Radiology Review Manual

fourth edition

Help

Introduction

Musculoskeletal System
DDx, Anatomy, Disorders

Central Nervous System - Skull and Spine
DDx, Anatomy, Disorders

Central Nervous System - Brain
DDx, Anatomy, Disorders

Orbit
DDx, Anatomy, Disorders

Ear, Nose, And Throat
DDx, Anatomy, Disorders

Chest
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Breast
DDx, Anatomy, Disorders

Heart and Great Vessels
DDx, Anatomy, Disorders

Liver, Bile Ducts, Pancreas, and Spleen
DDx, Anatomy, Disorders

Gastrointestinal Tract
DDx, Anatomy, Disorders

Urogenital Tract
DDx, Anatomy, Disorders

Obstetrics and Gynecology
DDx, Anatomy, Disorders

Nuclear Medicine, Statistics, Contrast

Product Information

LIPPINCOTT WILLIAMS & WILKINS
A Wolters Kluwer Company

Dahnert's Radiology Review Manual on CD-ROM

Navigation

Product Features

Navigation

Viewing Content

The *Dahnert's Radiology Review Manual on CD-ROM* home page is organized similarly to the table of contents in the print version. Topics that are underlined and in blue text are "hot." You can click on a "hot" topic (and down a hierarchy of topics, as applicable) to go to that section of text.

Browser Toolbar

The horizontal row of buttons near the top of the browser window is called the "toolbar." These buttons are not unique to this product and have basically the same functionality for any web site you view. These commonly-used features are accessible by clicking a toolbar button. For detailed information, refer to the online Help for your browser.

Do use the toolbar to:

- Go back and forth between previously-viewed pages (using the BACK and FORWARD buttons).
- Print the currently-displayed page.

Do NOT use the toolbar to:

- Search this site. Clicking the Search toolbar button will activate the Search across the web. To limit your search to this product, see Search.
- Go to the Home page. Clicking the Home toolbar button will take you to your browser's default page. To go to the Home page of this product, click the "Home" text link at the top of any page in this product.

Product Features

Search

Use the extensive word search capabilities to retrieve information on a specific topic of interest.

Basic search

1. Go to the Home page. (If you are already there, go to Step 2.)
2. Within the search box, enter your search term(s).
3. Click the **Search** button to process the search. (Note: Do not use the Search button on the browser toolbar.)
4. The Search Results page will display all of the entries containing your term. Click the desired entry to go to that section of the text.

Multiple Word Search

To retrieve material relating to multiple terms, enter your search terms one after another, spacing once between words and using no punctuation. For example, entering *prostate hypertrophy carcinoma* would retrieve a list of topics containing all three words.

Exact Phrase Search

To retrieve material relating to an exact phrase, enter the phrase within quotation marks. For example, entering *"benign prostatic hypertrophy"* would retrieve a list of topics containing this phrase.

Print

1. Go to the topic you wish to print.
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3. At the Print dialog box, select any applicable print options and click **OK**. (If you need to set up your printer, click **Setup** from the Print dialog box.)

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Home

INTRODUCTION

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Home

MUSCULOSKELETAL SYSTEM

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OSTEOMA

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CENTRAL NERVOUS SYSTEM

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Differential diagnosis of skull and spine disorders

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CAUDA EQUINA SYNDROME

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- Frontoethmoidal Encephalocele (13-15%)
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PROGRESSIVE MULTIFOCAL LEUKOENCEPHALOPATHY

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ORBIT

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Differential diagnosis of orbital disorders

OPHTHALMOPLEGIA

ANOPIA

OCULAR TRAUMA

ORBIT

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- Intraconal Lesion
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- Optic Nerve Enlargement

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LYMPHOMA OF ORBIT

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- Opacified Sinus & Expansion / Destruction

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- Airway Obstruction In Children
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- Aryepiglottic Cyst

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- Lymph Node Enlargement Of Neck
- Congenital Cystic Lesions Of Neck
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Thyroid Nodule

Discordant Thyroid Nodule

Hot Thyroid Nodule

Cold Thyroid Nodule

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Deep spaces of suprahyoid head & neck

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Ear, nose, and throat disorders

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MUCOCELE

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- Single-cavity Cochlea
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RHINOCEREBRAL MUCORMYCOSIS

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- Painless Thyroiditis
- Acute Suppurative Thyroiditis

WARTHIN TUMOR

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Home

CHEST

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Differential diagnosis of chest disorders

PNEUMONIA

- Lobar Pneumonia
- Lobular Pneumonia
- Interstitial Pneumonia
- Cavitating Pneumonia
- Pulmonary Infiltrates In Neonate
- Recurrent Pneumonia In Childhood
- Gram-negative Pneumonia
- Mycotic Infections Of Lung
- Hypersensitivity To Organic Dusts
- Drug-induced Pulmonary Damage

PULMONARY MASS

- Differential-diagnostic Features Of Lung Masses
- Benign Lung Tumor
- Solitary Nodule / Mass
- Large Pulmonary Mass
- Cavitating Lung Nodule
- Shaggy Pulmonary Nodule
- Hemorrhagic Pulmonary Nodule
- Multiple Nodules And Masses
- Pneumoconiosis Classification
- Pleura-based Lung Nodule
- Focal Area Of Ground-glass Attenuation
- Intrathoracic Mass Of Low Attenuation

PULMONARY CALCIFICATIONS

- Multiple Pulmonary Calcifications
- Calcified Pulmonary Nodules

LUCENT LUNG LESIONS

- Hyperlucent Lung
- Localized Lucent Lung Defect
- Multiple Lucent Lung Lesions
- Pulmonary Cyst
- Multiple Thin-walled Cavities

MEDIASTINUM

- Mediastinal Shift
- Pneumomediastinum
- Mediastinal Fat
- Acute Mediastinal Widening
- Mediastinal Mass
- Low-attenuation Mediastinal Mass
- Mediastinal Cysts
- Hilar Mass
- Eggshell Calcification Of Nodes
- Enlargement Of Azygos Vein

THYMUS

- Thymic Mass
- Diffuse Thymic Enlargement

TRACHEA & BRONCHI

- Tracheal Tumor
- Endobronchial Tumor
- Bronchial Obstruction
- Mucoid Impaction
- Signet-ring Sign
- HRCT Classification Of Bronchiolar Disease
- Bronchial Wall Thickening
- Broncholithiasis

PLEURA

- Pneumothorax
- Pleural Effusion
- Hemothorax
- Solitary Pleural Mass
- Multiple Pleural Densities
- Pleural Thickening
- Apical Cap
- Pleural Calcification

DIAPHRAGM

- Bilateral Diaphragmatic Elevation
- Unilateral Diaphragmatic Elevation

CHEST WALL

- Chest Wall Lesions
- Lung Disease With Chest Wall Extension
- Malignant Tumors Of Chest Wall In Children
- Pancoast Syndrome

PULMONARY MALFORMATION

NEONATAL LUNG DISEASE

- Mediastinal Shift & Abnormal Aeration
- Reticulogranular Densities In Neonate
- Hyperinflation In Newborn
- Hyperinflation In Child

PULMONARY HEMORRHAGE

BEDSIDE CHEST RADIOGRAPHY

Function and anatomy of lung

Chest disorders

BRONCHIOLITIS OBLITERANS WITH ORGANIZING PNEUMONIA (BOOP)

BRONCHIOLOALVEOLAR CARCINOMA

BRONCHOGENIC CARCINOMA

BRONCHOGENIC CYST

BRONCHOPULMONARY DYSPLASIA

BRONCHOPLEURAL FISTULA

BRONCHOPULMONARY SEQUESTRATION

- Intralobar Sequestration (75-86%)
- Extralobar Sequestration (14-25%)

CANDIDIASIS

- CASTLEMAN DISEASE
- Localized / Unicentric Angiofollicular Lymph Node Hyperplasia
- Generalized / Multicentric Angiofollicular Lymph Node Hyperplasia

CHRONIC EOSINOPHILIC PNEUMONIA

CHRONIC MEDIASTINITIS

- Mediastinal Granuloma
- Fibrosing Mediastinitis

CHURG-STRAUSS SYNDROME

CHYLOTHORAX

COAL WORKERS PNEUMOCONIOSIS

COCCIDIOIDOMYCOSIS

- Primary Coccidioidomycosis
- Chronic Respiratory Coccidioidomycosis
- Disseminated Coccidioidomycosis (in 1%)

CONGENITAL LOBAR EMPHYSEMA

CONGENITAL LYMPHANGIECTASIA

CONGENITAL PULMONARY VENOLOBAR SYNDROME

CRYPTOCOCCOSIS

CYSTIC ADENOMATOID MALFORMATION

CYSTIC FIBROSIS

DIAPHRAGMATIC HERNIA

- Congenital Diaphragmatic Hernia
- Traumatic Diaphragmatic Hernia

EMPHYSEMA

- Centrilobular Emphysema
- Panacinar Emphysema
- Paracicatricial Emphysema
- Paraseptal Emphysema

EMPYEMA

EXTRAMEDULLARY PLASMACYTOMA

EXTRINSIC ALLERGIC ALVEOLITIS

FAT EMBOLISM

FOCAL ORGANIZING PNEUMONIA

FRACTURE OF TRACHEA / BRONCHUS

GOODPASTURE SYNDROME

GRANULOMA OF LUNG

HAMARTOMA OF CHEST WALL

HAMARTOMA OF LUNG

HEREDITARY HEMORRHAGIC TELANGIECTASIA

HISTOPLASMOSIS

- Pulmonary Histoplasmosis

HYDATID DISEASE

HYPOGENETIC LUNG SYNDROME

IDIOPATHIC INTERSTITIAL PNEUMONIA

- Acute Interstitial Pneumonia
- Subacute Interstitial Pneumonia
- Chronic Interstitial Pneumonia

IDIOPATHIC PULMONARY HEMOSIDEROSIS

KARTAGENER SYNDROME

KLEBSIELLA PNEUMONIA

LANGERHANS CELL HISTIOCYTOSIS

LEGIONELLA PNEUMONIA

LIPOID PNEUMONIA

LÖFFLER SYNDROME

LUNG TORSION

LUNG TRANSPLANT

- Acute Rejection Of Lung Transplant
- Anastomotic Complications Of Lung Transplant
- Chronic Rejection Of Lung Transplant
- Hyperacute Rejection
- Posttransplantation Infection
- Posttransplantation Lymphoproliferative Disease
- Reperfusion Edema

LYMPHANGIOMYOMATOSIS

LYMPHANGITIC CARCINOMATOSIS

LYMPHOID INTERSTITIAL PNEUMONIA

LYMPHOMA

- Hodgkin Disease
- Non-Hodgkin Lymphoma
- Non-Hodgkin Lymphoma In Childhood

MECONIUM ASPIRATION SYNDROME

MEDIASTINAL LIPOMATOSIS

MESOTHELIOMA

- Benign Mesothelioma
- Malignant Mesothelioma

METASTASIS TO LUNG

- Solitary Metastatic Lung Nodule
- Calcifying Lung Metastases (<1%)
- Cavitating Lung Metastases (4%)
- Hemorrhagic Lung Metastases

- Endobronchial Metastases
- Lung Metastases In Childhood

METASTASIS TO PLEURA

MYCOPLASMA PNEUMONIA

NEAR DROWNING

NEONATAL PNEUMONIA

NOCARDIOSIS

NONTUBERCULOUS MYCOBACTERIAL INFECTION OF LUNG

PANBRONCHIOLITIS

PARAGONIMIASIS OF LUNG

PERICARDIAL CYST

PNEUMATOCELE

PNEUMOCOCCAL PNEUMONIA

PNEUMOCYSTOSIS

PNEUMONECTOMY CHEST

POSTOBSTRUCTIVE PNEUMONIA

PROGRESSIVE MASSIVE FIBROSIS

PSEUDOLYMPHOMA

PSEUDOMONAS PNEUMONIA

PULMONARY ARTERIAL MALFORMATION

PULMONARY CAPILLARY HEMANGIOMATOSIS

PULMONARY CONTUSION

PULMONARY INTERSTITIAL EMPHYSEMA

PULMONARY LYMPHANGIOMATOSIS

PULMONARY MAINLINE GRANULOMATOSIS

PULMONARY THROMBOEMBOLIC DISEASE

- Acute Thromboembolic Pulmonary Arterial Hypertension
- Chronic Thromboembolic Pulmonary Arterial Hypertension

PULMONARY VENOUS VARIX

RADIATION PNEUMONITIS

RESPIRATORY DISTRESS SYNDROME OF NEWBORN

RHEUMATOID LUNG

ROUND PNEUMONIA

SARCOIDOSIS

SEPTIC PULMONARY EMBOLI

SIDEROSIS

SILICOSIS

- Acute Silicoproteinosis
- Chronic Simple Silicosis
- Complicated Silicosis
- Silicotuberculosis
- Caplan Syndrome

SJÖGREN SYNDROME

STAPHYLOCOCCAL PNEUMONIA

STREPTOCOCCAL PNEUMONIA

SWYER-JAMES SYNDROME

SYSTEMIC LUPUS ERYTHEMATOSUS

TALCOSIS

TERATOID TUMOR OF MEDIASTINUM

THORACIC PARAGANGLIOMA

THYMIC CYST

THYMIC HYPERPLASIA

THYMOLIPOMA

THYMOMA

- Noninvasive = Benign Thymoma
- Invasive [Malignant] Thymoma

TORSION OF LUNG

TRACHEOBRONCHOMEGALY

TRACHEOBRONCHOPATHIA OSTEOCHONDROPLASTICA

TRANSIENT TACHYPNEA OF THE NEWBORN

TRAUMATIC LUNG CYST

TUBERCULOSIS

- Primary Pulmonary Tuberculosis
- Postprimary Pulmonary Tuberculosis
- Miliary Pulmonary Tuberculosis

UNILATERAL PULMONARY AGENESIS

VARICELLA-ZOSTER PNEUMONIA

VIRAL PNEUMONIA

WEGENER GRANULOMATOSIS

- Limited Wegener Granulomatosis
- Midline Granuloma

WILLIAMS-CAMPBELL SYNDROME

WILSON-MIKITY SYNDROME

ZYGOMYCOSIS

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Home

BREAST

Differential diagnosis of breast disorders

Breast anatomy and mammographic technique

BREAST ANATOMY

- Lobes
- Terminal Duct Lobular Unit (TDLU)
- Components Of Normal Breast Parenchyma
- Parenchymal Breast Pattern (László Tabár)

MAMMOGRAPHIC FILM READING TECHNIQUE

MAMMOGRAPHIC TECHNIQUE

- Factors Affecting Mammographic Image Quality

Breast disorders

BREAST CANCER

- Epidemiology Of Breast Cancer
- Breast Cancer Evaluation
- Screening Of Asymptomatic Patients
- Role Of Mammography
- Role Of Breast Ultrasound
- Role Of Breast MRI
- Role Of Stereotaxic Biopsy

BREAST CYST

- Simple Breast Cyst
- Complex Breast Cyst

CARCINOMA OF MALE BREAST

CHRONIC ABSCESS OF BREAST

CYSTOSARCOMA PHYLLODES

DERMATOPATHIC LYMPHADENOPATHY

EPIDERMAL INCLUSION CYST

FAT NECROSIS OF BREAST

FIBROADENOMA

- Juvenile / Giant / Cellular Fibroadenoma

FIBROCYSTIC CHANGES

- Adenosis
- Sclerosing Adenosis
- Fibrosis
- Atypical Lobular Hyperplasia
- Atypical Ductal Hyperplasia
- Intraductal Papillomatosis

GALACTOCELE

GRANULAR CELL TUMOR

GYNECOMASTIA

HAMARTOMA OF BREAST

HEMATOMA OF BREAST

JUVENILE PAPILLOMATOSIS

LACTATING ADENOMA

LIPOMA OF BREAST

LYMPHOMA OF BREAST

MAMMARY DUCT ECTASIA

MAMMOPLASTY

- Augmentation Mammoplasty
- Reduction Mammoplasty

MASTITIS

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Home

HEART AND GREAT VESSELS

Differential diagnosis of cardiovascular disorders

Cardiovascular anatomy and echocardiography

Cardiovascular disorders

- Symptomatic CoA
- Asymptomatic CoA

CONGENITAL ABSENCE OF PULMONARY VALVE

CONGESTIVE HEART FAILURE

CONSTRICTIVE PERICARDITIS

CORONARY ARTERY FISTULA

COR TRIATRIATUM

DEEP VEIN THROMBOSIS

DOUBLE-OUTLET RIGHT VENTRICLE

DUCTUS ARTERIOSUS ANEURYSM

EBSTEIN ANOMALY

EISENMENGER COMPLEX

EISENMENGER SYNDROME

ENDOCARDIAL CUSHION DEFECT

ENDOCARDIAL FIBROELASTOSIS

FLAIL MITRAL VALVE

HYPOPLASTIC LEFT HEART SYNDROME

HYPOPLASTIC RIGHT VENTRICLE

IDIOPATHIC DILATATION OF PULMONARY ARTERY

INTERRUPTION OF AORTIC ARCH

INTERRUPTION OF PULMONARY ARTERY

INTRAVENOUS DRUG ABUSE

ISCHEMIC HEART DISEASE

KAWASAKI SYNDROME

MICROSCOPIC POLYANGIITIS

MITRAL REGURGITATION

MITRAL STENOSIS

MITRAL VALVE PROLAPSE

MYOCARDIAL INFARCTION

- Right Ventricular Infarction

MYXOMA

- Carney Syndrome

PATENT DUCTUS ARTERIOSUS

- PDA In Premature Infant
- Beneficial PDA
- Nonbeneficial PDA

PENETRATING AORTIC ULCER

PERICARDIAL DEFECT

PERSISTENT FETAL CIRCULATION

POLYARTERITIS NODOSA

POLYSPLENIA SYNDROME

POPLITEAL ARTERY ENTRAPMENT SYNDROME

PRIMARY PULMONARY HYPERTENSION

PSEUDOCOARCTATION

PULMONARY ATRESIA

PULMONARY VENO-OCCLUSIVE DISEASE

PULMONIC STENOSIS

- Subvalvular Pulmonic Stenosis
- Valvular Pulmonic Stenosis
- Supravalvular Pulmonic Stenosis

RAYNAUD SYNDROME

- Raynaud Disease
- Raynaud Phenomenon

RHABDOMYOMA OF HEART

SINGLE VENTRICLE

SINUS OF VALSALVA ANEURYSM

SPLENIC ARTERY ANEURYSM

SUBCLAVIAN STEAL SYNDROME

- Partial Subclavian Steal Steal Syndrome
- Occult Subclavian Steal Syndrome

SUPERIOR VENA CAVA SYNDROME

SYPHILITIC AORTITIS

TAKAYASU ARTERITIS

TEMPORAL ARTERITIS

TETRALOGY OF FALLOT

- Pink Tetralogy
- Pentalogy Of Fallot
- Trilogy Of Fallot

THORACIC OUTLET SYNDROME

TRANSPOSITION OF GREAT ARTERIES

- Complete Transposition oOf Great Arteries
- Corrected Transposition Of Great Arteries

TRICUSPID ATRESIA

TROUSSEAU SYNDROME

TRUNCUS ARTERIOSUS

- Hemitruncus
- Pseudotruncus Arteriosus

VENTRICULAR ANEURYSM

- True Ventricular Aneurysm
- Pseudoaneurysm Of Ventricle

VENTRICULAR SEPTAL DEFECT

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LIVER, BILE DUCTS, PANCREAS, AND SPLEEN

Differential diagnosis of hepatic, biliary, pancreatic, and splenic disorders

RIGHT UPPER QUADRANT PAIN

LIVER

- Diffuse Hepatic Enlargement
- Increased Liver Attenuation
- Generalized Increase In Liver Echogenicity
- Primary Benign Liver Tumor
- Primary Malignant Liver Tumor
- Focal Liver Lesion
- Solitary Echogenic Liver Mass
- Bulls-eye Lesions Of Liver
- Cystic Liver Lesion
- Vascular "Scar" Tumor Of Liver
- Low-density Mass In Porta Hepatis
- Low-density Hepatic Mass With Enhancement
- Fat-containing Liver Mass
- Hepatic Calcification
- Portal Venous Gas
- Hyperperfusion Abnormalities Of Liver
- Dampening Of Hepatic Vein Doppler Waveform
- Aberrant Hepatic Artery

GALLBLADDER

- Nonvisualization Of Gallbladder On OCG
- Nonvisualization Of Gallbladder On US
- High-density Bile
- Displaced Gallbladder
- Alteration In Gallbladder Size
- Diffuse Gallbladder Wall Thickening
- Focal Gallbladder Wall Thickening
- Mobile Intraluminal Mass In Gallbladder
- Comet-tail Artifact In Liver And Gallbladder
- Echogenic Fat In Hepatoduodenal Ligament

BILE DUCTS

- Gas In Biliary Tree
- Obstructive Jaundice In Adult
- Neonatal Obstructive Jaundice
- Large Nonobstructed CBD
- Filling Defect In Bile Ducts
- Bile Duct Narrowing
- Papillary Stenosis
- Periampullary Tumor

- Double-duct Sign
- Congenital Biliary Cysts

PANCREAS

- Congenital Pancreatic Anomalies
- Pancreatic Calcification
- Fatty Replacement & Atrophy Of Pancreas
- Pancreatic Mass
- Pancreatic Neoplasm
- Hypervascular Pancreatic Tumors
- Pancreatic Cyst
- Hyperamylasemia

SPLEEN

- Nonvisualization Of Spleen
- Small Spleen
- Splenomegaly
- Splenic Lesion
- Solid Splenic Lesion
- Cystic Splenic Lesion
- Increased Splenic Density
- Splenic Calcification
- Iron Accumulation In Spleen
- Hyperechoic Splenic Spots

Anatomy of liver, bile ducts, and pancreas

Disorders of liver, biliary tract, pancreas, and spleen

ECHINOCOCCAL DISEASE

- Echinococcus Granulosus
- Echinococcus Multilocularis

EPIDERMOID CYST OF SPLEEN

EPITHELIOID HEMANGIOENDOTHELIOMA

FATTY LIVER

- Diffuse Fatty Infiltration
- Focal Fatty Infiltration

FOCAL NODULAR HYPERPLASIA

GALLBLADDER CARCINOMA

GLYCOGEN STORAGE DISEASE

HEMOCHROMATOSIS

- Genetic Hemochromatosis
- Secondary Hemochromatosis

HEPATIC ABSCESS

- Amebic Abscess
- Pyogenic Liver Abscess

HEPATIC ADENOMA

HEPATIC ANGIOSARCOMA

HEPATIC CYST

HEPATIC HEMANGIOMA

- Cavernous hemangioma of liver
- Infantile Hemangioendothelioma Of Liver

HEPATITIS

- Acute Hepatitis
- Chronic Hepatitis

HEPATOBLASTOMA

HEPATOCELLULAR CARCINOMA

- Fibrolamellar Hepatocellular Carcinoma

HYPERPLASTIC CHOLECYSTOSIS

- Cholesterolosis
- Adenomyomatosis Of Gallbladder

HYPOSPLENISM

LIPOMA OF LIVER

LIVER TRANSPLANT

- Vascular Complications In Liver Transplant
- Parenchymal Complications In Liver Transplant
- Biliary Complications In Liver Transplant

LYMPHOMA OF LIVER

MACROCYSTIC ADENOMA OF PANCREAS

MESENCHYMAL HAMARTOMA OF LIVER

METASTASES TO LIVER

- Calcified Liver Metastases
- Hypervascular Liver Metastases
- Hemorrhagic Liver Metastases

SCHISTOSOMIASIS

SCHWACHMAN-DIAMOND SYNDROME

SOLID AND PAPILLARY NEOPLASM OF PANCREAS

SPLENIC ANGIOSARCOMA

SPLENIC HAMARTOMA

SPLENIC HEMANGIOMA

SPLENIC INFARCTION

SPLENIC TRAUMA

SPLENOSIS

SPONTANEOUS PERFORATION OF COMMON BILE DUCT

THOROTRASTOSIS

UNDIFFERENTIATED SARCOMA OF LIVER

WANDERING SPLEEN

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Home

GASTROINTESTINAL TRACT

LIPPINCOTT WILLIAMS & WILKINS
A Wolters Kluwer Company

Differential diagnosis of gastrointestinal disorders

ABNORMAL INTRA-ABDOMINAL AIR

- Abnormal Air Collection
- Pneumoperitoneum
- Pseudopneumoperitoneum
- Pneumoretroperitoneum
- Pneumatosis Intestinalis
- Soap-bubble Appearance In Abdomen Of Neonate

ABDOMINAL CALCIFICATIONS & OPACITIES

- Opaque Material In Bowel
- Diffuse Abdominal Calcifications
- Focal Alimentary Tract Calcifications
- Abdominal Wall Calcifications
- Abdominal Vascular Calcifications

ABNORMAL INTRA-ABDOMINAL FLUID

- Ascites
- Fluid Collections
- Intra-abdominal Cyst In Childhood

MECHANICAL INTESTINAL OBSTRUCTION

- Common Causes Of Obstruction In Children
- Gastric Outlet Obstruction
- Duodenal Obstruction
- Jejunal And Ileal Obstruction
- Colonic Obstruction

ILEUS

- Localized Ileus
- Intestinal Pseudoobstruction

ESOPHAGUS

- Esophageal Contractions
- Abnormal Esophageal Peristalsis
- Diffuse Esophageal Dilatation
- Air Esophagogram
- Abnormal Esophageal Folds
- Esophageal Inflammation
- Esophageal Ulceration
- Double-barrel Esophagus
- Esophageal Diverticulum
- Tracheobronchoesophageal Fistula
- Long Smooth Esophageal Narrowing
- Focal Esophageal Narrowing
- Esophageal Filling Defect

- Esophageal Mucosal Nodules / Plaques
- Extrinsic Esophageal Impression

STOMACH

- Widened Retrogastric Space
- Gastric Pneumatosis
- Gastric Atony
- Narrowing Of Stomach
- Intramural-extramucosal Lesions Of Stomach
- Gastric Filling Defects
- Filling Defect Of Gastric Remnant
- Thickened Gastric Folds
- Gastric Ulcer
- Bulls-eye Lesions
- Complications Of Postoperative Stomach
- Lesions Involving Stomach And Duodenum

DUODENUM

- Extrinsic Pressure Effect On Duodenum
- Thickened Duodenal Folds
- Duodenal Filling Defect
- Duodenal Tumor
- Enlargement Of Papilla Of Vater
- Duodenal Narrowing
- Dilated Duodenum
- Postbulbar Ulceration

SMALL BOWEL

- Small Bowel Diverticula
- Small Bowel Ulcer
- Separation Of Bowel Loops
- Normal Small Bowel Folds & Diarrhea
- Dilated Small Bowel & Normal Folds
- Abnormal Small Bowel Folds
- Atrophy Of Folds
- Ribbonlike Bowel
- Delayed Small Bowel Transit
- Multiple Stenotic Lesions Of Small Bowel
- Small Bowel Filling Defects
- Small Bowel Tumors

CECUM

- Ileocecal Valve Abnormalities
- Coned Cecum
- Cecal Filling Defect

COLON

- Colon Cutoff Sign
- Colonic Thumbprinting
- Colonic Urticaria Pattern

Anatomy and function of gastrointestinal tract

Gastrointestinal disorders

ACHALASIA

ADENOMA OF SMALL BOWEL

ADENOMATOUS COLONIC POLYP

ADENOCARCINOMA OF SMALL BOWEL

AFFERENT LOOP SYNDROME

AIDS

AMEBIASIS

AMYLOIDOSIS

ANGIODYSPLASIA OF COLON

ANISAKIASIS

ANORECTAL MALFORMATION

ANTRAL MUCOSAL DIAPHRAGM

APPENDICITIS

ASCARIASIS

BANNAYAN-RILEY-RUVALCABA SYNDROME

BARRETT ESOPHAGUS

BEHÇET SYNDROME

- Intestinal Behçet Disease

BEZOAR

BLUNT ABDOMINAL TRAUMA

- Hemoperitoneum
- Hypovolemia
- Blunt Trauma To Spleen
- Blunt Trauma To Liver (20%)
- Blunt Trauma To Gallbladder (2%)
- Blunt Trauma To GI Tract (5%)
- Blunt Trauma To Pancreas (3%)
- Blunt Trauma To Kidney
- Blunt Trauma To Ureteropelvic junction (rare)
- Blunt Trauma To Bladder

BOERHAAVE SYNDROME

BRUNNER GLAND HYPERPLASIA

BURKITT LYMPHOMA

CARCINOID

CATHARTIC COLON

CHAGAS DISEASE

CHALASIA

CHRONIC IDIOPATHIC INTESTINAL PSEUDOOBSTRUCTION

COLITIS CYSTICA PROFUNDA

COLORECTAL CARCINOMA

- Lynch Syndrome
- Rectal Cancer

COLONIC VOLVULUS

CONGENITAL INTESTINAL ATRESIA

CRICOPHARYNGEAL ACHALASIA

COWDEN DISEASE

CROHN DISEASE

CRONKHITE-CANADA SYNDROME

DESMOID TUMOR

DIAPHRAGM DISEASE

DISACCHARIDASE DEFICIENCY

DISTAL INTESTINAL OBSTRUCTION SYNDROME

DIVERTICULAR DISEASE OF COLON

- Prediverticular Disease Of Colon
- Colonic Diverticulosis
- Colonic Diverticulitis
- Colonic Diverticular Hemorrhage

DUMPING SYNDROME

DUODENAL ATRESIA

DUODENAL DIVERTICULUM

DUODENAL ULCER

DUODENAL VARICES

DUPLICATION CYST

- Colonic Duplication Cyst
- Duodenal Duplication Cyst
- Esophageal Duplication Cyst
- Gastric Duplication Cyst
- Rectal Duplication Cyst
- Small Bowel Duplication Cyst
- Thoracoabdominal Duplication

ECTOPIC PANCREAS

ENTERIC CYST

EOSINOPHILIC GASTROENTERITIS

EPIPLOIC APPENDAGITIS

ESOPHAGEAL ATRESIA & TRACHEOESOPHAGEAL FISTULA

- Esophageal Atresia Without Fistula (8-9%)
- Esophageal Atresia With Fistula
- Tracheo-esophageal Fistula Without Atresia (6%)

ESOPHAGEAL CANCER

ESOPHAGEAL INTRAMURAL PSEUDODIVERTICULOSIS

ESOPHAGEAL PERFORATION

ESOPHAGEAL VARICES

ESOPHAGEAL WEB

ESOPHAGITIS

- Acute Esophagitis

- Candida Esophagitis
- Caustic Esophagitis
- Chronic Esophagitis
- Cytomegalovirus Esophagitis
- Drug-induced Esophagitis
- Herpes Esophagitis
- Human Immunodeficiency Virus Esophagitis
- Reflux Esophagitis
- Viral esophagitis

FAMILIAL ADENOMATOUS POLYPOSIS

GALLSTONE ILEUS

GANGLIOCYTIC PARAGANGLIOMA

GARDNER SYNDROME

GASTRIC CARCINOMA

- Early Gastric Cancer (20%)
- Advanced Gastric Cancer (T2 lesion and higher)

GASTRIC DIVERTICULUM

GASTRIC POLYP

GASTRIC ULCER

- Benign Gastric Ulcer
- Malignant Gastric Ulcer

GASTRIC VARICES

GASTRIC VOLVULUS

GASTRITIS

- Corrosive Gastritis
- Emphysematous Gastritis
- Erosive Gastritis
- Phlegmonous Gastritis

GIARDIASIS

GLYCOGEN ACANTHOSIS

GRAFT-VERSUS-HOST DISEASE

HELICOBACTER PYLORI INFECTION

HEMANGIOMA OF SMALL BOWEL

HENOCH-SCHÖNLEIN PURPURA

HERNIA

- External Hernia
- Internal Hernia
- Hiatal Hernia
- Umbilical Hernia

HIRSCHSPRUNG DISEASE

HODGKIN DISEASE

HYPERPLASTIC POLYP OF COLON

HYPERTROPHIC PYLORIC STENOSIS

- Infantile Form Of Hypertrophic Pyloric Stenosis
- Adult Form Of Hypertrophic Pyloric Stenosis

- Focal Pyloric Hypertrophy

IMPERFORATE ANUS

INTESTINAL LYMPHANGIECTASIA

INTRALUMINAL DUODENAL DIVERTICULUM

INTRAMURAL ESOPHAGEAL RUPTURE

INTUSSUSCEPTION

ISCHEMIC COLITIS

JEJUNOILEAL DIVERTICULAR DISEASE

JUVENILE POLYPOSIS

KAPOSI SARCOMA

LADD BANDS

LEIOMYOMA

- Esophageal Leiomyomatosis
- Leiomyoma Of Esophagus
- Leiomyoma Of Small Bowel
- Leiomyoma Of Stomach

LEIOMYOSARCOMA

- Leiomyosarcoma Of Small Bowel
- Leiomyosarcoma Of Stomach
- Carney Syndrome

LIPOMA

LYMPHANGIOMA

LYMPHOGRANULOMA VENEREUM

LYMPHOID HYPERPLASIA

LYMPHOMA OF GASTROINTESTINAL TRACT

MALIGNANT MELANOMA

MALLORY-WEISS SYNDROME

MALROTATION

MASTOCYTOSIS

MECKEL DIVERTICULUM

MECONIUM ILEUS

MECONIUM PERITONITIS

MECONIUM PLUG SYNDROME

MELANOSIS COLI

MÉNÉTRIÈR DISEASE

MESENTERIC LYMPHADENITIS

MESENTERIC ISCHEMIA

MESOTHELIAL CYST

METASTASES TO SMALL BOWEL

METASTASES TO STOMACH

MIDGUT VOLVULUS

MUCOCELE OF APPENDIX

NECROTIZING ENTEROCOLITIS

PELVIC LIPOMATOSIS + FIBROLIPOMATOSIS

PERITONEAL MESOTHELIOMA

PERITONEAL METASTASES

PEUTZ-JEGHERS SYNDROME

POSTCRICOID DEFECT

POSTINFLAMMATORY POLYPOSIS

PRESBYESOPHAGUS

PROGRESSIVE SYSTEMIC SCLEROSIS

PROLAPSED ANTRAL MUCOSA

PSEUDOMEMBRANOUS COLITIS

PSEUDOMYXOMA PERITONEI

RADIATION INJURY

RETAINED GASTRIC ANTRUM

RETRACTILE MESENTERITIS

SCHATZKI RING

SMALL LEFT COLON SYNDROME

SOLITARY RECTAL ULCER SYNDROME

SPRUE

STRONGYLOIDIASIS

SUPERIOR MESENTERIC ARTERY SYNDROME

TAILGUT CYST

TOXIC MEGACOLON

TUBERCULOSIS

TURCOT SYNDROME

TYPHLITIS

ULCERATIVE COLITIS

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WALDENSTRÖM MACROGLOBULINEMIA

WHIPPLE DISEASE

ZENKER DIVERTICULUM

ZOLLINGER-ELLISON SYNDROME

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Home

UROGENITAL TRACT

Differential diagnosis of urogenital disorders

RENAL FAILURE

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- Chronic Renal Failure
- Musculoskeletal Manifestations Of CRF

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ARTERIAL HYPOTENSION

HYPERCALCEMIA

POLYCYTHEMIA

URINARY TRACT INFECTION

WETTING

MALE INFERTILITY

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BLADDER TRAUMA

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GANGLIONEUROMA

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WOLMAN DISEASE

ZELLWEGER SYNDROME

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Home

OBSTETRICS AND GYNECOLOGY

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Differential diagnosis of obstetic and gynecologic disorders

GENERAL OBSTETRICS

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VELAMENTOUS CORD INSERTION

NUCLEAR MEDICINE

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About the Author

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Dedication:

To my dear wife Sue,
to our children Mathias and Patrick
who mean so much to me

About the Author:

Wolfgang Dähnert was born in Hamburg, Germany. He studied medicine at the universities of Dusseldorf and Mainz, where he graduated in 1975. After internship and a short surgical residency he enrolled in a 4-year radiology residency program at the Johannes-Gutenberg University in Mainz and received his German certification for radiology in 1982. In 1984 he started a 2-year fellowship in ultrasound and computed tomography at the Johns Hopkins Hospital in Baltimore and was appointed Clinical Instructor at the same institution in 1986. During his Hopkins years he sat for the FLEX exam, and the radiology specialty exam with the American Board of Radiology. During these three years the foundation of Radiology Review Manual was laid. Between 1987 and 1989 he worked as Assistant Professor of Radiology in ultrasound at Thomas Jefferson Hospital in Philadelphia. During these three years Radiology Review Manual was taken to fruition. Since December of 1989 he has been associated with Clinical Diagnostic Radiology & Nuclear Medicine, a large subspecialized radiology group practice in Phoenix, Arizona, providing radiology services to Good Samaritan Regional Medical Center, St. Joseph's Hospital and Medical Center, both tertiary care hospitals in Phoenix, Good Samaritan Hospital in Lake Havasu City, and the Children's Hospital of Phoenix.

"Nothing in the world can take the place of persistence.
Talent will not; nothing is more common than unsuccessful men
with talent. Genius will not; unrewarded genius is almost a proverb.
Education will not; the world is full of educated derelicts.
Persistence and determination alone are omnipotent."

Calvin Coolidge 1872-1933
Vice President 1921-1923
President 1923-1929

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PREFACE

The depth of medical knowledge and scope of image interpretation expected from an average general radiologist has soared over the last two decades. The emergence of subspecialties within radiology is witness to this development. Books have become available on so many different imaging topics and in such a large number that it is impossible even for the avid reader to consume them all, catalogue them, and have instant access to them. While some radiologists have the luxury to practice exclusively in their area of special interest with impressive expertise, many practice a much broader scope of diagnostic radiology and find themselves occasionally in situations where recollections have become nebulous. I know that I regret my inability to recall many facts or - more frustrating - where to look them up. In a busy practice it is simply not possible to take time out and disappear in the library.

Radiology Review Manual has become my carry-on memory jogger, in an attempt to put into a single reference much of the information that is or could be relevant to my practice. I use it like a dictionary, always available at my workstation. It is published under the assumption that many colleagues practice like I do: trying to do a good job vis-a-vis significant time constraints. This concept has resonated well with the radiologic community. The popularity of the "green giant" or the "green bible", as it has been dubbed by residents, confirms the usefulness of this type of publication. At the time of this writing approximately 28,000 copies have been sold, one half outside the United States of America.

Radiology Review Manual was created in preparation for the specialty exam as the "book under the pillow." I have to credit the idea to publish this material to several residents at the Johns Hopkins Hospital who urged me to do so. Over the years, this material has been changed and expanded. Our voluminous field of diagnostic radiology makes it necessary to use an outline style for the sake of conserving space and thus provides only an extract of information. This may, at times, jeopardize the full meaning of statements when the context is lost. It should be kept in mind that this book is not intended for the novice and that it requires familiarity with the subject of radiology and the background information of major textbooks.

How to use this book:
The organization of this book has caused a major headache as any topic can be looked at from various points of view. I have selected just one of many possibilities to avoid redundancy. The material is presented in a manner that is in keeping with the topics of the current board exam. Unfortunately, this grouping is inconsistent, sectioning off by age (Pediatric Radiology) and image modality (Nuclear Medicine, Ultrasound). In order to avoid repetition, pediatric entities are subsumed within organ systems. Ultrasound and Nuclear Medicine are used from head to heel and consequently are mentioned in all body sections. However, Nuclear Medicine is treated in a separate section when emphasis is on technique and functional aspects not covered elsewhere. The skull and spine, a crossing point of many subspecialties, are dealt with as the first part of the CNS section. A section on eye, ear, nose, and throat topics is placed at the end of the section on CNS disorders. Small chapters on statistics and contrast media are added.

The organization within the individual chapters follows the practical approach of reading films. The initial step of film interpretation is the description of radiologic patterns that serves to identify categories in which they belong. Therefore, radiologic patterns for differential diagnoses are found in the first portion of a chapter. Once the diagnostic possibilities have been reviewed in brief outline, one can look up detailed information about a disease entity in the last segment of a chapter. The disease entities are presented in alphabetical order. Both these segments are separated by a few pages of functional, anatomic, or embryologic aspects. Occasionally, important clinical signs and their differential diagnoses, relevant to the practice of radiology, are included in the first portion of a chapter. Mnemonics (which I personally abhor) have been liberally added by request. Accepted therapies for contrast reactions are printed on the inside of the back cover page for immediate access. A table of contents and abbreviations used throughout the book are found in front. A user-friendly index, which selectively refers to those pages with significant information concludes the manual. Notice that many systemic diseases will be mentioned in more than one chapter with some unavoidable redundancy. However, emphasized are those manifestations of the disease that occur within the organ under which it is listed. The index also includes so-called "buzz words" that are miraculously attached to diseases.

The backbone of the book are disease entities, radiologic symptoms, as well as lists of differential diagnosis. Disease entities are headed by their most commonly used name with other designations listed below. As a radiologic diagnosis should be entertained in context with its probability to be correct, percentages in regard to frequency of signs and symptoms are included liberally, often giving the lowest and the highest number found in the literature. The truth may be somewhere in between for a nonselected patient population, and occasionally a third number is provided between the high and low number as the most frequently cited. Arbitrary choices have been made in situations when different or contradictory results are found in the literature - unfortunately, an occurrence not at all infrequent.

Lists of differential diagnoses can be presented in many fashions. There is no right or wrong way, but there certainly is a chaotic versus an organized approach. An orderly thought process portrays familiarity with a problem. Examinees have always felt that "nailing" the diagnosis is secondary, but including it in one's consideration is paramount to a successful exam. Accordingly, an attempt is made to categorize differential diagnostic considerations or etiologies of certain diseases in a manner digestible for recapitulation. It is a common experience that this is not always possible, logically satisfactory, or complete.

Acknowledgement:
The information contained herein has been gathered over several years and stems from various sources. The most significant ones are the journals dedicated to imaging with brilliant review articles, in particular the practice-oriented publication of Radiographics, ACR syllabi, handouts from various CME courses, hand-written notes taken during lectures, as well as feed-back from candidates having taken the board exam. Anecdotal contributions can no longer be traced. I realize, in retrospect, that this may present a problem when certain statements appear unlikely and their verification has to be left to the user. For my defense, I can only say that I have tried to extract all data as diligently as possible.

The following textbooks have been particularly helpful and deserve mention: Barkovich AJ: Pediatric Neuroimaging; Burgener FA, Kormano M: Differential Diagnosis in Conventional Radiology; Chapman S, Nakielny R: Aids to Radiological Differential Diagnosis; Davidson AJ: Radiology of the Kidney; Eideken J: Roentgen Diagnosis of Diseases of Bone; Fraser RG, Pare JAP: Diagnosis of Diseases of the Chest; Gedgaudas E, Moller JH, Castaneda-Zuniga WR, Amplatz K: Cardiovascular Radiology; Harnsberger HR: Handbooks in Radiology, Head and Neck Imaging; Kadir S: Diagnostic Angiography; Kirks DR: Practical Pediatric Imaging; Margulis AR, Burhenne HJ: Alimentary Tract Radiology; Megibow AJ, Balthazar EJ: Computed Tomography of the Gastrointestinal Tract; Mittelstaedt CA: Abdominal Ultrasound; Newton TH, Hasso AN, Dillon WP: Computed Tomography of the Head and Neck in Modern Neuroradiology; Reed JC: Chest Radiology: Plain Film Patterns and Differential Diagnosis; Reeder MM, Felson B: Gamuts in Radiology; Sanders RC, James AE: Ultrasonography in Obstetrics and Gynecology; Resnick D, Niwayama G: Diagnosis of Bone and Joint Disorders; Romero R, Pilu G, Jeanty P, Ghidini A, Hobbins JC: Prenatal Diagnosis of Congenital Anomalies; Swischuk LE: Plain Film Interpretation in Congenital Heart Disease; Tabar L, Dean PB: Teaching Atlas of Mammography; Taveras JM, Ferrucci JT: Radiology - Diagnosis - Imaging - Intervention.

I would like to acknowledge the input of numerous teachers, residents, and fellows at the Johns Hopkins Hospital in Baltimore, Thomas Jefferson University Hospital in Philadelphia as well as many colleagues that have helped subsequently. I am particularly indebted to the following individuals for reviewing the separate sections of this book: Christopher Canino, Thomas Chang, Adam E. Flanders, Keith Haidet, Charles Intenzo, David Karasick, Stephen Karasick, Alfred B. Kurtz, Esmond M. Mapp, Joel Raichlen, Paul Spirn, Robert M. Steiner, and C. Amy Wilson. My special thanks go to Flavius ("Buddy") Guglielmo, who supplied me with probably the largest collection of mnemonics in existence. While completing his training at Thomas Jefferson University Hospital, he compiled a long list of memory joggers together with Tom Helinek and Les Folio with contributions from Barbara McComb, Barry Tom, and Ron Wachsberg. Thomas S. Chang of Montefiori University Hospital in Pittsburgh has made valuable suggestions for improvement. My thanks also go to my colleague Ross Levatter for his thorough review of the section on nuclear medicine.

Finally, my thanks go to Charles W. Mitchell, senior editor at Williams & Wilkins, and his staff who have been able to reduce the paper weight of this edition and have kept its price reasonable and affordable for residents. They have also created a CD-ROM version, released in October 1997, for those who use computers at their reading stations or love to lug around their portable personal computers.

I sincerely hope that Radiology Review Manual will serve you in the same manner it has helped me in preparation for the board exam, in teaching situations, and particularly in my daily work assignments.

Phoenix, September 1998

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ABBREVIATIONS

√	radiologic sign
●	clinical sign, symptom
=	equals, is
@	at anatomic location of
/	or, per
+	and, plus, with
±	with or without
<	less than
>	more than, over
◊	important comment
AAA	abdominal aortic aneurysm
ABC	aneurysmal bone cyst
AC	abdominal circumference
ACA	anterior cerebral artery
ACE	angiotensin converting enzyme
ACom	anterior communicating artery
ACTH	adrenocorticotropic hormone
ADEM	acute disseminated encephalo-myelitis
ADH	antidiuretic hormone
AFP	alpha-fetoprotein
AICA	anterior inferior cerebellar artery
AIDS	acquired immune deficiency syndrome
ALL	acute lymphoblastic leukemia
AMA	antimitochondrial antibody
AML	acute myeloblastic leukemia
AML	angiomyolipoma
aML	anterior mitral valve leaflet
ANA	antinuclear antibodies
Angio	angiography
ANT	anterior
Ao	aorta
AP	anteroposterior
APUD	amine precursor uptake and decarboxylation
APVR	anomalous pulmonary venous return
ARA-C	arabinoside C
ARDS	acute respiratory distress syndrome
AS	aortic stenosis
ASA	acetylsalicylic acid
ASD	atrial septal defect
ASH	asymmetric septal hypertrophy
aTL	anterior tricuspid valve leaflet
ATN	acute tubular necrosis
AV	arteriovenous
AV	atrioventricular
AVF	arteriovenous fistula
AVM	arteriovenous malformation
AVN	avascular necrosis
AVNA	atrioventricular node artery
Ba	barium
BCDDP	breast cancer detection demonstration project
BCG	bacille Calmette-Guérin
BE	barium enema
BIDA	butyl iminodiacetic acid
BIH	benign intracranial hypertension
BKG	background
BOOP	Bronchiolitis obliterans organizing pneumonia
BP	blood pressure
BPD	biparietal diameter
BPH	benign prostatic hyperplasia
bpm	beats per minute
BPP	biophysical profile
BSA	body surface area
Bx	biopsy
Ca	calcium
CAD	coronary artery disease
CAM	cystic adenomatoid malformation
CBD	common bile duct
CC	craniocaudad
CCA	common carotid artery
CCAM	congenital cystic adenomatoid malformation
CCK	cholecystokinin
CDC	Center for Disease Control
CECT	contrast-enhanced computed tomography
CEMR	contrast-enhanced MR
CFI	color flow imaging

cGy	centigray = rad
CHD	common hepatic duct; congenital heart defect
CHF	congestive heart failure
CLL	chronic lymphatic leukemia
CMC	carpometacarpal
CML	chronic myelogenous leukemia
CMV	cytomegalovirus
CNS	central nervous system
CO	carbon monoxide
CoA	coarctation of aorta
COPD	chronic obstructive pulmonary disease
CPA	cerebellopontine angle
CPPD	calcium pyrophosphate dihydrate
CPR	cardiopulmonary resuscitation
CRT	cathode ray tube
CSF	cerebrospinal fluid
CST	contraction stress test
CT	cardiothoracic ratio
CT	computed tomography
CVA	cerebrovascular accident
CWP	coal worker's pneumoconiosis
Cx	complication
CXR	chest x-ray
DCIS	ductal carcinoma in situ
DDx	differential diagnosis
DES	diethylstilbestrol
DIC	disseminated intravascular coagulation
DIDA	diethyl iminodiacetic acid
DIL	drug-induced lupus erythematosus
DIP	desquamative interstitial pneumonia
DIP	distal interphalangeal
DISH	diffuse idiopathic skeletal hyperostosis
DISIDA	diisopropyl iminodiacetic acid
DIT	diiodotyrosine
DMSA	dimercaptosuccinic acid
DTPA	diethylenetriamine pentaacetic acid
DVT	deep vein thrombosis
Dx	diagnosis
EAC	external auditory canal
ECA	external carotid artery
ECD	endocardial cushion defect
ECF	extracellular fluid
ECG	electrocardiogram
ECHO	echocardiogram
ED	end-diastole
EDV	end-diastolic volume
EEG	electroencephalogram
EF	ejection fraction
EFW	estimated fetal weight
EG	eosinophilic granuloma
eg	exempli gratia
EHDP	ethylene hydroxydiphosphonate
ERC	endoscopic retrograde cholangiography
ES	end-systole
esp.	especially
ESR	erythrocyte sedimentation rate
ESV	end-systolic volume
F	female
FDA	Federal Drug Administration
FDG	fluorodeoxyglucose
FEV	forced expiratory volume
FIGO	Fédération Internationale de Gynécologie et d'Obstétrique
FISP	fast imaging with steady-state precession
FLASH	fast low-angle shot
FN	false negative
FNH	follicular nodular hyperplasia
FP	false positive
FRC	functional residual capacity
FS	fractional shortening
FSH	follicle stimulating hormone
FUO	fever of unknown origin
FWHM	full-width at half-maximum
GA	gestational age
GB	gallbladder
GBM	glioblastoma multiforme
GBS	group B streptococcus
Gd	gadolinium
GE	gastroesophageal
GER	gastroesophageal reflux
GFR	glomerular filtration rate
GI	gastrointestinal
GIST	gastrointestinal stromal tumor
GMRH	germinal matrix-related hemorrhage

GN	glomerulonephritis
GNRH	gonadotropin releasing hormone
GRE	gradient refocused echo
GU	genitourinary
Hb	hemoglobin
HC	head circumference
hCG	human chorionic gonadotropin
Hct	hematocrit
HD	Hodgkin disease
HIAA	hydroxyindole acetic acid
HIDA	hepatic 2,6-dimethyl iminodiacetic acid
HIP	health insurance plan
Histo	histology
HIV	human immunodeficiency virus
HL	Hodgkin lymphoma
HOCM	hypertrophic obstructive cardiomyopathy; high-osmolarity contrast media
HPT	hyperparathyroidism
HRCT	high-resolution CT
HSA	human serum albumin
HSE	herpes simplex encephalitis
HSG	hysterosalpingography
HSV	herpes simplex virus
HTLV	human T-cell lymphotropic virus
HU	Hounsfield unit
HWP	hepatic wedge pressure
Hx	history
IAC	internal auditory canal
ICA	internal carotid artery
IDA	iminodiacetic acid
IDM	infant of diabetic mother
IDP	iminodiphosphonate
ie	id est
IHSS	idiopathic hypertrophic subaortic stenosis
IM	intramuscular
IMA	inferior mesenteric artery
In	indium
IPF	idiopathic pulmonary fibrosis
IPH	idiopathic pulmonary hemosiderosis
IR	inversion recovery
IRP	international reference preparation
IS	ileosacral; international standard
IUD	intrauterine device
IUGR	intrauterine growth retardation
IV	intravenous
IVC	inferior vena cava
IVH	intraventricular hemorrhage
IVP	intravenous pyelogram
IVS	intraventricular septum
IVU	intravenous urogram
KCC	Kulchitzky cell carcinoma
KUB	kidney + ureter + bladder on one film
L	left
L-DOPA	3-(3,4-dihydroxyphenyl)-levo-alanin
LA	left atrium
LAD	left anterior descending
LAO	left anterior oblique
LAT	lateral
LATS	long-acting thyroid stimulating
LAV	lymphadenopathy-associated virus
LCA	left coronary artery
LCIS	lobular carcinoma in situ
LCX	left circumflex coronary artery
LDH	lactate dehydrogenase
LE	lupus erythematosus
LES	lower esophageal sphincter
LGA	large for gestational age
LH	luteinizing hormone
LIP	lymphocytic interstitial pneumonitis
LL	lower lobes
LLL	left lower lobe
LLQ	left lower quadrant
Lnn	lymph nodes
LOCM	low-osmolarity contrast media
LPA	left pulmonary artery
LPO	left posterior oblique
LSD	lysergic acid diethylamide
LUL	left upper lobe
LUQ	left upper quadrant
LV	left ventricle
LVET	left ventricular ejection time
LVFT2	left ventricular slow filling time

LVOT	left ventricular outflow tract
LVT1	left ventricular fast filling time
M	male
MA	menstrual age
MAA	macroaggregated albumin
MAG	mercaptoacetyltriglycine
MAI	Mycobacterium avium intracellulare
MCA	middle cerebral artery
MCDK	multicystic dysplastic kidney
MCK	multicystic kidney
MCP	metacarpophalangeal
MDP	methylene diphosphonate
MEA	multiple endocrine adenomas
MEN	multiple endocrine neoplasms
MFH	malignant fibrous histiocytoma
MIBG	metaiodobenzylguanidine
MID	multi-infarct dementia
MIT	monoiodotyrosine
ML	middle lobe
MLCN	multilocular cystic nephroma
MLO	mediolateral oblique
MMAA	mini-microaggregated albumin colloid
MMFR	maximal midexpiratory flow rate
MPS	mucopolysaccharidosis
MR	magnetic resonance
MS-AFP	maternal serum a-fetoprotein
MTP	metatarsophalangeal
MUGA	multiple gated acquisition
MV	mitral valve
Myelo	myelography
N.B.	nota bene
NBS	National Bureau of Standards
NEC	necrotizing enterocolitis
NECT	nonenhanced computed tomography
NHL	non-Hodgkin lymphoma
NPH	normal pressure hydrocephalus
NPH	nucleus pulposus herniation
npl	neoplasm
NPO	nulla per os
NSAID	nonsteroidal anti-inflammatory drug
NST	nonstress test
NTD	neural tube defect
NUC	nuclear medicine
OB-US	obstetrical ultrasound
OCG	oral cholecystogram
OCVM	occult vascular malformation
OHP	orthogonal-hole test pattern
OHSS	ovarian hyperstimulation syndrome
OIH	orthoiodohippurate
P	phosphorus
PA	posteroanterior
PA	pulmonary artery
PAC	premature atrial contraction
PAH	para-aminohippurate
PAP	primary atypical pneumonia
PAP	pulmonary alveolar proteinosis
PAPVR	partial anomalous pulmonary venous return
PAS	periodic acid Schiff
Path	pathology
PAVM	pulmonary arteriovenous malformation
PBF	pulmonary blood flow
PCA	posterior cerebral artery
PCAVC	persistent complete atrioventricular canal
PCKD	polycystic kidney disease
PCom	posterior communicating artery
PCP	Pneumocystis carinii pneumonia
PCWP	pulmonary capillary wedge pressure
PD	posterior descending artery
PDA	patent ductus arteriosus
PE	pulmonary embolism
PEEP	positive end expiratory pressure
PEP	preejection period
PET	positron emission tomography
pHPT	primary hyperparathyroidism
PICA	posterior inferior cerebellar artery
PIE	pulmonary infiltrate with eosinophilia
PIE	pulmonary interstitial emphysema
PIOPED	prospective investigation of pulmonary embolus detection
PIP	proximal interphalangeal
PIPIDA	paraisopropyl iminodiacetic acid
PLES	parallel-line-equal spacing
PM	photomultiplier
PMF	progressive massive fibrosis

PML	progressive multifocal leukoencephalopathy
pML	posterior mitral valve leaflet
PMN	polymorphonuclear
PMT	photomultiplier tube
PNET	primitive neuroectodermal tumor
PO	per oral
POST	posterior
PPD	purified protein derivative
PPG	photoplethysmography
PPLO	pleuropneumonia-like organism
ppm	posterior papillary muscle
PS	pulmonary stenosis
PSS	progressive systemic sclerosis
PTC	percutaneous transhepatic cholangiography
PTH	parathyroid hormone
pTL	posterior tricuspid valve leaflet
PTU	propylthiouracil
PVC	polyvinyl chloride
PVE	periventricular echogenicity
PVH	pulmonary venous hypertension
PVL	periventricular leukomalacia
PVNS	pigmented villonodular synovitis
PYP	pyrophosphate
PVR	pulse volume recording; postvoid residual
R	right
RA	rheumatoid arthritis
RA	right atrium
RAO	right anterior oblique
RBC	red blood cell
RCA	right coronary artery
RCC	renal cell carcinoma
RDS	respiratory distress syndrome
RES	reticuloendothelial system
RI	resistive index
RIND	reversible ischemic neurologic deficit
RISA	radioiodine serum albumin
RLL	right lower lobe
RLQ	right lower quadrant
RML	right middle lobe
ROC	receiver operating characteristic
ROI	region of interest
RPA	right pulmonary artery
RPF	renal plasma flow
RPO	right posterior oblique
RTA	renal tubular acidosis
RUL	right upper lobe
RV	residual volume
RV	right ventricle
RVOT	right ventricular outflow tract
Rx	therapy
S/P	status post
SAE	subcortical arteriosclerotic encephalopathy
SAG	sagittal
SAH	subarachnoid hemorrhage
SAM	systolic anterior motion of mitral valve
SANA	sinoatrial node artery
SBE	subacute bacterial endocarditis
SBO	salpingo-oophorectomy
SD	standard deviation
SE	spin echo
SGA	small for gestational age
sHPT	secondary hyperparathyroidism
SIJ	sacroiliac joint
SFA	superficial femoral artery
SLE	systemic lupus erythematosus
SMA	superior mesenteric artery
SMV	superior mesenteric vein
Sn	stannum
SOB	small bowel obstruction
SONK	spontaneous osteonecrosis of knee
S/P	status post
SPECT	single photon emission
SQ	subcutaneous
STIR	short tau inversion recovery
SV	stroke volume
SVC	superior vena cava
T1WI	T1-weighted image
T2WI	T2-weighted image
TAH	total abdominal hysterectomy
TAPVR	total anomalous pulmonary venous return
TB	tuberculosis
TBG	thyroxin-binding globulin
TBPA	thyroxin-binding prealbumin

TCC	transitional cell carcinoma
TDLU	terminal ductal lobular unit
TE	tracheoesophageal fistula
TGA	transposition of great arteries
tHPT	tertiary hyperparathyroidism
TIA	transitory ischemic attack
TLC	total lung capacity
TN	true negative
TOF	tetralogy of Fallot
TORCH	toxoplasmosis, rubella, cytomegalovirus, herpes virus
TP	true positive
TR	repetition time
TRH	thyrotropin-releasing hormone
TRV	transverse
TSH	thyroid-stimulating hormone
TURP	transurethral resection of prostate
TV	tidal volume
UGI	upper gastrointestinal series
UIP	usual interstitial pneumonia
UL	upper lobe
UPJ	ureteropelvic junction
US	ultrasound
USP XX	United States Pharmacopoeia, 20th edition
UTI	urinary tract infection
UVJ	ureterovesical junction
VC	vital capacity
VIP	vasoactive intestinal peptides
VMA	vanillylmandelic acid
V/Q	ventilation perfusion
VS	interventricular septum
VSD	ventricular septal defect
WBC	white blood cells
WDHA	watery diarrhea, hypokalemia, achlorhydria
WDHH	watery diarrhea, hypokalemia, hypochlorhydria
XGP	xanthogranulomatous pyelonephritis

DIFFERENTIAL-DIAGNOSTIC GAMUT OF BONE DISORDERS
Conditions to be considered = "dissect bone disease with a DIATTOM" **D**ysplasia + **D**ystrophy **I**nfection **A**nomalies of development **T**umor + tumorlike conditions **T**rauma **O**steochondritis + ischemic necrosis **M**etabolic disease DYSPLASIA= disturbance of bone growthDYSTROPHY= disturbance of nutrition

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Notes:

DELAYED BONE AGE

A.CONSTITUTIONAL1.Familial2.IUGRB.METABOLIC1.Hypopituitarism2.Hypothyroidism3.Hypogonadism (Turner syndrome)4.Cushing disease, steroid therapy5.Diabetes mellitus6.Rickets7.MalnutritionC.SYSTEMIC DISEASE1.Congenital heart disease2.Renal disease3.GI disease: celiac disease, Crohn disease, ulcerative colitis4.AnemiaD.SYNDROME1.Trisomies2.Noonan disease3.Cornelia de Lange4.Cleidocranial dysplasia5.Lesch-Nyhan disease6.Metatrophic dwarfism

Notes:

Diffuse Osteosclerosis *mnemonic:*"5 MS To PROoF"**M**etastases **M**yelofibrosis **M**astocytosis **M**elorheostosis **M**etabolic:hypervitaminosis D, fluorosis, hypothyroidism, phosphorus poisoning**S**ickle cell anemia **T**uberous sclerosis **P**yknodysostosis, **P**aget disease **R**enal osteodystrophy **O**steopetrosis **F**luorosis

Notes:

Constitutional Sclerosing Bone Disease 1.Engelmann-Camurati disease2.Infantile cortical hyperostosis3.Melorheostosis4.Osteopathia striata5.Osteopetrosis6.Osteopoikilosis7.Pachydermoperiostosis8.Pyknodysostosis9.Van Buchem disease10.Williams syndrome

Notes:

Solitary Osteosclerotic Lesion A.DEVELOPMENTAL1.Bone islandB.VASCULAR1.Old bone infarct2.Aseptic / ischemic / avascular necrosisC.HEALING BONE LESION(a)trauma: callus formation(b)benign tumor: fibrous cortical defect / nonossifying fibroma, brown tumor; bone cyst(c)malignant tumor: lytic metastasis after radiation, chemo-, hormone therapyD.INFECTION / INFLAMMATION(low-grade chronic infection / healing infection) 1.Osteoid osteoma2.Chronic / healed osteomyelitis: bacterial, tuberculous, fungal3.Sclerosing osteomyelitis of Garré4.Granuloma5.Brodie abscessE.BENIGN TUMOR1.Osteoma2.Ossifying fibroma3.Enchondroma / osteochondroma4.OsteoblastomaF.MALIGNANT TUMOR1.Osteoblastic metastasis (prostate, breast)2.Lymphoma3.Sarcoma: osteo-, chondro-, Ewing sarcomaG.OTHERS1.Sclerotic phase of Paget disease2.Fibrous dysplasia

Notes:

Multiple Osteosclerotic Lesions A.FAMILIAL1.Osteopoikilosis2.Enchondromatosis = Ollier disease3.Melorheostosis4.Multiple osteomas: associated with Gardner syndrome5.Osteopetrosis6.Pyknodysostosis7.Osteopathia striata8.Chondrodystrophia calcificans congenita= congenital stippled epiphyses 9.Multiple epiphyseal dysplasia = Fairbank diseaseB.SYSTEMIC DISEASE1.Mastocytosis = urticaria pigmentosa2.Tuberous sclerosis

Notes:

Dense Metaphyseal Bands *mnemonic:*"**H**eavy **C**retins **S**ift **S**currilously through **R**ickety **S**ystems"**H**eavy metal poisoning (lead, bismuth, phosphorus) **C**retinism **S**yphilis, congenital **S**curvy **R**ickets (healed) **S**ystemic illness also:normal variant; methotrexate therapy*mnemonic:*"DENSE LINES"**D**-vitamin intoxication **E**lemental arsenic, bismuth, phosphorus **N**ormal variant **S**ystemic illness **E**strogen to mother during pregnancy **L**eukemia, **L**ead poisoning **I**nfection (TORCH), **I**diopathic hypercalcemia **N**ever forget rickets **E**arly hypothyroidism **S**curvy, **S**ickle cell disease

Notes:

Bone-within-bone Appearance =endosteal new bone formation1.Normal(a)thoracic + lumbar vertebrae (in infants)(b)growth recovery lines (after infancy)2.Infantile cortical hyperostosis (Caffey)3.Sickle cell disease / thalassemia4.Congenital syphilis5.Osteopetrosis / oxalosis6.Radiation7.Acromegaly8.Paget disease*mnemonic:*"BLT PLT RSD RSD"**B**ismuth ingestion **L**ead ingestion **T**horium ingestion **P**etrosis (osteopetrosis) **L**eukemia **T**uberculosis **R**ickets **S**curvy **D** toxicity (vitamin D) **RSD** (reflex sympathetic dystrophy)

Notes:

OSTEOPENIA
=decrease in bone density*Categories:* 1.Osteoporosis = decreased osteoid production2.Osteomalacia = undermineralization of osteoid3.Hyperparathyroidism4.Multiple myeloma / diffuse metastases

Osteoporosis Osteomalacia Localized Osteopenia Bone Marrow Edema Transverse Lucent Metaphyseal Lines Frayed Metaphyses

Notes:

Osteoporosis =reduced bone mass of normal composition secondary to(a) osteoclastic resorption (85%) (trabecular, endosteal, intracortical, subperiosteal)(b) osteocytic resorption (15%)*Incidence:*7% of all women between ages 35-40 years; 1 in 3 women > age 65 years*Etiology:* A.CONGENITAL DISORDERS1.Osteogenesis imperfecta (the only osteoporosis with bending)2.HomocystinuriaB.IDIOPATHIC (bone loss begins earlier + proceeds more rapidly in women)1.Juvenile osteoporosis: <20 years2.Adult osteoporosis: 20-40 years3.Postmenopausal osteoporosis: >50 years (40-50% lower trabecular bone mineral density in elderly than in young women)4.Senile osteoporosis: >60 yearsprogressively decreasing bone density at a rate of 8% in females; 3% in males C.NUTRITIONAL DISTURBANCESscurvy; protein deficiency (malnutrition, nephrosis, chronic liver disease, alcoholism, anorexia nervosa, kwashiorkor, starvation), calcium deficiency D.ENDOCRINOPATHYCushing disease, hypogonadism (Turner syndrome, eunuchoidism), hyperthyroidism, hyperparathyroidism, acromegaly, Addison disease, diabetes mellitus, pregnancy E.RENAL OSTEODYSTROPHYdecrease / same / increase in spinal trabecular bone; rapid loss in appendicular skeleton F.IMMOBILIZATION = disuse osteoporosisG.COLLAGEN DISEASE, RHEUMATOID ARTHRITISH.BONE MARROW REPLACEMENTinfiltration by lymphoma / leukemia, multiple myeloma, diffuse metastases, marrow hyperplasia secondary to hemolytic anemia I.DRUG THERAPYheparin (15,000-30,000 U for >6 months), methotrexate, corticosteroids, vitamin A J.RADIATION THERAPYK.LOCALIZED OSTEOPOROSISSudeck dystrophy, transient osteoporosis of hip, regional migratory osteoporosis of lower extremities ● serum calcium, phosphorus, alkaline phosphatase frequently normal ● hydroxyproline may be elevated during acute stage*Technique:* (1)Single photon absorptiometrymeasures primarily cortical bone of appendicular bones, single-energy I-125 radioisotope sourceSite:distal radius (= wrist bone density), os calcisDose:2-3 mremPrecision:1-3%(2)Dual photon absorptiometryradioactive energy source with two photon peaks; should be reserved for patients <65 years of age because of interference from osteophytosis + vascular calcifications Site:vertebrae, femoral neckDose:5-10 mrem;Precision: 2-4%(3)Quantitative computed tomographyhigh-turnover cancellous bone + low-turnover compact bone can be measured separately Site:vertebrae L1-L3, other sites(a)single energy:300-500 mrem;6-25% precision(b)dual energy:750-800 mrem;5-10% precision(4)Dual energy radiography = quantitative digital radiography = dual energy x-ray absorptiometryx-ray tube produces a two-peak energy spectrumSite:vertebrae, femoral neckDose:<3 mrem;Precision: 1-2% ◊Radiographs are insensitive prior to bone loss of 25-30% ◊Bone scans do NOT show a diffuse increase in activityLocation:axial skeleton (lower dorsal + lumbar spine), proximal humerus, neck of femur, wrist, ribs√ decreased number + thickness of trabeculae√ cortical thinning (endosteal + intracortical resorption)√ juxtaarticular osteopenia with trabecular bone predominance√ delayed fracture healing with poor callus formation(DDx: abundant callus formation in osteogenesis imperfecta + Cushing syndrome)@Spine√ diminished radiographic density√ vertical striations (= marked thinning of transverse trabeculae with relative accentuation of vertical trabeculae along lines of stress)√ prominence of endplates√ "picture framing" (= accentuation of cortical outline with preservation of external dimensions secondary to endosteal + intracortical resorption)√ compression deformities with protrusion of intervertebral disks√ biconcave vertebrae√ Schmorl nodes√ wedging√ decreased height of vertebrae√ absence of osteophytes *Cx:*(1)Fractures at sites rich in labile trabecular bone (eg, vertebrae, wrist) in postmenopausal osteoporosis(2)Fractures at sites containing cortical + trabecular bone (eg, hip) in senile osteoporosis*Rx:*calcitonin, sodium fluoride, diphosphonates, parathyroid hormone supplements, estrogen replacement

Notes:

Osteomalacia =accumulation of excessive amounts of uncalcified osteoid with bone softening + insufficient mineralization of osteoid due to(a)high remodeling rate: excessive osteoid formation + normal / little mineralization(b)low remodeling rate: normal osteoid production + diminished mineralization*Etiology:* (1)dietary deficiency of vitamin D_3 + lack of solar irradiation(2)deficiency of metabolism of vitamin D:-chronic renal tubular disease-chronic administration of phenobarbital (alternate liver pathway)-diphenylhydantoin (interferes with vitamin D action on bowel)(3)decreased absorption of vitamin D:-malabsorption syndromes (most common)-partial gastrectomy (self-restriction of fatty foods)(4)decreased deposition of calcium in bone-diphosphonates (for treatment of Paget disease)*Histo:*excess of osteoid seams + decreased appositional rate ● bone pain / tenderness; muscular weakness ● serum calcium slightly low / normal ● decreased serum phosphorus ● elevated serum alkaline phosphatase√ uniform osteopenia√ fuzzy indistinct trabecular detail of endosteal surface√ thin cortices of long bone√ coarsened frayed trabeculae decreased in number + size√ bone deformity from softening: hourglass thorax, bowing of long bones, buckled / compressed pelvis√ increased incidence of fractures, biconcave vertebral bodies√ mottled skull√ pseudofractures

Notes:

Localized Osteopenia 1.Disuse atrophy*Etiology:*local immobilization secondary to(a)fracture (more pronounced distal to fracture site)(b)neural paralysis(c)muscular paralysis2.Reflex sympathetic dystrophy = Sudeck dystrophy3.Regional migratory osteoporosis, transientosteoporosis of hip4.Osteolytic tumor5.Lytic phase of Paget disease6.Inflammation: rheumatoid arthritis, osteomyelitis, tuberculosis7.Early phase of bone infarct and hemorrhage8.Burns + frostbite

Notes:

Bone Marrow Edema =hypointensity on T1WI + hyperintensity on T2WI1.Transient osteoporosis of hip2.Osteonecrosis = early stage of AVN3.Trauma(a)"bone bruise"(b)radiographically occult fracture in elderly women4.Infection = osteomyelitis5.Infiltrative neoplasm

Notes:

Transverse Lucent Metaphyseal Lines *mnemonic:* "LINING"**L**eukemia **I**llness, systemic (rickets, scurvy) **N**ormal variant **I**nfection, transplacental (congenital syphilis) **N**euroblastoma metastases **G**rowth lines

Notes:

Frayed Metaphyses *mnemonic:*"CHARMS"**C**ongenital infections (rubella, syphilis) **H**ypophosphatasia **A**chondroplasia **R**ickets **M**etaphyseal dysostosis **S**curvy

Notes:

PERIOSTEAL REACTION

1.Trauma, hemophilia2.Infection3.Inflammatory: arthritis4.Neoplasm5.Congenital:physiologic in newborn6.Metabolic:hypertrophic osteoarthropathy, thyroid acropachy, hypervitaminosis A7.Vascular:venous stasis

Solid Periosteal Reaction Interrupted Periosteal Reaction Symmetric Periosteal Reaction In Adulthood Periosteal Reaction In Childhood

Notes:

Solid Periosteal Reaction =reaction to periosteal irritant√ even + uniform thickness >1 mm√ persistent + unchanged for weeks*Patterns:* (a)thin: eosinophilic granuloma, osteoid osteoma(b)dense undulating: vascular disease(c)thin undulating: pulmonary osteoarthropathy(d)dense elliptical: osteoid osteoma; long-standing malignant disease (with destruction)(e)cloaking: storage disease; chronic infection

Notes:

Interrupted Periosteal Reaction =pleomorphic, rapidly progressing process undergoing constant change(a)lamellated = "onion skin": acute osteomyelitis; malignant tumor (osteosarcoma, Ewing sarcoma)(b)perpendicular = "sunburst": osteosarcoma; Ewing sarcoma; chondrosarcoma; fibrosarcoma; leukemia; metastasis; acute osteomyelitis(c)amorphous: malignancy (deposits may represent extension of tumor / periosteal response); osteosarcoma(d)Codman triangle: hemorrhage; malignancy (osteosarcoma, Ewing sarcoma); acute osteomyelitis; fracture

Notes:

Symmetric Periosteal Reaction In Adulthood 1.Vascular insufficiency (lower extremity)2.Hypertrophic osteoarthropathy3.Pachydermoperiostosis4.Thyroid acropachy5.Fluorosis6.Rheumatoid arthritis7.Psoriatic arthritis8.Reiter syndrome9.Idiopathic-degenerative

Notes:

Periosteal Reaction In Childhood (a)benign1.Physiologic (up to 35%): symmetric involvement of diaphyses during first 1-6 months of life2.Battered child syndrome3Infantile cortical hyperostosis <6 months of age4.Hypervitaminosis A5.Scurvy6.Osteogenesis imperfecta7.Congenital syphilis(b)malignant1.Multicentric osteosarcoma2.Metastases from neuroblastoma + retinoblastoma3.Acute leukemia*mnemonic:*"PERIOSTEAL SOCKS"**P**hysiologic, **P**rostaglandin **E**osinophilic granuloma **R**ickets **I**nfantile cortical hyperostosis **O**steomyelitis **S**curvy **T**rauma **E**wing sarcoma **A**-hypervitaminosis **L**eukemia + neuroblastoma **S**yphilis **O**steosarcoma **C**hild abuse **K**inky hair syndrome **S**ickle cell disease

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Notes:

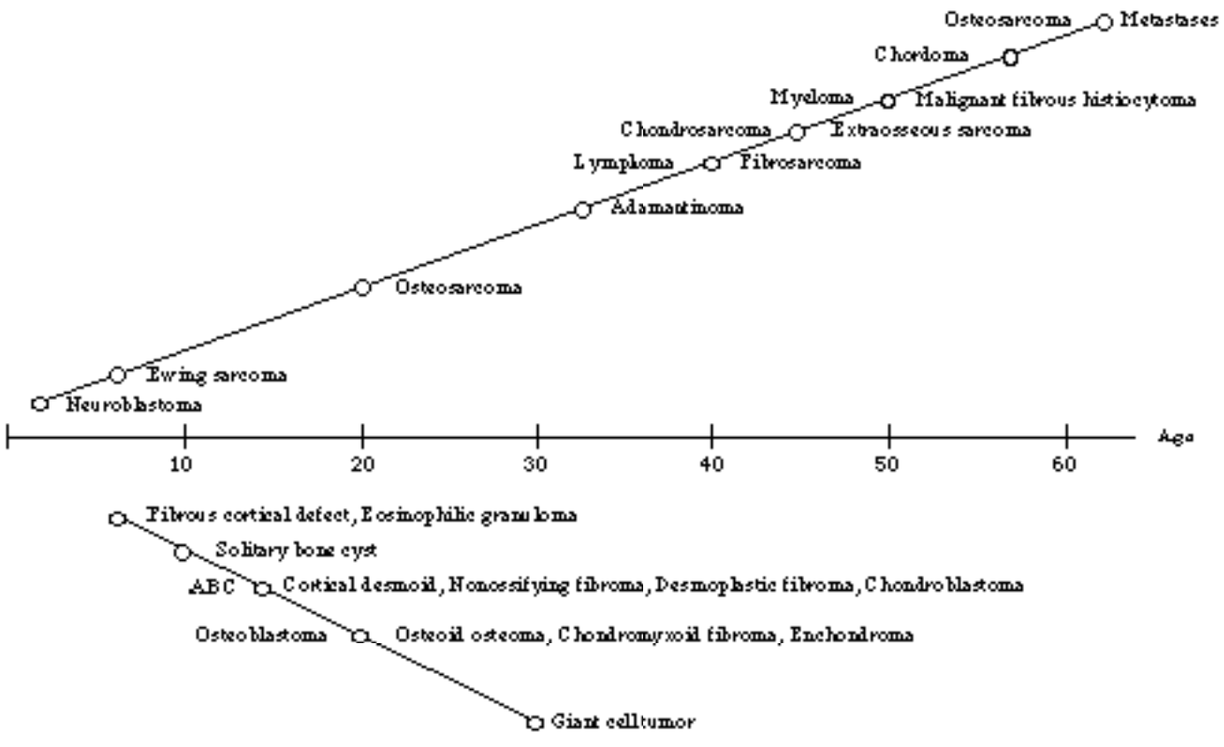
BONE TUMOR

Assessment of aggressiveness A.BENIGN1.Diagnosis certain: no further work-up necessary2.Asymptomatic lesion with highly probable diagnosis may be followed clinically3.Symptomatic lesion with highly probable diagnosis may be treated without further work-upB.CONFUSING LESIONnot clearly categorized as benign or malignant; needs staging work-up C.MALIGNANTneeds staging work-up *Staging work-up:* Bone scan:identifies polyostotic lesions (eg, multiple myeloma, metastatic disease, primary osteosarcoma with bone-forming metastases, histiocytosis, Paget disease)Chest CT:identifies metastatic deposits + changes further work-up and therapy*Local staging with MR imaging:* (1)Margins: encapsulated / infiltrating(2)Compartment: intra- / extracompartmental(3)Intraosseous extent + skip lesions(4)Soft-tissue extent (DDx: hematoma, edema)(5)Joint involvement(6)Neurovascular involvementLocal assessment with CT:matrix / rim calcifications

Age Incidence of Malignant Bone Tumors Tumor Matrix of Bone Tumors Pattern of Bone Destruction Tumor Position in Transverse Plane Tumor Position in Longitudinal Plane Tumorlike Conditions

Notes:

Age Incidence of Malignant Bone Tumors ◊80% of bone tumors are correctly determined on the basis of age alone! Age (years)Tumor----------0.1Neuroblastoma0.1-10Ewing tumor in tubular bones (diaphysis)10 -30Osteosarcoma (metaphysis); Ewing tumor in flat bones30 -40Reticulum cell sarcoma (similar histology to Ewing tumor); fibrosarcoma; malignant giant cell tumor (similar histology to fibrosarcoma); parosteal sarcoma; lymphoma>40Metastatic carcinoma; multiple myeloma; chondrosarcoma



Average Age for Occurrence of Benign and Malignant Bone Tumors

SARCOMAS BY AGE: *mnemonic:*"**E**very **O**ther **R**unner **F**eels **C**rampy **P**ain **O**n **M**oving"**E**wing sarcoma 0 -10 years**O**steogenic sarcoma10-30 years**R**eticulum cell sarcoma20-40 years**F**ibrosarcoma20-40 years**C**hondrosarcoma40-50 years**P**arosteal sarcoma40-50 years**O**steosarcoma60-70 years**M**etastases60-70 years ROUND CELL TUMORS: arise in mid shaft; osteolytic; reactive new bone formation; no tumor new bone *mnemonic:*"LEMON"**L**eukemia, **L**ymphoma **E**wing sarcoma, **E**osinophilic granuloma **M**ultiple myeloma **O**steomyelitis **N**euroblastoma MALIGNANCY WITH SOFT-TISSUE INVOLVEMENT *mnemonic:*"**M**y **M**other **E**ats **C**hocolate **F**udge **O**ften"**M**etastasis **M**yeloma **E**wing sarcoma **C**hondrosarcoma **F**ibrosarcoma **O**steosarcoma

Notes:

Tumor Matrix of Bone Tumors ***Cartilage-forming Bone Tumors*** A.BENIGN1.Enchondroma2.Parosteal chondroma3.Chondroblastoma4.Chondromyxoid fibroma5.OsteochondromaB.MALIGNANT1.Chondrosarcoma√ centrally located ringlike / flocculent / flecklike radiodensities ***Bone-forming Tumors*** A.BENIGN1.Osteoma2.Osteoid osteoma3.Osteoblastoma4.Ossifying fibromaB.MALIGNANT1.Osteogenic sarcoma √ inhomogeneous / homogeneous radiodense collections of variable size + extent ***Fibrous Connective Tissue Tumors*** A.BENIGN FIBROUS BONE LESIONS(a)cortical1.Benign cortical defect2.Avulsion cortical irregularity(b)medullary1.Herniation pit2.Nonossifying fibroma3.Ossifying fibroma4.Congenital generalized fibromatosis(c)corticomedullary1.Nonossifying fibroma2.Ossifying fibroma3.Fibrous dysplasia4.Cherubism5.Desmoplastic fibroma6.FibromyxomaB.MALIGNANT1.Fibrosarcoma ***Tumors of Histiocytic Origin*** A.LOCALLY AGGRESSIVE1.Giant cell tumor2.Benign fibrous histiocytomaB.MALIGNANT1.Malignant fibrous histiocytoma ***Tumors of Fatty Tissue Origin*** A.BENIGN1.Intraosseous lipoma2.Parosteal lipomaB.MALIGNANT1.Intraosseous liposarcoma◊Lipomas follow the signal intensity of subcutaneous fat in all sequences! ***Tumors of Vascular Origin*** <1% of all bone tumors A.BENIGN1.Hemangioma2.Glomus tumor3.Lymphangioma 4.Cystic angiomatosis5.HemangiopericytomaB.MALIGNANT1.Malignant hemangiopericytoma2.Angiosarcoma = hemangioendotheliomaMetastatic sites:lung, brain, lymph nodes, other bones ***Tumors of Neural Origin*** A.BENIGN1.Solitary neurofibroma2.NeurilemomaB.MALIGNANT1.Neurogenic sarcoma = malignant schwannoma

Notes:

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Pattern of Bone Destruction A.GEOGRAPHIC BONE DESTRUCTIONIndicative of slow-growing usually benign tumor √ well-defined smooth / irregular margin√ short zone of transitionB.MOTH-EATEN BONE DESTRUCTIONIndicative of more rapid growth as in malignant bone tumor / osteomyelitis √ less well defined / demarcated lesional margin√ longer zone of transition*mnemonic:*"H LEMMON"**H**istiocytosis X **L**ymphoma **E**wing sarcoma **M**etastasis **M**ultiple myeloma **O**steomyelitis **N**euroblastoma C.PERMEATIVE BONE DESTRUCTIONAggressive bone lesion with rapid growth potential (eg, Ewing sarcoma) √ poorly demarcated lesion imperceptibly merging with uninvolved bone√ long zone of transitionD.SIZE OF LESIONPrimary malignant tumors are larger than benign tumors E.ELONGATED LESION√ greatest lesional diameter is >1 1/2 times the least diameterEwing sarcoma, reticulum cell sarcoma, chondrosarcoma, angiosarcoma

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Notes:

Tumor Position in Transverse Plane A.CENTRAL MEDULLARY LESION1.Enchondroma2.Solitary bone cystB.ECCENTRIC MEDULLARY LESION1.Giant cell tumor2.Osteogenic sarcoma, chondrosarcoma, fibrosarcoma3.Chondromyxoid fibromaC.CORTICAL LESION1.Nonossifying fibroma2.Osteoid osteomaD.PAROSTEAL / JUXTACORTICAL LESION1.Juxtacortical chondroma2.Osteochondroma3.Parosteal osteogenic sarcoma

Notes:

Tumor Position in Longitudinal Plane A.EPIPHYSEAL LESION1.Chondroblastoma2.Intraosseous ganglion3.Giant cell tumor (originating in metaphysis)*mnemonic:*"CAGGIE"**C**hondroblastoma **A**neurysmal bone cyst **G**iant cell tumor **G**eode **I**nfection **E**osinophilic granuloma [after 40 years of age throw out "CEA" and insert metastases / myeloma] B.METAPHYSEAL LESION1.Nonossifying fibroma2.Chondromyxoid fibroma3.Solitary bone cyst4.Osteochondroma5.Brodie abscess6.Osteogenic sarcoma, chondrosarcomaC.DIAPHYSEAL LESION1.Round cell tumor (eg, Ewing sarcoma)2.Nonossifying fibroma3.Solitary bone cyst4.Aneurysmal bone cyst5.Enchondroma6.Osteoblastoma7.Fibrous dysplasia*mnemonic:*"FEMALE"**F**ibrous dysplasia **E**osinophilic granuloma **M**etastasis **A**damantinoma **L**eukemia, **L**ymphoma **E**wing sarcoma

Notes:

Tumorlike Conditions 1.Solitary bone cyst2.Juxta-articular ("synovial") cyst3.Aneurysmal bone cyst4.Nonossifying fibroma; cortical defect; cortical desmoid5.Eosinophilic granuloma6.Reparative giant cell granuloma7.Fibrous dysplasia (monostotic; polyostotic)8.Myositis ossificans9."Brown tumor" of hyperparathyroidism10.Massive osteolysis

Notes:

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Bubbly Bone Lesion *mnemonic:*"FOG MACHINES"**F**ibrous dysplasia, **F**ibrous cortical defect **O**steoblastoma **G**iant cell tumor **M**yeloma (plasmacytoma), **M**etastases from kidney, thyroid, breast **A**neurysmal bone cyst / **A**ngioma **C**hondromyxoid fibroma, **C**hondroblastoma **H**istiocytosis X, **H**yperparathyroid brown tumor, **H**emophilia **I**nfection (Brodie abscess, Echinococcus, coccidioidomycosis) **N**onossifying fibroma **E**nchondroma, **E**pithelial inclusion cyst **S**imple unilocular bone cyst ***Infectious Bubbly Lesion*** 1.Brodie abscess (Staph. aureus)2.Coccidioidomycosis3.Echinococcus4.Atypical mycobacterium5.Cystic tuberculosis ***Blowout Lesion*** A.METASTASESCarcinoma of thyroid, kidney, breast B.PRIMARY BONE TUMOR1.Fibrosarcoma2.Multiple myeloma (sometimes)3.Aneurysmal bone cyst4.Hemophilic pseudotumor

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Notes:

Nonexpansile Unilocular Well-demarcated Bone Defect 1. Fibrous cortical defect2.Nonossifying fibroma3.Simple unicameral bone cyst4.Giant cell tumor5.Brown tumor of HPT6.Eosinophilic granuloma7.Enchondroma8.Epidermoid inclusion cyst9.Posttraumatic / degenerative cyst10.Pseudotumor of hemophilia11.Intraosseous ganglion12.Histiocytoma13.Arthritic lesion14.Endosteal pigmented villonodular synovitis15.Fibrous dysplasia16.Infectious lesion

Notes:

Nonexpansile Multilocular Well-demarcated Bone Defect 1.Aneurysmal bone cyst2.Giant cell tumor3.Fibrous dysplasia4.Simple bone cyst

Notes:

Expansile Unilocular Well-demarcated Osteolysis 1.Simple unicameral bone cyst2.Enchondroma3.Aneurysmal bone cyst4.Juxtacortical chondroma5.Nonossifying fibroma6.Eosinophilic granuloma7.Brown tumor of HPT

Notes:

Poorly Demarcated Osteolytic Lesion Without Periosteal Reaction A.NONEXPANSILE1.Metastases from any primary neoplasm2.Multiple myeloma3.HemangiomaB.EXPANSILE1.Chondrosarcoma2.Giant cell tumor3.Metastasis from kidney / thyroid

Notes:

Poorly Demarcated Osteolytic Lesion With Periosteal Reaction 1.Osteomyelitis2.Ewing sarcoma3.Osteosarcoma

Notes:

Mixed Sclerotic And Lytic Lesion A.WITH SEQUESTRUM: osteomyelitisB.WITHOUT SEQUESTRUM:1.Osteomyelitis2.Tuberculosis3.Ewing sarcoma4.Metastasis5.Osteosarcoma

Notes:

Trabeculated Bone Lesion 1.Giant cell tumor: delicate thin trabeculae2.Chondromyxoid fibroma: coarse thick trabeculae3.Nonossifying fibroma: loculated4.Aneurysmal bone cyst: delicate, horizontally oriented trabeculae5.Hemangioma: striated radiating trabeculae

Notes:

Lytic Bone Lesion Surrounded By Marked Sclerosis *mnemonic:*"BOOST"**B**rodie abscess **O**steoblastoma **O**steoid osteoma **S**tress fracture **T**uberculosis

Notes:

Multiple Lytic Lesions *mnemonic:*"FEEMHI"**F**ibrous dysplasia **E**nchondromas **E**osinophilic granuloma **M**etastases, **M**ultiple myeloma **H**yperparathyroidism (brown tumors), **H**emangiomas **I**nfection

Notes:

Lytic Bone Lesion In Patient <30 Years Of Age *mnemonic:*"CAINES"**C**hondroblastoma **A**neurysmal bone cyst **I**nfection **N**onossifying fibroma **E**osinophilic granuloma **S**olitary bone cyst

Notes:

Lytic Bone Lesion On Both Sides Of Joint *mnemonic:*"SAC"**S**ynovioma **A**ngioma **C**hondroid lesion

Notes:

DWARFISM

Classification: (1)OSTEOCHONDRODYSPLASIA=abnormalities of cartilage / bone growth and development(a)identifiable at birth:-usually lethal: achondrogenesis, fibrochondrogenesis, thanatophoric dysplasia, short rib syndrome-usually nonlethal: chondrodysplasia punctata, camptomelic dysplasia, achondroplasia, diastrophic dysplasia, chondroectodermal dysplasia, Jeune syndrome, spondyloepiphyseal dysplasia congenita, mesomelic dysplasia, cleidocranial dysplasia, oto-palato-digital syndrome(b)identifiable in later life: hypochondroplasia, dyschondrosteosis, spondylometaphyseal dysplasia, acromicric dysplasia(c)abnormal bone density: osteopetrosis, pyknodysostosis, Melnick-Needles syndrome(2)DYSOSTOSIS=malformation of individual bones singly / in combination(a)with cranial + facial involvement:craniosynostosis, craniofacial dysostosis (Crouzon), acrocephalosyndactyly, acrocephalopolysyndactyly, branchial arch syndromes (Treacher-Collins, Franceschetti, acrofacial dysostosis, oculo-auriculo-vertebral dysostosis, hemifacial microsomia, oculo-mandibulo-facial syndrome (b)with predominant axial involvement:vertebral segmentation defects (Klippel-Feil), Sprengel anomaly, spondylocostal dysostosis, oculovertebral syndrome (c)with predominant involvement of extremities:acheiria (= absence of hands), apodia (= absence of feet), polydactyly, syndactyly, camptodactyly, Rubinstein-Taybi syndrome, pancytopenia-dysmelia syndrome (Fanconi), Blackfan-Diamond anemia with thumb anomaly, thrombocytopenia-radial aplasia syndrome, cardiomelic syndromes (Holt-Oram), focal femoral deficiency, multiple synostoses (3)IDIOPATHIC OSTEOLYSIS=disorders associated with multifocal resorption of bone(4)CHROMOSOMAL ABERRATION(5)PRIMARY METABOLIC DISORDER(a)calcium / phosphorus: hypophosphatasia(b)complex carbohydrates: mucopolysaccharidosis *Terminology:* Micromelia=shortening involves entire limb(eg, humerus, radius + ulna, hand)Rhizomelia=shortening involves proximal segment (eg, humerus)Mesomelia=shortening involves intermediate segment (eg, radius + ulna)Acromelia=shortening involves distal segment(eg, hand)

Micromelic Dwarfism Acromelic Dwarfism Rhizomelic Dwarfism Osteochondrodysplasia Lethal Bone Dysplasia Nonlethal Dwarfism Late-onset Dwarfism
Hypomineralization In Fetus Large Head In Fetus Narrow Chest In Fetus Platyspondyly Bowed Long Bones In Fetus Bone Fractures In Fetus

Notes:

Micromelic Dwarfism =disproportionate shortening of entire legA.Mild micromelic dwarfism1.Jeune syndrome2.Ellis-van Creveld syndrome= chondroectodermal dysplasia 3.Diastrophic dwarfismB.Mild bowed micromelic dwarfism1.Camptomelic dysplasia2.Osteogenesis imperfecta, type IIIC.Severe micromelic dwarfism1.Thanatophoric dysplasia2.Osteogenesis imperfecta, type II3.Homozygous achondroplasia4.Hypophosphatasia5.Short-rib polydactyly syndrome6.Fibrochondrogenesis

Notes:

Acromelic Dwarfism =distal shortening (hands, feet)1.Asphyxiating thoracic dysplasia

Notes:

Rhizomelic Dwarfism =shortening of proximal segments (humerus, femur)*mnemonic:*"MA CAT"**M**etatrophic dwarfism **A**chondrogenesis (most severe shortening) **C**hondrodysplasia punctata (autosomal recessive) Thanatophoric dysplasia **A**chondroplasia, heterozygous

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Notes:

Osteochondrodysplasia A.Failure of(a)articular cartilage: spondyloepiphyseal dysplasia(b)ossification center: multiple epiphyseal dysplasia(c)proliferating cartilage: achondroplasia(d)spongiosa formation: hypophosphatasia(e)spongiosa absorption: osteopetrosis(f)periosteal bone: osteogenesis imperfecta(g)endosteal bone: idiopathic osteoporosisB.Excess of(a)articular cartilage: dysplasia epiphysealis hemimelica(b)hypertrophic cartilage: enchondromatosis(c)spongiosa: multiple exostosis(d)periosteal bone: progressive diaphyseal dysplasia(e)endosteal bone: hyperphosphatemia

Notes:

Lethal Bone Dysplasia in order of frequency 1.Thanatophoric dysplasia2.Osteogenesis imperfecta type II3.Achondrogenesis type I + II4.Jeune syndrome (may be nonlethal)5.Hypophosphatasia, congenital lethal form6.Chondroectodermal dysplasia (usually nonlethal)7.Chondrodysplasia punctata, rhizomelic type8.Camptomelic dysplasia9.Short-rib polydactyly syndrome10.Homozygous achondroplasia◊Lethal short-limbed dysplasias typically are manifest on sonograms before 24 weeks MA!

Notes:

Nonlethal Dwarfism 1.Achondroplasia (heterozygous)2.Asphyxiating thoracic dysplasia3.Chondroectodermal dysplasia4.Chondrodysplasia punctata5.Spondyloepiphyseal dysplasia (congenital)6.Diastrophic dwarfism7.Metatrophic dwarfism8.Hypochondroplasia

Notes:

Late-onset Dwarfism 1.Spondyloepiphyseal dysplasia tarda2.Multiple epiphyseal dysplasia3.Pseudoachondroplasia4.Metaphyseal chondrodysplasia5.Dyschondrosteosis6.Cleidocranial dysostosis7.Progressive diaphyseal dysplasia

Notes:

Hypomineralization In Fetus A.DIFFUSE1.Osteogenesis imperfecta2.HypophosphatasiaB.SPINE1.Achondrogenesis

Notes:

Large Head In Fetus 1.Achondroplasia2.Thanatophoric dysplasia

Notes:

Narrow Chest In Fetus 1.Short-rib polydactyly syndrome2.Asphyxiating thoracic dysplasia3.Chondroectodermal dysplasia4.Camptomelic dysplasia5.Thanatophoric dwarfism6.Homozygous achondroplasia7.Achondrogenesis8.Hypophosphatasia

Notes:

Platyspondyly 1.Thanatophoric dysplasia2.Osteogenesis imperfecta type II3.Achondroplasia

Notes:

Bowed Long Bones In Fetus 1.Campomelic syndrome2.Osteogenesis imperfecta3.Thanatophoric dysplasia4.Hypophosphatasia

Notes:

Bone Fractures In Fetus 1.Osteogenesis imperfecta2.Hypophosphatasia3.Achondrogenesis

Notes:

LIMB REDUCTION ANOMALIES
Amelia=absence of limbHemimelia=absence of distal partsPhocomelia=proximal reduction with distal parts attached to trunk

Aplasia / Hypoplasia of Radius Pubic Bone Maldevelopment

Notes:

Aplasia / Hypoplasia of Radius *mnemonic:*"**T**he **F**urry **C**at **H**it **M**y **D**og"**T**hrombocytopenia-absent radius syndrome **F**anconi anemia **C**ornelia de Lange syndrome **H**olt-Oram syndrome **M**yositis ossificans progressiva (thumb only) **D**iastrophic dwarfism ("hitchhikers thumb")

Notes:

Pubic Bone Maldevelopment *mnemonic:*"CHIEF"**C**leidocranial dysostosis **H**ypospadia, epispadia **I**diopathic **E**xstrophy of bladder **F** for syringomyelia

Notes:

Bone Overdevelopment 1.Marfan syndrome2.Klippel-Trénaunay syndrome3.Nerve territory-oriented macrodactyly(a)Macrodystrophia lipomatosa(b)Fibrolipomatous hamartoma with macrodactyly

Notes:

Erlenmeyer Flask Deformity =expansion of distal end of long bones, usually femur1.Gaucher disease, Niemann-Pick disease2.Rickets3.Anemia, eg, thalassemia4.Fibrous dysplasia5.Osteopetrosis6.Heavy metal poisoning7.Metaphyseal dysplasia = Pyle disease8.Down syndrome9.Achondroplasia10.Rheumatoid arthritis11.Hypophosphatasia*mnemonic:*"TOP DOG"**T**halassemia **O**steopetrosis **P**yle disease **D**iaphyseal aclasis **O**llier disease **G**aucher disease

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Notes:

Approach to Arthritis *mnemonic:*"ABCDES"**A**lignment **B**one mineralization **C**artilage loss **D**istribution **E**rosion **S**oft tissues

Notes:

Signs of Arthritis *Prevalence of arthritis:*15% of population in USAConventional x-ray: √ narrowing of radiologic joint space(a)uniform = inflammatory arthritis(b)nonuniform = degenerative arthritis√ evidence of disease on both sides of joint:√ osteopenia√ subchondral sclerosis√ erosion√ subchondral cyst formation√ malalignment√ joint effusion√ joint bodiesNUC: √ increase in regional blood flow (active disease)√ distribution of diseaseMR: √ irregularity + narrowing of articular cartilage√ Gd-DTPA enhancement of synovium (active disease)

Notes:

Classification of Arthritides A.SEPTIC ARTHRITIS1.Tuberculous2.Pyogenic3.Lyme arthritis4.Fungal arthritis: Candida, Coccidioides immitis, Blastomyces dermatitidis, Histoplasma capsulatum, Sporothrix schenckii, Cryptococcus neoformans, Aspergillus fumigatusB.COLLAGEN / COLLAGEN-LIKE DISEASE1.Rheumatoid arthritis2.Ankylosing spondylitis3.Psoriatic arthritis4.Rheumatic fever5.SarcoidosisC.BIOCHEMICAL ARTHRITIS1.Gout2.Chondrocalcinosis3.Ochronosis4.Hemophilic arthritisD.DEGENERATIVE JOINT DISEASE = OsteoarthritisE.TRAUMATIC1.Secondary osteoarthritis2.Neurotrophic arthritis3.Pigmented villonodular synovitisF.ENTEROPATHIC ARTHROPATHY(a)INFLAMMATORY BOWEL DISEASE1.Ulcerative colitis (in 10-20%)2.Crohn disease (in 5%): peripheral arthritis increases with colonic disease3.Whipple disease (in 60-90% transient intermittent polyarthritis: sacroiliitis, spondylitis)Resection of diseased bowel is associated with regression of arthritic symptomatology!(b)INFECTIOUS BOWEL DISEASEInfectious agents: Salmonella, Shigella, Yersinia (c)after intestinal bypass surgery SPONDYLARTHRITIS + positive HLA-B 27 HISTOCOMPATIBILITY COMPLEX 1.Ankylosing spondylitis95%2.Reiter disease80%3.Arthropathy of inflammatory bowel disease75%4.Psoriatic spondylitis70%5.Normal population10%

Notes:

Synovial Disease with Decreased Signal Intensity = hemosiderin deposition 1.Pigmented villonodular synovitis2.Rheumatoid arthritis3.Hemophilia

Notes:

Chondrocalcinosis *mnemonic:* "WHIP A DOG"**W**ilson disease **H**emochromatosis, **H**emophilia, **H**ypothyroidism, 1° **H**yperparathyroidism (15%), **H**ypophosphatasia, Familial **H**ypomagnesemia **I**diopathic (aging) **P**seudogout (CPPD) **A**rthritis (rheumatoid, postinfectious, traumatic, degenerative), **A**myloidosis, **A**cromegaly **D**iabetes mellitus **O**chronosis **G**out *mnemonic:*"3 Cs"**C**rystalsCPPD,sodium urate (gout)Cationscalcium (any cause of hypercalcemia), copper, iron**C**artilage degenerationosteoarthritis, acromegaly, ochronosis

Notes:

Subchondral Cyst =SYNOVIAL CYST = SUBARTICULAR PSEUDOCYST= NECROTIC PSEUDOCYST = GEODES *Etiology:*bone necrosis allows pressure-induced intrusion of synovial fluid into subchondral bone; in conditions with synovial inflammation*Cause:*(1) Osteoarthritis (2) Rheumatoid arthritis(3) Osteonecrosis (4) CPPD size of cyst usually 2-35 mm may be large + expansile (especially in CPPD)*DDx:*(1)Giant cell tumor(2)Pigmented villonodular synovitis(3)Metastasis(4)Intraosseous ganglion(5)Hemophilia

Notes:

Loose Intraarticular Bodies 1.Osteochondrosis dissecans2.Synovial osteochondromatosis3.Chip fracture from trauma4.Severe degenerative joint disease5.Neuropathic arthropathy

Notes:

Premature Osteoarthritis *mnemonic:*"COME CHAT"**C**alcium pyrophosphate dihydrate arthropathy **O**chronosis **M**arfan syndrome **E**piphyseal dysplasia **C**harcot joint = neuroarthropathy **H**emophilic arthropathy **A**cromegaly **T**rauma

Notes:

Arthritis With Periostitis 1.Juvenile rheumatoid arthritis2.Psoriatic arthritis3.Reiter syndrome4.Infectious arthritis

Notes:

Arthritis With Demineralization *mnemonic:*"HORSE"**H**emophilia **O**steomyelitis **R**heumatoid arthritis, **R**eiter disease **S**cleroderma **E**rythematosus, systemic lupus

Notes:

Arthritis Without Demineralization 1.Gout2.Neuropathic arthropathy3.Psoriasis4.Reiter disease5.Pigmented villonodular synovitis*mnemonic:*"PONGS"**P**soriatic arthritis **O**steoarthritis **N**europathic joint **G**out **S**arcoidosis

Notes:

Articular Disorders Of The Hand + Wrist 1.Osteoarthritis = degenerative joint disease=abnormal stress with minor + major traumatic episodes*Target areas:*DIP, PIP, 1st CMC, trapezioscaphoid√ sclerosis + osteophytes2.Erosive osteoarthritis = inflammatory osteoarthritis*Age:*predominantly middle-aged / postmenopausal women ● acute inflammatory episodes*Target areas:*DIP, PIP, 1st CMC, trapezioscaphoid√ subchondral "gull wing" erosions√ rare ankylosis3.Psoriatic arthritis=rheumatoid variant / seronegative spondyloarthropathy; peripheral manifestation in monarthritis / asymmetric oligoarthritis / symmetric polyarthritis*Target areas:*all hand + wrist joints (commonly distal)√ "mouse ears" marginal erosions√ new bone formation4.Rheumatoid arthritis=synovial proliferative granulation tissue = pannus*Target areas:*PIP, MCP, all wrist joints, ulnar styloid√ marginal poorly defined erosions√ joint deformities5.Gouty arthritis ● monosodium urate crystals in synovial fluid ● asymptomatic periods from months to years*Target areas:*commonly CMC + all hand joints√ development of chronic tophaceous gout√ well-defined erosions with overhanging edge (often periarticular)√ joint space narrowing6.Calcium pyrophosphate dihydrate crystal deposition disease = CPPD*Target areas:*MCP, radiocarpal√ chondrocalcinosis√ "degenerative changes" in unusual locations√ no erosions7.SLE=myositis, symmetric polyarthritis, deforming nonerosive arthropathy, osteonecrosis*Target areas:*PIP, MCP√ reversible deformities8.Scleroderma = progressive systemic sclerosis (PSS)*Target areas:*DIP, PIP, 1st CMC√ tuft resorption√ soft-tissue calcifications

Notes:

Arthritis Involving Distal Interphalangeal Joints *mnemonic*:"POEM"**P**soriatic arthritis **O**steoarthritis **E**rosive osteoarthritis **M**ulticentric reticulohistiocytosis

Notes:

Ankylosis Of Interphalangeal Joints *mnemonic:*"S - Lesions"1.P**s**oriatic arthritis2.Ankylo**s**ing spondylitis3.Ero**s**ive osteoarthritis4.**S**till disease

Notes:

Sacroiliitis *Anatomy:* only anterior inferior aspect of sacroiliac apposition is covered with cartilage (1 mm thick hyalin cartilage on iliac side, 3-5 mm thick fibrous cartilage on sacral side); 2-5 mm normal joint width *Positioning:*Ferguson view = AP projection with 23° angulation toward head A.BILATERAL SYMMETRICAL1.Ankylosing spondylitis√ small regular erosion = loss of definition of white cortical line on iliac side√ ankylosis√ ossification of intraosseous ligaments2.Rheumatoid arthritis (in late stages)√ joint space narrowing without reparation√ osteoporosis√ ankylosis may occur3.Deposition arthropathy: gout, CPPD, ochronosis, acromegaly√ slow loss of cartilage√ subchondral reparative bone + osteophytes4.Enteropathic arthropathy:B.BILATERAL ASYMMETRICAL1.Psoriatic arthritis√ large + extensive erosive + reparative process√ occasional ankylosis2.Reiter syndrome3.Juvenile rheumatoid arthritisC.UNILATERAL1.Infection2.Osteoarthritis from abnormal mechanical stress√ irregular narrowing of joint space with subchondral bone repair√ osteophytes at anterosuperior / -inferior aspect of joint (may resemble ankylosis) *DDx:*Hyperparathyroidism√ subchondral bone resorption on iliac side resembling erosion + widening of joint

Notes:

Sacroiliac Joint Widening *mnemonic:*"CRAP TRAP"**C**olitis **R**heumatoid arthritis **A**bscess (infection) **P**arathyroid disease **T**rauma **R**eiter syndrome **A**nkylosing spondylitis **P**soriasis

Notes:

Sacroiliac Joint Fusion *mnemonic:*"CARPI" **C**olitic spondylitis **A**nkylosing spondylitis **R**eiter syndrome **P**soriatic arthritis **I**nfection (TB)

Notes:

Widened Symphysis Pubis *mnemonic:*"EPOCH"**E**xstrophy of the bladder **P**rune belly syndrome **O**steogenesis imperfecta **C**leidocranial dysostosis **H**ypothyroidism

Notes:

Arthritis Of Interphalangeal Joint Of Great Toe 1.Psoriatic arthritis2.Reiter disease3.Gout4.Degenerative joint disease

Notes:

Enthesopathy Enthesis = osseous attachment of tendon composed of 4 zones, ie, tendon itself + unmineralized fibrocartilage + mineralized fibrocartilage + bone
Cause: 1.Degenerative disorder2.Seronegative arthropathies: ankylosing spondylitis, Reiter disease, psoriatic arthritis3.Diffuse idiopathic skeletal hyperostosis4.Acromegaly5.Rheumatoid arthritis (occasionally)Location:at site of tendon + ligament attachment√ bone proliferation (enthesophyte)√ calcification of tendon + ligament√ erosion

Notes:

Epiphyseal / Apophyseal lesion 1.Chondroblastoma2.Brodie abscess3.Fungal / tuberculous infection4.Langerhans cell histiocytosis5.Osteoid osteoma6.Chondromyxoid fibroma7.Enchondroma8.Bone cyst9.Foreign-body granuloma

Notes:

Stippled Epiphyses 1.Normal variant2.Avascular necrosis3.Hypothyroidism4.Chondrodysplasia punctata5.Multiple epiphyseal dysplasia6.Spondyloepiphyseal dysplasia7.Hypoparathyroidism8.Down syndrome9.Trisomy 1810.Fetal warfarin syndrome11.Homocystinuria (distal radial + ulnar epiphyses = pathognomonic)12.Zellweger cerebrohepatorenal syndrome

Notes:

Epiphyseal Overgrowth 1.Juvenile rheumatoid arthritis2.Hemophilia3.Healed Legg-Perthes disease4.Tuberculous arthritis5.Pyogenic arthritis (chronic)6.Fungal arthritis7.Epiphyseal dysplasia hemimelica8.Fibrous dysplasia of epiphysis9.Winchester syndrome

Notes:

Ring Epiphysis 1.Severe osteoporosis2.Healing rickets3.Scurvy

Notes:

Epiphyseolysis =SLIPPED EPIPHYSIS (zone of maturing hypertrophic cartilage affected, not zone of proliferation)1.Idiopathic / juvenile epiphyseolysis*Age:*12-15 years (? puberty-related hormonal dysregulation) ● adiposogenital type; tall stature2.Renal osteodystrophy3.Hyperparathyroidism in chronic renal disease4.Hypothyroidism5.Radiotherapy

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Notes:

Childhood Fractures 1.Greenstick fracture=incomplete fracture of soft growing bone with intact periosteum2.Bowing fracture3.Traumatic epiphyseolysis4.Battered child syndrome5.Epiphyseal plate injury

Notes:

Pseudarthrosis In Long Bones 1.Nonunion of fracture2.Fibrous dysplasia3.Neurofibromatosis4.Osteogenesis imperfecta5.Congenital: clavicular pseudarthrosis

Notes:

Exuberant callus Exuberant Callus Formation 1.Steroid therapy / Cushing syndrome2.Neuropathic arthropathy3.Osteogenesis imperfecta4.Congenital insensitivity to pain5.Paralysis6.Renal osteodystrophy7.Multiple myeloma8.Battered child syndrome

Notes:

Rib Lesions A.BENIGN RIB TUMOR1.Fibrous dysplasia (most common benign lesion)√ predominantly posterior location2.Eosinophilic granuloma3.Benign cortical defect4.Hemangioma of bone5.Enchondroma: at costochondral / costovertebral junction6.Osteochondroma: at costochondral / costovertebral junction7.Giant cell tumor8.Aneurysmal bone cystB.PRIMARY MALIGNANT RIB TUMOR1.Chondrosarcoma (calcified matrix)2.Osteosarcoma (rare)3.FibrosarcomaC.SECONDARY MALIGNANT RIB TUMOR-in adult:1.Metastasis (most common malignant lesion)2.Multiple myeloma3.Desmoid tumor-in child:1.Ewing sarcoma2.Metastatic neuroblastomaD.TRAUMATIC RIB DISORDER1.Healing fracture(a)cough fractures: 4-9th rib in anterior axillary line(b)fatigue fracture: 1st rib (from carrying a heavy back pack)2.Radiation osteitisE.Aggressive granulomatous infections=osteomyelitis

Notes:

Rib Notching On Inferior Margin =minimal scalloping to deep ridges along the neurovascular groove◊Minor undulations in the inferior ribs are normal!◊The medial third of posterior ribs near transverse process of vertebrae may be notched normally!A.ARTERIAL*Cause:*intercostal aa. function as collaterals to descending aorta / lung(a)Aorta: coarctation, thrombosis(b)Subclavian artery: Blalock-Taussig shunt(c)Pulmonary artery: pulmonary stenosis, tetralogy of Fallot, absent pulmonary arteryB.VENOUS*Cause:*enlargement of intercostal veins(a)AV malformation of chest wall(b)Superior vena cava obstructionC.NEUROGENIC1.Intercostal neuroma2.Neurofibromatosis3.Poliomyelitis / quadriplegia / paraplegiaD.OSSEOUS1.Hyperparathyroidism2.Thalassemia3.Melnick-Needles syndrome

Notes:

Rib Notching On Superior Margin 1.Rheumatoid arthritis2.Scleroderma3.Systemic lupus erythematosus4.Hyperparathyroidism5.Restrictive lung disease6.Marfan syndrome

Notes:

Ribbon Ribs 1.Osteogenesis imperfecta2.Neurofibromatosis

Notes:

Bulbous Enlargement Of Costochondral Junction 1.Rachitic rosary2.Scurvy3.Achondroplasia

Notes:

Wide Ribs 1.Marrow hyperplasia (anemias)2.Fibrous dysplasia3.Paget disease4.Achondroplasia5.Mucopolysaccharidoses

Notes:

Expansile Rib Lesion *mnemonic:*"FEEL THE CLAMP"**F**ibrous dysplasia **E**osinophilic granuloma **E**nchondroma **L**ymphoma **T**uberculosis **H**ematopoiesis **E**wing sarcoma **C**hondromyxoid fibroma **L**eukemia **A**neurysmal bone cyst **M**etastases **P**lasmacytoma

Notes:

Short Ribs 1.Achondroplasia2.Achondrogenesis3.Thanatophoric dysplasia4.Asphyxiating thoracic dysplasia5.Mesomelic dwarfism6.Short rib-polydactyly syndrome7.Spondyloepiphyseal dysplasia8.Enchondromatosis9.Chondroectodermal dysplasia (Ellis-van Creveld)

Notes:

Dense Ribs 1.Osteopetrosis2.Mastocytosis3.Fluorosis

Notes:

Hyperlucent Ribs 1.Osteopetrosis2.Cushing disease3.Acromegaly4.Scurvy

Notes:

Absence Of Outer End Of Clavicle 1.Rheumatoid arthritis2.Hyperparathyroidism3.Posttraumatic osteolysis4.Metastasis / multiple myeloma5.Cleidocranial dysplasia

Notes:

Penciled Distal End Of Clavicle *mnemonic:*"SHIRT Pocket"**S**cleroderma **H**yperparathyroidism **I**nfection **R**heumatoid arthritis **T**rauma **P**rogeria

Notes:

Destruction Of Medial End Of Clavicle *mnemonic:*"MILERS"**M**etastases **I**nfection **L**ymphoma **E**osinophilic granuloma **R**heumatoid arthritis **S**arcoma

Notes:

Carpal Angle =angle of 130° formed by tangents to proximal row of carpal bonesA.DECREASED CARPAL ANGLE (<124°)1.Turner syndrome2.Hurler syndrome3.Morquio syndrome4.Madelung deformityB.INCREASED CARPAL ANGLE (>139°)1.Down syndrome2.Arthrogryposis3.Bone dysplasia with epiphyseal involvement

---

Notes:

Metacarpal Sign =tangent between 4th + 5th metacarpals intersects 3rd metacarpal = shortening of 4th metacarpal1.Idiopathic2.Gonadal dysgenesis: Turner syndrome, Klinefelter syndrome3.Pseudo- and pseudopseudohypoparathyroidism4.Ectodermal dysplasia = Cornelia de Lange syndrome5.Hereditary multiple exostoses6.Peripheral dysostosis7.Basal cell nevus syndrome8.Melorheostosis *mnemonic:*"**P**ing **P**ong **I**s **T**ough **T**o **T**each"**P**seudohypoparathyroidism **P**seudopseudohypoparathyroidism **I**diopathic **T**rauma **T**urner syndrome **T**risomy 13-18

Notes:

Lucent Lesion In Finger A.BENIGN TUMOR1.Giant cell tumor2.Aneurysmal bone cyst3.Brown tumor 4Hemophilic pseudotumor5.Epidermoid inclusion cyst6Glomus tumor7.Solitary bone cyst8.Osteoblastoma9.EnchondromaB.MALIGNANT TUMOR1.Osteosarcoma2.Fibrosarcoma3.Metastasis from lung, breast, malignant melanoma *mnemonic:*"GAMES PAGES"**G**lomus tumor **A**rthritis (gout, rheumatoid) **M**etastasis (lung, breast) **E**nchondroma **S**imple cyst (inclusion) **P**ancreatitis **A**neurysmal bone cyst **G**iant cell tumor **E**pidermoid **S**arcoid

Notes:

Resorption Of Terminal Tufts A.TRAUMA1.Amputation2.Burns, electric injury3.Frostbite4.Vinyl chloride poisoningB.NEUROPATHIC1.Congenital indifference to pain2.Syringomyelia3.Myelomeningocele4.Diabetes mellitus5.LeprosyC.COLLAGEN-VASCULAR DISEASE1.Scleroderma2.Dermatomyositis3.Raynaud disease D.METABOLIC1.HyperparathyroidismE.INHERITED1.Familial acroosteolysis2.Pyknodysostosis3.Progeria = Werner syndrome4.PachydermoperiostosisF.OTHERS1.Sarcoidosis2.Psoriatic arthropathy3.Epidermolysis bullosa

Notes:

Acroosteolysis 1.Acroosteolysis: (a) acquired, (b) familial2.Massive osteolysis3.Essential osteolysis4.Ainhum disease ***Acquired Acroosteolysis*** *mnemonic:*"PETER's DIAPER SPLASH"**P**soriasis, **P**orphyria **E**hlers-Danlos syndrome **T**hrombangitis obliterans **E**rgot therapy **R**aynaud disease **D**iabetes, **D**ermatomyositis, **D**ilantin therapy **I**njury (thermal + electrical burns, frostbite) **A**rteriosclerosis obliterans **P**VC (polyvinylchloride) worker **E**pidermolysis bullosa **R**heumatoid arthritis, **R**eiter syndrome **S**cleroderma, **S**arcoidosis **P**rogeria, **P**yknodysostosis **L**eprosy, **L**esch-Nyhan syndrome **A**bsence of pain **S**yringomyelia **H**yperparathyroidism also in:yaws; Kaposi sarcoma; pachydermoperiostosis√ lytic destructive process involving distal + middle phalanges√ NO periosteal reaction√ epiphyses resist osteolysis until late Acroosteosclerosis =focal opaque areas + endosteal thickening1.Incidental in middle-aged women2.Rheumatoid arthritis3.Sarcoidosis4.Scleroderma5.Systemic lupus erythematosus6.Hodgkin disease7.Hematologic disorders

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Notes:

Fingertip Calcifications 1.Scleroderma / CREST syndrome2.Raynaud disease3.Systemic lupus erythematosus4.Dermatomyositis5.Calcinosis circumscripta universalis6.Hyperparathyroidism

Notes:

Syndactyly =osseous ± cutaneous fusion of digits1.Apert syndrome2.Carpenter syndrome3.Down syndrome4.Neurofibromatosis5.Poland syndrome6.Others

Notes:

Polydactyly Frequently associated with: 1.Carpenter syndrome2.Ellis-van Creveld syndrome3.Meckel-Gruber syndrome4.Polysyndactyly syndrome5.Short rib-polydactyly syndrome6.Trisomy 13

Notes:

Clinodactyly =curvature of finger in mediolateral plane1.Normal variant2.Down syndrome3.Multiple dysplasia4.Trauma, arthritis, contractures

Notes:

Brachydactyly =shortening / broadening of metacarpals ± phalanges1.Idiopathic2.Trauma3.Osteomyelitis4.Arthritis5.Turner syndrome6.Osteochondrodysplasia7.Pseudohypoparathyroidism, Pseudopseudohypoparathyroidism8.Mucopolysaccharidoses9.Cornelia de Lange syndrome10.Basal cell nevus syndrome11.Hereditary multiple exostoses

Notes:

Snapping Hip Syndrome A.INTRAARTICULAR1.Osteocartilaginous bodiesB.EXTRA-ARTICULAR = tendon slippage1.fascia lata / gluteus maximus over greater trochanter2.iliopsoas tendon over iliopectineal eminence3.long head of biceps femoris over ischial tuberosity4.iliofemoral ligament over anterior portion of hip capsule

Notes:

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Protrusio Acetabuli =acetabular floor bulging into pelvis√ acetabular line projecting medially to ilioischial line by >3 mm (in males) / >6 mm (in females)√ crossing of medial + lateral components of pelvic "teardrop" (U-shaped radiodense area medial to hip joint with (a) lateral aspect = acetabular articular surface (b) medial aspect = anteroinferior margin of quadrilateral surface of ilium)A.UNILATERAL1.Tuberculous arthritis2.Trauma3.Fibrous dysplasiaB.BILATERAL1.Rheumatoid arthritis2.Paget disease3.Osteomalacia *mnemonic:*"PROT"**P**aget disease **R**heumatoid arthritis **O**steomalacia (HPT) **T**rauma

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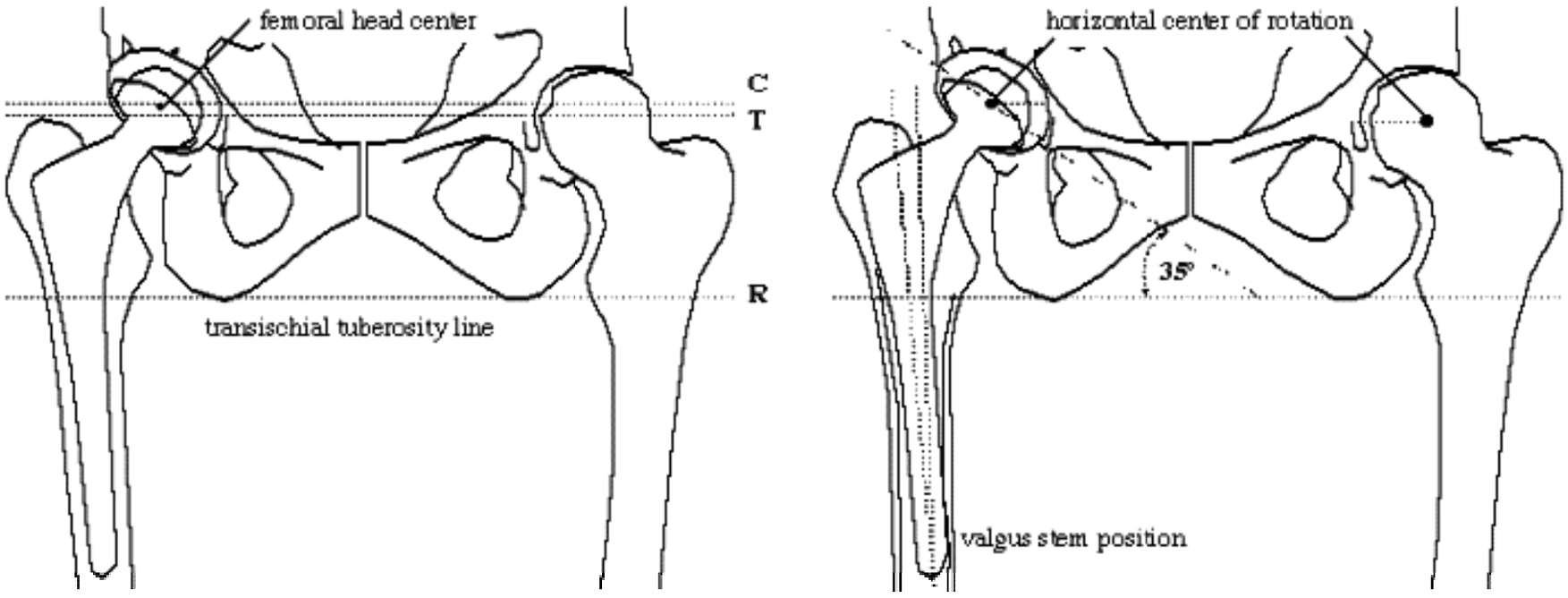
Notes:

Pain With Hip Prosthesis Approximately 120,000 hip arthroplasties per year in USA 1.Heterotopic ossification2.Trochanteric bursitis3.Prosthetic fracture / periprosthetic fracture / cement fracture4.Dislocation5.Loosening (10-30% after 10 years)(a)aseptic loosening (most common)*Cause:*mechanical wear + tear(b)septic loosening (1-9%)*Organism:*Staphylococcus epidermidis (50%), Staphylococcus aureus, PeptostreptococcusPlain film: √ subsidence of prosthesis√ area of lucency >2 mm at bone-cement interface√ focal lytic area (due to foreign body granuloma / abscess)√ rapid bone resorption (due to particulate debris / infection)√ extensive periostitis (in infection, but rare)NUC (83% sensitive, 88% specific): √ increased uptake of bone agent, gallium-67, indium-111-labeled leukocytes, complementary technetium-labeled sulfur colloid + combinationsArthrography: √ irregularity of joint pseudocapsule√ filling of nonbursal spaces / sinus tracts / abscess cavitiesAspiration of fluid under fluoroscopy (12-93% sensitive, 83-92% specific for infection): √ injection of contrast material to confirm intraarticular location

Notes:

Evaluation Of Total Hip Arthroplasty Measurements *Reference line:*transischial tuberosity line (R)



1.Leg length = vertical position of acetabular component=comparing level of greater / lesser tuberosity (T) with respect to line RHigh placement:shorter leg, less effective muscles crossing the hip jointLow placement:longer leg, muscles stretched to point of spasm with risk of dislocation2.Vertical center of rotation=distance from center of femoral head (C) to line R3.Horizontal center of rotation=distance from center of femoral head (C) to teardrop / other medial landmarkLateral position:iliopsoas tendon crosses medial to femoral head center of rotation increasing risk of dislocation4.Lateral acetabular inclination = horizontal version=angle of cup in reference to line R (40° ± 10° desirable)Less angulation:stable hip, limited abductionGreater angulation:risk of hip dislocation 5.Varus / neutral / valgus stem positionVarus position:tip of stem rests against medial endosteum, increased risk for looseningValgus position:tip of stem rests against lateral endosteum, not a significant problem 6.Acetabular anteversion (15° ± 10° desirable)=lateral radiograph of groinRetroversion:risk of hip dislocation7.Femoral neck anteversionworks synergistically with acetabular anteversion, true angle assessed by CT ***Radiographic findings*** A.NORMAL√ irregular cement-bone interface=normal interdigitation of polymethylmethacrylate (PMMA) with adjacent bone remodeling providing a mechanical interlock◊PMMA is not a glue!√ thin lucent line along cement-bone interface=0.1-1.5 mm thin connective tissue membrane ("demarcation") along cement-bone interface accompanied by thin line of bone sclerosisB.ABNORMAL√ wide lucent zone at cement-bone interface=≥2 mm lucent line along bone-cement interface due to granulomatous membrane*Cause:*component loosening ± reaction to particulate debris (eg, PMMA, polyethylene)√ lucent zone at metal-cement interface along proximal lateral aspect of femoral stem=suboptimal metal-cement contact at time of surgery / loosening√ well-defined area of bone destruction(= histiocytic response, aggressive granulomatous disease) *Cause:*granulomatous reaction as response to particulate debris / infection / tumor√ asymmetric positioning of femoral head within acetabular component*Cause:*acetabular wear / dislocation of femoral head / acetabular disruption / liner displacement / deformity√ cement fracture*Cause:*loosening

Notes:

Tibiotalar Slanting =downward slanting of medial tibial plafond1.Hemophilia2.Still disease3.Sickle cell disease4.Epiphyseal dysplasia5.Trauma

Notes:

Abnormal Foot Positions A.FOREFOOT1.Varus = adduction=axis of 1st metatarsal deviated medially relative to axis of talus2.Valgus = abduction=axis of 1st metatarsal deviated laterally relative to axis of talus3.Inversion = supination=inward turning of sole of foot4.Eversion = pronation=outward turning of sole of footB.HINDFOOTtalipes (talus, pes) = any deformity of the ankle and hindfoot 1.Equinus=hindfoot abnormality with reversal of calcaneal pitch so that the heel cannot touch the ground2.Calcaneal foot=very high calcaneal pitch so that forefoot cannot touch the ground3.Pes planus = flatfoot=low calcaneal pitch + (usually) heel valgus + forefoot eversion4.Pes cavus=high calcaneal pitch (fixed high arch)

Notes:

Clubfoot = Talipes Equinovarus Common severe congenital deformity characterized by ● equinus of heel (reversed calcaneal pitch) ● heel varus (talocalcaneal angle of almost zero on AP view with both bones parallel to each other) ● metatarsus adductus (axis of 1st metatarsal deviated medially relative to axis of talus)1.Arthrogryposis multiplex congenita2.Chondrodysplasia punctata3.Neurofibromatosis4.Spina bifida5.Myelomeningocele

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Notes:

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Rocker-bottom Foot = Vertical Talus √ vertically oriented talus with increased talocalcaneal angle on lateral view√ dorsal navicular dislocation at talonavicular joint√ heel equinus√ rigid deformity*Associated with:* Arthrogryposis multiplex congenita; spina bifida; trisomy 13-18

Notes:

Heel Pad Thickening =heel pad thickening >25 mm (normal <21 mm)*mnemonic:*"MAD COP"**M**yxedema **A**cromegaly **D**ilantin therapy **C**allus **O**besity **P**eripheral edema

Notes:

Histologic Classification Of Soft-tissue Lesions A.FATTY1.Lipoma2.Angiolipoma3.LiposarcomaB.FIBROUS1.Fibroma2.Nodular fasciitis3.Aggressive fibromatosis / desmoid4.FibrosarcomaC.MUSCLE1.Rhabdomyoma2.Leiomyoma3.Rhabdomyosarcoma4.LeiomyosarcomaD.VASCULAR1.Hemangioma2.Hemangiopericytoma3.HemangiosarcomaE.LYMPH1.Lymphangioma2.Lymphangiosarcoma3.Lymphadenopathy in lymphoma / metastasisF.SYNOVIAL1.Nodular synovitis2.Pigmented villonodular synovitis3.Synovial sarcomaG.NEURAL1.Neurofibroma2.Neurilemoma3.Ganglioneuroma4.Malignant neuroblastoma5.NeurofibrosarcomaH.CARTILAGE AND BONE1.Myositis ossificans2.Extraskeletal osteoma3.Extraskeletal chondroma4.Extraskeletal chondrosarcoma5.Extraskeletal osteosarcoma

Notes:

Fat-containing Soft-tissue Masses A.BENIGN LIPOMATOUS TUMORS1.Lipoma2.Intra- / intermuscular lipoma3.Synovial lipoma4.Lipoma arborescens = diffuse synovial lipoma5.Neural fibrolipoma = fibrolipomatous tumor of nerve6.Macrodystrophia lipomatosaB.LIPOMA VARIANTS1.Lipoblastoma (exclusively in infancy + early childhood)2.**Lipomatosis** = diffuse overgrowth of mature adipose tissue infiltrating through the soft tissues of affected extremity / trunk 3.**Hibernoma** = rare benign tumor of brown fat; often in peri- / interscapular region, axilla, thigh, chest wall√ marked hypervascularityC.MALIGNANT LIPOMATOUS TUMOR1.LiposarcomaD.OTHER FAT-CONTAINING TUMORS1.Hemangioma2.ElastofibromaE.LESIONS MIMICKING FAT-CONTAINING TUMORS1.Myxoid tumors: intramuscular myxoma, extraskeletal myxoid chondrosarcoma, myxoid malignant fibrous histiocytoma2.Neural tumors: neurofibroma, neurilemoma, malignant schwannoma√ 73% have tissue attenuation less than muscle3.Hemorrhage

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Notes:

Muscle Hyperintensity On STIR Images A.INFLAMMATION1.Polymyositis2.Dermatomyositis3.Inclusion body myositisB.CELLULAR INFILTRATE1.Lymphoma2.Bacterial myositisC.EDEMAD.RHABDOMYOLYSIS1.Sport / electric injury2.Diabetic muscular infarction3.Focal nodular myositis4.Metabolic myopathy: eg, phosphofructokinase deficiency, hypokalemia, alcohol overdose5.Viral myositisE.TRAUMATIC DENERVATION

Notes:

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Extraskeletal Osseous + Cartilaginous Tumors A. OSSEOUS SOFT-TISSUE TUMORS √ cloudlike "cumulus" type of calcification1.Myositis ossificans2.Fibrodysplasia ossificans progressiva3.Soft-tissue osteoma4.Extraskeletal osteosarcoma5.Myositis ossificans variants(a)Panniculitis ossificans(b)Fasciitis ossificans(c)Fibro-osseous pseudotumor of digitsB.CARTILAGINOUS SOFT-TISSUE TUMORS√ arcs and rings, spicules and floccules of calcification1.Synovial osteochondromatosis2.Soft-tissue chondroma3.Extraskeletal chondrosarcoma *DDx:* (1)Synovial sarcoma(2)Benign mesenchymoma= lipoma with chondroid / osseous metaplasia (3)Malignant mesenchymoma= 2 or more unrelated sarcomatous components (4)Calcified / ossified tophus of gout(5)Ossified soft-tissue masses of melorheostosis(6)**Pilomatricoma** = calcifying epithelioma of Malherbe ● lesion arises from hair matrix cells with slow growth confined to the subcutaneous tissue of the face, neck, upper extremities√ central sandlike calcifications (84%)√ peripheral ossification (20%)(7)Tumoral calcinosis

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Notes:

Soft-tissue Calcification ***Metastatic Calcification*** =deposit of calcium salts in previously normal tissue(1) as a result of elevation of Ca x P product above 60-70 (2) with normal Ca x P product after renal transplant Location:lung (alveolar septa, bronchial wall, vessel wall), kidney, gastric mucosa, heart, peripheral vessels*Cause:* (a)Skeletal deossification1.1° HPT2.Ectopic HPT production (lung / kidney tumor)3.Renal osteodystrophy + 2° HPT4.Hypoparathyroidism(b)Massive bone destruction1.Widespread bone metastases2.Plasma cell myeloma3.Leukemia(c)Increased intestinal absorption1.Hypervitaminosis D2.Milk-alkali syndrome3.Excess ingestion / IV administration of calcium salts4.Prolonged immobilization5.Sarcoidosis(d)Idiopathic hypercalcemia ***Dystrophic Calcification*** =in presence of normal serum Ca + P levels secondary to local electrolyte / enzyme alterations in areas of tissue injury*Cause:* (a)Metabolic disorder without hypercalcemia1.Renal osteodystrophy with 2° HPT2.Hypoparathyroidism3.Pseudohypoparathyroidism4.Pseudopseudohypoparathyroidism5.Gout6.Pseudogout = chondrocalcinosis7.Ochronosis = alkaptonuria8.Diabetes mellitus(b) Connective tissue disorder1.Scleroderma2.Dermatomyositis3.Systemic lupus erythematosus(c)Trauma1.Neuropathic calcifications2.Frostbite3.Myositis ossificans progressiva4.Calcific tendinitis / bursitis(d)Infestation1.Cysticercosis2.Dracunculosis (guinea worm)3.Loiasis4.Bancroft filariasis5.Hydatid disease6.Leprosy(e)Vascular disease1.Atherosclerosis2.Media sclerosis (Mönckeberg)3.Venous calcifications4.Tissue infarction (eg, myocardial infarction)(f)Miscellaneous1.Ehlers-Danlos syndrome2.Pseudoxanthoma elasticum3.Werner syndrome = progeria4.Calcinosis (circumscripta, universalis, tumoral calcinosis)5.Necrotic tumor ***Generalized Calcinosis*** (a)Collagen vascular disorders1.Scleroderma2.Dermatomyositis(b)Idiopathic tumoral calcinosis(c)Idiopathic calcinosis universalis

Notes:

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Interstitial Calcinosis
Calcinosis Circumscripta 1.Acrosclerosis: granular deposits around joints of fingers + toes, fingertips2.Scleroderma: acrosclerosis + absorption of ends of distal phalanges3.Dermatomyositis: extensive subcutaneous deposits4.Varicosities: particularly in calf5.1° Hyperparathyroidism: infrequently periarticular calcinosis6.Renal osteodystrophy with 2° hyperparathyroidism: extensive vascular deposits even in young individuals7.Hypoparathyroidism: occasionally around joints; symmetrical in basal ganglia8.Vitamin D intoxication: periarticular in rheumatoid arthritis (puttylike); calcium deposit in tophi ***Calcinosis Universalis*** Progressive disease of unknown origin *Age:*children + young adults√ plaquelike calcium deposits in skin + subcutaneous tissues; sometimes in tendons + muscles√ NO true bone formation

Notes:

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Soft-tissue Ossification =formation of trabecular bone1.Myositis ossificans progressiva / circumscripta2.Paraosteoarthropathy3.Soft-tissue osteosarcoma4.Parosteal osteosarcoma5.Posttraumatic periostitis = periosteoma6.Surgical scar7.Severely burned patient

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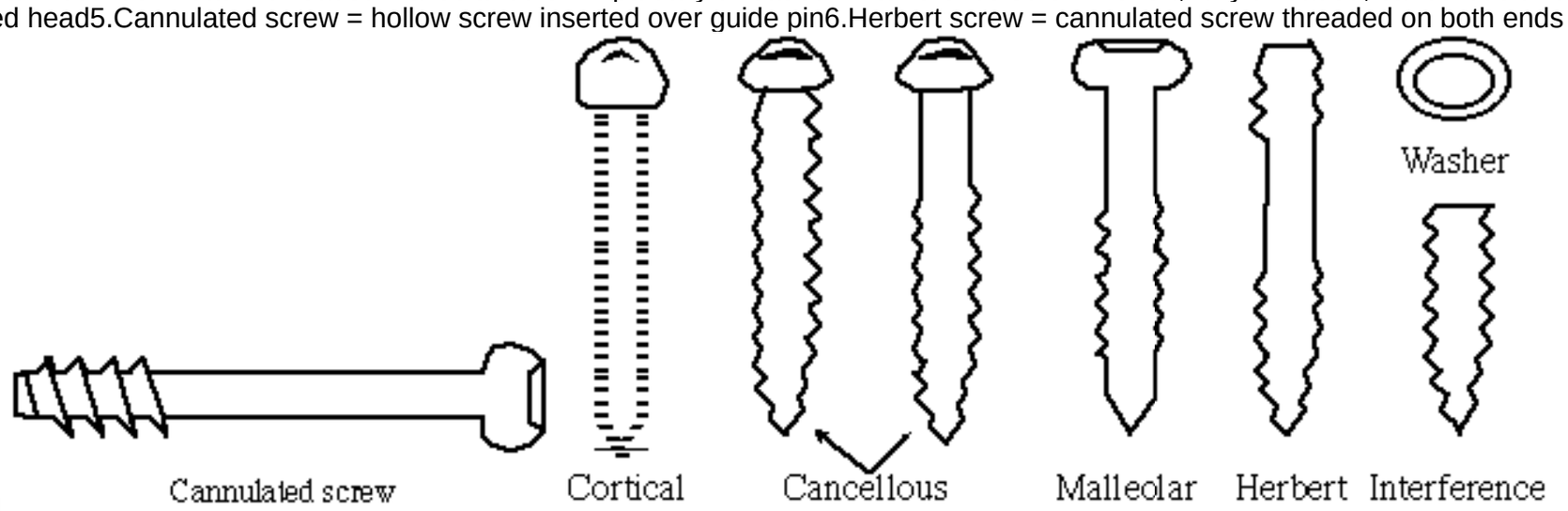
Notes:

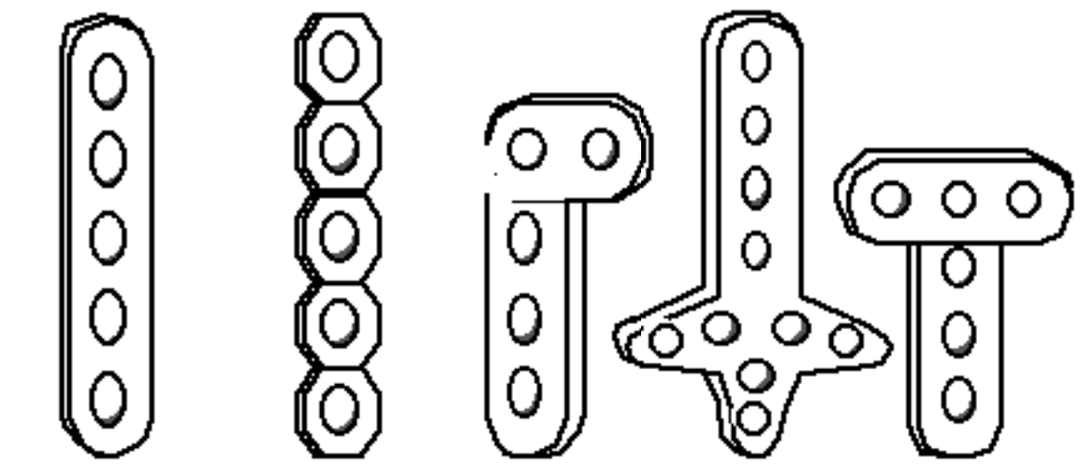
Connective Tissue Disease =CTD = [COLLAGEN VASCULAR DISEASE]=group of disorders that share a number of clinical + laboratory features ● Features:(a)relatively specific: arthritis, myositis, Raynaud phenomenon with digital ulceration, tethered skin in extremities + trunk, malar rash sparing nasolabial folds, morning stiffness(b)relatively nonspecific: polyarthralgias (most common initial symptom), myalgias, mottling of extremities, muscle weakness + tenderness ● Laboratory findings:(a)relatively specific: ANA in peripheral rim / nucleolar pattern, anti-DNA, elevated muscle enzyme(b)relatively nonspecific: ANA in homogeneous pattern, anti-single-stranded DNA, positive rheumatoid factor *Types and most distinctive features:* 1.Rheumatoid arthritispositive rheumatoid factor, prominent morning stiffness, symmetric erosive arthritis2.Systemic lupus erythematosusmalar rash, photosensitivity, serositis, renal disorders with hemolytic anemia, leukopenia, lymphopenia, thrombocytopenia, positive antinuclear antibody (ANA)3.Sjögren syndromedry eyes + mouth, abnormal Schirmer test4.SclerodermaRaynaud phenomenon, skin thickening of distal extremities proceeding to include proximal extremities + chest + abdomen, positive ANA in a nucleolar pattern5.Polymyositis, dermatomyositisheliotrope rash over eyes, proximal muscle weakness, elevated muscle enzymes, inflammation at muscle biopsy ***Mixed Connective Tissue Disease*** = disorder that shares distinctive features of ≥2 different connective tissue diseases in same patient (eg, overlapping features of SLE, PSS, polymyositis) ● pulmonary hypertension (due to interstitial pulmonary fibrosis / intimal proliferation of pulmonary arterioles)

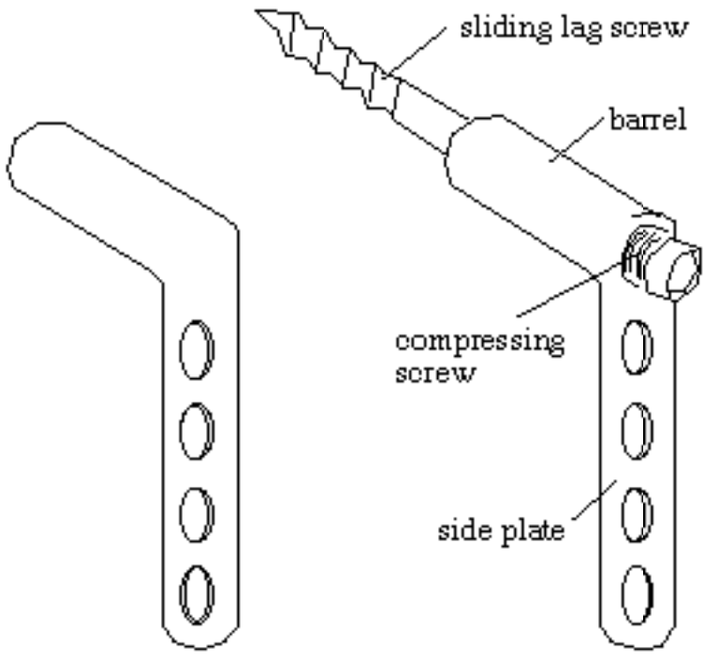
Notes:

Internal Fixation Devices A.Screws1.Cortical screw = threaded over entire length, shallow closely spaced threads, blunt tip2.Cancellous screw = wide thread diameter with varying length of smooth shank between head + threads3.Malleolar screw = partially threaded4.Interference screw = short, fully threaded, cancellous thread pattern, self-tapping tip, recessed head5.Cannulated screw = hollow screw inserted over guide pin6.Herbert screw = cannulated screw threaded on both ends with different pitches, no screw head

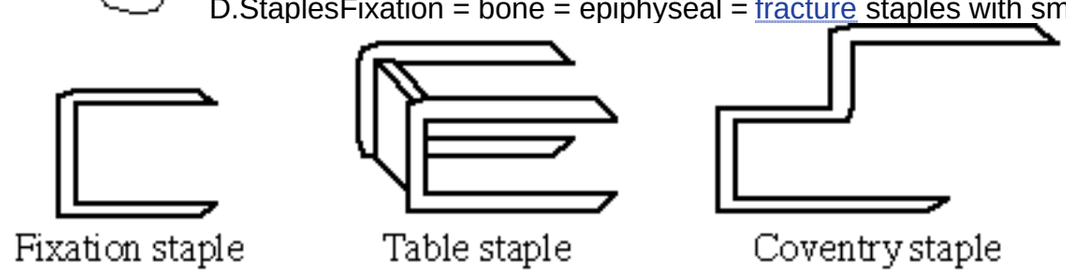


B.Washer1.Flat washer = increase surface area over which force is distributed2.Serrated washer = spiked edges used for affixing avulsed ligamentsC.Plates-Compression plate = used for compression of stable fractures-Neutralization plate = protects fracture from bending, rotation + axial-loading forces-Buttress plate = support of unstable fractures in compression / axial loading1.Straight plate(a)straight plate with round holes(b)dynamic compression plate = oval holes(c)tubular plate = thin pliable plate with concave inner surface(d)reconstruction plate = thin pliable plate to allow bending, twisting, contouring2.Special platesT-shaped, L-shaped, Y-shaped, cloverleaf, spoon, cobra, condylar blade plate, dynamic compression screw system





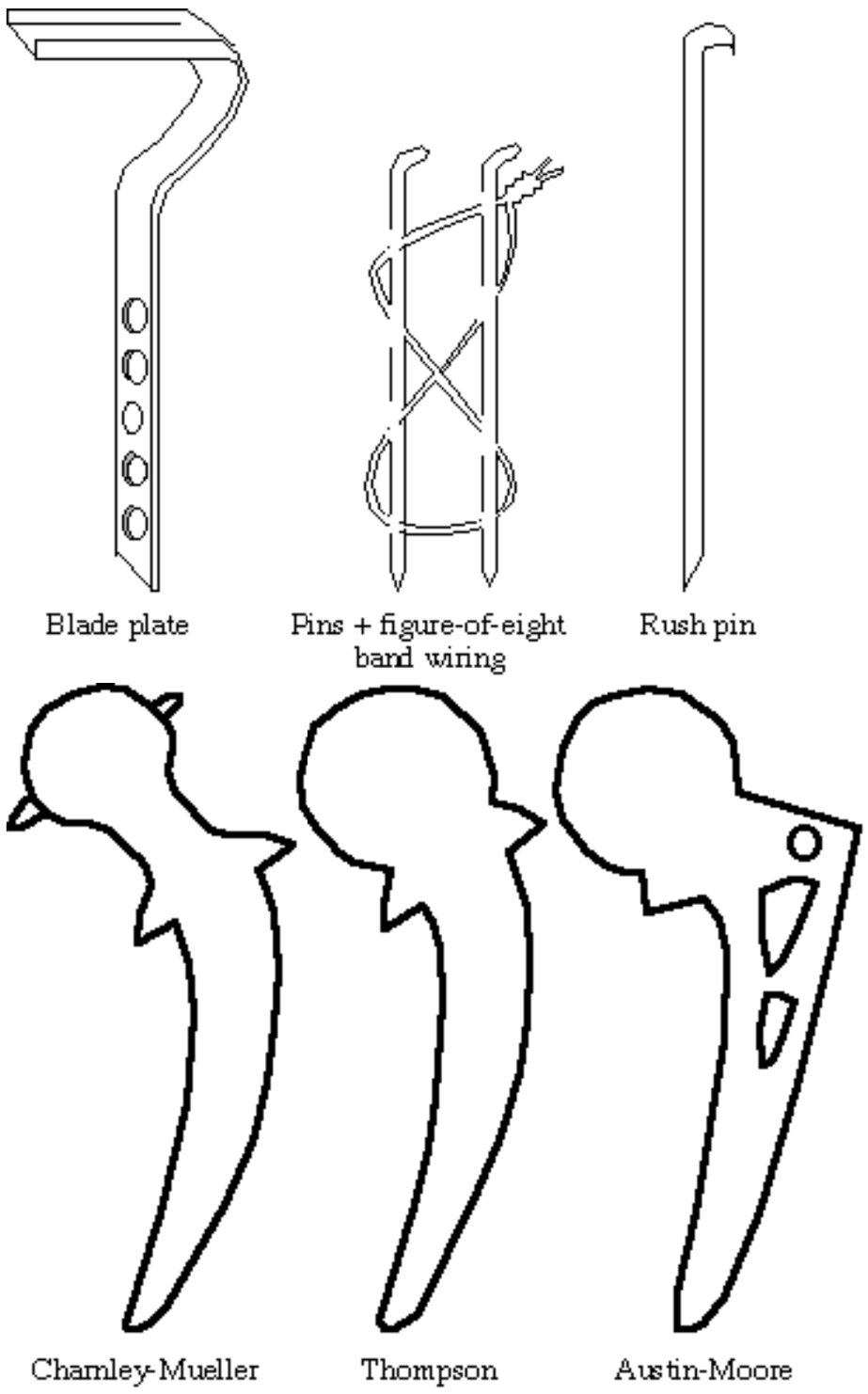
D.StaplesFixation = bone = epiphyseal = fracture staples with smooth / barbed surface -Coventry = stepped osteotomy staple-stone = table staple



E.Wires1.K wire = unthreaded segments of extruded wire of variable thickness2.Cerclage wiring = wire placed around bone3.Tension band wiring = figure-of-eight wire placed on tension side of bone

Notes:

External Fixation Devices =smooth / threaded pins / wires attached to an external frame(a)unilateral pin = enters bone only from one side1.Steinmann pin = large-caliber wire with pointed tip2.Rush pin = smooth intramedullary pin3.Schanz screw = pin threaded at one end to engage cortex, smooth at other end to connect to external fixation device4.Knowles pin (for femoral neck fracture)(b)transfixing pin = passes through extremity supported by external fixation device on both ends



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Intramedullary Fixation Devices (a)nail = driven into bone without reaming(b)rod = solid / hollow device with blunted tip driven into reamed channel(c)interlocking nail = accessory pins / screws / deployable fins placed to prevent rotation1.Rush pin = beveled end + hooked end2.Ender nail = oval in cross section3.Sampson rod = slightly curved rigid rod with fluted surface4.Küntscher nail = cloverleaf in cross section with rounded tip

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Notes:

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A Wolters Kluwer Company

Calcium A.99% in boneB.serum calcium(a)protein-bound fraction (albumin)(b) ionic (pH-dependent) 3% as calcium citrate / phosphate in serum*Absorption:*facilitated by vitamin D*Excretion:*related to dietary intake; >500 mg/24 hours = hypercalciuria

Notes:

Phosphorus *Absorption:*requires sodium; decreased by aluminum hydroxide gel in gut*Excretion:*increased by estrogen, parathormone decreased by vitamin D, growth hormone, glucocorticoids

Notes:

Parathormone *Major stimulus:*low levels of serum calcium ions (action requires vitamin D presence)*Target organs:* (a) BONE:increase in osteocytic + osteoclastic activity mobilizes calcium + phosphate= bone resorption(b) KIDNEY:(1)increase in tubular reabsorption of calcium(2)decrease in tubular reabsorption of phosphate (+ amino acids)= phosphate diuresis(c) GUT:increased absorption of calcium + phosphorus *Major function:* ● increase of serum calcium levels ● increase in serum alkaline phosphatase (50%)

	PTH ACTION	NET EFFECT	
Principal:	(1) phosphate diuresis (2) resorption of Ca + P from bone	(1) Serum:	increase in Ca decrease in P
Secondary:	(3) resorption of Ca from gut (4) reabsorption of Ca from renal tubule	(2) Urine:	increase in Ca increase in P

Notes:

Radiology Review Manual
fourth edition

Vitamin D Metabolism required for (1)adequate calcium absorption from gut(2)synthesis of calcium-binding protein in intestinal mucosa(3)parathormone effects (stimulation of osteoclastic + osteocytic resorption of bone)*Biochemistry:* inactive form of vitamin D_3 present through diet / exposure to sunlight; vitamin D_3 is converted into 25-OH-vitamin D_3 by liver and then converted into 1,25-OH vitamin D_3 (= hormone) by kidney Stimulus for conversion:(1)hypophosphatemia(2)PTH elevation*Action:* (a) INTESTINE:(1)increased absorption of calcium from bowel(2)increased absorption of phosphate from distal small bowel(b) BONE:(1)proper mineralization of osteoid(2)mobilization of calcium + phosphate (potentiates parathormone action)(c) KIDNEY:(1)increased absorption of calcium from renal tubule(2)increased absorption of phosphate from renal tubule

---

Notes:

Calcitonin secreted by parafollicular cells of thyroid *Major stimulus:*increase in serum calcium*Target organs:* (a) BONE:(1)inhibits parathormone-induced osteoclasis by reducing number of osteoclasts(2)enhances deposition of calcium phosphate; responsible for sclerosis in renal osteodystrophy(b) KIDNEY:inhibits phosphate reabsorption in renal tubule(c) GUT:increases excretion of sodium + water into gut*Major function:*decreases serum calcium + phosphate

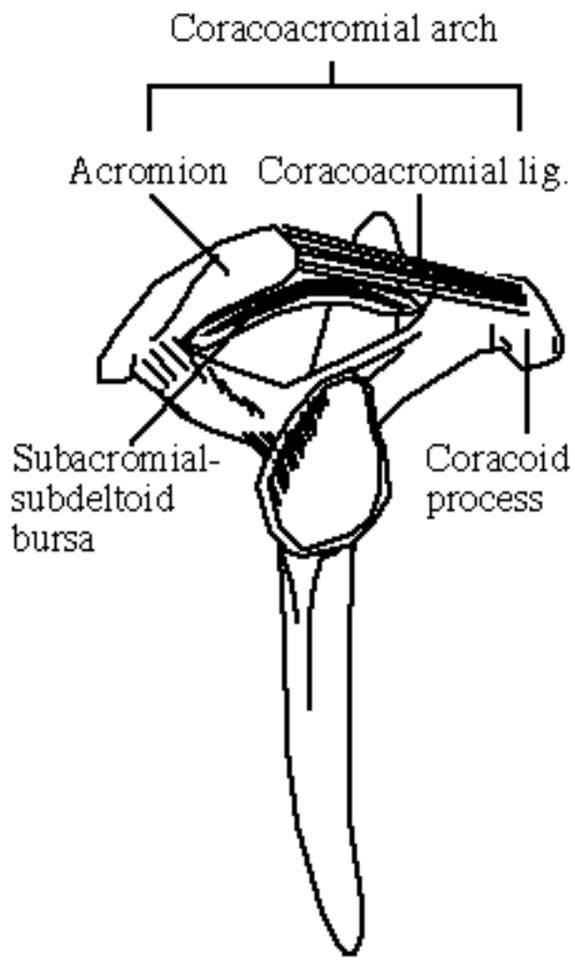
Notes:

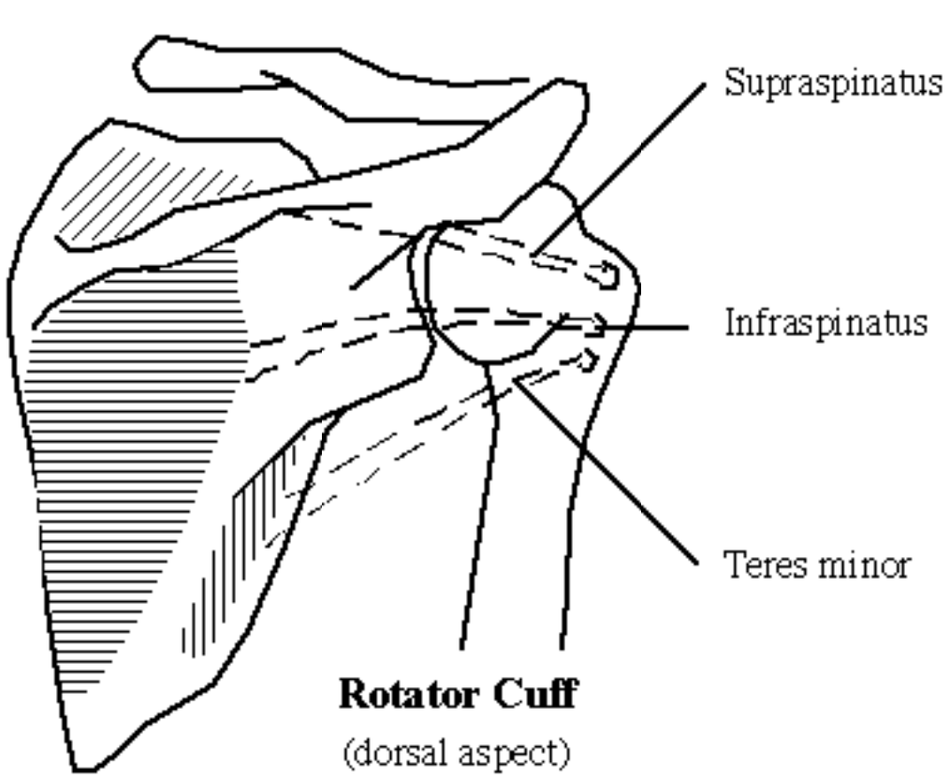
PHYSIS
Four distinct zones of cartilage in longitudinal layers (1)Germinal zone = small cellsadjacent to epiphyseal ossification center(2)Proliferating zone = flattened cellsarranged in columns(3)Hypertrophic zone = swollen vacuolated cells(4)Zone of provisional calcification

Notes:

SHOULDER

Rotator cuff muscles
mnemonic:"SITS"**S**upraspinatus**I**nfraspinatus**T**eres minor**S**ubscapularis





Muscle Attachments of Shoulder

Notes:

Muscle Attachments of Shoulder

*Name of muscle*OriginInsertion

Deltoidlateral third of claviculedeltoid tuberosity of humerus lateral border of acromiondeltoid tuberosity of humerus lower part of spinous process of scapuladeltoid tuberosity of humerus

Subscapularismedial 2/3 of costal surface of scapulasuperior aspect of lesser tubercle of humerus

Pectoralis major

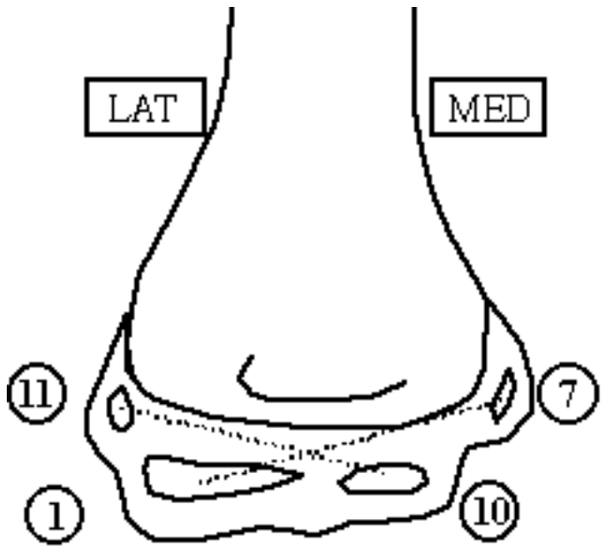
- clavicular portionmedial half of claviclecrest of greater tubercle of humerus - sternocostal portionmanubrium + corpus of sternumcrest of greater tubercle of humerus - abdominal portionanterior sheath of rectus abdominiscrest of greater tubercle of humerus **Pectoralis minor**2nd / 3rd-5th ribssuperomedial aspect of coracoid process

Biceps brachii - long headsupraglenoid tubercle of scapulatuberosity of radius - short headtip of coracoid processtuberosity of radius **Coracobrachialis**tip of coracoid processmedial surface of middle third of humerus **Supraspinatus**supraspinatous fossa of scapulagreater tubercle of humerus, highest facet

Infraspinatusinfraspinatous fossa of scapulagreater tubercle of humerus, middle facet **Teres minor**upper 2/3 of lateral border of scapula greater tubercle of humerus, lower facet **Teres major**dorsum of inferior angle of scapula inferior crest of lesser tubercle of humerus

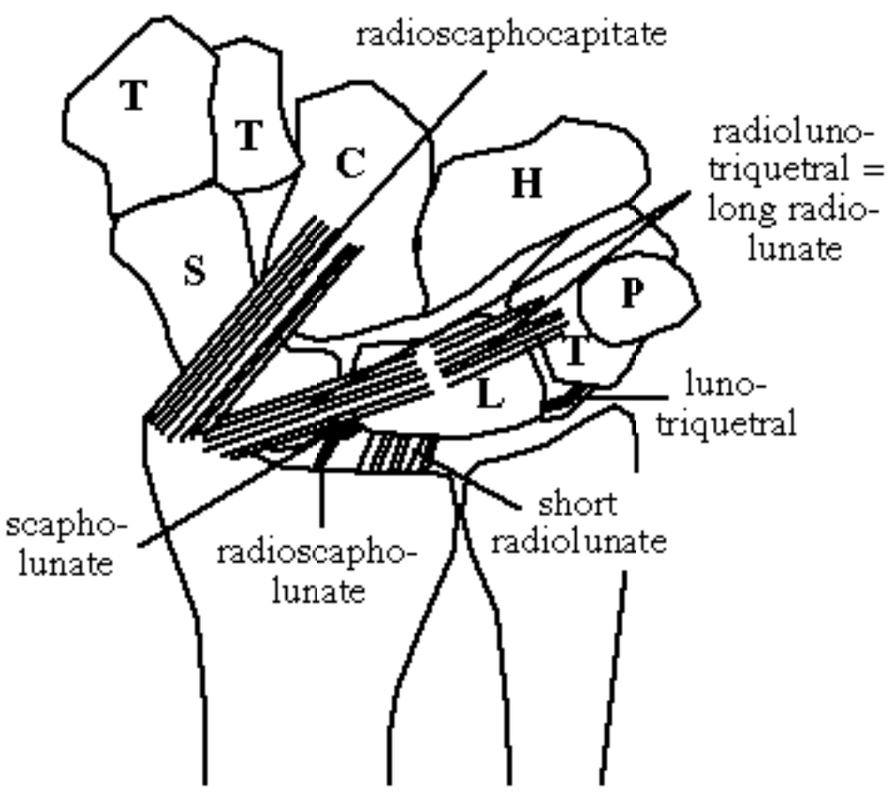
Notes:

OCCURRENCE OF BONE CENTERS AT ELBOW
mnemonic:"CRITOE"**C**apitellum1 year(3-6 months)**R**adial head4 years(3-6 years)**I**nternal humeral epicondyle 7 years(4-6 years, last to fuse)**T**rochlea10 years(9-10 years)**O**lecranon10 years(6-10 years)**E**xternal humeral epicondyle 11 years(9-12 years) *mnemonic:* "Nelsons X: 1, 7, 10, 11 years"

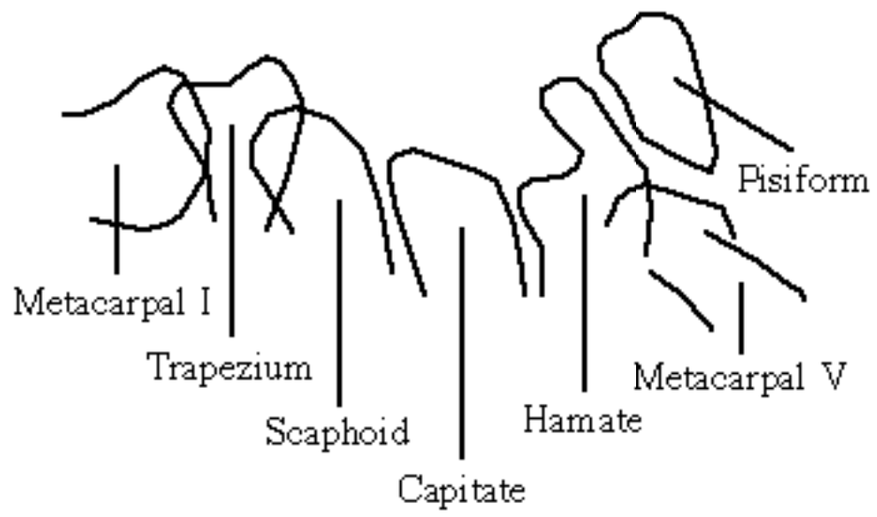


Notes:

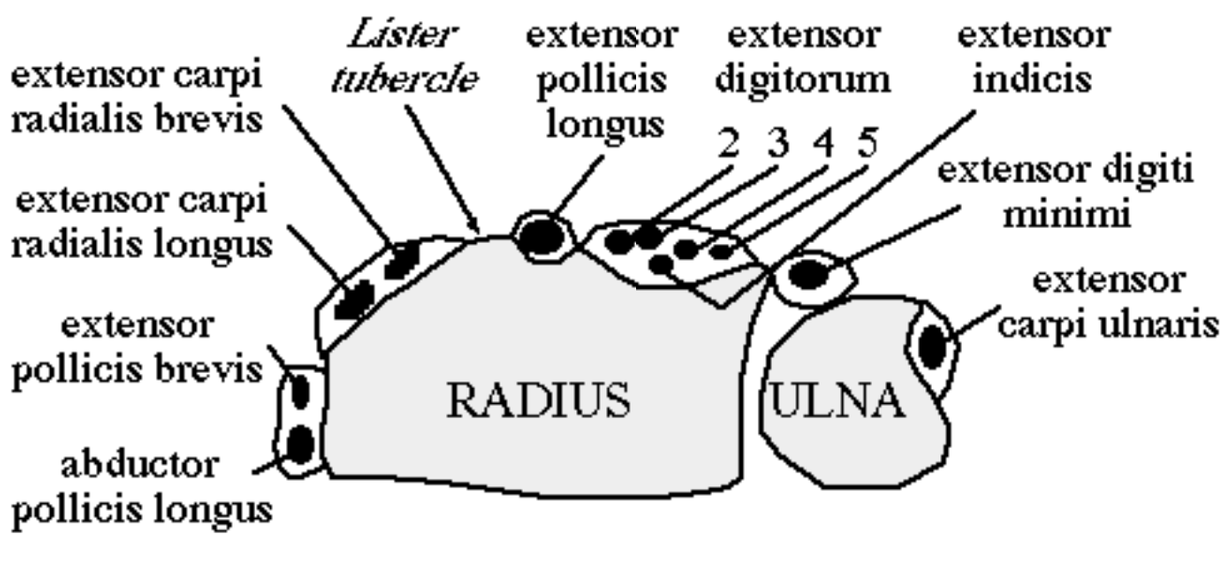
CARPAL BONES
mnemonic:"**S**ome **L**overs **T**ry **P**ositions **T**hat **T**hey **C**an't **H**andle"proximal rowdistal row **S**caphoid**T**rapezium**L**unate**T**rapezoid**T**riquetrum**C**apitate**P**isiform**H**amate ◊
Remember that trapezium comes before trapezoid in the dictionary as well!



Carpal Bones and Ligaments
(volar aspect)



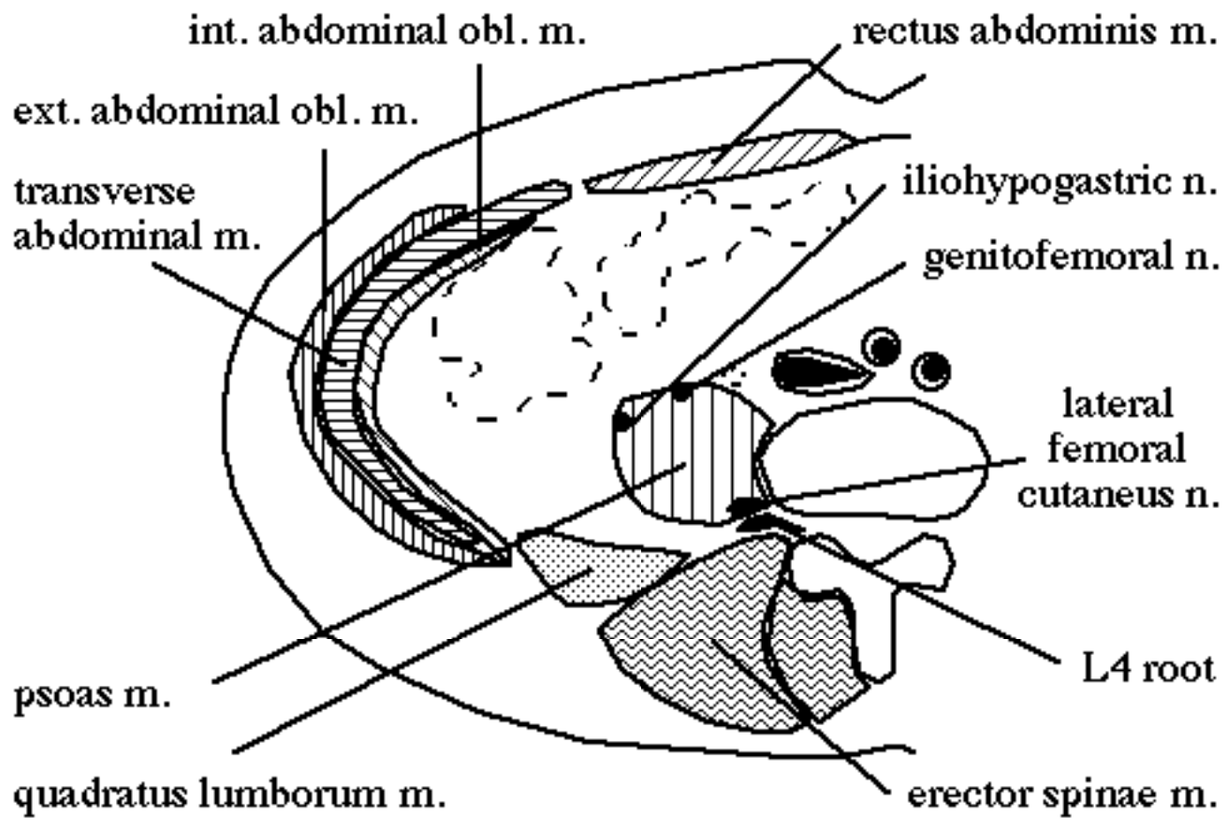
Carpal Tunnel View



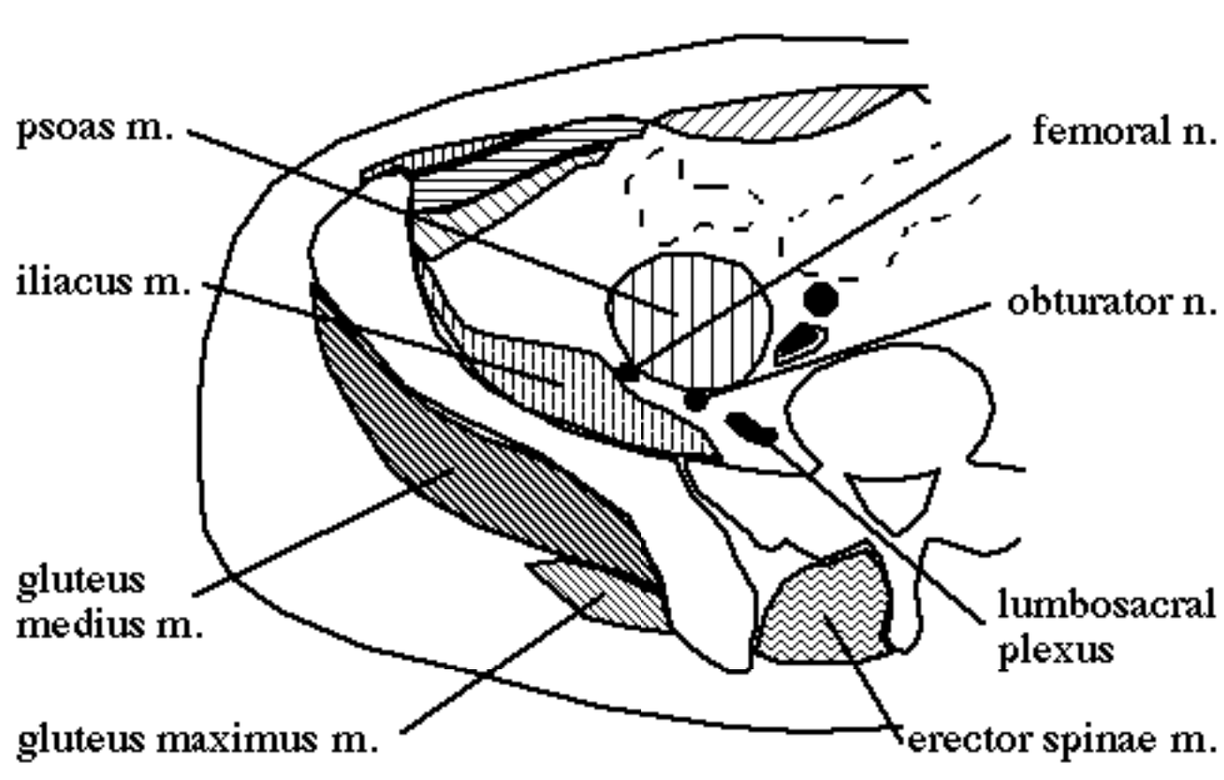
Wrist Cross Section of Distal Radioulnar Joint With the 6 Extensor Compartments

Notes:

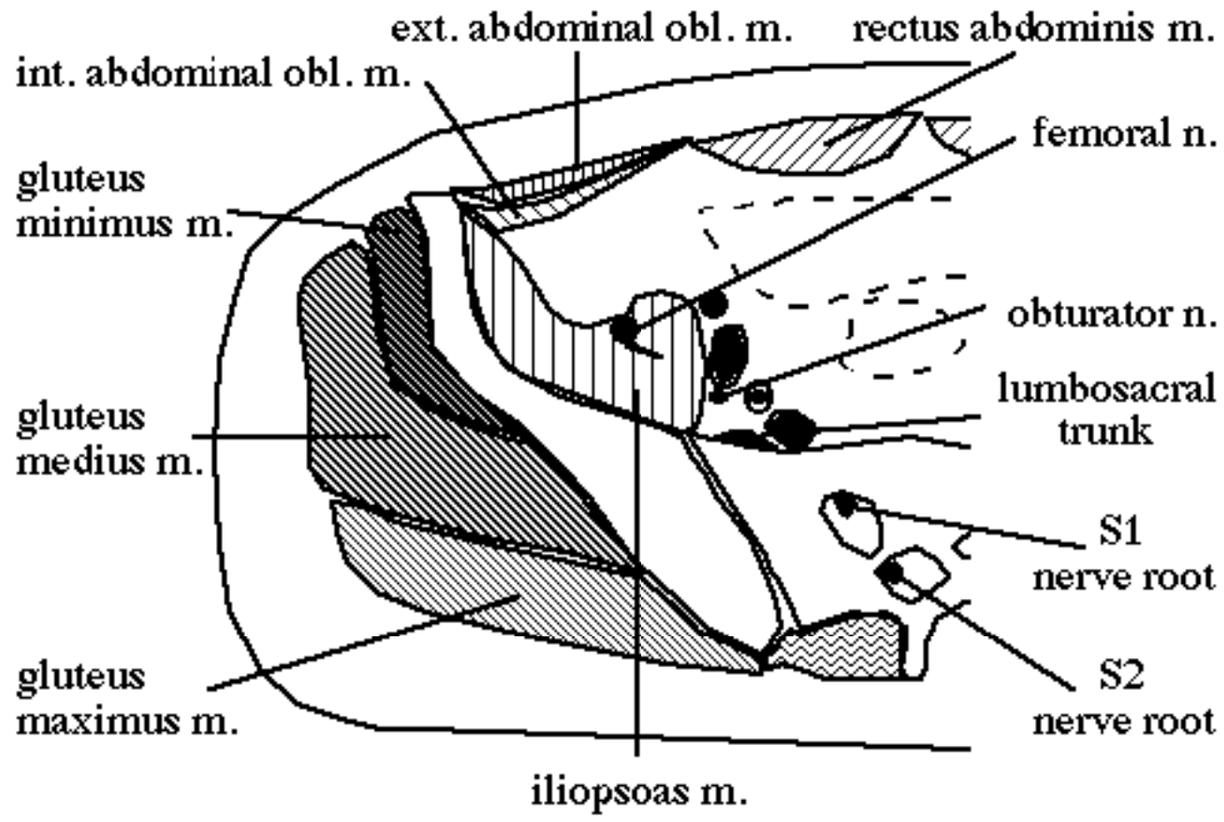
LEG



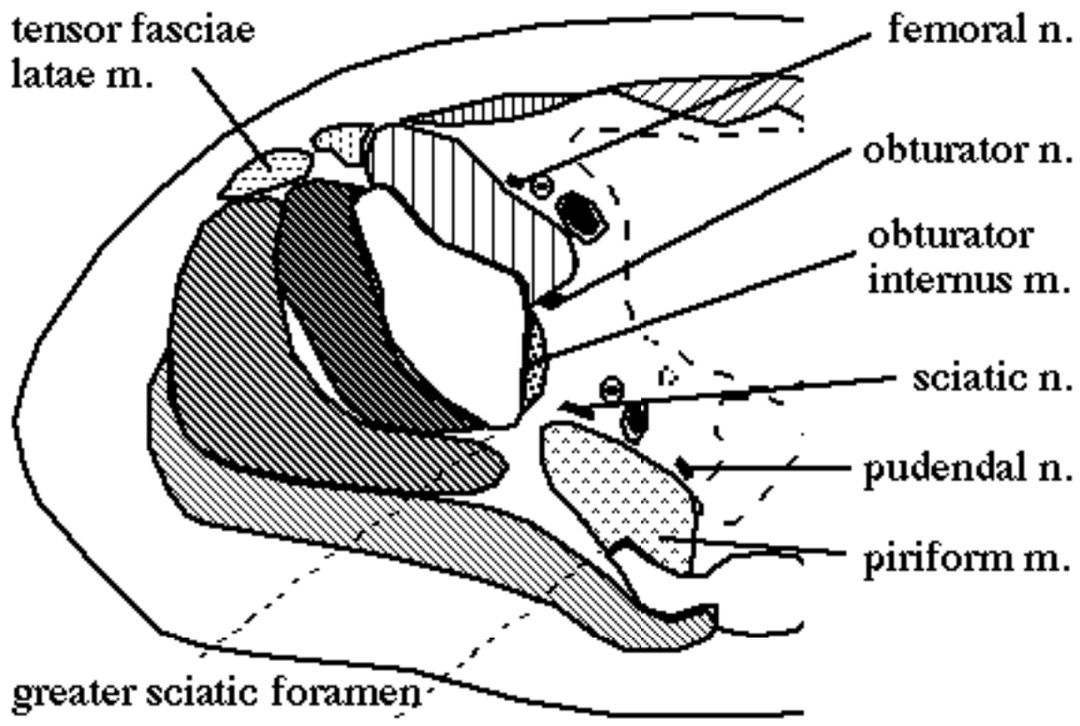
Cross-section Through L4-5



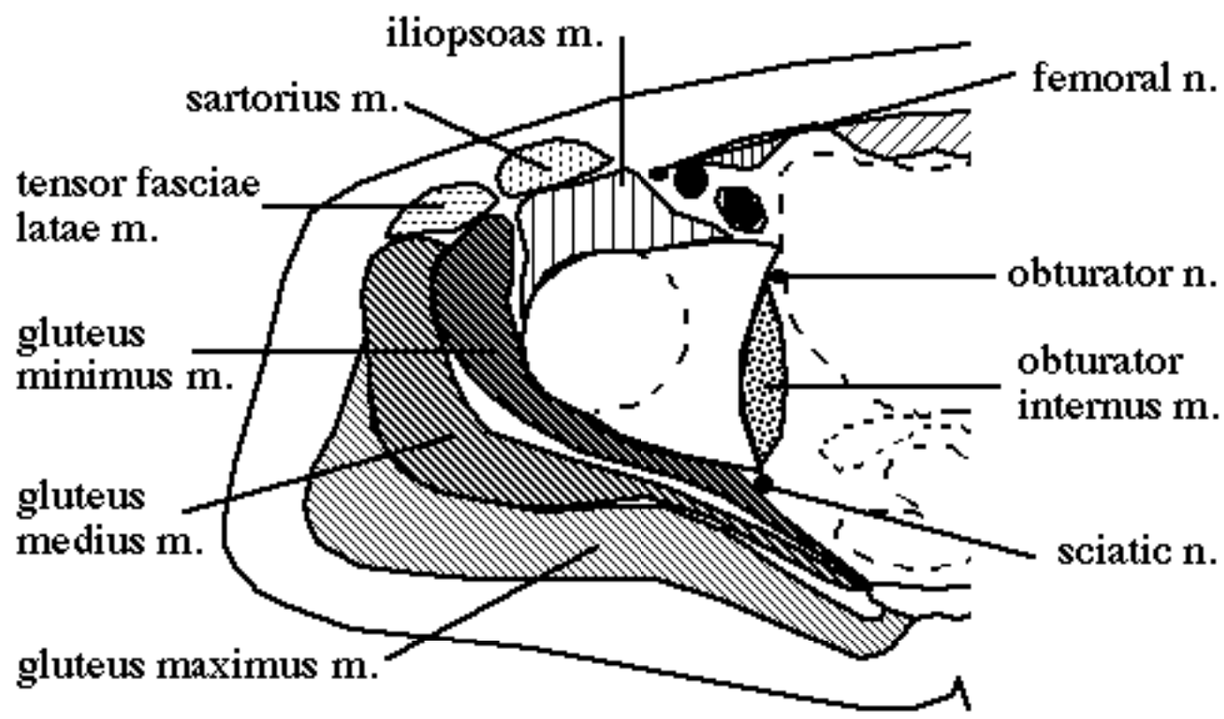
Cross-section ThroughL5-S1



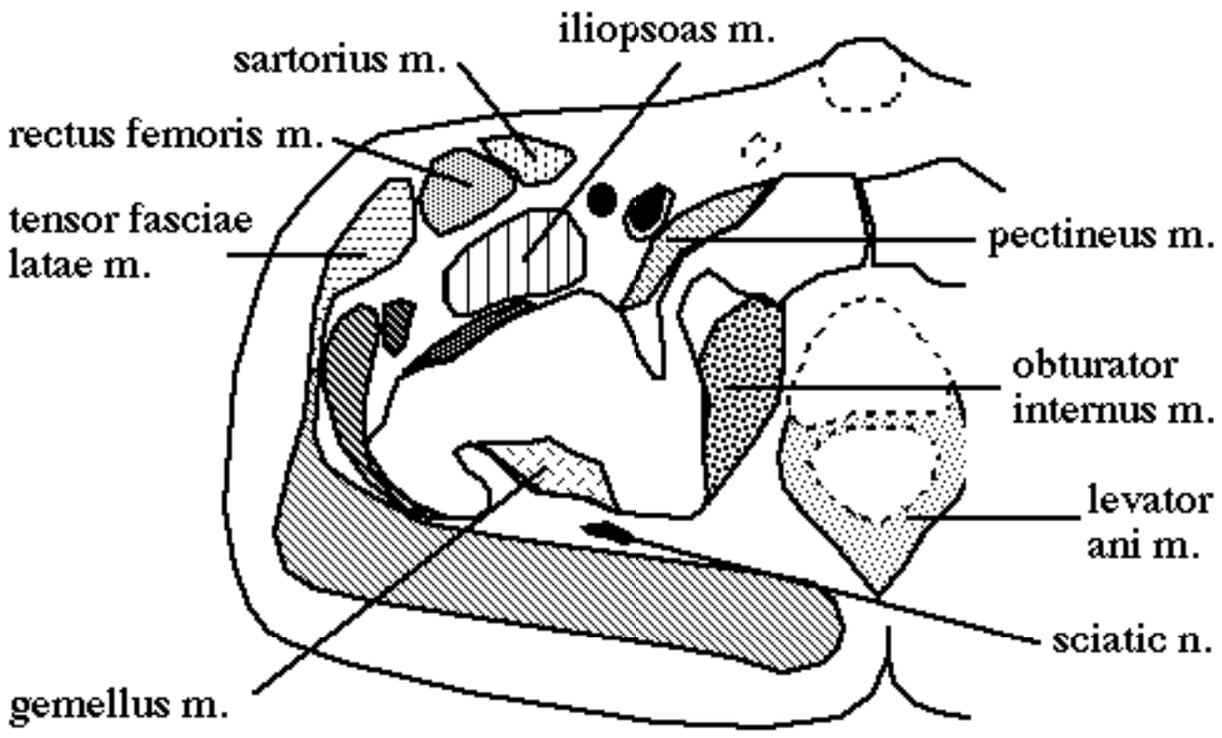
Cross-section Through S1-2



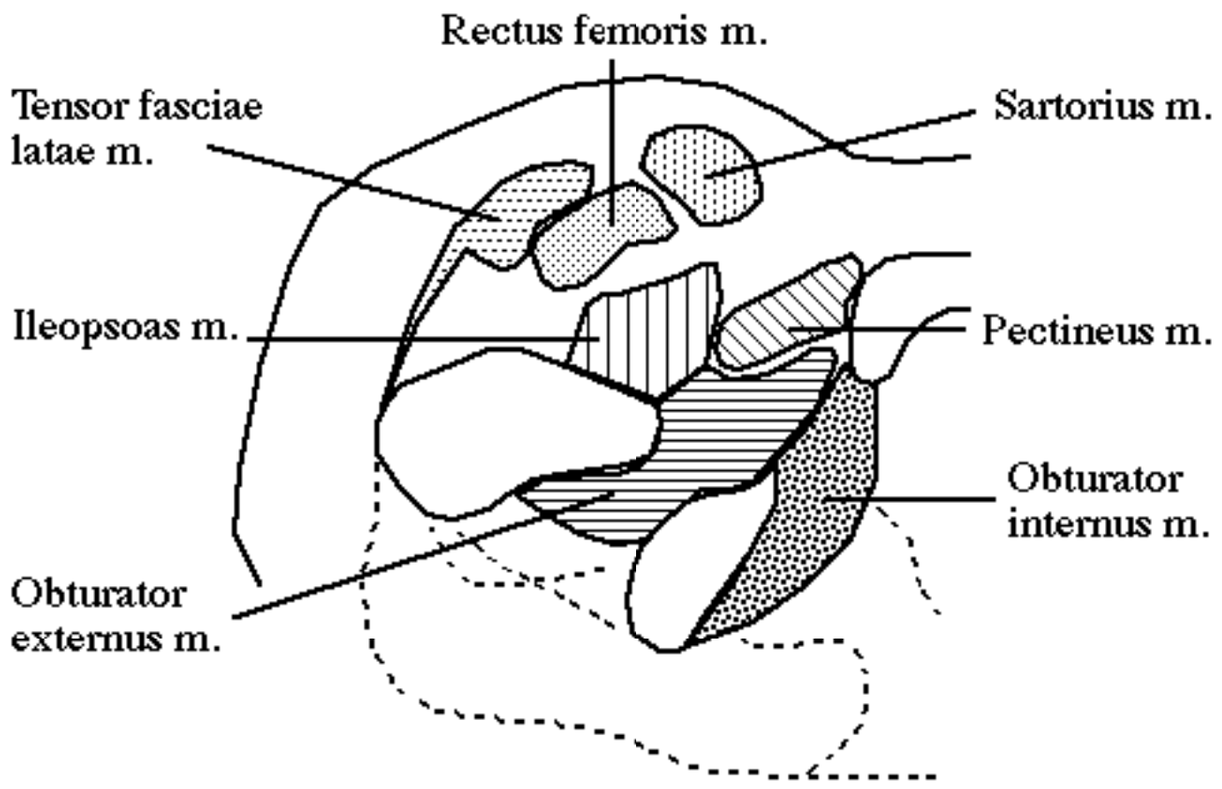
Cross-section Through S4



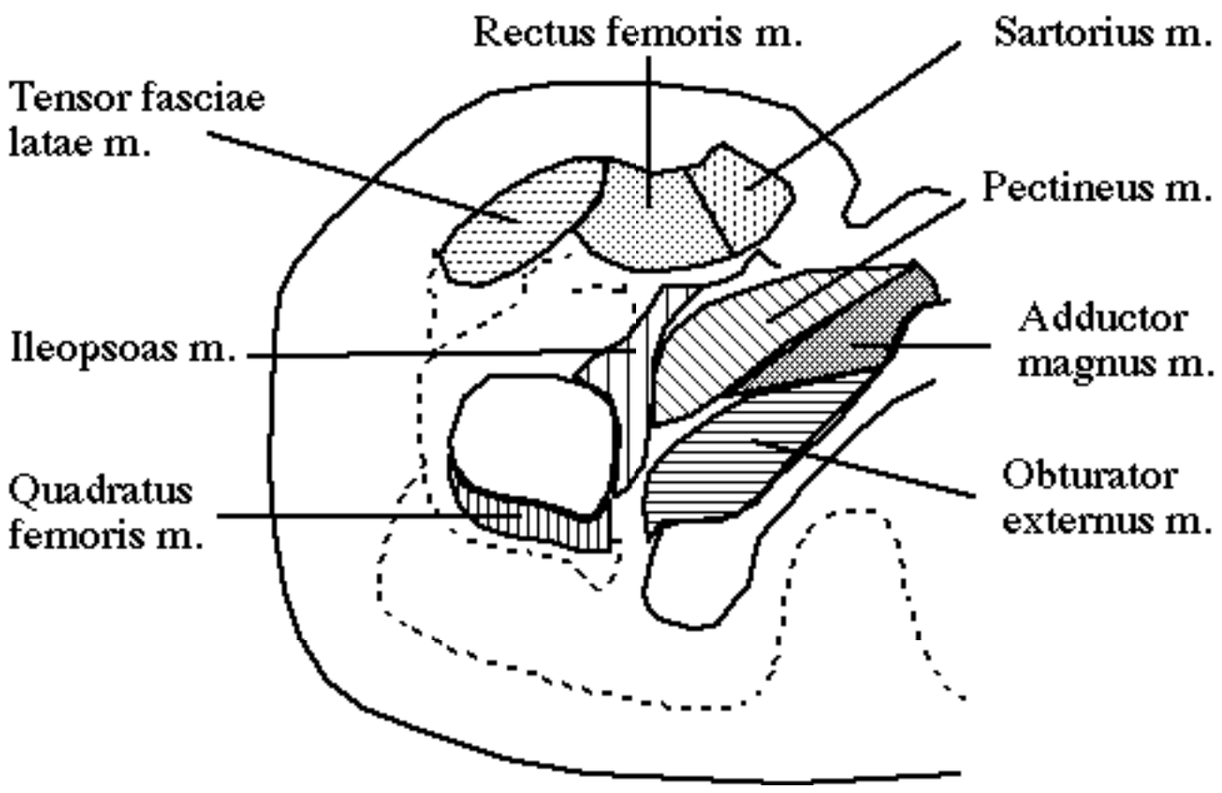
Cross-section Through Acetabular Roof



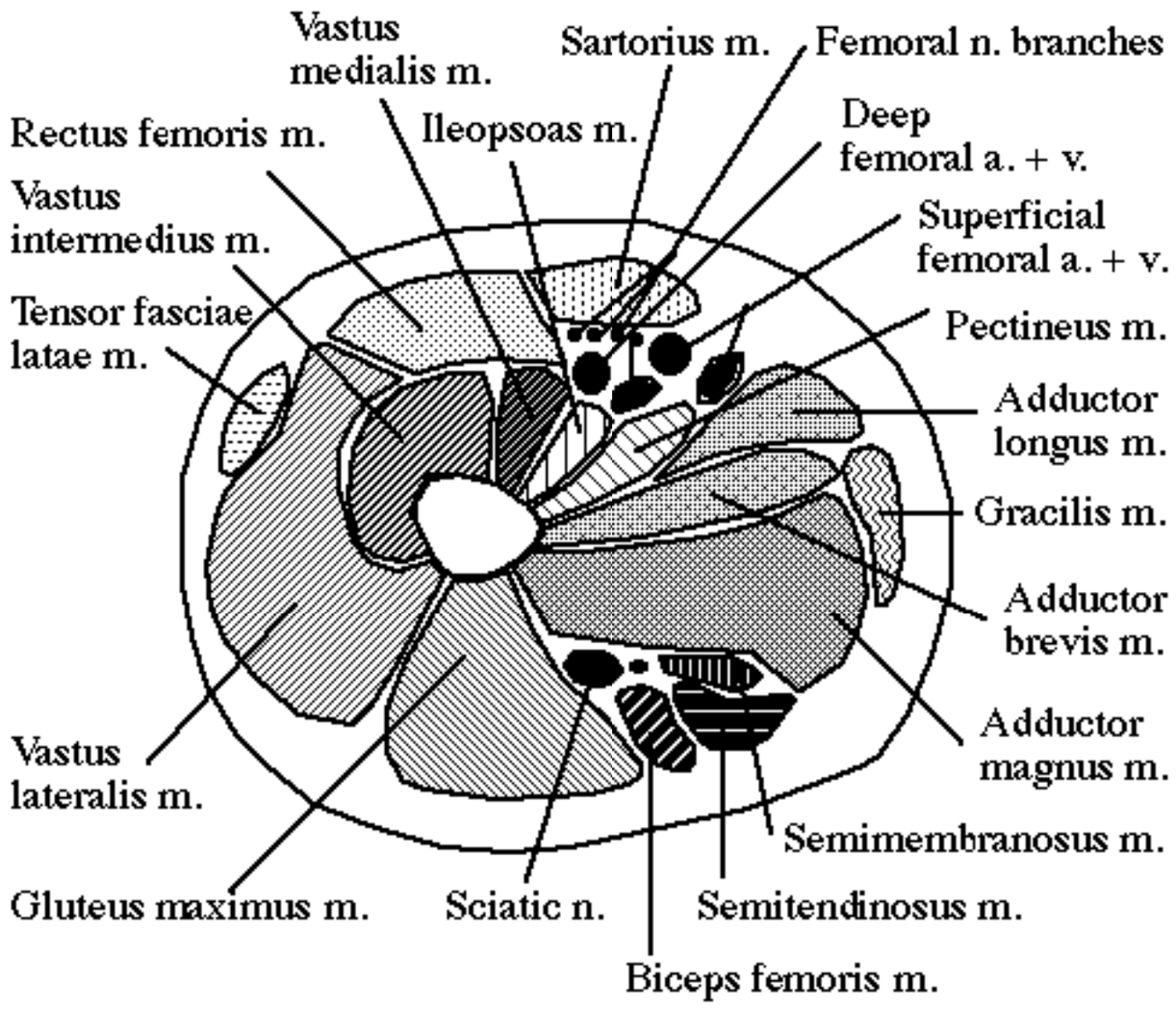
Cross-section Through Greater Trochanter



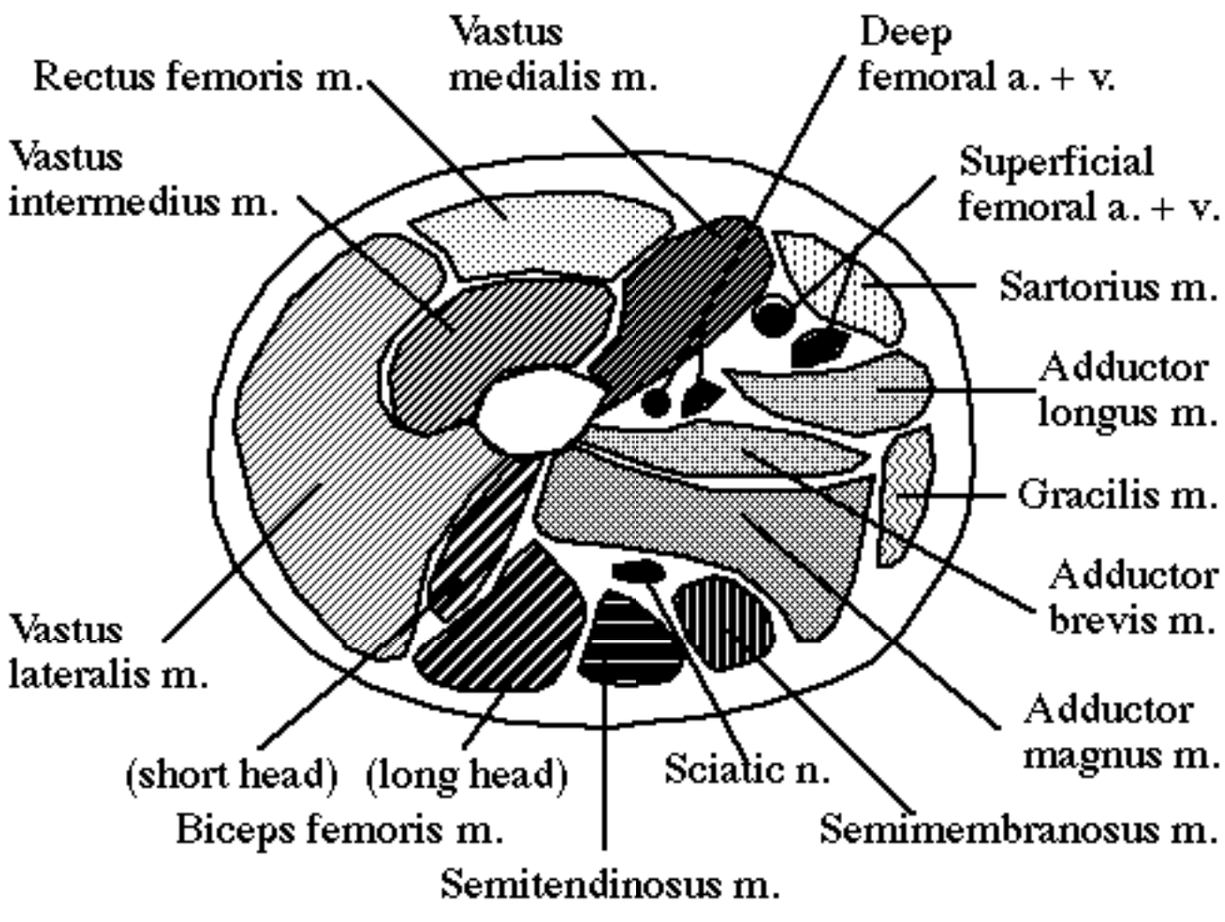
Cross-section Through Obturator Foramen



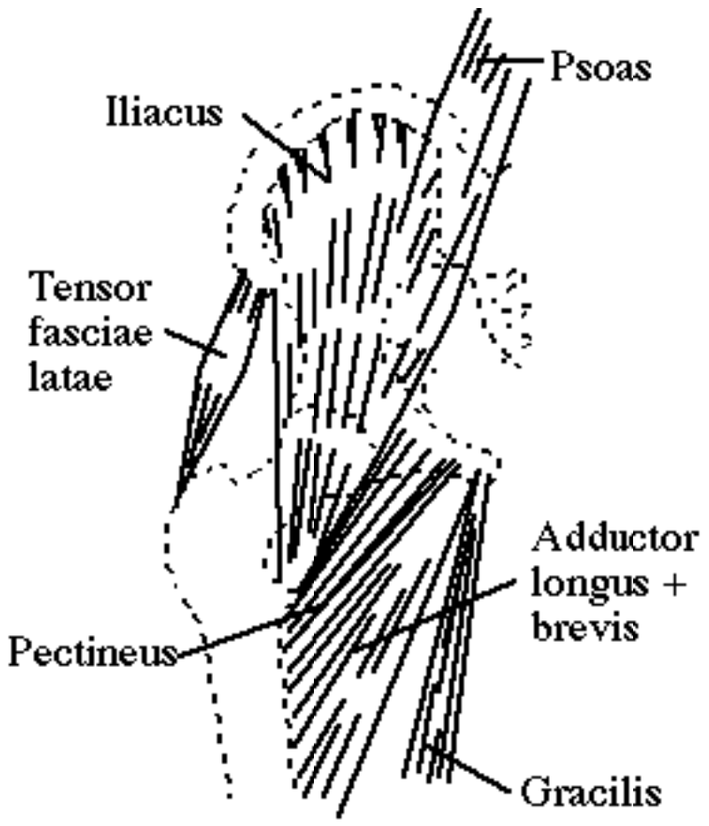
Cross-section Through Level of Minor Trochanter



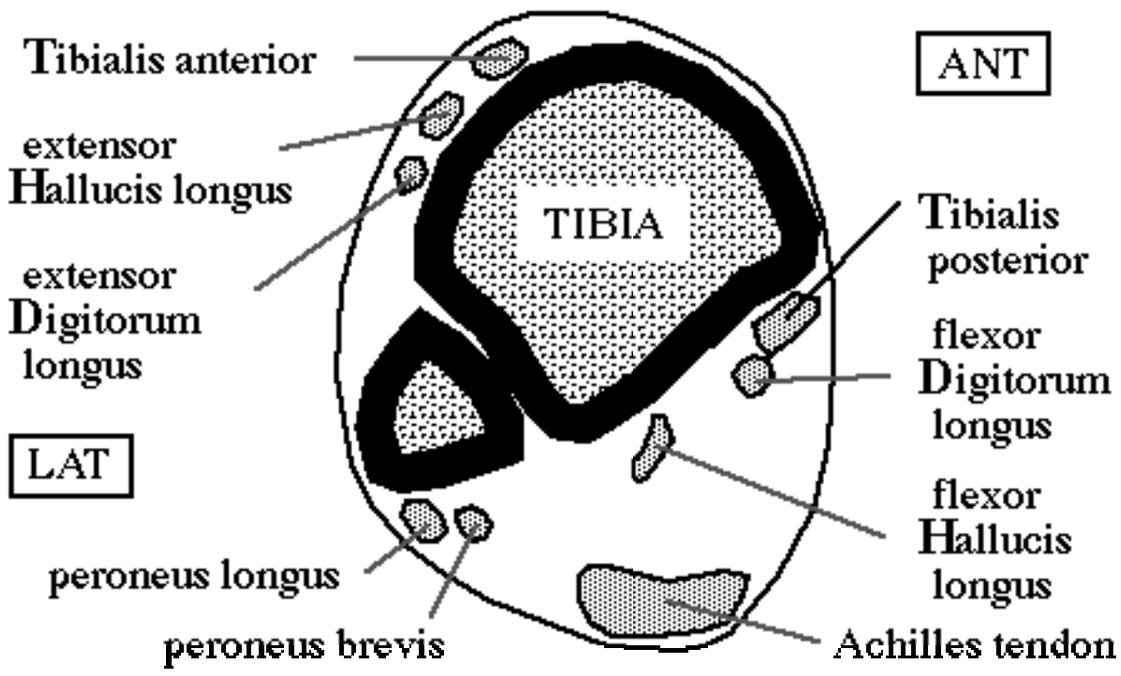
Cross-section Through Proximal Thigh



Cross-section Through Mid Thigh



Musculature About the Hip



Cross-section Through Distal Right Leg
mnemonic for posterior tendons: "**T**om, **D**ick and **H**arry"

Tibialis posterior**D**igitorum longus (flexor)**H**allucis longus (flexor)

Muscle Attachments of Thigh Anterior Cruciate Ligament (ACL) Posterior Cruciate Ligament (PCL) Medial (Tibial) Collateral Ligament Lateral (Fibular) Collateral Ligament

---

Notes:

Muscle Attachments of Thigh

Name of muscleOriginInsertion

Gracilisinferior pubic ramuspes anserinus**Semimembranosus**ischial tuberositymedial tibial condyle**Semitendinosus**ischial tuberositypes anserinus**Biceps femoris** - long headischial tuberosityfibular head- short headlateral linea asperafibular head**Adductor** - longussuperior pubic ramusmedial linea aspera- magnusinferior pubic ramusmedial linea aspera**Sartorius**anterior superior iliac spinepes anserinus**Quadriceps** - rectusanterior inferior iliac spinepatellar tendon- vastus lateralisgreater trochanterpatellar tendon- vastus medialismedial intertrochanteric linepatellar tendon**Iliopsoas** - iliacusiliumlesser trochanter- psoaslumbar spinelesser trochanter**Tensor fasciae latae**anterior superior iliac spineanterolateral tibia **Cruciate ligaments**

⚱Both cruciate ligaments are intracapsular but extrasynovial!

Notes:

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fourth edition

Anterior Cruciate Lignament (ACL) Origin:inner face of lateral femoral condyleInsertion:noncartilaginous region of anterior aspect of intercondylar eminence of tibiaAnatomy:several distinct bundles of fibers(1) posterior bulk = spiraling together at femoral origin (2) anteromedial bundle diverging at tibial insertion √ thin solid taut dark band (sagittal MR with knee in extension) almost parallel to intercondylar roof(= Blumensaat line) √ thin hypointense band parallel to inner aspect of lateral femoral condyle + fanlike configuration toward tibial spine (coronal MR) √ thin ovoid hypointense band proximally, elliptical configuration distally with higher intensity (axial MR) √ greater signal intensity than posterior cruciate ligament (due to anatomy)

Notes:

Posterior Cruciate Ligament (PCL) Origin:in a depression posterior to intercondylar region of tibia below joint surface Insertion:most distal + anterior aspect of inner face of medial femoral condyle√ thick dark band slightly posteriorly convex (arclike course on sagittal MR with knee in extension)√ medial to ACL (coronal MR)
Collateral ligaments of knee joint

Notes:

Medial (Tibial) Collateral Ligament Origin:just distal to adductor tubercle of femurInsertion:anteromedial face of tibiadistal to level of tibial tubercle about 5 cm below joint line(a)deep portion:-meniscofemoral ligament-meniscotibial ligaments(b)superficial portion-vertical band from femoral epicondyle to pes anserinus-posterior oblique ligament = posterior oblique band from femoral epicondyle to semimembranosus tendon deep and superficial dark bands separated by a thin bursa + fatty tissue (on coronal MR)

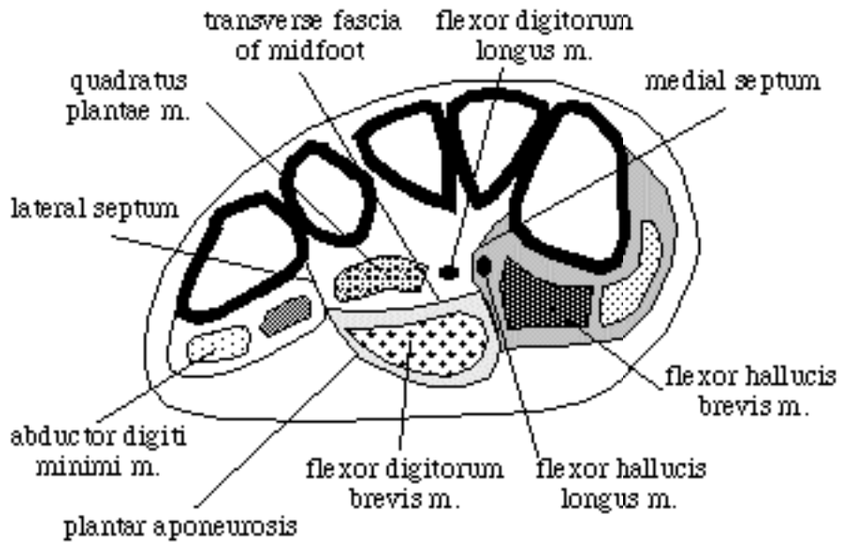
Notes:

Lateral (Fibular) Collateral Ligament Origin:lateral aspect of lateral femoral condyleInsertion:styloid process of fibular head\ bicipital tendon + iliotibial band join lateral collateral ligament

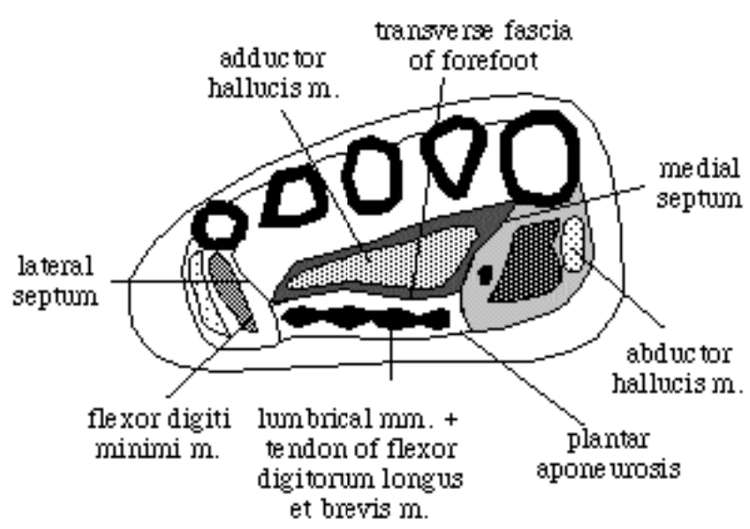
Notes:

FOOT AND ANKLE

Plantar Compartments of the Foot



Plantar Compartments of the Midfoot



Plantar Compartments of the Forefoot

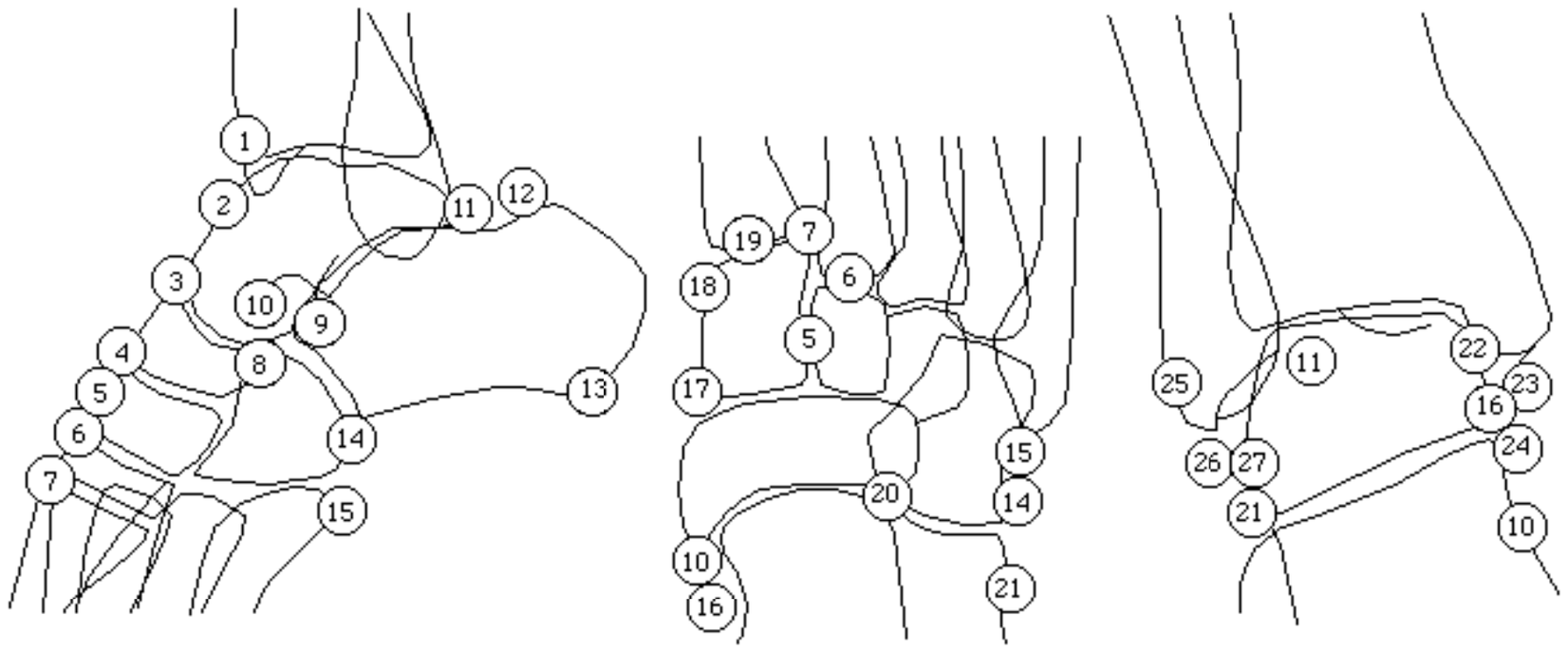
Medial compartment = bordered by medial septum (extending from plantar aponeurosis to navicular bone, medial cuneiform bone, and lateral border of plantar surface of 1st metatarsal bone);
contains abductor hallucis m. + flexor hallucis brevis m. + flexor hallucis longus tendon

Lateral compartment = bordered by lateral septum (extending from plantar aponeurosis to medial surface of 5th metatarsal bone);
contains abductor m. + short flexor m. + opponens m. of 5th toe

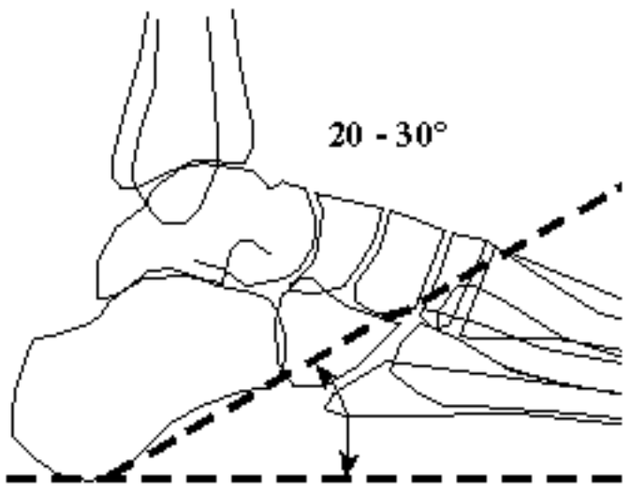
Central compartment = bordered by medial + lateral septa; communicates directly with posterior compartment of calf;
subdivided by horizontal septa: adductor hallucis m. separated from quadratus plantae m.
contains flexor digitorum brevis m. + flexor digitorum longus tendon + quadratus plantae m. + lumbricales mm.. + adductor hallucis m.

Deep subcompartment = bordered by transverse fascia of forefoot; separated from quadratus plantae m.; contains adductor hallucis m.

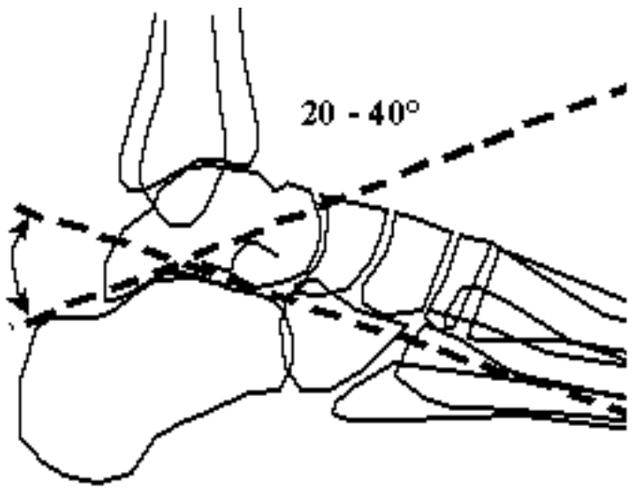
Accessory Ossicles of the Foot and Ankle



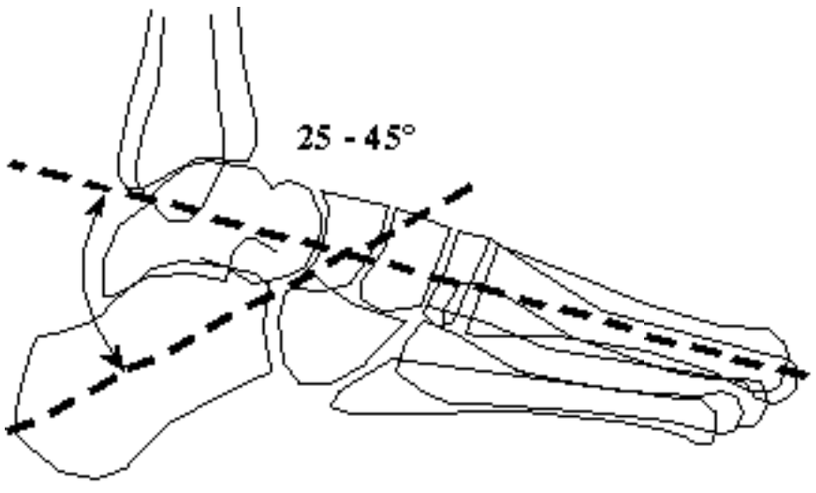
1 Os talotibiale
2 Os supratalare
3 Os supranaviculare
4 Os infranaviculare
5 Os intercuneiforme
6 Os cuneometatarsale II dorsale
7 Os intermetatarsale
8 Secondary cuboid
9 Calcaneus secundarius
10 Os tibiale externum
11 Trigonum
12 Os accessorium supracalcaneum
13 Os subcalcis
14 Os peroneum
15 Os vesalianum
16 Talus accessorius
17 Os cuneonaviculare mediale
18 Sesamum tibiale anterius
19 Os cuneometatarsale I plantare
20 Cuboides secundarium
21 Os trochleare calcanei
22 Sesamoid talus - int. malleolus
23 Os subtibiale
24 Os sustentaculi
25 Os retinaculi
26 Os subfibulare
27 Talus secundarius



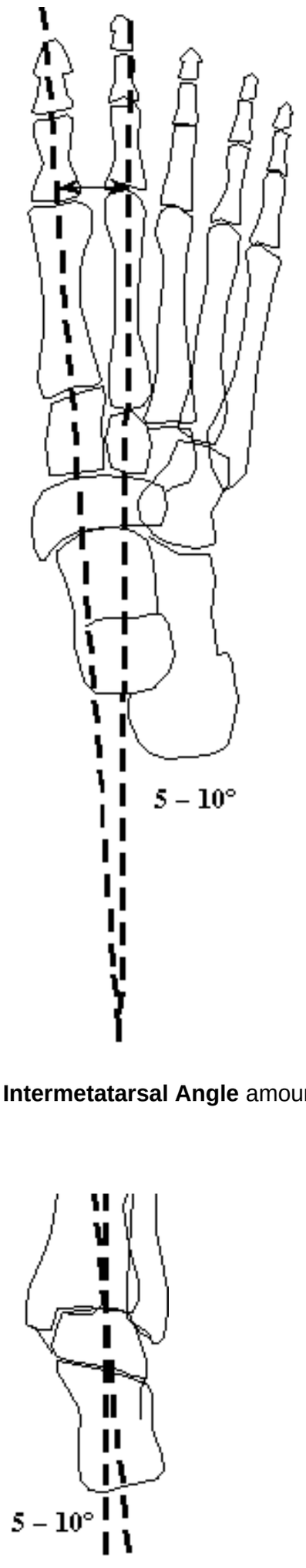
Calcaneal Pitch = Calcaneal Inclination Angle = determines longitudinal arch of foot; angle between line drawn along the inferior border of calcaneus connecting the anterior and posterior prominences + line representing the horizontal surface



Boehler Angle = angle between first line drawn from posterosuperior prominence of calcaneus anteriorly to sustentaculum tali + second line drawn from anterosuperior prominence posteriorly to sustentaculum tali; measures integrity of calcaneus

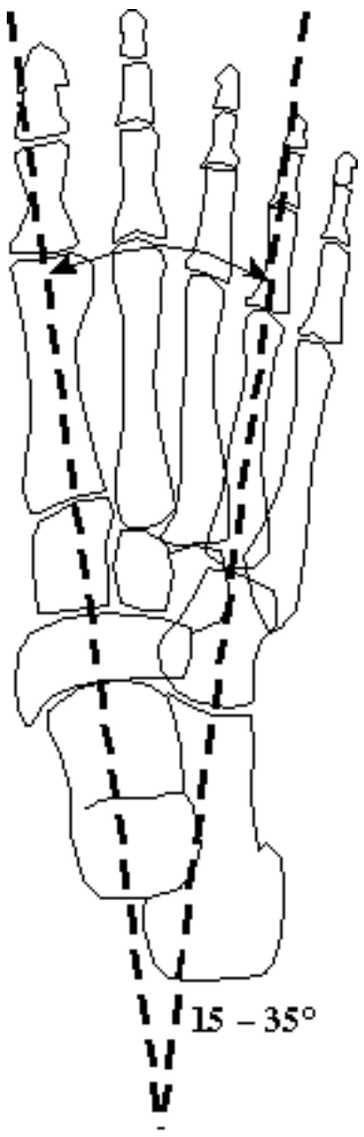


Talocalcaneal Angle on LAT View = angle between lines drawn through mid-transverse planes of talus + calcaneus; the midtalar line parallels the longitudinal axis of the first metatarsal

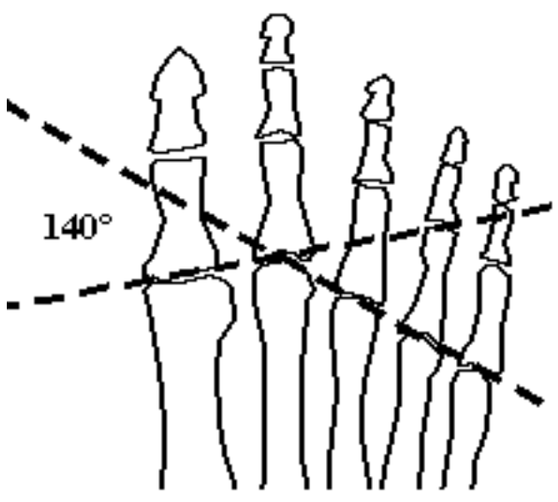


Intermetatarsal Angle amount that 1st + 2nd metatarsals diverge from each other

Heel Valgus cannot be measured directly on radiographs but inferred from the talocalcaneal angle and estimated on coronal CT sections



Talocalcaneal Angle on AP View = KITE ANGLE = the midtalar and midcalcaneal lines parallel the 1st + 4th metatarsals; angle is greater in infants



Angle of Metatarsal Heads = obtuse angle formed by lines tangential to metatarsal heads

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Notes:

ACHONDROGENESIS
=autosomal recessive lethal chondrodystrophy characterized by extreme micromelia, short trunk, large craniumTRIAD:(1)severe short-limb dwarfism(2)lack of vertebral calcification(3)large head with normal / decreased calvarial ossification*Birth prevalence:*2.3:100,000*Path:*disorganization of cartilageA.TYPE I = Parenti-Fraccaro disease=defective enchondral + membranous ossification√ complete lack of ossification of calvarium + spine + pelvis√ absent sacrum + pubic bone√ extremely short long bones without bowing, especially femur, radius, ulna√ thin ribs with multiple fractures (frequent)B.TYPE II = Langer-Saldino disease=defective enchondral ossification only√ good ossification of skull vault√ nonossification of lower lumbar vertebrae + sacrum√ short + stubby horizontal ribs without fractures ● often subcutaneous edema√ irregular flared metaphyses (esp. humerus)√ short trunk with narrow chest + protruding abdomen√ redundant soft tissues√ polyhydramnios (common)√ increase in HC:AC ratio*Prognosis:*lethal often in utero / within few hours or days after birth (respiratory failure)*DDx:*often confused with thanatophoric dwarfism

Notes:

Heterozygous Achondroplasia ◊Prototype of rhizomelic dwarfism!=autosomal dominant / sporadic (80%) disease with quantitatively defective endochondral bone formation; related to advanced paternal age; epiphyseal maturation + ossification unaffected*Incidence:*1:26,000-66,000 births, most common of lethal bone dysplasias; M < F ● normal intelligence + motor function ● neurologic defects ● classically circus dwarfs@Skull ● flat nasal bridge (hypoplastic base of skull) ● brachycephaly with enlarged bulging forehead (nonprogressive hydrocephalus) ● relative prognathism√ large calvarium with frontal bossing√ broad mandible√ shortened base of skull + small foramen magnum√ communicating hydrocephalus caused by constricted basicranium + foramen magnum (obstruction of basal cisterns + aqueduct)@Chest & spine ● protuberant abdomen ● prominent buttocks√ squaring of inferior scapular margin√ narrow chest with short anteriorly flared ribs√ hypoplastic bullet- / wedge-shaped vertebra= rounded anterior beaking of vertebra in upper lumbar spine (DDx: Hurler disease)√ posterior vertebral scalloping√ scoliosis√ spinal stenosis (ventrodorsal + interpediculate space) in lumbar spine√ laminar thickening√ bulging discs√ wide intervertebral foramina√ lumbar angular kyphosis (gibbus) + sacral lordosis @Pelvis ● rolling gait from backward tilt of pelvis and hip joints√ square-shaped flattened iliac bones with tombstone configuration ("champagne glass")√ lack of flaring of iliac wings√ horizontal acetabula (flat acetabular angle)√ small sacrosciatic notch @ Extremities ● short stubby limbs + fingers ● trident hand = separation of 2nd + 3rd digit and inability to approximate 3rd + 4th finger ● limited range of motion of elbow√ brachydactyly (short tubular bones of hand + feet), especially short proximal + middle phalanges√ "trumpet" appearance with short long bones and metaphyseal flaring (normal width of metaphysis)√ predominantly rhizomelic shortness of long bones (femur, humerus)√ short femoral necks√ limb bowing√ "ball-in-socket" epiphysis = broad V-shaped distal femoral metaphysis in which epiphysis is incorporated√ high position of fibular head (fibula less short)√ short ulna with thick proximal + slender distal end OB-US (diagnosable >21-27th week GA): √ shortening of proximal long bones: femur length <99th percentile between 21 and 27 weeks MA√ increased BPD, HC, HC:AC ratio√ decreased FL:BPD ratio√ normal mineralization, no fractures√ normal thorax + normal cardiothoracic ratio√ three-pronged (= trident) hand = 2nd + 3rd + 4th finger of similarly short length without completely approximating each other (= PATHOGNOMONIC)*Cx:*(1)Hydrocephalus + syringomyelia (small foramen magnum)(2)Recurrent ear infection (poorly developed facial bones)(3)Neurologic complications (compression of spinal cord, lower brain stem, cauda equina, nerve roots): apnea and sudden death(4)Crowded dentition + malocclusion*Prognosis:*long life*DDx:*various mucopolysaccharidoses

Notes:

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Homozygous Achondroplasia =hereditary autosomal dominant disease with severe features of achondroplasia (disproportionate limb shortening, more marked proximally than distally)*Risk:*marriage of two achondroplasts to each other√ large cranium with short base + small face√ flattened nose bridge√ short ribs with flared ends√ hypoplastic vertebral bodies√ decreased interpedicular distance√ short squared innominate bones√ flattened acetabular roof√ small sciatic notch√ short limb bones with flared metaphyses√ short, broad, widely spaced tubular bones of hand*Prognosis:*often stillborn; lethal in neonatal period (from respiratory failure)*DDx:*thanatophoric dysplasia

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Notes:

ACROCEPHALOSYNDACTYLY
=syndrome characterized by(1)increased height of skull vault due to generalized craniosynostosis (= acrocephaly, oxycephaly)(2)syndactyly of fingers / toes Type I:Apert syndrome = acrocephalosyndactylyType II: Vogt cephalosyndactylyType III:Acrocephalosyndactyly with asymmetry of skull + mild syndactylyType IV:Wardenburg typeType V:Pfeiffer type

Notes:

ACROOSTEOLYSIS, FAMILIAL
dominant inheritance *Age:*onset in 2nd decade; M:F = 3:1 ● sensory changes in hands + feet ● destruction of nails ● joint hypermobility ● swelling of plantar of foot with deep wide ulcer + ejection of bone fragments@Skull√ wormian bones√ craniosynostosis√ basilar impression√ protuberant occiput√ resorption of alveolar processes + loss of teeth@Spine√ spinal osteoporosis ± fracture√ kyphoscoliosis + progressive decrease in height

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ACROMEGALY
*Etiology:*excess growth hormone due to eosinophilic adenoma / hyperplasia ● gigantism in children (DDx: **Soto syndrome** of cerebral gigantism = large skull, mental retardation, cerebral atrophy, advanced bone age)√ osseous enlargement (phalangeal tufts, vertebrae)√ flared ends of long bone√ cystic changes in carpals, femoral trochanters√ osteoporosis@Hand ● spadelike hand√ widening of terminal tufts@Skull√ prognathism (= elongation of mandible) in few cases√ sellar enlargement + erosion√ enlargement of paranasal sinuses: large frontal sinuses (75%)√ calvarial hyperostosis (especially inner table)√ enlarged occipital protuberance@Vertebrae√ posterior scalloping in 30% (secondary to pressure of enlarged soft tissue)√ anterior new bone√ loss of disk space (weakening of cartilage)@Soft tissue√ heel pad >25 mm@ Joints √ premature osteoarthritis (commonly knees)

Notes:

ACTINOMYCOSIS
*Organism:*Actinomyces israelii, Gram-positive anaerobic pleomorphic small bacterium with proteolytic activity, superficially resembling the morphology of a hyphal fungus; closely related to mycobacteria*Histo:*mycelial form in tissue; rod-shaped bacterial form normally inhabiting oropharynx (dental caries, gingival margins, tonsillar crypts) + GI tract*Predisposed:*individuals with very poor dental hygiene, immunosuppressed patientsLocation:mandibulofacial > intestinal > lung*Types:* (1)Mandibulo- / cervicofacial actinomycosis (common)*Cause:*poor oral hygiene ● draining cutaneous sinuses ● "sulfur granules" in sputum / exudate = colonies of organisms arranged in circular fashion = mycelial clumps with thin hyphae 1-2 mm in diameter√ osteomyelitis of mandible (most frequent bone involved) with destruction of mandible around tooth socket√ no new-bone formation√ spread to soft tissues at angle of jaw + into neck(2)Abdominal / ileocecal actinomycosis (60%)*Cause:*rupture / surgery of appendix; IUD useLocation:initially localized to cecum / appendix ● fever, leukocytosis, mild anemia ● weight loss, nausea, vomiting, pain ● chronic sinus in groin√ fold thickening + ulcerations (resembling Crohn disease)√ rupture of abdominal viscus (usually appendix)√ fistula formation√ abscess in liver (15%), retroperitoneum, psoas muscle (containing yellow "sulfur granules"= 1-2 mm colony of gram-positive bacilli) (3)Pleuropulmonary actinomycosis*Cause:*hematogenous spread / inhalation@Lung ● draining chest wall sinuses (spread through fascial planes)√ consolidation extending across interlobar fissures (acute airspace pneumonia rare)√ cavitary lesion (abscess) √ pleuritis + empyema@ Vertebra + ribs √ destruction of vertebra with preservation of disk + small paravertebral abscess without calcification (DDx to tuberculosis: disk destroyed, large abscess with calcium)√ thickening of cervical vertebrae around margins√ destruction / thickening of ribs@ Tubular bones of hands √ destructive lesion of mottled permeating type√ cartilage destruction + subarticular erosive defects in joints (simulating TB) *Rx:*surgical débridement + penicillin

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ADAMANTINOMA
=(MALIGNANT) ANGIOBLASTOMA = locally aggressive / malignant lesion*Histo:*pseudoepithelial cell masses with peripheral columnar cells in a palisade pattern with varying amounts of fibrous stroma; areas of squamous / tubular / alveolar / vessel transformation; prominent vascularity; resembles ameloblastoma of the jaw*Age:*25-50 years, commonest in 3rd-4th decade ● frequently history of trauma ● local swelling ± painLocation:middle 1/3 of tibia (90%), fibula, ulna, carpals, metacarpals, humerus, shaft of femur√ eccentric round osteolytic lesion with sclerotic margin, may have additional foci in continuity with major lesion (CHARACTERISTIC)√ may show mottled density√ bone expansion frequent√ often multiple*Prognosis:*tendency to recur after local excision; after several recurrences pulmonary metastases may develop*DDx:*fibrous dysplasia (possibly related)

Notes:

AINHUM DISEASE
=DACTYLOLYSIS SPONTANEA[ainhum = fissure, saw, sword] *Etiology:*unknown*Histo:*hyperkeratotic epidermis with fibrotic thickening of collagen bundles below; chronic lymphocytic inflammatory reaction may be present; arterial walls may be thickened with narrowed vessel lumina*Incidence:*up to 2%*Age:*usually in males in 4th + 5th decades; Blacks (West Africa) + their American descendants; M > F ● deep soft-tissue groove forming on medial aspect of plantar surface of proximal phalanx with edema distally ● painful ulceration may developLocation:mostly 5th / 4th toe (rarely finger); near interphalangeal joint; mostly bilateral√ sharply demarcated progressive bone resorption of distal / middle phalanx with tapering of proximal phalanx to complete autoamputation (after an average of 5 years)√ osteoporosis*Rx:*early surgical resection of groove with Z plasty*DDx:*(1)Neuropathic disorders (diabetes, leprosy, syphilis)(2)Trauma (burns, frostbite)(3)Acroosteolysis from inflammatory arthritis, infection, polyvinyl chloride exposure(4)Congenitally constricting bands in amniotic band syndrome

Notes:

AMYLOIDOSIS
=accumulation + infiltration of a chemically diverse group of protein polysaccharides in body tissues; tends to form around capillaries + endothelial cells of larger blood vessels causing ultimately vascular obliteration with infarction*Path:*stains with Congo red ● bone pain ● periarticular rubbery soft-tissue swelling + stiffness (shoulders, hips, fingers) ● Bence-Jones protein (without myeloma)√ periarticular soft-tissue swelling (amyloid deposited in synovium, joint capsule, tendons, ligaments) ± extrinsic osseous erosion√ subluxation of proximal humerus + femoral neck√ osteoporosis√ coarse trabecular pattern (DDx: sarcoidosis)√ focal medullary lytic lesion with endosteal scalloping (± secondary invasion + erosion of articular bone)√ pathologic fractures may occur (vertebral fracture)

Notes:

ANEURYSMAL BONE CYST
=expansile lesion of bone containing thin-walled blood-filled cystic cavities; name derived from roentgen appearance*Etiology:* (a)primary ABC (65-99%)local circulatory disturbance as a result of trauma(b)secondary ABC (1-35%)arising in preexisting bone tumor causing venous obstruction / arteriovenous fistula: giant cell tumor (39%), osteoblastoma, chondroblastoma, angioma, telangiectatic osteosarcoma, solitary bone cyst, fibrous dysplasia, xanthoma, chondromyxoid fibroma, nonossifying fibroma, metastatic carcinoma*Histo:*"intraosseous arteriovenous malformation" with honeycombed spaces filled with blood + lined by granulation tissue / osteoid; areas of free hemorrhage; sometimes multinucleated giant cells; solid component predominates in 5-7%*Types:* 1.INTRAOSSEOUS ABC=primary cystic / telangiectatic tumor of giant cell family, originating in bone marrow cavity, slow expansion of cortex; rarely related to history of trauma2.EXTRAOSSEOUS ABC=posttraumatic hemorrhagic cyst; originating on surface of bones, erosion through cortex into marrow*Age:*peak age 16 years (range 10-30 years); in 75% <20 years; F > M ● pain of relatively acute onset with rapid increase of severity over 6-12 weeks ● ± history of trauma ● neurologic signs (radiculopathy to quadriplegia) if in spineLocation:(a)spine (12-30%) with slight predilection for posterior elements; thoracic > lumbar > cervical spine (22%); involvement of vertebral body (40-90%); may involve two contiguous vertebrae (25%)(b)long bones: eccentric in metaphysis of femur, tibia, humerus, fibula; pelvis√ purely lytic eccentric radiolucency√ aggressive expansile ballooning lesion of "soap-bubble" pattern + thin internal trabeculations√ rapid progression within 6 weeks to 3 months√ sclerotic inner portion√ almost invisible thin cortex (CT shows integrity)√ tumor respects epiphyseal plate√ no periosteal reaction (except when fractured)CT: √ "blood-filled sponge" = fluid-fluid levels due to blood sedimentation (in 10-35%)MR: √ multiple cysts of different signal intensity representing different stages of blood by-products√ low-signal intensity rim = intact thickened periosteal membraneNUC: √ "doughnut sign" = peripheral increased uptake (64%)Angio: √ hypervascularity in lesion periphery (in 75%) *Prognosis:*20-30% recurrence rate*Rx:*complete resection; embolotherapy; radiation therapy (subsequent sarcoma possible)*Cx:*(1)pathologic fracture (frequent)(2)extradural block with paraplegia*DDx:*(1)Giant cell tumor (particularly in spine)(2)Hemorrhagic cyst (end of bone / epiphysis, not expansile)(3)Enchondroma(4)Metastasis (renal cell + thyroid carcinoma)(5)Plasmacytoma(6)Chondro- and fibrosarcoma(7)Fibrous dysplasia(8)Hemophilic pseudotumor(9)Hydatid cyst

Notes:

ANGIOMATOSIS
= diffuse infiltration of bone / soft tissue by hemangiomatous / lymphangiomatous lesions *Age:*first 3 decades of life*May be associated with:* chylothorax, chyloperitoneum, lymphedema, hepatosplenomegaly, cystic hygroma A.OSSEOUS ANGIOMATOSIS (30-40%) ● indolent courseLocation:femur > ribs > spine > pelvis > humerus > scapula > other long bones > clavicle√ osteolysis with honeycomb / latticework ("hole-within-hole") appearance√ may occur on both sides of joint*DDx:*solitary osseous hemangiomaB.CYSTIC ANGIOMATOSIS=extensive involvement of bone*Histo:*endothelial lined cysts in bone*Age:*peak 10-15 years; range of 3 months to 55 yearsLocation:long bones, skull, flat bones√ multiple osteolytic metaphyseal lesions of 1-2 mm to several cm with fine sclerotic margins + relative sparing of medullary cavity√ may show overgrowth of long bone√ endosteal thickening√ sometimes associated with soft-tissue mass ± phleboliths√ chylous pleural effusion suggests fatal prognosis*DDx:*(other polyostotic diseases as) histiocytosis X, fibrous dysplasia, metastases, Gaucher disease, congenital fibromatosis, Maffucci syndrome, neurofibromatosis, enchondromatosisC.SOFT-TISSUE ANGIOMATOSIS (60-70%)=VISCERAL ANGIOMATOSIS ● poor prognosisD.ANGIOMATOUS SYNDROMES1.Maffucci syndrome2.Osler-Weber-Rendu syndrome3.Klippel-Trénaunay-Weber disease4.Kasabach-Merritt syndrome5.Gorham disease

Notes:

ANGIOSARCOMA
= aggressive vascular malignancy with frequent local recurrence + distant metastasis *Histo:*vascular channels surrounded by hemangiomatous / lymphomatous cellular elements with high degree of anaplasia*Age:*M:F = 2:1*Associated with:* **Stewart-Treves syndrome**
=angiosarcoma with chronic lymphedema developing in postmastectomy patientsLocation:skin (in 33%); soft tissue (in 24%);bone (in 6%): tibia (23%), femur (18%), humerus (13%), pelvis (7%)*DDx:*hemangioendothelioma, hemangiopericytoma

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Notes:

ANKYLOSING SPONDYLITIS

= chronic inflammatory disease of unknown etiology primarily affecting spine *Age:*15-35 years; M:F = 4:1-10:1;Caucasians:Blacks = 3:1*Associated with:*(1)ulcerative colitis, regional enteritis(2)iritis in 25%(3)aortic insufficiency + atrioventricular conduction defect ● HLA-B 27 positive in 96% ● insidious onset of low back pain + stiffnessLocation:axial skeleton; HALLMARK is sacroiliac joint involvement; peripheral skeleton (10-20%)√ temporomandibular joint space narrowing, erosions, osteophytosis@Hand (30%)Target area:MCP, PIP, DIP√ exuberant osseous proliferation√ osteoporosis, joint space narrowing, osseous erosions (deformities less striking than in rheumatoid arthritis)@Sacroiliac / symphysis pubis√ initially sclerosis of joint margins primarily on iliac side (bilateral + symmetric late in disease, may be unilateral + asymmetric early in disease)√ later irregularities + widening of joint (cartilage destruction)√ bony fusion@ Spine √ straightening / squaring of anterior vertebral margins = osteitis of anterior corners√ reactive sclerosis of corners of vertebral bodies√ asymmetric erosions of laminar + spinous process at level of lumbar spine√ marginal syndesmophyte formation = thin vertical radiodense spicules bridging the vertebral bodies= ossification of outer fibers of annulus fibrosus (NOT anterior longitudinal ligament) √ "trolley-track" sign on AP view = central line of ossification (supraspinous + interspinous ligaments) with two lateral lines of ossification (apophyseal joints)√ "bamboo" spine on AP view = undulating contour due to syndesmophytes; prone to fracture resulting in pseudarthrosis√ diskal ballooning ± diskal calcification√ apophyseal + costovertebral ankylosis√ periostitic "whiskering": ischial tuberosity, iliac crest, ischiopubic rami, greater femoral trochanter, external occipital protuberance, calcaneus√ dorsal arachnoid diverticula in lumbar spine with erosion of posterior elements (Cx: cauda equina syndrome)√ atlantoaxial subluxation@ Chest √ bilateral upper lobe pulmonary fibrosis (1%) with upward retraction of hila (DDx: tuberculosis)@Cardiovascular1.Aortitis (5%) of ascending aorta ± aortic valve insufficiency*Prognosis:*20% progress to significant disability; occasionally death from cervical spine fracture / aortitis*DDx:*(1)Reiter syndrome (unilateral asymmetric SI joint involvement, paravertebral ossifications)(2)Psoriatic arthritis (unilateral asymmetric SI joint involvement, paravertebral ossifications)(3)Inflammatory bowel disease

Notes:

ANTERIOR TIBIAL BOWING
=WEISMANN-NETTER SYNDROME = congenital painless nonprogressive bilateral anterior leg bowing*Age:*beginning in early childhood ● may be accompanied by mental retardation, goiter, anemia√ anterior bowing of tibia + fibula, bilaterally, symmetrically at middiaphysis√ thickening of posterior tibial + fibular cortices√ minor radioulnar bowing√ kyphoscoliosis√ extensive dural calcification*DDx:*Luetic saber shin (bowing at lower end of tibia + anterior cortical thickening)

Notes:

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APERT SYNDROME

@Skull√ oxycephalic skull + flat occiput√ hypertelorism + bilateral exophthalmos√ underdeveloped paranasal sinuses√ underdeveloped maxilla with prognathism√ high pointed arch of palate√ prominent vertical crest in middle of forehead (increased intracranial pressure)√ V-shaped anterior fossa due to elevation of lateral margins of lesser sphenoid√ sella may be enlarged√ cervical spine may be fused@Hand & feet√ fusion of distal portions of phalanges, metacarpals / carpals (2nd, 3rd + 4th digit)√ absence of middle phalanges√ missing / supernumerary carpal / tarsal bones√ pseudarthroses

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Notes:

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ARTERIOVENOUS FISTULA OF BONE

Etiology:(a)acquired (usually gunshot wound)(b)congenitalLocation:lower extremity most frequent√ soft-tissue mass√ presence of large vessels√ phleboliths (DDx: long-standing varicosity)√ accelerated bone growth√ cortical osteolytic defect (= pathway for large vessels into medulla)√ increased bone density

Notes:

ARTHROGRYPOSIS
=ARTHROGRYPOSIS MULTIPLEX CONGENITA= nonprogressive congenital syndrome complex characterized by poorly developed + contracted muscles, deformed joints with thickened periarticular capsule and intact sensory system*Pathophysiology:* congenital / acquired defect of motor unit (anterior horn cells, nerve roots, peripheral nerves, motor endplates, muscle) early in fetal life with immobilization of joints at various stages in their development *Cause:*? neurotropic agents, toxic chemicals, hard drugs, hyperthermia, neuromuscular blocking agents, mytotic abnormalities, mechanical immobilization*Incidence:*0.03% of newborn infants; 5% risk of recurrence in sibling*Path:*diminution in size of muscle fibers + fat deposits in fibrous tissue*Associated with:* (1)neurogenic disorders (90%)(2)myopathic disorders(3)skeletal dysplasias(4)intrauterine limitation of movement (myomata, amniotic band, twin, oligohydramnios)(5)connective tissue disordersDistribution:all extremities (46%), lower extremities only (43%), upper extremities only (11%); peripheral joints >> proximal joints; symmetrical ● clubfoot ● congenital dislocation of hip ● claw hand ● diminished muscle mass ● skin webs√ flexion + extension contractures√ osteopenia ± pathologic fractures√ congenital dislocation of hip√ carpal coalition√ vertical talus√ calcaneal valgus deformity

Notes:

ASPHYXIATING THORACIC DYSPLASIA
=JEUNE DISEASE = autosomal recessive disorder*Incidence:*100 cases*Associated with:*renal anomalies (hydroureter), PDA ● reduced thoracic mobility (abdominal breathing) + frequent pulmonary infections ● progressive renal failure + hypertension@ Chest √ markedly narrow + elongated bell-shaped chest√ normal size of heart leaving little room for lungs√ horizontal clavicles at level of 6th cervical vertebra√ short horizontal ribs + irregular bulbous costochondral junction@ Pelvis √ trident pelvis (retardation of ossification of triradiate cartilage)√ small iliac bone flared + shortened in cephalocaudal diameter ("wineglass" pelvis)√ short ischial + pubic bones √ reduced acetabular angle√ premature ossification of capital femoral epiphysis@ Extremities √ rhizomelic brachymelia (humerus, femur) = long bones shorter + wider than normal√ metaphyseal irregularity√ postaxial hexadactyly√ shortening of distal phalanges + cone-shaped epiphyses in hands + feet@ Kidneys √ enlarged kidneys with linear streaking on nephrogramOB-US: √ proportionate shortening of long bones√ small thorax with decreased circumference√ increased cardiothoracic ratio√ occasionally polydactyly√ polyhydramnios *Prognosis:*neonatal death in 80% (respiratory failure + infections)*DDx:*Ellis-van Creveld syndrome

Notes:

AVASCULAR NECROSIS
= AVN = OSTEONECROSIS = ASEPTIC NECROSIS = consequence of interrupted blood supply to bone with death of cellular elements *Histo:* (a)cellular ischemia leading to death of hematopoietic cells (in 6-12 hours), osteocytes (in 12-48 hours) and lipocytes (in 2-5 days)(b)necrotic debris in intertrabecular spaces + proliferation and infiltration by mesenchymal cells + capillaries(c)mesenchymal cells differentiate to osteoblasts on the surface of dead trabeculae synthesizing new bone layers + resulting in trabecular thickening*Pathogenesis:* (1)obstruction of extra- and intraosseous vessels by arterial embolism, venous thrombosis, traumatic disruption, external compression (increased marrow space pressure)(2)cumulative stress from cytotoxic factors *Cause:* A.Traumatic interruption of arteries@femoral head:1.Femoral neck fracture (60-75%)2.Dislocation of hip joint (25%)3.Slipped capital femoral epiphysis (15-40%)@carpal scaphoid:4-6 months after fracture (in 10-15%), in 30-40% of nonunion of scaphoid fracture Site:proximal fragment (most common)@humeral head (infrequent)B.Embolization of arteries1.Hemoglobinopathy: sickle-cell disease2.Nitrogen bubbles: Caisson diseaseC.Vasculitis1.Collagen-vascular disease: SLE2.Radiation exposureD.Abnormal accumulation of cells1.Lipid-containing histiocytes: Gaucher disease2.Fat cells: steroid therapyE.Idiopathic1.Spontaneous osteonecrosis of knee2.Legg-Calvé-Perthes disease3.Freiberg disease *mnemonic:*"PLASTIC RAGS"**P**ancreatitis, **P**regnancy **L**egg-Perthes disease, **L**upus erythematosus **A**lcoholism, **A**therosclerosis **S**teroids **T**rauma (femoral neck fracture, hip dislocation) **I**diopathic (Legg-Perthes disease), **I**nfection **C**aisson disease, **C**ollagen disease (SLE) **R**heumatoid arthritis, **R**adiation treatment **A**myloid **G**aucher disease **S**ickle cell disease *mnemonic:*"GIVE INFARCTS"**G**aucher disease **I**diopathic (Legg-Calvé-Perthes, Köhler, Chandler) **V**asculitis (SLE, polyarteritis nodosa, rheumatoid arthritis) **E**nvironmental (frostbite, thermal injury) **I**rradiation **N**eoplasia (-associated coagulopathy) **F**at (prolonged corticosteroid use increases marrow) **A**lcoholism **R**enal failure + dialysis **C**aisson disease **T**rauma (femoral neck fracture, hip dislocation) **S**ickle cell disease NO predisposing factors in 25%! Location:femoral head (most common), humeral head, femoral condyles

Avascular Necrosis of Hip Blount Disease Calvé-Kümmel-Verneuil Disease Freiberg Disease Kienböck Disease Köhler Disease Legg-Calvé-Perthes Disease Panner Disease Preiser Disease Spontaneous Osteonecrosis of Knee

Notes:

Avascular Necrosis of Hip ◊Involvement of one hip increases risk to contralateral hip to 70%!*Age:*20-50 years ● hip / groin / thigh / knee pain ● limited range of motion Plain film (positive only several months after symptoms): √ radiolucent crescent parallel to articular surface secondary to subchondral structural collapse of necrotic segmentSite:anterosuperior portion of femoral head (best seen on frogleg view)√ preservation of joint space (DDx: arthritis)√ flattening of articular surface√ increased density of femoral head (compression of bony trabeculae following microfracture of nonviable bone, calcification of dendritic marrow, creeping substitution = deposition of new bone) *Classification* (Steinberg): Stage O=normalStage I=normal / barely detectable trabecular mottling; abnormal bone scan / MRIStage IIA=focal sclerosis + osteopeniaStage IIB=distinct sclerosis + osteoporosis + early crescent signStage IIIA=subchondral undermining ("crescent sign") + cyst formationStage IIIB=mild alteration in femoral head contour / subchondral fracture + normal joint spaceStage IV=marked collapse of femoral head + significant acetabular involvementStage V=joint space narrowing + acetabular degenerative changes NUC (80-85% sensitivity): ◊Bone marrow imaging (with radiocolloid) more sensitive than bone imaging (with diphosphonates)◊More sensitive than plain films in early AVN (evidence of ischemia seen as much as 1 year earlier)◊Less sensitive than MR*Technique:*imaging improved with double counts, pinhole collimation√ early: cold = photopenic defect (interrupted blood supply)√ late: "doughnut sign" = cold spot surrounded by increased uptake secondary to(a)capillary revascularization + new-bone synthesis(b) degenerative osteoarthritis CT (utilized for staging of known disease): √ staging upgrades in 30% compared with plain films MR (90-100% sensitivity for symptomatic disease): *Prevalence* of clinically occult disease: 6% ◊MR imaging changes reflect the death of marrow fat cells (not death of osteocytes with empty lacunae)!◊Sagittal images particularly useful!
Classification (Mitchell): *StageT1T2analogous to* AhighintermediatefatBhighhighsubacute bloodClowhighfluid / edemaDlowlowfibrosis
EARLY AVN: √ decreased Gd-enhancement on short-inversion-recovery (STIR) images (very early) √ low-signal intensity band with sharp inner interface + blurred outer margin on T1WI within 12-48 hours (= mesenchymal + fibrous repair tissue, amorphous cellular debris, thickened trabecular bone) seen as(a)band extending to subchondral bone plate(b)complete ring (less frequent)√ "double-line sign" on T2WI (in 80%) [MORE SPECIFIC] = juxtaposition of inner hyperintense band (granulation tissue) + outer hypointense band (chemical shift artifact / fibrosis + sclerosis) ADVANCED AVN: √ "pseudohomogeneous edema pattern"= inhomogeneous large areas of mostly decreased signal intensity on T1WI √ hypo- to hyperintense lesion on T2WI√ contrast-enhancement of interface + surrounding marrow + within lesion SUBCHONDRAL FRACTURE: √ predilection for anterosuperior portion of femoral head (sagittal images!)√ cleft of low-signal intensity running parallel to the subchondral bone plate within areas of fatlike signal intensity on T1WI √ hyperintense band (= fracture cleft filled with articular fluid / edema) within the intermediate- or low-signal-intensity necrotic marrow on T2WI√ lack of enhancement within + around fracture cleft EPIPHYSEAL COLLAPSE: √ focal depression of subchondral bone *Cx:*early osteoarthritis through collapse of femoral head + joint incongruity in 3-5 years if left untreated*Rx:*(1)core decompression (for grade 0-II): most successful with <25% involvement of femoral head(2)osteotomy (for grade 0-II)(3)arthroplasty / arthrodesis / total hip replacement (for grade >III)*DDx:*bone marrow edema (ill-delimited marrow changes, no reactive interface); epiphyseal fracture (speckled / linear hypointense areas, focal depression of epiphyseal contour)

Notes:

Blount Disease =TIBIA VARA=avascular necrosis of medial tibial condyle*Age:*>6 years ● limping, lateral bowing of leg√ medial tibial condyle enlarged + deformed (DDx: Turner syndrome)√ irregularity of metaphysis (medially + posteriorlyprolonged with beak)

Notes:

Calvé-Kümmel-Verneuil Disease =VERTEBRAL OSTEOCHONDROSIS = VERTEBRA PLANA = avascular necrosis of vertebral body*Age:*2-15 years√ uniform collapse of vertebral body into flat thin disk√ increased density of vertebra√ neural arches NOT affected√ disks are normal with normal intervertebral disk space√ intravertebral vacuum cleft sign (PATHOGNOMONIC)*DDx:*Eosinophilic granuloma, metastatic disease

Notes:

Freiberg Disease =osteochondrosis of head of 2nd (3rd / 4th) metatarsal*Age:*10-18 years; M:F = 1:3 ● metatarsalgia, swelling, tendernessEarly: √ flattening, increased density, cystic lesions of metatarsal head√ widening of metatarsophalangeal jointLate: √ osteochondral fragment√ sclerosis + flattening of metatarsal head√ increased cortical thickening

Notes:

Kienböck Disease =LUNATOMALACIA= avascular necrosis of lunate bone *Predisposed:*individuals engaged in manual labor with repeated / single episode of trauma *Age:*20-40 years*Associated with:* ulna minus variant (short ulna) in 75% ● progressive pain + soft-tissue swelling of wrist Location:uni- > bilateral (usually right hand)√ initially normal radiograph√ fracture / osteonecrosis of lunate√ increased density + altered shape + collapse of lunate*Cx:*scapholunate separation, ulnar deviation of triquetrum, degenerative joint disease in radiocarpal / midcarpal compartments*Rx:*ulnar lengthening / radial shortening, lunate replacement

Notes:

Köhler Disease =avascular necrosis of tarsal scaphoid*Age:*3-10 years; boys√ irregular outline√ fragmentation√ disklike compression in AP direction√ increased density√ joint space maintained√ decreased / increased uptake on radionuclide study

Notes:

Legg-Calvé-Perthes Disease =COXA PLANA = idiopathic avascular necrosis of femoral head in children; one of the most common sites of AVN; 10% bilateral*Age:*(a)4-8 years: M:F = 5:1(b)adulthood: **Chandler disease**
*Cause:*trauma in 30% (subcapital fracture, epiphyseolysis, esp. posterior dislocation), closed reduction of congenital hip dislocation, prolonged interval between injury and reduction*Pathophysiology:* femoral head blood supply insufficient (epiphyseal plate acts as a barrier in ages 4-10; ligamentum teres vessels become nonfunctional; blood supply is from medial circumflex artery + lateral epiphyseal artery only) *Stages:* I=histologic + clinical diagnosis without radiographic findingsII=sclerosis ± cystic changes with preservation of contour + surface of femoral headIII=loss of structural integrity of femoral headIV=in addition loss of structural integrity of acetabulum ● 1 week-6 months (mean 2.7 months) duration of symptoms prior to initial presentation: limping, pain NUC (may assist in early diagnosis): √ decreased uptake (early) in femoral head = interruption of blood supply√ increased uptake (late) in femoral head =(a) revascularization + bone repair (b) degenerative osteoarthritis √ increased acetabular activity with associated degenerative joint disease X-RAY: Early signs: √ femoral epiphysis smaller than on contralateral side (96%)√ sclerosis of femoral head epiphysis (sequestration + compression) (82%)√ slight widening of joint space due to thickening of cartilage, failure of epiphyseal growth, presence of joint fluid, joint laxity (60%)√ ipsilateral bone demineralization (46%)√ alteration of pericapsular soft-tissue outline due to atrophy of ipsilateral periarticular soft tissues (73%)√ rarefaction of lateral + medial metaphyseal areas of neck√ NEVER destruction of articular cortex as in bacterial arthritisLate signs: √ delayed osseous maturation of a mild degree√ "radiolucent crescent line" of subchondral fracture= small archlike subcortical lucency (32%)√ subcortical fracture on anterior articular surface (best seen on frogleg view)√ femoral head fragmentation√ femoral neck cysts (from intramedullary hemorrhage in response to stress fractures)√ loose bodies (only found in males)√ coxa plana = flattened collection of sclerotic fragments (over 18 months)√ coxa magna = remodeling of femoral head to become wider + flatter in mushroom configuration to match widened metaphysis + epiphyseal plateCT: √ loss of "asterisk" sign (= starlike pattern of crossing trabeculae in center of femoral head) with distortion of asterisk and extension to surface of femoral headMR: √ normal signal intensity in marrow of femoral epiphysis replaced by low signal intensity on T1WI + high signal intensity on T2WI = "asterisk" sign√ "double-line" sign (80%) = sclerotic nonsignal rim producing line between necrotic + viable bone edged by a hyperintense rim of granulation tissue√ fluid within fracture plane√ hip joint incongruity: lateral femoral head uncovering, labral inversion, femoral head deformity*Cx:*severe degenerative joint disease in early adulthood

Notes:

Panner Disease =osteonecrosis of capitellum

Notes:

Preiser Disease =nontraumatic osteonecrosis of scaphoid

Notes:

Spontaneous Osteonecrosis of Knee = SONK *Cause:*? meniscal tear (78%), trauma with resultant microfractures, vascular insufficiency, degenerative joint disease, severe chondromalacia, gout, rheumatoid arthritis, joint bodies, intra-articular steroid injection (45-85%)*Age:*7th decade (range 13-83 years) ● acute onset of painLocation:weight-bearing medial condyle more toward epicondylus (95%), lateral condyle (5%), may involve tibial plateau√ radiographs usually normal (within 3 months after onset)√ positive bone scan within 5 weeks (most sensitive)√ flattening of weight-bearing segment of medial femoral epicondyle√ radiolucent focus in subchondral bone + peripheral zone of osteosclerosis√ horizontal subchondral fracture (within 6-9 months) + osteochondral fragment√ periosteal reaction along medial side of femoral shaft (30-50%)*Cx:*osteoarthritis

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Notes:

BASAL CELL NEVUS SYNDROME
=GORLIN SYNDROME = syndrome of autosomal dominant inheritance characterized by(1)multiple cutaneous basal cell carcinomas(2)jaw cysts(3)ectopic calcifications(4)skeletal anomalies ● multiple nevoid basal cell carcinomas (nose, mouth, chest, back) at mean age of 19 years; after puberty aggressive, may metastasize ● pitlike defects in palms + soles*Associated with:*high incidence of medulloblastoma in children√ multiple mandibular + maxillary cysts (dentigerous cysts + ectopic dentition)√ anomalies of upper 5 ribs: bifid, fused, dysplastic√ bifid spinous processes, spina bifida√ scoliosis (cervical + upper thoracic)√ hemivertebrae + block vertebrae√ Sprengel deformity (scapula elevated, hypoplastic, bowed)√ brachydactyly√ extensive calcification of falx + tentorium√ ectopic calcifications of subcutaneous tissue, ovaries, sacrotuberous ligaments, mesentery√ bony bridging of sella turcica

Notes:

BATTERED CHILD SYNDROME
= CAFFEY-KEMPE SYNDROME = CHILD ABUSE = PARENT / INFANT TRAUMATIC STRESS SYNDROME = NONACCIDENTAL TRAUMA◊Most common cause of serious intracranial injuries in children <1 year of age; 3rd most common cause of death in children after sudden infant death syndrome + true accidents*Prevalence:*1.7 million cases reported + 833,000 substantiated in United States in 1990 (45% neglected children, 25% physically abused, 16% sexually abused children); resulting in 2,500-5,000 deaths/year; 5-10% of children seen in emergency rooms*Age:*usually <2 years ● skin burns, bruising, lacerations, hematomas (SNAT = suspected nonaccidental trauma)@Skeletal trauma (50-80%)Site:multiple ribs, transverse fracture of sternum, costochondral / costovertebral separation, lateral end of clavicles, scapula, acromion, skull, anterior-superior wedging, vertebral compression, vertebral fracture dislocation, disk space narrowing, spinous processes, tibia, metacarpus√ multiple asymmetric fractures in different stages of repair (HALLMARK = repeated injury)√ separation of distal epiphysis√ marked irregularity + fragmentation of metaphyses (DDx: osteochondritis stage of congenital syphilis; infractions of scurvy)√ "corner" fracture (11%) = "bucket-handle" fracture= avulsion of an arcuate metaphyseal fragment overlying the lucent epiphyseal cartilage secondary to sudden twisting motion of extremity about knee, elbow, distal tibia, fibula, radius, ulna (periosteum easily pulled away from diaphysis but tightly attached to metaphysis)√ isolated spiral fracture (15%) of diaphysis secondary to external rotatory force applied to femur / humerus√ extensive periosteal reaction from large subperiosteal hematoma (DDx: scurvy, copper deficiency)√ exuberant callus formation at fracture sites√ cortical hyperostosis extending to epiphyseal plate (DDx: not in infantile cortical hyperostosis)√ avulsion fracture of ligamentous insertion; frequently seen without periosteal reaction @Head trauma (13-25%)Most common cause of death + physical disability (1)Impact injury with translational force: skull fracture (flexible calvaria + meninges decrease likelihood of skull fractures), subdural hematoma, brain contusion, cerebral hemorrhage, infarction, generalized edema(2)Whiplash injury with rotational force: shearing injuries + associated subarachnoid hemorrhage ● bulging fontanelles, convulsionsSkull film (associated fracture in 1%): √ linear fracture > comminuted fracture > diastases (conspicuously absent)CT: √ subdural hemorrhage (most common): interhemispheric location most common√ subarachnoid hemorrhage√ epidural hemorrhage (uncommon)√ cerebral edema (focal, multifocal, diffuse)√ acute cerebral contusion as ovoid collection of intraparenchymal blood with surrounding edemaMR:more sensitive in identifying hematomas of differing ages√ white matter shearing injuries as areas of prolonged T1 + T2 at corticomedullary junction, centrum semiovale, corpus callosum@ Viscera (3%) Second leading cause of death in child abuse *Cause:*crushing blow to abdomen (punch, kick)*Age:*often >2 years√ small bowel / gastric rupture√ hematoma of duodenum / jejunum√ contusion / laceration of lung, pancreas, liver, spleen, kidney√ traumatic pancreatic pseudocyst *Cx:*(1)Brain atrophy (up to 100%)(2)Infarction (50%)(3)Subdural hygroma(4)Encephalomalacia(5)Porencephaly*DDx:*normal periostitis of infancy, osteogenesis imperfecta, congenital insensitivity to pain, infantile cortical hyperostosis, Menkes kinky hair syndrome, Schmid-type chondrometaphyseal dysplasia, scurvy, congenital syphilitic metaphysitis

Notes:

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BENIGN CORTICAL DEFECT
=developmental intracortical bone defect*Age:*usually 1st-2nd decade; uncommon in boys <2 years of age; uncommon in girls <4 years of age ● asymptomaticSite:metaphysis of long bone√ well-defined intracortical round / oval lucency√ usually <2 cm long√ sclerotic margins*Cx:*pathologic / avulsion fracture following minor trauma (infrequent)*Prognosis:*(1)Spontaneous healing resulting in sclerosis / disappearance(2)Ballooning of endosteal surface of cortex = fibrous cortical defect(3)Medullary extension resulting in nonossifying fibroma

Notes:

BONE INFARCT
Etiology: A.Occlusion of vessel:(a)thrombus: thromboembolic disease, sickle cell anemia (SS + SC hemoglobin), polycythemia rubra vera(b)fat: pancreatitis (intramedullary fat necrosis from circulating lipase), alcoholism(c)gas: Caisson disease, astronautsB.Vessel wall disease:1.Arteritis: SLE, rheumatoid arthritis, polyarteritis nodosa, sarcoidosis2.ArteriosclerosisC.Vascular compression by deposition of:(a)fat: corticosteroid therapy (eg, renal transplant, Cushing disease)(b)blood: trauma (fractures + dislocations)(c)inflammatory cells: osteomyelitis, infection, histiocytosis X(d)edema: radiation therapy, hypothyroidism, frostbite(e)substances: Gaucher disease (vascular compression by lipid-filled histiocytes), goutD.Others: idiopathic, hypopituitarism, pheochromocytoma (microscopic thrombotic disease), osteochondroses

Medullary Infarction Cortical Infarction

Notes:

Medullary Infarction ◊Nutrient artery is the sole blood supply for diaphysis!Location:distal femur, proximal tibia, iliac wings, ribs, humeri(a)Acute phase:√ NO radiographic changes without cortical involvement√ area of rarefaction√ bone marrow scan: diminished uptake in medullary RES for long period of time√ bone scan: photon-deficient lesion within 24-48 hours; increased uptake after collateral circulation established(b)Healing phase: (complete healing / fibrosis / calcification)√ demarcation by zone of serpiginous / linear calcification + ossification parallel to cortex√ dense bone indicating revascularization

Notes:

Cortical Infarction ◊Requires compromise of (a) nutrient artery and(b) periosteal vessels!*Age:*particularly in childhood where periosteum is easily elevated by edema √ avascular necrosis = osteonecrosis√ osteochondrosis dissecans*Cx:*(1)Growth disturbances√ cupped / triangular / coned epiphyses√ "H-shaped" vertebral bodies(2)Fibrosarcoma (most common), malignant fibrous histiocytoma, benign cysts(3)Osteoarthritis

Notes:

BONE ISLAND
=ENOSTOSIS = ENDOSTEOMA = COMPACT ISLAND= FOCAL SCLEROSIS = SCLEROTIC BONE ISLAND= CALCIFIED MEDULLARY DEFECT=focal lesion of densely sclerotic (compact) bone nesting within spongiosa*Age:*any age (mostly 20-80 years of age); grows more rapidly in children*Histo:*nest of lamellar compacted bone with haversian system embedded within medullary canal*Pathogenesis:* ? misplaced cortical hamartoma, ? developmental error of endochondral ossification as a coalescence of mature bone trabeculae with failure to undergo remodeling ● asymptomaticLocation:ilium + proximal femur (88-92%), ribs, spine (1-14%), humerus, phalanges (not in skull)√ round / oval solitary osteoblastic lesion with abrupt transition to surrounding normal trabecular bone√ long axis of bone island parallels long axis of bone√ usually 2-10 mm in size; lesion >2 cm in longest axis = GIANT BONE ISLAND√ "brush border" = "thorny radiations" = sharply demarcated margins with feathery peripheral radiations (HALLMARK)√ may show activity on bone scan, esp. if large (33%)√ may demonstrate slow growth / decrease in size (32%)√ NO involvement of cortex / radiolucencies / periosteal reaction*Prognosis:*may increase to 8-12 cm over years (40%); may decrease / disappear*DDx:*(1)Osteoblastic metastasis (aggressive, break through cortex, periosteal reaction)(2)Low-grade osteosarcoma (cortical thickening, extension beyond medullary cavity)(3)Osteoid osteoma (pain relieved by aspirin, nidus)(4)Benign osteoblastoma(5)Involuted nonossifying fibroma replaced by dense bone scar(6)Eccentric focus of monostotic fibrous dysplasia(7)Osteoma (surface lesion)

Notes:

BRUCELLOSIS
=multisystemic zoonosis of worldwide distribution; endemic in Saudi Arabia, Arabian Peninsula, South America, Spain, Italy (secondary to ingestion of raw milk / milk products)*Organism:*small Gram-negative nonmotile, nonsporing, aflagellate, nonencapsulated coccobacilli: Brucella abortus, B. suis, B. canis, B. melitensis*Histo:*small intracellular pathogens shed in excreta of infected animals (urine, stool, milk, products of conception) cause small noncaseating granuloma within RESLocation:commonest site of involvement is reticulo-endothelial system; musculoskeletal system ● 1-3 weeks between initial infection + symptoms✧Radiologic evidence of disease in 69% of symptomatic sites!@Brucellar spondylitis (53%)*Age:*40 years is average age at onset ● pain, localized tenderness, radiculopathy, myelopathyLocation:lumbar (71%) > thoracolumbar (10%) > lumbosacral (8%) > cervical (7%) > thoracic (4%)(a)focal form√ bone destruction at diskovertebral junction (anterior aspect of superior endplate)√ associated with bone sclerosis + anterior osteophyte formation + small amount of gas(b)diffuse form: entire vertebral endplate / whole vertebral body affected with spread to adjacent disks + vertebral bodies√ bone destruction associated with sclerosis√ small amount of disk gas (25-30%)√ obliteration of paraspinal muscle-fat planes√ no / minimal epidural extension*DDx:*TB (paraspinal abscess, gibbus)@Extraspinal disease(a)Brucellar synovitis (81%)Location:knee > sacroiliac joint > shoulder > hip > sternoclavicular joint > ankle > elbowSite:organism localized in synovial membrane ● serosanguinous sterile joint effusion(b)Brucellar destructive arthritis (9%)√ indistinguishable from tuberculous / pyogenic arthritis(c)Brucellar osteomyelitis (2%) ● pain, tenderness, swelling(d)Brucellar myositis (2%)*Dx:*serologic tests (enzyme-linked immunosorbent assay, counterimmunoelectrophoresis, rose bengal plate test*Rx:*combination of aminoglycosides + tetracyclines*DDx:*fibrous dysplasia, benign tumor, osteoid osteoma

Notes:

CAISSON DISEASE
=DECOMPRESSION SICKNESS = THE BENDS*Etiology:*during too rapid decompression = reduction of surrounding pressure (ascent from dive, exit from caisson / hyperbaric chamber, ascent to altitude) nitrogen bubbles form (nitrogen more soluble in fat of panniculus adiposus, spinal cord, brain, bones containing fatty marrow) ● "the bends" = local pain in knee, elbow, shoulder, hip ● neurologic symptoms (paresthesia, major cerebral / spinal involvement) ● "chokes" = substernal discomfort + coughing(embolization of pulmonary vessels)Location:mostly in long tubular bones of lower extremity (distal end of shaft + epiphyseal portion); symmetrical lesions√ early: area of rarefaction√ healing phase: irregular new-bone formation with greater density√ peripheral zone of calcification / ossification√ ischemic necrosis of articular surface with secondary osteoarthritis

Notes:

CALCIUM PYROPHOSPHATE DIHYDRATE CRYSTAL DEPOSITION DISEASE
= PSEUDOGOUT = FAMILIAL CHONDROCALCINOSIS =most common crystalline arthropathy*Types:*1.Osteoarthritic form (35-60%)2.Pseudogout = acute synovitis (10-20%)3.Rheumatoid form (2-6%)4.Pseudoneuropathic arthropathy (2%)5.Asymptomatic with tophaceous pseudogout (common)*Associated with:***h**yperparathyroidism, **h**ypothyroidism, **h**emochromatosis, **h**ypomagnesemia *Prevalence:*widespread in older population; M:F = 3:2 ● calcium pyrophosphate crystals in synovial fluid + within leukocytes (characteristic weakly positive birefringent diffraction pattern) ● acute / subacute / chronic joint inflammationLocation:(a)knee (especially meniscus + cartilage of patellofemoral joint)(b)wrist (triangular fibrocartilage in distal radioulnar joint bilaterally)(c)pelvis (sacroiliac joint, symphysis)(d)spine (annulus fibrosis of lumbar intervertebral disk; NEVER in nucleus pulposus as in ochronosis)(e)shoulder (glenoid), hip (labrum), elbow, ankle, acromioclavicular joint√ polyarticular chondrocalcinosis (in fibro- and hyaline cartilage)√ disproportionate narrowing of patellofemoral joint√ involvement of tendons, bursae, pinnae of the ear√ pyrophosphate arthropathy resembles osteoarthritis: joint space narrowing, extensive subchondral sclerosis√ large subchondral cyst (HALLMARK)√ numerous intra-articular bodies (fragmentation of subchondral bone)

Notes:

CAMPOMELIC DYSPLASIA
=sporadic / autosomal recessive dwarfism*Incidence:*0.05:10,000 births*Associated with:* 1.Hydrocephalus (23%)2.Congenital heart disease (30%): VSD, ASD, tetralogy, AS3.Hydronephrosis (30%) ● pretibial dimple √ macrocephaly, cleft palate, micrognathia (90-99%)@Chest & spine√ hypoplastic scapulae (92%)√ narrow bell-shaped chest√ hypoplastic vertebral bodies + nonmineralized pedicles (especially lower cervical spine)@Pelvis√ vertically narrowed iliac bones√ vertical inclination of ischii√ wide symphysis√ narrow iliac bones with small wings√ shallow acetabulum@Extremities (lower extremity more severely affected)√ dislocation of hips + knees√ anterior bowing (= campo) of long bones: marked in tibia + moderate in femur√ hypoplastic fibula√ small secondary ossification center of knee√ small primary ossification center of talus√ clubfootOB-US: √ bowing of tibia + femur√ decreased thoracic circumference√ hypoplastic scapulae√ ± cleft palate*Prognosis:*death usually <5 months of age (within first year in 97%) due to respiratory insufficiency

Notes:

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CARPAL TUNNEL SYNDROME
=entrapment syndrome caused by chronic pressure on the median nerve within the carpal tunnel*Cause:*repetitive wrist / finger flexion; carpal tunnel crowding by cyst / mass / flexor tendon tendinitis or tenosynovitis / anomalous origin of lumbrical muscles*Pathogenesis:*probably ischemia with venous congestion (stage 1), nerve edema from anoxic damage to capillary endothelium (stage 2), impairment of venous + arterial blood supply (stage 3) ● nocturnal hand discomfort ● weakness, clumsiness, finger paresthesiasMR: √ "pseudoneuroma" of median nerve = swelling of median nerve proximal to carpal tunnel√ swelling of nerve within carpal tunnel√ increased signal intensity of nerve on T2WI√ volar bowing of flexor retinaculum√ swelling of tendon sheath (due to tenosynovitis)√ mass(es) within carpal tunnel√ marked enhancement (nerve edema = breakdown of blood-nerve barrier)√ no enhancement (ischemia) provoked by wrist held in an extended / flexed position

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CARPENTER SYNDROME

=ACROCEPHALOPOLYSYNDACTYLY type 2autosomal recessive ● retardation ● hypogonadism√ patent ductus arteriosus√ acro(oxy)cephaly√ preaxial polysyndactyly + soft-tissue syndactyly

Notes:

CHONDROBLASTOMA

=CODMAN TUMOR = BENIGN CHONDROBLASTOMA = CARTILAGE-CONTAINING GIANT CELL TUMOR*Incidence:*1% of primary bone neoplasms (700 cases in world literature)*Age:*peak in 2nd decade (range of 8-59 years); 10-26 years (90%); M:F = 2:1; occurs before cessation of enchondral bone growth*Path:*derived from primitive cartilage cells*Histo:*polyhedral chondroblasts + multinucleated giant cells + nodules of pink amorphous material (= chondroid) = epiphyseal chondromatous giant cell tumor (resembles chondromyxoid fibroma); "chicken wire" calcification = pericellular deposition of calcification is virtually PATHOGNOMONIC ● symptomatic for months to years prior to treatment ● mild joint pain, tenderness, swelling (joint effusion) ● limitation of motionLocation: (a)long bones (80%): proximal femur + greater trochanter (23%), distal femur (20%), proximal tibia (17%), proximal humerus (17%)◊2/3 in lower extremity, 50% about knee◊may occur in apophyses (minor + greater trochanter, patella, greater tuberosity of humerus)(b)flat bones: near triradiate cartilage of innominate bone(c)short tubular bones of hand + feetSite:eccentric medullary, subarticular location with open growth plate (98% begin within epiphysis); tumor growth may continue to involve metaphysis (50%) + rarely diaphysis√ oval / round eccentrically placed lytic lesion of epiphysis√ 1-4 cm in diameter occupying < one-half of epiphysis√ well-defined sclerotic margin, lobulated in 50%√ punctate / irregular calcifications in 25-30-50% (cartilaginous clumps better visualized by CT)√ intact cortical border√ thick periosteal reaction in metaphysis (50%) / joint involvement√ periostitis of adjacent metaphysis / diaphysis (30-50%)√ open growth plate in majority of patientsMR: ◊MR tends to overestimate extent + aggressiveness due to large area of reactive edema!√ intermediate to low signal intensity on T2WI relative to fat√ extensive intramedullary signal abnormalities consistent with bone marrow edema√ peripheral rim of very low signal intensity√ hypointense changes on T1WI + hyperintense on T2WI in adjacent soft tissues (muscle edema) in 50%√ ± joint effusion*Prognosis:*almost always benign; may become locally aggressive; rarely metastasizes*Dx:*surgical biopsy*Rx:*curettage + bone chip grafting (recurrence in 25%)*DDx:*(1)Ischemic necrosis of femoral head (may be indistinguishable, more irregular configuration)(2)Giant cell tumor (usually larger + less well demarcated, not calcified, older age group with closed growth plate)(3)Chondromyxoid fibroma(4)Enchondroma(5)Osteomyelitis (less well-defined, variable margins)(6)Aneurysmal bone cyst(7)Intraosseous ganglion(8)Langerhans cell histiocytosis (less well-defined, variable margins)(9)Primary bone sarcoma

Notes:

CHONDRODYSPLASIA PUNCTATA
=CONGENITAL STIPPLED EPIPHYSES=DYSPLASIA EPIPHYSEALIS PUNCTATA=CHONDRODYSTROPHIA CALCIFICANS CONGENITA =proportional / mesomelic dwarfism*Etiology:*peroxisomal disorder characterized by fibroblast plasmalogen deficiency*Incidence:*1:110,000 birthsA.AUTOSOMAL RECESSIVE CHONDRODYSPLASIA PUNCTATA = RHIZOMELIC TYPE*Associated with:*CHD (common) ● flat face ● congenital cataracts ● ichthyotic skin thickening ● mental retardation ● cleft palate√ multiple small punctate calcifications of varying size in epiphyses (knee, hip, shoulder, wrist), in base of skull, in posterior elements of vertebrae, in respiratory cartilage and soft tissues (neck, rib ends) before appearance of ossification centers√ prominent symmetrical shortening of femur + humerus (rarely all limbs symmetrically affected)√ congenital dislocation of hip√ flexion contractures of extremities√ clubfeet√ metaphyseal splaying of proximal tubular bones (in particular about knee)√ thickening of diaphyses√ prominent vertebral + paravertebral calcifications√ coronal clefts in vertebral bodies*Prognosis:*death usually <1 year of age*DDx:*Zellweger syndromeB.CONRADI-HÜNERMANN DISEASE= NONRHIZOMELIC TYPE more common milder nonlethal variety; autosomal dominant ● normal intelligence√ more widespread but milder involvement as above*Prognosis:*survival often into adulthood *Cx:*respiratory failure (severe underdevelopment of ribs), tracheal stenosis, spinal cord compression*DDx:*(1)Cretinism (may show epiphyseal fragmentation, much larger calcifications within epiphysis)(2)Warfarin embryopathy(3)Zellweger syndrome

Notes:

CHONDROECTODERMAL DYSPLASIA
=ELLIS-VAN CREVELD SYNDROME = MESODERMAL DYSPLASIA=autosomal recessive acromesomelic dwarfism*Incidence:*120 cases; in inbred Amish communities*Associated with:*congenital heart disease in 50% (single atrium, ASD, VSD) ● ectodermal dysplasia:-absent / hypoplastic brittle spoon-shaped nails-irregular + pointed teeth, partial anodontia, teeth may be present at birth-scant / fine hair ● obliteration of maxillary mucobuccal space (thick frenula between alveolar mucosa + upper lip) ● strabismus ● genital malformations: epispadia, hypospadia, hypoplastic external genitalia, undescended testicles√ hepatosplenomegaly √ accelerated skeletal maturation√ normal spine@ Skull √ wormian bones√ cleft lip@ Chest √ long narrow thorax in AP + transverse dimensions√ horizontal ribs + elevated clavicles@ Pelvis √ small flattened ilium√ trident shape of acetabulum with indentation in roof + bony spur (almost pathognomonic)√ acetabular + tibial exostoses@ Extremities √ thickening + shortening of all long bones, more severe in forearms + lower legs (radius + tibia > humerus + femur)√ excessive shortening of fibula√ widening of proximal tibial shaft + delayed development of tibial plateau√ dislocation of radial head (due to shortening of ulna)√ carpal / tarsal coalition = frequent fusion of two / more carpal (hamate + capitate) + tarsal bones√ supernumerary carpal bones√ hypoplasia / absence of terminal phalanges + cone-shaped epiphyses√ postaxial polydactyly common (usually finger, rarely toe) ± syndactyly of hands + feet√ carpal fusion (after complete ossification) OB-US: √ proportional shortening of long bones√ small thorax with decreased circumference√ increased cardiothoracic ratio√ ASD√ polydactyly *Prognosis:*death within first month of life in 33% (due to respiratory / cardiac complications)*DDx:*Asphyxiating thoracic dysplasia (difficult distinction); rhizomelic achondroplasia

Notes:

CHONDROMALACIA PATELLAE

= pathologic softening of patellar cartilage *Cause:*trauma, tracking abnormality of patella ● anterior knee pain ● asymptomatic (incidental arthroscopic diagnosis)

Classification of Chondromalacia Patellae		
Grade	Arthroscopic pathology	T1 WI of MRI
1	softening + swelling of articular cartilage	focal hypointense areas not extending to cartilage surface / subcondral bone
2	blistering of articular cartilage producing deformity of surface	focal hypointense areas extending to cartilage surface with preservation of sharp cartilage margins
3	surface irregularity + cartilage fibrillation with minimal extension to subchondral bone ("brush-border sign")	focal hypointense areas extending to articular surface but not to osseous surface; loss of sharp dark margin between articular cartilage of patella + trochlea
4	ulceration with exposure of subchondral bone	focal hypointense areas extending from subchondral bone to cartilage surface; cartilage thinned to subchondral bone

Notes:

CHONDROMYXOID FIBROMA
Rare benign cartilaginous tumor; initially arising in cortex *Incidence:*<1% of all bone tumors*Histo:*chondroid + fibrous + myxoid tissue (related to chondroblastoma); may be mistaken for chondrosarcoma*Age:*peak 2nd-3rd decade (range of 5-79 years);M:F = 1:1 ● slowly progressive local pain, swelling, restriction of motionLocation:(a)long bones (60%): about knee (50%), proximal tibia (82% of tibial lesions), distal femur (71% of femoral lesions), fibula(b)short tubular bones of hand + feet (20%)(c)flat bones: pelvis, ribs (classic but uncommon)Site:eccentric, metaphyseal (47-53%), metadiaphyseal (20-43%), metaepiphyseal (26%), diaphyseal(1-10%), epiphyseal (3%)√ expansile ovoid lesion with radiolucent center + oval shape at each end of lesion√ long axis parallel to long axis of host bone (1-10 cm in length and 4-7 cm in width)√ geographic bone destruction (100%)√ well-defined sclerotic margin (86%)√ expanded shell = bulged + thinned overlying cortex (68%)√ partial cortical erosion (68%)√ scalloped margin (58%)√ septations (57%) may mimic trabeculations√ stippled calcifications within tumor in advanced lesions (7%)√ NO periosteal reaction (unless fractured)*Prognosis:*25% recurrence rate following curettage*Cx:*malignant degeneration distinctly unusual*DDx:*(1) Aneurysmal bone cyst (2) Simple bone cyst(3) Nonossifying fibroma (4) Fibrous dysplasia(5) Enchondroma (6) Chondroblastoma(7) Eosinophilic granuloma (8) Fibrous cortical defect (9) Giant cell tumor

Notes:

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CHONDROSARCOMA
A.PRIMARY CHONDROSARCOMAB.SECONDARY CHONDROSARCOMAas a complication of a preexisting skeletal abnormality such as1.Osteochondroma2.Enchondroma3.Parosteal chondromaMetastases (uncommon) to:lungCT: √ chondroid matrix mineralization of "rings and arcs" (CHARACTERISTIC) in 70%√ nonmineralized portion of tumor hypodense to muscle (high water content of hyaline cartilage)√ extension into soft-tissuesMR: √ low to intermediate signal intensity on T1WI√ high signal intensity on T2WI + hypointense areas (due to mineralization)

Peripheral Chondrosarcoma Central Chondrosarcoma Clear Cell Chondrosarcoma Extraskeletal Chondrosarcoma

Notes:

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Peripheral Chondrosarcoma =EXOSTOTIC CHONDROSARCOMA = malignant degeneration of hereditary multiple exostoses and rarely of a solitary exostosis (beginning in cartilaginous cap of osteochondroma)*Peak age:*5th-6th decade; M:F = 1.5:1 ● asymptomatic / pain + swelling (45%)Location:pelvis, scapula, sternum, ribs, ends of humerus / femur, skull, facial bones√ unusually large soft-tissue mass attached to bone√ flocculent / streaky chondroid calcification (CHARACTERISTIC) √ dense radiopaque center with streaks radiating to periphery (not marginated)√ thickening of cortex at site of attachment√ late destruction of bone*DDx:*(1)Osteochondroma (densely calcified with multiple punctate calcifications)(2)Parosteal osteosarcoma (more homogeneous density of calcified osteoid)

Notes:

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Central Chondrosarcoma =ENDOSTEAL CHONDROSARCOMA*Incidence:*3rd most common primary bone tumor (1st multiple myeloma, 2nd osteosarcoma)*Histo:*arises from chondroblasts (tumor osteoid is never formed)*Age:*median 45 years; 50% >40 years; 10% in children (rapidly fatal); M:F = 2:1 ● hyperglycemia as paraneoplastic syndrome (85%)Location:neck of femur, pubic rami, proximal humerus, ribs, skull (sphenoid bone, cerebellopontine angle, mandible), sternum, spine (3-12%)Site:central + meta- / diaphysis√ expansile osteolytic lesion 1 to several cm in size√ short transition zone ± sclerotic margin (well defined from host bone)√ ± small irregular punctate / snowflake type of calcification; single / multiple√ late: loss of definition + break through cortex√ endosteal cortical thickening, sometimes at a distance from the tumor√ presence of large soft-tissue mass*DDx:*benign enchondroma, osteochondroma, osteosarcoma, fibrosarcoma

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Notes:

Clear Cell Chondrosarcoma ◊Usually mistaken for chondroblastoma because of low grade malignancy (may be related)!*Histo:*small lobules of tissue composed of cells with centrally filled vesicular nuclei surrounded by large clear cytoplasm*Age:*19-68 years, predominantly after epiphyseal fusionLocation:proximal femur, proximal humerus, proximal ulna, lamina vertebrae (5%); pubic ramusSite:epiphysis√ single lobulated oval / round sharply marginated lesion of 1-2 cm in size√ surrounding increased bone density√ aggressive rapid growth over 3 cm√ may contain calcifications√ bone often enlarged√ indistinguishable from conventional chondrosarcoma / chondroblastoma (slow growth over years)

Notes:

Extraskeletal Chondrosarcoma *Incidence:*2% of all soft-tissue sarcomas***Myxoid Extraskeletal Chondrosarcoma*** (most common) *Histo:*surrounded by fibrous capsule + divided into multiple lobules by fibrous septa; delicate strands of small elongated chondroblasts are suspended in an abundant myxoid matrix; foci of mature hyaline cartilage are rare*Mean age:*50 years (range 4-92 years); M > F ● slowly growing soft-tissue mass ● pain + tenderness (33%)◊Metastatic in 40-45% at time of presentation!Location:extremities (thigh most common)Site:deep soft tissues; subcutis (25%)√ lobulated soft-tissue mass WITHOUT calcification / ossification√ usually between 4 and 7 cm in diameterMR: √ approximately equal to muscle on T1WI + equal to fat on T2WI√ may mimic a cyst / myxoma*Prognosis:*45% 10-year survival rate; 5-15 years survival after development of metastases ***Extraskeletal Mesenchymal Chondrosarcoma*** 50% of all mesenchymal chondrosarcomas arise in soft tissues *Histo:*proliferation of small primitive mesenchymal cells with scattered islands of cartilage; hemangiopericytoma-like vascular pattern*Bimodal age distribution:*M = F(a)tumors of head + neck in 3rd decade (common): meninges, periorbital region(b)tumors of thigh + trunk in 5th decade ● frequently metastasized to lungs + lymph nodes√ matrix mineralization (50-100%) characterized as rings + arcs / flocculent + stippled calcification / dense mineralizationMR: √ approximately equal to muscle on T1WI + equal to fat on T2WI√ signal voids from calcifications√ homogeneous enhancement*Prognosis:*25% 10-year survival rate

Notes:

CLEIDOCRANIAL DYSOSTOSIS
=CLEIDOCRANIAL DYSPLASIA = MUTATIONAL DYSOSTOSIS=delayed ossification of midline structures (particularly of membranous bone)Autosomal dominant disease @ Skull ● large head√ diminished / absent ossification of skull (in early infancy)√ wormian bones√ widened fontanelles + sutures with delayed closure√ persistent metopic suture√ brachycephaly + prominent bossing√ large mandible√ high narrow palate (± cleft)√ hypoplastic paranasal sinuses√ delayed / defective dentition@Chest√ hypoplasia / absence (10%) of clavicles (defective development usually of lateral portion, R > L (DDx: congenital pseudarthrosis of clavicle)√ thorax may be narrowed + bell-shaped√ supernumerary ribs√ incompletely ossified sternum√ hemivertebrae, spondylosis (frequent)@ Pelvis √ delayed ossification of bones forming symphysis pubis (DDx: bladder exstrophy)√ hypoplastic iliac bones@Extremities√ radius short / absent√ elongated second metacarpals√ pseudoepiphyses of metacarpal bases√ short hypoplastic distal phalanges of hand√ pointed terminal tufts√ coned epiphyses√ coxa vara = deformed / absent femoral necks√ accessory epiphyses in hands + feet (common)OB-US: √ cephalopelvic disproportion (large fetal head + narrow birth canal of affected maternal pelvis) necessitates cesarean section

Notes:

COCCIDIOIDOMYCOSIS
*Histo:*chronic granulomatous process in bones, joints, periarticular structuresLocation:(a)bones: most frequently in metaphyses of long bones + medial end of clavicle, spine, ribs, pelvis / bony prominences of patella, tibial tuberosity, calcaneus, olecranon, acromion(b)weight-bearing joints (33%): knee, ankle, wrist, elbow ● "desert rheumatism"(c)tenosynovitis of hand, bursitis√ focal areas of destruction, formation of cavities (early)= bubbly bone lesion √ bone sclerosis surrounding osteolysis (later, rare)√ proliferation of overlying periosteum√ destruction of vertebra with preservation of disk space√ psoas abscess indistinguishable from tuberculosis, may calcify√ joints rarely infected (usually monarticular from direct extension of osteomyelitic focus): synovial effusion, osteopenia, joint space narrowing, bone destruction, ankylosis√ soft-tissue abscesses common*DDx:*tuberculosis

Notes:

CONGENITAL INSENSITIVITY TO PAIN WITH ANHYDROSIS
=rare autosomal recessive disorder presumably on the basis of abnormal neural crest development*Age:*presenting at birth*Incidence:*15 reported cases *Path:*absence of dorsal + sympathetic ganglia, deficiency of neural fibers <6 µm in diameter + disproportionate number of fibers of 6-10 µm in diameter ● history of painless injuries + burns (DDx: familialdysautonomia, congenital sensory neuropathy, hereditary sensory radicular neuropathy, acquired sensory neuropathy, syringomyelia) ● abnormal pain + temperature perception ● burns, bruises, infections are common ● biting injuries of fingers, lips, tongue ● absence of sweating ● mental retardationCRITERIA:(1) defect must be present at birth(2) general insensitivity to pain (3) general mental / physical retardation √ epiphyseal separation in infancy (epiphyseal injuries result in growth problems)√ metaphyseal fractures in early childhood√ diaphyseal fractures in late childhood√ Charcot joints = neurotrophic joints (usually weight-bearing joints) with effusions + synovial thickening√ ligamentous laxity√ bizarre deformities + gross displacement + considerable hemorrhage (unnoticed fractures + dislocations)√ osteomyelitis + septic arthritis may occur + progress extensively*DDx:*(1) sensory neuropathies (eg, diabetes mellitus),(2) hysteria, (3) syphilis, (4) mental deficiency, (5) syringomyelia, (6) organic brain disease

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CORNELIA DE LANGE SYNDROME
=Amsterdam dwarfism ● mental retardation (IQ <50) ● hirsutism; hypoplastic genitalia ● feeble growling cry ● high forehead; short neck ● arched palate ● bushy eyebrows meeting in midline + long curved eyelashes ● small nose with depressed bridge; upward tilted nostrils; excessive distance between nose + upper lip√ small + brachycephalic skull√ hypoplasia of long bones (upper extremity more involved)√ forearm bones may be absent√ short radius + elbow dislocation√ thumbs placed proximally (hypoplastic 1st metacarpal)√ short phalanges + clinodactyly of 5th finger

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CORTICAL DESMOID
=AVULSIVE CORTICAL IRREGULARITY=PERIOSTEAL / SUBPERIOSTEAL DESMOID= SUBPERIOSTEAL / CORTICAL ABRASION= SUBPERIOSTEAL CORTICAL DEFECT=rare fibrous lesion of the periosteum*Age:*peak 14-16 years (range of 3-17 years); M:F = 3:1*Histo:*shallow defect filled with proliferating fibroblasts, multiple small fragments of resorbing bone (microavulsions) at tendinous insertions ● no localizing signs / symptomsLocation:posteromedial aspect of medial femoral epicondyle along medial ridge of linea aspera at attachment of adductor magnus aponeurosis; 1/3 bilateral√ area of cortical thickening√ 1-2 cm irregular, shallow, concave saucerlike crater with sharp margin√ lamellated periosteal reaction√ localized cortical hyperostosis proximally (healing phase)◊May be confused with a malignant tumor (eg, osteosarcoma) / osteomyelitis!

Notes:

CRI-DU-CHAT SYNDROME
=deletion of short arm of 5th chromosome (5 p) ● generalized dwarfism due to marked growth retardation ● failure to thrive ● peculiar high-pitched cat cry (hypoplastic larynx) ● antimongoloid palpebral fissures ● strabismus ● profound mental retardation ● round facies ● low set ears*Associated with:*congenital heart disease (obtain CXR!)√ agenesis of corpus callosum√ microcephaly√ hypertelorism√ small mandible√ faulty long-bone development√ short 3rd, 4th, 5th metacarpals√ long 2nd, 3rd, 4th, 5th proximal phalanges√ horseshoe kidney*Dx:*made clinically

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CROUZON DISEASE
=CRANIOFACIAL SYNOSTOSIS / DYSOSTOSIS=Apert syndrome without syndactyly=characterized by skull + cranial base deformities secondary to craniosynostosis, maxillary hypoplasia, shallow orbits, ocular proptosis*Prevalence:*1:25,000*Etiology:*autosomal dominant inheritance (in 67%) ● parrot-beak nose ● strabismus ● deafness ● mental retardation ● dental abnormalities√ acro(oxy)cephaly / brachycephaly / scaphocephaly / trigonocephaly / "cloverleaf" skull (premature craniosynostosis) √ hypertelorism + exophthalmos√ hypoplastic maxilla (relative prominence of mandible)OB-US: √ cloverleaf appearance (coronal view) + bilateral frontal indentations (axial view) of skull√ increased interorbital distance + ocular proptosis√ mild ventriculomegaly

Notes:

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CRUCIATE LIGAMENT INJURY

A.COMPLETE TEAR√ failure to identify ligament√ amorphous areas of high signal intensity on T1WI + T2WI with inability to define ligamentous fibers√ focal discrete complete disruption of all visible fibersB.PARTIAL / INTRASUBSTANCE TEAR√ abnormal signal intensity within substance of ligament with some intact + some discontinuous fibers

Anterior Cruciate Ligament Injury (ACL) Posterior Cruciate Ligament Injury (PCL)

Notes:

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Anterior Cruciate Ligament Injury (ACL) ◊If the ACL appears intact in one of the sagittal oblique sequences discordant findings in other sequences can be disregarded!Site:intrasubstance tear near insertion of femoral condyle (frequently); bone avulsion (rarely)√ hyperintense signal (= focal fluid collection / soft-tissue edema) replacing the tendon substance in acute tear√ mass (hematoma + torn fibers) in intercondylar notch near femoral attachment√ concavity of anterior margin of ligament*Indirect findings:* ◊The indirect signs of ACL injury have a low sensitivity but high specificity!√ bone bruise in lateral compartment (posterolateral tibia + mid lateral femur) in >50%√ deepening of lateral femoral sulcus >1.5 mm√ posterior displacement of posterior horn of lateral meniscus >3.5 mm behind tibial plateau√ anterior translation of tibia (= anterior drawer sign)√ PCL bowing = angle between proximal + distal limbs of PCL <105°False-positive Dx: (1)slice thickness / interslice gap too great(2)adjacent fluid / synovial proliferation(3)cruciate ganglion / synovial cyst*Associated injuries:* meniscal tear (lateral > medial) in 65%
Chronic ACL Tear √ often complete absence of ligament√ bridging fibrous scar within intercondylar notch (simulating an intact ligament) ***Partial ACL Tear*** (15%) ◊ Extremely difficult to diagnose! 40-50% of partial tears are missed on MR! ● positive Lachman test (in 12-30%)√ MR primary signs positive for injury (in 33-43%)

Notes:

Posterior Cruciate Ligament Injury (PCL) *Prevalence:*2-23% of all knee injuries√ midsubstance of PCL most frequently involved (best seen on sagittal images)√ bone avulsion from posterior tibial insertion (<10%), best seen on lateral plain film*Mechanism:* (1)Direct blow to proximal anterior tibia with knee flexed (dashboard accident)√ midsubstance PCL tear√ injury to posterior joint capsule√ bone contusion at anterior tibial plateau + femoral condyles farther posteriorly(2)Hyperextension of knee√ avulsion of tibial attachment of PCL (with preservation of PCL substance)√ ± ACL rupture√ bone contusion in anterior tibial plateau + anterior aspect of femoral condyles(3)Severe ab-/adduction + rotational forces√ + injury to collateral ligaments *Associated with:* coexistent ligamentous injury in 70% anterior cruciate ligament27-38%medial collateral ligament20-23%lateral collateral ligament6-7%medial meniscal tear32-35%lateral meniscal tear28-30%bone marrow injury35-36%effusion64-65% ◊In 30% of cases injury of PCL is isolated! ● posterior tibial laxity ● difficult to evaluate arthroscopically unless ACL torn

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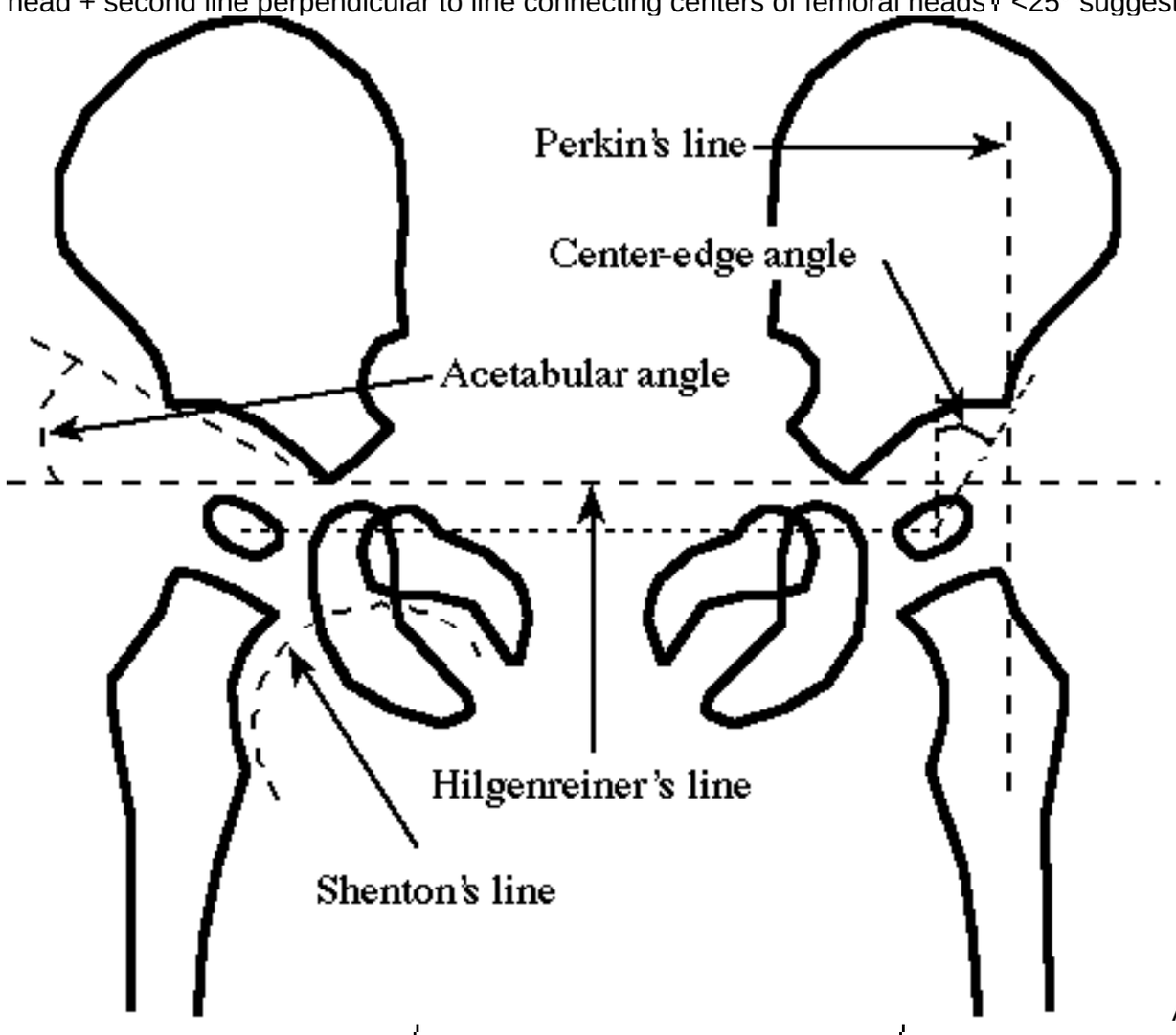
Notes:

DERMATOMYOSITIS

=POLYMYOSITIS=inflammatory myopathy of unknown etiology with diffuse nonsuppurative inflammation of striated muscle*Pathophysiology:*damaged chondroitin sulfate no longer inhibits calcification*Path:*atrophy of muscle bundles followed by edema and coagulation necrosis, fibrosis, calcification*Histo:*mucoid degeneration with round cell infiltrates concentrated around blood vessels*Age:*bimodal: 5-15 and 50-60 years; M:F = 1:2 ● elevated muscle enzymes (creatinine kinase, aldolase) ● myositis-specific autoantibodies: anti-Jo-1(a)anti-aminoacyl-tRNA synthetase ● arthritis, Raynaud phenomenon, fever, fatigue ● interstitial lung disease*Prognosis:*requires prolonged treatment(b)anti-Mi-2 antibodies: ● V-shaped chest rash (= shawl rash) ● cuticular overgrowth*Prognosis:*good response to medication(c)anti-signal recognition particle antibodies ● abrupt onset myositis ± heart involvement@ Skeleton √ linear + confluent calcifications in soft tissues of extremities (quadriceps, deltoid, calf muscles), elbows, knees, hands, abdominal wall, chest wall, axilla, inguinal region) in 75%√ pointing + resorption of terminal tufts √ rheumatoid-like arthritis (rare)√ "floppy-thumb" sign*Cx:*flexion contractures; soft-tissue ulceration@ Chest ● respiratory muscle weakness√ disseminated pulmonary infiltrates (reminiscent of scleroderma)@ Myocardium √ changes similar to skeletal muscle@ GI tract ● dysphagia√ atony + dilatation of esophagus√ atony of small intestines + colon ACUTE FORM ● fever, joint pain, lymphadenopathy, splenomegaly, subcutaneous edema*Prognosis:*death within a few monthsCHRONIC FORM =insidious onset with periods of spontaneous remission and relapse ● low-grade fever, muscular aches + pains, edema ● muscle weakness (due to active inflammation, necrosis, muscle atrophy with fatty replacement, steroid-induced myopathy)◊first symptom in 50% ● skin erythema: heliotrope rash (= dusky erythema of eyelids) with periorbital edema, Gottron sign (= scaly erythematous papules of knuckles, major joints and upper body)◊first symptom in 25%*Cx:*high incidence of malignant neoplasms in GI tract, lung, kidney, ovary, breast*Dx:*muscle biopsy (normal in up to 15%)

Notes:

DEVELOPMENTAL DYSPLASIA OF HIP (DDH)
=CONGENITAL DYSPLASIA OF HIP=abnormal laxity of ligaments + joint capsule resulting in dislocation / subluxation / dysplasia irrespective of prenatal (congenital) or postnatal onset*Etiology:* (a)mechanical:-oligohydramnios (restricted space in utero)-firstborn (tight maternal musculature)-breech position (hyperflexion of hip results in shortening of iliopsoas muscle; L > R)(b)physiologic:-maternal estrogen (not inactivated by immature fetal liver) blocking cross-linkage of collagen fibrils*Incidence:*0.15% of neonates*Increased risk:* (1)infants born in frank breech position (25%; risk of breech:vertex = 6:1)(2)congenital torticollis (10%)(3)skull-molding deformities, neuromuscular disorders (eg, myelodysplasia)(4)family history of DDH (6%): 6% risk for subsequent sibling of normal parents, 36% risk for subsequent sibling of one affected parent; 12% risk for patients own children(5)foot deformities [metatarsus adductus, clubfoot](2%) *Increased prevalence:*females, firstborns (60%), pregnancy with oligohydramnios *Age:*most dislocations probably occur after birth;M:F = 1:4 -Caucasians > Blacks *Classification:* Type 1=DISLOCATABLE UNSTABLE HIP*Incidence:*0.25-0.85% of all newborn infants;2/3 are firstborns√ slight increase in femoral anteversion√ mild marginal abnormalities in acetabular cartilage√ early labral eversion*Prognosis:*60% will become stable after 1 week; 88% will become stable by age of 2 monthsType 2=SUBLUXED HIP√ loss of femoral head sphericity√ increased femoral anteversion√ early labral inversion√ shallow acetabulumType 3=DISLOCATED HIP√ accentuated flattening of femoral head√ shallow acetabulum√ limbus formation (= inward growth + hypertrophy of labrum) ● positive Ortolani (reduction) test = reduction of proximal femur into the acetabulum by progressive abduction of flexed hips ± associated with audible "click" ● positive Barlow (dislocation) test = displacement of proximal femur by progressive adduction with downward pressure on flexed hips ● Alli sign = Galeazzi sign = affected knee is lower with knees bent in supine position ● asymmetric skin folds + shortening of thigh on dislocated side ● Trendelenburg test = visible drooping + shortening on dislocated side with child standing on both feet, then one footLocation:left:right:bilateral = 11:1:4Radiologic lines: 1.Line of Hilgenreiner =line connecting superolateral margins of triradiate cartilages2.Acetabular angle / index=slope of acetabular roof = angle that lies between Hilgenreiners line and a line drawn from most superolateral ossified edge of acetabulum to superolateral margin of triradiate cartilage√ >30° strongly suggests dysplasia3.Perkins line=vertical line to Hilgenreiners line through the lateral rim of acetabulum4.Shentons curved line=arc formed by inferior surface of superior pubic ramus (= top of obturator foramen) + medial surface of proximal femoral metaphysis to level of lesser trochanter√ disruption of line (DDx: coxa valga)5.Center-edge angle=angle subtended by one line drawn from the acetabular edge to center of femoral head + second line perpendicular to line connecting centers of femoral heads√ <25° suggests femoral head instability

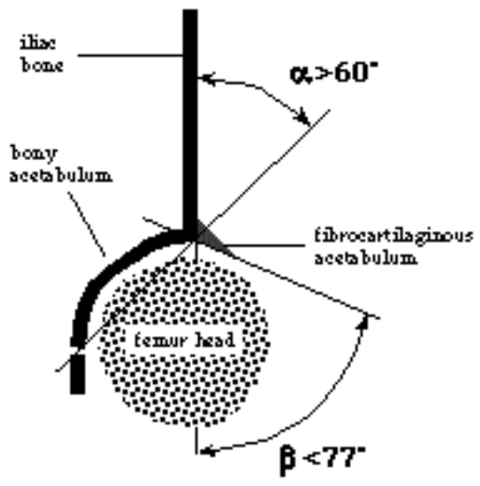


AP pelvic radiograph: >6-8 weeks of age (von Rosen view = legs abducted 45° + thighs internally rotated) √ proximal + lateral migration of femur√ eccentric position of femoral epiphysis (position estimated by a circle drawn with a diameter equivalent to width of femoral neck)√ interrupted discontinuous arc of Shentons line√ line drawn along axis of femoral shaft will not pass through upper edge of acetabulum but intersect the anterior-superior iliac spine (during Barlow maneuver)√ apex of metaphysis lateral to edge of acetabulum√ femoral shaft above horizontal line drawn through the Y-synchondroses√ unilateral shortening of vertical distance from femoral ossific nucleus / femoral metaphysis to Hilgenreiners line√ femoral ossific nucleus / medial beak of femoral metaphysis outside inner lower quadrant of coordinates established by Hilgenreiners + Perkins lines√ acetabular dysplasia = shallow incompletely developed acetabulum√ development of false acetabulum√ delayed ossification of femoral epiphysis (usually evident between 2nd and 8th month of life)
US (practical only up to 8-10 months of age): √ direct visualization of unossified femoral head

Sonographic Hip Types

Type	Description	alpha- / beta angle	
1	mature hip	>60°	
1A	narrow cartilaginous roof		<55°
1B	wide cartilagenous roof		>55°
2	deficient bony acetabulum		
2A	physiologic <3 months	50 – 59°	
2B	delayed ossification >3 months		
2C	concentric but unstable; *critical range*	43 – 49°	70 – 77°
2D	decentered = subluxed		>77°
3	eccentric = dislocated	<43°	
4	severe dysplasia with inverted labrum		

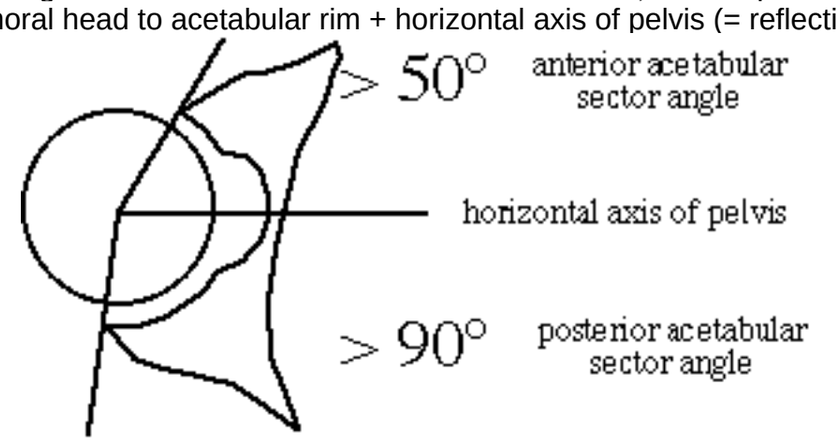
(1) alpha angle = angle between straight lateral edge of ilium + bony acetabular margin (on coronal view); determines sonographic hip type
(2) beta angle = angle between straight lateral edge of ilium + fibrocartilaginous acetabulum; determines nuances of sonographic hip type



Coronal View of Left Hip

√ femoral head position at rest in neutral position: normal / subluxed = decentered / dislocated = eccentric√ hip instability under motion + stress maneuvers: normal / lax = subluxable / subluxed / dislocatable = unstable / dislocated reducible / dislocated irreducible√ subluxability up to 6 mm is normal in newborns (still under influence

of maternal hormones); decreasing to 3 mm by 2nd day of life◊examination should be performed >2 weeks of age!√ dislocatable (= concentric but unstable) hip can be pushed out of hip joint (Barlow positive) by "piston" maneuver (= pushing / pulling in AP direction with hip flexed)√ posterior + superior dislocation of head against ilium √ dislocated (= eccentric) hip can be reduced (Ortolani positive)√ hypoechoic femoral head not centered over triradiate cartilage between pubis + ischium (on transverse view)√ increased amount of soft-tissue echoes ("pulvinar") between femoral head and acetabulum√ cartilaginous acetabular labrum interposed between head and acetabulum (inverted labrum)√ disparity in presence + size of ossific nucleus√ disparity in size of femoral head√ equator sign = <50% of femoral head lies medial to line drawn along iliac bone (on coronal view);58% to 33% coverage is indeterminate, <33% coverage is abnormal√ delayed ossification of acetabular corner √ wavy contour of bony acetabulum with only slight curvature√ abnormally acute alpha angle (= angle between straight lateral edge of ilium + bony acetabular margin)◊ 4°-6° interobserver variation!*Prognosis:*alpha-angle <50° at birth / 50° - 59° after 3 months indicates significant risk for dislocation without treatment; follow-up at 4-week intervals are recommendedCT: sector angle = angle between line drawn from center of femoral head to acetabular rim + horizontal axis of pelvis (= reflection of acetabular support) √ anterior acetabular sector angle <50°√ posterior acetabular sector angle <90°



Acetabular sector angles (in normal right hip)

Cx:(1)Avascular necrosis of femoral head(2)Intra-articular obstacle to reduction(a)pulvinar = fibrofatty tissue at apex of acetabulum(b)hypertrophy of ligamentum teres(c)labral hypertrophy / inversion(3)Extra-articular obstacle to reduction(a)iliopsoas tendon impingement on anterior joint capsule with infolding of joint capsule*Rx:*(1)Flexion-abduction-external rotation brace (Pavlik harness) / splint / spica cast(2)Femoral varus osteotomy(3)Pelvic (Salter) / acetabular rotation(4)Increase in acetabular depth (Pemberton)(5)Medialization of femoral head (Chiari)

Notes:

DESMOPLASTIC FIBROMA
=INTRAOSSEOUS DESMOID TUMOR=rare locally aggressive benign neoplasm of bone with borderline malignancy resembling soft-tissue desmoids / musculoaponeurotic fibromatosis*Incidence:*107 cases in world literature*Histo:*intracellular collagenous material in fibroblasts with small nuclei*Age:*mean of 21 years (range 15 months to 75 years); in 90% <30 years; M:F = 1:1 ● slowly progressive pain + local tenderness ● palpable mass*Location:*mandible (26%), ilium (14%), >50% in long bones (femur [14%], humerus [11%], radius [9%], tibia [7%], clavicle), scapula, vertebra, calcaneus*Site:*central meta- / diaphyseal (if growth plate open); may extend into epiphysis with subarticular location (if growth plate closed)√ geographic (96%) / moth-eaten (4%) bone destruction without matrix mineralization√ narrow (96%) / poorly defined (4%) zone of transition√ no marginal sclerosis (94%)√ residual columns of bone with "pseudotrabeculae" are CLASSIC (91%)√ bone expansion (89%); may grow to massive size (simulating aneurysmal bone cyst / metastatic renal cell carcinoma)√ breach of cortex + soft-tissue mass (29%)*Cx:*pathologic fracture (9%)*Prognosis:*52% rate of local recurrence*Rx:*wide excision*DDx:*(1)Giant cell tumor (round rather than oval, may extend into epiphysis + subchondral bone plate)(2)Fibrous dysplasia (occupies longer bone, contains mineralized matrix, often with sclerotic rim)(3)Aneurysmal bone cyst (eccentric blowout appearance rather than fusiform)(4)Chondromyxoid fibroma (eccentric with delicate marginal sclerosis + scalloped border)

Notes:

DIASTROPHIC DYSPLASIA
=DIASTROPHIC DWARFISM = EPIPHYSEALDYSOSTOSIS= autosomal recessive severe rhizomelic dwarfism secondary to generalized disorder of cartilage followed by fibrous scars + ossifications ● diastrophic = "twisted" habitus ● "cauliflower ear" = ear deformity from inflammation of pinna ● laryngomalacia ● lax + rigid joints with contractures ● normal intellectual development@ Axial skeleton √ cleft palate (25%)√ cervical spina bifida occulta√ hypoplasia of odontoid√ severe progressive kyphoscoliosis of lumbar spine (not present at birth)√ narrowed interpedicular space in lumbar spine√ short + broad bony pelvis√ posterior tilt of sacrum@ Extremities √ severe micromelia (predominantly rhizomelic= humerus + femur shorter than distal long bones √ widened metaphysis√ flattened epiphysis (retardation of epiphyseal ossification) with invagination of ossification centers into distal ends of femora√ multiple joint flexion contractures (notably of major joints)√ dislocation of one / more large joints (hip, elbow), lateral dislocation of patella√ coxa vara (common)√ medially bowed metatarsals√ clubfoot = severe talipes equinovarus√ ulnar deviation of hands√ oval + hypoplastic 1st metacarpal bone + abducted proximally positioned thumb = "hitchhikers thumb" (CHARACTERISTIC)√ bizarre carpal bones with supernumerary centers√ widely spaced fingersOB-US: √ proportionately shortened long bones√ hitchhiker thumb√ clubfeet√ joint contractures√ abnormal spinal curvature*Prognosis:*death in infancy (due to abnormal softening of tracheal cartilage)

Notes:

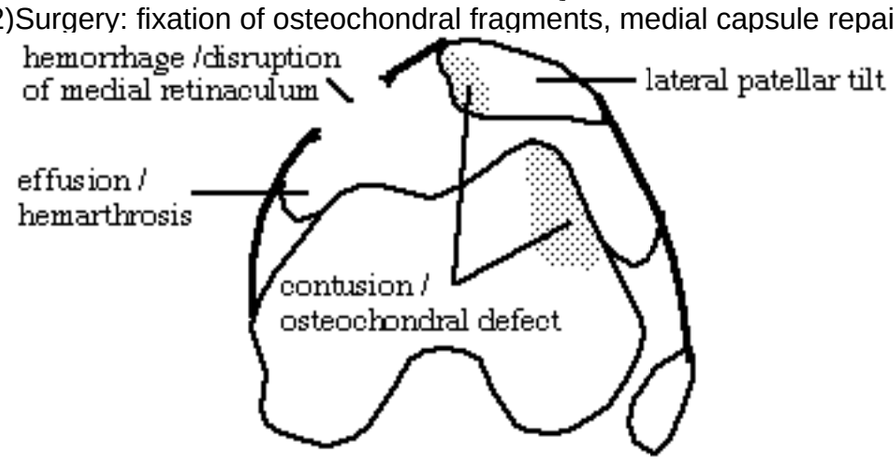
DIFFUSE IDIOPATHIC SKELETAL HYPEROSTOSIS
=DISH = FORESTIER DISEASE = ANKYLOSING HYPEROSTOSIS= common ossifying diathesis characterized by bone proliferation at sites of tendinous + ligamentous attachment (enthesis) *Etiology:* (1)may be caused by altered vitamin A metabolism (elevated plasma levels of unbound retinol)(2)long-term ingestion of retinoid derivatives for dermatologic disorders (eg, Accutane®);? hypertrophic variant of spondylosis deformans *Age:*>50 years; M:F = 3:1 ● pain, tenderness in extraspinal locations ● restricted motion of vertebral column ● hyperglycemia ● positive HLA-B27 in 34%Location:lower thoracic > lower cervical > entire lumbar spine √ anterior + lateral right-sided osteophytes of vertebral column (not on left because of aorta)√ disk spaces well preserved, no apophyseal ankylosis, no sacroiliitis√ flowing ossification along anterior / anterolateral aspect of at least 4 contiguous vertebral bodies√ "whiskering" at iliac crest, ischial tuberosity, trochanters√ spurs of olecranon process of ulna + calcaneus (plantar + posterior surface) + anterior surface of patella√ broad osteophytes at lateral acetabular edge, inferior portions of sacroiliac joints, superior aspect of symphysis pubis√ ossification of iliolumbar + sacrotuberous + sacroiliac ligaments (high probability for presence of spinal DISH, DDx: fluorosis)√ ossification of coracoclavicular ligament, patellar ligament, tibial tuberosity, interosseous membranes√ increased incidence of hyperostosis frontalis interna*DDx:*(1)Fluorosis (increased skeletal density)(2)Acromegaly (posterior scalloping, skull features)(3)Hypoparathyroidism(4)X-linked hypophosphatemic vitamin D-resistant rickets(5)Ankylosing spondylitis (squaring of vertebral bodies, coarser syndesmophytes, sacroiliitis, apophyseal alteration)(6)Intervertebral osteochondrosis (vacuum phenomenon, vertebral body marginal sclerosis, decreased intervertebral disk height)

Notes:

Hip Dislocation *Incidence:*5% of all dislocationsA.POSTERIOR HIP DISLOCATION (80-85%)Mechanism:classical dashboard injury (= flexed knee strikes dashboard)*Associated with:*fractures of posterior rim of acetabulum, femoral headB.ANTERIOR HIP DISLOCATION (5-10%)1.anterior obturator dislocation2.superoanterior / pubic hip dislocation*Associated with:*fractures of acetabular rim, greater trochanter, femoral neck, femoral head (characteristic depression on posterosuperior and lateral portion)C.CENTRAL ACETABULAR FRACTURE-DISLOCATIONMechanism:force applied to lateral side of trochanter

Notes:

Patellar Dislocation = TRANSIENT LATERAL PATELLAR DISLOCATION *Incidence:*2-3% of all knee injuries*Mechanism:*during attempt to slow forward motion while pivoting medially on a planted foot; internal rotation of femur and quadriceps contraction produces a net lateral force*Associated with:*medial meniscal tear / major ligamentous injury in 31%*Age:*young physically active people ● hemarthrosis (most common cause of hemarthrosis in young conscripts) ● swelling + tenderness of medial retinaculum◊>50% not clinically diagnosed initially!√ increased signal intensity / thickening / disruption of medial patellar retinaculum√ lateral patellar tilt√ contusion / microfracture / osteochondral injury of nonarticular surface of lateral femoral condyle + medial articular surface of patella√ hemarthrosis*Rx:*(1)Temporary immobilization + rehabilitation: successful in 75%(2)Surgery: fixation of osteochondral fragments, medial capsule repair, lateral retinacular release, vastus medialis et lateralis rearrangement, medial retinaculum reefing



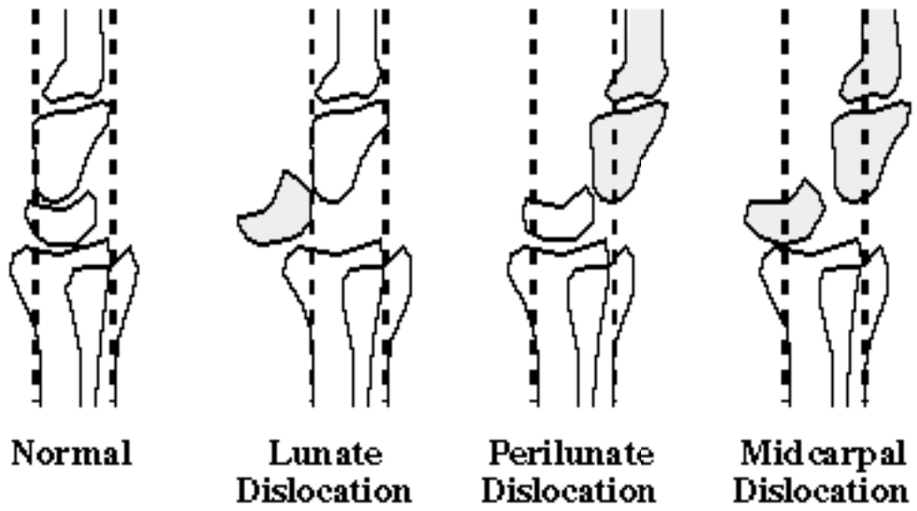
MR Imaging Signs of Patellar Dislocation

Notes:

Shoulder Dislocation ***Sternoclavicular dislocation*** (3%) Acromioclavicular dislocation (12%) Glenohumeral dislocation (85%) ◊Glenohumeral joint dislocations make up >50% of all dislocations!A.ANTERIOR / SUBCORACOID SHOULDER DISLOCATION (96%)*Types:*subcoracoid, subglenoid, subclavicular, intrathoracic*Mechanism:*external rotation + abduction; 40% recurrent*Age:*in younger individuals*May be associated with:* √ fracture of greater tuberosity (15%)√ Bankart lesion = fracture of anterior glenoid rim (originally only referring to injury of anterior band of glenohumeral ligament)√ fracture of anterior rim of glenoid√ Hill-Sachs defect (50%) = depression fracture of posterolateral surface of humeral head at / above level of coracoid process (impaction against glenoid rim in subglenoid type)B.POSTERIOR SHOULDER DISLOCATION (2-4%)*Cause:*(a)traumatic: convulsive disorders /electric shock therapy(b)nontraumatic: voluntary, involuntary, congenital, developmental*Types:*subacromial, subglenoid, subspinous◊In >50% unrecognized initially + subsequently misdiagnosed as frozen shoulder!◊Average interval between injury and diagnosis is 1 year!√ rim sign (66%) = distance between medial border of humeral head + anterior glenoid rim <6 mm*May be associated with:* √ trough sign (75%) = "reverse Hill-Sachs" = compression fracture of anteromedial humeral head (tangential Grashey view of glenoid!)√ fracture of posterior glenoid rim√ avulsion fracture of lesser tuberosityC.INFERIOR SHOULDER DISLOCATION (1-2%)=LUXATIO ERECTA = extremity held over head in fixed position with elbow flexed*Mechanism:*severe hyperabduction of arm resulting in impingement of humeral head against acromion√ humeral articular surface faces inferiorly*Cx:*rotator cuff tear; fracture of acromion ± inferior glenoid fossa ± greater tuberosity; neurovascular injuryD.SUPERIOR SHOULDER DISLOCATION (<1%)=humeral head driven upward through rotator cuff*May be associated with:*fracture of humerus, clavicle, acromion*DDx:*drooping shoulder (transient phenomenon after fracture of surgical neck of humerus due to hemarthrosis / muscle imbalance)

Notes:

Wrist Dislocation *Mechanism:* fall on outstretched hand*Incidence:*10% of all carpal injuries***Lunate Dislocation*** Perilunate Dislocation 2-3 times more common than lunate dislocation accompanied by fracture in 75% (= transscaphoid perilunate dislocation)√ most commonly dorsal dislocation***Rotary Subluxation of Scaphoid*** =tearing of interosseous ligaments of lunate, scaphoid, capitate*Mechanism:*acute dorsiflexion of wrist; may be associated with rheumatoid arthritis√ gap >4 mm between scaphoid + lunate (PA view)√ foreshortening of scaphoid√ ring sign of distal pole of scaphoid***Midcarpal Dislocation***



Notes:

DOWN SYNDROME

=MONGOLISM = TRISOMY 21 (95% nondisjunction,5% translocation)*Incidence:*1:870 liveborn infants, most common karyotype / chromosomal abnormality in U.S. ● mental retardation ● hypotonia in infancy ● characteristic facies ● Simian crease@ Skull √ hypotelorism√ persistent metopic suture (40-79%) after age 10√ hypoplasia of sinuses + facial bones√ microcrania (brachycephaly)√ delayed closure of sutures + fontanelles√ dental abnormalities (underdeveloped tooth No. 2)√ flat-bridged nose@Axial skeleton√ atlantoaxial subluxation (25%)√ anterior scalloping of vertebral bodies√ "squared vertebral bodies" = centra high and narrow= positive lateral lumbar index (ratio of horizontal to vertical diameters of L2) @Chest√ congenital heart disease (40%): endocardial cushion defect, VSD, tetralogy of Fallot√ hypersegmentation of manubrium= 2-3 ossification centers (90%) √ gracile ribs; 11 pairs of ribs (25%)@Pelvis√ flaring of iliac wings (decreased iliac angle + index)= "Mickey Mouse ears" / "elephant ears" √ flattening of acetabular roof (decreased acetabular angle)√ tapering of ischial rami@Extremities√ metaphyseal flaring√ clinodactyly (50%); widened space between first two digits of hands + feet√ hypoplastic and triangular middle + distal phalanges of 5th finger = acromicria (DDx: normal individuals, cretins, achondroplastic dwarfs)√ pseudoepiphyses of 1st + 2nd metacarpals@Gastrointestinal√ umbilical hernia√ "double bubble" sign (8-10%) = duodenal atresia / stenosis / annular pancreas√ tracheoesophageal fistula√ anorectal anomalies√ Hirschsprung diseaseOB-US: ● triple-marker screening test: ● low maternal alpha-fetoprotein (20-30%) ● increased HCG (DDx: decreased in trisomy 18) ● decreased unconjugated estriol (ue3) ● advanced maternal age◊in 1:385 livebirths for women >35 years of age◊HOWEVER: 80% of fetuses with Down syndrome are born to mothers <35 years of age√ occipital-nuchal skin thickening ≥6 mm during 19-24 weeks (in 45-80%) / ≥5 mm during 14-18 weeks on transcerebellar diameter view (69% positive predictive value, 0.5% false positives)√ ratio of measured-to-expected femur length ≤0.91 [expected femur length: -9.3105 + 0.9028 x BPD] (sensitivity 40%, specificity 95%, false-positive rate of 2-7%, 0.3% PPV for low-risk population [1:700], 1% PPV for high-risk population [1:250])√ elevated BPD / femur ratio (secondary to short femur)√ ratio of measured-to-expected humerus length ≤0.90 [expected humerus length: -7.9404 + 0.8492 x BPD] (1-2% PPV for low-risk population; 3% PPV for high-risk population)√ major structural malformations: √ VSD / complete AV canal (50%)√ cystic hygroma, resolved by 20th week MA√ omphalocele√ double bubble of duodenal atresia (8-10%), not apparent before 22 weeks GA√ hydrothorax√ mild cerebral ventricular dilatation√ agenesis of corpus callosum√ imperforate anus√ mild fetal pyelectasis (17-25%)√ hyperechoic bowel at <20 weeks GA (15%, in 0.6% of normals)√ intracardiac echogenic focus, usually in left ventricle= thickening of papillary muscle (18%, in 5% of normals) √ sandal-gap deformity = separation of great toe (45%)√ hypoplasia of middle phalanx of 5th digit resulting in clinodactyly (= inward curve) in 60%√ flared ilium = iliac wings rotated toward coronal plane at SIJ describing an angle of >70° with each other√ brachycephaly√ small cerebellum√ IUGR (in 30%)√ polyhydramnios*Cx:*leukemia (increased frequency 3-20 x)

Notes:

DYSCHONDROSTEOSIS

=LÉRI-LAYANI-WEILL SYNDROME = mesomelic long-bone shortening (forearm + leg); autosomal dominantM:F = 1:4 ● limited motion of elbow + wrist√ bilateral Madelung deformity√ radial shortening in relation to ulna√ bowing of radius laterally + dorsally√ dorsal subluxation of distal end of ulna√ carpal wedging between radius + ulna (due to triangular shape of distal radial epiphysis + underdevelopment of ulna)*DDx:*Pseudo-Madelung deformity (from trauma / infection)

Notes:

DYSPLASIA EPIPHYSEALIS HEMIMELICA
=TREVOR DISEASE = TARSOEPIPHYSEAL ACLASIS=eccentric usually medial epiphyseal cartilaginous overgrowth of one / more epiphyses; spontaneous occurrence*Age:*2-4 years; M > F*May be associated with:* hemihypertrophy ● limitation of joint mobility (due to localized painless mass)Location:localized to tarsus, carpus, knee, ankle;occasionally generalized√ osteochondroma-like growth from one side of epiphysis*Cx:*genu valgum*DDx:*osteochondroma

Notes:

ECHINOCOCCUS OF BONE

Occurs occasionally in the U.S.; usually in foreign-born individuals; bone involvement in 1% *Histo:*no connective tissue barrier; daughter cysts extend directly into bone@Pelvis, sacrum, rarely long tubular bones√ round / irregular regions of rarefaction√ multiloculated lesion (bunch of grapes)√ no sharp demarcation (DDx: chondroma, giant cell tumor) with secondary infection√ thickening of trabeculae with generalized perifocal condensation√ cortical breakthrough with soft-tissue mass@Vertebra√ sclerosis without pathologic fracture√ intervertebral disks not affected√ vertebral lamina often involved√ frequently involvement of adjacent ribs

Notes:

EHLERS-DANLOS SYNDROME
=group of autosomal dominant diseases of connective tissue characterized by abnormal collagen synthesis*Types:*10 types have been described that differ clinically, biochemically, and genetically*Age:*present at birth; predominantly in males ● hyperelasticity of skin ● fragile brittle skin with gaping wounds and poor healing ● molluscoid pseudotumors over pressure points ● hyperextensibility of joints ● joint contractures with advanced age ● bleeding tendency (fragility of blood vessels) ● blue sclera, microcornea, myopia, keratoconus, ectopia lentis@Soft tissues√ multiple ovoid calcifications (2-10 mm) in subcutis / in fatty cysts ("spheroids"), most frequently in periarticular areas of legs√ ectopic bone formation@Skeleton√ hemarthrosis (particularly in knee)√ malalignment / subluxation / dislocation of joints on stress radiographs√ recurrent dislocations (hip, patella, shoulder, radius, clavicle)√ precocious osteoarthrosis (predominantly in knees)√ ulnar synostosis√ kyphoscoliosis√ spondylolisthesis√ spina bifida occulta@Chest√ diaphragmatic hernia√ panacinar emphysema + bulla formation√ tracheobronchomegaly + bronchiectasis@Arteries√ aneurysm of great vessels, aortic dissection, tortuosity of arch, ectasia of pulmonary arteries◊AORTOGRAPHY CONTRAINDICATED!(Cx following arteriography: aortic rupture, hematomas) @GI tract√ ectasia of gastrointestinal tract

Notes:

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ELASTOFIBROMA
=benign tumorlike lesion forming as a reaction to mechanical friction*Incidence:*in 24% of women + 11% of men >55 years (autopsy study)*Age:*elderly; M:F = 1:2*Histo:*enlarged irregular serrated elastic hypereosinophilic fibers, collagen, scattered fibroblasts, occasional lobules of adipose tissue ● asymptomatic ● may remain clinically inapparentLocation:between inferior margin of scapula + posterior chest wall; bilateral in 25%√ inhomogeneous poorly defined lesion of soft-tissue attenuation similar to muscle√ well-defined intermediate-signal intensity lesion with interlaced areas of fat-intensity signal on T1WI + T2WI

Notes:

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ENCHONDROMA
=benign cartilaginous growth in medullary cavity; bones preformed in cartilage are affected (NOT skull)*Age:*10-30 years; M:F = 1:1*Histo:*lobules of hyaline cartilage ● usually asymptomatic, painless swellingLocation:(frequently multiple = enchondromatosis)(a)in 40% small bones of wrists + hand (most frequent tumor here), distal + mid aspects of metacarpals, proximal / middle phalanges(b)femur, tibia, humerus, radius, ulna, foot, ribSite:central + diaphyseal, epiphysis only affected after closure of growth plate√ oval / round lucency near epiphysis with fine marginal line√ scalloped endosteum√ ground-glass appearance√ calcification: pinhead, stippled, flocculent, "rings and arcs" pattern√ bulbous expansion of bone with thinning of cortex√ Madelung deformity = bowing deformities of limb, discrepant length√ NO cortical breakthrough / periosteal reaction*Cx:*(1)pathologic fracture(2)malignant degeneration in long-bone enchondromas in 15-20%*DDx:*(1)Epidermoid inclusion cyst (phalangeal tuft, Hx of trauma, more lucent)(2)Unicameral bone cyst (rare in hands, more radiolucent)(3)Giant cell tumor of tendon sheath (commonly erodes bone, soft-tissue mass outside bone)(4)Fibrous dysplasia (rare in hand, mostly polyostotic)(5)Bone infarct(6)Chondrosarcoma

Notes:

ENCHONDROMATOSIS
=OLLIER DISEASE = DYSCHONDROPLASIA=MULTIPLE ENCHONDROMATOSIS=nonhereditary failure of cartilage ossification*Age:*early childhood presentation ● growth disparity with leg / arm shortening ● hand + feet deformityLocation:predominantly unilateral monomelic distribution (a) localized (b) regional (c) generalized√ rounded masses / columnar streaks of decreased density from epiphyseal plate into diaphysis of long bones = cartilaginous rests√ bony spurs pointing toward the joint (DDx: exostosis points away from it)√ cartilaginous areas show punctate calcifications with age√ associated with dwarfing of the involved bone due to impairment of epiphyseal fusion√ clublike deformity of metaphyseal region√ cartilaginous metaphyseal expansion with cortical expansion + thinning + breakthrough√ bowing deformities of limb bones√ discrepancy in length = Madelung deformity (radius, ulna)√ small bones of feet + hands: aggressive deforming tumors that may break through cortex secondary to tendency to continue to proliferate√ fanlike radiation of cartilage from center to crest of ilium*Cx:*sarcomatous transformation (in 25-50%): osteosarcoma (young adults); chondro- / fibrosarcoma (in older patients)

Maffucci Syndrome

Notes:

Maffucci Syndrome =nonhereditary mesodermal dysplasia characterized by enchondromatosis + multiple soft-tissue cavernous hemangiomas*Age:*generally not before puberty; M > F ● multiple nodules particularly on digits + extremities (cavernous hemangiomas) ● normal intelligenceLocation:unilateral involvement / marked asymmetry; distinct predilection for hands + feet√ phleboliths may be present√ striking tendency for enchondromata to be very large projecting into soft tissues√ growth disturbance of long bones (common)*Cx:*(1)malignant transformation of(a)enchondroma to chondrosarcoma (15-20%)(b)soft-tissue hemangioma to vascular sarcoma (in 3-5%)(2)increased prevalence of ovarian carcinoma, pancreatic carcinoma, CNS glioma

Notes:

ENGELMANN-CAMURATI DISEASE
=PROGRESSIVE DIAPHYSEAL DYSPLASIA=ENGELMANN DISEASE = RIBBING DISEASE (as forme fruste)Autosomal dominant*Age:*5-25 years, M > F ● neuromuscular dystrophy = delayed walking (18-24 months) with wide-based waddling gait; often misdiagnosed as muscular dystrophy / poliomyelitis ● weakness + easy fatigability ● bone pain + tenderness usually in midshaft of long bones ● underdevelopment of muscles secondary to malnutrition ● NORMAL laboratory valuesLocation:usually symmetrical; NO involvement of hands, feet, ribs, scapulae@Skull (initially affected)√ amorphous increase in density at base of skull@Long bones (bilateral symmetrical distribution)√ fusiform enlargement of diaphyses with cortical thickening (endosteal + periosteal accretion of mottled new bone) and progressive obliteration of medullary cavity; symmetrical involvement√ progression of lesions along long axis of bone toward either end√ abrupt demarcation of lesions (metaphyses + epiphyses spared)√ relative elongation of extremities√ NORMAL epiphyses + metaphyses*DDx:*(1)Chronic osteomyelitis (single bone)(2)Hyperphosphatasemia (high alkaline phosphatase levels)(3)Paget disease (age, new-bone formation, increased alkaline phosphatase)(4)Infantile cortical hyperostosis (fever; mandible, rib, clavicles; regresses, <1 year of age)(5)Fibrous dysplasia (predominantly unilateral, subperiosteal new bone)(6)Osteopetrosis (very little bony enlargement)(7)Vitamin A poisoning

Notes:

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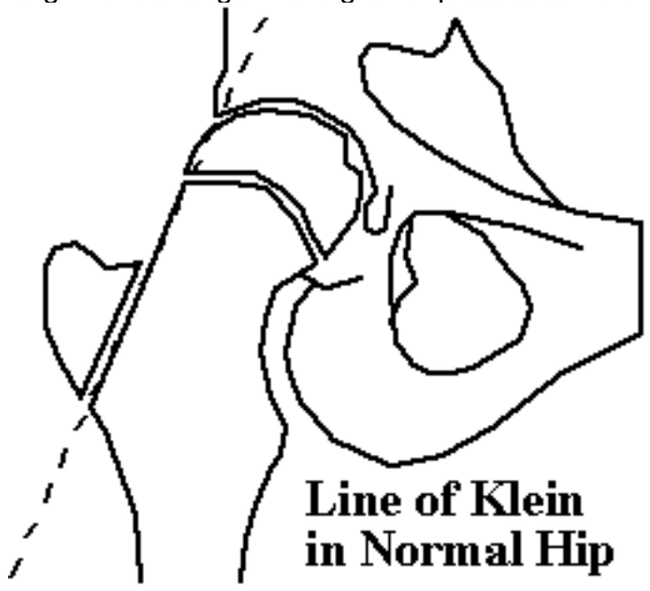
EPIDERMOID INCLUSION CYST
=INTRAOSSEOUS KERATIN CYST = IMPLANTATION CYST*Age:*2nd-4th decade;M > F*Histo:*stratified squamous epithelium, keratin, cholesterol crystals (soft white cheesy contents) ● history of trauma (implantation of epithelium under skin with secondary bone erosion) ● asymptomaticLocation:superficially situated bones such as calvarium (typically in frontal / parietal bone), phalanx (usually terminal tuft of middle finger), L > R hand, occasionally in foot√ well-defined round osteolysis with sclerotic margin√ cortex frequently expanded + thinned√ NO calcifications / periosteal reaction / soft-tissue swelling√ pathologic fracture often without periosteal reaction *DDx:*(a)in finger: glomus tumor, enchondroma (rare in terminal phalanx)(b)in skull: infection, metastasis (poorly defined), eosinophilic granuloma (beveled margin)

Notes:

EPIPHYSEOLYSIS OF FEMORAL HEAD
=SLIPPED CAPITAL FEMORAL EPIPHYSIS=atraumatic fracture through hypertrophic zone of physeal plate*Frequency:*2:100,000 people*Etiology:* growth spurt, renal osteodystrophy, rickets, childhood irradiation, growth hormone therapy, trauma (Salter-Harris type I epiphyseal injury)*Pathogenesis:*widening of physeal plate during growth spurt + change in orientation of physis from horizontal to oblique increases shear forces*Age:*overweight 8-17 year old boys (mean age for boys 13, for girls 11 years); M > F; black > white*Associated with:* (a)malnutrition, endocrine abnormality, developmental dysplasia of hip (during adolescence)(b)delayed skeletal maturation (after adolescence) ● hip pain (50%) / knee pain (25%) for 2-3 weeksLocation:usually unilateral; bilateral in 20-37% (at initial presentation in 9-18%)√ widening of epiphyseal plate (preslip phase)√ irregularity + blurring of physeal physis√ demineralization of neck metaphysis√ posteromedial displacement of head (acute slip)√ decrease in neck-shaft angle with alignment change in the growth plate to a more vertical orientation√ line of Klein (= line drawn along superior edge of femoral neck) fails to intersect the femoral head



√ epiphysis appears smaller due to posterior slippage:early slips are best seen on cross-table LAT viewCAVE:positioning into a frogleg view may cause further displacement√ sclerosis + irregularity of widened physis (chronic slip)√ metaphyseal blanch sign = area of increased opacity in proximal part of metaphysis (healing response) Grading (based on femoral head position): milddisplaced by <1/3 of metaphyseal diametermoderatedisplaced by 1/3-2/3 of diameterseveredisplaced by >2/3 of metaphyseal diameter *Cx:*(1)Chondrolysis = acute cartilage necrosis (7-10%)=rapid loss of >50% of thickness of cartilage√ joint space <3 mm(2)Avascular necrosis of femoral head (15%) risk increases with advanced degree of slip, delayed surgery for acute slip, anterior pin placement, large number of fixation pins, subcapital osteotomy(3)Pistol-grip deformity = broadening + shortening of femoral neck in varus deformity(4)Degenerative osteoarthritis (90%)(5)Limb-length discrepancy due to premature physeal closure*Rx:*(1) limitation of activity, (2) prophylactic pinningAttempted reductions increase risk of AVN!

Notes:

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ESSENTIAL OSTEOLYSIS

Progressive slow bone-resorptive disease *Histo:*proliferation + hyperplasia of smooth muscle cells of synovial arterioles√ progressive osteolysis of carpal + tarsal bones √ thinned pointed proximal ends of metacarpals + metatarsals √ elbows show same type of destruction√ bathyrocephalic depression of base of skull*DDx:*(1) Massive osteolysis = Gorham disease (local destruction of contiguous bones, usually not affecting hands / feet) (2) Tabes dorsalis (3) Leprosy (4) Syringomyelia (5) Scleroderma (6) Raynaud disease (7) Regional posttraumatic osteolysis (8) Ulcero-mutilating acropathy (9) Mutilating forms of rheumatoid arthritis (10) Acrodynia mutilante (nonhereditary)

Notes:

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EWING SARCOMA

=EWING TUMOR*Incidence:*4-10% of all bone tumors (less common than osteo- / chondrosarcoma); most common malignant bone tumor in children◊Clinically, radiologically, and histologically very similar to PNET!*Histo:* small round cells, uniformly sized + solidly packed (DDx: lymphoma, osteosarcoma, myeloma, neuroblastoma, carcinoma, eosinophilic granuloma) invading medullary cavity and entering subperiosteum via Haversian canals producing periostitis, soft-tissue mass, osteolysis; glycogen granules present (DDx to reticulum cell sarcoma); absence of alkaline phosphatase (DDx to osteosarcoma) *Age:*peak 15 years (range 5 months to 54 years); in 30% <10 years; in 39% 11-15 years; in 31% >15 years; in 50% <20 years; in 95% 4-25 years;M:F = 2:1; Caucasians in 96% ● severe localized pain ● soft-tissue mass ● fever, leukocytosis, anemia (in early metastases) simulating infectionLocation: femur (25%), pelvis-ilium (14%), tibia (11%), humerus (10%), fibula (8%), ribs (6%) (a)long bones in 60%:metadiaphysis (44%), middiaphysis (33%), metaphysis (15%), metaepiphyseal (6%), epiphyseal (2%); usually no involvement of epiphysis as tumor originates in medullary cavity with invasion of Haversian system (b)flat bones in 40%: pelvis, scapula, skull, vertebrae (in 3-10%; sacrum > lumbar > thoracic > cervical spine); ribs (in 7% > age 10; in 30% < age 10)◊>20 years of age predominantly in flat bones◊<20 years of age predominantly in cylindrical bones (tumor derived from red marrow) √ 8-10 cm long lytic lesion in shaft of long bone (62% lytic, 23% mixed density, 15% dense)√ mottled "moth-eaten" destructive permeative lesion (72%) (late finding)√ penetration into soft tissue (55%) with preservation of tissue planes (DDx: osteomyelitis with diffuse soft-tissue swelling)√ early fusiform lamellated "onionskin" periosteal reaction (53%) / spiculated = "sunburst" / "hair-on-end" (23%), Codman triangle√ cortical thickening (16%)√ cortical destruction (18%)√ ± cortical sequestration√ reactive sclerotic new bone (30%)√ bone expansion (12%)√ Ewing sarcoma of rib: disproportionately large inhomogeneous soft-tissue mass with large intrathoracic + minimal extrathoracic componentMetastases to:lung, bones, regional lymph nodes in 11-30% at time of diagnosis,in 40-45% within 2 years of diagnosis*Cx:*pathologic fracture (5-14%)*Prognosis:*60-75% 5-year survival*DDx:*(1)Multiple myeloma (older age group)(2)Osteomyelitis (duration of pain <2 weeks)(3)Eosinophilic granuloma (solid periosteal reaction)(4)Osteosarcoma (ossification in soft tissue, near age 20, no lamellar periosteal reaction)(5)Reticulum cell sarcoma (clinically healthy, between 30 and 50 years, no glycogen)(6)Neuroblastoma (< age 5)(7)Anaplastic metastatic carcinoma (>30 years of age)(8)Osteosarcoma(9)Hodgkin disease

Notes:

EXTRAMEDULLARY HEMATOPOIESIS
=compensatory response to deficient bone marrow blood cell production*Etiology:* prolonged erythrocyte deficiency due to(1)destruction of RBC:acquired hemolytic anemia, sickle cell anemia, thalassemia, hereditary spherocytosis, idiopathic severe anemia, erythroblastosis fetalis(2)inability of normal blood-forming organs to produce erythrocytes:iron deficiency anemia, pernicious anemia, myelofibrosis, myelosclerosis, polycythemia, carcinomatous / lymphomatous replacement of bone marrow (leukemia, Hodgkin disease)◊NO hematologic disease in 25% ● absence of pain, bone erosion, calcification ● chronic anemiaSites:in areas of fetal erythropoiesis@spleen, liver, lymph nodes@adrenal glands@mediastinum, heart, thymus@lung@renal pelvis@gastrointestinal lymphatics@dura mater (falx cerebri and over brain convexity)@cartilage, broad ligaments@thrombi, adipose tissue√ frequently bilateral paraspinal masses with round + lobulated margins between T8 and T12√ extramedullary hematopoiesis may compress cord√ splenomegaly / absent spleen√ lack of calcification / bone erosion

Notes:

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FAMILIAL IDIOPATHIC ACROOSTEOLYSIS
=HAJDU-CHENEY SYNDROME=rare bizarre entity of unknown etiologyLocation:may be unilateral ● fingernails remain intact ● sensory changes + plantar ulcers rare √ pseudoclubbing of fingers + toes with osteolysis of terminal + more proximal phalanges√ genu varum / valgum√ hypoplasia of proximal end of radius√ subluxation of radial head√ scaphocephaly, basilar impression√ wide sutures, persistent metopic suture, Wormian bones, poorly developed sinuses√ kyphoscoliosis√ severe osteoporosis + fractures at multiple sites (esp. of spine)√ protrusio acetabuli

Notes:

FANCONI ANEMIA
=autosomal recessive disease with severe hypoplastic anemia + skin pigmentation + skeletal and urogenital anomalies ● skin pigmentation (melanin deposits) in 74% (trunk, axilla, groin, neck) ● anemia onset between 17 months and 22 years of age ● bleeding tendency (pancytopenia) ● hypogonadism (40%) ● microphthalmia (20%) √ anomalies of radial component of upper extremity (strongly suggestive):√ absent / hypoplastic / supernumerary thumb√ hypoplastic / absent radius√ absent / hypoplastic navicular / greater multangular bone√ slight / moderate dwarfism√ minimal microcephaly√ renal anomalies (30%): renal aplasia, ectopia, horseshoe kidney*Prognosis:*fatal within 5 years after onset of anemia; patients family shows high incidence of leukemia

Notes:

FARBER DISEASE
=DISSEMINATED LIPOGRANULOMATOSIS*Histo:*foam cell granulomas; lipid storage of neuronal tissue (accumulation of ceramide + gangliosides) ● hoarse weak cry ● swelling of extremities; generalized joint swelling ● subcutaneous + periarticular granulomas ● intermittent fever, dyspnea ● lymphadenopathy√ capsular distension of multiple joints (hand, elbow, knee)√ juxta-articular bone erosions from soft-tissue granulomas√ subluxation / dislocation√ disuse / steroid deossification*Prognosis:*death from respiratory failure within 2 years

Notes:

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FIBROCHONDROGENESIS

=autosomal recessive lethal short-limb skeletal dysplasia*Incidence:*5 cases√ severe micromelia + broad dumbbell-shaped metaphyses√ flat + clefted pear-shaped vertebral bodies√ short + cupped ribs√ frontal bossing√ low-set abnormally formed ears*Prognosis:*stillbirth / death shortly after birth*DDx:*(1)Thanatophoric dysplasia(2)Metatropic dysplasia(3)Spondyloepiphyseal dysplasia

Notes:

FIBRODYSPLASIA OSSIFICANS PROGRESSIVA
=MYOSITIS OSSIFICANS PROGRESSIVA (misnomer since primarily connective tissues are affected)=rare slowly progressive sporadic / autosomal dominant disease with variable penetrance characterized by remissions + exacerbations of fibroblastic proliferation, subsequent calcification + ossification of subcutaneous fat, skeletal muscle, tendons, aponeuroses, ligaments*Histo:*edema with proliferating fibroblasts in a loose myxoid matrix; subsequent collagen deposition plus calcification + ossification of collagenized fibrous tissue in the center of nodules*Age:*presenting by age 2 years (50%) ● initially subcutaneous painful masses on neck, shoulders, upper extremities ● progressive involvement of remaining musculature of back, chest, abdomen, lower extremities ● lesions may ulcerate and bleed ● muscles of back + proximal extremities become rigid followed by thoracic kyphosis ● inanition secondary to jaw trismus (masseter, temporal muscle) ● "wry neck" = torticollis (due restriction of sternocleidomastoid muscle) ● respiratory failure (thoracic muscles affected) ● conductive hearing loss (fusion of middle ear ossicles) A.ECTOPIC OSSIFICATION√ rounded / linear calcification in neck / shoulders, paravertebral region, hips, proximal extremity, trunk, palmar + plantar fascia forming ossified bars + bony bridges√ ossification of voluntary muscles, complete by 20-25 years (sparing of sphincters + head)B.SKELETAL ANOMALIESmay appear before ectopic ossification ● clinodactyly√ microdactyly of big toes (90%) and thumbs (50%)= usually only one large phalanx present / synostosis of metacarpal + proximal phalanx (first sign) √ phalangeal shortening of hand + foot (middle phalanx of 5th digit)√ shortened 1st metatarsal + hallux valgus (75%)√ shortened metacarpals + metatarsals √ shallow acetabulum√ short widened femoral neck√ thickening of medial cortex of tibia√ progressive fusion of posterior arches of cervical spine√ narrowed AP diameter of cervical + lumbar vertebral bodies√ ± bony ankylosisCAVE:surgery is hazardous causing accelerated ossification at the surgical site

Notes:

FIBROMA OF SOFT TISSUE

*Histo:*hypocellular highly collagenic tumor*Age:*3rd and 4th decades; M > FLocation:tendon sheath of distal upper extremity√ slowly growing lesion 1-5 cm in sizeMR: √ small hypointense nodule on all pulse sequences

Notes:

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FIBROMATOSIS

=DESMOID TUMOR=benign aggressively growing lesionLocation:shoulder, pelvis, abdomen, thighSite:fascia in / around muscle√ mostly <10 cm in diameterMR: √ poorly defined (with invasion of fat / muscle) / lobulated well-defined lesion√ isointense with muscle on T1WI√ hyperintense (hypercellular) / hyperintense with areas of low intensity (intermixed with fibrous components) / hypointense (hypocellular) on T2WI*Cx:*compresses / engulfs adjacent structures

Notes:

FIBROSARCOMA

*Incidence:*4% of all primary bone neoplasm*Etiology:* A.PRIMARY FIBROSARCOMA (70%)B.SECONDARY FIBROSARCOMA (30%)1.following radiotherapy of giant cell tumor / lymphoma / breast cancer2.underlying benign lesion: Paget disease (common); giant cell tumor, bone infarct, osteomyelitis, desmoplastic fibroma, enchondroma, fibrous dysplasia (rare)3.dedifferentiation of low-grade chondrosarcoma*Histo:*spectrum of well to poorly differentiated fibrous tissue proliferation; will not produce osteoid / chondroid / osseous matrix*Age:*predominantly in 3rd-5th decade (range of 8-88 years); M:F = 1:1Metastases to:lung, lymph nodes ● localized painful massLocation:tubular bones in young, flat bones in older patients; femur (40%), tibia (16%) (about knee in 30-50%), jaw, pelvis (9%); rare in small bones of hand + feet or spinal columnSite:eccentric at diaphyseal-metaphyseal junction into metaphysis; intramedullary / periosteal A.CENTRAL FIBROSARCOMA=intramedullary√ well-defined lucent bone lesion√ thin expanded cortex√ aggressive osteolysis with geographic / ragged / permeative bone destruction + wide zone of transition√ occasionally large osteolytic lesion with cortical destruction, periosteal reaction + soft-tissue invasion√ sequestration of bone may be present(DDx: eosinophilic granuloma, bacterial granuloma) √ sparse periosteal proliferation (uncommon)√ intramedullary discontinuous spread√ no calcification*DDx:*malignant fibrous histiocytoma, myeloma, telangiectatic osteosarcoma, lymphoma, desmoplastic fibroma, osteolytic metastasisB.PERIOSTEAL FIBROSARCOMA=rare tumor arising from periosteal connective tissueLocation:long bones of lower extremity, jaw√ contour irregularity of cortical border√ periosteal reaction with perpendicular bone formation may be present√ rarely extension into medullary cavity *Cx:*pathologic fracture (uncommon)*Prognosis:*20% 10-year survival*DDx:*(1)Osteolytic osteosarcoma (2nd-3rd decade)(2)Chondrosarcoma (usually contains characteristic calcifications)(3)Aneurysmal bone cyst (eccentric blown-out appearance with rapid progression)(4)Malignant giant cell tumor (begins in metaphysis extending toward joint)

Notes:

FIBROUS CORTICAL DEFECT
*Incidence:*30% of children; M:F = 2:1*Age:*peak age of 7-8 years (range of 2-10 years); mostly before epiphyseal closure*Histo:*fibrous tissue from periosteum invading underlying cortex ● asymptomaticLocation:metaphyseal cortex of long bone; posterior medial aspect of distal femur, proximal tibia, proximal femur, proximal humerus, ribs, ilium, fibula√ round when small, average diameter of 1-2 cm√ oval, extending parallel to long axis of host bone√ cortical thinning + expansion may occur√ smooth, well-defined / scalloped margins√ larger lesions are multilocular√ involution over 2-4 years*Prognosis:* (a)potential to grow and encroach on the medullary cavity leading to nonossifying fibroma(b)bone islands in the adult may be residue of incompletely involuted cortical defect

Notes:

FIBROUS DYSPLASIA
=LICHTENSTEIN-JAFFE DISEASE=benign fibro-osseous developmental anomaly of the mesenchymal precursor of bone, manifested as a defect in osteoblastic differentiation and maturation*Cause:*probable gene mutation during embryogenesis*Age:*1st-2nd decade (highest incidence between 3 and 15 years), 75% before age 30; progresses until growth ceases; M:F = 1:1*Histo:*medullary cavity replaced by immature matrix of collagen with small irregularly shaped trabeculae of immature "woven" bone + inadequate mineralization; never replaced by mature lamellar bone *Types:* A.MONOSTOTIC FORM (70-80%) ● usually asymptomatic until 2nd-3rd decadeLocation:ribs (28%), proximal femur (23%), craniofacial bones (10-25%)B.POLYOSTOTIC FORM (20-30%)*Age:*mean age of 8 years ● 2/3 symptomatic by age 10 ● leg pain, limp, pathologic fracture (75%) ● abnormal vaginal bleeding (25%)Location:unilateral + asymmetric; femur (91%), tibia (81%), pelvis (78%), foot (73%), ribs, skull + facial bones (50%), upper extremities, lumbar spine (14%), clavicle (10%), cervical spine (7%)Site:metadiaphysis√ leg length discrepancy (70%)√ "shepherds crook" deformity (35%)√ facial asymmetry√ tibial bowing√ rib deformityC.CRANIOFACIAL FORM = LEONTIASIS OSSEA*Incidence:*in 10-25% of monostotic form / in 50% of polyostotic form / isolated ● cranial asymmetry ● facial deformity ● exophthalmos ● visual impairmentLocation:sphenoid, frontal, maxillary, ethmoid bones > occipital, temporal bones√ unilateral overgrowth of facial bones + calvarium (NO extracranial lesions)√ outward expansion of outer table maintaining convexity (DDx: Paget disease with destruction of inner + outer table)√ prominence of external occipital protuberance*Cx:*neurologic deficit secondary to narrowed cranial foramina (eg, blindness)D.CHERUBISM (special variant)=autosomal dominant disorder of variable penetrance*Age:*childhood; more severe in males√ symmetric involvement of mandible + maxilla*Prognosis:*regression after adolescence *May be associated with:* (a)endocrine disorders:-precocious puberty in girls-hyperthyroidism-hyperparathyroidism: renal stones, calcinosis-acromegaly-diabetes mellitus-Cushing syndrome: osteoporosis, acne-growth retardation(b)soft-tissue myxoma (rare): typically multiple intramuscular lesions VARIANT:**McCune-Albright syndrome** (10%)(1)polyostotic unilateral fibrous dysplasia(2)"coast of Maine" café-au-lait spots (35%)(3)endocrine dysfunction: menarche in infancy (20%), hyperthyroidism ● swelling + tenderness ● limp, pain (± pathologic fracture) ● increased alkaline phosphatase ● advanced skeletal + somatic maturation (early) ● coast of Maine café-au-lait spots = yellowish to brownish patches of cutaneous pigmentation with irregular / serrated border, predominantly on back of trunk (30-50%), buttocks, neck, shoulders; often ipsilateral to bone lesions (DDx: "coast of California" spots of neurofibromatosis) Common location: rib cage (30%), craniofacial bones [calvarium, mandible] (25%), femoral neck + tibia (25%), pelvis Site:metaphysis is primary site with extension into diaphysis (rarely entire length)√ normal bone architecture altered + remodeled√ lesions in medullary cavity: radiolucent / "ground-glass" appearance / increased density√ trabeculated appearance due to reinforced subperiosteal bone ridges in wall of lesion√ expansion of bones (ribs, skull, long bones)√ well-defined sclerotic margin of reactive bone = rind√ endosteal scalloping with thinned / lost cortex (ribs, long bones) and intervening normal cortex is HALLMARK√ lesion may undergo calcification + enchondral bone formation = fibrocartilaginous dysplasia√ increased activity on bone scan during early perfusion + on delayed images@Skull ● skull deformity with cranial nerve compromise ● proptosisLocation:frontal bone > sphenoid bone; hemicranial involvement (DDx: Paget disease is bilateral)√ sclerotic skull base, may narrow neural foramina (visual + hearing loss)√ widened diploic space with displacement of outer table, inner table spared (DDx: Paget disease, inner table involved)√ obliteration of sphenoid + frontal sinuses due to encroachment by fibrous dysplastic bone√ inferolateral displacement of orbit√ sclerosis of orbital plate + small orbit + hypoplasia of frontal sinuses (DDx: Paget disease, meningioma en plaque)√ occipital thickening√ cystic calvarial lesions, commonly crossing sutures√ mandibular cystic lesion (very common)= osteocementoma, ossifying fibroma @Pelvis + ribs√ cystic lesions (extremely common)√ protrusio acetabuli@Extremities ● short stature as adult / dwarfism√ premature fusion of ossification centers√ epiphysis rarely affected before closure of growth plate√ bowing deformities + discrepant limb length (tibia, femur) due to stress of normal weight bearing√ "shepherds crook" deformity of femoral neck= coxa vara√ pseudarthrosis in infancy = osteofibrous dysplasia (DDx: neurofibromatosis)√ premature onset of arthritis*Cx:*(1)Transformation into osteo- / chondro- / fibrosarcoma or malignant fibrous histiocytoma(0.5-1%, more often in polyostotic form) ● increasing pain√ enlarging soft-tissue mass√ previously mineralized lesion turns lytic(2)Pathologic fractures: transformation of woven into lamellar bone may be seen, subperiosteal healing without endosteal healing*DDx:* (1)HPT (chemical changes, generalized deossification, subperiosteal resorption)(2)Neurofibromatosis (rarely osseous lesions, cystic intraosseous neurofibroma rare, café-au-lait spots smooth, familial disease)(3)Paget disease (mosaic pattern histologically, radiographically identical to monostotic cranial lesion)(4)Osteofibrous dysplasia (almost exclusively in tibia of infants, monostotic, lesion begins in cortex)(5)Nonossifying fibroma(6)Simple bone cyst(7)Giant cell tumor (no sclerotic margin)(8)Enchondromatosis(9)Eosinophilic granuloma(10)Osteoblastoma(11)Hemangioma(12)Meningioma

Notes:

Benign fibrous histiocytoma *Incidence:*0.1% of all bone tumors*Histo:*interlacing bundles of fibrous tissue in storiform pattern (whorled / woven) interspersed with mono- / multinucleated cells resembling histiocytes, benign giant cells, and lipid-laden macrophages; resembles nonossifying fibroma / fibroxanthoma*Age:*23-60 years

- localized intermittently painful soft-tissue swelling*Location:*long bone, pelvis, vertebra (rare)*Site:*typically in epiphysis / epiphyseal equivalent√ well-defined radiolucent lesion with septa / soap-bubble appearance / no definable matrix√ may have reactive sclerotic rim√ narrow transition zone (= nonaggressive lesion)√ no periosteal reaction*Rx:*curettage*DDx:*nonossifying fibroma (childhood / adolescence, asymptomatic, eccentric metaphyseal location)

Notes:

Atypical Benign Fibrous Histiocytoma *Histo:*"atypical aggressive" features = mitotic figures present lytic defect with irregular edges*Prognosis:*may metastasize

Notes:

Malignant Fibrous Histiocytoma =MFH = MALIGNANT FIBROUS XANTHOMA=XANTHOSARCOMA = MALIGNANT HISTIOCYTOMA = FIBROSARCOMA VARIANT*Histo:*spindle-cell neoplasm of a mixture of fibroblasts + giant cells resembling histiocytes with nuclear atypia and pleomorphism in pinwheel arrangement; closely resembles high-grade fibrosarcoma (= fibroblastic cells arranged in uniform pattern separated by collagen fibers)(a)pleomorphic-storiform subtype (50-60%)(b)myxoid subtype (25%)(c)giant cell subtype (5-10%)(d)inflammatory subtype (5-10%)(e)angiomatoid subtype (<5%)*Age:*10-90 (average 50) years; peak prevalence in 5th decade; more frequent in Caucasians;M:F = 3:2Location:potential to arise in any organ (ubiquitous mesenchymal tissue); soft tissues >> bone ***Soft-tissue MFH*** *Incidence:*20-30% of all soft-tissue sarcomas; most common primary malignant soft-tissue tumor of late adult life◊Any deep-seated invasive intramuscular mass in a patient >50 years of age is most likely MFH!Location:extremities (75%), [lower extremity (50%), upper extremity (25%)], retroperitoneum (15%), head + neck (5%)Site:within large muscle groups ● large painless soft-tissue mass with progressive enlargement over several months√ mass usually 5-10 cm in size with increase over months / years√ poorly defined curvilinear / punctate peripheral calcifications / ossifications (in 5-20%)√ cortical erosion of adjacent bone (HIGHLY SUGGESTIVE FEATURE)CT: √ well-defined soft-tissue mass with central hypodense area = myxoid MFH (DDx: hemorrhage, necrosis, leiomyosarcoma with necrosis, myxoid lipo- / chondrosarcoma)√ enhancement of solid componentsMR: √ inhomogeneous poorly defined lesion iso- / hyperintense to muscle on T1WI + hyperintense on T2WI*Prognosis for soft-tissue MFH:* larger + more deeply located tumors have a worse prognosis; 2-year survival rate of 60%; 5-year survival rate of 50%; local recurrence rate of 44%; metastatic rate of 42% (lung, lymph nodes, liver, bone) *DDx:*(1)Liposarcoma (younger patient, presence of fat in >40%, calcifications rare)(2)Rhabdomyosarcoma(3)Synovial sarcoma (cortical erosion)
Osseous MFH *Prevalence:*5% of all primary malignant bone tumors ● painful, tender, rapidly enlarging mass ● pathologic fracture (20%)*Associated with:* prior radiation therapy, bone infarcts, Paget disease, fibrous dysplasia, osteonecrosis, fibroxanthoma (= nonossifying fibroma), enchondroma, chronic osteomyelitis◊20% of all osseous MFH arise in areas of abnormal bone!Location:femur (45%), tibia (20%), 50% about knee; humerus (10%); ilium (10%); spine; sternum; clavicle; rarely small bones of hand + feetSite:central metaphysis of long bones (90%); eccentric in diaphysis of long bones (10%)√ radiolucent defect with ill-defined margins (2.5-10 cm in diameter)√ extensive mineralization / small areas of focal metaplastic calcification√ permeation + cortical destruction√ expansion in smaller bones (ribs, sternum, fibula, clavicle)√ occasionally lamellated periosteal reaction (especially in presence of pathologic fracture)√ soft-tissue extension*Cx:*pathologic fracture (30-50%)*DDx:*(1) metastasis (2) fibrosarcoma (often with sequestrum) (3) reticulum cell sarcoma (4) osteosarcoma (5) giant cell tumor (6) plasmacytoma ***Pulmonary MFH*** (extremely rare) √ solitary pulmonary nodule without calcification√ diffuse infiltrateNUC: √ increased uptake of Tc-99m MDP (mechanism not understood)√ increased uptake of Ga-67 citrateUS: √ well-defined mass with hyperechoic + hypoechoic (necrotic) areasCT: √ mass of muscle density with hypodense areas (necrosis)√ invasion of abdominal musculature, but not IVC / renal veins (DDx to renal cell carcinoma)Angio: √ hypervascularity + early venous return

Notes:

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FOCAL FIBROCARTILAGINOUS DYSPLASIA OF TIBIA

Associated with: tibia vara*Age:*9-28 months*Histo:*dense hypocellular fibrous tissue resembling tendon with lacuna formation ● slight shortening of affected legLocation:insertion of pes anserinus (= tendinous insertion of gracilis, sartorius, semitendinous muscles) distal to proximal tibial physis; unilateral involvement√ unilateral tibia vara√ well-defined elliptic obliquely oriented lucent defect in medial tibial metadiaphyseal cortex√ sclerosis along lateral border of lesion√ absence of bone margin superomedially*Prognosis:*resolution in 1-4 years*DDx:*(1)Unilateral Blount disease (typically bilateral in infants, varus angulation of upper tibia, decreased height of medial tibial metaphysis, irregular physis)(2)Chondromyxoid fibroma, eosinophilic granuloma, osteoid osteoma, osteoma, fibroma, chondroma (not associated with tibia vara, soft-tissue mass)

Notes:

FRACTURE

=soft-tissue injury in which there is a break in the continuity of bone or cartilage*General description:* (1)OPEN / [CLOSED]open Fx=communication between fractured bone + skin(2)[COMPLETE] / INCOMPLETEcomplete Fx=all cortical surfaces disruptedincomplete Fx=partial separation of bonegreenstick Fx=break of one cortical margin only due to tensionbuckle / torus Fx=buckling of cortex due to compressionbowing Fx=plastic deformity of bonelead-pipe Fx=combination of greenstick + torus Fx(3)SIMPLE / COMMINUTEDsimple Fx=noncomminutedcomminuted Fx=>2 fragmentssegmental Fx=isolated segment of shaftbutterfly fragment=V-shaped fragment not completely circumscribed by cortex(4)DIRECTION OF FRACTURE LINE in relation to long axis of bone:transverse, oblique, oblique-transverse, spiral *Special terminology:* avulsion Fx=fragment pulled off by tendon / ligament from parent bonetranschondral Fx=cartilaginous surface involvedchondral Fx=cartilage alone involvedosteochondral Fx=cartilage + subjacent bone involved *Description of anatomic positional changes:* =change in position of distal fracture fragment in relation to proximal fracture fragment LENGTH = longitudinal change of fragments distraction=increase from original anatomic lengthshortening=decrease from original anatomic length-impacted=fragments driven into each other-overriding=also includes latitudinal changes-overlapping=bayonet apposition DISPLACEMENT =latitudinal change of anatomic axis:-undisplaced-anterior, posterior, medial / ulnar, lateral / radial ANGULATION / TILT =long axes of fragments intersect at the fracture apex:-medial / lateral, ventral / dorsal-varus=angular deviation of distal fragment toward midline on frontal projection-valgus=angular deviation of distal fragment away from midline on frontal projectioneg, "ventral angulation of fracture apex" eg, "in anatomic / near anatomic alignment" ROTATION ◊Difficult to detect radiographically!√ differences in diameters of apposing fragments√ mismatch of fracture line geometry-internal / external rotation NUC: *Typical time course:* 1.Acute phase (3-4 weeks)abnormal in 80% <24 hours, in 95% <72 hours◊elderly patients show delayed appearance of positive scan√ broad area of increased tracer uptake (wider than fracture line)2.Subacute phase (2-3 months) = time of most intense tracer accumulation√ more focal increased tracer uptake corresponding to fracture line3.Chronic phase (1-2 years)√ slow decline in tracer accumulation√ in 65% normal after 1 year; >95% normal after 3 years*Return to normal:* ◊non-weight-bearing bone returns to normal more quickly than weight-bearing bone ◊rib fractures return to normal most rapidly◊complicated fractures with orthopedic fixation devices take longest to return to normal1.Simple fractures: 90% normal by 2 years2.Open reduction / fixation: <50% normal by 3 years3.Delayed union: slower than normal for type of fracture4.Nonunion: persistent intense uptake in 80%5.Complicated union (true pseudarthrosis, soft-tissue interposition, impaired blood supply, presence of infection)√ intense uptake at fracture ends √ decreased uptake at fracture site6.Vertebral compression fractures: 60% normal by 1 year; 90% by 2 years; 97% by 3 years

Pathologic Fracture Stress Fracture Epiphyseal Plate Injury Apophyseal Injury Elbow Fracture Forearm Fracture Foot Fracture

Notes:

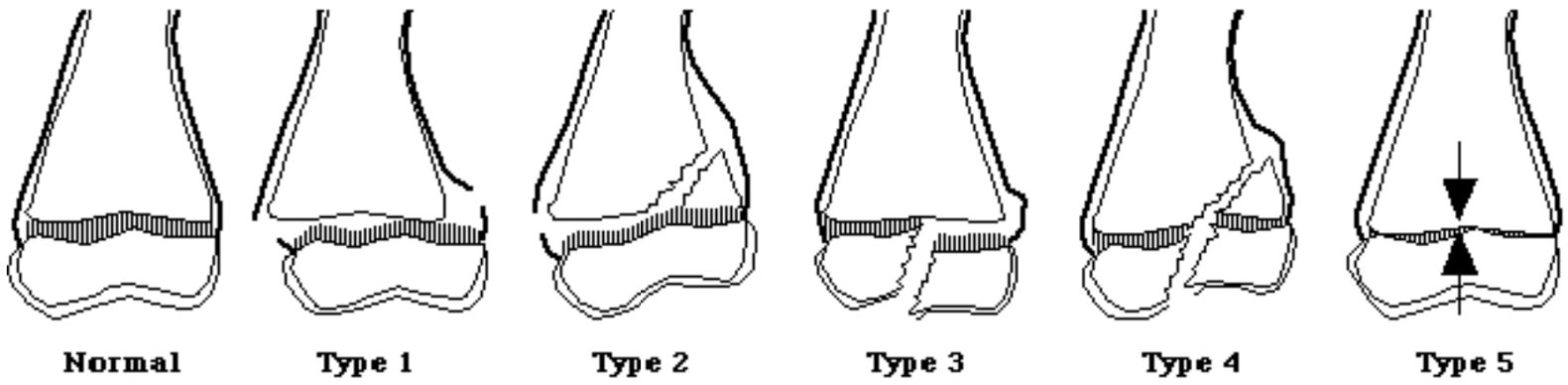
Pathologic Fracture =fracture at site of preexisting osseous abnormality*Cause:*tumor, osteoporosis, infection, metabolic disorder

Notes:

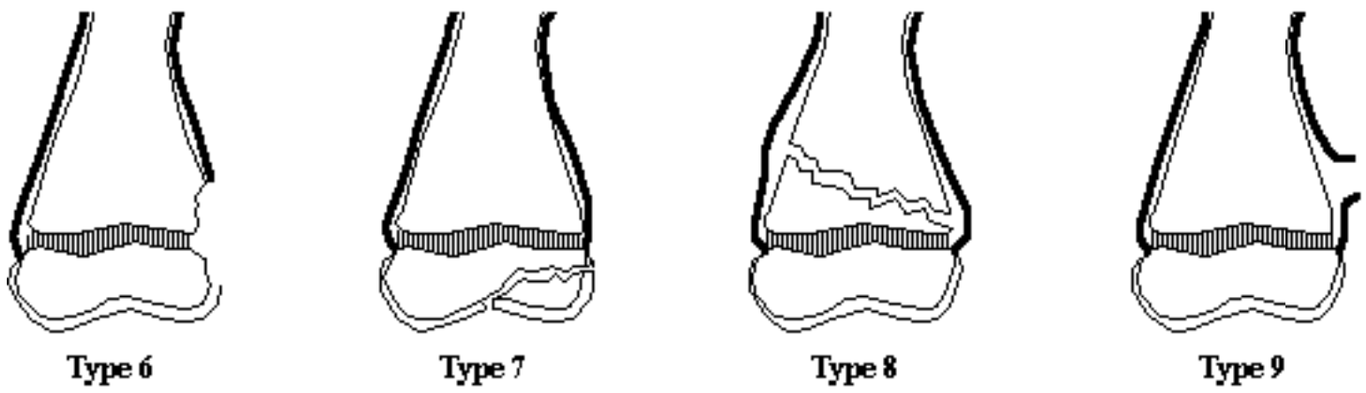
Stress Fracture =fractures produced as a result of repetitive prolonged muscular action on bone that has not accommodated itself to such action***Insufficiency Fracture*** =normal physiologic stress applied to bone with abnormal elastic resistance / deficient mineralization*Cause:* 1.Osteoporosis2.Rheumatoid arthritis3.Paget disease4.Fibrous dysplasia5.Osteogenesis imperfecta6.Osteopetrosis7.Osteomalacia / rickets8.Hyperparathyroidism9.Renal osteodystrophy10.Radiation therapy11.Prolonged corticosteroid treatment Location:lower extremity (calcaneus, tibia, fibula), thoracic vertebra, sacrum, ilium, pubic bone **Pelvic Insufficency Fracture** ● severe pain in lower back, buttock, groin ● walking ability impaired*Incidence:*1.8-5% of women >55 years*Predisposed:*postmenopausal womenLocation:sacral ala, parasymphyseal region of os pubis, pubic rami, supra-acetabular region, iliac blades, superomedial portion of ilium*Types:* (a)occult fracture:Site:sacrum > supra-acetabulum, ilium√ sclerotic band, cortical disruption, fracture line◊Often obscured by overlying bowel gas!(b)aggressive fracture:Site:parasymphysis, pubic rami√ exuberant callus formation, osteolysis + fragments (with prolonged or delayed healing / chronic nonunion)CAVE:fracture may be misdiagnosed as neoplasm; interpretation also histologically difficultNUC: √ butterfly / H-shaped ("Honda sign") / asymmetric incomplete H-shaped pattern of sacral uptake√ pelvic outlet view for parasymphyseal fxCT (most accurate modality): √ sclerotic band, linear fracture line, cortical disruption, fragmentation, displacement◊ Excludes bone destruction + soft-tissue masses!*Prognosis:*healing in 12-30 months ***Fatigue Fracture*** =normal bone subjected to repetitive stresses (none of which is singularly capable of producing a fracture) leading to mechanical failure over time*Risk factors:*new / different / rigorous repetitive activity; female sex; increased age; Caucasian race; low bone mineral density; low calcium intake; fluoride treatment for osteoporosis; condition resulting in altered gait ● activity-related pain abating with rest ● constant pain with continued activity1.**Clay shovelers fracture**: spinous process of lower cervical / upper thoracic spine2.**Clavicle**: postoperative (radical neck dissection)3.**Coracoid process of scapula**: trap shooting4.**Ribs**: carrying heavy pack, golf, coughing5.**Distal shaft of humerus**: throwing ball6.**Coronoid process of ulna**: pitching ball, throwing javelin, pitchfork work, propelling wheelchairs7.**Hook of hamate**: swinging golf club / tennis racquet / baseball bat8.**Spondylolysis** = pars interarticularis of lumbar vertebrae: ballet, lifting heavy objects, scrubbing floors9.**Femoral neck**: ballet, long-distance running10.**Femoral shaft**: ballet, marching, long-distance running, gymnastics11.**Obturator ring of pelvis**: stooping, bowling, gymnastics12.**Patella**: hurdling13.**Tibial shaft**: ballet, jogging14.**Fibula**: long-distance running, jumping, parachuting15.**Calcaneus**: jumping, parachuting, prolonged standing, recent immobilization16.**Navicular**: stomping on ground, marching, prolonged standing, ballet17.**Metatarsal** (commonly 2nd MT): marching, stomping on ground, prolonged standing, ballet, postoperative bunionectomy18.**Sesamoids of metatarsal**: prolonged standing X-ray (15% sensitive in early fractures, increasing to 50% on follow-up): -cancellous (trabecular) bone (notoriously difficult to detect)√ subtle blurring of trabecular margins√ faint sclerotic radiopaque area of peritrabecular callus (50% change in bone density needed)√ sclerotic band (due to trabecular compression + callus formation) usually perpendicular to cortex-compact (cortical) bone√ "gray cortex sign" = subtle ill definition of cortex√ intracortical radiolucent striations (early)√ solid thick lamellar periosteal new bone formation√ endosteal thickening (later)√ follow-up radiography after 2-3 weeks of conservative therapy NUC ("gold standard" = almost 100% sensitive): √ abnormal uptake within 6-72 hours of injury (prior to radiographic abnormality)√ "stress reaction" = focus of subtly increased uptake√ focal fusiform area of intense cortical uptake√ abnormal uptake persists for monthsMR (very sensitive modality; fat saturation technique most sensitive to detect increase in water content of medullary edema / hemorrhage): √ diminished marrow signal intensity on T1WI√ increased marrow signal intensity on T2WI√ low-intensity band contiguous with cortex on T2WI = fracture line of more advanced lesion CT (least sensitive modality): helpful in:longitudinal stress fracture of tibia; in confusing pediatric stress fracture (to detect endosteal bone formation) *DDx:* (1)Shin splints (activity not increased in angiographic / blood-pool phase)√ long linear uptake on posteromedial (soleus muscle) / anterolateral (tibialis anterior muscle) tibial cortex on delayed images (from stress to periosteum at muscle insertion site)(2)Osteoid osteoma (eccentric, nidus, solid periosteal reaction, night pain)(3)Chronic sclerosing osteomyelitis (dense, sclerotic, involving entire circumference, little change on serial radiographs)(4)Osteomalacia (bowed long bones, looser zones, gross fractures, demineralization)(5)Osteogenic sarcoma (metaphyseal, aggressive periosteal reaction)(6)Ewing tumor (lytic destructive appearance with soft-tissue component, little change on serial radiographs)

Notes:

Epiphyseal Plate Injury *Prevalence:*6-18-30% of bone injuries in children <16 years of age*Peak age:*12 yearsLocation:distal radius (28%), phalanges of hand (26%), distal tibia (10%), distal phalanges of foot (7%), distal humerus (7%), distal ulna (4%), proximal radius (4%), metacarpals (4%), distal fibula (3%)*Mechanism:*80% shearing force; 20% compressionResistance to trauma: ligament > bone > physis (hypertrophic zone most vulnerable) **Salter-Harris classification** (considering probability of growth disturbance) ⚱Prognosis is worse in lower extremities (ankle + knee) irrespective of Salter-Harris type!



Salter-Harris Classifcation of Epiphyseal Plate Injuries



Rang and Ogden's Additions to Salter-Harris

mnemonic:"SALTR"**S**lip of physis=type 1**A**bove physis=type 2**L**ower than physis=type 3**T**hrough physis=type 4**R**ammed physis=type 5 ***Salter Type 1***(6-8.5%)=slip of epiphysis due to shearing force separating epiphysis from physisLine of cleavage:confined to physisLocation:most commonly in phalanges, distal radius (includes: apophyseal avulsion, slipped capital femoral epiphysis)√ displacement of epiphyseal ossification center*Prognosis:*favorable irrespective of location ***Salter Type 2***(73-75%)=shearing force splits growth plateLine of fracture:through physis + extending through margin of metaphysis separating a triangular metaphyseal fragment (= "corner sign")Location:distal radius (33-50%), distal tibia + fibula, phalanges*Prognosis:*good, may result in minimal shortening ***Salter Type 3***(6.5-8%)=intra-articular fracture, often occurring after partial closure of physisLine of fracture:vertically / obliquely through epiphysis + extending horizontally to periphery of physisLocation:distal tibia, distal phalanx, rarely distal femur√ epiphysis split vertically*Prognosis:*fair (imprecise reduction leads to alteration in linearity of articular plane) ***Salter Type 4***(10-12%)Location:lateral condyle of humerus, distal tibia√ fracture involves metaphysis + physis + epiphysis*Prognosis:*guarded (may result in deformity + angulation) ***Salter Type 5***(<1%)=crush injury with injury to vascular supplyLocation:distal femur, proximal tibia, distal tibia*Often associated with:*fracture of adjacent shaft√ no immediate radiographic finding√ shortening of bone + cone epiphysis / angular deformity on follow-up*Prognosis:*poor (impairment of growth in 100%) ***Triplane Fracture*** *(6%)* Location:distal tibia, lateral condyle of distal humerus√ vertical fracture of epiphysis + horizontal cleavage plane within physis + oblique fracture of adjacent metaphysis MR: √ focal dark linear area (= line of cleavage) within bright physis on gradient echo images (GRE) *Cx:*(1)progressive angular deformity from segmental arrest of germinal zone growth with formation of a bone bridge across physis = "bone bar"(2)limb length discrepancy from total cessation of growth(3)articular incongruity from disruption of articular surface(4)Bone infarction in metaphysis / epiphysis

Notes:

Apophyseal Injury *Mechanism:*avulsive force Physis under secondary ossification center is weakest part!*At risk:*hurdlers, sprinters, cheerleaders (repetitive to and fro adduction / abduction + flexion / extension) ● pain, point tenderness, swelling

LocationMuscle origin / insertion

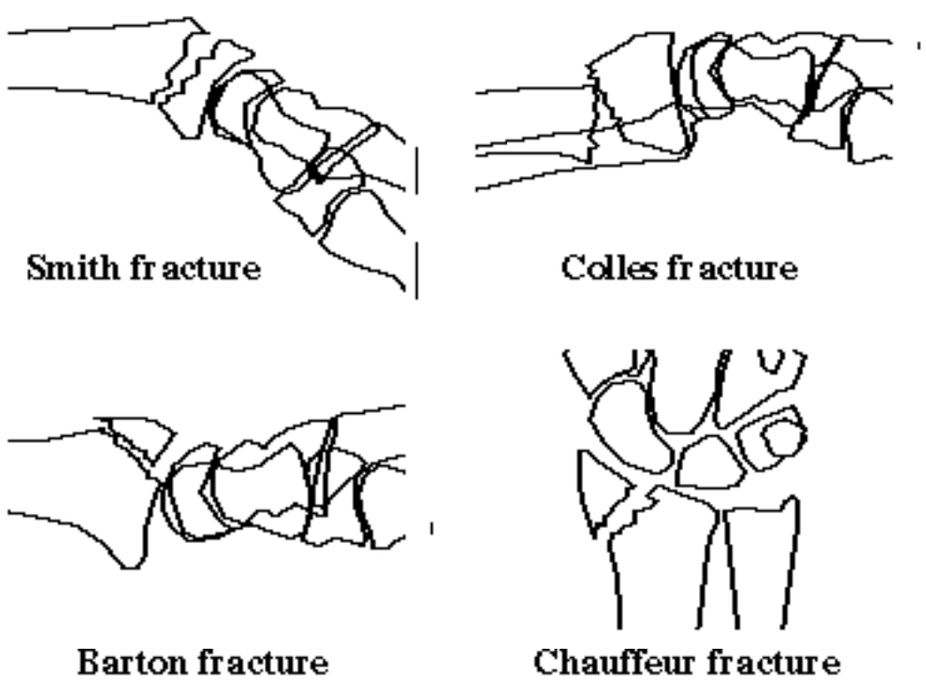
anterior superior iliac spinesartorius muscle +tensor fasciae femoris m.anterior inferior iliac spinerectus femoris musclelesser trochanterpsoas muscleischial tuberosityhamstring musclegreater trochantergluteal muscleiliac crestabdominal musclesymphysis pubisadductor muscle
√ irregularity at site of avulsion√ displaced pieces of bone of variable size√ abnormal foci of ossification

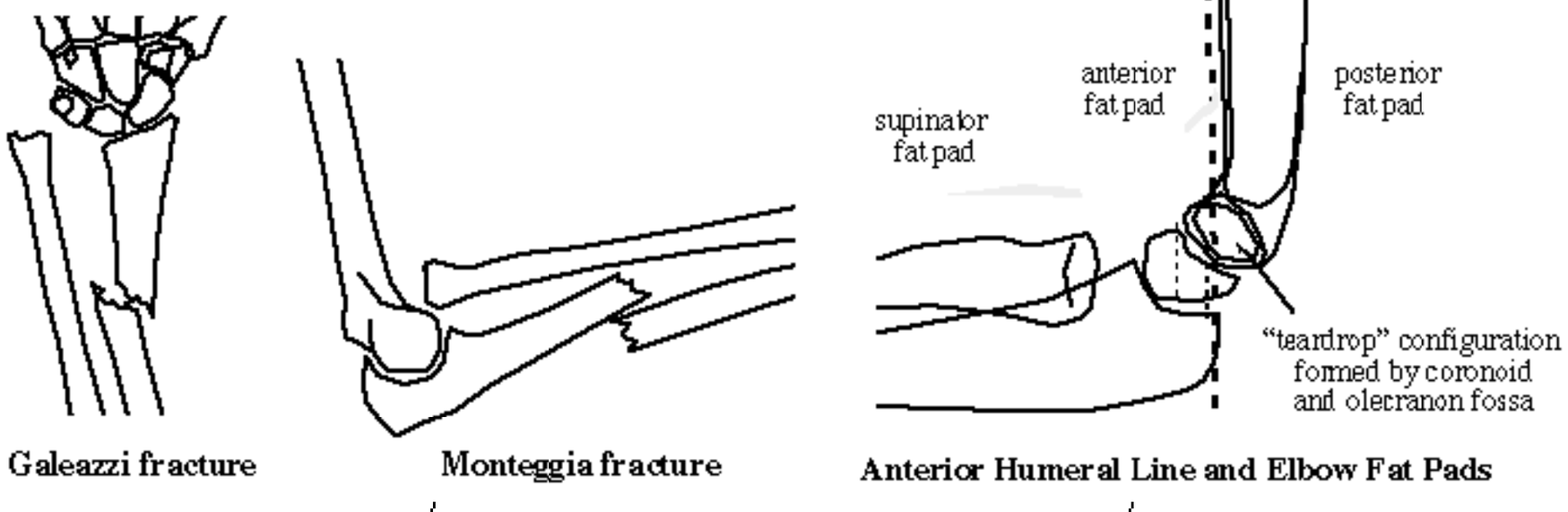
Notes:

Elbow Fracture common among children 2-14 years of age @Soft-tissue√ displacement of anterior + posterior fat pads(= elbow joint effusion with supracondylar / lateral condylar / proximal ulnar fractures) √ supinator fat pad (= fracture of proximal radius)√ focal edema medially (= medial epicondyle fx) / laterally (= lateral condyle fx)@Humerus (80%)Supracondylar fracture (55%) *Mechanism:*hyperextension with vertical stress√ transverse fracture line√ distal fragment posteriorly displaced / tilted√ anterior humeral line intersecting anterior to posterior third of capitellum (on lateral x ray) **Lateral condylar fracture (20%)** *Mechanism:*hyperextension with varus stress√ fracture line between lateral condyle + trochlea / through capitellum**Medial epicondylar fracture** (5%) *Mechanism:*hyperextension with valgus stress√ avulsion of medial epicondyle (by flexor muscles of forearm)√ may become trapped in joint space (after reduction of concomitant elbow dislocation)@Radius (10%)*Mechanism:*hyperextension with valgus stress√ Salter-Harris type II / IV fracture√ transverse metaphyseal / radial neck fracture*Mechanism:*hyperextension with varus stress√ dislocation as part of Monteggia fracture (from rupture of annular ligament)@Ulna (10%)√ longitudinal linear fracture through proximal shaft*Mechanism:*hyperextension with vertical stress√ transverse fracture through olecranon*Mechanism:*hyperextension with valgus / varus stress; blow to posterior elbow in flexed position√ coronoid process avulsion*Mechanism:*hyperextension-rotation associated with forceful contraction of brachial m.

Notes:

Forearm Fracture

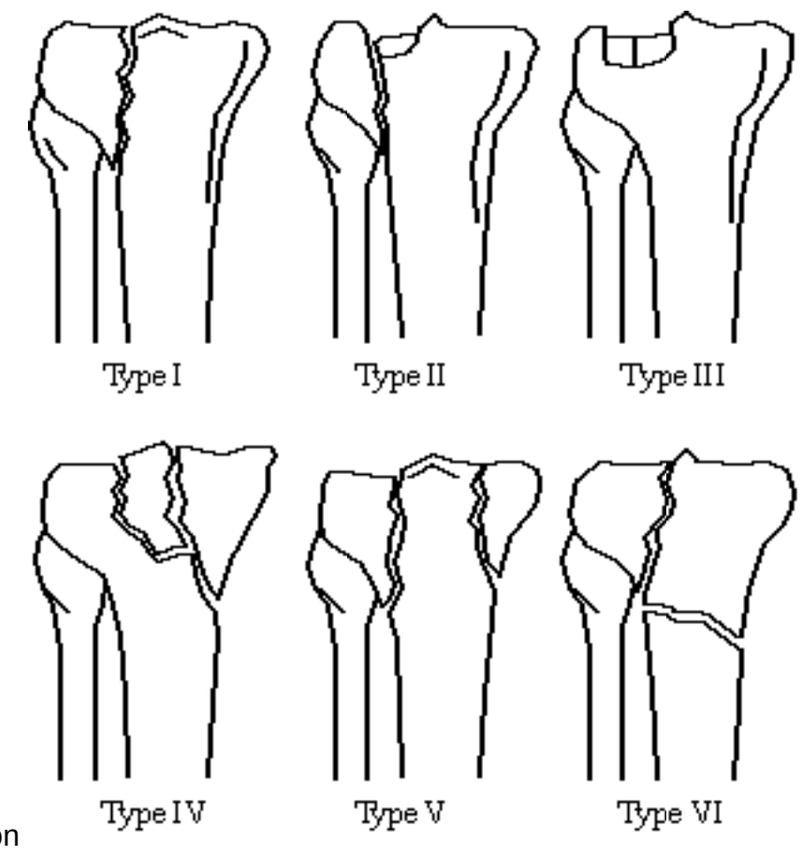




Barton Fracture

*Mechanism:*fall on outstretched hand√ intra-articular oblique fracture of dorsal lip of distal radius√ carpus dislocates with distal fragment up and back on radius *Chauffeur Fracture* =name derived from direct trauma to radial side of wrist sustained from recoil of crank used in era of hand-cranked automobiles=HUTCHINSON FRACTURE*Mechanism:*acute dorsiflexion + abduction of hand√ triangular fracture of radial styloid process *Colles Fracture* Most common fracture of forearm *Mechanism:*fall on outstretched hand√ radial fracture in distal 2 cm ± ulnar styloid fracture√ dorsal displacement of distal fragment√ "silver-fork" deformity*Cx:*posttraumatic arthritis*Rx:*anatomic reduction important*Significant postreduction deformity:* 1.Residual positive ulnar variance >5 mm indicates unsatisfactory outcome in 40%2.Dorsal angulation of palmar tilt >15° decreases grip strength + endurance in >50% *Galeazzi Fracture Mechanism:*fall on outstretched hand with elbow flexed√ radial fracture in distal third + subluxation / dislocation of distal radioulnar joint√ dorsal angulation√ ulnar plus variance (= radial shortening) of >10 mm implies complete disruption of interosseous membrane = complete instability of radioulnar joint*Cx:*(1)high incidence of nonunion, delayed union, malunion (unstable fracture)(2)limitation of pronation / supination *Monteggia Fracture Mechanism:*direct blow to the forearm√ anteriorly angulated proximal ulnar fracture + anterior dislocation of radiohumeral joint√ may have associated wrist injury*Cx:*nonunion, limitation of motion at elbow, nerve abnormalities Reverse Monteggia Fracture =dorsally angulated proximal ulnar fracture + posterior dislocation of radial head *Smith Fracture* =REVERSE COLLES FRACTURE*Mechanism:*hyperflexion with fall on back of hand√ distal radial fracture√ ventral displacement of fragment√ radial deviation of hand√ "garden spade" deformity*Cx:*altered function of carpus Hand Fracture *Bennett Fracture Mechanism:*forced abduction of thumb√ intra-articular fracture / dislocation of base of 1st metacarpal√ small fragment of 1st metacarpal continues to articulate with trapezium√ lateral retraction of 1st metacarpal shaft by abductor pollicis longus*Rx:*anatomic reduction important, difficult to keep in anatomic alignment*Cx:*pseudarthrosis *Boxers Fracture Mechanism:*direct blow with clenched fist√ transverse fracture of distal metacarpal (usually 5th) *Gamekeepers Thumb* =SKIERS THUMB; originally described as chronic lesion in hunters strangling rabbits*Incidence:*6% of all skiing injuries; 50% of skiing injuries to the hand*Mechanism:*violent abduction of thumb with injury to ulnar collateral ligament (UCL) in 1st MCP (faulty handling of ski pole)√ disruption of ulnar collateral ligament of 1st MCP joint, usually occurring distally near insertion on proximal phalanx√ radial stress examination necessary to document ligamentous disruption√ displacement of UCL superficial to aponeurosis of adductor pollicis (= Stener lesion) [torn end of UCL may be marked by avulsed bone fragment] *Navicular Fracture* =SCAPHOID FRACTURE◊Most frequently fractured of all carpal bones*Mechanism:*fall on dorsiflexed outstretched hand ● pain + tenderness at anatomic snuff boxRadiographic misses: 25-33-65% N.B.: If initial radiograph negative, reexamine in 2 + 6 weeks after treatment with short-arm spica cast! MR:high sensitivityBone scan:up to 100% sensitive, 93% PPV after 2-3 days*Prognosis:*dependent on√ displaced fracture = >1 mm offset / angulation / rotation of fragments (less favorable)√ location (blood supply derived from distal part):-distal 1/3 (10%) = usually fragments reunite-middle-third (70%) = failure to reunite in 30%-proximal 1/3 (20%) = failure to reunite in 90%√ orientation of fracture-transverse / horizontal oblique = relatively stable-vertical oblique (less common) = unstable◊Good prognosis with distal fracture + no displacement + no ligamentous injury!◊Less favorable prognosis with displaced / comminuted fracture + proximal pole fracture!*Cx:*avascular necrosis of proximal fragment *Rolando Fracture* √ comminuted intra-articular fracture through base of thumb*Prognosis:* worse than Bennetts fracture (difficult to reduce) Pelvic Fracture *Malgaigne Fracture Mechanism:*direct trauma ● shortening of involved extremity√ vertical fractures through one side of pelvic ring(1) superior to acetabulum (2) inferior to acetabulum (3) ± sacroiliac dislocation / fracture *Bucket Handle Fracture* √ double vertical fracture through superior and inferior pubic rami + sacroiliac joint dislocation on contralateral side

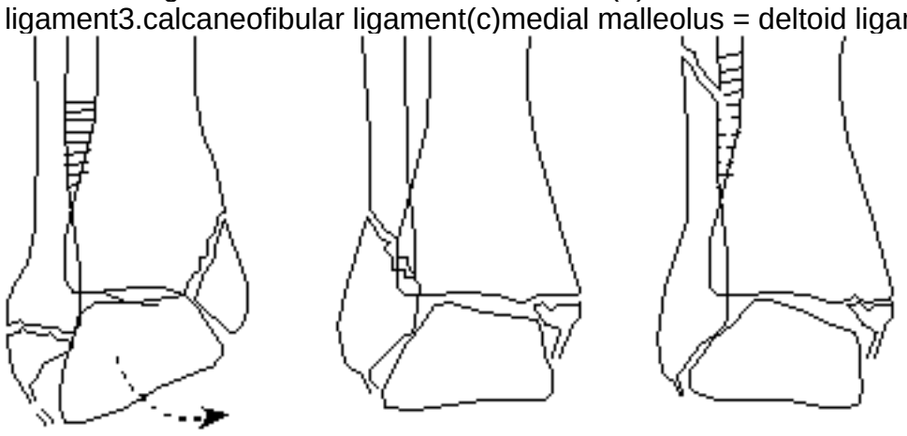
Knee Fracture *Segond Fracture Mechanism:*external rotation + varus stress causing excessive tension on the lateral capsular ligament*Associated with:*lesion of anterior cruciate ligament (75-100%), meniscal tear (67%) ● anterolateral instability of the knee√ small cortical avulsion fracture of proximal lateral tibial rim immediately distal to lateral plateau *Tibial Plateau Fracture* (Schatzker classification) *Mechanism:*valgus force ("bumper / fender fracture" from lateral force of automobile against a



pedestrians fixed knee) / compression force often in extension
Type I=wedge-shaped pure cleavage fracture6%Type II=combined cleavage + median25%compression fractureType III=pure compression fracture36%Type IV=medial plateau fracture with a split /10%depressed comminutionType V =bicondylar fracture, often with3%inverted Y appearanceType VI=transverse / oblique fracture with20%separation of metaphysis fromdiaphysis♁Lateral plateau fractures (type I-III) are most common!♁Fractures of medial plateau are associated with greater violence and higher percentage of associated injuries!

Notes:

Foot Fracture ***Ankle*** ***Fracture*** *Incidence:*ankle injuries account for 10% of all emergency room visits; 85% of all ankle sprains involve lateral ligamentsLigamentous connections at ankle: (a)binding tibia + fibula1.anterior inferior tibiofibular ligament(= tibiofibular syndesmosis) 2.posterior inferior tibiofibular ligament3.transverse tibiofibular ligament4.interosseous membrane(b)lateral malleolus85% of all ankle sprains involve these ligaments: 1.anterior talofibular ligament2.posterior talofibular ligament3.calcaneofibular ligament(c)medial malleolus = deltoid ligament with1.navicular portion2.sustentaculum portion3.talar portion



Supination-AdductionSupination-AbductionPronation-External Rotation

A.SUPINATION-ADDUCTION=INVERSION-ADDUCTION INJURY*Mechanism:* (1)avulsive forces affect lateral ankle structures(2)impactive forces secondary to talar shift stress medial structures√ sprain / rupture of lateral collateral ligament◊anterior tibiofibular ligament ruptures alone in 66%◊injury of all 3 lateral ligaments in 20%*Prognosis:*chronic lateral ankle instability in 10-20%√ transverse avulsion of malleolus sparing tibiofibular ligaments√ oblique fracture of medial malleolus ± posterior lip fracture B.SUPINATION-ABDUCTION= EVERSION / EXTERNAL ROTATION *Mechanism:* (1)avulsive forces on medial structures(2)impacting forces on lateral structures (talar impact)√ lateral subluxation of talus√ oblique / spiral fracture of lateral malleolus√ partial disruption of tibiofibular ligament√ sprain / rupture / avulsion of deltoid ligament√ transverse fracture of medial malleolus(a)**Pott** fracture√ fracture of fibula above an intact tibiofibular ligament(b)**Dupuytren** fracture√ fracture of fibula above a disrupted tibiofibular ligament C. PRONATION-EXTERNAL ROTATION = EVERSION + EXTERNAL ROTATION√ tear of tibiofibular ligament / avulsion of anterior tubercle (Tillaux-Chaput) / avulsion of posterior tubercle (Volkmann)√ tear of interosseous membrane = lateral instability√ fibular fracture higher than ankle joint (Maisonneuve fracture if around knee) ***Chopart*** ***Fracture*** √ fracture / dislocation through midtarsal joint (calcaneocuboid + talonavicular)√ commonly associated with fractures of the bones abutting the joint ***Jones*** ***Fracture*** *Mechanism:*plantar flexion + inversion (stepping off a curb)√ transverse avulsion fracture of base of 5th metatarsal (insertion of peroneus brevis tendon) ***Lisfranc*** ***Fracture*** *Mechanism:*metatarsal heads fixed and hindfoot forced plantarward and into rotation√ fracture / dislocation of tarsometatarsal joints ***Calcaneal*** ***Fracture*** *Incidence:*most commonly fractured tarsal bone; 60% of all tarsal fractures; 2% of all fractures in the body; commonly bilateral*Mechanism:*fall from heights*May be associated with:* lumbar vertebral fracture*Age:*95% in adults, 5% in children-adulthood:intra-articular (75%), extra-articular (25%)-childhood:extra-articular (63-92%)*Classification:* (a)Extra-articular fracture of calcaneal tuberosity:beak type, vertical, horizontal, medial avulsion(b)Intra-articular fracture-subtalar joint involvement: undisplaced, displaced, comminuted-calcaneocuboid joint involvement√ apex of lateral talar process does not point to "crucial angle" of Gissane√ Boehler angle decreased below 28°-40°

Notes:

FROSTBITE
Cause:(1)cellular injury + necrosis from freezing process(2)cessation of circulation secondary to cellular aggregates + thrombi forming as a result of exposure to low temperatures below -13° Celsius (usually cold air) ● firm white numb areas in cutis (separation of epidermal-dermal interface)Location:feet, hands (thumb commonly spared due to protection by clenched fist) Early changes: √ soft-tissue swelling + loss of tissue at tips of digits CHILD √ fragmentation / premature fusion / destruction of distal phalangeal epiphyses√ secondary infection, articular cartilage injury, joint space narrowing, sclerosis, osteophytosis of DIP√ shortening + deviation / deformity of fingersADULT √ osteoporosis (4-10 weeks after injury)√ periostitis√ acromutilation (secondary to osteomyelitis + surgical removal) + tuftal resorption (result of soft-tissue loss)√ small round punched-out areas near edge of joint√ interphalangeal joint abnormalities (simulating osteoarthritis)√ calcification / ossification of pinna Angio: √ vasospasm, stenosis, occlusion√ proliferation of arterial + venous collaterals (in recovery phase)Bone scintigraphy: √ persistent absence of uptake (= lack of vascular perfusion) indicates nonviable tissue*Rx:*selective angiography with intraarterial reserpine

Notes:

GANGLION
Ganglion=mucin-containing cyst arising from tendon sheath / joint capsule / bursa / subchondral bone lined by flat spindle-shaped cellsSynovial cyst=cyst continuous with joint capsule lined by synovial cells (term is used by some synonymously with ganglion)

Soft-tissue Ganglion Intraosseous Ganglion Periosteal Ganglion

Notes:

Soft-tissue Ganglion =cystic tumorlike lesion usually attached to a tendon sheath*Origin:*synovial herniation / coalescence of smaller cysts formed by myxomatous degeneration of periarticular connective tissue ● uni- / multilocular swellingLocation:hand, wrist, footSite:arise from tendon, muscle, semilunar cartilage√ soft-tissue mass with surface bone resorption√ periosteal new-bone formation√ arthrography may demonstrate communication with joint / tendon sheath√ internal septations*Rx:*steroid injection may improve symptomatology

Notes:

Intraosseous Ganglion =benign subchondral radiolucent lesion WITHOUT degenerative arthritis ● mild localized pain (4% of patients with unexplained wrist pain)*Age:*middle age*Origin:*(1)mucoid degeneration of intraosseous connective tissue perhaps due to trauma / ischemia(2)penetration of juxtaosseous soft-tissue ganglion into underlying bone (occasionally)*Path:*uni- / multilocular cyst surrounded by fibrous lining, containing gelatinous materialLocation:epiphysis of long bone (medial malleolus, femoral head, proximal tibia, carpal bones) / subarticular flat bone (acetabulum)√ well-demarcated solitary 0.6-6 cm lytic lesion√ sclerotic margin√ NO communication with joint√ increased radiotracer uptake on bone scintigraphy (in 10%)*DDx:*posttraumatic / degenerative cyst

Notes:

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Periosteal Ganglion = cystic structure with viscid / mucinous contents*Incidence:*11 cases in literature*Age:*39-50 years; M > F ● swelling, mild tendernessLocation:long tubular bones of lower extremity√ cortical erosion / scalloping / reactive bone formation√ NO intraosseous component (endosteal surface intact)CT: √ well-defined soft-tissue mass adjacent to bone cortex with fluid contentsMR: √ homogeneous isointense signal to muscle on T1WI√ homogeneous hyperintense signal to fat on T2WI√ NO internal septations (DDx to soft-tissue ganglion)*DDx:*periosteal chondroma without matrix calcification, cortical desmoid, subperiosteal aneurysmal bone cyst, acute subperiosteal hematoma (history of trauma / blood dyscrasia), subperiosteal abscess (involvement of adjacent bone marrow)*Rx:*surgical excision (local recurrence possible)

Notes:

GARDNER SYNDROME
=autosomal dominant syndrome characterized by(1) osteomas (2) soft-tissue tumors (3) colonic polypsLocation of osteomas:paranasal sinuses; outer table of skull (frequent); mandible (at angle)√ endosteal cortical thickening / osteomas in any bone√ may have solid periosteal cortical thickening√ osteomas / exostoses may protrude from periosteal surface√ wavy cortical thickening of superior aspect of ribs√ polyps:colon, stomach, duodenum, ampulla of Vater, small intestine*Cx:*high incidence of carcinoma of duodenum / ampulla of Vater

Notes:

GAUCHER DISEASE
=rare autosomal recessive disorder / dominant (in a few), common among Ashkenazi Jews; M < F*Etiology:*deficiency of lysosomal hydrolase acid ß-glycosidase (= glucocerebrosidase) leads to accumulation of glucosyl ceramide within cells of RES (liver, spleen, bone marrow, lung, lymph nodes)*Histo:*bone-marrow aspirate shows Gaucher cells (kerasin-laden histiocytes)*Types:* (1)Rapidly fatal infantile form = type 2: 1-12 months ● early onset of significant hepatosplenomegaly ● severe progressive neurologic symptoms: seizures, mental retardation, spasticity*Prognosis:*fatal during first 2 years of life(2)Juvenile form = type 3: 2-6 years ● mild neurologic involvement*Prognosis:*survival into adolescence(3)Adult form = type 1 (most common form in USA)*Prognosis:*longest time of survival; pulmonary involvement / hepatic failure may lead to early death ● hepatosplenomegaly, impairment of liver function, ascites ● elevated serum acid phosphatase ● pancytopenia, anemia, leukopenia, thrombocytopenia (hypersplenism) ● hemochromatosis (yellowish brown pigmentation of conjunctiva + skin) ● dull bone pain; bone involvement in 75%Location:axial skeleton, distal femur, pelvis, predominantly proximal + other long bones√ generalized osteopenia (decrease in trabecular bone density)√ striking cortical thinning + bone widening√ endosteal scalloping (due to marrow packing)√ Erlenmeyer flask deformity of distal femur + proximal tibia√ numerous sharply circumscribed lytic lesions resembling metastases / multiple myeloma (marrow replacement)√ periosteal reaction = cloaking√ weakening of subchondral bone + degenerative arthritis√ bone infarction in long-bone metaphyses (common)√ H-shaped / "step-off" / biconcave "fish-mouth" vertebra@Spleen√ multiple nodular lesions of low attenuation without enhancement on CT / hypoechoic or hyperechoic on US (= clusters of RES cells laden with glucosyl ceramide)@Lung√ diffuse reticulonodular infiltrates at lung bases (= infiltration with Gaucher cells)*Cx:*◊>90% have orthopedic complications at some time(1)pathologic fractures + compression fractures of vertebrae(2)osteonecrosis of femoral head, humeral head, wrist, ankle (common)(3)osteomyelitis (increased incidence)(4)myelosclerosis in long-standing disease(5)repeated pulmonary infections*Prognosis:*highly variable clinical course; strong relationship between splenic volume and disease severity

Notes:

GIANT CELL REPARATIVE GRANULOMA
=GIANT CELL REACTION*Histo:*numerous giant cells in exuberant fibrous matrix, osteoid formation, areas of hemorrhage*Peak age:*2nd + 3rd decade (range from childhood to 76 years); M:F = 1:1Location:mandible, maxilla, small bones of hand + feet ● pain + mass in affected bone√ expansile lytic defect with thinning of overlying cortex√ periosteal reaction may be present√ soft-tissue swelling / extension beyond cortex√ no matrix calcification*Cx:*pathologic fracture*Rx:*curettage (50% recurrence rate) / local excision*DDx:*(1)Enchondroma (same location, matrix calcification)(2)Aneurysmal bone cyst (rare in small bones of hand + feet, typically prior to epiphyseal closure)(3)Giant cell tumor (more aggressive appearance)(4)Infection (clinical)(5)Brown tumor of HPT (periosteal bone resorption, abnormal Ca + P levels)

Notes:

GIANT CELL TUMOR

=OSTEOCLASTOMA = probably arise from zone of intense osteoclastic activity in skeletally immature patients*Incidence:*4.2% of all primary bone tumors; 21% of benign skeletal tumors*Histo:*multinucleated osteoclastic giant cells intermixed throughout a spindle cell stroma (giant cells characteristic of all reactive bone disease, seen in pigmented villonodular synovitis, benign chondroblastoma, nonosteogenic fibroma, chondromyxoid fibroma, fibrous dysplasia)*Age:*in 98.3% after (in 1.7% before) epiphyseal plate fusion; 14% < age 20; 70-80% between 20 and 40 years; M:F = 1:1*May be associated with:*Paget disease (in 50-60% located in skull + facial bones) ● tenderness + pain at affected site ● weakness + sensory deficits (if in spine)Location: (a)85% in long bones-lower extremity (50-60% about knee):distal end of femur > proximal end of tibia-upper extremity (away from elbow):distal end of radius > proximal end of humerus(b)15% in flat bones: pelvis, sacrum near SIJ (common, 2nd only to chordoma) > thoracic > cervical > lumbar spine (5-7%), rib (anterior / posterior end), skullSite:eccentric in metaphysis of long bones, adjacent to / in ossified epiphyseal line, subarticular if epiphyseal plate is fused (MOST TYPICAL)√ expansile solitary lytic bone lesion ("soap bubble"), large at diagnosis√ conspicuous peripheral trabeculae without tumor matrix√ no sclerosis / periosteal reaction (aggressive rapid growth) in absence of fracture√ may break through bone cortex with cortical thinning, soft-tissue invasion (25%), pathologic fracture (5%)√ destruction of vertebral body with secondary invasion of posterior elements (DDx: ABC, osteoblastoma)√ frequently vertebral collapse√ involves adjacent vertebral disks + vertebrae, crosses sacroiliac joint√ may cross joint space in long bones (exceedingly rare)NUC: √ diffusely increased uptake ± "doughnut" sign of central photopeniaAngio: √ hypervascular lesionCT: √ tumor of soft-tissue attenuation with foci of low attenuation (hemorrhage / necrosis)√ well-defined margins ± thin rim of sclerosisMR: √ heterogeneous signal intensity with low to intermediate intensity on T1WI + T2WI (63-96%) due to collagen + hemosiderin content√ focal cystic areas√ low-signal-intensity pseudocapsule*Cx:*15% malignant within first 5 years (M:F = 3:1); metastases to lung*Prognosis:*locally aggressive; 40-60% recurrence rate*Rx:*complete resection; excision + radiation therapy*DDx:*(1)Aneurysmal bone cyst (in posterior elements of spine with invasion of vertebral body)(2)Brown tumor of HPT (lab values)(3)Cartilage tumor: chondroblastoma, enchondroma (not epiphyseal), chondromyxoid fibroma, chondrosarcoma(4)Bone abscess(5)Hemangioma(6)Fibrous dysplasia

Notes:

GLOMUS TUMOR
= hamartoma composed of cells derived from neuromyo-arterial apparatus (regulating blood flow in skin) **Glomus body** = encapsulated oval organ of 300 µm length; located in reticular dermis (= deepest layer of skin); concentrated in tips of digits (93-501/cm^2); composed of an afferent arteriole, an anastomotic vessel (= Sucquet-Hoyer canal lined by endothelium + surrounded by smooth muscle fibers), a primary collecting vein, the intraglomerular reticulum + capsule *Histo:*(a) vascular (b) myxoid (c) solid form*Prevalence:*1-5% of soft-tissue tumors of hand*Age:*mostly in 4-5th decade ● joint tenderness + pain (on average of 4-7 years duration prior to diagnosis) ● Love test = eliciting pain by applying precise pressure with a pencil tip ● Hildreth sign = disappearance of pain after application of a tourniquet proximally on arm (PATHOGNOMONIC) @SUBUNGUAL GLOMUS TUMOR√ increased distance between dorsum of phalanx + underside of nail (25%)√ extrinsic bone erosion (14-25-65%), often with sclerotic border√ small hypoechoic tumor by US (>3 mm detectable)√ homogeneously high-signal-intensity lesion on T2WI (detectable if >2 mm in diameter)@GLOMUS TUMOR OF BONE occasionally within bone√ resembles enchondroma*DDx:*(1)Mucoid cyst (painless, in proximal nail fold, communicating with DIP joint, associated with osteoarthritis)(2)Angioma (more superficially located)

Notes:

GOUT
=deposition of positively birefringent monosodium urate monohydrate crystals in poorly vascularized tissues (synovial membranes, articular cartilage, ligaments, bursae) leading to destruction of cartilage*Age:*>40 years; males (in women gout may occur after menopause)*Cause:* A.Idiopathic Gout*Incidence:*0.3%; M:F = 20:1(1)Overproduction of uric acid (phosphoribosyl transferase deficiency)(2)Abnormality of renal urate excretionB.Secondary Goutrarely cause for radiographically apparent disease (1)Myeloproliferative disorders + sequelae of their treatment: polycythemia vera, leukemia, lymphoma, multiple myeloma(2)Blood dyscrasias(3)Endocrinologic: myxedema, hyperparathyroidism(4)Chronic renal failure(5)Enzyme defects: glycogen storage disease(6)Vascular: myocardial infarction, hypertension(7)Lead poisoning *Stages:* (1)asymptomatic hyperuricemia(2)acute monarticular gout(3)polyarticular gout(4)chronic tophaceous gout = multiple large urate deposits Location:(a)joints: hands + feet (1st MTP joint most commonly affected = podagra), elbow, wrist (carpometacarpal compartment especially common), knee, shoulder, hip, sacroiliac joint (15%, unilateral)(b)ear > bones, tendon, bursaInvolvement of hip + spine is rare◊Radiologic features usually not seen until 6-12 years after initial attack◊Radiologic features present in 50% of inflicted patients @Soft tissues√ calcific deposits in gouty tophi in 50% (sodium urate crystals not radiopaque, only after calcium deposition)√ eccentric juxta-articular lobulated soft-tissue masses (hand, foot, ankle, elbow, knee)√ bilateral effusion of bursae olecrani (PATHOGNOMONIC)√ aural calcification@Joint√ preservation of joint space initially (important clue!)√ absence of periarticular demineralization (DDx:rheumatoid arthritis)√ erosion of joint margins (resembling rheumatoid arthritis) but with sclerosis√ cartilage destruction (late in course of disease)√ periarticular swelling (in acute monarticular gout)√ chondrocalcinosis (menisci, articular cartilage of knee) resulting in secondary osteoarthritis √ round / oval subarticular cysts up to 3 cm@Bone√ "punched-out" lytic bone lesion ± sclerosis of margin= "mouse / rat bite" from erosion of long-standing soft-tissue tophus√ "overhanging margin" (40%) = elevated osseous spicule in sites of tophus formation associated with erosion of adjacent bone (in intra- and extra-articular locations) (HALLMARK)√ ischemic necrosis of femoral / humeral heads√ bone infarction due to deposits at vascular basement membrane (DDx: bone island) *Coexisting disorders:* 1.Psoriasis2.Glycogen storage disease Type I3.Hypo- and hyperparathyroidism4.Down syndrome5.Lesch-Nyhan syndrome (choreoathetosis, spasticity, mental retardation, self-mutilation of lips + fingertips)◊NOT associated with rheumatoid arthritis!*Rx:*colchicine, allopurinol (effective treatment usually does not improve roentgenograms)

Notes:

GRANULOCYTIC SARCOMA
=CHLOROMA = MYELOBLASTOMA=solid tumor consisting of primitive precursors of the granulocytic series of WBCs (myeloblasts, promyelocytes, myelocytes)*Associated with:*AML (3-8%), CML (1%), polycythemia rubra vera, myelofibrosis with myeloid metaplasia, hypereosinophilic syndrome ● 60% are of green color (chloroma) due to high levels of myeloperoxidase (30% are white / gray / brown depending on preponderance of cell type + oxidative state of myeloperoxidase)Location:orbit, subcutaneous tissue, paranasal sinus, lymph node, bone, organs; often multipleSite:propensity for bone marrow (arises from bone marrow traversing haversian canal + reaching the periosteum), perineural + epidural tissue√ osteolysis with ill-defined margins√ homogeneous enhancement on CT / MR(DDx to hematoma / abscess)MR: √ isointense to brain / bone marrow / muscle on T1WI + T2WI*Prognosis:*resolution under chemotherapy ± radiation therapy; recurrence rate of 23%*DDx:*osteomyelitis, histiocytosis X, neuroblastoma, lymphoma, multiple myeloma

Notes:

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HEMANGIOENDOTHELIAL SARCOMA
= HEMANGIOENDOTHELIOMA =HEMANGIOEPITHELIOMA=neoplasm of vascular endothelial cells of intermediate aggressiveness with either benign or malignant behavior*Histo:*irregular anastomosing vascular channels lined by one / several layers of atypical anaplastic endothelial cells*Age:*4th-5th decade; M:F = 2:1 ● history of trauma / irradiation

Soft-tissue Hemangioendothelioma (common) Osseous Hemangioendothelioma (rare)

Notes:

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Soft-tissue Hemangioendothelioma (common)
Location:deep tissues of extremitiesSite:in 50% closely related to a vessel (often a vein)

Notes:

Osseous Hemangioendothelioma (rare)

*Age:*2nd-3rd decade of life; M > FLocation:calvarium, spine, femur, tibia, humerus, pelvis; multicentric lesions in 30% often with regional distribution (less aggressive)√ eccentric lesion in metaphysis of long bones√ osteolytic aggressively destructive area with indistinct margins (high grade)√ well-demarcated margins with scattered bony trabeculae (low grade)√ osteoblastic area in vertebrae, contiguous through several vertebraeMetastases to:lung (early)*Prognosis:*26% 5-year survival rate*DDx:*aneurysmal bone cyst, poorly differentiatedfibrosarcoma, highly vascular metastasis, alveolar rhabdomyosarcoma

Notes:

HEMANGIOMA
A.CAPILLARY HEMANGIOMA (most common)=small-caliber vessels lined by flattened epithelium Site:skin, subcutaneous tissue; vertebral body*Age:*first few years of life(a)Juvenile capillary hemangioma = strawberry nevus*Prevalence:*1:200 births; in 20% multiple*Prognosis:*involutes in 75-90% by age 7 years(b)Verrucous capillary hemangioma(c)Senile capillary hemangioma√ enlarged arteries + arteriovenous shunting√ pooling of contrast material B.CAVERNOUS HEMANGIOMA=dilated blood-filled spaces lined by flattened endotheliumSite:deeper soft tissues, frequently intramuscular; calvarium*Age:*childhood√ phleboliths = dystrophic calcification in organizing thrombus√ large cystic spaces√ enlarged arteries + arteriovenous shunting√ pooling of contrast material*Prognosis:*NO involution C. ARTERIOVENOUS HEMANGIOMA =persistence of fetal capillary bed with abnormal communications of an increased number of normal / abnormal arteries and veins*Etiology:*(?) congenital arteriovenous malformation*Age:*young patientsSite:soft tissues(a)superficial lesion without arteriovenous shunting(b)deep lesion with arteriovenous shunting ● limb enlargement, bruit ● distended veins, overlying skin warmth ● Branham sign = reflex bradycardia after compression√ large tortuous serpentine feeding vessels √ fast blood flow + dense staining√ early draining veins D.VENOUS HEMANGIOMA=thick-walled vessels containing muscleSite:deep soft tissues of retroperitoneum, mesentery, muscles of lower extremities*Age:*adulthood√ ± phleboliths√ serpentine vessels with slow blood flow√ vessels oriented along long axis of extremity (in 78%) + neurovascular bundle (in 64%)√ multifocal involvement (in 37%)√ muscle atrophy with increased subcutaneous fat√ may be normal on arterial angiography

Osseous Hemangioma Soft-tissue Hemangioma

Notes:

Osseous Hemangioma *Incidence:*10%*Histo:*mostly cavernous; capillary type is rare*Age:*4th-5th decade; M:F = 2:1 ● usually asymptomatic @Vertebra (28% of all skeletal hemangiomas)*Incidence:*in 5-11% of all autopsies; multiple in 1/3*Histo:*capillary hemangioma interspersed in fatty matrix◊The larger the degree of fat overgrowth, the less likely the lesion will be symptomatic!*Age:*>40 years; femaleLocation:in lower thoracic / upper lumbar spine√ "accordion" / "corduroy" / "honeycomb" vertebra= coarse vertical trabeculae with osseous reinforcement adjacent to bone resorption caused by vascular channels (also in multiple myeloma, lymphoma, metastasis)√ bulge of posterior cortex√ extraosseous extension beyond bony lesion (with cord compression)√ paravertebral soft-tissue extension√ lesion enhancementCT: √ polka-dot appearance = small punctate areas of sclerosis (= thickened vertical trabeculae)MR: √ mottled pattern of low-to-high intensity on T1WI + very-high intensity on T2WI depending on degree of adipose tissue (CHARACTERISTIC)*Cx:*vertebral collapse (unusual), spinal cord compression@Calvarium (20% of all hemangiomas)Location:frontal / parietal regionSite:diploe√ <4 cm round osteolytic lesion with sunburst / weblike / spoke-wheel appearance of trabecular thickening √ expansion of outer table to a greater extent than inner table producing palpable lump@Flat bones & long bones (rare)-ribs, clavicle, mandible, zygoma, nasal bones, metaphyseal ends of long bones (tibia, femur, humerus)√ radiating trabecular thickening√ bubbly bone lysis creating honeycomb / latticelike / "hole-within-hole" appearanceMR: √ serpentine vascular channels with low signal intensity on T1WI + high signal intensity on T2WI (= slow blood flow) / low signal intensity on all sequences (= high blood flow)NUC (bone / RBC-labeled scintigraphy): √ photopenia / moderate increased activity

Notes:

Soft-tissue Hemangioma *Incidence:*7% of all benign tumors; most frequent tumor of infancy + childhoodNonvascular elements: fat, smooth muscle, fibrous tissue, thrombus, bone√Fat overgrowth may be so extensive that some areas of lesion may be misdiagnosed as lipoma!*Age:*primarily in children; M < F ● intermittent change in size ● painful ● bluish discoloration of overlying skin (rare) ● may dramatically increase in size during pregnancyLocation:usually intramuscular; synovia (<1% of all hemangiomas); common in head and neck√ nonspecific soft-tissue mass√ may extend into bone√ ± longitudinal / axial bone overgrowth (secondary to chronic hyperemia)√ may contain phleboliths (30% of lesions, SPECIFIC)√ nonspecific curvilinear / amorphous calcifications√ may contain such large amounts of fat as to be indistinguishable from lipomaCT: √ poorly defined mass with attenuation similar to muscle√ areas of decreased attenuation approximating subcutaneous fat (= fat overgrowth)MR: √ poorly marginated mass isointense to muscle on T1WI√ areas with increased signal intensity on T1WI in periphery of lesion extending into septations (= fat)√ well-marginated markedly hyperintense mass on T2WI (increased free water content in stagnant blood)√ tubular structures with blood flow characteristics (flow void / inflow enhancement; contrast enhancement)√ phleboliths as low-intensity areas inside lesion√ high-signal-intensity areas on T1WI + T2WI (= hemorrhage)US: √ complex mass√ low-resistance arterial signal (occasionally) *Synovial Hemangioma* ● repetitive bleeding into jointLocation:knee (60%), elbow (30%)*DDx:*hemophilic arthropathy (polyarticular)

Notes:

HEMANGIOPERICYTOMA
=borderline tumor with benign / locally aggressive / malignant behavior (counterpart of glomus tumor)*Age:*4th-5th decade; M:F = 1:1*Path:*large vessels predominantly in tumor periphery*Histo:*cells packed around vascular channels containing cystic + necrotic areas; arising from cells of Zimmerman that are located around vessels@Soft tissue=deep-seated well-circumscribed lesion arising in muscleLocation:lower extremity in 35% (thigh), pelvic cavity, retroperitoneum ● painless slowly growing mass up to 20 cm@Bone (rare)Location:lower extremity, vertebrae, pelvis, skull (dura similar to meningioma)√ osteolytic lesions in metaphysis of long / flat bone√ subperiosteal large blowout lesion (similar to aneurysmal bone cyst)Angio: √ displacement of main artery√ pedicle of tumor feeder arteries√ spider-shaped arrangement of vessels encircling tumor√ small corkscrew arteries√ dense tumor stain*DDx:*hemangioendothelioma, angiosarcoma

Notes:

HEMOCHROMATOSIS

1.PRIMARY HEMOCHROMATOSIS=autosomal recessive / indeterminate inheritance (abnormal iron-loading gene) in thalassemia, sideroblastic anemia2.SECONDARY HEMOCHROMATOSIS=excessive iron absorption in anemias, myelofibrosis, portacaval shunt, exogenous administration of iron, porphyria cutanea tarda, beer brewed in iron vessels + deposition of excessive iron in liver, pancreas, spleen, GI tract, kidney, gonads, heart, endocrine glands (pituitary, hypothalamus)*Age:* >40 years; M:F = 10:1 (females protected by menstruation) ● cirrhosis ● "bronzed diabetes" ● congestive heart failure ● skin pigmentation ● hypogonadism ● arthritic symptoms (30%) ● increase in serum iron@SkeletonSite:most commonly in hands (metacarpal heads, particularly 2nd + 3rd MCP joints), carpal + proximal interphalangeal joints, knees, hips√ generalized osteoporosis√ small subchondral cystlike rarefactions with fine rim of sclerosis (metacarpal heads)√ arthropathy in 50% with iron deposition in synovium√ uniform joint space narrowing√ enlargement of metacarpal heads√ eventually osteophyte formation√ chondrocalcinosis in >60%, knees most commonly affected(a)calcium pyrophosphate deposition (inhibition of pyrophosphatase enzyme within cartilage which hydrolyzes pyrophosphate to soluble orthophosphate)(b)calcification of triangular cartilage of wrist, menisci, annulus fibrosus, ligamentum flavum, symphysis pubis, Achilles tendon, plantar fascia@Brain MRI:√ marked loss in signal intensity of anterior lobe of pituitary gland (iron deposition)*Cx:*hepatoma (in 30%)*Prognosis:*death from CHF (30%), death from hepatic failure (25%)*DDx:*(1)Pseudogout (no arthropathy)(2)Psoriatic arthritis (skin + nail changes)(3)Osteoarthritis (predominantly distal joints in hands)(4)Rheumatoid arthritis(5)Gout (may also have chondrocalcinosis)

Notes:

HEMOPHILIA
=X-linked deficiency / functional abnormality of coagulation factor VIII (= hemophilia A) in >80% / factor IX (= hemophilia B = Christmas disease)*Incidence:*1:10,000 males@Hemarthrosis (most common)*Histo:*hypertrophic synovial membrane with pannus formation that erodes cartilage, loss of subchondral bone plate, formation of subarticular cysts ● tense red warm joint with decreased range of motion (muscle spasm) ● fever, elevated WBC (DDx: septic arthritis)Location:in knee, ankle, elbow√ soft-tissue swelling of joint√ enlargement of epiphysis (secondary to synovial hyperemia)√ thinning of joint cartilage (particularly patella) secondary to cartilage destruction√ erosion of articular surface with multiple subchondral cysts√ superimposed degenerative joint disease√ "squared" patella√ widening of intercondylar notch √ medial "slanting" of tibiotalar joint√ juxta-articular osteoporosis@Hemophilic pseudotumor (1-2%)=posthemorrhagic cystic swelling within muscle + bone characterized by pressure necrosis + destruction(a)juvenile form = usually multiple intramedullary expansile lesions without soft-tissue mass in small bones of hand / feet (before epiphyseal closure)(b)adult form = usually single intramedullary expansile lesion with large soft-tissue mass in ilium / femur(c)soft-tissue involvement of retroperitoneum (psoas muscle), bowel wall, renal collecting system√ mixed cystic expansile lesion√ bone erosion + pathologic fractureCT: √ sometimes encapsulated mass containing areas of low attenuation + calcificationsMR: √ hemorrhage of varying ageN.B.:Needle aspiration / biopsy / excision may cause fistulae / infection / uncontrolled bleeding!*Rx:*palliative radiation therapy (destroys vessels prone to bleed) + transfusion of procoagulation factor concentrate

Notes:

HEREDITARY HYPERPHOSPHATASIA
="JUVENILE PAGET DISEASE" = rare autosomal recessive disease with sustained elevation of serum alkaline phosphatase, especially in individuals of Puerto Rican descent*Histo:*rapid turnover of lamellar bone without formation of cortical bone; immature woven bone is rapidly laid down, but simultaneous rapid destruction prevents normal maturation*Age:*1st-3rd year; usually stillborn ● rapid enlargement of calvarium + long bones ● dwarfism ● cranial nerve deficit (blind, deaf) ● hypertension ● frequent respiratory infections ● pseudoxanthoma elasticum ● elevated alkaline phosphatase√ deossification = decreased density of long bones with coarse trabecular pattern√ metaphyseal growth deficiency√ wide irregular epiphyseal lines (resembling rickets in childhood), persistent metaphyseal defects (40% of adults)√ bowing of long bones + fractures with irregular callus√ widened medullary canal with cortical thinning (cortex modeled from trabecular bone)√ skull greatly thickened with wide tables, cotton wool appearance√ vertebra planaOB-US: √ diagnosis suspected in utero in 20%*Cx:*pathologic fractures; vertebra plana universalis*DDx:*(1)Osteogenesis imperfecta(2)Polyostotic fibrous dysplasia(3)Paget disease (> age 20, not generalized)(4)Pyle disease (spares midshaft)(5)van Buchem syndrome (only diaphyses > age 20, no long-bone bowing)(6)Engelmann syndrome (lower limbs)

Notes:

HEREDITARY MULTIPLE EXOSTOSES
=DIAPHYSEAL ACLASIS*Inheritance:*autosomal dominant (unaffected female may be carrier)*Age:*discovered between 2 and 10 years; M:F = 2:1*Path:*ectopic cartilaginous rest in metaphysis + defect in periosteum; cap of hyaline cartilage; often bursa formation over cap ● usually painless mass near joints ● tendons, blood vessels, nerves may be impaired ● mechanical limitation of joint movementLocation:multiple + usually bilateral; common sites are knee, elbow, scapula, pelvis, ribsSite:metaphyses of long bones near epiphyseal plate (distance to epiphyseal line increases with growth)√ cortex + cancellous bone of exostosis contiguous to host bone√ slope on epiphyseal side + right angle on diaphyseal side of stalk = points away from joint + toward center of shaft√ occasionally small punctate calcifications incartilaginous cap√ shortening of 4th + 5th metacarpals√ supernumerary fingers / toes√ Madelung / reversed Madelung deformity = radius usually longer + bowed√ occasionally results in disproportionate shortening of an extremity, radioulnar synostosis, dislocation of radial head *Prognosis:*exostosis begins in childhood; stops growing when nearest epiphyseal center fusesCx:(1)Cord compression secondary to involvement of posterior spinal elements(2)Malignant transformation to chondrosarcoma in <5%; iliac bone commonest site; growth with irregularity of outline + fuzziness; sudden painful growth spurt

Notes:

HEREDITARY SPHEROCYTOSIS
=autosomal dominant congenital hemolytic anemia*Age:*anemia begins in early infancy to late adulthood ● rarely severe anemia ● jaundice ● spherocytes in peripheral smear√ bone changes rare (due to mild anemia); long bones rarely affected√ widening of diploe with displacement + thinning of outer table√ hair-on-end appearance*Rx:*splenectomy corrects anemia even though spherocytemia persists√ improvement in skeletal alterations following splenectomy

Notes:

HERNIATION PIT
=SYNOVIAL HERNIATION PIT = CONVERSION DEFECT=ingrowth of fibrous + cartilaginous elements from adjacent joint through perforation in cortex*Histo:*fibroalveolar tissue*Age:*usually in older individuals ● may be symptomatic ● no clinical significanceLocation:anterior superolateral aspect of proximal femoral neck; uni- or bilateralSite:subcortical√ well-circumscribed round lucency√ usually <1 cm in diameter√ reactive thin sclerotic border√ hyperintense area on T2WI √ bone scan may be positive

Notes:

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HOLT-ORAM SYNDROME
Autosomal dominant; M < F *Associated with CHD:*secundum type ASD (most common), VSD, persistent left SVC, tetralogy, coarctation ● intermittent cardiac arrhythmia ● bradycardia (50-60/min)Location:upper extremity only involved; symmetry of lesions is the rule; left side may be more severely affected√ aplasia / hypoplasia of radial structures: thumb, 1st metacarpal, carpal bones, radius√ "fingerized" hypoplastic thumb / triphalangeal thumb√ slender elongated hypoplastic carpals + metacarpals√ hypoplastic radius; absent radial styloid√ shallow glenoid fossa (voluntary dislocation of shoulder common)√ hypoplastic clavicula√ high arched palate√ cervical scoliosis√ pectus excavatum

Notes:

HOMOCYSTINURIA
Autosomal recessive disorder *Etiology:*cystathionine B synthetase deficiency results in defective methionine metabolism with accumulation of homocystine + homocysteine in blood and urine; causes defect in collagen / elastin structure ● thromboembolic phenomena due to stickiness of platelets ● ligamentous laxity ● downward + inward dislocation of lens (DDx: upward + outward dislocation in Marfan syndrome) ● mild / moderate mental retardation ● crowding of maxillary teeth and protrusion of incisors ● malar flush√ arachnodactyly in 1/3 (DDx: Marfan syndrome)√ microcephaly√ enlarged paranasal sinuses√ osteoporosis of vertebrae (biconcave / flattened / widened vertebrae)√ scoliosis√ pectus excavatum / carinatum (75%)√ osteoporosis of long bones (75%) with bowing + fracture√ children: metaphyseal cupping (50%); enlargement of ossification centers in 50% (knee, carpal bones); epiphyseal calcifications (esp. in wrist, resembling phenylketonuria); delayed ossification√ Harris lines = multiple growth lines√ genu valgum, coxa valga, coxa magna, pes cavus√ premature vascular calcifications*Prognosis:*death from occlusive vascular disease / minor vascular trauma

	Marfan Syndrome	Homocystinuria
Inheritance:	autosomal dominant	autosomal recessive
Biochemical defect:	not known	cystathionine synthetase
Osteoporosis:	no	yes
Spine:	scoliosis	biconcave vertebrae
Lens dislocation:	upward	downward
Arachnodactyly:	100%	33%

Notes:

HYPERPARATHYROIDISM
=uncontrolled production of parathyroid hormone*Age:*middle age; M:F = 1:3*Histo:*decreased bone mass secondary to increased number of osteoclasts, increased osteoid volume (defect in mineralization), slightly increased number of osteoblasts ● increase in parathyroid hormone (100%) ● increase in serum alkaline phosphatase (50%) ● elevation of serum calcium (due to accelerated bone turnover and increased calcium absorption) +decrease in serum phosphate (30%) ● hypotonicity of muscles, weakness, constipation, difficulty in swallowing, duodenal / gastric peptic ulcer disease (secondary to hypercalcemia) ● polyuria, polydipsia (hypercalciuria + hyperphosphaturia) ● renal colic + renal insufficiency (nephrocalculosis + nephrocalcinosis) ● rheumatic bone pain + tenderness (particularly at site of brown tumor), pathologic fracture secondary to brown tumorA.BONE RESORPTION(a)subperiosteal (most constant + specific finding; virtually PATHOGNOMONIC of hyperparathyroidism):√ lacelike irregularity of cortical margin; may progress to scalloping / spiculation (pseudoperiostitis)Site:phalangeal tufts (earliest involvement), radial aspect of middle phalanx of 2nd + 3rd finger beginning in proximal metaphyseal region (early involvement), bandlike zone of resorption in middle / base of terminal tuft, distal end of clavicles, medial tibia plateau, medial humerus neck, medial femoral neck, distal ulna, superior + inferior margins of ribs in midclavicular line, lamina dura of skull and teeth(b)subchondral:√ pseudowidening of joint space√ collapse of cortical bone + overlying cartilage with development of erosion, cyst, joint narrowing (similar to rheumatoid arthritis)Site:DIP joint (most commonly 4th + 5th digit), MCP joint, PIP joint, distal clavicle, acromioclavicular joint (clavicular side), "pseudowidening" of sacroiliac joint (iliac side), sternoclavicular joint, symphysis pubis, "scalloping" of posterior surface of patella, Schmorl nodes; typically polyarticular(c)cortical (due to osteoclastic activity within haversian canal):√ intracortical tunneling√ scalloping along inner cortical surface (endosteal resorption)(d)trabecular:√ spotty deossification with indistinct + coarse trabecular pattern√ granular salt and pepper skull√ loss of distinction between inner and outer table√ ground-glass appearance(e)subligamentous:√ bone resorption with smooth scalloped / irregular ill-defined marginsSite:inferior surface of calcaneus (long plantar tendons + aponeurosis), inferior aspect of distal clavicle (coracoclavicular ligament), greater trochanter (hip abductors), lesser trochanter (iliopsoas), anterior inferior iliac spine (rectus femoris), humeral tuberosity (rotator cuff), ischial tuberosity (hamstrings), proximal extensor surface of ulna (anconeus), posterior olecranon (triceps) B.BONE SOFTENING√ basilar impression of skull√ wedged vertebrae, kyphoscoliosis, biconcave vertebral deformities√ bowing of long bones√ slipped capital femoral epiphysisC.BROWN TUMOR=OSTEOCLASTOMA*Cause:*PTH-stimulated osteoclastic activity (more frequent in 1° HPT; in 1.5% of 2° HPT)*Path:*localized replacement of bone by vascularized fibrous tissue (osteitis fibrosa cystica) containing giant cells; lesions may become cystic following necrosis + liquefactionLocation:jaw, pelvis, rib, metaphyses of long bones (femur), facial bones, axial skeletonSite:often eccentric / cortical; frequently solitary√ expansile lytic well-marginated cystlike lesion(DDx: giant cell tumor)√ endosteal scalloping√ destruction of midportions of distal phalanges with telescoping D.OSTEOSCLEROSISMore frequent in 2° HPT *Cause:*? PTH-stimulated osteoblastic activity, ? role of calcitonin (poorly understood)Site:strong predilection for axial skeleton, pelvis, ribs, clavicles, metaphysis + epiphysis of appendicular skeleton√ "rugger jersey spine" (resembling the stripes on rugby jerseys) = sclerosis of vertebral endplates with intervening normal osseous density E.SOFT-TISSUE CALCIFICATIONMore frequent in 2° HPT; metastatic calcification when Ca x P product >70 mg/dL (a)cornea, viscera (lung, stomach, kidney)(b)periarticular in hip, knee, shoulder, wrist(c)arterial tunica media (resembling diabetes mellitus)(d)Chondrocalcinosis (15-18%) = calcification of hyaline / fibrous cartilage in menisci, wrist, shoulder, hip, elbow F.EROSIVE ARTHROPATHY ● asymptomatic √ simulates rheumatoid arthritis with preserved joint spaces G.PERIOSTEAL NEW-BONE FORMATION*Cause:*PTH-stimulation of osteoblastsSite:pubic ramus along iliopectineal line (most frequent), humerus, femur, tibia, radius, ulna, metacarpals, metatarsals, phalanges√ linear new bone paralleling cortical surface; may be laminated; often separated from cortex by radiolucent zone√ increase in cortical thickness (if periosteal reaction becomes incorporated into adjacent bone) *Sequelae:* 1.Renal stones / nephrocalcinosis (70%)2.Increased osteoblastic activity (25%) ● increased alkaline phosphatase(a)osteitis fibrosa cystica√ subperiosteal bone resorption + cortical tunneling√ brown tumors (primary HPT)(b)bone softening√ fractures3.Peptic ulcer disease (increased gastric secretion from gastrinoma)4.Calcific pancreatitis5.Soft-tissue calcifications (2° HPT)6.Marginal joint erosions + subarticular collapse (DIP, PIP, MCP)

Primary Hyperparathyroidism Secondary Hyperparathyroidism Tertiary Hyperparathyroidism Ectopic Parathormone Production

Notes:

Primary Hyperparathyroidism =pHPT = 1° HPT = hypercalcemia due to uncontrolled secretion of parathormone by one / more hyperfunctioning parathyroid glands featuring(1)brown tumor(2)chondrocalcinosis (20-30%)requires surgical Rx*Incidence:*25 / 100,000 per year; incidence of bone lesions in HPT is 25-40%*Etiology:* (a)Parathyroid adenoma (87%): single (80%); multiple (7%)(b)Parathyroid hyperplasia (10%): chief cell (5%); clear cell (5%)(c)Parathyroid carcinoma (3%)*Histo:*increased number of osteoclasts, increased osteoid volume (defect in mineralization), slightly increased osteoblasts = decreased bone mass*Age:*3rd-5th decade; M:F = 1:3*Associated with:* (a)Wermer syndrome = MEA I (+ pituitary adenoma + pancreatic islet cell tumor)(b)Sipple syndrome = MEA II (+ medullary thyroid carcinoma + pheochromocytoma)X-ray (skeletal involvement in 20%): √ thin cortices with lacy cortical pattern (subperiosteal bone resorption)√ brown tumor (particularly in jaw + long bones)√ osteitis cystica fibrosa (= intertrabecular fibrous connective tissue)NUC: √ normal bone scan in 80%√ foci of abnormal uptake: calvarium (especially periphery), mandible, sternum, acromioclavicular joint, lateral humeral epicondyles, hands√ increased uptake in brown tumors√ extraskeletal uptake: cornea, cartilage, joint capsules, tendons, periarticular areas, lungs, stomach√ normal renal excretion [except in stone disease / calcium nephropathy (10%)]*Rx:*pathologic glands identified by experienced surgeons in 90-95% on initial neck exploration (ectopic + supernumerary glands often overlooked at operation; recurrent hypercalcemia in 3-10%)Surgical risk for repeat surgery6.6%recurrent laryngeal nerve injury20.0%permanent hypoparathyroidism<1.0%perioperative mortality

Notes:

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Secondary Hyperparathyroidism =sHPT = 2° HPT = diffuse / adenomatous hyperplasia of all four parathyroid glands as a compensatory mechanism in any state of hypocalcemia featuring(1) soft-tissue calcifications (2) osteosclerosis◊requires medical Rx*Etiology:* (a)renal osteodystrophy (renal insufficiency + osteomalacia / rickets)(b)calcium deprivation, maternal hypoparathyroidism, pregnancy, hypovitaminosis D(c)rise in serum phosphate leading to decrease in calcium by feedback mechanism ● low to normal calcium levels ● Ca^{2+} PO_4^{2-} solubility product often exceededNUC: √ "superscan" in 2° HPT:√ absent kidney sign√ increased bone-to-soft tissue uptake ratio√ increased uptake in calvarium, mandible, acromioclavicular region, sternum, vertebrae, distal third of long bones, ribs√ diffuse Tc-99m MDP uptake in lungs (60%)

Notes:

Tertiary Hyperparathyroidism =tHPT = 3° HPT = development of autonomous PTH adenoma in patients with chronically overstimulated hyperplastic parathyroid glands (renal insufficiency);requires surgical Rx*Clue:*(a)intractable hypercalcemia(b)inability to control osteomalacia by vitamin D administration

Notes:

Ectopic Parathormone Production =pseudohyperparathyroidism as paraneoplastic syndrome in bronchogenic carcinoma + renal cell carcinoma

Notes:

HYPERTROPHIC OSTEOARTHROPATHY

=HYPERTROPHIC PULMONARY OSTEOARTHROPATHY*Etiology:*(1)Release of vasodilators which are not metabolized by lung(2)Increased flow through AV shunts(3)Reflex peripheral vasodilation (vagal impulses)(4)Hormones: estrogen, growth hormone, prostaglandinA.THORACIC CAUSES(a)malignant tumor (0.7-12%): bronchogenic carcinoma (88%), mesothelioma, lymphoma, pulmonary metastasis from osteogenic sarcoma, melanoma, renal cell carcinoma, breast cancer(b)benign tumor: benign pleural fibroma, tumor of ribs, thymoma, esophageal leiomyoma, pulmonary hemangioma, pulmonary congenital cyst(c)chronic infection / inflammation: pulmonary abscess, bronchiectasis, blastomycosis, TB (very rare); cystic fibrosis, interstitial fibrosis(d)congenital heart disease with R-to-L shuntB. EXTRATHORACIC CAUSES(a)GI tract: ulcerative colitis, amebic + bacillary dysentery, intestinal TB, Whipple disease, Crohn disease, gastric ulcer, bowel lymphoma, gastric carcinoma(b)liver disease: biliary + alcoholic cirrhosis, posthepatic cirrhosis, chronic active hepatitis, bile duct carcinoma, benign bile duct stricture, amyloidosis, liver abscess(c)undifferentiated nasopharyngeal carcinoma, pancreatic carcinoma, chronic myelogenous leukemia ● burning pain, painful swelling of limbs, and stiffness of joints: ankles (88%), wrists (83%), knees (75%), elbows (17%), shoulders (10%), fingers (7%) ● peripheral neurovascular disorders: local cyanosis, areas of increased sweating, paresthesia, chronic erythema, flushing + blanching of skin ● hippocratic fingers + toes (clubbing) ● hypertrophy of extremities (soft-tissue swelling)Location:tibia + fibula (75%), radius + ulna (80%), proximal phalanges (60%), femur (50%), metacarpus + metatarsus (40%), humerus + distal phalanges (25%), pelvis (5%); unilateral (rare)Site:in diametaphyseal regions√ periosteal proliferation of new bone, at first smooth then undulating + rough, most conspicuous on concavity of long bones (dorsal + medial aspects)√ regression of periosteal reaction after thoracotomy√ soft-tissue swelling ("clubbing") of distal phalangesBone scan (reveals changes early with greater sensitivity + clarity): √ symmetric diffusely increased uptake along cortical margins of diaphysis + metaphysis of tubular bones of the extremities with irregularities√ increased periarticular uptake (= synovitis)√ scapular involvement in 2/3√ mandible ± maxilla abnormal in 40%

Notes:

HYPERVITAMINOSIS A

*Age:*usually infants + children ● anorexia, irritability ● loss of hair, dry skin, pruritus, fissures of lips ● jaundice, enlargement of liver√ separation of cranial sutures secondary to hydrocephalus (coronal > lambdoid) in children <10 years of age, may appear within a few days√ symmetrical solid periosteal new-bone formation along shafts of long + short bones (ulna, clavicle)√ premature epiphyseal closure + thinning of epiphyseal plates√ accelerated growth√ tendinous, ligamentous, pericapsular calcifications√ changes usually disappear after cessation of vitamin A ingestion*DDx:*Infantile cortical hyperostosis (mandible involved)

Notes:

HYPERVITAMINOSIS D

=excessive ingestion of vitamin D (large doses act like parathormone) ● loss of appetite, drowsiness, headaches ● polyuria, polydipsia, renal damage ● anemia ● diarrhea ● convulsions ● excessive phosphaturia (parathormone decreases tubular absorption) ● hypercalcemia + hypercalciuria√ deossification√ widening of provisional zone of calcification√ cortical + trabecular thickening√ alternating bands of increased + decreased density near / in epiphysis (zone of provisional calcification)√ vertebra outlined by dense band of bone + adjacent radiolucent line within√ dense calvarium√ metastatic calcinosis in (a) arterial walls (between age 20 and 30) (b) kidneys = nephrocalcinosis (c) periarticular tissue (puttylike) (d) premature calcification of falx cerebri (most consistent sign!)

Notes:

HYPOPARATHYROIDISM

Etiology: A.Idiopathic Hypoparathyroidism=rare condition of unknown cause ● round face, short dwarflike, obese ● mental retardation ● cataracts ● dry scaly skin, atrophy of nails ● dental hypoplasia (delayed tooth eruption, impaction of teeth, supernumerary teeth)B.Secondary Hypoparathyroidism=accidental removal / damage to parathyroid glands in thyroid surgery / radical neck dissection (5%); I-131 therapy (rare); external beam radiation; hemorrhage; infection; thyroid carcinoma; hemochromatosis (iron deposition) ● tetany = neuromuscular excitability (numbness, cramps, carpopedal spasm, laryngeal stridor, generalized convulsions) ● hypocalcemia + hyperphosphatemia ● normal / low serum alkaline phosphatase√ premature closure of epiphyses√ hypoplasia of tooth enamel + dentine; blunting of roots√ generalized increase in bone density in 9%√ localized thickening of skull√ sacroiliac sclerosis√ bandlike density in metaphysis of long bones (25%), iliac crest, vertebral bodies√ thickened lamina dura (inner table) + widened diploe√ deformed hips with thickening + sclerosis of femoral head + acetabulum@Soft tissue√ intracranial calcifications in basal ganglia, choroid plexus, occasionally in cerebellum√ calcification of spinal and other ligaments√ subcutaneous calcifications√ ossification of muscle insertions√ ectopic bone formation

	HypoPT	PseudoHypoPT	PseudopseudoHypoPT
Serum-Ca	down	down	norm
Serum-P	up	up	norm
AlkaPhos	down/norm	down/norm	norm

Response to PTH-Injection	Norm / HypoPT	PseudoHypoPT
Urine-AMP	up	norm
Urine-P	up	norm
Plasma-AMP	up	norm

Notes:

HYPOPHOSPHATASIA
=autosomal recessive congenital disease with low activity of serum-, bone-, liver-alkaline phosphatase resulting in poor mineralization (deficient generation of bone crystals)*Incidence:*1:100,000*Histo:*indistinguishable from rickets ● phosphoethanolamine in urine as precursor of alkaline phosphatase ● normal serum calcium + phosphorus A.GROUP I = neonatal = congenital lethal form√ marked demineralization of calvarium ("caput membranaceum" = soft skull)√ lack of calcification of metaphyseal end of long bones√ streaky irregular spotty margins of calcification√ cupping of metaphysis√ angulated shaft fractures with abundant callus formation√ short poorly ossified ribs√ poorly ossified vertebrae (especially neural arches)√ small pelvic bonesOB-US: √ high incidence of intrauterine fetal demise√ increased echogenicity of falx (enhanced sound transmission secondary to poorly mineralized calvarium)√ poorly mineralized short bowed tubular bones + multiple fractures√ poorly mineralized spine√ short poorly ossified ribs√ polyhydramnios*Prognosis:*death within 6 months B.GROUP II = juvenile severe formonset of symptoms within weeks to months ● moderate / severe dwarfism ● delayed weight bearing√ resembles rickets√ separated cranial sutures; craniostenosis in 2nd year*Prognosis:*50% mortality C.GROUP III = adult mild formrecognized later in childhood / adolescence / adulthood ● dwarfism√ clubfoot, genu valgum√ demineralization of ossification centers (at birth / 3-4 months of age)*Prognosis:*excellent; after 1 year no further progression D.GROUP IV = latent formheterozygous state ● normal / borderline levels of alkaline phosphatase ● patients are small for age ● disturbance of primary dentition√ bone fragility + healed fractures√ enlarged chondral ends of ribs√ metaphyseal notching of long bones√ Erlenmeyer flask deformity of femur

Notes:

HYPOTHYROIDISM

A.Childhood = CRETINISM:*Frequency:*1:4,000 live births have congenital hypothyroidism*Cause:*sporadic hypoplasia / ectopia of thyroid√ delayed skeletal maturation (appearance + growth of ossification centers, epiphyseal closure)√ fragmented stippled epiphyses√ wide sutures / fontanelles with delayed closure√ delayed dentition √ delayed / decreased pneumatization of sinuses + mastoids√ hypertelorism√ dense vertebral margins√ demineralization√ hypoplastic phalanges of 5th fingerMR: √ reduced myelination of brain (usually beginning during midgestation)OB-US: √ fetal goiter (especially in hyperthyroid mothers treated with methimazole / propylthiouracil / I-131)B.Adulthood:√ calvarial thickening / sclerosis√ wedging of dorsolumbar vertebral bodies√ coxa vara with flattened femoral head√ premature atherosclerosisNo skeletal changes with adult onset!

Notes:

INFANTILE CORTICAL HYPEROSTOSIS
=CAFFEY DISEASE=uncommon self-limiting proliferative bone disease of infancy; remission + exacerbations are common*Cause:*? infectious; ? autosomal dominant with variable expression + incomplete penetrance / sporadic occurrence (rare)*Age:*<6 months, reported in utero; M:F = 1:1*Histo:*inflammation of periosteal membrane, proliferation of osteoblasts + connective tissue cells, deposition of immature bony trabeculae ● sudden, hard, extremely tender soft-tissue swellings over bone ● irritability, fever ● ± elevated ESR, increased alkaline phosphatase ● leukocytosis, anemiaLocation:mandible (80%) > clavicle > ulna + others (except phalanges + vertebrae + round bones of wrists and ankles)Site:hyperostosis affects diaphysis of tubular bones asymmetrically, epiphyses spared√ massive periosteal new-bone formation + perifocal soft-tissue swelling√ "double-exposed" ribs √ narrowing of medullary space (= proliferation of endosteum)√ bone expansion with remodeling of old cortex*Prognosis:*usually complete recovery by 30 months*Rx:*mild analgesics, steroids **Chronic Infantile Hyperostosis** Disease may persist or recur intermittently for years√ bowing deformities, osseous bridging, diaphyseal expansion ● delayed muscular development, crippling deformities *DDx:*(1) Hypervitaminosis A (rarely <1 year of age)(2) Periostitis of prematurity (3) Healing rickets(4) Scurvy (uncommon <4 months of age)(5) Syphilis (focal destruction) (6) Child abuse(7) Prostaglandin administration (usually following 4-6 weeks of therapy) (8) Osteomyelitis(9) Leukemia (10) Neuroblastoma (11) Kinky hair syndrome (12) Hereditary hyperphosphatasia

Notes:

INFANTILE MYOFIBROMATOSIS
=GENERALIZED HAMARTOMATOSIS = CONGENITAL MULTIPLE FIBROMATOSIS = MULTIPLE VASCULAR LEIOMYOMAS = DESMOFIBROMATOSIS=rare disorder characterized by proliferation of fibroblasts*Cause:*unknown*Frequency:*most common fibromatosis in childhood*Age:*at birth (in 60%), <2 years (in 89%)*Path:*well-marginated soft-tissue lesion 0.5-3 cm in diameter with scarlike consistency ± infiltration of surrounding tissues*Histo:*spindle-shaped cells in short bundles and fascicles in periphery of lesion; hemangiopericytoma-like pattern in center with necrosis, hyalinization, calcification (1)Solitary lesion (50-75%)dermis, subcutis, muscle (86%) Location:head, neck, trunk, bone (9%), GI tract (4%)*Prognosis:*spontaneous regression in 100%; recurrence after surgical excision in 7-10%(2)Multicentric disease (25-50%)Location:skin (98%), subcutis (98%), muscle (98%), bone (57%), viscera (25-37%): lung (28%), heart (16%), GI tract (14%), pancreas (9%), liver (8%)*Prognosis:*related to extent + location of visceral lesions with cardiopulmonary + GI involvement as harbingers of poor prognosis (death in 75-80%); spontaneous regression (33%) ● firm nodules in skin, subcutis, muscle ● ± overlying scarring of skin with ulceration@SkeletonLocation:any bone may be involved; commonly in femur, tibia, rib, pelvis, vertebral bodies, calvarium; often symmetricSite:metaphysis of long bones√ eccentric lobulated lytic foci with smooth margins 0.5 -1.0 cm in size√ well-defined with narrow zone of transition√ initially no sclerosis; sclerotic margin with healing√ osseous foci may increase in size and number√ healing leaves little residual abnormality√ unusual osseous findings:√ periosteal reaction, pathologic fracture√ vertebra plana, kyphoscoliosis with posterior scalloping of vertebral bodiesNUC (bone scan): √ increased / little radiotracer uptake*DDx:*(1)Langerhans cell histiocytosis (skin lesions)(2)Neurofibromatosis (multiple masses)(3)Osseous hemangiomas / lymphangiomatosis / lipomatosis(4)Metastatic neuroblastoma(5)Multiple nonossifying fibromas(6)Enchondromatosis(7)Unusual infection(8)Fibrous dysplasia@Soft tissue√ solid mass with central necrosis√ central / peripheral solitary / multiple calcifications√ ± contrast enhancementCT: √ attenuation similar to muscleMR: √ hypo- to hyperintense mass on T1WI + T2WI*DDx:*(1)Neurofibromatosis(2)Infantile fibrosarcoma, leiomyosarcoma(3)Angiomatosis@Lung√ interstitial fibrosis, reticulonodular infiltrates√ discrete mass√ generalized bronchopneumonia@GI tract√ diffuse narrowing with multiple small filling defects

Notes:

IRON DEFICIENCY ANEMIA
*Age:*infants affected*Cause:* (1) inadequate iron stores at birth (2) deficient iron in diet (3) impaired gastrointestinal absorption of iron (4) excessive iron demands from blood loss (5) polycythemia vera (6) cyanotic CHD √ widening of diploe + thinning of tables with sparing of occiput (no red marrow)√ hair-on-end appearance of skull√ osteoporosis in long bones (most prominent in hands)√ absence of facial bone involvement

Notes:

JACCOUD ARTHROPATHY

After subsidence of frequent severe attacks of rheumatic fever *Path:*periarticular fascial + tendon fibrosis without synovitis ● rheumatic valve diseaseLocation:primarily involvement of hands; occasionally in great toe√ muscular atrophy√ periarticular swelling of small joints of hands + feet√ ulnar deviation + flexion of MCP joints most marked in 4th + 5th finger√ NO joint narrowing / erosion

Notes:

JUVENILE APONEUROTIC FIBROMA

Rare benign fibrous tumor *Histo:*cellular dense fibrous tissue with focal chondral elements infiltrating adjacent structures (= cartilaginous tumor)*Age:*children + adolescents; male preponderanceLocation:deep palmar fascia of hand + wrist√ soft-tissue mass overlying inflamed bursa (oftenmistaken for calcified bursitis)√ stippled calcifications√ interosseous soft-tissue mass of forearm + wrist√ bone erosion may occur*DDx:*synovial sarcoma, chondroma, fibrosarcoma, osteosarcoma, myositis ossificans

Notes:

KLINEFELTER SYNDROME
47,XXY (rarely XXYY) chromosomal abnormality *Incidence:*1:750 live births (probably commonest chromosomal aberration) ● testicular atrophy (hyalinization of seminiferous tubules) = small / absent testes, sterility (azoospermia) ● eunuchoid constitution: gynecomastia; paucity of hair on face + chest; female pubic escutcheon ● mild mental retardation ● high level of urinary gonadotropins + low level of 17-ketosteroids after puberty ◊NO distinctive radiological findings!√ may have delayed bone maturation√ failure of frontal sinus to develop√ small bridged sella turcica√ ± scoliosis, kyphosis√ ± coxa valga√ ± metacarpal sign (short 4th metacarpal)√ accessory epiphyses of 2nd metacarpal bilaterally **47,XXX = Superfemale Syndrome** ● usually over 6 feet tall; subnormal intelligence; frequently antisocial behavior

Notes:

KLIPPEL-TRÉNAUNAY SYNDROME
=sporadic (nonhereditary) rare mesodermal abnormality that usually affects a single lower limb characterized by a triad of: (1)port-wine nevus = unilateral large flat infiltrative cutaneous capillary hemangioma often in dermatomal distribution on affected limb; may fade in 2nd-3rd decade(2)gigantism = overgrowth of distal digits / entire extremity (especially during adolescent growth spurt) involving soft-tissue + bone(3)varicose veins on lateral aspect of affected limb; usually ipsilateral to hemangioma*Pathogenesis:* superficial lateral venous channel of large caliber thought to represent the fetal lateral limb bud vein that has failed to regress; tissue overgrowth is secondary to impaired venous return *Age:*usually in children; M:F = 1:1*Associated with:* -polydactyly, syndactyly, clinodactyly, oligodactyly, ectrodactyly, congenital dislocation of hip-hemangiomas of colon / bladder (3-10%)-spinal hemangiomas + AVMs-hemangiomas in liver / spleen-lymphangiomas of limbLocation:lower extremity (10-15 x more common than upper extremity); bilateral in <5%√ increased metatarsal / metacarpal + phalangeal size√ cortical thickening √ punctate calcifications (phleboliths) in pelvis (bowel wall, urinary bladder)√ pulmonary vein varicosities√ cystic lung lesionsVenogram: √ aplasia / hypoplasia of lower extremity veins (18-40%): ? selective flow of contrast material up the lateral venous channel may fail to opacify the deep venous system√ valveless collateral venous channels (? persistent lateral limb bud vein = Klippel-Trénaunay vein) draining into deep femoral vein / iliac veinsColor Doppler US: √ normal deep veins*DDx:*(1)**Parke-Weber syndrome**= congenital persistence of multiple microscopic AV fistulas + spectrum of Klippel-Trénaunay-Weber syndrome(2)Neurofibromatosis (café-au-lait spots, axillary freckling, cutaneous neurofibromas, macrodactyly secondary to plexiform neurofibromas, wavy cortical reaction, early fusion of growth plate, limb hypertrophy not as extensive / bilateral)(3)Beckwith-Wiedemann syndrome (aniridia, macroglossia, cryptorchidism, Wilms tumor, broad metaphyses, thickened long-bone cortex, advanced bone age, periosteal new-bone formation, hemihypertrophy)(4)Macrodystrophia lipomatosis (hyperlucency of fat, distal phalanges most commonly affected, overgrowth ceases with puberty, usually limited to digits)(5)Maffucci syndrome (cavernous hemangiomas, soft tissue hypertrophy, phleboliths, multiple enchondromas)

Notes:

LANGERHANS CELL HISTIOCYTOSIS
=HISTIOCYTOSIS X (former name)=poorly understood group of disorders characterized by proliferation of Langerhans cells (normally responsible for first-line immunologic defense in the skin)*Cause:*uncertain (? primary proliferative disorder possibly due to defect in immunoregulation; neoplasm)*Path:*influx of eosinophilic leukocytes simulating inflammation; reticulum cells accumulate cholesterol + lipids (= foam cells); sheets or nodules of histiocytes may fuse to form giant cells, cytoplasm contains (? viral) Langerhans bodies*Histo:*Langerhans cells are similar to mononuclear macrophages + dendritic cells as the two major types of nonlymphoid mononuclear cells involved in immune + nonimmune inflammatory response; derived from promonocytes (= bone marrow stem cell)*Age:*any age, mostly presenting at 1-4 years; M:F = 1:1Location:bone + bone marrow, lymph nodes, thymus, ear, liver and spleen, gallbladder, GI tract, endocrine system*DDx:*osteomyelitis, Ewing sarcoma, leukemia, lymphoma, metastatic neuroblastoma

Letterer-Siwe Disease Hand-Schüller-Christian Disease Eosinophilic Granuloma

Notes:

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Letterer-Siwe Disease =acute disseminated, fulminant form of histiocytosis X characterized by wasting, pancytopenia (from bone marrow dysfunction), generalized lymphadenopathy, hepatosplenomegaly*Incidence:*1: 2,000,000; 10% of histiocytosis X*Age:*several weeks after birth to 2 years*Path:*generalized involvement of reticulum cells; may be confused with leukemia ● hemorrhage, purpura (secondary to coagulopathy) ● severe progressive anemia / pancytopenia ● intermittent fever ● failure to grow / malabsorption + hypoalbuminemia ● skin rash: scaly erythematous seborrhea-like brown to red papulesLocation:especially pronounced behind ears, in axillary, inguinal, and perineal areas√ hepatosplenomegaly + lymphadenopathy (most often cervical)√ obstructive jaundice@ Bone involvement (50%): √ widespread multiple lytic lesions; "raindrop" pattern in calvarium*Prognosis:*70% mortality rate

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Notes:

Hand-Schüller-Christian Disease =chronic disseminated form of histiocytosis X(15-40%) in 10% characterized by a triad of(1) exophthalmos (2) diabetes insipidus (3) lytic skull lesions *Path:*proliferation of histiocytes, may simulate Ewing sarcoma*Age at onset:*5-10 years (range from birth to 40 years); M:F = 1:1 ● diabetes insipidus (30-50%) often with large lytic lesion in sphenoid bone / panhypopituitarism ● otitis media with mastoid + inner ear invasion ● exophthalmos (33%), sometimes with orbital wall destruction ● generalized eczematoid skin lesions (30%) ● ulcers of mucous membranes (gingiva, palate)@Bone√ osteolytic skull lesions with overlying soft-tissue nodules√ "geographic skull" = ovoid / serpiginous destruction of large area√ "floating teeth" with mandibular involvement√ destruction of petrous ridge + mastoids + sella turcica@Orbit√ diffuse orbital disease with multiple osteolytic bone lesions@ Soft tissue √ hepatosplenomegaly (rare) with scattered granuloma√ lymphadenopathy (may be massive)√ gallbladder wall thickening (from infiltration)@ Lung √ cyst + bleb formation with spontaneous pneumothorax (25%)√ ill-defined diffuse nodular infiltration often progressing to fibrosis + honeycomb lung*Prognosis:*spontaneous remissions + exacerbations

Notes:

Eosinophilic Granuloma =most benign variety of histiocytosis X (60-80%) localized to bone*Age:*5-10 years (highest frequency); range 2-30 years; <20 years (in 75%); M:F = 3:2*Path:*bone lesions arise within medullary canal (RES)*Histo:*proliferation of histiocytes + infiltrate by variable number of inflammatory cells (eosinophils, lymphocytes, neutrophils, plasma cells) ● eosinophilia in blood + CSFLocation:monostotic involvement in 50-75%; calvarium > mandible > large long bones of upper extremity > ribs > pelvis > vertebrae@Skull (50%)Site:diploic space of parietal bone (most commonly involved) + temporal bone (petrous ridge, mastoid)√ round / ovoid punched-out lesion with serrated + beveled edge*DDx:*venous lake, arachnoid granulation, parietal foramen, epidermoid cyst, hemangioma√ sharply marginated without sclerotic rim (DDx: epidermoid with bone sclerosis)√ sclerotic margin during healing phase (50%)√ "hole-within-hole" appearance = uneven involvement of inner + outer table√ "button sequestrum" = central bone density within lytic lesion√ soft-tissue mass overlying the lytic process in calvarium (often palpable)√ isodense homogeneously enhancing mass in hypothalamus / pituitary gland@Orbit√ benign focal mass ± infiltration of orbital bones@Mastoid process ● intractable otitis media with chronically draining ear (in temporal bone involvement)√ destructive lesion near mastoid antrum*DDx:*mastoiditis, cholesteatoma, metastasis*Cx:*extension to middle ear may destroy ossicles leading to deafness@Jaw ● gingival + contiguous soft-tissue swelling√ "floating" teeth, fracture@Axial skeleton (25%)√ "vertebra plana" = "coin on edge" = Calvé disease (6%) = collapse of vertebra (most commonly thoracic); preserved disk space; rare involvement of posterior elements; no kyphosis; most common cause of vertebra plana in children√ lytic lesion in supra-acetabular region@ Proximal long bones (15%) ● painful bone lesion + swellingSite:mostly diaphyseal, epiphyseal lesions are uncommon√ expansile lytic lesion with ill-defined / sclerotic edges√ endosteal scalloping, widening of medullary cavity√ cortical thinning, intracortical tunneling√ erosion of cortex + soft-tissue mass√ laminated periosteal reaction (frequent), may show interruptions√ may appear rapidly within 3 weeks√ lesions respect joint space + growth plate@ Lung involvement (20%) *Incidence:*0.05 to 0.5 / 100,000 annually*Age:*peak between 20 and 40 years√Strong association between smoking + primary pulmonary Langerhans cell histiocytosis!√ 3-10 mm nodules√ reticulonodular pattern with predilection for apices√ may develop into honeycomb lung√ recurrent pneumothoraces (25%)√ rib lesions with fractures (common)√ pleural effusion, hilar adenopathy (unusual)NUC: √ negative bone scans in 35% (radiographs more sensitive)√ bone lesions generally not Ga-67 avid√ Ga-67 may be helpful for detecting nonosseous lesions*Prognosis:*excellent with spontaneous resolution of bone lesions in 6-18 months

Notes:

LAURENCE-MOON-BIEDL SYNDROME

● retardation ● obesity ● hypogonadism√ craniosynostosis√ polysyndactyly

Notes:

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LEAD POISONING
=PLUMBISM*Path:*lead concentrates in metaphyses of growing bones (distal femur > both ends of tibia > distal radius) leading to failure of removal of calcified cartilaginous trabeculae in provisional zone ● loss of appetite, vomiting, constipation, abdominal cramps ● peripheral neuritis (adults), meningoencephalitis (children) ● anemia ● lead line at gums (adults)√ bands of increased density at metaphyses of tubular bones (only in growing bone)√ lead lines may persist√ clubbing if poisoning severe (anemia)√ bone-in-bone appearance*DDx:*(1)Healed rickets(2)Normal increased density in infants <3 years of age

Notes:

LEPROSY
=HANSEN DISEASE*Organism:*Mycobacterium leprae*Types:* (1)lepromatous: in cutis, mucous membranes, viscera(2)neural: enlarged indurated nodular nerve trunks; anesthesia, muscular atrophy, neurotrophic changes(3)mixed form Osseous changes in 15-54% of patients: SPECIFIC SIGNS Location:center of distal end of phalanges / eccentric√ ill-defined areas of decalcification, reticulated trabecular pattern, small rounded osteolytic lesions, cortical erosions√ joint spaces preserved√ healing phase: complete resolution / bone defect with sclerotic rim + endosteal thickening√ nasal spine absorption + destruction of maxilla, nasal bone, alveolar ridge√ enlarged nutrient foramina in clawlike hand√ erosive changes of ungual tufts NONSPECIFIC SIGNS √ soft-tissue swelling; calcification of nerves√ contractures / deep ulcerations√ neurotrophic joints (distal phalanges in hands, MTP in feet, Charcot joints in tarsus)

Notes:

LEUKEMIA OF BONE

A.CHILDHOODmost common malignancy of children *Histo:*acute lymphoblastic leukemia (in 75%) ● migratory para-articular arthralgias (25-50%) due to adjacent metaphyseal lesions (may be confused with acute rheumatic fever / rheumatoid arthritis) ● fever, elevated erythrocyte sedimentation rate ● hepatosplenomegaly, occasionally lymphadenopathy◊Peripheral blood smears may be negative in aleukemic form! Skeletal manifestations in 50-90%: (a)Diffuse osteopenia (most common pattern)√ diffuse demineralization of spine + long bones (= leukemic infiltration of bone marrow + catabolic protein / mineral metabolism)√ coarse trabeculation of spongiosa (due to destruction of finer trabeculae)√ multiple biconcave / partially collapsed vertebrae (14%)(b)"Leukemic lines" (40-53% in acute lymphoblastic leukemia):√ transverse radiolucent metaphyseal bands, uniform + regular across the width of metaphysis (= leukemic infiltration of bone marrow / osteoporosis at sites of rapid growth)Location:large joints (proximal tibia, distal femur, proximal humerus, distal radius + ulna)√ horizontal / curvilinear bands in vertebral bodies + edges of iliac crest√ dense metaphyseal lines after treatment(c)Focal destruction of flat / tubular bones:√ multiple small clearly defined ovoid / spheroid osteolytic lesions (destruction of spongiosa, later cortex) in 30-60%√ moth-eaten appearance, sutural widening, prominent convolutional markings of skull◊Lytic lesions distal to knee / elbow in children are suggestive of leukemia (rather than metastases)!(d)Isolated periostitis of long bones (infrequent):√ smooth / lamellated / sunburst pattern of periosteal reaction (cortical penetration by sheets of leukemic cells into subperiosteum) in 12-25%(e)Metaphyseal osteosclerosis + focal osteoblastic lesion (very rare) √ osteosclerotic lesions (late in disease due to reactive osteoblastic proliferation)√ mixed lesions (lytic + bone-forming) in 18%*Dx:*sternal marrow / peripheral blood smear*Cx:*proliferation of leukemic cells in marrow leads to extraskeletal hematopoiesis*DDx:*metastatic neuroblastoma, Langerhans cell histiocytosisB.ADULTHOODDeath usually occurs before skeletal abnormalities manifest √ osteoporosis√ solitary radiolucent foci (vertebral collapse)√ permeating radiolucent mottling (proximal humerus)

Notes:

LIPOBLASTOMA
=postnatal proliferation of mesenchymal cells with a spectrum of differentiation ranging from prelipoblasts (spindle cells) to mature adipocytes*Path:*immature adipose tissue separated by septa into multiple lobules *Histo:*uni- and multivacuolated lipoblasts interspersed between spindle / stellate mesenchymal cells; suspended in myxoid stroma*Age:*<3 years of age; M:F = 2:1Location:subcutaneous tissue of extremities, neck, trunk, perineum, retroperitoneum fatty tumor with enhancing soft-tissue component*DDx:*liposarcoma (extremely rare in children)

Notes:

LIPOMA OF BONE
=INTRAOSSEOUS LIPOMA*Incidence:*<1:1,000 primary bone tumors*Age:*any (4th-6th decade); M:F = 1:1*May be associated with:*hyperlipoproteinemia ● asymptomatic / localized bone painLocation:calcaneus, extremities (proximal femur > tibia, fibula, humerus), ilium, skull, mandible, maxilla, ribs, vertebrae, sacrum, coccyx, radiusSite:metaphysis√ expansile nonaggressive radiolucent lesion√ loculated / septated appearance (trabeculae)√ thin well-defined sclerotic border√ ± thinned cortex (NO cortical destruction)√ NO periosteal reaction√ may contain clump of calcification centrally (= dystrophic calcification from fat necrosis)◊VIRTUALLY DIAGNOSTIC:@Calcaneus√ in triangular region between major trabecular groups (LAT projection)√ calcified / ossified nidus@Proximal femur√ on / above intertrochanteric line√ marked ossification of margins of lesion◊Radiographic appearance similar to unicameral bone cyst (infarcted lipoma = unicameral bone cyst ?)*DDx:*fibrous dysplasia, simple bone cyst, posttraumatic cyst, giant cell tumor, desmoplastic fibroma, chondromyxoid fibroma, osteoblastoma

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Notes:

LIPOMA OF SOFT TISSUE

Most common mesenchymal tumor composed of mature adipose tissue *Histo:*mature fat cells (adipocytes) that are uniform in size + shape, occasionally have fibrous connective tissue as septations; fat unavailable for systemic metabolism ● stable size after initial period of discernible growth*Age:*5th-6th decade; M > FLocation: (a)superficial = subcutaneous lipoma (more common) in posterior trunk, neck, proximal extremities(b)deep lipoma in retroperitoneum, chest wall, deep soft tissue of hands + feet; multiple in 5-7% (up to several hundred tumors)√ mass of fat opacity / density / intensity identical to subcutaneous fat√ cortical thickening (with adjacent parosteal lipoma)CT: √ well-defined + homogeneous tumor with low attenuation coefficient (-65 to -120 HU)√ no enhancement following IV contrast materialMR: √ well-defined + homogeneous, often with septations√ signal intensity characteristics similar to subcutaneous fat: hyperintense on T1WI + moderately intense on T2WI√ differentiation from other lesions by fat suppression technique

Angiolipoma Benign Mesenchymoma Lipoma Arborescens Neural Fibrolipoma

Notes:

Angiolipoma =lesion composed of fat separated by small branching vessels*Age:*2nd + 3rd decade; 5% familial incidence ● tenderLocation:upper extremity, trunk√ signal characteristics of fat + mixed with varying numbers of large / small vessels√ mostly encapsulated lesion, may infiltrate

Notes:

Benign Mesenchymoma = long-standing lipoma with chondroid + osseous metaplasia Infiltrating Lipoma =INTRAMUSCULAR LIPOMA = relatively common benign lipomatous tumor extending between muscle fibers that become variably atrophic*Peak age:*5th-6th decade; M > FLocation:thigh (50%), shoulder, upper arm

Notes:

Lipoma Arborescens =DIFFUSE SYNOVIAL LIPOMA = lipoma-like lesion composed of hypertrophic synovial villi distended with fat, probably reactive process to chronic synovitisLocation:knee; monarticular*Frequently associated with:* degenerative joint disease, chronic rheumatoid arthritis, prior trauma

Notes:

Neural Fibrolipoma =FIBROLIPOMATOUS HAMARTOMA OF NERVE=rare tumorlike condition characterized by sausage-shaped / fusiform enlargement of a nerve by fibrofatty tissue*Age:*early adulthood before age 30 years / at birth*Histo:*infiltration of epineurium + perineurium by fibrofatty tissue with separation of nerve bundles ● soft slowly enlarging mass ● pain, tenderness, decreased sensation, paresthesiaLocation:volar aspect of hand, wrist, forearmSite:median n. (most frequently), ulnar n., radial n., brachial plexus;*May be associated with:* macrodactyly (in 2/3) = **macrodystrophia lipomatosa** √ may not be visible radiographicallyMR: √ longitudinally oriented, cylindrical, linear / serpiginous structures of signal void about 3 mm in diameter (= nerve fascicles with epi- and perineural fibrosis) separated by areas of fat signal intensity (= mature fat infiltrating the interfascicular connective tissue)US: √ "cablelike appearance" = alternating hyper- and hypoechoic bands on US*DDx:*cyst, ganglion, lipoma, traumatic neuroma, plexiform neurofibroma, vascular malformation

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Notes:

LIPOSARCOMA
Malignant tumor of mesenchymal origin with bulk of tumor tissue differentiating into adipose tissue *Incidence:*12-18% of all malignant soft-tissue tumors; 2nd most common soft-tissue sarcoma in adults (after malignant fibrous histiocytoma)*Age:*5th-6th decade*Histo:*(a)well-differentiated(b)myxoid in 40-50% (most common): proliferating fibroblasts, plexiform capillary pattern, myxoid matrix, fat amount <10%(c)round cell = poorly differentiated myxoid(d)pleomorphic ● usually painless mass (may be painful in 10-15%)Location:trunk (42%), lower extremity (41%), upper extremity (11%), head + neck (6%); particularly in thigh + retroperitoneumSpread:hematogenous to lung, visceral organs; myxoid liposarcoma shows tendency for serosal + pleural surfaces, subcutaneous tissue, bone√ nonspecific soft-tissue mass (frequently fat is radiologically not detectable)√ inhomogeneous mass with soft-tissue + fatty components√ enhancement after IV contrast material (contradistinction to lipoma)√ concomitant mass in retroperitoneum / thigh (in up to 10% of myxoid liposarcomas) as multicentric lesion / metastasis√ mass of near water density / hypoechoic / hypointense on T1WI + hyperintense on T2WI in myxoid liposarcoma (high content of myxoid cells)

Notes:

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LYME ARTHRITIS
*Agent:*spirochete Borrelia burgdorferi; transmitted by tick Ixodes dammini *Histo:*inflammatory synovial fluid, hypertrophic synovia with vascular proliferation + cellular infiltration ● history of erythema chronicum migrans ● endemic areas: Lyme, Connecticut, first recognized location; now also throughout United States, Europe, Australia ● recurrent attacks of arthralgias within days to 2 years after tick bite (80%)Location:mono- / oligoarthritis of large joints (especially knee)Ψ erosion of cartilage / bone (4%)*Rx:*antibiotics*DDx:*(1) Rheumatic fever (2) Rheumatoid arthritis(3) Gonococcal arthritis (4) Reiter syndrome

Notes:

LYMPHANGIOMA
=sequestered noncommunicating lymphoid tissue lined by lymphatic endothelium*Cause:*congenital obstruction of lymphatic drainage*Subtypes:* (1)Capillary lymphangioma (rare)Location:subcutaneous tissue(2)Cavernous lymphangiomaLocation:about the mouth + tongue(3)Cystic lymphangioma (most common)=cystic hygroma*Associated with:*hydrops fetalis, Turner syndromeLocation:head, neck (75%), axilla (20%), extension into mediastinum (3-10%) ● soft fluctuant mass Lymphangiomas are frequently a mixture of subtypes! *Age:*found at birth (50-65%); within first 2 years of life (90%)Location:soft tissue; bone (rare) √ multilocular cystic lesion with fibrous septations√ occasionally serpentine vascular channels√ opacification during lymphangiography / direct puncture√ clear / milky fluid on aspiration*DDx:* hemangioma (blood on aspiration)

Notes:

LYMPHOMA OF BONE
=RETICULUM CELL SARCOMA = HISTIOCYTIC LYMPHOMA = PRIMARY LYMPHOMA OF BONE (the generalized form of reticulum cell sarcoma is lymphoma); 2-6% of all primary malignant bone tumors in children*Incidence of bone marrow involvement:* 5-15% in Hodgkin disease; 25-40% in non-Hodgkin lymphoma ◊bone marrow involvement indicates progression of disease◊bone marrow imaging-guidance for biopsy!NUC: 40% sensitivity; 88% specificityMR: 65% sensitivity; 90% specificity*Histo:*sheets of reticulum cells, larger than those in Ewing sarcoma (DDx: myeloma, inflammation, osteosarcoma, eosinophilic granuloma)*Age:*any age; peak age in 3rd-5th decade; 50% <40 years; 35% <30 years; M:F = 2:1 ● striking contrast between size of lesion + patients well-beingLocation:lower femur, upper tibia (40% about knee), humerus, pelvis, scapula, ribs, vertebraSite:dia- / metaphysis√ cancellous bone erosion (earliest sign)√ mottled permeative pattern of separate coalescent areas√ late cortical destruction√ lamellated / sunburst periosteal response (less than in Ewing sarcoma)√ lytic / reactive new-bone formation√ associated soft-tissue mass without calcification√ synovitis of knee joint common*Cx:*pathologic fracture (most common among malignant bone tumors)*Prognosis:*50% 5-year survival*DDx:*(1)Osteosarcoma (less medullary extension, younger patients)(2)Ewing tumor (systemic symptoms, debility, younger patients)(3)Metastatic malignancy (multiple bones involved, more destructive)

Notes:

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MACRODYSTROPHIA LIPOMATOSA
=rare nonhereditary congenital form of localized gigantism = neural fibrolipoma with macrodactyly*Path:*striking increase in adipose tissue in a fine fibrous network involving periosteum, bone marrow, nerve sheath, muscle, subcutaneous tissue*May be associated with:*syn-, clino-, polydactyly ● painlessLocation:2nd or 3rd digit of hand / foot; unilateral;one / few adjacent digits may be involved in the distribution of the median / plantar nerves√ long + broad splayed phalanges with endosteal + periosteal bone deposition √ overgrowth of soft tissue, greatest at volar + distal aspects√ slanting of articular surfaces√ lucent areas of fat (DIAGNOSTIC)*Prognosis:*accelerated maturation possible; growth stops at puberty*DDx:*fibrolipomatous hamartoma associated with macrodystrophia lipomatosa (indistinguishable), Klippel-Trénaunay-Weber syndrome, lymphangiomatosis, hemangiomatosis, neurofibromatosis, chronic vascular stimulation, Proteus syndrome

Notes:

MARFAN SYNDROME
=ARACHNODACTYLY = autosomal dominant familial disorder of connective tissue with high penetrance but extremely variable expression, new mutations in 15%*Etiology:*fibrillin gene defect on chromosome 15 resulting in abnormal cross-linking of collagen fibers *Prevalence:*5:100,000; M:F = 1:1 A.MUSCULOSKELETAL MANIFESTATIONS ● tall thin stature with long limbs, arm span greater than height ● muscular hypoplasia + hypotonicity ● scarcity of subcutaneous fat (emaciated look)√ generalized osteopenia@Skull ● elongated face√ dolichocephaly√ prominent jaw√ high arched palate@Hand ● Steinberg sign = protrusion of thumb beyond the confines of the clenched fist (found in 1.1% of normal population) ● metacarpal index (averaging the 4 ratios of length of 2nd through 5th metacarpals divided by their respective middiaphyseal width) >8.8 (male) or 9.4 (female)√ arachnodactyly = elongation of phalanges + metacarpals√ flexion deformity of 5th finger@Foot√ pes planus√ clubfoot√ hallux valgus√ hammer toes√ disproportionate elongation of 1st digit of foot@Spine ● ratio of measurement between symphysis and floor + crown and floor >0.45√ pectus carinatum / excavatum (common)√ scoliosis / kyphoscoliosis (45-60%)√ increased incidence of Scheuermann disease and spondylosis√ dural ectasia√ increased interpedicular distance√ posterior scalloping√ presacral + lateral sacral meningoceles√ expansion of sacral spinal canal√ enlargement of sacral foramina√ winged scapulae@Joints ● ligamentous laxity + hypermobility + instability√ premature osteoarthritis√ patella alta√ genu recurvatum√ recurrent dislocations of patella, hip, clavicle, mandible√ slipped capital femoral epiphysis√ progressive protrusio acetabuli (50%), bilateral > unilateral, F > M B. OCULAR MANIFESTATIONS ● bilateral ectopia lentis, usually upward + outward (secondary to poor zonular attachments) ● glaucoma, macrophthalmia ● hypoplasia of iris + ciliary body ● contracted pupils (absence of dilator muscle) ● myopia, retinal detachment ● strabismus, ptosis ● blue sclera ● megalocornea = flat enlarged thickened corneaC.CARDIOVASCULAR MANIFESTATIONS (60-98%)affecting mitral valve, ascending aorta, pulmonary artery, splenic + mesenteric arteries (occasionally)◊ Cause of death in 93%! ● chest pain, palpitations, shortness of breath, fatigue ● mid-to-late systolic murmur + one / more clicks*Associated with*congenital heart defect (33%): incomplete coarctation, ASD @Aorta (cause of death in 55%)*Histo:*myxomatous degeneration of aortic annulus√ "tulip bulb aorta" = symmetrical dilatation of aortic sinuses of Valsalva slightly extending into ascending aorta (58%)√ annuloaortic ectasia = combination of aortic root dilatation + aortic regurgitation√ fusiform aneurysm of ascending aorta, rarely beyond innominate artery (due to cystic medial necrosis)√ aortic wall calcification rare*Cx:*(1)Aortic regurgitation (in 81% if root diameter >5 cm; in 100% if root diameter >6 cm (2) Aortic dissection (3) Aortic rupture (secondary to progressive aortic root dilatation)@Mitral valve*Histo:*myxomatous degeneration of valve leads to redundancy + laxness ● mid-to-late systolic murmur + one / more clicks√ "floppy valve syndrome" (95%) = redundant chordae tendineae with mitral valve prolapse + regurgitation*Cx:*(1) Mitral regurgitation (2) Rupture of chordae tendineae (rare)@Coarctation (mostly not severe)@Pulmonary artery aneurysm + dilatation of pulmonary arterial root (43%)@Cor pulmonale (secondary to chest deformity) D.PULMONARY MANIFESTATIONS√ cystic lung disease√ recurrent spontaneous pneumothoraces E.ABDOMINAL MANIFESTATION√ recurrent biliary obstruction *DDx:*(1)Homocystinuria (osteoporosis)(2)Ehlers-Danlos syndrome(3)Congenital contractural arachnodactyly (ear deformities, NO ocular / cardiac abnormalities)(4)Type III MEN (medullary thyroid carcinoma, mucosal neuromas, pheochromocytoma, marfanoid habitus)

Notes:

MASSIVE OSTEOLYSIS
=GORHAM DISEASE = "VANISHING BONE" SYNDROME = PHANTOM BONE = HEMANGIOMA OF BONE=infrequent disorder of unknown etiology with unpredictable course + progression*Incidence:*>100 cases described*Histo:*massive proliferation of hemangiomatous / lymphangiomatous tissue with large sinusoid spaces + fibrosis*Age:*children + adults <40 years*Associated with:*soft-tissue hemangiomas without calcifications ● frequently history of severe trauma (50%) ● little / no painLocation:any bone; most commonly major long bones (humerus, shoulder, mandible), innominate bone, spine, thorax, short tubular bones of hand + feet (unusual) √ progressive relentless destruction of bone√ lack of reaction (no periosteal reaction, no repair)√ advancing edge of destruction not sharply delineated√ tapering margins of bone ends at sites of osteolysis with conelike spicule of bone (early changes)√ no respect for joints√ may destroy all bones in a particular area

Notes:

MASTOCYTOSIS
=URTICARIA PIGMENTOSA=mast cell accumulation in multiple organs*Age:*<6 months (50%)*Associated with:* myeloproliferative disorders, acute non-lymphatic leukemia, malignant lymphoma, mast cell leukemia ● hyperpigmented skin lesions exhibiting "wheal and flare" phenomenon when disturbed ● pruritus, flushing ● pancytopenia (chronic neutropenia) @Skeletal involvement (70%) ● bone and joint pain√ osteoporosis (due to release of heparin + prostaglandin by mast cells activating osteoclasts)√ scattered well-defined sclerotic foci with focal / diffuse involvement (due to release of histamine by mast cells promoting osteoblastic activity); often alternating with areas of bone rarefactionPredilected sites:skull, spine, ribs, pelvis, humerus, femur@Abdomen√ hepatosplenomegaly√ lymphadenopathy: retroperitoneal, periportal, mesenteric√ thickening of omentum, + mesentery√ ascites@GI tract ● nausea, vomiting, diarrhea√ thickened nodular irregular folds in small bowel (due to infiltration by mast cells, lymphocytes, plasma cells)√ duodenal ulcers (due to release of histamine increasing gastric acid secretion)

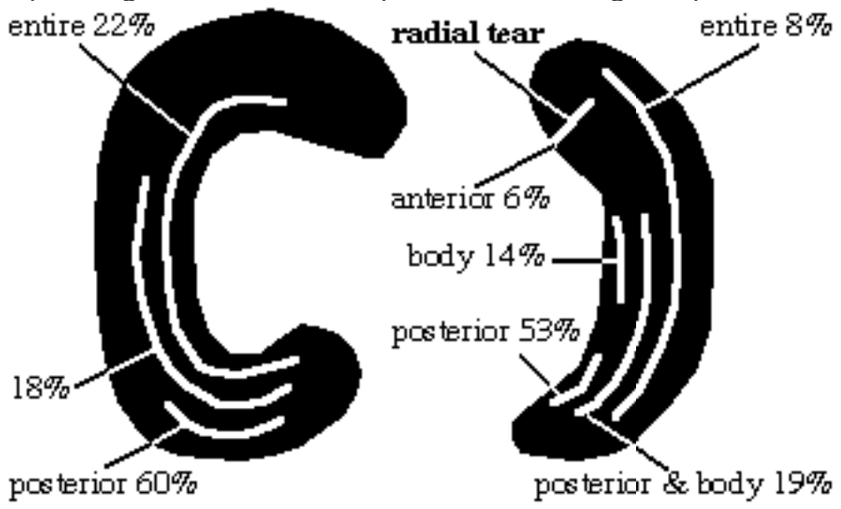
Notes:

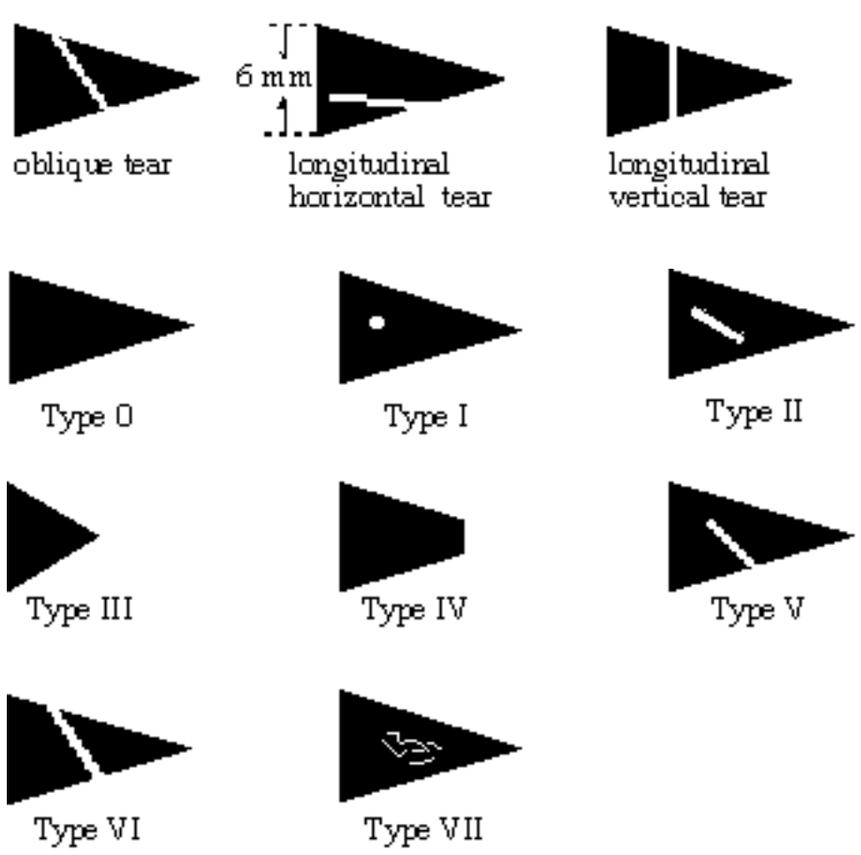
MELORHEOSTOSIS
Nonhereditary disease of unknown etiology; often incidental finding *Age:*slow chronic course in adults; rapid progression in children*Associated with:* osteopoikilosis, osteopathia striata, tumors / malformations of blood vessels (hemangioma, vascular nevi, glomus tumor, AVM, aneurysm, lymphedema, lymphangiectasia) ● severe pain + limited joint motion (bone may encroach on nerves, blood vessels, or joints) ● thickening + fibrosis of overlying skin (resembling scleroderma) ● muscle atrophy (frequent)Location:diaphysis, usually monomelic with at least two bones involved in dermatomal distribution (follows spinal sensory nerve sclerotomes); entire cortex / limited to one side of cortex; more common in lower limb; skull, spine, ribs rarely involved√ "candle wax dripping" = continuous / interrupted streaks / blotches of sclerosis along tubular bone beginning at proximal end extending distally with slow progression√ may cross joint with joint fusion√ small opacities in scapula + hemipelvis (similar to osteopoikilosis)√ discrepant limb length√ flexion contractures of hip + knee√ genu valgum / varus√ dislocated patella√ ossified soft-tissue masses (27%)*DDx:*(1)Osteopoikilosis (generalized)(2)Fibrous dysplasia (normal bone structure not lost, not as dense)(3)Engelmann disease(4)Hyperostosis of neurofibromatosis, tuberous sclerosis, hemangiomas(5)Osteoarthropathy

Notes:

MENISCAL TEAR

Type of tear: A.LONGITUDINAL TEAR1.Horizontal cleavage tear*Cause:*usually degenerative*Associated with:*meniscal cystSite:primarily involving the central horizontal plane of meniscus beginning at inner margin2.Bucket handle tear*Cause:* traumaticSite:usually in medial rarely in lateral meniscus√ longitudinal vertical tear with unstable displaced inner fragment3.Peripheral tear*Cause:* traumatic√ vertical tear in peripheral third of meniscusB.OBLIQUE TEARSite:common in midportion of medial meniscus√ both horizontal and vertical components√ commonly extending to inferior surface of meniscus1.Parrot beak tear*Cause:*usually degenerativeSite:in body of lateral meniscus near the junction of body + posterior horn√ fraying of free edge2.Flap tear = oblique + incomplete tear*Cause:*traumatic, at times degenerativeC.TRANSVERSE TEAR = RADIAL TEARSite:posterior + midportion of lateral meniscus√ peripheral displacement of meniscus√ "absent" / gray meniscus posteriorly*Cx:*lack of resistance to hoop stressesD.MENISCOCAPSULAR SEPARATION=tearing of peripheral attachments of meniscus√ linear regions of fluid separating meniscus from capsule√ uncovering of a portion of tibial plateau owing to inward movement of separated meniscus





MR Classification

GradeTypeMR findingPPV for tear 00normal meniscus1%1Iglobular / punctate intrameniscal signal2%2IIlinear signal not extending to surface5%IIIshort tapered apex of meniscus23%IVtruncated / blunted apex of meniscus71%3Vsignal extending to only one surface85%3VIsignal extending to both surfaces95%3VIIcomminuted reticulated signal pattern82%

◊Diagnosis of tear hinges on surface involvement!◊Intrameniscal signal may be a sign of persistent vascularity in children + young adults (type VII)!◊Truncation artifact + magic angle artifact may cause increased intrameniscal signal!◊Grade 3 signal identified only on a single image is unlikely to be confirmed as a tear at surgery! Site of injury: (a)medial meniscus in 45%: no isolated tears of body / anterior horn(b)lateral meniscus in 22%: posterior horn involved in 80% of all lateral meniscal tears(c)both menisci involved in 33% *Associated with:*ligamentous injury ● asymptomatic in up to 20% of older individuals √ signal extending to articular surface (type V + VI)√ notch sign = linear signal intensity becoming wider as it extends toward meniscal surface indicates type V finding (tapering toward surface = type II finding)√ meniscal cyst = implies presence of meniscal tear*DDx:*synovial cyst, tendon sheath fluid, fluid within normal synovial recess, fluid collection remote from meniscus *MR sensitivity, specificity, and accuracy:* Tear ofSensitivitySpecificityAccuracymedial meniscus95%88%59-92%lateral meniscus81%96%87-92%anterior cruciate lig.91-96%posterior cruciate lig.up to 99%◊MR has a high negative predictive value!◊60-97% accuracy for arthrography◊84-99% accuracy for arthroscopy (poor at posterior horn of medial meniscus)

Interpretative errors (12% for experienced radiologist): Lateral meniscus:5.0% FN (middle + posterior horn)1.5% FP (posterior horn)Medial meniscus:2.5% FN (posterior horn)2.5% FP (posterior horn) PITFALLS: A.Normal variants simulating tears:1.Superior recess on posterior horn of medial meniscus2.Popliteal hiatus√ hiatus of popliteal tendon separates lateral meniscus from joint capsule◊Seen above posterior aspect of lateral meniscus on most superficial sagittal slice!◊Tendon moves behind + inferior to meniscus on adjacent deeper sections!3.Transverse ligamentCourse: connects anterior horns of both menisci √ overrides superior aspect of menisci before completely fusing to menisci◊Trace the cross section of the transverse ligament through the infrapatellar fat pad on more central images!4.Meniscofemoral ligamentsOrigin:superior + medial aspect of posterior horn of lateral meniscusAttachment:medial femoral condyle√ demonstrated in 1/3 of cases on SAG images(a)Wrisberg ligament√ posterior to posterior cruciate ligament(b)Humphry ligament√ anterior to posterior cruciate ligament◊Finding usually limited to single most medial image!5.Soft tissue between capsule + medial meniscusB.Healed meniscus√ persistent grade 3 signal at least up to 6 months√ S/P meniscectomy (false-positive type IV finding)C.Degenerative changes√ grade 1 signal = globular increase in intensity√ grade 2 signal = linear signal not extending to articular surfaceD.Discoid meniscus=abnormally shaped enlarged discus-like meniscus*Prevalence:*in 1.5-15.5%*Age:*children, adolescentsSide:lateral >> medial meniscus√ centrally displaced fragment with meniscus apparently of normal size (coronal images)

Notes:

MESOMELIC DWARFISM

=heritable bone dysplasia with shortening of intermediate segments (radius + ulna or tibia + fibula)A.**Langer type**autosomal recessive ● mental impairment√ mesomelic shortening of limbs√ hypoplasia of ulna + fibula√ hypoplasia of mandible with short condylesB.**Nievergelt type**autosomal dominant√ severe mesomelic shortening of lower limbs√ marked thickening of tibia + fibula in central portion√ clubfoot (frequent)C.Reinhardt type:autosomal dominantD.Robinow type:autosomal dominantE.Werner type:autosomal dominantF.**Lamy-Bienenfeld type**autosomal dominant ● ligamentous laxity√ shortening of radius + ulna + tibia√ absent fibula√ normal femur + humerus √ shortening of all long bones at birth, most marked in tibia + radius√ modeling deformity with widening of diaphysis√ mild to moderate bowing√ hypoplasia of fibula with absent lateral malleolus√ short + thick ulna with hypoplastic distal end√ Madelung deformity of wrist√ hypoplasia of a vertebral body may be present

Notes:

METAPHYSEAL CHONDRODYSPLASIA

=severe short-limbed dwarfism√ metaphyseal flaring (Erlenmeyer flask deformity) extending into diaphysisA.**Schmid type** (most common)autosomal dominant ● waddling gateDistribution:more marked in lower limbs; mild involvement of hands + wrists√ shortened bowed long bones√ widened epiphyseal growth plates√ irregular widened cupped metaphyses√ coxa vara√ genu varum*DDx:*vitamin D-refractory ricketsB.**McKusick type**autosomal recessive (eg, in Amish) ● sparse brittle hair, deficient pigmentation ● normal intelligence√ shortening of long bones with normal width√ cupped + widened metaphyses with lucent defects√ short middle phalanges + narrow distal phalanges becoming triangular and bullet-shaped (more frequent in hands than feet)√ widened costochondral junctions + cystic lucenciesC.**Jansen type** (less common)sporadic occurrence with wide spectrum ● intelligence normal / retarded ● serum calcium levels often elevatedDistribution:symmetrical involvement of all long + short tubular bones√ widened epiphyseal plates√ expanded irregular + fragmented metaphyses (unossified cartilage extending into diaphyses)*DDx:*ricketsD.**Pyle disease** = Metaphyseal dysplasia ● often tall ● often asymptomaticDistribution:major long bones, tubular bones of hands, medial end of clavicle, sternal end of ribs, innominate bone√ splaying of proximal + distal ends of long bones with thinned cortex√ relative constriction of central portion of shafts√ craniofacial hyperostosis√ genu valgum

Notes:

METASTASES TO BONE
◊15-100 times more common than primary skeletalneoplasms! *Frequency:*
if primary knownif primary unknown
breast35%prostate25%prostate30%lymphoma15%lung10%breast10%kidney 5%lung10%uterus2%thyroid 2%stomach2%colon1%others13%

METASTASES OF PRIMARY BONE TUMORS 1.Osteosarcoma: 2% with distant metastases, adjuvant therapy has changed the natural history of the disease in that bone metastases occur in 10% of osteosarcomas without metastases to the lung2.Ewing sarcoma: 13% with distant metastases
SOLITARY BONE LESION ◊of all causes only 7% due to metastasis◊in patients with known malignancy due to metastasis (55%), due to trauma (25%), due to infection (10%)Location:axial skeleton (64-68%), ribs (45%), extremities (24%), skull (12%) *mnemonic:*"**S**everal **K**inds **O**f **H**orribly **N**asty **T**umors **L**eap **P**romptly **T**o **B**one"**S**arcoma, **S**quamous cell carcinoma **K**idney tumor **O**varian cancer **H**odgkin disease **N**euroblastoma **T**esticular cancer **L**ung cancer **P**rostate cancer **T**hyroid cancer **B**reast cancer Breast cancer:extensive osteolytic lesions; involvement of entire skeleton; pathologic fractures commonThyroid / kidney:often solitary; rapid progression with bone expansion (bubbly); frequently associated with soft-tissue mass (distinctive)Rectum / colon:may resemble osteosarcoma with sunburst pattern + osteoblastic reactionHodgkin tumor:upper lumbar + lower thoracic spine, pelvis, ribs; osteolytic / occasionally osteoblastic lesionsNeuroblastoma:extensive destruction, resembles leukemia (metaphyseal band of rarefaction), mottled skull destruction + increased intracranial pressure, perpendicular spicules of boneEwing tumor:extensive osteolytic / osteoblastic reaction *Mode of spread:*through bloodstream / lymphatics / direct extensionLocation:predilection for marrow-containing skeleton (skull, spine, ribs, pelvis, humeri, femora)√ single / multiple lesions of variable size√ usually nonexpansile√ joint spaces + intervertebral spaces preserved (cartilage resistant to invasion)

Osteolytic Bone Metastases Osteoblastic Bone Metastases Mixed Bone Metastases Expansile / Bubbly Bone Metastases Permeative Bone Metastases Bone Metastases With "Sunburst" Periosteal Reaction (infrequent) Bone Metastases With Soft-tissue Mass Calcifying Bone Metastases Skeletal Metastases In Children Skeletal metastases in adult Role Of Bone Scintigraphy In Bone Metastases Role Of Magnetic Resonance Imaging

Notes:

Osteolytic Bone Metastases *Most common cause:* neuroblastoma (in childhood); lung cancer (in adult male); breast cancer (in adult female), thyroid cancer; kidney; colon √ may begin in spongy bone (associated with soft tissue mass in ribs) √ vertebral pedicles often involved (not in multiple myeloma)

Notes:

Osteoblastic Bone Metastases = evidence of slow-growing neoplasm Primary:prostate, breast, lymphoma, malignant carcinoid, medulloblastoma, mucinous adenocarcinoma of GI tract, TCC of bladder, pancreas, neuroblastoma*Most common cause:* prostate cancer (in adult male); breast cancer (in adult female) *mnemonic:* "5 **B**ees **L**ick **P**ollen"**B**rain (medulloblastoma) **B**ronchus **B**reast **B**owel (especially carcinoid) **B**ladder **L**ymphoma **P**rostate √ frequent in vertebrae + pelvis√ may be indistinguishable from Paget disease

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Notes:

Calcifying Bone Metastases *mnemonic:* "BOTTOM" **B**reast **O**steosarcoma **T**esticular **T**hyroid **O**vary **M**ucinous adenocarcinoma of GI tract

Notes:

Skeletal Metastases In Children 1.Neuroblastoma (most often)2.Retinoblastoma3.Embryonal rhabdomyosarcoma4.Hepatoma5.Ewing tumor

Notes:

Skeletal metastases in adult *mnemonic:* "**C**ommon **B**one **L**esions **C**an **K**ill **T**he **P**atient"**C**olon **B**reast **L**ung **C**arcinoid **K**idney **T**hyroid **P**rostate

Notes:

Role Of Bone Scintigraphy In Bone Metastases *Pathophysiology:*accumulation of tracer at sites of reactive bone formationFalse-negative scan:very aggressive metastasesFalse-positive scan:degeneration, healing fractures, metabolic disordersBaseline bone scan: (a)high sensitivity for many metastatic tumors to bone (particularly carcinoma of breast, lung, prostate); 5% of metastases have normal scan; 5-40% occur in appendicular skeleton(b)substantially less sensitive than radiographs in infiltrative marrow lesions (multiple myeloma, neuroblastoma, histiocytosis)(c)screening of asymptomatic patients-useful in: prostate cancer, breast cancer-not useful in: non-small-cell bronchogenic carcinoma, gynecologic malignancy, head and neck cancer√ multiple asymmetric areas of increased uptake√ axial > appendicular skeleton (dependent on distribution of bone marrow); vertebrae, ribs, pelvis involved in 80%√ superscan in diffuse bony metastasesFollow-up bone scan: √ stable scan = suggestive of relatively good prognosis√ increased activity in:(a)enlargement of bone lesions / appearance of new lesions indicate progression of the disease(b)"healing flare" phenomenon (in 20-61%)= transient increase in lesion activity secondary to healing under antineoplastic treatment concomitant with increased sclerosis, detected at 3.2 ± 1.4 months after initiation of hormonal / chemotherapy, of no additional favorable prognostic value(c)avascular necrosis particularly in hips, knees, shoulders caused by steroid therapy(d)osteoradionecrosis / radiation-induced osteosarcoma√ decreased activity in:(a)predominately osteolytic destruction(b)metastases under radiotherapy; as early as 2-4 months with minimum of 2000 rads ROLE OF BONE SCAN IN BREAST CANCER Routine preoperative bone scan not justified: StageI:unsuspected metastases in 2%, mostly single lesionStageII:unsuspected metastases in 6%StageIII:unsuspected metastases in 14% Follow-up bone scan: At 12 months no new cases; at 28 months in 5% new metastases; at 30 months in 29% new metastases Conversion from normal:StageI:in 7%StageII:in 25%StageIII:in 58%☝With axillary lymph node involvement conversion rate 2.5 x that of those without☝Serial follow-up examinations are important to assess therapeutic efficacy + prognosis! ROLE OF BONE SCAN IN PROSTATE CANCER Stage B: 5% with skeletal metastasesStage C:10% with skeletal metastasesStage D:20% with skeletal metastasesTest sensitivities for detection of osseous metastases: (a)Scintigraphy 1.0(b)Radiographic survey 0.68(c)Alkaline phosphatase 0.5(d)Acid phosphatase 0.5*DDx*:pulmonary metastasis (SPECT helpful in distinguishing nonosseous lung from overlying rib uptake)

Notes:

Role Of Magnetic Resonance Imaging ideal for bone marrow imaging due to high contrast between bone marrow fat + water-containing metastatic deposits (1)Focal lytic lesion:√ hypointense on T1WI + hyperintense on T2WI(2)Focal sclerotic lesion:√ hypointense on T1WI + T2WI(3)Diffuse inhomogeneous lesions:√ inhomogeneously hypointense on T1WI + hyperintense on T2WI(4)Diffuse homogeneous lesions:√ homogeneously hypointense on T1WI + hyperintense on T2WI

Notes:

METATROPHIC DYSPLASIA
=HYPERPLASTIC ACHONDROPLASIA= METATROPHIC DWARFISM metatrophic = "changeable" (change in proportions of trunk to limbs over time secondary to developing kyphoscoliosis in childhood) ● longitudinal double skin fold overlying coccyx√ long bones short with dumbbell-like / trumpet-shaped configuration (exaggerated metaphyseal flaring)√ "hourglass" phalanges (short with widened ends)√ wide separation of major joint spaces (thick articular cartilage)√ delayed ossification of flat irregular epiphyses @Chest√ cylindrical narrowed elongated thorax√ short + wide ribs√ pectus carinatum@Vertebrae√ odontoid hypoplasia with atlantoaxial instability√ progressive kyphoscoliosis√ platyspondyly + very wide intervertebral spaces√ wedge- / keel-shaped vertebral bodies@Pelvis√ coccygeal appendage similar to a tail (rare but CHARACTERISTIC)√ short squared iliac bones + irregular acetabula√ narrowed greater sciatic notch*Prognosis:*compatible with life, increased disability from kyphoscoliosis*DDx:*achondroplasia, mucopolysaccharidoses

Notes:

MUCOPOLYSACCHARIDOSES
=lysosomal storage disorder from deficiency of specific lysosomal enzymes involved in degradation ofmucopolysaccharides

Mucopolysaccharidoses

Type	*Eponym*	*Inheritance*	*Enzyme Deficiency*	*Urinary Glycosaminoglycan*	*Neurologic Signs*
I-H	Hurler	autosomal recessive	alpha-L-iduronidase	dermatan sulfate	marked
II	Hunter	X-linked recessive	iduronate sulfatase	dermatan / heparan sulfate	mild to moderate
III	Sanfilippo	autosomal recessive		heparan sulfate	mental deterioration
	A		heparan sulfate sulfatase		
	B		N-acetyl-alpha-D-glucosaminidase		
	C		alpha-glucosamine-N-acetyl-transferase		
	D		N-acetylglucosamine-6-sulfate sulfatase		
IV	Morquio A–D	autosomal recessive	N-acetylgalactosamine-6-sulfate sulfatase beta-galactosidase	keratan sulfate	none
I-S (V)	Scheie	autosomal recessive	alpha-L-iduronidase	heparan sulfate	none
VI	Maroteaux-Lamy	autosomal recessive	arylsulfatase B	dermatan sulfate	none
VII	Sly	autosomal recessive	beta-glucuronidase	dermatan sulfate heparan sulfate	variable

Type I= HurlerTypeV= ScheieTypeII= HunterTypeVI= Maroteaux-LamyTypeIII= SanfilippoTypeVII= SlyTypeIV= Morquio◊All autosomal recessive except for Hunter (X-linked)!*Associated with:*valvular heart disease ● corneal clouding ● retardation (prominent in types I, II, III, VII) ● skeletal involvement dominates in types IV and VI √ scaphocephaly, macrocephaly; thick calvarium; hypertelorism√ platyspondyly with kyphosis + dwarfism√ irregularity at anterior aspect of vertebral bodies√ atlantoaxial subluxation (laxity of transverse ligament / hypoplasia or absence of odontoid)√ limb contractures√ broad hands√ hepatosplenomegaly@Brain√ brain atrophy√ varying degree of hydrocephalus√ multiple white matter changes within cerebral hemispheres (diffuse hypodense areas, prolongation of T1 + T2)*Cx:*cord compression at atlantoaxial joint (types IV + VI)*Dx:*combination of clinical features, radiographic abnormalities correlated with genetic + biochemical studies*Prenatal Dx:*occasionally successful analysis of fibroblasts cultured from amniotic fluid

Hurler Syndrome Morquio Syndrome

Notes:

Hurler Syndrome =GARGOYLISM = PFAUNDLER-HURLER DISEASE=MPS I-H; autosomal recessive disease*Cause:*homozygous for MPS III gene with excess chondroitin sulfate B due to deficient X-L iduronidase (= Hurler corrective factor)*Incidence:*1:10,000 births*Age:*usually appears >1st year ● dwarfism ● progressive mental deterioration after 1-3 years ● large head; sunken bridge of nose; hypertelorism ● early corneal clouding progressing to blindness ● "gargoyle" features = everted lips + protruding tongue ● teeth widely separated + poorly formed ● progressive narrowing of nasopharyngeal airway ● protuberant abdomen (secondary to dorsolumbar kyphosis + hepatosplenomegaly) ● urinary excretion of chondroitin sulfate B (dermatan sulfate) + heparan sulfate ● Reilly bodies (metachromic granules) in white blood cells or bone marrow cells @Skull (earliest changes >6 months of age)√ frontal bossing√ calvarial thickening√ premature fusion of sagittal + lambdoid sutures√ deepening of optic chiasm√ enlarged J-shaped sella (undermining of anterior clinoid process)√ small facial bones√ wide mandibular angle + underdevelopment of condyles√ communicating hydrocephalus@Extremities√ thick periosteal cloaking of long-bone diaphyses (early changes)√ swelling of diaphyses + tapering of either end: distal humerus, radius, ulna, proximal ends of metacarpals, ribs√ enlargement of shaft due to dilatation of medullary canal with cortical thinning √ deossification√ flexion deformities of knees + hips√ trident hands; clawing (occasionally)√ delayed maturation of irregular carpal bones@Spine√ thoracolumbar kyphosis with lumbar gibbus√ oval centra with normal / increased height + anterior beak at T12/L1/L2√ long slender pedicles√ spatulate rib configuration@Pelvis√ widely flared iliac wings√ constriction of iliac bones√ coxa valga*Prognosis:*death by age 10-15 years

Notes:

Morquio Syndrome =KERATOSULFATURIA = MPS IV;autosomal recessive; excess keratosulfate*Incidence:*1:40,000 births*Etiology:*N-acetylgalactosamine-6-sulfatase deficiency resulting in defective degradation of keratin sulfate (mainly in cartilage, nucleus pulposus, cornea)*Age:*normal at birth; skeletal changes manifest within first 18 months ● excessive urinary excretion of keratan sulfate ● normal intelligence ● weakness + hypotonia ● dwarfism with short trunk (<4 feet tall) ● head thrust forward + sunken between high shoulders ● normal intelligence ● corneal opacities evident around age 10 ● progressive deafness ● short nose, wide mouth, spacing between teeth ● semicrouching stance + knock knees from flexion deformities of knees + hips @Skull√ mild dolichocephaly√ hypertelorism√ poor mastoid air cell development√ short nose + depression of bridge of nose√ prominent maxilla@Chest√ increased AP diameter + marked pectus carinatum√ slight lordosis with wide short ribs√ bulbous costochondral junctions√ failure of fusion of sternal segments@Spine√ hypoplasia / absence of odontoid process of C2√ C1-C2 instability with anterior subluxation√ thick C2-body with narrowing of vertebral canal√ atlas close to occiput / posterior arch of C1 within foramen magnum√ platyspondyly = universal vertebra plana esp. affecting lumbar spine (DDx: normal height in Hurler syndrome)√ ovoid vertebral bodies with central anterior beak / tongue at lower thoracic / upper lumbar vertebrae√ mild gibbus at thoracolumbar transition = low dorsal kyphosis√ exaggerated lumbar lordosis√ widened intervertebral disk spaces@Pelvis√ "goblet-shaped" / "wineglass" pelvis = constricted iliac bodies + elongated pelvic inlet + flared iliac wings√ oblique hypoplastic acetabular roofs@Femur√ initially well-formed femoral head epiphysis, involution + fragmentation by age 3-6 years√ lateral subluxation of femoral heads; later hip dislocation√ wide femoral neck + coxa valga deformity@Tibia√ delayed ossification of lateral proximal tibial epiphysis√ sloping of superior margin of tibia plateau laterally + severe genu valgum@Hand & foot√ short bones of forearm with widening of proximal ends√ delayed appearance + irregularity of carpal centers√ small irregular carpal bones√ proximally pointed short metacarpals 2-5√ enlarged joints; hand + foot deformities (flat feet)√ ulnar deviation of hand *Cx:*cervical myelopathy (traumatic quadriplegia / leg pains / subtle neurologic abnormality) most common cause of death secondary to C2 abnormality; frequent respiratory infections (from respiratory paralysis)*Rx:*early fusion of C1-C2*Prognosis:*may live to adulthood*DDx:*(1)Hurler syndrome (normal / increased vertebral height; vertebral beak inferior)(2)Spondyloepiphyseal dysplasia (autosomal dominant, present at birth, absent flared ilia / deficient acetabular ossification, small acetabular angle, deficient ossification of pubic bones, varus deformity of femoral neck, minimal involvement of hand + foot, myopia)

Notes:

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MULTIPLE EPIPHYSEAL DYSPLASIA

=FAIRBANK DISEASE = ? tarda form of chondrodystrophia calcificans congenita√ mild limb shortening√ irregular mottled calcifications of epiphyses (in childhood + adolescence)√ epiphyseal irregularities + premature degenerative joint disease, especially of hips (in adulthood)√ short phalanges*DDx:* Legg-Perthes disease, hypothyroidism

Notes:

MULTIPLE MYELOMA
Most common primary malignant neoplasm in adults *Histo:*normal / pleomorphic plasma cells (not pathognomonic), may be mistaken for lymphocytes (lymphosarcoma, reticulum cell sarcoma, Ewing tumor, neuroblastoma)(a)diffuse infiltration: myeloma cells intimately admixed with hematopoietic cells(b)tumor nodules: displacement of hematopoietic cells by masses entirely composed of myeloma cells*Age:*usually 5th- 8th decade; 98% >40 years; rare < age 30; M:F = 2:1(a)DISSEMINATED FORM: >40 years of age (98%); M:F = 3:2(b)SOLITARY FORM: mean age 50 years ● bone pain (68%) ● normochromic normocytic anemia (62%) ● RBC rouleau formation ● renal insufficiency (55%) ● hypercalcemia (30-50%) ● proteinuria (88%) ● Bence-Jones proteinuria (50%) ● increased globulin production (monoclonal gammopathy)Location: A.DISSEMINATED FORM:scattered; axial skeleton predominant site; vertebrae (50%) > ribs > skull > pelvis > long bones (distribution correlates with normal sites of red marrow)B.SOLITARY FORM:vertebrae > pelvis > skull > sternum > ribsC.SPINAL PLASMA CELL MYELOMA√ sparing of posterior elements (no red marrow) (DDx: metastatic disease)√ paraspinal soft-tissue mass with extradural extension√ scalloping of anterior margin of vertebral bodies (osseous pressure from adjacent enlarged lymph nodes)√ generalized osteoporosis with accentuation of trabecular pattern, especially in spine (early)√ punched out appearance of widespread osteolytic areas (skull, long bones) with endosteal scalloping and uniform size√ diffuse osteolysis (pelvis, sacrum)√ expansile osteolytic lesions (ballooning) in ribs, pelvis, long bones√ soft-tissue mass adjacent to bone destruction(= extrapleural + paraspinal mass adjacent to ribs / vertebral column)√ periosteal new-bone formation exceedingly rare√ involvement of mandible (rarely affected by metastatic disease)√ sclerosis may occur after chemotherapy, radiotherapy, fluoride administration√ sclerotic form of multiple myeloma (1-3%)(a)solitary sclerotic lesion: frequently in spine(b)diffuse sclerosis*associated with* **POEMS syndrome**:**P**olyneuropathy **O**rganomegaly **E**ndocrine abnormalities **M**-protein **S**kin changes MR (recognition dependent on knowledge of normal range of bone marrow appearance for age): √ hypointense focal areas on T1WI (25%)√ hyperintense focal areas on T2WI (53%)√ absence of fatty infiltration (nonspecific) SENSITIVITY OF BONE SCANS VS. RADIOGRAPHS Radiographs:in 90% of patients and 80% of sitesBone scan:in 75% of patients and 24-54% of sitesGallium scan:in 55% of patients and 40% of sites◊30% of lesions only detected on radiographs◊10% of lesions only detected on bone scans *Cx:*(1)renal involvement frequent(2)predilection for recurrent pneumonias (leukopenia)(3)secondary amyloidosis in 6-15% (4)pathologic fractures occur often *Prognosis:*20% 5-year survival; death from renal insufficiency, bacterial infection, thromboembolism*DDx:* - with osteopenia:(1) Postmenopausal osteoporosis(2) Hyperparathyroidism- with lytic lesion:(1) Metastatic disease(2) Amyloidosis(3) Myeloid metaplasia- with sclerotic lesion:(1) Osteopoikilosis(2) Lymphoma(3) Osteoblastic metastasis(4) Mastocytosis(5) Myelosclerosis(6) Fluorosis(7) Lymphoma(8) Renal osteodystrophy **Myelomatosis** √ generalized deossification without discrete tumors√ vertebral flattening

Notes:

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MYELOPROLIFERATIVE DISORDERS
=autonomous clonal disorder initiated by an acquired pluripotential hematopoietic stem cell*Types:* 1.Polycythemia vera2.Chronic granulomatous leukemia = chronic myelogenous leukemia3.Essential idiopathic thrombocytopenia4.Agnogenic myeloid metaplasia (= primary myelofibrosis + extramedullary hematopoiesis in liver + spleen) *Pathophysiology:* -self-perpetuating intra- and extramedullary hematopoietic cell proliferation without stimulus-trilinear panmyelosis (RBCs, WBCs, platelets)-myelofibrosis with progression to myelosclerosis-myeloid metaplasia = extramedullary hematopoiesis (normocytic anemia, leukoerythroblastic anemia, reticulocytosis, low platelet count, normal / reduced WBC count)

Notes:

MYELOSCLEROSIS
=AGNOGENIC MYELOID METAPLASIA=MYELOPROLIFERATIVE SYNDROME=PSEUDOLEUKEMIA= hematologic disorder of unknown etiology with gradual replacement of bone marrow elements by fibrosis Characterized by (1)extramedullary hematopoiesis(2)progressive splenomegaly(3)anemia(4)variable changes in number of granulocytes + platelets; often predated by polycythemia vera*Age:*usually >50 years*Path:*fibrous / bony replacement of bone marrow; extramedullary hematopoiesis*Associated with:*metastatic carcinoma, chemical poisoning, chronic infection (TB), acute myelogenous leukemia, polycythemia vera, McCune-Albright syndrome, histiocytosis ● dyspnea, weakness, fatigue, weight loss, hemorrhage ● normochromic normocytic anemia; polycythemia may precede myelosclerosis in 59% ● dry marrow aspirateLocation:red marrow-containing bones in 40% (thoracic cage, pelvis, femora, humeral shafts, lumbar spine, skull, peripheral bones)√ splenomegaly√ widespread diffuse increase in density (ground glass)√ "jail-bar" ribs√ sandwich / rugger jersey spine√ generalized increase in bone density in skull + obliteration of diploic space; scattered small rounded radiolucent lesions; or combination of bothNUC: √ diffuse increased uptake of bone tracer in affected skeleton, possibly "superscan"√ increased uptake at ends of long bones *DDx:*(a)with splenomegaly: chronic leukemia, lymphoma, mastocytosis(b)without splenomegaly: osteoblastic metastases, fluorine poisoning, osteopetrosis, chronic renal disease

Notes:

MYOSITIS OSSIFICANS
=PSEUDOMALIGNANT OSSEOUS TUMOR OF SOFT TISSUE = EXTRAOSSEOUS LOCALIZED NONNEOPLASTIC BONE AND CARTILAGE FORMATION = MYOSITIS OSSIFICANS CIRCUMSCRIPTA = HETEROTOPIC OSSIFICATION=benign solitary self-limiting ossifying soft-tissue mass typically occurring within skeletal muscle*Age:*adolescents, young athletic adults; M > F*Path:*lesion rimmed by compressed fibrous connective tissue + surrounded by atrophic skeletal muscle (myositis = misnomer since no primary inflammation of muscle present)*Histo:* (a)early: focal hemorrhage + degeneration + necrosis of damaged muscle; histiocytic invasion; central nonossified core of proliferating benign fibroblasts + myofibroblasts; mesenchymal cells enclosed in ground substance assume characteristics of osteoblasts with subsequent mineralization + peripheral bone formation(b)intermediate age (3-6 weeks): "zone phenomenon" with central area of cellular variation and atypical mitotic figures (impossible to differentiate from soft-tissue sarcoma); middle zone of immature osteoid; outer zone of well-formed mature trabeculated dense bone ● history of direct trauma (75%) ● pain, tenderness, soft-tissue massLocation:large muscles of extremities (80%)(a)within muscle: anterolateral aspect of thigh + arm; temporal muscle; small muscles of hands; gluteal muscle; **"riders bone"** (adductor longus); **"fencers bone"** (brachialis); **"dancers bone"** (soleus); breast, elbow, knee(b)periosteal at tendon insertion: **Pellegrini-Stieda disease** (medial collateral ligament of knee)√ faint calcifications develop in 2-6 weeks after onset of symptoms √ well-defined partially ossified soft-tissue mass apparent by 6-8 weeks, becoming smaller + mature by 5-6 months√ radiolucent zone separating lesion from bone (DDx: periosteal sarcoma on stalk)√ ± periosteal reactionCT: √ well-defined mineralization at periphery of lesion after 4-6 weeks + less distinct lucent center(DDx: sarcoma with ill-defined periphery + calcified ossific center)√ diffuse ossification in mature lesionMR: Early phase: √ mass with poorly defined margins√ inhomogeneously hyperintense to fat on T2WI√ isointense to muscle on T1WI√ contrast enhancementIntermediate phase: √ isointense / slightly hyperintense core on T1WI, increasing in intensity on T2WI√ rim of curvilinear areas of decreased signal intensity surrounding the lesion (= peripheral mineralization / ossification)√ increased peritumoral signal intensity on T2WI(= edema of diffuse myositis)√ focal signal abnormality within bone marrow (= marrow edema)Mature phase: √ well-defined inhomogeneous mass with signal intensity approximating fat√ decreased signal intensity surrounding lesion + within (dense ossification + fibrosis, hemosiderin from previous hemorrhage)NUC: √ intense tracer accumulation on bone scan (directly related to deposition of calcium in damaged muscle)√ in phase of mature ossification activity becomes reduced + surgery may be performed with little risk of recurrenceAngio: √ diffuse tumor blush + fine neovascularity in early active phase√ avascular mass in mature healing phase *Prognosis:*? resorption in 1 year*DDx:* ◊In early stages difficult to differentiate histologically + radiologically from soft-tissue sarcomas!(1)Osteosarcoma(2)Synovial sarcoma(3)Fibrosarcoma(4)Chondrosarcoma(5)Rhabdomyosarcoma(6)Parosteal sarcoma (usually metaphyseal with thick densely mineralized attachment to bone)(7)Posttraumatic periostitis (ossification of subperiosteal hematoma with broad-based attachment to bone)(8)Acute osteomyelitis (substantial soft-tissue edema + early periosteal reaction)(9)Tumoral calcinosis (periarticular calcific masses of lobular pattern with interspersed lucent soft-tissue septa)(10) Osteochondroma (stalk contiguous with normal adjacent cortex + medullary space)

Myositis Ossificans Variants

Notes:

Myositis Ossificans **Variants** ***Panniculitis ossificans*** Location:subcutis of mostly upper extremities√ less prominent zoning phenomenon***Fasciitis ossificans*** Location:fascia***Fibro-osseous pseudotumor of digits*** =FLORID REACTIVE PERIOSTITIS*Age:*mean age of 32 years (range 4 -64 years);M:F = 1:2 ● fusiform swelling / massLocation:predominantly fingers (2nd > 3rd > 5th), occasionally toesSite:proximal > distal > middle phalanx√ radiopaque soft-tissue mass with radiolucent band between mass + cortex√ visible calcifications (50%)√ focal periosteal thickening (50%)√ cortical erosion (occasionally)

Notes:

NAIL-PATELLA SYNDROME
=FONG DISEASE = ILIAC HORNS = FAMILIAL / HEREDITARY OSTEO-ONYCHODYSPLASIA=OSTEO-ONYCHODYSOSTOSIS = HOOD SYNDROME=ELBOW-PATELLA SYNDROME=rare autosomal dominant disorder characterized by symmetrical meso- and ectodermal anomalies*Etiology:*? enzymatic defect in collagen metabolism*Age:*evident in 2nd + 3rd decades ● aplasia / hypoplasia of thumb + index fingernails ● bilateral spooning / splitting / ridging of fingernails ● abnormal gait ● abnormal pigmentation of iris ● renal dysfunction (secondary to abnormal glomerular basement membrane): proteinuria, hematuria, failure later in life√ bilateral posterior iliac horns in 80% (occasionally capped by an epiphysis) DIAGNOSTIC√ flared iliac crest with protuberant anterior iliac spines√ genu valgum due to asymmetrical development of femoral condyles√ prominent tibial tubercles√ fragmentation / hypoplasia / absence of patella; frequently with recurrent lateral dislocations √ radial head / capitellum hypoplasia with subluxation / dislocation of radial head dorsally and increased carrying angle of elbow (DDx: congenital dislocation of radial head)√ clinodactyly of 5th finger√ short 5th metacarpal√ flexion contractures of hip, knee, elbow, fingers, foot√ deltoid, triceps, quadriceps hypoplasia√ mandibular cysts (occasionally)√ scoliosis√ renal osteodystrophy*DDx:*(1)Seckel syndrome(2)"Bird-headed dwarfism" (absence of patella, radial head dislocation)(3)Popliteal pterygium syndrome (absence of patella, toenail dysplasia)

Notes:

NECROTIZING FASCIITIS
*Incidence:*500 cases in literature*Age:*58 ± 14 years; M>F*Cause:*deep internal infection / malignancy (perforated duodenal ulcer / retroperitoneal appendix, retroperitoneal / perirectal infection, infiltrating rectal / sigmoid carcinoma*Predisposed:*patients with diabetes, cancer, alcohol / drug abuse, poor nutrition*Organism:*Staphylococcus, E. coli, Bacteroides, Streptococcus, Peptostreptococcus, Klebsiella, Proteus, C. perfringens (5-15%) (multiple organisms in 75%)*Histo:*necrotic superficial fascia, leukocytic infiltration of deep fascial layers; fibrinoid thrombosis of arterioles + venules with vessel wall necrosis; microbial infiltration of destroyed fascia ● indolent (1-21 days delay before diagnosis) ● nonspecific symptoms: severe pain, fever, leukocytosis, shock, altered mental status ● crepitus (50%), overlying skin may be completely intactLocation:lower extremity, arm, neck, back, male perineum / scrotum (= **Fournier gangrene**)√ asymmetric fascial thickening with fat stranding (80%) from fluid√ gas in soft-tissues dissecting along fascial planes from gas-forming organisms (in 55%)√ associated deep abscess (35%)√ ± secondary muscle involvement *Prognosis:*poor with delay in diagnosis*Rx:*extensive surgical débridement*DDx:*(1)myonecrosis (infection originating in muscle)(2)fasciitis-panniculitis syndromes (chronic swelling of skin + underlying soft-tissues + fascial planes in arm + calf)(3)soft-tissue edema of CHF / cirrhosis (symmetrical diffuse fat stranding)

Notes:

NEUROPATHIC OSTEOARTHROPATHY

=NEUROTROPHIC JOINT = CHARCOT JOINT=traumatic arthritis due associated with loss of sensation + proprioception of affected limb*Pathogenesis:*(1)decreased pain sensation produces repetitive trauma(2)sympathetic dysfunction results in local hyperemia + bone resorption*Cause:* A.Congenital1.Myelomeningocele2.Congenital indifference to pain = asymboliaB.Acquired(a)central neuropathy1.Injury to brain / spinal cord2.Syringomyelia (in 1/3 of patients): shoulder, elbow3.Neurosyphilis = tabes dorsalis (in 15-20% of patients): hip, knee, ankle, tarsals4.Spinal cord tumors / infection(b)peripheral neuropathy1.Diabetes mellitus (most common cause, although incidence low): ankle, foot, hand2.Leprosy3.Peripheral nerve injury(c)others1.Scleroderma, Raynaud disease, Ehlers-Danlos syndrome2.Rheumatoid arthritis, psoriasis3.Amyloid infiltration of nerves, adrenal hypercorticismC.Iatrogenicprolonged use of pain-relieving drugs *mnemonic:*"DS6"**D**iabetes **S**yphilis**S**pina bifida**S**teroids**S**yringomyelia**S**pinal cord injury**S**cleroderma *Pathology:* (a)atrophic resorptive / hyperemic phase:osteoclasts + macrophages remove bone + cartilage debris making bone susceptible to fractures + joint destruction(b)hypertrophic reparative sclerotic phase ● no history of trauma ● swollen + warm joint with normal WBC count + ESR (infection may coexist) ● usually painless joint; pain at presentation (in 1/3) with decreased response to deep pain + proprioception ● joint changes frequently precede neurologic deficit ● synovial fluid: frequently xanthochromic / bloody, lipid crystals (from bone marrow)√ persistent joint effusion (first sign)√ narrowing of joint space√ speckled calcification in soft tissue (= calcification of synovial membrane)√ fragmentation of eburnated subchondral bone√ NO juxta-articular osteoporosis (unless infected)√ "bag-of-bones" appearance in late stage (= marked deformities around joint)*mnemonic:* "6 Ds"√ **D**ense subchondral bone (= sclerosis) √ **D**egeneration (= attempted repair by osteophytes)√ **D**estruction of articular cortex (with sharp margins resembling those of surgical amputation)√ **D**eformity ("pencil point" deformity of metatarsal heads)√ **D**ebris (loose bodies)√ **D**islocation (nontraumatic)√ subluxation of joints (laxity of periarticular soft tissues)√ talonavicular displacement with midfoot arthropathy (common in diabetic neuropathy)√ progressive rapid bone resorption√ joint distension (by fluid, hypertrophic synovitis, osteophytes, subluxation)MR: √ decreased signal intensity in bone marrow on T1WI + T2WI (due to osteosclerotic changes)@Spine (involved in 6-21%):√ lysis / sclerosis of intervertebral + facet joints√ scoliosis√ large osteophytes with beaking

Notes:

NODULAR SYNOVITIS
= GIANT CELL TUMOR OF TENDON SHEATH *Histo:*very cellular tumor with a capsule that separates the tumor into lobulesLocation:soft tissue of hand, occasionally lower extremity√ lobulated lesion with well-defined nodules up to 4 cm in size√ located along tendon sheath (CHARACTERISTIC)MR: √ low signal intensity on T1WI + T2WI (hemosiderin deposition)

Notes:

NONOSSIFYING FIBROMA
=FIBROXANTHOMA = NONOSTEOGENIC FIBROMA=XANTHOMA = XANTHOGRANULOMA OF BONE=FIBROUS METAPHYSEAL-DIAPHYSEAL DEFECT=FIBROUS MEDULLARY DEFECT*Incidence:*up to 40% of all children >2 years of age*Etiology:*lesion resulting from proliferative activity of a fibrous cortical defect that has expanded into medullary cavity*Histo:*whorled bundles of spindle-shaped fibroblasts + scattered multinucleated giant cells + foamy xanthomatous cells*Age:*8-20 years; 75% in 2nd decade of life ● usually asymptomaticLocation:shaft of long bone; mostly in bones of lower extremity, especially about knee (distal femur + proximal tibia); distal tibia; fibulaSite:eccentric metaphyseal, several cm shaftward from epiphysis, mostly intramedullary, rarely purely diaphysealMultiple fibroxanthomas (in 8-10%) *Associated with:*neurofibromatosis, fibrous dysplasia, Jaffe-Campanacci syndrome√ multilocular ovoid bubbly osteolytic area√ alignment along long axis of bone, about 2 cm in length√ dense sclerotic border toward medulla; V- or U-shaped at one end√ endosteal scalloping + thinning ± overlying bulge√ migrates toward center of diaphysis√ resolves with age√ minimal / mild uptake on bone scan*Prognosis:*spontaneous healing in most cases*Cx:*(1)Pathologic fracture (not uncommon)(2)Hypophosphatemic vitamin D-resistant rickets + osteomalacia (tumor may secrete substance that increases renal tubular resorption of phosphorus)*DDx:*(1)Adamantinoma (midshaft of tibia)(2)Chondromyxoid fibroma (bulging of cortex more striking)

Jaffe-Campanacci Syndrome

Notes:

Jaffe-Campanacci Syndrome =nonossifying fibroma with extraskeletal manifestations in children ● mental retardation ● hypogonadism ● ocular defect ● cardiovascular congenital defect ● café-au-lait spots

Notes:

NOONAN SYNDROME
=PSEUDO-TURNER = MALE TURNER SYNDROME=phenotype similar to Turner syndrome but with normal karyotype (occurs in both males + females)Striking familial incidence ● short / may have normal height ● webbed neck ● agonadism / normal gonads ● delayed puberty ● mental retardation√ osteoporosis√ retarded bone age√ cubitus valgus@Skull√ mandibular hypoplasia with dental malocclusion√ hypertelorism√ biparietal foramina√ dolichocephaly, microcephaly / cranial enlargement√ webbed neck@Chest√ sternal deformity: pectus excavatum / carinatum√ right-sided congenital heart disease (valvar pulmonic stenosis, ASD, eccentric hypertrophy of left ventricle, PDA, VSD)√ coronal clefts of spine√ may have pulmonary lymphangiectasis@Gastrointestinal tract√ intestinal lymphangiectasia√ eventration of diaphragm√ renal malrotation, renal duplication, hydronephrosis, large redundant extrarenal pelvis*DDx:*Turner syndrome (mental retardation rare, renal anomalies frequent)

Notes:

OCHRONOSIS
=ALKAPTONURIA = inherited absence of homogentisic acid oxidase with excessive homogentisic acid production + deposition in connective tissue including cartilage, synovium, and bone*Histo:*abnormally pigmented cartilage subject to deterioration resulting in calcification + denudation of cartilaginous tissueM:F = 2:1 ● black pigment in soft tissues (in 2nd decade): yellowish skin; gray pigmentation of sclera; bluish tinge of ears + nose cartilage ● alkaptonuria with black staining of diapers ● heart failure, renal failure (pigment deposition)@Spine*Age:*middle ageSite:lumbar region with progressive ascension√ laminated calcification of multiple intervertebral disks√ disk space drastically narrowed√ multiple "vacuum" phenomena (common)√ osteoporosis of adjoining vertebrae√ massive osteophytosis + ankylosis of spine (in older patient)√ spotty calcifications in tissue anterior to vertebral bodies@Joints√ hypertrophic changes in humeral head√ severe premature progressive osteoarthritic changes in shoulder, knee, hip, spine of young patients√ intra-articular osseous bodies√ small calcifications in para-articular soft tissues + tendon insertions

Notes:

ORODIGITOFACIAL SYNDROME
=OROFACIODIGITAL SYNDROME=group of heterogeneous defects, probably representing varying expressivity, involving face, oral cavity, and limbs
*Etiology:*autosomal trisomy of chromosome No. 1 with 47 chromosomes; X-linked dominant *Sex:*nuclear chromatin pattern female (lethal in male)*Associated with:*renal polycystic disease ● mental retardation ● hypertelorism ● cleft lip + tongue, lingual hamartoma ● bifid nasal tip√ cleft in palate + jaw bone√ hypoplasia of mandible (micrognathia) + occiput of skull√ hypodontia√ clinodactyly, syndactyly, brachydactyly (metacarpals may be elongated), polysyndactyly, duplication of hallux

Notes:

OSGOOD-SCHLATTER DISEASE
=traumatically induced disruption of the attachment of the patellar ligament to the tibial tuberosity (NOT osteonecrosis); bilateral in 25%*Age:*10-15 years; M > F*Cause:*trauma (common in sports that involve jumping, kicking, squatting) = ? cartilaginous avulsion fracture, ? tendinitis ● local pain + tenderness on pressure ● swelling of overlying soft tissue√ soft-tissue swelling in front of tuberosity (= edema of skin + subcutaneous tissue)√ thickening of distal portion of patellar tendon√ indistinct margin of patellar tendon√ increased radiodensity of infrapatellar fat pad√ avulsion with separation of small ossicles from the developing ossification center of tibial tuberosity√ single / multiple ossifications in avulsed fragment√ comparison with other side (irregular development normal)MR: √ increased signal intensity at tibial insertion site of patellar tendon on T1WI + T2WI√ distension of deep infrapatellar bursa√ bone marrow signal changes in tibial tuberosity + tibial apophysis (rare)*Cx:*nonunion of bone fragment, patellar subluxation, chondromalacia, avulsion of patellar tendon, genu recurvatum*Rx:*immobilization / steroid injection*DDx:*(1)normal ossification pattern of tibial tuberosity between ages 8-14 (no symptoms)(2)Osteitis: tuberculous / syphilitic(3)Soft-tissue sarcoma with calcifications

Notes:

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OSLER-WEBER-RENDU SYNDROME
=HEREDITARY HEMORRHAGIC TELANGIECTASIA=autosomal dominant systemic fibrovascular dysplasia of all vessels resulting in(1)telangiectasias(2)arteriovenous malformations (AV hemangiomas)(3)aneurysms ● frequent bleeding into mucous membranes, skin, lungs, genitourinary system, gastrointestinal system (due to vascular weakness) ● congestive heart failure (due to AV shunting)

Notes:

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OSSIFYING FIBROMA
Closely related to fibrous dysplasia + adamantinoma *Age:*2nd-4th decade; M < F*Histo:*maturing cellular fibrous spindle cells with osteoblastic activity producing many calcific cartilaginous + bone densitiesLocation: frequently in face @Mandible, maxilla ● painless expansion of tooth-bearing portion of jaw√ 1-5 cm well-circumscribed round / oval tumor√ moderate expansion of intact cortex√ homogeneous tumor matrix√ dislodgment of teeth@Tibia√ eccentric ground-glass lesion (resembling fibrous dysplasia)*Cx:*frequent recurrences

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Notes:

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OSTEITIS CONDENSANS ILII

*Incidence:*2% of population*Cause:*chronic stress secondary to instability of pubic symphysis*Age:*young multiparous women ● associated with low back pain when instability of pubic symphysis present√ triangular area of sclerosis along inferior anterior aspect of ileum adjacent to SI joint (joint space uninvolved)√ similar triangle of reparative bone on sacral side√ usually bilateral + symmetric; occasionally unilateral√ sclerosis dissolves in 3-20 years following stabilization of pubic symphysis*DDx:*(1)Ankylosing spondylitis (affects ilium + sacrum, joint space narrowing, involvement of other bones)(2)Rheumatoid arthritis (asymmetric, joint destruction)(3)Paget disease (thickened trabecular pattern)

Notes:

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OSTEOARTHRITIS

=DEGENERATIVE JOINT DISEASE = decreased chondroitin sulfate with age creates unsupported collagen fibrils followed by cartilage degeneration√ joint space narrowing√ sclerosis / eburnation of subchondral bone in areas of stress√ subchondral cyst formation (geodes)√ osteophytosis at articular margin / nonstressed area@Hand + foot*Target area:*1st MCP; trapezioscaphoid; DIP > PIP; 1st MTP√ radial subluxation of 1st metacarpal base√ Bouchard nodes = osteophytosis at PIP joint√ Heberden nodes = osteophytosis at DIP joint:M:F = 1:10 @Hip√ superior migration of femoral head (less frequently medial / axial)√ femoral + acetabular osteophytes, sclerosis, cyst formation√ thickening / buttressing of medial femoral cortex@Knee√ medial femorotibial compartment usually first to be involved√ varus deformity@Spine√ sclerosis + narrowing of intervertebral apophyseal joints√ osteophytosis usually associated with discogenic disease

Erosive Osteoarthritis Early Osteoarthritis

Notes:

Erosive Osteoarthritis =inflammatory form of osteoarthrosis*Predisposed:*postmenopausal femalesSite:DIP + PIP joints of hands; bilateral + symmetric√ "bird-wing" / "sea-gull" joint configuration = central erosions√ may lead to bony ankylosis*DDx:*Rheumatoid arthritis, Wilson disease, chronic liver disease, hemochromatosis

Notes:

Early Osteoarthritis *mnemonic:*"**E**arly **O**steo**A**rthritis"**E**piphyseal dysplasia, multiple **O**chronosis **A**cromegaly

Notes:

OSTEOBLASTOMA

=GIANT OSTEOID OSTEOMA = OSTEOGENIC FIBROMA OF BONE = OSSIFYING FIBROMA=rare benign tumor with unlimited growth potential + capability of malignant transformation*Incidence:*<1% of all primary bone tumors; 3% of all benign bone tumors*Age:*mean age of 16-19 years; 6-30 years (90%); 2nd decade (55%); 3rd decade (20%); M:F = 2:1*Path:*lesion >1.5 cm; smaller lesions are classified as osteoid osteoma*Histo:*numerous multinucleated giant cells (osteoclasts), irregularly arranged osteoid + bone; very vascular connective tissue stroma with interconnecting trabecular bone; trabeculae broader + longer than in osteoid osteoma ● asymptomatic in <2% ● dull localized pain of insidious onset (84%), worse at night in 7-13% ● response to salicylates in 7% ● localized swelling, tenderness, decreased range of motion (29%) ● painful scoliosis in 50% (with spinal / rib location) secondary to muscle spasm, may be convex toward side of tumor ● paresthesias, mild muscle weakness, paraparesis, paraplegia (due to cord compression) ● occasional systemic toxicity (high WBC, fever) Location: (rarely multifocal) (a)spine (33-37%): 62-94% in posterior elements, secondary extension into vertebral body (28-42%); cervical spine (31%), thoracic spine (34%), lumbar spine (31%), sacrum (3%)(b)long bones (26-32%): femur (50%), tibia (19%), humerus (19%), radius (8%), fibula (4%); unusual in neck of femur(c)small bones of hand + feet (15-26%): dorsal talus neck (62%), calcaneus (4%), scaphoid (8%), metacarpals (8%), metatarsals (8%)(d)calvarium + mandible (= cementoblastoma)Site:diaphyseal (58%), metaphyseal (42%); eccentric (46%), intracortical (42%), centric (12%), may be periosteal √ similar to osteoid osteoma:√ radiolucent nidus >2 cm (range of 2-12 cm) in size√ well demarcated (83%) √ ± stippled / ringlike small flecks of matrix calcification√ reactive sclerosis (22-91%) / no sclerosis (9-56%)√ progressive expansile lesion that may rapidly increase in size (25%):√ cortical expansion (75-94%) / destruction (20-22%)√ tumor matrix radiolucent (25-64%) / ossified (36-72%)√ sharply defined soft-tissue component√ thin shell of periosteal new bone (58-77%) / no periosteal reaction√ scoliosis (35%)√ osteoporosis due to disuse + hyperemia in talar location√ rapid calcification after radiotherapyCT: √ multifocal matrix mineralization, sclerosis√ expansile bone remodeling, thin osseous shellNUC: √ intense focal accumulation of bone agent (100%)Angio: √ tumor blush in capillary phase (50%)MR: √ low to intermediate signal intensity on T1WI√ mixed intermediate to high intensity on T2WI√ surrounding edema *Prognosis:*10% recurrence after excision; incomplete curettage can effect cure due to cartilage production + trapping of host lamellar bone*DDx:* (1)Osteo- / chondrosarcoma (periosteal new bone)(2)Osteoid osteoma (dense calcification + halo of bone sclerosis, stable lesion size <2 cm due to limited growth potential)(3) Cartilaginous tumors (lumpy matrix calcification(4) Giant cell tumor (no calcification, epiphyseal involvement)(5)Aneurysmal bone cyst(6)Osteomyelitis(7)Hemangioma(8)Lipoma(9)Epidermoid(10) Fibrous dysplasia(11) Metastasis(12) Ewing sarcoma

Notes:

OSTEOCHONDROSIS DISSECANS
=OSTEOCHONDRITIS DISSECANS=OSTEOCHONDRAL FRACTURE=fragmentation + possible separation of a portion of the articular surface*Etiology:* (1)subchondral fatigue fracture as a result of shearing, rotatory / tangentially aligned impaction forces(2)? autosomal dominant trait associated with short stature, endocrine dysfunction, Scheuermann disease, Osgood-Schlatter disease, tibia vara, carpal tunnel syndrome*Age:*adolescence; M > F ● asymptomatic / vague complaints ● clicking, locking, limitation of motion ● swelling, pain aggravated by movementLocation:(a)knee: medial (in 10% lateral) femoral condyle close to fossa intercondylaris; bilateral in 20-30%(b)humeral head(c)capitellum of elbow(d)talus√ purely cartilaginous fragment unrecognized on plain film√ fracture line parallels joint surface√ mouse = osteochondrotic fragmentLocation:posterior region of knee joint, olecranon fossa, axillary / subscapular recess of glenohumeral joint√ mouse bed = sclerosed pit in articular surface√ soft-tissue swelling, joint effusion*DDx:*spontaneous osteonecrosis, neuroarthropathy, degenerative joint disease, synovial osteochondromatosis

Notes:

OSTEOFIBROUS DYSPLASIA
=entity previously mistaken for fibrous dysplasia*Age:*newborn up to 5 years*Histo:*fibrous tissue surrounding trabeculae in a whorled storiform patternLocation:normally confined to tibia (middiaphysis in 50%), lesion begins in anterior cortex; ipsilateral fibula affected in 20%√ enlargement of tibia with anterior bowing√ cortex thin / invisible√ periosteal expansion√ sclerotic margin (DDx: nonosteogenic fibroma, chondromyxoid fibroma)√ spontaneous regression in 1/3*Cx:*pathologic fracture in 25%, fractures will heal with immobilization; infrequently complicated by pseudarthrosis*DDx:*fibrous dysplasia, Paget disease

Notes:

OSTEOGENESIS IMPERFECTA
=PSATHYROSIS = FRAGILITAS OSSIUM = LOBSTEIN DISEASE=heterogeneous group of a generalized connective tissue disorder leading to micromelic dwarfism characterized by bone fragility, blue sclerae, and dentinogenesis imperfecta*Incidence:*overall in 1:28,500 (20,000-60,000) live births; M:F = 1:1*Histo:*immature collagen matrix*Clinical types:* 1.OSTEOGENESIS IMPERFECTA CONGENITA=disease manifest at birth (occurring in utero); autosomal dominant; corresponds to type II; lethal variety2.OSTEOGENESIS IMPERFECTA TARDA=usually not manifest at birth; recessive / sporadic corresponds to type I + IV; nonlethal variety ● soft skull (caput membranaceum) ● hyperlaxity of joints ● blue sclerae ● poor dentition ● otosclerosis ● thin loose skin√ diffuse demineralization, deficient trabecular structure, cortical thinning√ defective cortical bone: increase in diameter of proximal ends of humeri + femora; slender fragile bone; multiple cystlike areas√ multiple fractures + pseudarthrosis with bowing (vertebral bodies, long bones)√ normal / exuberant callus formation√ rib thinning / notching√ thin calvarium√ sinus + mastoid cell enlargement√ thickened undermineralized otic capsule (= otosclerosis)√ wormian bones persisting into adulthood√ basilar impression (= platybasia)√ biconcave vertebral bodies + Schmorl nodes, increased height of intervertebral disk space√ bowing deformities after child begins to walk*Cx:*(1)impaired hearing / deafness from otosclerosis (20-60%)(2)death from intracranial hemorrhage (abnormal platelet function)*Dx:*chorionic villous sampling

Osteogenesis Imperfecta Type I Osteogenesis Imperfecta Type II Osteogenesis Imperfecta Type III Osteogenesis Imperfecta Type IV

Notes:

Osteogenesis Imperfecta **Type I** Autosomal dominant; compatible with life *Age at presentation:* 2-6 years
● blue sclerae ● presenile deafness ● normal / abnormal dentinogenesis√ infants of normal weight + length√ osteoporosis√ fractures in neonate (occurring during delivery)OB-US: √ marked bowing of long bones√ NO IUGR

Notes:

Osteogenesis Imperfecta **Type II** =CONGENITAL LETHAL OIAutosomal recessive / sporadic; perinatal lethal form *Incidence:*1:54,000 births; most frequent variety ● blue sclerae ● ligamentous laxity + loose skin√ shortened broad crumpled long bones√ bone angulations, bowing, demineralization√ localized bone thickening from callus formation√ thin beaded ribs ± fractures resulting in bell-shaped / narrow chest√ thin poorly ossified skull√ spinal osteopenia√ platyspondlyOB-US: A normal sonogram after 17 weeks MA excludes the diagnosis! √ increased through-transmission of skull (extremely poor mineralization)√ unusually good visualization of brain surface√ unusually good visualization of orbits√ increased visualization of intracranial arterial pulsations√ abnormal compressibility of skull vault with transducer√ decreased visualization of skeleton√ multiple fetal fractures + deformities of long bones + ribs√ wrinkled appearance of bone (= more than one fracture in single bone) √ beaded ribs (callus formation around fractures)√ abnormally short limbs√ small thorax (collapse of thoracic cage)√ decreased fetal movement√ infants small for gestational age (frequent)√ polyhydramnios*Prognosis:*stillborn / death shortly after birth due to pulmonary hypoplasia*DDx:*congenital hypophosphatasia, achondrogenesis type I

Notes:

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Osteogenesis Imperfecta **Type III** =SEVERE PROGRESSIVELY DEFORMING OIAutosomal recessive / dominant; progressively deforming disorder compatible with life ● bluish sclerae during infancy which turn pale with time ● joint hyperlaxity (50%)√ decreased ossification of skull√ normal vertebrae + pelvis√ progressive deformities of limbs + spine into adulthood √ shortened + bowed long bones√ ± rib fractures√ multiple fractures present at birth in 2/3 of cases√ fractures heal wellOB-US: √ short + bowed long bones√ fractures√ humerus almost normal in shape√ normal thoracic circumference*Prognosis:*progressive limb + spine deformities during childhood / adolescence

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Notes:

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Osteogenesis Imperfecta **Type IV** Autosomal dominant; mildest form with best prognosis ● normal scleral color ● little tendency to develop hearing loss√ tubular bones of normal length; mild femoral bowing may occur√ osteoporosisOB-US: √ bowing of long bones

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Notes:

OSTEOID OSTEOMA
=benign skeletal neoplasm composed of osteoid + woven bone less than 1.5 cm in diameter per definition*Incidence:*12% of benign skeletal neoplasms*Etiology:*? inflammatory response*Histo:*small nidus of osteoid-laden interconnected trabeculae with a background of highly vascularized fibrous connective tissue surrounded by zone of reactive bone sclerosis; osteoblastic rimming; indistinguishable from osteoblastoma*Age:*10-20 years (51%); 2nd + 3rd decade (73%); 5-25 years (90%); range of 19 months-56 years; uncommon <5 and >40 years of age; M:F = 2:1; uncommon in Blacks ● tender to touch + pressure ● local pain (95-98%), weeks to years in duration, worse at night, decreased by activity ● salicylates give relief in 20-30 minutes in 75-90% ● prostaglandin E2 elevated 100-1000 x normal within nidus (probable cause of pain and vasodilatation) Location: (a)meta- / diaphysis of long bones (73%): upper end of femur (43%), hands (8%), feet (4%); frequent in proximal tibia + femoral neck, fibula, humerus; no bone exempt(b)spine (10-14%): predominantly in posterior elements (50% in pedicle + lamina + spinous process; 20% in articular process) of lumbar (59%), cervical (27%), thoracic (12%), sacral (2%) segments ● painful scoliosis, focal / radicular pain ● gait disturbance, muscle atrophy(c)skull, rib, ischium, mandible, patella *Classification:* **Cortical osteoid osteoma** (most common) = nidus within cortex √ solid / laminated periosteal reaction √ fusiform sclerotic cortical thickening in shaft of long bone √ radiolucent area within center of osteosclerosis **Cancellous Osteoid Osteoma** (intermediate frequency) = intramedullary ◊Intra-articular lesion difficult to identify with delay in diagnosis of 4 months-5 years!Site:juxta- / intra-articular at femoral neck, vertebral posterior elements, small bones of hands + feet √ little osteosclerosis / sclerotic cortex distant to nidus (functional difference of intra-articular periosteum) √ joint space widened (effusion, synovitis) **Subperiosteal Osteoid Osteoma** (rare) =round soft-tissue mass adjacent to boneSite:juxta- / intra-articular at medial aspect of femoral neck, hands, feet (neck of talus) √ juxtacortical mass excavating the cortex (bony pressure atrophy) with almost no reactive sclerosis √ round / oval radiolucent nidus (75%) of <1.5 cm in size √ variable surrounding sclerosis ± central calcification √ painful scoliosis concave toward lesion / kyphoscoliosis / hyperlordosis / torticollis with spinal location (due to spasm) √ may show extensive synovitis + effusion + premature loss of cartilage with intra-articular site (lymphofollicular synovitis) √ osteoarthritis (50%) with intra-articular site 1.5-22 years after onset of symptomatology √ regional osteoporosis (probably due to disuse) ◊Radiographically difficult areas: vertebral column, femoral neck, small bones of hand + feetNUC: √ intensely increased radiotracer uptake (increased blood flow + new-bone formation) √ double density sign = small area of focal activity (nidus) superimposed on larger area of increased tracer uptakeCT (for detection + precise localization of nidus): √ small well-defined round / oval nidus surrounded by variable amount of sclerosis √ nidus enhances on dynamic scan √ nidus with variable amount of mineralization (50%): punctate / amorphous / ringlike / denseMR (diminished conspicuity of lesion compared with CT): √ nidus isointense to muscle on T1WI √ signal intensity increases to between that of muscle + fat / remains low on T2WI √ perinidal inflammation of bone marrow (63%) √ perinidal soft-tissue inflammation / edema (47%) √ synovitis + joint effusion with intra-articular siteAngio: √ highly vascularized nidus with intense circumscribed blush appearing in early arterial phase + persisting late into venous phase*Prognosis:*no growth progression, infrequent regression*Rx:*(1)complete surgical excision of nidus (reactive bone regresses subsequently)(2)percutaneous CT-guided removal(3)percutaneous ablation with radio-frequency electrode / laser / alcohol*DDx:* (1)Cortical osteoid osteoma: Brodie abscess, sclerosing osteomyelitis, syphilis, bone island, stress fracture, osteosarcoma, Ewing sarcoma, osteoblastic metastasis, lymphoma, subperiosteal aneurysmal bone cyst, osteoblastoma (progressive growth)(2)Intra-articular osteoid osteoma: inflammatory / septic / tuberculous / rheumatoid arthritis, nonspecific synovitis / Legg-Calvé-Perthes disease

Notes:

OSTEOMA
= benign tumor of membranous bone (hamartoma) *Age:*adult life*Associated with:*Gardner syndrome (multiple osteomas + colonic polyposis)Location:inner / outer table of calvarium (usually from external table), paranasal sinuses (frontal / ethmoid sinuses), mandible, nasal bones well-circumscribed round extremely dense structureless lesion usually <2 cm in size **Fibrous Osteoma** Probably a form of fibrous dysplasia *Age:*childhood less dense than osteoma / radiolucent expanding external table without affecting internal table*DDx:*endostoma, bone island, bone infarct (located in medulla)

Notes:

Acute Osteomyelitis *Age:*most commonly affects children*Organisms:* (a) newborns: S. aureus, Group B streptococcus, Escherichia coli (b) children: S. aureus (blood cultures in 50% positive) (c) adults: S. aureus (60%), enteric species (29%), Streptococcus (8%) (d) drug addicts: Pseudomonas (86%), Klebsiella, Enterobacteriae; (57 days average delay in diagnosis) (e) sickle cell disease: Salmonella *Cause:* (1)Genitourinary tract infection (72%)(2)Lung infection (14%)(3)Dermal infection (14%): direct contamination from a soft-tissue lesion in diabetic patient*Pathogenesis:* (a)hematogenous spread(b)direct implantation from a traumatic / iatrogenic source(c)extension from adjacent soft-tissue infectionLocation: @Lower extremity (75%): over pressure points in diabetic foot@Vertebrae (53%): lumbar (75%) > thoracic > cervical@Radial styloid (24%)@Sacroiliac joint (18%) ● leukocytosis + fever (66%) A.ACUTE NEONATAL OSTEOMYELITIS*Age:*onset <30 days of age ● little / no systemic disturbance√ multicentric involvement more common; often joint involvement√ bone scan falsely negative / equivocal in 70%B.ACUTE OSTEOMYELITIS IN INFANCY *Age:*<18 months of age*Pathomechanism:*spread to epiphysis because transphyseal vessels cross growth plate into epiphysis√ striking soft-tissue component√ subperiosteal abscess with extensive periosteal new bone*Cx:*frequent joint involvement*Prognosis:*rapid healingC.ACUTE OSTEOMYELITIS IN CHILDHOOD*Age:*2-16 years of age*Pathomechanism:* transphyseal vessels closed; metaphyseal vessels adjacent to growth plate loop back toward metaphysis locating the primary focus of infection into metaphysis; abscess formation in medulla with cortical spread√ sequestration frequent√ periosteal elevation (with disruption of periosteal blood supply)√ small single / multiple osteolytic areas in metaphysis√ extensive periosteal reaction parallel to shaft (after 3-6 weeks); may be "lamellar nodular"(DDx: osteoblastoma, eosinophilic granuloma)√ shortening of bone with destruction of epiphyseal cartilage√ growth stimulation by hyperemia + premature maturation of adjacent epiphysis√ midshaft osteomyelitis less frequent site√ serpiginous tract with small sclerotic rim (PATHOGNOMONIC)D.ACUTE OSTEOMYELITIS IN ADULTHOOD√ delicate periosteal new bone√ joint involvement common Radiographs: √ initial radiographs often normal (notoriously poor in early phase of infection for as long as 10 days)√ localized soft-tissue swelling adjacent to metaphysis with obliteration of usual fat planes (after 3-10 days)√ area of bone destruction (lags 7-14 days behind pathologic changes)√ involucrum = cloak of laminated / spiculated periosteal reaction (develops after 20 days)√ sequestrum = detached necrotic cortical bone (develops after 30 days)√ cloaca formation = space in which dead bone residesMR: √ bone marrow hypointense on T1WI + hyperintense on T2WI (= water-rich inflammatory tissue)*DDx:*neuropathic osteoarthropathy, aseptic arthritis, acute fracture, recent surgery√ focal / linear cortical involvement hyperintense on T2WI√ hyperintense halo surrounding cortex on T2WI = subperiosteal infection√ hyperintense line on T2WI extending from bone to skin surface + enhancement of borders (= sinus tract) Abscess characteristics: √ hyperintense enhancing rim (= hyperemic zone) around a central focus of low intensity (= necrotic / devitalized tissue) on contrast-enhanced T1WI√ hyperintense fluid collection surrounded by hypointense pseudocapsule on T2WI + contrast-enhancement of granulation tissue√ hyperintense adjacent soft tissues on T2WI√ fat-suppressed contrast-enhanced imaging (88% sensitive + 93% specific compared with 79% + 53% for nonenhanced MR imaging) NUC (accuracy approx. 90%): (1)Ga-67 scans: 100% sensitivity; increased uptake 1 day earlier than for Tc-99m MDP✧Gallium helpful for chronic osteomyelitis!(2)Static Tc-99m diphosphonate: 83% sensitivity5-60% false-negative rate in neonates + children because of (a) masking effect of epiphyseal plates (b) early diminished blood flow with infection (c) spectrum of uptake pattern from hot to cold(3)Three-phase skeletal scintigraphy:92% sensitivity, 87% specificity*Phase 1:*Radionuclide angiography = perfusion phase of regional blood flow*Phase 2:*"blood pool" images*Phase 3:*"bone uptake"*Limitations:* diagnostic difficulties in children, in posttraumatic / postoperative state, diabetic neuropathy (poor blood supply), neoplasia, septic arthritis, Paget disease, healed osteomyelitis, noninfectious inflammatory process *DDx:*cellulitis (decrease in activity over time)(4)WBC-scan:(a)In-111-labeled leukocytes: best agent for acute infections(b)Tc-99m labeled leukocytes: preferred over In-111-leukocyte imaging especially in extremities✧WBC scans have largely replaced gallium imaging for acute osteomyelitis due to improved photon flux + improved dosimetry (higher dose allowed relative to In-111) allowing faster imaging + greater resolution√ "cold" area in early osteomyelitis subsequently becoming "hot" if localized to long bones / pelvis (not seen in vertebral bodies)√ local increase in radiopharmaceutical uptake (positive within 24-72 hours)*Cx:*(1) Soft-tissue abscess (2) Fistula formation(3) Pathologic fracture (4) Extension into joint(5) Growth disturbance due to epiphyseal involvement (6) Neoplasm (7) Amyloidosis(8) Severe deformity with delayed treatment

Notes:

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Chronic Osteomyelitis √ thick irregular sclerotic bone with radiolucencies, elevated periosteum, chronic draining sinus ***Sclerosing Osteomyelitis of Garré*** =low-grade infection, no purulent exudateLocation:mandible (most commonly)√ focal bulge of thickened cortex (sclerosing periosteal reaction)*DDx:*osteoid osteoma, stress fracture ***Chronic Recurrent Multifocal Osteomyelitis*** =benign self-limited disease of unknown etiology*Age:*children + adolescents; M:F = 1:2*Histo:*nonspecific subacute / chronic osteomyelitis ● pain, soft-tissue swelling, limited motionLocation:tibia > femur > clavicle > fibulaSite:metaphyses of long bones; often symmetric√ small areas of bone lysis, often confluent

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Notes:

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Brodie Abscess =subacute pyogenic osteomyelitis (smoldering indolent infection)*Organism:*S. aureus (most common)*Histo:*granulation tissue + eburnation*Age:*more common in children; M > FLocation:predilection for ends of tubular bones (proximal / distal tibial metaphysis most common); carpal + tarsal bonesSite:metaphysis, rarely traversing the open growth plate; epiphysis (children + infants)√ central area of lucency surrounded by dense rim of reactive sclerosis√ lucent channel-like / tortuous configuration extending toward growth plate (PATHOGNOMONIC)√ periosteal new-bone formation√ ± adjacent soft-tissue swelling√ may persist for many monthsMR: √ "double line" effect = high signal intensity of granulation tissue surrounded by low signal intensity of bone sclerosis on T2WI√ well-defined low- to intermediate-signal lesion outlined by low-signal rim on T1WI*DDx:*Osteoid osteoma

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Epidermoid Carcinoma *Etiology:*complication of chronic osteomyelitis (0.2-1.7%)*Histo:*squamous cell carcinoma (90%); occasionally: basal cell carcinoma, adenocarcinoma, fibro-sarcoma, angiosarcoma, reticulum cell sarcoma, spindle cell sarcoma, rhabdomyosarcoma, parosteal osteosarcoma, plasmacytoma*Age:*30-80 (mean 55) years; M >> F*Latent period:*20-30 (range of 1.5-72) years ● history of childhood osteomyelitis ● exacerbation of symptoms with increasing pain, enlarging mass ● change in character / amount of sinus drainageLocation:at site of chronically / intermittently draining sinus; tibia (50%), femur (21%)√ lytic lesion superimposed on changes of chronic osteomyelitis√ soft-tissue mass√ pathologic fracture*Prognosis:* (1)early metastases in 14-20-40% (within 18 months)(2)no recurrence in 80%

Notes:

OSTEOPATHIA STRIATA
=VOORHOEVE DISEASE ● usually asymptomatic (similar to osteopoikilosis)Location:all long bones affected; the only bone sclerosis primarily involving metaphysis (with extension into epi- and diaphysis)√ longitudinal striations of dense bone in metaphysis√ radiating densities of "sunburst" appearance from acetabulum into ileum

Notes:

OSTEOPETROSIS
=ALBERS-SCHÖNBERG DISEASE = MARBLE BONE DISEASE = rare hereditary disorder*Path:*defective osteoclast function with failure of proper reabsorption + remodeling of primary spongiosum; bone sclerotic + thick but structurally weak + brittle A.INFANTILE AUTOSOMAL RECESSIVE TYPE ● failure to thrive ● premature senile appearance of facies ● severe dental caries ● anemia, leukocytopenia, thrombocytopenia (severe marrow depression) ● cranial nerve compression (optic atrophy, deafness) ● hepatosplenomegaly (extramedullary hematopoiesis) ● lymphadenopathy ● subarachnoid hemorrhage (due to thrombocytopenia)*May be associated with:* renal tubular acidosis + cerebral calcification *Prognosis:*survival beyond middle life uncommon (death due to recurrent infection, massive hemorrhage, terminal leukemia)B.BENIGN ADULT AUTOSOMAL DOMINANT TYPE ● 50% asymptomatic ● recurrent fractures, mild anemia ● occasionally cranial nerve palsy*Prognosis:*normal life expectancy √ diffuse osteosclerosis = generalized dense amorphous structureless bones with obliteration of normal trabecular pattern; mandible least commonly involved√ cortical thickening with medullary encroachment√ Erlenmeyer flask deformity = clublike long bones due to lack of tubulization + flaring of ends√ bone-within-bone appearance√ "sandwich" vertebrae√ alternating sclerotic + radiolucent transverse metaphyseal lines (phalanges, iliac bones) as indicators of fluctuating course of disease√ longitudinal metaphyseal striations√ obliteration of mastoid cells, paranasal sinuses, basal foramina by osteosclerosis√ sclerosis predominantly involving base of skull; calvaria often spared *Cx:*(1)usually transverse fractures (common because of brittle bones) with abundant callus + normal healing(2)crowding of marrow (myelophthisic anemia + extramedullary hematopoiesis)(3)frequently terminates in acute leukemia*Rx:*bone marrow transplant *DDx:*(1)Heavy metal poisoning(2)Melorheostosis (limited to one extremity)(3)Hypervitaminosis D(4)Pyknodysostosis(5)Fibrous dysplasia of skull / face

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OSTEOPOIKILOSIS
=OSTEOPATHIA CONDENSANS DISSEMINATAOften autosomal dominant; M > F ● asymptomatic*Histo:*compact bone islandsLocation:in most metaphyses + epiphyses (rarely extending into midshaft); concentrated at glenoid + acetabulum, wrist, ankle, pelvis; rare in skull, ribs, vertebral centra, mandible√ small foci of ovoid / lenticular opacification (2-10 mm) in cancellous bone√ long axis of lesions parallel to long axis of bone*Prognosis:*not progressive, no change after cessation of growth*DDx:*(1)Epiphyseal dysplasia (metaphyses normal)(2)Melorheostosis (diaphyseal involvement)

Notes:

OSTEORADIONECROSIS
*Cause:*deleterious effect of radiation on osteoblasts, osteoclasts, vascular damage, increased susceptibility of irradiated bone to infection*Time of onset:*1-3 years following radiation therapy*Dose:*>6,000 cGy in adults; >2,000 cGy in children√ focal lytic area with abnormal bone matrix√ ± cortical thinning from chronic infection√ ± pathologic fracture*DDx:*neoplastic involvement (soft-tissue mass)

Notes:

OSTEOSARCOMA
Most common malignant primary bone tumor in young adults + children; 2nd most common primary malignant bone tumor after multiple myeloma *Prevalence:*4-5:1,000,000; 15% of all primary bone tumors confirmed at biopsy *Types & Frequency:* A.Conventional osteosarcoma:-high-grade intramedullary75%-telangiectatic4.5-11%-low-grade intraosseous4-5%-small cell1-4%-osteosarcomatosis3-4%-gnathic6-9%B.Surface / juxtacortical osteosarcoma:4-10%-intracorticalrare-parosteal65%-periosteal25%-high-grade surface10%C.Extraskeletal4%D.Secondary osteosarcoma5-7% *Prognosis:*dependent on age, sex, tumor size, site, classification; best predictor is degree of tissue necrosis in postresection specimen following chemotherapy (91% survival with tumor necrosis >90%, 14% survival with <90% tumor necrosis)

Extraskeletal Osteosarcoma High-grade Intramedullary Osteosarcoma High-grade Surface Osteosarcoma Intracortical Osteosarcoma Low-grade Intraosseous Osteosarcoma Osteosarcoma of Jaw Osteosarcomatosis Parosteal Osteosarcoma Periosteal Osteosarcoma Secondary Osteosarcoma Small-cell Osteosarcoma Telangiectatic Osteosarcoma

Notes:

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Extraskeletal Osteosarcoma =located within soft tissue without attachment to bone / periosteum*Incidence:*1% of soft-tissue sarcomas*Histo:*variable amounts of neoplastic osteoid + bone + cartilage; frequently associated with fibrosarcoma, malignant fibrous histiocytoma, malignant peripheral nerve sheath tumor*Mean age:*50 years; 94% >30 years of age; M > FLocation:lower extremity (thigh in 42-47%), upper extremity (12-23%), retroperitoneum (8-17%), buttock, back, orbit, submental, axilla, abdomen, neck, kidney, breast ● slowly growing soft-tissue mass ● painful + tender (25-50%) ● history of trauma (12-31%): in preexisting myositis ossificans / site of intramuscular injection√ often deep-seated + fixed soft-tissue tumor (average diameter of 9 cm)√ focal / massive area of mineralization (>50%)√ increased radionuclide uptake on bone scan*Prognosis:* (1)multiple local recurrences (in 80-90%) after interval of 2 months to 10 years(2)metastases after interval of 1 month to 4 years: lungs (81-100%), lymph nodes (25%), bone, subcutis, liver(3)death within 2-3 years (>50%) with tumor size as major predictor

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Notes:

High-grade Intramedullary Osteosarcoma =CENTRAL OSTEOSARCOMA = CONVENTIONAL OSTEOSARCOMA*Histo:*arising from undifferentiated mesenchymal tissue; forming fibrous / cartilaginous / osseous matrix (mostly mixed) that produces osteoid / immature bone(a)osteoblastic (50-80%)(b)chondroblastic (5-25%)(c)fibroblastic-fibrohistiocytic (7-25%)*Age:*bimodal distribution 10- 25 years and >60 years; 21% <10 years; 68% <15 years; 70% between 10 and 30 years; M:F = 3:2 to 2:1; >35 years: related to preexisting condition ● painful swelling (1-2 months duration) ● fever (frequent) ● slight elevation of alkaline phosphatase ● diabetes mellitus (paraneoplastic syndrome) in 25%Location:long bones (70-80%), femur (40-45%), tibia (16-20%); 50-55% about knee; proximal humerus (10-15%); cylindrical bone <30 years; flat bone (ilium) >50 yearsSite:origin in metaphysis (90-95%) / diaphysis (2-11%) /epiphysis (<1%); growth through open physis with extension into epiphysis (75-88%)Doubling time:20-30 day √ usually large bone lesion of >5-6 cm when first detected√ cloudlike density (90%) / almost normal density / osteolytic (fibroblastic type)√ aggressive periosteal reaction: sunburst / hair-on-end / onion-peel = laminated / Codman triangle√ moth-eaten bone destruction + cortical disruption √ soft-tissue mass with tumor new bone (osseous / cartilaginous type)√ transphyseal spread before plate closure (75-88%); physis does NOT act as a barrier to tumor spread√ spontaneous pneumothorax (due to subpleural metastases)NUC (bone scintigraphy): √ intensely increased activity on blood flow, blood pool, delayed images (hypervascularity, new-bone formation)√ soft-tissue extension demonstrated, especially with SPECT√ bone scan establishes local extent (extent of involvement easily overestimated due to intensity of uptake), skip lesions, metastases to bone + soft tissuesCT: √ soft-tissue attenuation (nonmineralized portion) replacing fatty bone marrow√ low attenuation (higher water content of chondroblastic component / hemorrhage / necrosis)√ very high attenuation (mineralized matrix)MR (preferred modality): √ tumor of intermediate signal intensity on T1WI + high signal intensity on T2WI√ clearly defines marrow extent (best on T1WI), vascular involvement, soft-tissue component (best on T2WI)*Evaluate for:* (1)extent of marrow + soft-tissue involvement(2)invasion of epiphysis(3)joint (19-24%) + neurovascular involvement(4)viable tumor + mineralized matrix for biopsy*Metastases* (in 2% at presentation): (a)hematogenous lung metastases (15%): calcifying; spontaneous pneumothorax secondary to subpleural cavitating nodules rupturing into pleural space(b)lymph nodes, liver, brain (may be calcified)(c)skeletal metastases uncommon (unlike Ewing sarcoma); skip lesions = discontinuous tumor foci in marrow cavity in 1-25%*Cx:*(1)pathologic fracture (15-20%)(2)radiation-induced osteosarcoma (30 years delay)*Rx:*chemotherapy followed by wide surgical resection*Prognosis:*60-80% 5-year survival(1)amputation: 20% 5-year survival; 15% develop skeletal metastases; 75% dead within <2 years(2) multidrug chemotherapy: 55% 4-year survival more proximal lesions carry higher mortality (0% 2-year survival for axial primary) Predictors of poor outcome: metastasis at presentation, soft-tissue mass >20 cm, pathologic fracture, skip lesions in marrowPredictors of poor response to chemotherapy: no change / increase in size of soft-tissue mass, increase in bone destruction*DDx:*Osteoid osteoma, sclerosing osteomyelitis, Charcot joint

Notes:

High-grade Surface Osteosarcoma Location:femur, humerus, fibulaSite:diaphysis√ similar to periosteal osteosarcoma√ often involve entire circumference of bone√ frequent invasion of medullary canal*Prognosis:*identical to conventional intramedullary osteosarcoma

Notes:

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Intracortical Osteosarcoma Rarest form of osteosarcoma *Histo:*sclerosing variant of osteosarcoma which may contain small foci of chondro- or fibrosarcomaLocation:femur, tibia√ tumor <4 cm in diameter√ intracortical geographic bone lysis√ tumor margin may be well defined with thickening of surrounding cortex√ metastases in 29%

Notes:

Low-grade Intraosseous Osteosarcoma =WELL-DIFFERENTIATED / SCLEROSING OSTEOSARCOMA*Age:*most frequently 3rd decade; M:F = 1:1 ● protracted clinical course with nonspecific symptomsLocation:about the kneeSite:metaphysis; often with extension into epiphysis√ may have well-defined margins + sclerotic rim√ diffuse sclerosis√ expansile remodeling of bone√ subtle signs of aggressiveness: bone lysis, focally indistinct margin, cortical destruction, soft-tissue mass, periosteal reaction*Cx:*transformation into high-grade osteosarcoma*DDx:*fibrous dysplasia, nonossifying fibroma, chondrosarcoma, chondromyxoid fibroma

Notes:

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Osteosarcoma of Jaw =GNATHIC OSTEOSARCOMA*Average age:*34 years (10-15 years older than in conventional osteosarcoma)*Histo:*chondroblastic predominance (~50%), osteoblastic predominance (~25%); better differentiated (grade 2 or 3) than conventional osteosarcoma (grade 3 or 4) ● simulating periodontal disease: rapidly enlarging mass, lump, swelling ● paresthesia (if inferior alveolar nerve involved) ● painful / loose teeth, bleeding gum Location:body of mandible (lytic), alveolar ridge of maxilla (sclerotic), maxillary antrum√ osteolytic / osteoblastic / mixed pattern√ osteoid matrix (60-80%)√ aggressive periosteal reaction for mandibular lesion√ soft-tissue mass (100%)√ opacification of maxillary sinus (frequent in maxillary lesions)*Prognosis:*40% 5-year survival rate (lower probability of metastases, lower grade)*DDx:*metastatic disease (lung, breast, kidney), multiple myeloma, direct invasion by contiguous tumor from oral cavity, Ewing sarcoma, primary lymphoma of bone, chondrosarcoma, fibrosarcoma, acute osteomyelitis, ameloblastoma, Langerhans cell histiocytosis, giant cell reparative granuloma, "brown tumor" of HPT

Notes:

Osteosarcomatosis =MULTIFOCAL OSTEOSARCOMA=MULTIPLE SCLEROTIC OSTEOSARCOMA*Etiology:*(a) multicentric type of osteosarcoma(b) multiple metastatic bone lesions*Classification* (Amstutz): TypeImultiple synchronous bone lesions occurring within 5 months + patient ≤18 years of ageTypeIImultiple synchronous bone lesions occurring within 5 months + patient >18 years of ageTypeIIIaearly metachronous metastatic osteosarcoma occurring 5 to 24 months after diagnosisType IIIblate metachronous metastatic osteosarcoma occurring >24 months after diagnosis*Age:*Amstutz type I = 4-18 (mean 11) yearsAmstutz type II = 19-63 (mean 30) yearsSite:metaphysis of long bones; may extend into epiphyseal plate / begin in epiphysis√ multicentric simultaneously appearing lesions with a radiologically dominant tumor (97%)√ smaller lesions are densely opaque (osteoblastic)√ lesions bilateral + symmetrical√ early: bone islands√ late: entire metaphysis fills with sclerotic lesions breaking through cortex√ lesions are of same size√ lung metastases (62%)*Prognosis:*uniformly poor with mean survival of 12 (range, 6-37) months*DDx:*heavy metal poisoning, sclerosing osteitis, progressive diaphyseal dysplasia, melorheostosis, osteopoikilosis, bone infarction, osteopetrosis

Notes:

Parosteal Osteosarcoma *Frequency:*4% of all osteosarcomas; 65% of all juxtacortical osteosarcomas*Origin:*outer layer of periosteum; slowly growing lesion with fulminating course if tumor reaches medullary canal*Histo:*low-grade lesion with higher-grade regions (22-64%), invasion of medullary canal (8-59%); fibrous stroma + extensive osteoid with small foci of cartilage*Age:*peak age 38 years (range of 12-58 years); 50% > age 30 (for central osteosarcoma 75% < age 30); M:F = 2:3Location:posterior aspect of distal femur (50-65%), either end of tibia, proximal humerus, fibula, rare in other long bonesSite:metaphysis (80-90%) ● palpable mass √ large lobulated "cauliflower-like" homogeneous ossific mass extending away from cortex√ "string sign" = initially fine radiolucent line separating tumor mass from cortex (30-40%)√ tumor stalk (= attachment to cortex) grows with tumor obliterating the radiolucent cleavage plane√ cortical thickening without aggressive periosteal reaction√ tumor periphery less dense than center (DDx: myositis ossificans with periphery more dense than center + without attachment to cortex)√ large soft-tissue component with osseous + cartilaginous elements*Prognosis:*80-90% 5- and 10-year survival rates (best prognosis of all osteosarcomas)*DDx:*osteochondroma, myositis ossificans, juxtacortical hematoma, extraosseous osteosarcoma

Notes:

Periosteal Osteosarcoma *Origin:*deep layer of periosteum*Histo:*intermediate-grade lesion; highly chondroblastic lesion with smaller areas of osteoid formation*Age:*peak 10-20 years (range of 13-70 years)Location:femur and tibia (85-95%), ulna and humerus (5-10%)Site:diaphysis / metadiaphysis of long bone; limited to periphery of cortex with normal endosteal margin + medullary canal (resembles parosteal sarcoma)√ tumor 7-12 cm in length, 2-4 cm in width, involving 50% of osseous circumference√ tumor base closely attached to cortex over entire extent of tumor√ tumor lies in apparent depression on bone surface causing scalloped surface of thickened diaphyseal cortex√ short spicules of new bone perpendicular to shaft extending into broad-based elliptical soft-tissue mass√ solid (cortical thickening) / aggressive periosteal reaction (Codman triangle) at upper and lower margins of lesion√ NO cortical destruction / medullary cavity invasion√ chondroblastic areas of low attenuation on CT, hypointense on T1WI, very hyperintense on T2WI*Prognosis:*80-90% cure rate (better prognosis than central osteosarcoma with 50% 5-year survival but worse than parosteal osteosarcoma)*DDx:*juxtacortical chondrosarcoma

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Secondary Osteosarcoma *Cause:*malignant transformation within benign process(1)Paget disease (67-90%)◊0.2-7.5% of patients with Paget disease develop osteosarcoma dependent on extent of disease(2)sequela of irradiation (6-22%) 2-40 years ago (malignant fibrous histiocytoma most common; fibrosarcoma 3rd most common)◊0.02-4% of patients with radiation therapy develop osteosarcoma related to exposure dose (usually >1,000 cGy)(3)osteonecrosis, fibrous dysplasia, metallic implants, osteogenesis imperfecta, chronic osteomyelitis, retinoblastoma (familial bilateral type)*Path:*high-grade anaplastic tissue with little / no mineralization*Age:*middle-aged / late adulthood√ aggressive bone destruction in area of preexisting condition associated with large soft-tissue mass*Prognosis:*<5% 5-year survival rate

Notes:

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Small-cell Osteosarcoma *Age:*similar to conventional osteosarcoma; M:F = 1:1Histo:small round blue cells (similar to Ewing sarcoma) lacking cellular uniformity and consistently producing fine reticular osteoidLocation:distal femurSite:metaphysis with frequent extension into epiphysis; diaphysis (in 15%)√ predominantly permeative lytic medullary lesion√ cortical breakthrough√ aggressive periosteal reaction√ associated soft-tissue mass*Prognosis:*extremely poor

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Telangiectatic Osteosarcoma =MALIGNANT BONE ANEURYSM*Frequency:*4-11% of all osteosarcomas*Age:*3-67 (mean 20) years; M:F = 3:2*Path:*malignant osteoid-forming sarcoma of bone with large blood-filled vascular channels*Histo:*hemorrhagic + cystic + necrotic spaces occupying >90% of the lesion before therapy; blood-filled cavernous vessels lined with osteoclastic giant cellsLocation:about knee (62%); distal femur (48%), proximal tibia (14%), proximal humerus (16%)Site:metaphysis (90%); extension into epiphysis (87%)√ geographic bone destruction with a wide zone of transition√ marked aneurysmal expansion of bone (19%)√ fluid-fluid levels (90%)√ nodular calcific foci of osteoid (61-81%)√ "doughnut sign" = peripherally increased uptake with central photopenia on bone scan*DDx:*aneurysmal bone cyst (no enhancing rim of viable tumor along lesion periphery)

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OXALOSIS

Rare inborn error of metabolism *Etiology:*excessive amounts of oxalic acid combine with calcium and deposit throughout body (kidneys, soft tissue, bone) ● hyperoxaluria = urinary excretion of oxalic acid >50 mg/ day ● progressive renal failure√ osteoporosis = cystic rarefaction + sclerotic margins in tubular bones on metaphyseal side, may extend throughout diaphysis√ erosions on concave side of metaphysis near epiphysis (DDx: hyperparathyroidism)√ bone-within-bone appearance of spine√ nephrocalcinosis (2° HPT: subperiosteal resorption, rugger jersey spine, sclerotic metaphyseal bands)*Cx:*pathologic fractures

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PACHYDERMOPERIOSTOSIS
=OSTEODERMOPATHIA HYPERTROPHICANS (TOURAINE-SOLENTE-GOLE) = PRIMARY HYPERTROPHIC OSTEOARTHROPATHYAutosomal dominant
*Age:*3-38 years with progression into late 20s / 30s;M >> F ● large skin folds of face + scalpLocation:epiphyses + diametaphyseal region of tubular bones; distal third of bones of legs + forearms (early); distal phalanges rarely involved√ enlargement of paranasal sinuses√ irregular periosteal proliferation of phalanges + distal long bones (hand + feet) beginning in epiphyseal region at tendon / ligament insertions√ thick cortex, BUT NO narrowing of medulla√ clubbing√ may have acroosteolysis*Prognosis:*progression ceases after several years*DDx:*pulmonary osteoarthropathy, thyroid acropachy

Notes:

PAGET DISEASE

=OSTEITIS DEFORMANS = multifocal chronic skeletal disease due to chronic paramyxoviral infection*Prevalence:*3% of individuals >40 years; 10% of persons >80 years; higher prevalence in northern latitudes*Age:*>55 years (in 3%); >85 years (in 10%); unusual <40 years; M:F = 2:1*Histo:*increased resorption + increased bone formation; newly formed bone is abnormally soft with disorganized trabecular pattern ("mosaic pattern") causing deformity(a)ACTIVE PHASE = OSTEOLYTIC PHASE= aggressive bone resorption with lytic lesions, replacement of hematopoietic bone marrow by fibrous connective tissue with numerous large vascular channels (b)INACTIVE PHASE = QUIESCENT PHASE= decreased bone turnover with skeletal sclerosis + cortical accretion + loss of excessive vascularity (c)MIXED PATTERN (common)= lytic + sclerotic phases usually coexist ● asymptomatic (1/5) ● fatigue ● enlarged hat size ● peripheral nerve compression ● neurologic disorders from compression of brainstem (basilar invagination) ● hearing loss, blindness, facial palsy (narrowing of neural foramina) - rare ● pain from(a)primary disease process - rare(b)pathologic fracture(c)malignant transformation(d)degenerative joint disease / rheumatic disorder aggravated by skeletal deformity ● local hyperthermia of overlying skin ● high-output congestive heart failure from markedly increased perfusion (rare) ● increased alkaline phosphatase (increased bone formation) ● hydroxyproline increased (increased bone resorption) ● normal serum calcium + phosphorusSites:usually polyostotic + asymmetric; pelvis (75%) > lumbar spine > thoracic spine > proximal femur > calvarium > scapula > distal femur > proximal tibia > proximal humerusSensitivity:scintigraphy + radiography (60%)scintigraphy only (27%)radiography only (13%)√ thick coarse trabeculae + cortical thickening√ cystlike areas (fat-filled marrow cavity / blood-filled sinusoids / liquefactive degeneration + necrosis of proliferating fibrous tissue) @ Skull (involvement in 29-65%) √ inner + outer table involved√ diploic widening√ osteoporosis circumscripta = well-defined lysis, most commonly in calvarium anteriorly, occasionally in long bones (destructive active stage)√ "cotton wool" appearance = mixed lytic + blastic pattern of thickened calvarium (late stage)√ basilar invagination with encroachment on foramen magnum√ deossification + sclerosis in maxilla√ sclerosis of base of skull@Long bones (almost invariable at end of bone; rarely in diaphysis)√ "candle flame" / "blade of grass" lysis = advancing tip of V-shaped lytic defect in diaphysis of long bone originating in subarticular site (CHARACTERISTIC)√ lateral curvature of femur, anterior curvature of tibia (commonly resulting in fracture)@Small / flat bones√ bubbly destruction + periosteal successive layering@ Pelvis √ thickened trabeculae in sacrum, ilium; rarefaction in central portion of ilium√ thickening of iliopectineal line√ acetabular protrusion (DDx: metastatic disease not deforming) + secondary degenerative joint disease@ Spine (upper cervical, low dorsal, midlumbar) √ lytic / coarse trabeculations at periphery of bone√ "picture-frame vertebra" = bone-within-bone appearance = enlarged square vertebral body with reinforced peripheral trabeculae + radiolucent inner aspect, typically in lumbar spine√ "ivory vertebra" = blastic vertebra with increased density√ ossification of spinal ligaments, paravertebral soft tissue, disk spaces Bone scan: √ usually markedly increased uptake (symptomatic lesions strikingly positive)√ normal scan in some sclerotic burned-out lesions√ marginal uptake in lytic lesions√ enlargement + deformity of bonesBone marrow scan: √ sulfur colloid bone marrow uptake is decreased (marrow replacement by cellular fibrovascular tissue)MR: √ hypointense area / area of signal void on T1WI + T2WI (cortical thickening, coarse trabeculation)√ widening of bone√ reduction in size + signal intensity of medullary cavity (replacement of high-signal-intensity fatty marrow by increased medullary bone formation)√ focal areas of higher signal intensity than fatty marrow (= cystlike fat-filled marrow spaces)√ areas of decreased signal intensity within marrow on T1WI + increased intensity on T2WI (= fibrovascular tissue resembling granulation tissue) *Cx:*(1)Associated neoplasia (0.7-20%)(a)sarcomatous transformation into osteosarcoma (22-90%), fibrosarcoma /malignant fibrous histiocytoma (29-51%), chondrosarcoma (1-15%)√ osteolysis in pelvis, femur, humerus(b)giant cell tumor (3-10%)√ lytic expansile lesion in skull, facial bones(c)lymphoma, plasma cell myeloma(2)Fracture(a)"banana fracture" = tiny horizontal cortical infractions on convex surfaces of lower extremity long bones (lateral bowing of femur, anterior bowing of tibia);(b)compression fractures of vertebrae (soft bone despite increased density)(3)Extradural spinal block (bone-forming phase / compression fractures) with neurologic deficits(4)Early-onset osteoarthritis*Rx:*calcitonin, diphosphonate, mithramycin *Detection of recurrence:* (a)in 1/3 detected by bone scan(b)in 1/3 detected by biomarkers (alkaline phosphatase, urine hydroxyproline)(c)in 1/3 by scan + biomarkers simultaneously√ diffuse (most common) / focal increase in tracer uptake√ extension of uptake beyond boundaries of initial lesion*DDx:*Osteosclerotic metastases, Hodgkin disease, vertebral hemangioma

Notes:

PARAOSTEOARTHROPATHY

=HETEROTOPIC BONE FORMATION = ECTOPIC OSSIFICATION = MYOSITIS OSSIFICANSCommon complication following surgical manipulation, total hip replacement (62%) and chronic immobilization (spinal cord injury / neuromuscular disorders) *Mechanism:*pluripotent mesenchymal cell lays down matrix for formation of heterotopic bone similar to endosteal bone*Causes:*para- / quadriplegia (40-50%), myelomeningocele, poliomyelitis, severe head injury, cerebrovascular disease, CNS infections (tetanus, rabies), surgery (commonly following total hip replacement) *Evolution:*calcifications seen 4-10 weeks following insult; progression for 6-14 months; trabeculations by 2-3 months; stable lamellar bone ankylosis in 5% by 12-18 months √ largest quantity of calcifications around joints, especially hip, along fascial planes√ disuse osteoporosis of lower extremities√ renal calculi (elevation of serum calcium levels) *Radiographic grading system* (Brooker): 0no soft-tissue ossificationIseparate small foci of ossificationII>1 cm gap between opposing bone surfaces of heterotopic ossificationsIII<1 cm gap between opposing bone surfacesIVbridging ossification Bone scan: √ tracer accumulation in ectopic bone√ assessment of maturity for optimal time of surgical resection (indicated by same amount of uptake as normal bone)*Cx:*Ankylosis in 5%*Rx:*1000-2000 rad within 4 days following surgical removal

Notes:

PHENYLKETONURIA

High incidence of x-ray changes in phenylalanine-restricted infants: √ metaphyseal cupping of long bones (30-50%), especially wrist √ calcific spicules extending vertically from metaphysis into epiphyseal cartilage (DDx to rickets) √ sclerotic metaphyseal margins √ osteoporosis √ delayed skeletal maturation *DDx:* Homocystinuria

Notes:

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PHOSPHORUS POISONING

Etiology:(1)ingestion of metallic phosphorus (yellow phosphorus)(2)treatment of rachitis or TB with phosphorized cod liver oilLocation:long tubular bones, ilium√ multiple transverse lines (intermittent treatment with phosphorus)√ lines disappear after some years

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PIERRE ROBIN SYNDROME

May be associated with: CHD, defects of eye and ear, hydrocephalus, microcephaly ● glossoptosis√ micrognathia = hypoplastic receding mandible√ arched ± cleft palate√ rib pseudarthrosis*Cx:*airway obstruction (relatively large tongue), aspiration

Notes:

PIGMENTED VILLONODULAR SYNOVITIS
=PVNS = benign highly vascular synovial proliferation*Cause:*frequent history of antecedent trauma*Histo:*(1)hyperplasia of undifferentiated connective tissue with multinucleated large cells ingesting hemosiderin / lipoid (foam / giant cells)(2)villonodular appearance of synovial membrane ± fibrosis(3)pressure erosion / invasion of adjoining bone*Age:*mainly 2nd-4th decade (range 12-68 years);50% <40 years; M < F ● hemorrhagic "chocolate" effusion without trauma ● insidious onset of swelling, pain of long duration ● decreased range of motion, joint lockingLocation:knee, ankle, hip, elbow, shoulder, tarsal + carpal joints; predominantly monarticular (DDx: degenerative arthritis)√ soft-tissue swelling around joint (effusion + synovial proliferation)√ dense soft-tissues (hemosiderin deposits)√ subchondral pressure erosion at margins of joint√ multiple sites of deossification appearing as cysts√ NO calcifications, osteoporosis, joint space narrowing (until late)MR: √ masses of synovial tissue in a joint with effusion√ scalloping / truncation of prefemoral fat pad√ predominantly low signal intensity on all sequences (due to presence of iron) is CHARACTERISTIC√ often heterogeneous low + high signal intensity on T2WI (hemosiderin deposits in masses + para-articular fat)*DDx:*hemosiderin deposits in other diseases (eg, rheumatoid arthritis)*Rx:*synovectomy, arthrodesis, arthroplasty, radiation*DDx:*Synovial sarcoma (solitary calcified mass outside joint); synovial hemangioma INTRA-ARTICULAR LOCALIZED NODULAR SYNOVITIS = synovial lining without hemosiderin

Notes:

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POLAND SYNDROME

May be associated with: aplasia of mamilla / breastAutosomal recessive √ unilateral absence of the sternocostal head of the pectoralis major muscle√ ipsilateral syndactyly + brachydactyly√ rib anomalies

Notes:

POLIOMYELITIS

√ osteoporosis √ soft-tissue calcification / ossification √ intervertebral disk calcification √ rib erosion commonly on superior margin of 3rd + 4th rib (secondary to pressure from scapula) √ "bamboo" spine (resembling ankylosing spondylitis) √ sacroiliac joint narrowing

Notes:

POPLITEAL CYST

=BAKER CYST = synovial cyst in the posterior aspect of knee joint communicating with posterior joint capsule*Prevalence:*19% in general orthopedic patients, 61% in patients with rheumatoid arthritis*Pathophysiology:* formed by escape of synovial effusion into one of the bursae; fluid trapped by one-way valvular mechanism (a)Bunsen-type valve = expanding cyst compresses the communicating channel(b)ball-type valve = ball composed of fibrin + cellular debris plugs the communication channel*Etiology:*(1)arthritis (rheumatoid arthritis most common)(2)internal derangement (meniscal / anterior cruciate ligament tears)(3)pigmented villonodular synovitis ● pseudothrombophlebitis syndrome (= pain + swelling in calf) ● cellulitis (after leakage / rupture)Location:(a)gastrocnemio-semimembranous bursa = posterior to gastrocnemius muscle at level of medial condyle(b)supralateral bursa = between lateral head of gastrocnemius muscle + distal end of biceps muscle superior to lateral condyle (uncommon)(c)popliteal bursa = beneath lateral meniscus + anterior to popliteal muscle (uncommon)√ communication with bursa (documented on arthrogram) √ hypointense collection on T1WI + hyperintense on T2WI*Types:* 1.Intact cyst√ smooth contour 2.Dissected cyst√ smooth contour extending along fascial planes (usually between gastrocnemius + soleus)3.Ruptured cyst√ leakage into calf tissues*DDx of other synovial cysts about the knee:* (1)Meniscal cyst (at lateral / medial side of joint line; associated with horizontal cleavage tears)(2)Tibiofibular cyst (at proximal tibiofibular joint which communicates with knee joint in 10%)(3)Cruciate cyst (surrounding anterior / posterior cruciate ligaments following ligamentous injury)

Notes:

PROGERIA
=HUTCHINSON-GILFORD SYNDROME=autosomal recessive inheritance; most commonly in populations with consanguineous marriages (Japanese, Jewish)
*Age:*shortly after adolescence; M:F = 1:1Characteristic habitus + stature: ● symmetric retardation of growth ● absent adolescent growth spurt ● dwarf with short stature + light body weight ● spindly extremities with stocky trunk ● beak-shaped nose + shallow orbitsPremature senescence: ● birdlike appearance ● graying of hair + premature baldness ● hyperpigmentation ● voice alteration ● diffuse arteriosclerosis ● bilateral cataracts ● osteoporosisScleroderma-like skin changes: ● atrophic skin + muscles ● circumscribed hyperkeratosis ● telangiectasia ● tight skin ● cutaneous ulcerations ● localized soft-tissue calcificationsEndocrine abnormalities: ● diabetes ● hypogonadism √ generalized osteoporosis@Skull √ thin cranial vault √ delayed sutural closure + wormian bones √ hypoplastic facial bones (maxilla + mandible)@Chest √ narrow thorax + slender ribs √ progressive resorption with fibrous replacement of outer portions of thinned clavicles (HALLMARK) √ coronary artery + heart valve calcifications with cardiac enlargement@Extremities & joints √ short + slender long bones √ coxa valga √ valgus of humeral head √ acroosteolysis of terminal phalanges (occasionally) √ flexion + extension deformities of toes (hallux valgus, pes planus) √ excessive degenerative joint disease of major + peripheral joints √ neurotrophic joint lesions (feet) √ widespread osteomyelitis + septic arthritis (hands, feet, limbs)@Soft tissue √ soft-tissue atrophy of extremities √ soft-tissue calcifications around bony prominences (ankle, wrist, elbow, knee) √ peripheral vascular calcifications = premature atherosclerosis*Prognosis:*most patients die in their 30s / 40s from complications of arteriosclerosis (myocardial infarction, stroke) or neoplasm (sarcoma, meningioma, thyroid carcinoma)*DDx:*Cockayne syndrome (mental retardation, retinal atrophy, deafness, family history)

Notes:

PSEUDOACHONDROPLASIA

- normal face + head√ limb shortening√ irregular epiphyses√ scoliosis√ coxa vara√ marked shortening of bones in hands + feet

Notes:

PSEUDOFRACTURES
=LOOSER LINES = LOOSER ZONES = OSTEOID SEAMS = MILKMAN SYNDROME = insufficiency stress fractures + nonunion (incomplete healing due to mineral deficiency)*Path:*area of unmineralized woven bone occurring at sites of mechanical stress / nutrient vessel entry*Associated with:* (1) Osteomalacia / rickets (2) Paget disease ("banana fracture") (3) Osteogenesis imperfecta tarda (4) Fibrous dysplasia (5) Organic renal disease in 1% (6) Renal tubular dysfunction (7) Congenital hypophosphatasia (8) Congenital hyperphosphatasia ("juvenile Paget disease") (9) Vitamin D malabsorption / deficiency (10) Neurofibromatosis *mnemonic:*
"POOF"**P**aget disease **O**steomalacia **O**steogenesis imperfecta **F**ibrous dysplasia Common locations: scapulae (axillary margin, lateral + superior margin), medial femoral neck + shaft, pubic + ischial rami, ribs, lesser trochanter, ischial tuberosity, proximal 1/3 of ulna, distal 1/3 of radius, phalanges, metatarsals, metacarpals, clavicle √ typically bilateral + symmetric at right angles to bone margin√ paralleled by marginal sclerosis in later stages√ healing fracture with little or no callus response√ 2-3 mm stripe of lucency at right angle to cortex (= osteoid seams formed within stress-induced infractions (PATHOGNOMONIC) + nonunion (= incomplete healing due to mineral deficiency)

Notes:

PSEUDOHYPOPARATHYROIDISM
=PHypoPT = congenital X-linked dominant abnormality with renal + skeletal resistance to PTH due to (1) end organ resistance (2) presence of antienzymes (3) defective hormone*May be associated with:*hyperparathyroidism due to hypocalcemia; F > M ● short obese stature ● mental retardation ● corneal + lenticular opacity ● abnormal dentition (hypoplasia, delayed eruption, excessive caries) ● hypocalcemia + hyperphosphatemia (resistant to PTH injection) ● normal levels of PTH√ brachydactyly in bones in which epiphysis appears latest (metacarpal, metatarsal bones I, IV, V) (75%)√ accelerated epiphyseal maturation resulting in dwarfism + coxa vara / valga√ multiple diaphyseal exostoses (occasionally)√ calcification of basal ganglia + dentate nucleus√ calcification / ossification of skin + subcutaneous tissue

	PHypoPT	PPHypoPT
√ calcification of basal ganglia	44%	8%
√ soft-tissue calcifications	55%	40%
√ metacarpal shortening (4 +5 always involved)	75%	90%
√ metatarsal shortening (3 +4 involved)	70%	99%

Notes:

PSEUDOPSEUDOHYPOPARATHYROIDISM

=PPHypoPT = different expression of same familial disturbance with identical clinical + radiographic features as pseudohypoparathyroidism ● short stature, round facies ● NO blood chemical changes (normal calcium + phosphorus) ● normal response to injection of PTH √ brachydactyly

		PHypoPT	PPHypoPT
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Notes:

PSORIATIC ARTHRITIS
Uncommon disease involving synovium + ligamentous attachments with propensity for sacroiliitis / spondylitis classified as seronegative spondyloarthropathy 6/c *Incidence:*<5% of patients with psoriasis (peripheral arthritis in 5%, sacroiliitis in 29%, peripheral arthritis + sacroiliitis in 10%)*Path:*synovial inflammation (less prominent than in rheumatoid arthritis) with early fibrosis of proliferative synovium; bony proliferation at joint margins / tendon insertions / subperiosteum*Types:* (1)true psoriatic arthritis (31%)(2)psoriatic arthritis resembling rheumatoid arthritis (38%)(3)concomitant rheumatoid + psoriatic arthritis (31%) ● skin rash precedes / develops simultaneously with onset of arthritis in 85%◊Arthritis antedates dermatological changes by an interval of up to 20 years! ● pitting, discoloration, hyperkeratosis, subungual separation, ridging of nails (in 80%) ● positive HLA-B27 in 80% ● negative rheumatoid factorLocation:widely variable distribution + asymmetry with involvement of lower + upper extremitiesdistinctive pattern: terminal interphalangeal joints, ray distribution, unilateral polyarticular asymmetrical distribution√ NO / minimal juxta-articular osteoporosis (early stage); frequent osteoporosis (later stages)√ periosteal reaction frequent @Hand + foot*Target area:*DIP, PIP, MCP√ "sausage digit" = soft-tissue swelling of entire digit√ asymmetrical destruction of distal interphalangeal joints (erosive polyarthritis) + osseous resorption√ bony ankylosis (10%)√ "pencil-in-cup" deformity = erosions with ill-defined margins + adjacent proliferation at periosteal new bone (CHARACTERISTIC)√ ivory phalanx = sclerosis of terminal phalanx (28%)√ destruction of interphalangeal joint of 1st toe with exuberant periosteal reaction + bony proliferation at distal phalangeal base (PATHOGNOMONIC)√ poorly defined diffuse new bone formation at attachment of Achilles tendon + plantar aponeurosis√ erosions at superior / posterior margin of calcaneus (20%)√ acroosteolysis (occasionally)@Axial skeleton√ "floating" osteophyte = large bulky vertically oriented asymmetrical paravertebral soft-tissue calcification involving the disk annulus (not endplates), separate from edges of vertebraeLocation:lower cervical, thoracic, upper lumbar spine√ squaring of vertebrae in lumbar region√ sacroiliitis (40%) = asymmetrical / unilateral sacroiliac joint widening, increased density, fusion√ apophyseal joint narrowing + sclerosis√ atlantoaxial subluxation + odontoid abnormalities *DDx:*(1)Reiter syndrome (affects only lower extremity)(2)Rheumatoid arthritis (bilaterally symmetric well-defined erosions, juxta-articular osteoporosis)

Notes:

PYKNODYSOSTOSIS
=autosomal recessive disease;probably variant of cleidocranial dysostosis*Age:*children; M:F = 2:1 ● dwarfism (resembling osteopetrosis) ● mental retardation (10%) ● widened hands + feet ● dystrophic nails ● yellowish discoloration of teeth ● characteristic facies (beaked nose, receding jaw)√ brachycephaly + platybasia√ wide cranial sutures, wormian bones√ thick skull base√ hypoplasia of mandible + obtuse mandibular angle√ hypoplasia + nonpneumatization of paranasal sinuses√ nonsegmentation of C1/2 and L5/S1√ generalized increased density of long bones with thickened cortices√ clavicular dysplasia√ hypoplastic tapered terminal tufts√ multiple spontaneous fractures *DDx:*(1)Osteopetrosis (no mandibular / skull abnormality, no phalangeal hypoplasia, no transverse metaphyseal bands, anemia, Erlenmeyer flask deformity; "bone-within-bone" appearance)(2)Cleidocranial dysostosis (no dense bones / terminal phalangeal hypoplasia, short stature)

Notes:

RADIATION INJURY TO BONE

*Pathogenesis:*vascular compromise with obliterative endarteritis + periarteritis followed by damage to osteoblasts with decreased matrix production (growing bone + periosteal new bone most sensitive)*Dose effects:* >300 rad:microscopic changes>400 rad:growth retardation<600-1200 rad:histological recovery retained>1200 rad:pronounced cellular damage to chondrocytes; bone marrow atrophy + cartilage degeneration after >6 months; vascular fibrosisA.BONE GROWTH DISTURBANCE √ growth plate widening in 1-2 months, often returning to normal by 6 months√ joint space widening after 8-10 months√ metaphyseal bowing√ ricketlike irregularity + fraying of metaphysis√ abnormal tubulation + premature fusion of physisB.RADIATION OSTEITIS=bone mottling due to osteopenia + coarse trabeculation + focally increased bone density √ osteopenia about 1 year after radiation√ periostitis√ increased fragility with sclerosis (= insufficiency fx)√ avascular necrosis√ osteoradionecrosisMR: √ increased intensity of spinal bone marrow on T1WI + T2WI corresponding to radiation port (fatty infiltration)*DDx:*recurrent malignancy, radiation-induced sarcoma, infectionC.BENIGN NEOPLASMMost likely in patients <2 years of age at treatment; with doses of 1600-6425 rads *Latent period:*1.5-14 years1.Exostosis = Osteochondroma2.OsteoblastomaD.MALIGNANT NEOPLASM=RADIATION-INDUCED SARCOMA*Latency period:*3-55 (average of 11-14) years*Minimum dose:*1,660-3,000 rad*Criteria:*(a)malignancy occurring within irradiated field(b)latency period of >5 years(c)histologic proof of sarcoma(d)microscopic evidence of altered histology of the original lesion*Histo:*1.Osteosarcoma (90%) = 4-11% of all osteogenic sarcomas2.Fibrosarcoma > chondrosarcoma > malignant fibrous histiocytoma ● pain, soft-tissue mass, rapid progression of lesion

Notes:

REFLEX SYMPATHETIC DYSTROPHY
=CAUSALGIA = SHOULDER-HAND SYNDROME = POSTTRAUMATIC OSTEOPOROSIS = SUDECK DYSTROPHY=serious + potentially disabling condition with poorly understood origin + cause*Etiology:* (1)Trauma in >50% (fracture, frostbite; may be trivial)affects 0.01% of all trauma patients(2)Idiopathic in 27% (immobilization, infection)(3)Myocardial ischemia in 6%(4)CNS disorders in 6%affects 12-21% of patients with hemiplegia(5)Discogenic disease in 5% ● burning pain, tenderness, allodynia, hyperpathia ● soft-tissue swelling ± pitting edema out of proportion to degree of injury ● dystrophic skin + nail changes ● sudomotor changes: hyperhidrosis + hypertrichosis ● vasomotor instability (Raynaud phenomenon, local vasoconstriction / -dilatation) ● end-stage (after 6-12 months): contractures, atrophy of skin + soft tissues Location:hands and feet distal to injury√ periarticular soft-tissue swelling√ patchy osteopenia (50%) as early as 2-3 weeks after onsets of symptoms (DDx: disuse osteopenia)√ generalized osteopenia = ground-glass appearance (endosteal + intracortical excavation; subperiosteal bone resorption; lysis of juxta-articular + subchondral bone)NUC (3-phase bone scan): √ increased flow + increased blood pool + increase in periarticular uptake on delayed images in affected part (60%)√ diminished flow / delayed uptake (15-20%)*Rx:*sympathetic block, a-/b-adrenergic blocking agents, nonsteroidal anti-inflammatory drugs, radiation therapy, hypnosis, acupuncture, acupressure, transcutaneous nerve stimulation, physiotherapy, calcitonin, corticosteroids, early mobilization

Notes:

REITER SYNDROME
=triad of (1) arthritis (2) uveitis (3) urethritis; 98% male*Types:* (1)endemic (venereal)(2)epidemic (postdysenteric) ● Hx of sexual exposure / diarrhea 3-11 days before onset of urethritis ● mucocutaneous lesions (keratosis blennorrhagia, balanitis circinata sicca) ● uveitis, conjunctivitis ● positive HLA-B27 in 76%Location:asymmetric mono- / pauciarticular√ polyarthritis√ articular soft-tissue swelling + joint space narrowing in 50% (particularly knees, ankles, feet)√ widening + inflammation of Achilles + patella tendons√ "fluffy" periosteal reaction (DISTINCTIVE) at metatarsal necks, proximal phalanges, calcaneal spur, tibia + fibula at ankle and knee√ juxta-articular osteoporosis (rare in acute stage) CHRONIC CHANGES ● recurrent joint attacks in a few cases√ calcaneal spur at insertion of plantar fascia + Achilles tendon√ periarticular deossification√ marginal erosions, loss of joint space√ bilateral sacroiliac changes indistinguishable from ankylosing / psoriatic spondylitis√ isolated osteophyte usually in thoracolumbar area, separated from vertebral body*Cx:*gastric ulcer + hemorrhage; aortic incompetence; heart block; amyloidosis

Notes:

RELAPSING POLYCHONDRITIS
=generalized recurring inflammation + destruction of cartilage in joints, ears, nose, larynx, airways*Etiology:*acquired metabolic disorder (? abnormal acid mucopolysaccharide metabolism / hypersensitivity / altered immunity*Histo:*loss of cytoplasm in chondrocytes; plasma cell + lymphocyte infiltration ● saddle-nose deformity ● swollen + tender ears, cauliflower ears ● hearing loss (obstruction of external auditory meatus) ● cough, hoarseness, dyspnea (collapse of trachea) ● arthralgia @Head√ calcification of pinna of ear@Chest√ ectasia + collapsibility with narrowing of trachea and mainstem bronchi√ generalized + localized emphysema √ aortic aneurysm (10%), mostly in ascending aorta, may be multiple / dissecting√ costochondritis@Bone√ periarticular osteoporosis√ erosive changes in carpal bones resembling rheumatoid arthritis√ soft-tissue swelling around joints + styloid process of ulna√ erosive irregularities in sacroiliac joints√ disk space erosion + increased density of articular plates*Rx:*corticosteroids

Notes:

RENAL OSTEODYSTROPHY
=constellation of musculoskeletal abnormalities that occur with chronic renal failure as a combination of(a) osteomalacia (adults) / rickets (children) (b) 2° HPT with osteitis cystica fibrosa + soft-tissue calcifications (c) osteosclerosis (d) soft-tissue + vascular calcifications *Classification:* (1)Glomerular form = acquired renal disease: chronic glomerulonephritis (common)(2)Tubular form = congenital renal osteodystrophy:1.Vitamin D-resistant rickets = hypophosphatemic rickets2.Fanconi syndrome = impaired resorption of glucose, phosphate, amino acids, bicarbonate, uric acid, sodium, water3.Renal tubular acidosis*Pathogenesis:* (a)Renal insufficiency causes a decrease in vitamin D conversion into the active $1,25(OH)_2D_3$ (done by 25-OH-D-1-a hydroxylase, which is exclusive to renal tissue mitochondria); vitamin D deficiency slows intestinal calcium absorption; *vitamin D resistance predominates* and calcium levels stay low (Ca x P product remains almost normal secondary to hyperphosphatemia); low calcium levels lead to OSTEOMALACIA; additional factors responsible for osteomalacia are inhibitors to calcification produced in the uremic state, aluminium toxicity, dysfunction of hepatic enzyme system(b)Renal insufficiency with diminished filtration results in phosphate retention; maintenance of Ca x P product lowers serum calcium directly, which in turn increases PTH production (2° HPT); *2° HPT predominates* associated with mild vitamin D resistance and leads to an increase in Ca x P product with SOFT-TISSUE CALCIFICATION in kidney, lung, joints, bursae, blood vessels, heart as well as OSTEITIS FIBROSA(c)Mixture of (a) and (b): increased serum phosphate inhibits vitamin D activation via feedback regulation ● phosphate retention ● hypocalcemia A.OSTEOPENIA (in 0-25-83%)=diminution in number of trabeculae + thickening of stressed trabeculae = increased trabecular pattern*Cause:* combined effect of(1)Osteomalacia (reduced bone mineralization due to acquired insensitivity to vitamin D / antivitamin D factor)(2)Osteitis fibrosa cystica (bone resorption)(3)Osteoporosis (decrease in bone quantity)Contributing factors: chronic metabolic acidosis, poor nutritional status, pre- and posttransplantation azotemia, use of steroids, hyperparathyroidism, low vitamin D levels *Cx:*fracture predisposition (lessened structural strength) with minor trauma / spontaneously; fracture prevalence increases with duration of hemodialysis + remains unchanged after renal transplantationSite:vertebral body (3-25%), pubic ramus, rib (5-25%)√ Milkman fracture / Looser zones (in 1%)√ metaphyseal fractures*Prognosis:*osteopenia may remain unchanged / worsen after renal transplantation + during hemodialysis B.RICKETS (children)*Cause:*in CRF normal vessels fail to develop orderly along cartilage columns in zone of provisional calcification; this results in disorganized proliferation of the zone of maturing + hypertrophying cartilage and disturbed endochondral calcificationLocation:most apparent in areas of rapid growth such as knee joints√ diffuse bone demineralization√ widening of growth plate√ irregular zone of provisional calcification√ metaphyseal cupping + fraying√ bowing of long bones, scoliosis√ diffuse concave impression at multiple vertebral endplates, basilar invagination√ slipped epiphysis (10%): capital femoral, proximal humerus, distal femur, distal radius, heads of metacarpals + metatarsals√ general delay in bone age C.SECONDARY HPT (in 6-66%)*Cause:*inability of kidneys to adequately excrete phosphate leads to hyperplasia of parathyroid chief cells (2° HPT); excess PTH affects the development of osteoclasts, osteoblasts, osteocytes ● hyperphosphatemia ● hypocalcemia ● increased PTH levels√ subperiosteal, cortical, subchondral, trabecular, endosteal, subligamentous bone resorption√ osteoclastoma = brown tumor = osteitis fibrosa cystica in 1.5-1.7% (due to PTH-stimulated osteoclastic activity; more common in 1° HPT)√ periosteal new-bone formation (8-25%)√ chondrocalcinosis (more common in 1° HPT) D.OSTEOSCLEROSIS (9-34%)One of the most common radiologic manifestations; most commonly with chronic glomerulonephritis; may be the sole manifestation of renal osteodystrophy √ diffuse chalky density: thoracolumbar spine in 60% (rugger jersey spine); also in pelvis, ribs, long bones, facial bones, base of skull (children)*Prognosis:*may increase / regress after renal transplantation E.SOFT-TISSUE CALCIFICATIONS(a)metastatic secondary to hyperphosphatemia (solubility product for calcium + phosphate $[Ca^{2+} \times PO_4^{-2}]$ exceeds 60-75 mg/dL in extracellular fluid), hypercalcemia, alkalosis with precipitation of calcium salts(b)dystrophic secondary to local tissue injuryLocation: (a)arterial (27-83%): in medial + intimal elastic tissueLocation:dorsal pedis a., forearm, hand, wrist, leg√ pipestem appearance without prominent luminal involvement(b)periarticular (0-52%): multifocal, frequently symmetric, may extend into adjacent joint ● chalky fluid / pastelike material ● inflammatory response in surrounding tenosynovial tissue√ discrete cloudlike dense areas√ fluid-fluid level in tumoral calcinosis*Prognosis:*often regresses with treatment(c)visceral (79%): heart, lung, stomach, kidney√ fluffy amorphous "tumoral" calcification *Rx:*1.Decrease of phosphorus absorption in bowel (in hyperphosphatemia)2.Vitamin D_3 administration (if vitamin D resistance predominates)3.Parathyroidectomy for 3° HPT (= autonomous HPT)

Congenital Renal Osteodystrophy

Notes:

Congenital Renal Osteodystrophy *Vitamin D-Resistant Rickets* =PHOSPHATE DIABETES = PRIMARY HYPOPHOSPHATEMIA = FAMILIAL HYPOPHOSPHATEMIC RICKETS =rare X-linked dominant disorder of renal tubular reabsorption characterized by(1)impaired resorption of phosphate in proximal renal tubule (due to defect in renal brush-border membrane)(2) inappropriately low synthesis of 1,25 dihydroxyvitamin D_3 [$1,25(OH)_2D_3$] in renal tubules resulting in decreased intestinal resorption of calcium + phosphate*Age:*<1 year ● hypophosphatemia + hyperphosphaturia ● elevated serum alkaline phosphatase ● normal plasma + urine calcium ● normal / low serum $1,25(OH)_2D_3$√ classic rachitic changes√ skeletal deformity, particularly bowed legs√ retarded bone age; dwarfism if untreated√ osteosclerosis / bone thickening (from overabundance of incompletely calcified matrix)*Rx:*phosphate infusion + large doses of vitamin D*DDx:*vitamin-D-deficient and -dependent rickets (absence of muscle weakness + seizures + tetany) ***Fanconi Syndrome*** Triad of (1) hyperphosphaturia (2) amino aciduria (3) renal glucosuria (normal blood glucose) *Etiology:*renal tubular defect√ rickets, osteomalacia, osteitis fibrosa, osteosclerosis*Prognosis:*functional renal impairment likely when bone changes occur*Rx:*large doses of vitamin D + alkalinization ***Renal Tubular Acidosis*** ● systemic acidosis, bone lesions√ rickets, osteomalacia, pseudofractures, nephrocalcinosis, osteitis fibrosa (rare)(a)Lightwood syndrome = salt-losing nephritis (self-limited form) ● NO nephrocalcinosis(b)Butler-Albright syndrome (severe form) ● nephrocalcinosis

Notes:

RHEUMATOID ARTHRITIS
=generalized connective tissue disease=Type III hypersensitivity = delayed hypersensitivity=immune complex disease (= formation of antigen-antibody complexes with complement fixation) *Cause:*genetic predisposition; ? reaction to antigen from Epstein-Barr virus / certain strains of E. coli*Age:*highest incidence 40-50 years; M:F = 1:3 if <40 years; M:F = 1:1 if >40 years*Pathogenesis:*injury to synovial endothelial cells; synovitis with synovial hypertrophy leads to impaired nutrition with chondronecrosis, joint narrowing, subluxation, and ankylosis *Diagnostic criteria* of American Rheumatism Association (at least 4 criteria should be present): (1) morning stiffness for ≥1 hour (2) swelling of ≥3 joints, particularly of wrist, metatarsophalangeal or proximal interphalangeal joints for >6 weeks (3) symmetric swelling (4) typical radiographic changes (5) rheumatoid nodules (6) positive rheumatoid factor ● morning stiffness ● fatigue, weight loss, anemia ● carpal tunnel syndrome ● rheumatoid factor (positive in 85-94%) = IgM-antibody= agglutination of sensitized sheep RBCs closely correlating with disease severity; false positive: normal (5%), asbestos workers with fibrosing alveolitis (25%), viral / bacterial / parasitic infection, other inflammatory diseases ● antinuclear antibodies (positive in many) ● LE cells (positive in some) ● positive latex flocculation test ● hormonal influence:(a)decrease in activity during pregnancy(b)men with RA have low testosterone levels Location:symmetric involvement of diarthrodial joints*Target areas:* all five MCP, PIP, interphalangeal joint of thumb, all wrist compartments (especially radiocarpal, inferior radioulnar, pisiform-triquetral joints); medial aspect of MTP + interphalangeal joints of foot (esp. great toe); earliest changes seen in 2nd + 3rd MCP, 3rd PIP EARLY SIGNS: √ fusiform periarticular soft-tissue swelling (result of effusion)√ regional osteoporosis (disuse + local hyperthermia)√ widened joint space√ marginal + central bone erosions (less common in large joints); site of first erosion is classically base of proximal phalanx of 4th finger√ changes in the ulnar styloid + distal radioulnar joint√ atlantoaxial subluxation >2.5 mm (in >6%)√ giant synovial cyst LATE SIGNS: √ diffuse loss of interosseous space√ flexion + extension contractures with ulnar subluxation + dislocation√ marked destruction + fractures of joint space√ extensive destruction of bone ends√ bony fusion√ elevation of humeral heads (tear / atrophy of rotator cuff)√ resorption of distal clavicle√ erosion of superior margins of posterior portions of ribs 3-5√ destruction + narrowing of disk spaces + irregular vertebral body outlines + absence of osteophytosis√ destruction of zygapophyseal joints without osteophyte formation√ resorption of spinous processes√ "stepladder appearance" of cervical spine due to subaxial subluxations√ protrusio acetabuli (from osteoporosis)√ synovial herniation + cysts (eg, popliteal cyst)√ calcaneal plantar spur*DDx:*SLE, psoriatic arthritis, seronegative spondylarthropathies EXTRA-ARTICULAR MANIFESTATIONS (76%) (a)**Felty syndrome** (<1%)=rheumatoid arthritis (present for >10 years) + splenomegaly + neutropenia*Age:*40-70 years; F > M; rare in Blacks ● rapid weight loss ● therapy refractory leg ulcers ● brown pigmentation over exposed surfaces of extremities(b)Sjögren syndrome (15%)=keratoconjunctivitis + xerostomia + rheumatoid arthritis(c)Pulmonary manifestations√ pleural effusion, mostly unilateral, without change for months, usually not associated with parenchymal disease√ interstitial fibrosis with lower lobe predominance√ rheumatoid nodules (30%): well-circumscribed, peripheral, with frequent cavitation√ Caplan syndrome (= hyperimmune reactivity to silica inhalation with rapidly developing multiple pulmonary nodules)√ pulmonary hypertension secondary to arteritis(d)Subcutaneous nodules(in 5-35% with active arthritis) over extensor surfaces of forearm + other pressure points (eg, olecranon) without calcifications (DDx to gout) (e)Cardiovascular involvement1.Pericarditis (20-50%)2.Myocarditis (arrhythmia, heart block)3.Aortitis (5%) of ascending aorta ± aortic valve insufficiency(f)Rheumatoid vasculitisMimics periarteritis nodosa ● polyneuropathy, cutaneous ulceration, gangrene, polymyopathy, myocardial / visceral infarction(g)Neurologic sequelae1.Distal neuropathy (related to vasculitis)2.Nerve entrapment (atlantoaxial subluxation, carpal tunnel syndrome, Baker cyst)(h)Lymphadenopathy (up to 25%)√ splenomegaly (1-5%)

Cystic Rheumatoid Arthritis Juvenile Rheumatoid Arthritis

Notes:

Cystic Rheumatoid Arthritis =intraosseous cystic lesions as dominant feature*Pathogenesis:*increased pressure in synovial space from joint effusion decompresses through microfractures of weakened marginal cortex into subarticular bone◊increase in size + extent of cysts correlates with increased level of activity + absence of synovial cysts*Age:*as above; M:F = 1:1 ● seronegative in 50%√ juxta-articular subcortical lytic lesions with well-defined sclerotic margins√ relative lack of cartilage loss, osteoporosis, joint disruption*DDx:*gout (presence of urate crystals), pigmented villonodular synovitis (monarticular)

Notes:

Juvenile Rheumatoid Arthritis =rheumatoid arthritis in patients <16 years of age;M < F*Classification:* (1)Juvenile-onset adult type (10%) ● IgM RA factor positive; age 8-9; poor prognosis√ erosive changes; perfuse periosteal reaction; hip disease with protrusio(2)Polyarthritis of the ankylosing spondylitic type ● iridocyclitis; boys age 9-11 years√ peripheral arthritis; fusion of greater trochanter; complete fusion of both hips; heel spur(3)**Still disease**
(a) systemic (b) polyarticular (c) pauciarticular + iridocyclitis (30%) ● fever, rash, lymphadenopathy, hepatosplenomegaly; pericarditis, dwarfism ● fatal kidney disease in 20%*Age:*2-4 and 8-11 years of age; M < FLocation:involvement of carpometacarpal joints ("squashed carpi" in adulthood), hind foot, hip (40-50%)√ periosteal reaction of phalanges; broadening of bones; accelerated bone maturation + early fusion (stunting of growth) ● morning stiffness, arthralgia ● subcutaneous nodules (10%) ● skin rash (50%) ● fever, lymphadenopathyLocation:early involvement of large joints (hips, knees, ankles, wrists, elbows); later of hands + feet√ radiologic signs similar to rheumatoid arthritis (except for involvement of large joints first, late onset of bony changes, more ankylosis, wide metaphyses)√ periarticular soft-tissue swelling√ thinning of joint cartilage√ large cystlike lesions removed from articular surface (invasion of bone by inflammatory pannus); rare in children√ articular erosions at ligamentous + tendinous insertion sites√ joint destruction may resemble neuropathic joints√ juxta-articular osteoporosis√ "balloon epiphyses" + "gracile bones" (epiphyseal overgrowth + early fusion with bone shortening secondary to hyperemia)@Hand / foot√ "rectangular" phalanges (periostitis + cortical thickening)√ ankylosis in carpal joints@Axial skeleton√ ankylosis of cervical spine (apophyseal joints), sacroiliac joints√ subluxation of atlantoaxial joint (66%)√ thoracic spinal compression fractures@Chest√ ribbon ribs√ pleural + pericardial effusions√ interstitial pulmonary lesions (simulating scleroderma, dermatomyositis)√ solitary pulmonary nodules, may cavitate *Prognosis:*complete recovery (30%); secondary amyloidosis

Notes:

RICKETS
=osteomalacia during enchondral bone growth*Age:*4-18 months*Histo:*zone of preparatory calcification does not form, heap up of maturing cartilage cells; failure of osteoid mineralization also in shafts so that osteoid production elevates periosteum ● irritability, bone pain, tenderness ● craniotabes ● rachitic rosary ● bowed legs ● delayed dentition ● swelling of wrists + anklesLocation:metaphyses of long bones subjected to stress are particularly involved (wrists, ankles, knees)√ poorly mineralized epiphyseal centers with delayed appearance√ irregular widened epiphyseal plates (increased osteoid)√ increase in distance between end of shaft and epiphyseal center√ cupping + fraying of metaphysis with threadlike shadows into epiphyseal cartilage (weight-bearing bones)√ cortical spurs projecting at right angles to metaphysis√ coarse trabeculation (NO ground-glass pattern as in scurvy)√ periosteal reaction may be present√ deformities common (bowing of soft diaphysis, molding of epiphysis, fractures)√ bowing of long bones√ frontal bossing*mnemonic:*"RICKETS"**R**eaction of periosteum may occur **I**ndistinct cortex **C**oarse trabeculation **K**nees + wrists + ankles mainly affected **E**piphyseal plates widened + irregular **T**remendous metaphysis (fraying, splaying, cupping) **S**pur (metaphyseal)

Causes Of Rickets Classification Of Rickets

Notes:

Causes Of Rickets I.*ABNORMALITY IN VITAMIN D METABOLISM*
Associated with reactive hyperparathyroidismA.Vitamin D deficiency(a)Dietary lack of vitamin D= famine osteomalacia(b)Lack of sunshine exposure(c)Malabsorption of vitamin D=gastroenterogenous rickets1.pancreatitis + biliary tract disease2.steatorrhea, celiac disease, postgastrectomy3.inflammatory bowel diseaseB.Defective conversion of vitamin D to 25-OH-cholecalciferol in liver1.Liver disease2.Anticonvulsant drug therapy (= induction of hepatic enzymes that accelerate degradation of biologically active vitamin D metabolites)C.Defective conversion of 25-OH-D3 to 1,25-OH-D3 in kidney1.Chronic renal failure = renal osteodystrophy2.Vitamin D-dependent rickets = autosomal recessive enzyme defect of 1-OHase II.*ABNORMALITY IN PHOSPHATE METABOLISM*
not associated with hyperparathyroidism secondary to normal serum calciumA.Phosphate deficiency1.Intestinal malabsorption of phosphates2.Ingestion of aluminum salts [$Al(OH)_2$] forming insoluble complexes with phosphate3.Low phosphate feeding in prematurely born infants4.Severe malabsorption state5.Parenteral hyperalimentationB.Disorders of renal tubular reabsorption of phosphate1.Renal tubular acidosis (renal loss of alkali)2.deToni-Debré-Fanconi syndrome = hypophosphatemia, glucosuria, aminoaciduria3.Vitamin D-resistant rickets4.Cystinosis5.Tyrosinosis6.Lowe syndromeC.Hypophosphatemia with nonendocrine tumors=Oncogenic rickets = elaboration of humeral substance which inhibits tubular reabsorption of phosphates1.Sclerosing hemangioma2.Hemangiopericytoma3.Ossifying mesenchymal tumor4.Nonossifying fibromaD.Hypophosphatasia III. *CALCIUM DEFICIENCY* 1.Dietary rickets = milk-free diet (extremely rare)2.Malabsorption3.Consumption of substances forming chelates with calcium

Notes:

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Classification Of Rickets I.Primary vitamin D-deficiency ricketsII.Gastrointestinal malabsorptionA.Partial gastrectomyB.Small intestinal disease: gluten-sensitiveenteropathy / regional enteritisC.Hepatobiliary disease: chronic biliary obstruction / biliary cirrhosisD.Pancreatic disease: chronic pancreatitisIII.Primary hypophosphatemia; vitamin D-deficiency ricketsIV.Renal diseaseA.Chronic renal failureB.Renal tubular disorders: renal tubular acidosisC.Multiple renal defectsV.Hypophosphatasia + pseudohypophosphatasiaVI.Fibrogenesis imperfecta osseumVII.Axial osteomalaciaVIII.MiscellaneousHypoparathyroidism, hyperparathyroidism, thyrotoxicosis, osteoporosis, Paget disease, fluoride ingestion, ureterosigmoidostomy, neurofibromatosis, osteopetrosis, macroglobulinemia, malignancy

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Notes:

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ROTATOR CUFF LESIONS

SUBACROMIAL PAIN SYNDROME (1)Impingement syndrome(2)Rotator cuff tendinitis(3)Degeneration without impingement(4)Shoulder instability with secondary impingement(5)Instability without impingement

Impingement Syndrome Glenohumeral Instability Rotator Cuff Tear Subacromial-Subdeltoid Bursitis Supraspinatus Tendinopathy / Tendinosis

Notes:

Impingement Syndrome =lateral shoulder pain with abduction; common cause of rotator cuff tears; NOT radiographic diagnosis*Age:*lifelong process; 1st stage <25 years; 2nd stage 25-40 years; complete rotator cuff tear >40 years*Pathophysiology:* movement of humerus impinges rotator cuff tendons against coracoacromial arch resulting in microtrauma, which causes inflammation of subacromial bursa (= fibrous thickening of subacromial bursa) / rotator cuff (critical zone of rotator cuff = supraspinatus tendon 2 cm from its attachment to humerus) ◊Impingement pathophysiology may be secondary to primary instability!*Impingement anatomy:* narrowing of subacromial space secondary to (1)acquired degenerative subacromial osteophyte / enthesophyte from(a)bony outgrowth along coracoacromial ligament(b)acromioclavicular joint osteoarthritis(2)congenital subacromial hook of anterior acromion(= subacromial spur) ◊Impingement syndrome may exist without impingement anatomy! ● painful arc of motion√ subacromial enthesophyte√ alteration in acromial shape + orientation√ thickening of coracoacromial ligament *Cx:*(1)partial / complete tear (may be precipitated by acute traumatic event on preexisting degenerative changes)(2)cuff tendinitis / degenerative tendinosis*Dx:*Lidocaine impingement test (= subacromial lidocaine injection relieves pain)*Rx:*acromioplasty (= removal of a portion of the acromion), removal of subacromial osteophytes, removal / lysis / débridement of coracoacromial ligament, resection of distal clavicle, removal of acromioclavicular joint osteophytes

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Glenohumeral Instability Glenohumeral stability is dependent on a functional anatomic unit (= anterior capsular mechanism) formed by: glenoid labrum, joint capsule, superior + middle + anteroinferior + posteroinferior glenohumeral ligaments, coracohumeral ligament, subscapularis tendon, rotator cuff *Age:*<35 years*Frequency:*acute, recurrent, fixed*Cause:*traumatic, microtraumatic, atraumaticDirection:anterior > multidirectional > inferior > posteriorType of lesions: labral abnormalities (compression, avulsion, shearing), capsular / ligamentous tear / avulsion *Associated lesions:* Hill-Sachs fracture, trough line fracture, glenoid fracture, labral cyst Normal clefts may exist within labrum!False positive for labral separation: (1)Articular cartilage deep to labrum(2)Glenohumeral ligaments passing adjacent to labrum

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Notes:

Rotator Cuff Tear *Etiology:*(1)Attritional change + tendon degeneration due to aging, repeated microtrauma as a result of impingement between humeral head + coracoacromial arch, overuse of shoulder from professional / athletic activities(2)Acute trauma (rare)*Age:*most commonly >50 yearsLocation:"critical zone" of supraspinatus tendon 1 cm medial to tendon attachment (area of relative hypovascularity) *Classification:* EXTENT OF TEAR (a)incomplete rupture = **partial tear** involves either bursal or synovial surface or remains intratendinous(b)complete rupture = **full-thickness** tear bridging subacromial bursa and glenohumeral joint-pure transverse tear-pure vertical / longitudinal tear-tear with retraction of tendon edges-global tear = **massive tear** / avulsion of cuff involving more than one of the tendonsTOPOGRAPHY OF TEAR (a)extent in frontal plane: nondisplaced, minimally displaced, dramatically displaced(b)extent in anterior direction: supraspinatus tendon + coracohumeral ligament + subscapularis tendon(c)extent in posterior direction: supraspinatus tendon + infraspinatus + teres minor tendon Arthrography (71-100% sensitive, 71-100% specific for combined full + partial thickness tears) √ opacification of subacromial-subdeltoid bursa MR (41-100% sensitive and 79-100% specific for combined full + partial thickness tears): √ discontinuity of cuff with retraction of musculotendinous junction√ focal / generalized intense / markedly increased signal intensity on T2WI (= fluid within cuff defect) in <50%√ fluid within subacromial-subdeltoid bursa (MOST SENSITIVE)√ low / moderate signal intensity on T2WI (= severely degenerated tendon, intact bursal / synovial surface, granulation / scar tissue filling the region of torn tendinous fibers)√ cuff defect with contour irregularity√ abrupt change in the signal character at boundary of the lesion√ supraspinatus muscle atrophy (MOST SPECIFIC)PITFALLS: √ hyperintense focus in distal supraspinatus tendon√ gray signal isointense to muscle on all pulse sequences(a)partial volume averaging with superior + lateral infraspinatus tendon(b)vascular "watershed" area(c)magic angle effect = orientation of collagen fibers at 55° relative to main magnetic field√ hyperintense focus within rotator cuff on T2WI(a)partial volume averaging with fluid in biceps tendon sheath / subscapularis bursa(b)partial volume averaging with fat of peribursal fat(c)motion artifacts: respiration, vascular pulsation, patient movement√ fatty atrophy of muscle(a)impingement of axillary / suprascapular nn. = quadrilateral space syndrome US (scans in hyperextended position, 75-100% sensitive, 43-97% specific, 65-95% negative predictive value, 55-75% positive predictive value): √ nonvisualization of rotator cuff (large tear), most reliable sign√ deltoid muscle directly on top of humeral head√ defect filled with hypoechoic thickened bursa + fat (with hypervascularity on color Doppler) between deltoid and humeral head√ focal nonvisualization of rotator cuff, reliable sign√ "naked tuberosity sign" = retracted tendon leaves a bare area of bone√ folding of bursal + peribursal fat tissue into focal defect√ discontinuity of rotator cuff filled with joint fluid / hypoechoic reactive tissue√ abrupt + sharply demarcated focal thinning√ small comma-shaped area of hyperechogenicity (small tear filled with granulation tissue / hypertrophied synovium)False negative:longitudinal tear, partial tearFalse positive:intra-articular biceps tendon, soft-tissue calcification, small scar / fibrous tissue

Notes:

Subacromial-Subdeltoid Bursitis common finding in rotator cuff tears √ peribursal fat totally / partially obliterated + replaced by low-signal-intensity tissue on all pulse sequences√ fluid accumulation within bursa

Notes:

Supraspinatus Tendinopathy / Tendinosis *Cause:*impingement, acute / chronic stress*Histo:*mucinous + myxoid degeneration√ increase in signal intensity in tendon on proton-density images without disruption of tendon√ tendinous enlargement + inhomogeneous signal pattern

Notes:

RUBELLA
=GERMAN MEASLES*Incidence:*endemic rate of 0.1%*Age:*infants (in utero transmission) ● neonatal dwarfism (intrauterine growth retardation) ● failure to thrive ● retinopathy, cataracts, deafness ● mental deficiency with encephalitis + microcephaly ● thrombocytopenic purpura, petechiae, anemia√ "celery-stalk" sign (50%) = metaphyseal irregular margins + coarsened trabeculae extending longitudinally from epiphysis; distal end of femur > proximal end of tibia, humerus√ no periosteal reaction√ hepatosplenomegaly + adenopathy√ pneumonitis@Cardiovascular:√ congenital heart disease (PDA)√ peripheral pulmonary artery stenosis√ necrosis of myocardium@CNS√ punctate / nodular calcifications√ porencephalic cysts√ occasionally microcephaly*Prognosis:*osseous manifestations disappear in 1-3 months*DDx:*(1)CMV(2)Congenital syphilis (diaphysitis + epiphysitis)(3)Toxoplasmosis

Notes:

RUBINSTEIN-TAYBI SYNDROME
=BROAD THUMB SYNDROME=rare sporadic syndrome without known chromosomal / biochemical markers; M:F = 1:1 ● small stature ● mental, motor, language retardation@Characteristic facies ● beaked / straight nose ± low nasal septum ● antimongoloid slant of palpebral fissures ● epicanthic folds ● broad fleshy nasal bridge ● high-arched palate ● dental abnormalities@Ophthalmologic findings ● strabismus, ptosis, refractive errors@Cutaneous findings ● keloids, hirsutism, simian crease ● flat capillary hemangioma on forehead / neck@Musculoskeletal findings√ short broad "spatulate" terminal phalanges of thumb and great toe ± angulation deformity (MOST CONSISTENT + CHARACTERISTIC FINDING)√ radial angulation of distal phalanx (50%) caused by trapezoid / delta shape of proximal phalanx√ tufted "mushroom-shaped" fingers + webbing√ thin tubular bones of hand + feet√ club feet√ skeletal maturation retardation√ dysplastic ribs√ spina bifida occulta√ scoliosis√ flat acetabular angle + flaring of ilia@Genitourinary tract anomalies√ bilateral renal duplication√ renal agenesis√ bifid ureter√ incomplete / delayed descent of testes@Cardiovascular abnormalities√ atrial septal defect√ patent ductus arteriosus√ coarctation of aorta√ valvular aortic stenosis√ pulmonic stenosisOB-US: √ decreased head circumference√ small for gestational age*Cx in infancy:*obstipation, feeding problems, recurrent upper respiratory infection

Notes:

SAPHO SYNDROME
=**S**ynovitis, **A**cne, **P**almoplantar pustulosis, **H**yperostosis, **O**steitis=PUSTULOTIC ARTHROSTEITIS=STERNOCLAVICULAR HYPEROSTOSIS=association between rheumatologic and cutaneous lesions (= seronegative spondyloarthropathy)Delay of several years can separate osseous from cutaneous lesions!*Etiology:*? variant of psoriasis*Age:*young to middle-aged adults; M:F = 1:1 ● palmoplantar pustulosis (52%) = chronic eruption of yellowish intradermal sterile pustules on palms + soles ● severe acne (15%) = acne fulminans, acne conglobata ● pain, soft-tissue swelling, limitation of motion at skeletal site of involvement@Sternoclavicular joint (70-90%)Site:insertion of costoclavicular ligament, clavicles, manubrium sterni√ osteolysis at beginning of disease√ hyperostosis + osteosclerosis√ arthritis + ankylosis of sternoclavicular joint@Axial skeleton (33%)√ osteosclerosis of one / more vertebral bodies√ disk space narrowing + endplate erosion√ paravertebral ossifications (mimicking marginal / nonmarginal syndesmophytes / massive bridging)√ unilateral sacroiliitis + associated osteosclerosis of adjacent iliac bone@Appendicular skeleton (30%)Location:distal femur, proximal tibia, fibula, humerus, radius, ulnaSite:metaphysis√ osteosclerosis / osteolysis + periosteal new bone formation with aggressive appearance@JointsLocation:knee, hip, ankle, DIP of hand√ synovial inflammation with juxta-articular osteoporosis (early)√ joint narrowing, marginal erosion, hyperostosis, enthesopathy (later)*Prognosis:*chronic course with unpredictable exacerbations + remissions*Rx:*nonsteroidal anti-inflammatory drugs, corticosteroids, analgesics, cyclosporine*DDx:*infectious osteomyelitis / spondylitis, osteosarcoma, Ewing sarcoma, metastasis, Paget disease, aseptic necrosis of clavicle

Notes:

SARCOIDOSIS

Osseous involvement in 6-15-20% ● unimpaired joint function, joints are rarely involvedLocation:small bones of hands + feet (middle + distal phalanges)√ reticulated "lacelike" trabecular pattern in metaphyseal ends of middle + distal phalanges, metacarpals, metatarsals√ well-defined cystlike lesions of varying size√ neuropathy-like destruction of terminal phalanges (DDx: scleroderma)√ phalangeal endosteal sclerosis + periosteal new bone (infrequent)√ vertebral involvement unusual: destructive lesions with sclerotic margin√ diffuse sclerosis of multiple vertebral bodies√ paravertebral soft-tissue mass (DDx: indistinguishable from tuberculosis)√ osteolytic changes in skull

Notes:

SCURVY
=BARLOW DISEASE = vitamin C deficiency with defective osteogenesis from abnormal osteoblast function*Age:*6-9 months (maternal vitamin C protects for first 6 months) ● irritability ● tenderness + weakness of lower limbs ● scorbutic rosary of ribs ● bleeding of gums (teething) ● legs drawn up + widely spread = pseudoparalysisLocation:distal femur (esp. medial side), proximal and distal tibia + fibula, distal radius + ulna, proximal humerus, sternal end of ribs√ Wimberger ring = sclerotic ring around epiphysis indicating loss of epiphyseal density√ white line of Fränkel = metaphyseal zone of preparatory calcification (DDx: lead / phosphorus poisoning, bismuth treatment, healing rickets)√ Trümmerfeld zone = radiolucent zone on shaft side of Fränkels white line (site of subepiphyseal infraction)√ Parke corner sign = subepiphyseal infraction / comminution resulting in mushrooming / cupping of epiphysis (DDx: syphilis, rickets)√ Pelkan spurs = metaphyseal spurs projecting at right angles to shaft axis√ "ground-glass" osteoporosis (CHARACTERISTIC)√ cortical thinning√ subperiosteal hematoma with calcification of elevated periosteum (sure radiographic sign of healing)√ soft-tissue edema (rare)

Notes:

SEPTIC ARTHRITIS
Organism: most often due to S. aureus; Gonorrhea (indistinguishable from tuberculous arthritis, but more rapid); Brucellar arthritis (indistinguishable from tuberculosis, slow infection); Salmonella (commonly associated with sickle cell disease / Gaucher disease) (a)<4 years of age:Streptococcus pyogenes, S. aureus, Haemophilus influenzae(b)>4 years of age:S. aureus(c)>10 years of age:S. aureus, Neisseria gonorrhoeaeLocation:lower extremity (75%) with hip + knee in 90% ● pain, limp, pseudoparalysis ● warmth, swelling ● septic clinical picture ● bacteremia, leukocytosisACUTE SIGNS: √ initial radiographs frequently normal√ soft-tissue swelling (first sign secondary to local hyperemia + edema)√ joint distension (effusion) ± subluxation of hip and humerus in children√ joint space narrowing = rapid development of destruction of articular cartilage (not in tuberculous arthritis)SUBACUTE SIGNS after 8-10 days: √ small erosions in articular cortex / loss of entire cortical outline (marginal erosions in tuberculosis)√ reactive bone sclerosis in underlying bone√ subchondral bone destruction (by synovial proliferation)√ defective reparation / ankylosis (if entire cartilage is destroyed)√ local bone atrophy (immobility)√ metaphyseal bone destruction (if osteomyelitis is source of septic joint)*Dx:*prompt arthrocentesis + blood culture*Cx:*(1)bone growth disturbance (lengthening, shortening, angulation)(2)chronic degenerative arthritis(3)ankylosis(4)osteonecrosis

Notes:

SHIN SPLINTS
=SHIN SORENESS = MEDIAL TIBIAL STRESS SYNDROME = SOLEUS SYNDROME=nonspecific term describing exertional lower leg pain*Incidence:*75% of exertional leg pain*Cause:*? atypical stress fracture, traction periostitis, compartment syndrome ● diffuse tenderness along posteromedial tibia in its middle to distal aspectLocation:posterior / posteromedial tibial cortex Plain radiographs: √ normal / longitudinal periosteal new boneBone scintigraphy: √ normal radionuclide angiogram + blood-pool phase (DDx to stress fracture)√ linear longitudinal uptake on delayed imagesMR: √ marrow edema / hemorrhage√ periosteal fluid

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SHORT-RIB POLYDACTYLY SYNDROME
=group of autosomal recessive disorders characterized by short limb dysplasia, constricted thorax, postaxial polydactyly (on ulnar / fibular side)TYPE I= SALDINO-NOONAN SYNDROMETYPE II= MAJEWSKI TYPETYPE III= NAUMOFF TYPETYPE BEEMER√ severe micromelia√ pointed femurs at both ends (type I); widened metaphyses (type III)√ narrow thorax√ extremely short horizontally oriented ribs√ distorted underossified vertebral bodies + incomplete coronal clefts√ polydactyly√ cleft lip / palate*Prognosis:*uniformly lethal

Notes:

SICKLE CELL DISEASE
Abnormal hemoglobins: HbS =DNA mutation substituting glutamic acid in position 6 on b-chain with valineHbC =DNA mutation substituting glutamic acid in position 6 on b-chain with lysine(a)homozygous = HbSS = sickle cell anemia(b)heterozygous = HbSA = sickling trait but no anemia(c)heterozygous variants:-HbSC (less severe form)-HbS b-thalassemia anemia (seen occasionally)*Incidence:*8-13% of American Blacks carry sickling factor (HbS); 1:40 with sickle cell trait will manifest sickle cell anemia (HbSS); 1:120 with sickle cell trait will manifest HbSC disease*Pathogenesis:* altered shape + plasticity of RBCs under lowered oxygen tension lead to increased blood viscosity, stasis, "log jam" occlusion of small blood vessels, infarction, necrosis, superinfection; damage of intima occurs most frequently in vessels with high flow rates (terminal ICA); sickling occurs in areas of (a) slow flow (spleen, liver, renal medulla) (b) rapid metabolism (brain, muscle, fetal placenta) ● chronic hemolytic anemia (increased sequestration of sickled RBCs in spleen), jaundice ● chronic leg ulcers, priapism ● abdominal crisis ● rheumatism-like joint pain ● skeletal pain (osteomyelitis, cellulitis, bone marrow infarction) ● splenomegaly (in children + infants), later organ atrophy*Cx:*high incidence of infections (lung, bone, brain)*Prognosis:*death <40 years (1)DEOSSIFICATION DUE TO MARROW HYPERPLASIA√ porous decrease in bone density of skull (25%)√ widening of diploe with decrease in width of outer table (22%)√ vertical hair-on-end striations (5%)√ osteoporosis with thinning of trabeculae√ biconcave "fish" vertebrae (bone softening) in 70%√ widening of medullary space + thinning of cortices√ coarsening of trabecular pattern in long + flat bones√ rib notching√ pathologic fractures (2)THROMBOSIS AND INFARCTIONLocation:in diaphysis of small tubular bones (children); in metaphysis + subchondrium of long bones (adults)√ osteolysis (in ACUTE infarction)√ dystrophic medullary calcification√ periosteal reaction (bone-within-bone appearance)√ juxtacortical sclerosis√ Lincoln log = Reynold sign = H-vertebrae = steplike endplate depression√ articular disintegration√ collapse of femoral head (DDx: Perthes with involvement of metaphysis)MR: √ diffusely decreased signal of marrow on short + long TR/TE images (= hematopoietic marrow replacing fatty marrow)√ focal areas of decreased signal intensity on short TR/TE + increased intensity on long TR/TE (= acute marrow infarction)√ focal areas of decreased signal intensity on short TR/TE + long TR/TE images (= old infarction / fibrosis) (3)SECONDARY OSTEOMYELITIS*Organism:* Salmonella in unusual frequency, also Staphylococcus√ periostitis (DDx: indistinguishable from bone infarction)√ dactylitis = hand-foot syndrome (4)GROWTH EFFECTS (secondary to diminished blood supply)Location: particularly in metacarpal / phalanx√ bone shortening = premature epiphyseal fusion√ epiphyseal deformity with cupped metaphysis√ cup / peg-in-hole defect of distal femur√ diminution in vertebral height (shortening of stature + kyphoscoliosis) @Chest√ cardiomegaly + CHF@Gallbladder√ cholelithiasis@Brain*Pathophysiology:* chronic anemia produces cerebral hyperemia, hypervolemia, impaired autoregulation (a)cerebral blood flow cannot be increased leading to infarction in time of crisis(b)increased cerebral blood flow produces epithelial hyperplasia of large intracranial vessels (terminal ICA / proximal MCA) resulting in thrombus formation ● stroke (5-17%): ischemic infarction (70%), ischemia of deep white matter (25%), hemorrhage (20%), embolic infarctionAngio (in 87% abnormal): √ arterial stenosis / occlusion of supraclinoid portion of ICA + proximal segments of ACA and MCA√ moyamoya syndrome (35%)√ distal branch occlusion (secondary to thrombosis / embolism)√ aneurysm (rare)CT: √ cerebral infarction (mean age of 7.7 years)√ subarachnoid hemorrhage (mean age of 27 years) @Kidney ● hematuria ● hyposthenuria ● nephrotic syndrome ● renal tubular acidosis (distal) ● hyperuricemia ● progressive renal insufficiency√ normal urogram (70%)√ papillary necrosis (20%)√ focal renal scarring (20%)√ smooth large kidney (4%)MR: √ decreased cortical signal on T2-weighted images (renal cortical iron deposition) @Spleen√ splenomegaly < age 10 (in patients with heterozygous sickle cell disease)*Cx:*splenic rupture√ splenic infarction√ hemosiderosis **Functional asplenia** =anatomically present nonfunctional spleen ● Howell-Jolly bodies, siderocytes, anisocytosis, irreversibly sickled cells√ normal-sized / enlarged spleen on CT√ absence of tracer uptake on sulfur colloid scan **Autosplenectomy** =autoinfarction of spleen in homozygous sickle cell disease (function lost by age 5)*Histo:*extensive perivascular fibrosis with deposition of hemosiderin + calcium√ small (as small as 5-10 mm) densely calcified spleen **Acute splenic sequestration crisis** =sudden trapping of large amount of blood in spleen*Cause:*obstruction of small intrasplenic veins / sinusoids*Age:*(a)homozygous: infancy / childhood(b)heterozygous: any age ● sudden splenic enlargement ● rapid fall in hematocrit + rise in reticulocytes√ enlarged spleen√ multiple lesions at periphery of spleen: hypoechoic by US, of low attenuation by CT, hyperintense on T1WI + T2WI (due to hemorrhage)*Prognosis:*in 50% death <2 years of age (due to hypovolemic shock) Bone marrow scintigraphy: √ usually symmetric marked expansion of hematopoietic marrow beyond age 20 involving entire femur, calvarium, small bones of hand + feet (normally only in axial skeleton + proximal femur and humerus)√ bone marrow defects indicative of acute / old infarctionTc-99m diphosphonate scan: √ increased overall skeletal uptake (high bone-to-soft tissue ratio)√ prominent activities at knees, ankles, proximal humerus (delayed epiphyseal closure / increased blood flow to bone marrow)√ bone marrow expansion (calvarial thickening with relative decrease in activity along falx insertion)√ decreased / normal uptake on bone scan within 24 hours in acute infarction / posthealing phase following infarction (cyst formation)√ increased uptake on bone scan after 2-10 days persistent for several weeks in healing infarction√ increased uptake on bone scan within 24-48 hours in osteomyelitis√ increased blood-pool activity + normal delayed image on bone scan in cellulitis√ renal enlargement with marked retention of tracer in renal parenchyma (medullary ischemia + failure of countercurrent system) in 50%√ persistent splenic uptake (secondary to degeneration, atrophy, fibrosis, calcifications)

Sickle Cell Trait SC Disease Sickle-Thal Disease

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Sickle Cell Trait Hb SA carrier; mild disease with few episodes of crisis + infection; sickling provoked only under extreme stress (unpressurized aircraft, anoxia with CHD, prolonged anesthesia, marathon running) *Incidence:* in 8-10% of American Blacks ● may have normal blood count ● recurrent gross hematuria ● splenic infarction

Notes:

SC Disease Hb SC carrier *Incidence:*3% of American Blacks ● retinal hemorrhages ● hematuria due to multiple infarctions√ aseptic necrosis of hip

Notes:

Sickle-Thal Disease Resembling clinically Hb SS patients ● anemia (no normal adult hemoglobin)√ persistent splenomegaly

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SINDING-LARSEN-JOHANSSON DISEASE
=osteochondrosis of inferior pole of patella, often bilateral (NOT osteonecrosis / epiphysitis / osteochondritis)*Cause:*traction with contusion + subsequent tendinitis / traumatic avulsion of bone; repeated subluxation ± dislocation of patella*Age:*adolescents (often 10-14 years)*Predisposed:*cerebrospastic children ● tenderness + soft-tissue swelling over lower pole of patella√ peripatellar soft-tissue swelling√ calcification / ossification of patellar tendon√ small bone fragments at lower pole of patella (LAT view)MR: √ hypointense area on T1WI + hyperintense on T2WI in inferior pole of patella + surrounding soft tissues

Notes:

SMALLPOX

5% of infants Location:elbow bilateral; metaphysis of long bones√ rapid bone destruction spreading along shaft√ periosteal reaction√ endosteal + cortical sclerosis frequent√ premature epiphyseal fusion with severe deformity√ ankylosis is frequent

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SOFT-TISSUE CHONDROMA
=EXTRASKELETAL CHONDROMA = CHONDROMA OF SOFT PARTS*Incidence:*1.5% of all benign soft-tissue tumors*Age:*30-60 years (range 1-85 years); M:F = 1.2:1*Histo:*adult-type hyaline cartilage with areas of calcification + ossification; myxoid change; regions of increased cellularity + cytologic atypia ● slow-growing soft-tissue mass ● occasionally pain + tendernessLocation:hand (54-64%) + foot (20-28%)√ lobulated well-defined extraskeletal mass <2 cm in size√ may contain calcifications (33-70%) with ringlike appearance / ossifications√ scalloping of adjacent bone with sclerotic reactionMR: √ high signal intensity on T2WI√ intermediate signal intensity on T1WI*Rx:*local excision*Prognosis:*15-25% recurrence rate*DDx:*(1)Extraskeletal myxoid chondrosarcoma (deep-seated in large muscles of upper + lower extremities, pelvic + shoulder girdles)(2)Periosteal chondroma

Notes:

SOFT-TISSUE OSTEOMA
=OSTEOMA OF SOFT PARTS (extremely rare)*Histo:*mature lamellar bone with well-defined haversian system; bone marrow, myxoid, vascular, fibrous connective tissue between bone trabeculae; collagenous capsule blending into benign hyaline cartilageLocation:head (usually posterior part of tongue), thigh√ ossified mass√ intense tracer accumulation, greater than adjacent bone

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Notes:

SOLITARY BONE CYST
= UNICAMERAL / SIMPLE BONE CYST *Incidence:*up to 5% of primary bone lesionsEtiology:? trauma (synovial entrapment at capsular reflection), ? vascular anomaly (blockage of interstitial drainage)*Histo:*cyst filled with clear yellowish fluid often under pressure, wall lined with fibrous tissue + hemosiderin, giant cells may be present*Age:* 3-19 years (80%); occurs during active phase of bone growth; M:F = 3:1 ● asymptomatic, unless fracturedLocation:proximal femur + proximal humerus (60-75%), fibula, at base of calcaneal neck (4%, >12 years of age), talus; rare in ribs, ilium, small bones of hand + feet (rare), NOT in spine / calvarium; solitary lesionSite:intramedullary centric metaphyseal, adjacent to epiphyseal cartilage (during active phase) / migrating into diaphysis with growth (during latent phase), does not cross epiphyseal plate√ 2-3 cm oval radiolucency with long axis parallel to long axis of host bone√ fine sclerotic boundary√ scalloping + erosion of internal aspect of underlying cortex√ photopenic area on bone scan (if not fractured)√ "fallen fragment" sign if fractured (20%) = centrally dislodged fragment falls into a dependent position *Prognosis:*mostly spontaneous regression*Cx:*pathologic fracture (65%)DDx:(1) Enchondroma (calcific stipplings) (2) Fibrous dysplasia (more irregular lucency) (3) Eosinophilic granuloma (4) Chondroblastoma (epiphyseal) (5) Chondromyxoid fibroma (more eccentric + expansile) (6) Giant cell tumor (7) Aneurysmal bone cyst (eccentric) (8) Hemorrhagic cyst (9) Brown tumor

Notes:

SOLITARY OSTEOCHONDROMA
=OSTEOCARTILAGINOUS EXOSTOSIS=hyperplastic / dysplastic bone disturbance; growth ends when nearest epiphyseal plate fuses◊Most common benign growth of the skeleton!*Etiology:*displaced or aberrant physeal cartilage (? microtrauma); radiation induced with latency period of 17 months to 9 years in patient younger than 2 years receiving >2,500 cGy*Age:*1st-3rd decade; M>F*Path:*continuity of lesion with marrow + cortex of host bone (HALLMARK)*Histo:*cartilage cap containing a basal surface with enchondral ossification (cortex + marrow space) ● usually painless mass; painful with impingement of nerves / blood vesselsLocation:long-bone metaphysis of femur, humerus, proximal radius, tibia (50% about knee); scapula; rib; pelvis; spine (1-5%, commonly cervical, esp. C2); in any bone that develops by enchondromal calcification*Type:*(a) pedunculated form (b) broad-based sessile form (c) calcific form √ cortical bone with cartilaginous cap√ grows at right angles + toward diaphysis (tendon pull)√ continuity of bone cortex to host bone√ continuity of medullary marrow space to host bone√ metaphyseal widening *Cx:*(1)Impingement on nerves / blood vessels(2)Malignant transformation into chondro- / osteosarcoma (<1%)Signs of malignant degeneration *mnemonic:*"GLAD PAST"**G**rowth after epiphyseal fusion **L**ucency (new radiolucency) **A**dditional scintigraphic activity **D**estruction (cortical) **P**ain after puberty **A**nd **S**oft-tissue mass **T**hickened cartilaginous cap *Rx:*surgical excision (recurrence unusual)

Notes:

SOLITARY PLASMACYTOMA
=represents early stage of multiple myeloma, precedes multiple myeloma by 1-20 years*Age:*5th-7th decade ● negative marrow aspiration; no IgG spike in serum / urineA. SOLITARY MYELOMA OF BONE Site:thoracic / lumbar spine (most common) > pelvis > ribs > sternum, femora, humeri (common)√ solitary "bubbly" osteolytic grossly expansile lesion√ poorly defined margins, Swiss-cheese pattern√ frequently pathologic fracture (collapse of vertebra)*DDx:*giant cell tumor, aneurysmal bone cyst, osteoblastoma, solitary metastasis from renal cell / thyroid carcinomaB.EXTRAMEDULLARY PLASMACYTOMALocation:majority in head + neck; 80% in nasal cavity, paranasal sinuses, upper airways of trachea, lung parenchyma

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Spondyloepiphyseal Dysplasia Congenita Autosomal dominant / sporadic (most) ● disproportionate dwarfism with spine + hips more involved than extremities ● waddling gait + muscular weakness ● flat facies ● short neck ● deafness√ cleft palate@Axial skeleton√ ovoid vertebral bodies + severe platyspondyly (incomplete fusion of ossification centers + flattening of vertebral bodies)√ hypoplasia of odontoid process (Cx: cervical myelopathy)√ progressive kyphoscoliosis (short trunk) involving thoracic + lumbar spine√ narrowing of disk spaces (resulting in short trunk)√ broad iliac bases + deficient ossification of pubis√ flat acetabular roof@Chest√ bell-shaped thorax√ pectus carinatum@Extremities√ normal / slightly shortened limbs√ severe coxa vara + genu valgum√ multiple accessory epiphyses in hands + feet√ talipes equinovarus*Cx:*(1)Retinal detachment, myopia (50%)(2)Secondary arthritis in weight-bearing joints

Notes:

Spondyloepiphyseal Dysplasia Tarda Sex-linked recessive form with milder manifestation + later clinical onset *Age:*apparent by 10 years; exclusive to males√ hyperostotic new bone along posterior 2/3 of vertebral endplate (PATHOGNOMONIC)√ platyspondyly with depression of anterior 1/3 of vertebral body√ narrowing with calcification of disk spaces + spondylitic bridging√ short trunk√ dysplastic joints (eg, flattened femoral heads)√ premature osteoarthritis*DDx:*Ochronosis

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Notes:

SPRENGEL DEFORMITY
=failure of descent of scapula secondary to fibrous / osseous omovertebral connection*Associated with:* Klippel-Feil syndrome, renal anomalies ● webbed neck ● shoulder immobility√ elevation of scapula

Notes:

SYNOVIAL OSTEOCHONDROMATOSIS
=SYNOVIAL CHONDROMATOSIS = JOINT CHONDROMA=benign self-limiting proliferative + metaplastic changes in the synovium with formation of intrasynovial cartilaginous / osteocartilaginous nodules*Cause:*hyperplastic synovium with cartilage metaplasia (foci <2-3 cm); loose body may remain free floating / conglomerate with other loose bodies into large mass / reattach to synovium with either reabsorption or continued growth*Histo:*foci of hyaline cartilage with mineralized chondroid matrix beneath synovial surface + within subsynovial connective tissue; hypercellularity + nuclear atypia may be confused with malignancy*Age:*presents in 3rd-5th decade; M:F = 2-4:1 ● slow-growing soft-tissue mass in joint ● progressive joint pain for several years with limitation of motion / locking ● ± hemorrhagic joint effusionLocation:knee (most common with >50%, in 10% bilateral) elbow > hip > shoulder > ankle > wrist; usually monarticular, occasionally bilateralSites:within joint / tendon sheath / ganglion / bursa√ multiple calcified / ossified loose bodies in a single joint(bony shell of remodeled lamellar bone is rare)√ size of nodules varies between a few mm to several cm√ varying degrees of bone mineralization (1/3 of chondromas show no radiopacity)√ pressure erosion of adjacent bone in joints with tight capsule (eg, hip)√ widening of joint space (from accumulation of loose bodies)√ NO osteoporosisCT: √ intra-articular soft-tissue mass of near water attenuation containing multiple small calcificationsMR: √ lobulated intra-articular mass isointense to muscle on T1WI + hyperintense to muscle on T2WI containing multiple foci of low signal intensity*Cx:*(1)long-standing disease may lead to degenerative arthritis (from chronic mechanical irritation + destruction of articular cartilage by loose bodies)(2)malignant dedifferentiation to chondrosarcoma*Rx:*removal of loose bodies (recurrence is common)*DDx:*(1)Synovial sarcoma, chondrosarcoma(2)Osteochondral fracture (Hx of trauma), osteochondritis dissecans, osteonecrosis(3)Secondary chondromatosis = joint surface disintegration (rheumatoid arthritis, neuropathic arthropathy, tuberculous arthritis, degenerative joint disease)(4)Pigmented villonodular synovitis, synovial hemangioma, lipoma arborescens

Notes:

SYNOVIOMA
=SYNOVIAL SARCOMA=slow-growing expansile malignant tumor originating in the synovial lining / bursa / tendon sheath; uncommonly intra-articular*Incidence:*10% of soft-tissue sarcomas*Histo:*fibrosarcomatous + synovial component*Age:*3rd-5th decade; M:F = 2:3 ● painful soft-tissue massLocation:knee (most common), hip, ankle, elbow, wrist, hands, feet; usually solitary√ large spheroid well-defined soft-tissue mass√ lesion about 1 cm removed from joint cartilage√ amorphous calcifications (1/3), often at periphery√ involvement of adjacent bone (11-20%):√ periosteal reaction√ bone remodeling (pressure from tumor)√ invasion of cortex with wide zone of transition√ juxta-articular osteoporosisMR: √ low signal intensity on T1WI√ inhomogeneously increased signal intensity on T2WI√ multilocular appearance with internal septation√ fluid-fluid levels (previous hemorrhage)*Rx:*local excision / amputation + radiation / chemotherapy

Notes:

SYPHILIS OF BONE

Congenital syphilis Transplacental transmission cannot occur <16 weeks gestational age ● positive rapid plasma reagin (measures quantity of antibodies to assess new infection / efficacy of Rx) ● positive microhemagglutination test for Treponema pallidum (remains reactive for life)√ pneumonia alba√ hepatomegalyLocation:symmetrical bilateral osteomyelitis involving multiple bones (HALLMARK)A.Early phase√Skeletal radiography abnormal in 19% of infected newborns without overt disease!1.Metaphysitis√ lucent metaphyseal band adjacent to thin / widened zone of provisional calcification (disturbance in enchondral bone growth)√ frayed edge of metaphyseal-physeal junction (osteochondritis) = erosions + lytic defects 2.Diaphyseal periostitis = "luetic diaphysitis"√ solid / lamellated periosteal new-bone growth = bone-within-bone appearance3.Spontaneous epiphyseal fractures causing Parrot pseudopalsy (DDx: battered child syndrome)4.Bone destruction√ marginal destruction of spongiosa + cortex along side of shaft with widening of medullary canal (in short tubular bones)√ patchy rarefaction in diaphysis5.Wimberger sign√ symmetrical focal bone destruction of medial portion of proximal tibial metaphysis (ALMOST PATHOGNOMONIC)B.Late phase ● Hutchinson triad = dental abnormality, interstitial keratitis, 8th nerve deafness√ frontal bossing of Parrot = diffuse thickening of outer table√ saddle nose + high palate (syphilitic chondritis + rhinitis)√ short maxilla (maxillary osteitis)√ thickening at sternal end of clavicle√ "saber-shin" deformity = anteriorly convex bowing in upper 2/3 of tibia with bone thickening **Acquired Syphilis** =TERTIARY SYPHILIS resembles chronic osteomyelitis√ dense bone sclerosis of long bones√ irregular periosteal proliferation + endostealthickening with narrow medulla√ extensive calvarial bone proliferation with mottled pattern (anterior half + lateral skull) in outer table (DDx: fibrous dysplasia, Paget disease)√ ill-defined lytic destruction in skull, spine, long bones (gumma formation)√ enlargement of clavicle (cortical + endosteal new bone)√ Charcot arthropathy = neuropathic joints (lower extremities + spine)

Notes:

TARSAL COALITION
=abnormal fibrous / cartilaginous / bony fusion of two or more tarsal ossification centersMost important congenital problem of calcaneus clinically ● asymptomatic / painful pes planus with peroneal spasm*Age:*fibrous coalition at birth, ossification during 2nd decade of life√ bone bars on lateral radiographs between calcaneus, talus, navicular (CT superior to other imaging)√ both feet affected in 20%*Types:* (1)calcaneonavicular coalition (30%)M:F = 1:1 ● rigid flat foot ± pain in 2nd decade of life√ hypoplastic talar head√ narrowed calcaneonavicular joint with indistinct articular margins(2)talocalcaneal coalition (60%) ● painful peroneal spastic flat foot, relieved by restSite:middle facet (most frequently)√ prominent talar beak (66%) arising from dorsal aspect of head / neck of talus√ "ball-and-socket" ankle mortise√ asymmetric anterior talocalcaneal joint*DDx:*acquired intertarsal ankylosis (infection, trauma, arthritis, surgery)

Notes:

THALASSEMIA SYNDROMES

PHYSIOLOGIC HEMOGLOBINS (a)in adulthood:Hb A (98% = 2 a- and 2 b-chains); Hb A_2 (2% = 2 a- and 2 d-chains) (b)in fetal life, rapidly decreasing up to 3 months of newborn period: Hb F (= 2 a- and 2 g-chains) A.ALPHA-THALASSEMIA=decreased synthesis of a-chains leading to excess of b-chains + g-chains (Hb H = 4 b-chains; Hb Bart = 4 g-chains) ● disease begins in intrauterine life as no fetal hemoglobin is produced ● homozygosity is lethal (lack of oxygen transport)B.BETA-THALASSEMIA=decreased synthesis of b-chains leading to excess of a-chains + g-chains (= fetal hemoglobin) ● disease manifest in early infancy(a)homozygous defect = thalassemia major = Cooley anemia(b)heterozygous defect = thalassemia minor

Thalassemia Major Thalassemia Minor

Notes:

Thalassemia Major =COOLEY ANEMIA = MEDITERRANEAN ANEMIA= HEREDITARY LEPTOCYTOSIS = beta-thalassemia trait inherited from both parents (= homozygous) *Incidence:*1% for American Blacks; 7.4% for Greek population; 10% for certain Italian populations*Age:*develops after newborn period ● retarded growth ● elevated serum bilirubin ● hyperpigmentation of skin ● hyperuricemia ● secondary sexual characteristics retarded, normal menstruation rare (primary gonadotropin insufficiency from iron overload in pituitary gland) ● hypochromic microcytic anemia (Hb 2-3 g/dL), nucleated RBC, target cells, reticulocytosis, decrease in RBC survival, leukocytosis ● susceptible to infection (leukopenia secondary to splenomegaly) ● bleeding diathesis (secondary to thrombocytopenia)@Skull:√ widening of diploic space with coarsened trabeculations and displacement + thinning of outer table (from marrow hyperplasia)√ severe hair-on-end appearance (frontal bone, NOT inferior to internal occipital protuberance)√ impediment of pneumatization of maxillary antra + mastoid sinuses√ lateral displacement of orbits√ rodent facies = ventral displacement of incisors (marrow overgrowth in maxillary bone) with dental malocclusion@Peripheral skeleton:√ earliest changes in small bones of hands + feet (>6 months of age)√ widened medullary spaces with thinning of cortices√ osteoporosis = atrophy + coarsening of trabeculae (marrow hyperplasia)√ Erlenmeyer flask deformity = bulging of normally concave outline of metaphyses√ premature fusion of epiphyses (10%), usually at proximal humerus + distal femur√ arthropathy (secondary to hemochromatosis + CPPD + acute gouty arthritis)√ regression of peripheral skeletal changes (as red marrow becomes yellow)@Chest:√ cardiac enlargement + congestive heart failure (secondary to anemia)√ paravertebral masses (= extramedullary hematopoiesis)√ costal osteomas = expanded posterior aspect of ribs with thinned cortices@Abdomen:√ hepatosplenomegaly√ gallstones*Cx:*(1)Pathologic fractures(2)Sequelae of iron overload from transfusion therapy (absent puberty, diabetes mellitus, adrenal insufficiency, myocardial insufficiency)*Prognosis:*usually death within 1st decade

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Thalassemia Minor =beta-thalassemia trait inherited from one parent (= heterozygous) ● usually asymptomatic except for periods of stress (pregnancy, infection) ● microcytic hypochromic anemia (Hb 9-11 g/dL) ● occasionally jaundice + splenomegaly

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Notes:

THANATOPHORIC DYSPLASIA
=sporadic lethal skeletal dysplasia characterized by severe rhizomelia (micromelic dwarfism)*Incidence:*6.9:100,000 births; 1:6,400-16,700 births; most common lethal bone dysplasia ● hypotonic infants ● protuberant abdomen ● extended arms + abducted externally rotated thighs@Head√ large head with short base of skull + prominent frontal bone√ occasionally trilobed cloverleaf skull = "Kleeblattschädel"@Chest√ narrow chest√ short horizontal ribs with cupped anterior ends√ small scapula + normal clavicles@Spine√ normal length of trunk√ reduction of interpediculate space of last few lumbar vertebrae√ extreme generalized platyspondyly = severe H-shaped vertebra plana√ excessive intervertebral space height@Pelvis√ iliac wings small + square (vertical shortening but wide horizontally)√ flat acetabulum √ narrow sacrosciatic notch√ short pubic bones@Extremities√ severe micromelia + bowing of extremities√ metaphyseal flaring = "telephone handle" appearance of long bones√ thornlike projections in metaphyseal area OB-US (findings may be seen very early in pregnancy): √ polyhydramnios (71%)√ short-limbed dwarfism with extremely short + bowed "telephone receiver"-like femurs√ extremely small hypoplastic thorax with short ribs + narrowed in anteroposterior dimension√ protuberant abdomen√ macrocrania with frontal bossing ± hydrocephalus (increased HC:AC ratio)√ "cloverleaf skull" (in 14%) (DDx: encephalocele)√ diffuse platyspondyly√ redundant soft tissues*Prognosis:*often stillborn; uniformly fatal within a few hours / days after birth (respiratory failure)*DDx:*(1)Ellis-van Creveld syndrome (extra digit, acromesomelic short limbs)(2)Asphyxiating thoracic dysplasia (less marked bone shortening, vertebrae spared)(3)Short-rib polydactyly syndrome(4)Homozygous achondroplasia (both parents affected)

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THROMBOCYTOPENIA-ABSENT RADIUS SYNDROME

=TAR SYNDROME = rare autosomal recessive disorder*Age:*presentation at birth*May be associated with:* CHD (33%): ASD, tetralogy ● platelet count <100,000/mm^3 (decreased production by bone marrow)√ usually bilateral radial aplasia / hypoplasia√ uni- / bilaterally hypoplastic / absent ulna / humerus√ defects of hands, feet, legs*Prognosis:*death in 50% in early infancy (hemorrhage)

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THYROID ACROPACHY

*Onset:*after 18 months following thyroidectomy for hyperthyroidism (does not occur with antithyroid medication)*Incidence:*1-10% ● clubbing, soft-tissue swelling ● eu- / hypo- / hyperthyroid stateLocation:diaphyses of phalanges + metacarpals of hand; less commonly feet, lower legs, forearms ● thick spiculated lacy periosteal reaction*DDx:*(1)Pulmonary osteoarthropathy (painful)(2)Pachydermoperiostosis(3)Fluorosis (ligamentous calcifications)

Notes:

TRANSIENT REGIONAL OSTEOPOROSIS
*Cause:*unknown; ? overactivity of sympathetic nervous system + local hyperemia similar to reflex sympathetic dystrophy syndrome, trauma, synovitis, transient ischemia

Regional Migratory Osteoporosis Transient Osteoporosis of Hip

Notes:

Regional Migratory Osteoporosis =rapid onset of self-limiting episodes of severe localized osteoporosis and pain but repetitive occurrence of same symptoms in other regions of the same or opposite lower extremity ● rapid onset of local pain ● diffuse erythema, swelling, increased heat ● significant disability due to severe pain on weight-bearing*Age:*middle-aged malesLocation:usually lower extremity (ie, ankle, knee, hip, foot)√ rapid localized osteoporosis within 4-8 weeks after onset migrating from one joint to another; may affect trabecular / cortical bone√ linear / wavy periosteal reaction√ preservation of subchondral cortical bone√ no joint space narrowing, bone erosionMR: √ affected area has low signal intensity on T1WI, high signal intensity on T2WI (= bone marrow edema)NUC: √ increased activity*Prognosis:*persists for 6-9 months in one area; cycle of symptoms may last for several years*Rx:*variable response to analgesics / corticosteroids ***Partial Transient Osteoporosis*** =variant of regional migratory osteoporosis with more focal pattern of osteoporosis, which may eventually become more generalized(a)Zonal form = portion of bone involved, ie, one femoral condyle / one quadrant of femoral head(b)Radial form = only one / two rays of hand / foot involved

Notes:

Transient Osteoporosis of Hip =self-limiting disease of unknown etiology*Age:*typically in middle-aged males / in 3rd trimester of pregnancy in females involving left hip; M > F ● spontaneous onset of hip and groin pain, usually progressive over several weeks ● painful swelling of joint followed by progressivedemineralization ● rapid development of disability, limp, decreased range of motionSite:hip most commonly affected; generally only one joint at a time√ progressive marked osteoporosis of femoral head, neck, acetabulum (3-8 weeks after onset of illness)√ virtually PATHOGNOMONIC striking loss of subchondral cortex of femoral head + neck region√ NO joint space narrowing / subchondral bone collapseNUC: √ markedly increased uptake on bone scan without cold spots / inhomogeneities (positive before radiograph)MR: √ diffuse bone marrow edema involving femoral head + neck + sometimes intertrochanteric region√ small joint effusion*Cx:*pathologic fracture common*Prognosis:*spontaneous recovery within 2-6 months; recurrence in another joint within 2 years possible*DDx:*(1)AVN (cystic + sclerotic changes, early subchondral undermining)(2)Septic / tuberculous arthritis (joint aspiration)(3)Monarticular rheumatoid arthritis(4)Metastasis(5)Reflex sympathetic dystrophy(6)Disuse atrophy(7)Synovial chondromatosis(8)Villonodular synovitis

Notes:

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TRANSIENT SYNOVITIS OF HIP
=OBSERVATION HIP = TRANSITORY SYNOVITIS = TOXIC SYNOVITIS = COXITIS FUGAX= nonspecific inflammatory reaction; most common nontraumatic cause of acute limp in a child *Etiology:*unknown*Age:*5-10 (average 6) years; M:F = 2:1 ● developing limp over 1-2 days ● pain in hip, thigh, knee ● Hx of recent viral illness (65%) ● mild fever (25%)√ radiographs usually normal√ joint effusion√ displacement of femur from acetabulum√ displacement of psoas line√ lateral displacement of gluteal line (least sensitive + least reliable)√ regional osteoporosis (? hyperemia, disuse)*Prognosis:*complete recovery within a few weeks*Dx:*per exclusion*Rx:*non-weight-bearing treatment*DDx:*trauma, Legg-Perthes disease, acute rheumatoid arthritis, acute rheumatic fever, septic arthritis, tuberculosis, malignancy

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Notes:

TREACHER COLLINS SYNDROME
=MANDIBULOFACIAL DYSOSTOSIS=autosomal dominant disease (with new mutations in 60%) characterized by bilateral malformations of eyes, malar bones, mandible, and ears resulting in birdlike face*Cause:*defect in growth of 1st + 2nd branchial arches before the 7th to 8th week of gestation ● antimongoloid eye slant (drooping lateral lower eyelids due to hypoplasia of lateral canthal tendon of orbicular muscle) ● sparse / absent lashes in lower eye lids, coloboma ● dysplastic low-set auricles ● preauricular skin tags / fistulas ● conductive hearing loss (common) ● extension of scalp hair growth onto cheek√ craniosynostosis√ egg-shaped orbits = drooping of outer inferior orbital rim√ sunken cheek due to marked hypoplasia of zygomatic arches (= malar hypoplasia)√ hypoplasia of lateral wall of orbits + shallow / incomplete orbital floor√ hypoplasia of maxilla + maxillary sinus√ pronounced micrognathia = mandibular hypoplasia with broad concave curve on lower border of body √ microtia with small middle ear cavity√ deformed / fused / absent auditory ossicles√ atresia / stenosis of external auditory canal√ high-arched / cleft palateOB-US: √ polyhydramnios (from swallowing difficulty)*Prognosis:*early respiratory problems (tongue relatively too large for hypoplastic mandible)*DDx:*(1)Goldenhar-Gorlin syndrome (unilateral microtia + midface anomalies, hemivertebrae, block vertebrae, vertebral hypoplasia, microphthalmia, coloboma of upper lid)(2)Acrofacial dysplasia (limb malformations)(3)Crouzon disease (maxillary hypoplasia with protrusion of mandible, hypertelorism, exophthalmos, craniosynostosis)

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TRISOMY D SYNDROME
=Trisomy 13-15 group syndrome*Etiology:*additional chromosome in D group; high maternal age ● severe mental retardation ● hypertonic infant ● cleft lip + palate*Associated with:*capillary hemangioma of face + upper trunk ● hypotelorism ● coloboma, cataract, microphthalmia ● malformed ear with hypoplastic external auditory canal ● hyperconvex nails√ postaxial polydactyly@Skull√ deficient ossification of skull√ cleft / absent midline structures of facial bones√ poorly formed orbits√ slanting of frontal bones√ microcephaly√ arrhinencephaly√ holoprosencephaly@Chest√ thin malformed ribs√ diaphragmatic hernia (frequent)√ congenital heart disease*Prognosis:*death within 6 months of age

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Notes:

TRISOMY E SYNDROME

= Trisomy 16-18 group syndrome *Etiology:*additional chromosome at 18 or E group location*Sex:*usually female ● hypertonic infants ● mental + psychomotor retardation ● typical facies: micrognathia, high narrow palate with small buccal cavity, low-set deformed ears ● flexed ulnar-deviated fingers + short adducted thumb ● 2nd finger overlapping of 3rd (CHARACTERISTIC)*Associated with:*congenital heart disease in 100% (PDA, VSD); hernias; renal anomalies; eventration of diaphragm√ stippled epiphyses@Skull√ thin calvarium√ persistent metopic suture√ prominent occiput√ hypoplastic mandible (most constant feature) + maxilla@Chest√ increase in AP diameter of thorax√ hypoplastic sternum√ hypoplastic clavicles (DDx: cleidocranial dysostosis)√ slender + tapered ribs√ diaphragmatic eventration (common)@Pelvis √ small pelvis with forward rotation of iliac wings√ increased obliquity of acetabulum@Hand & foot√ adducted thumb = short 1st metacarpal + phalanges (DIAGNOSTIC)√ overlap of 2nd on 3rd finger (DIAGNOSTIC)√ flexed ulnar-deviated fingers√ short 1st toe√ varus deformities of forefoot + dorsiflexion of toes√ rocker bottom foot / extreme pes planus (frequent)OB-US: √ hydrocephalus√ cystic hygroma√ diaphragmatic hernia√ clubfoot√ overlapping index finger√ choroid plexus cyst (30%)*Prognosis:*child rarely survives beyond 6 months of age

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TUBERCULOSIS OF BONE

*Incidence:*3-5% of tuberculous patients, 30% in patients with extrapulmonary tuberculosis*Age:*any, rare in 1st year of life, M:F = 1:1 ● negative skin test excludes diagnosis ● history of active pulmonary disease (in 50%)Location:vertebral column, hip, knee, wrist, elbow*Pathogenesis:* 1.Hematogenous spread from(a)primary infection of lung (particularly in children)(b)quiescent primary pulmonary site / extraosseous focus2.Reactivation: especially in hip

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Tuberculous Arthritis Tuberculous Osteomyelitis Tuberculous Spondylitis

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Notes:

Tuberculous Arthritis =joint involvement usually secondary to adjacent osteomyelitis*Incidence:*84% of skeletal tuberculosis*Pathophysiology:*synovitis with pannus formation leads to chondronecrosis*Age:*middle-aged / elderly ● chronic pain, weakness, muscle wasting ● soft-tissue swelling, draining sinus ● joint fluid: high WBC count, low glucose level, poor mucin clot formation (similar to rheumatoid arthritis)Location:hip, knee > elbow, wrist, sacroiliac joint, glenohumeral, articulation of hand + foot√ Phemister triad:1.gradual narrowing of joint space due to slow cartilage destruction (DDx: cartilage destruction in pyogenic arthritis is much quicker)2.peripherally located (= marginal) bone erosions3.juxta-articular osteoporosisEarly radiographs: √ joint effusion (hip in 0%, knee in 60%, ankle in 80%)√ extensive osteopenia (deossification) adjacent to primarily weight-bearing joints√ soft tissues normalLate radiographs: √ small cystlike erosions along joint margins in non-weight-bearing line opposing one another (DDx: pyogenic arthritis erodes articular cartilage)√ no joint space narrowing for months√ articular cortical bone destruction earlier in joints with little unopposed surfaces (hip, shoulder)√ infection of subchondral bone forming "kissing sequestra"√ increased density with extensive soft-tissue calcifications in healing phase*Cx:*fibrous ankylosis, leg shortening*Dx:*synovial biopsy (in 90% positive), culture of synovial fluid (in 80% positive)

Notes:

Tuberculous Osteomyelitis *Incidence:*16% of skeletal tuberculosis*Age:*children <5 years (0.5-14%), rare in adults ● painless swelling of hand / footLocation:any boneSite:(a)epiphysis with spread to joint / spread from adjacent affected joint (most common)(b)metaphysis with transphyseal spread (in child) (DDx: pyogenic infections usually do not extend across physis)(c)diaphysis (<1%)√ initially round / oval poorly defined lytic lesion with minimal / no surrounding sclerosis√ varying amounts of eburnation + periostitis√ advanced epiphyseal maturity / overgrowth (due to hyperemia) ± limb shortening from premature physeal fusion√ cystic tuberculosis = well-marginated osseous lesions(a)in children (frequent): in peripheral skeleton, ± symmetric distribution, no sclerosis(b)in adults: in skull / shoulder / pelvis / spine, with sclerosis√ spina ventosa = tuberculous dactylitis = digit with exuberant periosteal new-bone formation of fusiform appearance secondary to erosion of endosteal cortex with lamellated / solid periosteal thickening in hands + feet

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Notes:

Tuberculous Spondylitis =POTT DISEASE=destruction of vertebral body + intervertebral disk by tuberculous mycobacterium*Incidence:*<1% of patients with tuberculosis; 25-60% of all skeletal tuberculosis*Age:*children / adults; M > F ● insidious onset of back pain, stiffness ● local tenderness ● NO pulmonary lesions in 50%Location:thoracolumbar area (L1 most common), frequent involvement of multiple contiguous segmentsSite:vertebral body (82%) > posterior elements (18%)*Spread:* (a)hematogenous spread via paravertebral venous plexus of Batson: separate foci in 1-4%(b)contiguous into disk by penetrating subchondral bone plate + cartilaginous endplate (c)subligamentous spread beneath paraspinal ligaments to adjacent vertebral bodies√ erosion and collapse of vertebral endplates leads to narrowing of vertebral interspaces (first change)N.B.:vertebral disk space maintained longer than in pyogenic arthritis (disk itself preserved but fragmented)√ destruction of centra√ vertebra plana in children√ angular kyphotic deformity (= gibbus) in adults√ vertebra within a vertebra (= growth recovery lines)√ ivory vertebra (= reossification as healing response to osteonecrosis)√ large cold fusiform abscess in paravertebral gutters / psoas, commonly bilateral, ± anterolateral scalloping of vertebral bodies√ amorphous / teardrop-shaped calcification in paraspinal area between L1 + L5 (DDx: nontuberculous abscess rarely calcifies)√ "gouge defect" = mild contour irregularity of anterior and lateral aspect of vertebral body (= erosion from subligamentous extension of tuberculous abscess)*Cx:*angular kyphosis (= gibbus deformity), scoliosis, ankylosis, osteonecrosis, paralysis (spinal cord compression from abscess, granulation tissue, bone fragments, arachnoiditis)*Prognosis*:26-30% mortality rate*DDx:*1.Pyogenic spondylitis (rapid destruction, multiple abscess cavities, no thickening / calcification of abscess rim, little new-bone formation, posterior elements not involved)2.Neoplasia (multiple noncontiguous lesions, no disk destruction, little soft-tissue involvement)

Notes:

TUMORAL CALCINOSIS
=LIPOCALCINOGRANULOMATOSIS=rare disease with progressive large nodular juxta-articular calcified soft-tissue masses in patients with normal serum calcium + phosphorus and no evidence of renal, metabolic, or collagen-vascular disease *Etiology:*autosomal dominant (1/3) with variable clinical expressivity; unknown biochemical defect of phosphorus metabolism responsible for abnormal phosphate reabsorption + 1,25-dihydroxy-vitamin D formation *Path:*multilocular cystic lesions with creamy white fluid (hydroxyapatite) + many giant cells (granulomatous foreign body reaction) surrounded by fibrous capsule *Age:* onset mostly within 1st / 2nd decade (range of 1-79 years); M:F = 1:1; predominantly in Blacks ● progressive painful / painless soft-tissue mass with overlying skin ulceration + sinus tract draining chalky milklike fluid ● swelling ● limitation of motion ● hyperphosphatemia + hypervitaminosis D ● normal serum calcium, alkaline phosphatase, renal function, parathyroid hormone @Soft tissueLocation:para-articular in hips > elbows > shoulders > feet, ribs, ischial spines; single / multiple joints; ALMOST NEVER knees; usually along extensor surface of joints (? initially a calcific bursitis)√ dense loculated multiglobular homogeneously calcified soft-tissue mass of 1-20 cm in size√ radiolucent septa (= connective tissue)√ ± fluid-fluid levels with milk-of-calcium consistency√ underlying bones NORMAL√ increased tracer uptake of soft-tissue masses on bone scan@Bone√ diaphyseal periosteal reaction (diaphysitis)√ patchy areas of calcification in medullary cavity (calcific myelitis)@Teeth√ bulbous root enlargement√ pulp stones = intrapulp calcifications@Pseudoxanthoma elasticum-like features√ calcinosis cutis = skin calcifications√ vascular calcifications√ angioid streaks of retina *Prognosis:*tendency for recurrence after incomplete excision*Rx:*phosphate depletion*DDx:*Chronic renal failure on hemodialysis, CPPD, paraosteoarthropathy, hyperparathyroidism

Notes:

TURNER SYNDROME
=due to nondisjunction of sex chromosomes as(1) complete monosomy (45,XO) (2) partial monosomy (structurally altered second X chromosome) (3) mosaicism (XO + another sex karyotype) *Incidence:*1:3,000-5,000 livebirths*Associated with:*coarctation, aortic stenosis, horseshoe kidney (most common) ● sexual infantilism: primary amenorrhea, absent secondary sex characteristics ● short stature; absence of prepubertal growth spurt ● webbed neck; low irregular nuchal hair line ● shield-shaped chest + widely spaced nipples ● mental deficiency (occasionally) ● high palate; thyromegaly ● multiple pigmented nevi; keloid formation ● idiopathic hypertension; elevated urinary gonadotropins@General√ normal skeletal maturation with growth arrest at skeletal age of 15 years√ delayed fusion of epiphyses > age 20 years√ osteoporosis during / after 2nd decade (gonadal hormone deficiency)√ coarctation of aorta (10%); aortic stenosis√ renal ectopia / horseshoe kidney√ lymphedema@Skull√ basilar impression; basal angle >140°√ parietal thinning√ small bridged sella√ hypertelorism@Axial skeleton√ hypoplasia of odontoid process + C1√ osteochondrosis of vertebral plates√ squared lumbar vertebrae; kyphoscoliosis√ deossification of vertebrae√ small iliac wings; late fusion of iliac crests√ android pelvic inlet with narrowed pubic arch + small sacrosciatic notches@Chest√ thinning of lateral aspects of clavicles√ thinned + narrowed ribs with pseudonotching@Hand + arm√ positive metacarpal sign = relative shortening of 4th metacarpal = tangential line along heads of 5th + 4th metacarpals intersects 3rd metacarpal√ positive carpal sign = narrowing of scaphoid-lunate-triquetrum angle <117°√ phalangeal preponderance = length of proximal + distal phalanx exceeds length of 4th metacarpal by >3 mm√ shortening of 2nd + 5th middle phalanx (also in Down syndrome)√ "drumstick" distal phalanges = slender shaft + large distal head√ "insetting" of epiphyses into bases of adjacent metaphyses (phalanges + metacarpals)√ Madelung deformity = shortening of ulna / absence of ulnar styloid process√ cubitus valgus = bilateral radial tilt of articular surface of trochlea√ deossification of carpal bones@Knee√ tibia vara = enlarged medial femoral condyle + depression of medial tibial plateau (DDx: Blount disease)√ small exostosis-like projection from medial border of proximal tibial metaphysis@Foot√ deossification of tarsal bones√ shortening of 1st, 4th, and 5th metatarsals√ pes cavusOB-US: √ large nuchal cystic hygroma√ lymphangiectasia with generalized hydrops√ symmetrical edema of dorsum of feet√ CHD (20%): coarctation of aorta (70%), left heart lesions√ horseshoe kidney **Bonnevie-Ullrich Syndrome** = infantile form of Turner syndrome (1)congenital webbed neck(2)widely separated nipples(3)lymphedema of hands + feet

Notes:

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VAN BUCHEM DISEASE
=GENERALIZED CORTICAL HYPEROSTOSISmay be related to hyperphosphatasemia ● paralysis of facial nerve ● auditory + ocular disturbances (in late teens secondary to foraminal encroachment) ● increased alkaline phosphataseLocation:skull, mandible, clavicles, ribs, long-bone diaphyses√ symmetrical generalized sclerosis + thickening of endosteal cortex√ obliteration of diploe√ spinous processes thickened + sclerotic*DDx:*(1)Osteopetrosis (sclerosis of all bones, not confined to diaphyses)(2)Generalized hyperostosis with pachydermia (involves entire long bones, considerable pain, skin changes)(3)Hyperphosphatasia (infancy, widened bones but decreased cortical density)(4)Engelmann disease (rarely generalized, involves lower limbs)(5)Pyle disease (does not involve middiaphyses)(6)Polyostotic fibrous dysplasia (rarely symmetrically generalized, paranasal sinuses abnormal, skull involvement)

Notes:

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WILLIAMS SYNDROME

=IDIOPATHIC HYPERCALCEMIA OF INFANCY ● elfin facies, dysplastic dentition ● neonatal hypercalcemia ● mental retardation@Skeletal manifestations√ osteosclerosis (secondary to trabecular thickening)√ dense broad zone of provisional calcification√ radiolucent metaphyseal bands√ dense vertebral endplates + acetabular roofs√ bone islands in spongiosa√ metastatic calcification√ craniostenosis@Cardiovascular manifestations√ supravalvular aortic stenosis, aortic hypoplasia √ pulmonic stenosis√ stenoses of major vessels (innominate, carotids, renal arteries)@GI and GU tract:√ colonic diverticula√ bladder diverticula*Prognosis:*spontaneous resolution after 1 year in most*Rx:*withhold vitamin D + calcium*DDx:*Hypervitaminosis D

Notes:

WILSON DISEASE
=HEPATOLENTICULAR DEGENERATION=autosomal recessive disease with excessive copper retention (= copper toxicosis)*Prevalence:*1:33,000-200,000; 1:90 persons is a heterozygous carrier*Cause:*alteration of chromosome 13 resulting in inability of liver to excrete copper into bile;hypothetically due to either (a)lysosomal defect in hepatocytes, or(b)deficiency of biliary copper-binding proteins, or(c)persistence of fetal mode of copper metabolism, or(d)hepatic synthesis of high-affinity copper-binding proteins)*Age of onset:*7-50 years; hepatic manifestations predominate in children; neuropsychiatric manifestations predominate in adolescents + adults*Histo:*macrovesicular fat deposition in hepatocytes, glycogen degeneration of hepatocyte nuclei, Kupffer cell hypertrophy Stage 1asymptomatic copper accumulation in hepatocytic cytosolStage 2redistribution of copper into hepatic lysosomes + circulation from saturated hepatocytic cytosol(a)gradual redistribution is asymptomatic(b)rapid redistribution causes fulminant hepatic failure / acute intravascular hemolysisStage 3cirrhosis, neurologic, ophthalmologic, renal dysfunction may be reversible with therapy ● tremor, rigidity, dysarthria, dysphagia (excessive copper deposition in lenticular region of brain) ● intellectual impairment, emotional disturbance ● Kayser-Fleischer ring (= green pigmentation surrounding limbus corneae) is DIAGNOSTIC ● jaundice / portal hypertension (liver cirrhosis) ● elevated copper concentration in serum ceruloplasmin (BEST SCREENING TEST) ● decreased incorporation of orally administered radiolabeled copper into newly synthesized ceruloplasminSkeletal manifestations (in 2/3): √ generalized deossification may produce pathologic fractures@Joints: shoulder (frequent), knee, hip, wrist, 2nd-4th MCP joints ● articular symptoms in 75%: pain, stiffness, gelling of joints√ subarticular cysts√ premature osteoarthritis (narrowing of joint space + osteophyte formation) √ osteochondritis dissecans√ chondrocalcinosis√ premature osteoarthrosis of spine, prominent Schmorl nodes, wedging of vertebrae, irregularities of vertebral plates@BrainLocation:basal ganglia, rarely thalamus√ cerebral white matter atrophy√ hypodensities, prolongation of T1 + T2*Cx:*rickets + osteomalacia (secondary to renal tubular dysfunction) in minority of patients*Rx:*life long pharmacologic therapy with chelation agents (penicillamine / trientine / zinc); liver transplantation

Notes:

Skull and spine disorders

GLIOMA OF SPINAL CORD

HEMANGIOBLASTOMA OF SPINE

KLIPPEL-FEIL SYNDROME

KÜMMELL DISEASE

LEPTOMENINGEAL CYST

LIPOMA OF SPINE

- Intradural Lipoma
- Lipomyelomeningocele
- Fibrolipoma of Filum Terminale

LÜCKENSCHÄDEL

MENINGIOMA OF SPINE

METASTASES TO SPINE

METASTASES TO SPINAL CORD

- CSF Seeding of Intracranial Neoplasms

MYELOCYSTOCELE

MYELOMENINGOCELE

NEURENTERIC CYST

OSSIFYING FIBROMA

OSTEOMYELITIS OF VERTEBRA

PERINEURAL SACRAL CYST

SACRAL AGENESIS

SACROCOCCYGEAL TERATOMA

SCHEUERMANN DISEASE

SPINAL STENOSIS

SPLIT NOTOCHORD SYNDROME

SPONDYLOLISTHESIS

- Isthmic Spondylolisthesis = open-arch type
- Degenerative Spondylolisthesis = closed-arch type

SPONDYLOLYSIS

- Spondylolysis of Cervical Spine

SYRINGOHYDROMYELIA

- Hydromyelia
- Syringomyelia
- Reactive Cyst

TETHERED CORD

TERATOMA OF SPINE

LUMBOSACRAL POSTSURGICAL SYNDROME
=signs of dysfunction and disability + pain and paresthesia following surgery*Cause:* A.Biomechanical failure1.Primary disk herniation2.Recurrent disk herniation(onset 1 week - 1 month) B.Failure of surgical treatment1.Residual disk herniation(onset <1 week) 2.Perioperative intraspinal hemorrhage(onset <1 week) 3.Spinal / meningeal / neural inflammation(onset 1 week - 1 month) 4.Intraspinal scar formation (onset >1 month)(a)Epidural fibrosis√ enhancing epidural plaque / mass (b)Fibrosing arachnoiditis√ clumping of nerve roots√ adhesion of roots to wall of thecal sac√ abnormal enhancement of thickened meninges + matted nerve roots5.Remote phenomena unrelated to spine

Notes:

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FAILED BACK SURGERY SYNDROME
=failure of improvement following back surgery in 5-15%Interpretation in immediate postoperative period difficult, stabilization of findings occurs in 2-6 monthsA.OSSEOUS CAUSES1.Spondylolisthesis2.Central stenosis3.Foraminal stenosis4.PseudarthrosisB.SOFT-TISSUE CAUSES1.Adhesive arachnoiditis thickened irregular clumped nerve roots2.Infection3.Hemorrhage4.Epidural fibrosis (scarring) heterogeneous enhancement on early T1WI (maximum at about 5 minutes post injection)5.Recurrent disk herniation no enhancement on early T1WI (appears enhanced ≥30 minutes post injection)C.SURGICAL ERRORS1.Wrong level / side of surgery2.Direct nerve injury*mnemonic:*"ABCDEF"**A**rachnoiditis **B**leeding **C**ontamination (infection) **D**isk (residual / recurrent / new level) **E**rror (wrong disk excised) **F**ibrosis (scar)

Notes:

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CAUDA EQUINA SYNDROME
=constellation of signs + symptoms resulting from compressive lesion in lower lumbar spinal canal*Cause:* (1)Displaced disk fragment(2)Intra- / extramedullary tumor(3)Osseous: Paget disease, osteomyelitis, osteoarthrosis of facet joints, complication of ankylosing spondylitis ● diminished sensation in lower lumbar + sacral dermatomes ● wasting + weakness of muscles ● decreased ankle reflexes ● impotence ● disturbed sphincter function + overflow incontinence ● decreased sphincter tone

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Notes:

Mandibular Hypoplasia = **Micrognathia** A.WITH ABNORMAL EARS1.Treacher-Collins syndrome2.Goldenhar syndrome = facio-auriculo-vertebral spectrum (x-rays of vertebrae!)3.Langer-Giedion syndrome (IUGR, protruding ears)B.ABNORMALITIES OF EARS + OTHER ORGANS1.Miller syndrome (severe postaxial hand anomalies)2.Velo-cardio-facial syndrome (hand + cardiac lesions)3.Otopalatodigital syndrome - type II (hand abnormalities)4.Stickler syndrome (ear anomalies not severe)5.Pierre-Robin syndrome (large fleshy ears)C.NO EAR ANOMALIES1.Pyknodysostosis

Notes:

Destruction Of Temporomandibular Joint *mnemonic:*"HIRT"**H**yperparathyroidism **I**nfection **R**heumatoid arthritis **T**rauma

Notes:

Radiolucent Lesion Of Mandible A.SHARPLY MARGINATED LESION(a)around apex of tooth1.Radicular cyst2.Cementinoma(b)around unerupted tooth1.Dentigerous cyst2.Ameloblastoma(c)unrelated to tooth1.Simple bone cyst2.Fong disease3.Basal cell nevus syndrome B.POORLY MARGINATED LESIONS"floating teeth": suggestive of primary / secondary malignancy resorption of tooth root: hallmark of benign process(a)Infection1.Osteomyelitis: actinomycosis(b)Radiotherapy1.Osteoradionecrosis(c)Malignant neoplasm1.Osteosarcoma (1/3 lytic, 1/3 sclerotic, 1/3 mixed)2.Local invasion from gingival / buccal neoplasms (more common)3.Metastasis from breast, lung, kidney in 1% (in 70% adenocarcinoma)(d)Other1.Eosinophilic granuloma: "floating tooth"2.Fibrous dysplasia3.Osteocementoma4.Ossifying fibroma (very common)

Notes:

Tooth Mass A. CYSTIC LESION 1. **Radicular cyst** (commonest)*Cause:*deep carious lesion / deep filling / traumaSite:intimately associated with apex of nonvital tooth √ apical lucency2.**Ameloblastoma = adamantinoma of jaw**
locally aggressive lesion from enamel-type epithelial tissue elements around tooth; 1/3 arise from dentigerous cyst *Age:*4 - 5th decade; M:F = 1:1Location:mandible (75%), maxilla (25%), in region of bicuspids + molars (angle of mandible commonly affected)√ uni- / multilocular lytic lesion with scalloped margin + cortical expansion √ may be associated with impacted tooth / resorption of the root of a tooth*Prognosis:*frequently local recurrence even more aggressive after excision3.**Primordial cyst**
arising from follicle of tooth that never developed √ absent tooth4.**Giant cell reparative granuloma**
unrelated to tooth (nonodontogenic) √ lucent smooth multiloculated lesion5.**Traumatic bone cyst**
in association with vital tooth √ sharply marginated lucent lesion with fingerlike projections between roots6.**Dentigerous cyst**
=epithelial-lined cyst from odontogenic epithelium developing around unerupted toothLocation:maxilla (may expand into maxillary sinus), posterior mandible√ cystic expansile lesion containing tooth*Cx:*may degenerate into ameloblastoma (rare) B.SCLEROTIC LESION 1.**Cementinoma** = fibro-osteoma = periapical cemental dysplasia*Histo:*spindle-cell fibroblastic proliferation + cementum*Age:*30-40 years of age; most common in womenLocation:in anterior portion of mandible, at apex of vital tooth√ often multicentric√ mixed lucent + sclerotic lesion with little expansion, calcifies with time*DDx:*ossifying fibroma, fibrous dysplasia, Paget disease2.True cementoma = benign cementoblastoma3.Gigantiform cementoma4.**Hypercementosis**
= bulbous enlargement of a root (a)idiopathic(b)associated with Paget disease5.Benign fibro-osseous lesions(a)ossifying fibroma: young adults; mandible > maxilla(b)monostotic fibrous dysplasia: M < F, younger patients(c)condensing osteitis = focal chronic sclerosing osteitis√ near apex of nonvital tooth6.Paget diseaseinvolvement of jaw in 20%; maxilla > mandible Location:bilateral, symmetric involvement√ widened alveolar ridges√ flat palate√ loosening of teeth√ hypercementosis√ may cause destruction of lamina dura7.**Torus mandibularis** = exostosisSite:midline of hard palate; lingual surface of mandible in region of bicuspids

Notes:

Sutural Abnormalities
Wide Sutures =>10 mm at birth, >3 mm at 2 years, >2 mm at 3 years of age; (sutures are splittable up to age 12-15; complete closure by age 30)A.NORMAL VARIANTin neonate + prematurity; growth spurt occurs at 2-3 years and 5-7 years B.CONGENITAL UNDEROSSIFICATIONosteogenesis imperfecta, hypophosphatasia, rickets, hypothyroidism, pyknodysostosis, cleidocranial dysplasia C.METABOLIC DISEASEhypoparathyroidism; lead intoxication; hypo- / hypervitaminosis A D.RAISED INTRACRANIAL PRESSURE*Cause:*(1) intracerebral tumor (2) subdural hematoma (3) hydrocephalus*Age:*seen only if <10 years of ageLocation:coronal > sagittal > lambdoid > squamosal sutureE.INFILTRATION OF SUTURES*Cause:*metastases to meninges from(1) neuroblastoma (2) leukemia(3) lymphoma√ poorly defined marginsF.RECOVERYfrom (1) deprivational dwarfism (2) chronic illness (3) prematurity (4) hypothyroidism ***Craniosynostosis*** =CRANIOSTENOSIS = premature closure of sutures (normally at about 30 years of age)*Age:*often present at birth; M:F = 4:1*Etiology:* A.Primary craniosynostosisB.Secondary craniosynostosis(a)hematologic: sickle cell anemia, thalassemia(b)metabolic: rickets, hypercalcemia, hyperthyroidism, hypervitaminosis D(c)bone dysplasia: hypophosphatasia, achondroplasia, metaphyseal dysplasia, mongolism, Hurler disease, skull hyperostosis, Rubinstein-Taybi syndrome(d)syndromes: Crouzon, Apert, Carpenter, Treacher-Collins, cloverleaf skull, craniotelencephalic dysplasia, arrhinencephaly(e)microcephaly: brain atrophy / dysgenesis(f)after shunting procedures*Types:* Sagittal suture most commonly affected followed by coronal suture 1.**Scaphocephaly** = **Dolichocephaly** (55%)premature closure of sagittal suture (long skull)2.**Brachycephaly** = **Turricephaly** (10%)premature closure of coronal / lambdoid sutures (short tall skull)3.**Plagiocephaly** (7%)unilateral early fusion of coronal + lambdoidal suture (lopsided skull)4.**Trigonocephaly**: premature closure of metopic suture (forward pointing skull)5.**Oxycephaly**: premature closure of coronal, sagittal, lambdoid sutures 6.**Cloverleaf skull** = Kleeblattschädel:intrauterine premature closure of sagittal, coronal, lambdoid sutures;*May be associated with:* thanatophoric dwarfism√ sharply defined thickened sclerotic suture margins√ delayed growth of BPD in early pregnancy

Notes:

Wormian Bones =intrasutural ossicles in lambdoid, posterior sagittal, temporosquamosal sutures; normal up to 6 months of age (most frequently)*mnemonic*:"PORK CHOPS I"**P**yknodysostosis **O**steogenesis imperfecta **R**ickets in healing phase **K**inky hair syndrome **C**leidocranial dysostosis **H**ypothyroidism / **H**ypophosphatasia **O**topalatodigital syndrome **P**rimary acroosteolysis (Hajdu-Cheney) / **P**achydermoperiostosis / **P**rogeria **S**yndrome of Down **I**diopathic

Notes:

Increased Skull Thickness A. GENERALIZED 1.Chronic severe anemia (eg, thalassemia, sickle cell disease)2.Cerebral atrophy following shunting of hydrocephalus3.Engelmann disease: mainly skull base4.Hyperparathyroidism5.Acromegaly6.OsteopetrosisB. FOCAL 1.Meningioma2.Fibrous dysplasia3.Paget disease4.Dyke-Davidoff-Mason syndrome5.Hyperostosis frontalis interna=dense hyperostosis of inner table of frontal bone; M < F *mnemonic:*"HIPFAM"**H**yperostosis frontalis interna **I**diopathic **P**aget disease **F**ibrous dysplasia **A**nemia (sickle cell, iron deficiency, thalassemia, spherocytosis) **M**etastases ***Hair-on-end Skull*** *mnemonic:*"HI NEST"**H**ereditary spherocytosis **I**ron deficiency anemia **N**euroblastoma Enzyme deficiency (glucose-6-phosphate dehydrogenase deficiency causes hemolytic anemia) **S**ickle cell disease **T**halassemia major ***Leontiasis Ossea*** =overgrowth of facial bones causing leonine (lionlike) facies1.Fibrous dysplasia2.Paget disease3.Craniometaphyseal dysplasia4.Hyperphosphatasia

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Notes:

Abnormally Thin Skull A. GENERALIZED 1.Obstructive hydrocephalus2.Cleidocranial dysostosis3.Progeria4.Rickets5.Osteogenesis imperfecta6.CraniolacuniaB. FOCAL 1. Neurofibromatosis 2. Chronic subdural hematoma 3. Arachnoid cyst
Inadequate Calvarial Calcification 1.Achondroplasia2.Osteogenesis imperfecta3.Hypophosphatasia

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Notes:

Osteolytic Lesion Of Skull A. NORMAL VARIANT 1.Emissary veinconnecting venous systems inside + outside skull √ bony channel <2 mm in width2.Venous lake=outpouching of diploic vein√ extremely variable in size, shape, and number√ irregular well-demarcated contour3.Pacchionian granulations√ usually multiple lesions with irregular contour in parasagittal location (within 3 cm of superior sagittal sinus) primarily involving the inner table*Associated with:* impressions by arachnoid granulations4.Parietal foraminanonossification of embryonal rests in parietal fissure; bilateral at superior posterior angles of parietal bone; hereditary transmission B. TRAUMA 1.Surgical burr hole2.Leptomeningeal cystC. INFECTION 1.Osteomyelitis3.Syphilis2.Hydatid disease4.TuberculosisD. CONGENITAL 1.Epidermoid / dermoid2.Neurofibromatosis (asterion defect)3.Meningoencephalocele4.Fibrous dysplasia5.Osteoporosis circumscripta of Paget diseaseE. BENIGN TUMOR 1.Hemangioma2.Brown tumor3.Eosinophilic granulomaF. MALIGNANT TUMOR 1.Solitary / multiple metastases2.Multiple myeloma3.Leukemia4.Neuroblastoma ***Solitary Lytic Lesion In Skull*** *mnemonic:*"HELP MFT HOLE"**H**emangioma **E**pidermoid / dermoid **L**eptomeningeal cyst **P**ostop, **P**aget disease **M**etastasis, **M**yeloma **F**ibrous dysplasia Tuberculosis **H**yperparathyroidism Osteomyelitis **L**ambdoid defect (neurofibromatosis) **E**osinophilic granuloma ***Multiple Lytic Lesions In Skull*** *mnemonic:*"BAMMAH"**B**rown tumor **A**VM **M**yeloma **M**etastases **A**myloidosis **H**istiocytosis

Notes:

Lytic Area In Bone Flap *mnemonic:*"RATI"**R**adiation necrosis **Avascular necrosis** **Tumor** Infection

Notes:

Button Sequestrum *mnemonic:*"TORE ME"**T**uberculosis **O**steomyelitis **R**adiation **E**osinophilic granuloma **M**etastasis **E**pidermoid

Notes:

Absent Greater Sphenoid Wing *mnemonic:*"M FOR MARINE"**M**eningioma **F**ibrous dysplasia **O**ptic glioma **R**elapsing hematoma **M**etastasis **A**neurysm **R**etinoblastoma **I**diopathic **N**eurofibromatosis **E**osinophilic granuloma

Notes:

Absence Of Innominate Line =OBLIQUE CAROTID LINE=vertical line projecting into orbit (on PA skull film) produced by orbital process of sphenoidA.CONGENITAL1.Fibrous dysplasia2.NeurofibromatosisB.INFECTIONC.TUMOR

Notes:

Widened Superior Orbital Fissure *mnemonic:*"A FAN"**A**neurysm (internal carotid artery) **F**istula (cavernous sinus) **A**denoma (pituitary) **N**eurofibroma

Notes:

Tumors Of The Central Skull Base A.DEVELOPMENTAL1.EncephaloceleB.INFECTION / INFLAMMATION1.Extension from paranasal sinus / mastoid infection2.Complication of trauma3.Fungal disease: mucormycosis in diabetics, aspergillosis in immunosuppressed patients4.Sinus + nasopharyngeal sarcoidosis5.Radiation necrosisC.BENIGN1.Juvenile angiofibroma2.Meningioma3.Chordoma4.Pituitary tumor5.Paget disease6.Fibrous dysplasiaD.MALIGNANT1.Metastasis: prostate, lung, breast2.Chondrosarcoma3.Nasopharyngeal carcinoma4.Rhabdomyosarcoma5.Perineural tumor spread: head + neck neoplasm

Notes:

Craniovertebral Junction **Anomaly** ***Basilar Invagination*** =primary developmental anomaly with abnormally high position of vertebral column prolapsing into skull base*Associated with:*Chiari malformation, syringohydromyelia in 25-35%*Cause:* 1.Condylus tertius = ossicle at distal end of clivus√ pseudojoint with odontoid process / anterior arch of C12.Condylar hypoplasia√ lateral masses of atlas may be fused to condyles√ violation of Chamberlain line√ widening of atlantooccipital joint axis angle √ tip of odontoid >10 mm above bimastoid line3.Basiocciput hypoplasia√ shortening of clivus√ violation of Chamberlain line√ clivus-canal angle typically decreased4.Atlantooccipital assimilation=complete / partial failure of segmentation between skull + 1st cervical vertebra√ violation of Chamberlain line√ clivus-canal angle decreased*May be associated with:*fusion of C2 + C3*Cx:*atlantoaxial subluxation (50%); sudden death ● limitation in range of motion of CVJ√ abnormal craniometry√ C-spine + foramen magnum bulge into cranial cavity√ elevation of posterior arch of C1 ***Basilar Impression*** =acquired form of basilar invagination with bulging of C-spine and foramen magnum into cranial cavity*Cause:*Paget disease, Osteomalacia, rickets, fibrous dysplasia, hyperparathyroidism, Hurler syndrome, osteogenesis imperfecta, skull base infection*mnemonic:*"PF ROACH"**P**aget disease **F**ibrous dysplasia **R**ickets **O**steogenesis imperfecta, **O**steomalacia **A**chondroplasia **C**leidocranial dysplasia **H**yperparathyroidism, **H**urler syndrome

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Notes:

Platybasia =anthropometric term referring to flattening of skull base*May be associated with:*basilar invagination ● cord symptoms√ craniovertebral = clivus-canal angle becomes acute (<150°)√ Welcher basal angle = sphenoid angle >140°√ bowstring deformity of cervicomedullary junction

Notes:

Atlas Anomalies A.POSTERIOR ARCH ANOMALIES

Atlas Ossification Center
secondary centers

Axis Ossification Center
secondary centers

1.Posterior atlas arch rachischisis (4%)Location:midline (97%), lateral through sulcus of vertebral artery (3%)√ absence of arch-canal line (LAT view)√ superimposed on odontoid process / axis body simulating a fracture (open-mouth odontoid view)2.Total aplasia of posterior atlas arch3.Keller-type aplasia with persistence of posterior tubercle4.Aplasia with uni- / bilateral remnant + midline rachischisis5.Partial / total hemiaplasia of posterior archB. ANTERIOR ARCH ANOMALIES 1.Isolated anterior arch rachischisis (0.1%)2.Split atlas = anterior + posterior arch rachischisis√ plump rounded anterior arch overlapping the odontoid process making identification of predental space impossible (LAT view)√ duplicated anterior margins (LAT view)

Notes:

Axis Anomalies

1.Persistent ossiculum terminale = Bergman ossicle√ unfused odontoid process >12 years of age*DDx:*type 1 odontoid fracture2.Odontoid aplasia (extremely rare)3.Os odontoideum=independent os cephalad to axis body in location of odontoid process√ absence of odontoid process√ anterior arch of atlas hypertrophic + situated too far posterior in relation to axis body*Cx:*atlantoaxial instability*DDx:*type 2 odontoid fracture (uncorticated margin) ***Odontoid Erosion*** *mnemonic:*"P LARD"**P**soriasis **L**upus erythematosus **A**nkylosing spondylitis **R**heumatoid arthritis **D**own syndrome

Notes:

Atlantoaxial Subluxation =displacement of atlas with respect to axis(1)Posterior atlantoaxial subluxation (rare)(2)Anterior atlantoaxial subluxation (common)=distance between dens + anterior arch of C1 (measurement along midplane of atlas on lateral view):(a)predental space:>2.5 mm; >4.5 mm (in children)(b)retrodental space:<18 mm*Causes of subluxation:* (a)Congenital1.Occipitalization of atlas0.75% of population; fusion of basion + anterior arch of atlas 2.Congenital insufficiency of transverse ligament3.Os odontoideum / aplasia of dens4.Down syndrome (20%)5.Morquio syndrome6.Bone dysplasia(b)Arthritisdue to laxity of transverse ligament or erosion of dens 1.Rheumatoid arthritis2.Psoriatic arthritis3.Reiter syndrome4.Ankylosing spondylitis5.SLErare: in gout + CPPD (c)Inflammatory processPharyngeal infection in childhood, retropharyngeal abscess, coryza, otitis media, mastoiditis, cervical adenitis, parotitis, alveolar abscess dislocation 8-10 days after onset of symptoms(d)Trauma (very rare without odontoid fracture)(e)Marfan disease *mnemonic:*"JAP LARD"**J**uvenile rheumatoid arthritis **A**nkylosing spondylitis **P**soriatic arthritis **L**upus erythematosus **A**ccident (trauma) **R**etropharyngeal abscess, **R**heumatoid arthritis **D**own syndrome PSEUDOSUBLUXATION =ligamentous laxity in infants allows for movement of the vertebral bodies on each other, esp. C2 on C3

Notes:

SPINAL DYSRAPHISM
=abnormal / incomplete fusion of midline embryologic mesenchymal, neurologic, bony structuresExternal signs (in 50%) ● subcutaneous lipoma ● spastic gait disturbance ● hypertrichosis ● foot deformities ● pigmented nevi ● absent tendon reflexes ● skin dimple ● sinus tract ● bladder + bowel dysfunction ● pathologic plantar response

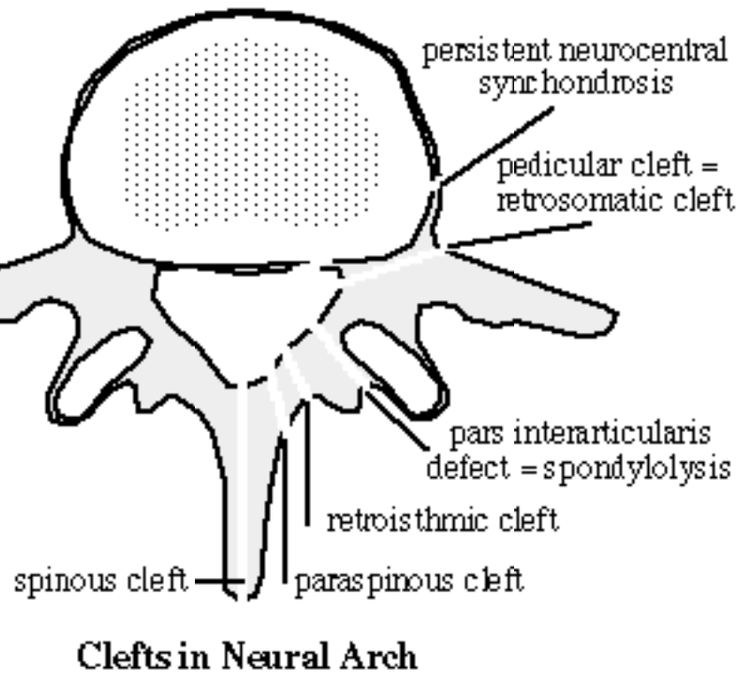
Spina Bifida Segmentation Anomalies Of Vertebral Bodies

Notes:

Spina Bifida =incomplete closure of bony elements of the spine (lamina + spinous processes) posteriorly***Spina Bifida Occulta*** = OCCULT SPINAL DYSRAPHISM =skin covered defect; 15% of spinal dysraphism ● rarely leads to neurologic deficit in itself*Associated with:* vertebral defect (85 - 90%), lumbosacral dermal lesion (80%), ie, hairy tuft, dimple, sinus, nevus, hyperpigmentation, hemangioma, subcutaneous mass 1.Diastematomyelia2.Lipomeningocele3.Tethered cord syndrome4.Filum terminale lipoma5.Intraspinal dermoid6.Epidermoid cyst7.Myelocystocele8.Split notochord syndrome9.Meningocele10.Dorsal dermal sinus11.Tight filum terminale syndrome ***Spina Bifida Aperta*** =SPINA BIFIDA CYSTICA=posterior protrusion of all / parts of the contents of the spinal canal through a bony spina bifida; 85% of spinal dysraphism ● associated with neurologic deficit in >90%1.Simple meningocele=herniation of CSF-filled sac without neural elements2.Myelocele=midline plaque of neural tissue lying exposed at the skin surface3.Myelomeningocele=a myelocele elevated above skin surface by expansion of subarachnoid space ventral to neural plaque4.Myeloschisis= surface presentation of neural elements completely uncovered by meninges

Notes:

Segmentation Anomalies Of Vertebral Bodies during 9 - 12th week of gestation two ossification centers form for the ventral + dorsal half of vertebral body



Clefts in Neural Arch

1.**Asomia** = agenesis of vertebral body√ complete absence of vertebral body√ hypoplastic posterior elements may be present2.**Hemivertebra**
(a)Unilateral wedge vertebra√ right / left hemivertebra√ scoliosis at birth(b)Dorsal hemivertebra√ rapidly progressive kyphoscoliosis(c)Ventral hemivertebra (extremely rare)3.**Coronal cleft**
=failure of fusion of anterior + posterior ossification centers*May be associated with:*premature male infant, Chondrodystrophia calcificans congenitaLocation:usually in lower thoracic + lumbar spine√ vertical radiolucent band just behind midportion of vertebral body; disappears mostly by 6 months of life4.**Butterfly vertebra**
=failure of fusion of lateral halves secondary to persistence of notochordal tissue*May be associated with:*anterior spina bifida ± anterior meningocele√ widened vertebral body with butterfly configuration (AP view)√ adaptation of vertebral endplates of adjacent vertebral bodies5.**Block vertebra**
= congenital vertebral fusion Location:lumbar / cervical√ height of fused vertebral bodies equals the sum of heights of involved bodies + intervertebral disk√ "waist" at level of intervertebral disk space6.Hypoplastic vertebra7.Klippel-Feil syndrome

Notes:

Small Vertebral Body 1.Radiation therapy during early childhood in excess of 1000 rads 2.Juvenile rheumatoid arthritisLocation:cervical spine√ atlantoaxial subluxation may be present√ vertebral fusion may occur3.Eosinophilic granulomaLocation:lumbar / lower thoracic spine√ compression deformity / vertebra plana4.Gaucher disease=deposits of glucocerebrosides within RES√ compression deformity5.Platyspondyly generalisata=flattened vertebral bodies associated with many hereditary systemic disorders (achondroplasia, spondyloepiphyseal dysplasia tarda, mucopolysaccharidosis, osteopetrosis, neurofibromatosis, osteogenesis imperfecta, thanatophoric dwarfism)√ disk spaces of normal height ***Vertebra Plana*** *mnemonic:*" FETISH"**F**racture (trauma, osteogenesis imperfecta) **E**osinophilic granuloma **T**umor (metastasis, myeloma, leukemia) **I**nfection **S**teroids (avascular necrosis) **H**emangioma ***Signs Of Acute Vertebral Collapse On MRI*** 1.OSTEOPOROSIS√ retropulsion of posterior bone fragment2.MALIGNANCY√ epidural soft-tissue mass√ no residual normal marrow signal intensity√ abnormal enhancement

Notes:

Enlarged Vertebral Body 1.Paget disease√ "picture framing"; bone sclerosis2.Gigantism√ increase in height of body + disk3.Myositis ossificans progressiva√ bodies greater in height than width√ osteoporosis√ ossification of ligamentum nuchae

Notes:

Enlarged Vertebral Foramen 1.Neurofibroma2.Congenital absence / hypoplasia of pedicle3.Dural ectasia (Marfan syndrome, Ehlers-Danlos syndrome)4.Intraspinal neoplasm5.Metastatic destruction of pedicle

Notes:

Cervical Spine Fusion *mnemonic:*"SPAR BIT"**S**enile hypertrophic ankylosis (DISH) **P**soriasis, **P**rogressive myositis ossificans **A**nkylosing spondylitis **R**eiter disease, **R**heumatoid arthritis (juvenile) **B**lock vertebra (Klippel-Feil) **I**nfection (TB) **T**rauma

Notes:

Vertebral Border Abnormality *Straightening Of Anterior Border* 1.Ankylosing spondylitis2.Paget disease3.Psoriatic arthritis4.Reiter disease5.Rheumatoid arthritis6.Normal variant ***Anterior*** Scalloping Of Vertebrae 1.Aortic aneurysm2.Lymphadenopathy3.Tuberculosis4.Multiple myeloma (paravertebral soft-tissue mass) ***Posterior Scalloping Of Vertebrae*** in conditions associated with dural ectasia A.INCREASED INTRASPINAL PRESSURE1.Communicating hydrocephalus2.EpendymomaB.MESENCHYMAL TISSUE LAXITY1.Neurofibromatosis (secondary to dural ectasia / spinal tumor)2.Marfan syndrome3.Ehlers-Danlos syndrome4.Posterior meningoceleC.BONE SOFTENING1.Mucopolysaccharidoses: Hurler, Morquio, Sanfilippo2.Acromegaly (lumbar vertebrae)3.Ankylosing spondylitis (lax dura acting on osteoporotic vertebrae)4.Achondroplasia *mnemonic:*"DAMN MALE SHAME"**D**ermoid **A**nkylosing spondylitis **M**eningioma **N**eurofibromatosis **M**arfan syndrome **A**cromegaly **L**ipoma **E**pendymoma **S**yringohydromyelia **H**ydrocephalus **A**chondroplasia **M**ucopolysaccharidoses **E**hlers-Danlos syndrome

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Notes:

Bony Projections From Vertebra 1.Hurler syndrome = gargoylism√ rounded appearance of vertebral bodies√ mild kyphotic curve with smaller vertebral body at apex of kyphosis displaying tonguelike beak at anterior half (usually at T12 / L1)√ "step-off" deformities along anterior margins2.Hunter syndromeless severe changes than in Hurler syndrome 3.Morquio disease√ flattened + widened vertebral bodies√ anterior "tonguelike" elongation of central portion of vertebral bodies4.Hypothyroidism = cretinism√ small flat vertebral bodies√ anterior "tonguelike" deformity (in children only)√ widened disk spaces + irregular endplates5.Spondylosis deformans√ osteophytosis along anterior + lateral aspects of endplates with horizontal + vertical course as a result of shearing of the outer annular fibers (Sharpey fibers connecting the annulus fibrosus to adjacent vertebral body)6.Diffuse idiopathic skeletal hyperostosis (DISH)= Forestier disease √ flowing calcifications + ossifications along anterolateral aspect of >4 contiguous thoracic vertebral bodies ± osteophytosis7.Ankylosing spondylitis√ bilateral symmetric syndesmophytes (ossification of annulus fibrosus)√ "bamboo spine"√ "discal ballooning" = biconvex intervertebral disks secondary to osteoporotic deformity of endplates√ straightening of anterior margins of vertebral bodies (erosion)√ ossification of paraspinal ligaments8.Fluorosis√ vertebral osteophytosis + hyperostosis√ sclerotic vertebral bodies + kyphoscoliosis√ calcification of paraspinal ligaments ***Spine Ossification*** 1.Syndesmophyte = ossification of annulus fibrosus*Associated with:*ankylosing spondylitis, ochronosis2.Osteophyte=ossification of anterior longitudinal ligament*Associated with:*osteoarthritis3.Flowing anterior ossification=ossification of disk, anterior longitudinal ligament, paravertebral soft tissues*Associated with:*diffuse idiopathic skeletal hyperostosis4.Paravertebral ossification*Associated with:*psoriatic arthritis, Reiter syndrome

Notes:

Vertebral Endplate Abnormality 1.Osteoporosis (senile / steroid-induced)√ "Fish-mouth vertebrafish-mouth vertebrae" (DDx: osteomalacia, Paget disease, hyperparathyroidism)√ bone sclerosis along endplates2.Sickle cell disease√ "H-vertebrae" = compression of central portions from subchondral infarcts (DDx: other anemias, Gaucher disease)3.Schmorl node=intraosseous herniation of nucleus pulposus at center of weakened endplate in disk herniation / Scheuermann disease4.Limbus vertebrae=intraosseous herniation of disk material at junction of vertebral bony rim of centra + endplate (anterosuperior corner)5."Ring" epiphysis=normal aspect of developing vertebra (between 6 and 12 years of age)√ small steplike recess at corner of anterior edge of vertebral body6.Renal osteodystrophy√ "rugger-jersey spine" = horizontal bands of increased opacity subjacent to vertebral endplates7.Myelofibrosis√ "rugger-jersey spine"8.Osteopetrosis√ "sandwich" / "hamburger" vertebrae = sclerotic endplates alternate with radiolucent midportions of vertebral bodies

Notes:

Bullet-shaped Vertebral Body *mnemonic:*"HAM"**H**ypothyroidism **A**chondroplasia **M**orquio syndrome

Notes:

Bone-within-bone Vertebra ="ghost vertebra" following stressful event during vertebral growth phase in childhood1.Stress line of unknown cause2.Leukemia3.Heavy metal poisoning4.Thorotrast injection, TB5.Rickets6.Scurvy7.Hypothyroidism8.Hypoparathyroidism

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Notes:

Ivory Vertebra *mnemonic:*"LOST FROM CHOMP"**L**ymphoma **O**steopetrosis **S**ickle cell disease **T**rauma **F**luorosis **R**enal osteodystrophy **O**steoblastic metastasis **M**yelosclerosis **C**hronic sclerosing osteomyelitis Hemangioma **O**steosarcoma **M**yeloma **P**aget disease

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Notes:

Expansile Lesion Of Vertebrae A.INVOLVEMENT OF MULTIPLE VERTEBRAEMetastases, multiple myeloma / plasmacytoma, lymphoma, hemangioma, Paget disease, angiosarcoma, eosinophilic granuloma B.INVOLVEMENT OF TWO / MORE CONTIGUOUS VERTEBRAEOsteochondroma, chordoma, aneurysmal bone cyst, myeloma C.BENIGN LESION1.Osteochondroma (1-5% with solitary osteochondromas, 7-9% with hereditary multiple exostoses) commonly cervical, esp. C2; commonly rising from posterior elements2.Osteoblastoma (30-40% in spine)M:F = 2:1; equal distribution in spine; posterior elements (lamina, pedicle), may involve body if large; expansile lesion with sclerotic / shell-like rim, foci of calcified tumor matrix in 50%3.Giant cell tumor (5-7% in spine)commonly sacrum, expansile lytic lesion of vertebral body with well-defined borders; secondary invasion of posterior elements; malignant degeneration in 5-20% after radiation therapy4.Osteoid osteoma (10-25% in spine)commonly lower thoracic / upper lumbar spine, posterior elements (pedicle, lamina, spinous process), painful scoliosis with concavity toward lesion5.Aneurysmal bone cyst (12-30% in spine)thoracic > lumbar > cervical spine, posterior elements with frequent extension into vertebral bodies, well-defined margins, may arise from primary bone lesion (giant cell tumor, fibrous dysplasia) in 50%, may involve two contiguous vertebrae 6.Hemangioma (30% in spine)10% incidence in general population; commonly lower thoracic / upper lumbar spine, vertebral body, "accordion" / "corduroy" appearance 7.Hydatid cyst (1% in spine)slow-growing destructive lesion, well-defined sclerotic borders, endemic areas 8.Paget diseasevertebral body ± posterior elements, enlargement of bone, "picture framing"; bone sclerosis 9.Eosinophilic granuloma (6% in spine)most often cervical / lumbar spine, vertebral body, "vertebra plana"; multiple involvement common 10.Fibrous dysplasia (1% in spine)vertebral body, nonhomogeneous trabecular "ground glass" appearance 11.Enostosis (1-14% in spine)Location:T1-T7 > L2-L3 D.MALIGNANT1.Chordoma (15% in spine)most common nonlymphoproliferative primary malignant tumor of the spine in adults; particularly C2, within vertebral body; violates disk space 2.Metastases (especially from lung, breast)*Age:*>50 years of age;*Clue:*pedicles often destroyed3.Multiple myeloma / plasmacytoma*Clue:*vertebral pedicles usually spared4.Angiosarcoma10% involve spine, most commonly lumbar 5.Chondrosarcoma (3-12% in spine)2nd most common nonlymphoproliferative primary malignant tumor of the spine in adults Site:vertebral body (15%), posterior elements (40%), both (45%)√ involvement of adjacent vertebra by extension through disk (35%)6.Ewing sarcoma and PNETmost common nonlymphoproliferative primary malignant tumor of the spine in children; metastases more common than primary Site:vertebral body with extension to posterior elements√ diffuse sclerosis + osteonecrosis (69%)7.Osteosarcoma (0.6-3.2% in spine)*Average age:*4th decadeLocation:lumbosacral segmentsSite:vertebral body, posterior elements (10-17%)√ may present as "ivory vertebra"8.Lymphoma ***Blowout Lesion Of Posterior Elements*** *mnemonic:*"GO APE"**G**iant cell tumor **O**steoblastoma **A**neurysmal bone cyst **P**lasmacytoma **E**osinophilic granuloma

Notes:

Bone Tumors Favoring Vertebral Bodies *mnemonic:*"CALL HOME"**C**hordoma **A**neurysmal bone cyst **L**eukemia **L**ymphoma **H**emangioma **O**steoid osteoma, **O**steoblastoma **M**yeloma, **M**etastasis **E**osinophilic granuloma ***Primary Vertebral Tumors In Children*** in order of frequency: 1.Osteoid osteoma2.Benign osteoblastoma3.Aneurysmal bone cyst4.Ewing sarcoma

Notes:

Primary Tumor Of Posterior Elements *mnemonic:*"A HOG"**A**neurysmal bone cyst **H**ydatid cyst, **H**emangioma Osteoblastoma, **O**steoid osteoma **G**iant cell tumor

Notes:

Vacuum Phenomenon In Intervertebral Disk Space =liberation of nitrogen gas from surrounding tissues into clefts with an abnormal nucleus or annulus attachment*Incidence:*in up to 20% of plain radiographs / in up to 50% of spinal CT in patients > age 40*Cause:* 1.Primary / secondary degeneration of nucleus pulposus2.Intraosseous herniation of disk (= Schmorl node)3.Spondylosis deformans4.Adjacent vertebral metastatic disease with vertebral collapse5.Infection (extremely rare)

Notes:

Intervertebral Disk Calcification *mnemonic:*" A DISC SO WHITE"**A**myloidosis, **A**cromegaly **D**egenerative Infection **S**pinal fusion **C**PPD **S**pondylitis ankylosing **O**chronosis **W**ilson disease **H**emochromatosis, **H**omocystinuria, **H**yperparathyroidism **I**diopathic skeletal hyperostosis **T**raumatic **E**tceteras: Gout and other causes of chondrocalcinosis

Notes:

Intervertebral Disk Ossification *Associated with:*fusion of vertebral bodies1.Ankylosing spondylitis2.Ochronosis3.Sequela of trauma4.Sequela of disk-space infection5.Degenerative disease

Notes:

Schmorl Node =chondrification defects where periosteal vessels penetrate cartilage plates of disk√ concave defects at upper and lower vertebral endplates with sharp margins produced by superior / inferior herniation of disk materialMR: √ node of similar signal intensity as disk√ low signal intensity of rim√ associated with narrowed disk space*DDx: mnemonic:* "SHOOT"**S**cheuermann disease **H**yperparathyroidism **O**steoporosis **O**steomalacia **T**rauma

Notes:

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Intramedullary Lesion 15% of spinal canal tumors in adults; 6% of spinal cord tumors in children (1/3 of spinal neoplasms in childhood) A.TUMOR(a)primary:1.Ependymoma (60% of all spinal cord tumors)2.Astrocytoma (25%)3.Oligodendroglioma (3%)4.Epidermoid, dermoid, teratoma (1-2%)5.Lipoma (1%)Location:-cervical region: astrocytoma-thoracic region: teratoma-dermoid, astrocytoma-lumbar region: ependymoma, dermoid(b)metastatic: eg, malignant melanoma, breast, lungB.CYSTIC LESIONmay show delayed filling of cystic space on CT-myelography 1.Syringomyelia2.Hydromyelia3.Reactive cyst4.Hemangioblastoma (2 - 4%)C.VASCULAR1.Cord concussion = reversible local edema2.Hemorrhagic contusion3.Cord transection4.AVMD.CHRONIC INFECTION1.Sarcoid2.Transverse myelitis3.Multiple sclerosis *mnemonic:*"IM ASHAMED"**I**nflammation (multiple sclerosis, sarcoidosis, myelitis) **M**edulloblastoma **A**strocytoma **S**yringomyelia / hydromyelia **H**ematoma, **H**emangioblastoma **A**rteriovenous malformation **M**etastasis **E**pependymoma **D**ermoid

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Notes:

Intradural Extramedullary Mass 1.Neurofibroma (25-35%)2.Meningioma (25-45% of all spinal tumors)3.Lipoma4.Dermoidcommonly conus / cauda equina; associated with spinal dysraphism (1/3) 5.Ependymomacommonly filum terminale; NO spinal dysraphism 6."Drop metastases" from CNS tumors7.Metastases from outside CNS8.Arachnoid cyst9.Neurenteric cyst10.Hemangioblastoma *mnemonic:*"MAMA N"**M**etastasis **A**rachnoiditis **M**eningioma **A**VM, **A**rachnoid cyst **N**eurofibroma

Notes:

Epidural Extramedullary Lesion Epidural space = space between dura mater + bone containing epidural venous plexus, lymphatic channels, connective tissue, fat *Incidence:*30% of all spinal tumorsA.TUMOR(a) benign 1.Dermoid, epidermoid2.Lipoma: over several segments3.Fibroma4.Neurinoma (with intradural component)5.Meningioma (with intradural component)6.Ganglioneuroblastoma, ganglioneuroma(b) malignant 1.Hodgkin disease2.Lymphoma: most commonly in dorsal space3.Metastasis: breast, lung - most commonly from involved vertebrae without extension through dura4.Paravertebral neuroblastomaB.DISK DISEASE1.Bulging disk2.Herniated nucleus pulposus3.Sequestered nucleus pulposusC.OSSEOUS: spinal stenosis, spondylosisD.INFLAMMATION: epidural abscessE.HEMATOMAF.SYNOVIAL CYST *mnemonic:*"MANDELIN"**M**etastasis (drop mets from CNS tumor), **M**eningioma **A**rachnoiditis, **A**rachnoid cyst **N**eurofibroma **D**ermoid / epidermoid **E**pendymoma **L**ipoma **I**nfection (TB, Cysticercosis) **N**ormal but tortuous roots

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Notes:

Tumors Of Nerve Roots And Nerve Sheaths =NEURINOMA A.ARISING FROM NERVE SHEATH1.**Schwannoma**=encapsulated benign slowly growing neoplasm arising from Schwann cellsSchwann cell = cell that surrounds cranial, spinal, and peripheral nerves producing myelin sheath around axons thus providing mechanical protection, serving as a tract for nerve regeneration ◊NOTE that myelin sheaths within brain substance are made by oligodendrocytes!◊Usually sporadic tumor, but 5 - 20% of patients with solitary intracranial schwannomas have type 2 neurofibromatosis!*Histo:*cellular component (Antoni type A tissue) + myxoid component (Antoni type B tissue)Location: (a)extracranial: (most commonly) cervical spine roots, vagus nerve, sympathetic plexus(b)intracranial: mostly from sensory nerves, vestibulocochlear (VIII) cranial nerve (most common), trigeminal (V) cranial nerve (2nd most common)√ solitary fusiform well-encapsulated lesionMR: √ dark line surrounding the lesion (= capsule) frequently seen2.**Neurofibroma**= tumor of nerve sheath composed of Schwann cells + fibroblasts with involvement of nerve, nerve fibers run through mass *Histo:*swirls of neuronal elements*Associated with:* neurofibromatosis type 1;M:F = 1:1 ◊Potential for malignant transformation!◊The spinal neurofibroma is rarely sporadic and usually a sign of type 1 neurofibromatosis!◊Only 10% of patients with neurofibromas have von Recklinghausen disease!Location:any level, but particularly cervical(a)peripheral nerves√ nonencapsulated well-circumscribed fusiform mass of peripheral nerves(b) intradural extramedullary mass√ well-defined mass with dumbbell configuration (= extradural component extends through neural foramen)√ widening of intervertebral foramen + erosion of pedicles√ scalloping of vertebral bodies√ hypodense (CHARACTERISTIC) approaching characteristics of water / isodense to skeletal muscle√ usually NO contrast enhancementMR: √ homogeneous mass isointense to cord on T1WI√ hyperintense tumor on T2WI compared with surrounding fat√ "target sign" = low signal-intensity center on T2WI (due to collagen + condensed Schwann cells)*DDx:*conjoined nerve root sleeveB.ORIGINATING FROM NERVE1.**Neuroma**= posttraumatic lesion forming at end of severed nerve 2.Neurilemmoma**Neurilemmoma**=nerve fibers diverge and course over the surface of the tumor mass

Notes:

Cord Lesions A.INFLAMMATION1.Multiple sclerosis2.Acute disseminated encephalomyelitis3.Acute transverse myelitis√ involves half the cross-sectional area of cord4.Lyme disease5.Devic syndromeB.INFECTION1.Cytomegalovirus2.Progressive multifocal leukoencephalopathy3.HIVC.VASCULAR1.Anterior spinal artery infarct √ affects central gray matter first√ extends to anterior two-thirds of cord2.Venous infarct / ischemia√ starts centrally progressing centripetallyD.NEOPLASM

Notes:

Cord Atrophy 1.Multiple sclerosis2.Amyotrophic lateral sclerosis3.Cervical spondylosis4.Sequelae of trauma5.Ischemia6.Radiation therapy7.AVM of cord

Notes:

Delayed Uptake Of Water-Soluble Contrast In Cord lesion 1.Syringohydromyelia2.Cystic tumor of cord3.Osteomalaciaexceedingly rare:4.Demyelinating disease5.Infection6.Infarction

Notes:

Extra-arachnoid Myelography A.SUBDURAL INJECTION√ spinal cord, nerve roots, blood vessels not outlined√ irregular filling defects√ slow flow of contrast material √ CSF pulsations diminished√ contrast material pools at injection site within anterior / posterior compartmentsB.EPIDURAL INJECTION√ contrast extravasation along nerve roots√ contrast material lies near periphery of spinal canal√ intraspinal structures are not well outlined

Notes:

Destructive Sacral Lesion *mnemonic:*"SPACEMON"**S**arcoma **P**lasmacytoma **A**neurysmal bone cyst **C**hordoma **E**pendymoma **M**etastasis **O**steomyelitis **N**euroblastoma ***Sacral Neoplasms*** 1.Metastases from breast, prostate, kidney, cervix, colon2.Multiple myeloma3.Chordoma (most common primary)4.Giant cell tumor (most common benign tumor)5.Sacrococcygeal teratoma

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Notes:

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SPINAL FIXATION DEVICES

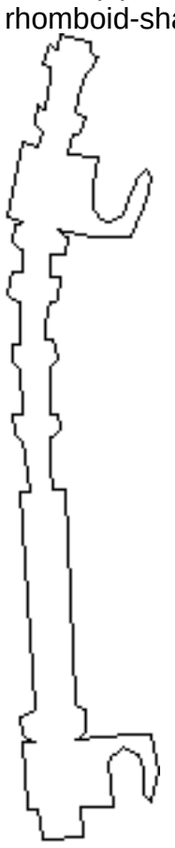
Function:(1)to restore anatomic alignment in fractures (fracture reduction)(2)to stabilize degenerative disease(3)to correct congenital deformities (scoliosis)(4)to replace diseased / abnormal vertebrae (infection, tumor)

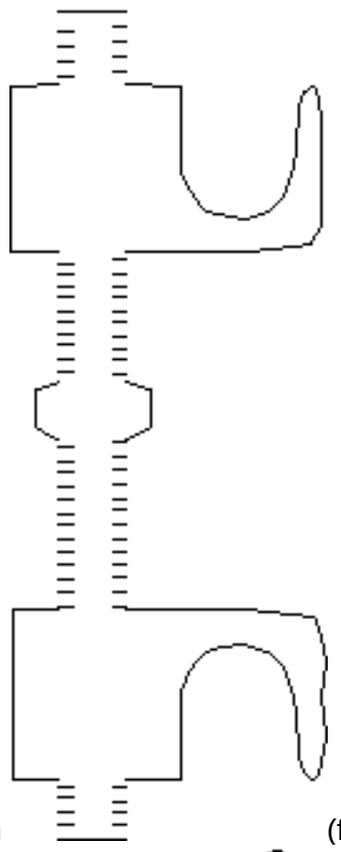
Posterior Fixation Devices Anterior Fixation Devices

Notes:

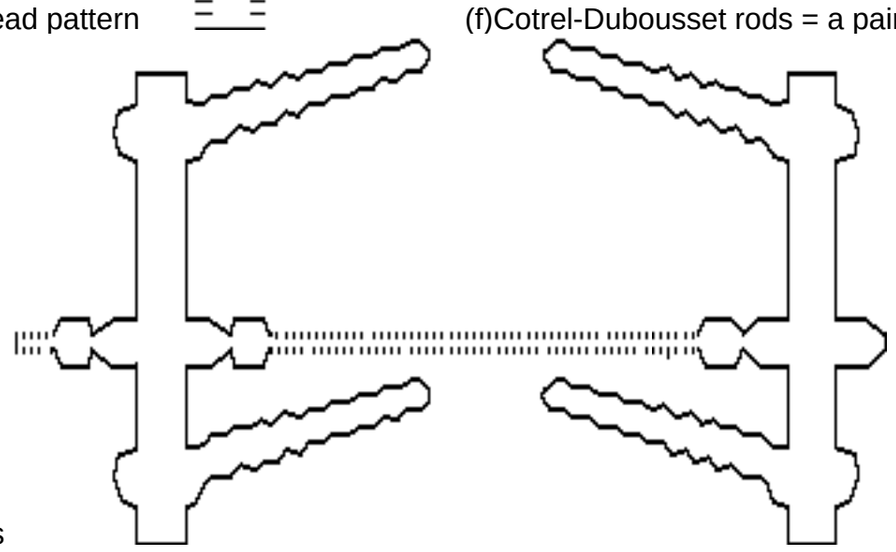
LIPPINCOTT WILLIAMS & WILKINS
A Wolters Kluwer Company

Posterior Fixation Devices using paired / unpaired rods attached with 1.Sublaminar wiring= passing a wire around lamina + rod 2.Interspinous wiring= passing a wire through a hole in the spinous process; a Drummond button prevents the wire from pulling through the bone 3.Subpars wiring= passing a wire around the pars interarticularis 4.Laminar / sublaminar hooksused on rods for compression / distraction forces to be applied to pedicles / laminae (a)upgoing hook curves under lamina(b)downgoing hook curves over lamina5.Pedicle / transpedical screws6.Rods(a)Luque rod = straight / L-shaped smooth rod 6-8 mm in diameter(b)O-ring fixator, rhomboid-shaped bar, Luque rectangle, segmental rectangle = preshaped loop to form a flat rectangle(c)Harrington distraction rod(d)Harrington compression rod



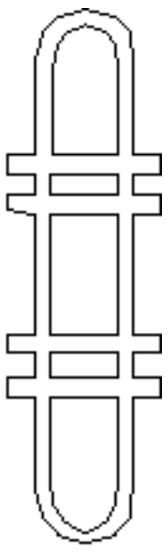


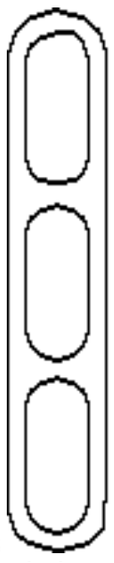
(e)Knodt rod = threaded distraction rod with a central fixed nut (turnbuckle) and opposing thread pattern



(f)Cotrel-Dubousset rods = a pair of rods with a serrated surface connected by a cross-link with ≥4 laminar hooks / pedicle screws

7.Plates(a)Roy-Camille plates= simple straight plates with round holes (b)Luque plates= long oval holes with clips encircling the plate

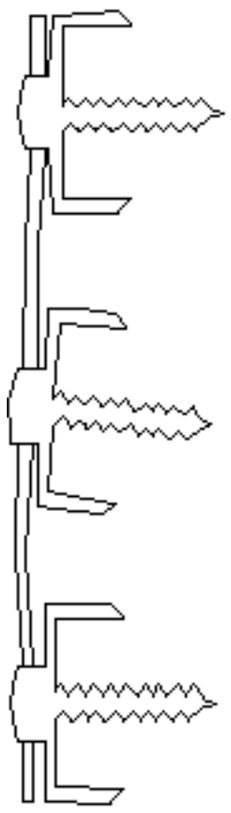




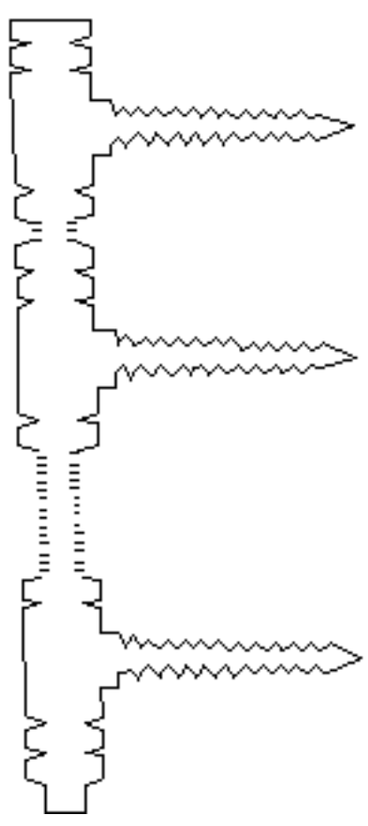
(c)Steffee plates = straight plates with long slots
8.Translaminar screw= cancellous screws for single level fusion 9.Percutaneous pinning= (hollow) interference screws placed across disk level

Notes:

Anterior Fixation Devices 1.Dwyer device= screws threaded into vertebral body over staples embedded into vertebral body connected by braided titanium wire; placed on convex side of spine



2.Zielke device= modified Dwyer system replacing cable with solid rod 3.Kaneda device= 2 curved vertebral plates with staples attached to vertebral bodies with screws, plates connected by 2 threaded rods attached to screw heads 4.Dunn device(similar to Kaneda device, discontinued)



Notes:

FORAMINA OF BASE OF SKULL
on inner aspect of middle cranial fossa 3 foramina are oriented along an oblique line in the greater sphenoidal wing from anteromedial behind the superior orbital fissure to posterolateral *mnemonic:*"rotos"foramen **rot**undum foramen **o**vale foramen **s**pinosum

Foramen Rotundum Foramen Ovale Foramen Spinosum Foramen Lacerum Foramen Magnum Pterygoid Canal Hypoglossal Canal Jugular Foramen

Notes:

Foramen Rotundum =canal within greater sphenoid wing connecting middle cranial fossa + pterygopalatine fossaLocation:inferior and lateral to superior orbital fissureCourse:extends obliquely forward + slightly inferiorly in a sagittal direction parallel to superior orbital fissure*Contents:* (a)nerves:V_2 (maxillary nerve)(b)vessels:(1)artery of foramen rotundum(2)emissary vv.√ best visualized by coronal CT

Notes:

Foramen Ovale =canal connecting middle cranial fossa + infratemporal fossaLocation:medial aspect of sphenoid body, situated posterolateral to foramen rotundum (endocranial aspect) + at base of lateral pterygoid plate (exocranial aspect)*Contents*: (a)nerves:(1)V_3 (mandibular nerve)(2)lesser petrosal nerve (occasionally)(b)vessels:(1)accessory meningeal artery(2)emissary vv.

Notes:

Foramen Spinosum Location:on greater sphenoid wing posterolateral to foramen ovale (endocranial aspect) + lateral to eustachian tube (exocranial aspect)*Contents:* (a)nerves:(1)recurrent meningeal branch of mandibular nerve(2)lesser superficial petrosal nerve(b)vessels:(1)middle meningeal a.(2)middle meningeal v.

Notes:

Foramen Lacerum Fibrocartilage cover (occasionally), carotid artery rests on endocranial aspect of fibrocartilage Location:at base of medial pterygoid plate*Contents:*(inconstant)(a)nerve:nerve of pterygoid canal (actually pierces cartilage)(b)vessel:meningeal branch of ascending pharyngeal a.

Notes:

Foramen Magnum *Contents:* (a)nerves:(1)medulla oblongata(2)cranial nerve XI (spinal accessory n.)(b)vessels:(1)vertebral a.(2)anterior spinal a.(3)posterior spinal a.

Notes:

Pterygoid Canal =VIDIAN CANAL=within sphenoid body connecting pterygopalatine fossa anteriorly to foramen lacerum posteriorly Location:at base of pterygoid plate below foramen rotundum*Contents:* (a)nerves:Vidian nerve = nerve of pterygoid canal= continuation of greater superficial petrosal nerve (from cranial nerve VII) after its union with deep petrosal nerve (b)vessel:Vidian artery = artery of pterygoid canal= branch of terminal portion of internal maxillary a. arises in pterygopalatine fossa + passes through foramen lacerum posterior to Vidian n.

Notes:

Hypoglossal Canal =ANTERIOR CONDYLAR CANALLocation:in posterior cranial fossa anteriorly above condyle starting above anterolateral part of foramen magnum, continuing in an anterolateral direction + exiting medial to jugular foramen*Contents:* (a)nerves:cranial nerve XII (hypoglossal nerve)(b)vessels:(1)pharyngeal artery(2)branches of meningeal artery

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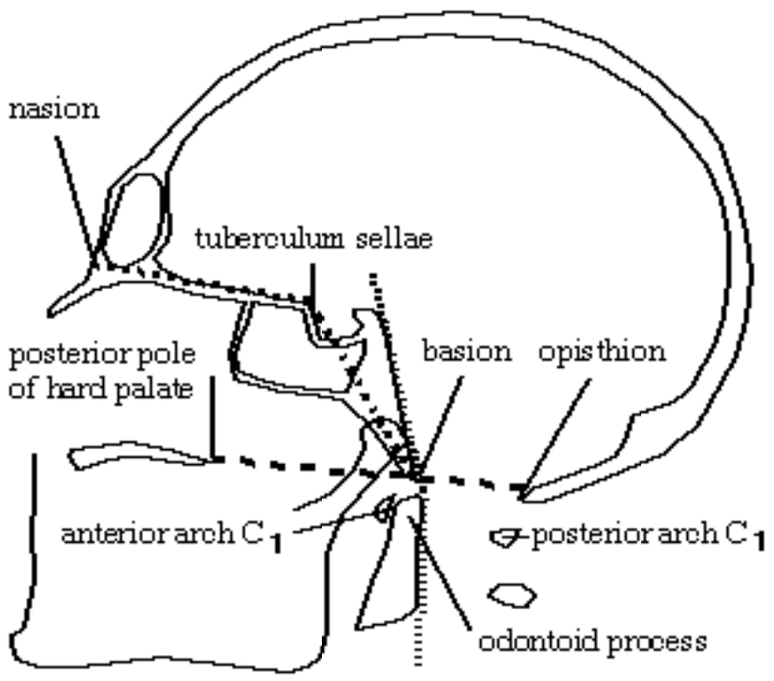
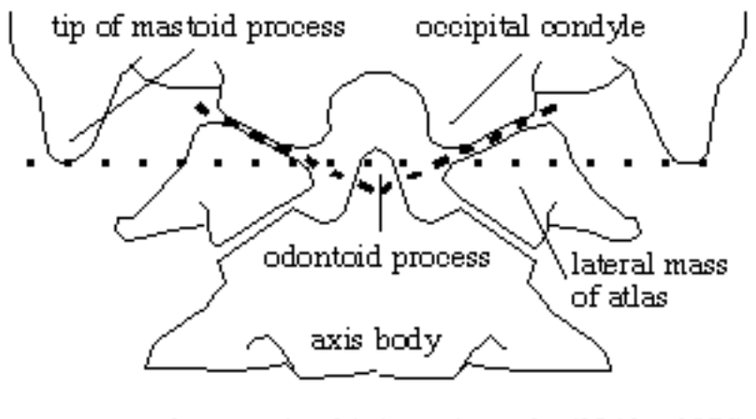
Notes:

Jugular Foramen Location:at the posterior end of petro-occipital suture directly posterior to carotid orifice(a)anterior part:(1)inferior petrosal sinus(2)meningeal branches of pharyngeal artery + occipital artery(b)intermediate part:(1)cranial nerve IX (glossopharyngeal nerve)(2)cranial nerve X (vagus nerve)(3)cranial nerve XI (spinal accessory nerve)(c)posterior part:internal jugular vein

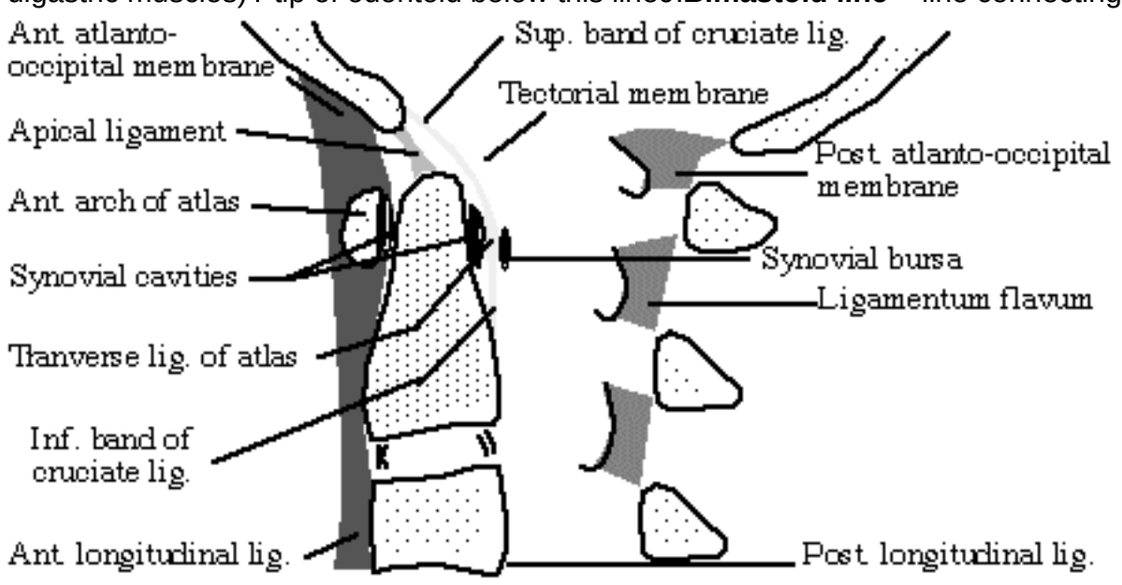
Notes:

CRANIOVERTEBRAL JUNCTION
Craniometry:

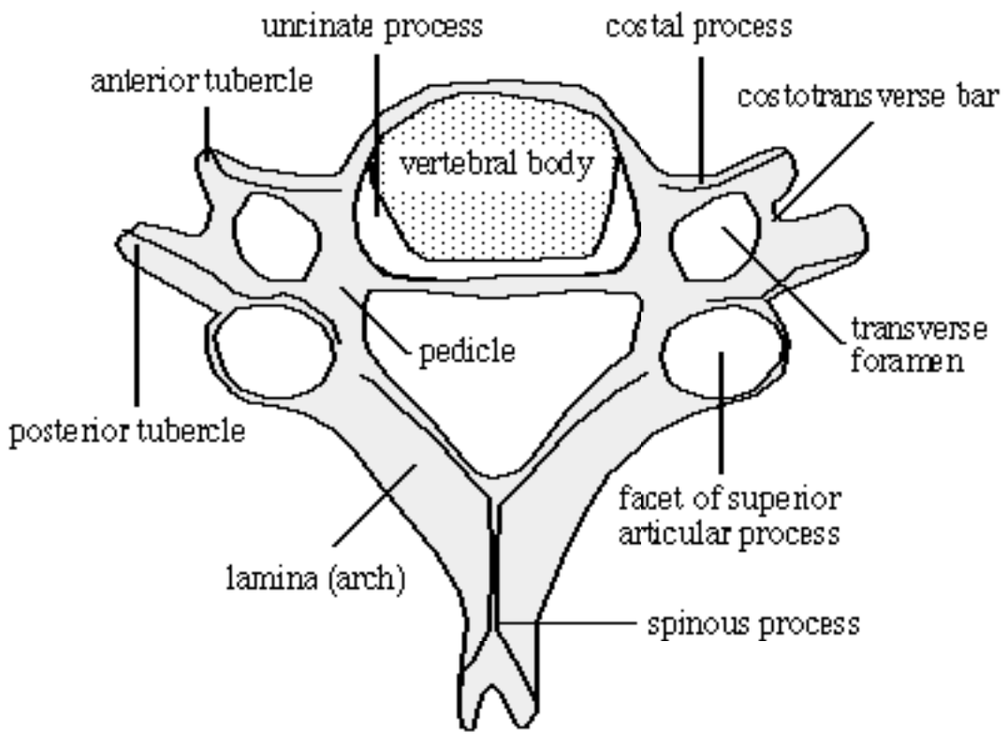




-LATERAL VIEW1.**Chamberlain line** = line between posterior pole of hard palate + opisthion (= posterior margin of foramen magnum)√ tip of odontoid process usually lies below / tangent to Chamberlain line√ tip of odontoid process may lie up to 1 ± 6.6 mm above the Chamberlain line2.**McGregor line** = line between posterior pole of hard palate + most caudal portion of occipital squamosal surface◊substitute to Chamberlain line if opisthion not visible√ tip of odontoid <5 mm above this line3.**Wackenheim clivus baseline**
=BASILAR LINE = line along clivus√ usually falls tangent to posterior aspect of tip of odontoid process4.**Craniovertebral angle** = clivus-canal angle=angle formed by line along posterior surface of axis body and odontoid process + basilar line√ ranges from 150° in flexion to 180° in extension√ ventral spinal cord compression may occur at <150°5.**Welcher basal angle**
=formed by nasion-tuberculum line and tuberculum-basion line√ angle averages 132° (should be <140°)6.**McRae line** = line between anterior lip (= basion) to posterior lip (= opisthion) of foramen magnum√ tip of odontoid below this line -ANTEROPOSTERIOR VIEW 7.**Atlanto-occipital joint axis angle**
=formed by lines drawn parallel to both atlanto-occipital joints√ lines intersect at center of odontoid process√ average angle of 125° (range of 124° to 127°)8.**Digastric line** = line between incisurae mastoideae (origin of digastric muscles)√ tip of odontoid below this line9.**Bimastoid line** = line connecting the tips of both mastoid processes√ tip of odontoid <10 mm above this line



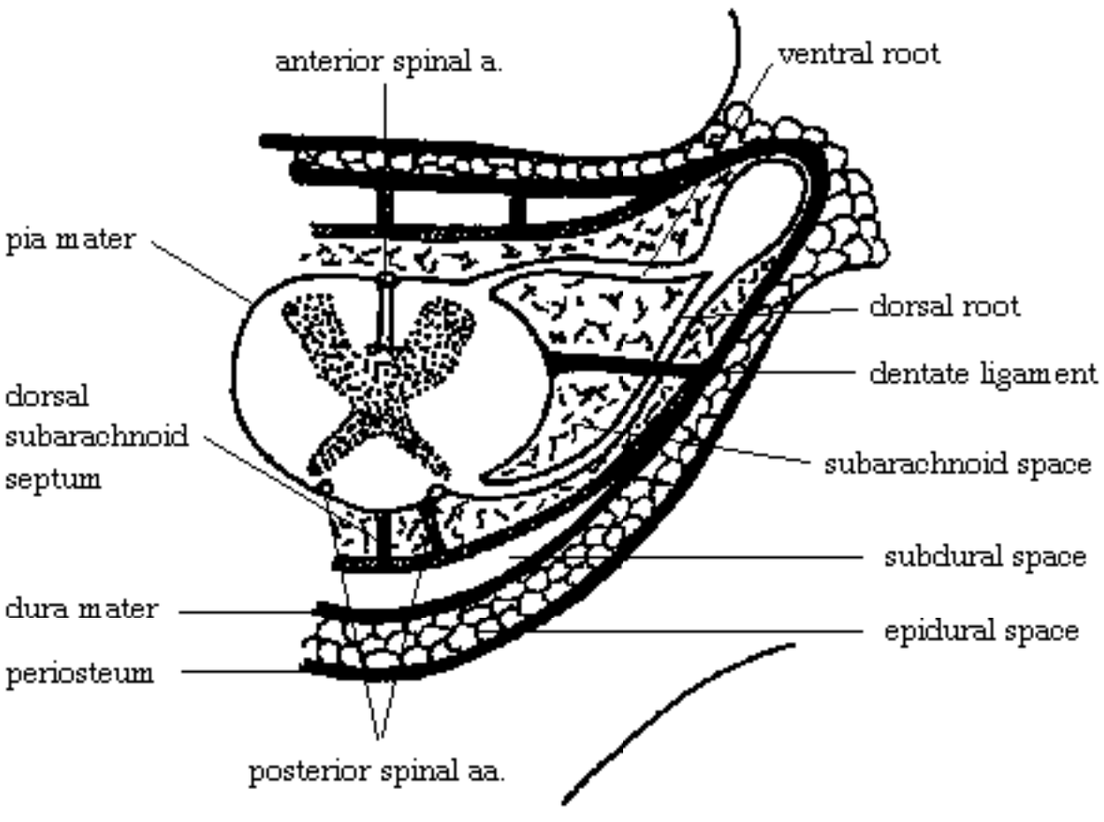
Joints and Ligaments of Occipito-atlanto-axial Region



Typical Cervical Vertebra
(cranial aspect)

Notes:

MENINGES OF SPINAL CORD



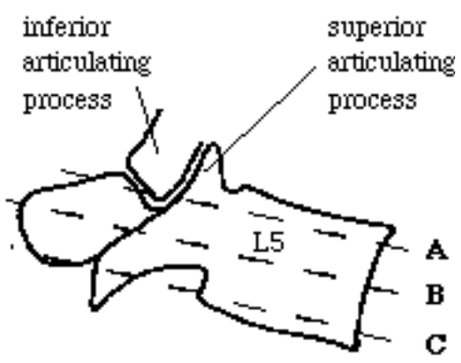
A.PERIOSTEUM= continuation of outer layer of cerebral dura mater B.EPIDURAL SPACEconsists of loose areolar tissue + rich plexus of veins (a)cervical + thoracic spine: spacious posteriorly, potential space anteriorly(b)lower lumbar + sacral spine: may occupy more than half of cross-sectional area C.DURA=continuation of meningeal / inner layer of cerebral dura mater; ends at 2nd sacral vertebra + forms coccygeal ligament around filum terminale; sends tubular extensions around spinal nerves; is continuous with epineurium of peripheral nerves*Attachment:*at circumference of foramen magnum, bodies of 2nd + 3rd cervical vertebrae, posterior longitudinal ligament (by connective tissue strands) D.SUBARACHNOID SPACE= space between arachnoid and pia mater containing CSF, reaching as far lateral as spinal ganglia dentate ligament partially divides CSF space into an anterior + posterior compartment extending from foramen magnum to 1st lumbar vertebra, is continuous with pia mater of cord medially + dura mater laterally (between exiting nerves) dorsal subarachnoid septum connects the arachnoid to the pia mater (cribriform septum) E.PIA MATER= firm vascular membrane intimately adherent to spinal cord, blends with dura mater in intervertebral foramina around spinal ganglia, forms filum terminale, fuses with periosteum of 1st coccygeal segment

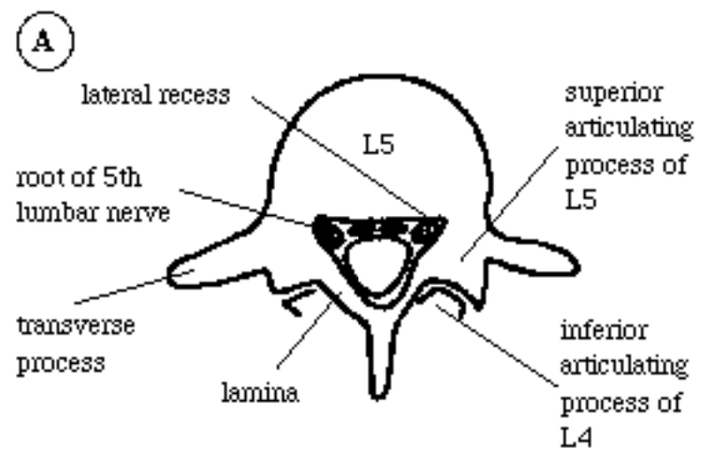
Notes:

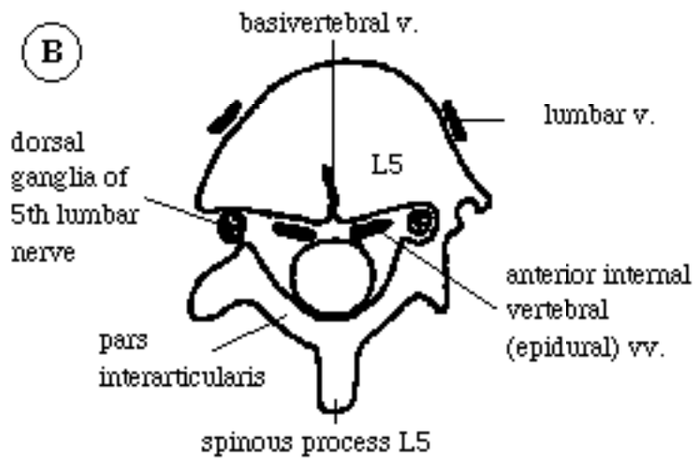
THORACIC SPINE
-12 load-bearing vertebrae-posterior arch (= pedicles, laminae, facets, transverse processes) handles tensional forces-vertebral bodies:(a)height of vertebrae anteriorly 2-3 mm less than posteriorly = mild kyphotic curvature(b)AP diameter: gradual increase from T1 to T12(c)transverse diameter: gradual increase from T3 to T12

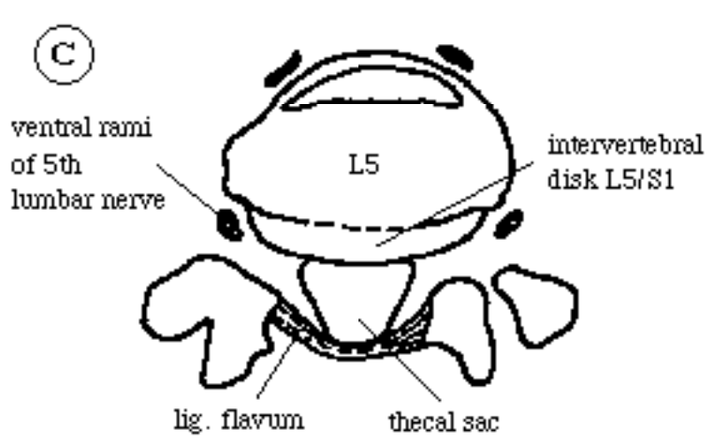
Notes:

THORACOLUMBAR SPINE (T11-L2)
-anterior column = anterior longitudinal ligament, anterior annulus fibrosus, anterior vertebral body-middle column = posterior longitudinal ligament, posterior annulus fibrosus, posterior vertebral body margin◊Integrity of the middle column is synonymous with stability!-posterior column = posterior elements + ligaments









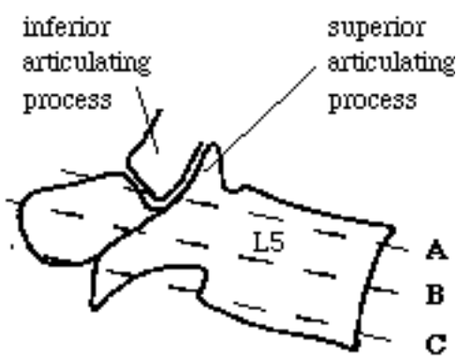
Notes:

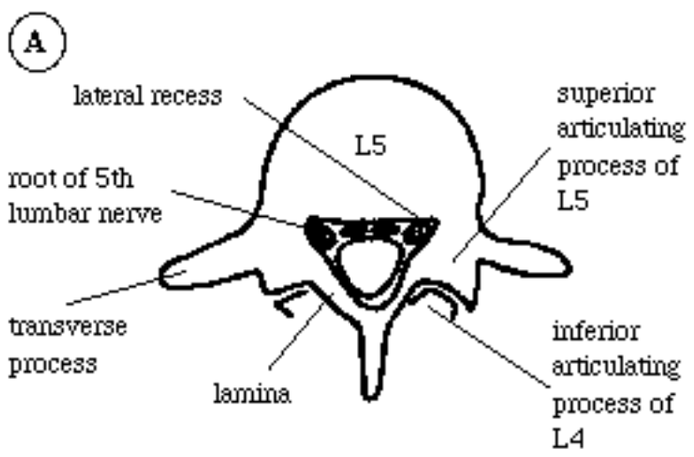
NORMAL POSITION OF CONUS MEDULLARIS

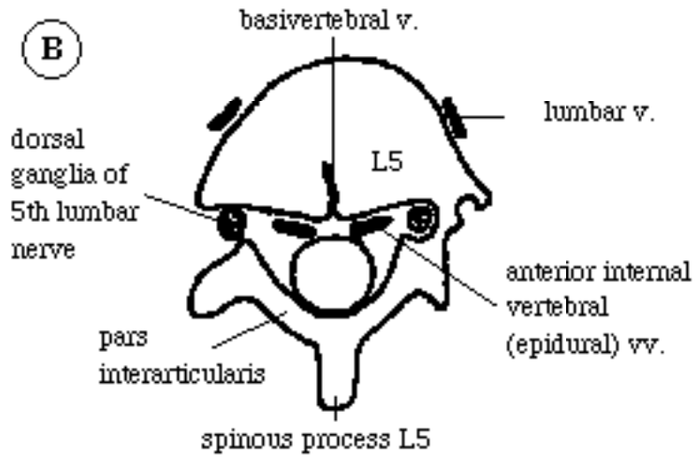
Vertebral bodies grow more quickly than spinal cord during fetal period of <19 weeks MA!No significant difference regardless of age! Inferior-most aspect of conus: L1-L2 level:normal (range T12 to L3)L2-L3 or higher:in 97.8%L3 level:indeterminate (in 1.8%)L3-L4 / lower:abnormalby 3 month:above inferior endplate of L2 (in 98%) N.B.:If conus is at / below L3 level, a search should be made for tethering mass, bony spur, thick filum!

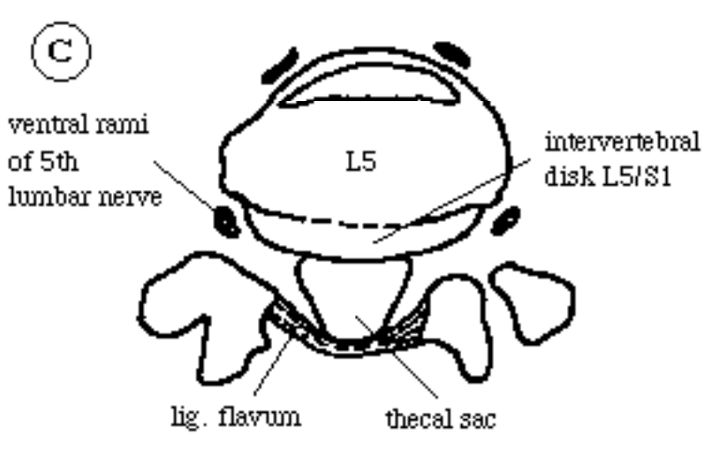
Notes:

CROSS-SECTIONS THROUGH 5TH LUMBAR VERTEBRA



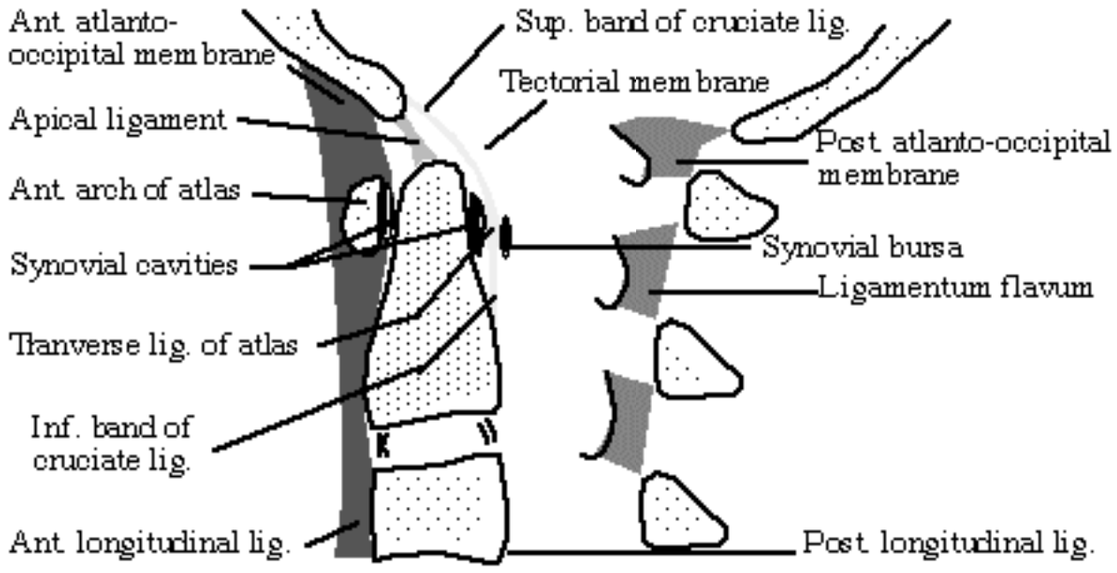






Notes:

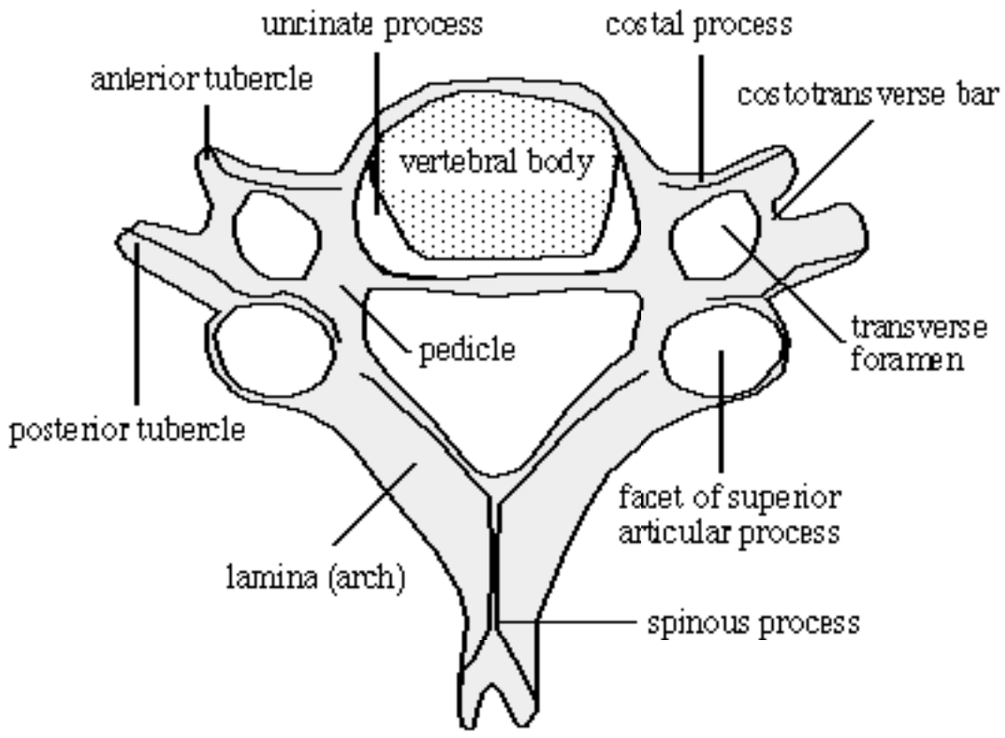
JOINTS AND LIGAMENTS OF OCCIPITAL-ATLANTO-AXIAL REGION



Joints and Ligaments of Occipito-atlanto-axial Region

Notes:

TYPICAL CERVICAL VERTEBRA



Typical Cervical Vertebra
(cranial aspect)

Notes:

BIRTH TRAUMA
1.**Caput succedaneum**
=localized edema in presenting portion of scalp, frequently associated with microscopic hemorrhage + subcutaneous hyperemia*Cause:*common after vaginal delivery ● soft superficial pitting edema√ crosses suture lines2.**Subgaleal hemorrhage**
=hemorrhage subjacent to aponeurosis covering scalp beneath the occipito-frontalis muscle ● may become symptomatic secondary to blood loss ● firm fluctuant mass increasing in size after birth ● may dissect into subcutaneous tissue of neck ● usually resolves over 2-3 weeks3.**Cephalohematoma**
=hematoma beneath outer layer of periosteum*Cause:*incorrect application of obstetric forceps / skull fracture during birth*Incidence:*1-2% of all deliveriesLocation:most commonly parietal ● firm tense mass ● usually increase in size after birth ● resolution in few weeks to months√ crescent-shaped lesion adjacent to outer table of skull √ will not cross cranial suture line√ may calcify / ossify causing thickening of diploe4.Skull fracture*Incidence:*1% of all deliveries√ CT shows associated intracranial hemorrhage5.Subdural hemorrhage(a) convexity hematoma (b) interhemispheric hematoma (c) posterior fossa hematoma 6.**Benign subdural effusion**
=benign condition that resolves spontaneously ● clear / xanthochromic fluid with elevated protein level√ extracerebral fluid collection accompanied by ventricular dilatation (= communicating hydrocephalus caused by impaired CSF absorption of these subdural fluid collections)

Notes:

INCREASED INTRACRANIAL PRESSURE

1.Intracranial mass2.Hydrocephalus3.Malignant hypertension4.Diffuse cerebral edema5.Increased venous pressure6.Elevated CSF protein7.Pseudotumor cerebri ● papilledema √ enlargement of perioptic nerve subarachnoid space

Notes:

PROLACTIN ELEVATION

Normal level: up to 25 ng/mL *Cause:* 1.Interference with hypothalamic-pituitary axis:(a)hypothalamic tumor(b)parasellar tumor(c)pituitary adenoma(d)sarcoidosis(e)histiocytosis(f)traumatic infundibular transection2.Pharmacologic agentsalpha-methyldopa, reserpine, phenothiazine, butyrophenone, tricyclic antidepressants, oral contraceptives 3.Hypothyroidism (TRH also stimulates prolactin)4.Renal failure5.Cirrhosis6.Stress / recent surgery7.Breast examination8.Pregnancy9.Lactation

Notes:

STROKE

=generic term designating a heterogeneous group of cerebrovascular disorders*Incidence:* 3rd leading cause of death in United States (after heart disease + cancer); 2nd leading cause of death due to cardiovascular disease in U.S.; 2nd leading cause of death in patients >75 years of age; 450,000 new cases per year; 160 new strokes per 100,000 population per year; leading cause of death in Orient *Age:*>55 years; M:F = 2:1*Risk factors:*heredity, hypertension (50%), smoking, diabetes (15%), obesity, familial hypercholesterolemia, myocardial infarction, atrial fibrillation, congestive heart failure, alcoholic excess, oral contraceptives, high anxiety + stress*Etiology:* A.NONVASCULAR (5%): eg, tumor, hypoxiaB.VASCULAR (95%)1.Brain infarction = ischemic stroke (80%)(a)Occlusive atheromatous disease of extracranial (35%) / intracranial (10%) arteries = large vessel disease between aorta + penetrating arterioles-critical stenosis, thrombosis,-plaque hemorrhage / ulceration / embolism(b)Small vessel disease of penetrating arteries (25%) = lacunar infarct(c)Cardiogenic emboli (6-15-23%)Ischemic heart disease with mural thrombus -acute myocardial infarction (3% risk/year)-cardiac arrhythmiaValvular heart disease -postinflammatory (rheumatic) valvulitis-infective endocarditis (20% risk/year)-nonbacterial thrombotic endocarditis (30% risk/year)-mitral valve prolapse (low risk)-mitral stenosis (20% risk/year)-prosthetic valves (1-4% risk/year)Nonvalvular atrial fibrillation (6% risk/year) Left atrial myxoma (27-55% risk/year) (d)Nonatheromatous disease (5%)-elongation, coil, kinks (up to 20%)-fibromuscular dysplasia (typically spares origin + proximal segment of ICA)-aneurysm (rare) may occur in cervical / petrous portion / intracranially-dissection: traumatic / spontaneous (2%)-cerebral arteritis (Takayasu, collagen disease, lymphoid granulomatosis, temporal arteritis, Behçet disease, chronic meningitis, syphilis)-postendarterectomy thrombosis / embolism / restenosis(e)Overactive coagulation (5%)2.Hemorrhagic stroke (20%)(a)Primary intracerebral hemorrhage (15%)-Hypertensive hemorrhage (40-60%)-Amyloid angiopathy (15-25%)-Vascular malformation (10-15%)-Drugs: eg, anticoagulants (1-2%)-Bleeding diathesis (<1%): eg, hemophilia(b)Vasospasm due to nontraumatic SAH (4%)-Ruptured aneurysm (75-80%)-Vascular malformation (10-15%)-"Nonaneurysmal" SAH (5-15%)(c)Veno-occlusive disease (1%): sinus thrombosis *May be preceded by TIA* ◊10-14% of all strokes are preceded by TIA!◊60% of all strokes ascribed to carotid disease are preceded by TIA!*Prognosis:* (1)death during hospitalization (25%): alteration in consciousness, gaze preference, dense hemiplegia have a 40% mortality rate(2)survival with varying degrees of neurologic deficit (75%)(3)good functional recovery (40%)◊Hypodensity involving >50% of MCA territory has a fatal outcome in 85%!◊Clinical diagnosis inaccurate in 13%! *Role of imaging:* 1.Confirm clinical diagnosis2.Identify primary intracerebral hemorrhage3.Detect structural lesions mimicking stroke: tumor, vascular malformation, subdural hematoma4.Detect early complications of stroke:cerebral herniation, hemorrhagic transformation*Indications for cerebrovascular testing:* 1.TIA = transient ischemic attack2.Progression of carotid disease to 95-98% stenosis3.Cardiogenic cerebral emboli*Temporal classification:* 1.**TIA** = transient ischemic attack2.**RIND** = reversible ischemic neurologic deficit=fully reversible prolonged ischemic event resulting in minor neurologic dysfunction for >24 hours*Incidence:*16 per 100,000 population per year3.**Progressing stroke** = stepwise / gradually progressing accumulative neurologic deficit evolving over hours / days4.**Slow stroke** = rare clinical syndrome presenting as developing neuronal fatigue with weakness in lower / proximal upper extremity after exercise; occurs in patients with occluded internal carotid artery5.**Completed stroke** = severe + persistent stable neurologic deficit = cerebral infarction (death of neuronal tissue) as end stage of prolonged ischemia ● level of consciousness correlates well with size of infarction *Prognosis:*6-11% recurrent stroke rate

Notes:

TRANSIENT ISCHEMIC ATTACK
=brief episode of transient focal neurological deficit owing to ischemia of <24 hours duration with return to pre-attack status*Incidence:*31 per 100,000 population per year; increasing with age up to 300; 105,000 new cases per year in United States; M > F*Cause:*(1)embolic: usually from ulcerative plaque at carotid bifurcation(2)hemodynamic: fall in perfusion pressure distal to a high-grade stenosis / occlusion*Risk factors:* (1)Hypertension (linear increase in probability of stroke with increase in diastolic blood pressure)(2)Cardiac disorders (prior myocardial infarction, angina pectoris, valvular heart disease, dysrhythmia, congestive heart failure)(3)Diabetes mellitus(4)Cigarette smoking (weak)*Prognosis:*5.3% stroke rate per year for 5 years after first TIA; per year 12% increase of stroke / myocardial infarction / death; complete stroke in 33% within 5 years; complete stroke in 5% in 1 month A.CAROTID TIA (2/3) ● carotid attacks <6 hours in 90% ● transient weakness / sensory dysfunction CLASSICALLY in(a)hand / face with embolic event(b)proximal arm + lower extremity with hemodynamic event (watershed area)-motor dysfunction = weakness, paralysis, clumsiness of one / both limbs on same side-sensory alteration = numbness, loss of sensation, paresthesia of one / both limbs on same side-speech / language disturbance = difficulty in speaking (dys- / aphasia) / writing, in comprehension of language / reading / performing calculations-visual disturbance = loss of vision in one eye, homonymous hemianopia, amaurosis fugax ● paresis (mono-, hemiparesis) in 61% ● paresthesia (mono-, hemiparesthesia) in 57% ● amaurosis fugax (= transient premonitory attack of impaired vision due to retinal ischemia) in 12% caused by transient hypotension or emboli of platelets / cholesterol crystals which may be revealed by funduscopy ● facial paresthesia in 30% B.VERTEBROBASILAR TIA (1/3) ● vertebrobasilar events <2 hours in 90%-motor dysfunction = as with carotid TIA but sometimes changing from side to side including quadriplegia, diplopia, dysarthria, dysphagia-sensory alteration = as with carotid TIA usually involving one / both sides of face / mouth / tongue-visual loss = as with carotid TIA including uni- / bilateral homonymous hemianopia-disequilibrium of gait / postural disturbance, ataxia, imbalance / unsteadiness-drop attack = sudden fall to the ground without loss of consciousness ● binocular visual disturbance in 57% ● vertigo in 50% ● paresthesia in 40% ● diplopia in 38% ● ataxia in 33% ● paresis in 33% ● headaches in 25% ● seizures in 1.5%
Accelerating / crescendo TIA =repeated periodic events of neurologic dysfunction with complete recovery to normal in interphase *Rx:*1.Carotid endarterectomy (1% mortality, 5% stroke)2.Anticoagulation3.Antiplatelet agent: aspirin, ticlopidine-in patients with recently symptomatic TIA / minor stroke + >70% carotid artery stenosis: prophylactic carotid endarterectomy + chronic low-dose aspirin therapy

Notes:

Radiology Review Manual
fourth edition

INFECTION IN IMMUNOCOMPROMISED PATIENTS
*Cause:*underlying malignancy, collagen disease, cancer therapy, AIDS, immunosuppressive therapy in organ transplants*Organism:*Toxoplasma, Nocardia, Aspergillus, Candida, Cryptococcus√ poorly defined hypodense zones with rapid enlargement in size + number, particularly affecting basal ganglia + centrum semiovale (poorly localized + encapsulated infection with poor prognosis)√ ring / nodular enhancement (sufficient immune defenses): Toxoplasma, Nocardia√ enhancement may be blunted by steroid Rx*AIDS may be associated with:* thrombocytopenia, lymphoma, plasmacytoma, Kaposi sarcoma, progressive multifocal leukoencephalopathy

Notes:

TRIGEMINAL NEUROPATHY

● facial pain, numbness, weakness of masticatory muscles, trismus ● diminished / absent corneal reflex ● abnormal jaw reflex ● decreased pain / touch / temperature sensation ● atrophy of masticatory muscles ● **tic douloureux** = paroxysmal facial pain (usually confined to V_2 and V_3) mainly caused by neurovascular compression (tortuous elongated superior cerebellar artery / anterior inferior cerebellar artery / vertebrobasilar dolichoectasia / venous compression)A.BRAIN STEM LESION1.Vascular: infarct, AVM2.Neoplastic: glioma, metastasis3.Inflammatory: multiple sclerosis (1-8%), herpes rhombencephalitis4.Other: syringobulbiaB.CISTERNAL CAUSES1.Vascular: aneurysm, AVM, vascular compression2.Neoplastic: acoustic schwannoma, meningioma, trigeminal schwannoma, epidermoid cyst, lipoma, metastasis3.Inflammatory: neuritisC.MECKEL CAVE + CAVERNOUS SINUS1.Vascular: carotid aneurysm2.Neoplastic: meningioma, trigeminal schwannoma, epidermoid cyst, lipoma, pituitary adenoma, base of skull neoplasm, metastasis, perineural tumor spread3.Inflammatory: Tolosa-Hunt syndromeD.EXTRACRANIAL1.Neoplastic: neurogenous tumor, squamous cell carcinoma, adenocarcinoma, lymphoma, adenoid cystic carcinoma, mucoepidermoid carcinoma, melanoma, metastasis, perineural tumor spread2.Inflammatory: sinusitis3.Other: masticator space abscess, trauma

Notes:

DEMENTIA

1.Alzheimer disease2.Pick disease3.Normal pressure hydrocephalus4.Subdural hematoma5.Brain mass

Notes:

CLASSIFICATION OF CNS ANOMALIES

A.DORSAL INDUCTION ANOMALY=defects of neural tube closure1.Chiari malformation:at 4 weeks2.Encephalocele:at 4 weeks3.Anencephaly4.Spinal dysraphism5.HydromyeliaB.VENTRAL INDUCTION ANOMALY1.Holoprosencephaly:5 - 6 weeks2.Septo-optic dysplasia:6 - 7 weeks3.Dandy-Walker malformation:7 - 10 weeks4.Agenesis of septum pellucidumC.NEURONAL PROLIFERATION & HISTOGENESIS1.Neurofibromatosis:5 weeks - 6 months2.Tuberous sclerosis:5 weeks - 6 months3.Primary hydranencephaly:>3 months4.Neoplasia5.Vascular malformation (vein of Galen, AVM, hemangioma)D.NEURONAL MIGRATION ANOMALYdue to infection, ischemia, metabolic disorders 1.Schizencephaly:2 months2.Agyria + pachygyria:3 months3.Gray matter heterotopia:5 months4.Dysgenesis of corpus callosum:2 - 5 months5.Lissencephaly6.Polymicrogyria7.Unilateral megalencephalyE.DESTRUCTIVE LESIONS1.Hydranencephaly2.Porencephaly3.Hypoxia4.Toxicosis5.Inflammatory disease (TORCH)(a)**To**xoplasmosis(b)**R**ubella√ punctate / nodular calcifications√ porencephalic cysts√ occasionally microcephaly(c)**C**ytomegalic inclusion disease√ typically punctate / stippled / curvilinear periventricular calcifications√ often hydrocephalus(d)**H**erpes simplex

Absence Of Septum Pellucidum Phakomatoses

Notes:

Absence Of Septum Pellucidum 1.Holoprosencephaly2.Callosal agenesis3.Septo-optic dysplasia4.Schizencephaly5.Severe chronic hydrocephalus6.Destructive porencephaly

Notes:

Phakomatoses =NEUROCUTANEOUS SYNDROMES=NEUROECTODERMAL DYSPLASIAS=development of benign tumors / malformations especially in organs of ectodermal origin1.Neurofibromatosis2.Tuberous sclerosis3.von Hippel-Lindau disease4.Sturge-Weber-Dimitri syndrome5.Ataxia-telangiectasia

Notes:

DEGENERATIVE DISEASES OF CEREBRAL HEMISPHERES
=progressive fatal disease characterized by destruction / alteration of gray and white matter *Etiology:*genetic; viral infection; nutritional disorders (eg, anorexia nervosa, Cushing syndrome); immune system disorders (eg, AIDS); exposure to toxins (eg, CO); exposure to drugs (eg, alcohol, methotrexate + radiation) Leukodystrophy = degenerative diffuse sclerosis with symmetrical bilateral white matter lesions Leukoencephalopathy = disease of white matter A.DEMYELINATING DISEASE=normal myelin destroyed by disease process1.Multiple sclerosis (most frequent primary demyelinating disease)2.Alzheimer disease (most common of diffuse gray matter degenerative diseases)3.Parkinson disease (most common subcortical degenerative disease)4.Creutzfeldt-Jakob disease5.Menkes disease (sex-linked recessive disorder of copper metabolism)6.Progressive multifocal leukoencephalopathy7.Disseminated necrotizing leukoencephalopathy8.Globoid cell leukodystrophy9.Spongiform degeneration10.Cockayne syndrome11.Spongiform leukoencephalopathy12.Myelinoclastic diffuse sclerosis (Schilder disease) B.DYSMYELINATING DISEASE=metabolic disorder (= enzyme deficiency) resulting in deficient / absent myelin sheaths(a)macrencephalic:1.Alexander disease (frontal areas affected first)2.Canavan disease (white matter diffusely affected)(b)hyperdense thalami, caudate nuclei, corona radiata1.Krabbe disease(c)family history (X-linked recessive)1.X-linked adrenoleukodystrophy2.Pelizaeus-Merzbacher disease(d)others1.Metachromatic leukodystrophy (most common hereditary leukodystrophy)2.Binswanger disease (SAE)3.Multi-infarct dementia (MID)4.Pick disease5.Huntington disease6.Wilson disease7.Reye syndrome8.Mineralizing microangiopathy9.Diffuse sclerosis

Notes:

Cerebral Atrophy =irreversible loss of brain substance + subsequent enlargement of intra- and extracerebral CSF-containing spaces (hydrocephalus ex vacuo = ventriculomegaly)A.DIFFUSE BRAIN ATROPHY*Cause:* (a)Trauma, radiation therapy(b)Drugs (dilantin, steroids, methotrexate, marijuana, hard drugs, chemotherapy), alcoholism, hypoxia(c)Demyelinating disease (multiple sclerosis, encephalitis)(d)Degenerative diseaseeg, Alzheimer disease, Pick disease, Jakob-Creutzfeldt disease(e)Cerebrovascular disease + multiple infarcts(f)Advancing age, anorexia, renal failure√ enlarged ventricles + sulciB.FOCAL BRAIN ATROPHY*Cause:*vascular / chemical / metabolic / traumatic / idiopathic (Dyke-Davidoff-Mason syndrome)C.REVERSIBLE PROCESS SIMULATING ATROPHY (in younger people)*Cause:*anorexia nervosa, alcoholism, catabolic steroid treatment, pediatric malignancy√ prominent sulci√ ipsilateral dilatation of basal cisterns + ventricles√ ex vacuo dilatation of ventricles√ thinning of gyri

Notes:

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Cerebellar Atrophy A.WITH CEREBRAL ATROPHY= generalized senile brain atrophy B.WITHOUT CEREBRAL ATROPHY1.Olivopontocerebellar degeneration / Marie ataxia / Friedreich ataxia ● onset of ataxia in young adulthood2.Ataxia-telangiectasia3.Ethanol-toxicity: predominantly affecting midline (vermis)4.Phenytoin-toxicity: predominantly affecting cerebellar hemispheres5.Idiopathic degeneration secondary to carcinoma(= paraneoplastic), usually oat cell carcinoma of lung 6.Radiotherapy7.Focal cerebellar atrophy:(a) infarction (b) traumatic injury

Notes:

Extra-axial Tumor *mnemonic:*"MABEL"**M**eningioma **A**rachnoid cyst **B**ony lesion **E**pidermoid **L**eukemic / lymphomatous infiltration

Intra- versus extra-axial mass

	intra-axial	extra-axial
Relationship to dura / bone	no attachment until advanced	contiguous
Local bony changes	uncommon	common
Displacement of cortex	toward dura / bone	away from bone
Subarachnoid cistern	effaced	widened
Feeding arteries	pial feeding arteries	dural feeding arteries

Notes:

Leptomeningeal Disease A.INFLAMMATION1.Langerhans cell histiocytosis2.Sarcoidosis3.Wegener granulomatosis4.Chemical meningitis: rupture of epidermoidB.INFECTION1.Bacterial meningitis2.Tuberculous meningitis3.Fungal meningitis4.NeurosyphilisC.TUMOR(a)Primary meningeal tumor:1.Meningioma2.Glioma: primary leptomeningeal glioblastomatosis / gliosarcomatosis3.Melanoma / melanocytoma4.Sarcoma5.Lymphoma(b)CSF-spread from primary CNS tumor1.Medulloblastoma2.Germinoma3.Pineoblastoma(c)Metastasis1.Breast carcinoma2.Lymphoma / leukemia3.Lung carcinoma4.Malignant melanoma5.Gastrointestinal carcinoma6.Genitourinary carcinomaD.TRAUMA1.Old subarachnoid hemorrhage2.Surgical scarring from craniotomy3.Lumbar puncture

Notes:

Pericerebral Fluid Collection In Childhood A.ENLARGED SUBARACHNOID SPACE(a)due to macrocephaly(b)due to brain atrophy√ superficial cortical veins cross subarachnoid space to reach superior sagittal sinus√ wide sulci, normal configuration of gyri√ normal / prominent size of ventriclesB.SUBDURAL FLUID COLLECTION(1)Subdural hygroma(2)Subdural empyema / abscess (due to meningitis)(3)Subdural hematoma√ superficial cortical veins are prevented to cross subarachnoid by presence of arachnoid / neomembrane√ wide interhemispheric fissure

Notes:

Ventriculomegaly A.MACROCEPHALY ● increased intraventricular pressure(a)Obstruction to CSF flow1.Communicating hydrocephalus2.Noncommunicating hydrocephalus(b)Overproduction of CSF = nonobstructive hydrocephalus(c)NeoplasmB.MICROCEPHALY ● normal intraventricular pressure(a)Primary failure of brain growth-dysgenesis1.Holoprosencephaly2.Aneuploidy syndromes (trisomies)3.Migrational (<6 layers)-environment: alcohol, drugs, toxins-infection: TORCH(b)Loss of brain mantle-Infection: TORCH-Vascular accident:1.Hydranencephaly2.Schizencephaly3.Porencephaly-Hemorrhage:1.Porencephaly2.LeukomalaciaC.NORMOCEPHALY

Notes:

Colpocephaly =dilatation of trigones + occipital horns + posterior temporal horns of lateral ventricles1.Agenesis of corpus callosum2.Arnold-Chiari malformation3.Holoprosencephaly

Notes:

Intraventricular tumor *Prevalence:*10% of all intracranial neoplasms1.Ependymoma20%2.Astrocytoma18%3.Colloid cyst12%4.Meningioma11%5.Choroid plexus papilloma 7%6.Epidermoid / dermoid6%7.Craniopharyngioma 6%8.Medulloblastoma5%9.Cysticercosis5%10.Arachnoid cyst4%11.Subependymoma2%12.AVM2%13.Teratoma1%14.Metastasis15.Intraventricular neurocytoma16.Oligodendroglioma ***Tumor In 4th Ventricle*** 1.Choroid plexus papilloma2.Ependymoma / glioma3.Hemangioblastoma4.Vermian metastasis5.AVM6.Epidermoid tumor (rare)7.Inflammatory mass8.Cyst ***Tumor In 3rd Ventricle*** 1.Colloid cyst2.Glioma3.Aneurysm4.Craniopharyngioma5.Ependymoma6.Meningioma7.Choroid plexus papilloma8.Intraventricular neurocytoma

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Notes:

Periventricular Hypodensity 1.Encephalomalacia√ slightly denser than CSF2.Porencephaly=cavity communicating with ventricle / cistern from intracerebral hemorrhage*Associated with:* dilated ventricle, sulci, and fissures √ CSF density 3.Resolving hematoma ● Hx of previously demonstrated hematoma√ may show ring enhancement + compression of adjacent structures4.Cystic tumor√ mass effect + contrast enhancement

Notes:

Enhancing Ventricular Margins (a)Subependymal spread of metastatic tumor1.Bronchogenic carcinoma (especially small cell carcinoma)2.Melanoma3.Breast carcinoma(b)Subependymal seeding of CNS primary1.Glioma2.Ependymoma(c)Ependymal seeding of CNS primary1.Medulloblastoma2.Germinoma(d)Primary CNS lymphoma / systemic lymphoma(e)Inflammatory ventriculitis

Notes:

Periventricular Calcifications In A Child 1.Tuberous sclerosis2.Congenital infection: CMV, toxoplasmosis

Notes:

Periventricular T2WI-hyperintense Lesions A.YOUNG PATIENTS1.Multiple sclerosis2.Migraine: in 41% with classic migraine, in 57% with complicated migraine; presumed to represent vasculitis-induced small infarcts3.Vasculitic disorder: SLE, Behçet disease, sickle cell disease√ triad of deep white matter lesions + cortical infarcts + hemorrhage4.Acute disseminated encephalomyelitis (ADE)= postviral leukoencephalopathy 5.Virchow-Robin space=small invaginations of subarachnoid space following pia mater along perforating nutrient end vessels into brain substanceLocation:inferior third of putamen; usually bilateral√ 1-2 mm round lesions isointense to CSF (well seen on coronal sections through centrum semiovale + on low-axial sections at level of anterior commissure)6.Leukodystrophy: in children√ symmetric diffuse confluent involvement7.Ependymitis granularis=symmetrically focal areas of hyperintensity on T2WI anterior + lateral to frontal horns in normal individuals*Histo:*patchy loss of ependyma with paucity of hydrophobic myelin, which allows migration of fluid out of the ventricle into interstitiumB.ELDERLY1.**État criblé** (sievelike) / gliosis=deep white matter ischemia=extensive number of perivascular fluid spaces predominantly at arteriolar level as part of subacute arteriosclerotic encephalopathy*Cause:*chronic ischemia due to arteriosclerosis of long penetrating arteries arising from circle of Willis (lenticulostriate + thalamo-perforators) = small vessel disease*Predisposed:*cigarette smoker, hypertensive patient*Histo:*lipohyalin deposits within vessel walls followed by partial demyelination, gliosis, interstitial edema*Incidence:*in 10% without risk factors, in 84% with risk factors and symptoms*Age:*>60 years (in 30-60%)Location:periventricular white matter > optic radiation > basal ganglia > centrum semiovale > brainstem (usually spares corpus callosum + subcortical U-fibers)√ multiple focal lesions <2 mm2.Lacunar infarctionC.PATIENTS WITH AIDS1.HIV encephalitis:√ well-defined "patchy" / ill-defined "dirty white matter"√ central atrophy2.Toxoplasmosis3.Lymphoma4.Progressive multifocal leukoencephalopathy (PML)D.PATIENTS WITH TRAUMA1.Diffuse axonal / shearing injury2.Diffuse white matter injury=radiation-induced demyelination of periventricular white matter*Cause:*whole-brain irradiation ● subclinical3.Diffuse necrotizing leukoencephalopathy*Cause:*intrathecal methotrexate ± whole brain irradiation ● rapidly deteriorating clinical course√ confluent pattern with scalloped margins within periventricular white matter extending out to subcortical U-fibersE.PATIENTS WITH HYDROCEPHALUS1.Transependymal CSF flow√ smooth halo of even thickness

Notes:

Diffusely Swollen Hemispheres A.METABOLIC1.Metabolic encephalopathy: eg, uremia, Reye syndrome, ketoacidosis2.Anoxia: cardiopulmonary arrest, near-drowning, smoke inhalation, ARDSB.NEUROVASCULAR1.Hypertensive encephalopathy2.Superior sagittal sinus thrombosis3.Head trauma4.Pseudotumor cerebriC.INFLAMMATIONeg, herpes encephalitis, CMV, toxoplasmosis

Notes:

Edema Of Brain =increase in brain volume due to increased tissue-water content (80% for gray matter + 68% for white matter is normal) *Etiology:* (a)Cytotoxic edemareversible increase in intracellular water content secondary to ischemia / anoxia (axonal pallor)(b)Vasogenic edema (most common form)increase in pinocytotic activity with passage of protein across vessel wall into intercellular space (lack of contrast enhancement means breakdown of blood-brain barrier is not the cause); associated with primary brain neoplasm, metastases, hemorrhage, infarction, inflammation*Types:* 1.Hydrostatic edemarapid increase / decrease in intracranial pressure2.Interstitial edema increase in periventricular interstitial spaces secondary to transependymal flow of CSF with elevated intraventricular pressure3.Hypoosmotic edemaproduced by overhydration from IV fluid / inappropriate secretion of antidiuretic hormone4.Congestive brain swellingrapid accumulation of extravascular water as a result of head trauma; may become irreversible (brain death) if intracranial pressure equals systolic blood pressure√ decreased distinction between gray + white matter√ compressed slitlike lateral ventricles√ compression of cerebral sulci + perimesencephalic cisternsCT: √ areas of hypodensity◊Edema is always greatest in white matter!√ mass effect: flattening of gyri, displacement + deformation of ventricles, midline shift√ return to normal from nonhemorrhagic edema / brain atrophy from white matter shearing injuryMR: √ decreased intensity on T1WI, increased intensity on T2WI√ enhancement with gadoliniumUS: √ generalized / focal increase of parenchymal echogenicity with featureless appearance√ decreased resistive indices

Notes:

Brain Herniation 1.Subfalcinecontralateral shift of midline structures under falx cerebri 2.Transtentorial(a)upward: displacement of cerebellum through tentorial incisura(b)downward-anterior: uncal herniation (most common) caused by lesions in anterior half of brain-posterior: herniation of parahippocampal gyrus-total: herniation of entire hippocampus3.Retroalarherniation of frontal lobe posteriorly across edge of sphenoid ridge 4.Transforaminalherniation of inferior mesial portions of cerebellum downward through foramen magnum

Notes:

Cholesterol-containing CNS Lesions 1.Epidermoid inclusion cyst2.Cholesterol granuloma3.Acquired epidermoid of middle ear4.Congenital cholesteatoma of middle ear5.Craniopharyngioma

Notes:

Cyst With A Mural Nodule 1.Pilocytic astrocytoma (childhood)2.Ganglioglioma3.Pleomorphic xanthoastrocytoma4.Glioblastoma multiforme5.Hemangioblastoma (posterior fossa, spinal cord)

Notes:

Midline Cyst 1.**Cavum septi pellucidi** = "5th ventricle"=thin triangular membrane consisting of two glial layers covered laterally with ependyma separating the frontal horns of lateral ventricles*Incidence:*in 80% of term infants; in 15% of adultsLocation:posterior to genu of corpus callosum, inferior to body of corpus callosum, anterosuperior to anterior pillar of fornix√ extends to foramen of Monro√ may dilate + cause obstructive hydrocephalus (rare)2.**Cavum vergae** = "6th ventricle"=cavity posterior to columns of fornix; contracts after about 6th gestational month*Incidence:*in 30% of term infants; in 15% of adultsLocation:posterior to fornix, anterior to splenium of corpus callosum, inferior to body of corpus callosum, superior to transverse fornix√ posterior midline continuation of cavum septi pellucidi beyond foramen of Monro3.**Cavum veli interpositi** =extension of quadrigeminal plate cistern above 3rd ventricle to foramen of Monro, laterally bounded by columns of fornix + thalamus4.Colloid cyst: anterior + superior to cavum septi pellucidi5.Arachnoid cyst: in region of quadrigeminal plate cistern√ curvilinear margins

Notes:

Posterior Fossa Cystic Malformation 1.Dandy-Walker malformation2.Dandy-Walker variant3.Megacisterna magna4.Arachnoid pouch

Notes:

Suprasellar Low-density Lesion With Hydrocephalus A.CYST1.Arachnoid cyst2.Ependymal cyst of 3rd ventricle3.Parasitic cyst of 3rd ventricle (cysticercosis)4.Dilated 3rd ventricle (in aqueductal stenosis)B.CYSTIC MASS1.Epidermoid2.Hypothalamic pilocytic astrocytoma3.Cystic craniopharyngioma Nota bene:Cystic lesion may be inapparent within surrounding CSF; metrizamide cisternography is helpful in detection + to exclude aqueduct stenosis

Notes:

Mesencephalic Low-density Lesion 1.Normal: decussation of superior cerebellar peduncles at level of inferior colliculi2.Syringobulbiafound in conjunction with syringomyelia, Arnold-Chiari malformation, trauma√ CSF density centrally√ intrathecal contrast enters central cavity3.Brainstem infarction√ abnormal contrast enhancement after 1 week√ well-defined low-attenuation region without enhancement after 2-4 weeks4.Central pontine myelinolysiscomatose patient receiving rapid correction / overcorrection of severe hyponatremia (following prolonged IV fluid administration / alcoholism) *Pathophysiology:* rapid correction of sodium releases myelinotoxic compounds by gray matter components resulting in loss of myelin (osmotic myelinolysis) with preservation of neurons + axons ● spastic quadriparesis + pseudobulbar palsy ● progression to pseudocoma (locked-in syndrome) in 3-5 days√ diminished attenuation in central region of pons√ ± extrapontine lesions in basal ganglia, thalami*Prognosis:*10% survival rate beyond 6 months5.Brainstem glioma√ mass with indistinct margins + vague enhancement6.Metastasis√ well-defined contrast enhancement7.Granuloma in TB / sarcoidosis (rare)

Notes:

Intracranial Pneumocephalus *Cause:* A.TRAUMA (74%):(a)fracturein 3% of all skull fractures; in 8% of fractures involving paranasal sinuses (frontal > ethmoid > sphenoid > mastoid) or base of skull (b)penetrating injuryB.NEOPLASM INVADING SINUS (13%):1.Osteoma of frontal / ethmoid sinus2.Pituitary adenoma3.Mucocele, epidermoid4.Malignancy of paranasal sinusesC.INFECTION WITH GAS-FORMING ORGANISM (9%):in mastoiditis, sinusitis D.SURGERY (4%):hypophysectomy, paranasal sinus surgery *Mechanism* (dural laceration): (1)ball-valve mechanism during straining, coughing, sneezing(2)vacuum phenomenon secondary to loss of CSF*Time of onset:*on initial presentation (25%), usually seen within 4-5 days, delay up to 6 months (33%)*Mortality:*15%*Cx:*1.CSF rhinorrhea (50%)2.Meningitis / epidural / brain abscess (25%)3.Extracranial pneumocephalus = air collection in subaponeurotic space

Notes:

Intracranial Calcifications *mnemonic:*"PINEEAL"**P**hysiologic **I**nfection **N**eoplasm **E**ndocrine **E**mbryologic **A**rteriovenous **L**eftover Ls A.PHYSIOLOGIC INTRACRANIAL CALCIFICATIONSB.INFECTIONTORCH (toxoplasmosis, CMV, rubella, herpes), healed abscess, hydatid cyst, granuloma (tuberculoma, actinomycosis, coccidioidomycosis, cryptococcosis, mucormycosis), cysticercosis, trichinosis, paragonimiasis *mnemonic:* **C**M**V** calcifications are **c**ircum**v**entricular **T**oxoplasma calcifications are in**t**raparenchymal C.NEOPLASMCraniopharyngioma (40-80%), oligodendroglioma (50-70%), chordoma (25-40%), choroid plexus papilloma (10%), meningioma (20%), pituitary adenoma (3-5%), pinealoma (10-20%), dermoid (20%), lipoma of corpus callosum, ependymoma (50%), astrocytoma (15%), after radiotherapy, metastases (1-2%, lung > breast > GI tract) N.B.:astrocytomas calcify less frequently but are the most common tumor*mnemonic:*"Ca^{2+} COME"**C**raniopharyngioma **A**strocytoma, **A**neurysm, **C**horoid plexus papilloma **O**ligodendroglioma **M**eningioma, **M**edulloblastoma **E**pendymoma D.ENDOCRINEHyperparathyroidism, hypervitaminosis D, hypoparathyroidism, pseudohypoparathyroidism, CO poisoning, lead poisoning E.EMBRYOLOGICNeurocutaneous syndromes (tuberous sclerosis, Sturge-Weber, neurofibromatosis), Fahr disease, Cockayne syndrome, basal cell nevus syndrome F.ARTERIOVENOUSAtherosclerosis, aneurysm, AVM, occult vascular malformation, hemangioma, subdural + epidural hematomas, intracerebral hemorrhage G.LEFTOVER LsLipoma, lipoid proteinosis, lissencephaly

Physiologic Intracranial Calcification 1.Pineal calcification*Age:*no calcification <5 years of age, in 8-10% at 8-14 years of age, in 40% by 20 years of age, 2/3 of adult population√ amorphous / ringlike calcification <3 mm from midline usually <10 mm in diameter√ approximately 30 mm above highest posterior elevation of pyramidsCAVE:pineal calcification >14 mm suggests pineal neoplasm (teratoma / pinealoma) 2.Habenula*Incidence:*approximately in 1/3 of population*Age:*>10 years of age√ posteriorly open C-shaped calcification 4-6 mm anterior to pineal gland3.Choroid plexusmay calcify in all ventricles: most commonly in glomus within atrium of lateral ventricles, near foramen of Monro, tela choroidea of 3rd ventricle, roof of 4th ventricle, along foramina of Luschka *Age:*>3 years of age√ 20-30 mm behind + slightly below pineal on lateral projection, symmetrical on AP projection*DDx:*neurofibromatosis4.Dura, falx cerebri, falx cerebelli, tentorium*Incidence:*10% of population*Age:*>3 years of age*DDx:*basal cell nevus syndrome (Gorlin syndrome), pseudoxanthoma elasticum, congenital myotonic dystrophy5.Petroclinoid ligament (= reflection of tentorium)between tip of dorsum sellae and apex of petrous bone *Age:*>5 years of age6.Interclinoid ligament= interclinoid bridging 7.Arteriosclerosis: particularly intracavernous segment of ICA, basilar a., vertebral a.8.Basal ganglia

Notes:

Increased Density Of Falx 1.Subarachnoid hemorrhage2.Interhemispheric subdural hematoma3.Diffuse cerebral edema (= increased density relative to low-density brain)4.Dural calcifications (hypercalcemia from chronic renal failure, basal cell nevus syndrome, hyperparathyroidism)5.Normal falx (can be normal in pediatric population)

Notes:

Intraparenchymal Hemorrhage *mnemonic:*"ITHACANS"**I**nfarction (hemorrhagic) **T**rauma **H**ypertensive hemorrhage **A**rteriovenous malformation **C**oagulopathy **A**neurysm, **A**myloid angiopathy **N**eoplasm: metastasis / primary neoplasm **S**inus thrombosis

Notes:

Dense Cerebral Mass *Substrate:*calcification / hemorrhage / dense proteinA.VESSEL1.Aneurysm2.Arteriovenous malformation3.Hematoma (acute / subacute)B.TUMOR1.Lymphoma2.Medulloblastoma3.Meningioma4.Metastasis(a)from mucinous-producing adenocarcinoma(b)hemorrhagic metastases: melanoma, choriocarcinoma, hypernephroma, bronchogenic carcinoma, breast carcinoma (rarely)

Notes:

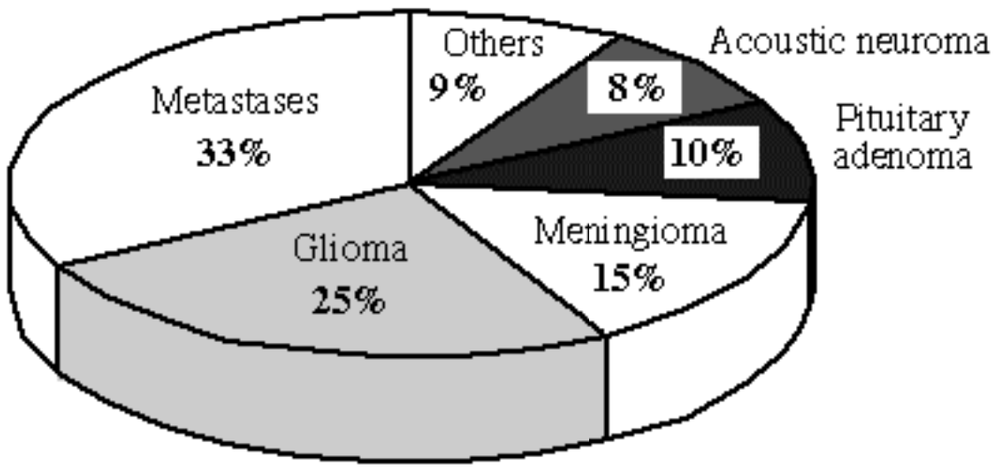
Dense Lesion Near Foramen Of Monro A.INTRAVENTRICULAR LESION1.Colloid cyst2.Meningioma3.Choroid plexus tumor / granuloma4.AVM of septal, thalamostriate, internal cerebral veinsB.PERIVENTRICULAR MASS1.Primary CNS lymphoma2.Tuberous sclerosis(a)subependymal tuber(b)giant cell astrocytoma3.Metastasis from mucin-producing adenocarcinoma / hemorrhagic metastasis (melanoma, choriocarcinoma, hypernephroma, bronchogenic carcinoma, breast carcinoma)4.Glioblastoma of septum pellucidumC.MASSES PROJECTING SUPERIORLY FROM SKULL BASE1.Pituitary adenoma2.Craniopharyngioma3.Aneurysm4.Dolichoectatic basilar artery

Notes:

Classification Of Primary CNS Tumors A.TUMORS OF BRAIN AND MENINGES(a)GliomasAstrocytoma (50%) 1.Astrocytoma (astrocytoma grades I - II)2.Glioblastoma (astrocytoma grades III - IV)Oligodendroglioma Paraglioma 1.Ependymoma2.Choroid plexus papillomaGanglioglioma Medulloblastoma (b)Pineal tumor1.Germinoma2.Teratoma3.Pineocytoma4.Pineoblastoma(c)Pituitary tumor1.Pituitary adenoma2.Pituitary carcinoma(d)Meningioma(e)Nerve sheath tumor1.Schwannoma2.Neurofibroma(f)Miscellaneous1.Sarcoma2.Lipoma3.HemangioblastomaB.TUMORS OF EMBRYONAL REMNANTS(a)Craniopharyngioma(b)Colloid cyst(c)Teratoid tumor1.Epidermoid2.Dermoid3.Teratoma

Notes:

Incidence Of Brain Tumors =9% of all primary neoplasms (5th most common primary neoplasm); 5-10 cases per 100,000 population per year; account for 1.2% of autopsied deathsIN ALL AGE GROUPS:IN PEDIATRIC AGE GROUP:Glioma34%Astrocytoma50%Meningioma17%Medulloblastoma15%Metastasis12%Ependymoma10%Pituitary adenoma6%Craniopharyngioma6%Neurinoma4%Choroid plexus papilloma2%Sarcoma3%Granuloma3%Craniopharyngioma2%Hemangioblastoma2%



Intracranial Tumors in Adult Population

Notes:

CNS Tumors Presenting At Birth 1.Hypothalamic astrocytoma2.Choroid plexus papilloma / carcinoma3.Teratoma4.Primitive neuroectodermal tumor5.Medulloblastoma6.Ependymoma7.Craniopharyngioma

Notes:

CNS Tumors In Pediatric Age Group *Incidence:* 2.4:100,000 (<15 years of age); 2nd most common pediatric tumor (after leukemia); 15% of all pediatric neoplasms; 15-20% of all primary brain tumors; M > F ● increased intracranial pressure ● increasing head sizeA.SUPRATENTORIAL (50%)*Age:*first 2-3 years of lifeCovering of brain:dural sarcoma, schwannoma, meningioma (3%)Cerebral hemisphere:astrocytoma (37%), oligodendrogliomaCorpus callosum :astrocytoma3rd ventricle:colloid cyst, ependymomaLateral ventricle:ependymoma (5%), choroid plexus papilloma (12%)Optic chiasm:craniopharyngioma (12%), optic nerve glioma (13%), teratoma, pituitary adenomaHypothalamus:glioma (8%), hamartomaPineal region:germinoma, pinealoma, teratoma (8%)B.INFRATENTORIAL (50%)*Age:*4-11 yearsCerebellum:astrocytoma (31-33%), medulloblastoma (26-31%)Brainstem:glioma (16-21%)4th ventricle:ependymoma (6-14%), choroid plexus papilloma*mnemonic:*"BE MACHO"**B**rainstem glioma **E**pendymoma **M**edulloblastoma **A**VM **C**ystic astrocytoma **H**emangioblastoma **O**ther ***Supratentorial MidlineTumors*** 1.Optic + hypothalamic glioma (39%)2.Craniopharyngioma (20%)3.Astrocytoma (9%)4.Pineoblastoma (9%)5.Germinoma (6%)6.Lipoma (6%)7.Teratoma (3.5%)8.Pituitary adenoma (3.5%)9.Meningioma (2%)10.Choroid plexus papilloma (2%) ***Supratentorial IntraventricularTumors*** (a)Lateral ventricle (3/4)1.Choroid plexus tumor (44%)2.Giant cell astrocytoma in tuberous sclerosis (19%)3.Hemangioma in Sturge-Weber syndrome (12%)(b)Third ventricle (1/4)1.Astrocytoma (13%)2.Choroid plexus tumor (6%)3.Meningioma (6%)
CLASSIFICATION BY HISTOLOGY 1.Astrocytic tumors (33.5%)2."Primitive" neuroectodermal tumor = PNET (21%) highly malignant neoplasms originating from germinal matrix + containing glial + neural elements-Medulloblastoma (16%)-Ependymoblastoma (2.5%)-PNET of cerebral hemisphere (2.5%)3.Mixed gliomas (16%)4.Malformative tumors (11.5%)-Craniopharyngioma (5.5%)-Lipoma (4.5%)-Dermoid cyst (1%)-Epidermal cyst (0.5%)5.Choroid plexus tumors (4%)6.Ependymal tumors (4%)7.Tumors of meningeal tissues (3.5%)-Meningioma (3%)-Meningeal sarcoma (0.5%)8.Germ cell tumors (2.5%)-Germinoma (1.5%)-Teratomatous tumor (1%)9.Neuronal tumors-Gangliocytoma (1.5%)10.Tumors of neuroendocrine origin-Pituitary adenoma (1%)11.Oligodendroglial tumors (0.5%)12.Tumors of blood vessel-Hemangioma (1%)

Notes:

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Multifocal CNS Tumors A.METASTASES FROM PRIMARY CNS TUMOR(a)via commissural pathways: corpus callosum, internal capsule, massa intermedia(b)via CSF: ventricles / subarachnoid cisterns(c)satellite metastasesB.MULTICENTRIC CNS TUMOR(a)true multicentric gliomas (4%)(b)concurrent tumors of different histology (coincidental)C.MULTICENTRIC MENINGIOMAS (3%) without neurofibromatosisD.MULTICENTRIC PRIMARY CNS LYMPHOMAE.PHAKOMATOSES1.Generalized neurofibromatosis:meningiomatosis, bilateral acoustic neuromas, bilateral optic nerve gliomas, cerebral gliomas, choroid plexus papillomas, multiple spine tumors, AVMs 2.Tuberous sclerosis:subependymal tubers, intraventricular gliomas (giant cell astrocytoma), ependymomas 3.von Hippel-Lindau disease:retinal angiomatosis, hemangioblastomas, congenital cysts of pancreas + liver, benign renal tumors, cardiac rhabdomyomas

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Notes:

CNS Tumors Metastasizing Outside CNS *mnemonic:*"MEGO"**M**edulloblastoma **E**pendymoma **G**lioblastoma multiforme **O**ligodendroglioma

Notes:

Calcified Intracranial Mass *mnemonic:*"Ca^{2+} COME"**C**raniopharyngioma **A**strocytoma, **A**neurysm **C**horoid plexus papilloma **O**ligodendroglioma **M**eningioma **E**pendymoma

Notes:

Avascular Mass Of Brain *mnemonic:*"TEACH"**T**umor: astrocytoma, metastasis, oligodendroglioma **E**dema **A**bscess **C**yst, **C**ontusion **H**ematoma, **H**erpes

Notes:

Jugular Foramen Mass 1.Glomus tumor2.Meningioma3.Neuroma4.Metastasis

Notes:

Dumbbell Mass Spanning Petrous Apex 1.Large trigeminal schwannoma2.Meningioma3.Epidermoid cyst

Notes:

Posterior Fossa Tumor In Adult *Extra-axialIntra-axial* 1. Acoustic neuroma1.Metastasis (lung, breast) 2. Meningioma2.Hemangioblastoma 3. Chordoma3.Lymphoma 4. Choroid plexus papilloma4.Lipoma 5. Epidermoid

Notes:

Cystic Mass In Cerebellar Hemisphere 1.Hemangioblastoma2.Cerebellar astrocytoma3.Metastasis4.Lateral medulloblastoma (= "cerebellar sarcoma")5.Choroid plexus papilloma with lateral extension

Notes:

Cerebellopontine Angle Tumor =extra-axial tumor arising in CSF-filled space bound by pons + cerebellar hemisphere + petrous bone*Incidence:*5-10% of all intracranial tumors ● cranial neuropathy: high frequency hearing loss (n. VIII), tinnitus + facial motor dysfunction (n. V II), facial sensory dysfunction (n. V), taste disturbance (chorda tympani) ● signs of posterior fossa mass effect: headache, nausea, vomiting, disequilibrium, ataxia ● hemifacial spasm, trigeminal neuralgia (tic douloureux)√ may widen CSF space (cistern) in 25%√ bone erosion / hyperostosis √ sharp margination with brain*Types:* 1.Acoustic neuroma = schwannoma (80-90%):from intracanalicular portion of 8th cranial nerve2.Meningioma (10-18%)2nd most common extra-axial mass in posterior fossa; <5% of all intracranial meningiomas; larger + more hemispheric in shape + more homogeneously enhancing than acoustic neuroma 3.Epidermoid inclusion cyst (5-9%)4.Arachnoid cyst (<1%)5.Aneurysm of basilar / vertebral / posterior inferiorcerebellar artery:congenital berry aneurysm / saccular aneurysm / atherosclerotic dolichoectasia 6.Choroid plexus papilloma7.Ependymoma8.Trigeminal neuromafrom gasserian ganglion within Meckel cave in the most anteromedial portion of petrous pyramid / trigeminal nerve root 9.Glomus jugulare tumorwithin adventitia of bulb of jugular vein at base of petrous bone with invasion of posterior fossa 10.Chordoma11.Exophytic brainstem glioma*Histo:*usually diffuse fibrillary astrocytoma12.Metastasis (0.2-2%)13.Lipoma (<1%) *mnemonic:*"**E**ver **G**rave **C**erebello**P**ontine **A**ngle **M**asses"**E**pidermoid **G**lomus jugulare tumor **C**hondroma, **C**hordoma, **C**holesteatoma **P**ituitary tumor, **P**ontine glioma (exophytic) **A**coustic + trigeminal neuroma, **A**neurysm of basilar / vertebral artery, **A**rachnoid cyst **M**eningioma, **M**etastasis LOW-ATTENUATION EXTRA-AXIAL LESION: 1.Acoustic schwannoma (occasionally low-density mass)2.Epidermoid tumor3.Arachnoid cyst

Notes:

Lesion Expanding Cavernous Sinus A.TUMOR1.Trigeminal schwannoma2.Pituitary adenoma3.Parasellar meningioma4.Parasellar metastasis5.Invasion by tumor of skull baseB.VESSEL1.Internal carotid artery aneurysm2.Carotid-cavernous fistula3.Cavernous sinus thrombosisC.TOLOSA-HUNT SYNDROME= granulomatous invasion of cavernous sinus

Notes:

Gyral Enhancement A.MENINGEAL TUMOR(a)Meningeal carcinomatosis from systemic tumor:eg, breast carcinoma, small cell carcinoma of lung, malignant melanoma, lymphoma / leukemia (b)Seeding primary CNS tumor:1.Medulloblastoma2.Pineoblastoma3.EpendymomaB.MENINGITISpyogenic, tuberculous, fungal, cysticercosis, sarcoidosis C.SEQUELAE OF SUBARACHNOID HEMORRHAGE(from fibroblastic proliferation) D.SUBACUTE BRAIN INFARCT *mnemonic:*"CAL MICE"**C**erebritis **A**rteriovenous malformation **L**ymphoma **M**eningitis **I**nfarct **C**arcinomatosis **E**ncephalitis

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Notes:

Ring-enhancing Lesion Of Brain *Cause:* A.NEOPLASM1.Primary neoplasm: high-grade glioma, meningioma, lymphoma, leukemia, pituitary macroadenoma, acoustic neuroma, craniopharyngioma2.Metastatic carcinoma + sarcomaB.ABSCESS1.Abscess: bacterial, fungal, parasitic2.Empyema of epidural / subdural / intraventricular spacesC.HEMORRHAGIC-ISCHEMIC LESION1.Resolving infarction2.Aging hematoma3.Operative bed following resection4.Thrombosed aneurysmD.DEMYELINATING DISORDER1.Radiation necrosis2.Tumefactive demyelinating lesion ("singular sclerosis")3.Necrotizing leukoencephalopathy after methotrexate *Pathogenesis:* (1)hypervascular margin of lesion = granulation tissue / peripheral vascular channels / hypervascular tumor capsule(2)breakdown of blood-brain barrier = leakage of contrast out of abnormally permeable vessels into extracellular fluid space(3)hypodense center = avascular / hypovascular (requires time to fill) / cystic degeneration*Incidence of ring blush:* abscess (in 73%); glioblastoma (in 48%); metastasis (in 33%); grade II astrocytoma (in 26%) [NOT in grade I astrocytoma] *mnemonic:*"MAGIC DR"**M**etastasis **A**bscess / cerebritis **G**lioma, **G**lioblastoma multiforme **I**mpact, **I**nfarct (resolving) **C**ontusion **D**emyelinating disease **R**esolving hematoma ***Ring-enhancing Lesion Crossing Corpus Callosum*** *mnemonic:*"GAL"**G**lioblastoma multiforme (butterfly glioma) **A**strocytoma **L**ymphoma

Notes:

Dense And Enhancing Lesions 1.Aneurysm2.Meningioma3.CNS lymphoma4.Medulloblastoma5.Metastasis

Notes:

Multifocal Enhancing Lesions 1.Multiple infarctions2.Arteriovenous malformations3.Multifocal primary / secondary neoplasms4.Multifocal infectious processes5.Demyelinating diseases: eg, multiple sclerosis

Notes:

Innumerable Small Enhancing Cerebral Nodules A.METASTASESB.PRIMARY CNS LYMPHOMAC.DISSEMINATED INFECTION1.Cysticercosis2.Histoplasmosis3.TuberculosisD.INFLAMMATION1.Sarcoidosis2.Multiple sclerosisE.SUBACUTE MULTIFOCAL INFARCTIONfrom hypoperfusion, multiple emboli, cerebral vasculitis (SLE), meningitis, cortical vein thrombosis

Notes:

Enhancing Lesion In Internal Auditory Canal A.NEOPLASTIC1.Acoustic schwannoma2.Ossifying hemangiomaB.NONNEOPLASTIC1.Sarcoidosis2.Meningitis3.Postmeningitic / postcraniotomy fibrosis4.Vascular loop of anterior inferior cerebellar a.

Notes:

Classification Of Vascular CNS Anomalies A.VASCULAR MALFORMATION(a)arterial = arteriovenous malformation (AVM)1.Facial / brain arteriovenous malformation2.Vein of Galen malformation(b)capillary = telangiectasia1.Facial port wine stain ● commonly asymptomatic(c)venous = venous malformation=tangle of abnormal varices of a "caput-medusae" / "spoked-wheel" configuration draining into a dilated cortical vein ● soft + compressible without thrills / pulsations ● distension with Valsalva maneuver ● commonly asymptomaticLocation:white matter with normal intervening brain parenchyma*Cx (uncommon):*hemorrhage, ischemia1.Venous angioma2.Sinus pericranii(d)lymphatic1.Cystic hygroma(e)combinations1.Sturge-Weber disease2.Rendu-Osler-Weber diseaseB.VASCULAR TUMOR1.Hemangioma(a)capillary hemangioma: seen in children, involution by 7 years of age in 95%(b)cavernous hemangioma: seen in adults, no involution√ thrombosed blood + hemosiderin√ normal angiogram2.Hemangiopericytoma3.Hemangioendothelioma4.Angiosarcoma Occult / cryptic vascular malformation 1.Cavernous hemangioma2.Capillary telangiectasia

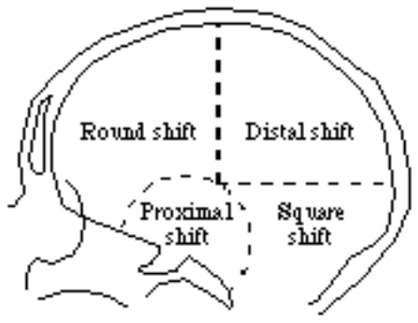
Notes:

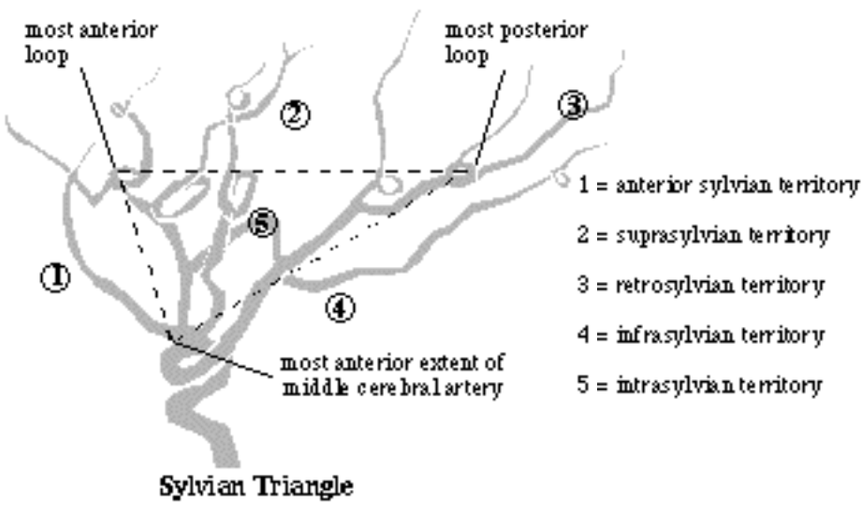
Occlusive Vascular Disease (a)Embolic state:√ single vascular territory(b)Hypoperfusive state:√ multiple vascular territories*Cause:* 1.Vasospasm from subarachnoid hemorrhage 2.Embolic infarction (50%)(a)thrombus (atrial fibrillation, valvular disease, Atheromatous plaques of extracerebral arteries, fibromuscular dysplasia, intracranial aneurysm, surgery, paradoxic emboli, sickle cell disease, atherosclerosis, thrombotic thrombocytopenic purpura) ● fluctuating blood pressures ● hypercoagulability√ cerebral petechial hemorrhage within cortical / basal gray matter during 2nd week (from fragments of embolus) in up to 40%; initial ischemia is followed by reperfusion (= HALLMARK of embolic infarction)√ "supernormal artery" on NECT = high-density material lodged in cerebral vessel near major bifurcations √ atheromatous narrowing of vessels(b)fat(c)nitrogen3.Watershed infarctinvolving deep white matter between two adjacent vascular beds in global hypoperfusion secondary to poor cardiac output / cervical carotid artery occlusion ◊6% of cerebral infarcts have hemorrhage (red infarct) ● stroke (3rd most common cause of death in USA, 5% of stroke syndromes are caused by underlying tumor) ● TIA = transitory ischemic attack: clears within 24 hours ● RIND = reversible ischemic neurologic deficit: still evident >24 hours with eventual total recovery ● amaurosis fugax = transient monocular blindness ● weakness / numbness in an extremity ● aphasia ● dizziness, diplopia, dysarthria (Vertebrobasilar ischemia)4.Hypertension(a)Hypertensive encephalopathy√ diffuse white matter hypodensity (edema secondary to arterial spasm)(b)Hypertensive hemorrhageLocation:basal ganglia (putamen, external capsule), thalamus, pons, cerebellum(c)Lacunar infarction(d)Subcortical arteriosclerotic encephalopathy5.Amyloidosisinvolvement of small- + medium-sized arteries of meninges + cortex ● normotensive patient >65 years of age√ multiple simultaneous / recurrent cortical hemorrhages6.Vasculitis(a)Bacterial meningitis, TB, syphilis, fungus, virus, rickettsia(b)Collagen-vascular disease: Wegener granulomatosis, polyarteritis nodosa, SLE, scleroderma, dermatomyositis(c)Granulomatous angitis: giant cell arteritis, sarcoidosis, Takayasu disease, temporal arteritis(d)Inflammatory arteritis: rheumatoid arteritis, hypersensitivity arteritis, Behçet disease, lymphomatoid granulomatosis(e)Drug-induced: IV amphetamine, ergot preparations, oral contraceptives(f)Radiation arteritis = mineralizing microangiopathy(g)Moyamoya disease7.Anoxic encephalopathycardiorespiratory arrest, near-drowning, drug overdose, CO poisoning 8.Venous thrombosis
Multiple Infarctions typical in extracranial occlusive disease, cardiac output problems, small vessel disease; in 6% from a shower of emboli Location:usually bilateral + supratentorial (3/4); supra- and infratentorial (1/4)

Notes:

Displacement Of Vessels

A.ARTERIAL SHIFT(a)Pericallosal arteries1.Round shift = frontal lesion anterior to coronal suture2.Square shift = lesion behind foramen of Monro in lower half of hemisphere3.Distal shift = posterior to coronal suture in upper half of hemisphere4.Proximal shift = basifrontal lesion / anterior middle cranial fossa including anterior temporal lobe(b)Sylvian triangle=branches of MCA within sylvian fissure on outer surface of insula form a loop upon reaching the upper margin of the insula; serves as angiographic landmark for localizing supratentorial massesLocation of lesion: -anterior sylvianfrontal region-suprasylvianposterior frontal + parietal-retrosylvianoccipital, parieto-occipital-infrasylviantemporal lobe + extracerebral region-intrasylvianusually due to meningioma-lateral sylvianfrontal, frontotemporal, parietotemporal-central sylviandeep posterior frontal, basal ganglia





Sylvian Triangle

B.CEREBRAL VEINS=indicate the midline of the posterior part of the forebrain showing the exact location of the roof of the 3rd ventricle

Notes:

Bilateral Basal Ganglia Lesions In Childhood Basal ganglia are susceptible to damage during childhood because of high energy requirements (ATP) mandating a rich blood supply + high concentration of trace metals (iron, copper, manganese) ● increased irritability, lethargy, dystonia ● seizure, behavioral changes√ bilateral necrosis of basal ganglia ACUTE CAUSES A.Compromise of vascular supply1.Hemolytic-uremic syndromecausing microthrombosis of basal ganglia, thalami, hippocampi, cortex 2.Encephalitis (usually viral agents)B.Compromise of nutrient supply1.Hypoxia: respiratory arrest, near drowning, strangling, barbiturate intoxication2.Hypoglycemia√ hemorrhage rarely seen3.Osmotic myelinolysis√ associated central pontine location commonC.Acute poisoning1.Carbon monoxide√ preferentially affects globus pallidusrare in children: 2.Hydrogen sulfide3.Cyanide poisoning4.Methanol poisoning CHRONIC CAUSES A.Inborn errors of metabolism1.Leigh disease=subacute necrotizing encephalomyelopathy=autosomal recessive disorder characterized by deficiencies in pyruvate carboxylase, pyruvate dehydrogenase complex, cytochrome *c* oxidase resulting in anaerobic ATP production ● lactic acidosis (elevated ratio of lactate to pyruvate in CSF + serum)√ propensity to involve putamen2.Wilson disease=hepatolenticular degeneration=increased deposition of copper in brain + liver ● decreased levels of serum copper + ceruloplasmin ● increased urinary copper excretion√ cell damage of lenticular nucleus (= lenslike configuration of putamen + globus pallidus)3.Mitochondrial encephalomyelopathies=subset of lactic acidemias with structurally abnormal mitochondria ● "ragged red" fibers in muscle biopsy4.Maple syrup urine disease=inability to catabolize branched-chain amino acids (leucine, isoleucine, valine) ● urine smells of maple syrup5.Methylmalonic acidemia=group of genetically distinct autosomal recessive disorders of organic acid metabolism affecting conversion of methylmalonyl-CoA to succinyl-CoA ● accumulation of methylmalonic acid in blood + urineB.Degenerative disease1.Huntington disease2.Dystrophic calcificationsC.Dysmyelinating diseasebasal ganglia are a mixture of gray + white matter 1.Canavan disease2.Metachromatic leukodystrophyD.Others1.Neurofibromatosis type 1

Notes:

Low-attenuation Lesion In Basal Ganglia 1.Poisoning: carbon monoxide, barbiturate intoxication, hydrogen sulfide poisoning, cyanide poisoning, methanol intoxication2.Hypoxia3.Hypoglycemia4.Hypotension (lacunar infarcts)5.Wilson disease

Notes:

Basal Ganglia Calcification *Prevalence in children:*1.1 - 1.6%A.PHYSIOLOGIC WITH AGINGB.ENDOCRINE1.Hypoparathyroidism, pseudo~, pseudopseudo~ (60%)2.Hyperparathyroidism3.HypothyroidismC.METABOLIC1.Leigh disease2.Mitochondrial cytopathy(a)Kearns-Sayre syndrome = ophthalmoplegia, retinal pigmentary degeneration, complete heart block, short stature, mental deterioration(b)MELAS = **M**itochondrial myopathy, **E**ncephalopathy, **L**actic acidosis, **A**nd **S**troke(c)MERRF = **M**yoclonic **E**pilepsy with **R**agged **R**ed **F**ibers3.Fahr disease = familial cerebrovascular ferrocalcinosisD.CONGENITAL / DEVELOPMENTAL1.Familial idiopathic symmetric basal ganglia calcification2.Hastings-James syndrome3.Cockayne syndrome4.Lipoid proteinosis = hyalinosis cutis5.Neurofibromatosis6.Tuberous sclerosis7.Oculocraniosomatic disease8.Methemoglobinopathy9.Down syndromeE. INFLAMMATION / INFECTION1.Toxoplasmosis, congenital rubella, CMV2.Measles, chicken pox3.Pertussis, Coxsackie B virus4.Cysticercosis5.Systemic lupus erythematosus6.AIDSF.TRAUMA1.Childhood leukemia following methotrexate therapy2.S/P radiation therapy3.Birth anoxia, hypoxia4.Cardiovascular eventG. TOXIC 1.Carbon monoxide poisoning2.Lead intoxication3.Nephrotic syndrome *mnemonic:*"BIRTH"**B**irth anoxia **I**diopathic (most common), **I**nfarct **R**adiation therapy **T**oxoplasmosis / CMV **H**ypoparathyroidism / pseudoHPT

Notes:

Linear Echogenic Foci In Thalamus + Basal Ganglia A.IN UTERO INFECTION=destruction of wall of lenticulostriate arteries + replacement by deposits of amorphous granular material1.TORCH agents: Toxoplasma, rubella virus, cytomegalovirus, herpes virus2.Syphilis3.Human immunodeficiency virusB.CHROMOSOMAL ABNORMALITY1.Down syndrome2.Trisomy 13C.OTHERS (anoxic injury?)1.Perinatal asphyxia, respiratory distress syndrome, cyanotic congenital heart disease, necrotizing enterocolitis2.Fetal alcohol syndrome3.Nonimmune hydrops

Notes:

Destruction Of Sella 1.Pituitary adenoma2.Suprasellar tumor3.Carcinoma of sphenoid + posterior ethmoid sinus√ opacification of sinus + destruction of walls√ associated with nasopharyngeal mass (common)4.Nasopharyngeal carcinoma(a)squamous cell carcinoma(b)lymphoepithelioma = Schmincke tumor = non-keratinizing form of squamous cell carcinoma√ sclerosis of adjacent bone5.Metastasis to sphenoidfrom breast, kidney, thyroid, colon, prostate, lung, esophagus 6.Primary tumor of sphenoid bone (rare)osteogenic sarcoma, giant cell tumor, plasmacytoma 7.Chordoma8.Mucocele of sphenoid sinus (uncommon)9.Enlarged 3rd ventricleaqueductal stenosis from infratentorial mass, maldevelopment

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Notes:

J-shaped Sella *mnemonic:*"CONMAN" **C**hronic hydrocephalus **O**ptic glioma, **O**steogenesis imperfecta **N**eurofibromatosis **M**ucopolysaccharidosis **A**chondroplasia **N**ormal variant

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Notes:

Enlarged Sella A.PRIMARY TUMOR1.Pituitary adenoma2.Craniopharyngioma3.Meningioma: hyperostosis4.Optic glioma: J-shaped sellaB.PITUITARY HYPERPLASIA1.Hypothyroidism2.Hypogonadism3.Nelson syndrome (occurring in 7% of patients subsequent to adrenalectomy)C.CSF-SPACE1.Enlarged 3rd ventricle2.Hydrocephalus3.Empty sellaD.VESSEL1.Arterial aneurysm2.Ectatic internal carotid artery *mnemonic:*"CHAMPS"**C**raniopharyngioma **H**ydrocephalus (empty sella) **A**VM, **A**neurysm **M**eningioma **P**ituitary adenoma **S**arcoidosis, TB ***Pituitary Gland Enlargement*** 1.Neoplasm: eg, pituitary gland adenoma2.Hypertrophy: primary precocious puberty, primary hypothyroidism3.Lymphocytic hypophysitis4.Infection5.Severe dural AV fistula

Notes:

Intrasellar Mass 1.Pituitary adenoma / carcinoma (most common cause)2.Craniopharyngioma (2nd most common cause)3.Meningioma: from surface of diaphragm / tuberculum sellae4.Chordoma5.Metastasis: lung, breast, prostate, kidney, GI tract, spread from nasopharynx6.Intracavernous ICA aneurysm: bilateral in 25%7.Pituitary abscess: rapidly expanding mass associated with meningitis8.Empty sella9.Rathke cleft cyst: commonly at junction of anterior + posterior pituitary gland10.Granular cell tumor = myeloblastoma: benign neoplasm of posterior pituitary gland11.Granuloma: sarcoidosis, giant cell granuloma, TB, syphilis, eosinophilic granuloma12.Lymphoid adenohypophysitis13.Pituitary hyperplasia, eg, in Nelson syndrome

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Notes:

Hypointense Lesion Of Sella 1.Empty sella2.Pituitary stone (= pituilith)= sequela of autonecrosis of pituitary adenoma 3.Intrasellar aneurysm4.Persistent trigeminal artery5.Calcified meningioma6.Pituitary hemochromatosis (anterior pituitary lobe only)

Notes:

Parasellar Mass 1.Meningioma: tentorium cerebelli2.Neurinoma (III, IV, V_1 , V_2, VI)3.Metastasis: lung, breast, kidney, GI tract, spread from nasopharynx4.Epidermoid5.Aneurysm6.Carotid-cavernous fistula *mnemonic:*"SATCHMO"**S**ella neoplasm with superior extension, **S**arcoidosis **A**neurysm, ectatic carotid, carotid-cavernous sinus fistula, **A**rachnoid cyst **T**eratoma: dysgerminoma (usually), dermoid, epidermoid **C**raniopharyngioma, **C**hordoma **H**ypothalamic glioma, **H**istiocytoma, **H**amartoma **M**etastatic disease, **M**eningioma, **M**ucocele **O**ptic nerve glioma, neuroma

Notes:

Suprasellar Mass 1.Meningioma2.Craniopharyngioma: in 80% suprasellar3.Chiasmal + optic nerve gliomain 38% of neurofibromatosis; adolescent girls; *DDx:* chiasmal neuritis4.Hypothalamic glioma5.Hamartoma of tuber cinereum6.Infundibular tumormetastasis (esp. breast); glioma; lymphoma / leukemia; histiocytosis X; sarcoidosis, tuberculosis√ diameter of infundibulum >4.5 mm immediately above level of dorsum; cone-shaped (on coronal scan)7.Germinomamalignant tumor similar to seminoma (= "ectopic pinealoma") √ frequently calcified (teratoma)√ CSF spread (germinoma + teratocarcinoma)√ enhancement on CECT (common)8.Epidermoid / dermoid√ cystic lesion containing calcifications + fat√ minimal / no contrast enhancement9.Arachnoid cyst ● hydrocephalus (common), visual impairment ● endocrine dysfunction*Age:*most common in infancy10.Enlarged 3rd ventricle extending into pituitary fossa11.Suprasellar aneurysm√ rim calcification + eccentric position
Suprasellar Mass with Low Attenuation 1.Craniopharyngioma2.Dermoid / epidermoid3.Arachnoid cyst4.Lipoma5.Simple pituitary cyst6.Glioma of hypothalamus
Suprasellar Mass With Mixed Attenuation A.IN CHILDREN1.Hypothalamic-chiasmatic glioma2.Craniopharyngioma3.Hamartoma of tuber cinereum4.HistiocytosisB.IN ADULTS1.Suprasellar extension of pituitary adenoma2.Craniopharyngioma3.Epidermoid cyst4.Thrombosed aneurysm5.Low-grade hypothalamic / optic glioma6.Inflammatory lesion: sarcoidosis, TB, sphenoid mucocele ***Suprasellar Mass With Calcification*** A.CURVILINEAR1.Giant carotid aneurysm2.CraniopharyngiomaB.GRANULAR1.Craniopharyngioma2.Meningioma3.Granuloma4.Dermoid cyst / teratoma5.Optic / hypothalamic glioma (rare)

Notes:

Enhancing Supra- and Intrasellar Mass 1.Pituitary adenoma2.Meningioma3.Germinoma4.Hypothalamic glioma5.Craniopharyngioma

Notes:

Perisellar Vascular Lesion 1.ICA aneurysmGiant aneurysms are >2.5 cm in diameter√ destruction of bony sella / superior orbital fissure√ calcified wall / thrombus√ CECT enhancement, nonuniform with thrombosis2.Ectatic carotid artery√ curvilinear calcifications√ encroachment upon sella turcica3.Carotid-cavernous sinus fistula

Notes:

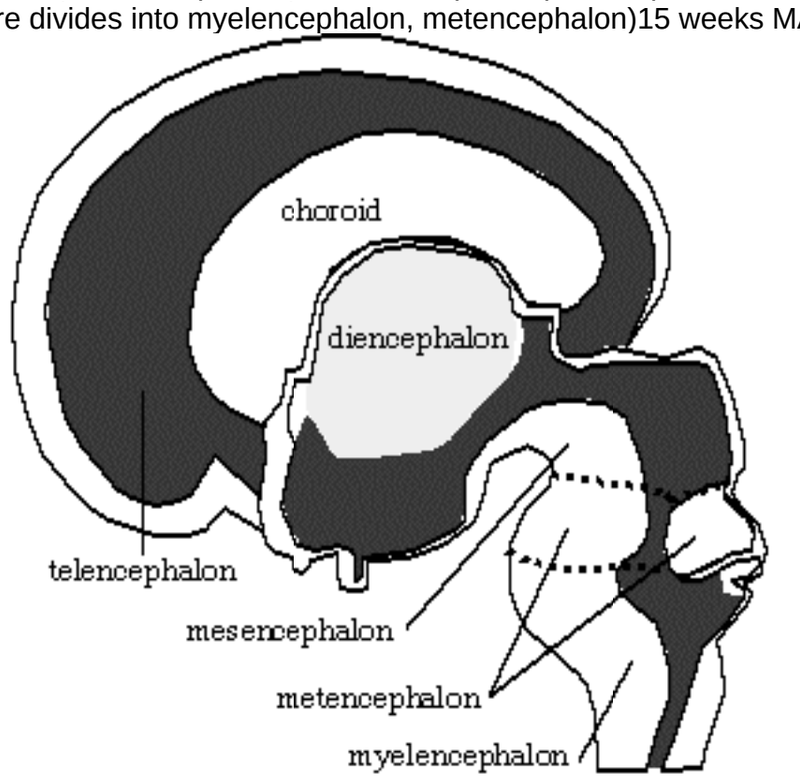
Classification Of Pineal Gland Tumors *Incidence of pineal mass:* <1% of all intracranial tumors, 4% of all childhood intracranial masses, 9% of all intracranial masses in Asia A.PRIMARY TUMOR(a)Germ cell origin (2/3)-forming embryonic tissue1.Germinoma (40 - 50%)2.Embryonal cell carcinoma3.Teratoma (15%): benign mature teratoma, benign immature teratoma, malignant teratoma-forming extraembryonic tissue4.Choriocarcinoma (<5%)5.Endodermal sinus tumor = yolk sac tumor(b)Pineal parenchymal cell origin (<15%)1.Pineocytoma2.Pineoblastoma(c)Other cell origin1.Retinoblastoma (trilateral retinoblastoma = left eye + right eye + pineal gland2.Astrocytoma3.Ependymoma4.Meningioma5.Hemangiopericytoma(d)Cysts1.Pineal cyst2.Malignant teratoma3.AVM, vein of Galen aneurysm4.Arachnoid cyst5.Inclusion cyst (dermoid / epidermoid)B.SECONDARY TUMORMetastasis:eg, lung carcinoma *DDx considerations:* -female:likely NOT germ cell tumor-hypodense matrix:likely NOT pineal cell tumor-distinct tumor margins:probably pineocytoma / teratoma / germinoma-calcification:likely NOT teratocarcinoma, metastasis, germinoma-CSF seeding:NOT teratoma-intense enhancement:likely NOT teratoma

Notes:

Intensely Enhancing Mass In Pineal Region 1.Germinoma2.Pineocytoma / -blastoma3.Pineal teratocarcinoma4.Glioma of brainstem / thalamus5.Subsplenial meningioma6.Vein of Galen aneurysm

Notes:

Neurulation neural plate=CNS originates as a plate of thickened ectoderm on the dorsal aspect of the embryoneural crest=elevation of the lateral margins of the neural plate; forms the peripheral nervous systemneural tube=invagination between the two neural crests; its wall forms the brain + spinal cord; its lumen forms the ventricles + spinal canal4.6 weeks MA:formation of neural tube5.6 weeks MA:rostral neuropore closes5.9 weeks MA:caudal neuropore closes6.0 weeks MA:3 primary brain vesicles develop (prosencephalon, mesencephalon, rhombencephalon) development of cervical flexure7.0 weeks MA:2 additional primary brain vesicles form out of rhombencephalon (pontine flexure divides into myelencephalon, metencephalon)15 weeks MA:dorsal portion of alar plates bulging into 4th ventricle have fused in



Sagittal Section Through Brain at 10–11 Weeks GA

midline to form cerebellar vermis

Notes:

Brain Growth =increase in thickness of brain mantle with relative constant ventricular width Most rapid brain growth from 12 to 24 weeks MA!

Notes:

Neuronal Migration 7th weeksubependymal neuronal proliferation = germinal matrix8th weekradial migration to cortex along radial glial fibers

Notes:

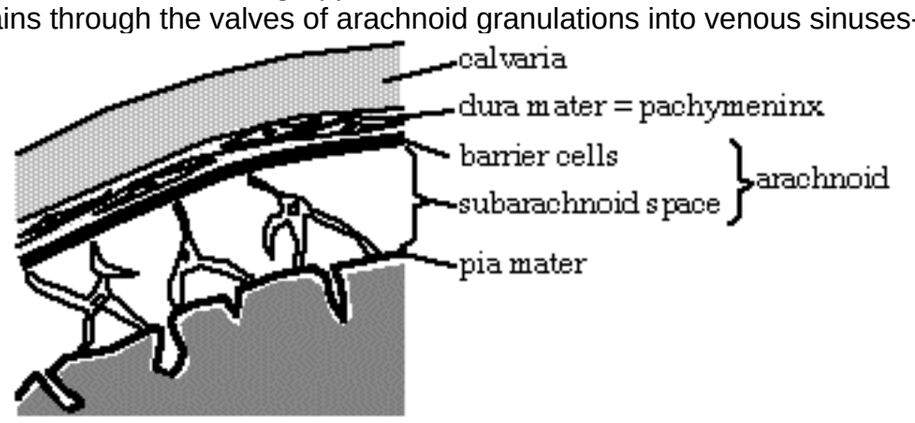
CLASSIFICATION OF BRAIN ANATOMY

A.PROSENCEPHALON = forebrain√ cerebrum, lateral ventricles, choroid, thalami, cerebellum sonographically visible at 12 weeks MA1.**Telencephalon** = cerebrum=cerebral hemispheres, putamen, caudate nucleus2.**Diencephalon** =thalamus, hypothalamus, epithalamus (= pineal gland + habenula), globus pallidusB.MESENCEPHALON = midbrain=short segment of brainstem above pons; traverses the hiatus in tentorium cerebelli; contains cerebral peduncles, tectum, colliculi (corpora quadrigemina)C.RHOMBENCEPHALON = hindbrain√ posterior cystic space of 4th ventricle sonogra-phically detectable between 8 and 10 weeks MA1.**Metencephalon** = cerebellar hemispheres, vermis2.**Myelencephalon** = medulla oblongata, ponsD.BRAINSTEM = mesencephalon + myelencephaloncontains(a)cranial nerve nuclei(b)sensory and motor tracts between thalamus, cerebral cortex, and spinal cord(c)reticular formation controlling respiration, blood pressure, gastrointestinal function, centers for arousal and wakefulness

Notes:

MENINGES OF BRAIN

A.CALVARIAB.EPIDURAL SPACE=created when dura becomes detached from calvariaC.PACHYMENINGES = DURA(a)outer dural layer= highly vascularized periosteum of calvaria (b)space for venous sinuses(c)inner dural layer= meningeal layer derived from meninx D.SUBDURAL SPACE=cleft formed in pathologic states within inner layer of duraE.LEPTOMENINGES1.Arachnoid=closely applied to inner surface of dura2.Subarachnoid space*Histo:*fine connective tissue + cellular septa link pia and arachnoid-contains CSF that drains through the valves of arachnoid granulations into venous sinuses-forms basal cisterns3.Pia materF.SUBPIAL



Meninges of Brain

SPACE=perivascular (Virchow-Robin) space

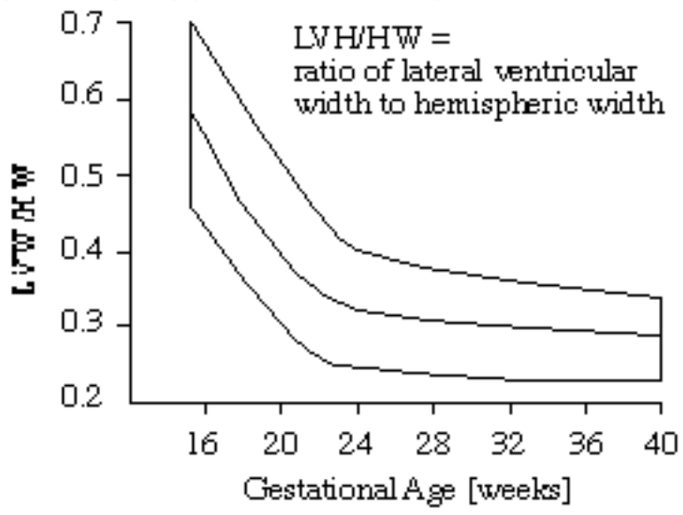
Notes:

CEREBROSPINAL FLUID
Total volume: 50 mL in newborn, 150 mL in adult Composition: inorganic salts like those in plasma, traces of protein + glucose *Production:* 0.3 - 0.4 mL/min resulting in 500 mL/day; secreted into ventricles by choroid plexuses (80 - 90%), 10-20% formed by parenchyma of the cerebrum + spinal cord *Circulation:* from ventricles through foramina of Magendie + Luschka of 4th ventricle into cisterna magna + basilar cisterns; 80% of CSF flows initially into suprasellar cistern + cistern of lamina terminalis, the ambient / superior cerebellar cisterns, eventually ascending over superolateral aspects of each hemisphere; 20% initially enters spinal subarachnoid space + eventually recirculates into cerebral subarachnoid space

Cerebral aqueduct

Notes:

Cerebral aqueduct pulsatile flow (due to brain motion during cardiac cycle) + net outflow into 4th ventricle; diameter of 2.6-4.2 mm; peak outflow velocity of 6-51 mm/sec; inflow velocity of 3-28 mm/sec *Absorption:* into venous system by (a)arachnoid villi of superior sagittal sinus (villi behave as one-way valves with an opening pressure between 20 - 50 mm of CSF)(b)cranial + spinal nerves with eventual absorption by lymphatics (50%)(c)prelymphatic channels of capillaries within brain parenchyma(d)vertebral venous plexuses, intervertebral veins, posterior intercostal + upper lumbar veins into azygos + hemiazygos veins



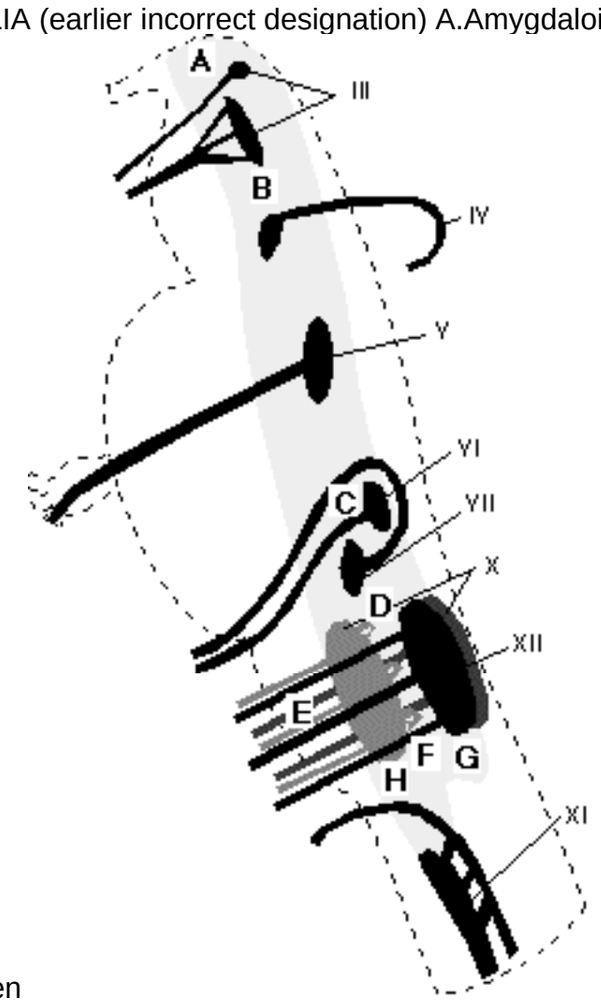
Notes:

PITUITARY GLAND
=HYPOPHYSIS CEREBRI within hypophysial fossa of sphenoid, covered superiorly by sellar diaphragm(= dura mater) which has an aperture for the infundibulum centrally Size: adult size is achieved at puberty Height in adult females=7 (range 4-10) mmHeight in adult males=5 (range 3- 7) mmShape: √ flat / downwardly convex superior border√ upwardly convex during puberty, pregnancy, in hypothyroidism (due to hyperplasia)A.ANTERIOR LOBE=larger anterior portion of adenohypophysis comprising 80% of pituitary gland volume*Origin:*ectodermal derivative of stomodeum*Function:* (a)chromophil cells1.acidophil cells = a cells growth hormone = somatotropin (STH), prolactin = lactogenic hormone (LTH) 2.basophil cells = b cellsadrenocorticotropin = adrenocorticotropic hormone (ACTH), thyrotropin = thyroid-stimulating hormone (TSH), follicle-stimulating hormone (FSH), interstitial-cell-stimulating hormone (ICSH), luteinizing hormone (LH), melanocyte-stimulating hormone (MSH) (b)chromophobe cells = 50% of epithelial cell population, of unknown significanceMRI: √ larger homogeneous component isointense to white matter on T1WI + T2WI√ prominent contrast enhancement (during first 3 minutes) due to lack of blood-brain barrier√ hyperintense in the newborn fading to normal adult signal by 2nd month of lifeB.PARS INTERMEDIA=posterior portion of adenohypophysis; separated from anterior lobe by hypophysial cleft in fetal life*Origin:*pouch of Rathke *Function:*termination point of short hypothalamic axons elaborating tropic hormones (= releasing factors + prolactin inhibiting factor), which are carried to anterior lobe via the portal system√ not visible with imaging techniquesC.POSTERIOR LOBE=major portion of neurohypophysis*Origin:*diencephalic outgrowth (termination point of axons from supraoptic + paraventricular nuclei of hypothalamus)*Function:*storage site for vasopressin (= antidiuretic hormone [ADH]) + oxytocin transported from paraventricular + supraoptic nuclei of hypothalamus along neurosecretory hypothalamohypophysial tractMRI: √ hyperintense on T1WI + isointense on T2WI in comparison with anterior lobe (? due to relaxing agent of phospholipid / neurosecretory granules / vasopressin)√ isointense in 10% of normal individualsD.PITUITARY STALK = INFUNDIBULUMarises from anterior aspect of floor of 3rd ventricle (infundibular recess) *Histo:*formed from axons of cells lying in supraoptic + paraventricular nuclei of hypothalamus√ joins posterior lobe at junction of anterior + posterior lobes√ up to 3 mm thick superiorly, up to 2 mm thick inferiorly√ usually in midline, may be slightly tilted to one sideMRI: √ prominent contrast enhancement

Notes:

BASAL NUCLEI

= BASAL GANGLIA (earlier incorrect designation) A.Amygdaloid bodyB.ClaustrumC.Corpus striatum(1)Caudate(2)Lentiform nucleus(a)pallidum = globus pallidus(b)putamen

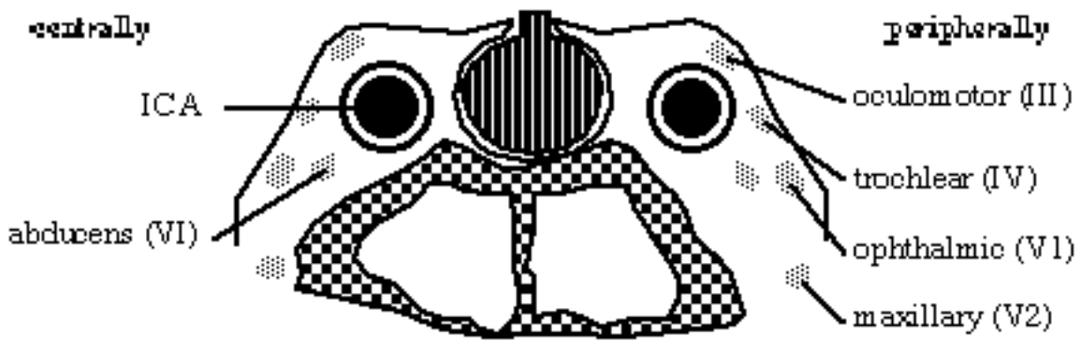


Cranial Nuclei of Brainstem and Reticular Formation

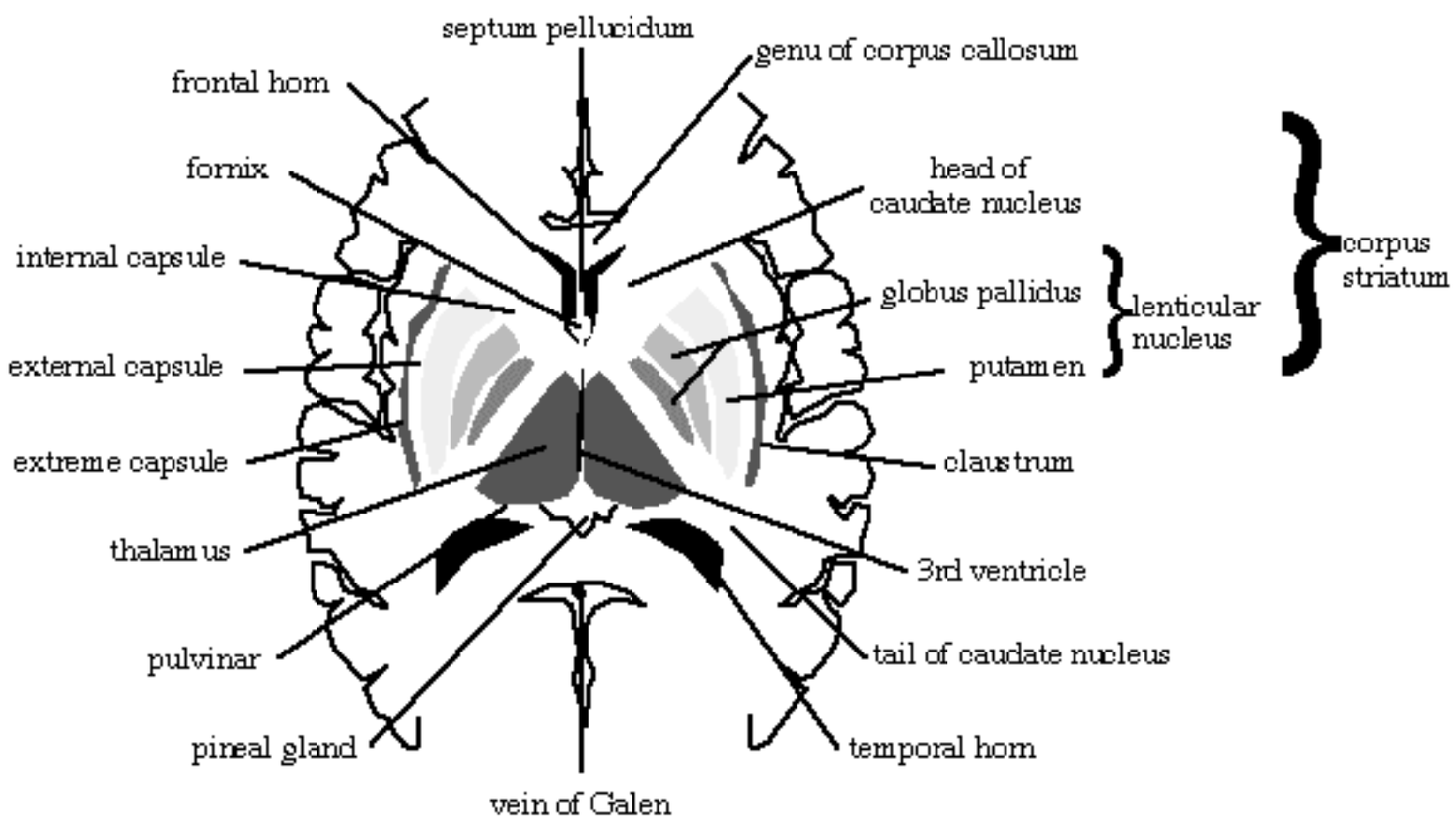
A = sleep, wakefulness, consciousness
B = visual spatial orientation, higher autonomic coordination of food intake
C = pneumotaxic center, coordination of breathing and circulation
D = swallowing
E = blood pressure, cardiac activity, vascular tone
F = expiration
G = area postrema = trigger zone for vomiting
H = inspiration

Notes:

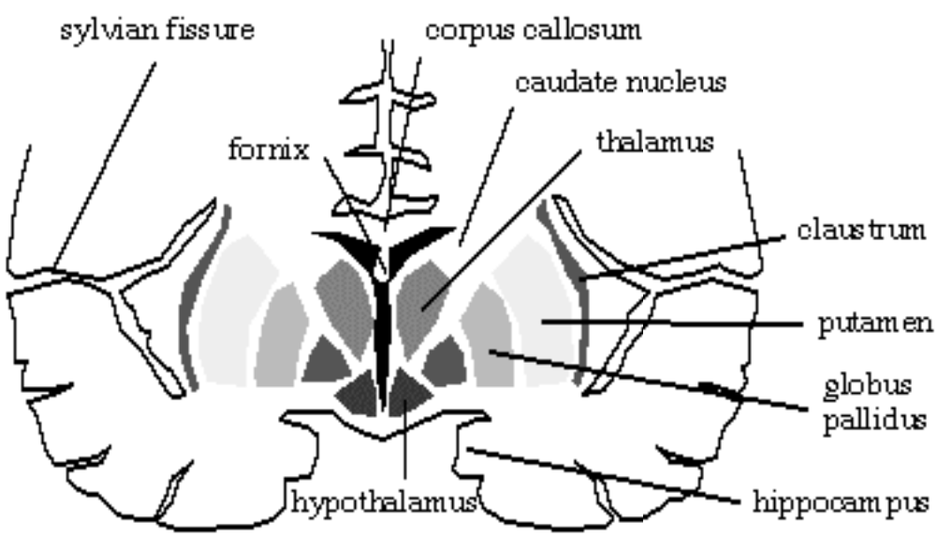
DIAGRAMS



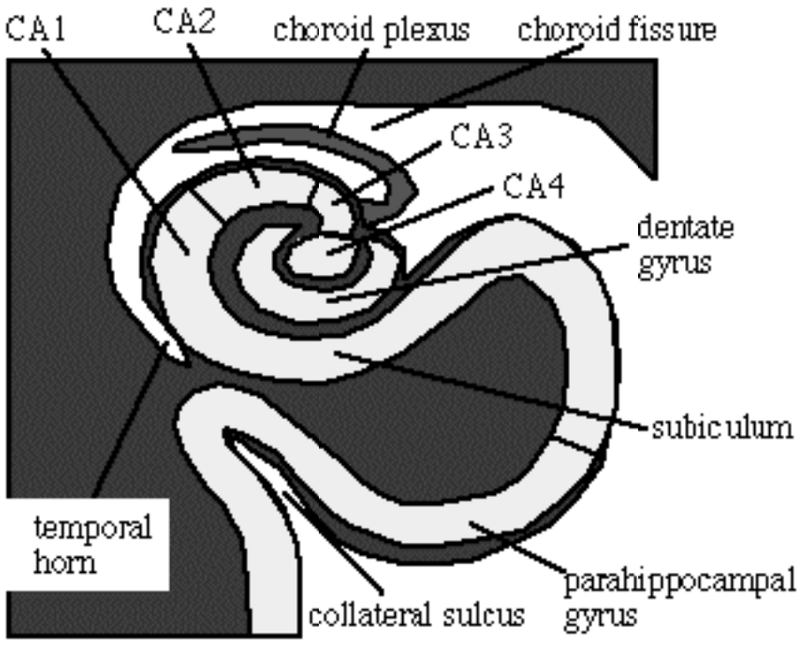
Cavernous Sinus (coronal view)



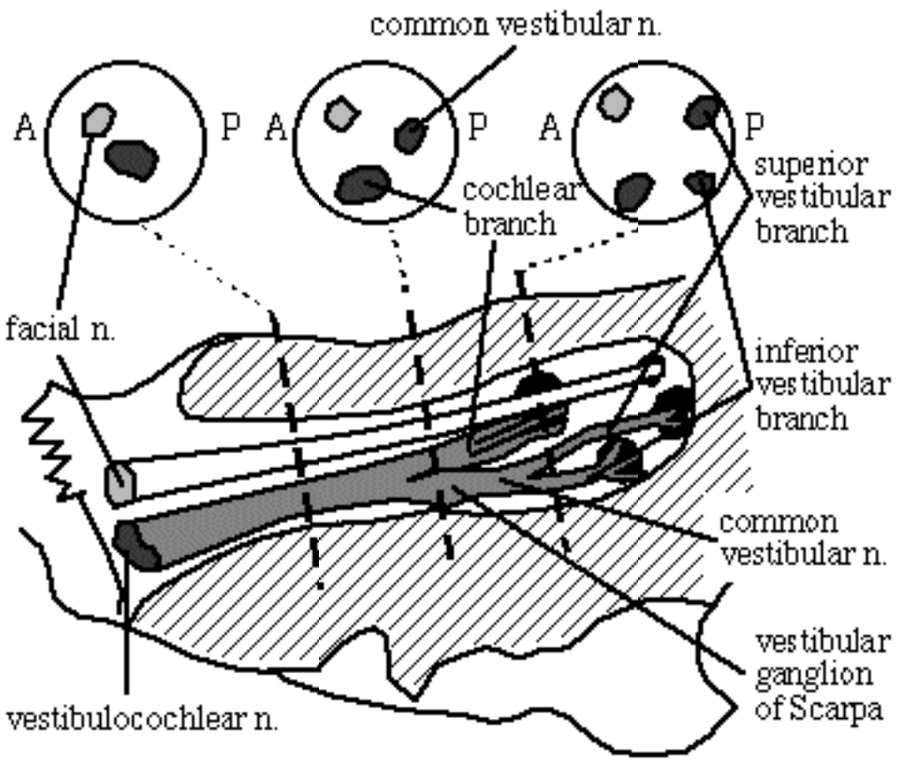
Axial Section Trough Level of Third Ventricle



Coronal Section Through Level of Basal Ganglia



Right Medial Temporal Lobe



InternalAuditory Canal

Posterior wall of IAC is removed; cross sections through IAC are displayed above; A = anterior, P = posterior

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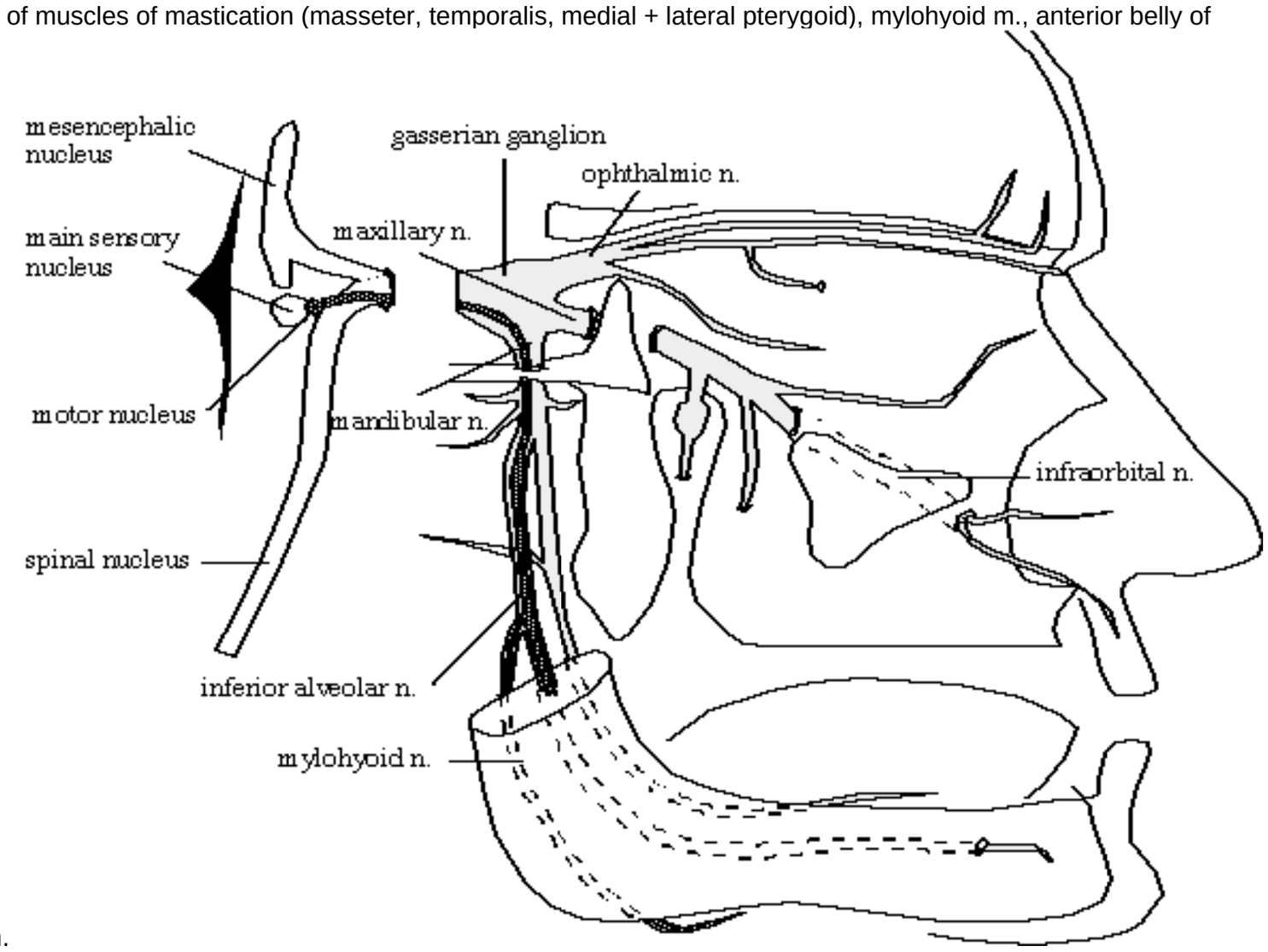
Notes:

PINEAL GLAND
Development: from area of ependymal thickening at the most caudal portion of roof of 3rd ventricle that evaginates into a pinecone-shaped mass during 7th week of gestation; initially contains ependyma lining in central cavity that connects with 3rd ventricle *Function:* 1.regulation of long-term biologic rhythm (eg, onset of puberty)2.regulation of short-term biologic rhythm (eg, diurnal / circadian) due to photoperiodic clues via accessory optic pathway*Histo:* (a)pinealocytes with dendritic processes (= neuronal cells) make up 95% of population(b)neuroglial supporting cells make up 5% of populationLocation:attached to upper aspect of posterior border of 3rd ventricle, lies within CSF of quadrigeminal cistern, anterior to pineal gland is cistern of velum interpositum (= cistern of transverse fissure)Size:8 mm long, 4 mm wide

Notes:

TRIGEMINAL NERVE (V)
Nuclei: (1)mesencephalic nucleus: proprioception extends to level of inferior colliculus(2)main sensory nucleus: tactile sensation(3)motor nucleus: motor innervation(4)spinal nucleus: pain + temperature sensation extends to level of 2nd cervical vertebraLocation:in tegmentum of lateral pons, along anterolateral aspect of 4th ventricle*Course:* -through prepontine cistern-exits through porus trigeminus (= opening in dura)-enters Meckel cave with dura mater + leptomeninges forming trigeminal cistern (= CSF-filled subarachnoid space)-forms gasserian ganglion (= trigeminal ganglion) which contains cell bodies of sensory fibers except those for proprioceptionTrifurcation into 3 principal branches: (1)**ophthalmic nerve** (V_1)Course:in lateral wall of cavernous sinusExit:superior orbital fissureSupply:sensory innervation of scalp, forehead, nose, globe ● mediates afferent aspect of corneal reflex(2)**maxillary nerve** (V_2)Course:between lateral dural wall of cavernous sinus + skull baseExit:through foramen rotundum into pterygopalatine fossaSupply:sensory innervation of middle third of face, upper teethMain trunk:infraorbital nerve(3)**mandibular nerve** (V_3)Course:NOT through cavernous sinusExit:through foramen ovale into masticator spaceSupply:(a)sensory innervation of lower third of face, tongue, floor of mouth, jaw(b)motor innervation of muscles of mastication (masseter, temporalis, medial + lateral pterygoid), mylohyoid m., anterior belly of digastric m., tensor tympani m., tensor veli palatini m.



Notes:

FACIAL NERVE (VII)

Nuclei: (1)Motor nucleus: ventrolateral deep in reticular formation of the caudal part of the ponsIntrapontine course: -dorsomedially towards 4th ventricle-curving anterolaterally around upper pole of abducent nucleus (= **geniculum**)-descending anterolaterally through reticular formationInnervation to:stapedius m., stylohyoid m., posterior belly of digastric m., occipitalis m., buccinator, muscles of facial expression, platysma(2)Nucleus solitarius (sensory nucleus):-**nervus intermedius**: sensation from anterior 2/3 of tongue, skin on + adjacent to ear(3)Superior salivatory nucleus (parasympathetic secretomotor innervation)-greater petrosal n.: secretion of lacrimal glands, nasal cavity, paranasal sinuses-chorda tympani: submandibular gland, sublingual glands*Course:* -from lateral aspect of pontomedullary junction-coursing anterolaterally in cerebellopontine angle cistern to internal auditory canal (IAC)-motor root of facial n. in anterosuperior groove of vestibulocochlear n. with nervus intermedius between them*mnemonic:*"seven up"-labyrinthine segment (in fallopian canal) travels anteromedially to **geniculate ganglion**
-turns posteriorly and horizontally along medial wall of mesotympanum (= anterior tympanic segment) below lateral semicircular canal just above the oval window-turns inferiorly at second genu in pyramidal eminence + descends through anterior mastoid(= medial wall of aditus ad antrum) *Exit:*from skull base through stylomastoid foramen*Branches:* (1)**Greater superficial petrosal nerve** (parasympathetic + motor fibers) arises from geniculate ganglion, runs anteromedially, and exits at the facial hiatus on the anterior surface of the temporal bone + passes under Meckel cave near foramen lacerum-forms **vidian nerve** after receiving sympathetic fibers from deep petrosal nerve which surrounds the internal carotid artery(2)**Stapedial nerve** (motor fibers) arises from proximal descending facial n.(3)**Chorda tympani** (sensory + parasympathetic fibers) leaves facial n. about 6 mm above stylomastoid foramen-ascends forward in a bony canal (= posterior canaliculus)-perforates posterior wall of tympanic cavity-crosses medial to handle of the malleolus underneath mucosa of tympanic cavity-reenters bone at medial end of petrotympanic fissure (= posterior canaliculus)-joins the lingual nerve (= branch of V_3) containing sensory fibers from anterior 2/3 of tongue + secretomotor fibers for submandibular and sublingual glands

Notes:

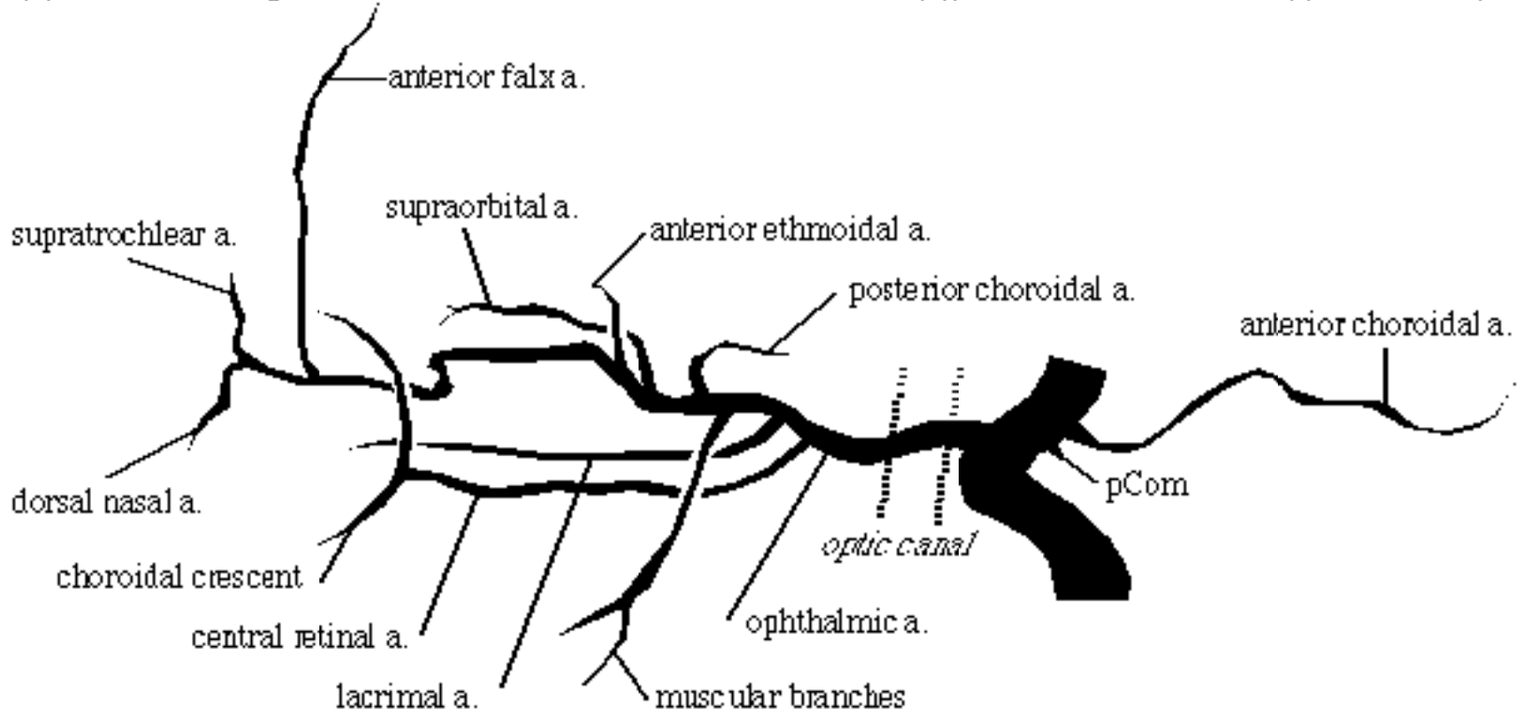
Common Carotid Artery ● 70% of blood flow is delivered to ICA√ shares waveform characteristics of both internal + external carotid arteries√ velocity increases toward the aorta (9 cm/sec for each cm of distance from the carotid bifurcation) ***Carotid bifurcation*** = physiologic stenosis due to inertial forces of blood flow diverting main-flow stream from midvessel to a path along vessel margin at flow divider Location:lateral to upper border of thyroid cartilage; at level of C3-4 intervertebral disc*Branches:*ECA arises anterior + medial to ICA (95%)

Notes:

External Carotid Artery Branches *mnemonic:*"**A**ll **S**ummer **L**ong **E**mily **O**gled **P**eter's **S**porty **I**suzu"**A**scending pharyngeal artery **S**uperior thyroid artery **L**ingual artery **E**xternal maxillary = facial artery **O**ccipital artery **P**osterior auricular artery **S**uperficial temporal artery **I**nternal maxillary artery

Notes:

Internal Carotid Artery A.CERVICAL SEGMENTascends posterior and medial to ECA; enters carotid canal of petrous bone; NO branches B.PETROUS SEGMENTascends briefly, in carotid canal bends anteromedially in a horizontal course (anterior to tympanic cavity + cochlea); exits near petrous apex through posterior portion of foramen lacerum; ascends to juxtasellar location where it pierces dural layer of cavernous sinus *Branches:* 1.**Caroticotympanic a.**: to tympanic cavity, anastomoses with anterior tympanic branch of maxillary a. + stylomastoid a.2.**Pterygoid (vidian) a.**: through pterygoid canal; anastomoses with recurrent branch of greater palatine a.C.CAVERNOUS SEGMENTascends to posterior clinoid process, then turns anteriorly + superomedially through cavernous sinus; exits medial to anterior clinoid process piercing dura *Branches:* 1.**Meningohypophyseal trunk** (a)tentorial branch(b)dorsal meningeal branch(c)inferior hypophysial branch2.**Anterior meningeal a.**: supplies dura of anterior fossa; anastomoses with meningeal branch of posterior ethmoidal a.3.Cavernous rami supply trigeminal ganglion, walls of cavernous + inferior petrosal sinuses D.SUPRACLINOID SEGMENTascends posterior + lateral between oculomotor + optic nerve *Branches: mnemonic:*"OPA"**O**phthalmic a. **P**osterior communicating a. **A**nterior choroidal a. 1.**Ophthalmic a.** exits from ICA medial to anterior clinoid process, travels through optic canal inferolateral to optic nerve(a)recurrent meningeal branch: dura of anterior middle cranial fossa(b)posterior ethmoidal a.: supplies dura of planum sphenoidale(c)anterior ethmoidal a.
2.**Superior hypophysial a.**: optic chiasm, anterior lobe of pituitary3.**Posterior communicating a.** (pCom)4.**Anterior choroidal a.**
5.**Middle + anterior cerebral arteries** (MCA, ACA)



Notes:

Carotid Siphon flow direction:C4 - C1 (a)C4 segment=before origin of ophthalmic a.(b)C3 segment =genu of ICA(c)C2 segment =supraclinoid segment after origin of ophthalmic a.(d)C1 segment =terminal segment of ICA between pCom + ACA

Notes:

Anterior Cerebral Artery (ACA) A. HORIZONTAL PORTION = A 1 SEGMENT = segment between origin and anterior communicating a. (aCom) (a)Inferior branchessupply superior surface of optic nerve + chiasm (b)superior branchespenetrate brain to supply anterior hypothalamus, septum pellucidum, anterior commissure, fornix columns, anterior inferior portion of corpus striatum (largest striatal artery = medial lenticulostriate artery = recurrent **artery of Heubner** for anteroinferior portion of head of caudate, putamen, anterior limb of internal capsule) B. INTERHEMISPHERIC PORTION = A 2 SEGMENT = segment after origin of anterior communicating a. (aCom); ascends in cistern of lamina terminalis *Branches:* 1.**Medial orbitofrontal a.**: along gyrus rectus2.**Frontopolar a.** 3.**Callosomarginal a.**: within cingulus gyrus4.**Pericallosal a.**: over corpus callosum within callosal cistern(a)Superior internal parietal a.: anterior portion of precuneus + convexity of superior parietal lobule(b)Inferior internal parietal a.(c)Posterior pericallosal a.from callosomarginal / pericallosal artery: -Anterior + middle + posterior internal frontal aa.-Paracentral a.: supplies precentral + postcentral gyri*Supply:*anterior 2/3 of medial cerebral surface+ 1 cm of superomedial brain over convexity

Notes:

Middle Cerebral Artery =largest branch of ICA arising lateral to optic chiasm; passes horizontal in lateral direction just ventral to anterior perforated substance to enter sylvian fissure where it divides into 2 / 3 / 4 branches*Branches:*1.**Anterior temporal a.**2.**Ascending frontal a.** (candelabra) / prefrontal a.3.**Precentral a.** = Pre-Rolandic a.4.**Central a.** = Rolandic a.5.**Anterior parietal a.** = Post-Rolandic a.6.**Posterior parietal a.**7.**Angular a.**8.**Middle temporal a.**9.**Posterior temporal a.**10.**Temporo-occipital a.***Supply:*lateral cerebrum, insula, anterior + lateral temporal lobe

Notes:

Posterior Cerebral Artery originates from bifurcation of basilar artery within inter-peduncular cistern (in 15% as a direct continuation of posterior communicating artery); lies above oculomotor nerve and circles midbrain above the tentorium cerebelli *Branches:* 1.Mesencephalic perforating branches: tectum + cerebral peduncles2.Posterior thalamoperforating aa.: midline of thalamus + hypothalamus3.Thalamogeniculate aa.: geniculate bodies + pulvinar4.Posterior medial choroidal a.: circles midbrain parallel to PCA; enters lateral aspect of quadrigeminal cistern; passes lateral and above pineal gland and enters roof of 3rd ventricle; supplies quadrigeminal plate + pineal gland5.Posterior lateral choroidal a.: courses lateral and enters choroidal fissure; anterior branch to temporal horn + posterior branch to choroid plexus of trigone and lateral ventricle + lateral geniculate body6.Cortical branches:(a)Anterior inferior temporal a.(b)Posterior inferior temporal a.(c)Parieto-occipital a.(d)Calcarine a.(e)Posterior pericallosal a.*Supply:*medial + posterior temporal lobe, medial parietal lobe, occipital lobe

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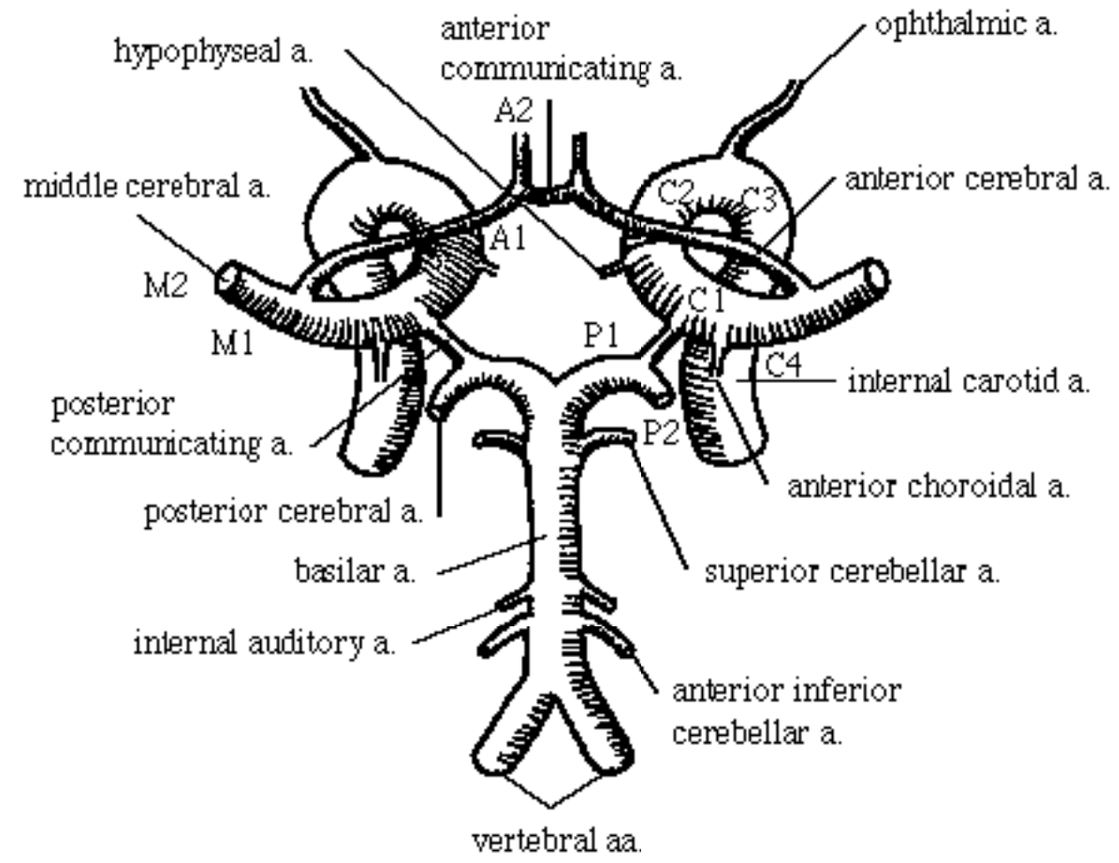
Notes:

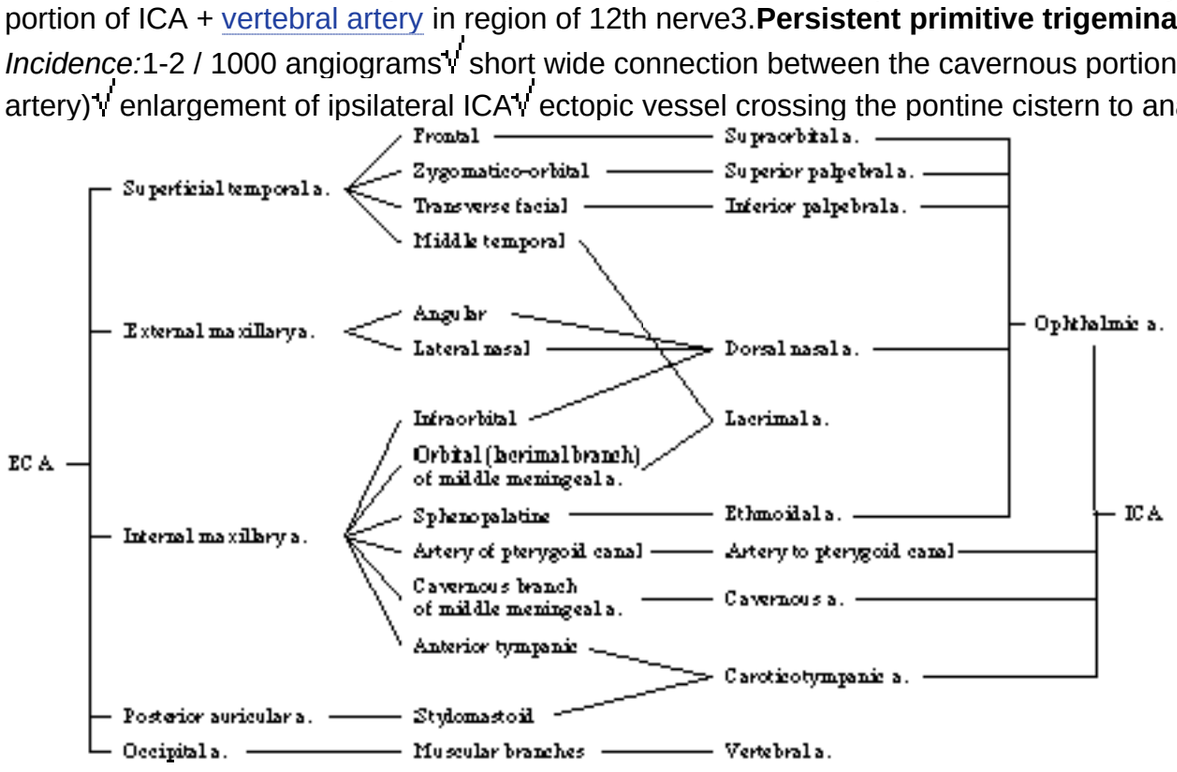
Arterial Anastomoses Of The Brain Anastomoses via the arteries at the base of the brain A.CIRCLE OF WILLIS1.right ICA - right ACA - aCom - left ACA - left ICA2.ICA - pCom - basilar a.3.ICA - anterior choroidal a. - posterior choroidal a. - PCA - basilar a.



CIRCLE OF WILLIS

B.DEVELOPMENTAL ANOMALYthree transient embryonal carotid-basilar anastomoses appearing consecutively in fetal life: 1. **Primitive hypoglossal artery** =arterial connection between the intrapetrosal portion of ICA and proximal portion of basilar artery2. **Primitive acoustic (otic) artery** =arterial connection between cervical portion of ICA + vertebral artery in region of 12th nerve3.**Persistent primitive trigeminal artery**

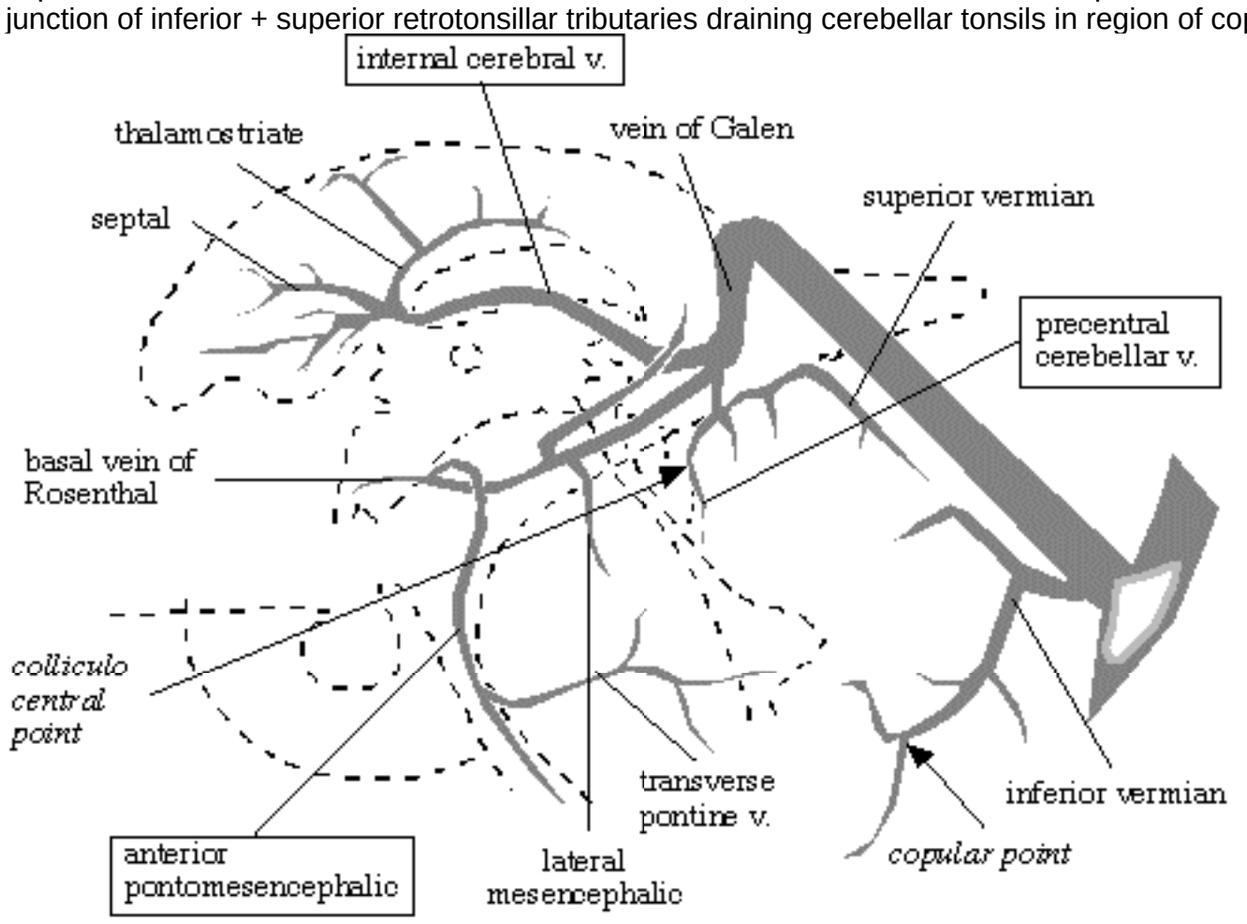
*Incidence:*1-2 / 1000 angiograms√ short wide connection between the cavernous portion of ICA and upper third of basilar artery (beneath posterior communicating artery)√ enlargement of ipsilateral ICA√ ectopic vessel crossing the pontine cistern to anastomose with basilar artery



Anastomoses via surface vessels A.Leptomeningeal anastomoses of the cerebrum:ACA - MCA - PCA B.Leptomeningeal anastomoses of the cerebellum:Superior cerebellar a. - AICA - PICA Rete mirabile ECA - middle meningeal a. / superficial temporal a. - leptomeningeal aa. - ACA / MCA

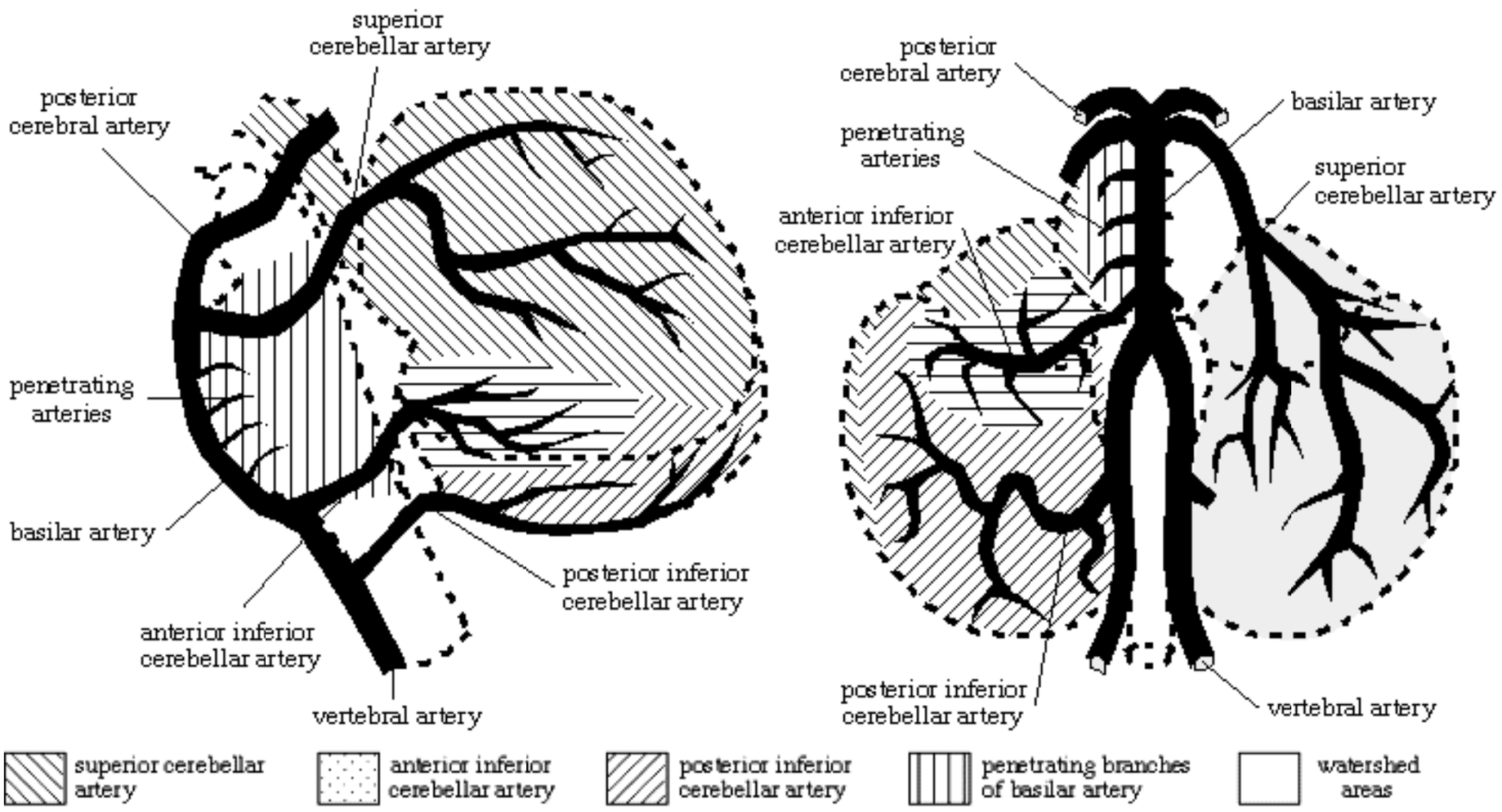
Notes:

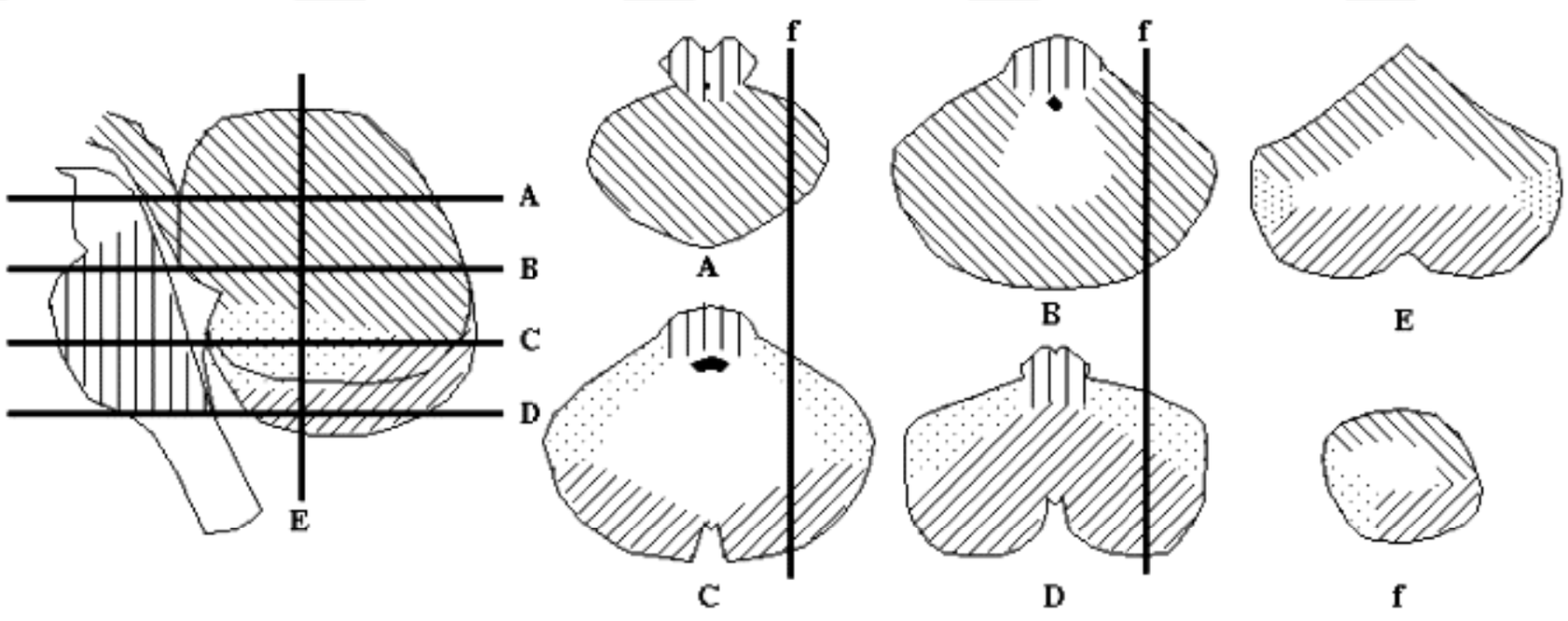
Cerebral Veins Important vascular markers: 1.Pontomesencephalic v. = anterior border of brainstem2.Precentral cerebellar v. = position of tectum◊colliculocentral point = midpoint of Twinings line at knee of precentral cerebellar vein3.Venous angle = acute angle at junction of thalamostriate with internal cerebral v. = posterior aspect of foramen of Monro4.Internal cerebral vv. = demarcate caudad border of splenium of corpus callosum superiorly + pineal gland inferiorly5.Copular point = junction of inferior + superior retrotonsillar tributaries draining cerebellar tonsils in region of copular pyramids of vermis

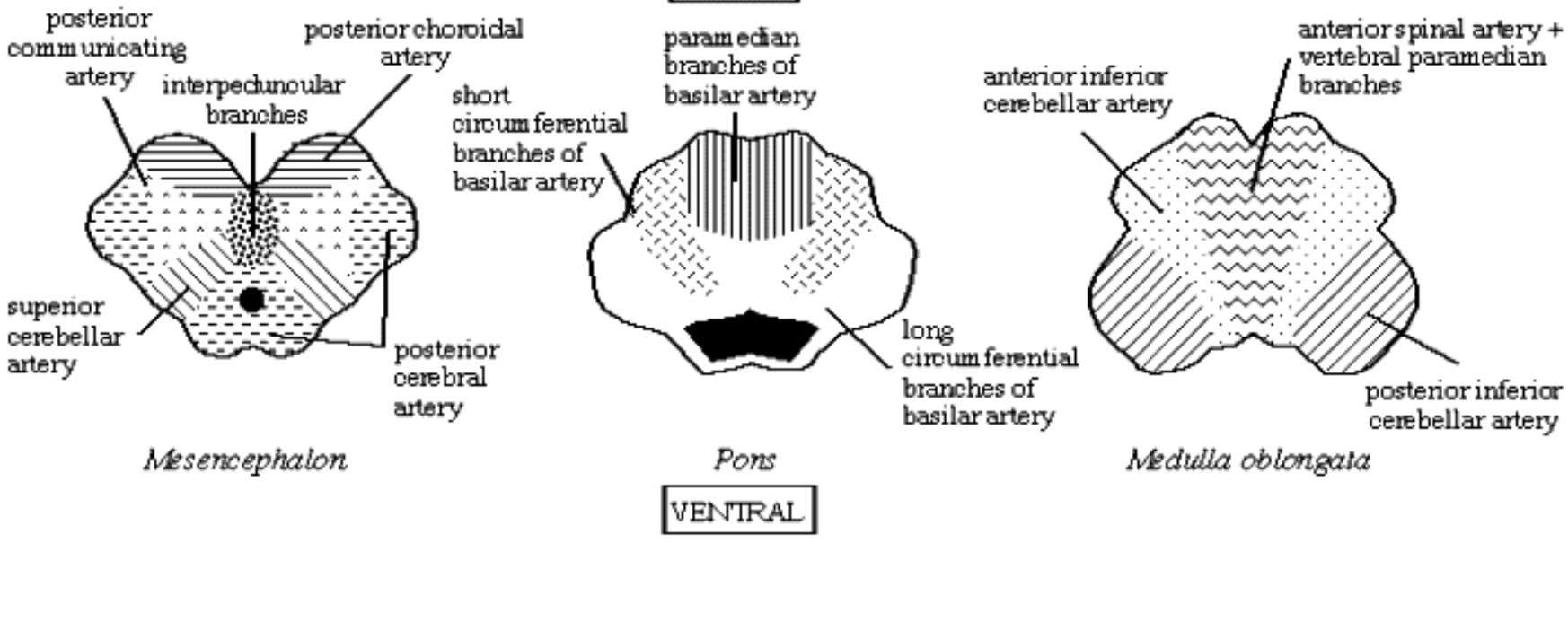


Notes:

Vertebral Artery originates from subclavian a. proximal to thyrocervical trunk; left vertebral a. usually greater than right cerebral a.; left vertebral a. may originate directly from aorta (5%) A.PREVERTEBRAL SEGMENTascends posterosuperiorly between longus colli + anterior scalene muscle; enters transverse foramina at C6 *Branches:*muscular branchesB.CERVICAL SEGMENTascends through transverse foramina in close proximity to uncinate processes *Branches:*1.**Anterior meningeal a.**
C.ATLANTIC SEGMENTexits transverse foramen of atlas; passes posteriorly in a groove on superior surface of posterior arch of atlas; pierces atlanto-occipital membrane + dura mater to enter cranial cavity *Branches:*1.**Posterior meningeal branch** to posterior falx + tentoriumD.INTRACRANIAL SEGMENTascends anteriorly + laterally around medulla to reach midline at pontomedullary junction; anastomoses with contralateral side to form basilar artery at clivus *Branches:* 1.**Anterior + posterior spinal a.** 2.**Posterior inferior cerebellar a.** (PICA)3.**Anterior inferior cerebellar a.** (AICA)4.**Internal auditory a.**
5.**Superior cerebellar a.**
6.**Posterior cerebral a.** (PCA)7.Medullary + pontine perforating branches may terminate in common AICA-PICA trunk





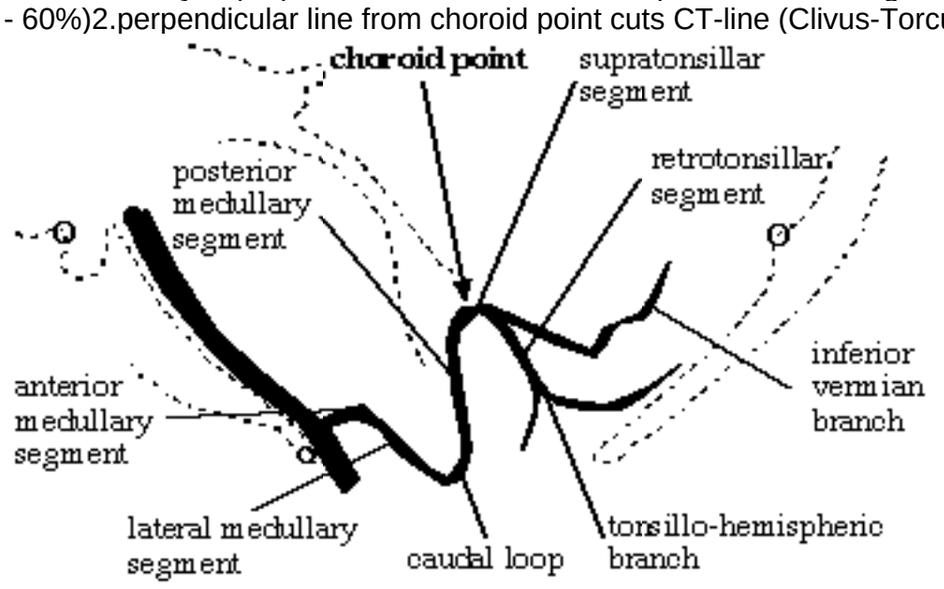


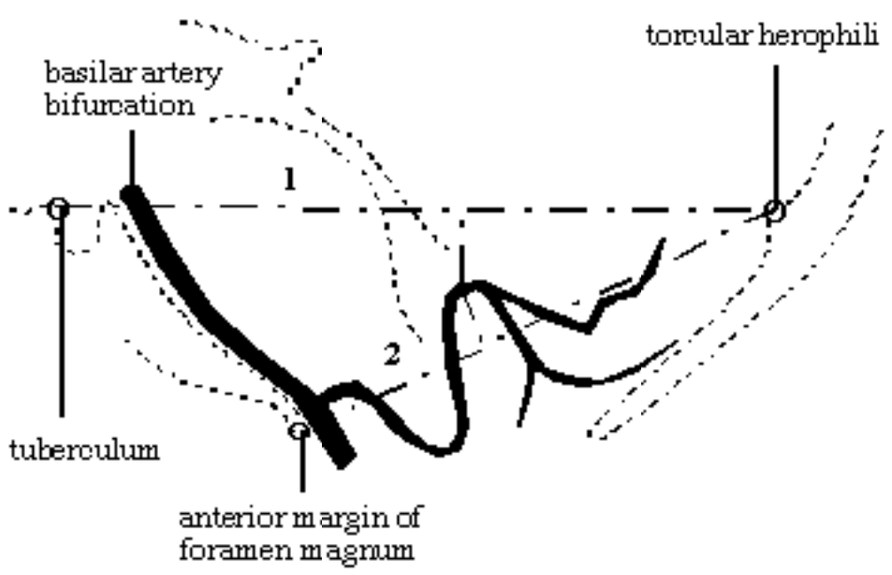
Notes:

Anterior Inferior Cerebellar Artery =AICA = first branch of basilar artery*Supply:* lateroinferior part of pons, middle cerebellar peduncle, floccular region, anterior petrosal surface of cerebellar hemisphere ◊Quite variable course + vascular supply with reciprocal relation between vascular territories of AICA + PICA!

Notes:

Posterior Inferior Cerebellar Artery =PICA = last and largest branch of vertebral artery*Parts:* 1.Premedullar segment = caudal loop around medulla, may descend below level of foramen magnum2.Retromedullar segment = ascending portion up to the level of 4th ventricle and tonsils3.Supratonsillar segment = the most cranial point is the choroidal pointP1 segment=horizontal segment between origin of PICA + pComP2 segment=segment downstream from pCom take-off *Variations:*commonly asymmetric; hypoplastic / absent in 20% [vascular supply then provided by anterior inferior cerebellar artery (AICA)]*Supply:* inferoposterior surface of cerebellar hemisphere adjacent to occipital bone, ipsilateral part of inferior vermis, inferior portion of deep white matter only Orthotopic **choroid point** established by: 1.perpendicular line from choroid point onto Twinings line = TTT-line (Twinings Tuberculum-Torcular line) bisects TTT-line (length of anterior portion 52 - 60%)2.perpendicular line from choroid point cuts CT-line (Clivus-Torcular line) <1 mm anterior / <3 mm posterior to junction of anterior and middle thirds of CT-line





Notes:

Superior Cerebellar Artery =SCA = last but one branch of basilar artery*Supply:* superior aspect of cerebellar hemisphere (tentorial surface), ipsilateral superior vermis, largest part of deep white matter including dentate nucleus, pons

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Notes:

Pyogenic Abscess =focal area of necrosis beginning in area of cerebritis with formation of surrounding membrane*Cause:* 1.Extension from paranasal sinus infection (41%) / mastoiditis / otitis media (5%) / facial soft-tissue infection / dental abscess2.Generalized septicemia (32%):(a)lung (most common): bronchiectasis, empyema, lung abscess, bronchopleural fistula, pneumonia(b)heart (less common): CHD with R-L shunt, AVM, bacterial endocarditis(c)osteomyelitis3.Penetrating trauma or surgery4.Cryptogenic (25%)*Predisposed:*diabetes mellitus, patients on steroids / immunosuppressive drugs, congenital / acquired immunologic deficiency*Organism:*Anaerobic streptococcus (most common), Bacteroides, Staphylococcus; in 20% multiple organisms; in 25% sterile contents*Pathophysiology:* Stage I:vascular congestion, petechial hemorrhage, edemaStage II:cerebral softening + necrosisStage III:(after 2-3 weeks) liquefaction, cavitation + capsule consisting of inner layer of granulation tissue, a middle collagenous layer and an outer astroglial layer; edema outside abscess capsuleLocation:typically at corticomedullary junction; frontal + temporal lobes; supratentorial : infratentorial = 2:1NCCT: √ zone of low density with mass effect (92%)√ slightly increased rim density (4%), development of collagen layer takes 10-14 days√ gas within lesion (4%) is diagnostic of gas-forming organismCECT: √ ring enhancement (90%) with peripheral zone of edema√ homogeneous enhancement in lesions <0.5 cm√ edema + contrast enhancement suppressed by steroids√ smooth regular 1-3 mm thick wall with relative thinning of medial wall (secondary to poorer blood supply of white matter)√ multiloculation + subjacent daughter abscess in white matterMR: (most sensitive modality)√ centrally increased / variable intensity with hypointense rim on T2WI√ outside border of increased signal intensity on T2WI (edema)*Cx:*(1)Development of daughter abscesses toward white matter(2)Rupture into ventricular system / subarachnoid space (thinner abscess capsule formation on medial wall of abscess related to fewer blood vessels) producing ventriculitis ± meningitis*Dx helpful features:* -multiple lesions at gray-white matter border-clinical history of altered immune status-R-to-L shunt: eg, pulmonary AV fistula-foreign travel-high-risk behavior: eg, IV drug abuse*DDx:*primary / metastatic neoplasm, subacute infarction, resolving hematoma

Notes:

Granulomatous Abscess 1.Tuberculoma2.Sarcoid abscess3.Fungal abscess: eg, Cryptococcus*Predisposed:* immunocompromised patients√ enhancement of leptomeningeal surface√ nodular / ring-enhancing parenchymal lesion*Cx:*Communicating hydrocephalus (secondary to thick exudate blocking basal cisterns)

Notes:

ACRANIA
=EXENCEPHALY=developmental anomaly characterized by partial / complete absence of membranous neurocranium + complete but abnormal development of brain tissue*Incidence:*25 cases reported*Cause:*impaired migration of mesenchyme to its normal location under the calvarial ectoderm resulting in failure for development of dura mater + skull + musculature*Time:*develops after closure of anterior neuropore during 4th week*May be associated with:* cleft lip, bilateral absence of orbital floors, metatarsus varus, talipes, cervicothoracic spina bifida ● ± elevation of maternal serum AFP√ absence of calvarium√ normal ossification of chondrocranium (face, skull base)√ hemispheres surrounded by thin membrane*Prognosis:*uniformly lethal; progression to anencephaly (brain destruction secondary to exposure to amniotic fluid + mechanical trauma)*DDx:*encephalocele, anencephaly, osteogenesis imperfecta, hypophosphatasia

Notes:

ADRENOLEUKODYSTROPHY
=BRONZED SCLEROSING ENCEPHALOMYELITIS=inherited metabolic disorder characterized by progressive demyelination of cerebral white matter + adrenal insufficiency*Etiology:*defective peroxisomal fatty acid oxidation due to impaired function of lignoceryl-coenzyme A ligase with accumulation of saturated very long chain fatty acids (cholesterol esters) in white matter + adrenal cortex + testes*Dx:*assay of plasma, red cells, cultured skin fibroblasts for the presence of increased amounts of very long chain fatty acids*Mode of inheritance:* (a)X-linked recessive in boys (common)(b)autosomal recessive in neonates (uncommon)*Histo:*PAS cytoplasmic inclusions in brain, adrenals, other tissues*Age:*3-10 years (X-linked recessive) ● deteriorating vision (27%), loss of hearing (50%) ● ataxia ● optic disk pallor ● adrenal gland insufficiency (abnormal increased pigmentation, elevated ACTH levels) ● altered behavior, attention disorder, mental deterioration, deathLocation:disease process usually starts in central occipital white matter, advances anteriorly through internal + external capsules + centrum semiovale, centripetal progression to involve subcortical white matter, interhemispheric spread via corpus callosum particularly splenium, involvement of optic radiation ± auditory system ± pyramidal tractCT: √ large symmetric low-density lesions in occipitoparietotemporal white matter (80%) advancing toward frontal lobes + cerebellum√ thin curvilinear / serrated enhancing rims near edges of lesion√ initial frontal lobe involvement (12%)√ calcifications within hypodense areas (7%)√ cerebral atrophy in late stage (progressive loss of cortical neurons)MR: √ hypointensity on T1WI in affected areas (hypointense atrophic splenium of corpus callosum)√ hyperintense bilateral confluent areas on T2WI*Prognosis:*usually fatal within several years after onset of symptoms
Adrenomyeloneuropathy =clinically milder form with later age of onset ● symptoms of spinal cord demyelination + peripheral neuropathy

Notes:

AGENESIS OF CORPUS CALLOSUM
=COMPLETE DYSGENESIS OF CORPUS CALLOSUM=failure of formation of corpus callosum originating from the lamina terminalis at 7-13 weeks from where a phalanx of callosal tissue extends backward arching over the diencephalon; usually developed by 20 weeks*Incidence:*0.7-5.3%*Cause:*congenital, acquired (infarction of ACA)*Histo:*axons from cerebral hemispheres that would normally cross continue along medial walls of lateral ventricles as longitudinal callosal bundles of Probst that terminate randomly in occipital + temporal lobes
Associated with: (a)CNS anomalies (85%):1.Dandy-Walker cyst (11%)2.Interhemispheric arachnoid cyst may be continuous with 3rd and lateral ventricles3.Hydrocephalus (30%)4.Midline intracerebral lipoma of corpus callosum often surrounded with ring of calcium (10%)5.Arnold-Chiari II malformation (7%)6.Midline encephalocele7.Porencephaly8.Holoprosencephaly9.Hypertelorism median cleft syndrome10.Polymicrogyria, gray-matter heterotopia(b)Cardiovascular, gastrointestinal, genitourinary anomalies (62%)(c)Abnormal karyotype (trisomy 13, 15, 18)
● normal brain function in isolated agenesis ● intellectual impairment; seizures√ absence of septum pellucidum + corpus callosum + cavum septi pellucidi√ longitudinal bundles of Probst create crescentic lateral ventricles√ colpocephaly (= dilatation of trigones + occipital horns + posterior temporal horns in the absence of splenium√ "bat-wing" appearance of lateral ventricles (= wide separation of lateral ventricles with straight parallel parasagittal orientation with absent callosal body)√ laterally convex frontal horns in case of absent genu of corpus callosum√ "high-riding third ventricle" = upward displacement of widened 3rd ventricle often to level of bodies of lateral ventricle√ anterior interhemispheric fissure adjoins elevated 3rd ventricle ± communication (PATHOGNOMONIC)√ "interhemispheric cyst" = interhemispheric CSF collection as an upward extension of 3rd ventricle√ enlarged foramina of Monro√ "sunburst gyral pattern" = dysgenesis of cingulate gyrus with characteristic radial orientation of cerebral sulci from the roof of the 3rd ventricle (on sagittal images)√ failure of normal convergence of calcarine + parieto-occipital sulci√ persistent eversion of cingulate gyrus (rotated inferiorly + laterally) with absence on midsagittal images√ incomplete formation of Ammon's horn in the hippocampus
OB-US (>22 weeks GA): √ absence of septum pellucidum√ "teardrop" ventriculomegaly = disproportionate enlargement of occipital horns = colpocephaly√ dilated + elevated 3rd ventricle√ radial array pattern of medial cerebral sulciAngio: √ wandering straight posterior course of pericallosal arteries (lateral view)√ wide separation of pericallosal arteries secondary to intervening 3rd ventricle (anterior view)√ separation of internal cerebral veins√ loss of U-shape in vein of Galen
DDx:(1)Prominent cavum septi pellucidi + cavum vergae (should not be mistaken for 3rd ventricle)(2)Arachnoid cyst in midline (suprasellar, collicular plate) raising and deforming the 3rd ventricle and causing hydrocephalus

Partial Agenesis of Corpus Callosum

Notes:

Partial Agenesis of Corpus Callosum =milder form of callosal dysgenesis (best seen on MR)depending on time of arrested growth (anteroposterior development of genu + body + splenium, however, rostrum forming last)(a)genu only(b)genu + part of the body(c)genu + entire body(d)genu + body + splenium (without rostrum)

Notes:

AIDS

=DNA retrovirus infection attacking monocytes + macrophages which leads to deficient cell-mediated immunity*Incidence:*1% of population in United States is HIV-seropositive; 187,000 new cases in 1991*Histo:*formation of microglial nodules instead of granulomas in 75-80% of autopsied brains ● neurologic symptoms as initial complaint in 10%, ultimately afflict up to 40-60%: headache, memory loss, confusion, dementia, focal deficit from mass lesion◊Any male with neurologic symptoms between age 20 and 50 has AIDS until proven otherwise◊Unusual presentations are clues to HIV infection:pan-sinusitis, mastoiditis, parotid cysts, cervical adenopathy, hypointense spine

DIFFUSE CHANGES: (1)HIV / CMV encephalopathy (most common complication)both viruses occur always in combination ● dementia in up to 60% during course of disease ● cognitive dysfunction in up to 90%√ patchy white matter lesions (= subacute leukoencephalitis) in 31%

FOCAL CHANGES: (1)Toxoplasmosis (50-70%)(2)Primary CNS lymphoma (20-30%)*Prevalence:*in 75% at autopsy◊Initial manifestation in 0.6% of AIDS patients◊2% of AIDS patients develop primary CNS lymphoma at some point during their illness(3)Progressive multifocal leukoencephalopathy (10-20%)(4)Fungal, granulomatous, viral, bacterial infection(a)CryptococcosisLocation:extension along Virchow-Robin spaces√ hydrocephalus + cortical / central atrophy (with inadequate immune response)√ enhancing granulomatous meningitis (with sufficient immune response)√ bilateral nonenhancing hyperintense abnormalities in lenticulostriate region(= gelatinous pseudocyst) on T2WI(b)Other opportunistic CNS infections: tuberculosis, neurosyphilis◊With multiple CNS lesions toxoplasmic encephalitis is the more likely diagnosis!◊With a single CNS lesion the probability of lymphoma is at least equal to toxoplasmosis!*Rx:*azidothymidine (AZT)

Notes:

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ALEXANDER DISEASE
=FIBRINOID LEUKODYSTROPHY*Age:*as early as first few weeks of life ● macrocephaly ● failure to attain developmental milestones ● progressive spastic quadriparesis ● intellectual failureLocation:frontal white matter gradually extending posteriorly into parietal region + internal capsuleCT: √ low-density white matter lesion√ contrast enhancement near tip of frontal hornMR: √ prolonged T1 + T2 relaxation times*Prognosis:*death in infancy / early childhood

Notes:

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ALZHEIMER DISEASE

most common of diffuse gray matter diseases with large loss of cells from cerebral cortex + other areas ● slowly progressing memory loss, dementia√ "cracked walnut" appearance = symmetrically enlarged sulci in high-convexity area√ focal atrophic change in medial temporal lobe√ smooth periventricular halo of hyperintensity (50%)

Notes:

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ANENCEPHALY
=lethal anomaly with failure of closure of the rostral end of the neural tube by 5.6 weeks MA◊Associated with highest AF-AFP and MS-AFP values; >90% will be detected with MS-AFP ≥2.5 MoM*Incidence:*1:1,000 births (3.5:1,000 in South Wales); M:F = 1:4; most common congenital defect of CNS; 50% of all neural tube defects*Recurrence rate:*3-4%*Etiology:*multifactorial (genetic + environmental)*Path:*absence of cerebral hemispheres + cranial vault; partial / complete absence of diencephalic + mesencephalic structures; hypophysis + rhombencephalic structures usually preserved*Risk factors:*family history of neural tube defect; twin pregnancy*Associated anomalies:* spinal dysraphism (17-50%), cleft lip / palate (2%), clubfoot (2%), umbilical hernia, amniotic band syndrome √ absence of bony calvarium cephalad to orbits√ ± cranial soft-tissue mass (= angiomatous stroma)√ bulging froglike eyes√ short neck√ polyhydramnios (40-50%) after 26 weeks GA (due to failure of normal fetal swallowing) / oligohydramnios*Dx:*in 100% >14 weeks GA*Prognosis:*uniformly fatal within hours to days of life; in 53% premature birth; in 68% stillbirth*DDx:*acrania, encephalocele, amniotic band syndrome

Notes:

ANEURYSM OF CNS
Etiology: (a)congenital (97%) = "berry aneurysm" in 2% of population (in 20% multiple); associated with aortic coarctation + adult polycystic kidney disease(b)infectious (3%) = mycotic aneurysm(c)arteriosclerotic: fusiform shape(d)traumatic(e)neoplastic(f)fibromuscular disease(g)collagen vascular disease*Risk factors:* (1)family history for aneurysms in 1st- / 2nd-degree relatives(2)female gender(3)age >50 years(4)cigarette smoking(5)oral contraceptives / pregnancy(6)Marfan syndrome, pseudoxanthoma elasticum, Ehlers-Danlos syndrome(7)polycystic kidney disease(8)asymmetry of circle of Willis(9)cerebral arteriovenous malformation*Pathogenesis:*arterial wall deficient in tunica media + external elastic lamina (natural occurrence with advancing age)
Location of aneurysm: A.by autopsy:(a)circle of Willis (85%):MCA bifurcation (25%), aCom (25%), pCom (18%), distal ACA (5%), ICA at bifurcation (4%), ophthalmic a. (4%), anterior choroidal a. (4%) (b)posterior fossa (15%)basilar bifurcation (7%), basilar trunk (3%), vertebral-PICA (3%), PCA (2%) B.by angiography (= symptomatic aneurysms):pCom (38%) > aCom (36%) > MCA bifurcation (21%) > ICA bifurcation > tip of basilar artery (2.8%) C.by risk of bleeding: 1-2% per yearaCom (70% bleed), pCom (2nd highest risk) ◊Aneurysms at bifurcations / branching points are at greatest risk for rupture!
MULTIPLE ANEURYSMS *Cause:*congenital in 20-30%, mycotic in 22%*mnemonic:*"FECAL P"**F**ibromuscular dysplasia **E**hlers-Danlos syndrome **C**oarctation **A**rteriovenous malformation **L**upus erythematosus **P**olycystic kidney disease (adult) ◊35% of patients with one MCA aneurysm have one on the contralateral side (= mirror image aneurysms)!◊simultaneous aneurysm + AVM in 4-15%

CECT: detection rate of aneurysms at pCom (40%), aCom / MCA, basilar artery (80%) Angio (all 4 cerebral vessels): √ contrast outpouching√ <2 mm infundibuli typically occur at pCom / anterior choroidal a. origin√ mass effect in thrombosed aneurysm◊2nd arteriogram within 1-2 weeks detects aneurysm in 10-20% following negative 1st angiogram!
Prognosis: (1)Death in 10% within 24 hours from concomitant intracerebral hemorrhage, extensive brain herniation, massive infarcts + hemorrhage within brainstem; 45% mortality within 30 days (25% prior to admission)(2)Complete recovery in 58% of survivors(3)Cerebral ischemia + infarction(4)Rebleeding rate: 12-20% within 2 weeks, 11-22% within 30 days, up to 50% within 6 months (increased mortality); thereafter 4% risk/year*Surgical mortality rate:*50% for ruptured, 1-3% for unruptured aneurysms*Cx:*subdural hematoma

Ruptured Berry Aneurysm Giant Aneurysm Mycotic Aneurysm Supraclinoid Carotid Aneurysm Cavernous Sinus Aneurysm

Notes:

Ruptured Berry Aneurysm *Incidence:*28,000 cases/year = 10 cases/10,000 people/year*Age:*50-60 years of age; M:F = 1:2*Rupture size:*5-15 mm ● "worst headache of ones life" ● neck stiffness, nausea, vomiting ● sudden loss of consciousness (in up to 45%) ● history of warning leak / sentinel hemorrhage hours to days earlier *Clues for which aneurysm is bleeding:* (a)the largest aneurysm (87%)(b)anterior communicating artery (70%)(c)contralateral side of all visualized aneurysms (60%), nonvisualization due to spasm*mnemonic:*"BISH"**B**iggest **I**rregular contour **S**pasm (adjacent) **H**ematoma location Location of blood suggesting accurately in 70% the site of the ruptured aneurysm: (a)according to location of subarachnoid hemorrhage:1.Anterior chiasmatic cistern:aCom2.Septum pellucidum:aCom3.Intraventricular:aCom, ICA, MCA4.Sylvian fissure:MCA, ICA, pCom5.Anterior pericallosal cistern:ACA, aCom6.Symmetric distribution insubarachnoid space:ACA + basilar a.(b)according to location of cerebral hematoma:1.inferomedial frontal lobe:aCom2.temporal lobe:MCA3.corpus callosum:pericallosal artery(c)intraventricular hemorrhagefrom aneurysms at aCom, MCA, pericallosal artery (CAVE: blood may have entered in retrograde manner from subarachnoid location)

Notes:

Giant Aneurysm =aneurysm larger than 2.5 cm in diameter, usually presenting with intracranial mass effect*Incidence:*25% of all aneurysms*Age:*no age predilection; M:F = 2:1Location:(arise from arteries at the base of the brain)(a)middle fossa: cavernous segment of ICA (43%), supraclinoid segment of ICA, terminal bifurcation of ICA, middle cerebral artery(b)posterior fossa: at tip of basilar artery, AICA, vertebral arterySkull film: √ predominantly peripheral curvilinear calcification (22%)√ bone erosion (44%)√ pressure changes on sella turcica (18%)CECT: √ "target sign" = centrally opacified vessel lumen + ring of thrombus + enhanced fibrous outer wall√ simple ring-blush (75%) of fibrous outer wall with total thrombosis√ little / no surrounding edemaMR: √ mixed signal intensity (combination of subacute + chronic hemorrhage, calcification)*Cx:*subarachnoid hemorrhage in <30%

Notes:

Mycotic Aneurysm =3% of all intracranial aneurysms, multiple in 20%*Source:*subacute bacterial endocarditis (65%), acute bacterial endocarditis (9%), meningitis (9%), septic thrombophlebitis (9%), myxomaLocation:peripheral to first bifurcation of major vessel (64%); often located near surface of brain especially over convexities(a)suprasellar cistern = circle of Willis(b)inferolateral sylvian fissure = middle cerebral artery trifurcation(c)genu of corpus callosum = origin of callosomarginal artery(d)bottom of 3rd ventricle = pericallosal a.NCCT: √ aneurysm rarely visualized; indirect evidence from focal hematoma secondary to rupture√ zone of increased density / calcification√ increased density in subarachnoid, intraventricular, intracerebral spaces (extravasated blood)√ focal / diffuse lucency of brain (edema / infarction / vasospasm)CECT: √ intense homogeneous enhancement within round / oval mass contiguous to vessels√ incomplete opacification with mural thrombus*Cx:*develop recurrent bleeding more frequently than congenital aneurysms

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Notes:

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Supraclinoid Carotid Aneurysm =38% of intracranial aneurysmsSite:(a)at origin of pCom (65%)(b)at bifurcation of internal carotid artery (23%)(c)at origin of ophthalmic artery (12%) medial to anterior clinoid process; most likely to become giant aneurysm*Presentation:*bitemporal hemianopia (extrinsic compression on chiasm)" calcification is rare (frequent in atherosclerotic cavernous sinus aneurysm)

Notes:

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Cavernous Sinus Aneurysm *Age:*20-70 years, peak 5th-6th decade; F >> M*Cause:*sinus thrombophlebitis ● progressive visual impairment ● cavernous sinus syndrome: trigeminal nerve pain, oculomotor nerve paralysisSite:extradural portion of cavernous sinus ICA√ undercutting of anterior clinoid process√ erosion of lateral half of sella√ erosion of posterior clinoid process√ invasion of middle cranial fossa√ enlargement of superior orbital fissure√ erosion of tip of petrous pyramid√ rimlike calcification (33%)√ displacement of thin bony margins without sclerosis*Rx:*often inoperable; balloon embolization ± parent artery occlusion

Notes:

AQUEDUCTAL STENOSIS

=focal reduction in size of aqueduct at level of superior colliculi / intercollicular sulcus (normal range of 0.2-1.8 mm^2)*Embryology:* aqueduct develops about the 6th week of gestation + decreases in size until birth due to growth pressure from adjacent mesencephalic structures *Incidence:*0.5-1:1,000 births; most frequent cause of congenital hydrocephalus (20-43%); recurrence rate in siblings of 1-4.5%;M:F = 2:1*Etiology:* (a)postinflammatory (50%): secondary to perinatal infection (toxoplasmosis, CMV, syphilis, mumps, influenza virus) or intracranial hemorrhage=destruction of ependymal lining of aqueduct with adjacent marked fibrillary gliosis(b)developmental: aqueductal forking (= marked branching of aqueduct into channels) / narrowing / transverse septum (X-linked recessive inheritance in 25% of males)(c)neoplastic (extremely rare): pinealoma, meningioma, tectal astrocytoma (may be missed on routine CT scans, easily differentiated by MR)*May be associated with:* other congenital anomalies (16%): thumb deformities enlargement of lateral + 3rd ventricles with normal-sized 4th ventricle (4th ventricle may be normal with communicating hydrocephalus) *Prognosis:*11-30% mortality

Notes:

ARACHNOID CYST
=CSF-containing intra-arachnoid cyst without ventricular communication / brain maldevelopment*Incidence:*1% of all intracranial masses*Origin:* (1)congenital: arising from clefts / duplication / "splitting" of arachnoid membrane with expansion by CSF due to secretory activity of arachnoid cells= **true arachnoid cyst**(2)acquired: following surgery / trauma / subarachnoid hemorrhage / infection in neonatal period / associated with extra-axial neoplasm = loculation of CSF surrounded by arachnoidal scarring with expansion by osmotic filtration / ball-valve mechanism = **leptomeningeal cyst** = **secondary arachnoid cyst** = **acquired arachnoid cyst***Histo:*cyst filled with clear fluid, thin wall composed of cleaved arachnoid membrane lined by ependymal / meningothelial cells*Age:*presentation at any time during life ● often asymptomatic ● symptomatic due to mass effect, hydrocephalus, seizures, headaches, hemiparesis, intracranial hypertension, craniomegaly, developmental delay, visual loss, precocious puberty, bobble-head doll syndromeLocation: (arise in CSF cisterns between brain + dura) (a)floor of middle fossa near tip of temporal lobe (sylvian fissure) in 50%(b)suprasellar / chiasmatic cistern (may produce endocrinopathy) in 10%(c)posterior fossa (1/3): cerebellopontine angle (11%), quadrigeminal plate cistern (10%), in relationship to vermis (9%), prepontine / interpeduncular cistern (3%)(d)interhemispheric fissure, cerebral convexity, anterior infratentorial midline√ forward bowing of anterior wall of cranial fossa + elevation of sphenoid ridge√ extra-axial unilocular thin-walled CSF-density cyst with well-defined smooth angular margins√ compression of subarachnoid space + subjacent brain (minimal mass effect)√ may erode inner table of calvarium√ NO enhancement (intrathecal contrast penetrates into cyst on delayed scans)√ NO calcificationsMR (best modality): √ well-circumscribed lesion with same uniform signal intensity as CSF ± mass effect*Cx:*(1)hydrocephalus (30-60%)(2)concurrent subdural / intracystic hemorrhage*Prognosis:*favorable if removed before onset of irreversible brain damage *Rx:*fenestration / cyst-peritoneal shunting*CT-DDx:* epidermoid cyst, dermoid, subdural hygroma, infarction, porencephaly *US-DDx:* choroid plexus cyst, porencephalic cyst (communicates with ventricle), cystic tumor (solid components), midline cyst associated with agenesis of corpus callosum, dorsal cyst associated with holoprosencephaly, Dandy-Walker cyst (extension of 4th ventricle, developmental delay), vein of Galen aneurysm

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ARTERIOVENOUS FISTULA
=abnormal communication between artery + vein resulting in tremendous amount of flow due to high pressure gradient; leading to enlargement + elongation of draining veins *Cause:* (1)Vessel laceration (delay between trauma + clinical manifestation due to delayed lysis of hematoma surrounding arterial laceration)(2)Angiodysplasia: fibromuscular disease, neurofibromatosis, Ehlers-Danlos syndrome(3)Congenital fistula ● pulsatile mass + thrill / bruit ● ± neurologic symptoms / deficit (due to arterial steal)Location: (a)carotid-cavernous sinus fistula (most common)(b)vertebral artery fistula(c)external carotid fistula (rare)

Notes:

ARTERIOVENOUS MALFORMATION
=congenital abnormality consisting of a nidus of abnormal dilated tortuous arteries + veins with racemose tangle of closely packed pathologic vessels resulting in shunting of blood from arterial to venous side without intermediary capillary bed*Prevalence:*most common vascular lesion*Histo:*affected arteries have thin walls (no elastica, small amount of muscularis); intervening gliotic brain parenchyma between vessels*Age:*80% by end of 4th decade; 20% <20 years of age ● headaches, seizures (nonfocal in 40%), mental deterioration ● progressive hemispheric neurologic deficit (50%) ● ictus from acute intracranial hemorrhage (50%)Location: (a)supratentorial (90%): parietal > frontal > temporal lobe > paraventricular > intraventricular region > occipital lobe(b)infratentorial (10%)Vascular supply: (a)pial branches of ICA in 73% of supratentorial location, in 50% of posterior fossa location(b)dural branches of ECA in 27% with infratentorial lesions√ NO mass effectSkull film: √ speckled / ringlike calcifications (15-30%)√ thinning / thickening of skull at contact area with AVM√ prominent vascular grooves on inner table of skull (dilated feeding arteries + draining veins) in 27%NCCT: √ irregular lesion with large feeding arteries + draining veins√ mixed density (60%): dense large vessels + hemorrhage + calcifications√ isodense lesion (15%): may be recognizable by mass effect√ low density (15%): brain atrophy due to ischemia√ not visualized (10%)CECT: √ serpiginous dense enhancement in 80% (tortuous dilated vessels)√ No enhancement in thrombosed AVM√ No avascular spaces within AVM√ lack of mass effect / edema (unless thrombosed / bleeding)√ rapid shunting√ thickened arachnoid covering√ adjacent atrophic brainMR: √ flow void (imaging with GRASS gradient echo + long TR sequences)Angio: √ grossly dilated efferent + afferent vessels with a racemose tangle ("bag of worms")√ arteriovenous shunting into at least one early draining vein√ negative angiogram (compression by hematoma / thrombosis)
Cx:(1)Hemorrhage (common): bleeding on venous side due to increased pressure / ruptured aneurysm (5%)(2)Infarction*Prognosis:*10% mortality; 30% morbidity; 2-3% yearly chance of bleeding increasing to 6% in year following 1st bleed + 25% in year following 2nd bleed

Wyburn-Mason Syndrome

Notes:

Wyburn-Mason Syndrome =telangiectasias of skin + retinal cirsoid aneurysm + AVM involving entire optic tract (optic nerve, thalamus, geniculate bodies, calcarine cortex);*May be associated with:*AVMs of posterior fossa, neck, mandible / maxilla presenting in childhood

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Notes:

ASTROCYTOMA
*Incidence:*70-75% of all primary intracranial tumors; most common brain tumor in children (40-50% of all primary pediatric intracranial neoplasms)Location: cerebral hemisphere (lobar), thalamus, pons, midbrain, may spread across corpus callosum (incidence of occurrence proportional to amount of white matter); no particular lobar distribution; (a)in adults: central white matter of cerebrum (15-30% of all gliomas)(b)in children: cerebellum (40%) + brainstem (20%), supratentorial (30%)
Well-differentiated = Low-grade Astrocytoma *Incidence:*9% of all primary intracranial tumors*Age:*20- 40 years; M > F*Path:*benign nonmetastasizing; poorly defined borders with infiltration of white matter + basal ganglia + cortex; NO significant tumor vascularity / necrosis / hemorrhage; blood-brain barrier may remain intact*Histo:*homogeneous relatively uniform appearance with proliferation of well-differentiated multipolar fibrillary / protoplasmic astrocytes; mild nuclear pleomorphism + mild hypercellularity; mitoses rareLocation:posterior fossa in children, supratentorial in adults (typically lobar); distribution proportional to amount of white matter√ may develop a cyst with high-protein content (rare)CT: √ usually hypodense lesion with minimal mass effect + NO peritumoral edema√ well-defined tumor margins√ central calcifications (frequent)√ minimal / no contrast enhancement (normal capillary endothelial cells)MR: √ well-defined hypointense lesion with little mass effect / vasogenic edema / heterogeneity on T1WI√ hyperintense on T2WI√ little / no enhancement on Gd-DTPA√ cyst with content hyperintense to CSF (protein content)√ hyperintense area within tumor mass (paramagnetic effect of methemoglobin)√ inhomogeneous gadolinium-DTPA enhancement of tumor noduleAngio: √ majority avascular*Prognosis:*3-10 years postoperative survival; occasionally converting into more malignant form several years after presentation

Anaplastic Astrocytoma Pilocytic Astrocytoma

Notes:

Anaplastic Astrocytoma *Incidence:*11% of all primary intracranial neoplasms*Path:*frequently vasogenic edema; NO necrosis / hemorrhage*Histo:*less well differentiated with greater degree of hypercellularity + pleomorphism, multipolar fibrillary / protoplasmic astrocytes; mitoses + vascular endothelial proliferation commonLocation:typically lobarDistribution:proportional to amount of white matterMR: √ well-defined slightly heterogeneous hypointense lesion on T1WI with prevalent vasogenic edema√ hyperintense on T2WI√ ± enhancement on Gd-DTPA*Prognosis:*2 years postoperative survival

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Notes:

Pilocytic Astrocytoma =JUVENILE PILOCYTIC ASTROCYTOMA=most benign histologic subtype of astrocytoma without progression to high-grade glioma*Histo:*alternating pattern of compact bipolar pilocytic (hairlike) astrocytes arranged mostly around vessels + loosely aggregated protoplasmic astrocytes undergoing microcystic degeneration *Age:*predominantly in children + young adults; peak age between birth and 9 years of age; M:F = 1:1*Associated with:*neurofibromatosisLocation:cerebellum, hypothalamus (around 3rd ventricle), optic nerve / chiasm√ mural tumor nodule located in wall of cerebellar cyst√ multilobulated / dumbbell appearance along optic pathway√ rarely calcifies√ micro- / macrocysts in cerebellar location√ increased heterogeneous signal intensity on early Gd-DTPA enhanced T1WI; homogeneous enhancement on delayed images*Prognosis:*relatively benign clinical course, almost never recurs after surgical excision; NO malignant transformation to anaplastic form*DDx:*metastasis, hemangioblastoma, atypical medulloblastoma

WHO Classification of Astrocytomas

Grade I	Circumscribed astrocytoma	generally benign well-circumscribed tumor, specific unique histologic features for each tumor, pilocytic astrocytoma (most common), subependymal giant cell astrocytoma; no tendency to progress to higher grade; low rate of recurrence
Grade II	Astrocytoma	diffusely infiltrating; well-differentiated; minimal pleomorphism or nuclear atypia; no vascular proliferation / necrosis
Grade III	Anaplastic astrocytoma	pleomorphism and nuclear atypia; increased cellularity; mitotic activity; vascular proliferation + necrosis absent
Grade IV	Glioblastoma multiforme	marked vascular proliferation and necrosis; increased cellularity; anaplasia + pleomorphism; variable mitotic activity; cell type may be poorly differentiated, fusiform, round or multinucleated

Notes:

ATAXIA-TELANGIECTASIA
=autosomal recessive disorder characterized by telangiectasias of skin + eye, cerebellar ataxia, sinus + pulmonary infections, immunodeficiencies, propensity to develop malignancies*Incidence:*1:40,000 livebirths*Path:*neuronal degradation + atrophy of cerebellar cortex (? from vascular anomalies) ● cerebellar ataxia at beginning of walking age ● progressive neurologic deterioration ● oculomotor abnormalities, dysarthric speech, choreaathetosis, myoclonic jerks ● mucocutaneous telangiectasias: bulbar conjunctiva, ears, face, neck, palate, dorsum of hands, antecubital + popliteal fossa ● recurrent bacterial + viral sinopulmonary infections√ cerebellar cortical atrophy: diminished cerebellar size, dilatation of 4th ventricle, increased cerebellar sulcal prominence√ cerebral hemorrhage (rupture of telangiectatic vessels)√ cerebral infarct (emboli shunted through vascular malformations in lung)*Cx:* 1.Bronchiectasis + pulmonary failure (most common cause of death)2.Malignancies (10-15%): lymphoma, leukemia, epithelial malignancies

Notes:

BINSWANGER DISEASE
=ENCEPHALOPATHIA SUBCORTICALIS PROGRESSIVA=LEUKOARIAOSIS = SUBCORTICAL ARTERIOSCLEROTIC ENCEPHALOPATHY (SAE)*Cause:*arteriosclerosis affecting the poorly collateralized distal penetrating arteries (perforating medullary arteries, thalamoperforators, lenticulostriates, pontine perforators); positive correlation with hypertension + aging*Path:*ischemic demyelination / infarction*Age:*>60 years ● psychiatric changes, intellectual impairment, slowly progressive dementia, transient neurologic deficits, seizures, spasticity, syncopeLocation:periventricular white matter, centrum semiovale, basal ganglia; subcortical white matter "U" fibers + corpus callosum are spared√ multifocal hypodense lesions (periventricular, centrum semiovale) with sparing of U fibers√ lacunar infarcts in basal ganglia√ sulcal enlargement + dilated lateral ventricles (brain atrophy)MR: √ focal areas of increased signal intensity on T2WI(= "unidentified bright objects")
*DDx:*leukodystrophy, progressive multifocal leukoencephalopathy, multiple sclerosis

Notes:

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CANAVAN DISEASE
=SPONGIFORM LEUKODYSTROPHY=rare form of leukodystrophy as an autosomal recessive disorder, most common in Ashkenazi Jews*Incidence:*<100 reported cases *Cause:*deficiency of aspartoacyclase leading to accumulation of *N*-acetylaspartic acid in brain, plasma, urine, CSF*Histo:*spongy degeneration of white matter with astrocytic swelling + mitochondrial elongation*Age:*3-6 months ● marked hypotonia ● progressive megalencephaly ● seizures ● failure to attain motor milestones ● spasticity ● intellectual failure ● optic atrophy with blindness ● swallowing impairment√ diffuse symmetric white matter abnormality√ may involve basal ganglia√ cortical atrophyCT: √ low-density white matterMR: √ white matter hypointense on T1WI + hyperintense on T2WI*Prognosis:*death in 2nd-5th year of life*Dx:*(1)elevation of *N*-acetylaspartic acid in urine(2)deficiency of aspartoacyclase in cultured skin fibroblasts

Notes:

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CAPILLARY TELANGIECTASIA
=CAPILLARY ANGIOMA=abnormal dilated capillaries separated by normal neural tissue; commonly "cryptic"*May be associated with:* hereditary Rendu-Osler-Weber syndrome, ataxia-telangiectasia syndrome, irradiation (latency period of 5 months to 22 years) *Age:*typically in elderly ● usually asymptomatic (incidental finding at necropsy)Location:mostly in pons / midbrain; usually multiple / may be solitary↓ poorly defined area of dilated vessels (resembling petechiae)↓ best delineated with MR (due to hemorrhage)*Cx:*punctate hemorrhage (uncommon), gliosis + calcifications (rare)*Prognosis:*bleeding in pons usually fatal*DDx:*cavernous angioma (identical on images)

Notes:

CAVERNOUS HEMANGIOMA OF BRAIN

= CAVERNOUS ANGIOMA = CAVERNOMA *Path:*well-circumscribed nodule of honeycomblike large sinusoidal vascular spaces separated by fibrous collagenous bands without intervening neural tissue; slow blood flow in vascular channels*Age:*3rd-6th decade; M > F ● seizures (commonly presenting symptom)Location:cerebrum (mainly subcortical) > pons > cerebellum; solitary > multiple√ NO obvious mass effect / edemaNCCT: √ extensive calcifications = hemangioma calcificans (20%)√ small round hyperdense region (CLUE)√ minimal surrounding edemaCECT: √ minimal / intense enhancement√ low-attenuation areas due to thrombosed portionsMR: √ well-defined area of mixed signal intensity centrally(= "mulberry"-shaped lesion) with a mixture of√ increased signal intensity (= extracellular methemoglobin / slow blood flow / thrombosis)√ decreased intensity (= deoxyhemoglobin / intracellular methemoglobin / hemosiderin / calcification)√ surrounded by hypointense rim (= hemosiderin) on T2WIAngio: √ negative = "cryptic / occult vascular malformation"*Cx:*hemorrhage of varying ages*DDx:*(1)Hemorrhagic neoplasm (edema, mass effect)(2)Small AVM (thrombosed / small feeding vessels, associated hemorrhage)(3)Capillary angioma (no difference)

Notes:

CEPHALOCELE
=mesodermal defect in skull + dura with extracranial extension of intracranial structuresENCEPHALOCELE=herniation of brain tissue + meninges + CSFCRANIAL MENINGOCELE=herniation of meninges + CSF only*Prevalence:* 1-4 per 10,000 livebirths; 5-6-20% of all craniospinal malformations; predominant neural axis anomaly in fetuses spontaneously aborted <20 weeks GA *Cause:* failure of surface ectoderm to separate from neuroectoderm early in embryonic development @Skull base(1)faulty closure of neural tube (without mesenchyme membranous cranial bone cannot develop)(2)failure of basilar ossification centers to unite@Calvarium(1)defective induction of bone(2)pressure erosion of bone by intracranial mass / cyst*In 60% associated with:* (1)Spina bifida (7-30%)(2)Corpus callosum dysgenesis(3)Chiari malformation(4)Dandy-Walker malformation(5)Meckel-Gruber syndrome (= occipital encephalocele + microcephaly + cystic dysplastic kidneys + polydactyly)(6)Amniotic band syndrome: multiple irregular asymmetric off-midline encephaloceles(7)Migrational abnormalities(8)Chromosomal anomalies in 44% (trisomy 18) ● MS-AFP elevated in 3% (skin-covered in 60%) ● CSF rhinorrhea ● meningitis*Prognosis:*dependent on associated malformations + size and content of lesion; 21% liveborn; 50% survival in liveborns, 74% retarded*Risk of recurrence:*3% (25% with Meckel syndrome)*DDx:*teratoma, cystic hygroma, iniencephaly, scalp edema, hemangioma, branchial cleft cyst, cloverleaf skull

Occipital Encephalocele (75%) Frontoethmoidal Encephalocele (13-15%) Sphenoidal Encephalocele (10%) Parietal Encephalocele (10-12%)

Notes:

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Occipital Encephalocele (75%) Most common encephalocele in Western Hemisphere *Associated with:*Dandy-Walker malformation, Chiari malformation ● external occipital massLocation:supra- and infratentorial structures involved with equal frequency√ skull defect (visualized in 80%)√ flattening of basiocciput√ ventriculomegaly √ lemon sign = inward depression of frontal bones (33%)√ cyst-within-a-cyst (ventriculocele = herniation of 4th ventricle into cephalocele)√ acute angle between mass + skin line of neck and occiput*DDx:*cystic hygroma

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Notes:

Frontoethmoidal Encephalocele (13-15%) =sincipital cephalocele Most common variety in Southeast Asia *Cause:* failure of anterior neuropore located near optic recess to close normally at 4th week GA *Types:* nasoethmoidal, nasofrontal, naso-orbital, interfrontal *Associated with:* midline craniofacial dysraphism (dysgenesis of corpus callosum, interhemispheric lipoma, anomalies of neural migration)√ external mass near dorsum of nose, orbits, forehead √ hypertelorism = increase in interorbital distance

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Notes:

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Sphenoidal Encephalocele (10%) =basal encephalocele*Age:*present at end of first decade of life ● clinically occult ● mass in nasal cavity, nasopharynx, mouth, posterior portion of orbit ● mouth breathing due to nasopharyngeal obstruction ● nasopharyngeal mass increasing with Valsalva ● diminished visual acuity with hypoplasia of optic discs ● hypothalamic-pituitary dysfunction*Associated with:*agenesis of corpus callosum (80%)*Types:* (a)sphenopharyngeal = through sphenoid body(b)spheno-orbital = through superior orbital fissure(c)sphenoethmoidal = through sphenoid + ethmoid(d)transethmoidal = through cribriform plate(e)sphenomaxillary = through maxillary sinus

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Parietal Encephalocele (10-12%) *Associated with:*dysgenesis of corpus callosum, large interhemispheric cyst√ hole in sphenoid bone (seen on submentovertex film) √ cranium bifidum = cranioschisis = "split cranium" (= skull defect) = smooth opening with well-defined sclerotic rim of cortical bone√ hydrocephalus in 15-80% (from associated aqueductal stenosis, Arnold-Chiari malformation, Dandy-Walker cyst)√ nonenhancing expansile homogeneous paracranial mass√ mantle of cerebral tissue often difficult to image in encephalocele (except with MR)√ intracranial communication often not visualized√ metrizamide / radionuclide ventriculography diagnostic√ microcephaly (20%)√ polyhydramnios*DDx:*(1)sonographic refraction artifact at skull edge(2)clover leaf skull (temporal bone may be partially absent)

Notes:

CEREBELLAR ASTROCYTOMA

2nd most frequent tumor of posterior fossa in children *Incidence:*10-20% of pediatric brain tumors*Histo:*mostly grade I*Age:*children > adults; no specific age peak; M:F = 1:1*Path:* (1)cystic lesion with tumor nodule ("mural nodule") in cyst wall (50%); (midline astrocytomas cystic in 50%, hemispheric astrocytomas cystic in 80%)(2)solid mass with cystic (= necrotic) center (40-45%)(3)solid tumor without necrosis (<10%) ● cerebellar signs: truncal ataxia, dysdiadochokinesiaLocation:originating in midline with extension into cerebellar hemisphere (30%) > vermis > tonsils > brainstem√ calcifications (20%): dense / faint / reticular / punctate / globular; mostly in solid variety√ may develop extreme hydrocephalus (quite large when finally symptomatic)CT: √ round / oval cyst with density of cyst fluid > CSF√ round / oval / plaquelike mural nodule with intense homogeneous enhancement √ cyst wall slightly hyperdense + nonenhancing(= compressed cerebellar tissue) √ uni- / multilocular cyst (= necrosis) with irregular enhancement of solid tumor portions√ round / oval lobulated fairly well-defined iso- / hypodense solid tumor with hetero- / homogeneous enhancementMR: √ hypointense on T1WI + hyperintense on T2WI√ enhancement of solid tumor portionAngio:√ avascular*Prognosis:* malignant transformation exceedingly rare -40% 25-year survival rate for solid cerebellarastrocytoma-90% 25-year survival rate for cystic juvenile pilocytic astrocytoma*DDx of solid astrocytoma:* (1)medulloblastoma (hyperdense mass, noncalcified)(2)ependymoma (fourth ventricle, 50% calcify)*DDx of cystic astrocytoma:* (1)Hemangioblastoma (lesion <5 cm)(2)Arachnoid cyst(3)Trapped 4th ventricle(4)Megacisterna magna(5)Dandy-Walker cyst

Notes:

CEREBRITIS

=focal area of inflammation within brain substanceCT: √ area of decreased density ± mass effect√ no contrast enhancement (initially) / central or patchy enhancement (later)MR: √ focal area of increased intensity on T2WI*Cx:*brain abscess

Notes:

Chiari I Malformation (adulthood) ="cerebellar tonsillar ectopia" = herniation of cerebellar tonsils below a line connecting basion with opisthion (= foramen magnum)◊ Frequently isolated hindbrain abnormality of little consequence without supratentorial anomalies!*Proposed causes:* (a)small posterior fossa(b)disproportionate CSF absorption from subarachnoid spinal space(c)cerebellar overgrowth*Associated with:* (1)syringohydromyelia (20-30%)(2)hydrocephalus (25-44%)(3)malformation of skull base + cervical spine:(a)basilar impression (25%)(b)craniovertebral fusion, eg, occipitalization of C1 (10%), incomplete ossification of C1-ring (5%)(c)Klippel-Feil anomaly (10%)(d)platybasia◊NOT associated with myelomeningocele! ● benign cerebellar ectopia <3 mm of no clinicalconsequence; 3-5 mm of uncertain significance;>5 mm clinical symptoms likely ● no symptoms in childhood (unless associated with hydrocephalus / syringomyelia) ● may have cranial nerve dysfunction / dissociated anesthesia of lower extremities in adulthood√ downward displacement of cerebellar tonsils + medial part of the inferior lobes of the cerebellum 5 mm below the level of the foramen magnum√ inferior pointing peglike / triangular tonsils√ obliteration of cisterna magna√ elongation of 4th ventricle which remains in normal position√ slight anterior angulation of lower brainstem

Notes:

Chiari II Malformation (childhood) =ARNOLD-CHIARI MALFORMATION=most common and serious complex of anomalies secondary to a too small posterior fossa involving hindbrain, spine, mesodermHALLMARK is dysgenesis of hindbrain with (1)caudally displaced 4th ventricle(2)caudally displaced brainstem(3)tonsillar + vermian herniation through foramen magnum*Associated with:* (a)spinal anomalies(1)lumbar myelomeningocele (>95%)(2)syringohydromyelia(b)supratentorial anomalies(1)dysgenesis of corpus callosum (80-85%)(2)obstructive hydrocephalus (50-98%) following closure of myelomeningocele(3)absence of septum pellucidum (40%)(4)excessive cortical gyration(stenogyria = histologically normal cortex; polymicrogyria = histologically abnormal cortex) NOT associated with basilar impression / C1-assimilation / Klippel-Feil deformity! ● newborn: respiratory distress, apneic spells, bradycardia, impaired swallowing, poor gag reflex, retrocollis, spasticity of upper extremities ● teenager: gradual loss of function + spasticity of lower extremitiesSkull film: √ Lückenschädel (most prominent near torcular herophili / vertex) in 85% = dysplasia of membranous skull disappearing by 6 months of age√ scalloping of clivus + posterior aspect of petrous pyramids (from pressure of cerebellum) in 70-90% leading to shortening of IAC√ small posterior fossa√ enlarged foramen magnum + enlarged upper spinal canal secondary to molding in 75%√ absent / hypoplastic posterior arch of C1 (70%)@Supratentorial√ hydrocephalus (duct of Sylvius dysfunctional but probe patent); may not become evident until after repair of myelomeningocele (90%)√ colpocephaly (= enlargement of occipital horns + atria) due to maldeveloped occipital lobes√ hypoplasia / absence of splenium + rostrum of corpus callosum (80-90%)√ "bat-wing" configuration of frontal horns on coronal views = frontal horns pointing inferiorly with blunt superolateral angle secondary to prominent impressions by enlarged caudate nucleus√ "hourglass ventricle" = small biconcave 3rd ventricle secondary to large massa intermedia√ interdigitation of medial cortical gyri (hypoplasia + fenestration of falx in up to 100%)√ wide prepontine + supracerebellar cisterns√ nonvisualization of aqueduct (in up to 70%)√ stenogyria = multiple small closely spaced gyri at medial aspect of occipital lobe secondary to dysplasia (in up to 50%)@ Cerebellum √ "cerebellar peg" = protrusion of vermis + hemispheres through foramen magnum (90%) resulting in craniocaudal elongation of cerebellum √ hypoplastic poorly differentiated cerebellum (poor visualization of folia on sagittal images) secondary to severe degeneration√ elongated / obliterated vertically oriented thin-tubed 4th ventricle with narrowed AP diameter exiting below foramen magnum (40%)√ obliteration of CPA cistern + cisterna magna by cerebellum growing around brainstem√ dysplastic tentorium with wide U-shaped incisura inserting close to foramen magnum (95%)√ "tectal beaking" = fusion of midbrain colliculi into a single beak pointing posteriorly and invaginating into cerebellum√ V-shaped widened quadrigeminal plate cistern (due to hypoplasia of cingulate gyri)√ "towering cerebellum" = "pseudomass" = cerebellar extension above incisura of tentorium√ triple peak configuration = corners of cerebellum wrapped around brainstem pointing anteriorly + laterally (on axial images)√ flattened superior portion of cerebellum secondary to temporoparietal herniation√ vertical orientation of shortened straight sinus@ Spinal cord √ medulla + pons displaced into cervical canal√ "cervicomedullary kink" = herniation of medulla posterior to spinal cord (up to 70%) at level of dentate ligaments√ widened anterior subarachnoid space at level of brainstem + upper cervical spine (40%)√ AP diameter of pons narrowed√ upper cervical nerve roots ascend toward their exit foramina√ syringohydromyelia√ low-lying often tethered conus medullaris below L2OB-US: √ "banana sign" = cerebellum wrapped around posterior brainstem + obliteration of cisterna magna due to small posterior fossa√ hydrocephalus

Notes:

Chiari III Malformation most severe rare abnormality; probably unrelated to type I and II Chiari malformation √ low occipital / high cervical meningomyelo-encephalocele*Prognosis:*survival usually not beyond infancy

Notes:

Chiari IV Malformation extremely rare anomaly probably erroneously included as type of Chiari malformation √ agenesis of cerebellum √ hypoplasia of pons √ small + funnel-shaped posterior fossa

Notes:

CHOROID PLEXUS CYST

=cyst arising from folding of neuroepithelium with trapping of secretory products + desquamated cells*Incidence:*0.9-3.6% in sonographic population; 50% of autopsied brains*Histo:*no epithelial lining, filled with clear fluid ± debris*May be associated with:* aneuploidy (76% with trisomy 18, 17% with trisomy 21, 7% with triploidy / Klinefelter syndrome) ◊In absence of other anomalies 1% of fetuses with choroid plexus cysts will have trisomy 18!◊In presence of other anomalies 4% of fetuses with choroid plexus cysts will have trisomy 18!◊40-71% of autopsied fetuses with trisomy 18 have choroid plexus cysts bilaterally >10 mm in diameter◊Risk of chromosomal abnormality not linked to size, bilaterality, gestational age at appearance / disappearance ● usually asymptomaticLocation:frequently at level of atrium; uni- / bilateral√ single / multiple round anechoic cysts√ ≥3 mm in size (average 4.5 mm, up to 25 mm)*Cx:*hydrocephalus (if cyst large)*Prognosis:*90% disappear by 28th week; may persist; in 95% of no significance*OB-management:* a choroid plexus cyst should stimulate a thorough sonographic examination at >19 weeks; if no other sonographic abnormalities are identified, the yield of abnormal karyotype is low so that the risk of trisomy 18 (1:450-500) is lower than risk of fetal loss due to amniocentesis (approximately 1:200-300) *Risk of karyotype abnormality:* 10 x with 1 additional defect 600 x with ≥2 additional defects *DDx:*Choroid plexus pseudocyst in the inferolateral aspect of atrium (? corpus striatum) on oblique coronal plane which elongates by turning transducer

Notes:

CHOROID PLEXUS PAPILLOMA

*Incidence:*0.5-0.6% of all intracranial tumors; 2-5% of brain tumors in childhood*Age:*20-40% <1 year of age; 86% <5 years of age; middle age; in 75% <2 years of age; M >> F*Path:*large aggregation of choroidal fronds producing great quantities of CSF; occasionally found incidentally on postmortem examination*Pathophysiology:*abnormal rate of CSF production of 1.0 mL/min (normal rate = 0.2 mL/min) ● signs of increased intracranial pressureLocation: (a)glomus of choroid plexus in trigone of lateral ventricles, L > R (in children)(b)4th ventricle + cerebellopontine angle (in adults)(c)3rd ventricle (unusual)(d)multiple in 7%√ large mass with smooth lobulated border√ small foci of calcifications (common)√ engulfment of glomus of choroid plexus (distinctive feature)√ asymmetric diffuse ventricular dilatation(CSF overproduction / decreased absorption secondary to obstruction of arachnoid granulations from repeated occult hemorrhage) √ dilatation of temporal horn in atrial location (obstruction)√ growth into surrounding white matter (occasionally, more common a feature of choroid plexus carcinoma)CT: √ iso- / mildly hyperdense with intense homogeneous enhancement on CECTMR: √ isointense / slightly hyperintense lesion on T1WI + slightly hypointense on T2WI relative to white matter√ surrounded by hypointense signal on T1WI + hyperintense signal on T2WI (CSF)√ intraventricular enhancing island of tumor on Gd-DTPAUS: √ echogenic mass adjacent to normal choroid plexusAngio: √ supplied by anterior + posterior choroidal arteries*Cx:*(1)transformation into malignant choroid plexus papilloma = choroid plexus carcinoma(2)hydrocephalus (in children) secondary to increased intracranial pressure from CSF-overproduction*Rx:*surgical removal (24% operative mortality) cures hydrocephalus*DDx:*intraventricular meningioma, ependymoma, metastasis, cavernous angioma, xanthogranuloma, astrocytoma

Notes:

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COCKAYNE SYNDROME

=autosomal recessive diffuse demyelinating disease*Age:*beginning at age 1 ● dwarfism ● progressive physical + mental deterioration ● retinal atrophy + deafness√ brain atrophy / microcephaly√ calcifications in basal ganglia + cerebellum√ skeletal changes superficially similar to progeria*DDx:*Progeria

Notes:

COLLOID CYST
*Incidence:*2% of glial tumors of ependymal origin;0.5-1% of CNS tumors *Histo:*ciliated + columnar epithelium; mucin-secreting; squamous cells of ependymal origin; tough fibrous capsule*Age:*young adults; M > F ● positional headaches (transient obstruction secondary to ball-valve mechanism at foramen of Monro) ● gait apraxia ● change in mental status ± dementia (related to increased intracranial pressure) ● papilledema (may become medical emergency with acute herniation)Location:exclusively arising from inferior aspect of septum pellucidum protruding into anterior portion of 3rd ventricle between columns of fornix √ ± sellar erosion√ spherical iso- / hyperdense lesion on NCCT with smooth surface√ fluid contents:(a)in 20% similar to CSF (= isodense)(b)in 80% mucinous fluid, proteinaceous debris, hemosiderin, desquamated cells (= hyperdense)√ may show enhancement of border (draped choroid plexus / capsule)√ 3rd ventricular enlargement (to accommodate cyst anteriorly)√ asymmetric lateral ventricular enlargement (invariably)√ occasionally widens septum pellucidumMR: √ lesion hyperintense on T1WI + hyperintense on T2WI in 60% (related to large protein molecules / paramagnetic effect of magnesium, copper, iron in cyst)*DDx:*meningioma, ependymoma of 3rd ventricle (rare) with enhancement

Notes:

CORTICAL CONTUSION
=traumatic injury to cortical surface of brain*Incidence:*most common type of primary intra-axial lesion; in 21% of head trauma patients; children:adults = 2:1*Path:*tissue necrosis, capillary disruption, petechial hemorrhage followed by liquefaction + edema after 4-7 days*Mechanism:*linear acceleration-deceleration forces / penetrating trauma 1.**Coup** = direct impact on stationary brain2.**Contrecoup** = impact of moving brain on stationary calvarium opposite to the site of the coupLocation:multiple bilateral lesions;-common: along anterior + lateral + inferior surfaces of frontal lobe (in orbitofrontal, inferior frontal, and rectal gyri above cribriform plate, planum sphenoidale, lesser sphenoid wing) and temporal lobe (just above petrous bone / posterior to greater sphenoid wing)-less frequent: in parietal + occipital lobes, cerebellar hemispheres, vermis, cerebellar tonsils-often bilateral / beneath an acute subdural hematoma ● confusion (mild initial impairment) ● focal cerebral dysfunction ● seizures, personality changes ● focal neurologic deficits (late changes)CT (sensitive only to hemorrhage in acute phase): ◊Look for scalp swelling to focus your attention on the location of the coup!√ focal / multiple (29%) poorly defined areas of low attenuation with irregular contour (edema) intermixed with a few tiny areas of increased density (petechial hemorrhage)√ diffuse cerebral swelling without hemorrhage in immediate posttraumatic period (common in children) due to hyperemia / ischemic edema√ some degree of contrast enhancement (leaking new capillaries)√ isodense hemorrhage after 2-3 weeks√ true extent of lesions becomes more evident with progression of edema + cell necrosis + mass effect over ensuing weeksMR (best modality for initial detection of contusional edema + accurate portrayal of extent of lesions): √ hemorrhagic lesions (detected in 50% of all contusions):√ initially decreased intensity (deoxyhemoglobin of acute hemorrhage) surrounded by hyperintense edema on T2WI√ hyperintense on T1WI + T2WI in subacute phase (secondary to Met-Hb)√ hyperintense gliosis + hypointense hemosiderin on T2WI in chronic phase√ nonhemorrhagic lesions hypointense on T1WI + hyperintense on T2WI*Cx:*(1)Encephalomalacia (= scarred brain)(2)Porencephaly (= formation of cystic cavity lined with gliotic brain and communicating with ventricles / subarachnoid space)(3)Hydrocephalus as a result from adhesions caused by subarachnoid blood

Notes:

CRANIOPHARYNGIOMA
*Incidence:*3-4% of all intracranial neoplasms; 15% of supratentorial + 50% of suprasellar tumors in children; most common suprasellar mass*Origin:*from epithelial rests along vestigial craniopharyngeal duct (Rathke cleft / pouch within intermediate lobe of pituitary gland)*Path:*benign tumor originating from neuroepithelium in craniopharyngeal duct + primitive buccal epithelium*Histo:*cystic (rich in liquid cholesterol) / complex / solid*Age:*from birth-7th decade; bimodal age distribution: age peaks in 1st-2nd decade (75%) + in 5th decade (25%); M > F ● diabetes insipidus (compression of pituitary gland) ● growth retardation (compression of hypothalamus) ● bitemporal hemianopia (compression of optic nerve chiasm) ● headaches from hydrocephalus (compression of foramen of Monro / aqueduct of Sylvius)Location: (a)pituitary stalk / tuber cinereum(b)suprasellar (20%)(c)intrasellar (10%)(d)intra- and suprasellar (70%)**Ectopic craniopharyngioma:** (e)floor of anterior 3rd ventricle (more common in adults)(f)sphenoid boneSkull films: √ normal sella (25%)√ enlarged J-shaped sella with truncated dorsum√ thickening + increased density of lamina dura in floor of sella (10%)√ extensive sellar destruction (75%)√ curvilinear / flocculent / stippled calcifications / lamellar ossification; calcifications seen in youth in 70-90%, in adults in 30-40%CT: √ multilobulated inhomogeneous suprasellar mass√ solid (15%) / mixed (30%) / cystic lesion (54-75%) [cystic appearance secondary to cholesterol, keratin, necrotic debris with higher density than CSF]√ enhancement of solid lesion, peripheral enhancement of cystic lesion√ marginal hyperdense lesion (calcification / ossification) in 70-90% in childhood tumors +30-50% of adult tumors √ ± obstructive hydrocephalus√ extension into middle > anterior > posterior cranial fossa (25%)MR (relatively ineffective in demonstrating calcifications): √ mostly hyperintense, but also iso- / hypointense on T1WI (variable secondary to hemorrhage / cholesterol-containing proteinaceous fluid)√ markedly hyperintense on T2WI√ marginal enhancement of solid components with gadopentetate dimeglumineAngio: √ usually avascular√ lateral displacement, elevation, narrowing of supraclinoid segment of ICA√ posterior displacement of basilar artery*DDx:*(1)Epidermoid (no contrast enhancement)(2)Rathke cleft cyst (small intrasellar lesion)

Notes:

CYSTICERCOSIS OF BRAIN

larva of pork tapeworm (Taenia solium) frequently involving CNS, muscles, heart, fat tissue *Infection:* (1)Ingestion of ova by fecal-oral route; embryophore is dissolved by gastric acid and enzymes + oncosphere is liberated(2)Ingestion of uncooked contaminated pork containing cysticerci; tapeworm develops in intestinal lumen + releases eggs*Organism:* embryos invade intestinal wall + enter circulation + disseminate in varies parts of body; embryo develops into a cysticercus (= complex wall surrounding a cavity containing vesicular fluid + scolex); following ingestion of cysticercus by definitive host a tapeworm develops within the intestinal tract *Incidence:*CNS involvement in up to 90%Location:meninges (39%) esp. in basal cisterns, parenchyma (20%), intraventricular (17%), mixed (23%), intraspinal (1%) A.ACUTE PAHSE (= focal meningoencephalitis) ● focal seizures√ single / multiple small focal enhancing lesions; transitory with resolution in a few months√ diffusely edematous white matter√ homogeneously enhancing small nodules often with extensive edema (DDx: metastases without edema)B.CHRONIC PHASE (= involution with subsequent calcification + cyst formation)√ small focal calcifications (= probably dead larvae); may appear within 8 months to 10 years after acute infection along gray-white matter junction√ well-defined cystic areas of CSF density without associated edema (= living larvae)√ "ricelike" muscle calcifications rarely visible√Cysts incite edema upon death of larvae!

RADIOGRAPHIC TYPES 1.Parenchymal type√ multiple / solitary cystic lesions up to 6 cm in size; many terminate as calcified granulomata (larvae not dead unless completely calcified)√ encephalitic form may occur in children2.Meningeal / racemose type√ ventricular dilatation indicating diffuse meningeal inflammatory process√ lucent cystic lesions in basal cisterns (= racemose cysts) with variable enhancement, usually located in cerebellopontine angle / suprasellar cistern3.Intraventricular type√ obstructive hydrocephalus caused by blockage within various portions of ventricular system from solitary / multiple cysts4.Mixed type (frequent)

Notes:

CYTOMEGALOVIRUS INFECTION

Most common intrauterine infection *Incidence:*0.4-2.3% of liveborn infants ● asymptomatic (90%) ● sensorineural hearing loss, chorioretinitis, mental retardation, neurologic deficits√ intrauterine growth retardation√ ascites√ hydrops@CNS√ periventricular calcifications√ ventricular dilatation√ microcephaly

Notes:

DANDY-WALKER MALFORMATION
=characterized by (1) enlarged posterior fossa with high position of tentorium (2) dys- / agenesis of cerebellar vermis (3) cystic dilatation of 4th ventricle filling nearly entire posterior fossa*Cause:*dysmorphogenesis of roof of 4th ventricle with failure to incorporate the area membranacea into developing choroid plexus; proposed originally as congenital atresia of foramina of **L**uschka (**l**ateral) + **M**agendie (**m**edian) not likely since foramina are not patent until 4th month*Incidence:*12% of all congenital hydrocephaly*Path:*defect in vermis connecting an ependyma-lined retrocerebellar cyst with 4th ventricle (PATHOGNOMONIC)
Associated anomalies : -midline CNS anomalies (in >60%)(1)dysgenesis of corpus callosum (20-25%), lipoma of corpus callosum(2)holoprosencephaly (25%)(3)malformation of cerebral gyri (dysplasia of cingulate gyrus) (25%)(4)cerebellar heterotopia + malformation of cerebellar folia (25%)(5)malformation of inferior olivary nucleus(6)hamartoma of tuber cinereum(7)syringomyelia(8)cleft palate(9)occipital encephalocele (<5%)-other CNS anomalies:(1)polymicrogyria / gray matter heterotopia (5-10%)(2)schizencephaly(3)lumbosacral meningocele-non-CNS anomalies (25%)(1)polydactyly, syndactyly(2)Klippel-Feil syndrome(3)Cornelia de Lange syndrome(4)cleft palate(5)facial angioma(6)cardiac anomaliesSkull film: √ large skull secondary to hydrocephalus + dolichocephaly√ diastatic lambdoid suture√ disproportionately large expanded posterior fossa√ torcular herophili and lateral sinuses high above lambdoid angle = torcular-lambdoid inversionCT / US / MR: √ absence / hypoplasia of cerebellar vermis:total (25%), partial (75%)√ superiorly displaced superior vermis cerebelli√ small + widely separated cerebellar hemispheres √ anterior + lateral displacement of ± hypoplastic cerebellar hemispheres√ large posterior fossa cyst with extension through foramen magnum = diverticulum of roofless 4th ventricle√ elevated insertion of tentorium cerebelli√ cerebellar hemispheres in apposition without intervening vermis following shunt procedure√ absence of falx cerebelli√ scalloping of petrous pyramids√ ventriculomegaly (in 72% open communication with 3rd ventricle; in 39% patent 4th ventricle; in 28% aqueductal stenosis; in 11% incisural obstruction); present prenatally in 30%, by 3 months of age in 75%√ anterior displacement of ponsAngio: √ high position of transverse sinus√ elevated great vein of Galen√ elevated posterior cerebral vessels√ anterosuperiorly displaced superior cerebellar arteries above the posterior cerebral arteries√ small / absent PICA with high tonsillar loop*Cx:*trapping of cyst above tentorium = "keyhole configuration"*Prognosis:*fetal demise in 66%; 22-50% mortality during 1st year of life*DDx:*(1)Posterior fossa extra-axial cyst(2)Arachnoid cyst (normal 4th ventricle, patent foramina, intact vermis)(3)Isolated 4th ventricle(4)Megacisterna magna = giant cisterna magna (enlarged posterior fossa, enlarged cisterna magna, intact vermis, normal 4th ventricle)(5)Porencephaly

Dandy-Walker Variant Dandy-Walker Complex Pseudo-Dandy-Walker Malformation

Notes:

Dandy-Walker Variant characterized by (1)variable hypoplasia of posteroinferior portion of vermis leading to communication between 4th ventricle and cisterna magna(2)cerebellar dysgenesis(3)cystic dilatation of 4th ventricle(4)NO enlargement of posterior fossa More common than Dandy-Walker malformation; accounts for 1/3 of all posterior fossa malformations*Cause:*focal insult to developing cerebellum*Associated CNS anomalies:* agenesis of corpus callosum (21%), cerebral gyral malformation (21%), heterotopia, holoprosencephaly (10%), diencephalic cyst (10%), posterior fossa meningoencephalocele (10%) *Other associated anomalies:* polydactyly; cardiac, renal, facial anomalies; abnormal karyotype (29%) √ 4th ventricle smaller + better formed√ retrocerebellar cyst smaller√ communication between retrocerebellar cyst and subarachnoid space through a patent foramen of Magendie may be present√ posterior fossa smaller than in usual Dandy-Walker syndromeOB-US: √ incomplete closure of vermis is normal until 18 weeks GA!

Notes:

Dandy-Walker Complex =continuum of anomalies, including Dandy-Walker malformation + Dandy-Walker variant + megacisterna magna, characterized by partial / complete dysgenesis of vermis cerebelli*Cause:*broad insult to alar plate from a variety of abnormalities*Associated with:* A.Inherited genetic syndromes-autosomal recessive:1.Meckel-Gruber syndrome2.Ellis-van Creveld syndrome3.Walker-Warburg syndrome-autosomal dominant:1.X-linked cerebellar hypoplasia2.Aicardi syndromeB.Abnormal karyotype (33%)1.Duplications of chromosomes 5p, 8p, 8q2.Trisomies 9, 13, 18C.Infection1.Virus: CMV, rubella2.Protozoan: toxoplasmosisD.Teratogen: alcohol, sodium warfarinE.Multifactorial

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Notes:

Pseudo-Dandy-Walker Malformation =developing rhombencephalon during 1st trimester√ fluid-filled space in posterior aspect of fetal head

Notes:

DERMOID OF CNS

=pilosebaceous mass lined with skin appendages originating from inclusion of epithelial cells + skin appendages during closure of neural tube *Incidence:* 1% of all intracranial tumors *Path:* ectodermal + mesodermal lesion = squamous epithelium, mesodermal cells (hair follicles, sweat + sebaceous glands) *Age:* <30 years (appears in adulthood secondary to slow growth); M < FLocation: (a)spinal canal (most common): extra- / intramedullary in lumbosacral region (b)posterior fossa within vermis / 4th ventricle (predilection for midline)(c)posterior to superior orbital fissure, may be associated with bone defect ● bouts of chemical / bacterial meningitis possible√ thick-walled inhomogeneous mass with focal areas of fat√ mural / central calcifications / bone (possible)√ may have sinus tract to skin surface (dermal sinus) if located in midline at occipital / nasofrontal region√ fat-fluid level if cyst ruptures into ventricles, fat droplets in subarachnoid space√ NO contrast enhancementMR: √ variointense on T1WI (hyperintense with contents of liquefied cholesterol products)√ shortened T1 + T2 relaxation times (= fat)

Notes:

DIFFUSE AXONAL INJURY

=WHITE MATTER SHEARING INJURY*Incidence:*most common type of primary traumatic injury in patients with severe head trauma (48%)*Cause:*indirect injury due to rotational acceleration / deceleration forces (not necessarily with direct impact to head)*Pathogenesis:* cortex and deep structures move at different speed resulting in shearing stress along the course of white matter tracts especially at gray-white matter junction with axonal tears followed by wallerian degeneration *Path:*much of the injury is only microscopic*Histo:*multiple axonal retraction balls (HALLMARK), numerous perivascular hemorrhages ■ severe impairment of consciousness
Location (according to severity of trauma): (a)lobar white matter at corticomedullary junction (67%): parasagittal region of frontal lobe + periventricular region of temporal lobe; occasionally in parietal + occipital lobes(b)internal + external capsule, corona radiata, cerebellar peduncles (c)corpus callosum (21%): 3/4 of lesions in posterior body + splenium√ often associated with intraventricular hemorrhage(d)brainstem: posterolateral quadrants of midbrain + upper pons; superior cerebellar peduncles especially vulnerable
√ sparing of cortex√ 20% of lesions with small central areas of petechial hemorrhageCT: √ foci of decreased density (usually seen when >1.5 cm in size)MR (most sensitive modality): √ multiple small oval / round foci of decreased signal intensity on T1WI + increased signal on T2WI*Prognosis:*poor due to sequelae (may go on to die without signs of high intracranial pressure)

Notes:

DIFFUSE SCLEROSIS

sporadic, young adults, fulminant course ● dementia, deafness ● low-attenuation regions in both hemispheres without symmetry

Notes:

DYKE-DAVIDOFF-MASON SYNDROME
=CEREBRAL HEMIATROPHY = INFANTILE / CONGENITAL HEMIPLEGIA = SYNDROME OF HEMICONVULSIONS, HEMIPLEGIA, AND EPILEPSY= unilateral cerebral atrophy with ipsilateral small skull *Cause:*insult to immature brain resulting in neuronal loss + impaired brain growth:(a)prenatal: congenital malformation, infection, vascular insult(b)perinatal: birth trauma, anoxia, hypoxia, intracranial hemorrhage(c)postnatal: trauma, tumor, infection, prolonged febrile seizures ● seizures ● hemiparesis (typically spastic hemiplegia) ● mental retardation*Age:*presents in adolescence√ unilateral thickening of skull√ unilateral decrease in size of cranial fossa√ unilateral overdevelopment of sinuses√ contraction of a hemisphere / lobe√ compensatory enlargement of adjacent ventricle + sulci with midline shift

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Notes:

EMPTY SELLA SYNDROME
=extension of subarachnoid space into sella turcica, which becomes exposed to CSF pulsations secondary to defect in diaphragma sellae; characterized by normal / molded pituitary gland + normal or enlarged sella (empty sella = misnomer)*Incidence:*24% in autopsy study
A.PRIMARY EMPTY SELLA (anatomic spectrum)*Incidence:*10% of adult population; M:F = 1:4*Probable causes:* (1)pituitary enlargement followed by regression during pregnancy(2)involution of a pituitary tumor(3)congenital weakness of diaphragma sellae●occurs more frequently in patients with increased intracranial pressure ● usually asymptomatic ● increased risk for CSF rhinorrhea ● NO endocrine abnormalitiesB.SECONDARY EMPTY SELLA=postsurgical when diaphragma sellae has been disrupted ● visual disturbance ● headaches
√ slowly progressive symmetrical / asymmetrical (double floor) enlargement of sella√ remodeled lamina dura remains mineralized√ small rim of pituitary tissue displaced posteriorly + inferiorly√ infundibulum sign = infundibulum extends to floor of sella*DDx:*cystic tumor, large herniated 3rd ventricle (displaced infundibulum)

Notes:

Subdural Empyema 20% of all intracranial bacterial infections *Cause:*paranasal sinusitis, otitis media, calvarial osteomyelitis, infection after craniotomy or ventricular shunt placement, penetrating wound, contamination of meningitis-induced subdural effusionLocation:frontal + inferior cranial space in close proximity to paranasal sinuses; 80% over convexity extending into interhemispheric fissure or posterior fossa√ hypo- / isodense crescentic / lentiform zone adjacent to inner table√ may show mass effect (sulcal effacement, ventricular compression, shift)√ thin curvilinear rim of enhancement (7-10 days later) adjacent to brain√ severe sinusitis / mastoiditis (may be most significant indicator)*Mortality:*30% (neurosurgical emergency)*Cx:*venous thrombosis, infarction, seizures, hemiparesis, hemianopia, aphasia, brain abscess*DDx:*subacute / chronic subdural hematoma

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Notes:

Epidural Empyema *Cause:*same as above ● no neurologic deficits (dura minimizes pressure exerted on brain)√ thick enhancing rim

Notes:

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ENCEPHALITIS
=term generally reserved for diffuse inflammatory process of viral etiology (herpes simplex, California encephalitis, Eastern equine encephalitis, St. Louis encephalitis, Western equine encephalitis)√ diffuse mild cerebral edema√ small infarctions / hemorrhage (less frequent)

Acute Hemorrhagic Leukoencephalitis Herpes Simplex Encephalitis (HSE) Postinfectious Encephalitis

Notes:

Acute Hemorrhagic Leukoencephalitis =fulminant myelinoclastic disease of CNS=hyperacute form of acute disseminated encephalomyelitis*Cause:*immunoreactive disease following prodromal illness (minor upper respiratory viral infection, ulcerative colitis)*Path:*marked edema, brain softening*Histo:*necrotizing angiitis of venules + capillaries within white matter with extravasation of PMNs + lymphocytes; fibrinoid necrosis of affected capillaries + surrounding tissues; confluent hemorrhages with ball-and-ring configuration due to diapedesis of RBCs ● progressive coma, motor disturbance, speech difficulty, seizures ● pyrexia, leukocytosis ● pleocytosis, elevated protein in spinal fluidLocation:unilateral disease; parietal + posterior frontal white matter at level of centrum semiovale (sparing subcortical U-fibers + cortex) > basal ganglia, cerebellum, brainstem, spinal cord√ rapid development of profound mass effect resembling infarction√ multiple punctate white matter hemorrhages√ extensive hypoattenuation virtually confined to hemispheric white matter*Prognosis:*usually results in death*DDx:*(1)Herpes simplex encephalitis (cortical lesions in temporal + inferior frontal lobes + insular region, no imaging findings until 3-5 days after onset of significant symptoms)(2)Tumefactive multiple sclerosis(3)Osmotic demyelination(4)Toxic encephalopathy: lipophilic solvent, methanol(5)Hypertensive encephalopathy: eclampsia, thrombotic thrombocytopenic purpura

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Notes:

Herpes Simplex Encephalitis (HSE) =most common cause of nonepidemic necrotizing meningoencephalitis in USA*Organism:*HSV type I (in adults); HSV type II (in neonates from transplacental infection) ● confusion, disorientation ● preceding viral syndrome, fever, headache, seizuresLocation:temporal > frontal > parietal lobes; propensity for limbic system (olfactory tract, temporal lobes, cingulate gyrus, insular cortex); predominantly unilateralCT (principal role is to identify biopsy site): √ may be negative in first 3 days√ poorly defined bilateral areas of decreased attenuation√ spared putamen forms sharply defined concave / straight border (DDx: infarction, glioma)√ compression of lateral ventricles, sylvian fissure (brain edema)√ patchy peripheral / gyral / cisternal enhancement (50%), may persist for several months√ tendency for hemorrhage + rapid dissemination in brainMR: √ increased signal intensity on T2WINUC: *Agents:*standard brain imaging (eg, Tc-99m DTPA), newer brain agents (eg, I-123 iodoamphetamine / Tc-99m HMPAO)SPECT imaging improves sensitivity √ characteristic focal increase in activity in temporal lobes on brain scintigraphy (blood-brain barrier breakdown)*Dx:*fluorescein antibody staining / viral culture from brain biopsy*Mortality:*70%*Rx:*adenine arabinoside*DDx:*low-grade glioma, infarct, abscess

Human Immunodeficiency Virus Encephalitis often in combination with CMV encephalitis *Histo:*microglial nodules + perivascular multinucleated giant cells accompanying gliosis of deep white + gray matter√ predominantly central CNS atrophy√ symmetric periventricular / diffuse white matter disease without mass effect (hypodense on CT, high intensity on T2WI)

Notes:

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Postinfectious Encephalitis following exanthematous viral illness / vaccination **Acute disseminated encephalomyelitis (ADEM)** =autoimmune reaction against patients white matter following measles, mumps, varicella, pertussis, rubella infection / vaccination ● seizures + focal neurologic signs 7-14 days after clinical onset of viral infection*Histo:*diffuse perivenous inflammatory process resulting in areas of demyelinationLocation:subcortical white matter of both hemispheres asymmetricallyCT: √ hypodense white matterMR: √ focal areas of hyperintensity on T2WI√ may demonstrate contrast enhancement*Rx:*corticosteroids result in dramatic improvement*Prognosis:*complete recovery / some permanent neurologic damage (10-20%)*DDx:*simulating multiple sclerosis (rarely recurrent episodes as in multiple sclerosis)

Notes:

EPENDYMOMA
=in majority benign slow-growing neoplasm of mature well-differentiated ependymal cells lining the ventricles*Incidence:*most commonly in children; 5-9% of all primary CNS neoplasms; 15% of posterior fossa tumors in children; 63% of spinal intramedullary gliomas*Histo:*benign aggregates of ependymocytes in form of perivascular pseudorosettes; may have papillary pattern (difficult DDx from choroid plexus papilloma)*Age:*(a)supratentorial: at any age (atrium / foramen of Monro)(b)posterior fossa: <10 years; age peaks at 5 and 34 years; M:F = 0.8:1*Associated with:*neurofibromatosis ● increased intracranial pressure (90%)Location: (a)infratentorial: floor of 4th ventricle (70% of all intracranial ependymomas)(b)supratentorial: frontal > parietal > temporoparietal juxtaventricular region (uncommonly intraventricular), lateral ventricle, 3rd ventricle(c)conus (40-65% of all spinal intramedullary gliomas)in children: infratentorial in 70%, supratentorial in 30% √ small cystic areas in 15-50% (central necrosis)√ fine punctate multifocal calcifications (25-50%)√ intratumoral hemorrhage (10%)√ frequently grows into brain parenchyma extending to cortical surface (particularly in frontal + parietal lobes)√ may invaginate into ventricles√ expansion frequently through foramen of Luschka into cerebellopontine angle (15%) or through foramen of Magendie caudad into cisterna magna (up to 60%) (CHARACTERISTIC)√ direct invasion of brainstem / cerebellum (30-40%)√ insinuation around blood vessels + cranial nerves√ communicating hydrocephalus (100%) secondary to protein exudate elaborated by tumor clogging resorption pathwaysCT: √ sharply marginated multilobulated iso- / slightly hyperdense 4th ventricular mass√ thin well-defined low-attenuation halo (distended effaced 4th ventricle)√ heterogeneous / moderately uniform enhancement of solid portions (80%)MR: √ low to intermediate heterogeneous signal intensity on T1WI√ hypointense tumor margins on T1WI + T2WI in 64% (hemosiderin deposits)√ foci of high-signal intensity on T2WI (= necrotic areas / cysts) + low signal intensity (= calcification / hemorrhage)√ fluid-fluid level within cysts√ homogeneous Gd-DTPA enhancement of tumor*Cx:*subarachnoid dissemination via CSF (rare) (DDx: malignant ependymoma, ependymoblastoma)*Rx:*surgery (difficult to resect due to adherence to surrounding brain) + radiation (partially radiosensitive) + chemotherapy
DDx of cerebellar ependymoma: (1)Astrocytoma (hypodense, displaces 4th ventricle from midline, cystic lucency, intramedullary)(2)Medulloblastoma (hyperdense, calcifications in only 10%)(3)Trapped 4th ventricle (no contrast enhancement)

Notes:

EPIDERMOID OF CNS

=EPIDERMOID [INCLUSION] CYST=benign tumor with extremely slow linear growth resulting from desquamation of epithelial cells from tumor wall*Incidence:*0.2-1.8% of all primary intracranial neoplasms; most common congenital intracranial tumor*Etiology:*inclusion of ectodermal epithelial tissue from pharyngeal pouch of Rathke / pluripotential cells during closure of neural tube in 5th week of fetal life (early inclusion results in midline lesion, later inclusion results in more lateral location)*Path:*"pearly tumor" = well-defined solid lesion with glistening irregular nodular surface; soft flaky desquamated keratinaceous debris rich in cholesterol + triglycerides = PRIMARY / CONGENITAL CHOLESTEATOMA*Histo:*tumor lined by simple stratified cuboidal squamous epithelium; surrounded by thin band of collagenous connective tissue; tumor center of lamellar appearance due to desquamation*Age:*10-60 years, peak age in 4th-5th decade; tumor slowly expands over decades by continued desquamation of the lining thus becoming symptomatic in adulthood; M:F = 1:1 ● facial pain ● cranial nerve palsies from CP angle epidermoids (50%) ● hydrocephalus in suprasellar epidermoids ● chemical meningitis (secondary to leakage of tumor contents into subarachnoid space) in middle cranial fossa epidermoids

Site:midline / paramidline; intradural (90%) / extradural; transspatial growth (= extension from one into another intracranial space)Location:(a)cerebellopontine angle (40%, account for 5% of CP angle tumors)(b)suprasellar region, perimesencephalic cisterns (14%)(c)within ventricles, brainstem, brain parenchyma(d)skull vault√ soft lesion conforming to + molding itself around brain surfaces√ may intimately surround vessels + cranial nerves rather than displacing them (limited resectability)√ little mass effect, no edema / hydrocephalus√ NO contrast enhancement√ may be associated with dermal sinus tract at occipital / nasofrontal region if midline in locationCT: √ typically lobulated round homogeneous mass with density similar to CSF (between water and -20 HU)√ occasionally hyperdense due to high protein content, saponification of keratinaceous debris, prior hemorrhage into cyst, ferrocalcium / iron-containing pigment, abundance of PMNs√ bony erosion with sharply defined well-corticated margins√ calcification (25%)√ peripheral enhancement (perilesional inflammation)MR: √ lamellated onionskin appearance with septations (layer-on-layer accretion of desquamated material)√ "black epidermoid" = signal intensity similar to CSF: heterogeneously hypointense lesion on T1WI + hyperintense on T2WI (due to cholesterol in solid crystalline state + keratin within tumor + CSF within tumor interstices)√ "white epidermoid" (rare) = hyperintense on T1WI + isointense on T2WI due to presence of triglycerides + polyunsaturated fatty acids√ hypointense on T2WI (very rare) due to calcification, low hydration, viscous secretion, paramagnetic iron-containing pigmentAngio: √ avascularCisternography: √ papillary / frondlike surface with contrast material extending into tumor interstices*Rx:*surgical resection (complicated by adherence to surrounding brain + cranial nerves, spillage of cyst contents with chemical meningitis, CSF seeding + implantation)*DDx:*arachnoid cyst (smooth surface, earlier diffusion), cystic schwannoma, adenomatoid tumor, atypical meningioma, chondroma, chondrosarcoma, chordoma, calcified neurogenic tumor, teratoma, calcified astrocytoma, ganglioglioma

Notes:

EPIDURAL HEMATOMA OF BRAIN

=EXTRADURAL HEMATOMA = within potential space between naked inner table + calvarial periosteum (dura layer), which is bound down at suture margins*Incidence:* 2% of all serious head injuries; in <1% of all children with cranial trauma; uncommon in infants*Age:*more common in younger patients (dura more easily stripped away from skull)*Associated with:* (1)skull fracture in 75-85 -95% (best demonstrated on skull radiographs)◊Skull fractures frequently not visible in children!(2)subdural hemorrhage(3)contusion*Mechanism of injury:* (a)laceration of (middle) meningeal artery / vein adjacent to inner table from fracture of calvarium (91%)(b)avulsion of venous vessels from points of calvarial perforations(c)disruption of dural venous sinuses (transverse / superior sagittal sinus) due to diastatic fracture of lambdoid / coronal suture [major cause in younger children]*Time of presentation:*within first few days of injury (80%), 4-21 days (20%) ● transient loss of consciousness (= brief period of unconsciousness from concussion of brainstem) ● lucent interval (in <33%) ● somnolence (24-96 hours after accident) due to accumulation of epidural hematoma◊DANGEROUS because of focal mass effect + rapid onset (neurosurgical emergency unless small)! ● progressive deterioration of consciousness to coma ● focal neurologic signs: 3rd nerve palsy (sign of cerebral herniation), hemiparesis◊Most commonly clinically significant if located in temporoparietal region!◊Only a minority of skull fractures across the middle meningeal artery groove result in epidural hematomas!*Types:* Iacute epidural hematoma(58%) from arterial bleedingIIsubacute hematoma(31%)IIIchronic hematoma(11%) from venous bleedingLocation: (a)in 66% temporoparietal (most often from laceration of middle meningeal artery)(b)in 29% at frontal pole, parieto-occipital region, between occipital lobes, posterior fossa (most often from laceration of dural sinuses by fracture)◊NO crossing of sutures unless diastatic fracture of suture present!CT: √ fracture line in area of epidural hematoma√ expanding biconvex (lenticular = elliptical) extra-axial fluid collection (most frequent) = under high pressure√ usually does not cross suture lines√ fresh extravasating blood (30-50 HU) / coagulated blood (50-80 HU) in acute stage√ hematoma usually homogeneous / rarely inhomogeneously "swirled" (due to mixture of clotted + unclotted blood indicating active bleeding)√ mass effect ("compression cone effect") with effacement of gyri + sulci from:-epidural hematoma (57%)-hemorrhagic contusion (29%)-cerebral edematous swelling (14%)√ separation of venous sinuses / falx from inner table of skull◊The ONLY hemorrhage displacing falx / venous sinuses away from inner table!√ marked stretching of vessels√ signs of arterial injury (rare): contrast extravasation, arteriovenous fistula, middle meningeal artery occlusion, formation of false aneurysmMR: √ low intensity of fibrous dura mater allows differentiation of epidural from subdural blood in the late subacute phase (extracellular methemoglobin) with hyperintensity on T1WI + T2WIAngio: √ meningeal arteries displaced away from inner table of skull*Rx:*after surgical evacuation return of ventricular system to midline◊Epidural hematoma at another site may be unmasked following surgical decompression!*DDx:*Chronic subdural hematoma (may have similar biconvex shape, crosses suture lines, stops at falx, no associated skull fracture, no displaced dura on MRI)

Notes:

FIBROMUSCULAR DYSPLASIA
=nonatherosclerotic angiopathy of unknown pathogenesis*Incidence:*<1% of cerebral angiographies*Age:*2/3 >50 years; M:F = 1:9*Associated with:*brain ischemia (up to 50%), intracranial aneurysms (up to 30%), intracranial tumors (30%), bruits, traumaLocation:cervical + intracranial ICA (85%), vertebral artery (7%); both anterior + posterior circulation (8%); bilateral (60-65%)simultaneous involvement of renal / muscular arteries in 3%Angio: length of affected vessel from 0.5 cm to several cm*Types:* 1.Medial fibroplasia = fibromuscular hyperplasia (80%) string of beads = alternating zones of widening + narrowing tubular narrowing2.Intimal fibroplasia smooth concentric tubular narrowing (DDx: Takayasu arteritis, sclerosing arteritis, vessel spasm, arterial hypoplasia)3.Subadventitial hyperplasia4.Atypical fibromuscular dysplasia(= ? variant of intimal fibroplasia) web = smooth / corrugated mass involving only one wall of vessel + projecting into lumen (DDx: atherosclerotic disease, posttraumatic aneurysm) *Cx:*dissection (in 3%), macroaneurysm*Prognosis:*tends to remain stable / minimal progression*Rx:*only when symptoms progress

Notes:

GLIOBLASTOMA MULTIFORME
Most malignant form of all gliomas / astrocytomas; end stage of progressive severe anaplasia of preexisting Grade I / II astrocytoma (not from embryologic glioblasts) *Incidence:*most common primary brain tumor; 50% of all intracranial tumors; 1-2% of all malignancies; 20,000 cases per year*Age:*all ages; peak incidence at 65-75 years;M:F = 3:2; more frequently in whites*Genetics:*Turcot syndrome, neurofibromatosis type 1, Li-Fraumeni syndrome (familial neoplasms in various organs based on abnormal p53 tumor-suppressor gene)*Path:*multilobulated appearance; quite extensive vasogenic edema (transudation through structurally abnormal tumor vascular channels); deeply infiltrating neoplasm; hemorrhage; necrosis is essential for pathologic diagnosis (HALLMARK)*Histo:*highly cellular, often bizarrely pleomorphic / undifferentiated multipolar astrocytes; common mitoses + prominent vascular endothelial proliferation; no capsule; pseudopalisading(= viable neoplastic cells forming an irregular border around necrotic debris as the tumor outgrows its blood supply)*Subtypes:*(a)giant cell GBM = monstrocellular sarcoma(b)small cell GBM = gliosarcoma = Feigin tumorLocation: (a)hemispheric: white matter of centrum semiovale: frontal > temporal lobes; common in pons, thalamus, quadrigeminal region; relative sparing of basal ganglia + gray matter*DDx:*solitary metastasis, tumefactive demyelinating lesion ("singular sclerosis"), atypical abscess(b)callosal: "butterfly glioma" may grow exophytically into ventricle(c)posterior fossa: pilocytic astrocytoma, brainstem astrocytoma(d)extra-axial: primary leptomeningeal glioblastomatosis(e)multifocal: in 2-5%Spread: (a)direct extension following white matter tracts into corpus callosum (36%); readily crosses midline = "butterfly" glioma (clue: invasion of septum pellucidum); frontal + temporal gliomas tend to invade basal ganglia; may invade pia, arachnoid and dura (mimicking meningioma)(b)subependymal carpet after reaching the surface of the ventricles(c)via CSF (<2%)(d)hematogenous (extremely rare)√ osteoblastic bone lesionNECT: √ inhomogeneous low-density mass with irregular shape + poorly defined margins (hypodense solid tumor / cavitary necrosis / tumor cyst / peritumoral "fingers of edema")√ considerable mass effect: compression + displacement of ventricles, cisterns, brain parenchyma√ iso- / hyperdense portions (hemorrhage) in 5%√ rarely calcifies (if coexistent with lower-grade glioma / after radio- or chemotherapy)CECT: Enhancement pattern: contrast enhancement due to breakdown of blood-brain barrier / neovascularity / areas of necrosis (a)diffuse homogeneous enhancement(b)nonhomogeneous enhancement(c)ring pattern (occasionally enhancing mass within the ring)(d)low-density lesion with contrast fluid level (leakage of contrast)√ almost always ring blush of variable thickness: multiscalloped ("garland"), round / ovoid; may be seen surrounding ventricles (subependymal spread); tumor usually extends beyond margins of enhancement√ sedimentation level secondary to cellular debris / hemorrhage / accumulated contrast material in tumoral cystMR: √ poorly defined lesion with some mass effect / vasogenic edema / heterogeneity√ hemosiderin deposits (gradient echo images)√ hemorrhage (hypointensity on T2WI and T2*-WI)√ T1WI + gadolinium-DTPA enhancement separate tumor nodules from surrounding edema, central necrosis and cyst formationAngio: √ wildly irregular neovascularity + early draining veins√ avascular lesionPET: √ increase in glucose utilization rate*Rx:*surgery + radiation therapy + chemotherapy*Prognosis:*16-18 months postoperative survival (frequent tumor recurrence due to uncertainty during surgery about tumor margins)
Multifocal GBM (1)Spread of primary GBM(2)Multiple areas of malignant degeneration in diffuse low-grade astrocytoma ("gliomatosis cerebri")(3)Inherited / acquired genetic abnormality

Notes:

Gangliocytoma rare benign tumor *Incidence:*0.1%*Histo:*purely neuronal tumor (no glial components); ganglion cells without stain for glial fibrillary acetic protein

Notes:

Ganglioglioma glial component that may show neoplastic differentiation √ cyst formation + calcifications √ contrast enhancement

Notes:

GLIOMA
=malignant tumors of glial cells growing along white matter tracts, tendency to increase in grade with time; may be multifocal*Incidence:*30-40% of all primary intracranial tumors√ contrast enhancement:◊increases in proportion to degree of anaplasia◊diminished intensity of enhancement with steroid therapy
CELL OF ORIGIN 1.AstrocyteAstrocytoma2.OligodendrocyteOligodendroglioma3.EpendymaEpendymoma4. MedulloblastMedulloblastoma;(PNET = primitive neuroectodermal tumor) 5.Choroid plexusChoroid plexus papilloma
FREQUENCY OF INTRACRANIAL GLIOMAS Glioblastoma multiforme51%Astrocytoma25%Ependymoma6%Oligodendroglioma6%Spongioblastoma polare 3%Mixed gliomas 3%Astroblastoma 2%
*Age peak:*middle adult lifeLocation:cerebral hemispheres; spinal cord;brainstem + cerebellum (in children)

Brainstem Glioma Hypothalamic / Chiasmatic Glioma

Notes:

Brainstem Glioma *Incidence:*1%; 12-15% of all pediatric brain tumors; 20-30% of infratentorial brain tumors*Histo:*usually anaplastic astrocytoma / glioblastoma multiforme with infiltration along fiber tracts*Age:*in children + young adults; peak age 3-13 years; M:F = 1:1 ● become clinically apparent early before ventricular obstruction occurs ● ipsilateral progressive multiple cranial nerve palsies ● contralateral hemiparesis ● cerebellar dysfunction: ataxia, nystagmus ● eventually respiratory insufficiencyLocation:pons > midbrain > medulla; often unilateral at medullopontine junction◊Medullary + mesencephalic gliomas are more benign than pontine gliomas!Growth pattern: (a)diffuse infiltration of brainstem with symmetric expansion + rostrocaudal spread into medulla / thalamus + spread to cerebellum(b)focally exophytic growth into adjacent cisterns (cerebellopontine, prepontine, cisterna magna)√ asymmetrically expanded brainstem√ flattening + posterior displacement of 4th ventricle + aqueduct of Sylvius√ compression of prepontine + interpeduncular cistern (in upward transtentorial herniation)√ paradoxical widening of CP angle cistern with tumor extension into CP angle√ paradoxical anterior displacement of 4th ventricle with tumor extension into cisterna magnaCT: √ isodense / hypodense mass with indistinct margins√ hyperdense foci (= hemorrhage) uncommon√ absent / minimal / patchy contrast enhancement (50%)√ ring enhancement in necrotic / cystic tumors (most aggressive)√ prominent enhancement in exophytic lesion√ hydrocephalus uncommon (because of early symptomatology)MR: (better evaluation in subtle cases)√ hypointense on T1WI + hyperintense on T2WI√ ± engulfment of basilar arteryAngio: √ anterior displacement of basilar artery + anterior pontomesencephalic vein√ posterior displacement of precentral cerebellar vein√ posterior displacement of posterior medullary + supratonsillar segments of PICA√ lateral displacement of lateral medullary segment of PICA*Prognosis:*10-30% 5-year survival rate*Rx:*radiation therapy*DDx:*focal encephalitis, resolving hematoma, vascular malformation, tuberculoma, infarct, multiple sclerosis, metastasis, lymphoma

Notes:

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Hypothalamic / Chiasmatic Glioma Point of origin often undeterminable: hypothalamic gliomas invade chiasm, chiasmatic gliomas invade hypothalamus *Incidence:*10-15% of supratentorial tumors in children*Age:*2-4 years; M:F = 1:1*Associated with:* von Recklinghausen disease (20-50%) ● diminished visual acuity (50%) with optic atrophy ● diencephalic syndrome (in up to 20%): marked emaciation, pallor, unusual alertness, hyperactivity, euphoria ● obese child ● sexual precocity ● diabetes insipidus√ obstructive hydrocephalus√ suprasellar hypodense lobulated mass with dense inhomogeneous enhancement√ hypointense on T1WI + hyperintense on T2WI√ cyst formation, necrosis, calcifications render lesion inhomogeneous*DDx:*hypothalamic hamartoma

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Notes:

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GLOBOID CELL LEUKODYSTROPHY
=KRABBE DISEASE*Cause:*deficiency of galactosylceramide beta-galactosidase resulting in cerebroside accumulation + destruction of oligodendrocytes*Dx:*biochemical assay from white blood cells / skin fibroblasts*Age:*3-6 months ● restlessness + irritability ● marked spasticity ● optic atrophy ● hyperacusis√ symmetric hyperdense lesions in thalami, caudate nuclei, corona radiata√ decreased attenuation of white matter√ brain atrophy with enlargement of ventricles*Prognosis:*death within first few years of life

Notes:

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HALLERVORDEN-SPATZ DISEASE
rare metabolic disorder with abnormal iron retention in basal ganglia *Age:*2nd decade of life*Histo:*hyperpigmentation + symmetrical destruction of globus pallidus + substantia nigra ● progressive gait impairment + rigidity of limbs ● slowing of voluntary movements, dysarthria ● choreoathetotic movement disorder ● mental deteriorationCT: √ low- (= tissue destruction) / high-density (= dystrophic calcification) foci in globus pallidusMR: √ initially hypointense globus pallidus on T2WI (= iron deposition)√ later hyperintense foci on T2WI (= tissue destruction + gliosis)

Notes:

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HAMARTOMA OF CNS
rare tumor (a)sporadic(b)associated with tuberous sclerosis; may degenerate into giant cell astrocytoma *Age:*0-30 yearsLocation:temporal lobe, hamartoma of tuber cinereum, subependymal in tuberous sclerosis√ cyst with little mass effect, possibly with focal calcifications√ usually no enhancement

Notes:

HEAD TRAUMA

*Incidence:*0.2-0.3% annually in United States; peak at 550/100,000 people aged 15-24 years; second peak >50 years of age*Classification:* A.Primary traumatic lesion(a)primary neuronal injury1.Cortical contusion2.Diffuse axonal injury3.Subcortical gray matter injury= injury to thalamus ± basal ganglia 4.Primary brainstem injury(b)primary hemorrhages (from injury to a cerebral artery / vein / capillary)1.Subdural hematoma2.Epidural hematoma3.Intracerebral hematoma4.Diffuse hemorrhage (intraventricular, subarachnoid)(c)primary vascular injuries1.Carotid-cavernous fistula2.Arterial pseudoaneurysmLocation:branches of ACA + MCA, intra-cavernous portion of ICA, pCom3.Arterial dissection / laceration / occlusion4.Dural sinus laceration / occlusion(d)traumatic pia-arachnoid injury1.Posttraumatic arachnoid cyst2.Subdural hygroma(e)cranial nerve injuryB.Secondary traumatic lesion ● deterioration of consciousness / new neurologic signs some time after initial injury1.Major territorial arterial infarction*Cause:*prolonged transtentorial / subfalcine herniation pinching the artery against a rigid dural marginLocation:PCA, ACA territory2.Boundary + terminal zone infarction3.Diffuse hypoxic injury4.Diffuse brain swelling / edema5.Pressure necrosis from brain herniation*Cause:*increased intracranial pressureLocation:cingulate, uncal, parahippocampal gyri, cerebellar tonsils6.Secondary "delayed" hemorrhage7.Secondary brainstem injury (mechanical compression, secondary (Duret) hemorrhage in tegmentum of rostral pons + midbrain, infarction of median / paramedian perforating arteries, necrosis)8.Other (eg, fatty embolism, infection) ● **Duret hemorrhage** = hemorrhage in lateral brainstem due to massive temporal lobe herniation ● **Kernahorn notch** = contusion of contralateral brainstem caused by pressure of free edge of tentorium

Pathomechanism: A.Direct impact on brain due to fracture / skull distortion√ superficial neural damage localized to immediate vicinity of calvarial injury1.Cortical laceration due to depressed fracture fragment2.Epidural hematomaB.Indirect injury irrespective of skull deformation(a)compression-rarefaction strain = change in cell volume without change in shape (rare)(b)shear strain = change in shape without change in volume by-rotational acceleration forces (more common)√ bilateral multiple superficial / deep lesions possibly remote from the site of impact1.Cortical contusion (brain surface)2.Diffuse axonal injury (white matter)3.Brainstem + deep gray matter nuclei-linear acceleration forces (less common)1.Subdural hematoma2.Small superficial contusion

Intracerebral Hemorrhage Extracerebral Hemorrhage Other Posttraumatic Lesions

Notes:

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Intracerebral Hemorrhage 1.Hematoma=blood separating relatively normal neurons(a)shear-strain injury (most common)(b)blunt / penetrating trauma (bullet, ice pick, skull fracture fragment)*Incidence:*2-16% of trauma victimsLocation:low frontal + anterior temporal white matter / basal ganglia (80-90%) ● frequently no loss of consciousness ● development may be delayed in 8% of head injuries√ well-defined homogeneously increased density2.Cortical contusion=blood mixed with edematous brain√ poorly defined area of mixed high and low densities, may increase with time3.Intraventricular hemorrhage=potential complication of any intracranial hemorrhage◊For earliest detection focus on occipital horns!

Notes:

Extracerebral Hemorrhage 1.Subdural hematomain adults:dura inseparable from skull2.Epidural hematomain children:dura easily stripped away from skull3.Subarachnoid hemorrhagecommon accompaniment to severe cerebral trauma

Notes:

Other Posttraumatic Lesions 1.Pneumocephalus2.Penetrating foreign body
Indications for radiographic skull series: Only in conjunction with positive CT scan findings! 1.Evaluation of depressed skull fracture / fracture of base of the skull
Indications for CT: 1.Loss of consciousness (more than transient)2.Altered mental status during observation3.Focal neurologic signs4.Clinically suspected basilar fracture5.Depressed skull fracture (= outer table of fragment below level of inner table of calvarium) 6.Penetrating wound (eg, bullet)7.Suspected acute subarachnoid hemorrhage, epidural / subdural / parenchymal hematomaCT report addresses: √ midline shift√ localized mass effect√ distortion / effacement of basal, perimesencephalic, suprasellar, quadrigeminal cisterns√ pressure on brainstem, brainstem abnormality√ hemorrhage / contusion: extra-axial, intra-axial, subarachnoid, intraventricular√ edema: generalized / localized√ hydrocephalus√ presence of foreign bodies, bullet, bone fragments, air√ base of skull, face, orbit√ scalp swelling
Indications for MR: 1.Postconcussive symptomatology2.Diagnosis of small sub- / epidural hematoma3.Suspected diffuse axonal (shearing) injury, cortical contusion, primary brainstem injury4.Vascular damage (eg, pseudoaneurysm formation due to basilar skull fracture)
Sequelae of head injury: 1.Posttraumatic hydrocephalus (1/3)=obstruction of CSF pathways secondary to intracranial hemorrhage; develops within 3 months2.Generalized cerebral atrophy (1/3)=result of ischemia + hypoxia3.Encephalomalacia√ focal areas of decreased density, but usually higher density than CSF4.Pseudoporencephaly=CSF-filled space communicating with ventricle / subarachnoid space from cystic degeneration5.Subdural hygroma=localized collection of CSF in subdural space secondary to (a) result of chronic subdural hematoma (b) arachnoidal tear acting as a ball valve*Age:*most often in elderly + young children√ may resolve spontaneously6.Leptomeningeal cyst=progressive protrusion of leptomeninges through traumatic calvarial defect7.Cerebrospinal fluid leak ● rhinorrhea, otorrhea (indicating basilar fracture with meningeal tear)8.Posttraumatic abscesssecondary to (a) penetrating injury (b) basilar skull fracture (c) infection of traumatic hematoma 9.Parenchymal injurybrain atrophy, residual hemoglobin degradation products, wallerian-type axonal degeneration, demyelination, cavitation, microglial scarring *Prognosis:*up to 10% fatal; 5-10% with some degree of neurologic deficit*Mortality:*25/100,000 per year (traffic-related in 20-50%, gunshot 20-40%; falls)

Notes:

HEMANGIOBLASTOMA OF CNS

=benign autosomal dominant tumor of vascular origin*Incidence:*1-2.5% of all intracranial neoplasms*Age:*(a)adulthood (>80%): 20-50 years, average age of 33 years; M > F(b)childhood (<20%): in von Hippel-Lindau disease (10-20%); girls*Associated with:* (a)von Hippel-Lindau disease, may have multiple hemangioblastomas (only 20% of patients show other stigmata)(b)pheochromocytoma (often familial)(c)syringomyelia(d)spinal cord hemangioblastomas ● erythrocythemia in 20% (tumor elaborates stimulant)Location: paravermian cerebellar hemisphere > spinal cord > cerebral hemisphere / brainstem; multiple lesions in 10%√ solid (1/3) / cystic / cystic + mural nodule√ solid portion often intensely hemorrhagic√ almost never calcifiesCT: √ cystic sharply marginated mass of CSF-density (2/3)√ peripheral mural nodule with homogeneous enhancement (50%√ occasionally solid with intense homogeneous enhancementMR: √ well-demarcated tumor mass moderately hypointense on T1WI + T2WI√ hyperintense areas on T1WI (= hemorrhage)√ hypointense areas on T1WI + hyperintense areas on T2WI (= cyst formation)√ intralesional vermiform areas of signal dropout(= high-velocity blood flow) √ heterogeneous enhancement on Gd-DTPA with nonenhancing foci of cyst formation + calcification + rapidly flowing blood √ perilesional Gd-DTPA enhancing areas of slow-flowing blood vessels feeding + draining the tumor√ peripheral hyperintense rim on T2WI (= edema)Angio: √ densely stained tumor nidus within cyst ("contrast loading")√ staining of entire rim of cyst√ draining vein*DDx:*(1)Cystic astrocytoma (>5 cm, calcifications, larger nodule, thick-walled lesion, no angiographic contrast blush of mural nodule, no erythrocythemia)(2)Arachnoid cyst (if mural nodule not visualized)(3)Metastasis (more surrounding edema)

Notes:

HEMATOMA OF BRAIN

=INTRACEREBRAL HEMATOMA*Etiology:* A.Very common1.Chronic hypertension*Age:*>60 yearsLocation:external capsule and basal ganglia (putamen in 50%) / thalamus (25%), pons + brainstem (10%), cerebellum (10%), cerebral hemisphere (5%)2.Trauma3.Aneurysm4.AVMB.Common1.Hemorrhagic infarction = hemorrhagic transformation of stroke2.Amyloid angiopathy: elderly patients3.Coagulopathy4.Drug abuse: methamphetamines, cocaine5.Bleeding into tumor (eg, metastasis, glioma)C.Uncommon1.Venous infarction2.Eclampsia3.Septic emboli4.Vasculitis (especially fungal)5.Encephalitis

Stages of Cerebral Hematomas Basal Ganglia Hematoma

Notes:

Stages of Cerebral Hematomas Progression:hematoma gradually "snowballs" in size, dissects along white matter tracts; may decompress into ventricular system / subarachnoid spaceResolution:resorption from outside toward the center; rate depends on size of hematoma (usually 1-6 weeks)FALSE-NEGATIVE CT: 1.impaired clotting2.anemia√ iso- / hypodense stage

MR Appearance of Intracerebral Hematoma

Phase	Age	Compartment	Hemoglobin	T1	T2	Comments
hyperacute	<24 hr	intracellular	oxyhemoglobin	iso	hyper	hyperacute bleed in <1 hr
						deoxygenation
acute	1 – 3 d	intracellular	deoxyhemoglobin	hypo	hypo	within clotted intact hypoxic RBCs
		extracellular	deoxyhemoglobin	iso	iso	after lysis of RBCs
subacute						oxidation
early	>3 d	intracellular	methemoglobin	hyper	hypo	within intact RBCs inside retracting clot
late	>7 d	extracellular	methemoglobin	hyper	hyper	after lysis of RBCs
chronic	>14 d					
center		extracellular	hemichromes	iso	hyper	non–iron-containing heme pigments
rim		intracellular	hemosiderin	hypo	hypo	within macrophages, present for years
		fibrous tissue		hypo	hypo	
		edema		iso	hyper	

mnemonic:	"DD-BD-BB-DD on T1/T2"		
Dark-Dark	acute	0–2 days	deoxyhemoglobin
Bright-Dark	early subacute	3 – 7 days	intracellular methemoglobin
Bright-Bright	late subacute	8 – 14 days	extracellular methemoglobin
Dark-Dark	chronic	>14 days	hemosiderin

Hyperacute Hemorrhage *Time period:*<24 hoursSubstrate:fresh oxygenated arterial blood contains 95% diamagnetic (= no unpaired electrons) intracellular oxyhemoglobin (Fe^{2+}) with higher water contents than white matter; oxyhemoglobin persists for 6-12 hours)NCCT: √ homogeneous consolidated high-density lesion (50-70 HU) with irregular well-defined margins increasing in density during day 1-3 (hematoma attenuation dependent on hemoglobin concentration + rate of clot retraction)√ usually surrounded by low attenuation (edema, contusion) appearing within 24-48 hours(a)irregular shape in trauma(b)spherical + solitary in spontaneous hemorrhage√ less mass effect compared with neoplasmsMR (less sensitive than CT during first hours): √ little difference to normal brain parenchyma = center of hematoma iso- to hypointense on T1WI + minimally hyperintense on T2WI√ peripheral rim of hypointensity (= degraded blood products as clue for presence of hemorrhage)

Acute Hematoma *Time period:*1-3 daysSubstrate:paramagnetic (= 4 unpaired electrons) intracellular deoxyhemoglobin (Fe^{2+}); deoxyhemoglobin persists for 3 daysMR: √ slightly hypo- / isointense on T1WI (= paramagnetic deoxyhemoglobin within clotted intact hypoxic RBCs does not cause T1 shortening)√ very hypointense on T2WI (progressive concentration of RBCs, blood clot retraction, and fibrin production shorten T2)√ surrounding tissue isointense on T1WI / hyperintense on T2WI (edema)

Early Subacute Hematoma *Time period:*3-7 daysSubstrate:intracellular strongly paramagnetic (= 5 unpaired electrons) methemoglobin (Fe^{3+}); (inhomogeneously distributed within cells)NCCT: √ increase in size of hemorrhagic area over days / weeks√ high-density lesion within 1st week; often with layeringMR: √ very hyperintense on T1WI (= oxidation of deoxyhemoglobin to methemoglobin results in marked shortening of T1)(a)beginning peripherally in parenchymal hematomas(b)beginning centrally in partially thrombosed aneurysm (oxygen tension higher in lumen)*DDx:*melanin, high-protein concentration, flow-related enhancement, gadolinium-based contrast agent√ very hypointense on T2WI (= intracellular methemoglobin causes T2 shortening)

Late Subacute Hematoma *Time period:*7-14 daysSubstrate:extracellular strongly paramagnetic met-hemoglobin (homogeneously distributed)NCCT: √ gradual decrease in density from periphery inward (1-2 HU per day) during 2nd + 3rd weekCECT: √ peripheral rim enhancement at inner border of perilesional lucency (1-6 weeks after injury) in 80% (secondary to blood-brain barrier breakdown / luxury perfusion / formation of hypervascular granulation tissue)√ ring blush may be diminished by administration of corticosteroidsMR: √ hyperintense on T1WI (= RBC lysis allows free passage of water molecules across cell membrane)√ hyperintense on T2WI (= compartmentalization of methemoglobin is lost due to RBC lysis)√ surrounding edema isointense on T1WI + hyperintense on T2WI

Chronic Hematoma *Time period*span style="margin-top: 0; margin-left: 90pt">>14 days Substrate:superparamagnetic **ferritin** (= soluble + stored in intracellular compartment) and **hemosiderin** (= insoluble + stored in lysosomes) cause marked field inhomogeneitiesNCCT: √ isodense hematoma from 3rd-10th week with perilesional ring of lucencyCT: √ hypodense phase (4-6 weeks) secondary to fluid uptake by osmosis√ decreased density (3-6 months) / invisible√ after 10 weeks lucent hematoma (encephalomalacia due to proteolysis and phagocytosis + surrounding atrophy) with ring blush (DDx: tumor)MR: √ rim slightly hypointense on T1WI + very hypointense on T2WI (= superparamagnetic hemosiderin + ferritin within macrophages); rim gradually increases over weeks in thickness, eventually fills in entire hematoma = HALLMARK√ center hyperintense on T1WI + T2WI (= extracellular methemoglobin of lysed RBCs just inside the darker hemosiderin ring); present for months to 1 year √ surrounding hyperintensity on T2WI (= edema + serum extruded from clot) with associated mass effect should resorb within 4-6 weeks (DDx: malignant hemorrhage)
Prognosis:(1)herniation (if 3-4 cm in size)(2)death (if >5 cm in size)

Notes:

Basal Ganglia Hematoma =rupture of small distal microaneurysms in the lenticulostriate arteries in patients with poorly controlled systemic arterial hypertension*Cx:*(1)Dissection into adjacent ventricles (2/3)(2)Porencephaly(3)Atrophy with ipsilateral ventricular dilatation

Notes:

HETEROTOPIC GRAY MATTER
=collection of cortical neurons in an abnormal location secondary to arrest of migrating neuroblasts from ventricular walls to brain surface between 7-24 weeks of GA*Frequency:*3% of healthy population*May be associated with:* agenesis of corpus callosum, aqueductal stenosis, microcephaly, schizencephaly ● seizuresLocation: (1)nodular form: usually symmetric bilaterally in subependymal region / periventricular white matter with predilection for posterior + anterior horns(2)laminar form: deep / subcortical regions within white matter (less common)√ single / multiple bilateral subependymal nodules along lateral ventricles√ NO surrounding edema, isointense with gray matter on all sequences, no contrast enhancement*DDx:*subependymal spread of neoplasm, subependymal hemorrhage, vascular malformation, tuberous sclerosis, intraventricular meningioma, neurofibromatosis

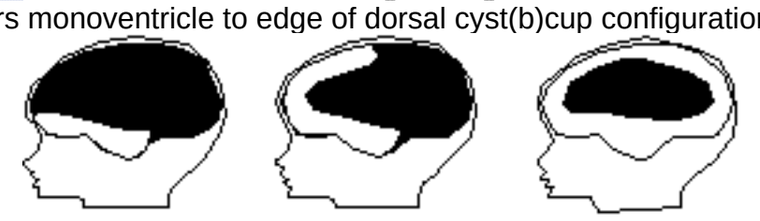
Notes:

HOLOPROSENCEPHALY
=lack of cleavage / diverticulation of the forebrain(= prosencephalon) laterally (cerebral hemispheres), transversely (telencephalon, diencephalon), horizontally (optic + olfactory structures) as a consequence of arrested lateral ventricular growth in 6-week embryo; cortical brain tissue develops to cover the monoventricle and fuses in the midline; posterior part of the monoventricle becomes enlarged and saclike⚱Septum pellucidum always absent!*Incidence:*1:16,000; M:F = 1:1A.ALOBAR=no hemispheric developmentB.SEMILOBAR=some hemispheric developmentC.LOBAR=frontal and temporal lobation + small monoventricle*Associated with:*polyhydramnios (60%), renal + cardiac anomalies; chromosomal anomalies (predominantly trisomy 13 + 18)Associated borderline syndromes secondary to diencephalic malformation: 1.Anophthalmia2.Microphthalmia3.Aplasia of pituitary gland4.Olfactogenital dysplasia5.Septo-optic dysplasia*DDx:* 1.Severe hydrocephalus (roughly symmetrically thinned cortex)2.Dandy-Walker cyst (normal supratentorial ventricular system)3.Hydranencephaly (frontal + parietal cortex most severely affected)4.Agenesis of corpus callosum with midline cyst (lateral ventricles widely separated with pointed superolateral margin)

Alobar Holoprosencephaly Semilobar Holoprosencephaly Lobar Holoprosencephaly

Notes:

Alobar Holoprosencephaly =extreme form in which the prosencephalon does not divide ● minimal motor activity, little sensory response (ineffective brain function); seizures ● severe facial anomalies ("the face predicts the brain"):1.Normal face in 17%2.Cyclopia (= midline single orbit); may have proboscis (= fleshy supraorbital prominence) + absent nose3.Ethmocephaly = 2 hypoteloric orbits + proboscis between eyes and absence of nasal structures4.Cebocephaly = 2 hypoteloric orbits + single nostril with small flattened nose + absent nasal septum5.Median cleft lip + cleft palate + hypotelorism6.Others: micrognathia, trigonocephaly (early closure of metopic suture), microphthalmia, microcephaly√ thalami fused√ protrusion of anteriorly placed fused thalami + basal ganglia into monoventricle√ absence of: septum pellucidum, 3rd ventricle, falx cerebri, interhemispheric fissure, corpus callosum, fornix, optic tracts, olfactory bulb (= arrhinencephaly), internal cerebral veins, superior + inferior straight sagittal sinus, vein of Galen, tentorium, sylvian fissure, opercular cortex√ crescent-shaped holoventricle = single large ventricle without occipital or temporal horns√ large dorsal cyst occupying most of calvarium + widely communicating with single ventricle√ "horseshoe" / "boomerang" configuration of brain= peripheral rim of cerebral cortex displaced rostrally (coronal plane)(a)pancake configuration = cortex covers monoventricle to edge of dorsal cyst(b)cup configuration =



more cortex visible posteriorly(c)ball configuration = complete covering of monoventricle without dorsal cyst √ midbrain, brainstem, cerebellum structurally normal√ pancakelike cerebrum in posterior cranium√ cerebral mantle pachygyric√ midline clefts in maxilla + palate*Prognosis:*death within 1st year of life / stillborn*DDx:*massive hydrocephalus, hydranencephaly

Notes:

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Semilobar Holoprosencephaly =intermediate form with incomplete cleavage ofprosencephalon (more midline differentiation + beginning of sagittal separation) ● mild facial anomalies: midline cleft lip + palate ● hypotelorism ● mental retardation√ single ventricular chamber with partially formed occipital horns + rudimentary temporal horns√ peripheral rim of brain tissue is several cm thick√ partially fused thalami anteriorly situated + abnormally rotated resulting in small 3rd ventricle√ absence of septum pellucidum + corpus callosum + olfactory bulb√ rudimentary falx cerebri + interhemispheric fissure form caudally with partial separation of occipital lobes√ incomplete hippocampal formation*Prognosis:*infants survive frequently into adulthood

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Notes:

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Lobar Holoprosencephaly =mildest form with two cerebral hemispheres + two distinct lateral ventricles◊May be part of septo-optic dysplasia! ● usually not associated with facial anomalies except for hypotelorism ● mild to severe mental retardation, spasticity, athetoid movements√ closely apposed bodies of lateral ventricles with distinct occipital + frontal horns√ mild dilatation of lateral ventricles√ colpocephaly√ unseparated frontal horns of angular squared shape + flatroof (on coronal images) due to dysplastic frontal lobes√ dysplastic anterior falx + interhemispheric fissure√ absence of septum pellucidum + sylvian fissures√ corpus callosum usually present √ hippocampal formation nearly normal√ basal ganglia + thalami may be fused / separated√ pachygyria (= abnormally wide + plump gyri), lissencephaly (= o gyri)*Prognosis:*survival into adulthood

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Notes:

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HYDATID DISEASE OF BRAIN

=canine tapeworm (Echinococcus granulosus) in sheep-and cattle-grazing areasLocation:liver (60%), lung (25%), CNS (2%) subcortical√ usually single, large round, sharply marginated smooth-walled hypodense cyst√ no significant surrounding edema; no rim enhancement√ development of daughter cysts (after rupture / following diagnostic puncture)

Notes:

HYDRANENCEPHALY
=liquefaction necrosis of cerebral hemispheres replaced by a thin membranous sac of leptomeninges in outer layer + remnants of cortex and white matter in inner layer, filled with CSF + necrotic debris*Incidence:*0.2% of infant autopsies*Etiology:*absence of supraclinoid ICA system (? vascular occlusion / infection with toxoplasmosis or CMV) = ultimate form of porencephaly ● seizures; respiratory failure; generalized flaccidity ● decerebrate state with vegetative existence√ normal skull size / macrocrania / microcrania√ complete filling of hemicranium with membranous sac√ absence of cortical mantle (inferomedial aspect of temporal lobe, inferior aspect of frontal lobe, occipital lobe may be identified in some patients)√ brainstem usually atrophic√ cerebellum almost always intact√ thalamic, hypothalamic, mesencephalic structures usually preserved + project into cystic cavity√ central brain tissue can be asymmetric√ choroid plexus present√ falx cerebri + tentorium cerebelli usually intact, may be deviated in asymmetric involvement, may be incomplete / absent*Prognosis:*not compatible with prolonged extrauterine life (no intellectual improvement from shunting)*DDx:*(1)Severe hydrocephalus (some identifiable cortex present)(2)Alobar holoprosencephaly (facial midline anomalies)(3)Schizencephaly (some spared cortical mantle)

Notes:

HYDROCEPHALUS

=excess of CSF due to imbalance of CSF formation + absorption resulting in increased intraventricular pressure*Pathophysiology:* A.Overproduction (rare)B.Impaired absorption1.Blockage of CSF flow within ventricular system, cisterna magna, basilar cisterns, cerebral convexities2.Blockage of arachnoid villi / lymphatic channels of cranial nerves, spinal nerves, adventitia of cerebral vessels

Compensated hydrocephalus = new equilibrium established at higher intracranial pressure due to opening of alternate pathways (arachnoid membrane / stroma of choroid plexus / extracellular space of cortical mantle = transependymal flow of CSF)

Skull film: signs of raised intracranial pressure A.YOUNG INFANT / NEWBORN√ increase in craniofacial ratio√ bulging of anterior fontanelle√ sutural diastasis√ macrocephaly + frontal bossing√ "hammered silver" appearance = prominent digital impressions (wide range of normals in 4-10 years of age)B.ADOLESCENT / ADULT (changes in sella turcica)√ atrophy of anterior wall of dorsum sellae√ shortening of the dorsum sellae producing pointed appearance√ erosion / thinning / discontinuity of floor of sella√ depression of floor of sella with bulging into sphenoid sinus√ enlargement of sella turcica*DDx:*osteoporotic sella (aging, excessive steroid hormone)

Signs favoring hydrocephalus over white matter atrophy: √ commensurate dilatation of temporal horn with lateral ventricles (most reliable sign)√ narrowing of ventricular angle (= angle between anterior / superior margins of frontal horns at level of foramen of Monro) due to concentric enlargement√ Mickey Mouse ears on axial scans√ enlargement of frontal horn radius (= widest diameter of frontal horns taken at 90° angle to long axis of frontal horn)√ rounding of frontal horn shape√ enlargement of ventricular system disproportionate to enlargement of cortical sulci (due to compression of brain tissue against skull + consequent sulcal narrowing)√ interstitial edema from transependymal flow of CSF√ periventricular hypodensity√ rim of prolonged T1 + T2 relaxation times surrounding lateral ventricles

Hydrocephalic distortion of ventricles + brain: √ atrial diverticulum = herniation of ventricular wall through choroidal fissure of ventricular trigone into supracerebellar + quadrigeminal cisterns√ dilatation of suprapineal recess expanding into posterior incisural space resulting in inferior displacement of pineal gland / shortening of tectum in rostral-caudal direction / elevation of vein of Galen√ enlargement of anterior recess of 3rd ventricle extending into suprasellar cistern

Obstructive Hydrocephalus Nonobstructive Hydrocephalus Congenital Hydrocephalus Infantile Hydrocephalus Normal Pressure Hydrocephalus

Notes:

Obstructive Hydrocephalus = obstruction to normal CSF flow + absorption
Communicating Hydrocephalus =EXTRAVENTRICULAR HYDROCEPHALUS= elevated intraventricular pressure secondary to blockade beyond the outlet of 4th ventricle within the subarachnoid pathways*Incidence:*38% of congenital hydrocephaly*Pathophysiology:* unimpeded CSF flow through ventricles, impeded CSF flow over convexities / impeded reabsorption by arachnoid villi *Cause:* subarachnoid hemorrhage (most common cause), meningeal carcinomatosis (medulloblastoma, germinoma, leukemia, lymphoma, adenocarcinoma), purulent / tuberculous meningitis, subdural hematoma, craniosynostosis, achondroplasia, Hurler syndrome, venous obstruction (obliteration of superior sagittal sinus), absence of Pacchioni granulations √ symmetric enlargement of lateral, 3rd, and often 4th ventricles√ dilatation of subarachnoid cisterns√ normal / effaced cerebral sulci√ symmetric low attenuation of periventricular white matter (transependymal CSF flow)√ delayed ascent of radionuclide tracer over convexities√ persistence of radionuclide tracer in lateral ventricles for up to 48 hoursChanges after successful shunting: √ diminished size of ventricles + increased prominence of sulci√ cranial vault may thicken*Cx:*subdural hematoma (result from precipitous decompression)
Noncommunicating Hydrocephalus =INTRAVENTRICULAR HYDROCEPHALUS=blockade of CNS flow within the ventricular system with dilatation of ventricles proximal to obstruction*Pathogenesis:*increased CSF pressure causes ependymal flattening with breakdown of CSF-brain barrier leading to myelin destruction + compression of cerebral mantle (brain damage)Location: (a)Lateral ventricular obstruction*Cause:*ependymoma, intraventricular glioma, meningioma(b)Foramen of Monro obstruction*Cause:*3rd ventricular colloid cyst, tuber, papilloma, meningioma, septum pellucidum cyst / glioma, fibrous membrane (post infection), giant cell astrocytoma(c)Third ventricular obstruction*Cause:*large pituitary adenoma, teratoma, craniopharyngioma, glioma of 3rd ventricle, hypothalamic glioma(d)Aqueductal obstruction*Cause:*Congenital web / atresia (often associated with Chiari malformation), fenestrated aqueduct, tumor of mesencephalon / pineal gland, tentorial meningioma, S/P intra-ventricular hemorrhage or infection(e)Fourth ventricular obstruction*Cause:*Congenital obstruction, Dandy-Walker syndrome, inflammation (TB), tumor within 4th ventricle (ependymoma), extrinsic compression of 4th ventricle (astrocytoma, medulloblastoma, large CPA tumors, posterior fossa mass), isolated / trapped 4th ventricle
√ enlarged lateral ventricles (enlargement of occipital horns precedes enlargement of frontal horns)√ effaced cerebral sulci√ periventricular edema with indistinct margins (especially frontal horns)√ radioisotope cisternography: no obstruction if tracer reaches ventricle√ change in RI indicates increased intracranial pressure (→RI 47-132% versus 3-29% in normals)

Notes:

Nonobstructive Hydrocephalus =secondary to rapid CSF production*Cause:*Choroid plexus papilloma√ ventricle near papilloma enlarges√ intense radionuclide uptake in papilloma√ enlarged anterior / posterior choroidal artery and blush

Notes:

Congenital Hydrocephalus =multifactorial CNS malformation during the 3rd / 4th week after conception*Etiology:* (1) aqueductal stenosis (43%) (2) communicating hydrocephalus (38%) (3) Dandy-Walker syndrome (13%) (4) other anatomic lesions (6%) (a)Genetic factors: spina bifida, aqueductal stenosis (X-linked recessive trait with a 50% recurrence rate for male fetuses), congenital atresia of foramina of Luschka and Magendie (Dandy-Walker syndrome; autosomal recessive trait with 25% recurrence rate), cerebellar agenesis, cloverleaf skull, trisomy 13-18(b)Nongenetic etiology: tumor compressing 3rd / 4th ventricle, obliteration of subarachnoid pathway due to infection (syphilis, CMV, rubella, toxoplasmosis), proliferation of fibrous tissue (Hurler syndrome), Chiari malformations, vein of Galen aneurysm, choroid plexus papilloma, vitamin A intoxication*Incidence:*0.3-1.8:1,000 pregnancies*Associated with:* (a)Intracranial anomalies (37%): hypoplasia of corpus callosum, encephalocele, arachnoid cyst, arteriovenous malformation(b)extracranial anomalies (63%): spina bifida in 25-30% (with spina bifida hydrocephalus is present in 80%), renal agenesis, multicystic dysplastic kidney, VSD, tetralogy of Fallot, anal agenesis, malrotation of bowel, cleft lip / palate, Meckel syndrome, gonadal dysgenesis, arthrogryposis, sirenomelia(c)chromosomal anomalies (11%): trisomy 18 + 21, mosaicism, balanced translocation ● elevated amniotic alpha-fetoprotein levelOB-US: (assessment difficult prior to 20 weeks GA as ventricles ordinarily constitute a large portion of cranial vault) √ "dangling choroid plexus sign" = choroid plexus not touching medial + lateral walls of lateral ventricles with downside choroid falling away from medial wall + upside choroid falling away from lateral wall√ lateral width of ventricular atrium ≥10 mm (size usually constant between 16 weeks MA and term)❁88% of fetuses with sonographically detected neural axis anomalies have atrial width >10 mm√ BPD >95th percentile (usually not before third trimester)√ polyhydramnios (in 30%)*Recurrence rate:*<4%*Mortality:*(1) fetal death in 24%(2) neonatal death in 17% *Prognosis:*poor with(1) associated anomalies (2) shift of midline (porencephaly) (3) head circumference >50 cm (4) absence of cortex (hydranencephaly) (5) cortical thickness <10 mm

Notes:

Infantile Hydrocephalus ● ocular disturbances: paralysis of upward gaze, abducens nerve paresis, nystagmus, ptosis, diminished pupillary light response ● spasticity of lower extremities (from disproportionate stretching of paracentral corticospinal fibers)*Etiology: mnemonic:* "**A VP**-**S**hunt **C**an **D**ecompress **T**he **H**ydrocephalic **C**hild"**A**queductal stenosis **V**ein of Galen aneurysm **P**ostinfectious **S**uperior vena cava obstruction **C**hiari II malformation **D**andy-Walker syndrome **T**umor **H**emorrhage **C**horoid plexus papilloma

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Notes:

Normal Pressure Hydrocephalus =NPH = ADAM SYNDROME=pressure gradient between ventricle + brain parenchyma in spite of normal CSF pressure*Cause:*communicating hydrocephalus with incomplete arachnoidal obstruction from neonatal intraventricular hemorrhage, spontaneous subarachnoid hemorrhage, intracranial trauma, infection, surgery, carcinomatosis*mnemonic:*"PAM the HAM"**P**aget disease **A**neurysm **M**eningitis **H**emorrhage (from trauma) **A**chondroplasia **M**ucopolysaccharidosis *Pathophysiology of CSF:* (?) brain pushed toward cranium from ventricular enlargement; brain unable to expand during systole thus compressing lateral + 3rd ventricles + expressing large CSF volume through aqueduct; reverse dynamic during diastole; "water-hammer" force of recurrent ventricular expansion damages periventricular tissues *Age:*50-70 years ● normal opening pressure at lumbar puncture ● dementia, gait apraxia, incontinence*mnemonic:*wacky, wobbly and wet√ communicating hydrocephalus with prominent temporal horns√ ventricles dilated out of proportion to any sulcal enlargement√ upward bowing of corpus callosum√ flattening of cortical gyri against inner table of calvarium (DDx: rounded gyri in generalized atrophy)MR: √ pronounced aqueductal flow void (due to diminished compliance of normal pressure hydrocephalus)√ periventricular hyperintensity (due to transependymal CSF flow)*Rx:*CSF shunting (only 50% improved)

Notes:

HYPOTHALAMIC HAMARTOMA
=HAMARTOMA OF TUBER CINEREUM=rare congenital malformation composed of normal neuronal tissue arising from posterior hypothalamus in region of tuber cinereum*Age:*<2 years of age; M > F*Histo:*heterotopic collection of neurons, astrocytes, oligodendroglial cells (closely resembling histologic pattern of tuber cinereum) ● isosexual precocious puberty (due to LRH secretion) ● gelastic seizures, hyperactivity ● neurodevelopmental delayLocation:mamillary bodies / tuber cinereum of thalamus, rarely within hypothalamus itself√ well-defined round / oval mass projecting from base of brain into suprasellar / interpeduncular cistern√ attached to tuber cinereum / mamillary bodies by thin stalk (pedunculated)√ remain stable in size over time; up to 4 cm in diameterCT: √ round homogeneous mass isodense with brain tissue√ NO enhancementMR: √ well-defined round pedunculated mass suspended from tuber cinereum / mamillary bodies√ isointense on T1WI + iso- / slightly hyperintense on T2WI (imaging characteristics of gray matter)√ no gadolinium-enhancement

Notes:

IDIOPATHIC INTRACRANIAL HYPERTENSION
=PSEUDOTUMOR CEREBRI = BENIGN INTRACRANIAL HYPERTENSION (BIH)secondary to (a) elevation in blood volume (85%) (b) decrease in regional cerebral blood flow with delayed CSF absorption (10%) *Etiology:* 1.Sinovenous occlusive disease, SVC occlusion, obstruction of dural sinus, obstruction of both internal jugular veins2.Dural AVM3.S/P brain biopsy with edema4.Endocrinopathies5.Hypervitaminosis A6.Hypocalcemia7.Menstrual dysfunction, pregnancy, menarche, birth control pills8.Drug therapy*Predilection for:* obese young to middle-aged women ● headache ● papilledema ● elevated opening pressures on lumbar puncture√ normal ventricular size / pinched ventricles√ increased volume of subarachnoid space

Notes:

INFARCTION OF BRAIN

=brain cell death leading to coagulation necrosis*Pathophysiology:* distal microstasis occurs within 2 minutes after occlusion of cerebral artery; regional cerebral blood flow is acutely decreased in area of infarction + remains depressed for several days at center of infarct; arterial circulation time may be prolonged in entire hemisphere; rapid development of vasodilatation due to hypoxia, hypercapnia, tissue acidosis; delayed filling + emptying of arterial channels in area of infarction (= arteriolar-capillary block) well into venous phase; by end of 1st week regional blood flow commonly increases to rates even above those required for metabolic needs (= hyperemic phase = luxury perfusion) *Detection rate by CT:* 80% for cortex + mantle, 55% for basal ganglia, 54% for posterior fossa positive correlation between degree of clinical deficit and CT sensitivityCT sensitivity: on day of ictus:48%1-2 days later:59%7-10 days later:66%10-11 days later:74%Location:cerebrum:cerebellum = 19:1;(a)supratentorial-cerebral mantle (70%) in territory of MCA (50%), PCA (10%), watershed between MCA + ACA (7%), ACA (4%)-basal ganglia + internal capsule (20%)(b)infratentorial (10%)upper cerebellum (5%), lower cerebellum (3%), pons + medulla (2%)

Hyperacute Ischemic Infarction Acute Ischemic Infarction Subacute Ischemic Infarction Chronic Ischemic Infarction Hemorrhagic Infarction Basal Ganglia Infarct Laminar Necrosis Lacunar Infarction TIA and RIND

Notes:

Hyperacute Ischemic Infarction *Time period:*<12 hoursCT: √ normal (in 10-60%)√ "hyperdense artery sign" = acute intraluminal thrombus (25-50% of acute MCA occlusions)√ obscuration of lentiform nucleus (50-80% of MCA occlusions)√ calcified intraluminal embolus (rare)MR (more sensitive than CT): √ parenchymal swelling due to cytotoxic edema(= increased intracellular water) can be seen by 2 hours post ictus (best on T1WI)NUC: √Newer imaging agents (eg, Tc-99m HM-PAO) may be positive within minutes of the event, while CT and MR are normal√ hemispheric hypoperfusion throughout all phases√ defect corresponding to nonperfused vascular territory√ "flip-flop sign" in radionuclide angiogram (15%)= decreased uptake during arterial + capillary phase followed by increased uptake during venous phase √ "luxury perfusion syndrome" (14%) = increased perfusion

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Notes:

Acute Ischemic Infarction *Histo:*cortical cytotoxic edema (from loss of vascular autoregulation) followed by white matter vasogenic edema(a)Substage I (12-24 hours)NCCT: √ low-density basal ganglia√ effacement of gray-white matter junction, eg, "insular ribbon sign" = hypodense extreme capsule no longer distinguishable from insular cortex)√ subtle sulcal effacement (8%)CECT: √ no iodine accumulation in affected cortical regionMR (routinely positive by 4-6 hours post ictus): √ subtle narrowing of sulci√ increase in thickness of cortex (= gyral swelling)√ blurring of gray-white matter junction on T2- and proton-density images√ contrast-enhanced cortical arterial vessels in area of brain injury (due to slow arterial blood flow provided by collateral circulation via leptomeningeal anastomoses)√ subtle low-signal intensity on T1WI, high-signal intensity on T2WI (masking of gyral infarcts on heavily T2WI due to sulcal CSF intensity)MRA: √ absence of flow for infarcts >2 cm in diameter(b)Substage II (1-7 days)NCCT: √ hypodense wedge-shaped lesion with base at cortex in a vascular distribution (in 70%) due to vasogenic + cytotoxic edema √ mass effect (23-75%): sulcal effacement, transtentorial herniation, displaced subarachnoid cisterns + ventricles√ "bland infarct" may be transformed into hemorrhagic infarct after 2-4 days (due to leakage of blood from ischemically damaged capillary endothelium following lysis of intraluminal clot + arterial reperfusion)CECT: √ gyral enhancement along cortexMR: √ intravascular enhancement sign (77%)= Gd-pentetate enhancement of vessels supplying infarct after 1-3 days √ meningeal enhancement sign = Gd-pentetate enhancement of meninges adjacent to infarct after 2-6 daysAngio: √ narrowed / occluded vessels supplying the area of infarction√ delayed filling + emptying of involved vessels√ early draining vein√ luxury perfusion of infarcted area (rare) = loss of small vessel autoregulation due to local increase in pH

Notes:

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Subacute Ischemic Infarction *Time period:*7-30 days = paradoxical phase with resolution of edema + onset of coagulation necrosisNCCT: √ "fogging phenomenon" = low-density area less apparent√ decrease of mass effect + ex vacuo dilatation of ventricles (in 57%)√ ± transient calcification (especially in children)CECT: √ gyral blush + ring enhancement (breakdown of blood-brain barrier + luxury perfusion) for 2-8 weeks (in 65-80% within first 4 weeks)√ no enhancement in 1/5 of patientsMR: *Histo:*vasogenic edema (= increased extracellular water) due to disruption of blood-brain barrier√ hypointense on T1WI, hyperintense on T2WI√ gyriform parenchymal Gd-pentetate enhancement◊Gyriform parenchymal enhancement permits differentiation of subacute from chronic infarction!

Notes:

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Chronic Ischemic Infarction *Time period:*>30 days*Histo:*demyelination + gliosis complete (focal brain atrophy after 8 weeks)√ cerebral atrophy + encephalomalacia + gliosis (HALLMARKS)NCCT: √ cystic foci of CSF-density (= encephalomalacia) in vascular distributionMR: √ patchy region with increased intensity on T2WI√ gliosis (hyperintense on T2WI) often surrounding encephalomalacic region√ wallerian degeneration (= anterograde degeneration of axons secondary to neuronal injury) of corticospinal tracts in the wake of old large infarcts that involve the motor cortex

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Notes:

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Hemorrhagic Infarction *Etiology:*lysis of embolus / opening of collaterals / restoration of normal blood pressure following hypotension / hypertension / anticoagulation causes extravasation in reperfused ischemic brain*Incidence:*6% of clinically diagnosed brain infarcts, 20% of autopsied brain infarcts*Path:*petechial hemorrhages in various degrees of coalescenceLocation:corticomedullary junctionCT: √ hyperdensity appearing within a previously imaged hypodense acute ischemic infarct = hemorrhagic transformation (in 50-72%)MR: √ hypointense area on T2WI within edema marking gyri = deoxyhemoglobin of acute hemorrhage√ hyperintense area on T1WI = methemoglobin of subacute hematoma

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Notes:

Basal Ganglia Infarct =occlusion of small penetrating arteries at base of brain (lenticulostriate / thalamoperforating arteries)= lacunar infarct (infarcts <1 cm in size)
Cause: (1) Embolism (2) Hypoperfusion (3) Carbon monoxide poisoning (4) Drowning (5) Vasculopathy (hypertension, microvasculopathy, aging) √ dense homogeneous enhancement outlining caudate nucleus, putamen, globus pallidus, thalamus √ dense round nodular enhancement / peripheral ring enhancement

Notes:

Laminar Necrosis =ischemic changes affecting deep layers of the cortex (layers 3, 5, 6 very sensitive to oxygen deprivation)MR: (a)acute stage√ linear cortical hyperintensity on T1WI√ contrast enhancement√ white matter edema on T2WI(b)chronic stage√ thin hypointense cortex√ hyperintense white matter√ enlargement of CSF spaces

Notes:

Lacunar Infarction =small deep infarcts in the distal distribution of penetrating vessels (lenticulostriate, thalamoperforating, pontine perforating arteries, recurrent artery of Heubner)*Cause:*occlusion of small penetrating end arteries at base of brain due to fibrinoid degeneration*Predisposed:*hypertensive / diabetic patients*Incidence:*20% of cerebral infarctions*Path:*lacune = cavitated infarct resulting in small hole traversed by cobweblike fibrous strandsHisto:"microatheroma" = hyalinization + arteriolar sclerosis resulting in thickening of vessel wall + luminal narrowing ● pure motor / pure sensory stroke ● ataxic hemiparesisLocation:upper two-thirds of putamen > caudate > thalamus > pons > internal capsule√ small discrete foci of hypodensity between 3 mm and 15 mm in size (most <1 cm in diameter) √ higher in signal intensity than CSF (due to marginal gliosis)√ unilateral pontine infarcts are sharply marginated at midline

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Notes:

TIA and RIND √ hypodense small lesions located peripherally near / within cortex without enhancement√ lesions detected in only 14%, contralateral lesion present in 14% (CT of marginal value)

Notes:

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INIENCEPHALY
=complex developmental anomaly characterized by (1) exaggerated lordosis (2) rachischisis (3) imperfect formation of skull base at foramen magnumM:F = 1:4
Associated with other anomalies in 84%: anencephaly, encephalocele, hydrocephalus, cyclopia, absence of mandible, cleft lip / palate, diaphragmatic hernia, omphalocele, gastroschisis, single umbilical artery, CHD, polycystic kidney disease, arthrogryposis, clubfoot √ dorsal flexion of head√ abnormally short + deformed spine*Prognosis:*almost uniformly fatal*DDx:*(1) Anencephaly(2) Klippel-Feil syndrome (3) Cervical myelomeningocele

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Notes:

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INTRAVENTRICULAR NEUROCYTOMA
=INTRAVENTRICULAR NEUROBLASTOMA=benign primary neoplasm of lateral + 3rd ventricles*Incidence:*unknown; tumor frequently mistaken for intraventricular oligodendroglioma*Age:*20-40 years*Histo:*uniform round cells with central round nucleus + fine chromatin stippling ± perivascular pseudorosettes, focal microcalcifications (closely resembling oligodendroglioma but with neuronal differentiation into synapselike junctions)Location:body ± frontal horn of lateral ventricle, may extend into 3rd ventricle√ entirely intraventricular well-circumscribed tumor, coarsely calcified (69%), containing cystic spaces (85%)√ mild to moderate contrast enhancement√ attachment to septum pellucidum CHARACTERISTIC√ ± hemorrhage into tumor / ventricle√ hydrocephalus√ peritumoral edema extremely uncommonMR: √ isointense relative to cortical gray matter on T1WI + T2WI with heterogeneous areas due to calcifications, cystic spaces, vascular flow voids (62%)*Rx:*complete surgical resection*DDx:* (1)Intraventricular oligodendroglioma (no hemorrhage)(2)Astrocytoma (peritumoral edema in 20%)(3)Meningioma (almost exclusively in trigone, >30 years of age)(4)Ependymoma (in + around 4th ventricle / trigone, in childhood)(5)Subependymoma (in + around 4th ventricle, young adults)(6)Choroid plexus papilloma (body + posterior horn of lateral ventricle, intense enhancement, younger patient)(7)Colloid cyst (anterior 3rd ventricle / foramen of Monroe, calcifications uncommon)(8)Craniopharyngioma (extraventricular origin)(9)Teratoma + dermoid cyst (fat attenuation)

Notes:

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JAKOB-CREUTZFELDT DISEASE
=rare transmissible disease developing over weeks*Cause:*"prion" = protein devoid of functional nucleic acid; ? slow-virus infection*Age:*older adults*Histo:*classified as "spongiform encephalopathy" ● rapidly progressive dementia, ataxia, myoclonus√ hyperintense lesions in head of caudate nucleus + putamen, bilaterally on T2WI√ NO gadolinium-enhancement of lesions√ NO white matter involvement*Prognosis:*usually fatal within 1 year of onset

Notes:

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JOUBERT SYNDROME

● episodic hyperpnea ● abnormal eye movement ● ataxia, mental retardation*Path:*(1)nearly total aplasia of cerebellar vermis(2)dysplasia + heterotopia of cerebellar nuclei(3)near total absence of pyramidal decussation(4)anomalies in structure of inferior olivary nuclei, descending trigeminal tract, solitary fascicle, dorsal column nuclei√ 4th ventricle triangle-shaped at mid-level + bat-wing-shaped superiorly√ cerebellar hemispheres appose one another in midline√ superior cerebellar peduncles surrounded by CSF

Notes:

LIPOMA
=congenital tumor developing within subarachnoid space as a result of abnormal differentiation of the meninx primitiva (which differentiates into pia mater, arachnoid, inner meningeal layer of dura mater)*Incidence:*<1% of brain tumors*Age:*presentation in childhood / adulthood*Associated with congenital anomalies:* (a)in anterior location: various degrees of agenesis of corpus callosum (in 50-80%)(b)in posterior location (in <33%) ● asymptomatic in 50%Location: (usually in subarachnoid space) callosal cistern (25-50%), sylvian fissure, quadrigeminal cistern, chiasmatic cistern, interpeduncular cistern, CP angle cistern, cerebellomedullary cistern, tuber cinereum, choroid plexus of lateral ventricle CT: √ well-circumscribed mass with CT density of -100 HU√ occasionally calcified rim (esp. in corpus callosum)√ no enhancementMR: √ hyperintense mass on T1WI + less hyperintense on T2WI (CHARACTERISTIC)

Lipoma of Corpus Callosum

Notes:

Lipoma of Corpus Callosum =congenital pericallosal tumor not actually involving the corpus callosum as a result of faulty disjunction of neuroectoderm from cutaneous ectoderm during process of neurulation*Incidence:*approx. 30% of intracranial lipomas*Associated with:* (1)anomalies of corpus callosum (30% with small posterior lipoma, 90% with large anterior lipoma)(2)frontal bone defect (frequent) = encephalocele(3)cutaneous frontal lipoma ● in 50% symptomatic: ● seizure disorders, mental retardation, dementia ● emotional lability, headaches ● hemiplegiaPlain film: √ midline calcification with associated lucency of fat densityCT: √ area of marked hypodensity immediately superior to lateral ventricles with possible extension inferiorly between ventricles / anteriorly into interhemispheric fissure√ curvilinear peripheral / nodular central calcification within fibrous capsule (more common in anterior compared with posterior lipomas)MR: √ hyperintense midline mass superior + posterior to corpus callosum on T1WI√ no callosal fibers dorsal to lipoma√ branches of pericallosal artery frequently course through lipoma*DDx:*dermoid (denser, extra-axial), teratoma

Notes:

LISSENCEPHALY
="smooth brain" = AGYRIA-PACHYGYRIA COMPLEX=most severe of neuronal migration anomalies; autosomal recessive disease with abnormal cortical stratification**agyria** = absence of gyri on brain surface Pachygyria**pachygyria** = focal / diffuse area of few broad flat gyri
A.COMPLETE LISSENCEPHALY = AGYRIAmost frequently parieto-occipital in location B.INCOMPLETE LISSENCEPHALY=areas of both agyria + pachygyria, pachygyric areas most frequently in frontal + temporal regions
*Histo:*thick gray + thin white matter with only four cortical layers I, III, V, VI (instead of six layers)*Often associated with:* (1)CNS anomalies: microcephaly, hydrocephalus, agenesis of corpus callosum, hypoplastic thalami(2)micromelia, clubfoot, polydactyly, camptodactyly, syndactyly, duodenal atresia, micrognathia, omphalocele, hepatosplenomegaly, cardiac + renal anomalies ● micrencephaly ● severe mental retardation ● hypotonia + occasional myoclonic spasm ● early seizures refractory to medication√ smooth thickened cortex with diminished white matter√ figure-eight appearance of cerebrum on axial images due to shallow widened vertically oriented sylvian fissures√ absent / shallow sulci and gyri (brain looks similar to that in fetuses <23 weeks GA)√ middle cerebral arteries close to inner table of calvarium (absence of sulci)√ small splenium + absent rostrum of corpus callosum√ hypoplastic brainstem (lack of formation of corticospinal + corticobulbar tracts)√ ventriculomegaly (atrium + occipital horns)√ midline round calcification in area of septum pellucidum (CHARACTERISTIC)√ polyhydramnios (50%)*Prognosis:*death by age 2*DDx:*Polymicrogyria (= formation of multiple small gyri mimicking pachygyria on CT + MR, most common around sylvian fissures, broad thickened gyri with frequent gliosis subjacent to polymicrogyric cortex as the most important differentiating feature)

Notes:

LYMPHOID HYPOPHYSITIS
=rare inflammatory autoimmune disorder with lymphocytic infiltration of pituitary gland*Associated with:*thyrotoxicosis + hypopituitarism*Age:*almost exclusively in early postpartum women ● headaches, vision loss, inability to lactate / to resume normal menses ● enlarged homogeneously enhancing pituitary gland*Prognosis:*spontaneous regression*Rx:*steroids (reduction in pituitary size on follow-up)

Notes:

LYMPHOMA

A.PRIMARY LYMPHOMA (93%) = RETICULUM CELL SARCOMA = HISTIOCYTIC LYMPHOMA = MICROGLIOMAincreased incidence (350-fold) in immunocompromised patients: AIDS, renal transplant, Wiskott-Aldrich syndrome, immunoglobulin deficiency A, rheumatoid arthritis, progressive multifocal leukoencephalopathy*Associated with:*intraocular lymphomaB.SECONDARY (7%) = SYSTEMIC LYMPHOMALocation:tendency for dura mater + leptomeninges ● palsies of cranial nerves III, VI, VII◊Primary lymphoma is indistinguishable from secondary!Clues:(1) multicentric involvement of deep hemispheres(2) association with immunosuppression(3) rapid regression with corticosteroids / radiation therapy = "ghost tumor"*Prevalence:*0.3-2% of all intracranial tumors; 7-15% of all primary brain tumors (equivalent to meningioma + low-grade astrocytoma); M > F◊Only 0.8% of lymphomas are primary CNS lesions*Peak age:*30-50 years; M:F = 2:1*Histo:*atypical pleomorphic B-cells mixed with reactive T-cells infiltrate blood vessel walls + cluster within perivascular (Virchow-Robin) spaces simulating vasculitis ● symptoms of rapidly enlarging mass (60%) ● symptoms of encephalitis (<25%) ● stroke (7%) ● cranial nerve palsy, demyelinating disease ● personality changes, headaches, seizures ● cerebellar signs, motor dysfunction ● CSF cytology positive in 4-25-43%: elevated protein, mononuclear / blast / other lymphoma cellsLocation:suapratentorial:posterior fossa = 3-9:1;paramedian structures preferentially affected; white matter + corpus callosum (55%), deep central gray matter of basal ganglia + thalamus + hypothalamus (17%), posterior fossa + cerebellum (11%), spinal cord (1%); multicentricity in 11-47%Site:tendency to abut ependyma + meninges (12-30%); "butterfly pattern" of frontal lobe lymphoma; dural involvement may mimic meningioma (rare)Spread:typically infiltrating; may cross anatomic boundaries + midline, diffuse leptomeningeal spread; subependymal spread + ventricular encasement
√ commonly large discrete solitary lesion (57%)◊Large lesion suggests lymphoma!√ small + symmetric multiple nodular lesions (43-81%)√ diffusely infiltrating lesion with blurred margins√ usually mildly hyperdense (33%) / occasionally isodense / low-density area (least common)√ little mass effect with significant peritumoral edema √ homogeneously dense + well-defined / irregular + patchy periventricular contrast enhancement√ commonly thick-walled ring enhancement√ spontaneous regression (unique feature)MR (superior to CT): √ well-demarcated round / oval / gyral-shaped (rare) mass√ relatively little mass effect for size√ isointense / slightly hypointense relative to gray matter on T1WI√ hypo- to isointense / hyperintense (less common) relative to gray matter on T2WI√ ring pattern (= central necrosis with densely cellular rim in hyperintense "sea of edema") typical in immunocompromised patients√ intense ring-shaped contrast enhancement on T1WI√ irregular sinuous / gyral-like contrast enhancement or homogeneous enhancement√ solid homogeneous enhancement in immunocompetent patient√ irregular heterogeneous ringlike mass in immunocompromised patient√ periventricular enhancement is highly SPECIFIC (DDx: CMV ependymitis)Angio: √ avascular mass / tumor neovascularity√ focal blush in late arterial-to-capillary phase persisting well into venous phase√ arterial encasement√ dilated deep medullary veinsNUC: √ increased uptake of C-11 methionine on PET√ increased uptake of thallium-201 on SPECT*Prognosis:*median survival of 45 days for AIDS patients; median survival of 3.3 months for immuno-competent patients; improved with radiation therapy (4.5-20 months) + chemotherapy*DDx:* A.Neoplastic disorders(1)Glioma (may be bilateral with involvement of basal ganglia + corpus callosum, may show dense homogeneous enhancement with vascularity)(2)Metastases (known primary, at gray-white matter junction)(3) Primitive neuroectodermal tumor (4)MeningiomaB.Infectious disease (multicentricity)(1)Abscess, especially toxoplasmosis (large edema)(2)Sarcoidosis(3)TuberculosisC.Demyelinating disease(1)Multiple sclerosis(2)Progressive multifocal leukoencephalopathy

Spinal Epidural Lymphoma Leukemia

Notes:

Spinal Epidural Lymphoma (a)invasion of epidural space through intervertebral foramen from paravertebral lymph nodes(b)destruction of bone with vertebral collapse (less common)(c)direct involvement of CNS (rare)

Notes:

Leukemia CNS affected in 10% of patients with acute leukemia √ enlargement of ventricles + sulci due to atrophy (31%) √ sulcal / fissural / cisternal enhancement (meningeal infiltration) in 5%*Prognosis:*3-5 months survival if untreated

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Notes:

MESIAL TEMPORAL SCLEROSIS
*Cause:*long-standing temporal lobe epilepsy*Histo:*marked neuronal loss throughout hippocampal subfields with relative sparing of the CA2 subfield*Mechanism for excitotoxicity-induced neuronal death:* seizures cause excessive neuronal depolarization which cause overproduction of excitory amino acid neurotransmitters which cause excessive activation of N-methyl-D-aspartate receptors which cause unregulated entry of Ca^{2+} which causes neuronal swelling with cytotoxic edema √ increased signal intensity + decreased volume of hippocampus compared to contralateral side on T2WI*Associated limbic system findings:* √ ipsilateral atrophy of fornix (55%)√ ipsilateral atrophy of mamillary body (26%)*Associated extrahippocampal abnormalities:* √ increased signal intensity of anterior temporal lobe cortex (38%)√ cerebral hemiatrophy (1%)

Notes:

MEDULLOBLASTOMA
most malignant infratentorial neoplasm; most common neoplasm of posterior fossa in childhood (followed by cerebellar astrocytoma) *Incidence:*15-20% of all pediatric intracranial tumors; 30-40% of all posterior fossa neoplasms in children; 2-10% of all intracranial gliomas*Origin:*from external granular layer of inferior medullary velum (= roof of 4th ventricle)*Histo:*completely undifferentiated cells (50%), desmoplastic variety (25%), glial / neuronal differentiation (25%)*Age:*40% within first 5 years of life; 75% in first decade; between ages 5-14 (2/3); between ages 15-35(1/3); M:F = 2-4:1 ● duration of symptoms <1 month prior to diagnosis: nausea, vomiting, headache, increasing head size, ataxiaSite: (a)vermis cerebelli + roof of 4th ventricle (younger age group) in 91%(b)cerebellar hemisphere (older age group)Size:usually >2 cm in diameter√ well-defined vermian mass with widening of space between cerebellar tonsils√ encroachment on 4th ventricle / aqueduct with hydrocephalus (85-95%)√ shift / invagination of 4th ventricle√ rapid growth with extension into cerebellar hemisphere / brainstem (more often in adults)√ extension into cisterna magna + upper cervical cord, occasionally through foramina of Luschka into cerebellopontine angle cistern√ mild / moderate surrounding edema (90%)CT: Classic features in 53%: √ slightly hyperdense (70%) / isodense (20%) / mixed (10%) lesion√ rapid intense homogeneous enhancement (97%) due to usually solid tumorAtypical features: √ cystic / necrotic areas (10-16%) with lack of enhancement√ calcifications in 13%√ hemorrhage in 3%√ supratentorial extensionMR: √ mixed / hypointense on T1WI√ hypo- / iso- / hyperintense on T2WI√ usually homogeneous Gd-DTPA enhancement with hypointense rim√ cerebellar folia blurred*Cx:*(1)Subarachnoid metastatic spread (30-100%) via CSF pathway to spinal cord + cauda equina ("drop metastases" in 40%), cerebral convexities, sylvian fissure, suprasellar cistern, retrograde into lateral + 3rd ventricle√ continuous "frosting" of tumor on pia(2)Metastases outside CNS (axial skeleton, lymph nodes, lung) after surgery*Rx:*surgery + radiation therapy (extremely radiosensitive)*DDx of midline medulloblastoma:* ependymoma, astrocytoma (hypodense) *DDx of eccentric medulloblastoma:* astrocytoma, meningioma, acoustic neuroma

Notes:

MENINGIOMA
*Incidence:*most common extra-axial tumor; 15-18% of intracranial tumors in adults; 1-2% of primary brain tumors in children; 33% of all incidental intracranial neoplasms *Origin:*derived from meningothelial cells concentrated in arachnoid villi (= "arachnoid cap cells") which penetrate the dura (villi are numerous in large dural sinuses, in smaller veins, along root sleeves of exiting cranial + spinal nerves, choroid plexus)*Histologic classification:* -benign behavior pattern(a)fibroblastic type = fibrous typeinterwoven bands of spindle cells + collagen + reticulin fibers (b)transitional type = mixed typefeatures of meningothelial + fibroblastic forms -aggressive imaging appearance(c)meningothelial = syncytial typeforming a syncytium of closely packed cells with indistinct borders (d)angioblastic / malignant typeprobably hemangiopericytoma / hemangioblastoma arising from vascular pericytes *Age:*peak incidence 45 years (range 35-70 years); rare <20 years (in children >50% malignant, M > F); M:F = 1:2 to 1:4*Associated with:*type 2 neurofibromatosis (multiple meningiomas, occurrence in childhood)10% of patients with multiple meningiomas have type 2 neurofibromatosis!Most common radiation-induced CNS tumor with latency period of 19-35 years varying with dosage!*Types:* (1)Globular meningioma (most common):compact rounded mass with invagination of brain; flat at base; contact to falx / tentorium / basal dura / convexity dura (2)Meningioma en plaque:pronounced hyperostosis of adjacent bone particularly along base of skull; difficult to distinguish hyperostosis from tumor cloaking the inner table (DDx: Paget disease, chronic osteomyelitis, fibrous dysplasia, metastasis) (3)Multicentric meningioma (2-9%):16% in autopsy series; tendency to localize to a single hemicranium; present clinically at earlier age; global / mixed; CSF seeding is exceptional; in 50% associated with neurofibromatosis type 2
Location: (a)convexity = lateral hemisphere (20-34%)(b)parasagittal = medial hemisphere (18-22%)-falcine meningioma (5%) below superior sagittal sinus, usually extending to both sides(c)sphenoid ridge + middle cranial fossa (17-25%)(d)frontobasal (10%)(e)posterior fossa (9-15%)-cerebellar convexity (5%)-tentorium cerebelli (2-4%)-cerebellopontine angle (2-4%)-clivus (<1%)(f)spine (12%)Atypical location: (a)cerebellopontine angle (<5%)(b)optic nerve sheath (<2%)(c)intraventricular (2-5%): 80% in lateral (L > R), 15% in 3rd, 5% in 4th ventricle; from infolding of meningeal tissue during formation of choroid plexusMost common trigonal intraventricular mass in adulthood!(d)ectopic = extradural (<1%): intradiploic space, outer table of skull, scalp, paranasal sinus, parotid gland, parapharyngeal space, mediastinum, lung, adrenal gland
Plain film: √ hyperostosis at site close to / within bone (exostosis, enostosis, sclerosis)Hyperostosis does NOT indicate tumor infiltration!√ blistering at paranasal sinuses (ethmoid, sphenoid) ± sclerosis (= pneumosinus dilatans)√ enlarged meningeal grooves (if location in vault), enlarged foramen spinosum√ calcification (= psammoma bodies)CT: √ sharply demarcated well-circumscribed slowly growing mass√ wide attachment to adjacent dura mater√ "cortical buckling" of underlying brain√ isodense / hyperdense lesion (psammomatous calcifications) on NECT√ calcifications in circular / radial pattern (20%) (DDx: osteoma)√ "intraosseous meningioma" = permeation of bone with intra- and extracerebral soft-tissue component (DDx: fibrous dysplasia)√ hyperostosis of adjacent bone (18%)√ intense uniform enhancement on CECT (absence of blood-brain barrier)√ minimal peritumoral edema (in up to 75%): NO correlation between tumor size + amount of edema (DDx: intra-axial lesion)√ cystic component: major in 2%, minor in 15%MR (100% detection rate with gadolinium DTPA): √ hypo- to isointense on T1WI + iso- to hyperintense on T2WI (intensity depends on amount of cellularity versus collagen elements)√ homogeneous / heterogeneous texture (tumor vascularity, cystic changes, calcifications)√ arcuate bowing of white matter + cortical effacement√ tumor-brain interface of low-intensity vessels + high-intensity cerebrospinal cleft on T2WI√ contrast enhancement for 3-60 minutes on T1WI as high as 148% over brain parenchyma√ "dural tail" sign = curvilinear area of enhancement tapering off from the margin of tumor along dural surface in 60% (= dural tumor infiltration / reactive hypervascularity / reactive hyperplastic changes)
Angio: √ "mother-in-law" phenomenon (contrast material shows up early and stays late into venous phase)√ "sunburst" / "spoke-wheel" pattern of tumor vascularity with hypervascular cloudlike stain√ early draining vein (rare: perhaps in angioblastic meningioma)√ en plaque meningioma is poorly vascularizedVascular supply: A.External carotid artery (almost always):1.vault: middle meningeal artery2.sphenoid plane + tuberculum: recurrent meningeal branch of ophthalmic a.3.tentorium: meningeal branch of meningohypophyseal trunk of ICA4.clivus + posterior fossa: vertebral artery / ascending pharyngeal artery5.falx: partly middle meningeal artery + othersB.Internal carotid artery (rare):1.intraventricular: choroidal vessels*Cx:*local invasion of venous sinuses
ATYPICAL MENINGIOMA (15%) 1.Low attenuation area of necrosis, old hemorrhage, cyst formation, fat (DDx: malignant glioma, metastasis)(a)**Cystic meningioma** (2-4%)*Frequency:*55-65% in 1st year of life; 10% in childrentype I =intratumoral central / eccentric cyst (ischemic necrosis, microcystic degeneration, breakdown of hemorrhagic products); often associated with meningothelial / microcystic / atypical / malignant histologic subtypestype II =extratumoral intraparenchymal cyst (arachnoid cyst / reactive gliosis / liquefactive necrosis of adjacent brain)type III = trapped CSF (DDx: cystic / necrotic glioma)(b)**Lipoblastic meningioma** (5%)metaplastic change of meningothelial cells into adipocytes 2.Heterogeneous / ring enhancement (secondary to bland tumor infarction / necrosis in aggressive histologic variants / true cyst formation from benign fluid accumulation)3."En plaque" morphology4."Comma shape" = combination of semilunar component bounded by dural interface + spherical component growing beyond dural margin5.Sarcomatous transformation with spread over hemisphere + invasion of cerebral parenchyma (leptomeningeal supply)6.**Meningeal hemangiopericytoma**√ multilobulated contour√ narrow dural base / "mushroom" shape√ large intratumoral vascular signals√ bone erosion√ prominent peritumoral edema√ multiple irregular feeding vessels on angiogram

Sphenoid Wing Meningioma Suprasellar Meningioma

Notes:

Sphenoid Wing Meningioma 1.Hyperostotic meningioma en plaque ● slowly progressive unilateral painless exophthalmos ● numbness in distribution of cranial nerve V_1 + V_2 ● headaches, seizures2.Meningioma arising from middle third of sphenoid ridge ● headaches, seizures√ compression of regional frontal + temporal lobes3.Meningioma arising from clinoid process√ encasement of carotid + middle cerebral arteries√ compression of optic nerve + chiasm4.Meningioma of planum sphenoidale√ subfrontal growth + posterior growth into sella turcica and clivus√ hyperostotic blistering of planum sphenoidale

Notes:

Suprasellar Meningioma *Incidence:*10% of all intracranial meningiomas*Origin:*from arachnoid + dura along tuberculum sellae / clinoids / diaphragma sellae / cavernous sinus with secondary extension into sella; NOT from within pituitary fossa ● hypothalamic / pituitary dysfunction (rare)√ irregular hyperostosis = blistering adjacent to sinus (HALLMARK of meningiomas at planum sphenoidale / tuberculum sellae)√ pneumatosis sphenoidale = increased pneumatization of sphenoid in area of anterior clinoids + dorsum sellae (DDx: normal variant)√ broad base of attachment√ intense homogeneous enhancement (may be impossible to differentiate from supraclinoid carotid aneurysm on CT)√ blood supply: posterior ethmoidal branches of ophthalmic artery, branches of meningohypophyseal trunkMR: √ large mass isointense to gray matter on T1WI + T2WI√ hyperintense flattened pituitary gland within floor of sella√ marked homogeneous enhancement on T1WI*DDx:*metastasis, glioma, lymphoma

Notes:

Radiology Review Manual
fourth edition

MENINGITIS

1.Pachymeningitis: affecting dura mater2.Leptomeningitis: affecting pia matter / arachnoid (most common) ● headaches, stiff neck ● confusion, disorientation ● positive CSF lab analysisROLE of CT and MR: (1)to exclude parenchymal abscess, ventriculitis, localized empyema(2)to evaluate paranasal sinuses / temporal bone as source of infection(3)to monitor complications: hydrocephalus, subdural effusion, infarction

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Purulent Meningitis Granulomatous Meningitis

Notes:

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Purulent Meningitis *Cause:*otitis media / sinusitis*Organism:* (a)adults:Meningococcus, Streptococcus pneumoniae, Haemophilus influenzae, Neisseria meningitidis, Staphylococcus aureus(b)children:Escherichia coli, Citrobacter, b-hemolytic StreptococcusNECT: √ often normal√ increased density in subarachnoid space (increased vascularity), esp. in children√ small ventricles secondary to diffuse cerebral edemaCECT: √ marked curvilinear meningeal enhancement over cerebrum (frontal + parietal lobes) and interhemispheric + sylvian fissures√ obliteration of basal cisterns with enhancement (common)MR (most sensitive modality): √ hyperintense plaques on T2WI√ leptomeningeal enhancement with Gd-DTPA*Cx:* (1)Cerebritis(2)Ventriculitis = ependymitis (secondary to retrograde spread)(3)Brain atrophy(4)Brain infarction (arteritis, venous thrombosis)(5)Subdural effusion [sterile subdural effusion secondary to H. influenzae meningitis (in children) may turn into empyema](6)Hydrocephalus (cellular debris blocking foramen of Monro, aqueduct, 4th ventricular outlet / intraventricular septa / arachnoid adhesions)(7)Cranial nerve dysfunction*Prognosis:* ◊Cerebral infarction + edema are predictive of poor outcome◊Enlargement of ventricles + subarachnoid spaces + subdural effusions have no predictive value*Mortality:*10% (5th common cause of death in children between 1 and 4 years of age)*DDx:*meningeal carcinomatosis

Notes:

Granulomatous Meningitis *Histo:*thick exudate, perivascular inflammation, granulation tissue + reactive fibrosis(1)Tuberculous meningitis = basilar meningitis:part of generalized miliary tuberculosis / primary tuberculous infection; in infants + small children(2)Sarcoidosismay be associated with single / multiple intracerebral masses(3)Fungal meningitis: cryptococcosis, candidiasis, coccidioidomycosis (endemic), blastomycosis, mucormycosis (diabetics), nocardiosis, actinomycosis, aspergillosis (under chronic corticosteroid therapy) ● acute life-threatening process / chronic indolent disease*May be associated with:* cerebritis, abscess formation√ hydrocephalusCT: √ obliteration of basal cisterns, sylvian fissure, suprasellar cistern (isodense cisterns secondary to filling with debris)√ intense contrast enhancement of gyri + involved subarachnoid spaces√ calcification of meninges√ decreased attenuation of white matterMR: √ high-signal intensity of basilar cisterns on T2WI√ enhancement with gadopentetate dimeglumine*Cx:*(1)hydrocephalus (obliteration of basal cisterns; blocking of CSF flow + CSF absorption)(2)infarction (due to arteritis)

Notes:

METACHROMATIC LEUKODYSTROPHY
=MLD = most common hereditary (autosomal recessive) leukodystrophy (dysmyelinating disorder)
*Cause:*deficiency of arylsulfatase A resulting in severe deficiency of myelin lipid sulfatide within macrophages + Schwann cells*Age of presentation:*before age 3 (2/3),in adolescence (1/3)
A.LATE INFANTILE FORM*Age:*2nd year of life ● gait disorder + strabismus ● impairment of speech ● spasticity + tremor ● intellectual deterioration*Prognosis:*death within 4 years of onsetB.JUVENILE FORM*Age:*5-7 yearsC.ADULT FORM ● organic mental syndrome ● progressive corticospinal, corticobulbar, cerebellar, extrapyramidal signs
√ progressive loss of hemispheric brain tissueCT: √ symmetric low density of white matter adjacent to ventricles (esp. centrum ovale and frontal horns)√ progressive atrophy√ no contrast enhancementMR: √ progressive symmetrical areas of hypointensity on T1WI√ hyperintensity on T2WI (increased water)*Prognosis:*death within several years

Notes:

METASTASES TO BRAIN

*Incidence:*14-37% of all intracranial tumors*Metastatic primary:* Six tumors account for 95% of all brain metastases: 1.Bronchial carcinoma (47%): RARELY squamous cell carcinoma2.Breast carcinoma (17%)3.GI-tract tumors (15%): colon, rectum4.Hypernephroma (10%)5.Melanoma (8%)6.Choriocarcinoma*In childhood:* 1.Leukemia / lymphoma2.Neuroblastoma*Brain metastases from sarcomas are exceptionally rare!Location:* (a)corticomedullary junction of brain (most characteristic)(b)subarachnoid space = carcinomatous meningitis (15%)(c)subependymal spread (frequent in breast carcinoma)(d)skull (5%)

HEMORRHAGIC METASTASES (in 3-4%): 1.Malignant melanoma2.Choriocarcinoma3.Oat cell carcinoma of lung4.Renal cell carcinoma5.Thyroid carcinoma√ hyperdense without contrast√ hypervascular with contrast*mnemonic:*"MATCH"**M**elanoma **A**naplastic lung carcinoma Thyroid carcinoma **C**horiocarcinoma **H**ypernephroma

CYSTIC METASTASES: 1.Squamous cell carcinoma of lung2.Adenocarcinoma of lung

CALCIFIED METASTASES: 1.Mucin-producing neoplasm2.Cartilage- / bone-forming sarcoma3.Effective radiochemotherapy

Presentation: -multiple lesions (2/3), single lesion (1/3)-cerebral hemispheres (57%), cerebellum (29%), brainstem (32%)-nodular deposits to dura are common√ multiple lesions of different sizes + locations√ surrounding edema usually exceeds tumor volumeCT: √ solid enhancement in small tumors / ringlike enhancement in large tumorsMR: (a combination of T2WI + contrast-enhanced T1WI offer greatest sensitivity) √ hypointense mass relative to edema on T2WI√ hypointensity more pronounced in melanoma + mucinous adenocarcinoma (paramagnetic effect)√ homogeneous / ring / nodular mixed enhancement after Gd-DTPA; often more than one metastatic focus identified in region of colliding edema√ asymmetric enhancement of dura with dural spread√ leptomeningeal enhancement (eg, in metastatic ependymoma)

Notes:

MICROCEPHALY
=clinical syndrome characterized by a head circumference below the normal range*Incidence:*1.6:1,000 or 1:6,200-1:8,500 births*Etiology:* (1)Undiagnosed intrauterine infection (toxoplasmosis, rubella, CMV, herpes, syphilis), toxic agents, drugs hypoxia, radiation, maternal phenylketonuria(2)Premature craniosynostosis(3)Chromosomal abnormalities (trisomies 13, 18, 21)(4)Meckel-Gruber syndrome*Often associated with:* micrencephaly, macrogyria, pachygyria, atrophy of basal ganglia, decrease in dendritic arborization, holoprosencephaly √ AC:HC discrepancy√ head circumference <3 S.D. below the mean√ apelike sloping of forehead√ dilatation of lateral ventricles√ poor growth of fetal cranium√ intracranial contents may not be visible (rare)*Prognosis:*normal to severe mental retardation (depending on degree of microcephaly)

Notes:

MINERALIZING MICROANGIOPATHY
=RADIATION-INDUCED LEUKOENCEPHALOPATHY=sequelae of radiotherapy combined with methotrexate therapy for leukemia*Incidence:*in 25-30% after >9 months after treatment*Age:*childhood*Cause:*deposition of calcium within small vessels of previously irradiated brain parenchyma ● 85% without neurologic deficitsCT: √ thin reticular / serrated linear / punctate calcifications near corticomedullary junction, especially in basal ganglia + frontal and posterior parietal lobes√ symmetric low-attenuation process in white matter near corticomedullary areaMR: √ confluent diffuse periventricular distribution spreading peripherally with an irregular scalloped edge

Notes:

MOYAMOYA DISEASE
=progressive obstructive / occlusive cerebral arteritis affecting distal ICA at bifurcation into its branches (anterior 2/3 of circle of Willis), usually involving both hemispheres*Etiology:*unknown*Age:*predominantly in children + young adults*Path:*endothelial hyperplasia + fibrosis without associated inflammatory reaction ● headaches ● behavioral disturbances ● recurrent hemiparetic attacks√ bilateral stenosis / occlusion of supraclinoid portion of internal carotid extending to proximal portions of middle + anterior cerebral arteries√ large network of vessels in basal ganglia ("puff of smoke") + upper brainstem fed by basilar artery, anterior + middle cerebral arteries (dilatation of lenticulostriate + thalamoperforating arteries)√ anastomoses between dural meningeal + leptomeningeal arteries*Cx:*subarachnoid hemorrhage (occasionally)

Moyamoya Syndrome

Notes:

Moyamoya Syndrome *Etiology:*neurocutaneous syndromes (neurofibromatosis), bacterial meningitis, periarteritis nodosa, head trauma, tuberculosis, oral contraceptives, atherosclerosis, sickle cell anemia

Notes:

MULTIPE SCLEROSIS
=most frequent form of chronic inflammatory demyelinating disease of unknown etiology, which reduces the lipid content and brain volume; characterized by a relapsing + remitting course*Prevalence:*6:10,000 (higher frequency in cooler climates; increased incidence with positive family history)*Cause:*? viral / autoimmune mechanism*Peak age:*25-30 (range of 20-50) years; M:F = 2:3*Histo:* (a)acute stage: perivenular inflammation (at junctions of pial veins) with-hypercellularity (= infiltration of lipid-laden macrophages + lymphocytes)-well-demarcated demyelination (destruction of oligodendroglia with loss of myelin sheath)-reactive astrocytosis (= gliosis), initially with preservation of axons (= denuded axons) resulting in scar (= white matter plaque)(b)chronic stage: plaques advance to fibrillary gliosis with reduction in inflammatory component*Clinical forms:*(a)relapsing remitting(b)relapsing progressive(c)chronic progressive ● waxing and waning course with ● numbness, dysesthesia, burning sensations ● signs of brain neoplasm: headaches, seizures, dizziness, nausea, weakness, altered mental status ● ataxia, diplopia ● optic neuritis = retrobulbar pain, central loss of vision, afferent pupillary defect (Marcus Gunn pupil) ● trigeminal neuralgia (1-2%) ● Schumacher criteria: (1) CNS dysfunction (2) involvement of two / more parts of CNS (3) predominant white matter involvement (4) two / more episodes lasting >24 hours less than 1 month apart (5) slow stepwise progression of signs + symptoms (6) at onset 10-50 years of age ● Rudick red flags (suggests diagnosis other than MS):(1) no eye findings (2) no clinical remission (3) totally local disease (4) no sensory findings (5) no bladder involvement (6) no CSF abnormality @Brain◊number + extent of plaques correlate with duration of disease + degree of cognitive impairmentLocation: subependymal periventricular location (along lateral aspects of atria + occipital horns), corpus callosum, internal capsule, centrum semiovale, corona radiata, optic nerves, chiasm, optic tract, brainstem (ventrolateral aspect of pons at 5th nerve root entry), cerebellar peduncles, cerebellum; rather symmetric involvement of cerebral hemispheres; subcortical U fibers NOT spared √ lesion size: 1-25 (majority between 5 and 10) mm√ large lesions may masquerade as brain tumors√ lesions usually without mass effect / edema unless acute√ ovoid lesions (86%) oriented with their long axis perpendicular to ventricular walls (due to perivenous demyelination; pathologically described as "Dawson fingers")√ chronic plaques do not enhance (due to intact blood-brain barrier)CT: √ normal CT scan (18%)√ nonspecific atrophy of brain (45%): enlarged ventricles, prominent sulci√ periventricular (near atria) multifocal nonconfluent lesions with distinct margins (location not always correlating well with symptoms)(a)NECT:isodense / lucent(b)CECT:transient enhancement during acute stage (active demyelination) for about 2 weeks; may require double dose of contrast; ultimately disappearance / permanent scarMR (modality of choice; 95% specific): √ well-marginated discrete foci of varying size with high-signal intensity on T2WI + proton density images (= loss of hydrophobic myelin produces increase in water content); hypointense on T1WI√ Gd-DTPA enhancement of lesions on T1WI (up to 8 weeks following acute demyelination with breakdown of blood-brain barrier)√ lesions on undersurface of corpus callosum (CHARACTERISTIC sagittal images)
@Spinal cord◊Most common demyelinating process of spinal cord!◊In 12% without coexistent intracranial plaques! ● number + extent of plaques correlate with degree of disabilityLocation:predilection for cervical regionSite:eccentric involvement of dorsal + lateral elements abutting subarachnoid space√ atrophic plaques oriented along spinal cord axis√ length of plaque usually less than 2 vertebral body segments + width less than half of cross section√ acute tumefactive MS = cord swelling + enhancement*DDx:*(1)Cord tumor (follow-up after 6 weeks without decrease in size of lesion)(2)Infection(3)Acute transverse myelitis (after viral illness / vaccination) *Rx:*steroids (inciting rapid decrease in size of lesions + loss of enhancement)*DDx:* (1)White matter ischemic disease (patients >50 years of age, lesions <5 mm, not infratentorial)(2)Acute disseminated encephalomyelitis, subacute sclerosing panencephalitis (lesions of similar age)(3)AIDS, CNS vasculitis, migraine, radiation injury, lymphoma, sarcoidosis, tuberculosis, systemic lupus erythematosus, cysticercosis, metastases, multifocal glioma, neurofibromatosis, contusions

Notes:

MYELINOCLASTIC DIFFUSE SCLEROSIS
=SCHILDER DISEASE=rare demyelinating disorder with episodic recurrence and remission*Age:*children > adults; M:F = 1:1*Histo:*selective confluent demyelination with relative axonal sparing, perivascular inflammatory infiltrate, reactive astrocytosis (indistinguishable from multiple sclerosis) ● hemiplegia, aphasia, ataxia, blindness ● swallowing difficulties, progressive dementia ● increased intracranial pressureLocation:centrum semiovale√ large bilateral white matter lesions with mass effect√ enhancement with IV contrast material*Rx:*usually responsive to corticosteroids*DDx:*(1)Acute disseminated encephalomyelitis (history of recent viral illness, monophasic course, lesions less confluent, no mass effect / enhancement)(2)Adrenoleukodystrophy (bilaterally symmetric, confluent lesions, parietal location)(3)Tumor, abscess, infarct

Notes:

Germinal Matrix Bleed =GERMINAL MATRIX-RELATED HEMORRHAGE
Germinal matrix = highly vascular gelatinous subependymal tissue adjacent to lateral ventricles in which the cells that compose the brain are generated; has its largest volume around 26 weeks GA; decreases in size with increasing fetal maturity; usually involutes by 32-34 weeks of gestation Location:greatest portion of germinal matrix above caudate nucleus in floor of lateral ventricle, tapering as it sweeps from frontal horn posteriorly into temporal horn, roof of 3rd + 4th ventricleArterial supply:via Heubner artery from ACA, striate branches of MCA, anterior choroidal a., perforating branches from meningeal aa.Capillary network:persisting immature vascular rete= large irregular endothelial-lined channels devoid of connective tissue support (collagen and muscle) Venous drainage:terminal vv., choroidal v., thalamostriate v. course anteriorly + feed into internal cerebral v. which has a posterior course
Risk factors: (1) prematurity (2) low birth weight (3) sex (M:F = 2:1) (4) multiple gestations (5) trauma at delivery (6) prolonged labor (7) hyperosmolarity (8) hypocoagulation (9) pneumothorax (10) patent ductus arteriosus *Etiology:*hypoxia with loss of autoregulation*Pathogenesis:*rupture of friable vascular bed due to(1)fluctuating cerebral blood flow in preterm infants with respiratory distress(2)increase in cerebral blood flow with(a)systemic hypertension (pneumothorax, REM sleep, handling, tracheal suctioning, ligation of PDA, seizures, instillation of mydriatics)(b)Rapid volume expansion (blood, colloid, hyperosmolar glucose / sodium bicarbonate)(c)Hypercarbia (RDS, asphyxia)(3)increase in cerebral venous pressure with labor and delivery, asphyxia (= impairment in exchange of oxygen and carbon dioxide), respiratory disturbances(4)decrease in cerebral blood flow with systemic hypotension followed by reperfusion(5)platelet and coagulation disturbance *Incidence:*in premature neonates <32 weeks of age; in 43% of infants <1,500 g (in 65% of 500-700 g infants, in 25% of 701-1,500 g infants) ; in up to 50% without prenatal care, in 5-10% with prenatal care Location:region of the caudate nucleus and thalamostriate groove (= caudothalamic notch) remains metabolically active the longest; in 80-90% in infants <28 weeks of MA age*Time of onset:*36% on first day, 32% on second day, 18% on first 3 day of life; by 6th day 91% of all intracranial bleeds have occurredGRADES (Papile classification) I:subependymal hemorrhage confined to germinal matrix (GMH) on one / both sidesII:subependymal hemorrhage ruptured into nondilated ventricle (IVH)III:intraventricular hemorrhage (IVH) with ventricular enlargement: (a) mild, (b) moderate, (c) severeIV:extension of germinal matrix hemorrhage into brain parenchyma (IPH)*Serial scans:*5-10-day intervals
US (100% sensitivity + 91% specificity for lesions >5 mm; 27% sensitivity + 88% specificity for lesions ≤5 mm):Germinal matrix hemorrhage (grade I) √ well-defined ovoid area of increased echogenicity (= fibrin mesh within clot) inferolateral to floor of frontal horn ± body of lateral ventricle√ bulbous enlargement of caudothalamic groove anterior to termination of choroid plexus*DDx:*choroid plexus (attached to inferomedial aspect of ventricular floor, tapers toward caudothalamic groove, never anterior to foramen of Monro)√ resolving bleed develops central sonolucency√ outcome: (1) complete involution (2) thin echogenic scar (3) subependymal cyst
Mild intraventricular hemorrhage (grade II) √ echogenic material filling a portion of lateral ventricles (acute phase) becoming sonolucent in a few weeks√ clot may gravitate into occipital horns√ vertical band of echogenicity between thalami on coronal scans (blood in 3rd ventricle)√ irregular bulky choroid plexus (clot layered on surface of choroid plexus)√ temporarily increased echogenicity of ventricular wall (= subependymal white halo between 7 days and 6 weeks after hemorrhagic event)
Extensive intraventricular hemorrhage (grade III) √ intraventricular cast of blood distending the lateral ventricles√ ± extension of hemorrhage into basal cisterns, cavum septi pellucidi√ hemorrhage becomes progressively less echogenic√ temporarily thickened echogenic walls of ventricles ("ventriculitis")
Intraparenchymal hemorrhage (grade IV) *Cause:*(a)extension of hemorrhage originating from germinal matrix (unusual)(b)separate hemorrhage within infarcted periventricular tissue (frequent)Location:on side of largest amount of IVH, commonly lateral to frontal horns / in parietal lobe, rare in occipital lobe + thalamus√ homogeneous highly echogenic intraparenchymal mass with irregular margins√ central hypoechogenicity (liquefying hematoma after 10-14 days)√ retracted clot settles to dependent position (3-4 weeks)√ complete resolution by 8-10 weeks results in anechoic area (= porencephalic cyst)
CT: Most sensitive + definite means to define site + extent of hemorrhage, especially in subdural hemorrhage, cerebral parenchymal hemorrhage, posterior fossa lesion √ hyperdense bleed only visible up to 7 days before it becomes isodense*Cx:* (1)Posthemorrhagic hydrocephalus (30-70%)√Severity of hydrocephalus directly proportional to size of original hemorrhage!*Cause:* (a)temporary blockage of arachnoid villi by particulate blood clot (within days), often transient with partial / total resolution(b)obliterative fibrosing arachnoiditis often in cisterna magna (within weeks); frequently leads to permanent progressive ventricular dilatation (50%)√ thickened echogenic ventricular wallsTime of onset:by 14 days (in 80%) ● delayed clinical signs because of compressible premature brain parenchyma√ ventricular dilatation, particularly affecting the occipital horns (amount of compressible immature white matter is larger posteriorly)*DDx:* ventriculomegaly secondary to periventricular cerebral atrophy (occurring slowly over several weeks)(2)Cyst formation(a)cavitation of hemorrhage(b)unilocular subependymal cyst(c)unilocular porencephalic cyst(3)Mental retardation, cerebral palsy(4)Death in 25% (IVH most common cause of neonatal death)*Prognosis:* (1)Grade I + II: good with normal developmental scores (12-18% risk of handicap)(2)Grade III + IV: 54% mortality; 30-40% risk of handicap (spastic diplegia, spastic quadriparesis, intellectual retardation)

Notes:

Choroid Plexus Hemorrhage affects primarily full-term infants *Cause:*birth trauma, asphyxia, apnea, seizures√ echogenicity of choroid plexus same as hemorrhage√ nodularity of choroid plexus√ enlargement of choroid plexus >12 mm in AP diameter√ left-right asymmetry >5 mm√ intraventricular hemorrhage without subependymal hemorrhage*Cx:*intraventricular hemorrhage (25%)

Notes:

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Intracerebellar Hemorrhage *Cause:* (a)full-term infant: traumatic delivery, intermittent positive pressure ventilation, coagulopathy(b)premature infant: subependymal germinal matrix hemorrhage up to 30 weeks gestation*Incidence:*16-21% of autopsies√ echogenicity of vermis same as hemorrhage√ echogenic mass in less echogenic cerebellar hemisphere (coronal scan most useful)√ nonvisualization / deformity of 4th ventricle√ asymmetry in thickness of paratentorial echogenicity is a sign of subarachnoid hemorrhage*Prognosis:*poor + frequently fatal

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Notes:

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Intraventricular Hemorrhage *Etiology:* (a)germinal matrix hemorrhage ruptures through ependymal lining at multiple sites(b) bleeding from choroid plexusRoute of hemorrhage: blood dissipates throughout ventricular system + aqueduct of Sylvius, passes through foramina of 4th ventricle, collects in basilar cistern of posterior fossa ● seizures, dystonia, obtundation, intractable acidosis ● bulging anterior fontanelle, drop in hematocrit, bloody / proteinaceous CSF ● IVH usually cleared within 7-14 daysCx:(1)Intracerebral hemorrhage(2)Hydrocephalus

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Notes:

Periventricular Leukoencephalopathy ***Periventricular Leukomalacia*** =PVL = perinatal hypoxic-ischemic encephalopathy=principal ischemic lesion of the premature infant characterized by focal coagulation necrosis of deep white matter as a result of ischemic infarction involving the watershed (= arterial border) zones between central and peripheral vascularityVascular supply: (a)ventriculopedal branches penetrating cerebrum from pial surface are derived from MCA ± PCA ± ACA(b)ventriculofugal branches extending from ventricular surface are derived from choroidal arteries ± striate arteries*Incidence:* 7-22% at autopsy (88% of infants between 900 and 2,200 g surviving beyond 6 days); in 34% of infants <1,500 g; in 59% of infants surviving longer than 1 week on assisted ventilation; only 28% detected by cranial sonography *Histo:*edema, white matter necrosis, evolution of cysts + cavities / diminished myelin; nonhemorrhagic : hemorrhagic PVL = 3:1*Pathogenesis:* immature autoregulation of periventricular vessels secondary to deficient muscularis of arterioles limits vasodilation in response to hypoxemia + hypercapnia + hypotension of perinatal asphyxia (hypoxic-ischemic encephalopathy) ● "cerebral palsy" (in 6.5% of infants <1,800 g) ● spastic diplegia (81%) > quadriparesis (necrosis of descending fibers from motor cortex) ● choreaathetosis, ataxia ● ± mental retardation ● severe visual / hearing impairment ● convulsive disordersLocation: bilateral white matter subjacent to external angle of lateral ventricular trigones, involving particularly the centrum semiovale (frontal horn + body), optic (occipital horn), and acoustic (temporal horn) radiations

US (50% sensitivity + 87% specificity): Early changes (2 days to 2 weeks after insult) √ increased periventricular echogenicity (PVE) (DDx: echogenic periventricular halo / blush of fiber tracts in normal neonates, white matter gliosis, cortical infarction extending into deep white matter)√ bilateral often asymmetric zones, occasionally extending to cortex√ infrequently accompanied by IVHLate changes (1-3-6 weeks after development of echodensities): √ periventricular cystic PVL = cystic degeneration of ischemic areas (= multiple small never septated periventricular cysts in relationship to lateral ventricles; the larger the echodensities, the sooner the cyst formation)√ brain atrophy secondary to thinning of periventricular white matter always at trigones, occasionally involving centrum semiovale√ ventriculomegaly (after disappearance of cysts) with irregular outline of body + trigone of lateral ventricles√ deep prominent sulci abutting the ventricles with little / no interposed white matter (DDx: schizencephaly)√ enlarged interhemispheric fissureCT (not sensitive in early phase): √ periventricular hypodensity (DDx: immature brain with increased water + incomplete myelination)MR (not sensitive in early phase): √ hypointense areas on T1WI√ hyperintense periventricular signals on T2WI in peritrigonal region√ thinning of posterior body + splenium of corpus callosum (= degeneration of transcallosal fibers)
Prognosis: major neurologic problem / death in up to 62%; PVL localized to frontal lobes show relative normal development; generalized PVL results in neurologic deficits in close to 100% *DDx:*tissue damage from ventriculitis (sequelae of meningitis), metabolic disorders, in utero ischemia (eg, maternal cocaine abuse)
Periventricular Hemorrhagic Infarction =hemorrhagic necrosis of periventricular white matter, usually large + asymmetric*Incidence:*in 15-25% of infants with IVH*Pathogenesis:* (a)germinal matrix hemorrhage with intraventricular blood clot (in 80%)(b)ischemic periventricular leukomalacialead to obstruction of terminal veins with sequence of venous congestion + thrombosis + infarction *Histo:*perivascular hemorrhage of medullary veins near ventricular angle*Associated with:*the most severe cases of intraventricular hemorrhage*Age:*peak occurrence on 4th postnatal day ● spastic hemiparesis (affecting lower + upper extremities equally) / asymmetric quadriparesis (in 86% of survivors)Location:lateral to external angle of lateral ventricle on side of more marked IVH: 67% unilateral; 33% bilateral but asymmetric
Early changes (hours to days after major IVH): √ unilateral / asymmetric bilateral triangular "fan-shaped" echodensities√ extension from frontal to parietooccipital regions / localized (particularly in anterior portion of lesion)Late changes: √ single large cyst = porencephaly√ bumpy ventricle / false accessory ventricle*Prognosis:*59% overall mortality with echodensities >1 cm; in 64% major intellectual deficits
Encephalomalacia =more extensive brain damage than PVL; may include all of white matter in subcortex + cortex*Associated with:* (1)Neonatal asphyxia(2)Vasospasm(3)Inflammation of CNS√ small ventricles (edema) with diffuse damage√ increased parenchymal echogenicity making it difficult to define normal structures√ decreased vascular pulsations√ transcranial Doppler:(a)group I (good prognosis)√ normal flow profile, normal velocities, normal resistive index(b)group II (guarded prognosis)√ increase in peak-systolic + end-diastolic flow velocities + decreased resistive index(c)group III (unfavorable prognosis)√ reduced diastolic flow + decreased peak systolic and diastolic velocities + increased resistive index√ ventricular enlargement + atrophy√ extensive multicystic encephalomalacia with cysts often not communicating

Notes:

NEUROBLASTOMA

Age at presentation:<2 years (50%); <4 years (75%);<8 years (90%); peak age <3 years ● abdominal mass (45%) ● neurologic signs (20%) ● bone pain / limp (20%) ● orbital ecchymosis / proptosis (12%) ● catecholamine production (95%) with paroxysmal episodes of flushing, tachycardia, hypertension, headaches, sweating, intractable diarrhea, acute cerebellar encephalopathy ● positive bone marrow aspiration (70%)Location:adrenal gland (67%), chest (13%), neck (5%), intracranial (2%); commonly involvement of multiple skeletal sitesNUC (overall sensitivity of detection better than radiography): CAVE:symmetric lytic neuroblastoma metastases occur frequently in metaphyseal areas where normal epiphyseal activity obscures lesions√ purely lytic lesions may present as photopenic areas√ soft-tissue uptake of Tc-99m phosphate in 60%√ frequently Ga-67 uptake in primary site of neuroblastoma*Prognosis:*2-year survival (a) in 60% for age <1 year(b) in 20% for ages 1-2 years (c) in 10% for ages >2 years

A.PRIMARY CEREBRAL NEUROBLASTOMA (rare)*Age:*childhood / early adolescence√ large hypodense / mixed-density mass with well-defined margins√ intratumoral coarse dense calcifications√ central cystic / necrotic zones with hemorrhage*Cx:*metastasizes via subarachnoid space to dura + calvarium

B.SECONDARY NEUROBLASTOMA (common)metastatic to:@ liver @ skeleton √ osteolysis with periosteal new-bone formation√ sutural diastasis√ hair-on-end appearance of skull@ orbit: √ unilateral proptosis◊Neuroblastoma usually not metastatic to brain!

Olfactory Neuroblastoma

Notes:

Olfactory Neuroblastoma =very malignant tumor arising from olfactory mucosa*Types:*1.Esthesioneuroepithelioma2.Esthesioneurocytoma3.Esthesioneuroblastoma√ mass in superior nasal cavity with extension into ethmoid + maxillary sinuses*Cx:*distant metastases in 20%

Notes:

NEUROFIBROMATOSIS

=autosomal dominant inherited disorder, probably of neural crest origin affecting all 3 germ cell layers, capable of involving any organ system*Path:* pure neurofibromas (= tumor of nerve sheath with involvement of nerve, nerve fibers run through mass) + neurilemomas (nerve fibers diverge and course over the surface of the tumor mass); frequently combined (1)discrete round mass(2)plexiform = tortuous tangles / fusiform enlargement of peripheral nerves (PATHOGNOMONIC of neurofibromatosis type 1)*Histo:*proliferation of fibroblasts + Schwann cells

Peripheral Neurofibromatosis (90%) Neurofibromatosis with Bilateral Acoustic Neuromas

Notes:

Peripheral Neurofibromatosis (90%) =NEUROFIBROMATOSIS TYPE 1 = NF-1=VON RECKLINGHAUSEN DISEASE= dysplasia of mesodermal + neuroectodermal tissue with potential for diffuse systemic involvement; autosomal dominant with abnormalities of long arm of chromosome **17**: *von Recklinghausen* has **17** letters; 50% spontaneous mutants; variable expressivity *Incidence:*1:2,000-4,000; M:F = 1:1; most common of phakomatoses
Diagnostic criteria (at least two must be present): (1)>6 café-au-lait spots >5 mm in greatest diameter (>15 mm in postpubertal individuals)(2)≥2 neurofibromas of any type / one plexiform neurofibroma(3)freckling in axilla / inguinal region(4)optic glioma(5)≥2 Lisch nodules (= pigmented hamartomas of iris)(6)distinctive osseous lesion (eg, sphenoid dysplasia / thinning of long bone cortex) ± pseudarthrosis(7)first-degree relative (parent, sibling, child) with peripheral neurofibromatosis
May be associated with: (1)MEA IIb (pheochromocytoma + medullary carcinoma of thyroid + multiple neuromas)(2)CHD (10 fold increase): pulmonary valve stenosis, ASD, VSD, IHSS
A.CNS MANIFESTATIONS@Intracranial1.Optic pathway gliomaisolated to single optic nerve ± extension to other optic nerve, chiasm, optic tracts *Histo:*pilocytic astrocytoma with perineural / subarachnoid spread (optic nerve is embryologically part of hypothalamus and develops gliomas instead of schwannomas)√in up to 30% of all neurofibromatosis patients√10% of all optic nerve gliomas are associated with neurofibromatosis2.Cerebral gliomasastrocytomas of tectum, brainstem, gliomatosis cerebri (= unusual confluence of astrocytomas) 3.Hydrocephalusobstruction usually at aqueduct of Sylvius *Cause:*benign aqueductal stenosis, glioma of tectum / tegmentum of mesencephalon4.Vascular dysplasia=occlusion / stenosis of distal internal carotid artery, proximal middle / anterior cerebral artery√ moyamoya phenomenon (60-70%) 5.Schwannomas of cranial nerves 3-12 (most commonly 5 + 8)6.Craniofacial plexiform neurofibromas=locally aggressive congenital lesion composed of tortuous cords of Schwann cells, neurons + collagen with progression along nerve of origin (usually small unidentified nerves)Location:commonly orbital apex, superior orbital fissure7.CNS hamartomas (up to 75-90%)=probably dysmyelinating lesions (may resolve)Location:pons, basal ganglia (most commonly in globus pallidus), thalamus, cerebellar white matter√ multiple foci of isointensity on T1WI + hyperintensity on T2WI without mass effect (= "unidentified bright objects")8.Vacuolar / spongiotic myelinopathy (in 66%)Location:basal ganglia (esp. in globus pallidus), cerebellum, internal capsule, brainstem√ nonenhancing hyperintense foci on T2WI@Spine1.Paraspinal neurofibromas√ tumors of varying sizes at nearly every level throughout the spinal canal√ enlargement of neural foramina due to "dumbbell" neurofibroma of spinal nerves√ fusiform / spherical low-attenuation mass (20-30 HU)√ slightly hyperintense to muscle on T1WI, hyperintense periphery + hypointense core on T2WI√ hypoechoic well-circumscribed cylindrical lesion√ spinal cord displaced to contralateral side2.Lateral / intrathoracic meningocele=diverticula of thecal sac extending through widened neural foramina*Cause:*dysplasia of meninges focally stretched by CSF pulsationsLocation:thoracic level (most common)√ erosion of bony elements with marked posterior scalloping√ widening of neural foramina (due to protrusion of spinal meninges)
B.SKELETAL MANIFESTATIONS (in 30-40-80%) ● dwarfism caused by scoliosis@ Orbit √ Harlequin appearance to orbit = partial absence of greater and lesser wing of sphenoid bone + orbital plate of frontal bone (failure of development of membranous bone)√ hypoplasia + elevation of lesser wing of sphenoid√ defect in sphenoid bone ± extension of middle cranial fossa structures into orbit√ concentric enlargement of optic foramen (optic glioma)√ enlargement of orbital margins + superior orbital fissure (plexiform neurofibroma of peripheral and sympathetic nerves within orbit / optic nerve glioma)√ sclerosis in the vicinity of optic foramen (optic nerve sheath meningioma)√ deformity + decreased size of ipsilateral ethmoid + maxillary sinus@Skull√ macrocranium + macroencephaly√ calvarial defect adjacent to left lambdoid suture = parietal mastoid (rare)@ Spine √ sharply angled kyphoscoliosis (50%) in lower thoracic + lumbar spine; kyphosis predominates over scoliosis; incidence increases with age*Cause:*abnormal development of vertebral bodies√ hypoplasia of pedicles, transverse + spinous processes√ posterior scalloping of vertebral bodies with dural ectasia (secondary to weakened meninges allowing transmission of normal CSF pulsations)@ Chest √ twisted "ribbonlike" ribs in upper thoracic segments accompanying kyphoscoliosis√ localized cortical notches / depression of inferior margins of ribs (DDx: aortic coarctation)√ intrathoracic meningoceles√ lung + mediastinal neurofibromas√ progressive pulmonary interstitial fibrosis@ Appendicular skeleton √ anterolateral bowing of lower half of tibia (most common) / fibula (frequent) / upper extremity (uncommon) ± pseudarthrosis secondary to deossification with bowing-fracture in 1st year of life√ atrophic thinned / absent fibulas√ periosteal dysplasia = traumatic subperiosteal hemorrhage with abnormal easy detachment of periosteum from bone√ subendosteal sclerosis√ bone erosion from periosteal / soft-tissue neurofibromas√ intramedullary longitudinal streaks of increased density√ single / multiple cystic lesions within bone (? deossification / nonossifying fibroma)√ focal gigantism = unilateral overgrowth of a limb bone; marked enlargement of a digit in a hand / foot (overgrowth of ossification center)

C.NEURAL CREST TUMORS1.Pheochromocytoma: ● hypertension in adults2.Parathyroid adenomas: ● hyperparathyroidism

D.VASCULAR LESIONSSchwann cell proliferation within vessel wall 1.Cranial artery stenosis2.Renal artery stenosis: very proximal, funnel-shaped (one of the most common causes of hypertension in childhood)3.Renal artery aneurysm4.Thoracic / abdominal aortic coarctation
E.GI TRACT MANIFESTATIONS (10-25%) ● pain, intestinal bleeding ● obstruction (simulating Hirschsprung disease (with plexiform neurofibromas of colon)Location:jejunum > stomach > ileum > duodenum; retroperitoneal / paraspinal*Associated with:*increased prevalence of carcinoid tumors + GI stromal tumors(a)solitary pattern = single neurofibroma, neuroma, ganglioneuroma, schwannoma√ subserosal / submucosal filling defect ("mucosal ganglioneurofibromatosis")(b)plexiform pattern = regional enlargement of nerve root trunks√ mass effect on adjacent barium-filled loops√ multiple eccentric polypoid filling defects involving mesenteric side of small bowel√ mesenteric fat trapped within entangled network (15-30 HU) CHARACTERISTIC√ multiple leiomyomas ± ulcer*Cx:*intussusception
F.OCULAR MANIFESTATIONS (6%) ● pulsatile exophthalmos / unilateral proptosis (herniation of subarachnoid space + temporal lobe into orbit) ● buphthalmos = congenital glaucoma (aberrant mesodermal tissue obstructing canal of Schlemm)1.Plexiform neurofibroma (most common)2.Pigmented iris hamartomas <2 mm (Lisch nodules) in >90%, mostly bilateral; appear in childhood3.Optic glioma: in 12% of patients, in 4% bilateral; 75% in 1st decade√ extension into optic chiasm (up to 25%), optic tracts + optic radiation√ increased intensity on T2WI if chiasm + visual pathways involved4.Perioptic meningioma5.Choroidal hamartoma: in 50% of patients
G.SKIN MANIFESTATIONS1.Café-au-lait spotsof "coast of California" type (= smooth outline): ≥6 in number >5 mm in greatest diameter usually develop within 1st year of life / >15 mm in size in postpubertal individuals 2.Axillary freckling (in 66%)3.Cutaneous neurofibromabegin to appear around puberty (a)localized = fibroma molluscum = string of pearls along peripheral nerve(b)plexiform neurofibroma = elephantiasis neuromatosa
*Cx:*malignant transformation to malignant neurofibromas + malignant schwannomas (3-15%), glioma, xanthomatous leukemia

Notes:

Neurofibromatosis with Bilateral Acoustic Neuromas =NEUROFIBROMATOSIS TYPE 2 = NF-2=CENTRAL NEUROFIBROMATOSIS=rare autosomal dominant syndrome characterized by propensity for developing multiple schwannomas, meningiomas, and gliomas of ependymal derivation*mnemonic:*"MISME"**M**ultiple **I**nherited **S**chwannomas **M**eningiomas **E**pendymomas *Incidence:*1:50,000 births*Etiology:*deletion on the long arm of chromosome 22; in 50% new spontaneous mutation◊ Neurofibromatosis **2** is located on chromosome **22**!*Symptomatic age:*during 2nd / 3rd decade of life*Diagnostic criteria:* (1)Bilateral 8th cranial nerve masses(2)First-degree relative with unilateral 8th nerve mass, neurofibroma, meningioma, glioma (spinal ependymoma), schwannoma, juvenile posterior subcapsular lenticular opacity ● NO Lisch nodules, skeletal dysplasia, optic pathway glioma, vascular dysplasia, learning disability ● café-au-lait spots (<50%): pale, <5 in number ● cutaneous neurofibroma: minimal in size + number / absent@Intracranial1.Bilateral acoustic schwannomas (*sine qua non*)Site:superior / inferior division of vestibular n.√ usually asymmetric in size2.Schwannoma of other cranial nerves*Frequency:*trigeminal n. > facial n.◊Nerves without Schwann cells are excluded: olfactory nerve, optic nerve3.Multiple meningiomas: intraventricular in choroid plexus of trigone, parasagittal, sphenoid ridge, olfactory groove, along intracranial nerves4.Meningiomatosis = dura studded with innumerable small meningiomas5.Glioma of ependymal derivation@Spinal ● symptoms of cord compressionA.Extramedullary1.Multiple paraspinal neurofibromas2.Meningioma of spinal cord (thoracic region)B.Intramedullary1.Spinal cord ependymomas

Notes:

NEUROMA
*Prevalence:*8% of all intracranial tumors*Age:*20-50 years ● slow growth; not painful

Acoustic Neuroma Trigeminal Neuroma

Notes:

Acoustic Neuroma =VESTIBULAR SCHWANNOMA = ACOUSTIC SCHWANNOMA = NEURILEMMOMA◊Most common neoplasm of internal auditory canal / cerebellopontine angle!*Prevalence:*5-10% of all intracranial tumors; 85% of all intracranial neuromas; 80-90% of all cerebellopontine angle tumors*Age:*(a)sporadic tumor: 35-60 years; M:F = 1:2(b)type 2 neurofibromatosis: 2nd decade*Histo:* encapsulated neoplasm composed of proliferating fusiform Schwann cells with (a)highly cellular dense regions (Antoni A) with reticulin + collagen, and(b)loose areas with widely separated cells (Antoni B) in a reticulated myxoid matrix; common degenerative changes with cyst formation, vascular features, lipid-laden foam cells*May be associated with:*central neurofibromatosis◊Solitary intracranial schwannoma is associated with type 2 neurofibromatosis in 5-25%!◊Bilateral acoustic schwannomas allow a presumptive diagnosis of type 2 neurofibromatosis! ● long history of slowly progressive unilateral sensorineural hearing loss affecting high-frequency sounds more severely (in 95%) ● tinnitus ● diminished corneal reflex ● unsteadiness, vertigo, ataxia, dizziness (<10%) ● pain*Doubling time:*2 years
Location: (a)arises from within internal auditory canal (IAC)(b)may arise in cerebellopontine angle cistern at opening of IAC (= porus acusticus) with intracanalicular extension in 5%
Site:(a)in 85% from the vestibular portion of 8th nerve (around vestibular ganglion of Scarpa / at the glial-Schwann cell junction)(b)in 15% from the cochlear portion √ round mass centered on long axis of IAC forming acute angles with petrous bone√ funnel-shaped component extending into IAC√ IAC enlargement / erosion (70-90%)√ widening / obliteration of ipsilateral cerebellopontine angle cistern√ shift / asymmetry of 4th ventricle with hydrocephalus√ degenerative changes (cystic areas ± hemorrhage) with tumors >2-3 cmPlain film: √ erosion of IAC: a difference in canal height of >2 mm is abnormal + indicates a schwannoma in 93%CT: √ isodense small / hypodense large solid tumor√ cyst formation in tumor (= central necrosis) / adjacent to tumor (= extramural arachnoid cyst) in 15% of large tumors√ usually uniformly dense tumor enhancement with small tumors (50% may be missed without CECT) / ring enhancement with large tumors√ NO calcification√ intrathecal contrast / carbon dioxide insufflation (for tumors <5 mm)MR (most sensitive test with Gd-DTPA enhancement): √ iso- / slightly hypointense on T1WI relative to brain√ intensely enhancing homogeneous mass / ringlike enhancement (if cystic) after Gd-DTPA√ hyperintense on T2WI (DDx: meningioma remains hypo- / isointense)Angio: √ elevation + posterior displacement of anterior inferior cerebellar artery (AICA) on basal view√ elevation of the superior cerebellar artery (large tumors)√ displacement of basilar artery anteriorly / posteriorly + contralateral side√ compression / posterior + lateral displacement of petrosal vein√ posterior displacement of choroid point of PICA√ vascular supply frequently from external carotid artery branches√ rarely hypervascular tumor with tumor blush*DDx:*ossifying hemangioma (bony spiculations)

Notes:

Trigeminal Neuroma =TRIGEMINAL SCHWANNOMA*Incidence:*2-5% of intracranial neuromas, 0.26% of all brain tumors*Origin:*arising from gasserian ganglion within Meckel cave at the most anteromedial portion of the petrous pyramid / trigeminal nerve root*Age:*35- 60 years; M:F = 1:2Symptoms of location in middle cranial fossa: ● facial paresthesia / hypesthesia ● exophthalmos, ophthalmoplegiaSymptoms of location in posterior cranial fossa: ● facial nerve palsy ● hearing impairment, tinnitus ● ataxia, nystagmusLocation:(in any segment of trigeminal nerve)(a)middle cranial fossa (46%) = gasserian ganglion(b)posterior cranial fossa (29%)(c)in both fossae (25%)(d)pterygoid fossa / paranasal sinuses (10%)√ erosion of petrous tip√ enlargement of contiguous fissures, foramina, canals√ dumbbell / saddle-shaped mass (extension into middle cranial fossa + through tentorial incisura into posterior fossa)√ isodense mass with dense inhomogeneous enhancement (tumor necrosis + cyst formation)√ distortion of ipsilateral quadrigeminal cistern√ displacement + cutoff of posterior 3rd ventricle√ anterior displacement of temporal horn√ angiographically avascular / hypervascular mass

Notes:

OLIGODENDROGLIOMA
=uncommon form of slowly growing glioma; presenting with large size at time of diagnosis*Incidence:*2-10% of intracranial gliomas; 5-7% of all primary intracranial neoplasms*Histo:*mixed glial cells (50%), astrocytic components (30%); hemorrhage + cyst formation infrequent*Age:*30-50 years ● seizuresLocation:most commonly in cerebral hemispheres (propensity for periphery of frontal lobes) involving cortex + white matter, thalamus, corpus callosum; occasionally around / in ventricles ("subependymal oligodendroglioma") rare in cerebellum + spinal cord√ large nodular clumps of calcifications (in 45% on plain film; in 90% on CT)CT: √ round / oval hypodense lesion with mass effect (75%)√ commonly no / minimal tumor enhancement (75%), pronounced in high-grade tumors√ may be adherent to dura (mimicking meningiomas)√ ± erosion of inner table of skull√ cystic changes (uncommon)√ edema (in 50% of low-grade, in 80% of high-grade tumors)MR: √ well-circumscribed heterogeneous hypointense lesion on T1WI + hyperintense on T2WI√ little edema / mass effect√ solid / peripheral / mixed enhancement√ calcification may not be detected*Cx:*malignant metaplasia + CSF seeding*DDx:*(1)Astrocytoma (no large calcifications)(2)Ganglioglioma (in temporal lobes + deep cerebral tissues(3)Ependymoma (enhancing tumor, often with internal bleeding producing fluid levels)(4)Glioblastoma (infiltrating, enhancing, edema, no calcifications)

Notes:

PARAGONIMIASIS OF BRAIN

Oriental lung fluke (Paragonimus westermani) producing arachnoiditis, parenchymal granulomas, encapsulated abscesses √ isodense / inhomogeneous masses surrounded by edema√ ring enhancement

Notes:

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PELIZAEUS-MERZBACHER DISEASE
= rare X-linked sudanophilic leukodystrophy (5 types with different times of onset, rate of progression, genetic transmission) *Age:*neonatal period ● bizarre pendular nystagmus + head shaking ● cerebellar ataxia ● slow psychomotor developmentCT: √ hypodense white matter√ progressive white matter atrophyMR: √ lack of myelination (appearance of newborn retained)√ hyperintense internal capsule, optic radiations, proximal corona radiata on T1WI√ near complete absence of hypointensity in supratentorial region on T2WI√ mild / moderate prominence of cortical sulci*Prognosis:*death in adolescence / early adulthood

Notes:

PICK DISEASE

= rare form of presenile dementia similar to Alzheimer disease; may be inherited with autosomal dominant mode; M < F √ focal cortical atrophy of anterior frontal + anterior temporal lobes √ dilatation of frontal + temporal horns of lateral ventricle

Notes:

PINEAL CYST
= small nonneoplastic cyst of pineal gland *Incidence:*25-40% on autopsy, 4% on MRI*Types:* (a)developmental = persistence of ependymal-lined pineal diverticulum(b)degenerative = glial-lined secondary cavitation within area of gliosis ● never associated with Parinaud syndrome ● never cause of hydrocephalus ● may be symptomatic when largeCT: √ normal-sized gland (80%), slightly >1 cm in 20%√ isodense to CSF in surrounding cistern (infrequently noted)MR: √ sharply marginated ovoid mass in pineal region√ slight impression on superior colliculi (sagittal image)√ isointense to CSF on T1WI + slightly hyperintense to CSF on T2WI (due to phase coherence in cysts but not in moving CSF)√ may have higher signal intensity than CSF due to high protein content√ contrast may diffuse from enhanced rim of residual pineal tissue into fluid center (no blood-brain barrier) on delayed sequence images

Notes:

PINEAL GERMINOMA
=DYSGERMINOMA = PINEALOMA = ATYPICAL TERATOMA (former inaccurate names)√"pinealoma" = misnomer referring to any pineal mass= malignant primitive germ cell neoplasm *Incidence:*most common pineal tumor (>50% of all pineal tumors)*Histo:*identical to testicular seminoma + ovarian dysgerminoma, NO capsule facilitates invasion*Age:*10-25 years; M:F = 10:1*May be associated with:*ectopic pinealoma = secondary focus in inferior portion of 3rd ventricle ● precocious puberty frequent in children <10 years of age ● Parinaud syndrome = paralysis of upward gaze (compression of mesencephalic tectum)Location of germinomas:pineal gland (80%), suprasellar region (20%), basal ganglia, thalamus√ displacement of calcified pineal gland√ hydrocephalus (compression of aqueduct of Sylvius)√ well-defined lesion restricted to pineal gland√ may infiltrate quadrigeminal plate / thalamus CT: √ infiltrating variodense homogeneous mass (attenuation usually similar to gray matter)√ rarely psammomatous calcifications within tumor, but pineal calcifications in 100% (40% in normal population)√ moderate / marked uniform contrast enhancementMR: √ round / lobular well-circumscribed relatively homogeneous mass isointense to gray matter√ hypointense mass on T2WI (occasionally)√ strong Gd-DTPA enhancement*Cx:*metastatic spread via CSF (frequent)*Rx:*combination of irradiation (very radiosensitive) + chemotherapy (adriamycin, cisplatin, cyclophosphamide)*Prognosis:*75% survival after radiation therapy alone

Notes:

PINEAL TERATOCARCINOMA
=highly malignant variant of germ cell tumors*Types:*1.Choriocarcinoma2.Embryonal cell carcinoma3.Endodermal sinus tumor*Histo:*arising from primitive germ cells, frequently containing more than one cell type*Age:*<20 years; males ● Parinaud syndrome ● tumor markers elevated in serum + CSF√ intratumoral hemorrhage (esp. choriocarcinoma)√ invasion of adjacent structures√ intense homogeneous contrast enhancement*Cx:*seeding via CSF

Notes:

PINEAL TERATOMA
=benign tumor containing one / all three germ cell layers (pineal region most common site of teratomas)*Incidence:*15% of all pineal masses (2nd most common tumor in pineal region)*Age:*<20 years; M:F = 2-8:1 ● Parinaud syndrome = paralysis of upward gaze (compression / infiltration of superior colliculi) ● hypothalamic symptoms ● headache ● somnolence (related to hydrocephalus)Location:pineal, parapineal, suprasellar, 3rd ventricle√ well-defined rounded / irregular lobulated extremely heterogenous mass of fat, cartilage, hair, linear / nodular calcifications + cysts◊Fat is absent in all other pineal tumors!√ may show heterogeneous / rimlike contrast enhancement (limited to solid-tissue areas)Angio: √ elevation of internal cerebral vein√ posterior displacement of precentral veinCT: √ heterogeneous mass with fat, calcification, cystic + solid areasMR: √ variegated appearance on all pulse sequences with hyperintense areas of fat on T1WI*Cx:*chemical meningitis with spontaneous rupture

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PINEOBLASTOMA
=highly malignant tumor derived from primitive pineal parenchymal cells*Histo:*unencapsulated highly cellular primitive small round cell tumor (similar to medulloblastoma, neuroblastoma, retinoblastoma)*Age:*any age, more common in children; M < FCT: √ poorly marginated iso- / slightly hyperdense mass√ may contain dense tumor calcifications√ peripherally displaced preexisting normal pineal calcification (= "exploded pineal pattern") √ intense homogeneous contrast enhancementMR: √ iso- / moderately hypointense on T1WI + iso- / hyperintense on T2WI√ dense homogeneous Gd-DTPA enhancement*Spread:* (1)direct extension posteriorly with invasion of cerebellar vermis + anteriorly into 3rd ventricle(2)throughout CSF (frequent) along meninges / via ventricles

Notes:

PINEOCYTOMA

=rare slow-growing unencapsulated tumor composed of mature pineal parenchymal cells*Age:*any age; M:F = 1:1√ well-marginated slightly hyperdense / isodense mass √ dense focal tumor calcifications possible√ peripherally displaced preexisting normal pineal calcification (= "exploded pineal pattern") √ well-defined homogeneous enhancementMR: √ intermediate intensity on T1WI + T2WI√ may be isointense to CSF but containing trabeculations (DDx to pineal cyst)√ mild to moderate Gd-DTPA enhancement*Cx:*some metastasize via CSF

Notes:

PITUITARY ADENOMA

=benign slow-growing neoplasm arising fromadenohypophysis (= anterior lobe); most common tumor of adenohypophysis*Prevalence:*5-10-18% of all intracranial neoplasms ● pituitary hyperfunction / hypofunction / visual field defect

FORMER CLASSIFICATION: (a)Chromophobe adenoma (80%)associated with hypopituitarism; elevation of prolactin, TSH, GH serum levels √ greatest sella enlargement; calcified in 5%however: functioning microadenomas are part of chromophobe adenomas(b)Acidophilic / eosinophilic adenoma (15%)increased GH secretion (acromegaly), prolactin, TSH √ tumor of intermediate size(c)Basophilic adenoma (5%)associated with ACTH secretion (Cushing syndrome), LH, FSH √ small tumor

Plain film: (UNRELIABLE !) √ enlargement of sella + sloping of sella floor√ erosion of anterior + posterior clinoid processes√ erosion of dorsum sellae√ calcification in <10%√ may present with mass in nasopharynx

Functioning Pituitary Adenoma Nonfunctioning Pituitary Adenoma Pituitary Macroadenoma Pituitary Microadenoma

Notes:

Functioning Pituitary Adenoma Adenoma may secrete multiple hormones! 1.PROLACTINOMA (30%)most common of pituitary adenomas; approximately 50% of all cranial tumors at autopsy; M << F ● prolactin levels do not closely correlate with tumor size◊Any mass compressing the hypothalamus / pituitary stalk diminishes the tonic inhibitory effect of dopaminergic factors, which originate there, resulting in hyperprolactinemia!
Female: *Age:*15-44 years (during childbearing age) ● infertility ● amenorrhea ● galactorrhea ● elevated prolactin levels (normal <20 ng/mL)◊>75% of patients with serum prolactin levels >200 ng/mL will show a pituitary tumor!
Male: ● headache ● impotence + decreased libido ● visual disturbance √ characteristic lateral location, anteriorly / inferiorly; variable in size*Rx:*bromocriptine
2.CORTICOTROPHIC ADENOMA (14%)*Function:*ACTH-secreting tumor*Age:*30-40 years; M:F = 1:3√ central location; posterior lobe; usually <5 mm in size√ sampling of inferior petrosal sinuses (95% diagnostic accuracy compared with 65% for MRI) ● **Cushing disease**=truncal obesity, abdominal striae, glycosuria, osteoporosis, proximal muscle weakness, hirsutism, amenorrhea, hypertension, elevated cortisol levels in plasma and urine*Rx:*(1)suppression by high doses of dexamethasone of 8 mg/day(2)surgical resection difficult because ACTH adenomas usually require resection of an apparently normal gland (tumor small + usually not on surface)
3.SOMATOTROPHIC ADENOMA (14%) ● gigantism, acromegaly, elevated GH >10 ng/mL, no rise in GH after administration of glucose / TRH*Histo:*(a)densely granulated type(b)sparsely granulated type: clinically more aggressive√ hypodense region, may be less well-defined, variable size
4.GONADOTROPH CELL ADENOMA (7%)secrets follicle-stimulating hormone (FSH) / luteinizing hormone (LH) √ slow-growing often extending beyond sella
5.THYROTROPH CELL ADENOMA (<1%)secrets thyroid-stimulating hormone (TSH) √ often large + invasive pituitary adenoma
6.PLURIHORMONAL PITUITARY ADENOMA (>5%)
CECT (dynamic bolus injection): √ upward convexity of gland√ increased height >10 mm√ deviation of pituitary stalk√ floor erosion of sella√ gland asymmetry√ focal hypodensity (most specific for adenoma)√ shift of pituitary tuft / density change in region of adenomaMR: Highest sensitivity on coronal nonenhanced T1WI (70%) + 3 D FLASH sequence (69%) + combination of both (90%) ◊1/3 of lesions are missed with enhancement◊1/3 of lesions are missed without enhancement√ focus of low signal intensity on T1WI√ focus of high-signal intensity on T2WI√ focal hypointensity within normally enhancing gland*DDx:*simple pituitary cyst (= Rathke cleft cyst)

Notes:

Nonfunctioning Pituitary Adenoma 1.NULL CELL ADENOMA=hormonally inactive pituitary tumor with no histologic / immunologic / ultrastructural markers to indicate its cellular derivation*Prevalence:*17% of all pituitary tumors*Age:*older patient√ slow-growing2.ONCOCYTOMA*Prevalence:*10% of all pituitary tumors ● clinically + morphologically similar to null cell adenoma

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Pituitary Macroadenoma =tumor >10 mm in size, usually endocrinologically inactive (70-80% of pituitary adenomas)*Incidence:*10%; M:F = 1:1*Age:*2560 years ● symptoms of mass effect: hypopituitarism, bitemporal hemianopia (with superior extension), pituitary apoplexy, hydrocephalus, cranial nerve involvement (III, IV, VI)Extension into:suprasellar cistern / cavernous sinus / sphenoid sinus + nasopharynx (up to 67% are invasive)√ occasionally tumor hemorrhage√ lucent areas correspond to cysts / focal necrosis√ invasion of cavernous sinus: encasement of carotid artery (surest sign)CT: √ tumor isodense to brain tissue√ erosion of bone (eg, floor of sella)√ calcifications infrequentMR: (allows differentiation from aneurysm) √ homogeneous enhancement*Cx:* (1)Obstructive hydrocephalus (at foramen of Monro)(2)Encasement of carotid artery(3)Pituitary apoplexy (rare)*DDx:* (1)Metastasis (more bone destruction, rapid growth)(2)Pituitary abscess

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Notes:

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Pituitary Microadenoma =very small adenomas <10 mm ● usually become clinically apparent by hormone production (20-30% of all pituitary adenomas)◊prolactin elevation (>25 ng/mL in females)4-8 x normal:adenoma demonstrated in 71%>8 x normal:adenoma demonstrated in 100% ● **incidentaloma** = nonfunctioning microadenoma / pituitary cyst√ NO imaging features to distinguish between different types o adenomasMRI: √ small mass of hypointensity on pre- and postcontrast T1WI (nonenhancing)√ occasionally isointense on precontrast images + hyperintense on postcontrast images√ enhancement on delayed images√ focal bulge on surface of gland√ focal depression of sellar floor√ deviation of pituitary stalk

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Notes:

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PITUITARY APOPLEXY
*Cause:*massive hemorrhage into pituitary adenoma (especially in patients on bromocriptine for pituitary adenoma) / dramatic necrosis / sudden infarction of pituitary gland◊25% of patients with pituitary hemorrhage will present with apoplexy!**Sheehan syndrome** = postpartum infarction of anterior pituitary gland ● severe headache, nausea, vomiting ● hypertension ● stiff neck ● sudden visual-field defect, ophthalmoplegia ● obtundation (frequent) ● hypopituitarism (eg, secondary hypothyroidism)◊ Area of destruction must be >70% to produce pituitary insufficiency!√ enlargement of pituitary glandNCCT: √ increased density ± fluid levelMR: √ bright signal from presence of hemoglobin on T1WI with persistence over hyperintensity on T2WI√ intermediate signal intensity from deoxyhemoglobin on T1WI + T2WI

Notes:

PORENCEPHALY
=focal cavity as a result of localized brain destructionA.AGENETIC PORENCEPHALY = Schizencephaly (= true porencephaly) B.ENCEPHALOCLASTIC PORENCEPHALY*Time of injury:*during first half of gestation*Histo:*necrotic tissue completely reabsorbed without surrounding glial reaction (= liquefaction necrosis)MR: √ smooth-walled cavity filled with CSF on all pulse sequences (= porencephalic cyst)√ lined by white matterC.ENCEPHALOMALACIA=Pseudoporencephaly = Acquired porencephaly*Cause:*infectious, vascular*Time f injury:*after end of 2nd trimester (brain has developed capacity for glial response)Location:parasagittal watershed areas with sparing of periventricular region + ventricular wall CT: √ hypodense regionsMR: √ hypointense on T1WI + hyperintense on T2WI√ surrounding hyperintense rim on T2WI = gliosis)√ glial septa coursing through cavity identified on T1WI + proton density imagesUS: √ septations in cavity well visualized

Notes:

POSTVIRAL LEUKOENCEPHALOPATHY
=ACUTE DISSEMINATED ENCEPHALOMYELITIS=autoimmune process ● several weeks following an exanthematous viral infection / vaccination (measles, rubella, chickenpox, Epstein-Barr virus, mumps, pertussis) ● seizures, focal neurologic deficits√ multifocal white matter abnormalities, occasionally deep gray matter involvement√ sparing of cortical gray matter√ no additional lesions on follow-up exam*Prognosis:*resolution of neurologic deficits within 1 month (80-90%)

Notes:

PRIMITIVE NEUROECTODERMAL TUMOR
=PNET = group of very undifferentiated tumors arising from germinal matrix cells of primitive neural tube*Incidence:*<5% of supratentorial neoplasms in children*Age:*mainly in children <5 years of age; M:F = 1:1*Histo:*highly cellular tumors composed of >90-95% of undifferentiated cells (histologically similar to medulloblastoma, pineoblastoma, peripheral neuroblastoma) ● signs of increased intracranial pressure / seizuresLocation: (a)supratentorial: deep cerebral white matter (most commonly in frontal lobe), pineal gland, in thalamic + suprasellar territories (least frequently)(b)posterior fossa (= medulloblastoma)
√ large cellular lesion with tendency for necrosis (65%), cyst formation, calcifications (71%), hemorrhage (10%)√ thin rim of edema√ contrast enhancement of solid tumor portionCT: √ solid tumor portions hyperdense (due to high nuclear to cytoplasmic ratio)MR: √ mildly hypointense on T1WI + hyperintense on T2WI√ remarkably inhomogeneous due to cyst formation + necrosis√ areas of signal dropout due to calcifications√ hyperintense areas on T1WI + variable intensity on T2WI due to hemorrhage√ inhomogeneously enhancing mass with tumor nodules + ringlike areas surrounding central necrosis after Gd-DTPA

Notes:

PROGRESSIVE MULTIFOCAL LEUKOENCEPHALOPATHY

=PML = rapidly progressive fatal demyelinating disease in patients with impaired immune system (chronic lymphocytic leukemia, lymphoma, Hodgkin disease, carcinomatosis, AIDS, tuberculosis, sarcoidosis, organ transplant)*Etiology:*virus infection (probably latent papovavirus= JC virus) *Pathophysiology:*destruction of oligodendrogliocytes leading to areas of demyelination + edema*Histo:*intranuclear inclusion bodies within swollen oligodendrocytes (viral particles in nuclei), absence of significant perivenous inflammation ● progressive neurologic deficits, visual disturbances, dementia, ataxia, spasticity ● normal CSF fluidLocation:predilection for parietooccipital regionSite:subcortical white matter spreading centrally√ NO contrast enhancementCT: √ multicentric confluent white matter lesions of low attenuation with scalloped borders along cortex√ NO mass effectMR: √ patchy high-intensity lesions of white matter away from ependyma in asymmetric distribution on T2WI√ sparing of cortical gray matter*Prognosis:*death usually within 6 months*DDx in early stages:*primary CNS lymphoma

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REYE SYNDROME
=hepatitis + encephalitis following viral upper respiratory tract infection with Hx of large doses of aspirin ingestion*Age:*in children + young adults ● obtundation rapidly progressing to coma√ initially (within 2-3 days) small ventricles√ later progressive enlargement of lateral ventricles + sulci√ markedly diminished attenuation of white matter*Mortality:*15-85% (from white matter edema + demyelination)*Dx:*liver biopsy

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Notes:

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SARCOIDOSIS OF CNS

=inflammatory disorder characterized by presence of noncaseating granulomas; mostly in Blacks*Incidence:*CNS involvement in 1-8% (in up to 15% of autopsies) ● cranial neuropathy (facial > acoustic > optic > trigeminal nerves) secondary to granulomatous infiltration + leptomeningeal fibrosis (50-75%) ● peripheral neuropathy + myopathy ● aseptic meningitis (20%) ● diffuse encephalopathy, dementia ● pituitary + hypothalamic dysfunction (eg, diabetes insipidus in 5-10%) ● generalized / focal seizures (herald poorer prognosis) ● multiple sclerosislike symptoms (from multifocal parenchymal involvement) ● prompt improvement following therapy with steroidsLocation:dura mater, leptomeninges, subarachnoid space, peripheral nerves, brain parenchyma, ventricular system√Affects meninges + cranial nerves more often than the brain!√ diffuse meningeal enhancement (most common) / meningeal nodules (less common) from leptomeningeal invasionSite:particularly in basal cisterns (suprasellar, sellar, subfrontal regions) with extension to optic chiasm, hypothalamus, pituitary gland, cranial nerves where exiting brainstem√ focal / widespread infarcts of peripheral gray matter / at gray-white matter junction (periarteritis)√ dense enhancement of falx + tentorium (granulomatous invasion of dura)√ isodense / hyperdense homogeneously enhancing small single / multiple nodules (invasion of brain parenchyma via perivascular spaces of Virchow-Robin)Site:periphery of parenchyma, intraspinal√ communicating / obstructive hydrocephalus is the most common finding (from arachnoiditis / adhesions)

Notes:

SCHIZENCEPHALY

=AGENETIC PORENCEPHALY = TRUEPORENCEPHALY = "split brain"=full-thickness CSF-filled parenchymal cleft lined by gray matter extending from subarachnoid space to subependyma of lateral ventricles*Frequency:*1:1,650*Cause:*segmental developmental failure of cell migration to form cerebral cortex / vascular ischemia of portion of germinal matrix*Time of injury:*30-60 days of gestation*Often associated with:*polymicrogyria, microcephaly, gray matter heterotopia*Types:* (a)clefts with fused lips(may be missed in imaging planes parallel to the plane of cleft) √ walls appose one another obliterating CSF space(b)clefts with separated / open lips √ CSF fills cleft from lateral ventricle to subarachnoid space ● seizure disorder ● mild / moderate developmental delay ● range of normal mentation to severe mental retardation ● blindness possible (optic nerve hypoplasia in 33%)Location:most commonly near pre- and postcentral gyri (sylvian fissure); uni- / (mostly) bilateral; in middle cerebral artery distribution √ polymicrogyria / pachygyria of cortex adjacent to cleft √ full-thickness cleft through hemisphere with irregular margins √ gray-matter lining of cleft (PATHOGNOMONIC) extending through entire hemisphere √ bilateral often symmetric intracranial cysts, usually around sylvian fissure √ asymmetrical dilatation of lateral ventricles with midline shift √ wide separation of lateral ventricles + squaring of frontal lobes √ absence of cavum septi pellucidi (80 - 90%) + corpus callosum*Prognosis:*severe intellectual impairment, spastic tetraplegia, blindness*DDx:*(1)Pseudoporencephaly = Acquired porencephaly= local parenchymal destruction secondary to vascular / infectious / traumatic insult (almost always unilateral)(2)Arachnoid cyst(3)Cystic tumor

Notes:

SEPTO-OPTIC DYSPLASIA
=DeMORSIER SYNDROME=rare anterior midline anomaly with (1) hypoplasia of optic nerves (2) hypoplasia / absence of septum pellucidum; often considered a mild form of lobar holoprosencephalyM:F = 1:3 *Cause:*insult between 5-7th week of GA*Associated with:*schizencephaly (50%) ● hypothalamic hypopituitarism (66%):diabetes insipidus (in 50%), growth retardation (deficient secretion of growth hormone + thyroid stimulating hormone) ● diminished visual acuity (hypoplasia of optic discs), nystagmus, occasionally hypotelorism ● seizures, hypotonia√ small optic canals√ hypoplasia of optic nerves + chiasm + infundibulum√ dilatation of chiasmatic + suprasellar cisterns√ fused dilated boxlike frontal horns squared off dorsally + pointing inferiorly√ bulbous dilatation of anterior recess of 3rd ventricle√ hypoplastic / absent septum pellucidum√ thin corpus callosum

Notes:

SINUS PERICRANII

=subperiosteal venous angiomas adherent to skull and connected by anomalous diploic veins to a sinus / cortical vein ● soft painless scalp mass that reduces under compressionLocation:frontal bone√ calvarial thinning + defectCT: √ sessile sharply marginated homogeneous densely enhancing mass adjacent to outer table of skull, perforating it and connecting it with another similar structure beneath the inner tableAngio: √ extracalvarial sinus may not opacify secondary to slow flow

Notes:

SPONGIFORM LEUKOENCEPHALOPATHY

rare, hereditary, > age 40 ● deteriorating mental function√ confluent areas of diminished attenuation

Notes:

STURGE-WEBER-DIMITRI SYNDROME
=ENCEPHALOTRIGEMINAL ANGIOMATOSIS=MENINGOFACIAL ANGIOMATOSIS= vascular malformation with capillary venous angiomas involving face, choroid of eye, leptomeninges *Cause:*persistence of transitory primordial sinusoidal plexus stage of vessel development; usually sporadic ● seizures (80%) in 1st year of life: usually focal involving the side of the body contralateral to nevus flammeus ● mental deficiency (>50%) ● increasing crossed hemiparesis (35-65%) ● hemiatrophy of body contralateral to facial nevus (secondary to hemiparesis) ● homonymous hemianopia@FACIAL MANIFESTATION ● congenital facial port-wine stain (nevus flammeus)=telangiectasia of trigeminal region; usually 1st ± 2nd division of 5th nerve; usually unilateral-V_1 associated with occipital lobe angiomatosis-V_2 associated with parietal lobe angiomatosis-V_3 associated with frontal lobe angiomatosis@CNS MANIFESTATION√ leptomeningeal venous angiomas confined to pia materLocation: parietal > occipital > frontal lobes Angio: √ capillary blush√ abnormally large veins in subependymal + periventricular regions√ abnormal deep medullary veins draining into internal cerebral vein (= venous shunt)√ failure to opacify superficial cortical veins in calcified region (markedly slow blood flow / thrombosis of dysgenetic superficial veins)√ cortical hemiatrophy beneath meningeal angioma due to anoxia (steal)√ "tram track" gyriform cortical calcifications >2 years of age; in layers 2-3(-4-5) of opposing gyri underlying pial angiomatosis; bilateral in up to 20%Location:temporo-parieto-occipital area, occasionally frontal, rare in posterior fossa√ subjacent white matter hypodense on CT with slight prolongation of T1 + T2 relaxation times (gliosis)√ choroid plexus enlargement ipsilateral to angiomatosis√ ipsilateral thickening of skull + orbit (bone apposition as result of subdural hematoma secondary to brain atrophy)√ elevation of sphenoid wing + petrous ridge√ enlarged ipsilateral paranasal sinuses + mastoid air cells√ thickened calvarium (= widening of diploic space)@ORBITAL MANIFESTATION (30%)ipsilateral to nevus flammeus: ● congenital glaucoma (30%)√ choroidal hemangioma (71%)√ dilatation + tortuosity of conjunctival + episcleral + iris + retinal vessels√ buphthalmos = enlarged + elongated globe as result of increased intraocular pressure*Cx:*retinal detachment@VISCERAL MANIFESTATIONlocalized / diffuse angiomatous malformation located in intestine, kidneys, spleen, ovaries, thyroid, pancreas, lungs
*DDx:*Klippel-Trenaunay syndrome, Wyburn-Mason syndrome

Notes:

SUBARACHNOID HEMORRHAGE

Cause: A.Spontaneous(1) ruptured aneurysm (72%) (2) AV malformation (10%) (3) hypertensive hemorrhage (4) hemorrhage from tumor (5) embolic hemorrhagic infarction (6) blood dyscrasia, anticoagulation therapy (7) eclampsia (8) intracranial infection (9) spinal vascular malformation (10) cryptogenic in 6% (negative 4-vessel angiography; seldom recurrent) B.Trauma (common)concomitant to cerebral contusion (a)injury to leptomeningeal vessels at vertex(b)rupture of major intracerebral vessels (less common)Location:(a)focal, overlying site of contusion(b)interhemispheric fissure, paralleling falx cerebri(c)spread diffusely throughout subarachnoid space (rare in trauma)*Pathophysiology:*irritation of meninges by blood + extra fluid volume increases intracranial pressure ● acute severe headache ("worst in life"), vomiting ● altered state of consciousness: drowsiness, sleepiness, stupor, restlessness, agitation, coma ● spectrophotometric analysis of CSF obtained by lumbar puncture

NCCT (60-90% accuracy of detection depending on time of scan; sensitivity depends on amount of blood; accuracy high within 4-5 days of onset): ◊May occur in only two locations if subtle!√ increased density in basal cisterns, superior cerebellar cistern, sylvian fissure, cortical sulci, intraventricular, intracerebral√ along interhemispheric fissure = on lateral aspect irregular dentate pattern due to extension into paramedian sulci with rapid clearing after several daysMR (relatively insensitive within first 48 hours): √ deoxyhemoglobin effects not appreciable in acute phase (secondary to higher oxygen tension in CSF, counterbalancing effects of very long T2 of CSF, pulsatile flow effects of CSF)√ low-signal intensity on brain surfaces in recurrent subarachnoid hemorrhages (hemosiderin deposition)*Prognosis:*clinical course depends on amount of subarachnoid blood*Cx:* (1)Acute obstructive hydrocephalus (in <1 week) secondary to intraventricular hemorrhage / ependymitis obstructing aqueduct of Sylvius or outlet of 4th ventricle(2)Delayed communicating hydrocephalus (after 1 week) secondary to fibroblastic proliferation in subarachnoid space and arachnoid villi interfering with CSF resorption(3)Cerebral vasospasm + infarction (develops after 72 hours, at maximum between 5-17 days, amount of blood is prognostic parameter)(4)Transtentorial herniation (cerebral hematoma, hydrocephalus, infarction, brain edema)

Notes:

SUBDURAL HEMATOMA OF BRAIN

*Incidence:*in 5% of head trauma patients; in 15% of closed head injuries; in 65% of head injuries with prolonged interruption of consciousness*Age:*predominantly in infants + elderly (large subarachnoid space with freedom to move in cerebral atrophy)*Cause:*direct trauma, sudden de-/acceleration; forceful coughing / sneezing / vomiting in elderly; occasionally in blood clotting disorder / during anticoagulation therapy◊No consistent relationship to skull fractures!*Pathogenesis:* differential movement of brain + adherent cortical veins with respect to skull + attached dural sinuses tears the "bridging veins" (= subdural veins), which connect cerebral cortex to dural sinuses and travel through the subarachnoid and subdural space Location:subdural space = potential space between pia-arachnoid membrane (leptomeninges) + dura mater; freely extending across suture lines, limited only by interhemispheric fissure and tentorium*DDx:*(1)Arachnoid cyst (extension into sylvian fissure)(2)Subarachnoid hemorrhage (extension into sulci)

Acute Subdural Hematoma Subacute Subdural Hematoma Chronic Subdural Hematoma

Notes:

Acute Subdural Hematoma Usually follows severe trauma, manifest within hours after injury Time frame:<3-4 days old*Associated with:*underlying brain injury (50%) with worse long-term prognosis than epidural hematoma, skull fracture (1%)Location: (a)over cerebral convexity, frequent extension into interhemispheric fissure, along tentorial margins, beneath temporal + occipital lobes; NO crossing of midline(b)bilateral in 15-25% of adults (common in elderly) and in 80-85% in infants√ extra-axial peripheral crescentic fluid collection between skull and cerebral hemisphere usually with√ concave inner margin (hematoma minimally pressing into brain substance)√ convex outer margin following normal contour of cranial vault√ occasionally with blood-fluid level√ after surgical evacuation: underlying parenchymal injury becomes more obvious √ after healing: ventricular + sulcal enlargementCT: √ hyperdense (<1 week) / isodense (1-2 weeks) / hypodense (3-4 weeks)False-negative CT scan: high-convexity location, beam-hardening artifact, volume averaging with high density of calvarium obscuring flat "en plaque" hematoma, too narrow window setting, isodense hematoma due to delay in imaging 10-20 days post injury / due to low hemoglobin content of blood / lack of clotting, CSF-dilution from associated arachnoid tear ◊38% of small subdural hematomas are missed!Aids in detection of acute subdural hematoma: √ thickening of ipsilateral portion of skull (hematoma of similar pixel brightness as bone)√ "subdural window" setting = window level of 40 HU + window width of 400 HU√ effacement of adjacent sulci√ sulci not traceable to brain surface √ ipsilateral ventricular compression / distortion√ displacement of gray-white matter interface away from ipsilateral inner table√ midline shift (often greater than width of subdural hematoma due to underlying brain contusion)√ contrast enhancement of cortex but not of subdural hematomaAids in detection of bilateral subdural hematomas: √ "parentheses" ventricles√ ventricles too small for patients ageMR:refer to HEMATOMA OF BRAINUS (neonate): Limitations: (a)convexity hematoma may be obscured by pie-shaped display + loss of near-field resolution◊Use contralateral transtemporal approach!(b)small loculations may be missed√ linear / elliptical space between cranial vault + brain√ flattened gyri + prominent sulci√ ± distortion of ventricles, extension into interhemispheric space*Cx:*Arteriovenous fistula (meningeal artery + vein caught in fracture line)*Prognosis:*may progress to subacute + chronic stage / may disappear spontaneously*Mortality:*35-50% (higher number due to associated brain injury, mass effect, old age, bilateral lesions, rapid rate of hematoma accumulation, surgical evacuation >4 hours)

Interhemispheric Subdural Hematoma Most common acute finding in child abuse (whiplash forces on large head with weak neck muscles) √ predominance for posterior portion of interhemispheric fissure√ crescentic shape with flat medial border√ unilateral increased attenuation with extension along course of tentorium√ anterior extension to level of genu of corpus callosum

Subdural Hemorrhage in Newborn *Cause:*mechanical trauma during delivery (excessive vertical molding of head)1.Posterior fossa hemorrhage(a)tentorial laceration with rupture of vein of Galen / straight sinus / transverse sinus(b)occipital osteodiastasis = separation of squamous portion from exoccipital portion of occipital bone√ high-density thickening of affected tentorial leaf extending down posterior to cerebellar hemisphere (better seen on coronal views)√ mildly echogenic subtentorial collection*Cx:*death from compression of brainstem, acute hydrocephalus2.Supratentorial hemorrhage(a)laceration of falx near junction with tentorium with rupture of inferior sagittal sinus (less common than tentorial laceration)√ hematoma over corpus callosum in inferior aspect of interhemispheric fissure(b)convexity hematoma from rupture of superficial cortical veins√ usually unilateral subdural convexity hematoma accompanied by subarachnoid blood√ underlying cerebral contusion√ sonographic visualization of convexities difficult

Notes:

Subacute Subdural Hematoma Time frame:4-20 daysCT: √ isodense hematoma (1-3 weeks) may be recognizable by mass effect with effacement of cortical sulci, deviation of lateral ventricle, midline shift, white matter buckling, displacement of gray-white matter junction√ contrast enhancement of inner membrane*AID in Dx:*contrast enhancement defines cortical-subdural interfaceMR: √ modality of choice in subacute stage because of high sensitivity for Met-Hb on T1WI (esp. superior to CT during isodense phase, for small subdural hematoma, for hematomas oriented in the CT scan plane, eg, tentorial subdural hematoma)

Notes:

Chronic Subdural Hematoma = result of(a)resolving phase of medically managed acute subdural hematoma(b)repeated episodes of subclinical hemorrhage until becoming symptomaticTime frame:>20 days old = 3 weeks and older*Histo:*hematoma enclosed by thick + vascular membrane which forms after 3-6 weeks*Pathogenesis:*vessel fragility accounts for repeated episodes of rebleeding following minor injuries that tear fragile capillary bed within neomembrane surrounding subdural hematoma*Predisposing factors:* alcoholism, increased age, epilepsy, coagulopathy, prior placement of ventricular shunt ◊>75% occur in patients >50 years of age!

● history of antecedent trauma often absent (25-48%) ● ill-defined neurologic signs + symptoms: cognitive deficit, behavioral abnormality, nonspecific headache√ crescent-shaped configuration (early) conforming to contour of brain√ often biconvex lenticular = medially concave configuration (late), esp. after compartmentalization secondary to formation of fibrous septa√ different attenuations within compartments√ low-density lesion of intermediate attenuation between CSF + brain, sometimes as low as CSF√ high-density components of collection (after common rebleeding)√ fluid-sedimentation levels (sedimented fresh blood with proteinaceous fluid layered above)√ displacement / absence of sulci, displacement of ventricles + parenchyma√ No midline shift if bilateral (25%)√ CECT demonstrates medially displaced cortical vein or membrane around hematoma (1-4 weeks after injury)*DDx:*Acute epidural hematoma (similar biconvex shape)

Notes:

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SUBDURAL HYGROMA
=CSF-fluid collection within subdural space; common in children*Cause:*traumatic tear in arachnoid with secondary ball valve mechanism*Time of onset:*6-30 days following trauma√ radiolucent crescent-shaped collection (as in acute subdural hematoma)√ no evidence of blood products (DDx to subdural hematoma)MR: √ isointense to CSF / hyperintense to CSF on T1WI (increased protein content)*Prognosis:*often spontaneous resorption*DDx:*(1)Enlarged subarachnoid space(2)Subdural empyema(3)Subdural hematoma

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TERATOMA OF CNS
*Incidence:*0.5% of primary intracranial neoplasms; 2% of intracranial tumors before age 15*Histo:*mostly benign, occasionally containing primitive elements + highly malignantLocation:pineal + parapineal region > floor of 3rd ventricle > posterior fossa > spine (associated with spina bifida) √ heterogeneous midline lesion, occasionally homogeneous soft-tissue mass (DDx: astrocytoma)√ contains fat + calcium√ hydrocephalus (common)

Notes:

TOXOPLASMOSIS OF BRAIN

*Organism:*obligate intracellular protozoan parasite Toxoplasma gondii, can live in any cell except for nonnucleated RBCs; felines are definite host*Infection:*ingestion of undercooked meat containing cysts or sporulated oocysts / transplacental transmission of trophozoites; acquired through blood transfusion + organ transplantation*Prevalence of seropositivity:* 11-16% of urban adults in United States; up to 90% of European adults *Histo:*inflammatory solid / cystic granulomas as a result of glial mesenchymal reaction surrounded by edema + microinfarcts due to vasculitis*Affected tissue:* @Gray + white matter of brain◊Most common cause of focal CNS infection in patients with AIDS!@Retina: most common retinal infection in AIDS@Alveolar lining cells (4%):mimics Pneumocystis carinii pneumonia @Heart (rare):cardiac tamponade / biventricular failure @Skeletal muscle ● asymptomatic ● lymphadenopathy ● malaise, fever
A.AIDS INFECTION = toxoplasmic encephalitis=reactivation of a chronic latent infection in >95%*Path:*well-localized indolent granulomatous process / diffuse necrotizing encephalitis ● focal neurologic deficit of subacute onset (50-89%) ● seizures (15-25%) ● pseudotumor cerebri syndromeLocation:basal ganglia (75%), scattered throughout brain parenchyma at gray-white matter junction√ multiple / solitary (up to 39%) lesions with nodular / thin-walled (common) ring enhancement√ surrounding white matter edema√ double-dose delayed CT scans with higher detection rate for multiple lesions (64-72%)√ ± hemorrhage and calcifications after therapy*Dx:*improvement on therapy with pyrimethamine + sulfadiazine within 1-2 weeks / biopsy*DDx:*CNS lymphoma (particularly with single lesion)◊Multiple lesions suggest toxoplasmosis!B.INTRAUTERINE INFECTIONTime of fetal infection:chances of transplacental transmission greater in late pregnancyScreening:impractical due to high false-positive rate ● Toxoplasma gondii found in ventricular fluid ● chorioretinitis ● mental retardation√ multiple irregular, nodular / cystlike / curvilinear calcifications in periventricular area + choroid plexus (= necrotic foci); bilateral; 1-20 mm in size; increasing in number + size (usually not developed at time of birth)√ hydrocephalus with return to normal / persistence of large head size√ thickened vault, sutures apposed / overlappingOB-US (as early as 20 weeks MA): √ sonographic findings in only 36%√ evolving symmetric ventriculomegaly√ intracranial periventricular + hepatic densities√ increased thickness of placenta√ ascites◊Microcephaly is NOT a feature of toxoplasmosis!Dx:elevated toxospecific IgM levels in fetal blood*Dx:*demonstration of elongated teardrop-shaped trophozoites in histologic sections of tissue

Notes:

TUBERCULOMA OF BRAIN
=result of granuloma formation within cerebral substance*Incidence:*0.15% of intracranial masses in Western countries, 30% in underdeveloped countries*Age:*infant, small child, young adult*Associated with:* tuberculous meningitis in 50% ● history of previous extracranial TB (in 60%)Location:more common in posterior fossa (62%), cerebellar hemispheres; may be associated with tuberculous meningitis√ solitary (70%) / multiple (30-60%) lesions; may be multiloculatedNCCT: √ isodense (72%) / hyperdense lesion of 0.5-4 cm in diameter with mass effect (93%)√ surrounding edema (72%) less marked than in pyogenic abscess√ central calcification (29%)CECT: √ homogeneous enhancement√ ring blush (nearly all) with smooth / slightly shaggy margins + thick wall around an isodense center (DDx: in pyogenic abscess less thick + more regular)√ "target sign" = central calcification in isodense lesion + ring-blush (DDx: giant aneurysm)√ homogeneous blush in tuberculoma en plaque along dural plane (6%) (DDx: meningioma en plaque)MR: √ isointense lesion on T1WI√ hypointense lesion ± hyperintense core on T2WI*DDx:*other CNS infection (esp. toxoplasmosis), lymphoma, atypical meningioma, radiation necrosis

Notes:

TUBEROUS SCLEROSIS
=BOURNEVILLE DISEASE = EPIPLOIA=neuroectodermal disorder characterized by TRIAD consisting of(1) Adenoma sebaceum (30%) (2) Seizures (80%) (3) Mental retardation (70%) *mnemonic:*zits, fits, nitwits*Frequency:*1:150,000 livebirths*Cause:*autosomal dominant with low penetrance (frequent skips in generations); gene loci 9q34 and 16p13; spontaneous mutations in 50-80%*Prognosis:*30% dead by age 5; 75% dead by age 20
@CNS INVOLVEMENT ● myoclonic seizures (80-90%): often first + most common sign of tuberous sclerosis with onset at 1st-2nd year, decreasing in frequency with age ● mental retardation (50-82%): mild to moderate (1/3) moderate to severe (2/3); progressive; observed in adulthood; common if onset of seizures before age 5 years 1.**Subependymal hamartomas**Location:along ventricular surface of caudate nucleus, on lamina of sulcus thalamo-striatus immediately posterior to foramen of Monro (most often), along frontal + temporal horns or 3rd + 4th ventricle (less commonly)√ multiple subependymal nodules with "candle drippings" appearance at lining of lateral ventricles√ calcification with increasing age (in up to 88%)M: √ subependymal nodules protruding into adjacent ventricle isointense with white matter√ minimal / no contrast enhancement2.**Giant cell astrocytoma**=large subependymal nodule located near foramen of Monro with tendency for enlargement + growth into ventricle*Incidence:*5-15%; M:F = 1:1√ hydrocephalus (obstruction at foramen of Monro)√ hypo- / isodense well-demarcated rounded lesion in the region of foramen of Monro√ hypo- / isointense on T1WI + hyperintense on T2WI√ uniformly enhancing mass√ frequent extension into frontal horn / body of lateral ventricle*Cx:*degeneration into higher grade astrocytoma
3.**Tubers** (in 56%)=CORTICAL / SUBCORTICAL HAMARTOMAS*Histo:*clusters of atypical glial cells surrounded by giant cells with frequent calcifications (if >2 years of age) = hamartomas*Frequency:*multiple (75%); bilateral (30%)√ noncalcified hypodense brain lesions of abnormal myelination within broadened cortical gyri√ cortical tubers calcified (in 15% <1 year of age, in 50% by age 10)MR: √ relaxation time similar to white matter (if uncalcified)√ multiple nodules of high-signal intensity on T2WI, iso- / hypointense on T1WI (fibrillary gliosis / demyelination)
4.**Heterotopic gray matter islands in white matter***Histo:*grouping of bizarre and gigantic neuronal cells associated with gliosis + areas of demyelination CT: √ hypodense well-defined regions within cerebral white matter without contrast enhancement√ calcification of all / part of noduleMR: √ subtle hypointense region on T1WI + well-defined hyperintense area on T2WI
DDx of CNS lesions: (1)Intrauterine CMV / Toxoplasma infection (smaller lesions, brain atrophy, microcephaly)(2)Basal ganglia calcification in hypoparathyroidism / Fahr disease (location)(3)Sturge-Weber, calcified AVM (diffuse atrophy, not focal)(4)Heterotopic gray matter (along medial ventricular wall, isodense, associated with agenesis of corpus callosum, Chiari malformation)
@SKIN INVOLVEMENT ● Adenoma sebaceum (80-90%) = wartlike nodules of brownish red color averaging 4 mm in size with bimalar distribution ("butterfly rash")*Age:*first discovered at age 1-5 years; family history in 30%*Path:*small hamartomas from neural elements with blood vessel hyperplasia = angiofibromasLocation:nasolabial folds, eventually covers nose + middle of cheeks ● Shagreen rough skin patches (80%) = "pigskin"= "peau dorange" = patches of fibrous hyperplasia; in intertriginous + lumbar location ● Ash leaf patches = hypopigmented macules shaped like ash / spearmint leaf on trunk + extremities (earliest manifestation in infancy); may be visible only under ultraviolet light ● Ungual fibromas (15-50%): sub- / periungual with erosion of distal tuft ● Café-au-lait spots: incidence similar to that in general population
@OCULAR INVOLVEMENT ● Phakoma (>50%) = whitish disk-shaped retinal hamartoma = astrocytic proliferation in / near optic disc, often multiple + usually in both eyes√ small calcifications in region of optic nerve head√ optic nerve glioma

@RENAL INVOLVEMENT ● renal failure in severe cases (5%); hypertension1.Angiomyolipoma (38%): usually multiple + bilateral; risk of spontaneous hemorrhage (subcapsular / perinephric)2.Multiple cysts of varying size in cortex + medulla mimicking adult polycystic kidney disease (15%)*Path:*cysts lined by columnar epithelium with foci of hyperplasia projecting into cyst lumen3.Renal cell carcinoma (3%), bilateral in 40%
@LUNG INVOLVEMENT (1%)√ interstitial fibrosis in lower lung fields + miliary nodular pattern may progress to honeycomb lung (lymphangioleiomyomatosis = smooth muscle proliferation around blood vessels)√ cystic changes of lung parenchyma√ spontaneous pneumothorax (50%)√ chylothorax√ cor pulmonale
@HEART INVOLVEMENT ● congenital cardiomyopathy√ circumscribed / diffuse subendocardial rhabdomyoma (in 5%)√ aortic aneurysm

@BONE INVOLVEMENT√ sclerotic calvarial patches (45%) = "bone islands" involving diploe + internal table; frontal + parietal location√ thickening of diploe (long-term phenytoin therapy)√ bone islands in pelvic brim, vertebrae, long bones√ periosteal thickening of long bones√ bone cysts with undulating periosteal reaction in distal phalanges (most common), metacarpals, metatarsals (DDx: sarcoid, neurofibromatosis)
@OTHER VISCERAL INVOLVEMENT1.Adenomas + lipomyomas of liver2.Adenomas of pancreas3.Tumors of spleen
@VASCULAR INVOLVEMENT (rare)√ thoracic + abdominal arterial aneurysms*Path:*vascular dysplasia with intimal + medial abnormalities of large muscular + musculoelastic arteries

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UNILATERAL MEGALENCEPHALY
=hamartomatous overgrowth of all / part of a cerebral hemisphere with neuronal migration defects ● intractable seizure disorder at early age, hemiplegia ● developmental delay√ moderately / marked enlargement of hemisphere√ ipsilateral ventriculomegaly proportionate to enlargement of affected hemispherestraightened frontal horn of ipsilateral ventricle pointing anterolaterally√ neuronal migration defects√ polymicrogyria√ pachygyria√ heterotopia of gray matter√ white matter gliosis (low density in white matter on CT, prolonged T1 + T2 relaxation times on MR)*Rx:*partial / complete hemispheric resection

Notes:

VEIN OF GALEN ANEURYSM

=central AVM directly draining into secondarily enlarged vein of Galen (aneurysm is a misnomer)*Anatomical types:* type 1=AV fistula fed by enlarged arterial branches leading to dilatation of vein Galen + straight sinus + torcular herophilitype 2=angiomatous malformation involving basal ganglia + thalami ± midbrain draining into vein of Galentype 3=transitional AVM with both featuresFeeding vessels: (a)posterior cerebral artery, posterior choroidal artery (90%)(b)anterior cerebral artery + anterior choroidal artery(c)middle cerebral artery + lenticulostriate + thalamic perforating arteries (least common)*Age at presentation:*detectable in utero >30 weeks GA; M:F = 2:1(a)neonatal pattern (0-1 month) ● high-output cardiac failure (36%) due to massive shunting(b)infant pattern (1-12 months) ● macrocrania from obstructive hydrocephalus ● seizures(c)adult pattern (>1 year) ● headaches ± intracranial hemorrhage ● ± hydrocephalus ● focal neurologic deficits (5%) due to steal of blood from surrounding structures ● cranial bruit*May be associated with:* porencephaly, nonimmune hydrops√ smoothly marginated midline mass posterior to indented 3rd ventricle√ prominent serpiginous network in basal ganglia, thalami, midbrain√ dilated straight + transverse sinus + torcular herophil√ dilatation of lateral + 3rd ventricle (37%)NCCT: √ round well-circumscribed homogeneous slightly hyperdense mass in region of 3rd ventricular outlet√ hyperdense intracerebral hematoma (ruptured AVM)√ focal hypodense zones (ischemic changes)√ rim calcification (14%)CECT: √ marked homogeneous enhancement of serpentine structures + vein of Galen + straight sinusOB-US: √ median tubular cystic space with high-velocity turbulent flow demonstrated by pulsed / color Doppler√ brain infarction / leukomalacia (steal phenomenon with hypoperfusion)√ cardiac enlargement (high-output heart failure)√ dilated veins of head + neck√ hydrocephalus (aqueductal obstruction / posthemorrhagic impairment of CSF absorption)MR: √ areas of signal voidAngio: necessary to define vascular anatomy for surgical / endovascular intervention *Cx:*subarachnoid hemorrhage*Rx:*ligation, excision, embolization of vessels from transtorcular / transarterial approach*Prognosis:*56% overall mortality; 91% neonatal mortality*DDx:* pineal tumor, arachnoid / colloid / porencephalic cyst

Notes:

VENOUS ANGIOMA
=cluster of dilated medullary veins, which drain into an enlarged vein; bleed rarely◊Can be considered a normal variant!*Histo:*venous channels without internal elastic lamina, separated by gliotic neural tissue that may calcify; probably representing persistent fetal venous system√ no arterial vessels√ "umbrella" configuration = multiple small radially oriented veins at periphery of lesion converging to a single larger vein◊Associated with increased incidence of cavernous angiomas which can bleed!*DDx:*Sturge-Weber disease (diffuse pial angiomatosis with venous-type capillaries)

Notes:

VENOUS SINUS THROMBOSIS
Septic causes (esp. in childhood): mastoiditis, sub- / epidural empyema, meningitis, encephalitis, brain abscess, face + scalp cellulitis, septicemia *Aseptic causes:* (a)Tumor compressing sinuses: meningioma, leukemia(b)Trauma: fracture through sinus wall, cranial surgery(c)Low-flow state: CHF, CHD, dehydration, shock(d)Hypercoagulability: polycythemia vera, idiopathic thrombocytosis, thrombocytopenia, sickle cell disease, cryofibrinogenemia, pregnancy, contraceptive steroids, disseminated intravascular coagulopathy(e)Chemotherapy: eg, ARA-C ● headaches, drowsiness, fever, nausea, vomiting ● stroke symptomatology, seizuresNCCT: √ high-attenuation material (clotted blood) in sagittal sinus / straight sinus / cerebral cortical vein = "cord sign" (rare)√ compression of lateral ventricles in 32% (infarction / edema)√ unilateral (2/3) / bilateral (1/3) parenchymal hemorrhage involving gray + white matter (20%)CECT: √ "delta sign" / "empty triangle" = filling defect in straight sinus / superior sagittal sinus (in 70%)√ gyral enhancement in periphery of infarction (30-40%)√ intense tentorial enhancement secondary to collaterals (rare)√ dense transcortical medullary veinAngio: √ nonfilling of thrombosed sinus√ filling of cortical veins, deep venous system, cavernous sinus√ parasagittal hemorrhages (highly specific for superior sagittal sinus thrombosis) secondary to cortical venous infarctionMR: √ high signal within sinus on T1WI + T2WI*Prognosis:*high mortality

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VENTRICULITIS
=EPENDYMITIS = inflammation of ependymal lining of one / more ventricles*Cause:*(1)rupture of periventricular abscess (thinner capsule wall medially)(2)retrograde spread of infection from basal cisternsCECT (necessary for diagnosis): √ thin uniform enhancement of involved ependymal lining√ often associated with intraventricular inflammatory exudate + septations*Cx:*obstructive hydrocephalus (occlusion at foramen of Monro / aqueduct)*DDx:*ependymal metastases, lymphoma, infiltrating glioma

Notes:

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VENTRICULOPERITONEAL SHUNT

A.SHUNT MALFUNCTION*Cause:*occlusion of catheter by choroid plexus / glial tissue, disconnection of tubes ● symptoms of increased intracranial pressure ● persistent bulging of anterior fontanelle ● excessive rate of head growth√ increasing ventricular size√ shuntogram (by scintigram / contrast radiography) determines site of obstruction√ brain edema tracking along shunt + within interstices of centrum semiovale (with partial obstruction)√ formation of white matter cyst surrounding ventricular catheter

B.SHUNT INFECTION*Incidence:*1-5% ● intermittent low-grade fever ● anemia, dehydration, hepatosplenomegaly ● stiff neck ● swelling + redness over shunting tract ● peritonitis√ ventriculitis (= enlarged ventricles with irregular enhancing ventricular wall ± septations)

C.ABDOMINAL COMPLICATIONS1.Ascites2.Pseudocyst formation3.Perforation of viscus / abdominal wall4.Intestinal obstruction

D.SUBDURAL HEMATOMA*Cause:*precipitous drainage of markedly enlarged ventricles*Age:*usually seen in children >3 years of age*Prognosis:*small hematomas are insignificant

E.GRANULOMATOUS LESION=rare granulomatous reaction adjacent to shunt tube within / near ventricle√ irregular contrast-enhancing mass along course of shunt tube

F.SLIT VENTRICLE SYNDROME=symptoms from shunt failure in absence of ventricular enlargement (poorly defined syndrome)√ normal imaging studies

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VISCERAL LARVA MIGRANS OF BRAIN

roundworm nematode (Toxocara canis) → small calcific nodules, especially in basal ganglia + periventricular*DDx:* tuberous sclerosis

Notes:

VON HIPPEL-LINDAU DISEASE
= vHL = RETINOCEREBELLAR ANGIOMATOSIS =inherited neurocutaneous dysplasia complex; autosomal dominant (gene located on chromosome 3p25-p26) with 80-100% penetrance + variable delayed expressivity; grouped under hereditary phakomatosis; in 20% familial*Age at onset:*2nd-3rd decade; M:F = 1:1
Diagnostic criteria: (a)>1 hemangioblastoma of CNS(b)1 hemangioblastoma + visceral manifestation(c)1 manifestation + known family history
@CNS MANIFESTATION*Age at presentation:*25-35 years ● cerebellar symptoms: vertigo, dysdiadochokinesia, dysmetria, Romberg sign ● signs of increased intracranial pressure: headache, vomiting ● vision changes: reactive retinal inflammation with exudate + hemorrhage, retinal detachment, glaucoma, cataract, uveitis, decreasing visual acuity, eye pain ● spinal cord symptoms (uncommon): loss of sensation, impaired proprioception
1.Retinal angiomatosis = **von Hippel tumor** (>45%) earliest manifestation of disease; multiple in up to 66%, bilateral in up to 50%*Dx:*indirect ophthalmoscopy + fluorescein angiography√ small tumors rarely detected by imaging studies√ globe distortion√ thick calcified retinal density (calcified angioma-induced hematoma)US: √ small hyperechoic solid masses, most in temporal retina*Cx:*(1)repeated vitreous hemorrhage (frequent)(2)exudative retinal detachment posteriorly
2.Hemangioblastomas of CNS = **Lindau tumor** (40%)=most commonly recognized manifestation of vHL disease*Age:*15-40 yearsSite:cerebellum (65%), brainstem (20%), spinal cord (15%); multiple lesions in 10-15%√4-20% of single hemangioblastomas occur in von Hippel-Lindau disease!CT: √ large cystic lesion with 3-15 mm mural nodule (75%)√ solid enhancing lesion (10%)√ enhancing lesion with multiple cystic areas (15%)√ intense tumor blush / blushing mural nodule√ NO calcifications (DDx: cystic astrocytoma calcifies in 25%)MR (modality of choice): √ hypointense cystic component on T1WI (slightly hyperintense to CSF due to protein content); hyperintense on T2WI√ small tubular areas of flow void within mural nodule (= enlarged feeding + draining vessels); intense contrast enhancement of mural nodule√ slightly hypointense solid lesion on T1WI; hyperintense on T2WI; intense contrast enhancementAngio: √ intense staining of mural nodule ("mother-in-law phenomenon" = tumor blush comes early, stays late, very dense)√ presence of feeding vessels*Prognosis:*most frequent cause of morbidity and mortality; frequent recurrence after resection@LABYRINTH1.Endolymphatic sac neoplasm=aggressive adenomatous tumor with mixed histologic features ● sensorineural hearing lossLocation:retrolabyrinthine temporal boneSite:endolymphatic sac√ aggressive lytic lesion containing intratumoral osseous spicules + areas of hemorrhage√ heterogeneous enhancement with hyperintense areas on T1WI + T2WI (due to hemorrhage)@HEART1.Rhabdomyoma
@KIDNEYS ● polycythemia due to elevated erythropoietin level (in 15% with hemangioblastoma, in 10% with renal cell carcinoma)1.Cortical renal cysts (75%)multiple + bilateral (may be confused with adult polycystic kidney disease) 2.Renal cell carcinoma (20-45%)*Age:*20-50 years√ multicentric in 87%, bilateral in 10-75%, may arise from cyst wall√ sensitivity: 35% for angiography, 37% for US, 45% for CT (due to inability to reliably distinguish between cystic RCC, cancer within cyst, atypical cyst) √ 50% metastatic at time of discovery*Prognosis:*RCC is cause of death in 30-50% as the second most frequent cause of mortality!3.Renal adenoma4.Renal hemangioma
@ADRENAL pheochromocytoma (in up to 10-17%), bilateral in up to 40%; confined to certain families
@EPIDIDYMIS1.Cystadenoma of epididymis
@PANCREAS1.Pancreatic cystadenoma / cystadenocarcinoma2.Pancreatic islet cell tumor3.Pancreatic hemangioblastoma4.Pancreatic cysts (in 30%); incidence in autopsies up to 72%√ usually multiple and multilocular cysts
@LIVER1.Liver hemangioma2.Adenoma@OTHERS1.Paraganglioma2.Cysts in virtually any organ: liver, spleen, adrenal, epididymis, omentum, mesentery, lung, bone
MULTIPLE ORGAN NEOPLASMS @Kidney:renal cell carcinoma (up to 40%), renal angioma (up to 45%)
@Liver:adenoma, angioma
@Pancreas:cystadenoma / adenocarcinoma@Epididymis:adenoma@Adrenal gland:pheochromocytoma

MULTIPLE ORGAN CYSTS (1)Kidney (usually multiple cortical cysts in 75-100% at early age, most common abdominal manifestation)
(2)Pancreas (in 9-72% often numerous cysts; second most common affected abdominal organ)(3)Others: liver, spleen, omentum, mesentery, epididymis, adrenals, lung, bone

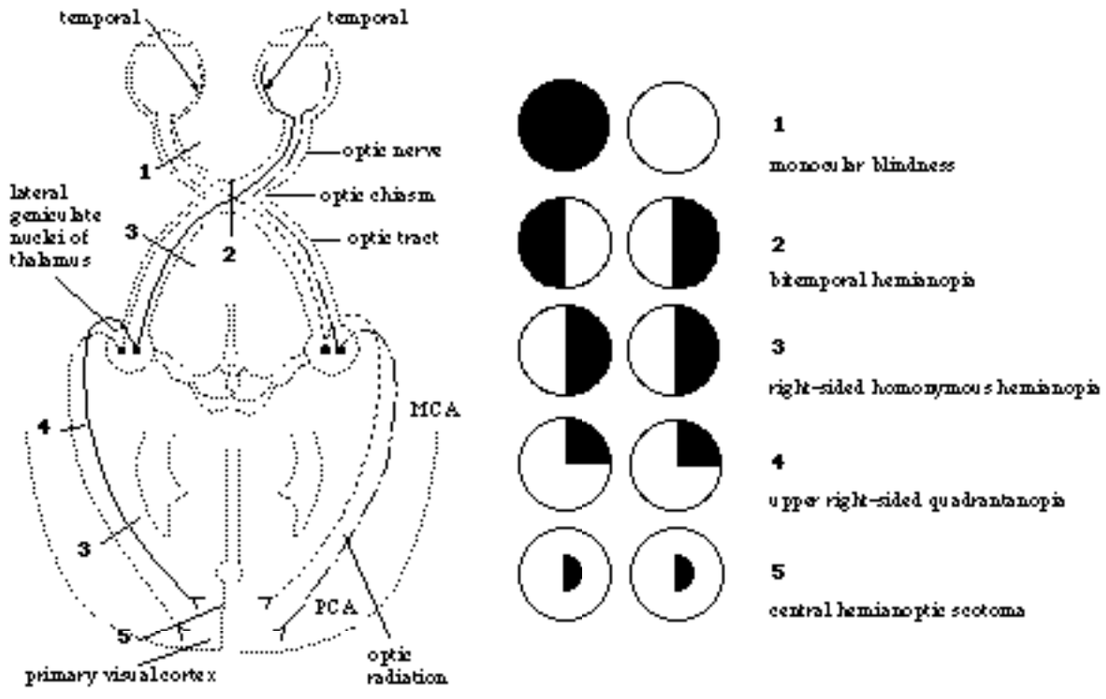
Notes:

OPHTHALMOPLEGIA
Lesions of 1.Oculomotor nerve (III)innervates medial rectus, superior rectus, inferior rectus, inferior oblique muscle, pupilloconstrictor, levator palpebrae 2.Trochlear nerve (IV)innervates superior oblique muscle 3.Abducens nerve (VI)innervates lateral rectus muscle

Notes:

ANOPIA

[numbers are referring to drawing] A.MONOCULAR DEFECTS1=monocular blindness (optic nerve lesion in fracture of optic canal, amaurosis fugax)B.BILATERAL HETERONYMOUS DEFECTS2=bitemporal hemianopia (chiasmatic lesion)C.BILATERAL HOMONYMOUS DEFECTS3=homonymous hemianopia4=upper right-sided quadrantanopia5=central hemianoptic scotoma3,4,5=most common type of hemianopia (CVA, brain tumor)



Notes:

OCULAR TRAUMA
Types:(a)Simple / complicated contusion with rupture of ocular wall(b)Simple / perforating injury to the globe(c)Foreign bodyEvaluate for: (1)vitreous hemorrhage (2)retinal detachment (3)choroidal detachment (4)alteration in position / texture of lens (5)thickening / rupture of ocular wall (6)Hematoma in retro-ocular space (7)Vascular complications: central renal artery occlusion, carotid-cavernous fistula, fistula of angular vein (8)Foreign body in globe (95% sensitivity for US) / orbit (50% sensitivity for US)

Notes:

Spectrum Of Orbital Disorders A.INFLAMMATORY DISEASE1.Tissue-specific inflammation:orbital cellulitis, optic neuritis, scleritis, myositis 2.Panophthalmitis3.Pseudotumor of orbitB.CYSTIC DISEASE1.Dermoid cyst2.Mucocele3.Retro-ocular cyst (developmental)C.VASCULAR DISEASE1.Cavernous angioma2.Capillary angioma3.Lymphangioma4.Varix5.Carotid-cavernous fistulaD.TUMORS1.Rhabdomyosarcoma2.Optic nerve glioma3.Meningioma4.Lymphoma5.Metastasis

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Notes:

Intraconal Lesion *mnemonic:* "**M**el **M**et **R**ita **M**ending **H**ems **O**n **P**oor **C**harlies **G**rave"**M**elanoma **M**etastasis **R**etinoblastoma **M**eningioma **H**emangioma **O**ptic glioma **P**seudotumor **C**ellulitis **G**rave disease
Intraconal Lesion With Optic Nerve Involvement 1.Optic nerve glioma2.Optic nerve sheath meningioma (10% of orbital neoplasm)3.Optic neuritis4.Inflammatory pseudotumor (may surround optic nerve)5.Intraorbital lymphoma (may surround optic nerve, older patient)6.Elevated intracranial pressure=distension of optic sheath√ bilateral tortuous enlarged optic nerve-sheath complex
Intraconal Lesion Without Optic Nerve Involvement 1.Cavernous hemangioma2.Orbital varix3.Carotid-cavernous fistula4.Arteriovenous malformationleast common of orbital vascular malformations (congenital, idiopathic, traumatic)√ irregularly shaped intensely enhancing mass of enlarged vessels√ associated with dilated superior / inferior ophthalmic vein5.Hematoma6.Lymphangioma7.Neurilemoma√ commonly adjacent to superior orbital fissure, inferior to optic nerve√ local bone erosion

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Notes:

Extraconal Lesion ***Extraconal-intraorbital Lesion*** A.BENIGN TUMOR1. Dermoid cyst2.**Teratoma**
<1% of all pediatric orbital tumors √ ± areas of fat, cartilage, bone√ expansion of bony orbit ± bone defect3. Capillary hemangioma4.Lymphangioma5.Plexiform neurofibroma6. Inflammatory orbital pseudotumor7.Histiocytosis Xlesion usually arises from bone B.MALIGNANT TUMOR1.Lymphoma / Leukemia2. Metastasis3.Rhabdomyosarcoma ***Extraconal-extraorbital Lesion*** A.FROM SINUSmaxillary / sphenoid sinuses are rare locations of origin 1.Tumor:squamous cell carcinoma (80%), adenocarcinoma, adenoid cystic carcinoma, lymphoma2.Paranasal sinusitis:most common cause of orbital infection; *Origin:*from ethmoid sinuses (in children), from frontal sinus (in adolescence)*Organism:*Staphylococcus, Streptococcus, Pneumococcus√ preseptal / orbital edema / cellulitis√ subperiosteal / orbital abscess√ mucormycosis (in diabetics) destroys bone + extends into cavernous sinus*Cx:*(1) epidural abscess (2) subdural empyema (3) cavernous sinus thrombosis (4) meningitis (5) cerebritis (6) brain abscess3.MucoceleB.FROM SKIN1.Orbital cellulitisC.FROM LACRIMAL GLAND√ mass arising from superolateral aspect of orbit *mnemonic:*"MOLD"**M**etastasis **O**thers (rhabdomyosarcoma, lymphangioma, sinus lesion) **L**ymphoma, **L**acrimal gland tumor **D**ermoid

Notes:

Orbital Mass In Childhood 1.Dermoid cyst46%2.Inflammatory lesion16%3.Dermolipoma7%4.Capillary hemangioma4%5.Rhabdomyosarcoma4%6.Leukemia / lymphoma2%7.Optic nerve glioma2%8.Lymphangioma2%9.Cavernous hemangioma1% *mnemonic:*"LO VISHON"**L**eukemia, **L**ymphoma **O**ptic nerve glioma **V**ascular malformation: hemangioma, lymphangioma **I**nflammation **S**arcoma: ie, rhabdomyosarcoma **H**istiocytosis Orbital pseudotumor, **O**steoma **N**euroblastoma ***Primary Malignant Orbital Tumors*** 1.Retinoblastoma86.0%2.Rhabdomyosarcoma8.1%3.Uveal melanoma2.3%4.Sarcoma1.7% ***Secondary Malignant Orbital Tumors*** 1.Leukemia36.7%2.Sarcoma14.3%3.Hodgkin lymphoma11.0%4.Neuroblastoma9.2%5.Wilms tumor6.7%6.Non-Hodgkin lymphoma5.6%7.Histiocytosis3.9%8.Medulloblastoma3.5% ***Orbital Cystic Lesion*** 1.Abscess2.Intraorbital hematoma3.Dermoid cyst4.Lacrimal cyst5.Lymphangioma6.Hydatid cyst ***Orbital Vascular Tumors*** 1.Orbital varix2.Arteriovenous malformation3.Carotid-cavernous fistula4.Hemangioma: capillary / cavernous5.Blood cyst6.Arterial malformation7.Glomus tumor8.Hemangiopericytoma

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Notes:

Mass In Superolateral Quadrant Of Orbit 1.Lacrimal gland tumor2.Dermoid cyst3.Metastasis (breast, prostate, lung)4.Lymphoma5.Leukemic infiltration of lacrimal gland6.Sarcoidosis7.Wegener granulomatosis8.Pseudotumor9.Frontal sinus mucocele

Notes:

Extraocular Muscle Enlargement A.ENDOCRINE1.Grave disease (50%)2.Acromegaly B.INFLAMMATION1.**Myositis**

- rapid onset of proptosis, erythema of lids, conjunctival injection Location:single muscle (in adults); multiple muscles (in children)√ enlarged extraocular muscle√ positive response to steroids2.Orbital cellulitis3.Sjögren disease, Wegener granulomatosis, lethal midline granuloma, SLE4.Sarcoidosis5.Foreign-body reaction C.TUMOR1.Pseudotumor2.Rhabdomyosarcoma3.Metastasis, lymphoma, leukemia D.VASCULAR1.Spontaneous / traumatic hematoma2.Arteriovenous malformation3.Carotid-cavernous sinus fistula

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Notes:

Spectrum Of Ocular Disorders A.CONGENITAL1.Persistent hyperplastic primary vitreous2.Coats disease3.Coloboma4.Congenital cataractB.VITREORETINAL1.Vitreous hemorrhage2.Retinal detachment3.Choroidal detachment4.Endophthalmitis5.Retinoschisis6.Retrolental fibroplasiaC.TUMOR1.Retinoblastoma2.Choroidal hemangioma3.Retinal angiomatosis4.Melanocytoma5.Choroidal osteomaD.TRAUMA

Notes:

Microphthalmia =congenital underdevelopment / acquired diminution of globeA.BILATERAL with cataract1.Congenital rubella2.Persistent hyperplastic vitreous3.Retinopathy of prematurity4.Retinal folds5.Lowe syndrome√ small globe + small orbitB.UNILATERAL1.Trauma / surgery / radiation therapy2.Inflammation with disorganization of eye (phthisis bulbi)√ shrunken calcified globe + normal orbit

Notes:

Macrophthalmia =enlargement of globeA.WITHOUT INTRAOCULAR MASS(a)generalized enlargement1.Axial myopia (most common cause)√ enlargement of globe in AP direction√ ± thinning of sclera2.Buphthalmos3.Juvenile glaucoma4.Connective tissue disorder: Marfan syndrome, Ehlers-Danlos syndrome, Weill-Marchesani syndrome (congenital mesodermal dysmorphodystrophy), homocystinuria√ "wavy" contour of sclera(b)focal enlargement1.**Staphyloma** =sacculation of posterior pole of globe (or berrylike protrusion of cornea)*Prevalence:*increasing with size of globe*Cause:*axial myopia (temporal side of optic disc / anteriorly / along equator), trauma, scleritis, necrotizing infection√ focal bulge + thinning of sclera*Cx:*advanced chorioretinal degeneration (77%), choroid retraction from optic disc, posterior vitreous detachment, choroidal hemorrhage, retinal detachment, cataract, glaucoma2.Apparent enlargement due to contralateral microphthalmiaB.WITH INTRAOCULAR MASS(rare cause for enlargement) (a)with calcifications:1.Retinoblastoma(b)without calcifications:1.Melanoma2.Metastasis

Notes:

Ocular Lesion *Intraocular Calcifications* 1.Retinoblastoma (>50% of all cases)2.Astrocytic hamartoma3.**Choroidal osteoma** = rare juxtapapillary tumor of mature bone *Age:*young woman; may be bilateral√ small flat very dense curvilinear mass aligned with choroidal margin of globe*DDx:*calcified choroidal angioma4. **Optic drusen** =accretions of hyaline material on / near surface of optic disc; often familial ● headache, visual field defects ● pseudopapilledema√ small flat / round calcification at junction of retina + optic nerve√ bilateral in 75%5.Scleral calcifications(a)in systemic hypercalcemic states (HPT, hypervitaminosis D, sarcoidosis, secondary to chronic renal disease)(b)in elderly: at insertion of extraocular muscles6.Retrolental fibroplasia7. **Phthisis bulbi** secondary to trauma or infection √ small contracted calcified disorganized nonfunctioning globe *mnemonic:*"NMR CT"**N**eurofibromatosis **M**elanoma (hyperdense melanin) **R**etinoblastoma **C**horoidal osteoma **T**uberous sclerosis ***Noncalcified Ocular Process*** 1.Uveal melanoma2.Metastasis86% of ocular lesions within globe; usually in vascular choroid *Origin:*breast, lung, GI tract, GU tract, cutaneous melanoma, neuroblastoma√ bilateral in 30%3. Choroidal hemangioma 4. **Vitreous lymphoma** √ diffuse ill-defined soft-tissue density5. Developmental anomalies (a)**Primary glaucoma** = enlargement of eye secondary to narrowing of Schlemm canal(b)Coloboma(c)Staphyloma

Notes:

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Vitreous Hemorrhage *Cause:*trauma, surgical intervention, arterial hypertension, retinal detachment, ocular tumor, Coats diseaseUS: √ numerous irregular, poorly defined, mobile low-intensity echoes√ voluminous hyperechoic fibrin clots not fixed to optic nerve (DDx to retinal detachment)*Prognosis:*complete absorption / development of vitreous membranes (repetitive episodes)

Notes:

Dense Vitreous In Pediatric Age Group 1.Retinoblastoma2.Persistent hyperplastic primary vitreous3.Coats disease4.Norrie disease5.Retrolental fibroplasia6.Sclerosing endophthalmitis

Notes:

Retinal Detachment *Cause:*trauma, tumor, exudative / inflammatory process, scarUS: √ curvilinear area of high echogenicity fixed at optic disk (= papilla) + extending to ora serrata√ V-shaped (with total detachment)√ in one quadrant only (partial detachment)√ thick folded retina with loss of mobility (long-standing detachment)√ subretinal space normal / occupied by blood, inflammation / tumor (depending on cause)*DDx:*vitreous membranes, choroidal detachment (point of fixation not at papilla)

Notes:

Choroidal Detachment *Cause:*trauma, surgical intervention, spontaneousUS: √ two convex lines emerging from both walls of the vitreous + advancing to ciliary body with posterior fixation outside the macula√ minimal / no choroidal membrane mobility

Notes:

Leukokoria =abnormal white / pinkish / yellowish pupillary light reflex [from Greek *leuko* = white and *koria* = pupil]A.TUMOR1.Retinoblastoma (most common cause - 58%)2.Retinal astrocytic hamartoma (3%):associated with tuberous sclerosis + von Recklinghausen disease 2.Medulloepithelioma (rare)B.DEVELOPMENTAL1.Persistent hyperplastic primary vitreous(2nd most common cause - 28%) 2.Coats disease (16%)3.Retrolental fibroplasia (3-5%)4.Coloboma of choroid / optic discC.INFECTION1.Uveitis2.Larval granulomatosis (16%)D.DEGENERATIVE1.Posterior cataractE.TRAUMA1.Retinopathy of prematurity (5%)2.Organized vitreous hemorrhage3.Long-standing retinal detachment ***Leukokoria In Normal-sized Eye*** A.CALCIFIED MASS1.Retinoblastoma2.Retinal astrocytomaB.NONCALCIFIED MASS1.Toxocaral endophthalmitis2.Coats disease ***Leukokoria With Microphthalmia*** A.UNILATERAL1.Persistent hyperplastic primary vitreous (PHPV)B.BILATERAL1.Retinopathy of prematurity2.Bilateral PHPV

Notes:

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Optic Nerve Enlargement A.TUMOR:1.Optic nerve glioma2.Optic nerve sheath meningioma3.Infiltration by leukemia / lymphomaB.FLUID:1.Perineural hematoma2.Papilledema of intracranial hypertension3.Patulous subarachnoid spaceC.INFLAMMATION:1.Optic neuritis2.Sarcoidosis √ fusiform thickening=lens-shaped thickening of nerve-sheath complex(a)with central lucency: meningioma(b)without central lucency: optic nerve glioma√ excrescentic thickening=single / multiple nodules along nerve-sheath complex usually due to tumor√ tubular enlargement=uniform enlargement of nerve-sheath complex(a)with central lucency: subarachnoid process (metastases, perineuritis, meningioma, perineural hemorrhage)(b)without central lucency: papilledema, leukemia, lymphoma, sarcoid, optic nerve glioma

Notes:

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Lacrimal Gland Lesion A.INFLAMMATION1.Dacryoadenitis2. **Mikulicz syndrome** =nonspecific enlargement of lacrimal + salivary glands*Associated with:*sarcoidosis, lymphoma, leukemia3.**Sjögren syndrome**
=lymphocytic infiltration of lacrimal + salivary glands ● decreased lacrimation, xerostomia*Often associated with:* rheumatoid arthritis, systemic lupus erythematosus, scleroderma, polymyositis 4.SarcoidosisB.TUMOR(a)benign:granuloma, cyst, benign mixed tumor (= pleomorphic adenoma)(b)malignant:malignant mixed tumor (= pleomorphic adenocarcinoma), adenoid cystic carcinoma, lymphoma, metastasis (rare)

Notes:

Lacrimal Gland Enlargement *mnemonic:*"MELD"**M**etastasis **E**pithelial tumor **L**ymphoid tumor **D**ermoid BILATERAL LACRIMAL GLAND MASSES *mnemonic:*"LACS"**L**ymphoma **A**nd **C**ollagen-vascular disease **S**arcoidosis

Notes:

Superior Orbital Fissure Boundaries (Grays Anatomy): - medial:sphenoid body- above:lesser wing of sphenoid = optic strut- below:greater wing of sphenoid- lateral:small segment of frontal bone*Contents:* (a)nerves:IIIoculomotor n.IVtrochlear n.V_1ophthalmic branch of trigeminal n.:(a) lacrimal nerve(b) frontal nerveVIabducens n.sympathetic filaments of internal carotid plexus (b)veins:superior + inferior ophthalmic vein(c)arteries:1.meningeal branch of lacrimal artery2.orbital branch of middle meningeal artery

Notes:

Inferior Orbital Fissure Location:between floor + lateral wall of orbit; connects with pterygopalatine + infratemporal fossa*Contents:* (a)nerves:infraorbital + zygomatic nn.branches from pterygopalatine ganglion(b)veins:connection between inferior orbital v. + pterygoid plexus

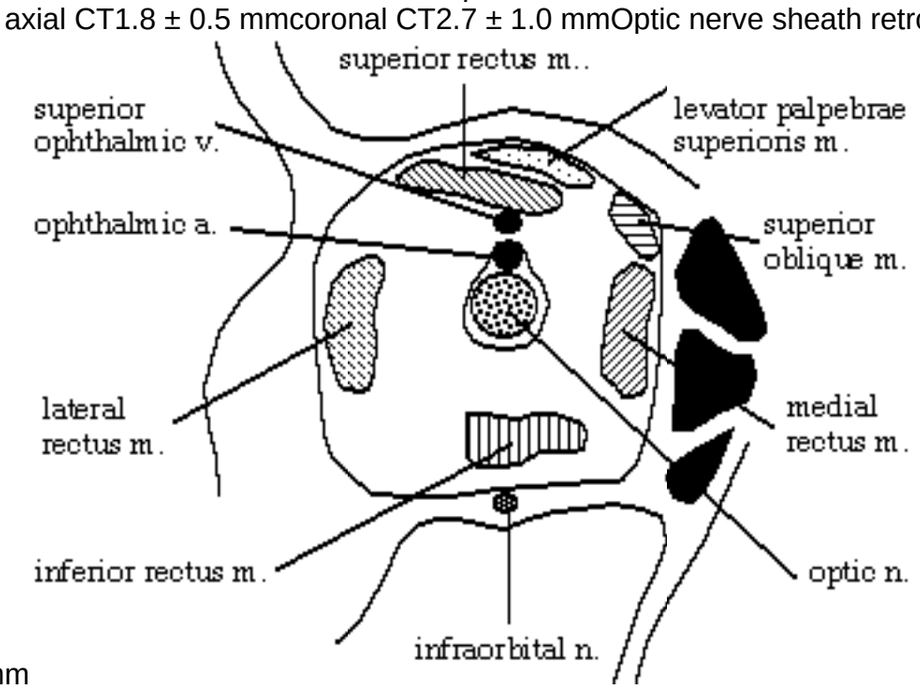
Notes:

Optic Canal completely formed by lesser wing of sphenoid *Contents:* (a)nerve:optic nerve (I)(b)vessel:ophthalmic a.

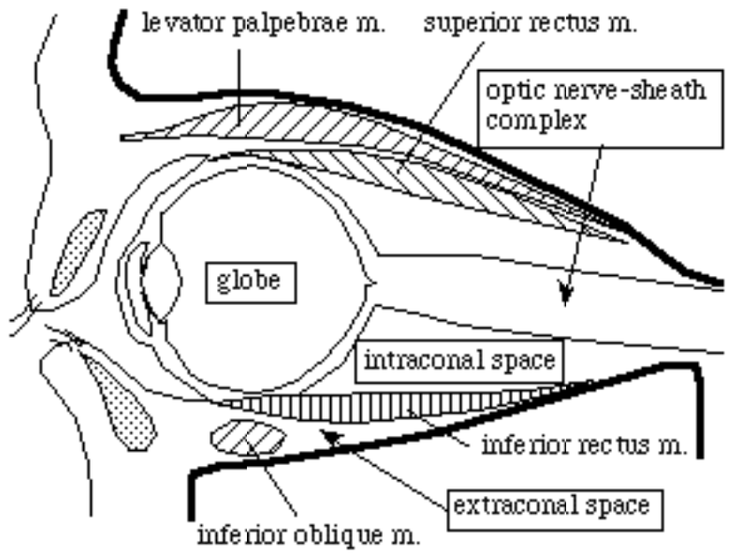
Notes:

NORMAL ORBIT MEASUREMENTS

Muscles medial rectus muscle4.1 ± 0.5 mminferior rectus muscle4.9 ± 0.8 mmsuperior rectus muscle3.8 ± 0.7 mmlateral rectus muscle2.9 ± 0.6 mmsuperior oblique muscle2.4 ± 0.4 mmSuperior ophthalmic vein axial CT1.8 ± 0.5 mmcoronal CT2.7 ± 1.0 mmOptic nerve sheath retrobulbar5.5 ± 0.8 mmwaist4.2 ± 0.6 mmGlobe position behind interzygomatic line9.9 ± 1.7 mm



Coronal Orbital Tomogram Through Midorbit



Orbital Spaces

globe:	subdivided into anterior + posterior segments by lens
optic nerve-sheath complex:	optic nerve surrounded by meningeal sheath as extension from cerebral meninges
intraconal space:	orbital fat, ophthalmic a., superior ophthalmic v., nerves I, III, IV, V_1, VI
conus:	incomplete fenestrated musculofascial system extending from bony orbit to anterior third of globe, consists of extraocular muscles + interconnecting fascia
extraconal space:	between muscle cone + bony orbit containing fat, lacrimal gland, lacrimal sac, portion of superior ophthalmic v.

Notes:

BUPTHALMOS
=HYDROPHTHALMOS = MEGOPHTHALMOS=diffuse enlargement of eye in children secondary to increased intraocular pressure*Cause:* 1.Congenital / infantile glaucoma2.Neurofibromatosis type 1: obstruction of canal of Schlemm by membranes / masses composed of aberrant mesodermal tissue3.Sturge-Weber syndrome4.Lowe (cerebrohepatorenal) syndrome5.Ocular mesodermal dysplasia (eg, Axenfeld or Rieger anomalies)6.Homocystinuria7.Aniridia8.Acquired glaucoma (rare)*Pathophysiology:* obstruction of canal of Schlemm located between cornea + iris leads to decreased resorption of aqueous humor (= anterior chamber fluid) with scleral distension → uniformly enlarged globe without mass of round / oval / bizarre shape*Rx:*goniotomy (increases the angle of anterior chamber); trabeculotomy (lyses adhesions)

Notes:

CAROTID-CAVERNOUS SINUS FISTULA
=abnormal communication between internal carotid artery + veins of cavernous sinus*Etiology:* (1)Trauma: laceration of ICA within cavernous sinus(a)usually secondary to basal skull fracture (cavernous ICA + small cavernous branches fixed to dura) (b)penetrating trauma(2)Spontaneous: rupture of an intracavernous ICA aneurysm*Route of drainage:* (a)superior ophthalmic vein (common)(b)contralateral cavernous sinus(c)petrosal sinus(d)cortical veins (rare) ● pulsating exophthalmos, chemosis, conjunctival edema ● persistent orbital bruit ● restricted extraocular movement ● decrease in vision due to increase in intraocular pressure (50%) = indication for emergent treatment√ enlarged edematous extraocular muscles√ dilatation of superior ophthalmic vein / facial veins / internal jugular vein√ focal / diffuse enlargement of cavernous sinus√ occasionally sellar erosion / enlargement√ enlargement of superior orbital fissure (in chronic phase)US + MR: √ arterial flow in cavernous sinus + superior ophthalmic veinAngio: √ ipsilateral ICA contrast injection shows wall of ICA to be incomplete√ contralateral ICA contrast injection + compression of involved ICA√ early opacification of veins of cavernous sinus√ retrograde flow through dilated superior ophthalmic v.*Rx:*latex / silicone balloon detached inside cavernous sinus to plug laceration (ocular signs resolve within 7-10 days)

Notes:

CHOROIDAL HEMANGIOMA
=vascular hamartoma*Age:*10-20 years (most common benign tumor in adults)*May be associated with:*Sturge-Weber syndromeLocation:posterior pole temporal to optic disk (70%)√ 0.5-3-mm small tumor√ focal thickening of posterior wall of globe√ enhancement similar to choroid√ retinal detachment (frequent)US: √ hyperechoic homogeneous mass*DDx:*melanoma (choroidal excavitation)

Notes:

COATS DISEASE
=RETINAL TELANGIECTASIA=Pseudoglioma = congenital idiopathic primary vascular malformation of the retina characterized by(1)multiple abnormal telangiectatic retinal vessels(2)lack of blood-retina barrier causing leakage of a lipoproteinaceous exudate into retina + subretinal space with secondary detachment of retina*Age:*6-8 years (but present at birth); M:F = 2:1 ● strabismus ● may present with leukokoria (if retina massively detached) [16% of leukokoria cases] ● loss of vision, secondary glaucoma ● cholesterol crystals at funduscopyLocation:unilateral in 90%*Associated with:*√ retinal detachment√ slight microphthalmia√ NO focal mass / calcification (HALLMARK)US: √ clumpy particulate echoes in subretinal space (due to cholesterol crystals suspended in fluid)√ vitreous + subretinal hemorrhage (frequent)*DDx:*unilateral noncalcifying retinoblastoma (before 3 years of age, no microphthalmia)CT: √ unilateral dense vitreous in normal-sized globeMR: √ hyperintense subretinal exudate on T1WI + T2WI (due to mixture of protein + lipid) / hypointense on T2WI (cholesterol crystals + membranous lipids)√ abnormal enhancement of retina at ora serrata + of detached retinal leaves*DDx:*(1)Persistent hyperplastic primary vitreous (thick tubular retrolental mass)(2)Retinopathy of prematurity*Rx:*photocoagulation / cryotherapy to obliterate telangiectasias (in early stages)

Notes:

COLOBOMA
[Greek koloboun, to mutilate] = incomplete closure of embryonic choroidal fissure affecting eyelid / lens / iris / choroid / retina / macula; autosomal dominant trait with variable penetrance (30%) and expression; bilateral in 60% *Time of insult:*6th week of GA*May be associated with:*encephalocele, agenesis of corpus callosumLocation:in 50% bilateral√ cystic outpouching (= herniation) of vitreous at site of optic nerve attachment√ small globe*DDx:*microphthalmos with cyst = duplication cyst, axial (high) myopia

Notes:

CONGENITAL CATARACT

=opacification of lens*Etiology:*infection, hereditaryLocation:frequently bilateralUS: √ increase in thickness + echogenicity of posterior wall of lens ± intralenticular echoes

Notes:

DACRYOADENITIS

=infection of lacrimal gland*Organism:*staphylococci (most common), mumps, infectious mononucleosis, influenza√ homogeneous enlargement of lacrimal gland√ ± compression of globe

Notes:

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DERMOID CYST OF ORBIT

Most common benign orbital tumor in childhood (45% of all masses) *Age:*1st decade*Histo:*contains keratin, hair, stratified epithelium + dermal appendages within thick capsule; usually arises in fetal cleavage planes (sutures)Location:in anterior extraconal orbit, upper temporal quadrant (60%), upper nasal quadrant (25%)√ well-defined cystic mass ± negative HU numbers√ thick surrounding capsule√ ± expansion / erosion of bony orbitUS: √ encapsulated heterogeneous mass with variable cystic componentMR: √ high signal intensity on T1WI + T2WI

Notes:

Infectious Endophthalmitis *Organism:*bacteria (rare in childhood, trauma, idiopathic), fungi, parasites*Cause:* (a)exogenous endophthalmitis: most commonly related to eye injury / surgery(b)endogenous endophthalmitis: hematogenous spread from distant source of infectionUS: √ medium- to high-intensity echoes dispersed throughout vitreous (DDx: echoes in vitreous hemorrhage are more mobile)CT: √ increased attenuation of vitreous√ uveal-scleral thickening√ decreased attenuation of lens

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Notes:

Sclerosing Endophthalmitis =TOXOCARA CANIS ENDOPHTHALMITIS=granulomatous uveitis resulting in subretinal exudate, retinal detachment, organized vitreous*Age:*2-6-12 years*Mode of infection:* playing in soil contaminated by viable infective eggs from dog excrement (common in playgrounds) *Organism:*helminthic nematode Toxocara canis causing visceral / ocular larva migrans (0.5 mm long, 20 µm wide); endemic throughout world; especially common in southeastern United States*Life cycle:* egg hatches into larva within intestines of definite host (dog) + develops into adult worm; alternatively dog may eat infective-stage larvae from intestines / viscera of other animals; in noncanine host larvae will not develop into adult worm, but burrow through intestinal wall and migrate to liver, lung, and other tissue including brain + eye *Pathophysiology:* migration through human tissue produces a severe eosinophilic reaction that becomes granulomatous; spreads hematogenously to temporal choroid *Path:*retina elevated + distorted + partially replaced by an inflammatory mass containing abundant dense scar tissue; subjacent choroid infiltrated with chronic inflammatory cells including eosinophils; proteinaceous subretinal exudate ● red "hot" eye, photophobia, pain ● anterior chamber flare cells, keratic precipitates ● vitreous synechia ● vitreitis = accumulation of cellular debris in vitreous ● leukokoria (16% of cases of childhood leukokoria) ● fever, hepatomegaly, pneumonitis, convulsions ● peripheral blood eosinophiliaLocation:usually unilateral√ eye of normal size without calcifications√ secondary retinal detachmentUS: √ hypoechoic mass in peripheral fundus√ ± calcificationsCT: √ intravitreal mass√ focal uveoscleral thickening (granulomatous reaction around larva) with contrast enhancement√ increased density of vitreous cavityMR: √ enhancing granuloma isointense to vitreous on T1WI√ mass usually hyperintense relative to vitreous on T2WI, occasionally hypointense (due to dense fibroconnective tissue)*Cx:*retinal detachment (due to subretinal fluid / vitreoretinal traction), cataract*Dx:*(1)Enzyme-linked immunosorbent assay (ELISA) on blood serum / vitreous aspirate(2)Histologic identification of organism*DDx:*retinoblastoma

Notes:

GRAVES DISEASE OF ORBIT
=THYROID OPHTHALMOPATHY= ENDOCRINE EXOPHTHALMOS =increase in orbital pressure produces ischemia, edema, fibrosis of muscles*Etiology:*produced by long-acting thyroid-stimulating factor (LATS); probably immunologic cross-reactivity against antigens shared by thyroid + orbital tissue*Age:*adulthood; 5% younger than 15 years; M:F = 1:4*Histo:*deposition of hygroscopic mucopolysaccharides + glycoprotein (early) + collagen (late); infiltration by mast cells and lymphocytes, edema, muscle fiber necrosis, lipomatosis, fatty degeneration*Time of onset:*signs + symptoms usually develop within one year of the onset of hyperthyroidism ● proptosis◊Most common cause of uni- / bilateral proptosis in adult! ● lid lag = upper eyelid retraction ● periorbital swelling ● conjunctival injection ● restricted ocular motility (correlates with increase in mean muscle diameters) ● progressive optic neuropathy (5%) ● hyperthyroidism; euthyroidism (in 10-15%); severity of orbital involvement unrelated to degree of thyroid dysfunction STAGING (Werner's modified classification): StageI:eyelid retraction without symptomsStageII:eyelid retraction with symptomsStageIII:proptosis >22 mm without diplopiaStageIV:proptosis >22 mm with diplopiaStageV:corneal ulcerationStageVI:loss of sight Location: bilateral in 70-85%; single muscle in 10%; asymmetrical involvement in 10-30%; all muscles equally affected with similar proportional enlargements; superior muscle group most commonly when only single muscle involved [former notion: inferior > medial > superior rectus muscle + levator palpebrae > lateral rectus muscle *mnemonic:*"IM SLow"**I**nferior **M**edial **S**uperior **L**ateral] √ proptosis = globe protrusion >21 mm anterior to interzygomatic line on axial scans at level of lens√ swelling of muscles maximally in midportion (relative sparing of tendinous insertion at globe) = "Coke-bottle" sign√ slight uveal-scleral thickening√ apical crowding = orbital apex involved late (pressure on optic nerve)√ dilatation of superior ophthalmic vein (compromised orbital venous drainage at orbital apex due to enlarged extraocular muscles)√ increase in diameter of retrobulbar optic nerve sheath (dural distension due to accumulation of CSF in subarachnoid space with optic neuropathy)√ increased density of orbital fat (late)√ anterior displacement of lacrimal gland√ intracranial fat herniation through superior ophthalmic fissure (best correlation with compressive neuropathyMR: √ high signal intensity in enlarged eye muscles on T2WI (edema in acute inflammation)*Prognosis:*in 90% spontaneous resolution within 3-36 months; in 10% decrease in visual acuity (corneal ulceration / optic neuropathy)*Rx:*short- and long-term steroid therapy, cyclosporine, radiation, surgical decompression, correction of eyelid position*DDx:*pseudotumor (usually includes tendon of eye muscles)

Notes:

HEMANGIOMA OF ORBIT

Most common benign orbital tumor Location:83-94% retrobulbar (intraconal)√ sharply demarcated oval mass in superior-temporal portion of conus (2/3) often sparing orbital apex√ displacement (not involvement) of optic nerve√ expansion of bony orbit√ uniform / inhomogeneous (when thrombosed) enhancement√ small calcifications (phleboliths)√ puddling of contrast material on angiographyUS: √ well-defined encapsulated mass of intermediate echogenicity√ absent / poor predominantly venous flow

Capillary Hemangioma Of Orbit Cavernous Hemangioma Of Orbit

Notes:

Capillary Hemangioma Of Orbit most common vascular tumor of orbit in children; 5-15% of all pediatric orbital masses *Age:*first 2 weeks of life; 95% in <6 months of age;M < F*Histo:*proliferation of endothelial cells with multiple capillaries ● proptosis, chemosis (= edema) of eyelid + conjunctiva exaggerated by crying ● associated with skin angioma (90%)Location:anterior part of orbit, occasionally posterior√ mass with enhancement equal to / greater than orbital muscle√ poorly marginated (suggesting malignant cause)√ activity in radionuclide flow studiesUS: √ poorly defined heterogeneous mass of intermediate echogenicity√ abundant internal flow decreasing with age*Prognosis:*often increase in size for 6-10 months followed by spontaneous involution within 1-2 years

Notes:

Cavernous Hemangioma Of Orbit *Frequency:*usually tumor of adulthood; 12-15% of all orbital masses; 1-2% of childhood orbital masses*Age:*20-40 years; F > M*Histo:*large dilated venous channels with flattened endothelial cells surrounded by fibrous pseudocapsule ● slowly progressive unilateral proptosis, diplopia, diminished visual acuity (optic nerve compression)

Notes:

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INFECTION OF ORBIT
*Cause:*bacterial infection extending from paranasal sinuses (especially ethmoid + frontal sinuses), face, eyelid, nose, teeth, lacrimal sac through thin lamina papyracea + valveless facial veins into orbit*Organism:*staphylococci, streptococci, pneumococci ● lid edema, ocular pain, ophthalmoplegia ● fever, elevated WBCLocation:preseptal = periorbital soft tissue; subperiosteal; peripheral = extraconal fat; extraocular muscles; central = intraconal fat; optic nerve complex; globe; lacrimal gland*Cx:*epidural abscess, subdural empyema, cavernous sinus thrombosis, cerebral abscess, osteomyelitis

Abscess Of Orbit Cellulitis Of Orbit Edema Of Orbit

Notes:

Abscess Of Orbit Location:most commonly in subperiosteal space on medial wall√ subperiosteal fluid collection√ displacement of thickened periosteal membrane + increased enhancement√ displacement of adjacent fat + extraocular musclesMR: √ hyperintensity on T1WI + T2WI

Notes:

Cellulitis Of Orbit ● limitation of ocular movements ● fever√ thickening of eyelids + septum√ proptosis√ scleral thickening√ enlargement + displacement of extraocular muscles (frequently medial rectus muscle)√ increased attenuation of retro-orbital fat + obliteration of fat planes√ opacification of ethmoid + maxillary sinusesUS: √ diffuse hypoechoic area invading retrobulbar fat*Rx:*antibiotics + corticosteroids*DDx:*cannot be differentiated from edema, chloroma, leukemic infiltrate

Notes:

Edema Of Orbit Location:usually confined to preseptal structures (eyelid, face); involvement of orbital structures (rare)√ swelling of eyelids / face√ increased attenuation of orbital fat + obliteration of fat planes√ displacement + enlargement of extraocular musclesMR: √ hyperintensity on T2WI

Notes:

LYMPHANGIOMA OF ORBIT

*Incidence:*3.5:100,000; 1-2% of orbital childhood masses; 8% of expanding orbital lesions*Histo:*dilated lymphatics, dysplastic venous vessels, smooth muscle, areas of hemorrhage(a)simple / capillary lymphangioma=lymphatic channels of capillary size(b)cavernous lymphangioma=dilated microscopic channels(c)cystic hygroma=macroscopic multilocular cystic mass*Age:*1st decade or later (mean age of 6 years) ● proptosis (sudden proptosis from spontaneous intratumoral hemorrhage = CARDINAL FEATURE; exacerbated during upper respiratory infections [rare]) ● associated with lesions on lid, conjunctiva, cheek ● coincident lymphangiomatous cysts in oral mucosa Location:usually medial to optic nerve with intra- and extraconal component, crossing anatomic boundaries (conal fascia / orbital septum);may involve conjunctiva + lid√ poorly defined multilobulated inhomogeneous lesion√ single / multiple cystlike areas with rim enhancement (after hemorrhage) = blood cyst = "chocolate cyst"√ areas of enhancement (= venous channels) / ring enhancement (after hemorrhage)√ rarely contains phleboliths (DDx: hemangioma, orbital varix)√ mild to moderate enlargement of orbitUS: √ area of predominantly cystic heterogeneous texture with infiltrative bordersMR: √ may show hematoma of various duration within lesion *Prognosis:*no involution, progression slows with termination of body growth*DDx:*orbital varix

Notes:

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LYMPHOMA OF ORBIT

Usually presents without evidence of systemic disease; subsequent development of systemic disease frequent *Incidence:*3rd most common cause of proptosis after orbital pseudotumor + cavernous hemangioma; in 8% of leukemia; in 3-4% of lymphoma*Age:*50 years on average*Type:*usually non-Hodgkin B-cell lymphoma; Burkitt lymphoma with orbit as primary manifestation; Hodgkin disease rare ● painless swelling of eyelid ● exophthalmos (late in course of disease)Location:extraconal (especially lacrimal gland, anterior extraconal space, retrobulbar) > intraconal > optic nerve-sheath complex; may be bilateral◊Lacrimal gland is a common site for leukemic infiltration!*Growth types:* (a)well-defined high-density mass (most commonly about lacrimal gland)(b)diffuse infiltration (tends to involve entire intraconal region)√ slight to moderate enhancementUS: √ solitary / multiple hypoechoic homogeneous masses with infiltrative borders

Notes:

METASTASIS TO ORBIT
*Origin:*only in 50% known; carcinoma of breast + lung (adults); neuroblastoma > Ewing sarcoma, leukemia, Wilms tumor (children)Location:12% intraorbital, 86% intraocular especially in posterior temporal portion of uvea (vascular layer between retina + sclera) near macula; may be bilateral CT: √ small areas of thickening + increased density√ subretinal fluid

Notes:

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NORRIE DISEASE
=RETINAL DYSPLASIA=X-linked recessive disease with; ? inherited form of persistent hyperplastic primary vitreous ● seizures, mental retardation (50%) ● hearing loss, deafness by age 4 (30%) ● bilateral leukokoria + microphthalmia ● cataract, blindness (absence of retinal ganglion cells)√ microphthalmia√ dense vitreous with blood-fluid level√ cone-shaped central retinal detachment√ calcifications

Notes:

OCULAR TRAUMA

● clinical evaluation: testing of visual acuity, slit-lamp evaluation of cornea + anterior segment, intraocular pressure measurement, funduscopyUS (used if ocular media opaque due to vitreous hemorrhage / hyphema / traumatic cataract) 1.Vitreous hemorrhage (53%) ● visual loss frequent√ echogenic material moving freely within vitreous chamber during eye movement*Cx:*retinal detachment (vitreous traction secondary to fibrovascular ingrowth following hemorrhage)*Rx:*vitrectomy2.Total retinal detachment (18%)√ slightly thick line of "V" shape with apex at optic disk√ retina remains bound down at ora serrata3.Vitreous detachment (11%)√ thin undulate mobile line moving away from posterior aspect of globe during eye motion4.Intraocular foreign body (7%)*Cx:*siderosis (if metallic); endophthalmitis5.Choroidal detachment (5%)√ convex lines projecting into the eye from periphery of globe, with most posterior aspect at some distance anterior to + separate from optic disk√ immobile during eye movement6.Lens dislocation (3%)7.Retrohyaloid hemorrhage (2%)√ echogenic material remaining behind detached vitreous capsule during eye movement8.Focal retinal detachment (2%)√ elevated immobile line close to sclera at periphery of globe

Notes:

OPTIC NERVE GLIOMA
=JUVENILE PILOCYTIC ASTROCYTOMA =most common cause of optic nerve enlargement*Incidence:*1% of all intracranial tumors, 2% of childhood orbital masses; 80% of primary tumors of optic nerve*Histo:*proliferation of well-differentiated astrocytes= low-grade glial neoplasm; most commonly pilocytic astrocytoma (in children) + glioblastoma (in adults)*Age:*1st decade (80%); peak age around 5 years; M < F*Associated with:*neurofibromatosis in 10-50%(± bilateral optic gliomas)◊15% of patients with neurofibromatosis have optic nerve gliomas! ● decreased visual acuity, minimal axial proptosis√ tubular / fusiform / excrescentic well-circumscribed enlargement of optic nerve√ posterior extension along optic tracts in 60-70% (indicates nonresectability)√ calcifications (rare)√ same attenuation as normal optic nerve; slight contrast enhancement√ ipsilateral optic canal enlargement (90%) >3 mm / 1 mm difference compared with contralateral sideUS: √ well-defined homogeneous mass of medium echogenicity inseparable from optic nerveMR:more sensitive than CT in detecting intracanalicular + intracranial extent√ isointense to muscle on T1WI√ hyperintense on T2WI*DDx:*optic nerve sheath meningioma (no intracranial extension along optic pathway)

Notes:

OPTIC NERVE SHEATH MENINGIOMA
= PERIOPTIC MENINGIOMA *Incidence:*10% of all intraorbital neoplasms; <2% of intracranial meningiomas*Age:*middle-aged + elderly females; slightly more aggressive in children*Occasionally associated with:* neurofibromatosis (usually in teenagers) *Primary origin:*arising from arachnoid rests in the meningeal investiture of optic nerves in orbit / middle fossa ● progressive loss of visual acuity over months (optic atrophy), proptosis√ ± enlargement of optic canal√ tubular (most commonly) / fusiform / excrescentic thickening of optic nerve√ sphenoid bone hyperostosis√ frequently calcified (HIGHLY SUGGESTIVE)US: √ hypoechoic tumor with irregular borderCECT: enhancement is the rule √ dense linear bands (axial view) as "tram tracks" / ringlike (coronal view) due to tumor enhancement around nonenhancing optic nerve√ minimal extension into optic canal (not uncommon)MR: √ extrinsic soft-tissue mass surrounding optic nerve √ hypointense to fat on T1WI

Notes:

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OPTIC NEURITIS
=nerve involvement by inflammation, degeneration, demyelination*Etiology:*(1) multiple sclerosis (involves optic nerve in 1/3) (2) inflammation secondary to ocular infection (3) degeneration (toxic, metabolic, nutritional) (4) ischemia (5) meningitis / encephalitis◊45-80% of patients develop multiple sclerosis within 15 years of their first episode of optic neuritis! ● ipsilateral orbital pain on eye movement ● sudden onset of unilateral loss of vision over several hours to several daysCT: √ normal / mildly enlarged optic nerve + chiasm√ may show enhancementMR: √ mild enlargement + enhancement of optic nerve well demonstrated on axial T1WI*Prognosis:*spontaneous improvement of visual acuity within 1-2 weeks

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Notes:

PERSISTENT HYPERPLASTIC PRIMARY VITREOUS

=rare condition with persistence + proliferation of embryonic hyaloid vascular system of primary vitreous due to arrest of normal regression*May be associated with:* any severe ocular malformation / optic dysplasia / trisomy 13 ◊Bilaterality is a feature of a congenital syndrome (Norrie disease, Warburg disease)! -Primary vitreous=fibrillar ectodermal meshwork + mesodermal tissue consisting of embryonic hyaloid vascular system; appears during 1st month of life; extends between lens + retina; involutes by 6th month of gestation-Hyaloid artery= important source of intraocular nutrition until 8th month of gestation; arises from dorsal ophthalmic artery at 3rd week of gestation; grows anteriorly with branches supplying vitreous + posterior aspect of lens -Secondary / adult vitreousbegins to form during 3rd gestational month; a watery mass of loose collagen fibers + hyaluronic acid gradually replaces primary vitreous, which is reduced to a small S-shaped remnant (hyaloid canal = Cloquet canal) and serves as lymph channel ● unilateral leukokoria (2nd most common cause) [2-3% of leukokoria cases] ● seizures, mental deficiency, hearing loss ● ± cataract ● ophthalmoscopy: S-shaped tubular mass extending between posterior surface of lens + region of optic nerve head; lens opacity may preclude diagnosis √ microphthalmia = small hypoplastic globe√ retinal detachment (due to vitreoretinal traction in 30%)US: √ hyperechoic band extending from posterior pole of globe to posterior surface of lens (= embryonic rest of primary vitreous)√ central anechoic line (= persistent hyaloid artery) visible in cases of echogenic vitreous hemorrhage√ hyperechoic band extending from papilla to ora serrata (= retinal detachment)CT: √ enhancing cone-shaped central retrolental density extending from lens through vitreous body to back of orbit, just lateral to optic nerve√ small optic nerve√ deformity of globe + lens√ hyperdense vitreous (from previous hemorrhage)√ fluid-fluid levels from breakdown of recurrent hemorrhage in subhyaloid (between vitreous + retina) / subretinal space (between sensory + pigment epithelium)√ NO calcifications MR: √ hyperintense vitreous body on T1WI + T2WI from chronic blood degradation products (methemoglobin) / proteinaceous fluid√ hypo- to isointense thin triangular band with base near optic disc and apex at posterior surface of lens√ marked enhancement of fibrovascular mass within vitreous*Cx:*(1)Glaucoma, cataract from recurrent spontaneous intravitreal hemorrhage (due to friable vessels)(2)Proliferation of embryonic tissue(3)Retinal detachment from organizing hemorrhage / traction(4)Hydrops / atrophy of globe + resorption of lens(5)Phthisis bulbi (scarred shrunken eye)

Notes:

PSEUDOTUMOR OF ORBIT
=IDIOPATHIC INFLAMMATORY PSEUDOTUMOR=nongranulomatous inflammatory process affecting all intraorbital soft tissues*Etiology:* (a)cause not apparent at time of study: bacterial, viral, foreign body(b)systemic disease presently not apparent: sarcoidosis, collagen, endocrine(c)idiopathic: probably abnormal immune response*Incidence:*25% of all cases of unilateral exophthalmos; most common cause of an intraorbital mass lesion in adult*Age:*young female*Histo:*lymphocytic infiltrate*May be associated with:* Wegener granulomatosis, sarcoidosis, fibrosing mediastinitis, retroperitoneal fibrosis, thyroiditis, cholangitis, vasculitis, lymphoma ● unilateral painful ophthalmoplegia ● proptosis, chemosis, lid injection ● limitation of ocular movementLocation:retrobulbar fat (76%), extraocular muscle (57%), optic nerve (38%), uveal-scleral area (33%), lacrimal gland (5%)(a)tumefactive type (common)√ discrete / poorly defined intra- / extraconal mass= "pseudotumor" close to surface margin of globe (b)myositic type (unusual)√ enlargement of one / more extraocular muscles close to insertion in globe with ill-defined margins√ typically involves muscles + tendon insertions (DDx to Graves disease with muscle involvement only)√ increased density of retro-orbital fat (may involve anterior compartment) √ thickening and enhancement of sclera near Tenon capsule√ enlarged lacrimal gland√ proptosisMR: √ lesion isointense to fat on T2WI *Prognosis:* (1) remitting / chronic + progressive course (2) rapid dramatic + lasting response to steroid therapy *DDx:*(1)lymphoma (may be confused with lymphoma clinically, radiographically, pathologically)(2)thyroid ophthalmopathy (tapering of distal muscles, painless proptosis)(3)radiation therapy

Notes:

RETINAL ASTROCYTOMA
=low-grade neoplasm / hamartoma arising from the nerve fiber layer of retina / optic nerve, usually associated with tuberous sclerosis*Etiology:*tuberous sclerosis (53%); neurofibromatosis type 1 (14%); sporadic (33%)*Path:*usually multiple + bilateral in tuberous sclerosis;(1)small flat noncalcified semitranslucent lesion in posterior / peripheral retina(2)"mulberry" lesion = raised white tumor in posterior retina with fine nodularity containing calcifications + cystic fluid accumulations*Histo:*spindle-shaped fibrous astrocytes ● leukokoria (3% of all childhood cases of leukokoria) ● asymptomatic, progressive loss of visionLocation:retina near optic disc√ retinal mass ± enhancement√ typically unilateral (DDx to drusen)*Cx:*(1)Central retinal vein occlusion + secondary hemorrhage(2)Neovascular glaucoma(3)Extensive tumor necrosis

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Notes:

RETINOBLASTOMA
=rare malignant congenital intraocular tumor arising from primitive photoreceptor cells of retina (included in primitive neuroectodermal tumor group)*Types:* (A)Nonheritable form (66%)(1)Sporadic postzygotic somatic mutation (subsequent generations unaffected)*Mean age at presentation:*23 months√ unilateral disease(2)Chromosomal anomaly=monosomy 13 / deletions of 13q*Associated with:*microcephaly, ear changes, facial dysmorphism, mental retardation, finger + toe abnormalities, malformation of genitalia(B)Heritable form(1)Heritable sporadic form (20-25%)=sporadic germinal mutation (50% chance to occur in subsequent generations)*Mean age at presentation:*12 months√ bilateral retinoblastomas in 66%(2)Familial retinoblastoma (5-10%)=autosomal dominant with abnormality of band 14 in chromosome 13 (95% penetrance)*Mean age at presentation:*8 months√ usually 3 to 5 ocular tumors per eye√ bilateral tumors in 66%Risk of secondary nonocular malignancy: osteo~, chondro~, fibrosarcoma, malignant fibrous histiocytoma (20% risk within 10 years, >90% by 30 years of age) **Trilateral retinoblastoma** (rare variant) =bilateral retinoblastomas + neuroectodermal pineal tumor (pineoblastoma)**Quadrilateral retinoblastoma** =trilateral retinoblastoma + 4th focus in suprasellar cistern *Incidence:*1:15,000-34,000 livebirths; most common intraocular neoplasm in childhood; 1% of all pediatric malignancies*Age:*mean age at presentation is 18 months;98% in children <5 years of age; M:F = 1:1*Path:*(1)Exophytic form = proliferation into subretinal space with detachment of retina + invasion of vascular choroid (hematogenous spread)(2)Endophytic form = centripetal tumor invasion causing floating islands of tumor within semiliquid vitreous ± anterior chamber(3)Diffuse form = thin en-plaque lesion extending along retina*Histo:*(a)Flexner-Wintersteiner rosettes (in 50%)= neuronal cells line up around an empty central zone filled with polysaccharides◊Very specific for retinoblastomas!(b)Homer-Wright rosettes = neuronal cells line up around a central area containing a cobweb of filaments (also found in other primitive neuroectodermal tumors)(c)"fleurettes" = flowerlike groupings of tumor cells that form photoreceptor elements (specific for retinal differentiation) ● "cats eye" = leukokoria (whitish mass behind lens) in 60%◊About 50% of all childhood leukokoria are caused by retinoblastoma! ● decreased visual acuity, heterochromia iridis ● strabismus (crossed eyes), proptosis (less common) ● hyphema ● iris neovascularization, phthisis bulbi ● ocular pain from secondary angle-closure glaucomaLocation:posterolateral wall of globe (most commonly); 60% unilateral; 40% bilateral + frequently synchronous (90% bilateral in inherited forms)√ normal ocular sizeUS: √ heterogeneous hyperechoic solid intraocular mass√ cystic appearance upon tumor necrosis√ secondary retinal detachment in all cases√ acoustic shadowing (in 75%)√ vitreous hemorrhage frequentCT: √ solid smoothly marginated lobulated retrolental hyperdense mass in endophytic type (rarer exophytic type grows subretinally causing retinal detachment)√ partial punctate / nodular calcification (50-75-95%)◊Retinoblastoma is the most common cause of orbital calcifications!√ dense vitreous (common)√ extraocular extension (in 25%): optic nerve enlargement, abnormal soft tissue in orbit, intracranial extension√ contrast enhancement usual√ ± macrophthalmiaMR: √ iso- to mildly hyperintense tumor on T1WI relative to vitreous + moderate to marked enhancement√ distinctly hypointense on T2WI (similar to uveal melanoma)√ subretinal exudate usually hyperintense on T1WI + T2WI (proteinaceous fluid)*Cx:*(1)Metastases to: meninges (via subarachnoid space), bone marrow, lung, liver, lymph nodes(2)Radiation-induced sarcomas develop in 15-20%*Prognosis:*spontaneous regression in 1%;√ calcifications = favorable prognostic sign√ contrast enhancement = poor prognostic sign*Mortality:* (a)choroidal invasion: 65% if significant, 24% if slight(b)optic nerve invasion:<10%if not invaded15%if through lamina cribrosa44%if significantly posterior to lamina cribrosa(c)margin of resection not free of tumor: >65%*DDx:*(1)Retinoma = retinocytoma (benign variant)(2)Toxocara canis infection (no calcification)(3)Retrolental fibroplasia (microphthalmia)(4)Coats disease (subretinal exudation, no calcification)(5)Norrie disease (retinal dysplasia)(6)Persistent hyperplastic primary vitreous (hypoplastic globe, no calcification)

Notes:

RETROLENTAL FIBROPLASIA
=RETINOPATHY OF PREMATURITY=bilateral often asymmetric postnatal fibrovascular organization of vitreous humor which usually leads to retinal detachment*Pathophysiology:* retinal vascularization occurs in 4th-9th months of fetal life progressing from the papilla to the periphery; vascularization is incomplete in premature neonates especially in temporal sectors *Predisposed:*premature infants with respiratory distress syndrome requiring prolonged oxygen therapySeverity directly related to: (1) degree of prematurity (2) birth weight (3) amount of oxygen used in therapy ● leukokoria in severe cases (traction retinal detachment, usually bilateral + temporal) [3-5% of all childhood leukokoria cases] ● Ophthalmoscopic stages:1st stage=arteriolar narrowing of most immature vessels at the border of the vascular-avascular retina (from spasm as a reaction to hyperoxygenation)2nd stage=dilatation + elongation + tortuosity of retinal vessels (after oxygen withdrawal)3rd stage=retinal neovascularization with growth into vitreous leads to vitreous hemorrhage4th stage=fibrosis with retraction of fibrovascular tissue + retinal detachment √ bilateral microphthalmia ± retinal detachmentUS: √ hyperechoic tracts extending from temporal side of periphery of retina to vitreous behind the lensCT: √ dense vitreous bilaterally (neovascular ingrowth)√ ± dystrophic calcifications in choroid + lens (late stage)MR: √ hyperintense vitreous on T1WI + T2WI (from chronic subretinal hemorrhage)√ hypointense retrolental mass (apposition of detached leaves of retina displaced from retinal pigment layer) *Prognosis:* (1)spontaneous regression of vitreous neovascularization (85-95%) ± retinal detachment(2)progression to cicatricial stage characterized by formation of dense membrane of gray-white vascularized tissue in retrolental vitreous + retinal detachment + microphthalmia*DDx:*(1)Retinoblastoma (calcifications in eye of normal size)

Notes:

RHABDOMYOSARCOMA
Most common primary malignant orbital tumor in childhood ◊10% occur primarily in orbit◊10% metastasize to / invade orbit*Incidence:*3-4% of all pediatric orbital masses*Histo:*arising from undifferentiated mesenchyma of orbital soft tissues (not from striated muscle)(1) embryonal type (75%) (2) alveolar type (15%) (3) pleomorphic type (10%) *Age at presentation:*average 7 years; 90% by 16 years of age; M > F*Rarely associated with:* neurofibromatosis ● rapidly progressive exophthalmos + proptosis of upper lidLocation:superior orbit / retrobulbar (71%), lid (22%), conjunctiva (7%)√ large soft-tissue density mass with ill-defined margins (extraocular muscles not involved)√ ± extension into preseptal space, adjacent sinus, nasal cavity, intracranial cavity with bony erosion√ may show significant enhancementUS: √ heterogeneous well-defined irregular mass of low to medium echogenicityMetastases:lung, bone marrow, cervical lymph nodes (rare)*Prognosis:*(1)40% survival after exenteration(2)80-90% survival after radiation therapy (4,000-5,000 rad) + chemotherapy (vincristine, cyclophosphamide, adriamycin)*DDx:*pseudotumor, lymphoma

Notes:

UVEAL MELANOMA

Most common primary intraocular neoplasm in adult Caucasian *Age:*50-70 yearsLocation:choroid (85-93%) > ciliary body (4-9%) > iris (3-6%); almost always unilateral ● retinal detachment, vitreous hemorrhage ● astigmatism, glaucomaUS: √ small flat hyperechoic solid massCT: √ ill-defined hyperdense thickening of wall of globe with inward bulgeMR: √ sharply circumscribed hyperintense lesion on T1WI (paramagnetic properties of melanin)Metastases to:globe, optic nerve; liver, lung, subcutis

Notes:

VARIX OF ORBIT

Etiology:(a)Congenital: venous malformation / venous wall weakness(b)Acquired: intraorbital / intracranial AVM ● intermittent exophthalmos associated with straining ● frequent blindness√ involvement of superior / inferior orbital vein; phleboliths rare√ may produce bony erosion without sclerotic reaction√ enlargement of mass during Valsalva maneuver / jugular vein compression√ well-defined markedly enhancing mass√ spontaneous thrombosis (common)US: √ anechoic tubular / oval structure ± thrombus√ venous flow increasing with ValsalvaMR: √ flow void (rapid flow) / flow-related enhancement (slow flow)

Notes:

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WARBURG DISEASE
=autosomal recessive syndrome characterized by(1)bilateral persistent hyperplastic primary vitreous(2)hydrocephalus, lissencephaly(3)mental retardation ● bilateral leukokoria + microphthalmia

Notes:

FACIAL NERVE PARALYSIS
A.INTRACRANIAL SEGMENT(a)intra-axialbrainstem glioma, metastasis, multiple sclerosis, cerebrovascular accident, hemorrhage ● cranial nerve VI also involved(b)extra-axialCPA tumor (acoustic neuroma, meningioma, epidermoid), CPA inflammation (sarcoidosis, basilar meningitis), vertebrobasilar dolichoectasia, AVM, aneurysm ● cranial nerve VIII also involvedB.INTRATEMPORAL SEGMENTfracture, cholesteatoma, paraganglioma, hemangioma, facial nerve schwannoma, metastasis, Bell palsy, otitis media ● loss of lacrimation, hyperacusis, loss of tasteC.EXTRACRANIAL PAROTID SEGMENTforceps delivery, penetrating facial trauma, parotid surgery, parotid malignancy, malignant otitis externa ● preservation of lacrimation, stapedius reflex, taste

Notes:

Hearing Deficit A.CONDUCTIVE HEARING LOSS ● decrease in air conduction via EAC, tympanic membrane, ossicular chain, oval window (sound via headphones) ● normal bone conduction (sound via bone oscillator)(a)destruction of ossicular chain: otitis media(b)restriction of ossicular chain: fenestral otosclerosis◊CT is the modality of choice! B.SENSORINEURAL HEARING LOSS (most common) ● elevated conduction thresholds for bone + air(a)sensory / cochlear SNHL = damage to cochlea / organ of Corti (less common)-bony labyrinth(1)demineralization: otosclerosis (otospongiosis), osteogenesis imperfecta, Paget disease, syphilis(2)congenital deformity: cochlear dys- / aplasia, Michel anomaly, Mondini dysplasia, enlarged vestibular aqueduct syndrome, X-linked sensorineural hearing loss(3)traumatic lesion: transverse fracture, perilymphatic fistula, cochlear concussion(4)destructive lesion: inflammatory lesion, neoplastic lesion◊CT is the modality of choice!-membranous labyrinth(1)enhancement: labyrinthitis, Cogan syndrome (early phase of autoimmune interstitial keratitis), intralabyrinthine schwannoma, site of postinflammatory perilymphatic fistula(2)obliteration: labyrinthitis ossificans, Cogan syndrome (late phase)(3)hemorrhage: trauma, labyrinthitis, coagulopathy, tumor fistulization(4)Meniere disease (vertigo + fluctuating sensory sensorineural hearing loss)◊MRI is the modality of choice!(b)neural / retrocochlear SNHL (more common)=abnormalities of neurons of spiral ganglion + central auditory pathways-IAC / cerebellopontine angle(1)Neoplastic lesions: vestibular / trigeminal schwannoma (acoustic neuroma in 1%), meningioma, arachnoid cyst, epidermoid cyst, leptomeningeal carcinomatosis, lymphoma, lipoma, hemangioma(2)nonneoplastic lesion: sarcoidosis, meningitis, vascular loop, siderosis-intra-axial auditory pathway(brain stem, thalamus, temporal lobe) (1)ischemic lesion(2)neoplastic lesion(3)traumatic lesion(4)demyelinating lesion◊MRI is the modality of choice!

Notes:

Pulsatile Tinnitus ± Vascular Tympanic Membrane =perception of a rhythmic cardiac synchronous soundA.No abnormality (20%)B.Congenital vascular variants (21%)1.Aberrant ICA=result of anastomosis of enlarged inferior tympanic artery with enlarged caroticotympanic artery when cervical ICA is underdeveloped 2.Dehiscent jugular bulb√ absence of bony plate separating jugular bulb from middle ear cavity√ jugular bulb bulges into middle ear cavity 3.High-riding nondehiscent jugular bulb (= jugular megabulb)√ high jugular bulb with diverticulum projecting cephalad into petrous temporal boneC.Acquired vascular lesions (25%)1.Dural AVM2.Extracranial arteriovenous fistula3.High-grade stenotic vascular lesion:carotid artery atherosclerosis, fibromuscular dysplasia, carotid artery dissection 4.Aneurysm involving horizontal segment of petrous ICAD.Temporal bone tumors (31%)1.Paraganglioma (27%):glomus tympanicum, glomus jugulare 2.Meningioma3.HemangiomaE.Miscellaneous1.Cholesterol granuloma

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Notes:

Temporal Bone Sclerosis 1.Otosclerosis = otospongiosis2.**Paget disease** = osteoporosis circumscripta ● sensorineural / mixed hearing loss (cochlear involvement / stapes fixation in oval window)√ usually lytic changes beginning in petrous pyramid + progressing laterally; otic capsule last to be affected√ calvarial changes ± basilar impression3.**Fibrous dysplasia**
monostotic with temporal bone involvement ● painless mastoid swelling ● conductive hearing loss (from narrowing of EAC / middle ear)√ homogeneously dense thickened bone (fibro-osseous tissue less dense than calvarial bone)√ expanded bone with preserved cortex√ lytic lesions (less frequent)√ sparing of membranous labyrinth, facial nerve canal, IAC is the rule4.Osteogenesis imperfecta√ changes similar to otosclerosis**van der Hoeve syndrome** = osteogenesis imperfecta + otosclerosis + blue sclera 5.Meningioma6.Otosyphilis: labyrinthitis + osteitis7.Metastasis8.Ossifying fibroma9.Osteosarcoma10.Osteopetrosis

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Notes:

External Ear Masses A.CONGENITAL1.AtresiaB. INFLAMMATORY 1.Malignant external otitis2.**Keratosis obturans**
bilateral process in association with chronic sinusitis + bronchiectasis *Age:*<40 years3.CholesteatomaC.BENIGN TUMOR1.**Exostosis** = surfers ear*Cause:*irritation by cold water√ bony mass projecting into EAC; often multiple + bilateral2.**Osteoma**
√ may invade adjacent bone; single in EAC / mastoid3.**Ceruminoma**
from apocrine + sebaceous glands; bone erosion mimics malignancy D.MALIGNANT TUMOR1.Squamous cell carcinoma ● often long history of chronic suppurative otitis media = "malignant otitis"2.Basal cell carcinoma3.Melanoma, adenocarcinoma, adenoid cystic carcinoma4.Metastases(a)hematogenous: breast, prostate, lung, kidney, thyroid(b)direct spread: skin, parotid, nasopharynx, brain, meninges(c)systemic:leukemia, lymphoma, myeloma5.Histiocytosis X: in 15% of patients

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Notes:

Middle Ear Masses A.CONGENITAL1.**Aberrant internal carotid artery**

● vascular tympanic membrane ● pulsatile tinnitus√ tubular soft-tissue density entering middle ear cavity posterolateral to cochlea, crossing mesotympanum along cochlear promontory, exiting anteromedial to become horizontal portion of carotid canal√ protrusion into middle ear without bony margin2.Dehiscent jugular bulb**Dehiscent jugular bulb**

● pulsatile tinnitus ● vascular tympanic membrane√ middle ear soft-tissue mass contiguous with jugular foramen√ absence of bony plate separating jugular bulb from posteroinferior middle ear*DDx:*Jugular megabulb (rises above floor of EAC but with preservation of bony plate)B. INFLAMMATORY 1.Cholesteatoma2.Cholesterol granuloma3.Granulation tissue√ linear strands partially opacifying middle ear cavity without bony erosionC.BENIGN TUMOR1.Glomus tumor (multiple in 10%; 8% malignant)(a)Glomus tympanicum: at cochlear promontory√ seldom erodes bone(b)Glomus jugulare: at jugular foramen√ invasion of middle ear from below√ destruction of bony roof of jugular fossa + bony spur separating vein from carotid artery2.Facial neuroma ● persistent Bell palsy (in 5% caused by neurinoma)Location:intracanalicular > IAC√ tubular mass in enlarged / scalloped facial canal3.Ossifying hemangioma4.Choristoma = ectopic mature salivary tissue5.MeningiomaD.MALIGNANT TUMOR1.Squamous cell carcinoma2.Metastasis3.RhabdomyosarcomaLocation: orbit > nasopharynx > ear 4.Adenocarcinoma (rare), adenoid cystic carcinoma ***Mass On The Promontory*** [promontory = bone over basal turn of cochlea] 1.Glomus tympanicum2.Congenital cholesteatoma3.Aberrant carotid artery4.Persistent stapedial artery

Notes:

Inner Ear Masses A.CONGENITAL1.Congenital / primary cholesteatoma = epidermoid tumor (3rd most common CPA tumor)B.INFLAMMATION1.Cholesterol granuloma2.Petrous apex mucoceleC.TUMOR1.Glomus jugulare tumor2.Hemangioma, fibro-osseous lesion3.Metastasis4.Facial nerve neurinoma5.Large CPA tumors: acoustic neuroma, meningioma (2nd most common CPA tumor)

Notes:

Opacification Of Maxillary Sinus A.WITHOUT BONE DESTRUCTION1.Sinus aplasia / hypoplasia*Age:*NOT routinely visualized at birth, by age 6 antral floor at level of middle turbinate, by age 15 of adult size Location:uni- / bilateral depression of orbital floor with enlargement of orbit lateral displacement of lateral wall of nasal fossa with large turbinate2.Maxillary dentigerous cystusually containing a tooth / crown; without tooth = primordial dentigerous cyst 3.Ameloblastoma4.Acute sinusitis air-fluid levelB.WITH BONE DESTRUCTION1.Maxillary sinus tumor2.Infection: aspergillosis, mucormycosis, TB, syphilis3.Wegener granulomatosis; lethal midline granuloma4.Blowout fracture

Notes:

Paranasal Sinus Masses 1.Mucocele2.Mucous retention cyst=smoothly marginated soft-tissue mass from obstruction of small seromucinous gland (commonly in floor of maxilla)3.Sinonasal polyp4.Antrochoanal polyp5.Inverting papilloma6.Sinusitis7.Carcinoma

Notes:

Granulomatous Lesions Of Sinuses A.Chronic irritants1.Beryllium2.Chromate salts B.Infection1.Tuberculosis2.Actinomycosis3.Rhinoscleroma4.Yaws5.Blastomycosis6.Leprosy7.Rhinosporidiosis8.Syphilis9.Leishmaniosis10.Glanders C.Autoimmune disease1.Wegener granulomatosis D.Lymphoma-like lesions1.Midline granuloma E.Unclassified1.Sarcoidosis

Notes:

Hyperdense Sinus Secretions 1.Inspissated secretions2.Fungal sinusitis3.Hemorrhage into sinus4.Chronic sinusitis infected with bacteria (in particular in very long-standing disease / cystic fibrosis)

Notes:

Opacified Sinus & Expansion / Destruction *mnemonic:*"PLUMP FACIES"**P**lasmacytoma **L**ymphoma **U**nknown etiology: Wegener granulomatosis **M**ucocele **P**olyp **F**ibrous dysplasia, **F**ibroma (ossifying) **A**neurysmal bone cyst, **A**ngiofibroma **C**ancer **I**nverting papilloma **E**sthesioneuroblastoma **S**arcoma: ie, rhabdomyosarcoma

Notes:

Nasal Vault Masses A.BENIGN1.Sinonasal polyp2.Inverted papilloma3.Hemangioma ● history of epistaxis4.Pyogenic granuloma√ pedunculated lobular mass5.Granuloma gravidarum=nasal hemangioma of pregnancy6.Hemangiopericytoma7.Juvenile nasopharyngeal angiofibroma√ arises in superior nasopharynx with extension into nose via posterior choanaB.MALIGNANT1.Lymphoma2.Melanoma3.Vascular metastasis

Notes:

Mass In Nasopharynx *mnemonic:*"NASAL PIPE"**N**asopharyngeal carcinoma **A**ngiofibroma (juvenile) **S**pine / skull fracture **A**denoids **L**ymphoma **P**olyp **I**nfection **P**lasmacytoma **E**xtension of neoplasm (sphenoid / ethmoid sinus ca.)

Notes:

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Parapharyngeal Space Mass 1.Asymmetric pterygoid venous plexus√ racemose, enhancing area along medial border of lateral pterygoid muscle2.Abscess*Origin:*pharyngitis (most common), dental infection, parotid calculus disease, penetrating trauma3.Atypical second branchial cleft cyst*Age:*child / young adult ● protruding parotid gland ● bulging posterolateral pharyngeal wall√ cystic mass projecting from deep margin of faucial tonsil toward skull base4.Pleomorphic adenoma of ectopic salivary tissue

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Notes:

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Pharyngeal Mucosal Space **Mass** 1.Asymmetric fossa of Rosenmüller=lateral pharyngeal recess = asymmetry in amount of lymphoid tissue2.Tonsillar abscess ● sore throat, fever, painful swallowing3.Postinflammatory retention cyst√ 1-2-cm well-circumscribed cystic mass4.Postinflammatory calcification ● remote history of severe pharyngitis√ multiple clumps of calcification5.Benign mixed tumor ● pedunculated mass arising from minor salivary glands√ oval / round well-circumscribed mass protruding into airway6.Squamous cell carcinoma√ infiltrating mass with epicenter medial to + invading parapharyngeal space√ middle-ear fluid (eustachian tube malfunction)√ cervical adenopathy7.Non-Hodgkin lymphoma8.Minor salivary gland malignancy9.Thornwaldt cyst

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Notes:

Masticator Space Mass 1.Asymmetric accessory parotid gland*Incidence:*21% of general populationLocation:usually on surface of masseter muscle√ prominent salivary gland tissue2.Benign masseteric hypertrophy*Cause:*bruxism (= nocturnal gnashing of teeth) √ homogeneous enlargement of one / both masseters3.Odontogenic abscess ● bad dentition + trismus4.Sarcoma (chondro-, osteo-, soft-tissue sarcoma)√ infiltrating mass with mandibular destruction5.Malignant schwannoma√ tubular mass along cranial nerve $V_3$6.Non-Hodgkin lymphoma7.Infiltrating squamous cell carcinoma

Notes:

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Carotid Space **Mass** A.VASCULAR LESION1.Ectatic common / internal carotid artery2.Carotid artery aneurysm / pseudoaneurysm3.Asymmetric internal jugular vein4.Jugular vein thrombosisB.BENIGN TUMOR1.Paraganglioma (carotid body tumor + glomus vagale)2.Schwannoma3.Neurofibroma of cranial nerves IX, X, XIC.MALIGNANT TUMOR1.Nodal metastasis from squamous cell carcinoma2.Non-Hodgkin lymphoma

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Notes:

Retropharyngeal Space **Mass** A.INFECTION1.Reactive lymph adenopathy√ nodes >10 mm in diameter2.Abscess√ bow-tie shapeB.BENIGN TUMOR1.Hemangioma2.LipomaC.MALIGNANT TUMOR1.Metastasis from squamous cell carcinoma, melanoma, thyroid carcinoma 2.Non-Hodgkin lymphoma3.Direct invasion by squamous cell carcinoma

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Notes:

Prevertebral Space **Mass** A.PSEUDOTUMOR1.Anterior disk herniation2.Vertebral body osteophyteB.INFLAMMATION1.Vertebral body osteomyelitis2.AbscessC.TUMOR1.Chordoma2.Vertebral body metastasis: lung, breast, prostate, non-Hodgkin lymphoma

Notes:

Inspiratory Stridor In Children 1.Croup2.Congenital subglottic stenosis3.Subglottic hemangioma4.Airway foreign body5.Esophageal foreign body6.Epiglottitis

Notes:

Airway Obstruction In Children ***Nasopharyngeal Narrowing*** (a)Congenital:Choanal atresia, choanal stenosis, encephalocele(b)Inflammatory:Adenoidal enlargement, polyps(c)Neoplastic:Juvenile angiofibroma, rhabdomyosarcoma, teratoma, neuroblastoma, lymphoepithelioma(d)Traumatic:Foreign body, hematoma, rhinolith ***Oropharyngeal Narrowing*** (a)Congenital:Glossoptosis + micrognathia (Pierre Robin, Goldenhar, Treacher Collins syndrome), macroglossia (cretinism, Beckwith-Wiedemann syndrome)(b)Inflammatory:Abscess, tonsillar hypertrophy(c)Neoplastic:Lingular tumor / cyst(d)Traumatic:Hematoma, foreign body ***Retropharyngeal Narrowing*** =potential space (normally <3/4 of AP diameter of adjacent cervical spine in infants / <3 mm in older children)(a)Congenital:Branchial cleft cyst, ectopic thyroid(b)Inflammatory:Retropharyngeal abscess(c)Neoplastic:Cystic hygroma (originating in posterior cervical triangle with extension toward midline + into mediastinum), neuroblastoma, neurofibromatosis, hemangioma(d)Traumatic:Hematoma, foreign body(e)Metabolic:Hypothyroidism ***Vallecular Narrowing*** =valleys on each side of glossoepiglottic folds between base of tongue + epiglottis(a)Congenital:Congenital cyst, ectopic thyroid, thyroglossal cyst(b)Inflammatory:Abscess(c)Neoplastic:Teratoma(d)Traumatic:Foreign body, hematoma ***Supraglottic Narrowing*** =area between epiglottis and true vocal cords(a)Congenital:Aryepiglottic fold cyst(b)Inflammatory:Acute bacterial epiglottitis, angioneurotic edema(c)Neoplastic:Retention cyst, cystic hygroma, neurofibroma(d)Traumatic:Foreign body, hematoma, radiation, caustic ingestion(e)Idiopathic:Laryngomalacia ***Glottic Narrowing*** =area of true vocal cords(a)Congenital:Laryngeal atresia, laryngeal stenosis, laryngeal web (anterior commissure)(b)Neoplastic:Laryngeal papillomatosis(c)Neurogenic:Vocal cord paralysis (most common)(d)Traumatic:Foreign body, hematoma ***Subglottic Narrowing*** =short segment between undersurface of true vocal cords + inferior margin of cricoid cartilage is the narrowest portion of childs airway(a)Congenital:Congenital subglottic stenosis(b)Inflammatory:Croup(c)Neoplastic:Hemangioma, papillomatosis(d)Traumatic:Acquired stenosis (result of prolonged endotracheal intubation in 5%), granuloma(e)Idiopathic:Mucocele = mucous retention cyst (rare complication of prolonged endotracheal intubation) ***Tracheal Narrowing*** A.ANTERIOR COMPRESSION(a)Congenital1.Congenital goiter2.Innominate artery syndrome*Cause:*crowding of thoracic inlet by cervical herniation of an enlarged thymus with development of focal tracheomalacia ● ablation of right radial pulse by rigid endoscopic pressure√ posterior tracheal displacement√ focal collapse of trachea at fluoroscopy√ pulsatile indentation of anterior tracheal wall by innominate artery on MRI*Rx:*surgical attachment of innominate artery to manubrium(b)Inflammatory1.Cervical / mediastinal abscess(c)Neoplastic1.Cervical / intrathoracic teratoma√ amorphous calcifications + ossifications2.Thymoma3.Thyroid tumors4.Lymphoma(d)Traumatic: HematomaB.POSTERIOR TRACHEAL COMPRESSION(a)Congenital1.Vascular ring-complete: double aortic arch, right aortic arch-incomplete: anomalous right subclavian artery√ posterior indentation of esophagus + trachea2.Pulmonary sling=anomalous left pulmonary artery arising from right pulmonary artery, passing between trachea + esophagus en route to left lung3.Bronchogenic cystmost common between esophagus + trachea at level of carina (b)inflammatory: abscess(c)neoplastic: neurofibroma(d)traumatic: esophageal foreign body, esophageal stricture, hematomaC.INTRINSIC TRACHEAL CAUSES(a)Congenital:1.Congenital tracheal stenosis: generalized / segmental = complete cartilaginous ring (instead of horseshoe shape) 2.Congenital tracheomalacia = immaturity of tracheal cartilage ● expiratory stridor √ tracheal collapse on expiration(b)Neoplastic: papilloma, fibroma, hemangioma(c)Traumatic: acquired stenosis (endotracheal + tracheostoma tubes), granuloma, acquired tracheomalacia (cartilage degeneration after inflammation, extrinsic pressure, bronchial neoplasia, TE fistula, foreign body)

Notes:

Tracheal Tumor 1.Adenomatoid cystic carcinoma2.Squamous cell carcinoma3.Carcinoid4.Squamous cell papilloma5.Mucoepidermoid carcinoma

Notes:

Vocal Cord Paralysis 1.Birth injury2.Arnold-Chiari malformation3.Intracranial tumor4.Mediastinal mass / cyst5.Vascular ring6.Thyroidectomy7.Malignancy√ fixed vocal cords (fluoroscopy)

Notes:

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Epiglottic Enlargement A.NORMAL VARIANT1.Prominent normal epiglottis2.Omega epiglottisB.INFLAMMATION1.Acute / chronic epiglottitis2.Angioneurotic edema3.Stevens-Johnson syndrome4.Caustic ingestion5.Radiation therapyC.MASSES1.Epiglottic cyst2.Aryepiglottic cyst3.Foreign body

Notes:

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Aryepiglottic Cyst 1.Retention cyst2.Lymphangioma3.Cystic hygroma4.Thyroglossal cyst ● may be symptomatic at birth✓ well-defined mass in aryepiglottic fold

Notes:

Solid Neck Masses In Childhood 1.Lymphadenopathy2.Fibromatosis colli3.Malignancy: neuroblastoma (most common), lymphoma4.Teratoma5.Hemangioma6.Lipoma7.Thyroid mass8.Ectopic thymus

Notes:

Lymph Node Enlargement Of Neck A.NORMAL LYMPH NODES√ few small oval hypoechoic√ ± central linear echogenicity (= invaginating hilar fat)√ larger in transverse than anteroposterior dimensionB.MALIGNANT LYMPH NODES√ increased anteroposterior diameter√ prominent calcifications suggestive of medullary thyroid cancer√ minimal axial diameter of 11 mm (in squamous cell carcinoma)CT: √ marginal enhancement

Notes:

Congenital Cystic Lesions Of Neck ◊95% of all branchial cleft anomalies arise from 2nd branchial apparatus!1.**Second branchial cleft cyst** =incomplete obliteration of 2nd branchial cleft tract (cervical sinus) resulting in sinus tract / fistula / cyst*Age:*young to middle-aged adultLocation:parotid space near mandibular angle, parapharyngeal space ● history of multiple parotid abscesses unresponsive to drainage + antibiotics ● otorrhea (if connected to external auditory canal)√ cystic oval / round mass near mandibular angle√ displacement of sternocleidomastoid muscle posteriorly, carotid artery + jugular vein posteromedially, submandibular gland anteriorly√ may insinuate between internal + external carotid artery (PATHOGNOMONIC)√ cyst may enlarge after upper respiratory tract infection / injury*DDx:*necrotic neural tumor, cervical abscess, submandibular gland cyst, cystic lymphangioma, necrotic metastatic / inflammatory lymphadenopathy 2.**First branchial cleft cyst**
Residual embryonic tract begins near submandibular triangle + ascends through the parotid gland, terminates at junction of cartilaginous + bony external auditory canal *Incidence:*8% of all branchial cleft anomalies*Age:*middle-aged women ● enlarging mass near lower pole of parotid gland*DDx:*inflammatory parotid cyst, benign cystic parotid tumor, necrotic metastatic lymphadenopathy 3.**Cervical thymic cyst**
forms along migratory tract of thymic tissue into mediastinum *Age:*<5 years of age; M > FNo association with myasthenia gravis! Location:from angle of mandible to anterior mid-neck√ uni- / multilocular mostly unilateral cyst 4.**Parathyroid cyst**
*Age:*30-50 years ● hormonally inactive√ noncolloidal cyst near lower pole of thyroid gland 5.Thyroglossal duct cyst6.Lymphangioma / cystic hygroma 7.**Dermoid cyst**
(1)Cystic teratoma(a)epidermoid cyst = lined by simple squamous epithelium without adnexal structures(b)dermal cyst = epithelial-lined cyst containing hair + sebaceous glands(c)teratoid cyst = lined with squamous / respiratory epithelium containing derivatives of skin appendages + endoderm + mesoderm(2)Nonteratomatous epithelial-lined cystLocation: -dorsum of nose in infants (most common)-midline anterior floor of mouth:(a)sublingual between mylohyoid muscle + tongue (DDx: inclusion cyst, ranula)(b)submental between platysma + mylohyoid muscle 8.Ectopic bronchogenic cyst ● stridor√ indentation of trachea

Notes:

Branchial Fistula 1.Third branchial fistula*Internal opening:*piriform fossa anterior to fold formed by internal laryngeal nerve*Course:*through thyrohyoid membrane, over hypoglossal nerve, between internal + external carotid arteries, caudolateral / posterolateral to proximal internal + common carotid arteries*External opening:*at base of neck anterior to sternocleidomastoid muscle2.Fourth branchial fistula*Internal opening:*apex of piriform sinus*Course:*between cricoid + thyroid cartilage, below cricothyroid muscle, caudal course between trachea + carotid vessels, deep to clavicle into mediastinum, looping forward below aorta (left side) / right subclavian artery (right side), ascending posterior to common carotid artery, passing over hypoglossal nerve*External opening:*at base of neck anterior to sternocleidomastoid muscle

Notes:

Air-containing Masses Of Neck 1.Laryngocele2.Tracheal diverticulumarising from anterior wall of trachea close to thyroid3.Zenker diverticulum4.Lateral pharyngeal diverticulumlocated in tonsillar fossa / vallecula / pyriform fossa

Notes:

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Parotid Gland Enlargement A.LOCALIZED INFLAMMATORY DISEASE1.Chronic recurrent sialadenitis2.Sialosis3.Sarcoidosis4.Tuberculosis5.Cat-scratch fever6.Syphilis7.Abscess8.Reactive adenopathyB.SYSTEMIC AUTOIMMUNE RELATED DISEASE1.Sjögren disease (= myoepithelial sialadenitis)2.Mikulicz diseaseC.NEOPLASM(a)benign tumor1.Pleomorphic / monomorphic adenoma2.Cystadenolymphoma (= Warthin tumor)3.Benign lymphoepithelial cysts (AIDS)4.Lipoma5.Facial neuroma6.Oncocytoma(b)primary malignant tumor1.Mucoepidermoid carcinoma2.Adenoid cystic carcinoma (= cylindroma)3.Malignant mixed tumor4.Adenocarcinoma5.Acinus cell carcinoma(c)metastatic tumor◊Parotid gland undergoes late encapsulation, which leads to incorporation of lymph nodes!1.Squamous cell carcinoma2.Melanoma3.Non-Hodgkin lymphomaD.LYMPHOPROLIFERATIVE DISORDER1.Lymphoma2.Primary Non-Hodgkin lymphomaE.CONGENITAL1.First branchial cleft cyst

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Notes:

Multiple Lesions Of Parotid Gland 1.Warthin tumor2.Metastases to lymph nodes: squamous cell carcinoma of skin, malignant melanoma, Non-Hodgkin lymphoma3.Benign lymphoepithelial cysts (AIDS)

Notes:

Congenital Dyshormonogenesis 1.Trapping defect=defective cellular uptake of iodine into thyroid, salivary glands, gastric mucosa;◊high doses of inorganic iodine facilitate diffusion into thyroid permitting a normal rate of thyroid hormone synthesis◊normal ratio of iodine concentrations for gastric juice:plasma = 20:1√ nearly entire dose of administered radioiodine is excreted within 24 hours2.Organification defect=deficient peroxidase activity, which catalyzes the oxidation of iodide by H_2O_2 to form monoiodotyrosine (MIT) / diiodotyrosine (DIT) ● high serum TSH ● low serum T_4 ● diffuse symmetric thyromegaly√ high thyroidal uptake of radioiodine / pertechnetate√ rapid I-131 turnover√ positive perchlorate washout test**Pendred syndrome** = autosomal recessive trait of deficient peroxidase regeneration characterized by hypothyroidism + goiter + nerve deafness 3.Deiodinase (dehalogenase) defect=deficient deiodination of MIT / DIT to release iodide which is reutilized to synthesize thyroid hormone production ● hypothyroidism ● identification of MIT + DIT in serum + urine following administration of I-131 ● "intrinsic" iodine deficiency goiter√ high thyroidal I-131 uptake√ rapid intrathyroidal turnover of I-1314.Thyroxin-binding globulin (TBG) deficiency ● abnormal T_4 transport ● low bound serum T_4 concentration ● euthyroid5.End-organ resistance to thyroid hormone ● high serum T_4 ● euthyroid / hypothyroid ● growth retardation√ goiter√ stippled epiphyses

Notes:

Hyperthyroidism 1.Graves disease (most common)2.Toxic nodular goiter3.Iodine-induced hyperthyroidism = Jod-Basedow4.Thyroiditis(a)Hashimoto thyroiditis = chronic lymphocytic thyroiditis(b)Subacute thyroiditis = de Quervain thyroiditis(c)Painless thyroiditisUS: √ decrease in overall echogenicity√ discrete nodules (50%)5.Thyrotoxicosis medicamentosa / factitiasurreptitious self-administration of thyroid hormones 6.Struma ovarii= ovarian teratoma containing thyroid tissue 7.Hydatidiform mole / choriocarcinoma / testicular trophoblastic carcinoma= stimulation of thyroid by HCG 8.Pituitary hyperthyroidism = pituitary neoplasm ● ± acromegaly ● ± hyperprolactinemia9.Thyroid carcinoma / hyperfunctioning metastasesvery rare (25 cases)
Hypothyroidism A.PRIMARY HYPOTHYROIDISM (most common)=thyroids inability to produce sufficient thyroid hormone1.Agenesis of thyroid2.Congenital dyshormonogenesis3.Chronic thyroiditis4.Previous radioiodine therapy5.Ectopic thyroid (1:4,000)B.SECONDARY HYPOTHYROIDISM=failure of anterior pituitary to release sufficient quantities of TSH1.Sheehan syndrome2.Head trauma3.Pituitary tumor (primary / secondary)4.Aneurysm5.SurgeryC.TERTIARY / HYPOTHALAMIC HYPOTHYROIDISM=failure of hypothalamus to produce sufficient amounts of TRH

Notes:

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Decreased / No Uptake Of Radiotracer A.BLOCKED TRAPPING FUNCTION1.Iodine load (most common)=dilution of tracer within flooded iodine pool (from administration of radiographic contrast / iodine-containing medication) ◊Suppression usually lasts for 4 weeks!2.Exogenous thyroid hormone (replacement therapy)suppresses TSH release B. BLOCKED ORGANIFICATION1.Antithyroid medication (propylthiouracil (PTU) / methimazole) / goitrogenic substances◊ Tc-99m uptake not inhibitedC.DIFFUSE PARENCHYMAL DESTRUCTION1.Subacute / chronic thyroiditisD.HYPOTHYROIDISM1.Congenital hypothyroidism2.Surgical / radioiodine ablation3.Thyroid ectopia (struma ovarii, intrathoracic goiter)*mnemonic:*"H MITTE"**H**ypothyroidism (congenital) **M**edications: PTU, perchlorate, Cytomel, Synthroid, Lugol solution **I**odine overload (eg, after IVP) **T**hyroid ablation (surgery, radioiodine) **T**hyroiditis (subacute / chronic) **E**ctopic thyroid hormone production

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Notes:

Increased Uptake Of Radiotracer *mnemonic:*"THRILLEr"**T**hyroiditis (early Hashimoto) **H**yperthyroidism (diffuse / nodular) **R**ebound after withdrawal of antithyroid medication **I**odine starvation **L**ow serum albumin **L**ithium therapy **E**nzyme defect

Notes:

Prominent Pyramidal Lobe =distal remnant of thyroid descent tract1.Normal variant: present in 10%2.Hyperthyroidism3.Thyroiditis4.S/P thyroid surgery*DDx:*esophageal activity from salivary excretion (disappears after glass of water)

Notes:

Thyroid Calcifications =benign calcifications = stromal calcifications in adenoma√ coarse calcifications with rough outline√ alignment along periphery of lesion√ irregular distribution ***Psammoma Bodies*** =microcalcifications (<1 mm) occur in 54% of thyroid neoplasms√ seen on xeroradiography in 94%1. Papillary carcinoma61%2. Follicular carcinoma26%3.Undifferentiated carcinoma13%

Notes:

Cystic Areas In Thyroid 15-25% of all thyroid nodules! A.Anechoic fluid + smooth regular wall:1.Colloid accumulation in goiter = colloid-filled dilated macrofollicle2.Simple cyst (extremely uncommon)B.Solid particles + irregular outline:1.Hemorrhagic colloid nodule2.Hemorrhagic adenoma (30%)3.Necrotic papillary cancer (15%)4.Liquefaction necrosis in adenoma / goiter5.Abscess6.Cystic parathyroid tumor ● bloody fluid = benign / malignant lesion ● clear amber fluid = benign lesion●Cystic lesions often yield insufficient numbers of cells!

Notes:

Thyroid Nodule *Incidence:*(increasing with age)(a)4-8% by palpation (>2 cm in 2%, 1-2 cm in 5%,<1 cm in 1%); M:F = 1:4(b)50% by autopsy / thyroid US if clinically normal: multiple in 38%, solitary in 12% (occult small cancers found in 4%)A.THYROID ADENOMA1.Colloid / adenomatous nodule = adenomatous hyperplasia / degenerative involuted nodule (42-77%)2.Follicular adenoma (15-40%)3.Ectopic parathyroid adenomaB.INFLAMMATION / HEMORRHAGE1.Inflammatory lymph node in subacute + chronic thyroiditis2.Hemorrhage / hematoma: frequently associated with adenomas3.AbscessC.CARCINOMA (8-17%)1.Thyroid carcinoma(a)papillary carcinoma (70%)(b)follicular (15%)(c)medullary carcinoma (5-10%)(d)anaplastic carcinoma (5%)(e)thyroid lymphoma (5%)2.Nonthyroidal neoplasmmetastasis from breast, lung, kidney, malignant melanoma, Hodgkin disease 3.Hürthle cell carcinoma√ very thin hypoechoic halo4.Carcinoma in situ√ echogenic area inside a goiter nodule Role of fine-needle aspiration biopsy (FNAB): (large-needle biopsy has more complications with no increase in diagnostic yield) ◊FNAB as initial test leads to a better selection of patients for surgery than any other test!Diagnostic accuracy of 70-97%: (a)70-80% negative(b)10% positive specimens (3-6% false-positive rate often due to Hashimoto thyroiditis)(c)10-20% indeterminateUp to 20% nondiagnostic (too few cells) material Role of imaging: ◊ Imaging cannot reliably distinguish malignant + benign nodules! (a)radionuclide scanning-useful in indeterminate cytology◊Hyperfunctioning nodule is almost always benign!(b)ultrasound-best method to determine volume of nodule-useful during follow-up to distinguish nodular growth from intranodular hemorrhage

Notes:

Discordant Thyroid Nodule =nodule hyperfunctioning on Tc-99m pertechnetate scan + hypofunctioning on I-131 scan, which indicates reduced organification capacity*Cause:* 1.Malignancy:follicular / papillary carcinoma2.Benign lesion:follicular adenoma / adenomatous hyperplasia(autonomous nontoxic nodules have accelerated iodine turnover and discharge radioiodine as hormone within 24 hours)

Notes:

Hot Thyroid Nodule *Incidence:* 8% of Tc-99m pertechnetate scans1.Adenoma(a)Autonomous adenoma = TSH-independent ● euthyroid (80%), thyrotoxicosis (20%)√ partial / total suppression of remainder of gland(b)Adenomatous hyperplasia = TSH-dependentsecondary to defective thyroid hormone production 2.Thyroid carcinoma (extremely rare)√ discordant uptake N.B.:any hot nodule on Tc-99m scan must be imaged with I-123 to differentiate between autonomous or cancerous lesion

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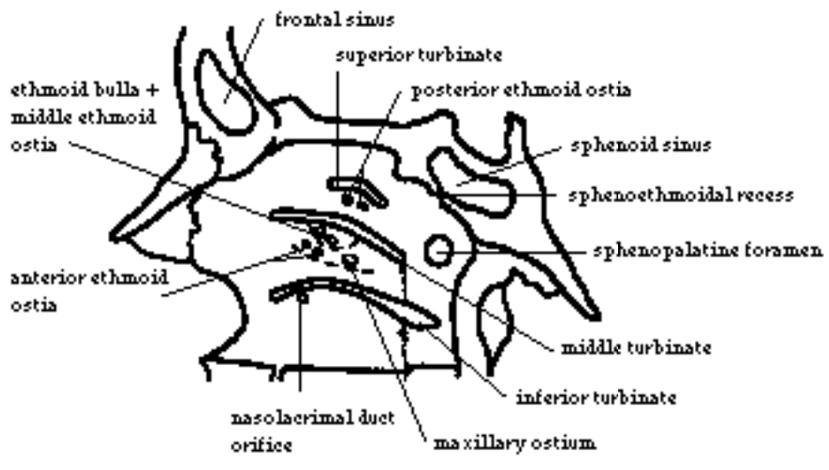
Notes:

Cold Thyroid Nodule A.BENIGN TUMOR1. Nonfunctioning adenoma2. Cyst (11-20%)3. Involutional nodule4. Parathyroid tumorB.INFLAMMATORY MASS1. Focal thyroiditis2. Granuloma3. AbscessC.MALIGNANT TUMOR1. Carcinoma2. Lymphoma3. MetastasisUS features of cold nodule: √ hypoechoic (71%)√ isoechoic (22%)√ mixed echogenicity (4%)√ hyperechoic (3%)√ cystic (rarely malignant)◊A palpable hypofunctioning nodule in a patient with Graves disease is likely malignant!
mnemonic:"CATCH LAMP"**C**olloid cyst **A**denoma (most common) **T**hyroiditis **C**arcinoma **H**ematoma **L**ymphoma, **L**ymph node **A**bscess **M**etastasis (kidney, breast) **P**arathyroid
Probability Of A Cold Nodule To Represent Thyroid Cancer: ◊Solitary cold nodules by scintigraphy are multinodular by US in 20-25%!(a)15-25% for solitary cold nodule(b)1-6% for multiple nodules (DDx: multinodular goiter)(c)with history of neck irradiation in childhood-solitary nodule found in 70%(cancerous in 31%) -multiple nodules found in 25%(cancerous in 37%) -normal thyroid scan found in 5%(cancer detected in 20%)

Notes:

PARANASAL SINUSES
Mucus production of 1 L/day; mucus blanket turns over every 20-30 minutes; irritants are propelled toward nasopharynx at a rate of 1 cm/minute



View of Lateral Nasal Wall (turbinates removed)

Maxillary Sinus Ethmoid Sinuses Frontal Sinus Sphenoid Sinus

Notes:

Maxillary Sinus Size:6-8 cm at birthWalls:roof = floor of orbit; posterior wall abuts pterygopalatine fossaExtension: 4-5 mm below level of nasal cavity by age 12Ostium:maxillary ostium + infundibulum enter middle meatus within posterior aspect of hiatus semilunaris; additional ostia may be presentPlain film:present at birth; visible at 4-5 months; completely developed by 15 years of ageVariations:sinus hypoplasia in 9%; aplasia in 0.4%

Notes:

Ethmoid Sinuses Size:adult size by age 12; 3-18 air cells per sideWalls:roof = floor of anterior cranial fossa;lateral wall = lamina papyraceaPlain film:very small at birth; visible at 1 year of age; completely developed by puberty(a)**anteromedial ethmoid air cells**
2-8 cells with a total area of 24 x 23 x 11 mm Ostia:opening into anterior aspect of hiatus semilunaris of middle meatus (anterior group), opening into ethmoid bulla (middle group)*Agger nasi cells* =anteriormost ethmoid air cells in front of the attachment of middle turbinate to cribriform plate near the lacrimal duct=anterior, lateral + inferior to frontoethmoidal recess = anteromedial margin of orbitPrevalence:present in >90%*Ethmoidal bulla* =ethmoidal air cell above + posterior to infundibulum + hiatus semilunaris, located outside the lamina papyracea at the lateral wall of the middle meatus*Haller cells* =anterior ethmoid air cells inferolateral to ethmoidal bulla, on lateral wall of infundibulum, along inferior margin of orbit / roof of maxillary sinus, protruding into maxillary sinusPrevalence:10-45%(b)**posterior ethmoid air cells**
1-8 cells, larger cells, total area smaller than that of anteromedial group Location:behind the basal (= ground) lamella of the middle turbinateOstium:into superior meatus / supreme meatus, ultimately draining into sphenoethmoidal recess of nasal cavity*Onodi cell* =most posterior ethmoid air cell pneumatized into sphenoid bone ± surrounding the optic canalLocation:superolateral to sphenoid sinus

Notes:

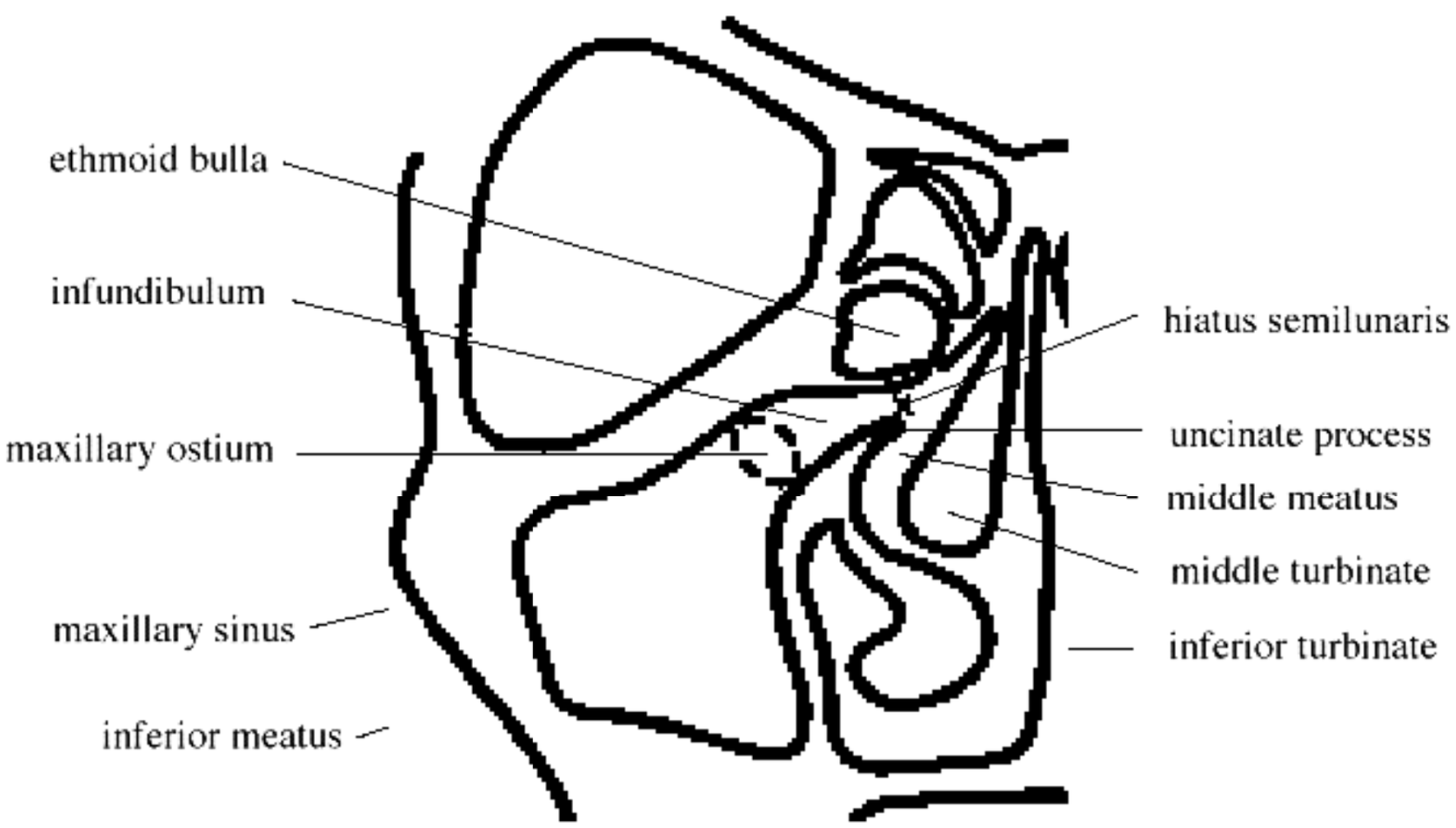
Frontal Sinus Size:28 x 24 x 20 mm in adults, rapid growth until the late teensWalls:posterior wall = anterior cranial fossa; inferior wall = anterior portion of roof of orbitOstium:into frontal recess of middle meatus via frontoethmoidal recess (= nasofrontal duct)Plain film:visible at age 6 yearsVariations:sinus aplasia in up to 4% (in 90% with Down syndrome)

Notes:

Sphenoid Sinus Size:20 x 23 x 17 mm in adults, small evagination of sphenoethmoidal recess at birth, invasion of sphenoid bone begins at age 5 years; aerated extensions into pterygoid plates (44%) + into clinoid processes (13%)Walls:roof = floor of sella turcica; anterior wall shared with ethmoid sinuses; posterior wall = clivus; inferior wall = roof of nasopharynxOstium:10 mm above sinus floor into sphenoethmoidal recess posterior to superior meatus at level of sphenopalatine foramenPlain film:appears by 3 years of age; continues to grow posteriorly + inferiorly into the sella until adulthood

Notes:

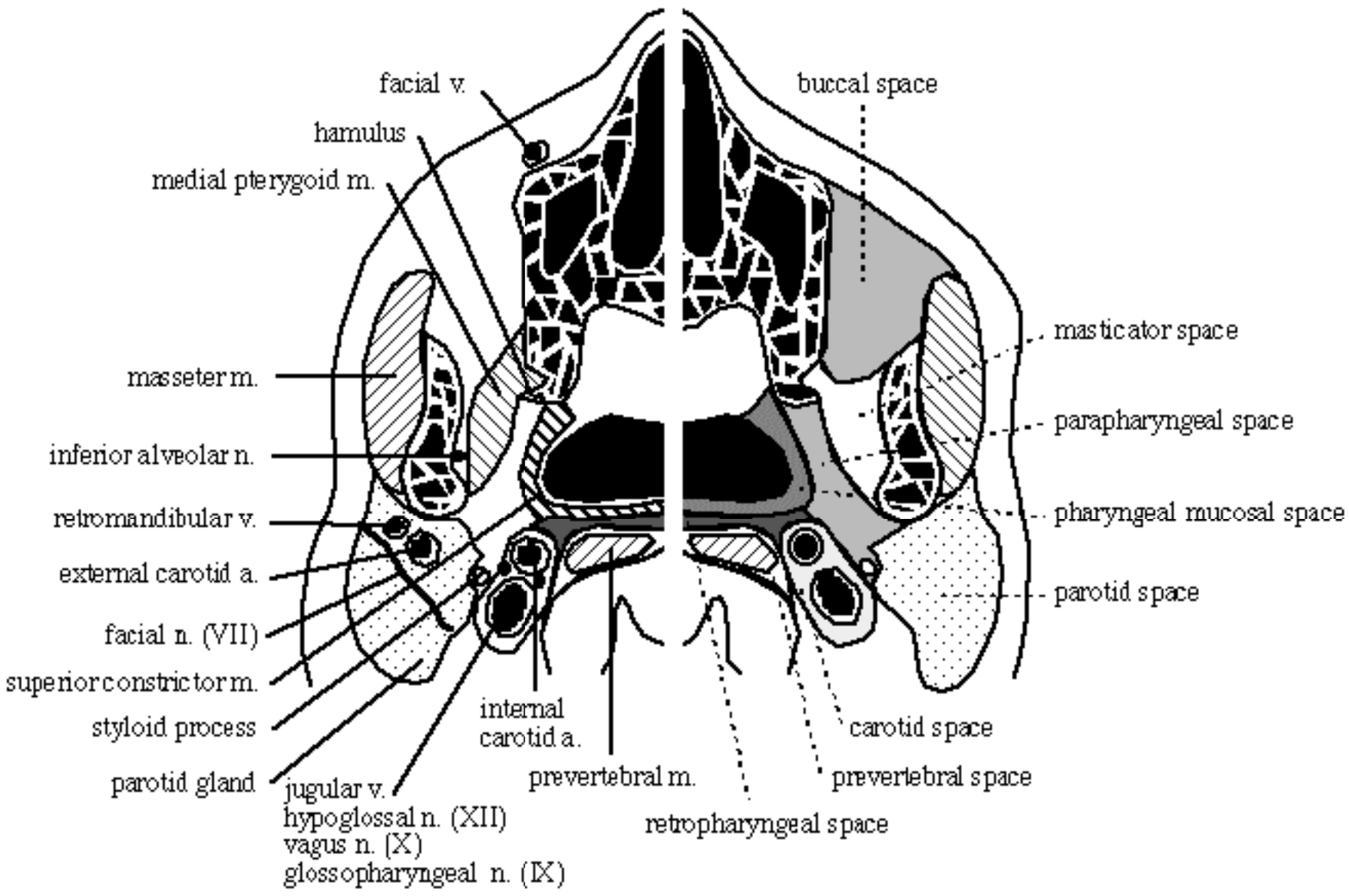
OSTIOMEATAL UNIT
=area of superomedial maxillary sinus + middle meatus as the common mucociliary drainage pathway of frontal maxillary, and anterior + middle ethmoid air cells into the noseCoronal CT:visualized on two or three 3-mm-thick sections Components: 1.Infundibulum= flattened conelike passage between inferomedial border of orbit / ethmoid bulla (laterally) + uncinate process (medially) + maxillary sinus (inferiorly) + hiatus semilunaris (superiorly) 2.Uncinate process=key bony structure in lateral nasal wall below hiatus semilunaris in middle meatus defines hiatus semilunaris together with adjacent ethmoid bulla pneumatized in <2.5% of patients3.Ethmoid bulla located in cephalad recess of middle meatus4.Hiatus semilunarisfinal segment for drainage of maxillary sinus; located just inferior to ethmoid bulla in middle meatus Ostia: (1)multiple ostia from anterior ethmoid air cells (at its anterior aspect)(2)maxillary ostium infundibulum (at its posterior aspect) Anatomic variations predisposing to ostiomeatal narrowing: 1.Concha bullosa (4-15%) = aerated / pneumatized middle turbinate2.Intralamellar cell = air cell within vertical portion of middle turbinate3.Oversized ethmoid bulla4.Haller cells5.Uncinate process bulla6.Bowed nasal septum7.Paradoxical middle turbinate = convexity of turbinate directed toward lateral nasal wall (10-26%)8.Deviation of uncinate process These conditions are not disease states per se!



Coronal Scan of Ostiomeatal Unit

Notes:

Branchial cleft development
-6 paired branchial arches are responsible for formation of lower face + neck-each branchial cleft arch contains a central core of cartilage + muscle, a blood vessel and a nerve-arches form 5 ectodermal "clefts" / grooves on outer aspect of neck + 5 endodermal pharyngeal pouches separated by a membrane Formation:during 4th-6th



week of embryonic development
Transaxial Scan Through Level of Lower Nasopharynx

1st Branchial Arch =maxillomandibular arch
(a)large ventral / mandibular prominence*forms:*mandible, incus, malleus, muscles of mastication(b)small dorsal / maxillary prominence*forms:*maxilla, zygoma, squamous portion of temporal bone, cheek, portions of external ear*nerve:*mandibular division of trigeminal nerve*pouch forms:* mastoid air cells + eustachian tube*cleft forms:* external auditory canal + tympanic cavity **2nd Branchial Arch =** Hyoid Arch *nerve:*facial nerve*arch forms:*thyroid gland, stapes, portions of external ear, muscles of facial expression*pouch forms:* palatine tonsil + tonsillar fossacleft involutes completely by 9th fetal week; 2nd arch overgrows 2nd + 3rd + 4th clefts to form *cervical sinus* which creates a tract that runs from supraclavicular area just lateral to carotid sheath, turns medially at mandibular angle between external + internal carotid artery, terminates in tonsillar fossa **3rd Branchial Arch** sunk into retrohyoid depression *nerve:*glossopharyngeal nerve*arch forms:*glossoepiglottic fold, superior constrictor m., internal carotid a., parts of hyoid bone*pouch forms:* (a)thymus gland, which descends into mediastinum by 9th fetal week(b)inferior parathyroid glands passing down with the thymus **4th Branchial Arch** sunk into retrohyoid depression *nerve:*superior laryngeal branch of vagus nerve*arch forms:*epiglottis + aryepiglottic folds, thyroid cartilage, cricothyroid m., left component of aortic arch, right component of right proximal subclavian a.*pouch forms:*superior parathyroid glands, apex of piriform fossa*cleft forms:*ultimobranchial body, which provides parafollicular = "C" cells of thyroid **5th + 6th Branchial Arches** cannot be recognized externally *nerve:*recurrent laryngeal branch of vagus nerve

Notes:

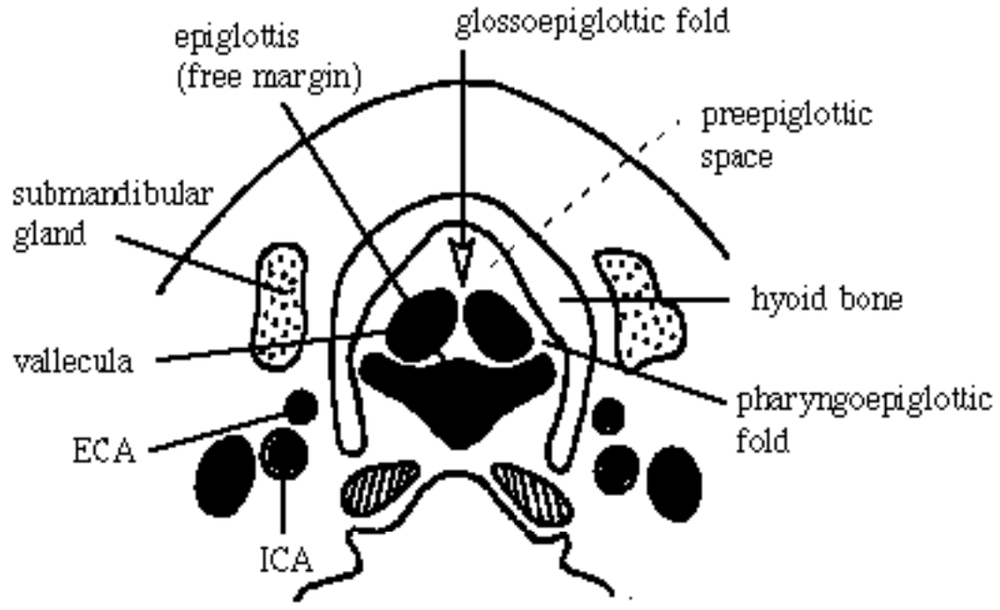
Oral cavity
comprises lip, upper + lower gingiva, buccal mucosa, hard palate, floor of mouth, anterior 2/3 of tongue

Notes:

Oropharynx
consists of (a)pharyngeal wall between nasopharynx + pharyngoepiglottic fold(b)soft palate(c)tonsillar region(d)tongue baseBorders: (a)superior: soft palate and Passavant ridge (= ridge of pharyngeal muscle that opposes the soft palate when soft palate is elevated)(b)anterior: plane that joins the posterior border of soft palate, anterior tonsillar pillars, circumvallate papillae(c)posterior: posterior pharyngeal wall(d)inferior: vallecula(e)lateral: tonsillar region consisting of anterior tonsillar pillar (= palatoglossus muscle) + palatine / faucial tonsil + posterior tonsillar pillar (= palatopharyngeus muscle)

Notes:

Hypopharynx
=compartment of aerodigestive tract between hyoid bone + inferior aspect of cricoid cartilage1.Pyriform sinuses=two symmetric lateral stalactites of air hanging from hypopharynx behind larynx-inferior wall: level of cricoarytenoid joint-anteromedial wall: lateral wall of aryepiglottic fold-lateral wall: abuts posterior ala of thyroid cartilage-posterior wall: most lateral aspect of posterior hypopharyngeal wall2.Postcricoid area = pharyngoesophageal junction extends from level of arytenoid cartilages to inferior border of cricoid cartilage-anterior wall of hypopharynx = posterior wall of lower larynx = "party wall"3.Posterior hypopharyngeal wallextends from

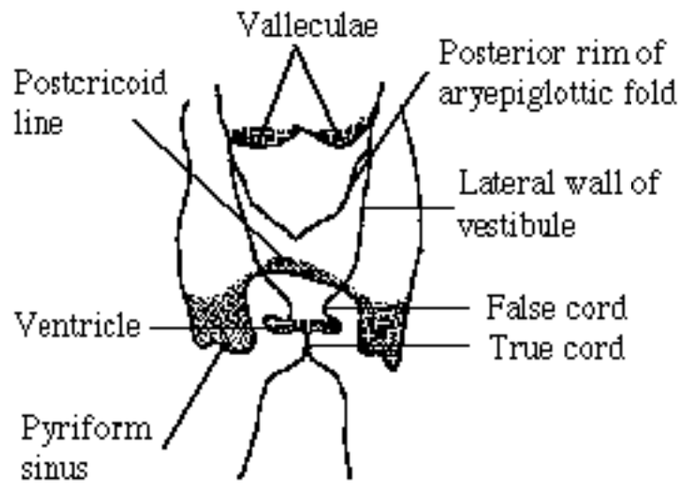


level of valleculae to cricoarytenoid joints

Hyoid Bone Level

Notes:

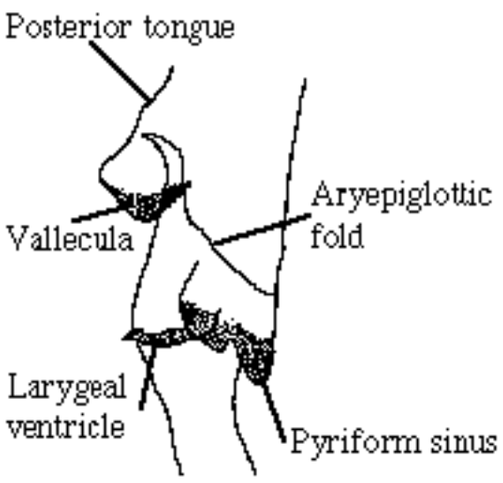
Larynx

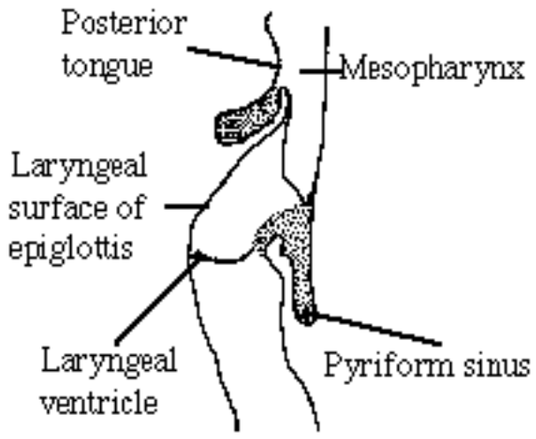


Vertical length:44 mm (males), 36 mm (females), at 4th-6th cervical vertebrae

Frontal Laryngopharyngogram

During Phonation

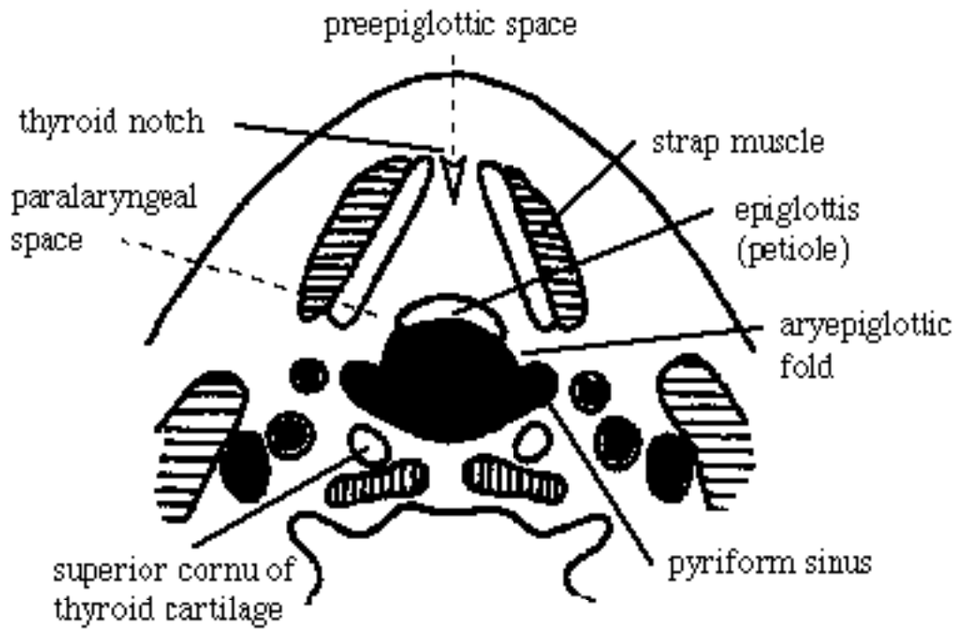




Lateral Laryngogram

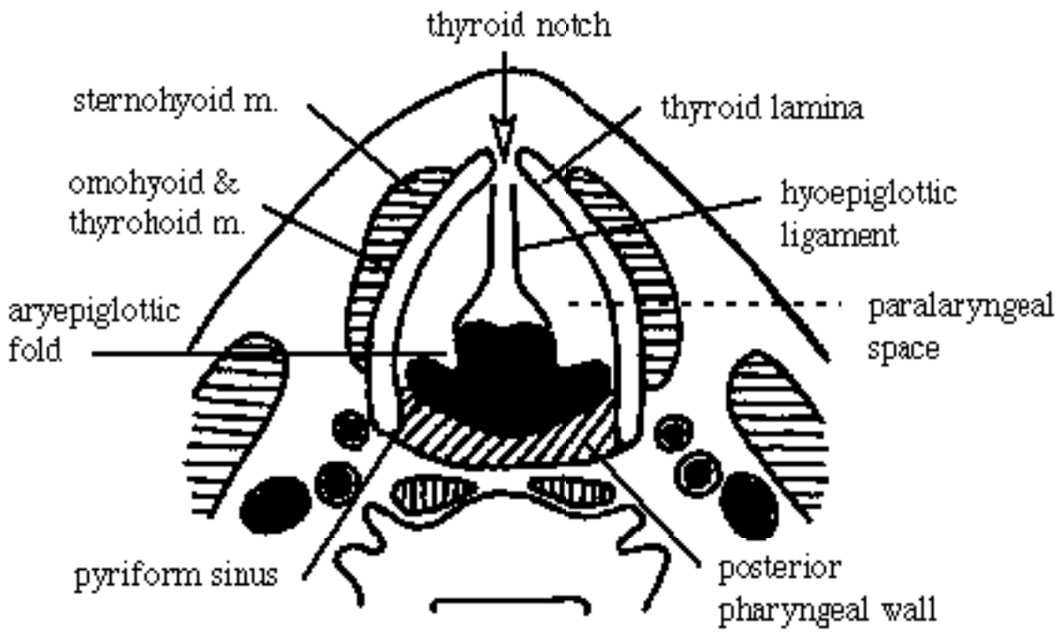
during phonation and during quiet breathing

A.SUPRAGLOTTISextends from tongue base + valleculae to laryngeal ventricle 1.Vestibule=airspace within supraglottic larynx2.Epiglottis=leaf-shaped cartilage that functions as a lid to endolarynx(a)petiole = stem of epiglottis(b)thyroepiglottic ligament = connects petiole to thyroid cartilage inferiorly(c)hyoepiglottic ligament = connects epiglottis to hyoid bone anteriorly, covered by a mucosal fold between the valleculae (glossoepiglottic fold)(d)"free margin" = superior portion of epiglottis3.False vocal cords=ventricular folds = inferior continuation of aryepiglottic folds = mucosal surface of ventricular ligaments; forming superior border of laryngeal ventricle4.Arytenoid cartilages5.Aryepiglottic folds=mucosal reflections between cephalad portion(= arytenoid processes) of arytenoid cartilage + inferolateral margin of epiglottis√ soft-tissue folds forming border between lateral pyriform sinuses + central laryngeal lumen 6.Laryngeal ventricle=fusiform fossa bounded by crescentic edge of false cords superiorly + straight margin of true cords inferiorly√ generally not visible on axial scans7.Preepiglottic space√ low-density tissue between anterior margin of epiglottis + thyroid cartilage8.Paralaryngeal space√ low-density tissue between true + false cords and thyroid cartilage√ continuous with preepiglottic

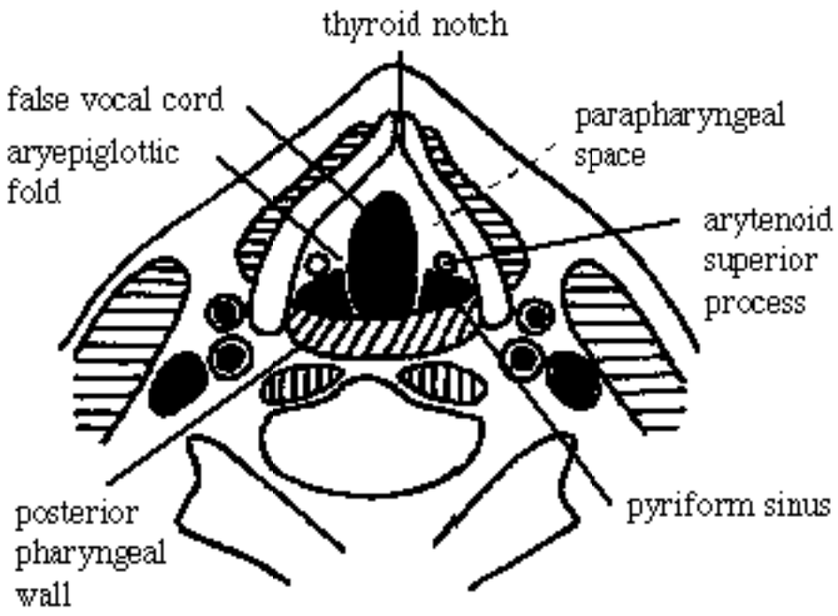


space anteriorly + aryepiglottic folds superiorly

High Supraglottic Level

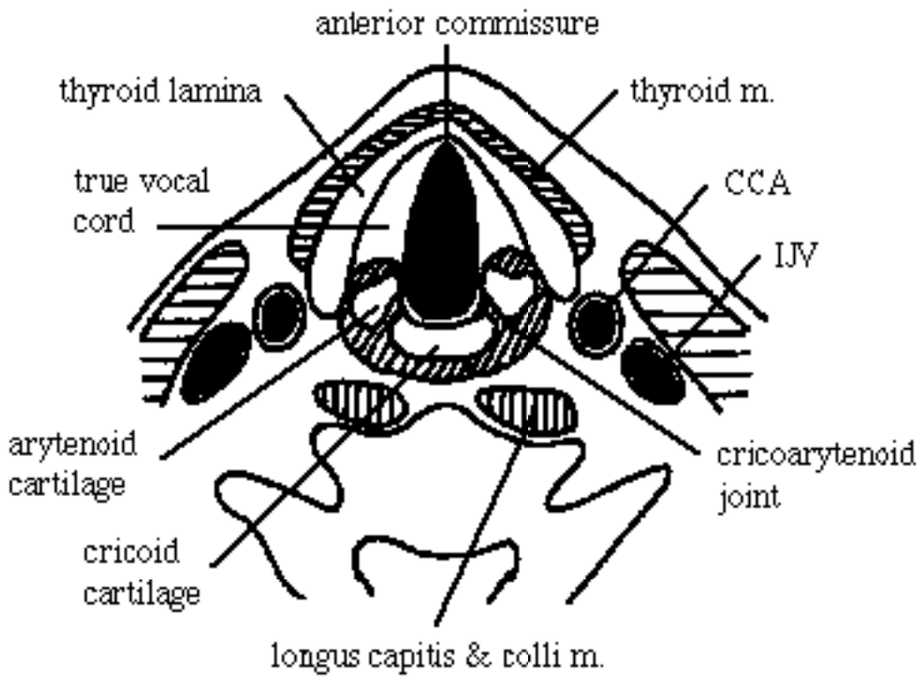


Mid Supraglottic Level



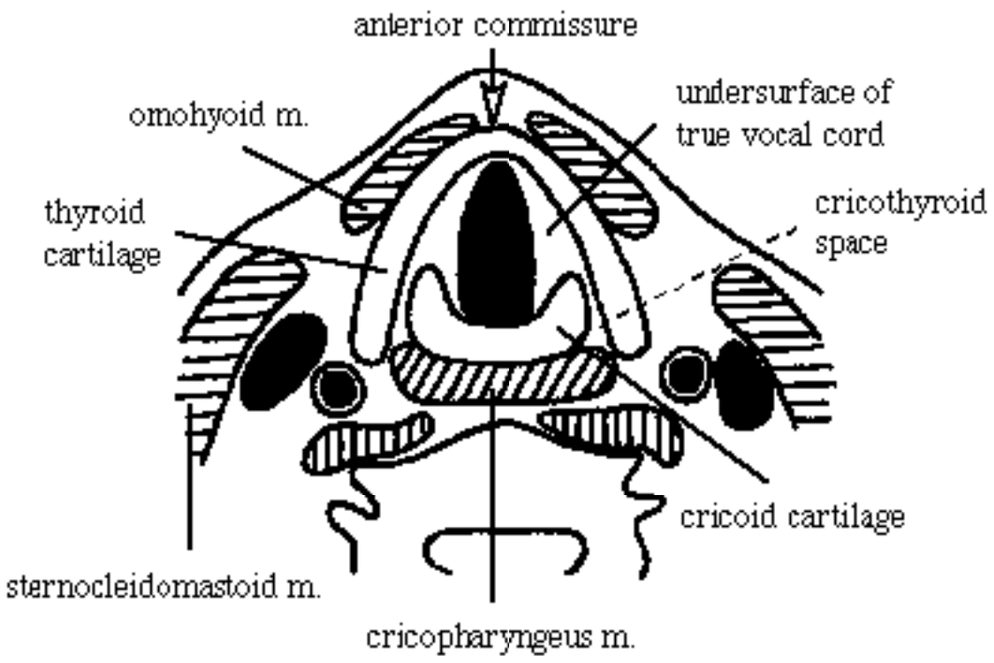
Low Supraglottic Level

B.GLOTTIS1.True vocal cords=extend from vocal process of arytenoid cartilage to anterior commissure√ vocal cords adduct during phonation of "E" / breath holding2.Anterior commissure=midline laryngeal mucosa covering anterior portions of the true vocal cords where they abut the laryngeal surface of the thyroid cartilage √ <1 mm soft tissue behind thyroid cartilage (during abduction of vocal cords with quiet breathing)3.Posterior commissure=midline laryngeal mucosal surface between



attachment of true vocal cords to the arytenoid cartilages
Glottic Level

C.SUBGLOTTISextends from undersurface of true vocal cords to inferior surface of cricoid cartilage 1.Conus elasticus=fibroelastic membrane extending from cricoid cartilage to medial margin of true vocal cords + forming lateral wall of subglottis



Undersurface of True Cord

Notes:

Pharyngeal mucosal space adenoids, faucial + lingual tonsils superior + middle constrictor muscles salpingopharyngeal muscle levator palatini muscle torus tubarius

Notes:

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Parapharyngeal space fat internal maxillary artery ascending pharyngeal artery pharyngeal venous plexus branches of cranial nerve V_3

Notes:

Retropharyngeal space fat medial + lateral retropharyngeal nodes

Notes:

Prevertebral space prevertebral muscles scalene muscles vertebral artery + vein brachial plexus phrenic nerve

Notes:

Carotid space Carotid fascia extends from skull base to aortic arch (a)below hyoid bone:common carotid artery internal jugular vein cranial nerve X (vagus nerve) (b)at level of nasopharynx:internal carotid artery internal jugular vein cranial nerves IX - XII

Notes:

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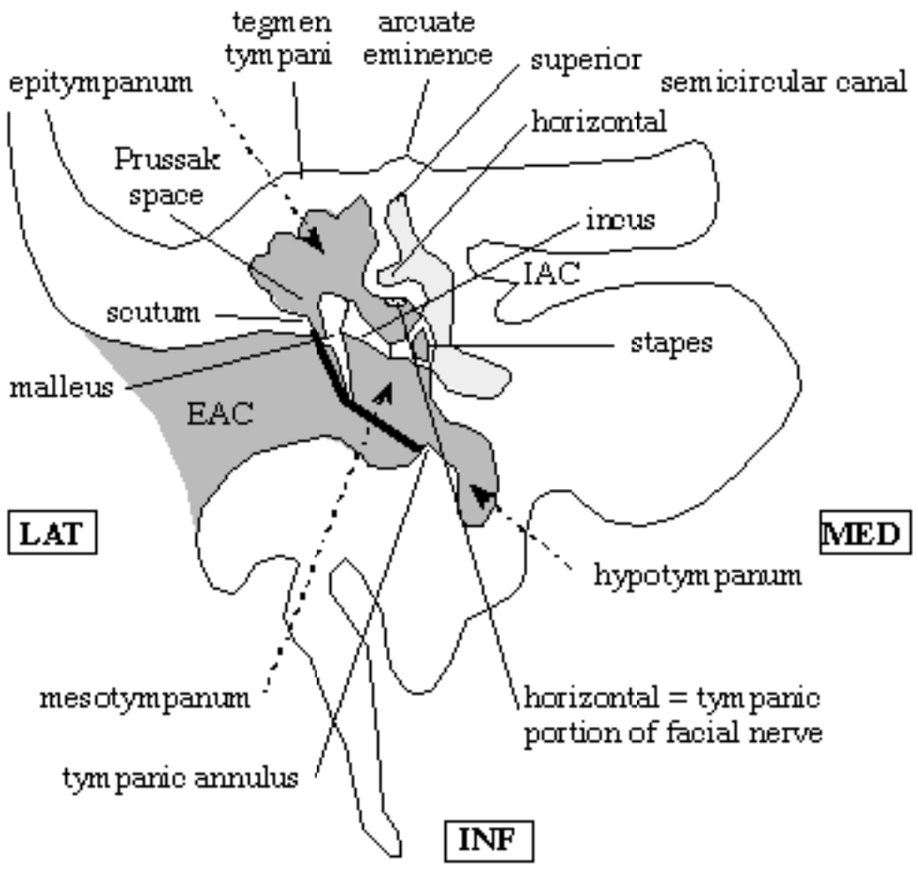
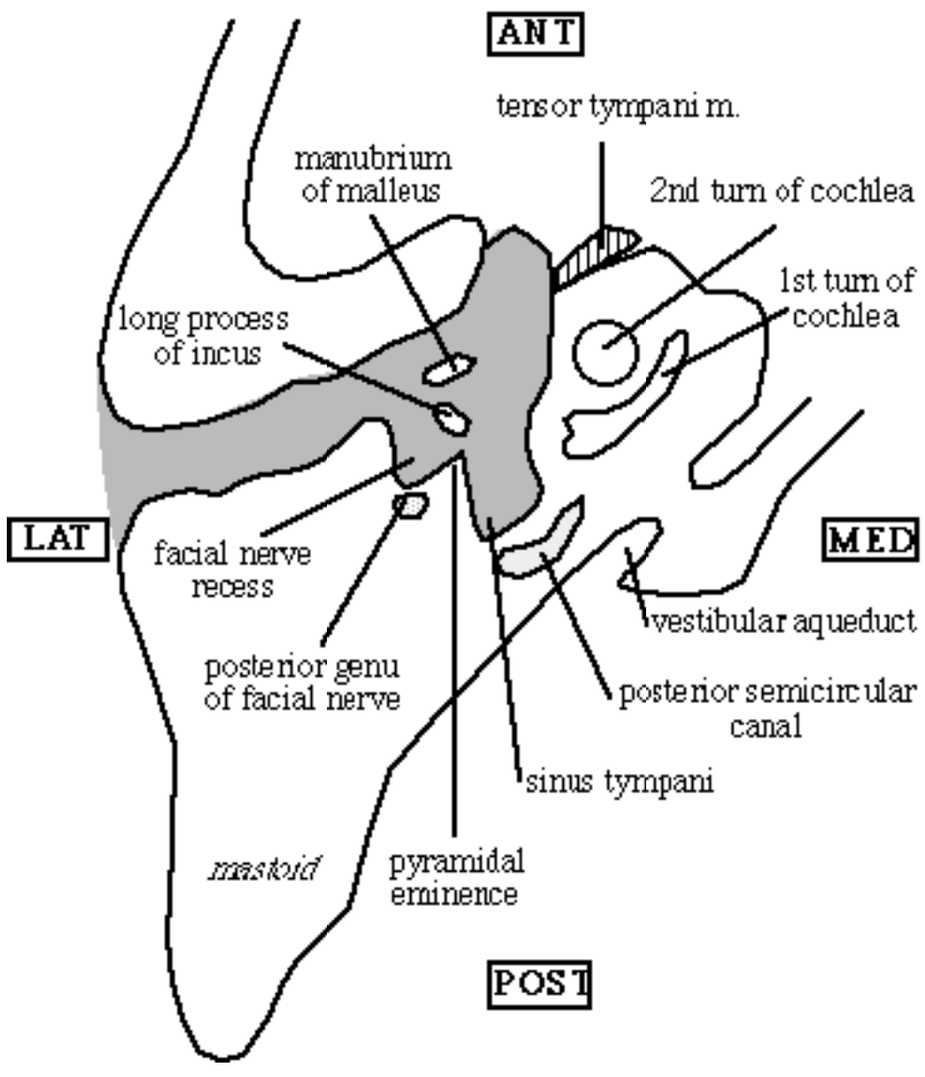
Parotid space parotid gland intraparotid lymph nodes external carotid + internal maxillary arteries retromandibular vein facial nerve

Notes:

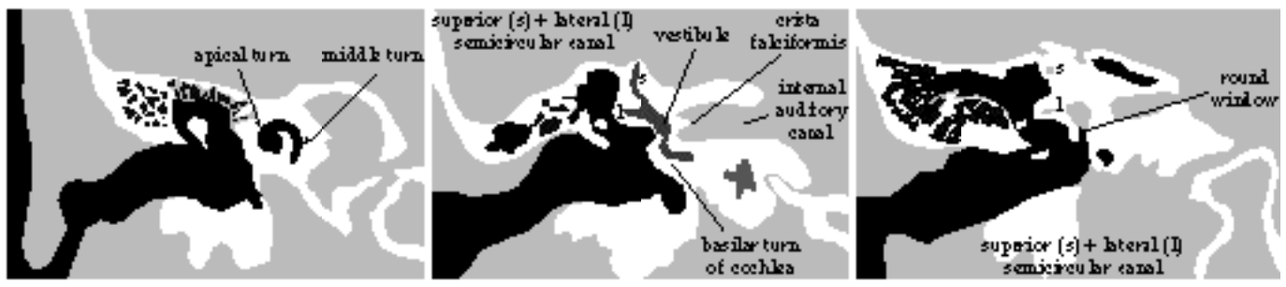


Temporal bone

Coronal Tomogram of Temporal Bone

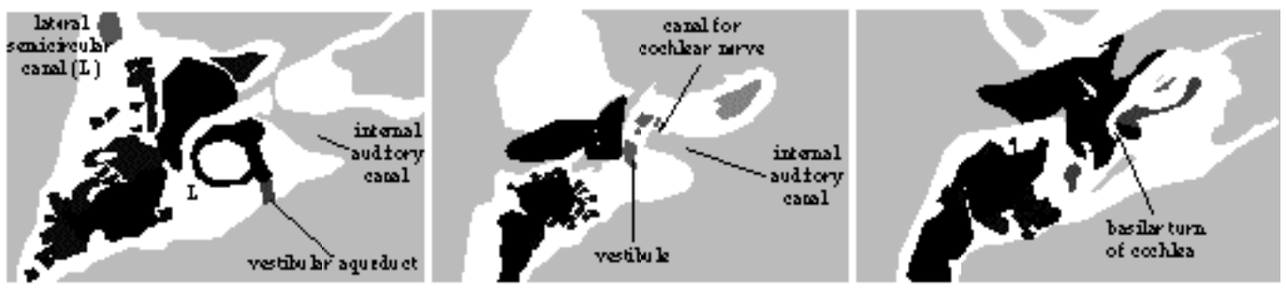


Axial Tomogram of Temporal Bone



Most anterior scan through the cochlea | Scan at vestibular level | Most posterior scan through round window

Coronal Scan of Normal Right Ear



Most superior scan through lateral semicircular canal | Scan through the vestibular level | Most inferior scan through the basilar turn of cochlea

Axial Scan of Normal Right Ear

A.SQUAMOUS PORTION=lateral wall of middle cranial fossa + floor of temporal fossaB.MASTOID PORTION1.Mastoid antrum2.Aditus ad antrumconnects epitympanum (= attic) of middle ear cavity to mastoid antrum 3.Körner septum=small bony projection extending inferiorly from roof of mastoid antrum as part of petrosquamosal suture between lateral + medial mastoid air cellsC.PETROUS PORTION = inner ear1.Tegmen tympani=roof of tympanic cavity2.Arcuate eminence=prominence of bone over superior semicircular canal3.Internal auditory canal (IAC)(a)Porus acusticus internus=opening of internal auditory canal(b)Modiolus=entrance to cochlea(c)Crista falciformis=horizontal bony septum in IAC4.Vestibular aqueduct=transmits endolymphatic duct5.Cochlear aqueduct=transmits perilymphatic duct6.Petrous apex=separated from clivus by petro-occipital fissure + foramen lacerumD.TYMPANIC PORTION1.External auditory canal (EAC)medial border formed by tympanic membrane, which attaches superiorly at scutum + inferiorly at tympanic annulus E.STYLOID PORTION

Notes:

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MIDDLE EAR

Borders: -anterior wall = carotid wall-posterior wall = mastoid wall including(a)facial nerve recess for descending facial nerve(b)pyramidal eminence for stapedius muscle(c)sinus tympani (clinically blind spot)-superior wall= tegmen tympani-inferior wall= jugular wall-lateral wall= tympanic membrane-medial wall= labyrinthine wallA.EPITYMPANUM=tympanic cavity above the line drawn between the inferior tip of scutum + tympanic portion of facial nerveContents:malleus head, body + short process of incus, Prussak space (= area between incus + lateral wall of epitympanum)B.MESOTYMPANUM=tympanic cavity between inferior tip of scutum + line drawn parallel to inferior aspect of bony EACContents:manubrium of malleus, long process of incus, stapes, tensor tympani muscle (innervated by V_3), stapedius muscle (innervated by VII)C.HYPOTYMPANUM=shallow trough in floor of middle ear

Notes:

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INNER EAR
1.Cochlea2 1/2 turns, basal first turn opens into round window posteriorly, encircles central bony axis of modiolus 2.Vestibule=largest part of membranous labyrinth with subunits of utricle + saccule (not separately visualized); separated from middle ear by oval window3.Semicircular canals-superior semicircular canal forms convexity of arcuate eminence-posterior semicircular canal points posteriorly along line of petrous ridge-lateral / horizontal semicircular canal juts into epitympanum4.Cochlear aqueductcontains 8 mm long perilymphatic duct, extends from basal turn of cochlea to lateral border of jugular foramen paralleling IAC Function:regulates CSF + perilymphatic fluid pressure5.Vestibular aqueductencompasses endolymphatic duct, extends from vestibule to endolymphatic sac Function:equilibration of endolymphatic fluid pressure

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FACIAL NERVE
Segments: (a)intracranial segment=from brainstem to porus acusticus internus(b)internal auditory canal=in anterosuperior portion of IAC(c)labyrinthine segment=short segment curling anteriorly over top of cochlea; terminates in anterior genu (geniculate ganglion)(d)tympanic segment=segment from anterior to posterior genu just underneath lateral semicircular canal(e)mastoid segment=from posterior genu to stylomastoid foramen(f)parotid segment=extracranial segment between superficial + deep lobes of parotid glandFunction: 1.Lacrimation (via greater superficial petrosal nerve)2.Stapedius reflex: sound damping3.Taste of anterior 2/3 of tongue (via chorda tympani nerve to lingual nerve)4.Facial expression (platysma)5.Secretion of lacrimal + submandibular + sublingual glands (via nervus intermedius)

Notes:

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THYROID HORMONES
free hormone:T_4(0.03%)T_3(0.4%)Thyroxin-binding globulin (TBG):binds T_4(70%)and T_3(38%)Thyroxin-binding prealbumin (TBPA):binds T_4(10%)and T_3(27%)Albumin:binds T_4(20%)and T_3(35%) A.ELEVATION OF TBG1.Pregnancy2.Estrogen administration3.Genetic trait B.REDUCTION IN TBG1.Androgens2.Anabolic steroids3.Glucocorticoids4.Nephrotic syndrome5.Chronic hepatic disease C.INHIBITION OF T_4 BINDING TO TBG: salicylates

Notes:

PARATHYROID GLANDS

A.SUPERIOR PARATHYROID GLANDSEmbryology:derived from 4th pharyngeal pouches, descending together with thyroid gland in close relationship to its posterolateral lobesLocation:superior dorsal surface of thyroid gland / intrathyroidal B.INFERIOR PARATHYROID GLANDSEmbryology:derived from 3rd pharyngeal pouches migrating caudally with thymusLocation:anywhere near / in thyroid, carotid bifurcation, lower neck, mediastinumC.SUPERNUMERARY PARATHYROID GLANDS5th / 6th gland may occupy an ectopic site◊Up to 12 parathyroids may be present! *Embryology:*parathyroid glands develop by 6 weeks GA + migrate into neck at 8 weeks Size:6 x 4 x 1 mm = 25-40 mg Surgical success rates for finding parathyroid glands: -95% for initial cervical exploration-60% for repeat surgical explorationCause for failure:overlooking an adenoma, multiple abnormal glands, diffuse hyperplasiaLocalization technique: US (75% sensitivity), thallium-technetium subtraction scintigraphy, MR (88% sensitivity)

DUPLEX IDENTIFICATION OF CAROTID ARTERIES

Notes:

DUPLEX IDENTIFICATION OF CAROTID ARTERIES

Criteria	Duplex Identification External Carotid Artery	Internal Carotid Artery
SIZE	usually smaller than ICA	usually larger than ECA
LOCATION	oriented medially + anteriorly toward face	oriented laterally + posteriorly toward mastoid process
BRANCHES	gives off arterial branches (superior thyroidal a. as 1st branch)	NO arterial branches
WAVE FORM	high-resistance flow pattern supplying capillary beds in skin + muscle √ forward systolic component √ early diastolic flow reversal occasionally followed by another forward component √ little / no flow in late diastole	low-resistance flow pattern supplying capillary bed in brain √ high-velocity forward systolic component √ sustained strong forward flow in diastole √ stagnant eddy with flow reversal opposite to flow divider in carotid bulb
MANEUVER	oscillations on temporal tap maneuver	

Notes:

ADENOID CYSTIC CARCINOMA
=CYLINDROMA*Incidence:*4-15% of all salivary gland tumors*Histo:*(a) tubular (b) cribriform (c) solid*Age:*3rd-9th decade; maximum between 40 and 70 yearsLocation: @Minor salivary glands (most common; 25-31% of malignant neoplasms in minor salivary glands) ● nasal obstruction + swelling@Submandibular gland (15% of tumors in this gland)@Parotid gland (2-6% of tumors in this gland; arises from peripheral parotid ducts with propensity for perineural spread along facial nerve) ● hard mass + facial nerve pain / paralysis√ infiltrating parotid massMR: √ hypo- to hyperintense (high signal corresponds to low cellularity) on T2WIMetastases to:lung, cervical lymph nodes, bone, liver*Prognosis:*slow relentless malignant course with repeat recurrences; the greater the cellularity, the worse the prognosis (requires entire tumor); 60-69% 5-year survival rate; 40% 10-year survival rate*Rx:*repeat surgical excision + radiation therapy

Notes:

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APICAL PETROSITIS
=PETROUS APICITISchronic > acute apicitis *Etiology:*spread from middle ear + mastoid infection; requires presence of air cells in petrous apices (which is found in 30% of population)*Organism:*Pseudomonas, enterococcus ● **Gradenigo syndrome** = otorrhea (otitis media) + retro-orbital pain (trigeminal pain) + 6th nerve palsy√ air cell opacification (fluid in ipsilateral middle ear + mastoid)√ bone destruction (osteomyelitis)MR: √ enhancing mass about petrous tip*Cx:*epidural abscess;cranial nerve palsy (abducens, trigeminal, vagus) *Mortality:*up to 20% (prior to antibiotic era)*Rx:*intravenous antibiotics, myringotomy, surgery

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Notes:

BENIGN MIXED TUMOR OF PAROTIS
=PLEOMORPHIC ADENOMA*Incidence:*80% of all benign parotid tumors*Histo:*mixture of epithelial + myoepithelial cells*Age:*usually >50 years ● slow-growing lump in cheek √ round / oval / lobulated sharply marginated mass√ rarely dystrophic calcifications√ variable contrast enhancementCT: √ low-density center if large (mucoid matrix)MR: √ hyperintense mass on T2WI√ hyperintense areas in center (mucoid matrix)

Notes:

CAROTID ARTERY DISSECTION
=hematoma within media splitting off the vessel wall and causing a false lumen within media
Etiology: A.SPONTANEOUS CAROTID DISSECTION(1)nonrecalled minor / trivial trauma(2)primary arterial disease: Marfan syndrome (fibromuscular dysplasia in 15%), cystic medial necrosis *Associated with:*hypertension (36%), smoking (47%), migraine (11%)B.TRAUMATIC CAROTID DISSECTIONblunt / penetrating trauma (automobile accident, boxing, accidental hanging, diagnostic carotid compression, manipulative therapy) *Associated with:*fracture through carotid canal *Incidence:*2% of strokes in persons aged 40-60 years*Age:*18-76 years (66% between 35 and 50 years) ● unilateral anterior headache (86%), neck pain (25%) ● TIA / stroke (58%), amaurosis fugax (12%) ● oculosympathetic paresis = Horner syndrome (52%) ● bruit (48%)Location:cervical ICA usually at level of C1-2 (60%), vertebral artery (20%), both ICA + vertebral artery (10%); multiple simultaneous dissections (33%); bilateral carotid dissections (15%), bilateral vertebral dissections (5%)Site:(a)Subintimal dissection = close to intima(b)Subadventitial dissection = close to adventitiaUS (50% accuracy) Angiography: √ string sign = elongated tapered irregular luminal stenosis extending to base of skull (76%)√ abrupt luminal reconstitution at level of bony carotid canal (42%)√ fingerlike / saccular aneurysm (40%), often in upper cervical / subcranial region√ intimal flap (29%), sometimes creating double-barrel lumen√ slow ICA-MCA flow√ tapered "flamelike" / "radish taillike" occlusion (17%), often distal to carotid bulbMR: √ pseudoenlargement of external diameter of artery (= intramural hematoma) *Cx:*(1)Thromboemboli due to stenosis(2)Subarachnoid hemorrhage (with intracranial location)(3)Secondary aneurysm*Prognosis:*complete / excellent recovery (8%)*Rx:*best therapy not clear; anticoagulants

Notes:

CAROTID ARTERY STENOSIS

High-grade ICA stenosis is associated with increased risk for TIA, stroke, carotid occlusion, embolism arising from thrombi forming at site of narrowing Increased risk for stroke: (a)significant ICA stenosis (compromised blood flow)Reduction of blood flow occurs at 50-60% diameter stenosis / 75% area stenosis◊2% risk of stroke with nonsignificant stenosis◊16% incidence of stroke with significant stenosis◊2% incidence of subsequent stroke following endarterectomy(b)intraplaque hemorrhage (embolic stroke)*Histo:* arteriosclerosis = generic term for all structural changes resulting in hardening of the arterial wall 1.Diffuse intimal thickening=growth of intima through migration of medial smooth muscle cells into subendothelial space through fenestrations in internal elastic lamella associated with increasing amounts of collagen, elastic fibers, glycosaminoglycans*Age:*beginning at birth slowly progressing to adult life2.Atherosclerosis=intimal pool of necrotic, proteinaceous + fatty substances within hardened arterial wallLocation:large + medium-sized elastic and muscular arteries(a)fatty streak = superficial yellow-gray flat intimal lesion characterized by focal accumulation of subendothelial smooth muscle cells + lipid deposits(b)fibrous plaque = whitish protruding lesion consisting of central core of lipid + cell debris surrounded by smooth muscle cells, collagen, elastic fibers, proteoglycans; a fibrous cap separates the lipid core (= atheroma) from the vessel lumen(c)complicated lesion = fibrous plaque with degenerative changes such as calcification, plaque hemorrhage, intimal ulceration / rupture, mural thrombosis*Plaque hemorrhage* from thin-walled blood vessels in vascularized plaque may cause ulceration, thrombosis + embolism, and luminal narrowing◊in 93% of symptomatic patients◊in 27% of asymptomatic patients*Plaque ulceration* exposes thrombogenic subendothelial collagen + lipid-rich material◊frequent in plaques occupying >85% of lumen◊12.5% stroke incidence per year3.Mönckeberg sclerosis = medial calcification4.Hypertensive arteriosclerosis

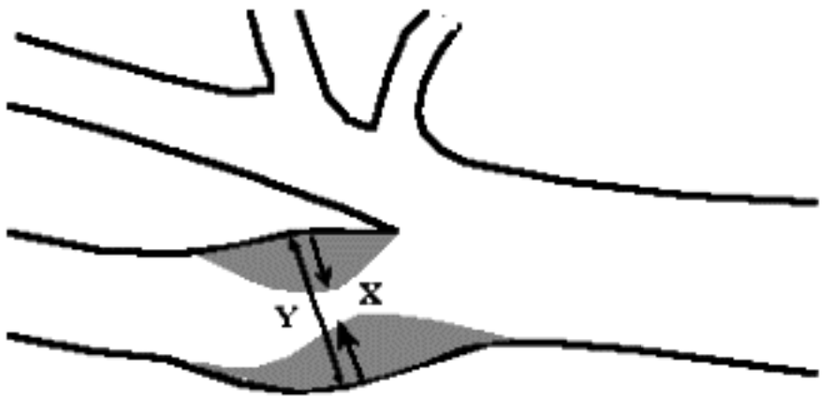
Predilection sites of arterial stenosis: Incidence of lesions StenosisOcclusion Right ICA origin33.8%8.6%Left ICA origin34.1%8.7%Right vertebral artery origin18.4%4.8%Left vertebral artery origin22.3%2.2%Right carotid siphon6.7%9.0%Left carotid siphon6.6%9.2%Basilar artery7.7%0.8%Right MCA3.5%2.2%Left MCA4.1%2.1%

Course Of Cartid Artery Stenosis: 1.Stable stenosis (68%)2.Progressive stenosis to >50% diameter reduction (25%) Angiography: @Extracranial√ smooth asymmetrical excrescence encroaching upon vessel lumen√ crater / niche = ulceration√ mound within base of crater = mural thrombus√ Holman carotid slim sign = diffuse narrowing of entire ICA distal to high-grade stenosis due to decrease in perfusion pressure√ occlusion of ICA @Intracranial√ carotid siphon stenosis√ retrograde flow in ophthalmic artery filled from ECA√ small vessel occlusion√ focal areas of slow flow√ early draining vein = reactive hyperemia = "luxury perfusion" due to shunting between arterioles + venules surrounding an area of ischemia√ ICA-MCA slow flow = delayed arrival + washout of ICA-MCA distribution in comparison to ECA *Carotid endarterectomy:* Benefit:17% reduction of ipsilateral stroke at 2 years in patients with >70% carotid stenosis (NASCET = North American Symptomatic Carotid Endarterectomy Trial)Risk:1% mortality; 2% risk of intraoperative neurologic deficit

Carotid Duplex Ultrasound Carotid Plaque Errors In Duplex Ultrasound

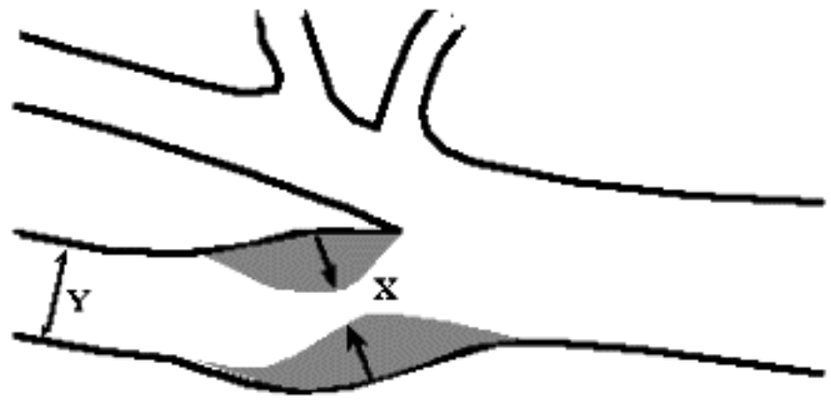
Notes:

Carotid Duplex Ultrasound *Indications for carotid duplex US:* (1)Screening for suspected extracranial carotid disease(a)high-grade flow-limiting stenosis(b)low-grade stenosis with hemorrhage(2)Nonhemispheric neurologic symptomatology(3)History of transient ischemic attack / stroke(4)Asymptomatic carotid bruit(5)Retinal cholesterol embolus(6)Preoperative evaluation before major cardiovascular surgery(7)Intraoperative monitoring of vascular patency during endarterectomy(8)Sequential evaluation after endarterectomy(9)Monitoring of known plaque during medical treatment
Grading Of Carotid Stenosis =severity of stenosis is primarily graded as a ratio of lumen diameter narrowing NOT reduction in cross sectional area*Limitations:* 1.Calcifications >1 cm in length◊A jet associated with an >70% stenosis usually travels at least 1 cm downstream!2.Contralateral high grade stenosis=ipsilateral ICA functions as collateral with increased blood flow velocities◊Use velocity ratios to compensate for this effect!*Accuracy of duplex scans:* (in comparison to arteriography for ICA lesions) 91-94% sensitivity, 85-99% specificity for >50% ICA diameter stenosis Incorporating B-mode and Doppler spectrum analysis A.NO LESION√ peak systolic velocity (PSV) < 125 cm/sec√ clear window under systole√ no spectral broadening√ no evidence of plaqueB.MINIMAL DISEASE=0-15% diameter reduction√ PSV < 125 cm/sec√ clear window under systole√ minimal spectral broadening in deceleration phase of systole√ minimal plaqueC.MODERATE DISEASE=16-49% diameter reduction√ peak systole <125 cm/sec√ no window under systole√ poststenotic spectral broadening throughout systole◊End-diastolic velocity (EDV) remains normal in <50% diameter reduction!√ moderate plaqueD.SEVERE DISEASE = HEMODYNAMICALLY SIGNIFICANT LESION(a)50-59% stenosis√ PSV 120-130 cm/sec√ EDV 30-40 cm/sec(b)60-79% stenosis√ PSV of 131-250 cm/sec√ EDV of 40-100 cm/sec(c)≥60 % stenosis√ end diastolic velocity of >80 cm/sec(d)50-79% diameter reduction√ peak velocity ratio of ICA/CCA >1.5√ peak systole >125 cm/s√ marked poststenotic spectral broadening throughout cardiac cycle(e)**>70% stenosis** (benefit of endarterectomy documented in NASCET study)√ peak systole >230 cm/s√ end diastole >100 cm/sec√ peak velocity ratio of ICA/CCA >4.0√ peak systolic velocity ICA ÷ end diastolic velocity CCA >15(f)80-99% diameter reduction√ PSV of >250 cm/sec√ EDV of >100 cm/sec√ no window under systole√ poststenotic spectral broadening throughout systole√ "string sign" on color Doppler with slow-flow sensitivity setting E.OCCLUDED VESSEL√ no signal in ICA on longitudinal / transverse images (color sensitivity + velocity scale must be set low enough to clearly discern flow signals within internal jugular vein)√ absence of diastolic flow in CCA (high impedance flow)√ diastolic flow reversal in CCA√ increased diastolic flow in ECA (if ECA assumes the role of primary supplier of blood to brain)√ increase in peak systolic velocities in contralateral ICA (due to collateral flow)*Limitations:* poor visualization due to calcification, tortuosity, increased depth of artery, "high" bifurcation **Common Carotid Waveform Analysis** A.DISTAL OBSTRUCTION√ high-pulsatility waveform (pulsatility changes occur only with >80% stenosis)√ reduced amplitudeB.PROXIMAL OBSTRUCTION√ low-amplitude damped waveform **Hemodynamic Variations of Carotid Artery Stenosis** A.MORPHOLOGY OF STENOSIS1.Degree of stenosis: velocities increase up to a luminal diameter of 1.0-1.5 mm2.Length of stenosis: peak velocities decrease with length of stenosis√ use the same angle + steering direction when following a patient for disease progressionB.PHYSIOLOGIC VARIABILITY◊A range of velocities may be encountered with a given degree of stenosis!◊ICA/CCA ratio obviates effects of physiologic variability!◊Compare left with right waveforms to avoid errors!◊Measure volume flow (more sensitive because of contralateral compensatory flow increase)*Cause:* 1.Cardiac output2.Pulse rate3.Flow velocity: increased with obstruction in collateral vessels, decreased with proximal obstruction in same vessel4.Normal helical nature of blood flow with many different velocity vectors + nonaxial blood flow not detectable by color Duplex imaging5.Peripheral resistance6.Arterial compliance7.Hypertension8.Blood viscosity



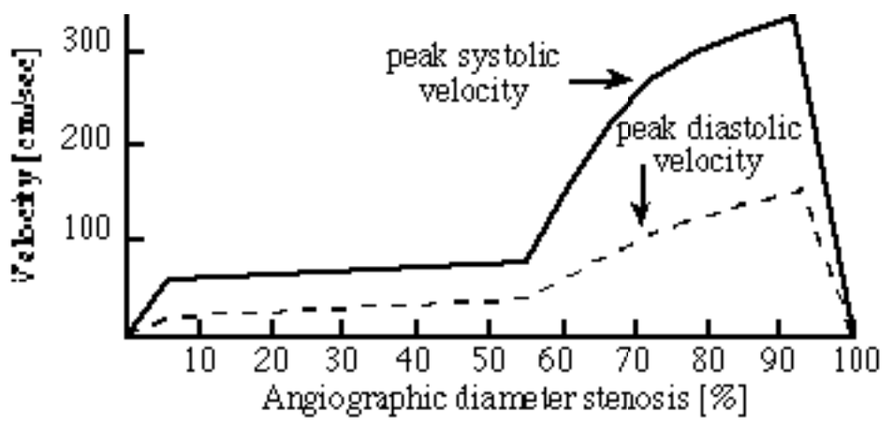
% STENOSIS (ECST) = (Y–X) / Y · 100

ECST = European Carotid Surgery Trial



% STENOSIS (NASCET) = (Y–X) / Y · 100

NASCET = North American Symptomatic Carotid Endarterectomy Trial



Doppler Parameters in Internal Carotid Artery Stenosis

Doppler Spectrum Analysis							
Diameter stenosis classification	(%)	ICA/CCA peak systolic ratio	ICA/CCA peak diastolic ratio	Peak systolic velocity (cm/sec)	kHz†	Peak diastolic velocity (cm/sec)	kHz†
normal – mild	0 – 40	<1.5	<2.6	<110 > 25	<3.5	<40	<1.5
moderate	41 – 59	<1.8	<2.6	>120	>3.5	<40	<1.5
severe	60 – 79	>1.8	>2.6	>130	>5.0	>40	>1.5
critical	80 – 99	>3.7	>5.5	>250	>8.0	>80 – 135	>4.5
† = based on 5MHz pulsed Doppler carrier frequency at 60° flow angle (Blackshear)							

	0 – 39	<1.8	<2.4	<110		<40	
	40 – 59	<1.8	<2.4	<130		<40	
	60 – 79	>1.8	>2.4	>130		>40	
	80 – 99	>3.7	>5.5	>250		>100	
(Bluth)							

	0 – 50	<2:1		<125		<40	
	50 – 75	>2:1		125 – 225		40 – 100	
	75 – 90	>3:1	>5:1	225 – 325		>100	
	>90	>4:1	>9:1	>325		>100	
	>95	resistive CCA	distortion	may be decreased		may be decreased	
(Gosink)							

	1 – 15						
	16 – 49			(<125)	<4.0		
	50 – 79			(≥125)	≥4.0	(<140)	<4.5
(Fell)	80 – 99					(≥140)	≥4.5
(Strandness)	occlusion			no flow detected			
† = based on 5MHz pulsed Doppler carrier frequency at 60° flow angle (University of Washington)							

Notes:

Carotid Plaque

FORMATION THEORY 1.Stagnant eddy that rotates at outer vessel margin (opposite to the flow divider in area of flow separation + low shear stress) leads to net influx of fluid into subendothelial tissue with progressive deposition of lipids + smooth muscle cell proliferation2.Increased likelihood of intraplaque hemorrhage (vascularization of plaque with fragile vessels derived from vasa vasorum / from lumen) + fissuring from a critical size on◊As the degree of stenosis increases, it is more likely that plaques become denser + more heterogeneous demonstrating an irregular surface!

PLAQUE DENSITY 1.Hypoechoic = low-echogenicity plaque=fibrofatty plaque / hemorrhage√ echogenicity less than sternocleidomastoid muscle√ flow void / flow disturbance on color Duplex2.Isoechoic plaque=smooth muscle cell proliferation / laminar thrombus√ echogenicity equal to sternocleidomastoid muscle + lower than adventitia3.Hyperechoic = moderately echogenic plaque=fibrous plaque√ echogenicity higher than sternocleidomastoid muscle + similar to adventitia4.Calcification = strongly echogenic plaque√ acoustic shadow impairs visualization of intima PLAQUE TEXTURE 1.Homogeneous plaque = stable plaque*Histo:*deposition of fatty streaks + fibrous tissue; rarely shows intraplaque hemorrhage / ulcerations*Prognosis:* ◊neurologic deficits develop in 4%◊ipsilateral infarction on CT in 12%◊ipsilateral symptoms develop in 22%◊progressive stenosis develops in 18%√ homogeneous uniform echo pattern with smooth surface (acoustic impedance similar to blood) 2.Heterogeneous plaque=unstable plaque = mixture of high, medium and low level echoes with smooth / irregular surface; may fissure / tear resulting in intraplaque hemorrhage / ulceration + thrombus formation (embolus / increasing stenosis)B-mode ultrasound has 90-94% sensitivity, 75-88% specificity, 90% accuracy for intraplaque hemorrhage *Histo:*lipid-laden macrophages, monocytes, leukocytes, necrotic debris, cholesterol crystals, calcifications*Prognosis:*◊neurologic deficits develop in 27%◊ipsilateral infarction on CT in 24%◊ipsilateral symptoms develop in 50%◊progressive stenosis develops in 77%√ anechoic areas within plaque (= hemorrhage / lipid deposition / focal plaque degeneration) √ heterogeneous complex echo pattern

PLAQUE SURFACE CHARACTERISTICS =US unreliable due to poor visualization of intima*Categories:*-smooth-mildly irregular-markedly irregular-ulcerated 1.Intimal thickening*Histo:*fatty streaks√ wavy / irregular line paralleling vessel wall extending >1 mm into vessel lumen2.Ulcerated plaque*Accuracy:*60% sensitive, 60-70% specific◊The presence of intraplaque hemorrhage is much more common than normally appreciated◊Neither arteriography nor US has proved reliable!√ isolated crater of >2 mm within surface of plaque demonstrated on transverse + longitudinal images√ reversed flow vortices extending into plaque crater demonstrated by color Doppler√ proximal + distal undercutting of plaque√ anechoic area within plaque extending to surface

Notes:

Errors In Duplex Ultrasound

1.Error in proper localization of stenosis (6%)*Cause:*ECA stenosis placed into ICA / carotid bifurcation or vice versa2.Mistaking patent ECA branches for carotidbifurcation (4%)*Cause:* complete occlusion of ICA not recognized√ disparity in position of bifurcation√ no difference in pulsatility waveform√ high-resistance waveform in CCA3.Interpreter error in estimating severity of stenosis (2.5%)usually overestimation, rarely underestimation√ absence of one / more components for diagnosis which are(a) significant elevation of peak velocity (b) poststenotic turbulence (c) extension of high velocity into diastole 4.Superimposition of ECA + ICA (2%)*Cause:*strict coronal orientation of ECA + ICA√ superimposition can be avoided by rotation of head to opposite side5.Severe stenosis mistaken for occlusionminimal flow not detectable; angiogram necessary with delayed images6.Weak signals misinterpreted as occlusion7.Normal / weak signals in severe stenosis*Cause:*severe stenosis causes a decrease in blood flow + peak velocity with return to normal velocity levels√ high resistivity in CCA8.Point of maximum frequency shift not identified*Cause:*extremely small lumen / short segment of stenosis√ unexplained (poststenotic) coarse turbulence√ ipsilateral ECA collateral flow√ abnormal CCA resistivity9.Stenosis obscured by plaque / strong Doppler shift in overlying vessel10.Inaccessible stenosis√ abnormal CCA resistivity√ abnormal oculoplethysmography 11.Unreliable velocity measurements(a)higher velocities: hypertension, severe bradycardia, obstructive contralateral carotid disease (b)lower velocities: arrhythmia, aortic valvular lesion, severe cardiomyopathy, proximal obstructive carotid lesion ("tandem lesion"), >95% ICA stenosis(c)aliasing = high velocities are displayed in reversed direction below zero baseline due to Doppler frequency exceeding half the pulse repetition frequency *Remedy:*shift zero baseline, increase pulse repetition frequency, increase Doppler angle, decrease transducer frequency, use continuous-wave Doppler probe Indirect Methods Of Evaluation 1.Oculoplethysmography (OPG)=measurement of ophthalmic artery pressure + pulse arrival time by air calibrated system*Contraindications:*glaucoma, retinal detachment, recent eye surgery / trauma, lens implants 2.Periorbital bidirectional Doppler=insonation of frontal + supraorbital arteries to assess flow direction around orbit and to detect crossover flow through the circle of Willis (through contra- and ipsilateral compression) 3.Transcranial Doppler=insonation to establish flow direction in basal cerebral arteries through temporal bone (MCA, ACA, PCA, terminal portion of ICA), foramen magnum (both vertebral arteries, basilar artery), orbit (carotid siphon)√Nondiagnostic in up to 35%!

Notes:

CHOANAL ATRESIA
*Etiology:*failure of perforation of oronasal membrane which normally perforates by 7th week EGA◊Associated with other anomalies in 50%!A.BONY SEPTATION (85%)B.MEMBRANOUS SEPTATION (15%)

Notes:

CHOLESTEATOMA
=KERATOMA = epithelium-lined sac filled with keratin debris leading to bone destruction by pressure + demineralizing enzymes

Primary Cholesteatoma Secondary Cholesteatoma

Notes:

Primary Cholesteatoma =CONGENITAL CHOLESTEATOMA = EPIDERMOID CYST (2%)=derived from aberrant embryonic ectodermal rests in temporal bone (commonly petrous apex) / epidural space / meninges ● conductive hearing loss in child with NO history of middle ear inflammatory disease ● cholesteatoma seen through intact tympanic membrane*Associated with:* EAC dysplasiaLocation: (a)epitympanum(b)petrous pyramid: internal auditory canal first involved(c)meninges: scooped out appearance of petrous ridge(d)cerebellopontine angle: erosion of porus, shortening of posterior canal wall(e)jugular fossa: erosion of posteroinferior aspect of petrous pyramid

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Notes:

Secondary Cholesteatoma =INFLAMMATORY CHOLESTEATOMA=ACQUIRED EPIDERMOID (98%)*Cause:* ingrowth of squamous cell epithelium of EAC through tympanic membrane (= eardrum) secondary to (a)repeated episodes of ear inflammation with invagination of posterosuperior retraction pocket(b)marginal perforation of eardrum*Age:*usually >40 years ● whitish pearly mass behind intact tympanic membrane (invasion of middle ear cavity and mastoid) diagnosed otoscopically in 95% ● facial paralysis (compression of nerve VII at geniculate ganglion) ● conductive hearing loss (compromise of nerve VIII in internal auditory canal / involvement of cochlea or labyrinth) ● severe vertigo (labyrinthine fistula)*Types:* 1.**Pars flaccida cholesteatoma** = Primary acquired cholesteatoma = Attic cholesteatoma (most common)√ increasing width of attic√ initially destruction of lateral wall of attic, particularly the drum spur (scutum) with invasion of Prussak space√ extension posteriorly through aditus ad antrum into mastoid antrum√ destruction of Körner septum2.**Pars tensa cholesteatoma** = Secondary acquired cholesteatoma (less frequent)√ displacement of auditory ossicles√ erosion of ossicular chain: first affecting long process of incus √ nondependent homogeneous mass√ perforation of tympanic membrane posterosuperiorly (pars flaccida = Shrapnell membrane)√ poorly pneumatized mastoid (frequent association)√ erosion of tegmen tympani (with more extensive cholesteatoma) producing an extradural mass√ destruction of labyrinthine capsule (less common) involving the lateral semicircular canal first√ erosion of facial canalMRI: √ iso- / hypointense relative to cortex on T1WI√ no enhancement with Gd-DTPA (enhancement is related to granulation tissue) *Cx:*(1)Intratemporal: ossicular destruction, facial nerve paralysis (1%), labyrinthine fistula, automastoidectomy, complete hearing loss(2)Intracranial: meningitis, sigmoid sinus thrombosis, temporal lobe abscess, CSF rhinorrhea*DDx:*chronic otitis media, granulation tissue= cholesterol granuloma, brain herniation through tegmen defect, neoplasm (rhabdomyosarcoma, squamous cell carcinoma)

Notes:

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CHOLESTEROL GRANULOMA
=CHOLESTEROL CYST=acquired inflammatory lesion of petrous bone*Histo:*cholesterol crystals surrounded by foreign-body giant cells; embedded in fibrous connective tissue with varying proportions of hemosiderin-laden macrophages, chronic inflammatory cells and blood vessels; brownish fluid contains cholesterol crystals + blood (= "chocolate cyst") ● blue (vascular) tympanic membrane without pulsatile tinnitus√ ossicles remain intactCT:√ nonenhancing middle ear massMRI: √ hyperintense signal on T1WI + T2WI secondary to methemoglobin (DDx to cholesteatoma, which is isointense to brain on T1WI)

Notes:

CHRONIC RECURRENT SIALADENITIS

● painful periodic unilateral enlargement of parotid gland ● milky discharge may be expressedSialography: √ Stensen duct irregularly enlarged / sausage-shaped√ pruning of distal parotid ducts√ ± calculiCT: √ diffusely enlarged dense gland√ dilated Stensen duct ± calculi*Cx:*Mucocele

Notes:

COGAN SYNDROME

=AUTOIMMUNE INTERSTITIAL KERATITISMR:√ membranous labyrinthine enhancement

Notes:

CROUP
=ACUTE LARYNGOTRACHEOBRONCHITIS=ACUTE VIRAL SPASMODIC LARYNGITIS=lower respiratory tract infection*Organism:*parainfluenza, respiratory syncytial virus*Age:*>6 months of age, peak incidence 2-3 years ● history of viral lower respiratory infection ● hoarse cry + "brassy" cough ● inspiratory difficulty with stridor ● fever√ thickening of vocal cords√ NORMAL epiglottis + aryepiglottic folds√ "steeple sign" = subglottic "inverted V" = symmetrical funnel-shaped narrowing 1-1.5 cm below lower margins of pyriform sinuses on AP radiograph (loss of normal "shouldering" of air column caused by mucosal edema + external restriction by cricoid), accentuated on expiration, paradoxical inspiratory collapse, less pronounced during expiration√ narrow + indistinct subglottic trachea on lateral radiograph√ inspiratory ballooning of hypopharynx (nonspecific sign of any acute upper airway obstruction)√ distension of cervical trachea on expiration*Prognosis:*usually self-limiting

Notes:

CYSTIC HYGROMA
=CYSTIC LYMPHANGIOMA = most common form of lymphangioma=single / multiloculated fluid-filled cavities on either side of fetal neck + head (localized form) ± trunk (generalized form)*Cause:*congenital blockage of lymphatic drainage(= noncommunication of jugular lymphatic sac with jugular vein) *Incidence:*1:6,000 pregnancies*Age:*50-65% present at birth;up to 90% evident by age 2*Histo:*hugely dilated cystic lymphatic spaces *Associated with:* (a)chromosomal abnormalities in 60-80% (in particular when detected in 2nd trimester)(1)Turner syndrome (45 XO, mosaic) in 40-80%(2)Trisomies 13, 18, 21, 13q, 18p, 22(3)Noonan syndrome(4)Distichiasis-lymphedema syndrome(5)Familial pterygium colli(6)Roberts, Cumming, Cowchock syndrome(7)Achondrogenesis type II(8)Lethal pterygium syndrome(b)exposure to teratogens(1)Fetal alcohol syndrome(2)aminopterin(3)trimethadione*Types:* (1)Cystic hygroma with abnormal peripheral lymphatic system√ lymphangioma in posterior compartment of neck√ septations (indicate high probability for aneuploidy, development of hydrops, and perinatal death)(2)Diffuse lymphangiectasia√ lymphangioma of chest + extremities√ peripheral lymphedema + nonimmune hydrops(3)Isolated cystic hygroma(a)axillary lymph sac malformation √ lymphangioma restricted to axilla(b)jugular lymph sac malformation√ lymphangioma restricted to lateral neck(c)internal thoracic + paratracheal lymph sac malformation√ lymphangioma within mediastinum(d)combined lymph sac malformation(e)thoracic duct malformation√ thoracic duct cyst ● AF- / MS-AFP may be elevated ● ± dyspnea / dysphagia with encroachment upon trachea, pharynx, esophagus ● rapid increase in size (from infection / hemorrhage) Location:posterior neck (75%), mediastinum (3-10%, in 1/2 extension from neck), axilla (20%), chest wall (14%), face (10%), retroperitoneum, abdominal viscera, groin, scrotum, bones√ thin-walled fluid-filled structure with multiple septa + solid cyst wall components√ isolated nuchal cysts√ webbed neck (= pterygium colli) following later communication with jugular veins√ nonimmune hydrops (43%)√ progressive peripheral edema√ fetal ascites√ oligo- / polyhydramnios / normal amount of fluid√ bradycardiaMR: √ hyperintense on T2WI√ low to high signal intensity on T1WI (depending on protein content of fluid)√ ± fluid-fluid level (if hemorrhage present)*Cx:*(1)Compression of airways / esophagus(2)Slow growth / sudden enlargement (hemorrhage, inflammation)*Prognosis:* (1)Intrauterine demise (33%)(2)Mortality of 100% with hydrops(3)Spontaneous regression (10-15%)Favorable for localized lesions of anterior neck + axilla ◊Only 2-3% of fetuses with posterior cystic hygroma become healthy living children!*Rx:*surgical excision (difficult since mass does not follow tissue planes)*DDx:*twin sac of blighted ovum, cervical meningocele, encephalocele, cystic teratoma, nuchal edema, branchial cleft cyst, vascular malformation, lipoma, abscess **Pseudocystic Hygroma** = PSEUDOMEMBRANE =anechoic space bordered by specular reflection on posterior aspect of fetal neck during 1st trimester*Cause:*? developing integument√ NO prominent posterior bulge / internal septations

Notes:

EPIGLOTTITIS
= ACUTE BACTERIAL EPIGLOTTITIS = life-threatening infection with edema of epiglottis + aryepiglottic folds *Organism:*Haemophilus influenzae type B, Pneumococcus, Streptococcus group A*Age:*>3 years, peak incidence 6 years ● abrupt onset of respiratory distress with inspiratory stridor ● severe dysphagia*Location:*purely supraglottic lesion; associated subglottic edema in 25%Lateral radiograph should be taken in erect position only! (frontal view irrelevant) √ enlargement of epiglottis + thickening of aryepiglottic folds √ circumferential narrowing of subglottic portion of trachea during inspiration √ ballooning of hypopharynx + pyriform sinuses √ cervical kyphosis*Cx:*Mortal danger of suffocation secondary to hazard of complete airway closure; patient needs to be accompanied by physician experienced in endotracheal intubation

Notes:

EXTERNAL AUDITORY CANAL DYSPLASIA
*Incidence:*1:10,000 births; family history in 14%*Etiology:*(a) isolated (b) Trisomy 13, 18, 21 (c) Turner syndrome (d) Maternal rubella (e) Craniofacial dysostosis (f) Mandibulofacial dysostosisSPECTRUM 1.Stenosis of EAC2.Fibrous atresia of EAC3.Bony atresia (in position of tympanic membrane)4.Decreased pneumatization of mastoid (mastoid cells begin to form in 7th fetal month)5.Decreased size / absence of tympanic cavity6.Ossicular changes (rotation, fusion, absence)7.Ectopic facial nerve = anteriorly displaced vertical (mastoid) portion of facial nerve canal8.Decrease in number of cochlear turns / absence of cochlea9.Dilatation of lateral semicircular canal ● bilateral in 29%; M:F = 6:4 ● pinna deformity ● stenotic / absent auditory canal*Cx:*congenital cholesteatoma (infrequent)

Notes:

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FIBROMATOSIS COLLI

*Cause:*pressure necrosis with secondary fibrosis of sternocleidomastoid muscle from birth trauma ● history of difficult delivery (forceps) ● anterior neck mass during first 2 weeks of life, which may grow over 2-4 additional weeks ● Torticollistorticollis (14-20%)Location:lower 2/3 of sternocleidomastoid muscleUS: √ well-defined mass within sternocleidomastoid muscle√ hypo- over iso- to hyperechoic mass depending on duration of disorderCT: √ isoattenuating muscle enlargement*Prognosis:*gradual spontaneous resolution over 4-8 months with / without treatment*Rx:*(1)muscle stretching exercise (2) surgery in 10%*DDx:* (1)Neuroblastoma (heterogeneous solid mass with calcifications)(2)Rhabdomyosarcoma(3)Lymphoma (well-defined round /oval masses along cervical lymph node chain)(4)Cystic hygroma (anechoic region with septations)(5)Branchial cleft cyst(6)Hematoma

Notes:

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Longitudinal Fracture Of Temporal Bone (75%) =fracture parallel to the axis of petrous pyramid arising in squamosa of temporal bone through tegmen tympani, EAC (external auditory canal), middle ear, terminating in foramen lacerum ● bleeding from EAC (disruption of tympanic membrane) ● NO neurosensory hearing loss ● otorrhea (CSF leak with ruptured tympanic membrane; rare) ● conductive hearing loss (dislocation of auditory ossicles - most commonly incus as the least anchored ossicle) ● facial nerve palsy (10-20%) due to edema / fracture of facial canal near geniculate ganglion; frequent spontaneous recovery√ pneumocephalus√ herniation of temporal lobe√ incudostapedial joint dislocation (weakest joint)√ "ice cream" (malleus) has fallen off the "cone" (incus) on direct coronal CT scan√ fracture of "molar tooth" on direct sagittal CT scan√ mastoid air cells opaque / with air-fluid levelPlain film views:Stenvers / Owens projection

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Notes:

Transverse Fracture Of Temporal Bone (25%) =fracture perpendicular to axis of petrous pyramid originating in occipital bone extending anteriorly across the base of skull + across the petrous pyramid ● irreversible neurosensory hearing loss (fracture line across apex of IAC / labyrinthine capsule) ● persistent vertigo ● facial nerve palsy in 50% (injury in IAC); less frequent spontaneous recovery because of disruption of nerve fibers ● rhinorrhea (CSF leak with intact tympanic membrane) ● bleeding into middle earPlain film views:posteroanterior (transorbital) + Towne projection

Notes:

GLOMUS TUMOR
=CHEMODECTOMA = NONCHROMAFFIN PARAGANGLIOMA = GLOMERULOCYTOMA=slow-growing vascular lesion arising from glomus body*Origin:*tumor arising from nonchromaffin paraganglion cells of neuroectodermal origin; differs from adrenal medulla only in its nonchromaffin feature*Histo:*acidophil-epitheloid cells in contact with endothelial cells of a vessel; storage of catecholamines (usually nonfunctioning); histologically similar to pheochromocytoma*Age:*range of 6 months to 80 years; peak age in 5-6th decade; F:M = 4:1*Associated with:*pheochromocytomaLocation:anywhere in paraganglionic tissue between glomus jugulotympanicum and base of bladder: carotid body, skull base, temporal region, trachea, periaortic region, mandible, ciliary ganglion of the eye, retroperitoneal region, cervical vagus nerve, laryngeal branches of vagus nerveSynchronous multicentricity in 3-26%: (a) autosomal dominant in 25-35% (b) nonhereditary in <5%

Glomus Tympanicum Tumor Glomus Jugulare Tumor Glomus Vagale Tumor Carotid Body Tumor

Notes:

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Glomus Tympanicum Tumor Most common tumor in middle ear ● hearing loss, pulsatile tinnitus ● reddish purple mass behind tympanic membraneLocation:tympanic plexus on cochlear promontory of middle earCT (bone algorithm preferred): √ globular soft-tissue mass abutting promontory√ intense enhancement√ usually small at presentation (early involvement of ossicles)√ erosion + displacement of ossicles√ inferior wall of middle ear cavity intactAngio: √ difficult to visualize because of small size

Notes:

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Glomus Jugulare Tumor Most common tumor in jugular fossa with intracranial extension Glomus jugulotympanicum tumor = large glomus jugulare tumor growing into the middle ear *Origin:*adventitia of jugular vein ● tinnitus, hearing loss ● vascular tympanic membraneLocation:at dome of jugular bulb√ soft-tissue mass in jugular bulb region / hypotympanum / middle ear space√ intense enhancement√ destruction of posteroinferior petrous pyramid + corticojugular spine of jugular foramen√ destruction of ossicles (usually incus), otic capsule, posteromedial surface of petrous boneMR: √ "salt and pepper" appearance due to multiple small tumor vesselsAngio: (film entire neck for concurrent glomus tumors!) √ hypervascular mass with persistent homogeneous reticular stain√ invasion / occlusion of jugular bulb by thrombus / tumor√ supplied by tympanic branch of ascending pharyngeal artery, meningeal branch of occipital artery, posterior auricular artery via stylomastoid branch, internal carotid artery, internal maxillary artery√ arteriovenous shunting*Cx:*malignant transformation with metastases to regional lymph nodes (in 2-4%)

Notes:

Glomus Vagale Tumor *Origin:*near ganglion nodosum of vagus nerve at base of skull close to jugular foramenExtension:(a)downward into parapharyngeal space (2/3)(b)intracranially (dumbbell shape) ● slow growing + asymptomatic√ spherical / ovoid mass with sharp interfacing margins and homogeneous enhancement√ highly vascular mass + neovascularity + intense tumor blush*Cx:*malignant transformation with metastases in 15% to regional lymph nodes + lung (other paragangliomas in 10%)

Notes:

Carotid Body Tumor *Embryology:* derived from mesoderm of 3rd branchial arch + neural crest ectoderm cells, which differentiate into sympathogonia (= forerunner of paraganglionic cells) ◊Chemodectoma is misnomer (not derived from chemoreceptor cells)!*Histo:*nests of epithelioid cells ("Zellballen") with granular eosinophilic cytoplasm separated by trabeculated vascularized connective tissue◊chromaffin-positive granules(= catecholamines) may be present *Function of carotid body:* 5 x 3 x 2 mm carotid body regulates pulmonary ventilation through afferent input by way of glossopharyngeal nerve to the medullary reticular formation Stimulus:hypoxia > hypercapnia > acidosis Effect:increase in respiratory rate + tidal volume; increase in sympathetic tone (heart rate, blood pressure, vasoconstriction, elevated catecholamines) ● painless pulsatile firm neck mass below the angle of the jaw, laterally mobile but vertically fixedLocation:adventitia of carotid bifurcation; bilateral in 5% with sporadic occurrence, in 32% with autosomal dominant transmission√ enhancing oval mass with splaying of ICA + ECA*Cx:*malignant transformation in 6% with metastases to regional lymph nodes, brachial plexus, cerebellum, lung, bone, pancreas, thyroid, kidney, breast

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Notes:

Adenomatous Goiter =MULTINODULAR GOITERUS: (89% sensitivity, 84% specificity, 73% positive predictive value, 94% negative predictive value) √ increased size + asymmetry of gland √ multiple 1-4 cm solid nodules √ areas of hemorrhage + necrosis √ coarse calcifications may occur within adenoma (secondary to hemorrhage + necrosis)

Notes:

Diffuse Goiter US: √ increase in glandular size, R lobe > L lobe √ NO focal textural changes √ calcifications not associated with nodules

Notes:

Iodine-deficiency Goiter Not a significant problem in United States because of supplemental iodine in food *Etiology:*chronic TSH stimulation ● low serum T_4 √ high I-131 uptake ***Jod-Basedow Phenomenon*** (2%) =development of thyrotoxicosis (= excessive amounts of T_4 synthesized + released) if normal dietary intake is resumed / iodinated contrast medium administered*Incidence:*most common in individuals with long-standing multinodular goiter*Age:*>50 years √ multinodular goiter with in- / decreased uptake (depending on iodine pool)

Notes:

Toxic Nodular Goiter =PLUMMER DISEASE=autonomous function of one / more thyroid adenomas*Peak age:*4-5th decade; M:F = 1:3 ● elevated T_4 ● suppressed TSH√ nodular thyroid with hot nodule + suppression of remainder of gland√ stimulation scan will disclose normal uptake in remainder of gland√ increased radioiodine uptake by 24 hours of approximately 80%*Rx:* (1)I-131 treatment with empirical dose of 25-29 mCi (hypothyroidism in 5-30%)(2)Surgery (hypothyroidism in 11%)(3)Percutaneous ethanol injection (hypothyroidism in <1%, transient damage of recurrent laryngeal nerve in 4%)

Notes:

Intrathoracic Goiter =extension of cervical thyroid tissue / ectopic thyroid tissue (rare) into mediastinum*Incidence:*5% of resected mediastinal masses; most common cause of mediastinal massesLocation:usually anterior, 25% posterior exclusively on right side ● mostly asymptomatic ● symptoms of tracheal + esophageal + recurrent laryngeal nerve compression√ continuity with cervical thyroid / lack of continuity (with narrow fibrous / vascular pedicle)√ mass of high HU + well-defined borders√ frequent focal calcifications√ inhomogeneous texture with low-density areas(= degenerative cystic areas) √ marked + prolonged enhancement

Notes:

GRAVES DISEASE
=DIFFUSE TOXIC GOITER=autoimmune disorder with thyroid stimulating antibodies (LATS) producing hyperplasia + hypertrophy of thyroid gland*Peak age:*3rd-4th decade; M:F = 1:7 ● elevated T_3 + T_4 ● depressed TSH production ● dermopathy = pretibial myxedema (5%) ● ophthalmopathy = periorbital edema, lid retraction, ophthalmoplegia, proptosis, malignant exophthalmos√ diffuse thyroid enlargement√ uniformly increased uptake√ incidental nodules superimposed on preexisting adenomatous goiter (5%)US:(identical to diffuse goiter)√ global enlargement of 2-3 x the normal size√ normal / diffusely hypoechoic pattern√ hyperemia on color Doppler*Rx:*I-131 treatments (for adults):*Dose:*80-120 µCi/g of gland with 100% uptake (taking into account estimated weight of gland + measured radioactive iodine uptake for 24 hours)*Cx:*10-30% develop hypothyroidism within 1st year + 3%/year rate thereafter

Notes:

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HYPOPHARYNGEAL CARCINOMA

*Histo:*squamous cell carcinoma*May be associated with:* Plummer-Vinson syndrome(= atrophic mucosa, achlorhydria, sideropenic anemia) affecting women in 90% ● sore throat, intolerance to hot / cold liquids (early signs) ● dysphagia, weight loss (late signs) ● cervical adenopathy (in 50% at presentation)*Stage:* T1tumor limited to one subsiteT2tumor involves >1 subsite / adjacent site without fixation of hemilarynxT3same as T2 with fixation of hemilarynxT4invasion of thyroid / cricoid cartilage / soft tissue of neck

Pyriform Sinus Carcinoma Postcricoid Carcinoma Posterior Pharyngeal Wall Carcinoma

Notes:

Pyriform Sinus Carcinoma *Incidence:*60% of hypopharyngeal carcinomas ● may escape clinical detection if located at inferior tip; often origin of "cervical adenopathy with unknown primary" (next to primaries in lingual + faucial tonsils and nasopharynx)√ invasion of posterior ala of thyroid cartilage, cricothyroid space, soft tissue of neck in T4 lesion *Prognosis:*poor due to early soft-tissue invasion

Notes:

Postcricoid Carcinoma *Incidence:*25% of hypopharyngeal carcinomas√ difficult assessment due to varying thickness of inferior constrictor + prevertebral muscles*Prognosis:*25% 5-year survival (worst prognosis)

Notes:

Posterior Pharyngeal Wall Carcinoma *Incidence:*15% of hypopharyngeal carcinomas√ invasion of retropharyngeal space with extension into oro- and nasopharynx√ retropharyngeal adenopathy

Notes:

INVERTED PAPILLOMA
=INVERTING PAPILLOMA = ENDOPHYTIC PAPILLOMA = SQUAMOUS CELL PAPILLOMA = TRANSITIONAL CELL PAPILLOMA = CYLINDRICAL EPITHELIOMA= SCHNEIDERIAN PAPILLOMA *Incidence:*4% of all nasal neoplasms; most common of epithelial papillomas; commonly occurring after nasal surgery*Cause:*unknown; association with human papillomavirus-11*Age:*40-60 years; M:F = 3-5:1*Path:*vascular mass with prominent mucous cyst inclusions interspersed throughout epithelium*Histo:*hyperplastic epithelium inverts into underlying stroma rather than in an exophytic direction; high intracellular glycogen content Squamous cell carcinoma coexistent in 5.5-27%!Location:uniquely unilateral (bilateral in <5%)(a)most often arising from the lateral nasal wall with extension into ethmoid / maxillary sinuses, at junction of antrum + ethmoid sinuses(b)paranasal sinus (most frequently maxillary antrum)(c)nasal septum (5.5-18%) ● unilateral nasal obstruction, epistaxis, postnasal drip, recurrent sinusitis, sinus headache ● distinctive absence of allergic history√ commonly involves antrum + ethmoid sinus√ widening of infundibulum / outflow tract of antrum√ destruction of medial antral wall / lamina papyracea of orbit, anterior cranial fossa (pressure necrosis) in up to 30%√ septum may be bowed to opposite side (NO invasion)√ homogeneous enhancementMR: √ may have intermediate to low intensity on T2WI (DDx: squamous cell carcinoma, olfactory neuroblastoma, melanoma, small cell carcinoma)*Cx:*(1) cellular atypia / squamous cell carcinoma (10%)(2) recurrence rate of 15-78% *Rx:*complete surgical extirpation (lateral rhinotomy with en bloc excision of lateral nasal wall)

Notes:

JUVENILE ANGIOFIBROMA
=most common benign nasopharyngeal tumor, can grow to enormous size and locally invade vital structures*Incidence:*0.5% of all head and neck neoplasms*Age:*teenagers (mean age of 15 years); almost exclusively in males ● recurrent + severe epistaxis (59%) ● nasal speech due to nasal obstruction (91%) ● facial deformity (less common) Location:nasopharynx / posterior naresExtension:posterolateral wall of nasal cavity; via pterygopalatine fossa into retroantral region / orbit / middle cranial fossa; laterally into infratemporal fossa√ widening of pterygopalatine fossa (90%) with anterior bowing of posterior antral wall√ invasion of sphenoid sinus (2/3) from tumor erosion through floor of sinus√ widening of inferior + superior orbital fissures (spread into orbit via inferior orbital fissure + into middle cranial fossa via superior orbital fissure)√ highly vascular nasopharyngeal mass (only enhances on CT scan immediately after bolus injection); supplied primarily by internal maxillary arteryMR: √ intermediate signal intensity on T1WI with discrete punctate areas of hypointensity (secondary to highly vascular stroma)NOTE:Biopsy contraindicated!

Notes:

LABYRINTHITIS

*Cause:*viral infection (mumps, measles) > bacterial infection > syphilis, autoimmune, toxins ● sudden hearing loss, vertigo, tinnitusMR:√ faint diffuse enhancement of labyrinth on T1WI (HALLMARK) **Ramsay Hunt syndrome** = herpes zoster oticus ● mucosal vesicles of external auditory canal√ intracanalicular 8th nerve enhancement **Tympanogenic Labyrinthitis** *Cause:*agent enters through oval / round window in middle ear infection **Meningogenic Labyrinthitis** *Cause:*agent propagates along IAC / cochlear aqueduct in meningitisLocation:often bilateral***Labyrinthitis Ossificans*** =LABYRINTHITIS OBLITERANS = SCLEROSING LABYRINTHITIS = CALCIFIC / OSSIFYING COCHLEITIS*Cause:*suppurative infection (tympanogenic, meningogenic, hematogenic) in 90%, trauma, surgery, tumor, severe otosclerosis*Pathophysiology:*progressive fibrosis + ossification of granulation tissue within labyrinth ● bi - / unilateral profound deafness√ loss of normal fluid signal within labyrinth on T2WI (early in course of disease)√ inner ear structures filled with bone

Notes:

LARYNGEAL CARCINOMA
98% of all malignant laryngeal tumors; in 2% sarcomas *Risk factors:*smoking, alcohol abuse, airborne irritants *Histo:*squamous cell carcinomaSuggestive of lymph node metastasis: √ lymph node >1.5 cm in cross section√ proximity to laryngeal mass√ cluster of >3 lymph nodes 6-15 mm in size ***Supraglottic carcinoma***
*Incidence:*20-30% of all laryngeal cancersMetastases:early to lymph nodes of deep cervical chain, in 25-55% at time of presentation ● symptomatic late in course of disease (often T3 / T4)*Stage:* T1tumor confined to site of originT2involvement of adjacent supraglottic site / glottis without cord fixationT3tumor limited to larynx with cord fixation or extension to postcricoid area / medial wall of pyriform sinus / preepiglottic spaceT4extension beyond larynx with involvement of oropharynx (base of tongue) / soft tissue of neck / thyroid cartilage A.ANTERIOR COMPARTMENT1.**Epiglottic carcinoma**
√ circumferential relatively symmetric growth√ extension into preepiglottic space ± base of tongue ± paraglottic space*Prognosis:*better than for tumors of posterolateral compartmentB.POSTEROLATERAL COMPARTMENT1.**Aryepiglottic fold (marginal supraglottic) carcinoma**
√ exophytic growth from medial surface of aryepiglottic fold√ growth into fixed portion of epiglottis + paraglottic (= paralaryngeal) space2.**False vocal cord / laryngeal ventricle carcinoma**
√ submucosal spread into paraglottic space√ ± destruction of thyroid cartilage√ ± involvement of true vocal cords*Prognosis:*poorer than for cancer of the anterior compartment

Glottic Carcinoma Subglottic Carcinoma

Notes:

Glottic Carcinoma *Incidence:*50-60% of all laryngeal cancers ● early detection due to hoarseness*Stage:* T1tumor confined to vocal cord with normal mobilityT2supra- / subglottic extension ± impaired mobilityT3fixation of true vocal cordT4destruction of thyroid cartilage / extension outside larynx*Patterns of tumor invasion:* (1)anterior extension into anterior commissure√ >1 mm thickness of anterior commissure√ invasion of contralateral vocal cord via anterior commissure(2)posterior extension to arytenoid cartilage, posterior commissure, cricoarytenoid joint(3)subglottic extension√ tumor >5 mm inferior to level of vocal cords(4)deep lateral extension into paralaryngeal space*Prognosis:*T1 carcinoma rarely metastasizes (0-2%) due to absence of lymphatics within true vocal cords

Notes:

Subglottic Carcinoma *Incidence:*5% of all laryngeal cancers ● late detection due to minimal symptomatology*Stage:* T1confined to subglottic areaT2extension to vocal cords ± mobilityT3tumor confined to larynx + cord fixationT4cartilage destruction / extension beyond larynx*Prognosis:*poor due to early metastases to cervical lymph nodes (in 25% at presentation)

Notes:

LARYNGEAL PAPILLOMATOSIS

=RECURRENT RESPIRATORY PAPILLOMATOSIS◊Squamous papilloma is the most common benign tumor of the larynx!*Etiology:*human papilloma virus types 6 + 11 (Papova virus causing genital condyloma acuminatum)*Histo:*core of vascular connective tissue covered by stratified squamous epithelium*Age of onset:*1-54 years; M:F = 1:1;bimodal distribution (a)<10 years (diffuse involvement) = juvenile onset papillomatosis; probably caused by transmission from mother to child during vaginal delivery(b)21-50 years (usually single papilloma) ● progressive hoarseness / aphonia ● repeated episodes of respiratory distress ● inspiratory stridor, asthmalike symptoms ● cough ● recurrent pneumonia ● hemoptysisLocation:(a) uvula, palate (b) vocal cord (c) subglottic extension (50-70%) (d) pulmonary involvement (1-6%)√ thickened lumpy cords√ bronchiectasis*Cx:* (1)Tracheobronchial papillomatosis (2-5%)Location:lower lobe + posterior predilection√ solid pulmonary nodules in mid + posterior lung fields√ 2-3 cm large thin-walled cavity with 2-4 mm thick nodular wall (foci of squamous papillomas enlarge centrifugally, undergo central necrosis, cavitate)√ peripheral atelectasis + obstructive pneumonitis(2)Pulmonary papillomatosisfrom aerial dissemination (bronchoscopy, laryngoscopy, tracheal intubation) 10 years after initial diagnosis √ irregularities of tracheal / bronchial walls√ noncalcified granulomata progressing to cavitation(3)Malignant transformation into invasive squamous cell carcinoma*Rx:*CO_2 laser resection / surgical excision

Notes:

LARYNGOCELE
=abnormally dilated appendix / sacculus of laryngeal ventricle (= anteriorly located blind pouch within laryngeal ventricle between false + true vocal cords; normal appendix relatively large in infancy, visible in 10% of adults during phonation)*Pathogenesis:*chronic increase in intraglottic pressure*Cause:*excessive coughing, playing wind instrument, blowing glass, obstruction of appendicular ostium (= secondary laryngocele) by chronic granulomatous disease, laryngeal neoplasm*Types:* (a)internal = in parapharyngeal space confined within thyrohyoid membrane + supraglottis(b)external = protrusion above thyroid cartilage + through thyrohyoid membrane presenting as lateral neck mass near hyoid bone(c)mixed (44%) = internal + external component joined through connection at thyrohyoid membrane ● hoarseness / stridor (internal laryngocele) ● anterior neck mass just below angle of mandible (external laryngocele)Site:unilateral (80%), bilateral (20%)√ cystic mass that can be followed to level of ventricle√ increase in size during Valsalva maneuver√ decrease in size during compression√ may be filled with fluid*Cx:*infection (pyolaryngocele), formation of mucocele

Notes:

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LARYNGOMALACIA

=immaturity of cartilage; most common cause of stridor in neonate + young infant ● only cause of stridor to get worse at rest√ hypercollapsible larynx during inspiration (supraglottic portion only)√ backward bent of epiglottis + anterior kink of aryepiglottic folds during inspiration*Prognosis:*transient (disappears by age 1 year)

Notes:

LINGUAL THYROID
=solid embryonic rest of thyroid tissue, which remains ectopic along the tract of thyroglossal duct*Incidence:*in 10% of autopsies (within tongue <3 mm); M << F ● may be only functioning thyroid tissue (70-80%) ● asymptomatic (usually) ● may enlarge causing dysphagia / dyspneaLocation:midline dorsum of tongue near foramen cecum (majority), thyroglossal duct, tracheaCT:√ small focus of intrinsic high attenuation*Cx:*malignancy in 3% (papillary carcinoma)

Notes:

LYMPHANGIOMA

=congenital lymphatic malformation*Incidence:*5.6% of all benign lesions of infancy + childhood*Age:*present at birth in 50-65%, clinically apparent by end of 2nd year in 80-90%Lymphatic development: endothelial buds from veins in jugular region form confluent plexuses, which develop into rapidly enlarging bilateral juguloaxillary lymph sacs (7.5 weeks GA); these fused lymph sacs extend craniad and dorsolateral with extensive outgrowth of lymph vessels in all directions; connection with internal jugular vein at level of confluence with external jugular vein persists on the left side *Pathogenesis:* failure of drainage from primordial lymph sacs into veins / sequestration of lymphatic tissue with failure to join central lymphatic channels / abnormal budding of lymph vessels with loss of connection with lymphatic primordia *Classification* (on basis of size of lymphatic spaces): (1)Cystic lymphangioma = cystic hygroma=multilocular mass with enormously dilated lymphatic channels of varying sizeLocation: neck, axilla, mediastinum √ low signal intensity on T1WI √ high signal intensity on T2WI(2)Cavernous lymphangioma=mildly dilated cavernous lymphatic spaces with cysts of intermediate sizeLocation:tongue, floor of mouth, salivary glands √ penetration of contiguous structures √ same signal intensities as cystic lymphangioma + fibrous stromal component of low intensity on T1WI + T2WI(3)Capillary / simple lymphangioma (least common)= capillary-sized lymphatic channels Location:epidermis + dermis of proximal limbs(4)Vasculolymphatic malformationcomposed of lymphatic + vascular elements, eg, lymphangiohemangioma *Histo:*endothelial-lined lymphatic channels containing serous / milky fluid + separated by connective tissue stroma ● asymptomatic soft / semifirm mass ● may cause dyspnea / dysphagiaLocation:anywhere in developing lymphatic system(a)posterior triangle of neck (most common), with extension into mediastinum in 3-10% ● visible at birth in 65% ● clinically apparent by end of 2nd decade in 90%(b)anterior mediastinum (<1%)(c)axilla, chest wall, groin*Cx:*infection, airway compromise, chylothorax, chylopericardium*Prognosis:*spontaneous regression (10-15%)*Rx:*surgical excision (treatment of choice) with recurrence rate of up to 15%

Notes:

MALIGNANT EXTERNAL OTITIS
=severe bacterial infection of the soft tissues + bones of base of skull*Organism:*almost always Pseudomonas aeruginosa*Age:*elderly*Predisposed:*diabetes mellitus / immunocompromised ● unrelenting otalgia, headache ● purulent otorrhea unresponsive to topical antibiotics ● may cause malfunction of nerves VII, IX, X, XILocation:at bone-cartilage junction of EAC*Spread of infection:* (a)inferiorly into soft tissues inferior to temporal bone, parotid space, nasopharyngeal masticator space(b)posteriorly into mastoid(c)anteriorly into temporomandibular joint(d)medially into petrous apexCT: √ soft-tissue density in external auditory canal (100%)√ fluid in mastoid / middle ear (89%)√ disease around eustachian tube (64%)√ obliteration of fat planes beneath temporal bone (64%)√ involvement of parapharyngeal space (54%)√ masticator space disease (27%)√ mass effect in nasopharynx (54%)√ bone erosion of clivus (9%)√ intracranial extension (9%)*Cx:*bone destruction, osteomyelitis, abscess*Prognosis:*20% recurrence rate*DDx:*malignant neoplasm

Notes:

MUCOCELE
=end stage of a chronically obstructed sinus*Incidence:*most common lesion to cause expansion of paranasal sinus; increased incidence in cystic fibrosis*Etiology:*obstructed paranasal sinus ostium*Path:*expanded sinus cyst lined by mucosa with accumulated secretions and desquamations*Age:*usually adulthood ● history of chronic nasal polyposis + pansinusitis ● commonly present with unilateral proptosis ● decreased visual acuity, visual field defect ● palpable mass in superomedial aspect of orbit (frontal mucocele) ● intractable headachesLocation: *mnemonic:*"fems"**f**rontal (60%) > **e**thmoid (30%) > **m**axillary (10%) > **s**phenoid (rare) √ soft-tissue density mass√ sinus cavity expansion (DDx: never in sinusitis)√ bone demineralization + remodeling at late stage but NO bone destruction (impossible DDx from neoplasm)√ surrounding zone of bone sclerosis / calcification of edges of mucocele (from chronic infection)√ macroscopic calcification in 5% (especially with superimposed fungal infection)√ uniform enhancement of thin rimUS: √ homogeneous hypoechoic massMR: √ signal intensity varies with state of hydration, protein content, hemorrhage, air content, calcification, fibrosis√ hypointense on T1WI + signal void on T2WI due to inspissated debris + fungus√ peripheral enhancement pattern (DDx from solid enhancement pattern of neoplasms) *Cx:*(1)protrusion into orbit displacing medial rectus muscle laterally(2)expansion into subarachnoid space resulting in CSF leak(3)mucopyocele = superimposed infection (rare)*DDx:*paranasal sinus carcinoma, Aspergillus infection (enlargement of medial rectus muscle + optic nerve, focal / diffuse areas of increased attenuation), chronic infection, inverting papilloma

Notes:

MUCOEPIDERMOID CARCINOMA

=most common malignant lesion of parotid gland*Path:*arises from glandular ductal epithelium ● rock-hard mass ● pain / itching over course of facial nerve ● facial nerve paralysis well-circumscribed parotid mass (low-grade lesion) / infiltrating poorly marginated lesion (high-grade lesion)

Notes:

Cochlear Aplasia =**Michel aplasia** = Michel anomaly = agenesis of osseous + membranous labyrinth (rare)*Cause:*arrested development at 4 weeks GA ● total sensorineural hearing loss√ region of otic capsule normally occupied by cochlea is replaced by dense labyrinthine + pneumatized bone√ flat medial wall of middle ear (= undeveloped horizontal semicircular canal)√ hypoplasia of internal auditory canal√ dysplasia of vestibule = marked enlargement into region of lateral + superior semicircular canals*DDx:*labyrinthitis obliterans (no loss of lateral convexity of medial wall of middle ear)

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Single-cavity Cochlea =saccular defect / cavity in otic capsule in the position normally occupied by cochlea without recognizable modiolus, osseous spiral lamina, interscalar septum ● profound hearing loss discovered in early childhood*May be associated with:*recurrent bacterial meningitis, perilymphatic fistula of oval window cystic cochlea (= developed basal turn, middle + apical turn occupy common nondeveloped space)

Notes:

Insufficient Cochlear Turns =normal basilar turn + varying degrees of hypoplasia of middle and apical turns***Mondini malformation*** =absence of anterior 1 1/2 turns of cochlea often with preservation of the basilar turn*Cause:*in utero insult at 7 weeks GA*Frequency:*2nd most common imaging finding in children with sensorineural hearing loss ● some high-frequency hearing preserved ● vertigo ● otorrhea, rhinorrhea, recurrent meningitis (perilymphatic fistula caused by absence / defect of stapes footplate)✓ absence of cochlear apex*May be associated with:*deformity of vestibule + semicircular canals + vestibular aqueduct

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Notes:

Anomalies Of Membranous Labyrinth Scheibe dysplasia = abnormal cochlea + saccule Alexander dysplasia = dysplasia of basal turn √ normal CT findings

Notes:

Small Internal Auditory Canal =decrease in the diameter of IAC due to hypoplasia / aplasia of cochlear nerve (portion of cranial nerve VIII) ● total sensorineural hearing loss√ hypoplastic anteroinferior quadrant of IAC

Notes:

Large Vestibule *Associated with:* underdeveloped lateral semicircular canal ● sensorineural hearing deficit (most common cause)√ lateral semicircular canal smaller√ vestibule extends further into lateral + superior aspects of otic capsule

Notes:

Large Vestibular Aqueduct =Enlarged vestibular aqueduct syndrome*Age:*manifests around 3 years*Frequency:*most common imaging abnormality detected in children with sensorineural hearing loss ● unilateral congenital deafness (commonly missed) ● vertigo, tinnitus (in 50%)Location:bilateral in 50-66%√ vestibular aqueduct >1.4-2 mm in diameter measured halfway between posterior petrous bone and common crus at level of vestibule√ vestibular aqueduct larger than superior and posterior semicircular canals

Notes:

OTOSCLEROSIS
=OTOSPONGIOSIS=replacement of dense otic capsule by highly vascular spongy bone in active phase (misnomer) with restoration of density during reparative sclerotic phase*Etiology:*unknown; frequently hereditary*Age:*adolescent / young adult Caucasian; M:F = 1:2 A.STAPEDIAL = FENESTRAL OTOSCLEROSIS(80-90%)Location:anterior oval window margin (= fissula ante fenestram); bilateral in 85% ● tinnitus early in course (2/3) ● progressive conductive hearing loss (stapes fixation in oval window)√ oval window too wide (lytic phase)√ new bone formation on anterior oval window margin ± posterior oval window margin ± round window√ complete plugging of oval window = obliterative otosclerosis (in 2%) B.COCHLEAR = RETROFENESTRAL OTOSCLEROSIS (10-20%)*Invariably associated with:* fenestral otosclerosis ● progressive sensorineural hearing loss (involvement of otic capsule / cytotoxic enzyme diffusion into fluid of membranous labyrinth) ● Schwartze sign = reddish hue behind tympanic membrane when promontory involved√ "double ring / double lucent" = lucent halo around cochlea (may appear as 3rd turn to cochlea) in early phase√ bony proliferation in reparative sclerotic phase difficult to diagnose because of same density as cochlea*DDx:*Paget disease, osteogenesis imperfecta, syphilis

Notes:

PARANASAL SINUS CARCINOMA
Location:maxillary sinus (80%), nasal cavity (10%), ethmoid sinus (5-6%), frontal + sphenoid sinus (rare)

Maxillary Sinus Carcinoma Nasopharyngeal Carcinoma Ethmoid Sinus Carcinoma

Notes:

Maxillary Sinus Carcinoma *Incidence:*80% of all paranasal sinus carcinomas*Histo:*squamous cell carcinoma (80%)*Age:*>40 years in 95%; M:F = 2:1 ● asymmetry of face, tumor in oral / nasal cavity√ bone destruction (in 90%) predominates over expansion√ nodal metastases in 10-18%

Notes:

Nasopharyngeal Carcinoma *Incidence:*10% of paranasal sinus carcinomas; 0.25-0.5% of all malignant tumors in whites; M>F*Predisposed:*Chinese population*Histo:*squamous cell carcinoma (>85%), nonkeratinizing ~, undifferentiated carcinoma*Mean age:*40 years ● asymptomatic for a long time ● history of chronic sinusitis / nasal polyps (15%) ● unilateral nasal obstructionLocation:turbinates (50%) > septum > vestibule > posterior choanae > floorExtension: (a)lateral + superior: through sinus of Morgagni(= natural defect in superior portion of lateral nasopharyngeal wall) into cartilaginous portion of eustachian tube + levator veli palatini muscle±masticator space and pre- and poststyloid parapharyngeal spaces±involvement of levator + tensor veli palatini muscle, 3rd division of nerve V, petroclinoid fissure±foramen lacerum of skull base encasing internal carotid artery±cavernous sinus (along ICA / mandibular nerve / direct skull base invasion)(b)anterior: posterior nasal cavity + pterygopalatine fossa(c)inferior (1/3): submucosal spread along lateral pharyngeal wall + anterior and posterior tonsillar pillars√ polypoid or papillary (2/3) √ bone invasion (1/3)MR: √ signal intensity similar to that of adjacent mucosa

Notes:

Ethmoid Sinus Carcinoma *Incidence:*5-6% of paranasal sinus carcinomas*Histo:*squamous cell carcinoma (>90%), sarcoma, adenocarcinoma, adenoid cystic carcinoma; frequently secondarily involved from maxillary sinus carcinoma ● nasal obstruction, bloody discharge ● anosmia, broadening of nose

Notes:

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PHARYNGEAL ABSCESS

*Etiology:*spread of infection from tonsils / pharynx*Age:*children > adults ● trismus (most common presenting symptom) from involvement of pterygoid muscle ● sore throat ● low-grade fever√ isodense / low-density mass with unsharp margins√ rim enhancement*Cx:*Mycotic aneurysm of carotid artery (within 10 days)

Notes:

RAMSAY-HUNT SYNDROME

=HERPES ZOSTER OTICUS ● vesicles in mucosa of external auditory canal ↓ intracanalicular 8th nerve enhancement

Notes:

RETROPHARYNGEAL ABSCESS / HEMORRHAGE
*Etiology:*upper respiratory tract infection, perforating injury of pharynx / esophagus, suppuration of infected lymph node*Organism:*Staphylococcus, mixed flora*Age:*usually <1 year ● fever, neck stiffness, dysphagia√ thickness of retropharyngeal space >3/4 of AP diameter of vertebral body√ reversal of cervical lordosis√ anterior displacement of airway√ may contain gas and gas-fluid level

Notes:

RHABDOMYOSARCOMA
=most common soft-tissue tumor in children; *Frequency:* 5-10% of all malignant solid tumors in children <15 years of age (ranking 4th after CNS neoplasm, neuroblastoma, Wilms tumor); 3rd most common primary childhood malignancy of head + neck (following brain tumors + retinoblastomas); 10-25% of all sarcomas; annual incidence of 4.5:1,000,000 white + 1.3:1,000,000 black children *Age:*2-5 years (peak prevalence); <10 years (70%); M:F = 2:1*Histo:* (a)embryonal rhabdomyosarcoma (>50%)subtype:polyploidal form = sarcoma botryoides = grapelike(b)alveolar rhabdomyosarcoma (worst prognosis)(c)pleomorphic rhabdomyosarcoma (mostly in adults) ● cranial nerve palsyLocation:head + neck (28-36%), trigone + bladder neck (18-21%), orbit (10%), extremities (18-23%), trunk (7-8%), retroperitoneum (6-7%), perineum + anus (2%), other sites (7%)Site:paranasal sinus, middle ear, nasopharyngeal musculature (1/3); most common primary extracranial tumor invading the cranial vault in childhoodMetastases:lymph nodes (50%), lung, bone √ bulky nasopharyngeal mass√ extension into cranial vault through fissures + foramina (up to 35%) usually involving cavernous sinus√ bone destruction√ uniform enhancementCT: √ isodense with brain√ expanded foramen / fissureMR (imaging modality of choice): √ signal intensity intermediate between muscle and fat on T1WI + hyperintense on T2WI *Prognosis:*12.5% 5-year survival

Notes:

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RHINOCEREBRAL MUCORMYCOSIS
=paranasal sinus infection caused by nonseptated fungi Rhizopus arrhizus and Rhizopus oryzae*Spread:*fungus first involves nasal cavity, then extends into maxillary / ethmoid sinuses / orbits / intracranially along ophthalmic artery / cribriform plate (frontal sinuses are spared)*Predisposed:* (1) poorly controlled diabetes mellitus (2) chronic renal failure (3) cirrhosis (4) malnutrition (5) cancer (6) prolonged antibiotic therapy (7) steroid therapy (8) cytotoxic drug therapy (9) AIDS (10) extensive burns
● black crusting of nasal mucosa (in diabetics) ● small ischemic areas (invasion of arterioles + small arteries)√ nodular thickening involving nasal septum + turbinates √ mucoperiosteal thickening + clouding of ethmoids√ focal areas of bone destruction*Cx:*(1) blindness (2) cranial nerve palsy (3) hemiparesis*Prognosis:*high mortality rate

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Notes:

SARCOIDOSIS

Blacks:Whites = 10:1 Location:eye, lacrimal glands, salivary glands (40%), larynx (5%), involvement of intra- and extraparotid lymph nodes (rare)√ granulomas may enhance√ enlargement of optic canal (optic neuritis)√ thickening of larynx with enhancement of granulomas√ multiple small granulomas of septum + turbinates

Heerfordt Syndrome (1)Parotid enlargement ● diffuse bilateral painless enlargement (10-30%) ● xerostomiaCT: √ diffusely dense multinodular gland / enlargement of lymph nodes within gland(2)Uveitis(3)Facial nerve paralysis

Notes:

SIALOSIS
=nontender noninflammatory recurrent enlargement of parotid gland*Cause:*cirrhosis, alcoholism, diabetes, malnutrition, hormonal insufficiency (ovarian / pancreatic / thyroid), drugs (sulfisoxazole, phenylbutazone), radiation therapy*Histo:*serous acinar hypertrophy + fatty replacement of glandSialography: √ sparse peripheral ductsCT: √ enlarged / normal-sized gland√ diffusely dense gland in end stage

Notes:

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SINONASAL POLYPOSIS
=benign sinonasal mucosal lesion*Incidence:*in 25% of patients with allergic rhinitis; in 15% of patients with asthma*Cause:*allergic rhinitis (atopic hypersensitivity), asthma, cystic fibrosis (child), Kartagener syndrome, nickel exposure, nonneoplastic hyperplasia of inflamed mucous membranesLocation:commonly maxillary antrum √ rounded masses within nasal cavity enlarging sinus ostium√ expansion of sinus√ thinning of bony trabeculae ± erosive changes at anterior skull base√ usually peripheral / occasionally solid heterogeneous enhancement*DDx:*cancer, fungal infection

Antrochoanal Polyp Angiomatous Polyp

Notes:

Antrochoanal Polyp =benign antral polyp, which widens the sinus ostium and extends into nasal cavity; 5% of all nasal polyps*Age:*teenagers + young adults√ antral clouding√ ipsilateral nasal mass√ smooth mass enlarging the sinus ostium√ NO sinus expansion

Notes:

Angiomatous Polyp =derivative of choanal polyp (following ischemia of polyp with secondary neovascularity along its surface)*DDx:* juvenile angiofibroma (involvement of pterygopalatine fossa)

Notes:

SINUSITIS

Incidence: most common paranasal sinus problem; most common chronic disease diagnosed in United States (31,000,000 people affected each year); complicating common colds in 0.5% (3-4 colds/year in adults, 6-8 colds/year in children) *Pathogenesis:* mucosal congestion as a result of viral infection leads to apposition of mucosal surfaces resulting in retention of secretions with bacterial superinfection (1)Obstruction of major ostia(a)middle meatus draining frontal, maxillary, anterior ethmoid sinus(b)sphenoethmoidal recess draining posterior ethmoid sphenoid sinus(2)Ineffective mucociliary clearing secondary to contact of two mucosal surfaces*Predisposing anatomic variants:* (1)greater degree of nasal septal deviation(2)horizontally oriented uncinate processNOT concha bullosa, paradoxical turbinate, Haller cells, uncinate pneumatization Location: (1)Infundibular pattern (26%)=isolated obstruction of inferior infundibulum just above the maxillary sinus ostium√ limited maxillary sinus disease(2)Ostiomeatal unit pattern (25%)√ middle meatus opacification(3)Sphenoethmoidal recess obstruction (6%)√ sphenoid / posterior ethmoid sinus inflammation(4)Sinonasal polyposis pattern√ enlargement of ostia, thinning of adjacent bone√ air-fluid levelsPlain films (Waters, Caldwell, lateral, submental vertex views): 1.**Acute sinusitis**

√ air-fluid level [from retention of secretions secondary to mucosal swelling leading to ostial dysfunction] (54% sensitive, 92% specific in maxillary sinus)√ hyperintense secretions on T2WI (95% water content + 5% proteinaceous macromolecules)2.**Chronic sinusitis**

√ mucosal swelling >5 mm thick on Waters view (99% sensitive, 46% specific in maxillary sinus)√ bone remodeling + sclerosis (from osteitis)√ polyposis√ hyperattenuating lesion on NCCT (due to inspissated secretions / fungal disease)√ hypointense secretions on T1WI + T2WI due to inspissated material with chronic obstruction (DDx: air)CT: to map bony anatomy for surgical planning MR: √ sinus thickening with high signal intensity on T2WI + low intensity on T1WI√ near solid secretions with >28% protein concentration are hypointense on both T1WI + T2WI simulating air√ rim gadolinium enhancement (DDx to neoplasms which enhance centrally) A.ALLERGIC SINUSITIS√ involves multiple sinuses√ bilaterally symmetric√ uniform enhancement√ sinonasal polyposisB.BACTERIAL SINUSITIS*Organism:* (a)acute phase: Streptococcus pneumoniae + Haemophilus influenzae (>50%), beta-hemolytic streptococcus, Moraxella catarrhalis(b)chronic phase: staphylococcus, streptococcus, corynebacteria, Bacteroides, fusobacteria√ solitary antral disease (obstruction of sinus ostium)√ uniform enhancementC.MYCOTIC / FUNGAL SINUSITIS*Organism:*Aspergillus fumigatus, mucormycosis, bipolaris, Drechslera, Curvularia, Candida√ polypoid lesion / fungus ball (= extramucosal infection due to saprophytic growth on retained secretions, usually caused by Aspergillus)√ infiltrating fungal sinusitis (in immune-competent host)√ fulminant fungal sinusitis (aggressive infection in immune-compromised individual / diabetics)CT: √ punctate calcifications (= calcium phosphate / calcium sulfonate deposition near mycelium)MR: √ dark on T2WI secondary to high fungal mycelial iron, magnesium, manganese content from aminoacid metabolism(DDx: inspissated secretions / polypoid disease) *Dx:*failure to respond to antibiotic therapy *Cx:* (1)Mucous retention cyst (10%)(2)Mucocele(3)Orbital extension through neurovascular foramina, dehiscences, or thin bones: orbital cellulitis, (4)Septic thrombophlebitis(5)Intracranial extension: meningitis, epidural abscess, subdural empyema, venous sinus thrombosis, cerebral abscess*Rx:*functional endoscopic sinus surgery (amputation of uncinate process, enlargement of infundibulum + maxillary ostium, creation of common channel for anterior ethmoid air cells, complete / partial ethmoidectomy)

Notes:

SUBGLOTTIC HEMANGIOMA

Most common subglottic soft-tissue mass causing upper respiratory tract obstruction in neonates ● crouplike symptoms in neonatal period ● hemangiomas elsewhere (skin, mucosal membranes) in 50%√ eccentric thickening of subglottic portion of trachea (AP view)√ arises from posterior wall below true cords (lateral view)

Notes:

SUBGLOTTIC STENOSIS

A.CONGENITAL SUBGLOTTIC STENOSIS ● crouplike symptoms, often self-limiting diseaseLocation:1-2 cm below vocal cords√ circumferential symmetrical narrowing of subglottic portion of trachea during inspiration√ NO change in degree of narrowing with expirationB.ACQUIRED SUBGLOTTIC STENOSISfollowing prolonged endotracheal intubation (in 5%)

Notes:

THORNWALDT CYST
=midline congenital pouch / cyst lined by ectoderm within nasopharyngeal mucosal space*Origin:*persistent focal adhesion between notochord + ectoderm extending to the pharyngeal tubercle of the occipital bone *Incidence:*4% of autopsies*Peak age:*15-30 years ● asymptomatic incidental finding ● persistent nasopharyngeal drainage ● halitosis ● foul taste in mouthLocation:posterior roof of nasopharynx√ smoothly marginated cystic mass of few mm to 3 cm in size√ low density, not enhancing√ NO bone erosion*Cx:*infection of cyst*DDx:* Rathke pouch (occurs in craniopharyngeal canal located anteriorly + cephalad to Thornwaldt cyst)

Notes:

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THYROGLOSSAL DUCT CYST

Embryogenesis: thyroglossal duct = duct along which thyroid gland descends to its final position from foramen cecum at base of tongue passing anteriorly / posteriorly / through precursor of hyoid bone; duct usually involutes by 8th week of fetal life; thyroid elements remain in thyroglossal duct in 5% *Histo:*cyst lined by squamous cell mucosaAge:<10 years in 50%; 2nd peak at 20-30 years ● midline neck mass ● ± history of previous incision and drainage of an "abscess" in area of cystLocation:suprahyoid (20%), hyoid (15%), infrahyoid (65%)√ midline / paramedian cystic mass of 2-4 cm√ infrahyoid strap muscles beak over edge of cyst*Cx:*infection; thyroglossal duct carcinoma (<1%)*Rx:*complete surgical removal

Notes:

Adenomatous Nodule (42-77%)
=COLLOID NODULE = ADENOMATOUS HYPERPLASIA = DEGENERATIVE INVOLUTED NODULE *Cytology:*abundant colloid + benign follicular cells with uniform slightly large nuclei, arranged in a honeycomb pattern (difficult DDx from follicular tumors)√ often multiple nodules by US / scintigraphy / surgery√ mostly hypofunctioning, rarely hyperfunctioning√ solid form = incompletely encapsulated, poorly demarcated nodules merging with surrounding tissue√ cystic form (= colloid cyst) = anechoic areas in nodule (hemorrhage / colloid degeneration√ calcific deposits

Notes:

Follicular Adenoma (15-40%)
=monoclonal tumor arising from follicular epithelium*Path:*single lesion with well-developed fibrous capsule*Histo subtypes:* (a)Simple colloid (macrofollicular) adenoma:most common form(b)Microfollicular (fetal) adenoma(c)Embryonal (trabecular) adenoma(d)Hürthle-cell (oxyphil / oncocytic) adenoma: large single polygonal cells with abundant granular cytoplasm + uniform eccentric nuclei + no colloid(e)Atypical adenoma(f)Adenoma with papillae(g)Signet-ring adenoma√5% of microfollicular adenomas, 5% of Hürthle-cell adenomas, 25% of embryonal adenomas prove to be follicular cancers with careful study! *Functional status:* (1)Toxic adenoma(2)Toxic multinodular goiter = hyperfunctioning adenoma within multinodular goiter; usually occurs in nodule >2.5 cm in size(3)Nonfunctioning adenoma√ mass with increased / decreased echogenicity√ "halo sign" = complete hypoechoic ring with regular border surrounding isoechoic solid mass

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Notes:

THYROID CARCINOMA

*Incidence:*12,000 new cancers/year in United States; clinically silent cancers in up to 35% at autopsy / surgery (usually papillary carcinomas of <1.0 cm in size)*Age:*<30 years; M > F ● history of neck irradiation ● rapid growth ● stone-hard nodule√ hypoechoic mass√ irregular ill-defined border without halo√ NO hemorrhage / liquefaction necrosis Radiation-induced Thyroid Cancer Incidence increases with doses of thyroidal irradiation from 6.5-1,500 rad (higher doses are associated with hypothyroidism) *Peak occurrence:*5-30 (up to 50) years post irradiationThyroid abnormalities in 20%: (a)in 14% adenomatous hyperplasia, follicular adenoma, colloid nodules, thyroiditis(b)in 6% thyroid cancer◊Nondetectable microscopic foci of cancer in 25% of patients operated on for benign disease!◊In patients with multiple cold nodules frequency of cancer is 40%

WHOLE-BODY SCAN in metastatic thyroid carcinoma *Indication:*to detect metastases of thyroid carcinoma after total thyroidectomy; preferred over bone scan (only detects 40%) for skeletal metastases◊Metastases not detectable in presence of normal functioning thyroid tissue because uptake is much less in metastases◊Tc-99m pertechnetate is useless because of high background activity + lack of organification◊False-negative I-131 scan in 24% secondary to nonfunctioning metastases *Technique:* (1)T_4 replacement therapy discontinued(2)short-acting T_3 is administered for 4-6 weeks(3)T_3 replacement therapy discontinued 10-14 days prior to whole-body scan(4)measurement of TSH level to confirm adequate elevation (TSH >50 mIU/mL; administration of exogenous TSH not desirable because of uneven stimulation)(5)oral administration of 5-10 mCi I-131(6)whole-body scan after 24, 48, 72 hours (low background activity)N.B.:posttherapy scan (1 week after therapeutic dose) identifies more lesions than diagnostic scan Normal sites of accumulation: nasopharynx, salivary glands, stomach, colon, bladder, liver (I-131-labeled thyroxine produced by carcinoma is metabolized in liver), breasts in lactating women (breast feeding must be terminated after administration of I-131) CONTRAINDICATED during pregnancy!

TREATMENT for follicular / papillary cancer: (1)Surgery: total thyroidectomy + modified radical neck dissection(2)Postoperative radioiodine treatment with I-131 (multiple treatments are usually necessary)◊Radioiodine therapy only appropriate for papillary / mixed / follicular thyroid carcinomas (NOT for medullary or anaplastic carcinomas)(a)ablative dose to destroy remaining thyroid tissue 6 weeks following surgery; no thyroid hormone replacement 3-4 weeks prior to therapyDose =[(weight (g) x 80-120 µCi/g) ÷ % uptake of I-123 by 24 hours] x 100approx. 100 mCi I-131 orally(b)treatment of metastases*Dose:*100-200 mCi (dose increase with regional lymph node / lung / bone metastases to 150, 175, 200 mCi)Administration of 150 mCi of I-131 with an uptake of 0.5% per gram of tumor tissue and a biologic half-life of 4 days will produce 25,000 rads to tumor)◊Rapid turnover rates may exist in some metastases (lower dose advisable)◊Treatment of large tumors incomplete (range of beta radiation is a few mm)*Cx:*radiation thyroiditis, radiation parotitis, GI-symptoms (nausea, diarrhea), minimal bone marrow depression, leukemia (2%), anaplastic transformation (uncommon), lung fibrosis (with extensive pulmonary metastases and dose >200 mCi)(3)Thyroid replacement therapyexogenous thyroid hormone to suppress TSH stimulation of metastases (4)External radiation therapy for anaplastic carcinoma + metastases without iodine uptake FOLLOW-UP:thyroglobulin >50 ng/mL indicates functioning metastases following complete ablation of thyroid tissue

Papillary Carcinoma Of Thyroid Follicular Carcinoma Of Thyroid Anaplastic Carcinoma Of Thyroid Medullary Carcinoma Of Thyroid

Notes:

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Papillary Carcinoma Of Thyroid 60% of all thyroid carcinomas *Peak age:*5th decade; F > M*Histo:*unencapsulated well-differentiated tumor(a)purely papillary(b)mixed with follicular elements (more common, especially under age 40)Metastases: (1)Lymphogenic spread to regional lymph nodes (40%, in children almost 90%)(2)Hematogenous spread to lung (4%), bone (rare) ● carcinoma elaborates thyroglobulinNUC: √ tumor usually concentrates radioiodine (even some purely papillary tumors)US: √ tumor of decreased echogenicity√ purely solid / complex mass with areas of necrosis, hemorrhage, cystic degenerationX-ray: √ punctate / linear psammomatous calcifications at tumor periphery*Rx:*lobectomy + isthmectomy for papillary cancer <1.5 to 2.0 cm in size isolated to one lobe*Prognosis:*90% 10-year survival for occult + intrathyroidal cancer; 60% 10-year survival for extrathyroidal cancer; worse prognosis with increasing age

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Notes:

Follicular Carcinoma Of Thyroid 20% of all thyroid cancers; slow growing *Peak age*:5th decade; F > M*Histo:*encapsulated well-differentiated tumor without papillary elements; in 25% multifocal; cytologically impossible to distinguish between well-differentiated follicular carcinoma + follicular adenoma (vascular invasion is only criteria)Early hematogenous spread to: (a)lung(b)bone (30%): almost always osteolytic (more frequent than in papillary carcinoma) ● carcinoma elaborates thyroglobulin√ psammoma bodies + stromal calcium depositsNUC: √ usually concentrates pertechnetate, but fails to accumulate I-123US: √ indistinguishable from benign follicular adenoma*Prognosis:*90% 10-year survival with slight / equivocal angioinvasion; 35% 10-year survival with moderate / marked angioinvasion

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Notes:

Anaplastic Carcinoma Of Thyroid 4-15% of all thyroid cancers *Age:*6th-7th decade; M:F = 1:1√ intrathoracic extension in up to 50%√ ± invasion of carotid a., internal jugular v., larynxNUC: √ NO radioiodine uptakeCT: √ mass with inhomogeneous attenuation√ areas of necrosis (74%)√ calcifications (58%)√ regional lymphadenopathy (74%)*Prognosis:*5% 5-year survival; average survival time of 6-12 months

Notes:

Medullary Carcinoma Of Thyroid 1-5% of all thyroid cancers; sporadic / familial *Histo:*arises from parafollicular C-cells, associated with amyloid deposition in primary + metastatic sites*Mean age*:60 years for sporadic variety;in adolescence with MEN *May be associated with:* (1)MEN IIa = pheochromocytoma + parathyroid hyperplasia (Sipple syndrome)(2)MEN IIb = without parathyroid componentMetastases:early spread to lymph nodes (50%), lung, liver, bone ● elevated calcitonin (from tumor production) stimulated by pentagastrin + calcium infusion√ mass of 2 to 26 mm√ granular calcifications within fibrous stroma / amyloid masses (50%)NUC: √ NO uptake by radioiodine / pertechnetate√ frequently shows increased uptake of Tl-201CT: √ mass of low attenuation (no iodine concentration)*Prognosis:* 90% 10-year survival without nodal metastases 42% 10-year survival with nodal metastases *Rx:*total thyroidectomy + modified radical neck dissection

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Hashimoto Thyroiditis =CHRONIC LYMPHOCYTIC THYROIDITISMost frequent cause of goitrous hypothyroidism in adults in the USA (iodine-deficiency is the more common cause worldwide) *Etiology:*autoimmune process with marked familial predisposition; antibodies are typically present; functional organification defect*Peak age:*4th-5th decade; M > F ● firm rubbery lobular goiter ● gradual painless enlargement ● thyrotoxicosis in early stage (4%) ● decreased thyroid reserve ● hypothyroidism at presentation (20%)√ moderate enlargement of both lobes (18%)NUC: √ low tracer uptake (occasionally increased) with poor visualization (4%)√ prominent pyramidal lobe√ positive perchlorate washout test√ patchy tracer distribution√ multiple (40%) / single cold defects (28%) / normal thyroid (8%)US: √ initially heterogeneous diffusely decreased echogenicity + slight lobulation of contour√ marked hyperemia on color Doppler√ later densely echogenic (fibrosis) + acoustical shadows*Cx:*hypothyroidism

Notes:

DeQuervain Thyroiditis =SUBACUTE THYROIDITIS*Etiology:*probably viral*Histo:*lymphocytic infiltration + granulomas + foreign body giant cells*Peak age:*2nd-5th decade; M:F = 1:5 ● upper respiratory tract infection precedes onset of symptoms by 2-3 weeks ● painful tender gland + fever; only mild enlargement ● hyperthyroidism (50%) secondary to severe destruction ● short-lived hypothyroidism (25%) secondary to hormone depletion of glandNUC: √ abnormally low radioiodine uptake with clinical and laboratory evidence of hyperthyroidism√ poor visualization of thyroid (initially)√ single / multiple hypofunctional areas (occasionally) √ increased uptake during phase of hypothyroidism (late event) *Cx:*permanent hypothyroidism (rare)*Prognosis:*usually full recovery

Notes:

Painless Thyroiditis *Histo:*resembles chronic lymphocytic thyroiditis ● clinical presentation similar to subacute thyroiditis ● NOT painful / tender

Notes:

Acute Suppurative Thyroiditis US: √ focal / diffuse enlargement; possibly abscess √ decreased echogenicity

Notes:

WARTHIN TUMOR
=PAPILLARY CYSTADENOMA LYMPHOMATOSUM*Incidence:*2nd most common benign tumor of parotid gland; bilateral in 10%*Age:*about 50 years; M > F*Origin:*from heterotopic salivary gland tissue within parotid lymph nodes ● slow-growing mass√ well-circumscribed single / multiple tumors in parotid region usually 3-4 cm in sizeMR: √ hypointense compared with fat / surrounding parotid tissue on T2WI

Notes:

HEMOPTYSIS
*Source:*bronchial a. (most common), pulmonary a.A.TUMOR1.Carcinoma (35%)2.Bronchial adenomaB.BRONCHIAL WALL INJURY1.Foreign body erosion2.Bronchoscopy / biopsyC.VASCULAR1.COPD2.Pulmonary embolus with infarction3.Venous hypertension (most common)4.Arteriovenous malformation5.Rupture of pulmonary artery aneurysm:TB, vasculitis, trauma, neoplasm, abscess, septic embolus, indwelling catheter D.INFECTION1.Chronic bronchitis2.Bronchiectasis, mouthful (15%)3.Tuberculosis (Rasmussen aneurysm)4.Aspergillosis5.Abscess ⚱In the majority of patients no cause is found!⚱The two most common identifiable causes are bronchial carcinoma + bronchiectasis!

Notes:

PULMONARY DISEASE ASSOCIATED WITH CIGARETTE SMOKING

1.Bronchogenic carcinoma2.Chronic bronchitis3.Centrilobular emphysema4.Panacinar emphysema with a-1-antitrypsin deficiency5.Respiratory bronchiolitis-associated interstitial lung disease6.Pulmonary Langerhans cell histiocytosis

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ABNORMAL LUNG PATTERNS

1.Mass=any localized density not completely bordered by fissures / pleura2.Consolidative (alveolar) pattern=commonly produced by filling of air spaces with fluid (transudate / exudate) / cells / other material, ALSO by alveolar collapse, airway obstruction, confluent interstitial thickeningground glass=hazy area of increased attenuation not obscuring bronchovascular structuresconsolidation=marked increase in attenuation with obliteration of underlying anatomic features3.Interstitial pattern4.Vascular pattern(a)increased vessel size: CHF, pulmonary arterial hypertension, shunt vascularity, lymphangitic carcinomatosis(b)decreased vessel size: emphysema, thromboembolism5.Bronchial pattern√ wall thickening: bronchitis, asthma, bronchiectasis√ density without air bronchogram (= complete airway obstruction)√ lucency of air trapping (= partial airway obstruction with ball-valve mechanism)

Notes:

ALVEOLAR (CONSOLIDATIVE) PATTERN

Classic appearance of airspace consolidation: *mnemonic:* "A^2BC^3"√ **A**cinar rosettes:rounded poorly defined nodules in size of acini (6-10 mm), best seen at periphery of densities√ **A**ir alveologram / bronchogram√ **B**utterfly / bat-wing distribution: perihilar / bibasilar√ **C**oalescent / confluent cloudlike ill-defined opacities√ **C**onsolidation in diffuse, perihilar / bibasilar, segmental / lobar, multifocal / lobular distribution√ **C**hanges occur rapidly (labile / fleeting)HRCT: √ poorly marginated densities within primary lobule (up to 1 cm in size)√ rapid coalescence with neighboring lesions in segmental distribution√ predominantly central location with sparing of subpleural zones√ air bronchograms

Diffuse Airspace Disease Localized Airspace Disease Acute Alveolar Infiltrate Chronic Alveolar Infiltrate CT Angiogram Sign HRCT Of Small Airway Disease

Notes:

Diffuse Airspace Disease A.INFLAMMATORY EXUDATE = "PUS"1.Lobar pneumonia2.Bronchopneumonia: especially Gram-negative organisms3.Unusual pneumonias(a)viral: extensive hemorrhagic edema especially in immunocompromised patients with hematologic malignancies + transplants(b)Pneumocystis(c)fungal: Aspergillus, Candida, Cryptococcus, Phycomycetes(d)tuberculosis4.AspirationB.HEMORRHAGE = "BLOOD"1.Trauma: contusion2.Pulmonary embolism, thromboembolism3.Bleeding diathesis: leukemia, hemophilia, anticoagulants, DIC4.Vasculitis: Wegener granulomatosis, Goodpasture syndrome, SLE, mucormycosis, aspergillosis, Rocky Mountain spotted fever, infectious mononucleosis5.Idiopathic pulmonary hemosiderosis6.Bleeding metastases: choriocarcinomaC.TRANSUDATE = "WATER"1.Cardiac edema2.Neurogenic edema3.Hypoproteinemia4.Fluid overload5.Renal failure6.Radiotherapy7.Shock8.Toxic inhalation9.Drug reaction10.Adult respiratory distress syndromeD.SECRETIONS = "PROTEIN"1.Alveolar proteinosis2.Mucus pluggingE.MALIGNANCY = "CELLS"1.Bronchioloalveolar cell carcinoma2.LymphomaF.INTERSTITIAL DISEASE simulating airspace disease, eg, "alveolar sarcoid" *mnemonics:* "**P**lease **P**ut **A** **H**ot-**L**ight **A**t **T**he **S**ithouse **F**irst""AIRSPACED"**P**ulmonary edema**A**spiration**P**neumonia**I**nhalation, **I**nflammatory**A**lveolar proteinosis,**R**enal (uremia)carcinoma, microlithiasis**S**arcoidosis**H**yaline membrane disease,**P**roteinosis (alveolar)**H**emorrhage, **H**eroin**A**lveolar cell carcinoma**L**ymphoma**C**ardiovascular (CHF)**A**spiration**E**mboli**T**uberculosis**D**rug reaction, **D**rowning**S**arcoidosis**F**ungus

Notes:

Localized Airspace Disease *mnemonic:*"4PS & TAIL"**P**neumonia **P**ulmonary edema **P**ulmonary contusion **P**ulmonary interstitial edema **T**uberculosis **A**lveolar cell carcinoma **I**nfant **L**ymphoma

Notes:

Acute Alveolar Infiltrate *mnemonic:*"I 2 CHANGE FAST"**I**nfarct **I**nfection **C**ontusion **H**emorrhage **A**spiration **N**ear drowning **G**oodpasture syndrome **E**dema **F**ungus **A**llergic sensitivity **S**hock lung **T**uberculosis

Notes:

Chronic Alveolar Infiltrate *mnemonics:* "PALS GET MOD""STALLAG"**P**roteinosis**S**arcoidosis**A**lveolar cell carcinoma**T**uberculosis**L**ymphoma**A**lveolar cell ca.**S**arcoidosis**L**ymphoma**G**ranulomatosis**L**ipoid pneumonia**E**osinophilic granuloma**A**lveolar proteinosis**T**uberculosis**G**oodpasture syndr.**M**icrolithiasis **O**il aspiration **D**IP (not consolidative)

Notes:

CT Angiogram Sign =homogeneous low attenuation of lung consolidation which allows vessels to be clearly seen1.Lobar bronchioloalveolar cell carcinoma2.Lobar pneumonia3.Pulmonary lymphoma4.Extrinsic lipid pneumonia5.Pulmonary infarction6.Pulmonary edema

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Notes:

HRCT Of Small Airway Disease *Cause:* 1.Bronchiolitis obliterans2.Bronchiolitis obliterans with organizing pneumonia3.Small airway disease of smokers4.Asthma5.Infection: TB, aspiration pneumonia, viral pneumonia6.Diffuse panbronchiolitis7.Extrinsic allergic alveolitisA.DIRECT SIGNS√ ringlike tubular structures in lung periphery (= wall thickening + dilatation of bronchioles)√ nodules / branching linear structures in lung periphery (= obliterated airways through wall thickening / filling with mucus or debris)B.INDIRECT SIGNS√ air trapping = area of decreased attenuation from collateral air drift / ball-valve effect distal to occluded / stenotic airway more prominent on expiration√ mosaic perfusion = scattered areas of air trapping √ subsegmental atelectasis = wedge-shaped area of ground-glass attenuation√ centrilobular emphysema = destruction of small airways + surrounding parenchyma in the center of the pulmonary lobule√ centrilobular airspace nodule = acinar nodule = <1 cm ill-defined nodule of ground-glass attenuation (from inflammation within alveolar space) less prominent on expiration *DDx:*1.Cystic lung disease (thin septum surrounds area of air attenuation, central vessel not present)2.Panlobular emphysema (distortion of vascular + septal architecture, bullae)
Inhomogeneous Lung Attenuation On HRCT A.GROUND-GLASS OPACITY DUE TO INFILTRATIVE LUNG DISEASE√ areas of higher attenuation with nodular / centrilobular distribution√ pulmonary vessels uniform in size in areas of differing attenuation√ increase in lung attenuation in low- and high-attenuation areas on expiratory HRCTB.MOSAIC PERFUSION = patchwork of normal and air-attenuated segments√ vessels in areas of low attenuation are smaller in 94% (due to differential blood flow)√ normal / dilated arteries in areas of hyperattenuation in 77%1.Mosaic perfusion due to air trapping√ attenuation differences are accentuated on expiratory HRCT2.Mosaic perfusion due to vascular obstruction√ increase in lung attenuation in low- and high-attenuation areas on expiratory HRCT

Notes:

EOSINOPHILIC LUNG DISEASE
=PULMONARY INFILTRATION WITH BLOOD / TISSUE EOSINOPHILIA (PIE)*Classification:* 1.IDIOPATHIC EOSINOPHILIC LUNG DISEASE(a)Transient pulmonary eosinophilia = Löffler syndrome ● peripheral eosinophilia (b)Acute / chronic eosinophilic pneumonia ● no peripheral eosinophilia 2.EOSINOPHILIC LUNG DISEASE OF SPECIFIC ETIOLOGY(a)drug induced: nitrofurantoin, penicillin, sulfonamides, ASA, tricyclic antidepressants, hydrochlorothiazide, cromolyn sodium, mephenesin(b)parasite induced: tropical eosinophilia (ascariasis, schistosomiasis), strongyloidiasis, ancylostomiasis (hookworm), filariasis, Toxocara canis (visceral larva migrans), Dirofilaria immitis, amebiasis (occasionally - in right lower + middle lobe)(c)fungus induced: allergic bronchopulmonary aspergillosis, bronchocentric granulomatosis(d)Pulmonary eosinophilia with asthma3.EOSINOPHILIC LUNG DISEASE ASSOCIATED WITH ANGIITIS ± GRANULOMATOSIS(a)Wegener granulomatosis(b)Polyarteritis nodosa(c)Churg-Strauss syndrome(d)Lymphomatoid granulomatosismay lead to lymphoma √ CXR similar to Wegener granulomatosis(e)Bronchocentric granulomatosis=granulomas forming around bronchi + vasculitis ● often associated with long history of asthma √ bronchial obstruction(f)Necrotizing "sarcoidal" angiitis(g)Rheumatoid disease(h)Scleroderma(i)Dermatomyositis(j)Sjögren syndrome(k)CREST

Notes:

INTERSTITIAL LUNG DISEASE
=thickening of lung interstices (= interlobular septa)Over 200 diseases affect the interstitium of the lung!A.MAJOR LYMPHATIC TRUNKS1. Lymphangitic carcinomatosis2. Congenital pulmonary lymphangiectasiaB.PULMONARY VEINS (increased pulmonary venous pressure)1. Left ventricular failure2. Venous obstructive diseaseC.SUPPORTING CONNECTIVE TISSUE NETWORK1. Interstitial edema2. Chronic interstitial pneumonia3. Pneumoconioses4. Collagen-vascular disease5. Interstitial fibrosis6. Amyloid7. Tumor infiltration within connective tissue8. Desmoplastic reaction to tumor*Path:*stereotypical inflammatory response of alveolar wall to injury(a)acute phase: fluid + inflammatory cells exude into alveolar space, mononuclear cells accumulate in edematous alveolar wall(b)organizing phase: hyperplasia of type II pneumocytes attempt to regenerate alveolar epithelium, fibroblasts deposit collagen(c)chronic stage: dense collagenous fibrous tissue remodels normal pulmonary architecture *Characterizing criteria:* (a)zonal distribution:-upper / lower lung zones-axial (core) / parenchymal (middle) / peripheral(b)volume loss(c)time course(d)interstitial lung pattern

Interstitial Lung Pattern On CXR Distribution Of Interstitial Disease Chronic Diffuse Infiltrative Lung Disease On HRCT Generalized Interstitial Disease Diffuse Fine Reticulations Coarse Reticulations Reticulonodular Disease Nodular Disease Chronic Interstitial Disease Simulating Airspace Disease End-stage Lung Disease

Notes:

Interstitial Lung Pattern On CXR 1.LINEAR FORM(a)reticulations= network of interlacing lines in all directions (b)Kerley lines = septal lines= thickened connective septa √ Kerley A lines = relatively long fine linear shadows in upper lungs, deep within lung parenchyma√ Kerley B lines = short horizontally oriented lines extending to pleura, perpendicular to pleura in costophrenic angles + retrosternal clear space√ Kerley C lines = "spider web" appearance covering entire lung2.NODULAR FORM=small sharp numerous uniform nodules with even distribution3.DESTRUCTIVE FORM = honeycomb lung ***Signs Of Acute Interstitial Disease*** √ peribronchial cuffing = thickened bronchial wall + peribronchial sheath (when viewed end on)√ thickening of interlobular fissures√ Kerley-lines√ perihilar haze = blurring of hilar shadows√ blurring of pulmonary vascular markings√ increased density at lung bases√ small pleural effusions ***Signs Of Chronic Interstitial Disease*** √ irregular visceral pleural surface√ **reticulations** = innumerable interlacing line shadows suggesting a mesh(a)fine reticulations = early potentially reversible / minimal irreversible alveolar septal abnormality(b)coarse reticulationsin 75% related to environmental disease, sarcoidosis, collagen-vascular disorders, chronic interstitial pneumonia√ **nodularity**
in 90% related to infectious / noninfectious granulomatous process, metastatic malignancy, pneumoconioses, amyloidosis √ **linearity**
cardiogenic / noncardiogenic interstitial pulmonary edema, lymphangitic malignancy, diffuse bronchial wall disorders (cystic fibrosis, bronchiectasis, hypersensitivity asthma) √ **honeycombing** = usually subpleural clustered cystic air spaces <1 cm in diameter with thick well-defined walls set off against a background of increased lung density (end-stage lung)◊HRCT approximately 60% more sensitive than CXR

Notes:

Distribution Of Interstitial Disease A.MIDLUNG / PERIHILAR DISEASE(a)Acute rapidly changing 1.Pulmonary edema2.Pneumocystis pneumonitis3.Early extrinsic allergic alveolitis(b)Chronic slowly progressive 1.Lymphangitic carcinomatosisoften unilateral, associated with adenopathy, pleural effusion B.PERIPHERAL LUNG DISEASE(a)Acute rapidly changing 1.Interstitial pulmonary edema with Kerley B lines (most common)2.Active fibrosing alveolitis(b)Chronic slowly progressive 1.Secondary pulmonary hemosiderosis
C.UPPER LUNG DISEASE(a)Chronic slowly progressive ± volume loss1.Postprimary TB (common)2.Silicosis (common)(b)Chronic slowly progressive with volume loss1.Sarcoidosis (common)2.Ankylosing spondylitis (rare)3.Sulfa drugs (rare)(c)Chronic slowly progressive without volume loss1.Extrinsic allergic alveolitis2.Eosinophilic granuloma3.Aspiration pneumonia4.Postradiation pneumonitis5.Recurrent Pneumocystis carinii pneumonia (PCP) in a patient receiving aerosolized pentamidine prophylaxis *mnemonic:*"SHIRT CAP"**S**arcoidosis **H**istoplasmosis **I**diopathic **R**adiation therapy **T**uberculosis (postprimary) **C**hronic extrinsic alveolitis **A**nkylosing spondylitis **P**rogressive massive fibrosis
D.LOWER LUNG DISEASEUsually chronic slowly progressive + with volume loss 1.Usual interstitial pneumonia (common)2.Rheumatoid lung disease (common)3.Scleroderma (common)4.Chronic aspiration pneumonia with fibrosis more regional / unilateral5.Asbestosis (posterior aspect of lung base) *mnemonics*:
Basilar distributionApical distribution
"BAD LASS RIF""CASSET"**B**ronchiectasis**C**ystic fibrosis**A**spiration**A**nkylosing spondylitis**D**ermatomyositis**S**ilicosis**L**ymphangitic spread**S**arcoidosis**A**sbestosis**E**osinophilic granuloma**S**arcoidosis**T**uberculosis, fungus**S**cleroderma **R**heumatoid arthritis **I**diopathic pulmonary fibrosis Furadantin

Notes:

Chronic Diffuse Infiltrative Lung Disease On HRCT maximum resolution = 300 µm 1.Interlobular septal thickening=interstitial fluid / fibrosis / cellular infiltrates(a)smooth septal thickening: pulmonary edema, lymphangitic carcinomatosis(b)beaded septa / septal nodules: lymphangitic carcinomatosis(c)irregular septa imply fibrosis-distorted lobules: fibrosis-no architectural distortion of lobules: edema / infiltration2.Reticular densities(a)predominantly subpleural small reticular elements of 6-10 mm in diameter with small cystic changes ("honeycombing")*Associated with:* interstitial fibrosis, lymphangioleiomyomatosis, amyloidosis(b)fine diffusely distributed network of 2-3 mm basic elements*Associated with:* miliary TB, reactions to methotrexate-lower lung zones in subpleural areas:idiopathic pulmonary fibrosis, collagen vascular disease, asbestosis -mid lung zone / all lung zones:chronic extrinsic allergic alveolitis -mid + upper lung zones: sarcoidosis3.Nodules(a)interstitial nodulessarcoidosis, histiocytosis X, silicosis, coal worker pneumoconiosis, tuberculosis, hypersensitivity pneumonitis, metastatic tumor, amyloidosis √ perihilar peribronchovascular, centrilobular, interlobular septa, subpleural(b)airspace noduleslobular pneumonia, transbronchial spread of TB, bronchiolitis obliterans organizing pneumonia (BOOP), pulmonary edema √ ill-defined nodules, a few mm to 1 cm in size√ peribronchiolar + centrilobular-along bronchoarterial bundles + interlobular septa + subpleural: sarcoidosis-upper zone: silicosis, coal-workers pneumoconiosis-centrilobular: extrinsic allergic alveolitis4.Ground-glass attenuation=hazy increase in lung opacity without obscuration of underlying vessels◊Often indicative of an acute, active, and potentially treatable process!(a)minimal alveolar wall thickening = early interstitial lung disease(b)minimal airspace filling = alveolitis(c)partial collapse of alveoli(d)increased capillary blood volume = edema(e)normal expiration-peripheral in lower lung zones: DIP, UIP-mid + upper lung zones: sarcoidosis-"crazy paving" appearance: alveolar proteinosis-mosaic perfusion: chronic thromboembolism, bronchiolitis obliterans5.Consolidation=increase in lung opacity with obscuration of underlying vessels ± air bronchograms-subpleural in mid + upper lung zones: chronic eosinophilic pneumonia-subpleural + peribronchial: BOOP-focal: bronchioloalveolar cell carcinoma, lymphoma6.Cystic airspaces=circumscribed air-containing lesions with well-defined walls*Associated with:* lymphangioleiomyomatosis, pulmonary Langerhans-cell granulomatosis, honeycomb lung

Notes:

Generalized Interstitial Disease *mnemonic:*"HIDE FACTS"**H**amman-Rich, **H**emosiderosis **I**nfection, **I**rradiation, **I**diopathic **D**ust, **D**rugs **E**osinophilic granuloma, **E**dema **F**ungal, **F**armers lung **A**spiration (oil), **A**rthritis (rheumatoid, ankylosing spondylitis) **C**ollagen disease **T**umor, **T**B, **T**uberous sclerosis **S**arcoidosis, **S**cleroderma
***Interstitial Lung Disease* With Increased Lung Volume** *mnemonic:*"ELECTS"**E**mphysema with interstitial lung disease **L**ymphangiomyomatosis **E**osinophilic granuloma **C**ystic fibrosis **T**uberous sclerosis **S**arcoidosis

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Notes:

Diffuse Fine Reticulations ***Acute Diffuse Fine Reticulations*** A.ACUTE INTERSTITIAL EDEMA1. Congestive heart failure2. Fluid overload3. Uremia4. HypersensitivityB.ACUTE INTERSTITIAL PNEUMONIA1. Viral pneumonia2. Mycoplasma pneumonia3. Pneumocystis carinii pneumonia *mnemonic:*"HELP"**H**ypersensitivity **E**dema **L**ymphoproliferative **P**neumonitis (viral) ***Chronic Diffuse Fine Reticulations*** A.VENOUS OBSTRUCTION1.Atherosclerotic heart disease2.Mitral stenosis3.Left atrial myxoma4.Pulmonary veno-occlusive disease5.Sclerosing mediastinitisB.LYMPHATIC OBSTRUCTION1.Lymphangiectasia (pediatric patient)2.Mediastinal mass (lymphoma)3.Lymphoma / leukemia4.Lymphangitic carcinomatosis:predominantly basilar distribution (a)bilateral (breast, stomach, colon, pancreas)(b)unilateral (lung tumor)5. Lymphocytic interstitial pneumonitisC.INHALATIONAL DISEASE1. Silicosis: small nodules + reticulations2. Asbestosis: basilar distribution, pleural thickening + calcifications3. Hard metals4. Allergic alveolitisD.GRANULOMATOUS DISEASEfrom a nodular to a reticular pattern if (a)nodules line up along bronchovascular bundles(b)interlobular septa show fibrotic changes1. Sarcoidosis:hilar + mediastinal adenopathy (may have disappeared)2. Eosinophilic granuloma: upper lobe distributionE.CONNECTIVE-TISSUE DISEASEreticulations in late stages1.Rheumatoid lung2.Scleroderma3.Systemic lupus erythematosusF.DRUG REACTIONSG.IDIOPATHIC1.Usual interstitial pneumonitis (UIP)2.Desquamative interstitial pneumonitis (DIP)3.Tuberous sclerosis: smooth muscle proliferation4.Lymphangiomyomatosis5.Idiopathic pulmonary hemosiderosis6.Alveolar proteinosis (late complication)7.Amyloidosis8.Interstitial calcification (chronic renal failure)*mnemonic:*"LIFE lines"**L**ymphangitic spread **I**nflammation / infection **F**ibrosis **E**dema

Notes:

Coarse Reticulations =architectural destruction of interstitium = end-stage scarring of lung = interstitial pulmonary fibrosis= **honeycomb lung**
√ coarse reticular interstitial densities with intervening cystic spaces√ rounded radiolucencies <1 cm in areas of increased lung density√ small lung volume (decreased compliance)*Cx:*(1)intercurrent pneumothoraces(2)bronchogenic carcinoma = scar carcinoma*Cause:* A.INHALATIONAL DISEASE(a)Pneumoconioses1.Asbestosis: basilar distribution, shaggy heart, pleural thickening + calcifications2.Silicosis:upper lobe predominance, ± pleural thickening, ± hilar and mediastinal lymphadenopathy3. Berylliosis (b)Chemical inhalation (late)1. Silo-fillers disease (nitrogen dioxide) 2. Sulfur dioxide, chlorine, phosgene, cadmium (c)Extrinsic allergic alveolitis(= hypersensitivity to organic dusts) (d)Oxygen toxicitysequelae of RDS therapy with oxygen (e)Chronic aspirationeg, mineral oil: localized process in medial basal segments / middle lobe B.GRANULOMATOUS DISEASE1.Sarcoidosis2.Eosinophilic granulomaC.COLLAGEN-VASCULAR DISEASE1.Rheumatoid lung2.Scleroderma3.Ankylosing spondylitis: upper lobes4.SLE: rarely produces honeycombingD.IATROGENIC1.Drug hypersensitivity2.RadiotherapyE.IDIOPATHIC1.Usual interstitial pneumonitis (UIP)honeycombing in 50%, severe volume loss in 45% 2.Desquamative interstitial pneumonitis (DIP)honeycombing in 12.5%, severe volume loss in 23% 3.Lymphangiomyomatosis4.Tuberous sclerosis (rare)5.Neurofibromatosis (rare)6.Pulmonary capillary hemangiomatosis (rare)*DDx:*bronchiectasis, cavitary metastases (rare)
Reticulations & Pleural Effusion A.ACUTE1. Edema2. Infection: viral, Mycoplasma (very rare)B.CHRONIC1. Congestive heart failure2. Lymphangitic carcinomatosis3. Lymphoma / leukemia4. SLE5. Rheumatoid disease6. Lymphangiectasia7. Lymphangiomyomatosis8. Asbestosis ***Reticulations & Hilar Adenopathy***
1. Sarcoidosis2. Silicosis3. Lymphoma / leukemia4. Lung primary: particularly oat cell carcinoma5. Metastases: lymphatic obstruction / spread6. Fungal disease7. Tuberculosis8. Viral pneumonia (rare combination)

Notes:

Reticulonodular Disease *mnemonic:* "**P**lease **D**on't **E**at **S**tale **T**una **F**ish **S**andwiches **E**very **M**orning"**P**neumoconiosis **D**rugs **E**osinophilic granuloma **S**arcoidosis **T**uberculosis **F**ungal disease **S**chistosomiasis **E**xanthem (measles, chickenpox) **M**etastases (thyroid) ***Reticulonodular Pattern &*** Lower Lobe Predominance *mnemonic:*"CIA"**C**ollagen vascular disease **I**diopathic **A**sbestosis

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Notes:

Nodular Disease =round moderately well marginated opacity <3 cm in maximum diameterA.GRANULOMATOUS LUNG DISEASE(a)Infections: eg, tuberculosis(b)Fungal disease: eg, histoplasmosis(c)Silicosis(d)Vasculitis: eg, Wegener granulomatosisB.NEOPLASM(a)metastatic lung diseases: eg, thyroid cancer(b)lymphoma(c)bronchioloalveolar cell carcinomaC.OTHER DISEASE(a)drug-induced: methotrexate(b)nongranulomatous vasculitis(c)sarcoidosis

Macronodular Disease √ nodules >5 mm in diameter*mnemonic:*"GAMMA WARPS"**G**ranuloma (EG, fungus) **A**bscess **M**etastases **M**ultiple myeloma **A**VM **W**egener granulomatosis **A**myloidosis **R**heumatoid lung **P**arasites (Echinococcus, Paragonimiasis) **S**arcoidosis ***Micronodular Disease*** =discrete 3-5-7 mm small round focal opacity of at least soft-tissue attenuation 1.Granulomatous disease (miliary tuberculosis, histoplasmosis)2.Hypersensitivity (organic dust)3.Pneumoconiosis (inorganic dust, thesaurosis= prolonged hair spray exposure) 4.Sarcoidosis5.Metastases (thyroid, melanoma)6.Histiocytosis X7.Chickenpox

Diffuse Fine Nodular Disease & Miliary Nodules √ very small 1-4 mm sharply defined nodules of interstitial disease (a)Inhalational disease1.Silicosis + coal workers pneumoconiosis2.Berylliosis3.Siderosis4.Extrinsic allergic alveolitis (chronic phase)(b)Granulomatous disease1.Eosinophilic granuloma2.Sarcoidosis (with current / previous adenopathy)(c)Infectious disease1.Tuberculosis2.Fungus:histoplasmosis, coccidioidomycosis, blastomycosis, aspergillosis (rare), cryptococcosis (rare)3.Bacteria:salmonella, nocardiosis4.Virus:varicella (more common in adults), Mycoplasma pneumonia(d)MetastasesThyroid carcinoma, melanoma, adenocarcinoma of breast, stomach, colon, pancreas (e)Alveolar microlithiasis (rare)(f)Bronchiolitis obliterans(g)Gaucher disease *mnemonic:*"TEMPEST"**T**uberculosis + fungal disease **E**osinophilic granuloma **M**etastases (thyroid, lymphangitic carcinomatosis) **P**neumoconiosis, Parasites **E**mbolism of oily contrast **S**arcoidosis **T**uberous sclerosis Fine Nodular Disease In Afebrile Patient 1.Inhalational disease2.Eosinophilic granuloma3.Sarcoidosis4.Metastases5.Fungal infection (late stage)6.Miliary tuberculosis (rare) **Fine nodular disease in febrile patient** 1.Tuberculosis2.Fungal infection (early stage)3.Pneumocystis4.Viral pneumonia

Notes:

Chronic Interstitial Disease Simulating Airspace Disease A.REPLACEMENT OF LUNG ARCHITECTURE BY AN INTERSTITIAL PROCESS(a)NeoplasticHodgkin disease, histiocytic lymphoma (b)Benign cellular infiltratelymphocytic interstitial pneumonia, pseudolymphoma (c)Granulomatous diseasealveolar sarcoidosis (d)Fibrosis B.EXUDATIVE PHASE OF INTERSTITIAL PNEUMONIA1. UIP2. Adult respiratory distress syndrome3. Radiation pneumonitis4. Drug reaction5. Reaction to noxious gases C.CELLULAR FILLING OF AIR SPACE1. Desquamative interstitial pneumonia2. Pneumocystis carinii pneumonia

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Notes:

End-stage Lung Disease A.DISTRIBUTION1.Usual interstitial pneumonia√ subpleural distribution + lower lobe predominance2.Asbestosis√ subpleural distribution + lower lobe predominance + pleural thickening3.Sarcoidosis√ peribronchovascular distribution + upper lobe predominance4.Extrinsic allergic alveolitis√ diffuse random distribution + patchy areas of ground-glass attenuation B.CYSTIC SPACES WITH WELL-DEFINED WALLS1.Langerhans cell histiocytosis√ upper lobe predominance2.Lymphangioleiomyomatosis√ no zonal predominance C.CONGLOMERATE FIBROTIC MASSES1.Sarcoidosis√ peribronchovascular distribution2.Silicosis√ bronchi splayed around masses3.Talcosis√ areas of high attenuation (= talc deposits) ***Honeycomb Lung*** *mnemonic:* "HIPS RDS""SHIPS BOATS"**H**istiocytosis X**S**arcoidosis**I**nterstitial pneumonia**H**istiocytosis**P**neumoconiosis**I**diopathic (UIP)**S**arcoidosis**P**neumoconiosis**S**cleroderma**R**heumatoid lung**B**leomycin, **B**usulfan**D**ermatomyositis**O**xygen toxicity**S**cleroderma**A**rthritis (rheumatoid), **A**myloidosis, **A**llergic alveolitis**T**uberous sclerosis, **T**B**S**torage disease (Gaucher)

Notes:

Ground-glass Attenuation 1.Desquamative interstitial pneumonia2.Extrinsic allergic alveolitis3.Sarcoidosis4.Usual interstitial pneumonia5.Alveolar proteinosis6.Cryptogenic organizing pneumonia

Notes:

Opacification Of Hemithorax *mnemonic:*"FAT CHANCE"**F**ibrothorax **A**denomatoid malformation **T**rauma (ie, hematoma) **C**ollapse, **C**ardiomegaly **H**ernia **A**genesis of lung **N**eoplasm (ie, mesothelioma) **C**onsolidation **E**ffusion

Notes:

Atelectasis *Cause:* A.TUMOR1.Bronchogenic carcinoma (2/3 of squamous cell carcinoma occur as endobronchial mass with persistent / recurrent atelectasis or recurrent pneumonia)2.Bronchial carcinoid3.Metastases: primary tumor of kidney, colon, rectum, breast, melanoma4.Lymphoma (usually as a late presentation)5.Lipoma, granular cell myoblastoma, amyloid tumor, fibroepithelial polypB.INFLAMMATION1.Tuberculosis (endobronchial granuloma, broncholith, bronchial stenosis)2.Right middle lobe syndrome (chronic right middle lobe atelectasis)3.Sarcoidosis (endobronchial granuloma - rare)C.MUCUS PLUG1.Severe chest / abdominal pain (postoperative patient)2.Respiratory depressant drug (morphine; CNS illness)3.Chronic bronchitis / bronchiolitis obliterans4.Asthma5.Cystic fibrosis6.Bronchopneumonia (peribronchial inflammation)D.OTHER1.Large left atrium (mitral stenosis + left lower lobe atelectasis)2.Foreign body (aspiration of food, endotracheal intubation)3.Broncholithiasis4.Amyloidosis5.Wegener granulomatosis6.Bronchial transection √ local increase in lung density√ crowding of pulmonary vessels√ bronchial rearrangement√ displacement of fissures√ displacement of hilus√ mediastinal shift√ elevation of hemidiaphragm√ cardiac rotation√ approximation of ribs√ compensatory overinflation of normal lung*Types:* A.OBSTRUCTIVE ATELECTASIS**Resorptive atelectasis** *Pathophysiology:* sum of partial gas pressures in venous blood perfusing atelectatic region is less than atmospheric pressure, which is responsible for gradual resorption of air trapped distal to site of obstruction; continuing secretion into small airways leads to consolidation (postobstructive pneumonitis / bacterial infection) *Cause:*bronchiolar obstruction by1. Tumor2. Stricture3. Foreign body4. Mucus plug5. Bronchial rupture ● airless collapse within minutes to hoursMR: √ high signal intensity on T2WI in atelectatic areaB.NONOBSTRUCTIVE ATELECTASIS*Pathophysiology:* pathway between bronchial system + alveoli is maintained because bronchi are less compliant than lung parenchyma + remain patent; secretions can be eliminated + convective airflow to distal bronchioles remains ● collapsed lung not completely airless (up to 40% residual air)MR: √ low-signal intensity on T2WI in atelectatic area **Passive atelectasis** =pleural space-occupying process1. Pneumothorax2. Hydrothorax / hemothorax3. Diaphragmatic hernia4. Pleural masses: metastases, mesothelioma **Adhesive atelectasis** =decrease in surfactant production1.Respiratory distress syndrome of the newborn (hyaline membrane disease)2.Pulmonary embolism: edema, hemorrhage, atelectasis3.Intravenous injection of hydrocarbon **Cicatrizing atelectasis** =parenchymal fibrosis causing decreased lung volume1.Tuberculosis / histoplasmosis (upper lobes)2.Silicosis (upper lobes)3.Scleroderma (lower lobes)4.Radiation pneumonitis (nonanatomical distribution)5.Idiopathic pulmonary fibrosis **Discoid atelectasis** *mnemonic:*"EPIC"**E**mbolus **P**neumonia **I**nadequate inspiration **C**arcinoma, obstructing

Notes:

Multifocal Ill-defined Densities =densities 5-30 mm resulting in airspace fillingA.INFECTION1.Bacterial bronchopneumonia2.Fungal pneumonia:histoplasmosis, blastomycosis, actinomycosis, coccidioidomycosis, aspergillosis, cryptococcosis, mucormycosis, sporotrichosis 3.Viral pneumoniainitially may have interstitial appearance =tracheitis, bronchitis, bronchiolitis, peribronchial infiltrate, interstitial septa infiltrates, injury to alveolar cells, hyaline membranes, necrosis of alveolar walls with blood, edema, fibrin, macrophages in alveoli(a)Influenza: cavitary lesion confirms superimposed infection(b)Varicella / herpes zoster: 10% of adults; 2-5 days after rash(c)Rubeola (measles) = before / with onset of rash; following overt measles = giant cell pneumonia(d)Cytomegalic inclusion virus: features suggestive of bronchopneumonia(e)Coxsackie, parainfluenza, adenovirus, respiratory syncytial virus4.Tuberculosis (primary infection)5.Rocky Mountain spotted fever6.Pneumocystis cariniiB.GRANULOMATOUS DISEASE1.Sarcoidosis (alveolar form secondary to peribronchial granulomas)2.Eosinophilic granulomaC.VASCULAR1.Thromboembolic disease2.Septic emboli3.Vasculitis(a)Wegener granulomatosis(b)Wegener variants: limited Wegener, lymphomatoid granulomatosis(c)Infectious vasculitis = invasion of pulmonary arteries: mucormycosis, invasive form of aspergillosis, Rocky Mountain spotted fever(d)Goodpasture syndrome(e)SclerodermaD.NEOPLASTIC1.Bronchioloalveolar cell carcinoma=only primary lung tumor to produce multifocal ill-defined densities with air bronchograms2.Alveolar type of lymphoma=massive accumulation of tumor cells in interstitium with compression atelectasis + obstructive pneumonia3.Metastases(a)Choriocarcinoma: hemorrhage (however rare)(b)Vascular tumors: malignant hemangiomas4.Waldenström macroglobulinemia5.Angioblastic lymphadenopathy6.Mycosis fungoides7.Amyloid tumorE.IDIOPATHIC INTERSTITIAL DISEASE1.Lymphocytic Interstitial Pneumonitis (LIP)2.Desquamative Interstitial Pneumonitis (DIP)3.Pseudolymphoma = localized form of LIP4.Usual Interstitial Pneumonitis (UIP)F.INHALATIONAL DISEASE1.Allergic alveolitis: acute stage (eg, farmers lung)2.Silicosis3.Eosinophilic pneumoniaG.DRUG REACTIONS

Notes:

Diffuse Infiltrates In Immunocompromised Cancer Patient *mnemonic:*"FOLD"**F**ailure (CHF) **O**pportunistic infection **L**ymphangitic tumor spread **D**rug reaction **Segmental & Lobar Densities** A.PNEUMONIA1. Lobar pneumonia2. Lobular pneumonia3. Acute interstitial pneumonia4. Aspiration pneumonia5. Primary tuberculosisB.PULMONARY EMBOLISM(rarely multiple / larger than subsegmental) C.NEOPLASM1. Obstructive pneumonia2. Bronchioloalveolar cell carcinomaD.ATELECTASIS

Notes:

Chronic Infiltrates ***Chronic Infiltrates In Childhood*** *mnemonic:*"ABC'S"**A**sthma, **A**gammaglobulinemia, **A**spiration **B**ronchiectasis **C**ystic fibrosis **S**equestration, intralobar ***Chronic Multifocal Ill-defined Opacities*** 1.Organizing pneumonia2.Granulomatous disease3.Allergic alveolitis4.Bronchioloalveolar cell carcinoma5.Lymphoma ***Chronic Diffuse Confluent Opacities*** 1.Alveolar proteinosis2.Hemosiderosis3.Sarcoidosis

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Notes:

Ill-defined Opacities With Holes A.INFECTION1.Necrotizing pneumonias:Staphylococcus aureus, ß-hemolytic streptococcus, Klebsiella pneumoniae, E. coli, Proteus, Pseudomonas, anaerobes 2.Aspiration pneumonia:mixed Gram-negative organisms 3.Septic emboli4.Fungus:histoplasmosis, blastomycosis, coccidioidomycosis, cryptococcosis 5.TuberculosisB.NEOPLASM1.Primary lung carcinoma2.Lymphoma (cavitates very rarely)C.VASCULAR + COLLAGEN-VASCULAR DISEASE1.Emboli with infarction2.Wegener granulomatosis3.Necrobiotic rheumatoid nodulesD.TRAUMA1.Contusion with pneumatoceles

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Notes:

Perihilar "Bat-wing" Infiltrates *mnemonic:*"**P**lease, **P**lease, **P**lease, **S**tudy **L**ight, **D**on't **G**et **A**ll **U**ptight"**P**ulmonary edema **P**roteinosis **P**eriarteritis **S**arcoidosis **Lymphoma** Drugs **G**oodpasture syndrome **A**lveolar cell carcinoma **U**remia

Notes:

Peripheral "Reverse Bat-wing" Infiltrates *mnemonic:*"REDS"**R**esolving pulmonary edema **E**osinophilic pneumonia **D**esquamative interstitial pneumonia **S**arcoidosis

Notes:

Recurrent Fleeting Infiltrates 1.Löffler disease2.Bronchopulmonary aspergillosis / bronchocentric granulomatosis3.Asthma4.Subacute bacterial endocarditis with pulmonary emboli

Notes:

Tubular Density A.Mucoid impactionB.Vascular malformation1.Arteriovenous malformation2.Pulmonary varix

Notes:

PULMONARY EDEMA

Transcapillary flow dependent on (1) hydrostatic pressure (2) colloid osmotic pressure (3) capillary permeability A.INCREASED HYDROSTATIC PRESSURE(a)cardiogenic (most common)= pulmonary venous hypertension1.Heart disease: left ventricular failure, mitral valve disease, left atrial myxoma2.Pulmonary venous disease: primary veno-occlusive disease, mediastinal fibrosis3.Pericardial disease: pericardial effusion, constrictive pericarditis (extremely rare)4.Drugs: antiarrhythmic drugs; drugs depressing myocardial contractility (beta-blocker)(b)noncardiogenic1.Renal failure2.IV fluid overload3.Hyperosmolar fluid (eg, contrast medium)(c)neurogenic? sympathetic venoconstriction in cerebrovascular accident, head injury, CNS tumor, postictal state B.DECREASED COLLOID OSMOTIC PRESSURE1.Hypoproteinemia2.Transfusion of crystalloid fluid3.Rapid reexpansion of lungC.INCREASED CAPILLARY PERMEABILITYEndothelial injury from (a)physical trauma: parenchymal contusion, radiation therapy(b)aspiration injury:1.Mendelson syndrome (gastric contents)2.Near drowning in sea water / fresh water3.Aspiration of hypertonic contrast media(c)inhalation injury:1.Nitrogen dioxide = silo-fillers disease2.Smoke (pulmonary edema may be delayed by 24-48 hours)3.Sulfur dioxide, hydrocarbons, carbon monoxide, beryllium, cadmium, silica, dinitrogen tetroxide, oxygen, chlorine, phosgene, ammonia, organophosphates(d)injury via bloodstream1.Vessel occlusion: shock (trauma, sepsis, ARDS) or emboli (fat, amniotic fluid, thrombus)2.Circulating toxins: snake venom, paraquat3.Drugs: heroin, morphine, methadone, aspirin, phenylbutazone, nitrofurantoin, chlorothiazide4.Anaphylaxis: transfusion reaction, contrast medium reaction, penicillin5.Hypoxia: high altitude, acute large airway obstruction *mnemonic:*"ABCDEFGHI - PRN"**A**spiration **B**urns **C**hemicals **D**rugs (heroin, nitrofurantoin, salicylates) **E**xudative skin disorders **F**luid overload **G**ram-negative shock **H**eart failure **I**ntracranial condition **P**olyarteritis nodosa **R**enal disease **N**ear drowning

Interstitial Pulmonary Edema Pulmonary Edema With Cardiomegaly Pulmonary Edema Without Cardiomegaly Noncardiogenic Pulmonary Edema Unilateral Pulmonary Edema

Notes:

Interstitial Pulmonary Edema often marked dissociation between clinical signs + symptoms + roentgenographic evidence nothing differentiates it from other interstitial lesions does not necessarily develop before alveolar pulmonary edema NOT typical for bacterial pneumonia

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Notes:

Pulmonary Edema With Cardiomegaly 1.Cardiogenic2.Uremic (with cardiomegaly from pericardial effusion / hypertension)

Notes:

Pulmonary Edema **Without Cardiomegaly** *mnemonic:*"U DOPA"**U**remia **D**rugs **O**verhydration **P**ulmonary hemorrhage **A**cute myocardial infarction, **A**rrhythmia

Notes:

Noncardiogenic Pulmonary Edema *mnemonic:*"The alphabet"**A**RDS, **A**lveolar proteinosis, **A**spiration, **A**naphylaxis **B**leeding diathesis, **B**lood transfusion reaction **C**NS (increased pressure, trauma, surgery, CVA, cancer) **D**rowning (near), **D**rug reaction **E**mbolus (fat, thrombus) **F**luid overload, **F**oreign-body inhalation **G**lomerulonephritis, **G**oodpasture syndrome, **G**astrografin aspiration **H**igh altitude, **H**eroin, **H**ypoproteinemia **I**nhalation (SO_2, smoke, CO, cadmium, silica) **-** Narcotics, **N**itrofurantoin **O**xygen toxicity **P**ancreatitis - **R**apid reexpansion of pneumothorax / removal of pleural effusion - **T**ransfusion **U**remia

Notes:

Unilateral Pulmonary Edema A.IPSILATERAL = on side of preexisting abnormality(a) filling of airways 1.Unilateral aspiration / pulmonary lavage2.Bronchial obstruction (drowned lung)3.Pulmonary contusion(b) increased pulmonary venous pressure 1.Unilateral venous obstruction2.Prolonged lateral decubitus position(c)pulmonary arterial overload1.Systemic artery-to-pulmonary artery shunt (Waterston, Blalock-Taussig, Pott procedure)2.Rapid thoracentesis (rapid reexpansion)B.CONTRALATERAL = opposite to side of abnormality(a)pulmonary arterial obstruction1.Congenital absence / hypoplasia of pulmonary artery2.Unilateral arterial obstruction3.Pulmonary thromboembolism(b)loss of lung parenchyma1.Swyer-James syndrome2.Unilateral emphysema3.Lobectomy4.Pleural disease

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Notes:

PNEUMONIA

"Classic" pneumonia pattern: 1. Lobar distribution:Streptococcus pneumoniae2. Bulging fissure:Klebsiella3. Pulmonary edema:Viral pneumonia, Pneumocystis pneumonia4. Pneumatocele:Staphylococcus5. Alveolar nodules:Varicella, bronchogenic spread of TB *Distribution:* A.SEGMENTAL / LOBAR-Normal host: S. pneumoniae, Mycoplasma, virus-Compromised host: S. pneumoniaeB.BRONCHOPNEUMONIA-Normal host: Mycoplasma, virus, Streptococcus, Staphylococcus, S. pneumoniae-Compromised host: Gram-negative, Streptococcus, Staphylococcus-Nosocomial: Gram-negative, Pseudomonas, Klebsiella, Staphylococcus-Immunosuppressed: Gram-negative, Staphylococcus, Nocardia, Legionella, Aspergillus, PhycomycetesC.EXTENSIVE BILATERAL-Normal host: virus (eg, influenza), Legionella-Compromised host: candidiasis, Pneumocystis, tuberculosisD.BILATERAL LOWER LOBE-Normal host: anaerobic (aspiration)-Compromised host: anaerobic (aspiration)E.PERIPHERAL-Noninfectious eosinophilic pneumonia *Transmission:* A.COMMUNITY-ACQUIRED PNEUMONIA*Organism:*viruses, S. pneumoniae, Mycoplasma*Mortality:*10%B.NOSOCOMIAL PNEUMONIA(a)Gram-negative organism (>50%): Klebsiella pneumoniae, P. aeruginosa, E. coli, Enterobacter(b)Gram-positive organism (10%): S. aureus, S. pneumoniae, H. influenzae

Lobar Pneumonia Lobular Pneumonia Interstitial Pneumonia Cavitating Pneumonia Pulmonary Infiltrates In Neonate Recurrent Pneumonia In Childhood Gram-negative Pneumonia Mycotic Infections Of Lung Hypersensitivity To Organic Dusts Drug-induced Pulmonary Damage

Notes:

Lobar Pneumonia =ALVEOLAR PNEUMONIA=pathogens reach peripheral air space, incite exudation of watery edema into alveolar space, centrifugal spread via small airways, pores of Kohn + Lambert into adjacent lobules + segments√ nonsegmental sublobar consolidation√ round pneumonia (= uniform involvement of contiguous alveoli)(a)Streptococcus pneumoniae(b)Klebsiella pneumoniae (more aggressive); in immunocompromised + alcoholics(c)any pneumonia in children(d)atypical measles√ expansion of lobe with bulging of fissures√ lung necrosis with cavitation*DDx:*Aspiration, pulmonary embolus

Notes:

Lobular Pneumonia =BRONCHOPNEUMONIA=combination of interstitial + alveolar disease (injury starts in airways involves bronchovascular bundle, spills into alveoli, which may contain edema fluid, blood, leukocytes, hyaline membranes, organisms)*Organisms:* (a)Staphylococcus aureus, Pseudomonas pneumoniae: thrombosis of lobular artery branches with necrosis + cavitation(b)Streptococcus, Klebsiella, Legionnaires bacillus, Bacillus proteus, E. coli, anaerobes (Bacteroides + Clostridia), Nocardia, actinomycosis(c)Mycoplasma√ small fluffy ill-defined acinar nodules, which enlarge with time√ lobar + segmental densities with volume loss from airway obstruction secondary to bronchial narrowing + mucus plugging

Notes:

Interstitial Pneumonia *Acute Interstitial Pneumonia* =NONBACTERIAL PNEUMONIAinitially predominantly affecting interstitial tissues *Organisms:*viruses, Mycoplasma, Pneumocystis ● often subacute atypical pneumonia√ diffuse interstitial process with peribronchial thickening√ segmental / lobar densities (mucus plugging + damage of surfactant-producing type 2 alveolar cells) ***Chronic Interstitial Pneumonia*** =diverse group of inflammatory disorders that can progress to pulmonary fibrosis*Modified Liebow classification:* 1.Usual interstitial pneumonia (UIP)2.Desquamative interstitial pneumonia (DIP)3.Bronchiolitis obliterans with organizing pneumonia (BOOP)added: 4.Acute interstitial pneumonia = Hamman-Rich syndrome5.Nonspecific interstitial pneumonitis6.Respiratory bronchiolitis-associated interstitial lung diseaseno longer included: 1.Lymphoid interstitial pneumonia (LIP)=potentially malignant lymphoproliferative disorder2.Giant cell interstitial pneumonia (GIP)=manifestation of hard-metal pneumoconiosis

Notes:

Cavitating Pneumonia 1. Staphylococcus aureus2. Haemophilus influenzae3. S. pneumoniaeother Gram-negative organisms (eg, Klebsiella) ***Cavitating Opportunistic Infections*** A.FUNGAL INFECTIONS1.Aspergillosis2.Nocardiosis3.Mucormycosis (= phycomycosis)B.SEPTIC EMBOLI1.Anaerobic organismsC.STAPHYLOCOCCAL ABSCESSD.TUBERCULOSISnummular form ◊Repeated infections in same patient are not necessarily due to same organism!*DDx:*Metastatic disease in carcinoma / Hodgkin lymphoma

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Notes:

Pulmonary Infiltrates In Neonate *mnemonic:*"I HEAR"**I**nfection (pneumonia) **H**emorrhage **E**dema **A**spiration **R**espiratory distress syndrome

Notes:

Recurrent Pneumonia In Childhood A.IMMUNE PROBLEM1.Immune deficiency2.Chronic granulomatous disease of childhood (males)3.Alpha 1-antitrypsin deficiencyB.ASPIRATION1.Gastroesophageal reflux2.H-type tracheoesophageal fistula3.Disorder of swallowing mechanism4.Esophageal obstruction, impacted esophageal foreign bodyC.UNDERLYING LUNG DISEASE1.Sequestration2.Bronchopulmonary dysplasia3.Cystic fibrosis4.Atopic asthma5.Bronchiolitis obliterans6.Sinusitis7.Bronchiectasis8.Ciliary dysmotility syndromes9.Pulmonary foreign body

Notes:

Gram-negative Pneumonia In 50% cause of nosocomial necrotizing pneumonias (including staphylococcal pneumonia) *Predisposed:*elderly, debilitated, diabetes, alcoholism, COPD, malignancy, bronchitis, Gram-positive pneumonia, treatment with antibiotics, respirator therapy*Organisms:* 1.Klebsiella4.Proteus2.Pseudomonas5.Haemophilus3.E. coli6.Legionella√ airspace consolidation (Klebsiella)√ spongy appearance (Pseudomonas)√ affecting dependent lobes (poor cough reflex without clearing of bronchial tree)√ bilateral√ cavitation common*Cx:* (1) exudate / empyema (2) bronchopleural fistula

Notes:

Mycotic Infections Of Lung A.IN HEALTHY SUBJECTS1. Histoplasmosis2. Coccidioidomycosis3. BlastomycosisB.OPPORTUNISTIC INFECTION1. Aspergillosis2. Candidiasis3. Mucormycosis (phycomycosis)Growth:(a) mycelial form(b) yeast form (depending on environment)*Source of contamination:* (a) soil (b) growth in moist areas (apart from Coccidioides immitis) (c) contaminated bird / bat excreta

Notes:

Hypersensitivity To Organic Dusts A.TRACHEOBRONCHIAL HYPERSENSITIVITYlarge particles reaching the tracheobronchial mucosa (pollens, certain fungi, some animal / insect epithelial emanations) 1.Extrinsic asthma2.Hypersensitivity aspergillosis3.Bronchocentric granulomatosis4.Byssinosis in cotton-wool workersB.ALVEOLAR HYPERSENSITIVITY= HYPERSENSITIVITY PNEUMONITIS = EXTRINSIC ALLERGIC ALVEOLITIS small particles of <5 µ reaching alveoli

Notes:

Drug-induced Pulmonary Damage A.CHEMOTHERAPEUTIC AGENTS1.BUSULFAN = Myleran® (for CML)Dose-dependent toxicity after 3-4 years on the drug in 1-10% √ diffuse linear pattern (occasionally reticulonodular / nodular pattern)√ partial / complete clearing after withdrawal of drug*DDx:*Pneumocystis pneumonia, interstitial leukemic infiltrate2.BLEOMYCIN (for squamous cell carcinoma, lymphoma, testicular tumor)Toxicity at doses >300 mg (in 3-6%); increased toxicity with age + radiation therapy + high oxygen concentrations √ subpleural linear / nodular opacities in lower lung zones occurring after 1-3 months following beginning of therapy3.NITROSOUREAS = BCNU, CCNU (for glioma, lymphoma, myeloma)Incidence of 50% after doses >1500 mg/m^2 √ linear / finely nodular opacities (following treatment of 2-3 years)√ high incidence of pneumothorax4.METHOTREXATE, PROCARBAZINE (for AML, psoriasis, pemphigus)Not dose-related, usually self-limited despite continuation of therapy ● blood eosinophilia (common)√ linear / reticulonodular process (time delay of 12 days to 5 years, usually early)√ acinar filling pattern (later)√ transient hilar adenopathy + pleural effusion (on occasion)*DDx:*Pneumocystis pneumoniaB.NITROFURANTOIN (Macrodantin®)(a)acute disorder with fever + eosinophilia (common)(b)chronic reaction with interstitial fibrosis (less common), may not be associated with peripheral eosinophilia ● positive for ANA + LE cells√ bilateral basilar interstitial opacities√ prompt resolution after withdrawal from drugC.HEROIN, PROPOXYPHENE, METHADONEOverdose followed by pulmonary edema in 30-40% √ bilateral widespread airspace consolidation√ aspiration pneumonia in 50-75%D.SALICYLATES ● asthma√ pulmonary edema (with chronic ingestion)E.INTRAVENOUS CONTRAST AGENT√ pulmonary edemaF.AMIODARONE (for refractory ventricular arrhythmia) ● pulmonary insufficiency after 1-12 months in 14-18% on long-term therapy√ alveolar + interstitial infiltrates√ peripheral consolidation√ pleural thickening adjacent to consolidation√ consolidated lung parenchyma has attenuation values of iodine

Notes:

Differential-diagnostic Features Of Lung Masses

DDx Of Lung Masses On CXR √ corona radiata = spiculations strongly suggestive of primary malignancy◊89% of irregular / spiculated lesions are malignant!√ lucencies / air bronchogram(a)cavitation◊A thin-walled cavity of ≤4 mm is benign in 94%!(b)infiltrative spread with air bronchogram:bronchioloalveolar cell carcinoma, lymphoma, resolving pneumonia √ calcifications(a)central / complete:granuloma(b)peripheral:granuloma, tumor√ decrease in size with time: benign lesion◊ Bronchogenic carcinoma may show temporary decrease in size due to infarction - necrosis - fibrosis - retraction sequence!√ absence of growth over 2 years: benign lesion√ increase in size with time:masses with "doubling times" (refers to volume not diameter) of <1 month / >16 months are unlikely to be malignant (a)very rapid growth:osteosarcoma, choriocarcinoma, testicular neoplasm, organizing infectious process, infarct (thromboembolism, Wegener granulomatosis) (b)very slow growth:hamartoma, bronchial carcinoid, inflammatory pseudotumor, granuloma, low-grade adenocarcinoma, metastases from renal cell carcinoma √ nodule >3 cm is suspect for malignancy√ satellite nodules (in association with larger peripheral nodule):-in 99% due to inflammatory disease (often TB)-in 1% due to primary lung cancer√ lobulation(a)organizing mass(b)tumor with multiple cell types growing at different rates (eg, hamartoma)◊79% of sharply defined marginated lesions are benign!√ bubblelike areas of low attenuation: bronchioloalveolar cell carcinoma (in 50%)√ focal collection of fat within smoothly marginated lung nodule: hamartoma√ vessel leading to mass: pulmonary varix, AVM ***DDx Of Lung Masses On Thin-section CT*** √ air bronchogram in nodules <2 cm in diameter:in 65% malignant, in 5% benign√ spiculation: in 87% malignant, in 55% benign√ pleural tag: in 25% malignant, in 9% benign√ presence of calcification, fat, smooth edge are suggestive of benignancy√ in 31% calcifications (usually >164 HU) were not detected on CXR√ CECT (2-5 minutes after administration):benign neoplasms + granulomas enhance <15 HU; malignant neoplasms enhance >25 HU

Notes:

Benign Lung Tumor A.CENTRAL LOCATION1.Bronchial polyp2.Bronchial papilloma3.**Granular cell myoblastoma**
= cell of origin from neural crest *Age:*middle-aged, esp. Black women endobronchial lesion in major bronchiB.PERIPHERAL LOCATION1.Hamartoma2.Leiomyomabenign metastasizing leiomyoma, history of hysterectomy 3.Amyloid tumornot associated with amyloid of other organs / rheumatoid arthritis / myeloma 4.Intrapulmonary lymph node5.Arteriovenous malformation6.Endometrioma, fibroma, neural tumor, chemodectomaC.CENTRAL / PERIPHERAL1.Lipoma:(a) subpleural (b) endobronchialD.PSEUDOTUMOR1.Fibroxanthoma / xanthogranuloma2.Plasma cell granuloma3.Sclerosing hemangiomamiddle-aged woman, RML / RLL (most commonly), may be multiple 4.Pseudolymphoma5.Round atelectasis6.Pleural pseudotumor = accumulation of pleural fluid within interlobar fissure

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Notes:

Solitary Nodule / Mass *Incidence:* (a)roentgenographic survey of low-risk population: <5% of masses are cancerous(b)on surgical resection: 40% malignant tumors, 40% granulomas A.INFLAMMATION / INFECTION1.Granuloma (most common lung mass):Sarcoidosis (1/3), tuberculosis, histoplasmosis, coccidioidomycosis, nocardiosis, cryptococcosis, talc, Dirofilaria immitis (dog heartworm), gumma, atypical measles infection 2.Fluid-filled cavity: abscess, hydatid cyst, bronchiectatic cyst, bronchocele3.Mass in preformed cavity: fungus ball, mucoid impaction4.Rounded atelectasis5.Inflammatory pseudotumor: fibroxanthoma, histiocytoma, plasma cell granuloma, sclerosing hemangioma6.Paraffinoma = lipoid granuloma7.Focal organizing pneumoniaB.MALIGNANT TUMORS(a)Malignant primaries of lung1.Bronchogenic carcinoma (66%, 2nd most common mass)2.Lymphoma3.Primary sarcoma of lung4.Plasmacytoma (primary / secondary)5.Clear cell carcinoma, carcinoid, giant cell ca.(b)Metastases (4th most common cause)in adults:kidney, colon, ovary, testesin children:Wilms tumor, osteogenic sarcoma, Ewing sarcoma, rhabdomyosarcomaC.BENIGN TUMORS(a)lung tissue:hamartoma (6%, 3rd most common lung mass)(b)fat tissue:lipoma (usually pleural lesion)(c)fibrous tissue:fibroma(d)muscle tissue:leiomyoma(e)neural tissue:schwannoma, neurofibroma, paraganglioma(f)lymph tissue:intrapulmonary lymph node(g)deposits:amyloid, splenosis, endometrioma, extramedullary hematopoiesisD.VASCULAR1.Arteriovenous malformation2.Hemangioma3.Hematoma4.Organizing infarct5.Pulmonary venous varix6.Pseudoaneurysm of pulmonary artery7.Rheumatoid / vasculitic noduleE.DEVELOPMENTAL1.Bronchogenic cyst (fluid-filled)2.Pulmonary sequestrationF.INHALATIONAL1.Silicosis (conglomerate mass)2.Mucoid impaction (allergic aspergillosis)G.MIMICKING DENSITIES1.Fluid in interlobar fissure2.Mediastinal mass3.Pleural mass (mesothelioma)4.Chest wall density: nipple, rib lesion, skin tumor (mole, neurofibroma, lipoma)5.Artifacts: buttons, snaps
mnemonic:"**B**ig **S**olitary **P**ulmonary **M**asses **C**ommonly **A**ppear **H**opeless **A**nd **L**onely"**B**ronchogenic carcinoma **S**olitary metastasis, **S**equestration **P**seudotumor **M**esothelioma **C**yst (bronchogenic, neurenteric, echinococcal) **A**denoma, **A**rteriovenous malformation **H**amartoma, **H**istoplasmosis **A**bscess, **A**ctinomycosis **L**ymphoma

Notes:

Large Pulmonary Mass *mnemonic:*"CAT PIES"**C**arcinoma (large cell, squamous cell, cannon ball metastasis **A**bscess **T**oruloma (Cryptococcus) **P**seudotumor, **P**lasmacytoma **I**nflammatory **E**chinococcal disease **S**arcoma, **S**equestration

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Notes:

Cavitating Lung Nodule A.NEOPLASM(a)Lung primary:1.Squamous cell carcinoma2.Adenocarcinoma3.Bronchioloalveolar carcinoma (rare)4.Hodgkin disease (rare)(b) Metastases (4% cavitate):1.Squamous cell carcinoma (2/3)nasopharynx (males), cervix (females), esophagus 2.Adenocarcinoma (colorectal)3.Sarcoma: Ewing sarcoma, osteo-, myxo-, angiosarcoma4.Melanoma5.Seminoma, teratocarcinoma6.Wilms tumorB.COLLAGEN-VASCULAR DISEASE1.Wegener granulomatosis + Wegener variant2.Rheumatoid nodules + Caplan syndrome3.SLE4.Periarteritis nodosa (rare)C.GRANULOMATOUS DISEASE1.Histiocytosis X2.Sarcoidosis (rare)D.VASCULAR DISEASE1.Pulmonary embolus with infarction2.Septic emboli (Staphylococcus aureus)E.INFECTION1.Bacterial: pneumatoceles from staphylococcal / Gram-negative pneumonia2.Mycobacterial: TB3.Fungal: nocardiosis, cryptococcosis, coccidioidomycosis (in 10%), aspergillosis4.Parasitic: echinococcosis (multiple in 20-30%), paragonimiasisF.TRAUMA1.Traumatic lung cyst (after hemorrhage)2.Hydrocarbon ingestion (lower lobes)G.BRONCHOPULMONARY DISEASE1.Infected bulla2.Cystic bronchiectasis3.Communicating bronchogenic cyst *mnemonic:* "CAVITY"**C**arcinoma (squamous cell), **C**ystic bronchiectasis **A**utoimmune disease (Wegener granulomatosis, rheumatoid lung) **V**ascular (bland / septic emboli) **I**nfection (abscess, fungal disease, TB, Echinococcus) **T**rauma **Y**oung = congenital (sequestration, diaphragmatic hernia, bronchogenic cyst)
Pulmonary Mass With Air Bronchogram 1.Bronchioloalveolar carcinoma2.Lymphoma3.Pseudolymphoma4.Kaposi sarcoma5.Blastomycosis ***Air-crescent Sign*** =air in a crescentic shape separating the outer wall of a nodule / mass from an inner sequestrum1.Invasive pulmonary aspergillosis2.Noninvasive mycetoma3.Septic emboli4.Cavitating benign + malignant neoplasms5.Echinococcal cyst6.TB with Rasmussen aneurysms (most are too small to be identified on CXR)

Notes:

Shaggy Pulmonary Nodule *mnemonic:*"**S**haggy **S**ue **M**ade **L**oving **A** **R**eally **W**ild **F**antasy **T**oday"**S**arcoidosis, alveolar type **S**eptic emboli **M**etastasis **L**ymphoma, **L**ung primary, **L**ymphomatoid granulomatosis **A**lveolar cell carcinoma **R**heumatoid lung **W**egener granulomatosis **F**ungus **T**uberculosis

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Notes:

Hemorrhagic Pulmonary Nodule √ CT halo sign = central area of soft-tissue attenuation surrounded by a halo of ground-glass attenuation*Causes:*
A.HEMORRHAGIC INFARCTION1.Early invasive aspergillosis2.Hematogenous candidiasis3.Herpes simplex, CMV, varicella-zoster virusB.VASCULITIS1.Wegener granulomatosisC.FRAGILITY OF NEOVASCULAR TISSUE1.Kaposi sarcoma2.Metastatic angiosarcomaD.BRONCHOARTERIAL FISTULA1.CoccidioidomycosisE.TRAUMA1.Following lung biopsy

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Notes:

Multiple Nodules And Masses √ homogeneous masses with sharp border√ no air alveolo- / bronchogram A.TUMORS(a)malignant1.Metastases:from breast, kidney, GI tract, uterus, ovary, testes, malignant melanoma, sarcoma, Wilms tumor 2.Lymphoma (rare)3.Multiple primary bronchogenic carcinomas (synchronous in 1% of all lung cancers)(b)benign1.Hamartoma (rarely multiple)2.AV malformations3.AmyloidosisB.VASCULAR LESIONS1.Thromboemboli with organizing infarcts2.Septic emboli with organized infarctsC.COLLAGEN-VASCULAR DISEASE1.Wegener granulomatosis: vasculitis with organizing infarcts2.Wegener variants3.Rheumatoid nodules: tendency for periphery, occasionally cavitatingD.INFLAMMATORY GRANULOMAS1.Fungal: coccidioidomycosis, histoplasmosis, cryptococcosis2.Bacterial: nocardiosis, tuberculosis3.Viral: atypical measles4.Parasites: hydatid cysts, paragonimiasis5.Sarcoidosis: large accumulation of interstitial granulomas6.Inflammatory pseudotumors: fibrous histiocytoma, plasma cell granuloma, hyalinizing pulmonary nodules, pseudolymphoma *mnemonic:*"SLAM DA PIG"**S**arcoidosis **L**ymphoma **A**lveolar proteinosis **M**etastases **D**rugs **A**lveolar cell carcinoma **P**neumonias **I**nfarcts **G**oodpasture syndrome
Small Pulmonary Nodules *mnemonic:*"MALTS"**M**etastases (esp. thyroid) **A**lveolar cell carcinoma **Lymphoma**, **L**eukemia **T**B **S**arcoid
Pulmonary Nodules & Pneumothorax 1.Osteosarcoma2.Wilms tumor3.Histiocytosis

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Notes:

Pneumoconiosis Classification *according to ILO (International Labour Office)* A.TYPE OF OPACITIES1.Silicosis, coal workers pneumoconiosisnodular opacities:p=<1.5 mmq=1.5-3 mmr=3-10 mm2..Asbestosislinear opacities:s=finet=mediumu=coarse / blotchyB.PROFUSION / SEVERITY0=normal1=slight2=moderate3=advancedintermediate grading: 2/2= definitely moderate profusion2/3=moderate, possibly advanced profusion ***Pneumoconiosis With Mass*** Anthracosilicosis with: 1.Granuloma (histoplasmosis, TB, sarcoidosis)2.Bronchogenic carcinoma (incidence same as in general population)3.Metastasis4.Progressive massive fibrosis5.Caplan syndrome (rheumatoid nodules)

Notes:

Pleura-based Lung Nodule √ ill-defined / sharply defined lesion mimicking a true pleural mass √ associated linear densities in lung parenchyma*Cause:* 1.Granuloma (fungus, tuberculosis)2.Inflammatory pseudotumor3.Metastasis4.Rheumatoid nodule5.Pancoast tumor6.Lymphoma7.Infarct: Hampton hump8.Atelectatic pseudotumor

Notes:

Focal Area Of Ground-glass Attenuation 1.Bronchioloalveolar cell carcinoma2.Pulmonary infiltrate with eosinophilia syndrome(a)simple pulmonary eosinophilia(b)idiopathic hypereosinophilic syndrome(c)parasitic infection3.Lymphoma4.Hemorrhagic nodule

Notes:

Intrathoracic Mass Of Low Attenuation A.CYSTS1.Bronchogenic / neurenteric / pericardial cyst2.Hydatid diseaseB.FATTY SUBSTRATE1.Hamartoma2.Lipoma3.Tuberculous lymph node4.Lymphadenopathy in Whipple diseaseC.NECROTIC MASSES1.Resolving hematoma2.Treated lymphoma3.Metastases from ovary, stomach, testes

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Notes:

Multiple Pulmonary Calcifications A.INFECTION1.Histoplasmosis2.Tuberculosis3.Chickenpox pneumoniaB.INHALATIONAL DISEASE1.SilicosisC.MISCELLANEOUS1.Hypercalcemia2.Mitral stenosis3.Alveolar microlithiasis

Notes:

Calcified Pulmonary Nodules *mnemonic:*"**HAM TV S**tation"**H**istoplasmosis, **H**amartoma Amyloid, **A**lveolar microlithiasis Mitral stenosis, **M**etastasis (thyroid, osteosarcoma, mucinous carcinoma) **T**uberculosis **V**aricella **S**ilicosis ◊Central / laminated / popcorn / diffuse calcifications are characteristic of benign solitary lung nodules!

Notes:

Hyperlucent Lung

Bilateral Hyperlucent Lung A.FAULTY RADIOLOGIC TECHNIQUE1.Overpenetrated filmB.DECREASED SOFT TISSUES1.Thin body habitus2.Bilateral mastectomyC.CARDIAC CAUSE of decreased pulmonary blood flow1.Right-to-left shunt:Tetralogy of Fallot (small proximal pulmonary vessels), pseudotruncus, truncus type IV, Ebstein malformation, tricuspid atresia 2.Eisenmenger physiology of left-to-right shunt:ASD, VSD, PDA (dilated proximal pulmonary vessels) D.PULMONARY CAUSE of decreased pulmonary blood flow(a)Decrease of vascular bed:1.Pulmonary embolismbilaterality is rare; localized areas of hyperlucency (Westermark sign) (b)Increase in air space:1.Air trapping (reversible changes):acute asthmatic attack, acute bronchiolitis (pediatric patient) 2.Emphysema3.Bulla4.Bleb5.Interstitial emphysema ***Unilateral Hyperlucent Lung*** A.FAULTY RADIOLOGIC TECHNIQUE1.Rotation of patientB.CHEST WALL DEFECT1.Mastectomy2.Absent pectoralis muscle (Poland syndrome)C.INCREASED PULMONARY AIR SPACEwith decreased pulmonary blood flow (a)Large airway obstruction with air trapping@Bronchial compression:hilar mass (rare), cardiomegaly compressing LLL bronchus @Endobronchial obstruction with air trapping (collateral air drift):foreign body, broncholith, bronchogenic carcinoma, carcinoid, bronchial mucocele (b)Small airway obstruction1.Bronchiolitis obliterans2.Swyer-James / Macleod syndrome3.Emphysema (particularly bullous emphysema)4.Emphysema + unilateral lung transplant(c)Pneumothorax (in supine patient)D.PULMONARY VASCULAR CAUSE of decreased pulmonary blood flow1.Pulmonary artery hypoplasia2.Pulmonary embolism3.Congenital lobar emphysema4.Compensatory overaeration

Notes:

Localized Lucent Lung Defect A.CAVITY = tissue necrosis with bronchial drainage(a)InfectionBacterial pneumonia 1.Pyogenic infection = abscess = necrotizing pneumonia:Staphylococcus, Klebsiella, Pseudomonas, anaerobes, b-hemolytic streptococcus, E. coli, mixed Gram-negative organisms 2.Aspiration pneumonia = gravitational pneumonia: mixed Gram-negative organisms, anaerobes Granulomatous infection 1.Tuberculosiscavitation indicates active infectious disease with risk for hematogenous / bronchogenic dissemination2.Fungal infection: nocardiosis (in immunocompromised), coccidioidomycosis (any lobe, desert Southwest), histoplasmosis, blastomycosis, mucormycosis, sporotrichosis, aspergillosis, cryptococcosis√ very thin-walled cavities less likely to follow apical distribution of TB / histoplasmosis3.Sarcoidosis (stage IV, upper lobe predominance)4.Angioinvasive organism (septic lung infarction followed by cavity formation): Aspergillus, Mucorales, Candida, Torulopsis, P. aeruginosaParasitic infestation: hydatid disease (b)NeoplasmPrimary lung tumor: 16% of peripheral lung cancers (in particular in squamous cell carcinoma (30%); also in bronchioloalveolar cell carcinoma Metastasis (usually multiple) 1.Squamous cell carcinoma (nasopharynx, esophagus, cervix) in 2/32.Adenocarcinoma (lung, breast, GI)3.Osteosarcoma (rare)4.Melanoma5.Lymphoma (rare): with adenopathy; cavities often secondary to opportunistic infection with nocardiosis + cryptococcosis(c)Vascular occlusion1.Infarct (thromboembolic, septic)2.Wegener granulomatosis3.Rheumatoid arthritis(d)Inhalational1.Silicosis with coal workers pneumoconiosis-complicating tuberculosis-ischemic necrosis of center of conglomerate mass (rare)B.CYST(a)Cystic bronchiectasis1.Cystic fibrosis (more obvious in upper lobes)2.Agammaglobulinemia (predisposed to recurrent bacterial infections)3.Recurrent bacterial pneumonias√ multiple thin-walled lucencies with air-fluid levels in lower lobes4.Childhood infection: tuberculosis, pertussis5.Allergic bronchopulmonary aspergillosis (in asthmatic patients)√ involvement of proximal perihilar bronchi6.Kartagener syndrome (ciliary dysmotility)(b)Pneumatocele1.Postinfectious pneumatocele2.Traumatic pneumatocele: lung hematoma / hydrocarbon inhalation(c)Congenital lesion (rare)1.Multiple bronchogenic cysts2.Intralobar sequestration: multicystic structure in lower lobes3.Congenital cystic adenomatoid malformation (CCAM) Type I4.Diaphragmatic hernia (congenital / traumatic)(d)Centrilobular / bullous emphysema(e)Honeycomb lung

Notes:

Multiple Lucent Lung Lesions for details see causes of localized lucent lung defect A.CAVITIES(a)Infection1.Bacterial pneumonia: cavitating pneumonia, lung abscess2.Granulomatous infection: TB, sarcoidosis3.Fungal infection: coccidioidomycosis4.Parasitic infection: echinococcosis5.Protozoan infection: pneumocystosis(b)Neoplasm(c)Vascular1.Thromboembolic + septic infarcts2.Wegener granulomatosis3.Rheumatoid arthritis4.Angioinvasive organism (septic lung infarction followed by cavity formation): Aspergillus, Mucorales, Candida, Torulosis, P. aeruginosaB.CYSTS(a)Cystic bronchiectasis1.Cystic fibrosis (more obvious in upper lobes)2.Agammaglobulinemia (predisposed to recurrent bacterial infections)3.Recurrent bacterial pneumonias4.Tuberculosis5.Allergic bronchopulmonary aspergillosis (in asthmatic patients)(b)Pneumatoceles(c)Congenital lesions (rare)1.Multiple bronchogenic cysts2.Intralobar sequestration:multicystic structure in lower lobes 3.Congenital cystic adenomatoid malformation (CCAM) Type I4.Diaphragmatic hernia (congenital / traumatic)(d)Centrilobular / bullous emphysema: blebs, bullae(e)Tuberous sclerosis + lymphangiomyomatosis(f)Honeycomb lung(g)Juvenile pulmonary polyposis

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Notes:

Pulmonary Cyst =round circumscribed space surrounded by an epithelial / fibrous wall of uniform / varied thickness containing air / liquid / semisolid / solid material A.CONGENITAL CYST1.Cystic adenomatoid malformation2.Congenital lobar emphysema3.Bronchial atresia4.Bronchogenic cyst5.Sequestration B.ACQUIRED CYST1.Pneumatocele (traumatic / infectious)2.Pseudocyst (from interstitial emphysema)3.Hydatid disease4.**Bleb** = cystic air collection within visceral pleura; mostly apical with narrow neck; associated with spontaneous pneumothorax5.**Bulla** = sharply demarcated dilated air space within lung parenchyma >1 cm in diameter with epithelialized wall <1 mm thick due to destruction of alveoli (= air cyst in localized / centrilobular / panlobular emphysema) ● usually asymptomatic√ typically at lung apex√ slow progressive enlargement*Cx:*1.Spontaneous pneumothorax2."Vanishing lung" = large area of localized emphysema causing atelectasis + dyspnea*Rx:*surgical resection if bulla >33% of hemithorax ***Multiple Pulmonary Cysts*** A.INFECTION1.Tuberculosis2.Pneumocystis carinii pneumonia in AIDSB.VASCULAR-EMBOLIC1.Cavitating septic emboli√ often seen at end of feeding vessel2.Angioinvasive infection (invasive pulmonary aspergillosis, candida, P. aeruginosa)3.Pulmonary vasculitis (Wegener granulomatosis)C.DILATATION OF BRONCHI = bronchiectasis√ bronchial wall thickeningD.DISRUPTION OF ELASTIC FIBER NETWORK1.Centrilobular emphysema2.Panlobular emphysema√ lobular architecture preserved with bronchovascular bundle in central position, areas of lung destruction without arcuate contour3.Lymphangiomyomatosis√ randomly scattered cysts in otherwise normal lung4.Tuberous sclerosis√ associated skin abnormalities, mental retardation, epilepsy5.Air-block disease (adult respiratory distress syndrome, asthma, bronchiolitis, viral / bacterial pneumonia)E.REMODELING OF LUNG ARCHITECTURE=honeycombing of idiopathic pulmonary fibrosis (= fibrosing alveolitis)√ 3-10 mm small irregular thick-walled cystic air spaces usually of comparable diameter surrounded by abnormal lung parenchyma√ predominantly peripheral + basilar distributionF.MULTIFACTORIAL / UNKNOWN1.Langerhans cell histiocytosis √ cysts with walls of variable thickness√ combination of nodules ± cavitation√ septal thickening√ predominant distribution in upper lung zones2.Klippel-Trenaunay syndrome3.Juvenile tracheolaryngeal papillomatosis4.Neurofibromatosis√ cystic air spaces predominantly apical ***Cystlike Pulmonary Lesions*** *mnemonic:* "C.C., I BAN WHIPS"**C**occidioidomycosis **C**ystic adenomatoid malformation **I**nfection **B**ronchogenic cyst, **B**ronchiectasis, **B**owel **A**bscess **N**eoplasm **W**egener granulomatosis **H**ydatid cyst, **H**istiocytosis X **I**nfarction **P**neumatocele **S**equestration

Notes:

Multiple Thin-walled Cavities *mnemonic:*"BITCH"**B**ullae + pneumatoceles **I**nfection (TB, cocci, staph) **T**umor (squamous cell carcinoma) **C**ysts (traumatic, bronchogenic) **H**ydrocarbon ingestion ***Mass Within Cavity*** 1.Mycetoma = aspergilloma2.Tissue fragment within carcinoma3.Necrotic lung within abscess4.Disintegrating hydatid cyst5.Intracavitary blood clot

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Notes:

Mediastinal Shift =displacement of heart, trachea, aorta, hilar vesselsexpiration film, lateral decubitus film (expanded lung down), fluoroscopy help to determine side of abnormalityA.DECREASED LUNG VOLUME1.Atelectasis2.Postoperative (lobectomy, pneumothorax)3.Hypoplastic lung / lobe small pulmonary artery + small hilum decreased peripheral pulmonary vasculature irregular reticular vascular pattern (bronchial origin) without converging on the hilum4.Bronchiolitis obliterans = Swyer-James syndromeB.INCREASED LUNG VOLUME=**air trapping** = retention of excess gas in all / part of the lung, especially during expiration, as a result of (a) complete / partial airway obstruction, or (b) local abnormalities in pulmonary compliance@Major bronchus1.Foreign body obstructing main-stem bronchus (common in children) with ball-valve mechanism + collateral air drift contralateral mediastinal shift increasing with expiration@ Emphysema1.Bullous emphysema (localized form) large avascular areas with thin lines2.Congenital lobar emphysema: only in infants3.Interstitial emphysema pattern of diffuse coarse lines;Cx of positive pressure ventilation therapy @ Cysts / masses1.Bronchogenic cyst: with bronchial connection + check-valve mechanism2.Cystic adenomatoid malformation3.Large mass (pulmonary, mediastinal)C.PLEURAL SPACE ABNORMALITY1.Large unilateral pleural effusion:opaque hemithorax through empyema, congestive failure, metastases 2.Tension pneumothorax:not always complete collapse of lung 3.Large diaphragmatic hernia:usually detected in neonatal period 4.Large massD.Partial absence of pericardium / pectus excavatum shift of heart without shift of trachea, aorta, or mediastinal border

Notes:

Pneumomediastinum *Pathophysiology:* alveolar rupture with air tracking along bronchovascular sheath into mediastinum + facial planes of the neck producing subcutaneous emphysema *Frequency:*in 1% of patients with pneumothorax√ streaky lucencies of air in mediastinum (look at thoracic inlet on PA + retrosternal space on LAT film)√ "continuous diaphragm" sign = lucency connecting both domes of hemidiaphragms√ "V-sign of Naclerio" = air between lower thoracic aorta + diaphragm √ "spinnaker-sail" sign in children = air outlining the thymusA.SPONTANEOUS PNEUMOMEDIASTINUM*Age:*neonates (0.05-1%), 2nd-3rd decade*Causes:* (a)rupture of marginally situated alveoli from sudden rise in intraalveolar pressure (acute asthma, aspiration pneumonia, hyaline membrane disease, measles, giant cell pneumonia, coughing, vomiting, strenuous exercise, parturition, diabetic acidosis)(b)tumor erosion of trachea / esophagus(c)pneumoperitoneum / retropneumoperitoneum= extension from peritoneal / retroperitoneal / deep fascial planes of the neck*Cx:***air block** = buildup of pressure impeding blood flow in low-pressure veins; particularly common in neonatal period B.TRAUMATIC PNEUMOMEDIASTINUM (rare)1.Pulmonary interstitial emphysema=disruption of marginal alveoli with gas traveling toward mediastinum due to positive pressure ventilation2.Bronchial / tracheal rupture√ commonly associated with pneumothorax3.Esophageal rupture (diabetic acidosis, alcoholic, Boerhaave syndrome)4.Iatrogenic - accidentalneck / chest / abdominal surgery, subclavian vein catheterization, mediastinoscopy, bronchoscopy, gastroscopy, recto-sigmoido-colonoscopy, electrosurgery with intestinal gas explosion, positive pressure ventilation, intubation, barium enema

Notes:

Mediastinal Fat A.MEDIASTINAL LIPOMATOSISB.FAT HERNIATION=omental fat herniating into chest1.Foramen of Morgagni=cardiophrenic-angle mass, R >> L side2.Foramen of Bochdalek=costophrenic-angle mass, almost always on left3.Paraesophageal hernia = perigastric fat through phrenicoesophageal membraneCT: √ fat with fine linear densities (= omental vessels)C.LIPOMAun- / encapsulated with variable amount of fibrous septa √ smooth + sharply defined boundaries*DDx:*Liposarcoma, lipoblastoma (infancy), fat-containing teratoma, thymolipoma (inhomogeneous, higher CT numbers, poor demarcation, ± invasion of surrounding structures)D.MULTIPLE SYMMETRIC LIPOMATOSISrare entity without involvement of anterior mediastinal / cardiophrenic / paraspinal areas √ compression of trachea √ periscapular lipomatous masses

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Notes:

Acute Mediastinal Widening 1.Rupture of aorta / brachiocephalic arteries2.Venous hemorrhage: traumatic / iatrogenic (malpositioning of central venous line)3.Congestive heart failure (venous dilatation)4.Rupture of esophagus5.Rupture of thoracic duct6.Magnification on supine radiograph

Notes:

Mediastinal Mass (excluding hyperplastic thymus glands, granulomas, lymphoma, metastases) 1. Neurogenic tumors(28%):malignant in 16%2. Teratoid lesions(19%):malignant in 15%3. Enterogenous cysts(16%)4. Thymomas(13%):malignant in 46%5. Pericardial cysts(7%)◊75% of all mediastinal tumors are benign (in all age groups)◊1/3 diagnosed on routine chest x-ray◊2/3 found in association with symptoms (pain, cough, shortness of breath)◊80% of malignant tumors are symptomatic ***Thoracic Inlet Lesions*** 1.Thyroid mass1-3% of all thyroidectomies have a mediastinal component; 1/3 of goiters are intrathoracic Location:anterior (80%) / posterior (20%) mediastinum √ displacement of trachea posteriorly + laterally (anterior goiter)√ displacement of trachea anteriorly + esophagus posteriorly + laterally (posterior goiter)√ inhomogeneous density (cystic spaces, high-density iodine contents of >100 HU)√ focal calcifications (common)√ marked + prolonged contrast enhancement√ connection to thyroid gland√ vascular displacement + compressionNUC (rarely helpful as thyroid tissue may be nonfunctioning): √ ± uptake on I-123 / I-131 scan (pertechnetate sufficient with modern gamma cameras, SPECT imaging may be helpful)2.Cystic hygroma3-10% involve mediastinum; childhood 3.Lymphoma4.Other tumors: adenoma, carcinoma, ectopic thymoma ***Anterior Mediastinal Mass*** *mnemonic:* "4 Ts"**T**hymoma **T**eratoma **T**hyroid tumor / goiter **T**errible lymphoma A.SOLID THYMIC LESIONS1.Thymoma (benign, malignant): most common2.Normal thymus (neonate)3.Thymic hyperplasia (child)4.Thymolipoma5.LymphomaB.SOLID TERATOID LESIONS1.Teratoma2.Embryonal cell carcinoma3.Choriocarcinoma4.SeminomaC.THYROID / PARATHYROID1.Substernal thyroid / intrathoracic goiter(10% of all mediastinal masses) 2.Thyroid adenoma / carcinoma3.**Ectopic parathyroid adenoma**:ectopia in 10-22% (62-81% in anterior mediastinum / thymus, 30% within thyroid tissue, 8% in posterior superior mediastinum) D.LYMPH NODES1.Lymphoma (Hodgkin, NHL): may arise in thymus, more common in young adults2.Metastases3.Benign lymph node hyperplasia4.Angioblastic lymphadenopathy5.Mediastinal lymphadenitis: sarcoidosis / granulomatous infectionE.CARDIOVASCULAR1.Tortuous brachiocephalic artery2.Aneurysm of ascending aorta3.Aneurysm of sinus of Valsalva4.Dilated SVC5.Cardiac tumor6.Epicardial fat-padF.CYSTS1.Cystic hygroma2.Bronchogenic cyst3.Extralobar sequestration4.Thymic cysts / dermoid cysts5.Pericardial cyst: (a) true cyst(b) pericardial diverticulum6.Pancreatic pseudocystG.OTHERS1.Neural tumor (vagus, phrenic nerve)2.Paraganglioma3.Hemangioma / lymphangioma4.Mesenchymal tumor (fibroma, lipoma)5. Sternal tumors(a)metastases from breast, bronchus, kidney, thyroid(b)malignant primary (chondrosarcoma, myeloma, lymphoma)(c)benign primary (chondroma, aneurysmal bone cyst, giant cell tumor)6.Primary lung / pleural tumor(invading mediastinum) 7.Mediastinal lipomatosis:(a)Cushing disease(b)Corticosteroid therapy8.Morgagni hernia / localized eventration9.Abscess ***Middle Mediastinal Mass*** *mnemonic:*"HABIT^5"**H**ernia, **H**ematoma **A**neurysm **B**ronchogenic cyst / duplication cyst **I**nflammation (sarcoidosis, histoplasmosis, coccidioidomycosis, primary TB in children) **T**umors - remember the 5 L's: **L**ung, especially oat cell carcinoma **L**ymphoma **L**eukemia **L**eiomyoma **L**ymph node hyperplasia A.LYMPH NODES◊90% of masses in the middle mediastinum are malignant(a)Neoplastic adenopathy1.Lymphoma (Hodgkin: NHL = 2 : 1)2.Leukemia (in 25%): lymphocytic > granulocytic3.Metastasis (bronchus, lung, upper GI, prostate, kidney)4.Angioimmunoblastic lymphadenopathy(b)Inflammatory adenopathy1.Tuberculosis / histoplasmosis (may lead to fibrosing mediastinitis)2.Blastomycosis (rare) / coccidioidomycosis3.Sarcoidosis (predominant involvement of paratracheal nodes)4.Viral pneumonia (particularly measles + cat-scratch fever)5.Infectious mononucleosis / pertussis pneumonia6.Amyloidosis7.Plague / tularemia8.Drug reaction9.Giant lymph node hyperplasia= Castleman disease10.Connective tissue disease (rheumatoid, SLE)11.Bacterial lung abscess(c)Inhalational disease adenopathy1.Silicosis (eggshell calcification also in sarcoidosis + tuberculosis)2.Coal workers pneumoconiosis3.BerylliosisB.FOREGUT CYST1.Bronchogenic / respiratory cyst: cartilage, respiratory epithelium2.Enteric cyst = esophageal duplication cyst3.Extralobar sequestration (anomalous feeding vessel)4.Hiatal hernia5.Esophageal diverticula: Zenker, traction, epiphrenicC.PRIMARY TUMORS (infrequent)1.Carcinoma of trachea2.Bronchogenic carcinoma3.Esophageal tumor:leiomyoma, carcinoma, leiomyosarcoma 4.Mesothelioma5.Granular cell myoblastoma of trachea (rare)D.VASCULAR LESIONS1.Aneurysm of transverse aorta2.Distended veins (SVC, azygos vein)3.Hematoma

Posterior Mediastinal Mass A.NEOPLASMNeurogenic tumor (largest group): 30% malignant (a)Tumor of peripheral nerve origin ● more common in adulthood√ 80% appear as round masses with sulcus√ lower attenuation than muscle (in 73%)1.Schwannoma = neurilemoma (32%): derived from sheath of Schwann without nerve cells2.Neurofibroma (10%): contains Schwann cells + nerve cells, 3rd + 4th decade3.Malignant schwannoma(b)Tumor of sympathetic ganglia origin ● more common in childhood√ 80% are elongated with tapered borders1.Ganglioneuroma (23-38%): second most common tumor of posterior mediastinum after neurofibroma2.Neuroblastoma (15%): highly malignant undifferentiated small round cell tumor originating in sympathetic ganglia, <10 years of age3.Ganglioneuroblastoma (14%): both features, spontaneous maturation possible(c)Tumors of paraganglia origin (rare)1.Chemodectoma = paraganglioma (4%)2.Pheochromocytoma√ rib spreading, erosion, destruction√ enlargement of neural foramina (dumbbell lesion)√ scalloping of posterior aspect of vertebral body√ scoliosisCT:√ low-density soft-tissue mass (lipid contents)Spine tumor: metastases (eg, bronchogenic carcinoma, multiple myeloma), ABC, chordoma, chondrosarcoma, Ewing sarcoma Lymphoma Invasive thymoma Mesenchymal tumor (fibroma, lipoma, leiomyoma) Hemangioma Lymphangioma Thyroid tumor B.INFLAMMATION / INFECTION1.Infectious spondylitis: pyogenic, tuberculous, fungal√ destruction of endplates + disk space√ paravertebral soft-tissue mass2.Mediastinitis3.Lymphoid hyperplasia4.Sarcoidosis (in 2%, typically asymptomatic patient)5.Pancreatic pseudocystC.VASCULAR MASS1.Aneurysm of descending aorta (curvilinear calcification; elderly)2.Enlarged azygos + accessory hemiazygos vein3.Esophageal varices4.Congenital vascular anomalies: aberrant subclavian artery, double aortic arch, pulmonary sling, interruption of IVC with azygos / hemiazygos continuationD.TRAUMA1.Aortic aneurysm / pseudoaneurysm2.Hematoma3.Loculated hemothorax4.Traumatic pseudomeningoceleE.FOREGUT CYST√ cysts may demonstrate peripheral rimlike calcifications1.Bronchogenic cyst2.Enteric cyst3.Neurenteric cyst4.Extralobar sequestrationF.FATTY MASS1.Bochdalek hernia2.Mediastinal lipomatosis3.Fat-containing tumors: lipoma, liposarcoma, teratoma (rare)G.OTHER1.Loculated pleural effusion2.Pancreatic pseudocyst3.Lateral meningocele (neurofibromatosis; enlarged neural foramen)4.Extramedullary hematopoiesis:in chronic bone marrow deficiency; paraspinal area rich in RES-elements √ splenomegaly; widening of ribs5."Pseudomass" of the newborn *mnemonic:*"BELLMAN"**B**ochdalek hernia **E**xtramedullary hematopoiesis **L**ymphadenopathy **L**ymphangioma **M**eningocele (lateral) **A**neurysm **N**eurogenic tumor ***Aorticopulmonary Window Mass*** 1.Adenopathy2.Traumatic aortic pseudoaneurysm3.Pulmonary artery aneurysm4.Bronchogenic cyst5.Tumor of tracheobronchial tree6.Esophageal tumor7.Neurogenic tumor8.Mediastinal abscess ***Hypervascular Mediastinal Mass*** 1.Paraganglioma2.Metastasis: typically renal cell carcinoma3.Castleman disease4.Hemangioma5.Sarcoma6.Tuberculosis7.Sarcoidosis ***Cardiophrenic-angle Mass*** A.Lesion of pericardium1.Pericardial cyst2.Intrapericardiac bronchogenic cyst3.Benign intrapericardiac neoplasm:teratoma, leiomyoma, hemangioma, lipoma 4.Malignant neoplasm:mesothelioma, metastasis (lung, breast, lymphoma, melanoma) B.Cardiac lesion: aneurysmC.Others:masses arising from lung, pleura, diaphragm, abdomen **Right Cardiophrenic-angle Mass** A.Heart1.Aneurysm (cardiac ventricle, sinus of Valsalva)2.Dilated right atriumB.Peri- / epicardium1.Epicardial fat-pad / lipoma (most common cause)√ triangular opacity in cardiophrenic angle less dense than heart√ increase in size under corticosteroid treatment2.Pericardial cystC.Diaphragm1.Diaphragmatic hernia of Morgagni2.Diaphragmatic lymph node (esp. in Hodgkin disease + breast cancer)D.Anterior mediastinal massE.Primary lung massF.Paracardiac varices

Notes:

Low-attenuation Mediastinal Mass A.FLUID1.Foregut cyst2.Lymphocele3.Seroma4.Hematoma5.Abscess6.Hydatid diseaseB.LYMPH NODE1.Tuberculous lymph nodes2.Metastasis from thyroid / testicular tumor3.Lymphoma: treated / untreatedC.PRIMARY NEOPLASM1.Neurogenic tumor2.Fat-containing neoplasm

Notes:

Mediastinal Cysts =21% of all primary mediastinal tumors, mostly developmental1.Pericardial cyst2.Thymic cyst3.FOREGUT CYST(a)Bronchogenic cyst (54-63%)(b)Esophageal duplication cyst**(c)Neurenteric cyst (least common)**
4.**Lateral meningocele**

=outpouching of leptomeninges through intervertebral foramen*Etiology:*in 75% neurofibromatosis√ spinal abnormalities (kyphoscoliosis, scalloping of dorsal vertebrae, enlargement of intervertebral foramen, pedicle erosion, thinning of ribs)5.**Hydatid cyst**

Location:paravertebral gutter√ erosion of ribs + vertebrae6.**Thoracic duct cyst**
rare, filled with chyle *Etiology:*degenerative / lymphangiomatous7.Posttraumatic lymphocele=contained pleural / mediastinal lymph collection ● history of prolonged chylous chest tube drainage*Time of onset:*several months after injury8.Cystic hygroma9.Parathyroid cystuncommon as mediastinal mass

Notes:

Hilar Mass A.LARGE PULMONARY ARTERIES√ enlargement of main pulmonary artery√ abrupt change in vessel caliber√ enlarged pulmonary artery compared with bronchus (in same bronchovascular bundle)√ cephalization√ enlargement of right ventricle (RAO 45°, LAO 60°)*Cause:* 1.Chronic obstructive disease (emphysema)2.Chronic restrictive interstitial lung disease (idiopathic fibrosis, cystic fibrosis, rheumatoid arthritis, sarcoidosis)3.Pulmonary embolic disease (acute massive / chronic)4.Idiopathic pulmonary hypertension5.Left-sided heart failure + mitral stenosis6.Congenital heart disease with left-to-right shunt(a)acyanotic: ASD, VSD, PDA(b)cyanotic (admixture lesions): transposition of great vessels, truncus arteriosusB.DUPLICATION CYSTC.UNILATERAL HILAR ADENOPATHY(a)NEOPLASTIC1.Bronchogenic carcinoma (most common)2.Metastases (lack of mediastinal involvement exceptional)3.Lymphoma(b)INFLAMMATORY1.Tuberculosis (primary) in 80%2.Fungal infection: histoplasmosis, coccidioidomycosis, blastomycosis3.Viral infections: atypical measles4.Infectious mononucleosis5.Drug reaction6.Sarcoidosis (in 1-3%)7.Bilateral lung abscess*mnemonic:*"**F**at **H**ila **S**uck"**F**ungus **H**odgkin disease **S**quamous / oat cell carcinoma D.BILATERAL HILAR ADENOPATHY(a)NEOPLASTIC1.Lymphoma (50% in Hodgkin disease)2.Metastases3.Leukemia4.Primary bronchogenic carcinoma5.Plasmacytoma(b)INFLAMMATORY1.Sarcoidosis (in 70-90%)2.Silicosis3.Histiocytosis X4.Idiopathic pulmonary hemosiderosis5.Chronic berylliosis(c)INFECTIOUS1.Rubella, ECHO virus, varicella, mononucleosis *mnemonic:*"**P**lease **H**elen **L**ick **M**y **P**opsicle **S**tick"**P**rimary TB **H**istoplasmosis **L**ymphoma **M**etastases **P**neumoconiosis **S**arcoidosis

Notes:

Eggshell Calcification Of Nodes A.PNEUMOCONIOSIS1.Silicosis (5%)2.Coal workers pneumoconiosis (1.3-6%)not seen in: asbestosis, berylliosis, talcosis, baritosis B.SARCOIDOSIS (5%)C.FUNGAL + BACTERILA INFECTION (rare):1.Tuberculosis2.Histoplasmosis3.CoccidioidomycosisD.FIBROSING MEDIASTINITISE.LYMPHOMA FOLLOWING RADIATION THERAPY

Notes:

Enlargement Of Azygos Vein Normal azygos vein (on upright CXR): ≤7 mm A.COLLATERAL CIRCULATION1.Portal hypertension2.SVC obstruction / compression below azygos vein3.IVC obstruction / compression4.Interrupted IVC with azygos continuation5.Partial anomalous venous return (rare)6.Pregnancy7.Hepatic vein occlusionB.RIGHT ATRIAL HYPERTENSION1.Right-sided heart failure2.Constrictive pericarditis3.Large pericardial effusion

Notes:

Thymic Mass 1.Thymoma2. Thymolipoma3.Thymic cyst4.Thymic carcinoid

Notes:

Diffuse Thymic Enlargement 1.Thymic hyperplasia2.Thymic infiltrationby leukemia, Hodgkin lymphoma, non-Hodgkin lymphoma, histiocytosis ● presence of adenopathy elsewhere√ no pleural implants3.Thymic hemorrhage

Notes:

Tracheal Tumor ● asthma symptomatology ● hoarseness, cough ● wheeze (inspiratory with extrathoracic lesion, expiratory with intrathoracic lesion) ● hemoptysisA.BENIGN1.Cartilaginous tumor (hamartoma)2.Squamous cell papilloma3.Fibroma / lipoma4.Hemangioma5.Granular cell myoblastoma6.Granuloma (inflammatory, TB, fungus)7.Amyloid tumorB.MALIGNANT1.Squamous-cell carcinoma (commonest primary)2.Adenoid cystic carcinoma = cylindroma3.Metastasis from renal cell carcinoma, colon cancer, malignant melanoma4.Lymphoma5.Plasmacytoma

Notes:

Endobronchial Tumor 1.Neuroendocrine tumor (typical / atypical carcinoid)2.Mucoepidermoid carcinoma3.Adenoid cystic carcinoma4.Hamartoma5.Leiomyoma6.Myoblastoma7.Mucous gland adenoma8.Squamous cell carcinoma

Notes:

Bronchial Obstruction 1.Foreign body: most commonly in young children2.Granulomatous disease: due to granuloma formation in bronchial wall / extrinsic compression by adenopathy3.Broncholiths = erosion of calcified nodes into bronchial lumen4.Stenosis / atresia5.Neoplasm(a)Bronchogenic carcinoma(b)Adenoid cystic carcinoma(c)Mucoepidermoid tumor(d)Hamartoma *mnemonic:*"MEATFACE"**M**ucus plug **E**ndobronchial granulomatous disease **A**denoma **T**uberculosis **F**oreign body **A**myloid, **A**tresia (bronchial) **C**ancer (primary) **E**ndobronchial metastasis

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Notes:

Mucoid Impaction =BRONCHIAL MUCOCELE = BRONCHOCELE=accumulation of inspissated secretions (mucus / pus / inflammatory products) within bronchial lumen; usually associated with bronchial dilatationA.WITH BRONCHIAL OBSTRUCTION in the presence of collateral air drift1.Bronchial obstruction by neoplasm:bronchogenic carcinoma / adenoma 2.Bronchial atresiaB.WITHOUT BRONCHIAL OBSTRUCTION1.Asthma (most frequent cause): esp. during acute attack or convalescent phase2.Fluid-filled bronchiectasis: history of childhood pneumonia; peripheral distribution3.Bronchopulmonary aspergillosis: central perihilar bronchiectasis4.Cystic fibrosis5.Chronic bronchitis

Notes:

Signet-ring Sign =ring of opacity in association with smaller round soft-tissue opacity (usually thick-walled bronchus + adjacent pulmonary artery / dilated bronchial artery)1.Bronchiectasis2.Multifocal bronchioloalveolar carcinoma3.Metastatic adenocarcinoma

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Notes:

Radiology Review Manual
fourth edition

HRCT Classification Of Bronchiolar Disease [CT findings are nonspecific and must be interpreted in the appropriate clinical context] √ nodules and branching lines1.Acute infectious bronchiolitis in infants and young children (RSV, adenovirus, Mycoplasma)2.Diffuse panbronchiolitis in Orientals3.Chronic inflammation: asthma, chronic bronchitis, bronchiectasis√ ground-glass attenuation and consolidation1.BOOP2.Respiratory bronchiolitis = smokers bronchiolitis√ low attenuation and mosaic perfusion1.Constrictive bronchiolitis2.Swyer-James syndrome√ bronchiolocentric infiltrates1.Extrinsic allergic alveolitis2.Sarcoidosis (perivenular nodules)3.Pneumoconiosis: asbestosis, silicosis

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Notes:

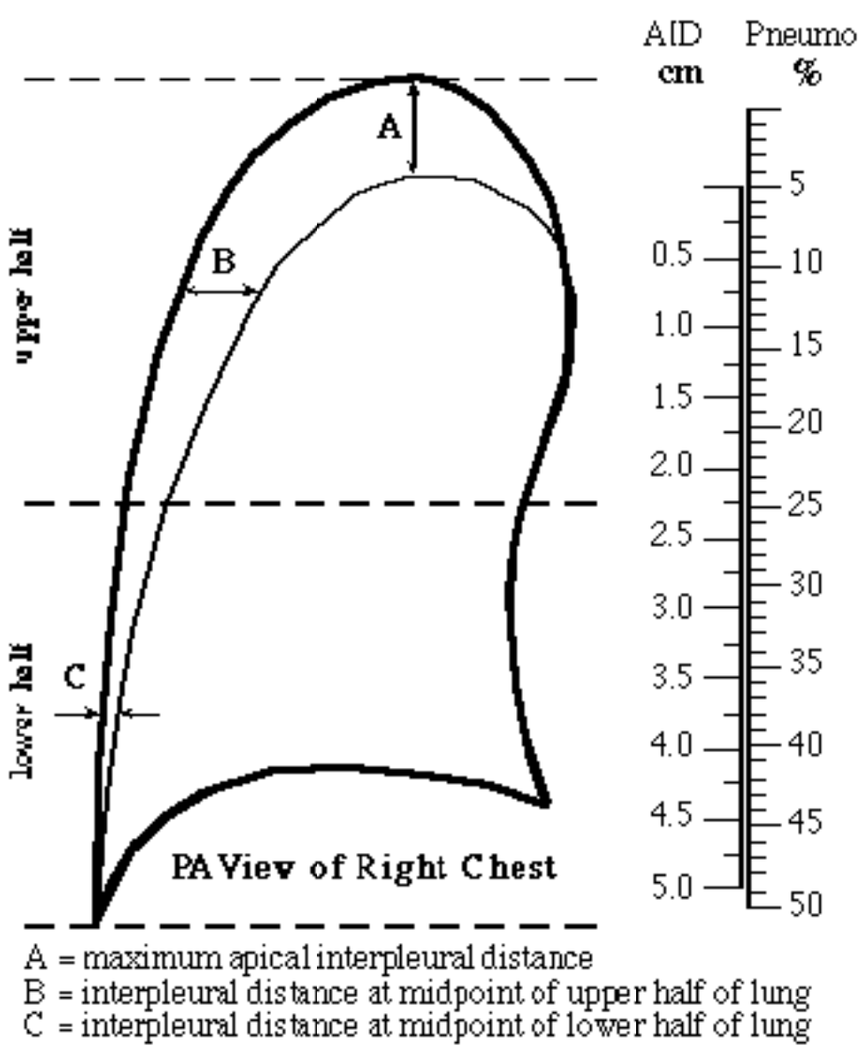
Bronchial Wall Thickening ◊Apparent thickness of bronchial wall varies with lung window chosen on CT: a mean window that is too low can make bronchial wall appear abnormal!A.PERIBRONCHOVASCULAR1.Sarcoidosis2.Lymphangitic carcinomatosis3.Kaposi sarcoma4.Lymphoma5.Pulmonary edemaB.BRONCHIAL WALL1.Airway diseaseC.MUCOSA

Notes:

Broncholithiasis 1.Histoplasmosis2.Tuberculosis3.Cryptococcosis4.Actinomycosis5.Coccidioidomycosis√ calcified lymph node within / adjacent to affected bronchus √ bronchial obstruction: atelectasis, airspace disease, bronchiectasis, air trapping√ absence of associated soft-tissue mass

Notes:

Pneumothorax =accumulation of air in the pleural space*Pathophysiology:*disruption of visceral pleura / trauma to parietal pleura ● pleuritic back / shoulder pain, dyspnea (in 80-90%)*Etiology:* 1.Penetrating trauma2.Blunt trauma(a)rib fracture(b)increased intrathoracic pressure against closed glottis: lung contusion / laceration(c)bronchial rupture√ fallen lung sign = hilum of lung below expected level within chest cavity√ persistent pneumothorax with functioning chest tube√ mediastinal pneumothorax3.Iatrogenictracheostomy, central venous catheter, PEEP ventilator (3-16%), thoracic irradiation 4.**Primary / idiopathic spontaneous pneumothorax**
*Cause:*rupture of subpleural blebs in apical region of lung*Age:*20-40 years; M:F = 8:1; esp. in patients with tall asthenic stature; mostly in smokers ● chest pain (69%) ● dyspnea*Prognosis:*recurrence in 30% on same side, in 10% on contralateral side*Rx:*simple aspiration (in >50% success) / tube thoracostomy (in 90% effective)5.Other causes:(a)Neonatal disease: meconium aspiration, respirator therapy for hyaline membrane disease(b)Malignancy: primary lung cancer, lung metastases (esp. osteosarcoma, pancreas, adrenal, Wilms tumor)(c)Pulmonary infections: tuberculosis, necrotizing pneumonia, coccidioidomycosis, hydatid disease, pertussis, acute bacterial pneumonia, staphylococcal septicemia, AIDS (Pneumocystis carinii, Mycobacterium tuberculosis, atypical mycobacteria)(d)Cx of honeycomb lung: pulmonary fibrosis, cystic fibrosis, sarcoidosis, scleroderma, eosinophilic granuloma, interstitial pneumonitis, histiocytosis X, rheumatoid lung, idiopathic pulmonary hemosiderosis, pulmonary alveolar proteinosis, biliary cirrhosis(e)Spasmodic asthma, diffuse emphysema√Chronic obstructive pulmonary disease is the most common predisposing disorder of secondary spontaneous pneumothorax(f)**Catamenial pneumothorax** = recurrent spontaneous pneumothorax during menstruation associated with endometriosis of the diaphragm; R >> L(g)Marfan syndrome, Ehlers-Danlos syndrome(h)Pulmonary infarction(i)Lymphangiomyomatosis + tuberous sclerosis*mnemonic:*"THE CHEST SET"**T**rauma **H**oneycomb lung, **H**amman-Rich syndrome **E**mphysema, **E**sophageal rupture **C**hronic obstructive pulmonary disease **H**yaline membrane disease **E**ndometriosis **S**pontaneous, **S**cleroderma **T**uberous sclerosis **S**arcoma (osteo-), **S**arcoidosis **E**osinophilic granuloma **T**uberculosis + fungus *Types:* 1.Closed pneumothorax = intact thoracic cage2.Open pneumothorax = "sucking" chest wound3.**Tension pneumothorax**
= accumulation of air within pleural space due to free ingress + limited egress of air *Pathophysiology:* intrapleural pressure exceeds atmospheric pressure in lung during expiration (check-valve mechanism) *Frequency:*in 3-5% of patients with spontaneous pneumothorax, higher in barotrauma√ displacement of mediastinum / anterior junction line√ deep sulcus sign = on frontal view larger lateral costodiaphragmatic recess than on opposite side√ diaphragmatic inversion√ total / subtotal lung collapse√ collapse of SVC / IVC / right heart border (decreased systemic venous return + decreased cardiac output)N.B.:Medical emergency!4.**Tension hydropneumothorax**
√ sharp delineation of visceral pleural by dense pleural space√ mediastinal shift to opposite side√ air-fluid level in pleural space on erect CXR PNEUMOTHORAX SIZE
Average Interpleural Distance (AID) = (A + B + C) ÷ 3 [in cm] converts to percentage of pneumothorax



A = maximum apical interpleural distance
B = interpleural distance at midpoint of upper half of lung
C = interpleural distance at midpoint of lower half of lung

Radiographic signs in upright position: √ white margin of visceral pleura separated from parietal pleuraDDx: skin fold, air trapped between chest wall soft tissues, hair braid) √ absence of vascular markings beyond visceral pleural margin *Radiographic signs in supine position:* 1.Anteromedial pneumothorax (earliest location)√ outline of medial diaphragm under cardiac silhouette√ sharp delineation of mediastinal contours (SVC, azygos vein, left subclavian artery, anterior junction line, superior pulmonary vein, heart border, IVC, deep anterior cardiophrenic sulcus, pericardial fat-pad)2.Subpulmonic pneumothorax (second most common location)√ hyperlucent upper abdominal quadrant√ deep lateral costophrenic sulcus√ sharply outlined diaphragm in spite of parenchymal disease√ visualization of anterior costophrenic sulcus√ visualization of inferior surface of lung3.Apicolateral pneumothorax (least common location)√ visualization of visceral pleural line4.Posteromedial pneumothorax (in presence of lower lobe collapse)√ lucent triangle with vertex at hilum√ V-shaped base delineating costovertebral sulcus5.Pneumothorax outlines pulmonary ligament *Prognosis:*resorption of pneumothorax occurs at a rate of 1.25% per day (accelerated by increasing inspired oxygen concentrations)

Notes:

Pleural Effusion A.TRANSUDATE (protein level of 1.5-2.5 g/dL)*Pathophysiology:*result of systemic abnormalities causing an outpouring of low-protein fluid(a)Increased hydrostatic pressure1.Congestive heart failure (in 65%)bilateral (88%); right-sided (8%); left-sided (4%); least amount on left side due to cardiac movement, which stimulates lymphatic resorption 2.Constrictive pericarditis (in 60%)(b)Decreased colloid-oncotic pressure-decreased protein production1.Cirrhosis with ascites (in 6%):right-sided (67%)-protein loss / hypervolemia1.Nephrotic syndrome (21%), overhydration, glomerulonephritis (55%), peritoneal dialysis2.Hypothyroidism(c)Chylous effusion◊Most frequent cause of isolated pleural effusion in newborn with 15-25% mortality! ● chylomicrons + lymphocytes in fluid B.EXUDATE*Pathophysiology:*increased permeability of abnormal pleural capillaries with release of high-protein fluid into pleural space*Criteria:* ● pleural fluid total protein / serum total protein ratio of >0.5 ● pleural fluid LDH / serum LDH ratio of >0.6 ● pleural fluid LDH >2/3 of upper limit of normal for serum LDH (upper limit for LDH ~200 IU) ● pleural fluid specific gravity >1.016 ● protein level >3 g/dL √ effusion with septation / low-level echoes√ "split pleura" sign on CECT = thickened enhancing visceral + parietal pleura separated by fluid√ extrapleural fat thickening of >2 mm + increased attenuation (edema / inflammation) (a)Infection1.**Empyema** =parapneumonic effusion characterized by presence of pus ± positive culture-*exudative phase* = inflammation of visceral pleura results in increased capillary permeability with weeping of high-protein fluid into pleural space-*fibrinopurulent phase* = inflammatory cells + neutrophils pour into pleural space + fibrin deposition on pleural surfaces-*organizing phase* = recruitment of fibroblasts + capillaries results in deposition of collagen + granulation tissue on pleural surfaces = pleural fibrosis*Rx:*decortication if active infection persists *Organism:*S. aureus, gram-negative + anaerobic bacteria ● positive Gram stain ● positive culture (anaerobic bacteria most frequent) ● gross pus (WBC >15, 000/cm^3) ● pH <7.0 ● LDH >1000 IU/L ● glucose <40 mg/dL2.Parapneumonic effusion (in 40%)=any effusion associated with pneumonia / lung abscess / bronchiectasis without criteria for an empyema3.Tuberculosis (in 1%):high protein content (75 g/dL), lymphocytes >70%, positive culture (only in 20-25%) 4.Fungi: Actinomyces, Nocardia5.Parasites: amebiasis (secondary to liver abscess in 15-20%), Echinococcus6.Mycoplasma, rickettsia (in 20%) **empyema necessitatis** = chronic empyema attempting to decompress through chest wall (in TB, actinomycosis, aspergillosis, blastomycosis, nocardiosis) (b)Malignant disease (in 60%) ● positive cytologic results*Cause:*lung cancer (26-49%), breast cancer (8-24%), lymphoma (10-28%, in 2/3 chylothorax), ovarian cancer (10%), malignant mesothelioma containing hyaluronic acid (5%)*Pathogenesis:* -pleural metastases (increase pleural permeability)-lymphatic obstruction (pleural vessels, mediastinal nodes, thoracic duct disruption)-bronchial obstruction (loss of volume + resorptive surface)-hypoproteinemia (secondary to tumor cachexia)*Rx:*sclerosing agents: doxycycline, bleomycin, talc (c)VascularPulmonary emboli (in 15-30% of all embolic events): often serosanguinous (d)Abdominal disease1.Pancreatitis / pancreatic pseudocyst / pancreaticopleural fistula (in 2/3):√ usually left-sided pleural effusion ● high amylase levels2.Boerhaave syndrome:left-sided esophageal perforation 3.Subphrenic abscess√ pleural effusion (79%)√ elevation + restriction of diaphragmatic motion (95%)√ basilar platelike atelectasis / pneumonitis (79%)4.Abdominal tumor with ascites5.**Meigs-Salmon syndrome** =primary pelvic neoplasms (ovarian fibroma, thecoma, granulosa cell tumor, Brenner tumor, cystadenoma, adenocarcinoma, fibromyoma of uterus) cause pleural effusion in 2-3%; ascites + hydrothorax resolve with tumor removal6.Endometriosis7.Bile fistula (e)Collagen-vascular disease1.Rheumatoid arthritis (in 3%):unilateral; R > L (in 75%), recurrent alternating sides; pleural effusion relatively unchanged in size for months; predominantly in men; LOW GLUCOSE content of 20-50 mg/dL (in 70-80%) without increase following IV infusion of glucose (DDx: TB, metastatic disease, parapneumonic effusion) 2.SLE (in 15-74%)most common collagenosis to give pleural effusion, bilateral in 50%; L > R √ enlargement of cardiovascular silhouette (in 35-50%)3.Wegener granulomatosis (in 50%)4.Sjögren syndrome5.Mixed connective tissue disease6.Periarteritis nodosa7.Postmyocardial infarct syndrome (f)Traumatichemorrhagic, chylous, esophageal rupture, thoracic / abdominal surgery, intrapleural infusion = "infusothorax" (0.5%), radiation pneumonitis (g)Miscellaneous1.Sarcoidosis2.Uremic pleuritis (in 20% of uremic patients)3.Drug-induced effusion CXR: √ first 300 ml not visualized on PA view (collect in subpulmonic region first, then spill into posterior costophrenic sinus)√ lateral decubitus views may detect as little as 25 ml√ hemidiaphragm + costophrenic sinuses obscured√ extension upward around posterior > lateral > anterior thoracic wall (mediastinal portion fixed by pulmonary ligament + hilum)√ meniscus-shaped semicircular upper surface with lowest point in midaxillary line√ associated collapse of ipsilateral lung Massive pleural effusion: √ enlargement of ipsilateral hemithorax√ displacement of mediastinum to contralateral side√ severe depression / flattening / inversion of ipsilateral hemidiaphragm√ visible air bronchogramSubpulmonic effusionSubpulmonic / subdiaphragmatic / infrapulmonary pleural effusion: √ peak of dome of pseudodiaphragm laterally positioned√ acutely angulated costophrenic angle√ increased distance between stomach bubble and lung√ blunted posterior costophrenic sulcus√ thin triangular paramediastinal opacity (mediastinal extension of pleural effusion)√ flattened pseudodiaphragmatic contour anterior to major fissure (on lateral CXR)CT: √ fluid outside diaphragm√ fluid elevating crus of diaphragm√ indistinct fluid-liver interface√ fluid posteromedial to liver (= bare area of liver)CAVE:"central oval" sign of ascites may be seen in subpulmonic effusion with inverted diaphragm ***Unilateral Pleural Effusion*** ◊The majority of massive unilateral pleural effusions are malignant (lymphoma, metastatic disease, primary lung cancer)!1.Neoplasm2.Infection: TB3.Collagen vascular disease4.Subdiaphragmatic disease5.Pulmonary emboli6.Trauma: fractured rib7.Chylothorax ***Left-sided Pleural Effusion*** 1.Spontaneous rupture of the esophagus2.Dissecting aneurysm of the aorta3.Traumatic rupture of aorta distal to left subclavian artery4.Transection of distal thoracic duct5.Pancreatitis: left-sided (68%), right-sided (10%), bilateral (22%)6.Pancreatic + gastric neoplasm***Right-sided Pleural Effusion*** 1.Congestive heart failure2.Transection of proximal thoracic duct3.Pancreatitis***Pleural Effusion & Large Cardiac Silhouette*** 1.Congestive heart failure (most common)√ cardiomegaly√ prominence of upper lobe vessels + constriction of lower lobe vessels√ prominent hilar vessels√ interstitial edema (fine reticular pattern, Kerley lines, perihilar haze, peribronchial thickening)√ alveolar edema (perihilar confluent ill-defined densities, air bronchogram)√ "phantom tumor" = fluid localized to interlobar pleural fissure (in 78% in right horizontal fissure)2.Pulmonary embolus with right-sided heart enlargement3.Myocarditis / pericarditis with pleuritis(a)viral infection(b)tuberculosis(c)rheumatic fever (poststreptococcal infection)4.Tumor: metastatic, mesothelioma5.Collagen-vascular disease(a)SLE (pleural + pericardial effusion)(b)rheumatoid arthritis
Pleural Effusion & Subsegmental Atelectasis 1.Postoperative (thoracotomy, splenectomy, renal surgery) secondary to thoracic splinting + small airway mucous plugging2.Pulmonary embolus3.Abdominal mass4.Ascites5.Rib fractures ***Pleural Effusion & Lobar Densities*** 1.Pneumonia with empyema2.Pulmonary embolism3.Neoplasm(a)bronchogenic carcinoma (common)(b)lymphoma4.Tuberculosis ***Pleural Effusion & Hilar Enlargement*** 1.Pulmonary embolus2.Tumor(a)bronchogenic carcinoma(b)lymphoma(c)metastasis3.Tuberculosis4.Fungal infection (rare)5.Sarcoidosis (very rare)

Notes:

Hemothorax A.TRAUMA1.Closed / penetrating injury2.Surgery3.Interventional procedures: thoracentesis, pleural biopsy, catheter placementB.BLEEDING DIATHESIS1.Anticoagulant therapy2.Thrombocytopenia3.Factor deficiencyC.VASCULAR1.Pulmonary infarct2.Arteriovenous malformation3.Aortic dissection4.Leaking atherosclerotic aneurysmD.MALIGNANCY1.Mesothelioma2.Lung cancer3.Metastasis4.LeukemiaE.OTHER1.Catamenial hemorrhage2.Extramedullary hematopoiesis √ rapidly enlarging high-attenuation pleural effusion on CT √ heterogeneous attenuation √ hyperattenuating areas of debris √ fluid-hematocrit level

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Notes:

Solitary Pleural Mass =density with incomplete border and tapered superior + inferior borders, difficult to distinguish from chest wall mass (rib destruction reliable sign of chest wall mass) 1.Loculated pleural effusion ("vanishing tumor")2.Organized empyema3.Metastasis4.Local benign mesothelioma5.Subpleural lipoma: may erode adjacent rib6.Hematoma7.Mesothelial cyst8.Neural tumor: schwannoma, neurofibroma9.**Localized fibrous tumor of pleura**
10. **Fibrin bodies**
= 3-4 cm large tumorlike concentrations of fibrin forming in serofibrinous pleural effusions; usually near lung base

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Notes:

Multiple Pleural Densities √ diffuse pleural thickening with lobulated borders 1.Loculated pleural effusion:infectious, hemorrhagic, neoplastic 2.Pleural plaques3.Metastasis (most common cause)*Origin:*lung (40%), breast (20%), lymphoma (10%), melanoma, ovary, uterus, GI tract, pancreas, sarcoma◊Metastatic adenocarcinoma histologically similar to malignant mesothelioma!4.Diffuse malignant mesotheliomaalmost always unilateral, associated with asbestos exposure 5.Invasive thymoma (rare)√ contiguous spread, invasion of pleura, spreads around lung√ NO pleural effusion6.**Thoracic splenosis**
=autotransplantation of splenic tissue to pleural space following thoracoabdominal trauma; discovered 10-30 years later ● asymptomatic / recurrent hemoptysis√ one or several nodules in left pleura / fissures measuring several mm to 6 cm√ positive Tc-99m-sulfur colloid scan, indium-111-labeled platelets, Tc-99m-labeled heat-damaged RBCs *mnemonic:*"**M**ary **T**yler **M**oore **L**ikes **L**emon"**M**etastases (especially adenocarcinoma) **T**hymoma (malignant) **M**alignant mesothelioma **L**oculated pleural effusion **L**ymphoma

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Notes:

Pleural Thickening A.TRAUMA1.**Fibrothorax** (most common cause)=organizing effusion / hemothorax / pyothorax√ dense fibrous layer of approx. 2 cm thickness; almost always on visceral pleura√ frequent calcification on inner aspect of pleural peelB.INFECTION1.Chronic empyema: over bases; history of pneumonia; parenchymal scars2.Tuberculosis / histoplasmosis: lung apex; associated with apical cavity3.Aspergilloma: in preexisting cavity concomitant with pleural thickeningC.COLLAGEN-VASCULAR DISEASE1.Rheumatoid arthritis: pleural effusion fails to resolveD.INHALATIONAL DISORDER1.Asbestos exposure: lower lateral chest wall; basilar interstitial disease (<25%); thickening of parietal pleura with sparing of visceral pleura2.TalcosisE.NEOPLASM(a)Metastases: often nodular appearance; may be obscured by effusion(b)Diffuse malignant mesothelioma(c)Pancoast tumorF.OTHER1.**Pleural hyaloserositis**
*Path:*hyaline sclerotic tissue = cartilagelike whitish sugar icing appearance (Zuckerguss) with occasional calcification2.Mimicked by extrathoracic musculature, 1st + 2nd rib companion shadow, subpleural fat, focal scarring around old rib fractures *mnemonic:*"TRINI"**T**rauma (healed hemothorax) **R**heumatoid arthritis (collagen vascular disease) **I**nhalation disease (asbestosis, talcosis) **N**eoplasm **I**nfection

Notes:

Apical Cap 1.Inflammatory process: TB, healed empyema2.Postradiation fibrosis3.Neoplasm4.Vascular abnormality5.Mediastinal hemorrhage6.Mediastinal lipomatosis7.Peripheral upper lobe collapse

Notes:

Pleural Calcification A.INFECTION1.Healed empyema2.Tuberculosis (and Rx for TB: pneumothorax / oleothorax), histoplasmosisB.TRAUMA1.Healed hemothorax = fibrothorax: ● Hx of significant chest trauma√ irregular plaques of calcium usually in visceral pleura√ healed rib fracture2.Radiation therapyC.PNEUMOCONIOSIS1.Asbestos-related pleural disease (most common):√ combination of basilar reticular interstitial disease (<1/3) + pleural thickening√ calcifications of parietal pleura frequently diagnostic (diaphragmatic surface of pleura, bilateral but asymmetric)2.Talcosis: similar to asbestos-related disease3.Bakelite4.Muscovite micaD.HYPERCALCEMIA1.Pancreatitis2.Secondary hyperparathyroidism in chronic renal failure / sclerodermaE.MISCELLANEOUS1.Mineral oil aspiration2.Pulmonary infarction *mnemonic:*"TAFT"**T**uberculosis **A**sbestosis **F**luid (effusion, empyema, hematoma) **T**alc

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Notes:

Bilateral Diaphragmatic Elevation A.Shallow inspiration (most frequent)B.Abdominal causesObesity, pregnancy, ascites, large abdominal mass C.Pulmonary causes(1) Bilateral atelectasis (2) Restrictive pulmonary disease (SLE) D.Neuromuscular disease(1) Myasthenia gravis (2) Amyotrophic lateral sclerosis

Notes:

Unilateral Diaphragmatic Elevation 1.Subpulmonic pleural effusion√ dome of pseudodiaphragm migrates toward the costophrenic angle and flattens2.Altered pulmonary volume(a)Atelectasis√ associated pulmonary density(b)Postoperative lobectomy / pneumonectomy√ rib defects, metallic sutures(c)Hypoplastic lung√ small hemithorax (more often on the right), crowding of ribs, mediastinal shift, absent / small pulmonary artery, frequently associated with dextrocardia + anomalous pulmonary venous return3.Phrenic nerve paralysis(a)Primary lung tumor(b)Malignant mediastinal tumor(c)Iatrogenic(d)Idiopathic√ paradoxic motion on fluoroscopy (patient in lateral position sniffing)4.Abdominal disease(a)Subphrenic abscess: history of surgery, accompanied by pleural effusion(b)Distended stomach / colon(c)Interposition of colon(d)Liver mass (tumor, echinococcal cyst, abscess)5.Diaphragmatic hernia6.Eventration of diaphragm7.Traumatic rupture of diaphragmAssociated with rib fractures, pulmonary contusion, hemothorax 8.Diaphragmatic tumorMesothelioma, fibroma, lipoma, lymphoma, metastases

Notes:

Chest Wall Lesions A.EXTERNAL1.Cutaneous lesion: moles, neurofibroma2.Nipples3.ArtifactB.NEOPLASTIC1.Mesenchymal tumor(a)Lipoma (common): growing between ribs presenting as intrathoracic + subcutaneous mass; CT diagnostic)(b)Muscle tumor, fibroma2.Neural tumorSchwannoma, neurofibroma (may erode ribs inferiorly with sclerotic bone reaction), neuroma, neuroblastoma 3.Vascular tumorHemangioma, lymphangioma, hemangiopericytoma, aneurysm, false aneurysm 4.Bone tumor *(see also Rib lesion)*
C.TRAUMATIC1.Hematoma2.Rib fractureD.INFECTIOUScellulitis, pyomyositis, abscess, necrotizing fasciitis 1.Actinomycosis (parenchymal infiltrate, pleural effusion, chest wall mass, rib destruction, cutaneous fistulas)2.Aspergillosis, nocardiosis, blastomycosis, tuberculosis (rare)3.Pyogenic: Staphylococcus, KlebsiellaE.CHEST WALL INVASION1.Peripheral lung cancer (eg, Pancoast tumor)2.Recurrent breast cancer3.Lymphomatous nodes√ incomplete border sign (due to obtuse angle)√ smooth tapering borders (tangential views)√ tumor pedicle suggests a benign tumor

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Notes:

Lung Disease With Chest Wall Extension A.Infectious1.Actinomycosis2.Nocardia3.Blastomycosis4.TuberculosisB.Malignant tumor1.Bronchogenic carcinoma2.Lymphoma3.Metastases4.Mesothelioma5.Breast carcinoma6.Internal mammary nodeC.Benign tumor1.Capillary hemangioma of infancy2.Cavernous hemangioma3.Extrapleural lipoma4.Abscess5.Hematoma

Notes:

Malignant Tumors Of Chest Wall In Children 1.Ewing sarcoma of rib (most common)(a)older child: rib involvement in 7%, predominant involvement of pelvis + lower extremity(b)child <10 years:rib involvement in 30%2.Rhabdomyosarcomarelatively common in children + adolescents √ sclerosis / destruction / scalloping of cortex (local extension to contiguous bone)√ may calcifyMetastases to: lung, occasionally lymph nodes*Prognosis:*infiltrative growth with high risk of local recurrence3.Neuroblastoma10% present as chest wall mass √ may calcify4.**Askin tumor**
=uncommon tumor probably arising from intercostal nerves in young Caucasian females*Path:*neuroectodermal small cell tumor containing neuron-specific enolase (may also be found in neuroblastoma)√ rib destruction√ pleural effusionMetastases to: bone, CNS, liver, adrenal *DDx:*Chest wall hamartoma in infancy

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Notes:

Pancoast Syndrome =superior sulcus tumor invading brachial plexus + sympathetic stellate ganglionCLINICAL TRIAD: 1.Ipsilateral arm pain2.Muscle wasting of hand3.Horner syndrome = enophthalmos, ptosis, miosis, anhidrosis*Cause:*lung cancer (most common), breast cancer, multiple myeloma, metastases, lymphoma, mesothelioma

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Notes:

PULMONARY MALFORMATION
=SEQUESTRATION SPECTRUM1.Congenital lobar emphysema2.Bronchogenic cyst3.Congenital cystic adenomatoid malformation4.Bronchopulmonary sequestration5.Hypogenetic lung syndrome6.Pulmonary arteriovenous malformation

Notes:

Mediastinal Shift & Abnormal Aeration A.SHIFT TOWARD LUCENT LUNG1.Diaphragmatic hernia2.Chylothorax3.Cystic adenomatoid malformationB.SHIFT AWAY FROM LUCENT LUNG1.Congenital lobar emphysema2.Persistent localized pulmonary interstitial emphysema3.Obstruction of main-stem bronchus (by anomalous or dilated vessel / cardiac chamber)

Notes:

Reticulogranular Densities In Neonate 1.Respiratory distress syndrome (90%): premature infant, inadequate surfactant2.Immature lung: premature infant, normal surfactant3.Transient tachypnea of the newborn4.Neonatal group-B streptococcal pneumonia5.Idiopathic hypoglycemia6.Congestive heart failure7.Early pulmonary hemorrhage8.Infant of diabetic mother

Notes:

Hyperinflation In Newborn 1.Fetal aspiration syndrome2.Neonatal pneumonia3.Pulmonary hemorrhage4.Congenital heart disease5.Transient tachypnea (mild)

Notes:

Hyperinflation In Child *mnemonic:*"BUMP FAD"**B**ronchiectasis **U**pper airway obstruction **M**ucoviscidosis **P**neumonia (esp. staph) **F**oreign body (ball-valve mechanism) **A**sthma **D**ehydration (diarrhea, acidosis)

Notes:

PULMONARY HEMORRHAGE
A.WITHOUT RENAL DISEASE1.Bleeding diathesis: leukemia2.Anticoagulation therapy3.Disseminated intravascular coagulation4.Blunt trauma5.Idiopathic pulmonary hemosiderosis6.Limited Wegener granulomatosis7.Infectious diseases8.Exogenous agents: D-penicillamine, lymphangiographyB.WITH RENAL DISEASE1.Goodpasture syndrome = anti-basement membrane antibody disease2.Collagen vascular disease + systemic vasculitides: SLE, Wegener granulomatosis, polyarteritis nodosa, Henoch-Schönlein purpura, Behçet disease3.Rapidly progressive glomerulonephritis ± immune complexesC.HEMORRHAGIC PNEUMONIA1.Bacteria:Legionnaires disease2.Viruses:CMV, herpes3.Fungi:Aspergillosis, mucormycosis

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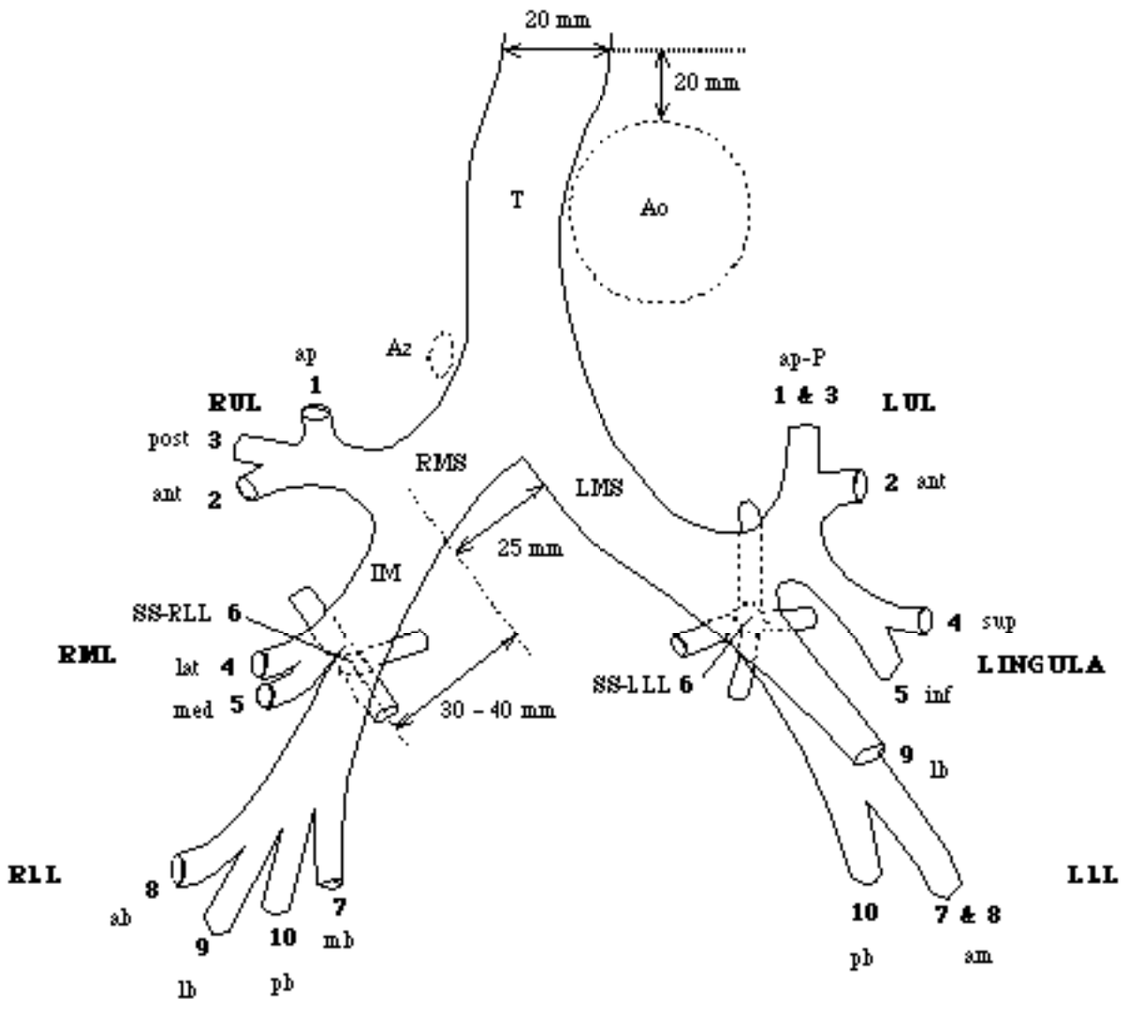
Notes:

BEDSIDE CHEST RADIOGRAPHY

Unexpected findings:in 37-43%Change in diagnostic approach / therapy: in 27% *Indications:* A.Apparatus position + complications1.Malposition of tracheal tube (12%)2.Malposition of central venous line (9%)B.Cardiopulmonary disease1.Congestive heart failure2.Pleural effusion3.Atelectasis4.Alveolar disease5.Air leak6.Lung trauma7.Thoracic bleeding8.Mediastinal disease

Notes:

BRONCHOPULMONARY ANATOMY



Bronchopulmonary Anatomy

Ao	=	aortic arch	RMS	=	right mainstem bronchus (2nd order bronchus)
Az	=	azygos vein	LMS	=	left mainstem bronchus
T	=	trachea (1st order bronchus)	IM	=	intermediate bronchus
SS-RLL	=	superior segment right lower lobe	SS-LLL	=	superior segment left lower lobe

RUL = right upper lobe
- 1 = apical
- 2 = anterior
- 3 = posterior

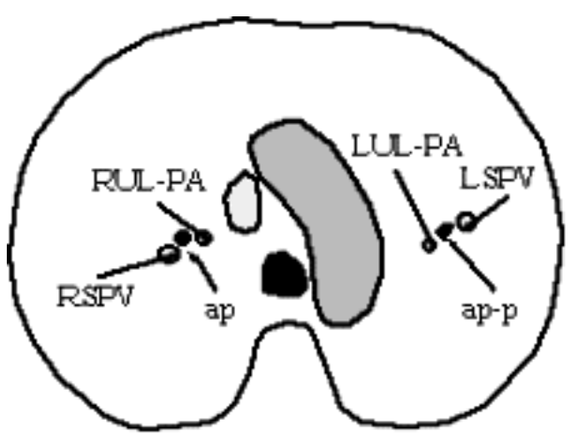
RML = right middle lobe
- 4 = lateral
- 5 = medial

RLL = right lower lobe
- 6 = superior
- 7 = mediobasal
- 8 = anterobasal
- 9 = lateobasal
- 10 = posterobasal

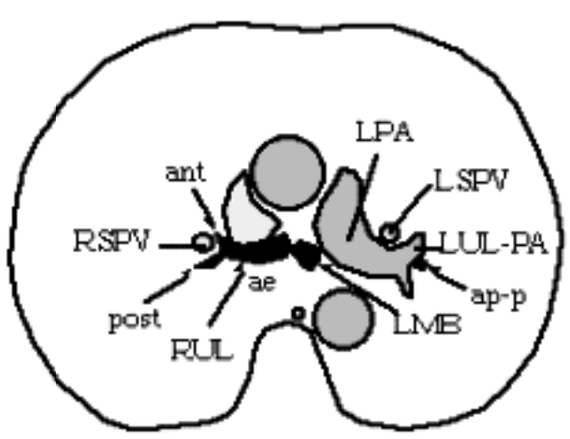
LUL = left upper lobe (3rd order bronchus)
- 1&3 = apicoposterior segment
- 2 = anterior (4th order bronchus)
- 4 = superior lingula
- 5 = inferior lingula

LLL = left lower lobe
- 6 = superior
- 7&8 = anteromedial
- 9 = lateobasal
- 10 = posterobasal

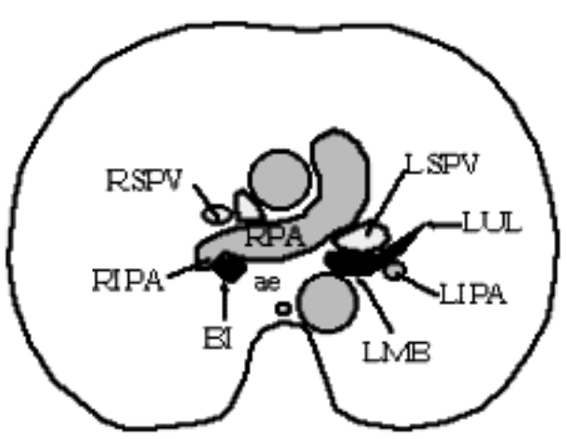
Order of lower lobe bronchi in frontal projection from lateral to medial:
mnemonic "ALPm" = Anterior-Lateral-Posterior-medial



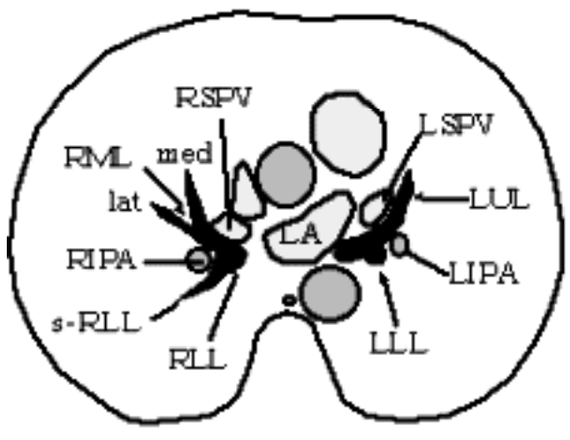
Level of apical segmental bronchus



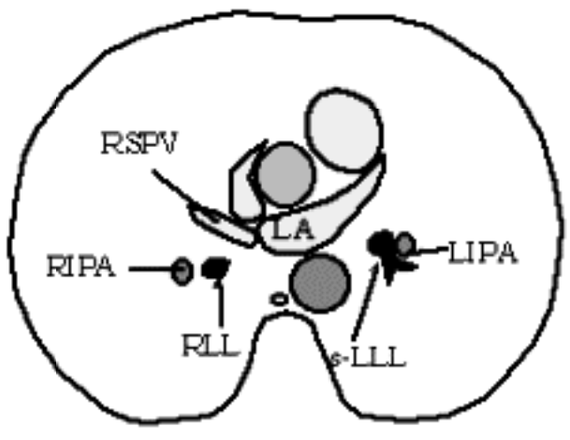
Level of right upper lobe bronchus



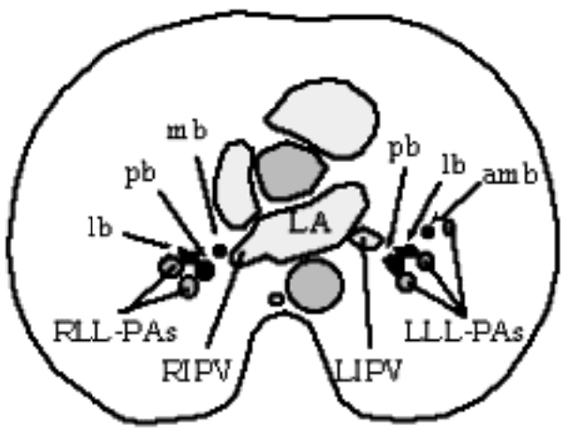
Level of bronchus intermedius



Level of right middle lobe bronchus



Level of left superior segmental bronchus

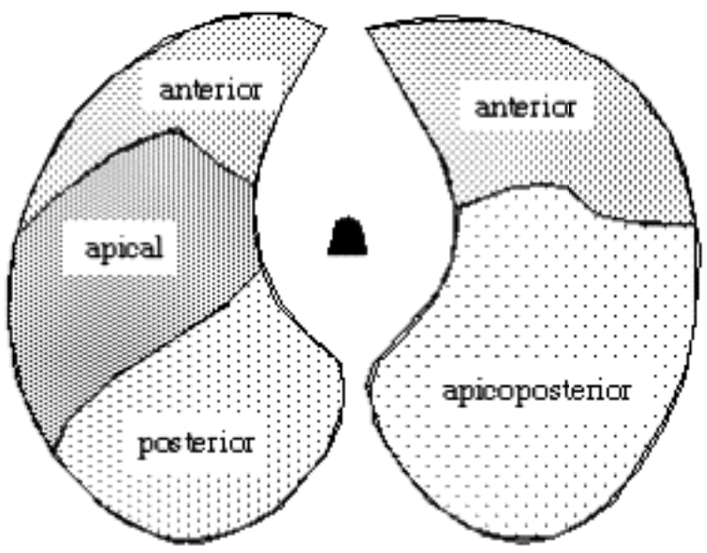


Level of lower lobe bronchi

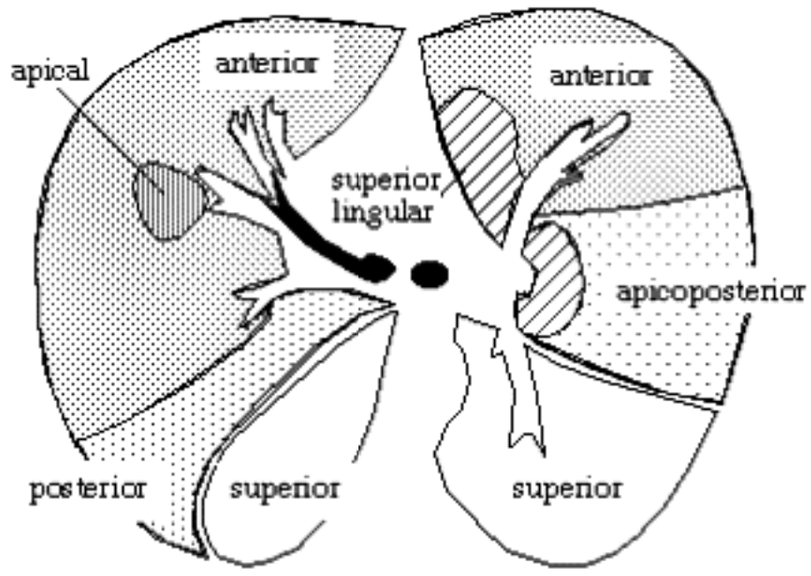
Right				Left			
ant	= anterior RUL	pb	= posterobasal RLL	amb	= anteromediobasal LLL	ap-p	= apicoposterior LUL
ap	= apical RUL	post	= posterior RUL	lb	= laterobasal LLL	LLL	= left lower lobe
BI	= bronchus intermedius	RLL	= right lower lobe	LMB	= left main bronchus	LUL	= left upper lobe
lat	= lateral RML	RML	= right middle lobe	pb	= posterobasal LLL	s-LLL	= superior segment
mb	= mediobasal RLL	RUL	= right upper lobe				
med	= medial RML	s-RLL	= superior segment	ae	= azygoesophageal recess		

RIPV / LIPV	= right / left inferior pulmonary vein	RIPA / LIPA	= right / left inferior pulmonary artery
RPA / LPA	= right / left pulmonary artery	RLL-PAs / LLL-PAs	= right / left lower lobe pulmonary arteries
RUL-PA / LUL-PA	= right / left upper lobe pulmonary artery	RSPV / LSPV	= right / left superior pulmonary vein

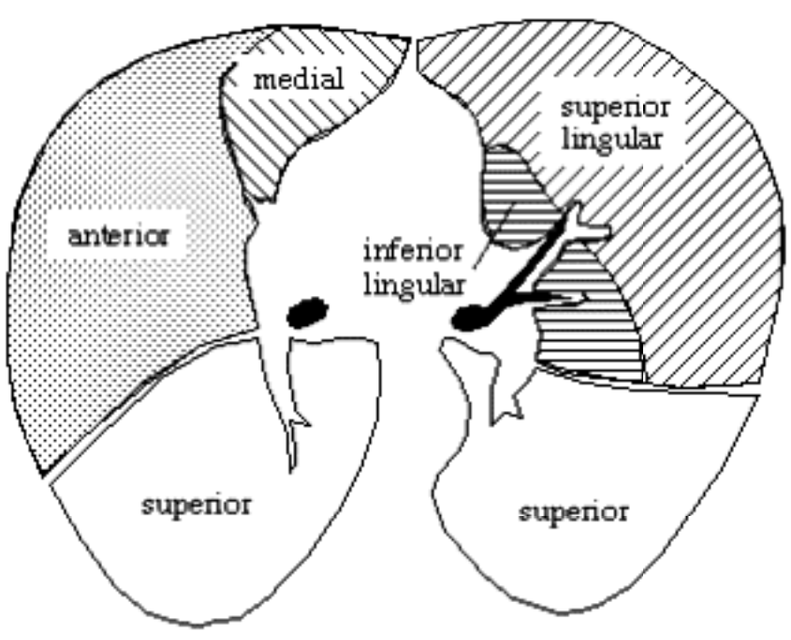
Cross-sectional Anatomy of Bronchovascular Divisions



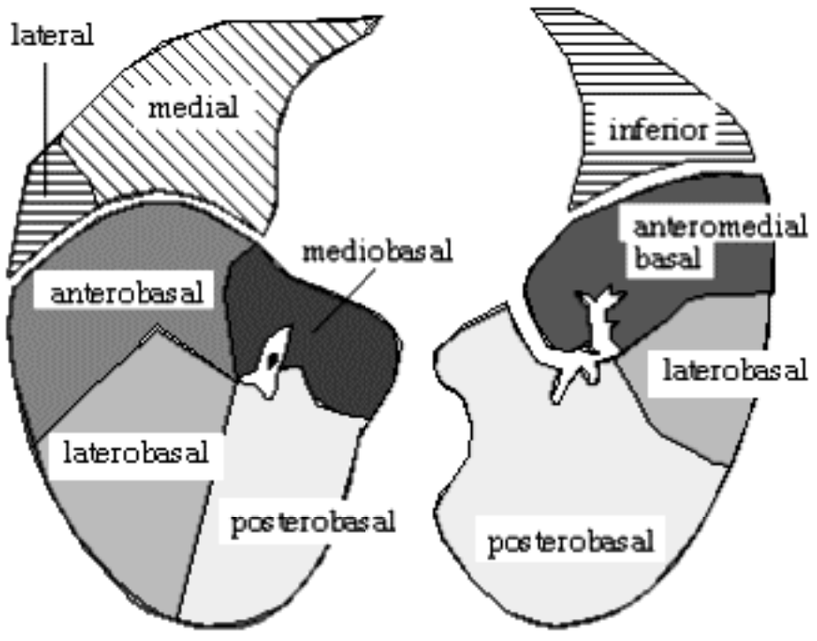
Aortic arch level



Left pulmonary artery level



Right pulmonary artery level



Cardiac ventricular level

Cross-sectional anatomy of lung segments

Notes:

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Embryology Of Airways first 5 weeks GAlung buds grow from ventral aspect of primitive foregut; *pulmonary agenesis*
5th week GAtrachea + esophagus separate5-16 weeksformation of tracheobronchial tree with bronchi, bronchioles, alveolar ducts, alveoli; *bronchogenic cyst* (= abnormal budding); *pulmonary hypoplasia* (= fewer than expected bronchi)16-24 weeksdramatic increase in number + complexity of airspaces and blood vessels; *small airways + reduction in number and size of acini*

Notes:

Airway =conducting branches for the transport of air;~300,000 branching airways from trachea to bronchiole with an average of 23 airway generations*Definition:* bronchus=cartilage in wallbronchiole=absence of cartilage-membranous bronchiole = purely air conducting-respiratory bronchiole = containing alveoli in their walls-lobular bronchiole = supplies secondary pulmonary lobule; may branch into 3 or more terminal bronchioles-terminal bronchiole = last generation of purely conducting bronchioles; each supplying one acinussmall airways=diameter <2 mm = small cartilaginous bronchi + membranous and respiratory bronchioles; account for 25% of airway resistancelarge airways=diameter >2 mm; account for 75% of airway resistanceHRCT of normal lung (window level -700 HU, window width 1,000-1,500): √ -875 ± 18 HU at inspiration;√ -620 ± 43 HU at expiration

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Notes:

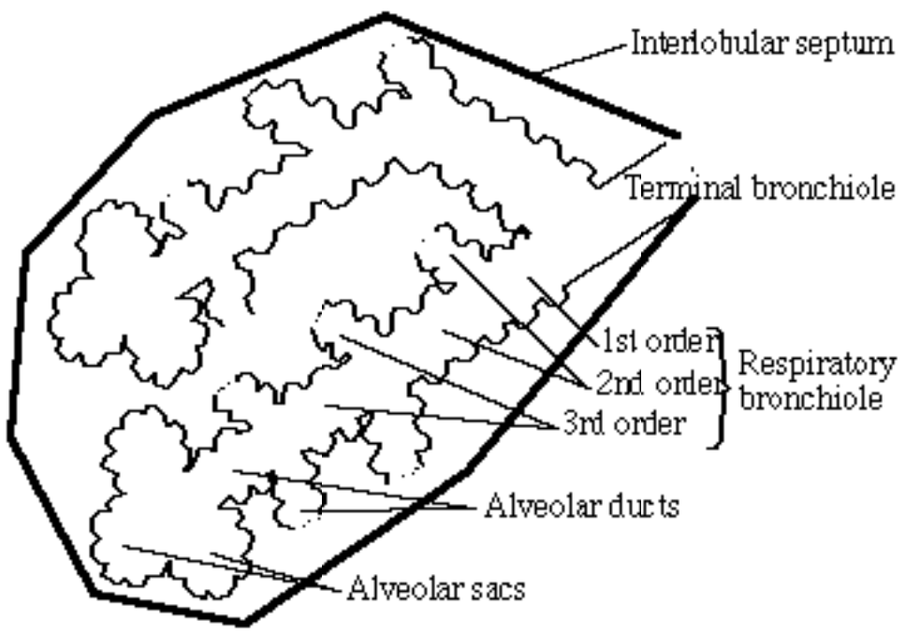
Acinus =functionally most important subunit of lung = all parenchymal tissue distal to one terminal bronchiole comprising 2-5 generations of respiratory bronchioles + alveolar ducts + alveolar sacs + alveoli√ radiologically not visible

Notes:

Primary Pulmonary Lobule =alveolar duct + air spaces connected with it

Notes:

Secondary Pulmonary Lobule =REID LOBULE=smallest portion of lung surrounded by connective tissue septa = basic anatomic + functional pulmonary unit appearing as an irregular polyhedron measuring 10-25 mm on each side; separated from each other by thin fibrous interlobular septa (100 µm); supplied by 3-5 terminal bronchioles; contains 3-24 aciniContents: -centrally = lobular core: branches of terminal bronchioles (0.1 mm wall thickness is below the resolution of HRCT) + pulmonary arterioles (1 mm)-peripherally (in interlobular septa): pulmonary veins + lymph vesselsHRCT: √ barely visible fine lines of increased attenuation in contact with pleura (= interlobular septa); best developed in subpleural areas of-UL + ML:anterior + lateral + juxtamediastinal-LL:anterior + diaphragmatic regions√ dotlike / linear / branching structures (= pulmonary arterioles) near center of secondary pulmonary lobule 3-5 mm from pleura



The Secondary Pulmonary Lobule

Notes:

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Surfactant =surface-active material essential for normal pulmonary functionSubstrate: phospholipids (phosphatidylcholine, phosphatidylglycerol), other lipids, cholesterol, lung-specific proteins Production: type II pulmonary alveoli synthesize + transport + secrete lung surfactant; earliest production around 18th week of gestation (in amniotic fluid by 22nd week of gestation) Action: increases lung compliance, stabilizes alveoli, enhances alveolar fluid clearance, reverses surface tension, protects against alveolar collapse during respiration, protects epithelial cell surface, reduces opening pressure + precapillary tone **LUNG INTERSTITIUM** *Division*Components axialbronchovascular sheathslymphatics middle (parenchymal)alveolar wall (interalveolar septum) peripheralpleurasubpleural connective tissueinterlobular septa (enclosing pulmonary veins, lymphatics, walls of cortical alveoli)

Notes:

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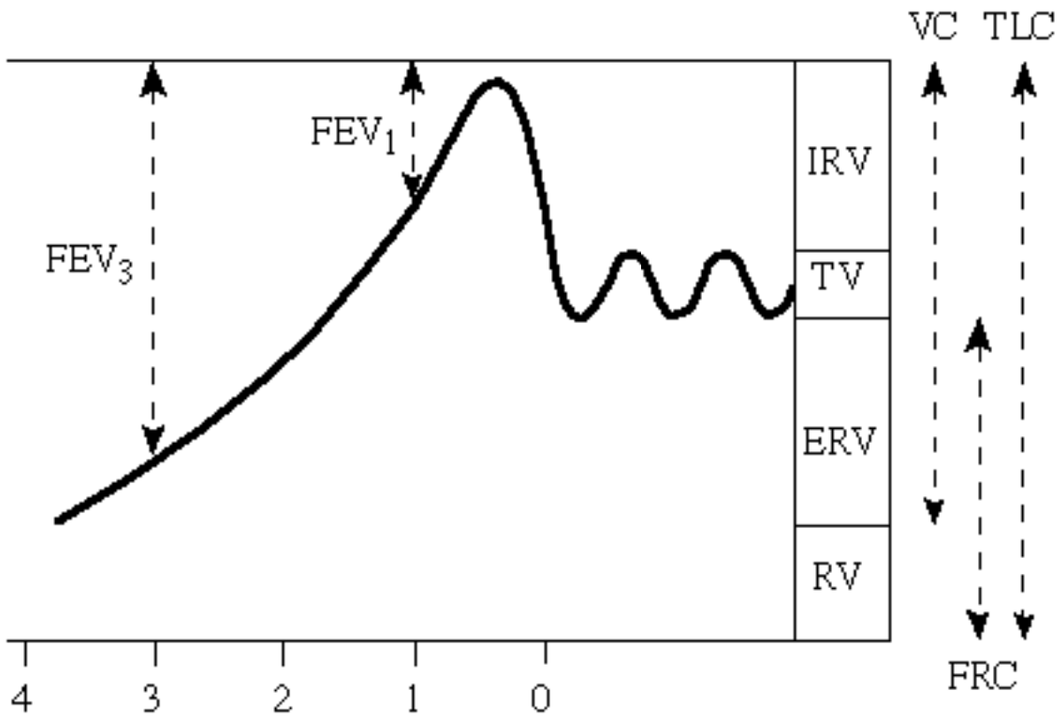
Lung Volumes & Capacities



1.Tidal volume (**TV**)=amount of gas moving in and out with each respiratory cycle2.Residual volume (**RV**)=amount of gas remaining in the lung after a maximal expiration3.Total lung capacity (**TLC**)=gas contained in lung at the end of a maximal inspiration4.Vital capacity (**VC**)=amount of gas that can be expired after a maximal inspiration without force5.Functional residual capacity (**FRC**)=volume of gas remaining in lungs at the end of a quiet expiration

Notes:

Changes In Lung Volumes A.DECREASED VC:1.Reduction in functioning lung tissue due to(a)space-occupying process (eg, pneumonia, infarction)(b)surgical removal of lung tissue2.Process reducing overall volume of the lungs (eg, diffuse pulmonary fibrosis)3.Inability to expand lungs due to(a)muscular weakness (eg, poliomyelitis)(b)increase in abdominal volume (eg, pregnancy)(c)pleural effusion B.INCREASED FRC and RV:characteristic of air trapping and overinflation (eg, asthma, emphysema) *Associated with:* increased TLCC.DECREASED FRC and RV:1.Process reducing overall volume of lungs (eg, diffuse pulmonary fibrosis)2.Process that occupies volume within alveoli (eg, alveolar microlithiasis)3.Process that elevates diaphragm (eg, ascites, pregnancy), usually associated with decreased TLC

Notes:

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Flow Rates A.Spirometric measurements:1.Forced expiratory volume (FEV)=amount of air expired during a certain period (usually 1 + 3 sec);Normal values: **FEV_1** = 83%; **FEV_3** = 97% 2.Maximal midexpiratory flow rate (MMFR)=amount of gas expired during the middle half of forced expiratory volume curve (largely effort independent)Indicator of small airway resistance 3.Flow-volume loop=gas flow is plotted against the actual volume of lung at which this flow is occurringUseful in identifying obstruction in large airways B.Resistance in small airwaysClosing volume = lung volume at which dependent lung zones cease to ventilate because of airway closure in small airway disease or loss of lung elastic recoil ● decrease in FEV, MMFR, MBC:(a)expiratory airway obstruction (reversible as in spasmodic asthma / irreversible as in emphysema)(b)respiratory muscle weakness

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Notes:

Diffusing Capacity =rate of gas transfer across the alveolocapillary membrane in relation to a constant pressure difference across it; measured by the carbon monoxide diffusion method*Reduction:* 1.Ventilation / perfusion inequality: less CO is taken up by poorly ventilated or poorly perfused areas (eg, emphysema)2.Reduction of total surface area (eg, emphysema, surgical resection)3.Reduction in permeability from thickening of alveolar membrane (eg, cellular infiltration, edema, interstitial fibrosis)4.Anemia with lack of hemoglobin

Notes:

Arterial Blood Gas Abnormalities ● decreased pulmonary arterial O_2:1.alveolar hypoventilation2.impaired diffusion3.abnormal ventilation/perfusion ratios4.anatomic shunting ● elevated pulmonary arterial CO_2:1.alveolar hypoventilation2.impaired ventilation / perfusion ratios

Notes:

V/Q Inequality A.NORMAL(a)blood flow decreases rapidly from base to apex(b)ventilation decreases less rapidly from base to apex◊V/Q is low at base and high at apex◊Pulmonary arterial O_2 is substantially higher at apex◊Pulmonary arterial CO_2 is substantially higher at baseB.ABNORMALchiefly resulting from non- / underventilated lung regions (non- / underperfused regions do not result in blood gas disturbances)

Notes:

Compliance =relationship of the change in intrapleural pressure to the volume of gas that moves into the lungsA.DECREASED COMPLIANCEedema, fibrosis, granulomatous infiltration B.INCREASED COMPLIANCEemphysema (faulty elastic architecture) √ height of diaphragm at TLC can provide some indication of lung compliance, particularly valuable in sequential roentgenograms for comparison in:1. Diffuse interstitial pulmonary edema2. Diffuse interstitial pulmonary fibrosis

Notes:

THYMUS *Origin:*residual thymic tissue in neck in 1.8 - 21%*Embryogenesis:* dorsal + ventral wings of 3rd (and possibly 4th) branchial pouch begin to form the primordia of the inferior parathyroid and thymic glands at 4th-5th week of gestation; both glands separate from pharyngeal wall + migrate caudally and medially with the thymus pulling the inferior parathyroid glands along the thymopharyngeal tract; thymic primordium fuses with its contralateral counterpart inferior to thyroid gland; thymic tail thins + disappears by 8th week *Thymic weight:* increases from birth to age 11 - 12 years (22 ± 13 g in neonate, 34 ± 15 g at puberty); ratio of thymic weight to body weight decreases with age (involution after puberty, total fatty replacement after age 60) √ measurement (perpendicular to axis of aortic arch):<18 mm before age 20; <13 mm after age 20√ triangular shape like an arrowhead (62%), bilobed (32%), single lobe (6%)√ muscular density of 30 HU (before puberty)√ flat / concave borders with abundant fat (after puberty)√ detected in 83% of subjects <50 years of age;in 17% of subjects >50 years of age◊atrophies under stress (due to increase in endogenous steroids)

Ectopic Tymus

Notes:

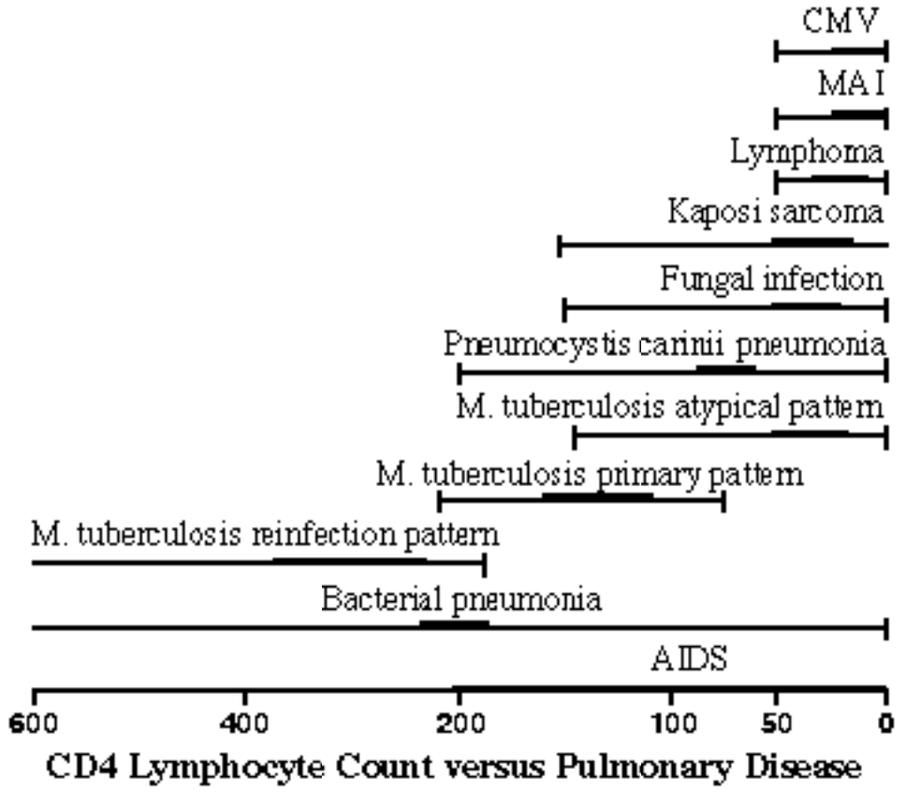
Ectopic Tymus √ solid mass√ cystic mass (= endodermal-lined cavity of thymopharyngeal duct / cystic degeneration of Hassall corpuscles or glandular epithelium)(1)Unilateral failure of thymic primordium to descend√ neck mass of thymic tissue on one side of neck√ ipsilateral absence of normal thymic lobe√ parathyroid tissue within ectopic thymus(2)Small rest of thymus left behind within thymopharyngeal tract during migration√ neck mass√ normally positioned bilobed thymus(3)Atypical location: trachea, skull base, intrathyroidal

Notes:

ACUTE EOSINOPHILIC PNEUMONIA
*Etiology:*idiopathic (no evidence of infection / exposure to potential antigens) with abrupt increase in lung cytokines*Age:*32 ± 17 years; M>F*Histo:*eosinophilic infiltrates + pulmonary edema (from release of eosinophilic granules altering vascular permeability) ● acute respiratory failure in previously healthy individuals ● markedly elevated levels of eosinophils in bronchoalveolar lavage fluid ● no peripheral eosinophilia ● acute febrile illness of 1-5 days duration, myalgia√ bilateral interstitial + air space opacities√ pleural effusion*Rx:*IV corticosteroids*Dx:*bronchoscopy with bronchopulmonary lavage*DDx:*chronic eosinophilic pneumonia (infiltrates with peripheral predominance)

Notes:

AIDS
=Acquired immune deficiency syndrome=ultimately fatal disease characterized by HIV seropositivity, specific opportunistic infections, specific malignant neoplasms (Kaposi sarcoma, Burkitt lymphoma, primary lymphoma of brain)=patient with CD4 cell count <200 cells/µL (normal range, 800-1,200 cells/µL)*Incidence:*2 million Americans are infected with HIV + 270,000 have AIDS (estimate in 1993); >50% develop pulmonary disease **AIDS-related complex** (ARC) = GENERALIZED LYMPHADENOPATHY SYNDROME =prodromal phase of HIV seropositivity, generalized lymphadenopathy, CNS diseases other than those associated with AIDS*Time interval:*approximately 10 years between seroconversion + clinical AIDS ● weight loss, malaise, diarrhea ● fever, night sweats, lymphadenopathy ● lymphopenia with selective decrease in helper T-cells *Organism:*human immunodeficiency virus (HIV) = human T-cell lymphotropic virus type III (HTLV III) = lymphadenopathy-associated virus (LAV)*Pathomechanism:* HIV retrovirus attaches to CD4 molecule on surface of T-helper lymphocytes + macrophages + microglial cells; after cellular invasion HIV genetic information is incorporated into cells chromosomal DNA; virus remains dormant for weeks to years; after an unknown stimulus for viral replication CD4 lymphocytes are destroyed (normal range of 800-1,000 cells/mm^3) and others become infected leading to impairment of the immune system; CD4 lymphocyte number and function decreases (at an approximate rate of 50-80 cells/year) CD4 Lymphocyte Count vs. HIV disease status (cells/mm^3) <300-400thrush, hairy leukoplakia<200-400Pneumocystis pneumonia<150cerebral toxoplasmosis<100intestinal CMV + MAI infection<50AIDS-related lymphoma



CD4 Lymphocyte Count versus Pulmonary Disease

*Prognosis:*median survival with CD4 lymphocyte count <50 cells/mm^3 is 12 months*Transmission by:*intimate sexual contact, exposure to contaminated blood / bloody body secretions*Groups at risk:* 1.Homosexual males (74%)2.IV drug abusers (16%)3.Recipients of contaminated blood products (3%)4.Sexual partner of drug abuser + bisexual man5.Infants born to woman infected with AIDS virus◊HIV antibodies present in >50% of homosexuals + 90% of IV drug abusers!◊Rate of heterosexual transmission is increasing! *Clinical classification:* group Iacute HIV infection with seroconversiongroup IIasymptomatic HIV infectiongroup IIIpersistent generalized lymphadenopathygroup IVother HIV disease-subgroup Aconstitutional disease-subgroup Bneurologic disease-subgroup Csecondary infectious disease-subgroup Dsecondary cancers-subgroup Eother conditions AIDS-defining pulmonary conditions (CDC, 1987): (1)Tracheal / bronchial / pulmonary candidiasis(2)Pulmonary CMV infection(3)Herpes simplex bronchitis / pneumonitis(4)Kaposi sarcoma(5)Immunoblastic / Burkitt lymphoma(6)Pneumocystis carinii pneumonia A.LYMPHADENOPATHY*Cause:* reactive follicular hyperplasia = HIV adenopathy (50%), AIDS-related lymphoma (20%), mycobacterial infection (17%), Kaposi sarcoma (10%), metastatic tumor, opportunistic infection with multiple organisms, drug reaction Location:mediastinum, axilla, retrocrural B.OPPORTUNISTIC INFECTIONaccounts for majority of pulmonary disease ◊Pulmonary infection is often the first AIDS-defining illness!1.Pneumocystis carinii pneumonia (60-80%)20-40% develop >1 episode during disease ● CD4+ T helper lymphocyte cell count ≤200/mm^3 ● subacute insidious onset with malaise, minimal cough√ bilateral ground-glass infiltrates without effusion / adenopathy√ bilateral perihilar interstitial infiltrates√ diffuse bilateral alveolar infiltrates*Mortality:*in 25% fatal 2.Fungal disease (<5%)(a)Cryptococcus neoformans pneumonia (2-15%)usually associated with brain / meningeal disease √ segmental infiltrate + superimposed pulmonary nodules ± lymphadenopathy ± pleural effusion(b)Histoplasma capsulatum√ typically diffuse nodular / miliary pattern at time of diagnosis√ normal CXR in up to 35%(c)Coccidioides immitis√ diffuse infiltrates + thin-walled cavities(d)Candida albicans(e)Aspergillus: less common + less invasive due to relative preservation of neutrophilic function 3.Mycobacterial infection (20%):(a)M. tuberculosis (increasing frequency):◊AIDS patients are 500 times more likely to become infected than general population!√ postprimary TB pattern with upper-lobe cavitating infiltrate (CD4 lymphocyte count of 200-500 cells/mm^3)√ primary TB pattern with lung infiltrate / lung masses + hilar / mediastinal lymphadenopathy + pleural effusion (CD4 lymphocyte count of 50-200 cells/mm^3)√ atypical TB pattern with diffuse reticular / nodular infiltrates (CD4 lymphocyte count of <50 cells/mm^3)√ adenopathy of low attenuation with rim enhancement on CECT(b)M. avium-intracellulare (5%) √ adenopathy, pulmonary infiltrates, nodules, miliary disease(c)M. kansasii and others4.Bacterial pneumonia (5-30%):(a)Haemophilus influenzae, Streptococcus pneumoniae, Staphylococcus aureus(b)Nocardia pneumonia (<5%)usually occurs in cavitating pneumonia √ segmental / lobar alveolar infiltrate ± cavitation ± ipsilateral pleural effusion5.CMV pneumoniamost frequent infection found at autopsy (49-81%), diagnosed before death in only 13-24%; high combined prevalence with Kaposi sarcoma 6.Toxoplasmosis C.TUMOR1.Kaposi sarcoma (15%)Location:lung involvement (20%) preceded by widespread skin + organ involvementSite:peribronchovascular distribution (best appreciated on CT)√ numerous fluffy ill-defined nodules / asymmetric clusters in a vague perihilar distribution√ interlobular septal thickening√ pleural effusion (30%)√ lymphadenopathy (10-35%), late in disease2.AIDS-related lymphoma of B-cell origin (2-5%)primarily immunoblastic NHL / Burkitt lymphoma / non-Burkitt lymphoma; occasionally Hodgkin disease Location:pulmonary involvement (9-31%), CNS, GI tract, liver, spleen, bone marrowSite:primarily extranodal√ solitary / multiple well-defined pulmonary nodules often coexistent with pleural effusion ± axillary / supraclavicular / cervical / hilar adenopathy√ alveolar infiltrates, paraspinal masses D.LYMPHOID INTERSTITIAL PNEUMONITIS*Age:*in children <13 years of age E.SEPTIC EMBOLI F.PREMATURE DEVELOPMENT OF BULLAE (40%) with disposition to spontaneous pneumothorax

Notes:

ADULT RESPIRATORY DISTRESS SYNDROME
=SHOCK LUNG = POSTTRAUMATIC PULMONARY INSUFFICIENCY = HEMORRHAGIC LUNG SYNDROME = RESPIRATOR LUNG = STIFF LUNG SYNDROME = PUMP LUNG = CONGESTIVE ATELECTASIS = OXYGEN TOXICITY=severe unexpected life-threatening acute respiratory distress characterized by abrupt onset of marked dyspnea, increased respiratory effort, severe hypoxemia associated with widespread airspace consolidation *Histo:* (a)up to 12 hours:fibrin + platelet microemboli(b)12-24 hours:interstitial edema(c)24-48 hours:capillary congestion, extensive interstitial + alveolar proteinaceous edema + hemorrhage, widespread microatelectasis, destruction of type I alveolar epithelial cells(d)5-7 days:extensive hyaline membrane formation, hypertrophy + hyperplasia of type II alveolar lining cells(e)7-14 days:extensive fibroblastic proliferation in interstitium + within alveoli, rapidly progressing collagen deposition + fibrosis; almost invariably associated with infection *Predisposed:* hemorrhagic / septic shock, massive trauma (pulmonary / general body), acute pancreatitis, aspiration of liquid gastric contents, heroine / methadone intoxication, massive viral pneumonia, traumatic fat embolism, near-drowning, conditions leading to pulmonary edema *mnemonic:*"DICTIONARIES"**D**isseminated intravascular coagulation **I**nfection **C**aught drowning **T**rauma **I**nhalants: smoke, phosgene, NO_2 O_2 toxicity **N**arcotics + other drugs **A**spiration **R**adiation **I**ncludes pancreatitis **E**mboli: amniotic fluid, fat **S**hock: septic, hemorrhagic, cardiogenic, anaphylactic CXR: √ NO cardiomegaly / pleural effusion-up to 12 hours:√ characteristic 12-hour delay between clinical onset of respiratory failure and CXR abnormalities-12-24 hours:√ patchy ill-defined opacities throughout both lungs-24-48 hours:√ massive airspace consolidation of both lungs-5-7 days:√ consolidation becomes inhomogeneous (resolution of alveolar edema)√ local areas of consolidation (pneumonia)->7 days:√ reticular / bubbly lung pattern (diffuse interstitial + airspace fibrosis) Complication of continuous positive pressure ventilation (= **barotrauma**) *Path:* (a)rupture of alveoli along margins of interlobular septa + vascular structures(b)air dissection along interlobular septa + perivascular spaces (= interstitial emphysema)(c)interstitial air rupturing into pleural space(= pneumothorax) / into mediastinum(= pneumomediastinum)√ mottled air opacities often outlining bronchovascular bundles√ large subpleural cysts without definable wall usually at diaphragmatic + mediastinal surface compressing adjacent lung

Notes:

ALPHA-1 ANTITRYPSIN DEFICIENCY
=rare autosomal recessive disorderAlpha-1 antitrypsin (glycoprotein) is synthesized in liver + released into serum Action:proteolytic inhibitor of trypsin, chymotrypsin, elastase, plasmin, thrombin, kallikrein, leukocytic + bacterial proteases; neutralizes circulating proteolytic enzymesMode of injury from deficiency: PMNs + alveolar macrophages sequester into lung during recurrent bacterial infections + release elastase, which digests basement membrane *Age:*early age of onset (20-30 years); M:F = 1:1 ● rapid + progressive deterioration of lung function ● chronic sputum production (50%)√ severe panacinar emphysema with basilar predominance√ reduction in size + number of pulmonary vessels in lower lobes√ redistribution of blood flow to unaffected upper lung zones√ bullae at both lung bases√ marked flattening of diaphragm√ minimal diaphragmatic excursion√ multilobar cystic bronchiectasis (40%)*Cx:*hepatic cirrhosis (in homozygotic individuals)

Notes:

ALVEOLAR MICROLITHIASIS
=very rare disease of unknown etiology characterized by myriad of calcospherites (= tiny calculi) within alveoli*Age peak:*30-50 years; begins in early life; has been identified in utero; M:F = 1:1; in 50% familial (restricted to siblings) ● usually asymptomatic (70%) ● dyspnea on exertion (reduction in residual volume) ● cyanosis, clubbing of fingers ● striking discrepancy between striking radiographic findings and mild clinical symptoms ● NORMAL serum calcium + phosphorus levels√ very fine, sharply defined, sandlike micronodulations(<1 mm)√ diffuse involvement of both lungs√ intense uptake on bone scan*Prognosis:* (a)late development of pulmonary insufficiency secondary to interstitial fibrosis(b)disease may become arrested(c)microliths may continue to form / enlarge*DDx:*"Mainline" pulmonary granulomatosis = IV abuse of talc-containing drugs such as methadone (rarely as numerous + scarring + loss of volume)

Notes:

ALVEOLAR PROTEINOSIS
=PULMONARY ALVEOLAR PROTEINOSIS (PAP)=accumulation of PAS positive phospholipid material in alveoli (= surfactant)*Etiology:*?; associated with dust exposure (eg, silicoproteinosis is histologically identical to PAP), immunodeficiency, hematologic + lymphatic malignancies, AIDS, chemotherapy*Pathophysiology:* (a)overproduction of surfactant by granular pneumocytes(b)defective clearance of surfactant by alveolar macrophages*Histo:*alveoli filled with proteinaceous material (the ONLY pure airspace disease), normal interstitium*Age peak:*30-50 years (age range 2-70 years);M:F = 3:1 ● asymptomatic (10-20%) ● gradual onset of dyspnea + cough ● weight loss, weakness, hemoptysis ● defect in diffusing capacity√ "bat-wing" consolidation of ground-glass pattern, predominant at bases√ small acinar nodules + coalescence + consolidation√ patchy peripheral / primarily unilateral infiltrates (rare)√ reticular / reticulonodular / linear interstitial pattern with Kerley B lines (late stage)√ slow clearing over weeks or months√ slow progression (1/3), remaining stable (2/3)√ NO adenopathy, NO cardiomegaly, NO pleural effusionHRCT: √ patchy ground-glass opacity√ smooth septal thickening*Cx:*infections (frequently secondary to poorly functioning macrophages + excellent culture medium): Nocardia asteroides (most common), mycobacterial, fungal, Pneumocystis, CMV*Prognosis:* highly variable course with clinical and radiologic episodes of exacerbation + remissions (a)50% improvement / recovery(b)30% death within several years under progression*Rx:*bronchopulmonary lavage*DDx:* (a)during acute phase: pulmonary edema, diffuse pneumonia, ARDS(b)in chronic stage:1.Idiopathic pulmonary hemosiderosis (boys, symmetric involvement of mid + lower zones, progression to nodular + linear pattern)2.Hemosiderosis (bleeding diathesis)3.Pneumoconiosis4.Hypersensitivity pneumonitis5.Goodpasture syndrome (more rapid changes, renal disease)6.Desquamative interstitial pneumonia ("ground glass" appearance, primarily basilar + peripheral)7.Pulmonary alveolar microlithiasis (widespread discrete intraalveolar calcifications primarily in lung bases, rare familial disease)8.Sarcoidosis (usually with lymphadenopathy)9.Lymphoma10.Bronchioloalveolar cell carcinoma (more focal, slowly enlarging with time)

Notes:

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AMNIOTIC FLUID EMBOLISM
=most common cause of maternal peripartum death ● dyspnea ● shock during / after labor + delivery*Pathogenesis:*Amniotic debris enters maternal circulation resulting in (1) pulmonary embolization (2) anaphylactoid reaction (3) DIC√ usually fatal before radiographs obtained√ may demonstrate pulmonary edema

Notes:

AMYLOIDOSIS
=disease characterized by an extracellular deposit of proteinaceous twisted ß-pleated sheet fibrils of great chemical diversity*Histo:*protein (immunoglobulin) / polysaccharide complex; affinity for Congo red stain@ Lung involvement *Incidence:*1° amyloidosis (in up to 70%),2° amyloidosis (rare) A.TRACHEOBRONCHIAL TYPE (most common) ● hemoptysis (most frequent complaint) ● stridor, cough, dyspnea, hoarseness, wheezing√ multiple nodules protruding from wall of trachea / large bronchi√ diffuse rigid narrowing of a long tracheal segment√ prominent bronchovascular markings√ destructive pneumonitisB.NODULAR TYPE*Age:*>60 years of age; M:F = 1:1 ● usually asymptomatic√ mediastinal / hilar adenopathy√ solitary / multiple parenchymal nodules in a peripheral / subpleural location ± central calcification / ossification; slow growth over years√ ± pleural effusion*DDx:*metastatic disease, granulomatous disease, rheumatoid lung, sarcoidosis, mucoid impactionC.DIFFUSE PARENCHYMAL TYPE (least common)*Age:*>60 years of age ● usually asymptomatic with normal CXR ● cough + dyspnea with abnormal CXR √ widespread small irregular densities (exclusively interstitial involvement) ± calcification√ may become confluent ± honeycombing*DDx:*idiopathic interstitial fibrosis, pneumoconiosis (especially asbestosis), rheumatoid lung, Langerhans cell histiocytosis, scleroderma

Notes:

ANKYLOSING SPONDYLITIS

*Incidence:*1% of patients with ankylosing spondylitis*Histo:*interstitial + pleural fibrosis with foci of dense collagen deposition, NO granulomas ● bone manifestations obvious + severeLocation:apices / upper lung fields√ uni- / bilateral, coarse, linear shadows + cavities√ bronchiectasis may be present√ superinfection, especially with aspergillosis (mycetoma formation) / atypical mycobacteria*DDx:*other causes of pulmonary apical fibrosis (primary infection by fungi / mycobacteria; cancer)

Notes:

ASBESTOS-RELATED DISEASE
Substances: aspect (length-to-diameter) ratio effects carcinogenicity: eg, aspect ratio of 32 = 8 µm long, 0.25 µm wide -commercial amphiboles: crocidolite, amosite-commercial nonamphiboles / serpentines: chrysotile-noncommercial contaminating amphiboles: actinolite, anthophyllite, tremolite(a) relatively benign:(1) Chrysotile (white asbestos) in Canada(2) Anthophyllite in Finland, North America(3)Tremolite(b)relatively malignant:(1)Crocidolite (blue / black asbestos) in South Africa, Australia(2)Amosite (brown asbestos)◊Very fine fibers (crocidolite) associated with largest number of pleural disease!*Occupational exposure:* (a)asbestos mining + milling(b)insulation, textile manufacturing, construction, ship building, gaskets, brake linings

Pulmonary Asbestosis Asbestos-related Pleural Disease Atelectatic Asbestos Pseudotumor Lung Cancer In Asbestos-related Disease

Notes:

Pulmonary Asbestosis =(term asbestosis reserved for) chronic progressive diffuse interstitial fibrosis*Incidence:*in 49-52% of industrial asbestos exposure*Latency period:*40-45 years*Histo:*interstitial fibrosis begins in peribronchiolar areas, then progresses to involve adjacent alveoli*Diagnostic criteria:* 1.reliable history of exposure2.appropriate time interval between exposure + detection3.CXR evidence4.restrictive pattern of lung impairment5.abnormal diffusing capacity6.bilateral crackles at posterior lung bases, not cleared by cough ● dyspnea ● restrictive pulmonary function testsLocation:more severe in lower subpleural zones (concentration of asbestos fibers under pleura)√ small irregular opacities (NOT rounded as in coal / silica)√ confined to lung bases, progressing superiorly√ septal lines (= fibrous thickening around secondary lobules)√ "shaggy" heart border = obscuration secondary to parenchymal + pleural changes√ ill-defined outline of diaphragm√ honeycombing (uncommon)√ rarely massive fibrosis, predominantly at lung bases without migration toward hilum (DDx from silicosis / CWP)√ NO hilar adenopathy√ Ga-67 uptake gives a quantitative index of inflammatory activityHRCT: √ subpleural pulmonary arcades = branching linear structures most prominent posteriorly (initial finding) = centrilobular peribronchiolar fibrosis√ curvilinear subpleural lines parallel to + within 1 cm of pleura (30%) = multiple subpleural dotlike reticulonodularities connected to the most peripheral branch of pulmonary artery √ parenchymal band = linear <5 cm long + several mm wide opacity, often extending to pleura, which may be thickened + retracted at site of contact√ reticulation = network of linear densities, usually posteriorly at lung bases√ honeycombing = multiple cystic spaces <1 cm in diameter with thickened walls√ thickened interlobular septal lines√ thickened intralobular lines

Notes:

Asbestos-related Pleural Disease 1.Focal Pleural Plaques (65%)=hyalinized collagen in submesothelial layer of parietal pleura*Incidence:*most common manifestation of exposure; 6% of general population will show plaques*Latent period:*in 10% after 20 years; in 50% after 40 years*Histo:*dense hypocellular undulating collagen fibers often arranged in a basket weave pattern ± focal / massive calcificationsLocation:bilateral + multifocal; posterolateral midportion of chest wall between 7-10th rib; aponeurotic portion of diaphragm; mediastinum; following rib contours; visceral pleura + apices + costophrenic angles typically spared ● asymptomatic√ usually focal area of pleural thickening (<1 cm thick) with edges thicker than central portions of plaque; in 48% only finding; in 41% with parenchymal changes; stable over time√ no hilar adenopathy√ usually not calcified*DDx:*chest wall fat, rib fractures, rib companion shadows 2.Diffuse Pleural Thickening (17%)=diffuse thickening of parietal pleura (visceral pleura involved in 90%, but difficult to demonstrate) ● may cause restriction of pulmonary function*May be associated with:* rounded atelectasis√ bilateral process with "shaggy heart" appearance (20%)√ smooth; difficult to assess when viewed en face√ thickening of interlobar fissures√ focally thickened diaphragm√ obliterated costophrenic angles (minority of cases) 3.Pleural Calcification (21-25-60%)detected by radiography in 25%, by CT in 60% *Overall incidence:*20%*Latent period:*>20 years to become visible; in 40% after 40 years*Histo:*calcification starts in parietal pleura; calcium deposits may form within center of plaques√ dense lines paralleling the chest wall, mediastinum, pericardium, diaphragm (bilateral diaphragmatic calcifications with clear costophrenic angles are (PATHOGNOMONIC)√ advanced calcifications are leaflike with thick-rolled edges*DDx:*talc exposure, hemothorax, empyema, therapeutic pneumothorax for TB (often unilateral, extensive sheetlike, on visceral pleura) 4.Pleural Effusion (21%)Earliest asbestos-related pleural abnormality, frequently followed by diffuse pleural thickening + rounded atelectasis *Prevalence:*3% (increases with increasing levels of asbestos exposure)*Latent period:*8-10 years after exposure ● **benign asbestos pleurisy** ● may be associated with chest pain (1/3) ● usually small sterile, serous / hemorrhagic exudate√ recurrent bilateral effusions√ ± plaque formation*DDx:*TB, mesothelioma

Notes:

Atelectatic Asbestos Pseudotumor =ROUNDED ATELECTASIS = "FOLDED LUNG"= infolding of redundant pleura accompanied by segmental / subsegmental atelectasisLocation:posteromedial / posterolateral lower lobe (most common); frequently bilateral√ 2.5-8 cm focal subpleural mass abutting a region of thickened pleura √ size + shape show little progression, occasionally decrease in size√ volume loss in adjacent lungCT: √ rounded / lentiform / wedge-shaped outline√ contiguous to areas of diffuse pleural thickening ± calcification√ partial interposition of lung between pleura + mass√ "crows feet" = linear bands radiating from mass into lung parenchyma (54%)√ "vacuum cleaner" / "comet tail" sign = bronchovascular markings emanating from nodular subpleural mass + coursing toward ipsilateral hilum √ "Swiss cheese" air bronchogram (18%)

Notes:

Lung Cancer In Asbestos-related Disease Occurrence related to: (a)cumulated dose of asbestos fibers(b)smoking (synergistic carcinogenic effect)Increased risk by factor of up to 90 in smokers versus a factor of 5 in nonsmokers!Up to 25% of asbestos workers who smoke develop lung cancer!(c)preexisting interstitial disease(d)occupational exposure to known carcinogen*Latency period:* 25-35 years*Associated with:* increased incidence of gastric carcinoma*Histo:*bronchioloalveolar cell carcinoma (most common); bronchogenic carcinoma (adenocarcinoma + squamous cell)Location: at lung base / in any location if associated with smoking

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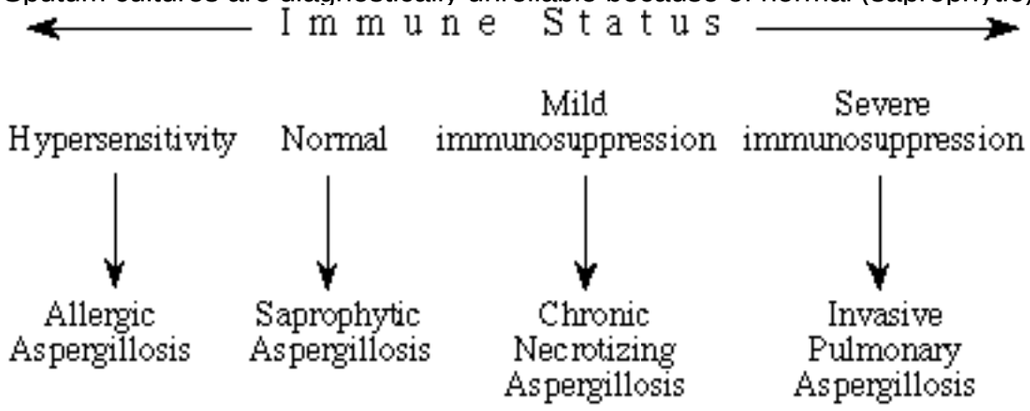
Notes:

ASPERGILLOSIS

Organism: Aspergillus fumigatus = intensely antigenic ubiquitous soil fungus existing as (a)conidiophores = reproductive form releasing thousands of spores(b)hyphae (= matured spores) characterized by 45° dichotomous branching pattern*Occurrence:* commonly in sputum of normal persons, ability to invade arteries + veins facilitating hematogenous dissemination M:F = 3:1 *Predisposed:* (a)preexisting lung disease (tuberculosis, bronchiectasis)(b)impairment of immune system (alcoholism, advanced age, malnutrition, concurrent malignancy, poorly controlled diabetes, cirrhosis, sepsis)*Cx:*dissemination to heart, brain, kidney, GI tract, liver, thyroid, spleen Sputum cultures are diagnostically unreliable because of normal (saprophytic) colonization of upper airways!



Noninvasive Aspergillosis Semi-invasive Aspergillosis Invasive Pulmonary Aspergillosis Allergic Bronchopulmonary Aspergillosis Pleural Aspergillosis

Notes:

Noninvasive Aspergillosis =SAPROPHYTIC ASPERGILLOSIS=noninvasive colonization of preexisting cavity / cyst in immunologically normal patients with cavitary disease [tuberculosis, sarcoidosis (common), bronchiectasis, bullous lung disease, carcinoma] ● sputum blood-streaked / severe hemoptysis (45-70%) ● elevated serum precipitins level for Aspergillus (50%)√ solid round gravity-dependent mass within preexisting spherical / ovoid thin-walled cavity (= Mounod sign)*Histo:*mycetoma = aspergilloma = **fungus ball**

= masslike collection of intertwined hyphae matted together with fibrin, mucus, cellular debris colonizing a pulmonary cavity √ crescent-shaped air space separates fungus ball from cavity wall√ fungus ball may calcify in scattered / rimlike fashion√ pleural thickening adjacent to preexisting cyst / cavity, commonly first sign before visualizing mycetoma

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Notes:

Semi-invasive Aspergillosis =CHRONIC NECROTIZING ASPERGILLOSIS=chronic cavitary slowly progressive disease in patients with preexisting lung injury (COPD, radiation therapy), mild immune suppression, or debilitation (alcohol, diabetes) ● symptoms mimicking pulmonary tuberculosis ↓ progressive consolidation (usually upper lobe) ↓ development of air crescent and fungus ball*Dx:*pathologic examination demonstrating local tissue invasion

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Notes:

Invasive Pulmonary Aspergillosis =often fatal form in severely immunocompromised patients (most commonly in lymphoma / leukemia patients with prolonged granulocytopenia) with absolute neutrophil count of <500*Path:*endobronchial fungal proliferation followed by transbronchial vascular invasion eventually causes widespread hemorrhage + thrombosis of pulmonary arterioles + ischemic tissue necrosis + systemic dissemination; fungus ball = devitalized sequestrum of lung infiltrated by fungi ● Hx of series of bacterial infections + unremitting fever ● pleuritic chest pain (mimicking emboli) ● progression of pulmonary infiltrates despite broad-spectrum antibiotics(a)early signs:√ CT halo sign = single / multiple 1-3 cm peripheral nodules (= necrotic lung) with halo of ground-glass attenuation (= hemorrhagic edema)√ patchy localized bronchopneumonia(b)signs of progression√ enlargement of nodules into diffuse bilateral consolidation√ development into large wedge-shaped pleural-based lesions√ air-crescent sign = cavitation of existing nodule (air crescent between sequestrum and lung) 1-3 weeks after granulocyte recovery◊has better prognosis than consolidation without cavitation (feature of resolution phase)*Dx:*branching hyphae at tissue examination

Notes:

Allergic Bronchopulmonary Aspergillosis =hypersensitivity toward aspergilli in patients with long-standing asthma*Incidence:*in 1-2% of patients with asthma, in 10% of patients with cystic fibrosis; most common + clinically important form*Age:*mostly young patients (begins in childhood); may be undiagnosed for 10-20 years A.ACUTE ALLERGIC BRONCHOPULMONARY ASPERGILLOSISType I reaction = immediate hypersensitivity (IgE-mediated) *Histo:*alveoli filled with eosinophilsB.CHRONIC ALLERGIC BRONCHOPULMONARY ASPERGILLOSISType III reaction = delayed immune complex response = Arthus reaction (IgG-mediated) *Histo:*bronchial damage secondary to Aspergillus antigen reacting with IgG antibodies, immune complexes activate complement leading to tissue injury *Pathophysiology:* inhaled spores are trapped in segmental bronchi of individuals with asthma, germinate, and form hyphae; immunologic response coupled with proteolytic enzymes causes pulmonary infiltrates + tissue damage + central bronchiectasis *Criteria:* (a)Primary diagnostic criteria:*acronym:*ARTEPICS**A**sthma (84-96%) **R**oentgenographic transient or fixed pulmonary infiltrates **T**est for A. fumigatus positive: immediate skin reaction **E**osinophilia in blood between 8% and 40% **P**recipitating antibodies to A. fumigatus (70%) **I**gE in serum elevated **C**entral bronchiectasis (late manifestation that proves diagnosis) **S**erum-specific IgE and IgG A. fumigatus levels elevated (b)Secondary diagnostic criteria (less common):1.Aspergillus fumigatus mycelia in sputum2.Expectoration of brown sputum plugs (54%)3.Arthus reaction (= late skin reactivity with erythema + induration) to Aspergillus antigen *Staging:* Iacute phase with all primary diagnostic criteriaIIclearing of pulmonary infiltrates with declining IgE levelsIIIall criteria of stage I reappear after emissionIVcorticosteroid dependencyVirreversible lung fibrosis ● flulike symptoms: fever, headache, malaise, weight loss, fleeting chest pain√ migratory pneumonitis = transient recurrent "fleeting" alveolar patchy subsegmental / lobar infiltrates in upper lobes (50%), lower lobes (20%), middle lobe (7%), both lungs (65%); may persist for >6 months√ central varicose / cystic bronchiectasis√ "tramlike" bronchial walls (edema)√ 1-2 cm ring shadows (= bronchus on end) around hilum + upper lobes (HALLMARK)√ "finger-in-glove / toothpaste shadow" = V- or Y-shaped central mucus plugs in 2nd order bronchi of 2.5-6 cm in length remaining for months + growing in size√ lobar consolidation (in 32%)√ atelectasis (in 14%) with collateral air drift√ cavitation (in 14%) secondary to postobstructive abscess√ hyperinflation (due to bronchospasm)√ pulmonary fibrosis + retraction√ hilar elevation due to lobar shrinkage√ emphysema√ NORMAL peripheral bronchi√ UNUSUAL are aspergilloma in cavity (7%), empyema, pneumothorax*DDx:*hypersensitivity pneumonitis or allergic asthma (no hyphae in sputum, normal levels of IgE + IgG to A. fumigatus), tuberculosis, lipoid pneumonia, Löffler syndrome, bronchogenic carcinoma

Notes:

Pleural Aspergillosis =Aspergillus empyema in patients with pulmonary tuberculosis, bacterial empyema, bronchopleural fistula pleural thickening

Notes:

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ASPIRATION OF SOLID FOREIGN BODY

*Age:*in 50% <3 years*Source:*in 85% vegetable origin (peanut, barley grass)Location:almost exclusively in lower lobes; R:L = 2:1√ obstructive overinflation (68%) + reflex vasoconstriction√ collapse (14-53%)√ infiltrate (11%)√ radiopaque foreign body (9%)√ air trapping (expiratory / lateral decubitus film)NUC: √ ventilation defect (initial breath) + retention (washout)*Cx:*bronchiectasis (from long retention)*DDx:*impacted esophageal foreign body

Notes:

ASPIRATION PNEUMONIA
Predisposing conditions: (1)CNS disorders / intoxication: alcoholism, mental retardation, seizure disorders, recent anesthesia(2)Swallowing disorders: esophageal motility disturbances, head + neck surgery ● low-grade fever ● productive cough ● choking on swallowingLocation:gravity-dependent portions of lung, posterior segments of upper lobes + lower lobes in bedridden patients, frequently bilateral, right middle + lower lobe with sparing of left lung is commonA.ACUTE ASPIRATION PNEUMONIA*Cause:*acid, food particles, anaerobic bacteria from GI tract provoke edema, hemorrhage, inflammatory cellular response, foreign-body reaction√ segmental consolidation in dependent portionB.CHRONIC ASPIRATION PNEUMONIA*Cause:*repeated aspiration of foreign material from GI tract over long time / mineral oil (eg, in laxatives)*Associated with:*Zenker diverticulum, esophageal stenosis, achalasia, TE fistula, neuromuscular disturbances in swallowing√ recurring segmental consolidation√ progression to interstitial scarring (= localized honeycomb appearance)√ bronchopneumonic infiltrates of variable location over months / years√ residual peribronchial scarringUpper GI: √ abnormal swallowing / aspiration

Notes:

ASTHMA
=episodic reversible bronchoconstriction secondary to hypersensitivity to a variety of stimuliA.INTRINSIC ASTHMA*Age:*middle age*Pathogenesis:* probably autoimmune phenomenon caused by viral respiratory infection and often provoked by infection, exercise, pharmaceuticals; no environmental antigen B.EXTRINSIC ASTHMA = ATOPIC ASTHMA*Pathogenesis:* secondary to antigens producing an immediate hypersensitivity response (type I); reagin sensitizes mast cells to release histamine followed by increased vascular permeability, edema, small muscle contraction; effects primarily bronchi causing airway obstruction *Nonoccupational allergens:* pollens, dog + cat fur, tamarind seed powder, castor bean, fungal spores, grain weevil *Occupational allergens:* (a)natural substances: wood dust, flour, grain, beans(b)pharmaceuticals: antibiotics, ASA(c)inorganic chemicals: nickel, platinum *Path:*bronchial plugging with large amounts of viscid tenacious mucus (eosinophils, Charcot-Leyden crystals), edematous bronchial walls, hypertrophy of mucous glands + smooth muscle ACUTE SIGNS: ● during asthmatic attack low values for FEV + MMFR and abnormal V/Q ratios ● increased resistance to airflow due to(a) smooth muscle contraction in airway walls (b) edema of airway wall caused by inflammation (c) mucus hypersecretion with airway plugging ● normal diffusing capacity√ hyperexpansion of lungs = severe overinflation + air trapping√ flattened diaphragmatic dome√ deepened retrosternal air space√ peribronchial cuffing (inflammation of airway wall)√ bronchial dilatation√ localized areas of hypoattenuationCHRONIC CHANGES: Normal chest x-ray in 73%, findings of abnormalities depend on (a)age of onset (<15 years of age in 31%; >30 years of age in none)(b)severity of asthma √ central ring shadows = bronchiectasis√ scars (from recurrent infections) *Cx:* (1)Pneumonia (2 x as frequent as in nonasthmatics)√ peripheral pneumonic infiltrates (secondary to blocked airways)(2)Atelectasis (5-15%) from mucoid impaction(3)Pneumomediastinum (5%), pneumothorax, subcutaneous emphysema; predominantly in children(4)Emphysema(5)Allergic bronchopulmonary aspergillosis with central bronchiectasis

Notes:

ATYPICAL MEASLES PNEUMONIA
=clinical syndrome in patients who have been previously inadequately immunized with killed rubeola vaccine and are subsequently exposed to the measles virus (= type III immune complex hypersensitivity); noted in children who have received live vaccine before 13 months of age ● 2- to 3-day prodrome of headache, fever, cough, malaise ● maculopapular rash beginning on wrists + ankles (sometimes absent) ● postinfectious migratory arthralgias ● history of exposure to measles√ extensive nonsegmental consolidation, usually bilateral√ hilar adenopathy (100%)√ pleural effusion (0-70%)√ nodular densities of 0.5-10 cm in diameter in peripheral location, may calcify and persist up to 30 months

Notes:

BARITOSIS

=inhalation of nonfibrogenic barium sulfate ● asymptomatic ● normal pulmonary function (benign course)√ bilateral nodular / patchy opacities, denser than bone (high atomic number)√ similar to calcified nodules√ NO cor pulmonale, NO hilar adenopathy√ regression if patient removed from exposure

Notes:

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BEHÇET SYNDROME
=rare multisystem disease of unknown origin characterized by(1)aphthous stomatitis(2)genital ulceration(3)iritis ● positive pathergy test = unusual hypersensitivity to pricking with formation of pustules at site of needle prick within 24-48 hours ● skin changes: erythema nodosum, folliculitis, papulopustular lesions ● arthritis, encephalitis ● epididymitis@Chest (5%)√ multiple peripheral subpleural opacities (due to hemorrhage, necrotic pulmonary infarctions)√ increased radiopacity near hila (pulmonary artery aneurysm)@Veins (25%)√ large vein occlusion; may cause SVC syndrome√ subcutaneous thrombophlebitis@Arteries√ arterial occlusion / pulseless disease√ aneurysm of large arteries (in 2%)

Notes:

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BERYLLIOSIS
=chronic granulomatous disorder as a result of beryllium-specific cell-mediated immune response (= delayed hypersensitivity reaction after exposure to acid salts from extraction of beryllium oxide)*Substance:*one of the lightest metals (atomic weight 9), marked heat resistance, great hardness, fatigue resistance, no corrosion*Occupational exposure:*fluorescent light bulb factories*Histo:*noncaseating granulomas within interstitium + along vessels + in bronchial submucosa ● positive beryllium lymphocyte transformation test (blood test of T-lymphocyte response to beryllium)A.ACUTE BERYLLIOSIS (25%)√ pulmonary edema following an overwhelming exposureB.CHRONIC BERYLLIOSISwidespread systemic disease of liver, spleen, lymph nodes, kidney, myocardium, skin, skeletal muscle; removed from lungs + excreted via kidneys *Latent period:*5-15 years√ fine nodularity (granulomas similar to sarcoidosis)√ irregular opacities, particularly sparing apices + bases √ hilar + mediastinal adenopathy (may calcify)√ emphysema in upper lobes + interstitial fibrosis√ pneumothorax in 10%HRCT: √ diffuse small parenchymal nodules (57%)√ septal lines (50%)√ patches of ground-glass attenuation (32%)√ hilar adenopathy (21-35%), only in the presence of parenchymal abnormalities√ bronchial wall thickening (46%)√ pleural irregularities (25%)*DDx:*(1)Nodular pulmonary sarcoidosis (indistinguishable)(2)Asbestosis without hilar adenopathy

Notes:

BLASTOMYCOSIS
=NORTH AMERICAN BLASTOMYCOSIS = GILCHRIST DISEASE = CHICAGO DISEASE=rare systemic mixed pyogenic + granulomatous fungal infection*Organism:*soil-born saprophytic dimorphic fungus Blastomyces dermatitidis, mycelial phase in soil + round thick-walled yeast form with broad-based budding in mammals*Geographic distribution:* worldwide; endemic in central + southeastern United States (Ohio + Mississippi river valleys, vicinity of Great Lakes), Africa, Canada (northern Ontario), Central + South America (acquired through activities in woods) *Age:*several months of age to 80 years (peak between 25 and 50 years of age)*Mode of infection:* inhalation of fungal conidia (primary portal of entry); spread to extrapulmonary sites, eg, skin, bone (often direct extension from skin lesion resembling actinomycosis), joints *Predisposed:*elderly, immunocompromised*Histo:* (a)exudative phase: accumulation of numerous neutrophils with infecting organism(b)proliferative phase: proliferation of epitheloid granulomas + giant cells with central microabscesses containing neutrophils and yeast forms ● mouth ulcers ● fever, cough, weight loss, chest pain (majority) ● crusted verrucous lesions on exposed body areas@Lung ● Clinical patterns following pulmonary infection:(a)severe pulmonary symptoms(b)asymptomatic pulmonary infection with spontaneous resolution(c)disseminated disease to single / multiple organs indolent for several years(d)extrapulmonary manifestation involving male GU system, skeleton, skin√ segmental / lobar airspace disease in lower lobes in acute illness (26-61%)√ solitary / multiple irregular nodular masses / satellite lesions in paramediastinal location√ air bronchogram in area of consolidation / mass (87%)√ interstitial disease√ cavitation if communicating with airway (13%)√ hilar / mediastinal lymph node enlargement (<25%)@Bone√ marked destruction ± surrounding sclerosis√ periosteal reaction in long bones, but not in short bones√ multiple osseous lesions are frequent√ vertebral bodies + intervertebral disks are destroyed (similar to tuberculosis)√ psoas abscess√ lytic skull lesions + soft-tissue abscess√ usually monarticular arthritis: knee > ankle > elbow > wrist > hand@GU tract (20%): prostate, epididymis *Dx:*(1)culture of organism(2)silver stain microscopy of tissues*Prognosis:*spontaneous resolution of acute disease in up to 4 weeks; disease may reactivate for up to 3 years*Rx:*(1)amphotericin B IV: 8-10 weeks for noncavitary + 10-12 weeks for cavitary lesions(2)ketoconazole*DDx:*other pneumonias (ie, bacterial, tuberculous, fungal), pseudolymphoma, malignant neoplasm (ie, alveolar cell carcinoma, lymphoma, Kaposi sarcoma)

Notes:

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BONE MARROW TRANSPLANTATION
=intravenous infusion of hematopoietic progenitor cells from patients own marrow (autologous transplant) / HLA-matched donor (allogenic transplant) to reestablish marrow function after high-dose chemotherapy and total body irradiation for lymphoma, leukemia, anemia, multiple myeloma, congenital immunologic defects, solid tumors*Cx:*pulmonary complications in 40-60%

Neutropenic Phase Pulmonary Complications Early Phase Pulmonary Complications Late Phase Pulmonary Complications

Notes:

Neutropenic Phase Pulmonary Complications *Time:*2-3 weeks after transplantation1.Angioinvasive aspergillosis√ nodule surrounded by halo of ground-glass attenuation (= fungal infection spreading into lung parenchyma and surrounding area of hemorrhagic infarction)√ segmental / subsegmental consolidation(= pulmonary infarction)√ cavitation of nodule with air-crescent sign (during recovery phase with resolving neutropenia)√ <5 mm centrilobular nodules to 5 cm peribronchial consolidation (= airway invasion with surrounding zone of hemorrhage / organizing pneumonia)2.Diffuse alveolar hemorrhage (20%) ● hemosiderin-laden macrophages on lavage√ bilateral areas of ground-glass attenuation / consolidation3.Pulmonary edema*Cause:*infusion of large volumes of fluid combined with cardiac + renal dysfunction√ prominent pulmonary vessels, interlobar septal thickening, ground-glass attenuation, pleural effusions4.Drug toxicity*Cause:*bleomycin, busulfan, bischloronitrosurea (carmustine), methotrexate√ bilateral areas of ground-glass attenuation / consolidation / reticular attenuation (= fibrosis)

Notes:

Early Phase Pulmonary Complications *Time:*up to 100 days after transplantation1.CMV pneumonia (23%)√ multiple small nodules + associated areas of consolidation + ground-glass attenuation(= hemorrhagic nodules)2.Pneumocystis carinii pneumonia√ diffuse / predominantly perihilar / mosaic pattern of ground-glass attenuation with sparing of some secondary pulmonary lobules3.Idiopathic interstitial pneumonia (12%)√ nonspecific findings (diagnosis of exclusion)

Notes:

Late Phase Pulmonary Complications *Time:*after 100 days post transplantation1.Bronchiolitis obliterans (in up to 10%)2.BOOP3.Chronic graft-versus-host diseaseinfections, chronic aspiration, bronchiolitis obliterans, lymphoid interstitial pneumonia

Notes:

BRONCHIAL ADENOMA
=misnomer secondary to locally invasive features, tendency for recurrence, and occasional metastasis to extrathoracic sites (10%) = low-grade malignancy*Incidence:*6-10% of all primary lung tumors*Age:*mean age of 35-45 years (range 12-60 years); 90% occur <50 years of age; most common primary lung tumor under age 16; M:F = 1:1; Whites:Blacks = 25:1*Path:*arises from duct epithelium of bronchial mucous glands (predominant distribution of Kulchitsky cells at bifurcations of lobar bronchi)*Types: mnemonic:*"CAMP"**C**arcinoid90%**A**denoid cystic carcinoma = Cylindroma6%**M**ucoepidermoid carcinoma 3%**P**leomorphic carcinoma1%Location:most commonly near / at bifurcation of lobar / segmental bronchi; central:peripheral = 4:1- 48% on right:RLL (20%), RML (10%), RUL (7%), main right bronchus (8%), intermediate bronchus (3%)- 32% on left:LLL (13%), LUL (12%), main left bronchus (6%), lingular bronchus (1%) ● hemoptysis (40-50%) ● atypical asthma ● persistent cough ● recurrent obstructive pneumonia ● asymptomatic (10%) √ complete obstruction / air trapping in partial obstruction (rare) / nonobstructive (10-15%)√ obstructive emphysema√ recurrent postobstructive infection: pneumonitis, bronchiectasis, abscess√ atelectasis / consolidation of a lung / lobe / segment (78%)√ collateral air drift may prevent atelectasis√ solitary round / oval slightly lobulated pulmonary nodule (19%) of 1-10 cm in size√ hilar enlargement / mediastinal widening= central endo- / exobronchial massCT: √ well-marginated sharply defined mass√ in close proximity to an adjacent bifurcation with splaying of bronchus√ coarse peripheral calcifications in 1/3 (cartilaginous / bony transformation)√ may exhibit marked homogeneous enhancementBiopsy:risky secondary to high vascularity of tumor*Prognosis:*95% 5-year survival rate, 75% 15-year survival rate after resection

Carcinoid Cylindroma Mucoepidermoid Carcinoma Pleomorphic Adenoma

Notes:

Carcinoid =NEUROENDOCRINE CARCINOMA=slow-growing low-grade malignant tumor*Incidence:*12-15% of all carcinoid tumors in the body; 1-4% of all bronchial neoplasms*Age peak:*5th decade (range of 2nd-9th decade); 4% occur in children + adolescents; M:F = 2:1; very uncommon in Blacks*Path:* originates from neurosecretory cells of bronchial mucosa (= Kulchitsky cells = argentaffine cells) just as small cell cancer; part of APUD (amine precursor uptake and decarboxylation) system = chromaffin paraganglioma, which produces serotonin, ACTH, norepinephrine, bombesin, calcitonin, ADH, bradykinin *Pathologic classification:* (KCC = **Kulchitsky cell carcinoma**) KCC I=**classic carcinoid** (least aggressive);=bronchial adenoma (misnomer)=central location with endobronchial growth; usually <2.5 cm in size + well-defined; younger patient; M:F = 1:10; lymph node metastases in 3%KCCII=**atypical carcinoid** (25% of carcinoid tumors); mass usually >2.5 cm with well-defined margins; older patient; M:F = 3:1; lymph node metastases in 40-50%; metastases to brain, liver, bone (in 30%)KCCIII=**small cell carcinoma** (most aggressive); mediastinal lymphadenopathy; ill-defined tumor margins◊Rarely cause for carcinoid syndrome or Cushing syndrome! ● recurrent unifocal pneumonitis, hemoptysis ● wheezing, persistent cough, dyspnea, chest pain ● carcinoid syndrome (rare) ● endobronchial exophytic mass at endoscopyLocation:58-90% central in lobar / segmental bronchi, 10-42% peripheral; located in submucosa; endobronchial / along bronchial wall / exobronchial√ polypoid tumor with average size of 2.2 cm√ most extend through bronchial wall thus involving bronchial lumen + parenchyma (= collar button lesion)√ calcification / ossification (26-33%): central carcinoid (43%), peripheral carcinoid (10%)√ vascular tumor supplied by bronchial circulation√ cavitation (rare)√ segmental / lobar atelectasis√ obstructive pneumonitis√ bronchiectasis + pulmonary abscessMalignant potential: low Metastases: (a)regional lymph nodes in 25%(b)distantly in 5% (adrenal, liver, brain, skin, osteoblastic bone metastases)*Prognosis:* 95% 5-year survival rate for classic carcinoids; 57-66% 5-year survival rate for atypical carcinoids

Notes:

Cylindroma =ADENOID CYSTIC CARCINOMA (7%)Second most common primary tumor of trachea *Path:*mixed serous + mucous glands; resembles salivary gland tumor*Histo:* Grade 1:tubular + cribriform; no solid subtype√ entirely intraluminal Grade 2:tubular + cribriform; <20% solid subtype√ predominantly intraluminal Grade 3:solid subtype >20%√ predominantly extraluminal*Age peak:*4th-5th decade ● typical Hx of refractory "asthma" ● hemoptysis, cough, stridor, wheezing ● dysphagia, hoarseness√ endotracheal mass with extratracheal extensionMalignant potential: more aggressive than carcinoid with propensity for local invasion + distant metastases (lung, bone, brain, liver) in 25% *Rx:*tracheal resection + adjunctive radiotherapy*Prognosis:*8.3 years mean survival

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Mucoepidermoid Carcinoma *Path:*squamous cells + mucus-secreting columnar cells; resembles salivary gland tumor√ may involve trachea = locally invasive tumor√ sessile / polyploid endobronchial lesion

Notes:

Pleomorphic Adenoma =MIXED TYPE = extremely rare

Notes:

BRONCHIAL ATRESIA
=local obliteration of proximal lumen of a segmental bronchus*Proposed causes:* (a)local interruption of bronchial arterial perfusion >15 weeks GA (when bronchial branching is complete)(b)tip of primitive bronchial bud separates from bud and continues to develop*Path:*normal bronchial tree distal to obstruction patent and containing mucus plugs; alveoli distal to obstruction air-filled through collateral air drift*Associated with:*lobar emphysema, cystic adenomatoid malformation ● minimal symptoms, apparent later in childhood (most by age 15) / adult lifeLocation:apicoposterior segment of LUL (>>RUL / ML)√ decreased perfusion√ overexpanded segment (collateral air drift with expiratory air-trapping)√ fingerlike opacity lateral to hilum (= mucus plug distal to atretic lumen) is CHARACTERISTICOB-US (detected >24 weeks MA): √ large echogenic fetal lung mass = fluid-filled lung distal to obstruction√ dilated fluid-filled bronchus*Rx:*no treatment because mostly asymptomatic*DDx:*Congenital lobar emphysema (no mucus plug)

Notes:

BRONCHIECTASIS

=localized mostly irreversible dilatation of bronchi often with thickening of the bronchial wall*Etiology:* A.Congenital1.Structural defect of bronchi: bronchial atresia, Williams-Campbell syndrome2.Abnormal mucociliary transport: Kartagener syndrome3.Abnormal secretions: mucoviscidosis = cystic fibrosisB.Congenital / acquired immune deficiency (usually IgG deficiency):chronic granulomatous disease of childhood, alpha 1-antitrypsin deficiency C.Postinfectious: measles, whooping cough, Swyer-James syndrome, allergic bronchopulmonary aspergillosis, chronic granulomatous infection (TB)D.Bronchial obstruction: neoplasm, inflammatory nodes, foreign bodyE.Aspiration / inhalation: gastric contents / inhaled fumes (late complication)F.Pulmonary fibrosis: "**traction bronchiectasis**" due to increased elastic recoil with bronchial dilatation + mechanical distortion of bronchi by fibrosis *Imaging definition on HRCT (modality of choice):* (1)lack of tapering of bronchi (in 80% = most sensitive finding)(2)internal diameter of bronchus larger than adjacent pulmonary artery (in 60%)(3)bronchi visible within 1 cm of pleura (in 45%)(4)mucus-filled dilated bronchi (in 6%) *Classification:* 1.**Cylindrical / tubular / fusiform bronchiectasis** least severe type reversible if associated with pulmonary collapse √ 16 subdivisions of bronchi√ square abrupt ending with lumen of uniform diameter and same width as parent bronchusHRCT (study of choice): √ "tram lines" (horizontal course)√ "signet-ring sign" (vertical course) = cross-section of dilated bronchus + branch of pulmonary artery2.**Varicose bronchiectasis** Rare, associated with Swyer-James syndrome √ 4-8 subdivisions of bronchi√ beaded contour with normal pattern distally3.**Saccular / cystic bronchiectasis** most severe type *Associated with:* severe bronchial infection√ <5 subdivisions of bronchi√ progressive ballooning dilatation toward periphery with diameter of saccules >1 cm√ irregular constrictions may be present√ dilatation of bronchi on inspiration, collapse on expirationHRCT: √ string of cysts = "string of pearls" (horizontal course) / cluster of cysts = "cluster of grapes"√ air-fluid level (frequent)*Age:*predominantly pediatric disease ● chronic cough ● recurrent infection with expectoration of purulent sputum ● shortness of breath ● hemoptysis (50%)*Associated with:*obliterative + inflammatory bronchiolitis (in 85%)Location:posterior basal segments of lower lobes, bilateral (50%), middle lobe / lingula (10%), central bronchiectasis in bronchopulmonary aspergillosis√ normal radiograph in 7%√ increase in size of lung markings (retained secretions)√ loss of definition of lung markings (peribronchial fibrosis)√ crowding of lung markings (if associated with atelectasis)√ cystic spaces ± air-fluid levels <2 cm in diameter (dilated bronchi)√ honeycomb pattern (in severe cases)√ compensatory hyperinflation of uninvolved ipsilateral lung√ increased background density√ frequent exacerbations + resolutions (due to superimposed infections)*Cx:*frequent respiratory infections*DDx of CT appearance:* (1)emphysematous blebs (no definable wall thickness, subpleural location)(2)"reversible bronchiectasis" = temporary dilatation during pneumonia with return to normal within 4-6 months

Notes:

BRONCHIOLITIS OBLITERANS
=CONSTRICTIVE BRONCHIOLITIS = OBLITERATIVE BRONCHIOLITIS =inflammation of bronchioles leading to (sometimes reversible) obstruction of bronchiolar lumen*Etiology:* (1)Inhalation: 1-3 weeks after exposure to toxic fumes (isocyanates, phosgene, ammonia, sulfur dioxide, chlorine)(2)Postinfectious: Mycoplasma (children), virus (older individual); *see Swyer-James syndrome*
(3)Drugs: penicillamine(4)Connective tissue disorder: rheumatoid arthritis, scleroderma, systemic lupus erythematosus(5)Chronic rejection: lung transplant, heart-lung transplant (30-50%)(6)Chronic graft-versus-host disease: bone marrow transplant(7)Cystic fibrosis (as a complication of repeated episodes of pulmonary infection)(8)Idiopathic (in immunocompetent patients)*Path:*submucosal and peribronchiolar fibrosis= irreversible fibrosis of small airway walls with narrowing / obliteration of airway lumina by granulation tissue *Peak age:*40-60 years; M:F = 1:1 ● insidious onset of dyspnea over many months ● obstructive pulmonary function tests ● no response to antibiotics ● persistent nonproductive cough√ normal CXR (in up to 40%)√ hyperinflated lungs = limited disease with connective tissue plugs in airways√ bronchiectasis√ decreased vascularity (reflex vasoconstriction)HRCT (paired expiration-inspiration images: √ "mosaic perfusion" of lobular air trapping (85-100%)=patchy areas of decreased lung attenuation alternating with areas of normal attenuation√ areas of decreased attenuation containing vessels of decreased caliber (due to alveolar hypoventilation + secondary vasoconstriction of alveoli distal to bronchiolar obstruction)√ areas of increased attenuation containing vessels of increased caliber (uninvolved areas with compensatory increased perfusion)√ bronchial wall thickening (87%)√ bronchiectasis (66-80%)√ patchy air trapping on expiratory scans (due to collateral airdrift into postobstructive alveoli) = failure of volume / attenuation change between expiratory + inspiratory images√ "tree-in-bud" appearance of bronchioles = centrilobular branching structures and nodules caused by peribronchiolar thickening + bronchiolectasis with secretions (the only direct, but uncommon sign)√ centrilobular ground-glass opacities*Rx:*steroids may stop progression*DDx:*(1)Bacterial / fungal pneumonia (response to antibiotics, positive cultures)(2)Chronic eosinophilic pneumonia (young female, eosinophilia in 2/3)(3)Usual interstitial pneumonia (irregular opacities, decreased lung volume)

Notes:

BRONCHIOLITIS OBLITERANS WITH ORGANIZING PNEUMONIA (BOOP)
=PROLIFERATIVE BRONCHIOLITIS=CRYPTOGENIC ORGANIZING PNEUMONITIS (COP)*Prevalence:*20-30% of all chronic infiltrative lung disease*Cause:*postobstructive pneumonia, organizing adult respiratory distress syndrome, lung cancer, extrinsic allergic alveolitis, pulmonary manifestation of collagen vascular disease, pulmonary drug toxicity, silo filler disease, idiopathic (50%)*Path:*granulation tissue polyps filling the lumina of alveolar ducts and respiratory bronchioles (bronchiolitis obliterans) + variable degree of infiltration of interstitium and alveoli with macrophages (organizing pneumonia)Bronchiolitis obliterans component not present in up to 1/3!*Histo:*plugs of immature fibroblasts (Masson bodies) covered with low cuboidal epithelium which may spread through collateral air drift pathways*Age:*40-70 years; M:F = 1:1 ● clinical + functional + radiographic manifestation of organizing pneumonia ● nonproductive cough, dyspnea (1-4-month history), preceded by a brief flulike illness with sore throat, low-grade fever, malaise (in 33%) ● late respiratory crackles ● restrictive pulmonary function tests + diminished diffusing capacity on pulmonary function tests ● unresponsive to broad-spectrum antibiotics ● no organism identifiedLocation:mainly mid + lower lung zones; often subpleural (50%) and peribronchiolar distribution (30-50%)CXR: frequently mixture of: √ uni- / bilateral patchy alveolar airspace consolidation (25-73%), often subpleural√ 3-5 mm nodules (up to 50%)√ irregular linear opacities (15-42%)√ unilateral focal / lobar consolidation (5-31%)√ pleural thickening (13%)√ cavitation / pleural effusion (<5%)HRCT: √ patchy airspace consolidation (80%)(a)bilateral in 90% involving all lung zones(b)subpleural distribution in 50-60%√ patchy ground-glass opacities (due to alveolitis) in 60%√ 3-5 mm centrilobular nodules (30-50%) due to organized pneumonia√ air bronchograms = cylindrical bronchial dilatation in areas of airspace consolidation (36-70%)√ pleural effusion (28-35%)√ adenopathy (27%)*Rx:*improvement with corticosteroid therapy (in 84% of patients with idiopathic form)*Prognosis:*persistent abnormalities (30%); 10% mortality due to progressive / recurrent disease*Dx:*tissue examination from open lung biopsy

Notes:

BRONCHIOLOALVEOLAR CARCINOMA
=ALVEOLAR CELL CARCINOMA = BRONCHIOLAR CARCINOMA*Incidence:*1.5-6% of all primary lung cancers (increasing incidence to ? 20-25%)*Etiology:*development from type II alveolar epithelial cells, subtype of adenocarcinoma*Age:*40-70 years; M:F = 1:1 (strikingly high in women)*Path:*peripheral neoplasm arising beyond a recognizable bronchus with tendency to spread locally using lung structure as a stroma (= **lepidic** growth)*Histo:*cuboidal / columnar cells grow along alveolar walls + septa without disrupting the lung architecture or pulmonary interstitium (serving as "scaffolding" for tumor growth); subtype of adenocarcinomaSubtypes: (a)mucinous (80%): mucin-secreting tall columnar peglike bronchiolar cells; more likely multicentric; 26% 5-year survival rate(b)nonmucinous (20%): cuboidal type II alveolar pneumocytes with production of surfactant / nonciliated bronchiolar (Clara) cells; more localized + solitary; 72% 5-year survival rate *Risk factors:*localized pulmonary fibrosis (tuberculous scarring, pulmonary infarct) in 27%, diffuse fibrotic disease (scleroderma), previous exogenous lipid pneumonia ● history of heavy smoking (25-50%) ● often asymptomatic (even with disseminated disease) ● cough (35-60%), hemoptysis (11%) ● bronchorrhea = abundant white mucoid / watery expectoration (5-27%); can produce hypovolemia + electrolyte depletion; unusual + late manifestation only with diffuse bronchioloalveolar carcinoma ● shortness of breath (15%) ● weight loss (13%), fever (8%)Location:peripherally, beyond a recognizable bronchusSpread:tracheobronchial dissemination = cells detach from primary tumor + attach to alveolar septa elsewhere in ipsi- / contralateral lung; lymphogenous + hematogenous dissemination (in 50-60%) A.LOCAL FORM (60-90%)1.Ground-glass attenuation=early stage (due to lepidic growth pattern along alveolar septa with relative lack of acinar filling)√ ground-glass haziness√ bubblelike hyperlucencies / pseudocavitation√ airway dilatation√ lesion persists / progresses within 6-8 weeks2.Single mass (43%)√ well-circumscribed focal mass in peripheral / subpleural location arising beyond a recognizable bronchus√ "rabbit ears" / pleural tags / triangular strand / "tail sign" (55%) = linear strands extending from nodule to pleura (desmoplastic reaction / scarring granulomatous disease / pleural indrawing)√ spiculated margin = sunburst appearance (73%)√ "open bronchus sign" = air bronchogram = tumor / mucus surrounding aerated bronchus ± narrowing / stretching / spreading of bronchi√ pseudocavitation (= dilatation of intact air spaces from desmoplastic reaction / bronchiectasis / focal emphysema) in 50-60%♦2nd most common cell type associated with cavitation after squamous cell√ heterogeneous attenuation (57%)√ confined to single lobe√ rarely evolving into diffuse form√ slowly progressive growth on serial radiographs√ NO atelectasis√ negative FDG PET results in 55%*Prognosis:*70% surgical cure rate for tumor <3 cm; 4-15 years survival time with single nodule B.DIFFUSE FORM = Pneumonic form (10-40%)1.Diffuse consolidation (30%)√ acinar airspace consolidation + air bronchogram + poorly marginated borders√ airspace consolidation may affect both lungs (mucus secretion)√ ± cavitation within consolidation√ "CT angiogram sign" = low-attenuation consolidation does not obscure vessels (mucin-producing subtype)2.Lobar form√ ± expansion of a lobe with bulging of interlobar fissures3.Multinodular form (27%)√ multiple bilateral poorly / well-defined nodules similar to metastatic disease√ multiple poorly defined areas of ground-glass attenuation / consolidation√ pleural effusion (8-10%)*Prognosis:*worse with extensive consolidation / multifocal / bilateral disease; death within 3 years with diffuse disease

Notes:

BRONCHOGENIC CARCINOMA
=LUNG CANCER = LUNG CARCINOMAMost frequent cause of cancer deaths in males (35% of all cancer deaths) and females (21% of all cancer deaths); most common malignancy of men in the world; 6th leading cancer in women worldwide *Prevalence:*in 1991 161,000 new cases; 143,000 deaths*Age at diagnosis:* 55-60 years (range 40-80 years);M:F = 1.4:1 ● asymptomatic (10-50%) usually with peripheral tumors ● symptoms of central tumors: ● cough (75%), wheezing, pneumonia ● hemoptysis (50%), dysphagia (2%) ● symptoms of peripheral tumors: ● pleuritic / local chest pain, dyspnea, cough ● Pancoast syndrome, superior vena cava syndrome ● hoarseness ● symptoms of metastatic disease (CNS, bone, liver, adrenal gland) ● paraneoplastic syndromes ● cachexia of malignancy ● clubbing + hypertrophic osteoarthropathy ● nonbacterial thrombotic endocarditis ● migratory thrombophlebitis ● ectopic hormone production: hypercalcemia, syndrome of inappropriate secretion of antidiuretic hormone, Cushing syndrome, gynecomastia, acromegaly*Types:* 1.**Adenocarcinoma** (50%)◊Most common cell type seen in women + nonsmokers!Intermediate malignant potential (slow growth, high incidence of early metastases) *Histo:*formation of glands / intracellular mucinSubtype:bronchioloalveolar carcinomaLocation:almost invariably develops in periphery; frequently found in scars (tuberculosis, infarction, scleroderma, bronchiectasis) + in close relation to preexisting bullae√ solitary peripheral subpleural mass (52%) / alveolar infiltrate / multiple nodules√ may invade pleura + grow circumferentially around lung mimicking malignant mesothelioma√ upper lobe distribution (69%)√ air broncho- / bronchiogram on HRCT (65%)√ calcification in periphery of mass (1%)√ smooth margin / spiculated margin due to desmoplastic reaction with retraction of pleura 2.**Squamous cell carcinoma** = **epidermoid carcinoma** (30-35%)◊Strongly associated with cigarette smoking*Histo:*mimics differentiation of the epidermis by producing keratin ("epidermoid carcinoma"); central necrosis is common*Histogenesis:*chronic inflammation with squamous metaplasia, progression to dysplasia + carcinoma in situ ● positive sputum cytology◊Most common cell type diagnosed that is radiologically occult! ● hypercalcemia from tumor-elaborated parathyroid hormonelike substance◊Slowest growth rate, lowest incidence of distant metastases(a)Central location within main / lobar / segmental bronchus (2/3)√ large central mass ± cavitation√ distal atelectasis ± bulging fissure (due to mass)√ postobstructive pneumonia◊All cases of pneumonia in adults should be followed to complete radiologic resolution!√ airway obstruction with atelectasis (37%)(b)Solitary peripheral nodule (1/3)√ characteristic cavitation (in 7-10%)◊Squamous cell carcinoma is the most common cell type to cavitate!√ invasion of chest wall◊Squamous cell carcinoma is the most common cell type to cause Pancoast tumor! 3.**Small cell undifferentiated carcinoma** (15%)◊Strongly associated with cigarette smokingRapid growth + high metastatic potential (early metastases in 60-80% at time of diagnosis); should be regarded as systemic disease regardless of stage; virtually never resectable *Path:*arises from bronchial mucosa with growth in submucosa + subsequent invasion of peribronchial connective tissue*Histo:*small uniform oval cells with scant cytoplasm; nuclei with stippled chromatin; numerous mitoses + large areas of necrosis; in 20% coexistent with non-small cell histologic types (most frequently squamous cell)Subtype:oat cell cancer with hyperchromatic nuclei; ? related to Kulchitsky cell carcinomas ● smooth-appearing mucosal surface endoscopically ● ectopic hormone production: Cushing syndrome, inappropriate secretion of ADH◊Most common primary lung cancer causing superior vena caval obstruction (due to extrinsic compression / endoluminal thrombosis / invasion)!Location:90% central within lobar / mainstem bronchus (primary tumor rarely visualized) √ typically large hilar / perihilar mass often associated with mediastinal widening (from adenopathy)√ extensive necrosis + hemorrhage√ small lung lesion (rare)Staging evaluation: CT of abdomen + head, bone scintigraphy, bilateral bone marrow biopsies 4.**Undifferentiated large cell carcinoma** (<5%)◊Strongly associated with smokingIntermediate malignant potential; rapid growth + early distant metastases*Histo:*tumor cells with abundant cytoplasm + large nuclei + prominent nucleoli; diagnosed per exclusion due to lack of squamous / glandular / small cell differentiationSubtype:giant cell carcinoma with very aggressive behavior + poor prognosis√ large bulky usually peripheral mass >6 cm (50%)√ large area of necrosis√ pleural involvement√ large bronchus involved in central lesion (50%)
RISK FACTORS: (1)cigarette smoking (squamous cell carcinoma + small cell carcinoma)-related to number of cigarettes smoked, depth of inhalation, age at which smoking began◊85% of lung cancer deaths are attributable to cigarette smoking!◊Passive smoking may account for 25% of lung cancers in nonsmokers!(2)radon gas: may be the 2nd leading cause for lung cancer with up to 20,000 deaths per year(3)industrial exposure: asbestos, uranium, arsenic, chlormethyl ether(4)concomitant disease: chronic pulmonary scar + pulmonary fibrosis**Scar carcinoma** 7% of lung tumors; 1% of autopsies *Origin:*related to infarcts (>50%), tuberculosis scar (<25%)*Histo:*adenocarcinoma (72%), squamous cell carcinoma (18%)Location:upper lobes (75%)◊45% of all peripheral cancers originate in scars! PRESENTATION √ solitary peripheral mass with corona radiata / pleural tail sign / satellite lesion√ cavitation (16%): usually thick-walled with irregular inner surface; in 4/5 secondary to squamous cell carcinoma, followed by bronchioloalveolar carcinoma√ central mass (38%): common in small cell carcinoma√ unilateral hilar enlargement (secondary to primary tumor / enlarged lymph nodes)Nodes on CT:0-10 mm negative, 10-20 mm indeterminate, >20 mm positive√ anterior + middle mediastinal widening (suggests small cell carcinoma)√ segmental / lobar / lung atelectasis (37%) secondary to airway obstruction (particularly in squamous cell carcinoma)√ "S sign of Golden" = incomplete lobar collapse with bulging contour produced by primary central tumor√ rat tail termination of bronchus√ bronchial cuff sign = focal / circumferential thickening of bronchial wall imaged end-on (early sign)√ local hyperaeration (due to check-valve type endobronchial obstruction, best on expiratory view)√ mucoid impaction of segmental / lobar bronchus (due to endobronchial obstruction)√ persistent peripheral infiltrate (30%) = postobstructive pneumonitis√ NO air bronchogram √ pleural effusion (8-15%)√ bone erosion of ribs / spine (9%)√ involvement of main pulmonary artery (18%); lobar + segmental arteries (53%) may result in additional peripheral radiopacity (due to lung infarct)√ calcification in 7% on CT (histologically in 14%) usually eccentric / finely stippled(a)preexisting focus of calcium engulfed by tumor(b)dystrophic calcium within tumor necrosis(c)calcium deposit from secretory function of carcinoma (eg, mucinous adenocarcinoma)Angio: √ bronchogenic carcinoma supplied by bronchial circulation√ distortion / stenosis / occlusion of pulmonary arterial circulation MULTIPLE PRIMARY LUNG CANCERS *Incidence:*0.72-3.5%; in 1/3 synchronous, in 2/3 metachronous◊10-32% of patients surviving resection of a lung cancer will develop a second primary!*Dx:*biopsy mandatory for proper therapy because the tumor may have a different cell type PARANEOPLASTIC MANIFESTATIONS 1.Carcinomatous neuromyopathy (4-15%)2.Migratory thrombophlebitis3.Hypertrophic pulmonary osteoarthropathy (3-5%)4.Endocrine manifestations (15%) usually with small cell carcinoma: Cushing syndrome, inappropriate secretion of ADH, HPT, excessive gonadotropin secretion LOCATION 60-80% arise in segmental bronchi -central: small cell carcinoma, squamous cell carcinoma (sputum cytology positive in 70%); arises in central airway often at points of bronchial bifurcation, infiltrates circumferentially, extends along bronchial tree-peripheral: adenocarcinoma, large cell carcinoma-upper lobe: lower lobe = right lung : left lung = 3 : 2-most common site: anterior segment of RUL-**Pancoast tumor** (3%) = superior pulmonary sulcus tumor, frequently squamous cell carcinoma ● atrophy of muscles of ipsilateral upper extremity due to lower brachial plexus involvement ● Horner syndrome (enophthalmos, miosis, ptosis, anhidrosis) due to sympathetic chain + stellate ganglion involvement√ apical pleural thickening / mass√ ± soft-tissue invasion / bone destruction-SVC obstruction (5%): often in small cell carcinoma TNM STAGING T1:<3 cm in diameter, surrounded by lung / visceral pleuraT2:>3 cm in diameter / invasion of visceral pleura / lobar atelectasis / obstructive pneumonitis / at least 2 cm from carinaT3:tumor of any size; less than 2 cm from carina / invasion of parietal pleura, chest wall, diaphragm, mediastinal pleura, pericardium; pleural effusionT4:invasion of heart, great vessels, trachea, esophagus, vertebral body, carina / malignant effusionN1:peribronchial / ipsilateral hilar nodesN2:ipsilateral mediastinal nodesN3:contralateral hilar / mediastinal nodes STAGING FOR SMALL CELL LUNG CANCER Limited disease: 1.Primary in one hemithorax2.Ipsilateral hilar adenopathy3.Ipsilateral supraclavicular adenopathy4.Ipsi- and contralateral mediastinal adenopathy5.Atelectasis6.Paralysis of phrenic + laryngeal nerve7.Small effusion without malignant cellsExtensive disease (60-80%): 1.Contralateral hilar adenopathy2.Contralateral supraclavicular adenopathy3.Chest wall infiltration4.Carcinomatous pleural effusion5.Lymphangitic carcinomatosis6.Superior vena cava syndrome7.Metastasis to contralateral lung8.Extrathoracic metastases to bone (38%), liver (22-28%), bone marrow (17-23%), CNS (8-15%), retroperitoneum (11%), other lymph nodes*Prognosis:*7-11 months median survival; 15-20% 2-year disease-free survival rate SPREAD 1.direct local extension2.hematogenous (small cell ca.)3.lymphatic spread (squamous cell ca.); tumor in 10% of normal-sized lymph nodes4.transbronchial spread-least common DISTANT METASTASES @Bone(a)Marrow: in 40% at time of presentation(b)Gross lesions in 10-35%:Location:vertebrae (70%), pelvis (40%), femora (25%)√ osteolytic metastases (3/4)√ osteoblastic metastases (1/4):in small cell carcinoma / adenocarcinoma√ occult metastases in 36% of bone scans@Adrenals: in 37% at time of presentation@Brain: asymptomatic metastases on brain scan in 7% (30% at autopsy), in 2/3 multiple@Kidney, GI tract, liver, abdominal lymph nodes@Lung-to-lung metastases (in up to 10%, usually in late stage)*Cx:* 1.Diaphragmatic elevation (phrenic nerve paralysis)2.Hoarseness (laryngeal nerve involvement, left > right)3.SVC obstruction (5%): lung cancer is cause of all SVC obstructions in 90%4.Pleural effusion (10%): malignant, parapneumonic, lympho-obstructive5.Dysphagia: enlarged nodes, esophageal invasion6.Pericardial invasion: pericardial effusion, localized pericardial thickening / nodular masses*Prognosis:*mean survival time <6 months; 10-15% overall 5-year survival; survival at 40 months: squamous cell 30% > large cell 16% > adenocarcinoma 15% > oat cell 1%*Rx:* (1)Surgical resection for non-small cell histologic typesUnresectable: involvement of heart, great vessels, trachea, esophagus, vertebral body, malignant pleural effusion(2)Adjuvant chemotherapy + radiation therapy in extensive resectable disease(3)Chemotherapy for small cell carcinoma + radiation therapy for bulky disease, CNS metastases, spinal cord compression, SVC obstruction

Notes:

BRONCHOGENIC CYST
=budding / branching abnormality of ventral diverticulum of primitive foregut (ventral segment = tracheobronchial tree; dorsal segment = esophagus) between 26 and 40 days of embryogenesis*Incidence:*most common intrathoracic foregut cyst (54-63% in surgical series)*Histo:*thin-walled cyst filled with mucoid material, lined with columnar respiratory epithelium, mucous glands, cartilage, elastic tissue, smooth muscle ● contains mucus / clear or turbid fluid√ sharply outlined round / oval mass√ may contain air-fluid levelCT: √ cyst contents of water density (50%) / higher density (50%)OB-US: √ single unilocular pulmonary cyst√ echogenic distended lung obstructed by bronchogenic cyst A.MEDIASTINAL BRONCHOGENIC CYST (86%)*Associated with:*spinal abnormalitiesM:F = 1:1 ● usually asymptomatic ● stridor, dysphagiaLocation:pericarinal (52%), paratracheal (19%), esophageal wall (14%), retrocardiac (9%); usually on right√ rarely communicate with tracheal lumen√ may show esophageal compressionB.INTRAPULMONARY BRONCHOGENIC CYST (14%)M > F ● infection (75%) ● dyspnea, hemoptysis (most common)Location:lower:upper lobe = 2:1; usually medial third√ 36% will eventually contain air*DDx:*solitary pulmonary nodule, cavitated neoplasm, cavitated pneumonia, lung abscess

Notes:

BRONCHOPULMONARY DYSPLASIA
=RESPIRATOR LUNG = complication of prolonged respirator therapy of intermittent PEEP with high oxygen concentration = oxygen toxicity + barotrauma Stage I (2-3 days):√ RDS pattern of hyaline membrane diseaseStage II (4-10 days):√ complete opacification with air bronchogram;associated with congestive failure from PDA Stage III(10-20 days):√ "spongy" / "bubbly" coarse linear densities, esp. in upper lobes√ hyperaeration of lung√ lower lobe emphysemaStage IV(after 1 month):√ same pattern;40% mortality if not resolved by 1 month *Cx:*(1)abnormal pulmonary function(2)increased frequency of lower respiratory tract infections*Prognosis:* (1)complete clearing over months / years (1/3)(2)retained linear densities in upper lobe emphysema (29%)*DDx:*(1) Diffuse neonatal pneumonia (2) Meconium aspiration (3) Total anomalous pulmonary venous return (4) Congenital pulmonary lymphangiectasia (5) Cystic fibrosis (6) Idiopathic pulmonary fibrosis (7) Pulmonary interstitial emphysema (8) Wilson-Mikity syndrome

Notes:

BRONCHOPLEURAL FISTULA
=BRONCHOPULMONARY FISTULA=communication between the bronchial system / lung parenchyma + pleural space*Cause:* (a)Trauma1.Complication of resectional surgery (pneumonectomy, lobectomy, bullectomy)2.Blunt / penetrating trauma3.Barotrauma(b)Lung necrosis1.Putrid lung abscess2.Necrotizing pneumonia: Klebsiella, H. influenzae, Staphylococcus, Streptococcus; tuberculosis; fungus; Pneumocystis3.Infarction(c)Airway disease1.Bronchiectasis (very rare)2.Emphysema complicated by pneumonia / pneumothorax(d)Malignancy: lung carcinoma with postobstructive pneumonia / tumor necrosis following therapy ● large / persistent air leak ● acute / chronic empyemaHRCT: √ direct visualization of bronchopleural fistula (in 50%)√ peripheral air + fluid collection (indirect sign) *Dx:*(1) Introduction of methylene blue into pleural space, in 65% dye appears in sputum(2) Sinography (3) Bronchography*Rx:*tube thoracostomy, open drainage, decortication, thoracoplasty, muscle-pedicle closure, transbronchial occlusions

Notes:

BRONCHOPULMONARY SEQUESTRATION
=congenital malformation consisting of(1)nonfunctioning lung segment(2)no communication with tracheobronchial tree(3)systemic arterial supply*Incidence:*0.15-6.4% of all congenital pulmonary malformations; 1.1-1.8% of all pulmonary resections√ usually >6 cm in size√ round / oval, smooth, well-defined solid homogeneous mass near diaphragm with mass effect√ occasionally fingerlike appendage posteriorly + medially (anomalous vessel)√ contrast enhancement of sequestration at the same time as thoracic aorta on rapid sequential CT scans√ multiple / single air-fluid levels if infected√ surrounded by recurrent pulmonary consolidation in a lower lobe that never clears completely√ may communicate with esophagus / stomach◊Pulmonary sequestration with communication to GI tract is termed **bronchopulmonary foregut malformation**!*DDx:*bronchiectasis, lung abscess, empyema, bronchial atresia, congenital lobar emphysema, cystic adenomatoid malformation, intrapulmonary bronchogenic cyst, Swyer-James syndrome, pneumonia, arteriovenous fistula, primary / metastatic neoplasm, hernia of Bochdalek

Bronchopulmonary Sequestrations

	INTRALOBAR	EXTRALOBAR
Prevalence	75%	25%
Pleural investment	visceral pleura	own pleura
Venous drainage	pulmonary veins	systemic veins
Symptomatic	adulthood	first 6 month
Etiology	acquired	developmental
Congen. anomalies	15%	50%

Intralobar Sequestration (75-86%) Extralobar Sequestration (14-25%)

Notes:

Intralobar Sequestration (75-86%)
=enclosed by visceral pleura of affected pulmonary lobe but separated from bronchial tree*Etiology:*controversial(1)probably acquired in majority of patients(2)early appearance of congenital accessory tracheobronchial bud leads to incorporation within one pleural investment*Path:*chronic inflammation fibrosis: multiple irregular cordlike adhesions to mediastinum, diaphragm, parietal pleura; multiple cysts filled with fluid / thick gelatinous / purulent material; vascular sclerosis*Age at presentation:*adulthood (50% >20 years); M:F = 1:1*Associated with congenital anomalies in 6-12%:* skeletal deformities (4%): scoliosis, rib + vertebral anomalies; esophagobronchial diverticula (4%); diaphragmatic hernia (3%); cardiac (including tetralogy of Fallot); renal: failure of ascent + rotation; cerebral anomalies; congenital pulmonary venolobar syndrome ● about 50% have symptoms by age 20; asymptomatic in 15% ● pain, repeated infection in same location (eg, recurrent acute lower lobe pneumonias) ● high-output congestive heart failure (in neonatal period) from L-to-L shunt ● cough + sputum production, hemoptysisLocation:posterobasal segments, rarely upper lung / within fissure; L:R = 3:2CXR: √ recurrent / persistent pneumonia localized to lower lobe√ cavitation and cysts ± fluid levels◊Aeration of sequestered lung via Kohn pores / communication with tracheobronchial tree!Bronchogram: √ NO communication of rudimentary bronchial system of sequestration with tracheobronchial tree (rare exceptions)Angio: √ usually single large artery (mean diameter of 6 mm) coursing through inferior pulmonary ligament from-distal thoracic aorta (73%)-proximal abdominal aorta (22%)-celiac / splenic artery-intercostal artery (4%)-anomalous branch of coronary artery√ multiple aa. in 16% (with vessel diameter of <3 mm)√ combined systemic + pulmonary arterial supply√ venous drainage via-normal pulmonary veins to L atrium (in 95%)-azygos / hemiazygos vv. / intercostal vv. / SVC into R atrium (in 5%)CT: √ single / multiple thin-walled cysts containing fluid / mucus / pus / air-fluid level / air alone√ mucus-impacted ectatic bronchi (= fat density) in sequestered lung√ emphysema bordering normal lung (37%)=postobstructive hyperinflation of sequestered lung√ homogeneous / inhomogeneous soft-tissue mass with irregular borders√ irregular enhancement (rare)√ one / two anomalous systemic arteries arising from aorta (DDx: AVM, interrupted pulmonary artery, isolated anomaly, chronic infection / inflammation of lung or pleura, surgically created shunt)√ premature atherosclerosis of anomalous arteries◊Mucoid impaction of bronchus surrounded by hyperinflated lung is CHARACTERISTIC!OB-US: √ spherical homogeneous highly echogenic mass√ anomalous systemic artery seen by color Doppler*Cx:*massive spontaneous nontraumatic pleural hemorrhage, chronic inflammation, fibrosis *DDx of mass:*neurogenic tumor, lateral thoracic meningocele, extramedullary hematopoiesis, pleural tumor*DDx of cavity:*lung abscess, necrotizing pneumonia, fungal / mycobacterial pneumonia, cavitating neoplasm, empyema*DDx of cysts:*pulmonary abscess, empyema, bronchiectasis, emphysema, bronchogenic foregut cyst, pericardial cyst, eventration of diaphragm, congenital cystic malformation

Notes:

Extralobar Sequestration (14-25%)
=accessory lobe with its own pleural sheath(= "Rokitansky lobe"), which prevents collateral air drift resulting in an airless round mass *Etiology:*development of an anomalous accessory / supernumerary tracheobronchial foregut bud*Path:*single ovoid / rounded / pyramidal airless lesion between 0.5 and 15 cm (generally 3 to 6 cm) in size*Histo:*resembles normal lung with diffuse dilatation of bronchioles + alveolar ducts + alveoli; dilatation of subpleural + peribronchiolar lymph vessels; covered by mesothelial layer overlying fibrous connective tissue; congenital cystic adenomatoid malformation type II is present in 15-25%*Incidence:*0.5-6% of all congenital lung lesions*Age:*neonatal presentation; 61% within first 6 months of life; occasionally in utero; M:F = 4:1*Associated with congenital anomalies in 15-65%:* @Lung: congenital diaphragmatic hernia (20-30%), eventration / diaphragmatic paralysis (up to 60%), cystic adenomatoid malformation (15-25%), lobar emphysema, bronchogenic cyst, pectus excavatum, congenital pulmonary venolobar syndrome◊May coexist / form part of spectrum with CAM@Heart: anomalous pulmonary venous return, cardiac / pericardial anomalies (8%)@GI tract: epiphrenic diverticula (2%), TE fistula (1.5%), duplication of GI tract, ectopic pancreas@Others: renal anomaly, vertebral anomaly ● respiratory distress + cyanosis + CHF in newborn (due to shunting of blood) ● feeding difficulties ● asymptomatic (rarely becomes infected) in 10% Location:L:R = 4:1; typically within pleural space in posterior costodiaphragmatic sulcus between diaphragm + lower lobe (63-77%); mediastinum; within pericardium; within / below diaphragm (5-15%) √ airless (NO communication with bronchial tree);in presence of air connection with GI tract is inferred√ may contain cystic areas√ mediastinal shift (if large)Angio (diagnostic): √ arterial supply from-aorta as single / several small branches (80%)-splenic, gastric, subclavian, intercostal branches (15%)-pulmonary artery (5%)√ venous drainage via-systemic veins (80%) to R heart (IVC, azygos, hemiazygos, SVC, portal vein)-pulmonary vein (25%)CXR: √ single well-defined homogeneous triangular mass (most commonly located adjacent to posterior medial hemidiaphragm)√ NO air bronchograms√ small "bump" on hemidiaphragm / inferior paravertebral region√ opaque hemithorax ± ipsilateral pleural effusion (if sequestration large)√ ± air-fluid levelCT: √ homogeneous well-circumscribed soft-tissue density mass (no bronchial communication)NUC (radionuclide angiography): √ lack of perfusion during pulmonary phase followed by rapid perfusion in systemic phase*DDx:*intrathoracic kidney, scimitar syndrome (with systemic supply to affected lung), hepatic herniation through diaphragmOB-US: ◊The vast majority in fetuses are extralobar!√ conical / triangular homogeneous highly echogenic mass (many interfaces from multiple microscopically dilated structures)√ color duplex may demonstrate vascular supply√ polyhydramnios (? esophageal compression, excessive fluid secretion by sequestration)√ fetal hydrops (? venous compression)√ edema, ascites√ hydrothorax (obstructed lymphatics + veins in torsed sequestration)*DDx for chest lesion:* congenital cystic adenomatoid malformation, neuroblastoma, teratoma, diaphragmatic hernia *DDx for infradiaphragmatic lesion:* neuroblastoma, teratoma, adrenal hemorrhage, mesoblastic nephroma, foregut duplication *Cx:*infection (in cases of communication with bronchus / GI tract)*Rx:*resection (delineation of vascular supply helpful)*Prognosis:*favorable (worse if pulmonary hypoplasia present); decreases in size / disappears in up to 65% before birth ***Esophageal /*** Gastric Lung =rare variant of pulmonary sequestration*Age:*infancy (as it is symptomatic) ● cough related to feeding ● recurrent pulmonary infections√ communication of bronchial tree of sequestered lung with esophagus / stomach

Notes:

CANDIDIASIS
*Organism:*ubiquitous human saprophyte (Candida albicans most commonly) characterized by blastospheres (yeasts) admixed with hyphae / pseudohyphae (conventional stains)*At risk:*patient with lymphoreticular malignancy*Entry:*(a)aspiration(b)hematogenous dissemination from GI tract / infected central venous catheter
● prolonged fever despite broad-spectrum antibacterial coverage ● cough, hemoptysis√ patchy airspace consolidation in lower lobe distribution√ interstitial pattern√ diffuse micro- / macronodular disease√ pleural effusion (25%)

CASTLEMAN DISEASE Localized / Unicentric Angiofollicular Lymph Node Hyperplasia Generalized / Multicentric Angiofollicular Lymph Node Hyperplasia

Notes:

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CASTLEMAN DISEASE
= ANGIOFOLLICULAR LYMPH NODE HYPERPLASIA =GIANT LYMPH NODE HYPERPLASIA= ANGIOMATOUS LYMPHOID HAMARTOMA= LYMPHOID HAMARTOMA=benign masses of lymphoid tissue of unknown etiologySize:up to 16 cm in diameterCT: √ well-defined mass of muscle density√ spotty central calcification√ enhancing rim (vascular capsule)√ marked enhancement almost equal to aorta (in hyalin-vascular type)√ slight enhancement (in plasma cell type)Angio: √ mass with multiple feeding vessels√ dense homogeneous blush (hyalin-vascular type)√ some hypervascularity (plasma cell type)*DDx:*indistinguishable from lymphoma

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Notes:

Localized / Unicentric Angiofollicular Lymph Node Hyperplasia A.Hyaline-vascular type (76-91%)*Cause:*chronic antigenic stimulation / developmental abnormality of lymphoid tissue*Age:*4th decade; M:F = 1:1*Path:*vascular proliferation + hyalinization with small follicle centers penetrated by capillaries, capillary proliferation in interfollicular areasLocation:mediastinal + cervical lymph nodes ● asymptomatic in 97% B.Plasma cell type (10-24%)*Cause:*chronic viral antigenic stimulation*Average age:*22 years; M:F = 1:1*Path:*sheets of plasma cells between normal / enlarged folliclesLocation:mesenteric + retroperitoneal lymph nodes ● cough, dyspnea, hemoptysis ● lassitude, weight loss, fever ● growth retardation ● elevated sedimentation rate ● IgG, IgM, IgA hypergammaglobulinemia (50%) ● refractory microcytic anemia*Prognosis:*treatment ~100% curative*Rx:*(1)complete surgical resection(2)radiation + steroid therapy

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Notes:

Generalized / Multicentric Angiofollicular Lymph Node Hyperplasia A.HYPERPLASIA WITHOUT NEUROPATHY*Cause:*disordered immunoregulation with polyclonal plasma cells from viral infection*Mean age:*57 years; M>F ● fatigue, anorexia, skin lesions, CNS disorders√ peripheral multicentric adenopathy√ hepatosplenomegaly√ salivary gland enlargement√ ± pulmonary lesions*Rx:*systemic chemotherapy + corticosteroids + irradiation*Prognosis:*mean survival of 27 months B.HYPERPLASIA WITH NEUROPATHY*Cause:*immunoregulatory deficits with uncontrolled B-cell proliferation + interleukin-6 dysregulation*Mean age:*40-60 years; M:F = 2:1 ● skin lesions: hypertrichosis, hirsutism, sclerodermatous thickening, hyperpigmentation, hemangiomas ● distal symmetric sensorimotor neuropathy (50%) ● papilledema, pseudotumor cerebri (66%) ● monoclonal IgG (75%)*Rx:*surgical resection, irradiation, chemotherapy*Prognosis:*mean survival of 24-33 months

Notes:

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CHRONIC EOSINOPHILIC PNEUMONIA
=numerous eosinophils, macrophages, histiocytes, lymphocytes, PMNs within lung interstitium + alveolar sacs*Etiology:*unknown*Age:*middle-age; M < F ● common history of atopia (may occur during therapeutic desensitization procedure) ● adult onset asthma (wheezing) ● high fever, malaise, dyspnea (DDx to Löffler syndrome) ● peripheral blood eosinophilia (with rare exceptions)√ homogeneous alveolar lung infiltrates with distribution at lung periphery = "photographic negative" of pulmonary edema√ frequently bilateral nonsegmental√ unchanged for many days / weeks (DDx to Löffler syndrome)√ fast regression of infiltrates under steroids*Rx:*dramatic response to steroid therapy (within 3-10 days)

Notes:

CHRONIC MEDIASTINITIS
Etiology: (1)Granulomatous infection: histoplasmosis (most frequent), tuberculosis, actinomycosis, Nocardia(2)Mediastinal granuloma(3)Fibrosing mediastinitis(4)Radiation therapy

Mediastinal Granuloma Fibrosing Mediastinitis

Notes:

Mediastinal Granuloma =relatively benign massive coalescent adenitis with caseating / noncaseating lesions*Cause:*primary lymph node infection (commonly tuberculosis / histoplasmosis)*Histo:*thin fibrous capsule surrounding granulomatous lesion√ lymphadenopathy*DDx:*fibrosing mediastinitis (infiltrative, rare)

Notes:

Fibrosing Mediastinitis =SCLEROSING MEDIASTINITIS = MEDIASTINAL COLLAGENOSIS=diffuse fibrotic infiltration throughout mediastinum*Cause:*abnormal host immune response to Histoplasma antigen (organisms recovered in 50%); autoimmune disease, methysergide-induced*May be associated with:* retroperitoneal fibrosis, orbital pseudotumor, Riedel struma*Histo:*infiltrative, often invasive fibrotic process with minimal / no apparent granulomatous fociAge:2nd-5th decade of life ● cough, dyspnea, hemoptysis ● dysphagia ● superior vena cava syndrome ● cor pulmonale (secondary to pulmonary arterial hypertension caused by compression of pulmonary arteries / veins)Location:upper half of mediastinum in paratracheal region + anterior to trachea + near hilumSite:right > left√ widening of upper mediastinum √ lobulated (in 86% calcified) paratracheal / hilar massNUC: √ decreased / absent perfusion with normal ventilation*Cx:*(1)Compression of SVC (64%) + pulmonary veins (4%)(2)Chronic obstructive pneumonia (narrowing of trachea / central bronchi) in 5%(3)Esophageal stenosis (3%)(4)Pulmonary infarcts + fibrosis (narrowing of pulmonary artery)(5)Prominent intercostal arteries (narrowing of pulmonary artery)*DDx:*(1)Swyer-James syndrome(2)Congenital absence of pulmonary artery(3)Embolus to main pulmonary artery(4)Bronchogenic carcinoma(5)Lymphoma(6)Metastatic carcinoma

Notes:

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CHURG-STRAUSS SYNDROME
=variant of polyarteritis nodosaCLASSIC TRIAD: (1)Allergic rhinitis and asthma(2)Eosinophilic infiltrative disease(a)eosinophilic pneumonia(b)eosinophilic gastroenteritis(3)Systemic small-vessel vasculitis with granulomatous inflammationusually develops within 3 years of onset of asthma ● ANCA (antineutrophil cytoplasmic autoantibodies) in 70% ● eosinophilia (almost 100%): peripheral eosinophilia in >30%@Kidney: less frequent + less severe renal disease compared with Wegener granulomatosis + microscopic polyangiitis@Heart: coronary arteritis, myocarditis (accounting for 50% of deaths)@CNS: neuropathy

Notes:

CHYLOTHORAX
=leakage of chyle (= lymph containing chylomicrons= suspended fat) from thoracic duct or its branches into pleural space secondary to obstruction / disruption of thoracic duct (in 2%) *Route of* ***thoracic duct****:* Origin:arises from cisterna chyli anterior to L1/2 (10-15 mm in diameter and 5-7 cm long)Course:enters thorax through aortic hiatus; ascends in right prevertebral location (between azygos vein + descending aorta); swings to left at T4-6 posterior to esophagus; ascends for a short distance along right of aorta; crosses behind aortic arch; runs ventrally at T3 between left common carotid artery + left subclavian arteryTermination:3-5 cm above clavicle at venous angle(= junction of left subclavian + internal jugular veins) Variation:two (33%) or more (in up to 50%) main ducts each consisting of up to 8 separate channels*Etiology:* A.Developmental defects1.Thoracic duct atresia2.Lymphangiectasia3.Lymphangioma4.Lymphangiomatosis (rare): mediastinal / thoracic cystic hygroma of neck growing into mediastinum5.Lymphangioleiomyomatosis ± tuberous sclerosisB.Trauma1.Closed / penetrating chest trauma / birth trauma (25%): latent period of 10 days2.Surgery (2nd most common cause): esophagectomy / cardiovascular surgery, esp. coarctation repair (0.5%), retroperitoneal surgery, neck surgery3.Subclavian venous catheterC.Neoplasm (54%)1.Lymphoma (most common cause)2.Metastatic cancerD.Fibrosing conditions1.Mediastinitis2.Tuberculosis3.Filariasis (rare)E.Obstruction of central venous system / thoracic ductF.Idiopathic / cryptogenic (15%): most common cause in neonatal periodG.Transdiaphragmatic passage of chylous ascites *Age:*in full-term infants; may be present in utero;M:F = 2:1*Incidence:*1:10,000 deliveries*May be associated with:* Trisomy 21, TE-fistula, extralobar lung sequestration, congenital pulmonary lymphangiectasia ● high in neutral fat + fatty acid (low in cholesterol): ● triglyceride level >110 mg/dL ● milky viscoid fluid (chylomicrons) after ingestion of milk / formula and clear during fasting√ usually unilateral loculated pleural effusion(a)right chylothorax due to duct disruption inferior to T5-6 (more common)(b)left-sided chylothorax if duct disrupted above T5-6√ low attenuation (fat) / high attenuation (protein content)√ ± leakage of lymphangiographic contrast√ polyhydramnios (? result of esophageal compression)*Cx:*(1)Pulmonary hypoplasia(2)Hydrops (congestive heart failure secondary to impaired venous return)*Rx:*(1)Thoracentesis (leading to loss of calories, lymphocytopenia, hypogammaglobulinemia)(2)Total parenteral nutrition(3)Thoracic duct ligation (if drainage exceeds 1500 mL/day for adults or 100 mL/yr-age/day for children >5 years of age; drainage >14 days)(4)Pleuroperitoneal shunt; tetracycline pleurodesis; mediastinal radiation; intrapleural fibrin glue; pleurectomy

Notes:

COAL WORKERS PNEUMOCONIOSIS
=CWP = ANTHRACOSIS = ANTHRACOSILICOSIS=coal dust inhalation taken up by alveolar macrophages, in part cleared by mucociliary action (particle size >5 µ), in part deposited around bronchioles + alveoli, coal dust in itself is inert, but admixed silica is fibrogenic **Simple CWP** =aggregates of coal dust = coal macules(usually <3 mm) NO progression in absence of further exposure *Histo:*development of reticulin fibers associated with bronchiolar dilatation (focal emphysema) + bronchiolar artery stenosis (decreased capillary perfusion) ● poor correlation between symptoms, physiologic findings + roentgenogram ● small round 1-5 mm opacities, frequently in upper lobes (radiographically only seen through superposition after an exposure of >10 years) ● nodularity correlates with amount of collagen (NOT amount of coal dust)*Cx :*(1)Chronic obstructive bronchitis(2)Focal emphysema(3)Cor pulmonale

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Notes:

COCCIDIOIDOMYCOSIS
*Organism:*dimorphic soil fungus Coccidioides immitis; arthrospores in desert soil spread by wind aerosolized in dry dust; highly infectious*Geographic distribution:* endemic in southwest desert of USA (San Joaquin Valley, central southern Arizona, western Texas, southern New Mexico) + northern Mexico + in parts of Central + South America; similar to histoplasmosis *Mode of infection:*deposited in alveoli after inhalation + maturation into large thick-walled spherules with release of hundreds of endospores*Dx:*(1)culture of organism(2)spherules in pathologic material (demonstrated with Gomori-methenamine silver stain)(3)positive skin test(4)complement fixation titer

Primary Coccidioidomycosis Chronic Respiratory Coccidioidomycosis Disseminated Coccidioidomycosis (in 1%)

Notes:

Primary Coccidioidomycosis =ACUTE RESPIRATORY COCCIDIOIDOMYCOSIS ● 60-80% asymptomatic ● "valley fever" = influenza-like symptoms ● desert rheumatism (33%) = immune-complex-mediated arthritis (most commonly in ankle) ● rash, erythema nodosum / multiforme (5-20%)√ segmental / lobar consolidation√ patchy infiltrates mainly in lower lobes (46-80%) frequently subpleural + abutting fissures√ peribronchial thickening√ hilar adenopathy (20%)√ pleural effusion (10%)

Notes:

Chronic Respiratory Coccidioidomycosis *Prevalence:*5% of infected patients ● symptoms of postprimary tuberculosis ● hemoptysis in 50%√ one / several well-defined nodules (= coccidioidomycoma) of 5-30 mm in size (in 5%)√ persistent / progressive consolidation√ "grape skin" thin-walled cavities (in 10-15%), in 90% solitary, 70% in anterior segment of upper lobes (DDx: TB), 3% rupture into pleural space due to subpleural location (pneumothorax / empyema / persistent bronchopleural fistula)√ bronchiectasis√ mediastinal adenopathy (10-20%)

Notes:

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Disseminated Coccidioidomycosis (in 1%) =secondary phase of hematogenous spread to meninges, bones, skin, lymph nodes, subcutaneous tissue, joints (except GI tract) ● skin granulomas / abscesses√ micronodular "miliary" lung pattern√ pericardial effusion

Notes:

CONGENITAL LOBAR EMPHYSEMA
=progressive overdistension of one / multiple lobesM:F = 3 :1 *Etiology:* (a)deficiency / dysplasia / immaturity of bronchial cartilage(b)endobronchial obstruction (mucosal fold / web, prolonged endotracheal intubation, inflammatory exudate, inspissated mucus)(c)bronchial compression (PDA, aberrant left pulmonary artery, pulmonary artery dilatation)(d)polyalveolar / macroalveolar hyperplasia*Associated with:*CHD in 15% (PDA, VSD) ● respiratory distress (90%) + progressive cyanosis within first 6 months of lifeLocation:LUL (42-43%), RML (32-35%), RUL (20%), two lobes (5%)√ hazy masslike opacity immediately following birth (delayed clearance of lung fluid in emphysematous lobe over 1-14 days)√ air trapping√ hyperlucent expanded lobe (after clearing of fluid)√ compression collapse of adjacent lobes√ contralateral mediastinal shift√ widely separated vascular markings*Mortality:*10%*Rx:*surgical resection

Notes:

CONGENITAL LYMPHANGIECTASIA

1.PRIMARY PULMONARY LYMPHANGIECTASIA (2/3)=abnormal development of lungs between 14-20th week of GA characterized by anomalous dilatation of pulmonary lymph vessels*Path:*subpleural cysts, ectatic tortuous lymph channels in pleura, interlobular septa + along bronchoarterial bundles; NO obstruction*Age:*usually manifest at birth; 50% stillborn; M = F*May be associated with:*total anomalous pulmonary venous return, hypoplastic left heart, Noonan syndrome ● respiratory distress within few hours of birthSite:diffuse involvement of both lungs, occasionally only in one / two lobes (with good prognosis)√ marked prominence of coarse interstitial markings (simulating interstitial edema)√ hyperinflation√ scattered radiolucent areas (dilated airways)√ patchy areas of pneumonia + atelectasis√ pneumothorax*Prognosis:*in diffuse form invariably fatal at <2 months of age2.GENERALIZED LYMPHANGIECTASIA=DIFFUSE LYMPHANGIOMA=proliferation of mainly lymphatic vascular spaces with relentless systemic progression *Age:*children, young adultsLocation:widespread visceral + skeletal involvement√ diffuse pulmonary interstitial disease√ chylous effusions in pleural + pericardial spaces√ ± lytic bone lesions√ lymphangiographic pooling of contrast material in dilated lymphatic channels / lymph nodes 3.LOCALIZED LYMPHANGIOMA=rare benign usually cystic lesion*Histo:*collection of dilated + proliferated lymph vessels (? hamartoma / benign neoplasm / focal sequestration of ectatic lymph tissue)*Age:*first 3 years of life; M = F ● asymptomatic (33%) ● dyspnea (from tracheal compression)Location:neck (80%), mediastinum, axilla, extremity√ discrete featureless mass√ may have chylous / pleural effusion√ may have lytic lesion in contiguous skeleton*Prognosis:*propensity for local recurrence*DDx:*hemangioma 4.SECONDARY LYMPHANGIECTASIASecondary to elevated pulmonary venous pressure in CHD (TAPVR)

Notes:

CONGENITAL PULMONARY VENOLOBAR SYNDROME
=unique form of lung hypoplasia / aplasia affecting one / more lobes in a constellation of distinctly different congenital anomalies of the thorax that often occur together; M:F = 1:1.4A.MAJOR COMPONENTS1.Hypogenetic lung (69%): lobar agenesis / aplasia / hypoplasia2.Partial anomalous pulmonary venous return (31%)=scimitar syndrome3.Absence of pulmonary artery (14%)4.Pulmonary sequestration (24%)5.Systemic arterialization of lung without sequestration (10%)6.Absence / interruption of inferior vena cava (7%)7.Duplication of diaphragm = accessory diaphragm (7%)=thin membrane in right hemithorax fused anteriorly with the diaphragm coursing posterosuperiorly to join with the posterior chest wall + trapping all / part of RML / RLL√ accessory fissurelike oblique line above right posterior costophrenic sinus (if trapped lung is aerated)√ solid mass along posterior right hemidiaphragm (if trapped lung is unaerated)CT: √ ovoid area of increased density in posterior right hemithorax (= dome of accessory diaphragm) B.MINOR COMPONENTS1.Tracheal trifurcation (extremely rare): 2 mainstem bronchi supply the right lung2.Eventration of diaphragm3.Partial absence of diaphragm4.Phrenic cyst5.Horseshoe lung6.Esophageal / gastric lung7.Anomalous superior vena cava8.Absence of left pericardium√ The most constant components of the syndrome are hypogenetic lung + PAPVR! *Associated with:* (1)Vascular anomalies: hypoplastic artery, anomalous venous return, systemic arterial supply(2)Anomalies of hemidiaphragm on affected side:√ retrosternal band on lateral CXR due to mediastinal rotation √ phrenic cyst√ diaphragmatic hernia√ accessory hemidiaphragm(3)Hemivertebrae + scoliosis(4)CHD (25-50%): secundum-type ASD, VSD, tetralogy of Fallot, PDA, coarctation of aorta, hypoplastic left heart, double-outlet right ventricle, double-chambered right atrium, endocardial cushion defect, persistent left SVC, pulmonary stenosis ● asymptomatic (40%) ● may have dyspnea / recurrent infectionsLocation:right-sided predominance; M:F = 1.0:1.4√ hypoplasia / aplasia of one / more lobes of the lung with errors of lobation (bilateral left bronchial branching pattern / horseshoe lung)√ "scimitar vein" (90%) = partial anomalous pulmonary venous return (commonly infradiaphragmatic into IVC / portal vein / hepatic vein / R atrium), on CXR seen only in 1/3√ systemic arterial supply to abnormal segment my be present from thoracic aorta (bronchial, intercostal, transpleural) or abdominal aorta (celiac artery, transdiaphragmatic)√ reticular densities (enlarged bronchial / transpleural arterial collaterals)√ small hilus (absent / small pulmonary artery)√ small right hemithorax + mediastinal shift√ haziness of right heart border√ cardiac dextroposition (in right lung hypoplasia)√ anomalies of bony thorax / thoracic soft tissues√ absent inferior vena cava√ rib hypoplasia / malsegmentation√ rib notchingCT: √ small hemithorax + mediastinal shift√ abnormalities of bronchial branching√ anomalously located pulmonary fissure√ discontinuity of hemidiaphragm√ pulmonary arterial hypoplasia√ hyparterial right bronchus (instead of eparterial)√ one / more vessels increasing in diameter toward diaphragm√ rind of subpleural fatty tissue in affected hemithorax√ lack of normal venous confluence of right lung *DDx:*meandering pulmonary vein, dextrocardia, hypoplastic lung, Swyer-James syndrome

Notes:

CRYPTOCOCCOSIS
=TORULOSIS = EUROPEAN BLASTOMYCOSIS*Organism:*encapsulated unimorphic yeastlike fungus Cryptococcus neoformans; spherical single-budding yeast cell with thick capsule, stains with India ink; often in soil contaminated with pigeon excreta*Histo:*granulomatous lesion with caseous necrotic center*Predisposed:*opportunistic invader in diabetics + immunocompromised patients ● low-grade meningitis (affinity to CNS); M:F = 4:1@Lung√ well-circumscribed mass (40%) of 2-10 cm in diameter, usually peripheral location√ lobar / segmental consolidation (35%)√ cavitation (15%)√ hilar / mediastinal adenopathy (12%)√ calcifications (extremely rare)√ interstitial pneumonia (rare, in AIDS patients)@Musculoskeletal√ osteomyelitis (5-10%)√ arthritis (rare, usually from extension of osteomyelitis)

Notes:

CYSTIC ADENOMATOID MALFORMATION

=CAM = congenital cystic abnormality of the lung characterized by an intralobar mass of disorganized pulmonary tissue communicating with bronchial tree + having normal vascular supply + drainage but delayed clearance of fetal lung fluid*Incidence:*25% of congenital lung disorders;95% of congenital cystic lung lesions *Cause:*arrest of normal bronchoalveolar differentiation between 5th-7th week of gestation with overgrowth of terminal bronchioles*Path:*proliferation of bronchial structures at the expense of alveolar saccular development, modified by intercommunicating cysts of various size (adenomatoid overgrowth of terminal bronchioles, proliferation of smooth muscle in cyst wall, absence of cartilage)TYPE I(50%):*Histo:*single / multiple large cyst(s) >20 mm lined by ciliated pseudostratified columnar epithelium, mucus-producing cells in 1/3*Prognosis:*excellent following resectionTYPE II(40%):*Histo:*multiple cysts 5-12 mm lined by ciliated cuboidal / columnar epithelium*Prognosis:*poor secondary to associated abnormalitiesTYPE III(10%):*Histo:*solitary large bulky firm mass of bronchuslike structures lined by ciliated cuboidal epithelium with 3-5 mm small microcysts*Prognosis:*poor secondary to pulmonary hypoplasia / hydrops *In 25% associated with:* cardiac malformation, pectus excavatum, renal agenesis, prune-belly syndrome, jejunal atresia, chromosomal anomaly, bronchopulmonary sequestration *Age of detection:*children, neonates, fetus; M:F = 1:1 ● respiratory distress + severe cyanosis in first week of life (66%) / within first year of life (90%) due to compression of normal lung + airways ● superimposed chronic recurrent infection (10%) after first year of lifeLocation:equal frequency in all lobes (middle lobe rarely affected); more than one lobe involved in 20%; mostly unilateral without side preferenceCXR: √ almost always unilateral expansile mass with well-defined margins (80%)√ multiple air- / occasionally fluid-filled cysts√ sometimes solid appearance (retained fetal lung fluid / type III lesion)√ compression of adjacent lung√ contralateral shift of mediastinum (87%)√ hypoplastic ipsilateral lung√ proper position of abdominal viscera√ spontaneous pneumothorax (late sign)CT: ◊Postnatally becoming obstructed and filled with air√ solitary / multiple fluid or air-fluid filled cysts with thin walls√ surrounding focal emphysematous changesOB-US: √ single large cyst / multiple large cysts of 2-10 cm in diameter (Type I)√ multiple small cysts of 5-12 mm in diameter (Type II)√ large homogeneously hyperechoic mass compared to liver (Type III)√ contralateral mediastinal shift (89%)√ polyhydramnios (25-75%, ? from compression of esophagus or increased fluid production by abnormal lung) / normal fluid (28%) / oligohydramnios (6%)√ fetal ascites (62-71%)√ fetal hydrops in 33-81% (decreased venous return from compression of heart / vena cava)*Risk of recurrence:*none*Cx:*ipsi- / bilateral pulmonary hypoplasia*Prognosis:*50% premature, 25% stillborn◊Polyhydramnios, ascites, hydrops indicate a poor outcome!◊CAM becomes smaller in fetuses in many cases + occasionally almost disappears by birth!*DDx:*(1)Congenital lobar emphysema(2)Diaphragmatic hernia(3)Bronchogenic cyst (small solitary cyst near midline)(4)Neurenteric cyst(5)Bronchial atresia(6)Bronchopulmonary sequestration (less frequently associated with polyhydramnios / hydrops)(7)Mediastinal / pericardial teratoma

Notes:

CYSTIC FIBROSIS
=MUCOVISCIDOSIS = FIBROCYSTIC DISEASE=autosomal recessive multisystem disease characterized by mucous plugging of exocrine glands secondary to(a)dysfunction of exocrine glands forming a thick tenacious material obstructing conducting system(b)reduced mucociliary transport*Incidence:*1:2,000-1:2,500 livebirths; almost exclusively in Caucasians (5% carry a CF mutant gene allele); unusual in Blacks (1:17,000), Orientals, Polynesians◊The most common inherited disease among Caucasian Americans!*Cause:*cystic fibrosis gene (= transmembrane conductance regulator gene) on long arm of chromosome 7 creates a defective transmembrane ion transport protein through deletion of an amino-acid; the normal product represents an epithelial chloride channel that supplies luminal water by osmosis; >230 different gene mutations (in 70% →F_{508})*Screening* (for 6 most common mutations of CF gene): carrier detection rate of 85% of Northern Europeans, 90% of Ashkenazi Jews, 50% of American Blacks *Age at diagnosis:*1st year of life (70%), by age 4 years (80%), by age 12 years (90%); mean age of 2.9 years; M:F = 1:1 ● elevated concentrations of sodium + chloride (>40 mmol/L for infants) in sweat ● decreased urinary PABA excretion ● infertility in males ● increased susceptibility to infection by Staphylococcus aureus + Pseudomonas aeruginosa*Prognosis:*median survival of 28 years; pulmonary complications are the most predominant cause of morbidity and death (90%) @Lung ● chronic cough ● recurrent pulmonary infections (reduced mucociliary clearance encourages Pseudomonas colonization) ● progressive respiratory insufficiency due to obstructive lung disease Location:predilection for apical + posterior segments of upper lobes√ "fingerlike" mucus plugging (mucoid impaction in dilated bronchi) within 1st month of life√ subsegmental / segmental / lobar atelectasis with right upper lobe predominance (10%)√ progressive cylindrical / cystic bronchiectasis (in 100% at >6 months of age) ± air-fluid levels due to prolonged mucus plugging preponderant in upper lobes√ parahilar linear densities + peribronchial cuffing√ focal peripheral / generalized hyperinflation secondary to collateral air drift into blocked airways)√ hilar adenopathy√ large pulmonary arteries (pulmonary arterial hypertension)√ recurrent local pneumonitis (initiated by staphylococcus / Haemophilus influenza, succeeded by Pseudomonas)√ allergic bronchopulmonary aspergillosis (with bronchial dilatation + mucoid impaction)CT: √ cylindrical (varicose / cystic) bronchiectasis√ peribrochial thickening√ bronchiectatic cyst (= bronchus directly leading into sacculation) in 56%√ interstitial cysts in 32%√ emphysematous bulla (= peripheral air space with long pleural attachment + without communication to bronchus) in 12%√ periseptal emphysema√ mucus plugs = tubular structures ± branching pattern√ subsegmental / segmental collapse / consolidationsNUC: √ matched patchy areas of decreased ventilation + perfusion *Cx:*(1)Pneumothorax (rupture of bulla / bleb), common + recurrent(2)Hemoptysis(3)Cor pulmonale(4)Hypertrophic pulmonary osteoarthropathy (rare) *Cause of death:*massive mucus plugging (95%)
@GI tract (85-90%) ● chronic obstipation ● failure to thrive√ gastroesophageal reflux (21-27%) due to transient inappropriate lower esophageal sphincter relaxation√ meconium plug syndrome (25%, most common cause of colonic obstruction in the infant)√ distal intestinal obstruction syndrome (10-15-47%)= meconium ileus equivalent syndrome (in older child / young adult)√ meconium ileus (10-16% at birth)◊Earliest clinical manifestation of cystic fibrosis!√ fibrosing colonopathy = stricture of right colon with longitudinal shortening secondary to high-dose lipase supplementation√ thickened nodular duodenal mucosal folds (due to unbuffered gastric acid, production of abnormal mucus, Brunner gland hypertrophy)√ mild generalized small bowel dilatation with diffuse distortion + thickening of mucosal folds (at times involving colon + rectum)√ large distended colon with mottled appearance (retained bulky dry stool)√ pneumatosis intestinalis of colon (5%) from air block phenomena of obstructive pulmonary disease√ "microcolon" = colon of normal length but diminished caliber√ "jejunization of colon" = coarse redundant + hyperplastic colonic mucosa (distended crypt goblet cells)√ Crohn disease√ appendicitis√ rectal prolapse between 6 months and 3 years in untreated patients (18-23%)*Cx:*gastrointestinal perforation with meconium peritonitis (50%), volvulus of dilated segments, bowel atresia, intussusception at an average age of 10 years (1%)
@Liver√ steatosis (30%) due to untreated malabsorption, dietary deficiencies, hepatic dysfunction, medications√ focal / multilobular biliary cirrhosis from inspissated bile ● signs of portal hypertension (clinically in 4-6%, autoptic in up to 50%)√ portal hypertension (in 1% of biliary cirrhosis) + hepatosplenomegaly + hypersplenism
@Biliary tree*Histo:*mucus-containing cysts in gallbladder wall ● cholestasis (secondary to CBD obstruction) ● symptoms of gallbladder disease (3.6%)√ sludge (33%)√ cholelithiasis (12-24%): mostly cholesterol stones due to (1) interrupted enterohepatic circulation after ileal resection / (2) ileal dysfunction in distal intestinal obstruction syndrome√ gallbladder atony√ microgallbladder (25% at autopsy)√ thickened trabeculated gallbladder wall√ subepithelial cysts of gallbladder wall√ atresia / stenosis of cystic duct @Pancreas*Histo:*dilatation of acini + cyst formation due to obstruction from protein plugs as a result of precipitation of relatively insoluble proteins*Path:*progressive ductectasia, pancreatic atrophy, increased pancreatic lobulation, fibrosis due to recurrent acute pancreatitis, replacement by fat ● steatorrhea + malabsorption + fat intolerance due to exocrine pancreatic insufficiency in 80-90% without affecting endocrine function (once 98% of entire pancreas is damaged)◊ Cystic fibrosis is the most common cause of exocrine pancreatic insufficiency in patients <30 years of age! ● abdominal pain, bloating, flatulence, failure to thrive ● diabetes mellitus (secondary to pancreatic fibrosis) in 1% of children + 13% of adults ● acute pancreatitis (clinically rare)√ diffuse pancreatic atrophy without fatty replacement√ lipomatous pseudohypertrophy of pancreas√ generalized increased echogenicity (70-100%)√ complete / partial fatty replacement (-90 to -120 HU)√ calcific chronic pancreatitis√ pancreatic cystosis = microscopic / 1-3 mm small cysts replacing pancreas (common), occasionally macroscopic cysts up to 12 cm @Skull √ sinusitis with opacification of well-developed maxillary, ethmoid, sphenoid sinuses√ hypoplastic frontal sinuses OB-US: √ hyperechogenic bowel (in up to 60-70% of fetuses affected with cystic fibrosis)*Prognosis:*median survival of 28 years; 2.3 deaths/100 patients from cardiorespiratory causes (78%), hepatic disease (4%)

Notes:

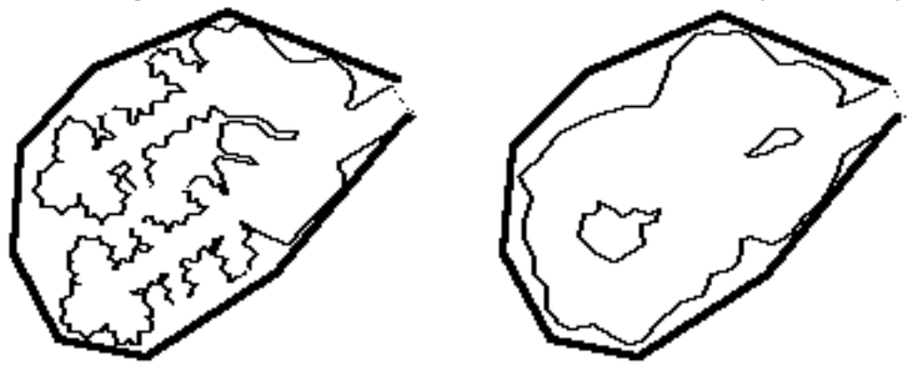
Congenital Diaphragmatic Hernia =absence of closure of the pleuroperitoneal fold by 9th week of gestational age*Embryology:* ventral component of diaphragm formed by septum transversum during 3rd-5th week GA; gradually extends posteriorly to envelop esophagus + great vessels; fuses with foregut mesentery to form the posteromedial portions of the diaphragm by 8th week GA; lateral margins of diaphragm develop from muscles of the thoracic wall; the posterolaterally located pleuroperitoneal foramina (Bochdalek) close last *Incidence:*1: 2,200-3,000 livebirths (0.04%);M:F = 2:1; most common intrathoracic fetal anomaly ◊Delayed onset following group B streptococcal infection!*Etiology:* (1)delayed fusion of diaphragm (spontaneous self-correction may occur) / premature return of bowel from its herniated position within the umbilical coelom(2)insult that inhibits / delays normal migration of the gut + closure of the diaphragm between 8-12th week of embryogenesis *Classification (Wiseman):* I.herniation early during bronchial branching leading to severe bilateral pulmonary hypoplasia; uniformly fatalII.herniation during distal bronchial branching leading to unilateral pulmonary hypoplasia; survival possibleIII.herniation late in pregnancy with compression of otherwise normal lung; excellent prognosisIV.postnatal herniation with compression of otherwise normal lung; excellent prognosis *Associated anomalies in 20% of liveborn and in 90% of stillborn fetuses:* 1.CNS (28%): neural tube defects2.Gastrointestinal (20%): particularly malrotation, oral cleft, omphalocele3.Cardiovascular (9-23%)4.Genitourinary (15%)5.Chromosomal abnormalities (4%): trisomy 18 + 216.Spinal defects7.IUGR (with concurrent major abnormality in 90%) Location:L:R = 5-9:1◊Right-sided hernias are frequently fatal!(1)**Bochdalek hernia** (85-90%)=posterolateral defect caused by maldevelopment / defective fusion of the cephalic fold of the pleuroperitoneal membranes*Incidence:*1:2,200-12,500 livebirthsLocation:left (80%), right (15%), bilateral (5%)Herniated organs: (a)on left: omental fat (6%), bowel, spleen, left lobe of liver, stomach (rare), kidney, pancreas(b)on right: part of liver, gallbladder, small bowel, kidney*mnemonic:*"4 Bs"**B**ochdalek **B**ack (posterior location) **B**abies (age at presentation) **B**ig (usually large) (2)**Morgagni hernia** (1-2%)=anteromedial parasternal defect (space of Larrey) caused by maldevelopment of septum transversum; R > L*Incidence:*1:100,000Herniated organs:omental fat, transverse colon, liver*Often associated with:* chromosomal abnormality, mental retardation, heart defects, pericardial deficiency (a)abdominal viscera / fat may herniate into pericardial sac(b)heart may herniate into upper abdomen*mnemonic:*"4 Ms"**M**orgagni **M**iddle (anterior + central location) **M**ature (present in older children) **M**inuscule (usually small) (3)Septum transversum defect = defect in central tendon(4)Hiatal hernia = congenitally large esophageal orifice(5)**Eventration** (5%) = upward displacement of abdominal contents secondary to a congenitally thin hypoplastic diaphragm*Unilateral eventration may be associated with:* Beckwith-Wiedemann syndrome, trisomy 13, trisomy 15, trisomy 18 *Bilateral eventration may be associated with:* toxoplasmosis, CMV, arthrogryposis Location:anteromedial on right, total involvement on left side; R:L = 5:1√ small diaphragmatic excursions√ often lobulated diaphragmatic contour ● respiratory distress in neonatal period (life-threatening deficiency of small airways + alveoli) ● scaphoid abdomen*Herniated organs:* small bowel (90%), stomach (60%), large bowel (56%), spleen (54%), pancreas (24%), kidney (12%), adrenal gland, liver, gallbladder√ bowel loops in chest√ contralateral shift of mediastinum + heart√ complete (1-2%) / partial absence of diaphragm√ absence of stomach, small bowel in abdomen√ passage of nasogastric tube under fluoroscopic control entering intrathoracic stomach√ incomplete rotation + anomalous mesenteric attachment of bowel OB-US (diagnosis possible by 18 weeks GA): √ solid / multicystic / complex chest mass√ mediastinal shift√ nonvisualization of fetal stomach below diaphragm√ fetal stomach at level of fetal heart√ peristalsis of bowel within fetal chest (inconsistent)√ paradoxical motion of diaphragm with fetal breathing (defect in diaphragm sonographically not visible)√ scaphoid fetal abdomen with reduced abdominal circumference√ herniated liver frequently surrounded by ascites√ polyhydramnios (common, due to partial esophageal obstruction or heart failure) / normal fluid volume / oligohydramnios√ swallowed fetal intestinal contrast appears in chest (CT amniography confirms diagnosis)*Cx:*(1)Bilateral pulmonary hypoplasia(2)Persistent fetal circulation (postsurgical pulmonary hypertension)*Prognosis:*(1)Stillbirth (35-50%)(2)Neonatal death (35%)◊Survival is determined by size of defect + time of entry + associated anomalies (34% survival rate if isolated, 7% with associated anomalies)Indicators for poor prognosis: large intrathoracic mass with marked mediastinal shift, IUGR, polyhydramnios, hydrops fetalis, detection <25 weeks MA, intrathoracic liver, dilated intrathoracic stomach, other malformations *Mortality:*in 10% death before surgery;40-50% operative mortality;(a)stomach intrathoracic vs. intraabdominal = 60% vs. 6%(b)polyhydramnios vs. normal amniotic fluid = 89% vs. 45%*DDx:*Congenital adenomatoid malformation, mediastinal cyst (bronchogenic, neuroenteric, thymic)

Notes:

Traumatic Diaphragmatic Hernia *Prevalence:*0.8-5.0% of all trauma patients; 5% of all diaphragmatic hernias, but 90% of all strangulated diaphragmatic herniasEtiology of traumatic rupture of diaphragm: (a)blunt trauma (5-50%) due to marked increase in intraabdominal pressure: motor vehicle accident, fall from height, bout of hyperemesis; L:R = 3:1, bilateral rupture in <3.6%(b)penetrating trauma (50%): knife, bullet, repair of hiatus hernia◊usually <1 cm in diameter; detected at surgery*Herniated organs in order of frequency:* stomach, colon, small bowel, omentum, spleen, kidney, pancreas ● may be asymptomatic for months / years following trauma, onset of symptoms may be so long delayed that traumatic event is forgotten ● virtually all become ultimately symptomatic, most in <3 years ● **Bergqvist triad**:(1) rib fractures (2) fracture of spine / pelvis (3) traumatic rupture of diaphragm Location:90-98% on left side; posterolateral portion of diaphragm medial to spleenSize:most tears are >10 cm in lengthCXR: ◊The first posttraumatic CXR is abnormal in only 28-64%!√ nonvisualization of diaphragmatic contour√ abnormally elevated contour of hemidiaphragmCave:cephalad margin of bowel may simulate an elevated diaphragm (look for haustra)√ lower lobe mass / consolidation (herniated solid organ / omentum / airless bowel loop)√ inhomogeneous mass with air-fluid level in left hemithorax√ displacement of mediastinum + lung to contralateral side√ mushroomlike mass of herniated liver in right hemithorax√ "hourglass" constriction of afferent + efferent bowel loops at orifice√ hydrothorax / hemothorax indicates strangulation√ nasogastric tube above suspected level of hemidiaphragmN.B.:tube first dips below diaphragm (rent spares esophageal hiatus with gastroesophageal junction remaining in its normal position)√ location of diaphragm may be documented by1. gas-filled bowel constricted at site of diaphragmatic laceration2. barium studyCT (61% sensitive, 87% specific): √ abrupt discontinuity of hemidiaphragm√ herniation of omentum / abdominal viscera into thorax√ "collar sign" = focal constriction of viscera at level of diaphragm√ "absent diaphragm sign" = failure to see diaphragm*Associated injuries:* √ fractures of lower ribs√ perforation of hollow viscus√ rupture of spleen*Reasons fore diagnostic misses:* (1)left-sided defect covered by omentum(2)right-sided defect sealed by liver(3)positive pressure ventilation*Cx:*life-threatening strangulation of bowel / stomach occurs in majority◊90% of strangulated hernias are traumatic!*DDx:*eventration, diaphragmatic paralysis

Notes:

EMPHYSEMA
=group of pulmonary diseases characterized by permanently enlarged air spaces distal to terminal bronchioles accompanied by destruction of alveolar walls + local elastic fiber network The clinical term "chronic obstructive pulmonary disease (COPD)" should not be used in image interpretation! It encompasses: asthma, chronic bronchitis, emphysema!*Prevalence:*1.65 million people in United States*Cause:*imbalance in elastase-antielastase system (due to increase in elastase activity in smokers / a_1-antiprotease deficiency) causing proteolytic destruction of elastin resulting in alveolar wall destruction ● dyspnea on exertion ● irreversible expiratory airflow obstruction (due to decreased elastic recoil from parenchymal destruction) ● decreased carbon monoxide diffusing capacityCXR (moderately sensitive, highly specific): √ hyperinflated lung (most reliable sign)√ low hemidiaphragm (= at / below 7th anterior rib)√ flat hemidiaphragm (= <1.5 cm distance between line connecting the costo- and cardiophrenic angles + top of midhemidiaphragm) √ retrosternal air space >2.5 cm√ "barrel chest" = enlarged anteroposterior chest diameter√ saber-sheath trachea√ pulmonary vascular pruning + distortion (± pulmonary arterial hypertension)√ right-heart enlargement√ bullaeHRCT: √ well-defined areas of abnormally decreased attenuation without definable wall (<-910 HU)*Rx:*lung volume reduction surgery



Centrilobar Emphysema Panacinar Emphysema

Centrilobular Emphysema Panacinar Emphysema Paracicatricial Emphysema Paraseptal Emphysema

Notes:

Centrilobular Emphysema =CENTRIACINAR EMPHYSEMA = PROXIMAL ACINAR EMPHYSEMA=emphysematous change selectively affecting the acinus at the level of 1st + 2nd generations of respiratory bronchioles (most common form)*Path:*normal + emphysematous alveolar spaces adjacent to each other*Histo:*enlargement of respiratory bronchioles + destruction of centrilobular alveolar septa in the center of the secondary pulmonary lobule; CHARACTERISTICALLY surrounded by normal lung; distal alveoli spared; severity of destruction varies from lobule to lobule*Predisposed:*smokers (in up to 50%), coal workers*Cause:*excess protease with smoking (elastase is contained in neutrophils + macrophages found in abundance in lung of smokers) ● blue bloaterSite:apical and posterior segments of upper lobe + superior segment of lower lobe (relatively greater ventilation-perfusion ratio in upper lobes favors deposition of particulate matter and release of elastase in upper lungs)CXR (80% sensitivity for moderate / severe stages): √ irregular scattered area of radiolucency (best appreciated if lung opacified by edema / pneumonia / hemorrhage) = area of bullae, arterial depletion + increased markings√ hyperinflated lungHRCT: √ "emphysematous spaces" (= focal area of air attenuation) >1 cm in diameter with central dot / line (representing the centrilobular artery of secondary pulmonary lobule) without definable wall and surrounded by normal lung√ pulmonary vascular distortion + pruning with lack of juxtaposition of normal lung (advanced stage)

Notes:

Panacinar Emphysema =PANLOBULAR EMPHYSEMA = DIFFUSE EMPHYSEMA = GENERALIZED EMPHYSEMA (rare)=emphysematous change involving the entire acinus=uniform nonselective destruction of all air spaces throughout both lungs*Path:*uniform enlargement of acini from respiratory bronchioles to terminal alveoli (from center to periphery of secondary pulmonary lobule) secondary to destruction of lung distal to terminal bronchiole*Cause:* autosomal recessive a-1-antitrypsin deficiency in 10-15% (proteolytic enzymes carried by leukocytes in blood gradually destroy lung unless inactivated by a-1-protease inhibitor)*Age:*6th-7th decade (3rd-4th decade in smokers) ● pink pufferSite:affects whole lung, but more severe at lung bases (due to greater blood flow)CXR: √ hyperinflated lung√ decreased pulmonary vascular markings√ lung destruction extremely uniformHRCT: √ diffuse simplification of lung architecture with pulmonary septal and vascular distortion + pruning (difficult to detect early, ie, prior to considerable lung destruction for lack of adjacent normal lung)√ paucity of vessels√ bullae

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Notes:

Paracicatricial Emphysema =PERIFOCAL / IRREGULAR EMPHYSEMA=airspace enlargement + lung destruction developing adjacent to areas of pulmonary scarring*Usual cause:*granulomatous inflammation, organized pneumonia, pulmonary infarction*Path:*no consistent relationship to any portion of secondary lobule / acinus; frequently associated with bronchiolectasis producing "honeycomb lung" ● little functional significanceCXR (rarely detectable): √ fine curvilinear reticular opacities + interposed radiolucent areasHRCT: √ low-attenuation areas adjacent to areas of fibrosis (diagnosable only in the absence of other forms of emphysema)

Notes:

Paraseptal Emphysema =DISTAL ACINAR EMPHYSEMA = LOCALIZED EMPHYSEMA = LINEAR EMPHYSEMA=focal enlargement + destruction of air spaces in one site in otherwise normal lung*Path:*predominant involvement of alveolar ducts + sacsSite:characteristically within subpleural lung and adjacent to interlobular septa + vesselsCXR: √ area of lucency, frequently sharply demarcated from normal lung√ bands of radiopacity (residual vessels / interstitium) may be presentHRCT: √ peripheral low-attenuation area with remainder of lung normal*Cx:*spontaneous pneumothorax; bullae formation

Notes:

EMPYEMA

Stage I"exudative" stage = inflamed pleura weeps proteinaceous fluid into pleural space = sterile exudate ● elevated number of PMNs ● pH >7.20; glucose >40 mg/dL (2.2 mmol/L);LDH <1000 II"fibropurulent" stage = accumulation of neutrophils + fibrin deposition on pleural surfaces-early stage II empyema ● WBCs >5 x 10^9/mm^3, but no gross pus ● pH between 7.0 and 7.2 ● glucose level >40 mg/dL-late stage II empyema ● frank pus ● pH <7.0 ● glucose level <40 mg/dL*Cx:*multiloculation*Rx:*chest tube drainage III"organization" stage = fibroblast infiltration forming "pleural peel / pleural rind"*Cx:*limited expansion of lung*Rx:*decortication (with persistent sepsis despite appropriate antibiotic Rx + drainage / persistent thick pleural rind trapping underlying lung)CT: √ thickening of parietal pleura in 60% on NECT, in 86% on CECT√ increased thickness + density of paraspinal subcostal tissue (inflammation of extrapleural fat)√ curvilinear enhancement of chest wall boundary in 96% (inflammatory hyperemia of pleura)√ "split pleura" sign = pleural fluid between enhancing thickened parietal + visceral pleura√ gas bubbles in pleural space (gasforming organism / bronchopleural fistula)*DDx:*simple / complicated parapneumonic effusion (negative Gram + culture stain), malignant effusion after sclerotherapy, malignant invasion of chest wall, mesothelioma, pleural tuberculosis, reactive mesothelial hyperplasia, pleural effusion of rheumatoid disease

Notes:

EXTRAMEDULLARY PLASMACYTOMA
Uncommon form; relatively benign course (dissemination may be found months / ears later or not at all); questionable if precursor to multiple myeloma *Age :*35-40 years; M:F = 2:Location:air passages (50%) predominantly in upper nose and oral cavity; conjunctiva (37%); lymph nodes (3%) ● usually not associated with increased immunoglobulin titer or amyloid deposition ● mass of one to several cm in size with well-defined lobulated border*Classification:* 1.Medullary plasmacytoma2.Multiple myeloma:(a)scattered involvement of bone(b)myelomatosis of bone3.Extramedullary plasmacytoma*DDx:* (1)MULTIPLE MYELOMA=malignant course with soft-tissue involvement in 50-73%:(a)microscopic infiltration(b)enlargement of organs(c)formation of tumor mass (1/3) ● usually associated with protein abnormalities ● may have amyloid deposition*Age incidence:*50-85 years ◊ tends to occur late in the course of the disease and indicates a poor prognosis (0-6% 5-year survival)

Notes:

EXTRINSIC ALLERGIC ALVEOLITIS
= HYPERSENSITIVITY PNEUMONITIS =characterized by an inappropriate host response to inhaled organic allergens that are often related to patients occupation*Cause:*exposure to organic dust of <5 µm particle size acting as antigen*Histo:*diffuse predominantly mononuclear cell inflammation of bronchioles (bronchiolitis) + pulmonary parenchyma (alveolitis); ill-defined granulomas of <1 mm in diameter ● asymptomatic (10-40%) ● recurrent episodes of fever, chills, dry cough, dyspnea following exposure after 6-hour interval ● resolution of episodic symptoms after cessation of exposure, abate spontaneously over 1-2 days ● insidious onset of gradually progressive dyspnea ● reduction in vital capacity, diffusing capacity, arterial PO_2 ● intracutaneous injection of antigen results in delayed hypersensitivity reaction ● presence of serum precipitins against antigen ● positive aerosol provocation inhalation test ● markedly increased cell count with often >50% T-lymphocytes on bronchoalveolar lavageLocation:predominantly midlung zones, occasionally lower lung zones, rarely upper lung zones*Specific antigens* for immune complex disease (Type III = Arthus reaction): 1.**Farmers lung** from moldy hay (Thermoactinomyces vulgaris or Micropolyspora faeni)2.Hypersensitivity pneumonitis from forced-air equipment = **Pandoras pneumoniti**s with heating / humidifying / air conditioning systems (thermophilic actinomycetes)3.**Bird-fanciers lung**, pigeon breeders lung from protein in bird serum / excrements / feathers4.**Mushroom workers lung** from mushroom compost (Thermoactinomyces vulgaris or Micropolyspora faeni)5.**Bagassosis** from moldy sugar cane in sugar mill (contamination with Thermoactinomyces sacchari /vulgaris and Micropolyspora faeni)6.**Malt workers lung** from malt dust (Aspergillus clavatus)7.**Maple bark disease** from moldy maple bark in saw mill (Cryptostroma corticale)8.**Suberosis** from moldy cork dust (Penicillium frequentans)9.**Sequoiosis** from redwood dust (Graphium species)*Thermophilic actinomycetes* = bacteria <1 µm in diameter with morphologic characteristics of fungi; found in soil, grains, compost, fresh water, forced-air heating, cooling system, humidifier, air-conditioning system A.ACUTE EXTRINSIC ALLERGIC ALVEOLITIS=heavy exposure to inciting antigen in domestic, occupational, atmospheric environment*Histo:*filling of air spaces by polymorph neutrophils + lymphocytes*Onset of symptoms after exposure:* 4-8 hours ● fever, chills, malaise, chest tightness, cough, dyspnea ● scanty mucoid expectoration ● frontal headache, arthralgia (common)√ No CXR abnormalities in 30-95%√ diffuse acinar consolidative pattern (edema + exudate filling alveoli) resolving within a few days√ lymph node enlargement (unusual, more common with recurrence)CT: √ small + medium rounded opacities (large active granulomas)√ diffuse dense airspace consolidation (confluent collections of intraalveolar histiocytes, interstitial + intraalveolar edema)*Dx:*classical presentation of a known exposure history + typical symptoms + detection of serum precipitins to suspected antigen B.SUBACUTE EXTRINSIC ALLERGIC ALVEOLITIS=less intense but continuous exposure to inhaled antigens, usually in domestic environment*Histo:*predominantly interstitial lymphocytic infiltrate, poorly defined granulomas, cellular bronchiolitis*Onset of symptoms after exposure:* weeks - months ● recurrent respiratory / systemic symptoms: breathlessness upon exertion, fever + cough, weight loss, muscle + joint pain√ changes may be completely reversible if present less than 1 year√ interstitial nodular / reticulonodular patternCT: √ poorly defined centrilobular micronodules <5 mm (cellular bronchiolitis + small granulomas)√ widespread patchy ground-glass attenuation in 52% (obstructive pneumonitis, filling of alveoli by large mononuclear cell infiltrates)√ areas of decreased attenuation + mosaic perfusion (86%) C.CHRONIC EXTRINSIC ALLERGIC ALVEOLITIS=prolonged insidious dust exposure*Onset of symptoms after exposure:* months - years ● insidious progressive exertional dyspnea indistinguishable from idiopathic pulmonary fibrosis*Histo:*proliferation of epithelial cells + predominantly peribronchiolar interstitial fibrosisLocation:usually in mid zones, relative sparing of lung apices + costophrenic sulci√ irregular linear opacities (fibrosis)√ loss of lung volume (cicatrization atelectasis)√ pleural effusion (rare)√ lymph node enlargement may occurCT: √ honeycombing without zonal predominance√ focal air trapping / diffuse emphysema√ coexistent subacute changes (due to continuing exposure)*Rx:*mask, filter, industrial hygiene, alterations in forced-air ventilatory system, change in patients habits / occupation / environment

Notes:

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FAT EMBOLISM
=obstruction of pulmonary vessels by fat globules followed by chemical pneumonitis from unsaturated plasma fatty acids producing hemorrhage / edema*Incidence:*in necropsy series in 67-97% of patients with major skeletal trauma, however, symptomatic fat embolism syndrome in <10% (M > F)*Onset:*24-72 hours after trauma ● dyspnea (progressive pulmonary insufficiency) ● fever ● systemic hypoxemia ● mentation changes: headaches, confusion ● petechiae (50%) from coagulopathy (release of tissue thromboplastin)√ initial chest film usually negative (normal up to 72 hours)√ platelike atelectasis√ bilateral diffuse alveolar infiltrates√ consolidation (may progress to ARDS)NUC: √ mottled peripheral perfusion defects (1-4 days after injury), later enlarging secondary to pneumonic infiltrates

Notes:

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FOCAL ORGANIZING PNEUMONIA
=unresolving pneumonia / pneumonia with incomplete resolution beyond 8 weeks*Prevalence:*5-10% of all pneumonias (87% of pneumonias resolve within 4 weeks, 12% within 4-8 weeks)*Predisposing factors:*? age, diabetes mellitus, chronic bronchitis, overuse of antibiotics*Histo:*organization of intraalveolar exudate + thickening of alveolar septa / chronic inflammatory change of bronchial mucosa + obstructive lesion in bronchioles with organization ● cough, sputum, fever, hemoptysis (in 1/4)√ ill-defined localized parenchymal abnormality with irregular margin√ decrease in size of mass within 3-4 weeksHRCT: √ flat / ovoid lesion with irregular margin in subpleural location / along bronchovascular bundle√ ± satellite lesions (44%) + air bronchogram (22%)

Notes:

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FRACTURE OF TRACHEA / BRONCHUS

Location:(a)mainstem bronchus 1-2 cm distal to carina (80%); R > L(b)just above carina (20%)√ fracture of first 3 ribs (53-91%), rare in children√ pneumothorax (70%) √ mediastinal ± subcutaneous emphysema√ absence of pleural effusion√ collapsed lung falling to dependent position (loss of anchoring support in bronchial transection)√ atelectasis (may be late development)√ inadequate reexpansion of lung despite chest tube (due to large air leak)*Prognosis:*30% mortality (in 15% within 1 hour)

Notes:

GOODPASTURE SYNDROME
=autoimmune disease characterized by(1) glomerulonephritis (2) circulating antibodies against glomerular + alveolar basement membrane (3) pulmonary hemorrhage *Pathogenesis:* cytotoxic antibody-mediated disease = Type II hypersensitivity; alveolar basement membrane becomes antigenic (perhaps viral etiology); IgG / IgM antibody with complement activation causes cell destruction + pulmonary hemorrhage, leads to hemosiderin deposition and pulmonary fibrosis *Age peak:*26 years (range 17-78 years); M:F = 7:1 ● iron-deficiency anemia ● hepatosplenomegaly ● systemic hypertension@ Lung ● preceding upper respiratory infection (in 2/3) + renal disease ● mild hemoptysis (72%) with hemosiderin-laden macrophages in sputum, commonly precedes the clinical manifestations of renal disease by several months ● cough, dyspnea, basilar rales√ patchy alveolar filling pattern with predominance in perihilar area + lung bases√ air bronchogram√ consolidation at lung bases + central lung fields√ gradually interstitial pattern (due to septal thickening) = organization of hemorrhage√ hilar lymph noes may be enlarged during acute episodes@ Kidney ● glomerulonephritis with IgG deposits in characteristic linear pattern in glomeruli ● hematuria*Prognosis:*death within 3 years (average 6 months) because of renal failure*Rx:*cytotoxic chemotherapy, plasmapheresis, bilateral nephrectomy*DDx:*idiopathic pulmonary hemosiderosis

Notes:

GRANULOMA OF LUNG
Cause: A.SarcoidosisB.Non-sarcoid granulomatous disease(a)infectious-bacterial: TB, gumma-opportunistic: cryptococcosis-parasitic: Dirofilaria immitis (dog heartworm)-fungal: histoplasmosis, coccidioidomycosis, nocardiosis(b)noninfectious-foreign body: talc, beryllium, algae, pollen, cellulose, lipids, abuse of nasally inhaled drugs, aspiration of medication-angiocentric lymphoproliferative disease-vasculitides-extrinsic allergic alveolitis-Langerhans cell histiocytosis-pulmonary hyalinizing granuloma-peribronchial granuloma-chronic granulomatous disease of childhood*Histo:*epithelial cells, lymphocytes, macrophages, giant cells of Langhans type*Frequency:*constitutes the majority of solitary pulmonary nodules ● nonproductive cough ● shortness of breath ● spontaneous pneumothoraxCXR: ◊CXR detection requires multiple granulomas / clusters of granulomas (individual granuloma too small)!√ central nidus of calcification in a laminated / diffuse pattern√ absence of growth for at least 2 yearsCT (most effective in nodules ≤3 cm of diameter with smooth discrete margins): √ 50-60% of pulmonary nodules demonstrate unsuspected calcification by CT*DDx:*Carcinoma (in 10% eccentric calcification in preexisting scar / nearby granuloma / true intrinsic stippled calcification in larger lesion)

Notes:

HAMARTOMA OF CHEST WALL
=MESENCHYMOMA (incorrect as it implies neoplasm)=focal overgrowth of normal skeletal elements with a benign self-limited course; extremely rare*Age:*1st year of life√ moderate / large extrapleural well-circumscribed mass affecting one / more ribs√ ribs near center of mass partially / completely destroyed√ ribs at periphery deformed / eroded√ significant amount of calcification / ossification (DDx: aneurysmal bone cyst)√ mass compresses underlying lung*Rx:*resection curative

Notes:

HAMARTOMA OF LUNG
=composed of tissues normally found in this location in abnormal quantity, mixture, and arrangement*Incidence:*0.25% in population (autopsy); 6-8% of all solitary pulmonary lesions; most common benign lung tumor*Etiology:* 1.Congenital malformation of a displaced bronchial anlage2.Hyperplasia of normal structures3.Cartilaginous neoplasm4.Response to inflammation*Path:*columnar, cuboidal, ciliated epithelium, fat (in 50%), bone, cartilage (predominates), muscle, vessels, fibrous tissue, calcifications, plasma cells originating in fibrous connective tissue beneath mucous membrane of bronchial wall*Age peak:*5th + 6th decade; M:F = 3:1 ● mostly asymptomatic ● hemoptysis (rare) ● cough, vague chest pain, fever (with postobstructive pneumonitis)Location:2/3 peripheral; endobronchial in 10%; multiplicity (rare)√ round smooth lobulated mass <4 cm (averages 2.5 cm)√ calcification in 15% (almost pathognomonic if of chondroid "popcorn" type)√ fat in 50% (detection by CT)√ cavitation (extremely rare)√ growth patterns: slow / rapid / stable with later growth√ usually 5 mm increase in diameter per yearHRCT: √ fat density detectable in 34% (-80 to -120 HU)√ calcium + fat detectable in 19%*DDx:*Lipoid pneumonia (ill-defined mass / lung infiltrate)

Notes:

HEREDITARY HEMORRHAGIC TELANGIECTASIA
=RENDU-OSLER-WEBER SYNDROME=hereditary multiorgan abnormality of vascular structure*Etiology:*gene encoding a protein that binds transforming growth factor*Path:*direct connections between arteries + veins with absence of capillaries (telangiectases are small AVMs)(a)small telangiectasis = focal dilatation of postcapillary venules with prominent stress fibers in pericytes along luminal borders(b)fully developed telangiectasis = markedly dilated + convoluted venules with excessive layers of smooth muscle without elastic fibers directly connecting to dilated arterioles@Nose (telangiectasis of nasal mucosa) ● recurrent epistaxis: more severe over time in 66%; begins by age 10, present by age 21 in most cases@Skin (present in most cases by age 40)telangiectases of lips, tongue, palate, fingers, face, conjunctiva, trunk, arms, nail beds @Lung (in 5-15%)*see* PULMONARY ARTERIAL MALFORMATION @CNS (cerebral or spinal AVMs) ● subarachnoid hemorrhage ● seizure; paraparesis (less common)@GI tract (stomach, duodenum, small bowel, colon)occasionally associated with AVMs / angiodysplasia ● recurrent GI bleeding (in 5th-6th decade)@Liverpresence of multiple AVMs / atypical cirrhosis ● high cardiac output failure (due to L-to-R shunt)

Notes:

HISTOPLASMOSIS
*Prevalence:*nearly 100% in endemic area; up to 30% in Central + South America, Puerto Rico, West Africa, Southeast Asia*Organism:*Histoplasma capsulatum = dimorphic fungus; worldwide most often in temperate climates; widespread in soil enriched by bird droppings of central North America (endemic in Ohio, Mississippi, St. Lawrence River valley; exists as a spore in soil + transforms into yeast form at normal body temperatures*Infection:*inhalation of wind-borne spores (microconidia of 2-6 µm, macroconidia of 6-14 µm) which germinate within alveoli releasing yeast forms which are phagocytized but not killed by macrophages; invasion of pulmonary lymphatics with spread to hilar + mediastinal lymph nodes; hematogenous dissemination of parasitized macrophages throughout reticuloendothelial system (spleen!)*Path:*spores incite formation of epitheloid granulomas, necrosis, calcification *Dx:*(1)Culture (sputum, lung tissue, urine, bone marrow, lymph node)(2)Identification of yeast forms stained with PAS / Gomori methenamine silver(3)Complement fixation test (absolute titer of 1:64 or 4-fold rise in convalescent titer suggest active / recent infection)(4)Serum immunodiffusion: agar gel diffusion test (H precipitin band)*Rx:*ketoconazole

Pulmonary Histoplasmosis

Notes:

Pulmonary Histoplasmosis A.ACUTE HISTOPLASMOSIS ● mostly asymptomatic and self-limiting illness (in 99.5%) ● fever, cough, malaise simulating viral upper respiratory infection 3 weeks after massive inoculum / in debilitated patients (infants, elderly) ● positive skin test for histoplasmosis√ generalized lymphadenopathy√ bilateral nonsegmental bronchopneumonic pattern with tendency to clear in one area + appear in another√ multiple nodules changing into hundreds of punctate calcifications (usually >4 mm) after 9-24 months√ "target lesion" = central calcification is PATHOGNOMONIC√ hilar / mediastinal lymph node enlargement (DDx: acute viral / bacterial pneumonia)√ "popcorn" calcification of mediastinal lymph nodes >10 mm√ >5 splenic calcifications (40%)CT: √ paratracheal / subcarinal mass with regions of low attenuation (necrosis) + enhancing septa B.CHRONIC HISTOPLASMOSIS (0.03%)*Predisposed:*individuals with chronic obstructive pulmonary disease*Age:*adult middle-aged white men*Pathophysiology:*hyperimmune reaction ● cough, low-grade fever, night sweats simulating postprimary tuberculosis√ segmental wedge-shaped peripheral consolidation of moth-eaten appearance from scattered foci of emphysematous lung√ fibrosis in apical posterior segments of upper lobes (indistinguishable from postprimary TB) adjacent to emphysematous blebs C.DISSEMINATED HISTOPLASMOSIS*Predisposed:*impaired T-cell immunity; AIDS*Prevalence:*1:50,000 exposed individuals*Pathophysiology:*progression of exogenous infection / reactivation of latent focus ● acute rapidly fatal infection ● fever, weight loss, anorexia, malaise ● cough (<50%) ● abdominal pain, nausea, vomiting, diarrhea ● chronic intermittent illness ● low-grade fever, weight loss, fatigue ● adrenal insufficiency√ normal CXR (>50%)√ miliary / diffuse reticulonodular pattern rapidly progressing to diffuse airspace opacification√ hilar + mediastinal adenopathy√ hepatosplenomegaly*Cx:*arthritis (most often knee), tenosynovitis, osteomyelitis D.DELAYED MANIFESTATIONS√ **histoplasmoma** (= continued growth of primary focus at 0.5-2.8 mm/year) adjacent to pleura + typically with laminated calcific rings;*in 20% associated with:* mediastinal granulomas√ broncholithiasis√ **mediastinal granuloma** (more common)=direct infection of mediastinal lymph nodes*Histo:*involved nodes with varying degrees of central caseation ± calcification ● usually asymptomaticLocation:subcarinal / right paratracheal / hilar lymph nodes√ widened mediastinum (enlarged nodes + veins)√ lobulated mass of low-density lymph nodes 3-10 cm in thickness surrounded by a 2-5 mm thick fibrous capsule crisscrossed by irregularly shaped septa (CHARACTERISTIC)√ displacement of SVC / esophagus√ fibrosing mediastinitis (less common)◊Organism recovered in only 50%!

Notes:

HYDATID DISEASE
=ECHINOCOCCOSIS ● asymptomatic ● eosinophilia (<25%) ● cough, expectoration, fever ● positive Casoni skin test in 60% ● hypersensitivity reaction (if cyst rupture occurs)√ solitary (75%) / multiple (25%) sharply circumscribed spherical / ovoid masses√ size of 1-10 cm in diameter (16-20 weeks doubling time)√ cyst communicating with bronchial tree√ "meniscus sign", "double arch sign," "moon sign," "crescent sign" (5%) = rupture of pericyst with air dissection between peri- and exocyst√ "water lily sign," "sign of the Camalotte" = collapsed cyst membrane floating on the fluid√ air-fluid level = rupture of all cyst walls√ hydropneumothorax√ calcification of cyst wall (<6%)√ rib + vertebral erosion (rare)√ mediastinal cyst: posterior (65%), anterior (26%), middle (9%) mediastinum

Notes:

HYPOGENETIC LUNG SYNDROME
=collective name for congenital underdevelopment of one / more lobes of a lung separated into 3 forms:1.**Pulmonary agenesis**
=complete absence of a lobe + its bronchusCT: √ missing bronchus + lobe(s)2.**Pulmonary aplasia**
=rudimentary bronchus ending in blind pouch + absence of parenchyma + vessels*Incidence:*1:10,000; R:L = 1:1CT: √ absence of ipsilateral pulmonary artery√ bronchus terminates in dilated blind pouch√ absence of ipsilateral pulmonary tissue3.**Pulmonary hypoplasia** (38%)=completely formed but congenitally small bronchus with rudimentary parenchyma + small vessels*Developmental causes:* (a)Idiopathic(b)Extrathoracic compression1.Oligohydramnios2.Fetal ascites3.Membranous diaphragm(c)Thoracic cage compression1.Thoracic dystrophies2.Muscular disease(d)Intrathoracic compression1.Diaphragmatic defect2.Excess pleural fluid3.Large intrathoracic cyst / tumorCT: √ small bronchus + lobe ◊Hypogenetic lung is the most constant component of congenital pulmonary venolobar syndrome!*May be associated with:*congenital tracheal stenosis, bronchitis, bronchiectasisLocation:R:L = 3:1; RML (65%) > RUL (40%) > RLL (20%) > LUL (20%) > LLL (15%); multiple lobes (45%) ● usually asymptomatic (in isolated hypogenetic lung) ● exertional dyspnea√ small ipsilateral hemithorax + elevated hemidiaphragm√ diminished pulmonary vascularity on involved side√ small hilum on involved side (absent / small pulmonary artery)√ mediastinum + heart shifted toward involved side√ indistinct cardiomediastinal border on involved side√ diminished radiolucency on involved side√ large ipsilateral apical cap + blunted costophrenic angle√ broad retrosternal band of opacity (LAT view) **Horseshoe Lung** =uncommon variant of hypogenetic lung syndrome in which RLL crosses midline between esophagus and heart + fuses with opposite lung√ oblique fissure in left lower hemithorax (if both lungs separated by pleural layers)√ pulmonary vessels + bronchi crossing midline

Notes:

Acute Interstitial Pneumonia = AIP = [ACCELERATED INTERSTITIAL PNEUMONIA] = DIFFUSE ALVEOLAR DAMAGE = IDIOPATHIC ARDS = ACUTE DIFFUSE INTERSTITIAL FIBROSIS = HAMMAN-RICH SYNDROME =rapidly progressive fulminant disease of unknown etiology that usually occurs in previously healthy subjects + produces diffuse alveolar damage*Path:*temporally homogeneous organizing diffuse alveolar damage; little mature collagen deposition / architectural distortion / honeycombing (as opposed to UIP)*Histo:*thickening of alveolar wall due to alveolar edema + inflammatory cells; extensive alveolar damage with hyaline membrane formation; marked interstitial fibroblast proliferation with stabilizing nonprogressive scarring*Mean age:*50 years; M=F ● prodromal viral upper respiratory infection: cough, fever ● rapidly increasing dyspnea + acute respiratory failure ● requires ventilation within days to (1-4) weeksLocation:mainly lower lung zonesSite:predominantly central / subpleural (in 22%)CXR: √ progressive extensive bilateral airspace opacification: symmetric, bilateral, basilarCT: √ diffuse extensive bilateral airspace consolidation (in 67%) with basal predominance (similar to ARDS)√ patchy (67%) / diffuse (38%) bilateral ground-glass opacities√ anteroposterior lung attenuation gradient*Dx:*negative bacterial / viral / fungal cultures; no inhalational exposure to noxious agents; no pulmonary drug toxicity*Prognosis:*death within 1-6 months (60-90%); recovery in 12%

Notes:

Subacute Interstitial Pneumonia *BOOP* see BRONCHIOLITIS OBLITERANS ***Nonspecific Interstitial Pneumonia With Fibrosis*** =NONCLASSIFIABLE INTERSTITIAL PNEUMONIA=interstitial pneumonia that cannot be classified as UIP / DIP / acute interstitial pneumonia / BOOP*Histo:*temporal uniformity of (a)cellular interstitial infiltrate with little / no fibrosis (48%)(b)inflammation + fibrosis (38%)(c)dense fibrosis dominant (14%);occasionally intraalveolar accumulation of macrophages + focal areas of bronchiolitis obliterans organizing pneumonia *Cause:*collagen vascular disease (16%), inhalational exposure to noxious agents (17%), recent surgery / severe pneumonia / ARDS (8%)*Mean age:*46 years; M<F ● dyspnea + dry cough (1-week to 5-year history)Location:no zonal predominance√ normal CXR in 14%√ bibasilar irregular linear opacities + airspace consolidation√ normal / slightly decreased lung volumeCT: √ bilateral areas of scattered ground-glass opacities (100%)√ bibasilar airspace consolidation (71%)√ irregular linear opacities (29%)√ bronchial dilatation in areas of consolidation (71%)√ mediastinal lymphadenopathy (29%)√ NO honeycombing*Prognosis:*11% overall mortality*Rx:*corticosteroids (clinical + functional + radiographic improvement in 50-86%)*DDx:*usual interstitial pneumonia (irregular reticular pattern + honeycombing involving subpleural + lower lung zones ***Respiratory Bronchiolitis - Interstitial lung Disease*** =interstitial pneumonia of smokers in which respiratory bronchiolitis is associated with limited peribronchiolar interstitial inflammation; ? early manifestation of DIP*Mean age:*36 years; M=F*Cause:*heavy cigarette smoking*Histo:*accumulation of brown-pigmented macrophages in respiratory bronchioles + surrounding air spaces ● mild dyspnea + cough ● pulmonary function test: mixed restrictive + obstructive√ normal CXR (21%)√ diffuse bibasilar small linear + nodular opacities (71%)√ bibasilar atelectasis (12%)√ bronchial wall thickeningCT: √ scattered ground-glass opacities (66%)√ centrilobular micronodules√ centrilobular emphysema*Prognosis:*excellent (after cessation of smoking / corticoid therapy)

Notes:

Chronic Interstitial Pneumonia =ORGANIZING INTERSTITIAL PNEUMONIA= CHRONIC DIFFUSE SCLEROSING ALVEOLITIS ***Usual Interstitial Pneumonia*** =UIP = IDIOPATHIC PULMONARY FIBROSIS (IPF) = MURAL TYPE OF FIBROSING ALVEOLITIS= CRYPTOGENIC FIBROSING ALVEOLITIS =commonest (90%) form of idiopathic interstitial pneumonia (may represent late stage of DIP)*Etiology:*50% idiopathic; 25% familial; drug exposure (bleomycin, cyclophosphamide (Cytoxan®), busulfan, nitrofurantoin); 20-30% associated with collagen vascular disease / immunologic disorder (mostly rheumatoid arthritis)*Pathophysiology:* repetitive episodes of lung injury to the alveolar wall causing alveoli to flood with proteinaceous fluid + cellular debris; incomplete lysis of intraalveolar fibrin; type II pneumocytes regenerate over the intraalveolar collagen incorporating the fibrous tissue into alveolar septa (= injury-inflammation-fibrosis sequence) *Mean age:*64 years; M>F*Path:*simultaneous presence of inflammatory cell infiltration + fibrotic alveolar walls + honeycombing + areas of normal lung tissue (= temporal variegation)*Histo:*proteinaceous exudate in interstitium + hyaline membrane formation in alveoli; necrosis of alveolar lining cells followed by cellular infiltration of mono- and lymphocytes + regeneration of alveolar lining; intraalveolar histiocytes; proliferation of fibroblasts + deposition of collagen fibers + smooth muscle proliferation; progressive disorganization of pulmonary architecture ● progressive dyspnea, dry cough, fatigue (over 1-3 years) ● "Velcro" rales = crepitations ● clubbing of fingers (83%) ● lymphocytosis on bronchoalveolar lavage (marker of alveolitis) ● pulmonary function tests: restrictive defects + decreased diffusing capacity for carbon monoxide √ occasionally ground-glass pattern in early stage of alveolitis (alveolar wall injury, interstitial edema, proteinaceous exudate, hyaline membranes, infiltrate of monocytes + lymphocytes) in 15-62%√ bilateral diffuse linear / small irregular reticulations (100%); basilar (85%) + peripheral (59%)√ reticulonodular pattern = superimposition of linear opacities√ heart border "shaggy"√ honeycombing = numerous cystic spaces (up to 74%)√ elevated diaphragm = progressive loss of lung volume (45-75%)√ 1.5-3 mm diffusely distributed nodules (15-29%)√ pleural effusion (4-6%), pleural thickening (6%)√ pneumothorax in 7% (in late stages)√ normal CXR (2-8%)HRCT (88% sensitive): Location:lung bases (68-80%)Site:predominantly subpleural regions (79%)√ patchy distribution with areas of normal parenchyma, active alveolitis, early + late fibrosis present at the same time (HALLMARK)√ irregular linear opacities (82%) with architectural distortion of secondary pulmonary lobule √ interlobular septal thickening (10%)√ subpleural areas of honeycombing with cystic spaces outlined by thick fibrous walls (up to 96%)√ subpleural lines (= fibrosis / functional atelectasis)√ small peripheral convoluted cysts (= traction bronchiectasis) in 50%√ ground-glass opacities (= diffuse inflammatory mononuclear cell infiltrates of active disease + fibroblast proliferation) in 65-76%*Cx:*bronchogenic carcinoma (more frequent occurrence)*Rx:*response to steroids in only 10-15%*Prognosis:*average survival of 3-6 years; 45% 5-year mortality rate (overall 87%); no recovery ***Desquamative Interstitial Pneumonia*** =DIP = DESQUAMATIVE TYPE OF FIBROSING ALVEOLITIS=second commonest (although rare) form of interstitial pneumonia with more benign course than UIP, may be self-limited disease or lead to UIP*Mean age:*42 years (approximately 8 years younger than in UIP); M>F*Path:*filling of alveolar spaces with foamy histiocytes + relative preservation of lung architecture + mild fibrosis (temporally homogeneous)*Histo:*alveoli lined by large cuboidal cells + filled with heavy accumulation of mononuclear cells (macrophages, NOT desquamated alveolar cells); relative preservation of alveolar anatomy; histologic uniformity from field to field*Predisposed:*smokers (history in up to 90%) ● asymptomatic ● weight loss ● dyspnea + nonproductive cough (for 6-12 months) ● clubbing of fingers ● mild pulmonary function abnormalities√ normal chest x-ray (3-22%)√ "ground-glass" alveolar pattern sparing costophrenic angles (25-33%), diffuse ground-glass opacities (15%)√ linear irregular opacities (60%), bilateral + basilar (46-73%)√ lung nodules (15%)√ honeycombing (13%)√ preserved lung volumeHRCT: Location:mainly middle + lower lung zones (73%); bilateral + symmetric (86%)Site:predominantly subpleural distribution (59%)√ patchy ground-glass attenuation√ irregular linear opacities (= fibrosis) + architectural distortion (50%)√ honeycombing + traction bronchiectasis (32%)√ fibrosis of lower lung zones in late stage*Prognosis:*better response to corticosteroid Rx than UIP (in 60-80%); median survival of 12 years; 5% 5-year mortality rate (overall 16-27%)

Notes:

IDIOPATHIC PULMONARY HEMOSIDEROSIS
=IPH = probable autoimmune process with clinical + radiologic remissions + exacerbations characterized by eosinophilia + mastocytosis, immunoallergic reaction, pulmonary hemorrhage, iron deficiency anemia*Age:*(a)Chronic form: most commonly <10 years of age(b)Acute form (rare): in adults; M:F = 2:1 ● iron deficiency anemia ● clubbing of fingers ● hepatosplenomegaly (25%) ● bilirubinemia ● recurrent episodes of severe hemoptysis√ bilateral patchy alveolar-filling pattern (= blood in alveoli); initially for 2-3 days with return to normal in 10-12 days unless episode repeated√ reticular pattern (= deposition of hemosiderin in interstitial space) later√ moderate fibrosis after repeated episodes√ hilar lymph nodes may be enlarged during acute episodes*Prognosis:*death within 2-20 years (average survival 3 years)*DDx:*SECONDARY PULMONARY HEMOSIDEROSIScaused by mitral valve disease√ septal lines (NOT in idiopathic form)√ lung ossifications (NOT in idiopathic form)

Notes:

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KARTAGENER SYNDROME
=IMMOTILE / DYSMOTILE CILIA SYNDROME*Incidence:*1:40,00; high familial incidence*Etiology:*abnormal mucociliary function secondary to generalized deficiency of dynein arms of cilia affecting respiratory epithelium, auditory epithelium, spermTriad:(1)Situs inversus (50%)(2)Sinusitis(3)Bronchiectasis ● deafness ● infertility (abnormal sperm tails)*Associated anomalies:* transposition of great vessels, tri- / bilocular heart, pyloric stenosis, postcricoid web, epispadia

Notes:

KLEBSIELLA PNEUMONIA
Most common cause of Gram-negative pneumonias; community acquired *Incidence:*responsible for 5% of adult pneumonias*Organism:*Friedländer bacillus = encapsulated, nonmotile, Gram-negative rod*Predisposed:*elderly, debilitated, alcoholic, chronic lung disease, malignancy ● bacteremia in 25%√ propensity for posterior portion of upper lobe / superior portion of lower lobe√ dense lobar consolidation√ bulging of fissure (large amounts of inflammatory exudate) CHARACTERISTIC but unusual√ empyema (one of the most common causes)√ patchy bronchopneumonia may be present√ uni- / multilocular cavities (50%) appearing within 4 days√ pulmonary gangrene = infarcted tissue (rare)*Cx:*meningitis, pericarditis*Prognosis:*mortality rate 25-50%*DDx:*Acute pneumococcal pneumonia (bulging of fissures, abscess + cavity formation, pleural effusion / empyema frequent)

Notes:

LANGERHANS CELL HISTIOCYTOSIS
=EOSINOPHILIC GRANULOMA=HISTIOCYTOSIS X = LANGERHANS CELL GRANULOMATOSIS=group of disorders of unknown origin characterized by granulomatous infiltration of lungs, bone, skin, lymph nodes, brain, endocrine glands*Manifestation:* (a)multisystem disease with poor prognosis(b)confined to one system: most commonly eosinophilic granuloma of bone*Histo:* granuloma containing Langerhans cells, foamy histiocytes, lymphocytes, plasma cells, eosinophils Langerhans cell -dendritic antigen-presenting cell found in basal layer of skin + in liver (Kupffer cell), lymph nodes, spleen, bone marrow, lung-contains unique mostly rod-shaped cytoplasmatic inclusion bodies known as Birbeck granules (identifiable only with electron microscopy)*Age:*most frequently in 3rd-4th decade (range 3 months to 69 years); M:F = 4:1; Caucasians >> Blacks @Pulmonary Langerhans cell histiocytosis*Pathogenesis:* heavy cigarette smoking in young men with accumulation + activation of Langerhans cells (90% smokers) as a result of excess neuroendocrine cell hyperplasia + secretion of bombazine-like peptides *Path:* multifocal granulomatous infiltration centered on walls of bronchioles (= bronchiolitis) often extending into surrounding alveolar interstitium with subsequent bronchiolar destruction leading to thick-walled cysts presumably caused by check-valve bronchial obstruction + pneumothorax (no necrosis); in end-stage disease foci of LCG are replaced by fibroblasts forming CHARACTERISTIC stellate "starfish" scars with central remnants of persisting inflammatory cells◊CXR abnormalities more severe than clinical symptoms + pulmonary function tests! ● asymptomatic (up to 25%) ● nonproductive cough (75%) ● combination of obstructive + restrictive pulmonary function: presenting with pneumothorax in 15% ● fatigue, weight loss, fever (15-30%) ● dyspnea (40%) ● chest pain (25%) from pneumothorax / eosinophilic granuloma in rib ● diabetes insipidus (10-25%) ● lymphocytosis with predominance of T-suppressor cells on bronchoalveolar lavage (DDx: excess of T-helper cells in sarcoidosis)Location:usually bilaterally symmetric, upper lobe predominance, sparing of costophrenic angles*Evolutionary sequence:* nodule - cavitated nodule - thick-walled cyst - thin-walled cyst√ ill-defined / stellate nodules 3-10 mm (granuloma stage)√ diffuse fine reticular / reticulonodular pattern (cellular infiltrate)√ "honeycomb lung" = multiple 1-5 cm cysts + subpleural blebs (fibrotic stage)√ increased lung volumes in 1/3 (most other fibrotic lung diseases have decreased lung volumes!)√ pleural effusion (8%), hilar adenopathy (unusual)√ cavitation of large nodules (rare)√ thymic enlargementHRCT (combination virtually diagnostic): √ complex / branching thin-walled cysts <5 mm in size equally distributed in central + peripheral lung zones√ centrilobular peribronchiolar nodules√ intervening lung appears normal*DDx for nodules:* sarcoidosis, hypersensitivity pneumonitis, berylliosis, TB, atypical TB, metastases, silicosis, coal workers peumoconiosis *DDx for cysts:* emphysema, bronchiectasis, idiopathic pulmonary fibrosis, lymphangiomyomatosis *Cx:*1.Recurrent pneumothorax in 25% (from rupture of subpleural cysts) CHARACTERISTIC2.Pulmonary hypertension3.Superimposed Aspergillus fumigatus infection*Prognosis:*improvement (50%), stable (33%), rapid progression (20%) @ Bone involvement: √ lytic bone lesions (skull, ribs, pelvis)√ vertebra plana *Prognosis:*poor with multisystem disease + organ dysfunction (especially with skin lesions); complete / partial regression (13-55%), progression (7-21%); 2-25% mortality*Rx:*cessation of smoking, chemotherapy (vincristine sulfate, prednisone, methotrexate, 6-mercaptopurine)*DDx:*sarcoidosis (equal sex distribution, always multisystem disease, not related to smoking, erythema nodosum, bilateral hilar lymphadenopathy, lung cavitation + pneumothorax rare, epitheloid cells)

Notes:

LEGIONELLA PNEUMONIA
=LEGIONNAIRES DISEASE*Organism:*Legionella pneumophila, 1-2 µm, aerobic, gram-negative bacillus, weakly acid-fast, silver-impregnation stain*Predisposed:*middle-aged / elderly, immunosuppressed, alcoholism, chronic obstructive lung disease, diabetes, cancer, cardiovascular disease, chronic renal failure, transplant recipients*Transmission:*direct inhalation (air conditioning systems)*Prevalence:*6% of community-acquired pneumonias*Histo:*leukocytoclastic fibrinopurulent pneumonia with histiocytes in intraalveolar exudate ● fever ● absence of sputum / lack of purulence (22-75%)Clue:involvement of other organs with ● diarrhea (0-25%), myalgia, toxic encephalopathy ● liver + renal disease ● hyponatremia (20%) ● elevated serum transaminase / transpeptidase levels ● lack of quick response to penicillin / cephalosporin / aminoglycoside*Concomitant infection (in 5-10%):* Streptococcus pneumoniae, Chlamydia pneumoniae, Mycobacterium tuberculosis, Pneumocystis carinii Location:unilateral / bilateral (less frequent); lobar / segmental√ patchy bronchopneumonia (= multifocal consolidation)√ moderate volume of pleural effusion (6-30-63%)√ cavitation (rare)*Cx:*progressive respiratory failure (most common cause of death; 6% mortality in healthy patients)*Rx:*erythromycin

Notes:

LIPOID PNEUMONIA

*Etiology:*aspiration of vegetable / animal / mineral oil (most common)*Predisposed:*elderly, debilitated, neuromuscular disease, swallowing abnormalities (eg, scleroderma)Mineral oil =inert pure hydrocarbon that does not initiate cough reflex*Path:*pool of oil surrounded by giant cell foreign body reaction (mineral oil aspiration) / initially hemorrhagic bronchopneumonia (animal fat) ● mostly asymptomatic ● fever, constitutional symptoms ● lipid-laden macrophages in sputum / lavage fluid ● oil droplets in bronchial washing / needle aspirateLocation:predilection for RML + lower lobes√ homogeneous segmental airspace consolidation (most common)√ interstitial reticulonodular pattern (rare)√ paraffinoma = circumscribed peripheral mass (granulomatous reaction + fibrosis often causing stellate appearance)√ slow progression / no changeCT: √ mass of low-attenuation approaching that of subcutaneous fat

Notes:

LÖFFLER SYNDROME
=disorder of unknown etiology characterized by local areas of transient parenchymal consolidation associated with blood eosinophilia*Path:*interstitial + alveolar edema containing a large number of lymphocytes ● no / mild symptoms ● eosinophilia ● history of atopia√ single / multiple areas of homogeneous ill-defined consolidation√ uni- or bilateral, nonsegmental distribution, predominantly in lung periphery√ transient + shifting in nature (changes within one to several days)*Prognosis:*may undergo spontaneous remission

Notes:

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LUNG TORSION
=rare complication of severe chest trauma*Mechanism:*compression of lower thorax with lung twisted through 180°; usually in presence of a large amount of pleural air / fluid*Age:*almost invariably in children√ main lower lobe artery sweeping upward toward apex√ lower lung vessels diminutive√ unusual configuration of lobar collapse√ lung infarction = opacification of involved lung (from edema + hemorrhage into air spaces)

Notes:

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LUNG TRANSPLANT
Indications: emphysema, cystic fibrosis, CHD, idiopathic pulmonary fibrosis, a-1-antitrypsin deficiency, primary pulmonary hypertension, sarcoidosis, pneumoconiosis, malignancy *Survival rate:*90% 1-month survival, 70% 1-year survival

Acute Rejection Of Lung Transplant Anastomotic Complications Of Lung Transplant Chronic Rejection Of Lung Transplant Hyperacute Rejection Posttransplantation Infection Posttransplantation Lymphoproliferative Disease Reperfusion Edema

Notes:

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Acute Rejection Of Lung Transplant *Incidence:*60-80% with 2-3 significant episodes in first 3 months*Histo:*mononuclear cell infiltrate around arteries, veins, bronchioles, alveolar septa with alveolar edema (initially) + fibrinous exudate (later)*Time of onset:*first episode 5-10 days after transplantation; occasionally by 48 hours ● drop in arterial oxygen pressure WITHOUT infection /airway obstruction / fluid overload ● pyrexia, fatigue, decreased exercise tolerance√ heterogeneous opacities in perihilar areas√ ground-glass attenuation on HRCT√ new increasing pleural effusion + septal thickening (most common, 90% specific, 68% sensitive) WITHOUT concomitant signs of LV dysfunction (increase in cardiac size / vascular pedicle width / vascular redistribution)√ subpleural edema, peribronchial cuffing, airspace disease*Dx:*(1)transbronchial biopsy(2)rapid improvement of radiologic abnormalities after treatment with IV bolus of corticosteroids for 3 days*Rx:*methylprednisolone, polyclonal T-cell antibody (antithymocyte globulin), monoclonal antibodies (CD3, OKT3), lymphoid irradiation

Notes:

Anastomotic Complications Of Lung Transplant 1.Airway dehiscence (2-8%)↓ presence of extraluminal air collections at anastomotic site (80%)2.Airway stricture*DDx:*telescoped anastomosis*Rx:*laser resection, balloon bronchoplasty3.Vascular stenosis4.Diaphragmatic hernia from omentopexyProcedure:omental pedicle is harvested at time of transplantation through a small diaphragmatic incision + wrapped around anastomosis to prevent dehiscence

Notes:

Chronic Rejection Of Lung Transplant *Prevalence:*24%*Path:*obliterative bronchiolitis (36%), interstitial pneumonitis, rejection-mediated vasculopathy*Time of onset:*3-75 months after transplantation ● persistent coughing and wheezing ● slowly worsening exertional dyspnea√ increased / diminished lung volumes√ central + peripheral bronchiectasis√ localized airspace disease√ partial lobar atelectasis√ thin irregular areas of increased opacity√ pleural thickening√ diminished peripheral lung markings√ nodular / reticular opacities associated with peribronchial thickening

Notes:

Hyperacute Rejection =rejection in cases of an immunoglobulin G donor-specific HLA antibody positive crossmatch*Path:*acute diffuse alveolar damage

Notes:

Posttransplantation Infection *Cause:*immunosuppression, reduced mucociliary clearance, interruption of lymphatic drainage, direct contact of transplant with environment via airwaysA.INFECTION OF LUNG TRANSPLANT*Prevalence:*35-50%; major cause of morbidity + mortality in early postoperative period*Cause:*? absent cough reflex, impaired mucociliary transport in denervated lung*Organism:*bacteria (23%) > CMV > Aspergillus > Pneumocystis(1)within 1st month:gram-negative bacteria, fungi (candidiasis, aspergillosis)(2)after 1st month:CMV, Pneumocystis carinii, bacteria, fungi ● fever, leukocytosis√ lobar / multilobar consolidation (due to bacterial > fungal pathogens)√ diffuse heterogeneous / ground-glass opacities (due to viral / disseminated fungal pathogens)√ nodular opacities (due to fungal / unusual bacterial pathogens / CMV / septic emboli)*Cx:*may progress rapidly to respiratory failure + death*Dx:*transbronchial / open biopsy (80% accurate)B.EXTRAPULMONARY INFECTIONthoracotomy wound infection, bacteremia, sepsis, empyema, central venous line infection

Notes:

Posttransplantation Lymphoproliferative Disease *Incidence:*4%*Histo:*spectrum from benign polyclonal proliferation of lymphoid tissue to non-Hodgkin lymphoma*Associated with:*Epstein-Barr virus*Time of onset:*1 month to several years; related to immunosuppressive regimen√ solitary / multiple discrete nodules√ mediastinal / hilar lymphadenopathy

Notes:

Reperfusion Edema =REIMPLANTATION RESPONSE=infiltrate appearing within 48 hours after transplantation unrelated to fluid overload, LV failure, infection, atelectasis, or rejection; diagnosed by exclusion*Pathogenesis:*permeability edema due to lymphatic disruption, pulmonary denervation, organ ischemia, trauma*Histo:*fluid accumulation in interstitium consistent with noncardiogenic pulmonary edemaTime course:manifests within 24 hours, peaks at 2nd-4th postoperative day, resolves at variable rate ranging from days to 1-2 weeks to months ● increasing hypoxia before extubation; poor correlation between radiographic severity + physiologic parametersLocation:perihilar areas + basal regions√ perihilar haze / rapid uni- or bilateral heterogeneously dense interstitial and/or air-space disease*Dx:*per exclusion (radiographic changes not due to LV failure, hyperacute rejection, fluid overload, infection, atelectasis)

Notes:

LYMPHANGIOMYOMATOSIS
=LYMPHANGIOLEIOMYOMATOSIS=? forme fruste of tuberous sclerosis=rare disorder characterized by (1) gradually progressive diffuse interstitial lung disease (2) recurrent chylous pleural effusions (3) recurrent pneumothoraces*Etiology:*unknown*Age:*17-50 years, exclusively in women of childbearing age*Histo:*proliferation of atypical smooth muscle in pulmonary lymphatic vessels, blood vessels, and airways*Pathogenesis:* proliferated smooth muscle obstructs (a) bronchioles (trapping of air, overinflation, formation of cysts, pneumothorax), (b) venules (pulmonary edema, hemorrhage, hemosiderosis), (c) lymphatics (thickening of lymphatics, chylothorax) *May be associated with:* Tuberous sclerosis (lung involvement in 1%) ● progressive exertional dyspnea + cough ● disease aggravated by pregnancy + oral contraceptives ● hemoptysis (30-40%), chyloptysis ● radiologic-physiologic discrepancy = severe airflow obstruction (reduced FEV_1, reduced ratio of FEV_1 to forced vital capacity) despite relatively normal findings on CXR ● combination of restrictive + obstructive ventilatory defects: hypoxia, markedly impaired diffusing capacity ● positive immunohistochemical staining of LAM cells with HMB-45 (monoclonal antibody for melanocytic lesion) √ classic signs:√ coarse reticular interstitial pattern (caused by summation of multiple cyst walls)√ recurrent large chylothorax (20-50-75%)√ recurrent pneumothorax (40-50% at presentation; in 80% during course of disease)√ normal / increased lung volume◊The only interstitial lung disease to develop increase in lung volume! √ Kerley-B lines√ pulmonary cysts + honeycombing√ occasionally chylous ascites√ mediastinal + retroperitoneal adenopathy (from smooth muscle proliferation)HRCT: √ numerous randomly scattered thin-walled (<2 mm) cysts of various sizes (0.5-6 cm) surrounded by normal lung parenchyma√ bronchovascular bundles at periphery of cyst walls√ consolidations (due to hemorrhage following destruction of pulmonary microvasculature) @Kidney√ multiple hamartomas lacking fat (50%)√ simple cysts (occasionally large enough to lead to renal insufficiency) *Dx:*open / transbronchial lung biopsy*Prognosis:*8.5-year survival rate of 38-78%; death within 10 years from progressive pulmonary insufficiency *DDx:* (1)Histiocytosis (cyst walls more variable in thickness and in upper lobes, nodules + septal thickening)(2)Idiopathic pulmonary fibrosis = fibrosing alveolitis (small irregular thick-walled cysts + predominantly peripheral interstitial thickening(3)Emphysema (lobular architecture preserved with bronchovascular bundle in central position, areas of lung destruction without arcuate contour)(4)Bronchiectasis (bronchial wall thickening)(5)Tuberous sclerosis (associated skin abnormalities, mental retardation, epilepsy)(6)Neurofibromatosis (cystic air spaces predominantly in apical location)

Notes:

LYMPHANGITIC CARCINOMATOSIS
=INTERSTITIAL CARCINOMA=tumor cell accumulation within connective tissue (bronchovascular bundles, interlobular septa, subpleural space, pulmonary lymphatics) from tumor embolization of blood vessels followed by lymphatic obstruction, interstitial edema, and collagen deposition (fibrosis from desmoplastic reaction when tumor cells extend into adjacent pulmonary parenchyma)*Incidence:*7% of all pulmonary metastasesTumor origin:bronchogenic carcinoma, carcinoma of breast (56%), stomach (46%), thyroid, pancreas, larynx, cervix*mnemonic:* "**C**ertain **C**ancers **S**pread **B**y **P**lugging **T**he **L**ymphatics" **C**ervix**P**ancreas**C**olon**T**hyroid**S**tomach**L**arynx**B**reast*Path:*(1) interstitial edema (2) interstitial fibrotic changes (3) lymphatic dilatation (4) tumor cells within connective tissue planes ● dyspnea (often preceding radiographic abnormalities) ● rarely dry cough + hemoptysisLocation:bilateral; unilateral if secondary to lung primaryCXR (accuracy 23%): √ normal chest radiograph√ reticular densities√ coarsened bronchovascular markings√ Kerley A + B lines√ small lung volume√ hilar adenopathy (20-50%)HRCT: √ well-defined smoothly thickened polygonal reticular network of 10-25 mm in diameter (= thickened interlobular septa)√ irregular / nodular = "beaded" thickening of interlobular septa√ central dot within secondary pulmonary lobule= thickened centrilobular bronchovascular bundle√ subpleural thickening√ pleural effusion (30-50%)√ hilar / mediastinal lymphadenopathy (30-50%)*Prognosis:*death within 1 year*DDx:*(1)Fibrosing alveolitis (peripheral predominance)(2)Extrinsic allergic alveolitis (no polygonal structures, pleural changes rare)(3)Sarcoidosis (nodules of irregular outline more frequent in upper lobes, polygonal structures uncommon)

Notes:

LYMPHOID INTERSTITIAL PNEUMONIA
=LYMPHOCYTIC INTERSTITIAL PNEUMONITIS=LIP = lymphoproliferative disorder characterized by diffuse lymphocytic infiltration of pulmonary interstitium / diffuse lymphoid hyperplasia (probably immunologic disorder) with frequently chronic + progressive course*Histo:*diffuse interstitial infiltrate of polyclonal lymphocytes + plasma cells; many cases reclassified as lymphoma*Associated with:*Sjögren syndrome, systemic lupus erythematosus, myasthenia gravis, pernicious anemia, chronic active hepatitis, AIDS◊Indicative of AIDS when present in child under 13 years of age! ● dyspnea + cough ● cyanosis + clubbing (50%) ● enlargement of salivary glands (20%) ● NO lymphocytosis or history of atopia ● monoclonal gammopathy (usually IgM)√ fine reticular changes in both lungs√ resembling airspace disease (in severe form)√ reticulonodular pattern*Rx:*responsive to steroids Localized form = PSEUDOLYMPHOMA

Notes:

LYMPHOMA

7th leading cause of death from cancer in United States *Pathogenesis:*? viral causeHD:contiguous spread requires scanning of abnormal area onlyNHL:noncontiguous spread requires scanning of chest, abdomen, pelvis @Thorax◊Hodgkin disease more common in thorax than NHL at presentation (HD in 85%, NHL in 45%)1.Lymphadenopathyanterior mediastinal, pretracheal, hilar, subcarinal, axillary, periesophageal, paracardiac, superior diaphragmatic internal mammary lymph nodes 2.Lung parenchyma involvement (HD in 12%, NHL in 4%)3.Pleural + subpleural lymphoma (up to 30%) @Abdomen1.Periaortic adenopathyHD in 25%NHL in 49%2.Mesenteric adenopathyHD in 4%NHL in 51%3.Liver involvementHD in 8%NHL in 14%√ hepatomegaly with involvementHD in <30%NHL in 57%HD:commonly diffuse infiltrating processNHL:diffuse infiltrating / discrete tumor nodules4.Splenic involvementHD in 37%NHL in 41%HD:most common site of abdominal involvementNHL:3rd most common site of abdominal involvement; may be initial manifestation in large cell NHL◊Staging laparotomy necessary as 2/3 of tumor nodules <1 cm in size5.Gastrointestinal involvementin 10% of patients with abdominal lymphoma (uncommon in HD, common in histiocytic NHL); NHL accounts for 80% of all gastric lymphomas 6.Renal involvementlate manifestation, most commonly in NHL 7.Adrenal involvementmore common in NHL 8.Extranodal involvementmore frequent with histologically diffuse forms of NHL

Comparison of Histologic Classifications of Non-Hodgkin Lymphoma	
International Working Formulation	*Rappaport Classification*
Low grade	
A. Small lymphocytic	Well-differentiated lymphocytic
B. Follicular, predominantly small cleaved cell	Nodular, poorly differentiated lymphocytic
C. Follicular, mixed small and large cell	Nodular, mixed
Intermediate grade	
D. Follicular, predominantly large cell	Nodular, histiocytic
E. Diffuse, small cleaved cell	Diffuse, poorly differentiated lymphocytic
F. Diffuse, mixed small and large cell	Diffuse, mixed
G. Diffuse, large cell, cleaved or noncleaved	...
High grade	
H. Diffuse large cell, immunoblastic	...
I. Small, noncleaved cell	...
J. Lymphoblastic	Undifferentiated

Hodgkin Disease Non-Hodgkin Lymphoma Non-Hodgkin Lymphoma In Childhood

Notes:

Hodgkin Disease 40% of all lymphomas; disease of T cells *Age:*bimodal distribution at 25-30 years + >70 years ● asymptomatic unilateral cervical adenopathy*Histo:*Reed-Sternberg cell characteristic 1.nodular sclerosis: most common, localized, good prognosis; greatest adenopathy in anterior mediastinum2.lymphocyte predominance: uncommon, localized, excellent prognosis, majority <35 years3.mixed cellularity: more commonly abdominal than mediastinal, less favorable prognosis4.lymphocyte depletion: uncommon, disseminated, older patients, rapidly fatal *Ann Arbor Staging Classification:* StageI=limited to one / two contiguous anatomic regions on same side of diaphragmI_E=single extralymphatic organ / siteStageII=>2 anatomic regions / two noncontiguous regions on same side of diaphragmII_E=with extralymphatic organ / siteStageIII=on both sides of diaphragm, not extending beyond lymph nodes, spleen (Stage III_S), Waldeyers ringIII_E=with extralymphatic organ / siteStageIV=organ involvement (bone marrow, bone, lung, pleura, liver, kidney, GI tract, skin) ± lymph node involvementSubstage A=absence of systemic symptomsSubstage B=fever, night sweats, pruritus, ≥10% weight loss @CHEST INVOLVEMENT*At presentation:*67% with intrathoracic diseaseSites of lymphoid aggregates: 1. Lymph nodes in mediastinum2. Lymph nodes at bifurcation of 1st + 2nd order bronchi3. Encapsulated lymphoid collections on thoracic surface deep to parietal pleura4. Unencapsulated nodules at points of divisions of more distally situated bronchi, bronchioles, and pulmonary vessels5. Unencapsulated lymphoid aggregates within peribronchial connective tissue6. Small accumulations of lymphocytes in interlobular septa + lymphatic channels A.INTRAPULMONARY MANIFESTATIONSin 15-30-40% during disease duration; most commonly in nodular sclerosing type; invariably subsequent to hilar adenopathy 1.Bronchovascular form (most common type of involvement):√ coarse reticulonodular pattern contiguous with mediastinum = direct extension from mediastinal nodes along lymphatics√ nodular parenchymal lesions√ miliary nodules√ endobronchial involvement√ lobar atelectasis secondary to endobronchial obstruction (rare)√ cavitation secondary to necrosis (rare)2.Subpleural form√ circumscribed subpleural masses√ pleural effusion (20-50%) from lymphatic obstruction3.Massive pneumonic form (68%)√ diffuse nonsegmental infiltrate (pneumonic type)√ massive lobar infiltrates (30%)√ homogeneous confluent infiltrates with shaggy borders√ air bronchogram4.Nodular form√ multiple nodules <1 cm in diameter (DDx: metastatic disease)B.EXTRAPULMONARY MANIFESTATIONS1.Mediastinal + Hilar LymphadenopathyMost common manifestation, present in 90-99%, in thorax commonly multiple lymph node groups involved Location: anterior mediastinal + retrosternal nodes commonly involved (DDx: sarcoidosis); confined to anterior mediastinum in 40%; 20% with mediastinal nodes have hilar lymphadenopathy also; hilar lymph nodes involved bilaterally in 50% Spread from anterior mediastinum to: other mediastinal locations, pleura, pericardium, chest wall ◊Involvement of multiple lymph node groups in 95%!√ CXR: on initial film adenopathy identified in 50%√ necrotic lymph nodes (commonly nodular sclerosing type) √ lymph nodes may calcify following radiation / chemotherapy2.Pleural Effusion (30%)3.Pleural Masses + Plaques(a)sternal erosion(b)invasion of anterior chest wall*Cx:* 1.Superimposed infection√ consolidation with bulging borders: necrotizing bacterial pneumonia√ multiple nodular foci: aspergillosis + nocardiosis√ bilateral diffuse consolidation: Pneumocystis carinii√ rapidly developing cavitation within consolidation: anaerobes / fungus*Dx:*by culture, sputum cytology, lung biopsy2.Drug toxicity @ BONE INVOLVEMENT (15%) √ frequently osteoblastic (28%), eg, ivory vertebrae√ osteolysis of sternum / ribs (direct invasion) *Cx:*increased risk for other malignancies from aggressive therapy (acute leukemia, NHL, radiation-induced sarcoma)

Notes:

Non-Hodgkin Lymphoma =NHL = disease of B cells*Incidence:*3% of all newly diagnosed cancers; 3rd most common cancer in childhood (behind leukemia + CNS neoplasms); 4 times more common than Hodgkin disease*Predisposed:*(40-100 times greater risk) congenital immunodeficiency syndromes, organ transplant patients undergoing immunosuppression, patients with HIV infection, collagen vascular diseases*Age:*all ages; median age of 55 years; M:F = 1.4:1 ● chest / shoulder pain, dyspnea, dysphagia ● CHF, hypotension, SVC syndrome *Modified Rappaport Classification:* =categorization according to histologic distribution of lymphomatous cellsA.Nodular form = organized in clusters1.Poorly differentiated lymphocytic (PDL)2.Mixed lymphocytic / histiocytic (mixed cell)3.Large cell (histiocytic)B.Diffuse form = distortion of tissue architecture1.well-differentiated lymphocytic (WDL)2.intermediate-differentiated lymphocytic (IDL)3.poorly differentiated lymphocytic (PDL)4.mixed lymphocytic / histiocytic large cell (histiocytic) (DLCL); undifferentiated Burkitt lymphoma; undifferentiated non-Burkitt lymphoma (pleiomorphic); lymphoblastic (LBL); unclassified *Luke and Collins classification:* =categorization by morphologic characteristics of cell + cell of origin (T-cell, B-cell, non-B, non-T cell)*Working Formulation Classification (Kiel / Lennert):* =categorization by gradeA.Low grade1.small lymphocytic (3.6%)median age 61 years, 59% 5-year survival 2.follicular, small cleaved cell (22.5%)median age 54 years, 70% 5-year survival 3.follicular, mixed (7.7%)median age 56 years, 50% 5-year survival B.Intermediate grade1.follicular, large cell (3.8%)median age 55 years, 45% 5-year survival 2.diffuse, small cleaved cell (6.9%)median age 58 years, 33% 5-year survival 3.diffuse, mixed (6.7%)median age 58 years, 38% 5-year survival 4.diffuse, large cell (19.7%)median age 57 years, 35% 5-year survival C.High grade1.large cell, immunoblastic (7.9%)median age 51 years, 32% 5-year survival 2.lymphoblastic (4.2%)median age 17 years, 26% 5-year survival 3.small noncleaved cell (5%)median age 30 years, 23% 5-year survival D.Miscellaneous (12%)composite, mycosis fungoides, histiocytic, extramedullary plasmacytoma *Staging:* same Ann Arbor system as for Hodgkin disease Extranodal involvement: @GI tract:stomach (3%), small bowel (5%), large bowel (2%), pancreas (0.7%), peritoneal nodules + ascites (1.4%) @Chest:lung (6%), pleural fluid (3.3%), pericardial fluid (0.7%), heart (0.2%) @GU tract (10%):kidneys (6%), testes (1.2%), ovaries (1.8%), uterus (1.2%) @Bone (3.8%)@CNS (2.4%)@Breast (1.2%)@Skin (6.4%)@Head and neck (1.7%)@Liver (14%)@Spleen (41%) Nodal involvement: @Paraaortic lymph nodes (49%)@Mesenteric lymph nodes (51%):predominantly in middle mediastinum, cardiophrenic angle ◊Single lymph node involvement is often the only manifestation of intrathoracic disease!@Splenic hilar lymph nodes (53%)◊Lymphography 89% sensitive + 86% specific Intrathoracic disease (40-50%): √ hilar + mediastinal adenopathy (DDx: sarcoidosis; anterior nodes favor lymphoma)◊Nodes frequently not involved!√ isolated lymph nodes may enhance (DDx: Castleman disease)√ lung nodules + air bronchograms√ pleural effusion *Prognosis:*unfavorable

Notes:

Non-Hodgkin Lymphoma In Childhood *Incidence:*3rd most common childhood malignancy (after leukemia + CNS tumors); 7% of all malignancies in children <15 years of age*Origin:*B or T cell (in 90%) located outside marrow; (rarely) non-B and non-T cells located within bone marrow*Age:*median age of 10 years; <15 years of age (most common); unusual <5 years of age; M > F ● chest pain, back pain, cough, dyspnea ● fever, anorexia, weight loss ● ± peripheral blood + bone marrow involvement (particularly in lymphoblastic NHL):with lymphoblastic bone marrow involvement of <25% patient is classified as having lymphoma*Staging (St. Jude):* Isingle extranodal tumor / single anatomic areaII(a)single extranodal tumor + regional nodes(b)≥2 nodal areas on same side of diaphragm(c)2 single extranodal tumors ± nodes on same side of diaphragm(d)primary gastrointestinal tract tumor ± nodesIII(a)2 single extranodal tumors on opposite sides of diaphragm(b)≥2 nodal areas on both sides of the diaphragm(c)primary intrathoracic tumors (mediastinum, pleura, thymus)(d)extensive primary intraabdominal disease(e)paraspinal / epidural tumorIVany of the above + initial CNS / bone marrow involvement Differences between adult and childhood NHL: CharacteristicsAdult NHLChildhood NHLPrimary sitenodalextranodalHistology50% follicular,diffuse50% diffuseGradelow, intermediate,highhighHistologic subtypemanythreeSex predilectionnone70% male *Prognosis:*80% cure rate with multiple-agent chemotherapy*DDx:*1.Acute lymphocytic leukemia (>25% lymphoblasts within bone marrow)2.Hodgkin disease (contiguous spread, nodes are site of origin)

1.**Undifferentiated / small noncleaved NHL** (39%);*Path:*non-Burkitt lymphoma; Burkitt lymphoma ● abdominal mass ± ascites ● pain similar to appendicitis / intussusceptionPrimary site:abdomen (distal ileum, cecum, appendix); ovariesCommon site:mesenteric, inguinal, iliac nodes; CNS; bone marrow; kidneyRare site:orbit, supradiaphragmatic paraspinal region, mediastinum, paranasal sinuses, bone, testes, pulmonary parenchyma*Cx:*"leukemic transformation" (= extensive bone marrow involvement) 2.**Lymphoblastic (T-cell) NHL** (28%) Primary site:mediastinum (66%)Common site:neck, thymus, liver, spleen, CNS, bone marrow, gonadsRare site:subdiaphragmatic (ileum, cecum, kidney, mesentery, retroperitoneum), orbit, paranasal sinus, thyroid, parotid ● respiratory distress, dysphagia ● SVC syndrome, pericardial tamponade 3.**Large cell (histiocytic) NHL** (26%)*Origin:*B cell, T cells (small percentage)Location:nodal + extranodalPrimary site:variable (Waldeyer ring, Peyer patches)Common site:peripheral lymph nodes, lung, bone, brain, skinRare site:hard palate, esophagus, trachea

Notes:

MECONIUM ASPIRATION SYNDROME
=most common cause of neonatal respiratory distress in full term / postmature infants (hyaline membrane disease most common cause in premature infants)*Etiology:*fetal circulatory accidents / placental insufficiency / postmaturity result in perinatal hypoxia + fetal distress with meconium defecated in utero*Pathogenesis:*meconium produces bronchial obstruction + chemical pneumonitis*Incidence:*10% of all deliveries have meconium-stained amniotic fluid, 1% of all deliveries have respiratory distress ● cyanosis (rare)√ large infant√ bilateral diffuse grossly patchy opacities (atelectasis + consolidation)√ hyperinflation with areas of emphysema (air trapping)√ spontaneous pneumothorax + pneumomediastinum(25%) requiring no therapy √ small pleural effusions (20%)√ NO air bronchograms√ rapid clearing usually within 48 hours*Cx:*morbidity from anoxic brain damage is high

Notes:

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MEDIASTINAL LIPOMATOSIS
=excess unencapsulated fat deposition*Etiology:* (a)Exogenous steroids (average daily dose of >30 mg prednisone): (1) chronic renal disease, renal transplant (5%) (2) collagen vascular disease, vasculitis (3) hemolytic anemia (4) asthma(5) dermatitis (6) Crohn disease (7) myasthenia gravis (b)Endogenous steroid elevation: (1) adrenal tumor(2) pituitary tumor / hyperplasia = Cushing disease (3) ectopic ACTH-production (carcinoma of the lung) (c)Obesity ● moon facies ● buffalo hump ● supraclavicular + episternal fatLocation:upper mediastinum (common), cardiophrenic angles + paraspinal areas (less common)√ upper mediastinal widening√ paraspinal widening√ increase in epicardial fat-pads√ symmetric slightly lobulated extrapleural deposits extending from apex to 9th rib laterallyOTHER FEATURES: √ osteoporosis√ fractures√ aseptic necrosis√ increased rectosacral distance

Notes:

Benign Mesothelioma =LOCALIZED FIBROUS MESOTHELIOMA=LOCALIZED FIBROUS TUMOR OF THE PLEURA=SOLITARY FIBROUS TUMOR OF PLEURA=BENIGN LOCALIZED MESOTHELIOMA=BENIGN PLEURAL FIBROMA = FIBROSING MESOTHELIOMA = PLEURAL FIBROMYXOMA*Incidence:*<5% of all pleural tumors◊No recognized association with asbestos exposure!*Age:*3rd-8th decade; mean age of 50-60 years;M:F = 1:1 *Path:*usually solitary mass arising from visceral pleura in 80% + parietal pleura in 20%*Histo:*tumor originates from submesothelial fibroblasts, lined by layer of mesothelial cells(a)relatively acellular fibrous tissue(b)rounded spindle-shaped densely packed cells(c)resembling hemangiopericytoma of lung ● asymptomatic in 50% ● cough, fever, dyspnea, chest pain (larger mass) ● digital clubbing (rare) + hypertrophic pulmonary osteoarthropathy in 20-35% ● episodic hypoglycemia (4%)√ sharply circumscribed spherical / ovoid lobular mass of 2-30 cm in diameter located near lung periphery / adjacent to pleural surface / within fissure √ sessile with smooth tapered margin (common) / pedunculated with obtuse angle toward chest wall (rare, benign feature)√ tumor may change in shape + location upon alteration of patients position (if pedunculated)√ areas of hemorrhage / necrosis may be present (favors malignancy)√ ipsilateral pleural effusion (rare) containing hyaluronic acidCT: √ substantial contrast enhancement√ heterogeneous enhancement due to myxoid degeneration + hemorrhageMR: √ hypointense on T1WI + hyperintense on T2WI*Cx:*malignant degeneration in 37%*DDx:*metastatic deposit*Rx:*excision is curative (recurrence rate lower for pedunculated versus nodular tumor)

Notes:

Malignant Mesothelioma =DIFFUSE MALIGNANT MESOTHELIOMA=most common primary neoplasm of pleura*Prevalence:*7-13:1,000,000 persons/year;2,000-3,000 cases/year in US*Etiology:*asbestos exposure (13-100%); zeolite (nonasbestos mineral fiber); chronic inflammation (TB, empyema); irradiation*Carcinogenic potential:* proportional to aspect ratio (= length-to-diameter) of fiber and durability in human tissue: crocidolite > amosite > chrysotile > actinolite, anthophyllite, tremolite ◊Occupational exposure of asbestos found in only 40-80% of all cases!◊5-10% of asbestos workers will develop mesothelioma (risk factor of 30 compared with general population)◊No relation to duration / degree of exposure or smoking history*Latency period:* 20-35-45 years (earlier than asbestosis; later than asbestos-related lung cancer)*Peak age:*50-70 years (66%); M:F = 2-4-6:1*Path:*multiple tumor masses involving predominantly the parietal pleura + to a lesser degree the visceral pleura; progression to thick sheetlike / confluent masses resulting in lung encasement*Histo:* (a) epithelioid (60%) (b) sarcomatoid (15%)(c) biphasic (25%); intracellular asbestos fibers in 25% *Associated with:*peritoneal mesothelioma; hypertrophic osteoarthropathy (10%)*Staging (Boutin modification of Butchart staging)* IAconfined to ipsilateral parietal / diaphragmatic pleuraIB+ visceral pleura, lung , pericardiumIIinvasion of chest wall / mediastinum (esophagus, heart, contralateral pleura) or metastases to thoracic lymph nodesIIIpenetration of diaphragm with peritoneal involvement or metastases to extrathoracic lymph nodesIVdistant hematogenous metastasesStage at presentation:II in 50%, III in 28%, I in 18%, IV in 4% ● nonpleuritic (56%) / pleuritic chest pain (6%) ● dyspnea (53%) ● fever + chills + sweats (30%) ● weakness, fatigue, malaise (30%) ● cough (24%), weight loss (22%), anorexia (10%) ● expectoration of asbestos bodies (= fusiform segmented rodlike structures = iron-protein deposition on asbestos fibers [a subset of ferruginous bodies])Spread: (a)contiguous: chest wall, mediastinum, contralateral chest, pericardium, diaphragm, peritoneal cavity; lymphatics, blood(b)lymphatic: hilar + mediastinal (40%), celiac (8%), axillary + supraclavicular (1%), cervical nodes(c)hematogenous: lung, liver, kidney, adrenal gland√ extensive irregular lobulated bulky pleural-based masses typically >5 cm / pleural thickening (60%)√ exudative / hemorrhagic unilateral pleural effusion (30-60-80%) without mediastinal shift ("frozen hemithorax" = fixation by pleural rind of neoplastic tissue); effusion contains hyaluronic acid in 80-100%; bilateral effusions (in 10%)√ distinct pleural mass without effusion (<25%)√ associated with pleural plaques in 50% = pathologic HALLMARK of asbestos exposure√ pleural calcifications (20%)√ circumferential encasement = involvement of all pleural surfaces (mediastinum, pericardium, fissures) as late manifestation√ extension into interlobar fissures (40-86%)√ rib destruction in 20% (in advanced disease)√ ascites (peritoneum involved in 35%)CT: √ pleural thickening (92%)√ thickening of interlobar fissure (86%)√ pleural effusion (74%)√ contraction of affected hemithorax (42%):√ ipsilateral mediastinal shift√ narrowed intercostal spaces√ elevation of ipsilateral hemidiaphragm√ calcified pleural plaques (20%)MR (best modality to determine resectability): √ minimally hyperintense relative to muscle on T1WI√ moderately hyperintense relative to muscle on T2WIMetastases to: ipsilateral lung (60%), hilar + mediastinal nodes, contralateral lung + pleura (rare), extension through chest wall + diaphragm *Prognosis:*10% of occupationally exposed individuals die of mesothelioma (in 50% pleural + in 50% peritoneal mesothelioma); mean survival time of 5-11 months*DDx:*pleural fibrosis from infection (TB, fungal, actinomycosis), fibrothorax, empyema, metastatic adenocarcinoma (differentiation impossible)*Dx:*video-assisted thoracoscopic surgery (postprocedural radiation therapy of all entry ports for tumor seeding of needle track [21%])

Notes:

METASTASIS TO LUNG

Pulmonary metastases occur in 30% of all malignancies; mostly hematogenous *Age:*>50 years (in 87%)FREQUENCY: *Origin of pulmonary mets*Probability of pulmonary mets 1.Breast22%Kidneyin 75%2.Kidney11%Osteosarcomain 75%3.Head and neck10%Choriocarcinomain 75%4.Colorectal 9%Thyroidin 65%5.Uterus 6%Melanomain 60%6.Pancreas 5%Breastin 55%7.Ovary 5%Prostatein 40%8.Prostate 4%Head and neckin 30%9.Stomach 4%Esophagusin 20% *Incidence of pulmonary metastases: mnemonic:* "CHEST" **C**horiocarcinoma60% **H**ypernephroma / Wilms tumor30 / 20%**E**wing sarcoma18%**S**arcoma (rhabdomyo- / osteosarcoma)21 / 15%**T**esticular tumor12%√ multiple nodules (in 75%) of varying sizes (most typical), 82% subpleural√ fine micronodular pattern: highly vascular tumor (renal cell, breast, thyroid, prostate carcinoma, bone sarcoma, choriocarcinoma)√ pneumothorax (2%): especially in children with bone tumorsCT: √ noncalcified multiple (>10) round lesions >2.5 cm likely to be metastatic√ connection to pulmonary arterial branches (75%)

Solitary Metastatic Lung Nodule Calcifying Lung Metastases (<1%) Cavitating Lung Metastases (4%) Hemorrhagic Lung Metastases Endobronchial Metastases Lung Metastases In Childhood

Notes:

Solitary Metastatic Lung Nodule ◊A solitary lung nodule represents a primary lung tumor in 62% in patients with known Hx of neoplasm◊5% of all solitary nodules are metastatic; most likely origin: colon carcinoma (30-40%), osteosarcoma, renal cell carcinoma, testicular tumor, breast carcinoma

Notes:

Calcifying Lung Metastases (<1%) *mnemonic:*"BOTTOM"**B**reast **O**steo- / chondrosarcoma **T**hyroid (papillary) **T**esticular **O**varian **M**ucinous adenocarcinoma + lung metastases following radiation / chemotherapy

Notes:

Cavitating Lung Metastases (4%) *mnemonic:*"**S**quamous **C**ell **M**etastases **T**end to **C**avitate"**S**quamous cell carcinoma, **S**arcoma **C**olon **M**elanoma **T**ransitional cell carcinoma **C**ervix, under **C**hemotherapy

Notes:

Hemorrhagic Lung Metastases √ ill-defined nodules1.Choriocarcinoma2.Renal cell carcinoma3.Melanoma4.Thyroid carcinoma

Notes:

Endobronchial Metastases √ segmental / subsegmental atelectasis1.Bronchogenic carcinoma2.Lymphoma3.Renal cell carcinoma4.Breast cancer5.Colon carcinoma

Notes:

Lung Metastases In Childhood *mnemonic:*"ROWE"**R**habdomyosarcoma **O**steosarcoma **W**ilms tumor **E**wing sarcoma

Notes:

METASTASIS TO PLEURA
1.Lung (36%)2.Breast (25%)3.Lymphoma (10%)4.Ovary (5%)5.Stomach (2%)

Notes:

MYCOPLASMA PNEUMONIA
=PRIMARY ATYPICAL PNEUMONIA (PAP)Commonest cause of nonbacterial pneumonia with a mild course (only 2% require hospitalization), usually lasts 2-3 weeks; only 10% of infected subjects develop pneumonia *Incidence:*10-33% of all pneumonias; autumn peak*Organism:*Eaton agent = pleuropneumonia-like organism (PPLO) *Age:*most common in ages 5-20 years (esp. in closed populations ● mild symptoms of cough + low fever, malaise, otitis ● mild leukocytosis (20%) ● most common respiratory cause of cold agglutinin production (60%)√ radiologic findings often diverge from clinical condition√ pulmonary infiltrates show a significant lag time√ fine interstitial infiltration from hilum into lower lobe (earliest change)√ alveolar infiltrates: unilateral (L > R) airspace consolidation in segmental lower lobe in 50%, bilateral in 10-40%√ small pleural effusions in 20%√ hilar adenopathy (rare) *Cx:*(1)Meningoencephalitis(2)Erythema nodosum, erythema multiforme, Stevens-Johnson syndrome*Prognosis:*20% with recurrent symptoms of pharyngitis + bronchitis ± infiltrations

Notes:

NEAR DROWNING

1.Sea-water drowning ● hemoconcentration, hypovolemia2.Fresh-water drowning ● hemodilution, hypervolemia ● hemolysis3.Secondary drowning(a)pneumonia with toxic debris(b)progressive pulmonary edema4.Dry drowning (20-40%)=laryngeal spasm prevents water from entering√ no roentgenographic abnormality *Similarities of all 4 types:* ● hypoxemia ● metabolic acidosis√ pulmonary edema√ hyaline membrane formation = considerable loss of protein from blood

Notes:

NEONATAL PNEUMONIA
Pathogenesis: (a)in utero infection (ascending from premature rupture of membranes or prolonged labor / transplacental route)(b)aspiration of infected vaginal secretions during delivery(c)infection after birth*Organism:* (1)Group B streptococcus (GBS): in low-birth-weight premature infants; 50% mortality√ radiographic picture may be identical to RDS (in 52%)√ appearance suggesting retained lung fluid / focal infiltrates (35%)√ normal CXR (13%)√ cardiomegaly√ pleural effusions (in 2/3, but RARE in RDS)√ delayed onset diaphragmatic hernia (evidenced by clinical deterioration)(2)Pneumococci: RDS-like(3)Listeria: RDS-like(4)Candida: progressive consolidation + cavitation(5)Chlamydia: bronchopneumonic pattern ● afebrile ● lower ventilatory pressure requirements√ bilateral focal / diffuse areas of opacities (may initially appear similar to fetal aspiration syndrome)√ hyperaeration√ may cause lobar atelectasis√ may cause pneumothorax / pneumomediastinum√ pleural effusion (exceedingly rare)

Notes:

NOCARDIOSIS

*Organism:*Gram-positive acid-fast bacterium resembling fungus*Predisposed:*immunocompromised√ multiple poorly / well-defined nodules ± cavitation√ lobar consolidation√ empyema without sinus tracts√ SVC obstruction (rare)

Notes:

NONTUBERCULOUS MYCOBACTERIAL INFECTION OF LUNG

=ATYPICAL TUBERCULOSIS*Organisms:* M. kansasii:lung infection in subjects with good immune statusM. marinum:"swimming pool granuloma"M. ulcerans:"Buruli ulcer" in tropical areasM. scrofulaceum:cervical lymphadenitis in infantsM. avium intracellulare:esp. in AIDS *Organism causing pulmonary disease (Runyon classification):* ubiquitous organisms as part of normal environmental flora 1.PhotochromogensM. kansasii, M. simiae, M. asiaticum ● colonies turn yellow with exposure to light◊70-80% of individuals from rural areas test positive on PPD-B (= antigen from M. kansasii)!2.ScotochromogensM. scrofulaceum, M. xenopi, M. szulgai, M. gordonae ● yellow colonies turn orange with exposure to light3.NonchromogensM. avium-intracellulare, M. malmoense, M. terrae ● white / beige colonies without color change4.Rapid growersM. fortuitum-chelonei ● appear in culture in 3-5 days (all other groups appear in culture in 2-4 weeks) *Histo:*lesions indistinguishable from M. tuberculosis*Source:*soil, water, dairy products, bird droppings*Infection:*inhalation of aerosolized water droplets (M. avium-intracellulare complex), food aspiration in patients with achalasia (M. fortuitum-chelonei), GI tract (in AIDS) ● cough (60-100%), hemoptysis (15-20%) ● asthma, dyspnea ● fever distinctly uncommon (10-13%) ● weakness + weight loss (up to 50%) ● weekly positive tuberculin skin test A.CLASSICAL FORM*Age:*6th-7th decade, in Whites (80-90%), M>F*Predisposing factors:* COPD (25-72%), previous TB (20-24%), interstitial lung disease (6%), smoking >30 pack-years (46%), alcohol abuse (40%), cardiovascular disease (36%), chronic liver disease (32%), previous gastrectomy (18%) Location:apical + anterior segments of upper lobes√ chronic fibronodular / fibroproductive apical opacities (indistinguishable from reactivation TB)√ cavitation in 80-95%√ apical pleural thickening in 37-56%√ additional patchy nodular alveolar opacities (due to bronchogenic spread) in ipsi- / contralateral lung in 40-70%√ adenopathy (0-4%)√ pleural effusion (5-20%)√ typically NO hilar elevationB.NONCLASSICAL FORM (20-30%)*Age:*7th-8th decade, 86% in Whites; M:F = 1:4*Predisposing factors:*NONELocation:predominantly in middle lobe + lingula √ multiple bilateral nodular opacities throughout both lungs in random distribution√ irregular curvilinear interstitial opacities (resembling bronchiectasis)C.ASYMPTOMATIC GRANULOMAS√ cluster of similar-sized nodulesD.ACHALASIA-RELATED INFECTIONwith M. fortuitum-chelonei E.DISSEMINATED DISEASEin immunocompromised patients: AIDS, transplant patients, lymphoproliferative disorders (esp., hairy cell leukemia), steroid + immunosuppressive therapy CT: √ multifocal bronchiectasis (79-94%), esp. middle lobe + lingula√ centrilobular nodules of varying sizes, usually <1 cm (= micronodules) in 76-97%√ bronchial wall thickening (97%)√ airspace disease (76%)√ cavitation (21%), esp. in upper lobes√ interlobular septal thickening (12%)◊ Unfavorable response to antituberculous therapy is suspicious for atypical TB!*DDx:*M. tuberculosis (bronchiectasis less common + less extensive), bronchiolitis obliterans, sarcoidosis, fungal disease

Notes:

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PANBRONCHIOLITIS

=inflammatory lung disease, prevalent in Orientals but rare in Europeans + North Americans*Pathogenesis:*unknownHRCT: √ centrilobular branching structures (segments of bronchiolectasis filled with secretions) + nodules surrounding respiratory bronchioles√ mosaic perfusion√ air trapping√ bronchial dilatation*DDx:*bronchiolitis obliterans

Notes:

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PARAGONIMIASIS OF LUNG
=parasitic disease caused by trematode Paragonimus (usually P. westermani = lung fluke) endemic to certain areas of East + Southeast Asia (China, Korea, Japan, Thailand, Laos, Philippines, India)*Infection:*ingestion of raw / incompletely cooked freshwater crab / crayfish infected with metacercaria; larva exists in small intestine + penetrates the intestinal wall + enters peritoneal cavity; larva penetrates diaphragm + pleura to enter the lung*Cycle:*from the final host (tiger, cat, dog, fox, weasel, opossum, human) eggs of worm pass to the outside with blood-streaked sputum; in fresh water ciliated embryos (miracidia) develop; they become tailed larvae (cercariae) after invading a fresh water snail; when the infected snail is eaten by a crustacean, their tails detach and they become 300 µm encysted larvae (metacercariae) @CNS ● meningoencephalitis (in 25%)√ shell-like / soap-bubble-like calcifications of varying size (~50%) CXR (pulmonary lesions in 83%, pulmonary + pleural lesions in 44%, pleural lesions in 17%): early findings (lesions occur 3-8 weeks after ingestion):√ uni- / bilateral pneumo- / hydropneumothorax (17%)√ uni- / bilateral pleural effusion (3-54%)√ focal patchy migrating airspace consolidation(= worm migration causing focal hemorrhagic pneumonia) (45%)√ lobar / segmental collapse (airway obstruction from egg granuloma / intrusion of worm)√ 2-4 mm thick and 2-7 cm long linear opacities abutting the pleura (41%) due to worm migration tracklater findings:√ lung cyst (cyst formation from infarction after arteriolar / venous obstruction by worm or egg; expansion of small airway by intraluminal parasite)√ thick-walled cyst (due to fibrosis)√ "eclipse effect" = eccentric thickening of cyst wall (due to intracystic one / two worms)√ thin-walled cyst (when cyst connected to airway)√ 10-15 mm nodules + masslike consolidation (24%) (due to cyst initially masked by pericystic airspace consolidation ± cyst filled with chocolate-colored necrotic fluid)√ bronchiectasis (35%) *DDx:*tuberculosis (nodular slowly changing lesion, residual fibrosis after treatment, no subpleural linear opacities)

Notes:

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PERICARDIAL CYST

Etiology: (1)defect in embryogenesis of coelomic cavities(2)sequela of pericarditis *Histo:*lined by single layer of mesothelial cells*Age:*30-40 years; M:F = 3:2 ● asymptomatic (50%)Location:(a)cardiophrenic angle (75%), R:L = 3:1 / 3:2, 25% higher; may extend into major fissure(b)mediastinum (rare)√ sharply marginated round / ovoid / triangular mass usually 3-8 cm (range 1-28 cm) in diameter√ change in size + shape with respiration / body position√ attenuation values of 20-40 HU, occasionally higher

Notes:

PNEUMATOCELE
=cystic air collection within lung parenchyma due to obstructive overinflation = regional obstructive emphysema◊does not indicate destruction of lung parenchyma◊ occurs during healing phase◊appears to enlarge while patient improves◊frequently multiple*Developmental theories:* (1)small bronchioles undergo severe distension secondary to check-valve endobronchial / peribronchial obstruction(2)focus of necrotic lung evacuates through a bronchus narrowed by edema / inflammation ; air space subsequently enlarges due to check-valve mechanism from enlarging pneumatocele / inflammatory exudate(3)air from ruptured alveoli / bronchioles dissects along interstitial interlobular tissue and accumulates between visceral pleura and lung parenchyma= subpleural emphysematous bulla = subpleural air cyst
A.PNEUMATOCELE ASSOCIATED WITH INFECTION*Organism:*Pneumococci, E. coli, Klebsiella, Staphylococcus (in childhood)√ appears within 1st week, disappears within 6 weeks√ thin-walled + completely air-filled cavity√ ± air-fluid level + wall thickening (during infection)√ pneumothorax√ spontaneous resolution (in most)B.TRAUMATIC PNEUMATOCELE = PNEUMATOCYST*Cause:* (a)air trapped within area of pulmonary laceration is initially obscured by surrounding contusion (hematoma); pneumatocyst appears within hours after blunt chest trauma(b)intensive inflammatory response from hydrocarbon (furniture polish, kerosene) inhalation / ingestion√ single / multiple pneumatoceles√ spontaneous resolution over several weeks to months

Notes:

PNEUMOCOCCAL PNEUMONIA

Most common Gram-positive pneumonia 90% community-acquired, 10% nosocomial *Incidence:*15% of all adulthood pneumonias, uncommon in child; peaks in winter + early spring; increased during influenza epidemics*Organism:*Streptococcus pneumoniae (formerly Diplococcus pneumoniae), Gram-positive, in pairs / chains, encapsulated, capsular polysaccharide responsible for virulence + serotyping*Susceptible:*elderly, debilitated, alcoholics, CHF, COPD, multiple myeloma, hypogammaglobulinemia, functional / surgical asplenia ● rusty blood-streaked sputum ● left-shift leukocytosis ● impaired pulmonary functionLocation:usually involves one lobe only; bias for lower lobes + posterior segments of upper lobes (bacteria flow under gravitational influence to most dependent portions as in aspiration)√ extensive airspace consolidation abutting against visceral pleura (lobar / beyond confines of one lobe through pores of Kohn) CHARACTERISTIC√ slight expansion of involved lobes√ prominent air bronchograms (20%)√ patchy bronchopneumonic pattern (in some)√ pleural effusion (parapneumonic transudate) uncommon with antibiotic therapy√ cavitation (rare, with Type III)*Variations* (modified by bronchopulmonary disease, eg, chronic bronchitis, emphysema): √ bronchopneumonia-like pattern√ effusion may be only presentation (esp. in COPD)√ empyema (with persistent fever)-in children:√ round pneumonia = sharply defined round lesion*Prognosis:*prompt response to antibiotics (if without complications); 5% mortality rate*Dx:*blood culture (positive in 30%)*Cx:*meningitis, endocarditis, septic arthritis, empyema (now rarely seen)

Notes:

PNEUMOCYSTOSIS
=PNEUMOCYSTIS CARINII PNEUMONIA◊Most common cause of interstitial pneumonia in immunocompromised patients, which quickly leads to airspace disease*Organism:* ubiquitous obligate extracellular protozoan / fungus Pneumocystis carinii (a)trophozoite develops into a cyst(b)cyst produces up to eight daughter sporozoites which are released at maturity + develop into trophozoites*Pathomechanism:* trophozoite attaches to cell membrane of type I alveolar pneumocytes with subsequent cell death + leakage of proteinaceous fluid into alveolar space *Predisposed:* (1)debilitated premature infants, children with hypogammaglobulinemia (12%)(2)AIDS (60-80%)(3)other immunocompromised patients: congenital immunodeficiency syndrome, lymphoproliferative disorders, organ transplant recipients (renal transplant patients in 10%), patients on long-term corticosteroid therapy (nephrotic syndrome, collagen vascular disease), patients on cytotoxic drugs [under therapy for leukemia (40%), lymphoma (16%)]◊Often associated with simultaneous infection by CMV, Mycobacterium avium-intracellulare, herpes simplex ● severe dyspnea + cyanosis over 3-5 days ● subacute insidious onset of malaise + minimal cough (frequent in AIDS patients) ● respiratory failure (5-30%) ● WBC slightly elevated (PMNs) ● lymphopenia (50%) heralds poor prognosis√ normal CXR in 10-39%√ bilateral diffuse symmetric finely granular / reticular interstitial / airspace infiltrates (in 80%) with perihilar + basilar distribution (CHARACTERISTIC central location)√ response to therapy within 5-7 days√ rapid progression to diffuse alveolar homogeneous consolidation (DDx: pulmonary edema)√ air bronchogram√ fine / coarse linear / reticular pattern = thickened coarse interstitial lung markings (in healing phase)√ pleural effusion + hilar lymphadenopathy (uncommon)√ atypical pattern (in 5%):√ isolated lobar disease / focal parenchymal opacities√ lung nodules ± cavitation√ hilar / mediastinal lymphadenopathy√ thin- / thick-walled regular / irregular cysts / cavities with predilection for upper lobes + subpleural regions√ effect of prophylactic use of aerosolized pentamidine:redistribution of infection to upper lobes √ cystic lung disease√ spontaneous pneumothorax, frequently bilateral (6-7%)√ disseminated extrapulmonary disease (1%):√ punctate / rimlike calcifications within enlarged lymph nodes + abdominal visceraCT: √ patchwork pattern (56%)=bilateral asymmetric patchy mosaic appearance with sparing of segments / subsegments of pulmonary lobe√ "ground-glass" pattern (26%)=bilateral diffuse air space disease (fluid + inflammatory cells in alveolar space) in symmetric distribution√ interstitial pattern (18%)=bilateral symmetric / asymmetric, linear / reticular markings (thickening of lobular septa)√ air-filled spaces (38%):(a)pneumatoceles = thin-walled spaces without lobar predilection resolving within 6 months(b)subpleural bullae (due to premature emphysema)(c)thin-walled cysts (? check-valve obstruction of small airways from aerosolized pentamidine)(d)necrosis of PCP granuloma√ pneumothorax (13%)√ lymphadenopathy (18%)√ pleural effusion (18%)√ pulmonary nodulesusually due to malignancy (leukemia, lymphoma, Kaposi sarcoma, metastasis) / septic emboli √ pulmonary cavitiesusually due to superimposed fungal / mycobacterial infection NUC: √ bilateral and diffuse Ga-67 uptake without mediastinal involvement prior to roentgenographic changes*DDx:*TB / MAI infection (with mediastinal involvement)*Dx:*(1) sputum collection (2) bronchoscopy with lavage (3) transbronchial / transthoracic / open lung Bx*Prognosis:*rapid fulminant disease; death within 2 weeks*Rx:*co-trimoxazole IV, nebulized pentamidine

Notes:

PNEUMONECTOMY CHEST

Early signs (within 24 hours): √ partial filling of thorax√ ipsilateral mediastinal shift + diaphragmatic elevationLate signs (after 2 months): √ complete obliteration of spaceN.B.:Depression of diaphragm / shift of mediastinum to contralateral side indicates a bronchopleural fistula / empyema / hemorrhage!

Notes:

POSTOBSTRUCTIVE PNEUMONIA
=chronic inflammatory disease distal to bronchial obstruction*Cause:* 1.Bronchogenic carcinoma (most commonly)2.Bronchial adenoma3.Granular cell myoblastoma (almost always tracheal lesion)4.Bronchostenosis*Histo:*"golden pneumonia" = cholesterol pneumonia endogenous lipid pneumonia = mixture of edema, atelectasis, round cell infiltration, bronchiectasis, liberation of lipid material from alveolar pneumocytes secondary to inflammatory reaction√ frequently associated with some degree of atelectasis√ persists unchanged for weeks√ recurrent pneumonia in same region after antibiotic treatment

Notes:

PROGRESSIVE MASSIVE FIBROSIS
=(PMF) = COMPLICATED PNEUMOCONIOSIS=CONGLOMERATE ANTHRACOSILICOSISMay develop / progress after cessation of dust exposure *Path:*avascular amorphous central mass of insoluble proteins stabilized by cross-links + ill-defined bundles of coarse hyalinized collagen at peripheryLocation:almost exclusively restricted to posterior segment of upper lobe / superior segment of lower lobe√ large >1 cm opacities initially in middle + upper lung zones at periphery of lung√ discoid contour (44%) = mass flat from front to back (thin opacity on lateral view, large opacity on PA view), medial border often ill-defined, lateral borders sharp + parallel to rib cage√ migration toward hila starting at lung periphery; bilateral symmetry√ apparent decrease in nodularity (incorporation of nodules from surroundings)√ cavitation (occasionally) due to ischemic necrosis / superimposed TB infection√ bullous scar emphysema√ pulmonary hypertension

Notes:

PSEUDOLYMPHOMA
=reactive benign lesion = localized form of lymphocytic interstitial pneumonitis (LIP); no progression to lymphoma*Histo:*aggregates of plasma cells, reticulin cells, large + small lymphocytes with preserved lymphoid architecture resembling lymphoma histologically without lymph node involvement*Associated with:*Sjögren syndrome ● mostly asymptomatic√ well-demarcated dense infiltrate√ infiltrate typically in central location extending to visceral pleura√ prominent air bronchogram√ NO lymphadenopathy*Prognosis:*occasionally progression to non-Hodgkin lymphoma*Rx:*most patients respond well to steroids initially

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Notes:

PSEUDOMONAS PNEUMONIA
=most dreaded nosocomial infection because of resistance to antibiotics in patients with debilitating diseases on multiple antibiotics + corticosteroids; rare in community*Organism:*Pseudomonas aeruginosa, Gram-negative ● bradycardia ● temperature with morning peaks√ widespread patchy bronchopneumonia (secondary to bacteremia; unlike other Gram-negative pneumonias)√ predilection for lower lobes√ extensive bilateral consolidation√ "spongelike pattern" with multiple nodules >2 cm(= extensive necrosis with formation of multiple abscesses) √ small pleural effusions

Notes:

PULMONARY ARTERIAL MALFORMATION

=PAVM = PULMONARY ARTERIOVENOUS ANEURYSM = PULMONARY ARTERIOVENOUS FISTULA= PULMONARY ANGIOMA = PULMONARY TELANGIECTASIA=abnormal vascular communication between pulmonary artery and vein (95%) or systemic artery and pulmonary vein (5%)*Etiology:* (a)congenital defect of capillary structure (common)(b)acquired in cirrhosis (hepatogenic pulmonary angiodysplasia), cancer, trauma, surgery, actinomycosis, schistosomiasis*Path:*hemangioma of cavernous type*Pathophysiology:* low-resistance extracardiac R-to-L shunt (which may result in paradoxical embolism); quantification with Tc-99m-labeled albumin microspheres by measuring fraction of dose reaching kidneys *Age:*3rd-4th decade; manifest in adult life, 10% in childhood*Occurrence:* (a)isolated abnormality (40%)(b)multiple (in 1/3)*associated with*Rendu-Osler-Weber syndrome (in 30-60-88%) = hereditary hemorrhagic telangiectasia Only 5-15% of patients with Rendu-Osler-Weber disease have pulmonary AVMs!*Types:* 1.Simple type (79%)=single feeding artery empties into a bulbous nonseptated aneurysmal segment with a single draining vein2.Complex type (21%)=more than one feeding artery empties into septated aneurysmal segment with more than one draining vein ● asymptomatic in 56% (until 3rd-4th decade) if AVM single and <2 cm ● orthodeoxia (= increased hypoxemia with PaO_2 <85 mm Hg in erect position due to gravitational shift of pulmonary blood flow to base of lung) ● cyanosis with normal-sized heart (R-to-L shunt) in 25-50%, clubbing ● bruit over lesion (increased during inspiration) ● dyspnea on exertion (60-71%) ● epistaxis (79%) ● palpitation, chest pain ● No CHFLocation:lower lobes (65-70%) > middle lobe > upper lobes; bilateral (8-20%); medial third of lung√ sharply defined, lobulated oval / round mass (90%) of 1 to several cm in size ("coin lesion")√ cordlike bands from mass to hilum (feeding artery + draining veins)√ in 2/3 single lesion, in 1/3 multiple lesions√ enlargement with advancing age√ change in size with Valsalva / Mueller maneuver / erect vs. recumbent position (decrease with Valsalva maneuver)√ phleboliths (occasionally)√ increased pulsations of hilar vesselsCT (98% detection rate): √ homogeneous circumscribed noncalcified nodule / serpiginous mass up to several cm in diameter√ vascular connection of mass with enlarged feeding artery + draining vein√ sequential enhancement of feeding artery + aneurysmal part + efferent vein on dynamic CTMR: (if contraindication to contrast / slow flow due to partial thrombosis / follow-up) √ signal void on standard spin echo / high signal intensity on GRASS imagesAngio (mostly obviated by MR / CT unless surgery or embolization contemplated) *Cx:*CNS symptoms are commonly the initial manifestation(1)Cerebrovascular accident: stroke (18%), transient ischemic attack (37%) secondary to paradoxical bland emboli(2)Brain abscess (5-9%) secondary to loss of pulmonary filter function for septic emboli(3)Hemoptysis (13%) secondary to rupture of PAVM into bronchus, most common presenting symptom(4)Hemothorax (9%) secondary to rupture of subpleural PAVM(5)Polycythemia*Prognosis:*26% morbidity, 11% mortality*DDx:* solitary / multiple pulmonary nodules*Rx:*embolization with coils / detachable balloons

Notes:

PULMONARY CAPILLARY HEMANGIOMATOSIS
=bilateral pulmonary disease behaving like a low-grade nonmetastatic vascular neoplasm with slowly progressive pulmonary hypertension*Histo:*sheets of thin-walled capillary blood vessels infiltrating pulmonary interstitium + invading pulmonary vessels, bronchioles, and pleura*Pathomechanism of pulmonary hypertension:* (a)veno-occlusive phenomenon secondary to invasion of small pulmonary veins(b)progressive vascular obliteration secondary to in situ thrombosis + infarction(c)pulmonary scar formation secondary to recurrent pulmonary hemorrhage*Age:*20-40 years ● dyspnea on exertion ● cor pulmonale: jugular venous distension, pedal edema, ECG-signs of RV failure (DDx: pulmonary veno-occlusive disease) ● elevated PA pressures + normal pulmonary wedge pressure ● hemoptysis + pleuritic chest pain in 1/3(DDx: pulmonary thromboembolic disease)CXR: √ diffuse reticulonodular pattern√ focal areas of interstitial fibrosis (recurrent episodes of pulmonary hemorrhage + thrombotic infarction)CT: √ thickening + nodularity of inter- and intralobular septa + walls of pulmonary veins√ areas of ground-glass attenuation (= increased perfusion to extensive proliferating hemangiomatous tissue)Angio: √ combination of increased flow (to hemangiomatous areas) + decreased flow (to regions of thrombosis, infarction, and scarring)*Prognosis:*death after 2- to 12-year interval from onset of symptoms*Rx:*bilateral lung transplantation*DDx:*(1)Pulmonary veno-occlusive disease(2)Idiopathic interstitial fibrosis(3)Primary pulmonary hypertension (no increase in lung markings)(4)Pulmonary hemangiomatosis (only in children, cavernous hemangiomas involving several organs)

Notes:

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PULMONARY CONTUSION
=most common manifestation of blunt chest trauma, esp. deceleration trauma*Path:*exudation of edema + blood into air space + interstitium*Time of onset:*apparent within 6 hours after trauma ● clinically inapparent ● hemoptysis (50%)Location:posterior (in 60%)Site:directly deep to site of impact / contrecoup√ irregular patchy / diffuse homogeneous extensive consolidation (CT is more sensitive)√ opacity may enlarge for 48-72 hours√ rapid resolution beginning 24-48 hours, complete within 2-10 days√ overlying rib fractures (frequent)CT: √ nonsegmental coarse ill-defined crescentic (50%) / amorphous (45%) opacification of lung parenchyma without cavitation√ "subpleural sparing" = 1-2 mm rim of uniformly nonopacified subpleural portion of lung*Cx:*pneumothorax*DDx:*fat embolism (1-2 days after injury)

Notes:

PULMONARY INTERSTITIAL EMPHYSEMA
=PIE = complication of respirator therapy with PEEP*Pathogenesis:* gas escapes from overdistended alveolus, dissects into perivascular sheath surrounding arteries, veins, and lymphatics, tracks into mediastinum forming clusters of blebs; **air-block** = compression + obstruction of pulmonary veins + mediastinal structures by interstitial pulmonary emphysema / pneumomediastinum / pneumothorax (obstruction esp. during expiration) ● sudden deterioration in patients condition during respiratory therapy√ elongated lucencies following distribution of bronchovascular tree√ circular densities√ bilateral, symmetrical distribution√ lobar overdistension (occasionally)*Cx:*pneumomediastinum, pneumothorax, subcutaneous emphysema, pneumopericardium, intracardiac air, pneumoperitoneum, pneumatosis intestinalis

Notes:

PULMONARY LYMPHANGIOMATOSIS

=increased number of communicating lymphatic channels√ smooth thickening of bronchovascular bundles + interlobular septaCT: √ diffuse increased attenuation of mediastinal fat√ mild perihilar infiltration√ pleural effusion√ pleural thickening

Notes:

PULMONARY MAINLINE GRANULOMATOSIS
=PULMONARY TALCOSIS=microscopic pulmonary embolism in drug addicts from IV injection of talc-containing drugs (ground tablets)*Drugs:*amphetamines, methylphenidate hydrochloride ("West coast"), tripelen amine ("blue velvet"), methadone hydrochloride, dilaudid, meperidine, pentazocine, propylhexedrine, hydromorphone hydrochlorideadded talc (= magnesium silicate) particles incite a granulomatous foreign-body reaction + subsequent fibrosis in perivascular distribution talc retinopathy (80%) = small glistening crystals angiothrombotic pulmonary hypertension + cor pulmonaleEarly changes: widespread micronodularity of "pinpoint" size (1-3 mm) with perihilar / basilar predominance well-defined nodules predominantly in middle zonesLate changes: loss of lung volume coalescent opacities similar to progressive massive fibrosis (DDx: in silicosis away from hila)*DDx of late changes:* (1)Progressive massive fibrosis of silicosis / coal workers pneumoconiosis(2)Chronic sarcoidosis*Dx:*lung biopsy

Notes:

PULMONARY THROMBOEMBOLIC DISEASE
=PULMONARY EMBOLISM (PE)*Prevalence:*630,000 Americans/year with missed / delayed diagnosis in 400,000 causing death in 120,000; diagnosed in 1% of all hospitalized patients; in 12-64% at autopsy; in 9-56% of patients with deep venous thrombosis*Age:*60% >60 years of age*Cause:*deep vein thrombosis (DVT) of LE in >90%; PE usually occurs within first 5-7 days of thrombus formation*Predisposing factors:*immobilization (56%), surgery (54%) *Pathophysiology:*A clot from the deep veins of the leg breaks off + fragments in right side of heart + showers lung with emboli varying in size◊On average >6-8 vessels are embolized! Class 1 = <20% of pulmonary arteries occluded ● asymptomatic ● normal arterial blood gas levels ● normal pulmonary + systemic hemodynamicsClass 2 = 20-30% of pulmonary arteries occlude ● anxiety, hyperventilation ● arterial PO_2 <80 torr ● PCO_2 <35 torrClass 3 = 30-50% of pulmonary arteries occluded ● dyspnea, collapse ● arterial PO_2 <65 torr ● arterial PCO_2 <30 torr ● elevated central venous pressureClass 4 = >50% of pulmonary arteries occluded ● shock, dyspnea ● arterial PO_2 <50 torr ● arterial PCO_2 <30 torr ● elevated central venous pressure ● mean PA pressure >20 mm Hg ● systolic blood pressure <100 mm Hg ● Classic triad (<33%):(1) hemoptysis (25-34%) (2) pleural friction rub (3) thrombophlebitis◊only 10-33% of patients with fatal PE are symptomatic for DVT◊DVT diagnosed ante mortem in <30%◊clinically suspected diagnosis accurate in 26-45%◊30% of patients with angiographically detected PE have negative bilateral venograms ("big bang" theory = clot embolizes in toto to lung leaving no residual in leg veins) ● may be asymptomatic ● false-positive clinical diagnosis in 62% ● acute dyspnea (81-86%) ● pleuritic chest pain (58-72%) ● apprehension (59%) ● cough (54-70%) ● tachycardia, tachypnea ● accentuated 2nd heart sound ● ECG changes (83%), mostly nonspecific: P-pulmonale, right-axis deviation, right bundle branch block, classic $S_1Q_3T_3$ pattern ● bronchospasm (histamine-mediated), bronchial plugging, rales (loss of surfactant) ● elevated levels of fibrinopeptide-A (FPA) = small peptide split off of fibrinogen during fibrin generation ● positive D-dimer assay (generated during clot lysis)Location of PE:bilateral emboli (in 45%), RT lung only (36%), LT lung only (18%); multiple emboli [3-6 on average] in 65%Distribution:RUL (16%), RML (9%), RLL (25%), LUL (14%), LLL (26%)Site:central = segmental / larger (in 58%);peripheral = subsegmental / smaller (in 42%); in subsegmental branches exclusively (in 30%)◊Emboli are occlusive in 40%! RESOLUTION OF PE (through fibrinolysis + fragmentation): in 8% by 24 hours, in 56% by 14 days, in 77% by 7 months; complete in 65%, partial in 23%, no resolution in 12% ◊Resolution less favorable with increasing age + cardiac disease◊Resolution improved with urokinase > heparin within first week (after 1 year 80% for both) A.EMBOLISM WITHOUT INFARCTION (90%)*Histo:*hemorrhage + edema√ normal chest film common (>29%), abnormal CXR in 40-93%◊A normal CXR has a negative predictive value of only 74%!√ platelike atelectasis ± segmental / lobar consolidation in lower lung zones + pleural effusion (most common findings with the lowest positive predictive value)√ Westermark sign = area of oligemia (due to vasoconstriction distal to embolus) in 2%√ Fleischner sign = local widening of artery by impaction of embolus (due to distension by clot / pulmonary hypertension developing secondary to peripheral embolization)√ "knuckle sign" = abrupt tapering of an occluded vessel distally B.EMBOLISM WITH INFARCTION (10-60%)=any opacity developing as a result of thromboembolic disease; more likely to develop in presence of cardiopulmonary disease with obstruction of pulmonary venous outflow (diagnosed in retrospect)*Histo:*(1)incomplete infarction = reversible transient hemorrhagic congestion / edema usually resolving over several days to weeks(2)complete infarction = hemorrhagic infarction with necrosis of lung parenchyma remaining permanently√ segmentally distributed wedge-shaped consolidation (54%)√ ± cavitation√ Hampton hump = pleural-based shallow consolidation in form of a truncated cone with base against pleural surface + convex medial border√ pleural effusion (54%)√ thoracentesis: bloody (65%), predominantly PMNs (61%), exudate (65%)√ NO air-bronchogram (hemorrhage into alveoli)√ "melting sign" = within few days to weeks regression from periphery toward center√ Fleischner lines = long-line shadows (fibrotic scar) from invagination of pleura at the base of the collapse resulting in pseudofissure√ platelike atelectasis (27%)√ cardiomegaly / CHF (17%)√ elevated hemidiaphragm (17%)√ subsequent nodular / linear scar CT (spiral CT equal to angio in detection of emboli within proximal arteries of ≤5th / 6th generation): ◊ Subsegmental intraluminal filling defects (in 30%) usually not detectable!◊Detection poor in middle lobe + lingular branches (in 18%)!√ peripheral wedge-shaped lung densities with the triangle base adjacent to pleural surface√ vascular connection to a branch of pulmonary artery√ peripheral rimlike contrast enhancement√ intraluminal filling defect in pulmonary artery NUC (VQ scan = guide for angiographic evaluation) interpreted in reference to Biello or PIOPED criteria (*see* page 910) √ low- / intermediate-probability scans (73%): additional studies recommended√ high-probability scan: in 12% normal angiogram Angio (indicated within 24 hours of indeterminate NUC scan): √ intraluminal defect (94%)√ abrupt termination of pulmonary arterial branch√ pruning + attenuation of branches√ wedge-shaped parenchymal hypovascularity√ absence of draining vein in affected segment√ tortuous arterial collaterals *Cx of pulmonary angiography (1-2%):* arrhythmia, endocardial injury, cardiac perforation, cardiac arrest, contrast reaction *Mortality rate of pulmonary angiography:*0.2-0.5%False-negative rate: 1-4-9% due to difficulty in visualizing subsegmental emboli (with only 30% interobserver agreement about presence of subsegmental emboli)

Acute Thromboembolic Pulmonary Arterial Hypertension Chronic Thromboembolic Pulmonary Arterial Hypertension

Notes:

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Acute Thromboembolic Pulmonary **Arterial Hypertension** Hypertension disappears as emboli lyse ● sudden onset of chest pain ● acute dyspnea ● hemoptysis occasionally*Mortality:* 3:1,000 surgical procedures; 200,000 deaths in 1975; 7-10% of all autopsies (death within first hour of PE in most patients); 26-30% if untreated; 8% if treated; fatal if >60% of pulmonary bed obstructed; healthy patients may survive obstruction of 50-60% of vascular bed *Rx:* 1.Heparin IV: 10,000-15,000 units as initial dose; 8,000-10,000 units/hour during diagnostic evaluation; continued for 10-14 days2.Streptokinase: better results with massive PE3.Urokinase: slightly better than streptokinase4.Coumadin: maintained for at least 3 months (15% complication rate)

Notes:

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Chronic Thromboembolic Pulmonary **Arterial Hypertension** ● history of previous embolic episodes ● dyspnea on exertion (DDx: interstitial lung disease) ● may be clinically silentCT (77% sensitive): √ vascular abnormalities:√ direct visualization of thrombus (70%)√ mural arterial irregularities ± abrupt narrowing / cutoff√ decrease in caliber of small branches + narrowing of peripheral pulmonary vessels√ main pulmonary artery diameter >28.6 mm√ parenchymal abnormalities:√ wedge-shaped pleura-based parenchymal bands with tip pointing to hila, often multiple, esp. involving lower lung (70%) = infarcted tissue replaced by scar√ scattered geometric areas of low attenuation in 55% (due to oligemia) associated with vessels of small cross-sectional diameter√ regional sharply demarcated areas of high attenuation (perfused lung on background of oligemic / nonperfused lung)√ cylindric bronchial dilatation of segmental / subsegmental bronchi (64%)

Notes:

PULMONARY VENOUS VARIX
=abnormal tortuosity + dilatation of pulmonary vein just before entrance into left atrium*Etiology:*congenital / associated with pulmonary venous hypertension ● usually asymptomatic; may cause hemoptysisLocation:medial third of either lung below hila close to left atrium√ well-defined lobulated round / oval mass√ change in size during Valsalva / Mueller maneuver√ opacification at same time as LA (on CECT)*Risk:*(1)death upon rupture during worsening heart failure(2)source of cerebral emboli*DDx:*pulmonary arteriovenous fistula

Notes:

RADIATION PNEUMONITIS
=damage to lungs after radiation therapy dependent on:(a)irradiated lung volume(b)radiation dose: unusual if <2000 R given in 2-3 weeks; common if >6000 R given in 5-6 weeks(c)fractionation of dose(d)concurrent / later chemotherapy*Pathologic phases:* (1)Exudative phase = edema fluid + hyaline membranes(2)Organizing phase(3)Fibrotic phase = interstitial fibrosis*Time of onset:*usually 4-6 months after treatmentLocation:confined to radiation port1.ACUTE RADIATION PNEUMONITIS(within 1-8 weeks after radiation therapy) *Path:*depletion of surfactant (1 week to 1 month later), plasma exudation, desquamation of alveolar + bronchial cells ● asymptomatic (majority) ● nonproductive cough, shortness of breath, weakness, fever (insidious onset) ● acute respiratory failure (rare)√ changes usually within portal entry fields√ patchy / confluent consolidation, may persist up to 1 month (exudative reaction)√ atelectasis + air bronchogram√ spontaneous pneumothorax (rare)CT: √ homogeneous slight increase in attenuation (2-4 months after therapy)√ patchy consolidation (1-12 months after therapy)√ nonuniform discrete consolidation (most common; 3 months to 10 years after therapy)*Prognosis:*recovery / progression to death / fibrosis*Rx:*steroids2.CHRONIC RADIATION DAMAGE(9-12 months after radiation therapy) *Histo:*permanent damage of endothelial + type I alveolar cells*May be associated with:* (1)thymic cyst(2)calcified lymph nodes (in Hodgkin disease)(3)pericarditis + effusion (within 3 years)√ severe loss of volume√ dense fibrous strands from hilum to periphery√ thickening of pleura√ pericardial effusionCT: √ solid consolidation (radiation fibrosis) + bronchiectasis (stabilized by 1 year after therapy)

Notes:

RESPIRATORY DISTRESS SYNDROME OF NEWBORN

=RDS = HYALINE MEMBRANE DISEASE=acute pulmonary disorder characterized by generalized atelectasis, intrapulmonary shunting, ventilation-perfusion abnormalities, reduced lung compliance*Cause:*immature surfactant production (usually begins at 18-20 weeks of gestational age) causing acinar atelectasis + dilatation of terminal airways*Predisposed:*perinatal asphyxia, cesarean section, infants of diabetic mothers, premature infants (<1000 g in 66%; 1000 g in 50%; 1500 g in 16%; 2000 g in 5%; 2500 g in 1%)*Onset:*<2-5 hours after birth, increasing in severity from 24 to 48 hours, gradual improvement after 48-72 hours; M:F = 1.8:1 ● abnormal retraction of chest wall ● cyanosis (carbon dioxide retention) ● expiratory grunting ● increased respiratory rate√ hypoaeration with loss of lung volume (counteracted by respirator therapy)√ reticulogranular pattern (coincides with onset of clinical signs)√ prominent air bronchograms (distension of compliant airways)√ bilateral + symmetrical distribution*Prognosis:*spontaneous clearing within 7-10 days (mild course in untreated survivors); death in 18% ACUTE COMPLICATIONS OF RDS (a)Barotrauma with air-block phenomena1.Parenchymal pseudocyst2.Pulmonary interstitial emphysema3.Pneumomediastinum, -thorax, -pericardium, -peritoneum,-retroperitoneum4.Subcutaneous emphysema5.Gas embolism(b)Diffuse opacity1.Worsening RDS2.Superimposed pneumonia3.Massive aspiration4.Pulmonary hemorrhage5.Congestive heart failure (PDA, fluid overload)(c)Persistent patency of ductus arteriosusoxygen stimulus is missing to close duct; gradual decrease in pulmonary resistance (by end of 1st week) leads to L-to-R shunt through PDA (d)Hemorrhage1.Pulmonary hemorrhage2.Intracranial hemorrhage(e)Necrotizing enterocolitis(f)Acute renal failure CHRONIC COMPLICATIONS OF RDS 1.Lobar emphysema2.Localized interstitial emphysema3.Delayed onset of diaphragmatic hernia4.Recurrent inspiratory tract infections5.Hyperinflation6 Bronchopulmonary dysplasia (10-20%)7.Retrolental fibroplasia8.Subglottic stenosis (intubation)*Rx:*exogenous surfactant intratracheally

Notes:

RHEUMATOID LUNG

*Incidence:*2-54% of patients with rheumatoid arthritis; M:F = 5:1 (although incidence of rheumatoid arthritis: M < F) ● rheumatoid arthritisStage 1:multifocal ill-defined alveolar infiltratesStage 2:fine interstitial reticulations (histio- and lymphocytes)Stage 3:honeycombing A.PLEURAL ABNORMALITIES (most frequent manifestation) ● Hx of pleurisy (21%)√ pleural effusion (3%): unilateral (92%), with little change over months; M:F = 9:1; most often without other pulmonary changes, may antedate rheumatoid arthritis ● exudate (with protein content >4 g/dL) ● low in sugar content (<30 mg/dL) without rise during glucose infusion (75%) ● low WBC high in lymphocytes ● positive for rheumatoid factor, LDH, RA cells√ pleural thickening, usually bilateralB.DIFFUSE INTERSTITIAL FIBROSIS (30%) ● restrictive ventilatory defectLocation:lower lobe predominance*Histo:*deposition of IgM in alveolar septa (DDx to IPF)√ punctate / nodular densities (mononuclear cell infiltrates in early stage) √ reticulonodular densities√ medium to coarse reticulations (mature fibrous tissue in later stage)√ honeycomb lung (uncommon in late stage)C.NECROBIOTIC NODULES (rare)=well-circumscribed nodular mass in lung, pleura, pericardium identical to subcutaneous nodules associated with advanced rheumatoid arthritis*Path:*central zone of eosinophilic fibrinoid necrosis surrounded by palisading fibroblasts; nodule often centered on necrotic inflamed blood vessel (? vasculitis as initial lesion) ● subcutaneous nodules (same histology)*Associated with:*interstitial lung disease√ well-circumscribed usually multiple nodules of 3-70 mm in size√ commonly located in lung periphery√ cavitation with thick symmetric walls + smooth inner lining (in 50%)√ NO calcificationD.CAPLAN SYNDROME=RHEUMATOID PNEUMOCONIOSIS=pneumoconiosis + rheumatoid arthritis in coal workers with rheumatoid disease;=hypersensitivity reaction to irritating dust particles in lungs of rheumatoid patients*Incidence:*2-6% of all men affected by pneumoconioses (exclusively in Wales)*Path:*disintegrating macrophages deposit a pigmented ring of dust surrounding the central necrotic core + zone of fibroblasts palisading the zone of necrosis√NOT necessarily evidence of long-standing pneumoconiosis ● concomitant with joint manifestation (most frequent) / may precede arthritis by several years ● concomitant with systemic rheumatoid nodules√ rapidly developing well-defined nodules of 5-50 mm in size with a tendency to appear in crops predominantly in upper lobes + in periphery of lung√ nodules may remain unchanged / increase in number / calcify√ background of pneumoconiosis√ pleural effusion (may occur)E.BRONCHIAL ABNORMALITIES (30%)√ bronchiectasis√ bronchiolitis obliterans (may be transient + related to penicillamine therapy)F.PULMONARY ARTERITIS=fibroelastoid intimal proliferation of pulmonary arteries ● pulmonary arterial hypertension + cor pulmonaleG.CARDIAC ENLARGEMENT(pericarditis + carditis / congestive heart failure) H.BONE ABNORMALITIES ON CXR√ arthritis of acromioclavicular joint, sternoclavicular joint, shoulder joint√ ankylosis of vertebral facet joints√ vertebral body collapse due to steroid use

Notes:

ROUND PNEUMONIA
=NUMMULAR PNEUMONIA = fairly spherical pneumonia caused by pyogenic organisms*Organism:*Haemophilus influenzae, Streptococcus, Pneumococcus*Age:*children >> adults ● cough, chest pain, feverLocation:always posterior, usually in lower lobes√ spherical infiltrate with slightly fluffy borders + air bronchogram√ triangular infiltrate abutting a pleural surface (usually seen on lateral view)√ rapid change in size and shape

Notes:

SARCOIDOSIS
=BOECK SARCOID [*sarcoid = sarcoma-like, Caesar Boeck describes skin lesions in 1899]*
=immunologically mediated multisystem granulomatous disease of unknown etiology with variable presentation, progression, and prognosis*Prevalence:*10-40:100,000 in United States*Age peak:*20-40 years; M:F = 1:3 (female predominance only in Black population); American Blacks:American Whites = 10:1 (rare in African / South American Blacks); more common in blood group A*Immunology:* unknown antigen activates alveolar macrophages which release -interleukin-1 (T-cell activator)-fibronectin (fibroblast chemotactic factor)-alveolar macrophage-derived growth factor (stimulates fibrosis)and activates T lymphocytes which release -interleukin-2 (stimulates growth of T-helper / cytolytic cells)-immune interferon (polyclonal B-cell activator)-monocyte chemotactic factor (attracts circulating monocytes and stimulates granuloma formation) *Histo:*alveolitis (earliest changes); noncaseating epithelioid granulomas [composed of lymphocytes, peripheral fibroblasts, multinucleated giant cells] with occasional minimal central necrosisLocation:along course of lymphatic vessels: subpleural, septal, perivascular, peribronchial*DDx:*indistinguishable from granulomas of berylliosis, treated TB, leprosy, fungal disease, hypersensitivity pneumonitis, Crohn disease, primary biliary cirrhosis ● angiotensin-converting enzyme (ACE) elevated in 70% [ACE is a product of macrophages and an indicator for the granuloma burden of the body]*DDx:*tuberculosis, leprosy, histoplasmosis, berylliosis, cirrhosis, hyperthyroidism, diabetes ● hypercalcemia + hypercalciuria in 2-15% [result of hydroxylation of 1,25-dihydroxy vitamin D in macrophages leading to increased intestinal resorption of calcium] ● Kveim-Stiltzbach test (positive in 70%) = intracutaneous injection of previously validated saline suspension of human sarcoid spleen / lymph nodes, rarely used ● functional pulmonary impairment (even with NO radiographic abnormality):-reduced VC + FRC + TLC [from generalized reduction in lung volume]-low lung compliance [from diffuse interstitial disease]-obstructive airway disease [from endobronchial lesions, peribronchial fibrosis]*Epidemiology:* found with varying frequency in every country in the world; higher prevalence in temperate climates compared to tropical regions (<10/100,000) A.ACUTE FORM = **Löfgren Syndrome** (17%) ● fever + malaise + bilateral hilar adenopathy ● erythema nodosum ● arthralgia of large joints ● (occasionally) uveitis + parotitisB.CHRONIC FORM ● asymptomatic (50%) ● fever, malaise, weight loss ● dry cough + shortness of breath (25%) ● hemoptysis in 4% (from endobronchial lesion / vascular erosion / cavitation) *Stage at presentation:* 0normal chest radiograph5%IIlymphadenopathy only50%IIlymphadenopathy + parenchymal disease30%IIIparenchymal disease only15%IVpulmonary fibrosis20%*Prognosis:* 75%complete resolution of hilar adenopathy33%complete resolution of parenchymal disease30%improve significantly20%irreversible pulmonary fibrosis (may persist unchanged for >15 years)10%mortality (cor pulmonale / CNS / lung fibrosis / liver cirrhosis)25%relapse (in 50% detected by CXR) @Bone (6-20%):√ phalangeal sclerosis of hands√ lytic cystic lesions with lacelike trabecular pattern @Muscle (25%): myopathy@Eyes (5-25%): uveitis, photophobia, blurred vision, glaucoma (rare)@ Myocardium (6-25%): ventricular arrhythmia, heart block, cardiomyopathy, congestive failure, angina, ventricular aneurysm @ CNS (9%): hypothalamus, basal granulomatous meningitis, facial nerve palsy @ Salivary gland (4%): bilateral parotid enlargement @Peripheral lymph node involvement (30%) @ Skin disease (10-30%) ● erythema nodosum = multiple bilateral tender erythematous nodules mostly on anterior aspect of lower extremities ● lupus pernio = indurated bluish-purple elevations mainly on nose + digits ● skin plaques / scars @Thoracic disease (90%)-adenopathy alone (43%)-adenopathy + parenchymal disease (41%)-parenchymal disease alone (16%)*Associated with:* tuberculosis in up to 13%√ intrathoracic lymphadenopathy (>85%)Location: (a)"1-2-3 sign" = Garland triad = bilateral hilar + right paratracheal groups (75-95%)(b)isolated unilateral hilar enlargement (1-8%)(c)mediastinal nodes are regularly enlarged on CT*Prognosis:*adenopathy commonly decreases as parenchymal disease gets worse; subsequent parenchymal disease in 32%; adenopathy does not develop subsequent to parenchymal disease√ eggshell calcification of lymph nodes (in 3% after 5 years, in 20% after 10 years)√ parenchymal disease (60%); without adenopathy in 16-20%◊Parenchymal granulomas are invariably present on open lung biopsy!Site:predominantly mid-zone involvement√ reticulonodular pattern (46%)√ acinar pattern (20%) = ill-defined 6-7 mm nodules / coalescent opacities√ "alveolar / acinar sarcoidosis" (2-10%) = multiple large nodules >10 mm ± air bronchogram (= coalescence of numerous interstitial granulomas)√ progressive fibrosis with upper lobe retraction + bullae (20%)√ end-stage lung (11%)√ airway disease√ tracheal stenosis√ bronchial stenosis (extrinsic compression by large lymph nodes / endobronchial granulomas)√ bronchiectasis (scarring / fibrosis)HRCT:√ irregular septal thickening√ perilymphatic nodules (= small nodules along bronchoarterial bundles and veins, in subpleural + interlobular septal lymphatics representing epitheloid cell granulomas)√ traction bronchiectasis (TYPICAL)√ ground-glass opacity (in alveolitis)√ honeycombing√ irregular / nodular bronchial wall thickening *Atypical manifestations (25%):*√ pleural effusion (2%) = exudate with predominance of lymphocytes, effusion clears in 2-3 months√ focal pleural thickening√ solitary / multiple pulmonary nodules√ cavitation of nodules (0.6%)√ isolated hilar / mediastinal nodal enlargement√ bronchostenosis (2%) with lobar / segmental atelectasis√ pulmonary arterial hypertension (periarterial granulomatosis without extensive pulmonary fibrosis)*Cx:*√ pneumothorax secondary to chronic lung fibrosis (rare)√ cardiomegaly from cor pulmonale (rare)√ aspergilloma formation in apical bulla (in >50% of stage IV disease)*Diagnostic criteria:* (1)compatible clinical + radiologic picture(2)noncaseous epitheloid granulomas on bronchial / transbronchial biopsy (diagnostic results in 60-95% and 80-95% respectively)(3)negative results of special stains / cultures for other entities ASSESSMENT OF ACTIVITY (1)ACE titer (= angiotensin I converting enzyme)(2)Bronchoalveolar lavage: 20-50% lymphocytes with number of T-suppressor lymphocytes 4-20 times above normal(3)Gallium scan√ uptake in lymph nodes + lung parenchyma + salivary glands (correlates with alveolitis + disease activity); monitor of therapeutic response (indicator of macrophage activity) @ Abdominal disease ● strikingly elevated ACE levels in 91%@Liver (pathologic involvement in 24-79%):√ hepatomegaly (18-29%)√ nodular lesions in liver and spleen in 5-15% (= coalescent granulomata) occurring within 5 years of diagnosis√ abdominal adenopathy (mean size of 2.6 cm)@Spleen (pathologic involvement in 24-59%):√ splenomegaly (20-33%)√ scattered nodular lesions (18%)@Lymphadenopathy (31%) ● frequently associated with thoracic adenopathy√ mean lymph node size of 2.6 cm@Stomach (60 cases):√ polypoid / nodular mass ± ulcer√ loss of antral compliance*DDx:*lymphoma @Genitourinary disease (0.2-5%)@Kidney:√ renal calculi@Scrotum (0.5%)√ hypoechoic lesions of epididymal + testicular sarcoidosis

Notes:

SEPTIC PULMONARY EMBOLI
=lodgement of an infected thrombus in a pulmonary artery*Organism:*S. aureus, Streptococcus*Predisposed:*IV drug abusers, alcoholism, immunodeficiency, CHD, dermal infection (cellulitis, carbuncles)*Source:* (a)infected venous catheter / pacemaker wires, arteriovenous shunts for hemodialysis, drug abuse producing septic thrombophlebitis (eg, heroin addicts), pelvic thrombophlebitis, peritonsillar abscess, osteomyelitis(b)tricuspid valve endocarditis (most common cause in IV drug abusers)*Age:*majority <40 years ● sepsis, cough, dyspnea, chest pain ● shaking chills, high fever, severe sinus tachycardiaLocation:predilection for lung bases√ multiple nondescript pulmonary infiltrates (initially)√ migratory infiltrates (old ones heal, new ones appear)√ cavitation (frequent), usually thin-walled√ pleural effusion (rare)CT (more sensitive than CXR): √ multiple peripheral parenchymal nodules ± cavitation / air bronchogram (83%)√ wedge-shaped subpleural lesion with apex of lesion directed toward pulmonary hilum (50%)√ feeding vessel sign = pulmonary artery leading to nodule (67%)√ cavitation (50%), esp. in staphylococcal emboli√ air bronchogram within pulmonary nodule (28%)*Cx:*empyema (39%)

Notes:

SIDEROSIS
=inert iron oxide / metallic iron deposits*Path:*iron phagocytosed by macrophages in alveoli / respiratory bronchioles, elimination from lung by lymphatic circulation*Occupational exposure:* arc welding, cutting / burning of steel, foundry workers, grinders, fettlers, polishers (jewelry industry) √ reticulonodular pattern (may disappear after exposure discontinued)√ small round opacities (indistinguishable from silica / coal)√ NO secondary fibrosis + NO hilar adenopathy (unless mixed dust inhalation as in siderosilicosis)

Notes:

SILICOSIS
=inhalation of silicon dioxide; most prevalent silicosis of progressive nature after termination of exposure; similar to CWP (because of silica component in CWP)*Substance:*Crystalline silica (quartz); one of the most widespread elements on earth*Occupational exposure:*tunneling, mining, quarrying, sandblasting, ceramic industry*Path:*small particles engulfed by macrophages; liberation of silica results in cell death; 2-3 mm nodules with layers of laminated connective tissue around smaller vessels*Cx:*predisposes to tuberculosis

Acute Silicoproteinosis Chronic Simple Silicosis Complicated Silicosis Silicotuberculosis Caplan Syndrome

Notes:

Acute Silicoproteinosis =acute silicosis of sandblasters; exposure may be <1 year*Associated with:* increased risk to develop autoimmune disease√ diffuse airspace disease

Notes:

Chronic Simple Silicosis At least 10-20 years of dust exposure before appearance of roentgenographic abnormality√ small 1-10 mm rounded opacities, beginning in upper + middle lung zones√ may calcify centrally in 5-10% (rather typical for silicosis)√ hilar lymphadenopathy, may calcify in 5% ("eggshell pattern")√ ± reticulonodular patternHRCT: √ nodules of 3-10 mm in size√ thickened intra- and interlobular lines√ subpleural curvilinear lines (peribronchiolar fibrosis)√ ground-glass pattern = mild thickening of alveolar wall + interlobular septa (fibrosis / edema)√ parenchymal fibrous bands√ pleura-based nodular irregularities√ traction bronchiectasis√ honeycombing

Notes:

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Complicated Silicosis √ conglomerate masses of nonsegmental distribution in middle + upper lung zones √ progressive massive fibrosis = sausage-shaped masses with ill-defined margins (in advanced stages) √ compensatory emphysema in unaffected portion √ slow change over years √ may cavitate

Notes:

Silicotuberculosis Doubtful synergistic relationship between silicosis + tuberculosis √ little change over years with intermittently positive sputa

Notes:

Caplan Syndrome More common in coal workers pneumoconiosis

Notes:

SJÖGREN SYNDROME
=MYOEPITHELIAL SIALADENITIS=probable autoimmune multisystem disorder (= collagen-vascular disease) characterized by dryness of mucous membranes affecting(1)salivary + lacrimal glands(2)mucosa + submucosa of pharynx(3)tracheobronchial tree(4)reticuloendothelial system(5)joints A.PRIMARY SJÖGREN SYNDROME=autoimmune exocrinopathy(a)recurrent parotitis in children(b)SICCA SYNDROME = Mikulicz disease=xerophthalmia + xerostomiaB.SECONDARY SJÖGREN SYNDROME*Associated with:* (a)connective tissue diseases1.Rheumatoid arthritis (55%)2.Systemic lupus erythematosus (2%)3.Progressive systemic sclerosis (0.5%)4.Psoriatic arthritis, primary biliary cirrhosis (0.5%)(b)lymphoproliferative disorders1.Lymphocytic interstitial pneumonitis (LIP)2.Pseudolymphoma (25%)3.Lymphoma (5%; 44 x increased risk): mostly B-cell lymphoma4.Waldenström macroglobulinemia*Age:*35-70 (mean 57) years; M:F = 1:9*Path:*benign lymphoepithelioma = lymphoid infiltrates in lacrimal + salivary glands, mucous glands of conjunctivae, nasal cavity, pharynx, larynx, trachea, bronchi ● xerophthalmia = dryness of eyes= keratoconjunctivitis sicca = desiccation of cornea + conjunctiva ● xerostomia = atrophy of salivary + parotid glands leading to diminished saliva production and dryness of mouth + lips ● xerorhinia = dryness of nose ● decreased sweating ● decreased vaginal secretions ● swelling of parotid gland: usually unilateral, recurrent ● rheumatoid factor (positive in up to 95%) ● ANA (positive in up to 80%) CXR: √ reticulonodular pattern (3-33-52%)√ patchy consolidation√ inspissated mucus:√ atelectasis√ recurrent pneumonia√ bilateral lower lobe bronchiectasis√ acute focal / lipoid pneumonia (secondary to oils taken to combat dry mouth)√ ± pleural effusionSialogram: √ nonobstructive punctate / globular / cavitary sialectasia (ducts + acini destroyed by lymphocytic infiltrates / infection) US of parotid gland: √ enlarged gland√ multiple scattered cysts bilaterally (= cystic dilatation of intraparotid ducts + glands)√ increased vascularity on color DopplerMR of parotid gland: √ inhomogeneous honeycomblike internal pattern (= areas of low intensity between nodular parenchyma of high signal intensity) on T2WI / Gd-enhanced T1WI*Cx:*Lymphoma (occurs in significant number of patients)

Notes:

STAPHYLOCOCCAL PNEUMONIA
Most common cause of bronchopneumonia (a)common nosocomial infection (patients on antibiotic drugs most susceptible)(b)accounts for 5% of community-acquired pneumonias (esp. in infants + elderly) ◊secondary invader to influenza (commonest cause of death during influenza epidemics)*Organism:*Staphylococcus aureus, Gram-positive, appears in clusters, coagulase-producing√ rapid spread through lungs√ empyema (esp. in children)√ pneumothorax, pyopneumothorax√ abscess formation√ bronchopleural fistulaA.in CHILDREN:√ rapidly developing lobar / multilobar consolidation√ pleural effusion (90%)√ pneumatocele (40-60%)B.in ADULTS:√ patchy often confluent bronchopneumonia of segmental distribution, bilateral in >60%√ segmental collapse (air bronchograms absent)√ late development of thick-walled lung abscess (25-75%)√ pleural effusion / empyema (50%) (DDx from other pneumonias) *Cx:*meningitis, metastatic abscess to brain / kidneys, acute endocarditis

Notes:

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STREPTOCOCCAL PNEUMONIA
*Incidence:*1-5% of bacterial pneumonias (rarely seen); most common in winter months*Organism:*Group A ß-hemolytic streptococcus = Streptococcus pyogenes, Gram-positive cocci appearing in chains*Predisposed:*newborns, following infection with measles*Associated with:*delayed onset of diaphragmatic hernia (in newborns)

- rarely follows tonsillitis + pharyngitis√ patchy bronchopneumonia√ lower lobe predominance (similar to staphylococcus)√ empyema*Cx:*(1)Residual pleural thickening (15%)(2)Bronchiectasis(3)Lung abscess(4)Glomerulonephritis

Notes:

SWYER-JAMES SYNDROME
=MACLEOD SYNDROME= UNILATERAL LOBAR EMPHYSEMA=IDIOPATHIC UNILATERAL HYPERLUCENT LUNG*Etiology:*acute viral bronchiolitis in infancy / early childhood (adenovirus, RSV) preventing normal development of lung*Path:*variant of postinfectious constrictive bronchiolitis with acute obliterative bronchiolitis, bronchiectasis, distal airspace destruction (developing in 7-30 months) ● asymptomatic ● cough, dyspnea on exertion, hemoptysis ● history of recurrent lower respiratory tract infections during childhoodLocation:one / both lungs (usually entire lung, occasionally lobar / subsegmental)√ unilateral hyperlucency of affected lung√ small hemithorax with decreased / normal volume (collateral air drift)√ air trapping during expiration*DDx:*no air trapping with proximal interruption of pulmonary artery (no hilum), hypogenetic lung syndrome, pulmonary embolus√ mild cylindrical bronchiectasis with paucity of bronchial subdivisions (cutoff at 4th-5th generation = "pruned tree" bronchogram)√ small ipsilateral hilum (diminuted hilar vessels + attenuated arteries)√ diminutive pulmonary vasculatureHRCT: √ bilateral areas of decreased attenuation√ areas of normal lung attenuation within hypoattenuating lung√ air trapping within hypoattenuating lung√ bronchiectasisAngio: √ "pruned tree" appearanceNUC: √ decreased perfusion√ decreased ventilation + delayed washout

Notes:

SYSTEMIC LUPUS ERYTHEMATOSUS
=most prevalent of the potentially grave connective tissue diseases characterized by involvement of vascular system, skin, serous + synovial membranes (type III immune complex phenomenon)*Incidence:*1:2,000; Blacks:Caucasians = 3:1; increased risk in relatives*Age:*women of child-bear age; M:F = 1:10 ● clinically heterogeneous due to different types of serum antibodies ● antinuclear DNA antibodies (87%) ● hypergammaglobulinemia (77%) ● LE cells (= antigen-antibody complexes engulfed by PMNs) in 78% ● chronic false-positive Wassermann test for syphilis (24%) ● Sjögren syndrome (frequent) ● anemia (78%) ● leukopenia (66%) ● thrombocytopenia (19%)@Skin changes (81%) ● "butterfly rash" (= facial erythema), discoid lupus erythematosus, alopecia, photosensitivity ● Raynaud phenomenon (15%)@Thoracic involvement (30-70%)◊affects respiratory system more commonly than any other connective tissue disease ● dyspnea, pleuritic chest pain (35%) ● respiratory dysfunction (>50%): single-breath diffusing capacity for carbon monoxide most sensitive indicator(a)Pulmonary changes*Cause:*chronic antibody damage to alveolar-capillary membrane√ lupus pneumonitis (acute form) = poorly defined patchy areas of increased density peripherally at lung bases (alveolar pattern) secondary to infection / uremia in 10%√ interstitial reticulations in lower lung fields (chronic form) in 3%√ fleeting platelike atelectasis in both bases (? infarction due to vasculitis)√ cavitating nodules (vasculitis)√ elevated sluggish diaphragms (progressive volume loss due to diaphragmatic dysfunction)√ hilar + mediastinal lymphadenopathy (extremely rare)(b)Pleural changes (most common manifestation)√ recurrent bilateral pleural effusions (70%) from pleuritis√ pleural thickening(c)Cardiovascular changes√ pericardial effusion (from pericarditis)√ cardiomegaly (primary lupus cardiomyopathy)@Joints ● arthralgia (95%)√ nonerosive arthritis of hands (characteristic) without deformity@Kidney*Incidence:*kidneys involved in 100% with renal disease developing in 50%*Histo:*focal membranous glomerulonephritis ● renal failure (fibrinoid thickening of basement membrane)√ aneurysms in interlobular + arcuate arteries (similar to polyarteritis nodosa)√ normal / decreased renal sizeUS: √ increased parenchymal echogenicity*Cx:*(1)Nephrotic syndrome (common)(2)Renal vein thrombosis (rare)*Prognosis:*end-stage renal disease is common cause of death@GI tract (in up to 50%) ● buccal erosions / ulcerations ● GI tract bleeding√ motility disorder of lower esophagus (similar to scleroderma)√ esophagitis ± ulcers√ gastritis√ mesenteric ischemia: colitis, pseudoobstruction, ileus, thumbprinting, luminal narrowing√ nodularity of folds√ pneumatosis intestinalis, perforation√ painful ascites√ hepatomegaly, hepatitis, cirrhosis√ splenomegaly*Prognosis:*60-90% 10-year survival; death from renal failure / sepsis / CNS involvement / myocardial infarction **Drug-induced Lupus Erythematosus** (DIL) ● temporary phenomenon*Agents:*procainamide, hydralazine, isoniazid, phenytoin account for 90%√ pulmonary + pleural disease more common than in SLE

Notes:

TALCOSIS
=prolonged inhalation of magnesium silicate dust containing amphibole fibers (tremolite and anthophyllite) and silicaTalcosis resembles: (1)Asbestosis (indistinguishable)√ massive and bizarre pleural plaques√ may encase lung with calcification(2)Silicosis√ small rounded + large opacities√ fibrogenic process (NO regression after removal of patient from exposure)

Notes:

TERATOID TUMOR OF MEDIASTINUM
=MEDIASTINAL GERM CELL TUMOR [= TERATOMA]The anterior mediastinum is the most common extragonadal site of primary germ cell tumors (1-3% of all germ cell tumors)!*Pathogenesis:*"misplaced" multipotential primitive germ cells during migration from yolk endoderm to gonad*Incidence:* -adults:15% of anterior mediastinal tumors-children: 24% of anterior mediastinal tumors16-28% of all mediastinal cysts!Occurs in same frequency as the usually larger thymoma!1/3 of primary neoplasms in this area are in children *Classes:*(1)Mature teratoma (solid)(2)Cystic teratoma (dermoid cyst)(3)Immature teratoma(4)Malignant teratoma (teratocarcinoma)(5)Mixed teratoma Location:mediastinum is 3rd most common site for teratoid lesions (after gonadal + sacrococcygeal location); 5% of all teratomas occur in mediastinum, mostly anterosuperiorly (in only 1% posteriorly)√ often inseparable from thymus gland A.BENIGN TERATOID TUMOR (75-86%)=MATURE TERATOMA=most common histologic type1.Epidermoid (52%) = ectodermal derivatives2.Dermoid (27%) = ecto- + mesodermal derivatives3.TeratomaTeratoma (21%) = ecto- + meso- + endodermal derivatives*Path:*spherical lobulated well-encapsulated tumor; typically multi- / unilocular cystic cavities with clear / yellow / brown liquid*Histo:* (a)ectoderm: skin, sebaceous material, hair, cysts lined by squamous epithelium(b)mesoderm: bone, cartilage, muscle(c)endoderm: GI + respiratory tissue, mucus glandsTumor capsule commonly has remnants of thymic tissue!Cyst formation is typical (usually lined by mucus-secreting tall epithelial cells)!*Age:*young adults / children; M = F ● asymptomatic (in up to 53%) ● cough, dyspnea, chest pain, pulmonary infection, respiratory distress (due to compression by large tumor)Location: (a)anterior superior mediastinum near thymus / within thymic parenchyma(b)posterior mediastinum (rare = 3-8%)√ rounded mass bulging into right / left hemithorax sharply demarcated against adjacent lung √ variations in density (may all be present):√ fat-fluid level (rare but SPECIFIC)√ water density√ homogeneous soft-tissue density (indistinguishable from lymphoma / thymoma)√ curvilinear peripheral / central calcification (20-43%, 4 x more common in benign lesions) in tumor wall / substance, ossification in mature bone√ visualization of tooth (PATHOGNOMONIC)√ often inseparable from thymic gland√ enhancement of rim / tissue septa*Prognosis:*approx. 100% 5-year survival rate*Rx:*complete surgical excision B.MALIGNANT TERATOID TUMOR (14-20%)*Histo:*similar to mature teratoma but with primitive / immature tissue elements; commonly neural tissue arranged in rosettes / primitive tubulesTeratocarcinoma / malignant teratoma = identical to teratoma with components of seminoma, endodermal sinus tumor, embryonal carcinoma, choriocarcinoma, sarcoma, carcinoma 1.**Seminoma** = germinoma = dysgerminomaTM2nd most common mediastinal germ cell tumor!Most common primary malignant germ cell tumor of mediastinum!*Incidence:*2-6% of all mediastinal tumors; 5-13% of all malignant mediastinal tumors*Age:*3rd-4th decade; M >> F; white*Histo:*uniform polyhedral / round cells arranged in sheets or forming small lobules separated by fibrous septa; varying amounts of mature lymphocytes*Path:*large unencapsulated well-circumscribed mass ● asymptomatic (20-30%) ● chest pain / pressure, shortness of breath, weight loss, hoarseness, dysphagia, fever ● SVC obstruction (10%) ● elevated serum levels of HCG (7-18%) ● elevated serum levels of LDH (80%) correlate with tumor burden + rate of tumor growthMetastases:to regional lymph nodes, lung, bone, liver√ large bulky well-marginated lobulated mass√ usually NO calcification√ homogeneous soft-tissue density with slight enhancement*Prognosis:*75-100% 5-year survival rate; death from distant metastases*Rx:*surgery + radiation therapy (very radiosensitive) ± cisplatin2.**Nonseminomatous malignant germ cell tumor**
(a)embryonic tissue(1)Embryonal carcinoma(b)extraembryonic tissue(1)Yolk sac = endodermal sinus tumor(2)Choriocarcinoma (least frequent)(c)combination = mixed germ cell tumor*Path:*large unencapsulated heterogeneous soft-tissue mass with tendency for invasion of adjacent structures*Age:*during 2nd to 4th decade M:F = 9:1; in children M = F*Associated with:*Klinefelter syndrome (in 20%), hematologic malignancy ● chest pain, dyspnea, cough, weight loss, fever, SVC syndrome (90-100%) ● elevated serum level of a-fetoprotein (80%) with endodermal sinus tumor / embryonal carcinoma ● elevated serum level of LDH (60%) ● elevated serum level of HCG (30%) [DDx: lung cancer; hepatocellular carcinoma; adenocarcinoma of pancreas, colon, stomach]Metastases to:lung, liver√ large tumor of heterogeneous texture with central hemorrhage / necrosis√ well circumscribed / with irregular margins√ enhancement of tumor periphery√ lobulation suggests malignancy√ invasion of mediastinal structures (SVC obstruction is ominous)√ pleural / pericardial effusion (from local invasion)Absence of primary testicular tumor / retroperitoneal mass proves primary!*Rx:*cisplatin-based chemotherapy + tumor resection*Prognosis:* 50% long-term survivors *Cx:* (1)Hemorrhage(2)Pneumothorax (from bronchial obstruction with air trapping + alveolar rupture)(3)Respiratory distress (rapid increase in size from fluid production) with compression of trachea / SVC (SVC syndrome)(4)Fistula formation to aorta, SVC, esophagus(5)Rupture into bronchus (expectoration of oily substance / trichoptysis in 5-14%, lipoid pneumonia)(6)Rupture into pericardium (pericardial effusion), pleural cavity (pleural effusion)*DDx:*thymoma

Notes:

THORACIC PARAGANGLIOMA
=CHEMODECTOMA=rare neural tumor arising from paraganglionic tissue *Age:*3rd-5th decade; M:F = 1:1Path:extremely vascular well-marginated / irregular mass that may adhere to / envelop / invade adjacent mediastinal structures (bronchus, spinal canal)*Histo:*anastomosing cords of granule-storing chief cells arranged in a trabecular pattern; identical appearance for benign and malignant tumors*May be associated with:* syn- / metachronous adrenal / extrathoracic paragangliomas; multiple endocrine neoplasia type 2; bronchial carcinoid tumor ● asymptomatic ● dyspnea, cough, chest pain, hemoptysis, neurologic deficits, SVC syndrome (if tumor large) ● signs of excessive catecholamine production: hypertension, headache, tachycardia, palpitations, tremorLocation:base of heart + great vessels (adjacent to pericardium / heart, within interatrial septum / left atrial wall); paravertebral sulciCT: √ sharply marginated 5-7 cm middle / posterior mediastinal mass√ hypodense areas due to extensive cystic degeneration / hemorrhage√ exuberant enhancementMR: √ heterogeneous intermediate signal intensity with areas of signal void from flowing blood on T1WI√ high signal intensity on T2WINUC (I-123 / I-131 metaiodobenzylguanidine): √ useful for localization as relatively specificAngio (may precipitate cardiovascular crisis): √ marked hypervascularity, multiple feeding vessels√ homogeneous capillary blush*Rx:*surgical excision with preoperative administration of a- or b-blockers (hypertensive crisis, tachycardia, dysrhythmia during manipulation)

Notes:

THYMIC CYST
*Incidence:*1-2% of mediastinal masses*Etiology:* (1)Congenital cyst (persistent tubular remnants of 3rd pharyngeal pouch = thymopharyngeal duct, develops during 5th-8th week of gestation)(2)Acquired reactive multilocular cysts = cystic transformation of duct epithelial structures induced by an inflammatory process: eg, HIV(3)Neoplastic cyst (cystic teratoma, cystic degeneration within a thymoma), S/P radiation therapy for Hodgkin disease*Associated with:* (1)Hodgkin disease (? thymic involvement / treatment-induced cystic degeneration)(2)myasthenia gravis (rare) ● commonly asymptomatic ● symptomatic when hemorrhage occursLocation:anterior mediastinum / lateral neck√ unilocular cyst with thin walls containing clear fluid / multilocular cyst with thick walls containing turbid fluid or gelatinous material√ may show partial wall calcification (rare)√ low-density fluid (0-10 HU), may be higher depending on cyst contentsUS: √ typically anechoic*DDx:*Benign thymoma, teratoma, dermoid cyst, Hodgkin disease, non-Hodgkin lymphoma, pleural fibroma

Notes:

THYMIC HYPERPLASIA
Most common anterior mediastinal mass in pediatric age group through puberty *Age:*particularly in young individual*Histo:*numerous active lymphoid germinal centers*Etiology:* 1.Hyperthyroidism (most common), Graves disease, treatment of primary hypothyroidism, idiopathic thyromegaly2.Rebound hyperplasia in children recovering from severe illness (eg, from burns), after treatment for Cushing disorder, after chemotherapy√ thymus may regrow more than 50% (transient overgrowth and reducible with steroids)3.Myasthenia gravis (65%)4.Acromegaly5.Addison disease√ normal thymus visible in 50% of neonates 0-2 years of age√ notch sign = indentation at junction of thymus + heart√ sail sign = triangular density extending from superior mediastinum√ wave sign = rippled border due to indentation from ribs√ shape changes with respiration + position

Notes:

THYMOLIPOMA
*Incidence:*2-9% of thymic tumors*Age:*3-60 years (mean age of 22 years); M:F = 1:1*Path:*lobulated pliable encapsulated tumor capable of growing to large size (in 68% >500 g, in 20% >2,000 g, the largest >16 kg)*Histo:*benign adult adipose tissue interspersed with areas of normal / hyperplastic / atrophic thymus tissue (thymic tissue <33% of tumor mass) ● chest pain, dyspnea, cough (in 50%)√ large lesions slump inferiorly from anterior mediastinum toward diaphragm√ may drape around heart enlarging cardiac silhouette on frontal view√ apparent elevation of diaphragm on lateral view√ NO compression / invasion of adjacent structures*DDx:*mediastinal lipoma (most common of intrathoracic fatty tumors), liposarcoma

Notes:

THYMOMA

◊Most common primary neoplasm of anterior superior mediastinum*Age:*majority >40 years; 70% occur in 5th-6th decade; less frequent in young adults, rare in children;M:F = 1:1*Associated with:*parathymic syndromes (40%) such as ● **Myasthenia gravis**:=autoimmune disorder characterized by antibodies against acetylcholine receptors of the postjunctional muscle membrane ● progressive weakness, fatigue ● fatigability of skeletal muscles innervated by cranial nerves, eg, ptosis, diplopia, dysphagia, dysarthria, drooling, difficulty with chewing ● elevated serum level of anti-acetylcholine receptor antibodies◊10-15-25% of patients with myasthenia gravis have a thymoma (in 65% due to thymic hyperplasia)◊7-30-54% of patients with thymoma have myasthenia gravis; removal of thymic tumor often results in symptomatic improvement; myasthenia gravis may develop after surgical thymoma excision*Rx:*edrophonium chloride ● Pure red cell aplasia = aregenerative anemia=almost total absence of marrow erythroblasts + blood reticulocytes resulting in severe normochromic normocytic anemia◊50% of patients with red cell aplasia have thymoma◊5% of patients with thymoma develop red cell aplasia ● Acquired hypogammaglobulinemia◊10% of patients with hypogammaglobulinemia have thymoma◊6% of patients with thymoma have hypogammaglobulinemia ● Paraneoplastic syndromes occur with thymic carcinoid (10%): eg, Cushing syndrome (ACTH production) ● chest pain, dyspnea, cough (33%)*Path:* round / ovoid slow-growing primary epithelial neoplasm with smooth / lobulated surface divided into lobules by fibrous septa; areas of hemorrhage + necrosis may form cysts (a)encapsulated = thick fibrous capsule ± calcifications(b)locally invasive = microscopic foci outside capsule(c)metastasizing = benign cytologic appearance with pleural + pulmonary parenchymal seeding(d)thymic carcinoma*Histo:* (a)biphasic thymoma (most common)= epithelial + lymphoid elements in equal amounts (b)predominantly lymphocytic thymoma= >2/3 of cells are lymphocytic (c)predominantly epithelial thymoma= >2/3 of cells are epithelial ◊Prognosis unrelated to cell type! ● asymptomatic (50% discovered incidentally) ● signs of mediastinal compression (25-30%):cough, dyspnea, chest pain, respiratory infection, hoarseness (recurrent laryngeal n.), dysphagia ● signs of tumor invasion (rare): SVC syndromeLocation:any anterior mediastinal location between thoracic inlet and cardiophrenic angle; rare in neck, other mediastinal compartments, lung parenchyma, or tracheobronchial treeSize:1-10 cm (up to 34 cm)

Noninvasive = Benign Thymoma Invasive [Malignant] Thymoma

Notes:

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Noninvasive = Benign Thymoma *Age peak:*5th-6th decade, almost all are >25 years of age√ oval / round lobulated sharply demarcated asymmetric homogeneous mass of soft-tissue density (equal to muscle), usually on one side of the midline√ abnormally wide mediastinum√ displacement of heart + great vessels posteriorlyCT: √ homogeneous soft-tissue mass with smooth / lobulated border partially / completely outlined by fat√ homogeneous enhancement√ areas of decreased attenuation (fibrosis, cysts, hemorrhage, necrosis)√ amorphous, flocculent central / curvilinear peripheral calcification (5-25%)MRI: √ isointense to skeletal muscle on T1WI√ increased signal intensity (approaching that of fat) on T2WI√ fluid characteristics of cysts with high water content

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Notes:

Invasive [Malignant] Thymoma◊Malignancy defined according to extent of invasion into adjacent mediastinal fat + fascia!*Frequency:*in 30-35% of thymomasStageI:intact capsuleStageII:pericapsular growth into mediastinal fatStageIII:invasion of surrounding organs such as lung, pericardium, SVC, aortaStageIVa:dissemination within thoracic cavity (metastases to pleura + lung in 6%)StageIVb:distant metastases (liver, bone, lymph nodes, kidneys, brain)√ heterogeneous attenuation√ spread by contiguity along pleural reflections, extension along aorta reaching posterior mediastinum / crus of diaphragm / retroperitoneum (transdiaphragmatic tumor extension)√ irregular interface with lung√ unilateral diffuse nodular pleural thickening / pleural masses encasing lung circumferentially√ vascular encroachment√ pleural effusion UNCOMMON*DDx:*malignant mesothelioma, lymphoma, thymic carcinoma / malignant germ cell tumor (older male, no diffuse pleural seeding), peripheral lung carcinoma (no dominant mediastinal mass), metastatic disease (not unilateral) *Rx:*radical excision ± adjuvant radiation therapy*Prognosis:*5-year survival of 93% for stage I, 86% for stage II, 70% for stage III, 50% for stage IV; 2-12% rate of recurrence for resected encapsulated thymomas

Notes:

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TORSION OF LUNG

*Incidence:*rare (<30 cases)*Cause:*compression of lower thorax, tear on inferior pulmonary ligament, completeness of fissures*Associated with:* surgery (lobectomy), trauma, diaphragmatic hernia, pneumonia, pneumothorax, bronchus-obstructing tumor *Histo:*± hemorrhagic infarction + excessive air trapping√ collapsed / consolidated lobe in unusual position√ hilar displacement of atelectatic-appearing lobe in an inappropriate direction√ alteration in normal course of pulmonary vasculature√ rapid opacification of an ipsilateral lobe after trauma / thoracic surgery (DDx: pleural effusion)√ change in position of opacified lobe on sequential radiographs√ bronchial cutoff / distortion√ lobar air trapping

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TRACHEOBRONCHOMEGALY
=MOUNIER-KUHN SYNDROME = primary atrophy / dysplasia of supporting structures of trachea + major bronchi with abrupt transition to normal bronchi at 4th-5th division*Incidence:*0.5-1.5%*Age:*discovered in 3rd-5th decade ● cough with copious sputum ● shortness of breath on exertion ● long history of recurrent pneumonias*May be associated with:*Ehlers-Danlos syndrome√ marked dilatation of trachea (>29 mm), right (>20 mm) + left (>15 mm) mainstem bronchi√ sacculated outline / diverticulosis of trachea on lateral CXR (= protrusion of mucous membrane between rings of trachea)√ may have emphysema, bullae in perihilar region

Notes:

TRACHEOBRONCHOPATHIA OSTEOCHONDROPLASTICA
=rare benign disease characterized by cartilaginous / osseous nodules projecting from submucosa into tracheobronchial lumen*Cause:*unknown; may be due to chronic inflammation, degenerative process, irritation by oxygen / chemical, metabolic disturbance, amyloidosis, tuberculosis, syphilis, heredity (high prevalence in Finland)*Pathogenetic theories:* (1)Ecchondrosis / exostosis of cartilage rings(2)Cartilaginous / osseous metaplasia of internal elastic fibrous membrane of trachea*Histo:*adipose tissue + calcified areas with foci of bone marrow; thinned normal overlying mucosa with inflammation + hemorrhage*Age:*in 50% >50 years (11-72 years); M:F = 3:1 ● usually asymptomatic (incidentally diagnosed) ● dyspnea, productive cough, hoarseness, hemoptysis, fever, recurrent pneumoniaLocation:distal 2/3 of trachea, larynx, lobar / segmental bronchi, entire length of trachea;spares posterior membrane of tracheaCXR: √ scalloped / linear opacities surrounding + narrowing the trachea (best on lateral view)CT: √ deformed thickened narrowed tracheal wall√ irregularly spaced 1-3 mm calcific submucosal nodules of trachea + bronchi (similar to plaques)*Dx:*bronchoscopy*DDx:*relapsing polychondritis, tracheobronchial amyloidosis, sarcoidosis, papillomatosis, tracheobronchomalacia

Notes:

TRANSIENT TACHYPNEA OF THE NEWBORN
=NEONATAL WET LUNG DISEASE = TRANSIENT RESPIRATORY DISTRESS OF THE NEWBORN= RETAINED FETAL LUNG FLUID*Incidence:*6%; most common cause of respiratory distress in newborn*Cause:*cesarean section, precipitous delivery, breech delivery, prematurity, maternal diabetes*Pathophysiology:* delayed resorption of fetal lung fluid (normal clearance occurs through capillaries (40%), lymphatics (30%), thoracic compression during vaginal delivery (30%) *Onset:* within 6 hours of life; peak at day 1 of age ● increasing respiratory rates during first 2-6 hours of life ● intercostal + sternal retraction ● normal blood gases during hyperoxygenation√ linear opacities + perivascular haze + thickened fissures + interlobular septal thickening (interstitial edema)√ mild hyperaeration√ mild cardiomegaly√ small amount of pleural fluid*Prognosis:* resolving within 1-4 days (retrospective diagnosis) *DDx:*(1) normal during first several hours of life(2) diffuse pneumonitis / sepsis (3) mild meconium aspiration syndrome (4) "drowned newborn syndrome" = clear amniotic fluid aspiration (5) alveolar phase of RDS (6) pulmonary venous congestion (7) pulmonary hemorrhage (8) hyperviscosity syndrome = thick blood (9) immature lung syndrome

Notes:

TRAUMATIC LUNG CYST

*Age:*children + young adults are particularly prone√ thin-walled air-filled cavity (50%) ± air-fluid level preceded by homogeneous well-circumscribed mass (hematoma) √ oval / spherical lesion of 2-14 cm in diameter√ single / multiple lesions; uni- or multilocular√ usually subpleural under point of maximal injury√ persistent up to 4 months + progressive decrease in size (apparent within 6 weeks)

Notes:

TUBERCULOSIS

*Prevalence:*10 million people worldwide, active TB develops in 5-10% of those exposed*Organism:*Mycobacterium = acid-fast aerobic rods staining red with carbol-fuchsin; M. tuberculosis (95%), atypical types increasing: M. avium-intracellulare, M. kansasii, M. fortuitum *Susceptible:*infants, pubertal adolescents, elderly, alcoholics, Blacks, diabetics, silicosis, measles, AIDS, sarcoidosis (in up to 13%)*Pathologic phases:* (a)exudative reaction (initial reaction, present for 1 month)(b)caseous necrosis (after 2-10 weeks with onset of hypersensitivity)(c)hyalinization = invasion of fibroblasts (granuloma formation in 1-3 weeks)(d)calcification / ossification(e)chronic destructive form in 10% (<1 year of age, adolescents, young adults)Spread:regional lymph nodes, hematogenous dissemination, pleura, pericardium, upper lumbar vertebrae*Mortality:*1:100,000 ● Positive PPD tuberculin test: 3 weeks after infection ● Negative PPD test:1.Overwhelming tuberculous infection (miliary TB)2.Sarcoidosis3.Corticosteroid therapy4.Pregnancy5.Infection with atypical Mycobacterium ENDOBRONCHIAL (ACINAR) TUBERCULOSIS *Path:*ulceration of bronchial mucosa followed by fibrosis leads to(a)bronchial stenosis (lobar consolidation)(b)bronchiectasis(c)acinar nodules reflecting airway spreadHRCT: √ airspace nodules√ "tree-in-bud" appearance = nodular opacities along centrilobular artery + bronchiole√ bronchiectasis TUBERCULOMA =manifestation of primary / postprimary TB√ round / oval smooth sharply defined mass√ 0.5-4 cm in diameter remaining stable for a long time√ lobulated mass (25%) √ satellite lesions (80%)√ may calcify CAVITARY TUBERCULOSIS =hallmark of reactivation tuberculosis= semisolid caseous material is expelled into bronchial tree after lysis √ moderately thick-walled cavity with smooth inner surface*Cx:* (1)dissemination to other bronchial segments√ multiple small acinar shadows remote from massive consolidation(2)colonization with Aspergillus√ aspergilloma

Primary Pulmonary Tuberculosis Postprimary Pulmonary Tuberculosis Miliary Pulmonary Tuberculosis

Notes:

Primary Pulmonary Tuberculosis *Mode of infection:*inhalation of infected airborne droplets*Age:*usually in childhood, becoming commoner in adults ● asymptomatic (91%) ● symptomatic (5-10%)Location:lower lobes, middle lobe, anterior segment of upper lobes√ in children: massive hilar (60%) / paratracheal (40%) / subcarinal lymphadenopathy (in children), in 80% on right side;in adults: mediastinal lymphadenopathy in 5-35-48% √ one / more areas of homogeneous ill-defined airspace consolidation of 1-7 cm in diameter in 25-50-78% (requires several weeks for complete clearing with antituberculous therapy)√ absent response to antibiotic Rx for "pneumonia"√ atelectasis (8-18%), esp. in right lung (anterior segment of upper lobe / medial segment of middle lobe) secondary to(a)endobronchial tuberculosis(b)bronchial / tracheal compression by enlarged lymph nodes (68%)√ pleural effusion (10% in childhood, 23-38% in adulthood) most commonly 3-7 months after initial exposure (from subpleural foci rupturing into pleural space)√ pneumonic reaction (mid or lower lung zones) with segmental / lobar consolidation√ calcified lung lesion (17%) / parenchymal scar <5 mm = **Ghon lesion**
√ calcified lymph node (36%) in hilus / mediastinum√ **Ranke complex** = Ghon lesion + calcified lymph node (22%)√ **Simon focus** = healed site of primary infection in lung apexCT: √ tuberculous adenopathy may demonstrate necrotic center with low attenuation after enhancement *Outcome of primary infection:* 1.Immunity prevents multiplication of organism (containment of initial infection by delayed hypersensitivity response + granuloma formation in 1-3 weeks)2.Progressive primary TB (inadequate immune mechanism with local progression) in 10%, most common in older children / teenagers3.Miliary tuberculosis (uncontrolled massive hematogenous dissemination overwhelming host defense system)4.Postprimary TB = reactivation TB (reactivation of dormant organisms after asymptomatic years)*Prognosis:*3.6% mortality rate*Cx:*(1)Bronchopleural fistula + empyema(2)Fibrosing mediastinitis

Notes:

Postprimary Pulmonary Tuberculosis =REACTIVATION TB = RECRUDESCENT TB= infection under the influence of acquired hypersensitivity and immunity secondary to longevity of bacillus + impairment of cellular immunity *Incidence:*1% per year in persons with normal immunity, up to 10% in persons with deficient T-cell immunity*Etiology:* (a)reactivation of focus acquired in childhood(b)initial infection in individual vaccinated with BCG(c)continuation of initial infection= progressive primary tuberculosis (rare)*Path:*foci of caseous necrosis with surrounding edema, hemorrhage, mononuclear cell infiltration; formation of tubercles = accumulation of epithelioid cells + Langhans giant cells; bronchial perforation leads to intrabronchial dissemination (19-21%)*Age:*predominantly in adulthoodSite:85% in apical + posterior segments of upper lobe, 10% in superior segment of lower lobe, 5% in mixed locations (anterior + contiguous segments of upper lobe); R > L (DDx: histoplasmosis tends to affect anterior segment) ***Local*** Exudative Tuberculosis √ chronic patchy / confluent ill-defined areas of acinar consolidation (87-91%)√ thin-walled cavitation with smooth inner surface (present in more advanced disease)√ cavity under tension (air influx + obstructed efflux)√ air-fluid level is strong evidence for superimposed bacterial / fungal infection√ accentuated drainage markings toward ipsilateral hilum√ acinar nodular pattern (20%) due to bronchogenic spread√ pleural effusion (18%)CT: √ micronodules in centrilobular location (62%)= solid caseation material in / surrounding the terminal / respiratory bronchioles √ interlobular septal thickening (34-54%)= increase in lymphatic flow as inflammatory response / impaired lymphatic drainage due to hilar lymphadenopathy ***Local***
Fibroproductive Tuberculosis √ sharply circumscribed irregular + angular masslike fibrotic lesion (in up to 7%)√ thick-walled irregular cavitation (HALLMARK) secondary to expulsion of caseous necrosis into airways, esp. in apical / posterior segments of upper lobes (rare in children, in up to 45-51% in adults)√ reticular pulmonary scars√ cicatrization atelectasis = volume loss in affected lobe√ bronchiectasis in apical / posterior segments of upper lobes√ pleural thickening√ apical cap = pleural rind = thickening of layer of extrapleural fat (3-25 mm) + pleural thickening (1-3 mm)√ tuberculous lymphadenitis√ calcified hilar / mediastinal nodes√ Rasmussen aneurysm = aneurysm of terminal branches of pulmonary artery within wall of TB cavity secondary inflammatory necrosis of the vessel wall (4% at autopsies of cavitary TB)√ central cavity near hilum√ enlargement of central solid component of cavity√ opacification of pseudoaneurysm on CT / angio

Notes:

Miliary Pulmonary Tuberculosis =massive hematogenous dissemination of organisms any time after primary infection*Cause:* (1)severe immunodepression during postprimary state of infection(2)impaired defenses during primary infection= PROGRESSIVE PRIMARY TB *Incidence:*2-3.5% of TB infections√ chronic focus often not identifiable√ radiographically recognizable after 6 weeks post hematogenous dissemination√ generalized granulomatous interstitial small foci of pinpoint to 2-3 mm size √ rapid complete clearing with appropriate therapyHRCT (earlier detection than CXR): √ diffusely scattered discrete 1-2 mm nodules*Cx:*dissemination via bloodstream affecting lymph nodes, liver, spleen, skeleton, kidneys, adrenals, prostate, seminal vesicles, epididymis, fallopian tubes, endometrium, meninges

Notes:

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UNILATERAL PULMONARY AGENESIS
=one-sided lack of primitive mesenchyme*Associated with:* anomalies in 60% (higher if right lung involved): PDA, anomalies of great vessels, tetralogy of Fallot (left-sided pulmonary agenesis), bronchogenic cyst, congenital diaphragmatic hernia, bone anomalies ● may be asymptomatic ● respiratory infections√ complete opacity of hemithorax√ ipsilateral absence of pulmonary artery + vein√ absent ipsilateral mainstem bronchus√ symmetrical chest cage with approximation of ribs√ overdistension of contralateral lung√ ipsilateral shift of mediastinum + diaphragm

Notes:

VARICELLA-ZOSTER PNEUMONIA

*Incidence:*14% overall; 50% in hospitalized adults*Age:*>19 years (90%); 3rd-5th decade (75%); contrasts with low incidence of varicella in this age group ● vesicular rash√ patchy diffuse airspace consolidation√ tendency for coalescence near hila + lung bases√ widespread nodules (30%) representing scarring√ tiny 2-3 mm calcifications widespread throughout both lungs (2%)*Cx:*unilateral diaphragmatic paralysis*Prognosis:*11% mortality rate

Notes:

VIRAL PNEUMONIA
*Organism:*Rhinovirus (43%), respiratory syncytial virus (12%), Mycoplasma (10%), Parainfluenza virus, adenovirus, Influenza-virus*Path:*necrosis of ciliated epithelial cells, goblet cells, bronchial mucous glands with frequent involvement of peribronchial tissues + interlobular septa*Age:*most common cause of pneumonia in children under 5 years of ageDistribution:usually bilateral√ hyperaeration + air trapping√ "dirty chest" = peribronchial cuffing + opacification√ perihilar linear densities (bronchial wall thickening)√ interstitial pattern√ airspace pattern (from hemorrhagic edema) in 50%√ pleural effusion (20%)√ hilar adenopathy (3%)√ striking absence of pneumatoceles, lung abscess, pneumothorax√ radiographic resolution lags 2-3 weeks behind clinical*Cx:*bronchiectasis; unilateral hyperlucent lung◊Atypical measles pneumonia does NOT show the typical radiographic findings of viral pneumonias!

Notes:

WEGENER GRANULOMATOSIS

=probable autoimmune disease characterized by systemic necrotizing granulomatous process with destructive angiitis*Path:*peribronchial necrotizing granulomas + vasculitis not intimately related to arteries*Mean age of onset:*40 years (range of all ages);M:F = 2:1CLASSIC TRIAD: (1)respiratory tract granulomatous inflammation(2)systemic small-vessel vasculitis(3)necrotizing glomerulonephritis @Upper respiratory tract (100% involvement)(similar to midline granuloma) (a)nasal cavity: ● epistaxis from nasal mucosal ulceration ● necrosis of nasal septum ● saddle nose deformity√ progressive destruction of nasal cartilage + bone (DDx: relapsing polychondritis)√ granulomatous masses filling nasal cavities(b)sinuses (maxillary antra most frequently): ● sinus pain, purulent sinus drainage, rhinorrhea√ thickening of mucous membranes of paranasal sinuses@Pulmonary disease ● stridor (from tracheal inflammation + sclerosis) ● intractable cough, occasionally with hemoptysis√ patchy alveolar infiltrates (with acute airspace pneumonia / pulmonary hemorrhage)√ widely distributed multiple irregular masses / nodules of varying sizes (up to 9 cm), especially in lower lung fields√ thick-walled cavities with irregular shaggy inner lining (25-50%)√ pleural effusion in 25%√ lymphadenopathy exceedingly rare*Cx:*(1)dangerous airway stenosis (15% of adults, 50% of children)(2)massive life-threatening pulmonary hemorrhage@Renal diseasefocal glomerulonephritis in 20% at presentation, as disease progresses in 83% *Histo:*focal necrosis, crescent formation, paucity/ absence of immunoglobulin deposits@ Other organ involvement: (a)Joints (56%): migratory polyarthropathy(b)Skin + muscle (44%): inflammatory nodular skin lesions, cutaneous purpura(c)Eyes + middle ear (29%): ocular inflammation, proptosis, otitis media(d)Heart + pericardium (28%): myocardial infarction (vasculitis)(e)CNS (22%): central / peripheral neuritis(a)involvement of abdominal viscera*Cx:*(1) Hypertension (2) Uremia (3) Facial nerve paralysis*Dx:*lung / renal biopsy*Prognosis:*death within 2 years from renal failure (83%) / respiratory failure*Rx:*corticosteroids, cytotoxic drugs (cyclophosphamide), renal transplantation

Limited Wegener Granulomatosis Midline Granuloma

Notes:

Limited Wegener Granulomatosis =Wegener granulomatosis WITHOUT renal involvement

Notes:

Midline Granuloma =mutilating granulomatous + neoplastic lesions limited to nose + paranasal sinuses with very poor prognosis; considered a variant of Wegener granulomatosis WITHOUT the typical granulomatous + cellular components

Notes:

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WILLIAMS-CAMPBELL SYNDROME

=congenital bronchial cartilage deficiency in the 4th to 6th bronchial generation either diffuse or restricted to focal areaHRCT: √ cystic bronchiectasis distal to 3rd bronchial generation√ emphysematous lung distal to bronchiectasis√ inspiratory ballooning + expiratory collapse of dilated segments

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WILSON-MIKITY SYNDROME
=PULMONARY DYSMATURITY=similarity to bronchopulmonary dysplasia in patients breathing room air; rarely encountered anymore*Predisposed:*premature infants <1500 g who are initially well ● gradual onset of respiratory distress between 10-14 days√ hyperinflation√ reticular pattern radiating from both hila√ small bubbly lucencies throughout both lungs (identical to bronchopulmonary dysplasia)*Prognosis:* resolution over 12 months

Notes:

ZYGOMYCOSIS
=PHYCOMYCOSIS=group of severe opportunistic sinonasal + pulmonary disease caused by a variety of Phycomycetes (soil fungi)*Organism:*ubiquitous Mucor (most common), Rhizopus, Absidia with broad nonseptated hyphae of irregular branching pattern *At risk:*immunoincompetent host with1.lymphoproliferative malignancies and leukemia2.acidotic diabetes mellitus3.immunosuppression through steroids, antibiotics immunosuppressive drugs (rare)*Entry:*inhalation / aspiration from sinonasal colonization*Path:*angioinvasive behavior similar to aspergillosis A.RHINOCEREBRAL FORM=involvement of paranasal sinuses (frontal sinus usually spared) with extension into:(a)orbit = orbital cellulitis(b)base of skull = meningoencephalitis + cerebritisB.PULMONARY FORM√ segmental homogeneous consolidation√ cavitary consolidation + air-crescent sign√ nodules (from arterial thrombi + infarction)√ rapidly progressive (often fatal) pneumonia*Dx:*culture of fungus from biopsy specimen / demonstration within pathologic material*DDx:*aspergillosis

Notes:

Asymmetric Breast Density A.OBVIOUS PATHOLOGIC LESION1.Stellate lesion2.Circular / ovoid lesion3.Calcifications4.CombinationB.PARENCHYMA1.Nodular densities + fat(a)normal TDLU(b)adenosis2.Linear densities + fat3.Fibrosis + fat4.Accessory breastC.FIBROSIS1.Postinflammatory fibrosis2.Posttraumatic fibrosis3.Desmoplastic reaction

Notes:

Diffuse Increase In Breast Density √ generalized increased density√ skin thickening√ reticular pattern in subcutisA.CANCER1."Inflammatory" breast cancer (angiolymphatic spread) ● rapid development of diffuse swelling, induration, skin redness + peau d'orange edema over 1/3 of breast surface*Dx:*skin biopsy2.Diffuse primary noninflammatory breast cancer3.Diffuse metastatic breast cancer4.Lymphoma / leukemiadue to obstructive lymphedema of breast B.INFECTIOUS MASTITISusually in lactating breast C.RADIATION(a)diffuse exudative edema within weeks after beginning of radiation therapy(b)indurational fibrosis months after radiation therapyD.EDEMA1.Lymphatic obstruction: extensive axillary / intrathoracic lymphadenopathy, mediastinal / anterior chest wall tumor, axillary surgery2.Generalized body edema: congestive heart failure (breast edema may be unilateral if patient in lateral decubitus position), hypoalbuminemia (renal disease, liver cirrhosis), fluid overloadE.HEMORRHAGE1.Posttraumatic2.Anticoagulation therapy3.Bleeding diathesisF.ACCIDENTAL INFUSION OF FLUIDinto subcutaneous tissue

Notes:

Mammographic Evaluation Of Breast Masses True mass or pseudomass? A.SIZE-well-defined nodules <1.0 cm are of low risk for cancer-"most likely benign" nodules approaching 1 cm should be considered for ultrasound / aspiration / biopsy B.SHAPE-increase in probability of malignancy: round < oval < lobulated < irregular < architectural distortion C.MARGIN (most important factor)-well-circumscribed mass with sharp abrupt transition from surrounding tissue is almost always benign-"halo" sign of apparent lucency = optical illusion of Mach effect + true radiolucent halo is almost always (92%) benign but not pathognomonic for benignity-microlobulated margin worrisome for cancer-obscured margin may represent infiltrative cancer-irregular ill-defined margin has a high probability of malignancy-spiculated margin due to (a) fibrous projections extending from main cancer mass (b) previous surgery (c) sclerosing duct hyperplasia (radial scar) D.LOCATION-intramammary lymph node typically in upper outer quadrant (in 5% of all mammograms)-large hamartoma + abscess common in retro- / periareolar location-sebaceous cyst in subcutaneous tissue E.X-RAY ATTENUATION = DENSITY-fat-containing lesions are never malignant-high-density mass suspicious for carcinoma (higher density than equal volume of fibroglandular tissue due to fibrosis)F.NUMBER-multiplicity of identical lesions decreases risk G.INTERVAL CHANGE-enlarging mass needs biopsy H.PATIENT RISK FACTORS-increasing age increases risk for malignancy-positive family history-history of previous abnormal breast biopsy-history of extramammary malignancy

Notes:

Well-circumscribed Breast Mass ◊Well-defined nonpalpable lesions have a 4% risk of malignancy!A.BENIGN1.Cyst (45%)2.Fibroadenoma3.Sclerosing adenoma4.Intraductal papilloma (intracystic / solid)5.Galactocele6.Sebaceous cystB.MALIGNANT1.Medullary carcinoma2.Mucinous carcinoma3.Intracystic papillary carcinoma4.Invasive ductal cancer not otherwise specified (rare)5.Pathologic intramammary lymph node6.Metastases to breast: melanoma, lymphoma / leukemia, lung cancer, hypernephroma ***Well-circumscribed De Novo Mass In Woman >40 Years Of Age*** 1.Cyst2.Papilloma3.Carcinoma4.Sarcoma (rare)5.Fibroadenoma (exceedingly rare)6.Metastasis (extremely rare)

Notes:

Fat-containing Breast Lesion ◊Fat contained within a lesion proves benignity!1.Lipoma2.Galactocele=fluid with high lipid content (last phase) ● during / shortly after lactation3.Traumatic lipid cyst = fat necrosis = oil cyst ● site of prior surgery / trauma4.Focal collection of normal breast fat ***Mixed Fat- And Water-density Lesion*** 1.Intramammary lymph node2.Galactocele3.Hamartoma = lipofibroadenoma = fibroadenolipoma4.Small superficial hematoma

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Notes:

Breast Lesion With Halo Sign A.HIGH-DENSITY LESION=vessels + parenchymal elements not seen in superimposed lesion1.Cyst2.Sebaceous cyst3.WartB.LOW-DENSITY LESION=vessels + parenchyma seen superimposed on lesion1.Fibroadenoma2.Galactocele3.Cystosarcoma phylloides

Notes:

Stellate / Spiculated Breast Lesion =mass / architectural distortion characterized by thin lines radiating from its marginsRisk of malignancy: -75% for nonpalpable spiculated masses-32% for nonpalpable irregular masses A.PSEUDOSTELLATE STRUCTURE=SUMMATION SHADOWScaused by fortuitous superimposition of normal fibrous + glandular structures; unveiled by rolled views, spot compression views ± microfocus magnification technique B."BLACK STAR"√ groups of fine fibrous strands bunched together√ circular / oval lucencies within center√ change in appearance on different views1.Radial scar = sclerosing duct hyperplasia2.Posttraumatic fat necrosisC."WHITE STAR"√ individual straight dense spicules√ central solid tumor mass√ little change in different views1.Invasive ductal carcinoma = scirrhous carcinoma=desmoplastic reaction + secondary retraction of surrounding structures ● clinical dimensions larger than mammographic size√ distinct central tumor mass with irregular margins√ length of spicules increase with tumor size√ localized skin thickening / retraction when spiculae extend to skin√ commonly associated with malignant-type calcifications2.Postoperative scar ● correlation with history + site of biopsy√ scar diminishes in size + density over time3.Postoperative hematoma ● clinical information√ short-term mammographic follow-up confirms complete resolution4.Breast abscess ● clinical information√ high-density lesion with flamelike contour5.Hyalinized fibroadenoma with fibrosis√ changing pattern with different projections√ may be accompanied by typical coarse calcifications of fibroadenomas6.Granular cell myoblastoma7.Fibromatosis8.Extra-abdominal desmoid *mnemonic:*"STARFASH"**S**ummation shadow **T**umor (malignant) **A**bscess **R**adial scar **F**ibroadenoma (hyalinized), **F**at necrosis **A**denosis (sclerosing) **S**car (postoperative) **H**ematoma (postoperative)

Notes:

Tumor-mimicking Lesions 1."Phantom breast tumor" = simulated mass(a)asymmetric density√ scalloped concave breast contour√ interspersed fatty elements(b)summation shadow = chance overlap of glandular breast structures√ failure to visualize "tumor" on more than one view2.Silicone injections3.Skin lesions(a)Dermal nevus√ sharp halo / fissured appearance(b)Skin calcifications√ lucent center (clue)√ superficial location (tangential views)(c)Sebaceous / epithelial inclusion cyst(d)Neurofibromatosis(e)Biopsy scar4.Lymphedema5.Lymph nodes*Frequency:*5.4% for intramammary nodesLocation:axilla, subcutaneous tissue of axillary tail, lateral portion of pectoralis muscle, intramammary (typically in upper outer quadrant)√ ovoid / bean-shaped mass(es) with fatty notch representing hilum√ central zone of radiolucency (fatty replacement of center) surrounded by "crescent" rim of cortex√ usually <1.5 cm (up to 4 cm) in size√ well-circumscribed with slightly lobulated margin6.Hemangioma

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Notes:

Solid Breast Lesion By Ultrasound ***Malignant Sonographic Characteristics*** √ spiculation = alternating straight lines radiating perpendicularly from surface of nodule(a)hypoechoic relative to echogenic fibrous tissue(b)hyperechoic relative to surrounding fat√ taller-than wide lesion = AP dimension greater than craniocaudal / transverse dimension√ angular margin = contour of junction between hypo- or isoechoic solid nodule and surrounding tissue at acute / obtuse / 90° angles√ acoustic shadowing behind all / part of nodule(= fibroelastic host response to scirrhous cancer)√ central part of solid lesion very hypoechoic with respect to fat√ punctate echogenic calcifications within hypoechoic mass (acoustic shadowing commonly not present)√ radial extension / branch pattern (= intraductal component of breast cancer)√ microlobulation = many small lobulations at surface of solid nodule (according to data from A.T. Stavros) CharacteristicSens.Specif.PPVRel. riskspiculation36.099.491.85.5taller than wide41.698.181.24.9angular margins83.292.067.54.0acoustic shadowing48.894.764.93.9branch pattern29.696.664.03.8markedly hypoechoic68.860.160.13.6calcifications27.296.359.63.6duct extension24.895.250.83.0microlobulation75.283.848.22.9◊ Approximately 5 malignant features are found per cancer. The combination of 5 findings increases the sensitivity to 98.4%! ***Benign Sonographic Characteristics*** √ absence of any malignant characteristics◊A single malignant feature prohibits classification of a nodule as benign!√ marked hyperechogenic well-circumscribed nodule compared to fat = normal stromal fibrous tissue (may represent a palpable pseudomass / fibrous ridge)√ smooth well-circumscribed ellipsoid shape√ 2-3 smooth well-circumscribed gentle lobulations√ thin echogenic capsule√ kidney-shaped lesion = intramammary lymph node◊If specific benign features are not found the lesion is classified as indeterminate! (according to data from A.T. Stavros) CharacteristicSens.Specif.NPVRel. riskhyperechoic100.07.4100.00.00≤3 lobulations99.219.499.20.05ellipsoid shape97.651.299.10.05thin echogenic capsule95.276.098.80.07

Notes:

BREAST CALCIFICATIONS

Indicative of focally active process; often requiring biopsy◊75-80% of biopsied clusters of calcifications represent a benign process◊10-30% of microcalcifications in asymptomatic patients are associated with cancers Composition:hydroxyapatite / tricalcium phosphate / calcium oxalate *Results of breast biopsies for microcalcification:* (without any other mammographic findings) (a)benign lesions (80%)1.Mastopathy without proliferation44%2.Mastopathy with proliferation28%3.Fibroadenoma4%4.Solitary papilloma2%5.Miscellaneous2%(b)malignant lesions (20%)1.Lobular carcinoma in situ10%in 8% no spatial relationship to LCIS2.Infiltrating carcinoma6%3.Ductal carcinoma in situ4%◊Positive biopsy rate of >35% is desirable goal! A.LOCATION(a)intramammary1.**Ductal microcalcifications**

√0.1-0.3 mm in size, irregular, sometimes mixed linear + punctate*Occurrence:*secretory disease, epithelial hyperplasia, atypical ductal hyperplasia, intraductal carcinoma2.**Lobular microcalcifications**

√smooth round, similar in size + density*Occurrence:* cystic hyperplasia, adenosis, sclerosing adenosis, atypical lobular hyperplasia, lobular carcinoma in situ, cancerization of lobules (= retrograde migration of ductal carcinoma to involve lobules), ductal carcinoma obstructing egress of lobular contents N.B.:lobular and ductal microcalcifications occur frequently in fibrocystic disease + breast cancer!(b)extramammary: arterial wall, duct wall, fibroadenoma, oil cyst, skin, etc.B.SIZE√malignant calcifications usually <0.5 mm; rarely >1.0 mmC.NUMBER√<4-5 calcifications per 1 cm^2 have a low probability for malignancyD.MORPHOLOGY(a)benign1.smooth round calcifications: formed in dilated acini of lobules2.solid / lucent-centered spheres: usually due to fat necrosis3.crescent-shaped calcifications that are concave on horizontal beam lateral projection=sedimented milk of calcium at bottom of cyst4.lucent-centered calcifications: around accumulated debris within ducts / in skin5.solid rod-shaped calcifications / lucent-centered tubular calcifications: formed within / around normal / ectatic ducts6.eggshell calcifications in rim of breast cysts7.calcifications with parallel track appearance= vascular calcifications(b)malignant=calcified cellular secretions / necrotic cancer cells within ducts√calcifications of-vermicular form-varying in size-linear / branching shapeE.DISTRIBUTION1.clustered heterogeneous calcifications: adenosis, peripheral duct papilloma, hyperplasia, cancer2.segmental calcifications within single duct network: suspect for multifocal cancer within lobe3.regional / diffusely scattered calcifications with random distribution throughout large volumes of breast: almost always benignF.TIME COURSEmalignant calcifications can remain stable for >5 years! G.DENSITY

Malignant Calcifications Benign Calcifications

Notes:

Malignant Calcifications 1.**Granular calcifications** = resembling fine grains of salt√ amorphous, dotlike / elongated, fragmented√ grouped very closely together√ irregular in form, size, and density2.**Casting calcifications** = fragmented cast of calcifications within ducts√ variable in size + length√ great variation in density within individual particles + among adjacent particles√ jagged irregular contour√ ± Y-shaped branching pattern√ clustered (>5 per focus within an area of 1 cm^2)

Notes:

Benign Calcifications 1.**Lobular calcifications =** arise within a spherical cavity of cystic hyperplasia, sclerosing adenosis, atypical lobular hyperplasia√ sharply outlined, homogeneous, solid, spherical "pearl-like"√ little variation in size√ numerous + scattered√ associated with considerable fibrosis(a)adenosis√ diffuse calcifications involving both breasts symmetrically(b)periductal fibrosis√ diffuse / grouped calcifications + irregular borders, simulating malignant process2.Sedimented milk of calcium*Frequency:*4%√ multiple, bilateral, scattered / occasionally clustered calcifications within microcysts√ smudge-like particles at bottom of cyst on vertical beam√ crescent-shaped on horizontal projection= "teacup-like"3.Plasma cell mastitis = periductal mastitis√ sharply marginated calcifications of uniform density = intraductal form√ sharply marginated hollow calcifications= periductal form4.Peripheral eggshell calcifications(a)with radiolucent lesion-liponecrosis micro- / macrocystica calcificans (= fatty acids precipitate as calcium soaps at capsular surface) as calcified fat necrosis / calcified hematoma√May mimic malignant calcifications!(b)with radiopaque lesion-degenerated fibroadenoma-macrocyst√ high uniform density in periphery√ usually subcutaneous√ no associated fibrosis5.Papilloma√ solitary raspberry configuration in size of duct√ central / retroareolar6.Degenerated fibroadenoma√ bizarre, coarse, sharply outlined, "popcornlike" very dense calcification within dense mass(= central myxoid degeneration)√ eggshell type calcification (= subcapsular myxoid degeneration)7.Arterial calcifications√ parallel lines of calcifications8.Dermal calcificationsSite:sebaceous glands√ hollow radiolucent center√ polygonal shape√ peripheral location (may project deep within breast even on 2 views at 90° angles)√ linear orientation when caught in tangent√ same size as skin poresProof:superficial marking technique9.Metastatic calcifications*Cause:*2° hyperparathyroidism (in up to 68%)

Notes:

Nipple Retraction 1.Positional2.Relative to inflammation / edema of periareolar tissue3.Congenital4.Acquired (carcinoma, ductal ectasia)

Notes:

Nipple Discharge ◊The most significant discharge comes from one breast + one orifice!◊The most common cause of bloody / serosanguinous discharge is intraductal papilloma!Type of discharge: A.Lactating breast: galactorrheaB.Nonlactating breast:(a)normal: white, yellowish, greenish-gray(b)abnormal:1.clear serous: cancer 2-7%, papilloma 35%, fibrocystic change 36%, ductectasia 11%2.bloody: cancer 6-16%, papilloma 61%, fibrocystic change 12%, ductectasia 2% ● exfoliative cytology not helpful (true positive in 11%)Site of origin: A.Lobules + terminal duct lobular unit:1.Galactorrhea2.Fibrocystic changesB.Larger lactiferous ducts (collecting duct, segmental duct, subsegmental duct)1.Solitary papilloma2.Papillary carcinoma3.Duct ectasiaGalactography: injection of 0.1-0.3 cm^3 of water-soluble contrast material through blunt 27-gauge pediatric sialography needle (0.4-0.6 mm outer diameter, tip bent 90°) *DDx of intraductal defects:* gas bubble, clot, inspissated secretions, solitary intraductal papilloma, epithelial hyperplastic lesion, duct carcinoma Galactographic filling defectssinglemultipleMultiple papilloma5.6%14.0%Cancer0.05%9.7%

Notes:

Secretory Disease 1.Retained lactiferous secretionsresult of incomplete / prolonged involution of lactiferous ducts√ branching pattern of fat density in dense breast (high lipid content)2.Prolonged inspissation of secretion + intraductal debris√ duct dilatation√ calcifications with linear orientation toward subareolar area a few mm long: rod-shaped / sausage-shaped / spherical with hollow center3.Galactocele4.Plasma cell mastitis

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Notes:

Skin Thickening Of Breast Normal skin thickness:0.8-3 mm; may exceed 3 mm in inframammary regionA.LOCLAIZED SKIN THICKENING1.Trauma (prior biopsy)2.Carcinoma3.Abscess4.Nonsuppurative mastitis5.Dermatologic conditionsB.GENERALIZED SKIN THICKENING◊Skin is thickened initially and to the greatest extent in the lower dependent portion of breast!√ overall increased density with coarse reticular pattern (= dilated lymph vessels + interstitial fluid triggering fibrosis)(a)Axillary lymphatic obstruction1.Primary breast cancer-advanced breast cancer-invasive comedocarcinoma in large area◊Primary breast cancer not necessarily seen due to small size / hidden location (axillary tail, behind nipple)!2.Primary malignant lymphatic disease (eg, lymphoma)(b)Intradermal + intramammary obstruction of lymph channels1.Lymphatic spread of breast cancer from contralateral side2.Inflammatory breast carcinoma = diffusely invasive ductal carcinoma(c)Mediastinal lymphatic blockage1.Sarcoidosis2.Hodgkin disease3.Advanced bronchial / esophageal carcinoma4.Actinomycosis(d)Advanced gynecologic malignanciesfrom thoracoepigastric collaterals1.Ovarian cancer2.Uterine cancer(e)Inflammation1.Acute mastitis2.Retromamillary abscess3.Fat necrosis4.Radiation therapy5.Reduction mammoplasty(f)Right heart failuremay be unilateral (R > L) / migrating with change in patient position (to avoid decubitus ulcer) (g)Nephrotic syndrome, anasarca1.Dialysis2.Renal transplant(h)Subcutaneous extravasation of pleural fluid following thoracentesis

Notes:

Axillary Lymphadenopathy =solid node >1.5 cm in size without fatty hilumA.MALIGNANT1.Metastasis from breast cancer in 26%2.Metastases from non-breast primary (melanoma, ovary) 3.Lymphoma / chronic lymphocytic leukemia (17%)B.BENIGN1.Nonspecific benign lymphadenopathy (29%)2.Sarcoidosis3.Collagen vascular disease: rheumatoid arthritis, systemic lupus erythematosus4.Psoriasis5.HIV-related adenopathy6.Reactive lymphadenopathy (breast infection / abscess / biopsy) *Radiographic features suspicious for malignancy:* √ size increase of >100% over baseline√ size >3.3 cm√ change in shape√ spiculation of margins√ intranodal microcalcifications (without history of gold therapy)√ loss of radiolucent center / hilar notch√ increase in density

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Notes:

Breast Imaging Reporting And Data System (BIRD) N=negativethere is nothing to comment on; breasts are symmetrical without masses, architectural disturbances / suspicious calcifications B=benign findingconfidently labeled, eg, calcified fibroadenoma, multiple secretory calcifications, fat-containing lesion such as oil cyst, lipoma, galactocele, mixed-density hamartoma, intramammary lymph node, implant P=probably benign finding - short interval follow-uphigh probability of benign with radiologists preference to establish its stability S=suspicious abnormality - consider biopsylesion without characteristic morphology of cancer but definite probability of being malignant M=highly suggestive of malignancybiopsy is mandatory

Notes:

Lexicon Descriptors For Reporting (ACR)
A.MASSsize shapecircular, oval, lobulated, irregularmarginscircumscribed, lobulated, obscured, indistinct, speculatedlocationbased on face of clock + depth in breastassociated findingsskin changes, calcifications, nipple retraction, trabecular thickeningattenuationrelative to an equal volume of breast tissue: high density, isodense, low density, fat densityB.CALCIFICATIONStypeskin, vascular, coarse, rodlike, eggshell, punctate, pleomorphic number size distributionclustered, linear, segmental, regional, scattered, multiple groupsassociated findingsskin changes, nipple retraction, architectural distortion, trabecular thickening

Notes:

Radiology Review Manual
fourth edition

Lobes 15 - 20 lobes disposed radially around nipple, each lobe has a main lactiferous duct of 2.0-4,5 mm converging at the nipple with an opening in the central portion of nipple Main duct:branches dichotomously eventually forming terminal ductal lobular units*Histo:*epithelial cells, myoepithelial cells surrounded by extralobular connective tissue with elastic fibers

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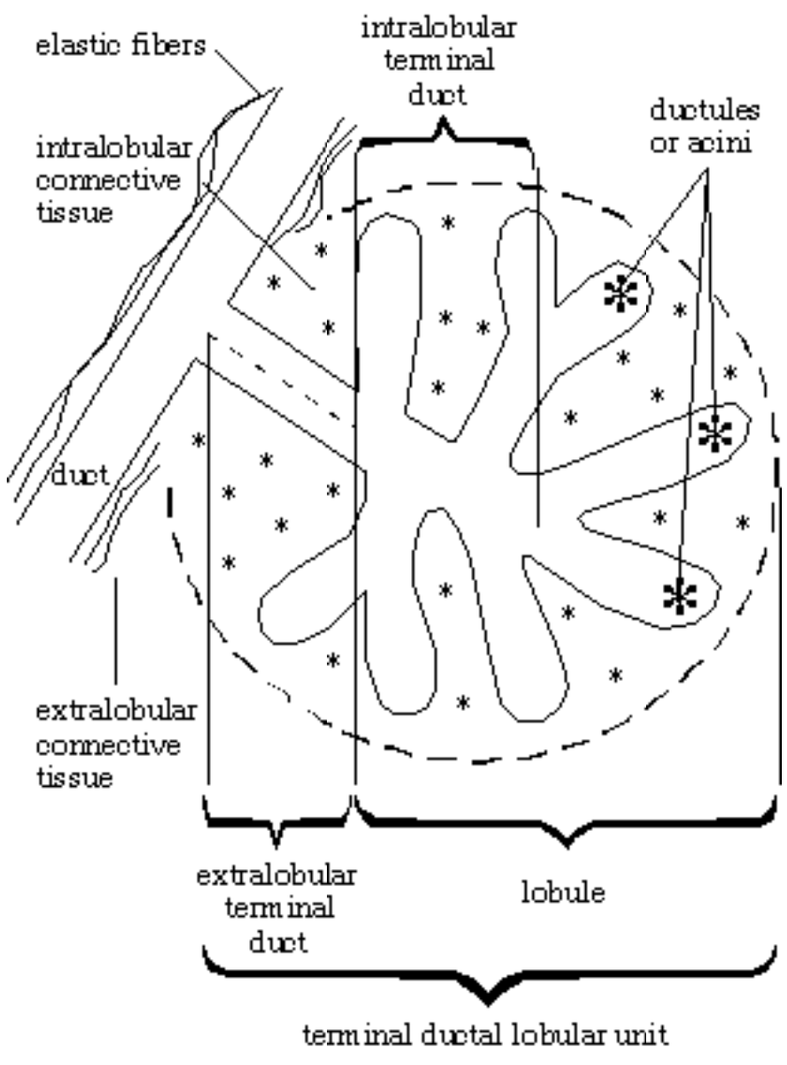
Notes:

Terminal Duct Lobular Unit (TDLU)
(1)Extralobular terminal duct*Histo:*lined by columnar cells + prominent coat of elastic fibers + outer layer of myoepithelium(2)Lobule(a)intralobular terminal duct*Histo:*lined by 2 layers of cuboidal cells + outer layer of myoepithelium(b)ductules / acini(c)intralobular connective tissueSize:1 - 8 mm (most 1 - 2 mm) in diameterChange: (a)reproductive age: cyclic proliferation (up to time of ovulation) + cyclic involution (during menstruation)(b)post menopause: regression with fatty replacementSignificance: TDLU is site of fibroadenoma, epithelial cyst, apocrine metaplasia, adenosis (= proliferation of ductules + lobules), epitheliosis (= proliferation of mammary epithelial cells within preexisting ducts + lobules), ductal + lobular carcinoma in situ, infiltrating ductal + lobular carcinoma



Notes:

Components Of Normal Breast Parenchyma 1.Nodular densities surrounded by fat(a)1 - 2 mm = normal lobules(b)3 - 9 mm = adenosis2.Linear densities=ducts and their branches + surrounding elastic tissue3.Structureless ground-glass density=stroma / fibrosis with concave contours

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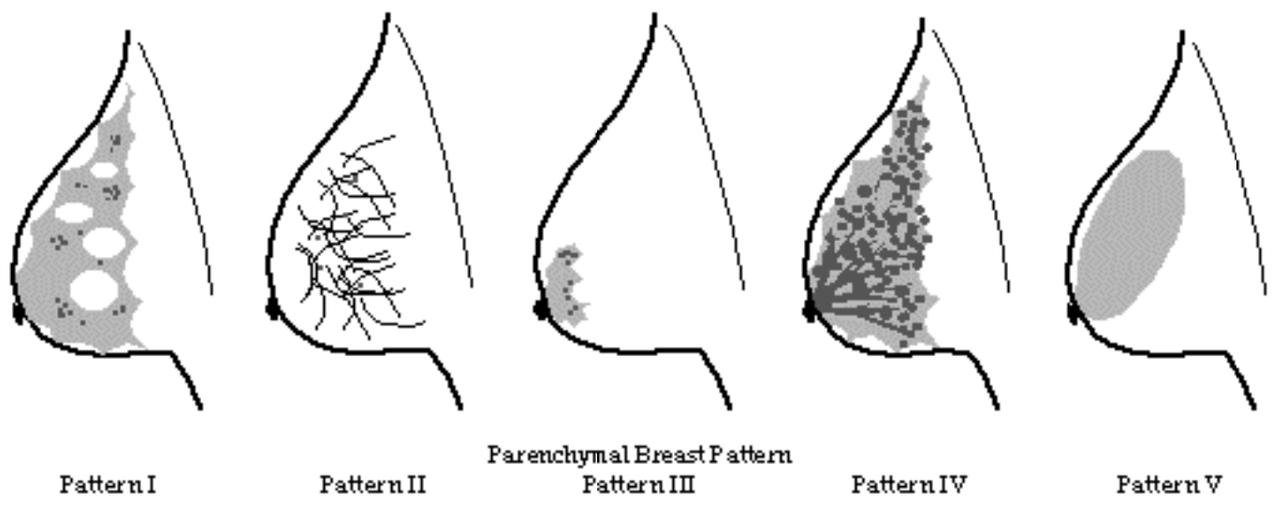
Notes:

Parenchymal Breast Pattern (László Tabár)



Pattern I named QDY = quasi dysplasia (for Wolfe classification) √ concave contour from Coopers ligaments√ evenly scattered 1 - 2 mm nodular densities (= normal terminal ductal lobular units)√ oval-shaped / circular lucent areas (= fatty replacement)**Pattern II** similar to N1 (Wolfe) √ total fatty replacement√ NO nodular densities**Pattern III** similar to P1 (Wolfe) √ normal parenchyma occupying <25% of breast volume in retroareolar location**Pattern IV** = adenosis pattern similar to P2 (Wolfe) *Cause:*hypertrophy + hyperplasia of acini within lobules*Histo:*small ovoid proliferating cells with rare mitoses√ scattered 3 - 7 mm nodular densities (= enlarged terminal ductal lobular units) = adenosis√ thick linear densities (= periductal elastic tissue proliferation with fibrosis) = fibroadenosis√ no change with increasing age (genetically determined)**Pattern V** similar to DY (Wolfe) √ uniformly dense parenchyma with smooth contour (= extensive fibrosis)

Notes:

MAMMOGRAPHIC FILM READING TECHNIQUE

1.Compare with earlier films2.Scan "forbidden" areas(a)"Milky Way" = 2 - 3 cm wide area parallel with the edge of the pectoral muscle on MLO projection(b)"No mans land" = fatty replaced area between posterior border of parenchyma + chest wall on CC projection(c)Medial half of breast on CC view3.Look for increased retroareolar density4.Look for parenchymal contour retraction5.Look for architectural distortion6.Look for straight lines superimposed on normal scalloped contour7.Compare left with right side8.Dont stop looking after one lesion is found

Notes:

MAMMOGRAPHIC TECHNIQUE

BEAM QUALITY Molybdenum target material with characteristic emission peaks of 17.9 + 19.5 keV (lower average energy than tungsten) FOCAL SPOT 0.1 - 0.4 mm (0.1 mm for magnification views) TUBE OUTPUT 80 - 100 mA EXPOSURE (a)without grid: 25 kV (optimum between contrast + penetration), exposure time of 1.0 seconds(b)with grid: 26 - 27 kV; exposure time of 2.3 seconds(c)microfocus magnification: 26 - 27 kV; 1.5 - 2.0 times magnification with 16 - 30 cm air gap(d)specimen radiography: 22 - 24 kVFILTER (a)beryllium window (absorbs less radiation than glass tube)(b)molybdenum filter (0.03 mm): allows more of lower energy radiation to reach breastREDUCTION OF SCATTER RADIATION (1)adequate compression (also improves contrast + decreases radiation dose)(2)beam collimation to <8 - 10 cm(3)air gap with microfocus magnification(greater spatial resolution, 2 - 3-fold increase in radiation exposure) (4)Moving gridgrid if compressed breast >5 cm / very dense breast (facilitates perception, 2 - 3-fold increase in radiation exposure) SCREEN-FILM COMBINATION (1)Intensifying screen phosphorsingle screen systems (2)Film-screen contact(3)Mammography film with minimal base fog, sufficient maximum density + contrastFILM PROCESSING (1)Processing time of 3 minutes (42 - 45 seconds in developing fluid) superior to 90-second processor for double-emulsion film (which creates underdevelopment + compensatory higher radiation exposure)(2)Developing temperature of 35° C (95° F)(3)Developing fluid replenishment rate:450 - 500 mL replenisher per square meter of film QUALITY CONTROL (1)Processor (daily)with sensito- / densitometric measurements (a)base fog <0.16 - 0.17(b)maximum density >3.50(c)contrast >1.9 - 2.0(2)X-ray unit (semiannually)(a)beam quality(b)phototimer *Average glandular dose:* <0.6 mGy per breast for nonmagnification film-screen mammogram (ACR accreditation requirement) Screen/film technique (molybdenum target; 0.03 mm molybdenum filter, 28 kVp): mean absorbed dose:0.05 rad for CC view0.06 rad for LAT view *Effective dose equivalent* H_E*:* screen-film mammography0.11 mSvxeroradiographic mammography0.78 mSvchest0.05 mSvskull0.15 mSvabdomen1.40 mSvlumbar spine2.20 mSv *Advantages of magnification mammography* 1.Sharpness effect = increased resolution2.Noise effect = noise reduced by a factor equal to the degree of magnification3.Air-gap effect = increased contrast by reduction in scattered radiation4.Visual effect = improved perception and analysis of small detail

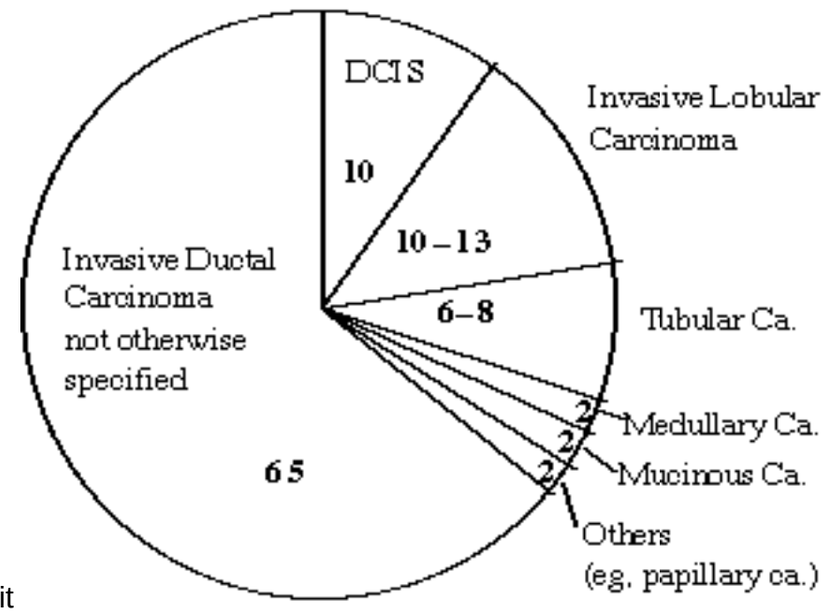
Factors Affecting Mammographic Image Quality

Notes:

Factors Affecting Mammographic Image Quality
A.RADIOGRAPHIC SHARPNESS=subjective impression of distinctness / perceptibility of structure boundary / edge 1.**Radiographic contrast**
=magnitude of optical density difference between structure of interest + surroundings influenced by (a)subject contrast=ratio of x-ray intensity transmitted through one part of the breast to that transmitted through a more absorbing adjacent part affected by-absorption differences in the breast (thickness, density, atomic number)-radiation quality (target material, kilovoltage, filtration)-scattered radiation (beam limitation, grid, compression)(b)receptor contrast=component of radiographic contrast that determines how the x-ray intensity pattern will be related to the optical density pattern in the mammogram affected by -film type-processing (chemicals, temperature, time, agitation)-photographic density-fog (storage, safelight, light leaks) 2.**Radiographic blurring**
=lateral spreading of a structural boundary(= distance over which the optical density between the structure and its surroundings changes) (a)motionreduced by compression + short exposure time (b)geometric blurringaffected by -focal spot: size, shape, intensity distribution-focus-object distance (= cone length)-object-image distance(c)receptor blurring=light diffusion (= spreading of the light emitted by the screen) affected by -phosphor thickness + particle size-light-absorbing dyes + pigments-screen-film contact B.RADIOGRAPHIC NOISE=unwanted fluctuation in optical density 1.**Radiographic mottle**
=optical density variations consist of(a)receptor graininess=optical density variation from random distribution of finite number of silver halide grains(b)quantum mottle (principal contributor to mottle)=variation in optical density from random spatial distribution of x-ray quanta absorbed in image receptoraffected by -film speed + contrast-screen absorption + conversion efficiency-light diffusion-radiation quality(c)structure mottle=optical density fluctuation from nonuniformity in the structure of the image receptor (eg, phosphor layer of intensifying screen)2.**Artifacts**
=unwanted optical density variations in the form of blemishes on the mammogram(a)improper film handling (static, crimp marks, fingerprints, scratches)(b)improper exposure (fog)(c)improper processing (streaks, spots, scratches)(d)dirt + stains

Notes:

BREAST CANCER



*Origin:*terminal ductal lobular unit (numbers are in percentage)

Distribution of Breast Cancers in Screening Population

A.NONINVASIVE BREAST CANCER (15%)=malignant transformation of epithelial cells lining mammary ducts + lobules confined within boundaries of basement membrane*Rx:*little data is available to provide insight into proper treatment 1.**Ductal carcinoma in situ** (DCIS)=intraductal carcinoma*Incidence:*10-25-40% in screening population; 70% of noninvasive carcinomas*Age:*most >55 years*Histo:*heterogeneous group of malignancies originating within extralobular terminal duct + without invasion of basement membraneSubgroups:comedocarcinoma, non-comedocarcinomas (solid, micropapillary, cribriform) ● may persist for years without palpable abnormality (in screening population) ● palpable mass / Paget disease of nipple / nipple discharge (in symptomatic patients)◊50% of DCIS are >5 cm in size◊Histologic size of DCIS is independent of histologic subgroup◊Almost all "comedo" type DCIS contain significant microcalcifications◊DCIS often involves the nipple + subareolar ducts*Spectrum of mammographic findings:*√ calcifications only (72%)√ soft-tissue abnormality + calcification (12%)√ soft-tissue abnormality only (10%)√ nonvisible (6%)*Prognosis:*20-50% develop invasive disease 5-10 years after initial diagnosis of DCIS*Rx:*(1)Simple / modified mastectomy: cure rate of almost 100%(2)Local excision alone: 25% rate of recurrence within 26 months in immediate vicinity of biopsy site(3)Local excision + radiotherapy: 2-17% rate of recurrence Treatment problems: 1.Occult invasion in 5-20% of patients2.Multifocality(= >1 focus in same quadrant of breast) 3.Multicentricity(= >1 focus in different quadrants of breast) in 14% of lesions <25 mm, in 100% of lesions >50 mm 4.Axillary metastases in 1-2%
(a)High nuclear grade DCIS ("**comedo type**")*Prevalence:*60% of all DCIS*Precursor:*none; one stage development*Path:*"comedo" = pluglike appearance of necrotic material that can be expressed from the cut surface*Characteristics:* }nuclear grade: large / intermediate nuclei, numerous mitoses, aneuploidy}growth pattern: predominantly solid cell proliferation; atypically micropapillary / cribriform}necrosis: extensive (HALLMARK)}calcifications (90%): dystrophic / amorphous within necrosis in center of dilated ductal system outlining most of the lobe in classic solid growth pattern ● estrogen- + progesterone-receptor negative ● overexpression of c-erb B-2 oncogene product and P53 suppressor gene mutation ● often symptomatic lesion with nipple discharge√ ductal system enlarged to 300-350 µ√ linear / branching pattern of calcifications scattered in a large part of lobe / whole lobe√ large solid high-density casting calcifications (fragmented, coalesced, irregular) in solid growth pattern√ "snake skin-like" / "birch tree flowerlike" dotted casting calcifications within necrosis of micropapillary / cribriform growth pattern√ palpable dominant mass without calcifications (very unusual)√ nipple discharge (rare)*Prognosis:*higher recurrence rate than noncomedo-group(b)Low nuclear grade DCIS ("**noncomedo** type")*Prevalence:*40% of all DCIS*Precursor lesion:* atypical ductal hyperplasia (ADH) with slight / moderate / severe atypia◊52-56% of ADH at core biopsy are associated with malignancy at excision!*Characteristics:* }nuclear grade: monomorphic small round nuclei, few / no mitoses}growth pattern: predominantly micropapillary / cribriform; atypically solid cell proliferation (often coexist)}necrosis: not present in classic micropapillary / cribriform growth pattern}calcifications (50%): laminated / psammoma-like due to active secretion by malignant cells into duct lumen√ fine granular "cotton ball" calcifications in micropapillary / cribriform growth pattern√ coarse granular "crushed stone" / "broken needle tip" / "arrowhead" calcifications in less common solid growth pattern◊Size of "noncomedo" DCIS often underestimated mammographically (? due to lower density of calcifications at periphery of lesion)!√ palpable dominant mass without calcifications (intracystic papillary carcinoma, multifocal papillary carcinoma in situ)√ nonpalpable asymmetric density with architectural distortion√ occasionally serous / bloody nipple discharge + ductal filling defects on galactography*Risk of recurrence:*2%*Prognosis:*30% eventually develop into invasive cancer*Dx:*surgical biopsy◊Core needle biopsy could result in diagnosis of only proliferative breast disease that is usually intermixed! 2.**Lobular carcinoma in situ** (LCIS)= arises in epithelium of blunt ducts of mammary lobules *Incidence:*0.8-3.6% in screening population; 3-6 % of all breast malignancies; 25% of noninvasive carcinomas; high incidence during reproductive age but decreasing with age*Age:*most 40-54 years (earlier than DCIS / invasive tumors) *Histo:*monomorphous small cell population filling + expanding ductules of the lobule◊Synchronous invasive cancer in 5%! ● not palpable√ mammographically occult√ may atypically present as a noncalcified mass (in 7%), calcifications + mass (in 10%), asymmetric opacity (2%)◊High frequency of multicentricity (70%) + bilaterality (30%)!*Dx:*incidental microscopic finding depending on accident of biopsy (performed for unrelated reasons + findings)*Prognosis:* 20-30% develop invasive ductal > lobular carcinoma within 20 years after initial diagnosis◊1% per year lifetime risk for invasive malignancy◊LCIS serves as a marker of increased risk for developing invasive carcinoma in either breast!*Rx:*recommendations range from observation (with follow-up examinations every 3-6 months + annual mammograms) to unilateral / bilateral simple mastectomy 3.**Intracystic papillary carcinoma in situ** (0.5-2%)=rare variant of noncomedo DCIS*Age:*average of 51 years ● well-circumscribed + freely movable ● aspiration may yield bloody fluid (cytology negative in 80%)√ intracystic mass on pneumocystography√ solid intracystic mass on US√ round benign appearing mass on mammography*Prognosis:*favorable B.INVASIVE BREAST CANCER (85%) 1.**Infiltrating / invasive ductal carcinoma** (65%) of no special type / otherwise not specified (NOS)10% false-negative ratio *Histo:* grade I=well-differentiatedgrade II=moderately differentiatedgrade III=poorly differentiated ● palpable in 70% ● larger by palpation than on mammogram√ spiculated mass (36%) is PRINCIPAL FINDING√ malignant calcifications (45-60%) 2.**Infiltrating / invasive lobular carcinoma** (5-10%)◊2nd most common type of breast cancer; 30-50% of patients will develop a second primary in same / opposite breast within 20 years◊Most frequently missed breast cancer (difficult to detect mammographically + clinically) with 19-43% false-negative rate (occult in dense breast)*Median age:*45-56 years; 2% of all ILC occur in women <35 years*Path:*multicentricity + bilaterality (in up to 1/3); tendency to grow around ducts, vessels, and lobules without destruction of anatomic structures (targetoid growth); no substantial connective tissue reaction*Histo:*20% grade I, 64% grade II, 16% grade IIIMetastases:GI tract, gynecologic organs, peritoneum, retroperitoneum, carcinomatous meningitis ● palpable in 69% ● area of subtle skin thickening / induration ● large hard mass / fine nodularityN.B.:may be seen on CC view only in many cases√ architectural distortion (= retraction of normal glandular tissue with thickening + disturbance of fibrous septa) in 18-30% is MOST COMMON MAMMOGRAPHIC FINDING*Histo:*straight single file of uniform small cells with round oval nuclei ("Indian files") growing around ducts resulting in subtle changes in architecture√ irregular spiculated mass >1 cm (16-28%)√ poorly defined mass ± spicules <1 cm (22%)√ asymmetric opacity (= ill-defined area of increased opacity without central tumor nidus) in 8-19%√ round / ovoid mass with regular borders (1%)√ microcalcifications (0-24%)√ retraction of skin (25%) + nipple (26%)√ skin thickening 3.**Tubular carcinoma** (6-8%)=well-differentiated form of ductal carcinoma(a)low grade: bilateral in 1:3(b)high grade: bilateral in 1:300*Associated with:*lobular carcinoma in situ in 40%*Mean age:*40-49 years ● positive family history in 40% ● nonpalpable√ high-opacity nodule with spiculated margins√ <17 mm in diameter; mean diameter of 8 mm*DDx:*radial scar 4.**Medullary carcinoma** (2%)=SOLID CIRCUMSCRIBED CARCINOMA◊Fastest growing breast cancer!*Path:*well-circumscribed mass with nodular architecture + lobulated contour; central necrosis is common in larger tumors; reminiscent of medullary cavity of bone*Histo:*intense lymphoplasmocytic reaction (reflecting host resistance); propensity for syncytial growth; no glands*Incidence:*11% of breast cancers in women <35 years of age; 40-50% of medullary cancers in women <50 years of age*Mean age:*46-54 years ● softer than average breast cancer√ well-defined round / oval noncalcified uniformly dense mass (hemorrhage) with lobulated margin√ may have partial / complete halo signUS:√ hypoechoic mass with some degree of through transmission√ distinct / indistinct margins√ large central cystic component*DDx:*fibroadenoma*Prognosis:*92% 10-year survival rate 5.**Mucinous / colloid carcinoma** (1.5-2%)*Path:* (a)pure form: aggregates of tumor cells surrounded by abundant pools of extracellular mucin (gelatinous / colloid fluid)(b)mixed form: contains areas of infiltrating ductal carcinoma not surrounded by mucin*Age:*1% in women <35 years; 7% of carcinomas in women >75 years ● slow growth rate of pure form ● "swish" / "crush" sensation during palpation ● 60% estrogen-receptor positive√ well-circumscribed usually lobulated mass of round /

ovoid shape√ pleomorphic clustered / clumped amorphous / punctate calcifications (rare)√ may enlarge fast (through mucin production)√ solid mass on US*Prognosis:*favorable
6.**Papillary carcinoma** (1-2-4%)=rare ductal carcinoma forming papillary structuresN.B.:Do not confuse with micropapillary / cribriform growth pattern of ductal carcinoma*Histo:*multilayered papillary projections extending from vascularized stalks; no myoepithelial layer (as in benign lesions); neurosecretory granules + positive CEA-reactivity in 85% (absent in benign lesions)*Types:* (a)multiple intraductal carcinomas with papillary configuration(b)Intracystic papillary carcinoma=in situ malignancy(c)invasive carcinoma with papillary growth pattern (microscopic frond formation)*Age:*25-89 (mean 50-60) years; peak age of 40-75 years ● palpable mass (67%) ● nipple discharge (22-35%) often tinged with blood ● rich in estrogen and progesterone receptorsLocation:single nodule in central portion of breast; multiple nodules extending from subareolar area to periphery of breast√ multinodular pattern (55%) = lobulated mass / cluster of well-defined contiguous nodules√ solitary well-circumscribed round / ovoid nodule with average diameter of 2-3 cm√ usually confined to single quadrant√ associated microcalcifications in 60%√ multiple filling defects / disruption of an irregular duct segment / complete obstruction of duct system at galactographyUS: √ solid hypoechoic mass with lobulated smooth margins + acoustic enhancement√ ± blood flow on color Doppler*Prognosis:*90% 5-year survival after simple mastectomy + axillary node dissection*DDx:*solitary central duct papilloma; multiple peripheral benign papillomas
C.PAGET DISEASE OF THE NIPPLE (5%) D.INFLAMMATORY BREAST CARCINOMA=tumor emboli within dermal lymphatics*Prevalence:*1-4% of breast cancers*Age:*52 years (on average)*Histo:*infiltrating ductal carcinomaLocation:L > R breast; bilaterality in 30-55% ● palpable tumor (63%) ● erythema of skin (13-64%) ● edema of skin (13%) ● nipple retraction (13%) ● palpable axillary adenopathy (up to 91%)√ tumor mass ± malignant-type calcifications√ diffusely increased breast density√ stromal coarsening (50%)√ thickening of Cooper ligaments√ extensive skin thickening (71%)*Prognosis:*2% 5-year survival; median survival time of 7 months (untreated) + 18 months (after radical mastectomy)*DDx:*breast abscess

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Epidemiology Of Breast Cancer Breast Cancer Evaluation Screening Of Asymptomatic Patients Role Of Mammography Role Of Breast Ultrasound Role Of Breast MRI Role Of Stereotaxic Biopsy

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Notes:

Epidemiology Of Breast Cancer *Incidence:* 2-5 breast cancers/1,000 women; in USA >142,000 new cases per year (of which 25,000 are in situ); 25% of all female malignancies One of 9 women will develop breast cancer during her life!*Age:*0.3-2% in women <30 years of age;15% in women <40 years of age; 85% in women >30 years of age *Mortality:*43,000 deaths per year Death rate has remained stable for past 60 years! ***Risk Factors (increasing risk):***
A.DEMOGRAPHIC FACTORS ● increasing age (66% of cancers in women >50 years):AgePrevalence of Cancer255:100,0001:19,6084080:100,0001:1,250451075:100,0001:9350180:100,0001:555553030:100,0001:3360240:100,0001:416Relative risk compared with woman of age 60: 30 years of age0.0760 years of age1.0035 years of age0.1970 years of age1.2740 years of age0.3580 years of age1.4550 years of age0.71 ● Whites > Blacks after age 40 ● Jewish women + nuns ● upper > lower social class ● unmarried > married women B.REPRODUCTIVE VARIABLES ● nulliparous > parousRelative risk compared with nulliparous: age at 1st pregnancy<19 years0.5age at 1st pregnancy20-30 years-age at 1st pregnancy30-34 years1.0age at 1st pregnancy>35 years>1.0 ● first full-term pregnancy after age 35: 2 x risk ● low parity > high parity ● early age at menarche (<12 years)Relative risk compared with onset of regular ovulatory cycle: menarche <12menarche >12immediately3.71.61-4 years2.31.6>5 years1.61.0 ● late age at menopauseRelative risk compared with menopause before age 44 years: natural menopause >55 years of age2.0 ● early bilateral oophorectomyRelative risk compared with menopause between ages 45-49 years: artificial menopause at 50-54 years1.34artificial menopause before age 450.77C.MULTIPLE PRIMARY CANCERS ● 4-5 x increase in risk for cancer in contralateral breast ● increased risk after ovarian + endometrial cancerD. FAMILY HISTORY ● breast cancer in first-degree relativeRelative risk compared with negative family Hx: (+) for mother1.8(+) for sister 2.5(+) for mother + sister5.6 ● 25% of patients with carcinoma have a positive family history ● carcinoma tends to affect successive generations approx. 10 years earlierE.BENIGN BREAST DISEASE ● 2-4 x increased risk with atypical hyperplasia Relative risk compared with no biopsy: benign breast disease in all patients1.5nonproliferative disease0.9proliferative disease without atypia1.6fibroadenoma + hyperplasia3.5atypical duct hyperplasia (ADH) no family history of breast cancer4.4family history of breast cancer8.9F.MAMMOGRAPHIC FEATURES ● prominent duct pattern + extremely dense breasts according to Wolfe classificationN1 (0.14%), P1 (0.52%), P2 (1.95%), DY (5.22%) G.RADIATION EXPOSUREexcess risk of 3.5-6 cases per 1,000,000 women per year per rad after a minimum latent period of 10 years (atomic bomb, fluoroscopy during treatment of tuberculosis, irradiation for postpartum mastitis) H.GEOGRAPHY ● Western + industrialized nations (highest incidence) ● Asia, Latin America, Africa (decreased risk)

Notes:

Breast Cancer Evaluation

PREDICTIVE VALUES OF RADIOGRAPHIC SIGNS FOR MALIGNANCY	
1. Classic mammographic findings of malignancy + palpable abnormality (only 3% of cancers present this way)	100%
2. Classic mammographic findings of malignancy + NO palpable finding (only 6% of cancers present this way)	74%
3. Indeterminate mammographic features + palpable mass	11%
4. Indeterminate mass + no palpable finding	5%
5. Mammographically benign mass	2%
6. Asymmetric density (mass questionable) + clinical finding	4%
7. Asymmetric density (mass questionable) + NO clinical finding	0%
8. Microcalcifications + clinical abnormality	25%
9. Microcalcifications + NO clinical abnormality (≥3 punctate irregular microcalcifications in area <1 cm^2)	21%
10. Vein dilatation	0%
11. Skin thickening	0%
12. Duct dilatation	0%

A.PRIMARY = LOCALIZING SIGNS OF BREAST CANCER1.Dominant mass seen on two views with(a)*spiculation* = stellate / star-burst appearance(= fine linear strands of tumor extension + desmoplastic response); "scirrhus" caused by: (1)infiltrating ductal carcinoma (75% of all invasive cancers)(2)invasive lobular carcinoma (occasionally)√ mass feels larger than its mammographic / sonographic size*DDx:*prior biopsy / trauma / infection(b)*smooth border* (1)intracystic carcinoma (rare): subareolar area; bloody aspiration(2)medullary carcinoma: soft tumor(3)mucinous / colloid carcinoma: soft tumor(4)papillary carcinoma√ "telltale" signs:lobulation, small comet tail, flattening of one side of the lesion, slight irregularity√ halo sign (= Mach band) may be present*DDx:*cyst (sonographic evaluation)(c)*lobulation* Appearance similar to fibroadenoma (only characteristic calcifications may exclude malignancy) ◊the likelihood of malignancy increases with number of lobulations ● clinical size of mass > radiographic size (Le Borgnes law) 2.Asymmetric density = star-shaped lesion√ distinct central tumor mass with volumetric rather than planar appearance (additional coned compression views!)√ denser relative to other areas (= vessels + trabeculae cannot be seen within high-density lesion)√ fat does not traverse density√ corona of spicules√ in any quadrant (but fatty replacement occurs last in upper outer quadrant)*DDx:*postsurgical fibrosis, traumatic fat necrosis, sclerosing duct hyperplasia3.MicrocalcificationsAssociated with malignant mass by mammogram in 40%, pathologically with special stains in 60%, on specimen radiography in 86% ◊20% of clustered microcalcifications represent a malignant process!(a)*shape:* fragmented, irregular contour, polymorphic, casting rod-shaped without polarity, Y-shaped branching pattern, granular "salt and pepper" pattern, reticular pattern(b)*density:* various densities(c)*size:* 100-300 µ (usually); rarely up to 2 mm(d)*distribution:* tight cluster over an area of 1 cm^2 or less is most suggestive; coursing along ductal system seen in ductal carcinoma with comedo elements4.Architectural distortiondue to desmoplastic reaction √ ragged irregular border*DDx:*postsurgical fibrosis5.Interval change(a)neodensity = de novo developing density (in 6% malignant)(b)enlarging mass (malignant in 10-15%)6.Enlarged single duct(low probability for cancer in asymptomatic woman with normal breast palpation) √ solitary dilated duct >3 cm long*DDx:*inspissated debris / blood, papilloma7.Diffuse increase in density (late finding)*Cause:*(1) plugging of dermal lymphatics with tumor cells(2) less flattening of sclerotic + fibrous elements of neoplasm in comparison with more compressible fibroglandular breast tissue
B.SECONDARY = NONLOCALIZING SIGNS OF BREAST CANCER1.Asymmetric thickening2.Asymmetric ducts, especially if discontinuous with subareolar area3.Skin changes(a)retraction = dimpling of skinfrom desmoplastic reaction causing shortening of Cooper ligaments / direct extension of tumor to skin *DDx:*trauma, biopsy, abscess, burns(b)skin thickening secondary to blocked lymphatic drainage / tumor in lymphatics ● peau dorange*DDx:*normal in inframammary region4.Nipple / areolar abnormalities(a)retraction / flattening of nipple*DDx:*normal variant(b)Paget disease = eczematoid appearance of nipple + areola in ductal carcinoma√ associated with ductal calcifications toward the nipple*DDx:*nipple eczema(c)nipple discharge ● spontaneous persistent discharge ● need not be bloody*DDx:*lactational discharge5.Abnormal veinsvenous diameter ratio of >1.4:1 in 75% of cancers; late sign + thus not very important 6.Axillary nodes (sign of advanced / occult cancer)√ >1.5 cm without fatty center*DDx:*reactive hyperplasia LOCATION OF BREAST MASSES benign + malignant masses are of similar distribution @upper outer quadrant (54%)@upper inner quadrant (14%)@lower outer quadrant (10%)@lower inner quadrant (7%)@retroareolar (15%)◊ Mediolateral oblique view is important part of screening because it includes largest portion of breast tissue + considers most common location of cancers! ***Metastatic Breast Cancer*** @Axillary lymph adenopathy*Incidence:*40-74%Risk for positive nodes:30% if primary >1 cm, 15% if primary <1 cm@Bone@Liver*Incidence:*48-60%US:√ hypoechoic (83%) / hyperechoic (17%) masses

Notes:

Screening Of Asymptomatic Patients *Definition of screening (World Health Organization):* A screening test must (a)be adequately sensitive and specific(b)be reproducible in its results(c)identify previously undiagnosed disease(d)be affordable(e)be acceptable to the public(f)include follow-up services *Guidelines of American Cancer Society, American College of Radiology, American Medical Association, National Cancer Institute:* 1.Breast self-examination to begin at age 202.Breast examination by physician every 3 years between 20-40 years, in yearly intervals after age 403.Baseline mammogram between age 35-40; follow-up screening based upon parenchymal pattern + family history4.Initial screening at 30 years if patient has first-degree relative with breast cancer in premenopausal years; follow-up screening based upon parenchymal pattern5.Mammography at yearly intervals after age 406.All women who have had prior breast cancer require annual follow-upAdditional recommendations: 1.Baseline mammogram 10 years earlier than age of mother / sister when their cancer was diagnosed2.Screening at 2-year intervals for women >70 years *Rate of detected abnormalities* 30 abnormalities in 1,000 screening mammograms: 20-23benign lesions7-10cancers ***Value Of Screening Mammography*** *Indication:* decrease in cancer mortality through earlier detection + intervention when tumor size small + lymph nodes negative; tumor grade of no prognostic significance in tumors <10 mm in size 1.Health Insurance Plan (HIP) 1963-1969randomized controlled study of 62,000 women aged 40-64 ● 25-30% reduction in mortality in women >50 years (followed for 18 years) ● 25% reduction in mortality in women 40-49 years (followed for 18 years); no significant effect at 5- and 10-year follow-up ● 19% of cancers found by mammography alone ● 61% of cancers found at physical examination ● effectiveness of screening <50 years of age is uncertain2.Breast Cancer Detection Demonstration Project (BCDDP) 1973-19804,443 cancers found in 283,000 asymptomatic volunteers ● 41.6% of cancers found by mammography alone (77% with negative nodes) ● 8.7% of cancers found by physical examination alone ● 59% of noninfiltrating cancers found by mammography alone ● 25% of cancers were intraductal (vs. 5% in previous series) ● 21% of cancers found in women aged 40-49 years (mammography alone detected 35.4%)3.Two-county Swedish trial 1977-1990randomized controlled study of 78,000 women in study group + 56,700 in control group aged 40-74 years (a)single MLO mammogram at 2-year intervals for women <50 years of age(b)single MLO mammogram at 3-year intervals for women ≥50 years of age ● 40% reduction in mortality at 7 years in women 50-74 years ● 0% reduction in mortality at 7 years in women 40-49 years OCCULT VERSUS PALPABLE CANCERS 27% are occult cancers (NO age difference) Positive axillary nodes:occult cancers (19%); palpable cancers (44%)10-year survival:occult cancers (65%); palpable cancers (25%)

Notes:

Role Of Mammography *Overall detection rate:* 58-69%; 8% if <1 cm in size*Mammographic accuracy:* 88%correctly diagnosed by radiologist27%detected only by mammography8%misinterpretations4%not detected15-30%positive predictive value (national average) ***Mammographically Missed Cancers*** False-negative screening mammogram = pathologic diagnosis of breast cancer within 1 year after negative mammogram with the following types of misses: (a)lesion could not be seen in retrospect (25-33%) = "acute cancer" = cancer surfacing in screening interval(b)cancer undetected by first reader but correctly identified by second reader (14%)(c)visible in retrospect on prior mammogram (61%)*Incidence:*approx. 10-25-30% of all cancers; approx. 3 cancers:2000 mammograms; 5-15-22% of palpable breast cancers*Cause:* 1.Misinterpretation (52%):(a)benign appearance (18%): medullary carcinoma, colloid carcinoma, intracystic papillary carcinoma, some infiltrating ductal carcinomas(b)present on previous mammogram (17%)(c)seen on one view only (9%)(d)site of previous biopsy (8%)2.Observer error (30-43%):overlooked, presence of obvious finding = "satisfied search" phenomenon, rushed interpretation, heavy caseload, extraneous distraction, eye fatigue 3.Technical error (5%):(a)poor image quality(b)failure to image region of interest4.Tumor biology:(a)small tumor size(b)failure to incite desmoplastic reaction (eg, invasive lobular carcinoma)(c)masked by dense breast parenchyma(d)no associated microcalcifications (approx. 50% of cancers)(e)developing soft-tissue radiopacity *Location of missed cancers:* retroglandular area (33%), lateral parenchyma (31%), central (18%), medial (13%), subareolar (4%)
Radiation-induced Breast Carcinoma ◊Lifetime risk with cumulative carcinogenic effect related to age!(a)women age <35: 7.5 additional cancers per 1 million irradiated women per year per rad(b)women age >35: 3.5 additional cancers per 1 million irradiated women per year per rad

Notes:

Role Of Breast Ultrasound *Indications:* Ultrasound is no screening tool!A.TARGETED EXAM(1)initial study of palpable lump in patient <30 years of age / pregnant / lactating Ultrasound will not add useful information in an area that contains only fatty tissue on a mammogram! (2)characterization of mammographic / palpable mass as fluid-filled / solid Ultrasound will add useful information if there is water-density tissue in the area of palpable abnormality Differentiation of cystic from solid lesion is the principal role of ultrasound!(3)additional evaluation of nonpalpable abnormality with uncertain mammographic diagnosis(4)search for focal lesion as cause for mammographic asymmetric density (5)confirmation of lesion seen in one mammographic projection onlyB.WHOLE-BREAST EXAM(1)Breast secretions(2)Suspected leaks from silicone implant(3)Follow-up of multiple known mammographic / sonographic lesions(4)Radiographically dense breast with strong family history of breast cancer(5)Metastases thought to be of breast origin, but with negative clinical + mammographic exam(6)Mammography not possible: "radiophobic" patient, bedridden patient, after mastectomyC.INTERVENTINAL PROCEDURE(1)Ultrasound-guided cyst aspiration(2)Ultrasound-guided core biopsy(3)Ultrasound-guided ductography, if(a)secretions cannot be expressed(b)duct cannot be cannulated *Accuracy:*98% accuracy for cysts; 99% accuracy for solid masses; small carcinomas have the least characteristic features

Notes:

Role Of Breast MRI *Indications:*ambiguous mammographic findings; positive clinical examination + negative mammographic findingsSensitivity:72-93-100%√ rapid enhancement reaching a markedly higher amplitude than parenchymal tissue*DDx:* fibroadenoma in premenopausal patient, ductal hyperplasia ± atypia, lobular neoplasia, inflammatory disease, scar <6 months old in nonirradiated breast, scar <18 months old in irradiated breast, fibrocystic change (apocrine metaplasia, sclerosing adenosis) √ intense early rim / peripheral enhancement (± central necrosis)√ malignant mass margination

Notes:

Role Of Stereotaxic Biopsy *Indications:*obviously malignant nonpalpable lesion, indeterminate likely benign lesion, anxiety over lesion*Types:*well-defined solid mass, indistinct / spiculated mass, clustered microcalcifications *Advantage:*single-stage surgical procedure*Problematic:*3-5 mm small lesion, fine scattered microcalcifications, indistinct density, area of architectural distortion*Excision:* radial scar suspected (in up to 28% associated with tubular carcinoma), lesion close to chest wall, lesion in axillary tail, very superficial lesion, atypia / atypical hyperplasia (in 49-61% associated with malignancy), carcinoma in situ (in 9-20% associated with invasion), branching microcalcifications suggestive of DCIS with comedo necrosis *Sensitivity:*85-99% with core needle biopsy (100% specific), 68-93% with fine-needle aspiration (88-100% specific)*Miss rate:*3-8% for stereotaxic biopsy, 3% for surgery

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Notes:

BREAST CYST

*Incidence:*most common single cause of breast lumps between 35 and 55 years of age*Age:*any; most common in later reproductive years + around menopause*Histo:*cyst wall lined by single layer of(a)flattened epithelial cells; cyst fluid with Na^+/ K^+ ratio ≥3(b)epithelial cells with apocrine metaplasia (secretory function); cyst fluid with Na^+/K^+ ratio <3*Cause:*fluid cannot be absorbed due to obstruction of extralobular terminal duct by fibrosis / intraductal epithelial proliferation ● size changes over time

Simple Breast Cyst Complex Breast Cyst

Notes:

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Simple Breast Cyst √ well-defined flattened oval / round (if under pressure) mammographic mass + surrounding halo(DDx: well-defined solid mass)√ solitary / multiple √ needle aspiration of fluid (proof) + postaspiration mammogram as new baselineUS (98-100% accuracy): ◊Correlate with palpation / mammogram as to size, shape, location, surrounding tissue density!√ spherical / ovoid lesion with anechoic center√ well-circumscribed thin echogenic capsule√ posterior acoustic enhancement (may be difficult to demonstrate in small / deeply situated cysts)√ thin edge shadows√ occasionally multilocular ± thin septations / cluster of cysts PNEUMOCYSTOGRAPHY (for symptomatic cysts): √ air remains mammographically detectable for up to 3 weeks√ therapeutic effect of air insufflation (equal to 60-70% of aspirated fluid volume): no cyst recurrence in 85-94% (40-45% cyst recurrence without air insufflation)

Notes:

Complex Breast Cyst =any cyst that does not meet criteria of simple cyst*Cause:*fibrocystic changes (vast majority), infection, malignancy (extremely rare)◊0.3% of all breast cancers are intracystic◊Patients with apocrine cysts are at greater risk to develop breast cancer!√ uniformly thick wall + tenderness = inflammation / infection√ diffuse low-level internal echoes (= "foam" cyst)(a)with mobility upon increase in power output= subcellular material like protein globs, floating cholesterol crystals, cellular debris(b)without mobility upon increase in power output= cells like foamy macrophages, apocrine metaplasia, epithelial cells, pus, blood√ fluid-debris level*Rx:*aspiration to rule out blood / pus√ thick septation / eccentric wall thickeningfurther characterized by protruding ill-defined outer margin, convex microlobulated inner margin ("mural nodule"), nonmobile mass with coarse heterogeneous echotexture, CD flow within thickening*Rx:*treated like solid nodule√ spongelike cluster of microcysts*Rx:*treated like solid nodule*Rx:*complete aspiration (assures benign cause), core needle biopsy (if partially / nonaspiratable)DDx:artifactual scatter in superficial / deep small cysts, fibroadenoma, papilloma, carcinoma CYST ASPIRATION ● inspection of cyst fluid:(a)normal: turbid greenish / grayish / black fluid(b)abnormal: straw-colored clear fluid / dark blood√ needle moves within nonaspiratable complex cyst√ fluid without blood should be discarded√ bloody fluid should be examined cytologically

Notes:

CARCINOMA OF MALE BREAST

*Incidence:*0.2%; 1,400 new cases/year with 300 deaths;3.7% of male breast carcinomas occur in men with Klinefelter syndrome!*Peak age:*60-69 years*At risk:*(males with increased estrogen levels)1.Klinefelter syndrome (20-fold risk over normals):XXY chromosomes 2.Liver disease: cirrhosis, schistosomiasis, malnutrition3.Radiation therapy to chest4.Occupational heat exposure (diminished testicular function)5.Testicular atrophy: injury, mumps-orchitis, undescended testis6.Jewish background7.Family history Gynecomastia is NOT a risk factor! *Histo:*infiltrating ductal carcinoma ● firm painless retroareolar / upper-outer-quadrant mass ● breast swelling, bloody nipple discharge, retractionLocation:L >R breast; bilaterality is uncommon√ resembles scirrhous carcinoma of female breast√ usually located eccentrically√ calcifications fewer + more scattered + more round + larger√ enlarged axillary nodes (in 50% at time of presentation)√ metastases to pleura, lung, bone, liverDelay in diagnosis from onset of symptoms: 6-18 months *Rx:*surgery, hormonal manipulation (85% estrogen receptor and 75% progesterone receptor positive)*Prognosis:*5-year survival rate for stage 1 = 82-100%for stage 2 = 44-77%, for stage 3 = 16-45%, stage 4 = 4-8% (not worse than for women!)*DDx:*breast abscess, gynecomastia, epidermal inclusion cyst

Notes:

CHRONIC ABSCESS OF BREAST

=COLD ABSCESS usually seen in lactating women ● fever, pain, increased WBC (clinical diagnosis) ● rapid response to antibioticsLocation:most commonly in central / subareolar area√ ill-defined mass of increased density with flamelike contour√ secondary changes common: architectural distortion, nipple + areolar retraction, lymphedema, skin thickening, pathologic axillary nodes√ liquefied center can be aspiratedUS: √ anechoic / nearly anechoic area with posterior enhancement

Notes:

CYSTOSARCOMA PHYLLODES
=GIANT FIBROADENOMA = ADENOSARCOMA=PHYLLODE TUMOR=usually benign giant form of intracanalicular fibroadenoma*Incidence:*1: 6,300 examinations; 0.3-1.5% of all breast tumors; 3% of all fibroadenomas*Age:*5th-6th decade (mean age of 45 years, occasionally in women <20 years of age*Histo:*similar to fibroadenoma but with increased cellularity + pleomorphism (wide variations in size, shape, differentiation) of its stromal elements; fibroepithelial tumor with leaflike (phyllodes) growth pattern = branching projections of tissue into cystic cavities; cavernous structures contain mucus; cystic degeneration + hemorrhage ● rapidly enlarging breast mass; periods of remission ● sense of fullness ● huge, firm, mobile, discrete, lobulated, smooth mass ● discoloration of skin, wide veins, shining skin √ large noncalcified mass with smooth polylobulated margins mimicking fibroadenoma√ rapid growth to large size (>6-8 cm), may fill entire breastUS: √ fluid-filled clefts in large tumors*Prognosis:*limited invasion frequently seen; 15-20% recurrence rate if not completely excised*Cx:*in 5-10% degeneration into malignant fibrous histiocytoma / fibrosarcoma / liposarcoma / chondrosarcoma / osteosarcoma with local invasion + hematogenous metastases to lung, pleura, bone (axillary metastases quite rare)

Notes:

DERMATOPATHIC LYMPHADENOPATHY
=benign reactive lymphadenopathy within breast associated with cutaneous rashes*Cause:*exfoliative dermatitis, erythroderma, psoriasis, atopic dermatitis, skin infection)*Histo:*follicular pattern retained, germinal centers enlarged, enlarged paracortical area with pale-staining cells (lymphocytes, Langerhans cells, interdigitating reticulum cells) ● mobile nontender firm subcutaneous nodulesLocation:often bilateralSite:predominantly upper outer quadrant regional subcentimeter masses with central / peripheral radiolucent notches

Notes:

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EPIDERMAL INCLUSION CYST
=benign cutaneous / subcutaneous lesion*Cause:*congenital, metaplasia, trauma (needle biopsy, reduction mammoplasty), obstructed hair follicle*Path:*cyst filled with keratinHisto:stratified squamous epithelium ● smooth round nodule attached to skin with blackened pore, movable against underlying tissue√ circumscribed round / oval iso- / high-density mass of 0.8-10.0 cm in diameter√ may contain heterogeneous microcalcificationsUS: √ circumscribed hypoechoic solid mass extending into dermis*DDx:*sebaceous cyst (epithelial cysts containing sebaceous glands)

Notes:

FAT NECROSIS OF BREAST
=TRAUMATIC LIPID CYST = OIL CYST = aseptic saponification of fat by tissue lipase after local destruction of fat cells with release of lipids + hemorrhage + fibrotic proliferation*Etiology:*direct external trauma, breast biopsy, reduction mammoplasty, irradiation, nodular panniculitis (Weber-Christian disease), ductal ectasia of chronic mastitis*Incidence:* 0.5% of breast biopsies*Histo:*cavity with oily material surrounded by "foam cells" (= lipid-laden macrophages) ● history of trauma in 40% (eg, prior surgery, radiation >6 months ago, reduction mammoplasty, lumpectomy) ● firm, slightly fixed mass ● skin retraction (50%) ● yellowish fatty fluid on aspirationLocation: anywhere; more common in areolar region; near biopsy site / surgical scar√ ill-defined irregular spiculated dense mass (indistinguishable from carcinoma if associated with distortion, skin thickening, retraction)√ well-circumscribed mass with translucent areas at center (= homogeneous fat density of oil cyst) surrounded by thin pseudocapsule (in old lesions)√ calcifies in 4-7% (= **liponecrosis macrocystica calcificans**)√ occasionally curvilinear / eggshell calcification in wall √ fine spicules of low density vary with projection√ localized skin thickening / retraction possibleUS: √ hypo- / anechoic mass with ill- / well-defined margins ± acoustic shadowing√ complex cyst with mural nodules / echogenic bands **Weber-Christian Disease** =nonsuppurative panniculitis with recurrent bouts of inflammation = areas of fat necrosis, involving subcutaneous fat + fat within internal organs ● accompanied by fever + nodules over trunk and limbs

Notes:

FIBROADENOMA

=ADULT-TYPE FIBROADENOMA=estrogen-induced benign tumor originating from TDLU; forms during adolescence; pregnancy + lactation are growth stimulants; regression after menopause (mucoid degeneration, hyalinization, involution of epithelial components, calcification)*Incidence:*3rd most common type of breast lesion after fibrocystic disease + carcinoma; most common benign solid tumor in women of childbearing age*Age:*mean age of 30 years (range 13-80 years); median age 25 years; most common breast tumor under age 25 years*Hormonal influence:* slight enlargement at end of menstrual cycle + during pregnancy; regresses after menopause; may occur in postmenopausal women receiving estrogen replacement therapy *Histo:* mixture of proliferated fibrous stroma + epithelial ductal structures(a)intracanalicular fibroadenoma compressing ducts(b)pericanalicular fibroadenoma without duct compression(c)combination ● firm, smooth, sometimes lobulated, freely movable mass ● in 35% not palpable ● NO skin fixation ● rarely tender / painful ● clinical size = radiographic sizeSize:1-5 cm (in 60%); multiple in 15-25%; bilateral in 4%√ circular / oval-shaped lesion of low density√ nodular / lobulated contour when larger (areas with different growth rates)√ smooth, discrete margins (indistinguishable from cysts when small)√ often with "halo" sign√ smoothly contoured calcifications of high + fairly equal density in 3% due to necrosis from regressive changes in older patients:(a)peripheral subcapsular myxoid degeneration√ peripheral marginal ringlike calcifications(b)central myxoid degeneration√ "popcorn" type of calcification (PATHOGNOMONIC)(c)calcifications within ductal elements√ pleomorphic linear ± branching pattern◊Calcifications enlarge as soft-tissue component regresses!US: √ round (3%) / oval mass (96%) with length-to-depth ratio of >1.4 (in carcinomas usually <1.4)√ hypoechoic similar to fat lobules (80-96%) / hyperechoic / mixed pattern / anechoic / isoechoic compared with adjacent fibroglandular tissue√ homogeneous (48-89%) / inhomogeneous (12-52%) texture√ regular (57%) / lobulated (15-31%) / irregular (6-58%) contour√ "hump and dip" sign = small focal contour bulge immediately contiguous with a small sulcus (57%)√ intratumoral bright echoes (10%)= macrocalcifications√ posterior acoustic enhancement (17-25%) / acoustic shadow without calcifications (9-11%)√ echogenic halo (capsule) with lateral shadowing

Juvenile / Giant / Cellular Fibroadenoma

Notes:

Juvenile / Giant / Cellular Fibroadenoma =fibroadenoma >5 cm in diameter / weighing >500 g*Cause:*hyperplasia + distortion of normal breast lobules secondary to hormonal imbalances between estradiol + progesterone levels*Age:*any (mostly in adolescent girls)*Histo:*more glandular + more stromal cellularity than adult type of fibroadenoma; ductal epithelial hyperplasia ● rapidly enlarging well-circumscribed nontender mass ● dilated superficial veins, stretched skin discrete mass with rounded borders *DDx:*medullary / mucinous / papillary carcinoma / carcinoma within fibroadenoma

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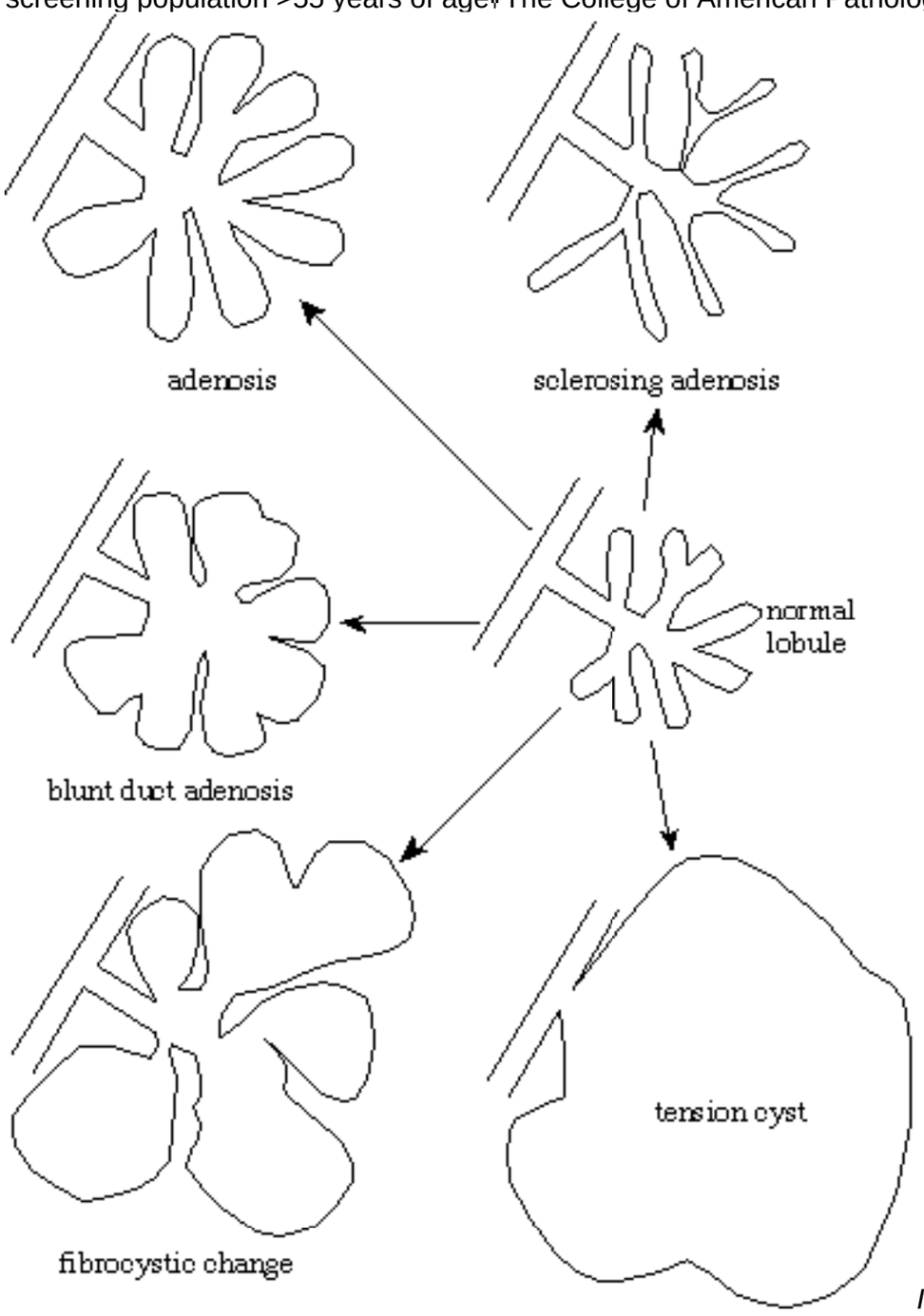
Notes:

FIBROCYSTIC CHANGES
=Mazoplasia = mastitis fibrosa cystica = chronic cystic mastitis = cystic disease = generalized breast hyperplasia = desquamated epithelial hyperplasia= fibroadenomatosis = mammary dysplasia = Schimmelbusch disease = fibrous mastitis = mammary proliferative disease ◊Not a disease since found in 72% of screening population >55 years of age◊The College of American Pathologists suggests to use the term "fibrocystic changes / condition" in mammography reports!



*Incidence:*most common diffuse breast disorder; in 51% of 3,000 autopsies*Age:*35-55 years*Etiology:*exaggeration of normal cyclical proliferation + involution of the breast with production + incomplete absorption of fluid by apocrine cells ● asymptomatic in macrocystic disease ● fullness, tenderness, pain in microcystic disease ● palpable nodules + thickening ● symptoms occur with ovulation; regression with pregnancy + menopause*Histo:* (1)overgrowth of fibrous connective tissue = stromal fibrosis, fibroadenoma(2)cystic dilatation of ducts + cyst formation (in 100% microscopic, in 20% macroscopic)(3)hyperplasia of ducts + lobules + acini = adenosis; ductal papillomatosis√ individual round / ovoid cysts with discrete smooth margins√ lobulated multilocular cyst√ enlarged nodular pattern (= fluid-distended lobules + extensive extralobular fibrous connective tissue overgrowth)√ "teacup-like" curvilinear thin calcifications with horizontal beam + low-density round calcifications in craniocaudal projection = milk of calcium (4%)√ "oyster pearl-like" / psammoma-like calcifications√ "involutional type" calcifications = very fine punctate calcifications evenly distributed within one / more lobes against a fatty background (from mild degree of hyperplasia in subsequently atrophied glandular tissue)US: √ ductal pattern, ductectasia, cysts, ill-defined focal lesions *Risk for Invasive Breast Carcinoma* A. NO INCREASED RISK1.Nonproliferative lesions: adenosis, florid adenosis, apocrine metaplasia without atypia, macro- / microcysts, duct ectasia, fibrosis, mild hyperplasia (more than 2 but not more than 4 epithelial cells deep), mastitis, periductal mastitis, squamous metaplasia2.FibroadenomaB.SLIGHTLY INCREASED RISK (1.5-2 times):1.Moderate + florid solid / papillary hyperplasia2.Papilloma with fibrovascular core3.Sclerosing adenosisC.MODERATELY INCRAESED RISK (5 times):Ductal / lobular atypical hyperplasia (borderline lesion with some features of carcinoma in situ) D.HIGH RISK (8-11 times):1.Atypical hyperplasia + family history of breast cancer2.Ductal / lobular carcinoma in situ

Adenosis Sclerosing Adenosis Fibrosis Atypical Lobular Hyperplasia Atypical Ductal Hyperplasia Intraductal Papillomatosis

Notes:

Adenosis =hyperplasia + hypertrophy of glandular elements√ increase in size of lobules to 3-7 mm√ "snowflake pattern" of widespread ill-defined nodular densities√ adenosis lobules are sonographically iso- to mildly hypoechoic compared with fat

Notes:

Sclerosing Adenosis =adenosis + reactive fibrosis = proliferating acinar structure maintaining a lobular configuration√ adenosis + diffusely scattered calcifications (calcifications in cystically dilated acinar structure)√ diffusely dense breast√ focally dense breast appearing as a nodule / spiculated lesion

Notes:

Fibrosis √ round / oval clustered microcalcifications with smooth contours + associated fine granular calcifications filling lobules

Notes:

Atypical Lobular Hyperplasia =proliferation of round cells of LCIS type growing along terminal ducts in permeative fashion (pagetoid growth) between benign epithelium + basal myoepithelium BUT NOT completely obliterating terminal ductal lumina / distending lobules (as in lobular carcinoma in situ)* no mammographic correlate

Notes:

Atypical Ductal Hyperplasia =low-grade intraductal proliferation with partial / incompletely developed features of noncomedo DCIS√ frequent calcifications

Notes:

Intraductal Papillomatosis = hyperplastic polypoid lesions within a duct *Age:*perimenopausal ● spontaneous bloody / serous / serosanguinous nipple discharge (most common cause of nipple discharge)✓ small retroareolar opacity (= dilated duct) extending 2-3 cm into breast✓ intraluminal filling defect on galactography

Notes:

GALACTOCELE
=retention of fatty material in areas of cystic duct dilatation appearing during / shortly after lactation*Cause:*? abrupt suppression of lactation*Age:*occurs during / shortly after lactation ● thick inspissated milky fluid (colostrum)Location:retroareolar area√ large radiopaque lesion of water density (1st phase)√ smaller lesion of mixed density + fat-water level with horizontal beam (2nd phase)√ small radiolucent lesion resembling lipoma√ ± fluid-calcium level

Notes:

GRANULAR CELL TUMOR

=GRANULAR CELL MYOBLASTOMA OF BREAST=benign tumor, occasionally locally invasive + metastasizing*Origin:*? Schwann cell, smooth muscle, or undifferentiated mesenchymal cell*Prevalence:*1:1,000 primary breast carcinomas*Age :*20-59 (mean 35) years; more common in Blacks*Histo:*rounded groups of large cells with small dark regular nuclei + abundant eosinophilic granular cytoplasm; not immunoreactive to cytokeratin + epithelial membrane antigen BUT to S-100 protein*DDx:*carcinoma, lymphoma, metastasis◊Fine-needle aspirate may be difficult to interpret!Location:tongue, skin, bronchial wall, subcutaneous breast tissue (6-8%)Site:more commonly other than upper outer quadrant ● asymmetric lump with slow growth, hardness, skin fixation / retraction, ulceration ● often fixed to pectoralis fascia√ well-circumscribed spiculated mass 1-3 cm in diameter√ stellate extensions (tumor insinuating itself into surrounding breast tissue)√ may exhibit acoustic shadow*Rx:*wide local excision

Notes:

GYNECOMASTIA
Cause: (1)Hormonal(a)puberty: high estradiol levels(b)older men: decline in serum testosterone levels(c)hypogonadism (Klinefelter syndrome, testicular neoplasm)(d)tumors: adrenal carcinoma, pituitary adenoma, testicular tumor, hyperthyroidism(2)Systemic disordersadvanced alcoholic cirrhosis, hemodialysis in chronic renal failure, chronic pulmonary disease (emphysema, TB), malnutrition (3)Drug-inducedestrogen treatment for prostate cancer, digitalis, cimetidine, thiazide, spironolactone, reserpine, isoniazid, ergotamine, marijuana (4)Neoplasm: hepatoma (with estrogen production)(5)Idiopathic*mnemonic:*"CODES"**C**irrhosis **O**besity **D**igitalis **E**strogen **S**pironolactone *Incidence:*85% of all male breast masses*Age:*adolescent boys (40%), men >50 years (32%)*Histo:*increased number of ducts, proliferation of duct epithelium, periductal edema, fibroplastic stroma, adipose tissue ● palpable firm mass >2 cm in subareolar regionLocation:bilateral (63%), left-sided (27%), right-sided (10%)√ mild prominence of subareolar ducts in flame-shaped distribution (focal type)√ homogeneously dense breast (diffuse type)*DDx:*pseudogynecomastia (= fatty proliferation)

Notes:

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HAMARTOMA OF BREAST
=FIBROADENOLIPOMA = LIPOFIBROADENOMA=ADENOLIPOMA*Incidence:* 2-16:10,000 mammograms*Mean age:*45 (27-88) years*Histo:*normal / dysplastic mammary tissue composed of dense fibrous tissue + variable amount of fat, delineated from surrounding tissue without a true capsule ● soft, often nonpalpable (60%)Location:retroareolar (30%),upper outer quadrant (35%)√ round / ovoid well-circumscribed mass usually > 3 cm√ mixed density with mottled center (secondary to fat)= "slice of sausage" pattern√ thin smooth pseudocapsule (= thin layer of surrounding fibrous tissue)√ peripheral radiolucent zone√ may contain calcifications*DDx:*liposarcoma, Cowden disease

Notes:

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HEMATOMA OF BREAST

Cause:(1)surgery / biopsy (most common)(2)blunt trauma(3)coagulopathy (leukemia, thrombocytopenia)(4)anticoagulant therapy√ well-defined ovoid mass (= hemorrhagic cyst)√ ill-defined mass with diffuse increased density (edema + hemorrhage)√ adjacent skin thickening / prominence of reticular structures√ regression within several weeks leaving (a) no trace(b) architectural distortion (c) incomplete resolution√ calcifications (occasionally)US: √ hypoechoic mass with internal echoes

Notes:

JUVENILE PAPILLOMATOSIS
*Path:*many aggregated cysts with interspersed dense stroma*Histo:*cysts lined by flat duct epithelium / epithelium with apocrine metaplasia, sclerosing adenosis, duct stasis; marked papillary hyperplasia of duct epithelium with often extreme atypia*Mean age:*23 years (range of 12-48 years) ● localized palpable tumor ● family history of breast cancer in 28% (affected first-degree relative in 8%; in one / more relatives in 28%)*Prognosis:*development of synchronous (4%) / metachronous (4%) breast cancer after 8-9 years.*DDx:*fibroadenoma

Notes:

LACTATING ADENOMA
=newly discovered painless mass during 3rd trimester of pregnancy / in lactating woman*Etiology:*? variant of fibroadenoma / tubular adenoma / lobular hyperplasia or de novo neoplasm*Path:*well-circumscribed yellow spherical mass with lobulated surface + rubbery firm texture and without capsule*Histo:*secretory lobules lined by granular and foamy to vacuolated cytoplasm + separated by delicate connective tissue ● firm freely movable painless mass√ homogeneously hypoechoic / isoechoic mass√ posterior acoustic enhancement (most) / shadowing√ fibrous septa*Prognosis:*regression after completion of breast feeding*DDx:*breast carcinoma (1:1,300-1:6,200 pregnancies)

Notes:

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LIPOMA OF BREAST

=usually solitary asymptomatic slow-growing lesion*Mean age:*45 years + postmenopause ● soft, freely movable, well delineated√ usually >2 cm√ radiolucent lesion easily seen in dense breast; almost invisible in fatty breast√ discrete thin radiopaque line (= capsule), seen in most of its circumference√ displacement of adjacent breast parenchyma√ calcification with fat necrosis (extremely rare)*DDx:*fat lobule surrounded by trabeculae / suspensory ligaments

Notes:

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LYMPHOMA OF BREAST

A. Primary lymphoma: 0.05-0.53% prevalenceB. Metastatic lymphoma*Histo:*large cell type NHL (majority), Hodgkin disease, leukemia, plasmacytoma*Age:*50-60 years; M < FLocation:right-sided predominance; 13% bilateral√ round / oval mass√ infiltrate with poorly defined borders√ skin thickening√ axillary nodes involved in 35%
PSEUDOLYMPHOMA =lymphoreticular lesion as an overwhelming response to trauma

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Notes:

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MAMMARY DUCT ECTASIA
=PLASMA CELL MASTITIS = VARICOCELE TUMOR OF BREAST = MASTITIS OBLITERANS= COMEDOMASTITIS = PERIDUCTAL MASTITIS= SECRETORY DISEASE OF BREAST=rare aseptic inflammation of subareolar area*Pathogenesis (speculative):* (1)Stasis of intraductal secretion leads to duct dilatation + leakage of inspissated material into parenchyma giving rise to an aseptic chemical mastitis (periductal mastitis); the extravasated material is rich in fatty acids = nontraumatic fat necrosis(2)Periductal inflammation causes damage to elastic lamina of duct wall resulting in duct dilatation *Histo:*ductal ectasia, heavily calcified ductal secretions; infiltration of plasma cells + giant cells + eosinophils*Mean age:*54 years ● often asymptomatic ● breast pain, nipple discharge, nipple retraction, mamillary fistula, subareolar breast mass Location:subareolar, often bilateral + symmetric; may be unilateral + focal√ dense triangular mass with apex toward nipple√ distended ducts connecting to nipple√ periphery blending with normal tissue√ multiple often bilateral dense round / oval calcifications with lucent center + polarity (= orientation toward nipple)(a)periductal√ oval / elongated calcified ring around dilated ducts with very dense periphery (surrounding deposits of fibrosis + fat necrosis)(b)intraductal√ fairly uniform linear, often "needle-shaped" calcifications of wide caliber, occasionally branching (within ducts / confined to duct walls)√ nipple retraction / skin thickening may occur*Sequela:*cholesterol granuloma*DDx:*breast cancer

Notes:

MAMMOPLASTY
=COSMETIC BREAST SURGERY

Augmentation Mammoplasty Reduction Mammoplasty

Notes:

Augmentation **Mammoplasty** Most frequently performed plastic surgery in U.S. *Frequency:*150,000 procedures in 1993 (80% for cosmesis, 20% for reconstruction); 2 million American women have breast implants (estimate)Methods: 1.Injection augmentation (no longer practiced): paraffin, silicone, fat from liposuction*Cx:*tissue necrosis resulting in dense, hard, tender breast masses2.Implants(a)spongelike masses of Ivalon, Etrheron, Teflon(b)Silicone elastomer (silastic) smooth / textured shell containing silicone gel / saline: >100 varieties-single lumen of polymerized methyl polysiloxane with smooth / textured outer silicone shell / polyurethane coating-double lumen with inner core of silicone + outer chamber of saline-triple lumen(c)expandable implant ± intraluminal valves = saline injection into port with gradual tissue expansion for breast reconstructionLocation:retroglandular / subpectoral3.Autogenous tissue transplantation(for breast reconstruction) with musculocutaneous flaps: transverse rectus abdominis muscle (TRAM), latissimus dorsi, tensor fascia lata, gluteus maximus *Mammographic technique for implants:* 1.Two standard views (CC and MLO views) for most posterior breast tissue◊22-83% of fibroglandular breast tissue obscured by implant depending on size of breast + location of implant + degree of capsular contraction on standard views!◊The false-negative rate of mammography increases from 10-20% to 41% in patients with implants!2.Two Eklund (= implant displacement) views (CC and 90° LAT views) for compression views of anterior breast tissue = "push-back" view = breast tissue pulled anteriorly in front of implant while implant is pushed posteriorly + superiorly thus excluding most of the implant *Cx of silicone-gel-filled implant:* 1.Capsular fibrosis, calcification, contracture (15-50%): more frequent with retroglandular implants ● distortion of breast contour with hard capsule√ crenulated contour (US helpful) √ capsular calcifications at periphery of prosthesis√ fibrous capsule delineated by US (unleaked silicone is echolucent)2.Implant migration*Cause:*overdistension of implant pocket at surgery 3.Rupture of prosthesis*Prevalence:*>50% after 12 years ● change in contour / location of implant ● flattening of implant ● breast pain A.INTRACAPSULAR RUPTURE (more common) = broken implant casing with silicone leakage contained by intact fibrous capsuleMammo (11-23% sensitive, 89-98% specific): √ bulging / peaking of implant contour US (59-70% sensitive, 57-92% specific, 49% accurate): √ "stepladder" sign = series of parallel horizontal echogenic straight / curvilinear lines inside implant (= collapsed implant shell floating within silicone gel)√ heterogeneous aggregates of low- to medium-level echogenicity (65% sensitive, 57% specific)N.B.:visualization of internal lumen within anechoic space in double-lumen implants can be confused on US with intracapsular rupture MR (81-94% sensitive, 93-97% specific, 84% accurate): √ "linguine" sign = multiple hypointense wavy lines within implant (= pieces of free-floating collapsed envelope surrounded by silicone gel)√ "inverted teardrop" / "noose" / "keyhole" / "lariat (= lasso)" sign = loop-shaped hypointense structure contiguous with implant envelope (= small focal invagination of shell with silicone on either side)√ = infolded polyurethane coat of a single lumen prosthesis√ hypointense subcapsular lines paralleling the fibrous capsule (= minimally displaced ruptured shell as early sign) (DDx: phase-encoding artifact caused by motion) B.EXTRACAPSULAR RUPTURE= extrusion + migration of silicone droplets through tear in both implant + overlying fibrous capsule ● palpable breast masses ● paresthesia of arm (from nerve impingement secondary to fibrosis surrounding silicone migrated to axilla / brachial plexus) ● silicone nipple discharge (rare)√ silicone droplets in breast√ axillary silicone lymphadenopathyUS: √ "snowstorm" pattern = markedly hyperechoic nodule with well-defined anterior but indistinct posterior margin and intense shadowing echogenic noise (= free silicone droplets mixing with breast tissue)√ occasionally "dirty" complex cyst (= larger collection of free silicone)(c)"gel bleed" = leakage of silicone through porous but intact implant gel4.Localized pain / paresthesia5.? development of autoimmune disorders (eg, scleroderma, lupus erythematosus)6.Infection / hematoma formation

Notes:

Reduction Mammoplasty √ swirled architectural distortion (in inferior breast best seen on mediolateral view) √ postsurgical distortion √ residual isolated islands of breast tissue √ fat necrosis √ dystrophic calcifications √ asymmetric tissue oriented in nonanatomic distribution

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Notes:

Puerperal Mastitis =usually interstitial infection during lactational period(a) through infected nipple cracks (b) hematogenous (c) ascending via ducts = galactophoritis *Organism:*staphylococcus, streptococcus ● tender swollen red breast (DDx: inflammatory carcinoma) ● enlarged painful axillary lymph nodes ● ± febrile, elevated ESR, leukocytosis√ diffuse increased density√ diffuse skin thickening√ swelling of breast√ enlarged axillary lymph nodes√ rapid resolution under antibiotic therapy

Notes:

Nonpuerperal Mastitis 1.Infected cyst2.Purulent mastitis with abscess formation3.Plasma cell mastitis4.Nonspecific mastitis

Notes:

Granulomatous Mastitis 1.Foreign-body granuloma2.Specific disease (TB, sarcoidosis, leprosy, syphilis, actinomycosis, typhus)3.Parasitic disease (hydatid disease, cysticercosis, filariasis, schistosomiasis)

Notes:

METASTASES TO BREAST

*Incidence:*1%*Mean age:*43 yearsPrimaries:leukemia / lymphoma > malignant melanoma > ovarian carcinoma > lung cancer > sarcoma In up to 40% no known history of primary cancer! solitary mass (85%), esp. in upper outer quadrant multiple masses skin adherence (25%) ± skin thickening axillary node involvement (40%)

Notes:

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PAGET DISEASE OF THE NIPPLE
=uncommon manifestation of breast cancer ● eczemalike scaling + excoriation of nipple and areola ● nipple discharge + itching*Histo:*Paget cell = large pleomorphic cells with pale cytoplasm invading the epidermis; histologically + biologically similar to comedocarcinoma*Associated with:* extensive invasive / noninvasive ductal carcinoma limited to one duct in subareolar area / remote + multicentric √ negative mammogram in 50% √ nipple / areolar thickening√ dilated duct√ linearly distributed microcalcifications√ retroareolar soft-tissue mass*Prognosis:*similar to infiltrating duct carcinoma

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Notes:

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PAPILLOMA OF BREAST
=usually benign proliferation of ductal epithelial tissue*Age:*30-77 years (juvenile papillomatosis = 20-26 years)*Histo:*hyperplastic proliferation of ductal epithelium; lesion may be pedunculated / broad-based; connective tissue stalk covered by epithelial cells proliferating in the form of apocrine metaplasia / solid hyperplasia may cause duct obstruction + distension to form an intracystic papilloma*DDx:*invasive papillary carcinoma

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Central Solitary Papilloma Peripheral Multiple Papillomas

Notes:

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Central Solitary Papilloma Location:subareolar within major ductNOT premalignant ● spontaneous bloody / serous / clear nipple discharge (52-100%)Most common cause of serous / sanguinous nipple discharge! ● "trigger point" = nipple discharge produced upon compression of area with papilloma ● intermittent mass disappearing with discharge√ negative mammogram / intraductal nodules in subareolar area√ asymmetrically dilated single duct√ subareolar amorphous coarse calcifications√ dilated duct with obstructing / distorting intraluminal filling defect on ductography (= galactography)*Cx:*0-14% frequency of carcinoma development

Notes:

Peripheral Multiple Papillomas Location:within terminal ductal lobular unit; bilateral in up to 14%*In 10-38% associated with:* atypical ductal hyperplasia, lobular carcinoma in situ, papillary + cribriform intraductal cancers, radial scar • nipple discharge (20%)√ round / oval / slightly lobulated well-circumscribed nodules√ segmental distribution with dilated ducts extending from beneath the nipple (20%)√ may be associated with coarse microcalcifications*Cx:*5% frequency of carcinoma development; increased risk dependent on degree of cellular atypia*Prognosis:*in 24% recurrence after surgical treatment

Notes:

RADIAL SCAR
=SCLEROSING DUCT HYPERPLASIA = INDURATIVE MASTOPATHY = FOCAL FIBROUS DISEASE= BENIGN SCLEROSING DUCTAL PROLIFERATION = NONENCAPSULATED SCLEROSING LESION = INFILTRATING EPITHELIOSIS =benign proliferative breast lesion (malignant potential is controversial); "scar" = fibroelastic center with surrounding stellate proliferation of contracted ducts + lobules*Incidence:*1-2/1,000 screening mammograms; in 2-16% of mastectomy specimens*Path:*entrapped tubules in sclerotic center surrounded by a corona of contracted ducts + lobules (sclerosing adenosis) and papillomatosis*Histo:*central core of elastosis (= acellular connective tissue and abundant deposits of elastin); one / more ducts obliterated by connective tissue*May be associated with:*tubular carcinoma, comedo carcinoma, invasive lobular carcinoma + contralateral breast cancer◊Avoid frozen section! ● rarely palpable√ mean diameter of 0.33 cm (range, 0.1-0.6 cm)√ irregular noncalcified mass often with architectural distortion√ variable appearance in different projections√ oval / circular translucent areas at center√ very thin long spicules, clumped together centrally√ radiolucent linear structures paralleling spicules√ no skin thickening / retraction*Rx:*surgical excision required for definite diagnosis*DDx:*carcinoma, postsurgical scar, fat necrosis, fibromatosis, granular cell myoblastoma

Notes:

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SARCOMA OF BREAST
*Incidence:*1% of malignant mammary lesions*Age:*45-55 years*Histo:*fibrosarcoma, rhabdomyosarcoma, osteogenic sarcoma, mixed malignant tumor of the breast, malignant fibrosarcoma and carcinoma, liposarcoma ● rapid growth√ smooth / lobulated large dense mass√ well-defined outline√ palpated size similar to mammographic size

Angiosarcoma

Notes:

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Angiosarcoma =highly malignant vascular breast tumor*Incidence:*200 cases in world literature; 0.04% of all malignant breast tumors; 8% of all breast sarcomas*Age:*3rd-4th decade of life*Histo:*hyperchromatic endothelial cells; network of communicating vascular spacesstage I:cells with large nucleolistage II:endothelial lining displaying tufting + intraluminal papillary projectionsstage III:mitoses, necrosis, marked hemorrhage*Metastasis:*hematogenous spread to lung, skin, subcutaneous tissue, bone, liver, brain, ovary; NOT lymphatic ● rapidly enlarging painless immobile breast mass√ skin thickening + nipple retraction√ large solitary mass with ill-defined nonspiculated border US: √ well-defined multilobulated hypoechoic mass with hyperechoic areas (from hemorrhage)*Prognosis:*1.9-2.1 years mean survival; 14% overall 3-year survival rate*Rx:*simple mastectomy without axillary lymph node dissection*DDx:*phyllodes tumor, lactating breast, juvenile hypertrophy◊ Frequently misdiagnosed as lymphangioma / hemangioma!

Notes:

Classification Of CHD

	Acyanotic	Cyanotic
Increased PBF + increased CT ratio	*L-R shunts* VSD ASD PDA ECD PAPVR	*T-lesions* Transposition Truncus arteriosus TAPVR "Tingles" (single ventricle / atrium) Tricuspid atresia (without RVOT obstruction)
Normal PBF + normal CT ratio	*L V outflow obstruction* AS Coarctation Interrupted aortic arch Hypoplastic left heart PS *L V inflow obstruction* Obstructed TAPVR Cor triatriatum Pulmonary vein atresia Congenital MV stenosis *Muscle disease* Cardiomyopathy Myocarditis Anomalous LCA	
Decreased PBF + normal CT ratio Cardiomegaly		*VSD present* Tetralogy of Fallot Tricuspid atresia (with PS + nonrestrictive ASD) Pulmonary atresia + VSD *Intact ventricular septum*

Presenting Age In CHD

AGE	SEVERE PVH	PVH + SHUNT VASCULARITY
0 – 2 days	Hypoplastic left heart Aortic atresia TAPVR below diaphragm Myocardiopathy in IDM	Hypoplastic left heart TAPVR above diaphragm Complete transposition
3 – 7 days		PDA in preterm infant
7 – 14 days	CoA + VSD / PDA Aortic valve stenosis Peripheral AVM Endocardial fibroelastosis Anomalous left coronary artery	Coarctation of aorta (CoA) AVM

Notes:

Incidence Of CHD In Liveborn Infants *Overall incidence:*8-9:1000 livebirths ● most common CHD: mitral valve prolapse (5-20%), bicuspid aortic valve (2%) [usually not recognized before late infancy / childhood] ● ASD + VSD + PDA account for 45% of all CHD ● 12 lesions account for 89% of all CHDVentricular septal defect30.3%Patent ductus arteriosus8.6%Pulmonary stenosis7.4%Septum secundum defect6.7%Coarctation of aorta5.7%Aortic stenosis5.2%Tetralogy of Fallot5.1%Transposition4.7%Endocardial cushion defect3.2%Hypoplastic right ventricle2.2%Hypoplastic left heart1.3%TAPVR1.1%Truncus arteriosus1.0%Single ventricle0.3%Double outlet right ventricle0.2%
High-risk pregnancy: (1) Previous sibling with CHD:2- 5%(2) Previous 2 siblings with CHD:10-15%(3) One parent with CHD:2-10%
Most common causes for CHF + PVH in neonate: 1.Left ventricular failure due to outflow obstruction2.Obstruction of pulmonary venous return

Notes:

CHD With Relatively Long Life Congenital lesions compatible with a relative long life are: 1.Mild tetralogy: mild pulmonic stenosis + small VSD2.Valvular pulmonic stenosis: with relatively normal pulmonary circulation3.Transposition of great vessels: some degree of pulmonic stenosis + large VSD4.Truncus arteriosus: delicate balance between systemic + pulmonary circulation5.Truncus arteriosus type IV: large systemic collaterals6.Tricuspid atresia + transposition + pulmonic stenosis7.Eisenmenger complex8.Ebstein anomaly9.Corrected transposition without intracardiac shunt

Notes:

Juxtaposition Of Atrial Appendages 1.Tricuspid atresia with transposition2.Complete transposition3.Corrected transposition of great arteries4.DORV

Notes:

Continuous Heart Murmur 1.PDA2.AP window3.Ruptured sinus of Valsalva aneurysm4.Hemitruncus5.Coronary arteriovenous fistula

Notes:

Congestive Heart Failure & Cardiomegaly *mnemonic:*"Ma McCae & Co."**M**yocardial infarction **a**nemia **M**alformation **c**ardiomyopathy **C**oronary artery disease **a**ortic insufficiency **e**ffusion **Co**arctation

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Notes:

Congenital Cardiomyopathy *mnemonic:*"CAVE G"**C**ystic medial necrosis of coronary arteries **A**berrant left coronary artery **V**iral **E**ndocardial fibroelastosis **G**lycogen storage disease (Pompe)

Notes:

Neonatal Cardiac Failure A.OBSTRUCTIVE LESIONS1.Coarctation of the aorta2.Aortic valve stenosis3.Asymmetrical septal hypertrophy / hypertrophic obstructive cardiomyopathyB.VOLUME OVERLOAD1.Congenital mitral valve incompetence2.Corrected transposition with left (= tricuspid) AV valve incompetence3.Congenital tricuspid insufficiency4.Ostium primum ASDC.MYOCARDIAL DYSFUNCTION / ISCHEMIA1.Nonobstructive cardiomyopathy2.Anomalous origin of LCA from pulmonary trunk3.Primary endocardial fibroelastosis4.Glycogen storage disease (Pompe disease)5.MyocarditisD.NONCARDIAC LESIONS1.AV fistulas: hemangioendothelioma of liver, AV fistula of brain, vein of Galen aneurysm, large pulmonary AV fistula2.Transient tachypnea of the newborn3.Intraventricular / subarachnoid hemorrhage4.Neonatal hypoglycemia (low birth weight, infants of diabetic mothers)5.Thyrotoxicosis (transplacental passage of LATS hormone)

Notes:

Syndromes With CHD ***5 p - (Cri-du-chat) Syndrome*** *Incidence of CHD:*20%
DiGeorge Syndrome =congenital absence of thymus + parathyroid glands1.Conotruncal malformation2.Interrupted aortic arch
Down Syndrome = MONGOLISM = TRISOMY 21 1.Endocardial cushion defect (25%)2.Membranous VSD3.Ostium primum ASD4. AV communis5.Cleft mitral valve6.PDA7.11 rib pairs (25%)8.Hypersegmented manubrium (90%)
Ellis-van Creveld Syndrome *Incidence of CHD:*50% ● polydactyly ● single atrium
Holt-Oram Syndrome = UPPER LIMB-CARDIAC SYNDROME *Incidence of CHD:*50%1.ASD2.VSD3.Valvular pulmonary stenosis4.Radial dysplasia
Hurler Syndrome Cardiomyopathy
Ivemark Syndrome *Incidence of CHD:*100% ● asplenia ● complex cardiac anomalies
Klippel-Feil Syndrome *Incidence of CHD:*5%1.Atrial septal defect2.Coarctation
Marfan Syndrome = ARACHNODACTYLY 1.Aortic sinus dilatation2.Aortic aneurysm3.Aortic insufficiency4.Pulmonary aneurysm
Noonan Syndrome 1.Pulmonary stenosis2.ASD3.Hypertrophic cardiomyopathy
Osteogenesis Imperfecta 1.Aortic valve insufficiency2.Mitral valve insufficiency3.Pulmonic valve insufficiency
Postrubella Syndrome ● low birth weight ● deafness ● cataracts ● mental retardation1.Peripheral pulmonic stenosis2.Valvular pulmonic stenosis3.Supravalvular aortic stenosis4.PDA
Trisomy 13-15 VSD, tetralogy of Fallot, DORV
Trisomy 16-18 VSD, PDA, DORV
Turner Syndrome ***(XO)*** = OVARIAN DYSGENESIS Incidence of CHD:35%1.Coarctation of the aorta (in 15%)2.Bicuspid aortic valve3.Dissecting aneurysm of aorta
Williams Syndrome = IDIOPATHIC HYPERCALCEMIA ● peculiar elfinlike facies ● mental + physical retardation ● hypercalcemia (not in all patients)1.Supravalvular aortic stenosis (33%)2.ASD, VSD3.Valvular + peripheral pulmonary artery stenosis4.Aortic hypoplasia, stenoses of more peripheral arteries

Notes:

Evaluation Of L-to-R Shunts A.AGE-Infants:(1)Isolated VSD(2)VSD with CoA / PDA / AV canal(3)PDA(4)Ostium primum-Children / adults:(1)ASD(2)Partial AV canal with competent mitral valve(3)VSD / PDA with high pulmonary resistance(4)PDA without murmurB.SEX99% chance for ASD / PDA in female patient C.CHEST WALL ANALYSIS√ 11 pair of ribs + hypersegmented manubrium:Down syndrome √ pectus excavatum + straight back:prolapsing mitral valve D.CARDIAC SILHOUETTE√ absent pulmonary trunk:corrected transposition with VSD; pink tetralogy √ left-sided ascending aorta:corrected transposition with VSD √ tortuous descending aorta:aortic valve incompetence + ASD √ huge heart:persistent complete AV canal (PCAVC); VSD + PDA; VSD + mitral valve incompetence √ enlarged left atrium:intact atrial septum; mitral regurgitation (endocardial cushion defect, prolapsing mitral valve + ASD)
DIFFERENTIAL DIAGNOSIS OF L-R SHUNTS
RARVPALALVProx. Ao
ASDincincincnlnlnlVSDnlincincincincnlPDAnlnlincincincoften inc

Shunt With Normal Left Atrium A.Precardiac shunt1.Anomalous pulmonary venous connectionB.Intracardiac shunt1.ASD (8%)2.VSD (25%)C.Postcardiac shunt1.PDA (12%)

Aortic Size In Shunts A.Extracardiac shunts√ aorta enlarged + hyperpulsatile1.PDAB.Pre- and intracardiac shunts√ aorta small but not hypoplastic1.Anomalous pulmonary venous return2.ASD3.VSD4.Common AV canal

Notes:

Abnormal Heart Chamber Dimensions A.LEFT VENTRICULAR VOLUME OVERLOAD1.VSD2.PDA3.Mitral incompetence4.Aortic incompetenceB.LEFT VENTRICULAR HYPERTROPHY1.Coarctation2.Aortic stenosisC.RIGHT VENTRICULAR VOLUME OVERLOAD1.ASD2.Partial APVR / total APVR3.Tricuspid insufficiency4.Pulmonary insufficiency5.Congenital / acquired absence of pericardium[6.Ebstein anomaly] - not truly RVD.RIGHT VENTRICULAR HYPERTROPHY1.Pulmonary valve stenosis2.Pulmonary hypertension3.Tetralogy of Fallot4.VSDE.Fixed subvalvular aortic stenosisF.Hypoplastic left / right ventricle, common ventricleG. Congestive cardiomyopathy

Notes:

Cardiomegaly In Newborn A.NONCARDIOGENIC1.Metabolic:(a)ion imbalance in serum levels of sodium, potassium, and calcium(b)hypoglycemia2.Decreased ventilation(a)asphyxia(b)transient tachypnea(c)perinatal brain damage3.Erythrocyte function(a)anemia(b)erythrocythemia4.Endocrine(a)glycogen storage disease(b)thyroid disease: hypo- / hyperthyroidism5.Infant of diabetic mother6.Arteriovenous fistula(a)vein of Galen aneurysm(b)hepatic angioma(c)chorioangiomaB.CARDIOGENIC1.Arrhythmia2.Myo- / pericarditis3.Cardiac tumor4.Myocardial infarction5.Congenital heart disease

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Notes:

CYANOTIC HEART DISEASE

Chemical cyanosis=PaO_2 ≤94%Clinical cyanosis=PaO_2 ≤85%◊Decrease in hemoglobin delays detectability!Most common cause of cyanosis -in newborn is transposition of great vessels-in child is tetralogy of Fallot!

A.OVERCIRCULATION VASCULARITY*mnemonic:* "5 Ts + CAD"1.**T**ransposition, complete2.**T**ricuspid atresia with transposition3.**T**runcus arteriosus4.**T**APVR above diaphragm5.**T**ingle ventricle6. **C**ommon atrium7.**A**ortic atresia8.**D**ORVB.DECREASED VASCULARITY (with R-to-L shunt)(a)at ATRIAL LEVEL1.Isolated pulmonary stenosis / atresia2.Tricuspid atresia without transposition with pulmonary stenosis3.Ebstein / Uhl malformation4.Congenital tricuspid regurgitation5.Pericardial effusion(b)at VENTRICULAR LEVEL1.Tetralogy of Fallot2.Single ventricle3.Tricuspid atresia without transposition without pulmonary stenosis4.DORV5.Asplenia syndrome6.Corrected transposition + VSDC.PULMONARY VENOUS HYPERTENSION1.Atresia of common pulmonary vein2.TAPVR below diaphragm3.Aortic atresia

N.B.:tricuspid atresia = the great mimicker

Increased Pulmonary Blood Flow With Cyanosis Decreased Pulmonary Blood Flow With Cyanosis

Notes:

Increased Pulmonary Blood Flow With Cyanosis = ADMIXTURE LESIONS = bidirectional shunt with 2 components: (a)mixing of saturated blood (L-R shunt) and unsaturated blood (R-L shunt)(b)NO obstruction to pulmonary blood flow
Evaluation process: √ PA segment absent = transposition√ PA segment present:(a)L atrium normal (= extracardiac shunt)= TAPVR(b)L atrium enlarged (= intracardiac shunt)= truncus arteriosus
N.B.:Overcirculation + cyanosis = complete transposition until proven otherwise!
ADMIXTURE LESIONS = T-LESIONS *mnemonic:*"5 **T**s + **CAD**"**T**ransposition of great vessels = complete TGV ± VSD (most common cause for cyanosis in neonate) **T**ricuspid atresia with or without transposition + VSD (2nd most common cause for cyanosis in neonate) **T**runcus arteriosus **T**otal anomalous pulmonary venous return (TAPVR) above diaphragm (a) supracardiac (b) cardiac (coronary sinus / right atrium) "**T**ingle" = single ventricle **C**ommon atrium **A**ortic atresia **D**ouble-outlet right ventricle (DORV type I) / Taussig-Bing anomaly (DORV type II) *Clues:* √ skeletal anomalies: Ellis-van Creveld syndrome (truncus / common atrium)√ polysplenia: common atrium√ R aortic arch: persistent truncus arteriosus√ ductus infundibulum: aortic atresia√ pulmonary trunk seen: supracardiac TAPVR; DORV; tricuspid atresia; common atrium√ ascending aorta with leftward convexity: single ventricle√ dilated azygos vein: common atrium + polysplenia + interrupted IVC; TAPVR to azygos vein√ left-sided SVC: vertical vein of TAPVR√ "waterfall" right hilum: single ventricle + transposition√ large left atrium (rules out TAPVR)√ prominent L heart border: single ventricle with inverted rudimentary R ventricle; levoposition of R atrial appendage (tricuspid atresia + transposition)√ age of onset ≤2 days: aortic atresia

Notes:

Decreased Pulmonary Blood Flow With Cyanosis =two components of (a) impedance of blood flow through right heart due to obstruction / atresia at pulmonary valve / infundibulum (b) R-to-L shunt; pulmonary circulation maintained through systemic arteries / PDA
mnemonic:"P2 TETT"**P**ulmonic stenosis with ASD **P**ulmonic atresia **T**etralogy of Fallot **E**bstein anomaly **T**ricuspid atresia with pulmonic stenosis **T**ransposition of great vessels with pulmonic stenosis
A.SHUNT AT VENTRICULAR LEVEL1.Tetralogy of Fallot2.Tetralogy physiology (associated with pulmonary obstruction):-Complete / corrected transposition-Single ventricle-DORV-Tricuspid atresia (PS in 75%)-Asplenia syndrome√ prominent aorta with L / R aortic arch; inapparent pulmonary trunk√ NORMAL R atrium (without tricuspid regurgitation)√ NORMAL-sized heart (secondary to escape mechanism into aorta)*Clues:* 1.Skeletal anomaly (eg, scoliosis): tetralogy (90%)2.Hepatic symmetry: asplenia3.Right aortic arch: tetralogy, complete transposition, tricuspid atresia4.Aberrant right subclavian artery: tetralogy5.Leftward convexity of ascending aorta: single ventricle with inverted right rudimentary ventricle, corrected transposition, asplenia, JAA (tricuspid valve atresia)B.SHUNT AT ATRIAL LEVEL1.**P**ulmonary stenosis / atresia with intact ventricular septum2.**E**bstein malformation + Uhl anomaly3.**T**ricuspid atresia (ASD in 100%)√ moderate to severe cardiomegaly√ R atrial dilatation√ R ventricular enlargement (secondary to massive tricuspid incompetence)√ inapparent aorta√ left aortic arch

Notes:

Increased Pulmonary Blood Flow Without Cyanosis =indicates L-R shunt with increased pulmonary blood flow (shunt volume >40%)A.WITH LEFT ATRIAL ENLARGEMENTIndicates shunt distal to mitral valve = increased volume without escape defect 1.VSD (25%): small aorta in intracardiac shunt2.PDA (12%): aorta + pulmonary artery of equal size in extracardiac shunt3.Ruptured sinus of Valsalva aneurysm (rare)4.Coronary arteriovenous fistula (very rare)5.Aortopulmonary window (extremely rare)B.WITH NORMAL LEFT ATRIUMIndicates shunt proximal to mitral valve = volume increased with escape mechanism through defect 1.ASD (8%)2.Partial anomalous pulmonary venous return (PAPVR) + sinus venosus ASD3.Endocardial cushion defect (ECD) (4%)

Notes:

Normal Pulmonary Blood Flow Without Cyanosis A.OBSTRUCTIVE LESION(a)Right ventricular outflow obstruction1.at level of pulmonary valve: subvalvular / valvular / supravalvular pulmonic stenosis2.at level of peripheral pulmonary arteries:peripheral pulmonary stenosis (b)Left ventricular inflow obstruction1.at level of peripheral pulmonary veins:pulmonary vein stenosis / atresia 2.at level of left atrium: cor triatriatum3.at level of mitral valve:supravalvular mitral stenosis, congenital mitral stenosis / atresia, "parachute" mitral valve (c)Left ventricular outflow obstruction1.at level of aortic valve:anatomic subaortic stenosis, functional subaortic stenosis (IHSS), valvular aortic stenosis, hypoplastic left heart, supravalvular aortic stenosis 2.at level of aorta:interruption of aortic arch, coarctation of aorta B.CARDIOMYOPATHY1.Endocardial fibroelastosis2.Hypertrophic cardiomyopathy3.Glycogen storage diseaseC.HYPERDYNAMIC STATE1.Noncardiac AVM (cerebral AVM, vein of Galen aneurysm, large pulmonary AVM, hemangioendothelioma of liver)2.Thyrotoxicosis3.Anemia4.PregnancyD.MYOCARDIAL ISCHEMIA1.Anomalous left coronary artery2.Coronary artery disease (CAD)

Notes:

Increased Pulmonary Vasculature A.OVERCIRCULATION=shunt vascularity = arterial + venous overcirculation(a)Congenital heart disease (most common)(1)L-R shunts (2) Admixture cyanotic lesions(b)High-flow syndromes(1) Thyrotoxicosis (2) Anemia (3) Pregnancy (4) Peripheral arteriovenous fistula √ diameter of right descending pulmonary artery larger than trachea just above aortic knob √ increased size of veins + arteries with size larger than accompanying bronchus (= "kissing cousin" sign), best seen just above hila on AP view √ enlarged hilar vessels (lateral view) √ visualization of vessels below 10th posterior ribB.PULMONARY VENOUS HYPERTENSION √ redistribution of flow (not seen in younger children) √ indistinctness of vessels with Kerley lines(= interstitial edema) √ alveolar edema √ fine reticulated patternC.PRECAPILLARY HYPERTENSION √ enlarged main + right and left pulmonary arteries √ abrupt tapering of pulmonary arteriesD.PROMINENT SYSTEMIC / AORTOPULMONARY COLLATERALS1.Tetralogy of Fallot with pulmonary atresia(= pseudotruncus) 2.VSD + pulmonary atresia (single ventricle, complete transposition, corrected transposition)3.Pulmonary-systemic collaterals √ coarse vascular pattern with irregular branching arteries (from aorta / subclavian arteries) √ small central vessels despite apparent increase in vascularity

Notes:

Decreased Pulmonary Vascularity =obstruction to pulmonary flow√ vessels reduced in size and number√ hyperlucent lungs√ small pulmonary artery segment + hilar vessels

Notes:

Normal Pulmonary Vascularity & Normal-sized Heart *mnemonic:*"MAN"**M**yocardial ischemia **A**fterload (= pressure overload problems) **N**ormal

Notes:

Pulmonary Arterial Hypertension =PAH = sustained pulmonary arterial pressure in systole >30 mm Hg, in diastole >15 mm Hg, mean pressure >20 mm Hg secondary to reduction in cross-sectional area of pulmonary vascular bed with concomitant increase in pulmonary vascular resistance *Pathogenesis:* A.PRIMARY PAH (rare) = plexogenic pulmonary arteriopathy = unknown cause / mechanismB.SECONDARY PAH (more common)(a)primary pleuropulmonic disease1.Parenchymal pulmonary disease= **cor pulmonale**: COPD, emphysema, chronic bronchitis, asthma, bronchiectasis, malignant infiltrate, granulomatous disease, cystic fibrosis, end-stage fibrotic lung, S/P lung resection, idiopathic hemosiderosis, alveolar proteinosis, alveolar microlithiasis 2.Alveolar hypoventilation= hypoxic pulmonary arterial hyperperfusion: chronic high altitude, sleep apnea, hypoventilation due to neuromuscular disease / obesity 3.Pleural disease + chest deformity fibrothorax, thoracoplasty, kyphoscoliosis(b)primary vascular disease1.Congenital heart disease-increased flow: large L-R shunt (Eisenmenger syndrome)-decreased flow: tetralogy of Fallot2.Capillary obliteration: chronic pulmonary thromboembolism, persistent fetal circulation, arteritides (eg, Takayasu)3.Venous obliteration: pulmonary venoocclusive disease(c)pulmonary venous hypertension
Histo: GradeI=hypertrophy of media of muscular pulmonary arteries + arteriolesGradeII=hypertrophy of muscle cells + proliferation of intima cells in small muscular arteries + arteriolesGradeIII=muscular hypertrophy + intimal thickening + subendothelial fibrosisGradeIV=occlusion of vessels with progressive dilatation of small arteries nearby; muscular hypertrophy less apparentGradeV=tortuous channels within proliferation of endothelial cells (= plexiform + angiomatoid lesions) + intraalveolar macrophagesGradeVI=thrombosis + necrotizing arteritis
√ "pruning" of pulmonary arteries = disproportionate increase in caliber of central fibrous arteries + decrease in caliber of smaller muscular arteries (from sustained increase in pressure)√ increase in vessel caliber of central + peripheral arteries (from sustained increase in flow by a factor of >2)√ calcification of central pulmonary vessels (PATHOGNOMONIC)√ NO increase of pulsations in middle third of lung√ normal-sized heart / right heart enlargement

Notes:

Cor Pulmonale *mnemonic:*"TICCS BEV"**T**horacic deformity **I**diopathic **C**hronic pulmonary embolism **C**OPD **S**hunt (ASD, VSD, etc) **B**ronchiectasis **E**mphysema **V**asculitis

Notes:

Pulmonary Venous Hypertension =INCREASED VENOUS PULMONARY PRESSURE=VENOUS CONGESTION=pulmonary capillary wedge pressure (PCWP) >15 mm Hg*Cause:* A.LEFT VENTRICULAR INFLOW TRACT OBSTRUCTION√ normal-sized heart with right ventricular hypertrophy√ prominent pulmonary trunk@proximal to mitral valve:√ normal-sized left atrium1.TAPVR below the diaphragm2.Primary pulmonary veno-occlusive disease3.Stenosis of individual pulmonary veins4.Atresia of common pulmonary vein5.Cor triatriatum6.Left atrial tumor / clot7.Supravalvular ring of left atrium8.Fibrosing mediastinitis9.Constrictive pericarditis@at mitral valve level√ enlarged left atrium1.Rheumatic mitral valve stenosis ± regurgitation (99%)√ enlarged left atrial appendage2.Congenital mitral valve stenosis3.Parachute mitral valve (= single bulky papillary muscle)B.LEFT VENTRICULAR FAILURE(a)ABNORMAL PRELOAD with secondary mitral valve incompetence (= volume overload)1.Aortic valve regurgitation2.Eisenmenger syndrome (= R-to-L shunt in VSD)3.High-output failure:noncardiac AVM (cerebral AVM, vein of Galen aneurysm, large pulmonary AVM, hemangioendothelioma of liver, iatrogenic), thyrotoxicosis, anemia, pregnancy (b)ABNORMAL AFTERLOAD(= pressure overload) = LV outflow tract obstruction 1.Hypoplastic left heart syndrome2.Aortic stenosis (supravalvular, valvular, anatomic subaortic)3.Interrupted aortic arch4.Coarctation of the aorta(c)DISORDERS OF CONTRACTION AND RELAXATION1.Endocardial fibroelastosis2.Glycogen storage disease (Pompe disease)3.Cardiac aneurysm4.Cardiomyopathy(a)congestive (alcohol)(b)hypertrophic obstructive cardiomyopathy (HOCM), particularly in IDM- asymmetric septal hypertrophy (ASH) - idiopathic hypertrophic subaortic stenosis (IHSS) (d)MYOCARDIAL ISCHEMIA1.Anomalous left coronary artery2.Coronary artery disease (CAD) √ moderate redistribution (PCWP 13-15 mm Hg)√ redistribution (PCWP 15-18 mm Hg)√ indistinct vessel margins due to interstitial edema (PCWP 18-25 mm Hg)√ alveolar pulmonary edema (PCWP >30 mm Hg)

Notes:

Pulmonary Artery-Bronchus Ratios =ratio of diameters of end-on segmental pulmonary artery + accompanying end-on bronchusA.ERECT CHEST FILM1.Normal (effect of gravity):upper lung zone0.85 ± 0.15lower lung zone 1.34 ± 0.252.Pulmonary plethora (balanced engorgement):upper lung zone1.62 ± 0.31lower lung zone1.56 ± 0.283.Decompensated CHF (redistribution from left-sided CHF):upper lung zone1.50 ± 0.25lower lung zone0.87 ± 0.20B.SUPINE CHEST FILM1.Normal (gravitational effect lost):upper lung zone1.01 ± 0.13lower lung zone1.05 ± 0.132.Decompensated CHF (inverted pattern / plethora pattern):upper lung zone1.49 ± 0.31lower lung zone0.96 ± 0.31

Notes:

Enlarged Aorta A. INCREASED VOLUME LOAD1.Aortic insufficiency2.PDAB. POSTSTENOTIC DILATATION1.Valvular aortic stenosisC.INCREASED INTRALUMINAL PRESSURE1.Coarctation2.Systemic hypertensionD.MURAL WEAKNESS / INFECTION1.Cystic media necrosis: Marfan / Ehlers-Danlos syndrome2.Congenital aneurysm3.Syphilitic aortitis4.Mycotic aneurysm5.Atherosclerotic aneurysm (compromised vasa vasorum)E.LACERATION OF AORTIC WALL1.Traumatic aneurysm2.Dissecting hematoma

Notes:

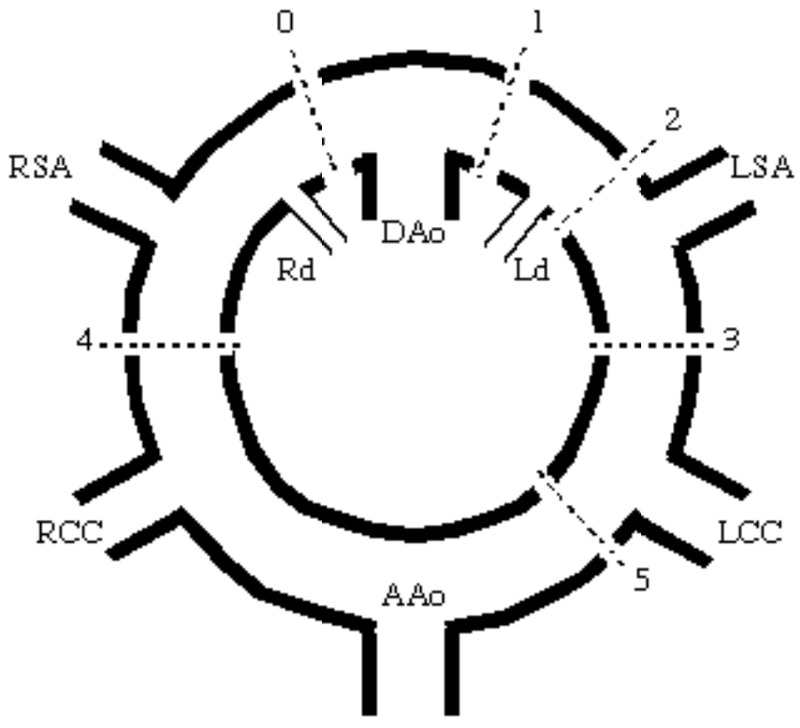
Aortic Wall Thickening 1.Intramural hematoma= aortic dissection without intimal tear 2.Aortitissegments of aortic arch + branch vessels 3.Atherosclerotic plaque irregular narrowing of aortic lumen4.Adherent thrombus

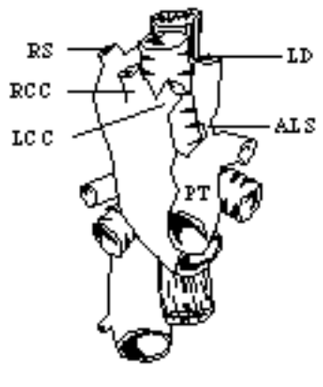
Notes:

Double Aortic Arch Common cause of vascular ring; usually isolated condition *Incidence:*55% of all vascular rings*Age:*usually detected in infancy ● usually asymptomatic ● stridor, dyspnea, recurrent pneumonia ● dysphagia (less common than respiratory symptoms, more common after starting baby on solids)Location:descending aorta in 75% on left, in 25% on right side; smaller arch anterior in 80%; right arch larger + more cephalad than left in 80%√ two separate arches arise from single ascending aorta√ each arch joins to form a single descending aorta√ impressions may be present on both sides of trachea: usually R > L √ small anterior tracheal impression√ broad posterior + bilateral esophageal indentationsCT: √ "four-artery sign" = each arch gives rise to 2 dorsal subclavian + 2 ventral carotid arteries evenly spaced around trachea on section cephalad to aortic arch*DDx:*right arch with aberrant left subclavian artery (indistinguishable by esophagram when dominant arch on right side)

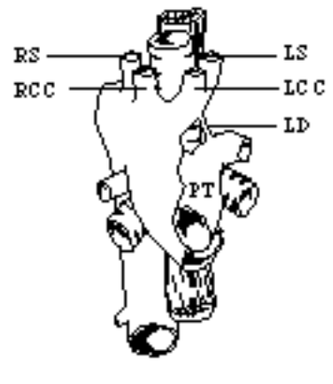
Notes:

Right Aortic Arch *Incidence:*1-2%INCIDENCE OF RIGHT AORTIC ARCH IN CONGENITAL HEART DISEASE 1.Truncus arteriosus35%2.Tetralogy of Fallot25%3.TGV10%4.Tricuspid atresia 5%5.Large VSD 2%Rare anomalies: 1.Corrected transposition50%2.Pseudotruncus50%3.Asplenia30%4.Pink tetralogy15%*mnemonic:* "TRU TETRA TRIC"**TRU**ncus arteriosus **TE**tralogy of Fallot **TRA**nsposition **TRIC**uspid atresia

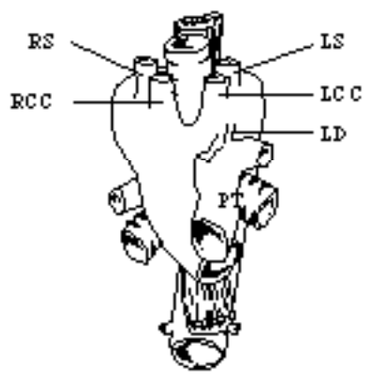




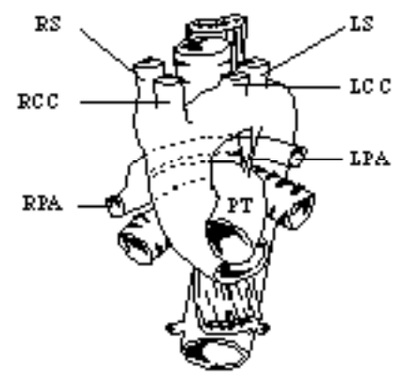
Right Aortic Arch with Aberrant Left Subclavian Artery



Right Aortic Arch with Mirror-image Branching



Double Aortic Arch
ALS = aberrant left subclavian a.
LS = left subclavian a.
LCC = left common carotid a.
LD = left ductus arteriosus
LPA = left pulmonary a.



Aberrant Left Pulmonary Artery
PT = pulmonary trunk
RCC = right common carotid a.
RPA = right pulmonary a.
RS = right subclavian a.

Right Aortic Arch With Aberrant Left Subclavian Artery =RAA with ALSA=interruption of embryonic left arch between left CCA and left subclavian artery; most common type of right aortic arch anomaly: 35-72%; 2nd most common cause of vascular ring after double aortic arch*Incidence:*1:2,500*Associated with:* congenital heart disease in 5-12%:1.Tetralogy of Fallot(2/3 = 8%)2.ASD ± VSD(1/4 = 3%)3.Coarctation(1/12 = 1%) ● usually asymptomatic (loose ring around trachea + esophagus) ● may be symptomatic in infancy / early childhood provoked by bronchitis + tracheal edema ● may be symptomatic in adulthood provoked by torsion of aorta√ left common carotid artery is first branch of ascending aorta√ left subclavian artery arises from descending aorta via the remnant of the left dorsal aortic root√ bulbous configuration of origin of LSA (= remnant of embryonic left arch) = retroesophageal aortic diverticulum = **diverticulum of Kommerell** (N.B.: originally described as diverticular outpouching at origin of right subclavian artery with left aortic arch)√ small rounded density left lateral to trachea√ impression on left side of esophagus simulating a double aortic arch (aortic diverticulum / ligamentum arteriosum)√ vascular ring (= left ductus extends from aortic diverticulum to left pulmonary artery)√ right aortic arch impression on tracheal air shadow√ right-sided esophageal indentation (right arch)√ masslike density silhouetting top of aortic arch just posterior to trachea on LAT CXR√ broad posterior impression on esophagus (left subclavian artery / aortic diverticulum)√ small anterior impression on trachea√ aorta descends on right side

Right Aortic Arch With Mirror-image Branching 2nd most common aortic arch anomaly: 24-60% =interruption of embryonic left arch between left subclavian artery and descending aorta; dorsal to left ductus arteriosus(a)Type 1 = interruption of left aortic arch distal to ductus arteriosus (common)*Associated with:*cyanotic congenital heart disease in 98%:1.Tetralogy of Fallot(87%)2.Multiple defects(7.5%)3.Truncus arteriosus(2-6%)4.Transposition (1-10%)5.Tricuspid atresia(5%)6.ASD ± VSD(0.5%) ◊25% of patients with tetralogy have right aortic arch!◊37% of patients with truncus arteriosus have right aortic arch!√ NO vascular ring, NO retroesophageal component√ NO structure posterior to trachea√ R arch impression on tracheal air shadow√ NORMAL barium swallow(b)Type 2 = interruption of left aortic arch proximal to ductus arteriosus (rare)true vascular ring (if duct persists); rarely associated with CHD

Right Aortic Arch With Isolated Left Subclavian Artery 3rd most common right aortic arch anomaly: 2% =interruption of embryonic left arch between(a) left CCA and left subclavian artery and (b) left ductus and descending aorta resulting in a connection of left subclavian artery with left pulmonary artery*Associated with:*tetralogy of Fallot√ left common carotid artery arises as the first branch√ left subclavian artery attaches to left pulmonary artery through PDA√ NO vascular ring, NO retroesophageal component ● congenital subclavian steal syndrome

Right Aortic Arch With Aberrant Left Brachiocephalic Artery Similar in appearance to R aortic arch + aberrant L subclavian artery

Notes:

Left Aortic aArch ***Left Aortic Arch With Aberrant Right Subclavian Artery*** =right subclavian artery arises as 4th branch from proximal descending aorta*Incidence:*0.4-2.3%; most common congenital aortic arch anomaly; in 37% of Down syndrome children with CHD*Associated with:*(1) Absent recurrent pharyngeal nerve(2) CHD in 10-15%Course: (a) behind esophagus (80%)(b) between esophagus + trachea (15%)(c) anterior to trachea (5%) ● asymptomatic / dysphagia lusoria (rare)√ soft-tissue opacity crossing the esophagus obliquely upward toward the right shoulder√ masslike opacity in right paratracheal region√ rounded opacity arising from superior aortic margin posterior to trachea + esophagus on LAT CXR√ dilated origin of aberrant subclavian artery (in up to 60%) = diverticulum of Kommerell = remnant of embryonic right arch √ unilateral L-sided rib notching (if aberrant right subclavian artery arises distal to coarctation)
Anomalous Innominate Artery Compression Syndrome =origin of R innominate artery to the left of trachea coursing to the right√ anterior tracheal compression

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Notes:

Bovine Aortic Arch =common origin of brachiocephalic trunk + left common carotid artery

Notes:

Cervical Aortic Arch *Associated with:*right aortic arch (in 2/3) ● pulsatile neck mass ● upper airway obstruction ● dysphagia√ mediastinal widening√ absence of normal aortic knob√ aortic arch near lung apex√ tracheal displacement to opposite side + anteriorly√ apparent cutoff of tracheal air column (secondary to crossing of descending aorta to side opposite of arch)*DDx:*carotid aneurysm

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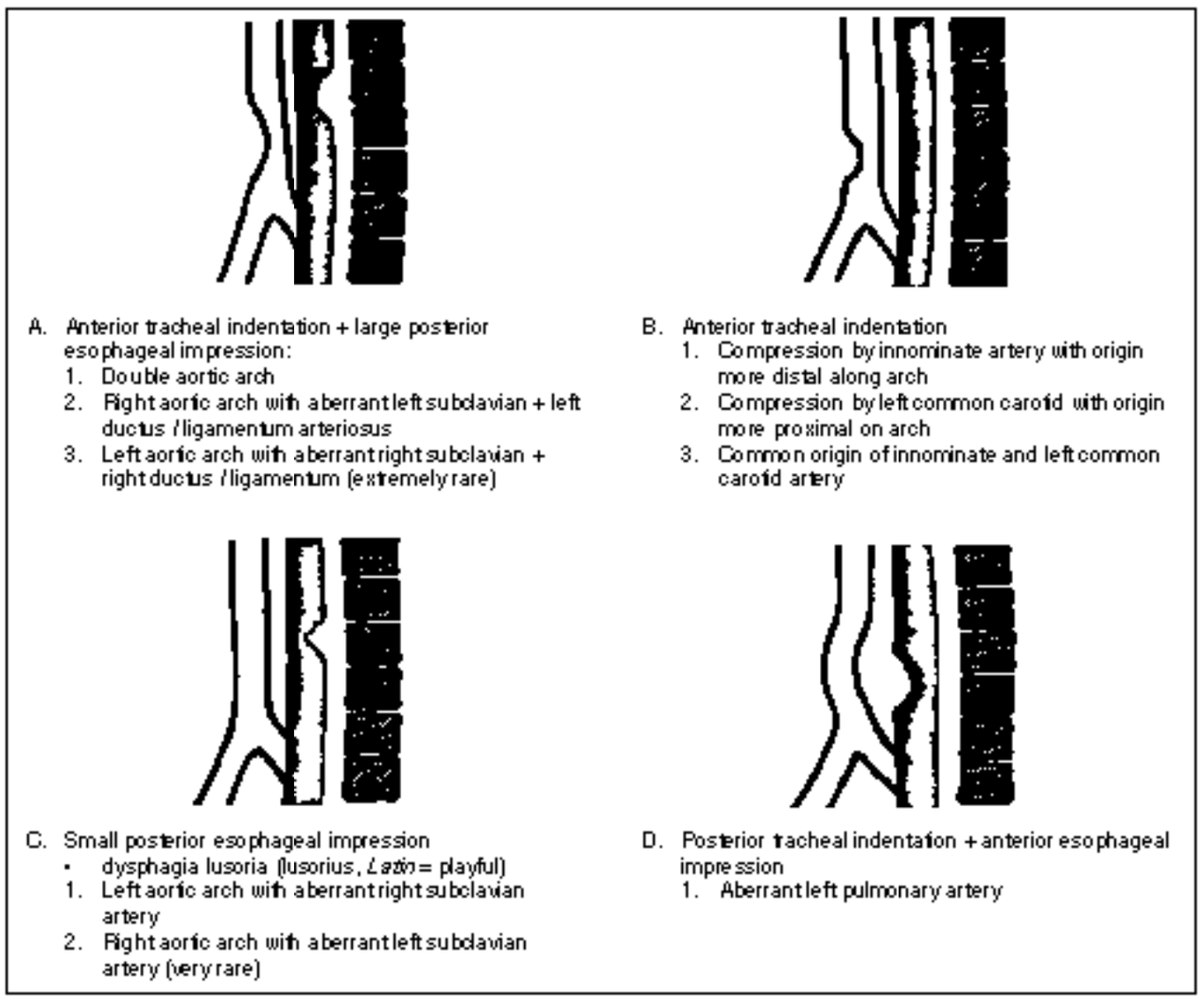
Notes:

Vascular Rings =anomaly characterized by encirclement of trachea + esophagus by aortic arch + branchesA.Usually symptomatic lesions ● chronic stridor, wheezing, recurrent pneumonia ● dysphagia, failure to thrive1.Double aortic arch with R descending aorta + L ductus arteriosus2.R aortic arch with R descending aorta + aberrant L subclavian artery + persistent L ductus / ligamentum teres3.L arch with L descending aorta + R ductus / ligamentum4.Aberrant L pulmonary artery = "pulmonary sling"*Frequency of CXR findings:* -frontal CXR:√ right aortic arch (85%)√ focal indentation of distal trachea (73%)-lateral CXR:√ anterior tracheal bowing (92%)√ increased retrotracheal opacity (79%)√ focal tracheal narrowing (77%)B.Occasionally symptomatic lesions1.Anomalous innominate2.Anomalous L common carotid artery / common trunk3.R aortic arch with L descending aorta + L ductus / ligamentumC.Usually asymptomatic lesions1.L aortic arch + aberrant R subclavian artery2.L aortic arch with R descending aorta3.R aortic arch with R descending aorta + mirror-image branching4.R aortic arch with R descending aorta + aberrant L subclavian artery5.R aortic arch with R descending aorta + isolation of L subclavian artery6.R aortic arch with L descending aorta + L ductus / ligamentum

Pattern of vascular compression of esophagus and trachea



Notes:

Aortic Stenosis A.ACQUIRED1.Takayasu aortitis2.Radiation aortitis3.Aortic dissection4.Infected aortic aneurysm with abscess5.Pseudoaneurysm from laceration6.Atherosclerosis (rare)7.Syphilitic aortitis (rare)B.CONGENITAL1.Williams syndrome2.Neurofibromatosis3.Rubella4.Mucopolysaccharidosis5.Hypoplastic left heart syndrome

Notes:

Abnormal Left Ventricular Outflow Tract LVOT = area between IVS + aML from aortic valve cusps to mitral valve leaflets 1.Membranous subaortic stenosis=crescent-shaped fibrous membrane extending across LVOT + inserting at aML√ diffuse narrowing of LVOT√ abnormal linear echoes in LVOT space (occasionally)2.Prolapsing aortic valve vegetation3.Narrowed LVOT (<20 mm)(a)Long-segment subaortic stenosis√ aortic valve closure in early systole with coarse fluttering√ high-frequency flutter of mitral valve in diastole (aortic regurgitation)√ symmetric LV hypertrophy(b)ASH / IHSS√ asymmetrically thickened septum bulging into LV + LVOT√ systolic anterior motion of aML (SAM)(c)Mitral stenosis(d)Endocardial cushion defect

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Notes:

Invisible Main Pulmonary Artery A.Underdeveloped = RVOT obstruction1.Tetralogy of Fallot2.Hypoplastic right heart syndrome (tricuspid / pulmonary atresia)B.Misplaced pulmonary artery1.Complete transposition of great vessels2.Persistent truncus arteriosus

Notes:

Unequal Pulmonary Blood Flow 1.Tetralogy of Fallot√ diminished flow on left side (hypoplastic / stenotic pulmonary artery in 40%)2.Persistent truncus arteriosus (esp. Type IV)√ diminished / increased blood flow to either lung3.Pulmonary valvular stenosis√ increased flow to left lung secondary to jet phenomenon

Notes:

Dilatation Of Pulmonary Trunk 1.Idiopathic dilatation of pulmonary artery2.Pulmonic valve stenosis√ poststenotic dilatation of trunk + left pulmonary a.3.Pulmonary regurgitation(a)severe pulmonic valve insufficiency(b)absence of pulmonic valve (may be associated with tetralogy)4.Congenital L-to-R shunts5.Pulmonary arterial hypertension6.Aneurysm: mycotic / traumatic

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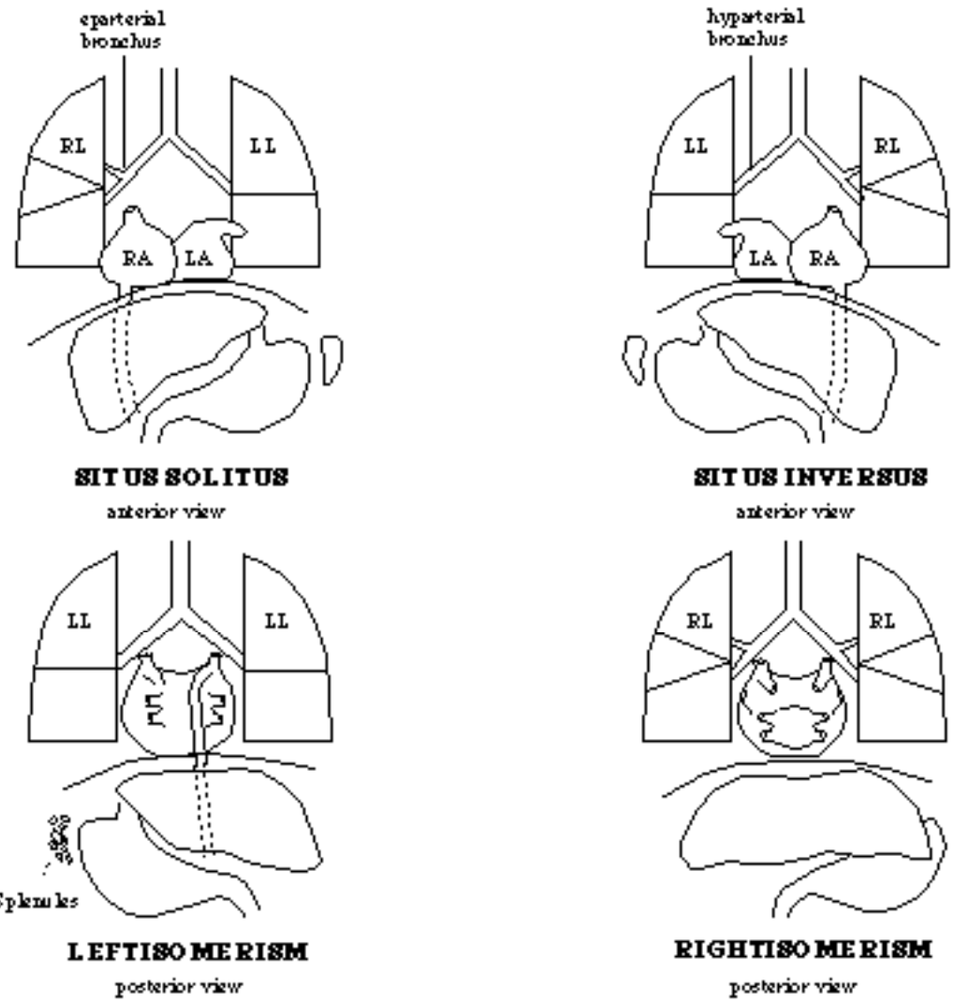
Notes:

SITUS
=term describing the position of atria, tracheobronchial tree, pulmonary arteries, thoracic + abdominal viscera



A.SITUS SOLITUS = normal situs=position of morphologic LA is the same as that of the aortic arch + stomach bubble + hyparterial bronchus + bilobed lung; the position of the morphologic RA is the same as that of the eparterial bronchus + trilobed lung1.Abdominal situs solitus√ liver + IVC are right-sided√ stomach, spleen, abdominal aorta are left-sided2.Cardiac situs solitus√ morphologic right atrium is right-sided√ morphologic left atrium is left-sided*Associated with:* (a)levocardia:<1% chance for CHD(b)dextrocardia:95% chance for CHDB.SITUS INVERSUS=mirror-image position of normal1.Abdominal situs inversus√ mirror-image position of abdominal organs2.Cardiac situs inversus√ morphologic right atrium is left-sided√ morphologic left atrium is right-sided*Associated with:* (a)dextrocardia = situs inversus totalis (usual variant): 3-5% chance for CHD, eg, Kartagener syndrome(b)levocardia (extremely rare): 95% chance for CHD
C.SITUS INDETERMINATUS / INDETERMINUS / AMBIGUUS=ambiguous relationship1.Abdominal situs ambiguus√ liver may be midline + symmetric√ bowel malrotations are typical2.Cardiac situs ambiguus√ atrial morphology indeterminate / bilateral right atria (right atrial isomerism) / bilateral left atria (left atrial isomerism)*Associated with:* (a)bilateral right isomerism / sidedness = asplenia syndrome(b)bilateral left isomerism / sidedness = polysplenia syndrome

Notes:

HETEROTAXIA

= CARDIOSPLENIC SYNDROMES = sporadic disorders with abnormal relationship between abdominal organs + tendency toward symmetric development of organs within trunk + associated cardiac anomolies

	Asplenia bilateral R sidedness	Polysplenia bilateral L sidedness
CLINICAL		
Presenting age	newborn / infant	infant / adult
Sex predominance	male	female
Cyanosis	severe	usually absent
Heart disease	severe	moderate / none (5 – 10%)
Howell-Jolly / Heinz bodies	present	absent
Spleen scan	no spleen	multiple small spleens
Characteristic ECG	none	abnormal P wave vector
Prognosis	poor	good
Mortality	high	low
PLAIN FILM		
Lung vascularity	decreased	normal / increased
Aortic arch	right / left	right / left
Cardiac apex	right / left / midline	right / left
Bronchi	bilateral eparterial	bilateral hyparterial
Minor fissure	possibly bilateral	none / normal
Stomach	midline / right / left	right / left
Liver	symmetrical / R / L	in various positions
Malrotation of bowel	yes (microgastria)	yes
CARDIOGRAPHY		
Coronary sinus	usually absent	sometimes absent
Atrial septum	common atrium (100%)	ASD (84%)
AV valve	atresia / common valve	normal / abnormal MV
Single ventricle	44%	infrequent
IVS	VSD	VSD common
Great vessels	d- / l-transposition (72%)	normal relationship
Pulmonary stenosis	the rule	frequent
Pulmonary veins	TAPVR	PAPVR (42%) TAPVR (6%)
Single coronary artery	19%	
SVC	bilateral (53%)	bilateral (33%)
IVC-aorta relationship	same side of spine	normal
IVC	normal	interrupted (84%) / normal
Azygos vein	inapparent	continuation R / L

Cardiac Position

Notes:

Cardiac Position
=determined by base-apex axis; no assumption is made regarding cardiac chamber / vessel arrangement
A.POSITION OF CARDIAC APEX1.Levocardia = apex directed leftward2.Dextrocardia = apex directed rightward3.Mesocardia = vertical / midline heart (usually with situs solitus)"i" atrial septum characteristically bowed into left atrium in cardiac situs solitus with dextrocardia + cardiac situs inversus with levocardia (DDx: juxtapositioned atrial appendages)B.CARDIAC DISPLACEMENTby extracardiac factors (eg, lung hypoplasia, pulmonary mass) 1.Dextropositionsuggests hypoplasia of ipsilateral pulmonary artery (PAPVR implies scimitar syndrome) 2.Levoposition3.MesopositionC.CARDIAC INVERSION=alteration of normal relationship of chambers1.D-bulboventricular loop2.L-bulboventricular loopD.TRANSPOSITION=alteration of anterior-posterior relationship of great vessels

Notes:

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CARDIAC TUMOR
*Prevalence:*0.017-0.08-0.3% ● weight loss, fever, malaise ● congestive heart failure, palpitations, heart murmur ● syncope ● dyspnea, cough, chest painLocation:pericardial, intramural, intracavitary

Malignant Heart Tumors Benign Heart Tumor In Adults Congenital Cardiac Tumor

Notes:

LIPPINCOTT WILLIAMS & WILKINS
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Malignant Heart Tumors *Prevalence:*25% of all cardiac tumors in adults10% of all cardiac tumors in children1.Sarcoma:undifferentiated sarcoma, angiosarcoma, rhabdomyosarcoma2.Malignant fibrous histiocytoma*Prevalence:*1-2% of all primary cardiac tumors*Age:*more common in adults than children3.Metastatic diseasemost commonly lung, melanoma, breast ◊20-40 times more frequent than primary tumor!4.Lymphoma*Incidence:*cardiac involvement in 29% on autopsy; pericardial involvement more frequent ● intractable congestive heart failure ● chest pain√ SVC obstruction5.Malignant teratoma6.Multiple cardiac myxomas

Notes:

Benign Heart Tumor In Adults 1.Myxoma (most common cardiac tumor)2.Papillary fibroelastoma3.Lipoma4.Hydatid cyst (uncommon):√ localized bulge of left cardiac contour√ curvilinear / spotty calcifications (resembling myocardial aneurysm)*Cx:*may rupture into cardiac chamber / pericardium

Notes:

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Congenital Cardiac Tumor *Incidence:* 1:10,000 1.Rhabdomyoma (58%): usually multiple masses2.Teratoma (20%): intrapericardiac, extracardiac√ multicystic mass3.Fibroma (12%): intramuralmay be associated with:Gorlin syndromeLocation:free LV wall / interventricular septum√ may be pedunculated√ calcification and cystic degeneration centrally√ tendency for slow growth*Cx:*fetal hydrops secondary to obstruction, pericardial effusion, fetal arrhythmia, fetal death4.Hemangioma (arise from RT atrium, pericardial effusion, skin hemangiomas), lymphangioma, neurofibroma, myxoma, mesothelioma:√ mass-occupying lesion impinging upon cardiac cavities

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Notes:

Pericardial Effusion =pericardial fluid >50 mL*Etiology:* A.SEROUS FLUID = transudatecongestive heart failure, hypoalbuminemia, irradiation B.BLOOD = hemopericardium(a)iatrogenic: cardiac surgery / catheterization, anticoagulants, chemotherapy(b)trauma: penetrating / nonpenetrating(c)acute myocardial infarction / rupture(d)rupture of ascending aorta / pulmonary trunk(e)coagulopathy(f)neoplasm: mesothelioma, sarcoma, teratoma, fibroma, angioma, metastasis (lung, breast, lymphoma, leukemia, melanoma)C.LYMPHneoplasm, congenital, cardiothoracic surgery, obstruction of hilum / SVC D.FIBRIN = exudate(a)infection: viral, pyogenic, TB(b)uremia: 18% in acute uremia; 51% in chronic uremia; dialysis patient(c)collagen disease: rheumatoid arthritis, SLE, acute rheumatic fever(d)hypersensitivity*mnemonic:*"CUM TAPPIT RV"**C**ollagen vascular disease **U**remia **M**etastasis **T**rauma **A**cute myocardial infarction **P**urulent infection **P**ost MI syndrome **I**diopathic **T**uberculosis **R**heumatoid arthritis **V**irus CXR: √ normal with fluid <250 mL / in acute pericarditis√ "water bottle configuration" = symmetrically enlarged cardiac silhouette√ loss of retrosternal clear space√ "fat-pad sign" = separation of retrosternal from epicardial fat line >2 mm (15%)√ rapidly appearing cardiomegaly + normal pulmonary vascularity√ "differential density sign" = increase in lucency at heart margin secondary to slight difference in contrast between pericardial fluid + heart muscle√ diminished cardiac pulsationsECHO: √ separation of epi- and pericardial echoes extending into diastole (rarely behind LA)√ volume estimates by M-mode:(a)separation only posteriorly = <300 mL(b)separation throughout cardiac cycle = 300-500 mL(c)plus anterior separation = >1000 mL

Notes:

Pneumopericardium *Etiology:*shearing mechanism of injury of the heart during blunt trauma*Path:*tear in fibrous pericardium, usually along the course of the phrenic nerve, allows pneumomediastinal air to enter √ thick shaggy soft-tissue density of fibrous pericardium separated by air from cardiac density√ air limited to distribution of pericardial reflection

Notes:

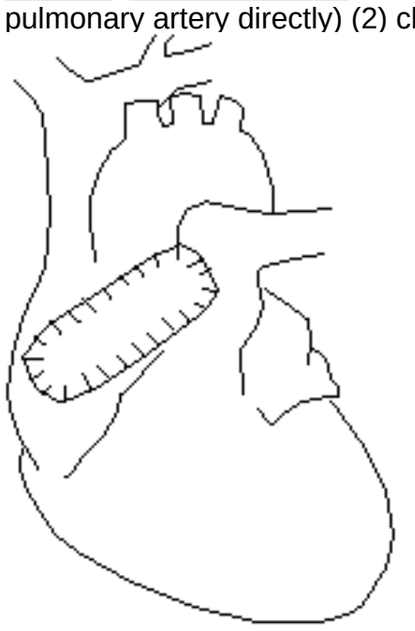
Vena cava anomalies 1.Retrocaval ureter = circumcaval ureter2.Duplicated IVC*Incidence:*0.2-3%*Etiology:*persistence of right + left supracardinal veins√ small / equal-sized left IVC formed by left iliac vein√ crossover to right IVC via left renal vein / or more inferiorly√ crossover usually anterior / rarely posterior to aorta*DDx:*left gonadal v./ a., inferior mesenteric v.3.Transposition of IVC = solitary left IVC*Incidence:*0.2-0.5%*Etiology:*persistence of left + regression of right supracardinal vein√ left IVC usually crosses over via left renal vein / or more inferiorly√ crossover usually anterior / rarely posterior to aorta4.Retroaortic left renal vein*Incidence:*1.8-2.4%*Etiology:*persistence of posterior intersupracardinal anastomosis + regression of anterior intersubcardinal anastomosis√ crossover usually below / occasionally at level of right renal vein5.Circumaortic left renal vein*Incidence:*1.5-8.7%*Etiology:*persistence of anterior intersubcardinal + posterior intersupracardinal anastomosis√ venous collar encircling aorta6.Interrupted IVC with azygos / hemiazygos continuation *see AZYGOS CONTINUATION*7.Persistent left SVC = Bilateral SVCs*Incidence:*0.3% of general population;4.3-11% of patients with CHD*Etiology:*failure of regression of left anterior + common cardinal veins + left sinus horn*May be associated with:*ASD, azygos continuation of IVC*Course:*lateral to aortic arch, anterior to left hilum√ left SVC drains into enlarged coronary sinus (common)√ left SVC drains into LA (rare) creating a R-to-L shunt (increased prevalence of CHD)√ hemiazygos arch formed by left superior intercostal vein + persistent left SVC (20%)√ absent / small left brachiocephalic vein (65%)√ absence of right SVC (10-18%)√ anastomosis between right + left anterior cardinal veins (in 35%)

Notes:

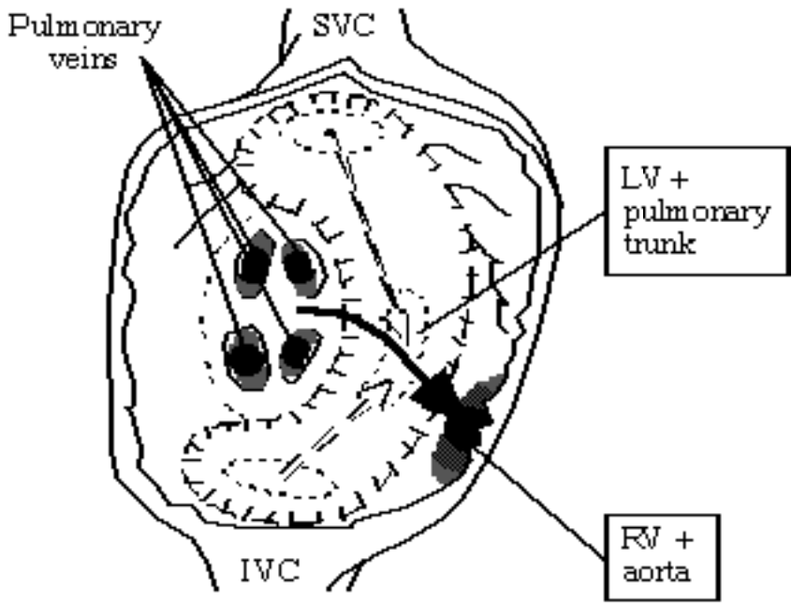
IVC Obstruction A.INTRINSIC OBSTRUCTION(a)neoplastic (most frequent)1.Renal cell carcinoma (in 10%), Wilms tumor2.Adrenal carcinoma, pheochromocytoma3.Pancreatic carcinoma, hepatic adenocarcinoma4.Metastatic disease to retroperitoneal lymph nodes (carcinoma of ovary, cervix, prostate)(b)nonneoplastic1.Idiopathic2.Proximally extending thrombus from femoroiliac veins3.Systemic disorders: coagulopathy, Budd-Chiari syndrome, dehydration, infection (pelvic inflammatory disease), sepsis, CHF4.Postoperative / traumatic phlebitis, ligation, plication, clip, cava filter, severe exertionB.INTRINSIC CAVAL DISEASE(a)neoplastic1.Leiomyoma, leiomyosarcoma, endothelioma(b)nonneoplastic1.Congenital membraneC.EXTRINSIC COMPRESSION(a)neoplastic1.Retroperitoneal lymphadenopathy (adults) due to metastatic disease, lymphoma, granulomatous disease (TB)2.Renal + adrenal tumors (children)3.Hepatic masses4.Pancreatic tumor5.Tumor-induced desmoplastic reaction (eg, metastatic carcinoid)(b)nonneoplastic1.Hepatomegaly2.Tortuous aorta / aortic aneurysm3.Retroperitoneal hematoma4.Massive ascites5.Retroperitoneal fibrosisD.FUNCTIONAL OBSTRUCTION1.Pregnant uterus2.Valsalva maneuver3.Straining / crying (in children)4.Supine position with large abdominal mass
E.COLLATERAL PATHWAYS1.Deep pathway: ascending lumbar veins to azygos vein (right) + hemiazygos vein (left) + intravertebral, paraspinal, extravertebral plexus (Batson plexus)2.Intermediate pathway: via periureteric plexus + left gonadal vein to renal vein3.Superficial pathway: external iliac vein to inferior epigastric vein + superior epigastric vein + internal mammary vein into subclavian vein4.Portal pathway: retrograde flow through internal iliac vein + hemorrhoidal plexus into inferior mesenteric vein + splenic vein into portal vein

Notes:

Surgical Procedures A.AORTICOPULMONARY WINDOW SHUNT=side-to-side anastomosis between ascending aorta and left pulmonary artery (reversible procedure)◊Tetralogy of FallotB.BLALOCK-HANLON PROCEDURE=surgical creation of ASD◊Complete transpositionC.BLALOCK-TAUSSIG SHUNT=end-to-side anastomosis of subclavian artery to pulmonary artery, performed ipsilateral to innominate artery / opposite to aortic archModified Blalock-Taussig shunt uses synthetic graft material such as polytetrafluoroethylene (Gore-Tex®) in an end-to-side anastomosis between subclavian artery + ipsilateral branch of pulmonary artery◊Tetralogy of Fallot, Tricuspid atresia with pulmonic stenosisD.FONTAN PROCEDURE=(1) external conduit from right atrium to pulmonary trunk (= venous return enters pulmonary artery directly) (2) closure of ASD: floor constructed from flap of atrial wall and roof from piece of prosthetic material◊Tricuspid atresia



E.GLENN SHUNT=end-to-side shunt between distal end of right pulmonary artery and SVC; reserved for patients with cardiac defects in which total correction is not anticipated◊Tricuspid atresiaF.POTT SHUNT=side-to-side anastomosis between descending aorta + left pulmonary artery◊Tetralogy of FallotG.MUSTARD PROCEDURE(a) removal of atrial septum (b) pericardial baffle placed into common atrium such that systemic venous blood is rerouted into left ventricle and pulmonary venous return into right ventricle and aorta ◊Complete transposition



Mustard Procedure (lateral view into opened right atrium)

H.RASHKIND PROCEDURE = balloon atrial septostomy◊Complete transpositionI.RASTELLI PROCEDUREexternal conduit (Dacron) with porcine valve connecting RV to pulmonary trunk ◊TranspositionJ.WATERSTON-COOLEY SHUNT=side-to-side anastomosis between ascending aorta and right pulmonary artery; (a) extrapericardial (WATERSTON) (b) intrapericardial (COOLEY)◊Tetralogy of Fallot

Notes:

Postoperative Thoracic Deformity A.ON RIGHT SIDE1.Systemic-PA shunt: Blalock-Taussig shunt, Waterston-Cooley shunt, Glenn shunt, Central conduit shunt2.Atrial septectomy: Blalock-Hanlon procedure3.VSD repair: through RA4.Mitral valve commissurotomyB.ON LEFT SIDE1.PDA2.Coarctation3.PA banding4.Mitral valve commissurotomy5.Systemic-PA shunt: Blalock-Taussig shunt, Pott shunt

Notes:

Heart Valve Prosthesis 1.Starr-Edwards√ caged ball◊predictable performance from large long-term experience2.Bjørk-Shiley / Lillehei-Kaster / St. Jude√ tilting disk◊ excellent hemodynamics, very low profile, durable3.Hancock / Carpentier-Edwards (= porcine xenograft)Ionescu-Shiley (= bovine xenograft)◊low incidence of thromboembolism, no hemolysis, central flow, inaudible

Notes:

CARDIAC CALCIFICATIONS

Detected by: fluoroscopy (at low-beam energies ≤75 kVp; 57% sensitivity) < digital subtraction fluoroscopy < conventional CT < ultrafast CT (96% sensitivity)@Coronary arteries*see below* @Cardiac valves◊Valvar calcification means stenosis - its amount is proportionate to degree and duration of stenosis!1.**Aortic valve** ● usually indicates significant aortic stenosis*Cause:*congenital bicuspid valve (70-85%) > atherosclerotic degeneration > rheumatic aortic stenosis (rare), syphilis, ankylosing spondylitis Location:above + anterior to a line connecting carina + anterior costophrenic angle (lateral view)(a)Stenotic congenital bicuspid valve ● calcium first detected at an average age of 28 years√ usually extensive cluster of heavy dense calcific deposits assuming a nodular contour√ poststenotic dilatation of ascending aorta(b)Degenerative aortic stenosis ● calcium first detected at an average age of 54 years◊In patients >65 years aortic valve calcification in 90% due to atherosclerosis!√ curvilinear shape of calcium outlining tricuspid leaflets√ diffuse dilatation + tortuosity of aorta (NO poststenotic dilatation)(c)Isolated rheumatic aortic stenosis ● calcium first detected at an average age of 47 years√ cluster of heavy dense calcific deposits without bicuspid contour2.**Mitral valve leaflet***Cause:*rheumatic heart disease (virtually always), mitral valve prolapseLocation:inferior to a line connecting carina + anterior costophrenic angle (on lateral view) ● calcium first detected in early thirties when patients become overtly symptomatic√ delicate calcification similar to coronary arteries (DDx: calcium in RCA / LCX)√ superior-to-inferior motion3.**Pulmonic valve***Cause:*tetralogy of Fallot, pulmonary stenosis, atrial septal defect√ calcific pattern similar to calcified mitral valve4.**Tricuspid valve** (extremely rare)*Cause:*rheumatic heart disease, septal defect, tricuspid valve defect, infective endocarditis

@Annulus=valve rings serve as fibrous skeleton of the heart for attachment of myocardial fibers + cardiac valves1.**Mitral annulus***Cause:*degenerative (physiologic in elderly)*Age:*>65 years*May be associated with:*mitral valve prolapse*Commonly associated with:*aortic valve calcium√ dense bandlike calcification starting at posterior aspect + progressing laterally frequently forming a "reversed C" / "U" / "J"*Cx:*mitral insufficiency, atrial fibrillation, heart block2.**Aortic annulus**√ usually in combination with degenerative aortic valve calcification3.**Tricuspid annulus***Associated with:*long-standing RV hypertensionLocation:right AV groove√ bandlike C-shaped configuration

@Pericardium*Cause:*idiopathic pericarditis, rheumatoid arthritis (5%), tuberculosis, viral, chronic renal failure, radiotherapy of mediastinumLocation:calcification over less pulsatile right-sided chambers, atrioventricular grooves, pulmonary trunk◊50% of patients with constrictive pericarditis show pericardial calcifications!*Cx:*constrictive pericarditis

@Myocardium*Cause:*infarction, aneurysm, rheumatic fever, myocarditisLocation:apex / anterolateral wall of LV (coincides with typical location of LV aneurysms)√ fine curvilinear contour outlines the aneurysm√ shaggy laminated calcification suggests associated calcification of mural thrombus√ coarse amorphous calcifications are caused by trauma, cardioversion, infection, endocardial fibrosis@Interventricular septumLocation:triangular fibrous area between mitral + tricuspid annuli (= trigona fibrosa) representing the basal segment of interventricular septum, closely related to bundle of His*Always associated with:* heavy calcification of mitral annulus / aortic valve *Cx:*heart block

@Left atrial wall*Cause:*rheumatic mitral valve disease(a)diffuse form ● patient usually in bilateral CHF + atrial fibrillation√ diffuse sheetlike calcification starting in the appendage sparing posterolateral wall on right side*Cx:*mural thrombus formation + emboli(b)localized form√ nodular calcific scar in posterior wall (= McCallum patch) due to injury from a forceful jet in mitral valve insufficiency@Cardiac tumoratrial myxoma (in 10% calcified), rhabdomyoma, fibroma, angioma, osteosarcoma, osteoclastoma @Endocardium*Cause*:cardiac aneurysm, thrombus, endocardial fibroelastosis@Pulmonary artery*Cause:*severe precapillary pulmonary arterial hypertension, syphilis@Ductus arteriosus(a)in adults:indicates patency of ductus with associated long-standing precapillary pulmonary hypertension(b)in children:ductus likely closed√ calcium deposition in ligament of Botallo

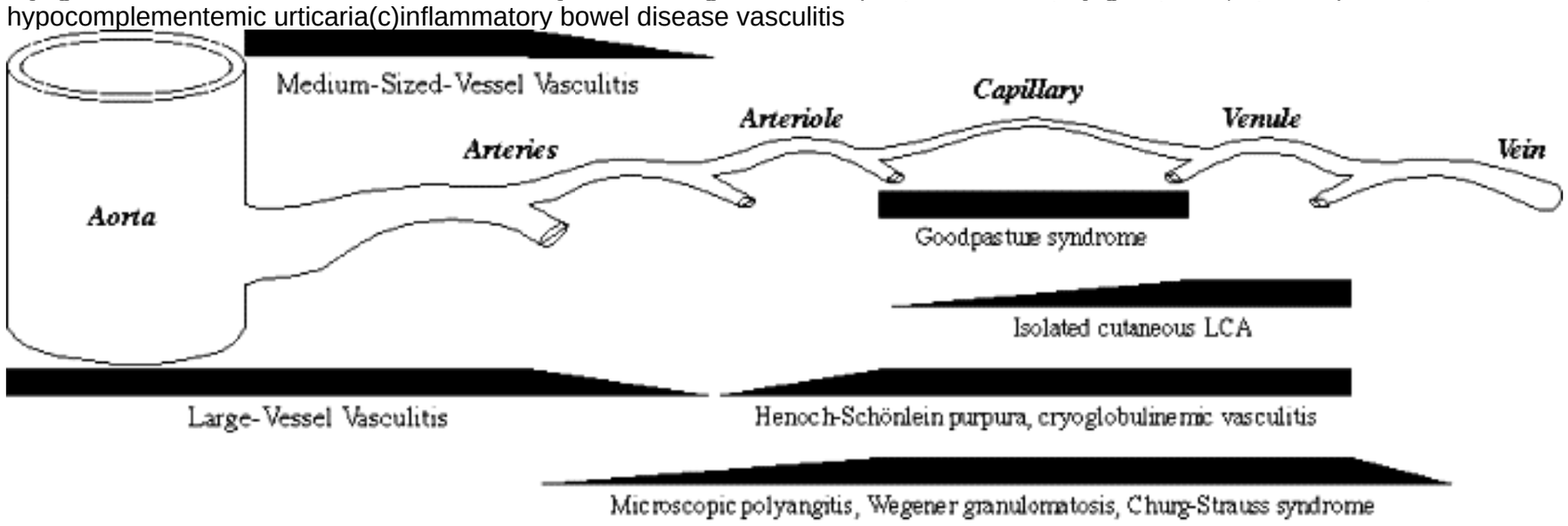
Coronary Artery Calcification Vasculitis

Notes:

Coronary Artery Calcification =due to (1) arteriosclerosis of intima (2) Mönckeberg medial sclerosis (exceedingly rare)*Histo:*calcified subintimal plaques◊Calcium is deposited in hemorrhagic areas within atheromatous plaques!CXR (detection rate up to 42%): ◊indicating more severe coronary artery diseaseFluoroscopy: (promoted as inexpensive screening test) (a)asymptomatic population-calcifications in 34% in asymptomatic male individuals-in 35% of patients with calcifications exercise test will be positive (without calcifications only in 4% positive)-calcifications indicate >50% stenosis with 72-76% sensitivity, 78% specificity); frequency of coronary artery calcifications with normal angiogram increases with age; predictive values in population <50 years as good as exercise stress test(b)symptomatic population-in 54% of symptomatic patients with ischemic heart disease◊In symptomatic patients 94% specificity for obstructive disease (>75% stenosis) of at least one of the three major vessels!Location: "coronary artery calcification triangle" = triangular area along mid left heart border, spine, and shoulder of LV containing left main coronary artery, proximal portions of LAD + LCX calcifications at autopsy: LAD (93%), LCX (77%), left main CA (70%), RCA (69%) ◊ parallel calcified lines (lateral view)*Prognosis:*58% 5-year survival rate with and 87% without calcifications

Notes:

Vasculitis A.LARGE-VESSEL VASCULITIS1.Giant cell (temporal) arteritis2.Takayasu diseaseB.MEDIUM-SIZED-VESSEL VASCULITIS1.Polyarteritis nodosa2.Kawasaki diseaseC.SMALL-VESSEL VASCULITIS(a)ANCA-associated small-vessel vasculitis(= antineutrophil cytoplasmic autoantibodies) 1.Wegener granulomatosis2.Churg-Strauss syndrome3.Microscopic polyangiitis(b)immune-complex small-vessel vasculitis1.Henoch-Schönlein purpura2.Essential cryoglobulinemic vasculitis3.Cutaneous leukocytoclastic angiitisothers:lupus, rheumatoid, Sjögren, Behçet, Goodpasture, serum sickness, drug-induced, hypocomplementemic urticaria(c)inflammatory bowel disease vasculitis



Noninfectious Vasculitides

Notes:

PULSUS ALTERNANS
=alternating arterial pulse height with regular cardiac rhythm1.Intrinsic myocardial abnormalitysevere left ventricular dysfunction (CHF, aortic valvular disease, hypothermia, hypocalcemia, hyperbaric stress, ischemia) 2.Alternating end-diastolic volumesabnormalities in venous filling + return (obstructed venous return, IVC balloon)

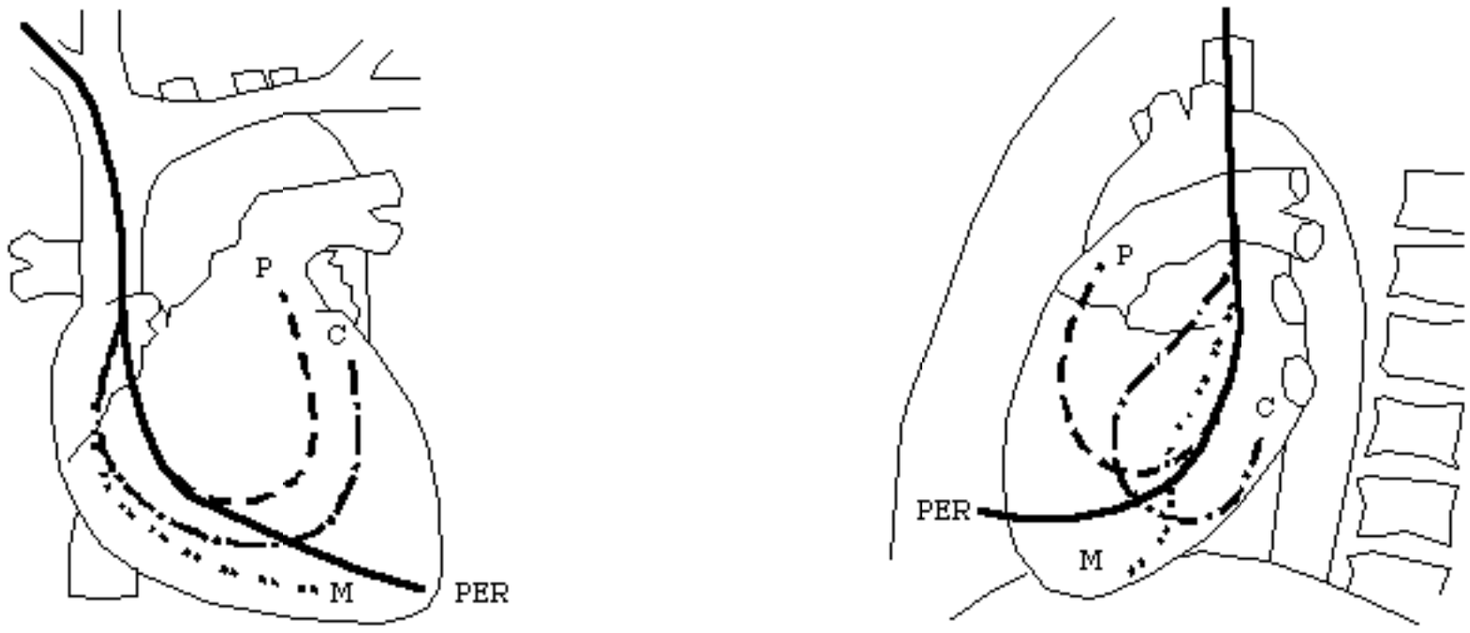
Notes:

ARTERIAL HYPERTENSION

A.ESSENTIAL (85-90%)B.RENAL PARENCHYMAL DISEASE (5-10%)C.POTENTIALLY CURABLE(1-2%)(a)vascular1.Renovascular disease2.Coarctation(b)hormonal1.Pheochromocytoma2.Cushing syndrome3.Primary aldosteronism4.Hyperthyroidism5.Myxedema(c)renal1.Unilateral renal disease

Notes:

CENTRAL VENOUS LINE POSITIONS

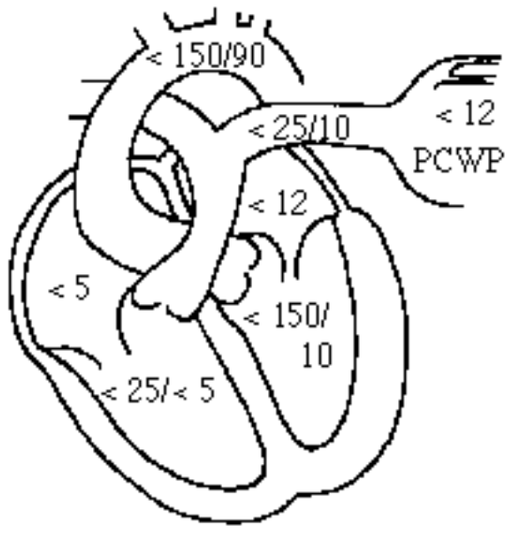


Central Venous Line Positions

C = coronary sinus, M = middle cardiac vein, P = main pulmonary artery, PER = perforation

Notes:

Normal Blood Pressures

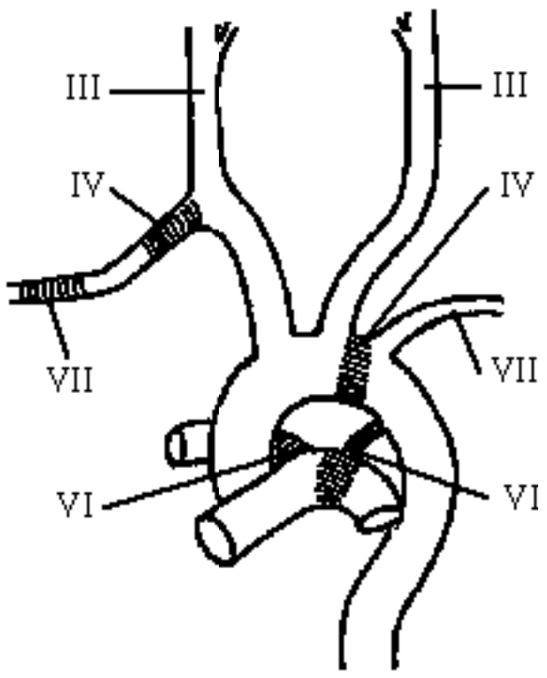


Normal Blood Pressures

PCWP = pulmonary capillary wedge pressure

Notes:

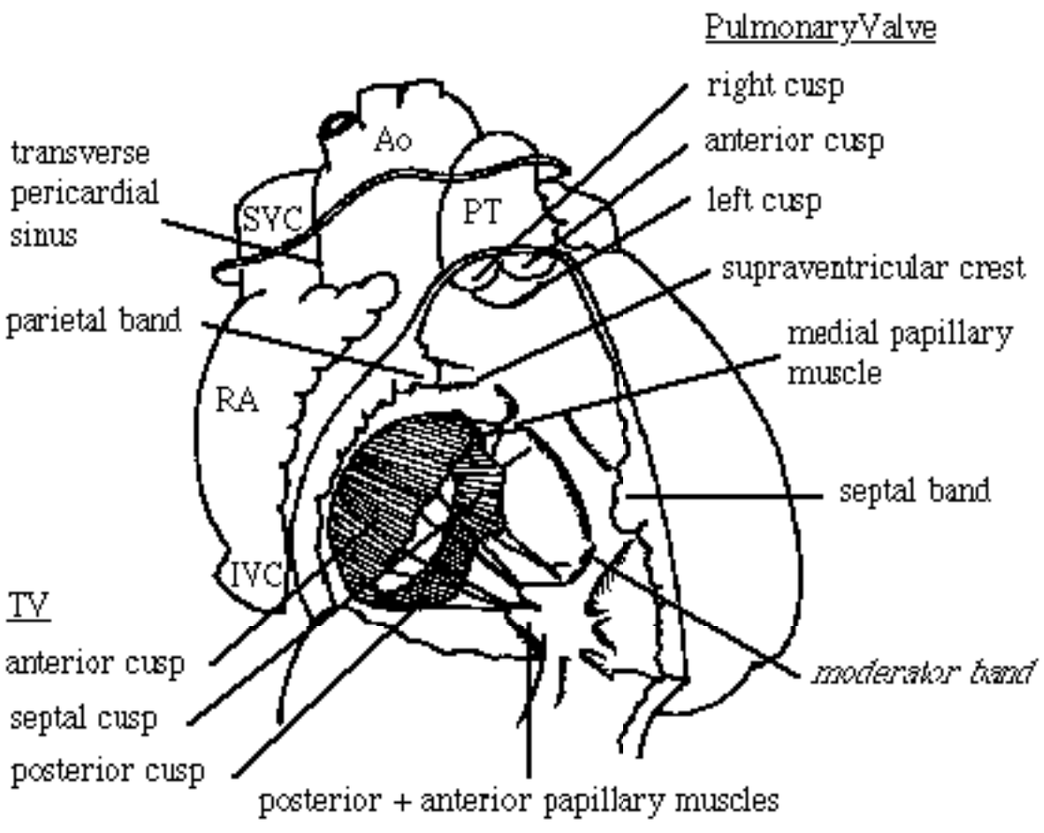
Development of Major Blood Vessels



Development of Major Blood Vessels
numbers refer to embryologic aortic arches
most portions of aortic arches I, II, V regress

Notes:

Right Ventricle Viewed from Front



Right Ventricle Viewed from Front
Demarcation between posteroinferior inflow portion and anterosuperior outflow portion by prominent muscular bands forming an almost circular orifice -parietal band-crista supraventricularis-septomarginal trabeculae (= septal band + moderator band)Anterior papillary muscle originates from moderator band!

Notes:

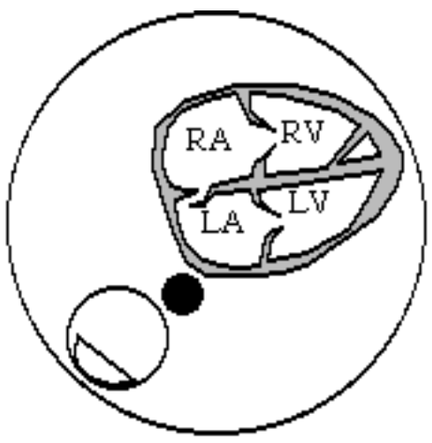
Sweep of Transducer From Aorta Toward Apex

Sweep of Transducer From Aorta Toward Apex

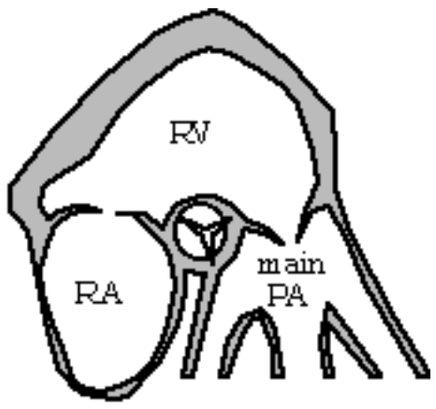
Area 1:recognized by parallel motion of both aortic walls (a) toward the transducer during systole (b) away from the transducer during diastole. Left atrial posterior wall (LAPW) does not move because of mediastinal attachment by pulmonary veins.
Aortic valve cusps (right coronary + noncoronary / left cusps) are positioned in middle of aorta during diastole, open abruptly during systole at onset of ventricular ejection in a "box-like" fashion.Aortic + LA dimension are similar in most cases.Area 2:Aortic-septal continuity = anterior aortic wall becomes interventricular septumAortic-mitral continuity = posterior aortic wall becomes anterior mitral valve leafletMitral valve with typical "M" configuration during diastole; motion of aML toward transducer during systole secondary to movement of whole mitral valve apparatusArea 3:posterior mitral valve leaflet (pML) = reciprocal "W-shaped" configuration; left ventricular posterior wall (LVPW) shows anterior motion during systole.Area 4:Chordae tendineae in continuity with mitral valve leaflets merge with a thick posterior band of echoes representing the posteromedial papillary muscle (ppm).

Notes:

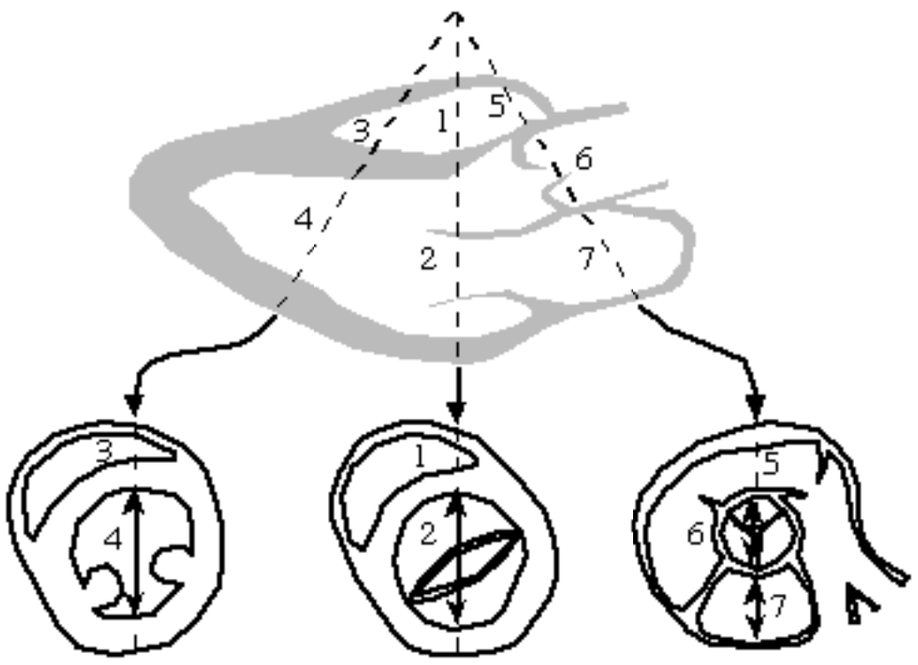
FETAL ECHOCARDIOGRAPHIC VIEWS



Fetal Four-Chamber View

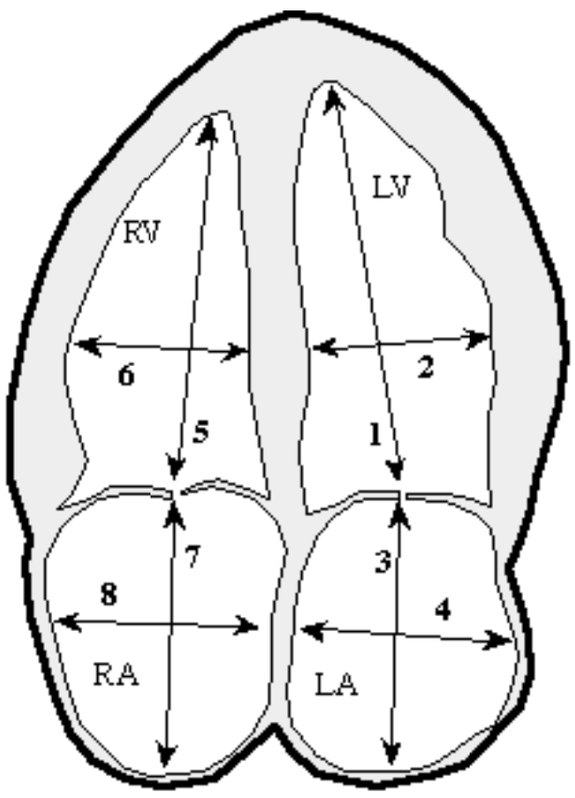


Fetal Short-axis View



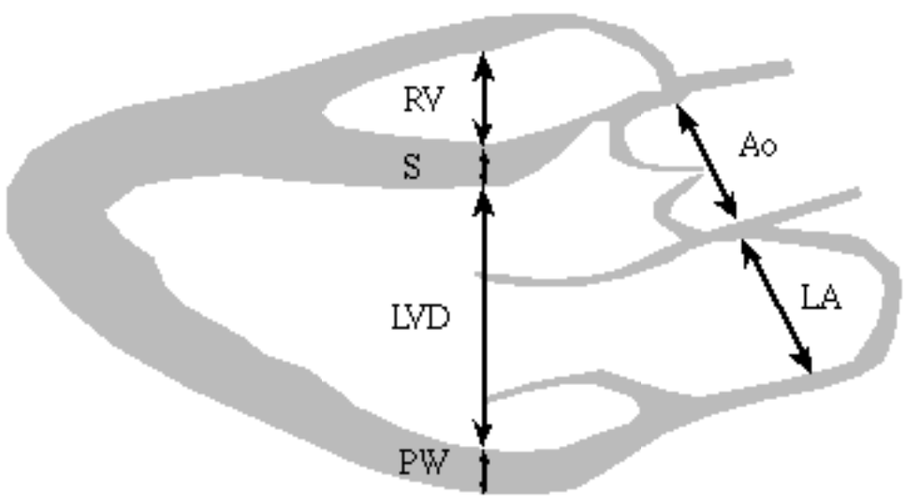
Parasternal Long- And Short-Axis Views

1, 3, 5 = RV dimension
6 = aortic root
7 = LA
2 = LV dimension at mitral level
4 = LV dimension at papillary muscle level



Apical 4-Chamber View

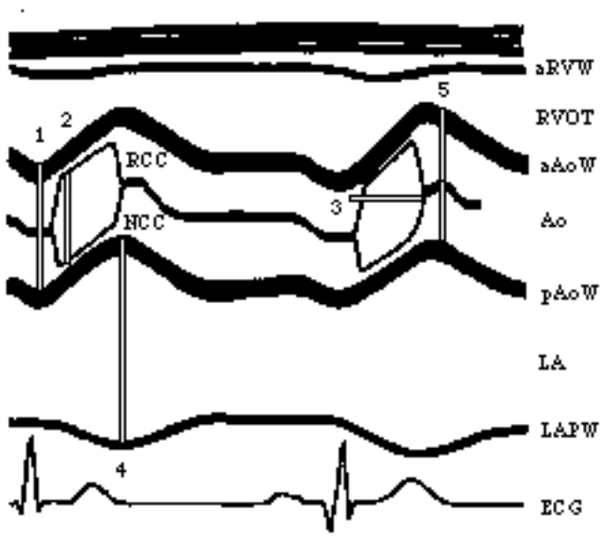
1 = LV long axis
2 = LV short axis
3 = LA major axis
4 = LA minor axis
5 = RV long axis
6 = RV short axis
7 = RA major axis
8 = RA minor axis



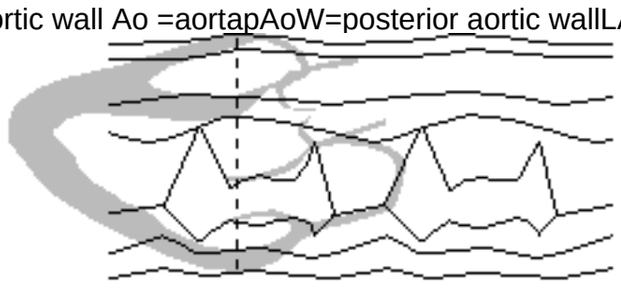
Parasternal Long-Axis View

Ao = aorta
LA = left atrium
LVD = left ventricular diameter
PW = posterior wall
RV = right ventricle
S = septum

A.FOUR-CHAMBER VIEW1.Position of heart within thorax2.Number of cardiac chambers3.Ventricular proportion4.Integrity of atrial + ventricular septa5.Position + size + excursion of AV valvesB.PARASTERNAL LONG-AXIS VIEW=LEFT VENTRICULAR OUTFLOW TRACT1.Continuity between ventricular septum + anterior aortic wall2.Caliber of aortic outflow tract3.Excursion of aortic valve leafletsC.SHORT-AXIS VIEW OF OUTFLOW TRACTS1.Spatial relationship between aorta + pulmonary artery2.Caliber of aortic + pulmonary outflow tractsD.AORTIC ARCH VIEW Identification of fetal RV √ RV lies closest to anterior chest wall√ foramen ovale flap seen within LA√ prominent moderator band + papillary muscles in RV**Echocardiogram of Aortic Root**

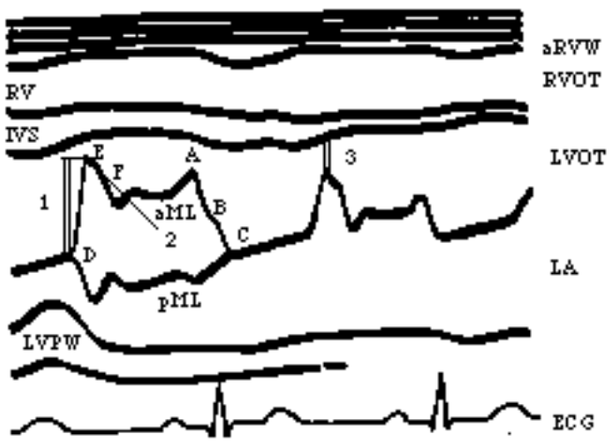


1 = **aortic root dimension**, measured atend-diastole at R-wave of ECG2.1 - 4.3 cm*increased in:*aneurysm of aorta,aortic insufficiency2 = **aortic cusp separation**: 1.7 - 2.5 cm*decreased in:*aortic stenosis,low stroke volume*increased in:*aortic insufficiency3=**left ventricular ejection time**
4 = **left atrial diameter**, measured atmoment of mitral valve opening2.3 - 4.4 cm5 =**eccentricity index of aortic valve**
cusps = ratio of anterior to posteriordimension (rarely used)<1.34 ÷ 1=**ratio of LA-to-aortic root**
dimension0.87 - 1.11aRVW =anterior right ventricular wallRVOT=right ventricular outflow tractaAoW=anterior aortic wall Ao =aortapAoW=posterior aortic wallLA=left

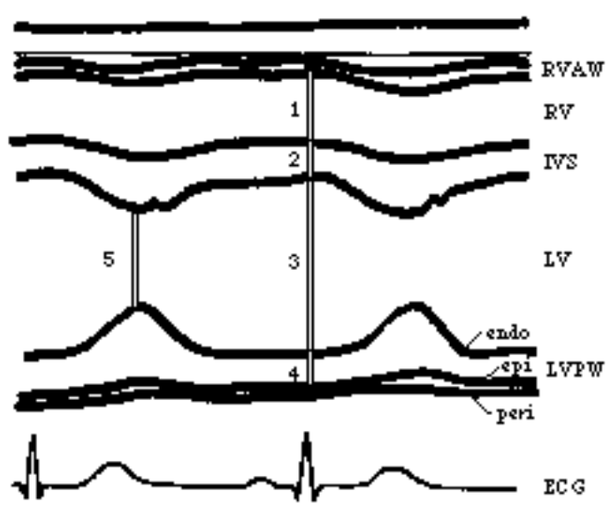


atriumLAPW =left atrial posterior wallNCC=noncoronary cuspRCC=right coronary cuspECG =electrocardiogram **Mitral Valve in Mid-Diastole**

Echocardiogram of Mitral Valve 1=**mitral valve excursion** = opening



amplitude of anterior leaflet of mitralvalve (DE amplitude)2 - 3 cm*decreased in:*nonpliable MV stenosis,low cardiac output,low compliance of LV*increased in:*MV prolapse,high flow through MV2=**E to F slope** = early diastolicposterior motion of anterior leaflet7 - 15 cm/sec*decreased in:*mitral valve stenosis,low compliance of LV3=**septal-mitral valve distance** =E point septal separation2.9 - 4.1 mm*decreased in:*ostium primum ASD,IHSS*increased in:*dilated LVRV=right ventricleIVS=interventricular septumLVPW=left ventricular posterior wallaML=anterior mitral valve leafletpML=posterior mitral valve leafletaRVW=anterior right ventricular wallRVOT=right ventricular outflow tractLVOT =left ventricular outflow tractLA=left atriumECG =electrocardiogramA=point of atrial contractionC=closure pointDE=opening secondary to passive ventricular fillingCD=systole with steady anterior drift of coapted leaflets (passive movement secondary to movement of entire heart toward chest wall) **Echocardiogram of Right and Left Ventricle**



1= **RV end-diastolic dimension**
(RVEDD) at R-wave of ECG0.7 - 2.3 cm*increased in:*RV volume overload2= **septal thickness** = end-diastolic IVSthickness at R-wave of ECG0.9 ± 0.06 cm*decreased in:*CAD*increased in:*asymmetric septalhypertrophy, IHSS3= **LV end-diastolic dimension**
(LVEDD) at R-wave of ECG4.6 ± 0.54 cm4= **LVPW thickness**, measured at end-diastole at peak of R-wave of ECG:0.94 ± 0.09 cm*increased in:*LV hypertrophy5= **LV end-systolic dimension**
(LVESD)2.9 ± 0.5 cm3 and 5=**fractional shortening of**
internal diameter= (EDD - ESD)/EDD x 1000.25 0.42IVS:LVPW thickness<1.3RVAW=right ventricular anterior wallRV=right ventricleIVS= interventricular septumLV= left ventricleLVPW= left ventricular posterior wallendo=endocardiumepi=epicardiumperi=pericardiumFractional shortening (FS) = [(end-diastolic size - systolic size) / end-diastolic size] x 100 - for LV=25 - 42%
- for IVS=28 - 62%
- for LVPW=36 - 70%

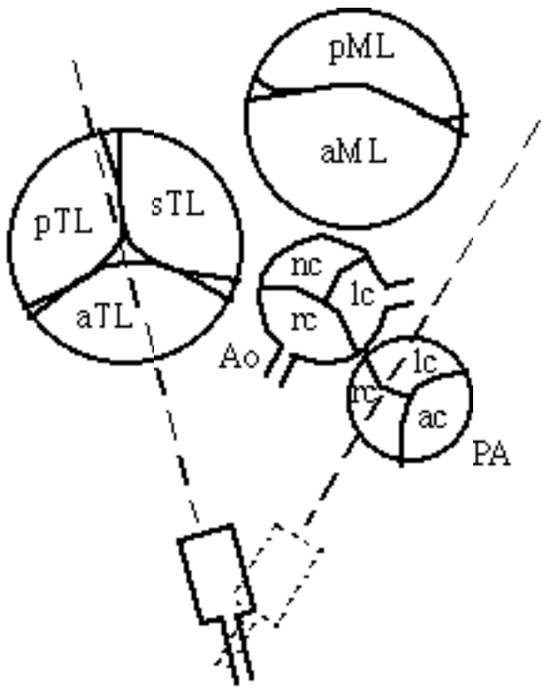


Diagram Showing the Relationship of the Four Cardiac Valves in Cross Section

aTL, pTL, sTL = anterior, posterior, septal tricuspid valve leaflets
aML, pML = anterior, posterior mitral valve leaflets
rc, lc, nc (Ao) = right, left, noncoronary cusps of aorta
rc, lc, ac (PA) = right, left, anterior cusps of pulmonary artery

---

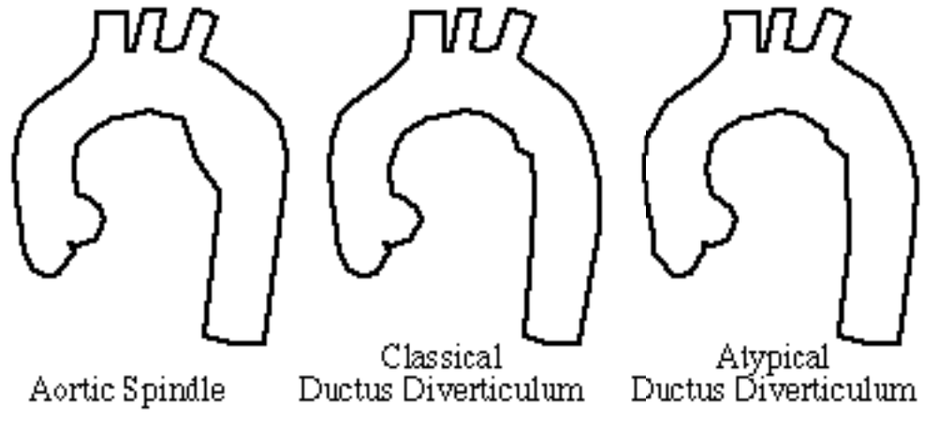
Notes:

Aortic Isthmus =narrowing of the aorta in newborn between left subclavian artery and ductus arteriosus*Age:*up to 2 months of age*Prognosis:*aortic isthmus disappears due to cessation of flow through ductus arteriosus + increased flow through narrowed region

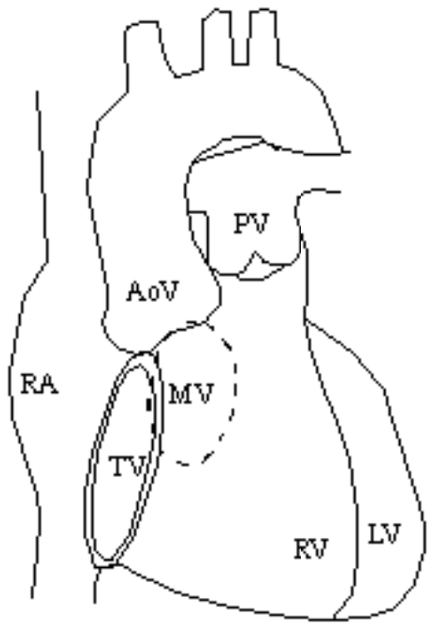
Notes:

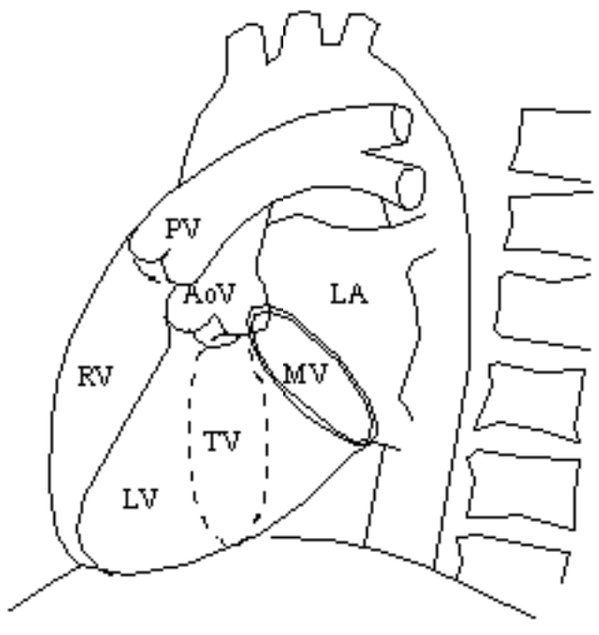
Aortic Spindle =normal variant of circumferential aortic bulge below isthmus region

Notes:



Ductus Diverticulum =focal bulge along anteromedial aspect of aortic isthmus *Frequency:*in 33% of infants, in 9% of adults√ focal bulge with smooth uninterrupted margins√ gently sloping symmetric shoulders (classic ductus diverticulum)√ shorter steeper slope superiorly + more gentle slope inferiorly (atypical ductus diverticulum)*DDx:*posttraumatic false aneurysm

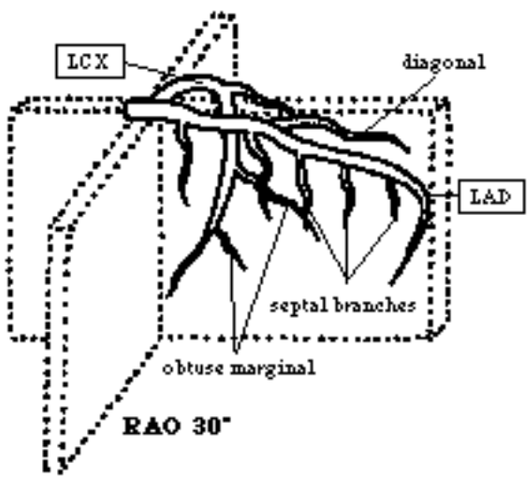


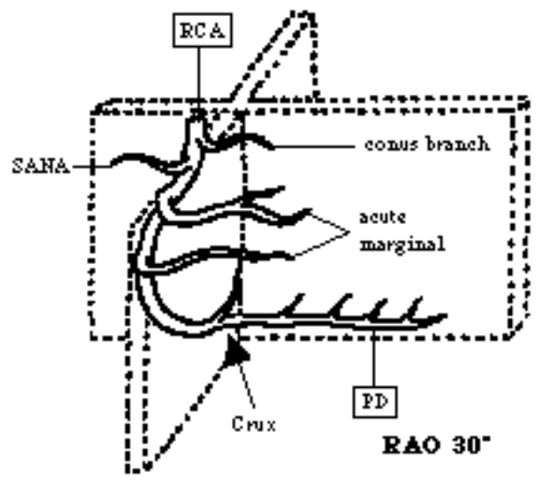


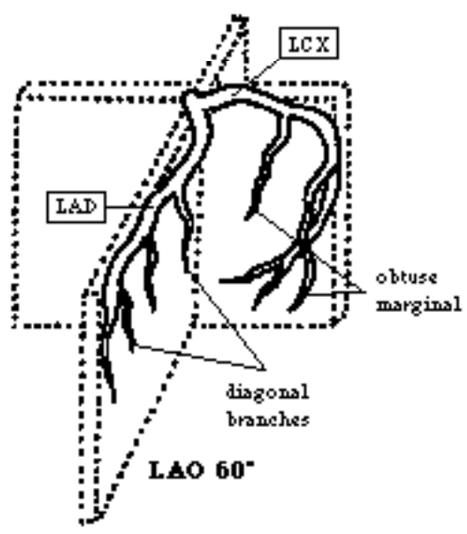
Heart Valve Positions
AoV = aortic valve, LA = left atrium, LV = left ventricle, MV = mitral valve, PV = pulmonic valve
RA = right atrium, RV = right ventricle, TV = tricuspid valve

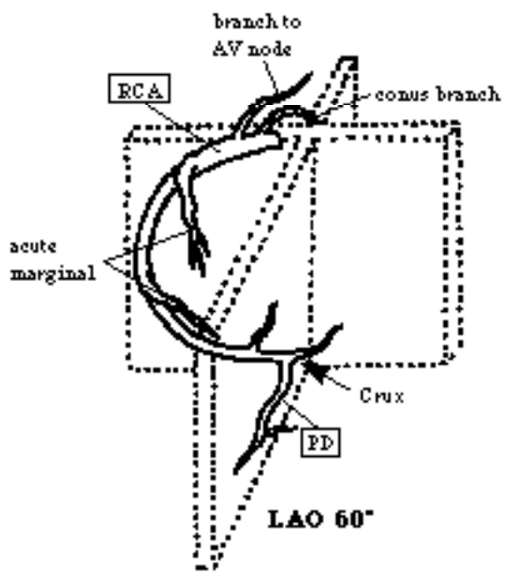
Notes:

CORONARY ARTERY ANATOMY









Anatomy of Left Coronary Artery
Marginals emanate from vessels in the AV groove (RCA, LXR) - on left side called obtuse marginal arteries- on right side called acute marginal arteries**Diagonals** emanate from vessel in the interventricular groove (LAD)Note: **D**iagonals from LA**D**
Coronary dominance the dominant vessel is the one that supplies the inferolateral wall of LV**AV-node branch** from RCA (in 90%) = conus branch (1st branch in 50%)**SA-node branch** from RCA (in >50%) **Anatomy of Right Coronary Artery**
Arteries in atrioventricular plane: RCA=right coronary arteryLCX=left circumflex artery, gives blood supply to anterolateral papillary muscle**Arteries in interventricular plane:** LAD=left anterior descending artery, gives blood supply to anterolateral papillary musclePD=posterior descending artery, gives blood supply to posteromedial papillary muscleSANA=sinoatrial node artery

Notes:

Radiology Review Manual
fourth edition

Coronary Artery Collaterals A.INTRACORONARY COLLATERALS=filling of a distal portion of an occluded vessel from the proximal portion via tortuous course outside the normal pathB.INTERCORONARY COLLATERALS=between different coronary arteries / between branches of the same arteryLocation:on epicardial surface, in atrial / ventricular septum, in myocardium1.proximal RCA to distal RCA(a)by way of acute marginal branches(b)from sinoatrial node artery (SANA) to atrioventricular node artery (AVNA) = Kugel collateral2.RCA to LAD(a)between PDA and LAD through ventricular septum / around apex(b)conus artery (1st branch of RCA) to proximal part of LAD(c)acute marginals of RCA to right ventricular branches of LAD3.distal RCA to distal LCX(a)posterolateral segment artery of RCA to distal LCX (in AV groove)(b)AVNA of RCA to LCX (through atrial wall)(c)posterolateral branch of RCA to obtuse marginal branches of LCX (over left posterolateral ventricular wall)4.proximal LAD to distal LAD(a)proximal diagonal to distal diagonal artery of LAD(b)proximal diagonal to LAD directly5.LAD to obtuse marginal of LCX

Notes:

Coronary Artery Dominance =vessel that supplies the inferior portion of left ventricleRCA in 80% LCA in 10% RCA + LCA (codominance with balanced supply) in 10%

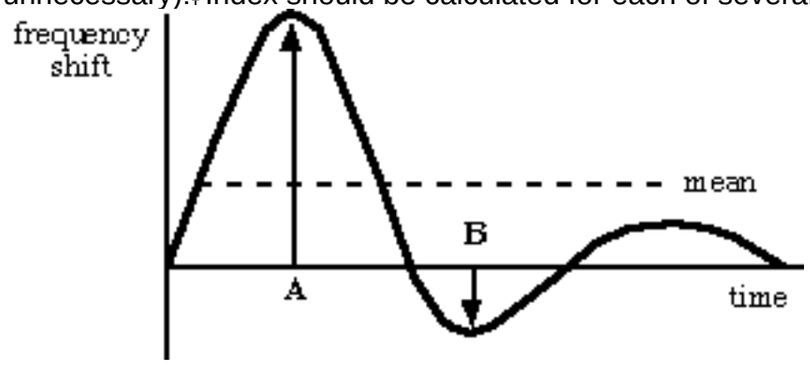
Notes:

Coronary Arteriography *Contrast agents:* 1.Monomeric ionic contrast material:(a)negative inotropic = depression of myocardial contractility due to hyperosmolality of sodium + decrease in total calcium(b)peripheral vasodilatation2.Meglumine diatrizoate (contains small quantities of sodium citrate + EDTA)3.Nonionic contrast material = slight increase in LV contractility*Mortality:*0.05%*Risk factors associated with death:* 1.multiple ventricular premature contractions2.congestive heart failure3.systemic hypertension4.severe triple-vessel coronary artery disease (highest risk)5.LV ejection fraction <30%6.Left main coronary artery stenosis*Projections:* (a)LAO + 20 - 30° caudocranial angulation proximal 1/3 of LAD + origin of first diagonal branch(b)LAO + 20 - 30° craniocaudal angulation = "spider view"Left main coronary artery, proximal LCX, first marginal / diagonal branches(c)RAO + 20 - 30° craniocaudal angulationProximal 1/3 of LCX + origin of its branches(d)RAO + 20 - 30° caudocranial angulationSeparation of LAD from diagonal branches*False-negative interpretation:* (1)eccentric lesion in 75%(2)foreshortening of vessel(3)overlap of other vessels remedied by angulated projections: improved diagnosis (50%), upgrade to more significant stenosis (30%), lesion unmasked (20%)

Notes:

PULSATILITY

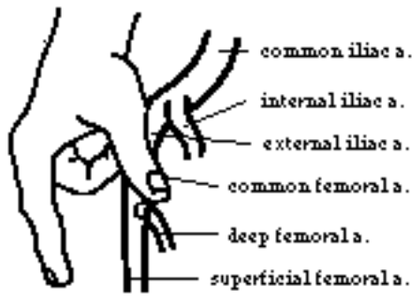
=assessment of vascular resistance (increased resistance reduces diastolic flow)Can be assessed in vessels too small / tortuous to be imaged (Doppler angle unnecessary)!Index should be calculated for each of several cardiac cycles (5 heartbeats adequate) an average value taken

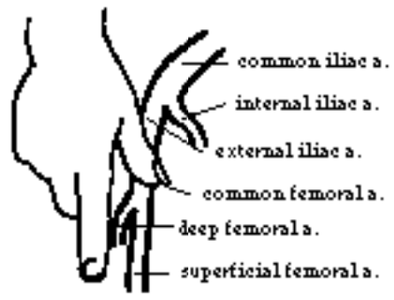


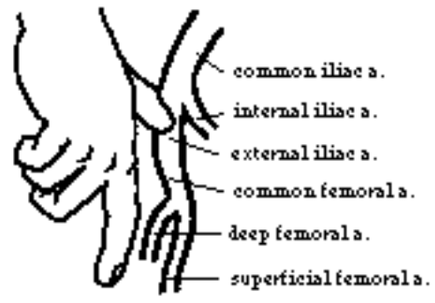
S=A= maximal systolic shiftD=B= end-diastolic frequency shift 1.Full pulsatility index of Gosling (PI_F) = $1/A_0^2$ $SA_i^2$2.Simplified pulsatility index (PI) = (S - D)/mean3.Resistance index (RI) = Pourcelot index= (S - D)/S or 1 - (D/S) 4.Stuart index = A/B ratio = S/D ratio5.B/A ratio = B(100%)/A DECREASE IN LUMEN DIAMETER VS. CROSS-SECTIONAL AREA decrease indecrease inlumen diametercross-sectional area20%36%40%64%60%84%80% 96%

Notes:

CONTENTS OF FEMORAL TRIANGLE
mnemonic: "NAVEL" (from lateral to medial)





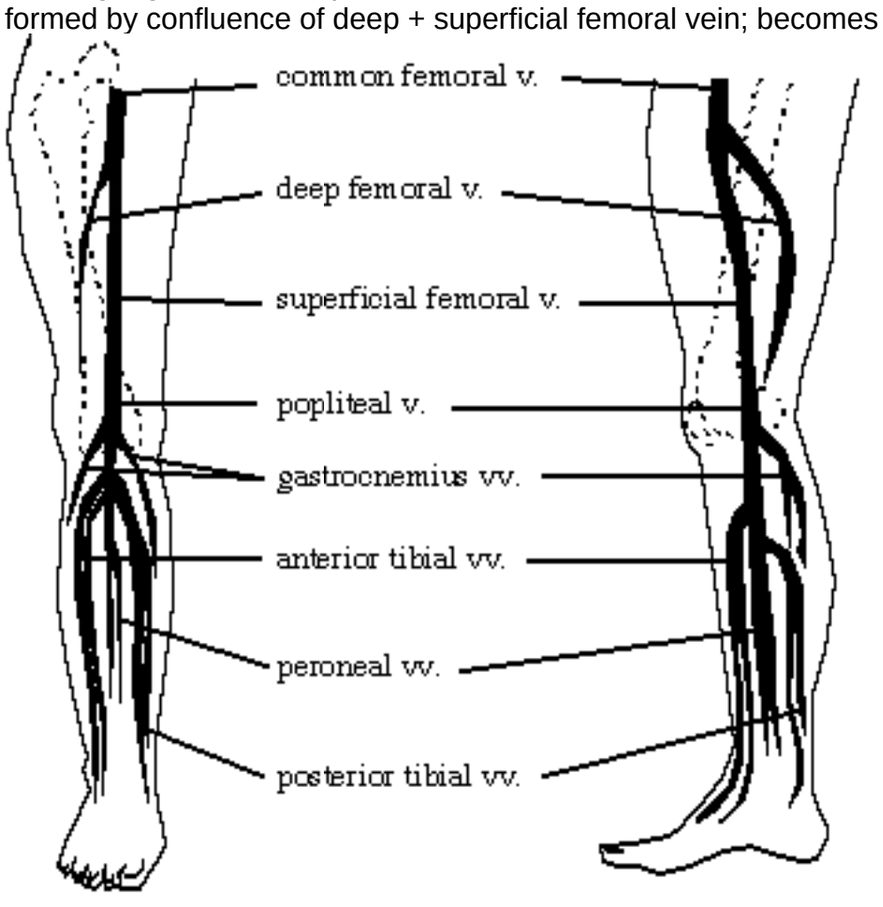


Nerve**A**rtery**V**ein**E**mpty space**L**ymphatics

Pelvic Arterial Anatomy (right side)

Notes:

Deep Veins Of Lower Extremity 3 paired stem veins of the calf accompany the arteries as venae commitantes + anastomose freely with each other: 1.**Anterior tibial veins**
draining blood from dorsum of foot, running within extensor compartment of lower leg close to interosseous membrane 2.**Posterior tibial veins**
formed by confluence of superficial + deep plantar veins behind ankle joint 3.**Peroneal veins**
directly behind + medial to fibula 4.Calf veins(a)**Soleal muscle veins**
baggy valveless veins in soleus muscle (= sinusoidal veins); draining into posterior tibial + peroneal veins or lower part of popliteal vein (b)**Gastrocnemius veins**
thin straight veins with valves; draining into lower + upper parts of popliteal vein 5.**Popliteal vein**
formed by stem veins of lower leg 6.**Femoral / superficial femoral vein**
continuation of popliteal vein; receives deep femoral vein about 9 cm below inguinal ligament 7.**Deep femoral vein**
draining together with superficial femoral vein into common femoral vein; may connect to popliteal vein (38%) 8.**Common femoral vein**
formed by confluence of deep + superficial femoral vein; becomes external iliac vein as it passes beneath inguinal ligament



Notes:

Superficial Veins Of Lower Extremity

1.**Greater saphenous vein**

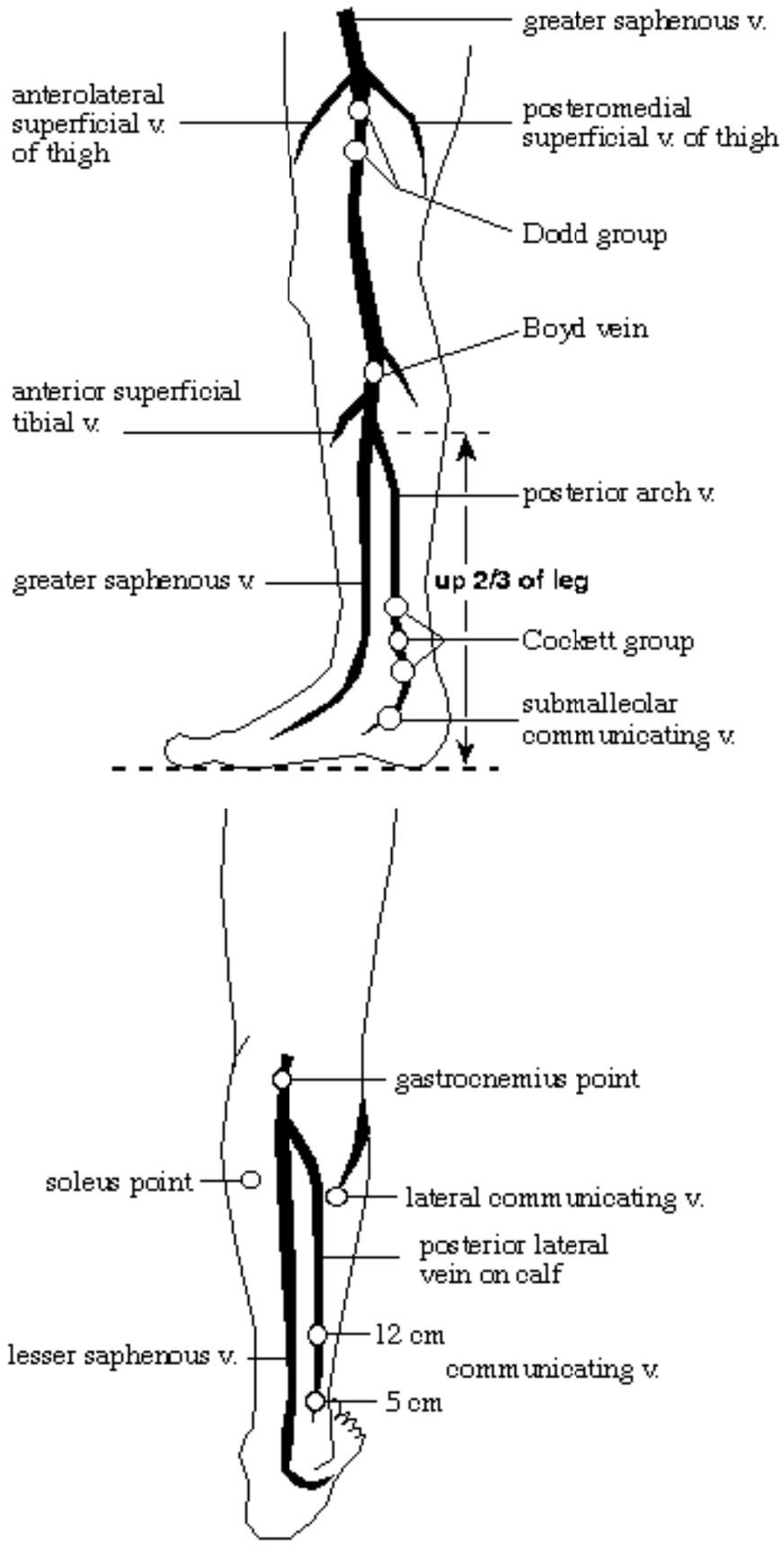
formed by union of veins from medial side of sole of foot with medial dorsal veins; ascends in front of medial malleolus; passes behind medial condyles of tibia + femur (a)Posterior arch veinconnected to deep venous system by communicating veins (b)**Anterior superficial tibial vein**

(c)**Posteromedial superficial thigh vein**

often connects with upper part of lesser saphenous vein (d)**Anterolateral superficial thigh vein**

(e)Tributaries in fossa ovalis-superficial inferior epigastric vein-superficial external pudendal vein-superficial circumflex iliac vein2.**Lesser saphenous vein**

originates at outer border of foot behind lateral malleolus as continuation of dorsal venous arch; enters popliteal vein between heads of gastrocnemius in popliteal fossa within 8 cm of knee joint (60%) or joins with greater saphenous vein via posteromedial / anterolateral superficial thigh veins (20%)

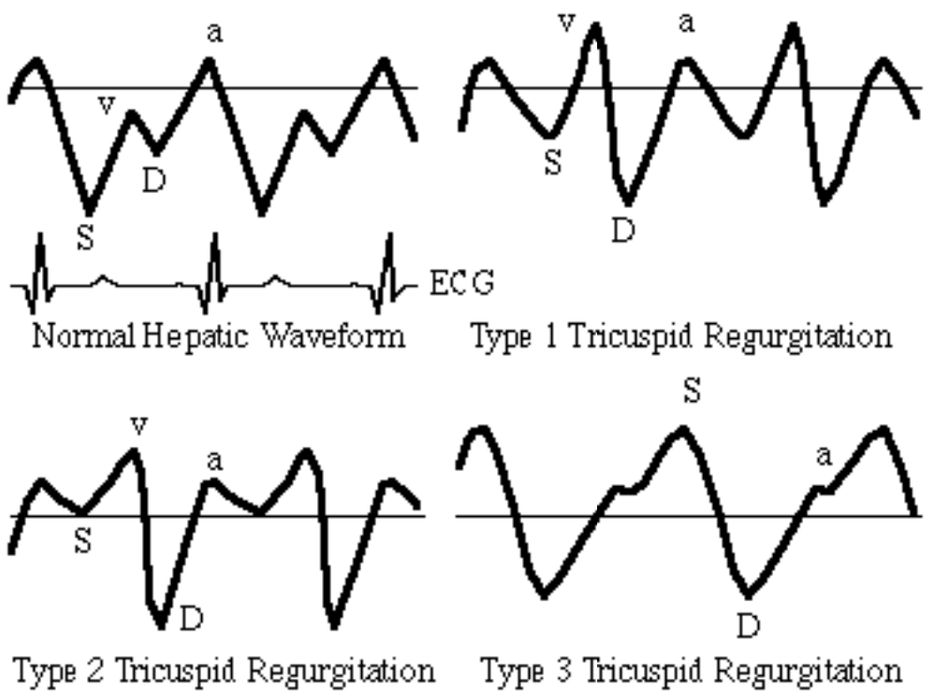


Notes:

Communicating = Perforating Veins >100 veins in each leg A.MEDIAL1.Submalleolar communicating vein2.**Cockett group**
group of 3 veins located 7, 12, 18 cm above the tip of medial malleolus connecting posterior arch vein with posterior tibial vein 3.**Boyd vein**
located 10 cm below knee joint connecting main trunk of greater saphenous vein to posterior tibial veins 4.**Dodd group**
group of 1 or 2 veins passing through Hunter canal (= subsartorial canal) to join greater saphenous vein with superficial femoral vein B.LATERAL1.**Lateral communicating vein**
located from just above lateral malleolus to junction of lower-to-mid thirds of calf connecting lesser saphenous vein with peroneal veins 2.**Posterior mid-calf communicating veins**
located posteriorly 5 + 12 cm above os calcis joining lesser saphenous vein to peroneal veins 3.**Soleal + gastrocnemius points**
joining short saphenous vein to soleal / gastrocnemius veins

Notes:

Doppler Waveforms of Hepatic Veins



Doppler Waveforms of Hepatic Veins

S wave = systolic wave resulting from negative RA pressure caused by atrial relaxation + movement of tricuspid anulus toward cardiac apex

v wave = resulting from elevated RA pressure caused by RA overfilling against a closed tricuspid valve; occurs in <50% of patients

D wave = diastolic wave resulting from negative RA pressure caused by opening of tricuspid valve + blood flow from RA into RV; equal to / smaller than S wave

a-wave = resulting from elevated RA pressure caused by RA contraction; in 66% of patients

Notes:

ABERRANT LEFT PULMONARY ARTERY
=PULMONARY SLING = failure of development / obliteration of left 6th aortic arch followed by development of a collateral branch of right pulmonary artery to supply the left lungSite:left PA passes above right mainstem bronchus + between trachea and esophagus on its way to left lung*Age at presentation:*neonate / infant / child*Associated with:* (1)"napkin-ring trachea" = absent pars membranacea (50%)(2)PDA (most common), ASD, persistent left SVC ● stridor (most common), wheezing, apneic spells, cyanosis ● respiratory infection ● feeding problems√ deviation of trachea to left√ "inverted-T" appearance of mainstem bronchi= horizontal course secondary to lower origin of right mainstem bronchus√ anterior bowing of right mainstem bronchus√ "carrot-shaped trachea" = narrowing of tracheal diameter in caudad direction resulting in functional tracheal stenosis√ obstructive emphysema / atelectasis of RUL + LUL√ low left hilum√ separation of trachea + esophagus at hilum by soft-tissue mass√ anterior indentation on esophagram

Notes:

AMYLOIDOSIS

=extracellular deposits of insoluble fibrillar protein ● asymptomatic / CHF (restrictive cardiomyopathy), arrhythmiaCXR: √ normal / generalized cardiomegaly√ pulmonary congestion√ pulmonary deposits of amyloidNUC: √ striking uptake of Tc-99m pyrophosphate greater than bone (50-90%)ECHO: √ granular sparkling appearance of myocardium√ LV wall thickening√ decreased LV systolic + diastolic function

Notes:

ANOMALOUS LEFT CORONARY ARTERY
=left coronary artery arises from pulmonary trunk (left sinus of Valsalva)*Hemodynamics:* with postnatal fall in pulmonary arterial pressure perfusion of LCA drops (ischemic left coronary bed), collateral circulation from RCA with flow reversal in LCA -adequate collateral circulation = lifesaving-inadequate collateral circulation = myocardial infarction-large collateral circulation = L-to-R shunt with volume overload of heart ● episodes of sweating, ashen color (angina symptomatology) ● ECG: anterolateral infarction ● continuous murmur (if collaterals large)√ dilatation of LV√ enlargement of LA√ normal pulmonary vascularity / redistribution*Rx:* (1)Ligation of LCA at its origin from pulmonary trunk(2)Ligation of LCA + graft of left subclavian artery to LCA(3)Creation of an AP window + baffle from AP window to ostium of LCA*DDx:*Endocardial fibroelastosis, viral cardiomyopathy (NO shocklike symptoms)

Notes:

Total Anomalous Pulmonary Venous Return = TAPVR = anomalous connection between pulmonary veins and systemic veins secondary to embryologic failure of the common pulmonary vein to join the posterior wall of the left atrium *Prevalence:*2% of CHD*Age:*symptomatic in 1st year of life*Associated with:* ASD / patent foramen ovale (necessary for survival), bronchopulmonary sequestration, pulmonary arteriovenous malformation, cystic adenomatoid malformation *Overall prognosis*:75% mortality rate within 1 year of birth if untreated
Subdiaphragmatic TAPVR Type I=SUPRACARDIAC TAPVR (52%)=drainage into left brachiocephalic vein / right + left persistent SVC / azygos vein; <10% obstructedType II=CARDIAC TAPVR (30%)=drainage into coronary sinus (80%) / RA*Hemodynamics*: -functional L-to-R shunt from pulmonary veins to right atrium-increased pulmonary blood flow(= overcirculation)-ASD restores oxygenated blood to left side-normal systemic venous pressure with increased flow through widened SVC-after birth CHF secondary to(a)mixture of systemic + pulmonary venous blood in RA(b)volume overload of RV ● cyanosis ● neck veins undistended (shunt level distally) ● R ventricular heave (= increased contact of enlarged RV with sternum) ● systolic ejection murmur (large shunt volume)√ "figure of 8" / "snowman" configuration of cardiac silhouette (= dilated SVC + left vertical vein)√ pretracheal density on lateral film (= left vertical vein)√ enlargement of RA + RV (= volume overload)√ normal LA (= ASD acts as escape valve)√ increased pulmonary blood flow (= overcirculation)√ absent connection of pulmonary veins to LA ***Sub- / Infradiaphragmatic TAPVR*** (12%) = Type III =drainage into portal vein / IVC / ductus venosus / left gastric vein with constriction of descending pulmonary vein by diaphragm en route through esophageal hiatus leading to pulmonary venous hypertension + RV pressure overload; >90% obstructed ● intense cyanosis + respiratory distress (R-to-L shunt through ASD)*Prognosis*:death within a few days of life*Associated with:*asplenia syndrome (80%), polysplenia√ unique appearance of pulmonary edema + pulmonary venous congestion with normal-sized heart (DDx: hyaline membrane disease)√ low anterior indentation on barium-filled esophagus ***Mixed Type Of TAPVR*** (6%) = Type IV =with various connections to R side of heart (6%)

Notes:

Partial Anomalous Pulmonary Venous Return = PAPVR May occur in isolation *Prevalence:*0.3-0.5% of patients with CHD*May be associated with:* (1)Atrial septal defect (25%)(a)RUL pulmonary vein enters SVC / RA (2/3)frequently associated with: sinus venosus type ASD (90%)√ RUL vein courses in a horizontal direction(b)LUL pulmonary vein enters brachiocephalic vein (1/3)*frequently associated with:* ostium secundum type ASD √ vertical mediastinal density lateral to aortic knob extending upward and medially with smooth curvilinear border (DDx: persistent left SVC)(2)Hypogenetic lung as a component of congenital pulmonary venolobar syndrome=SCIMITAR SYNDROME=part / all of the hypogenetic lung is drained by an anomalous veinAnomalous vein drains into: -IVC below right hemidiaphragm (33%)-suprahepatic portion of IVC (22%)-hepatic veins-portal vein (11%)-azygos vein-coronary sinus-right atrium (22%)-left atrium = "meandering pulmonary vein"◊ Drainage into suprahepatic portion of IVC / right atrium may be a clue for interruption of intrahepatic portion of IVC!*May be associated with:*systemic arterialization of the lung without sequestrationLocation:almost exclusively on right side√ tubular structure paralleling the right heart border in the configuration of a Turkish sword= "scimitar" (PA view) ● ASD symptomatologyCECT: √ nodular / tubular opacity (= anomalous vein), which opacifies in phase with pulmonary vein

Notes:

AORTIC ANEURYSM

Cause: 1.Atherosclerosis (73-80-90%):2.Traumatic (15-20%): following transection3.Congenital (2%): aortic sinus, post coarctation, ductus diverticulum4.Syphilis (19%): ascending aorta + arch5.Mycotic = bacterial dissection6.Cystic media necrosis (Marfan / Ehlers-Danlos syndrome, annuloaortic ectasia)7.Inflammation of media + adventitia:Takayasu arteritis, giant cell arteritis, relapsing polychondritis, rheumatic fever, rheumatoid arthritis, ankylosing spondylitis, Reiter syndrome, psoriasis, ulcerative colitis, systemic lupus erythematosus, scleroderma, Behçet disease, radiation 8.Increased pressure:systemic hypertension, aortic valve stenosis9.Abnormal volume load: severe aortic regurgitation TRUE ANEURYSM =permanent dilatation of all layers of weakened but intact wallFALSE ANEURYSM =focal perforation with all layers of wall disrupted; escaped blood contained by adventitia / perivascular connective tissue + organized bloodFUSIFORM ANEURYSM (80%) =circumferential involvementSACCULAR ANEURYSM =involvement of portion of wall

Abdominal Aortic Aneurysm (AAA) Atherosclerotic Aneurysm Degenerative Aneurysm Inflammatory Aortic Aneurysm Mycotic Aneurysm Syphilitic Aneurysm Thoracic Aortic Aneurysm Traumatic Aortic Pseudoaneurysm

Notes:

Abdominal Aortic Aneurysm (AAA) ◊There is no consensus regarding the definition of an atherosclerotic AAA!=focal widening >3 cm (ultrasound literature); twice the size of normal aorta / >4 cm (Bergan, Ann Surg 1984)Normal size of abdominal aorta >50 years of age: 12-19 mm in women; 14-21 mm in men *Prevalence:*1.4-8.2% in unselected population; in 6% >80 years of age; in 6-20% of patients with signs of atherosclerotic disease; M>F; Whites:Blacks = 3:1*Cause:*? genetic (10-fold increase in risk as first-degree relative of patient with AAA); structural defect of aortic wall caused by increased proteolysis; copper deficiency*Risk factors:*male sex, age >75 years, white race, prior vascular disease, hypertension, cigarette smoking, family history, hypercholesterolemia*Age:*>60 years; M:F = 5-9:1*Associated with*: (a)visceral + renal artery aneurysm (2%)(b)isolated iliac + femoral artery aneurysm (16%): common iliac (89%), internal iliac (10%), external iliac (1%)(c)stenosis / occlusion of celiac trunk / SMA (22%)(d)stenosis of renal artery (22-30%)(e)occlusion of inferior mesenteric artery (80%)(f)occlusion of lumbar arteries (78%)*Growth rate of aneurysm of 3-6 cm in diameter:* 0.39 cm / year ● asymptomatic (30%) ● abdominal mass (26%) ● abdominal pain (37%)◊Imaging should provide information about(a)the proximal extent of the aneurysm which determines the site of clamping of the aorta (origin of renal arteries)(b)the course of the left renal vein (retroaortic?)!Location:infrarenal (91-95%) with extension into iliac arteries (66-70%)Plain film:√ mural calcification (75-86%)US:√ >98% accuracy in size measurementNCCT: √ perianeurysmal fibrosis (10%), may cause ureteral obstruction√ "crescent sign" = peripheral high-attenuating crescent in aneurysm wall (= acute intramural hematoma) = **sign of impending rupture**
CECT: (a)ruptured aneurysm:√ anterior displacement of kidney√ extravasation of contrast material√ fluid collection / hematoma within posterior pararenal + perirenal spaces√ free intraperitoneal fluid√ perirenal "cobwebs"(b)contained leak√ laminated mural calcification√ periaortic mass of mixed / soft-tissue density√ lateral "draping" of aneurysm around vertebral body√ focal discontinuity of calcifications (unreliable)√ indistinct aortic wall (unreliable)Angio: √ focally widened aortic lumen >3 cm√ apparent normal size of lumen secondary to mural thrombus (11%)√ mural clot (80%)√ slow antegrade flow of contrast medium**Contained rupture** = extraluminal hematoma / cavity √ absent parenchymal stain = avascular halo√ displacement + stretching of aortic branches*Cx:* (1)Rupture (25%)(a)into retroperitoneum: commonly on left(b)into GI tract: massive GI hemorrhage(c)into IVC: rapid cardiac decompensation*Incidence:*aneurysm <4 cm in 10%, 4-5 cm in 23%, 5-7 cm in 25%, 7-10 cm in 46%, >10 cm in 60% ● sudden severe abdominal pain ± radiating into back ● faintness, syncope, hypotension*Prognosis:*64-94% die before reaching hospital*Increased risk:*size >6 cm, growth >5 mm / 6 months, pain + tenderness◊The exact moment of rupture is unpredictable!◊Cause of death in 1.3% of men >65 years!(2)Peripheral embolization(3)Infection(4)Spontaneous occlusion of aorta*Prognosis:*17% 5-year survival without surgery,50-60% 5-year survival with surgery *Rx:*surgery recommended if >5 cm in diameter;4-5% surgical mortality for nonruptured, 30-80% for ruptured aneurysm*Postoperative Cx:*(1)Left colonic ischemia (1.6%) with 10% mortality(2)Renal failure (14%)(3)0-8% mortality rate for elective surgery

Notes:

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Atherosclerotic Aneurysm *Incidence:*leading cause of thoracic aortic aneurysm*Histo:*diseased intima with secondary degeneration + fibrous replacement of media; ultimately wall of aneurysm composed of acellular + avascular connective tissue*Pathophysiology:* progressive weakening of media results in vessel dilatation + increased tension of vessel wall (law of Laplace = tensile stress varies with product of blood pressure and radius of vessel); compromise of mural vascular nutrition (vasa vasorum) causes further degeneration + progressive dilatation *Age:*elderly; M > FLocation:distal abdominal aorta > iliac a. > popliteal a. > common femoral a. > aortic + descending thoracic aorta > carotid a.Site:(1)infrarenal aorta (associated with thoracic aneurysm in 29%)(2)descending thoracic aorta distal to left subclavian artery(3)thoracoabdominal√ fusiform (80%), saccular (20%)*Cx:*rupture (cause of death in 50%): usually unrestrained + fatal in thoracic location

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Notes:

Degenerative Aneurysm =medial degenerationMost common cause of aneurysm in ascending aorta *Cause:*(1)genetically transmitted metabolic disorder: Marfan syndrome, Ehlers-Danlos syndrome(2)acquired: result of repetitive aortic injury + repair associated with aging

Notes:

Inflammatory Aortic Aneurysm =defined as triad of(1)thickened aneurysm wall(2)extensive perianeurysmal + retroperitoneal fibrosis(3)dense adhesions of adjacent abdominal organs*Frequency:*3-10% of all AAAs; M:F = 6:1 to 30:1*Mean age:*62-68 years ● abdominal / back pain ● weight loss + anorexia (20-41%) ● elevated ESR (40-88%) ● tender pulsatile abdominal mass (15-30%)*Comorbidities:*arterial hypertension (34-69%), arterial occlusive disease (10-47%), diabetes mellitus (3-13%), coronary artery disease (33-55%)√ entrapment of ureters (10-21%)√ sonolucent halo around aorta*Cx:*enlargement + rupture (lower rate than in noninflammatory aneurysm)

Notes:

Mycotic Aneurysm *Incidence:*2.6% of all abdominal aneurysmsA.PRIMARY MYCOTIC ANEURYSM (rare)unassociated with any demonstrable intravascular inflammatory process B.SECONDARY MYCOTIC ANEURYSM=aneurysm due to nonsyphilitic infection*Predisposing factors:* (1) IV drug abuse (2) bacterial endocarditis (12%) (3) immunocompromise (malignancy, alcoholism, steroids, chemotherapy, autoimmune disease, diabetes) (4) atherosclerosis (5) aortic trauma caused by accidents / aortic valve surgery / coronary artery bypass surgery / arterial catheterization *Mechanism:* (a)septicemia with abscess formation via vasa vasorum(b)septicemia with abscess formation via vessel lumen(c)direct extension of contiguous infection(d)preexisting intima laceration (trauma, atherosclerosis, coarctation)*Organism:*S. aureus (53%), Salmonella (33-50%), nonhemolytic Streptococcus, Pneumococcus, Gonococcus, Mycobacterium (contiguous spread from spine / lymph nodes)*Histo:*loss of intima + destruction of internal elastic lamella; varying degrees of destruction of muscularis of media + adventitia ● frequently insidious, fever ● positive blood culture in 50%Site:ascending aorta > abdominal visceral artery > intracranial artery > lower / upper extremity artery√ true aneurysm (majority)√ saccular structure arising eccentrically from aortic wall with rapid enlargement√ interrupted ring of aortic wall calcification√ periaortic gas collection√ adjacent vertebral osteomyelitis√ adjacent reactive lymph node enlargement*Cx:*(1)life-threatening rupture + hemorrhage (75%)(2)uncontrolled sepsis if untreated*Prognosis:*67% overall mortality

Notes:

Syphilitic Aneurysm Spectrum: 1.Uncomplicated syphilitic aortitis2.Syphilitic aortic aneurysm (mostly saccular)3.Syphilitic aortic vasculitis (aortic regurgitation)*Incidence:*12% of patients with untreated syphilis*Onset:*10-30 years after initial spirochete infection*Histo:*chronic inflammation of aortic adventitia + media beginning at vasa vasorum + leading to obstruction of vasa vasorum followed by nutritional impairment of media + loss of elastic fibers + smooth muscle fibers ● positive venereal disease research laboratory (VDRL) test ● positive microhemagglutination assay - Treponema pallidum (MHA-TP) testLocation:ascending aorta (36%), aortic arch (34%), proximal descending aorta (25%), distal descending aorta (5%), aortic sinuses (<1%)√ asymmetric enlargement of aortic sinuses (DDx to medial degeneration with symmetric enlargement)√ saccular (75%) / fusiform (25%) aneurysm√ pencil-thin dystrophic aortic wall calcification (up to 40%) most severe in ascending aorta, frequently obscured by thick coarse irregular calcifications of secondary atherosclerosis*Prognosis:*death in 2%, rupture in up to 40%; death within months of onset of symptoms if untreated

Notes:

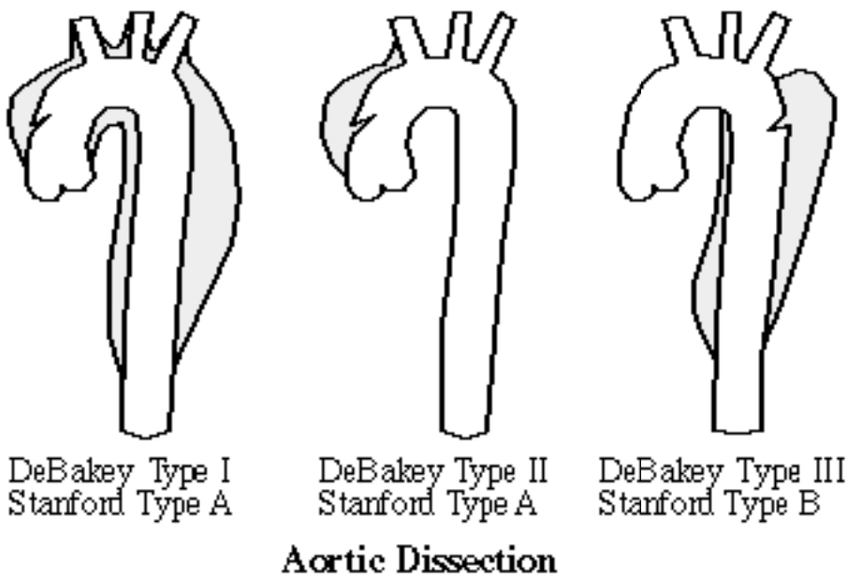
Thoracic Aortic Aneurysm Most common vascular cause of mediastinal mass! ◊10% of mediastinal masses are of vascular origin!Average diameter of thoracic aorta (<4-5 cm wide): -aortic root:3.6 cm-ascending aorta 1 cm proximal to arch:3.5 cm-proximal descending aorta:2.6 cm-middle descending aorta:2.5 cm-distal descending aorta:2.4 cm*Associated with:*hypertension, coronary artery disease, abdominal aneurysm*Mean age:*65 years; M:F = 3:1 ● substernal / back / shoulder pain (26%) ● SVC syndrome (venous compression) ● dysphagia (esophageal compression) ● stridor, dyspnea (tracheobronchial compression) ● hoarseness (recurrent laryngeal nerve compression)√ mediastinal mass with proximity to aorta√ wide tortuous aorta√ curvilinear peripheral calcifications (75%)√ circumferential / crescentic mural thrombus√ Angio: may show normal caliber secondary to mural thrombus*Cx:*(1)Rupture into mediastinum, pericardium, either pleural sac, extrapleural space√ high-attenuation fluid(2)Aortobronchopulmonary fistula√ consolidation of lung adjacent to aneurysm◊Most aneurysms rupture when >10 cm in size*Prognosis:*1-year survival 57%, 3-year survival 26%, 5-year survival 19% (60% die from ruptured aneurysm, 40% die from other causes)*Surgical mortality:*10%

Notes:

Traumatic Aortic Pseudoaneurysm =CHRONIC AORTIC PSEUDOANEURYSM◊2nd most common form of thoracic aortic aneurysm;◊most common type occurring in young patients*Incidence:*2.5% of patients who survive initial trauma of acute aortic transection√ usually calcified√ may contain thrombus*Cx:*(1)progressive enlargement (2)rupture (even years after insult)

Notes:

AORTIC DISSECTION
=spontaneous longitudinal separation of aortic intima + adventitia by circulating blood having gained access to the media of the aortic wall splitting it in two*Path:*(a)transverse tear in weakened intima (95-97%)(b)no intimal tear (3-5%) = INTRAMURAL HEMATOMA OF AORTA*Pathogenesis:* intimal tear results from combination of following factors: (1)medial degeneration decreases cohesiveness within aortic wall(2)persistent aortic motion secondary to beating heart results in stress within aortic wall(3)hydrodynamic forces accentuated by hypertension*Incidence:*3:1,000 (more common than all ruptures of thoracic + abdominal aorta combined); 1:205 autopsies; 2,000 cases/year in United States*Peak age:*60 years (range 13-87 years); M:F = 3:1*Predisposed:*(cystic medial necrosis / disease of aortic wall)◊Starts in fusiform aneurysms in 28%◊Does not occur in aneurysms <5 cm in diameter1.Hypertension (60-90%) 9.Bicuspid aortic valve2.Marfan syndrome (16%)10.S/P prosthetic valve 3.Ehlers-Danlos syndrome11.Trauma (rare)4.Relapsing polychondritis12.Catheterization5.Valvular aortic stenosis13.Pregnancy6.Turner syndrome14.Aortitis (eg, SLE)7.Behçet disease15.Cocaine abuse8.CoarctationNOT syphilis◊In women 50% of dissections occur during pregnancy! ● sharp tearing intractable anterior / posterior chest pain (75-95%) radiating to jaw, neck, low back(DDx: myocardial infarction) ● murmur ± bruit (65%) from aortic regurgitation ● asymmetric peripheral pulses + blood pressures (59%) ● absent femoral pulses (25%), reappearing after reentry ● pulse deficit: in up to 50% of type A dissection, in 16% of type B dissection ● hemodynamic shock (25%) ● neurologic deficits (25%): hemiplegia, paraparesis (due to compromise of anterior spinal artery of Adamkiewicz) ● persistent oliguria ● congestive heart failure (rare) due to acute aortic insufficiency ● recurrent arrhythmias / right bundle branch block ● signs of pericardial tamponade: clouded sensorium, extreme restlessness, dyspnea, distended neck veins *Types:* DeBakey Type I(29-34%)=ascending aorta + portion distal to archDeBakey Type II(12-21%)=ascending aorta onlyDeBakey Type III(50%)=descending aorta onlySubtype IIIA=up to diaphragmSubtype IIIB=below diaphragm Stanford Type A (70%)=ascending aorta ± arch in first 4 cm in 90%Stanford Type B (20-30%)=descending aorta only*mnemonic:***Aa**ffects **a**scending aorta and **a**rch; **Bb**egins **b**eyond **b**rachiocephalic vessels!



Aortic Dissection

Clinical classification: (1)Acute aortic dissection:<2 weeks old(2)Chronic aortic dissection:>2 weeks oldLocation of dissection (following helical flow pattern): -on anterior + right lateral wall of ascending aorta just distal to aortic valve (65%)-on superior + posterior wall of transverse aortic arch (10%)-on posterior + left lateral wall of upper descending aorta distal to left subclavian artery (20%)-more distal aorta (5%) usually terminating in left iliac artery (80%) / right iliac artery (10%) [involvement of left renal artery in 50%]◊An exit / distal tear / reentry occurs in 10%! CXR (best assessment from comparison with serial films): √ normal CXR in 25%√ "calcification sign" = inward displacement of atherosclerotic plaque by >4-10 mm from outer aortic contour (7%), can only be applied to contour of descending aorta secondary to projection, may be misleading in presence of periaortic soft-tissue mass / hematoma√ disparity in size between ascending + descending aorta√ irregular wavy contour / indistinct outline of aorta√ widening of superior mediastinum to >8 cm due to hemorrhage / large false channel (40-80%)√ cardiac enlargement (LV hypertrophy / hemopericardium)√ left pleural effusion (27%)√ atelectasis of lower lobe√ rightward displacement of trachea / endotracheal tubeECHO: (a)transthoracic US: 59-85% sensitive + 63-96% specific for type A dissection; poorer for type B(b)transesophageal US: up to 99% sensitive + 77-97% specific(c)intravascular in conjunction with aortography to differentiate true from false lumen√ intimal flap (seen in more than one view)√ pericardial fluid√ aortic insufficiencyFalse-positives:reverberation echoes from aneurysmal ascending aorta / calcified atheromatous plaque, postoperative periaortic hematoma Angio(86-88% sensitive, 75-94% specific):◊Aortography 1st choice for final confirmation + staging because of contrast limitation!Superior to any other technique in demonstrating -entry + reentry points (in 50%)-branch vessel involvement + coronary arteries-aortic insufficiency√ visualization of intimal / medial flap (75-79%) = linear radiolucency within opacified aorta√ "double barrel aorta" (87%) = opacification of two aortic lumens√ abnormal catheter position outside anticipated aortic course√ compression of true lumen by false channel (72-85%)√ aortic valvular regurgitation (30%)√ increase in aortic wall thickness >6-10 mm√ obstruction of aortic branches: left renal artery (25-30%)√ ulcerlike projections caused by truncated branches√ slower blood flow in false lumenFalse-negative: complete thrombosis of false channel (10%), intimal flap not tangential to x-ray beam False-positive: thickening of aortic wall due to aneurysm, aortitis, adjacent neoplasm / hemorrhage CECT (87-94% sensitive, 87-100% specific): within 4 hours (if patient responds rapidly to medical Rx); detection as accurate as angio with single-level dynamic scanning√ crescentic high-attenuation clot within false lumen√ internally displaced intimal calcification (DDx: calcification of thrombus on luminal surface or within)√ intimal flap separating two aortic channels (may be seen without contrast in anemic patients)False-negative: inadequate contrast opacification, thrombosed lumen misinterpreted as aortic aneurysm with mural thrombus False-positive: streak artifacts secondary to cardiac / aortic motion, opacified normal sinus of Valsalva, normal pericardial recess mistaken for thrombus MR (95-100% sensitive, 90-100% specific): √ intimal flap of medium intensity outlined by signal voids of rapidly flowing blood√ intimal flap more difficult to detect in presence of slow flow / thrombus√ "cobwebs" (= bands of medial elastic lamellae spanning the junction of the dissecting septum with the outer wall of the false lumen) mark the false lumen in 80%*Cx:*(1)Retrograde dissection(a)aortic insufficiency(b)occlusion of coronary artery (8%)(c)rupture into pericardial sac / pleural space: 70% mortality(d)rupture into RV, LA, vena cava, pulmonary artery producing large L-to-R shunt(2)occlusion / transient obstruction of major aortic branches (30%)(3)rupture of aorta(4)development of saccular aneurysm requiring surgery (15%)◊Organs may receive their blood supply through either the true or false lumen or both!*Rx:*(1)Reducing peak systolic pressure to 120-70 mm Hg (adequate alone for Type III = B, which rarely progresses proximally): death from rupture of aortic aneurysm in 46% of hypertensive + 17% of normotensive patients(2) Immediate surgical graft reinforcement of aortic wall (Type I, II = A) preventing rupture + progressive aortic valve insufficiency*Prognosis without Rx:* immediate death (3%); death within: 1 day (20-30%), 1 week (50-62%), 3 weeks (60%), 1 month (75%), 3 months (80%), 1 year (80-95%) *Prognosis with Rx:* 5-10% mortality rate following timely surgery; 40% 10-year survival rate after leaving hospital *DDx:*Penetrating ulcer of thoracic aorta (= atherosclerotic lesion of mid-descending aorta with ulceration extending through intima into aortic media)

Notes:

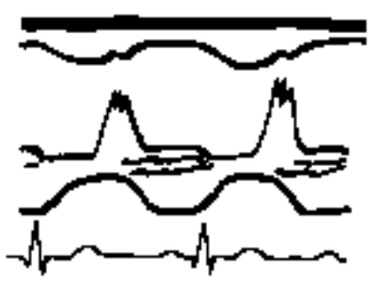
AORTIC GRAFT INFECTION

Classification: (1)PERIGRAFT INFECTION (2-6%) ● fever, chills, leukocytosis ● groin swelling / drainage(2)AORTOENTERIC FISTULA (0.6-2%) ● acute / chronic GI bleeding (may be occult) ● sepsisNormal postoperative course: ◊complete resolution of hematoma by 2-3 months◊disappearance of ectopic gas by 3-4 weeksCT (94% sensitive, 85% specific, 91% accurate): √ perigraft soft tissue√ ectopic gas (fistulous communication with bowel / gas-producing organism)√ focal bowel wall thickening (indicates fistula)√ >5 mm soft tissue between graft + surrounding wrap (beyond 7th postoperative week)√ focal discontinuity of calcified aneurysmal wrap*False positives:* perigraft hematoma in early postoperative period, pseudoaneurysm (in 15-20%) *Prognosis:*17-75% mortality; 30-50% morbidity

Notes:

AORTIC REGURGITATION

=AORTIC INSUFFICIENCY*Cause:* A.INTRINSIC AORTIC VALVE DISEASE1.Congenital bicuspid valve2.Rheumatic endocarditis3.Bacterial endocarditis (perforation / prolapse of cusp)4.Myxomatous valve associated with cystic medial necrosis5.Aortic valve prolapse6.Prosthetic valve: mechanical break, thrombosis, paravalvular leakB.PRIMARY DISEASE OF ASCENDING AORTA(a)Dilatation of aortic annulus1.Syphilitic aortitis2.Ankylosing spondylitis (5-10%)3.Reiter disease4.Rheumatoid arthritis5.Cystic medial necrosis: Marfan syndrome(b)Laceration = aortic dissection1.Deceleration trauma2.Hypertension *Pathogenesis:*progressive enlargement of diastolic + systolic LV dimensions result in increase in myocardial fiber length + increase in stroke volume; decompensation occurs if critical limit of fiber length is reached ● "water-hammer pulse" = twin-peaked pulse ● systolic ejection murmur + high-pitched diastolic murmur ● Austin Flint murmur = soft mid-diastolic or presystolic bruit√ LV enlargement (cardiothoracic ratio >0.55) + initially normal pulmonary vascularity (DDx: congestive cardiomyopathy, pericardial effusion)√ normal aorta (in intrinsic valve disease)√ dilatation ± calcification of ascending aorta (in aortic wall disease)√ tortuous descending aorta√ increased pulsations along entire aorta ECHO: √ aortic root dilatation√ high frequency flutter of aML (occasionally pML) during first 2/3 of diastole (CHARACTERISTIC)√ high frequency diastolic flutter of IVS (uncommon)√ diastolic flutter of aortic valve (SPECIFIC, but rare)√ premature aortic valve opening (high diastolic LV pressure)√ decreased MV opening (aML pushed posteriorly by regurgitant aortic jet)√ premature closure of mitral valve (high diastolic LV pressure produces MV closure before beginning of systole in severe acute aortic insufficiency)√ LV dilatation + large amplitude of LV wall motion (volume overload, increased ejection fraction):End-systolic LV diameterAction<50 mmyearly follow-up50-54 mm4- to 6-month follow-up>55 mmvalve replacement Doppler: √ slope of peak diastolic to end-diastolic velocity decrease >3 m/sec^2 in severe aortic regurgitation√ area of color Doppler regurgitant flow√ ratio of width of regurgitant beam to width of aortic root is good predictor of severity (color Doppler)



Mitral Valve in Severe Aortic Regurgitation

The valve is almost completely closed before onset of ventricular systole. Atrial contraction has little effect in reopening the valve. Complete closure occurs with ventricular systole. A high-velocity flutter of aML is present in diastole.

Notes:

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AORTIC RUPTURE
=blood leakage through aneurysmatic aortic wall*Pathogenesis:*small clefts occur at a fragile site within inner thrombus gradually expanding to outer layer of thrombus with gradual seepage of flowing blood into mural thrombus and aneurysmal wallCT: high-attenuation crescent sign (71%)

Notes:

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AORTIC STENOSIS

Aorta valve area decreased to <0.8 cm^2 = 0.4 cm^2/m^2 BSA (normal 2.5-3.5 cm^2) A.ACQUIRED AORTIC STENOSIS1.Rheumatic valvulitis (almost invariably associated with mitral valve disease)2.Fibrocalcific senile aortic stenosis (degenerative)B.CONGENITAL AORTIC STENOSIS (most common)=most frequent CHD associated with IUGR1.Subvalvular AS (30%)2.Valvular AS (70%): degeneration of bicuspid valve most common cause3.Supravalvular AS*Pathogenesis:*increased gradient across valve produces LV hypertrophy and diminished LV compliance; increased muscle mass may outstrip coronary blood supply (subendocardial myocardial ischemia with angina); LV decompensation leads to LV dilatation + pulmonary venous congestion ● asymptomatic for many years ● angina, syncope, heart failure ● systolic murmur ● carotid pulsus parvus et tardus ● diminished aortic component of 2nd heart sound ● sudden death in severe stenosis (20%) after exercise (diminished flow in coronary arteries causes ventricular dysrhythmias + fibrillation)√ poststenotic dilatation of ascending aorta (in 90% of acquired, in 70% of congenital AS)√ normal-sized / enlarged LV (small LV chamber with thick walls)@in adults >30 years√ calcification of aortic valve (best seen on RAO); indicates gradient >50 mm Hg√ discrete enlargement of ascending aorta (NO correlation with severity of stenosis)√ calcification of mitral annulus√ "left ventricular configuration" = concavity along mid left lateral heart border + increased convexity along lower left lateral heart border@in children / young adults√ prominent ascending aorta√ left ventricular heart configuration@in infancy:√ left ventricular stress syndromeECHO: √ thickened + calcified aortic valve with multiple dense cusp echoes throughout cardiac cycle (right > noncoronary > left coronary cusp)√ decreased separation of leaflets in systole with reduced opening orifice (13-14 mm = mild AS; 8-12 mm = moderate AS; <8 mm = severe AS)√ ± doming in systole√ dilated aortic root√ increased thickness of LV wall (= concentric LV hypertrophy)√ hyperdynamic contraction of LV (in compensated state)√ decreased mitral EF slope (reduced LV compliance)√ LA enlargement√ increased aortic valve gradient (Doppler)√ decreased aortic valve area (unreliable)*DDx:*calcification of aortic annulus in elderly / calcified coronary artery ostium (thickened cusp echoes only in diastole) *Prognosis:*depends on symptomatology (angina, syncope, CHF) secondary to subvalvular obstruction

Aortic Valve in Hypertrophic Subaortic Stenosisduring midsystole the aortic valve closes

Subvalvular Aortic Stenosis Valvular Aortic Stenosis Supravalvular Aortic Stenosis

Notes:

Subvalvular Aortic Stenosis =SUBAORTIC STENOSIS(a)Anatomic / fixed subaortic stenosis*Associated with:* cardiac defects in 50% (usually VSD)Type I:thin 1-2 mm membranous diaphragmatic stenosis, usually located within 2 cm or less of valve annulusType II:thick collarlike stenosisType III:irregular fibromuscular stenosisType IV:"tunnel subaortic stenosis" = fixed tunnel-like narrowing of LVOT = excessive thickening of only upper ventricular septum with normal mitral valve motion(b)Functional / dynamic subaortic stenosis1.Asymmetric septal hypertrophy (ASH)2.Idiopathic hypertrophic subaortic stenosis (IHSS)3.Hypertrophic obstructive cardiomyopathy (HOCM)may occur in infants of diabetic mothers √ asymmetrically thicker ventricular septum than free wall of LV (95%)√ normal / small left + right ventricular cavities (95%)√ systolic anterior motion of mitral valve√ lucent subaortic filling defect in systoleECHO: √ coarse systolic flutter of valve cusps√ opening of leaflets followed by rapid inward move in mid systole, leaflets may remain in partially closed position through latter portion of systole (to appose borders of the flow jet) *Cx:*mitral regurgitation (secondary to abnormal position of anterolateral papillary muscle preventing complete closure of MV in systole)

Notes:

Valvular Aortic Stenosis =fusion of commissures between cusps*Congenital types:* (a)bicuspid / unicuspid (in 95%): in 1-2% of population; M > F; commonly associated with coarctation of the aorta(b)tricuspid (5%)(c)dysplastic thickened aortic cusps√ valvular calcifications (in 60% of patients >24 years of age)@IN INFANT with critical aortic stenosis: ● intractable CHF in first days / weeks of life with severe dyspnea ● may simulate neonatal sepsis*Associated with:* L-to-R shunts (ASD, VSD)√ marked cardiomegaly (thickened wall of LV)√ pulmonary venous hypertension√ decreased ejection fraction√ doming of thickened valve cusps√ dilated ascending aorta*Rx:*emergency surgical dilatation@IN CHILD: ● asymptomatic until late in life√ normal pulmonary vascularity√ LV configuration with normal size of heart√ large posterior noncoronary cusp, smaller fused right + left cusps√ doming of thickened valve cusps√ eccentric jet of contrast√ poststenotic dilatation of ascending aortaECHO: √ increase in echoes from thickened deformed leaflets (maximal during diastole)√ decrease in leaflet separation

Aortic Valvular Stenosis

decrease separation of thickened deformed leaflets

Notes:

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Supravalvular Aortic Stenosis *Types:* (a)localized hourglass narrowing just above aortic sinuses(b)discrete fibrous membrane above sinuses of Valsalva(c)diffuse tubular hypoplasia of ascending aorta + branching arteries*Associated with:*peripheral PS, valvular + discrete subvalvular AS, Marfan syndrome, Williams syndrome dilatation + tortuosity of coronary arteries (may undergo early atherosclerotic degeneration secondary to high pressure)ECHO: narrowing of supravalvular aortic area (normal root diameter: 20-37 mm) normal movement of cusps

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Notes:

AORTIC TRANSECTION

=TRAUMATIC AORTIC RUPTURE = aortic tear from rapid horizontal deceleration / blunt chest trauma*Pathophysiology:* 1.Incomplete rupture (15%)(a)intimal hemorrhage without tear(b)transverse tear of intima(c)tear into media with subadventitial accumulation of blood (40-60%) = false aneurysm◊Aorta goes on to rupture completely within 24 hours in 50% of patients!2.Complete rupture (85%) with exsanguination before reaching a hospital3.Periaortic hemorrhage ± aortic injury ● interscapular severe chest pain, dyspnea, dysphagia ● hypertension of upper extremities = acute traumatic coarctation ● bilateral femoral pulse deficit ● systolic murmur in 2nd left parasternal interspaceSite:(a)Aortic isthmus just distal to left subclavian artery (88-95%): brachiocephalic arteries + ligamentum arteriosum fix aorta in this region(b)Aortic arch with avulsion of brachiocephalic trunk (4.5%)(c)Ascending aorta immediately above aortic valve (1%)*Cx:*aortic valve rupture, coronary artery laceration, hemopericardium + cardiac tamponade; NO mediastinal hematoma(d)Descending aorta (1.8%)CXR: N.B.:There are no plain CXR findings of aortic injury (since aortic integrity is maintained by intact adventitia)! The source of mediastinal hematoma are frequently the azygos, hemiazygos, paraspinal and intercostal vessels!◊Aortic injury is the cause of mediastinal hematoma in only 12.5%!√ normal admission CXR in 28% (radiographic signs may not develop until 6-36 hours): 96% NPV for supine CXR Most specific signs: √ deviation of nasogastric tube to the right of T4 spinous process (67%)√ depression of left mainstem bronchus anteroinferiorly >40° below the horizontal + toward right (53%)√ mediastinal width >8 cm at level of aortic knob (75%): 53-100% sensitive, 1-60% specific√ mediastinal width to chest width >0.25√ obscuration / irregularity of aortic arch contour (75%)√ leftward displacement of left mediastinal stripe abnormally extending above the level of aortic arch forming a left apical cap√ thickening of right paratracheal stripe >4-5 mm(= hematoma between pleura + trachea)√ left / right "apical cap" sign in 37% (= extrapleural hematoma along brachiocephalic vessels)√ opacification of aortopulmonary window√ loss of contour of descending aorta√ widening of left paraspinal interface >5 mm√ tracheal compression + displacement toward right (61%)√ rapidly accumulating commonly left-sided hemothorax without evident rib fracture (break in mediastinal pleura)√ fractures of 1st + 2nd rib (17%) *mnemonic:*"BAD MEAT"**B**ronchus depression (left main) **A**ortic silhouette shaggy **D**eath in 80-90% **M**ediastinal widening **E**nteric (nasogastric) tube displacement **A**pical cap **T**racheal shift NECT screening (55% sensitive, 65% specific): √ obliteration of aorta-fat interface with increased attenuation (= mediastinal hematoma)◊A negative CT examination for mediastinal hemorrhage has an almost 100% NPV for aortic injury!◊All patients with periaortic / middle / superior mediastinal hemorrhage require aortography! Save your contrast for that study!CECT: √ abrupt change in aortic contour at inner aortic wall √ aortic pseudoaneurysm√ intimal flap√ pseudocoarctation = diminished caliber of the descending aorta√ extravasation of contrast materialFalse positive: residual thymic tissue, atelectatic lung, pericardial recess, patient motion, streak artifacts, partial volume effect with pulmonary artery Angio (definitive means for diagnosis): True positive: In 20% of patients with mediastinal hematoma angio demonstrates acute traumatic aortic injury! √ traumatic false aneurysm√ tear of intima (5-10%) / media√ rupture with extravasation of contrast material√ posttraumatic dissection (11%)√ posttraumatic coarctation*DDx:*ductus diverticulum (in 10% of normals), aortic spindle, infundibula of brachiocephalic arterial branches, atherosclerotic aortic ulceration *Recommendation for work-up:* (1)normal well-defined mediastinal contours on CXR: no further imaging(2)unequivocally abnormal mediastinum on CXR: angiography (±for other reasons)(3)Clinically stable patient with equivocal CXR: CECT of thorax*Prognosis:* (1)70-85% fatal at scene of trauma(2)15-30% reach hospital (due to formation of periaortic hematoma + false aneurysm contained by adventitia ± surrounding connective tissue)(a)with surgical repair: 60-70% survive(b)no intervention: 80% dead within 1 hour; 85% dead within 24 hours, 98% dead within 10 weeks; chronic false aneurysm may develop in 2-5% at isthmus / descending aorta

Chronic Posttraumatic Aortic Pseudoaneurysm

Notes:

Chronic Posttraumatic Aortic Pseudoaneurysm =aneurysm existing for >3 months (amount of wall fibroplasia following rupture usually not sufficient to prevent subsequent rupture until at least 3 months after initial traumatic episode)*Incidence:*2-5% of patients surviving aortic transection >24-48 hours ● symptom-free period of months to years (in 11% >10 years) ● delayed clinical symptoms (42% within 5 years, 85% within 20 years): chest pain, back pain, dyspnea, cough, hoarseness, dysphagia, systolic murmur*Location:*descending aorta at level of lig. arteriosum filling the aorticopulmonary window (most commonly)√ well-defined rounded mass in left paramediastinal region√ ± inferior displacement of left mainstem bronchus*Cx:*CHF, partial obstruction of aortic lumen, bacterial endocarditis, aortoesophageal fistula, aortic dissection, obstruction of tracheobronchial tree, systemic emboli*Prognosis:*enlargement + eventual rupture;10-year survival rate:85% with surgical repair,66% without surgical repair

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Notes:

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AORTOPULMONIC WINDOW
=defect in septation process characterized by large round / oval communication between left wall of ascending aorta + right wall of pulmonary trunk ● clinically resembles PDACXR: √ shunt vascularity√ cardiomegaly (LA + LV enlarged)√ diminutive aortic knob√ prominent pulmonary trunkAngio (left ventriculogram / aortogram in AP / LAO projection): √ defect several mm above aortic valve√ pulmonary valve identified (DDx to truncus arteriosus)

Notes:

ARTERIOSCLEROSIS OBLITERANS

=ASO = hardening of the arteries*Prevalence:*2.4 million people in U.S.; in 1978 12% of autopsies had ASO as leading cause of death (excluding MI)*Etiology:*unknown*Contributing factors:* aging, diabetes (16-44%), hypertension, atherosclerosis Effect of hyperlipidemia: (a)High-density lipoproteins (HDL) have a protective effect: carry 25% of blood cholesterol(b)Low-density lipoproteins (LDL): carry 60% of blood cholesterol*Histo:*deposition of lipids, blood products, carbohydrates, begins as disruption of intimal surface; fatty streaks (as early as childhood); fibrous plaques (as early as 3rd decade); thrombosis, ulceration, calcification, aneurysm*Age:*50-70 years; M > F (after menopause)*Clinical classification:* (1)intermittent claudication = ischemic symptoms with exercise: calf, thigh, hip, buttock(2)ischemic symptoms at rest (indicative of multisegment disease) ● cramping / burning / aching pain ● cold extremity ● paresthesia ● trophic changes: hair loss, thickened nails ● ulcer, gangrene ● decreased / absent pulsesLocation:medium + large arteries; frequently at bifurcations; most frequent:-superficial femoral artery in adductor canal (diabetics + nondiabetics)-aortoiliac segment (nondiabetics)-tibioperoneal trunk (diabetics)*Prognosis:* accelerated by diabetes (34% will require amputation), hypertension, lipoprotein abnormalities, heart disease (decreased cardiac output resulting in increased blood viscosity from polycythemia), chronic addiction to tobacco (11.4% will require amputation), intermittent claudication (5-7% require amputation if nondiabetic = 1-2% per year), ischemic ulcer / rest pain (19.6% require amputation)

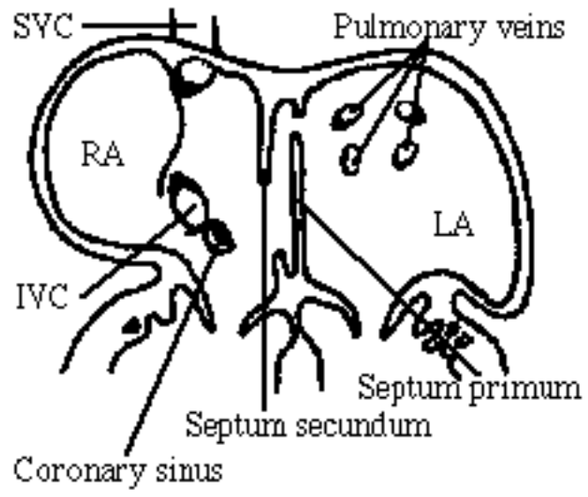
Notes:

ASPLENIA SYNDROME
=BILATERAL RIGHT-SIDEDNESS= IVEMARK SYNDROME*Incidence:*1:1,750 to 1:40,000 livebirths; M > F*Associated with:* (a)CHD (in 50%):TAPVR (almost 100%), endocardial cushion defect (85%), single ventricle (51%), TGA (58%), pulmonary stenosis / atresia (70%), dextrocardia (42%), mesocardia, VSD, ASD, absent coronary sinus, common atrium, common hepatic vein(b)GI anomalies:Partial / total situs inversus, annular pancreas, agenesis of gallbladder, ectopic liver, esophageal varices, duplication + hypoplasia of stomach, Hirschsprung disease, hindgut duplication, imperforate anus(c)GU anomalies (15%):Horseshoe kidney, double collecting system, hydroureter, cystic kidney, fused / horseshoe adrenal, absent left adrenal, bilobed urinary bladder, bicornuate uterus(d)Cleft lip / palate, scoliosis, single umbilical artery, lumbar myelomeningocele ● cyanosis in neonatal period / infancy (if severe cyanotic CHD) ● Howell-Jolly bodies = RBC inclusions in patients with absent spleen√ absent spleen@Lung√ bilateral trilobed lungs = bilateral minor fissures (SPECIFIC)√ bilateral eparterial bronchi (tomogram) = pulmonary arteries inferior to bronchi on PA view + projecting anterior to trachea on LAT view√ diminished pulmonary vascularity / pulmonary venous hypertension (TAPVR below diaphragm)√ bilateral SVC√ bilateral right atrial appendages@Abdomen√ absent spleen√ centrally located liver = hepatic symmetry√ stomach on right / left side / in central position√ juxtaposed IVC ("piggybacked") to aorta = abdominal aorta + IVC located on same side of spine (aorta usually posterior) (NEARLY PATHOGNOMONIC)*Prognosis:*80% mortality by end of 1st year of life

Notes:

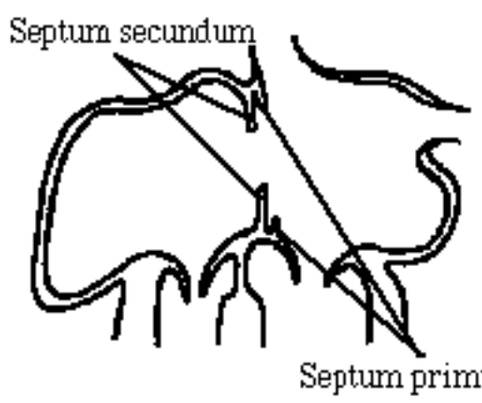
ATRIAL SEPTAL DEFECT

Most common congenital cardiac defect in subjects >20 years of age *Incidence:*8-14% of all CHD; M:F = 1:4*Age:*presentation frequently > age 40 secondary to benign course(a)mildly symptomatic (60%): dyspnea, fatigue, palpitations(b)severely symptomatic (30%): cyanosis, heart failure*Embryology:* 1.Septum primum = membrane growing from atrial walls toward endocardial cushion2.Ostium primum = temporary orifice between septum primum + endocardial cushion, which becomes obliterated by 5th week3.Ostium secundum = multiple small coalescing perforations in septum primum4.Septum secundum = membrane developing on right side of septum primum + covering part of ostium secundum5.Foramen ovale = orifice limited by septum secundum + septum primum6.Foramen ovale flap = lower edge of septum

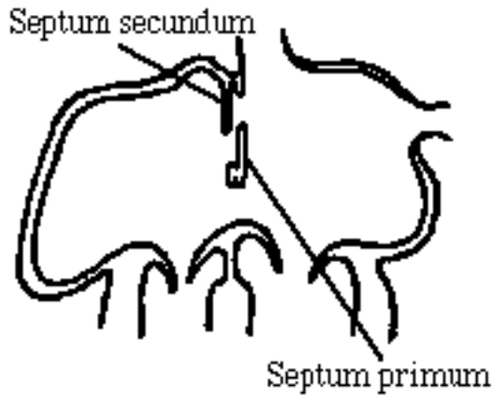


primum (foramen ovale patent in 6%, probe-patent in 25%; not considered an ASD) **Normal Newborn Heart**Atrial septum consists of two components (a) right side: septum secundum (muscular, firm) with posterior opening = foramen ovale (b) left side: septum primum (fibrous, thin) with anterior opening = ostium secundum

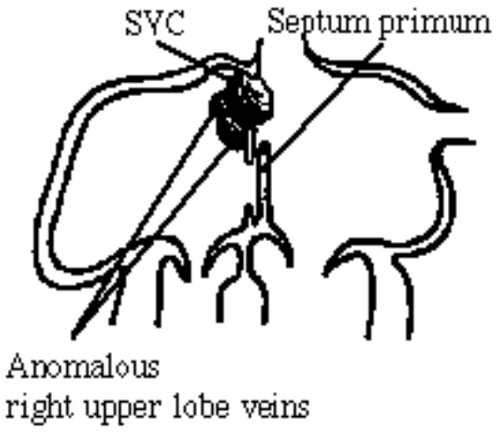
A.OSTIUM SECUNDUM ASD (60-70%)



=exaggerated resorptive process of septum primum leads to absence / fenestration of the foramen ovale flapLocation:in the body of the atrial chamber at fossa ovalisSize:large defect of 1-3 cm in diameter*May be associated with:* prolapsing mitral valve, pulmonary valve stenosis, tricuspid



atresia, TAPVR, hypoplastic left heart, interrupted aortic arch B.OSTIUM PRIMUM ASD (30%) =defect of atrioventricular endocardial cushionLocation:inferior to fossa ovalis at outlet portion of atrial septum*Almost always associated with:* endocardial cushion defects, cleft mitral valve,



anterior fascicular block C.SINUS VENOSUS ASD (5%) =defect of the superior inlet portion of the atrial septumLocation:superior to fossa ovalis near entrance of superior vena cava (SVC straddles ASD)*Associated with:*partial anomalous pulmonary venous return in 90% (RUL pulmonary veins connect to SVC / right atrium), Holt-Oram syndrome, Ellis-van Creveld syndrome D.LUTEMBACHER SYNDROME = ASD + mitral stenosis *Hemodynamics:* no hemodynamic perturbance in the fetus; after birth physiologic increase in LA pressure creates a L-to-R shunt (shunt volume may be 3-4 times that of systemic blood flow) with volume overload of RV leading to RV dilatation, right heart failure, pulmonary hypertension; diastolic pressure differences in atria determine direction of shunt; pulmonary pressure remains normal for decades before Eisenmenger syndrome sets in; pulmonary hypertension in young adulthood (6%) ● repeated respiratory infections ● feeding difficulties ● arrhythmias ● thromboembolism ● asymptomatic; occasionally discovered by routine CXR ● right ventricular heave ● fixed splitting of second heart sound with accentuation of pulmonary component ● ECG: right axis deviation + some degree of right bundle branch block ● exertional dyspnea after development of pulmonary arterial hypertension (= Eisenmenger syndrome) ● cyanosis may occur (shunt reversal to R-to-L shunt), typically

during 3rd-4th decade ● right heart failure in patients >40 years CXR: √ normal (if shunt <2 x systemic blood flow)√ "hilar dance" = increased pulsations of central pulmonary arteries (DDx: other L-to-R shunts)√ overcirculation (if pulmonary-to-systemic blood flow ≥2:1)√ loss of visualization of SVC (= clockwise rotation of heart due to RV hypertrophy)√ small appearing aorta with normal aortic knob√ normal size of LA after shunt reversal (due to immediate decompression into RA) in EISENMENGER SYNDROME√ enlargement of pulmonary trunk + arteries√ RV enlargementECHO: √ paradoxical interventricular septal motion (due to volume overload of RV)√ direct visualization of ASD (= lack of echoes of atrial septum) in subcostal view√ diastolic blood flow from interatrial septum crossing RA + tricuspid valve observed by color DopplerAngio: √ RA fills with contrast shortly after LA is opacified (on levophase of pulmonary angio in AP or LAO projection)√ injection into RUL pulmonary vein to visualize exact size + location of ASD (LAO 45° + C-C 45°)*Prognosis:* (1) Mortality: 0.6% in 1st decade; 0.7% in 2nd decade; 2.7% in 3rd decade; 4.5% in 4th decade; 5.4% in 5th decade; 7.5% in 6th decade; median age of death is 37 years(2)Spontaneous closure: 22% in infants <1 year; 33% between ages 1 and 2 years; 3% in children >4 years*Cx:*(1)Tricuspid insufficiency (secondary to dilatation of AV ring)(2) Mitral valve prolapse(3) Atrial fibrillation (in 20% 1st presenting symptom in patients > age 40)*Rx:*(if vascular changes still reversible = resistance of pulmonary-to-systemic system ≤0.7); 1% surgical mortality1.Surgical patch closure2.Rashkind foam + stainless steel prosthesis BENEFICIAL ASD =secundum type ASD serves an essential compensatory function in:1.Tricuspid atresiaRA blood reaches pulmonary vessels via ASD + PDA; improvement through Rashkind procedure 2.TAPVRsignificant shunt volume only available through ASD (VSD / PDA much less reliable) 3.Hypoplastic left heartsystemic circulation maintained via RV with oxygenated blood from LA through ASD into RA

Notes:

AZYGOS CONTINUATION OF IVC
=INTERRUPTED IVC WITH AZYGOS / HEMIAZYGOS CONTINUATION*Incidence:*0.2-0.6-2% of CHD*Etiology:*failure of right subcardinal vein to anastomose with hepatic vein resulting in drainage of suprarenal IVC to heart via cranial portion of supracardinal vein (ie, azygos vein)*May be associated with:* polysplenia syndrome (more common), asplenia syndrome (rare), indeterminate situs (= situs ambiguus), persistent left SVC, dextrocardia, transposed abdominal viscera, duplicated IVC, retroaortic left renal vein, congenital pulmonary venolobar syndrome √ enlargement of azygos arch to >7 mm√ widening of right paraspinal stripe contiguous with azygos arch (= enlarged paraspinal + retrocrural azygos veins)√ widening of left paraspinal stripe (= enlarged hemiazygos vein)√ absence of hepatic ± infrahepatic IVC √ drainage of hepatic veins directly into right atrium via suprahepatic segment of IVC (N.B.: IVC shadow present on LAT CXR!)√ drainage of iliac + renal veins via azygos / hemiazygos vein

Notes:

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BACTERIAL ENDOCARDITIS

Predisposed: 1.Rheumatic valve disease2.Mitral valve prolapse with mitral regurgitation3.Aortic stenosis, mitral stenosis, aortic regurgitation, mitral regurgitation4.Most CHD (VSD, TOF) except ostium secundum ASD5.Previous endocarditis6.Drug addicts:endocarditis of tricuspid valve causes multiple septic pulmonary emboli

7.Bicuspid aortic valve:responsible for 50% of aortic valvular bacterial endocarditis 8.Prosthetic valve:4% incidence of bacterial endocarditis √ exaggerated valve motion (= disintegration of suture line + regurgitation)

Valve Vegetations

Notes:

Valve Vegetations ECHO: √ usually discrete focal echodensities with sharp edges; may show fuzzy / shaggy nonuniform thickening of cusps (vegetations) in systole + diastole√ may appear as shaggy echoes that prolapse when the valve is closed (DDx to mitral valve prolapse)

Aortic Valve Endocarditis

Notes:

BUERGER DISEASE
=THROMBANGITIS OBLITERANS=idiopathic recurrent segmental obliterative vasculitis of small + medium-sized peripheral arteries + veins (panangiitis)*Incidence:*<1% of all chronic vascular diseases; more common in Israel, Orient, India*Etiology:*unknown*Histo:* (a)acute stage: multiple microabscesses within fresh / organizing thrombus; all layers of vessel wall inflamed but intact; internal elastic lamina may be damaged; multinucleated giant cells within microabscesses (PATHOGNOMONIC)(b)subacute stage: thrombus organization with little residual inflammation(c)chronic stage: lumen filled with organized recanalized thrombus, fibrosis of adventitia binds together artery, vein, and nerve*Associated with:*cigarette smoking (95%) ● instep claudication ± distal ulceration (symptoms abate on cessation of smoking + return on its resumption) ● Raynaud phenomenon (33%) Location:legs (80%), arms (10-20%)Site:starts in palmar + plantar vessels with proximal progression√ superficial + deep migratory thrombophlebitis (20-33%)√ arterial occlusions, tapered narrowing of arteries√ abundant corkscrew-shaped collaterals√ direct collateral following the path of the original artery (Martorell sign) in 80%√ skip lesions = multiple segments involved with portions of arterial wall remaining unaffected√ absence of generalized arteriosclerosis / arterial calcifications (90%)

Notes:

CARDIAC TAMPONADE
=significant compression of heart by fluid contained within pericardial sac resulting in impaired diastolic filling of ventricles*Cause:see* Pericardial effusion (page 489) ● tachycardia ● pulsus paradoxus = exaggeration of normal pattern= drop in systolic arterial pressure >10 mm Hg during inspiration (secondary to increase in right heart filling during inspiration at the expense of left heart filling) ● elevated central venous pressure with distended neck veins ● falling blood pressure ● distant heart sounds / friction rub ● ECG: reduced voltage, ST elevation, PR depression, nonspecific T-wave abnormalities√ normal lung fields + normal pulmonary vascularity√ rapid enlargement of heart size√ distension of SVC, IVC, hepatic + renal veins√ periportal edema√ hepatomegalyDoppler-US: √ episodes of high-velocity hepatopetal flow separated by long intervals of minimal flowECHO: √ diastolic collapse of RV√ cyclical collapse of either atrium*Rx:*pericardiocentesis / pericardial drainage

Notes:

Congestive Cardiomyopathy =DILATED CARDIOMYOPATHY*Etiology:* (a)Myocarditis: viruses, bacteria(b)Endocardial fibroelastosis = thickened endocardium + reduced contractility(c)Infants of diabetic mothers(d)Inborn error of metabolism: glycogenosis, mucolipidosis, mucopolysaccharidosis(e)Coronary artery disease: myocardial infarction, anomalous origin of left coronary artery, coronary calcinosis(f)Muscular dystrophies ● tendency for CHF√ cardiomegaly + poor contractility of ventricular wall√ global heart enlargement√ LA enlargement without enlargement of LA appendageECHO: √ enlarged LV with global hypokinesis√ IVS and LVPW of equal thickness with decreased amplitude of motion√ low-profile / "miniaturized" mitral valve√ mildly enlarged LA (elevated end-diastolic LV pressure)√ enlarged hypokinetic right ventricle

Notes:

Hypertrophic Cardiomyopathy =OBSTRUCTIVE CARDIOMYOPATHY=characterized by nondilated hypertrophy of left ventricle in the absence of cardiac / systemic disease that would cause LV hypertrophy1.SYMMETRIC / CONCENTRIC HYPERTROPHY (2-20%)(a) midventricular (b) diffuse (c) apical 2.ASYMMETRIC SEPTAL HYPERTROPHY (ASH)=IDIOPATHIC HYPERTROPHIC SUBAORTIC STENOSIS (IHSS) = SUBAORTIC STENOSIS= HYPERTROPHIC OBSTRUCTIVE CARDIOMYOPATHY =basal septum of LV disproportionately thickened3.APICAL HYPERTROPHY (2-3%)=myocardial wall thickening confined to apical portion of LV ● usually clinically benign ● giant inverted T waveLeft ventriculography: √ spade-shaped deformity of LV cavity*Pathophysiology:* -LV hypertrophy leads to subaortic stenosis, abnormal diastolic function, myocardial ischemia-rapid blood flow through narrow outflow tract causes the anterior leaflet of mitral valve to displace anteriorly toward septum during systole (Venturi effect)-mitral regurgitation (from displaced MV leaflet)*Etiology:*autosomal dominant transmission ● exertional angina + dyspnea, fatigue ● syncope, arrhythmia, sudden death√ prominent left midheart border (septal hypertrophy)

Systolic Anterior Motion (SAM) of MV in IHSS mitral valve leaflets move abruptly toward septum at a rate greater than the endocardium of the posterior wall; responsible for obstruction to blood ejected from LV ECHO: √ IVS >14 mm thick; posterolateral wall >11 mm thick; IVS:LVPW thickness >1.3:1 √ systolic anterior motion of mitral valve (SAM) causing narrowed LVOT in systole√ midsystolic closure of aortic valve√ increased LVOT gradient with late systolic peaking on Doppler

Notes:

Restrictive Cardiomyopathy *Etiology:*(a)infiltrative disease: amyloid, glycogen, hemochromatosis(b)constrictive pericarditis

Notes:

CHRONIC VENOUS STASIS DISEASE
=CHRONIC VENOUS INSUFFICIENCY=insufficiency / incompetence of venous valves in deep venous system of lower extremity*Cause:* (a)postphlebitic valvular incompetence: destruction of valve apparatus results in short thickened valves secondary to scar formation(b)primary valvular incompetence: shallow elongated redundant valve cusps prevent effective closure*Associated with:* incompetent venous valves in the calf (secondary to pressure dilatation from stasis in deep venous system) leading to superficial vein varicosities ● edema, induration (= fluid exudation from increased capillary pressure) ● ulceration (from minor trauma + decreased diffusion of oxygen secondary to fibrin deposits around capillaries) ● skin hyperpigmentation (= breakdown products of exudated RBCs) ● aching pain√ venous reflux on descending venography with Valsalva(a)82% in deep venous system alone(b)2% in saphenous vein alone(c)16% in bothbilateral in 75% *Grade:* 1=minimal incompetence= to level of upper thigh2=mild incompetence= to level of lower thigh3=moderate incompetence= to level of knee4=severe incompetence= to level of calf veins

Notes:

COARCTATION OF AORTA
M:F = 4:1; rare in Blacks

A.LOCALIZED COARCTATION [former classification=ADULT / POSTDUCTAL / JUXTADUCTAL TYPE] (most common type)=short discrete narrowing close to ligamentum arteriosum

◊Coexistent cardiac anomalies uncommon!Location:most frequent in juxtaductal portion of arch ● incidental finding late in life ● ductus usually closed√ shelflike lesion at any point along the aortic arch√ narrow isthmus above the lesion√ poststenotic aortic dilatation distally B.TUBULAR HYPOPLASIA

[former classification=INFANTILE / PREDUCTAL / DIFFUSE TYPE]=hypoplasia of long segment of aortic arch after origin of innominate artery
◊Coexistent cardiac anomalies common! ● CHF in neonatal period (in 50%)*Hemodynamics:* fetus:no significant change because only 10% of cardiac output flows through aortic isthmusneonate:determined by how rapidly the ductus closes; without concurrent VSD overload of LV leads to CHF in 2nd / 3rd week of life*Collateral circulation:* via subclavian artery and its branches: - intercostals- internal mammary- anterior spinal artery- scapular artery- lateral thoracic- transverse cervical artery*Associated with:* (in 50%): 1.Bicuspid aortic valve (in 25-50%), which may result in calcific aortic valve stenosis (after 25 years of age) + bacterial endocarditis2.Intracardiac malformations:PDA (33%), VSD (15%), aortic stenosis, aortic insufficiency, ASD, TGV, ostium primum defect, truncus arteriosus, double-outlet right ventricle3.Noncardiac malformations (13%):Turner syndrome (13-15%)4.Cerebral berry aneurysms5.Mycotic aneurysm distal to CoA *Prognosis:*11% mortality prior to 6 months of age *Rx:*ages 3-5 years are ideal time for operation (late enough to avoid restenosis + early enough before irreversible hypertension occurs); surgical correction past 1 year of age decreases operative mortality drastically; 3-11% perioperative mortality *Procedures:* 1.Resection + end-to-end anastomosis2.Patch angioplasty3.Subclavian flap (Waldhausen procedure) using left subclavian artery as a flap *Postsurgical Cx:* 1.Residual coarctation (in 32%)2.Subsequent obstruction (rare)3.Mesenteric arteritis: 2-3 days after surgery secondary to paradoxical hypertension from increased plasma renin ● abdominal pain, loss of bowel control4.Chronic persistent hypertension

Symptomatic CoA Asymptomatic CoA

Notes:

Symptomatic CoA ◊Second most common cause of CHF in neonate (after hypoplastic left heart)Time:(a)toward the end of 1st week of life in "critical stenosis"(b)more commonly presents in older child ● lower extremity cyanosis (in tubular hypoplasia) ● left ventricular failure (usually toward end of 1st week of life)√ generalized cardiomegaly√ increased pulmonary vascularity (L-to-R shunt through PDA / VSD)√ pulmonary venous hypertension / edema√ "figure 3 sign" hidden by thymus

Notes:

Asymptomatic CoA ● headaches (from hypertension) ● claudication (from hypoperfusion)√ "figure 3 sign" = indentation of left lateral margin of aortic arch in the region of aortic-pulmonic window (at site of coarctation and poststenotic dilatation)√ "reverse 3 sign" on barium esophagram√ elevated left ventricular apex (secondary to left ventricular hypertrophy)√ scalloped contouring of soft-tissues posterior to sternum (= dilated tortuous internal mammary arteries) on LAT CXR (in 28%)√ dilatation of brachiocephalic vessels + aorta proximal to stenosis√ obscuration of superior margin of aortic arch√ rib notching (in 75%; mostly in adults over age 20; unusual before age 6)Location:ribs 3-8 (most pronounced in 3rd + 4th ribs, less pronounced in lower ribs)Site:central + lateral thirds of posterior rib(a)bilateral(b)unilateral on left side: left aortic arch with aberrant right subclavian artery below CoA(c)unilateral on right side: right aortic arch with anomalous left subclavian artery below CoA

Notes:

CONGENITAL ABSENCE OF PULMONARY VALVE

Massive regurgitation between pulmonary artery and RV *Associated with in 90%:* VSD, tetralogy of Fallot (50%) ● cyanosis (not in immediate newborn period) ● repeated episodes of respiratory distress ● continuous murmur ● ECG:right ventricular hypertrophy√ prominent main, right, and left pulmonary artery√ RV dilatation (increased stroke volume)√ partial obstruction of right / left mainstem bronchus (compression by vessel)√ right-sided aorta (33%)

Notes:

CONGESTIVE HEART FAILURE
=elevation of microvascular pressure of lung; most common cause of interstitial + airspace edema of lungs*Cause:* (a)back pressure from LV: long-standing systemic hypertension, aortic valve disease, coronary artery disease, cardiomyopathy, myocardial infarction(b)obstruction proximal to LV: mitral valve disease, LA myxoma, cor triatriatum*Histo:* (a)Interstitial phase: fluid in loose connective tissue around conducting airways and vessels + engorgement of lymphatics(b)Alveolar phase: increase in alveolar wall thickness(c)Alveolar airspace phase: alveoli filled with fluid + loss of alveolar volume; pulmonary fibrosis upon organization of intra-alveolar fibrin (if chronic)√ large heart√ vascular congestion 1.**Interstitial pulmonary edema** (invariably precedes alveolar edema) ● NO abnormal physical finding ● hypoxemia (ventilation-perfusion inequality)√ loss of sharp definition of vascular markings√ thickening of interlobular septa (pulmonary venous wedge pressure 17-20 mm Hg)√ poorly defined increased bronchial wall thickness√ thickening of interlobar fissures (due to fluid in subpleural connective tissue layer)2.**Airspace edema** (when volume of capillary filtration exceeds that of lymphatic drainage) ● severe dyspnea / orthopnea ● tachypnea + cyanosis ● dry cough / copious frothy sputum ● hypoxemia (vascular shunting)√ poorly defined patchy acinar opacities√ coalescence of acinar consolidation, particularly in medial third of lung√ butterfly / bat-wing distribution of consolidation(= consolidated hilum + uninvolved lung cortex)

Notes:

CONSTRICTIVE PERICARDITIS
=fibrous thickening of pericardium interfering with filling of ventricular chambers through restriction of heart motion*Age:*30-50 years; M:F = 3:1*Etiology:* 1.Idiopathic (most common)2.Viral (Coxsackie B)3.Tuberculosis (formerly most common)4.Chronic renal failure5.Rheumatoid arthritis6.Neoplastic involvement7.Radiotherapy to mediastinum Causes of acute pericarditis: *mnemonic:*"MUSIC"**M**yocardial infarction (acute) **U**remia **S**urgery (cardiac) **I**nfection **C**ancer ● dyspnea ● abdominal enlargement (ascites + hepatomegaly) ● peripheral edema ● pericardial knock sound = loud early-diastolic sound ● neck vein distension ● Kussmaul sign = failure of venous pressure to fall with inspiration ● prominent X and Y descent on venous pressure curve√ linear / plaquelike pericardial calcifications (50%): predominantly over RV, posterior surface of LV, in atrioventricular groove√ dilatation of SVC, azygos vein√ small atria√ normal / small-sized heart (enlargement only due to preexisting disease)√ normal pulmonary vascularity / pulmonary venous hypertension√ straightening of right + left heart borders√ increase in ejection fraction (small EDV)CT: √ epicardium = visceral pericardium >2 mm thick√ dilatation of SVC + IVC√ reflux of contrast into coronary sinus√ flattening of right ventricle + curvature of interventricular septum toward left√ pleural effusion + ascitesECHO (nonspecific features): √ thickening of pericardium√ rapid early filling motion followed by flat posterior wall motion during diastasis period (= period between early rapid filling and atrial contraction)*Cx:*protein-losing enteropathy (increased pressure in IVC + portal vein)*DDx:*Cardiac tamponade, restrictive cardiomyopathy (eg, amyloid)

Notes:

CORONARY ARTERY FISTULA

=single / multiple fistulous connections between a coronary artery (R > L) and other heart structures*Abnormal communication with* (>90% right heart): RV > RA > pulmonary trunk > coronary sinus > SVC *Hemodynamics:*L-to-R shunt; pulmonary:systemic blood flow = <1.5:1 (usually)√ may have normal CXR (in small shunts)√ cardiomegaly + shunt vascularity (in large shunts)Angio: √ dilated tortuous coronary artery with anomalous connection

Notes:

COR TRIATRIATUM
=rare congenital anomaly in which a fibromuscular septum with a single stenotic / fenestrated / large opening separates the embryologic common pulmonary vein from the left atrium:(1)proximal / accessory chamber lies posteriorly receiving pulmonary veins(2)distal / true atrial chamber lies anteriorly connected to left atrial appendage + emptying into LV through mitral valve*Etiology:*failure of common pulmonary vein to incorporate normally into left atrium*Associated with:*ASD, PDA, anomalous pulmonary venous drainage, left SVC, VSD, tetralogy of Fallot, atrioventricular canal ● dyspnea, heart failure, failure to thrive ● clinically similar to mitral valve stenosis √ pulmonary venous distention + interstitial edema + dilatation of pulmonary trunk and pulmonary arteries (in severe obstruction)√ enlarged RA + RV√ mild enlargement of LAAngio: √ dividing membrane on levophase of pulmonary arteriogram*Prognosis* (if untreated): usually fatal within first 2 years of life; 50% 2-year survival; 20% 20-year survival *Rx:*surgical excision of obstructing membrane

Notes:

DEEP VEIN THROMBOSIS

= DVT *Incidence:*140,000-250,000 new cases per year in United States with an estimated sole / major cause of 50,000-200,000 deaths per year (15% of in-hospital deaths); 6-7 million stasis skin changes; in 0.5% cause of skin ulcers*Pathogenetic factors:* 1.Hypercoagulability2.Decreased blood flow / stasis3.Intimal injury4.Decreased fibrinolytic potential of veins5.Platelet aggregation*Risk factors:* 1.Surgery, esp. on legs / pelvis: orthopedic (45-50%) especially total hip replacement >50%), gynecologic (7-35%), neurosurgery (18-20%), urologic (15-35%), general surgery (20-25%)2. Severe trauma3.Prolonged immobilization: hemiplegic extremity, paraplegia + quadriplegia, casting / orthopedic appliances4.Malignancy (risk factor 2.5) = Trousseau syndrome5.Obesity (risk factor 1.5)6.Diabetes7.Pregnancy (risk factor 5.5) and for 8-12 weeks postpartum8.Medication: birth control pills, estrogen replacement, tamoxifen (risk factor 3.2)9.Decreased cardiac function: congestive heart failure, myocardial infarction (20-50%; risk factor 3.5)10.Age >40 years (risk factor 2.2) 11.Varicose veins12.Previous DVT (risk factor 2.5)13.Patients with blood group A > blood group 014.Polycythemia15.Smoking Location: 1.Dorsal veins of calf (± ascending thrombosis)2.Iliofemoral veins (± descending thrombosis)3.Peripheral + iliofemoral veins simultaneously4.rare: internal iliac v., ovarian v., ascending lumbar vv.L:R = 7:3 due to compression of left common iliac v. by left common iliac a. (arterial pulsations lead to chronic endothelial injury with formation of intraluminal spur, which is present in 22% of autopsies + in 90% of patients with DVT) ● Local symptoms due to obstruction / phlebitis usually only when (a) thrombus occlusive (b) clot extends into popliteal / more proximal vein (14-78% sensitivity, 4-21% specificity) ● warmth ● swelling (measurement of circumference) ● blanching of skin (phlegmasia dolens alba) / blue leg with complete obstruction (phlegmasia cerulea dolens) ● deep crampy pain in affected extremity, worse in erect position, improved while walking ● tenderness along course of affected vein ● Homans sign = calf pain with dorsal flexion of foot ● Payr sign = pain upon compression of sole of foot◊2/3 of deep vein thromboses are clinically silent◊Clinically suspected DVT only in 50% confirmed◊DVT symptomatology due to other causes in 15-35% of patients ◊Negative bilateral venograms in 30% of patients with angiographically detected pulmonary emboli (big bang theory = clot embolizes in toto to the lung leaving no residual in vein) Venography (89% sensitivity, 97% specificity): false negative in 11%, false positive in 5%;study aborted / nondiagnostic in 5%*Risk:*postvenography phlebitis (1-2%), contrast reaction, contrast material-induced skin slough, nephropathy√ intraluminal filling defect constant on all images√ nonfilling of calf veins√ inadequate filling of common femoral vein + external + common iliac veinsB-Mode US (88-100% sensitivity, 92-100% specificity, >90% accuracy for DVT in thigh and popliteal veins): √ lack of complete luminal collapse with venous compression (DDx: deformity + scarring from prior DVT; technical difficulties in adductor canal + distal deep femoral vein)√ visualization of clot within vein (DDx: slow flowing blood; machine noise)√ <75% increase in diameter of common femoral vein during Valsalva√ venous diameter at least twice that of adjacent artery suggests thrombus <10 days old Doppler US: √ absence of spontaneity (= any waveform recording), not reliable in peripheral veins√ continuous venous signal = absence of phasicity(= no cyclic variation in flow velocity with respiration, ie, decrease in expiration + increase in inspiration) is suspicious for proximal obstruction √ attenuation / absence of augmentation (= no increase in flow velocity with distal compression) indicates venous occlusion / compression in intervening venous segments√ pulsatile venous flow is a sign of congestive heart failure / pericardial effusion / cardiac tamponade / pulmonary embolism with pulmonary hypertension Venous Occlusion Plethysmography : -87-95-100% sensitivity, 92-100% specificity for above-knee DVT-17-33% sensitivity for below-knee DVT=temporary obstruction of venous outflow by pneumatic cuff around mid-thigh inflated above venous pressure leads to progressive increase in blood volume in lower leg; upon release of cuff limb quickly returns to resting volume with prompt venous runoff; limb blood volume changes are measured by *impedance plethysmography* in which a weak alternating current is passed through the leg; the electrical resistance varies inversely with blood volume; the current strength is held constant and voltage changes directly reflect blood volume changes√ initial rise in venous volume (= venous capacitance) diminished√ delay in venous outflow = "fall" measured at 3 seconds*False positives (6%):*severe cardiopulmonary disease, pelvic mass, reduced arterial inflow*False negatives:*calf vein thrombosis, small thrombus I-125-Labeled Fibrinogen: -90% sensitive for calf vein thrombus-60-80% sensitive for femoral vein thrombus-insensitive for thrombus in upper thigh / pelvis*Risk:*results not available for several days, transmission of viral infection*False positives:*hematoma, inflammation, wound, old small thrombus isolated in common femoral / iliac vein *Cx:* (1)Pulmonary embolism (50%): in 90% from lower extremity / pelvis; in 60% with proximal "free-floating" / "widow-maker" thrombus; occurs usually between 2nd to 4th (7th) day of thrombosis*Source of pulmonary emboli:* multiple sites (1/3), cryptogenic in 50%; (a)lower extremity(46%)(b)inferior vena cava(19%)(c)pelvic veins(16%)(d)mural heart thrombus(4.5%)(e)upper extremity(2%)*Likelihood of pulmonary embolism:* 77% for iliac veins, 35-67% for femoropopliteal vein, 0-46% for calf veins (2)Postphlebitic syndrome (PPS) in 20% of cases with DVT (= recanalization to a smaller lumen, focal wall changes) due to valvular incompetence(3)Phlegmasia cerulea / alba dolens (= severely impaired venous drainage resulting in gangrene) *Prognosis:*tibial / peroneal venous thrombi resolve spontaneously in 40%, stabilize in 40%, propagate into popliteal vein in 20%*Prophylaxis:*intermittent compression of legs, heparin, warfarin*Rx:* (1)Heparin IV(2)Systemic anticoagulation (warfarin) for ≥3 months decreases risk of recurrent DVT in initial 3 months from 50% to 3% + fatal pulmonary embolism from 30% to 8%; necessity for anticoagulation in DVT of calf veins is controversial(3)Caval filter (10-15%) in patients with contraindication / complication from anticoagulation or progression of DVT / PE despite adequate anticoagulation*DDx:*pseudothrombophlebitis (= signs + symptoms of DVT produced by popliteal cyst / traumatic hematoma)

Notes:

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DOUBLE-OUTLET RIGHT VENTRICLE
= DORV = TAUSSIG-BING HEART = most of the aorta + pulmonary artery arise from the RV secondary to maldevelopment of conotruncus Type 1=aorta posterior to pulmonary artery + spiraling course (most frequent)Type 2=Taussig-Bing heart = aorta posterior to pulmonary artery + parallel courseType 3=aorta anterior to pulmonary artery + parallel course*Hemodynamics:* fetus:no CHF in utero (in absence of obstructing other anomalies)neonate:ventricular work overload leads to CHF*Associated with:*VSD (100%), pulmonary stenosis (50%), PDA↓ aorta overriding the interventricular septum with predominant connection to RV↓ aorta posterior / parallel / anterior to pulmonary artery↓ LV enlargement (volume overload)

Notes:

DUCTUS ARTERIOSUS ANEURYSM
=fusiform aneurysm of ductus arteriosus, usually patent toward aorta + completely / incompletely occluded toward pulmonary artery*Incidence:*<100 cases*Classification:* (a)according to age: infantile, childhood, adult type(b)according to cause: congenital, infectious, traumatic*Pathogenesis:*? delay in closure, ? myxoid degeneration of ductus wall, ? abnormal elastic fibers*Age:*most <2 months of age ● dyspnea, tachypnea, hoarseness√ pulmonary artery displaced anteromedially√ distal aortic arch displaced laterallyCXR: √ left-sided upper mediastinal mass in aorticopulmonary window√ tracheal displacement to right + anteriorly / posteriorly√ consolidation of adjacent lung (compression, fibrosis, hemorrhage)CT:√ contrast-enhancing mass in classic locationECHO:√ cystic mass with pulsatile flow*Cx:*rupture, dissection, infection, thromboembolic disease, phrenic nerve compression*Prognosis:*usually fatal (without prompt surgery)

Notes:

EBSTEIN ANOMALY
=downward displacement of septal + posterior leaflets of dysplastic tricuspid valve with ventricular division into(a) a large superior atrialized portion and (b) a small inferior functional chamber *Etiology:*chronic maternal lithium intake (10%)*Hemodynamics:*tricuspid valve insufficiency leads to tricuspid regurgitation ("Ping-Pong" volume); may be followed by CHF in utero / in neonate (50%); survival into adulthood if valve functions normally*Associated with:*PDA, ASD (R-to-L shunt) ● cyanosis in neonatal period (R-to-L shunt), may improve / disappear postnatally with decrease in pulmonary arterial pressure ● systolic murmur (tricuspid insufficiency) ● Wolff-Parkinson-White syndrome (10%) = paroxysmal supraventricular tachycardia / right bundle branch block (responsible for sudden death)√ "boxlike / funnel-like" cardiomegaly (enlargement of RA + RV)√ extreme RA enlargement (secondary to insufficient tricuspid valve)√ IVC + azygos dilatation (secondary to tricuspid regurgitation)√ hypoplastic aorta + pulmonary trunk (the ONLY cyanotic CHD to have this feature)√ normal LA√ calcification of tricuspid valve may occurECHO: √ large "sail-like" tricuspid valve structure within dilated right heart√ tricuspid regurgitation identified by Doppler ultrasound*Prognosis:*50% infant mortality; 13% operative mortality*Rx:*1.Digitalis + diuretics2.Tricuspid valve prosthesis

Notes:

EISENMENGER COMPLEX
=EISENMENGER DEFECT=(1) high VSD ± overriding aorta with hypoplastic crista supraventricularis(2)RV hypertrophyand as consequence of increased pulmonary blood flow: (3) dilatation of pulmonary artery + branches(4)intimal thickening + sclerosis of small pulmonary arteries + arterioles ● cyanosis appears in 2nd + 3rd decade with shunt reversal

Notes:

EISENMENGER SYNDROME
=EISENMENGER REACTION=development of high pulmonary vascular resistance after many years of increased pulmonary blood flow secondary to L-to-R shunt (ASD, PDA, VSD), which leads to a bidirectional (= balanced) shunt and ultimately to R-to-L shunt*Etiology:* pulmonary microscopic vessels undergo reactive muscular hypertrophy, endothelial thickening, in situ thrombosis, tortuosity + obliteration; once initiated, pulmonary hypertension accelerates the vascular reaction, thus increasing pulmonary hypertension in a vicious cycle with RV failure + death √ pronounced dilatation of central pulmonary arteries (pulmonary trunk, main pulmonary artery, intermediate branches)√ pruning of peripheral pulmonary arteries√ enlargement of RV√ LA + LV return to normal size (with decrease of L-to-R shunt)√ pulmonary veins NOT distended (NO increase in pulmonary blood flow)√ NO redistribution of pulmonary veins (normal venous pressure)*Dx:*measurement of pulmonary artery pressure + flow via catheter

Notes:

ENDOCARDIAL CUSHION DEFECT
=ECD = ATRIOVENTRICULAR SEPTAL DEFECT=PERSISTENT OSTIUM ATRIOVENTRICULARE COMMUNE = PERSISTENT COMMONATRIOVENTRICULAR CANAL=persistence of primitive atrioventricular canal + anomalies of AV valves*Associated with:* (1)Down syndrome:in 25% of trisomy 21 an ECD is present;in 45% of ECD trisomy 21 is present(2)Asplenia, polysplenia A.INCOMPLETE / PARTIAL ECD=(1)Ostium primum ASD(2)Cleft in anterior mitral valve leaflet / trileaflet(3)Accessory short chordae tendineae arising from anterior MV leaflet insert directly into crest of deficient ventricular septum√ left atrioventricular valve usually has 3 leaflets with a wide cleft between anterior + septal leaflet√ "gooseneck" deformity secondary to downward attachment of anterior MV leaflet close to interventricular septum by accessory chordae tendineae√ communication between LA-RA or LV-RA, occasionally LV-RV√ right atrioventricular valve usually normalB.TRANSITIONAL / INTERMEDIATE ATRIOVENTRICULAR CANAL (uncommon)=(1)Ostium primum ASD(2)High membranous VSD(3)Wide clefts in septal leaflets of both AV valves(4)Bridging tissue between anterior + posterior common leaflet of both AV valvesC.COMPLETE ECD = AV COMMUNIS = COMMON AV CANAL=(1)Ostium primum ASD above(2)Posterior VSD below(3)One AV valve common to RV + LV with 5-6 leaflets(a)anterior common "bridging" leaflet(b)two lateral leaflets(c)posterior common "bridging" leaflet Type 1 =chordae tendineae of anterior bridging leaflet attached to both sides of ventricular septumType 2 =chordae tendineae of anterior leaflet attached medially to anomalous papillary muscle within RV, but unattached to septumType 3 =free-floating anterior leaflet with chordae attachments to septum; only type becoming symptomatic in infancy!√ common atrioventricular orifice√ oval septal defect consisting of a low ASD + high VSD√ atrial septum secundum usually spared ("common atrium" if absent)√ frequently associated with mesocardia / dextrocardia *Hemodynamics:* fetus:atrioventricular valves frequently incompetent leading to regurgitation + CHFneonate:L-to-R shunt after decrease of pulmonary vascular resistance resulting in pulmonary hypertension ● incomplete right bundle branch block (distortion of conduction tissue) ● left-anterior hemiblock CXR: √ increased pulmonary vascularity (= shunt vascularity)√ redistribution of pulmonary blood flow (mitral regurgitation)√ enlarged pulmonary artery√ diminutive aorta (secondary to L-to-R shunt)√ cardiac enlargement out of proportion to pulmonary vascularity (L-to-R shunt + mitral insufficiency)√ enlarged RV + LV√ enlarged RA (LV blood shunted to RA)√ normal-sized LA (secondary to ASD)ECHO: √ visualization of ASD + VSD + valve + site of insertion of chordae tendineae√ paradoxical anterior septal motion (secondary to ASD)√ atrioventricular insufficiency + shunts identified by Doppler ultrasoundAngio: AP projection: √ gooseneck deformity of LVOT (in diastole)√ cleft in anterior leaflet of mitral valve (in systole)√ mitral regurgitationHepatoclavicular projection in 45° LAO + C-C 45° (= 4-chamber view):√ best view to demonstrate LV-RA shunt√ best view to demonstrate VSD (inflow tract + posterior portion of interventricular septum in profile)LAT projection:√ irregular appearance of superior segment of anterior mitral valve leaflet over LVOT *Prognosis:*54% survival rate at 6 months, 35% at 12 months, 15% at 24 months, 4% at 5 years; 91% long-term survival with primary intracardiac repair, 4-17% operative mortality

Notes:

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ENDOCARDIAL FIBROELASTOSIS
=diffuse endocardial thickening of LV + LA from deposition of collagen + elastic tissue*Etiology:* (1)? viral infection(2)Secondary endocardial fibroelastosis=subendocardial ischemia in critical LVOT obstruction: aortic stenosis, coarctation, hypoplastic left heart syndrome ● sudden onset of CHF during first 6 months of life√ mitral insufficiency:(a) involvement of valve leaflets (b) shortening + thickening of chordae tendineae (c) distortion + fixation of papillary muscles √ enlarged LV = dilatation of hypertrophied LV from mitral regurgitation√ restricted LV motion√ enlarged LA√ pulmonary venous congestion + pulmonary edema√ LLL atelectasis (= compression of left lower lobe bronchus by enlarged LA)*Prognosis:*mortality almost 100% by 2 years of age

Notes:

FLAIL MITRAL VALVE

Cause: (1)ruptured chordae tendineae in rheumatic heart disease, ischemic heart disease, bacterial endocarditis(2)rupture of head of papillary muscle in acute myocardial infarction, chest traumaLocation:chordae to leaflet from posteromedial papillary muscle (single vessel blood supply)√ deep holosystolic posterior movement √ random anarchic motion pattern of flail parts in diastole√ excessively large amplitude of opening of aML

Notes:

HYPOPLASTIC LEFT HEART SYNDROME

=SHONE SYNDROME = AORTIC ATRESIA=underdevelopment of left side of heart characterized by (a) aortic valve atresia (b) hypoplastic ascending aorta (c) hypoplastic / atretic mitral valve (d) endocardial fibroelastosis giving rise to small LA + small LV + small ascending aorta*Incidence:*most common cause of CHF in neonate; responsible for 25% of all cardiac deaths in 1st week of life*Hemodynamics:* pulmonary venous return is diverted from LA to RA through herniated foramen ovale / ASD (L-to-R shunt); RV supplies (a) pulmonary artery (b) ductus arteriosus (c) descending aorta (antegrade flow) (d) aortic arch + ascending aorta + coronary circulation (retrograde flow) leading to RV work overload + CHF ● characteristically presents within first few hours of life ● ashen gray color (inadequate atrial L-to-R shunt with systemic underperfusion) ● myocardial ischemia (decreased perfusion of aorta + coronary arteries) ● cardiogenic shock, metabolic acidosis ● CHF (RV volume + pressure overload)OB-US: √ small left ventricular cavity (apex of LV and RV should be at same level)√ hypoplastic ascending aorta + aortic arch√ aortic coarctation (in 80%)ECHO: √ normal / enlarged LA√ small LV√ enlarged RA√ herniation + prolapse of foramen ovale flap into RA√ small / absent aortic root√ absent / grossly distorted mitral valve echoesAngio: √ retrograde flow in ascending aorta + aortic arch + coronary arteries via PDA√ stringlike ascending aorta <6 mm in diameter√ massive enlargement of RV + RVOT*Prognosis:*almost 100% fatal by 6 weeks*Rx:*(1) Norwood procedure = palliative attempt(2) Cardiac transplant

Notes:

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HYPOPLASTIC RIGHT VENTRICLE
=PULMONARY ATRESIA WITH INTACT VENTRICULAR SEPTUM=underdeveloped right ventricle due to pulmonary atresia in the presence of an intact interventricular septumType I=small RV secondary to competent tricuspid valve (more common)Type II=normal / large RV secondary to incompetent tricuspid valve*Hemodynamics:* fetus:L-to-R atrial shunt through foramen ovale; retrograde flow through ductus arteriosus into pulmonary vascular bedneonate:closure of ductus results in cyanosis, acidosis, death√ small right ventricular cavity (apex of RV + LV should be at same level)√ atresia of pulmonary valve√ hypoplastic proximal pulmonary artery√ secundum atrial septal defect (frequently associated)*Rx:*prostaglandin E1 infusion + valvotomy + systemic-pulmonary artery shunt

Notes:

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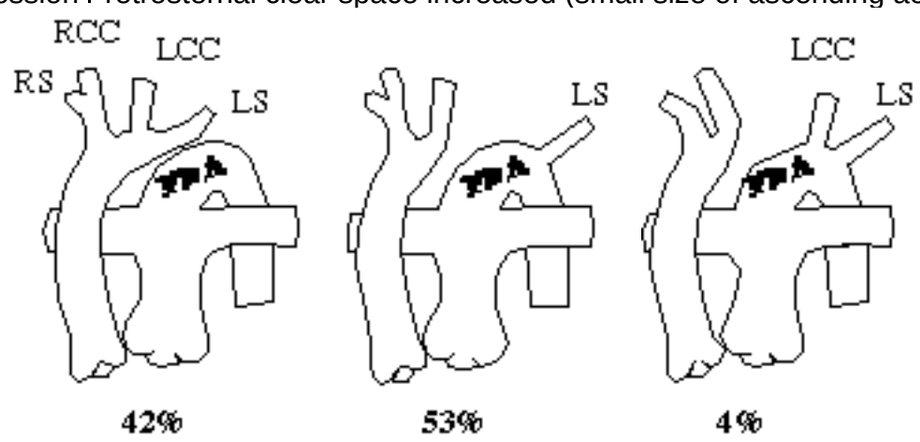
IDIOPATHIC DILATATION OF PULMONARY ARTERY

=CONGENITAL ANEURYSM OF PULMONARY ARTERY*Age:*adolescence; M < F ● systolic ejection murmur (in most cases)√ dilated main pulmonary artery√ normal peripheral pulmonary vascularity√ normal pulmonary arterial pulsations√ NO lateralization of pulmonary flow*Dx per exclusion*: 1.Absence of shunts, CHD, acquired disease2.Normal RV pressure3.No significant pressure gradient across pulmonic valve*DDx:*(1)Marfan syndrome(2)Takayasu arteritis

Notes:

INTERRUPTION OF AORTIC ARCH

=rare congenital anomaly as a common cause of death in the neonatal periodTrilogy:(1)Interrupted aortic arch(2)VSD(3)PDA (pulmonary blood supplies lower part of body)*Associated with* (in 1/3): 1.Bicuspid aortic valve2.Muscular subaortic stenosis3.ASD4.Truncus arteriosus5.Transposition6.Complete anomalous pulmonary venous return ● presents with CHFLocation: Type A:distal to left subclavian artery (42%)Type B:between left CCA and subclavian artery (53%) associated with: DiGeorge syndromeType C:between innominate and left CCA (4%) √ dilatation of right atrium + ventricle√ dilatation of pulmonary artery√ ascending aorta much smaller than pulmonary artery√ arch formed by pulmonary artery + ductus arteriosus gives the appearance of a low aortic arch√ aortic knob absent√ trachea in midline√ NO esophageal impression√ retrosternal clear space increased (small size of ascending aorta)√ increased pulmonary vascularity (L-to-R shunt)*Prognosis:*76% dead at end of 1st month



Interruption of Aortic Arch

Notes:

INTERRUPTION OF PULMONARY ARTERY
=pulmonary trunk continues only as one large artery to one lung while systemic aortic collaterals supply the other side*Associated with:* CHD (particularly if interruption on left side): 1.Tetralogy of Fallot2.Scimitar syndrome = congenital pulmonary venolobar syndrome3.PDA, VSD4.Pulmonary hypertension*Collateral supply:* 1.Arteries arising from arch + ascending aorta2.Bronchial vessels3.Intercostal vessels4.Branches from subclavian arteryLocation:usually opposite from aortic arch; R + L pulmonary artery equally involvedCXR: √ hypoplastic ipsilateral lung√ mediastinal shift toward involved lung√ hemidiaphragm may be elevated√ small hyperlucent ipsilateral chest with narrowed intercostal spaces√ "comma-shaped" small distorted hilar shadow√ asymmetry of pulmonary vascularity√ normal respiratory motion (normal aeration of hypoplastic lung)NUC:√ absent perfusion with normal aerationAngio:√ absent pulmonary artery *Rx:*surgical anastomosis between proximal + distal pulmonary artery (to prevent progressive pulmonary hypertension with dyspnea, cyanosis, hemoptysis, death)*DDx:*(1)Hemitruncus(2)Swyer-James syndrome (ipsilateral air trapping, reduced ventilation + perfusion)

Notes:

INTRAVENOUS DRUG ABUSE

Complications secondary to: (a)direct toxic effects of drugs or drug combinations (eg, heroin + cocaine / Talwin)(b)direct toxic effects of adulterants [eg, heroin is mixed ("cut") with quinine, baking soda, sawdust](c)septic preparation(d)injection technique(e)choice of injection site (eg, "groin hit" into femoral vein; "pocket shot" into jugular, subclavian, brachiocephalic vein)A.Cardiovascular complications1.Arterial pseudoaneurysmmay be followed by rupture with exsanguination / loss of limb 2.Arteriovenous fistula3.Arterial occlusion(a)at injection site due to intimal damage, thrombosis, spasm(b)distal to injection site due to embolization, spasm4.Venous thrombosis5.Intravenous migration of needle to heart / lungs6.Embolization of infectious agent / foreign body / air through inadvertent arterial injection ("hit the pink")7.Endocarditis (most commonly S. aureus)B.Soft-tissue complications1.Hematoma / abscess2.Foreign bodies3.Lymphadenopathy4.CellulitisC.Skeletal complications1.Osteomyelitis(a)direct contamination: eg, pubic bone ("groin hit") / clavicle ("pocket shot")(b)hematogenous: spine most commonly affected2.Septic arthritis: sacroiliac, sternoclavicular, symphysis pubis, hip, knee, wristD.Pleuropulmonary complications1.Pneumothorax ("pocket shot")2.Hemo- / pyothorax3.Septic pulmonary emboliE.Gastrointestinal complications1.Severe colonic ileus2.Colonic pseudoobstruction3.Necrotizing enterocolitis4.Liver abscessF.Genitourinary complications1.Focal / segmental glomerulosclerosis (heroin abuser)2.AmyloidosisG.CNS complications1.Spinal epidural abscess in 5-18% (from vertebral osteomyelitis)2.Cord compression (from collapsed vertebral body)3.Cerebral infarction (from subacute bacterial endocarditis, toxic effect of drug, spasm, intimal damage from "pocket shot")4.Intracranial hemorrhage (from trauma, hypertension, injection of anticholinergic drugs, vasculitis, rupture of mycotic aneurysm)5.Meningitis, cerebral abscess

Notes:

Radiology Review Manual
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ISCHEMIC HEART DISEASE
=CORONARY ARTERY DISEASE (CAD)*Incidence:*1.5 million/year; leading cause of death in industrial nations*Morbidity:*28.7 cases per 1,000 men per year*Mortality:*3.1 deaths per 1,000 men per year Noninvasive testing: 1.Noninvasive testing is of marginal benefit when disease prevalence is <0.2 / >0.72.Concordant thallium-201 and stress ECG are greater predictors of disease probability than either one used alone and/or when discordant3.Sequential thallium-201 and stress ECG are most useful to establish the diagnosis of CAD when pretest prevalence is intermediate + test results are concordant CXR: √ often normal√ coronary artery calcification√ pulmonary venous hypertension following acute infarction (40%)√ LV aneurysm ECHO: √ region of dilatation with disturbance of wall movement(1)Akinesis=no wall motion(2)Hypokinesis=reduced wall motion(3)Dyskinesis=paradoxical systolic expansion(4)Asynchrony=disturbed temporal sequence of contractionCoronary angiography: 1.2 million procedures per year

Notes:

KAWASAKI SYNDROME
= MUCOCUTANEOUS LYMPH NODE SYNDROME = acute febrile multisystem vasculitis of unknown cause involving large + medium-sized + small arteries with a predilection for the coronary arteries *Incidence:*average of 1.1:100,000 population per year*Histo:*panvasculitis*Age:*<5 years of age (in 85%); peak age of 1-2 years; M:F = 1.5:1*Associated with:*polyarthritis (30-50%), aseptic meningitis (25%), hepatitis (5-10%), pneumonitis (5-10%) ● fever >5 days ● mucosal reddening (injected fissured lips, injected pharynx, strawberry tongue) in 99% ● nonpurulent cervical lymphadenopathy (82%) ● maculopapular rash on extensor surfaces (99%) ● bilateral nonpurulent conjunctivitis (96%) ● erythema of palms + soles with desquamation (88%)@Cardiovascular system (1/3)1.Coronary artery abnormality (15-25%)√ coronary artery aneurysm: LCA (2/3), RCA (1/3); proximal segment in 70%; 48% regress, 37% diminish in size√ coronary artery stenosis (39%) due to thrombus formation in aneurysm + intimal thickening√ coronary artery occlusion (8%) in aneurysms >9 mm2.Myocarditis (25%)3.Pericarditis4.Valvulitis5.Atrioventricular conduction disturbance√ intestinal pseudoobstruction√ transient gallbladder hydrops*Prognosis:*0.4-3% mortality (from myocardial infarction / myocarditis with congestive heart failure / rupture of coronary artery aneurysm)*Rx:*aspirin (100 mg/kg per day) + gamma globulin*DDx:*infantile polyarteritis

Notes:

MICROSCOPIC POLYANGIITIS
=pauci-immune necrotizing small-vessel angiitis without granulomatous inflammation*Path:*necrotizing arteritis identical to polyarteritis nodosa but with vasculitis in arterioles, venules and capillaries ● ANCA (antineutrophil cytoplasmic autoantibodies) in >80% ● negative serologic tests for hepatitis B◊Most common cause of the pulmonary-renal syndrome!√ pulmonary infiltrates√ glomerulonephritis (90%)

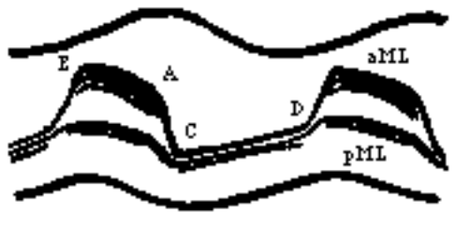
Notes:

MITRAL REGURGITATION

=MITRAL INSUFFICIENCY*Cause:* 1.Rheumatic heart disease(a)isolated: frequently seen in children(b)uncommon in adults (mostly combined with stenosis)2.Bacterial endocarditis3.Myocardial infarction with involvement of papillary muscle (posteromedial > anterolateral papillary m.)4.Congenital (short / abnormally inserted chordae tendineae)5.Marfan syndrome6.Corrected transposition with Ebstein-like anomaly7.Idiopathic hypertrophic subaortic stenosis (IHSS)8.Persistent ostium primum ASD with cleft mitral valve9.Mitral valve prolapse syndrome10.Functional / secondary (from dilatation of mitral ring in any condition with dilatation of LV)*Pathogenesis:*backward flow of blood from LV into LA during LV systole; increased volume of blood under elevated pressure causes dilatation of LA; marked increase in LV diastolic volume with little increase in LV diastolic pressure √ mild pulmonary venous hypertension (less than with mitral stenosis)√ LA + LV enlargement (cardiothoracic ratio >0.55)√ enlarged LA appendage (with history of previous rheumatic heart disease)√ mitral annular calcification (frequent) ECHO: √ LV volume overload√ normal-sized / enlarged LV√ increased septal + posterior wall motion√ increased EF slope√ early closure of aortic valve (LV stroke volume partially lost to LA)√ LA enlargement (in chronic MV insufficiency)√ bulging of interatrial septum to the right during systole√ Doppler is only diagnostic tool + allows assessment of severity

Notes:

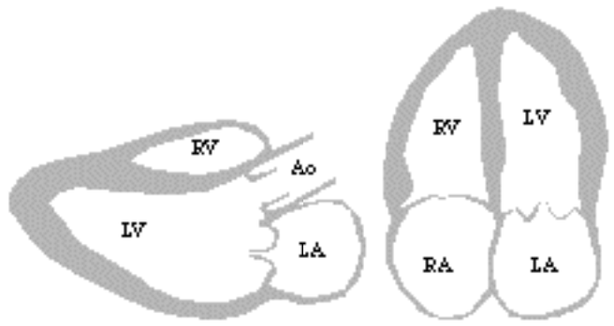
MITRAL STENOSIS
Acquired causes: principal cause:rheumatic heart diseaserare cause:mass obstructing LV inflow (tumor, myxoma, thrombus)M:F = 1:8 *Pathogenesis:* rise in left atrial + pulmonary vascular pressure throughout systole and into diastole; development of medial hypertrophy + intimal sclerosis in pulmonary arterioles leads to pulmonary arterial hypertension, RV hypertrophy, tricuspid regurgitation, RV dilatation, right heart failure ● history of rheumatic fever (in 50%) ● atrial fibrillation ● systemic embolization from thrombosis of atrial appendage *Stages* (according to degree of pulmonary venous hypertension): Stage 1:loss of hilar angle, redistributionStage 2:interstitial edemaStage 3:alveolar edemaStage 4:hemosiderin deposits + ossification √ calcification of valve leaflets (calcification of mitral annulus is a feature of age) √ prominent pulmonary artery segment (precapillary hypertension)√ small aorta (if forward cardiac output decreased)√ enlarged LA ± wall calcification√ "double density" seen through right upper cardiac border (AP view)√ bulge of superior posterior cardiac border below carina (lateral view)√ esophagus displaced toward right + posteriorly√ dilated left atrial appendage (not present with retracting clot)√ hypertrophy of RV√ dilatation of RV (tricuspid insufficiency / pulmonary hypertension)√ increase in cardiothoracic ratio√ diminution of retrosternal clear space√ IVC pushed backward (lateral view)√ redistribution of pulmonary blood flow to upper lobes (postcapillary pressure 16-19 mm Hg)√ interstitial pulmonary edema (postcapillary pressure 20-25 mm Hg)√ alveolar edema (postcapillary pressure 25-30 mm Hg)ECHO: √ thickening of leaflets toward free edge (fibrosis, calcification)√ flattening of EF slope = MV remains open throughout diastole due to persistently high LA pressure (crude index of severity of MV stenosis)√ diastolic anterior tracking of pML in 80% (secondary to diastolic anterior pull by larger + more mobile aML)√ diastolic doming of MV leaflets√ commissure fusion = increased echodensity + decreased leaflet motion at level of commissure√ area reduction of MV orifice: normal within 4-6 cm^2; mild narrowing with <2 cm^2; severe narrowing with <1 cm^2 (reproducible to within 0.3 cm^2)√ shortening + fibrosis of chordae tendineae√ abnormal septal motion = early diastolic dip of IVS due to rapid filling of RV (in severe MV stenosis)√ slowed LV filling pattern of small LV√ dilatation of LA (>5 cm increases risk of atrial fibrillation + left atrial thrombus)√ DE opening amplitude reduced to <20 mm indicating loss of valve pliability (DDx: low cardiac output state)√ absent A-wave common (atrial fibrillation)√ increase in valve gradient + pressure halftime on Doppler*Rx:*(1)Commissurotomy if valves pliable + calcium absent + MV regurgitation absent(2)Valve replacement for symptomatic patients with severely stenotic valves*DDx:* (1)Pseudomitral stenosis in decreased LV compliance (decreased EF slope, normal leaflet thickness + motion)(2)Rheumatic mitral insufficiency (indistinguishable findings + evidence of LV volume overload)(3)LA myxoma (mass behind MV + in LA)(4)Low cardiac output (apparent small valve orifice) LUTEMBACHER SYNDROME = rheumatic mitral valve stenosis + ASD

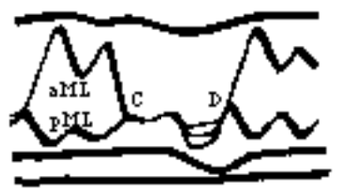


Classic Mitral Valve Stenosis

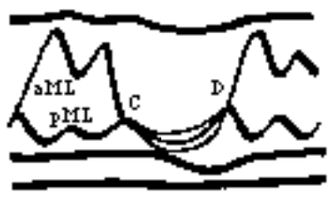
Notes:

MITRAL VALVE PROLAPSE
*Incidence:*2-6% of general population; 5-20% of young women; ? autosomal dominant inheritance*Age:*commonly 14-30 years*Cause:* (1)"Floppy mitral valve" = elongation of cusps + chordae leading to redundant valve tissue, which prolapses into LA during systole*Associated with:* (a)Skeletal abnormalities: scoliosis, straightening of thoracic spine, narrow anteroposterior chest dimension, pectus excavatum deformity of sternum(b) Barlowe syndrome = straight back syndrome(c)Marfan syndrome(d)Tricuspid valve prolapse(e)Long-standing ASD(2)Secondary MV prolapse:papillary muscle dysfunction, rupture chordae tendineae, rheumatic mitral insufficiency, primary pulmonary hypertension, ostium secundum ASD ● arrhythmias, palpitation, chest pain, light-headedness, syncope ● responsible for midsystolic click + late systolic murmur (when associated with mitral regurgitation)√ LA not enlarged (unless associated with significant mitral regurgitation)ECHO: √ interruption of CD line with bulge toward left atrium√ abrupt midsystolic posterior buckling of both leaflets (classic pattern)√ "hammocklike" pansystolic posterior bowing of both leaflets√ multiple scallops on mitral valve leaflets (short-axis parasternal view)√ valve leaflets may appear thickened (myxomatous degeneration + valve redundancy)√ mitral valve leaflets passing >2 mm posterior to plane of mitral annulus (apical 4-chamber view)√ hyperactive atrioventricular groove√ mitral annulus may be dilated >4.7 cm^2*DDx:*(1)Pericardial effusion (systolic posterior displacement of MV leaflets + entire heart)(2)Bacterial endocarditis (mimicked by locally thickened + redundant leaflets)





Mid systolic Mitral Valve Prolapse



Holosystolic Mitral Valve Prolapse

Notes:

MYOCARDIAL INFARCTION

*Incidence:*1,500,000 per year in United States resulting in 500,000 deaths (50% occur in asymptomatic individuals) ● atrioventricular block (common with inferior wall infarction as AV nodal branch originates from RCA); complete heart block has worse prognosis because it indicates a large area of infarctionCXR: √ normal-sized heart (84-95%) in acute phase if previously normal√ cardiomegaly: high incidence of congestive heart failure in anterior wall infarction, multiple myocardial infarctions, double- and triple-vessel CAD, LV aneurysmCECT: √ perfusion defect within 60-90 seconds after bolus injection√ delayed enhancement of infarcted tissue peaking at 10-15 minutes (due to accumulation of iodine in ischemic cells), size of enhanced area correlates well with size of infarct*Cx:*(myocardium is prone to rupture during 3rd-14th day post infarction) A.LEFT VENTRICULAR FAILURE (60-70%) ● "cardiac shock" = systolic pressure <90 mm Hg◊Signs of pulmonary venous hypertension are a good predictor of mortality (>30% if present, <10% if absent)√ progressive enlargement of heart√ haziness + indistinctness of pulmonary arteries√ increase in size of right descending pulmonary artery >17 mm√ pleural effusion√ septal lines√ perihilar ± peripheral parenchymal clouding√ alveolar pulmonary edema*Mortality:*30-50% with mild LV failure; 44% with pulmonary edema; 80-100% with cardiogenic shock; 8% in absence of LV failureB.ANEURYSM (12-15% of survivors)C.MYOCARDIAL RUPTURE (3.3%) ● occurs usually on 3rd-5th day post MI√ enlargement of heart (slow leakage of blood into pericardium)*Prognosis:*cause of death in 13% of all infarctions; almost 100% mortality D.RUPTURE OF PAPILLARY MUSCLE (1%)from infarction of posteromedial papillary muscle in inferior MI (common) / anterolateral papillary muscle in anterolateral MI (uncommon) ● sudden onset of massive mitral insufficiency ● unresponsive to medical management√ abrupt onset of severe persistent pulmonary edema√ minimal LV enlargement / normal-sized heart√ NO dilatation of LA (immediate decompression into pulmonary veins)*Prognosis:*70% mortality within 24 hours; 80-90% within 2 weeksE.RUPTURE OF INTERVENTRICULAR SEPTUM(0.5-2%) ● occurs usually within 4-21 days with rapid onset of L-to-R shunt ● Swan-Ganz catheterization: increase in oxygen content of RV, capillary wedge pressure may be within normal limits√ right-sided cardiac enlargement√ engorgement of pulmonary vasculature√ NO pulmonary edema (DDx to ruptured papillary muscle)*Prognosis:*24% mortality within 24 hours; 87% within 2 months; >90% in 1 yearF.DRESSLER SYNDROME (<4%)=POSTMYOCARDIAL INFARCTION SYNDROME*Etiology:*autoimmune reactionOnset:2-3 weeks (range 1 week-several months) following infarction ● relapses occur as late as 2 years after initial episode ● fever√ pericarditis + pericardial effusion√ pleuritis + pleural effusion√ pneumonitis

Right Ventricular Infarction

Notes:

Right Ventricular Infarction Right ventricle involved in 33% of left inferior myocardial infarction √ decreased RV ejection fraction √ accumulation of Tc-99m pyrophosphate*Prognosis:*in 50% RV ejection fraction returns to normal within 10 days*Cx:*(1)cardiogenic shock (unusual)(2)elevation of RA pressure(3)decrease of pulmonary artery pressure

Notes:

MYXOMA

Most common benign primary intracardiac tumor (true neoplasm) in adults, 40-50% of all cardiac tumors *Age:*30-60 years; M<F*Classification:*sporadic (most frequent);familial type (mean age of 24 years); complex type = Carney syndrome *Path:*(a)gelatinous, friable, papillary / villous pedunculated tumor(b)round / polypoid sessile tumor*Histo:*hypocellular amorphous acid mucopolysaccharide matrix covered by a monolayer of endothelial cells ● short history + rapid progression ● dyspnea, chest pain ● constitutional symptoms: ● fever, myalgia, arthralgia, weight loss ● leukocytosis, anemia, elevated ESR, ● hypergammaglobulinemia ● positional symptoms (ie, change with position): ● tachyarrhythmia, murmur ● syncopeLocation:LA:RA = 4:1; ventricles (exceptional); attached to atrial septum by small stalk near fossa ovalis (75%); may protrude into ventricle causing partial obstruction of atrioventricular valve√ generalized cardiac enlargement√ atrial obstruction (mimicking valvular stenosis)√ persistent defect in atrium / diastolic defect in ventricleA. LEFT ATRIAL MYXOMAwith obstruction of mitral valve: √ enlargement of LA√ pulmonary venous hypertension / edema√ ossific lung nodules√ NO enlargement of atrial appendage*Cx:*systemic emboli (27%) in 50% to CNS (stroke / "mycotic" aneurysm)B.RIGHT ATRIAL MYXOMAwith obstruction of tricuspid valve: √ enlargement of RA√ prominent SVC, IVC, azygos vein√ decreased pulmonary vascularity*Cx:* pulmonary emboli ECHO: (2D-ECHO is study of choice) √ hyperechoic mass ± mobile◊M-mode findings of only historical interest!√ dense echoes appearing posterior to aML soon after onset of diastole√ pML obscured√ tumor echoes can be traced into LA√ dilated LA√ reduced E-F slopeCT: √ intraluminal filling defectMR: √ hypointense on T1WI, hyperintense on T2WI*Rx:*surgical excision ± valvuloplasty / valve replacement*Prognosis:*5-14% recurrence rate*DDx:*(1)Thrombus (most commonly in LA + LV)(2)Other cardiac tumors: sarcoma, malignant mesenchymoma, metastasis

Carney Syndrome

Notes:

Carney Syndrome =COMPLEX MYOMA(1)multiple myxomas recurring at an increased rate(2)pigmented + myxomatous skin lesions(3)myxoid fibroadenomas of the breast(4)pituitary adenoma + testicular tumors(5)adrenocortical disease (Cushing disease)

Atrial Myxoma Prolapsing Into Mitral Valve Orifice

Note the interval between the opening of aML and pML and the moment that the tumor reaches its maximal anterior excursion at point E when a slight additional opening of the aML results; aML stays open during entire diastole as a result of obstruction to left atrial emptying.

Notes:

PATENT DUCTUS ARTERIOSUS
= PDA = persistence of left 6th aortic arch *Incidence:*9% of all CHD; M:F = 1:2*Associated with*:prematurity, birth asphyxia, high-altitude births, rubella syndrome, coarctation, VSD, trisomy 18 + 21*Normal physiology in mature infant:* increase in arterial oxygen pressure leads to constriction + closure of duct ◊functional closure due to muscular contraction within 10-15 hours◊anatomic closure due to subintimal fibrosis + thrombosis: in 35% by 2 weeks; in 90% by 2 months; in 99% by 1 year ● mostly asymptomatic ● congestive heart failure (rare) usually by 3 months of age if L-to-R shunt is large ● continuous murmur ● bounding peripheral pulses (intraaortic pressure runoff through PDA) CXR (mimics VSD): √ LA enlargement√ enlarged pulmonary artery segment√ increase of pulmonary vasculature (less flow directed to LUL)√ enlarged RV + LV√ enlarged ascending aorta + aortic arch (thymus may obscure this)√ prominent ductus infundibulum (diverticulum)= prominence between aortic knob + pulmonary artery segment√ obscured aortopulmonary window√ "railroad track" = calcified ductus arteriosusECHO: √ LA:Ao ratio ≥1.2:1 (signalizes significant L-to-R shunt)Angio: √ catheter course from RA to RV, main pulmonary artery, PDA, descending aorta√ communication from aorta (distal to left subclavian artery) to left pulmonary artery on AP / LAT / LAO aortogram

PDA In Premature Infant Beneficial PDA Nonbeneficial PDA

Notes:

PDA In Premature Infant Premature infant not subject to medial muscular hypertrophy of small pulmonary artery branches (which occurs in normal infants subsequent to progressive hypoxia in 3rd trimester) ● CHF*Cause:* (a)pulmonary artery pressure remains low without opposing any L-to-R shunts (PDA / VSD)(b)ductus arteriosus remains open secondary to hypoxia in RDS√ recurrence of alveolar airspace filling after resolution of RDS√ granular pattern of hyaline membrane disease becomes more opaque√ enlargement of heart (masked by positive pressure ventilation)*Rx:* (a)Medical therapy:(1)supportive oxygen, diuretics, digitalis(2)avoid fluid overload (not to increase shunt volume)(3)antiprostaglandins = indomethacin opposes prostaglandins, which are potent duct dilators(b)Surgical ligation

Notes:

Beneficial PDA = compensatory effect of PDA in: 1.Tetralogy of Fallotcyanosis usually occurs during closure of duct shortly after birth 2.Eisenmenger pulmonary hypertensionPDA acts as escape valve shunting blood to descending aorta 3.Interrupted aortic archsupply of lower extremity via PDA

Notes:

Nonbeneficial PDA in L-to-R shunts (VSD, aortopulmonic window) a PDA increases shunt volume

Notes:

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PENETRATING AORTIC ULCER
=characterized by ulceration of atheromatous plaque that disrupts the internal elastic lamina + results in hemorrhage into media / rupture through wall of aortaLocation:middle of descending thoracic aortaAngio: √ ulcerated atherosclerotic plaque√ aortic wall thickeningCECT: √ focally ulcerated plaque√ intramural hematoma cannot be differentiated from intraluminal thrombus / atherosclerotic plaqueMR: √ deeply ulcerated aortic plaque√ subacute hematoma in aortic wall indicated by high signal intensity on T1WI + T2WI (methemoglobin) either localized or mimicking type 3 dissection√ aortic rupture with contained hematoma*DDx:*(1)Aortic dissection (intimal flap, patent false lumen)(2)Atheroma / thrombus (low signal intensity on T1WI + T2WI)

Notes:

PERICARDIAL DEFECT
=failure of pericardial development secondary to premature atrophy of the left duct of Cuvier (cardinal vein), which fails to nourish the left pleuropericardial membrane*Incidence:*1:13,000; M:F = 3:1*Age at detection:*newborn to 81 years (mean 21 years)Location: (a)foraminal defect on left side(35%)(b)complete absence on left side(35%)(c)diaphragmatic pericardial aplasia(17%)(d)total bilateral absence(9%)(e)foraminal defect on right side(4%)*Associated with* (in 30%): (1)Bronchogenic cyst (30%)(2)VSD, PDA, mitral stenosis(3)Diphragmatic hernia, sequestration ● mostly asymptomatic ● ECG: right axis deviation, right bundle branch block ● palpitations, tachycardia, dyspnea, dizziness, syncope ● positional discomfort while lying on left side ● nonspecific intermittent chest pain (lack of pericardial cushioning, torsion of great vessels, tension on pleuropericardial adhesions, pressure on coronary arteries by rim of pericardial defect)√ size:-small foraminal defect=no abnormality-large defect=herniation of cardiac structures / lung-complete absence=levoposition of heart √ absence of left pericardial fat-pad√ levoposition of heart with lack of visualization of right heart border√ prominence / focal bulge in the area of RVOT, main pulmonary artery, left atrial appendage√ sharp margination + elongation of left heart border√ insinuation of lung between heart + left hemidiaphragm√ insinuation of lung between aortic knob + pulmonary a.√ increased distance between heart + sternum secondary to absence of sternopericardial ligament (cross-table lateral projection)√ pneumopericardium following pneumothorax√ NO tracheal deviation*Rx:*foraminal defect requires surgery because of(a) herniation + strangulation of left atrial appendage (b) herniation of LA / LV (1)closure of defect with pleural flap(2)resection of pericardium

Notes:

PERSISTENT FETAL CIRCULATION
=PERSISTENT PULMONARY HYPERTENSION OF THE NEWBORN=delay in transition from intra- to extrauterine pulmonary circulation*Cause:* primary disorder related to birth asphyxia, concurrent parenchymal lung disease (meconium aspiration, pneumonia, pulmonary hemorrhage, hyaline membrane disease, pulmonary hypoplasia), concurrent cardiovascular disease, hypoxic myocardial injury, hyperviscosity syndromes) ● labile PO_2 ● structurally normal heart

Notes:

POLYARTERITIS NODOSA

=PERIARTERITIS NODOSA = systemic necrotizing inflammation of medium-sized + small muscular arteries without glomerulonephritis or vasculitis in arterioles, capillaries, venules*Incidence:*rare (2 new cases/million/year); M > F*Etiology:*? deposition of immune complexes*Path:*mucoid degeneration + fibrinoid necrosis begins within media; absence of vasculitis in vessels other than arteries (DDx: necrotizing angiitis, mycotic aneurysm)*Histo:*polymorphonuclear cell infiltrate in all layers of arterial wall + perivascular tissue (acute phase), mononuclear cell infiltrate, intimal proliferation, thrombosis, perivascular inflammation (chronic stage)*Associated with:*hepatitis B antigenemia ● low-grade fever, myalgia, arthralgias ● malaise, abdominal pain, weight loss ● tender subcutaneous nodules (15%) ● elevated ESR, thrombocytosis, anemia ● peripheral neuropathy ● painless hematuriaLocation:all organs may be involved, kidney (85%), heart (65%), liver (50%), pancreas, bowel, CNS (cerebrovascular accident, seizure) @Kidney (most frequently affected organ)√ multiple small intrarenal aneurysms (interlobar, arcuate, interlobular arteries)√ aneurysms may disappear (thrombosis) or appear in new locations√ arterial narrowing + thrombosis (chronic stage / healing stage)√ multiple small cortical infarcts*Cx:*perinephric / subcapsular hemorrhage (rupture of aneurysm)@ Chest (involved in 70%) √ cardiac enlargement / pericardial effusion (14%)√ pleural effusion (14%)√ pulmonary venous engorgement (21%)√ massive pulmonary edema (4%)√ linear densities / platelike atelectasis (10%)√ wedge-shaped / round peripheral infiltrates of nonsegmental distribution (14%) (simulating thromboembolic disease with infarction)√ cavitation may occur√ interstitial lower lung field pneumonitis@Liver (66%)@Mesenteric vessels (50%) ● abdominal pain, ulcer formation, GI bleeding, intestinal infarction@Skeletal muscle (39%)@Skin (20%) Angiography (61% sensitivity, 80% true-positive rate): √ 1-5 mm saccular aneurysms of small + medium-sized arteries in 60-75% as a result of necrosis of the internal elastic lamina (HALLMARK)√ luminal irregularities + stenoses of arteries√ arterial occlusions + small tissue infarctions *Cx:*hypertension, renal failure, hemorrhage secondary to aneurysm rupture, organ infarction due to vessel thrombosis, gangrene of fingers / toes*Rx:*steroids (50% 5-year survival rate)

Notes:

POLYSPLENIA SYNDROME
=BILATERAL LEFT-SIDEDNESS*Age:*presentation in infancy / adulthood; M < F*Associated with:* (a)CHD (90-95%):APVR (70%), dextrocardia (37%), ASD (37%), ECCD (43-65%), pulmonic valvular stenosis (23%), TGA (13-17%), DORV (13-20%)(b)GI abnormalities:esophageal atresia, TE fistula, gastric duplication, preduodenal portal vein, duodenal webs + atresia, short bowel, mobile cecum, malrotation, semiannular pancreas, biliary atresia, absent gallbladder(c)GU anomalies (15%):renal agenesis, renal cysts, ovarian cysts(d)Vertebral anomalies, common celiac trunk-SMA ● heart murmur, CHF, occasional cyanosis ● leftward / superiorly directed P-wave vector ● heart block (due to ECCD) ● extrahepatic biliary obstruction√ bilateral morphologic LA appendages: pointed, tubular, narrow-based@Lung√ bilateral morphologic left lungs (68%), normal (18%), bilateral R-sided lungs (7%)√ bilateral hyparterial bronchi (= arteries projecting superior to bronchi on PA view + posterior to tracheobronchial tree on LAT view)√ normal / increased pulmonary vascularity√ bilateral SVC (50%)√ large azygos vein (MOST SPECIFIC sign) may mimic aortic arch√ absence of middle lobe fissure√ cardiac apex on R / in midline@Abdomen√ presence of ≥2 spleens (usually two major + indefinite number of splenules) located on both sides of the mesogastrium (esp. greater curvature of stomach)√ hepatic symmetry√ absence of gallbladder (50%)√ stomach on right (40%) / left side√ malrotation of bowel (80%)√ azygos / hemiazygos continuation with interruption of hepatic segment of IVC (65-70%)√ preduodenal portal veinOB-US: √ absence of intrahepatic IVC√ aorta anterior to spine in midline√ "double vessel" sign = 2 vessels of similar size in paraspinous location posterior to heart = aorta + azygos vein on left / right side of spine*Prognosis:*50% mortality by 4 months;75% mortality by 5 years; 90% mortality by midadolescence

Notes:

POPLITEAL ARTERY ENTRAPMENT SYNDROME

=popliteal artery classically winding medially and then inferiorly to the tendinous insertion of the medial head of the gastrocnemius*Incidence:*35 cases in American surgical literature; bilateral in up to 66%*Cause:*anomalous development and course of medial head of gastrocnemius muscle, which attaches to medial femoral condyle after development of primitive popliteal artery in 20 mm embryo slinging around lateral aspect of popliteal a.*Pathophysiology:* flow unimpeded when muscle relaxed; increased arterial angulation with muscle contraction (early); progressive intimal hyperplasia ("atheroma" = misnomer) due to microtrauma in area of repeated arterial compression; ultimately occlusion / thrombosis within aneurysm (late) *Age:*<35 years in 68%; age peaks at 17 and 47 years; M:F = 9:1 ● slowly progressive intermittent unilateral calf claudication (early) esp. during periods of prolonged standing ● acute ischemia of leg with permanent occlusion of popliteal a. (late)√ posterior tibial pulse obliterated during active plantar flexion against resistance√ PVR has 40% false-positive results√ ankle-arm index reduced during active muscle contraction√ Doppler waveforms of posterior tibial a. diminished during muscle contractionsAngio (biplanar views with hyperextended knee): √ medial deviation of artery (29%), popliteal stenosis (11%), poststenotic dilatation (8%)*Dx:* √ arteriography with typical medial deviation of popliteal a. before + after gastrocnemius contraction√ popliteal a. thrombosis / occlusion*Cx:*popliteal a. aneurysm*DDx:*cystic adventitial disease of popliteal a., arterial embolism, premature arteriosclerosis, popliteal aneurysm with thrombosis, popliteal a. trauma, popliteal a. thrombosis, Buerger disease, spinal cord stenosis (= neurogenic claudication)

Notes:

PRIMARY PULMONARY HYPERTENSION
=PLEXOGENIC PULMONARY ARTERIOPATHY*Diagnosis per exclusion:* clinically unexplained progressive pulmonary arterial hypertension without evidence for thromboembolic disease + pulmonary venoocclusive disease *Histo:*plexiform + angiomatoid lesions = tortuous channels within proliferation of endothelial cells*Age:*3rd decade; M < F ● dyspnea on exertion, syncope ● easy fatigability ● hyperventilation ● chest pain ● hemoptysis

Notes:

PSEUDOCOARCTATION
=AORTIC KINKING = elongated redundant thoracic aorta with acute kink / anterior buckling just distal to origin of left subclavian artery at lig. arteriosum=variant of coarctation without a pressure gradient*Age:*12-64 years*Associated with:* hypertension, bicuspid aortic valve, PDA, VSD, aortic / subaortic stenosis, single ventricle, ASD, anomalies of aortic arch branches ● asymptomatic ● ejection murmur ● NO pressure gradient across the buckled segment√ anteromedial deviation of aorta√ "chimney-shaped" high aortic arch (in children)√ rounded / oval soft-tissue mass in left paratracheal region + superior to presumed normally positioned aortic arch [secondary to elongation of ascending aorta + aortic arch] (in adults)√ anterior displacement of esophagus√ NO rib notching / dilatation of brachiocephalic arteries / LV enlargement / poststenotic dilatationAngio: √ high position of aortic arch√ "figure 3 sign" = notch in descending aorta at attachment of short ligamentum arteriosum*DDx:*true coarctation, aneurysm, mediastinal mass

Notes:

PULMONARY ATRESIA
=CONGENITAL ABSENCE OF PULMONARY ARTERY=atretic pulmonary valve with underdeveloped pulmonary artery distally*May be associated with:*hypogenetic lungCXR: √ small hemithorax of normal radiodensity√ mediastinal shift to affected side√ elevation of ipsilateral diaphragm√ reticular network of vessels on affected side (due to systemic collateral circulation from bronchial arteries)√ rib notching from prominence of intercostal arteries (due to large transpleural collateral vessels)OB-US: √ small / enlarged / normal right ventricle√ progressive atrial enlargement (tricuspid regurgitation)√ flow reversal in ductus arteriosus + main pulmonary artery (most reliable) **Pulmonary Atresia With Intact Interventricular Septum** *Associated with:* ASD (R-to-L shunt)Type I:no remaining RV, no tricuspid regurgitation√ moderately enlarged RA (depending on size of ASD)Type II:normal RV with tricuspid regurgitation√ massive enlargement of RA√ cardiomegaly (LV, RA) √ concave / small pulmonary artery segment√ diminished pulmonary vascularity

Notes:

PULMONARY VENO-OCCLUSIVE DISEASE
=fibrous narrowing of intrapulmonary veins in the presence of a normal left heart characterized by pulmonary arterial hypertension, pulmonary edema, normal wedge pressures*Age:*children, adolescents; M:F = 1:1*Histo:*fibrous narrowing + thrombosis in up to 95% of pulmonary veins√ pulmonary edema√ pleural effusions√ delayed filling of normal main pulmonary veins + left heart*Prognosis:*poor (no effective therapy)

Notes:

PULMONIC STENOSIS

Pulmonary artery stenosis without VSD = 8% of all CHD ● mostly asymptomatic ● cyanosis / heart failure ● loud systolic ejection murmur√ systolic doming of pulmonary valve (= incomplete opening)√ normal / diminished / increased pulmonary vascularity (depending on presence + nature of associated malformations)√ enlarged pulmonary trunk + left pulmonary artery (poststenotic dilatation)√ prominent left pulmonary artery + normal right pulmonary artery√ hypertrophy of RV with reduced size of RV chamber√ elevation of cardiac apex√ increased convexity of anterior cardiac border on LAO√ diminution of retrosternal clear space√ cor pulmonale √ mild enlargement of LA (reason unknown)√ calcification of pulmonary valves in older adults (rare)*Prognosis:*death at mean age of 21 years if untreated

Subvalvular Pulmonic Stenosis Valvular Pulmonic Stenosis Supravalvular Pulmonic Stenosis

Notes:

Subvalvular Pulmonic Stenosis A.INFUNDIBULAR PULMONIC STENOSIStypically in tetralogy of Fallot B.SUBINFUNDIBULAR PULMONIC STENOSIS=hypertrophied anomalous muscle bundles crossing portions of RV*Associated with:*VSD (73-85%)(a)low type:courses diagonally from low anterior septal side to crista posteriorly (b)high type:horizontal defect across RV below infundibulum

Notes:

Valvular Pulmonic Stenosis 1.CLASSIC / TYPICAL PULMONIC VALVE STENOSIS (95%)= commissural fusion of pulmonary cusps *Age of presentation:*childhood ● pulmonic click ● ECG: hypertrophy of RV√ thickened dome-shaped valve√ dilated main + left pulmonary artery√ jet of contrast*Rx:* balloon valvuloplasty 2.DYSPLASTIC PULMONIC VALVE STENOSIS (5%)=thickened redundant distorted cusps, immobile secondary to myxomatous tissue ● NO click√ NO poststenotic dilatation*Rx:*surgical resection of redundant valve tissueCXR: √ normal pulmonary vascularity√ normal-sized heartAngio: √ increase in trabecular pattern of RV√ hypertrophied crista supraventricularis (lateral projection) TRILOGY OF FALLOT (infantile presentation) (1) severe pulmonic valvular stenosis (2) hypertrophy of RV (3) ASD with R-to-L shunt (increased pressure in RA forces foramen ovale open)

Notes:

Supravalvular Pulmonic Stenosis 60% of all pulmonary valve stenoses Site of narrowing:pulmonary trunk, pulmonary bifurcation, one / both main pulmonary arteries, lobar pulmonary artery, segmental pulmonary arteryShape of narrowing: (a)localized with poststenotic dilatation(b)long tubular hypoplasia*May be associated with:* (1)Valvular pulmonic stenosis, supravalvular aortic stenosis, VSD, PDA, systemic arterial stenoses(2)Familial peripheral pulmonic stenoses + supravalvular aortic stenosis(3)Williams-Beuren syndrome: PS, supravalvular AS, peculiar facies(4)Ehlers-Danlos syndrome(5)Postrubella syndrome: peripheral pulmonic stenoses, valvular pulmonic stenosis, PDA, low birth weight, deafness, cataract, mental retardation(6)Tetralogy of Fallot / critical valvular pulmonic stenosis

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Notes:

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RAYNAUD SYNDROME
=episodic digital ischemia in response to cold / emotional stimuli*Pathogenesis:* (1)increase in vasoconstrictor tone(2)low blood pressure(3)slight increase in blood viscosity(4)immunologic factors (4-81%)(5)cold provocation ● exaggerated response of digit to cold / emotional stress: ● numbness + loss of tactile perception ● demarcated pallor / cyanosis ● hyperemic throbbing during rewarming ● sclerodactyly ● small painful ulcers at tip of digit

Raynaud Disease Raynaud Phenomenon

Notes:

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Raynaud Disease = PRIMARY VASOSPASM = SPASTIC FORM =exaggerated cold-induced constriction of smooth muscle cells in otherwise normal artery*Cause:*? acquired adrenoreceptor hypersensitivity*May be associated with:* early stages of autoimmune disorders*Age:*most common in young women ● usually affects all fingers of both hands equally√ normal segmental arm + digit pressures at room temperature√ peaked digit volume pulse = rapid rise in systole, anacrotic notch just before the peak, dicrotic notch high on the downslopePPG: √ flat-line tracing at low temperatures (10°-22°C) with sudden reappearance of normal waveform at 24-26°C = "threshold phenomenon"

Notes:

Raynaud Phenomenon =SECONDARY VASOSPASM WITH OBSTRUCTION=OBSTRUCTIVE FORM=digital artery occlusion due to stenotic process in normally constricting artery / associated with an abnormally high blood viscosity*Cause:* 1.Atherosclerosis (most frequent)(a)embolization from an upstream lesion(b)occlusion of major arteries supplying arm2.Arterial trauma3.End stage of many autoimmune disorders: eg, scleroderma, rheumatoid arthritis, systemic lupus erythematosus4.Takayasu disease5.Buerger disease6.Drug intoxication (ergot, methysergide)7.Dysproteinemia8.Primary pulmonary hypertension9.Myxedema ● normal vasoconstrictive response to cold√ reduced segmental arm + digit pressures at room temperaturePPG (76% sensitivity, 92% specificity): √ flat-line / barely detectable tracing at low temperature with gradual increase of amplitude upon rewarmingHand magnification angiography: 1.Baseline angiogram with ambient temperature2.Stress angiogram immediately following immersion of hand in ice water for 20 seconds

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Notes:

RHABDOMYOMA OF HEART
=benign hamartoma arising from myocardium*Prevalence:*most common cardiac tumor in infancy + childhood*Histo:*"spider cell" = central nucleus surrounded by clear cytoplasm and radial extensions*Associated with:* tuberous sclerosis (in 50-86%) ● asymptomatic (incidental detection) ● obstructed blood flow, murmur, arrhythmia ● heart failure ● supraventricular tachycardia (accessory conductive pathways within tumor)Location:usually multiple; ventricular wall with intramural growth + tendency to involve interventricular septum; atrial wall (rare)US: √ fetal nonimmune hydrops√ solid echogenic sessile mass ± intracavitary component bulging into ventricular outflow tract / atrioventricular valveMR: √ tumor hyperintense to myocardium on T1WI*Prognosis:*may regress spontaneously in patients <4 years of age*DDx:*fibroma (solitary centrally calcified + cystic tumor, in ventricular myocardium, associated with Gorlin syndrome), teratoma (single intrapericardial multicystic mass), hemangioma (arise from RT atrium, pericardial effusion, skin hemangiomas)

Notes:

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SINGLE VENTRICLE
= UNIVENTRICULAR HEART = DOUBLE INLET SINGLE VENTRICLE = failure of development of interventricular septum ± absence of one atrioventricular valve (mitral / tricuspid atresia) ± aortic / pulmonic stenosis ● conduction defect (aberrant anatomy of conduction system)√ two atrioventricular valves connected to a main ventricular chamber√ the single ventricle may be a LV (85%) / RV / undetermined√ a second rudimentary ventricular chamber may be present, which is located anteriorly (in left univentricle) / posteriorly (in right univentricle)√ rudimentary chamber ± connection to one great artery√ may be associated with tricuspid / mitral atresia

Notes:

SINUS OF VALSALVA ANEURYSM
=deficiency between aortic media + annulus fibrosis of aortic valve resulting in distension + eventual aneurysm formation*Age:*puberty to 30 years of ageSite:right sinus / noncoronary sinus (>90%)◊Right sinus usually ruptures into RV, occasionally into RA◊Noncoronary sinus ruptures into RA ● sudden retrosternal pain, dyspnea, continuous murmur√ shunt vascularity√ cardiomegaly√ prominent ascending aorta

Notes:

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SPLENIC ARTERY ANEURYSM
=most frequent of visceral artery aneurysms*Etiology:*medial degeneration with superimposed atherosclerosis, congenital, mycotic, pancreatitis, trauma, portal hypertension*Predisposed:*women with ≥2 pregnancies (88%)*May be associated with:*fibromuscular disease (in 20%)M:F = 1:2 ● usually asymptomatic ● pain, GI bleedingLocation:intra- / extrasplenic ● calcified wall of aneurysm (2/3)*Cx:*rupture of aneurysm (6-9%, higher during pregnancy) with up to 76% mortality*DDx:*renal artery aneurysm, tortuous splenic artery

Notes:

SUBCLAVIAN STEAL SYNDROME

=stenosis / obstruction of subclavian artery near its origin with flow reversal in ipsilateral vertebral artery at the expense of the cerebral circulation*Incidence:*2.5% of all extracranial arterial occlusions*Etiology:* (a)congenital: interruption of aortic arch, preductal infantile coarctation, hypoplasia of left aortic arch, hypoplasia / atresia / stenosis of an anomalous left subclavian artery with right aortic arch, coarctation with aberrant subclavian artery arising distal to the coarctation(b)acquired: atherosclerosis (94%), dissecting aneurysm, chest trauma, embolism, tumor thrombosis, inflammatory arteritis (Takayasu, syphilitic), ligation of subclavian artery in Blalock-Taussig shunt, complication of coarctation repair, radiation fibrosis*Age:*average 59-61 years; M:F = 3:1;Whites:Blacks = 8:2 *Associated with:*additional lesions of extracranial arteries in 81% ● lower systolic blood pressure by >20-40 mm Hg on affected side ● delayed weak / absent pulse in ipsilateral extremity ● Signs of vertebrobasilar insufficiency (40%): ● syncopal episodes initiated by exercising the ischemic arm ● headaches, nausea, vertigo, ataxia ● mono-, hemi-, para-, quadriparesis, paralysis ● diplopia, dysphagia, dysarthria, paresthesias around mouth ● uni- / bilateral homonymous hemianopia ● Signs of brachial insufficiency (3-10%): ● intermittent / constant pain in affected arm precipitated by increased activity of that arm ● paresthesia, weakness, coolness, numbness, burning in fingers + hand ● fingertip necrosisLocation:L:R = 3:1Color Doppler: √ reversal of vertebral artery flow, augmented by reactive hyperemia (blood pressure cuff inflated above systolic pressure for 5 minutes) / arm exerciseAngio: √ subclavian stenosis / occlusion (aortic arch injection)√ reversal of vertebral artery flow (selective injection of contralateral subclavian / vertebral artery)CAVE:"false steal" = transient retrograde flow in contralateral vertebral artery caused by high-pressure injection *Rx:*bypass surgery, PTA (good long-term results)

Partial Subclavian Steal Steal Syndrome Occult Subclavian Steal Syndrome

Notes:

Partial Subclavian Steal Steal Syndrome =retrograde flow in systole + antegrade flow in diastole

Notes:

Occult Subclavian Steal Syndrome =reverse flow seen only after provocative maneuvers, ie, ipsilateral arm exercise of 5 minutes / 5 minutes inflation of sphygmomanometer > systolic blood pressure levels

Notes:

SUPERIOR VENA CAVA SYNDROME
=obstruction of SVC with development of collateral pathways*Etiology:* (a)Malignant lesion (80-90%)1.Bronchogenic carcinoma (>50%)2.Lymphoma(b)Benign lesion1.Granulomatous mediastinitis (usually histoplasmosis, sarcoidosis, TB)2.Substernal goiter3.Ascending aortic aneurysm 4.Pacer wires / central venous catheters (23%)5.Constrictive pericarditis*Collateral routes:* 1.Esophageal venous plexus = "downhill varices" (predominantly upper 2/3)2.Azygos + hemiazygos veins3.Accessory hemiazygos + superior intercostal veins= "aortic nipple" (visualization in normal population in 5%)4.Lateral thoracic veins + umbilical vein5.Vertebral veins ● head and neck edema (70%) ● cutaneous enlarged venous collaterals ● headache, dizziness, syncope ● with benign etiology: slower onset + progression, both sexes, 25-40 years of age ● with malignancy: rapid progression within weeks, mostly males, 40-60 years of age ● proptosis, tearing ● dyspnea, cyanosis, chest pain ● hematemesis (11%)√ superior mediastinal widening (64%)√ encasement / compression / occlusion of SVC√ dilated cervical + superficial thoracic veins (80%)√ SVC thrombusNUC: √ increased tracer uptake in quadrate lobe + posterior aspect of medial segment of left lobe (umbilical pathway toward liver when injected in upper extremity)

Notes:

SYPHILITIC AORTITIS
=LUETIC AORTITIS*Incidence:*in 10-15% of untreated patients (accounts for death in 1/3)*Path:*periaortitis (via lymphatics), mesaortitis (via vasa vasorum) = primarily disease of media leading to secondary injury of intima, which predisposes the intima to premature calcific atherosclerosis*Age:*between 40 and 65 yearsSite:ascending aorta (36%), aortic arch (24%), descending aorta (5%), sinus of Valsalva (1%), pulmonary artery√ thick aortic wall (fibrous + inflammatory tissue)√ saccular (75%) / fusiform (25%) dilatation of ascending aorta√ small saccular aneurysms often protrude from fusiform aneurysm√ fine pencil-like calcifications of intima (15-20%) in ascending aorta, late in disease*Cx:*(1)stenosis of coronary ostia (intimal thickening)(2)aortic regurgitation (syphilitic valvulitis), rare*DDx:*degenerative calcification of ascending aorta (older population, no aneurysm, no aortic regurgitation)

Notes:

TAKAYASU ARTERITIS

=PULSELESS DISEASE = AORTITIS SYNDROME= AORTOARTERITIS = IDIOPATHIC MEDIAL AORTOPATHY = AORTIC ARCH SYNDROME=granulomatous inflammation of unknown pathogenesis affecting segments of aorta + major aortic branches + pulmonary arteries limited to persons usually <50 years of age◊The only form of aortitis that produces stenosis / occlusion of the aorta!*Etiology:*probably cell-mediated inflammation*Incidence:*2.6 new cases/million/year;2.2% (at autopsy)*Age:*12-66 years; M:F = 1:8; especially in Orientals*Histo:*(a)Acute stage: granulomatous infiltrative process focused on elastic fibers of media of arterial wall consisting of multinucleate giant cells, lymphocytes, histiocytes, plasma cells(b)Fibrotic stage (weeks to years): progressive fibrosis of vessel wall resulting in constriction from intimal proliferation / thrombotic occlusion / aneurysm formation (from extensive destruction of elastic fibers in the media); ultimately leads to fibrosis of intima + adventitia◊Morphologically indistinguishable from temporal arteritis! ● prepulseless / systemic phase of a few months to a year= nonspecific systemic signs + symptoms of fever, night sweats, weakness, weight loss, myalgia, arthralgia ◊Mean interval of 8 years between onset of symptoms and diagnosis ● pulseless phase = signs + symptoms of ischemia of limb (claudication, pulse deficit, bruits) + renovascular hypertension ● erythrocyte sedimentation rate (ESR) >20 mm/hour in 80%Location: Type I:classic pulseless type = brachiocephalic trunk + carotid arteries + subclavian arteriesType II:combination of type I + IIIType III:atypical coarctation type = thoracic and abdominal aorta distal to arch + its major branchesType IV:dilated type = extensive dilatation of the length of the aorta + its branchesCommonly involved: left subclavian artery (<50%), left common carotid artery (20%), brachiocephalic trunk, renal arteries, celiac trunk, superior mesenteric artery, pulmonary arteries (>50%) Infrequently involved: axillary, brachial, vertebral, iliac arteries (usually bilaterally), coronary arteries √ arterial wall thickening + contrast enhancement√ full-thickness calcification (chronic disease)@Aorta√ long + diffuse / short + segmental irregular stenosis / occlusion of major branches of aorta near their origins√ stenotic lesions of thoracic aorta > abdominal aorta√ frequent skipped lesions√ abundant collateralization (late phase)√ aneurysmal dilatation of aorta = diffusely dilated lumen with irregular contours (common in ascending aorta + arch)√ fusiform / saccular aortic aneurysms (10-15%) (common in descending thoracic + abdominal aorta)@Brachiocephalic arteries√ multisegmented dilatation of carotid artery producing segmental septa√ diffuse homogeneous circumferential thickening of vessel wall in proximal common carotid artery√ increase in flow velocity + turbulence√ distal CCA, ICA, ECA spared with dampened waveforms@Pulmonary arteries (50-80%)√ pulmonary arterial lesions specific for Takayasu arteritis : dilatation of pulmonary trunk (19%), nodular thrombi (3%), "pruned tree" appearance of pulmonary arteries (66%)√ systemic-pulmonary artery shuntsCXR: √ widened supracardiac shadow >3.0 cm√ wavy / scalloped appearance of lateral margin of descending aorta √ aortic calcification (15%) commonly in aortic arch + descending aorta√ focal decrease of pulmonary vascularity *Cx:*(1)Cerebrovascular accidents(2)Heart failure due to aortic regurgitation*DDx:*atherosclerosis, temporal arteritis (CCA not involved), fibromuscular dysplasia (in ICA not CCA), idiopathic carotid dissection (ICA), syphilitic aortitis (calcification of ascending aorta)*Rx:*steroids, angioplasty after decline of active inflammation

Notes:

TEMPORAL ARTERITIS
=CRANIAL / GRANULOMATOUS ARTERITIS=POLYMYALGIA RHEUMATICA = GIANT CELL ARTERITIS (poor choice because Takayasu disease is also a giant cell arteritis)=systemic granulomatous vasculitis limited to persons usually >50 years of age*Incidence:*1.7 new cases/million/year*Histo:* (a)acute stage: granulomatous infiltrative process focused on elastic fibers of arterial wall consisting of multinucleate giant cells, lymphocytes, histiocytes, plasma cells(b)fibrotic stage (weeks to years): progressive fibrosis of vessel wall resulting in constriction from intimal proliferation / thrombotic occlusion / aneurysm formation◊Morphologically indistinguishable from Takayasu arteritis!*Age peak:*65-75 years; M:F = 1:3 ● prodromal phase of flulike illness of 1-3 weeks: ● malaise, low-grade fever, weight loss, myalgia ● unilateral headache (50-90%) ● chronic stage: ● jaw claudication (while chewing + talking) ● palpable tender temporal artery ● neuro-ophthalmic manifestations: visual impairment /diplopia / blindness ● polymyalgia rheumatica (50%) = intense myalgia of shoulder + hip girdles ● erythrocyte sedimentation rate (ESR) of 40-140 mm/ hour (HALLMARK)Location:any artery of the body; mainly medium-sized branches of aortic arch (10%), external carotid artery branches (particularly temporal artery); extracranial arteries below neck (9%): subclavian > axillary > brachial > profunda femoris > forearm > calf; commonly bilateral + symmetric√ long smooth stenotic arterial segments with skip areas√ smooth tapered occlusions with abundance of collateral supply√ absence of atherosclerotic changes√ aortic root dilatation + aortic valve insufficiency*Dx:*biopsy of palpable temporal artery*Prognosis:*disease may be self-limiting (1-2 years); 10% mortality within 2-3 years

Notes:

TETRALOGY OF FALLOT

=underdevelopment of pulmonary infundibulum secondary to unequal partitioning of the conotruncus*Incidence:*8% of all CHD; most common CHD with cyanosis after 1 year of lifeTETRAD: 1.Obstruction of right ventricular outflow tract: usually at pulmonary infundibulum, occasionally at pulmonic valve2.VSD3.Right ventricular hypertrophy4.Aorta overriding the interventricular septum*Hemodynamics:* fetus:pulmonary blood flow supplied by retrograde flow through ductus arteriosus with absence of RV hypertrophy / IUGRneonate:R-to-L shunt bypassing pulmonary circulation with decrease in systemic oxygen saturation (cyanosis); pressure overload + hypertrophy of RV secondary to pulmonic-infundibular stenosis*Associated with:* 1.Bicuspid pulmonic valve (40%)2.Stenosis of left pulmonary artery (40%)3.Right aortic arch (25%)4.TE fistula5.Down syndrome6.Forked ribs, scoliosis7.Anomalies of coronary arteries in 10% (single RCA / LAD from RCA) ● cyanosis by 3-4 months of age (concealed at birth by PDA) ● dyspnea on exertion, clubbing of fingers and toes ● "squatting position" when fatigued (increases pulmonary blood flow) ● "episodic spells" = loss of consciousness ● polycythemia, lowered PO_2 values, systolic murmur in pulmonic area √ pronounced concavity in region of pulmonary artery trunk (small / absent PA)√ coeur en sabot (boot-shaped heart) = enlargement of right ventricle√ right-sided aortic arch in 25%√ marked reduction in caliber + number of pulmonary vessels√ asymmetric pulmonary vascularity√ reticular pattern with horizontal course usually in periphery (= prominent collateral circulation of pleuropulmonary connections)OB-US: √ dilated aorta overriding the interventricular septum√ usually perimembranous VSD√ mildly stenotic RV outflow tract√ NO RV hypertrophy in midtrimesterECHO: √ discontinuity between anterior aortic wall + interventricular septum (= overriding of the aorta)√ small left atrium√ RV hypertrophy with small right ventricular outflow tract√ widening of the aorta√ thickening of right ventricular wall + interventricular septum*Prognosis:*spontaneous survival without surgical correction in 50% up to age 7; in 10% up to age 21*Rx:*surgery in early childhood(a)palliative1.Blalock-Taussig shunt = end-to-side anastomosis of subclavian to pulmonary artery opposite aortic arch (64% survival rate at 15 years, 55% at 20 years)2.Pott operation on left = anastomosis of left PA with descending aorta3.Waterston-Cooley procedure = anastomosis between ascending aorta + right pulmonary artery4.Central shunt = Rastelli procedure = tubular synthetic graft between ascending aorta + pulmonary artery(b)corrective open cardiac surgery = VSD-closure + reconstruction of RV outflow tract by excision of obstructing tissue (82% survival rate at 15 years)Operative mortality:3-10%

Pink Tetralogy Pentalogy Of Fallot Trilogy Of Fallot

Notes:

Pink Tetralogy =infundibular hypertrophy in VSD (3%)

Notes:

Pentalogy Of Fallot =tetralogy + ASD

Notes:

Trilogy Of Fallot =pulmonary stenosis + RV hypertrophy + patent foramen ovale

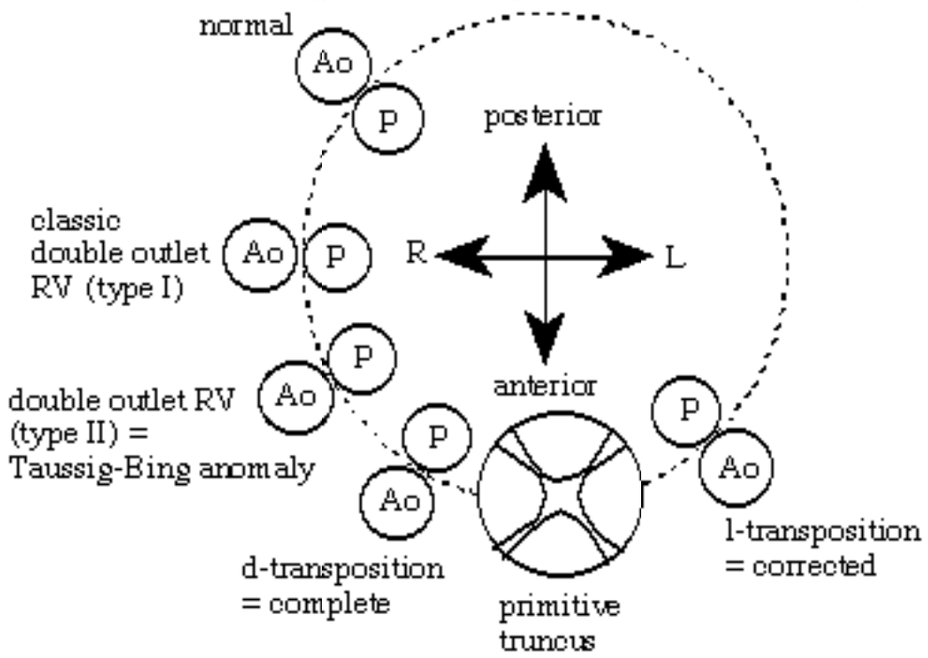
Notes:

THORACIC OUTLET SYNDROME

=compression of nerves, veins, and arteries between chest and arm*Cause:* A.CONGENITAL1.Cervical rib= elevation of floor of scalene triangle with decrease of costoclavicular space*Incidence:*0.5-1% of population◊5-10% of complete cervical ribs cause symptoms◊10-20% of symptomatic patients have a responsible cervical rib*Cx:*aneurysmal dilatation of subclavian a.2.Scalenus minimus muscle (rare)extending from transverse process of 7th cervical vertebra to 1st rib with insertion between brachial plexus + subclavian artery 3.Anterior scalene muscle = scalenus anticus syndrome (most common) = wide / abnormal insertion / hypertrophy of muscle4.Anomalous 1st rib = unusually straight course with narrowing of costoclavicular spaceB.ACQUIRED1.Muscular body habitus=arterial compression in pectoralis minor tunnel2.Slender body habituswith long neck, sagging shoulders 3.Fracture of clavicle / 1st rib (34%)with nonanatomic alignment / exuberant callus 4.Supraclavicular tumor / lymphadenopathy ● pain in forearm + hand which increases upon elevation of arm ● paresthesias of hand + fingers (numbness, "pins and needles") in 95% ● decreased skin temperature, discoloration of hand ● intermittent claudication of fingers (from ischemia) ● hyperabduction maneuver with obliteration of radial pulse (34%) ● Raynaud phenomenon (40%): episodic constriction of small vessels ● supraclavicular bruit (15-30%) Bidirectional Doppler: 1.Adson maneuver (for scalenus anticus muscle) = hold deep inspiration while neck is fully extended + head turned toward ipsilateral and opposite side2.Costoclavicular maneuver (compression between clavicle + 1st rib) = exaggerated military position with shoulders drawn back and downward3.Hyperabduction maneuver (compression by humeral head / pectoralis minor muscle) = extremity monitored through range of 180° abduction√ complete cessation of flow in one position Photoplethysmography: 1.Photo pulse transducer secured to palmar surface of one fingertip of each hand2.Arterial pulsations recorded with arm in(a)neutral position(b)extended 90° to side(c)180° over the head(d)in "military" position with arms at 90° + shoulders pressed back√ complete disappearance of pulse in one position Angio: √ abnormal course of distal subclavian artery√ focal stenosis / occlusion√ poststenotic dilatation of distal subclavian artery√ aneurysm√ stress test: bandlike / concentric constriction√ mural thrombus ± distal embolization√ venous thrombosis / obstruction *DDx:*Cervical disk disease, radiculopathy, spinal cord tumor, trauma to brachial plexus, arthritis, carpal tunnel syndrome, Pancoast tumor, peripheral arterial occlusive disease, aneurysm, causalgia, thromboembolism, Raynaud disease, vasculitis

Notes:

Complete Transposition oOf Great Arteries = TGA = D-TRANSPOSITION = failure of the aorticopulmonary septum to follow a spiral course characterized by (1) aorta originating from RV (2) pulmonary artery originating from LV (3) normal position of atria + ventricles*Incidence*:10% of all CHDVARIATIONS: 1.Complete TGA + intact interventricular septum2.Complete TGA + VSD: CHF due to VSD3.Complete TGA + VSD + PS: PS prevents CHF= longest survival *Hemodynamics*: fetus:no hemodynamic compromise with normal birth weightneonate:mixing of the 2 independent circulations necessary for survivalAdmixture of blood from both circulations via: (1) PDA + patent foramen ovale (when PDA closes worst prognosis) (2) VSD (in 50%) ● cyanosis (most common cause for cyanosis in neonate) 2nd most common cause of cyanosis after tetralogy of Fallot ● symptomatic 1-2 weeks following birthCXR: √ "egg-on-its-side" appearance of heart = narrow superior mediastinum secondary to hypoplastic thymus + hyperaeration + abnormal relationship of great vessels√ cardiac enlargement beginning 2 weeks after birth√ right heart enlargement√ enlargement of LA (with VSD)√ absent pulmonary trunk (99%) = PA located posteriorly in midline√ increased pulmonary blood flow (if not associated with PS)√ midline aorta (30%) / ascending aorta with convexity to the right√ right aortic arch in 3% (difficult assessment due to midline position + small size)OB-US: √ great arteries arise from ventricles in a parallel fashion√ aorta anterior + to right of pulmonary artery (in 60%; rarely side by side)*Prognosis*:overall 70% survival rate at 1 week, 50% at 1 month, 11% at 1 year by natural history*Rx*: (1)Prostaglandin E1 administration to maintain ductal patency(2)Rashkind procedure = balloon septostomy to create ASD(3)Blalock-Hanlon procedure = surgical creation of ASD(4)Mustard operation (corrective) = removal of atrial septum + creation of intraatrial baffle directing the pulmonary venous return to RV + systemic venous return to LV; 79% 1-year survival rate; 64-89% 5-year survival



Notes:

Corrected Transposition Of Great Arteries = CONGENITALLY CORRECTED TRANSPOSITION = L-TRANSPOSITION = anomalous looping of the primordial ventricles associated with lack of spiral rotation of conotruncal septum characterized by (1)Transposition of great arteries(2)Inversion of ventricles (LV on right side, RV on left side):(a)RA connected to morphologic LV(b)LA connected to morphologic RV(3)AV valves + coronary arteries follow their corresponding ventricles*Hemodynamics*:functionally corrected abnormality*Associated with:* (1)usually perimembranous VSD (in >50%)(2)pulmonic stenosis (in 50%)(3)anomaly of left (= tricuspid) atrioventricular valves (Ebstein-like)(4)dextrocardia (high incidence) ● atrioventricular block (malalignment of atrial + ventricular septa)CXR: √ abnormal convexity / straightening in upper portion of left heart border (ascending aorta arising from inverted RV)√ inapparent aortic knob + descending aorta (overlying spine)√ inapparent pulmonary trunk (rightward posterior position) = PREMIER SIGN√ humped contour of lower left heart border with elevation above diaphragm (anatomic RV) √ apical notch (= septal notch)√ increased pulmonary blood flow (if shunt present)√ pulmonary venous hypertension (if left-sided AV valve incompetent)√ LA enlargementAngio: √ original LV on right side: smooth-walled, cylinder- / cone-shaped with high recess emptying into aorta (= venous ventricle)√ original RV on left side: bulbous, triangular shape, trabeculated chamber with infundibular outflow tract into pulmonary trunk (= arterial ventricle)OB-US: √ great arteries arise from ventricles in a parallel fashion√ aortic valve separated from tricuspid valve by a complete infundibulum√ fibrous continuity between pulmonic valve + mitral valve*Prognosis*:(unfavorable secondary to additional cardiac defects) 40% 1-year survival rate, 30% 10-year survival rate

Notes:

TRICUSPID ATRESIA
2nd most common cause of pronounced neonatal cyanosis (after transposition) characterized by absent tricuspid valve, ASD, and small VSD (in most patients) *Incidence*:1.5% of all CHD1.TRICUSPID ATRESIA WITHOUT TRANSPOSITION (80%)(a) without PS (b) with PS (c) with pulmonary atresia 2. TRICUSPID ATRESIA WITH TRANSPOSITION (a) without PS (b) with PS [most favorable combination] (c) with pulmonary atresia◊Usually small VSD + PS (75%) restrict pulmonary blood flow ● progressive cyanosis from birth on, increasing with crying = OUTSTANDING FEATURE (inverse relationship between degree of cyanosis + volume of pulmonary blood flow) ● pansystolic murmur (VSD) ● ECG: left-axis deviationCXR (typical cardiac contour): √ left rounded contour = enlargement + hypertrophy of LV√ right rounded contour = enlarged RA√ flat / concave pulmonary segment√ normal / decreased pulmonary vascularity√ typical flattening of right heart border with transposition (in 15%)*Prognosis*:may survive well into early adulthood*Rx:* 1.Blalock-Taussig procedure (if pulmonary blood flow decreased in infancy)2.Glenn procedure = shunt between IVC + right PA (if total correction not anticipated)3.Fontan procedure = external conduit from RA to pulmonary trunk + closure of ASD (if pulmonary vascular disease has not developed)

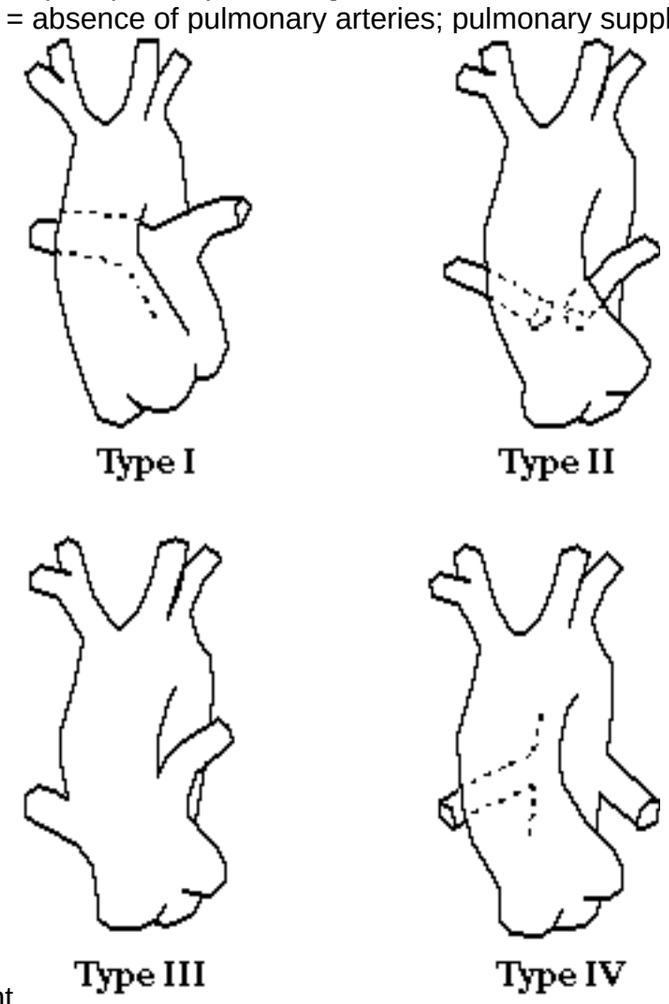
Notes:

TROUSSEAU SYNDROME
=PARANEOPLASTIC THROMBOEMBOLISM*Incidence:*1-11%; higher in terminally ill cancer patients*Tumors:*mucin-secreting adenocarcinoma of GI tract and pancreas (most common), lung, breast, ovary, prostate*Pathogenesis:*(?)(a)tumors activate coagulation + depress anticoagulant function(b)cancer cells cause injury to endothelial lining, activate platelets + coagulation*Type of lesion:*(1)Venous thrombosis(2)Arterial thromboembolism(3)Nonbacterial thrombotic endocarditis◊Patients with thromboembolism have an increased incidence of occult malignancy!*Prevalent criteria:* -absence of apparent cause for thromboembolism-age >50 years-multiple sites of venous thrombosis-simultaneous venous + arterial thromboembolism-resistance to oral anticoagulant therapy-associated other paraneoplastic syndromes-regression of thromboembolism with successful treatment of cancer ● disorders of consciousness (cerebral emboli) ● muscular pain + weakness (emboli to skeletal muscle) ● decompensated disseminated intravascular coagulation√ deep vein thrombosis√ pulmonary embolism√ nonbacterial thrombotic endocarditis (echocardiography)*Rx:*(1)Heparin (more successful than warfarin)(2)Greenfield filter

Notes:

TRUNCUS ARTERIOSUS

=PERSISTENT TRUNCUS ARTERIOSUS= SINGLE OUTLET OF THE HEART =abnormal septation of the conotruncus characterized by(1)one great artery arising from the heart giving rise to the coronary, pulmonary, and systemic arteries, straddling (2)large VSD*Incidence*:2% of all CHD*Types:* TypeI(50%)=main PA + aorta arise from common truncal valveTypeII(25%)=both pulmonary arteries arise from back of trunkTypeIII(10%)=both pulmonary arteries arise from side of trunkTypeIV="Pseudotruncus" = absence of pulmonary arteries; pulmonary supply from systemic collaterals arising from descending aortaSubtype A=infundibular VSD



presentSubtype B=VSD absent *Associated with:* (1)Right aortic arch (in 35%)cyanosis + shunt vascularity + right aortic arch= TRUNCUS(2)Forked ribs*Hemodynamics*: fetus:CHF only with incompetent valve secondary to massive regurgitation from truncus to ventriclesneonate:L-to-R shunt after decrease in pulmonary resistance (massive diversion of flow to pulmonary district) leads to CHF (ventricular overload) / pulmonary hypertension with time ● moderate cyanosis, apparent with crying ● severe CHF within first days / months of life (in large R-to-L shunt) ● systolic murmurCXR: √ cardiomegaly (increased LV volume)√ enlarged LA (50%) secondary to increased pulmonary blood flow√ large "aortic shadow" = truncus arteriosus√ "waterfall / hilar comma sign" = elevated right hilum (30%); elevated left hilum (10%)√ concave pulmonary segment (50%) (type I has left convex pulmonary segment)√ markedly increased pulmonary blood flow, may be asymmetricECHO: √ single arterial vessel overriding the interventricular septum (DDx: tetralogy of Fallot)√ frequently dysplastic + incompetent single semilunar valve with 3-6 leaflets (most commonly 3 leaflets)*Prognosis*:40% 6-months survival rate,20% 1-year survival rate*Rx*:Rastelli procedure (30% no longer operable at 4 years of age) = (a) artificial valve placed high in RVOT and attached via a Dacron graft to main pulmonary artery (b) closure of VSD

Hemitruncus Pseudotruncus Arteriosus

Notes:

Hemitruncus = rare anomaly characterized by (a)one pulmonary artery (commonly right PA) arising from trunk(b)one pulmonary artery arising from RV / supplied by systemic collaterals*Associated with:*PDA (80%), VSD, tetralogy (usually isolated to left PA) ● acyanotic

Notes:

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Pseudotruncus Arteriosus = TRUNCUS TYPE IV = severe form of tetralogy of Fallot with atresia of the pulmonary trunk; entire pulmonary circulation through bronchial collateral arteries (NOT a form of truncus arteriosus in its true sense); characterized by (1) pulmonary atresia (2) VSD with R-to-L shunt (3) RV hypertrophy
*Associated with:*right aortic arch in 50% ● cyanosis√ concavity in area of pulmonary segment√ commalike abnormal appearance of pulmonary artery √ absent normal right and left pulmonary artery (lateral chest film)√ esophageal indentation posteriorly (due to large systemic collaterals)√ prominent hilar + intrapulmonary vessels (= systemic collaterals)√ "coeur en sabot" = RV enlargement√ prominent ascending aorta with hyperpulsations

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Notes:

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VENTRICULAR ANEURYSM

A.CONGENITAL LEFT VENTRICULAR ANEURYSMrare, young Black adult (a)Submitral type:√ bulge at left middle / upper cardiac border(b)Subaortic type:√ small + not visualized√ heart greatly enlarged (from aortic insufficiency)B.ACQUIRED LEFT VENTRICULAR ANEURYSM=complication of myocardial infarction, Chagas disease ● may be asymptomatic + well tolerated for years ● occasionally associated with persistent heart failure, arrhythmia, peripheral embolization

True Ventricular Aneurysm Pseudoaneurysm Of Ventricle

Notes:

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True Ventricular Aneurysm =circumscribed noncontractile outpouching of ventricular cavity with broad mouth + localized dyskinesis*Cause:*sequela of transmural myocardial infarctionLocation: (a)left anterior + anteroapical: readily detected (anterior + LAO views)(b)inferior + inferoposterior: less readily detected (steep LAO + LPO views)Detection rate:50% by fluoroscopy; 96% by radionuclide ventriculography; frequently not visible on CXR√ localized bulge of heart contour = "squared-off" appearance of mid left lateral margin of heart border√ localized paradoxical expansion during systole (CHARACTERISTIC)√ rim of calcium in fibrotic wall (chronic), rare√ akinetic / severely hypokinetic segment√ left ventriculography in LAO, RAO is diagnostic√ wide communication with heart chamber (no neck)*Cx:*wall thrombus with embolization*Prognosis:*rarely ruptures

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Notes:

Pseudoaneurysm Of Ventricle =FALSE ANEURYSM = left ventricular rupture contained by fused layers of visceral + parietal pericardium / extracardiac tissue(a)cardiac rupture with localized hematoma contained by adherent pericardium; typically in the presence of pericarditis(b)subacute rupture with gradual / episodic bleeding*Etiology:*trauma, myocardial infarctionLocation:typically at posterolateral / diaphragmatic wall of LV√ left retrocardiac double density√ diameter of mouth smaller than the largest diameter of the globular aneurysm√ delayed filling*Cx:*high risk of delayed rupture (infrequent in true aneurysms)

Notes:

VENTRICULAR SEPTAL DEFECT
Most common CHD (25-30%): (a) isolated in 20% (b) with other cardiac anomalies in 5%;Acyanotic L-to-R shunt + right aortic arch (in 2-5%)= VSD 1.MEMBRANOUS = PERIMEMBRANOUS VSD(75-80%) Location:posterior + inferior to crista supraventricularis near commissure between right and posterior (= noncoronary) aortic valve cusps*May be associated with:* small aneurysms of membranous septum commonly leading to decrease in size of membranous VSD (their presence does not necessarily predict eventual complete closure) 2.SUPRACRISTAL = CONAL VSD (5-8%)Crista supraventricularis = inverted U-shaped muscular ridge posterior + inferior to pulmonary valve(a)RV view = VSD just beneath pulmonary valve with valve forming part of superior margin of defect(b)LV view = VSD just below commissure between R + L aortic valve cusps*Cx:*right aortic valve cusp may herniate into VSD (= aortic insufficiency)3. MUSCULAR VSD (5-10%)May consist of multiple VSDs; bordered entirely by myocardium Location:(a) inlet portion (b) trabecular portion(c) infundibular / outlet portion 4.ATRIOVENTRICULAR CANAL TYPE=ENDOCARDIAL CUSHION TYPE= POSTERIOR VSD (5-10%)Location:adjacent to septal + anterior leaflet of mitral valve; rare as isolated defect *Hemodynamics:* small bidirectional shunt during fetal life (similar pressures in RV + LV); after birth a decrease in pulmonary arterial pressure + increase in systemic arterial pressure occurs with development of L-to-R shunt (a)small VSD: little / no hemodynamic significance(b)large VSD: pulmonary vascular disease + hypertension will increase RV pressure; eventually leads to shunt reversal (R-to-L shunt)(c)very large VSD: gross right ventricular overload creates CHF soon after birthNATURAL HISTORY OF VSD causing reduction in pulmonary blood flow: 1.Spontaneous closurein 40% within first 2 years of life; 60% by 5 years (65% with muscular VSD, 25% with membranous VSD); with large VSD in 10%; with small VSD in 50% 2.Eisenmenger syndrome= progressive increase in pulmonary vascular resistance through intima + medial hyperplasia; occurs in 10% of large VSDs by 2 years of age 3.RVOT obstructioninfundibular hypertrophy in 3% = pink tetrad 4.Prolapse of right aortic valve cusp= aortic valve insufficiency

CLASSIFICATION: Group I:"maladie de Roger" = small shunt with defect <1 cm; normal pulmonary artery pressure, normal pulmonary vascular resistance; spontaneous closure ● asymptomatic ● heart murmur√ normal plain filmGroup II:moderate shunt with defect of 1-1.5 cm; intermediate pulmonary artery pressure; normal pulmonary vascular resistance; spontaneous closure in large percentage ● respiratory infections, mild dyspnea√ slight prominence of pulmonary vessels (45% shunt)√ slight enlargement of LAGroup III:nonrestrictive large shunt with size equal to aortic valve orifice; pulmonary artery pressure approaching systemic levels; slightly increased pulmonary vascular resistance; pulmonary blood flow 2-4 x systemic flow ● bouts of respiratory infections ● feeding problems, failure to thrive√ prominent pulmonary segment + vessels (= shunt vascularity)√ enlargement of LA + LV√ normal / small aortaGroup IV:Eisenmenger syndrome with shunt reversal into R-to-L shunt; irreversible increase in pulmonary vascular resistance (when pulmonary vascular resistance >0.75 of systemic vascular resistance) ● cyanotic, but less symptomatic; CHF rare√ decrease of pulmonary vessel caliber√ decrease in size of LA + LV CXR (with increase in size of VSD): √ enlargement of LA√ enlargement of pulmonary artery segment√ enlargement of LV√ RV hypertrophy√ increase in pulmonary blood flow (>45% of pulmonary blood flow from systemic circulation)√ Eisenmenger reactionECHO: √ lack of echoes in region of interventricular septum with sharp edges (DDx: artifactual dropout with sound beam parallel to septum); muscular VSD difficult to see√ LA enlargement√ prolapse of aortic valve cusp (in supracristal VSD)√ deformity of aortic cusp (in membranous VSD)Angio: Projections: (a)LAO 60° C-C 20° for membranous + anterior muscular VSD(b)LAO 45° C-C 45° (hepatoclavicular) for posterior endocardial cushion + posterior muscular VSD(c)RAO for supracristal VSD + assessment of RVOT√ RVOT / pulmonary valve fill without filling of RV chamber (in supracristal VSD)*Rx:* (a)large VSD + left heart failure at 3 months of age: aim is to delay closure until child is 18 months of age; pulmonary-to-systemic blood flow >2:1 requires surgery before pulmonary hypertension becomes manifest1.Digitalis + diuretics2.Pulmonary artery banding3.Patching of VSD: surgical approach through RA / through RV for supracristal VSD(b)small VSDs without increase in pulmonary arterial pressure are followed

Notes:

RIGHT UPPER QUADRANT PAIN

A.BILE DUCTS1.Biliary colic / bile duct obstruction2.Acute cholecystitis / cholangitisB.LIVER1.Acute hepatitis: alcoholic, viral, drug-related, toxic2.Hepatic abscess3.Hepatic tumor: metastases, hepatocellular carcinoma, hemangioma, focal nodular hyperplasia, hepatic adenoma4.Hemorrhagic cyst5.Hepatic congestion: acute hepatic congestion, Budd-Chiari syndrome6.Perihepatitis from gonococcal / chlamydial infection (Fitz-Hugh-Curtis syndrome)C.PANCREAS1.Acute pancreatitisD.INTESTINES1.Acute appendicitis2.Peripyloric ulcer3.Small bowel obstruction4.Irritable bowel5.Colitis / ileitis6.Intestinal tumorE.LUNG1.Pneumonia2.Pulmonary infarctionF.KIDNEY1.Acute pyelonephritis2.Ureteral calculus3.Renal / perirenal abscess4.Renal infarction5.Renal tumorG.OTHERS1.Costochondritis2.Herpes zoster

Notes:

Diffuse Hepatic Enlargement A.METABOLIC1.Fatty infiltration2.Amyloid3.Wilson disease4.Gaucher disease5.Von Gierke disease6.Niemann-Pick disease7.Weber-Christian disease8.GalactosemiaB.MALIGNANCY1.Lymphoma2.Diffuse metastases3.Diffuse HCC4.AngiosarcomaC.INFLAMMATION / INFECTION1.Hepatitis2.Mononucleosis3.Miliary TB, histoplasmosis, sarcoid4.Malaria5.Syphilis6.Leptospirosis7.Chronic granulomatous disease of childhood8.SarcoidosisD.VASCULAR1.Passive congestionE.OTHERS1.Early cirrhosis2.Polycystic liver disease

Notes:

Increased Liver Attenuation Abnormal deposits of substances with high atomic numbers A.IRON(a)diffuse iron accumulation1.Genetic / primary hemochromatosis2.Erythropoietic hemochromatosis3.Bantu siderosis4.Transfusional iron overload(b)focal iron accumulation1.Hemorrhagic metastases: choriocarcinoma, melanoma2.Hepatic adenoma3.Siderotic regenerative nodules of cirrhosis◊An iron-poor focus within a siderotic nodule on T2WI is suspect of HCC!4.Focal hemochromatosisB.COPPERWilson disease = hepatolenticular degeneration =increased copper deposits in liver + basal gangliaC.IODINEAmiodarone (= antiarrhythmic drug with 37% iodine by weight) √ 95-145 HU (range of normal for liver 30-70 HU)D.GOLDColloidal form of gold for therapy of rheumatoid arthritis E.THOROTRASTAlpha-emitter with atomic number of 90 F.THALLIUMAccidental / suicidal ingestion of rodenticides (lethal dose is 0.2-1.0 gram) G.ACUTE MASSIVE PROTEIN DEPOSITSH.GLYCOGEN STORAGE DISEASE *mnemonic:*"GG CHAT"**G**old therapy **G**lycogen storage disease **C**yclophosphamide **H**emochromatosis / hemosiderosis **A**miodarone **T**horotrast

Notes:

Generalized Increase In Liver Echogenicity 1.Fatty liver2.Steatohepatitis3.Cirrhosis (fibrosis + fatty liver)4.Chronic hepatitis5.Vacuolar degeneration

Notes:

Primary Benign Liver Tumor A.EPITHELIAL TUMORS(a)hepatocellular1.Regenerative nodules2.Adenomatous hyperplastic nodules3.Focal nodular hyperplasia4.Hepatocellular adenoma(b)cholangiocellular1.Bile duct adenoma2.Biliary cystadenomaB.MESENCHYMAL TUMORS(a)tumor of adipose tissue1.Lipoma2.Myelolipoma3.Angiomyolipoma(b)tumor of muscle tissue1.Leiomyoma(c)tumor of blood vessels1.Infantile hemangioendothelioma2.Hemangioma3.Peliosis hepatis(d)mesothelial tumor1.Benign mesotheliomaC.MIXED TISSUE TUMOR1.Mesenchymal hamartoma2.Benign teratomaD.MISCELLANEOUS1.Adrenal rest tumor2.Pancreatic rest

Notes:

Primary Malignant Liver Tumor A.EPITHELIAL TUMOR(a)hepatocellular1.Hepatoblastoma (7%)2.Hepatocellular carcinoma (75%)(b)cholangiocellular (6%)1.Cholangiocarcinoma2.Biliary cystadenocarcinomaB.MESENCHYMAL TUMOR(a)tumor of blood vessels1.Angiosarcoma2.Epithelioid hemangioendothelioma3.Kaposi sarcoma(b)other tumor1.Embryonal sarcoma2.FibrosarcomaC.TUMOR OF MUSCLE TISSUE1.Leiomyosarcoma2.RhabdomyosarcomaD.MISCELLANEOUS1.Carcinosarcoma2.Teratoma3.Yolk sac tumor4.Carcinoid5.Squamous carcinoma6.Primary lymphoma

Notes:

Focal Liver Lesion A.SOLITARY(a)benign1.Simple cyst / echinococcal cyst2.Cavernous hemangioma3.Abscess4.Hematoma / traumatic cyst5.Adenoma6.Focal nodular hyperplasia7.Fatty change(b)malignant1.Hepatoma2.Metastasis3.Peripheral cholangiocarcinomaB.MULTIPLE(a)benign1.Simple cysts2.Cavernous hemangioma3.Polycystic disease4.Multiple abscesses5.Caroli disease6.Adenoma7.Regenerating hepatic nodules8.Sarcoidosis(b)malignant1.Metastases (most common malignant liver tumor)2.Multifocal hepatoma3.Lymphoma

Notes:

Solitary Echogenic Liver Mass *mnemonic:*"**H**yperechoic **F**ocal **M**asses **A**ffecting the **L**iver"**H**ematoma, **H**epatoma, **H**emangioma, **H**emochromatosis **F**atty infiltration, **F**ocal nodular hyperplasia, **F**ibrosis **M**etastasis **A**denoma **L**ipoma

Notes:

Bulls-eye Lesions Of Liver 1.Candidiasis (in immunocompromised)2.Metastases3.Lymphoma, leukemia4.Sarcoidosis5.Septic emboli6.Other opportunistic infections7.Kaposi sarcoma

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Notes:

Cystic Liver Lesion A.NONNEOPLASTIC1.Congenital hepatic cyst2.Hematoma3.Echinococcal cyst4.Abscess5.Cystic liver disease6.Autosomal dominant polycystic diseaseB.NEOPLASTIC1.Mesenchymal hamartoma2.Undifferentiated sarcoma (embryonal sarcoma)3.Malignant mesenchymoma4.Biliary cystadenoma / cystadenocarcinoma<5% of intrahepatic cysts of biliary origin 5.Lymphangioma6.Necrotic neoplasm7.Cystic metastasis (ovarian / gastric carcinoma)

Notes:

Vascular "Scar" Tumor Of Liver 1.Focal nodular hyperplasia2.Hepatic adenoma3.Giant cavernous hemangioma4.Fibrolamellar hepatocellular carcinoma5.Well-differentiated hepatocellular carcinoma6.Hypervascular metastasis7.Intrahepatic cholangiocarcinoma

Notes:

Low-density Mass In Porta Hepatis 1.Choledochal cyst2.Hepatic cyst3.Pancreatic pseudocyst4.Enteric duplication5.Hepatic artery aneurysm6.Biloma

Notes:

Low-density Hepatic Mass With Enhancement 1.Hepatoma2.Hypervascular metastases (lesions that may be obscured after contrast injection: pheochromocytoma, carcinoid, melanoma)3.Cavernous hemangioma4.Focal nodular hyperplasia with central fibrous scar5.Hepatic adenoma

Notes:

Fat-containing Liver Mass 1.Hepatoma2.Angiomyolipoma

Notes:

Hepatic Calcification A.INFECTION1.Tuberculosis (48%), histoplasmosis, gumma, brucellosis2.Echinococcal cyst (in 33%)3.Chronic granulomatous disease of childhood4.Old pyogenic / amebic abscessB.VASCULAR1.Hepatic artery aneurysm2.Portal vein thrombosis3.HematomaC.BILIARY1.Intrahepatic calculiD.BENIGN TUMORS1.Congenital cyst2.Cavernous hemangioma3.Capsule of regenerating nodules4.Infantile hemangioendotheliomaE.PRIMARY MALIGNANT TUMOR1.Hepatoblastoma (10-20%)2.Cholangiocellular carcinomaF.METASTATIC TUMOR1.Mucinous carcinoma of colon, breast, stomach2.Ovarian carcinoma (psammomatous bodies)3.Melanoma, pleural mesothelioma, osteosarcoma, carcinoid, leiomyosarcoma *mnemonic:*"4H TAG MAP"**H**epatoma **H**emochromatosis **H**emangioma **H**ydatid disease **T**horotrast **A**bscess **G**ranulomas (healed) **M**etastases **A**bsent mnemonic **P**orcelain gallbladder

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Notes:

Portal Venous Gas ◊Should be considered a life-threatening event and sign of bowel infarction + gangrene until proved otherwise!*Etiology:* A.INTESTINAL NECROSIS (in 74% of adults)1.Bowel infarction secondary to arterial and venous occlusions (vascular accidents, superior mesenteric artery syndrome)2.Ulcerative colitis3.Necrotizing enterocolitis associated with mesenteric arterial thrombosis4.Perforated gastric ulcerB.GI OBSTRUCTION1.Small bowel obstruction (duodenal atresia)2.Imperforate anus3.Esophageal atresiaC.MISCELLANEOUS1.Hemorrhagic pancreatitis2.Sigmoid diverticulitis3.Intraabdominal abscess4.Pneumonia5.Iatrogenic injection of air during endoscopy6.Dead fetus7.Diabetes, diarrhea *mnemonic:*"BE NICE"**BE** (air embolism during double contrast barium enema) **N**ecrotizing enterocolitis **I**nfarction (mesenteric) **C**atheterization of umbilical vein **E**rythroblastosis fetalis *Pathogenesis:* 1.Luminal bacterial overgrowth with gas-forming organisms invading the submucosa and veins of the intestinal wall2.Intestinal necrosis with gas infiltrating directly through damaged intestinal wall into intestinal venules (bowel obstruction, ulcer)3.Elevated intraluminal pressure in conjunction with mucosal ulceration*Composition of colonic gas:* methane, carbon dioxide, oxygen, nitrogen, hydrogen √ branching linear gas densities in periphery of liver√ gas in mesenteric vessels√ pneumatosis of intestinal wallUS: √ intensely hyperechoic foci within lumen of portal vein + liver parenchymaDoppler: √ tall sharp bidirectional spikes (overloading of Doppler receiver from strong reflection of gas bubble in bloodstream) superimposed on normal portal vein spectrum *Prognosis:*often fatal within 1 week of diagnosis*DDx:*pneumobilia (central bile ducts close to liver hilum)

Notes:

Hyperperfusion Abnormalities Of Liver = areas of early enhancement on arterial-dominant phase due to decreased portal blood flow / formation of intrahepatic arterioportal shunts / increased aberrant drainage through hepatic veins A.LOBAR / SEGMENTAL1.Portal venous thrombosis2.Obstruction by malignant neoplasm3.Ligation of portal vein4.Cirrhosis with arterioportal shunt5.Hypervascular gallbladder diseaseB.SUBSEGMENTAL1.Obstruction of peripheral portal branches2.Percutaneous needle biopsy / ethanol ablation3.Acute cholecystitisC.SUBCAPSULAR of unknown originD.EARLY-ENHANCING PSEUDOLESIONS IN LEFT HEPATIC LOBE1.Aberrant venous drainage: gastric v., cystic v., capsular v.E.GENERALIZED HETEROGENEOUS1.Cirrhosis

Notes:

Dampening Of Hepatic Vein Doppler Waveform ="portalization" of hepatic vein flow pattern1.Liver cirrhosis2.Budd-Chiari syndrome3.Inferior vena cava obstruction4.Extrinsic compression of hepatic veins5.Various parenchymal abnormalities of liver

Notes:

Aberrant Hepatic Artery =hepatic artery coursing between IVC + portal vein1.Replaced right hepatic artery (50%)2.Right hepatic artery with early bifurcation of common hepatic artery into right + left hepatic arteries (20%)3.Accessory right hepatic artery (15%)4.Replacement of entire hepatic trunk to SMA (15%)

Notes:

Nonvisualization Of Gallbladder On OCG Peak opacification of gallbladder: 14-19 hours (13-35% of dose excreted in urine)A.EXTRABILIARY CAUSES1.Failure to ingest contrast2.Fasting3.Failure to reach absorptive surface of bowel(a)vomiting, nasogastric suction(b)esophageal / gastric obstruction(c)hiatal, umbilical, inguinal hernias(d)Zenker, epiphrenic, gastric, duodenal, jejunal diverticulum(e)gastric ulcer, gastrocolic fistula(f)malabsorption, diarrhea(g)postoperative ileus, severe trauma(h)inflammation: acute pancreatitis, acute peritonitis4.Deficiency of bile saltsCrohn disease, surgical resection of terminal ileum, liver disease, cholestyramine therapy, abnormal communication between biliary system and gastrointestinal tract B.INTRINSIC GALLBLADDER DISEASE1.Cholecystectomy2.Anomalous position3.Obstruction of cystic duct4.Chronic cholecystitis ***Oral Cholecystogram (OCG)*** *Dose:*6 x 0.5 g tablets 2 hours after evening mealA.PATIENT SELECTION ● bilirubin <5 mg% (not necessary if due to hemolysis)◊Contraindicated in serious liver disease!◊Relative contraindications in peritonitis, postoperative ileus, acute pancreatitis!B.TOXICITY1.Nausea + vomiting (also noted in 29% on placebo)2.Immediate anaphylactic response3.Delayed hypotensive reaction (increased risk in cirrhosis)4.Renal failure5.Precipitation of hyperthyroidism

Notes:

Nonvisualization Of Gallbladder On US 1.Contracted gallbladder2.Chronic cholecystitis3.Gallbladder carcinoma4.Perforation of gallbladder5.Congenital absence

Notes:

High-density Bile 1.Hemorrhagic cholecystitis2.Hemobilia3.Prior contrast administration(a)vicarious excretion of urographic agent(b)cholecystopaque4.Milk of calcium bile

Notes:

Displaced Gallbladder A.NORMAL IMPRESSIONby duodenum / colon (positional change) B.HEPATIC MASShepatoma, hemangioma, regenerating nodule, metastases, intrahepatic cyst, polycystic liver, hydatid disease, hepar lobatum (tertiary syphilis), granuloma, abscess C.EXTRAHEPATIC MASS1.Retroperitoneal tumor (renal, adrenal)2.Polycystic kidney3.Lymphoma4.Lymph node metastasis to porta hepatis5.Pancreatic pseudocyst

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Notes:

Alteration In Gallbladder Size NORMAL MEASUREMENTS Size:7-10 cm in length; 2-3.5 cm in widthCapacity:30-50 mLWall thickness:2-3 mm ***Enlarged Gallbladder*** = CHOLECYSTOMEGALY A.OBSTRUCTION1.Cystic duct obstruction (40%)(a)Hydrops: chronic cystic duct obstruction + distension with clear sterile mucus (white bile)(b)Empyema: acute / chronic obstruction with superinfection of bile2.Cholelithiasis causing obstruction (37%)3.Cholecystitis with cholelithiasis (11%)4.Courvoisier phenomenon (10%) = secondary to neoplastic process in pancreas / duodenal papilla / ampulla of Vater / common bile duct5.PancreatitisB.UNOBSTRUCTED (mostly neuropathic)1.S/P vagotomy2.Diabetes mellitus3.Alcoholism4.Appendicitis (in children)5.Narcotic analgesia6.WDHA syndrome7.Hyperalimentation8.Acromegaly9.Kawasaki syndrome10.Anticholinergics11.Bedridden patient with prolonged illness12.AIDS (in 18%)C.NORMAL (2%) ***Small Gallbladder*** 1.Chronic cholecystitis2.Cystic fibrosis: in 25% of patients3.Congenital hypoplasia / multiseptated gallbladder

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Notes:

Diffuse Gallbladder Wall Thickening =anterior wall of gallbladder >3 mmA.INTRINSIC1.Acute cholecystitis2.Chronic cholecystitis (10-25%)3.Xanthogranulomatous cholecystitis4.Hyperplastic cholecystosis (in 91% diffuse)5.Gallbladder perforation6.Sepsis7.Gallbladder carcinoma (in 41% diffuse)8.AIDS cholangiopathy (average of 9 mm in up to 55%)9.Sclerosing cholangitis10.Gallbladder varicesB.EXTRINSIC1.Hepatitis (in 80%)2.Hypoalbuminemia3.Renal failure4.Right heart failure5.Systemic venous hypertension6.Hepatic venous obstruction7.Ascites8.Multiple myeloma9.Portal node lymphatic obstruction10.Cirrhosis11.Acute myelogenous leukemia12.Brucellosis13.Graft-versus-host diseaseC.PHYSIOLOGIC=contracted gallbladder after eating

Notes:

Focal Gallbladder Wall Thickening A.METABOLIC1.Metachromatic sulfatides2.Hyperplastic cholecystosesB.BENIGN TUMOR1.Adenoma: glandular elements (0.2%)2.Papilloma: fingerlike projections (0.2%)3.Villous hyperplasia4.Fibroadenoma5.Cystadenoma: ? premalignant6.Neurinoma, hemangioma7.Carcinoid tumorC.MALIGNANT TUMOR1.Carcinoma of gallbladder: adenocarcinoma / squamous cell carcinoma (in 59% focal)2.Leiomyosarcoma3.Metastases: from malignant melanoma (15%), lung, kidney, esophagus, breast, carcinoid, Kaposi sarcoma, lymphoma, leukemiaD.INFLAMMATION / INFECTION1.Inflammatory polyp: in chronic cholecystitis2.Parasitic granuloma: Ascaris lumbricoides, Paragonimus westermani, Clonorchis, filariasis, Schistosoma, Fasciola3.Intramural epithelial cyst / mucinous retention cyst4.Xanthogranulomatous cholecystitis (in 9% focal)E.WALL-ADHERENT GALLSTONE = embedded stoneF.HETEROTOPIC MUCOSA1.Ectopic pancreatic tissue2.Ectopic gastric glands3.Ectopic intestinal glands4.Ectopic hepatic tissue5.Ectopic prostatic tissue ***Fixed Filling Defects In Gallbladder*** *mnemonic:*"PANTS"**P**olyp **A**denomyomatosis **N**eurinoma **T**umor, primary / secondary **S**tone, wall-adherent

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Notes:

Mobile Intraluminal Mass In Gallbladder 1.Tumefactive sludge2.Blood clot3.Nonshadowing stone

Notes:

Comet-tail Artifact In Liver And Gallbladder A.LIVER1.Foreign metallic body (eg, surgical clip)2.Intrahepatic calcification3.Pneumobilia4.Multiple bile duct hamartoma = von Meyenburg complexB.GALLBLADDER1.Rokitansky-Aschoff sinus2.Intramural stone3.Cholesterolosis of gallbladder

Notes:

Echogenic Fat In Hepatoduodenal Ligament =sign of pericholecystic inflammation1.Cholecystitis2.Perforated duodenal ulcer3.Pancreatitis4.Diverticulitis

Notes:

Gas In Biliary Tree *mnemonic:*"I GET UP"**I**ncompetent sphincter of Oddi (after sphincterotomy / passage of a gallstone) **G**allstone ileus **E**mphysematous cholecystitis (actually in gallbladder) **T**rauma **U**lcer (duodenal ulcer perforating into CBD) **P**ostoperative (eg, cholecystoenterostomy)

Notes:

Obstructive Jaundice In Adult *Etiology:* A.BENIGN DISEASE (76%) 1.Traumatic / operative stricture (44%)2.Calculi (21%)3.Pancreatitis (8%)4.Sclerosing cholangitis (1%)5.Recurrent pyogenic cholangitis6.Parasitic disease (ascariasis)7.Liver cysts8.Aortic aneurysmB.MALIGNANCY (24%)1.Pancreatic carcinoma (18%)2.Ampullary / duodenal carcinoma (8%)3.Cholangiocarcinoma (3%)4.Metastatic disease (2%)from stomach, pancreas, lung, breast, colon, lymphoma *Level and cause of obstruction:* A.INTRAPANCREATIC1.Choledocholithiasis◊Most common cause of biliary obstruction (in 15% of patients with cholelithiasis)!2.Chronic pancreatitis3.Pancreatic carcinomaB.SUPRAPANCREATIC (5%)=between pancreas + porta hepatis1.Cholangiocarcinoma2.Metastatic adenopathyC.PORTA HEPATIS (5%)1.Klatskin tumor2.Spread from adjacent tumor (GB, liver)3.Surgical strictureD.INTRAHEPATIC1.Cystadenoma, cystadenocarcinoma2.Mirizzi syndrome3.Caroli disease4.Cholangitis: recurrent pyogenic ~, sclerosing ~, AIDS cholangitis
Incidence of infected bile in bile duct obstruction: (a)incomplete / partial obstruction in 64%(b)complete obstruction in 10%◊Infection twice as high with biliary calculi than with malignant obstruction!*Organism:* E. coli (21%), Klebsiella (21%), Enterococci (18%), Proteus (15%)
Test SensitivIty For Common Bile Duct Obstruction 1.Intravenous cholangiographydepends on level of bilirubin: <1 mg/dL in 92%; <2 mg/dL in 82%; <3 mg/dL in 40%; >4 mg/dL in <10% False-negative rate: 45% *Cx:*adverse reactions in 4-10%2.US88-90% sensitivity for dilatation of CBD ◊in 27-95% correct level of obstruction determined by US◊in 23-81% correct cause of obstruction determined by US√ CBD >4-8 mm / 10% of patients age in years√ increase in CBD size after fatty meal√ "Swiss cheese sign" = abundance of fluid-filled structures on liver sections√ intrahepatic "double channel" / "shotgun" sign= two parallel tubular structures composed of portal vein + dilated intrahepatic bile ducts√ intrahepatic bile duct >2 mm / >40% of adjacent portal veinFalse-negative:not dilated in acute obstruction (in 70%), sclerosing cholangitis, intermittent obstruction from choledocholithiasisFalse-positive:dilated hepatic artery in cirrhosis / portal hypertension / hepatic neoplasm, patients after cholecystectomy3.CT100% visualization in tumorous obstruction, 60% in nontumorous obstruction 4.NUC√ delayed / nonvisualization of biliary system (93% specificity)√ vicarious excretion of tracer through kidneys*DDx:*Hepatocellular dysfunction (delayed clearance of cardiac blood pool)

Notes:

Neonatal Obstructive Jaundice =severe persistent jaundice in a child beyond 3-4 weeks of age *Cause:* A.INFECTION(a)bacterial:E. coli, syphilis, Listeria monocytogenes(b)viral:TORCH, hepatitis B, Coxsackie, echovirus, adenovirusB.METABOLIC(a)inherited:alpha-1-antitrypsin deficiency, cystic fibrosis, galactosemia, hereditary tyrosinemia(b)acquired:inspissated bile syndrome (= cholestasis due to erythroblastosis); cholestasis due to total parenteral nutritionC.BILIARY TRACT ABNORMALITIES(a)extrahepatic:biliary obstruction / hypoplasia / atresia, choledochal cyst, spontaneous perforation of bile duct, "bile plug" syndrome(b)intrahepatic:ductular hypoplasia / atresiaD.IDIOPATHIC NEONATAL HEPATITIS *mnemonic:*"CAN"**C**holedochal cyst **A**tresia **N**eonatal hepatitis
NUC-imaging regimen: (1)Premedication with phenobarbital (5 mg/kg/day) over 5 days to induce hepatic microsomal enzymes which enhance uptake and excretion of certain compounds and increase bile flow(2)IDA scintigraphy (50 µCi/kg; minimum of 1 mCi)(3)Imaging at 5-minute intervals for 1 hour + at 2, 4, 6, 8, 24 hours

Notes:

Large Nonobstructed CBD 1.Passage of stone (return to normal after days to weeks)2.Common duct surgery (return to normal in 30-50 days)3.Postcholecystectomy dilatation (in up to 16%)4.Intestinal hypomotility5.Normal variant (aging) Fatty-meal sonography (to differentiate from obstruction with 74% sensitivity, 100% specificity) *Method:*peroral Lipomul (1.5 mL/kg) followed by 100 mL of water [cholecystokinin causes contraction of gallbladder, relaxation of sphincter of Oddi, increase in bile secretion], CBD measured before and 45 / 60 minutes after stimulation√ little change / decrease in size = normal response√ increase in size >2 mm = partial obstruction

Notes:

Filling Defect In Bile Ducts A.ARTIFACT1.Pseudocalculus = contracted sphincter of Boyden + Oddi with smooth arcuate contour2.Air bubble: confirmed by positional changes3.Blood clot: spheroid configuration, spontaneous resolution with timeB.BILIARY CALCULIC.MIRIZZI SYNDROME D.NEOPLASM1.Cholangiocarcinoma: irregular stricture, intraluminal polypoid mass2.Others: ampullary carcinoma, hepatoma, villous tumor, hamartoma, carcinoid, adenoma, papilloma, fibroma, lipoma, neuroma, cystadenoma, granular cell myoblastoma, sarcoma botryoidesE.PARASITES1.Ascaris lumbricoides: long linear filling defect / discrete mass if coiled2.Liver fluke (Clonorchis sinensis, Fasciola hepatica): intrahepatic epithelial hyperplasia, periductal fibrosis, cholangitis, liver abscess, hepatic duct stones, common duct obstruction3.Hydatid cyst: after erosion into biliary tree

Notes:

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Bile Duct Narrowing A.BENIGN STRICTURE (44%)(a)trauma1.Postoperative stricture (95-99%)associated with cholecystectomy2.Blunt / penetrating trauma3.Hepatic artery embolization4.Infusion of chemotherapeutic agents(b)inflammation 1.Sclerosing cholangitis2.Recurrent pyogenic cholangitis3.Acute / chronic pancreatitis4.Pancreatic pseudocyst5.Perforated duodenal ulcer6.Erosion by biliary calculus7.Gallstones + cholecystitis8.Abscess9.Radiation therapy10.Papillary stenosis(c)congenital1.Choledochal cystB.MALIGNANT STRICTURE1.Pancreatic carcinoma2.Ampullary carcinoma3.Cholangiocarcinoma4.Compression by enlarged lymph node

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Notes:

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Papillary Stenosis *Etiology:* A.PRIMARY PAPILLARY STENOSIS (10%)1.Congenital malformation of papilla2.Sequelae of acute / chronic inflammation3.AdenomyosisB.SECONDARY PAPILLARY STENOSIS (90%)1.Mechanical trauma of stone passage (choledocholithiasis in 64%; cholecystolithiasis in 26%)2.Functional stenosis: associated with pancreas divisum, history of pancreatitis3.Reflex spasm4.Previous surgical manipulation5.Periampullary neoplasm√ prestenotic dilatation of CBD√ increase in pancreatic duct diameter (83%)√ long smooth narrowing / beak (fibrotic stenosis)√ prolonged bile-to-bowel transit time >45 minutes onTc-IDA scintigraphy

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Notes:

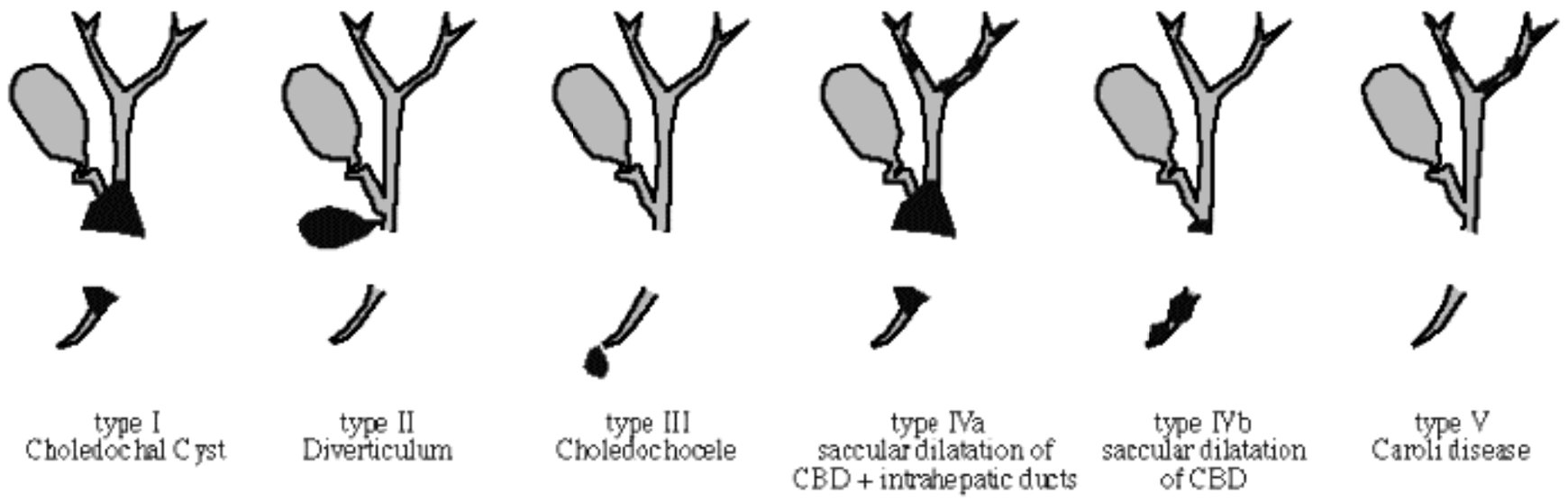
Periampullary Tumor 1.Pancreatic carcinoma (85%)2.Cholangiocarcinoma of distal common bile duct (6%)3.Ampullary tumor (4%)4.Duodenal wall tumoradenocarcinoma, adenoma, carcinoid, smooth muscle tumor

Notes:

Double-duct Sign =dilatation of common bile duct + pancreatic duct1.Ampullary tumor (most common)2.Other periampullary tumor3.Papillary stenosis4.Stone impacted in ampulla of Vater

Notes:

Congenital Biliary Cysts *(Todani classification)* I.Common bile duct cyst = choledochal cyst (77-87%)a.marked cystic dilatation of CBD + CHDb.focal segmental dilatation of CBD distallyc.cylindric dilatation of CBD + CHDII.Diverticulum of extrahepatic ducts (1.2-3%)originating from CBD / CHD III.Choledochocele (1.4-6%)IV.Multiple segmental cystsa.in intra- and extrahepatic ducts (19%)b.in extrahepatic ducts only (rare)V.Intrahepatic cysts = Caroli disease



Classification of Congenital Biliary Cysts

Notes:

Congenital Pancreatic Anomalies 1.Pancreas divisum2.Annular pancreas3.Agenesis of dorsal pancreas*May be associated with:* abnormal situs, polysplenia, intestinal malrotation

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Notes:

Pancreatic Calcification 1.CHRONIC PANCREATITISNumerous irregular stippled calcifications of varying size; predominantly intraductal (a)Alcoholic pancreatitis (in 20-50%): √ calcifications limited to head / tail in 25%(b)Biliary pancreatitis (in 2%)(c)Hereditary pancreatitis (in 35-60%): √ round calcifications throughout gland(d)Idiopathic pancreatitis(e)Pancreatic pseudocyst2.NEOPLASM(a)Microcystic adenoma (in 33%):√ "sunburst" appearance of calcifications(b)Macrocystic cystadenoma In 15%):√ amorphous peripheral calcifications(c)Adenocarcinoma (in 2%): with "sunburst" pattern(d)Cavernous lymphangioma / hemangioma:√ multiple phleboliths(e)Metastases from colon cancer3.INTRAPARENCHYMAL HEMORRHAGE(a)Old hematoma / abscess / infarction(b)Rupture of intrapancreatic aneurysm4.HYPERPARATHYROIDISM (in 20%):50% of patients develop chronic pancreatitis, concomitant nephrocalcinosis ◊indistinguishable from alcoholic pancreatitis5.CYSTIC FIBROSISFine granular calcifications imply advanced pancreatic fibrosis 6.HEMOCHROMATOSIS7.KWASHIORKOR = tropical pancreatitis:◊ indistinguishable from alcoholic pancreatitis

Notes:

Fatty Replacement & Atrophy Of Pancreas 1.Main pancreatic duct obstruction2.Cystic fibrosis3.Schwachman syndrome4.Hemochromatosis5.Viral infection6.Malnutrition

Notes:

Pancreatic Mass A.NEOPLASTIC1.Adenocarcinoma2.Islet cell tumor3.Cystadenoma / -carcinoma4.Solid and papillary neoplasm5.LymphomaB.INFLAMMATORY1.Acute pancreatitis2.Pseudocyst3.Pancreatic abscess

Notes:

Pancreatic Neoplasm *Origin:*-in99%exocrine ductal epithelium-in1%acinar portion of pancreatic glands-in0.1%malignant ampullary tumor with better prognosisA.EXOCRINE NEOPLASM(a)Ductal cell origin1.Ductal adenocarcinoma (90%)2.Ductectatic mucinous tumor=mucin-hypersecreting carcinoma3.Cystic neoplasm (10-15%)-serous microcystic neoplasm-mucinous macrocystic neoplasm4.Solid and papillary epithelial neoplasm (rare)5.Cystic changes of von Hippel-Lindau disease(b)Acinar cell origin1.Acinar cell carcinoma (1%)(c)Indeterminate origin1.Pancreaticoblastoma = infantile pancreatic carcinomaB.ENDOCRINE NEOPLASM(a)Nonfunctioning islet cell tumor(b)Functioning islet cell tumor1.Insulinoma2.Glucagonoma3.Gastrinoma4.Somatostatinoma5.VIPoma6."PP-oma" = pancreatic polypeptide7.CarcinoidC.NONEPITHELIAL ORIGIN1.Lymphoma(a)Primary lymphoma:<1% of pancreatic neoplasms (b)Secondary lymphoma√ large homogeneous solid mass, infrequently with central cystic area√ peripancreatic nodal masses√ peripancreatic vessels displaced + stretched2.Metastasesrenal cell carcinoma, melanoma, lung cancer, breast cancer, ovarian cancer, hepatocellular carcinoma, sarcoma

Notes:

Hypervascular Pancreatic Tumors A.PRIMARYIslet cell tumor, microcystic adenoma, solid and papillary epithelial neoplasm B.METASTASES fromangiosarcoma, leiomyosarcoma, melanoma, carcinoid, renal cell carcinoma, adrenal carcinoma, thyroid carcinoma

Notes:

Pancreatic Cyst 1.Pseudocyst (85%): secondary to obstructive tumor / trauma / acute pancreatitis (in 2-4%), chronic pancreatitis (in 10-15%) [develop within 10-20 days, consolidated after 6-8 weeks]2.Congenital cyst (rare)(a)solitary(b)multiple (when associated with cystic disease of the liver / other organs):adult polycystic kidney disease (hepatic cysts in 90% at autopsy); von Hippel-Lindau disease (pancreatic cysts in 72% at autopsy; in only 25% on CT) 3.Acquired cyst:(a)retention cyst (= exudate within bursa omentalis)(b)parasitic cyst: Echinococcus multilocularis, amebiasis(c)**mucinous ductal ectasia** (= obstruction of pancreatic duct as a result of filling with mucus)√ massive ductal dilatation√ intraluminal filling defects on ERCP4.Cystic pancreatic neoplasm (5-15%):<5% of all pancreatic tumors (a)microcystic adenoma = serous cystadenoma(b)macrocystic adenoma = mucinous cystic neoplasm(c)solid and cystic papillary epithelioid neoplasm(d)cystic islet cell tumor (rare)(e)Variants of pancreatic ductal adenocarcinoma (rare): mucinous colloid adenocarcinoma= ductectatic mucinous tumor of pancreas = mucin-hypersecreting carcinoma; papillary intraductal adenocarcinoma; adenosquamous carcinoma; anaplastic adenocarcinoma (f)pancreatic sarcoma (extremely rare)5.Cystic metastases (3-12% at autopsy): renal cell carcinoma, melanoma, lung tumors, breast carcinoma, hepatocellular carcinoma, ovarian carcinoma6.Retroperitoneal lymphangioma / hemangioma

Notes:

Hyperamylasemia A.PANCREATIC1.Acute / chronic pancreatitis2.Pancreatic trauma3.Pancreatic carcinomaB.GASTROINTESTINAL1.Perforated peptic ulcer2.Intestinal obstruction3.Peritonitis4.Acute appendicitis5.Afferent loop syndrome6.Mesenteric ischemia / infarction7.Portal vein thrombosisC.TRAUMA1.Burns2.Cerebral trauma3.PostoperativeD.OBSTETRICAL1.Pregnancy2.Ruptured ectopic pregnancyE.RENAL1.Transplantation2.Renal insufficiencyF.METABOLIC1.Diabetic ketoacidosis2.DrugsG.PNEUMONIAH.SALIVARY GLAND LESION1.Facial trauma2.Mumps

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Notes:

Nonvisualization Of Spleen 1.Asplenia syndrome2.Polysplenia syndrome3.Traumatic fragmentation of spleen4.Wandering spleen

Notes:

Small Spleen 1.Hereditary hypoplasia2.Irradiation3.Infarction4.Polysplenia syndrome5.Atrophy

Notes:

Splenomegaly √ inferior tip of spleen extends below tip of right lobe of liver√ AP diameter of spleen >2/3 of abdominal diameterA.CONGESTIVE SPLENOMEGALYheart failure, portal hypertension, cirrhosis, cystic fibrosis, portal / splenic vein thrombosis, acute splenic sequestration crisis of sickle cell disease B.NEOPLASMleukemia, lymphoma, metastases, primary neoplasm C.STORAGE DISEASEGaucher disease, Niemann-Pick disease, gargoylism, amyloidosis, diabetes mellitus, hemochromatosis, histiocytosis D.INFECTIONhepatitis, malaria, infectious mononucleosis, kala azar, leishmaniosis, brucellosis, TB, typhoid, syphilis, echinococcosis, subacute bacterial endocarditis E.HEMOLYTIC ANEMIAhemoglobinopathy, hereditary spherocytosis, primary neutropenia, thrombotic thrombocytopenic purpura F.EXTRAMEDULLARY HEMATOPOIESISosteopetrosis, myelofibrosis G.COLLAGEN VASCULAR DISEASEsystemic lupus erythematosus, rheumatoid arthritis, Felty syndrome H.SPLENIC TRAUMAI.OTHERS1.Sarcoidosis√ splenomegaly in up to 60%√ inhomogeneous enhancement after bolus injection (multiple 2-3 cm nodular lesions)√ necrotic mass with focal calcifications2.Hemodialysis

Notes:

Splenic Lesion *mnemonic:*"LCHAIM"**L**ymphoma **C**yst **H**ematoma **A**bscess **I**nfarct **M**etastasis

Notes:

Solid Splenic Lesion A.MALIGNANT TUMOR1.Lymphoma (Hodgkin disease, non-Hodgkin lymphoma, primary splenic lymphoma)-spleen involved in 70%◊ splenomegaly in non-Hodgkin lymphoma indicates involvement in most patients◊30% of patients with splenomegaly have no involvement from non-Hodgkin lymphoma◊ 30% of patients with lymphoma of any kind have splenic involvement without splenomegaly√ homogeneous splenomegaly (from diffuse infiltration)√ miliary nodules√ large 2-10 cm nodules (10-25%)√ nodes in splenic hilum (50%) in NHL; uncommon in Hodgkin disease2.Metastasis (7%)melanoma (6-34%), breast carcinoma (12-21%), bronchogenic carcinoma (9-18%), colon carcinoma (4%), renal cell carcinoma (3%), ovary (8%), prostate (6%), stomach (7%), pancreas, endometrial cancer 3.Angiosarcoma4.Malignant fibrous histiocytoma, leiomyosarcoma, fibrosarcomaB.BENIGN TUMOR1.Hamartoma = **Splenoma** √ solid / cystic splenic mass of low attenuation2.Hemangioma3.Hematopoietic4.Sarcoidosis√ nodular lesions in liver and spleen in 5-15% (= coalescent granulomata) occurring within 5 years of diagnosis√ hepatosplenomegaly√ abdominal adenopathy (mean size of 2.6 cm)5.Gaucher disease (islands of RES cells laden with glucosylceramide)6.Inflammatory pseudotumor7.LymphangiomaC.SPLENIC INFARCTION

Notes:

Cystic Splenic Lesion A.CONGENITAL1.Epidermoid cyst = true cyst = congenital cystB.VASCULAR1.Splenic laceration / fracture2.Hematoma3.**False cyst** = posttraumatic cyst = nonpancreatic pseudocyst of the spleen◊80% of all splenic cysts are pseudocysts (= secondary cysts)*Cause:*cystic end stage of old trauma, infection, infarction!√ internal echoes from debris√ calcifications within cyst wall may resemble eggshell√ smaller size than true cyst4.Cystic degeneration of infarct(a)occlusion of splenic a. / branches (hemolytic anemia, endocarditis, SLE, arteritides, pancreatic cancer)(b)venous thrombosis of splenic sinusoids (massive splenomegaly)5.Peliosis*Associated with:* Hodgkin disease, myeloma, disseminated cancer, TB, anabolic + contraceptive steroids, thorium dioxide injection, viral infection C.INFECTION / INFLAMMATION1.Pyogenic abscess*Prevalence:*0.1-0.7%*Cause:*hematogenous spread (75%), penetrating trauma (15%), infarction (10%)*Predisposed:*endocarditis, drug abuse, penetrating trauma, neoplasm, sickle cell disease ● fever, chills, LUQ pain (in <50%)√ irregular borders without capsule√ gas within abscess*Rx:*76% success rate for percutaneous drain2.Microabscesses*Organism:*fungus (especially Candida, Aspergillus, Cryptococcus)*Prevalence:*26% of splenic abscesses*Predisposed:*immunocompromised patient√ splenomegaly√ multiple hypoattenuating "target" lesions of 5-10 mm often associated with hepatic + renal involvement3.Granulomatous infection(a)Mycobacterium tuberculosis: miliary TB√ mild splenomegaly uncommon(b)M. avium-intracellulare√ marked splenomegaly in 20%4.Pneumocystis carinii infection√ splenomegaly + multiple hypoattenuating foci5.Parasitic cyst (Echinococcus)*Prevalence:*in <2% of patients with hydatid disease6.Pancreatic pseudocyst*Prevalence:*in 1.1-5% of patients with pancreatitis D.CYSTIC NEOPLASM1.Cavernous hemangioma◊Most common primary neoplasm of the spleen!2.Lymphangioma / lymphangiomatosis3.Lymphoma (most common malignant neoplasm!)4.Necrotic metastasis:◊In 7% of patients with widespread metastasis!malignant melanoma (in 50%); breast, lung, ovarian, pancreatic, endometrial, colonic, prostatic, carcinoma; chondrosarcoma

Notes:

Increased Splenic Density 1.Sickle cell anemia (in 5% of sicklers)2.Hemochromatosis3.Thorotrast exposure4.Lymphangiography

Notes:

Splenic Calcification

A.DISSEMINATED1.Phlebolith: visceral angiomatosis2.Granuloma (most common): histoplasmosis, TB, brucellosisB.CAPSULAR & PARENCHYMAL1.Pyogenic / tuberculous abscess2.Pneumocystis carinii infection2.Infarction (multiple)3.HematomaC.VASCULAR1.Splenic artery calcification2.Splenic artery aneurysm3.Splenic infarctD.CALCIFIED CYST WALL1.Congenital cyst2.Posttraumatic cyst3.Echinococcal cyst4.Cystic dermoid5.Epidermoid *mnemonic:*"HITCH"**H**istoplasmosis (most common) **I**nfarct (sickle cell disease) **T**uberculosis **C**yst (Echinococcus) **H**ematoma

Notes:

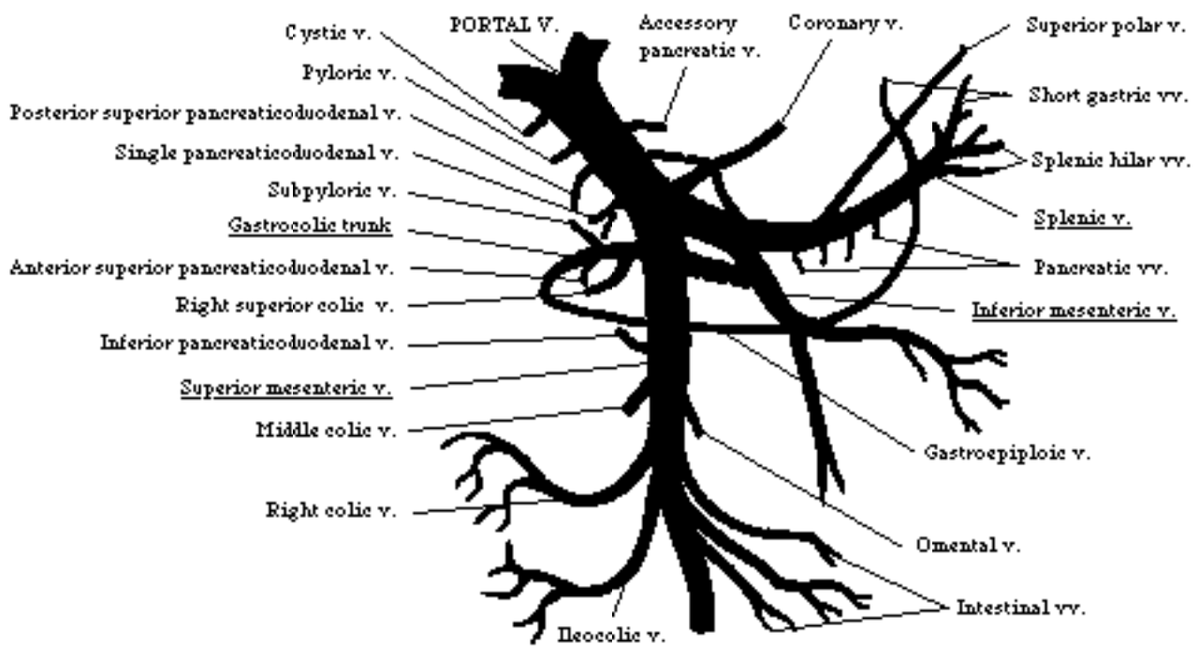
Iron Accumulation In Spleen A.DIFFUSE1.Multiple blood transfusions2.Sickle cell anemiaB.FOCAL1.Gamna Gandy bodies2.Angiosarcoma

Notes:

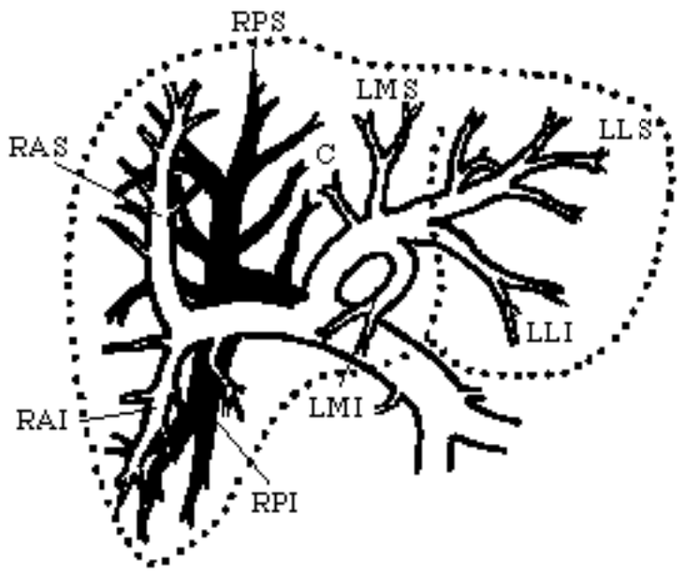
Hyperechoic Splenic Spots 1.Granulomas: miliary tuberculosis, histoplasmosis2.Phleboliths3.Lymphoma / leukemia4.Myelofibrosis5.Gamna-Gandy nodules (in portal hypertension)

Notes:

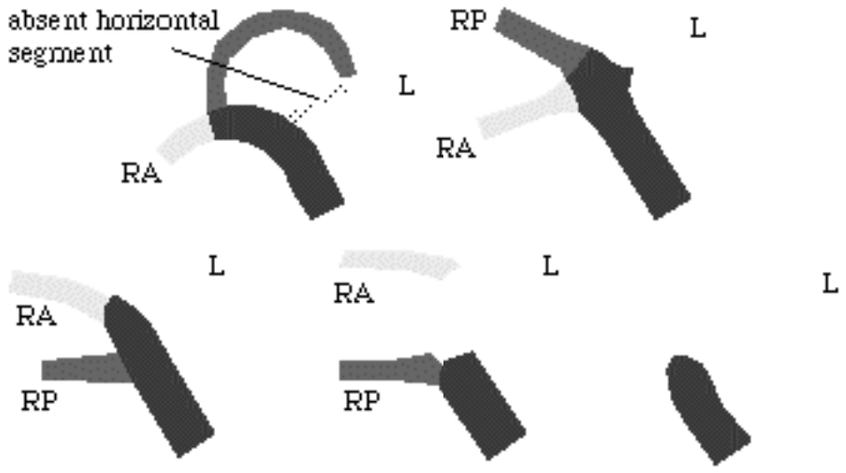
Portal Venous Anatomy



Extrahepatic Portal Vein Tributaries



Intrahepatic Portal Vein Branches



Variations of Intrahepatic Portal Venous System (20%)

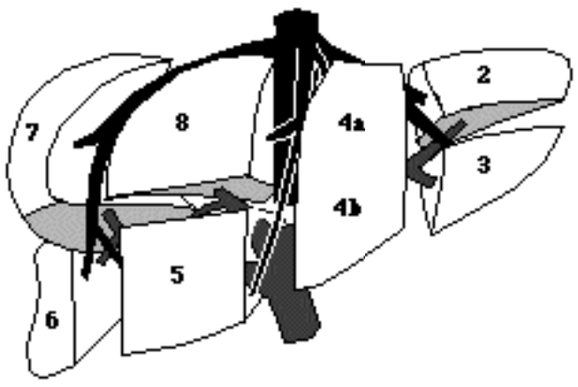
A. LEFT PORTAL VEIN
   1. Absence of horizontal segment (0.2%)

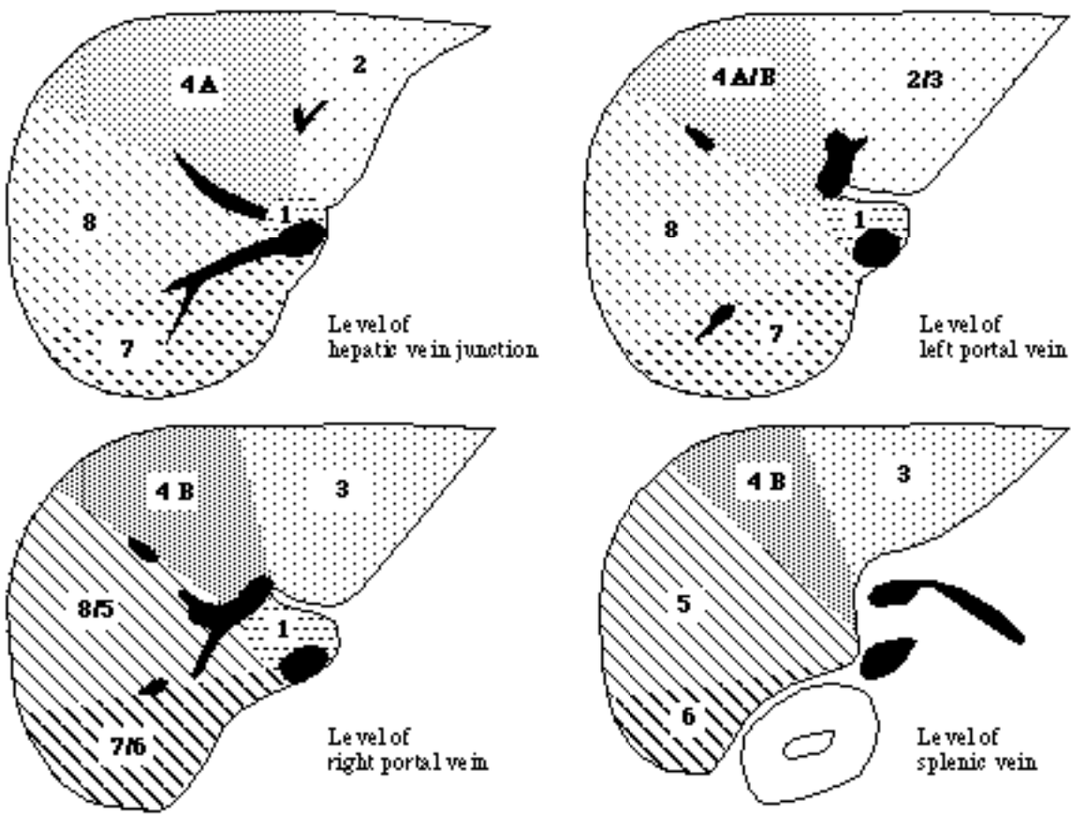
B. RIGHT PORTAL VEIN
   1. Trifurcation of main portal vein (11%)
   2. Origin of RP segment from main portal vein (5%)
   3. Origin of RA segment from left portal vein (4%)
   4. Absence of main right, RA, and RP portal segments

RA	=	right anterior segment	RPI	=	right posterior inferior	LMI	=	left median inferior
RAI	=	right anterior inferior	RPS	=	right posterior superior	LMS	=	left median superior
RAS	=	right anterior superior	C	=	caudate lobe	LLI	=	left lateral inferior
RP	=	right posterior segment	L	=	left portal vein	LLS	=	left lateral superior

Notes:

Functional Segmental Liver Anatomy based on distribution of 3 major hepatic veins: (a)middle hepatic veindivides liver into right and left lobe also separated by main portal vein scissura (Cantlie line) passing through IVC + long axis of gallbladder) (b)left hepatic veindivides left lobe into medial + lateral sectors (c)right hepatic veindivides right lobe into medial + lateral sectors Each of the four sections is further divided by an imaginary transverse line drawn through the right + left portal vein into anterior + posterior segments; the segments are numbered counterclockwise from IVC

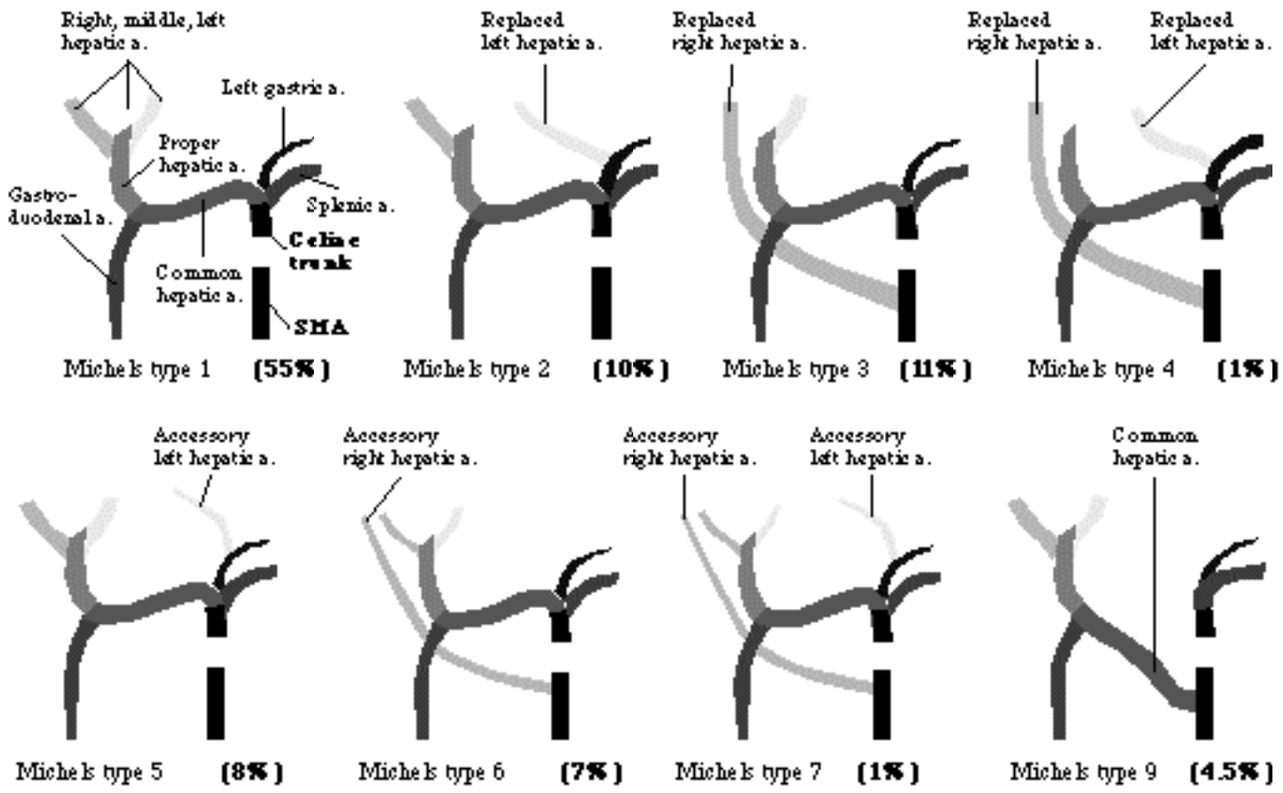




Functional Segmental Liver Anatomy			
(Goldsmith & Woodburne)		(Couinaud & Bismuth)	
CAUDATE LOBE			1
LEFT LOBE	Left lateral segment	Left lateral superior subsegment	2
		Left lateral inferior subsegment	3
	Left medial segment	Left medial superior subsegment	4 a
		Left medial inferior subsegment	4 b
RIGHT LOBE	Right anterior segment	Right anterior inferior subsegment	5
		Right anterior superior subsegment	8
	Right posterior segment	Right posterior inferior subsegment	6
		Right posterior superior subsegment	7

Notes:

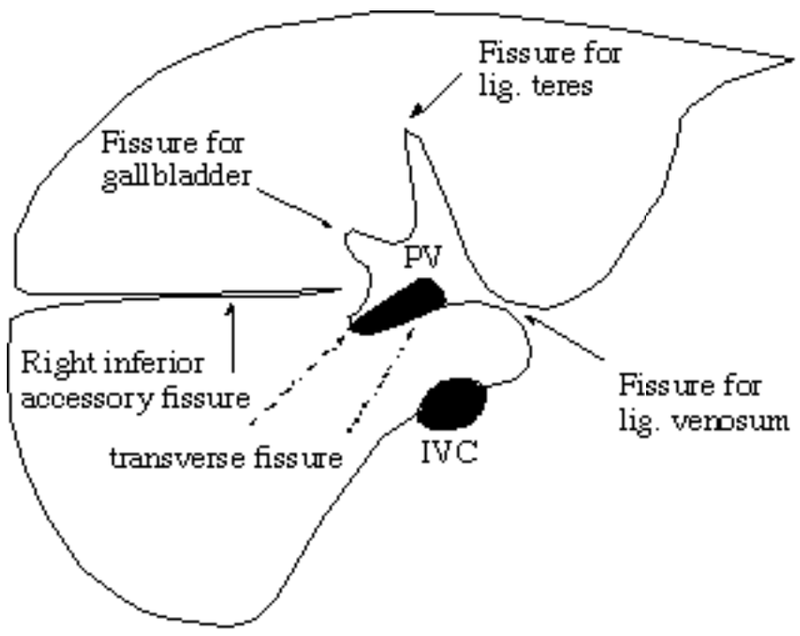
Hepatic Arterial Anatomy (Michels classification)



Type I (55%): -celiac trunk trifurcates into LT gastric a. + splenic a. + common hep. a.-common hep. a. divides into gastroduodenal a. + proper hep. a.-RT hep. a. + LT hep. a. arise from proper hep. a.-middle hep. a. (supplying caudate lobe) arises from(a)LT / RT hep. a.(b)proper hep. a. (in 10%)Type II (10%): -common hep. a. divides into gastroduodenal + RT hep. a.-LT hep. a. replaced to LT gastric a.-middle hep. a. from RT hep. a.Type III (11%): -common hep. a. divides into gastroduodenal + LT hep. a.-RT hep. a. replaced to superior mesenteric a.-middle hep. a. from LT hep. a.Type IV (1%): -common hep. a. divides into middle hep. a. + gastroduodenal a.-RT hep. a. + LT hep. a. are both replacedType V (8%): -accessory LT hep. a. arises from LT gastric a.Type VI (7%): -accessory RT hep. a. arises from superior mesenteric a.Type VII (1%): -accessory RT + LT hepatic a.Type VIII (2%): -combinations of accessory + replaced hepatic aa.Type IX (4.5%): -hepatic trunk replaced to superior mesenteric a.Type X (0.5%): -hepatic trunk replaced to LT gastric a.

Notes:

Hepatic Fissures



1.Fissure for ligamentum teres = umbilical fissure=invagination of ligamentum teres = embryologic remnant of obliterated umbilical vein connecting placental venous blood with left portal vein-located at dorsal free margin of falciform ligament-runs into liver with visceral peritoneum-divides left hepatic lobe into medial + lateral segments (divides subsegment 3 from 4)2.Fissure for ligamentum venosum=invagination of obliterated ductus venosus=embryologic connection of left portal vein with left hepatic vein-separates caudate lobe from left lobe of liver-lesser omentum within fissure separates the greater sac anteriorly from lesser sac posteriorly3.Fissure for gallbladder (GB)=shallow peritoneal invagination containing the GB-divides right from left lobe of liver4.Transverse fissure=invagination of hepatic pedicle into liver-contains horizontal portion of left + right portal veins5.Accessory fissures(a)Right inferior accessory fissure=from gallbladder fossa / just inferior to it to lateroinferior margin of liver(b)others (rare)

Notes:

Size Of Liver Sonographic measurements along vertical (craniocaudad) axis: (a)midclavicular line<13 cm=normal13.0-15.5 cm=indeterminate (in 25% of patients)>15.5 cm=hepatomegaly (87% accuracy)(b)preaortic line >10 cm(c)prerenal line >14 cm

Notes:

Normal Hemodynamics Parameter Of Liver Portal vein velocity:>11 cm/secCongestion index (= cross-sectional area of portal vein divided by average velocity): 0.070 ± 0.09Hepatic artery resistive index: 0.60-0.64 ± 0.06

Notes:

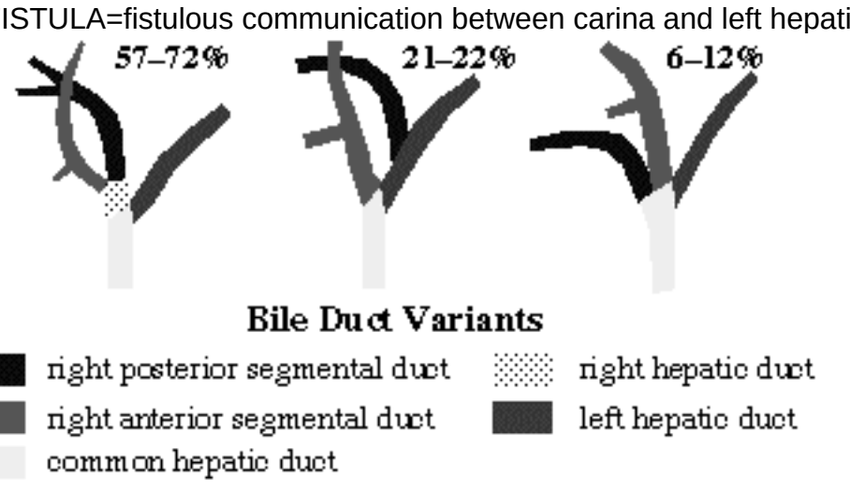
Liver Tests A.Alkaline phosphatase (AP)*Formation:*bone, liver, intestine, placenta*high increase:*cholestasis with extrahepatic biliary obstruction (confirmed by rise in gGT), drugs, granulomatous disease (sarcoidosis), primary biliary cirrhosis, primary + secondary malignancy of liver*mild increase:*all forms of liver disease, heart failureB.g-glutamyl transpeptidase (GGT)very sensitive in almost all forms of liver disease *Utility:*confirms hepatic source of elevated AP, may indicate significant alcohol useC.Transaminases*high increase:*viral / toxin-induced acute hepatitis(a)aspartate aminotransferase (AST; formerly serum glutamic oxaloacetic transaminase [SGOT])*Formation:*liver, muscle, kidney, pancreas, RBCs(b)alanine aminotransferase (ALT; formerly serum glutamic pyruvic transaminase [SGPT])*Formation:*primarily in liver ● rather specific elevation in liver diseaseD.Bilirubinhelps differentiate between various causes of jaundice (a)unconjugated / indirect bilirubin = insoluble in water*Formation:*breakdown of senescent RBCs*Metabolism:*tightly bound to albumin in vessels, actively taken up by liver, cannot be excreted by kidneys(b)conjugated / direct bilirubin = water-soluble*Formation:*conjugation in liver cells*Metabolism:*excretion into bile; not reabsorbed by intestinal mucosa + excreted in feces*Elevation:* -overproduction: hemolytic anemia, resorption of hematoma, multiple transfusions-decreased hepatic uptake: drugs, sepsis-decreased conjugation: Gilbert syndrome, neonatal jaundice, hepatitis, cirrhosis, sepsis-decreased excretion into bile: hepatitis, cirrhosis, drug-induced cholestasis, sepsis, extrahepatic biliary obstructionE.Lactic dehydrogenase (LDH)nonspecific and therefore not helpful *high increase:*primary or metastatic liver involvementF.Alpha fetoprotein (AFP)>400 ng/mLstrongly suggests that focal mass represents a hepatocellular carcinoma

Notes:

Normal Size Of Bile Ducts @CBD at point of maximum diameter:≤5 mm = normal; 6-7 mm = equivocal; ≥8 mm = dilated @CHD at porta hepatis + CBD in head of pancreas:5 mm @right intrahepatic duct just proximal to CHD:2-3mm @Cystic duct diameter:1.8 mmaverage length of 1-2 cm distal cystic duct posterior to CBD (in 95%), anterior to CBD (in 5%)

Notes:

Bile Duct Variants *Incidence:*2.4% of autopsies;13% of operative cholangiograms A.ABERRANT INTRAHEPATIC DUCTmay join CHD, CBD, cystic duct, right hepatic duct, gallbladder -anomalous right hepatic duct entering CHD / cystic duct (4-5%)*Cx:*(1)postoperative bile leak if severed(2)segmental biliary obstruction if ligatedB.CYSTIC DUCT ENTERING RIGHT HEPATIC DUCTC.DUCTS OF LUSCHKA=small ducts from hepatic bed draining directly into gallbladderD.DUPLICATION OF CYSTIC DUCT / CBDE.CONGENITAL TRACHEOBILIARY FISTULA=fistulous communication between carina and left hepatic duct ● infants with respiratory distress ● productive cough with bilious sputum√ pneumobilia



Bile Duct Variants

Notes:

Radiology Review Manual
fourth edition

Pancreaticobiliary Junction Variants A.Angle between CBD + pancreatic duct:(a)usually acute at 5°-30°(b)occasionally abnormal at up to 90°B.Sphincter of Oddi= muscle fibers encircling the CBD + pancreatic duct at choledochoduodenal junction (a)choledochal sphincter = encircles distal CBD(b)pancreatic duct sphincter (in 33% separate)C.Types of union between CBD + pancreatic duct:(a)2-10 (mean 5) mm short common channel (85%) with a diameter of 3-5 mm(b)separate entrances into duodenum(c)8-15 mm long common channel(d)pancreatic duct inserting into CBD >15 mm from entrance into duodenum(e)CBD inserting into pancreatic duct

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Notes:

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Agenesis Of Gallbladder *Incidence:*0.04 - 0.07 % (autopsy)*Associated with:* common: rectovaginal fistula, imperforate anus, hypoplasia of scapula + radius, intracardiac shunt rare: absence of corpus callosum, microcephaly, atresia of external auditory canal, tricuspid atresia, TE fistula, dextroposition of pancreas + esophagus, absent spleen, high position of cecum, polycystic kidney

Notes:

Hypoplastic Gallbladder (a)congenital(b)associated with cystic fibrosis

Notes:

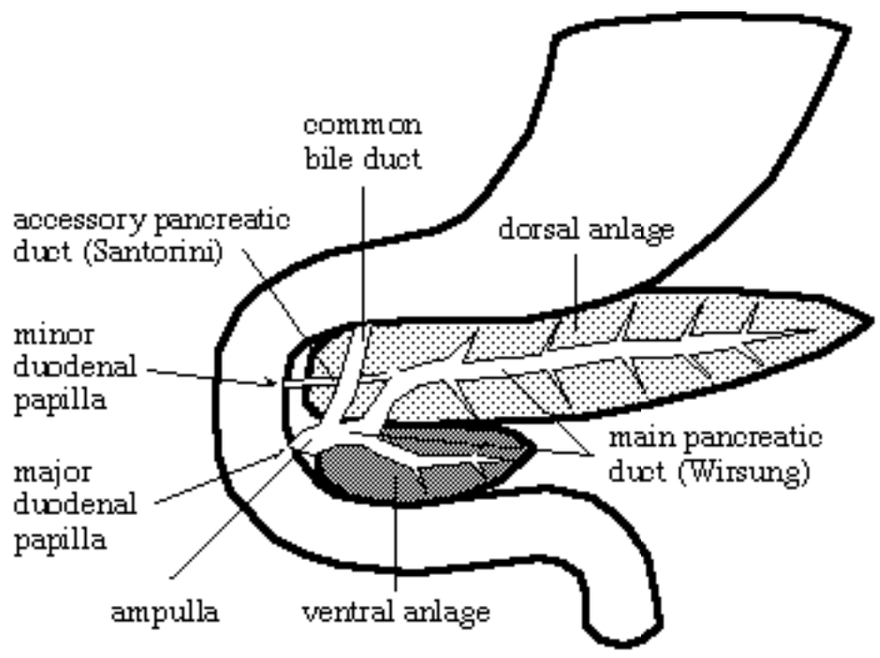
Septations Of Gallbladder A.LONGITUDINAL SEPTA1.Duplication of gallbladder=two separate lumens + two cystic ducts*Incidence:*1:3,000 to 1:12,0002.Bifid gallbladder = double gallbladder=two separate lumens with one cystic duct3.Triple gallbladder (extremely rare)B.TRANSVERSE SEPTA1.Isolated transverse septum2.PHRYGIAN CAP (2-6% of population)=kinking / folding of fundus ± septum3.Multiseptated gallbladder (rare)=multiple cystlike compartments connected by small pores*Cx:*stasis + stone formationC.GALLBLADDER DIVERTICULUM=persistence of cystohepatic duct

Notes:

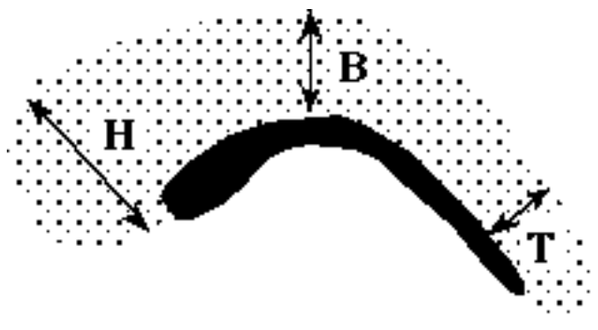
Gallbladder Ectopia Most frequent locations: (1) beneath the left lobe of the liver > (2) intrahepatic > (3) retrohepatic Rare locations: (4) within falciform ligament (5) within interlobar fissure (6) suprahepatic (lodged between superior surface of right hepatic lobe + anterior chest wall) (7) within anterior abdominal wall (8) transverse mesocolon (9) retrorenal (10) near posterior spine + IVC (11) intrathoracic GB (inversion of liver) *Associated with:* eventration of diaphragm "Floating GB" =gallbladder with loose peritoneal reflections, may herniate through foramen of Winslow into lesser sac"Torqued GB" = results in hydrops

Notes:

Pancreatic Development & Anatomy



Anatomy of Pancreatic Ducts



Pancreatic Diameters (on TRV image)H = head = 1.5 - 3.0 cm B = body = 1.2 - 2.5 cm C = tail = 1.0 - 2.5 cm

A.DORSAL ANLAGE (in mesoduodenum)*Origin:*arises from dorsal wall of duodenum◊forms cranial portion of head + isthmus + body + tail of pancreas-prone to atrophy (poor in polypeptides)√ drains to the minor papilla through accessory duct of Santorini B.VENTRAL ANLAGE (below primordial liver bud)*Origin:*ventral bud arises from ventral wall of duodenum and is composed of right + left lobes (the left ventral bud regresses completely), migrates to opposite side of duodenum + fuses with dorsal anlage during 6th week GA◊forms caudal portion of the pancreatic head + uncinate process + CBD-not prone to atrophy (rich in polypeptides)√ the ventral duct of Wirsung drains with the CBD through ampulla of Vater and becomes the major drainage pathway for the entire pancreas after fusion with the duct of Santorini C.MAIN PANCREATIC DUCT OF WIRSUNGdistal portion of dorsal duct connects with ventral duct; proximal portion of dorsal duct may disappear D.ACCESSORY PANCREATIC DUCT OF SANTORINI=proximal portion of dorsal duct which has not atrophied E.AMPULLA OF VATER=space within medial wall of second portion of duodenum below surface of papilla of Vater F.MAJOR DUODENAL PAPILLA = papilla of Vater◊drainage of common bile duct in 100%◊drainage of main pancreatic duct of Wirsung in 90% G.MINOR DUODENAL PAPILLA (present in 60%)◊drainage of accessory pancreatic duct of Santorini◊drainage of main pancreatic duct in 10% √ located a few cm orad to papilla of Vater

Notes:

SPLEEN

A.NORMAL SIZEin adults:12 cm length, 7-8 cm anteroposterior diameter, 3-4 cm thick; splenic index (LxWxH) of <480 in children:formula for length = 5.7 + 0.31 x age (in years)B.NORMAL WEIGHT150 (100-265) gestimated weight = splenic index x 0.55 C.CT ATTENUATION(a)without enhancement:40-60 HU; 5-10 HU less than liver (b)with enhancement:normal heterogeneous enhancement during parenchymal phase after bolus injection (due to varying blood flow rates through the cords of the red pulp) D.MR SIGNAL INTENSITY(a)on T1WI:liver > spleen > muscle(b)on T2WI:spleen > liver

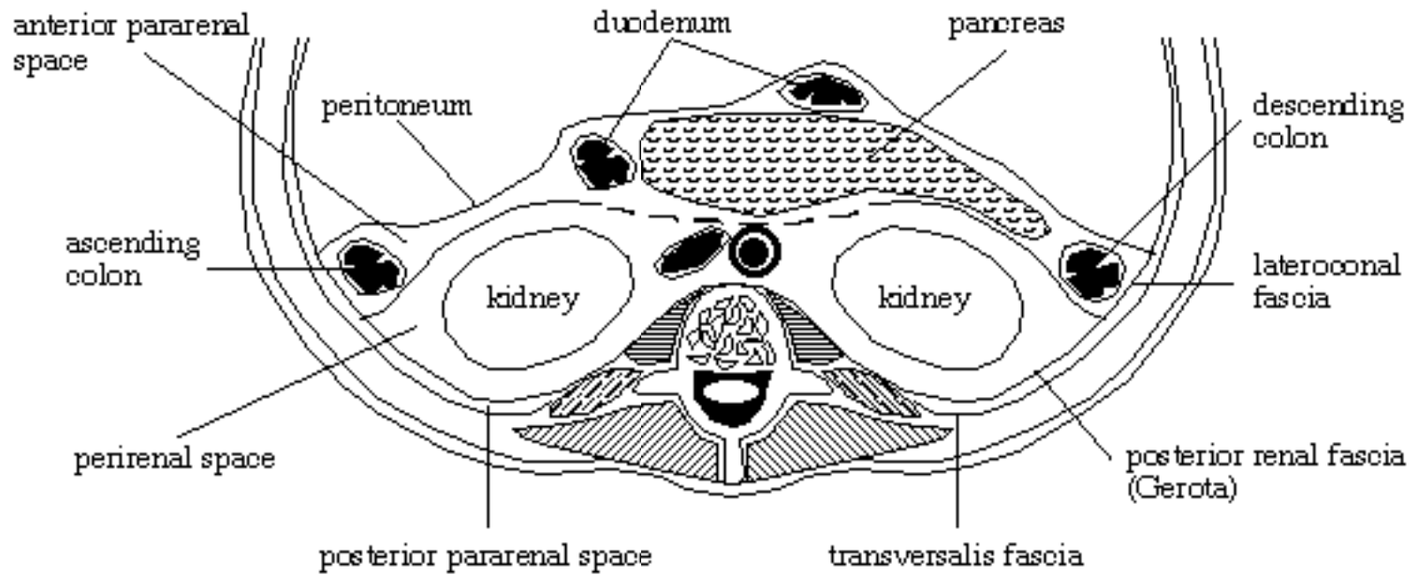
Notes:

IRON METABOLISM

Total body iron: 5 g (a)functional iron:4 gLocation:hemoglobin of RBCs, myoglobin of muscle, various enzymes(b)stored iron:1 gLocation:hepatocytes, reticuloendothelial cells of liver (Kupffer cells) + spleen + bone marrowAbsorption:1-2 mg/day through gutTransport:bound to transferrin intravascularly Deposition: (a)transferrin-transfer to:hepatocytes, RBC precursors in erythron, parenchymal tissues (eg, muscle) (b)phagocytosis by:reticuloendothelial cells phagocytize senescent erythrocytes (= extravascular hemolysis); RBC iron stored as ferritin / released and bound to transferrin

Notes:

EXTRAPERITONEAL SPACES



Extraperitoneal Spaces

Notes:

ACCESSORY SPLEEN
=failure of coalescence of several small mesodermal buds in the dorsal mesogastrium which comprise the spleen*Incidence:*10-30% of population; multiple in 10%◊ undergoes hypertrophy after splenectomy and is responsible for recurrence of hematologic disorders (idiopathic thrombocytopenic purpura, hereditary spherocytosis, acquired autoimmune hemolytic anemia, hypersplenism)Location:splenic hilum (most common), gastrosplenic ligament, other suspensory ligaments of spleen, rare in pancreas / pelvisNUC (Tc-99m sulfur colloid scan / spleen-specific Tc-99m denatured RBCs): √ usually <1 cm in diameter√ <10% identified when normal spleen present

Notes:

AMPULLARY TUMOR

=benign / malignant tumors arising from glandular epithelium of ampulla of Vater*Age:*6th + 7th decade; M:F = 2:1*Path:*average diameter of <3 cm*Histo:*(a)dysplastic epithelium in glandular / villous structures of tubular / villous adenoma(b)carcinoma in situ(c)invasive carcinoma often with desmoplastic reaction*Associated with:*familial adenomatous polyposis syndromes (eg, familial polyposis coli, Gardner syndrome) [100-200-fold risk], colon carcinoma ● malaise, epigastric pain, weight loss ● intestinal bleeding (tumor ulceration) ● intermittent jaundice (ductal obstruction) ● gray "aluminum / silver colored" stools (3%) ● chills, fever, RUQ pain (ascending cholangitis) in up to 20% ● endoscopy: tumor extending through orifice (63%), prominent papilla / submucosal mass (25%), not visualized (9%) TNM staging: T1:tumor confined to ampullaT2:tumor extending into duodenal wallT3:invasion of pancreas <2 cm deepT4:invasion of pancreas >2 cm deep International Union against Cancer staging: I=tumor confined to ampullaII=tumor extension into duodenal wall / pancreasIII=regional lymph node involvement (Lnn stations around head + body of pancreas, anterior + posterior pancreaticoduodenal, pyloric, common bile duct, proximal mesenteric)IV=invasion of pancreas >2 cm deep √ tumor often inapparent due to small sizeUGI: √ indentation of duodenal lumen at papilla of Vater with filling defect >1.5 cm√ surface irregularity + deep barium-filled crevices in villous tumorBiliary imaging: √ dilatation of most distal segment of common bile duct√ stenosis (circumferential tumor growth around ampulla / desmoplastic reaction)√ irregular predominantly polypoid filling defect√ ± pancreatic dilatation = double-duct sign (may be absent if tumor small / accessory pancreatic duct decompresses pancreatic system / main pancreatic duct drains into minor papilla)Endoscopic US (most sensitive technique): 87% staging accuracy *Rx:*Whipple procedure (= pancreaticoduodenectomy)*Prognosis:*28-70% 5-year survival for ampullary carcinomas (depending on stage)*DDx:* 1.Periampullary duodenal adenoma / adenocarcinoma (usually larger lesion with significant intraduodenal extension)2.Choledochocele (cystic lesion filling with biliary contrast)3.Brunner gland tumor, pancreatic rest ("myoepithelial hamartoma"), leiomyoma, carcinoid (often produce somatostatin)4.Duodenitis, pancreatitis5.Stone impaction in ampulla

Notes:

ANNULAR PANCREAS
=uncommon congenital anomaly wherein a ring of normal pancreatic tissue encircles the duodenum secondary to abnormal migration of ventral pancreas (head + uncinate); most common congenital anomaly of pancreas*Age at discovery:*childhood (50%); adulthood (50%)*Associated with:* other congenital anomalies (in 75%): esophageal atresia, TE fistula, duodenal atresia / stenosis, duodenal diaphragm, imperforate anus, malrotation, Down syndromeLocation:2nd portion of duodenum (85%);1st / 3rd portion of duodenum (15%) ● mostly asymptomatic with incidental discovery ● neonate:persistent vomiting (duodenal obstruction) ● adult:nausea, vomiting (60%), abdominal pain (70%), hematemesis (10%), jaundice (50%)√ polyhydramnios (in utero)√ "double bubble" = dilated duodenal bulb + stomach√ enlargement of pancreatic headUGI: √ eccentric narrowing with lateral notching + medial retraction of 2nd part of duodenum√ concentric narrowing of mid-descending duodenum√ reverse peristalsis, pyloric incompetencyERCP (most specific): √ normally located main duct in pancreatic body + tail√ small duct originating on anterior left + passing posteriorly around duodenum communicates with main duct (in 85%)*Cx:*increased incidence of(1)periampullary peptic ulcers (2)pancreatitis (15-20%) usually confined to pancreatic head and annulus*Rx:*gastrojejunostomy / duodenojejunostomy

Notes:

ASCARIASIS
Most frequent helminthic infection in humans *Organism:*Ascaris lumbricoides, 25-35 cm long as adult worm; life span of 1 year*Country:*644 million humans harbor the roundworm; 70- 90% in America; in United States endemic in: Appalachian range, southern + Gulf coast states*Prevalence:*25% of world population infected(a)in United States: 12% in blacks, 1% in whites(b)in parts of Africa, Asia, South America: 90%*Cycle:* ingestion of contaminated water / soil / vegetable; larvae penetrate intestinal wall; migrate into mesenteric lymphatics + veins into liver; reach lung via right heart + pulmonary artery; mature in pulmonary capillary bed to 2-3 mm length; burrow into alveoli; ascend in respiratory tract; are swallowed and again reach small intestine, where they become adult worms whose eggs leave the body by the fecal route
● abnormal liver function tests + biliary colic ● hypereosinophilia only present during acute stage of larval migration√ barium study√ cholangiography (49%)US: √ tubular echogenic filling defect with 2-4 mm wide central sonolucent line (= worm with digestive tract) within dilated common bile duct*Cx:*(1)Intestinal obstruction(2)Intermittent biliary obstruction with acute cholangitis, cholecystitis, pancreatitis(3)Liver abscess (rare)(4)Granulomatous stricture of extrahepatic bile ducts (rare)*Rx:*Mebendazole

Notes:

BANTI SYNDROME
=NONCIRRHOTIC IDIOPATHIC PORTAL HYPERTENSION = NONCIRRHOTIC PORTAL FIBROSIS = HEPATOPORTAL SCLEROSIS=syndrome characterized by (1) splenomegaly(2) hypersplenism (3) portal hypertension *Etiology:*increased portal vascular resistance possibly due to portal fibrosis + obliterative venopathy of intrahepatic portal branches*Histo:*slight portal fibrosis, dilatation of sinusoids, intimal thickening with eccentric sclerosis of peripheral portal vein walls*Age:*middle-aged women; rare in America + Europe but common in India + Japan ● elevated portal vein pressure (without cirrhosis, parasites, venous occlusion) ● normal liver function tests ● cytopenia (due to hypersplenism) ● normal / slightly elevated hepatic venous wedge pressure√ esophageal varices√ patent hepatic veins√ patent extrahepatic portal vein + multiple collaterals*Prognosis:*90% 5-year survival; 55% 30-year survival

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Notes:

BILIARY CYSTADENOCARCINOMA
=BILE DUCT CYSTADENOCARCINOMA=rare malignant multilocular cystic tumor originating from biliary cystadenoma*Histo:*(a)with ovarian stroma (good prognosis), in females only(b)without ovarian stroma (bad prognosis) ● hemorrhagic internal fluid√ nodularity with septations are suggestive of malignancy√ coarse calcifications*DDx:*no image differentiation from biliary cystadenoma

Notes:

BILIARY CYSTADENOMA

=BILE DUCT CYSTADENOMA=rare benign premalignant multilocular cystic tumor originating in bile ducts; probably deriving from ectopic nests of primitive biliary tissue*Incidence:*4.6% of all intrahepatic cysts of bile duct origin*Age:*>30 years (82%), peak incidence in 5th decade; M:F = 1:4; predominantly Caucasian*Path:*multiloculated cystic tumor with well-defined thick capsule containing proteinaceous fluid*Histo:*single layer of cuboidal / tall columnar biliary-type epithelium with papillary projections, subepithelial stroma resembling that of the ovary√Similar to mucinous cystic tumors of pancreas + ovaryLocation:intrahepatic bile ducts (85%); extrahepatic bile ducts (15%); right lobe (48%); left lobe (20-35%); both lobes (15-30%); gallbladder (rare) ● abdominal swelling with palpable mass (90%) ● dyspepsia, anorexia, nausea + vomiting ● jaundice√ mass of 1.5-35 cm in size√ up to 11 liters of clear / cloudy, serous / mucinous / gelatinous, purulent / hemorrhagic / bilious fluid containing hemosiderin / cholesterol / necrosis√ papillary excrescences + mural nodules√ septations between cystsUS: √ ovoid multiloculated anechoic mass with highly echogenic septations / papillary growths√ may contain fluid-fluid levelsCT: √ multiloculated mass of near water density√ contrast enhancement in wall + internal septa MR: √ locules with variable signal intensity on T1WI + T2WI depending on their protein contentAngio: √ avascular mass with small clusters of peripheral abnormal vessels√ stretching + displacement of vessels√ thin subtle blush of neovascularity in septa + wall*Cx:*malignant transformation into cystadenocarcinoma (indicated by invasion of capsule); rupture into peritoneum / retroperitoneum*Rx:*surgical resection (recurrence common)*DDx:*liver abscess, echinococcal cyst, cystic mesenchymal hamartoma (children + young adults), undifferentiated sarcoma (children + young adults), necrotic hepatic metastasis, cystic primary hepatocellular carcinoma

Notes:

BILIARY-ENTERIC FISTULA

*Incidence:*5% at cholecystectomy; 0.5% at autopsy*Etiology:*cholelithiasis (90%), acute / chronic cholecystitis, biliary tract carcinoma, regional invasive neoplasm, diverticulitis, inflammatory bowel disease, peptic ulcer disease, echinococcal cyst, trauma, congenital communicationCommunication with: duodenum (70%), colon (26%), stomach (4%), jejunum, ileum, hepatic artery, portal vein (caused death of Ignatius Loyola), bronchial tree, pericardium, renal pelvis, ureter, urinary bladder, vagina, ovary A.CHOLECYSTODUODENAL FISTULA (51-70%)1.Perforated gallstone (90%):associated with gallstone ileus in 20%2.Perforated duodenal ulcer (10%)3.Surgical anastomosis4.Gallbladder carcinomaB.CHOLECYSTOCOLIC FISTULA (13-21%)C.CHOLEDOCHODUODENAL FISTULA (13-19%)due to perforated duodenal ulcer diseaseD.MULTIPLE FISTULAE (7%)√ branching tubular radiolucencies, more prominent centrally√ barium filling of biliary tree√ multiple hyperechoic foci with dirty shadowing*DDx:*patulous sphincter of Oddi, ascending cholangitis, surgery (choledochoduodenostomy, cholecystojejunostomy, sphincterotomy)

Notes:

BUDD-CHIARI SYNDROME

=HEPATIC VENO-OCCLUSIVE DISEASE=global / segmental obstruction of hepatic venous outflow *Cause:* A.IDIOPATHIC (66%)B.THROMBOSIS(a)Hypercoagulable state: polycythemia rubra vera (1/3), oral contraceptives, pregnancy + postpartum state, paroxysmal nocturnal hemoglobulinuria (successive thrombosis of small veins), sickle cell disease*mnemonic:*"5 Ps"**P**aroxysmal nocturnal hemoglobulinuria **P**latelets (thrombocytosis) **P**ill (birth control pills) **P**regnancy **P**olycythemia rubra vera (b)Injury to vessel wall: phlebitis, trauma, hepatic radiation injury, chemotherapeutic + immunosuppressive drugs in patients with bone marrow transplants, venoocclusive disease from pyrrolizidine alkaloids (senecio) found in medicinal bush teas in JamaicaC.NONTHROMBOTIC OBSTRUCTION(a)Tumor growth into IVC / hepatic veins (renal cell carcinoma, hepatoma, adrenal carcinoma, metastasis, primary leiomyosarcoma of IVC)(b)Membranous obstruction of suprahepatic IVC= IVC diaphragm (believed to be a congenital web or an acquired lesion from long-standing IVC thrombosis); common cause in Oriental + Indian population (South Africa, India, Japan, Korea); very rare in Western countries (c) Right atrial tumor(d)Constrictive pericarditis(e)Right heart failure *Pathophysiology:*hepatic venous thrombosis leads to elevation of sinusoidal pressure which causes delayed / reversed portal venous inflow, ascites, alteration in hepatic morphologyM < F Location: Type I:occlusion of IVC ± hepatic veinsTypeII:occlusion of major hepatic veins ± IVCTypeIII:occlusion of small centrilobar veins√ hepatosplenomegaly (early sign)√ hypertrophy of caudate lobe (88%) [DDx: cirrhosis]√ ascites√ gallbladder wall thickening >6 mm√ nonvisualization of hepatic veins (75%) / vein diameter <3 mm (measured 2 cm from IVC)√ communications between right / middle hepatic vein and inferior right hepatic vein√ enlarged inferior right hepatic vein (18%)√ portal vein diameter >12 mm (in adults), >8 mm (in children)√ visualization of paraumbilical vein√ hypodensity in atrophic areas / periphery (82%) with inversion of portal blood flow√ patchy enhancement (85%) with normal portal blood flow√ ± narrowing / obstruction of intrahepatic IVCCT: √ enhancement of enlarged caudate lobe√ hypodense nonenhancing peripheral zones of liver (= reversed portal venous blood flow due to increased postsinusoidal pressure produced by hepatic venous obstruction)√ failure to identify hepatic veins√ hepatic vein thrombi (18-53%)MRI: √ reduction in caliber / complete absence of hepatic veins√ multiple comma-shaped intrahepatic flow voids(= intrahepatic collaterals)US: √ hepatic veins not visualized / reduced in size / filled with thrombus√ communicating collateral vessels√ reversed flow in hepatic veins√ absent / sluggish blood flow within IVCDoppler: -hepatic veins:√ absent / reversed / flat flow / loss of cardiac modulation in hepatic veins√ reversed flow in IVC-portal vein:√ flow demodulation = disappearance of portal vein velocity variations with breathing√ slow flow (<11 cm/sec) / hepatofugal flow in portal vein√ congestion index >0.1√ portal vein thrombosis (20%)-hepatic artery:√ resistive index >0.75NUC (Tc-99m sulfur colloid): √ central region of normal activity (hot caudate lobe) surrounded by greatly diminished activity (venous drainage of hypertrophied caudate lobe into IVC by separate vein)√ colloid shift to spleen + bone marrow√ wedge-shaped focal peripheral defectsAngio (inferior venocavography, hepatic venography): √ absence of main hepatic veins√ spider weblike appearance of collaterals + small hepatic veins√ stretching + draping of intrahepatic arteries with hepatomegaly√ inhomogeneous prolonged intense hepatogram with fine mottling√ large lakes of sinusoidal contrast accumulationPortography: √ central hepatic enhancement (normal hepatopetal flow)√ reversed portal flow in liver periphery (supplied only by hepatic artery)√ bidirectional / hepatofugal main portal vein flow

Acute Budd-Chiari Syndrome (1/3) Chronic Budd-Chiari Syndrome (2/3)

Notes:

Acute Budd-Chiari Syndrome (1/3)

◊Caudate lobe has not had time to hypertrophy! ● rapid onset of abdominal pain (liver congestion) ● insidious onset of intractable ascites√ hepatomegaly without derangement of liver function√ ascites (97%)CT: √ diffuse hypodensity on NECT√ early enhancement of caudate lobe + central portion around IVC with decreased enhancement peripherally√ hypodense lumina of hepatic veins on CECT√ decreased attenuation of enhancing areas with patchy inhomogeneous enhancement in liver periphery on delayed scans

Notes:

Chronic Budd-Chiari Syndrome (2/3)

● insidious onset of jaundice, intractable ascites ● portal hypertension, variceal bleeding√ enlargement of central region (= caudate lobe + adjacent central part of right lobe + medial segment of left lobe√ nonsegmental / lobar atrophy of affected liver (due to extensive fibrosis) with diminished attenuation before + after contrast administration√ progressive patchy enhancement radiating outward from major portal vessels (on dynamic bolus CT)√ "reticulated mosaic" enhancement = diffuse patchy lobular enhancement separated by irregular linear areas of low density in central area√ delayed homogeneous enhancement of entire liver after several minutesColor Doppler: √ "bicolored" hepatic veins (due to intrahepatic collateral pathways) are PATHOGNOMONIC *Dx:*liver biopsy*Rx:*anticoagulants, surgery / balloon dilatation (depending on etiology); portosystemic shunt; liver transplantation (for advanced cases)

Notes:

CANDIDIASIS OF LIVER
=almost exclusively seen in immunocompromised patients (leukemia, chronic granulomatous disease of childhood, renal transplant, chemotherapy for myeloproliferative disorders)◊Most common systemic fungal infection in immunocompromised patients! ● abdominal pain ● persistent fever in neutropenic patient whose leukocyte count is returning to normal ● elevated alkaline phosphatase√ hepatomegaly√ "target" / "bulls-eye" sign = multiple small hypoechoic / hypoattenuating masses with centers of increased echogenicity / attenuation◊Bulls-eye lesion becomes visible only when neutropenia resolves!NUC: √ uniform uptake / focal photopenic areas√ diminished Ga-67 uptake*Dx:*biopsy evidence of yeast / pseudohyphae in central necrotic portion of lesion*DDx:*metastases, lymphoma, leukemia, sarcoidosis, septic emboli, other infections, Kaposi sarcoma

Notes:

CAROLI DISEASE
=COMMUNICATING CAVERNOUS ECTASIA OF INTRAHEPATIC DUCTS=rare probably autosomal recessive disorder characterized by congenital segmental saccular cystic dilatation of major intrahepatic bile ducts*Etiology:*(a)? perinatal hepatic artery occlusion(b)? hypoplasia / aplasia of fibromuscular wall components*Age:*childhood + 2nd-3rd decade, occasionally in infancy; M:F = 1:1*Associated with:* medullary sponge kidney (in 80%), infantile polycystic kidney disease, renal tubular ectasia, choledochal cyst (rare), congenital hepatic fibrosis ● recurrent cramplike upper abdominal pain ● NO cirrhosis / portal hypertension√ multiple cystic structures converging toward porta hepatis as either localized / diffusely scattered cysts communicating with bile ducts (DDx: polycystic liver disease)√ segmental saccular / beaded appearance of intrahepatic bile ducts extending to periphery of liver√ portal radicles completely surrounded by dilated bile ducts = central dot sign on CT√ bridge formation across dilated lumina√ intraluminal bulbar protrusions√ frequent ectasia of extrahepatic ducts + CBD√ sludge / calculi in dilated ducts*Cx:*(1) bile stasis with recurrent cholangitis (2) biliary calculi (3) liver abscess (4) septicemia(5) increased risk for cholangiocarcinoma

Notes:

Intrahepatic Cholangiocarcinoma =CHOLANGIOCELLULAR CARCINOMA*Incidence:*1/3 of all malignancies originating in the liver; 8-13% of all cholangiocarcinomas; 2nd most common primary hepatic tumor after hepatoma*Types:* (1)Massive / nodular type(2)Diffuse (sclerosing cholangitis) type◊Cannot be depicted by cross-sectional imaging!*Histo:*adenocarcinoma arising from the epithelium of a small intrahepatic bile duct with prominent desmoplastic reaction (fibrosis); ± mucin and calcifications*Average age:*50-60 years; M > F ● abdominal pain (47%) ● palpable mass (18%) ● weight loss (18%) ● painless jaundice (12%)Spread:(a)local extension along duct(b)local infiltration of liver substance(c)metastatic spread to regional lymph nodes (in 15%)√ mass of 5-20 cm in diameter√ satellite nodules in 65%√ punctate / chunky calcifications in 18%√ calculi in biliary treeNUC:√ cold lesion on sulfur colloid / IDA scans√ segmental biliary obstruction√ may show uptake on gallium scanUS:√ dilated biliary tree√ predominantly homo- / heterogeneous mass√ hyper- (75%) / iso- / hypoechoic (14%) massCT:√ single predominantly homogeneous round / oval hypodense mass with irregular borders√ "peripheral washout sign" = early minimal / moderate rim enhancement with progressive concentric filling and clearing of contrast material in rim of lesion on delayed images√ marked homogeneous delayed enhancement (74%)MR:√ large central heterogeneous hypointense mass on T1WI√ hyperintense periphery (viable tumor) + large central hypointensity (fibrosis) on T2WIAngiography:√ avascular / hypo- / hypervascular mass√ stretched / encased arteries (frequent)√ neovascularity in 50%√ lack of venous invasion*Prognosis:*<20% resectable; 30% 5-year survival

Notes:

Extrahepatic Cholangiocarcinoma =BILE DUCT CARCINOMA *Age peak:*6th-7th decade, M:F = 3:2*Incidence:*<0.5% of autopsies; 90% of all cholangiocarcinomas; more frequent in Far East*Histo:*well-differentiated sclerosing adenocarcinoma (2/3), anaplastic carcinoma (11%), cystadenocarcinoma, adenoacanthoma, malignant adenoma, squamous cell = epidermoid carcinoma, leiomyosarcoma*Predisposed:* (1)Inflammatory bowel disease (10 x increased risk); incidence of 0.4-1.4% in ulcerative colitis; latent period of 15 years; tumors usually multicentric + predominantly in extrahepatic sites; GB involved in 15% (simultaneous presence of gallstones is rare)(2)Sclerosing cholangitis (10%)(3)Caroli disease (due to chronic biliary stasis)(4)Clonorchis sinensis infestation (Far East); most common cause worldwide(5)Thorotrast exposure(6)History of other malignancy (10%)(7)Previous surgery for choledochal cyst / congenital biliary atresia(8)Alpha-1-antitrypsin deficiency(9)Autosomal dominant polycystic disease(10)Cholecystolithiasis (20-50%), probably coincidental(11)Papillomatosis of bile ducts ● gradual onset of fluctuating painless jaundice ● cholangitis (10%) ● weight loss, fatigability ● intermittent epigastric pain ● elevated bilirubin + alkaline phosphatase ● enlarged tender liver*Growth pattern:* (1)Obstructive type (70-85%)√ U- / V-shaped obstruction with nipple, rattail, smooth / irregular termination(2)Stenotic type (10-25%)√ strictured rigid lumen with irregular margins + prestenotic dilatation(3)Polypoid / papillary type (5-6%)√ intraluminal filling defect with irregular margins*Spread:*(a)lymphatic spread: cystic + CBD nodes (>32%), celiac nodes (>16%), peripancreatic nodes, superior mesenteric nodes(b)infiltration of liver (23%)(c)peritoneal seeding (9%)(d)hematogenous (extremely rare): liver, peritoneum, lung Location: left / right hepatic ductin8-13%confluence of hepatic ductsin10-26%(Klatskin tumor)common hepatic ductin14-37%proximal CBDin15-30%distal CBDin30-50%cystic ductin6% UGI: √ infiltration / indentation of stomach / duodenumCholangiography (PTC or ERC best modality to depict bile duct neoplasm): √ exophytic intraductal tumor mass (46%), 2-5 mm in diameter√ frequently long / rarely short concentric focal stricture in infiltrating sclerosing cholangitic type with wall irregularities√ prestenotic diffuse / focal biliary dilatation (100%)√ progression of ductal strictures (100%)US / CT: √ dilatation of intrahepatic ducts without extrahepatic duct dilatation√ failure to demonstrate the confluence of L + R hepatic ducts√ mass within / surrounding the ducts at point of obstruction (21% visible on US, 40% visible on CT)√ infiltrating tumor visible as highly attenuating lesion in 22% on CT, in 13% on US√ exophytic tumor visible in 100% on CT as low-attenuation mass, in 29% on US√ polypoid intraluminal tumor visible as isoechoic mass within surrounding bile in 100% on US, in 25% on CTAngiography: √ hypervascular tumor with neovascularity (50%)√ arterioarterial collaterals along the course of bile ducts associated with arterial obstruction√ poor / absent tumor stain√ displacement / encasement / occlusion of hepatic artery + portal vein*Cx:*(1)Obstruction leading to biliary cirrhosis(2)Hepatomegaly(3)Intrahepatic abscess (subdiaphragmatic, perihepatic, septicemia)(4)Biliary peritonitis(5)Portal vein invasion*Prognosis:*median survival of 5 months; 1.6% 5-year survival; 39% 5-year survival for carcinoma of papilla of Vater*DDx:*benign stricture, chronic pancreatitis, sclerosing cholangitis, edematous papilla, idiopathic inflammation of CBD

Notes:

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Acute Cholangitis *Cause:* (a)benign disease:(1) stricture from prior surgery (36%) (2) calculi (30%) (3) sclerosing cholangitis (4) obstructed drainage catheter (5) parasitic infestation (b)malignant disease: ampullary carcinoma*Types:* A.ACUTE NONSUPPURATIVE ASCENDING CHOLANGITIS ● bile remains clear ● patient nontoxicB.ACUTE SUPPURATIVE ASCENDING CHOLANGITIS (14%)*Associated with:* obstructing biliary stone or malignancy ● septicemia, CNS depression, lethargy, mental confusion, shock (50%)Ψ purulent material fills biliary ducts *Organism:*E. coli > Klebsiella > Pseudomonas > Enterococci ● recurrent episodes of sepsis + RUQ pain ● Charcot triad (70%): fever + chills + jaundice ● bile cultures in 90% positive for infection*Cx:*miliary hepatic abscess formation*Prognosis:*100% mortality if not decompressed;40-60% mortality with treatment;13-16% overall mortality rate

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Notes:

AIDS Cholangitis *Organism:*CMV, Cryptosporidium ● RUQ pain, fever, jaundice ● elevated WBC count ● abnormal LFT (esp. serum alkaline phosphatase)√ irregular mild dilatation of intra- and extrahepatic bile ducts similar to sclerosing cholangitis√ stricture of distal CBD / papillary stenosis√ mural thickening of gallbladder + bile ducts√ ± pericholecystic fluid

Notes:

Primary Sclerosing Cholangitis =insidious progressive inflammatory disease causing multifocal strictures of intra- and extrahepatic bile ducts*Etiology:*idiopathic, ? hypersensitivity reaction (speculative)*Prevalence:*1% as common as alcoholic liver disease*Age:*<45 years (2/3); range 21-39-67 years;M:F = 7:3*Histo:* Stage 1:degeneration of epithelial bile duct cells + infiltration with lymphocytes ± neutrophils; inflammation + scarring + enlargement of periportal triads (pericholangitis)Stage 2:fibrosis + inflammation infiltrating periportal parenchyma with piecemeal necrosis of hepatocytes; enlargement of portal triads; bile ductopeniaStage 3:portal-to-portal fibrous septa; severe degenerative changes + disappearance of bile ducts; cholestasis in periportal + paraseptal hepatocytesStage 4:frank cirrhosis*Associated with:* (1)Inflammatory bowel disease (ulcerative colitis in 50-74%, Crohn disease in 13%)✓1-4% of patients with inflammatory bowel disease develop sclerosing cholangitis!(2)Cirrhosis, chronic active hepatitis, pericholangitis, fatty degeneration(3)Pancreatitis(4)Retroperitoneal / mediastinal fibrosis(5)Peyronie disease(6)Riedel thyroiditis, hypothyroidism(7)Retroorbital pseudotumor ● abnormal liver function tests: serum alkaline phosphatase, g-glutamyltransferase ● progressive chronic / intermittent obstructive jaundice (most frequent) ● history of previous biliary surgery (53%) + chronic / recurrent pancreatitis (14%) ● fever, night sweats, chills, RUQ pain, itching (10-15%)Location: 1.CBD almost always involved2.Intra- and extrahepatic ducts (68-89%)3.Cystic duct involved in 15-18%4.Intrahepatic ducts only (1-11-25%)5.Extrahepatic ducts only (2-3%) ✓ intrahepatic bile duct calculi (8%): soft black crushable stones / sandlike gritUS: ✓ brightly echogenic portal triads✓ echogenic biliary casts / punctate coarse calcifications along portal vein branchesCT: ✓ dilatation, stenosis, pruning, beading of intrahepatic bile ducts (80%)✓ dilatation, stenosis, wall nodularity, duct wall thickening, mural contrast enhancement of extrahepatic bile ducts (100%)✓ hepatic metastases + lymph nodes in porta hepatis✓ subtle foci of high attenuation in intrahepatic bile ductsCholangiography: ✓ multifocal strictures with predilection for bifurcations + skip lesions (uninvolved duct segments of normal caliber) involving intra- and extrahepatic bile ducts✓ "pruned tree" appearance (= opacification of central ducts + diffuse obstruction of peripheral smaller radicles)✓ "cobblestone" appearance (= coarse nodular mural irregularities) in 50%✓ small saccular outpouchings (diverticula / pseudodiverticula) = PATHOGNOMONIC✓ CLASSIC "beaded appearance" (= alternating segments of dilatation and focal circumferential stenoses)✓ new strictures + lengthening of strictures between 6 months and 6 years (<20%)✓ marked ductal dilatation (24%)✓ polypoid mass (7%)✓ gallbladder irregularities uncommonNUC (Tc-99m-IDA scan): ✓ multiple persistent focal areas of retention in distribution of intrahepatic biliary tree✓ marked prolongation of hepatic clearance✓ gallbladder visualized only in 70%*Cx:*(1)Biliary cirrhosis(2)Portal hypertension(3)Cholangiocarcinoma (6-12-15%)*Rx:*4th leading indication for liver transplantation*DDx:* (1)Sclerosing cholangiocarcinoma (progressive cholangiographic changes within 0.5-1.5 years of initial diagnosis, marked ductal dilatation upstream from a dominant stricture, intraductal mass >1 cm in diameter)(2)Acute ascending cholangitis (history)(3)Primary biliary cirrhosis (disease limited to intrahepatic ducts, strictures less pronounced, pruning + crowding of bile ducts, normal AMA titer)

Notes:

Recurrent Pyogenic Cholangitis =PRIMARY CHOLANGITIS = RECURRENT PYOGENIC HEPATITIS = ORIENTAL CHOLANGIOHEPATITIS = ORIENTAL CHOLANGITIS = HONG KONG DISEASE= INTRAHEPATIC PIGMENT STONE DISEASE *Etiology:*? clonorchis infestation; endemic to South China, Indochina, Taiwan, Japan, Korea*Incidence:*3rd most common cause of an acute abdomen in Hong Kong after appendicitis and perforated ulcer*Age:*20-50 years; M:F = 1:1*Associated intrabiliary infestation:* Clonorchis sinensis, Ascaris lumbricoides, Escherichia coli ● recurrent attacks of fever, chills, abdominal pain, jaundiceLocation:particularly in lateral segment of L lobe + posterior segment of R lobe√ marked dilatation of proximal intrahepatic ducts(3-4 mm) in 100%√ decreased arborization of intrahepatic radicles√ intrahepatic bile ducts filled with nonshadowing soft mudlike pigment (calcium bilirubinate) stones (64%)√ dilatation of CBD (68%) + choledocholithiasis (30%)√ bile duct strictures (22%)√ pneumobilia (3-52%)√ segmental hepatic atrophy (36%)*Cx:*liver abscess (18%), splenomegaly (14%), biloma (4%), pancreatitis (4%)*DDx:*complication of Caroli disease

Notes:

Secondary Sclerosing Cholangitis *Cause:* (1)chronic bacterial cholangitis from bile duct stricture / choledocholithiasis(2)ischemic bile duct damage from treatment with floxuridine(3)infectious cholangiopathy in AIDS(4)previous biliary tract surgery(5)congenital biliary tree anomalies(6)bile duct neoplasm

Notes:

Acute Cholecystitis *Etiology:*(a)in 80-95% cystic duct obstruction by impacted calculus; 85% disimpact spontaneously(b)in 10% acalculous cholecystitis*Pathogenesis:*chemical irritation from concentrated bile, bacterial infection, reflux of pancreatic secretions*Age peak:*5th-6th decade; M:F = 1:3 ● persisting (>6 hours) RUQ pain radiating to right shoulder / scapula / interscapular area (DDx: biliary colic usually <6 hours) ● nausea, vomiting, chills, fever ● RUQ tenderness + guarding ● ± leukocytosis, elevated levels of alkaline phosphatase and transaminase and amylase ● mild hyperbilirubinemia (20%) ● Murphy sign = inspiratory arrest upon palpation of GB area (falsely positive in 6% of patients with cholelithiasis) Oral cholecystography: √ nonvisualization / poor visualization of gallbladderUS (81-100% sensitivity, 60-100% specificity): √ GB wall thickening >3 mm (45-72% sensitive, 76-88% specific)√ hazy delineation of GB wall√ "halo sign" = GB wall lucency (in 8%) = 3-layered configuration with sonolucent middle layer (edema)√ striated wall thickening (62%) = several alternating irregular discontinuous lucent + echogenic bands within GB wall (100% PPV)√ GB hydrops = distension with AP diameter >5 cm or enlargement of greater than 4 x 10 cm√ positive sonographic Murphy sign (in 85-88%)= focal tenderness over gallbladder (63-94% sensitive, 85-93% specific, 72% NPV)false-negative Murphy sign: lack of patient responsiveness, pain medication, inability to press directly on GB (position deep to liver / protected by ribs), GB wall necrosis √ crescent-shaped / loculated pericholecystic fluid (in 20%) = inflammatory intraperitoneal exudate / abscess√ gallstones (83-98% sensitive, 52-77% specific)√ impacted gallstone in GB neck / cystic duct√ echogenic shadowing fat within hepatoduodenal ligament ± conspicuous color Doppler flow (due to inflammation)Color Doppler US: √ visualization of cystic artery >50% of the length of the gallbladder (30% sensitive, 98% specific)NUC (86-97% sensitivity, 73-100% specificity , 95-98% accuracy): √ visualization of biliary tract + bowel√ nonvisualization of GB during 1st hour (in 83%)√ nonvisualization of GB by 4 hours (99% specificity)√ nonvisualization of GB + CBD (in 13%)√ rim sign (34%) = increased activity in GB fossa conforming to inferior hepatic edge (= sign of hyperemia); predictive value of 57% for gangrenous GB + 94% for acute cholecystitis√ increased perfusion to GB fossa during "arterial phase" (in up to 80%) False-positive scans (10-12%) = nonvisualization of GB in absence of acute cholecystitis: congenital absence of GB, carcinoma of GB, chronic cholecystitis, acute pancreatitis, alcoholic liver disease, hepatocellular disease, severe intercurrent illness, total parenteral nutrition, hyperalimentation, prolonged fasting, recent feeding <4-6 hours prior to study Reduction to 2% false-positive scans through: (1)delayed images up to 4 hours(2)cholecystokinin (Sincalide®) injection 15 minutes prior to study(3)morphine IV (0.04 mg/kg) at 40 minutes + reimaging after 20 minutes (contraction of sphincter of Oddi + rise in intrabiliary pressure)False-negative scans (4.8%): dilated cystic duct *Cx:* (1)Gangrene of gallbladder√ shaggy, irregular, asymmetric wall (mucosal ulcers, intraluminal hemorrhage, necrosis)√ hyperechoic foci within GB wall (microabscesses in Rokitansky-Aschoff sinuses)√ intraluminal pseudomembranes (gangrene)√ coarse nonshadowing nondependent echodensities (= sloughed necrotic mucosa / sludge / pus / clotted blood within gallbladder)(2)Perforation of gallbladder (in 2-20%)(a)acute free perforation with peritonitis causing pericholecystic abscess in 33%(b)subacute localized perforation causing pericholecystic abscess in 48%(c)chronic perforation resulting in internal biliary fistula causing pericholecystic abscess in 18%Location:most commonly perforation of fundus√ gallstone lying free in peritoneal cavity√ sonolucent / complex collection surrounding GB(3)Empyema of gallbladder√ multiple medium / coarse highly reflective intraluminal echoes without shadowing / layering / gravity dependence (purulent exudate / debris)*mnemonic:*"GAME BEG"**G**angrene **A**bscess (pericholecystic) **M**irizzi syndrome **E**mphysematous cholecystitis **B**ouveret syndrome (= gallstone erodes into duodenum leading to duodenal obstruction) **E**mpyema **G**allstone ileus

Notes:

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Acute Acalculous Cholecystitis *Frequency:*5-15% of all acute cholecystitis cases*Etiology:* probably caused by decreased blood flow within cystic artery(1)depressed motility / starvation in trauma, burns, surgery, total parenteral nutrition, anesthesia, mechanical ventilation, narcotics, shock, congestive heart failure, arteriosclerosis, polyarteritis nodosa, SLE, diabetes mellitus(2)obstruction of cystic duct by extrinsic inflammation, lymphadenopathy, metastases(3)infection from Salmonella, cholera, Kawasaki syndrome√ thickened gallbladder wall >4-5 mm√ echogenic bile sludge√ gallbladder distension√ pericholecystic fluid in absence of ascites√ subserosal edema√ sloughed mucosal membrane√ Murphy sign = pain + tenderness with transducer pressure over the gallbladder*Cx:*gallbladder perforation*Prognosis:*6.5% mortality rate

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Notes:

Chronic Cholecystitis Most common form of gallbladder inflammation √ gallstones√ smooth / irregular GB wall thickening (mean of 5 mm)√ mean volume of 42 mLNUC: √ normal GB visualization in majority of patients√ delayed GB visualization (1-4 hours)√ visualization of bowel prior to GB (sensitivity 45%, specificity 90%)√ noncontractility / decreased response after CCK injection (decreased GB ejection fraction)

Notes:

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Emphysematous Cholecystitis =ischemia of gallbladder wall + infection with gas-producing organisms*Etiology:*calculous (70-80%) / acalculous cystic duct obstruction with inflammatory edema resulting in cystic artery occlusion*Organism:*Clostridium perfringens, Clostridium welchii, E. coli, staphylococcus, streptococcus*Age:*>50 years; M:F = 5:1*Predisposed:* diabetics (20-50%), debilitating diseases ● WBC count may be normal (1/3) ● point tenderness rare (diabetic neuropathy)Plain film: √ gas appears 24-48 hours after onset of symptoms√ air-fluid level in GB lumen, air in GB wall within 24-48 hours after acute episode√ pneumobilia (rare)US: √ high-level echoes outlining GB wall*Cx:*gangrene (75%); gallbladder perforation (20%)*Mortality:*15%*DDx:*1.Enteric fistula2.Incompetent sphincter of Oddi3.Air-containing periduodenal abscess4.Periappendiceal abscess in malpositioned appendix5.Lipomatosis of gallbladder

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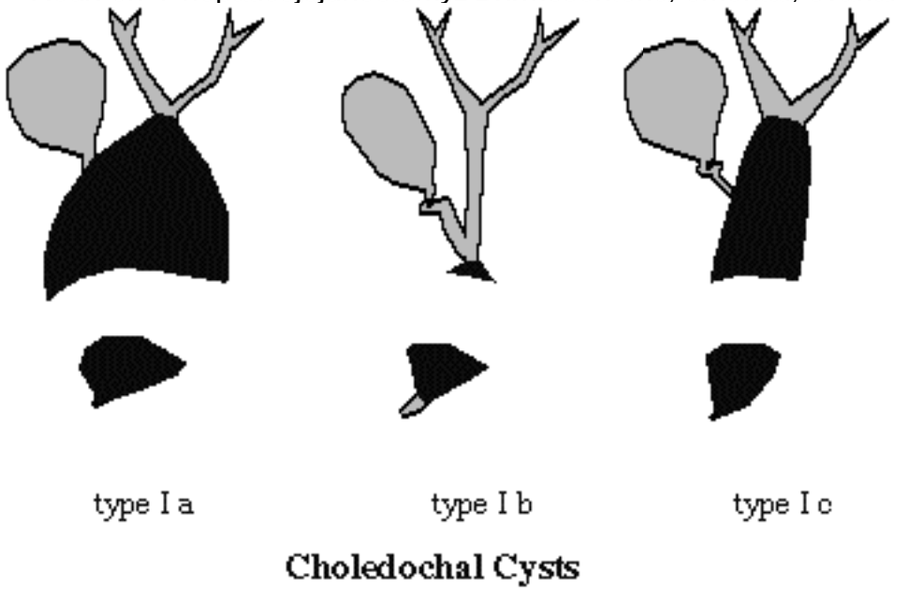
Notes:

Xanthogranulomatous Cholecystitis =FIBROXANTHOGRANULOMATOUS INFLAMMATION = CEROID GRANULOMAS OF THE GALLBLADDER=uncommon inflammatory disease of gallbladder characterized by presence of multiple intramural nodules*Etiology:* rupture of occluded Rokitansky-Aschoff sinuses with subsequent intramural extravasation of inspissated bile + mucin attracting histiocytes to phagocytose the insoluble cholesterol *Incidence:*1-2%*Age:*7th + 8th decadeHisto:mixture of ceroid (waxlike) xanthogranuloma with foamy histiocytes + multinucleated foreign body giant cells + lymphocytes + fibroblasts containing areas of necrosis (in newer lesions)*May be associated with:*gallbladder carcinoma (11%)√ preservation of 2-3 mm thick mucosal lining (in 82%)√ thickened gallbladder wall: 91% diffuse, 9% focal √ infiltration of pericholecystic fat: in 45% focal, in 54% diffuse√ hepatic extension (45%)√ biliary obstruction (36%)√ lymphadenopathy (36%)US: √ intramural hypoechoic nodulesCT: √ 5-20 mm small intramural hypoattenuating nodules√ poor / heterogeneous contrast enhancement*DDx:*gallbladder carcinoma (in 59% focal, in 41% diffuse thickening of gallbladder wall, multiple masses within liver)

Notes:

CHOLEDOCHAL CYST

=CYSTIC DILATATION OF EXTRAHEPATIC BILE DUCT= segmental aneurysmal dilatation of common bile duct without involvement of gallbladder / cystic duct; most common congenital lesion of bile ducts *Etiology:*anomalous junction of pancreatic duct and CBD proximal to duodenal papilla, higher pressure in pancreatic duct and absent ductal sphincter allows free reflux of enzymes into CBD resulting in weakening of CBD wall*Classification:* malunion of pancreaticobiliary duct Kimura Type I=pancreatic duct enters the proximal / mid CBDKimura Type II=CBD drains into pancreatic duct*Prevalence:*1:13,000 admissions; high prevalence in Japanese*Age:*<10 years (50%) + young adulthood, 80% diagnosed in childhood, 7% during pregnancy, occasionally detected up to 7th decade; M:F = 1:4*Histo:*fibrous cyst wall without epithelial lining*Associated with:* (1)dilatation, stenosis or atresia of other portions of the biliary tree (2%)(2)gallbladder anomaly (aplasia, double GB)(3)failure of union of left + right hepatic ducts (4)pancreatic duct + accessory hepatic bile ducts may drain into cyst(5)polycystic liver disease ● Classic triad (20-30% of adult patients):(1)intermittent obstructive jaundice (33-50%)(2)recurrent RUQ colicky pain (>75-90%), back pain(3)intermittent palpable RUQ abdominal mass (<25%) ● recurrent fever, chills, weight loss, pruritus*Types:* (a)marked cystic dilatation of CBD + CHD(b)focal segmental dilatation of CBD distally(c)cylindric dilatation of CBD + CHD√ size:diameter of 2 cm up to 15 cm (largest contained 13 liters)√ NO / mild peripheral intrahepatic bile duct dilatation√ may contain stones / sludgeUGI: √ soft-tissue mass in RUQ√ anterior displacement of 2nd portion of duodenum + distal portion of stomach / widening of C-loop with inferior displacement of duodenumUS: √ ballooned / fusiform cyst beneath porta hepatis separate from gallbladder√Communication with common hepatic / intrahepatic ducts needs to be demonstrated!√ abrupt change of caliber at junction of dilated segment to normal ducts√ intrahepatic bile duct dilatation (16%) secondary to stenosisOB-US (earliest diagnosis at 25 weeks MA): √ right-sided cyst in fetal abdomen + adjacent dilated hepatic ducts*DDx:*duodenal atresia; cyst of ovary, mesentery, omentum, pancreas, liverNUC with HIDA: (excludes effectively DDx of hepatic cyst, pancreatic pseudocyst, enteric duplication, spontaneous loculated biloma) √ photopenic area within liver that fills within 60 minutes + stasis of tracer within cyst√ prominent hepatic ductal activity (dilatation of ducts)*Cx:*1.Stones in gallbladder, within cyst, in intra-hepatic biliary tree, in pancreatic duct (8-50%)2.Recurrent pancreatitis (33%)3.Cholangitis (20%)4.Malignant transformation into bile duct carcinoma + gallbladder carcinoma (increasing with age, <1% in 1st decade, 7-14% > age 20)5.Cyst rupture with bile peritonitis (1.8%)6.Bleeding7.Biliary cirrhosis + portal hypertension*Rx:*excision of cyst + Roux-en-Y hepaticojejunostomy*DDx:*mesenteric, omental, ovarian, renal, adrenal, hepatic, pancreatic cyst, gastrointestinal duplication, hydronephrotic kidney



Choledochal Cysts

Notes:

CHOLEDOCHOCELE
=DUODENAL DUPLICATION CYST= ENTEROGENOUS CYST OF AMPULLA OF VATER / DUODENUM = INTRADUODENAL CHOLEDOCHAL CYST = DIVERTICULUM OF COMMON BILE DUCT= cystic dilatation of the distal / intramural duodenal portion of the CBD with herniation of CBD into duodenum (similar to ureterocele) *Etiology:* (1)congenital:(a)originates from tiny bud / diverticulum of distal CBD (found in 5.7% of normal population)(b)stenosis of ductal orifice / weakness of ductal wall(2)acquired: stone passage followed by stenosis + inflammation*Age:*33 years (manifestation usually in adulthood)*Types:*(a)CBD terminates in cyst, cyst drains into duodenum (common)(b)cyst drains into adjacent intramural portion of CBD (less common) ● biliary colic, episodic jaundice, nausea, vomiting√ stones / sludge are frequently presentUGI: √ smooth well-defined intraluminal duodenal filling defect in region of papilla√ change in shape with compression / peristalsisCholangiography (diagnostic): √ smooth clublike / saclike dilatation of intramural segment of CBD*Cx:*choledocholithiasis, pancreatitis*Rx:*sphincterotomy / sphincteroplasty*DDx:*choledochal cyst (involves more than only terminal portion of CBD)

Notes:

CHOLELITHIASIS

Predisposing factors: "female, forty, fair, fat, fertile, flatulent"(a)Hemolytic diseasesickle cell disease (7-37%), hereditary spherocytosis (43-85%), thalassemia, pernicious anemia (16-20%), prosthetic cardiac valves + mitral stenosis (hemolysis), cirrhosis (hemolysis secondary to hypersplenism), Rhesus / ABO blood group incompatibility (perinatal period) (b)Metabolic disorder = disruption of biliary lithogenic indexdiabetes mellitus, obesity, pancreatic disease, cystic fibrosis, hypercholesterolemia, hemosiderosis (20%), hyperparathyroidism, hypothyroidism, prolonged use of estrogens / progesterone, pregnancy (c)Cholestasis-hepatic dysfunction: hepatitis, neonatal sepsis-biliary tree malformation: Caroli disease-biliary obstruction: parasitic infection, benign / malignant strictures, foreign bodies (sutures, ascariasis)-prolonged fasting (total parenteral nutrition)-Methadone intake(d)Inflammatory bowel disease intestinal malabsorption has a 10 x increased risk of stone formation-Crohn disease (28-34%)(e)Genetic predisposition = familialNavaho, Pima, Chippewa Indians (f)Othersmuscular dystrophy GALLSTONES IN NEONATE ◊rare without predisposing factors*Associated with:*total parenteral nutrition, furosemide, GI dysfunction, prolonged fasting, phototherapy*Composition:* A.CHOLESTEROL STONE (70%)=main component of most calculi (70%)√ lucent (93%), calcified (7%)√ slightly hypodense compared with bile(a)pure cholesterol stones (10%): yellowish, soft√ buoyancy in contrast-enhanced bile√ density of <100 HU(b)mixture of cholesterol + calcium carbonate / bilirubinate (70%)√ laminated appearance√ radiopaque on plain film (15-20%)B.PIGMENT STONE (30%) ● black = compact "lacquer" of bilirubin derivatives with a high affinity for calcium carbonate ● brown = granular precipitate of calcium bilirubinate (in inflamed / infected gallbladders) contains <25% cholesterol√ multiple tiny faceted / spiculated homogeneously radiopaque stonesCT: √ usually denser than bile *Radioopacity:* √ lucent stones (84%):cholesterol (85%), pigment (15%) √ calcified stones (16% on plain film, 60% on CT):cholesterol (33%), pigment (67%) Location of calcium: √ calcium phosphate deposited centrally within cholesterol stones√ calcium carbonate deposited radially within aging cholesterol / peripherally around cholesterol + pigmented stones
FLOATING GALLSTONES (20-25%) (a)relatively pure cholesterol stones(b)gas-containing stones(c)rise in specific gravity of bile (1.03) from oral cholecystopaques (1.06) causing stones (1.05) to float GAS-CONTAINING GALLSTONES *Mechanism:*dehydration of older stones leads to internal shrinkage + dendritic cracks + subsequent nitrogen gas-filling from negative internal pressure√ "crow-foot" = "Mercedes-Benz" sign = radiating streaklike lucencies within stone, also responsible for buoyancy SLUDGE = calcium-bilirubinate granules + cholesterol crystals associated with biliary stasis secondary to prolonged fasting, parenteral nutrition, hyperalimentation, hemolysis, cystic duct obstruction, acute + chronic cholecystitis √ nonshadowing echogenic homogeneous mass shifting position slowly√ "sludge ball" = tumefactive sludge (DDx: gallbladder cancer)*DDx:*hemobilia, blood clot, parasitic infestation, mucus

Cholecystolithiasis Cholangiolithiasis

Notes:

Cholecystolithiasis *Incidence:*2% of children;10% of population; M:F = 1:3;in 3rd decade M:F = 2%:4%;in 7th decade M:F = 10%:25%*Peak age:*5th-6th decade • asymptomatic (60-65%); become symptomatic at a rate of 2% per year • **biliary colic** (misnomer) due to obstruction of cystic duct / common bile duct develops in 33% (18% overall risk in 20 years)=acute RUQ / epigastric / LUQ / precordial / lower abdominal pain increasing over seconds / minutes + remaining fairly steady for 4-6 hours • no tenderness upon palpationAbdominal plain film (10-16% sensitive) √ calcified gallstonesOCG (65-90% sensitive) √ filling defect in contrasted gallbladder lumen√ nonvisualization of gallbladder (25%) = inconclusiveCT (80% sensitive): √ hyperdense calcified gallstones in 60%√ hypodense cholesterol stones ≤140 HU = pure cholesterol stone (= ≥80% cholesterol content)◊Inverse relationship between CT attenuation number + cholesterol content√ gallstones isointense to bile in 21-24% and thus undetectable by CT (<30 HU)US (91-98% sensitive; in 5% falsely negative): √ mobile echogenic structure + acoustic shadowing within gallbladder (100% PPV)√ reverberation artifact√ nonvisualization of GB + collection of echogenic echoes with acoustic shadowing (15-25%)√ "double-arc shadow" = 2 echogenic curvilinear parallel lines separated by sonolucent rim (ie, GB wall + GB lumen + stone with acoustic shadowing)√ focal nonshadowing opacities <5 mm in diameter (in 70% gallstones)√ infrequently adherent to wallFALSE-NEGATIVE US (5%): contracted GB, GB in anomalous / unusual location, small gallstone, gallstone impacted in GB neck / cystic duct, immobile patient, obese patient, extensive RUQ bowel gas *Cx:*cholangitis, pancreatitis, fistula; cancer of GB + bile ducts (2-3 x more frequent)

Notes:

Cholangiolithiasis A.CHOLEDOCHOLITHIASIS◊Most common cause of bile duct obstruction!*Etiology:*(a)passed stones originating in GB(b)primary development in intra- / extrahepatic ducts*Incidence:*in 12-15% of cholecystectomy patients; in 3-4% of postcholecystectomy patients; in 75% of patients with chronic bile duct obstruction*Risk indicators for CBD stone:* (1)recent history of jaundice(2)recent history of pancreatitis(3)elevated serum bilirubin >17 µmol/L(4)elevated serum amylase >120 IU/L(5)dilated CBD >6 mm (16%)(6)obscured bile duct ● recurrent episodes of jaundice, chills, fever (25-50%) ● elevated transaminase (75%) ● spontaneous passage with stones <6 mm sizeCholangiography (most specific technique): √ stone visualization in 92%Peroperative cholangiography: prolongs operation by 30 minutes; 4% false-negatives; 4-10% false-positives US (22-82% sensitive): √ stone visualization in 13-75% (more readily with CBD dilatation + good visibility of pancreatic head)√ dilated ducts in 64-77% / normal-sized duct in 36%√ dilatation of CBD with administration of fatty meal / cholecystokinin√ no stone in gallbladder (11%)CT: √ stone visualization in 75-85% (isoattenuating to bile in 15-25%)√ target sign = intraluminal mass with crescentic ring (= stone of soft-tissue density) in 85%NUC:√ delayed bowel activity beyond 2 hours√ persistent hepatic + common bile duct activity to 24 hours√ prominent ductal activity beyond 90 minutes with visualization of secondary ducts B.STONE IN CYSTIC DUCT REMNANT:retained in 0.4% after surgery for choledocholithiasis

Notes:

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CHRONIC GRANULOMATOUS DISEASE OF CHILDHOOD
=recessive sex-linked immunodeficiency disorder resulting in purulent infections + granuloma formation primarily involving lymph nodes, skin, lungs*Etiology:*polymorphonuclear leukocyte dysfunction characterized by inability to generate hydrogen peroxide causing prolonged intracellular survival of phagocytized catalase-positive bacteria*Organism:*most commonly staphylococcus, Serratia marcescens, gram-negative enterococci*Path:*chronic infection with granuloma formation / caseation / suppuration*Age:*onset in childhood; M > F (more severe in boys) ● recurrent chronic infections: suppurative lymphadenitis, pyoderma ● chronic diarrhea ● perianal fistula + abscess@Chest√ chronic pneumonia√ hilar lymphadenopathy√ pleural effusions@Liver√ hepatosplenomegaly√ hepatic abscess√ liver calcifications@GI tract√ esophageal dysmotility, esophagitis, stricture√ gastric antral narrowing ± gastric outlet obstruction@Bone√ osteomyelitis

Notes:

CIRRHOSIS
=chronic liver disease characterized by diffuse parenchymal necrosis, regeneration and scarring with abnormal reconstruction of preexisting lobular architecture*Etiology:* A.TOXIC(1) Alcoholic liver disease in 75% (2) Drug-induced (prolonged methotrexate, oxyphenisatin, alpha- methyldopa, nitrofurantoin, isoniazid) (3) Iron overload (hemochromatosis, hemosiderosis) B.INFLAMMATION: Viral hepatitis, SchistosomiasisC.BILIARY OBSTRUCTION(1) Cystic fibrosis (2) Inflammatory bowel disease (3) Primary biliary irrhosis (4) Obstructive infantile cholangiopathy D.VASCULAR(1) Prolonged CHF = cardiac cirrhosis (2) Hepatic venoocclusive disease E.NUTRITIONAL(1) Intestinal bypass (2) Severe steatosis (3) Abetalipoproteinemia F.HEREDITARY(1) Wilson disease (2) Alpha-1-antitrypsin deficiency (3) Juvenile polycystic kidney disease (4) Galactosemia (5) Type IV glycogen storage disease (6) Hereditary fructose intolerance (7) Tyrosinemia (8) Hereditary tetany (9) Osler-Weber-Rendu syndrome (10) Familial cirrhosis G.IDIOPATHIC / CRYPTOGENIC*Cirrhosis in children:*biliary atresia, hepatitis, a-1-antitrypsin deficiency, tyrosinemia, hemochromatosis, Wilson disease, schistosomiasis*Morphology:* (a)micronodular cirrhosis (<3 mm): usually due to alcoholism, biliary obstruction, hemochromatosis, venous outflow obstruction, previous small-bowel bypass surgery, Indian childhood fibrosis(b)macronodular cirrhosis (3-15 mm, up to several cm): usually due to chronic viral hepatitis, Wilson disease, a-1-antitrypsin deficiency(c)mixed cirrhosis*Nodular lesions:* (a)**regenerative nodules** = localized proliferation of hepatocytes + supporting stroma(b)cirrhotic nodule = regenerative nodule largely / completely surrounded by fibrous septa(c)dysplastic nodule [adenomatous hyperplasia] = cluster of hepatocytes >1 mm in diameter with evidence of dysplasia; common in hepatitis B and C, a-1-antitrypsin deficiency, tyrosinemia(d)hepatocellular carcinoma *Associated with:*anemia, coagulopathy, hypoalbuminemia, cholelithiasis, pancreatitis, peptic ulcer disease, diarrhea, hypogonadism ● anorexia, weakness, fatigue, weight loss ● jaundice, continuous low-grade fever ● ascites, bleeding from esophageal varices, hepatic encephalopathy √ enlarged (early stage) / normal / shrunken liver√ shrinkage of right lobe (segments 5-8) and medial segment of left lobe (segments 4a + 4b) with concomitant hypertrophy of lateral segment of left lobe (segments 2 +3) and caudate lobe (segment 1):√ ratio of caudate to right lobe >0.65 on transverse images [sensitivity 43-84%, least sensitive in alcoholic cirrhosis, most sensitive in cirrhosis caused by hepatitis B; specificity 100%; 26% sensitivity; 84-96% accuracy] (DDx: Budd-Chiari syndrome) √ diameter of quadrate lobe (segment 4) <30 mm(= distance between left wall of gallbladder and ascending portion of left portal vein) due to selective atrophy (95% specific) √ widened porta hepatis + interlobar fissure√ surface nodularity + indentations (regenerating nodules)√ signs of portal hypertension√ splenomegaly√ ascites (failure of albumin synthesis, overproduction of lymph due to increased hydrostatic pressure in sinusoids / decreased splanchnic output due to portal hypertension)√ associated with fatty infiltration (in early cirrhosis)US (sensitivity 65-80%; DDx: chronic hepatitis, fatty infiltration): Hepatic signs: √ hepatomegaly (63%)√ hypertrophy of caudate lobe (26%)√ ratio of width of caudate lobe to width of right hepatic lobe >0.65 (43-84% sensitive, 100% specific)√ surface nodularity (88% sensitive, 82-95% specific)√ increased hepatic parenchymal echogenicity in 66% (as a sign of superimposed fatty infiltration)√ increased sound attenuation (9%) √ heterogeneous coarse (usually) / fine echotexture (7%)√ decreased / normal definition of walls of portal venules (sign of associated fatty infiltration NOT of fibrosis)√ occasional depiction of isoechoic regenerative nodules√ dilatation of hepatic arteries (increased arterial flow) with demonstration of intrahepatic arterial branches (DDx: dilated biliary radicals)√ increase in hepatic artery resistance after meal ingestion√ "portalization" of hepatic vein waveform = dampened oscillations of hepatic veins resembling portal vein flowExtrahepatic signs: √ splenomegaly√ ascites√ signs of portal hypertension CT: √ native + enhanced parenchymal inhomogeneity√ decreased attenuation (steatosis) in early cirrhosis√ isodense / hyperdense (siderotic) regenerative nodules√ nodular / lobulated liver contour√ predominantly portal venous supply to dysplastic nodules√ hypodense area adjacent to portal vein (= peribiliary cysts from obstructed extramural peribiliary glands) MR (problem-solving tool): √ no alteration of liver parenchyma√ regenerating nodules = hypointense lesions (due to iron deposits within nodules) with hyperintense septa (due to vascularity) on T2WI√ dysplastic nodule = iso- / hyperintense on T1WI + iso- / hypointense on T2WI√ HCC nodule = hypo- / iso- / hyperintense on T1WI + usually hyperintense on T2WI with marked enhancement during arterial phaseAngio: √ stretched hepatic artery branches (early finding)√ enlarged tortuous hepatic arteries = "corkscrewing" (increase in hepatic arterial flow)√ shunting between hepatic artery and portal vein√ mottled parenchymal phase√ delayed emptying into venous phase√ pruning of hepatic vein branches (normally depiction of 5th order branches) = postsinusoidal compression by developing nodulesNUC (Tc-99m-labeled sulfur colloid): √ high blood pool activity secondary to slow clearance√ colloid shift to bone marrow + spleen + lung√ shrunken liver with little or no activity + splenomegaly√ mottled hepatic uptake (pseudotumors) on colloid scan (normal activity on IDA scans!)√ displacement of liver + spleen from abdominal wall by ascites *Cx:*(1)Ascites: cause / contributor to death in 50%(2)Portal hypertension(3)Hepatocellular carcinoma (in 7-12%)(4)Cholangiocarcinoma*Fatality from:* esophageal variceal bleeding (in 25%), hepatorenal syndrome (10%), spontaneous bacterial peritonitis (5-10%), complications from treatment of ascites (10%)

Primary Biliary Cirrhosis

Notes:

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Primary Biliary Cirrhosis =CHRONIC NONSUPPURATIVE DESTRUCTIVE CHOLANGITIS*Histo:*idiopathic progressive destructive cholangitis of interlobar and septal bile ducts, portal fibrosis, nodular regeneration, shrinkage of hepatic parenchyma *Age:*35-55 years; M:F = 1:9 ● fatigue, pruritus ● xanthelasma / xanthoma (25%) ● hyperpigmentation (50%) ● insidious onset of pruritus (60%) ● IgM increased (95%) ● positive antimitochondrial antibodies (AMA) in 85-100%√ normal extrahepatic ducts√ cholelithiasis in 35-39%√ hepatomegaly (50%)√ tortuous intrahepatic ducts with narrowing + caliber variation / decreased arborization = "tree-in-winter" appearanceNUC: √ marked prolongation of hepatic Tc-99m IDA clearance√ uniform hepatic isotope retention√ normal visualization of GB and major bile ducts in 100% *DDx:*(1)Sclerosing cholangitis (young men)(2)CBD obstruction*Prognosis:*mean survival 6 (range 3-11) years after onset of cholestatic symptoms

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Notes:

CLONORCHIASIS
Rarely of clinical significance *Country:*Japan, Korea, Central + South China, Taiwan, Indochina*Organism:*Chinese liver fluke = Clonorchis sinensis *Cycle:*parasite cysts digested by gastric juice, larvae migrate up the bile ducts, remain in small intrahepatic ducts until maturity (10-30 mm in length), travel to larger ducts to deposit eggs*Infection:*snail + freshwater fish serve as intermediate hosts; infection occurs by eating raw fish; hog, dog, cat, man are definite hosts*Path:*(a)desquamation of epithelial bile duct lining with adenomatous proliferation of ducts + thickening of duct walls (inflammation, necrosis, fibrosis)(b)bacterial superinfection with formation of liver abscess ● remittent incomplete obstruction + bacterial superinfection multiple crescent- / stiletto-shaped filling defects within bile ducts *Cx:*(1)Bile duct obstruction (conglomerate of worms / adenomatous proliferation)(2)Calculus formation (stasis / dead worms / epithelial debris)(3)Jaundice in 8% (stone / stricture / tumor)(4)Generalized dilatation of bile ducts (2%)

Notes:

CONGENITAL BILIARY ATRESIA

Etiology:? variation of same infectious process as in neonatal hepatitis with additional component of sclerosing cholangitis or vascular injury*Histo:*proliferation of bile ducts in all portal triads*In 15% associated with:* polysplenia, trisomy 18 NUC [phenobarbital-augmented cholescintigraphy] (90-97% sensitivity, 63-94% specificity, 90% accuracy): preparation of patient with 5 ng/kg/d phenobarbital twice a day for 3-7 days to stimulate biliary secretion (via induction of hepatic enzymes + increase in conjugation + excretion of bilirubin)√ good hepatic activity within 5 min√ delayed clearance from cardiac blood pool√ NO biliary excretion√ NO visualization of bowel on delayed images at 6 and 24 hours√ increased renal excretion*DDx:*severe hepatocellular dysfunctionUS: √ normal (visualization of gallbladder in 20%)*Rx:*Kasai procedure (= portoenterostomy) (a)child <60 days of age: 90% success rate(b)child between 60 and 90 days of age:50% success rate (c)child >90 days of age: 17% success rate

Notes:

CONGENITAL HEPATIC FIBROSIS
=congenital cirrhosis with rapid + fatal progression*Histo:*fibrous tissue within hepatic parenchyma with excess numbers of distorted terminal interlobular bile ducts + cysts which rarely communicate with bile ducts*Age:*usually present in childhood resulting in early death*Associated with:*autosomal recessive type of polycystic kidney disease, medullary sponge kidney (80%) ● hepatosplenomegaly, portal hypertension ● predisposed to cholangitis + calculi√ "lollipop-tree" = ectasia of peripheral biliary radicles√ hepatosplenomegaly√ periportal fibrosis + portosystemic collaterals*Cx:*portal hypertension, hepatocellular carcinoma, cholangiocellular carcinoma

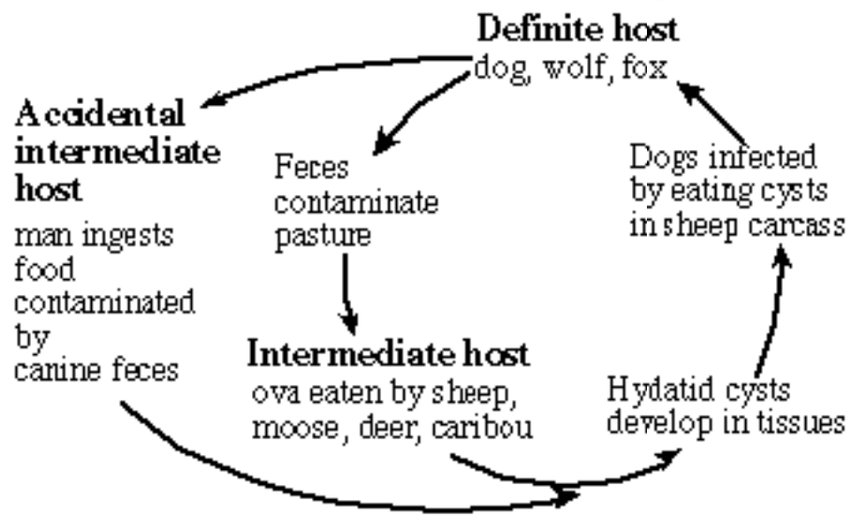
Notes:

DUCTECTATIC MUCINOUS TUMOR OF PANCREAS

=MUCIN-HYPERSECRETING CARCINOMA=rare intraductal tumor typified by voluminous mucin secretions*Site:*(a)main duct tumor causes diffuse segmental dilatation of the entire main pancreatic duct(b)branch duct tumor causes focal dilatation of affected branches; mainly in uncinate process ● endoscopy: inspissated mucus spilling out of a dilated hepatopancreatic ampulla√ mass usually in uncinate portion of pancreatic head√ cystic dilatation of pancreatic duct surrounded by thin rim of normal pancreatic parenchyma√ grapelike clusters of cysts containing thick mucinous secretions*Prognosis:*better than pancreatic adenocarcinoma

Notes:

Echinococcus Granulosus =HYDATID DISEASE=E. cysticus (more common); man is accidental host(a)pastoral (European) form: dog is definite host; intermediate hosts are cattle, sheep, horses, hogs; endemic in sheep-raising countries: Australia, New Zealand, North + East Africa, USSR, Mediterranean countries, Near + Middle East countries, Japan, Argentina, Chile, Uruguay(b)sylvatic (northern) form: wolf is definite host; intermediate hosts are deer, moose; endemic in northwestern Canada, Alaska*Cycle:*ingestion of contaminated material (eggs passed in feces of dog / other carnivore); eggs hatch in duodenum; larvae penetrate intestinal wall + mesenteric venules; larvae carried into portal circulation; larvae are filtered in capillaries of liver > lung > other organsOrgans:liver (73%); lung (14%); peritoneum (12%); kidney (6%); spleen (4%); spinal cord; brain; bladder; thyroid; prostate; heart; orbit (1-20%); bone*Histo:* A.ENDOCYST (parasitic component of capsule)=inner GERMINATIVE LAYER (resembling wet tissue paper) giving rise to brood capsules which may remain attached to cyst wall harboring up to 400,000 scolices / may detach + form sediment in cyst fluid = "hydatid sand" / may break up into numerous self-contained daughter cystsB.ECTOCYST = CYST MEMBRANE = laminated chitinlike substance secreted by parasiteC.PERICYST = highly vascularized adventitial layer (resembling egg white), organized host granulation tissue replaces tissue necrosis (due to compression of expanding cyst), marginal vascular rim of 0.5-4 mm ● pain / asymptomatic ● recurrent jaundice + biliary colic (transient obstruction by membrane fragments + daughter cysts expelled into biliary tree) ● blood eosinophilia (20-50%) ● urticaria + anaphylaxis (following rupture) ● Tests:1.Casoni intradermal test (60% sensitivity; may be falsely positive)2.Complement fixation double diffusion (65% sensitivity)3.Immunoelectrophoresis (most specific)4.Indirect hemagglutination (85% sensitivity)*Time to diagnosis:*11-81 (mean 51) years Location:right lobe > left lobe of liver; multiple cysts in 20%Size:up to 50 cm (average size of 5 cm), up to 16 liters of fluid Plain film: √ may have peripheral crescentic / curvilinear / polycyclic calcifications (10-33%), located in pericyst◊The presence of calcifications does not imply death of parasite!√ pneumohydrocyst (infection / communication with bronchial tree)US: √ complex heterogeneous mass (most common) √ well-defined anechoic cyst (common)√ "racemose" appearance = multiseptated cyst=daughter cysts internally and tangent to mother cyst (characteristic, but rare)√ floating undulating membrane / vesicles = separation of laminated membrane from pericyst (characteristic, but rare)◊Floating membrane does not indicate death of parasite!√ mass with eggshell calcification (least common)CT: √ well-demarcated low-density round masses of fluid attenuation ± internal septations√ enhancement of cyst wall + septations MR: √ hypointense rim surrounding multiloculated cystAngio: √ avascular area with splaying of arteries√ halo of increased density around cyst (inflammation / compressed liver)Cholangiography: √ cyst may communicate with bile ducts: right hepatic duct (55%), left hepatic duct (29%), CHD (9%), gallbladder (6%), CBD (1%) Percutaneous aspiration: ● fluid analysis positive for hydatid disease in 70% (fragments of laminated membrane in 54%; scolices in 15%; hooklets in 15%)◊Risk of anaphylactic shock (0.5%), asthma (3%), implantation of spilled protoscolices*Cx:*(1)Compression of vital structures(2)Infection(3)Rupture (25-90%)(a)contained = rupture of laminated membrane with cyst contents contained within pericyst(b)communicating = cyst contents escapes through biliary / bronchial tree(c)direct = tear of endocyst + ectocyst + pericyst with cyst contents spilling into pleural / peritoneal cavity (anaphylaxis, metastatic hydatidosis)*Rx:*(1)Surgery (in 10% recurrence)(2)Anthelmintics (albendazole, medendazole)(3)Injection of scolecidal agents (silver nitrate, 20 / 30% hypertonic saline solution, 0.5% cetrimide solution, 95% ethanol)



Parasitic Cycle of Echinococcus Granulosus

Notes:

Echinococcus Multilocularis =E. alveolaris = less common but more aggressive form of echinococcal diseasePrimary host:fox, wolfSecondary host:rodents (moles, lemmings, wild mice); domestic cat; dog*Endemic to:*eastern France, southern Germany, western Austria, much of Soviet Union, Japan, Alaska, Canada, some areas in Turkey*Infection:*eating wild fruits contaminated with fox / wolf feces; direct contact with fox / wolf; contact with dogs / cats that have ingested infested rodents*Path:*larvae proliferate by exogenous extension + penetration of surrounding tissue (= diffuse + infiltrative process resembling malignancy); chronic granulomatous reaction with central necrosis, cavitation, calcification*Histo:*daughter cysts with thick lamellar wall arising on outer surface of original cyst, rarely containing scolicesLocation:liver (access via portal vein); widespread hematogenous dissemination is not uncommon ● clinical manifestation 5-20 years after ingestion ● abdominal discomfort, jaundice, hepatomegaly ● eosinophilia √ aggressive growth pattern√ geographic infiltrating lesion with ill-defined margins√ invasion of IVC, diaphragm√ metastases to lung, heart, brain (in 10%)√ faint / dense amorphous / nodular / flame-shaped calcifications (dystrophic calcifications scattered throughout necrotic + granulomatous tissue)US: √ echogenic geographic ill-defined single / multiple solid masses√ ± irregular cystic areas√ propensity of spread to liver hilumCT: √ heterogeneous hypodense poorly marginated infiltrating masses√ pseudocystic necrotic regions of near water density surrounded by hyperdense solid component√ little / no enhancementAngio: √ intrahepatic arterial tapering + obstruction*Cx:*Budd-Chiari syndrome, IVC thrombosis, portal hypertension*Prognosis:*fatal within 10-15 years (if left untreated)*DDx:*hepatocellular carcinoma (biopsy!), large hemangioma (characteristic enhancement pattern), metastasis, epithelial hemangioendothelioma

Notes:

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EPIDERMOID CYST OF SPLEEN
= EPITHELIAL CYST = PRIMARY CYST OF SPLEEN *Cause:*infolding of peritoneal mesothelium / collection of peritoneal mesothelial cells trapped within splenic sulci*Histo:*(1) mesothelial lining (2) squamous epithelial lining = epidermoid cyst = squamous metaplasia from embryonic inclusions within preexisting mesothelial surface epithelium*Age:*2nd-3rd decade (average age of 18 years)*May be associated with:* polycystic kidney disease(a)unilocular + solitary (80%)(b)multiple + multilocular (20%)√ average size of 10 cm√ peripheral septations / cyst wall trabeculations (in 86%)√ curvilinear calcification in wall (9-25%)√ may contain cholesterol crystals, fat, blood*Cx:*trauma, rupture, infection

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Notes:

EPITHELIOID HEMANGIOENDOTHELIOMA

=primary malignant vascular tumor of liver (soft tissue, bone, lung)*Age:*average age of 45 years; M:F = 1:2*Possibly associated with:*oral contraceptives, exposure to vinyl chloride*Path:*multifocal nodules varying in size from a few mm to several cm involve both lobes of the liver (due to rapid perivascular extension); nodules may coalesce in liver periphery*Histo:*dendritic spindle-shaped cells + epithelioid round cells in a matrix of myxoid + fibrous stroma; neoplastic endothelial cells invade sinusoids + terminal hepatic veins + portal veins cutting off the tumors blood supply ● in 80%: abdominal pain, weakness, anorexia, jaundiceMetastases to:spleen, mesentery, lymph nodes, lung, bone√ multiple nodules (nodular form)√ peripheral subcapsular growth (diffuse form) without deforming liver contour√ increased tumor vascularity√ hypertrophy of uninvolved liverPlain film: √ hepatic calcifications (15%)US: √ typically hypoechoic lesions (due to central core of myxoid stroma)CT: √ low-attenuation masses on NECT, may become isoattenuating with rest of liver on CECT (due to vasoformative growth + compensatory hepatic arterial flow with portal vein occlusion)Angio: √ hyper- and hypovascularity (dependent upon degree of sclerosis + hyalinization)√ invasion ± occlusion of portal + hepatic veinsNUC: √ decreased perfusion to central myxoid tumor portion + increased perfusion to cellular areas on sulfur colloid scan√ photopenic defect on static sulfur colloid scan√ NOT gallium avid *Prognosis:*20% die within 2 years, 20% survive for 5-28 years ± treatment*DDx of multiple nodules:*metastatic disease*DDx of diffuse form:*sclerosing carcinoma, vaso-occlusive disease

Notes:

FATTY LIVER
= FATTY INFILTRATION OF THE LIVER = HEPATIC STEATOSIS *Cause:* A.METABOLIC DERANGEMENTpoorly controlled diabetes mellitus (50%), obesity, hyperlipidemia, acute fatty liver of pregnancy, protein malnutrition, parenteral hyperalimentation, malabsorption (jejunoileal bypass), glycogen storage disease, glycogen synthetase deficiency, cystic fibrosis, Reye syndrome, corticosteroids, severe hepatitis, trauma, congestive heart failure B.HEPATOTOXINSalcohol (>50%), carbon chlorides, phosphorus, amiodarone, chemotherapy *Histo:*hepatocytes with large cytoplasmatic fat vacuoles containing triglycerides; >5% fat of total liver weight
- NO abnormal liver function tests√ rapid change with time (few days to >10 months) depending on clinical improvement (abstinence from alcohol, improved nutrition) + degree of severity

Diffuse Fatty Infiltration Focal Fatty Infiltration

Notes:

Diffuse Fatty Infiltration √ hepatomegaly (75-80%) / normal sized liverPlain film: √ radiolucent liver sign = enlarged radiolucent liverUS (sensitivity >90%, accuracy 85-97%): √ increased sound attenuation (scattering of sound beam) = poor definition of posterior aspect of liver√ fine (more typical) / coarsened hyperechogenicity (compared with kidney)√ impaired visualization of borders of hepatic vessels√ attenuation of sound beam (feature of fat, NOT fibrosis)CT: √ areas of lower attenuation than normal portal vein / IVC density√ reversal of liver-spleen density relationship (spleen is normally 6-12 HU below liver density)√ hyperdense intrahepatic vascular structuresNUC: Tc-99m sulfur colloid scan: √ diffuse heterogeneous uptake (68%)√ reversal of liver-spleen uptake (41%)√ increased bone marrow uptake (41%)Xe-133 ventilation scan: √ increased activity during washout phase (38%)MR: √ slightly increased signal on T1WI + T2WI; relatively insensitive (10% fat by weight will alter SE signal intensities only by 5-15%)√ fat turns black with Dixon technique FAT-SPARED AREA in diffuse fatty infiltration *Cause:*direct drainage of systemic blood into liverLocation:(a)posterior edge of segment 4 = anterior to portal vein bifurcation (drainage of aberrant gastric vein)(b)next to gallbladder bed (drainage of cystic vein)(c)subcapsular skip areas√ hypoechoic ovoid / spherical / sheetlike mass√ NO mass effect (undisplaced course of vessels)*DDx:*tumor mass

Notes:

Focal Fatty Infiltration *Etiology:*? vascular origin, focal tissue hypoxiaDistribution:(a)lobar / segmental uniform lesions(b)lobar / segmental nodular lesions(c)perihilar lesions(d)diffuse nodular lesions(e)diffuse patchy lesionspredominantly in centrilobar + periportal regions, subcapsular distribution may be due to variants of blood supply (direct connections between peripheral portal radicles + perforating capsular / accessory cystic veins) Location:right lobe, caudate lobe, perihilar region√ fan-shaped lobar / segmental distribution with angulated / interdigitating geographic margins√ lesions extend to periphery of liver√ NO mass effect (undisplaced course of vessels, no bulging of liver contour)US: √ hyperechoic area with poorly defined / sharp margins√ multiple / rarely single echogenic nodules simulating metastases (rare)CT: √ patchy areas of decreased attenuation ranging from -40 to +10 HU (DDx: liver tumor)√ NO contrast enhancementMR (not sensitive for fat): √ high signal on T1WI + low / isointense signal on T2WINUC with colloid: √ no significant changes on sulfur colloid images (SPECT imaging may detect focal fatty infiltration)*DDx:*primary / secondary hepatic tumor

Notes:

FOCAL NODULAR HYPERPLASIA

=FNH = rare benign congenital hamartomatous malformation or reparative process in areas of focal injury; SPECIFIC DIAGNOSIS RARELY POSSIBLE*Cause:*(?) congenital arteriovenous malformation triggers focal hepatocellular hyperplasia owing to a regional increase in blood flow Oral contraceptives DO NOT cause FNH, but exert a trophic effect on its growth!*Incidence:*only 357 cases reported; 2nd most common benign tumor of liver; 4% of all primary hepatic tumors in pediatric population, 3-8% in adult population; twice as common as hepatocellular adenoma*Path:*localized, well-delineated, usually solitary (80-95%), subcapsular mass of numerous small lobules within an otherwise normal liver; no true capsule; frequently central fibrous scar in area of interconnection of fibrous bands (HALLMARK) containing centrally an arterial malformation with spiderlike branches supplying the component nodules*Histo:*composed of multiple spherical aggregates of hepatocytes often containing increased amounts of fat + triglycerides + glycogen; thick-walled arteries within fibrous septa radiating from the center toward the periphery; absent portal triads + central veins; bile duct proliferation within fibrous septa without connection to biliary tree; Kupffer cells; difficult differentiation from regenerative nodules of cirrhosis + hepatocellular adenoma*Age peak:*3rd-4th decade (range: 7 months to 75 years); M:F = 1:2-4*Associated with:*hepatic hemangioma (in 23%), meningioma, astrocytoma, arterial dysplasia of other organs in case of multiple FNH ● initially often asymptomatic (in 50-90% incidental finding) ● vague abdominal pain (10-15%) due to mass effect ● normal liver function ● hepatomegaly / abdominal mass √ size <5 cm (in 85%); right lobe:left lobe = 2:1√ well-circumscribed, nonencapsulated nodular cirrhotic-like mass in an otherwise normal liver√ NO calcifications√ pedunculated mass (in 5-20%)√ multiple masses (in 20%)NECT: √ iso- / slightly hypoattenuating homogeneous massCECT: √ transient intense hyperdensity (after 30-60 sec) on bolus injection followed rapidly by isodensity Lesion may be missed without precontrast study!√ hypodense central stellate scar = central fibrous core with radiating fibrous septa (15-33%) (DDx: fibrolamellar HCC)√ ± early enhancement of vessels traversing central scar√ hypodense mass during peak portal venous phase√ isodense mass following portal venous phase√ hyperdense central scar on delayed images (delayed washout of contrast from myxomatous scar tissue)US: √ iso- / hypo- / hyperechoic (33%) homogeneous mass√ hyperechoic central scar in 18%√ displacement of hepatic vesselsDoppler: √ enlarged afferent blood vessel with central arterial hypervascularity + centrifugal filling to the periphery in "spoke-wheel" pattern√ large draining veins at tumor margins√ may show high-velocity Doppler signals with arterial pulsatility from arteriovenous shunts NUC: Sulfur colloid scan: √ normal uptake (50-70%), hot spot (7-10%) Only FNH contains sufficient Kupffer cells to cause normal / increased uptake (almost PATHOGNOMONIC)! √ cold spot (30-50%)(DDx: hepatic adenoma, hemangioma, hepatoblastoma, liver herniation, hepatocellular carcinoma) Tc-HIDA: √ normal / increased uptake (40-70%), cold spot (60%)Tc-99m-tagged RBCs: √ increased uptake during early phase√ defect relative to liver on delayed images MR: √ usually homogeneous signal intensity of lesion√ iso- to hypointense on T1WI (94-100%)√ slightly hyper- to isointense on T2WI (94-100%)√ atypically hyperintense lesion on T1WI in 6%√ central scar hypointense on T1WI√ central scar hyperintense on T2WI in 75% (due to vascular channels + edema) / hypointense in 25% (absent or minimal edema)CEMR: √ dense enhancement in arterial phase√ isointense during portal venous phase√ hyperintense on delayed images√ late + prolonged enhancement of central scar√ occasionally prolonged enhancement (due to entrapment of Gd-DTPA by functioning hepatocytes inside tumor followed by 1% excretion into biliary tree)√ less uptake of IV superparamagnetic iron oxide than surrounding liver (uptake mechanism similar to that of sulphur colloid)Angio: √ discretely marginated hypervascular mass (90%) with intense capillary blush / hypovascular (10%)√ enlargement of main feeding artery with central blood supply (= "spoke-wheel" pattern in 33%)√ homogeneous parenchymal stain√ decreased vascularity in central stellate fibrous scar *Rx:*(1)Discontinuation of oral contraceptives(2)Resection of pedunculated mass(3)Diagnostic excisional biopsy for extensive tumor (FNH seldom requires surgery)*Cx:*rarely rupture with hemoperitoneum (increased incidence in patients on oral contraceptives - 14%)*DDx:* 1.Fibrolamellar carcinoma (scar calcified, metastases, retroperitoneal adenopathy, tumor hemorrhage + necrosis causing pain, hypointense scar on T2WI)2.Hepatic adenoma (10 cm large tumor, symptomatic due to propensity for hemorrhage in 50%, central scar atypical)3.Well-differentiated hepatocellular carcinoma (internal necrosis + hemorrhage, vascular invasion, metastases, rim-enhancement of pseudocapsule)4.Giant cavernous hemangioma (larger tumor, may calcify, globular peripheral enhancement followed by centripetal filling, retention of contrast on delayed images, CSF-like behavior on MRI)5.Hypervascular metastasis (hypovascular during portal venous phase, older patient)6.Intrahepatic cholangiocarcinoma (less vascular, dominant large central scar, metastases)

Notes:

GALLBLADDER CARCINOMA

Most common biliary cancer (9 x more common than extrahepatic bile duct cancer); 5th most common gastrointestinal malignancy (after colorectal, pancreatic, gastric, esophageal carcinoma); 3% of all intestinal neoplasms *Incidence:*0.4-4.6% of biliary tract operations; 6,500 deaths/year in United States*Peak age:*6-7th decade; M:F = 1:3-1:4◊85% occur in 6th decade or later!*Histo:*(a)well differentiated adenocarcinoma of scirrhous type (80-90%)(b)anaplastic carcinoma, squamous cell carcinoma, adenoacanthoma (10-20%)(c)carcinoid, sarcoma, basal cell carcinoma, lymphoma (extremely rare)*Stage:* Imucosa onlyIImucosa + muscularisIIImucosa + muscularis + serosaIVgallbladder wall + lymph nodesVhepatic / distant metastases *Predisposed:*patients with porcelain gallbladder (22%); gallbladder polyp >2 cm is likely malignant*Associated with:* (1)Gallstones in 64-98%◊Gallbladder carcinoma occurs in only 1% of all patients with gallstones!(2)Porcelain gallbladder (in 4-60%): prevalence of gallbladder carcinoma in 11-22% of autopsies(3)Inflammatory bowel disease (predominantly ulcerative colitis, less common in Crohn disease)(4)Familial polyposis coli(5)Chronic cholecystitis ● history of past GB disease (50%) ● malaise, vomiting, weight loss ● RUQ pain (54-76%) ● obstructive jaundice (35-74%) ● abnormal liver function tests (20-75%) Location:usually in body / fundus; rarely in cystic ductGrowth types: √ focal (59%) / diffuse (41%) thickening of GB wall√ polypoid / fungating intraluminal mass with wide base (14-25%)√ replacement of gallbladder by mass (37-70%)√ pericholecystic infiltration: in 76% focal, in 24% diffuse √ dilatation of biliary tree (38-70%)√ fine granular / punctate flecks of calcification (mucinous adenocarcinoma)OCG: √ nonvisualization of gallbladder (2/3)Metastases:in 75-77% at time of diagnosis(a)direct invasion of liver (34-89%), duodenum (12%), colon (9%), stomach, bile duct, pancreas, right kidney, abdominal wall(b)lymphatic spread (26-41-75%): porta hepatis, portacaval, lesser omental, superior + posterior pancreaticoduodenal, paraaortic nodes(c)intraperitoneal seeding (common)(d)hematogenous spread (less common): liver, lung, bones(e)neural spread (frequent): associated with more aggressive tumors(f)intraductal spread (least common): particularly in papillary adenocarcinoma *Cx:*perforation of gallbladder + abscess formation√ gallstones located within abscess*Prognosis:*75% unresectable at presentation; average survival is 6 months; 5% 1-year survival rate; 6% 5-year survival rate *DDx:*(1)Xanthogranulomatous cholecystitis (lobulated mass filling gallbladder + stones)(2)Acute / chronic cholecystitis (generalized gallbladder wall thickening <10 mm)(3)Liver tumor invading gallbladder fossa(4)Tumors from adjacent organs (pancreas, duodenum)(5)Metastases (melanoma, leukemia, lymphoma)(6)Polyps: cholesterol polyp, hyperplastic polyp, granulation polyp(7)Adenomyomatosis

Notes:

GLYCOGEN STORAGE DISEASE
=autosomal recessive diseases with varying severity and clinical syndromesA.VON GIERKE DISEASE (TYPE I)*Etiology:*defect in glucose-6-phosphatase with excess deposition of glycogen in liver, kidney, intestines*Dx:*failure of rise in blood glucose after glucagon administration*Age at presentation:*infancy√ hepatomegalyUS: √ increased echogenicity (glycogen / fat)CT: √ increased (glycogen) / normal / decreased (fat) parenchymal attenuation*Prognosis:*death in infancy, may survive into adulthood with early therapy*Cx:*(1) Hepatic adenoma(2) Hepatocellular carcinoma B.POMPE DISEASE (TYPE II)=abnormal metabolism with enlargement of myocardial cells due to glycogen deposition; similar to endocardial fibroelastosis*Etiology:*defect in lysosomal glucosidase√ massive cardiomegaly with CHF√ hepatomegaly*Prognosis:*sudden death in 1st year of life (due to conduction abnormalities); survival rarely beyond infancyC.CORI DISEASE (TYPE III)D.ANDERSEN DISEASE (TYPE IV)E.McARDLE DISEASE (TYPE V)F.HERS DISEASE (TYPE VI)

Notes:

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HEMOCHROMATOSIS
=excess iron deposition in various parenchymal organs (liver, pancreas, spleen, kidneys, heart) leading to cirrhosis with portal hypertension
[HEMOSIDEROSIS=increased iron deposition without organ damage]*Cause:*excess iron deposition from(a)increased GI absorption:1.Genetic hemochromatosis2.Erythropoietic hemochromatosis3.Bantu siderosis(b)IV blood transfusion(c)intravascular (extrasplenic) hemolysis

Genetic Hemochromatosis Secondary Hemochromatosis

Notes:

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Genetic Hemochromatosis =IDIOPATHIC / PRIMARY HEMOCHROMATOSIS=excessive absorption + parenchymal retention of dietary iron that favors accumulation within non-RES organs (liver, pancreas, heart, pituitary gland)*Cause:*autosomal recessive disorder (human-leukocyte antigen[HLA]-linked abnormal gene located on short arm of chromosome 6) with mucosal defect in intestinal wall / increased absorption of intestinal iron*Prevalence:*1:220 whites of northern European ancestry; homozygote frequency up to 0.25-0.50%; heterozygote carriers >10%*Pathophysiology:* absorbed iron is selectively bound to transferrin; increased transferrin saturation in portal circulation favors selective iron uptake by periportal hepatocytes as initial site of iron accumulation; RES cells are incapable of storing excess iron *Path:*excess iron stored as crystalline iron oxide (ferric oxyhydroxide) within cytoplasmic ferritin + lysosomal hemosiderin; iron overload affects parenchymal cells (liver, pancreas, heart) NOT Kupffer cells / RE cells of bone marrow + spleen (abnormal function of RES) ● asymptomatic during 1st decade of disease ● hyperpigmentation (90%) ● hepatomegaly (90%) ● arthralgias (50%) ● diabetes mellitus (30%) secondary to insulin resistance by hepatocytes + pancreatic b-cell damage from iron deposition ● CHF + arrhythmia (15%) ● loss of libido, impotence, amenorrhea, testicular atrophy, loss of body hair ● liver iron index > 2 (= liver iron concentration [micromoles per gram of dry weight] per patients age)CT (60% sensitivity for iron): √ diffuse / rarely focal increase in liver density (up to 75-130 HU)√ depiction of hepatic veins on NECT√ dual energy CT (at 80 + 120 kVp) can quantitate amount of iron depositionMR: (skeletal muscle = good signal intensity reference) √ significant signal loss in liver on T2WI with signal intensity equal to background noise√ normal pancreatic signal intensity in noncirrhotics√ pancreatic signal intensity equal to / less than muscle (in 90% of cirrhotic patients)√ normal signal intensity of spleen (in 86%) due to abnormal RES function*Dx:*liver biopsy*Cx:*(1)Periportal fibrosis resulting in cirrhosis (if iron concentration >22,000 µg/g of liver tissue)(2)Hepatocellular carcinoma (14-30%)(3)Insulin-dependent diabetes mellitus (30-60%)(4)Congestive cardiomyopathy (15%)*Rx:*phlebotomies in precirrhotic stage*Prognosis:*normal life expectancy with early diagnosis and treatment

Notes:

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Secondary Hemochromatosis *Cause:* (1)Erythrogenic hemochromatosis = increased absorption of iron secondary to erythroid hyperplasia in ineffective erythropoiesis (eg, thalassemia, NOT in sickle cell anemia)*Path:*no excess Kupffer cell iron(2)Bantu siderosis = excessive dietary iron from food preparation in iron containers (Kaffir beer)(3)Transfusional iron overload = patients receiving > 40 units of blood (iron storage capacity of RES = 10 g of iron)*Path:*iron deposition initially in RES (phagocytosis of intact RBC) with sparing of parenchymal cells of pancreas; after saturation of RES storage capacity parenchymal cells of other organs accumulate iron (liver, pancreas, myocardium)*Age:*4th-5th decade; M:F = 10:1 ● little clinical significanceMR: ↓ signal loss in liver on T2WI with signal intensity greater than background noise (iron in Kupffer cells)↓ splenic signal intensity less than muscle

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Notes:

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HEPATIC ABSCESS
=localized collection of pus in the liver resulting from any infectious process with destruction of the hepatic parenchyma + stroma*Types:*pyogenic (88%), amebic (10%), fungal (2%)Location:multiple in 50%√ hepatomegaly√ elevation of right hemidiaphragm√ pleural effusion√ right lower lobe atelectasis / infiltration√ gas within abscess (esp. Klebsiella)MR: √ hypointense on T1WI + hyperintense on T2WI (72%)√ perilesional edema (35%)√ "double target sign" on T2WI = hyperintense center (fluid) + hypointense sharply marginated inner ring (abscess wall) + hyperintense poorly marginated ring (perilesional edema)√ rim enhancement (86%)

Amebic Abscess Pyogenic Liver Abscess

Notes:

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Amebic Abscess *Organism:*Entamoeba histolytica*Etiology:*spread of viable amebae from colon to liver via portal system*Incidence:*in 1-25% of intestinal amebiasis*Age:*3rd-5th decade; M:F = 4:1 ● amebic dysentery ● amebic hepatitis (15%)Location:liver abscess (right lobe) in 2-25%; systemic dissemination by invasion of lymphatics / portal system (rare); liver:lung:brain = 100:10:1Size:2-12 cm; multiple liver abscesses in 25% √ nodularity of abscess wall (60%)√ internal septations (30%)√ not gas-containing (unless hepatobronchial / hepatoenteric fistula present)NUC: √ sensitivity of sulfur colloid scan is 98%√ photon-deficient area surrounded by rim of uptake on Ga-67 scanAspiration: typically opaque reddish / dirty brown / pink material ("anchovy paste" / "chocolate sauce"), usually sterile, parasite confined to margin of abscess *Cx:*(1)Diaphragmatic disruption (rare) is strongly suggestive of amebic abscess(2)Fistulization into colon, right adrenal gland, bile ducts, pericardium*Rx:*conservative treatment with chloroquine / metronidazole (Flagyl®)

Notes:

Pyogenic Liver Abscess *Organisms:*E. coli, aerobic streptococci, St. aureus, anaerobic bacteria (45%)*Incidence:*0.016%*Etiology:*(1)Ascending cholangitis from obstructive biliary tract disease (malignant / benign)(2)Portal phlebitis (suppurative appendicitis, colitis, diverticular disease)(3)Infarction from embolism / septicemia(4)Indwelling arterial catheters(5)Direct spread from contiguous infection (cholecystitis, peptic ulcer, subphrenic sepsis)(6)Trauma (rupture, penetrating wounds, biopsy, surgery)(7)Cryptogenic in 45% (invasion of cysts / dead tissue by pyogenic intestinal flora)*Age:*6th-7th decade; M > F ● pyrexia (79%) ● abdominal pain (68%) ● nocturnal sweating (43%) ● vomiting / malaise (39%) ● jaundice (0-20%) ● positive blood culture (50%)Location:solitary abscess in right lobe (40-75%), in left lobe (2-10%); multiple abscesses in 10-34-73% (more often of biliary than hematogenous origin)US: √ hypoechoic round lesion with well-defined mildly echogenic rim√ distal acoustic enhancement√ coarse clumpy debris / low-level echoes / fluid-debris level√ intensely echogenic reflections with reverberations (from gas) in 20-30%CT: √ inhomogeneous hypodense single / multiloculated cavity√ "double target sign" = wall-enhancement + surrounding hypodense zone (6-30%)√ "cluster sign" = several abnormal foci within the same anatomic area; suggestive of biliary originNUC: √ photon-deficient area on sulfur colloid + IDA scan√ Ga-67 citrate uptake in 80%√ In-111 tagged WBC uptake is highly specific (since WBCs normally go to liver, may need sulfur colloid test for correlation)*Cx:*(1) Septicemia(2) Rupture into right subphrenic space(3) Rupture into abdominal cavity(4) Rupture into pericardium(5) Empyema(6) Common hepatic duct obstruction*Mortality:*20-80%; 100% if unrecognized / untreated

Notes:

HEPATIC ADENOMA
=HEPATOCELLULAR ADENOMA = LIVER CELL ADENOMA=rare benign neoplasm, most frequent hepatic tumor in young women after use of contraceptive steroids*Path:*no true capsule; pseudocapsule due to compression of liver tissue containing multiple large vessels; high incidence of hemorrhage + necrosis + fatty change; no scar*Histo:*solitary spherical benign growth of hepatocytes; sheets of hepatocytes without portal veins or central veins; scattered thin-walled vascular channels + bile canaliculi; decrease in number of abnormally functioning Kupffer cells; hepatocytes contain increased amounts of glycogen ± fat*Age:*young women in childbearing age; not seen in males unless on anabolic steroids*Associated with:*oral contraceptives (2.5 x risk after 5-year use, 7.5 x risk after 9-year use, 25 x risk >9-year use), steroids, pregnancy, diabetes mellitus, type Ia glycogen storage disease (von Gierke) in 60%Pregnancy may increase tumor growth rate + lead to tumor rupture!Tumor remission may occur with dietary therapy leading to normal insulin, glucagon, and serum glucose levels • asymptomatic (20%) • RUQ pain as sign of mass effect (40%) / intratumoral or intraperitoneal hemorrhage (40%) • hepatomegalyLocation:right lobe of liver in subcapsular location (75%)√ round well-circumscribed mass; between 6-30 cm in size (average size of 8-10 cm)√ intraparenchymal / pedunculated (in 10%)√ unusual "nodule-in-nodule" appearance in large tumors (DDx: hepatocellular carcinoma) CT: √ round mass of decreased density; areas of necrosis (30-40%)√ hyperdense areas of fresh intratumoral hemorrhage (22-50%)√ transiently enhancing on arterial-phase images√ iso- / hypoattenuating on delayed-phase imagesUS: √ usually small well-demarcated solid echogenic / complex hyper- and hypoechoic heterogeneous mass with anechoic areas (if large)MR: √ inhomogeneous on all pulse sequences (indistinguishable from HCC)√ often hyperintense areas on T1WI (due to presence of fat-laden hepatocytes / hemorrhage)√ isointense (sheets of hepatocytes) and hyperintense areas (necrosis, hemorrhage) on T2WINUC: √ focal photopenic lesion on sulfur colloid scan (because lesion composed of hepatocytes + nonfunctioning Kupffer cells) surrounded by rim of increased uptake (due to compression of adjacent normal liver containing Kupffer cells); may show uptake equal to / slightly less than liver (23%) √ usually increased activity on HIDA scan√ NO gallium uptakeAngio: √ usually hypervascular mass√ homogeneous but not intense stain in capillary phase√ enlarged hepatic artery with feeders at tumor periphery (50%)√ hypo- / avascular regions (secondary to hemorrhage / necrosis)√ neovascularity CAVE: percutaneous biopsy carries high risk of bleeding! *Cx:*(1)Spontaneous hemorrhage with subcapsular hematoma / hemoperitoneum (41%)(2)Malignant transformation (? contiguous development of hepatocellular carcinoma)(3)Recurrence after resection*Rx:*surgical resection (to prevent rupture)*DDx:*hepatocellular carcinoma

Notes:

HEPATIC ANGIOSARCOMA

=HEMANGIOENDOTHELIAL SARCOMA = KUPFFER CELL SARCOMA = HEMANGIOSARCOMA*Prevalence:*0.14-0.25 per million; <2% of all primary liver neoplasms; most common sarcoma of liver (followed by fibrosarcoma > malignant fibrohistiocytoma > leiomyosarcoma)*Etiology:*(a)thorotrast = thorium dioxide (7-10%) with latent period of 15-24 years(b)arsenic(c)polyvinyl chloride (latent period of 4-28 years)*Associated with:*hemochromatosis, von Recklinghausen disease*Path:*(a)multifocal / multinodular lesions (71%) of up to >5 cm in size(b)large solitary mass with hemorrhage + necrosis*Histo:* (a)vessels lined with malignant endothelial cells(eg, sinusoids) causing atrophy of surrounding liver(b)vasoformative = forming poorly organized vessels(c)forming solid nodules of malignant spindle cells*Age:*6th-7th decade; M:F = 4:1 ● abdominal pain, weakness, fatigue, weight loss ● spontaneous hemoperitoneum (27%) ● jaundice ● NO elevation of a-fetoproteinEarly metastases to: lung, spleen (16%), porta hepatis nodes, portal vein, thyroid, peritoneal cavity, bone marrow (rapid metastatic spread) √ portal vein invasion√ hemorrhagic ascitesPlain film: √ circumferential displacement of residual thorotrastNUC: √ single / multiple photopenic areas on sulfur colloid scan√ increased gallium uptake√ perfusion blood pool mismatch (initial decrease followed by slow increase in RBC concentration) as in hemangioma on 3-phase red blood cell scanUS: √ solid / mixed mass with anechoic areas (hemorrhage / necrosis)√ multiple nodulesCT: √ hypodense masses with high-density regions (hemorrhage) / low-attenuation regions (old hemorrhage / necrosis)√ striking peripheral enhancement on dynamic CT as in large hemangiomaMR: √ hypointense on T1WI + hyperintense on T2WI√ peripheral Gd-pentetate enhancement on T1WIAngio: √ hypervascular stain around tumor periphery in late arterial phase with puddling; NO arterial encasementCAVE:Biopsy may lead to massive bleeding in 16%! Opt for open rather than percutaneous biopsy! *Prognosis:*rapid deterioration with median survival of 6 months (13 months under chemotherapy)*DDx for multiple lesions:*metastases*DDx for single lesion:*cavernous hemangioma

Notes:

HEPATIC CYST

=second most common benign hepatic lesion*Prevalence:*2-7%; increasing with ageA.ACQUIRED HEPATIC CYSTsecondary to trauma, inflammation, parasitic infestation, neoplasia B.CONGENITAL HEPATIC CYST=defective development of aberrant intrahepatic bile ducts*Incidence:*liver cysts detected at autopsy in 50%; in 22% detected during life*Age of detection:*5th-8th decade*Histo:*cysts surrounded by fibrous capsule + lined by columnar epithelium, related to bile ducts within portal triads; no communication with bile ducts*Associated with:* (1)Tuberous sclerosis(2)Polycystic kidney disease (25-33% have liver cysts)(3)Polycystic liver disease: autosomal dominant; M:F = 1:2; (50% have polycystic kidney disease) ● hepatomegaly (40%); pain (33%); jaundice (9%)Size of cyst:range from microscopic to huge (average 1.2 cm; in 25% largest cyst <1 cm; in 40% largest cyst >4 cm; maximal size of 20 cm); multiple cysts spread throughout liver (in 60%) / solitary cyst √ "cold spot" on IDA, Ga-68, Tc-99m sulfur colloid scans√ echo-free cyst, may show fluid-fluid interface*Rx:*sclerosing with minocycline hydrochloride (Dose: 1 mg per 1-mL cyst content up to 500 mg in 10 mL of 0.9% saline + 10 mL 1% lidocaine) following contrast opacification of cyst to confirm absence of communication with biliary tree / leakage into peritoneal cavity

Notes:

Cavernous hemangioma of liver most common benign liver tumor (78%); second most common liver tumor after metastases *Incidence:*1-4%; autopsy incidence 0.4-7.3%; increased with multiparityCause:? enlarging hamartoma present since birth,? true vascular neoplasm*Age:*rarely seen in young children; M:F = 1:5*Histo:*large vascular channels filled with slowly circulating blood; lined by single layer of mature flattened endothelial cells separated by thin fibrous septa; no bile ducts; thrombosis of vascular channels common resulting in fibrosis + hemorrhage + myxomatous degeneration + calcifications*Associated with:*(1) Hemangiomas in other organs(2) Focal nodular hyperplasia (3) Rendu-Osler-Weber disease ● asymptomatic if tumor small (50-70%) ● may present with spontaneous life-threatening hemorrhage if large (5%) ● hepatomegaly ● may enlarge during pregnancy ● abdominal discomfort + pain (from thrombosis in large hemangioma) ● Kasabach-Merritt syndrome (= hemangioma + thrombocytopenia) rareLocation:frequently peripheral / subcapsular in posterior right lobe of liver; 20% are pedunculated; multiple in 10-20%Size:<4 cm (90%); >10 cm = giant cavernous hemangioma√ may have central area of fibrosis = areas of nonenhancement / nonfilling / cystic space (occurrence increases with age)√ calcifications (phleboliths / septal calcifications) are extremely uncommonUS: √ uniformly hyperechoic (60-70%) mass due to multiple interfaces created by blood-filled spaces separated by fibrous septa√ inhomogeneous hypoechoic mass (up to 40%) in larger hemangiomas with well-defined thick / thin echogenic lobulated border due to hemorrhagic necrosis, scarring, myxomatous change centrally√ homogenous (58-73%) / heterogeneous (fibrosis, thrombosis, hemorrhagic necrosis)√ hypoechoic center possible√ may show acoustic enhancement (37-77%)√ unchanged in size / appearance (82%) on 1-to-6-year follow-up√ no Doppler signals / signals with peak velocity of <50 cm/secCT (combination of precontrast images, good bolus, dynamic scanning): √ well-circumscribed spherical / ovoid low-density mass√ may have areas of higher / lower density within mass√ typical pattern of low density on NECT + peripheral enhancement + complete fill-in on delayed images 3-30 minutes post IV bolus (55-89%)√ peripheral (72%) / central (in 8%) / diffuse dense (in 8%) enhancement√ complete (75%) / partial (24%) / no (2%) fill-in to isodensity in delayed phaseAngio (historical gold standard): √ dense opacification of well-circumscribed, dilated, irregular, punctate vascular lakes / puddles in late arterial + capillary phase starting at periphery in ring- / C-shaped configuration√ normal-sized feeders; AV shunting (very rare)√ contrast persistence late into venous phaseNUC (95% accuracy with SPECT): *Indication:*lesions >2 cm (detectable in 70-90%)√ delayed filling on Tc-99m labeled RBC scans (dose of 15-20 mCi) with increased activity on delayed images at 1-2 hours√ cold defect on sulfur colloid scansMR (90-95% accuracy): √ spheroid / ovoid (87%) mass with smooth well-defined lobulated margins (87%); no capsule√ homogeneous internal architecture if <4 cm, hypointense internal inhomogeneities if >4 cm (due to fibrosis)√ hypo- / isointense on T1WI; hyperintense "light bulb" appearance on T2WI (due to slow flowing blood) (DDx: hepatic cyst, hypervascular tumor, necrotic tumor, cystic neoplasm)√ uniform enhancement at 1 second in 40% of small hemangiomas <1.5 cm after gadolinium-DTPA√ peripheral nodular enhancement progressing centripetally with centrally uniform enhancement (50%) / persistent hypointensity (30%)Bx:may be biopsied safely provided normal liver is present between tumor + liver capsule√ nonpulsatile blood (73%)√ endothelial cells without malignancy (27%) *Prognosis:*no growth when <4 cm in diameter; giant cavernous hemangiomas may enlarge*Cx* (rare):(1)Spontaneous rupture (4.5%)(2)Abscess formation(3)Kasabach-Merritt syndrome (platelet sequestration)*DDx:*hypervascular malignant neoplasm / metastasis (quick homogeneous filling during arterial phase of small hemangiomas)

Notes:

Infantile Hemangioendothelioma Of Liver =INFANTILE HEPATIC HEMANGIOMA = CAPILLARY / CAVERNOUS HEMANGIOMA=most common benign hepatic tumor during first 6 months of life*Histo:*multiple anastomosing thick-walled vascular spaces similar to cavernous hemangioma lined by plump endothelial cells in single or (less often) multiple cell layers; areas of extramedullary hematopoiesis / thrombi; scattered bile ducts; involutional changes (infarction, hemorrhage, necrosis, scarring)*Classification:* (a)Hemangioendothelioma type 1 (more common): orderly proliferation of small blood vessels(b)Hemangioendothelioma type 2:more aggressive histologic pattern *DDx:*angiosarcoma(c)Cavernous hemangioma:dilated vascular spaces lined by flat endothelial cells ◊Relationship to adult cavernous hemangioma unknown!*Age at presentation:*<6 months in 85%, during 1st month in 33%, >1 year in 5%; M:F = 1:1.4-1:2 ● abdominal mass secondary to hepatomegaly ● cutaneous hemangiomas (9-45-87%) occur with multinodular form ● may present with high-output CHF secondary to AV shunts within tumor (8 -15-25%) ● **Kasabach-Merritt syndrome** (in 11%)=hemorrhagic diathesis due to platelet sequestration by tumor / disseminated intravascular coagulopathy; characterized by an association of hemangioma, or hemangioendothelioma, or angiosarcoma with thrombocytopenia and purpura) ● hemolytic anemiaSize:several mm up to 20 cm (average size of 3 cm)√ diffuse involvement of entire liver, rarely focal√ single mass (50%) / multiple masses (50%)√ enlargement of celiac + hepatic arteries + proximal aorta√ rapid decrease in aortic caliber below celiac trunk√ enlarged hepatic veins (increased venous flow)Plain film: √ fine speckled / fibrillary calcifications in 16% (DDx: hepatoblastoma, hamartoma, metastatic neuroblastoma)US: √ predominantly hypoechoic / complex / hyperechoic lesion√ multiple sonolucent areas (= enlarging vascular channels secondary to initial rapid growth) (DDx: mesenchymal hamartoma)OB-US: √ polyhydramnios + fetal hydropsCT: √ focal areas of low attenuation√ early peripheral enhancement (72%)√ variable delayed central enhancement (similar to cavernous hemangioma)MR: √ heterogeneous hypointense multinodular lesion on T1WI ± hyperintense areas of hemorrhage√ varying degrees of hyperintensity on T2WI (resembling adult hemangioma)√ decreasing signal intensity with fibrotic replacement on T2WINUC (sulfur colloid, tagged RBC): √ increased flow in viable portions of lesion during angiographic phase√ increased activity mixed with central photopenic areas (hemorrhage, necrosis, fibrosis) on delayed tagged RBC images√ photopenic defect on delayed sulfur colloid imagesAngio: √ enlarged, tortuous feeding arteries and stretched intrahepatic vessels√ hypervascular tumor with inhomogeneous stain; clusters of small abnormal vessels√ pooling of contrast material in sinusoidal lakes with rapid clearing through early draining veins (AV shunting) *Prognosis:*rapid growth in first 6 months followed by tendency to involute within 6-8 months; 32-75% survival rate in complicated cases*Cx:*(1)Congestive heart failure(2)Hemorrhagic diathesis(3)Obstructive jaundice(4)Hemoperitoneum (rupture of tumor) *Rx:*(1)No treatment if asymptomatic(2)Reduction in size with steroids / radiotherapy / chemotherapy(3)Embolization(4)Surgical resection / liver transplantation *DDx:*(1)Hepatoblastoma (>1 year of age, elevated a-fetoprotein, more heterogeneous)(2)Mesenchymal hamartoma (usually multilocular cystic mass)(3)Metastatic neuroblastoma (elevated catecholamines in urine, adrenal mass, nonenhancing multiple liver masses)

Notes:

Acute Hepatitis ● markedly elevated AST + ALT ● increase in serum-conjugated bilirubinUS: √ diffusely decreased parenchymal echogenicity√ increased brightness of portal triads ("starry sky" pattern) = centrilobular pattern (DDx: leukemic infiltrate, diffuse lymphomatous involvement, toxic shock syndrome)√ edema of gallbladder fossa√ thickening + increase in echogenicity of fat within falciform ligament, ligamentum venosum, porta hepatis, periportal connective tissue

Viral Markers of Hepatitis

Virus	Tests	Interpretation
HAV	Anti-HAV IgM	acute hepatitis (can remain positive for >1 year)
	Anti-HAV IgG	past hepatitis, lifelong immunity
HBV	HBsAg	acute / chronic disease
	Anti-HBc IgM	acute infection (if titer high); chronic infection (if titer low)
	Anti-HBc IgG	past / recent HBV contact (may be only serum indicator of past infection)
	HBe	active viral replication
	Anti-HBe	low / absent replicative state (typically present in long-standing HBV carriers)
	Anti-HBs	imunity after vaccination
	HBV-DNA	active viral replication
HCV	Anti-HCV	past / current infection
	RIBA	test for various viral components
	HCV-RNA	active viral replication
HDV	Anti-HDV IgM	acute / chronic infection
	Anti-HDV IgG	chronic infection (if titer high + IgM positive); past infection if titer low + IgM negative)
	HDV-RNA	active viral replication
HEV	Anti-HEV IgM	acute hepatitis
	Anti-HEV IgG	past hepatitis
	HEV-RNA	viral replication

Notes:

Chronic Hepatitis =process present for at least 6 months*Diseases:*autoimmune hepatitis; hepatitis B, C, D; cryptic hepatitis; chronic drug hepatitis; primary biliary cirrhosis; primary sclerosing cholangitis; Wilson disease; a-1-antitrypsin deficiencyUS: √ increased liver echogenicity√ coarsening of parenchymal texture√ silhouetting of portal vein walls = loss of definition of portal venules√ NO sound attenuation*Cx:*cirrhosis (10% for hepatitis B; 20-50% for hepatitis C)

Notes:

HEPATOBLASTOMA

*Incidence:*3rd most common abdominal tumor in children; most frequent malignant hepatic tumor in children (51%)*Incidence increased with:*hemihypertrophy, Beckwith syndrome *Histo:*(a)epithelial type = small cells resembling embryonal / fetal liver(b)mixed type = epithelial cells + mesenchymal cells (osteoid, cartilaginous, fibrous tissue)*Age:*<3 years; <18 months (in 50%); peak age between 18 and 24 months; range from newborn to 15 years; M:F = 2:1 ● upper abdominal mass, weight loss, nausea, vomiting ● precocious puberty (production of endocrine substances) ● persistently + markedly elevated a-fetoprotein (66%)Metastases to:lung (frequent)Location:right lobe of the liver√ usually solitary mass with an average size of 10-12 cm√ coarse calcifications / osseous matrix (12-30%)US: √ large heterogeneous echogenic mass, sometimes with calcifications, occasionally cystic areas (necrosis / extramedullary hematopoiesis)CT: √ hypointense tumor with peripheral rim enhancementMR: √ inhomogeneously hypointense on T1WI with hyperintense foci (hemorrhage)√ inhomogeneously hyperintense with hypointense bands (fibrous septa) on T2WINUC: √ photopenic defectAngio: √ hypervascular mass with dense stain√ marked neovascularity; NO AV-shunting√ vascular lakes may be present√ avascular areas (secondary to tumor necrosis)√ may show caval involvement (= unresectable) *Prognosis:*60% resectable; 75% mortality; better prognosis than hepatoma; better prognosis for epithelial type than mixed type*DDx:*hemangioendothelioma (fine granular calcifications), metastatic neuroblastoma, mesenchymal hamartoma, hepatocellular carcinoma (>5 years of age, no calcifications)

Notes:

HEPATOCELLULAR CARCINOMA

=HEPATOMA = most frequent primary visceral malignancy in the world; 80-90% of all primary liver malignancies; 2nd most frequent malignant hepatic tumor in children (39%) after hepatoblastoma*Incidence:*(a)in industrialized world: 0.2-0.8%(b)in sub-Saharan Africa, Southeast Asia, Japan, Greece, Italy: 5.5-20%*Peak age:*(a)industrialized world: 6th-7th decade; M:F = 2.5:1; fibrolamellar subtype (in 3-10%) below age 40 years(b)high incidence areas: 30-40 years;M:F = 5:1 (c)in children: >5 years of age; M:F = 4:3*Etiology:* 1.Cirrhosis (60-90%)*Latent period:*8 months to 14 years from onset of cirrhosis*Incidence of HCC:* -44% in macronodular (= postnecrotic) cirrhosis due to hepatitis B virus, alcoholism, hemochromatosis-6% in micronodular cirrhosis due to alcoholism5% of alcoholic cirrhotics develop HCC!(a)alcohol(c)cardiac(b)hemochromatosis(d)biliary atresia2.Chronic hepatitis B / C; 12% develop HCC3.Carcinogens(a)aflatoxin (b) siderosis (c) thorotrast(d)oral contraceptives / anabolic androgens4.Inborn errors of metabolism(a)a-1-antitrypsin deficiency(b)galactosemia(c)type I glycogen storage disease (von Gierke)(d)Wilson disease(e)tyrosinosis*mnemonic:*"WHAT causes HCC?"**W**ilson disease **H**emochromatosis **A**lpha-1-antitrypsin deficiency **T**yrosinosis **H**epatitis **C**irrhosis (alcoholic, biliary, cardiac) **C**arcinogens (aflatoxin, sex hormones, thorotrast) *Histo:*HCC cells resemble hepatocytes in appearance + structural pattern (trabecular, pseudoglandular = acinar, compact, scirrhous);(a)expansive encapsulated HCC: collapsed portal vein branches at capsule(b)infiltrative nonencapsulated HCC: portal venules communicate with tumoral sinusoids = often invasion of portal ± hepatic veinsGROWTH PATTERN: (a)solitary massive (27-50-59%):bulk in one (most often right) lobe with satellite nodules (b)multicentric small nodular (15-25%):small foci of usually <2 cm (up to 5 cm) in both hepatic lobes (c)diffuse microscopic (10 - 15-26%):tiny indistinct nodules closely resembling cirrhosis Vascular supply:hepatic artery, portal vein in 6% ● elevated a-fetoprotein (75-90%), negative in cholangiocarcinoma ● elevated liver function tests ● persistent RUQ pain, hepatomegaly, ascites ● fever, weight loss, malaise ● Paraneoplastic syndromes:(a)sexual precocity / gynecomastia(b)hypercholesterolemia(c)erythrocytosis (tumor produces erythropoietin)(d)hypoglycemia(e)hypercalcemia(f)carcinoid syndromeMetastases to: lung (most common = 8%), adrenal, lymph nodes, bone√ portal vein invasion (25-33-48%)√ arterioportal shunting (4-63%)√ invasion of hepatic vein (16%) / IVC (= Budd-Chiari syndrome)√ occasionally invasion of bile ducts√ calcifications in ordinary HCC (2-9-25%); however, common in fibrolamellar (30-40%) and sclerosing HCC √ hepatomegaly and ascites√ tumor fatty metamorphosis (2-17%)NUC: √ Sulfur colloid scan: single cold spot (70%), multiple defects (15-20%), heterogeneous distribution (10%)√ Tc-HIDA scan: cold spot / atypical uptake in 4% (delayed images)√ Gallium-scan: avid accumulation in 70-90% (in 63% greater, in 25% equal, in 12% less uptake than liver)CT (sensitivity of 63% in cirrhosis, 80% without cirrhosis): √ hypodense mass / rarely isodense / hyperdense in fatty liver√ dominant mass with satellite nodules√ mosaic pattern = multiple nodular areas with differing attenuation on CECT (up to 63%)√ diffusely infiltrating neoplasm√ encapsulated HCC = circular zone of radiolucency surrounding the mass (12-32-67%)False-positive:confluent fibrosis, regenerative noduleBiphasic CECT: √ enhancement during hepatic arterial phase (80%)√ decreased attenuation during portal venous phase with inhomogeneous areas of contrast accumulation√ isodensity on delayed scans (10%)√ thin contrast-enhancing capsule (50%) due to rapid washout√ wedge-shaped areas of decreased attenuation (segmental / lobar perfusion defects due portal vein occlusion by tumor thrombus)CT with intraarterial ethiodol injection: √ hyperdense mass detectable as small as 0.5 cm US (86-99% sensitivity, 90-93% specificity, 50-94% accuracy):√ hyperechoic HCC (13%) due to fatty metamorphosis or marked dilatation of sinusoids√ hypoechoic HCC (26%) due to solid tumor√ HCC of mixed echogenicity (61%) due to nonliquefactive tumor necrosis√ Doppler peak velocity signals >250 cm/secMR: √ hypointense (50%) / iso- to hyperintense (with fatty metamorphosis) on T1WI√ ring sign = well-defined hypointense capsule on T1WI (24-44%), double layer of inner hypointensity (fibrous tissue) + outer hyperintensity (compressed blood vessels + bile ducts) on T2WI in expansive type of HCC √ mildly hyperintense on T2WI√ Gd-DTPA enhancement peripherally (21%) / centrally (7%) / mixed (10%) / no enhancement (21%)√ improved lesion detectability after intravenous administration of superparamagnetic iron oxideAngio: √ "thread and streaks" = linear parallel vascular channels coursing along portal venous radicles seen with portal venous involvement√ in differentiated HCC: enlarged arterial feeders, coarse neovascularity, vascular lakes, dense tumor stain, arterioportal shunts√ in anaplastic HCC: vascular encasement, fine neovascularity, displacement of vessels + corkscrew-like vessels of cirrhosis*Prognosis:*>90% overall mortality; 17% resectability rate; 6 months average survival time; 30% 5-year survival time*Cx:*spontaneous rupture (in 8%)*Rx:*(1) Resection (2) I-131 antiferritin IgG (remission rate >40% up to 3 years)*DDx:*hepatocarcinoma, cholangiocarcinoma, focal nodular hyperplasia, hemangioma, hepatic adenoma

Fibrolamellar Hepatocellular Carcinoma

Notes:

Fibrolamellar Hepatocellular Carcinoma NO underlying cirrhosis or known risk factors ● a-fetoprotein negative*Age:*5-35 (mean 23) years; M:F = 1:1*Path:*well-circumscribed strikingly desmoplastic tumor with calcifications + fibrous central scar*Histo:*hepatocyte-like cells with granular eosinophilic cytoplasm growing in sheets / cords / trabeculae separated by broad bands of fibrous stroma arranged in parallel lamellae√ partially / completely encapsulated solitary mass 4-17 cm in diameter√ prominent central fibrous scar (45-60%)√ central stellate / trabecular calcifications (30-55%)CT: √ mass of low attenuation + varying degrees of enhancementMRI: √ homogeneous mildly hypointense tumor on T1WI; slightly hyperintense on T2WI√ hypointense central scar on T1WI + T2WIAngio: √ dense tumor stain without arterioportal shunting / neovascularity*Prognosis:*48% resectability rate; 32 months average survival time; 63% 5-year survival time*DDx:*focal nodular hyperplasia (hyperintense central scar on T2WI)

Notes:

HYPERPLASTIC CHOLECYSTOSIS
=variety of degenerative + proliferative changes of gallbladder wall characterized by hyperconcentration, hyperexcitability, and hyperexcretion*Incidence:*30-50% of all cholecystectomy specimens; M:F = 1:6

Cholesterolosis Adenomyomatosis Of Gallbladder

Notes:

Cholesterolosis = abnormal deposits of cholesterol esters in macrophages within lamina propria (foam cells) + in mucosal epithelium 1.STRAWBERRY GALLBLADDER=LIPID CHOLECYSTITIS = CHOLESTEROSIS=planar form = seedlike patchy / diffuse thickening of the villous surface pattern (disseminated micronodules)*Associated with:* cholesterol stones in 50-70% ● not related to serum cholesterol level radiologically not demonstrable2.CHOLESTEROL POLYP (90%)= polypoid form =abnormal deposit of cholesterol ester producing a villouslike structure covered with a single layer of epithelium and attached via a delicate stalk Most common fixed filling defect of GBLocation:commonly in middle 1/3 of gallbladder multiple small filling defects <10 mm in diameter *DDx:*papilloma, adenopapilloma, inflammatory granuloma

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Notes:

Adenomyomatosis Of Gallbladder =increase in number + height of mucosal folds*Histo:*hyperplasia of epithelial + muscular elements with mucosal outpouching of epithelium-lined cystic spaces into (46%) or all the way through (30%) a thickened muscular layer as tubules / crypts / saccules (= intramural diverticula= Rokitansky-Aschoff sinus); develop with increasing age*Incidence:*5% of all cholecystectomies*Age:*>35 years; M:F = 1:3*Associated with:*(1)Gallstones in 25-75%(2)Cholesterolosis in 33%A.Generalized form= **Adenomyomatosis** √ "pearl necklace gallbladder" = tiny extraluminal extensions of contrast on OCG (enhanced after contraction)B.Segmental formcompartmentalization most often in neck / distal 1/3 C.Localized form in fundus= **Adenomyoma** √ smooth sessile mass in GB fundus=solitary adenomyoma + extraluminal diverticula-like formationD.Annular form √ "hourglass" configuration of GB with transverse congenital septum

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Notes:

HYPOSPLENISM

=no uptake of Tc-99m sulfur colloidA.ANATOMIC ABSENCE OF SPLEEN1.Congenital asplenia = Ivemark syndrome2.SplenectomyB.FUNCTIONAL ASPLENIA=spleen anatomically present without uptake of Tc-99m sulfur colloid1.Circulatory disturbances:occlusion of splenic artery / vein, hemoglobinopathies (sickle cell disease, hemoglobin-SC disease, thalassemia), polycythemia vera, idiopathic thrombocytopenic purpura 2.Altered RES activity:thorotrast irradiation, combined splenic irradiation + chemotherapy, replacement of RES by tumor / infiltrate, splenic anoxia (cyanotic congenital heart disease), sprue 3.Autoimmune disease*Cx:*children at risk for pneumococcal pneumonia (liver partially takes over immune response later in life)C.FUNCTIONAL ASPLENIA + SPLENIC ATROPHYUlcerative colitis, Crohn disease, celiac disease, tropical sprue, dermatitis herpetiformis, thyrotoxicosis, idiopathic thrombocytopenic purpura, thorotrast D.FUNCTIONAL ASPLENIA + NORMAL / LARGE SPLEENSarcoidosis, amyloidosis, sickle cell anemia (if not infarcted) ● RBC (acanthocytes, siderocytes) ● lymphocytosis, monocytosis ● Howell-Jolly bodies (intraerythrocytic inclusions) ● thrombocytosis√ spleen not visualized on Tc-99m sulfur colloid√ Tc-99m heat-damaged RBCs / In-111 labeled platelets may demonstrate splenic tissue if Tc-99m sulfur colloid does not*Cx:*increased risk of infection (pneumococcus, meningococcus, influenza)

Notes:

LIPOMA OF LIVER

Extremely rare ● asymptomatic*May be associated with:* tuberous sclerosisUS: √ echogenic mass√ striking acoustic refraction (sound velocity in soft tissue 1,540 m/sec, in fat 1,450 m/sec)*Prognosis:*no malignant potential

Notes:

LIVER TRANSPLANT
Indication:(a)in childhood: biliary atresia (52%), acute fulminant hepatic failure (11%), a-1-antitrypsin deficiency (9%), cryptogenic cirrhosis (6%), chronic active hepatitis (4%) NORMAL POSTTRANSPLANT FINDINGS (1)Periportal edema (21%)*Cause:*? lymphedema in early posttransplantation period, occasionally associated with acute rejection√ "periportal collar" of low attenuation on CT + hyperechogenicity on US(2)Fluid collection around falciform ligament (11%)

Vascular Complications In Liver Transplant Parenchymal Complications In Liver Transplant Biliary Complications In Liver Transplant

Notes:

Vascular Complications In Liver Transplant 1.Anastomotic narrowing of IVC / portal vein◊Discrepancies in caliber between donor + recipient vessel have no pathologic significance! ● venous hypertension of lower part of body ● portal hypertension2.Thrombosis of IVC / portal vein3.Hepatic artery stenosis (11-13%)Location:at / near anastomotic site√ marked focal increase in velocity >200-300 cm/sec + poststenotic turbulence√ intrahepatic tardus et parvus waveform = slowed systolic acceleration time (SAT >0.08 sec) distal to stenosis (73% sensitive)√ diminished pulsatility (RI <0.5) due to ischemia4.Hepatic artery thrombosis (3-9-16% in adults, 9-19-42% in children)Time of onset:usually within first 2 months ● Three types of clinical presentation:(1)fulminant hepatic necrosis + rapid deterioration(2)bile leak, bile peritonitis, bacteremia, sepsis(3)relapsing bacteremia√ absence of hepatic artery flowFP Doppler (10%): low flow state, small vessel size, severe liver edema (in first 72 hours after transplantation, viral hepatitis, rejection) FN Doppler:arterial collaterals√ multiple hypoechoic lesions in liver periphery(= infarcts) *Mortality:*50-58%5.Hepatic artery pseudoaneurysm

Notes:

Parenchymal Complications In Liver Transplant 1.Rejection Can ONLY be diagnosed with liver biopsy!2.Infarction (10%) may calcify may liquefy developing into intrahepatic biloma3.Graft infection

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Notes:

Biliary Complications In Liver Transplant *Incidence:*13-25%1.Biliary obstruction(a)stricture at anastomosis(b)tension mucocele of allograft cystic duct remnant √ extrinsic mass compressing CHD√ fluid collection adjacent to CHD(c)intrahepatic strictureas complication of arterial ischemia 2.Bile leak(a)anastomotic site:70% within 1st month(b)T-tube exit site:50% within 10 days(c)bile duct necrosis (hepatic artery occlusion)◊The intrahepatic biliary epithelium is perfused solely by the hepatic artery!(d)after liver biopsy(e)common hepatic duct leak*Incidence:*4.3-23%

Notes:

Radiology Review Manual
fourth edition

LYMPHOMA OF LIVER

A.PRIMARY LYMPHOMA (rare)√ solid solitary mass B.SECONDARY LYMPHOMA (common)autoptic incidence of liver involvement: 60% in Hodgkin disease 50% in non-Hodgkin lymphoma Pattern: (a)infiltrative diffuse (most common): no alteration in hepatic architecture(b)focal nodular: detectable by cross-sectional imaging(c)combination of diffuse + nodular (3%)Detection rate (for CT, MRI): <10%

Notes:

MACROCYSTIC ADENOMA OF PANCREAS
= MUCINOUS CYSTIC NEOPLASM = MUCINOUS CYSTADENOMA / CYSTADENOCARCINOMA = thick-walled uni- / multilocular low-grade malignant tumor composed of large mucin-containing cystic spaces *Frequency:*10% of pancreatic cysts; 1% of pancreatic neoplasms*Mean age:*50 years (range of 20-95 years); in 50% between 40-60 years;M:F = 1:19 *Path:*large smooth round / lobulated multiloculated cystic mass encapsulated by a layer of fibrous connective tissue*Histo:*similar to biliary and ovarian mucinous tumors; cysts lined by tall columnar, mucin-producing cells subtended by a densely cellular mesenchymal stroma (reminiscent of ovarian stroma), often in papillary arrangement, lack of cellular glycogen(a)mucinous cystadenoma(b)mucinous cystadenocarcinoma = stratified papillary epithelium◊All mucinous cystic neoplasms should be considered as malignant neoplasms of low-grade malignant potentialLocation:often in pancreatic tail (90%) / body, infrequently in head ● asymptomatic ● abdominal pain, anorexia√ well-demarcated thick-walled mass of 2-36 (mean 10-12) cm in diameter√ multi- / unilocular large cysts >2 cm with thin septa <2 mm ◊A tumor with <6 cysts of >2 cm in diameter is in 93-95% a mucinous cystic neoplasm!√ solid papillary excrescences protrude into the interior of tumor (sign of malignancy)√ amorphous discontinuous peripheral mural calcifications (10-15%)√ hypovascular mass with sparse neovascularity√ vascular encasement and splenic vein occlusion may be present√ great propensity for invasion of adjacent organsUS: √ cysts may contain low-level echoesCT: √ internal septations may not be visualized without contrast enhancement√ cysts with attenuation values of water; may have different levels of attenuation within different cystic cavities√ enhancement of cyst wallsAngio: √ predominantly avascular mass√ cyst wall + solid components may demonstrate small areas of vascular blush + neovascularity√ displacement of surrounding arteries + veins by cystsMetastases: √ round thick-walled cystic lesions in liver*Prognosis:*invariable transformation into cystadenocarcinoma*Rx:*complete surgical excision (5-year survival rate of 74-90%)*DDx:* (1)Pseudocyst: inflammatory changes in peripancreatic fat, pancreatic calcifications, temporal evolution, history of alcoholism, elevated levels of amylase(2)Lymphangioma / hemangioma(3)Variants of ductal adenocarcinoma:(a)mucinous colloid adenocarcinoma / ductectatic mucinous tumor of pancreas = mucin-hypersecreting carcinoma(b)papillary intraductal adenocarcinoma(c)adenosquamous carcinoma: squamous component predisposes to necrosis + cystic degeneration(d)anaplastic adenocarcinoma: lymphadenopathy + metastases at time of presentation(4)Solid and cystic papillary epithelioid neoplasm: hemorrhagic cystic changes in 20%(5)Cystic islet cell tumor: hypervascular component(6)Cystic metastases: history of malignant disease(7)Atypical serous cystadenoma: smaller tumor with greater number of smaller cysts(8)Sarcoma(9)Infection: amebiasis, Echinococcus multilocularis

Notes:

MESENCHYMAL HAMARTOMA OF LIVER
=rare developmental cystic liver tumor*Histo:*disordered arrangement of primitive fluid-filled mesenchyme, bile ducts, hepatic parenchyma; stromal / cystic predominance with cysts of a few mm up to 14 cm in size; no capsule*Age peak:*15-24 months (range from newborn to 19 years); M:F = 2:1 ● slow progressive abdominal enlargement ● ± respiratory distress and lower extremity edemaLocation:right lobe:left lobe = 6:1; 20% pedunculated √ 16 cm average tumor size (range of 5-29 cm)√ grossly discernible cysts in 80%US: √ multiple rounded cystic areas on an echogenic background√ may appear solid in younger infant (when cysts are still small)CT: √ multiple lucencies of variable size + attenuationMR: √ varying signal intensity (varying concentrations of protein in cystic predominance type) / hypointense on T1WI (mesenchymal predominance type)√ marked hyperintensity of cystic locules / hypointense fibrosis on T2WINUC: √ one / more areas of diminished uptake on sulfur colloid scanAngio: √ hypovascular mass√ may show patchy areas of neovascularity√ enlarged irregular tortuous feeding vessels

Notes:

METASTASES TO LIVER

Incidence: liver is most common metastatic site after regional lymph nodes; incidence of metastatic carcinoma 20 x greater than primary carcinoma; metastases represent 22% of all liver tumors in patients with known malignancy; most common malignant lesion of the liver Enhancement characteristics compared with normal liver: √ lesion enhancement during arterial phase (metastases are supplied by hepatic artery)√ less enhancement during portal venous phase (metastases have a negligible portal venous supply)√ extracellular space agents accumulate more in tumor tissue (metastases have a larger interstitial space)*Organ of origin:*colon (42%), stomach (23%), pancreas (21%), breast (14%), lung (13%) ● hepatomegaly (70%) ● abnormal liver enzymes (50-75%) Location:both lobes (77%), right lobe (20%), left lobe (3%)Number:multiple (50-98%), solitary (2%)Size:>33% smaller than 2 cm √ involvement of liver + spleen typical in lymphoma + melanomaNUC:80-95% sensitivity in lesions >1.5 cm; lesions <1.5 cm are frequently missed; sensitivity increases with metastatic deposit size, peripheral location, and use of SPECTNECT:important for hypervascular tumors (eg, renal cell carcinoma, carcinoid, islet cell tumors) which may be obscured by CECTCECT: *Technique:* optimal is bolus technique with dynamic incremental scanning; sensitivity is decreased relative to NCCT if scans are obtained during equilibrium phase of contrast administration √ circumferential bead- or bandlike enhancement during arterial phase + peripheral washout on delayed images √ no (35%), peripheral (37%), mixed (20%), central (8%) enhancement√ complete isodense fill-in on delayed scans in 5% (DDx: hemangioma)◊CT-sensitivity 88-90%; specificity 99%; lesions of approx. 1 cm can usually be detected! CT-Angiography (most sensitive imaging modality): *Indication:*patients with potentially resectable isolated liver metastases / preoperative to partial hepatectomy for detection of additional metastases (additional lesions detected in 40-55%)(1)CT arteriography = angiography catheter in hepatic artery, detects lesions by virtue of increased enhancement(2)CT arterial portography = angiography catheter in SMA, detects hypodense lesions on a background of increased enhancement of normal surroundings in portal venous phase CT-delayed iodine scanning: =CT performed 4-6 hours following administration of 60 mg iodine results in detection of additional lesions in 27%

Calcified Liver Metastases Hypervascular Liver Metastases Hemorrhagic Liver Metastases Echogenic Liver Metastases Liver Metastases of Mixed Echogenicity Cystic Liver Metastases Echopenic Liver Metastases

Notes:

Calcified Liver Metastases *Incidence:*2-3%1. Mucinous carcinoma of GI tract (colon, rectum, stomach)2. Endocrine pancreatic carcinoma3. Leiomyosarcoma, osteosarcoma4. Malignant melanoma5. Papillary serous ovarian cystadenocarcinoma6. Lymphoma7. Pleural mesothelioma8. Neuroblastoma9. Breast cancer10. Medullary carcinoma of the thyroid11. Renal cell carcinoma 12. Lung carcinoma13. Testicular carcinoma *mnemonic for mucinous adenocarcinoma:* "COBS" **C**olon carcinoma **O**varian carcinoma **B**reast carcinoma **S**tomach carcinoma

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Notes:

Hypervascular Liver Metastases 1.Renal cell carcinoma2.Carcinoid tumor3.Colonic carcinoma4.Choriocarcinoma5.Breast carcinoma6.Melanoma7.Pancreatic islet cell tumor8.Ovarian cystadenocarcinoma9.Sarcomas10.Pheochromocytoma *mnemonic:*"CHIMP"**C**arcinoid, **C**olon cancer **H**ypernephroma **I**slet cell carcinoma **M**elanoma **P**heochromocytoma

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Notes:

Hemorrhagic Liver Metastases *mnemonic:*"CT BeComes MR"**C**olon carcinoma **T**hyroid carcinoma **B**reast carcinoma **C**horiocarcinoma **M**elanoma **R**enal cell carcinoma

Notes:

Echogenic Liver Metastases *Incidence:*25%1.Colonic carcinoma (mucinous adenocarcinoma) 54%2.Hepatoma 25%3.Treated breast carcinoma 21%

Notes:

Liver Metastases of Mixed Echogenicity *Incidence:*37.5%1.Breast cancer31%2.Rectal cancer20%3.Lung cancer17%4.Stomach cancer14%5.Anaplastic cancer11%6.Cervical cancer5%7.Carcinoid1%

Notes:

Cystic Liver Metastases 1.Mucinous ovarian carcinoma2.Colonic carcinoma3.Sarcoma4.Melanoma5.Lung carcinoma6.Carcinoid tumor*mnemonic:*"LC GOES"**L**eiomyosarcoma (and other sarcomas) **C**horiocarcinoma **G**astric carcinoma **O**varian carcinoma **E**ndometrial carcinoma **S**mall cell carcinoma

Notes:

Echopenic Liver Metastases *Incidence:*37.5%1.Lymphoma44%2.Pancreas36%3.Cervical cancer20%4.Lung (adenocarcinoma)5.Nasopharyngeal cancer
*Rx:*Exclusion criteria for metastasectomy:(1)advanced stage of primary tumor(2)>4 metastases(3)extrahepatic disease(4)<30% normal liver tissue / function available after resection

Notes:

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METASTASES TO PANCREAS
*Frequency:*3-10% (autopsy)Organ of origin:renal cell carcinoma (30%), bronchogenic carcinoma (23%), breast carcinoma (12%), soft-tissue sarcoma (8%), colonic carcinoma (6%), melanoma (6%)√ solitary (78%) / multiple (17%) ovoid masses with discrete smooth margins√ diffuse pancreatic enlargement (5%)CECT: √ heterogeneously (60%) / homogeneously (17%) hyperattenuating relative to pancreas√ hypoattenuating relative to pancreas (20%)√ isoattenuating relative to pancreas (5%)Concomitant intraabdominal metastases to: liver (36%), lymph nodes (30%), adrenal glands (30%) *DDx:*ductal pancreatic adenocarcinoma (uniformly nonenhancing mass, encasement of vessels)

Notes:

MICROCYSTIC ADENOMA OF PANCREAS

=SEROUS CYSTADENOMA = GLYCOGEN-RICH CYSTADENOMA=benign lobulated neoplasm composed of innumerable small cysts (1-20 mm) containing proteinaceous fluid separated by thin connective tissue septa*Incidence:*approximately 50% of all cystic pancreatic neoplasms*Histo:*cyst walls lined by cuboidal / flat glycogen-rich epithelial cells derived from centroacinar cells of pancreas (DDx: lymphangioma), thin fibrous pseudocapsule*Age:*34-88 years; mean age 65 years;82% over 60 years of age; M:F = 1:4 *Associated with:*von Hippel-Lindau syndrome ● pain, weight loss, jaundice ● palpable mass Location:any part of pancreas affected, slight predominance for head√ well-demarcated lobulated mass 1-25 (mean 5) cm in diameter with smooth / nodular contour√ innumerable small <2 cm cysts; uncommonly few large cysts (in <5%) / cyst up to 8 cm in diameter√ prominent central stellate scar (CHARACTERISTIC)√ amorphous central calcifications (in 33% on plain film) in dystrophic area of stellate central scar ("sunburst")√ pancreatic duct + CBD may be displaced, encased, or obstructedUS: √ solid predominantly echogenic mass with mixed hypoechoic + echogenic areas CT: √ attenuation values close to water√ contrast enhancementMR: √ delayed enhancement of scar on contrast-enhanced FLASH imagesAngio: √ hypervascular mass with dilated feeding arteries, dense tumor blush, prominent draining veins, neovascularity, occasional AV shunting, NO vascular encasement*Prognosis:*no malignant potential*Rx:*surgical excision / follow-up examinations

Notes:

MILK OF CALCIUM BILE
=LIMY BILE = CALCIUM SOAP = precipitation of particulate material with high concentration of calcium carbonate, calcium phosphate, calcium bilirubinate*Associated with:*chronic cholecystitis + gallstone obstruction of cystic duct√ diffuse opacification of GB lumen with dependent layering√ usually functionless GB on oral cholecystogramUS: √ intermediate features between sludge + gallstones

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Notes:

MIRIZZI SYNDROME
=extrinsic right-sided compression of common hepatic duct by large gallstone impacted in cystic duct / gallbladder neck / cystic duct remnant; accompanied by chronic inflammatory reaction*Frequently associated with:* formation of fistula between gallbladder and common hepatic duct ● jaundice√ normal CBD below level of impacted stone√ TRIAD:(1)gallstone impacted in GB neck(2)dilatation of bile ducts above level of cystic duct(3)smooth curved segmental stenosis of CHDCholangiography: √ partial obstruction of CHD due to external compression on lateral side of duct / eroding stone*DDx:*lymphadenopathy, neoplasm of GB / CHD

Notes:

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MULTIPLE BILE DUCT HAMARTOMA
=VON MEYENBURG COMPLEX*Incidence:*0.15-2.8% of autopsies*Etiology:*failure of involution of embryonic bile ducts*Histo:*cluster of proliferated bile ducts lined by single layer of cuboidal cells embedded in fibrocollagenous tissue with single ramified lumen, communication with biliary system usually obliterated*Associated with:*polycystic liver diseaseSize:0.1-10 mmCT: √ multiple irregular hypodense lesions of up to 10 mmUS: √ multiple small cysts / echogenic areas (if size not resolved) up to 10 mm ± comet-tail artifactAngio: √ multiple areas of abnormal vascularity in form of small grapelike clusters persisting into venous phase*DDx:*metastatic liver disease

Notes:

MULTIPLE ENDOCRINE NEOPLASIA
= MEN = MULTIPLE ENDOCRINE ADENOMAS (MEA) = familial autosomal dominant adenomatous hyperplasia characterized by neoplasia of more than one endocrine organ *Theory:*cells of involved principal organs originate from neural crest and produce polypeptide hormones in cytoplasmic granules which allow **a**mine **p**recursors **u**ptake and **d**ecarboxylation = APUD cellsreminder:TypeI= Wermer syndromePPPTypeII= Sipple syndrome (Type IIA)PMPTypeIII= Mucosal neuroma syndrome (Type IIB)MPM

MEAType IType IIType III			
Pituitary adenoma	+Parathyroid adenoma	++Medullary thyroid carcinoma	++Pancreatic island cell tumor+Pheochromocytoma++Ganglioneuromatosis+
MEN I Syndrome	MEN II Syndrome	MEN III Syndrome	

Notes:

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MEN I Syndrome =WERMER SYNDROME=autosomal dominant trait with high penetrance;M:F = 1:1*Cause:*genetic defect in chromosome 11*Organ involvement:* 1.Parathyroid hyperplasia (97%): multiglandular2.Pancreatic islet cell tumor (30-80%):◊Likely multiple + behaving malignant!◊Primary cause of morbidity + mortality!(a)gastrinoma = Zollinger-Ellison syndrome (most common type, in 50%), usually multicentric(b)insulinoma(c)VIPoma = WDHH-syndrome (watery diarrhea, hypokalemia, hypochlorhydria)3.Anterior pituitary gland tumor (15-50%):(a)nonfunctioning (b)prolactin, growth hormone, corticotropin, TSH4.Combination of parathyroid + pancreas + pituitary involvement (40%)5.Adrenocortical hyperplasia (up to 33-40%)6.Carcinoid7.Lipoma ● usually asymptomatic*May be associated with:* thyroid tumor (20%), thymoma, buccal mucosal tumor, colonic polyposis, Ménétrier disease

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Notes:

MEN II Syndrome =SIPPLE DISEASE = MEN Type IIA*Organ involvement:* 1.Medullary carcinoma of thyroid2.Pheochromocytoma: bilateral in 50%;malignant in 3% diagnosed before (in 10%) / after detection (in 17%) of medullary thyroid carcinoma 3.Parathyroid neoplasia ● ± hyperparathyroidism*May be associated with:*carcinoid tumors, Cushing disease

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Notes:

MEN III Syndrome =MUCOSAL NEUROMA SYNDROME = MEN Type IIB*Organ involvement:* 1.Medullary carcinoma of thyroid2.Pheochromocytoma3.Oral + intestinal neuroganglioneuromatosis◊Usually precedes the appearance of thyroid carcinoma + pheochromocytoma! ● long slender extremities (Marfanoid appearance) ● thickened lips (due to submucosal nodules) ● nodular deformity of tongue (mucosal neuromas of tongue often initially diagnosed by dentists) ● prognathism ● corneal limbus thickening ● constipation alternating with diarrhea@GI tract√ thickened / plaquelike colonic wall√ dilated colon with abnormal haustral markings√ alternating areas of colonic spasm + dilatation√ multiple submucosal neuromas throughout small bowel, may act as lead point for intussusception

Notes:

NEONATAL HEPATITIS

*Etiology:*CMV, hepatitis A/B, rubella, toxoplasmosis, spirochetes, idiopathic*Path:*multinucleated giant cells, bile ducts relatively free of bileNUC: *Technique:*often performed after pretreatment with phenobarbital (5 mg/kg x 5 days) to maximize hepatic function√ normal / decreased hepatic tracer accumulation√ prolonged clearance of tracer from blood pool√ bowel activity faint / delayed usually by 24 hours (best seen on lateral view; covering liver activity with lead shielding is helpful)√ gallbladder may not be visualized*Prognosis:*spontaneous remission*DDx:*biliary atresia

Notes:

PANCREAS DIVISUM
=most common anatomic variant of pancreas due to failure of fusion of the ventral and dorsal anlage at 8th week of fetal life with dorsal pancreatic duct (Santorini) draining through minor (accessory) papilla + ventral pancreatic duct (Wirsung) with CBD draining through major papilla*Prevalence:*4-9-14% in autopsy series;2-8% in ERCP series;3-7% in normal population;12-26% in patients with idiopathic recurrent pancreatitisHypothesis:relative / actual stenosis of minor papilla predisposes to nonalcoholic recurrent pancreatitis in dorsal segment ● clinical relevance continues to be debatedPancreatography: ONLY reliable means for diagnosis √ contrast injection into major papilla demonstrates only short ventral pancreatic duct with early arborization√ contrast injection into minor papilla fills dorsal pancreatic duct√ no communication between ventral + dorsal ductsCT: √ oblique fat cleft between ventral + dorsal pancreas (25%)√ failure to see union of dorsal + ventral pancreatic ducts (rare)

Notes:

PANCREATIC ACINAR CELL CARCINOMA

=rare neoplasm of exocrine origin*Age:*40-81 (mean 62) years; M:F = 86:14; 87% Caucasian ● increased serum lipase ± amylase ● syndrome of elevated lipase = ● disseminated subcutaneous + intraosseous fat necrosis (usually distal to knees / elbows) ● polyarthropathy ● skin lesions resembling erythema nodosum ● biliary obstruction distinctly uncommon√ lobulated well-defined mass of 2-15 cm in diameter√ thin enhancing capsule√ tumor necrosis usually present√ moderately vascular tumor + neovascularity + arterial and venous encasement*Prognosis:* median survival of 7-9 months *DDx:*(1)pancreatic adenocarcinoma (small, irregular, locally invasive, without capsule, biliary obstruction if located in head of pancreas)(2)Nonfunctioning islet-cell tumor(3)Microcystic cystadenoma(4)Solid and papillary epithelial neoplasm(5)Oncocytic tumor of pancreas

Notes:

PANCREATIC DUCTAL ADENOCARCINOMA

=DUCT CELL ADENOCARCINOMA (duct cells comprise only 4% of pancreatic tissue)*Incidence:*80 - 95% of nonendocrine pancreatic neoplasms; 5th leading cause of cancer death in the United States (27,000 per year)*Etiology:*alcohol abuse (4%), diabetes (2 x more frequent than in general population, particularly in females), hereditary pancreatitis (in 40%); cigarette smoking (risk factor 2 x)*Path:*scirrhous infiltrative adenocarcinoma with a dense cellularity + sparse vascularity*Mean age at onset:*55 years; peak age in 7th decade; M:F = 2:1 STAGEI= confined to pancreasII= + regional lymph node metastasesIII= + distant spread◊At presentation-65% of patients have advanced local disease / distant metastases-21% of patients have localized disease with spread to regional lymph nodes-14% of patients have tumor confined to pancreasExtension: (a)local extension beyond margins of organ (68%): posteriorly (96%), anteriorly (30%), into porta hepatis (15%), into splenic hilum (13%)(b)invasion of adjacent organs (42%):duodenum > stomach > left adrenal gland > spleen > root of small bowel mesenteryMetastases:liver (30-36%), regional lymph nodes >2 cm (15-28%), ascites from peritoneal carcinomatosis (7-10%), lungs (pulmonary nodules / lymphangitic), pleura, bone ● weight loss, anorexia, fatigue ● pain in hypochondrium radiating to back ● obstructive jaundice (75%): most frequent cause of malignant biliary obstruction ● new onset diabetes (25-50%), steatorrhea ● thrombophlebitis Location:pancreatic head (56-62%); body (26%); tail (12%)Size:2-10 cm (in 60% between 4-6 cm)UGI: √ "antral padding" = extrinsic indentation of the posteroinferior margin of antrum√ "Frostberg 3" sign = inverted 3 contour to the medial portion of the duodenal sweep√ spiculated duodenal wall + traction + fixation (neoplastic infiltration of duodenal mucosa / desmoplastic response)√ irregular / smooth nodular mass with ampullary carcinomaBE: √ localized haustral padding / flattening / narrowing with serrated contour at inferior aspect of transverse colon / splenic flexure√ diffuse tethering throughout peritoneal cavity (intraperitoneal seeding)CT (99% detection rate for dynamic CT scan; 89% in predicting nonresectability): √ pancreatic mass (95%) / diffuse enlargement (4%) / normal scan (1%)√ mass with central zone of diminished attenuation (75- 83%)√ pancreatic + bile duct obstruction without detectable mass (4%)√ duct dilatation (58%): 3/4 biductal, 1/10 isolated to one duct; dilated pancreatic duct (67%); dilated bile ducts (38%)√ atrophy of pancreatic body + tail (20%)√ calcifications (2%)√ postobstructive pseudocyst (11%)√ obliteration of retropancreatic fat (50%)√ thickening of celiac axis / SMA (invasion of perivascular lymphatics) in 60%√ dilated collateral veins (12%)√ thickening of Gerota fascia (5%)√ local tumor extension posteriorly, into splenic hilum, into porta hepatis (68%)√ contiguous organ invasion (duodenum, stomach, mesenteric root) in 42%US: √ hypoechoic pancreatic mass√ focal / diffuse (10%) enlargement of pancreas√ contour deformity of gland; rounding of uncinate process√ dilatation of pancreatic ± biliary ductMR (no diagnostic improvement over CT): √ hypointense lesion on fat-suppressed T1WI√ diminished enhancement on dynamic contrast imagesAngiography (70% accuracy): √ hypovascular tumor / neovascularity (50%)√ arterial encasement: SMA (33%), splenic artery (14%), celiac trunk (11%), hepatic artery (11%), gastroduodenal artery (3%), left renal artery (0.6%)√ venous obstruction: splenic vein (34%), SMV (10%)√ venous encasement: SMV (23%), splenic vein (15%), portal vein (4%)Cholangiography: √ "rat tail / nipplelike" occlusion of CBD√ nodular mass / meniscuslike occlusion in ampullary tumorsPancreatography (abnormal in 97%): √ irregular, nodular, rat-tailed, eccentric obstruction√ localized encasement with prestenotic dilatation√ acinar defect*Prognosis:* 10% 1-year survival, 2% 3-year survival, <1% 5-year survival; 14 months medial survival after curative resection, 8 months after palliative resection, 5 months without treatment; tumors resectable in only 8-15% at presentation, 5% 5-year survival rate after surgery *DDx:*focal pancreatitis, islet cell carcinoma, metastasis, lymphoma, normal variant

Notes:

PANCREATIC ISLET CELL TUMORS

*Origin:*embryonic neuroectoderm, derivatives of APUD (amine precursor uptake and decarboxylation) cell line arising from islet of Langerhans (APUDoma)*Prevalence:*1:1,000,000 population/year; isolated or part of MEN I syndrome (= Wermer syndrome)*Path:*(a)small tumor: solid well-demarcated(b)large tumor: cystic changes + necrosis + calcifications*Histo:*sheets of small round cells + numerous stromal vesselsAverage time from onset of symptoms to diagnosis is 2.7 years *Classification:*(a)functional (85%)(b)nonfunctional (below threshold of detectability) / hypofunctionalMetastases: in 60-90% to liver ± regional lymph nodes Hyperechoic liver metastasis is suggestive of islet cell tumor rather than pancreatic adenocarcinoma! calcifications highly suggestive of malignancyNUC: somatostatin receptor imaging with octreotide*DDx:* 1.Pancreatic ductal adenocarcinoma (hypovascular, smaller, encasement of SMA + celiac trunk)2.Microcystic adenoma (benign tumor, small cysts, older women)3.Metastatic tumor: renal cell carcinoma (clinical Hx)4.Solid and papillary epithelial neoplasm (young female, hemorrhagic areas)5.Paraganglioma6.Sarcoma (rare)

ACTH-producing Tumor Gastrinoma Glucagonoma Insulinoma Nonfunctioning Islet Cell Tumor Somatostatinoma VIPoma

Notes:

ACTH-producing Tumor rare cause of Cushing syndrome ● increased level of serum cortisol ● impaired glucose tolerance > central obesity > hypertension, oligomenorrhea > osteoporosis > purpura > striae > muscle atrophy*Prognosis:*almost all malignant with metastases at time of diagnosis

Notes:

Gastrinoma 2nd most common islet cell tumor; in a cells / d cells *Age:*8% in patients <20 years; M > F*Path:*(a)islet cell hyperplasia (10%)(b)benign adenoma (30%): in 50% solitary, in 50% multiple (especially in MEN I)(c)malignant (50-60%) with metastases to liver, spleen, lymph nodes, bone*Associated with:* MEN Type I (in 10-40%) ● Zollinger-Ellison syndrome: severe recurrent peptic ulcer disease (>90%), malabsorption, hypokalemia, gastric hypersecretion, hyperacidity / occasionally hypoacidity, diarrhea (from gastric hypersecretion)◊Only 1:1,000 patients with peptic ulcer disease has a gastrinoma! ● GI bleeding ● elevated serum levels of gastrinLocation: (a)87% in pancreas (50% solitary in head / tail)(b)ectopic (7-33%):-duodenal wall (13% in medial wall of duodenum = gastrinoma triangle)-peripancreatic nodes / spleen-stomach, jejunum-omentum, retroperitoneum-ovaryfrequently in "gastrinoma triangle" (= triangle defined by porta hepatis as apex of triangle + 2nd and 3rd parts of duodenum as the base) √ average tumor size 3.4 cm (up to 15 cm)√ occasionally calcifications√ homogeneous hypoechoic massAngio: √ hypervascular lesion (70%)√ hepatic venous sampling after intraarterial stimulation with secretinCT: √ transiently hyperdense on dynamic CT (majority) √ thickening of gastric rugal foldsMR: √ low-intensity mass on fat-suppressed T1WI√ diminished central + peripheral ring enhancement√ high-intensity mass on fat-suppressed T2WISensitivity of preoperative localization: 25% for US, 35% for CT, 20% for MRI, 42-63% for transhepatic portal venous sampling for gastrin, 68-70% for selective angiography, 77% for arteriography combined with intra-arterial injection of secretin *Rx:*surgery curative in 30%

Notes:

Glucagonoma Uncommon tumor; derived from a cells; M < F *Associated with:* MEN • necrolytic erythema migrans (erythematous macules / papules on lower extremity, groin, buttocks, face) in >70% of patients • diarrhea, diabetes, painful glossitis, weight loss, anemia • plasma glucagon level > 1,000 ng/LLocation:predominantly in pancreatic body / tail • tumor size 2.5-25 cm (mean 6.4 cm) with solid + necrotic components • hypervascular in 90%; successful angiographic localization in 15%*Cx:*deep vein thrombosis + pulmonary embolism*Prognosis:*in 60-80% malignant transformation (liver metastases at time of diagnosis in 50%); 55% 5-year survival rate

Notes:

Insulinoma Most common functioning islet cell tumor *Age:*4th-6th decade; M:F = 2:3*Associated with:*MEN Type I*Path:*(a)single benign adenoma (80-90%)(b)multiple adenomas / microadenomatosis (5-10%)(c) islet cell hyperplasia (5-10%)(d) malignant adenoma (5-10%) ● Whipple triad: starvation attack + hypoglycemia (fasting glucose <50 mg/dL) + relief by IV dextrose ● neuroglycopenic symptoms: headaches, confusion, coma ● hypoglycemia exacerbated by fasting results in frequent meals to avoid symptoms ● sweating, palpitations, tremor (secondary to catecholamine release in response to hypoglycemia) ● obesity ● firm rubbery palpable mass at surgery (in >90%)Location:no predilection for any part of pancreas, 2-5% in ectopic location; 10% multiple (especially in MEN I)√ average tumor size 1-2 cm; <1.5 cm in 70%US (20-75% preoperative and 75-100% endoscopic + intraoperative sensitivity) √ round / oval smoothly marginated solid homogeneously hypoechoic mass Angio: √ hypervascular tumor (66%): accurate angiographic localization in 50-90%√ transhepatic portal venous sampling (correct localization in 95%)√ hepatic venous sampling after intraarterial stimulation with calcium gluconateCECT (30-75% sensitivity): √ hypo- / iso- / hyperattenuating lesionMR: √ low signal intensity on fat-suppressed T1WI√ hyperintense on T2WI + dynamic contrast-enhanced + suppressed inversion recovery images*Prognosis:*malignant transformation in 5-10%*Rx:*surgery curative

Notes:

Nonfunctioning Islet Cell Tumor *Incidence:*3rd most common islet cell tumor after insulinoma + gastrinoma; 15-25% of all islet cell tumorsDerived from either alpha or beta cells *Age:*24-74 (mean 57) years ● mostly asymptomatic (hormonally quiescent) ● abdominal pain, jaundice, gastric variceal bleeding ● palpable mass, gastric outlet obstructionLocation:predominantly in pancreatic head√ tumor size 6-20 cm (>5 cm in 72%) with solid + necrotic components√ coarse nodular calcifications (20-25%)√ CT contrast enhancement in 83%√ hypoechoic mass √ late dense capillary stain√ large irregular pathologic vessels with early venous filling*Prognosis:*in 80-100% malignant transformation with metastases to liver + regional nodes; 60% 3-year survival; 44% 5-year survival*Rx:*may respond to systemic chemotherapy

Notes:

Somatostatinoma Derived from delta cells ● inhibitory syndrome = inhibitory action of somatostatin on other pancreatic + bowel peptides (growth hormone, TSH, insulin, glucagon, gastric acid, pepsin, secretin) ● diabetes, cholelithiasis, steatorrhea ● elevated level of somatostatinLocation:predominantly in pancreatic head√ tumor size 0.6-20 cm (average >4 cm)√ hypervascular*Prognosis:*50-90% malignant transformation; metastatic disease in 70% at time of initial diagnosis

Notes:

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VIPoma =solitary tumor liberating **V**asoactive **I**ntestinal **P**eptides acting directly on cyclic adenosine monophosphate within epithelial cells of bowel relaxing vascular smooth muscle; sporadic occurrence*Histo:*adenoma / hyperplasiaM:F = 1:2 ● **WDHA syndrome** = **w**atery **d**iarrhea + **h**ypokalemia + **a**chlorhydria (more recently + more accurately described as) **WDHH syndrome** = **w**atery **d**iarrhea + **h**ypokalemia + **h**ypochlorhydria = "pancreatic cholera" = **Verner-Morrison syndrome** ● dehydration due to massive diarrhea (>1 L/day)Location: (1)pancreas: from delta cells predominantly in pancreatic body / tail(2)extrapancreatic: retroperitoneal ganglioblastoma, pheochromocytoma, lung, neuroblastoma (in children)√ average size 5-10 cm with solid + necrotic tissue√ mostly hypervascular tumor√ dilatation of gallbladder*Prognosis:*in 50-80% malignant transformation*DDx:*small cell carcinoma of lung / neuroblastoma may also cause WDHH syndrome

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Notes:

PANCREATIC LIPOMATOSIS
=FATTY REPLACEMENT = FATTY INFILTRATION=deposition of fat cells in pancreatic parenchyma*Predisposing factors:* 1.Atherosclerosis of elderly2.Obesity3.Steroid therapy4.Diabetes mellitus5.Cushing syndrome6.Chronic pancreatitis7.Main pancreatic duct obstruction8.Cystic fibrosis9.Malnutrition / dietary deficiency10.Hepatic disease11.Hemochromatosis12.Viral infection13.Schwachman-Diamond syndrome√ fatty replacement often uneven√ increase in AP diameter of pancreatic head with focal fatty replacement = lipomatous pseudohypertrophy√ prominently lobulated external contourUS:√ increased pancreatic echogenicityCT:√ "marbling" of pancreatic parenchyma / total fatty replacement / lipomatous pseudohypertrophy

Pancreatic Fatty Sparing

Notes:

Pancreatic Fatty Sparing =sparing of fatty change in pancreatic head + uncinate process (ventral pancreatic anlage) as initial stage in pancreatic lipomatosis*Histo:*ventral pancreatic anlage has smaller + more densely packed acini with scanty / absent interacinar fatUS: √ rounded / triangular hypoechoic area within pancreatic head / uncinate process + diffusely increased echogenicity in remainder of glandCT: √ higher-density region of pancreatic head + uncinate process with diffusely decreased attenuation of pancreatic body + tail

Notes:

PANCREATIC PSEUDOCYST

=collection of pancreatic fluid encapsulated by fibrous tissue*Etiology:*(1)Acute pancreatitis ; pseudocysts mature in 6-8 weeks(2)Chronic pancreatitis(3)Posttraumatic(4)Pancreatic cancer*Incidence:*2-4% in acute pancreatitis;10-15% in chronic pancreatitis Location:2/3 within pancreasAtypical location (may dissect along tissue planes in 1/3): (a)intraperitoneal: mesentery of small bowel / transverse colon / sigmoid colon(b)retroperitoneal: along psoas muscle; may present as groin mass / in scrotum(c)intraparenchymal: liver, spleen, kidney(d)mediastinal (through esophageal hiatus > aortic hiatus > foramen of Morgagni > erosion through diaphragm): may present as neck massPlain film / contrast radiograph: √ smooth extrinsic indentation of posterior wall of stomach / inner duodenal sweep (80%)√ indentation / displacement of splenic flexure / transverse colon (40%)√ downward displacement of duodenojejunal junction√ gastric outlet obstruction√ splaying of renal collecting system / ureteral obstructionUS (pseudocyst detectable in 50-92%; 92-96% accuracy): √ usually single + unilocular cyst√ multilocular in 6%√ fluid-debris level / internal echoes (may contain sequester, blood clot, cellular debris from autolysis)√ septations (rare; sign of infection / hemorrhage)√ may increase in size (secondary to hypertonicity of fluid, communication with pancreatic duct, hemorrhage, erosion of vessel)√ obstruction of pancreatic duct / CBDCT: √ fluid in pseudocyst (0-30 HU)√ cyst wall calcification (extremely rare)Pancreatography: √ communication with pancreatic duct in up to 70%*Cx* (in 40%): 1.Rupture into abdominal cavity, stomach, colon, duodenum2.Hemorrhage / formation of pseudoaneurysm3.Infection√ gas bubbles (DDx: fistulous communication to GI tract)√ increase in attenuation of fluid contents4.Intestinal obstruction*Prognosis:*spontaneous resolution (in 20-50%) secondary to rupture into GI tract / pancreatic / bile duct*DDx:*pancreatic cystadenoma, cystadenocarcinoma, necrotic pancreatic carcinoma, fluid-filled bowel loop, fluid-filled stomach, duodenal diverticulum, aneurysm

Notes:

PANCREATIC TRANSPLANTATION
*Complications:*sepsis, rejection, pancreatitis, pseudocyst, pancreatic abscess (22%), anastomotic leak*Prognosis:*40% survival rate >1 year

Graft-vessel Thrombosis in Pancreatic Transplant (2-19%) Acute Rejection of Pancreatic Transplant

Notes:

Graft-vessel Thrombosis in Pancreatic Transplant (2-19%)
A.Early thrombosis<1 month after transplantation *Cause:*technical error in fashioning anastomosis, microvascular damage due to preservation injuryB.Late thrombosis>1 month after transplantation *Cause:*alloimmune arteritis with gradual occlusion of small blood vessels

Notes:

Acute Rejection of Pancreatic Transplant ● focal tenderness over transplant ● measurement of urinary + serum amylase, blood glucose (nonspecific for diagnosis of rejection)US: √ poor margination of transplant√ acoustic inhomogeneity√ dilated pancreatic duct

Notes:

PANCREATITIS

Cause: A.IDIOPATHIC (20%)B.ALCOHOLISM: acute pancreatitis (15%); chronic pancreatitis (70%)C.CHOLELITHIASIS: acute pancreatitis (75%); chronic pancreatitis (20%)D.METABOLIC DISORDERS1.Hypercalcemia in hyperparathyroidism (10%), multiple myeloma, amyloidosis, sarcoidosis2.Hereditary pancreatitis: autosomal dominant, only Caucasians affected, most common cause of large spherical pancreatic calcifications in childhood, recurrent episodes of pancreatitis, development into pancreatic carcinoma in 20-40%; pronounced dilatation of pancreatic duct; pseudocyst formation (50%); associated with type I hypercholesterolemia3.Hyperlipidemia Types I and V4.Kwashiorkor = Tropical pancreatitisE.INFECTION / INFESTATION1.Viral infection (mumps, hepatitis, mononucleosis)2.Parasites (ascariasis, clonorchis)F.TRAUMA1.Penetrating ulcer2.Blunt / penetrating trauma3.Surgery (in 0.8% of Billroth-II resections, 0.8% of splenectomies, 0.7% of choledochal surgery, 0.4% of aortic graft surgery)G.STRUCTURAL ABNORMALITIES1.Pancreas divisum2.CholedochoceleH.DRUGSAzathioprine, thiazide, furosemide, ethacrynic acid, sulfonamides, tetracycline, phenformin, steroids (eg, renal transplant), asparaginase, procainamide I.MALIGNANCYPancreatic carcinoma (in 1%), metastases, lymphoma *Theories of pathogenesis:* Reflux of bile / pancreatic enzymes / duodenal succus (a)terminal duct segment shared by common bile duct + pancreatic duct(b)obstruction at papilla of Vater from inflammatory stenosis, edema / spasm of sphincter of Oddi, tumor, periduodenal diverticulum(c)incompetent sphincter of Oddi

Acute Pancreatitis Chronic Pancreatitis

Notes:

Acute Pancreatitis =inflammatory disease of pancreas producing temporary changes with restoration of normal anatomy + function following resolution*Path:* 1.EDEMATOUS PANCREATITIS:edema, congestion, leukocytic infiltrates; mortality rate of 4% 2.NECROTIZING PANCREATITIS:proteolytic destruction of pancreatic parenchyma; mortality rate of 80-90% (a)HEMORRHAGIC PANCREATITIS:+ fat necrosis and hemorrhage (b)SUPPURATIVE PANCREATITIS:+ bacterial infection A. Diffuse pancreatitis (52%) B. Focal pancreatitis (48%): location of head:tail = 3:2 *Clinical stages:* I=EDEMATOUS PANCREATITIS (75%) ● rapid improvement following conservative therapy ● gradual decrease of elevated enzymes*Mortality:*1-5%II=PARTIALLY NECROTIZING PANCREATITIS ● delayed / no response to conservative therapy ● delayed / no normalization of enzymes ● leukocytosis of <16,000 ● hyperglycemia of <200 mg/100 mL ● hypocalcemia of >4 mval/L ● base deficit of <4 mval/L*Mortality:*30-75%III=TOTALLY NECROTIZING PANCREATITIS ● deterioration under conservative therapy ● leukocytosis of >16,000 ● hyperglycemia of >200 mg/100 mL ● hypocalcemia of <4 mval/L ● base deficit of >4 mval/L*Mortality:*100% (40% by 2nd day, 75% by 5th day, 100% by 10th day) ● acute abdominal pain (peaking after a few hours, resolving in 2-3 days), nausea, vomiting ● raised pancreatic amylase + lipase in blood + urine ● increased amylase-creatinine clearance ratio ● signs of hemorrhagic pancreatitis: ● Cullen sign = periumbilical ecchymosis ● Grey-Turner sign = flank ecchymosis ● Fox sign = infrainguinal ecchymosis√ NO findings on US / CT in 29%Abdominal film: √ "colon cutoff" sign = dilated transverse colon with abrupt change to a gasless descending colon (inflammation via phrenicocolic ligament causes spasm + obstruction at the splenic flexure impinging on a paralytic colon)√ "sentinel loop" (10-55%) = localized segment of gas-containing bowel in duodenum (in 20-45%) / terminal ileum / cecum√ "renal halo" sign = water-density of inflammation in anterior pararenal space contrasts with perirenal fat; more common on left side√ mottled appearance of peripancreatic area (secondary to fat necrosis in pancreatic bed, mesentery, omentum)√ intrapancreatic gas bubbles (from acute gangrene / suppurative pancreatitis)√ "gasless abdomen" = fluid-filled bowel associated with vomiting√ ascitesCXR (findings in 14-71%): √ pleural effusion (in 5%), usually left-sided, with elevated amylase levels (in 85%)√ left-sided diaphragmatic elevation√ left-sided subsegmental atelectasis (20%)√ parenchymal infiltrates, pulmonary infarction√ pulmonary edema, ARDS√ pleural empyema, pericardial effusion√ mediastinal abscess, mediastinal pseudocyst √ pancreatico-bronchial / -pleural / -pulmonary fistulaUGI: √ esophagogastric varices (from splenic vein obstruction)√ enlarged tortuous edematous rugal folds along antrum + greater curvature (20%)√ widening of retrogastric space (from pancreatic enlargement / inflammation in lesser sac)√ diminished duodenal peristalsis + edematous folds√ widening of duodenal sweep + downward displacement of ligament of Treitz√ Poppel sign = edematous swelling of papilla√ Frostberg inverted-3 sign = segmental narrowing with fold thickening of duodenum√ jejunal + ileal fold thickening (proteolytic spread along mesentery)BE: √ narrowing, nodularity, fold distortion along inferior haustral row of transverse colon ± descending colonCholangiography: √ long gently tapered narrowing of CBD√ prestenotic biliary dilatation√ smooth / irregular mucosal surfaceBone films (findings in 6%): secondary to metastatic intramedullary lipolysis + fat necrosis √ punched out / permeative destruction of cancellous bone + endosteal erosion√ aseptic necrosis of femoral / humeral heads√ metaphyseal infarcts, predominantly in distal femur + proximal tibiaUS (pancreatic visualization in 62-78%): √ hypoechoic diffuse / focal enlargement of pancreas√ dilatation of pancreatic duct (if head focally involved)√ perivascular cloaking = spread of inflammatory exudate along perivascular spaces√ extrapancreatic hypoechoic mass with good acoustic transmission (= phlegmonous pancreatitis)√ fluid collection: lesser sac (60%), L > R anterior pararenal space (54%), posterior pararenal space (18%), around left lobe of liver (16%), in spleen (9%), mediastinum (3%), iliac fossa, along transverse mesocolon / mesenteric leaves of small intestineFate of fluid collection:(a)complete resolution(b)pseudocyst formation(c)bacterial infection = abscess √ pseudocyst formation (52%): extension into lesser sac, transverse mesocolon, around kidney, mediastinum, lower quadrants of abdomenCT (pancreatic visualization in 98%): √ no detectable change in size / appearance (29%)√ hypodense (5-20 HU) mass in phlegmonous pancreatitis; may persist long after complete recovery√ hyperdense areas (50-70 HU) in hemorrhagic pancreatitis for 24-48 hours√ enlargement with convex margins + indistinctness of gland with parenchymal inhomogeneity√ thickening of anterior pararenal fascia√ non-contrast-enhancing parenchyma during bolus injection (= pancreatic necrosis)Angiography: √ may be normal√ hypovascular areas (15-56%)√ hypervascularity + increased parenchymal stain (12- 45%)√ venous compression secondary to edema√ formation of pseudoaneurysms (in 10% with chronic pancreatitis): splenic artery (50%), pancreatic arcades, gastroduodenal artery *Cx:* 1.Phlegmon (18%) = solid mass characterized by edema, infiltration of inflammatory cells + necrosis: extension into lesser sac, anterior pararenal space, transverse mesocolon, small bowel mesentery, retroperitoneum, pelvis2.Pseudocyst formation (10%)3.Hemorrhage (3%)4.Abscess (2-10%): 2-4 weeks after severe acute pancreatitis; most commonly due to E. coli √ may contain gas within pancreatic bed*DDx:*air secondary to intestinal fistula5.Pancreatic ascites6.Biliary duct obstruction7.Thrombosis of splenic vein / SMV8.Pseudoaneurysm(a)rupture into preexisting pseudocyst(b)digestion of arterial wall by enzymes*Incidence:*in up to 10% of severe pancreatitisLocation:splenic artery (most common), gastroduodenal, pancreatico-duodenal, hepatic artery*Mortality:*37% for rupture, 16-50% for surgery*Therapy:* 1.Conservative (NPO, gastric tube, atropine, analgesics, sedation, prophylactic antibiotics) for stage I2.Early surgery in stages II and III

Notes:

Chronic Pancreatitis =continuing inflammatory disease of pancreas characterized by irreversible damage to anatomy + functionA.CHRONIC CALCIFYING PANCREATITIS:√ protein plugs / calculi within ductal systemB.CHRONIC OBSTRUCTIVE PANCREATITIS: secondary to slow growing tumor / surgical duct ligation / ampullary stenosis√ dilatation of pancreatic duct√ normal sized / focally or diffusely enlarged / small atrophic gland√ calcifications uncommon ● acute exacerbation of epigastric pain (93%):decreasing with time due to progressive destruction of gland, usually painless after 7 years ● jaundice (42%) from common bile duct obstruction ● steatorrhea (80%) ● diabetes mellitus (58%) ● secretin test with decreased amylase + bicarbonate in duodenal fluid Plain film:√ numerous irregular calcifications (in 20-50% of alcoholic pancreatitis) PATHOGNOMONICUGI:√ displacement of stomach / duodenum by pseudocyst√ shrinkage / fold induration of stomach (DDx: linitis plastica)√ stricture of duodenumCholangiopancreatography (most sensitive imaging modality):√ slight ductal ectasia / clubbing of side branches (minimal disease)√ "nipping" = narrowing of the origins of side branches√ dilatation >2 mm, tortuosity, wall rigidity, main ductal stenosis (moderate disease)√ "beading, chain of lakes, string of pearls"=dilatation, stenosis, obstruction of main pancreatic duct + side branches (severe disease)√ intraductal protein plugs / calculi√ prolonged emptying of contrast material√ may have stenosis / obstruction + prestenotic dilatation of CBDUS / CT:√ irregular (73%) / smooth (15%) / beaded (12%) pancreatic ductal dilatation (in 41-68%)√ small atrophic gland (in 10-54%)√ pancreatic mostly intraductal calcifications (4-68%)√ inhomogeneous gland with increased echogenicity (62%)√ irregular pancreatic contour (45-60%)√ focal (12-32%) / diffuse (27-45%) pancreatic enlargement√ mostly mild biliary ductal dilatation (29%)√ intra- / peripancreatic pseudocysts (20-34%)√ segmental portal hypertension (= splenic vein thrombosis + splenomegaly) in 11%√ arterial pseudoaneurysm formation√ peripancreatic fascial thickening + blurring of organ margins (16%)√ ascites / pleural effusion (9%)MR:√ loss of signal intensity on fat-suppressed T1WI (from loss of aqueous protein in pancreatic acini secondary to fibrosis)√ diminished contrast enhancement (from loss of normal capillary network replaced by fibrous tissue)Angiography:√ increased tortuosity + angulation of pancreatic arcades + intrahepatic arteries (88%)√ luminal irregularities / focal fibrotic arterial stenoses (25-75%) / smooth beaded appearance√ irregular parenchymal stain√ venous compression / occlusion (20-50%)√ portoportal shunting + gastric varices without esophageal varices*Cx:*pancreatic carcinoma (2-4%), jaundice, pseudocyst formation, pancreatic ascites, thrombosis of splenic / mesenteric / portal vein*Rx:*surgery for infected pseudocyst, GI-bleeding from portal hypertension, common bile duct obstruction, gastrointestinal obstruction

Notes:

PASSIVE HEPATIC CONGESTION

*Cause:*CHF, constrictive pericarditis*Pathophysiology:*chronic central venous hypertension transmitted to hepatic sinusoids results in centrilobular congestion + eventually hepatic atrophy, necrosis, fibrosis ● abnormal liver function testsCT: √ globally delayed enhancement (36%)√ enhancement of portal veins + hepatic arteries + immediately adjacent parenchyma (56%)√ "reticulated mosaic" pattern = lobular patchy areas of enhancement separated by coarse linear regions of diminished attenuation (100%)√ diminished periportal attenuation (24%)√ diminished attenuation around intrahepatic IVC (8%)√ prominent IVC + hepatic vein enhancement (due to contrast reflux from right atrium into dilated IVC)*DDx:*Budd-Chiari syndrome (regional / lobular distribution of reticulated mosaic pattern, caudate lobe hypertrophy)

Notes:

PELIOSIS HEPATIS

[*pelios* , Greek = purple] =rare benign disorder characterized by multiple blood-filled cavities randomly distributed throughout liver*Cause:*(a)? acquired: chronic infection (TB), hepatotoxic drugs (androgen-anabolic steroids, chemotherapeutic agents) diabetes mellitus, chronic renal failure(b)? congenital: angiomatous malformation*Histo:*(1)**Phlebectatic** peliosis hepatis (early stage)= endothelial-lined cysts (= ? dilatation of central veins) communicating with dilated hepatic sinusoids + compression of surrounding liver (2)**Parenchymal** peliosis hepatis (late stage)= irregularly shaped cysts without lining communicating with dilated hepatic sinusoids + areas of liver cell necrosis *Associated with:*hormonally induced benign / malignant tumors*Age:*fetal life (rare) to adult life√ hepatomegalyAngio: √ multiple small (several mm to 1.5 cm) round collections of contrast medium scattered throughout liver in late arterial phase of hepatic arteriogram√ ± simultaneous opacification of hepatic veins*Prognosis:*reversible after drug withdrawal / progression to hepatic failure / intraperitoneal hemorrhage leading to death

Notes:

PERICHOLECYSTIC ABSCESS

*Cause:*subacute perforation of gallbladder wall subsequent to gangrene + infarction due to acute cholecystitis*Prevalence:*2-20%Location:(a)gallbladder bed (most common)√ area of low-level echoes in liver adjacent to gallbladder(b)intramural√ small area of low-level echoes within thickened gallbladder wall(c)intraperitoneal√ area of low-level echoes within peritoneal cavity adjacent to gallbladder*Rx:*(1)Emergency operation(2)Antibiotic treatment + elective operation(3)Percutaneous abscess drainage

Notes:

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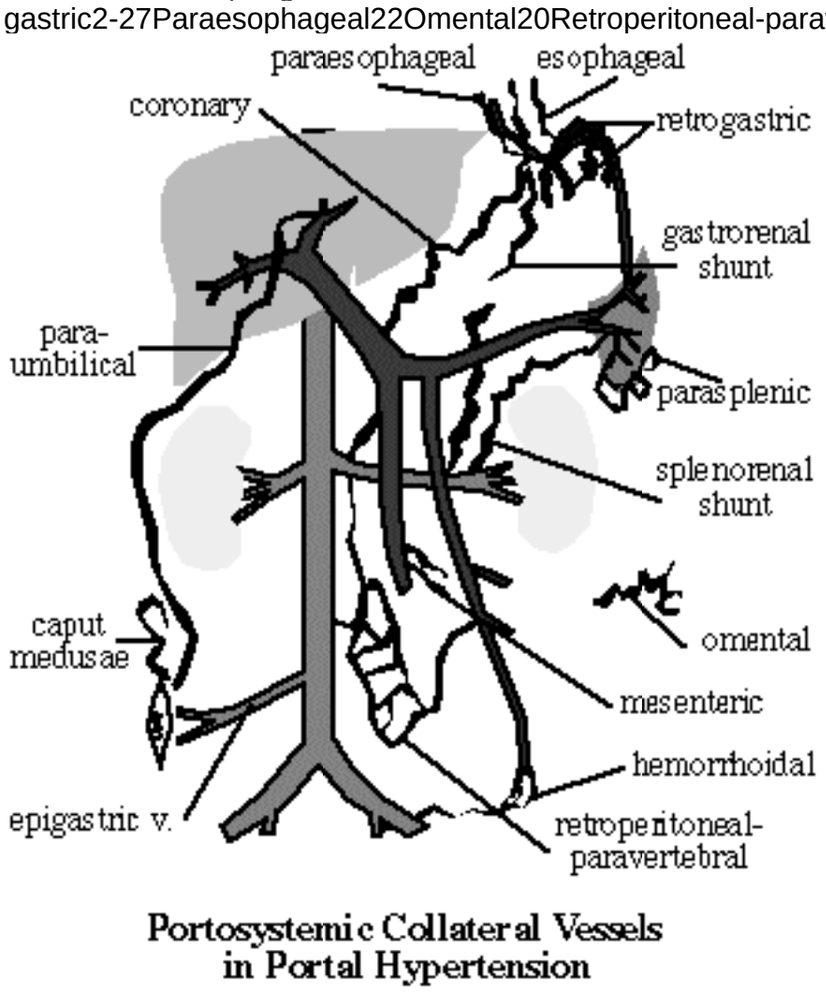
PORCELAIN GALLBLADDER
=calcium incrustation of gallbladder wall*Incidence:*0.6-0.8% of cholecystectomy patients;M:F = 1:5*Histo:*(a)flakes of dystrophic calcium within chronically inflamed + fibrotic muscular wall(b)microliths scattered diffusely throughout mucosa, submucosa, glandular spaces, Rokitansky-Aschoff sinuses*Associated with:*gallstones in 90%

- minimal symptoms√ curvilinear (muscularis) / granular (mucosal) calcifications in segment of wall / entire wall√ nonfunctioning GB on oral cholecystogram√ highly echogenic shadowing curvilinear structure in GB fossa (DDx: stone-filled contracted GB)√ echogenic GB wall with little acoustic shadowing (DDx: emphysematous cholecystitis)√ scattered irregular clumps of echoes with posterior acoustic shadowing*Cx:*10-20% develop carcinoma of gallbladder

Notes:

PORTAL HYPERTENSION

● normal hepatic blood flow of 550-900 mL/min (= 25% of cardiac output) passes through portal system (2/3) + through hepatic artery (1/3)*Classification:* A.DYNAMIC / HYPERKINETIC PORTAL HYPERTENSIONcongenital / traumatic / neoplastic arterioportal fistula B.INCREASED PORTAL RESISTANCE@Prehepatic-portal vein thrombosis (portal phlebitis, oral contraceptives, coagulopathy, neoplastic invasion, pancreatitis, neonatal omphalitis)-portal vein compression (tumor, trauma,lymphadenopathy, portal phlebosclerosis, pancreatic pseudocyst)@Intrahepatic (= obstruction of portal venules)-presinusoidal1.Congenital hepatic fibrosis2.Idiopathic noncirrhotic fibrosis3.Primary biliary cirrhosis4.a-1-antitrypsin deficiency5.Wilson disease6.Sarcoid liver disease7.Toxic fibrosis (arsenic, copper, PVC)8.Reticuloendotheliosis9.Myelofibrosis10.Felty syndrome11.Schistosomiasis12.Cystic fibrosis13.Chronic malaria-sinusoidal1.Hepatitis2.Sickle cell disease-postsinusoidal1.Cirrhosis (most frequent): Laennec cirrhosis, postnecrotic cirrhosis from hepatitis2.Venoocclusive disease of liver@Posthepatic1.Budd-Chiari syndrome2.Constrictive pericarditis3.CHF (tricuspid incompetence)*Pathophysiology:* continued elevated pressure despite formation of portal venous collateral vessels may be explained by (a)backward flow theory = hypodynamic flow theory=continuing increase in intrahepatic resistance + inadequate collateralization ● low / stagnant portal venous flow rates(b)forward flow theory = hyperdynamic flow theory=splanchnic flow increases secondary to splanchnic vasodilatation + increase in cardiac output to preserve hepatic perfusion ● increased portal venous flow rates >15 mL/min/kg ● elevated hepatic wedge pressure (HWP) = portal venous pressure (normal <10 mm Hg); normal values seen in presinusoidal portal hypertension ● **caput medusae** = drainage from paraumbilical + omental veins through superficial veins of chest (lateral thoracic vein to axillary vein; superficial epigastric vein to internal mammary vein and subclavian vein) + abdominal wall (circumflex iliac vein and superficial epigastric vein to femoral vein; inferior epigastric vein to external iliac vein) ● hemorrhaging esophageal varices (50%) @Splanchnic system:√ portal vein >13 mm (57% sensitivity, 100% specificity)√ SMV + splenic vein >10 mm; coronary vein >4 mm; recanalized umbilical vein >3 mm (size of vessels not related to degree of portal hypertension or presence of collaterals)√ loss of respiratory increase of splanchnic vein diameters (80% sensitivity, 100% specificity)√ portal vein aneurysm√ portal vein thrombosis√ cavernous transformation of portal vein√ increased echogenicity + thickening of portal vein wallsDoppler US: √ continuous portal vein flow without respiratory changes√ reduction of mean portal vein velocities to 7-12 cm/sec (normally 12-30 cm/sec)√ loss of flow increase in portal venous system during expiration√ may have hepatofugal flow within spontaneous splenorenal shunts (indicates high incidence of hepatic encephalopathy)√ dilated hepatic artery may demonstrate elevated resistive index >0.78@Portosystemic collaterals:*Type of Varices*Frequency (%)Coronary venous80-86Esophageal45-65Paraumbilical10-43Abdominal wall30Perisplenic30Retrogastric / gastric2-27Paraesophageal22Omental20Retroperitoneal-paravertebral18Mesenteric10Splenorenal10Gastrorenal7



Portosystemic Collateral Vessels in Portal Hypertension

√ varices = serpentine tubular rounded structures√ coronary (left gastric) vein >5-6 mm (in 26%)√ esophageal varices (= subepithelial + submucosal veins) supplied by anterior branch of left gastric vein√ paraesophageal varices (endoscopically not visible) supplied by posterior branch of coronary (= left gastric) vein draining into azygos + hemiazygos vv. + vertebral plexus◊NOT connected to esophageal varices!√ mediastinal / lung mass on CXR in 5-8%√ gallbladder wall varices in thickened gallbladder wall (in 80% associated with portal vein thrombosis)@Cruveilhier-von Baumgarten syndrome (20-35%)=recanalized paraumbilical veins (NOT recanalized umbilical veins)√ hypoechoic channel in ligamentum teres(a)size <2 mm (in 97% of normal subjects; in 14% of patients with portal hypertension)(b)size ≥2 mm (86% sensitivity for portal hypertension)√ arterial signal on Doppler US in 38%√ hepatofugal venous flow (82% sensitivity, 100% specificity for portal hypertension)@Spontaneous portosystemic shunts ● high frequency of hepatic encephalopathy1.Splenorenal / splenoadrenorenal shunt2.Gastrorenal shunt3.Mesenterorenal shunt (between SMV + right renal v.)4.Splenocaval shunt (between splenic v. + left hypogastric v.)5.Gastropulmonary shunt (between gastric / esophageal vv. and pericardiophrenic / inferior pulmonary vv.)6.Intrahepatic shunt (portal v. to hepatic v.)@Spleen√ splenomegaly (absence does not rule out portal hypertension)√ siderotic Gamna-Gandy nodules in 13% (= small foci of perifollicular + trabecular hemorrhage)√ multiple 3-8 mm low-intensity spots on FLASH / GRASS images√ multiple hyperechoic spots on US√ multiple faint calcifications on CT√ ascites *Cx:*Acute gastrointestinal bleeding (mortality of 30-50% during 1st bleeding)

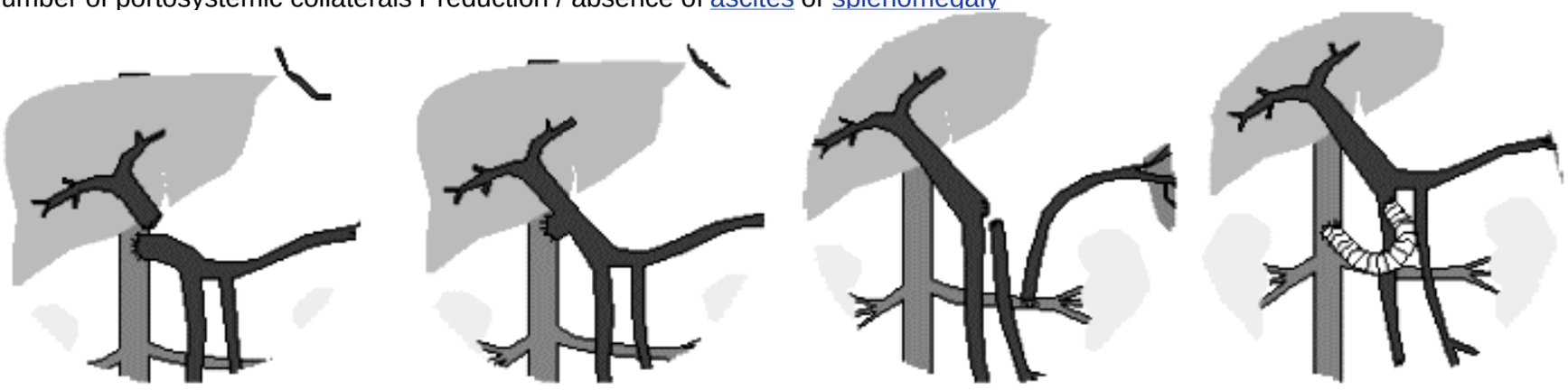
Segmental Portal Hypertension Portosystemic Surgical Connections Transjugular Intrahepatic Portosystemic Shunt (TIPS)

Notes:

Segmental Portal Hypertension =splenic vein occlusion / superior mesenteric vein occlusion

Notes:

Portosystemic Surgical Connections 1.Portacaval shunt=portal vein to IVC end-to-side / side-to-side2.Distal splenorenal shunt = Warren shunt (popular)=splenic vein to left renal vein3.Mesocaval shunt=synthetic graft between SMV and IVC(a)short "H-graft" to posterior wall of SMV(b)long "C-graft" to anterior wall of SMV(c)direct mesocaval shunt dividing IVC (rare)4.Mesoatrial shunt=polytetrafluoroethylene (PTFE) graft between anterior wall of SMV superior to pancreas and right atrium coursing through abdomen + diaphragm into right thoracic cavity*Doppler criteria for shunt patency:* √ increased local velocities√ turbulence + severe spectral broadening√ dilatation of recipient vein at shunt site√ phasic flow pattern in portal tributaries√ hepatofugal flow in intrahepatic portal vein branches√ reduction in size + number of portosystemic collaterals√ reduction / absence of ascites or splenomegaly



Surgical Portosystemic Shunts

Notes:

Transjugular Intrahepatic Portosystemic Shunt (TIPS) =portal decompression through percutaneously established shunt with expandable metallic stent between hepatic + portal veins within the liver*Indication:*patients with esophageal + gastric variceal hemorrhage / refractory ascites due to advanced liver disease with portal hypertension, hepatorenal syndrome*Type of stent:*10-mm Wall stent (curved), Palmaz stent (straight), Strecker stent, spiral Z stent*Shunt surveillance:*at regular 3-6 months intervals forA.MORPHOLOGY1.Ascites2.Portosystemic collaterals3.Size of spleen4.Diameter of stent (usually 8-10 mm)5.Configuration of stent: areas of narrowing6.Extension of stent into portal + hepatic veinsB.HEMODYNAMICS1.Direction of flow in: extrahepatic portal vein, RT + LT portal vein, SMV, splenic vein, all 3 hepatic veins, intrahepatic IVC, paraumbilical vein, coronary vein2.Peak blood flow velocity within main portal vein3.Peak blood flow velocity within proximal + mid + distal aspects of stent4.Hepatic artery: PSV, EDV, RI Pre- and post-TIPS baseline study under stable fasting conditions! Pre-TIPSPost-TIPSPortal vein velocity (cm/s)10-3040-60Mean portal vein velocity (cm/s)18 ± 655 ± 7Portal pressure (mm Hg)37 ± 822 ± 6Shunt peak velocity (cm/s)95 ± 58√ high-velocity turbulent flow (50-270 cm/sec) at least double that of pre-TIPS values√ superimposed cardiac + respiratory variations√ increase in hepatic artery velocities from 77 cm/sec (pre-TIPS) to 119 cm/sec (post-TIPS)*Cx:*1.Shunt obstruction2.Hepatic vein stenosis3.Vascular injury: hepatic artery pseudoaneurysm, arterioportal fistula4.Intrahepatic / subcapsular hematoma5.Hemoperitoneum (due to penetration of liver capsule)6.Transient bile duct dilatation (due to hemobilia)7.Bile collection8.stent dislodgment with embolization to right atrium, pulmonary artery, internal jugular vein*Mortality:*<2% (intraperitoneal hemorrhage) *TIPS failure Cause:*acute thrombosis, improper stent placement, intimal hyperplasia, hepatic vein stenosis, change in stent configuration, bulging of liver parenchyma into shunt 1.Shunt obstruction (38%)*Prevalence:*31% at 1 year, 42% at 2 years ● recurrent bleeding = shunt abnormality in 100%A.>50% stenosisTime of onset:in 30-80% within 12 months√ irregular filling defects along wall of shunt on color Doppler◊pseudointimal hyperplasia is isoechoic to blood!√ gradual decrease in shunt velocity over 1-6 months (due to intimal hyperplasia)√ maximal shunt velocity of <60 cm/sec (>95% sensitive + specific)√ in- / decrease in peak flow velocity in similar location within stent >50 cm/sec relative to initial baseline study√ velocity transition zone within stent with flow acceleration by a factor of 2√ decrease in maximal portal vein velocity >33% from baseline√ reversal of portal venous flow direction (100% sensitive, 92% specific, 71% PPV, 100% NPV)√ loss of pulsatility of portal / shunt flow√ change in flow direction in collateral veins from baseline√ retrograde flow in RHV (developing stenosis of right hepatic venous outflow tract)√ developing / worsening ascites / splenomegalyB.Occlusion√ absent flow within shunt√ echogenic material within stent-acute cause: leakage of bile into / around stent, prolonged procedural catheterization-delayed cause: pseudointimal hyperplasia, stent shortening with delayed stent expansion

Notes:

PORTAL VEIN THROMBOSIS

Etiology: A.IDIOPATHIC (mostly): ? neonatal sepsisB.SECONDARY:(1)Tumor invasion by HCC, cholangiocarcinoma, pancreatic carcinoma, gastric carcinoma / extrinsic compression by tumor(2)Trauma; umbilical venous catheterization(3)Blood dyscrasia; clotting disorder; estrogen therapy; severe dehydration; Cx of splenectomy (7%, higher in patients with myeloproliferative disorders)(4)Intraabdominal sepsis with phlebitis; perinatal omphalitis; pancreatitis; ascending cholangitis(5)Cirrhosis + portal hypertension (5%)*Age:*predominantly children, young persons ● abdominal pain ● portal systemic encephalopathy ● hematemesis (esophageal varices)√ nonvisualization of portal vein√ calcification within clot / wall of portal vein√ splenomegaly√ ascitesPlain film: √ hepatosplenomegaly√ enlarged azygos vein√ paraspinal varicesUGI: √ esophageal varices√ thickening of bowel wallUS: √ echogenic material within vessel lumen (67%)√ increase in portal vein diameter (57%)◊Malignant thrombus tends to distend vein + exhibit pulsatile flow, a bland thrombus does not!√ portosystemic collateral circulation (48%)√ enlargement of thrombosed segment >15 mm (38%)√ no flow on postprandial Doppler color scans√ cavernous transformation = **cavernoma** (19%)√ failure to visualize the extrahepatic portal vein√ presence of a racemose conglomerate of collateral veins with portal venous flow linking pancreas + duodenum + gallbladder fossa√ decrease in hepatic artery resistive index√ RI <0.50 (in acute occlusive portal vein thrombosis)√ minimal decrease / normal RI (in chronic portal vein thrombosis / nonocclusive thrombosis)√ thickening of lesser omentumCECT: √ low-density center in portal vein surrounded by peripheral enhancement√ portal vein density 20-30 HU less than aortic density after contrastMR: √ areas of flow void in portal area + abnormal signal intensity in main portal veinAngio: √ "thread and streaks" sign of tumor thrombus (streaky contrast opacification of tumor vessels) *Cx:*(1)Hepatic infarction(2)Bowel infarction

Notes:

POSTCHOLECYSTECTOMY SYNDROME

=symptoms recurring / persisting after cholecystectomy*Incidence:* mild recurrent symptoms in 9-25%; severe symptoms in 2.6-32% (result of 1,930 cholecystectomies): -completely cured (61%)-satisfactory improvement with(a) persistent mild dyspepsia (11%) (b) mild attacks of pain (24%) -failure with(a) occasional attacks of severe pain (3%) (b) continuous severe distress (1.7%) (c) recurrent cholangitis (0.7%) *Cause:* A.BILIARY CAUSES(a)Incomplete surgery1. Gallbladder / cystic duct remnant 2. Retained stone in cystic duct remnant 3. Overlooked CBD stone (b)Operative trauma1. Bile duct stricture 2. Bile peritonitis (c)Bile duct pathology1. Fibrosis of sphincter of Oddi 2. Biliary dyskinesia 3. Biliary fistula (d)Residual disease in neighboring structures1. Pancreatitis 2. Hepatitis 3. Cholangitis (e)Overlooked bile duct neoplasiaB.EXTRABILIARY CAUSES (erroneous preoperative diagnosis)(a)Other GI tract disease:1. Inadequate dentition 2. Hiatus hernia 3. Peptic ulcer 4. Spastic colon (b)Anxiety state, air swallowing(c)Abdominal angina(d)Carcinoma outside gallbladder(e)Coronary artery disease

Notes:

RICHTER SYNDROME
= development of large cell / diffuse histiocytic lymphoma in patients with CLL *Etiology:*transformation / dedifferentiation of CLL lymphocytes*Incidence in CLL patients:*3-10%*Median age:*59 yearsMedium time interval after diagnosis of CLL: 24 months ● fever (65%) without evidence of infection ● increasing lymphadenopathy + hepatosplenomegaly (46%) ● weight loss (26%) ● abdominal pain (26%)Location:bone marrow, lymph nodes, liver, spleen, bowel, lung, pleura, kidney, dura*Prognosis:*median survival time: 4 months from diagnosis of lymphoma; 14% rate of remission rate

Notes:

SCHISTOSOMIASIS

Worldwide major cause of portal hypertension: 200 million people affected *Types:* A.SCHISTOSOMA HAEMATOBIUMin Africa, Mediterranean, Southwest Asia B.SCHISTOSOMA MANSONIoccurs in >70 million inhabitants of parts of Africa, Caribbean, Arabic peninsula, West Indies, northern part of South America C.SCHISTOSOMA JAPONICUMcoastal areas of China, Japan, Formosa, Philippines, Celebes *Cycle:* cercariae enter lymphatics + blood system via thoracic duct; larvae are transported into mesenteric capillaries; mature in portal system + liver into worms; worms live in pairs in copula within portal vein + tributaries for 10-15 years; female swims against bloodflow to reach venules of urinary bladder (S. haematobium) or intestine + rectum (S. mansoni, S. japonicum); deposits eggs in wall of urinary bladder or intestines, eggs pass with urine + feces; hatch within water to release miracidia which infect snail hosts; cercariae emerge after maturation from snails *Infection:*cercariae penetrate human skin / buccal mucosa from contaminated water (slow-moving streams, irrigation canals, paddy fields, lakes)*Histo:*granulomatous reaction + fibrosis along portal vein branches ● clinically mild infection with chronic course@ Liver √ marked diffuse thickening of echogenic walls of portal venules = periportal fibrosis◊Schistosoma infection is the most frequent cause of liver fibrosis worldwide!√ hepatosplenomegaly√ portal vein dilatation in 73% (= portal hypertension)√ normal parenchymal echogenicity + small peripheral hyperechoic foci in 50% (= fibrosis of portal radicles)√ hyperechoic gallbladder bed √ thickened gallbladder wall@ GI tract √ gastric + esophageal varices√ polypoid bowel wall masses (esp. in sigmoid)√ granulomatous colitis√ strictures with extensive pericolic inflammation*Cx:*ileus

Notes:

SCHWACHMAN-DIAMOND SYNDROME
=rare congenital absence of pancreatic exocrine tissue, 2nd most frequent cause of exocrine pancreatic insufficiency in childhood ● pancreatic insufficiency ● recurrent respiratory and skin infections (secondary to bone marrow hypoplasia) ● dwarfism (metaphyseal dysostosis) ● normal electrolytes in sweat ● tends to improve with time√ total fatty replacement of pancreas

Notes:

SOLID AND PAPILLARY NEOPLASM OF PANCREAS

=SOLID AND CYSTIC TUMOR = PAPILLARY-CYSTIC NEOPLASM = SOLID AND PAPILLARY EPITHELIAL NEOPLASM=rare, low-grade malignant tumor; often misclassified as nonfunctioning islet cell tumor, cystadenoma, cystadenocarcinoma of pancreas*Prevalence:*0.17-2.7% of all nonendocrine pancreatic tumors*Mean age:*25 (range 10-74) years ; M:F = 1:9; especially in black and East Asian patients*Path:*large well-encapsulated mass with considerable hemorrhagic necrosis + cystic degeneration*Histo:*sheets + cords of cells arranged around a fibrovascular stroma ● vague upper abdominal discomfort and pain ● gradually enlarging abdominal massLocation:tail of pancreas (most frequently)√ well-encapsulated inhomogeneous round / lobulated pancreatic mass with solid + cystic portions√ may be completely cystic (when complicated by extensive necrosis + internal hemorrhage)√ fluid-debris level (20%)√ mean diameter of 9 cm (range 3-15 cm)√ ± stippled / punctate / amorphous dystrophic calcification (33%)√ hypovascular with no contrast enhancement / enhancement of solid tissue projecting toward center of massUS: √ echogenic mass with necrotic centerMR: √ high signal intensity on T1WI (consistent with hemorrhagic necrosis)*Prognosis:*(1)excellent after excision(2)metastases (in 4%): omentum, lymph nodes, liver*DDx:*(1)Microcystic adenoma (innumerable tiny cysts, older age group) (2)Mucinous cystic neoplasm (large uni- / multilocular cysts, older age group)(3)Nonfunctioning islet cell tumor (hypervascular)(4)Pleomorphic carcinoma of pancreas (smaller tumor in older patient)(5)Pancreaticoblastoma (childhood tumor)(6)Calcified hemorrhagic pseudocyst

Notes:

Radiology Review Manual
fourth edition

SPLENIC ANGIOSARCOMA

*Incidence:*rare, <100 cases in literature*Cause:*usually not due to thorotrast or toxic exposure to vinyl chloride / arsenic as in liver angiosarcoma*Age:*50-60 years ● splenomegaly, abdominal pain√ multiple nodules of varying size usually enlarging the spleen√ solitary complex mass with variable contrast enhancement√ metastasizes to liver (70%)√ spontaneous rupture (33%)MR: √ focal / diffuse hypointense foci on T1WI + T2WI (iron deposition from hemorrhage)*Prognosis:*20% survival rate after 6 months

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Notes:

SPLENIC HAMARTOMA
=rare nonneoplastic tumor composed of a mixture of normal splenic elements*Etiology:*congenital*May be associated with:*hamartomas elsewhere as in tuberous sclerosis*Histo:*(a)white pulp subtype = aberrant lymphoid tissue(b)red pulp subtype = aberrant complex of sinusoids(c)mixture (most common) ● asymptomaticCT: √ attenuation equal to splenic tissue√ prolonged enhancementMR: √ heterogeneously hyperintense on T2WI√ diffuse heterogeneous enhancement, more homogeneous on delayed images

Notes:

SPLENIC HEMANGIOMA

*Cause:*congenital, arising from sinusoidal epithelium*Prevalence:* 0.03-14% (autopsy); M > F◊Most common primary splenic tumor!*Age:* 20-50 years*Histo:*proliferation of vascular channels lined by single layer of endothelium; mostly of cavernous type; may contain areas of infarction, hemorrhage, thrombosis, fibrosis*May be associated with:*Klippel-Trénaunay-Weber syndrome (multiple hemangiomas) ● asymptomatic / pain + fullness in LUQ√ usually small single lesion <4 cm, up to 17 cm in size√ foci of speckled / snowflakelike calcificationsMR: √ hyperintense on T2WI√ progressive centripetal enhancement with persistent uniform enhancement on delayed images*Prognosis:*slow growth, thus becoming symptomatic in adulthood*Cx:*(1)Spontaneous splenic rupture (in up to 25%)(2)Kasabach-Merritt syndrome (= anemia, thrombocytopenia, coagulopathy) with large hemangioma(3)Malignant degeneration

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Notes:

SPLENIC INFARCTION

◊Most common cause of focal defects!*Cause:* 1.Embolic: bacterial endocarditis (responsible in 50%), atherosclerosis with plaque emboli, cardiac thrombus (atrial fibrillation, left ventricular thrombus), metastatic carcinoma2.Local thrombosis: sickle cell disease (leading to functional asplenia), myelo- / lymphoproliferative disorders (CML most common), polycythemia vera, myelofibrosis with myeloid metaplasia + splenomegaly, Gaucher disease3.Vasculitis: periarteritis nodosa4.Vascular compromise of splenic artery: focal inflammatory process (eg, pancreatitis), thrombus from splenic artery aneurysm, splenic torsion5.Therapeutic complication: transcatheter hepatic arterial embolization*mnemonic:*"PSALMS"**P**ancreatic carcinoma, **P**ancreatitis **S**ickle cell disease / trait **A**denocarcinoma of stomach **L**eukemia **M**itral stenosis with emboli **S**ubacute bacterial endocarditis ● LUQ pain, fever ● elevated erythrocyte sedimentation rate, leukocytosis ● abnormal lactate dehydrogenase levels √ single / multiple focal wedge-shaped peripheral defectsCT phases: (a)hyperacute phase (day 1)√ mottled area of increased attenuation on NECT (hemorrhage)√ large focal hyperattenuating lesion on CECT√ mottled pattern of contrast enhancement(b)acute (days 2-4) + subacute phase (days 4-8)√ focal progressively more well-demarcated areas of decreased attenuation without enhancement(c)chronic phase (2-4 weeks)√ size decreases + attenuation returns to normal√ complete resolution / residual contour defect√ areas of calcification*Cx:*superimposed infection, splenic rupture

Notes:

SPLENIC TRAUMA

◊Most frequently injured intraperitoneal organ in blunt abdominal trauma*Associated with:*other solid visceral / bowel injuries (29%); lower rib fractures in 44%, injury to left kidney in 10%, injury to left diaphragm in 2%*Technique:*scanning delay of 60-70 sec to avoid the phase of heterogeneous splenic enhancementCT sensitivity:>95% for splenic injury, but not reliable to determine need for surgical intervention◊Attenuation of active extravasation (80-370 HU) exceeds that of splenic parenchyma / clotted blood*Prognosis:*high PPV for surgery1.Intrasplenic laceration√ linear parenchymal defect√ almost always associated with hemoperitoneum2.Splenic fracture√ laceration traverses two capsular surfaces3.Subcapsular hematoma√ crescentic lesion along splenic margin flattening / indenting the normally convex lateral margin4.Perisplenic hematoma√ "sentinel clot" (= area of >60 HU adjacent to spleen) sensitive predictor of splenic injury5.Delayed splenic rupture=hemorrhage >48 hours after trauma*Prevalence:*0.3-20% of blunt splenic injuries*Time of onset:*in 70% within 2 weeks of injury,in 90% within 4 weeks of injury*Rx:*52% surgery (splenectomy (8%), splenorrhaphy), 48% nonsurgical management

Notes:

SPLENOSIS
=autotransplantation of splenic tissue to other sites following trauma*Age:*young men with history of trauma / splenectomy*Time of detection:*mean of 10 years (range of 6 months-32 years) after traumaLocation:diaphragmatic surface, liver, omentum, mesentery, peritoneum, pleura‵ multiple small encapsulated sessile implants (few mm -3 cm)‵ demonstrated by Tc-99m sulfur colloid; In-111 labeled platelets; Tc-99m heat-damaged RBC (best detection rate)*DDx:*accessory spleen

Notes:

SPONTANEOUS PERFORATION OF COMMON BILE DUCT

*Pathogenesis:*unknown (? CBD obstruction, localized mural malformation, ischemia, trauma)*Age:*5 weeks to 3 years of age ● vague abdominal distension ● mild persistent hyperbilirubinemia ● varying acholic stoolsUS: √ biliary ascites / loculated subhepatic fluid√ localized pseudocholedochal cyst in porta hepatisHepatobiliary scintigraphy: √ radioisotope diffusely throughout peritoneal cavity

Notes:

THOROTRASTOSIS
Thorotrast = 25% colloidal suspension of thorium dioxide; used as contrast agent between late 1920s and mid 1950s, in particular for cerebral angiography and liver spleen imaging; chemically inert with high atomic number of 90; >100,000 people injected **Thorium dioxide** = consists of 11 radioactive isotopes (thorium-232 is major isotope); decay by means of alpha, beta, and gamma emission; biologic half-life of 1.34 x 10^{10} years; hepatic dose of 1000-3000 rads in 20 years *Distribution:* phagocytosed by RES + deposited in liver (70%), spleen (30%), bone marrow, abdominal lymph nodes (20%) √ linear network of metallic density contrast material in spleen, lymph nodes, liver√ spleen may be shrunken / nonfunctional*Cx:*hepatic fibrosis, angiosarcoma (50%), cholangiocarcinoma, hepatocellular carcinoma (latency period of 3-40 years; mean 26 years)

Notes:

UNDIFFERENTIATED SARCOMA OF LIVER

=EMBRYONAL SARCOMA*Incidence:*4th / 5th most common liver tumor in pediatric population*Age:*<2 months (in 5%); 6-10 years (in 52%); by 15 years (in 90%); up to 49 years; M:F = 1:1*Histo:*primitive undifferentiated stellate / spindle-shaped sarcomatous cells closely packed in whorls + sheets / scattered loosely in a myxoid ground substance with foci of hematopoiesis (50%) ● painful RUQ mass and fever ● mild anemia + leukocytosis (50%) ● elevated liver enzymes (33%) ● fever (5%)Location:right lobe (75%); left lobe (10%); both lobes (15%)√ 7-14-21 cm in size√ well-defined margins (fibrous pseudocapsule)NUC: √ photodefect on sulfur colloid scanUS / CT: √ large intrahepatic mass with cystic areas up to 4 cm in diameter (myxoid stroma + necrosis + hemorrhage)√ discordant finding between US (solid) + CT (cystlike)Angio: √ hypo- / hypervascular with stretching of vessels√ scattered foci of neovascularity*Prognosis:*mostly results in death within 12 months*DDx:*mesenchymal hamartoma(a)solid lesion + cystic degeneration:hepatocellular carcinoma, fibrolamellar carcinoma, intrahepatic cholangiocarcinoma, angiosarcoma, epithelioid hemangio-endothelioma, other sarcomas, lymphoma, metastatic disease, hepatocellular adenoma (b)solitary cystic lesion:biliary cystadenoma / ~carcinoma, cystic degeneration of hepatocellular carcinoma, bacterial / parasitic abscess, metastatic disease, posttraumatic resolving hematoma

Notes:

WANDERING SPLEEN
=ABERRANT / FLOATING / PTOTIC / DRIFTING / DYSTOPIC / DISPLACED / PROLAPSED SPLEEN=excessively mobile spleen on an elongated pedicle displaced from its usual position in LUQ*Cause:*embryologically absent / malformed gastrosplenic + splenorenal ligaments; lax abdominal musculature during pregnancy*Age:*any (higher frequency in women of childbearing age) ● asymptomatic mobile abdominal / pelvic mass ● chronic vague lower abdominal / back pain ● nausea, vomiting, eructation, flatulence ● acute abdomen (with splenic infarction)√ empty splenic fossa√ inverted malpositioned stomach√ displaced large spleen (congestion during torsion) *Cx:* 1.Torsion with prolonged venous occlusion: perisplenitis, localized peritonitis, adhesions, venous thrombosis, hypersplenism2.Torsion with arterial occlusion: hemorrhagic infarction, subcapsular / intrasplenic hemorrhage, gangrene, degenerative cysts, functional asplenism3.GI complications:@Stomach: compression, distension, volvulus, traction diverticulum, varices@Small bowel: dilatation, obstruction@Colon: compression, volvulus, laxity, ptosis *Rx:*1.Splenectomy (4% postsplenectomy sepsis)2.Splenopexy3.Conservative treatment (if asymptomatic)

Notes:

Abnormal Air Collection 1.Abnormally located bowelChilaiditi syndrome (= colon interposed between liver and chest wall), inguinal hernia 2.Pneumoperitoneum3.Retropneumoperitoneumperforation of duodenum / rectum / ascending + descending colon, diverticulitis, ulcerative disease, endoscopic procedure 4.Gas in bowel wallgastric pneumatosis, phlegmonous gastritis, endoscopy, rupture of lung bulla 5.Gas within abscesslocated in subphrenic, renal, perirenal, hepatic, pancreatic space, lesser sac 6.Gas in biliary systemhepatobiliary fistula, surgery, duodenal ulcer, duodenal diverticulum, cancer, stone, patulous ampulla, emphysematous cholecystitis √ gas outlines choledochus ± gallbladder√ peripheral branches of bile ducts not filled*mnemonic:*"SITS"**S**tone **I**nflammation (emphysematous cholecystitis) **T**umor with fistula **S**urgery 7.Gas in portal venous system√ branching air within 2 cm of liver periphery

Notes:

Pneumoperitoneum *Etiology:* A.DISRUPTION OF WALL OF HOLLOW VISCUS(a)blunt / penetrating trauma1.Perforating foreign body (eg, thermometer injury to rectum, vaginal stimulator in rectum)2.Compressor air directed toward anus(b)iatrogenic perforation1.Laparoscopy / laparotomy (58%): absorbed in 1-24 days dependent on initial amount of air introduced and body habitus (80% in asthenic, 25% in obese patients)√After 3 days free air should be followed with suspicion!2.Leaking surgical anastomosis3.Endoscopic perforation4.Enema tip injury5.Diagnostic pneumoperitoneum(c)diseases of GI tract1.Perforated gastric / duodenal ulcer2.Perforated appendix3.Ingested foreign-body perforation4.Diverticulitis (ruptured Meckel diverticulum / sigmoid diverticulum, jejunal diverticulosis)5.Necrotizing enterocolitis with perforation6.Inflammatory bowel disease (eg, toxic megacolon)7.Obstruction* (gas traversing intact mucosa): neoplasm, imperforate anus, Hirschsprung disease, meconium ileus8.Ruptured pneumatosis cystoides intestinalis* with "balanced pneumoperitoneum" (= free intraperitoneal air acts as tamponade of pneumatosis cysts thus maintaining a balance between intracystic air + pneumoperitoneum)9.Idiopathic gastric perforation = spontaneous perforation in premature infants (congenital gastric muscular wall defect)B.THROUGH PERITONEAL SURFACE(a)transperitoneal manipulation1.Abdominal needle biopsy / catheter placement2.Mistaken thoracentesis / chest tube placement3.Endoscopic biopsy(b)extension from chest*1.Dissection from pneumomediastinum (positive pressure breathing, rupture of bulla / bleb, chest surgery)2.Bronchopleural fistula(c)rupture of urinary bladder(d)penetrating abdominal injuryC.THROUGH FEMALE GENITAL TRACT*(a)iatrogenic1.Perforation of uterus / vagina2.Culdocentesis3.Rubin test = tubal patency test4.Pelvic examination(b)spontaneous1.Intercourse, orogenital insufflation2.Douching3.Knee-chest exercise, water skiing, horseback ridingD.INTRAPERITONEAL1.Gasforming peritonitis2.Rupture of abscessNote * =asymptomatic spontaneous pneumoperitoneum without peritonitis√ air in lesser peritoneal sac√ gas in scrotum (through open processus vaginalis) Large collection of gas: √ abdominal distension, no gastric air-fluid level√ "football sign" = large pneumoperitoneum outlining entire abdominal cavity√ "double wall sign" = "Rigler sign" = "bas-relief sign"= air on both sides of bowel as intraluminal gas + free air outside (usually requires >1,000 mL of free intraperitoneal gas + intraperitoneal fluid) √ "telltale triangle sign" = triangular air pocket between 3 loops of bowel√ depiction of diaphragmatic muscle slips = two or three 6-13 cm long and 8-10 mm wide arcuate soft-tissue bands directed vertically inferiorly + arching parallel to diaphragmatic dome superiorly√ outline of ligaments of anterior inferior abdominal wall:√ "inverted V sign" = outline of both lateral umbilical ligaments (containing inferior epigastric vessels)√ outline of medial umbilical ligaments (obliterated umbilical arteries)√ "urachus sign" = outline of middle umbilical ligament
RUQ gas (best place to look for small collections): √ single large area of hyperlucency over the liver√ oblique linear area of hyperlucency outlining the posteroinferior margin of liver√ doge's cap sign = triangular collection of gas in Morison pouch (posterior hepatorenal space)√ outline of falciform ligament = long vertical line to the right of midline extending from ligamentum teres notch to umbilicus; most common structure outlined√ ligamentum teres notch = inverted V-shaped area of hyperlucency along undersurface of liver√ ligamentum teres sign = air outlining fissure of ligamentum teres hepatis (= posterior free edge of falciform ligament) seen as vertically oriented sharply defined slitlike / oval area of hyperlucency between 10th and 12th rib within 2.5-4.0 cm of right vertebral border 2-7 mm wide and 6-20 mm long√ "saddlebag / mustache / cupola sign" = gas trapped below central tendon of diaphragm√ parahepatic air = gas bubble lateral to right edge of liver

Notes:

Pseudopneumoperitoneum =process mimicking free airA.ABDOMINAL GAS(a)gastrointestinal gas1.Pseudo-wall sign = apposition of gas-distended bowel loops2.Chilaiditi syndrome3.Diaphragmatic hernia4.Diverticulum of esophagus / stomach / duodenum(b)extraintestinal gas1.Retroperitoneal air2.Subdiaphragmatic abscessB.CHEST1.Pneumothorax2.Empyema3.Irregularity of diaphragmC.FAT1.Subdiaphragmatic intraperitoneal fat2.Interposition of omental fat between liver + diaphragm

Notes:

Pneumoretroperitoneum *Cause:*(1)Traumatic rupture (usually duodenum)(2)Perforation of duodenal ulcer(3)Gas abscess of pancreas (usually extends into lesser sac)(4)Urinary tract gas (trauma, infection)(5)Dissected mediastinal air√ kidney outlined by gas√ outline of psoas margin ± gas streaks in muscle bundles

Notes:

Pneumatosis Intestinalis =PNEUMATOSIS CYSTOIDES INTESTINALIS = BULLOUS EMPHYSEMA OF THE INTESTINE = INTESTINAL GAS CYSTS = PERITONEAL LYMPHOPNEUMATOSIS◊Attributed to at least 58 causative factors!A.BOWEL NECROSIS / GANGRENE◊Most common + life-threatening cause!*Pathogenesis:*damage + disruption of mucosa with entry of gasforming bacteria into bowel wall (cysts contain 50% hydrogen = evidence of bacterial origin)necrotizing enterocolitis, ischemia + infarction (mesenteric thrombosis), neutropenic colitis, sepsis, volvulus, emphysematous gastritis, caustic ingestion B.MUCOSAL DISRUPTION*Pathogenesis:*increased intestinal gas pressure leads to overdistension and dissection of gas into bowel wall(a)intestinal obstruction:pyloric stenosis, annular pancreas, imperforate anus, Hirschsprung disease, meconium plug syndrome, obstructing neoplasm (b)intestinal trauma:endoscopy ± biopsy, biliary stent perforation, sclerotherapy, bowel surgery, postoperative bowel anastomosis, penetrating / blunt abdominal trauma, trauma of child abuse, intracatheter jejunal feeding tube, barium enema (c)infection / inflammation:peptic ulcer disease, intestinal parasites, tuberculosis, peritonitis, inflammatory bowel disease (Crohn disease, ulcerative colitis, pseudomembranous colitis), ruptured jejunal diverticula, Whipple disease, systemic amyloidosis D.INCREASED MUCOSAL PERMEABILITY*Pathogenesis:*defects in lymphoid tissue of bowel wall allows bacterial gas to enter bowel wall(a)immunotherapy: graft-versus-host disease, organ transplantation, bone marrow transplantation (b)others:AIDS enterocolitides, steroid therapy, chemotherapy, radiation therapy, collagen vascular disease (scleroderma, systemic lupus erythematosus, periarteritis dermatomyositis), intestinal bypass enteropathy, diabetes mellitus C.PULMONARY DISEASE*Pathogenesis:*alveolar rupture with air dissecting interstitially along bronchovascular bundles to mediastinum + retroperitoneally along vascular supply of visceraChronic obstructive pulmonary disease (chronic bronchitis, emphysema, bullous disease of lung), asthma, cystic fibrosis, chest trauma (barotrauma from artificial ventilation, chest tube), increased intrathoracic pressure associated with retching + vomiting *Path:*(a)microvesicular type = 10-100 mm cysts / bubbles within lamina propria(b)linear / curvilinear type = streaks of gas oriented parallel to bowel wallLocation:any part of GI tract; may be discontinuous with spread to distant sites along mesenterySite:subserosa > submucosa > muscularis > mesentery; mesenteric side >> antimesenteric side√ radiolucent clusters of cysts along contour of bowel wall (best demonstrated on CT)√ segmental mucosal nodularity (DDx: polyposis)√ ± pneumoperitoneum / pneumoretroperitoneum (asymptomatic large pneumoperitoneum may persist for months / years)√ ±gas in mesenteric + portal vein*Prognosis:* wide spectrum from innocuous to fatal; clinical outcome impossible to predict based on x-ray findings ◊linear gas collections have probably a more severe connotation◊pneumatosis of the colon is likely clinically insignificant◊extent of pneumatosis is inversely related to severity of disease

Notes:

Soap-bubble Appearance In Abdomen Of Neonate 1.Feces in infant fed by mouth2.Meconium ileus:gas mixed with meconium, usually RLQ3.Meconium plug:gas in and around plug, in distribution of colon4.Necrotizing enterocolitis: submucosal pneumatosis5.Atresia / severe stenosis: pneumatosis6.Hirschsprung disease:impacted stool, sometimes pneumatosis

Notes:

Opaque Material In Bowel *mnemonic:*"CHIPS"**C**hloral hydrate **H**eavy metals (lead) **I**ron **P**henothiazines **S**alicylates

Notes:

Diffuse Abdominal Calcifications 1.Cystadenoma of ovary√ granular, sandlike psammomatous calcifications2.Pseudomyxoma peritonei(a)pseudomucinous adenoma of ovary(b)mucocele of appendix3.Undifferentiated abdominal malignancy4.Tuberculous peritonitis√ mottled calcifications, simulating residual barium5.Meconium peritonitis6.Oil granuloma√ annular, plaquelike

Notes:

Focal Alimentary Tract Calcifications A.ENTEROLITHS1.Appendicolith: in 10-15% of acute appendicitis2.Stone in Meckel diverticulum3.Diverticular stone4.Rectal stone5.Proximal to partial obstruction (eg, tuberculosis, Crohn disease)B.MESENTERIC CALCIFICATIONS1.Dystrophic calcification of omental fat deposits + appendices epiploicae (secondary to infarction / pancreatitis / TB)2.Cysts: mesenteric cyst, hydatid cyst3.Calcified mesenteric lipomaC.INGESTED FOREIGN BODIEStrapped in appendix, diverticula, proximal to stricture 1.Calcified seeds + pits (bezoar)2.BirdshotD.TUMOR1.Mucocele of appendix√ crescent-shaped / circular calcification2.Mucinous adenocarcinoma of stomach / colon= COLLOID CARCINOMA √ small mottled / punctate calcifications in primary site ± in regional lymph node metastases, adjacent omentum, metastatic liver foci3.Gastric / esophageal leiomyoma: calcifies in 4%4.Lipoma

Notes:

Abdominal Wall Calcifications A.IN SOFT TISSUES1.Hypercalcemic states2.Idiopathic calcinosisB.IN MUSCLE(a)parasites:1.Cysticercosis = Taenia solium√ round / slightly elongated calcifications2.Guinea worm = dracunculiasis√ stringlike calcifications up to 12 cm long(b)injection sitesfrom quinine, bismuth, calcium gluconate, calcium penicillin (c)myositis ossificansC.IN SKIN1.Soft-tissue nodules: papilloma, neurofibroma, melanoma, nevi2.Scar:√ linear density3.Colostomy / ileostomy4.Tattoo markings

Notes:

Abdominal Vascular Calcifications A.ARTERIES1.Atheromatous plaques2.Arterial calcifications in diabetes mellitusB.VEINSphleboliths = calcified thrombus, generally seen below interspinous line 1.normal / varicose veins2.hemangiomaC.LYMPH NODES1.Histoplasmosis / tuberculosis2.Chronic granulomatous disease3.Residual lymphographic contrast4.Silicosis

Notes:

Ascites A.TRANSUDATE:(1) Cirrhosis (75%): poor prognostic sign (2) Hypoproteinemia, (3) CHF, (4) Constrictive pericarditis, (5) Chronic renal failure, (6) Budd-Chiari syndrome B.EXUDATE:(1) Carcinomatosis, (2) Polyserositis, (3) TB peritonitis, (4) Pancreatitis, (5) Meigs syndrome C.HEMORRHAGIC / CHYLOUS FLUID Early signs (accumulation in pelvis): √ round central density in pelvis + ill-defined bladder top√ thickening of peritoneal flank stripe√ space between properitoneal fat and gut >3 mmLate signs: √ Hellmer sign = medial displacement of lateral liver margins√ medial displacement of ascending + descending colon√ obliteration of hepatic + splenic angles√ bulging flanks√ gray abdomen√ floating centralized loops√ separation of loops ***High-density Ascites*** 1.Tuberculosis: 20-45 HU; may be lower2.Ovarian tumor3.Appendiceal tumor ***Neonatal Ascites*** A.GASTROINTESTINAL(a)perforation of hollow viscusmeconium peritonitis (b)inflammatory lesionsMeckel diverticulum, appendicitis (c)cyst rupturemesenteric / omental / choledochal cyst (d)bile leakagebiliary obstruction / perforation B.PORTOHEPATIC(a)extrahepatic portal vein obstructionatresia of veins, compression by mass (b)intrahepatic portal vein obstructionportal cirrhosis (neonatal hepatitis), biliary cirrhosis (biliary atresia) C.URINARY TRACT◊Urine ascites (most common cause) from lower urinary tract obstruction + upper urinary tract rupture: posterior / anterior urethral valves, ureterovesical / ureteropelvic junction obstruction, renal / bladder rupture, anterior urethral diverticulum, bladder diverticula, neurogenic bladder, extrinsic bladder massD.GENITALruptured ovarian cyst, hydrometrocolpos E.HYDROPS FETALISimmune hydrops, nonimmune hydrops (usually cardiac causes) F.MISCELLANEOUSchylous ascites, lymphangiectasia, congenital syphilis, trauma, idiopathic ***Chylous Ascites*** IN ADULTS:1.Inflammatory process(35%)2.Tumor(30%)3.Idiopathic(23%)4.Trauma(11%)5.Congenital(1%)IN CHILDREN:1.Congenital(39%)2.Inflammatory process(15%)3.Trauma(12%)4.Tumor(3%)5.Idiopathic(33%)

Notes:

Fluid Collections *mnemonic:* "BLUSCHINGS"**B**iloma **L**ymphocele, **L**ymphangioma, **L**ymphoma (almost anechoic by US) **U**rinoma **S**eroma **C**yst (pseudocyst, peritoneal inclusion cyst) **H**ematoma (aneurysm, AVM) **I**nfection, **I**nfestation (empyema, abscess, Echinococcus) **N**eoplasm (necrotic) **GI** tract (dilated loops, ileus, duplication) **S**erosa (ascites, pleural fluid, pericardial effusion)

Notes:

Intra-abdominal Cyst In Childhood 1.Omental cyst (greater omentum / lesser sac, multilocular)2.Mesenteric cyst (between leaves of small bowel mesentery)3.Choledochal cyst4.Intestinal duplication5.Ovarian cyst6.Pancreatic pseudocyst7.Cystic renal tumor8.Abscess9.Meckel diverticulum (communicates with GI tract)10.Lymphangioma11.Mesenteric lymphoma12.Intramural tumor

Notes:

MECHANICAL INTESTINAL OBSTRUCTION
=occlusion / constriction of bowel lumen

Common Causes Of Obstruction In Children Gastric Outlet Obstruction Duodenal Obstruction Jejunal And Ileal Obstruction Colonic Obstruction

Notes:

Common Causes Of Obstruction In Children NurseryIntestinal atresia, midgut volvulus, meconium ileus, Hirschsprung disease, small bowel atresia with meconium ileus, meconium plug syndrome, small left colon syndrome, imperforate anus, obstruction from duplication cystFirst 3 monthsInguinal hernia, Hirschsprung disease, midgut volvulus6 - 24 monthsIleocolic intussusceptionChildhoodAppendicitis

Notes:

Gastric Outlet Obstruction A.CONGENITAL LESION1.Antral mucosal diaphragm = antral web2.Gastric duplication: usually along greater curvature, abdominal mass in infancy3.Hypertrophic pyloric stenosisB.INFLAMMATORY NARROWING1.Peptic ulcer disease: cause in adults in 60-65%2.Corrosive gastritis3.Crohn disease, sarcoidosis, syphilis, tuberculosisC.MALIGNANT NARROWING1.Antral carcinoma: cause in adults in 30-35%2.Scirrhous carcinoma of pyloric channelD.OTHERS1.Prolapsed antral polyp / mucosa2.Bezoar3.Gastric volvulus4.Postoperative stomal edemaAbdominal plain film: √ large smoothly marginated homogeneous mass displacing transverse colon + small bowel inferiorly√ one / two air-fluid levels

Notes:

Duodenal Obstruction A.CONGENITAL1.Annular pancreas2.Peritoneal bands = Ladd bands3.Aberrant vesselB.INFLAMMATORY NARROWING1.Chronic duodenal ulcer scar2.Acute pancreatitis: phlegmon, abscess, pseudocyst3.Acute cholecystitis: perforated gallstoneC.INTRAMURAL HEMATOMA1.Blunt trauma (accident, child abuse)2.Anticoagulant therapy3.Blood dyscrasiaD.TUMORAL NARROWING1.Primary duodenal tumors2.Tumor invasion from pancreas, right kidney, lymph node enlargementE.EXTRINSIC COMPRESSION1.Aortic aneurysm2.PseudoaneurysmF.OTHERS1.Superior mesenteric artery syndrome from extensive burns, body cast, rapid weight loss, prolonged bed rest2.Bezoar (in gastrectomized patient) *mnemonic:*"VA BADD TU BADD"childadult **V**olvulus**T**umor**A**tresia**U**lcer**B**ands**B**ands**A**nnular pancreas**A**nnular pancreas**D**uplication**D**uplication**D**iverticulum**D**iverticulum Abdominal plain film: √ double-bubble sign = air-fluid levels in stomach + duodenum√ frequently normal due to absence of gas from vomiting

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Notes:

Jejunal And Ileal Obstruction =SMALL BOWEL OBSTRUCTION (SBO)*Technique:*best evaluated by CT (95% accurate, 94% sensitive, 96% specific)A.CONGENITAL1.Ileal atresia / stenosis2.Enteric duplication: located on antimesenteric side, mostly in ileum3.Midgut volvulus from arrest in rotation + fixation of small bowel during fetal life4.Mesenteric cyst from meconium peritonitis:located on mesenteric side 5.Meckel diverticulumB.EXTRINSIC BOWEL LESIONS1.Fibrous adhesions from previous surgery / peritonitis (in 75%) 2.Hernias (inguinal, femoral, umbilical, paraduodenal, foramen of Winslow, incisional, Spigelian, obturator)3.Volvulus4.Masses: neoplasm, abscessC.LUMINAL OCCLUSION1.Swallowed foreign body, bezoar, gallstone, bolus of Ascaris lumbricoides, inspissated milk2.Meconium ileus:√ microcolon in cystic fibrosis 3.Meconium ileus equivalent4.Intussusception (tumor, Meckel diverticulum, chronic ulcer, adhesion)5.Tumor (rare): eg, lipomaD.INTRINSIC BOWEL WALL LESION1.Strictures from neoplasm, Crohn disease, tuberculous enteritis, parasitic disease, potassium chloride tablets, surgical anastomosis, irradiation, massive deposition of amyloid2.Intramural hemorrhage: blunt trauma, Henoch-Schönlein purpura3.Vascular insufficiency: arterial / venous occlusion
Acquired Small Bowel Obstruction In Childhood *mnemonic:* "AAIIMM"**A**dhesions **A**ppendicitis **I**ntussusception **I**ncarcerated hernia **M**alrotation **M**eckel diverticulum
Small Bowel Obstruction In Adulthood *mnemonic:*"SHAVIT"**S**tone (gallstone ileus) **H**ernia **A**dhesion **V**olvulus **I**ntussusception **T**umor Plain abdominal radiograph (50-66% sensitive): √ "candy cane" appearance in erect position = >3 distended small bowel loops >3 cm with gas-fluid levels (>3-5 hours after onset of obstruction)√ disparity in size between obstructed loops and contiguous small bowel loops of normal caliber beyond site of obstruction√ small bowel positioned in center of abdomen √ little / no gas + stool in colon with complete mechanical obstruction after 12-24 hours√ "stretch sign" = erectile valvulae conniventes completely encircle bowel lumen √ "stepladder appearance" in low obstruction (the greater the number of dilated bowel loops, the more distal the site of obstruction)√ "string-of-beads" indicate peristaltic hyperactivity to overcome mechanical obstruction√ hyperactive peristalsis / aperistalsis = fatigued small bowelCAVE:little / no gas in small bowel from fluid-distended loops may lead one to overlook obstruction *Plain abdominal radiographic categories:* 1.Normal=absence of small intestinal gas / gas within 3-4 variably shaped loops <2.5 cm in diameter2.Mild small bowel stasis=single / multiple loops of 2.5-3 cm in diameter with ≥3 air-fluid levels3.Probable SBO pattern=dilated multiple gas- / fluid-filled loops with air-fluid levels + moderate amount of colonic gas4.Definite SBO pattern=clearly disproportionate gaseous / fluid distension of small bowel relative to colon UGI: √ "snake head" appearance = active peristalsis forms bulbous head of barium column in an attempt to overcome obstruction√ barium appears in colon >12 hours Enteroclysis for adhesive obstruction: √ abrupt change in caliber of bowel with normal caliber / collapsed bowel distal to obstruction√ stretched folds of normal pattern√ angulated + fixed bowel segment*Enteroclysis categories of SBO (Shrake):* (a)low-grade partial SBO=sufficient flow of contrast material through point of obstruction so that fold pattern beyond obstruction is readily defined(b)high-grade partial SBO=stasis + delay in arrival of contrast so that contrast material is diluted in distended prestenotic loop with minimal contrast in postobstructive loop leading to difficulty in defining fold pattern after transition point(c)complete SBO=no passage of contrast material 3-24 hours after start of examination CT (poor sensitivity for low-grade partial obstruction) US: √ small bowel loops dilated >3 cm√ length of dilated segment >10 cm√ increased peristalsis of dilated segment (may become paralytic in prolonged obstruction)√ colon collapsed Location of obstruction: (a)valvulae conniventes high + frequent = jejunum(b)valvulae conniventes sparse / absent = ileum

Closed Loop Obstruction =bowel obstruction at two points*Cause:*adhesion (75%), volvulus, incarcerated hernia√ U-shaped distended loop√ increasing intraluminal fluid√ fixation of bowel loop = no change in position√ "coffee bean sign" = gas-filled loop√ "pseudotumor" = fluid-filled loop√ U- or C-shaped dilated bowel loop on CT√ "beak sign" = point of obstruction on CT / UGI√ "whirl sign" = twisting of bowel + mesentery on CT√ stretched mesenteric vessels converging toward torsion
Strangulated Obstruction =triad of (1) mechanical obstruction proximal to the involved segment (2) closed-loop obstruction of the involved segment (3) venous congestion of the involved segmentCT: √ slight circumferential thickening of bowel wall√ increased wall attenuation√ target / halo sign√ serrated beak at site of obstruction (32-100% specific)√ unusual course of mesenteric vasculature√ mesenteric haziness due to edema (95% specific)√ diffuse engorgement of mesenteric vasculature√ poor / no enhancement of bowel wall (100% specific)√ delayed prolonged enhancement of bowel wall√ large amount of ascites√ pneumatosis intestinalis

Notes:

Colonic Obstruction *Incidence:*25% of all intestinal obstructionsA.NEONATAL COLONIC OBSTRUCTION1.Meconium plug syndrome2.Colonic atresia3.Anorectal malformation: rectal atresia, imperforate anusB.LUMINAL OBTURATION1.Fecal impaction√ bubbly pattern of large mass of stool2.Fecaloma3.Gallstone (in sigmoid narrowed by diverticulitis)4.IntussusceptionC.BOWEL WALL LESION(a)malignant (60-70% of obstructions):predominantly in sigmoid (b)inflammatory1.Crohn disease2.Ulcerative colitis3.Mesenteric ischemia4.Sigmoid diverticulitis (15%)√ stenotic segment >6 cm5.Acute pancreatitis(c)infectious:infectious granulomatous process (actinomycosis, tuberculosis, lymphogranuloma venereum), parasitic disease (amebiasis, schistosomiasis) (d)wall hematoma:blunt trauma, coagulopathy D.EXTRINSIC (a)mass impression1.Endometriosis2.Large tumor mass: prostate, bladder, uterus, tubes, ovaries3.Pelvic abscess4.Hugely distended bladder5.Mesenteritis6.Poorly formed colostomy (b)severe constriction1.Volvulus (3rd most common cause): sigmoid colon, cecum, transverse colon, compound volvulus (= ileosigmoid knot)2.Hernia: transverse colon in diaphragmatic hernia, sigmoid colon in left inguinal hernia3.Adhesion *Abdominal plain-film patterns:* (a)dilated colon only = competent ileocecal valve(b)dilated small bowel (25%) = incompetent ileocecal valve(c)dilated colon + dilated small bowel = ileocecal valve obstruction secondary to cecal overdistension√ gas-fluid levels distal to hepatic flexure (fluid is normal in cecum + ascending colon); sign not valid with diarrhea / saline catharsis / enema√ cecum most dilated portion (in 75% of cases); critical at 10 cm diameter (high probability for impending perforation)√The lower the obstruction, the more proximal the distension!BE:emergency barium enema of unprepared colon in suspected obstruction!contraindicated in toxic megacolon, pneumatosis intestinalis, portal vein gas, extraluminal gas

Notes:

ILEUS

[ileus = stasis / inability to push fluid along (term does not distinguish between mechanical and nonmechanical causes)] =ADYNAMIC / PARALYTIC / NONOBSTRUCTIVE ILEUS=derangement impairing proper distal propulsion of intestinal contents *Cause:* -in neonate:1.Hyperbilirubinemia2.Intracranial hemorrhage3.Aspiration pneumonia4.Necrotizing enterocolitis5.Aganglionosis-in child / adult:1.Postoperative ileus ● usually resolves by 4th postoperative day2.Visceral pain: obstructing ureteral stone, common bile duct stone, twisted ovarian cyst, blunt abdominal / chest trauma3.Intra-abdominal inflammation / infection: peritonitis, appendicitis, cholecystitis, pancreatitis, salpingitis, abdominal abscess, hemolytic-uremic syndrome, gastroenteritis4.Ischemic bowel disease5.Anticholinergic drugs: atropine, propantheline, morphine + derivatives, tricyclic antidepressants, dilantin, phenothiazines, hexamethonium bromide6.Neuromuscular disorder: diabetes, hypothyroidism, porphyria, lead poisoning, uremia, hypokalemia, amyloidosis, urticaria, sprue, scleroderma, Chagas disease, vagotomy, myotonic dystrophy, CNS trauma, paraplegia, quadriplegia7.Systemic disease: septic / hypovolemic shock, urticaria8.Chest disease: lower lobe pneumonia, pleuritis, myocardial infarction, acute pericarditis, congestive heart failure9.Retroperitoneal disease: hemorrhage (spine trauma), abscess *mnemonic:*"Remember the P's"**P**ancreatitis **P**endicitis **P**eptic ulcer **P**erforation **P**eritonitis **P**neumonia **P**orphyria **P**ostoperative **P**otassium deficiency **P**regnancy **P**yelonephritis ● intestinal sounds decreased / absent ● abdominal distension√ large + small bowel ± gastric distension√ decreased small bowel distension on serial films√ delayed but free passage of contrast material*Rx:*not amenable to surgical correction

Localized Ileus Intestinal Pseudoobstruction

Notes:

Localized Ileus =isolated distended loop of small / large bowel= SENTINEL LOOP Often associated with an adjacent acute inflammatory process *Etiology:* 1.Acute pancreatitis:duodenum, jejunum, transverse colon2.Acute cholecystitis:hepatic flexure of colon3.Acute appendicitis:terminal ileum, cecum4.Acute diverticulitis:descending colon5.Acute ureteral colic:GI tract along course of ureter

Notes:

Intestinal Pseudoobstruction A.Transient pseudoobstruction1.Electrolyte imbalance2.Renal failure3.Congestive heart failureB.Chronic pseudoobstruction1.Scleroderma2.AmyloidosisC.Idiopathic pseudoobstruction1.Chronic intestinal pseudoobstruction syndrome ● persistently decreased peristalsis + clinical obstruction*Age:*neonatal period / delayed for months + years2.Megacystis-microcolon-intestinal-hypoperistalsis syndrome

Notes:

Esophageal Contractions ◊Esophageal motor activity needs to be evaluated in recumbent position without influence of gravity!PERISTALTIC EVENT = coordinated contractions of esophagus PERISTALTIC SEQUENCE = aboral stripping wave clearing esophagus A.PRIMARY PERISTALSIS=orderly peristaltic sequence with progressive aboral stripping traversing entire esophagus with complete clearance of barium; centrally mediated (medulla) swallow reflex via glossopharyngeal + vagal nerve; initiated by swallowing√ rapid wave of inhibition followed by slower wave of contraction◊Normal peristaltic sequence will be interrupted by repetitive swallowing before peristaltic sequence is complete!B.SECONDARY PERISTALSIS=local peristaltic wave identical to primary peristalsis but elicited through esophageal distension = sensorimotor stretch reflex◊Esophageal motility can be evaluated with barium injection through nasoesophageal tube despite patient's inability to swallow!C.TERTIARY CONTRACTIONS=nonpropulsive esophageal motor event characterized by disordered up-and-down movement of bolus without clearing of esophagus *Cause:* 1.Presbyesophagus2.Diffuse esophageal spasm3.Hyperactive achalasia4.Neuromuscular disease:diabetes mellitus, Parkinsonism, amyotrophic lateral sclerosis, multiple sclerosis, thyrotoxic myopathy, myotonic dystrophy5.Obstruction of cardia:neoplasm, distal esophageal stricture, benign lesion, S/P repair of hiatal hernia◊Tertiary activity does not necessarily imply a significant motility disturbance!*Age:*in 5-10% of normal adults during 4th-6th decade(a)nonsegmental = partial luminal indentationLocation: in lower 2/3 of esophagus √ spontaneous repetitive nonpropulsive contraction√ "yo-yo" motion of barium√ "corkscrew" appearance = scalloped configuration of barium column√ "rosary bead" / "shish kebab" configuration= compartmentalization of barium column√ no lumen-obliterating contractions(b)segmental = luminal obliteration (rare)√ "curling" = erratic segmental contractions√ "rosary-bead" appearance

Notes:

Abnormal Esophageal Peristalsis A.PRIMARY MOTILITY DISORDERS1.Achalasia2.**Diffuse esophageal spasm**

- severe intermittent pain while swallowing√ compartmentalization of esophagus by numerous tertiary contractions*Dx:*extremely high pressures on manometry3.Presbyesophagus4.Chalasia5.Congenital TE fistula6.Intestinal pseudoobstructionB.SECONDARY MOTILITY DISORDERS(a)Connective tissue disease1.Scleroderma2.SLE3.Rheumatoid arthritis4.Polymyositis5.Dermatomyositis6.Muscular dystrophy(b)Chemical / physical injury1.Reflux / peptic esophagitis2.S/P vagotomy3.Caustic esophagitis4.Radiotherapy(c)InfectionFungal: candidiasisParasitic: Chagas diseaseBacterial:TB, diphtheriaViral:herpes simplex(d)Metabolic disease1.Diabetes mellitus2.Amyloidosis3.Alcoholism4.Electrolyte disturbances(e)Endocrine disease1.Myxedema2.Thyrotoxicosis(f)Neoplasm(g)Drug-relatedatropine, propantheline, curare (h)Muscle disease1.Myotonic dystrophy2.Muscular dystrophy3.Oculopharyngeal dystrophy4.Myasthenia gravis (disturbed motility only in striated muscle of upper 1/3 of esophagus)√ persistent collection of barium in upper third of esophagus√ findings reversed by cholinesterase inhibitor edrophonium (Tensilon®)(i)Neurologic disease1.Parkinsonism2.Multiple sclerosis3.CNS neoplasm4.Amyotrophic lateral sclerosis5.Bulbar poliomyelitis6.Cerebrovascular disease7.Huntington chorea8.Ganglioneuromatosis9.Wilson disease10.Friedreich ataxia11.Familial dysautonomia (Riley-Day)12.Stiff-man syndrome

Notes:

Diffuse Esophageal Dilatation =ACHALASIA PATTERN = MEGAESOPHAGUSA.ESOPHAGEAL MOTILITY DISORDER1.Idiopathic achalasia2.Chagas disease: patients commonly from South America; often associated with megacolon + cardiomegaly3.Postvagotomy syndrome4.Scleroderma5.Systemic lupus erythematosus6.Presbyesophagus7.Ehlers-Danlos syndrome8.Diabetic / alcoholic neuropathy9.Anticholinergic drugs10.Idiopathic intestinal pseudoobstruction= degeneration of innervation11.Amyloidosis: associated with macroglossia, thickened small bowel folds12.EsophagitisB.DISTAL OBSTRUCTION1.Infiltrating lesion of distal esophagus / gastric cardia (eg, carcinoma) = pseudoachalasia2.Benign stricture3.Extrinsic compression *mnemonic:*"MA'S TACO in a SHell"**M**uscular disorder (eg, myasthenia gravis) **A**chalasia **S**cleroderma **T**rypanosomiasis (Chagas disease) **A**myloidosis **C**arcinoma **O**bstruction **S**tricture (lye, potassium, tetracycline) **H**iatal hernia

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Notes:

Air Esophagogram 1.Normal variant2.Scleroderma3.Distal obstruction: tumor, stricture, achalasia4.Thoracic surgery5.Mediastinal inflammatory disease6.S/P total laryngectomy (esophageal speech)7.Endotracheal intubation + PEEP

Notes:

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Abnormal Esophageal Folds A.TRANSVERSE FOLDS1.**Feline esophagus**
frequently seen with gastroesophageal reflux; normally found in cats √ transient contraction of longitudinally oriented muscularis mucosae2.Fixed transverse foldsdue to scarring from reflux esophagitis √ stepladder appearance in distal esophagusB.LONGITUDINAL FOLDSnormally 1-2 mm wide in collapsed esophagus; >3 mm with submucosal edema / inflammation 1.Gastroesophageal reflux2.Opportunistic infection3.Caustic ingestion4.Irradiation*DDx:*1.Varices √ tortuous / serpentine folds that can be effaced by esophageal distension2.Varicoid carcinoma √ fixed rigid folds with abrupt demarcation due to submucosal spread

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Notes:

Esophageal Inflammation A.CONTACT INJURY(a)reflux related1.Peptic ulcer disease2.Barrett esophagus3.Scleroderma (patulous LES)4.Nasogastric intubation(b)caustic1.Foreign body2.Corrosives(c)thermicHabitual ingestion of excessively hot meals / liquids B.RADIATION INJURYC.INFECTION1.Candidiasis2.Herpes simplex virus / CMV3.DiphtheriaD.SYSTEMIC DISEASE(a)dermatologic disorderspemphigoid, epidermolysis bullosa (b)others:1.Crohn disease2.Graft-versus-host disease3.Behçet disease4.Eosinophilic gastroenteritis

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Notes:

Esophageal Ulceration A.PEPTIC1.Reflux esophagitis: scleroderma√ shallow / deep ulcers in distal esophagus2.Barrett esophagus3.Crohn disease√ aphthous ulcers in variable location4.Dermatologic disorders: benign mucous membrane pemphigoid, epidermolysis bullosa dystrophica, Behçet diseaseB.INFECTIOUS1.Herpes√ discrete superficial ulcers in midesophagus2.Cytomegalovirus √ large flat ulcer in mid- or distal esophagusC.CONTACT INJURY / EXTERNAL INJURY1.Corrosives: alkali, strictures in 50%2.Alcohol-induced esophagitis3.Drug-induced = "pill esophagitis":(a)antibiotics (tetracyclines), quinidine, potassium chloride√ discrete superficial ulcers in midesophagus(b)alendronate (= inhibitor of osteoclastic activity)√ long-segment involvement with severe ulceration4.Radiotherapy: smooth stricture >4500 rads√ shallow / deep ulcers conforming to radiation portal5.Nasogastric tube√ elongated stricture in middle + distal 1/36.Endoscopic sclerotherapyD.MALIGNANT1.Esophageal carcinoma Location: @Upper esophagus1.Barrett ulcer in islets of gastric mucosa@Midesophagus1.Herpes esophagitis2.CMV esophagitis3.Drug-induced esophagitis@Distal esophagus1.Reflux esophagitis2.CMV esophagitis*DDx:* 1.Sacculation=outpouching in distal esophagus due to asymmetric scarring in reflux esophagitis2.Esophageal intramural pseudodiverticula3.Artifact(a)tiny precipitates of barium(b)transient mucosal crinkling in inadequate distension(c)irregular Z-line

Notes:

Double-barrel Esophagus 1.Dissecting intramural hematoma from emetogenic injury2.Mallory-Weiss teartrauma, esophagoscopy (in 0.25%), bougienage (in 0.5%), ingestion of foreign bodies, spontaneous (bleeding diathesis) 3.Intramural abscess4.Intraluminal diverticulum5.Esophageal duplication (if communication with esophageal lumen present)

Notes:

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Esophageal Diverticulum 1.ZENKER DIVERTICULUM (pharyngoesophageal)2.INTERBRONCHIAL DIVERTICULUM=traction diverticulumresponse to pull from fibrous adhesions following lymph node infection (TB), contains all 3 esophageal layers Location:usually on right anterolateral wall of interbronchial segment√ calcified mediastinal nodes3.INTERAORTICOBRONCHIAL DIVERTICULUM= thoracic pulsion diverticulum Location:on left anterolateral wall between inferior border of aortic arch + upper margin of left main bronchus4.EPIPHRENIC DIVERTICULUM (rare)Location:usually on lateral esophageal wall, right > left, in distal 10 cm√ often associated with hiatus hernia5.INTRAMURAL ESOPHAGEAL PSEUDODIVERTICULOSIS√ outpouching from mucosal glands

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Notes:

Tracheobronchoesophageal Fistula A.CONGENITAL1.Congenital tracheoesophageal fistulaB.MALIGNANT1.Lung cancer2.Metastases to mediastinal lymph nodes3.Esophageal cancerOften following radiation treatment of these tumors! C.TRAUMATIC1.Instrumentation (esophagoscopy, bougienage, pneumatic dilatation)2.Blunt ("crush injury") / penetrating chest trauma3.Surgery4.Foreign-body perforation5.Corrosives6.Postemetic rupture = Boerhaave syndromeD.INFECTIOUS / INFLAMMATORY1.TB, syphilis, histoplasmosis, actinomycosis, Crohn disease2.Perforated diverticulum3.Pulmonary sequestration / cyst

Notes:

Long Smooth Esophageal Narrowing 1.Congenital esophageal stenosis√ at junction between middle + distal third√ weblike / tubular stenosis of 1 cm in length2.Surgical repair of esophageal atresia√ interruption of primary peristaltic wave at anastomosis√ secondary contractions may produce retrograde flow with aspiration√ impaction of food3.Caustic burns = alkaline burns4.Alendronate (= inhibitor of osteoclastic activity)5.Gastric acid: reflux, hyperemesis gravidarum6.Intubation: reflux + compromise of circulation7.Radiotherapy for esophageal carcinoma; tumor of lung, breast, or thymus; lymphoma; metastases to mediastinal lymph nodesOnset of stricture:usually 4-8 months post RxDose:3000-5000 rad8.Postinfectious: moniliasis (rare) ***Lower Esophageal Narrowing*** *mnemonic:*"SPADE"**S**cleroderma **P**resbyesophagus **A**chalasia; **A**nticholinergics **D**iffuse esophageal spasm **E**sophagitis

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Notes:

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Focal Esophageal Narrowing 1.**Web**
=1- to 2-mm thick (vertical length) area of complete / incomplete circumferential narrowing2.**Ring**
=5- to 10-mm thick (vertical length) area of complete / incomplete circumferential narrowing3.**Stricture**
=>10 mm in vertical length*mnemonic:*"LETTERS MC"**L**ye ingestion **E**sophagitis **T**umor **T**ube (prolonged nasogastric intubation) **E**pidermolysis bullosa **R**adiation **S**urgery, **S**cleroderma **M**oniliasis **C**ongenital

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Notes:

Esophageal Filling Defect A.BENIGN TUMORS<1% of all esophageal tumors (a)Submucosal tumor (75%)= nonepithelial, intramural 1.Leiomyoma (50% of all benign tumors)2.Lipoma, fibroma, lipoma, fibrolipoma, myxofibroma, hamartoma, hemangioma, lymphangioma, neurofibroma, schwannoma, granular cell myoblastoma√ primary wave stops at level of tumor√ proximal esophageal dilatation + hypotonicity√ rigid esophageal wall at site of tumoral implant√ disorganized / altered / effaced mucosal folds around defect√ tumor shadow on tangential view extending beyond esophageal margin(b)Mucosal tumor (25%) = epithelial, intraluminal1.Fibrovascular / inflammatory polyp; adenomatous polyp2.Squamous papilloma, fibropapilloma3.Villous adenoma, fibroadenoma√ no interruption of primary peristaltic wave√ well-circumscribed central radiolucent defect√ symmetric ampullary distension of esophagus around defect√ no change of mucosal pattern at periphery of defectB.MALIGNANT TUMORS1.Esophageal cancer, varicoid squamous cell carcinoma2.Gastric cancer3.Leiomyosarcoma, carcinosarcoma, pseudosarcoma4.Metastases: malignant melanoma, lymphoma(<1% of gastrointestinal lymphomas), stomach, lung, breast C.VASCULARvarices D.INFECTION / INFLAMMATIONCandida / herpes esophagitis, drug-induced inflammatory reaction E.CONGENITAL / NORMAL VARIANT1.Prolapsed gastric folds2.Esophageal duplication cyst(0.5-2.5% of all esophageal tumors) F.FOREIGN BODIESretained food particles (chicken bone, fish bone, pins, coins, small toys, meat), undissolved effervescent crystals, air bubbles

Notes:

Esophageal Mucosal Nodules / Plaques 1.Candida esophagitis√ diffuse / localized discrete plaques2.Reflux esophagitis (early stage)√ tiny poorly defined nodules in distal esophagus3.Barrett esophagus√ localized reticular pattern often adjacent to distal aspect of high stricture4.Glycogen acanthosis√ diffuse / localized nodules / plaques5.Superficial spreading carcinoma√ localized coalescent nodules / plaques6.Artifacts (undissolved effervescent agent, air bubbles, debris)

Notes:

Extrinsic Esophageal Impression ***Cervical Causes Of Esophageal Impression*** A.OSSEOUS LESIONS1.Anterior marginal osteophyte / DISH2.Anterior disk herniation3.Cervical trauma + hematoma4.Osteomyelitis5.Bone neoplasmB.ESOPHAGEAL WALL LESIONS(a)muscle1.Cricopharyngeus2.Esophageal web(b)vessel1.Pharyngeal venous plexus2.Lymph node enlargementC.ENDOCRINE ORGANS1.Thyroid / parathyroid enlargement (benign / malignant)2.Fibrotic traction after thyroidectomyD.Retropharyngeal / mediastinal abscess ***Thoracic Causes Of Esophageal Impression*** A.NORMAL INDENTATIONSaortic arch, left mainstem bronchus, left inferior pulmonary vein, diaphragmatic hiatus B.ABNORMAL VASCULATURERight-sided aortic arch, cervical aortic arch, aortic unfolding, aortic tortuosity, aortic aneurysm, double aortic arch ("reverse S"), coarctation of aorta ("reverse figure 3"), aberrant right subclavian artery =arteria lusoria (semilunar / bayonet-shaped imprint upon posterior wall of esophagus), aberrant left pulmonary artery (between trachea + esophagus), anomalous pulmonary venous return (anterior), persistent truncus arteriosus (posterior)C.CARDIAC CAUSES(a)enlargement of chambersleft atrial / left ventricular enlargement: mitral disease (esophageal displacement backward + to the right) (b)pericardial massespericardial tumor / cyst / effusion D.MEDIASTINAL CAUSESmediastinal tumor, lymphadenopathy (metastatic, tuberculous), inflammation, cyst E.PULMONARY CAUSESpulmonary tumor, bronchogenic cyst, atypical pulmonary fibrosis (retraction) F.ESOPHAGEAL ABNORMALITIES1.Esophageal diverticulum2.Paraesophageal hernia3.Esophageal duplication

Notes:

Widened Retrogastric Space A.PANCREATIC MASSES (most common cause)1.Acute + chronic pancreatitis2.Pancreatic pseudocyst3.Pancreatic cystadenoma + carcinomaB.OTHER RETROPERITONEAL MASSESsarcoma, renal tumor, adrenal tumor, lymph node enlargement, abscess, hematoma C.GASTRIC MASSES1.Leiomyoma, leiomyosarcomaD.OTHERS1.Aortic aneurysm2.Choledochal cyst3.Obesity4.Postsurgical disruptions + adhesions5.Ascites6.Gross hepatomegaly + enlarged caudate lobe7.Hernia involving omentum

Notes:

Gastric Pneumatosis A.INFECTION1.Emphysematous gastritisB.ISCHEMIA1.Gastric ulcer disease with intramural perforation2.Severe necrotizing gastroenteritis3.Gastric carcinoma4.Volvulus5.Gastric infarctionC.TRAUMA(a)Iatrogenic = gastric manipulation1.Recent gastroduodenal surgery2.Endoscopy (1.6%)(b)Ingested material:1.Corrosive gastritis2.Acid ingestionD.OVERDISTENSION (increased intraluminal pressure)1.Gastric outlet obstruction2.Volvulus3.Overinflation during gastroscopy4.Profuse severe vomitingE.DISSECTING AIR1.Rupture + dissection of subpleural blebs in bullous emphysema along esophageal wall / mediastinumF.IDIOPATHIC1.(Intramural / nonbacterial) gastric emphysema=cystic pneumatosis=benign idiopathic submucosal air lucencies√ thin discrete sharply defined streaks of gas in submucosa ± subserosa√ irregular radiolucent band of innumerable small bubbles with constant relationship to each other√ bulging of mucosa√ gas within portal venous system

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Notes:

Gastric Atony =gastric retention in the absence of mechanical obstruction*Pathophysiology:*reflex paralysis A.ACUTE GASTRIC ATONY(may develop within 24-48 hours) 1.Acute gastric dilatation: secondary to decreased arterial perfusion (ischemia, congestive heart failure) in old patients, usually fatal2.Postsurgical atony, ureteral catheterization3.Immobilization: body cast, paraplegia, postoperative state4.Abdominal trauma: especially back injury5.Severe pain: renal / biliary colic, migraine headaches, severe burns6.Infection: peritonitis, pancreatitis, appendicitis, subphrenic abscess, septicemiaB.CHRONIC GASTRIC ATONY1.Neurologic abnormalities: brain tumor, bulbar poliomyelitis, vagotomy, tabes2.Muscular abnormalities: scleroderma, muscular dystrophy3.Drug-induced atony: atropine, morphine, heroin, ganglionic blocking agents4.Electrolyte imbalance: diabetic ketoacidosis, hypercalcemia, hypocalcemia, hypokalemia, hepatic coma, uremia, myxedema5.Diabetes mellitus = gastroparesis diabeticorum (0.08% incidence)6.Emotional distress7.Lead poisoning8.Porphyria ● abdominal distension ● vascular collapse (decreased venous return) ● vomiting√ large stomach filled with air + fluid (up to 7,500 mL)√ retention of barium√ absent / diminished peristaltic activity√ patulous pylorus√ frequently dilated duodenum*DDx:*gastric volvulus, pyloric stenosis

Notes:

Narrowing Of Stomach =**linitis plastica** type of stenosisA.MALIGNANCY1.Scirrhous gastric carcinoma (involving portion / all of stomach)2.Hodgkin lymphoma, NHL3.Metastatic involvement (carcinoma of breast, pancreatic carcinoma, colonic carcinoma)B.INFLAMMATION1.Chronic gastric ulcer disease with intense spasm2.Pseudo-Billroth-I pattern of Crohn disease3.Sarcoidosis√ polypoid appearance, pyloric hypertrophy√ gastric ulcers, duodenal deformity 4.Eosinophilic gastritis5.Polyarteritis nodosa6.Stenosing antral gastritis / hypertrophic pyloric stenosisC.INFECTION1.Tertiary stage of syphilis√ absent mucosal folds + peristalsis√ no change over years2.Tuberculosis (rare)√ hyperplastic nodules / ulcerative lesion / annular lesion√ pyloric obstruction, may cross into duodenum3.Histoplasmosis4.Actinomycosis5.Strongyloidiasis6.Phlegmonous gastritis7.ToxoplasmosisD.TRAUMA1.Corrosive gastritis2.Radiation injury3.Gastric freezing4.Hepatic arterial chemotherapy infusionE.OTHERS1.Perigastric adhesions (normal mucosa, no interval change, normal peristalsis)2.Amyloidosis3.Pseudolymphoma4.Exogastric mass (hepatomegaly, pancreatic pseudocyst) *mnemonic:*"SLIMRAGE"**S**cirrhous carcinoma of stomach **L**ymphoma **I**nfiltration from adjacent neoplasm **M**etastasis (breast carcinoma) **R**adiation therapy **A**cids (corrosive ingestion) **G**ranulomatous disease (TB, sarcoidosis, Crohn) **E**osinophilic gastroenteritis
Antral Narrowing *mnemonic:*"SPICER"**S**arcoidosis, **S**yphilis **P**eptic ulcer disease **I**nfection (tuberculosis) **C**ancer, **C**rohn disease, **C**austic **E**osinophilic granuloma **R**adiation

Notes:

Radiology Review Manual
fourth edition

Intramural-extramucosal Lesions Of Stomach √ sharply delineated marginal / contour defect √ stretched folds over intact mucosa √ acute angle at margins √ may ulcerate centrally √ may become pedunculated and acquire polypoid appearance over years A.NEOPLASTIC1.Leiomyoma(48%)2.Neurogenic tumors(14%)3.Heterotopic pancreas(12%)4.Fibrous tumor(11%)5.Lipoma(7%)6.Hemangioma(7%)7.Glomus tumor(rare)8.Carcinoid9.Metastatic tumorB.INFLAMMATION / INFECTION1.Granuloma:(1) Foreign-body granuloma (2) Sarcoidosis (3) Crohn disease (4) Tuberculosis (5) Histoplasmosis 2.Eosinophilic gastritis3.Tertiary syphilis: infiltrative / ulcerative / tumorous type4.Echinococcal cystC.PANCREATIC ABNORMALITIES1.Ectopic pancreas2.Annular pancreas3.Pancreatic pseudocystD.DEPOSITS1.Amyloid2.Endometriosis3.Localized hematomaE.OTHERS1.Varices (ie, fundal)2.Duplications (4% of all GI tract duplications)

Notes:

Gastric Filling Defects A.INTRINSIC WALL LESIONS(a)benign (most common)1.Polyps: hyperplastic, adenomatous, villous, hamartomatous (Peutz-Jeghers syndrome, Cowden disease)2.Leiomyoma3.Granulomatous lesions:(a) Eosinophilic granuloma, (b) Crohn disease, (c) Tuberculosis ,(d) Sarcoidosis 4.Pseudolymphoma = benign reactive proliferation of lymphoid tissue5.Extramedullary hematopoiesis6.Ectopic pancreas7.Gastric duplication cyst8.Intramural hematoma9.Esophagogastric herniation(b)malignant1.Gastric carcinoma, lymphoma2.Gastric sarcoma: leiomyosarcoma, liposarcoma, leiomyoblastoma3.Gastric metastases: melanoma, breast, pancreas, colonB.EXTRINSIC IMPRESSIONS ON STOMACHin 70% nonneoplastic (extrinsic pseudotumors in 20%) (a)normal organs: organomegaly, tortuous aorta, heart, cardiac aneurysm(b)benign masses:cysts of pancreas, liver, spleen, adrenal, kidney; gastric duplication, postoperative deformity (eg, Nissen fundoplication) (c)malignant masses: enlarged celiac nodes(d)inflammatory lesion:left subphrenic abscess / hematoma -lateral displacement: enlarged liver, aortic aneurysm, enlarged celiac nodes-medial displacement: splenomegaly, mass in colonic splenic flexure, cardiomegaly, subphrenic abscessC.INTRALUMINAL GASTRIC MASSES1.Bezoar2.Foreign bodies: food, pills, blood clot, gallstoneD.TUMORS OF ADJACENT ORGANSpancreatic carcinoma + cystadenoma, liver carcinoma, carcinoma of gallbladder, colonic carcinoma, renal carcinoma, adrenal carcinoma, lymph node involvement E.THICKENED GASTRIC FOLDS

Notes:

Filling Defect Of Gastric Remnant A.IATROGENICsurgical deformity / plication defect, suture granuloma B.INFLAMMATORYbile reflux gastritis, hyperplastic polyps C.INTUSSUSCEPTION1.**Jejunogastric intussusception**
(efferent loop in 75%, afferent loop in 25%) (a)acute form: high intestinal obstruction, left hypochondriac mass, hematemesis(b)chronic / intermittent form: may be self-reducing"V" "coil spring" appearance of gastric filling defect2.Gastrojejunal / gastroduodenal mucosal prolapse ● often asymptomatic ● bleeding, partial obstructionD.NEOPLASTIC1.Gastric stump carcinoma: >5 years after resection for benign disease; 15% within 10 years; 20% after 20 years2.Recurrent carcinoma (10%) secondary to incomplete removal of gastric cancer 3.Malignancy at anastomosis (incomplete resection)E.INTRALUMINAL MATTER: bezoar
mnemonic:"PUBLICS"**P**olyp (hyperplastic polyp due to bile reflux) **U**lcer (anastomotic) **B**ezoar, **B**lind loop syndrome **L**oop (afferent loop syndrome) **I**ntussusception at gastrojejunostomy **C**ancer (recurrent, residual, de novo) **S**urgical deformity, **S**uture granuloma

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Notes:

Thickened Gastric Folds A.INFLAMMATION / INFECTION1.Inflammatory gastritis:alcoholic, hypertrophic, antral, corrosive, postirradiation, gastric cooling 2.Crohn disease3.Sarcoidosis4.Infectious gastritis:bacterial invasion, bacterial toxins from botulism, diphtheria, dysentery, typhoid fever, anisakiasis, TB, syphilis 5.PseudolymphomaB.MALIGNANCY1.Lymphoma2.Gastric carcinomaC.INFILTRATIVE PROCESS1.Eosinophilic gastritis2.AmyloidosisD.PANCREATIC DISEASE1.Pancreatitis2.Direct extension from pancreatic carcinomaE.OTHERS1.Zollinger-Ellison syndrome2.Ménétrièr disease3.Gastric varices *mnemonic:*"ZEAL VOLUMES C^3P^3"**Z**ollinger-**E**llison syndrome **A**myloidosis **L**ymphoid hyperplasia **V**arices **O**perative defect **L**ymphoma **U**lcer disease (peptic) **M**énétrièr disease **E**osinophilic gastroenteritis **S**yphilis **C**rohn disease, **C**arcinoma, **C**orrosive gastritis **P**ancreatitis, **P**ancreatic carcinoma, **P**ostradiation gastritis

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Notes:

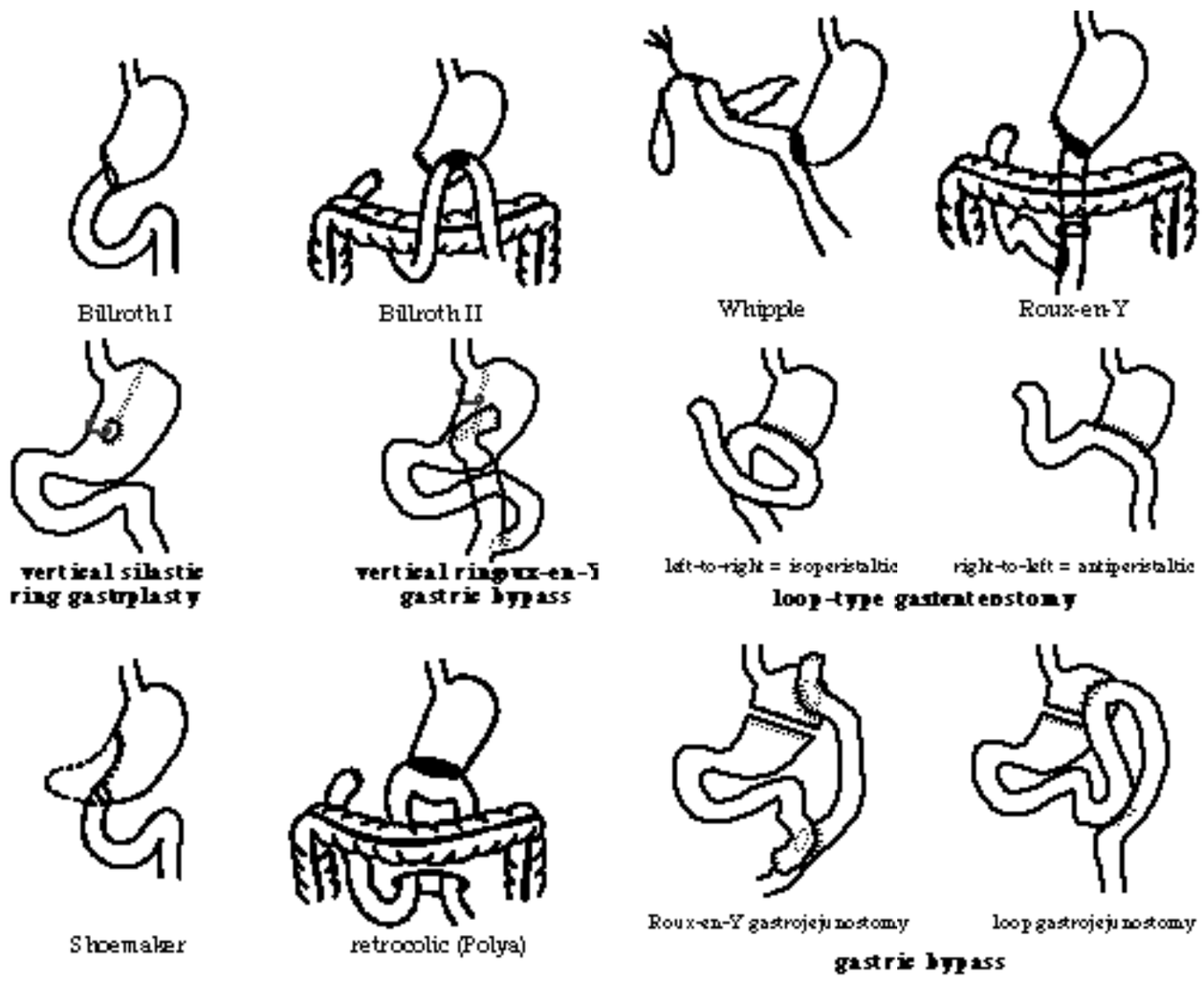
Gastric Ulcer A.HORMONAL1.Zollinger-Ellison syndrome2.Hyperparathyroidism (in 1.3-24%)duodenum:stomach = 4:1; M:F = 3:1 ◊Duodenal ulcers predominate in females!◊Gastric ulcers predominate in males! ● absence of gastric hypersecretion3.Steroid-induced ulcergastric > duodenal location; frequently multiple + deep ulcers; commonly associated with erosions ● bleeding (in 1/3)4.Curling ulcer (burn) (in 0.09-2.6%)5.Retained gastric antrumB.INFLAMMATION1.Peptic ulcer disease2.Gastritis3.Radiation-induced ulcerC.BENIGN MASS1.Leiomyoma2.Granulomatous disease3.Pseudolymphoma (lymphoid hyperplasia)D.MALIGNANT MASS1.Gastric carcinoma2.Lymphoma (2% of all gastric neoplasms)√ multiple ulcers with aneurysmal appearance3.Leiomyosarcoma, neurogenic sarcoma, fibrosarcoma, liposarcoma4.Metastases (a)hematogenic: malignant melanoma, breast cancer, lung cancer(b)per continuum: pancreas, colon, kidneyE.DRUGSASA: greater curvature

Notes:

Bulls-eye Lesions A.PRIMARY NEOPLASMS1.Leiomyoma, leiomyosarcoma2.Lymphoma3.Carcinoid4.Primary carcinomaB.HEMATOGENOUS METASTASES1.Malignant melanoma√ usually spares large bowel2.Breast cancer (15%)√ scirrhous appearance in stomach3.Cancer of lung4.Renal cell carcinoma5.Kaposi sarcoma6.Bladder carcinomaC.ECTOPIC PANCREASin duodenum / stomach D.EOSINOPHILIC GRANULOMAmost frequently in stomach

Notes:

Complications Of Postoperative Stomach 1.Filling defect of gastric remnant2.Retained gastric antrum3.Dumping syndrome4.Afferent loop syndrome5.Stomal obstruction(a)temporary reversible: edema of suture line, abscess / hematoma, potassium deficiency, inadequate electrolyte replacement, hypoproteinemia, hypoacidity(b)late mechanical: stomal ulcer (75%)*mnemonic:*"LOBULATING"**L**eaks (early) **O**bstruction (early) **B**ezoar **U**lcer (especially marginal) **L**oop (afferent loop syndrome) **A**nemia (macrocytic secondary to decreased intrinsic factor) **T**umor (? increased incidence) **I**ntussusception **N**ot feeling well after meals (dumping syndrome) **G**astritis (bile reflux)



Gastric Surgical Procedures

Notes:

Lesions Involving Stomach And Duodenum 1.Lymphoma: in <33% of patients with lymphoma2.Gastric carcinoma: in <5%, but 50 x more common than lymphoma 3.Peptic ulcer disease4.Tuberculosis: in 10% of gastric TB5.Crohn disease: pseudo-Billroth-I pattern6.Strongyloidiasis7.Eosinophilic gastroenteritis

Notes:

Extrinsic Pressure Effect On Duodenum A.BILE DUCTSnormal impression, dilated CBD, choledochal cyst B.GALLBLADDERnormal impression, gallbladder hydrops, Courvoisier phenomenon, gallbladder carcinoma, pericholecystic abscess C.LIVERhepatomegaly, hypertrophied caudate lobe, anomalous hepatic lobe, hepatic cyst, hepatic tumor D.RIGHT KIDNEYbifid collecting system, hydronephrosis, multiple renal cysts, polycystic kidney disease, hypernephroma E.RIGHT ADRENALadrenal carcinoma, enlargement in Addison disease F.COLONduodenocolic apposition due to anomalous peritoneal fixation, carcinoma of hepatic flexure G.VESSELSlymphadenopathy, duodenal varices, dilated arterial collaterals, aortic aneurysm, intramural / mesenteric hematoma
Widened duodenal sweep A.NORMAL VARIANTB.PANCREATIC LESION1.Acute pancreatitis2.Chronic pancreatitis3.Pancreatic pseudocyst4.Pancreatic carcinoma5.Metastasis to pancreas6.Pancreatic cystadenomaC.VASCULAR LESION1.Lymph node enlargement: lymphoma, metastasis, inflammation2.Cystic lymphangioma of the mesenteryD.RETROPERITONEAL MASS1.Aortic aneurysm2.Choledochal cyst

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Notes:

Thickened Duodenal Folds A.INFLAMMATION(a)within bowel wall:peptic ulcer disease, Zollinger-Ellison syndrome, regional enteritis, lymphoid hyperplasia, uremia (b)surrounding bowel wall:pancreatitis, cholecystitis B.INFECTIONgiardiasis, TB, strongyloidiasis, celiac disease C.NEOPLASIAlymphoma, metastases to peripancreatic nodes D.DIFFUSE INFILTRATIVE DISORDERWhipple disease, amyloidosis, mastocytosis, eosinophilic enteritis, intestinal lymphangiectasia E.VASCULAR DISORDERduodenal varices, mesenteric arterial collaterals, intramural hemorrhage (trauma, Schönlein-Henoch purpura), chronic duodenal congestion (congestive heart failure, portal venous hypertension); lymphangiectasia F.HYPOPROTEINEMIAnephrotic syndrome, Menetrier disease, protein-losing enteropathy G.GLANDULAR ENLARGEMENTBrunner gland hyperplasia, cystic fibrosis *mnemonic:*"BAD HELP"**B**runner gland hyperplasia **A**myloidosis **D**uodenitis (Z-E syndrome, peptic) **H**emorrhage **E**dema, **E**ctopic pancreas **L**ymphoma **P**ancreatitis, **P**arasites

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Notes:

Duodenal Filling Defect A.EXTRINSICgallbladder impression, CBD impression, gas-filled diverticulum B.INTRINSIC TO WALL(a)benign neoplastic massadenoma, leiomyoma, lipoma, hamartoma (Peutz-Jeghers syndrome), prolapsed antral polyp, Brunner gland adenoma, villous adenoma, islet cell tumor, gangliocytic paraganglioma (b)malignant neoplastic masscarcinoid tumor, adenocarcinoma, ampullary carcinoma, lymphoma, sarcoma, metastasis (stomach, pancreas, gallbladder, colon, kidney, melanoma), retroperitoneal lymph node involvement (c)nonneoplastic masspapilla of Vater, choledochocele, duplication cyst, pancreatic pseudocyst, duodenal varix, mesenteric artery collaterals, intramural hematoma, adjacent abscess, stitch abscess, ectopic pancreas, heterotopic gastric mucosa, prolapsed antral mucosa, Brunner gland hyperplasia, benign lymphoid hyperplasia C.INTRALUMINALblood clot, foreign body (fruit pit, gallstone, feeding tube)

Notes:

Duodenal Tumor ***Benign Duodenal Tumors*** 1.Leiomyoma(27%)2.Adenomatous polyp(21%)3.Lipoma(21%)4.Brunner gland adenoma(17%)5.Angiomatous tumor(6%)6.Ectopic pancreas(2%)7.Duodenal cyst(2%)8.Neurofibroma(2%)9.Hamartoma (2%) ***Malignant Duodenal Tumors*** 1.Adenocarcinoma (73%)Location:40% in duodenum, most often in 2nd + 3rd portion = periampullary neoplasm(a)suprapapillary: apt to cause obstruction + bleeding(b)peripapillary: extrahepatic jaundice(c)intrapapillary: GI bleeding*May be associated with:* Peutz-Jeghers syndrome √ annular / polypoid / ulcerativeMetastases:regional lymph nodes (2/3)*DDx:*(1) Primary bile duct carcinoma(2) Ampullary carcinoma2.Leiomyosarcoma (14%)most often beyond 1st portion of duodenum √ up to 20 cm in size√ frequently ulcerated exophytic mass3.Carcinoid (11%)4.Lymphoma (2%)√ marked wall thickening√ bulky periduodenal lymphadenopathy

Notes:

Enlargement Of Papilla Of Vater A.Normal variantidentified in 60% of UGI series; atypical location in 3rd portion of duodenum in 8%; 1.5 cm in diameter in 1% of normals B.Papillary edema1.Impacted stone2.Pancreatitis (Poppel sign)3.Acute duodenal ulcer disease4.PapillitisC.Perivaterian neoplasms=tumor mass + lymphatic obstruction1.Adenocarcinoma2.Adenomatous polyp (premalignant lesion)√ irregular surface + erosionsD.Lesions simulating enlarged papilla1.Benign spindle cell tumor2.Ectopic pancreatic tissue

Notes:

Duodenal Narrowing A.DEVELOPMENTAL ANOMALIES1.Duodenal atresia2.Congenital web / duodenal diaphragm3.Intraluminal diverticulum4.Duodenal duplication cyst5.Annular pancreas6.Midgut volvulus, peritoneal bands (Ladd bands)B.INTRINSIC DISORDERS(a)inflammation / infection1.Postbulbar ulcer2.Crohn disease3.Sprue4.Tuberculosis5.Strongyloidiasis(b)tumorduodenal / ampullary malignancy C.DISEASE IN ADJACENT STRUCTURES1.Pancreatitis, pseudocyst, pancreatic carcinoma2.Cholecystitis3.Contiguous abscess4.Metastases to pancreaticoduodenal nodes (lymphoma, lung cancer, breast cancer)D.TRAUMA1.Duodenal rupture2.Intramural hematomaE.VASCULAR1.Superior mesenteric artery syndrome2.Aorticoduodenal fistula3.Preduodenal portal vein (anterior to descending duodenum)

Notes:

Dilated Duodenum *Megaduodenum*= marked dilatation of entire C-loop*Megabulbus*= dilatation of duodenal bulb onlyA.VASCULAR COMPRESSIONsuperior mesenteric artery syndrome, abdominal aortic aneurysm, aorticoduodenal fistula B.PRIMARY DUODENAL ATONY(a)scleroderma, dermatomyositis, SLE(b)Chagas disease, aganglionosis, neuropathy, surgical / chemical vagotomy(c)focal ileus: pancreatitis, cholecystitis, peptic ulcer disease, trauma(d)altered emotional status, chronic idiopathic intestinal pseudoobstructionC.INFLAMMATORY / NEOPLASTIC INDURATION OF MESENTERIC ROOTCrohn disease, tuberculous enteritis, pancreatitis, peptic ulcer disease, strongyloidiasis, metastatic disease D.FLUID DISTENSIONceliac disease, Zollinger-Ellison syndrome

Notes:

Postbulbar Ulceration 1.Benign postbulbar peptic ulcer√ medial aspect of upper 2nd portion√ incisura pointing to ulcer√ occasionally barium reflux into common bile duct√ ring stricture√ stress- and drug-induced ulcers heal without deformity2.Zollinger-Ellison syndrome√ multiple ulcers distal to duodenal bulb√ thickening of folds + hypersecretion3.Leiomyoma4.Malignant tumors: (a)primariesadenocarcinoma, lymphoma, sarcoma (b)contiguous spreadpancreas, colon, kidney, gallbladder (c)hematogenous spreadmelanoma, Kaposi sarcoma (d)lymphogenic spreadmetastases to periduodenal lymph nodes 5.Granulomatous disease: Crohn disease, TB6.Aorticoduodenal fistula7.Mimickers: ectopic pancreas, diverticulum

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Notes:

Small Bowel Diverticula A.TRUE DIVERTICULA(a)Duodenal diverticula1.Racemose diverticula: bizarre, lobulated2.Giant diverticula3.Intraluminal diverticula: result of congenital web / diaphragm(b)Jejunal diverticulosis(c)Meckel diverticulumB.PSEUDODIVERTICULA1.Scleroderma2.Crohn disease3.Lymphoma4.Mesenteric ischemia5.Communicating ileal duplication6.Giant duodenal ulcer

Notes:

Small Bowel Ulcer ***Aphthous Ulcers Of Small Bowel*** A.INFECTION1.Yersinia enterocolitis (25%)2.Salmonellosis3.Tuberculosis4.RickettsiosisB.INFLAMMATION1.Crohn disease (22%)2.Behçet syndrome3.Reiter syndrome4.Ankylosing spondylitis ***Large Nonstenotic Ulcers Of Small Bowel*** 1.Primary nonspecific ulcer47% incidence2.Yersiniosis33%3.Crohn disease30%4.Tuberculosis18%5.Salmonellosis / shigellosis7%6.Meckel diverticulum5% ***Multiple Small Bowel Ulcers*** A.DRUGS1.Potassium tablets2.Steroids3.Nonsteroidal anti-inflammatory drugsB.INFECTION / INFLAMMATION1.Bacillary dysentery2.Ischemic enteritis3.Ulcerative jejunoileitis as complication of celiac diseaseC.TUMOR1.Neoplasms2.Intestinal lymphoma ***Cavitary Small Bowel Lesions*** 1.Lymphoma (exoenteric form)2.Leiomyosarcoma (exoenteric form)3.Primary adenocarcinoma4.Metastases (especially malignant melanoma)

Notes:

Separation Of Bowel Loops A.INFILTRATION OF BOWEL WALL / MESENTERY(a)inflammation / infection1.Crohn disease2.TB3.Radiation injury4.Retractile mesenteritis5.Intraperitoneal abscess(b)deposits1.Intestinal hemorrhage / mesenteric vascular occlusion2.Whipple disease3.Amyloidosis(c)tumor1.Carcinoid tumor: local release of serotonin responsible for muscular thickening + fibroplastic proliferation = desmoplastic reaction2.Primary carcinoma of small bowel (unusual presentation)3.Lymphoma4.NeurofibromatosisB.ASCITEShepatic cirrhosis (75%), peritonitis, peritoneal carcinomatosis, congestive heart failure, constrictive pericarditis, primary / metastatic lymphatic disease C.EXTRINSIC MASS1.Peritoneal mesothelioma, mesenteric tumors (fibroma, lipoma, fibrosarcoma, leiomyosarcoma, malignant mesenteric lymphoid tumor, metastases)2.Intraperitoneal abscess3.Retractile mesenteritis (fibrosis, fatty infiltration, panniculitis)

Notes:

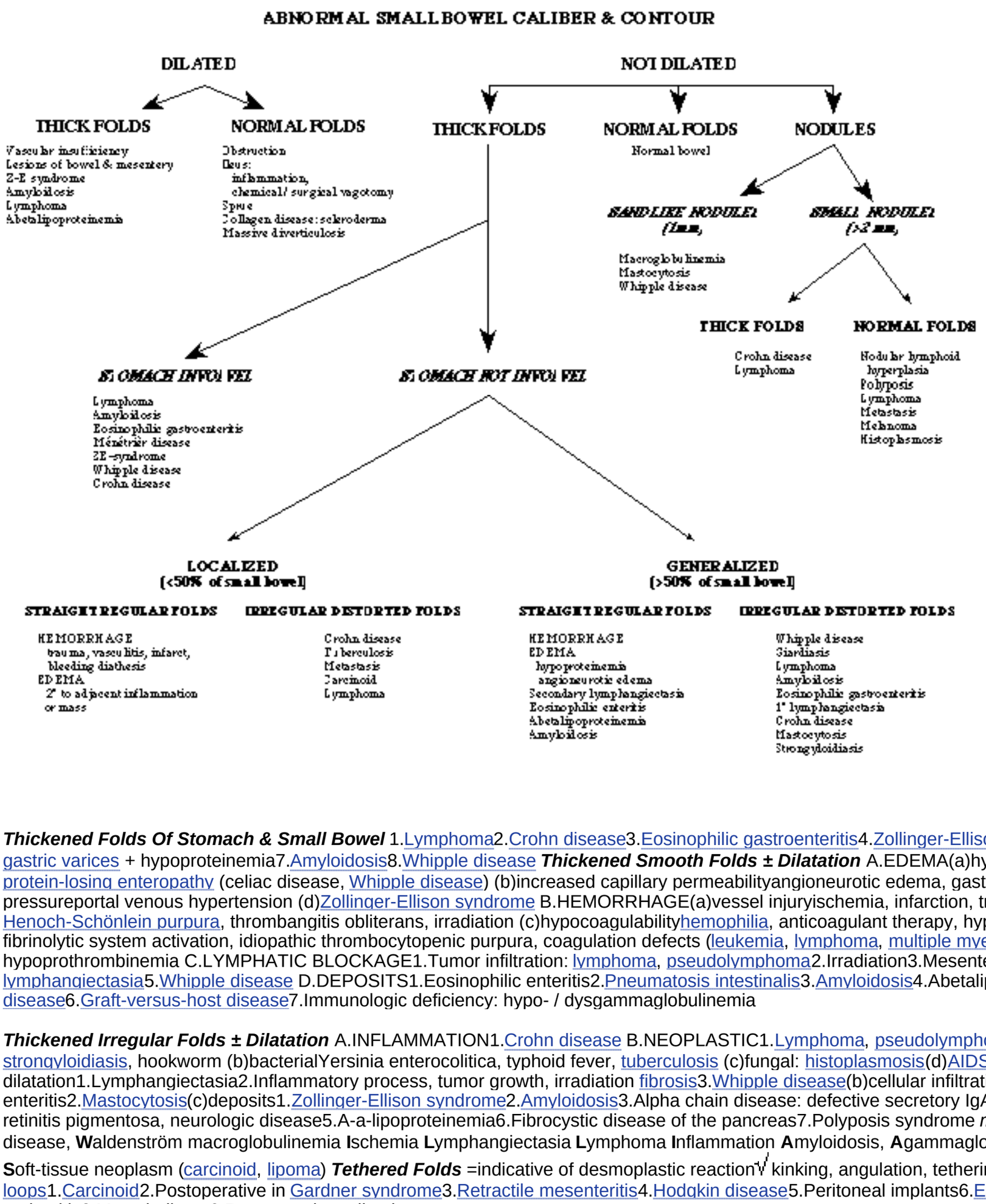
Normal Small Bowel Folds & Diarrhea 1.Pancreatic insufficiency2.Lactase deficiency3.Lymphoma / pseudolymphoma

Notes:

Dilated Small Bowel & Normal Folds *mnemonic:*"SOS"**S**prue **O**bstruction **S**cleroderma A.EXCESSIVE FLUID(a)mechanical obstructiondue to adhesion, hernia, neoplasm √ "string-of-beads sign" = air bubbles between mucosal folds in a fluid-filled small bowel√ "pseudotumor sign" = closed-loop obstruction(b)malabsorption syndromes1.Celiac disease, tropical + nontropical sprue2.Lactase deficiency B.BOWEL WALL PARALYSIS=functional ileus = adynamic ileus1.Surgical vagotomy2.Chemical vagotomy from drug effects:atropine-like substances, morphine, L-dopa, glucagon 3.Chagas disease4.Metabolic: hypokalemia, diabetes5.Intrinsic + extrinsic intra-abdominal inflammation 6.Chronic idiopathic pseudoobstruction C.VASCULAR COMPROMISE1.Mesenteric ischemia (atherosclerosis)2.Acute radiation enteritis3.Amyloidosis4.SLE D.BOWEL WALL DESTRUCTION1.Lymphoma2.Scleroderma (smooth muscle atrophy)3.Dermatomyositis

Notes:

Abnormal Small Bowel Folds



Thickened Folds Of Stomach & Small Bowel 1.Lymphoma2.Crohn disease3.Eosinophilic gastroenteritis4.Zollinger-Ellison syndrome5.Ménétrièr disease6.Cirrhosis = gastric varices + hypoproteinemia7.Amyloidosis8.Whipple disease ***Thickened Smooth Folds ± Dilatation*** A.EDEMA(a)hypoproteinemiacirrhosis, nephrotic syndrome, protein-losing enteropathy (celiac disease, Whipple disease) (b)increased capillary permeabilityangioneurotic edema, gastroenteritis (c)increased hydrostatic pressureportal venous hypertension (d)Zollinger-Ellison syndrome B.HEMORRHAGE(a)vessel injuryischemia, infarction, trauma (b)vasculitisconnective tissue disease, Henoch-Schönlein purpura, thrombangitis obliterans, irradiation (c)hypocoagulabilityhemophilia, anticoagulant therapy, hypofibrinogemia, circulating anticoagulants, fibrinolytic system activation, idiopathic thrombocytopenic purpura, coagulation defects (leukemia, lymphoma, multiple myeloma, metastatic carcinoma), hypoprothrombinemia C.LYMPHATIC BLOCKAGE1.Tumor infiltration: lymphoma, pseudolymphoma2.Irradiation3.Mesenteric fibrosis4.Intestinal lymphangiectasia5.Whipple disease D.DEPOSITS1.Eosinophilic enteritis2.Pneumatosis intestinalis3.Amyloidosis4.Abetalipoproteinemia5.Crohn disease6.Graft-versus-host disease7.Immunologic deficiency: hypo- / dysgammaglobulinemia

Thickened Irregular Folds ± Dilatation A.INFLAMMATION1.Crohn disease B.NEOPLASTIC1.Lymphoma, pseudolymphomaC.INFECTION(a)protozoangiardiasis, strongyloidiasis, hookworm (b)bacterialYersinia enterocolitica, typhoid fever, tuberculosis (c)fungal: histoplasmosis(d)AIDS-related infectionD.IDIOPATHIC(a)lymphatic dilatation1.Lymphangiectasia2.Inflammatory process, tumor growth, irradiation fibrosis3.Whipple disease(b)cellular infiltration1.Eosinophilic enteritis2.Mastocytosis(c)deposits1.Zollinger-Ellison syndrome2.Amyloidosis3.Alpha chain disease: defective secretory IgA system4.A-b-lipoproteinemia: recessive, retinitis pigmentosa, neurologic disease5.A-a-lipoproteinemia6.Fibrocystic disease of the pancreas7.Polyposis syndrome *mnemonic:*"G. WILLIAMS"**G**iardiasis **W**hipple disease, **W**aldenström macroglobulinemia **I**schemia **L**ymphangiectasia **L**ymphoma **I**nflammation **A**myloidosis, **A**gammaglobulinemia **M**astocytosis, **M**alabsorption **S**oft-tissue neoplasm (carcinoid, lipoma) ***Tethered Folds*** =indicative of desmoplastic reaction* kinking, angulation, tethering, separation of bowel loops1.Carcinoid2.Postoperative in Gardner syndrome3.Retractile mesenteritis4.Hodgkin disease5.Peritoneal implants6.Endometriosis7.Tuberculous peritonitis8.Mesothelioma9.Postoperative adhesions

Notes:

Atrophy Of Folds 1.Celiac disease2.Chronic radiation injury

Notes:

Ribbonlike Bowel =featureless / tubular nature of small bowel with effacement of folds1.Graft-versus-host disease2.Celiac disease3.Small bowel infection4.Injury from radiation / corrosive medication5.Allergy6.Ischemia7.Amyloid, mastocytosis8.Lymphoma, pseudolymphoma9.Crohn disease

Notes:

Delayed Small Bowel Transit =transit time >6 hours*mnemonic:*"SPATS DID"**S**cleroderma **P**otassium (hypokalemia) **A**nxiety **T**hyroid (hypothyroidism) **S**prue **D**iabetes (poorly controlled) **I**diopathic **D**rugs (opiates, atropine, phenothiazine)

Notes:

Multiple Stenotic Lesions Of Small Bowel 1.Crohn disease2.End-stage radiation enteritis3.Metastatic carcinoma4.Endometritis5.Eosinophilic gastroenteritis6.Tuberculosis7.Drug-induced (eg, potassium chloride tablets, NSAIDs)

Notes:

Small Bowel Filling Defects *Solitary Filling Defect* A.INTRINSIC TO BOWEL WALL(a)benign neoplasm: leiomyoma (97%), adenoma, lipoma, hemangioma, neurofibroma(b)malignant primary: adenocarcinoma, lymphoma (desmoplastic response), sarcoma, carcinoid(c)metastases: from melanoma, lung, kidney, breast(d)inflammation: inflammatory pseudotumor(e)infection: parasitesB.EXTRINSIC TO BOWEL WALL1.Duplication cyst2.EndometriomaC.INTRALUMINAL1.Gallstone ileus2.Parasites (ascariasis, strongyloidiasis)3.Inverted Meckel diverticulum4.Blood clot5.Foreign body, bezoar, pills, seeds ***Multiple Filling Defects Of Small Bowel*** A.POLYPOSIS SYNDROMES1.Peutz-Jeghers syndrome2.Gardner syndrome3.Disseminated gastrointestinal polyposis4.Generalized gastrointestinal juvenile polyposis5.Cronkhite-Canada syndromeB.BENIGN TUMORS1.Multiple simple adenomatous polyps2.Hemangioma3.Leiomyoma, neurofibroma4.Nodular lymphoid hyperplasia=normal terminal ileum in children + adolescents; may be associated with dysgammaglobulinemia√ symmetric fairly sharply demarcated filling defects5.Varices (= multiple phlebectasia in jejunum, oral mucosa, tongue, scrotum)C.MALIGNANT TUMORS1.Carcinoid tumor2.Lymphoma(a)primary lymphoma (rarely multiple)(b)secondary lymphoma: gastrointestinal involvement in 63% of disseminated disease; 19% in small intestine3.Metastases: melanoma > lung > breast > choriocarcinoma > kidney > stomach, uterus, ovary, pancreasD.INTRALUMINAL1.Gallstones2.Foreign bodies, food particles, seeds, pills3.Parasites: ascariasis, strongyloidiasis, hookworm, tapeworm ***Sandlike Lucencies Of Small Bowel*** 1.Waldenström macroglobulinemia2.Mastocytosis3.Histoplasmosis4.Nodular lymphoid hyperplasia5.Intestinal lymphangiectasia6.Eosinophilic gastroenteritis7.Lymphoma8.Crohn disease9.Whipple disease10.Yersinia enterocolitis11.Cronkhite-Canada syndrome12.Cystic fibrosis13.Food particles / gas bubbles14.Strongyloides stercoralis

Notes:

Small Bowel Tumors *Incidence:* 1:100,000; 1.5-6% of all GI neoplasmsMalignant:benign = 1:1 Symptomatic malignant:symptomatic benign = 3:1 Location of small bowel primaries: ileum (41%), jejunum (36%), duodenum (18%) ROENTGENOGRAPHIC APPEARANCE: (1)pedunculated intraluminal tumor, usually originating from mucosa√ smooth / irregular surface without visible mucosal pattern√ moves within intestinal lumen twice the length of the stalk(2)sessile intraluminal tumor without stalk, usually from tissues outside mucosa√ smooth / irregular surface without visible mucosal pattern(3)intra- / extramural tumor√ base of tumor greater than any part projecting into the lumen√ mucosal pattern visible, may be stretched(4)serosal tumor√ displacement of adjacent loops√ small bowel obstruction (rare)√ coil-spring pattern of intussusceptumCT:small bowel wall >1.5 cm thick*Cx:*small-bowel obstruction (in up to 10%) ***Benign Small Bowel Tumors*** ● asymptomatic (80%) ● melena, pain, weakness ● palpable abdominal mass (20%)*Types:* 1.Leiomyoma (36-49%)Location:any segment2.Lipoma (14-16%)Location:duodenum (32%), jejunum (17%), ileum (51%)√ fat-density on CT3.Adenoma (15-20%)4.Hemangioma (13-16%)5.Lymphangioma (5%)Location: duodenum > jejunum > ileum6.Neurogenic tumor (1%) ***Malignant Small Bowel Tumors*** At risk:Crohn disease, celiac disease, polyposis syndromes, history of small-bowel diverting surgery ● asymptomatic (10-30%) ● pain due to intermittent obstruction (80%) ● weight loss (66%) ● gastrointestinal blood loss (50%) ● palpable abdominal mass (50%)1.Carcinoid (25-41%)Location:predominantly distal ileum√ calcified mesenteric mass on CT2.Adenocarcinoma (25-26%)Location:duodenum (48%), jejunum (44%), ileum (8%)3.Lymphoma (16-17%)√ aneurysmal dilatation4.Gastrointestinal stromal tumor (GIST)= leiomyosarcoma (9-10%) Location:ileum (50%)5.Vascular malignancy (1%)6.Fibrosarcoma (0.3%)7.Metastatic tumor

Notes:

Ileocecal Valve Abnormalities A.Lipomatosis: >40 years of age, female√ stellate / rosette patternB.NEOPLASM1.Lipoma, adenomatous polyp, villous adenoma2.Carcinoid tumor3.Adenocarcinoma: 2% of all colonic cancers4.Lymphoma: often involving terminal ileumC.INFLAMMATION1.Crohn disease2.Ulcerative colitis√ patulous valve, fixed in open position3.Tuberculosis4.Amebiasis√ terminal ileum not involved (in United States)5.Typhoid fever, anisakiasis, schistosomiasis, actinomycosis6.Cathartic abuseD.PROLAPSE(a)antegrade:indistinguishable from lipomatosis / prolapsing mucosa / neoplasm(b)retrogradeE.INTUSSUSCEPTIONF.LYMPHOID HYPERPLASIA

Notes:

Coned Cecum A.INFLAMMATION1.Crohn disease√ involvement of ascending colon + terminal ileum2.Ulcerative colitis√ backwash ileitis (in 10%)√ gaping ileocecal valve3.Appendicitis4.Typhlitis5.Perforated cecal diverticulumB.INFECTION1.Tuberculosis√ colonic involvement more prominent than that of terminal ileum2.Amebiasis √ involvement of cecum in 90% of amebiasis√ thickened ileocecal valve fixed in open position√ reflux into normal terminal ileum√ skip lesions in colon3.Actinomycosis ● palpable abdominal mass ● indolent sinus tracts in abdominal wall4.Blastomycosis5.**Anisakiasis**
from ingestion of raw fish with ascaris-like nematode 6.Typhoid, YersiniaC.TUMOR1.Carcinoma of the cecum2.Metastasis to cecum

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Notes:

Radiology Review Manual
fourth edition

Cecal Filling Defect A.ABNORMALITIES OF THE APPENDIX1.Acute appendicitis / appendiceal abscess2.Crohn disease3.Inverted appendiceal stump / appendiceal intussusception4.Mucocele5.Myxoglobulosis6.Appendiceal neoplasm: carcinoid tumor (90%), leiomyoma, neuroma, lipoma, adenocarcinoma, metastasisB.COLONIC LESION1.Ameboma2.Primary cecal neoplasm3.Ileocolic intussusception4.Lipomatosis of ileocecal valveC.UNUSUAL ABNORMALITIES1.Ileocecal diverticulitis (in 50% < age 30 years)2.Solitary benign ulcer of the cecum3.Adherent fecolith (eg, in cystic fibrosis)4.Endometriosis5.Burkitt lymphoma *mnemonic:*"CECUM TIPSALE"**C**arcinoma **E**nteritis **C**arcinoid **U**lcerative colitis **M**ucocele of appendix **T**uberculosis **I**ntussusception **P**eriappendiceal abscess **S**tump of the appendix **A**meboma **L**ymphoma **E**ndometriosis

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Notes:

Colon Cutoff Sign =abrupt cutoff of gas column at splenic flexure1.Acute pancreatitis (inflammatory exudate along transverse mesocolon)2.Colonic obstruction3.Mesenteric thrombosis4.Ischemic colitis

Notes:

Colonic Thumbprinting =sharply defined fingerlike marginal indentations at contours of wall1.ISCHEMIA = Ischemic colitisocclusive vascular disease, hypercoagulability state, hemorrhage into bowel wall (bleeding diathesis, anticoagulants), traumatic intramural hematoma 2.INFLAMMATIONulcerative colitis, Crohn colitis 3.INFECTIONacute amebiasis, schistosomiasis, strongyloidiasis, cytomegalovirus (in renal transplant recipients), pseudomembranous colitis 4.MALIGNANT LESIONSlocalized primary lymphoma, hematogenous metastases 5.MISCELLANEOUSendometriosis, amyloidosis, pneumatosis intestinalis, diverticulosis, diverticulitis, hereditary angioneurotic edema *mnemonic:*"PSALM II"**P**seudomembranous colitis **S**chistosomiasis **A**mebic colitis **L**ymphoma **M**etastases (to colon) **I**schemic colitis **I**nflammatory bowel disease

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Notes:

Colonic Urticaria Pattern A.OBSTRUCTION1.Obstructing carcinoma2.Cecal volvulus3.Colonic ileusB.ISCHEMIAC.INFECTION / INFLAMMATION1.Yersinia enterocolitis2.Herpes3.Crohn diseaseD.URTICARIA

Notes:

Colonic Ulcers A.IDIOPATHIC1.Ulcerative colitis2.Crohn colitisB.ISCHEMIC1.Ischemic colitisC.TRAUMATIC1.Radiation injury2.Caustic colitisD.NEOPLASTIC1.Primary colonic carcinoma2.Metastases (prostate, stomach, lymphoma, leukemia)E.INFLAMMATORY1.Pseudomembranous colitis2.Pancreatitis3.Diverticulitis4.Behçet syndrome5.Solitary rectal ulcer syndrome6.Nonspecific benign ulcerationF.INFECTION(a)protozoan1.Amebiasis2.Schistosomiasis3.Strongyloidiasis(b)bacterial1.Shigellosis, salmonellosis2.Staphylococcal colitis3.Tuberculosis4.Gonorrheal proctitis5.Yersinia colitis6.Campylobacter fetus colitis(c)fungalhistoplasmosis, mucormycosis, actinomycosis, candidiasis (d)viral1.Lymphogranuloma venereum2.Herpes proctocolitis3.Cytomegalovirus (transplants) ***Aphthous Ulcers*** 1.Crohn disease2.Amebic colitis3.**Yersinia enterocolitis**

*Organism:*Gram-negative ● fever, diarrhea, RLQ painLocation:terminal ileum√ thickened folds + ulceration√ lymphoid nodular hyperplasia4.Salmonella, shigella infection5.Herpes virus infection6.Behçet syndrome7.Lymphoma8.Ischemia

Notes:

Multiple Bulls-eye Lesions Of Bowel Wall *mnemonic:*"MaCK CLaN"**M**elanoma and **C**arcinoma **K**aposi sarcoma **C**arcinoid **L**ymphoma and **N**eurofibromatosis

Notes:

Double-tracking Of Colon =longitudinal extraluminal tracks paralleling the colon1.Diverticulitis: generally 3-6 cm in length2.Crohn disease: generally >10 cm3.Primary carcinoma: wider + more irregular

Notes:

Colonic Narrowing A.CHRONIC STAGE OF ANY ULCERATING COLITIS(a)inflammatory:ulcerative colitis, Crohn colitis, solitary rectal ulcer syndrome, nonspecific benign ulcer (b)infectious:amebiasis, schistosomiasis, bacillary dysentery, TB, fungal disease, lymphogranuloma venereum, herpes zoster, cytomegalovirus, strongyloides (c)ischemic: ischemic colitis(d)traumatic:radiation injury, cathartic colon, caustic colitis B.MALIGNANT LESION(a)primary: colonic carcinoma (annular / scirrhous); complication of ulcerative colitis + Crohn colitis(b)metastatic:from prostate, cervix, uterus, kidney, stomach, pancreas, primary intraperitoneal sarcoma -hematogenous (eg, breast)-lymphangitic spread-peritoneal seedingC.EXTRINSIC PROCESS(a)inflammation:retractile mesenteritis, diverticulitis, pancreatitis (b)deposits:amyloidosis, endometriosis, pelvic lipomatosis D.POSTSURGICALadhesive bands, surgical anastomosis E.NORMALCannon point ***Localized Colonic Narrowing*** *mnemonic:*"SCARED CELL-MATE"**S**chistosomiasis **C**arcinoid **A**ctinomycosis **R**adiation **E**ndometriosis **D**iverticulitis **C**olitis **E**xtrinsic lesion **L**ymphoma **L**ymphogranuloma venereum **M**etastasis **A**denocarcinoma **T**uberculosis **E**ntamoeba histolytica ***Microcolon*** *mnemonic:*"MI MCA"**M**econium ileus **I**leal atresia **M**egacystis-microcolon-hypoperistalsis syndrome **C**olonic atresia (distal to atretic segment) **A**ganglionosis (Hirschsprung disease)

Notes:

Colonic Filling Defects *Submucosal Tumor* 1.Lipoma2.Carcinoid3.Leiomyoma4.Lymphangioma, hemangioma ***Single Colonic Filling Defect*** A.BENIGN TUMOR1.Polyp(hyperplastic, adenomatous, villous adenoma, villoglandular); most common benign tumor 2.LipomaMost common intramural tumor; 2nd most common benign tumor; M < F Location:ascending colon + cecum > left side of colon3.Carcinoid: 10% metastasize4.Spindle cell tumor(leiomyoma, fibroma, neurofibroma); 4th most common benign tumor; rectum > cecum 5.Lymphangioma, hemangiomaB.MALIGNANT TUMOR(a)primary tumor:carcinoma, sarcoma (b)secondary tumor:metastases (breast, stomach, lung, pancreas, kidney, female genital tract), lymphoma, invasion by adjacent tumors C.INFECTION1.Ameboma2.Polypoid granuloma: schistosomiasis, TBD.INFLAMMATION1.Inflammatory pseudopolyp: ulcerative colitis, Crohn disease2.Periappendiceal abscess3.Diverticulitis4.Foreign-body perforationE.NONSESSILE INTRALUMINAL BODY1.Fecal impaction2.Foreign body3.Gallstone4.Bolus of Ascaris wormsF.MISCELLANEOUS1.Endometriosis3rd most common benign tumor Location:sigmoid colon, rectosigmoid junction (at level of cul-de-sac) ● may cause bleeding (after invasion of mucosa)2.Localized amyloid deposition3.Suture granuloma4.Intussusception5.Pseudotumor (adhesions, fibrous bands)6.Colitis cystica profunda ***Multiple Colonic Filling Defects*** A.NEOPLASMS(a)polyposis syndrome:familial polyposis, Gardner syndrome, Peutz-Jeghers syndrome, Turcot syndrome, juvenile polyposis syndrome, disseminated gastrointestinal polyps, multiple adenomatous polyps (b)hematogenous metastases:from breast, lung, stomach, ovary, pancreas, uterus (c)multiple tumors-benign:neurofibromatosis, colonic lipomatosis, multiple hamartoma syndrome (Cowden disease) -malignant:lymphoma, leukemia, adenocarcinoma B.INFLAMMATORY PSEUDOPOLYPSulcerative colitis, Crohn colitis, ischemic colitis, amebiasis, schistosomiasis, strongyloidiasis, trichuriasis C.ARTIFACTSfeces, air bubbles, oil bubbles, mucous strands, ingested foreign body (eg, corn kernels) D.MISCELLANEOUSnodular lymphoid hyperplasia, lymphoid follicular pattern, hemorrhoids, diverticula, pneumatosis intestinalis, colitis cystica profunda, colonic urticaria, submucosal colonic edema secondary to obstruction, cystic fibrosis, amyloidosis, ulcerative pseudopolyps, proximal to obstruction *mnemonic:*"MILL P^3"**M**etastases (to colon) **I**schemia (thumbprinting) **L**ymphoma **L**ymphoid hyperplasia **P**olyposis **P**seudopolyposis (with inflammatory bowel disease); **P**neumatosis cystoides ***Carpet Lesions Of Colon*** =flat lobulated lesions with alteration of surface texture + little / no protrusion into lumenLocation:rectum > cecum > ascending colon*Cause:* A.NEOPLASMS1.Tubular / tubulovillous / villous adenoma2.Familial polyposis3.Adenocarcinoma4.Submucosal tumor spread (from adjacent carcinoma)B.MISCELLANEOUS1.Nonspecific follicular proctitis2.Biopsy site3.Endometriosis4.Rectal varices5.Colonic urticaria

Notes:

Colonic Polyp *Terminology:* 1.**Polyp** =mass projecting into the lumen of a hollow viscus above the level of the mucosa; usually arises from mucosa, may derive from submucosa / muscularis propria(a)neoplastic: adenoma / carcinoma(b)nonneoplastic: hamartoma / inflammatory polyp2.**Pseudopolyp** =scattered island of inflamed edematous mucosa on a background of denuded mucosa(a)pseudopolyposis of ulcerative colitis(b)"cobblestoning" of Crohn disease3.**Postinflammatory (filiform) polyp** =fingerlike projection of submucosa covered by mucosa on all sides following healing + regeneration of inflammatory (most common in ulcerative colitis) / ischemic / infectious bowel disease *Histologic classification:* A.ADENOMATOUS POLYPS=**Familial adenomatous polyposis syndrome** 1.Familial polyposis2.Gardner syndrome3.Turcot syndromeB.HAMARTOMATOUS POLYPS=HAMARTOMATOUS POLYPOSIS SYNDROMES1.Peutz-Jeghers syndrome (most in small bowel)2.Cowden disease3.Juvenile polyposis4.Cronkhite-Canada syndrome5.Bannayan-Riley-Ruvalcaba syndromeC.POLYPOSIS LOOK-ALIKES1.Inflammatory polyposis2.Lymphoid hyperplasia3.Lymphoma4.Metastases5.Pneumatosis coli ***Polyposis Syndromes*** =more than 100 polyps in number*Mode of transmission:* A.HEREDITARY(a)autosomal dominant1.Familial (multiple) polyposis2.Gardner syndrome3.Peutz-Jeghers syndrome(b)autosomal recessive1.Turcot syndromeB.NONHEREDITARY1.Cronkhite-Canada syndrome2.Juvenile polyposis

	Single Polyp	Multiple Polyps
Neoplastic (10%)		
— epithelial	1. Tubular adenoma 2. Tubulovillous adenoma 3. Villous adenoma 4. Turcot syndrome	1. Familial adenomatosis coli 2. Adenomatosis of GI tract 3. Gardner syndrome
— nonepithelial	1. Carcinoid 2. Leiomyoma 3. Lipoma 4. Hem-, lymphangioma 5. Fibroma, neurofibroma	
Nonneoplastic (90%)		
— unclassified	1. Hyperplastic polyp	1. Hyperplastic polyposis
— hamartomatous	1. Juvenile polyp 2. Peutz-Jeghers syndrome	1. Juvenile polyposis
— inflammatory	1. Ulcerative colitis 2. Benign lymphoid polyp 3. Fibroid granulation polyp	1. Cronkhite-Canada syndrome 2. Ulcerative colitis

Notes:

Rectal Narrowing 1.Pelvic lipomatosis + fibrolipomatosis2.Lymphogranuloma venereum3.Radiation injury of rectum4.Chronic ulcerative colitis

Notes:

Enlarged Presacral Space Normal width <5 mm in 95%; abnormal width >10 mm A.RECTAL INFLAMMATION / INFECTIONulcerative colitis, Crohn colitis, idiopathic proctosigmoiditis, radiation therapy B.RECTAL INFECTION1.Proctitis (TB, amebiasis, lymphogranuloma venereum, radiation, ischemia)2.DiverticulitisC.BENIGN RECTAL TUMOR1.Developmental cyst (dermoid, enteric cyst, tailgut cyst)2.Lipoma, neurofibroma, hemangioendothelioma3.Epidermal cyst4.Rectal duplicationD.MALIGNANT RECTAL TUMOR1.Adenocarcinoma, cloacogenic carcinoma2.Lymphoma, sarcoma, lymph node metastases3.Prostatic carcinoma, bladder tumors, cervical cancer, ovarian cancerE.BODY FLUIDS / DEPOSITS1.Hematoma: surgery, sacral fracture2.Pus: perforated appendix, presacral abscess3.Serum: edema, venous thrombosis4.Deposit of fat: pelvic lipomatosis, Cushing disease5.Deposit of amyloid: amyloidosisF.SACRAL TUMOR1.Sacrococcygeal teratoma, anterior sacral meningocele2.Chordoma, metastasis to sacrumG.MISCELLANEOUS1.Inguinal hernia containing segment of colon2.Colitis cystica profunda3.Pelvic lipomatosis

Notes:

Lesions Of Ischiorectal Fossa A.Congenital and developmental anomalies1.Gartner duct cyst2.Klippel-Trénaunay syndrome3.Tailgut cystB.Inflammatory and hemorrhagic lesions1.Fistula in ano2.Ischiorectal / perirectal abscess3.Extraperitoneal pelvic hematoma4.Rectal perforationC.Secondary neoplasmper direct extension / hematogenous spread: anorectal / prostatic / pelvic / sacral tumor; lung cancer; melanoma; lymphoma D.Primary neoplasm1.Aggressive angiomyxoma2.Lipoma3.Plexiform neurofibroma4.Anal adenocarcinoma5.Squamous cell carcinoma

Notes:

Peritoneal Mass A.SOLID MASS1.Peritoneal mesothelioma2.Peritoneal carcinomatosisB.INFILTRATIVE PATTERN1.Peritoneal mesotheliomaC.CYSTIC MASS1.Cystic mesothelioma2.Pseudomyxoma peritonei3.Bacterial / mycobacterial infection

Notes:

Omental Mass◊33% of primary omental tumors are malignant!◊Secondary neoplasms are more frequent than primary!A.SOLID MASS(a)benign: leiomyoma, lipoma, neurofibroma(b)malignant: leiomyosarcoma, liposarcoma, fibrosarcoma, lymphoma, peritoneal mesothelioma, hemangiopericytoma, metastases(c)Infection: tuberculosisB.CYSTIC MASShematoma

Notes:

Mesenteric Mass A.ROUND SOLID MASSES◊Benign primary tumors are more common than malignant primary tumors!◊Secondary neoplasms are more frequent than primary!◊Cystic are more common than solid tumors!◊Malignant solid tumors have a tendency to be located near root of mesentery, benign solid tumors in periphery near bowel!1.Metastases especially from colon, ovary (most frequent neoplasm of mesentery)2.Lymphoma3.Leiomyosarcoma (more frequent than leiomyoma)4.Neural tumor (neurofibroma, ganglioneuroma)5.Lipoma (uncommon), lipomatosis, liposarcoma6.Fibrous histiocytoma7.Hemangioma8.Desmoid tumor (most common primary)B.ILL-DEFINED MASSESmetastases (ovary), lymphoma, fibromatosis, fibrosing mesenteritis (associated with Gardner syndrome), lipodystrophy, mesenteric panniculitis C.STELLATE MASSESperitoneal mesothelioma, retractile mesenteritis, fibrotic reaction of carcinoid, radiation therapy, desmoid tumor, Hodgkin disease, tuberculous peritonitis, ovarian metastases, diverticulitis, pancreatitis◊A mesenteric mass with calcifications suggests carcinoid tumor !D.LOCULATED CYSTIC MASSES (2/3)cystic lymphangioma (most common), pseudomyxoma peritonei, cystic mesothelioma, mesenteric cyst, mesenteric hematoma, benign cystic teratoma, cystic spindle cell tumor (= centrally necrotic leiomyoma / leiomyosarcoma)

Notes:

Mesenteric / Omental Cysts ="BUBBLES OF THE BELLY"The first step is to determine the organ of origin!1.Lymphangioma2.Nonpancreatic pseudocyst=sequelae of mesenteric / omental hematoma / abscess*Path:*thick-walled, usually septated cystic mass with hemorrhagic / purulent contents3.Duplication cyst4.Mesothelial cyst5.Enteric cyst

Notes:

Umbilical Tumor A.PRIMARY (38%)benign / malignant neoplasm, skin tumor B.METASTASES (30%)="Sister Joseph nodule" ● firm painful nodule ● ± ulceration with serosanguinous / purulent discharge*Cause:*gastrointestinal cancer (50%), undetermined (25%), ovarian cancer, pancreatic cancer, small cell carcinoma of lung (very rare)Spread: (a)direct extension from anterior peritoneal surface(b)extension along embryonic remnants: falciform, median umbilical, omphalomesenteric ligaments(c)hematogenous(d)retrograde lymphatic flow from inguinal, axillary, paraaortic nodes(e)iatrogenic: laparoscopic tract, tract of percutaneous needle biopsyC.NONNEOPLASTIC1.Endometriosis (32%)2.Granuloma3.Incarcerated hernia

Notes:

Regional Patterns Of Lymphadenopathy @Retrocrural nodesAbnormal size:>6 mm*Common cause:*lung carcinoma, mesothelioma, lymphoma@Gastrohepatic ligament nodes=superior portion of lesser omentum suspending stomach from liverAbnormal size:>8 mm*Common cause:*carcinoma of lesser curvature of stomach, distal esophagus, lymphoma, pancreatic cancer, melanoma, colon + breast cancer*DDx:*coronary varices@Porta hepatis nodes=in porta hepatis extending down hepatoduodenal ligament, anterior + posterior to portal veinAbnormal size:>6 mm*Common cause:*carcinoma of gallbladder + biliary tree, liver, stomach, pancreas, colon, lung, breast*Cx:*high extrahepatic biliary obstruction@Pancreaticoduodenal nodes=between duodenal sweep + pancreatic head anterior to IVCAbnormal size:>10 mm*Common cause:*lymphoma, pancreatic head, colon, stomach, lung, breast cancer@Perisplenic nodes=in splenic hilumAbnormal size:>10 mm*Common cause:*NHL, leukemia, small bowel neoplasm, ovarian cancer, carcinoma of right / transverse colon@Retroperitoneal nodes=periaortic, pericaval, interaortocavalAbnormal size:>10 mm*Common cause:*lymphoma, renal cell, testicular, cervical, prostatic carcinomas@Celiac and superior mesenteric artery nodes=preaortic nodesAbnormal size:>10 mm*Common cause:*any intra-abdominal neoplasm@Pelvic nodes=along common, external + internal iliac vesselsAbnormal size:>15 mm*Common cause:*carcinoma of bladder, prostate, cervix, uterus, rectum

Notes:

Enlarged Lymph Node With Low-density Center 1.Tuberculosis, Mycobacterium avium-intracellulare2.Pyogenic infection3.Whipple disease4.Lymphoma5.Metastatic disease after radiation + chemotherapy

Notes:

GASTROINTESTINAL HEMORRHAGE

*Mortality:*approx. 10%Barium examination should be avoided in acute bleeders! *Source:* A.UPPER GASTROINTESTINAL HEMORRHAGE= bleeding site proximal to ligament of Treitz @ Esophagogastric junction 1.Esophageal varices (17%): 50% mortality2.Mallory-Weiss syndrome (7-14%): very low mortality@Stomach1.Acute hemorrhagic gastritis (17-27%)2.Gastric ulcer (10%)3.Pyloroduodenal ulcer (17-25%)*Mortality:* <10% if under age 60; >35% if over age 60@Other causes (14%): visceral artery aneurysm, vascular malformation, neoplasm, vascular-enteric fistula*Average mortality:* 8-10% B.LOWER GASTROINTESTINAL HEMORRHAGE@Small intestinetumor (eg, leiomyoma, metastases), ulcers, diverticula (eg, Meckel diverticulum), inflammatory bowel disease (eg, Crohn disease), vascular malformation, visceral artery aneurysm, aortoenteric fistula @Colorectal (70%)1.Diverticula (most common): hemorrhage in 25% of patients with diverticulosis; spontaneous cessation of bleeding in 80%; recurrent bleeding in 25%2.Colonic angiodysplasia = dilated submucosal arteries + veins overlying mucosal thinning (? secondary to mucosal ischemia)3.Colitis, tumors, mesenteric varices INFANTILE GASTROINTESTINAL BLEEDING (1)Peptic ulcer(2)Varices(3)Ulcerated Meckel diverticulum

Intramural Hemorrhage

Notes:

Intramural Hemorrhage A.VASCULITIS1.Henoch-Schönlein purpuraB.TRAUMAC.COAGULATION DEFECT1.Anticoagulant therapy2.Thrombocytopenia3.Disseminated intravascular coagulationD.DISEASES WITH COAGULATION DEFECT1.Hemophilia2.Leukemia, lymphoma3.Multiple myeloma4.Metastatic carcinoma5.Idiopathic thrombocytopenic purpuraE.ISCHEMIA (often fatal) ● abdominal pain ● melenaSite:submucosal / intramural / mesenteric √ "stacked coin" / "picket fence" appearance of mucosal folds (due to symmetric infiltration of submucosal blood)√ "thumbprinting" = rounded polypoid filling defect (due to focal accumulation of hematoma in bowel wall)√ separation + uncoiling of bowel loops√ narrowing of lumen + localized filling defects (asymmetric hematoma)√ no spasm / irritability√ mechanical obstruction + proximal distension of loops*Prognosis:*resolution within 2-6 weeks

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Notes:

GI ABNORMALITIES IN CHRONIC RENAL FAILURE AND RENAL TRANSPLANTATION

@Esophagus1.Esophagitis: candida, CMV, herpes@Stomach & duodenum1.Gastritis↓ thickened gastric folds (38%)↓ edema + erosions*Cause:* (a)imbalance of gastrin levels + gastric acid secretion due to (1) reduced removal of gastrin from kidney with loss of cortical mass (2) impaired acid feedback mechanism (3) hypochlorhydria(b)opportunistic infection (eg, CMV)2.Gastric ulcer (3.5%)3.Duodenal ulcer (2.4%)4.Duodenitis (47%)@ColonMore severely + frequently affected after renal transplantation 1.Progressive distention + pseudoobstruction*Contributing factors:* dehydration, alteration of diet, inactivity, nonabsorbable antacids, high-dose steroids 2.Ischemic colitis(a)primary disease responsible for end-stage renal disease (eg, diabetes, vasculitis)(b)trauma of renal transplantation3.Diverticulitis*Contributing factors:* chronic obstipation, steroids, autonomic nervous dysfunction 4.Pseudomembranous colitis5.Uremic colitis = nonspecific colitis6.Spontaneous colonic perforation*Cause:*nonocclusive ischemia, diverticula, duodenal + gastric ulcers@Pancreas1.Pancreatitis*Cause:*hypercalcemia, steroids, infection, immunosuppressive agents, trauma@General1.GI hemorrhage*Cause:*gastritis, ulcers, colonic diverticula, ischemic bowel, infectious colitis, pseudomembranous colitis, nonspecific cecal ulceration2.Bowel perforation (in 1-4% of transplant recipients)3.Opportunistic infection*Organism:* Candida, herpes, CMV, strongyloides 4.Malignancy(a) skin tumors (b) lymphoma

Notes:

Protein-losing Enteropathy A.DISEASE WITH MUCOSAL ULCERATION1.Carcinoma2.Lymphoma3.Inflammatory bowel disease4.Peptic ulcer diseaseB.HYPERTROPHIED GASTRIC RUGAE1.Ménétrièr diseaseC.NONULCERATIVE MUCOSAL DISEASE1.Celiac disease2.Tropical sprue3.Whipple disease4.Allergic gastroenteropathy5.Gastrocolic fistula6.Villous adenoma of colonD.LYMPHATIC OBSTRUCTION1.Intestinal lymphangiectasiaE.HEART DISEASE1.Constrictive pericarditis2.Tricuspid insufficiency

Notes:

Malabsorption =deficient absorption of any essential food materials within small bowel(1)PRIMARY MALABSORPTION=the digestive abnormality is the only abnormality present1.Celiac disease = nontropical sprue2.Tropical sprue3.Disaccharidase deficiencies(2)SECONDARY MALABSORPTION=occurring during course of gastrointestinal disease(a)enteric1.Whipple disease2.Parasites: hookworm, Giardia, fish tapeworm3.Mechanical defects: fistulas, blind loops, adhesions, volvulus, short circuits4.Neurologic: diabetes, functional diarrhea5.Inflammatory: enteritis (viral, bacterial, fungal, nonspecific)6.Endocrine: Zollinger-Ellison syndrome7.Drugs: neomycin, phenindione, cathartics8.Collagen disease: scleroderma, lupus, polyarteritis9.Lymphoma10.Benign + malignant small bowel tumors11.Vascular disease12.CHF, agammaglobulinemia, amyloid, abetalipoproteinemia, intestinal lymphangiectasia(b)gastricvagotomy, gastrectomy, pyloroplasty, gastric fistula (to jejunum, ileum, colon) (c)pancreaticpancreatitis, pancreatectomy, pancreatic cancer, cystic fibrosis (d)hepatobiliaryintra- and extrahepatic biliary obstruction, acute + chronic liver disease ***Roentgenographic Signs In Malabsorption*** √ SMALL BOWEL WITH NORMAL FOLDS + FLUID1.Maldigestion (deficiency of bile salt / pancreatic enzymes)2.Gastric surgery3.Alactasia √ SMALL BOWEL WITH NORMAL FOLDS + WET1.Sprue2.Dermatitis herpetiformis √ DILATED DRY SMALL BOWEL1.Scleroderma2.Dermatomyositis3.Pseudoobstruction: no peristaltic activity √ DILATED WET SMALL BOWEL1.Sprue2.Obstruction3.Blind loop √ THICKENED STRAIGHT FOLDS + DRY SMALL BOWEL1.Amyloidosis (malabsorption is unusual)2.Radiation3.Ischemia4.Lymphoma (rare)5.Macroglobulinemia (rare) √ THICKENED STRAIGHT FOLDS + WET SMALL BOWEL1.Zollinger-Ellison syndrome2.Abetalipoproteinemia: rare inherited disease characterized by CNS damage, retinal abnormalities, steatorrhea, acanthocytosis √ THICKENED NODULAR IRREGULAR FOLDS + DRY SMALL BOWEL 1.Lymphoid hyperplasia2.Lymphoma3.Crohn disease4.Whipple disease5.Mastocytosis √ THICKENED NODULAR IRREGULAR FOLDS + WET SMALL BOWEL1.Lymphangiectasia2.Giardiasis3.Whipple disease (rare)

Small Bowel Nodularity With Malabsorption *mnemonic:*"**W**hat **I**s **H**is **M**ain **A**im? **L**ay **E**ggs, **B**y **G**od"**W**hipple disease
Intestinal lymphangiectasia
Histiocytosis
Mastocytosis
Amyloidosis
Lymphoma, **L**ymph node hyperplasia
Edema
Blood
Giardiasis

Notes:

Cholecystokinin =CCK = 33 amino acid residues (former name: Pancreozymin); the 5 C-terminal amino acids are identical to those of gastrin, causing similar effects as gastrin*Produced in:* duodenal + upper intestinal mucosa*Released by:*fatty acids, some amino acids (phenylalanine, methionine), hydrogen ionsEffects: @Stomach(1)weakly stimulates HCl secretion(2)given alone:inhibits gastrin, which leads to decrease in HCl production(3)stimulates pepsin secretion(4)stimulates gastric motility@Pancreas(1)stimulates secretion of pancreatic enzymes(= Pancreozymin) (2)stimulates bicarbonate secretion (weakly by direct effect; strongly through potentiating effect on secretin)(3)stimulates insulin release@Liver(1)stimulates water + bicarbonate secretion@Intestine(1)stimulates secretion of Brunner glands(2)increases motility@Biliary tract(1)strong stimulator of gallbladder contraction(2)relaxation of sphincter of Oddi

Notes:

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Gastrin =17 amino acid peptide amide;PentagastrinPENTAGASTRIN =acyl derivative of the biologic active C-terminal tetrapeptide amide*Produced in:* antral cells + G-cells of pancreas*Released by:* (a)vagal stimulation, gastric distension(b)short-chain alcohol (ethanol, propanol)(c)amino acids (glycine, ß-alanine)(d)caffeine(e)hypercalcemiamediated by neuroendocrine cholinergic reflexes *Inhibited by:* drop in pH of antral mucosa to <3.5Effects: @Stomach:(1)stimulation of gastric HCl secretion from parietal cells, which in turn:(2)increases pepsinogen production by chief cells through local reflex(3)increase in antral motility(4)trophic effect on gastric mucosa (parietal cell hyperplasia)@Pancreas(1)strong increase in enzyme output(2)weakly stimulates fluid + bicarbonate output(3)stimulates insulin release@Liver(1)water + bicarbonate secretion@Intestine(1)stimulates secretion of Brunner glands(2)increases motility@Gallbladder(1)stimulates contraction@Esophagus(1)increases resting pressure of LES

Notes:

Glucagon *Produced in:* a-cells (and b-cells) of pancreas*Released by:*low blood glucose levelsEffects: @Intestines(1)lowers pressure of GE sphincter(2)hypotonic effect on duodenum > jejunum > stomach > colon@Hormones(1)releases catecholamines from the adrenal gland that paralyze intestinal smooth muscle(2)increases serum insulin + glucose levels (mobilization of hepatic glycogen)@Biliary tract(1)increases bile flow(2)relaxes gallbladder + sphincter of Oddi *Dose for radiologic imaging:*1 mg maximum◊IV administration causes a quick response + rapid dissipation of action!◊IM administration prolongs onset + increases length of action!*Half-life:*3-6 minutes*Side effects:*nausea + vomiting, weakness, dizziness (delayed onset of 1.5-4 hours after IM administration)*Contraindication:* (1)hypersensitivity / allergy to glucagon: urticaria, periorbital edema, respiratory distress, hypotension, coronary artery spasm (?), circulatory arrest(2)known hypertensive response to glucagon(3)pheochromocytoma: glucagon stimulates release of catecholamines(4)insulinoma: insulin-releasing effect may result in hypoglycemia(5)glucagonoma(6)poorly controlled diabetes mellitus

Notes:

Secretin *Produced in:* duodenal mucosa*Released by:* hydrogen ions providing a pH <4.5Effects: @Stomach(1)inhibits gastrin activity, which leads to decrease in HCl secretion(2)stimulates pepsinogen secretion by chief cells (potent pepsigogue)(3)decreases gastric and duodenal motility + contraction of pyloric sphincter@Pancreas(1)increases alkaline pancreatic secretions ($NaHCO_3$)(2)weakly stimulates enzyme secretion(3)stimulates insulin release@Liver(1)stimulates water + bicarbonate secretion (most potent choleretic)@Intestine(1)stimulates secretion of Brunner glands(2)inhibits motility@Esophagus(1)opens LES

Notes:

Lower Esophageal Anatomy A.**Esophageal Vestibule**
=saccular termination of lower esophagus with upper boundary at tubulovestibular junction + lower boundary at esophagogastric junction√ collapsed during resting state√ assumes bulbous configuration with swallowing(a)tubulovestibular junction = A level = junction between tubular and saccular esophagus(b)phrenic ampulla = bell-shaped part above diaphragm (term should be discarded because of dynamic changes of configuration)(c)submerged segment = infrahiatal part of esophagus√ widening / disappearance is indicative of gastroesophageal reflux disease (GERD)B.**Gastroesophageal Junction**
Site:at upper level of gastric sling fibers, straddles cardiac incisura demarcating the left lateral margin of GE junctionC.**Z line** = B level = squamocolumnar junction linenot acceptable criterion for locating GE junction Site:1-2 cm above gastric sling fibersD.**Lower Esophageal Sphincter**
=physiologic 2-4 cm high pressure zone corresponding to esophageal vestibule√ tightly closed during resting state√ assumes bulbous configuration with swallowing

Notes:

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Muscular Rings Of Esophagus *A Ring* =contracted / hypertrophied muscles in response to incompetent GE sphincter ● rarely symptomatic / dysphagiaLocation:at tubulovestibular junction = superior aspect of vestibule√ usually 2 cm proximal to GE junction at upper end of vestibule√ varies in caliber during the same examination, may disappear on maximum distension√ broad smooth narrowing with thick rounded margins√ visible only if tubular esophagus above + vestibule below are distended
B Ring =sling fibers representing a U-shaped thickening of inner muscle layers with open arm of U toward lesser curvature = inferior aspect of vestibuleLocation:< 2 cm from hiatal margins√ only visible when esophagogastric junction is above hiatus√ thin ledge-like ring just below the mucosal junction (Z line)

Notes:

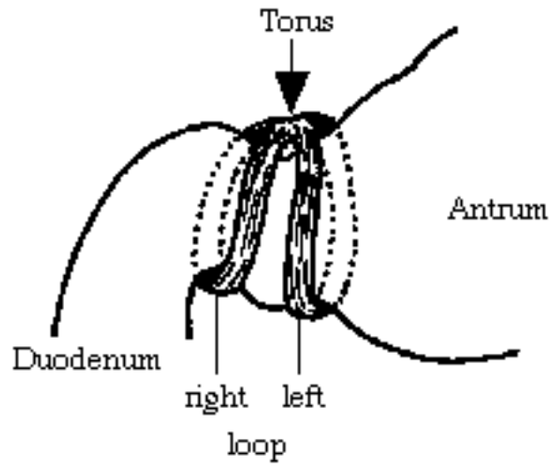
Gastric Cells 1.Chief cells=peptic / zymogenic cellsLocation:body + fundusproduce:pepsinogen2.Parietal cells=oxyntic cellsLocation:body + fundusproduce:H^+, Cl^-, intrinsic factor, prostaglandins3.Mucous neck cellsproduce:mucoprotein, mucopolysaccharide, aminopolysaccharide sulfate4.Argentaffine cells=enteroendocrine cellsLocation:body + fundusproduce:glucagon-like substance (A-cells), somatostatin (D-cells), vasoactive intestinal polypeptide (D_1-cells), 5-hydroxytryptamine (EC-cells)5.G-cellsLocation:pylorusproduce:gastrin

Notes:

Effect Of Bilateral Vagotomy = cholinergic denervation (1)decreased MOTILITY of stomach + intestines(2)decreased GASTRIC SECRETION(3)decreased TONE OF GALLBLADDER + bile ducts(4)increased TONE OF SPHINCTERS (Oddi + lower esophageal sphincter)

Notes:

Pylorus =fan-shaped specialized circular muscle fibers with:(a)distal sphincteric loop = right canalis loop√ corresponds to radiologic pyloric sphincter(b)proximal sphincteric loop = left canalis loop√ 2 cm proximal to distal sphincteric loop on greater curvature (seen during complete relaxation)(c)torus= fibers of both sphincters converge on the lesser curvature side to form a muscular prominence; prolapse of mucosa between sphincteric loops produces a niche simulating ulcer√ pyloric channel 5-10 mm long, wall thickness of 4-8 mm√ concentric indentation of the base of the duodenal bulb



Notes:

Duodenal Segments (1)duodenal bulb + short postbulbar segment:intraperitoneal + freely movable (2)descending duodenum:retroperitoneal attached to head of pancreas (3)horizontal = transverse segment:retroperitoneal crossing the spine (4)ascending portionretroperitoneal ascending to level of duodenojejunal junction VARIATIONS: (1)"mobile duodenum" / "water-trap duodenum"= long postbulbar segment with undulation / redundancy (2)duodenum inversum / duodenum reflexum= distal duodenum ascends to the right of spine to the level of duodenal bulb + then crosses spine horizontally + fixated in normal location

Notes:

Small Bowel Folds A.NORMAL FOLD THICKNESS@jejunum1.7-2.0 mm>2.5 mm pathologic@ileum1.4-1.7 mm>2.0 mm pathologicB.NORMAL NUMBER OF FOLDS@jejunum4-6 / inch@ileum3-5 / inchC.NORMAL FOLD HEIGHT@jejunum3.5-7.0 mm@ileum2.0-3.5 mmD.NORMAL LUMEN DIAMETER@upper jejunum3.0-4.0 cm>4.5 cm pathologic@lower jejunum2.5-3.5 cm>4.0 cm pathologic@ileum2.0-2.8 cm>3.0 cm pathologic RULE OF 3s: ◊wall thickness <3 mm◊ valvulae conniventes <3 mm◊diameter <3 cm◊air-fluid levels <3

Notes:

Normal Bowel Caliber *mnemonic:*"3-6-9-12"3 cmmaximal size of small bowel6 cmmaximal size of transverse colon9 cmmaximal size of cecum12 cmmaximal caliber of cecum before it may burst

Notes:

Small Bowel Peristalsis A.INCREASED1.Vagal stimulation2.Acetylcholine3.Anticholinesterase (eg, neostigmine)4.CholecystokininB.DECREASED1.Atropine (eg, Pro-Banthine®)2.Bilateral vagotomy

Notes:

Intestinal Gas A.INFLUX1.Aerophagia2 L2.Liberation from intestinal tract(a)neutralization of bicarbonate insecretions (CO_2)8 L(b)bacterial fermentation (CO_2, H_2,CH_4, H_2S)15 L3.Diffusion from blood (N_2, O_2, CO_2)B.EFFLUX1.Diffusion from intestines into bloodand expulsion from lung50 L2.Expulsion from anus2 L

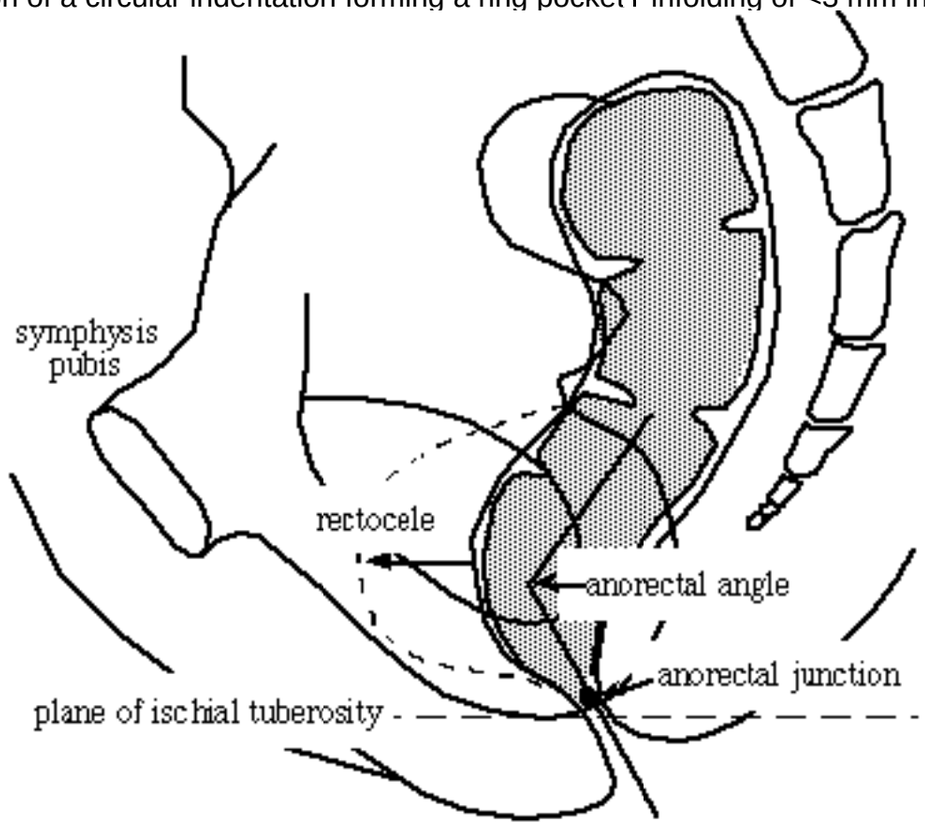
Notes:

Intestinal Fluid A.INFLUX1.Oral ingestion2.5 L2.Intestinal secretions8.2 Lsaliva1.5 Lbile0.5 L gastric secretions2.5 Lpancreatic secretions0.7 Lintestinal secretions3.0 LB.EFFLUX1.Peranal0.1 L2.Intestinal resorption (primarily in ileum+ ascending colon)10.6 L

Notes:

Defecography / Evacuation Proctography

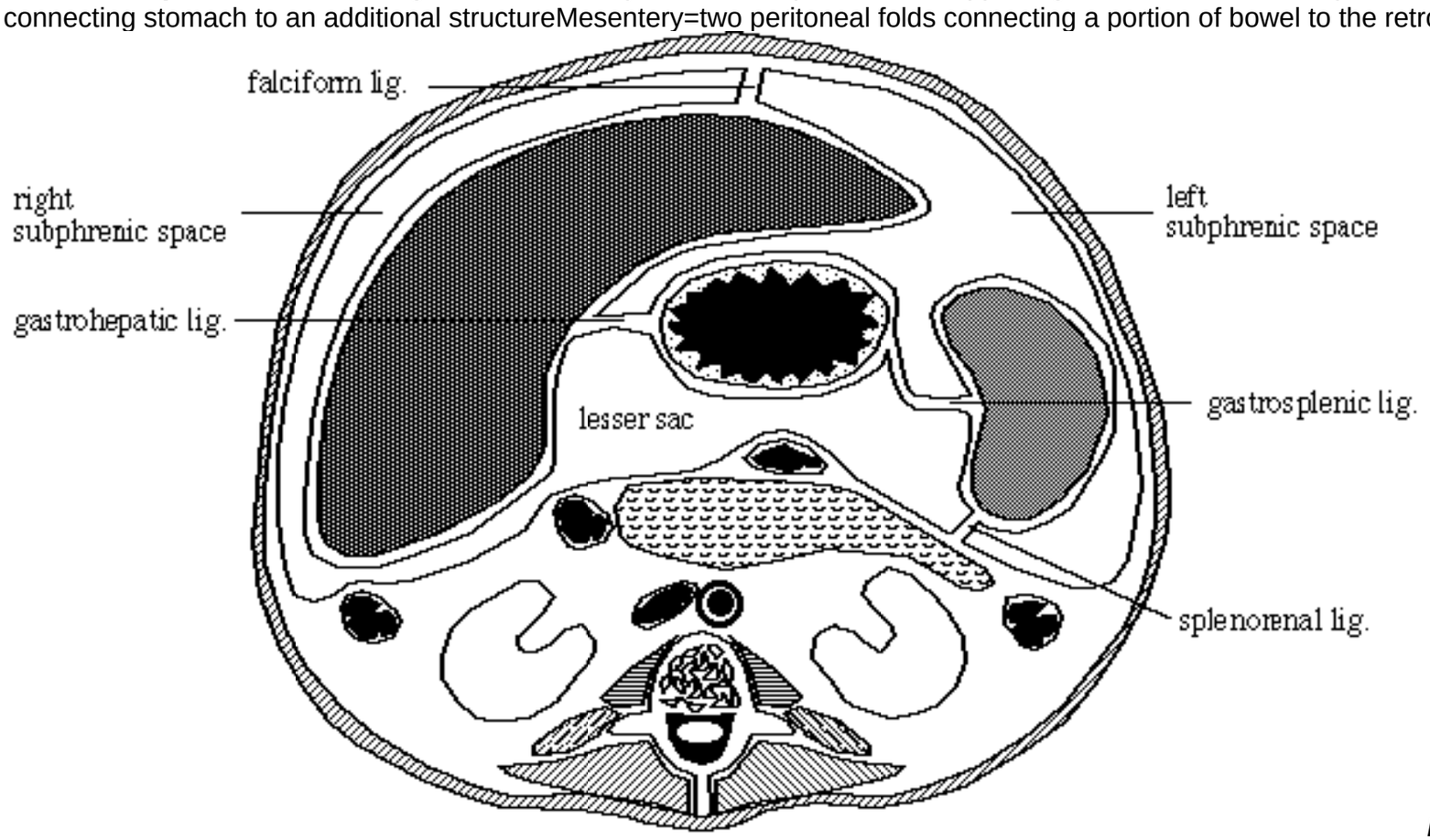
evacuation time=15 (range 5-40) secondsanorectal angle=angle formed between central axis of anal canal + line parallel to posterior wall of rectum√ 90° at rest and during voluntary contraction (squeeze maneuver)√ more obtuse during defecation straining (void) anorectal junction=point of taper of distal rectal ampulla as it merges with the anal canal; position of anorectal junction referenced to plane of ischial tuberosities = 0-3.5 cm; elevation during squeeze of 0-4.5 cm; elevation during void of -3.0-0 cm rectovaginal space=space between vagina and rectum perineum=area between external genital organs and anal verge rectocele=measurement of anteroposterior depth of convex wall protrusion extending beyond expected margin of normal rectal wallsmall<2 cm;moderate= 2-4 cm;large>4 cm peritoneocele=extension of rectouterine excavation to below upper third of vagina; containing liquid / bowel / omentum enterocele=bowel present in peritoneocele rectal prolapse=descent of entire thickness of rectal wall through anal verge rectal intussusception =descent of the entire thickness of the rectal wall possibly extending into anal canal; starting 6-11 cm above anus; accompanied by formation of a circular indentation forming a ring pocket√ infolding of <3 mm in width / > 3 mm in width / intraluminal narrowing / descent into anal canal / external prolapse



Defecographic Measurements

Notes:

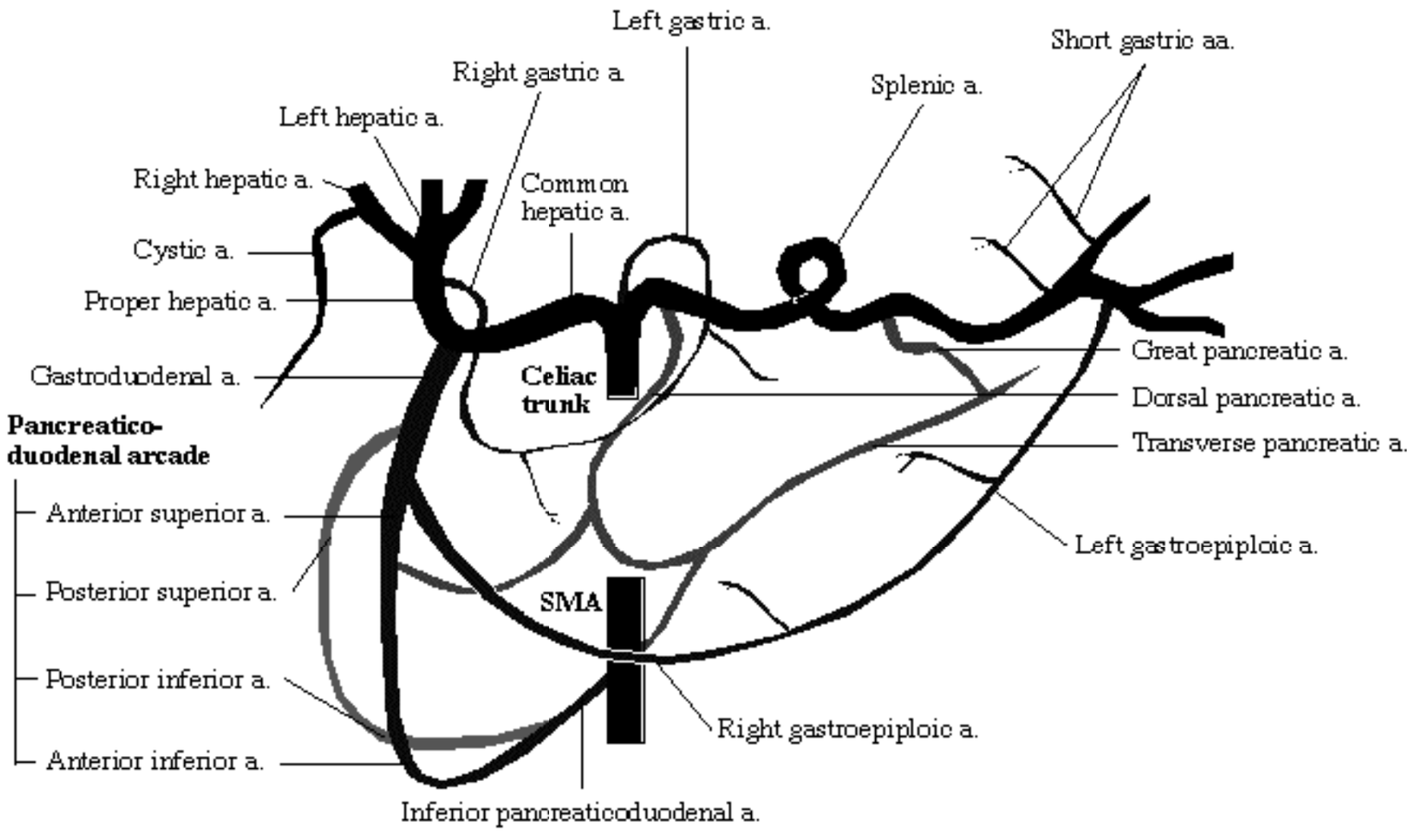
Peritoneal Spaces *Definitions:* Ligament=formed by two folds of peritoneum supporting a structure within the peritoneal cavityOmentum=specialized structure connecting stomach to an additional structureMesentery=two peritoneal folds connecting a portion of bowel to the retroperitoneum



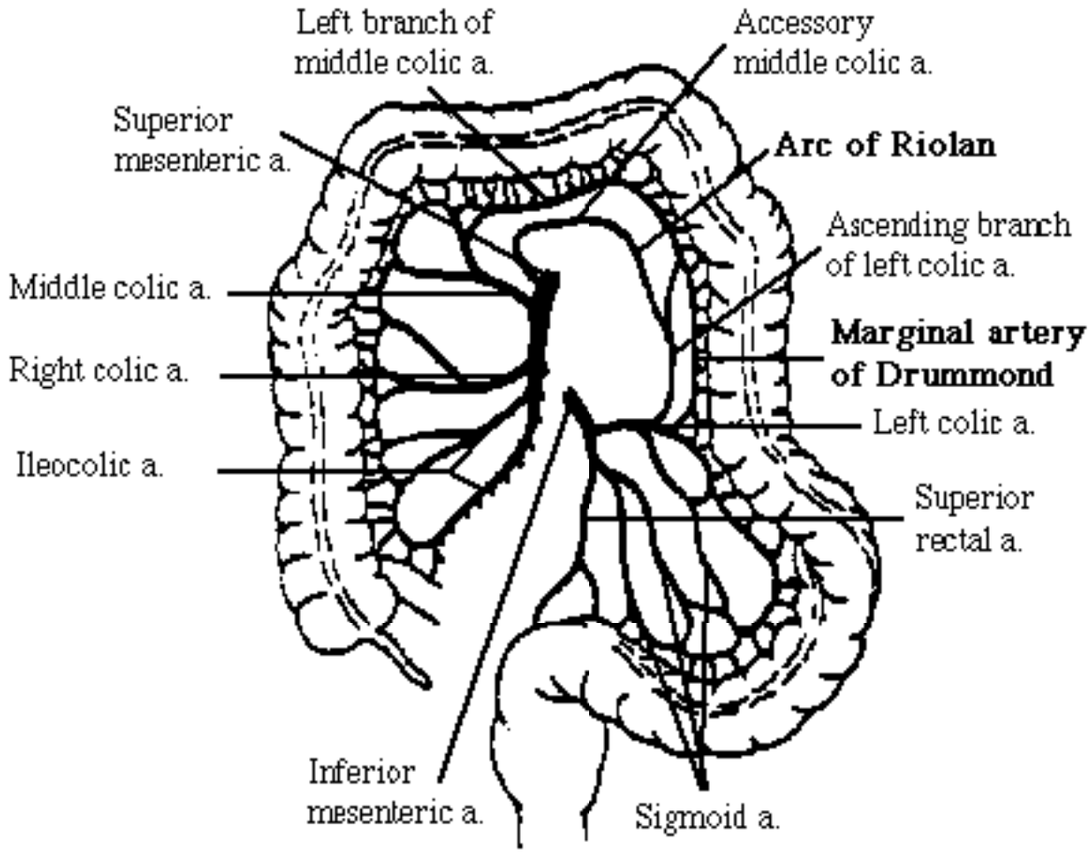
Embryology: above transverse mesocolon: A.RIGHT PERITONEAL SPACEforms perihepatic space + lesser sac: 1.Right subphrenic space:-located between right hepatic lobe + diaphragm-limited posteriorly by right superior reflection of coronary lig. + right triangular ligament2.Right subhepatic space:-divided into ● anterior right subhepatic space: located just posterior to porta hepatis, communicating with lesser sac via epiploic foramen (= foramen of Winslow) ● posterior right subhepatic space = Morison pouch = hepatorenal fossa◊Most dependent portion of the abdomen in supine patient!3.Bare area of liver-situated between reflections of right + left coronary ligaments-continuous with right anterior pararenal space4.Lesser sac: ● superior recess:-surrounds medial aspect of caudate lobe-separated from splenic recess by gastropancreatic fold ● splenic recess:-extends across midline to splenic hilum ● inferior recess:-separates stomach from pancreas + transverse mesocolon-anteriorly covered by lesser omentum5.Lesser omentum = combination of gastrohepatic ligament + hepatoduodenal ligament6.Right triangular ligament:-forms from coalescence of superior + inferior reflections of right coronary ligament-divides posterior aspect of right perihepatic space into right subphrenic space + posterior right subhepatic space B.LEFT PERITONEAL SPACEforms left subphrenic space 1.Left subphrenic space:-artificially divided into ● immediate subphrenic space:between diaphragm + gastric fundus ● perisplenic space:bounded inferiorly by phrenicocolic lig. ● subhepatic space = gastrohepatic recess:located between lateral segment of left hepatic lobe + stomach-separated from right subphrenic space by falciform ligament2.Left triangular ligament:-forms from coalescence of superior + inferior reflections of left coronary ligament-located along superior aspect of left hepatic lobe C.DORSAL MESENTERY gives rise to:1.Gastrophrenic ligament-courses through immediate subphrenic space -suspends stomach from dome of diaphragm2.Gastropancreatic ligament-formed by proximal left gastric artery-attaches posterior aspect of gastric fundus to retroperitoneum-partially separates superior recess of lesser sac from splenic recess 3.Phrenicocolic ligament-major suspensory ligament of spleen-attaches proximal descending colon to left hemidiaphragm-separates left subphrenic space from left paracolic gutter 4.Gastrosplenic ligament-remnant of dorsal mesentery-connects greater curvature of stomach with splenic hilum-contains short gastric vessels 5.Splenorenal ligament-connects posterior aspect of spleen to anterior pararenal space-contributes to left lateral + posterior border of lesser sac-encloses tail of pancreas + distal splenic artery + proximal splenic vein 6.Gastrocolic ligament-forms portion of anterior border of lesser sac-forms superior aspect of greater omentum-connects greater curvature of stomach with superior aspect of transverse colon-contains gastroepiploic vessels D.VENTRAL MESENTERY gives rise to: 1.Falciform ligament=sickle-shaped fold composed of two layers of peritoneum-attaches ventral surface of liver to anterior abdominal wall-its right layer continues into the superior layer of the coronary ligament, its left layer continues into the anterior layer of the left triangular ligament-contains ligamentum teres (= obliterated umbilical vein) in its free inferoposterior margin-continuous with fissure for ligamentum venosum 2.Gastrohepatic ligament:-arises in fissure of ligamentum venosum-connects medial aspect of liver to lesser curvature of stomach as part of lesser omentum-contains left gastric artery, coronary vein, lymph nodes
3.Hepatoduodenal ligament:-forms inferior edge of gastrohepatic ligament-forms anterior margin of epiploic foramen-extends from proximal duodenum to porta hepatis-contains common hepatic duct, common bile duct, hepatic artery, portal vein below transverse mesocolon: A.VENTRAL MESENTERY regressesB.DORSAL MESENTERY forms:1.Tansverse mesocolon:-suspends transverse colon from retroperitoneum along anteroinferior edge of pancreas-forms posteroinferior border of lesser sac-contains middle colic vessels2.Small bowel mesentery:-suspends small bowel from retroperitoneum-extends from ligament of Treitz to ileocecal valve-contains superior mesenteric vessels + lymph nodes3.Sigmoid mesocolon:-attaches sigmoid colon to posterior pelvic wall-contains sigmoid + hemorrhoidal vessels4.Greater omentum:-inferior continuation of gastrocolic ligament-formed by double reflection of dorsal mesogastrium thus composed of 4 layers of peritoneum5.Superior + inferior ileocecal recesses:-located above + below terminal ileum6.Retrocecal space:-present only if peritoneum reflects posterior to cecum7.Right + left paracolic gutters:-located lateral to ascending + descending colon8.Intersigmoid recess:-located along undersurface of sigmoid mesocolon

Notes:

BLOOD SUPPLY



Blood Supply of Stomach, Duodenum, and Pancreas



Blood Supply of Large Intestine

Notes:

ACHALASIA
=failure of organized peristalsis + relaxation at level of lower esophageal sphincter*Etiology:*(a)idiopathic: abnormality of Auerbach plexus / medullary dorsal nucleus; ? neurotropic virus, ? gastrin hypersensitivity(b)Chagas disease √ megaesophagus = dilatation of esophagus beginning in upper 1/3, ultimately entire length√ absence of primary peristalsis below level of cricopharyngeus√ nonperistaltic contractions√ "bird-beak" / "rat tail" deformity = V-shaped conical + symmetric tapering of stenotic segment with most marked narrowing at GE junction√ Hurst phenomenon = temporary transit through cardia when hydrostatic pressure of barium column is above tonic LES pressure√ sudden esophageal emptying after ingestion of carbonated beverage (eg, Coke)√ "vigorous achalasia" = numerous tertiary contractions in nondilated distal esophagus of early achalasia√ prompt relaxation of LES upon amyl nitrate inhalation (smooth-muscle relaxant)CXR: √ right convex opacity behind right heart border; occasionally left convex opacity if thoracic aorta tortuous√ right convex opacity may be tethered by azygos arch allowing for greater dilatation above + below√ air-fluid level (stasis in thoracic esophagus filled with retained secretions + alimentary residue)√ small / absent gastric air bubble√ anterior displacement + bowing of trachea (LAT view)√ patchy bilateral alveolar opacities resembling acute / chronic aspiration pneumonia (M. fortuitum-chelonei infection)*Cx:*esophageal carcinoma in 2-7% (usually midesophagus)*Rx:*pneumatic dilatation / surgical myotomy *DDx:*(1)Neoplasm (separation of gastric fundus from diaphragm; normal peristalsis; asymmetric tapering)(2)Peptic stricture of esophagus

Notes:

ADENOMA OF SMALL BOWEL
Location:duodenum (21%), jejunum (36%), ileum (43%) esp. ileocecal valve*Histo:*(1)Hamartomatous polyp (77%), multiple in 47%, 1/3 of multiple lesions associated with Peutz-Jeghers syndrome(2)adenomatous polyp (13%), may have malignant potential(3)polypoid gastric heterotopic tumor (10%)

Notes:

ADENOMATOUS COLONIC POLYP
=EPITHELIAL POLYPMost common benign colonic tumor (68-79%)*Predisposed:*previously detected polyp / cancer; family history of polyps / cancer; idiopathic inflammatory bowel disease; Peutz-Jeghers syndrome; Gardner syndrome; familial polyposis*Prevalence:*3% in 3rd decade; 10% in 7th decade;26% in 9th decadeLocation:rectum (21-34%); sigmoid (26-38%); ascending colon (9-12%); transverse colon (12-13%); descending colon (6-18%); multiple in 35-50% (usually <5-10 in number) *Histo:* 1.Tubular adenoma (75%)=cylindrical glandular structure lined by stratified columnar epithelium + nests of epithelium within lamina propria√ usually < 10 mm in diameter√ often pedunculated if >10 mm*malignant potential:*<10 mm in 1%; 10-20 mm in 10%; >20 mm in 35%2.Tubulovillous adenoma (15%)=mixture between tubular + villous adenoma*malignant potential:* <10 mm in 4%; 10-20 mm in 7%; >20 mm in 46%3.Villous adenoma (10%)=thin frondlike projections from surface with epithelium outlining their margins ("villous fronds") ● potassium depletion√ often >20 mm in diameter with papillary surface√ often broad-based sessile lesion√ heterogeneous low attenuation on CT (due to capacious mucin becoming trapped within papillary projections + crevices)*malignant potential:* <10 mm in 10%; 10-20 mm in 10%; >20 mm in 53% *Size & malignancy:* <5 mm in 0%; 5-9 mm in 1%; 10-20 mm in 10%; >20 mm in 46% malignant ◊All polyps >10 mm should be removed!◊Time for adenoma-carcinoma sequence probably averages 10-15 years! *Probability of coexistent colonic growth:* -synchronous adenoma in 50%-metachronous adenoma in 30-40%-synchronous adenocarcinoma in 1.5-5%-metachronous adenocarcinoma in 5-10% ● asymptomatic (75%) ● diarrhea, abdominal pain ● peranal hemorrhage (67%)Colonoscopy (incomplete in 16-43%) BE (rate of detection of polyps <10 mm higher with double than single contrast; false-negative rate of 7%): √ sessile flat / round polyp√ pedunculated polyp: stalk >2 cm in length almost always indicative of a benign polyp√ suggestive of malignancy: irregular lobulated surface, broad base = width of the base greater than height, retraction of colonic wall = dimpling / indentation / puckering at base of tumor, interval growth√ lacelike / reticular surface pattern CHARACTERISTIC for villous adenoma (occasionally in tubular adenoma)*DDx:*(1)Nonneoplastic: hyperplastic polyp, inflammatory pseudopolyp, lymphoid tissue, ameboma, tuberculoma, foreign-body granuloma, malacoplakia, heterotopia, hamartoma(2)Neoplastic subepithelial: lipoma, leiomyoma, neurofibroma, hemangioma, lymphangioma, endothelioma, myeloblastoma, sarcoma, lymphoma, enteric cyst, duplication, varix, pneumatosis, hematoma, endometriosis

Notes:

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ADENOCARCINOMA OF SMALL BOWEL

*Frequency:*about 50 times less common than colonic carcinoma*Risk factors:*Crohn disease, sprue, Peutz-Jeghers syndrome, Lynch syndrome II, congenital bowel duplication, ileostomy, duodenal / jejunal bypass surgery*Histo:*mostly moderately to well differentiated; may arise in villous tumors / de novo; no correlation between size and invasivenessLocation:duodenum (~50%, especially near ampulla), jejunum > ileum√ annular stricture with "overhanging edges" (60%)√ lobulated / ovoid polypoid sessile mass (41%)◊Duodenal tumors tend to be papillary / polypoid!√ ulcerated mass (27%)CT: √ soft-tissue mass with heterogeneous attenuation√ moderate contrast enhancement*Cx:*intussusception*DDx:*lymphoma (lymphadenopathy more bulky)

Notes:

AFFERENT LOOP SYNDROME
=PROXIMAL LOOP / BLIND LOOP SYNDROME=partial intermittent obstruction of afferent loop leading to overdistension of loop by gastric juices after Billroth-II gastrojejunostomy*Cause:*gastrojejunostomy with left-to-right anastomosis (= proximal jejunal loop attached to greater curvature instead of lesser curvature), mechanical factors (intussusception, adhesion, kinking), inflammatory disease, neoplastic infiltration of local mesentery or anastomosis, idiopathic motor dysfunction ● postprandial epigastric fullness relieved by bilious vomiting ● vitamin B_{12} deficiency with megaloblastic anemia ● afferent loop with abnormal bacterial flora (Gram negative, resembling colon in quality + quantity)Abdominal plain film: √ normal in 85% (no air in lumen of afferent loop)UGI: √ preferential emptying of stomach into proximal loop√ proximal loop stasis√ regurgitationCT: √ rounded water-density masses adjacent to head + tail of pancreas forming a U-shaped loop√ oral contrast material may not enter loop√ may result in biliary obstruction (increased pressure at ampulla)*Rx:*antibiotic therapy

Notes:

AIDS
◊Gastrointestinal involvement due to opportunistic infections + AIDS-associated neoplasms◊Pathologic abnormalities at multiple sites with single / several opportunistic organisms are frequent! AIDS-defining illness related to CD4 T-lymphocyte count [cells/µL]: <400extrapulmonary Mycobacterium tuberculosis, Kaposi sarcoma<200Candida albicans, Histoplasma capsulatum, Cryptosporidium species, Pneumocystis carinii, Non-Hodgkin lymphoma<100Cytomegalovirus, Herpes simplex virus, Mycobacterium avium complex A.VIRAL PATHOGENS1.**Cytomegalovirus infection**
◊Most common cause of life-threatening opportunistic viral infection in AIDS patients!*Organism:*double-stranded DNA virus of the herpes family*Infection:*ubiquitous among humans occurring at an early age in populations with poor sanitation + crowded living conditions◊Result of reactivation of latent virus in previously infected host!*Prevalence:*13% of all gastrointestinal diseases in AIDS patients*Path:*infection of endothelial cells leads to small vessel vasculitis resulting in hemorrhage, ischemic necrosis, ulceration*Histo:*large mononuclear epithelial / endothelial cells that contain intranuclear /cytoplasmatic inclusions with surrounding inflammationLocation:colon > small bowel (terminal ileum) > esophagus > stomach@Esophagus√ single / multiple large superficial ulcers@Small bowel√ luminal narrowing secondary to marked bowel wall thickening√ thickened irregular folds (vasculitis leading to thrombosis + ischemia)√ penetrating ulcer ± perforation√ CMV pseudotumor (uncommon)@Colon (CMV colitis) ● hematochezia, crampy abdominal pain, fever√ findings of toxic megacolon√ discrete small well-defined nodules (similar to lymphoid nodular hyperplasia) throughout entire colon√ aphthous ulcers on background of normal mucosa√ marked bowel wall thickening√ double-ring / target sign on CT (due to increased submucosal edema)√ ascites√ inflammation of pericolonic fat + fascia*Rx:*ganciclovir (effective in 75%)2.**Herpes simplex virus infection**
◊Result of reactivation of latent virus in previously infected host*Organism:*neurotropic DNA virus of herpes family*Prevalence:*70% for type 1, 16% for type 2 (endemic in United States); type 2 much more common in AIDS*Infection:*direct inoculation through mucous membrane contact; from dormant state in root ganglia reactivated + transported via efferent nerves to mucocutaneous surfaceLocation:oral cavity, esophagus, rectum, anus√ multiple small discrete ulcers3.**HIV infection**
◊Not an AIDS-defining illness!*Infection:*acute HIV-infection with transient immunosuppression / during AIDS√ >2 cm large solitary ulcer in the mid- or distal esophagus (HIV-infected cells cause alterations in cytokines resulting in infiltration of inflammatory cells into submucosa + destruction of mucosa)*Rx:*corticosteroids B.FUNGAL PATHOGENS1.**Candidiasis**
◊The absence of thrush does not exclude the diagnosis of candidal esophagitis!*Organism:*commensal fungus Candida albicans*Prevalence:*10-20% (in United States); up to 80% in developing countriesLocation:oral cavity, esophagus√ discrete linear / irregular longitudinally oriented filling defects in esophagus*Cx:*disseminated systemic candidiasis (rare + indicative of granulocytopenia from chemotherapy / direct inoculation via catheter)2.**Histoplasmosis**
*Organism:*dimorphic opportunistic fungus*Prevalence:*10% GI involvement with disseminated histoplasmosis in AIDS patientsLocation:colon > terminal ileum√ segmental inflammation / applecore lesion / bowel stricture√ hepatosplenomegaly√ mesenteric lymphadenopathy√ diffuse hypoattenuation of spleen C.PROTOZOAN PATHOGENS1.**Cryptosporidiosis**
◊One of the most common causes of enteric disease in AIDS patients!*Organism:*intracellular parasite Cryptosporidium*Prevalence:*16% (in United States) + up to 48% (in developing countries) in patients with diarrhea ● severe diarrhea with fluid loss of 10-17 L/dayLocation:jejunum > other small bowel > stomach > colon√ Cryptosporidium antritis (= area of focal gastric thickening + ulceration)√ small bowel dilatation (increased secretions)√ regular fold thickening + effacement (atrophy, blunting, fusion, loss of villi)√ "toothpaste" appearance of small bowel (mimicking sprue)√ dilution of barium (hypersecretion)√ marked antral narrowing (extensive inflammation)*Dx:*microscopic identification in stool / biopsy2.**Pneumocystosis**
◊Likely to occur inpatients treated with aerosolized pentamidine!*Organism:*eukaryotic microbe Pneumocystis carinii*Prevalence:*pulmonary infection in 75% of AIDS patients; in <1% disseminationLocation:liver, spleen, lymph nodes√ hepatic + splenic + nodal punctate calcifications√ multiple tiny echogenic foci in spleen√ multiple low-attenuation lesions of varying size in spleen (foamy eosinophilic material) with subsequently progressive rimlike / punctate calcifications D.BACTERIAL PATHOGENS1.**Tuberculosis**
◊Most common cause of serious HIV-related infection worldwide with tendency to occur earlier than other AIDS-defining opportunistic infections!*Prevalence:*4% (in United States) + 43% (in developing countries) of HIV-infected persons*Infection:*swallowing of infected sputum; hematogenous spread from pulmonary focus; direct extension from lymph nodeLocation:lymph nodes, liver, spleen, peritoneum, GI tract (especially ileum, colon, ileocecal valve)√ low-attenuation mesenteric lymphadenopathy (suggestive of necrosis)√ segmental ulceration√ inflammatory stricture√ hypertrophic lesion resembling polyp or mass2.**Mycobacterium avium complex infection**
=PSEUDO-WHIPPLE DISEASE◊Most common opportunistic infection of bacterial origin in AIDS patients!◊Most common nontuberculous mycobacterial infection in AIDS patients!*Organism:*facultative intracellular acid-fast bacillus M. avium / M. intracellulare*Infection:*invasion of Peyer patches + adjacent mesenteric lymph nodes*Histo:*true granulomas with Langhans giant cells and caseous necrosis are rare because infection occurs in patients with advanced disease and a CD4 cell count of <100/µL ● diarrhea, malabsorptionLocation:jejunum (most common)√ mild dilatation of middle + distal small bowel√ diffuse irregular mucosal fold thickening and nodularity without ulceration√ mesenteric + retroperitoneal lymphadenopathy (1.0-1.5 cm in size) with homogeneous soft-tissue attenuation causing segmental separation of small bowel loops√ hepatosplenomegaly√ multiple tiny echogenic foci in liver + spleen (occasionally large hypoechoic / low-attenuation lesions)*Dx:*(1)visualization of large numbers of intracellular acid-fast bacilli in foamy histiocytes of tissue specimens(2)tissue culture*DDx:*Whipple disease (positive with periodic acid-Schiff stain just like M. avium, but not with acid-fast stain, responsive to tetracyclines)
E.OTHER INFECTIONS1.**Bacillary angiomatosis**
*Organism:*Rickettsiales Bartonella henselae*Histo:*characteristic pattern of vascular proliferation with bacilliLocation:cutis (mimicking Kaposi sarcoma), liver, spleen, lymph nodes√ peliosis (blood-filled cystic spaces) of liver / spleen√ abdominal lymphadenopathy with contrast enhancement2.**Isospora belli**
◊Infection resembles cryptosporidiosis*Organism:*protozoan pathogen*Histo:*oval oocysts within bowel lumen / epithelial cells; localized inflammation; fold atrophyLocation:small intestine ● severe watery diarrhea√ fold thickening F.AIDS-ASSOCIATED NEOPLASMS1.Kaposi sarcoma2.**Non-Hodgkin lymphoma**
◊2nd most common AIDS-associated neoplasm*Prevalence:*in 4-10% of AIDS patients (60 times higher risk compared with general population); occurs in all AIDS risk groups*Histo:*multiclonal B-cell lymphoma of high or intermediate grade ● at initial presentation widely disseminated disease often with extranodal involvementLocation:CNS, bone marrow, GI tract (stomach, small bowel)@Stomach√ circumferential / focal wall thickening√ mural mass ± ulceration@Small bowel√ diffuse / focal wall thickening√ excavated mass√ solitary / multiple liver lesions *Differential diagnostic considerations:* 1.Splenomegaly (31-45%)*Cause:*nonspecific (most), lymphoma, infection (M. avium-intracellulare, P. carinii)2.Lymphadenopathy (21-60%)*Cause:*reactive hyperplasia (most), Kaposi sarcoma, lymphoma, infectionsSize:<3 cm in diameter (in 95%)3.Hepatomegaly (20%)*Cause:*nonspecific, hepatitis, fatty infiltration, lymphoma, Kaposi sarcoma4.AIDS-related cholangiopathy:*Organism:*CMV, Cryptosporidium√ papillary stenosis of CBD√ dilatation of extra- and intrahepatic bile ducts√ periductal fibrosis√ strictures + irregularities of bile ducts resembling primary sclerosing cholangitis√ intraluminal polypoid filling defects5.AIDS-related esophagitis:*Organism:*Candida, herpes simplex, CMV√ giant esophageal ulcer: HIV (76%), CMV (14%)√ esophageal fistula / perforation: tuberculosis, actinomycosis6.Gastritis*Organism:*CMV (GE junction + prepyloric antrum), Cryptosporidium (antrum)7.AIDS enteritis*Organism:*Cryptosporidium, M. avium complex8.AIDS colitis-ischemic bowel-acute appendicitis-neutropenic colitis-pseudomembranous colitis-infectious colitis / ileitis9.Bowel obstruction(a)infection(b)intussusception: Kaposi sarcoma, lymphoma

Notes:

AMEBIASIS
=primary infection of the colon by protozoan Entamoeba histolyticaCountries:worldwide distribution, most common in warm climates; South Africa, Egypt, India, Asia, Central + South America (20%); United States (5%)Route:contaminated food / water (human cyst carriers); cyst dissolves in small bowel; trophozoites settle in colon; proteolytic enzymes + hyaluronidase lyse intestinal epithelium; may embolize into portal venous + systemic blood system*Histo:*amebic invasion of mucosa + submucosa causing tiny ulcers, which spread beneath mucosa + merge into larger areas of necrosis; mucosal sloughing; secondary bacterial infection ● asymptomatic for months / years ● acute attacks of diarrhea (loose mucoid bloodstained stools) ● fever, headache, nauseaLocation:(areas of relative stasis) right colon + cecum (90%) > hepatic + splenic flexures > rectosigmoid√ loss of normal haustral pattern with granular appearance (edema, punctate ulcers)√ "collarbutton" ulcers√ cone-shaped cecum√ several cm long stenosis of bowel lumen in transverse colon, sigmoid colon, flexures (result of healing + fibrosis); in multiple segments√ ameboma = hyperplastic granuloma with bacterial invasion of amebic abscess; usually annular + constricting / intramural mass / cavity continuous with bowel lumen; shrinkage under therapy in 3-4 weeks√ ileocecal valve thickened + fixed in open position with reflux√ involvement of distal ileum (10%)*Dx:*stool examination / rectal biopsy*Cx:*(1)Toxic megacolon with perforation(2)Amebic abscess in liver (2%), brain, lung (transdiaphragmatic spread of infection), pericolic, ischiorectal, subphrenic space(3)Intussusception in children (due to ameboma)(4)Fistula formation (colovesical, rectovesical, rectovaginal, enterocolic)

Notes:

AMYLOIDOSIS
=group of heterogeneous disorders caused by interstitial deposits of a protein-polysaccharide in various organs leading to hypoxia, mucosal edema, hemorrhage, ulceration, mucosal atrophy, muscle atrophy*Histo:*amorphous eosinophilic hyaline material deposited around terminal blood vessels, stains with Congo red + crystal violet; amyloid fibrils have b-pleated sheet structure (= b fibrilloses)*Biochemical classification (1979):* 1.AL amyloidosis(A = amyloidosis, L = light chain immunoglobulin) ● monoclonal protein in serum + urine ● occurs in primary amyloidosis + myeloma-associated amyloidosis*Histo:*massive deposits in muscularis mucosae + submucosa√ thickening of folds with polyps / large nodules2.SAA amyloidosis (S = serum, AA = amyloid A) ● occurs in secondary = reactive amyloidosis*Histo:*expansion of lamina propria√ coarse mucosal pattern + innumerable fine granular elevations3.AF amyloidosis (A = amyloid, F = familial) ● AF prealbumin as precursor of fibrils ● occurs in familial amyloidosis4.AS amyloidosis (A = amyloid, S = senile) ● AS prealbumin as precursor of fibrils ● occurs in senile amyloidosis√ massive amyloid deposition5.AH amyloidosis (A = amyloid, H = hemodialysis) ● b_2 microglobulin as precursor of fibrils6.AE amyloidosis (A = amyloid, E = endocrine) ● calcitonin produced by medullary thyroid carcinoma is precursor of fibrils *Reimann classification (1935):* 1.Primary = idiopathic amyloidosis=probably autosomal dominant inheritance with immunologically determined dysfunction of plasma cells ● absence of discernible preceding / concurrent diseaseLocation:(predominant involvement of connective tissues + mesenchymal organs) heart (90%), lung (30-70%), liver (35%), spleen (40%), kidneys (35%), adrenals, tongue (40%), GI tract (70%), skin + subcutis (25%)√ tendency to nodular deposition2.Secondary amyloidosis (most common form) ● following / coexistent with prolonged infectious / inflammatory processes*Cause:*rheumatoid arthritis (in 20%), Still disease, tuberculosis, osteomyelitis, leprosy, chronic pyelonephritis, bronchiectasis, ulcerative colitis, Waldenström macro-globulinemia, familial Mediterranean fever, lymphoreticular malignancy, paraplegiaLocation:spleen, liver, kidneys (>80%), breast, tongue, GI tract, connective tissue√ small amyloid deposits3.Amyloidosis associated with multiple myeloma ● may precede development of multiple myeloma*Incidence:*10-15%√ primary amyloidosis with osteolytic lesions in myelomatous disease4.Tumor-forming / organ-limited amyloidosis ● related to primary type(a)hereditary = familial amyloidosis(b)senile amyloidosis (limited to heart / brain / pancreas / spleen)√ large localized masses ◊GI involvement in primary more common than in secondary amyloidosis! ● malabsorption (diarrhea, protein loss) ● occult GI bleeding ● obstruction ● macroglossia@Esophagus (11%)√ loss of peristalsis√ megaesophagus@Stomach (37%) ● postprandial epigastric pain + heartburn ● acute erosive hemorrhagic gastritis(a)diffuse infiltrative form√ small-sized stomach with rigidity + loss of distensibility simulating linitis plastica (from thickening of gastric wall)√ effaced rugal pattern√ diminished / absent peristalsis√ marked retention of food(b)localized infiltration (often located in antrum)√ irregularly narrowed + rigid antrum√ thickened rugae√ superficial erosions / ulcerations(c)amyloidoma = well-defined submucosal mass@Small bowel (74%)(a)diffuse form (more common)√ diffuse uniform thickening of valvulae conniventes in entire small bowel√ broadened flat undulated mucosal folds (mucosal atrophy)√ "jejunalization" of ileum√ impaired intestinal motility√ small bowel dilatation(b)localized form (less common) √ multiple pea- / marble-sized deposits√ pseudoobstruction = physical + plain-film findings suggesting mechanical obstruction with patent large + small bowel on barium examination (involvement of myenteric plexus)*Cx:*small bowel infarction@Colon (27%):√ pseudopolyps in colon@ Bone:√ bone cysts@Spleen:*Histo:*(a)nodular form involving lymph follicles(b)diffuse form infiltrating red pulp√ discrete masses√ splenomegaly (4-13%)*Cx:*spontaneous splenic rupture (from vascular fragility + acquired coagulopathy)*Dx:*by rectal / gingival biopsy*DDx:*Whipple disease, intestinal lymphangiectasia, lymphosarcoma

Notes:

ANGIODYSPLASIA OF COLON
=VASCULAR ECTASIA = ARTERIOVENOUS MALFORMATION*Cause:*?; acquired lesion*Associated with:*aortic stenosis (20%) *Incidence at autopsy:*2%*Age:*majority >55 yearsLocation:(a)cecum + ascending colon (majority)(b)descending + sigmoid colon (25%) ● chronic intermittent low-grade bleeding ● occasionally massive bleeding√ "vascular tufts" = cluster of vessels during arterial phase along antimesenteric border√ early opacification of ileocolic vein√ densely opacified dilated tortuous ileocolic vein into late venous phase√ contrast extravasation (unusual)

Notes:

ANISAKIASIS
=parasitic disease of GI tract*Cause:*ingestion of Anisakis larvae present in raw / undercooked fish (mackerel, cod, pollack, herring, whiting, bonito, squid) consumed as sashimi, sushi, ceviche, lomi-lomi*Organism:*worm with straight / serpentine / circular threadlike appearance◊Site of penetration by larvae determines clinical form!@Gastric anisakiasis ● acute gastric pain, nausea, vomiting a few hours after ingestion (DDx: acute gastritis, peptic ulcer, food poisoning, neoplasia) ● eosinophilia√ mucosal edema√ about 3-cm-long threadlike filling defects (= larvae) @Intestinal anisakiasis ● diffuse abdominal tenderness / colicky abdominal pain, nausea, vomiting (DDx: acute appendicitis, regional enteritis, intussusception, ileus, diverticulitis, neoplasia) ● leukocytosis without eosinophilia (frequent)*Histo:*marked edema, eosinophilic infiltrates, granuloma formation√ thickened folds√ disappearance of Kerckring folds√ thumbprinting / saw-tooth appearance√ irregular luminal narrowing√ eosinophilic ascites (DDx: eosinophilic gastroenteritis, hypereosinophilic syndrome)*Cx:*ileus@Colonic anisakiasis (rare)*DDx:*colonic tumor

Notes:

ANORECTAL MALFORMATION

(1)Imperforate anus(2)Cloacal malformation(3)Cloacal exstrophy*Embryology:* during weeks 3 and 4 the dorsal part of the yolk sac folds are incorporated into embryo forming the *primitive hindgut* consisting of distal part of transverse + descending + sigmoid colon, rectum, superior portion of anal canal, epithelium of urinary bladder, and most of the urethra; at 4 weeks the transverse *rectovesical septum* descends caudally between allantois and hindgut dividing the *cloaca* into *urogenital sinus* ventrally + *anorectal canal* dorsally; by 7th week the rectovaginal septum fuses with cloacal membrane creating a *urogenital membrane* ventrally + anal membrane dorsally; perineum is formed by fusion of rectovesical septum + cloacal membrane; *anal membrane* ruptures by 9th week *In 48% associated with:* (part of VACTERL syndrome) (1)GU anomalies (20%):renal agenesis / ectopia, vesicoureteral reflux, obstruction, hypospadia (3.1%); M > F; (2)Lumbosacral segmentation anomalies (30%): dysplasia, agenesis, hemivertebrae(3)GI anomalies (11%):esophageal atresia ± tracheoesophageal fistula (4%), duodenal atresia / stenosis (4)Cardiovascular anomalies (8%)(5)Abdominal wall (2%)(6)Cleft lip-cleft palate (1.6%)(7)Down syndrome (1.5%)(8)Meningomyelocele (0.5%) + occult myelodysplasia(9)Others (8%)
Caudal regression syndrome: anorectal atresia, sacral agenesis, renal agenesis / dysplasia, lower limb hypoplasia, sirenomelia

Notes:

ANTRAL MUCOSAL DIAPHRAGM

= antral web *Age range:*3 months to 80 years*Associated with:*gastric ulcer (30-50%) ● symptomatic if opening <1 cmLocation:usually 1.5 cm from pylorus (range 0-7 cm)√ constant symmetric band of 2-3 mm thickness traversing the antrum perpendicular to long axis of stomach√ "double bulb" appearance (in profile)√ concentric / eccentric orifice√ normal peristaltic activity

Notes:

APPENDICITIS

*Incidence:*7-12% in Western world population*Etiology:*obstruction of appendiceal lumen by lymphoid hyperplasia (60%), fecolith (33%), foreign bodies (4%), stricture, tumor, parasite; Crohn disease (in 25%)*Peak age:*2nd-3rd decade ● fever (56%) ● nausea + vomiting (40%) ● RLQ pain over appendix = McBurney sign (72%) ● leukocytosis (88%)False positive:7-45% (average 20%)False negative:7-33% (average 20%)◊32-45% rate of misdiagnosis in women between ages 20-40!Atypical location:within pelvis (30%), extraperitoneal (5%)Abdominal plain film (abnormalities seen in <50%):◊Plain-film findings become more distinctive after perforation, while clinical findings subside / simulate other diseases!√ usually laminated calcified appendicolith in RLQ (in 7-15%)◊Appendicolith + abdominal pain = 90% probability of acute appendicitis!◊Appendicolith in acute appendicitis means a high probability for gangrene / perforation!√ "cecal ileus" = gas-fluid level in cecum in gangrene(= local paralysis)√ thickening of cecal wall√ small bowel obstruction pattern = small bowel dilatation with air-fluid levels (in 43% of perforations)√ colon cutoff sign = amputation of gas at the hepatic flexure (in 20% of perforations) due to spastic ascending colon√ water-density mass + paucity / absence of intestinal gas in RLQ (in 24% of perforations)√ extraluminal gas (in 33% of perforations)√ gas loculation√ mottled bacteriogenic gas√ pneumoperitoneum (rare)√ focal increase in thickness of lateral abdominal wall in 32% (= edema between properitoneal fat line + cecum)√ loss of properitoneal fat line√ loss of pelvic fat planes around the bladder / right obturator (= fluid / pus in cul-de-sac)√ loss of definition of right inferior hepatic outline (= free peritoneal fluid)√ distortion of psoas margin + flank stripesBE / UGI (accuracy 50-84%): √ failure to fill appendix with barium (normal finding in up to 35%)√ indentation along medial wall of cecum (= edema at base of appendix / matted omentum / periappendiceal abscess)US (77-94% sensitive, 90% specific, 78-96% accurate; nondiagnostic study in 4% due to inadequate compression of RLQ); useful in ovulating women (false-negative appendectomy rate in males 15%, in females 35%): √ visualization of noncompressible appendix as a blind-ending tubular aperistaltic structure (seen only in 2% of normal adults, but in 50% of normal children) √ target appearance of ≥6 mm in total diameter on cross section (81%) / mural wall thickness ≥2 mm√ diffuse hypoechogenicity (associated with higher frequency of perforation)√ lumen may be distended with anechoic / hyperechoic material√ loss of wall layers√ visualization of appendicolith (6%)√ localized periappendiceal fluid collection√ prominent hyperechoic mesoappendix / pericecal fatColor Doppler US: √ increased conspicuity (= increase in size + number) of vessels in and around the appendix = hyperemia√ decreased resistance of arterial waveforms√ continuous / pulsatile venous flowCT (87-98% sensitive, 83-97% specific, 93% accurate): √ abnormal appendix√ distended lumen√ circumferentially thickened ± enhancing wall√ appendicolith = homogeneous / ringlike calcification (25%)√ periappendicular inflammation√ linear streaky densities in periappendicular / pericecal / mesenteric / pelvic fat√ phlegmon = pericecal soft-tissue mass√ pericecal / mesenteric / pelvic abscess = poorly encapsulated single / multiple fluid collection with air / extravasated contrast material√ focal cecal apical thickening (80%)√ "arrowhead" sign = funnel of contrast medium in cecum centering about occluded orifice of appendix*Cx:*perforation (13-30%)*DDx:*colitis, diverticulitis, epiploic appendagitis, small bowel obstruction, infectious enteritis, duodenal ulcer, pancreatitis, intussusception, Crohn disease, mesenteric lymphadenitis, ovarian torsion, pelvic inflammatory disease*Rx:*finding of appendicolith is sufficient evidence to perform prophylactic appendectomy in asymptomatic patients (50% have perforation / abscess formation at surgery)

Notes:

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ASCARIASIS
=most common parasitic infection in world; cosmopolitan occurrence; endemic along Gulf Coast, Ozark Mountains, Nigeria, Southeast Asia*Organism:*Ascaris lumbricoides = roundworm parasite, 15-35 cm in length; production of 200,000 eggs daily*Cycle*:infection by contaminated soil, eggs hatch in duodenum, larvae penetrate into venules / lymphatics, carried to lungs, migrate to alveoli and up the bronchial tree, swallowed, maturation in jejunum within 2.5 months*Age:*children age 1-10 years ● colic ● eosinophilia ● appendicitis ● hematemesis / pneumonitis ● jaundice (if bile ducts infested)Location:jejunum > ileum (99%), duodenum, stomach, CBD, pancreatic duct√ 15- to 35-cm-long tubular filling defects√ barium-filled enteric canal outlined within Ascaris √ whirled appearance, occasionally in coiled clusters ("bolus of worms")*Cx:*(1)Perforation of bowel(2)Mechanical obstruction

Notes:

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BANNAYAN-RILEY-RUVALCABA SYNDROME

=RUVALCABA-MYHRE-SMITH SYNDROME*Cause:*autosomal dominant transmission ● pigmented genital lesions√ hamartomatous intestinal polyps (in 45%): usually in distal ileum + colon√ macrocephaly√ subcutaneous and visceral lipomas + hemangiomas

Notes:

BARRETT ESOPHAGUS

=BARRETT SYNDROME=replacement of stratified squamous epithelium by metaplastic columnar epithelium (Barrett epithelium) containing goblet cells*Cause:*chronic gastroesophageal reflux with epithelial injury from esophagitis*Contributing factors:* genetic influence, reduced LES pressure, transient LES relaxation, hiatal hernia, delayed acid clearance, reduced acid sensitivity, duodenogastroesophageal reflux, alcohol, tobacco, chemotherapy, scleroderma (37%), S/P repair of esophageal atresia / esophagogastric resection / Heller esophagomyotomy *Histo:*(1)specialized columnar epithelium (proximal)(2)junctional-type epithelium (distal to above)(3)fundic-type epithelium (most distally)*Incidence:* in general 0.3-4%; 7-10-20% of patients with symptoms of reflux*Associated with:*moderate + severe esophagitis (94%), no / mild esophagitis (6%)*Age:*0-15 years and 40-88 years (mean of 55 years); M > F; mainly among Whites ● dysphagia (due to esophageal stricture) ● heartburn, substernal chest pain, regurgitation ● low-grade upper intestinal bleeding ● asymptomatic Location:middle to lower esophagusN.B.:the squamocolumnar junction does not coincide with the GE junction, is irregular and lies >2-3 cm orad from the gastroesophageal junctionDistribution:circumferential / focal √ several-cm-long stricture (71%) in midesophagus (40%) or lower esophagus (60%; DDx: peptic stricture without Barrett esophagus)√ large deep wide-mouthed peptic ulcer (= Barrett ulcer) at upwardly displaced squamocolumnar junction / within columnar epithelium√ fine reticular mucosal pattern (3-30%) located distally from stricture (DDx: gastroesophageal reflux, monilial + viral esophagitis, superficial spreading carcinoma)√ thickened irregular mucosal folds (28-86%)√ fine granular mucosal pattern (DDx: reflux esophagitis, acanthosis, leukoplakia, superficial spreading carcinoma, moniliasis / herpes simplex / CMV esophagitis)√ gastroesophageal reflux (45-63%)√ distal esophageal widening (34-66%; due to abnormal motility)√ hiatal hernia (75-94%)√ uptake of Tc-99m pertechnetate by columnar epithelium *Dx:*velvety pinkish red appearance of gastric-type mucosa extending from gastric mucosa into distal esophagus (endoscopy with biopsy)*Cx:*1.Ulceration ± penetration into mediastinum2.Stricture3.Adenocarcinoma (0-10-46%;) 40-fold higher risk than general population√ plaquelike / focal irregularity / nodularity / sessile polyps*Rx:*(1)stop smoking, avoid bedtime snacks + foods that lower LES pressure, lose excess weight(2)suppress gastric acidity: antacids, H_2-receptor antagonists (cimetidine, ranitidine, famotidine), H^+K^+-adenosintriphosphatase inhibitor (omeprazole)(3)improve LES pressure: metoclopramide, bethanechol(4)esophageal resection in high-grade dysplasia

Notes:

BEHÇET SYNDROME
=uncommon chronic multisystem inflammatory disorder of unknown etiology with relapsing course characterized by mucocutaneous-ocular symptoms as a triad of aphthous stomatitis, genital ulcers, ocular inflammation*Age at onset:*3rd decade; M:F = 2:1Major criteria:buccal + genital ulceration, ocular inflammation, skin lesionsMinor criteria:thrombophlebitis, GI + CNS lesions, arthritis, family history ● abdominal pain + diarrhea (50%)@ Mucocutaneous:aphthous stomatitis, papules, pustules, vesicles, folliculitis, erythema nodosum-like lesions@ Genital:ulcers on penis + scrotum / vulva + vagina@ Ocular:relapsing iridocyclitis, hypopyon, choroiditis, papillitis, retinal vasculitis@ Articular:mild nondestructive arthritis@ Vascular:migratory thrombophlebitis@ CNS:chronic meningoencephalitis@ Esophagus:ulceration, stenosis, perforation@ Small bowel:ulceration, perforation@ Colon:multiple discrete deep ulcers in normal mucosa (DDx: granulomatous / ulcerative colitis)

Intestinal Behçet Disease

Notes:

Intestinal Behçet Disease =presence of intestinal ulcers*Incidence:*<1%Location:terminal ileum, cecum√ deep round ulcers similar in appearance to peptic ulcers of stomach / duodenum√ multiple shallow / longitudinal / aphthoid ulcers*Cx:*panperitonitis with high mortality due to tendency for perforation at multiple sites*DDx:*Reiter syndrome, Steven-Johnson syndrome, SLE, ulcerative colitis, ankylosing spondylitis

Notes:

BEZOAR
=persistent concretions of foreign matter composed of accumulated ingested material in intestines (from Persian word padzahr = antidote, counterpoison)*Incidence:*0.4% (large endoscopic series)*Etiology:*material unable to exit stomach because of large size, indigestibility, gastric outlet obstruction, poor gastric motility (diabetes, mixed connective tissue disease, myotonic dystrophy, hypothyroidism)*Predisposition:* previous gastric surgery (vagotomy, pyloroplasty, antrectomy, partial gastrectomy), inadequate chewing, missing teeth, dentures, massive overindulgence of food with high fiber contents ● anorexia, bloating, early satiety / may be asymptomatic(a)**Phytobezoar** (55% of all bezoars):=poorly digested fibers, skin + seeds of fruits and vegetables usually forming in stomach, may become impacted in small bowel ● history of recent ingestion of pulpy foodsFood:oranges, persimmons (most common, unripe persimmons contain the tannin shibuol that forms a gluelike coagulum after contact with dilute acid) Site of impaction:stomach, jejunum, ileum√ intraluminal filling defect without constant site of attachment to bowel wall√ interstices filled with barium√ coiled-spring appearance (rare)√ partial / complete obstruction*Cx:*decubitus ulceration + pressure necrosis of bowel wall, perforation, peritonitis*DDx:*lobulated / villous adenoma, leiomyosarcoma, metastatic melanoma, intussusception(b)**Trichobezoar** (hair):80% are < age 30, almost exclusively in females; *Associated with:*gastric ulcer in 24-70%

Notes:

Hemoperitoneum ATTENUATION VALUES OF BLOOD during IV contrast administration and assuming an initially normal hematocrit without significant dilution from intraperitoneal fluid (ascites, urine, succus, lavage fluid) -serum (after hematocrit effect)0-20 HU-fresh unclotted blood30- 45 HU-clotted blood60-100 HU-active arterial extravasation>180 HULocation:paracolic gutters, pelvis√ "sentinel clot" sign = the highest attenuation value of blood clot marks the anatomic site of visceral injury√ high-density active arterial extravasation always surrounded by lower-density hematoma(DDx: extravasated oral contrast is not surrounded by lower-density material)

Notes:

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Hypovolemia √ "collapsed cava" sign = persistent flattening of IVC (due to decreased venous return)N.B.: abort CT examination as shock is imminent!√ small hypodense spleen (decreased enhancement)√ small aorta + mesenteric arteries (due to intense vasoconstriction)√ shock nephrogram = lack of renal contrast excretion√ "shock bowel" = generalized thickening of small bowel folds + increased enhancement + luminal fluid dilatation (due to vasoconstriction of mesenteric vessels)√ marked enhancement of adrenal gland

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Notes:

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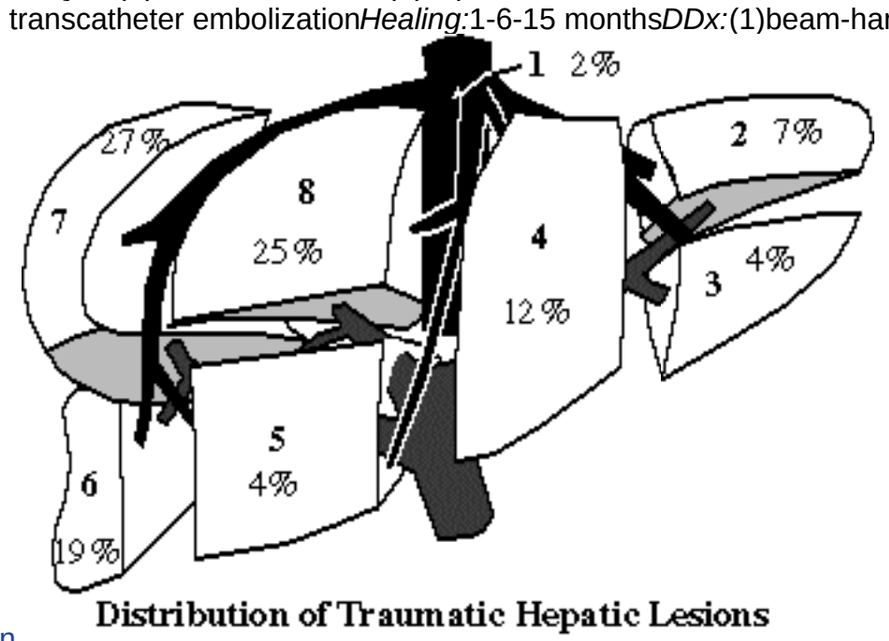
Blunt Trauma To Spleen ◊The spleen is the most frequently injured solid parenchymal organ within the abdomen!*Cause:*blunt trauma (most frequent)*Associated with:*rib fractures (in 40%), left renal injury◊20% of patients with left rib fractures have a splenic injury!◊25% of patients with left renal injury have a splenic injury!CECT (95% accuracy): √ mottled parenchymal enhancement = contusion√ hypoattenuating hematoma complete separation of splenic fragments (= fracture)√ crescentic region of low attenuation compressing normal parenchyma = subcapsular hematoma√ round hypodense inhomogeneous region ± hyperdense clot = intrasplenic hematoma√ hypoattenuating line connecting opposing visceral surfaces + perisplenic fluid = splenic laceration√ multiple lacerations = "shattered spleen"√ high-attenuation area = contrast extravasation / pseudoaneurysm√ hemoperitoneum (= disruption of splenic capsule)*Sequelae:*splenic pseudocyst (20-30 HU)*Cx:*delayed rupture up to 10 days later*Rx:*up to 91% of stable patients can be treated conservatively with observation; transcatheter embolization*DDx:*(1)Normal lobulation / splenic cleft (smoothly contoured, medially located)(2)Adjacent unopacified jejunum simulating splenic tissue(3)Early differential enhancement of red and white pulp (scan obtained within 20-50 sec)(4)Perisplenic fluid from ascites / urine / succus / bile / lavage

Notes:

Blunt Trauma To Liver (20%)
◊Second most frequently injured intra-abdominal viscus*Associated with:*splenic injury in 45%Location:R > L lobeSite:perivascular, paralleling right + middle hepatic arteries + posterior branches of right portal vein, avulsion of right hepatic vein from IVC (13%)◊Left lobe injury more often associated with damage to duodenum, pancreas, transverse colonCECT: √ hypoattenuating hematoma√ lenticular configuration (= subcapsular hematoma) usually resolving within 6-8 weeks√ irregular linear branching / round regions of low attenuation = laceration√ focal / diffuse periportal tracking (in up to 22%) due to dissecting hemorrhage / bile / dilated periportal lymphatics (secondary to elevated central venous pressure / injury to lymphatics)√ alteration in distribution of vessels + ducts√ hypodense wedge extending to liver surface = focal hepatic devascularization√ focal hyperdense (80-350 HU) area = active hemorrhage / pseudoaneurysm√ hemoperitoneum (inability of liver veins to contract)√ intrahepatic / subcapsular gas (usually due to necrosis)*Cx:*in up to 20%(1)delayed rupture (rare)(2)hemobilia(3)arteriovenous fistula / pseudoaneurysm(4)biloma ± infection(5)superinfection of hematoma / devascularized hepatic parenchyma*Rx:*conservative treatment in up to 80% in adults + 97% in children; transcatheter embolization*Healing:*1-6-15 months*DDx:*(1)beam-hardening artifact from adjacent ribs / from air-contrast level in stomach(2)Focal fatty infiltration



Distribution of Traumatic Hepatic Lesions

Notes:

Blunt Trauma To Gallbladder (2%)
*Associated with:*injury to liver , duodenum√ pericholecystic fluid (extraperitoneal location of GB)√ free intraperitoneal fluidCECT: √ blurred contour of GB√ focal thickening / discontinuity of GB wall√ intraluminal enhancing mucosal flap√ hyperattenuating blood within GB lumen√ mass effect on adjacent duodenum√ collapsed GB = GB rupture√ focal periportal tracking = GB ruptureUS: √ focal hypoechoic thickening√ echogenic mass within GB lumen

Notes:

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Blunt Trauma To GI Tract (5%)

Location:jejunum distal to ligament of Treitz > duodenum > ascending colon at ileocecal valve > descending colonCECT (88-92% sensitive): √ hypodense free fluid (85%), particularly in interloop location due to perforation√ focal bowel wall thickening > 3 mm = intramural hematoma (75%) ± intestinal obstruction√ focal discontinuity of bowel wall√ sentinel clot sign adjacent to bowel√ streaky hyperattenuating mesentery√ mesenteric hematoma (39%)√ hyperdense contrast enhancement of injured bowel wall = delayed venous transit time (20%)√ pneumoperitoneum (15-32%)√ extravasation of oral contrast material + gasN.B.:clinical signs + symptoms may be delayed for 24 hours (increasing mortality to 65%)

Notes:

Blunt Trauma To Pancreas (3%)
*Mechanism:*compression against vertebral column with shear across pancreatic neck*Associated with:*injury to liver, duodenum*Classification:* Iminor contusion / hematoma, capsule + major duct intactIIparenchymal injury without major duct injuryIIImajor ductal injuryIVsevere crush injuryLocation:junction of body + tail√ posttraumatic pancreatitis√ edema / fluid in peripancreatic fat√ focal / diffuse pancreatic enlargement√ irregularity of pancreatic contour√ area of low-attenuation laceration (actual site of laceration difficult to visualize)√ fluid around superior mesenteric artery√ fluid in transverse mesocolon / lesser sac√ fluid between pancreas and splenic vein√ thickening of anterior pararenal fasciaN.B.:24-48 hours delayed scans uncover findings not present earlier*Rx:*I + IIconservative management;III + IVneed surgery within 24 hours*Cx:*recurrent pancreatitis, pseudocyst, pseudoaneurysm, fistula, abscess (attendant mortality of 20%)

Notes:

Blunt Trauma To Kidney *Incidence:*10% of injuries in emergency room*Cause:*motor vehicle accident, contact sports, falls, fights, assaults*Mechanism:*direct blow (>80%) often lacerated by lower ribs, acceleration-deceleration (renal artery tear)*Associated with:*other organ injury in 20% ● >95% hematuria◊25% of patients with gross hematuria have significant injuries!◊24% of patients with renal pedicle injury have no hematuria!◊Only 1-2% with microhematuria (<35 RBCs per high-power field) have a severe renal injury!*Classification:* Icontusion + corticomedullary laceration (up to 85%)IIdeep laceration generally communicating with collecting system (10%)IIIcatastrophic injury: shattered kidney, renal artery pedicle injury (5%)IVUPJ avulsion / laceration (rare)Location:simultaneous upper + lower GU tract injury in <5%√ focal patchy areas of decreased enhancement / striated nephrogram = contusion√ irregular linear hypodense parenchymal areas = renal laceration√ laceration connecting two cortical surfaces = fracture√ multiple separated renal fragments ± perfusion = shattered kidney√ superficial crescentic hypodense area compressing adjacent parenchyma = subcapsular hematoma◊Subcapsular / perinephric hematoma usually proportional to extent of injury√ wedge-shaped perfusion defect = segmental arterial injury√ diffuse nonperfusion of kidney = devascularized kidney√ persistent nephrogram on delayed scans = renal vein thrombosisN.B.:Delayed images to check for urine leak!*Rx:*1.Blunt trauma I:expectant2.Blunt trauma II:controversial3.Blunt trauma III + IV:surgery4.Penetrating injury (stab wound, gunshot wound): surgery depending on location

Notes:

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Blunt Trauma To Ureteropelvic junction (rare)
=laceration (60%) / avulsion of ureter at UPJ*Mechanism:*tension on renal pedicle by sudden deceleration*Age:*usually young boys*Associated with:*fracture of transverse process (30%) ● gross / microscopic hematuria (53-60%)√ massive extravasation of contrast material medially in the region of UPJ√ nonfilling of affected ureter (with avulsion)√ ± circumferential perinephric urinoma

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Notes:

Blunt Trauma To Bladder *Associated with:*pelvic fracture in 70%*Indications for urethrogram:* ● blood at urethral meatus ● "floating" prostate ● inability to pass Foley catheter√ symphysis diastasis*CT cystogram:* √ focal thickening of bladder wall = contusion√ contrast extravasation = *see BLADDER RUPTURE*

Notes:

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BOERHAAVE SYNDROME
=complete transmural disruption of esophageal wall with extrusion of gastric content into mediastinum / pleural space secondary to food bolus impaction ● forceful vomiting with sudden onset of pain (substernal, left chest, in neck, pleuritic, abdominal) ● dyspnea ● NO hematemesis (blood escapes outside esophageal lumen)√ rent of 2-5 cm in length, 2-3 cm above GE junction, predominantly on left posterolateral wall√ pleural effusion on left >> right side / hydropneumothorax√ pneumomediastinum (single most important plain-film finding), pneumopericardium, subcutaneous air√ "V-sign of Naclerio" = localized mediastinal emphysema with air between lower thoracic aorta + diaphragm√ mediastinal widening√ air-fluid level within mediastinum√ extravasation of contrast medium into mediastinum / pleura

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Notes:

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BRUNNER GLAND HYPERPLASIA
*Etiology:*hyperplasia secondary to hyperacidity*Physiology:*secrete a clear viscous alkaline substance into crypts of LieberkühnMORPHOLOGIC TYPES: 1.Diffuse nodular hyperplasia2.Circumscribed nodular hyperplasia:in suprapapillary portion 3.Single adenomatous hyperplastic polyp:in duodenal bulb Location:duodenal glands begin in vicinity of pylorus extending distally within proximal 2/3 of duodenum√ multiple nodular filling defects (usually limited to 1st portion of duodenum√ "cobblestone appearance" (most common finding)√ occasionally single large mass ± central ulceration

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Notes:

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BURKITT LYMPHOMA
=most common type of non-Hodgkin lymphoma in children; initially described in Africa*Etiology:*tumor from undifferentiated B-cell-derived lymphocytes; associated with Epstein-Barr virus*Age:*children + young adults*Path:*resemblance to Hodgkin disease*Histo:*characteristic "starry sky" patternLocation:mandible (first), maxilla; multifocal (10%) ● jaw mass ● abdominal mass ● paraplegia ● NO peripheral leukemia√ usually intra-abdominal extranodal involvement with sparing of spleen A.ENDEMIC FORM OF BURKITT LYMPHOMAendemic in areas with malaria: tropical Africa, New Guinea 50% of all childhood cancers in central Africa *Age:*6-8 years@Mandible / maxilla√ grossly destructive lesion, spicules of bone growing at right angles√ large soft-tissue mass@ Other skeleton √ reminiscent of Ewing tumor / reticulum cell sarcoma√ lamellated periosteal reaction around major long bones B.NONENDEMIC FORM OF BURKITT LYMPHOMA*Age:*10-12 yearsLocation:abdominal involvement (69%): tumors of small bowel (terminal ileum), mesentery, retroperitoneum, ovary, uterus, salivary glands, thyroid, kidneys, bone marrow√ well-defined sharply marginated homogeneous tumors (75%)√ ascites (13%)√ renal masses / enlargement (5%)√ hydronephrosis (28%)√ conspicuous absence of lymph node disease√ pleural effusion (most common chest abnormality)*Rx:*dramatic response to chemotherapy*Prognosis:*long-term survival in 50%

Notes:

CARCINOID

=most common primary tumor of small bowel + appendix (>95% of all carcinoids); belongs to APUDomas; M:F = 2:1*Path:*firm yellow submucosal nodule arising from argentophil Kulchitsky cells in the crypts of Lieberkühn (= argentaffinoma); invasion into mesentery incites an intense fibrotic reaction*Histo:*low-grade malignancy = resemble adenocarcinomas but do not have their aggressive behavior; malignant through invasion of muscularis*Biochemistry:* tumor elaborates (1) ACTH (2) histamine (3) bradykinin (4) kallikrein (5) serotonin = 5-hydroxytryptamine (from tryptophan over 5-hydroxytryptophan), which is metabolized in liver by monamine oxidase into 5-hydroxyindole acetic acid (5-HIAA) and excreted in urine; 5-hydroxytryptophan is destroyed in pulmonary circulation ● asymptomatic (66%) ● pain / obstruction (19%) ● weight loss (16%) ● palpable mass (14%) ● **Carcinoid syndrome** (7% of small bowel carcinoids)caused by excess serotonin levels, requires that serotonin metabolism (to 5-HIAA in liver) is bypassed (a)with liver metastases(b)with primary pulmonary / ovarian carcinoids ● recurrent diarrhea (70%) ● right-sided endocardial fibroelastosis (35%) resulting in tricuspid regurgitation + pulmonary valve stenosis + right heart failure ● attacks precipitated by ingestion of food / alcohol ● asthmatic wheezing from bronchospasm (15%) ● desquamative skin lesions (5%) ● nausea & vomiting, fever ● hypotension ● cutaneous flushing (rare)*Metastases:* to lymph nodes, liver (in 90% of patients with carcinoid syndrome), lung, bone (osteoblastic) (a)incidence versus tumor sizetumor of<1 cm(in 75%)metastasizes in2%tumor of1-2 cm(in 20%)metastasizes in50%tumor of>2 cm(in 5%)metastasizes in85%(b)incidence versus locationtumor in ileum(in 28%)metastasizes in35%tumor in appendix(in 46%)metastasizes in3%tumor in rectum(in 17%)metastasizes in1%Liver metastases seen: best /(only) on:(a)NECT35%(3%)(b)CECT in HAP35%(14%)(c)CECT in PVP30%(3%)HAP = hepatic arterial-dominant phase of triple phase CT PVP = portal venous-dominant phase of triple phase CT RULE OF 1/3:◊1/3 occur in small bowel◊1/3 have metastases◊1/3 are multiple◊1/3 have a second malignancyLocation:between gastric cardia and anus@Appendix (30-45%)commonly benign; surgical incidence of 0.03-0.7% Site:tip (70%), middle (20%), base (10%) of appendix@Small bowel (25-35%)Location:ileum (91%); jejunum (7%), duodenum (2%); multiple in 15-35%@Rectum (10-15%): metastasize in 10%@Colon (5%): ascending colon, often malignant@Stomach (rare)@Other organs (5%): bronchus, thyroid, pancreas, biliary tract, teratomas (ovarian, sacrococcygeal, testicular)@may be multicentricUGI: √ small smooth submucosal mass (usually <2 cm) impinging eccentrically on lumen√ angulation + kinking of loops leading to obstruction (DIAGNOSTIC)√ spiculated / tethered appearance of mucosal folds (desmoplastic reaction)√ separation of loops due to large mesenteric metastasesCT: √ stellate radiating pattern + beading of mesenteric neurovascular bundles (desmoplastic reaction)√ retraction + shortening of mesentery√ displacement + kinking + separation of adjacent bowel loops√ segmental thickening of adjacent bowel loops (encasement of mesenteric vessels leads to chronic ischemia)√ calcification of mesenteric mass√ low-density lymphadenopathy (due to necrosis)√ liver metastases may become isodense following slow contrast infusionAngio: √ thickening + foreshortening of mesenteric vessels√ kinking of small- and medium-sized vessels with stellate configuration√ venous occlusion / mesenteric varices√ encasement of medium-sized vessels√ simulated hypervascularity secondary to fibrotic retraction of mesenteric vesselsNUC (I-123 MIBG imaging): √ uptake in 44-63% (higher frequency of radiotracer uptake in midgut carcinoids + with elevated serotonin levels)US: √ persistent fluid-distended appendix without typical signs of appendicitis*Cx:*second primary malignant neoplasm in other location (36% at necropsy)*Rx:*Somatostatin / SMS 201-995*DDx:*oat-cell carcinoma, pancreatic carcinoma, medullary thyroid carcinoma, retractile mesenteritis, desmoplastic carcinoma / lymphoma

Notes:

CATHARTIC COLON

=prolonged use of stimulant-irritant cathartics (>15 years) resulting in neuromuscular incoordination from chronically increased muscular activity + tonus*Agents:*castor oil, senna, phenolphthalein, cascara, podophyllum, aloinLocation:involvement of colon proximal to splenic flexure√ effaced mucosa with flattened smooth surface√ diminished / absent haustrations√ "pseudostrictures" = smoothly tapered areas of narrowing are typical (sustained tonus of circular muscles)√ poor evacuation of barium√ flattened + gaping ileocecal valve√ shortened but distensible ascending colon*DDx:*"burned-out" ulcerative colitis with right-sided predominance (very similar)

Notes:

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CHAGAS DISEASE
=damage of ganglion cells by neurotoxin liberated from protozoa Trypanosoma cruzi resulting in aperistalsis of GI tract + dilatation Endemic to Central + South America (esp. eastern Brazil) *Histo:*decreased number of cells in medullary dorsal motor nucleus + Wallerian degeneration of vagus + decrease / loss of argyrophilic cells in myenteric plexus of Auerbach*Peak age:*30-50 years; M:F = 1:1 ● intermittent / persistent dysphagia ● odynophagia (= fear of swallowing) ● foul breath, regurgitation, aspiration ● Mecholyl test: abnormal response indicative of deficient innervation; 2.5-10 mg methacholine subcutaneously followed by severe tetanic nonperistaltic contraction 2-5 minutes after injection, commonly in distal half of esophagus, accompanied by severe pain@Dilatative cardiomyopathy (myocarditis)@Megacolon (bowels move at intervals of 8 days to 5 months)*Cx:*impacted feces, sigmoid volvulus@Esophagus: changes as in achalasia

Notes:

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CHALASIA
=continuously relaxed sphincter with free reflux in the absence of a sliding hernia*Etiology:*elevated submerged segment*Causes:*(1)Delayed development of esophagogastric region in newborns(2)Scleroderma, Raynaud disease(3)S/P forceful dilatation / myotomy for achalasia√ free / easily induced reflux

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CHRONIC IDIOPATHIC INTESTINAL PSEUDOOBSTRUCTION
=nonpropulsive intestine characterized by impaired response to intestinal dilatation without definable cause; ? autosomal dominant*Age:*all ages, M:F = 1:1 ● recurrent attacks of abdominal distension, periumbilical pain, nausea, vomiting, constipation√ mild to marked gaseous distension of duodenum + proximal small bowel√ esophageal dilation + hypoperistalsis (lower third)√ excessive duodenal dilation (DDx: megaduodenum, superior mesenteric artery syndrome)√ ligament of Treitz may be placed lower than usual√ delayed transit of barium through affected segments√ disordered motor activity (fluoroscopy)

Notes:

COLITIS CYSTICA PROFUNDA
=rare benign condition characterized by submucosal mucus-containing cysts lined by normal colonic epithelium*Etiology:*probably related to chronic inflammation*Age:*primarily disease of young adults ● brief periods of bright red rectal bleeding ● mucous / bloody discharge ● intermittent diarrheaLocation:(a)localized to rectum (most commonly) / sigmoid(b)generalized colonic process (less common)√ nodular polypoid / cauliflower-like lesions <2 cm in size, containing no gas√ spiculations mimicking ulcers (barium-filled clefts between nodules)*DDx:*pneumatosis (rarely affects rectum)

Notes:

COLORECTAL CARCINOMA

Most common cancer of GI tract; 2nd most frequently diagnosed malignancy; 2nd most common cause of death from malignancy after lung cancer (in men) + breast cancer (in women) *Predisposed:*socioeconomic status; diet low in fiber + high in fat and animal protein; obesity (in men); asbestos workerSyndromes (6% of colorectal carcinomas): familial adenomatous polyposis syndrome (= familial polyposis, Gardner syndrome, Turcot syndrome), Peutz-Jeghers syndrome, hereditary nonpolyposis colon cancer syndrome *Risk factors:* 1.Colonic adenoma-malignancy in 5% of tubular adenomas-malignancy in 40% of villous adenomasProof of adenoma-carcinoma sequence: (a)frequent coexistence of adenoma + carcinoma(b)similar distribution within colon(c)consistent proportional prevalence in population having varied magnitudes of colon cancer risk(d)increased frequency of carcinoma in patients with adenomas(e)reduction of cancer incidence following endoscopic removal of polyps(f)all patients with familial adenomatous polyposis syndrome develop colon carcinoma if colon not removed(g)similarity of DNA + chromosomal constitution93% of colorectal carcinomas arise from adenomatous polypA patient with one adenoma has a 9% chance of having a colorectal carcinoma in next 15 years!It takes about 7 years for a 1-cm adenoma to become an invasive cancer!5% of adenomas 5 mm in size develop into invasive cancers (5 mm is considered critical mass of intraepithelial neoplasia)!2.Dysplasia of colon within flat mucosa3.Family history of benign / malignant colorectal tumors, 3-5 x risk in first-degree relatives4.Chronic ulcerative colitis (3-5% incidence; cumulative incidence of 26% after 25 years of colitic symptoms)5.Prominent lymphoid follicular pattern6.History of endometrial / breast cancer7.Crohn disease (particularly in bypassed loops / in vicinity of chronic fistula)8.Pelvic irradiation9.Ureterosigmoidostomy *Screening recommendations:* as / more effective than mammographic screening (a)for persons >50 years of age: annual fecal occult-blood test + sigmoidoscopy / BE every 3 to 5 years(b)for first-degree relatives of patients with colon cancer screening should start at age 40*Incidence:*15% of all newly diagnosed cancers; 13% of all cancer deaths; 156,000 new cases/year with 61,300 deaths; 6.5% lifetime probability of any White person to develop colorectal cancer; 3/100,000 in 30- to 34-year-olds; 532/100,000 for >85-year-olds *Age:*median age of 71 years for colon cancer; median age of 69 years for rectal cancer; M:F = 3:2*Histo:*(1)Adenocarcinoma with varied degrees of differentiation(2)Mucinous carcinoma (uncommon)(3)Squamous cell carcinoma + adenoacanthoma (rare) *Staging (modified Dukes = Astler-Coller classification):* Alimited to mucosaBinvolvement of muscularis propria B_1extension into muscularis propriaB_2extension through muscularis propria into serosa / mesenteric fat (35%)Clymph node metastases (50%)C_1+ growth limited to bowel wallC_2+ growth extending into adipose tissueDdistant metastases *Staging (UICC-AJCC Colorectal Cancer Staging System):* StageGrouping5-year survival0TisN0M0>95%IT1N0M0T2N0M075-100%IIT3N0M0T4N0M050-75%IIIany TN1M0any TN2,3M030-50%IVany Tany NM1<10%Legend: Tiscarcinoma in situT1invasion of submucosaT2invasion of muscularis propriaT3invasion of subserosa / pericolic tissueT4invasion of other organsN11 to 3 pericolic LnnN2>4 pericolic LnnN3any Lnn along course of a vascular trunk Metastases (lymphatic / hematogenous venous): 1.liver (75%; 15-20% at time of surgery)2.retroperitoneal + mesenteric nodes (10-15%)3.adrenal (10-14%)4.lung (5-50%)5.ovary (3-8%)6.psoas muscle tumor deposit7.malignant ascites8.bone (5%)9.brain (5%)Because of absence of lymphatics in lamina propria colon cancer will not metastasize until it penetrates the muscularis mucosa! ● rectal bleeding, iron deficiency anemia ● change in caliber of stools ● obstruction (poor prognostic indicator) ● hydronephrosis (13%) ● positive fecal occult blood testing (2-6% positive-result rate ; 5-10% positive predictive value; fails to detect 30 -50% of colorectal carcinomas + up to 75% of adenomas): Hemoccult (hematein), Hemoquant (porphyrins), Haemselect (hemoglobin) ● progressive elevation of carcinoembryonic antigen (CEA) >10 µg/L indicative of recurrent / metastatic diseaseLocation:rectum (15-33 -41%), sigmoid (20-37%), descending colon (10-11%), transverse colon (12%), ascending colon (8-16%), cecum (8-10%); "aging gut" = number of right-sided lesions increasing with age Colonoscopy: cecum not visualized in 10-36%; fails to detect 12% of colonic polyps (10% in areas never reached by colonoscope) *Cx:*perforation in 0.2% (0.02% for BE); death in 1:5,000 (1:50,000 for BE) BE (sensitivities for polyps >1 cm: single contrast 77-94%, double contrast 82-98%; for polyps <1 cm: single contrast 18-72%, double contrast 61-83%): √ fungating polypoid carcinoma; ● chronic bleeding, intussusception√ annular ulcerating carcinoma = "applecore lesion";=annular constriction is a result of tumor growing along the lymphatic channels which parallel the circular muscle fibers of the inner layer of the muscularis propria; longitudinal growth is limited with abrupt transition to normal mucosa ● colonic obstruction√ "saddle lesion" = growth characteristics between polypoid mass + annular constricting lesion√ scirrhous carcinoma = rare variant of diffusely infiltrating adenocarcinoma (signet-ring type); often seen in ulcerative colitis=circumferential + longitudinal tumor spread within the loose submucosal tissue between muscularis mucosa + muscularis propria ● long-segment stricture similar to linitis plastica√ curvilinear / mottled calcifications (rare) are CHARACTERISTIC of mucinous adenocarcinoma CT: staging accuracy of 48-90%, for lymph node metastases of 25-73% CT staging (poor accuracy compared with modified Duke classification): Stage 1intramural polypoid massStage 2thickening of bowel wallStage 3slight invasion of surrounding tissuesStage 4massive invasion of surrounding tissue + adjacent organs / distant metastases√ low-density mass + low-density lymph nodes in mucinous adenocarcinoma (= >50% of tumor composed of extracellular mucin)√ psammomatous calcifications in mucinous adenocarcinoma√ signs of Lnn involvement: single lymph node >1 cm in diameter / cluster of ≥3 nodes <1 cm / node of any size within mesenteryMR (staging accuracy of 73%, 40% sensitivity for lymph node metastases) *Prognosis:* Survival rate of 40-50% overall in 5 years (unchanged over past 40 years); 80-90% with Duke A; 70% with Duke B; 33% with Duke C; 5% with Duke D Recurrence in 1/3 of patients: (a)local recurrence at line of anastomosis (60%) within 1 year after resection in 50%, within 2 years after resection in 70-80%(b)distant metastases (26%)(c)local recurrence + metastases (14%)Risk after detection of colon cancer: of5%for synchronous colon cancerof14%for synchronous cancer with "sentinel polyp" of35%for additional adenomatous polypof3%for metachronous colon cancerof4%for extracolonic malignancy*Cx:*(1)Obstruction (frequently in descending + sigmoid colon)(2)Perforation(3)Intussusception(4)Pneumatosis cystoides intestinalis(5)Pseudomyxoma peritonei (from low-grade adenocarcinoma of colon)*DDx:*(1)Prolapsing ileocecal valve (change on palpation)(2)Spasm (intact mucosa, released by propantheline bromide)(3)Diverticulitis

Lynch Syndrome Rectal Cancer

Notes:

Lynch Syndrome =HEREDITARY NONPOLYPOSIS COLORECTAL CANCER SYNDROME=families with high incidence of colorectal cancers + increased incidence of synchronous and metachronous colorectal cancersA.Lynch I=no associated extracolonic cancerB.Lynch II=associated with extracolonic malignancy: transitional cell carcinoma of ureter + renal pelvis, adenocarcinoma of endometrium, stomach, small bowel, pancreas, biliary tract, brain, hematologic malignancy, carcinoma of skin + larynx*Etiology:*autosomal dominant abnormality of chromosome 2 with defect in DNA replication-repair process(a)accelerated adenoma-carcinoma sequence(b)dysplasia in flat mucosa of colon*Prevalence:*5-10% of patients with colon cancer;5 times more common than familial adenomatous polyposis syndrome*Mean age:*45 yearsLocation:70% proximal to splenic flexure*Prognosis:*better stage for stage than in other cancers (5-year survival rate of 65% versus 44% in sporadic cases)*Surveillance:*colonoscopy every 1-2 years from ages 22-35 years

Notes:

Rectal Cancer *Incidence:*45,000 rectal cancers/year in United States*Pathologic staging of rectal cancer:* Astler-Coller/TNMDescription5-year survival AT1,N0,M0limited to submucosa80%B1T2,N0,M0limited to muscularis propria70%B2T3,N0,M0transmural extension60-65%C1T2,N1,M0nodes (+), into muscularis35-45%C2T3,N1,M0nodes (+), transmural25%T4invasion of adjacent organsDM1distant metastasis<25% *Risk of recurrence:* 5%for T110%for T233% for T1,N1 + T2N125%for T366% for T3N150%for T4*Staging accuracy:* (1)Digital rectal examination: 68-75-83%; limited to lesions within 10 cm of anal verge(2)CT: 48-72-92%, better for more extensive regional spread; 25-73% for lymph node involvement(3)MR: 74-84-93% with tendency for overstaging(4)Transrectal ultrasound: 64-77-94% with tendency for overstaging; limited to lesions <14 cm from anal verge + nonstenotic lesions; 50-83% sensitivity for lymph node involvementTransrectal US (81% accuracy): Normal layers: (a) hyperechoic interface of balloon + mucosa (b) hypoechoic mucosa + muscularis mucosa (c) hyperechoic submucosa (d) hypoechoic muscularis propria (e) hyperechoic serosa √ hypoechoic mass disrupting rectal wall√ no interruption of hyperechoic submucosa = tumor confined to mucosa + submucosa√ no interruption of hyperechoic serosa = tumor confined to rectal wall√ break in outermost hyperechoic layer = tumor penetrates into perirectal fat√ irregular serrated outer border of muscularis propria (pseudopodia through serosa)√ hypoechoic perirectal lymph nodes (= tumor involvement)

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COLONIC VOLVULUS

=most common form of volvulusA.VOLVULUS OF CECUM*Associated with:* malrotation + long mesentery*Age peak:*20-40 years; M > F√ "kidney-shaped" distended cecum, usually positioned in LUQ√ tapered end of barium column points toward torsionB.VOLVULUS OF SIGMOID=sigmoid twists on mesenteric axisUsually in elderly / psychiatrically disturbed Degree of torsion:360° (50%), 180° (35%), 540° (10%)√ greatly distended paralyzed loop with fluid-fluid levels, mainly on left side, extending toward diaphragm (erect film)√ "coffee-bean sign" = distinct midline crease corresponding to mesenteric root in largely gas-distended loop (supine)√ "bird-of-prey sign" = tapered hooklike end of barium columnCT: √ "whirl sign" = tightly torsioned mesentery formed by twisted afferent + efferent loop

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CONGENITAL INTESTINAL ATRESIA
*Incidence:*1:300 livebirths*Cause:*usually sporadic vascular accidents (primary / secondary to volvulus or gastroschisis)Location:jejunum + ileum (70%), duodenum (25%), colon (5%); may involve multiple sites√ "triple bubble sign" = intraluminal gas in stomach + duodenal bulb + proximal jejunum as pathognomonic sign for jejunal atresia√ bulbous bowel segment sign = dilated loop of bowel just proximal to site of atresia (due to prolonged impaction of intestinal contents) with curvilinear termination√ gasless lower abdomen (gut usually air-filled by 4 hours after birth)√ meconium peritonitis (6%)√ polyhydramnios (in 50% with duodenal / proximal jejunal atresia; rarely in ileal / colonic atresia)*Prognosis:*88% survival for isolated atresia

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Notes:

CRICOPHARYNGEAL ACHALASIA
=hypertrophy of cricopharyngeus muscle (= upper esophageal sphincter) with failure of complete relaxation*Etiology:* 1.Normal variant without symptoms: seen in 5-10% of adults2.Compensatory mechanism to gastroesophageal reflux3.Neuromuscular dysfunction of deglutition(a)primary neural disorders:brainstem disorder (bulbar poliomyelitis, syringomyelia, multiple sclerosis, amyotrophic lateral sclerosis); central / peripheral nerve palsy; cerebrovascular occlusive disease; Huntington chorea (b)primary muscle disorder:myotonic dystrophy; polymyositis; dermatomyositis; sarcoidosis; myopathies secondary to steroids / thyroid dysfunction; oculopharyngeal myopathy (c)myoneural junction disorder:myasthenia gravis; diphtheria; tetanus ● mostly asymptomatic ● dysphagia◊Cineradiography / videotape recording required for demonstration!√ distension of proximal esophagus + pharynx√ smoothly outlined shelf- / liplike projection posteriorly at level of cricoid (= pharyngoesophageal junction) = level of C5/6√ barium may overflow into larynx + trachea*Cx:*Zenker diverticula*Rx:*cricopharyngeal myotomy

Notes:

COWDEN DISEASE
=MULTIPLE HAMARTOMA SYNDROME=autosomal dominant disease with high penetrance characterized by multiple hamartomas + neoplasms of endodermal, ectodermal, mesodermal origin*Incidence:*160 cases reported*Age:*2nd decade@Mucocutaneous tumors ● facial papules ● oral papillomas (lips, gingiva, tongue) ● palmoplantar keratosis, acral keratosis@Breast lesions (in 50%):√ fibrocystic disease + fibroadenomas√ breast cancer (20-30%): often bilateral + ductal@GI tract√ multiple hamartomatous polyps (in 30-60%, commonly in rectosigmoid)@Thyroid abnormalities (in 60-70%):√ adenomas + goiter√ follicular thyroid adenocarcinoma (3-4%)@Genitourinary lesions@Skeletal abnormalities

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Notes:

CROHN DISEASE
=REGIONAL ENTERITIS = disease of unknown etiology with prolonged + unpredictable course characterized by discontinuous + asymmetric involvement of entire GI tract*Prevalence:*2-3:100,000 white adults*Path:*transmural inflammation (noncaseating granuloma with Langhans giant cells and epitheloid cells, edema, fibrosis); obstructive lymphedema + enlargement of submucosal lymphoid follicles; ulceration of mucosa overlying lymphoid follicles*Age:*onset between 15-30 years; M:F = 1:1 ● recurrent episodes of diarrhea ● colicky / steady abdominal pain ● low-grade fever ● weight loss, anorexia ● occult blood + anemia ● perianal abscess / fistula (40%) ● malabsorption (30%)*Associated with:* erythema nodosum, pyoderma gangrenosum INTESTINAL MANIFESTATIONS @Esophagus (rare)@ Stomach (1-2%) = granulomatous gastritis √ pseudo-post Billroth-I appearance√ "rams horn sign" = poorly distensible smooth tubular narrowed antrum + widened pylorus + narrow duodenal bulb√ aphthous ulcers (= pinpoint erosions)√ cobblestone appearance of mucosa√ antral-duodenal fistula@ Duodenum (4-10%) almost always associated with gastric involvement Location:duodenal bulb + proximal half of duodenum√ superficial erosions / aphthoid ulcers (early lesion)√ thickened duodenal folds@ Small bowel (80%) = regional enteritis terminal ileum (alone / in combination in 95%); jejunum / ileum (15-55%) √ thickening + slight nodularity of circular folds√ aphthous ulcers√ cobblestone mucosa / ulceration√ commonly associated with medial cecal defect@ Colon (22-55%) = granulomatous colitis particularly on right side with rectum + sigmoid frequently spared √ tiny 1- to 2-mm nodular filling defects (lymphoid follicular pattern)√ aphthous ulcers with "target / bulls-eye" appearance√ "transverse stripe sign" = 1-cm-long straight stripes representing contrast medium within deep grooves of coarse mucosal folds√ long fistulous tracts parallel to bowel lumen@Appendicitis (20%)@ Rectum (14-50%) √ deep / collarbutton ulcers√ rectal sinus tracts*Phases:* (a)Earliest changes√ nodular enlargement of lymphoid follicles √ blunting / flattening / distortion / straightening / thickening of valvulae conniventes (obstructive lymphedema, usually first seen in terminal ileum)√ aphthous ulcers = nodules with shallow central barium collection up to 5 mm in diameterLocation: duodenal bulb, second portion of duodenum, terminal ileum (b)Advanced nonstenotic phase√ skip lesions (90%) = discontinuous involvement with intervening normal areas√ cobblestone appearance = serpiginous longitudinal + transverse ulcers separated by areas of edema√ thick + blunted small bowel folds (inflammatory infiltration of lamina propria + submucosa)√ straightening + rigidity of small bowel loops with luminal narrowing (spasm + submucosal edema)√ separation + displacement of small bowel loops (from lymphedematous wall thickening / increase in mesenteric fat / enlarged mesenteric lymph nodes / perforation with abscess formation)√ pseudopolyps = islands of hyperplastic mucosa between denuded mucosa√ inflammatory polypoid masses√ sessile / pedunculated / filiform postinflammatory polyps√ diffuse mucosal granularity due to 0.5- to 1-mm round lucencies (= blunted + fused villi seen en face)√ pseudodiverticula = pseudosacculations = bulging area of normal wall opposite affected scarred wall on antimesenteric side(c)Stenotic phase√ "string sign" = strictures (in 21%, most frequently in terminal ileum) / marked narrowing of rigid loops√ normal proximal loops may be dilated with stasis ulcers + fecolithsCT: √ homogeneous density of thickened bowel wall (DDx: ulcerative colitis with inhomogeneous attenuation)√ "double halo configuration" (50%) = intestinal lumen surrounded by inner ring of low attenuation(= edematous mucosa) + outer ring of soft-tissue density (= thickened fibrotic muscularis + serosa) (DDx: radiation enteritis, ischemia, mesenteric venous thrombosis, acute pancreatitis) √ luminal narrowing + proximal dilatation√ skip areas of asymmetric bowel wall thickening of 10-20 mm in 82% (DDx: ulcerative colitis with a mean thickness of 8 mm)√ "creeping fat" = massive proliferation of mesenteric fat (40%) with mass effect separating small bowel loops√ mesenteric adenopathy (18%)√ abscess (DDx: postoperative blind loop)US: √ "pseudokidney" / target sign = thickening of bowel wall (22-65-89%) of 5-20 mm (DDx: ulcerative colitis)√ circumferential diffusely hypoechoic bowel wall with loss of normal layering (due to transmural edema, inflammation, fibrosis)√ rigid + noncompressible bowel segment with reduction / loss of peristalsis√ hyperemia of gut wall + adjacent fat on color Doppler√ inflammatory mass = phlegmon (14%), abscess (4%)√ distended fluid-filled loops (12%)√ hypoechoic fistulous tract *Prognosis:*recurrence rate of up to 39% after resection (commonly at the site of the new terminal ileum, most frequently during first 2 years after resection); mortality rate of 7% at 5 years, 12% at 10 years after 1st resection *Cx:*(1)Fistula (33%):(a)enterocolic:most frequently between ileum and cecum (b)enterocutaneous (8-21%):rectum-to-skin; rectum-to-vagina (c)perineal fistula + sinus tracts◊Crohn disease is 3rd most common cause of fistula / sinus tracts (*DDx:* iatrogenic [most common cause], diverticula [2nd most common cause])!(2)Intramural sinus tracts(3)Abscess (DDx: acute appendicitis)(4)Free perforation (1-2%)(5)Toxic megacolon(6)Small bowel obstruction (15%)(7)Hydronephrosis (from ureteric compression, generally on right side)(8)Adenocarcinoma in ileum / colon (particularly in bypassed loops / in vicinity of chronic fistula)◊4-20 x increased risk of colonic adeno-carcinoma compared with general population with a latency period of 25-30 years!(9)Lymphoma in large + small bowel*DDx:*(1)Yersinia (in terminal ileum, resolution within 3-4 months)(2)Tuberculosis (more severe involvement of cecum, pulmonary TB)(3)Actinomycosis, histoplasmosis, blastomycosis, anisakiasis(4)Segmental infarction (acute onset, elderly patient)(5)Radiation ileitis (appropriate history)(6)Lymphoma (no spasm, luminal narrowing is uncommon, tumor nodules)(7)Carcinoid tumor (tumor nodules)(8)Eosinophilic gastroenteritis(9)Potassium stricture EXTRAINTESTINAL MANIFESTATIONS @Hepatobiliary1.Fatty infiltration of liver (steroid therapy, hyperalimentation)2.Hepatic abscess3.Gallstones (28-34%)3-5 x higher risk than expected; stone formation caused by interrupted enterohepatic circulation with malabsorption of bile salts in terminal ileum; risk correlates with length of diseased ileum / resected ileum / duration of disease 4.Acute cholecystitis5.Sclerosing cholangitis (10%) + hepatoma6.Bile duct + gallbladder carcinoma@Genitourinary1.Urolithiasis: oxalate (frequent) / urate stones2.Hydronephrosis3.Renal amyloidosis4.Focal cystitis5.Ileoureteral / ileovesical fistula (5-20%)@Musculoskeletal ● digital clubbing (11-40%) ● mild self-limiting seronegative peripheral migratory arthritis (15-22%): may precede bowel disease in 10%; severity + course correlates well with severity of intestinal disease; resection of diseased bowel leads to regression of symptoms1.Hypertrophic osteoarthropathy2.Ankylosing spondylitis (in 3-16%)◊Axial skeletal involvement usually precedes onset of GI symptoms! ● unrelated in severity / course to activity level of bowel disease√ symmetric bilateral sacroiliitis√ spondylitis with syndesmophytes3.Peripheral erosive arthritis√ small marginal erosions√ periostitis√ propensity for osseous ankylosis4.Avascular necrosis of femoral head (steroid Rx)5.Pelvic osteomyelitis (contiguous involvement)6.Septic arthritis7.Muscle abscess8.Retarded skeletal growth + maturation@Erythema nodosum, uveitis

Notes:

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CRONKHITE-CANADA SYNDROME
=nonneoplastic nonhereditary inflammatory polyps (as in juvenile polyposis) associated with ectodermal abnormalities; no familial predisposition*Incidence:*>100 cases described*Histo:*hamartomatous polyps resembling juvenile / retention polyps = multiple cystic spaces filled with mucin secondary to degenerative changes; expansion + inflammation of lamina propria*Age:*62 years (range 42-75 years); M < F ● exudative protein-losing enteropathy ● diarrhea (disaccharidase deficiency, bacterial overgrowth in small intestine) ● severe weight loss, anorexia ● abdominal pain ● nail atrophy ● brownish macules of hand + feet ● alopecia√ multiple polyps√ thickened gastric rugaeLocation:stomach (100%); small bowel (>50%); colon (100%)*Prognosis:*rapidly fatal in women within 6-18 months (cachexia); tendency toward remission in men

Notes:

DESMOID TUMOR
=uncommon benign tumor consisting of fibrous tissue with insidious growth [desmos = "band / tendon"]=subgroup of fibromatoses*Types:* 1.ABDOMINAL DESMOIDLocation:mesentery (most common mesenteric primary), musculoaponeurosis of rectus, internal oblique muscle; occasionally external oblique muscle2.EXTRA-ABDOMINAL DESMOID=musculoaponeurotic fibromatosisLocation:pelvis, chest wall, mediastinum *Age:*peak age in 3rd decade, 70% between 20 and 40 years of age; M:F = 1:3*Path:*poorly circumscribed coarsely trabeculated tumor resembling scar tissue, confined to musculature + overlying aponeurosis*Histo:*elongated spindle-shaped cells of uniform appearance, septated by dense bands of collagen, infiltration of adjacent tissue (DDx: low-grade fibrosarcoma, reactive fibrosis)*Associated with:*Gardner syndrome, multiple pregnancies, prior trauma • firm slowly growing deep-seated massSize:5-20 cm in diameterMR: √ hypointense to muscle on T1WI + variable intensity on T2WICT: √ ill-defined / well-circumscribed mass√ usually higher attenuation than muscle√ ± enhancement√ retraction, angulation, distortion of small / large bowel with mesenteric infiltrationUS: √ sharply defined + smoothly marginated mass of low / medium / high echogenicity*Cx:*compression / displacement of bowel / ureter, intestinal perforation*Prognosis:*locally aggressive growth; 25-65% recurrence rate*Rx:*local resection + radiotherapy, antiestrogen therapy*DDx:*(1)Malignant tumor: metastasis, fibrosarcoma, rhabdomyosarcoma, synoviosarcoma, liposarcoma, fibrous histiocytoma, lymphoma, (2)Benign tumor: neurofibroma, neuroma, leiomyoma(3)Acute hematoma

Notes:

DIAPHRAGM DISEASE
=small bowel webs due to NSAIDs*Effect of NSAID:*gastric irritation, ulceration of small intestines*Frequency:*in 10% of patients receiving long-term NSAID therapy*Path:*foci of submucosal fibrosis with interruption of adjacent muscularis mucosae ● blood + protein loss ● intermittent intestinal obstructionLocation:ileum > jejunumEnteroclysis: √ multiple concentric diaphragm-like strictures*DDx:*Crohn disease

Notes:

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DISACCHARIDASE DEFICIENCY
=enzyme deficiencies for any of the disaccharides (maltose, lactose, etc.)A.PRIMARYB.SECONDARY to other diseases (eg, Crohn disease)*Pathophysiology:*
(a)unabsorbed disaccharides produce osmotic diarrhea(b)bacterial fermentation produces short-chain volatile fatty acids causing further osmotic + irritant diarrhea√ normal small bowel series without added lactose√ abnormal small bowel series done with lactose (50 g added to 600 cm^3 of barium suspension)√ small + large bowel distension√ dilution of barium√ shortening of transit time

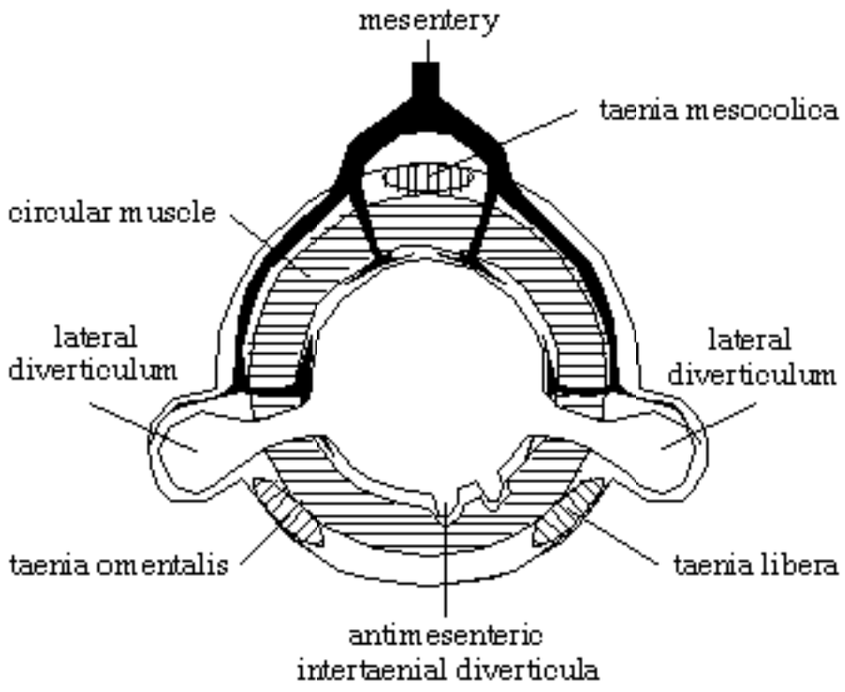
Notes:

DISTAL INTESTINAL OBSTRUCTION SYNDROME
=MECONIUM ILEUS EQUIVALENT=impaction of inspissated stool in distal part of ileum + proximal part of colon*Prevalence:*7-15-41% of children / adolescents with cystic fibrosis; 2% in patients <5 years of age*Cause:*tenacious intestinal mucus, steatorrhea due to pancreatic insufficiency, undigested food residue, disordered intestinal motility with increase in intestinal transit time, fecal stasis, dehydration*Age:*2nd-3rd decade of life ● recurrent bouts of colicky abdominal pain (from fecal impaction / constipation) in RLQ ● palpable cecal mass√ bubbly granular ileocecal soft-tissue mass in RLQ√ partial / complete small bowel obstruction (due to puttylike fecal material in terminal ileum / right colon)√ thickening of mucosal folds√ cystic fibrosis of lungCT: Location:cecum > ascending colon > transverse colon > descending colon (contiguous involvement)√ diffuse colonic thickening√ mural striation (50%)√ mesenteric soft-tissue infiltration (100%)√ increased pericolonic fat (60%)*Cx:*intussusception, volvulus*Rx:*stool softeners, oral polyethylene glycol-electrolyte solution (Go-lytely®), increasing dose of pancreatic enzyme supplements, mucolytic agents (N-acetylcysteine) orally / with Gastrografin® enema*DDx:*appendicitis, partial intestinal obstruction (adhesion / stricture from previous bowel surgery)

Notes:

DIVERTICULAR DISEASE OF COLON

=overactivity of smooth muscle causing herniation of mucosa + submucosa through muscle layers*Incidence:*5-10% in 5th decade; 33-48% over age 50; 50% past 7th decade; M:F = 1:1; most common affliction of colon in developed countries*Cause:*decreased fecal bulk (diet high in refined fiber + low in roughage)Location:in 80% in sigmoid (= narrowest colonic segment with highest pressure); in 17% distributed over entire colon; in 4-12% isolated to cecum / ascending colon



Cross Section through Colon

Prediverticular Disease Of Colon Colonic Diverticulosis Colonic Diverticulitis Colonic Diverticular Hemorrhage

Notes:

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Prediverticular Disease Of Colon =longitudinal + circular smooth muscle thickening with redundancy of folds secondary to myostatic contracture√ "saw-tooth sign" = crowding + thickening of haustral folds (shortening of colonic segment)√ plump marginal indentations√ superimposed muscle spasm (relieved by antispasmodics)*DDx:*hemorrhage; ischemia; radiation changes; pseudomembranous colitis

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Notes:

Colonic Diverticulosis =acquired herniations of mucosa + muscularis mucosae through the muscularis propria with wall components of mucosa, submucosa, serosa = false diverticula of pulsion typeSite: (a)lateral diverticula arise between mesenteric + antimesenteric teniae on opposite sides(b)antimesenteric intertaenial diverticula opposite of mesenteric sideIntramural type vasa recta (= nutrient arteries) pass through the circular muscle (weakness in muscular wall) and are carried over the fundus of the diverticula as it enlarges √ size: initially tiny (3- to 10-mm) V-shaped protrusions increasing up to several cm in diameter√ bubbly appearance of air-containing diverticula√ residual barium within diverticula from previous study√ spiky irregular outline (antimesenteric intertaenial ridge is typical site for intramural diverticula)√ smooth dome-shaped appendages with a short neck√ may be pointed, attenuated, irregular with variable filling√ circular line with sharp outer edge + fuzzy blurred inner edge (en face view in double contrast BE)√ **Giant sigmoid diverticulum** = large gas-containing cyst (air entrapment secondary to ball-valve mechanism) arising in left iliac fossaCT: √ diverticula√ distorted luminal contour + muscular hypertrophy

Notes:

Colonic Diverticulitis =perforation of diverticulum with intramural / localized pericolic abscess*Incidence:*5% of population; in 10-35% of diverticular disease; increasing frequency with age*Pathogenesis:*mucosal abrasion from inspissated fecal material leads to perforation of thin wall ● pain + local tenderness + mass in LLQ ● fever (25%), leukocytosis (36%) Location:sigmoid colon (most commonly)√ localized ileus√ ± pattern of small bowel obstruction (kinking / edema if small bowel adheres to abscess) √ gas in abscess / fistula√ pneumoperitoneum (rare)BE (77-86% sensitive): √ focal area of eccentric luminal narrowing caused by pericolic / intramural inflammatory mass√ marked thickening + distortion of mucosal folds√ mucosal tethering√ extraluminal contrast = PERIDIVERTICULITIS√ "double-tracking" = pericolonic longitudinal sinus tract√ pericolonic collection = peridiverticular abscess√ fistula to bladder / small bowel / vaginaCT (79-93% sensitive, 77% specific): √ poorly marginated hazy area of increased attenuation ± fine linear strands within pericolic fat (98%)√ diverticula (84%) = flask-shaped structures projecting through colonic wall + filled with air / barium / fecal material√ circumferential bowel wall thickening of >4 mm (70%)√ frank abscess (47%) = central liquid / gas√ fluid ± air of peritonitis (16%)√ fluid at root of mesentery√ fistula formation (14%):most commonly colovesical, also colovaginal, coloenteric, colocutaneous√ colonic obstruction (12%)√ intramural sinus tracts (9%)√ ureteral obstruction (7%)US (85-98% sensitive, 80-97% specific): √ thickening of bowel wall = >4 mm distance between echogenic lumen interface and serosa√ diverticula = round / oval hypo- / hyperechoic foci protruding from colonic wall with focal disruption of normal layer continuity ± internal acoustic shadowing√ inflammatory pericolic fat = regionally increased echogenicity adjacent to colonic wall ± ill-defined hypoechoic zones√ pericolic abscess*Prognosis:*(a)self-limiting (usually)(b)transmural perforation(c)superficial ulceration(d)chronic abscess*DDx:*(1)Colonic neoplasm (shorter segment, heaped-up margins, ulcerated mucosa)(2)Crohn colitis (double-tracking longer than 10 cm)*Rx:*antibiotics, surgery (in 25%), percutaneous abscess drainage

Notes:

Colonic Diverticular Hemorrhage Not related to diverticulitis *Incidence:*in 3-47% of diverticulosisLocation:75% located in ascending colon (larger neck + dome of diverticula) ● massive rectal hemorrhage without pain√ extravasation of radionuclide tracers√ angiographic contrast pooling in bowel lumen*Rx:*(1)transcatheter infusion of vasoconstrictive agents (Pitressin®)(2)embolization with Gelfoam®

Notes:

DUMPING SYNDROME

=early postprandial vascular symptomatology of sweating, flushing, palpitation, feeling of weakness and dizziness*Pathophysiology:*rapid entering of hypertonic solution into jejunum resulting in fluid shift from blood compartment into small bowel*Incidence:*1-5%; M:F = 2:1Roentgenologic findings not diagnostic! rapid emptying of barium into small bowel (= loss of gastric reservoir function)*Rx:*lying down, diet*DDx:*late postprandial hypoglycemia (90-120 minutes after eating)

Notes:

DUODENAL ATRESIA

=most common cause of congenital duodenal obstruction; second most common site of gastrointestinal atresias after ileum*Incidence:*1:10,000; M:F = 1:1*Etiology:*defective vacuolization of duodenum between 6th-11th weeks of fetal life; rarely from vascular insult (extent of obstruction usually involves larger regions with vascular insult)*Age at presentation:*first few days of life ● persistent bilious vomiting a few hours after birth / following 1st feeding ● rapid deterioration secondary to loss of fluids + electrolytesIsolated sporadic anomaly (30-52%) *Associated anomalies (in 60%):* (1)Down syndrome (20-33%);◊25% of fetuses with duodenal atresia have Down syndrome!◊<5% of fetuses with Down syndrome have duodenal atresia!(2)CHD (8-30-50%): endocardial cushion defect, VSD(3)Gastrointestinal anomalies (26%):esophageal atresia, biliary atresia, duodenal duplication, imperforate anus, small bowel atresia, intestinal malrotation, Meckel diverticulum, transposed liver, annular pancreas (20%) (4) Urinary tract anomalies (8%) (5) Vertebral + rib anomalies (37%) Location:(a)usually distal to ampulla of Vater (80%)(b)proximal duodenum (20%)√ "double bubble sign" = gas-fluid levels in duodenal bulb + gastric fundus√ total absence of intestinal gas in small / large bowel√ colon of normal caliberOB-US (usually not identified prior to 24 weeks GA): ● ± elevated AFP√ "double bubble sign" = simultaneous distension of stomach + 1st portion of duodenum, continuity of fluid between stomach + duodenum must be demonstrated√ increased gastric peristalsis√ polyhydramnios in 3rd trimester (100%)*Prognosis:*36% mortality in neonates*DDx:*(1)Prominent incisura angularis causing bidissection of stomach(2) Choledochal cyst(3)Annular pancreas(4)Peritoneal bands(5)Intestinal duplication*Cx:*prematurity (40%) secondary to preterm labor related to polyhydramnios

Notes:

DUODENAL DIVERTICULUM

*Incidence:*1-5% of GI studies; 22% of autopsiesA.PRIMARY DIVERTICULUM=mucosal prolapse through muscularis propriaLocation:2nd portion (62%), 3rd portion (30%), 4th portion (8%)Site:medial wall in region of papilla (88%), posteriorly (8%), lateral wall (4%)B.SECONDARY DIVERTICULUM=all layers of duodenal wall = true diverticulum as complication of duodenal / periduodenal inflammationLocation:almost invariably in 1st portion of duodenum ● mostly asymptomatic*Cx:*(1) Perforation +

peritonitis (2) Bowel obstruction(3) Biliary obstruction (4) Bleeding (5) Diverticulitis

Notes:

DUODENAL ULCER
*Incidence:*200,000 cases/year; 2-3 x more frequent than gastric ulcers; M:F = 3:1*Pathophysiology:*too much acid in duodenum from(a) abnormally high gastric secretion (b) inadequate neutralization *Predisposed:*cortisone therapy, severe cerebral injury, after surgery, chronic obstructive pulmonary diseaseLocation:(a)bulbar (95%):anterior wall (50%), posterior wall (23%), inferior wall (22%), superior wall (5%) (b)postbulbar (3-5%):majority on medial wall of supraampullary region; tendency for hemorrhage in 66%; M:F = 7:1 √ frequently small round / ovoid / linear ulcer niche√ "kissing ulcers" = ulcers opposite from each other on anterior + posterior wall√ giant duodenal ulcer >3 cm (rare) with higher morbidity + mortality; may be overlooked by simulating a normal / deformed duodenal bulb√ "cloverleaf deformity, hourglass stenosis" (healed stage) with prestenotic dilatation of recesses*Cx:*(1)Obstruction (5%)(2)Perforation (<10%): anterior > posterior wall; fistula to gallbladder(3)Penetration (<5%) = sealed perforation(4)Hemorrhage (15%): melena > hematemesis*Rx:*antral resection (Billroth I) + vagotomy

Notes:

DUODENAL VARICES

=dilated collateral veins secondary to portal hypertension (posterior superior pancreaticoduodenal vein)√ lobulated filling defects (best demonstrated in prone position, maximal luminal distension will obliterate them)√ commonly associated with fundal + esophageal varices

Notes:

DUPLICATION CYST

=uncommon congenital anomaly found anywhere along alimentary tract from tongue to anus*Incidence:*15% of pediatric abdominal masses are gastrointestinal duplication cysts*Theories of formation:* (1)Abortive twinning(2)Persistent embryologic diverticula(3)Split notochord(4)Aberrant luminal recanalization(5)Intrauterine vascular accidentassociated with alimentary tract atresia in 9% *Age:*presentation often in infancy / early childhood*Path:*spherical cyst / tubular structure located in / immediately adjacent to gastrointestinal tract; shares a common muscle wall + blood supply; has a separate mucosal lining; cyst contents are usually serous*Histo:*smooth muscle wall + lined with alimentary tract mucosa; ectopic mucosa squamous, transitional, ciliated mucosa; lymphoid aggregates; ganglion cells◊ Gastric mucosa + pancreatic tissue are the only ectopic tissues of clinical importance! ● respiratory distress (with esophageal duplication) ● palpable abdominal mass ● nausea, emesisLocation:ileum (30-33%), esophagus (17-20%), colon (13-30%), jejunum (10-13%), stomach (7%), pylorus (4%), duodenum (4-5%), ileocecal junction (4%), rectum (4%);◊In 7-15% concomitant duplications elsewhere in the alimentary tract!Site:on mesenteric aspect of alimentary canalMorphology: (a)large spherical / saccular cyst (82%)(b)small intramural cyst(c)tubular sausage-shaped cyst (18%): commonly along small + large bowel; frequently communicates with lumen of adjacent gut√ elongated tubular / spherical cystic mass√ muscular rim sign (= echogenic inner mucosal lining + hypoechoic outer rim) in 47%√ cyst paralleling normal bowel lumen*Cx:*bowel obstruction, intussusception, bleeding (due to presence of gastric mucosa / pressure necrosis of adjacent mucosa by cyst expansion / from intussusception)*DDx:* (1)Omental cyst (greater omentum / lesser sac, multilocular)(2)Mesenteric cyst (between leaves of small bowel mesentery)(3)Choledochal cyst(4)Ovarian cyst(5)Pancreatic pseudocyst(6)Cystic renal tumor(7)Abscess(8)Meckel diverticulum (communicates with GI tract)(9)Lymphangioma(10)Mesenteric lymphoma(11)Intramural tumor

Colonic Duplication Cyst Duodenal Duplication Cyst Esophageal Duplication Cyst Gastric Duplication Cyst Rectal Duplication Cyst Small Bowel Duplication Cyst Thoracoabdominal Duplication

Notes:

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Colonic Duplication Cyst *Incidence:*13% of all alimentary tract duplicationsA.CYSTIC COLONIC DUPLICATION (7%)*Path:*closed spherical cyst; contains gastric mucosa in 2% + ectopic pancreatic tissue in 5% ● abdominal mass, bowel obstruction, GI hemorrhageLocation:cecum (40%) ± intussusceptionB.COLORECTAL TUBULAR DUPLICATION (6%)=DUPLICATION OF THE HINDGUT=double-barreled duplication involving part / all of large bowel with "twin" segment on mesenteric / antimesenteric side*Symptomatic age:*neonatal period / infancy;M:F = 1:2 *May be associated with:* rectogenital / rectourinary fistula, duplication of internal / external genitalia, vertebral anomalies, multisystem congenital anomaly complex ● bowel obstruction ● passage of feces through vagina√ simultaneous opacification of true + twin colon√ duplication may terminate at(a)2nd functional anus(b)imperforate perineal orifice(c)fistulous communication with GU tractC.DOUBLE APPENDIX

Notes:

Duodenal Duplication Cyst *Incidence:*5% of all alimentary tract duplications*Path:*noncommunicating spherical cyst; may contain ectopic gastric mucosa in 21%, small bowel mucosa, pancreatic tissue ● obstruction, palpable abdominal mass ● hemorrhage (due to peptic ulceration) ● jaundice (due to biliary obstruction) ● pancreatitis (due to ectopic pancreatic tissue)Site:on mesenteric side of anterior wall of 1st + 2nd portion of duodenum√ mass in concavity of duodenal C-loop√ compression + displacement of 1st / 2nd portion of duodenum superiorly + anteriorly*Cx:*pancreatitis from perforation of duplication cyst *DDx:*pancreatic cyst, pancreatic pseudocyst, choledochal cyst, choledochocele, duodenal intramural tumor, pancreatic tumor

Notes:

Esophageal Duplication Cyst arises from foregut *Incidence:*10-20% of all alimentary tract duplications; 0.5-2.5% of all esophageal masses;M:F = 2:1*Path:*contains ectopic gastric mucosa in 43%*Histo:*contains no cartilage, lined by gastrointestinal tract epithelium*Associated with:* vertebral anomalies, esophageal atresia, small bowel duplication (18%)Location:adjacent to esophagus / within esophageal musculature at any level, paraspinal position; R:L = 2:1; in right pleural space detached from esophagus (rare)A.CERVICAL ESOPHAGUS (23%) ● asymptomatic enlarging lateral neck mass ● upper airway obstruction in newborn*DDx:*thyroglossal duct cyst, branchial cleft cyst, cystic hygroma, cervical tumor, cervical lymphadenopathyB.MIDESOPHAGUS (17%) ● severe upper airway obstruction in early infancy*DDx:*bronchogenic cyst, neurenteric cyst, intramural esophageal tumorC.DISTAL ESOPHAGUS (60%) ● frequently asymptomaticLocation:paraspinal*DDx:*bronchogenic cyst, neurenteric cyst, intramural esophageal tumor √ closed spherical cyst, almost never communicatingCXR: √ posterior mediastinal mass ± air-fluid level√ lobar consolidation + central cavitation (from autodigestion of lung tissue by gastric secretions)√ thoracic vertebral anomaliesUGI: √ displacement of esophagus by paraesophageal mass√ intramural extramucosal massUS: √ hypoechoic fluid-filled cyst + inner mucosal lining*Cx:*(1)Peptic ulceration (secondary to gastric mucosa)(2)Perforation (secondary to penetrating ulcer)(3)Hematemesis (from erosion into esophagus)(4)Hemoptysis + autodigestion of pulmonary tissue (from erosion into tracheobronchial tree)

Notes:

Gastric Duplication Cyst =intramural gastric cyst lined with secretory epithelium*Incidence:*7% of all alimentary tract duplications*Path:*noncommunicating spherical cyst (majority); may communicate with aberrant pancreatic duct; ectopic pancreatic tissue found in 37%*Symptomatic age:*infancy; in 75% detected before age 12; M:F = 1:2 ● pain (from overdistension of cyst, rupture with peritonitis, peptic ulcer formation, internal pancreatitis) ● vomiting, anemia, fever ● symptoms mimicking congenital hypertrophic pyloric stenosis (if duplication in antrum / pylorus)Most common site:greater curvature (65%)√ paragastric cystic mass up to 12 cm in size, indenting greater curvature√ seldom communicates with main gastric lumen at one or both ends√ may enlarge + ulcerate√ Tc-99m uptakeUS: √ cyst with two wall layers (inner echogenic layer of mucosa, outer hypoechoic layer of muscle)√ clear / debris-containing fluid*Cx:*(1)Partial / complete small bowel obstruction(2)Relapsing pancreatitis (with ductal communication)(3)Ulceration, perforation, fistula formation*DDx:*pancreatic cyst, pancreatic pseudocyst, mesenteric cyst, leiomyoma, adenomatous polyp, hamartoma, lipoma, neurofibroma, teratoma

Notes:

Rectal Duplication Cyst *Incidence:*4% of all alimentary tract duplications*Path:*spherical fluid-filled cyst; may contain duodenal / gastric mucosa + pancreatic tissueSite:posterior to rectum / anus√ communication with rectum / perianal fistula (in 20%)*Symptomatic age:*childhood ● constipation + fecal soiling ● palpable retrorectal / retroanal mass ● intractable excoriation of perianal skin (with chronic perianal fistula)√ cystic mass; may be echogenic (due to solid material ± gas from communication with rectum)*DDx:*anterior meningocele, sacrococcygeal teratoma, retrorectal abscess, pilonidal cyst, sacral bone tumor

Notes:

Small Bowel Duplication Cyst *Incidence:*most common of all alimentary tract duplications*Symptomatic age:*neonatal period (1/3); <2 years of age (in 72%)*Path:*contains ectopic gastric mucosa in 24%; ectopic pancreatic tissue in jejunum (8%)*May be associated with:* small bowel atresia ● neonatal bowel obstruction ● intussusception, palpable mass ● acute abdominal pain, hemorrhageLocation:ileum (33%), jejunum (10%), ileocecal (4%)√ low small bowel obstruction ± soft-tissue mass√ cyst may serve as lead point for intussusception*DDx:*mesenteric cyst, pancreatic pseudocyst, omental cyst, exophytic hepatic cyst, ovarian cyst

Notes:

Thoracoabdominal Duplication =FOREGUT DUPLICATION=long tubular cyst closed at its cranial end, passing through diaphragm through its own hiatus, in 60% communicating with normal duodenum / jejunum / ileum*Incidence:*2% of all alimentary tract duplications*Associated with:*thoracic vertebral anomalies*Histo:*gastric mucosa in 29%*Symptomatic age:*50% during neonatal period;80% within 1st year of life ● severe respiratory distress ● chest pain, GI bleeding, anemia√ tubular right posterior mediastinal mass ± air√ thoracic vertebral anomaly√ contrast material may enter through distal connection

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Notes:

ECTOPIC PANCREAS

=PANCREATIC REST*Incidence:*2-10% of autopsies; M:F = 2:1 ● asymptomaticLocation:distal greater curvature of antrum / pylorus (80%), duodenal bulb, jejunum, ileum, Meckel diverticulum; lesions may be multiple√ smooth cone- / nipple-shaped submucosal nodule 1-5 cm in size√ central umbilication representing orifice of filiform duct

Notes:

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ENTERIC CYST
=cyst lined by gastrointestinal mucosa without bowel wall*Etiology:*migration of small bowel / colonic diverticulum into mesentery / mesocolon*Path:*unilocular thin smooth-walled cyst with serous contents lined by enteric epithelium + thin fibrous wallUS: √ hypoechoic cystic mass, occasionally with septations*DDx:*duplication cyst (reduplication of bowel wall)

Notes:

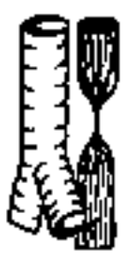
EOSINOPHILIC GASTROENTERITIS
=uncommon self-limited form of gastroenteritis with remissions + exacerbations characterized by infiltration of eosinophilic leukocytes into stomach / small bowel wall + usually marked peripheral eosinophilia*Cause:*unknown*Histo:*fibrous tissue + eosinophilic infiltrate of gastrointestinal mucosa*Age:*in children + young adults with allergy + eosinophilia A.EOSINOPHILIC GRANULOMA= FIBROUS POLYPOID LESION= INFLAMMATORY PSEUDOTUMOR=localized form / circumscribed type Location:almost exclusively in stomach (most common in antrum + pylorus)√ submucosal polypoid mass / pedunculated polypB.EOSINOPHILIC GASTROENTERITIS=diffuse type=eosinophilic infiltration of mucosa, submucosa, and muscular layers of small intestine ± stomach by mature eosinophils (? gastric pendant to Löffler syndrome) ● recurrent episodes of abdominal pain, diarrhea, vomiting ● weight loss ● hematemesis (from ulceration) ● peripheral eosinophilia, anemia ● history of systemic allergy / food allergyLocation:entire small bowel (particularly jejunum), distal stomach, omentum, mesenterySite:(a) mucosal (b) muscular (c) serosal (rare)@ Stomach (almost always limited to antrum) √"wet stomach"√ ulcers are rare(a)mucosal type√ enlarged gastric rugae / cobblestone nodules / polyps(b)muscular type√ thickened + rigid wall with narrowed gastric antrum / pylorus√ bulky intraluminal mass up to 9 cm in size*Cx:*pyloric obstruction*DDx:*hypertrophic gastritis, lymphoma, carcinoma@ Small bowel (involved in 50%) √ separation of small bowel loops(a)mucosal type ● malabsorption + hypoproteinemia√ thickening + distortion of folds predominantly in jejunum(b)submucosal / muscular type√ motility disturbance√ small-bowel obstruction√ effacement of mucosal pattern + narrowing of lumen(c)serosal type√ ascites*Prognosis:*tendency toward spontaneous remission*Rx:*steroids / removal of sensitizing agent

Notes:

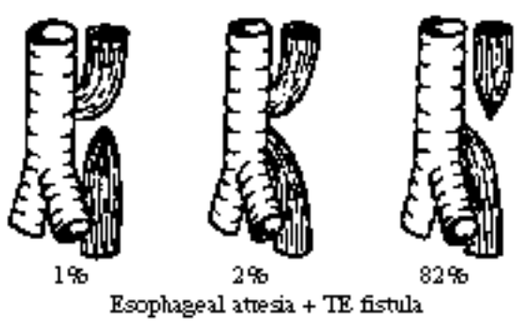
EPIPLOIC APPENDAGITIS
=rare inflammation of one of the 100 epiploic appendages*Cause:*(a)primary: torsion (exercise), venous thrombosis(b)secondary: inflammation of adjacent organ (eg, diverticulitis, appendicitis)*Histo:*acute infarction with fat necrosis, inflammation, thrombosed vessels with hemorrhagic suffusion ● abrupt onset of localized abdominal pain, gradually resolving over 3-7 days◊Almost never suspected preoperatively!Location:anterolaterally / (occasionally) anteromedially to ascending / descending / sigmoid colonUS: √ solid hyperechoic noncompressible ovoid mass√ hypoechoic margin (93%)CT: √ pericolonic oval-shaped pedunculated mass, 1-4 cm in diameter, with fat attenuation (approx. -60 HU)√ hyperattenuating peripheral rim + fat stranding√ thickening of adjacent visceral peritoneal lining (93%)*Prognosis:*spontaneous resolution*Rx:*conservative management*DDx:*torsion / infarction of greater omentum, diverticulitis, appendicitis

Notes:

ESOPHAGEAL ATRESIA & TRACHEOESOPHAGEAL FISTULA
=incomplete division of primitive foregut into respiratory + digestive tracts characterized by failure of formation of tubular esophagus + abnormal communication between esophagus + trachea; occurring at 3rd-5th week of intrauterine life*Incidence:*1:2,000-4,000 livebirths; most common sporadic congenital anomaly diagnosed in childhood*Risk of recurrence in sibling:* 1% *Associated anomalies* (17-56-70%):1.Cardiac (15-39%): patent ductus arteriosus, ASD, VSD, right-sided aortic arch (5%)2.Musculoskeletal (24%): radial ray hypoplasia, vertebral anomalies3.Gastrointestinal (20%): anorectal anomalies, duodenal atresia4.Genitourinary (12%): unilateral renal agenesis5.Chromosomal (3-19%): trisomy 18, 21, 13Trisomy 18 is present in 75-100% of fetuses + in 3-4% of neonates with esophageal atresia! *mnemonic:*"ARTICLES"**A**nal atresia **R**enal anomalies **T**E fistula **I**ntestinal atresia / malrotation **C**ardiac anomaly (PDA, VSD) **L**imb anomalies (radial ray hypoplasia, polydactyly) **E**sophageal atresia **S**pinal anomalies *mnemonic:*"VACTERL"**V**ertebral anomalies **A**norectal anomaly **C**ardiovascular anomalies **T**racheo- **E**sophageal fistula **R**enal anomalies **L**imb anomalies ● drooling from excessive accumulation of pharyngeal secretions (esophageal atresia = EA) ● obligatory regurgitation of ingested fluids (EA) ● coughing + choking during feeding (TEF) ● recurrent pneumonia + progressive respiratory distress of variable severity (tracheoesophageal fistula = TEF)Location:between upper 1/3 + lower 1/3 of esophagus just above carina √"coiled tube" = inability to pass feeding tube into stomach (esophageal atresia)√ retrotracheal air-filled pouch causing compression / displacement of esophagus√ gasless abdomen (esophageal atresia ± proximal TE fistula)√ bowel gas present in 90% (distal TE fistula / H-type fistula)√ non- / hypoperistaltic esophageal segment (6-15 cm) in midesophagus√ aspiration pneumonia, esp. in dependent upper lobes OB-US (anomalies not identified before 24 weeks GA): √ polyhydramnios in 33-60%◊TE-fistula with esophageal atresia is cause of polyhydramnios in only 3%!√ absence of fluid-distended stomach (in 10-41%; in remaining cases TE-fistula / gastric secretions allow some gastric distension)√ small abdomen (birth weight <10th percentile in 40%)√ distended proximal pouch of atretic esophagus *Cx after repair:* (1)Anastomotic leak(2)Recurrent TE fistula(3)Aspiration pneumonia secondary to(a)esophageal stricture(b)disordered esophageal motility distal to TE fistula(c)gastroesophageal reflux *DDx:*pharyngeal pseudodiverticulum (traumatic perforation of posterior pharynx from finger insertion into oropharynx during delivery / tube insertion)



Esophageal atresia 9%



1% 2% 82%
Esophageal atresia + TE fistula

TE fistula without esophageal atresia 6%

Esophageal Atresia Without Fistula (8-9%) Esophageal Atresia With Fistula Tracheo-esophageal Fistula Without Atresia (6%)

Notes:

Esophageal Atresia Without Fistula (8-9%)
Associated anomalies in 17% (mostly Down syndrome + other atresias of GI tract)

Notes:

Esophageal Atresia With Fistula 1.Proximal TE fistula (1%)2.Distal TE fistula (82-86%)3.Proximal + distal TE fistula (1-2%)Associated anomalies in 30% (mostly cardiovascular)

Notes:

Tracheo-esophageal Fistula Without Atresia (6%)
Associated anomalies in 23% (mostly cardiovascular)

Notes:

ESOPHAGEAL CANCER
Incidence:<1% of all cancers; 4-10% of all GI malignancies; 11,000 cases/year (United States in 1994); M:F = 4:1;Blacks:Whites = 2:1 *High-risk regions:*Iran, parts of Africa, Italy, China *Predisposing factors:* achalasia (risk factor of 1000 x), asbestosis, Barrett esophagus, celiac disease, ionizing radiation, caustic stricture (risk factor of 1000 x), Plummer-Vinson syndrome, tannins, alcohol, tobacco, history of oral / pharyngeal cancer, tylosis palmaris et plantaris *mnemonic:*"BELCH SPAT"**B**arrett esophagus **E**tOH abuse **L**ye stricture **C**eliac disease **H**ead and neck tumor **S**moking **P**lummer-Vinson syndrome **A**chalasia **T**ylosis *Cancer Staging:* TNM system: T1tumor invades lamina propria / submucosaT2tumor invades muscularis propria T3tumor invades adventitiaT4tumor invades adjacent structures Stage I=T1,N0,M0Stage III=T3,N1,M0Stage IIA=T2/3,N0,M0or T4,N0/1,M0Stage IIB=T1/2,N1,M0Stage IV=T1-4,N0/1,M1 CT staging (Moss): Stage 1intraluminal tumor / localized wall thickening of 3-5 mmStage 2localized / circumferential wall thickening >5 mmStage 3contiguous spread into adjacent mediastinum (trachea, bronchi, aorta, pericardium)√ loss of fat planes (nonspecific due to cachexia, often still resectable)√ mass in contact with aorta >90° arc (in 20-70% still resectable)√ displacement / compression of airway (90-100% accuracy for invasion)√ esophagotracheal / -bronchial fistula (unresectable)Stage 4distant metastases√ enlarged abdominal lymph nodes >10 mm (12-85% accuracy)√ hepatic, pulmonary, adrenal metastases√ direct erosion of vertebral body√ tumor >3 cm wide = high frequency of extra-esophageal spread*Histo:* (1)Squamous cell carcinoma (81-95%)(2)Adenocarcinoma (4-19%) arising from mucosal / submucosal glands or heterotopic gastric mucosa or columnar-lined epithelium (Barrett)(a)in 70% from Barrett esophagus(b)at gastroesophageal junction(3)Mucoepidermoid carcinoma, adenoid cystic carcinoma(4)Carcinosarcoma = pseudosarcoma = spindle-cell squamous carcinoma*Age:*in men >45 yearsLocation:usually middle third of esophagus√ large bulky polypoid smooth, lobulated, scalloped intraluminal mass, may be pedunculated(5)Leiomyosarcoma, rhabdomyosarcoma, fibrosarcoma, malignant lymphoma ● dysphagia (87-95%) of <6 months duration ● weight loss (71%) ● retrosternal pain (46%) ● regurgitation (29%)Location:upper 1/3 (15-20%); middle 1/3 (37-44%); lower 1/3 (38-43%) *Radiologic types:* (1)Polypoid / fungating form (most common)√ sessile / pedunculated tumor with lobulated surface√ protruding, irregular, polycyclic, overhanging, steplike "apple core" lesion(2)Ulcerating form√ large ulcer niche within bulging mass(3)Infiltrating form√ gradual narrowing with smooth transition(DDx: benign stricture) (4)Varicoid form = superficial spreading carcinoma*Histo:*longitudinal extension within wall without invasion beyond mucosa / submucosa√ tiny confluent nodules / plaques*DDx:*Candida esophagitis Metastases: (a)lymphogenic: anterior jugular chain + supraclavicular nodes (primary in upper 1/3); paraesophageal + subdiaphragmatic nodes (primary in middle 1/3); mediastinal + paracardial + celiac trunk nodes (primary in lower 1/3)(b)hematogenous: lung, liver, adrenal gland CXR: √ widened azygoesophageal recess with convexity toward right lung (in 30% of distal + midesophageal cancers)√ thickening of posterior tracheal stripe + right paratracheal stripe >4 mm (if tumor located in upper third of esophagus)√ widened mediastinum√ tracheal deviation√ posterior tracheal indentation / mass√ retrocardiac mass√ esophageal air-fluid level√ lobulated mass extending into gastric air bubble√ repeated aspiration pneumonia (with tracheoesophageal fistula) *Cx:*fistula formation to trachea (5-10%) / bronchi / mediastinum *Prognosis*:3-5-20% 5-year survival rate *Mean survival time:* 90 dayswith subdiaphragmatic lymphadenopathy180 dayswith local invasion + abdominal metastases480 dayswithout evidence of invasion / metastases *Rx:*(1)chemotherapy (fluorouracil, cisplatin, bleomycin sulfate, mitomycin) + surgery(2)chemotherapy + irradiation (~4,000 cGy)(3)chemotherapy + irradiation + surgery*Operative mortality:*3-8%

Notes:

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ESOPHAGEAL INTRAMURAL PSEUDODIVERTICULOSIS
=dilated excretory ducts of deep esophageal adnexal mucous glands*Etiology:*uncertain*Incidence:*about 100 cases in world literatureSite:diffuse / segmental involvement*In 90% associated with:* any severe esophagitis (most often reflux / Candida), esophageal stricture√ multiple tiny rounded / flask-shaped barium collections in longitudinal rows parallel to long axis of esophagus√ appear to "float" outside esophagus without apparent communication with lumen√ commonly associated with strictures in distal esophagus

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Notes:

ESOPHAGEAL PERFORATION

Cause: (1)Iatrogenic injury (most common cause, 55%): complication of endoscopy, dilatation of stricture, bougie, disruption of suture line following surgical anastomosis, attempted intubation(2)Spontaneous rupture = Boerhaave syndrome (15%): emetogenic injury of the esophagus from sudden increase in intra-abdominal pressure + relaxation of distal esophageal sphincter in the presence of a moderate to large amount of gastric contents(3)Closed chest trauma (10%)(4)Esophageal carcinoma(5)Retained foreign body (14%): coin, aluminum pop-tops, metallic button, safety pin, invisible plastic toy) leading to perforation (in pediatric age group)(6)Barrett ulcer ● pain, dysphagia, odynophagia ● rapid onset of overwhelming sepsis: fever, tachycardia, hypotension, shockPlain film (normal in 9-12%): √ pneumomediastinum√ subcutaneous emphysema of the neck√ delayed widening of the mediastinum (secondary to mediastinitis)√ hydrothorax (after rupture into pleural cavity), usually unilateral√ hydropneumothorax (often not initially seen)√ confirmation with contrast study (90% of contrast esophagrams are positive)CT: √ extraluminal air (92%; most useful sign)√ periesophageal / mediastinal fluid (92%)√ pleural effusion (75%)√ esophageal thickening√ extravasation of oral contrast materialEsophagography with: (1)water-soluble contrast material (10% false-negative results)(2)barium (if result with water-soluble material negative)A.UPPER / MID-ESOPHAGEAL PERFORATIONLocation:at level of cricopharyngeus muscle (most frequent)√ widening of upper mediastinum√ right-sided hydrothoraxB.DISTAL ESOPHAGEAL PERFORATION (more common)*Cause:*biopsy, dilatation of stricture, Boerhaave syndrome√ left-sided hydrothorax√ little mediastinal changes*Cx:*(1) Acute mediastinitis (2) Obstruction of SVC(3) Mediastinal abscess*Prognosis:*20-60% mortality

Notes:

ESOPHAGEAL VARICES

=dilated submucosal veins due to increased collateral blood flow from portal venous system to azygos systemA.UPHILL VARICES=collateral blood flow from portal vein via azygos vein into SVC (usually lower esophagus drains via left gastric vein into portal vein)*Cause:* (a)intrahepatic obstruction from cirrhosis(b)splenic vein thrombosis (usually gastric varices)(c)obstruction of hepatic veins(d)IVC obstruction below hepatic veins(e)IVC obstruction above hepatic vein entrance / CHF(f)marked splenomegaly / splenic hemangiomatosis (rare)√ varices in lower half of esophagusB.DOWNHILL VARICES=collateral blood flow from SVC via azygos vein into IVC / portal venous system (upper esophagus usually drains via azygos vein into SVC)*Cause:*obstruction of superior vena cava distal to entry of azygos vein most commonly due to lung cancer, lymphoma, retrosternal goiter, thymoma, mediastinal fibrosis√ varices in upper 1/3 of esophagus EXAMINATION TECHNIQUE (a)small amount of barium (not to obscure varices)(b)relaxation of esophagus (not to compress varices): refrain from swallowing because succeeding swallow initiates a primary peristaltic wave that lasts for 10-30 seconds; sustained Valsalva maneuver precludes from swallowing(c)in LAO projection with patient recumbent / in Trendelenburg position ± Valsalva maneuver / deep inspirationPlain film: √ lobulated masses in posterior mediastinum (visible in 5-8% of patients with varices)√ silhouetting of descending aorta √ abnormal convex contour of azygoesophageal recessUGI: √ thickened sinuous interrupted mucosal folds (earliest sign)√ tortuous radiolucencies of variable size + location√ "worm-eaten" smooth lobulated filling defects√ findings may be accentuated after sclerotherapyCT: √ thickened esophageal wall + lobulated outer contour√ scalloped esophageal luminal masses√ right- / left-sided soft-tissue masses(= paraesophageal varices)√ marked enhancement following dynamic CT*Cx:*bleeding in 28% within 3 years; exsanguination in 10-15%*DDx:*varicoid carcinoma of esophagus

Notes:

ESOPHAGEAL WEB
=ringlike esophageal constriction caused by thin mucosal membrane projecting into lumen; covered by squamous epithelium on superior + inferior surfaces*Age:*middle-aged females*? association with:* **Plummer-Vinson syndrome** = Paterson-Kelly syndrome (iron deficiency anemia, stomatitis, glossitis, dysphagia, thyroid disorder, spoon-shaped nails) *Cause:mnemonic:*"BIEP"**B**-ring (Schatzki ring) **I**diopathic (= transverse mucosal fold) **E**pidermolysis bullosa **P**lummer-Vinson disease *Path:*hyperkeratosis + chronic inflammation of submucosa ● mostly asymptomatic (unless severely stenosing)Location:in cervical esophagus near cricopharyngeus (most common) > thoracic esophagus; occasionally multiple√ visualized during maximal distension (in one tenth of a second)√ arises at right angles from anterior esophageal wall√ thin delicate membrane of uniform thickness of <3 mm*Cx:*high risk of upper esophageal + hypopharyngeal carcinoma*Rx:*(1)balloon dilatation(2)bougienage during esophagoscopy*DDx:*stricture (circumferential + thicker =1- to 2-mm thick [vertical length] area of complete / incomplete circumferential narrowing

Notes:

Acute Esophagitis *mnemonic for cause:* "CRIER" **C**orrosives, **C**rohn disease **R**eflux **I**nfection, **I**ntubation **E**pidermolysis bullosa **R**adiation therapy √ thickened >3-mm-wide folds with irregular lobulated contour√ mucosal nodularity (= multiple ulcerations + intervening edema)√ erosions√ vertically oriented ulcers usually 3-10 mm in length√ inflammatory esophagogastric polyp = proximal gastric fold extending across esophagogastric junction (rare)√ abnormal motility

Notes:

Candida Esophagitis =MONILIASIS = CANDIDIASIS√ Most common cause of infectious esophagitis!*Organism:*C. albicans, C. tropicalis; endogenous (majority) / transmitted by another human / animal; often discovered in diseased skin, GI tract, sputum, female genital tract, urine with an indwelling Foley catheter*Predisposed:* (a)individuals with depressed immunity: hematologic disease, renal transplant, leukemia, chronic debilitating disease, diabetes mellitus, steroids, chemotherapy, radiotherapy, AIDS◊Most common type of fungi found with opportunistic infections!(b)delayed esophageal emptying: scleroderma, strictures, achalasia, S/P fundoplication(c)antibiotics*Path:*patchy, creamy-white plaques covering a friable erythematous mucosa*Histo:*mucosal plaques = necrotic epithelial debris + fungal colonies ● dysphagia (= difficulty swallowing) ● severe odynophagia (= painful swallowing from segmental spasm) ● intense retro- / substernal pain ● associated with thrush (= oropharyngeal moniliasis) in 20-50-80%Location:predilection for upper 1/2 of esophagus√ involvement of long esophageal segments√ longitudinal plaques = grouping of tiny 1-2 mm nodular filling defects with linear orientation(= heaped-up areas of mucosal plaques)√ "cobblestone" appearance = mucosal nodularity in early stage (from growth of colonies on surface)√ **shaggy** / fuzzy / serrated contour (from coalescent plaques, pseudomembranes, erosions, ulcerations, intramural hemorrhage) in fulminant candidiasis√ narrowed lumen (from spasm, pseudomembranes, marked edema)√ "intramural diverticulosis" = multiple tiny indentations + protrusions√ sluggish / absent primary peristalsis√ strictures (rare)√ mycetoma resembling large intraluminal tumor (rare)*Diagnostic sensitivity:*endoscopy (97%), double contrast (88%), single contrast (55%)*Cx:*(1)systemic candidiasis ("microabscesses" in liver, spleen, kidney)(2)gastric bezoar due to large fungus ball (after long-standing esophageal candidiasis)*Rx:*Mycostatin®*DDx:*glycogen acanthosis, reflux esophagitis, superficial spreading carcinoma, artifacts (undissolved effervescent crystals, air bubbles, retained food particles), herpes esophagitis, acute caustic ingestion, intramural pseudo-diverticulosis, squamous papillomatosis, Barrett esophagus, epidermolysis bullosa, varices

Notes:

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Caustic Esophagitis =CORROSIVE ESOPHAGITIS*Corrosive agents:* lye (sodium hydroxide), washing soda (sodium carbonate), household cleaners, iodine, silver nitrate, household bleaches, Clinitest® tablets (tend to be neutralized by gastric acid) ◊Severity of injury dependent on contact time + concentration of corrosive material!*Associated with:*injury to pharynx + stomach (7-8%): antral burns more common with acid (buffering effect of gastric acid on alkali)Location:middle + lower thirds of esophagusStageI:acute necrosis from protein coagulation√ mucosal blurring (edema)√ diffusely atonic + dilated esophagus√ tertiary contractionsStageII:frank ulceration in 3-5 days√ ulceration + pseudomembranesStageIII:scarring + stricture from fibroblastic activity√ long segmental stricture after 10 days when acute edema subsides (7-30%)*Cx:*(1)Esophageal / gastric perforation during ulcerative stage(2)Squamous cell carcinoma in injured segment

Notes:

Chronic Esophagitis √ luminal narrowing with tapered transition to normal + proximal dilatation √ circumferential / eccentric stricture √ sacculations = pseudodiverticula

Notes:

Cytomegalovirus Esophagitis *Organism:*member of herpesvirus group*Associated with:*AIDS ● severe odynophagia√ diffusely normal mucosal background√ one / more **large ovoid flat ulcers** (up to several cm in size) near gastroesophageal junction√ discrete small superficial ulcers indistinguishable from herpes esophagitis (uncommon)*Rx:*ganciclovir (relatively toxic) *Dx:*endoscopic brushings, biopsy specimen, cultures

Notes:

Drug-induced Esophagitis *Agents:*tetracycline, doxycycline, potassium chloride, quinidine, aspirin, ascorbic acid, alprenolol chloride, emepronium bromide ● severe odynophagia ● history of taking medication with little / no water immediately before going to bedLocation:midesophagus at site of compression by aortic arch / left mainstem bronchus√ superficial solitary / several discrete / localized clusters of tiny ulcers distributed circumferentially√ dramatic healing of lesion 7-10 days after withdrawal of offending agent*DDx:*herpes esophagitis (less localized)

Notes:

Herpes Esophagitis ◊2nd most common cause of opportunistic infection!*Organism:*Herpes simplex virus type I (DNA core virus) secreted in saliva of 2% of healthy population*Age:*15-30 years; usually males ● history of recent exposure to sexual partners with herpetic lesions on lips / buccal mucosa ● flulike prodrome of 3-10 days (headaches, fever, sore throat, upper respiratory symptoms, myalgia) ● severe acute dysphagia / odynophagia*May be associated with:*oropharyngeal herpetic lesions / oropharyngeal candidiasisLocation:midesophagus (level of left main bronchus)√ initially vesicles / blisters that subsequently rupture√ **multiple small** discrete superficial punctate / linear / stellate (often "diamond shaped") **ulcers** surrounded by radiolucent halos of edematous mucosa√ intervening mucosa normal (without plaques)√ multiple plaquelike lesions (only with severe infection)*Rx:*oral / intravenous acyclovir*Dx:*rising serum titer for HSV type 1, viral culture, biopsy (immunofluorescent staining for HSV antigen, demonstration of intranuclear inclusions)*DDx:*drug-induced esophagitis, Crohn disease, esophageal intramural pseudodiverticulosis

Notes:

Human Immunodeficiency Virus Esophagitis ● maculopapular rash + ulcers of soft palate√ one / more **giant flat** ovoid / diamond-shaped **ulcers** (at time of seroconversion) indistinguishable from CMV esophagitis*Dx:*ONLY per exclusion*DDx:*CMV esophagitis, mycobacterial esophagitis, actinomycosis, potassium chloride, quinidine, caustic ingestion, nasogastric intubation, radiation therapy, endoscopic sclerotherapy

Notes:

Reflux Esophagitis =esophageal inflammation secondary to reflux of acid-peptic contents of the stomach; reflux occurs if resting pressure of LES <5 mm Hg (may be normal event if followed by rapid clearing)*Histo:*basal cell hyperplasia with wall thickening + thinning of epithelium, mucosal edema + erosions, inflammatory infiltrate*Determinants:*(1)Frequency of reflux(2)Adequacy of clearing mechanism(3)Volume of refluxed material(4)Potency of refluxed material(5)Tissue resistanceReflux preventing features: (1)Lower esophageal sphincter(2)Phrenoesophageal membrane(3)Length of subdiaphragmatic esophagus(4)Gastroesophageal angle of His (70-110°)*May be associated with:*sliding hiatal hernia (in most patients), scleroderma, nasogastric intubation ● heartburn, epigastric discomfort ● choking, globus hystericus ● retrosternal pain ● thoracic / cervical dysphagia Site:usually lower 1/3 / lower 1/2 with continuous disease extending proximally from GE junction √ segmental esophageal narrowing (edema / spasm / stricture)√ granular / finely nodular appearance of thickened longitudinal mucosal folds with poorly defined borders (mucosal edema + inflammation) in early stages√ single marginal ulcer / erosion at or adjacent to gastroesophageal junction√ multiple areas of superficial ulceration in distal esophagus√ prominent mucosal fold ending in polypoid protuberance within hiatal hernia / cardia√ interruption of primary peristalsis at inflamed segment√ nonperistaltic waves in distal esophagus following deglutition (85%)√ incomplete relaxation of LES (75%), incompetent sphincter (33%)√ acid test = abnormal motility elicited by acid barium (pH 1.7)√ "felinization" = transverse ridges of esophagus secondary to contraction of muscularis mucosae (similar to cat esophagus)NUC (pertechnetate): √ esophageal activity (Barrett esophagus similar to ectopic gastric mucosa) *Reflux tests:* 1.Reflux of barium in RPO position, may be elicited by coughing / deep respiratory movements / swallowing of saliva + water / anteflexion in erect position: only in 50% accurate2.Water-siphon test: in 5% false negative; large number of false positives3.Tuttle test = measurement of esophageal pH: 96% accurate4.Radionuclide gastroesophageal reflux test (typically combined with gastric emptying test):*Technique:*ROI drawn over distal esophagus + compared with time-activity curve over stomach, scaled to 4%√ esophageal activity >4% stomach activity *Cx of reflux:* (a)from acid + pepsin acting on esophageal mucosa:1.Motility disturbance2.Stricture3.Schatzki ring4.Barrett esophagus5.Iron-deficiency anemia6.Reflux / peptic esophagitis(b)from aspiration of gastric contents1.Acute aspiration pneumonia2.Mendelson syndrome3.Pulmonary fibrosis

Notes:

Viral esophagitis *Predisposed:*immunocompromised, eg, underlying malignancy, debilitating illness, radiation treatment, steroids, chemotherapy, AIDS

Notes:

FAMILIAL ADENOMATOUS POLYPOSIS
=FAMILIAL MULTIPLE POLYPOSIS = autosomal dominant disease with 80% penetrance (gene for familial polyposis localized on chromosome 5); sporadic occurrence in 1/3*Incidence:*1:7,000 to 1:24,000 livebirths*Histo:*tubular / villotubular adenomatous polyps; usually about 1,000 adenomas*Age:*polyps appear around puberty ● family history of colonic polyps (66%)Screening of family members after puberty! ● clinical symptoms begin during 3rd-4th decade (range 5-55 years) ● vague abdominal pain, weight loss ● diarrhea, bloody stools ● protein-losing enteropathy (occasionally)*Associated with:*(1)Hamartomas of stomach in 49%(2)Adenomas of duodenum in 25%(3)Periampullary carcinoma "carpet of polyps" = myriad of 2-3 mm (up to 2 cm) polypoid lesions@Colon (100%): more numerous in distal colon; always affecting rectum normal haustral pattern@Stomach (5%)@Small bowel (<5%)*Cx:*malignant transformation: colon > stomach > small bowel (in 12% by 5 years; in 30% by 10 years; in 100% by 20 years after diagnosis; age at carcinomatous development usually 20-40 years; multiple carcinomas in 48%)Periampullary carcinoma is the most common cause of death after prophylactic colectomy!*Rx:*prophylactic total colectomy in late teens / early twenties before symptoms develop +(1)Permanent ileostomy(2)Continent endorectal pull-through pouch(3)Kock pouch (= distal ileum formed into a one-way valve by invaginating the bowel at skin site) *DDx:*other polyposes, lymphoid hyperplasia, lymphosarcoma, ulcerative colitis with inflammatory pseudopolyps

Notes:

GALLSTONE ILEUS
*Incidence:*0.4-5% of all intestinal obstructions (20% of obstruction in patients >65 years; 24% of obstructions in patients >70 years); develops in <1% of patients with cholelithiasis; in 1 of 6 perforations; risk increases with age*Age:*average 65-75 years; M:F = 1:4 -7 ● previous history of gallbladder disease ● intermittent episodes of acute colicky abdominal pain (20-30%) ● nausea, vomiting, fever, distension, obstipation √ **Rigler triad** on plain film:1.Partial / complete intestinal obstruction (usually small bowel), "string of rosary beads" = multiple small amounts of air trapped between dilated + stretched valvulae conniventes (in 86%)2.Gas in biliary tree (in 69%)3.Ectopic calcified gallstone (in 25%): stones are commonly >2.5 cm in diameter√ change in position of previously identified gallstoneUGI / BE: √ well-contained localized barium collection lateral to first portion of duodenum (barium-filled collapsed GB + possibly biliary ducts)Fistulous communication: CHOLECYSTODUODENAL (60%), choledochoduodenal, cholecystocolic, choledochocolic, cholecystogastric √ identification of site of obstruction: terminal ileum (60-70%), proximal ileum (25%), distal ileum (10%), pylorus, sigmoid, duodenum (Bouveret syndrome)*Cx:*recurrent gallstone ileus in 5-10% (additional silent calculi more proximally)*Prognosis:*high mortality

Notes:

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GANGLIOCYTIC PARAGANGLIOMA
=rare benign tumor of the GI tract*Frequency:*<100 cases reported*Origin:*pancreatic endocrine rest that remained when the ventral primordium rotated around the duodenum*Age:*50-60 years of age; M:F = 2:1Location:almost exclusively in 2nd portion of duodenum near the ampulla of Vater on the medial / lateral wall of duodenum

- GI hemorrhage, abdominal pain√ polypoid smooth-surfaced intraluminal mass√ homogeneously enhancing mural / extrinsic solid mass of soft-tissue attenuation√ well-circumscribed hypoechoic mass contiguous with bowel√ no biliary duct dilatation*DDx:*adenocarcinoma (biliary duct dilatation, hypovascular), leiomyosarcoma (cystic internal hemorrhage / necrosis), hemangioma, duplication cyst, choledochal cyst, lipoma, hamartoma, inflammatory fibroid polyp (distal small bowel), lymphoma (isolated in stomach and ileum)

Notes:

GARDNER SYNDROME
=autosomal dominant disease (? variant of familial polyposis) characterized by a triad of (1) colonic polyposis (2) osteomas (3) soft-tissue tumors*Histo:*adenomatous polyps*Age:*15-30 years*Associated with:* ? MEA complex(1) periampullary / duodenal carcinoma (12%) (2) thyroid carcinoma (3) adrenal adenoma / carcinoma (4) parathyroid adenoma (5) pituitary chromophobe adenoma (6) carcinoid, adenoma of small bowel (7) retroperitoneal leiomyoma ● skin pigmentation◊Familial polyposis + Gardner syndrome may occur in the same family!◊Extraintestinal manifestations occur usually earlier than in intestinal polyposis!@PolyposisLocation:colon (100%), stomach (5-68%), duodenum (90%), small bowel (<5%)√ multiple colonic polyps appearing during puberty, increasing in number during 3rd-4th decade√ lymphoid hyperplasia of terminal ileum√ hamartomas of stomach@Soft-tissue tumors(a)sebaceous / epidermoid inclusion cysts (scalp, back, face, extremities)(b)fibroma, lipoma, leiomyoma, neurofibroma(c)desmoid tumors (3-29%); peritoneal adhesions (desmoplastic tendency); mesenteric fibrosis, retroperitoneal fibrosis, mammary fibromatosis, marked keloid formation, hypertrophied scars (anterior abdominal wall) arise 1-3 years after surgery ● GI / urinary tract obstruction@Osteomatosis of membranous bone (50%)Location:calvarium, mandible (81%), maxilla, ribs, long bones@Long bones√ localized wavy cortical thickening / exostoses√ slight shortening + bowing@Teeth√ odontoma, unerupted supernumerary teeth, hypercementosis√ tendency toward numerous caries (dental prosthesis at early age)*Cx:*malignant transformation in 100% (average age at death is 41 years if untreated)*Rx:*prophylactic total colectomy at about 20 years of age

Notes:

GASTRIC CARCINOMA

3rd most common GI malignancy after colorectal + pancreatic cancer, 6th leading cause of cancer deaths *Prevalence:*declining; 24,000 cases/year in USA*Risk factors:*smoking, nitrites, nitrates, pickled vegetables*Predisposed:*pernicious anemia (risk factor of 2), chronic atrophic gastritis, adenomatous + villous polyp (7-27% are malignant), gastrojejunostomy, Billroth II > Billroth I *Histo:*adenocarcinoma (95%); rarely squamous cell carcinoma / adenoacanthoma *Staging:* T1tumor limited to mucosa / submucosaT2tumor involves muscle / serosaT3tumor penetrates through serosaT4ainvasion of adjacent contiguous tissuesT4binvasion of adjacent organs, diaphragm, abdominal wallN1involvement of perigastric nodes within 3 cm of primary along greater / lesser curvatureN2involvement of regional nodes >3 cm from primary along branches of celiac axisN3paraaortic, hepatoduodenal, retropancreatic, mesenteric nodesM1distant metastases Location:mostly distal third of stomach + cardia; 60% on lesser curvature, 10% on greater curvature; esophagogastric junction in 30% Probability of malignancy of an ulcer: at lesser curvature 10-15%, at greater curvature 70%, in fundus 90% *Morphology:* 1.Polypoid / fungating carcinoma2.Ulcerating / penetrating carcinoma (70%)3.Infiltrating / scirrhous carcinoma (5-15%)= linitis plastica *Histo:*frequently signet ring cell type + increase in fibrous tissueLocation:antrum, fundus + body (38%)√ firmness, rigidity, reduced capacity of stomach, aperistalsis in involved area√ granular / polypoid folds with encircling growth4.Superficial spreading carcinoma=confined to mucosa / submucosa; 5-year survival of 90%√ patch of nodularity√ little loss of elasticity5.Advanced bulky carcinoma ● GI bleeding, abdominal pain, weight loss UGI: √ rigidity√ filling defect√ amputation of folds ± ulceration ± stenosis√ calcifications (mucinous adenocarcinoma)CT: √ irregular nodular luminal surface√ asymmetric thickening of folds√ mass of uniform density / varying attenuation√ wall thickness >6 mm with gas distension + 13 mm with positive contrast material distension√ increased density in perigastric fat √ enhancement exclusively in linitis plastica type√ nodules of serosal surface (= dilated surface lymphatics)√ diameter of esophagus at gastroesophageal junction larger than adjacent aorta (DDx: hiatal hernia)√ lymphadenopathy below level of renal pedicle (3%) *Metastases:* 1.along peritoneal ligaments(a)gastrocolic lig.: transverse colon, pancreas(b)gastrohepatic + hepatoduodenal lig.: liver2.local lymph nodes3.hematogenous: liver (most common), adrenals, ovaries, bone (1.8%), lymphangitic carcinomatosis of lung (rare) 4.peritoneal seeding:on rectal wall=Blumer shelfon ovaries=Krukenberg tumor5.left supraclavicular lymph node = Virchow node*Prognosis:* overall 5-year survival rate of 5-18%, mean survival time of 7-8 months; -85% 5-year survival in stage T1-52%5-year survival in stage T2-47%5-year survival in stage T3-17%5-year survival in stage N1-2- 5%5-year survival in stage N3

Prognostic Parameters of Gastric Carcinoma

Tumor Size	Metastases	Limited to Submucosa	5-Year Survival Rate
1 cm	11%		87%
2 cm	25%	70%	67%
3 cm	45%		35%
4 cm	59%	60%	33%
>4 cm	72%	33%	

Early Gastric Cancer (20%) Advanced Gastric Cancer (T2 lesion and higher)

Notes:

Early Gastric Cancer (20%)
=invasion limited to mucosa + submucosa (T1 lesion)*Classification of Japan Research Society for Gastric Cancer:* TypeIProtruded type = >0.5 cm height with protrusion into gastric lumen (10-20%)TypeIISuperficial type = <0.5 cm heightIIaslightly elevated surface (10-20%)IIbflat / almost unrecognizable (2%)IIcslightly depressed surface (50-60%)TypeIIIExcavated type (5-10%)

Notes:

Advanced Gastric Cancer (T_2 lesion and higher)
Bormann classification: Type 1broad-based elevated polypoid lesionType 2elevated lesion + ulceration + well-demarcated marginType 3elevated lesion + ulceration + ill-defined marginType 4ill-defined flat lesionType 5unclassified, no apparent elevation

Notes:

GASTRIC DIVERTICULUM
stomach is least common site of diverticula *Incidence:*1:600-2,400 of UGI studies*Etiology:*(a)traction secondary to scarring / periantral inflammation = true diverticulum(b)pulsion (less common) = false diverticulum*Age:*beyond 40 years*Often associated with:* aberrant pancreas in antral location Location:juxtacardiac on posterior wall (75%), prepyloric (15-22%), greater curve (3%)√ pliability + varying degrees of distension√ NO mass, edema or rigidity of adjacent folds *DDx:*small ulcer in intramural-extramucosal mass

Notes:

GASTRIC POLYP

*Incidence:*1.5-5%, most common benign gastric tumor *Associated with:*hyperacidity + ulcers, chronic atrophic gastritis, gastric carcinoma A.NONNEOPLASTIC1.INFLAMMATORY POLYP (75-90%)= HYPERPLASTIC POLYP = REGENERATIVE POLYP *Histo:*cystically dilated glands lined by gastric epithelium + acute and chronic inflammatory infiltrates in lamina propria*Associated with:*chronic atrophic gastritis, pernicious anemiaLocation:random distribution within stomach; usually multiple√ sharply delineated polyp with smooth circular border√ "Mexican hat sign" = stalk seen en face overlying the head of polyp√ sessile / pedunculated√ usually <2 cm in diameter without progression√ no contour defect of stomach*Prognosis:*no malignant potential2.HAMARTOMATOUS POLYP (rare)*Histo:*densely packed gastric glands + bundles of smooth muscle*Associated with:*Peutz-Jeghers syndrome√ sessile / pedunculated√ usually <2 cm in diameter3.RETENTION POLYP (rare)*Histo:*dilated cystic glands + stroma*Associated with:*Cronkhite-Canada syndrome B.NEOPLASTIC1.ADENOMATOUS POLYP (10-20%)=true neoplasm with malignant potential (10-80%, increasing with size)*Age:*increasing incidence with age; M:F = 2:1*Histo:*intestinal metaplasia (common) + marked cellular atypism*Associated with:*Gardner syndrome; coexistent with gastric carcinoma in 35%Location:more commonly in antrum (antrum spared in Gardner syndrome)√ broad-based elliptical / mushroom-shaped ± pedicle; often single√ usually >2 cm in diameter (in 80%)√ smooth / irregular lobulated contour2.VILLOUS POLYP (rare)√ trabeculated / lobulated slightly irregular contour*Cx:*malignant transformation *DDx:*(1)Ménétrièr disease (antrum spared)(2)Eosinophilic polyp (peripheral eosinophilia, linitis plastica appearance, small bowel changes)(3)Lymphoma(4)Carcinoma

Notes:

Benign Gastric Ulcer 95% of all gastric ulcers *Cause:* 1.Stress2.Burns = curling ulcer3.Cerebral disease = Cushing ulcer4.Uremia5.Severe prolonged illness6.Gastritis7.Steroid therapy8.Intubation9.Stasis ulcer proximal to pyloric / duodenal obstruction10.HPT (25% with ulcer disease)*Pathophysiology:* disrupted mucosal barrier (Helicobacter pylori) with vulnerability to acid + secretion of large volume of gastric juice containing little acid *Incidence:*5:10,000; 100,000/year (United States)*Age peak:*55-65 years; M:F = 1:1*Multiplicity:* (a)multiple in 2-8% (17-24% at autopsy), especially in patients on Aspirin®(b)coexistent duodenal ulcer in 5-64%; gastric:duodenal = 1:3 (adults) = 1:7 (children) ● abdominal pain: in 30% at night, in 25% precipitated by foodLocation:lesser curvature at junction of corpus + antrum within 7 cm from pylorus; proximal half of stomach in older patients (geriatric ulcer); adjacent to GE junction within hiatal hernia√ ulcer size usually <2 cm (range 1-250 mm); in 4% >40 mm√ Haudek niche = conical / collar button-shaped barium collection projecting outside gastric contour (profile view)√ Hampton line = 1-mm thin straight lucent line traversing the orifice of the ulcer niche (seen on profile view + with little gastric distension) = ledge of touching overhanging gastric mucosa of undermined benign ulcer√ ulcer collar = smooth thick lucent band interposed between the niche and gastric lumen (thickened rim of edematous gastric wall) in well-distended stomach√ ulcer mound = smooth, sharply delineated, gently sloping extensive tissue mass surrounding a benign ulcer (edema + lack of wall distensibility) in well-distended stomach√ ulcer crater = round / oval barium collection with smooth border on dependent side (en face view)√ halo defect = wide lucent band symmetrically surrounding ulcer resembling extensive ulcer mound (viewed en face)√ ring shadow: ulcer on nondependent side (en face view)√ radiating thick folds extending directly to crater edge fusing with the effaced marginal fold of the ulcer collar / halo of ulcer mound√ incisura defect = smooth, deep, narrow, sharp indentation on greater curvature opposite a niche on lesser curvature at / slightly below the level of the ulcer (spastic contraction of circular muscle fibers)*Prognosis:*healing in 50% by 3 weeks, in 100% by 6-8 weeks; slower healing in older patients; only complete healing proves benignancy*Cx:*bleeding, perforation

Notes:

Malignant Gastric Ulcer *Incidence:*5% of ulcers are malignant*Prognosis:*partial healing may occurLocation:anywhere within stomach; fundal ulcers above level of cardia are usually malignant√ ulcer location within gastric lumen, ie, not projecting beyond expected margin of stomach (profile view)√ eccentrically located ulcer within the tumor√ irregularly shaped ulcer√ shallow ulcer with width greater than depth√ nodular ulcer floor√ abrupt transition between normal mucosa + abnormal tissue at some distance (usually 2-4 cm) from ulcer edge√ rolled / rounded / shouldered edges surrounding ulcer√ nodular irregular folds approaching ulcer with fused / clubbed / amputated tips√ rigidity / lack of distensibility√ associated large irregular mass√ **Carman meniscus sign** = curvilinear lens-shaped intraluminal form of crater with convexity of crescent toward gastric wall and concavity toward gastric lumen (profile view, usually under compression) found in specific type of ulcerating carcinoma, seen only infrequently; wall aspect can also be concave / flat√ **Kirklin meniscus complex** = Carman sign (appearance of crater) + radiolucent slightly elevated rolled border

Notes:

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GASTRIC VARICES
*Cause:*portal hypertension (varices seen in 2-78%)Location:(a)esophagogastric junction (most common)(b)along lesser curvature (in 11-75% of patients with portal hypertension / cirrhosis)Feeding vessels: 1.Left gastric vein (between splenic vein + stomach)2.Short gastric veins (between spleen + fundus)3.Retrogastric vein (between splenic vein + esophagogastric junction) ● increased prevalence of portosystemic encephalopathy√ barium study: 65-89% rate of detection√ endoscopy: most practical method√ splenic portography√ hepatofugal blood flow along SMV into left gastric + splenic vein*Cx:*variceal bleeding in 3-10-36%◊Gastric varices bleed less frequently but more severely than esophageal varices!

Notes:

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GASTRIC VOLVULUS
=abnormal degree of rotation of one part of stomach around another part, usually requires >180° twisting to produce complete obstruction*Etiology:*(a)abnormality of suspensory ligaments (hepatic, splenic, colic, phrenic)(b)unusually long gastrohepatic + gastrocolic mesenteries*Usually associated with:*diaphragmatic abnormality:1.Paraesophageal hiatus hernia in 33%2.Eventration*Types:* A.ORGANOAXIAL VOLVULUSrotation around a line extending from cardia to pylorusB.MESENTEROAXIAL VOLVULUSrotation around an axis extending from lesser to greater curvature ● severe epigastric pain ● vigorous attempts to vomit without results ● inability to pass tube into stomach√ massively distended stomach in LUQ extending into chest√ incomplete / absent entrance of barium into stomach √ barium demonstrates area of twist*Cx:*intramural emphysema, perforation*DDx:*gastric atony, acute gastric dilatation, pyloric obstruction

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Notes:

Corrosive Gastritis *Agents:* (a)acid, formaldehyde ● clinically usually silentLocation:esophagus usually unharmed, severe gastric damage, duodenum may be involved (newer potent materials cause atypical distribution)(b)alkalineLocation:pylorus + antrum most frequently involvedA.ACUTE CHANGES (edema + mucosal sloughing)√ marked enlargement of gastric rugae + erosions / ulceration√ complete cessation of motor activity√ gas in portal venous system*Cx:*perforationB.CHRONIC CHANGES √ firm thick nonpliable wall√ stenotic / incontinent pylorus (if involved)√ gastric outlet obstruction (cicatrization) after 3-10 weeks

Notes:

Emphysematous Gastritis =rare but severe form of widespread phlegmonous gastritis subsequent to mucosal disruption characterized by gas in wall of stomach *Cause of mucosal disruption:* ingestion of toxic / corrosive substances (most common), alcohol abuse, trauma, gastric infarction, necrotizing enterocolitis, ulcer *Histo:*bacterial invasion of submucosa + subserosa*Organism:*hemolytic streptococcus, Clostridium welchii, E. coli, S. aureus ● explosive onset of abdominal pain, nausea, chills, fever, leukocytosis ● bloody foul-smelling emesis√ linear small gas bubbles within grossly thickened gastric wall√ may be associated with gas in portal vein*Cx:* cicatricial stenosis / sinus tract formation*Prognosis:*60-80% mortality

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Erosive Gastritis =HEMORRHAGIC GASTRITIS*Incidence:*0.5-10% of GI studies*Etiology* (in 50% without causative factors):(1)Peptic disease: emotional stress, alcohol, acid, corrosives, severe burns, anti-inflammatory agents (aspirin, steroids, phenylbutazone, indomethacin)(2)Infection: herpes simplex virus, CMV, Candida(3)Crohn disease: aphthoid ulcers identical in appearance to varioliform erosions*Histo:*epithelial defect not penetrating beyond muscularis mucosae ● 10-20% of all GI hemorrhages (usually without significant blood loss) ● vague dyspepsia, ulcerlike symptomsLocation:antrum, rarely extending into fundus; aligned on surface of gastric rugal folds√ varioliform erosion = tiny fleck of barium surrounded by radiolucent halo ("target lesion") <5 mm, usually multiple√ incomplete erosion = linear streaks / dots of barium without surrounding mound of edema / inflammation√ nodularity / scalloping of prominent antral folds√ contiguous duodenal disease may be present√ limited distensibility, poor peristalsis / atony, delayed gastric emptying

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Notes:

Phlegmonous Gastritis *Etiology:*septicemia, local abscess, postoperative stomach, complication of gastric ulcer / cancer*Organism:*Streptococcus*Path:*multiple gastric wall abscesses, which may communicate with lumen ● severe fulminating illness ● patient may vomit pusLocation:usually limited to stomach not extending beyond pylorus; submucosa is the most severely affected gastric layer√ barium dissection into submucosa + serosa

Notes:

GIARDIASIS

=overgrowth of commensal parasite Giardia lamblia*Organism:* Giardia lamblia (flagellated protozoan); often harmless contaminant of duodenum + jejunum in motile form (= trophozoite) attached to mucosa by suction disk, nonmotile form (= cyst) shed in feces; capable of pathogenic behavior with invasion of gut wall *Incidence:*1.5-2% of population in United States, infests 4-16% of inhabitants of tropical countries, found in 3-20% of children in parts of southern United States*Predisposed:*altered immune mechanism (dysgammaglobulinemia, nodular lymphoid hyperplasia of ileum)*Histo:*blunted villi (may be misdiagnosed as celiac disease especially in children), cellular infiltrate of acute + chronic inflammation in lamina propria ● abdominal pain, weight loss, failure to thrive (especially in children) ● spectrum from asymptomatic to severe debilitating diarrhea, steatorrhea (related to number of organisms) ● reduced fat absorption (simulating celiac disease)Location:most pronounced in duodenum + jejunum √ thickened distorted mucosal folds in duodenum + jejunum (mucosal edema) with normal ileum √ marked spasm + irritability with rapid change in direction + configuration of folds √ hypersecretion with blurring + indistinctness of folds √ hyperperistalsis with rapid transit time √ segmentation of barium (from motility disturbance + excess intraluminal fluid) √ ± lymphoid hyperplasia (associated with immunoglobulin deficiency state)*Dx:*(1)Detection of Giardia lamblia cysts in formed feces or trophozoites in diarrheal stools(2)Trophozoites in duodenal aspirate / jejunal biopsy*DDx:*Strongyloides / hookworm infection*Rx:*quinacrine (Atabrine®)

Notes:

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GLYCOGEN ACANTHOSIS
=benign degenerative condition with accumulation of cellular glycogen within squamous epithelial lining of esophagus; etiology unknown*Incidence:*in up to 15% of endoscoped patients*Age:*middle-aged / elderly individuals*Histo:*hyperplasia + hypertrophy of squamous mucosal cells secondary to increased glycogen; no malignant potential ● asymptomatic ● white oval mucosal plaques of 2-15 mm in diameter on otherwise normal appearing mucosaLocation:middle (common) / distal esophagus ● multiple 1-3 mm rounded nodules / plaques*Dx:*biopsy*DDx:*Candida esophagitis (lesions disappear under treatment in contrast to glycogen acanthosis)

Notes:

GRAFT-VERSUS-HOST DISEASE
=T lymphocytes from donor bone marrow cause selected epithelial damage of recipient target organsBone marrow transplantation for treatment of: leukemia, lymphoma, aplastic anemia, immunologic deficit, metabolic disorders of hematopoietic system, some metastatic disease*Incidence:*30-70% of patients with allogeneic (= donor genetically different from host) transplantTarget organs:GI tract (small bowel), skin, liver@Skin ● maculopapular rash on face, trunk, extremities@Liver ● elevation of hepatic enzymes ± liver failure@GI tract ● profuse secretory diarrhea ● abdominal cramping, fever, nausea, vomiting*Path:*severe mucosal atrophy / destruction√ shaggy fold thickening√ "ribbon bowel" = small bowel fold effacement with tubular appearance (DDx: viral enteritis, ischemia, celiac disease, radiation, soybean allergy)√ loss of haustration, spasm, edema, ulceration, granular mucosal pattern of colon (simulating ulcerative colitis)√ small bowel "cast" = prolonged coating of abnormal bowel for hours to days√ circular collections of contrast material on cross section + parallel tracks on longitudinal section√ severely decreased transit timeCT: √ abnormally enhancing thin layer of mucosa diffusely involving small + large bowel√ fluid-filled distended poorly opacified bowel (oral contrast material not given!)*Cx:*infection with opportunistic organisms, eg, Candida albicans, herpes virus, invasive fungal organisms, CMV, varicella-zoster virus, Epstein-Barr virus, hepatitis viruses, rotavirus, adenovirus, Coxsackie virus A and B, P. carinii, pneumococcus*Prognosis:*fatal in up to 15% (due to opportunistic infections)*Rx:*steroids + cyclosporine*DDx:*superinfection with enteroviruses

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HELICOBACTER PYLORI INFECTION
*Organism:*worldwide gram-negative spiral-shaped bacillus [formerly Campylobacter pylori]*Prevalence:*increasing with age; >50% of Americans >60 years of age*Path:*surface epithelial damage + inflammation with mucosal infiltration by neutrophils, plasma cells, and lymphoid nodulesLocation:gastric antrum > proximal half of stomachSite:beneath mucus layer on surface epithelial cells ● asymptomatic (vast majority) ● dyspepsia, epigastric pain√ gastritis√ thickened gastric folds√ polypoid gastritis mimicking malignant tumor√ enlarged areae gastricae√ gastric ulcer (60-80% prevalence of H. pylori)√ duodenal ulcer (90-100% prevalence of H. pylori)*Dx:*(1) Endoscopic brushings + biopsy(2)Breath test measuring urease activity after ingestion of carbon-14-labeled urea(3)Serologic test for IgG antibodies*Rx:*triple therapy (= bismuth + metronidazole + tetracycline / amoxicillin) results in 95% cure rate after 2 weeks of therapy

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HEMANGIOMA OF SMALL BOWEL

*Increased incidence in:*Turner syndrome, tuberous sclerosis, Osler-Weber-Rendu diseaseLocation:duodenum (2%), jejunum (55%), ileum (42%)√ multiple sessile compressible intraluminal filling defects√ nodular segmental mucosal abnormality√ phleboliths in intestinal wall

Notes:

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HENOCH-SCHÖNLEIN PURPURA
=most common systemic allergic vasculitis in children precipitated by bacterial / viral infection, allergies, insect sting, drugs (eg, penicillin, sulfonamides, aspirin)*Cause:*deposition of IgA-dominant immune complexes in venules, capillaries, and arterioles*Age:*children (peak age of 5 years) + adults ● most frequent manifestations: ● purpuric skin rash on legs + extensor surfaces on arms ● colicky abdominal pain + GI bleeding ● microscopic hematuria + proteinuria in 50% (from proliferative glomerulonephritis with IgA deposits demonstrated by immunofluorescence) ● often begins as an upper respiratory tract infection ● arthralgias√ thickened valvulae conniventes (due to hemorrhage + edema)*Cx:*renal insufficiency (10-20%), end-stage renal disease (5%)*Rx:*high doses of corticosteroids + azathioprine

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Notes:

External Hernia =bowel extending outside the abdominal cavity*Incidence:*95% of all herniasLocation: 1.Inguinal hernia2.Femoral hernia3.**Spigelian hernia** *Frequency:*2% of anterior abdominal hernias=acquired ventrolateral hernia through defect in aponeurosis between transverse and rectus muscle of abdomen at junction of semilunar + arcuate lines below umbilicus√ hernia sac dissects laterally to rectus abdominis muscle through a fibrous groove (= semicircular / spigelian line) √ hernia sac lies beneath an intact external oblique aponeurosis4.Petit lumbar triangle5.Obturator foramen6.Sciatic notch7.Diaphragmatic hernia (foramen of Bochdalek + Morgagni)8.Richter hernia = entrapment of antimesenteric border of bowel in hernia orifice, usually seen in older women with femoral hernias9.Perineal hernia (rare)(a)anterior perineal hernia = defect of urogenital diaphragm anterior to superficial transverse perineal m. + lateral to bulbocavernosus m. + medial to ischiocavernosus m. (only in females)(b)posterior perineal hernia = defect in levator ani m. / between levator ani m. and coccygeus m. posterior to superficial transverse perineal m.√ defecating proctography

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Notes:

Internal Hernia *Incidence:*5% of all hernias, responsible for <1% of mechanical small bowel obstruction*Classification of hernias:* (a) retroperitoneal: usually congenital containing a hernial sac 1.paraduodenal (ligament of Treitz)2.foramen of Winslow3.intersigmoid4.pericecal / ileocolic5.supravesical(b)anteperitoneal:small group of hernias without a peritoneal sac 1.transmesenteric (transverse / sigmoid mesocolon)2.transomental3.pelvic (including broad ligament) A.PARADUODENAL HERNIA (53%)(a)through fossa of Landzert on left side (3/4)√ lateral to 4th portion of duodenum and behind descending + transverse mesocolon(b)through fossa of Waldeyer on right side (1/4)√ caudal to SMA and inferior to 3rd portion of duodenumB.LESSER SAC HERNIA (<10%)through foramen of Winslow in retrogastric location Invaginated gut: ileum > jejunum, cecum, appendix, ascending colon, Meckel diverticulum, gallbladder, greater omentum C.HERNIA THROUGH BROAD LIGAMENT (very rare)after laceration / fenestration from surgery or during pregnancy

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Notes:

Hiatal Hernia *Associated with:*diverticulosis (25%), reflux esophagitis (25%), duodenal ulcer (20%), gallstones (18%) ***Sliding Hiatal Hernia*** (99%) =AXIAL HERNIA = CONCENTRIC HERNIA=esophagogastric junction remains in chest with portion of peritoneal sac forming part of wall of hernia*Etiology:*rupture of phrenicoesophageal membrane due to repetitive stretching with swallowing*Incidence:*increasing with age√ reducible in erect position√ epiphrenic bulge = entire vestibule + sleeve of stomach are intrathoracic√ distance between B ring (if visible) and hiatal margin >2 cm√ peristalsis ceases above hiatus (end of peristaltic wave delineates esophagogastric junction)√ tortuous esophagus having an eccentric junction with hernia√ numerous coarse thick gastric folds within suprahiatal pouch (>6 longitudinal folds)√ ± gastroesophageal refluxCT: √ dehiscence of diaphragmatic crura >15 mm√ pseudomass within / above esophageal hiatus√ increase in fat surrounding distal esophagus(= herniation of omentum through phrenicoesophageal ligament) *DDx:*normal temporary cephalad motion of esophagogastric junction by 1-2 cm into chest due to contraction of longitudinal muscle during esophageal peristalsis ***Paraesophageal Hernia*** (1%) =ROLLING HIATAL HERNIA = PARAHIATAL HERNIA = portion of stomach superiorly displaced into thorax with esophagogastric junction remaining in subdiaphragmatic position√ cardia in normal position√ herniation of portion of stomach anterior to esophagus√ frequently nonreducible√ may be associated with gastric ulcer of lesser curvature at level of diaphragmatic hiatus ***Totally Intrathoracic Stomach*** =defect in central tendon of diaphragm in combination with slight volvulus in transverse axis of stomach behind heart√ cardia may be intrathoracic (usually) / subdiaphragmatic√ great gastric curvature either on right / left side ***Congenitally Short Esophagus*** (not true hernia, very rare) =gastric ectopy by lack of lengthening of esophagus√ nonreducible intrathoracic gastric segment (in erect / supine position)√ cylindrical / round intrathoracic segment with large sinuous folds√ short straight esophagus√ circular narrowing at gastroesophageal junction, frequently with ulcer√ gastroesophageal reflux

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Umbilical Hernia =protrusion of abdominal contents / fat into anterior abdominal wall via umbilical ring*Prevalence:*4% of all hernias; M<F*Cause:*failed closure of umbilical ring, obesity, multiple pregnancies, intra-abdominal masses, liver failure, increased intra-abdominal pressure, weak abdominal wall√ may contain fat / small bowel / colon√ herniation of antimesenteric border of intestine (Richter hernia)√ Meckel diverticulum in hernial sac (Littré hernia)*Cx:*strangulation, incarceration*DDx:*paraumbilical, spigelian, epigastric, incisional hernia

Notes:

HIRSCHSPRUNG DISEASE
=AGANGLIONOSIS OF THE COLON = AGANGLIONIC MEGACOLON=absence of parasympathetic ganglia in muscle (Meissner plexus) + submucosal layers (Auerbach plexus) secondary to an arrest of craniocaudal migration of neuroblasts along vagal trunks before 12th week leading to relaxation failure of the aganglionic segment*Incidence:* 1:5,000-8,000 livebirths; usually sporadic; familial in 4% *Age:* full-term infant during first 6 weeks of life (70-80%); M:F = 4-9:1; extremely rare in premature infants *Associated with:*trisomy 21 (2%)Location:at varying distances proximal to anus, usually rectosigmoid(a)short segment disease(80%)(b)long segment disease(15%)(c)total colonic aganglionosis(5%)(d)skip aganglionosis = sparing of rectum (very rare) ● failure to pass meconium within first 24 hours of life ● intermittent constipation + paradoxical diarrhea (25%) ● rectal manometry with absence of spike activity√ "transition zone" = aganglionic segment appears normal in size√ dilatation of large + small bowel aborally from transition zone√ marked retention of barium on delayed films after 24 hours√ normal-appearing rectum in 33%√ 10- to 15-cm segment of persistent corrugated / convoluted rectum (= abnormal uncoordinated contractions of the aganglionic portion of colon) in 31% (DDx: colitis, milk allergy, normal intermittent spasm of rectum)N.B.:avoid digital exam / cleansing enema prior to radiographic studies!OB-US: √ dilated small bowel / dilated colon*Cx:*(1)Necrotizing enterocolitis(2)Cecal perforation (secondary to stasis, distension, ischemia)(3)Obstructive uropathy*Dx:*suction mucosal biopsy of rectum (increased acetylcholinesterase activity)*Rx:*(1)Swenson pull-through procedure(2)Duhamel operation(3)Soave procedure

Notes:

HODGKIN DISEASE

*Incidence:*0.75% of all cancers diagnosed each year*Age:*bimodal peaks at age 25-30 years and 75-80 years*Histo:*Reed-Sternberg cell = binucleate cell with prominent centrally located nucleolus (1)Lymphocyte predominance (5%)=abundance of normal-appearing lymphocytes + relative paucity of abnormal cells; often diagnosed in younger people; frequently early stage; systemic symptoms are uncommon; most favorable natural history(2)Nodular sclerosis (78%)=lymph nodes traversed by broad bands of birefringent collagen separating nodules, which consist of normal lymphocytes, eosinophils, plasma cells, and histiocytes; most common subtype; typically mediastinal involvement; 1/3 with systemic symptoms(3)Mixed cellularity (17%)=diffuse effacement of lymph nodes with lymphocytes, eosinophils, plasma cells + relative abundance of atypical mononuclear and Reed-Sternberg cells; more commonly advanced stage at presentation and older age(4)Lymphocyte depletion (1%)=paucity of normal-appearing lymphocytes + abundance of abnormal mononuclear and Reed-Sternberg cells; least common subtype with worst prognosis; associated with advanced stage and systemic symptomsSTAGE Iinvolvement of single lymph node regionIIinvolvement of $\geq$ 2 lymph node regions on same side of diaphragmIIIlymph node involvement on both sides of diaphragmIVdiffuse / disseminated involvement of $\geq$ 1 extralymphatic organs / tissues ± associated lymph node involvementE=extralymphatic siteS=splenic involvementA=absence of fever, night sweats, >10% weight loss in past 6 monthsB=presence of fever, night sweats, >10% weight loss in past 6 months ● painless lymphadenopathy ● alcohol-induced pain ● unexplained fevers, night sweat, weight loss ● generalized pruritusLocation:intestinal involvement uncommon (10-15%); duodenum + jejunum (67%); terminal ileum (20%)√ narrow rigid obstructive lesion√ abundance of desmoplastic reaction (DDx from NHL)√ infiltrating (60%); polypoid (26%); ulcerated (14%)*Prognosis:* excellent for isolated / localized disease

Notes:

HYPERPLASTIC POLYP OF COLON
=intestinal metaplasia consisting of mucous glands lined by a single layer of columnar epithelium; NO malignant potential*Path:*infolding of epithelium into the glandular lumenLocation:rectum√ usually <5 mm in diameter

Notes:

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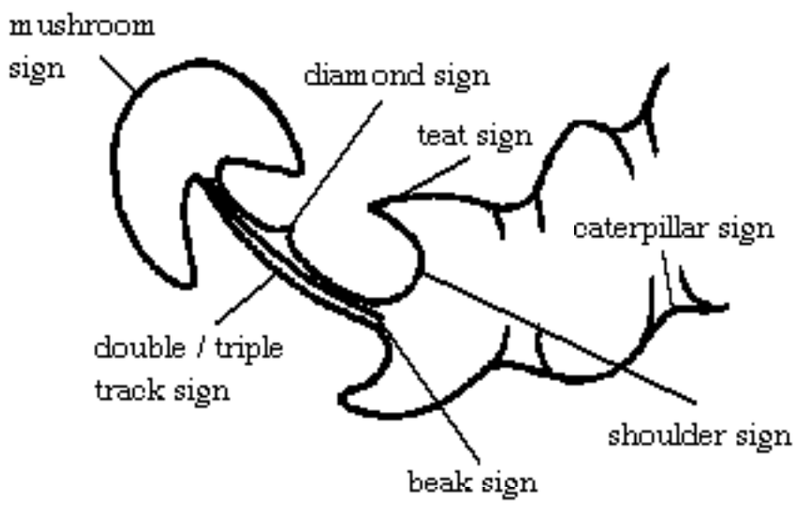
HYPERTROPHIC PYLORIC STENOSIS
=idiopathic hypertrophy and hyperplasia of circular muscle fibers of pylorus with proximal extension into gastric antrum*Incidence:*3:1,000; M:F = 4-5:1*Etiology:*inherited as a dominant polygenic trait; increased incidence in firstborn boys; acquired rather than congenital condition

Infantile Form Of Hypertrophic Pyloric Stenosis Adult Form Of Hypertrophic Pyloric Stenosis Focal Pyloric Hypertrophy

Notes:

Infantile Form Of Hypertrophic Pyloric Stenosis *Age:*manifestation at 2-8 weeks of life ● nonbilious projectile vomiting (sour formula / clear gastric contents) with progression over a period of several weeks after birth (15-20%) ● positive family history ● palpable olive-shaped mass (80% sensitive in experienced hands, up to 14% false positive) ● nasogastric aspirate >10 mL (92% sensitive, 86% specific)UGI (95% sensitivity): Precautions:(1)empty stomach via nasogastric tube before study(2)remove contrast at end of study√ pyloric wall thickness >10 mm√ elongation + narrowing of pyloric canal (2-4 cm in length)√ "double / triple track sign" = crowding of mucosal folds in pyloric channel√ "string sign" = passing of small barium streak through pyloric channel√ Twining recess = "diamond sign" = transient triangular tentlike cleft / niche in midportion of pyloric canal with apex pointing inferiorly secondary to mucosal bulging between two separated hypertrophied muscle bundles on the greater curvature side within pyloric channel√ "pyloric teat" = outpouching along lesser curvature due to disruption of antral peristalsis√ "antral beaking" = mass impression upon antrum with streak of barium pointing toward pyloric channel √ Kirklin sign = "mushroom sign" = indentation of base of bulb (in 50%)√ gastric distension with fluid√ active gastric hyperperistalsis√ "caterpillar sign" = gastric hyperperistaltic waves



US: √ "target sign" = hypoechoic ring of hypertrophied pyloric muscle around echogenic mucosa centrally on cross-section√ "cervix sign" = indentation of muscle mass on fluid-filled antrum on longitudinal section√ "antral nipple sign" = redundant pyloric channel mucosa protruding into gastric antrum√ pyloric volume >1.4 cm^3 (= 1/4 ÷ x [maximum pyloric diameter]^2 x pyloric length); most criteria independent of contracted or relaxed state (33% false negative)√ pyloric length (mm) + 3.64 x muscle thickness (mm) > 25√ pyloric muscle wall thickness ≥3 mm√ pyloric transverse diameter ≥13 mm with pyloric channel closed√ elongated pyloric canal ≥17 mm in length√ exaggerated peristaltic waves√ delayed gastric emptying of fluid into duodenum*Cx:*hypochloremic metabolic alkalosis*DDx:* 1.**Infantile pylorospasm** √ muscle thickness between 1.5 and 3 mm√ variable caliber of antral narrowing√ antral peristalsis√ delayed gastric emptying√ elongation of pylorus*Prognosis:*resolves in several days / ? early stage of evolving pyloric stenosis*Rx:*effective with metoclopramide hydrochloride2.Milk allergy3.Eosinophilic gastroenteritis

Notes:

Adult Form Of Hypertrophic Pyloric Stenosis (secondary to mild infantile form) ● acute obstructive symptoms uncommon ● nausea, intermittent vomiting ● postprandial distress, heartburn*Associated with:* (1) peptic ulcer disease (in 50-74%) (prolonged gastrin production secondary to stasis of food)(2)chronic gastritis (54%)√ persistent elongation (2-4 cm) + concentric narrowing of pyloric channel√ parallel + preserved mucosal folds√ antispasmodics show no effect on narrowing√ proximal benign ulcer (74%), usually near incisura

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Notes:

Focal Pyloric Hypertrophy =TORUS HYPERPLASIA=localized muscle hypertrophy on the lesser curvature=milder atypical form of HPS√ flattening of distal lesser curvature

Notes:

IMPERFORATE ANUS

*Prevalence:*1:5,000 live births A.LOW ANOMALY (55%)=bowel has passed through levator sling ● fistula to perineum / vulva*Rx:*readily reparableB.INTERMEDIATE DEFECT (least common)=bowel ends within levator muscle as a result of abnormality in posterior migration of rectum ● fistula opening low in vagina / vestibule*Rx:*2- / 3-stage operationC.HIGH ANOMALY=bowel ends above levator sling; M > F ● fistulous connection to perineum / vagina / posterior urethra (air in bladder in males; air in vagina in females)*Cx:*associated malformations more common + more severe*Rx:*multiple surgical procedures √ distance between rectal air and skin will not accurately outline the extent of atretic rectum and anus (varying length during crying with increase in abdominal pressure + contraction of levator ani muscle)US: √ ≤15 mm distance between anal dimple + distal rectal pouch on transperineal images indicates low lesionOB-US (earliest detection by 20-29 weeks GA): ● absent / low disaccharidase level in amniotic fluid√ dilated colon in lower pelvis with U- / S-shaped configuration ± intraluminal calcifications√ normal amniotic fluid (unless also TE fistula)√ absence of anal characteristics (= hypoechoic circular rim with central echogenic stripe)

Notes:

INTESTINAL LYMPHANGIECTASIA

A.CONGENITAL LYMPHANGIECTASIA = PRIMARY PROTEIN-LOSING ENTEROPATHY=generalized congenital malformation of lymphatic system with atresia of the thoracic duct + gross dilatation of small bowel lymphatics; usually sporadic; may be inherited*Age:*presentation before 30 years ● asymmetric generalized lymphedema (due to protein-losing enteropathy with hypoproteinemia) ● chylous pleural effusions (45%) ● diarrhea (60%), steatorrhea (20%) ● vomiting (15%) ● abdominal pain (15%) + distension ● decreased albumin + globulin ● lymphocytopenia (90%) ● decreased serum fibrinogen, transferrin, ceruloplasminB.ACQUIRED LYMPHANGIECTASIA*Causes leading to dilatation of intestinal lymphatics:* 1.Mesenteric adenitis2.Retroperitoneal fibrosis3.Diffuse small bowel lymphoma 4.Pancreatitis5.Pericardial effusion with obstruction of thoracic duct ● peripheral edema / anasarca (KEY SYMPTOM) ● chylous + serous effusion ● diarrhea, vomiting, abdominal pain, malabsorption, steatorrhea ● hypoproteinemia secondary to protein loss into intestinal lumen *Path:*dilatation of lymph vessels in mucosa + submucosa + abundance of foamy fat-staining macrophages (negative for PAS)√ diffuse symmetric marked enlargement of folds in jejunum + ileum (due to dilated intestinal lymphatics + hypoproteinemic edema)√ slight separation + rigidity of folds√ dilution of barium column (considerable increase in intestinal secretions from malabsorption)√ no / mild dilatation of bowelLymphangiogram (not always diagnostic): √ hypoplasia of lower extremity lymphatics√ occlusion of thoracic duct / large tortuous thoracic duct√ obstruction of cisterna chyli with backflow into mesenteric + intestinal lymphatics√ hypoplastic lymph nodes*Dx:*small bowel biopsy (dilated lymphatics in lamina propria + vascular core)*Rx:*low-fat diet with medium-chain triglycerides (direct absorption into portal venous system)*DDx:*(1)Whipple disease (more segmentation + fragmentation, wild folds)(2)Amyloidosis (edema + secretions usually absent)(3)Hypoalbuminemia (less pronounced symmetric thickening of folds, less prominent secretions)

Notes:

INTRALUMINAL DUODENAL DIVERTICULUM

=congenital lesion secondary to elongation of an incomplete duodenal diaphragm*Age at presentation:* in young adult ● easy satiety ● vomiting ● upper abdominal cramping painLocation: 2nd-3rd portion of duodenum√ barium-filled sac within duodenal lumen (pathognomonic picture) = "windsock, comma, teardrop" appearance√ anchored to the lateral wall of the duodenum√ "halo" sign = duodenal mucosa covers outer + inner wall of diverticulum

Notes:

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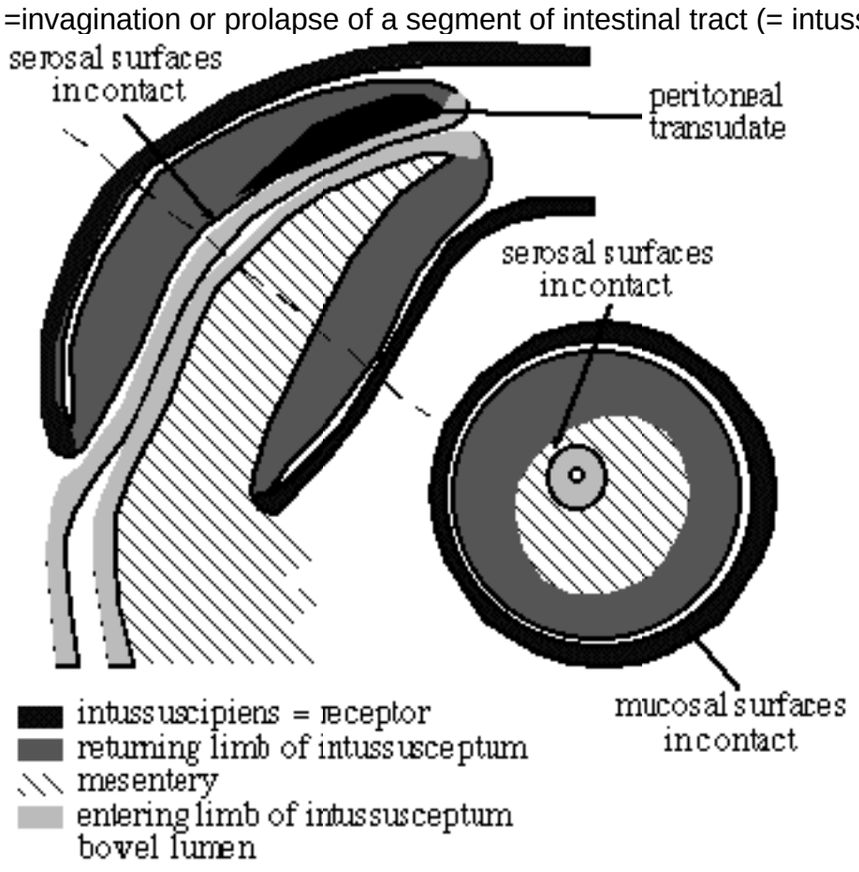
INTRAMURAL ESOPHAGEAL RUPTURE

=DISSECTING INTRAMURAL HEMATOMA=mucosal tear with dissecting hemorrhage into submucosa and involvement of venous plexus ● hematemesis√ intramural hematoma simulates retained solid material within lumen√ "mucosal stripe sign" = dissected mucosa floating within lumen

Notes:

INTUSSUSCEPTION
=invagination or prolapse of a segment of intestinal tract (= intussusceptum) into the lumen of adjacent intestine (= intussuscipiens)



A.IN CHILDREN (94%)Most common abdominal emergency of early childhood, leading cause of acquired bowel obstruction in childhood *Etiology:* (1)idiopathic (over 95%): mucosal edema + lymphoid hyperplasia following viral gastroenteritis; predominantly at ileocecal valve(2)lead point (5%): Meckel diverticulum (most common), lymphosarcoma, polyp, enterogenous cyst, duplication cyst, suture granuloma, appendiceal inflammation, Henoch-Schönlein purpura, inspissated meconium; usually >6 years of age*Age:*peak incidence between 6 months and 2 years; 3-9 months (40%); <1 year (50%); <2 years (75%); >3 years (<10%); M:F = 2:1 ● abrupt onset of violent crampy pain (90%), vomiting (85%) ● abdominal mass (60%) ● "currant jelly" bloody stools (60%)Location:ileocolic (75-95%) > ileoileal (4%) > colocolic*Cx:*vascular compromise secondary to incorporation of mesentery (hemorrhage, infarction, acute inflammation)B.IN ADULTS (6%)*Etiology:* (1)specific cause (80%): benign tumor (1/3), malignant tumor (1/5), lipoma, Meckel diverticulum, prolapsed gastric mucosa, aberrant pancreas, adhesions, foreign body, feeding tube, chronic ulcer (TB, typhoid), prior gastroenteritis, gastroenterostomy, traumawithout anatomic lead point: celiac disease, scleroderma, Whipple disease, fasting, anxiety, agonal state (2)idiopathic (20%) ● recurrent episodes of colicky pain, nausea, vomitingLocation:ileoileal (40%) > ileocolic (13%) Plain film (no abnormality in 25%): √ abdominal soft-tissue mass (50-60%), usually in RUQ√ loss of inferior hepatic margin√ small bowel obstruction (25%) with nipplelike termination of gas shadowAntegrade barium study: √ "coil spring" appearance√ beaklike abrupt narrowing of barium column demonstrating a central channelRetrograde barium study: √ convex intracolic mass + "coiled spring" patternUS (close to 100% sensitive): √ "doughnut / target / bulls eye sign" (on transverse scan) = concentric rings of alternating hypoechoic + hyperechoic layers (= intussuscipiens) with central hyperechoic portion (= mesentery of intussusceptum)√ "pseudokidney / sandwich / hay fork sign" (on longitudinal scan) = hypoechoic layers on each side of echogenic center of mesenteric fat√ peritoneal fluid trapped inside intussusception (associated with irreducibility + ischemia)√ color Doppler demonstrates mesenteric vessels dragged between entering + returning wall of intussusceptum◊Absence of blood flow suggests bowel necrosis!CT: √ "multiple concentric rings" = 3 concentric cylinders (central cylinder = canal + wall of intussusceptum; middle cylinder = crescent of mesenteric fat; outer cylinder = returning intussusceptum + intussuscipiens)√ proximal obstruction HYDROSTATIC / PNEUMATIC REDUCTION◊<1% mortality if reduction occurs <24 hours after onset!*Overall success rate:*70-85%*Contraindications:*pneumoperitoneum, peritonitis, hypovolemic shockTechnique: (1)Sedation with morphine sulfate (0.2 mg/kg IM) / fentanyl citrate IV (straining increases intraluminal pressure of distended colon)(2)Anal seal with 24-F Foley catheter + balloon inflation to size equal to interpediculate distance of L5; balloon pulled down to levator sling; taped to buttocks; both buttocks firmly taped together(3)60% wt/vol barium sulfate with container between 24-36 inches above level of anus(4)Maximally 3 attempts for 3 minutes each(5)Manual manipulation increases colonic pressure(6)Reduction should be accomplished within 10 minutes(7)Extensive reflux into small bowel desirable to exclude residual ileoileal intussusception**"Rule of 3s"**: (1)3.5 feet (105 cm) above table (=120 mm Hg)(2)3 attempts(3)3 minutes between attempts (delay allows venous congestion + edema to subside)Alternative medium: (1)1:4 Gastrografin®-water solution raised to a height of 5 feet (150 cm)(2)air: delivers higher intracolonic pressures, faster, less fluoroscopic time, smaller tears, less contamination of peritoneal cavity*Cx:*perforation (0.4-2%; colonic bursting pressure ~200 mm Hg); reduction of nonviable bowel; incomplete reduction; missed lead point*Prognosis:*3.5-10% rate of recurrence

Notes:

ISCHEMIC COLITIS
=nonocclusive vascular disease within the territory of the inferior mesenteric artery characterized by acute onset + rapid clinical and radiographic evolutionary changes*Etiology:*diminished blood flow within bowel wall (mucosa + submucosa most sensitive to ischemia); major mesenteric vessels usually patent*Precipitating factors:* (a)bowel obstruction: volvulus, carcinoma (proximal bowel segment affected)(b)thrombosis: cardiovascular disease, collagen vascular disease, sickle cell disease, hemolytic-uremic syndrome, oral contraceptives(c)trauma: history of aortoiliac reconstruction (2%) with ligation of IMA*mnemonic:*"VINTS"**V**asculitis **I**ncarceration (hernia, volvulus) **N**onocclusive ischemia (shock, CHF) **T**hrombosis (atherosclerosis, emboli, polycythemia vera, hyperviscosity) **S**pontaneous *Age:*>50 years ● abrupt onset of lower abdominal pain + rectal bleeding ● abdominal tenderness, diarrheaLocation:left colon (90%), splenic flexure = Griffith point (80%) + sigmoid ("watershed areas"), rectum sparedPlain film (usually normal): √ segmental thumbprinting = marginal indentations on mesenteric side (rare finding on plain film)BE (in 90% abnormal): ◊Single contrast may efface thumbprinting, but double contrast overall is more sensitive!√ thumbprinting (75%) due to submucosal hemorrhage + edema√ transverse ridging = markedly enlarged mucosal folds (spasm), some wall pliability is preserved√ serrated mucosa = inflammatory edema + superficial longitudinal / circumferential ulceration√ deep penetrating ulcers (late)CT: √ symmetric / lobulated segmental thickening of colonic wall√ irregular narrowed atonic lumen (= thumbprinting)√ curvilinear collection of intramural gas√ portal + mesenteric venous air√ blood clot in SMA / SMVUS: √ absence / barely visible color flow√ absence of arterial signals√ nonstratified (= indistinct layers) thickened bowel wall >3 mmAngio (findings similar to inflammatory disease): √ normal / slightly attenuated arterial supply√ mild acceleration of arteriovenous transit time√ small tortuous ectatic draining veins*Prognosis:* (1)Transient ischemia = complete resolution within 1-3 months(2)Stricturing ischemia = incomplete delayed healing√ narrowed foldless segment of several cm in length with smooth tapering margins(3)Gangrene with necrosis + perforation (extremely uncommon)

Notes:

JEJUNOILEAL DIVERTICULAR DISEASE
=JEJUNAL DIVERTICULOSIS=rarest form of gastrointestinal diverticular disease*Cause:*acquired mucosal herniation (= pulsion diverticulum) *Incidence:*0.5-2.3% on UGI; 0.3-4.5% of autopsy series; M > F*Age:*6th-7th decadesLocation:80% in jejunum, 15% in ileum (usually solitary), 5% in jejunum + ileumSite:on mesenteric border near entrance of vasa recti ● intermittent upper abdominal pain, flatulence, episodes of diarrhea (30%)Plain film: √ air-fluid levels in multiple diverticula√ slight dilatation of intestinal loops in area of diverticulaBE: √ may not fill (narrow neck / stagnant secretions)√ trapped barium on delayed film after 24 hours*Cx:* (1)Blind loop syndrome with bacterial overgrowth ● steatorrhea, diarrhea, malabsorption, weight loss ● megaloblastic anemia (overgrowth of coliform bacteria leads to deconjugation of bile acids + intraluminal metabolism of vitamin B12)(2)Free perforation = leading cause of pneumo-peritoneum without peritonitis (21-40% mortality)(3)Hemorrhage (few cases)(4)Diverticulitis(5)Intestinal obstruction

Notes:

JUVENILE POLYPOSIS

=rare autosomal dominant disease with variable penetrance characterized by development of multiple (>5) juvenile polyps in GI tract◊Most common familial / nonfamilial colonic polyp in children (75%)!*Categories:* A.Juvenile polyposis of infancy*Age:*4-6 years (range 1-10 years); M:F = 3:2 ● protein-losing enteropathy, diarrhea, hemorrhage ● rectal prolapse√ intussusceptionB.Colonic & generalized juvenile polyposis*Age:*in 85% manifested by 20 years of age ● prolapse of polyp / rectum ● rectal bleeding, anemia *Path:*hamartomatous polyps; adenomas may coexist*Histo:*little / no smooth muscle; hyperplasia of mucous glands; retention cysts develop with obstruction of gland orifices (multiple mucin-filled spaces); edematous inflamed expanded lamina propria*DDx:*familial adenomatous polyposis, Peutz-Jeghers syndrome ● rectal bleeding (95%) most commonly as intermittent bright red hematochezia ● anemia, pain ● diarrhea, constipation ● abdominal pain (from intussusception) ● rectal prolapse (rare)Location:rectosigmoid (80%); rare in small bowel + stomach; not in esophagus√ solitary polyp (75%); multiple polyps (1/3) of smooth round contour√ lesion of pinpoint size / up to several cm in diameter√ invariably on stalk of variable length*Dx:*(1)any number of polyps with family history(2)polyps throughout the GI tract(3)>5-10 polyps in colon*Cx:*colorectal cancer by 35 years of age (in 15%)*DDx:*solitary juvenile polyps (<5 polyps, 1% prevalence in children)

Notes:

KAPOSI SARCOMA
=multicentric malignant neoplasm originating from endothelial cells of lymphatic / blood vessels*Cause:*HIV regulatory protein (trans-activator target [TAT]) important for viral replication is thought to cause proliferation of Kaposi sarcoma cells*Incidence:*most common AIDS-related neoplasm (10-20-34%); in 51% of homosexual / bisexual men with AIDS; rare in hemophiliacs;M:F = 50:1 *Histo:*proliferation of spindle cells with numerous extravasated RBCs located in clefts between stromal cells @Skin (most frequent site)@Lymph nodes (2nd most frequent site):√ abdominal + pelvic lymphadenopathy with high contrast-enhancement (secondary to vascularity)Associated with high frequency of GI tract involvement @GI tract (40%, 3rd most frequent site): ● usually clinically silent ● concurrent with / after cutaneous disease◊GI tract is the only site of involvement in <5%!Location:anywhere within GI tract; often multifocal√ thickened nodular folds√ multiple submucosal nodules ± central umbilication√ polypoidal mass√ infiltrating lesion@Liver (34% at autopsy)infrequently contributes to morbidity + mortality √ multiple 5-12 mm nodules hyperechoic on US, hypoattenuating on NECT/CECT indistinguishable from multiple hemangiomas*DDx:*metastatic disease, fungal microabscesses, multiple areas of bacillary angiomatosis (= swollen venous lakes in liver)@Lung (18-47% of patients with cutaneous sarcoma):=late complication of AIDSSite:peribronchial + perivascular axial interstitium (91%); middle / lower lung zones (92%)√ coarsening of bronchovascular bundles√ tram track opacities√ peribronchial cuffing√ septal lines (38-71%)√ central perihilar coalescent consolidation ± air bronchograms in 45% (= confluent tumor)√ small (50%) / large (28%) pulmonary nodules(= tumor proliferation extending into parenchyma) √ pleural effusion (33-67%), chylothorax (rare)√ moderate lymphadenopathy (16%)@Lower extremities√ lytic cortical lesion√ subcutaneous nodules*Dx:*visualization + biopsy of mass with red-purple color

Notes:

LADD BANDS
=congenital peritoneal bands extending from cecum / hepatic flexure over anterior surface of 2nd / 3rd portion of duodenum causing duodenal obstruction at its 2nd portion (even without volvulus)*Associated with:* malrotation

Notes:

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LEIOMYOMA
Location:2/3 occur in stomach*Path:*arising from muscularis propria / submucosa / muscularis mucosae / smooth muscle of blood vessels within wall of viscus*Histo:*intersecting bands of muscle + fibrous tissue in a well-defined capsule*DDx:*fibroma, neurofibroma, hemangioma

Esophageal Leiomyomatosis Leiomyoma Of Esophagus Leiomyoma Of Small Bowel Leiomyoma Of Stomach

Notes:

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Esophageal Leiomyomatosis *Age:*6-18 (mean of 11) years; M >F*Cause:*(1)sporadic (50%)(2)familial disease (20%): leiomyomas of uterus, vulva, tracheobronchial tree, small bowel, rectum(3)Alport syndrome (30%) = nephritis, high-frequency sensorineural hearing loss, congenital cataractSite:distal third / half of esophagus ± extension into proximal stomach ● slowly progressive dysphagia over years√ smooth tapered narrowing of distal esophagus over an average length of 6 cm√ decreased / absent esophageal peristalsis√ smooth relatively symmetric defect at cardia (from thickened muscle bulging into gastric fundus)CT: √ marked circumferential wall thickening of up to 4 cm from mass with relatively low soft-tissue attenuation*DDx:*(1)primary achalasia (shorter narrowed segment)(2)secondary achalasia (older individual, recent onset of dysphagia)(3)stricture from reflux esophagitis(4)idiopathic muscular hypertrophy of the esophagus (in late adulthood, corkscrew appearance of esophagus with nonperistaltic contractions, cardia rarely involved)

Notes:

Leiomyoma Of Esophagus ◊Most common benign tumor of esophagus!*Incidence:*1:1,119 (autopsy study);50% of all benign esophageal tumors*Age:*young adults; 3% in children; M > F ● usually asymptomatic (due to slow growth) ● dysphagia, odynophagia, dyspepsia ● hematemesis if large (rare)Site:frequently lower + mid 1/3 of esophagus;intramural; multiple leiomyomas in 3-4%√ 2-15 cm large smooth well-defined intramural mass causing eccentric thickening of wall + deformity of lumen√ may have coarse calcifications◊Leiomyoma is the only calcifying esophageal tumor!√ ulceration uncommonCT: √ uniform soft-tissue density√ diffuse contrast enhancementCAVE:high percentage misdiagnosed as extrinsic lesion!

Notes:

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Leiomyoma Of Small Bowel Most common benign tumor of small bowel Location:duodenum (21%), jejunum (48%), ileum (31%); single in 97%Site:mainly serosal (50%), mainly intraluminal (20%), intramural (10%)Size:<5 cm (50%), 5-10 cm (25%), >10 cm (25%)Ψ small ulcer + large barium-filled cavity (central necrosis + communication with lumen)Ψ hypervascular

Notes:

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Leiomyoma Of Stomach 2nd most common benign gastric tumor (after gastric polyp), most common of calcified benign tumors Location: pars media (39%), antrum (26%), pylorus (12%), fundus (12%), cardia (10%)Site:intraluminal submucosal (60%), exophytic subserosal (35%), combined intramural-extramural dumbbell type mass (5%)√ average size of 4.5 cm√ ovoid mass with smooth margin + smooth surface (most frequently)√ forms right angle with gastric wall√ ulcerated in 50%√ pedunculated intraluminal tumor in submucosal growth (rare)√ "iceberg phenomenon" = large extraluminal component in subserosal growth√ calcifies in 4%*Cx:*(1)Hemorrhage (acute / chronic)(2)Obstruction (tumor bulk / intussusception)(3)Infection(4)Fistulization / perforation(5)Malignant degeneration (benign:malignant = 3:1)

Notes:

Leiomyosarcoma Of Small Bowel Location:duodenum (26%), jejunum (34%), ileum (40%)√ usually >6 cm in size√ nodular mass: intraluminal (10%), intraluminal pedunculated (5%), intramural (15%), chiefly extrinsic (66%)√ mucosa may be stretched + ulcerated (50%)√ may show central ulcer pit / fistula communicating with a large necrotic center√ intussusception

Notes:

Leiomyosarcoma Of Stomach *Incidence:*0.1-3% of all gastric malignancies*Age:*10-73 years; M > F*Histo:*pleomorphism, hypercellularity, mitotic figures, cystic degeneration, necrosis ● GI bleeding (from ulceration) ● obstruction*Metastases:* (a)hematogenous to liver, lung, peritoneum; rarely to bone + soft tissue(b)direct extension into omentum, retroperitoneum(c)lymph nodes (rare)Location:anterior / posterior wall of body of stomach√ average size of 12 cm√ intramural mass√ may be pedunculated√ large masses tend to be exogastric√ very frequently ulceratedCT: √ lobulated irregular outline√ central zones of low density (necrosis with liquefaction) √ air / positive contrast within tumor (= ulceration)√ dystrophic calcifications

Notes:

Carney Syndrome Triad of(1)Gastric epitheloid leiomyosarcoma(2)Functioning extraadrenal paraganglioma(3)Pulmonary chondromas*Incidence:*24 patients reported; M:F = 1:11

Notes:

LIPOMA
Most common submucosal tumor in colon *Incidence:*in colon in 0.25% (autopsy)Location:colon (particularly cecum + ascending colon) > duodenum > ileum > stomach > jejunum > esophagus ● asymptomatic ● crampy pain, hemorrhage (rare)√ smooth, sharply outlined, round / ovoid globular mass of 1-3 cm in diameter√ short thick pedicle in 1/3 caused by repeated peristaltic activity (prone to intussuscept)√ marked radiolucency√ change in shape + size on compression due to softness√ "squeeze sign" = sausage-shaped mass on postevacuation radiographsCT: √ sharply defined intramural mass of fat density*Cx:*intussusception (rare) / ulceration (rare)*Prognosis:*NO liposarcomatous degeneration

Notes:

LYMPHANGIOMA
=congenital malformation of lymphatic vessels*Path:*usually multiloculated large thin-walled cystic mass with chylous / serous / hemorrhagic fluid contentsLocation:mesentery√ proximal bowel dilatation (in partial bowel obstruction)US: √ multiseptated cystic mass with lobules√ fluid anechoic / with internal echoes / sedimentationCT: √ cystic mass with contents of water- to fat-densityMR: √ serous contents: hypointense on T1WI + hyperintense on T2WI√ hemorrhage / fat: hyperintense on T1WI + T2WI*Rx:*surgery (difficult due to intimate attachment to bowel wall)

Notes:

LYMPHOGRANULOMA VENEREUM
=LGV = sexually transmitted disease caused by virus Chlamydia trachomatis producing a nonspecific granulomatous inflammatory response in infected mucosa (mononuclear cells + macrophages), perirectal lymphatic invasionLocation:rectum, may extend to sigmoid + descending colonM:F = 3.4:1√ narrowing + shortening + straightening of rectosigmoid√ widening of retrorectal space√ irregularity of mucosa + ulcerations√ paracolic abscess√ fistula to pericolic area, rectum, vagina (common)*Rx:*tetracyclines effective in acute phase before scarring has occurred

Notes:

LYMPHOID HYPERPLASIA

*Incidence:*normal variant in 13% of BE examinations*Histo:*hyperplastic lymph follicles in lamina propria (Peyer patches), probably compensatory attempt for immunoglobulin deficiency*Etiology:* (1)Normal in child / young adult(2)Self-limiting local / systemic inflammation / infection / allergy(3)May be related to immunodeficiency / dysgammaglobulinemia with small bowel involvement*Age:*(a)generally in children <2 years(b)in adults invariably associated with late onset immunoglobulin deficiency (IgA, IgM)*Associated with:*splenomegaly, large tonsils, eczematous dermatitis, achlorhydria, pernicious anemia, acute pancreatitis, colonic carcinoma*At risk for:* (1)**Good syndrome** (10%) =gastric carcinoma + benign thymoma + lymphoid hyperplasia(2)Respiratory infections(3)Giardia lamblia infection (90%)(4)Functional thyroid abnormalitiesLocation:primarily jejunum, may involve entire small bowel, ascending colon + hepatic flexure, seldom in sigmoid / rectum ● malabsorption (diarrhea + steatorrhea) ● low serum concentrations of IgA, IgG, IgM√ mucosa studded with innumerable 1-3 mm small uniform polypoid lesions√ lesions may be umbilicated (uncommon)

Notes:

LYMPHOMA OF GASTROINTESTINAL TRACT

Classification: A.PRIMARY LYMPHOMA OF BOWEL(a) localized (b) diffuse *Predisposed:*Arabs + Middle Eastern Jews*Associated with:*celiac diseaseB.SECONDARY INTESTINAL LYMPHOMAas part of generalized systemic process*Incidence:*4-20% of all NHL; 10% of patients with abdominal lymphoma have bowel involvement*At risk:*long-standing celiac disease, AIDS, systemic lupus erythematosus, Crohn disease, history of chemotherapy*Median age:*60 years*Histo*: (1)T-cell malignant lymphoma (in celiac disease)(2)B-cell lymphoma(3)Immunoproliferative small intestinal disease(= Mediterranean lymphoma) (4)Low-grade B-cell lymphoma (= lymphoma of mucosa-associated lymphoid tissue)(5)Follicular lymphoma(6)Burkitt lymphoma (in children)(7)Mantle cell lymphoma(8)Hodgkin disease (<15%)*May be associated with:*enlargement of extra-abdominal lymph nodes, malabsorption*Radiographic types:* 1.Polypoid / nodular (47%)√ enlarged nodular folds2.Ulcerative (42%) √ ulcerative lesions, may be complicated by perforation√ aneurysmal configuration3.Diffusely infiltrating (11%)√ diffuse hoselike thickening of bowel wall√ decreased / absent peristalsis CT staging: StageItumor confined to bowel wallStageIIlimited to local nodesStageIIIwidespread nodal diseaseStageIVdisseminated to bone marrow, liver, other organsLocation:10-25% of NHL are extranodal; stomach > small bowel > colon > esophagus; multicentric in 10-50%√ enlargement of spleen√ bulky enlargement of regional lymph nodes@Esophagusleast common site of GI involvement (in <1%) @ Stomach 1-5% of all gastric malignancies; most common site of extranodal Hodgkin disease; 25% of extranodal lymphoma; mostly NHL with histiocytic cell type; isolated primary gastric malignancy in 10% Site:arises in lymphoid tissue of lamina propria; no predilection for any particular region of stomachDirect extension into:pancreas, spleen, transverse colon, liver√ flexibility of gastric wall preserved√ duodenum often affected when antrum involved√ circumscribed mass with endogastric / exogastric (25%) growth√ broad tortuous mucosal folds over large portions of stomach (diffuse form)√ large irregular ulcersCT: √ diffuse involvement of entire stomach (50%), typically more than half of gastric circumference√ segmental involvement (15%)√ ulcerated mass (8%)√ average wall thickness of 4-5 cm√ luminal irregularity (66%)√ hyperrugosity (58%)*Prognosis:*55% 5-year survival rate after resection@Small bowel1/5 of all small bowel malignancies; most common malignant small bowel tumor; multiple sites of involvement in 1/5; most common cause of intussusception in children >6 years Location:ileum (51%), jejunum (47%), duodenum (2%),Site:arising from lymphoid patches of Peyer*Types:* 1.Infiltrating lymphoma with plaquelike involvement of wall >5 cm in length (80%) / >10 cm in length (20%) (DDx: Crohn disease)√ ± ulceration (considerable excavation)√ desmoplastic response√ thickened valvulae with corrugated appearance√ aneurysmal dilatation (secondary to destruction of autonomic nerve plexus + muscle / tumor necrosis)2.Single / multiple polypoid mucosal / submucosal masses√ cobblestone defects due to lymphomatous polyps√ nodules may ulcerate√ may cause intussusception√ sprue pattern3.Endoexoenteric mass√ large mass with only small intramural component√ ± ulcer + fistulae + aneurysmatic dilatation4.Mesenteric / retroperitoneal adenopathy√ single / multiple extraluminal masses displacing bowel√ ill-defined confluent mass engulfing + encasing multiple loops of adjacent bowel√ "sandwich configuration" = mass surrounding mesenteric vessels that are separated by perivascular fat√ conglomerate mantle of retroperitoneal + mesenteric mass@ Colon Less commonly involved than stomach / small bowel; 1.5% of all abdominal lymphomas Location:cecum most commonly involved (85%)√ single mass > diffuse infiltration > polypoid lesion√ paradoxical dilatation√ gross mural circumferential / focal soft-tissue thickening (average size of 5 cm)√ slight enhancement√ massive regional + distant mesenteric + retroperitoneal adenopathy*DDx:*frequently resembles inflammatory disease / polyposis *Prognosis:*(a)71-82% 2-year survival rate in isolated bowel lymphoma(b)0% 2-year survival rate in stage IV disease with bowel involvement *Cx during chemotherapy:*perforation (9-40%), hemorrhage

Notes:

MALIGNANT MELANOMA
=develops from melanocytes derived from neural crest cells, arising in preexisting benign nevi*Incidence:*1% of all cancers @Skin primary*Clark staging:* LevelIall tumor cells above basement membrane (in situ lesion)LevelIItumor extends to papillary dermisLevelIIItumor extends to interface between papillary + reticular dermisLevelIVtumor extends between bundles of collagen of reticular dermisLevelVtumor invasion of subcutaneous tissue (in 87% metastatic)*Breslow staging:* thin<0.75 mm depth of invasionintermediate0.76-3.99 mm depth of invasionthick>4 mm depth of invasion METASTASES: latent period of 2-20 years after initial diagnosis (most commonly 2-5 years) Primary site:head + neck (79%), eye (77%), GU system (67%), GI tract (in up to 60%)@Lymphadenopathy-in 23% with level II + IV-in 75% with level V@Bone (11-17%) ● often initial manifestation of recurrence ● poor prognosisLocation:axial skeleton (80%), ribs (38%)@Lung (70% at autopsy)most common site of relapse; respiratory failure most common cause of death @Liver (17-23%; 58-66% at autopsy)√ single / multiple lesions 0.5-15 cm in size√ larger lesion often necrotic√ may be partially calcified@Spleen (1-5%; 33% at autopsy)√ single / multiple lesions of variable size√ solid / cystic@GI tract + mesentery (4-8%) ● abdominal pain, GI bleedingLocation:small intestine (35-50%), colon (14-20%), stomach (7-20%)√ multiple submucosal nodules ± "bulls-eye / target" appearance = central ulceration√ irregular amorphous cavity (exoenteric growth)√ intussusception (10-20%)@Kidney (up to 35% at autopsy)@Adrenal (11%, up to 50% at autopsy)@Subcutis*Prognosis:*30-40% eventually die from this tumor

Notes:

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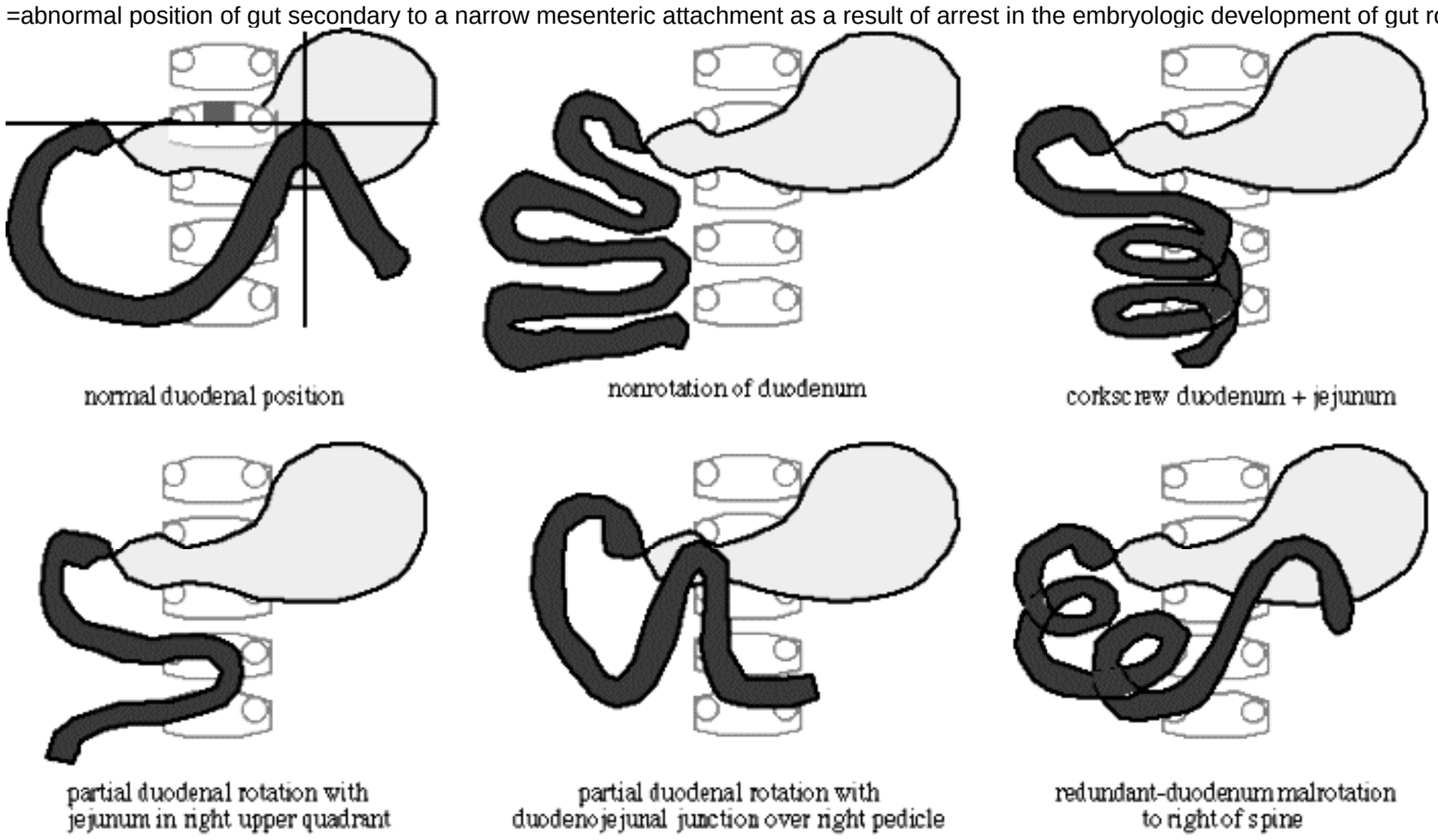
MALLORY-WEISS SYNDROME
=mucosal + submucosal tear with involvement of venous plexus*Pathophysiology:*violent projection of gastric contents against lower esophagus*Age:*30-60 years; M > F*Predisposed:*alcoholics ● history of repeated vomiting prior to hematemesis ● massive painless hematemesis Location:at / above / below (76%) esophagogastric junction√ longitudinal single tear in 77%, in 23% multiple tears√ extravasation of bariumAngio: √ bleeding site at gastric cardia*DDx:*peptic ulcer / ulcerative gastritis

Notes:

MALROTATION
=abnormal position of gut secondary to a narrow mesenteric attachment as a result of arrest in the embryologic development of gut rotation + fixation



Embryology: duodenojejunal + ileocolic segments of primitive digestive tube rotate by 270° in a counterclockwise direction about the omphalomesenteric vessels to cross beneath the vessels (future SMA + SMV); LUQ fixation at ligament of Treitz (an extension of the right crus of diaphragm) + fibrous tissue around celiac artery, located to left of L2) + RLQ fixation of cecum *Definition:*nonrotation ≤ 90°; malrotation = 90-270° *Associated with:*urinary pseudoobstruction, prune-belly syndrome, cloacal exstrophy Barium meal & barium enema: *Purpose:*guess the location of abnormal peritoneal fixation from position of bowel! √ clearly abnormal position of duodenum (81%): √ duodenum + jejunum to the right of spine (30%) √ corkscrew duodenum + jejunum (29%) √ duodenojejunal junction low + in midline (22%) √ unusual abnormal position of duodenum (16%): √ duodenojejunal junction over right pedicle √ duodenojejunal junction to left of spine but low √ duodenal redundancy to right of spine √ Z-shape configuration of duodenum + jejunum √ nonrotation = small bowel on right + colon on left (in 0.2% incidental finding in adults) √ abnormal position of duodenum + cecum (84%) √ normal position of duodenum (3%) √ normal position of cecum (in 5-20%) *DDx:*mobile cecum (15%) CT: √ SMV positioned to left of SMA (80%) √ aplastic / hypoplastic uncinate process of pancreas *Cx:*midintestinal volvulus, duodenal obstruction, internal herniation

Notes:

MASTOCYTOSIS
=systemic disease with mast cell proliferation in skin + RES (lamina propria of small bowel; bone; lymph nodes; liver; spleen) associated with eosinophils + lymphocytes *Age:*<6 months old (in 50%) *Categories:* Iindolent mastocytosis (most frequent) IImastocytosis associated with myeloproliferative / myelodysplastic hematologic disorder IIIaggressive / lymphadenopathic mastocytosis with eosinophilia IVmast cell leukemia (rare) ● diarrhea, malabsorption, steatorrhea, anorexia ● urticaria pigmentosa = cutaneous form (in 80-90%) ● abdominal pain, nausea, vomiting ● tachycardia, asthma, flushing, gastrointestinal upset, headache, pruritus (due to liberation of histamine / prostaglandin D_2) *caused by:*physical exertion, heat, certain foods, alcohol, nonsteroidal anti-inflammatory drugs @Stomach ulcer @ Small bowel √ generalized irregular distorted thickened folds ± wall thickening √ diffuse pattern of 2-3 mm sandlike mucosal nodules √ urticaria-like lesions of gastric + intestinal mucosa @ Reticuloendothelial system √ hepatomegaly √ Budd-Chiari hepatic veno-occlusive disease √ reversed portal venous flow √ cavernous transformation of portal vein √ splenomegaly (43-61%) √ ascites:(a) transudative secondary to liver disease (b)exudative from mast cell proliferation of peritoneum @ Bone √ sclerotic bone lesions *Dx:*skin / bone marrow biopsy; jejunal biopsy demonstrates an excess of mast cells *Cx:*(1)Peptic ulcer disease (histamine-mediated acid secretion) (2)Leukemia *Rx:* antihistamines, histamine decarboxylase inhibitors, sodium chromoglycase; steroids; splenectomy (for symptomatic splenomegaly / hypersplenism) *DDx:*carcinoid, pheochromocytoma

Notes:

MECKEL DIVERTICULUM
=persistence of the omphalomesenteric duct (= vitelline duct), which usually obliterates by 5th embryonic week ◊Most common congenital abnormality of the GI tract! *Incidence:*0.3-2-3% of population (at autopsy) *Age:*majority in children <10 years of age; M:F = 3:1 *Histo:*contains ectopic mucosa in 50%: gastric / pancreatic / colonic mucosa ◊Frequency of ectopic gastric mucosa: 15-34% overall; 60% in symptomatic children; in >95% with GI hemorrhage Location:within terminal 6 feet of ileum (= 30-90 cm from ileocecal valve); in 94% on antimesenteric border ● asymptomatic (20-40%) RULE OF 2s:(1)in 2% of population (2)symptomatic usually before age 2 (3)located within 2 feet of ileocecal valve (4)length of 2 inches NUC (>85% sensitivity, >95% specificity, >83-88% accuracy): √ accumulation of radiotracer in right lower quadrant coinciding with uptake of tracer in stomach N.B.:sensitivity drops after adolescence, because patients asymptomatic throughout childhood are less likely to have ectopic gastric mucosa ◊Tc-99m pertechnetate is excreted by mucoid cells of gastric mucosa, excretion is not dependent on presence of parietal cells *Preparation:* (1)No irritative measures for 48 hours (contrast studies, endoscopy, cathartics, enemas, drugs irritating GI tract) (2)Fasting for 3-6 hours (results in decreased gastric secretion + diminished bowel peristalsis) (3)Evacuation of bowel + bladder prior to study
*Dose:*5-20 mCi (100 µCi/kg) Tc-99m pertechnetate *Radiation dose:*0.54 rad/2 mCi for thyroid; 0.3 rad/2 mCi for large intestine; 0.2 rad/2 mCi for stomach *Imaging:*serial images in 5- to 10-minute intervals for 1 hour √ improved visualization through (a)pentagastrin = stimulates uptake (6 µg/kg SC 20 min prior to pertechnetate) (b)cimetidine = inhibits secretion (maximum 300 mg/dose IV 1 hour prior) (c)glucagon = decreases peristalsis (50 µg/kg IM 5-10 minutes prior) √ poor visualization with use of perchlorate + atropine (= depressed uptake) False-positive results: (1)Ectopic gastric mucosa in gastrogenic cyst, enteric duplication, normal small bowel, Barrett esophagus (2)Increased blood pool in AVM, hemangioma, hypervascular tumor, aneurysm (3)Duodenal ulcer, ulcerative colitis, Crohn disease, appendicitis, laxative abuse (4)Intussusception, intestinal obstruction, volvulus (5)Urinary tract obstruction, caliceal diverticulum (6)Anterior meningomyelocele (7)Poor technique *mnemonic:*"HA GUIDI" **H**emangioma **A**ppendicitis **G**astric ectopia **U**rinary obstruction **I**ntussusception **D**uplication of bowel **I**nflammatory bowel disease False-negative results: (1)Insufficient mass of ectopic gastric mucosa (2)Dilution of intraluminal activity (hemorrhage / hypersecretion) *mnemonic:*"MIS" **M**alrotation of ileum **I**rritable bowel in RLQ (rapid transit) **S**mall amount of ectopic gastric mucosa
Enteroclysis: √ elongated, smoothly marginated, clublike, intraluminal mass parallel to long axis of distal ileum = inverted Meckel diverticulum (20%) √ 0.5-20-cm-long blind pouch on the antimesenteric border of ileum with junctional fold pattern Angio (59% accuracy): √ presence of vitelline artery (= anomalous end branch of superior mesenteric artery) is PATHOGNOMONIC *Cx (in 20%):* (1)GI bleeding secondary to ulceration (in 95% due to ectopic gastric mucosa) (2)Acute diverticulitis (3)Intestinal obstruction secondary to intussusception (diverticulum acts as lead point) / volvulus (when omphalomesenteric diverticulum attached to umbilicus by fibrous band) (4)Malignant tumor (rare): carcinoma, sarcoma, carcinoid (5)Chronic abdominal pain

Notes:

MECONIUM ILEUS
=small bowel obstruction secondary to desiccated meconium pellets impacted in distal ileum *Age:*may develop in utero (in 15%) *Associated with:* cystic fibrosis with thick + sticky meconium due to deficiency of pancreatic secretions (in almost 100%) ◊Earliest clinical manifestation of cystic fibrosis! ◊Virtually all infants with meconium ileus prove to have cystic fibrosis ◊10-15% of infants with cystic fibrosis present with meconium ileus! ● abdominal distension, bilious emesis ● failure to pass meconium within 48 hours
√ numerous dilated small bowel loops without air-fluid levels (fluid not present) √ "bubbly" / "frothy" appearance of intestinal contents √ "soap-bubble" / "applesauce" appearance in RLQ (in 50-66%) √ multiple round / oval filling defects in distal ileum + colon √ microcolon (unused colon in antenatal obstruction) OB-US: √ unusual echogenic intraluminal areas in small bowel (DDx: normal transient inspissated meconium) √ usually polyhydramnios √ fluid-filled dilated small bowel *Cx (in 40-50%):*volvulus, ischemia, necrosis, stenosis, atresia, perforation, meconium peritonitis, pseudocyst *Rx:*(1)Nonionic contrast media enema (because of risk of bowel perforation) (2)17% Hypaque / Conray enema mixed with acetylcysteine (Mucomyst®) (3)Gastrografin® enema with Tween 80 (attention to fluid + electrolyte balance) *DDx:*Hirschsprung disease, small bowel atresia with meconium ileus, meconium plug syndrome, small left colon syndrome, imperforate anus, obstruction from duplication cyst

Notes:

MECONIUM PERITONITIS
=sterile chemical peritonitis secondary to perforation of bowel proximal to high-grade / complete obstruction that seals in utero due to inflammatory response *Incidence:*1:35,000 livebirths *Age:*antenatal perforation after 3rd month of gestation *Cause:* (1)Atresia (secondary to ischemic event) (50%) (a)of small bowel (usually ileum or jejunum) (b)of colon (uncommon) (2)Bowel obstruction (46%) (a)meconium ileus (b)volvulus, internal hernia (c)intussusception, congenital bands, Meckel diverticulum (3)Hydrometrocolpos ◊Meconium peritonitis due to cystic fibrosis diagnosed in utero in 8% + at birth in 15-40%! ◊Intraperitoneal meconium may calcify within 24 hours!
Types: (a)fibroadhesive type (most common): =intense chemical reaction of peritoneum, which seals off the perforation ● no evidence for active leak at birth √ dense mass with calcium deposits √ calcific plaques scattered throughout peritoneal cavity (b)cystic type: =cystic cavity formed by fixation of bowel loops surrounding the perforation site, which continues to leak meconium √ cyst outlined by calcific rim (c)generalized type: ● perforation occurs immediately antenatally ● active leakage of bowel contents √ complicated ascites
√ intra-abdominal calcifications (conspicuously absent in cystic fibrosis) √ peripherally calcified pseudocysts √ small flecks of calcifications scattered throughout abdomen √ larger aggregates of calcifications along inferior surface of liver / flank / processus vaginalis / scrotum √ obstructive roentgen signs following birth √ separation of bowel loops by fluid √ microcolon = "unused colon" √ meconium hydrocele producing labial mass OB-US: √ polyhydramnios (64-71%) √ fetal ascites (54-57%) √ bowel dilatation (27-29%) √ intra-abdominal bright echogenic mass √ multiple linear / clumped foci of calcifications (84%); may develop within 12 hours after perforation √ meconium pseudocyst = well-defined hypoechoic mass surrounded by an echogenic calcified wall (= contained perforation) *DDx:*(1)Intra-abdominal teratoma (2)Fetal gallstones (3)Isolated liver calcifications *Mortality:*up to 62% *Prognosis:*generally good; surgery may not be required when perforation site is completely healed

Notes:

MECONIUM PLUG SYNDROME
=local inspissation of meconium leading to low colonic obstruction *Age:*newborn infant (symptomatic within first 24 hours of life) *Cause:*cystic fibrosis (25%), Hirschsprung disease, prematurity, maternal magnesium sulfate treatment ● abdominal distension ● vomiting ● failure to pass meconium √ distended transverse + ascending colon + dilated small bowel (proximal to obstruction) √ occasionally bubbly appearance in colon (DDx: submucosal air in necrotizing enterocolitis) √ presacral pseudotumor (no gas in rectum) √ double-contrast effect = barium between meconium plug + colonic wall *Rx:*water-soluble enema *DDx:*Hirschsprung disease

Notes:

MELANOSIS COLI
=benign brown-black discoloration of colonic mucosa *Incidence:*10% of autopsies *Cause:*? chronic anthracene cathartic usage ● asymptomatic *Prognosis:*no malignant potential

Notes:

MÉNÉTRIÈR DISEASE
=GIANT HYPERTROPHIC GASTRITIS =HYPERPLASTIC GASTROPATHY =characterized by excessive mucus production and TRIAD of(1)Giant mucosal hypertrophy (2)Hypoproteinemia (3)Hypochlorhydria *Histo:*hyperplasia of glandular tissue + microcyst formation, mucosal thickness up to 6 mm (normal range: 0.6-1.0 mm) *Age:*20-70 years; M:F = 2:1 *Associated with:* benign gastric ulcer (13-72%) ● protein-losing enteropathy with hypoproteinemia + peripheral edema ● weight loss ● gastrointestinal bleeding ● absent / decreased acid secretion (>50%) epigastric pain vomiting Location:throughout fundus + body, particularly prominent along greater curvature, antrum usually spared (DDx to lymphoma: usually in antrum) √ markedly enlarged + tortuous gastric folds in spite of adequate gastric distension √ relatively abrupt demarcation between normal + abnormal areas √ marked hypersecretion (mucus) √ preserved pliability CT: √ wall thickening of proximal stomach √ nodular symmetric folds *DDx:*lymphoma, polypoid variety of gastric carcinoma, acute gastritis, chronic gastritis, gastric varices

Notes:

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MESENTERIC LYMPHADENITIS
=clinical entity whose symptoms relate to benign inflammation of lymph nodes in the bowel mesentery *Cause:*Yersinia enterocolitica, Y pseudotuberculosis, viral infection *Age:*children, young adults ● nausea, vomiting, diarrhea, fever ● diffuse / RLQ pain + tenderness Location:usually RLQ (immediately anterior to right psoas muscle in 78%, small bowel mesentery in 56%) √ enlarged mesenteric lymph nodes √ isolated ileal wall thickening (33%) √ colonic wall thickening (18%)
N.B.:visualization of entire normal appendix is necessary to differentiate from acute appendicitis! *DDx:*appendicitis (enlarged nodes immediately anterior to right psoas muscle in 40-82%, nodes less numerous + smaller), Crohn disease

Notes:

MESENTERIC ISCHEMIA
Acute Mesenteric Ischemia *Etiology*: (a)arterial:atheromatous disease, embolic disease, dissecting aortic aneurysm, fibromuscular hyperplasia, arteritis, endotoxin shock, hypoperfusion (shock, hypovolemia), disseminated intravascular coagulation, direct trauma, radiation -**occlusive mesenteric infarction** (90% mortality) 1.embolus (40-50%) just distal to middle colic a. 2.SMA thrombosis (20-40%) at origin + site of atherosclerotic narrowing (ostium stenosis) -**nonocclusive mesenteric ischemia** (10% mortality) = preexisting atherosclerosis with systemic low-flow state (cardiac failure / intraoperative hypotension, bowel vasospasm) (b)venous (<10%): young patient, often following abdominal surgery Location:superior mesenteric vein > inferior mesenteric vein > portal vein (c)incarceration of hernia, volvulus, constriction by adhesive bands, intussusception *Prevalence:*5% for SMA; 4% for celiac artery; 11% for inferior mesenteric artery *Pathophysiology:*mucosa is most sensitive area to anoxia from arterial / venous occlusion with early ulcerations leading to formation of strictures ● first crampy, then continuous abdominal pain with acute event ● cardiac disease predisposing to embolization ● gut emptying (vomiting / diarrhea) ● WBC >12,000/µl with left shift (80%) ● gross rectal bleeding Location:(a)any segment of small bowel (b)distal transverse colon, splenic flexure, cecum (most common) *Consequences:* dependent on magnitude of insult, duration of process, adequacy of collaterals (a)reversible ischemia 1.Complete restitution of bowel wall secondary to abundant collaterals 2.Healing with fibrosis + stricture formation (b)irreversible ischemia 1.Transmural infarction with bowel perforation
Plain film: √ gasless abdomen (= fluid-filled loops from exudation) (21%) √ bowel distension to splenic flexure (= perfusion territory of SMA) in 43% √ "thumbprinting" (36%) = thickening of bowel wall + valvulae (edema) √ small bowel pseudoobstruction (most frequently in thrombosis) √ pneumatosis = dissection of luminal gas into bowel wall (28%) √ mesenteric + portal vein gas (14%) √ ascites (14%) Barium: √ "scalloping / thumbprinting" = thickening of wall + valvulae √ "picket fencing" √ separation + uncoiling of loops √ narrowed lumen √ circumferential ulcer CT (26-73-82% sensitive): √ focal / diffuse bowel dilatation (10-56-71%) with gas (43%) / fluid (29%) √ portal venous gas (5-13-36%) / mesenteric vein gas (28%) √ pneumoperitoneum (7%) √ ascites (43%) √ mesenteric edema (a)arterial occlusion: √ thrombosis of SMA (4-18%) √ pneumatosis intestinalis (22-30%) √ thumbprinting (26%) = thickening of bowel wall √ lack of bowel wall enhancement with arterial occlusion (b)venous thrombosis: √ SMV / portal vein thrombosis (15%) √ thickened intestinal wall (64%) √ marked contrast enhancement Angio: √ occlusion / vasoconstriction / vascular beading √ embolus lodged at major branching points distal to first 3 cm of SMA
NUC: (a)IV / IA Tc-99m sulfur colloid / labeled leukocytes, Ga-citrate, Tc-99m pyrophosphate: √ tracer accumulation 5 hours after onset of ischemia (more intense uptake with transmural infarcts) (b)intraperitoneal injection of Xe-133 in saline is absorbed by intestine: √ decreased washout with abnormal perfusion of strangulated bowel *Prognosis*: (1)Massive infarction of small + large bowel if mesenteric embolization occurs proximal to middle colic artery (= limited collateral flow) (2)Focal segments of intestinal ischemia if mesenteric embolization occurs distal to middle colic artery (= good collateral flow) *Mortality:*70-80-92% for intestinal infarction
Chronic Mesenteric Ischemia =ABDOMINAL ANGINA = intermittent mesenteric ischemia in severe arterial stenosis with inadequate collateralization provoked by food ingestion ● postprandial abdominal pain 15-20 minutes after food intake (due to "gastric steal" diverting blood flow away from intestine) ● fear of eating large meals ● weight loss, malabsorption ● reflex emptying of bowel after eating Barium: (a)Subacute: √ flattening of one border √ pseudosacculation / pseudodiverticula on antimesenteric border (b)Chronic: √ 7- to 10-cm-long smooth pliable strictures √ dilatation of gut between strictures √ thinned + atrophic valvulae *Cx:*obstruction Duplex US: √ celiac trunk occlusion + retrograde perfusion of hepatic artery through SMA √ PSV >300 cm/sec and EDV >45 cm/sec in SMA √ peak systolic velocity >160 cm/sec in celiac trunk for >50% stenosis (57% sensitivity, 100% specificity) during fasting state

Notes:

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MESOTHELIAL CYST
=MESENTERIC / OMENTAL CYST *Etiology:*failure of mesothelial peritoneal surfaces to coalesce *Path:*unilocular thin-walled cyst usually with serous, occasionally chylous / hemorrhagic fluid contents *Histo:*lined by mesothelial cells + surrounded by thin layer of fibrous tissue Location:small bowel, mesentery (78%), mesocolon • asymptomatic √ single cyst up to several cm in size √ omental cysts may be pedunculated CT: √ near-water density / soft-tissue density √ ± fluid levels related to fat + water components *Cx:*torsion, hemorrhage, intestinal obstruction *DDx:*lymphangioma (septations)

Notes:

METASTASES TO SMALL BOWEL
*Origin:*colon > stomach > breast > ovary > uterine cervix > melanoma > lung > pancreas Spread: (1)Intraperitoneal seeding: primary mucinous tumor of ovary, appendix, colon; breast cancer (2)Hematogenous dissemination with submucosal deposits: malignant melanoma, breast carcinoma, lung carcinoma, Kaposi sarcoma (3)Direct extension from adjacent neoplasm: ovary, uterus, prostate, pancreas, colon, kidney √ fixation + tenting + transverse stretching (= across long axis) of folds secondary to mesenteric + peritoneal infiltration (most common form) UGI: √ single mass protruding into lumen resembling annular carcinoma √ "bulls-eye" lesions = multiple polypoid masses with sizable ulcer craters √ obstruction from kinking / annular constriction / large intraluminal mass √ compression by direct extension of primary tumor / involved nodes CT: √ soft-tissue density nodules / masses √ sheets of tissue causing thickening of bowel wall + mesenteric leaves √ fixation + angulation of bowel loops (in tumors with desmoplastic response) √ ascites

Notes:

METASTASES TO STOMACH

Organ of origin:malignant melanoma, breast, lung, colon, prostate, leukemia, secondary lymphoma ● GI bleeding + anemia (40%) ● epigastric pain √ solitary mass (50%) √ multiple nodules (30%) √ linitis plastica (20%): especially breast √ multiple umbilicated nodules: melanoma

Notes:

MIDGUT VOLVULUS
=torsion of entire gut around SMA due to a short mesenteric attachment of small intestine in malrotation *Age:*neonate / young infant; occasionally older child / adult *In 20% associated with:*(1)Duodenal atresia (2)Duodenal diaphragm (3)Duodenal stenosis (4)Annular pancreas *Pathophysiology:* degree of twisting can change due to natural movement of bowel + determines symptomatology; severe volvulus (= twist of 3 and a half turns) causes bowel necrosis ● acute symptoms in newborn (medical emergency): bile-stained vomiting (intermittent, postprandial, projectile); abdominal distension; shock ● intermittent obstructive symptoms in older child: recurring attacks of nausea, vomiting, and abdominal pain ● failure to thrive (hypoproteinemic gastroenteropathy as a result of lymphatic + venous obstruction) Plain film: √ dilated air-filled duodenal bulb + paucity of gas distally √ "double bubble sign" = air-fluid levels in stomach + duodenum √ isolated collection of gas-containing bowel loops distal to obstructed duodenum = gas-filled volvulus = closed-loop obstruction (from nonresorption of intestinal gas secondary to obstruction of mesenteric veins) Barium studies: √ duodenojejunal junction (ligament of Treitz) located lower than duodenal bulb + to the right of expected position √ spiral course of midgut loops = "apple-peel / twisted ribbon / corkscrew" appearance (in 81%) √ duodenal-fold thickening + thumbprinting (mucosal edema + hemorrhage) √ abnormally high position of cecum CT: √ whirl-like pattern of small bowel loops + adjacent mesenteric fat converging to the point of torsion (during volvulus) √ SMV to the left of SMA (NO volvulus) √ chylous mesenteric cyst (from interference with lymphatic drainage) US: √ clockwise whirlpool sign = color Doppler depiction of mesenteric vessels moving clockwise with caudal movement of transducer √ distended proximal duodenum with arrowhead-type compression over spine √ superior mesenteric vein to the left of SMA √ thick-walled bowel loops below duodenum + to the right of spine associated with peritoneal fluid Angio: √ "barber pole sign" = spiraling of SMA √ tapering / abrupt termination of mesenteric vessels √ marked vasoconstriction + prolonged contrast transit time √ absent venous opacification / dilated tortuous superior mesenteric vein *Cx:*intestinal ischemia + necrosis in distribution of SMA (bloody diarrhea, ileus, abdominal distension) *DDx:*pyloric stenosis (same age group, no bilious vomiting)

Notes:

MUCOCELE OF APPENDIX

Mucocele =distension of appendix with sterile mucus *Etiology:* (a)(perhaps) cystic dilatation of lumen secondary to obstruction by fecolith, foreign body, carcinoid, endometriosis, adhesions, volvulus (b)mucosal hyperplasia (c)mucinous cystadenoma (d)mucinous cystadenocarcinoma *Incidence:*0.07-0.3% of appendectomies *Mean age:*55 years; M:F = 1:4 *Associated with:*colonic adenocarcinoma (6-fold risk), mucin-secreting tumor of ovary ● asymptomatic (25%) ● acute / chronic right lower quadrant pain √ globular, smooth-walled, broad-based mass invaginating into cecum √ nonfilling of the appendix on BE √ peripheral rimlike calcifications frequent CT: √ round sharply defined mass with homogeneous content of near-water / soft-tissue attenuation US: √ purely cystic / cystic with fine internal echoes / complex cystic mass with high-level echoes √ gravity-dependent echoes = layering of protein macroaggregates / inspissated mucoid material NUC: √ intense early gallium uptake (affinity to acid mucopolysaccharides of mucus) *Cx:*(1)Rupture with pseudomyxoma peritonei (2)Torsion with gangrene + hemorrhage (3)Herniation into cecum with bowel obstruction

Myxoglobulosis =rare variant of mucocele of the appendix characterized by clusters of pearly white mucous balls intermixed with mucus ● usually asymptomatic ● may appear as acute appendicitis √ multiple 1- to 10-mm small rounded annular, nonlaminated calcified spherules (PATHOGNOMONIC) *DDx:*inverted appendiceal stump, acute appendicitis, carcinoma of the cecum

Notes:

NECROTIZING ENTEROCOLITIS
=NEC = ischemic bowel disease secondary to hypoxia, perinatal stress, infection (endotoxin), congenital heart disease *Incidence:*most common GI emergency in premature infants *Age:*develops >48-72 hours after birth; in 90% within first 10 days of life *Path:*acute inflammation + mucosal ulceration + widespread transmural necrosis *Organism:*not yet isolated; often occurs in miniepidemics within nursery *Predisposed:*premature infant (50-80%), Hirschsprung disease, bowel obstruction (small bowel atresia, pyloric stenosis, meconium ileus, meconium plug syndrome) ● blood-streaked stools (in 50%); explosive diarrhea ● bile emesis ● mild respiratory distress ● generalized sepsis Location:usually in terminal ileum (most commonly involved), cecum, right colon; rarely in stomach, upper bowel √ disarrayed bowel gas pattern (no longer normal array of polygons) √ distension of small bowel and colon (loops wider than vertebral body L1) ± air-fluid levels, commonly in RLQ (1st sign) √ tubular loops of bowel √ bowel wall thickening + "thumbprinting" √ "fixed" bowel = persistent abnormal loop of bowel without change on supine vs. prone films / for >24 hours √ pneumatosis intestinalis (80%) -in curvilinear shape (= subserosal) or -bubbly / cystic (= submucosal gas collection from gasforming organisms / dissection of intraluminal gas) √ "bubbly" appearance of bowel due to gas in wall / intraluminal gas / fecal matter (intraluminal contents are composed of blood, sloughed colonic mucosa, intraluminal gas, some fecal material) √ gas in portal venous system (frequently transient, does not imply hopeless outcome) √ ascites √ pneumoperitoneum (immediate surgery required) N.B.:Barium enema is contraindicated! May be used judiciously in selected cases with radiologic + clinical doubt! *Cx:*(1)Inflammatory stricture after healing (BE follow-up in survivors) (2)Bowel perforation in 12-32%

Notes:

PELVIC LIPOMATOSIS + FIBROLIPOMATOSIS
=nonmalignant overgrowth of adipose tissue with minimal fibrotic + inflammatory components compressing soft-tissue structures within pelvis *Age:*9-80 years (peak 25-60 years); M:F = 10:1; NO racial predominance for Blacks; obesity NOT contributing factor ● often incidental finding ● urinary frequency, flank pain, suprapubic tenderness ● recurrent urinary tract infections ● low back pain, fever √ elongation + narrowing of rectum √ elevation of rectosigmoid + sigmoid colon out of pelvis √ increase in sacrorectal space >10 mm √ stretching of sigmoid colon √ elongation + elevation of urinary bladder with symmetric inverted pear shape √ elongation of posterior urethra √ pelvic lucency; CT confirmatory √ medial / lateral displacement of ureters *Cx of fibrolipomatosis:* (1)Ureteral obstruction (40% within 5 years) (2)IVC obstruction

Notes:

PERITONEAL MESOTHELIOMA
=only primary tumor of peritoneum arising from mesothelial cells lining peritoneal cavity *Age:*55-66 years; M >> F *Associated with:*asbestos exposure Spread:intraperitoneal along serosal surfaces; direct invasion of liver, pancreas, bladder, bowel Location:pleura (67%), peritoneum (30-40%), pericardium (2.5%), processus vaginalis (0.5%) √ thickening of mesentery, omentum, peritoneum, bowel wall √ nodular masses in anterior parietal peritoneum becoming confluent cakelike √ disproportionately small amount of ascites √ areas of calcification (rare) CT: √ nodular irregular thickening of peritoneal surfaces √ localized masses √ infiltrating sheets of tissue √ foci of calcifications √ ascites of near-water density √ stellate configuration of neurovascular bundles √ pleated thickening of mesenteric leaves NUC: √ diffuse uptake of gallium-67 *Prognosis:*extremely poor due to advanced disease at presentation (most patients die within 1 year)
Cystic Mesothelioma =rare benign neoplasm without metastatic potential but tendency for local recurrence (in 27-50%) *Path:*multiple thin-walled cysts lined by mesothelial cells + filled with watery fluid; intermediate form between benign adenomatoid tumor + malignant peritoneal mesothelioma ◊Not associated with asbestos exposure! *Median age:*37 years; M << F Location:any peritoneal / omental surface, most frequently in pelvis ● contains watery fluid √ uni- / multilocular cystic tumor (cysts of 1 mm to 6 cm) without calcifications *DDx:*lymphangioma, ovarian carcinoma

Notes:

PERITONEAL METASTASES
=PERITONEAL CARCINOMATOSIS =intra-abdominal spread of malignant tumors *Origin:*(a)common: ovary, stomach, colon (b)less common: pancreas, uterus, bladder √ massive ascites √ desmoplastic reaction at (a) anterior border of rectum (Blumer shelf) (b) mesenteric side of terminal ileum CT: √ increased density of linear network in mesenteric fat √ loculated fluid collections in peritoneal cavity √ apparent thickening of mesenteric vessels (= fluid within leaves of mesentery) √ adnexal mass of cystic / soft-tissue density (= Krukenberg tumor) √ small nodular densities on peritoneal surface √ "omental cake" = thickening of greater omentum √ lobulated mass in pouch of Douglas √ calcified peritoneal implants in serous cystadenocarcinoma of ovary (in up to 40% with stage III / IV disease)

Notes:

PEUTZ-JEGHERS SYNDROME
=rare autosomal dominant disease with incomplete penetrance characterized by intestinal polyposis + mucocutaneous pigmentation (= hamartomatosis); often spontaneous mutation *Incidence:*1:7,000 livebirths; in 50% familial, in 50% sporadic; most frequent of polyposis syndromes to involve small intestines *Age:*25 years at presentation (range 10-30 years); M:F = 1:1 *Path:*multiple small sessile / large pedunculated polyps *Histo:*benign hamartomatous polyp with smooth muscle core arising from muscularis mucosae + extending treelike into lamina propria of polyp; misplaced epithelium in submucosa, muscularis propria, subserosa frequently surrounding mucin-filled spaces ● mucocutaneous pigmentation (similar to freckles) =1-5 mm small elongated melanin spots on mucous membranes (lower lips, gums, palate) + facial skin (nose, cheeks, around eyes) + volar aspects of toes and fingers (100%), becoming noticeable in first few years of life ● cramping abdominal pain (small bowel intussusception in 47%) ● rectal bleeding, melena (30%) ● prolapse of polyp through anus ● chronic hypochromic microcytic anemia Location:small bowel (jejunum + ileum > duodenum) > colon > stomach; mouth + esophagus spared @ Small bowel (>95%) √ multiple usually broad-based polyps separated by wide areas of intervening flat mucosa √ multilobulated surface of larger polyps √ myriad of 1- to 2-mm nodules of up to several cm = carpet of polyps √ intussusception usually confined to small bowel @ Colon + rectum (30%) √ multiple scattered 1- to 30-mm polyps; NO carpeting @ Stomach + duodenum (25%) √ diffuse involvement with multiple polyps @ Respiratory + urinary tract √ adenoma of bronchus + bladder *Cx:* (1)Transient intussusception (pedunculated polyp) (2)Carcinoma of GI tract (2-3%) (3)Carcinoma of pancreas (13%) (4)Carcinoma of breast (commonly bilateral + ductal) (5)Ovarian tumor (5%): ovarian sex cord tumor, mucinous cystic tumor, cystadenoma, granulosa cell tumor (6)Endometrial cancer: adenoma malignum of cervix (7)Testicular tumor: feminizing Sertoli cell tumor *Rx:*(1)Endoscopic removal of all polyps >5 mm (2)Surgery is reserved for obstruction, severe bleeding, malignancy *Prognosis:*decreased life expectancy (risk of cancer approaching 40% by 40 years of age) *DDx:*familial adenomatous polyposis, juvenile polyposis (similar age), Cowden syndrome, Cronkhite-Canada syndrome

Notes:

POSTCRICOID DEFECT
=variable defect seen commonly in the fully distended cervical esophagus; no pathologic value *Etiology:*redundancy of mucosa over rich postcricoid submucosal venous plexus *Incidence:*in 80% of normal adults Location:anterior aspect of esophagus at level of cricoid cartilage tumor- / weblike lesion with variable configuration during swallowing *DDx:*submucosal tumor, esophageal web (persistent configuration)

Notes:

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POSTINFLAMMATORY POLYPOSIS
=PSEUDOPOLYPOSIS =reepithelialized inflammatory polyps as sequelae of mucosal ulceration *Etiology:*ulcerative colitis (10-20%); granulomatous colitis (less frequent); schistosomiasis (endemic); amebic colitis (occasionally); toxic megacolon Location:most common in left hemicolon, may occur in stomach / small intestine √ sessile + frondlike appearance (often) √ filiform polyposis = multiple wormlike projections only attached at their bases (CHARACTERISTIC) *Pathogenesis:*ulcerative undermining of strips of mucosa with reepithelialization of denuded surfaces of tags + bowel wall *Prognosis:*NO malignant potential *DDx:*familial polyposis (polyps terminate in bulbous heads)

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PRESBYESOPHAGUS
=defect in primary peristalsis + LES relaxation associated with aging *Incidence:*15% in 7th decade; 50% in 8th decade; 85% in 9th decade *Associated with:*hiatus hernia, reflux ● usually asymptomatic √ impaired / no primary peristalsis √ often repetitive nonperistaltic tertiary contractions in distal esophagus √ mild / moderate esophageal dilatation √ poor LES relaxation *DDx:*diabetes, diffuse esophageal spasm, scleroderma, esophagitis, achalasia, benign stricture, carcinoma

Notes:

PROGRESSIVE SYSTEMIC SCLEROSIS
=PSS = multisystem connective tissue disorder (collagen-vascular disease) of unknown etiology characterized by widespread disorder of the microvasculature causing exuberant interstitial fibrosis with atrophy + sclerosis of many organ systems =SCLERODERMA = variety of skin disorders associated with hardening of skin; by extent of cutaneous involvement divided into: (a)DIFFUSE SCLERODERMA tends to involve older women; interstitial pulmonary fibrosis more severe; organ failure more likely (b)SYSTEMIC SCLEROSIS WITH LIMITED SCLERODERMA (formerly CREST syndrome) CREST features more common; pulmonary arterial hypertension more common + more severe)
May be associated with: other connective tissue diseases (especially SLE and polymyositis/dermatomyositis)
*Cause:*autoimmune condition with genetic predisposition, may be initiated by environmental antigen (eg, toxic oil syndrome in Spain through ingestion of adulterated rape seed oil / ingestion of L-tryptophan) *Peak age:*30-50 years; M:F = 1:3 *Histo:*vasculitis + submucosal fibrosis extending into muscularis, smooth muscle atrophy (initially hypertrophy and finally atrophy of collagen fibers)
● CREST:**C**alcinosis of skin **R**aynaud phenomenon **E**sophageal dysmotility **S**clerodactyly **T**elangiectasia ● antinuclear antibodies (30-80%): ● centromere antibody (ACA) specific for limited disease ● anti-topoisomerase-1 (= antiScl-70) identifies patients with diffuse cutaneous disease ● antibodies to extracellular matrix proteins and type I + IV collagen ● rheumatoid factor (35%) ● LE cells (5%) ● weakness, generalized debility *Prognosis:*50-67% 5-year survival rate

Gastrointestinal Scleroderma (in 40-45%) ◊Third most common manifestation of scleroderma (after skin changes + Raynaud phenomenon) ◊May precede other manifestations! ● abdominal pain, diarrhea ● multiple episodes of pseudoobstruction √ hepatomegaly @Esophagus (in 42-95%) ◊First GI tract location to be involved! ● dysphagia (50%) ● heartburn (30%) √ normal peristalsis above aortic arch (striated muscle in proximal 1/3 of esophagus) √ hypotonia / atony + hypokinesia / aperistalsis in lower 2/3 of esophagus (>50%) √ deficient emptying in recumbent position √ thin / vanished longitudinal folds √ mild to moderate dilatation of esophagus √ chalasia (= patulous lower esophageal sphincter) √ gastroesophageal reflux (70%) √ erosions + superficial ulcers (from asymptomatic reflux esophagitis: NO protective esophageal contraction) √ fusiform stricture usually 4-5 cm above gastroesophageal junction (from reflux esophagitis) √ esophageal shortening + sliding hiatal hernia *Cx:*peptic stricture, aspiration, Barrett esophagus, adenocarcinoma
@ Stomach (less frequent involvement) √ gastric dilatation √ decreased motor activity + delayed emptying @ Small bowel (in up to 45%) ◊PSS is rapidly progressing once small intestine is involved! ● malabsorption (delayed intestinal transit time + bacterial overgrowth) √ marked dilatation of small bowel (in particular duodenum = megaduodenum, jejunum) simulating small bowel obstruction CAVE:misdiagnosis of obstruction may lead to exploratory surgery! √ abrupt cutoff at SMA level (atrophy of neural cells with hypoperistalsis) √ prolonged transit time with barium retention in duodenum up to 24 hours √ "hidebound / accordion" pattern (60%) = sharply defined folds of normal thickness with decreased intervalvular distance (tightly packed folds) within dilated segment (due to predominant involvement of circular muscle) √ pseudodiverticula (10-40%) = asymmetric sacculations with squared tops + broad bases on mesenteric side (due to eccentric smooth muscle atrophy) √ pneumatosis cystoides intestinalis + pneumoperitoneum (occasionally) √ excess fluid with bacterial overgrowth (= "pseudo-blind loop syndrome") √ normal mucosal fold pattern *Cx:*intussusception without anatomic lead point
@ Colon (up to 40-50%) ● constipation (common), may alternate with diarrhea √ pseudosacculations + wide-mouthed "diverticula" on antimesenteric side (formed by repetitive bulging through atrophic areas) in transverse + descending colon √ eventually complete loss of haustrations (simulating cathartic colon) √ marked dilatation (may simulate Hirschsprung disease) √ stercoral ulceration (from retained fecal material) *Cx:*life-threatening barium impaction
DDx:(1)Dermatomyositis (similar radiographic findings) (2)Sprue (increased secretions, segmentation, fragmentation, dilatation most significant in midjejunum, normal motility) (3)Obstruction (no esophageal changes, no pseudodiverticula) (4)Idiopathic intestinal pseudoobstruction (usually in young people)

Pulmonary Scleroderma (in 10-25%) *Path:*almost 100% involvement in autopsy series *Histo:*thickening of basement membrane of alveoli + small arteries and veins ● slightly productive cough + progressive dyspnea ● hematemesis ● pulmonary function abnormalities in the absence of frank roentgenographic changes (typical dissociation of clinical, functional, and radiologic evidence) ● pericarditis Location:most prominent at both lung bases (where blood flow greatest) √ fine / coarse reticulations / diffuse interstitial infiltrates √ subpleural fibrocystic spaces (honeycombing) √ low lung volumes from progressive volume loss √ alveolar changes (secondary to aspiration of refluxed gastric contents with disturbed esophageal motility / mineral oil taken to combat constipation) √ air esophagram (DDx: achalasia, mediastinitis) √ pleural reaction / effusion distinctly uncommon *Cx:*(1)Pulmonary arterial hypertension (6-60%) (2)Increased incidence of lung cancer @ Heart: sclerosis of cardiac muscle ± cor pulmonale
Renal Scleroderma (25%) *Onset:*common within 3 years *Histo:*fibrinoid necrosis of afferent arterioles (also seen in malignant hypertension) √ renal cortical necrosis √ spotty inhomogeneous nephrogram (constriction + occlusion of arteries) √ concomitant arterial ectasia *Cx:*renal failure (from nephrosclerosis)

Musculoskeletal Scleroderma ● edema of distal portion of extremities ● thickened inelastic waxy skin most prominent about face + extremities ● symmetrical polyarthralgias (50-80%) ● Raynaud phenomenon (may proceed other symptoms by months / years) ● atrophy + thickening of skin and musculature (78%) @Fingers ● "sausage digit" = edema of digits associated with loss of transverse skin folds + lack of definition of subcutaneous fat √ "tapered fingers" = sclerodactyly = atrophy + resorption of soft tissues of fingertips + soft-tissue calcifications √ acroosteolysis = "penciling" / "autoamputation" = resorption of distal phalanges of hand (63%) beginning at volar aspect of terminal tufts with proximal progression √ calcinosis (25%) = punctate soft-tissue calcifications of fingertips, axilla, ischial tuberosity, forearm, elbow (over pressure area), lower leg, face √ calcifications around tendons. bursae, within joints
@Arthritis ● stiffness in small joints, occasionally in knee, shoulder, wrist ● lack of motility, eventually contractures √ arthritis of interphalangeal joints of hands (25%) Location:1st CMC, MCP, DIP, PIP √ central / marginal erosions (50%) √ resorption of palmar aspect of terminal phalanges (most frequent sign) √ bony erosions of carpal bones (trapezium), distal radius + ulna, mandible, ribs, lateral aspect of clavicle, humerus, acromion, mandible, cervical spine √ joint-space narrowing (late) *DDx:*rheumatoid, psoriatic, erosive arthritis √ soft-tissue swelling ± periarticular osteoporosis √ NO significant osteoporosis √ ± flexion contractures of fingers (from tendon sheath inflammation + fibrosis)
√ erosion of superior aspect of ribs √ widening of periodontal membrane

Notes:

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PROLAPSED ANTRAL MUCOSA

=prolapse of hypertrophic + inflammatory mucosa of gastric antrum into duodenum resulting in pyloric obstruction √ mushroom- / umbrella- / cauliflower-shaped filling defect at duodenal base √ filling defect varies in size + shape √ redundant gastric rugae can be traced from pyloric antrum through pyloric channel √ gastric hyperperistalsis

Notes:

PSEUDOMEMBRANOUS COLITIS
=CLOSTRIDIUM DIFFICILE DISEASE (more appropriate name because pseudomembranes are uncommon) *Cause:*overgrowth of Gram-positive Clostridium difficile in response to a decrease in normal intestinal flora *Etiologic agent:*cytotoxin produced by C. difficile *Predisposed:* (a)complication of antibiotic therapy with tetracycline, penicillin, ampicillin, clindamycin, lincomycin, amoxicillin, chloramphenicol, cephalosporins (b)complication of some chemotherapeutic agents: methotrexate, fluorouracil (c)following surgery / renal transplantation / irradiation; intestinal vascular insufficiency (d)shock, uremia (e)proximal to large bowel obstruction (f)debilitating diseases: lymphosarcoma, leukemia (g)immunosuppressive therapy with actinomycin D
*Histo:*pseudomembranes (exudate composed of leukocytes, fibrin, mucin, sloughed necrotic epithelium held in columns by strands of mucus) on a partially denuded colonic edematous mucosa (mucosa generally intact); reactive edema in lamina propria, submucosa, and eventually subserosa ● profuse watery diarrhea, abdominal cramps, tenderness ● fever, fecal blood, leukocytosis ● less common: chronic diarrhea, toxic megacolon, hyperpyrexia, leukemoid reaction, hypoalbuminemia with anasarca ● confluent small yellow plaques (= pseudomembranes) adherent to mucosal surface seen on endoscopy (50%) Location:rectum (95%); confined to right + transverse colon (5-27%) Plain film: √ adynamic ileus pattern = moderate gaseous distension of small bowel + colon √ "transverse banding" = marked thickening + distortion of haustral folds √ "thumbprinting" most prominent in transverse colon √ diffusely shaggy + irregular surface (confluent pseudomembranes) BE (CONTRAINDICATED in severe cases): √ "accordion-like" haustral thickening = contrast material trapped between distorted thickened closely spaced transverse edematous folds (simulating intramural tracts) √ pseudoulcerations = barium filling clefts between pseudomembranes √ irregular ragged polypoid contour of colonic wall √ discrete multiple plaquelike lesions of 2-4 mm in size (DDx: polyposis, nodular form of lymphoma) N.B.:Risk of colonic perforation! CT (85% sensitive, 48% specific): √ colonic wall thickening of 4-22 mm (61-88%) √ smooth circumferential thickening (44%) √ accordion sign (51-70%) = alternating bands of edematous haustral folds separated by intraluminal contrast material √ nodular thickening (17%) √ homogeneous enhancement due to hyperemia √ pericolonic stranding (42%) √ ascites (15-25%) √ NO colonic abnormality (12-39%)
Dx:(1)Stool assay for Clostridium difficile cytotoxin (detects toxin B): cumbersome to perform (2)Enzyme immunoassay test (up to 33% false-negative results): detects toxin A + B (3)Stool culture (95% sensitive): not available for 2 days (4)Pseudomembranes on proctosigmoidoscopy *Cx:*peritonitis *Prognosis:*15% mortality; most patients recover within 2 weeks *Rx:*discontinuation of suspected antibiotic + administration of vancomycin / metronidazole with attention to fluid and electrolyte balance

Notes:

PSEUDOMYXOMA PERITONEI
="jelly belly" = "gelatinous ascites" = slow insidious accumulation of large amounts of intraperitoneal gelatinous material *Etiology:*rupture of mucinous cystadenoma / cystadenocarcinoma of appendix (male) / ovary (female); rarely associated with malignancy of colon (<5%), stomach, uterus, pancreas, common bile duct, urachal duct, omphalomesenteric duct ● slowly progressive massive abdominal distension ● recurrent abdominal pain √ thickening of peritoneal + omental surfaces √ omental cake √ posterior displacement of bowel loops + mesentery √ voluminous septated / loculated pseudoascites √ several thin-walled cystic masses of different size throughout abdominal cavity √ scalloped contour of liver margins √ annular / semicircular calcifications (rare but highly suggestive) CT: √ tumor collection of very low attenuation (common) / soft-tissue density (rare) US: √ hypoechoic collection (common) / more solid appearance (rare) *DDx:*peritoneal metastases, pancreatitis with pseudocysts, pyogenic peritonitis, widespread echinococcal disease, ascites *Prognosis*:50% 5-year survival rate

Notes:

RADIATION INJURY

=obliterative endarteritis with irradiation in excess of 4,000-4,500 rads *Incidence:*5%; increased risk after pelvic surgery √ radiographic changes within field of radiation only

Radiation Gastritis Permanent radiographic findings of radiation injury appear 1 month to 2 years after therapy √ gastric ulceration + deformity (pylorus) √ enlargement + effacement of gastric folds √ antral narrowing + rigidity (similar to linitis plastica)

Radiation Enteritis Permanent radiographic findings of radiation injury appear >1-2 years following irradiation *Predisposed:*women (cancer of cervix, endometrium, ovary), patients with bladder cancer ● crampy abdominal pain (from intermittent obstruction) ● persistent diarrhea ● occult intestinal hemorrhage Location:ileum; concomitant radiation damage to colon / rectum √ irregular nodular thickening of folds with straight transverse course ± ulcerations √ serrated bowel margin √ thickened bowel wall with luminal narrowing √ multiple strictures + partial mechanical obstruction √ separation of adjacent bowel loops by >2 mm √ shortening of small bowel √ fixation + immobilization of bowel loops with similar radiographic appearance between examinations (from dense desmoplastic response to irradiation) CT: √ increased attenuation of mesentery *DDx*:Crohn disease, lymphoma, ischemia, hemorrhage

Radiation Injury Of Rectum Manifestation of radiation colitis can occur up to 15 years following irradiation *Predisposed:*90% in women (carcinoma of cervix) ● tenesmus, diarrhea, bleeding, constipation √ ridgelike appearance of mucosa (submucosal fibrosis) √ irregularly outlined ulcerations (rare) CT: √ narrowed partially distensible rectum √ thick homogeneous rectal wall √ "target sign" = submucosal circumferential lucency √ proliferation of perirectal fat >10 mm √ thickening of perirectal fascia √ "halo sign" = increase in pararectal fibrosis *Cx:*(1) Obstruction (2) Colovaginal / coloenteric fistula formation

Notes:

RETAINED GASTRIC ANTRUM

*Cause:*retention of endocrinologically active gastric antrum in continuity with pylorus + duodenum *Pathophysiology:*bathing of antrum in alkaline duodenal juice stimulates secretion of gastrin *Associated with:*gastric ulcers in 30-50% √ duodenogastric reflux of barium through pylorus (diagnostic) √ giant marginal ulcer / several marginal ulcers usually on jejunal side of anastomosis (large false-negative + false-positive rates; correct-positive rate of 28-60%) √ large amount of secretions √ edematous mucosa of jejunal anastomotic segment √ lacy / cobweblike small bowel pattern (hypersecretion) *Cx:*gastrojejunocolic fistula

Notes:

RETRACTILE MESENTERITIS
=CHRONIC FIBROSING MESENTERITIS = CHRONIC SUBPERITONEAL SCLEROSIS = MESENTERIC PANNICULITIS = LIPOSCLEROTIC MESENTERITIS = MESENTERIC LIPODYSTROPHY = MESENTERIC WEBER-CHRISTIAN DISEASE =rare disorder of unknown etiology characterized by fibrofatty thickening of small bowel mesentery *Etiology:*? trauma, previous surgery, ischemia *Path:*spectrum ranging from mesenteric lipodystrophy through mesenteric panniculitis to mesenteric fibrosis *Histo:*chronic inflammation with a dense collection of lymphocytes + plasma cells + lipid-laden macrophages; desmoplastic reaction; fat necrosis; calcifications *Associated with:* (1)Gardner syndrome, familial polyposis (2)Fibrosing mediastinitis, retroperitoneal fibrosis (3)Lymphoma, lymphosarcoma (4)Carcinoid tumor (5)Metastatic gastric / colonic carcinoma (6)Whipple lipodystrophy (7)Weber-Christian disease *Age:*most common in 6th decade; M:F = 2:1 ● crampy abdominal pain ● nausea + vomiting; mild weight loss ● low-grade fever
Location:root of mesentery extending toward mesenteric border of bowel Plain film: √ soft-tissue mass with calcifications √ ± thumbprinting (from vascular congestion) UGI: √ compression / distortion of duodenum near ligament of Treitz √ separation of small bowel loops with fixation, kinking, and angulation CT: √ mass of fat density interspersed with soft-tissue density (fibrous tissue) + calcifications √ mesenteric thickening with fine stellate pattern extending to bowel border √ retraction of small bowel loops √ single mesenteric soft-tissue mass (fibroma) √ multiple nodules throughout mesentery (fibromatosis) *Prognosis:*usually benign course *DDx:*metastatic gastric / colonic adenocarcinoma; carcinoid tumor; mesenteric lymphoma; liposarcoma of mesentery

Notes:

SCHATZKI RING
=LOWER ESOPHAGEAL MUCOSAL RING = constant lower esophageal ring (mucosal thickening) presumed to result from reflux esophagitis = thin annular peptic stricture *Incidence:*6-14% of population; old age > young age; M > F *Histo:*usually squamous epithelium on upper surface + columnar epithelium on undersurface; may be covered totally by squamous epithelium or columnar epithelium ● asymptomatic (if ring >20 mm) ● dysphagia (if ring <12 mm) Location:near the squamocolumnar junction; in region of B ring at inferior margin of lower esophageal sphincter √ permanently present nondistensible transverse ring with constant shape + size (range of 3-18 mm) √ 2- to 4-mm thick shelflike projection into lumen with smooth symmetric margins √ visible only with adequate distension of esophagogastric region and when located above the esophageal hiatus of the diaphragm √ best demonstrated in prone position during arrested deep inspiration with Valsalva maneuver while barium column passes through esophagogastric region √ short esophagus + intrahiatal / intrathoracic gastric segment = sliding hiatal hernia if Schatzki ring located 1-2 cm above diaphragmatic hiatus *Prognosis:*decrease in caliber over 5 years (in 25-33%) *Cx:*impaction of food bolus (associated with severe chest pain) *Rx:*(1)Proper mastication of food (2)Endoscopic rupture (3)Esophageal dilatation (radiographically often lack of caliber change after successful dilatation) *DDx:*annular peptic stricture (usually thicker, asymmetric, irregular surface, associated with thickened esophageal folds, serration of esophageal margins)

Notes:

SMALL LEFT COLON SYNDROME

*Cause:*transient functional colonic obstruction due to immaturity of mesenteric plexus *Age:*newborn infant *Associated with:*maternal diabetes mellitus (most common), maternal substance abuse; NOT related to cystic fibrosis √ colonic caliber becomes abruptly diminutive distal to splenic flexure √ bowel dilatation proximal to splenic flexure √ ± meconium plug (as a result and not the cause of obstruction) *Prognosis:*gradual resolution of functional immaturity over days to weeks

Notes:

SOLITARY RECTAL ULCER SYNDROME
=MUCOSAL PROLAPSE SYNDROME *Related disorders with common pathogenesis:* hamartomatous inverted polyp, colitis cystica profunda *Cause:*prolapse of anterior rectal wall resulting in mucosal ischemia due to traumatization of rectal mucosa by anal sphincter during defecation *Path:*small / large, single / multiple shallow ulcers; 25% broad-based, 18% patchy granular / velvety hyperemic mucosa; rectal stenosis through confluent circumferential lesion *Histo:*obliteration of lamina propria mucosae by fibromuscular proliferation of muscularis mucosae, streaming of fibroblasts + muscle fibers between crypts, misplaced mucosal glands deep to muscularis mucosae; diffuse increase in mucosal collagen ● chronic rectal bleeding ● passage of mucus ● disordered defecation ● tenesmus BE: √ ulcer (ulcerative type) √ polypoid lesion / nodules (polypoid type) √ flat granular mucosa (flat type) √ stricture Evacuation proctography: √ failure of anorectal angle to open while straining √ excessive perineal descent *Prognosis:* (1)Little change over time (2)Considerable change in appearance of lesion (3)Transfusions necessitated by massive blood loss *DDx:*invasive rectal carcinoma, Crohn disease

Notes:

SPRUE
=classic disease of malabsorption *Path:*villous atrophy (truncation) + elongation of crypts of Lieberkühn + round cell infiltration of lamina propria (plasma cells + lymphocytes)
Celiac Disease =NONTROPICAL SPRUE = GLUTEN-SENSITIVE ENTEROPATHY =characterized by malabsorption resulting from atrophy of small intestinal villi *Irritating agent:*gliadin polypeptides in wheat, rye, barley, oats *May be hereditary:*detected in 15% of 1st-degree relatives *Countries:*North America, Europe, Australia, India, Pakistan, Middle East, Cuba *Age:* childhood by age 2 years; 30-40 years with M<F; 40-60 years with M>F *Rx:*gluten-free diet: corn, rice, tapioca, soya, millet, vitamin supplements
Tropical Sprue *Etiology:*infectious agent cured with antibiotics; geographic distribution (India, Far East, Puerto Rico) *Age:*any age group ● glossitis ● hepatosplenomegaly ● macrocytic anemia + leukopenia *Prognosis:*spontaneous resolution after months / years *Rx:*responds well to folic acid + broad-spectrum antibiotics
● severe diarrhea, steatorrhea (CLASSIC but found only in minority of patients) ● crampy abdominal pain (from intussusception) ● lassitude, fatigue, weight loss ● stomatitis, anemia (iron / folate / vitamin B_{12} deficiency) ● bleeding diathesis ● neuropathy, depression ● infertility ● osteomalacia with bone pain ● dermatitis herpetiformis Location:patchy involvement of duodenum + jejunum > remainder of small bowel Small bowel follow-through: √ small bowel dilatation is HALLMARK in untreated celiac disease (70-95%), best seen in mid + distal jejunum (due to intestinal hypomotility); degree of dilatation related to severity of disease √ hypersecretion-related artifacts: √ air-fluid levels in small bowel (rare) √ segmentation = breakup of normal continual column of barium creating large masses of barium in dilated segments separated by stringlike strands from adjacent clumps due to excessive fluid; best seen on delayed films √ flocculation = coarse granular appearance of small clumps of disintegrated barium due to excess fluid best seen at periphery of intestinal segment; occurs especially with steatorrhea √ fragmentation = scattering = faint irregular stippling of residual barium resembling snowflakes associated with segmentation due to excessive fluid √ "moulage sign" (50%) = smooth contour with effaced featureless folds resembling tubular wax mold (due to atrophy of the folds of Kerckring); CHARACTERISTIC of sprue if seen in duodenum + jejunum √ long / normal / short transit time √ nonpropulsive peristalsis (flaccid + poorly contracting loops) √ normal / thickened / effaced mucosal folds (depending on degree of hypoproteinemia) √ colonlike haustrations in well-filled jejunum (secondary to spasm + cicatrization from transverse ulcers) √ "jejunization" of ileal loops (= adaptive response to decreased jejunal mucosal surface) = SPECIFIC √ transient nonobstructive intussusception (20%) without anatomic lead point √ "bubbly bulb" = peptic duodenitis = mucosal inflammation, gastric metaplasia, Brunner gland hyperplasia Enteroclysis: √ decreased number of folds in proximal jejunum (≤3 folds per inch) √ increased number of folds in distal ileum (>5 folds per inch) √ tubular featureless lumen √ mosaic pattern = 1-2 mm polygonal islands of mucosa surrounded by barium-filled distinct grooves (10%) CT: √ small bowel dilatation + increased fluid content ± mucosal fold thickening √ mild to moderate lymphadenopathy in mesentery / retroperitoneum (up to 12%)
Dx:(1)Jejunal / duodenal biopsy (2)Improvement of small bowel abnormalities after a few months on a gluten-free diet *Cause for relapse:*hidden dietary gluten, diabetes, bacterial overgrowth, intestinal ulceration, development of lymphoma *Cx:* (1)Ulcerative jejunoileitis =multiple chronic benign ulcers (sausage appearance of small bowel) with hemorrhage, perforation + obstruction *Age:*5th-6th decade Location:jejunum > ileum > colon ● response to gluten-free diet ceases *Prognosis:*frequently fatal *Rx:*small bowel resection (2)Hyposplenism (30-50%) √ small atrophic spleen (3)Cavitary mesenteric lymph node syndrome characterized by: (a)mesenteric lymph node cavitation (b)splenic atrophy (c)villous atrophy of small intestinal mucosa √ enlarged lymph nodes of low attenuation ± fat-fluid levels (filled with lipid-rich hyaline material) within jejunoileal mesentery *Prognosis:*usually fatal disorder (4)Malignant tumors (a)lymphoma (in 8%): commonly diffuse + nodular and of C-cell type *Peak prevalence:*7th decade √ enlarged nodular folds, ulcers, extrinsic mass effect (b)adenocarcinoma of small bowel (6%), rectum, stomach (c)squamous cell carcinoma of pharynx / esophagus (in 6%) during 6th-7th decade (5)Generalized lymphadenopathy with lymphocytosis (mimicking lymphoma) (6)Sigmoid volvulus (rare)
DDx: (1)Esophageal hypoperistalsis: scleroderma, idiopathic pseudoobstruction (2)Gastric abnormalities: Zollinger-Ellison syndrome, chronic granulomatous disease, eosinophilic enteritis, amyloidosis, malignancy (3)Tiny nodular defects on thickened folds: Whipple disease, intestinal lymphangiectasia, Waldenström macroglobulinemia (4)Small 1- to 3-mm nodules: lymphoid hyperplasia associated with giardiasis and immunoglobulin deficiency disease, diffuse lymphoma (5)Small nodules of varying sizes: systemic mastocytosis, amyloidosis, eosinophilic enteritis, Cronkhite-Canada syndrome (6)Bowel wall narrowing, kinking, scarring, ulceration: regional enteritis, bacterial / parasitic infection, carcinoid, vasculitis, ischemia, irradiation

Notes:

STRONGYLOIDIASIS
*Organism:*helminthic parasite Strongyloides stercoralis (2.2 mm long, 50µm in diameter); capable of reproducing within human host *Prevalence:*100 million cases globally; 4% in U.S. Country:tropical + subtropical regions, parts of Europe, southeastern U.S. (eastern Kentucky, rural Tennessee), Puerto Rico *Infection:*filiform larva enters body through skin / mucous membranes (from contaminated soil) *Cycle:*larva passes from subcutaneous / submucosal sites via venous circulation to lung; larva breaks into alveolar spaces and ascends via bronchi + trachea; larva swallowed; settles in duodenum + upper jejunum (lives in tunnels between enterocytes); parasitic adult female worms release eggs containing mature larvae into the intestinal lumen; ova hatch immediately into rhabditiform larvae and are passed to the environment *Path:*edema + inflammation of intestinal wall secondary to invasion by larvae; flattening of villi; ova in mucosal crypts ● asymptomatic for many years (in majority) ● larva currens = recurrent allergic cutaneous skin lesions of autoinfection ● severe malnutrition (malabsorption, steatorrhea) ● weight loss ● worms, larvae, eggs in stool ● peripheral eosinophilia ● elevated levels of immunoglobulin E √ paralytic ileus (massive invasion) √ edematous irregular mucosal folds, spasm, dilatation of proximal 2/3 of duodenum √ ulcerations √ stricture of 3rd + 4th part of duodenum √ rigid pipestem appearance + irregular narrowing of duodenum (in advanced cases)
Rx:thiabendazole (90% efficacy rate) *Prognosis:*high mortality in undernourished patients
HYPERINFECTION SYNDROME = extensive tissue invasion by larvae in patients with malignancy, autoimmune disease, malnutrition ● bacteremia, septicemia ● crampy abdominal pain, persistent vomiting, diarrhea CXR: √ fine miliary nodules / diffuse reticular opacities

Notes:

SUPERIOR MESENTERIC ARTERY SYNDROME
=VASCULAR COMPRESSION OF DUODENUM =WILKIE SYNDROME = CHRONIC DUODENAL ILEUS =BODY CAST SYNDROME =vascular compression of 3rd portion of duodenum within aortomesenteric compartment; probably representing a functional reflex dilatation *Etiology:*narrowing of angle between SMA + aorta to 10-22° (normal 45-65°): congenital, weight loss, visceroptosis due to loss of abdominal muscle tone (as in pregnancy), asthenic built, exaggerated lumbar lordosis, prolonged bed rest in supine position (body cast, whole-body burns, surgery) ● repetitive vomiting ● abdominal cramping √ megaduodenum = pronounced dilatation of 1st + 2nd portion of duodenum + frequently stomach, best seen in supine position √ vertical linear compression defect in transverse portion of duodenum overlying spine √ abrupt change in caliber distal to compression defect √ relief of compression by postural change into prone knee-elbow position

Notes:

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TAILGUT CYST
=RETRORECTAL CYSTIC HAMARTOMA *Cause:*incomplete regression of embryonic tailgut (= the portion distal to future anus) *Average age:*35 years; M<F *Histo:*several types of epithelia + elements of intestinal epithelium, smooth muscle within cyst wall ● asymptomatic / perirectal pain, rectal bleeding, urinary frequency Location:retrorectal / presacral space ± extension into ischiorectal fossa √ thin-walled multicystic / unilocular cyst adhering to sacrum / rectum √ fluid of clear / mucoid fluid with internal echoes *Cx:*(1)repeated perirectal abscesses, recurring anorectal fistula (2)degeneration into mucinous adenocarcinoma

Notes:

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TOXIC MEGACOLON
=acute transmural fulminant colitis with neurogenic loss of motor tone + rapid development of extensive colonic dilatation >5.5 cm in transverse colon (damage to entire colonic wall + neuromuscular degeneration) *Etiology:* 1.Ulcerative colitis (most common) 2.Crohn disease 3.Amebiasis, salmonellosis 4.Pseudomembranous colitis 5.Ischemic colitis *Histo:*widespread sloughing of mucosa + thinning of frequently necrotic muscle layers ● systemic toxicity ● profuse bloody diarrhea √ colonic ileus with marked dilatation of transverse colon √ few air-fluid levels √ increasing caliber of colon on serial radiographs without redundancy √ loss of normal colonic haustra + interhaustral folds √ coarsely irregular mucosal surface √ pseudopolyposis = mucosal islands in denuded ulcerated colonic wall √ pneumatosis coli ± pneumoperitoneum CT: √ distended colon filled with large amounts of fluid + air √ distorted haustral pattern √ irregular nodular contour of thin wall √ intramural air / small collections BE:CONTRAINDICATED due to risk of perforation *Prognosis:*20% mortality

Notes:

TUBERCULOSIS

Rarely encountered in Western Hemisphere, increased incidence in AIDS; usually associated with pulmonary tuberculosis (in 6-38%) *Etiology:* (1)Ingestion of tuberculous sputum (2)Hematogenous spread from tuberculous focus in lung to submucosal lymph nodes, associated with radiographic evidence of pulmonary TB in <50% (3)Primary infection by cow milk (Mycobacterium bovis) *Path:* (a)ulcerative form (most frequent): ulcers with their long axis perpendicular to axis of intestine, undermining + pseudopolyps (b)hypertrophic form: thickening of bowel wall (transmural granulomatous process) *Organism:*M. tuberculosis, M. bovis, M. avium-intracellulare *Age:*20-40 years ● weight loss, abdominal pain (80-90%) ● nausea, vomiting ● tuberculin skin test negative in most patients with primary intestinal TB Location:ileocecal area > ascending colon > jejunum > appendix > duodenum > stomach > sigmoid > rectum @Tuberculous peritonitis (in 1/3) ◊Most common presentation *Cause:*hematogenous spread / rupture of mesenteric node (a)wet type = exudative ascites with high protein contents + leukocytes (b)dry type = caseous adenopathy + adhesions (c)fibrotic type = omental cakelike mass with separation + fixation of bowel loops CT: √ high-density ascites (20-45 HU) √ enlarged lymph nodes (90%) with low-density centers in 40% (due to caseous necrosis) Location:peripancreatic + mesentery, retroperitoneum √ irregular masses of soft-tissue density in omentum + mesentery (common) *Cx:*small bowel obstruction (adhesions from serosal tubercles) @ Ileocecal area (80-90%) ◊Most commonly affected bowel *Cause:*relative stagnation of intestinal contents + abundance of lymphoid tissue (Peyer patches) √ Stierlin sign = rapid emptying of narrowed terminal ileum (due to persistent irritability) on BE √ thickened ileocecal valve (mass effect) √ Fleischner sign = "inverted umbrella" defect = wide gaping patulous ileocecal valve associated with narrowing of the immediately adjacent ileum + narrowed rigid cecum √ deep fissures + ulcers with sinus tracts / enterocutaneous fistulas / perforation *DDx:*Crohn disease, cecal carcinoma @Colon Site:segmental colonic involvement, esp. on right side √ rigid contracted cone-shaped cecum (spasm / transmural fibrosis) √ spiculations + wall thickening √ diffuse ulcerating colitis + pseudopolyps √ shortening + short hourglass strictures *DDx:*ulcerative colitis, Crohn disease, amebiasis (spares terminal ileum), colitis of bacillary dysentery, ischemic colitis, pseudomembranous colitis @Gastroduodenal Site:simultaneous involvement of pylorus + duodenum √ stenotic pylorus with gastric outlet obstruction √ narrowed antrum (linitis plastica appearance) √ antral fistula √ multiple large and deep ulcerations on lesser curvature √ thickened duodenal folds with irregular contour / dilatation *DDx:*carcinoma, lymphoma, syphilis @Esophagus ◊Least common GI tract manifestation *Cause:*secondary involvement from adjacent tuberculous lymphadenitis / primary TB √ deep ulceration √ stricture √ mass √ intramural dissection / fistula formation =sinus tract formation

Notes:

TURCOT SYNDROME
=autosomal recessive disease with (a)colonic polyposis (b)CNS tumors (especially supratentorial glioblastoma, occasionally medulloblastoma) *Age:*symptomatic during 2nd decade *Histo:*adenomatous polyps ● diarrhea ● seizures √ multiple 1-30 mm polyps in colon + rectum *Cx:*malignant transformation of colonic polyps in 100% *Prognosis:*death from brain tumor in 2nd + 3rd decade

Notes:

TYPHLITIS
=ILEOCECAL SYNDROME = NEUTROPENIC COLITIS =acute inflammation of cecum, appendix, and occasionally terminal ileum; initially described in children with leukemia + severe neutropenia; typhlos = "blind sac" = cecum *Cause:*leukemic / lymphomatous infiltrate, ischemia, focal pseudomembranous colitis, infection *Histo:*edema + ulceration of entire bowel wall; transmural necrosis with perforation possible *Organism:*CMV, Pseudomonas, Candida, Klebsiella, E. coli, B. fragilis, Enterobacter *Predisposed:*common in childhood leukemia, aplastic anemia, lymphoma, immunosuppressive therapy (eg, renal transplant), clinical AIDS ● abdominal pain, may be localized to RLQ ● watery diarrhea ● fullness / palpable mass in RLQ ● fever, neutropenia ● hematochezia / occult blood Location:cecum + ascending colon, appendix + distal ileum may become secondarily involved √ fluid-filled masslike density in RLQ √ distension of nearby small bowel loops √ thumbprinting of ascending colon √ circumferential thickening of cecal wall >4 mm √ occasionally pneumatosis CT (preferable examination due to risk of perforation): √ circumferential wall thickening (>1-3 mm) of cecum ± terminal ileum √ decreased bowel wall attenuation (edema) √ increased attenuation of adjacent fat + thickening of fascial planes (pericolonic inflammation) √ ± pericolonic fluid + intramural pneumatosis *Cx:*(1)Perforation (BE is a risky procedure) (2)Abscess formation *Rx:*early aggressive medical support (high doses of antibiotics + IV fluids) prior to development of transmural necrosis *DDx:*(1)Leukemic / lymphomatous deposits (more eccentric thickening) (2)Appendicitis with periappendicular abscess (normal cecal wall thickness) (3)Diverticulitis (4)Inflammatory bowel disease

Notes:

ULCERATIVE COLITIS
=common idiopathic inflammatory bowel disease with continuous concentric + symmetric colonic involvement *Etiology:*? hypersensitivity / autoimmune disease *Prevalence:*50-80:100,000 In high incidence areas of North America, Northern Europe, Australia *Path:*predominantly mucosal + submucosal disease with exudate + edema + crypt abscesses (HALLMARK) resulting in shallow ulceration *Age peak:*20-40 years + 60-70 years; M:F = 1:1 ● alternating periods of remission + exacerbation ● bloody diarrhea ● electrolyte depletion, fever, systemic toxicity ● abdominal cramps Extracolonic manifestations: ● iritis, erythema nodosum, pyoderma gangrenosum ● pericholangitis, chronic active hepatitis, primary sclerosing cholangitis, fatty liver ● spondylitis, peripheral arthritis, coincidental rheumatoid arthritis (10-20%) ● thrombotic complications Location:begins in rectum with proximal progression (rectum spared in 4%) (a)rectosigmoid in 95% (diagnosed by rectal biopsy); continuous circumferential involvement often limited to left side of colon (b)colitis extending proximally to splenic flexure = universal colitis (c)terminal ileum in 10-25% ("backwash ileitis") Plain film: √ hyperplastic mucosa, polypoid mucosa, deep ulcers √ diffuse dilatation with loss of haustral markings √ toxic megacolon √ free intraperitoneal gas √ complete absence of fecal residue (due to inflammation)
BE: (a)acute stage √ narrowing + incomplete filling (spasm + irritability) √ fine mucosal granularity = stippling of barium coat (from diffuse mucosal edema + hyperemia + superficial erosions) √ spicules + serrated bowel margins (tiny superficial ulcers) √ "collar button" ulcers (= undermining of ulcers) √ "double-tracking" = longitudinal submucosal ulceration over several cm √ hazy / fuzzy quality of bowel contour (excessive secretions) √ "thumbprinting" = symmetric thickening of colonic folds √ pseudopolyps = scattered islands of edematous mucosa + reepithelialized granulation tissue within areas of denuded mucosa √ widening of presacral space √ obliterated rectal folds = valves of Houston (43%) (b)subacute stage √ distorted irregular haustra √ inflammatory polyps = sessile frondlike / rarely pedunculated lesions (= localized mucosal inflammation resulting in polypoid protuberance) √ coarse granular mucosa (= mucosal replacement by granulation tissue) (c)chronic stage √ shortening of colon (= reversible spasm of longitudinal muscle) with depression of flexures √ "leadpipe" colon = rigidity + symmetric narrowing of lumen √ widening of haustral clefts / complete loss of haustrations (DDx: cathartic colon) √ "burnt-out colon" = fairly distensible colon without haustral markings + without mucosal pattern √ hazy / fuzzy quality of bowel contour (excessive secretions) √ postinflammatory polyps (12-19%) = small sessile nodules / long wormlike branching + bridging outgrowths (= filiform polyposis) √ "backwash ileitis" (5-30%) involving 4-25 cm of terminal ileum with patulous ileocecal valve + absent peristalsis + granularity
CT: √ wall thickening <10 mm
Cx: (1)Toxic megacolon ± perforation in 5-10% (DDx: granulomatous / ischemic / amebic colitis) ◊Most common cause of death in ulcerative colitis! (2)Colonic adenocarcinoma (3-5%): risk starts after 8-10 years of onset of disease; risk progresses at 0.5% for 10-20 years + at 0.9% thereafter; higher risk with pancolitis + onset of disease in <15 years of age Location:rectosigmoid > descending colon, distal transverse colon √ narrowed segment of 2-6 cm in length with eccentric lumen + irregular contour + flattened rigid tapered margins = scirrhous carcinoma √ annular / polypoid carcinoma (3)Colonic strictures (10%) smooth contour with fusiform pliable tapering margins, usually short + single stricture; commonly in sigmoid / rectum / transverse colon; usually after minimum of 5 years of disease; rarely cause for obstruction (DDx: colonic carcinoma)
DDx:(1)Familial polyposis (no inflammatory changes) (2)Cathartic colon (more extensive in right colon)
DDx between CROHN DISEASE and ULCERATIVE COLITIS: mnemonic:"LUCIFER M"
*Crohn Disease*Ulcerative Colitis
Locationright sideleft side **U**lcersdeepshallow **C**ontractionnoyes **I**leocecal valvethickenedgaping **F**istulaeyesno **E**ccentricityyesno **R**ate of carcinomaslight increasemarked increase **M**egacolonunusualyes

Notes:

VILLOUS ADENOMA

Villous Adenoma Of Colon *Incidence:*7% of all colonic tumors *Age:*presentation late in life;M = F Location:rectum + sigmoid (75%), cecum, ileocecal valve; 2% of all tumors in rectum + colon *Associated with:*other GI tumors (25%) ● sensation of incomplete evacuation ● rectal bleeding ● excretion of copious amounts of thick mucus ● fatigability, weakness ● electrolyte depletion syndrome in 4% (dehydration, hyponatremia, hypokalemia) √ may completely encircle the colon √ bulky tumor with spongelike corrugated appearance (barium within interstices) √ striated "brushlike" surface √ soft pliable tumor with change in shape √ innumerable mucosal projections (= fronds) with reticular / granular surface pattern (if villous elements constitute >75% of tumor, diagnosis can be made on BE) √ apparent decrease in size on postevacuation films *Cx:*malignant transformation / invasion (in 36%) related to size of tumor <5 cm (9%); >5 cm (55%); >10 cm (100%)

Villous Adenoma Of Duodenum More common in colon + rectum; fewer than 50 cases in world literature √ sessile, soft nonobstructive mass √ "lace" / "soap bubble" pattern √ preservation of peristaltic activity + bowel distensibility

Notes:

WALDENSTRÖM MACROGLOBULINEMIA
=low-grade lymphoid malignancy composed of mature plasmacytoid lymphocytes with production of abnormal monoclonal IgM protein *Incidence:*0.53 / 100,000 annually; frequency 10-15% that of multiple myeloma *Histo:*macroglobulin proteinaceous hyaline material fills lacteals in lamina propria of small bowel villi with secondary lymphatic distension + edema *Mean age:*63 years; M > F ● fatigue, weight loss ● diarrhea, steatorrhea, malabsorption ● anemia, bleeding diathesis ● IgM elevation ● hyperviscosity syndrome (20%) = bleeding, visual changes, neurologic abnormalities @Small bowel (rarely involved) √ small bowel dilatation √ uniform diffuse thickening of valvulae conniventes with spikelike configuration (jejunum + proximal ileum) √ granular surface of punctate filling defects (distended villi) @Bone marrow involvement (91-98%) (a)diffuse replacement of bone marrow (56%) (b)variegated replacement of bone marrow (35%) √ compression fractures of spine (48%) √ diffuse demineralization of spine √ lytic lesions on bone surveys (in up to 20%) MR (pre- and postcontrast T1WI preferred): √ marrow iso- / hypointense to muscle on T1WI √ enhancement of abnormal marrow on T1WI @Lymph nodes √ lymphadenopathy (43%) @Liver & spleen √ hepatosplenomegaly *Dx:*(1)characteristic M-spike in serum / urine electrophoresis (2)abnormal lymphplasmacytoid cells in bone marrow / lymph nodes *DDx:*multiple myeloma (lymphadenopathy rare, lytic lesions in 31%)

Notes:

WHIPPLE DISEASE
=INTESTINAL LIPODYSTROPHY =sporadically occurring chronic multisystem disease *Etiology:*thought to be caused by infection with an as yet unidentified gram-positive bacterium (Tropheryma whippelii) closely related to actinobacteria *Histo:*PAS-positive material (periodic acid Schiff) = glycoprotein within foamy macrophages in the submucosa of the jejunum (bacterial cell wall) + fat deposits within intestinal submucosa and lymph nodes causing lymphatic obstruction + dilatation
*Age:*4th-6th decade (mean age of onset, 50 years); M:F = 8:1; Caucasians
● recurrent and migratory arthralgias / nondeforming arthritis (65-95%); arthritis may precede Whipple disease in 10% up to 10 years ● malabsorption, steatorrhea, abdominal pain ● weight loss, low-grade fever ● polyserositis ● generalized peripheral lymphadenopathy (50%) ● hyperpigmentation of skin similar to Addison disease ● pale shaggy yellow plaques / erosions in postbulbar duodenum on endoscopy
Organ involvement:virtually every organ system, liver, intestines, joints, heart, lung, CNS, eyes, skin
√ moderate thickening of jejunal + duodenal folds (from mucosal + submucosal infiltration by PAS-positive macrophages combined with lymphatic obstruction) √ micronodularity (= swollen villi) and wild mucosal pattern √ hypersecretion, segmentation, fragmentation (occasionally if accompanied by hyperproteinemia) √ NO / minimal dilatation of small bowel √ NO rigidity of folds √ NO ulcerations √ normal transit time (approximately 3 hours) √ hepatosplenomegaly CT: √ bulky 3-4 cm large low-density lymph nodes in mesenteric root + retroperitoneum (due to extracellular neutral fat + fatty acids) √ thickening of bowel wall √ splenomegaly √ ascites √ pleuropericarditis √ sacroiliitis *Dx:*endoscopically guided biopsy of small bowel mucosa, abdominal / peripheral lymph node biopsy *Rx:*long-term broad-spectrum antibiotics (tetracycline) *DDx:*(1)Sprue (marked dilatation, no fold thickening, pronounced segmentation + fragmentation) (2)Intestinal lymphangiectasia (thickened folds throughout small bowel) (3)Amyloidosis (4)Lymphoma

Pseudo-Whipple disease in AIDS similar clinical picture caused by Mycobacterium avium intracellulare √ wall + fold thickening of small bowel loops √ mesenteric lymphadenopathy

Notes:

ZENKER DIVERTICULUM
=PHARYNGOESOPHAGEAL DIVERTICULUM =outpouching of posterior hypopharyngeal wall = pulsion diverticulum with herniation of mucosa + submucosa through oblique + transverse muscle bundles (pseudodiverticulum) of the cricopharyngeal muscle *Prevalence:*0.01-0.11% (overall); higher in elderly women (50% occur in 7th-8th decade) *Etiology:*cricopharyngeal dysfunction (cricopharyngeal achalasia / premature closure) results in increased intraluminal pressure *Associated with:*hiatal hernia, gastroduodenal ulcer, midesophageal diverticulum, esophageal spasm, achalasia ● compressible neck mass ● upper esophageal dysphagia (98%) ● regurgitation + aspiration of undigested food ● noisy deglutition ● halitosis (= foul breath) Location:at pharyngoesophageal junction in midline of Killian dehiscence / triangle of Laimer, at level of C5/6 √ posterior barium extension in upper half of semilunar depression on the posterior wall of esophagus (cricopharyngeal muscle) √ barium-filled sac extending caudally behind + usually to left of esophagus √ partial / complete obstruction of esophagus from external pressure of sac contents √ partial barium reflux from diverticulum into hypopharynx √ continual growth with successive enlargement CXR: √ air-fluid level in superior mediastinum *Cx:*aspiration pneumonia (30%); esophageal perforation; carcinoma (0.48%) *Rx:*surgical excision

Notes:

ZOLLINGER-ELLISON SYNDROME
=peptic ulcer diathesis associated with marked hypersecretion of gastric acid + gastrin-producing non-b islet cell tumor of pancreas *Cause:* A.GASTRINOMA (90%) = non-b islet cell tumor with continuous gastrin production B.PSEUDO Z-E SYNDROME = COWLEY SYNDROME = antral G-cell hyperplasia (10%) (increase in number of G-cells in gastric antrum) ● lack of gastrin elevation after secretin injection ● exaggerated gastrin elevation after protein meal *Age:*middle age; M > F
● Clinical tetrad: (1)Gastric hypersecretion: refractory response to histamine stimulation test concerning HCl concentration; increased basal secretion (>60% of augmented secretion is diagnostic) (2)Hypergastrinemia >1000 ng/L (during fasting) (3)Hyperacidity with basal acid output >15 mEq/h (4)Diarrhea (30%), steatorrhea (40%): may be sole complaint in 10%, frequently nocturnal; secondary to inactivation of pancreatic enzymes by large volumes of HCl ● severe intractable pain (90%) ● ulcer perforation (30%) ● positive secretin test = increase in serum gastrin level by >200 ng/L after administration of 2 IU/kg of secretin √ ulcers (atypical location + course should suggest diagnosis): Location:duodenal bulb (65%) + stomach (20%), near ligament of Treitz (25%), duodenal C-loop (5%), distal esophagus (5%) Multiplicity: solitary ulcer (90%), multiple ulcers (10%) √ recurrent / intractable ulcers √ marginal ulcers in postgastrectomy patient (a) on gastric side of anastomosis (b) on mesenteric border of efferent loop √ prominence of area gastricae (hyperplasia of parietal cell mass) √ enlargement of rugal folds √ sluggish gastric peristalsis (? hypokalemia) √ "wet stomach" = dilution of barium by excess secretions in nondilated nonobstructed stomach √ gastroesophageal reflux (common) + esophagitis √ dilatation of duodenum + upper small bowel (fluid overload) √ thickened folds in duodenum + jejunum (edema) √ rapid small-bowel transit time
mnemonic:"FUSED" **F**olds (thickened, gastric folds) **U**lcers (often multiple, postbulbar) **S**ecretions increased (refractory to histamine) **E**dema (of proximal small bowel) **D**iarrhea
Cx:(1)Malignant islet cell tumor (in 60%) (2)Liver metastases will continue to stimulate gastric secretion
Rx: (1)Control of gastric hypersecretion: (a)H2-receptor antagonist: cimetidine, ranitidine, famotidine (b)Hydrogen-potassium adenosine triphosphatase inhibitor (omeprazole) (2)Resection of gastrinoma if found (because of malignant potential) (3)Total gastrectomy

Notes:

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RENAL FAILURE
=reduction in renal function ● rise in serum creatinine >2.5 mg/dL

Acute Renal Failure Chronic Renal Failure Musculoskeletal Manifestations Of CRF

Notes:

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Acute Renal Failure = clinical condition associated with rapid steadily increasing azotemia ± oliguria (<500 mL urine per day) over days / weeks *Etiology:* A.PRERENAL=renal hypoperfusion secondary to systemic illness1.Fluid + electrolyte depletion2.Hemorrhage3.Hepatic failure + hepatorenal syndrome√ abnormally elevated resistive index4.Cardiac failure5.Sepsis√ resistive index <0.75 in 80% of kidneysB.RENAL (most common)1.Acute tubular necrosis:ischemia, nephrotoxins, radiographic contrast, hemoglobulinuria, myoglobulinuria, myocardial infarction, burns √ resistive index ≥0.75 in 91% of kidneys2.Acute glomerulonephritis + small vessel disease:acute poststrep glomerulonephritis, rapidly progressive glomerulonephritis, lupus, polyarteritis nodosa, Schönlein-Henoch purpura, subacute bacterial endocarditis, serum sickness, Goodpasture syndrome, malignant hypertension, hemolytic uremic syndrome, drug-related vasculitis, abruptio placentae √ normal resistive index <0.703.Acute tubulointerstitial nephritis:drug reaction, pyelonephritis, papillary necrosis √ abnormal resistive index4.Intrarenal precipitation (hypercalcemia, urate, myeloma protein)5.Arterial / venous obstruction6.Cortical necrosisC.POSTRENAL (5%) =result of outflow obstruction (rare)1.Prostatism2.Tumors of bladder, retroperitoneum, pelvis3.Calculus√ hydronephrosisD.CONGENITALbilateral renal agenesis / dysplasia / infantile polycystic kidney disease, congenital nephrotic syndrome, congenital nephritis, perinatal hypoxia *Incidence:*ATN + prerenal disease account for 75% of acute renal failure

Notes:

Chronic Renal Failure =decrease in renal function over months / years*Incidence:* end-stage renal disease in 0.01% of U.S. population; 85,000 patients/year undergo hemodialysis; 8,000 renal transplantations/year *Etiology:* A.INFLAMMATION / INFECTION1.Glomerulonephritis2.Chronic pyelonephritis3.Tuberculosis4.SarcoidosisB.VASCULAR1.Renal vascular disease2.Bilateral renal vein thrombosisC.DYSPROTEINEMIA1.Myeloma2.Amyloid3.Cryoglobulinemia4.Waldenström macroglobulinemiaD.METABOLIC1.Diabetes2.Gout3.Hypercalcemia4.Hyperoxaluria5.Cystinosis6.Fabry diseaseE.CONGENITAL1.Polycystic kidney disease2.Multicystic dysplastic kidney3.Medullary cystic disease4.Alport syndrome5.Infantile nephrotic syndromeF.MISCELLANEOUS1.Hepatorenal syndrome2.Radiation

Notes:

Musculoskeletal Manifestations Of CRF 1.Renal osteodystrophy = combination of 2° HPT, osteoporosis, osteosclerosis, osteomalacia, soft-tissue and vascular calcifications2.Aluminum toxicity (1-30%)*Cause:*ingestion of aluminum salts phosphate-binding antacids (to control hyperphosphatemia) ● aluminum serum level >100 ng/mL√ signs of osteomalacia (>3 insufficiency fractures with predominant involvement of ribs)√ avascular necrosis√ lack of osteosclerosis√ less evidence of subperiosteal resorption3.Amyloid deposition*Path:*amyloid consists of b_2-microglobulinOrgans:bone, tenosynovium (carpal tunnel syndrome), vertebral disk, articular cartilage + capsule, ligament, muscle4.Destructive spondyloarthropathy (15%)√ discovertebral junction erosion + sclerosis√ vertebral body compression√ disk space narrowing√ Schmorl node formation√ lack of osteophytosis√ facet involvement with subluxation5.Tendon rupture6.Crystal deposition diseaseType:calcium hydroxyapatite, CPPD, calcium oxalate, monosodium urate7.Osteomyelitis + septic arthritis8.Avascular necrosis (in up to 40%)

Notes:

DIABETES INSIPIDUS
A.Hypothalamic Diabetes Insipidus=vasopressin production is reduced to <10%*Cause:* (a)idiopathic (27%) rare familial (autosomal dominant X-linked) / sporadic disorder *Histo:*atrophic supraoptic nucleus ● never associated with anterior pituitary dysfunction(b)pituitary destruction by tumor / infiltrative disorder (32%):in childhood:hypothalamic glioma, tuber cinereum hamartoma, craniopharyngioma, histiocytosis, germinoma, leukemia, complication of meningitisin adulthood:sarcoidosis, metastasis ● in 60% associated with anterior pituitary dysfunction(c)pituitary destruction by surgery (20%) ● always associated with anterior pituitary dysfunction(d)head injury (17%) ● in 20% associated with anterior pituitary dysfunction●A lesion in the posterior pituitary will NOT produce diabetes insipidus, because it is just the storage space for vasopressin!B.Psychogenic Water Intoxication=compulsive intake of large amounts of fluid, which leads to inhibition of normal vasopressin production ● water deprivation testC.Primary Nephrogenic Diabetes Insipidus=rare sex-linked recessive genetic disorder with unresponsiveness of tubules + collecting system to vasopressin (in infants + young males)D.Secondary Nephrogenic Diabetes Insipidus*Cause:*drug toxicity, analgesic nephropathy, sickle cell anemia, hypokalemia, hypercalcemia, chronic uremic nephropathy, postobstructive uropathy, reflux nephropathy, amyloidosis, sarcoidosis

Notes:

ABNORMAL TUBULAR FUNCTION

A.PROXIMAL TUBULEreabsorbs almost all of glucose, amino acids, phosphate, bicarbonate ● glycosuria (Toni-Fanconi syndrome) ● aminoaciduria (cystinuria) ● phosphaturia (phosphate diabetes, thiazides) ● HCO_3^- wasting (proximal renal tubular acidosis)B.DISTAL TUBULEabsorbs most of water ● diabetes insipidus, secretes H^+ ● distal renal tubular acidosis

Notes:

ARTERIAL HYPOTENSION

*Cause:*intrarenal hypovolemia, primary vasoconstriction, reduced glomerular filtration, depletion of intratubular urine volume◊May occur as a contrast reaction!Urogram reverts to normal after reversion of hypotension! √ bilateral small smooth kidneys (compared with size on preliminary films)√ increasingly dense nephrogram√ usually NO opacification of collecting system√ initially opacification of collecting system if hypotension occurs during contrast injection

Notes:

HYPERCALCEMIA
mnemonic:"SHAMPOO DIRT"**S**arcoidosis **H**yperparathyroidism, **H**yperthyroidism **A**lkali-milk syndrome **M**etastases, **M**yeloma **P**aget disease **O**steogenesis imperfecta **O**steopetrosis **D** vitamin intoxication **I**mmobility **R**enal tubular acidosis **T**hiazides

Notes:

POLYCYTHEMIA
*Cause:*increased level of erythropoietin (acting on erythroid stem cells) secondary to a decrease in pO_2; erythropoietin precursor is produced in juxtaglomerular epitheloid cells of kidney + converted in bloodA.RENAL(a)intrarenal1.Vascular impairment2.Renal cell carcinoma (5%)3.Wilms tumor4.Benign fibroma5.Simple cyst (14%)6.Polycystic kidney disease(b)postrenal1.Obstructive uropathy (14%)B.EXTRARENAL(a)liver disease1.Hepatoma2.Regenerating hepatic cells(b)adrenal disease1.Pheochromocytoma2.Aldosteronoma3.Cushing diseaseC.CNS DISEASE1.Cerebellar hemangioblastomaD.Large uterine myomas NOT in:renal vein thrombosis, multicystic dysplastic kidney, medullary sponge kidney

Notes:

URINARY TRACT INFECTION

=pure growths of >100,000 organisms/mL urine*Prevalence:*3% of girls + 1% of boys during first 10 years of life*Underlying radiologic abnormality:* 1.Vesicoureteral reflux = VUR (30-40%)2.Obstructive uropathy (8%)3.Reflux nephropathy / scar formation (6%)The prevalence of an underlying radiologic abnormality depends on age, sex, and frequency of previous infections!*Imaging objective:* 1.Identify patients at risk for reflux nephropathy2.Detect reflux nephropathy / scars3.Detect obstructive uropathy4.Minimize radiation, morbidity, and costVCUG: for children <5 years of age with infection; normal results in 60-70% Renal cortical scintigraphy (DMSA / glucoheptonate): to detect acute pyelonephritis (risk for scarring) / scar; with VUR there is twice the risk of cortical defects than without VUR

Notes:

WETTING

1.**Enuresis**

=manifestation of neuromuscular vesicourethral immaturity; M:F = 3:2 ● intermittent wetting, usually at night during sleep ● often positive history of enuresis from one parent ● normal physical examination√ no structural abnormality; urography NOT indicated2.**Epispadia** =incomplete fusion of infravesical portion of urinary tract ● urinary incontinence from incompetent bladder neck / urethral sphincter√ abnormally wide symphysis pubis (>1 cm)3.Sacral agenesis=segmental defect (below S2) with deficiency of nerves that innervate bladder, urethra, rectum, feet◊Children of diabetic mothers are affected in 17%!4.**Extravesical infrasphincteric ectopic ureter**

only affects girls as boys do NOT have infrasphincteric ureteral orifices (a)ureter draining upper pole of duplex system exits below urethral sphincter (90%)(b)ureter draining single system with ectopic extravesical orifice (10%)5.**Synechia vulvae**

=adhesive fusion of minor labia directs urine primarily into vagina from where it dribbles out post micturition6.**Vaginal reflux**

in obese older girls with fat thighs and fat labia 7.Miscellaneousposterior urethral valves, urethral stricture, urethral diverticula

Notes:

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MALE INFERTILITY
A.CONGENITAL(a)Wolffian duct anomalies1.Renal agenesis / atrophy2.Vas deferens agenesis / cyst3.Seminal vesicle agenesis / cyst4.Ejaculatory duct cyst(b)Müllerian duct anomalies1.Müllerian duct cyst2.Utricle cystB.ACQUIRED1.Cowper duct cyst2.Prostatic cyst in peripheral zoneC.INFECTIOUS1.ProstatitisD.HORMONAL ● semen low in volume, acid pH, without fructose1.Seminal vesicle atrophy=seminal vesicles <7 mm in width2.Seminal vesicle hypoplasia=seminal vesicles <11 mm + >7 mm in width

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Notes:

ABNORMAL GAS IN URINARY TRACT

A.Renal emphysema = renal / perirenal gas1.Emphysematous pyelonephritis2.Emphysematous pyelitis3.Gasforming perinephric abscess4.Perinephric emphysemaB.Bladder1.Emphysematous cystitisC.Trauma1.Penetrating trauma2.Ureterosigmoidostomy, ileal conduit, catheterization with vesicoureteral reflux, percutaneous procedureCAVE:anomalous posterior position of colon3.Infarction of renal carcinoma (therapeutic / spontaneous)D.Fistula to urinary tractConnection:bronchus / cutis / GI tract (colon > duodenum > stomach > small bowel > appendix1.Inflammation: chronic purulent renal infection, diverticulitis, Crohn disease2.Neoplastic: colonic carcinoma

Notes:

Absent Renal Outline On Plain Film A.ABSENT KIDNEY1.Congenital absence2.S/P nephrectomyB.SMALL KIDNEY1.Renal hypoplasia2.Renal atrophyC.RENAL ECTOPIA1.Pelvic kidney2.Crossed fused ectopia3.Intrathoracic kidneyD.OBLITERATION OF PERIRENAL FAT1.Perirenal abscess2.Perirenal hematoma3.Renal tumors

Notes:

Nonvisualized Kidney On Excretory Urography A.ABSENCE OF KIDNEY1.Agenesis2.EctopiaB.LOSS OF PERFUSION1.Chronic infarction2.Unilateral renal vein thrombosis3.Fractured kidneyC.URINARY OBSTRUCTION1.Hydronephrosis2.Ureteropelvic junction obstructionD.REPLACED NORMAL RENAL PARENCHYMA1.Multicystic dysplastic kidney2.Unilateral polycystic kidney disease3.Renal tumor (RCC, TCC, Wilms tumor)4.Xanthogranulomatous pyelonephritis

Notes:

Unilateral Large Smooth Kidney A.PRERENAL(a)arterial:acute arterial infarction(b)venous:acute renal vein thrombosisB.INTRARENAL(a)congenital:duplicated pelvicaliceal system, crossed fused ectopia, multicystic dysplastic kidney, adult polycystic kidney (in 8% unilateral)(b)infectious:acute bacterial nephritis(c)adaptation:compensatory hypertrophyC.POSTRENAL(a)collecting system:obstructive uropathy *mnemonic:*"AROMA"**A**cute pyelonephritis **R**enal vein thrombosis **O**bstructive uropathy **M**iscellaneous (compensatory hypertrophy, duplication) **A**rterial obstruction (infarction)

Notes:

Bilateral Large Kidneys Average renal length by x-ray: M = 13 cm; F = 12.5 cm 1.PROTEIN DEPOSITIONamyloidosis, multiple myeloma 2.INTERSTITIAL FLUID ACCUMULATIONacute tubular necrosis, acute cortical necrosis, acute arterial infarction, renal vein thrombosis 3.CELLULAR INFILTRATION(a)Inflammatory cells: acute interstitial nephritis, acute bacterial nephritis(b)Malignant cells: leukemia / lymphoma4.PROLIFERATIVE / NECROTIZING DISORDERS(a)Glomerulonephritis (GN)acute (poststreptococcal) GN, rapidly progressive GN, idiopathic membranous GN, membrano-proliferative GN, lobular GN, IgA nephropathy, glomerulosclerosis, glomerulosclerosis related to heroin abuse (b)Multisystem diseasepolyarteritis nodosa, systemic lupus erythematosus, Wegener granulomatosis, allergic angiitis, diabetic glomerulosclerosis, Goodpasture syndrome (lung hemorrhage + glomerulonephritis), Schönlein-Henoch syndrome (anaphylactoid purpura), thrombotic thrombocytopenic purpura, focal glomerulonephritis associated with subacute bacterial endocarditis 5.URINE OUTFLOW OBSTRUCTIONbilateral hydronephrosis: congenital / acquired 6.HORMONAL STIMULUSacromegaly, compensatory hypertrophy, nephromegaly associated with cirrhosis / hyperalimentation / diabetes mellitus 7.DEVELOPMENTALbilateral duplication system, horseshoe kidney, polycystic kidney disease 8.MISCELLANEOUSacute urate nephropathy, glycogen storage disease, hemophilia, sickle cell disease, Fabry disease, physiologic response to contrast material and diuretics *mnemonic:*"FOG P"**F**luid:= edema of kidney (ATN, acute cortical necrosis)**O**ther:leukemia, acromegaly, sickle cell anemia, bilateral duplication, acute urate nephropathy**G**lomerular disease:acute GN, lupus, polyarteritis nodosa, diabetes mellitus**P**rotein deposition:multiple myeloma, amyloidosis

Notes:

Bilateral Small Kidneys A.PRERENAL = VASCULAR1.Arterial hypotension (acute)2.Generalized arteriosclerosis3.Atheroembolic disease4.Benign & malignant nephrosclerosisB.INTRARENAL1.Hereditary nephropathies:medullary cystic disease, hereditary chronic nephritis (Alport syndrome) 2.Chronic glomerulonephritis3.Amyloidosis (late)C.POSTRENAL1.Papillary necrosisD.CAUSES OF UNILATERAL SMALL KIDNEYoccurring bilaterally *mnemonic:*"CAPE HANA"**C**hronic glomerulonephritis **A**rteriosclerosis **P**apillary necrosis **E**mbolic disease (secondary to atherosclerosis) **H**ypotension **A**lport syndrome **N**ephrosclerosis **A**myloidosis (late)

Notes:

Unilateral Small Kidney A.PRERENAL = VASCULAR1.Lobar infarction2.Chronic infarction3.Renal artery stenosis4.Radiation nephritisB.INTRARENAL = PARENCHYMAL1.Congenital hypoplasia 2.Multicystic dysplastic kidney (in adult)3.Postinflammatory atrophyC.POSTRENAL = COLLECTING SYSTEM1.Reflux nephropathy = chronic atrophic pyelonephritis2.Postobstructive atrophy *mnemonic:*"RIP R HIP"**R**eflux atrophy **I**schemia (renal artery stenosis) **P**ostobstructive atrophy **R**adiation therapy **H**ypoplasia (congenital) **I**nfarction **P**ostinflammatory atrophy

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Notes:

Increased Echogenicity Of Renal Cortex =RENAL MEDICAL DISEASE = diffuse increase in cortical echogenicity with preservation of corticomedullary junction*Path:*deposition of collagen / calcium in interstitial, glomerular, tubular, vascular disease echointensity of cortex greater than liver / spleen ± equal to renal sinus renal size may be normal; enlarged kidneys suggest active stage of renal disease; small kidneys suggest chronic + often end-stage renal disease 1.Acute / chronic glomerulonephritis2.Renal transplant rejection3.Lupus nephritis4.Hypertensive nephrosclerosis5.Renal cortical necrosis6.Methemoglobulinuric renal failure7.Alport syndrome8.Amyloidosis9.Diabetic nephrosclerosis10.Nephrotoxin-induced acute tubular necrosis11.End-stage renal disease

Notes:

Hyperechoic Renal Pyramids In Children A.NEPHROCALCINOSIS(a)iatrogenic (most common cause):furosemide (Rx for BPD), vitamin D (Rx for hypophosphatemic rickets) (b)noniatrogenic:1.Idiopathic hypercalcemia2.Williams syndrome3.Absorptive hypercalcemia4.Hyperparathyroidism5.Milk-alkali syndrome6.Kenny-Caffey syndrome7.Distal renal tubular acidosis8.Malignant tumors9.Chronic glomerulonephritis10.Sjögren syndrome (distal RTA)11.SarcoidosisB.METABOLIC DISEASE1.Gout2.Lesch-Nyhan syndrome (urate)3.Fanconi syndrome4.Glycogen storage disease (distal RTA)5.Wilson disease (distal RTA)6.Alpha-1-antitrypsin deficiency7.Tyrosinemia8.Cystinosis9.Oxalosis10.Crohn diseaseC.HYPOKALEMIA1.Primary aldosteronism2.Pseudo-Bartter syndromeD.PROTEIN DEPOSITS1.Infant dehydration with presumed Tamm-Horsfall proteinuria2.Toxic shock syndromeE.VASCULAR CONGESTION1.Sickle cell anemiaF.INFECTION1.Candida / CMV nephritis2.AIDS-associated Mycobacterium avium-intracellulareG.FIBROSIS OF RENAL PYRAMIDSH.CYSTIC MEDULLARY DISEASE1.Medullary sponge kidney2.Congenital hepatic fibrosis with tubular ectasiaI.INTRARENAL REFLUX1.Chronic pyelonephritis

Notes:

Iron Accumulation In Kidney A.RENAL CORTEX1.Paroxysmal nocturnal hemoglobulinuria(= intravascular extrasplenic hemolysis) 2.Sickle cell anemiaB.RENAL MEDULLA1.Hemorrhagic fever with renal syndrome (uncommon viral illness caused by Hanta virus)Triad:(1)renal medullary hemorrhage(2)right atrial hemorrhage(3)necrosis of anterior pituitary

Notes:

Depression Of Renal Margins 1.Fetal lobation√ notching between normal calices2.Splenic impression√ flattened upper outer margin of left kidney3.Chronic atrophic pyelonephritis√ indentation over clubbed calices4.Renal infarct√ normal calices5.Chronic renal ischemia√ normal calices

Notes:

Enlargement Of Iliopsoas Compartment A.INFECTION(a)from retroperitoneal organs1.Renal infection2.Complicated pancreatitis3.Postoperative aortic graft infection(b)from spine1.Osteomyelitis / postoperative complication of bone surgery2.Discitis / postoperative complication from disk surgery(c)from GI tract1.Crohn disease2.Appendicitis(d)others1.Pelvic inflammatory disease / postpartum infection2.SepsisB.HEMORRHAGE1.Coagulopathy and anticoagulant therapy2.Ruptured aortic aneurysm3.Postoperative aneurysm repair / other surgery / traumaC.NEOPLASTIC DISEASE(a)Extrinsic1.Lymphoma2.Metastatic lymphadenopathy3.Bone metastases with soft-tissue involvement4.Retroperitoneal sarcoma(b)Intrinsic1.Muscle tumors2.Nervous system tumors3.Lipoma / liposarcomaD. MISCELLANEOUS1.Pseudoenlargement of psoas musclecompared to de facto atrophy of contralateral side in neuromuscular disease 2.Fluid collectionsurinoma, lymphocele, pancreatic pseudocyst, enlargement of iliopsoas bursa 3.Pelvic venous thrombosis diffuse swelling of all muscles (edema)

Notes:

Bilateral Renal Masses A.MALIGNANT TUMOR1.Malignant lymphoma / Hodgkin disease2.Metastases3.Renal cell carcinoma4.Wilms tumorB.BENIGN TUMOR1.Angiomyolipoma2.NephroblastomatosisC.CYSTS1.Adult polycystic kidney disease2.Acquired cystic kidney disease

Notes:

Renal Mass In Neonate A.UNILATERAL1.Multicystic kidney (15%)2.Hydronephrosis (25%)(a)UPJ obstruction(b)upper moiety of duplication3.Renal vein thrombosis4.Mesoblastic nephroma5.Rare: Wilms tumor, teratomaB.BILATERAL1.Hydronephrosis2.Polycystic kidney disease3.Multicystic kidney + contralateral hydronephrosis4.Nephroblastomatosis5.Bilateral multicystic kidney

Notes:

Renal Mass In Older Child A.SINGLE MASS1.Wilms tumor2.Multilocular cystic nephroma3.Focal hydronephrosis4.Traumatic cyst, abscess5.Renal cell carcinoma6.Malignant rhabdoid tumor7.Teratoma8.Clear cell sarcoma of kidney9.Intrarenal neuroblastomaB.MULTIPLE MASSES1.Nephroblastomatosis2.Multiple Wilms tumors3.Angiomyolipoma4.Lymphoma5.Leukemia6.Adult polycystic kidney disease7.Abscesses

Notes:

Growth Pattern Of Renal Tumors In Adults A.EXPANSILE GROWTH1.Renal cell carcinoma2.Oncocytoma3.Angiomyolipoma4.Juxtaglomerular tumor5.Metastatic tumor (eg, lymphoma)6.Mesenchymal tumorB.INFILTRATIVE GROWTH1.Lymphoma / leukemia2.Invasive transitional cell carcinoma3.Metastatic tumor4.Renal cell carcinoma (unusual)5.Xanthogranulomatous pyelonephritis

Notes:

Local Bulge In Renal Contour A.CYST1.Simple renal cystB.TUMOR1.Adenocarcinoma2.Angiomyolipoma3.PseudotumorC.INFECTION1.Subcapsular abscess2.XGPD.TRAUMA1.Subcapsular hematomaE.DILATED COLLECTING SYSTEM

Notes:

Unilateral Renal Mass ***Solid Renal Mass*** A.TUMORS(a)primary malignant:adenocarcinoma (83%), chromophobe carcinoma (4%), papillary neoplasm (14%), renal collecting duct carcinoma = Bellini duct carcinoma (1%), transitional cell carcinoma (8%), renal neuroendocrine tumors (carcinoid, small cell carcinoma), Wilms tumor (6%), renal sarcoma (2%) *in horseshoe kidney:* adenocarcinoma (45%), Wilms tumor (28%), transitional cell carcinoma (20%) (b)secondary malignant:malignant lymphoma / Hodgkin disease, metastases, invasive transitional cell carcinoma (c)benign:adenoma, oncocytoma, hamartoma (mesoblastic nephroma, angiomyolipoma, myolipoma, lipoma, leiomyoma, fibroma), hemangioma B.INFLAMMATORY MASSESacute focal bacterial nephritis, renal abscess, xanthogranulomatous pyelonephritis, malacoplakia, tuberculoma ***Fluid-filled Mass*** A.CYSTS1.Simple renal cyst2.Inherited cystic disease:multicystic dysplastic kidney disease (Potter type II), multilocular cystic nephroma 3.Focal hydronephrosisB.VASCULAR1.Arteriovenous malformation2.Arteriovenous fistula=single dilated artery + vein√ tortuous varices over time√ enlargement of renal vein*Cx:*hydronephrosis ◊Lesions <1 cm often cannot be clearly characterized◊Lesions 1-1.5 cm can often be ignored, particularly in elderly / patients with significant other disease

Notes:

Avascular Mass In Kidney *mnemonic:*"CHEAT"**C**yst **H**ematoma **E**dema **A**bscess **T**umor

Notes:

Hyperechoic Renal Nodule A.MALIGNANT TUMOR1.Renal cell carcinoma2.Angiosarcoma3.Liposarcoma4.Undifferentiated sarcoma5.LymphomaB.BENIGN TUMOR1.Angiomyolipoma2.Lipoma3.Oncocytoma4.Cavernous hemangiomaC.INFARCTD.HEMATOMA

Notes:

Hyperattenuating Renal Mass On NECT A.BENIGN1.Complicated benign cyst: hemorrhagic, protein-rich, gelatinous2.Leiomyoma3.Angiomyolipoma (rare)4.Thrombosed renal veinB.MALIGNANT1.Metastasis from thyroid carcinoma2.Renal cell carcinoma

Notes:

Low-density Retroperitoneal Mass 1.Lipoma√ sharply marginated, homogeneously fatty mass2.Lymphangioma√ similar to lipoma if enough fat content3.Adrenal myelolipoma√ density between fat + water√ usually nonhomogeneous, occasionally with hemorrhage ± calcifications4.Renal angiomyolipoma√ intrarenal component√ hypervascular with large feeding arteries, multiple aneurysms, laking without shunting, tortuous circumferential vessels, whorled parenchymal + venous phase5.Xanthogranulomatous pyelonephritis√ nonfunctioning kidney replaced by low-density material + central staghorn calculus6.Metastatic retroperitoneal tumors7.Renal cell carcinoma8.Fibrosarcoma, fibrous histiocytoma, mesenchymal sarcoma, malignant teratoma√ density close to muscle9.Liposarcoma

Notes:

Focal Area Of Increased Renal Echogenicity A.NONNEOPLASTIC1.Chronic renal infarction2.Acute focal bacterial nephritisB.BENIGN TUMOR1.Angiomyolipoma2.Cavernous renal hemangioma3.OncocytomaC.MALIGNANCY1.Renal cell carcinoma2.Angiosarcoma3.Undifferentiated sarcoma4.Metastasis

Notes:

Fat-containing Renal Mass 1.Angiomyolipoma2.Lipoma, liposarcoma3.Teratoma4.Wilms tumor5.Xanthogranulomatous pyelonephritis6.Oncocytoma engulfing renal sinus fat7.Renal cell carcinoma(a)invasion of perirenal fat(b)intratumoral metaplasia into fatty marrow (in 32% if RCCs <3 cm)

Notes:

Renal Sinus Mass A.TUMORS1.Transitional cell carcinoma2.Lymphoma3.Metastasis to sinus lymph nodes4.Mesenchymal tumor: lipoma, fibroma, myoma, hemangioma5.Plasmacytoma6.Myeloid metaplasiaB.MISCELLANEOUS1.Sinus lipomatosis2.Parapelvic cyst3.Saccular aneurysm4.Urinoma ***Hypoechoic Renal Sinus*** A.SOLID1.Fibrolipomatosis2.Column of Bertin3.Duplex kidney4.TCC / RCCB.CYSTIC1.Renal sinus cysts2.Caliectasis3.Dilated veins, varix4.Aneurysm, arteriovenous malformation

Notes:

Renal Pseudotumor =anomalies of lobar anatomy that may simulate a tumorA.PRIMARY1.**Large column of Bertin**
= large septum / cloison of Bertin = large cloison = focal cortical hyperplasia = benign cortical rest = focal renal hypertrophy=persistence of normal septal cortex / excessive infolding of cortex usually in the presence of partial or complete duplicationLocation:between upper and interpolar portion√ mass <3 cm in largest diameter√ lateral indentation of renal sinus√ "deformation" of adjacent calices + infundibula√ mass continuous with renal cortex√ enhancement pattern like renal cortex√ echogenicity similar to cortex2.**Dromedary hump**
=subcapsular nodule = splenic bump=secondary to prolonged pressure by spleen during fetal developmentLocation:in mid portion of lateral border of left kidney√ triangular contour + elongation of middle calyx√ enhancement pattern like renal cortex3.**Hilar lip**
=supra- / infrahilar bulge = medial part of kidney above / below sinusLocation:most frequently medial to left kidney just above renal pelvis (on transaxial scan)√ enhancement pattern like cortex with medulla4.**Fetal lobation**
= persistent cortical lobation = ren lobatus 14 individual lobes with centrilobar cortex located around calices 5.**Lobar dysmorphism**
complete diminutive lobe situated deep within renal substance with its own diminutive calyx in its central portion = calyx of nonresorbed normal junctional parenchyma between upper + lower subkidneys B.ACQUIRED1.**Nodular compensatory hypertrophy**
areas of unaffected tissue in the presence of focal renal scarring from chronic atrophic pyelonephritis (= reflux nephropathy), surgery, trauma, infarction; √ hypertrophy usually evident within 2 months; less likely to occur > age 50*DDx:*accessory spleen, medial lobule of spleen, splenosis, normal / abnormal bowel, pancreatic disease, gallbladder, adrenal abnormalities*Dx:*static radionuclide imaging / renal arteriography / CT

Notes:

Potter Classification =POTTER SYNDROME=any renal condition associated with severe oligohydramnios ● peculiar facies with wide-set eyes, parrot-beak nose, pliable low-set ears, receding chinTypeI:infantile PCKDTypeII:multicystic dysplastic kidney disease, multilocular cystic nephromaIIa:kidneys of normal / increased sizeIIb:kidneys reduced in sizeTypeIII:adult PCKD, tuberous sclerosis, medullary sponge kidneyTypeIV:small cortical cysts / cystic dysplasia secondary to ureteropelvic junction obstruction

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Notes:

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Renal Cystic Disease A.SIMPLE RENAL CYST1.Intrarenal2.ParapelvicB.POLYCYSTIC RENAL DISEASE1.Adult PCKD2.Infantile PCKD3.Glomerulocystic kidney disease=congenital disease with extremely variable presentation + prognosis*Path:*cysts within Bowman capsule ± tubular cysts ± multiple macroscopic cortical cystsC.CYSTIC MEDULLARY DISEASE1.Uremic medullary cystic disease2.Juvenile nephrophthisis3.Medullary sponge kidneyD.RENAL DYSPLASIA1.Multicystic dysplastic kidney2.Segmental / focal renal dysplasia3.Familial renal dysplasiaE.NEUROCUTANEOUS DYSPLASIA 1.Tuberous sclerosis2.Von Hippel-Lindau syndromeF.CYSTIC TUMORS1.Multilocular cystic nephroma2.Cystic Wilms tumor3.Cystic renal cell carcinomaG.ACQUIRED RENAL CYSTIC DISEASE1.Acquired cystic disease of uremia2.Infectious cysts (TB, Echinococcus, abscess)3.Medullary necrosis4.Pyelogenic cyst

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Notes:

Syndromes With Multiple Cortical Renal Cysts 1.Von Hippel-Lindau syndrome2.Tuberous sclerosis3.Meckel-Gruber syndrome4.Zellweger syndrome = cerebrohepatorenal syndrome5.Jeune syndrome6.Conradi syndrome = chondrodysplasia punctata7.Oro-facial-digital syndrome8.Trisomy 139.Turner syndrome10.Dandy-Walker malformation

Notes:

Multiloculated Renal Mass A.NEOPLASTIC DISEASE1.Cystic renal cell carcinoma2.Multilocular cystic renal tumor(a)cystic nephroma(b)cystic partially differentiated nephroblastoma3.Cystic Wilms tumor4.Necrotic tumor(a)mesoblastic nephroma(b)clear cell sarcomaB.RENAL CYSTIC DISEASE1.Localized renal cystic disease2.Septated cyst3.Multicystic dysplastic kidney3.Segmental multicystic dysplasia4.Complicated cystC.INFLAMMATORY DISEASE1.Echinococcus2.Segmental XGP3.Abscess4.MalacoplakiaD.VASCULAR LESIONS1.AV fistula2.Organizing hematoma

Notes:

Normal Nephrographic Phases 1.Vascular phase (= cortical arteriogram)=contrast material visible in interlobular arteries + glomeruliTiming after IV injection:10-15 sec (arm-to-kidney circulation time)Duration:transient vascular phase of <0.5 sec2.Cortical phase (= cortical nephrogram)=contrast medium in cortical capillaries + peritubular spaces + cortical tubular luminaTiming after IV injection:20-45 secTiming after intraarterial injection:2-3 secCT: √ exclusive renal cortical enhancement3.Parenchymal phase (= generalized / tubular nephrogram)=contrast material within loops of Henle + collecting tubulesTiming after IV injection:1-2 min (maximum)√ enhancement of both cortex and medulla4.Excretory phaseTiming after IV injection:beginning at 2-3 min

Notes:

Absence Of Nephrogram ***Global Absence Of Nephrogram*** *Pathophysiology:*complete renal ischemia secondary to occlusion of main renal artery1.Injury to vascular pedicle during blunt abdominal trauma2.Thromboembolic disease3.Renal artery dissection: spontaneous, traumatic, iatrogenic ***Segmental Absence Of Nephrogram*** A.SPACE-OCCUPYING PROCESS1.Neoplasm2.Cyst3.AbscessB.FOCAL RENAL INFARCTION1.Arterial embolus / thrombosis2.Vasculitis, collagen-vascular disease3.Sickle cell anemia4.Septic shock5.Renal vein thrombosis

Notes:

Rim Nephrogram =rim of cortex receiving collateral blood flow from capsular, peripelvic, and periureteric vessels Most specific indicator of renovascular compromise! 2-4 mm peripheral band of cortical opacification*Cause:*1.Acute total main renal artery occlusion: seen in 50% of cases with renal infarction2.Renal vein thrombosis3.Acute tubular necrosis4.Severe chronic urinary obstruction*DDx:*severe hydronephrosis (rim/shell nephrogram surrounding dilated calices)

Notes:

Unilateral Delayed Nephrogram A.OBSTRUCTIVE UROPATHYB.REDUCTION IN RENAL BLOODFLOW1.Renal artery stenosis2.Renal vein thrombosis

Notes:

Striated Nephrogram =stasis of contrast material in dilated collecting ducts on background of edematous renal parenchyma fine linear bands of alternating lucency + density parallel to axis of tubules + collecting ductsA.UNILATERAL1.Acute ureteric obstruction2.Acute bacterial nephritis / pyelonephritis3.Renal contusion4.Renal vein thrombosisB.BILATERAL1.Acute pyelonephritis2.Intratubular obstruction: Tamm-Horsfall proteinuria, rhabdomyolysis with myoglobinuria3.Systemic hypotension4.Autosomal recessive PCKD5.Medullary sponge kidney6.Medullary cystic disease *mnemonic:*"CHOIR BOY"**C**ontusion **H**ypotension (systemic) **O**bstruction (ureteral) **I**ntratubular obstruction **R**enal vein thrombosis **B**acterial nephritis (acute) **O**bstruction (ureteral) - it is so common! **Y**es, also cystic diseases: infantile PCKD, medullary cystic disease, medullary sponge kidney

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Notes:

Persistent Nephrogram A.BILATERAL GLOBAL1.Systemic hypotension2.Intratubular obstruction from protein: Tamm-Horsfall, Bence-Jones, myoglobin3.Tubular damage by contrast materialB.UNILATERAL GLOBAL1.Renal artery stenosis2.Renal vein thrombosis3.Urinary tract obstructionC.SEGMENTAL1.Obstructed moiety of duplicated collecting system2.Obstructing renal calculus3.Obstructing neoplasm4.Focal stricture5.Focal parenchymal disease: tubulointerstitial infection

Notes:

Abnormal Nephrogram Due To Impaired Perfusion A.SYSTEMIC HYPOTENSIVE REACTIONas reaction to contrast material / cardiac failure / dehydration / shock *Pathophysiology:* drop in perfusion pressure after contrast reaches kidney leads to increased salt + water reabsorption and slowed tubular transit √ prolonged bilateral dense nephrograms= persistent increasing nephrogram √ decrease in renal size√ loss of pyelogram after initial opacificationNUC (use of glomerular filtration agent [eg, Tc-99m DTPA] preferred) √ prolonged cortical transit + reduced excretion B.RENAL ARTERY STENOSIS√ decreased nephrographic opacity + rim nephrogram√ hyperconcentration in collecting system√ ureteral notchingNUC (glomerular filtration agent [eg, Tc-99m DTPA] preferred): √ decreased perfusion with prolonged excretory phaseC.IMPAIRED PERFUSION OF SMALL ARTERIESTrueta shunting = transient rerouting of blood flow from cortex to medulla *Cause:* (a)reflex spasm during arterial angiography secondary to catheter trauma / pressure injection of highly concentrated contrast medium(b)chronic renal disorders (collagen vascular disease, malignant nephrosclerosis, chronic glomerulonephritis)(c)necrotizing vasculitis (polyarteritis nodosa, scleroderma, hypertensive nephrosclerosis)CT, Angio: √ inhomogeneous opacification of cortexIVP: √ irregular cortical nephrogram = spotted nephrogramD.ACUTE VENOUS OUTFLOW OBSTRUCTIONin renal vein thrombosis √ obstructive nephrogram√ progressive increase in opacity of entire kidney

Notes:

Abnormal Nephrogram Due To Impaired Tubular Transit *Cause:* A.EXTRARENAL: ureteric obstruction (eg, stone)√ obstructive nephrogramNUC: before decrease in renal function use of glomerular filtration agent (eg, Tc-99m DTPA); with decrease in renal function use of plasma flow agents (eg, Tc-99m MAG3 / I-123 Hippuran) preferred √ continuous increase in renal activity√ dilatation of collecting systemB.INTRARENAL(a)segmental: limb of duplication system, caliceal obstruction, interstitial edema √ segmental nephrogram(b)protein precipitation: Tamm-Horsfall protein (a normal mucoprotein product of proximal nephrons), Bence Jones protein (multiple myeloma), uric acid precipitation (acute urate nephropathy), myoglobulinuria, hyperproteinuric state√ striated nephrogramNUC: before decrease in renal function use of glomerular filtration agent (eg, Tc-99m DTPA); with decrease in renal function use of plasma flow agents (eg, Tc-99m MAG3 / I-123 Hippuran) preferred √ prolonged cortical transit time + prolonged excretory phase

Notes:

Abnormal Nephrogram Due To Abnormal Tubular Function 1.Acute tubular necrosis√ immediate persistent nephrogram (common)√ progressive increasing opacity (rare)2. Contrast-induced renal failure ***Striated Angiographic Nephrogram*** =random patchy densities reflecting redistribution of blood flow from the cortical vasculature to the vasa recta of the medulla1.Obliterative diseases of the renal microvasculature:polyarteritis nodosa, scleroderma, necrotizing angiitis, catheter-induced vasospasm2.Acute bacterial nephritis3.Renal vein thrombosis ***Increasingly Dense Nephrogram*** =initially faint nephrogram becoming increasingly dense over hours to days*Mechanism:* (a)diminished plasma clearance of contrast material(b)leakage of contrast material into renal interstitial spaces(c) increase in tubular transit time *Cause:* A.VASCULAR = diminished perfusion1.Systemic arterial hypotension (bilateral)2.Severe main renal artery stenosis (unilateral)3.Acute tubular necrosis (in 33%): due to contrast material nephrotoxicity4.Acute renal vein thrombosisB.INTRARENAL1.Acute glomerular diseaseC.COLLECTING SYSTEM1.Intratubular obstruction(a)uric acid crystals (acute urate nephropathy)(b)precipitation of Bence Jones protein (myeloma nephropathy)(c)Tamm-Horsfall protein (severely dehydrated infants / children)2.Acute extrarenal obstruction: ureteral calculus

Notes:

Vicarious Contrast Material Excretion During IVP =biliary contrast material detected radiographically following intravenous administration of contrast material*Normal contrast excretion:* <2% of urographic dose of diatrizoates + iothalamates are handled by hepatobiliary excretion *Pathophysiology:* increase in protein binding due to prolonged intravascular contact + acidosis *Cause:* 1.Uremia (reduction in glomerular filtration + uremia-associated acidosis)2.Acute unilateral obstruction (increase in circulation time + transient intracellular acidosis)3.Spontaneous urinary extravasation (prolonged vascular contact of contrast material)

Notes:

Spontaneous Urinary Contrast Extravasation =SPONTANEOUS PYELORENAL BACKFLOW*Etiology:*physiologic "safety valve" for obstructed urinary tract with pressures of 80-100 mm Hg in collecting system due to ipsilateral ureteral obstruction from distal stone impaction; pressure is proportional to degree + duration of acute obstruction + dose of contrast material *Incidence:*0.1-18%; M > F (male ureter less compliant)*Criteria:* (a)absence of recent ureteral instrumentation(b)absence of previous renal / ureteral surgery(c)absence of destructive urinary tract lesion(d)absence of external trauma(e)absence of external compression(f)absence of pressure necrosis due to stone*Types:* 1.Pyelotubular backflow= opacification of terminal portions of collecting ducts (= papillary ducts = ducts of Bellini) as a physiologic phenomenon (in 13% with low osmolality + in 0.4% with high osmolality contrast media), wrongly termed "backflow"↓ wedge-shaped brushlike lines from calyx toward periphery2.Pyelosinus backflow= contrast extravasation from ruptured fornices along infundibula, renal pelvis, proximal ureter; most common form*Cx:*urinoma, retroperitoneal fibrosis3.Pyelointerstitial backflow=contrast flow from pyramids into subcapsular tubules4.Pelolymphatic backflow= contrast extravasation into periforniceal + peripelvic lymphatics↓ visualization of small lymphatics draining medially5.Pyelovenous backflow=forniceal rupture into interlobar / arcuate veins; very rare

Notes:

Widened Collecting System & Ureter *Fetal pyelectasis:* AP diameter of renal pelvis <5 mm<20 weeks MA<8 mm20-30 weeks MA<10 mm>30 weeks MAA.OBSTRUCTIVE UROPATHY1.Acute / chronic obstruction2.Obstructed upper pole moiety of duplicated systemB.NONOBSTRUCTIVE WIDENING(a)congenital1.Megacalicosisunderdevelopment of papillae, usually unilateral 2.Congenital primary megaureterwidened ureter with normally tapered distal end 3.Megacystis-megaureter syndrome4.Prune-belly syndrome(b)increased urine volume1.High-flow states: diabetes insipidus, osmotic diuresis, dehydrated patient undergoing rehydration, unilateral kidney2.Vesicoureteral reflux(c)atony of renal collecting system1.Infection: ie, acute pyelonephritis2.Pregnancy*Etiology:*? obstruction by enlarged ovarian veins / uterus; progesterone-induced decrease in ureteral tone*Incidence:*3-4% of pregnant womenTime:at end of 1st trimester, maximal in 3rd trimesterLocation:right (90%), left (67%); ureter widened only to pelvic brim*Prognosis:*resolution within a few weeks to 6 months after delivery3.Retroperitoneal fibrosis(d)distended urinary bladder(e)previous long-standing significant obstruction:dilatation remains in spite of relief of obstruction

Notes:

Caliceal Abnormalities A.OPACIFICATION OF COLLECTING TUBULES1.Pyelorenal backflow2.Medullary sponge kidneyB.PAPILLARY CAVITY1.Papillary necrosis2.Caliceal diverticulum3.Tuberculosis / brucellosisC.LOCALIZED CALIECTASIS1.Reflux nephropathy = chronic atrophic pyelonephritis2.Compound calyx3.Hydrocalyx4.Congenital megacalyx5.Localized postobstructive caliectasis6.Localized tuberculosis / papillary necrosisD. GENERALIZED CALIECTASIS1.Postobstructive atrophy2.Congenital megacalices3.Obstructive uropathy (hydronephrosis)4.Nonobstructive hydronephrosis 5.Diabetes insipidus

Notes:

Filling Defect In Collecting System *mnemonic:*"6 C's & 2 P's"**C**lot **C**ancer **C**yst **C**alculus **C**andida + other fungi **C**ystitis cystica **P**olyp **P**apilla (sloughed) ***Nonopaque Intraluminal Mass In Collecting System*** A.NONOPAQUE CALCULUSuric acid, xanthine, matrix √ smooth, rounded, not attachedB.TISSUE SLOUGH1.Papillary necrosis2.Cholesteatoma3.Fungus ball = conglomeration of fibrillar hyphae4.Inspissated debris ("mucopus")C.VASCULAR1.Blood clot: history of hematuria√ change in appearance over timeD.FOREIGN MATERIAL1.Airfrom bladder via reverse peristalsis, direct trauma, renoalimentary fistula 2.Foreign matter
Mucosal Mass In Collecting System NEOPLASTIC A.BENIGN TUMOR1.Aberrant papilla = papilla without calyx protruding into major infundibulum2.Endometriosis3.**Fibroepithelial polyp** = fibrous polyp=fibroepithelioma = vascular fibrous polyp = polypoid fibroma= mesodermal tumor with fibrovascular stroma + normal transitional cell epithelium*Age:*20-40 years ● intermittent abdominal / flank pain ● gross hematuria (rare)√ elongated cylindrical filling defect with smooth margins√ mobile on thin pedicleB.MALIGNANT TUMOR(a)Uroepithelial tumors1.Transitional cell carcinoma (85-91%)2.Squamous cell carcinoma (10-15%)*Predisposing factors:* calculi (50-60%), chronic infection, leukoplakia, phenacetin abuse √ infiltrating / superficially spreading3.Mucinous adenocarcinoma=metaplastic transformation4.Sarcoma (extremely rare)(b)Metastases: breast (most common), melanoma, stomach, lung, cervix, colon, prostate INFLAMMATION / INFECTION 1.Tuberculosis2.Candidiasis3.Schistosomiasis4.Pyeloureteritis cystica5.Leukoplakia6.Malacoplakia7.Xanthogranulomatous pyelonephritis VASCULAR 1.Submucosal hemorrhage:trauma, anticoagulant therapy, acquired circulating anticoagulants, complication of crystalluria / microlithiasis √ thumbprinting with progressive improvement2.Vascular notching:ureteropelvic varices, renal vein occlusion, IVC occlusion, vascular malformation, retroaortic left renal vein, "nutcracker" effect on left renal vein between aorta and SMA 3.Polyarteritis nodosa PROMINENT MUCOSAL FOLDS 1.Redundant longitudinal mucosal folds of intermittent hydronephrosis (UPJ obstruction, vesicoureteral reflux) or after relief of obstruction2.Chemical / mechanical irritation3.Urticaria (Stevens-Johnson syndrome= erythema multiforme bullosa) 4.Leukoplakia (= squamous metaplasia)5.Ureteral diverticulosis=rupture of the roofs of cysts in ureteritis cystica

Notes:

Effaced Collecting System A.EXTRINSIC COMPRESSION(1)Unilateral / bilateral global enlargement of renal parenchyma(2)Renal sinus masses: hemorrhage; parapelvic cyst; sinus lipomatosisB.SPASM / INFLAMMATION(1)Infection: acute pyelonephritis, acute bacterial nephritis, acute tuberculosis(2)HematuriaC.INFILTRATIONMalignant uroepithelial tumors D.OLIGURIA1.Antidiuretic state2.Renal ischemia3.Oliguric renal failure

Notes:

Retroperitoneal Calcification A.NEOPLASM1.Wilms tumor (in 10%)2.Neuroblastoma (in 50%): fine granular / stippled / amorphous3.Teratoma: cartilage / bone / teeth, pseudodigits, pseudolimbs4.Cavernous hemangioma: phlebolithsB.INFECTION1.Tuberculous psoas abscess2.Hydatid cystC.TRAUMA1.Old hematoma

Notes:

Calcified Renal Mass A calcified renal mass is malignant in 75% of cases! Lesions with (a) nonperipheral calcifications are malignant in 87%! (b) peripheral calcifications are malignant in 20%! A. TUMOR 1. Renal cell carcinoma (calcifies in 8-18%) √ calcifications generally nonperipheral, sometimes along fibrous capsule 2. Wilms tumor B. INFECTION 1. Abscess Tuberculous abscess frequently calcifies! Pyogenic abscess rarely calcifies! 2. Echinococcal cyst Renal involvement in 3% of hydatid disease; 50% of echinococcal cysts calcify 3. Xanthogranulomatous pyelonephritis √ large obstructive calculus in >70% C. CYSTS Calcification is related to prior hemorrhage or infection 1. Simple renal cyst (calcifies in 1%) 2. Multicystic dysplastic kidney (in adult) 3. Adult polycystic kidney disease 4. Milk of calcium (cyst, caliceal diverticulum, obstructed hydrocalyx) *DDx:* residual pantopaque used in cyst puncture D. VASCULAR 1. Subcapsular / perirenal hematoma 2. Renal artery aneurysm √ circular cracked eggshell appearance 3. Congenital / posttraumatic arteriovenous fistula

Notes:

Nephrocalcinosis =NEPHROLITHIASIS=calcium salts in renal parenchyma*Incidence:*0.1-6%; M > F*mnemonic:*"MARCH"**M**edullary sponge kidney **A**lkali excess **R**enal medullary / cortical necrosis, **R**TA **C**hronic glomerulonephritis **H**yperoxaluria, **H**ypercalcemia, **H**ypercalciuria
Medullary Nephrocalcinosis =calcifications involving the distal convoluted tubules in the loops of Henle*Incidence:*95% of all nephrocalcinoses*Cause:*
A.HYPERCALCIURIA(a)endocrine1.Hyperparathyroidism in 5% (primary >> secondary)2.Paraneoplastic syndrome of lung + kidney primary (ectopic parathormone production)3.Cushing syndrome4.Diabetes insipidus5.Hyperthyroidism(b)alimentary1.Milk-alkali syndrome (excess calcium + alkali = milk + antacids)2.Hypervitaminosis D3.Beryllium poisoning(c)osseous1.Osseous metastases, multiple myeloma2.Prolonged immobilization3.Progressive senile osteoporosis(d)renal1.Renal tubular acidosis (in 73% of primary RTA)2.Medullary sponge kidney3.**Bartter syndrome**
tubular disorder with potassium + sodium wasting, hyperplasia of juxtaglomerular apparatus, hyperaldosteronism, hypokalemic alkalosis, and normal blood pressure
(e)drug therapy1.Furosemide (in infants)2.Prolonged ACTH therapy3.Vitamin E (orally)4.Calcium (orally)(f)miscellaneous1.Sarcoidosis2.Idiopathic hypercalcuria3.Idiopathic hypercalcemia B.HYPEROXALURIA = OXALOSIS1.**Primary hyperoxaluria**
=Hereditary hyperoxaluria (more common)=rare autosomal recessive inherited enzyme deficiency of carboligase with diffuse oxalate deposition in kidneys, heart, blood vessels, lung, spleen, bone marrowType I=a-ketoglutarate-glyoxylate carboxylase deficiency ● glycolic aciduriaType II=D-glycerate dehydrogenase deficiency ● 1-glyceric aciduria*Age:*usually <5 years*Prognosis:*early death in childhood2.**Secondary hyperoxaluria**
=enteric hyperoxaluria (rare)*Cause:*disturbance of bile acid metabolism after jejunoileal bypass, ileal resection, blind loop syndrome, Crohn disease, increased ingestion (green leafy vegetables), pyridoxine deficiency, ethylene glycol poisoning, methoxyflurane anesthesia C.HYPERURICOSURIA1.Gouty kidney2.Lesch-Nyhan syndromeD.URINARY STASIS1.Milk-of-calcium in pyelocaliceal diverticulum2.Medullary sponge kidneyE.DYSTROPHIC CALCIFICATION1.Renal papillary necrosis
mnemonic:"HAM HOP"**H**yperparathyroidism **A**cidosis (renal tubular) **M**edullary sponge kidney **H**ypercalcemia / hypercalciuria (sarcoidosis, milk-alkali syndrome, hypervitaminosis D) **O**xalosis **P**apillary necrosis √ normal-sized / occasionally enlarged kidneys (medullary sponge kidney)√ grouped rounded / linear calcifications√ small poorly defined / large coarse granular calcifications in renal pyramidsUS: √ absence of hypoechoic papillary structures (earliest sign)√ hyperechoic rim at corticomedullary junction + around tip and sides of pyramids√ solitary focus of hyperechogenicity at tip of pyramid near fornix√ increased echogenicity of renal pyramids ± shadowing (no acoustic shadowing with small + light calcifications)*DDx of hyperechoic medulla in newborns:* oliguria with transient tubular blockage by Tamm-Horsfall proteinuria *Cx:*often followed by urolithiasis ***Cortical Nephrocalcinosis*** *Incidence:*5% of all nephrocalcinoses*Cause:* 1.Acute cortical necrosis2.Chronic glomerulonephritis3.Alport syndrome = hereditary nephritis + deafness4.Congenital oxalosis, primary hyperoxaluria5.Chronic paraneoplastic hypercalcemia6.Rejected renal transplant*mnemonic:*"COAG"**C**ortical necrosis (acute) **O**xalosis **A**lport syndrome **G**lomerulonephritis (chronic) US: √ homogeneously increased echogenicity of renal parenchyma > liver echogenicity

Notes:

Renovascular Hypertension =normalization of blood pressure following nephrectomy / reestablishment of normal renal blood flow (Dx made in retrospect)*Incidence:*1-5% of general population; 2nd most common cause of potentially curable hypertension*Pathophysiology:* usually >50% stenosis at any level in renovascular bed leads to mildly reduced pressure in glomerular afferent arteriole (pressure falls precipitously in >80% stenosis); reduced pressure stimulates release of renin followed by angiotensin-II, and aldosterone causing (a)constriction of efferent glomerular arterioles(b)increase in systemic hypertension(c)sodium retention*Cause:* 1.Atherosclerosis (60-90%) in individuals >50 years of age2.Fibromuscular dysplasia (10-35%) in women <40 years of age3.Neurofibromatosis4.Pheochromocytoma5.Fibrous bands (congenital stenosis, retroperitoneal fibrosis, postradiation artery stenosis)6.Arteritis (Buerger disease, polyarteritis nodosa, Takayasu disease, thrombangitis obliterans, syphilitic arteritis)7.Arteriovenous malformation / fistula8. Thromboembolic disease (eg, atrial fibrillation, prosthetic valve thrombi, cardiac myxoma, paradoxical emboli, atheromatous emboli)9. Renal artery aneurysm10.Extrinsic compression (eg, renal cyst, neoplasm, perirenal hematoma)11.Middle aortic syndrome, aortic dissection, dissecting aortic aneurysm12.Trauma Renal artery stenosis is present in 77% of hypertensive patients! Renal artery stenosis is present in 32-49% of normotensive patients! 15-20% of patients remain hypertensive after restoration of normal renal blood flow!*Rx:*(1)Relieving renal artery stenosis(2)Angiotensin-converting enzyme inhibitor ***Hypertension In Children*** *Prevalence:*1-3%1.Coarse renal cortex scarring (36%)2.Glomerulonephritis (23%)3.Coarctation of aorta (10%)4.Renovascular disease (10%)5.Polycystic renal disease (6%)6.Hemolytic-uremic syndrome (4%)7.Catecholamine excess [pheochromocytoma, neuroblastoma] (3%)8.Renal tumor (2%)9.Essential hypertension (3%)

Notes:

Renal Aneurysm A.EXTRARENAL ANEURYSM (2/3)1.Congenital2.Atherosclerotic3.Fibromuscular dysplasia4.Mycotic2.5% of all aneurysms *Cause:*bacteremia, SBE, perivascular extension of inflammation*Organism:*Streptococcus, Staphylococcus, Pneumococcus, SalmonellaLocations:thoracic aorta, SMA, peripheral branches of middle cerebral artery, large arteries of extremities, intrarenal (rare), in areas of preexisting vascular disease5.Neurofibromatosis6.Trauma + renal artery angioplastyB.INTRARENAL ANEURYSM (1/3)in interlobar and more peripheral branches 1.Congenital renal aneurysm*Age at Dx:* 30 years; M:F = 1:1 ● hypertension in 25% (from segmental renal ischemia)√ aneurysm close to vascular bifurcations, may calcify2.Atherosclerotic (may calcify)3.Polyarteritis nodosa4.SLE5.Drug-abuse vasculitisKidney most commonly affected organ *Cause:* (a)immunologic injury from circulating hepatitis antigen-antibody complexes producing a necrotizing angiitis(b)bacterial endocarditis(c)drug-related(d)impurity-relatedDrugs:methamphetamine, heroin, LSD√ multiple small aneurysms in interlobar branches near corticomedullary junction√ inhomogeneous spotty nephrogram6.Allergic vasculitis7.Neoplasm (renal cell carcinoma in 14%; adult Wilms tumor)8.Hamartoma (angiomyolipoma in 50%)9.Wegener granulomatosis10.Metastatic arterial myxoma11.Transplant rejection12.Neurofibromatosis*Cx:*(1) Hypertension (unusual) (2) Perinephric / retroperitoneal hemorrhage (3) Formation of AV fistula (4) Peripheral renal embolization(5) Thrombosis

Notes:

Spontaneous Renal Hemorrhage A.RENAL TUMOR (57-63%)(a)malignant (30-33%):RCC, TCC of renal pelvis, Wilms tumor, lipo-, fibro-, angiosarcoma (b)benign (24-33%):angiomyolipoma (16-20%), lipoma, adenoma, fibromyoma, ruptured hemorrhagic cyst B.VASCULAR DISEASE (18-26%)vasculitis (eg, polyarteritis nodosa in 13%), arteriovenous malformation, ruptured aneurysm, segmental renal infarction C.INFLAMMATION / INFECTION (7-10%)1/2 with + 1/2 without abscess D.COAGULOPATHYanticoagulation therapy, bleeding diathesis, long-term hemodialysis◊Surgical exploration must be considered to uncover a small renal tumor if the cause of hemorrhage is not determined radiologically! ***Subcapsular Hematoma*** √ subcapsular mass with flattening of renal parenchyma √ total resorption / formation of pseudocapsule with calcificationAngio: √ avascular mass*Cx:*Page kidney (ischemia, release of renin, hypertension)

Notes:

Renal Doppler A.NORMAL RENAL DOPPLER↓ resistive index (RI) of 0.70 = upper limit of normalElevation of RI: -significant systemic hypotension-markedly decreased heart rate-perinephric / subcapsular fluid collection-in neonates + infantsB.RENAL MEDICAL DISEASEElevation of RI more likely with vascular / tubulointerstitial process, less likely with glomerular disease May be useful in predicting clinical outcome in: -hemolytic-uremic syndrome-acute renal failure-nonazotemic patients with severe liver diseaseC.RENAL ARTERIAL STENOSISD.RENAL VEIN THROMBOSIS

Notes:

Ureteral Deviation A.LUMBAR URETER(a)lateral deviation (common):1.Hypertrophy of psoas muscle2.Enlargement of paracaval / para-aortic lymph nodes3.Aneurysmal dilatation of aorta4.Neurogenic tumors5.Fluid collections (abscess, urinoma, lymphocele, hematoma)(b)medial deviation:1.Retrocaval ureter (on right side only)2.Retroperitoneal fibrosisB.PELVIC URETER(a)medial deviation:1.Hypertrophy of iliopsoas muscle2.Enlargement of iliac lymph nodes3.Aneurysmal dilatation of iliac vessels4.Bladder diverticulum at UVJ (Hutch)5.Following abdominoperineal surgery + retroperitoneal lymph node dissection6.Pelvic lipomatosis(b) lateral deviation with extrinsic compression1.Pelvic mass (eg, fibroids, ovarian tumor)

Notes:

Megaureter A. VESICOURETERAL REFLUX(a)primary vesicoureteral reflux1.Primary reflux megaureterabnormal ureteral tunnel at UVJ 2.Prune belly syndrome(b)secondary vesicoureteral reflux1.Hypertonic neurogenic bladder2.Bladder outlet obstruction3.Posterior urethral valvesB.OBSTRUCTION(a)primary obstruction1.Intrinsic ureteral obstruction (stone, stricture, tumor)2.Ectopic ureter3.Ureterocele4.Ureteral duplication: tortuous dilated ureter of upper moiety(b)secondary obstruction1.Retroperitoneal obstruction: tumor, fibrosis, aortic aneurysm2.Bladder wall mass3.Bladder outlet obstruction: eg, prostatic enlargementC.NONREFLUX-NONOBSTRUCTED MEGAURETER1.Congenital primary megaureter = megaloureter2.Polyuria: eg, diabetes insipidus, acute diuresis3.Infection4.Ureter remaining wide after relief of obstruction *mnemonic:*"DiaPOUR"**D**iabetes insipidus **P**rimary megaureter **O**bstruction (recent / old) **U**VJ obstruction **R**eflux

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Notes:

Ureteral Stricture A.INTRINSIC CAUSE(a)mucosal1.Primary ureteral tumors(b)mural1.**Endometriosis** common disorder in menstruating women (15%); ureteral involvement is rare and indicates widespread pelvic disease√ abrupt smooth stricture of 0.5-2.5 cm length√ rectosigmoid involvement on BE2.Tuberculosis, schistosomiasis3.Traumaticureterolithotomy, endoscopic stone extraction, hysterectomy 4.Amyloidosis√ distal stricture with submucosal calcification5.Nonspecific (rare)B.EXTRINSIC CAUSE1.Endometriosisextrinsic form:intrinsic form = 4:1 2.Abscesstubo-ovarian, appendiceal, perisigmoidal 3.Inflammatory bowel disease(eg, Crohn disease, diverticulitis) 4.Radiation fibrosis5.Metastasescervix, endometrium, ovary, rectum, prostate, breast, lymphoma 6.Iliac artery aneurysm (with perianeurysmal fibrosis) *mnemonic:*"MISTER"**M**etastasis (extrinsic / intrinsic) **I**nflammation from calculus **S**chistosomiasis **T**uberculosis, **T**ransitional cell carcinoma, **T**rauma **E**ndometriosis + other periureteral inflammatory process **R**adiation therapy, **R**etroperitoneal fibrosis

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Notes:

Ureteral Filling Defect A.FIXED1.Urothelial neoplasm2.Metastasis3.Inflammation(a)ureteritis cystica(b)tuberculosis4.Fibroepithelial polyp5.EndometriosisB. MOBILE 1.Calculus2.Sloughed papilla3.Blood clot

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Notes:

Adrenal Medullary Disease 1.Neuroblastoma2.Ganglioneuroblastoma3.Ganglioneuroma4.Pheochromocytoma

Notes:

Adrenal Cortical Disease 1.Adrenal hyperplasia2.Adrenocortical adenoma3.Adrenocortical carcinoma4.Cushing syndrome5.Conn syndrome6.Adrenogenital syndrome ***Adrenocortical Hyperfunction*** 1.Cushing syndrome = hypercortisolism2.Conn syndrome = hyperaldosteronism√ solitary unilateral adrenal adenoma + normal contralateral gland on CT may be due to:(a)aldosterone-producing adrenocortical adenoma(b)renin-responsive aldosterone-producing adenoma(c)idiopathic hyperaldosteronism with dominant hyperplastic / nonfunctional adenoma3.Adrenogenital syndrome *DDx of Cushing syndrome* A.FOCAL UNILATERAL ADRENAL MASS√ 2-4 cm focal mass in one adrenal gland + atrophy of contralateral gland = adrenal adenoma√ >4 cm large focal mass with central necrosis in one adrenal gland + atrophy of contralateral gland = adrenal adenocarcinomaB.BILATERAL ADRENAL ENLARGEMENT√ diffuse uniform thickening = Cushing diseaseC.MULTIPLE BILATERAL ADRENAL NODULES√ macronodules = multinodular hyperplasia of long-standing Cushing disease√ large nodules (autonomous ACTH-independent) = massive macronodular hyperplasia√ small nodules = primary pigmented nodular adrenal disease

Notes:

Bilateral Large Adrenals *mnemonic:*"4 H PM"**H**odgkin disease **H**yperplasia **H**emorrhage **H**istoplasmosis / TB **P**heochromocytoma **M**etastasis

Notes:

Unilateral Adrenal Mass ◊CT attenuation<0HU=benign mass0-15HU=probably benign>15HU=indeterminate◊on 15-minute-delayed CECT scan:<25 HU benign lesion, >25 HU malignant lesion *Cause:*rapid contrast washout from benign lesions*mnemonic:*"PLAN My HAM"**P**heochromocytoma **L**ymphoma **A**denoma **N**euroblastoma **My**elolipoma **H**emorrhage **A**denocarcinoma **M**etastasis ***Small Unilateral Adrenal Tumor*** ◊Incidental discovery of adrenal mass in 1% of all CT!(a)mass <3 cm in diameter is likely (in 87%) benign(b)mass >5 cm in diameter is likely malignant1.Cortical adenoma (in 1-9% of autopsies)√ <10 HU imply (in 96%) an adenoma2.Metastasis (27% of all tumors): lung (40%), breast(20%), renal cell carcinoma, gastrointestinal tumors, melanoma◊50% of adrenal masses in oncologic patients represent benign nonhyperfunctioning adenomas!3.Pheochromocytoma4.Asymmetric hyperplasia5.Granulomatous disease (TB, histoplasmosis)√ diffuse enlargement / discrete mass√ ± central cystic changes ± calcification6.Myelolipoma: rare benign tumor composed of hematopoietic cells + fat similar to bone marrow ● may cause pain if large√ typically between -30 to -115 HU√ calcified in up to 20%*Cx:*retroperitoneal hemorrhage ***Large Solid Adrenal Mass*** 1.Cortical carcinoma2.Pheochromocytoma3.Neuroblastoma / ganglioneuroma4.Myelolipoma5.Metastasis6.Hemorrhage7.Inflammation8.Abscess (eg, histoplasmosis, tuberculosis)9.Hemangioma

Notes:

Cystic Adrenal Mass 1.Pseudocyst: old hemorrhage / infarction2.Vascular cystic space (endothelial lining): lymphangioma, hemangioma3.True cyst (epithelial lining): glandular cyst, embryonal cyst, mesothelial inclusion cyst4.Parasitic cyst: hydatid cyst5.Hemorrhagic complication / degeneration of a tumor:cystic adenoma, cystic pheochromocytoma, cystic adenomatoid tumor, cystic adrenocortical carcinoma, schwannoma6.Neuroblastoma (rare)7.Cortical adenoma with low density

Notes:

Adrenal Calcification A.TUMOR1.Neuroblastoma2.Pheochromocytoma3.Adrenal adenoma4.Adrenal carcinoma5.DermoidB.VASCULAR1.Hemorrhage (neonatal, sepsis)C.INFECTION1.Tuberculosis2.Histoplasmosis3.Waterhouse-Friderichsen syndromeD.ENDOCRINE1.Addison disease (TB) E.OTHERS1.Wolman disease

Notes:

Bilateral Narrowing Of Urinary Bladder A.WITH ELEVATION OF BLADDER FLOOR1.Pelvic lipomatosis2.Pelvic hematoma*Cause:*trauma, anticoagulant therapy, spontaneous rupture of blood vessels, blood dyscrasia (rare), bleeding neoplasm (rare)3.Chronic cystitisB.WITH SUPERIOR COMPRESSION OF BLADDER1.Thrombosis of IVC*Cause:*trauma, hypercoagulability state (oral contraceptives), extension of thrombi from lower extremity, abdominal sepsis, Budd-Chiari syndrome, compression of IVC by neoplasm√ collaterals through gonadal veins, ascending lumbar veins, vertebral plexus, retroperitoneal veins, portal vein (via hemorrhoidal veins)√ notching of distal ureter by ureteral veins2.Pelvic lymphadenopathy*Cause:*lymphoma (most often)√ polycyclic asymmetric compression of bladder √ medial displacement of pelvic segment of ureters√ lateral displacement of upper ureters3.Hypertrophy of iliopsoas muscles4.Bilateral pelvic masses(a)bilateral lymphocysts (following radical pelvic surgery)(b)bilateral urinomas(c)bilateral pelvic abscesses ***Pear-shaped Urinary Bladder*** *mnemonic:*"HALL"**H**ematoma **A**neurysm (bilateral common / external iliac artery) **L**ipomatosis **L**ymphadenopathy (pelvic)

Notes:

Small Bladder Capacity *Cause:* A.Thickened / fibrotic bladder wall1.Interstitial cystitis2.Tuberculous cystitis3.Cystitis cystica4.Schistosomiasis5.Trauma: surgical resection, radiation therapyB.Disuse of bladder ● urinary frequency ● progressive rise in bladder pressure during filling√ reduced bladder compliance√ thickened bladder wall + decreased bladder volume√ vesicoureteral reflux

Notes:

Bladder Wall Thickening Normal bladder wall thickness (regardless of age + gender): <5 mmin nondistended bladders<3 mmin well-distended bladdersA.TUMOR1.Neurofibromatosis B.INFECTION / INFLAMMATION1.Cystitis C. MUSCULAR HYPERTROPHY 1.Neurogenic bladder2.Bladder outlet obstruction (eg, posterior urethral valves)D. UNDERDISTENDED BLADDER

Notes:

Urinary Bladder Wall Masses A.CONGENITAL1.Congenital septum 2.Simple ureterocele3.Ectopic ureteroceleB.BLADDER TUMORSC.INFLAMMATION / INFECTION1.Cystitis: hemorrhagic ~, abacterial ~, bullous ~, edematous ~, interstitial ~, eosinophilic ~, granulomatous ~, emphysematous ~, cystitis cystica, cyclophosphamide cystitis, cystitis glandularis (premalignant lesion with villous lesions in bladder dome from proliferation of "intestine-like" glands in submucosa)2.Tuberculosis3.Schistosomiasis4.Malacoplakia5.Extravesical inflammation:(a)Diverticulitis(b)Crohn disease(c)endometriosisD.HEMATOMAafter instrumentation, surgery, trauma

Notes:

Bladder Tumor A.EPITHELIAL TUMORS (95%)1.Transitional cell carcinoma (90%)multicentric, aniline dyes 2.Squamous cell carcinoma (4%)worst prognosis; secondary to chronic disorders (infection, stricture, calculi), bladder diverticula, schistosomiasis 3.Adenocarcinoma (1%)most common in bladder exstrophy, less common in cystitis glandularis + urachal carcinoma (at dome of bladder in urachal remnant) B. NONEPITHELIAL TUMORS (a) primary benign tumors 1.Leiomyoma (most common) ● hematuria secondary to ulcerationSite:submucosal / intramural / subserosal2.Rhabdomyoma (rare)3.Hemangioma4.Neurofibroma / neurofibromatosisgeneralized neurofibromatosis in 60% 5.Nephrogenic adenoma*Associated with:*cystitis cystica / cystitis glandularis6.Endometriosison posterior wall, urinary symptoms in 80% 7.Pheochromocytoma (0.5%)from paraganglia of bladder wall; 7% are malignant ● adrenergic attack at micturition / bladder filling (headaches, weakness) ● intermittent hypertension ● elevated catecholamine levels(b) primary malignant tumors 1.Primary lymphoma2nd most common nonepithelial tumor of urinary bladder *Age:*40 years; M:F = 1:3Location:submucosal; at bladder base + trigone2.Rhabdomyosarcoma1st and 2nd decade of life 3.Leiomyosarcomararely at trigone; mainly >40 years of age (c) secondary tumors 1.Metastases1.5% of all bladder malignancies *Origin:*melanoma > stomach > breast > kidney > lung solitary / multiple nodules2.Lymphomabladder involved at autopsy: for NHL in 15%, for Hodgkin disease in 5% 3.Leukemiamicroscopic involvement in 22% at autopsy 4.Direct extension (common)from prostate, rectum, sigmoid, cervix, ovary

Notes:

Bladder Wall Calcification A.INFLAMMATION1.Schistosomiasis (50%)√ relatively normal distensibility2.Tuberculosis√ bladder markedly contracted3.Postirradiation cystitis4.Bacillary UTI (extremely uncommon)B.NEOPLASMTCC, squamous cell carcinoma, leiomyosarcoma, hemangioma, neuroblastoma, osteogenic sarcoma
mnemonic:"SCRITT"**S**chistosomiasis **C**ytoxan **R**adiation **I**nterstitial cystitis **T**uberculosis **T**ransitional cell carcinoma

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Notes:

Masses Extrinsic To Urinary Bladder A.NORMAL / ENLARGED ORGANS1.Uterus, leiomyomatous uterus, pregnant uterus2.Distended rectosigmoid3.Ectopic pelvic kidney4.Prostate cancer / BPHB.SOLID PELVIC TUMORS1.Lymphadenopathy2.Bone tumor from sacrum / coccyx3.Rectosigmoid mass4.Hip arthroplasty5.Neurogenic neoplasm, meningomyelocele6.Pelvic lipomatosis / liposarcoma C.CYSTIC PELVIC LESIONS(a) congenital / developmental 1.Urachal cyst2.Müllerian duct cyst3.Gartner duct cyst4.Anterior meningocele5.Hydrometrocolpos(b) related to trauma 1.Hematoma (eg, rectus sheath hematoma)2.Urinoma3.Lymphocele4.Abscess5.Aneurysm6.Mesenteric cyst(c) cyst of genitalia 1.Prostatic cyst2.Cyst of seminal vesicle3.Cyst of vas deferens4.Ovarian cyst5.Hydrosalpinx6.Vaginal cyst(d)cyst of urinary bladder1.Bladder diverticulum(e) cyst of GI tract 1.Peritoneal inclusion cyst2.Fluid-filled bowel

Notes:

VOIDING DYSFUNCTION

A.FAILURE TO STORE URINE ● urinary frequency, urgency, incontinence(a)bladder causes1.involuntary detrusor contractions-detrusor instability (idiopathic / neurogenic)-detrusor hyperreflexia (upper cord lesion)2.poor bladder compliance-detrusor hyperreflexia-bladder wall fibrosis3.sensory urgency-infection, inflammation, irritation-neoplasia4.vesicovaginal fistula5.psychogenic condition(b)sphincter causes1.Stress incontinence2.Sphincteric incontinence(c)extravesical ectopic insertion of ureter in femalesB.FAILURE TO EMPTY BLADDER ● poor flow, straining, hesitancy ● inability to completely empty bladder(a)bladder causes1.Detrusor areflexia (sacral arc lesion)2.Impaired detrusor contractility (myogenic)3.Psychogenic condition(b)bladder outlet obstruction:1.Bladder neck contracture2.Prostatic enlargement3.Detrusor-external sphincter dyssynergia4.Scarring from surgery / radiation therapy5.Ectopic ureterocele6.Urethral stenosis7.Urethral kinking (eg, due to cystocele)

Incontinence Prostatic Obstruction

Notes:

Incontinence 1.Stress incontinence2.Vesicovaginal / ureterovaginal fistula3.Overflow incontinencesecondary to lesions of sacral spinal cord / sacral reflex arc or severe outlet obstruction 4.Reflex voiding(a)hyperreflexive lesion (lesion of upper spinal cord)(b)uninhibited / unstable bladder5.Urge incontinence6.Continual dribbling(extravesical ectopic termination of ureter)7.Psychogenic incontinence ***Stress Incontinence*** =SPHINCTER WEAKNESS INCONTINENCE*Cause:* A.Female: congenital bladder neck weakness, pregnancy, childbirth, aging (secondary to changes in anatomic relationship of urethra + bladder base)B.Male: S/P prostatectomy with damage to distal sphincter ● frequency, urgency (involuntary filling of bladder neck)√ opening of bladder neck during coughing√ impairment of milk-back mechanism (= retrograde emptying of urethra during interruption of voiding phase does not occur)√ urethrovesical descent (in types I + II)Chain cystography: √ posterior urethrovesical angle (= angle between posterior urethra + bladder base) increased >100°√ upper urethral axis (= angle between upper urethra + vertical line) increased >35° ***Detrusor Instability***
=MOTOR URGE INCONTINENCE = UNSTABLE BLADDERCondition resembles that of immature bladder before toilet training Patient groups: (1)symptoms of nocturnal enuresis + frequency / incontinence dating back to childhood(2)idiopathic instability occurring in middle age(3)outflow obstruction commonly in men(4)degenerative instability secondary to cardiovascular + neurologic disease later in life ● frequency, urgency, urge incontinence, occasionally nocturia ● hesitancy + difficulty in voiding may occur in men without significant prostatic hypertrophy√ involuntary bladder contractions with no relationship to bladder distension√ progressively vigorous contractions during bladder filling√ postural instability limited to upright position√ impaired milk-back due to high bladder pressure√ strong aftercontractions following bladder emptying*Cx:*thickening of bladder wall, bladder diverticula*Rx:*treatment of obstruction, anticholinergic drug (oxybutynin), operative increase in bladder capacity
Sensitive Bladder (Sensory Urgency) *Cause:* cystitis (reduced compliance), some cases of stress incontinence (filling of bladder neck induces urgency) ● frequency, urgency, sometimes nocturia√ patient uncomfortable with low bladder filling√ no abnormal rise in bladder pressure√ normal voiding function
Detrusor-sphincter Dyssynergia =overactivity of bladder neck muscle with failure to relax at beginning of voiding*Cause:*spinal cord lesion / trauma above level of sacral outflow ● difficulty in voiding ± frequency ● lifelong history of poor stream√ collarlike indentation of bladder neck during voiding(= persistent / intermittent narrowing of membranous urethra)√ may have high voiding pressure + reduced flow√ trapping of contrast in urethra during interruption of flow√ massive reflux into prostatic ducts during voiding (due to high pressure within prostatic urethra)√ severely trabeculated "Christmas-tree" bladder + bilateral hydroureteronephrosis*Rx:*bladder neck incision ***Hinman Syndrome*** = NONNEUROGENIC NEUROGENIC BLADDER [NNNB] = DETRUSOR-SPHINCTER DYSSYNERGIA *Cause:*no neurologic / anatomic obstructive disease; distinctly abnormal family dynamics (in 50%)*Age:*some time after toilet training with onset during early / late childhood / puberty ● clinical criteria:(1)intact perineal sensation + anal tone(2)normal anatomy + function of lower extremities(3)absence of skin lesions overlying sacrum(4)normal lumbosacral spine at plain radiography(5)normal spinal cord at MR imaging√ high-pressure uninhibited detrusor contractions√ lack of coordination between detrusor contraction + periurethral striated sphincter relaxation√ inability to suppress bladder contractions√ normal response of detrusor muscle to reflex stimulation√ increased bladder capacity + pressure√ sphincter activity may increase paradoxically during detrusor contractionUS: √ trabeculated bladder√ dilatation of upper urinary tracts√ renal damageVCUG: √ urethra normal during early voiding√ urethral distension after contraction of external sphincter as voiding progresses√ ureterovesical obstruction / reflux*Rx:*suggestion therapy + hypnosis, bladder retraining, biofeedback, anticholinergic drugs

Notes:

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Prostatic Obstruction =urethral compression by hypertrophic prostatic tissue ● difficulty in voiding ● reduction in flow rate√ high-pressure bladder√ slow + prolonged flow√ increase in bladder capacity with reduced contractility (late)

Notes:

BLADDER TRAUMA

1.Bladder contusion (most common injury)2.Interstitial bladder injury (uncommon)=bladder tear without serosal involvement3.Bladder rupture(a)intraperitoneal rupture (30%)(b)extraperitoneal rupture(c)combined intra- and extraperitoneal rupture (5%)

Notes:

Acutely Symptomatic Scrotum =acute unilateral scrotal swelling ± pain*Cause:* epididymitis:torsion = 3:2<20 years of ageepididymitis:torsion = 9:1>20 years of age A.TORSION1.Torsion of testis (20%)=most common acute process in prepubertal age2.Torsion of testicular appendagesaccounts for 5% of scrotal pathology; both located near upper pole of testes *Frequency:* appendix testis:appendix epididymis = 9:1 √ 8-9 mm complex mass in superior aspect of scrotum without color Doppler flow signals√ mildly enlarged epididymis (75%)√ blood flow increased in epididymis (60%), scrotal wall (53%), testis (13%) simulating acute epididymo-orchitis3.Scrotal fat necrosis4.Strangulated herniaB.INFECTION / INFLAMMATION (75-80%)1.Acute epididymitis=most common acute process in postpubertal age2.**Orchitis** *Etiology:* (a)bacterial infection(b)complication of mumps in 20%:in adolescents + young adults; usually developing 4-5 days later; unilateral involvement in >90%; parotitis precedes orchitis in 84%, simultaneous in 3%, later in 4%, without parotitis in 10% 3.Intrascrotal abscessC.HEMORRHAGE1.Testicular traumaLocation:hematoma in scrotal wall, between layers of tunica vaginalis (= hematocele), in epididymis, in testis√ rapid change in echo character over time√ disruption of tunica albuginea (= testicular rupture)2.Hemorrhage into testicular tumorD.STRANGULATED HERNIA

Notes:

Scrotal Wall Thickening 1.Acute idiopathic scrotal edema*Incidence:*20-30% of all acute scrotal disorders*Age:*5-11 years (range 18 months to 14 years) ● subcutaneous scrotal edema, erythema ● minimal pain, afebrile, peripheral eosinophilia2.Epididymo-orchitis3.Testicular torsion4.Torsion of testicular / epididymal appendage5.Trauma6.Henoch-Schönlein purpura7.Cx of ventriculoperitoneal shunt8.Cx of peritoneal dialysis (? leakage of fluid into the anterior abdominal wall + dissection into scrotum)

Notes:

Scrotal Gas 1.Fournier gangrene2.Scrotal abscess3.Scrotal hernia with gas-containing bowel4.Scrotal emphysema from bowel perforation5.Extension of subcutaneous emphysema6.Air leakage + dissection due to faulty chest tube positioning

Notes:

Scrotal Mass Most frequent conditions: 1.Inflammation(48%)2.Hydrocele(24%)3.Torsion(9%)4.Varicocele(7%)5.Spermatocele(4%)6.Cysts(4%)7.Malignant tumor(2%)8.Benign tumor(0.7%)◊Sonographic differentiation of intra- from extratesticular mass is 80-95% accurate! A.INTRATESTICULAR MASS◊90-95% of testicular tumors are malignant!1.Malignant tumor2.Inflammation: focal orchitis3.Abscess4.Testicular infarction: torsion, endocarditis, trauma, leukemia, vasculitis, embolus ● soft to palpation√ hypoechoic wedge-shaped peripheral defect5.Hematoma6.Benign gonadal tumor7.Granulomatous disease: sarcoidosis8.Testicular cyst / tunica albuginea cyst9.Postbiopsy defect10.Adrenal rest ● increase in circulating corticotropin√ bilateral eccentric nodular masses ± acoustic shadowing B.MULTIPLE INTRATESTICULAR MASSES1.Primary testicular tumor2.Lymphoma / leukemia3.Chronic infections4.Metastases5.Sarcoidosis◊The prevalence of synchronous / metachronous bilateral testicular neoplasms is 1-3%! C.EXTRATESTICULAR FLUID COLLECTION1.Hydrocele, pyocele, hematocele (surgery, trauma, neoplasm)2.Varicocele3.Spermatocele=cyst filled with fluid + spermatozoa + cellular debris ● frequently following vasectomyLocation:commonly in head of epididymis √ up to a few cm in size ± septations4.Epididymal cyst=cyst without spermatozoaLocation:anywhere within epididymis5.Scrotal hernia D.PARATESTICULAR MASS◊ Only 4% of all scrotal tumors!(a)inflammatory mass1.Sarcoidosis of epididymis2.Inflammatory nodule of epididymitis3.Sperm granuloma*Cause:*sperm extravasation with granuloma formation4.Scrotal calculi = "scrotal pearls"*Cause:*fibrinous debris in long-standing hydrocele / following torsion of appendix testis or epididymis(b)paratesticular tumor◊The majority of paratesticular tumors are derived from the spermatic cord!-Benign Paratesticular tumor (70%)1.Cord lipoma (vast majority)2.**Adenomatoid tumor** (30%)=benign slow-growing mesothelial neoplasm *Age:*2nd-4th decade*Histo:*epithelial-like cells + fibrous stromaLocation:epididymis (particularly in globus minor), tunica albuginea, spermatic cord (rare)√ well-marginated solid mass with echogenicity equal to / greater than testis√ 0.4-5.0 cm in size3.Epidermoid inclusion cyst4.Polyorchidism5.Others: herniated omentum, adrenal rest, carcinoid, papillary cystadenoma of epididymis, cord leiomyoma, cord fibroma (= reactive nodular proliferation of paratesticular tissues), adrenal rest, cholesteatoma-Malignant Paratesticular Tumor (3-16%)1.Sarcomas:◊Sarcomas are the most common spermatic cord tumors after lipomas!(a)primarily in adults: undifferentiated sarcoma (30%), leiomyo-, lipo-, fibro-, myxochondro-sarcoma(b)children: embryonal sarcoma, rhabdomyosarcoma (20%)2.Mesothelioma of tunica (in 15% malignant)3.Metastases

Prepubertal Testicular Mass A.Germ cell tumors (70-90%): yolk sac tumor, teratomaB.Interstitial cell tumors: Leydig / Sertoli cell tumor, gonadoblastomaC.Leukemia, lymphoma, metastasesD.Others: adrenal rest, lipoma, hematoma, histiocytosis, tuberculous orchitis

Notes:

Calcification Of Male Genital Tract A.VAS DEFERENS1.Diabetes mellitus: in muscular outer layer2.Degenerative changes3.TB, syphilis, nonspecific UTI: intraluminalB.SEMINAL VESICLESgonorrhea, TB, schistosomiasis, bilharziasis C.PROSTATEcalcified corpora amylacea, TB

Notes:

Cystic Lesions Of Testis *Incidence:*4-10% (increasing with age) ● asymptomaticA.NONNEOPLASTIC1.**Testicular cyst**
● nonpalpable*Often associated with:*spermatoceleLocation:related to rete testis (in 92%)2.**Tunica albuginea cyst**
● palpable√ solitary small marginally located cyst3.**Intratesticular tubular ectasia**
=DILATATION OF RETE TESTIS*Age:*middle-aged to elderly*Often associated with:*spermatocele ● nonpalpableLocation: mediastinum testis√ elliptical hypoechoic mass with branching tubular structures ± cysts4.Congenital cystic dysplasia of testis (extremely rare)B.NEOPLASTIC◊24% of all testicular tumors have cystic component! ● palpable√ in combination with solid elements *DDx:*hematoma, inflammation, seminoma, Leydig cell tumor

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Notes:

Epididymal Enlargement With Hypoechoic Foci 1.Epididymitis2.Sperm granulomas3.Tuberculosis4.Lymphogranuloma venereum5.Granuloma inguinale6.Filarial granuloma7.Fungal disease8.Lymphoproliferative disease9.Metastases

Notes:

Cystic Lesions Of Epididymis 1.Epididymal cyst*Incidence:*in up to 40%*May be associated with:*intratesticular tubular ectasia√ single / multiple / bilateral*DDx:*loculated hydrocele2.Spermatocele√ may contain low-level echoes3.Cystic degeneration of epididymis

Notes:

Seminal Vesicle Cyst A.CONGENITAL*associated with:*renal dysgenesis, collecting system duplication, ectopic ureter, vas deferens agenesisB.ACQUIRED1.Autosomal dominant polycystic kidney disease bilateral seminal vesicle cysts2.Invasive bladder tumor3.Infection4.Benign prostatic hypertrophy5.Ejaculatory duct obstruction

Notes:

Large Utricle 1.Prune belly syndrome2.Imperforate anus of high type3.Down syndrome4.Hypospadia5.Posterior urethral valves

Notes:

Prostatic Cysts 1.**Müllerian duct cyst**
from remnants of paramesonephric (= müllerian) duct which has regressed by 3rd fetal month *Prevalence:*4-5% of male newborns; in 1% of men*Age:*discovered in 3rd-4th decade ● obstructive / irritative urinary tract symptoms ● suprapubic / rectal pain ● hematuria ● infertility (most common cause of ejaculatory duct obstruction)Location:arise from region of verumontanum slightly lateral to midline◊No communication with genital tract / urethra√ large intraprostatic cyst usually with extension superolaterally above prostate√ aspirate contains serous / mucous clear brown / green fluid (hemorrhage + debris), NOT spermatozoa√ rarely contains calculi*Cx:*infection, hemorrhage, carcinomatous transformation2.**Utricle cyst**
Secondary to dilatation of prostatic utricle (sometimes believed to be a remnant of the müllerian duct) *Age:*1st-2nd decade ● postvoid dribbling ● obstructive / irritative urinary tract symptoms ● suprapubic / rectal pain ● hematuria*Often associated with:* hypospadia, intersex disorders, incomplete testicular descent, ipsilateral renal agenesis Location:arise in midline from verumontanum◊Free communication with urethra√ 8- to 10-mm long cyst usually√ NO extension above prostate*Dx:*endoscopic catheterization with aspiration of white / brown fluid occasionally containing spermatozoa*Cx:*infection, hemorrhage, carcinomatous metaplasia3.**Ejaculatory duct cyst**
*Cause:*congenital / acquired obstruction of ejaculatory duct ● perineal pain, dysuria, ejaculatory pain ● hematospermiaLocation:along expected course of ejaculatory duct√ intraprostatic cyst within central zone√ aspirate contains spermatozoa with normal testicular function√ cyst commonly contains calculi√ cystic dilatation of ipsilateral seminal vesicle√ contrast injection into cyst outlines seminal vesicle4.**Cystic degeneration of BPH**
Most common cystic lesion of prostate Location: transition zone√ usually small cyst within nodules of benign prostatic hyperplasia 5.**Retention cyst**
=dilatation of glandular acini*Cause:*acquired obstruction of glandular ductule*Age:*5th-6th decadeLocation: transition / central / peripheral zone √ 1- to 2-cm smooth-walled unilocular cyst6.**Cavitary / diverticular prostatitis**
*Cause:*fibrosis of chronic prostatitis constricts ducts leading to stagnation of exudate + breakdown of intraacinar septa with cavity formation ● history of long-standing inflammatory condition √ "Swiss cheese" prostate7.**Prostatic abscess**
*Age:*5th-6th decade ● fever, chills ● urinary frequency, urgency, dysuria, hematuria ● perineal / lower back pain ● focally enlarged tender prostate√ hypo- / anechoic mass with irregular wall + septations8.Parasitic cyst (Echinococcus, bilharziasis)9.Cystic carcinoma ● hemorrhagic aspirate√ solid tissue invaginating into cyst

Notes:

Hypoechoic Lesion Of Prostate 1.Adenocarcinoma (35%)2.Benign prostatic hyperplasia (18%)√ rarely may originate in the peripheral zone3."Normal" prostatic tissue (18%)(a)cluster of prostate retention cysts(b)prominent ejaculatory ducts4.Acute / chronic prostatitis (14%)5.Granulomatous prostatitis (0.8%): most frequently due to Calmette-Guérin bacillus (BCG)6.Atrophy (10%) ● occurs in 70% of young healthy men◊May be confused with carcinoma histologically!7.Prostatic dysplasia (6%)

Notes:

Cowper (Bulbourethral) Gland Lesions Analogous to Bartholin glands in females *Prevalence:*2.3% (autopsy)Location:within urogenital diaphragm1.Retention cyst*Cx:*prenatal death from urinary obstruction2.Infectious / traumatic cyst ● asymptomatic (most) ● hematuria, bloody urethral discharge ● postvoid dribbling

Notes:

Urethral Tumors ***Benign Urethral Tumor*** 1.**Fibroepithelial polyp**
in child / young adult; transitional cell epithelium √ solitary, pedunculated fingerlike filling defect attached near verumontanum*Cx:*bladder outlet obstruction2.**Transitional cell papilloma**
older patient; in prostatic / bulbomembranous urethra; frequently associated with concomitant bladder papillomas 3.**Adenomatous polyp**
young men; adjacent to verumontanum *Histo:*columnar epithelium from aberrant prostatic epithelium ● hematuria4.**Penile squamous papilloma / condyloma acuminata**
in 5% of patients with cutaneous disease (glans penis) √ verrucous lesion in distal urethra, rarely extension into bladder5.Others:caruncle, urethral mucosal prolapse, inflammatory tags (in female)
Malignant Urethral Neoplasm *Incidence:*6th-7th decade, M:F = 1:5A.FEMALE ● urethral bleeding ● obstructive symptoms ● dysuria ● mass at introitus1.Squamous cell carcinoma (70%):distal 2/3 of urethra 2.Transitional cell carcinoma (8-24%):posterior 1/3 of urethra 3.Adenocarcinoma (18-28%):from periurethral glands of Skene B.MALE ● palpable urethral mass ● periurethral abscess ● obstructive symptoms ● cutaneous fistula ● bloody dischargeSite:bulbomembranous urethra (60%); penile urethra (30%); prostatic urethra (10%)1.Squamous cell carcinoma (70%)secondary to chronic urethritis from venereal disease (44%) + urethral strictures (88%)
2.Transitional cell carcinoma (16%)part of multifocal urothelial neoplasia, in 10% after cystectomy for bladder tumor 3.Adenocarcinoma (6%)in bulbous urethra originating in glands of Cowper / Littre 4.Melanoma, rhabdomyosarcoma, fibrosarcoma (rare)5.Metastases from bladder / prostatic carcinoma (rare)

Notes:

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AMBIGUOUS GENITALIA

=external genitalia that are not clearly of either sex*Prevalence:*1:1,000 live births ● cryptorchidism ● labial fusion ● clitoromegaly ● epi- / hypospadia*Cause:* A.Abnormal hormone levels1.congenital adrenal hyperplasia2.transplacental passage of hormones3.true hermaphroditismB.Anomalies of external genitalia not hormonally mediated (eg, micropenis) SEX=what a person is biologically; sex assignment based on (1) karyotype (2) gonadal biopsy (3) genital anatomyGENDER=what a person becomes socially

Female Pseudohermaphroditism Male Pseudohermaphroditism Gonadal Dysgenesis True Hermaphroditism

Notes:

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Female Pseudohermaphroditism =FEMALE INTERSEX*Cause:*exposure to excessive androgens in 1st trimester due to(a) congenital adrenogenital syndrome (b)maternal drug ingestion (progestational agents, androgens)(c) masculinizing ovarian tumor Karyotype: 46,XX ● masculinized external genitalia ● penislike clitoris (due to prominent corpora cavernosa + corpus spongiosum) ● rugose labioscrotum ● uterus + vagina may be filled with urine through urogenital sinus√ normal ovaries, fallopian tubes, uterus, vagina√ enlarged adrenal glands (adrenal hyperplasia)√ no testicular tissue / internal wolffian duct derivatives

Notes:

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Male Pseudohermaphroditism *Cause:*within fetal testis(a)decreased testosterone synthesis(b)decreased dihydrotestosterone production(= substance responsible for masculinization of external genitalia) due to 5a-reductase deficiency(b)no testosterone production due to early destruction / dysgenesis of testes(c)complete / incomplete androgen insensitivity due to androgen receptor defect (= testicular feminization)Karyotype:46,XY ● incompletely masculinized / ambiguous external genitalia[● apparent hypergonadotropic primary amenorrhea]√ commonly undescended normal / mildly defective bilateral testes√ prostatic tissue√ no müllerian duct derivatives (production of müllerian regression factor by testes not affected)√ occasionally blind-ending vaginal pouch emptying into perineum (= pseudovagina) / through urethra (= urogenital sinus)

Notes:

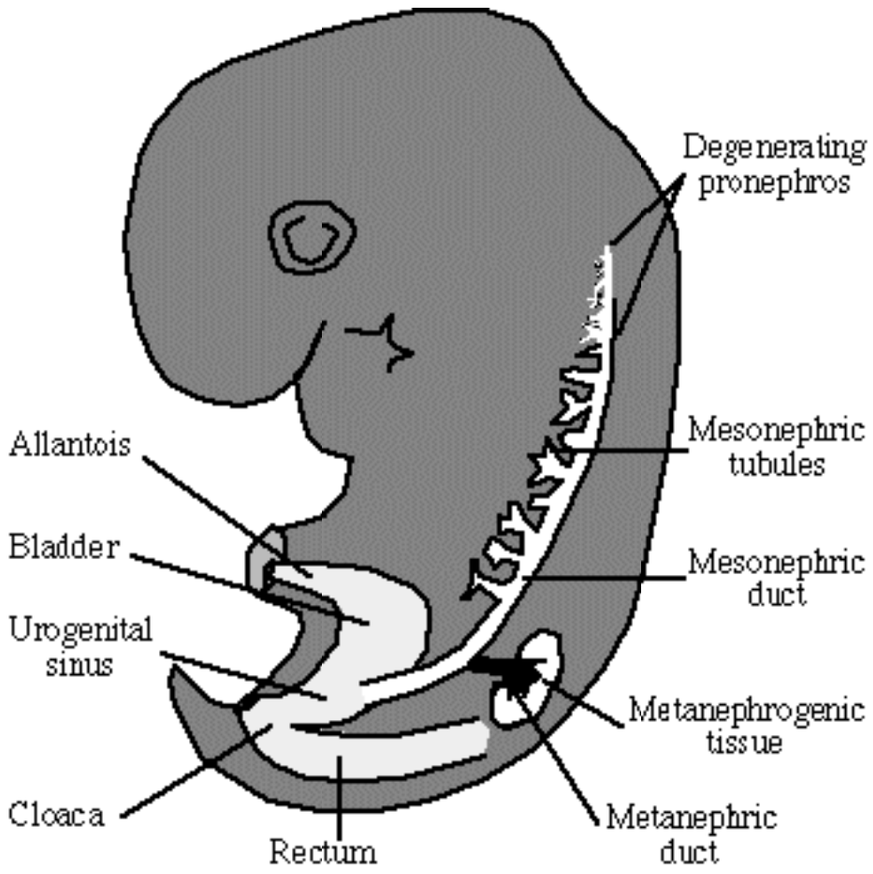
Gonadal Dysgenesis characterized by abnormal gonadal organization and function with gonads often partially / completely replaced by fibrous stroma (1)Mixed gonadal dysgenesis=testis on one side + gonadal streak on other sideKaryotype:45,XO/46,XY karyotype or other mosaics with a Y chromosome ● ambiguous external genitalia√ small / rudimentary uterus + vagina√ fallopian tube present on side of streak gonad√ urogenital sinus commonly empties at base of phallus√ dysgenetic gonads (with inability to secrete müllerian regression factor)*Cx:*gonadal neoplasia(2)Pure XY gonadal dysgenesisKaryotype:46, XY√ bilateral streak gonads / dysgenetic testes√ müllerian + wolffian duct derivatives both absent / partially developed(3)XY gonadal agenesis= vanishing testes syndrome = testicular resorption in early fetal life of unknown cause Karyotype:46,XY ● ambiguous external genitalia / female phenotype√ absent testes√ müllerian + wolffian duct derivatives both absent / partially developed

Notes:

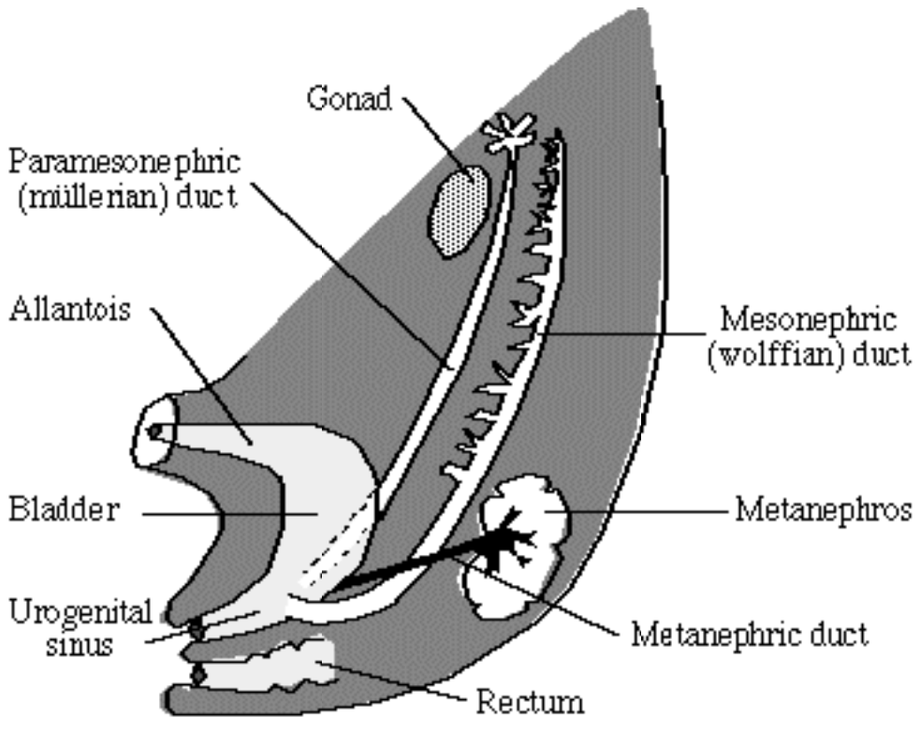
True Hermaphroditism = TRUE INTERSEX =condition characterized by presence of ovarian + testicular tissue either separate or in same gonad(= ovotestis in 64%)*Gonads:*(a)ovary on one + testis on other side (30%)(b)ovary / testis on one + ovotestis on other side (50%)(c)bilateral ovotestes (20%)Location:in pelvis (predominantly ovarian tissue); in scrotum / inguinal region (predominantly testicular tissue)*Incidence:*rare (500 cases in world literature); <10% of all intersex conditions*Age:*diagnosed within first 2 decades (75%)*Karyotype:*46,XX (80%) / 46,XY (10%) / mosaicism (10%)*Classification:* ClassI:normal female genitalia (80%)ClassII:enlarged clitorisClassIII:partially fused labioscrotal foldsClassIV:fused labioscrotal foldsClassV:hypoplastic scrotum + penoscrotal hypospadiaClassVI:normal male genitalia ● ambiguous external genitalia ● inguinal hernia ● lower abdominal pain (due to endometriosis) ● lower abdominal tumor (dysgerminoma, myomatous uterus)Reared as boy: ● cryptorchidism ● short penis ● slight degree of hypospadia ● urogenital sinus at base of penis ● penile urethra (extremely rare) ● effective spermatogenesis (rare)Reared as girl: ● development of breasts ● hematuria (= menstruation via urogenital sinus opening) in 50% ● internal female organs + female fertility ● amenorrhea ● separate urethral + vaginal openings (uncommon) √ hypoplastic uterus (in virtually 100%)√ ovotestis with heterogeneous appearance due to combination of testicular tissue + ovarian follicles√ internal gonadal duct fits the gonad:√ deferent duct on side of testis√ fallopian tube on side of ovary√ ipsilateral fallopian tube absent (suppression of development by fetal testis)√ testis / testicular portion of ovotestis usually dysgenetic

Notes:

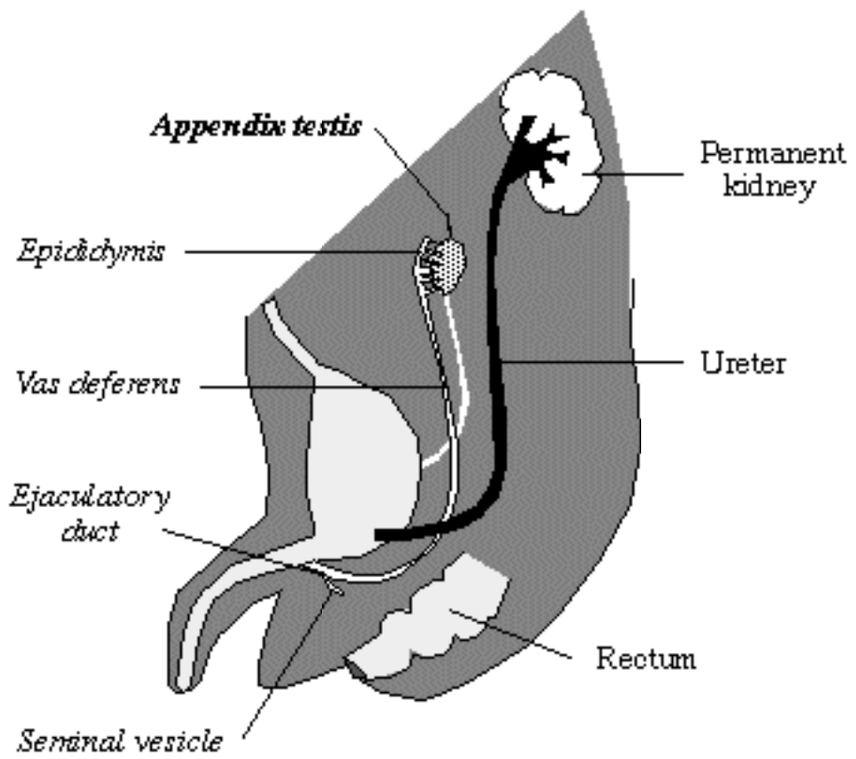
UROGENITAL EMBRYOLOGY



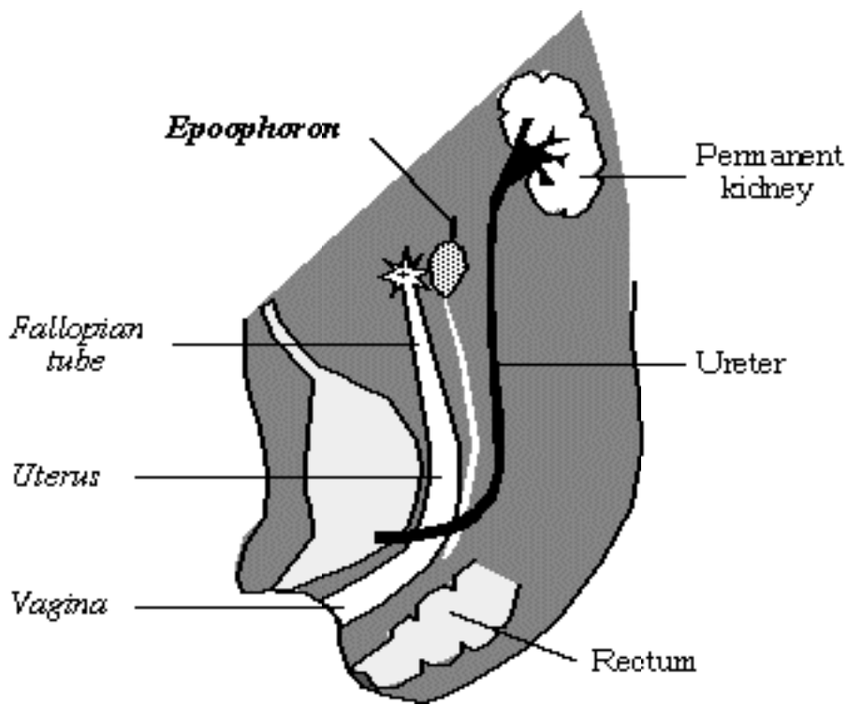
Embryo at 6th week



Embryo at 7th week



Male metanephros differentiation

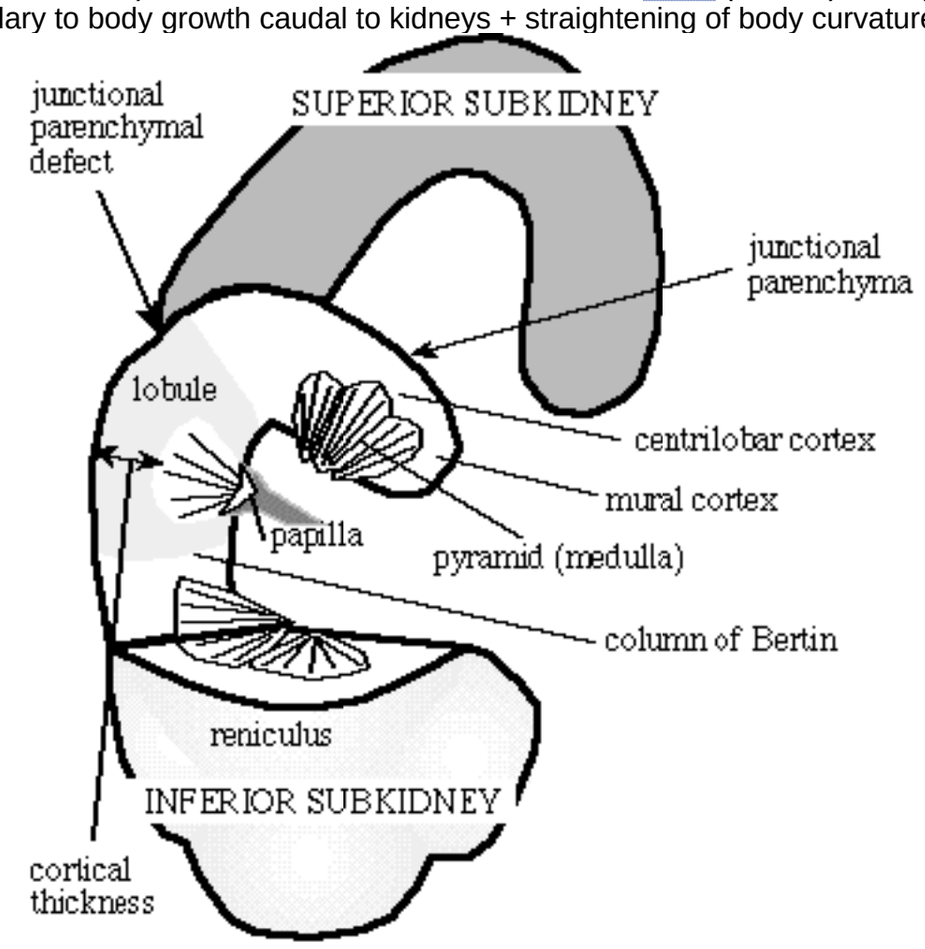


Female metanephros differentiation

Pronephros = Forekidney develops from mesoderm during 3rd week of gestation; involutes during 4th week of gestation; →vestigial remnant / completely absent **Mesonephros = Midkidney** develops during 4th week of gestation immediately caudal to pronephros, functions as interim kidney; degenerates around 8 weeks of gestation (a)mesonephric tubules →paradidymis, epididymis, efferent ductules (M); epinephron (F) (b)mesonephric (wolffian) duct →appendix epididymis, vas deferens, ejaculatory duct, seminal vesicles (M); vanishes (F) ***Paramesonephric (Müllerian) Duct*** (grows along mesonephric duct) Male:degenerates due to production of Müllerian inhibiting factor (MIF) by Sertoli cells of testis at about 6 weeks GA, remnants are prostatic utricle + appendix testisFemale:induced by wolffian duct at 5 weeks GA; grow caudally + join in midline + fuse with outgrowth of urogenital sinus; uterus, fallopian tubes **Metanephros = Hindkidney** = permanent kidney (1)metanephric diverticulum (**ureteric bud**) buds from mesonephric duct near its entry into the cloaca at 4th week; it grows toward nephrogenic cord which becomes the metanephric blastema + divides and forms→ureter (mesonephric duct)→renal pelvis (first 4 dividing generations of duct)→calices (second 4 dividing generations of duct)→collecting tubules (10-12 generations of duct)(2)**metanephric blastema** (= nephrogenic mesoderm) forms nephrons under the influence of ureteral bud, ie, the end of collecting tubules induce clusters of metanephric blastema cells(3)**metanephric vesicles** form within clusters of metanephric blastema cells + elongate into S-shaped tubules which, by 12th week of gestation, result in→glomerulus→proximal convoluted tubule→loop of Henle→distal convoluted tubule◊Polycystic kidney disease is believed to be a failure of linkage! **Urogenital Sinus** forms from cloaca →develops into bladder + urethra (+ prostate)

Notes:

Adult Kidney -forms by fusion of superior + inferior subkidneys(= metanephric lobes); the line of fusion runs obliquely forward and upward √ separation of upper + lower groups of calices√ indentation of cortical contour + echogenic line(= interrenicular septum = **junctional parenchymal defect**) delineates junctional parenchyma (often referred to as hypertrophic column of Bertin) -consists of 20,000 lobules within 14 lobes (reniculi)-initially located in pelvic region ventral to sacrum, ascending cranially at 9 weeks of gestation secondary to body growth caudal to kidneys + straightening of body curvature-renal hilum at first ventrally located, eventually rotating



Renal anatomy

medially by 90 degrees with renal ascent ***Reniculus*** = renal lobe =central core of medullary tissue enveloped by(a)centrilobar cortex (= cortical arch) that covers the base of the pyramid subsequently forming the renal cortex with loss of grooves(b)mural cortex that wraps around sides of pyramid and fuses with the mural cortex of adjacent lobe to form renal septum (= column of Bertin)√ ren lobatus (= interlobar surface grooves) present in fetus + infant, rare in adulthood

Notes:

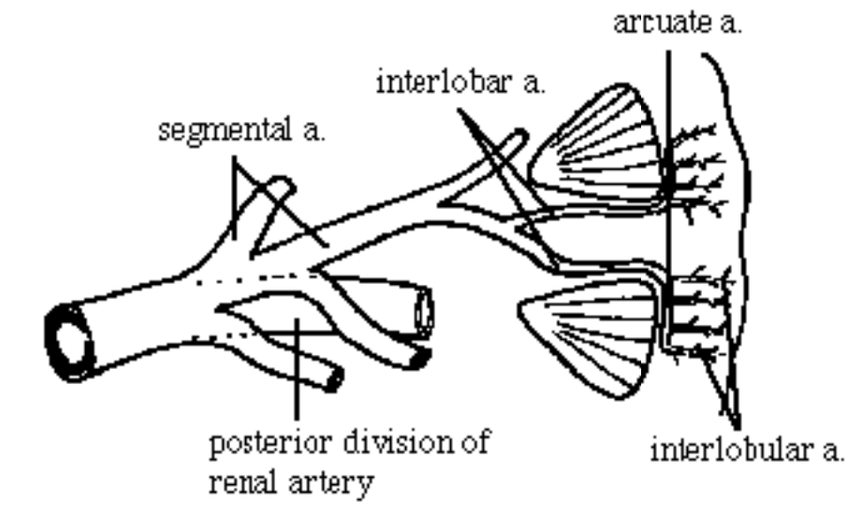
Renal Size (in cm)
-<1 year of age:4.98 + 0.155 x age (months)->1 year of age:6.79 + 0.22 x age (years)-adulthood:R kidney 10.74 ± 1.35 (SD);L kidney 11.10 ± 1.15 (SD);-ratio of renal length (RL) to distance between first 4 lumbar transverse processes (4TP) = 1.04 ± 0.22

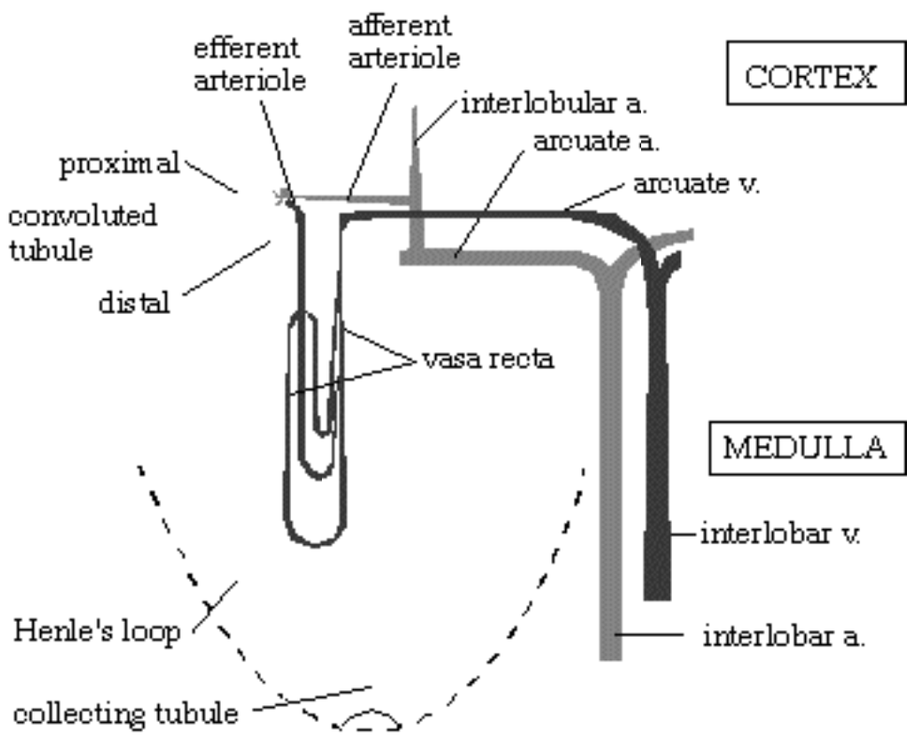
Notes:

Renal Echogenicity -neonate (up to 6 months of age): cortex may be more echogenic than adjacent normal liver / spleen (glomeruli occupy larger percentage of cortex in neonate)-adult: liver ≥ spleen ≥ renal cortex > renal medulla-renal sinus echogenicity less prominent in neonate because of paucity of fat

Notes:

Renal Vascular Anatomy 1st order:main renal artery2nd order:anterior + posterior division at / before hilum3rd order:5 segmental branches for each divisionAccessory renal artery =segmental arteries originating from the aortaAberrant renal artery =segmental artery arising from superior mesenteric artery / internal spermatic arteryResistive index:<0.701 SD of several measurements = 0.04

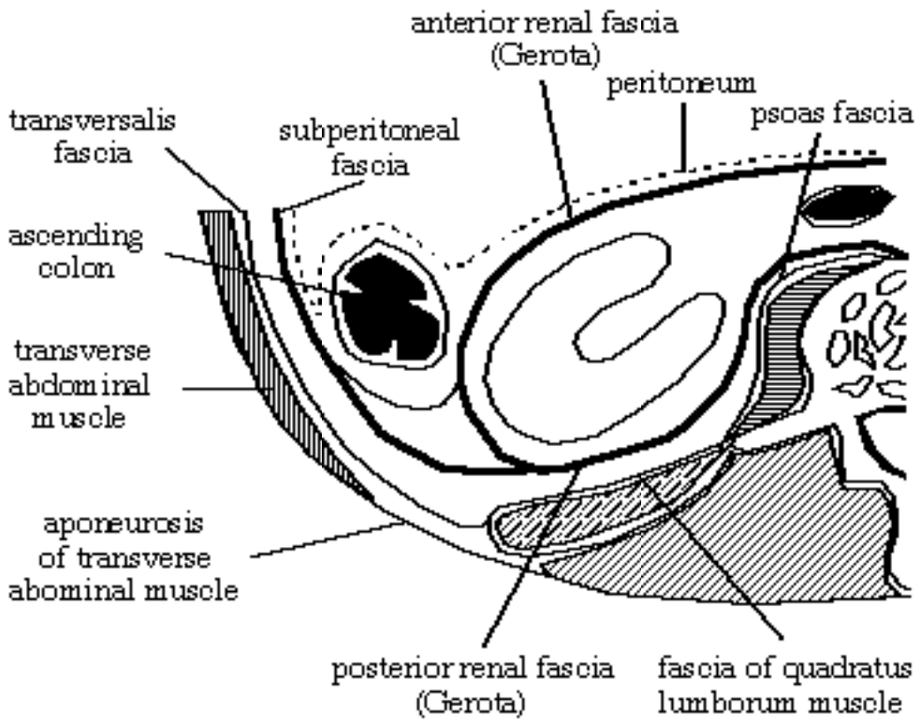




Renal Parenchymal Blood Supply

Notes:

Perirenal Compartments A.Anterior border:anterior renal fasciaB.Anterior pararenal space→superiorly joins with posterior renal fascia and attaches to crux of diaphragm→ in the middle blends with connective tissues of central prevertebral space around great vessels →inferiorly joins with posterior renal fascia and attaches to great vesselsC.Perirenal spacesubdivided into multiple compartments by incomplete bridging septa that attach to anterior + posterior renal fascia →forms inverted cone around adrenal gland + perirenal fat + upper half of kidney→forms cone around perirenal fat + lower pole of kidney→medially open communicating with central prevertebral spaceD.Posterior pararenal spaceE.Posterior border:posterior renal fascia (attaches to psoas muscle)



Gerota's fascia

Notes:

Antidiuretic Hormone (ADH)
Production site:supraoptic nuclei of hypothalamus, transported to neurohypophysisStimulus:fluid loss with increase in osmolalityEffects:(1)10 x increase in permeability of collecting ducts (= concentrated urine)(2)decreased blood flow through vasa recta leads to increased hypertonicity of interstitium (= countercurrent multiplier mechanism)

Notes:

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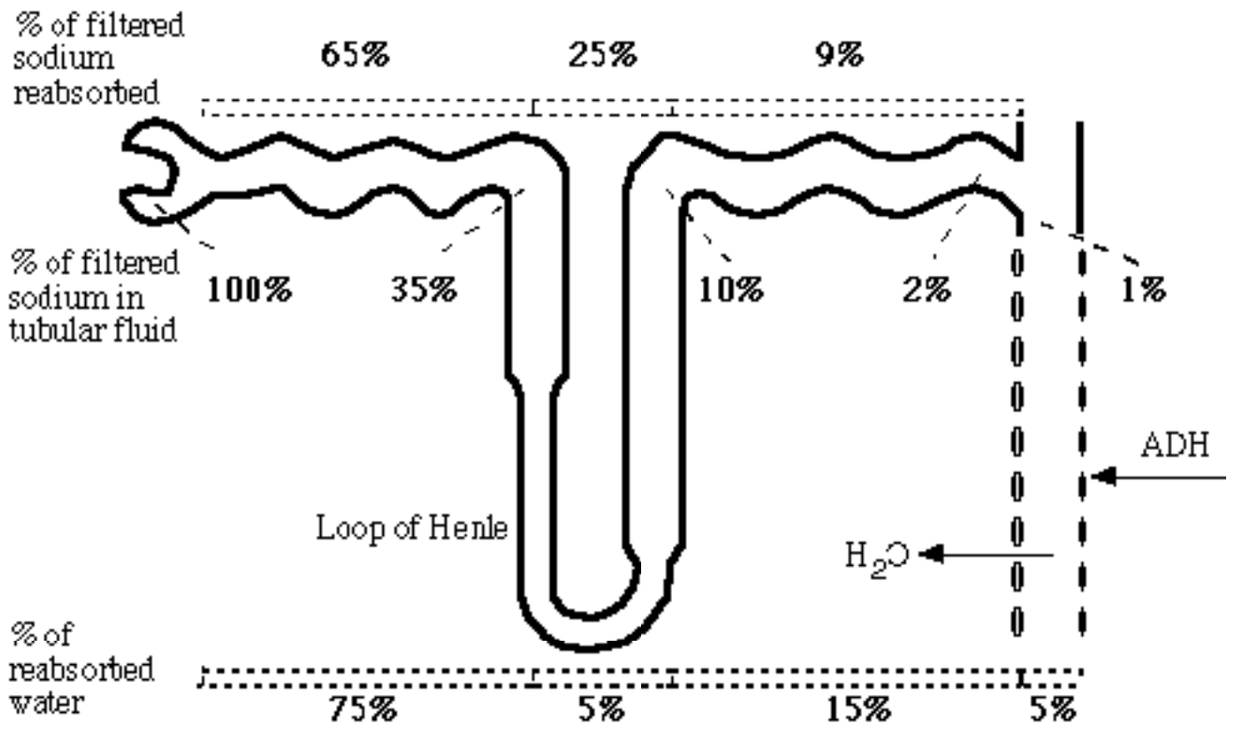
Renin-aldosterone Mechanism receptors in juxtaglomerular apparatus register the intraglomerular capillary hydraulic pressure, which is one of the main determinants of the glomerular filtration rate (GFR); the receptors regulate the release of **renin** as an autoregulatory feedback mechanism to maintain the intraglomerular hydraulic pressure; renin mediates conversion of angiotensin to angiotensin-I, which is then cleaved by a converting enzyme into angiotensin-II; **Angiotensin-II** effect: (a)constriction of efferent postglomerular arterioles, which increases intraglomerular capillary hydraulic pressure + GFR(b)systemic arteriolar constriction (= most potent vasoconstrictor of biologic systems), which causes systemic hypertension(c)release of **aldosterone**, which increases sodium retention by renal tubules-leads to an increase in blood volume + pressure if both kidneys are affected-leads to compensatory natriuresis if only one kidney is affected ACE inhibitors (eg, captopril) produce a dramatic decrease in blood pressure!

Notes:

RENAL PHSYIOLOGY

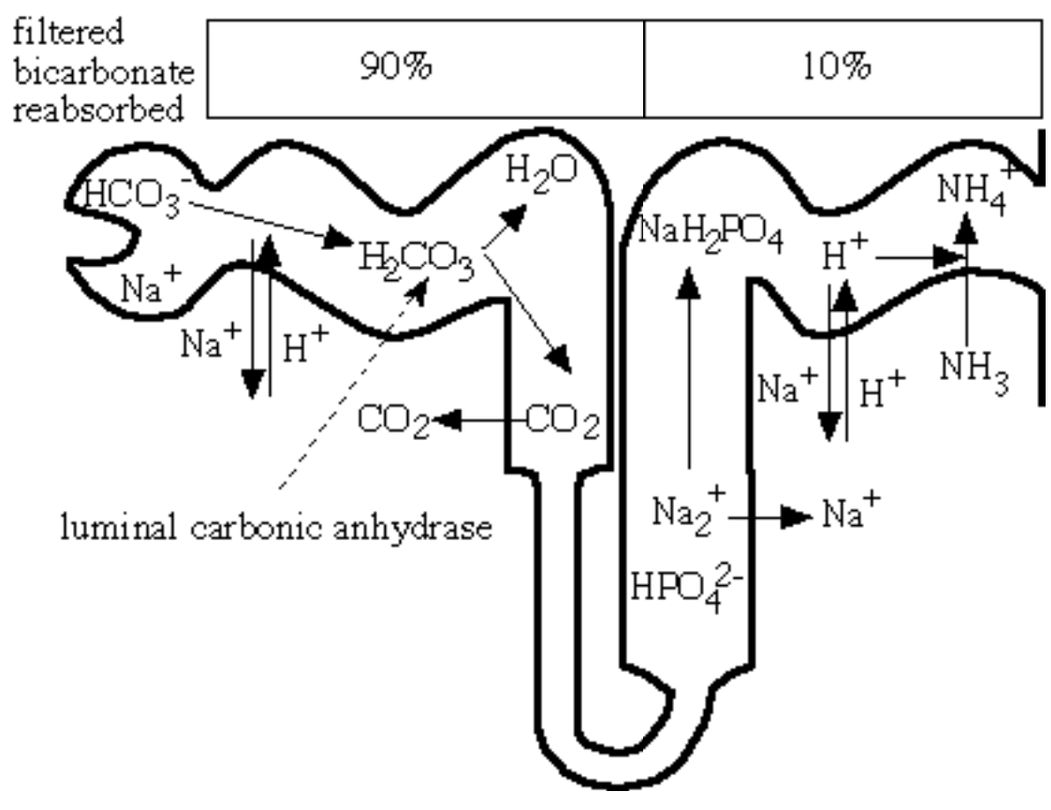


Perfusion:1.2-1.3 L of blood per minute (= 20-25% of total cardiac output)Urine output:1 L/dFiltration:substances of up to 4 nm (excluding substances >8 nm), threshold at molecular weight of approximately 40,000 **Glomerular Filtration Rate** (GFR) [P] x GFR = [U] x U_{vol} **GFR = {[U] x U_{vol}} / [P]** = 125 mL/min = 20% of RPF *Substrate:* inulin; Tc-99m DTPA **Tubular Secretion** (Tm) [U] x U_{vol} = [P] x GFR + Tm **Tm = [U] x U_{vol}** - [P] x GFR *Substrate:* p-aminohippurate (PAH); I-131 Hippuran **Renal Plasma Flow** (RPF) [P] x RPF = [U] x U_{vol} **RPF = [U] x U_{vol}** / [P] *Substrate:* p-aminohippurate [P]= concentration in plasmaGFR= glomerular filtration rate[U]= concentration in urineU_{vol}= urine volumeTm= transport maximum (across tubular cells)RPF= renal plasma flow

Renal Acidification Mechanism Renal Imaging In Newborn Infant Contrast Excretion

Notes:

Renal Acidification Mechanism Proximal tubule: reabsorption of 90% of filtered bicarbonate by luminal Na^+/H^+ exchange and Na^+/HCO_3^- cotransport at basolateral membrane regulated by:luminal carbonic anhydraseinfluenced by:luminal HCO_3^- concentration, extracellular fluid volume, parathormone, K^+, aldosteroneDistal nephron: active secretion of H^+ against a steep urine-to-blood gradient across luminal cell membrane by H^+-ATPase pump facilitated by Na^+ reabsorption resulting in reabsorption of 10% of filtered bicarbonate, formation of ammonium (NH_4^+) and titratable acidity Ammonium excretion: Ammonia (NH_3) is formed in proximal tubule as a product of catabolism of glutamine + other amino acids; combination with secreted H^+ to NH_4^+ takes place in distal nephron Titratable acidity: divalent basic phosphate is converted into monovalent acid form in distal tubule



Notes:

Renal Imaging In Newborn Infant ◊low glomerular filtration rate (GFR):-on first day of life:21% of adult values-by 2 weeks of age:44% of adult values-at end of 1st year:close to adult values◊limited capacity to concentrate urineIVP: √ occasional failure of renal visualizationNUC: √ improved visualization on radionuclide studies

Notes:

Contrast Excretion UROGRAPHIC DENSITY depends on [U] = [P] x GFR / U_{vol} 1. Concentration of contrast material in plasma [P] is a function of (a)total iodine dose(b)contrast injection rate(c)volume distributionRapid decline of concentration of contrast material in vessels is due to: (1)rapid mixing within vascular compartment(2)diffusion into extravascular extracellular fluid space (capillary permeation)(3)renal excretion2. Glomerular filtration rate (GFR): 99% filtered 3. Urine volume (U_{vol}), ie, activity of ADH: (a)in dehydrated state with increased ADH activity concentrations of contrast material are higher◊Dehydration is considered a risk-potentiating factor for nephrotoxicity!(b)in volume-expanded state with decreased ADH activity concentrations of contrast material are lower◊Patients with CHF require higher doses of contrast material!A.MEGLUMINEno metabolization, excreted by glomerular filtration alone Meglumine effect of osmotic diuresis: (a) lower concentration of urinary iodine per mL urine (b) greater distension of collecting system N.B.:Avoid meglumine in "at risk" patients (higher incidence of contrast reactions than sodium!)B.SODIUMextensive reabsorption by tubules with delayed excretion *Sodium effect of reabsorption:* (a)increased concentration of urinary iodine (improved visualization)(b)less distension of collecting system (ureteral compression necessary)

Notes:

Numerary Renal Anomaly 1.Supernumerary kidney2.Complete / partial renal duplication3.Abortive calix4.Unicaliceal (unipapillary) kidney

Notes:

Renal Underdevelopment 1.Congenital renal hypoplasia2.Renal agenesis3.Renal dysgenesis

Notes:

Renal Ectopia Normal location of kidneys: 1st-3rd lumbar vertebra *Incidence:*0.2% (autopsy series) ***Longitudinal Renal Ectopia*** Location:pelvic, sacral, lower lumbar level, intrathoracic; L > R√ must demonstrate aberrant arteries*DDx:*displacement through diaphragmatic hernia (nonaberrant); hypermobile kidney **Pelvic kidney** =ectopic kidney due to failure of renal ascent*Incidence:*1:725 births*May be associated with:* (1)vesicoureteral reflux(2)hydronephrosis due to abnormally high insertion of ureter into renal pelvis(3)hypospadia (common)(4)contralateral renal agenesis√ blood supply via iliac vessels / aorta√ nonrotation = anteriorly positioned renal pelvis (common) ***Crossed Renal Ectopia*** =kidney located on opposite side of midline from its ureteral orifice; usually L > R and crossed kidney inferior to normal kidney*Cause:*? faulty development of ureteral bud, vascular obstruction of renal ascent*Associated with:*obstruction urolithiasis, infection, reflux, megaureter, hypospadia, cryptorchidism, urethral valves, multicystic dysplasia(a)fused (common)(b)separate (rare)√ invariably aberrant renal arteries√ distal ureter inserts into trigone on the side of origin ***Renal Fusion*** ="lump, cake, disk, horseshoe"*Cx:*aberrant arteries may cross and obstruct ureter **Discoid / pancake kidney** =bilateral fused pelvic kidneys*Associated with:* abnormal testicular descent, tetralogy of Fallot, vaginal agenesis, sacral agenesis, caudal regression, anal anomalies ***Renal Malrotation*** √ collecting structures may be positioned ventrally (most common), lateral (rare), dorsal (rarer), transverse (along AP axis)√ "funny-looking calices" = developmental usually nonobstructive ectasia

Notes:

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ADRENAL ANATOMY
from periphery to centrum: (a)renin-angiotensin-dependent outer adrenal cortex:zona glomerulosa=mineralocorticoid (aldosterone)(b)corticotropin-dependent inner adrenal cortex:zona fasciculata=cortisolzona reticularis=sex hormones (androgen, estrogen)(c)medulla=norepinephrine, epinephrine *mnemonic:*"Glomerular Filtration Rate May Give Answers"**G**lomerulosa **F**asciculata **R**eticulosa **M**ineralocorticoids **G**lucocorticoids **A**ndrogens Normal size:3-5 x 3 x 1 cmNormal weight:3-5 gVisualizationby CT:Left side 100%, Right side 99%by US:Left side 45%, Right side 80%

Notes:

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SCROTAL ANATOMY
Scrotal wall thickness: 2-8 mm (3-6 mm in 89%) Tunica vaginalis =inferior extension of processus vaginalis of the peritoneumHydrocele: small to moderate in 14% of normals

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Testis Epididymis Spermatic Cord

Notes:

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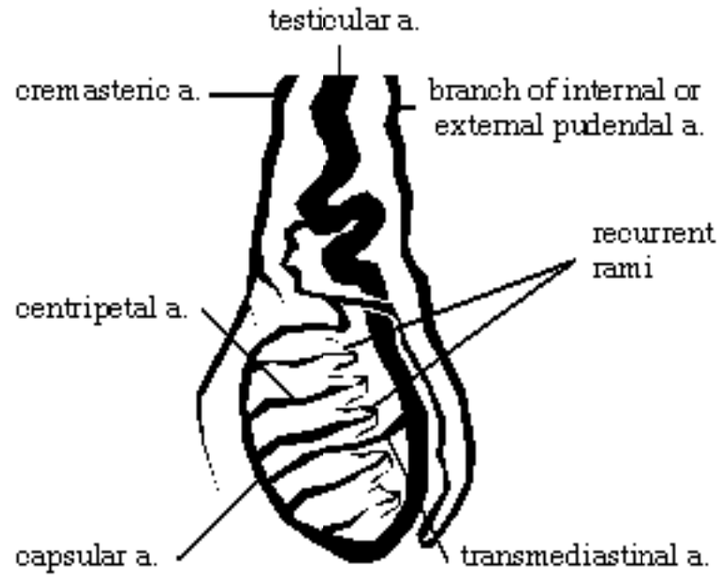
Testis Average size of testis: 3.8 x 3.0 x 2.5 cm (decreasing with age) Length of testis:3-5.5 cm (mature);1-1.5 cm (newborn)Testicular cysts:in 8% of normals (average size 2-3 mm), numbers increasing with age ***Appendix Testis*** =small stalked appendage at upper pole of testis= remnant of paramesonephric duct ***Tunica Albuginea*** =fibrous covering of testis, invaginating into testicular parenchyma at mediastinum testis; externally covered by visceral layer of tunica vaginalis; internally applied to tunica vasculosa carrying the capsular artery ***Mediastinum Testis*** =converging point of ~400 cone-shaped lobules separated by fibrous septa + seminiferous tubules forming tubuli recti and the rete testis within the mediastinum√ linear echogenic region extending longitudinally 5-8 mm from the edge ***Blood***



Arterial Supply of Scrotum

Flow To Testis PSV:4-10-19 cm/sEDV:2-5-8 cm/sRI:0.44-0.60-0.75

Notes:

Epididymis =tortuous tightly folded canal forming the efferent route from testis; consists of head (= globus major), body, tail (= globus minor)Size of globus major:11 x 7 x 6 mm (decreasing with age)Epididymal cysts:occur in 30% of normals (average size of 4 mm)Epididymal calcification:in 3%Appendix epididymis= occasionally duplicated, small stalked appendage of globus major

---

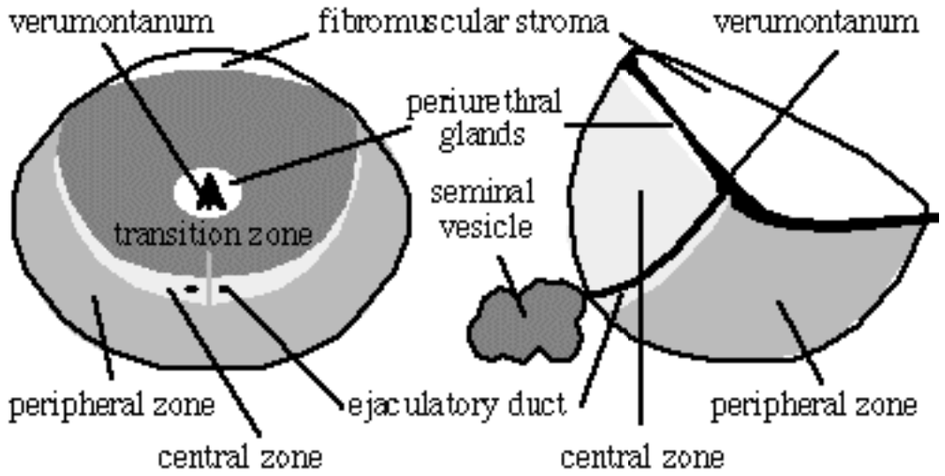
Notes:

Spermatic Cord =testicular + deferential + cremasteric aa., pampiniform plexus of veins, vas deferens, nerves, lymphatics

Notes:

ZONAL ANATOMY OF PROSTATE

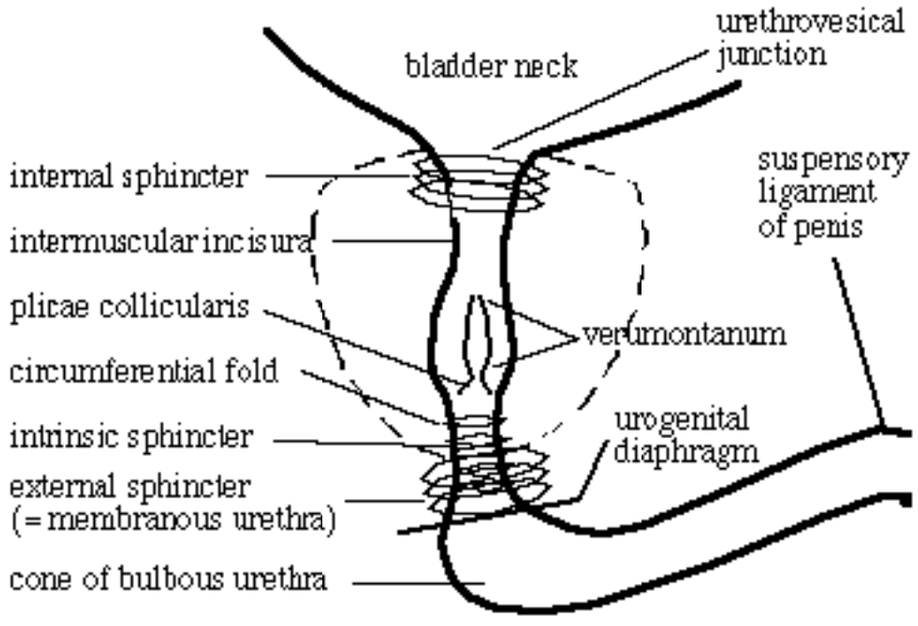
Normal weight:20 ± 6 gNormal size:2.8 cm (craniocaudad), 2.8 cm (anteroposterior), 4.8 cm (width)A.Outer gland1.Central zone:surrounds ejaculatory ducts from their entrance at prostatic base to verumontanum; 25% of glandular tissue2.Peripheral zone:extends from base of prostate to apex along rectal surface;70% of glandular tissueB.Inner gland1.Transition zone:on each side of internal sphincter; 4% of glandular tissue; enlarges with BPH2.Periurethral zone:surrounding urethra; 1% of glandular tissue



Transverse Section Through Prostate With BPH **Midsagittal Section Through Normal Prostate**

Notes:

Male Urethra extends through corpus spongiosum (composed of large venous sinuses) A.Posterior urethra1.Prostatic urethra = from vesical neck to triangular ligament-orifices of ducts from prostatic acini on floor-verumontanum = colliculus seminalis = prostatic utricle (fused end of müllerian ducts)-orifice of the two ejaculatory ducts2.Membranous urethra = portion traversing urogenital diaphragm-pea-sized bulbourethral glands of Cowper lie laterally + posteriorly between fasciae and sphincter urethrae within urogenital diaphragmB.Anterior = cavernous urethra1.Bulbous urethra2.Penile = pendulous urethra-many small branched tubular periurethral glands of Littré terminate in recesses (lacunae of Morgagni)*Cx:*recurring urethral discharge following chronic urethritis, latent gonorrheal urethritis, stricture formation3.Fossa navicularis



Urethrogram: normal urethral folds in LPO

Notes:

Female Urethra 3-5 cm in length, 6 mm in diameter urethral crest = posteriorly located prominent fold Two sets of glands: (a)urethral glands = terminate separately along entire length of urethra(b)paraurethral glands = glands of Skene (homologues of prostatic ducts) are formed by an interdependent conducting system and exit on either side of midline just posterior to urethral meatus draining into vaginal vestibule*Cx:*chronic gonorrheal urethritis1. Intrapelvic urethra =upper 2/3 of urethra that lies behind symphysis pubis2.Membranous urethrasurrounded by sphincter membranacea urethrae (weaker less important structure than in male) 3.Perineal urethralower 1/3 extending from superior fascia of urogenital diaphragm to meatus between labia minora

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Notes:

ABORTIVE CALYX
= developmental anomaly with short blind-ending outpouching of pyramid without papillary invaginationLocation:(a) renal pelvis(b) infundibulum (mostly upper pole)

Notes:

ACQUIRED CYSTIC KIDNEY DISEASE
=ACQUIRED CYSTIC DISEASE OF UREMIA=development of numerous fluid-filled renal cysts in patients with chronic renal failure undergoing hemodialysis Successful transplant probably stops development of additional cysts, but does not affect malignant potential!*Prevalence:*in 10-20% after 1-3 years, in 40-60% after 3-5 years, in 90% after 5-10 years of hemodialysis;in 25% of renal allograft recipients*Proposed etiologies:* (a)altered compliance of tubular basement membrane(b)intra- and extratubal obstruction due to focal proliferation of tubular epithelium(c)obstruction of ducts by interstitial fibrosis / oxalate crystals(d)toxicity from circulating metabolites (endogenous / exogenous toxins, mutagens, mitogens, growth factors)(e)vascular insufficiency*At increased risk:*older men*Histo:*cysts lined by flattened cuboidal / papillary epithelium*In 13-20% associated with:* (a)small papillary / tubular / solid clear-cell adenomas1 cm in diameter(b)renal cell carcinoma (in 3-6%): 7-year interval between transplantation + detection of RCC small end-stage kidneys (<280 g) multiple 0.5- to 3-cm cysts bilaterally (early = small,late = large) occasionally progressive renal enlargement due to cysts*Dx:*>3 cysts + NO history of hereditary cystic disease*Cx:*spontaneous hemorrhage into cyst (macrohematuria / retroperitoneal hemorrhage from cyst rupture)

Notes:

AIDS

● azotemia, proteinuria, hematuria, pyuria (in 38-68% sometime during illness) ● progressive renal failure (10%)1.**HIV nephropathy** (40%)=characterized by nephrotic-range proteinuria + rapidly progressive renal failure, primarily occurring in Black patients*Histo:*focal + segmental glomerulosclerosis, sparse interstitial infiltrates, severe tubular degenerative changes, interstitial tubular microcystic ectasia containing protein casts ● mild hypertension ● early + rapidly progressive renal failure with 100% mortality within 6 months√ global enlargement of both kidneysUS (best screening test): √ increased cortical echogenicity (33-68%)CT: √ medullary hyperattenuation (14%)√ striated nephrogram on CECTMRI: √ loss of corticomedullary differentiation*Prognosis:*death within 6 months2.Renal infection with Pneumocystis carinii (8%)√more frequent since introduction of prophylactic aerosolized pentamidine therapy encouraging extrapulmonic spread (<1%) due to inadequate systemic distribution of drug!√ punctate renal calcifications confined to cortex (DDx: CMV, Mycobacterium avium-intracellulare)√ associated calcifications in spleen, liver, lymph nodes, adrenal glands3.Renal lymphoma (3-12%)AIDS-related lymphoma: highly aggressive B-cell lymphomas (centroblastic, lymphoblastic, immunoblastic); NHL > Burkitt lymphoma, Hodgkin disease √ bilateral multiple renal masses√ direct extension of retroperitoneal lymphadenopathy engulfing kidney, renal sinus, ureter4.Cystitis (22%)*Organism:*routine Gram-negative species, Candida, beta-hemolytic streptococci, Salmonella, CMV√ bladder wall thickening

Notes:

ACUTE CORTICAL NECROSIS
=rare disorder with patchy / universal necrosis of renal cortex + proximal convoluted structures secondary to distension of glomerular capillaries with dehemoglobulinized RBCs; medulla and 1-2 mm of peripheral cortex are spared*Etiology:* (a)Obstetric patient (most often): abruptio placentae= premature separation of placenta with concealed hemorrhage (50%), septic abortion, placenta previa (b)Children: severe dehydration + fever, infection, hemolytic uremic syndrome, transfusion reaction(c)Adults: sepsis, dehydration, shock, myocardial failure, burns, snakebite, abdominal aortic surgery, hyperacute renal transplant rejection ● protracted + severe oliguria / anuria A.EARLY SIGNS√ diffusely enlarged smooth kidneys√ absent / faint nephrogramUS: √ loss of normal corticomedullary region with hypoechoic outer rim of cortexNUC: √ severely impaired renal perfusionB.LATE SIGNS√ small kidney (after a few months)√ "tramline" / punctate calcifications along margins of viable and necrotic tissue (as early as 6 days)US: √ hyperechoic cortex with acoustic shadowing*Prognosis:*poor chance of recovery

Notes:

ACUTE DIFFUSE BACTERIAL NEPHRITIS
=ACUTE SUPPURATIVE PYELONEPHRITIS=more severe and extensive form of acute pyelonephritis, which may lead to diffuse necrosis (phlegmon)*Organism:*Proteus, Klebsiella > E. coli*Predisposed:*diabetics (60%)

Notes:

ACUTE INTERSTITIAL NEPHRITIS
=infiltration of interstitium by lymphocytes, plasma cells, eosinophils, few PMNs + edema*Cause:*allergic / idiosyncratic reaction to drug exposure (methicillin, sulfonamides, ampicillin, cephalothin, penicillin, anticoagulants, phenindione, diphenylhydantoin) ● eosinophilia (develops 5 days to 5 weeks after exposure)√ large smooth kidneys with thick parenchyma√ normal / diminished contrast densityUS:√ normal / increased echogenicity

Notes:

ACUTE TUBULAR NECROSIS
=temporary reversible marked reduction in tubular flow rate*Etiology:* (a)DRUGS: bichloride of mercury, ethylene glycol (antifreeze), carbon tetrachloride, bismuth, arsenic, uranium, urographic contrast material (especially when associated with glomerulosclerosis in diabetes mellitus), aminoglycosides (gentamicin, kanamycin)(b)ISCHEMIA: major trauma, massive hemorrhage, postpartum hemorrhage, crush injury, myoglobulinuria, compartmental syndrome, septic shock, cardiogenic shock, burns, transfusion reaction, severe dehydration, pancreatitis, gastroenteritis, renal transplantation, cardiac surgery, biliary surgery, aortic resection*Pathophysiology:*profound reduction in renal blood flow due to elevated arteriolar resistance√ smooth large kidneys, especially increase in AP diameter >4.63 cm (due to interstitial edema)√ diminished / absent opacification of collecting system√ immediate persistent dense nephrogram (75%)√ increasingly dense persistent nephrogram (25%)√ diffuse calcifications (rare)US: √ normal to diminished echogenicity of medulla√ sharp delineation of swollen pyramids√ normal (89%) / increased (11%) echogenicity of cortex√ elevated resistive index ≥0.75 (in 91% excluding patients with hepatorenal syndrome); unusual in prerenal azotemiaAngio: √ normal arterial tree with delayed emptying of intrarenal vessels√ slightly delayed / normal venous opacificationNUC: √ poor concentration of Tc-99m glucoheptonate / Tc-99m DTPA√ well-maintained renal perfusion√ better renal visualization on immediate postinjection images than on delayed images√ progressive parenchymal accumulation of I-131 Hippuran / Tc-99m MAG3√ no excretion

Notes:

ADDISON DISEASE
=PRIMARY ADRENAL INSUFFICIENCY90% of adrenal cortex must be destroyed!*Course:*acute (adrenal apoplexy), subacute (disease present for <2 years), chronicCause: 1.Idiopathic adrenal atrophy (60-70%): likely autoimmune disorder2.Granulomatous disease: tuberculosis, sarcoidosis3.Fungal infection: histoplasmosis, blastomycosis, coccidioidomycosis4.Adrenal hemorrhage: anticoagulation therapy, trauma, bleeding, coagulation disorders, sepsis, shock5.Bilateral metastatic disease (rare)√ diminutive glands (in idiopathic atrophy + chronic inflammation)√ enlarged glands (acute inflammation, acute hemorrhage, metastasis)√ calcifications (in 25% of chronic course)

Notes:

ADRENAL CYST

*Prevalence:*0.064-0.180%*Path:*(a)endothelial lining (45-48%):1.Lymphangioma (93%)2.Hemangioma(b)epithelial lining = true cyst (9-10%):1.Glandular / retention cyst2.Embryonal cyst3.Cystic adenoma4.Mesothelial inclusion cyst(c)pseudocyst (39-42%):1.Previous hemorrhage / infarction2.Hemorrhagic complication of benign vascular neoplasm / malformation3.Cystic degeneration / hemorrhage of primary adrenal mass(d)parasitic cyst (7%): usually echinococcal*Age:*3rd-6th decades (most commonly); M:F = 1:3Location:mostly solitary; R:L = 1:1; bilateral in 8-10%√ well-defined uni- / multilocular√ wall thickness of up to 3 mm√ <5 cm in diameter in 50% (up to 20 cm)√ usually homogeneous with near-water density; higher attenuation with hemorrhage / intracystic debris / crystals√ lack of central enhancement ± wall enhancement√ calcifications:(a)peripheral / mural: rimlike / nodular (51-69%)(b)central: in intracystic septation (19%) / punctate within intracystic hemorrhage (5%)*Cx:*hypertension; hemorrhage; infection; rupture with retroperitoneal hemorrhage*DDx:*1.Cystic pheochromocytoma2.Cystic adenomatoid tumor3.Schwannoma4.Cystic adrenocortical carcinoma (thick-walled lesion >7 cm in size; extremely rare)5.Adrenal adenoma (contrast enhancement, no wall, no peripheral calcification)

Notes:

ADRENAL HEMORRHAGE

Cause: A.NEWBORN 1.Birth trauma: forceps / breech delivery2.Hypoxia due to prematurity3.Infants of diabetic mothers4.Septicemia5.Hemorrhagic disorders*Age:*1st week of lifeSite:R > L; bilateral in 10%B.ADULT1.Anticoagulant therapy: during initial 3 weeks2.Stress caused by sepsis: Waterhouse-Friderichsen syndrome3.Surgery: orthotopic liver transplantation4.Adrenal venous sampling5.Tumor6.Blunt abdominal trauma*Prevalence:*2% (in 28% of autopsies)Location:R:L = 9:1, bilateral in 20%√ round / oval hematoma (in 83%) located in medulla + stretching cortex around hematoma√ obliteration of gland by diffuse irregular hemorrhage (in 9%)√ uniform adrenal enlargement (in 9%)√ periadrenal hemorrhage causes ill-defined adrenal margin + stranding + asymmetric thickening of diaphragmatic crus√ mass displacing renal axis√ gradual decrease in size√ peripheral calcification occurring after 1 weekUS: √ initially echogenic becoming progressively hypoechoic (degeneration, lysis)CT: √ high-attenuation mass (50-90 HU) in acute / subacute stage

Notes:

ADRENOCORTICAL ADENOMA

A.NONHYPERFUNCTIONINGcharacterized by (a)normal lab values of adrenal hormones(b)NO pituitary shutdown of the contralateral gland(c)activity on NP-59 radionuclide scans*Incidence:*incidental finding in 0.6 -1.5% of CT examinations, in 3-9% at autopsy√ surveillance CT to confirm lack of growth*Rx:*surgical removal for masses 3-5 cm as indeterminate potentially malignant neoplasms*DDx:*metastasis B. HYPERFUNCTIONING 1.Primary hyperaldosteronism (= Conn syndrome)*Pathophysiology:*secretion of aldosterone by an adenoma is pulsatile√ ACTH infusion incites a dramatic increase in levels of cortisol + aldosterone for venous sampling2.Cushing syndrome (10%)3.Virilization:(a) hirsutism + clitoromegaly in girls (b) pseudopuberty in boys most common type of hormone elevation in children ● elevated testosterone levels >0.55 ng/mL4.Feminization (estrogen production)√ contralateral atrophic gland (secondary to ACTH suppression with autonomous adenoma)√ unilateral focus of I-131 NP-59 radioactivity + contralateral absence of iodocholesterol accumulation (DDx: hyperplasia [bilateral activity]) √ well-defined sharply marginated mass <5 cm in size (average size 2.0-2.5 cm)√ mild homogeneous enhancement√ adenoma may calcify CT: √ soft-tissue density / cystic density (mimicked by high cholesterol content) with poor correlation between functional status and HU number√ <10 HU on NECT is 73% sensitive + 96% specific for adenoma√ <37 HU on delayed CECT (>5-15 minutes after contrast injection) is DIAGNOSTIC of adenoma√ small adenomas <1 cm often go undetected√ contralateral gland often normal / atrophicAngio: √ tumor blush + neovascularity; occasionally hypovascular√ pooling of contrast material√ enlarged central vein with high flow√ arcuate displacement of intraadrenal veins√ bilateral adrenal venous sampling in up to 40% unsuccessful in localizingMR: √ mass iso- / hypointense (rarely hyperintense) to liver on T2WI√ mild enhancement + quick washout on Gd-dimeglumine enhanced study(DDx: metastases tend to have higher signal intensities [however 20-30% overlap])

Notes:

ADRENOCORTICAL CARCINOMA
*Prevalence:*0.3-0.4% of all pediatric neoplasms (3 times as likely than adrenal adenoma)*May be associated with:*hemihypertrophy, Beckwith-Wiedemann syndrome, astrocytomas ● 20% nonfunctioning ● 50% hyperfunctioning (in 10-15% Cushing syndrome)Size:usually >5 cm (median size 12 cm; in 16% <6 cm)√ frequently heterogeneous mass with irregular margins√ occasionally calcified√ invasion of IVC√ metastases to regional lymph nodes, kidney, renal veins, liver, diaphragm, lung, bone, brain◊Metastases are the only reliable sign of malignancy!◊Large size + calcifications suggest malignancy!CT: √ central areas of low attenuation (tumor necrosis) √ heterogeneous enhancement (foci of hemorrhage + central necrosis)US: √ complex echo pattern (due to hemorrhage + necrosis)MRI: √ hyperintense to liver on T2WIAngio: √ enlarged adrenal arteries√ neovascularity, occasionally with parasitization√ AV shunting; multiple draining veinsNUC: √ usually bilateral nonvisualization with I-131 NP-59 (carcinomatous side does not visualize because amount of uptake is small for size of lesion; contralateral side does not visualize because carcinoma is releasing sufficient hormone to cause pituitary feedback shutdown of contralateral gland)Biopsy:may appear histologically benign in well-differentiated adenocarcinoma*Prognosis:*0% 5-year survival rate*DDx:*metastasis (similar signal intensities on MR)

Notes:

ADRENOCORTICAL HYPERPLASIA

◊Responsible for 8% of Cushing syndrome and 10-20% of hyperaldosteronism!*Cause:* 1.Corticotropin-dependent (85%): pituitary causes, ectopic corticotropin production, production of corticotropin-releasing factor2.Primary pigmented nodular adrenocortical hyperplasia*Associated with:*Carney complex (spotty skin pigmentation, calcified Sertoli cell tumors of testes, cardiac and soft-tissue myxomas) 3.Primary aldosteronism (rare)*Incidence:*4 x increased in patients with malignancy*Age:*70-80% in adults; 19% in children*Types:* (1)Smooth hyperplasia (common)√ bilateral normal-sized glands√ thickened + elongated glands(2)Cortical nodular hyperplasia (less common)√ normal glands ± appreciable micronodular configuration√ thickened gland with macronodular configuration (nodules up to 2.5 cm)Angio: √ minimally increased hypervascularity√ focal accumulation of contrast medium√ normal venogram / may show enlarged glandNUC: √ asymmetric bilateral NP-59 uptake (related to urinary cortisol excretion) without dexamethasone suppression in Cushing syndrome√ bilateral foci of NP-59 uptake with dexamethasone suppression (nondiagnostic ≥5 days)

Notes:

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ADRENOGENITAL SYNDROMES

A.CONGENITAL TYPE=impaired cortisol + aldosterone synthesis secondary to enzyme defect (21-hydroxylase) with increased ACTH stimulation by pituitary gland (negative feedback mechanism)M < F ● excess of androgenic steroids ● ± salt wasting due to diminished mineralocorticoids ● virilization of female fetus ● precocious puberty in male ● pseudohermaphroditism (clitoral hypertrophy, ambiguous external genitalia, urogenital sinus)♀ symmetrically enlarged + thickened adrenal glands*Rx:*cortisone ± mineralocorticoidsB.ACQUIRED TYPEM < F (a)adrenal hyperplasia / adenoma / carcinoma(b)ovarian / testicular tumor(c)gonadotropin-producing tumor: pineal, hypothalamic, choriocarcinoma ● virilization ● Cushing syndrome

Notes:

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AMYLOIDOSIS

=accumulation of extracellular eosinophilic protein substances@Renal involvement*Incidence:*1° amyloidosis (35%), 2° amyloidosis (in >80%)√ smooth normal to large kidneys with increase in parenchymal thickness (early stage)√ small kidneys = renal atrophy (late stage)√ occasionally attenuated collecting system√ increase in cortical echogenicity (deposition of amyloid in glomeruli and interstitium) + prominence of corticomedullary junction + obscuration of arcuate aa.√ nephrographic density normal to diminishedUS: √ normal to increased echogenicity*Cx:*renal vein thrombosis

Notes:

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ANALGESIC NEPHROPATHY
=renal damage from ingestion of salicylates incombination with phenacetin / acetaminophen in a cumulative dose of 1 kg*Incidence:*United States (2-10%), Australia (20%)*Age:*middle-aged; M:F = 1:4 ● gross hematuria ● hypertension ● renal colic (passage of renal tissue) ● renal insufficiency (2-10% of all end-stage renal failures) ● **Analgesic syndrome**: history of psychiatric therapy, abuse of alcohol + laxatives, headaches, pain in cervical + lumbar spine, peptic ulcer, anemia, splenomegaly, arteriosclerosis, premature aging√ papillary necrosis√ scarring of renal parenchyma ("wavy outline"); bilateral in 66%, unilateral in 5%√ renal atrophy√ papillary urothelial tumors in calices / pelvis (mostly TCC / squamous cell carcinoma), in 5% bilateral

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Notes:

ANGIOMYOLIPOMA
=benign mesenchymal tumor of kidney=RENAL CHORISTOMA (= benign tumor composed of tissues not normally occurring within the organ of origin)=RENAL HAMARTOMA (improper name since fat and smooth muscle do not normally occur within renal parenchyma)*Prevalence:*0.3-3%*Path:*no true capsule, 88% extending through renal capsule, hemorrhage (characteristic lack of complete elastic layer of vessels predisposes to aneurysm formation); tumor continues to grow during childhood + early adulthood*Histo:*tumor composed of fat, smooth muscle, aggregates of thick-walled blood vessels*Types:* (1)Isolated AML (80%) = sporadic AMLsolitary + unilateral (in 80% on R side), NO stigmata of tuberous sclerosis *Age:*27-72 (mean 43) years of age; M:F = 1:4(2)AML associated with tuberous sclerosis (in 20%)◊AML in 80% of patients with tuberous sclerosiscommonly large + bilateral + multiple;◊may be the only evidence of tuberous sclerosis*Mean age:*17 years; M:F = 1:1 ● small lesions are asymptomatic (60%) ● acute flank / abdominal pain (due to hemorrhage) in 87% ● shock (due to massive retroperitoneal hemorrhage) ● hematuria (40%) ● palpable mass (47%)√ mostly <5 cm in diameter√ large component of exophytic extrarenal tumor (25%)√ calcifications (6%)Plain film: √ mass of fat lucency (in <10%)CT: √ well-marginated cortical heterogeneous tumor predominantly of fat density <-20 HU√ homogeneously high attenuation on NECT in 5% (due to minimal fat component)√ variable enhancement (smooth muscle, vessels)US: √ intensely echogenic tumor (due to high fat content)√ homogeneously isoechoic in 5% (due to minimal fat component)√ less echogenic areas due to hemorrhage, necrosis, dilated calycesMRI: √ intratumoral fat (fat-suppression technique)√ variable areas of high signal intensity on T1WI(DDx: hemorrhagic cyst, solid tumor)Angio: √ hypervascular mass (95%) with enlarged interlobar + interlobular feeding arteries, tortuous irregular aneurysmally dilated vessels (1/3), venous pooling, "sunburst" / "whorled" / "onion peel" appearance, no AV shunting*Cx:*hemorrhagic shock from bleeding into angiomyolipoma or into retroperitoneum◊Angiomyolipomas >4 cm bleed spontaneously in 50-60%!*Rx:*(1)annual follow-up of lesions <4 cm(2)emergency laparotomy (in 25%): nephrectomy, tumor resection(3)selective arterial embolization*DDx:*renal / perirenal lipoma or liposarcoma; Wilms tumor / renal cell carcinoma (occasionally contains fat)

Notes:

ARTERIOVENOUS MALFORMATION

(1)Congenital AVM(2)Acquired AVM: trauma, spontaneous rupture of aneurysm, very vascular malignant neoplasm*Histo:* (a)cirsoid = multiple coiled vascular channels grouped in cluster; supplied by one / more arteries; draining into one / more veins(b)cavernous = single well-defined artery feeding into a single vein (rare)√ large unifocal mass√ focally attenuated and displaced collecting system√ homogeneously enhancing mass √ curvilinear calcificationUS: √ tubular anechoic structure (DDx: hydronephrosis, hydrocalyx)

Notes:

BENIGN PROSTATIC HYPERTROPHY

=BENIGN PROSTATIC HYPERPLASIA*Prevalence:*50% between ages 51 + 60 years; 75-80% of all men >80 years of age*Histo:*fibromyoadenomatous nodule (most common), muscular + fibromuscular + fibroadenomatous + stromal nodules*Age:*initial growth onset <30 years of age; onset of clinical symptoms at 60 ± 9 years ● sensation of full bladder, nocturia ● trouble initiating micturition ● decreased urine caliber + force ● dribbling at termination of micturitionLocation:transition + periurethral zone proximal to verumontanum forming "lateral lobes" (82%), "median lobe" (12%)√ oval (61%) / round (22%) / pear-shaped (17%) enlargement of central gland√ posterior + lateral displacement of outer gland(= prostate proper) creating cleavage plane of fibrous tissue between hyperplastic tissue + compressed prostatic tissue (= surgical capsule) often demarcated by displaced intraductal calcifications*Cx:*bladder outflow obstruction*Rx:* (1)Surgery: open prostatectomy (glands >80 g), transurethral resection of prostate = TURP (glands <80 g)√Only 4-5% of patients need surgical treatment!(2)Drugs: a-blockers (for stromal hyperplasia); androgen deprivation (suppression of LHRH / inhibition of Leydig cell synthesis of testosterone / competition for androgen receptor binding sites) + a-blockers (for glandular hyperplasia)

Notes:

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BLADDER CALCULI
Etiology: 1.FOREIGN BODY NIDUS CALCULIfrom self-introduced objects, bladder wall-penetrating bone fragments, prostatic chips, nonabsorbable suture material, fragments of Foley balloon catheter, pubic hair, presence of intestinal mucosa (in bladder augmentation, ileal conduit, repaired bladder exstrophy) 2.STASIS CALCULIin bladder outflow obstruction, vesical diverticula, lower urinary tract infection (in particular Proteus), cystocele, neuropathic bladder dysfunction 3.MIGRANT CALCULI= renal calculi spontaneously passing into bladder 4.IDIOPATHIC / PRIMARY / ENDEMIC CALCULIin North Africa, India, Indonesia; in young boys of low socioeconomic class (nutritional deficiency?) ↓ single stone in 86%Rate of recurrence after removal:41%

Notes:

BLADDER CONTUSION

= intramural hematoma (most common bladder injury) √ no extravasation√ lack of normal distensibility√ crescent-shaped filling defect in contrast-distended bladder

Notes:

BLADDER DIVERTICULUM
=cavity formed by herniation of bladder mucosa through muscular wall, joined to the bladder cavity by aconstricted neck*Prevalence:*1.7% in children*Etiology:* A.PRIMARY / CONGENITAL / IDIOPATHIC DIVERTIVULA (40%)√ in 3% single diverticulum(a)with vesicoureteral reflux1.Hutch diverticulum in paraureteral region(b)without vesicoureteral refluxB.SECONDARY DIVERTICULA (60%)√ in 50% multiple diverticula(a)postoperative state(b)associated with bladder outlet obstruction1.Posterior urethral valves2.Urethral stricture3.Large ureterocele4.Neurogenic dysfunction5.Enlarged prostate6.Bladder neck stenosis(c)associated with syndromes1.Prune belly syndrome2.Menkes kinky-hair syndrome3.Williams syndrome4.Ehlers-Danlos type 9 syndrome5.Diamond-Blackfan syndromeC.MULTIPLE DIVERTICULA IN CHILDREN1.Neurogenic dysfunction2.Posterior urethral valves3.Prune belly syndrome *Average age:*57 years; M:F = 9:1Site:areas of congenital weakness of muscular wall at(a)ureteral meatus(b)posterolateral wall (Hutch diverticulum= paraureteral) *Cx:*(1)Vesical carcinoma in 0.8-7% secondary to chronic inflammation (average age 66 years)(2)Ureteral obstruction(3)Ureteral reflux

Notes:

BLADDER EXSTROPHY

=EPISPADIA-EXSTROPHY COMPLEX*Prevalence:*1:33,000 to 1:40,000 live births*Etiology:*incomplete retraction of cloacal membrane prevents normal midline migration of mesoderm resulting in incomplete midline closure of infraumbilical abdominal wall; size of persistent cloacal membrane at time of rupture accounts for different degrees of severity ● urinary bladder exposed + open anteriorly ● mucosa everted through abdominal wall defect ● bladder margins continuous with margins of abdominal wall ● epispadia (male); bifid clitoris (female) *May be associated with:* wide linea alba, omphalocele, limb defects (eg, club feet), renal malformation (horseshoe kidney, renal agenesis), incomplete testicular descent, GI obstruction, bilateral inguinal hernias, imperforate anus, cardiac anomalies, hydrocephalus, meningomyelocele √ ventral defect of infraumbilical abdominal wall√ low position of umbilicus√ pubic diastasis = widening of pubic symphysis *Cx:*urinary incontinence, infertility, pyelonephritis, bladder carcinoma (4%)*Rx:*primary closure, bladder excision with urinary diversionClosed Exstrophy = Pseudoexstrophy =persistent large cloacal membrane without rupture ● anterior wall of bladder covered by thin bilaminar epithelial membrane√ infraumbilical musculoskeletal defect√ subcutaneous position of bladder

Notes:

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BLADDER RUPTURE
Cystography: diagnostic in >85% false-negatives if tear sealed by hematoma / mesentery

Extraperitoneal Rupture Of Bladder (80%) Intraperitoneal Rupture Of Bladder (20%)

Notes:

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Extraperitoneal Rupture Of Bladder (80%)
*Cause:*pelvic fracture (sharp bony spicule) or avulsion tear at fixation points of puboprostatic ligamentsLocation:usually close to base of bladder anterolaterallyPlain film: √ "pear-shaped" bladder√ loss of obturator fat planes√ paralytic ileus√ upward displacement of ileal loopsContrast examination: √ flame-shaped contrast extravasation into perivesical fat, best seen on postvoid films, may extend into thigh / anterior abdominal wallUS: √ "bladder within a bladder" = bladder surrounded by fluid collection

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Notes:

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Intraperitoneal Rupture Of Bladder (20%)
Cause: (a)usually as a result of invasive procedure (cystoscopy), stab wound, surgery(b)blunt trauma with sudden rise in intravesical pressure (requires distended bladder)Location:usually at dome of bladder√ contrast extravasation into paracolic gutters√ contrast outlining small bowel loops√ uriniferous ascites

Notes:

CHOLESTEATOMA

=keratin ball = keratinized squamous epithelium shed into lumen ● history of UTIs ● repeated episodes of renal colicLocation:renal pelvis > upper ureter√ mottled / stringy filling defects in collecting system√ dilatation of pelvicaliceal system (with obstruction)√ calcification of keratinized material possible◊Not a premalignant condition!

Notes:

CHROMOPHOBE CARCINOMA OF KIDNEY

*Prevalence:*4% of renal cell neoplasms*Age:*median in 6th decade (31-75 years)*Histo:*cells with abundant cytoplasm containing numerous microvesicles; average size of 8 cm (range 1.3-20 cm)*Prognosis:*probably better than RCC

Notes:

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CHRONIC GLOMERULONEPHRITIS

*Cause:*after acute poststreptococcal glomerulonephritis ● late presentation without prior clinically apparent acute phase ● hypertension ● renal failure√ small smooth kidneys with wasted parenchyma√ normal papillae + calices√ patchy nephrogram with diminished density of contrast material√ cortical calcification (uncommon)US: √ increased echogenicity√ small kidneys with vicarious sinus lipomatosisAngio: √ marked reduction in renal blood flow + reflux of contrast material into aorta√ severely pruned + tortuous interlobar and arcuate arteries√ nonvisualization of interlobular arteries√ delayed contrast clearance from interlobar arteries

Notes:

CLEAR CELL SARCOMA OF KIDNEY
=rare highly malignant renal tumor of childhood with predilection for bone metastasis*Incidence:*up to 6% of renal tumors in children*Histo:*composed of well-defined polygonal to stellate cells with vacuolization, ovoid to rounded nuclei, prominent capillary pattern + tendency toward cyst formation separated by slightly thickened septa*Age:*1-6 years; M:F = 1:1 ● increasing abdominal girth + palpable abdominal mass ● lethargy, weight loss ● hematuria√ expansile mass (8-16 cm) with dominant soft-tissue component√ cystic component of varying size (few mm to 5 cm) + multiplicity (58%)√ amorphous / linear calcifications (25%)√ renal mass crossing midline (58%)US: √ inhomogeneous renal mass of soft-tissue density√ well-defined hypoechoic central area (= necrosis)√ mass of fluid-filled cystic spacesCT: √ inhomogeneous enhancement less than that of normal renal parenchyma√ low-attenuation areas (= necrosis)√ water-density areas (= cysts)*Prognosis:*worse than Wilms tumor*DDx:*cystic form of Wilms tumor, multilocular cystic nephroma, cystic dysplasia

Notes:

CONGENITAL RENAL HYPOPLASIA
=miniaturization with reduction in number of renal lobes, number of calices and papillae, amount of nephrons(+ smallness of cells)VARIANT:**Ask-Upmark kidney** = aglomerular focal hypoplasia√ unilateral small kidney√ decreased number of papillae + calices (5 or less)√ hypertrophied contralateral kidney√ absent renal artery√ hypoplastic disorganized renal veins

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Notes:

CONN SYNDROME

=PRIMARY HYPERALDOSTERONISM = PRIMARY ALDOSTERONISM = autonomous excess secretion of the mineralocorticoid aldosterone with hypertension + spontaneous hypokalemia=solitary adrenocortical adenoma (originally)*Incidence:*0.05-2% of hypertensive population*Age:*3rd-5th decade; M:F = 1:2 ● hypertension (secondary to hypernatremia) ● hypokalemia (80-90%, induced by administering large amounts of sodium chloride for 3-5 days): ● muscle weakness, cardiac arrhythmia ● carbohydrate intolerance ● nephrogenic diabetes insipidus ● depletion of magnesium ● metabolic alkalosis ● increased urinary excretion of aldosterone + metabolites ● nonsuppressible elevation in plasma aldosterone concentration ● suppressed plasma renin levels*Path:* (a)adenoma (65-89%): solitary aldosteronoma (65-70%); multiple (13%); microadenomatosis (6%)(b)bilateral adrenal hyperplasia (11-25-30%):= idiopathic hyperaldosteronism = focal / diffuse hyperplasia of glomerular zone accompanied by micro- / macroscopic nodules (c)adrenocortical carcinoma (<1%)√ small aldosteronoma of 1.7 cm average size (range0.5-3.5 cm); L > R, bilateral in 6%√ soft-tissue density / low attenuation◊Among hyperfunctioning adrenal adenomas aldosteronomas have the lowest attenuation!√ usually hypervascular, rarely hypovascular√ normal / nodular / multinodular adrenal gland(s) (with hyperplasia)Adrenal venography:76% accuracyAdrenal venous blood sampling:95% accuracy, 75% sensitivityCT :60-80% sensitivity NUC: √ I-131 NP-59 uptake following dexamethasone suppression√ bilateral early visualization (<5 days) implies adrenal hyperplasia√ unilateral early visualization implies adenoma√ late bilateral visualization (>5 days) may be normal*Dx:*elevated plasma aldosterone concentration + suppressed plasma renin activityDiagnostic endocrine tests: postural stimulation test, short saline infusion test, 18-hydroxycorticosterone concentration *Rx:*adrenalectomy for neoplasms (75% long-term cure rate for hypertension); medical treatment for hyperplasia

Notes:

CONTRAST NEPHROPATHY

=CONTRAST-INDUCED RENAL FAILURE=increase in serum creatinine of ≥1 mg/dL ± 25-50% of the baseline creatinine level after intravascular contrast administration*Patients at risk:* 1.Preexisting renal insufficiency2.Insulin-dependent diabetes mellitus3.Large volume of contrast media4.Concomitant administration of other nephrotoxic drugs: aminoglycosides, nonsteroidal anti-inflammatory agents5.American Heart Association class IV congestive heart failure6.Hyperuricemia◊A serum creatinine level of >4.5 mg/dL causes acute renal failure in 60% of nondiabetics + 100% of diabetics!*Previously considered but no longer accepted risk factors:* dehydration, hypertension, proteinuria, peripheral vascular disease, age >65 years, multiple myeloma *Mechanism:* increase in renal perfusion by vasodilatation (via prostaglandin I2 ± E2) followed by vasoconstriction (via angiotensin II, norepinephrine, vasopressin) *Time course:* (a)rise in serum creatinine within 1-2 days(b)peak at 4-7 days(c)return to normal by 10-14 days√ persistent nephrogram on plain film√ cortical attenuation >140 HU on CT with 24-hour delay*Recommendation:* ◊Employ nonionic contrast media (LOCM appears safe in patients without renal dysfunction / underlying risk factors in doses as large as 800 mL [300 mg iodine per mL])◊Do not exceed maximum allowed dose (Cigarroa formula for HOCM): Contrast limit (mL) 60% by weight = (5 mL x body weight (kg)) / (serum creatinine (mg/100 mL))

Notes:

CUSHING SYNDROME
=HYPERCORTISOLISM = excessive glucocorticoid secretion from either exogenous / endogenous sources*Etiology:* A.ACTH-INDEPENDENT1.Exogenous cortisol2.Primary adrenal abnormality (20%):(a)primary pigmented nodular adrenocortical hyperplasia (children, young adults)(b)adrenocortical adenoma (10-20% of cases; 10% in adults, 15% in children)(c)adrenocortical carcinoma (5-10% of cases; 10% in adults, 66% in children)B.ACTH-DEPENDENT=overproduction of corticotropin with adrenal hyperplasia (in up to 85%)1.Exogenous ACTH2.Paraneoplastic ectopic ACTH production (20%): oat cell carcinoma of lung (8%), liver cancer, prostate cancer, ovarian cancer, breast cancer, bronchial / thymic carcinoid, bronchial adenoma, pancreatic islet cell tumor (10%), medullary carcinoma of thyroid, thymoma, pheochromocytoma◊Bronchial + thymic carcinoids are often <1 cm at the time they produce Cushing syndrome!◊Islet cell tumors are large + often metastatic by the time they produce Cushing syndrome!3.**Cushing disease** (70% of endogenous causes)=adrenal hyperplasia due to overproduction of ACTH*Cause:*(1)basophilic / chromophobe adenoma(2)overactive pituitary(3)ACTH-producing primary elsewhere4.Hypothalamic dysfunction5.Production of corticotropin-releasing factor (rare)*Incidence:*1:1,000 autopsies; M:F = 1:4*Age:* 30-40 years (highest incidence); more often following pregnancy ● central / truncal obesity, buffalo hump, moon face, facial plethora ● purple striae, acne, hirsutism ● fatigue, weakness, amenorrhea ● impaired glucose tolerance / diabetes mellitus ● hypertension, atherosclerosis, edema ● elevated plasma cortisol levels ● excessive excretion of urinary 17-hydroxy-corticosteroids ● dexamethasone suppression test / metyrapone test√ retarded bone maturation√ most often axial osteoporosis√ stippled calvarium√ demineralized dorsum sellae√ excess callus formation*Cx:*(1)pathologic fractures of vertebrae + ribs with excessive callus formation(2)aseptic necrosis of hips(3)bone infarcts(4)delayed skeletal maturation in children

Notes:

CYSTITIS

=bacterial infection; more common in females ● frequency, dysuria, hematuria ● reduced bladder capacity√ cystogram insensitiveUS: √ focal / multifocal / circumferential isoechoic bladder wall thickening√ decrease in bladder wall thickening during bladder distension (eg, instillation of sterile saline via a urethral catheter)√ bullous lesions√ intact mucosa

Cystitis Cystica Emphysematous Cystitis Granulomatous Cystitis = Tuberculous Cystitis Hemorrhagic Cystitis Interstitial Cystitis Bullous Edema Of Bladder Wall

Notes:

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Cystitis Cystica = CYSTITIS FOLLICULARIS = CYSTITIS GLANDULARIS = BULLOUS CYSTITIS =nonspecific inflammatory process of bladder wall√ multiple small round cystlike mucosal elevations*Prognosis:*potentially malignant in adults

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Notes:

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Emphysematous Cystitis =uncommon complication of urinary tract infection by gasforming organism almost PATHOGNOMONIC of poorly controlled diabetes (= bacterial fermentation of glucose)*Age:*>50 years; M:F = 1:2*Predisposed:*diabetes mellitus, neurogenic bladder, bladder outlet obstruction, chronic UTI*Organism:*E. coli, E. aerogenes, P. mirabilis, S. aureus, streptococci, Clostridium perfringens, Nocardia, Candida*May be associated with:*emphysematous pyelitis / pyelonephritis ● pneumaturia (rare)Plain film: √ translucent streaky irregular area / ring of air bubbles in bladder wall√ intraluminal air-fluid levelUS: √ shadowing echogenic foci within area of bladder wall thickeningCT (most specific modality) *DDx:*(a)Gas within bladder:trauma, urinary tract instrumentation, enterovesical fistula (b)Gas extern to bladder:rectal gas, emphysematous vaginitis, pneumatosis cystoides intestinalis, gas gangrene of uterus

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Notes:

Granulomatous Cystitis = Tuberculous Cystitis

√ irritable hypertonic bladder with decreased capacity√ disease process usually starts at trigone spreading upward and laterally√ calcification of bladder wall (rare)

Notes:

Hemorrhagic Cystitis *Cause:*unclear(a)nonspecific: negative culture(b)bacterial: E. coli (in 17%)(c)viral (adenovirus in 19%): negative culture, viral exanthem(d)cytotoxic: cyclophosphamide (Cytoxan®), in 15% of patients within 1st year of treatment echogenic mobile clumps of solid material(= intraluminal blood clots)

Notes:

Interstitial Cystitis *Age:*postmenopausal female ● pink pseudoulceration of bladder mucosa characteristically at vertex of bladder (= Hunner ulcer)

Notes:

Bullous Edema Of Bladder Wall *Cause:*continuous internal contact with Foley catheter, involvement of bladder wall by external contact in pelvic inflammatory conditions (eg, Crohn disease, appendicitis, diverticulitis)√ smoothly thickened / polypoid redundant hypoechoic mucosa *DDx:*bladder neoplasm, ureterocele, pseudoureterocele, neurofibromatosis, pseudosarcomatous myofibroblastic proliferations

Notes:

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DIABETES MELLITUS
=multisystem disorder*Prevalence:*14 million patients in United States*Path:*macro- and microvascular disease; neuropathy increased susceptibility to infectionA.CHRONIC EFFECTS1.Papillary necrosis2.Renal artery stenosis3.Vas deferens calcificationB.URINARY TRACT INFECTIONS1.Renal and perirenal abscess2.Emphysematous pyelonephritis3.Emphysematous cystitis4.Fungal infection: Candida, Aspergillus5.Xanthogranulomatous pyelonephritisC.GENITAL INFECTION1.Fournier gangrene2.Postmenopausal tubo-ovarian abscess

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Diabetic Nephropathy Diabetic Cystopathy

Notes:

Diabetic Nephropathy =defined as persistent proteinuria (>500 mg of albumin/24 hours) + retinopathy + elevated blood pressure◊Most common cause of end-stage renal disease!*Incidence:*35-45% of IDDM; <20% of NIDDM;M > F*Histo:*diffuse intercapillary glomerulosclerosis*Mortality:*90% after 40 yearsEarly: √ renal enlargement (renal hypertrophy with glomerular expansion)Late: √ progressive decrease in size√ diffuse cortical hyperechogenicity with gradual loss of corticomedullary differentiation√ resistive index >0.7 (very late)IVP: √ contrast material may induce renal failure (= rise in serum creatinine level 1-5 days after exposure)◊Keep patient well hydrated with 0.45% saline!

Notes:

Diabetic Cystopathy *Cause:*autonomous peripheral neuropathy*Histo:*vacuolation of ganglion cells in bladder wall, giant sympathetic neurons, hypochromatic ganglion cells, demyelination ● insidious impairment of bladder sensation ● decreased reflex detrusor activity√ enlarged postvoid residual urine volume*Cx:*vesicoureteral reflux, recurrent pyelonephritis, pyohydronephrosis, overflow incontinence

Notes:

Acute Epididymitis =ACUTE EPIDIDYMO-ORCHITIS=most common acute pathologic process in postpubertal age secondary to ascending infection (usually beginning as prostatitis)*Incidence:*634,000 cases/year; <10 years in 0%;20-30 years in 72%*Organism:*E. coli + S. aureus (85%), Gonococcus (12%), TB (2%); nonspecific epididymitis in 20%(a)>35 years of age Escherichia coli + Proteus mirabilis(b)<35 years of age: Chlamydia trachomatis, Neisseria gonorrheae ● fever ● increasing pain over 1-2 days ● epididymal swelling + tenderness ● pyuria (95%) ● positive urine culture ● leukocytosis (50%) ● dysuria + frequency (25%) ● prostatic tenderness (infrequent)Location:may have focal involvement as in focal epididymitis (25%) often in epididymal tail◊Subsequent spread to testis is common: global orchitis (frequent), focal orchitis (10%)US: √ enlarged epididymis with decreased echogenicity√ reactive hydrocele + skin thickening√ enlarged spermatic cord containing hyperechoic fat√ thickening of tunica albuginea (in severe infection)Color Duplex (91% sensitive, 100% specific): √ increased number + concentration of identifiable vessels in affected region (= hyperemia)√ peak systolic velocity (PSV) >15 cm/s with PSV ratio >1.9 compared with normal side√ detection of venous flow√ diastolic flow reversal in testicular artery (due to epididymal edema with obstruction of venous outflow)NUC (true positive rate of 99%): √ symmetric perfusion of iliac + femoral vessels√ markedly increased perfusion through spermatic cord vessels (testicular + deferential arteries)√ curvilinear increased activity laterally in hemiscrotum on static images (also centrally if testis involved)√ increased activity of scrotal contents on static images (hyperemia + increased capillary permeability)*Rx:*antimicrobial therapy, scrotal elevation, bed rest, analgesics, ice packs*Cx:*(1)Focal / diffuse orchitis (20-40%)(2)Epididymal abscess (6%) / testicular abscess (6%)(3)Testicular infarction (3%) from extrinsic compression of testicular blood flow(4)Late testicular atrophy (21%)(5)Hydropyocele(6)Fournier gangrene*DDx:*(1)Testicular abscess (increased perfusion with centrally decreased uptake)(2)Hydrocele (normal perfusion, no uptake)(3)Testicular tumor (slightly increased perfusion; in- / decreased uptake; no associated epididymal hyperemia on CFI; positive tumor markers: HCG, AFP)

Notes:

Chronic Epididymitis US: √ enlarged hyperechoic epididymis

Notes:

ERECTILE DYSFUNCTION

=IMPOTENCE (term replaced due to negative connotation)=inability to obtain / maintain a penile erection sufficient for vaginal penetration in 50% or more attempts during intercourse*Physiology:* Psychogenic phase: stimuli from thalamic nuclei, rhinencephalon, limbic system converge in medial preoptic anterior hypothalamic area Neurologic phase: sacral nerve roots (S2-S4) contribute fibers to pelvic sympathetic plexus stimulation of cavernous n. (parasympathetic nerve) causes changes in blood flow resulting in full erection stimulation of pudendal n. (motor nerve) causes contraction of bulbocavernosus + ischiocavernosus muscle resulting in occlusion of veins + rigid erection *Risk factors:*hypertension, diabetes, smoking, CAD, peripheral vascular disease, pelvic trauma / surgery, blood lipid abnormalities, *Cause:* A.Organic cause (50%)1.Endocrine disorder (reducing serum testosterone / increasing serum prolactin2.Vascular disease (10-20%): increasing with age3.Neurologic disorder (10%): multiple sclerosis, spinal cord trauma, cervical spondylosis, spinal arachnoiditis, pelvic trauma, temporal lobe / idiopathic epilepsy, Alzheimer disease, Parkinson disease, tabes dorsalis, amyloidosis, primary autonomic insufficiency, cerebrovascular accidents, primary / metastatic tumor4.Chronic disease: diabetes mellitus, drugs (antihypertensives, anticonvulsants, alcohol, narcotics, psychotropic agents)5.Surgery: damage to pelvic sympathetic nerves / cavernous n. during radical prostatectomy / cystectomy Penile-brachial index (normal > 1.0) =highest penile artery pressure over mean brachial pressure <0.70 suggests large vessel disease *Rx:*nonsurgical external devices, sex therapy, surgery, intracavernosal injection of vasoactive agents, medical therapy

Notes:

FOURNIER GANGRENE
=FULMINANT FASCIITIS=uncommon potentially lethal necrotizing fasciitis of the scrotum*Incidence:*500 cases in literature*Organism:*(a)aerobes: S. aureus, E. coli, Proteus species, enterococci(b)anaerobes: Bacteroides fragilis, anaerobic streptococci, clostridia*Path:*cellulitis, myositis, fasciitis with soft-tissue necrosis*Histo:*thrombosis of subcutaneous vessels with gangrene of overlying skin*Age:*newborn to elderly*Predisposed:*diabetes mellitus (present in 40-60%) ● pain, fever, leukocytosis ● scrotal tenderness, erythema, swelling, crepitation✓In 95% primary focus of infection is recognizable (urethra, soft tissue of anorectal area, genital skin)!✓ gas in scrotal wall + perineum✓ scrotal skin thickening + normal testes*Mortality:*7-75%*Rx:*antibiotic therapy + surgery + hyperbaric oxygen*DDx:*epididymo-orchitis, gas-containing scrotal abscess, scrotal hernia with gas-containing bowel, scrotal emphysema from bowel perforation, extension of subcutaneous emphysema, air leakage + dissection due to faulty chest tube positioning

Notes:

GANGLIONEUROBLASTOMA
=tumor of sympathetic nervous system that is intermediate in cellular maturity between neuroblastoma and ganglioneuroma; metastatic potential*Incidence:*less common than neuroblastoma / ganglioneuroma*Age:*early childhood; M:F = 1:1Location:posterior mediastinum, abdomen↓ extension through neural foramen into epidural space↓ nerve root / spinal cord compression

Notes:

GANGLIONEUROMA
=benign neoplastic growth of autonomic ganglia=may represent end-stage of maturation of a neuroblastoma induced by chemotherapy / occurring spontaneously*Histo:*mixture of mature ganglion + Schwann cells*Age:*42-60% <20 years, 39% aged 20-39 years, 19% aged 40-80 years; M:F = 1:1Location:posterior mediastinum (25-43%); abdomen (52%), adrenal gland (20%); pelvis and neck (9%); oral + intestinal ganglioneuromatosis associated with MEN IIb ● respiratory symptoms, local pressure (40%) ● rarely hormone-active: diarrhea, sweating, hypertension, virilization, myasthenia gravis√ spherical / elliptical large well-defined encapsulated slow-growing mass√ tendency to surround blood vessels without compromising the lumen√ dumbbell-shaped large mass extending from paraspinous region through neural foramen into epidural space√ calcifications (8-27%)CT: √ homogeneous attenuation less than that of muscleMR: √ homogeneous + isointense with muscle on T1WI√ heterogeneous + hyperintense to muscle on T2WI*DDx:*neurofibroma (no calcification), schwannoma (no calcification), neuroblastoma (calcified)

Notes:

HEMANGIOMA OF URINARY BLADDER

*Incidence:*0.6% of primary bladder NEOPLASMS; 0.3% of all bladder tumors*Age:* <20 years (in >50%), M:F = 1:1*May be associated with:* (a)additional hemangiomas in 30%(b)Klippel-Trénaunay syndrome(c)Sturge-Weber syndrome*Histo:*capillary / venous / cavernous / hemangiolymphomatous form ● recurrent gross painless hematuria ● cutaneous hemangiomas over abdomen, perineum, thighs in 25-30%Location:dome, posterolateral wallSite:limited to submucosa (33%), muscular wall, perivesical tissue√ compressible solitary (2/3) / multiple (1/3) masses√ rounded well-marginated intraluminal mass√ diffuse bladder wall thickening + punctate calcifications (phleboliths)IVP: √ rounded / lobulated filling defectUS: √ solid predominantly hyperechoic mass√ hypoechoic spaces within thickened bladder wallCAVE:high risk of intractable hemorrhage at biopsy!

Notes:

HEMOLYTIC-UREMIC SYNDROME

◊Most common cause of acute renal failure in children requiring dialysis!=characterized by thrombotic microangiopathy with typical features of DIC*Cause:* (1)Infection: enterotoxic E. coli, Shigella dysenteriae I, Streptococcus pneumoniae, Salmonella typhi, Coxsackie virus, ECHO virus, adenovirus(2)Associated medical condition: pregnancy, SLE + other collagen vascular disease, malignancy, malignant hypertension(3)Drugs: oral contraceptives, cyclosporine, mitomycin, 5-fluorouracil*Pathogenesis:*capillary and endothelial injury to kidney leads to mechanical damage of RBCs + formation of hyaline microthrombi within renal vasculature + focal infarction*Age:*usually children <2 years*Histo:*microangiopathy including endothelial swelling + thrombus formation in glomerulus + renal arteriolesCLASSIC TRIAD: (1)microangiopathic hemolytic anemia(2)thrombocytopenia(3)acute oliguric / anuric renal failure leading to uremia ● recent bout of gastroenteritis (commonly with E. coli) ● sudden pallor, irritability ● bloody diarrhea ● dyspnea (due to fluid retention, heart failure, pleural effusion) ● convulsions ● rapid rise in blood urea nitrogen level out of proportion to plasma creatinine level (= result of cell lysis)@Kidney (sometimes only organ involved):√ kidneys of normal / slightly increased size√ hyperechoic cortexDoppler-US: √ diastolic flow absent / reversed / reduced(= increase in resistance to flow) √ return to normal waveforms predates return of urine outputScintigraphy: √ lack of renal perfusion@Liver: hepatomegaly, hepatitis@Pancreas: diabetes mellitus@Heart: myocarditis@Muscle: rhabdomyolysis@Intestines: perforation, intussusception, pseudomembranous colitis@Brain (20-50%): drowsiness, personality changes, coma, hemiparesis, seizures (up to 40%)*Prognosis:*complete spontaneous recovery (in 85%)

Notes:

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HEREDITARY CHRONIC NEPHRITIS
=ALPORT SYNDROME = probably autosomal dominant trait with presence of fat-filled macrophages ("foam cells") in the corticomedullary junction and medulla(a)males: progressive renal insufficiency, death usually < age 50(b)females: nonprogressive ● polyuria ● anemia ● salt wasting ● hyposthenuria ● nerve deafness ● ocular abnormalities (congenital cataracts, nystagmus, myopia, spherophakia) ● NO hypertension√ small smooth kidneys√ diminished density of contrast material√ cortical calcifications

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HORSESHOE KIDNEY
=two kidneys joined at poles by parenchymal / fibrous isthmus*Incidence:*1-4:1,000 births; 0.2-1% (autopsy series); M:F = 2-3:1*Associated with:* cardiovascular anomaly, skeletal anomaly, CNS anomaly, anorectal malformation, genitourinary anomaly (hypospadia, undescended testis, bicornuate uterus, ureteral duplication), trisomy 18, Turner syndrome in 50% with: (1)Caudal ectopia(2)Vesicoureteral reflux(3)Hydronephrosis√ fusion of R + L kidney at lower (90%) / upper (10%) pole√ renal long axis medially oriented√ isthmus at L4/5 between aorta + inferior mesenteric a.√ renal pelves and ureters situated anteriorly*Cx:*renal calculi

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Notes:

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HYDROCELE
=collection of fluid between parietal and visceral layers of tunica vaginalis; most common type of fluid collection in scrotum(A)PRIMARY = IDIOPATHIC HYDROCELEwithout predisposing lesion as congenital defect of lymphatic drainage (B)SECONDARY HYDROCELE(a)inflammation (epididymitis, epididymo-orchitis)(b)testicular tumor (in 10-40%)(c)trauma / postsurgical(d)torsion, infarction(C)CONGENITAL HYDROCELE=ascites in scrotum through communication with peritoneal cavity (= open processus vaginalis); may be associated with inguinal hernia(D)INFANTILE HYDROCELE=hydrocele with fingerlike extension into funicular process but without communication with peritoneal cavityUS: √ anechoic, good back wall, through transmission √ with low level echoes ± septations: hematocele / pyocele / cholesterol crystals

Notes:

HYDRONEPHROSIS

A.OBSTRUCTIVE UROPATHY = HYDRONEPHROSIS= dilatation of collecting structures without functional deficit B.OBSTRUCTIVE NEPHROPATHY = dilatation of collecting system with renal functional impairmentUS: Grading system of hydronephrosis: Grade 0=homogeneous central renal sinus complex without separationGrade 1=separation of central sinus echoes of ovoid configuration; continuous echogenic sinus periphery; 52% predictive value for obstructionGrade 2=separation of central sinus echoes of rounded configuration; dilated calices connecting with renal pelvis; continuity of echogenic sinus peripheryGrade 3=replacement of major portions of renal sinus; discontinuity of echogenic sinus peripheryAmount of collecting system dilatation depends on: (a)duration of obstruction(b)renal output(c)presence of spontaneous decompression◊Amount of residual renal cortex is of prognostic significance!

Acute Hydronephrosis Chronic Hydronephrosis Congenital Hydronephrosis Focal Hydronephrosis

Notes:

Acute Hydronephrosis *Cause:* (1)Passage of calculus with sites of stone impaction at points of ureteral narrowing:(a)ureterovesical junction (70%)(b)ureteropelvic junction(c)crossing of iliac vessels(2)Passage of blood clot (from carcinoma, AV malformation, trauma, anticoagulant therapy), sloughed necrotic papilla(3)Suture on ureter(4)Ureteral edema following instrumentation(5)Sulfonamide crystallization in nonalkalinized urine(6)Normal pregnancy ● pain (50%) ● urinary tract infection (36%) ● nausea + vomiting (33%)√ normal-sized kidney with normal parenchymal thickness√ increasingly dense nephrogram√ delayed opacification of collecting system (decreased glomerular filtration)√ increasingly dense nephrogram over time ("obstructed nephrogram")√ dilated collecting system + ureter√ widening of forniceal angles√ delayed images demonstrate site of obstruction at the end of a persistent column of contrast material in a dilated urinary collecting system√ vicarious contrast excretion through gallbladder (uncommon)NECT: √ dilatation of renal collecting system + ureter√ inflammation of perinephric ± periureteral fat√ calcified ureteral stone √ ureteral rim sign (77%) = thickening of ureteral wall secondary to edema from stone impaction with small stones (DDx: in *% of phleboliths)US: √ ureteral jet not detectable / continuous at low levelFalse-negatives: staghorn calculus filling entire collecting system, hyperacute renal obstruction (system not yet dilated), spontaneous decompression of obstruction, fluid-depleted patient with partial obstruction, dehydrated neonate False-positives: full bladder, increased urine flow (overhydration, medications, following urography, diabetes insipidus, diuresis in nonoliguric azotemia), acute pyelonephritis, postobstructive / postsurgical dilatation, vesicoureteral reflux Imposters: parapelvic cysts, sinus vessels, prominent extrarenal pelvis Duplex: √ mean RI of 0.77 ± 0.05 (0.63 ± 0.06 in nonobstructed kidney)Caution:RI often normal in chronic obstruction; nonobstructive renal disease may elevate RIs√ ≥0.08 difference in RI in right-to-left comparison with unilateral obstruction*Cx:*spontaneous urinary extravasation (10-18%) from forniceal / pelvic tear (= pyelosinus reflux)

Notes:

Chronic Hydronephrosis = most frequent cause of abdominal mass in first 6 months of life (25% of all neonatal abdominal masses) *Cause:* (a)acquired: benign + malignant tumors of the ureter; ureteral strictures; retroperitoneal tumor / fibrosis; neurogenic bladder; benign prostatic hyperplasia; cervical / prostatic carcinoma; pelvic mass (lymphoma, abscess, ovarian); urethral polyps; urethral neoplasm; acquired urethral strictures(b)congenital ● insidious course√ large kidney with wasted parenchyma√ diminished nephrographic density (decreased clearance)√ early "rim" sign (thin band of radiodensity surrounding calices)√ delayed opacification of collecting system√ moderate to marked widening of collecting system √ tortuous dilated ureterNUC: √ photopenic area during vascular phase√ accumulation of radionuclide tracer within hydronephrotic collecting system on delayed images*Cx:*superimposed infection (= pyonephrosis)

Notes:

Congenital Hydronephrosis Mostly isolated malformation *Incidence:*1:100-300 births*Risk of recurrence:*2-3% for siblings*Age at presentation:*25% by age 1 year,55% by age 5 years *Cause:* 1.UPJ obstruction (22-40-67%)2.Posterior urethral valves (18%)3.Ectopic ureterocele (14%)4.Prune belly syndrome (12%)5.Ureteral + UVJ obstruction (8%)6.Others: severe vesicoureteral reflux, bladder neck obstruction, hypertrophy of verumontanum, urethral diverticulum, congenital urethral strictures, anterior urethral valves, meatal stenosis*May be associated with:* Down syndrome (17-25%) ● palpable abdominal mass ● intermittent flank + periumbilical pain ● failure to thrive ● vomiting ● hematuria, infectionLocation:70% unilateralOB-US: √ AP diameter of renal pelvis ≥5 mm between 15-20 weeks, ≥8 mm at 20-30 weeks, ≥10 mm after 30 weeks MA√ ratio of AP diameter of renal pelvis to kidney >50%√ caliceal distension communicating with renal pelvis◊Postnatal evaluation after 4-7 days of age (because of decreased GFR + relative dehydration in first days of life)!*Prognosis:*parenchymal atrophy + renal impairment (dependent on severity + duration)

Notes:

Focal Hydronephrosis =HYDROCALICOSIS = HYDROCALYX = obstructed drainage of one portion of kidney*Cause:*(1)Congenital: partial / complete duplication(2)Infectious stricture: eg, TB(3)Infundibular calculus(4)Tumor(5)Trauma√ unifocal mass, commonly in upper pole√ absent polar group of calices (early)√ dilated polar group (late) with displacement of adjacent calices√ delayed opacification in obstructed group√ focally replaced nephrogram US: √ anechoic cystic lesion with smooth marginsCT: √ focal area of water density with smooth margin and thick wall

Notes:

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IMPOTENCE
=inability to have + maintain an erection adequate for sexual intercourse*Incidence:*10 million Americans*Cause:* A.ORGANIC (majority): diabetes (2 million), vascular disease, cancer surgery, spinal cord injury, pelvic trauma, endocrine problem, multiple sclerosis, alcoholism, drug-associated impotence(a)failure to initiate (neurogenic)(b)failure to fill (arteriogenic)(c)failure to store (venogenic)(d)end organ diseaseB.PSYCHOGENIC*Rx:*(1)Vascular reconstructive surgery(2)Oral / intracavernous pharmacotherapy(3)Vacuum erection devices(4)Penile prosthesis placement(a)nonhydraulic: semirigid, malleable, positionable(b)hydraulic*also see* ERECTILE DYSFUNCTION

Notes:

JUXTAGLOMERULAR TUMOR
=RENINOMA = very rare tumor arising from renin-producing juxtaglomerular cells*Incidence:*<30 cases reported*Age:*mean age of 31 years; 50% <21 years; M < F*Path:*small foci of hemorrhage + pseudocapsule*Histo:*tumor resembles hemangiopericytoma ● typical features of primary reninism: ● hypertension ● hyperreninemia ● secondary hyperaldosteronism ● moderate to severe headaches ● polydipsia, polyuria, enuresisLocation:just beneath renal capsule√ renal mass of usually 2-3 cm in sizeUS: √ echogenic mass ± areas of necrosis / hemorrhageCT (thin overlapping cuts): √ isodense tumor on NECT, hypodense on CECTAngio: √ angiographically hypo- / avascular tumor√ renal venous blood sampling yields high renin level on affected side*Dx:*combination of elevated renin without renal arterial lesion + hypovascular solid renal mass*DDx of renin elevation:* Wilms tumor, hypernephroma, lung cancer, paraovarian tumor, fallopian tube adenocarcinoma, epithelial liver hamartoma, orbital hemangiopericytoma, pancreatic cancer, angiolymphoid hyperplasia

Notes:

LEUKEMIA

◊Most common malignant cause of bilateral global renal enlargement!*Incidence:*renal involvement in 63% of autopsiesA.FOCALACCUMULATION OF LEUKEMIC CELLS (rare)**chloroma** (= granulocytic sarcoma) of acute myeloblastic leukemia, myeloblastoma, myeloblastic sarcoma ● may antedate other manifestations of leukemia√ unifocal mass in renal cortex / renal sinusB.DIFFUSE INVOLVEMENTleukemic cells infiltrate the interstitial tissue + renal sinus; tubules are replaced (more common in lymphocytic than in granulocytic forms); no relationship to peripheral white blood cell count ● renal impairment (from leukemic infiltrate, hyperuricemia, septicemia, hemorrhage) ● hypertension√ large kidneys bilaterally with smooth contours√ normal or diminished density on nephrogram√ occasionally attenuated collecting system (DDx: renal sinus lipomatosis)√ nonopaque filling defects on IVP (clot, uric acid)√ renal / subcapsular / perinephric hemorrhage frequent√ retroperitoneal lymphadenopathyUS: √ loss of definition + distortion of central sinus complex√ normal to increased coarse echoes throughout renal cortex + preservation of renal medullae√ single / multiple focal anechoic masses*DDx:* Hodgkin disease, malignant lymphoma, multiple myeloma

Notes:

LEUKOPLAKIA
=KERATINIZING SQUAMOUS METAPLASIA / DYSPLASIA = DYSKERATOSIS*Cause:*chronic infection (80%) / stones (40%)*Histo:*large confluent areas / scattered patches of squamous metaplasia of transitional cell epithelium with keratinization + cellular atypia in deeper layers*Peak age:*4th-5th decade;M:F = 1:1 (with involvement of renal pelvis) M:F = 4:1 (with involvement of bladder) ● hematuria (30%) ● recurrent UTIs ● pathognomonic passage of gritty flakes, soft-tissue stones, white chunks of tissue (desquamated keratinized epithelial layers) leading to colic, fever, chillsLocation:bladder > renal pelvis > ureter; bilateral in 10%√ corrugated / striated irregularities of pelvicaliceal walls, localized / generalized√ plaquelike intraluminal mass with "onion skin" pattern of contrast material in interstices√ caliectasis + pyelectasis common (with obstruction)√ ridging / filling defects of ureter√ associated with calculi in 25-50%*Cx:*premalignant condition for epidermoid carcinoma in 12% (controversial!)

Notes:

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LOBAR NEPHRONIA
= ACUTE FOCAL BACTERIAL NEPHRITIS = focal variant of acute pyelonephritis with single / multiple areas of suppuration + necrosis*Organism:*E. coli > Proteus > Klebsiella*Predisposed:*patients with altered host resistance (diabetes [60%], immunosuppression), chronic catheterization, mechanical / functional obstruction, trauma

- fever, flank pain, pyuria*Site:*usually involves entire renal lobe√ focal area of absent nephrogram / distorted pyelogram√ renal arteries displaced, renal veins compressed√ hypoechoic mass with ill-defined margins and disruption of corticomedullary border, NO fluid collection√ low attenuation zone with poorly defined transition to surrounding parenchyma√ Ga-67 uptake√ vesicoureteral reflux often present*Cx:*scarring, abscess

Notes:

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LOCALIZED CYSTIC DISEASE
=multiple simple cysts involving only one portion of the kidney ● no family history*Histo:*dilated ducts and tubules varying in size from mm to several cm*Prognosis:*not progressive

Notes:

LYMPHOMA
*Incidence:*in 2.7-6% renal involvement*Types:* A.NON-HODGKIN LYMPHOMArenal involvement detected in 5% of abdominal CT, in 33-65% of autopsies; occurs usually late in diseaseB.HODGKIN DISEASErenal involvement in 13% of autopsies*Patterns of involvement:* (a)primary renal lymphoma (very rare)(b)hematogenous dissemination:-single / multiple foci-diffuse infiltration(c)contiguous extension from adjacent pararenal lymphomatous disease, usually extranodal ● clinically silent (50%) ● flank pain, palpable mass, weight loss ● hematuria ● compromise of renal function (urinary tract obstruction, renal vein compression, diffuse infiltration of kidney, superimposed infarct, amyloidosis, hypercalcemia)√ unilateral:bilateral = 3:1√ multiple nodular masses (29-61%)√ invasion from retroperitoneal disease (11%) with involvement by transcapsular / transsinus extension√ single bulky tumor (7%), small solitary tumor (7-48%)√ diffuse infiltration (6-19%), microscopic infiltration (7%)CECT: √ usually homogeneous poorly marginated masses less dense than renal parenchymaUS: √ single / multiple anechoic / hypoechoic masses√ renal enlargement + decreased parenchymal echoes√ loss of renal sinus echoesAngio: √ neovascularity, encasement, vascular displacement (occasionally palisade-like configuration)

Notes:

MALACOPLAKIA
=uncommon chronic inflammatory response to Gram-negative infection*Organism:*E. coli (in 94%);diabetes mellitus predisposes*Histo:*submucosal histiocytic granulomas containing large foamy mononuclear cells (Hansemann macrophages) with intracytoplasmic basophilic PAS-positive inclusion bodies (Michaelis-Gutmann bodies) consisting of incompletely destroyed E. coli bacterium surrounded by lipoprotein membranes*Peak age:*5th-7th decade; M:F = 1:4 ● hematuria ● raised yellow lesion <3 cm in diameterLocation:bladder > lower 2/3 of ureter > upper ureter > renal pelvis; multifocal in 75%; bilateral in 50%√ multiple dome-shaped smooth mural filling defects√ scalloped appearance if lesions confluent√ generalized pelviureteral dilatation (if obstructive)√ displacement of pelvicaliceal system + distorted central sinus complex√ multifocal parenchymal masses may cause diminished / absent nephrogram*DDx:*pyeloureteritis cystica

Notes:

MALPOSITIONED TESTIS
=MALDESCENDED TESTIStestes are normally within scrotum by 28-32 weeks MA*Incidence:*early 3rd trimester in 10%; at birth in 3.7% (in babies >2,500 g in 3.4%; in premature babies in 30%); beyond 3 months of age in 1%*Test sensitivity:* MR:modality of choiceUS:20-88%; very sensitive in inguinal canal need to identify mediastinum testis (DDx: lymph node)CT:95% (testis <1 cm cannot be detected) no spermatic cord in inguinal canalVenography:50-90%*Cx:*(1)Sterility(2)Malignancy: most commonly seminoma, 30-50 x risk increase = 1:1,000 men/year, 4-11% of all testicular tumors found in cryptorchidism; risk remains increased even after orchiopexyAnnual screening until at least age 35!(3)Torsion: 10 x risk in cryptorchidism*Rx:*surgery at 9-12 months of age*DDx:*(1)Rudimentary testis(2)Pars intravaginalis gubernaculum= nonatrophied bulbous termination (3)Congenital absence = monorchia / anorchia (in 3-5%)

Cryptorchidism (20-29%) Ectopia Testis (1%) Pseudocryptorchidism (70%) Undescended Testis

Notes:

Cryptorchidism (20-29%)
=arrested descent of testis along its normal course*Associated with:*prune belly syndrome (bilateral cryptorchidism), Prader-Willi syndrome, Beckwith-Wiedemann syndrome, Noonan syndrome, Laurence-Moon-Biedl syndrome, trisomies 13, 18, 21 ● nonpalpable testisLocation:high scrotal position (50%); canalicular = between internal + external inguinal ring (20%); abdominal (10%); bilateral in 10%The most craniad possible point of an undescended testis is the lower pole of the ipsilateral kidney!√ failure to visualize testis within scrotum√ small atrophic testis with generalized decreased echogenicity + demonstrable mediastinum testis

Notes:

Ectopia Testis (1%)
=deviation from the usual pathwayLocation:interstitial = groin (on external oblique muscle), pubopenile = root of penis, perineal, femoral triangle, on opposite side

Notes:

Pseudocryptorchidism (70%)
=RETRACTILE TESTIS=unusually spastic cremasteric muscle

Notes:

Undescended Testis = retractile testis + cryptorchidism

Notes:

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MECKEL-GRUBER SYNDROME
=autosomal recessive disease characterized by occipital encephalocele, polycystic kidneys, polydactyly*Incidence:*1:12,000-50,000; more common among Yemenite Jews*Risk of recurrence:*25%; carrier frequency of 1:56 ● history of affected siblingsOB-US: √ large polycystic kidneys containing 2- to 10-mm cysts√ occipital encephalocele√ postaxial polydactyly√ microcephaly√ cleft lip and palate√ moderate-to-severe oligohydramnios (onset midtrimester)√ inability to visualize urine within fetal bladderOB management: 1.Chromosomal analysis to exclude trisomy 13 (if no prior family history)2.Option of pregnancy termination <24 weeks GA3.Nonintervention for fetal distress >24 weeks GA*Prognosis:*invariably fatal at birth due to pulmonary hypoplasia + renal failure*DDx:*trisomy 13

Notes:

MEDULLARY CYSTIC DISEASE
=NEPHRONOPHTHISIS*Histo:*variable number of medullary cysts (100 µ to 2 cm) + progressive periglomerular and interstitial fibrosis + tubular atrophy with dilatation of some proximal tubules*Types:* (1)MEDULLARY CYSTIC DISEASE = ADULT ONSETautosomal dominant, in young adults, rapidly progressive course with uremia + death in 2 years (2)JUVENILE NEPHRONOPHTHISIS = JUVENILE ONSETautosomal recessive, in children 3-5 years, average duration of 10 years before uremia and death occurs ● salt-wasting, polyuria, hyposthenuria, polydipsia ● failure to thrive, growth retardation (in early teens) ● uremia, severe anemia, normal sediment, hypertension (only in late phase)√ bilateral normal / small kidneys with smooth contour + thin cortexIVP: √ poor opacification of renal collecting system√ "medullary nephrogram" = medullary striations persistent for up to 2 hours; occasionally replaced by sharply defined multiple thin-walled lucenciesRetrograde pyelogram: √ communication between collecting system + cystsUS / CT: √ increased parenchymal echogenicity + loss of corticomedullary junction√ multiple small medullary / corticomedullary cysts

Notes:

MEDULLARY SPONGE KIDNEY
=dysplastic cystic dilatation of papillary + medullary portions of collecting ducts (first few generations of metanephric duct branchings)*Incidence:*0.5%*Age:*young to middle-aged adults; sporadic*May be associated with:*Ehlers-Danlos syndrome, parathyroid adenoma, Caroli disease ● often asymptomatic√ medullary nephrocalcinosis (40-80%) with one / more calculi up to 5 mm√ "bunch of flowers" = thick dense streaks of contrast material radiating from pyramids peripherally representing papillary cysts / ectatic ducts (DDx: dense papillary blush in normals)√ may be unilateral in 25%√ may involve only one pyramid / all pyramids (25%)*Cx:* urolithiasis, hematuria, infection*DDx:* (1)Normal variant ("papillary blush" without distinct streaks / nephrocalcinosis / pyramidal enlargement)(2)Renal tuberculosis (larger more irregular calcifications + cavitations + strictures + ulcerations)(3)Papillary necrosis (sloughed papilla + caliceal ring sign)(4)Medullary nephrocalcinosis (no ectatic ducts / cysts, calcifications beyond pyramids)(5)Juvenile polycystic kidney disease (bilateral renal enlargement + hepatic periportal fibrosis)(6)Caliceal diverticulum (small, solitary, located between pyramid)

Notes:

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MEGACALICOSIS
=CONGENITAL MEGACALICES = nonprogressive caliceal dilatation caused by hypoplastic medullary pyramids*Age:*any age; M >> F*May be associated with:*primary megaureter ● normal glomerular filtration rateSite:entire kidney / part of kidney; unilateral / bilateral√ kidney usually enlarged with prominent fetal lobation√ reduced parenchymal thickness (medulla affected, NOT cortex)√ mosaic-like arrangement of dilated calices (polygonal + faceted appearance, NOT globular as in obstruction)√ increased number of calices√ ABSENT caliceal cupping (semilunar instead of pyramidal configuration of papillae)√ NO dilatation of pelvis / ureters, NORMAL contrast excretion*Cx:*(1) Hematuria (2) Stone formation

Notes:

MEGACYSTIS-MICROCOLON SYNDROME
=MEGALOCYSTIS-MICROCOLON-INTESTINAL HYPOPERISTALSIS SYNDROME (MMIH)= functional obstruction of bladder + colon characterized by (1)enlarged urinary bladder(2)small colon(3)strikingly short small intestine suspended on a primitive dorsal mesentery(4)markedly enlarged hydronephrotic kidneys with little remaining parenchyma*Incidence:*26 cases reported; M:F = 1:7*May be associated with:* diaphragmatic hernia, PDA, teeth at birth ● distended abdomen (large bladder + dilated small bowel loops) ● overflow incontinence ● intestinal pseudo-obstruction (poor emptying of stomach, NO peristaltic activity of small bowel)OB-US: √ normal amount of amniotic fluid / polyhydramnios (in spite of dilated bladder = "nonobstructive obstruction")√ massive + progressive bladder distension with poor emptying√ bilateral megaloureters√ ± hydronephrosis√ female sexBE: √ microcolon (transient feature of "unused colon") with narrow rectum + sigmoid√ malrotation / malfixation or foreshortening of small bowelVCUG √ distended unobstructed bladder with poor / absent muscular function*Prognosis:*lethal in most cases (a few months of age)

Notes:

MEGALOURETER
=CONGENITAL PRIMARY MEGAURETER = TERMINAL URETERECTASIS = ACHALASIA OF URETER= URETEROVESICAL JUNCTION OBSTRUCTION = intrinsic congenital dilatation of lower juxtavesical orthotopic ureter *Cause:*aperistaltic juxtavesical (1.5 cm long) segment secondary to faulty development of muscle layers of ureter (functional, NOT mechanical obstruction)*Incidence:*all ages; second most common cause of hydronephrosis in fetus and newborn;M:F = 2-5:1 *Associated disorders* (in 40%): (a)contralateral: UPJ obstruction, reflux, ureterocele, ureteral duplication, renal ectopia, renal agenesis(b)ipsilateral: caliceal diverticulum, megacalicosis, papillary necrosis ● asymptomatic (mostly) ● pain ● abdominal mass ● hematuria ● infectionLocation:L:R = 3:1, bilateral in 15-40%√ prominent localized dilatation of pelvic ureter (up to 5 cm in diameter) usually not progressive, but may involve entire ureter + collecting system√ vigorous nonpropulsive to-and-fro motion in dilated segment√ functional smoothly tapered narrowing of intravesical ureter√ NO reflux, NO stenosis

Notes:

MESOBLASTIC NEPHROMA
=FETAL RENAL HAMARTOMA = LEIOMYOMATOUS HAMARTOMA = BENIGN CONGENITAL WILMS TUMOR = BENIGN FETAL HAMARTOMA = FETAL MESENCHYMAL TUMOR = BOLANDE TUMOR = CONGENITAL FIBROSARCOMA = FIBROMYXOMA=nonfamilial benign fibromyomatoid mass arising from renal connective tissue*Incidence:*most common renal neoplasm in neonate; 3% of all renal neoplasms in children*Age:*3 months mean age at presentation; may occasionally go undetected until adulthood; M > F*Histo:*smooth muscle cells + immature fibroblasts resembling leiomyoma containing trapped islands of embryonic glomeruli, tubules, vessels, hematopoietic cells, cartilage*In 14% associated with:*prematurity, polyhydramnios, GI + GU tract malformations, neuroblastoma ● large flank mass ● hematuria (20%) / hypertension (4%), anemia√ usually replaces 60-90% of renal parenchyma√ usually solid but may produce multiple cystic spaces√ NO sharp cleavage plane toward normal parenchyma, may extend beyond capsule√ calcifications (rare)√ NO venous extension (DDx from Wilms tumor)IVP: √ large noncalcified renal mass with distortion of collecting system√ usually NO herniation into renal pelvis (DDx from MLCN)US: √ evenly echogenic tumor with concentric echogenic + hypoechoic rings resembling uterine fibroids√ complex mass with hemorrhage + cyst formation + necrosisAngio: √ hypervascular mass with neovascularity + displacement of adjacent vessels*Cx:*transformation to metastasizing spindle cell sarcoma (rare)*Rx:*complete resection*Prognosis:*excellent

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METASTASES TO KIDNEY

Most common malignant tumor of the kidney (2-3 times as frequent as primaries in autopsy studies)!5th most common site of metastases (after lung, liver, bone, adrenals)!most common primaries: bronchus, breast, opposite kidney, non-Hodgkin lymphoma, colon less common primaries: stomach, cervix, ovary, pancreas, prostate, chloroma, myeloblastoma, myeloblastic sarcoma, melanoma (45% incidence), osteogenic sarcoma, choriocarcinoma (10-50% incidence), Hodgkin lymphoma, rhabdomyosarcoma ● usually asymptomatic bilateral multiple small masses (due to brief survival of patient)*DDx on CT:*lymphoma, bilateral RCC, multiple renal infarcts, acute focal bacterial nephritis, infiltrating TCC

Notes:

MULTICYSTIC DYSPLASTIC KIDNEY

=MULTICYSTIC DYSGENETIC KIDNEY (MCDK)= MULTICYSTIC KIDNEY (MCK) = Potter Type I◊Second most common cause of an abdominal mass in neonate (after hydronephrosis)!◊Most common form of cystic disease in infants!*Incidence:*1:10,000 (for bilateral MCDK); M:F = 2:1 (for unilateral MCDK); more common among infants of diabetic mothers*Risk of recurrence:*2-3%*Etiology:* (sporadic) generalized interference with ureteral bud function before 8-10 weeks of fetal life*Pathophysiology:*ureteral obstruction / atresia interferes with ureteral bud division + inhibits induction and maturation of nephrons; collecting tubules enlarge into cysts*Histo:*immature glomeruli + tubules reduced in number + whirling mesenchymal tissue, cartilage (33%), cysts ● abdominal mass ● asymptomatic if unilateral (may go undetected until adulthood) ● recurrent urinary tract infections, intermittent abdominal pain, nausea + vomiting, hematuria, failure to thrive ● fatal due to pulmonary hypoplasia if bilateralFatal form:bilateral MCDK (4.5-21%), contralateral renal agenesis (0-11%)Location: 1.UNILATERAL multicystic dysplastic kidneymost common form (80-90%); L:R = 2:1 secondary to pelvoinfundibular atresia *Associated with*anomalies of contralateral side in 20-40-50%:(1)Ureteropelvic junction obstruction (7-27%)(2) Horseshoe kidney (5-9%)(3)Ureteral anomalies (5%)(4)Renal hypoplasia (4%)(5)Vesicoureteral reflux(6)Malrotation(7)Renal agenesis*Associated with*ipsilateral anomalies:(1)Vesicoureteral reflux (25%)(2)Ectopic ureter2.SEGMENTAL / focal renal dysplasia="multilocular cyst" secondary to(a)high-grade obstruction of upper pole moiety in duplex kidney from ectopic ureterocele(b)single obstructed infundibulum3.BILATERAL cystic dysplasiain the presence of severe obstruction in utero from posterior urethral valves / urethral atresia with oligohydramnios + pulmonary hypoplasia *Types:* (1)Multicystic kidney (Potter IIa)√ large kidney with multiple large cysts + little visible renal parenchyma(2)Hypoplastic / diminutive form (Potter IIb)√ echogenic small kidney APPEARANCE RELATED TO SITE OF OBSTRUCTION @ureteropelvic junction√ single / several large / multiple medium-sized cysts in large kidney@distal ureter / urethra√ small / no cysts in small kidney APPEARANCE RELATED TO TIME OF INSULT (a)early onset between 8th-11th week√ small / atretic renal pelvis + calices√ 10-20 cysts + loss of reniform appearance(b)late onset = HYDRONEPHROTIC FORM√ large central cyst (= dilated pelvis) often communicating with cysts√ some renal function may be demonstrated √ large kidney with lobulated contour in infancy√ incidental finding of small kidney in adults (secondary to arrested growth)√ ipsilateral atretic ureter√ contralateral renal hypertrophy√ calcification: curvilinear / ringlike in wall of cysts in30% of adults, rarely in childrenIVP + NUC: ◊NUC preferred over IVP in first month of life as concentrating ability of even normal neonatal kidneys is suboptimal!√ no function (rarely faint contrast accumulation)US: √ normal renal architecture replaced √ random cysts of varying shape + size ("cluster of grapes") with largest cyst in peripheral nonmedial location (100% accurate)√ cysts separated by septa (100% accurate)√ central sinus complex absent (100% accurate)√ no communication between multiple cysts (93% accurate)√ no identification of parenchymal rim or corticomedullary differentiation (74% accurate)√ cysts begin to disappear in infancy√ kidney may be small + atrophic (as little as 1 g) / normal / large√ oligohydramnios in bilateral MCDK / unilateral MCDK + contralateral urinary obstructionAngio: √ absent / hypoplastic renal artery; angiography unnecessary since a DDx to long-standing functionless kidney is not possible OB-management: (1)Routine antenatal care + evaluation by pediatric urologist following delivery if unilateral(2)Option of pregnancy termination if ≤24 weeks GA(3)Nonintervention for fetal distress if >24 weeks GA *Cx:*(1)Renin-dependent hypertension (rare)(2)Malignancy in <1:330*Rx:*(1)follow-up(2)nephrectomy (in hypertension / if kidney does not involute)*DDx:*(1)hydronephrosis(2)renal dysplasia with cysts (associated with partial obstruction)

Notes:

MULTILOCULAR CYSTIC RENAL TUMOR

=rare nonhereditary benign renal neoplasm originating from metanephric blastema possibly representing the benign end of a spectrum with solid Wilms tumor at the malignant end= BENIGN MULTILOCULAR CYSTIC NEPHROMA = POLYCYSTIC NEPHROBLASTOMA = WELL-DIFFERENTIATED POLYCYSTIC WILMS TUMOR= BENIGN CYSTIC DIFFERENTIATED NEPHROBLASTOMA = CYSTIC PARTIALLY DIFFERENTIATED NEPHROBLASTOMA= MULTILOCULAR CYSTIC NEPHROMA = PERLMANN TUMOR = MULTILOCULAR RENAL CYST = CYSTIC ADENOMA / HAMARTOMA / LYMPHANGIOMA = PARTIALLY POLYCYSTIC KIDNEY*Age:*biphasic age + sex distribution: <4 years in 73% male, >4 years in 89% female(a)3 months to 2 years of age (65%), 5-30 years (5%); M:F = 2:1(b)>30 years (30%); M:F = 8:1◊90% of tumors in males occur in first 2 years of life (peak 3-24 months)!◊Most of the lesions in females occur between ages 4 and 20 or 40 and 60!*Path:*solitary large well-circumscribed multiseptated mass of noncommunicating fluid-filled loculi, surrounded by thick fibrous capsule + compressed renal parenchyma; cyst size between mm up to 4 cm*Histo:*(gross anatomic features are identical)1.**Cystic nephroma**
fibrous tissue septa of undifferentiated mesenchymal and primitive glomerulotubular elements surround cysts lined by flattened cuboidal epithelium; NO blastemal / other embryonal elements 2.**Cystic partially differentiated nephroblastoma**
=CPDNpredominantly cystic lesion with septa containing blastemal / other embryonal elements ● commonly asymptomatic painless abdominal mass ● ± sudden and rapid enlargement ● pain, hematuria, urinary tract infectionLocation:unilateral, often replacing an entire renal pole (usually lower pole) Size:average size of 10 cm (few cm to 33 cm)√ sharply well-circumscribed (characteristic) multiseptated cystic renal mass√ tumor surrounded by thick fibrous capsule√ cluster of noncommunicating "honeycombed" cysts of various sizes separated by thick septa√ smaller closely spaced cysts appear as solid nodules√ contrast enhancement of septations (secondary to tortuous fine vessels coursing through septa)√ curvilinear to flocculent calcification of septa / capsuleIVP: √ distortion of calices / hydronephrosis secondary to nonfunctional mass√ tendency for herniation of tumor cysts into renal pelvis (nonspecific, also seen with Wilms tumor + RCC)US: √ cluster of cysts separated by thick septa (SUGGESTIVE PATTERN)√ occasionally solid echogenic character (due to very small cysts / jellylike contents)CT: √ cysts with attenuation equal to / higher than water (gelatinous fluid) *Cx:*local recurrence / coexistent Wilms tumor (extremely rare)*Rx:*nephrectomy*DDx:*(1)Cystic Wilms tumor (overlapping age)(2)Clear cell sarcoma (poor prognosis)(3)Cystic mesoblastic nephroma (most common renal tumor of infancy)(4)Cystic RCC (mean age of 10 years)(5)Segmental form of multicystic dysplastic kidney

Notes:

MULTIPLE MYELOMA

◊It is essential that dehydration is avoided!*Impairment of renal function:* (1)Precipitation of abnormal proteins (Bence-Jones ± Tamm-Horsfall protein casts) into tubule lumen (30-50%)(2)Toxicity of Bence-Jones proteins on tubules(3)Impaired renal blood flow secondary to increased blood viscosity(4)Amyloidosis(5)Nephrocalcinosis from hypercalcemia◊Contrast-induced renal failure in multiple myeloma is not seen with greatly increased frequency! ● Tamm-Horsfall proteinuria (tubular cell secretion) √ smooth normal to large kidneys (initially), become small with time√ occasionally attenuated pelvo-infundibulo-caliceal system√ normal to diminished contrast material density; increasingly dense in acute oliguric failureUS: √ normal to increased echogenicityNUC in bone scintigraphy: √ nonspecific increased parenchymal activity

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MYCETOMA
=FUNGUS BALL*Organism:*typically Candida, Aspergillus, Mucor, Cryptococcus, Phycomycetes, Actinomycetes mostly mycelial (M-form) or occasionally yeast cells (Y-form)*Predisposed:*diabetics, debilitating illness, prolonged antibiotic therapy, leukemia, lymphoma, thymoma, immunosuppression ● flank pain, passing of tissue, hematuria (extremely rare) ● renal candidiasis associated with candidemia ● Candida cystitis preceded by vaginal candidiasis√ unilateral nonvisualization of kidney (most frequent)√ large irregular filling defect extending into dilated calices (retrograde contrast study)√ necrotizing papillitis from Candida nephritis (common)√ lacelike pattern (on antegrade contrast study)

Notes:

NEPHROBLASTOMATOSIS
=multiple / diffuse nephrogenic rests (= abnormally persistent nephrogenic cells with potential to form Wilms tumor)*Incidence:*in 41% with unilateral Wilms tumor, in 94% with metachronous contralateral Wilms tumor, in 99% with bilateral Wilms tumorPathogenesis:primitive renal tissue (metanephrogenic blastema) normally present up to 36 weeks of gestational age; embryonal renal tissue in mature kidney after birth retains potential to form nephroblastomatosis / Wilms tumor*Histo:*contains only primitive epithelial cell line without mesenchymal elements (as seen in Wilms tumor)*Age:*neonatal period, infancy, childhood*Associated with:* hemihypertrophy, sporadic aniridia, Klippel-Trénaunay syndrome, Beckwith-Wiedemann syndrome, trisomy 18, pseudohermaphroditism, splenic agenesis with hepatic malformation, Drash syndrome Site:(a)at periphery of renal lobe = perilobar nephrogenic rest associated with a 1-2% risk of Wilms tumor(b)within renal lobe = intralobar nephrogenic restassociated with 4-5% risk of Wilms tumor A.**Multifocal (juvenile) nephroblastomatosis**

most common form =discrete islands of rests in cortex / columns√ may escape detection with imaging√ ± deformation of pelvicaliceal structures√ kidneys may be enlargedB.**Superficial diffuse (late infantile) nephroblastomatosis**

=superficial continuous peripheral ring of rests around normal medulla + pyramid*Age:*<2 years√ nephromegaly◊Strong association with Wilms tumor!C.**Universal / panlobar (infantile) nephroblastomatosis**

rare form =entire renal parenchyma diffusely involved ● may develop renal failure√ bilateral renal enlargement US: √ subtle subcapsular hypoechoic / isoechoic / hyperechoic nodules√ nephromegaly with decreased parenchymal echoesCECT (preferred study): √ nonenhancing subcapsular nodules√ splaying + elongation of collecting systemMR (43% sensitivity, 58% sensitivity with enhancement): √ homogeneously hypointense lesions on T1WI√ homogeneously hypointense lesions on T2WI for sclerosing / involuting type of nephroblastomatosis√ isointense lesions on T2WI for hyperplastic / neoplastic type of nephroblastomatosis√ hypointense lesions on enhanced T1WI*Cx:*malignant transformation (enlargement of rest / development of mass)*Rx:*amenable to chemotherapy

Notes:

MYELOLIPOMA
*Prevalence:*0.08-0.2% (autopsy series)*Cause:*? metaplasia of adrenal cortical cells precipitated by chronic stress / degeneration*Path:*mature fat interspersed with hematopoietic cells resembling bone marrow + pseudocapsule*Histo:*variable mixture of myeloid cells, erythroid cells, megakaryocytes, lymphocytes*Associated with:*endocrine disorders in 7% (Cushing syndrome, 21-hydroxylase deficiency), nonhyperfunctioning adenoma (15%)Location:(a)adrenal gland (85%)(b)extraadrenal (15%): retroperitoneal (12%), intrathoracic (3%)*Site:*unilateral : bilateral = 10:1*Size:*mean diameter of 10.4 cmX-ray: √ lucent mass with rim of residual normal adrenal cortex√ calcifications (22%)US: √ heterogeneous predominantly hyperechoic (= fatty + myeloid tissue) mass with interspersed hypoechoic(= pure fat) regionsCT: √ large amounts of fat with interspersed "smoky" areas of higher attenuation of 20-30 HU (= admixture of fat + marrowlike elements)MR: √ hyperintense areas on T1WI (= predominantly fatty areas)√ intermediate intensity on T2WI similar to spleen√ hyperintense areas on fat-suppressed images(= marrowlike elements + hemorrhage) *Cx:*acute hemorrhage with increase in size (12%)*Dx:*percutaneous needle biopsy*Rx:*surgical excision not necessaryDDx:liposarcoma

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NEPHROGENIC ADENOMA
=uncommon benign metaplastic response to urothelial injury / prolonged irritation*Cause:*(a)trauma: accident, surgery, instrumentation, renal transplantation(b)irritation: calculi, chronic infection*Age:*3 weeks to 83 years; M:F = 3:1 (more common in females <20 years of age)*Path:*discrete raised papillary / polypoid areas projecting from epithelial surface*Histo:*variable number of small tubules + cysts + papillae lined with a single layer of cuboidal / low columnar cells ● hematuria, dysuria ● asymptomaticLocation:bladder (72%), renal pelvis, ureter, urethra; strong correlation between location + site of insult to urothelium√ filling defect*Rx:*resection / fulguration*DDx:*inflammatory / malignant urothelial lesions

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NEPHROGENIC DIABETES INSIPIDUS
=poor reabsorption of water in collecting ducts due to(1)lack of adequate vasopressin production(2)end-organ resistance to vasopressin*Cause:*(a)congenital1.X-linked recessive trait with variable expression2.Autosomal dominant form (rare)(b)acquired1.Obstructive uropathy2.Unilateral renal artery stenosis3.Acute tubular necrosis ● symptoms in infancy: ● vomiting secondary to hypernatremic dehydration ● mental retardation ● caloric growth failure (water favored over formula) ● symptoms after infancy: ● increased fluid intake ● avoiding urination √ bilateral hydroureteronephrosis*Rx:*thiazide diuretics, low-salt diet, encouragement of frequent micturition, indomethacin

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Notes:

NEUROBLASTOMA
Most common solid abdominal mass of infancy (12.3% of all perinatal neoplasms), 3rd most common malignant tumor in infancy (after leukemia + CNS tumors), 2nd most common tumor in childhood (Wilms tumor more common in older children), 7% of all childhood cancers; 15% of cancer deaths in children *Incidence:*1:7,100 to 1:10,000 livebirths; 500 cases per year in USA; 20% hereditary*Origin:*neural crest*Path:*round irregular lobulated mass of 50-150 g with areas of hemorrhage + necrosis*Histo:*small round cells slightly larger than lymphocytes with scant cytoplasm; Horner-Wright rosettes = one / two layers of primitive neuroblasts surrounding a central zone of tangled neurofibrillary processes*Age:*peak age at 2 years; 25% during 1st year;50% <2 years; 75% in <4 years; 90% in <8 years; occasionally present at birth; M:F = 1:1 *May be associated with:* aganglionosis of bowel, CHD ● pain + fever (30%) ● palpable abdominal mass (45-54%) ● bone pain, limp, inability to walk (20%) ● myoclonus of trunk + extremities ● cerebellar ataxia, nystagmus (20%) ● opsoclonus = spontaneous conjugate + chaotic eye movements (sign of cerebellar disease) ● orbital ecchymosis / proptosis (12%) ● intractable diarrhea (9%) due to increase in vasoactive intestinal polypeptides (VIP) ● increased catecholamine production (75-90%):in 95% excreted in urine as vanillylmandelic acid (VMA) / homovanillic acid (HVA) ● hypertension (up to 30%) ● acute cerebellar encephalopathy ● paroxysmal episodes of flushing, tachycardia, headaches, sweating ● rise in body temperature ● hyperglycemia*Stage:* Ilimited to organ of originIIregional spread not crossing midlineIIIextension across midlineIVmetastatic to distant lymph nodes, liver, bone, brain, lungIVsstages I + II with disease confined to liver, skin, bone marrow WITHOUT radiographic evidence of skeletal metastases *Metastases:* bone (60%), regional lymph nodes (42%), orbit (20%), liver (15%), intracranial (14%), lung (10%) ◊Metastases are first manifestation in up to 60%!**Hutchinson syndrome** (1) primary adrenal neuroblastoma (2) extensive skeletal metastases, particularly skull (3) proptosis (4) bone pain **Pepper syndrome** (1) primary adrenal neuroblastoma (2) massive hepatomegaly from metastases **Blueberry muffin syndrome** (1) primary adrenal neuroblastoma (2) multiple metastatic skin lesions ◊Bone marrow aspirate positive in 50-70% at time of initial diagnosis!◊2/3 of patients >2 years have disseminated disease!@Skeletal metastases:√ periosteal reaction√ osteolytic focus / multicentric lytic lesions√ lucent horizontal metaphyseal line√ vertical linear radiolucent streaks in metadiaphysis of long bones√ pathologic fracture√ vertebral collapse√ widened cranial sutures (subjacent dural metastases)√ sclerotic lesions with healing*DDx:*Ewing sarcoma, rhabdomyosarcoma, leukemia, lymphoma@Intracranial + maxillofacial metastases:Site:dura, brain substance@Pulmonary metastases:√ nodular infiltrates√ rib erosion√ mediastinal + retrocrural lymphadenopathy (common)Location:anywhere within sympathetic neural chain@abdomen(a) adrenal (36%): almost always unilateral (b) both adrenals (7-10%) (c) extraadrenal in sympathetic chain (18%) @thorax + posterior mediastinum (14%): aortic bodies@neck (5%): carotid ganglia@pelvis (5%): organ of Zuckerkandl@skull / esthesioneuroblastoma of olfactory bulb, cerebellum, cerebrum (2%)@other sites (10%): eg, intrarenal (very rare)@unknown (10%) √ large suprarenal mass with irregular shape + margins (82%)√ heterogeneous texture with low-density areas from hemorrhage + necrosis (55%)√ stippled / coarse calcifications (36-70%)√ "drooping lily" sign = displacement of kidney inferolaterally without distortion of collecting system√ hydronephrosis (24%)√ inseparable from kidney ± invasion of kidney (32%)√ propensity for extension into spinal canal through neural foramen with erosion of pedicles (15%)√ extension across midline (55%) (DDx: Wilms tumor)√ retroperitoneal adenopathy / contiguous extension (73%)√ retrocrural adenopathy (27%)√ encasement of IVC + aorta, celiac axis, SMA (32%)√ caval involvement = indicator of unresectability√ liver metastases (18-66%); invasion of liver (5%)Angio: √ hypo- / hypervascular massUS: √ hyper- / hypoechoic mass with acoustic shadowsNUC: √ focal uptake of I-131 / I-123 MIBG radioactivity (82% sensitivity; 88% specificity)√ tracer uptake on bone scan (60%)OB-US: ● maternal symptoms of catecholamine excess√ mixed cystic + solid mass in adrenal region√ may exhibit acoustic shadowing (calcifications)√ hydrops fetalis (severe anemia secondary to metastases to bone marrow, mechanical compression of IVC, hypersecretion of aldosterone) *2-year survival rate versus age at presentation:* 60%if patients age<1year20%if patients age1-2years10%if patients age>2years◊May revert to benign ganglioneuroma in 0.2%!*Survival rate versus stage:* 80%for stage I60%for stage II30%for stage III7%for stage IV 75-87%for stage IVs*DDx:*exophytic Wilms tumor, mesoblastic nephroma, multicystic kidney, retroperitoneal teratoma, adrenal hemorrhage, hepatic hamartoma / hemangioma, infradiaphragmatic sequestration

Notes:

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NEUROGENIC BLADDER
*Neuroanatomy:*bladder innervation of detrusor muscle by parasympathetic nerves S2-S4*Etiology:*congenital (myelomeningocele); trauma; neoplasm (spinal, CNS); infection (herpes, polio); inflammation (multiple sclerosis, syrinx); systemic disorder (diabetes, pernicious anemia)A.SPASTIC BLADDER"upper motor neuron" lesion above conus B.ATONIC BLADDER"lower motor neuron lesion" below conus

Notes:

ONCOCYTOMA
= PROXIMAL TUBULAR ADENOMA = BENIGN OXYPHILIC ADENOMA *Prevalence:*1-2-13% of renal tumors*Age:*median age around 65 (range of 26-94) years; M:F = 1.6:1 to 2.5:1*Path:*well-encapsulated tan-colored tumor of well-differentiated proximal tubular cells (benign adenoma) + oncocytes*Histo:*oncocytes = large epithelial cells with granular oxyphilic / eosinophilic cytoplasm (due to large number of mitochondria); no clear cytoplasm; similar oncocytic tumors seen in thyroid, parathyroid, salivary glands, adrenals ● majority asymptomatic, occasionally hypertension √ renal mass of 6-7.5 cm average size (0.1-26 cm) √ tumor of homogeneous low attenuation / hypoechogenicity (>50%) √ well-demarcated with pseudocapsule √ central stellate scar in 30% (in lesions >3 cm in diameter due to organization of central infarction + hemorrhage after tumor growth has outstripped blood supply) √ invasion of renal capsule / renal vein in large tumorsAngio: √ spoke-wheel configuration (80%), homogeneously dense parenchymal phase (71%) √ NO contrast puddling / arteriovenous shunting / renal vein invasionNUC: √ photopenic area (tubular cells do not function normally) on Tc-99m DMSA *Dx:*percutaneous needle biopsy unreliable Pathologic diagnosis requires entire tumor because well-differentiated renal cell carcinoma may have oncocytic features!*Rx:*local resection / heminephrectomy*Prognosis:*death from malignancy following surgery (3%)

Notes:

PAGE KIDNEY
=renin-angiotensin mediated hypertension caused by renal compression in a perinephric / subcapsular location*Etiology:*(1) Spontaneous hematoma (most common)(2) Blunt trauma (3) Cyst (4) Tumor √ stretching + splaying of intrarenal vessels√ slow arterial washout√ distortion of renal contour + thinning of renal parenchyma√ enlarged + displaced capsular artery

Notes:

PAPILLARY NECROSIS

=NECROTIZING PAPILLITIS = ischemic necrobiosis of medulla (loops of Henle + vasa recta) secondary to interstitial nephritis (interstitial edema) or intrinsic vascular obstruction*Cause: mnemonic:* "POSTCARD"**P**yelonephritis **O**bstructive uropathy **S**ickle cell disease **T**uberculosis, **T**rauma **C**irrhosis = alcoholism, **C**oagulopathy **A**nalgesic nephropathy **R**enal vein thrombosis **D**iabetes mellitus (50%) also:dehydration, severe infantile diarrhea, hemophilia, Christmas disease, acute tubular necrosis, transplant rejection, postpartum state, high-dose urography, intravesical instillation of formalin, thyroid cancer*Types:* 1.Necrosis in situ = necrotic papilla detaches but remains unextruded within its bed2.Medullary type (partial papillary slough) = single irregular cavity located concentric / eccentric in papilla with long axis paralleling the long axis of the papilla + communicating with calyx3.Papillary type (total papillary slough)*Phases:* (1)Enlargement of papilla (papillary swelling)(2)Fine projections of contrast material alongside papilla (tract formation)(3)Medullary cavitation / complete slough of papilla ● flank pain, dysuria, fever, chills ● ureteral colic ● acute oliguric renal failure ● hypertension ● proteinuria, pyuria, hematuria, leukocytosis Location:(a)localized / diffuse(b)bilateral distribution (systemic cause)(c)unilateral (obstruction, renal vein thrombosis, acute bacterial nephritis)√ normal or small kidney (analgesic nephropathy) / large kidney (acute fulminant)√ smooth / wavy renal contour (analgesic nephropathy)√ calcification of necrotic papilla: papillary / curvilinear / ringlikeIVP: √ subtle streak of contrast material extending from fornix parallel to long axis of papilla√ centric / eccentric, thin and short / bulbous cavitation of papilla√ widened fornix (necrotic shrinkage of papilla)√ ring shadow of papilla (outlining detached papilla within contrast material-filled cavity)√ club-shaped / saccular calyx (sloughed papilla)√ intraluminal nonopaque filling defect (sloughed papilla) in calyx / pelvis / ureter√ diminished density of contrast material in nephrogram; rarely increasingly dense√ wasted parenchymal thickness√ displaced collecting system (enlarged septal cortex from edema)US: √ multiple round / triangular cystic spaces in medulla with echo reflections of arcuate arteries at periphery of cystic spaces*Cx:*higher incidence of transitional cell carcinoma in analgesic abusers (8 x); higher incidence of squamous cell carcinoma*DDx:*(1)Postobstructive renal atrophy(2)Congenital megacalices (normal renal function)(3)Hydronephrosis (dilated infundibula)

Notes:

PARAGANGLIOMA
=rare neuroendocrine tumor arising from paraganglionic tissue found between base of skull and floor of pelvis; belong to amine-precursor-uptake decarboxylation (APUD) system characterized by cytoplasmic vesicles containing catecholamines *Types:* (1)Adrenal paraganglioma arising from adrenal medulla = pheochromocytoma(2)Aorticosympathetic paraganglioma associated with sympathetic chain + retroperitoneal ganglia(3)Parasympathetic paraganglioma including branchiomeric **chemodectoma**, vagal + visceral autonomic paraganglioma ● paroxysmal / permanent hypertension (due to secretion of vasopressor amines) with headache, pallor, perspiration, palpitations ● tumor may secrete catecholamine (= **functional paraganglioma**); proportion of hormonally active tumors high for pheochromocytomas, intermediate for aorticosympathetic paragangliomas, low for parasympathetic paragangliomas ● pheochromocytomas secrete norepinephrine + epinephrine, extraadrenal paragangliomas secrete only norepinephrine, some paragangliomas produce dopamine ● determination of free norepinephrine most sensitive with gas chromatography / high-pressure liquid chromatography (HPLC) performed on 24-hour urine specimensLocation of functioning paragangliomas: (a)adrenal medulla (>80%)(b)extraadrenal intraabdominal (8-16%)(c)extraadrenal in head, neck, chest (2-4%)(d)multiple paragangliomas in up to 20%, particularly in hereditary disorders (multiple endocrine neoplasia syndromes, neuroectodermal syndromes)*Cx:*malignant transformation in 2-10%

Notes:

PAROXYSMAL NOCTURNAL HEMOGLOBINURIA
=rare acquired disorder of nonmalignant bone marrow clones*Cause:*infection, transfusion, radiographic contrast material, exercise, drugs, immunization, surgery*Pathophysiology*:destruction of abnormally sensitive RBCs by activated complement; complement activation of abnormal platelets + release of thrombogenic material from lysed RBCs ● intravascular hemolysis: ● hemoglobinuria ● chronic iron deficiency anemia ● venous thrombosis: ● acute / chronic renal failure (small vessel thrombosis)MR: √ renal cortical iron deposition*Cx:*thrombosis due to hypercoagulable state (Budd-Chiari syndrome involving tertiary + secondary venous radicles, portal v., mesenteric v., splenic v.)

Notes:

PHEOCHROMOCYTOMA
=ADRENAL PARAGANGLIOMA= rare tumor of chromaffin tissue; responsible for 0.1% of hypertensions *Incidence:*0.13% in autopsy series; sporadic occurrence in 94%*Histo:*chromaffin tumor cells contain chromagranin within secretory granules, tumor tends to form "Zellballen" (cell balls)*Age:*5% in childhoodSymptomatology secondary to excess catecholamine production (norepinephrine / epinephrine): ● asymptomatic (9%) ● headaches, sweating, flushing, palpitations, anxiety, tremor ● nausea, vomiting, abdominal pain, chest pain ● paroxysmal (47%) / sustained (37%) hypertension(a) elevated catecholamine (b) functional renal vasoconstriction (c) renal artery stenosis (fibrosis, intimal proliferation, tumor encasement) ● hypoglycemia during hypertensive crisis ● elevated urine vanillylmandelic acid (VMA) in 54%;in up to 22% false-negative result because VMA not excreted*Associated with:* √ pheochromocytomas usually bilateral(1)Multiple endocrine neoplasia (MEN) in 6%: ● pheochromocytoma asymptomatic in 50%(a)Sipple syndrome = MEN type II (= type 2A)=medullary carcinoma of thyroid + parathyroid adenoma + pheochromocytoma(b)**Mucosal neuroma syndrome** = MEN type III(= type 2B) =medullary carcinoma of thyroid + intestinal ganglioneuromatosis + pheochromocytoma(2)Neuroectodermal disorder(a)tuberous sclerosis(b)von Hippel-Lindau disease(c)neurofibromatosis(3)Familial pheochromocytosis(4)**Carney syndrome** = paraganglioma + gastric epitheloid leiomyosarcoma + pulmonary chondroma*mnemonic:* "VEIN" **V**on Hippel-Lindau**E**ndocrine neoplasia (MEA 2)**I**nherited (congenital pheochromocytoma)**N**eurofibromatosisLocation:anywhere in sympathetic nervous system from neck to sacrum; subdiaphragmatic in 98%(a)adrenal medulla (85-90%)(b)extraadrenal (10-15% in adults, 31% in children):para-aortic sympathetic chain (8%), organ of Zuckerkandl at origin of inferior mesenteric artery (2-5%), gonads, urinary bladder (1%)Multiplicity:10% in nonfamilial adult cases32% in nonfamilial childhood cases65% in familial syndromesRULE OF TENS ("ten percent tumor"): **10**% bilateral / multiple**10**% extraadrenal**10**% malignant**10**% familial√ discrete round / oval mass with a mean size of 5 cm (range 3-12 cm)√ calcifications in 10%CT: localization accurate in 91% with tumor >2 cm in size; up to 40% in extraadrenal location are missed by CT; 93-100% sensitivity √ solid / cystic / complex mass with low-density areas secondary to hemorrhage / necrosis◊IV injection of iodinated contrast material may precipitate hypertensive crisis in patients not on alpha-adrenergic blockers!NUC:I-131 / I-123 MIBG (metaiodobenzylguanidine) scan (80-90% sensitivity; 98% specificity)Useful: (a)with clear clinical / laboratory evidence of tumor but no adrenal abnormality on CT / MRI(b)in detecting extraadrenal pheochromocytomas by whole-body scintigraphyUS: √ well-marginated purely solid (68%) / complex (16%) / cystic tumor (16%)√ homo- (46%) / heterogeneously (54%) solid tumor: isoechoic + hypoechoic (77%) / hyperechoic (23%) to renal parenchymaMRI: √ iso- / slightly hypointense to liver on T1WI√ extremely hyperintense on T2WI√ marked homo- / inhomogeneous enhancementAngio:intraarterial injection CONTRAINDICATED(induces hypertensive crisis) √ localization by aortography in >91%√ usually hypervascular lesion with intense tumor blush√ slow washout of contrast material√ enlarged feeding arteries + neovascularity ("spoke-wheel" pattern)√ parasitization from intrarenal perforating branches√ venous blood sampling (at different levels in IVC) *Cx:* malignancy in 2-14%; metastases (may be hormonally active) to bone, lymph nodes, liver, lung*Rx:*(1)Surgical removal curative(2)Alpha-adrenergic blocker (phenoxybenzamine / phentolamine)(3)Beta-adrenergic blocker (propranolol)(4)I-131 MIBG used to treat metastases*DDx:*nonfunctioning adrenal adenoma, adrenocortical carcinoma, adrenal cyst

Notes:

Autosomal Dominant Polycystic Kidney Disease =ADULT POLYCYSTIC KIDNEY DISEASE=Potter Type III=slowly progressive disease with nearly 100% penetrance and great variation in expressivity*Cause:*gene located on short arm of chromosome 16 (in 90%); spontaneous mutation in 10%*Incidence:*1:1,000 people carry the mutant gene; 3rd most prevalent cause of chronic renal failure*Risk of recurrence:*50%*Histo:*abnormal rate of tubule divisions (Potter Type III) with hypoplasia of portions of tubules left behind as the ureteral bud advances; cystic dilatation of Bowman capsule, loop of Henle, proximal convoluted tubule, coexisting with normal tissue*Mean age at diagnosis:* 43 years (neonatal / infantile onset has been reported); M:F = 1:1 Onset of cyst formation: -54% in 1st decade-72% in 2nd decade-86% in 3rd decademorphologic evidence in all patients by age 80 *Associated with:* (1)Cysts in: liver (25-50%), pancreas (9%); rare in lung, spleen, thyroid, ovaries, uterus, testis, seminal vesicles, epididymis, bladder(2)Aneurysm: saccular "berry" aneurysm of cerebral arteries (3-13%)(3)Mitral valve prolapse ● symptomatic at mean age of 35 years (cysts are growing with age) ● hypertension (50-70%) ● azotemia ● hematuria, proteinuria ● lumbar / abdominal pain√ bilaterally large kidneys with multifocal round lesions; unilateral enlargement may be the first manifestation of the disease√ cysts may calcify in curvilinear rim- / ringlike irregular amorphous fashion √ elongated + distorted + attenuated collecting system√ nodular puddling of contrast material on delayed images√ "Swiss cheese" nephrogram = multiple lesions of varying size with smooth margins√ polycystic kidneys shrink after beginning of renal failure, after renal transplantation, or on chronic hemodialysisNUC: poor renal function on Tc-99m DTPA scan √ multiple areas of diminished activity, cortical activity only in areas of functioning cortexUS: √ multiple cysts in cortical region (usually not seen prior to teens)√ diffusely echogenic when cysts small (children)√ renal contour poorly demarcatedOB-US: √ large echogenic kidneys similar to infantile PCKD (usually in 3rd trimester, earliest sonographic diagnosis at 14 weeks), can be unilateral√ macroscopic cysts (rare)√ normal amount of amniotic fluid / oligohydramnios (renal function usually not impaired) Atypical rare presentation: (a)unilateral adult PCKD(b)segmental adult PCKD(c)adult PCKD in utero / neonatal period *Cx:* (1)Death from uremia (59%) / cerebral hemorrhage (secondary to hypertension or ruptured aneurysm [13%]) / cardiac complications (mean age 50 years)(2)Renal calculi (3)Urinary tract infection(4)Cyst rupture(5)Hemorrhage(6)Renal cell carcinoma (increased risk)*DDx:* (1)Multiple simple cysts (less diffuse, no family history)(2)von Hippel-Lindau disease (cerebellar hemangioblastoma, retinal hemangiomas, occasionally pheochromocytomas)(3)Acquired uremic cystic disease (kidneys small, no renal function, transplant)(4)Infantile PCKD (usually microscopic cysts)

Notes:

Autosomal Recessive Polycystic Kidney Disease =INFANTILE POLYCYSTIC KIDNEY DISEASE=POLYCYSTIC DISEASE OF CHILDHOOD=Potter Type I*Incidence:*1: 6,000 to 1:50,000 livebirths; F > M; carrier frequency of 1:112*Path:* @kidney: abnormal proliferation + dilatation of collecting tubules resulting in multiple 1- to 2-mm cysts@liver: periportal fibrosis often with abnormal proliferation + dilatation of bile ducts@pancreas: pancreatic fibrosis A.ANTENATAL FORM (most common)90% of tubules show cystic changes ● onset of renal failure in utero ● Potter sequence√ oligohydramnios and dystocia (large abdominal mass)*Prognosis:*death from renal failure / respiratory insufficiency (pulmonary hypoplasia) within 24 hours in 75%, within 1 year in 93%; uniformly fatalB.NEONATAL FORM60% of tubules show ectasia + minimal hepatic fibrosis + bile duct proliferation ● onset of renal failure within 1st month of life*Prognosis:*death from renal failure / hypertension / left ventricular failure within 1st year of lifeC.INFANTILE FORM20% of renal tubules involved + mild / moderate periportal fibrosis ● disease appears by 3-6 months of age*Prognosis:*death from chronic renal failure / systemic arterial hypertension / portal hypertensionD.JUVENILE FORM10% of tubules involved + gross hepatic fibrosis + bile duct proliferation ● disease appears at 1-5 years of age*Prognosis:*death from portal hypertension◊The less severe the renal findings, the more severe the hepatic findings! @Lung√ severe pulmonary hypoplasia√ pneumothorax / pneumomediastinum@Liver ● portal venous hypertension√ tubular cystic dilatation of small intrahepatic bile ducts√ increase in liver echogenicity (from congenital hepatic fibrosis)@Kidneys√ bilateral gross renal enlargement√ faint nephrogram + blotchy opacification on initial images√ increasingly dense nephrogram√ poor visualization of collecting system√ "sunburst nephrogram" = striated nephrogram with persistent radiating opaque streaks (collecting ducts) on delayed images√ prominent fetal lobationCT: √ prolonged corticomedullary phaseUS: √ hyperechoic enlarged kidneys (unresolved 1- to 2-mm cystic / ectatic dilatation of renal tubules increase number of acoustic interfaces)√ increased renal through-transmission (high fluid content of cysts)√ loss of corticomedullary differentiation, poor visualization of renal sinus + renal borders√ occasionally discrete macroscopic cysts <1 cm√ compressed / minimally dilated collecting systemOB-US (diagnostic as early as 17 weeks GA): √ progressive renal enlargement with renal circumference:abdominal circumference ratio >0.30√ hyperechoic renal parenchyma√ nonvisualization of urine in fetal bladder (in severe cases)√ oligohydramnios (33%)√ small fetal thoraxOB management: (1)Chromosome studies to determine if other malformations present (eg, trisomy 13 / 18)(2)Option of pregnancy termination <24 weeks(3)Nonintervention for fetal distress >24 weeks if severe oligohydramnios present*Risk of recurrence:*25%*DDx:*Meckel-Gruber syndrome, adult polycystic kidney disease

Notes:

POSTERIOR URETHRAL VALVES
=congenital thick folds of mucous membrane located in posterior urethra (prostatic + membranous portion) distal to verumontanum Type I:(most common) mucosal folds (vestiges of Wolffian duct) extend anteroinferiorly from the caudal aspect of the verumontanum, often fusing anteriorly at a lower levelType II:(rare) mucosal folds extend anterosuperiorly from the verumontanum toward the bladder neck (nonobstructive normal variant, probably a consequence of bladder outlet obstruction)Type III:diaphragm-like membrane located below the verumontanum (= abnormal canalization of urogenital membrane)*Incidence:*1:5,000-8,000 boys; most common cause of urinary tract obstruction + leading cause of end-stage renal disease among boys*Time of discovery:*prenatal (8%), neonatal (34%), 1st year (32%), 2nd-16th year (23%), adult (3%) ● urinary tract infection (fever, vomiting) in 36% ● obstructive symptoms in 32% (hesitancy, straining, dribbling [20%], enuresis [20%]) ● palpable kidneys / bladder in neonate (21%) ● failure to thrive (13%) ● hematuria (5%)VCUG: √ vesicoureteral reflux, mainly on left side (<50%)√ fusiform distension + elongation of proximal posterior urethra persisting throughout voiding√ transverse / curvilinear filling defect in posterior urethra√ diminution of urethral caliber distal to severe obstruction√ hypertrophy of bladder neck√ trabeculation + sacculation of bladder wall√ large postvoid bladder residualUS: √ male gender√ oligohydramnios (related to severity + duration of obstruction)√ hypoplastic / multicystic dysplastic kidney (if early occurrence)√ bilateral hydroureteronephrosis (+ pulmonary hypoplasia)√ dilated renal pelvis may be absent in renal dysplasia / rupture of bladder / pelviureteral atresia√ overdistended urinary bladder (megacystis) in 30%√ thick-walled urinary bladder + trabeculations (best seen after decompression)√ urine leak: urinoma, urine ascites, urothorax√ posterior urethral dilatation (on perineal scan)√ dilated utricle (perineal scan)OB management: (1)Induction of labor as soon as fetal lung maturity established if diagnosed during last 10 weeks of pregnancy(2)Vesicoamniotic shunting may be contemplated if diagnosed remote from term (68% survivors) with good prognostic parameters of fetal urinary sodium <100 mEq/dL + chloride <90 mEq/dL + osmolality <210 mOsm/dL *Cx:*(1)Neonatal urine leak (ascites, urothorax, urinoma) in 13%(2)Neonatal pneumothorax / pneumomediastinum in 9%(3)Prune belly syndrome(4)Renal dysplasia (if obstruction occurs early during gestation)*Prognosis:*depends upon duration of obstruction prior to corrective surgery; poor prognosis if associated with vesicoureteral reflux; nephrectomy for irreversible damage (13%) *DDx:*(1) UPJ obstruction (2) UVJ obstruction (3) Primary megaureter (4) Massive vesicoureteral reflux (5) Megacystis-microcolon-intestinal hypoperistalsis syndrome

Notes:

POSTINFLAMMATORY RENAL ATROPHY
=acute bacterial nephritis with irreversible ischemia as an unusual form of severe Gram-negative bacterial infection in patients with altered host resistance in spite of proper antibiotic treatment*Histo:*occlusion of interlobar arteries / vasospasm√ small smooth kidney√ papillary necrosis in acute phase

Notes:

POSTOBSTRUCTIVE RENAL ATROPHY

= generalized papillary atrophy usually following successful surgical correction of urinary tract obstruction and progressing in spite of relief of obstruction√ small smooth kidney, usually unilateral √ dilated calices with effaced papillae√ thinned cortex

Notes:

PRIAPISM
=prolonged penile erection not associated with sexual arousal*Types:* (1)Low-flow form = veno-occlusive form (common)characterized by ischemia, venous stasis, pooling of blood within corpora cavernosa Cause:sickle cell disease, hematopoietic malignancy, hypercoagulable state ● painful erection√ sluggish intracavernosal flow √ decreased venous outflow√ decreased arterial inflow√ intracavernosal thrombosis*Rx:*cavernosal aspiration + irrigation, anticoagulation, shunt procedure*Cx:*impotence (in 50% in spite of Rx)(2)High-flow form (rare)characterized by unregulated arterial inflow of blood into corpora cavernosa usually due to arterial injury *Cause:*perineal / penile trauma ● subsequent persistent painless erectionColor Doppler US: √ focal blush of abnormal intracavernosal flow adjacent to cavernosal artery from arterial-sinusoidal fistula*Rx:*percutaneous transcatheter embolization; arterial ligation

Notes:

PROSTATE CANCER

Incidence: 8.7% in White males, 9.4% in Black males, increasing with age; less common in Asian population; 200,000 new cases in USA (1994); 2nd most common malignancy in males (after lung cancer); in 35% of men >45 years of age (autopsies) ◊One out of 11 males will develop prostate cancer!*Risk factors:*advancing age, presence of testes, cadmium exposure, animal fat intake*Histo:* nuclear anaplasia + large nucleoli in secretory cells, disturbed architecture, invasive growth Premalignant change: (1)Prostatic intraepithelial neoplasia (PIN)= premalignant lesion frequently associated with invasive carcinoma next to it / elsewhere in the gland (2)Atypical adenomatous hyperplasia = proliferation of newly formed small acini*Grading* (Gleason score 2-10): 1,2,3glands surrounded by 1 row of epithelial cells4absence of complete gland formation5sheets of malignant cellslow numbers refer to well-differentiated, high numbers to anaplastic tumors; primary predominant grade (1-5) is added to secondary less representative area with highest degree of dedifferentiation (1-5) ◊Gleason grading is in only 80% reproducible! ● Categories:1.Latent carcinoma = usually discovered at autopsy of a patient without signs or symptoms referable to the prostate (26-73%)2.Incidental carcinoma = discovered in 6-20% of specimens obtained during transurethral resection for clinically benign prostatic hyperplasia3.Occult carcinoma = found at biopsy of metastatically involved bone lesion / lymph node in a patient without symptoms of prostatic disease4.Clinical carcinoma = cancer detected by digital rectal examination based on induration / irregularity / nodule ● **Prostate-specific antigen** (PSA = glycoprotein produced by prostatic epithelium) may be elevated (a)monoclonal radioimmunoassay (Hybritech®); most commonly used: normal value of 0.1-4 ng/mL◊Cancers with PSA levels of <10 ng/mL are usually confined to gland!◊Cancers of <1 mL usually do not elevate PSA!◊19% of prostate cancers have normal PSA!◊16% of normal men have PSA >4 ng/mL◊Benign conditions with PSA elevation: benign prostatic hypertrophy, prostatitis, prostatic intraepithelial neoplasia(b)polyclonal radioimmunoassay (Proscheck®, Abbott PSA®)(c)enzyme-linked immunosorbent assay PSA density = volume corrected PSA level [= prostate volume (height x width x length x 0.523) / Hybritech® PSA value]: >0.12 (90% sensitive, 51% specific for cancer) ◊ Each gram of malignant prostate tissue results in about 10 times as much PSA in the serum as its benign counterpart! PSA "velocity" = serial PSA evaluation ◊If annual rate of PSA increase is >20% / >0.75 ng/ mL, the chances of cancer increase sharply! *Staging* (American Urological Association System, modified Jewitt-Whitmore staging system): ANo palpable lesionA_1focal well-differentiated tumor <1.5 cmA_2diffuse poorly differentiated tumor; >5% of chips from transurethral resection contain cancerBPalpable tumor confined to prostateB_1lesion <1.5 cm in diameter confined to one lobeB_2tumor ≥1.5 cm / involving more than one lobeCLocalized tumor with capsular involvementC_1capsular invasionC_2capsular penetrationC_3seminal vesicle involvementDDistant metastasisD_1involvement of pelvic lymph nodesD_2distant nodes involvedD_3metastases to bone / soft tissues / organs◊At initial presentation >75% have stage C + D!◊Escape routes through prostatic capsule are: (1) apex, (2) capsular margin at neurovascular bundle posterolaterally, (3) seminal vesicles!*Staging* (American Joint Committee on Cancer): T0No evidence of primary tumorT1Clinically inapparent nonpalpable nonvisible tumorT1a<3 microscopic foci of cancer / <5% of resected tissueT1b>3 microscopic foci of cancer / <5% of resected tissueT1ctumor identified by needle biopsyT2Tumor clinically present + confined to prostateT2atumor ≤1.5 cm, normal tissue on 3 sidesT2btumor >1.5 cm / in >1 lobeT2ctumor involves both lobesT3Extension through prostatic capsuleT3aunilateral extracapsular extensionT3bbilateral extracapsular extensionT3cinvasion of seminal vesiclesT4Tumor fixed / invading adjacent structures other than seminal vesiclesT4ainvasion of bladder neck, external sphincter, rectumT4binvasion of levator anus muscle and/or fixed to pelvic wallNInvolvement of regional lymph nodesN1metastasis in a single node ≤2 cmN2metastasis in a single node >2 and <5 cm / multiple lymph nodes affectedN3metastasis in a lymph node ≥5 cmMDistant metastasisM1anonregional lymph nodesM1bboneM1cother siteStaging accuracy for local / advanced disease: 46 / 66% for US, 57 / 77% for MR ◊Extracapsular disease is common at a tumor volume of >3.8 cm^3! Metastases to lymph nodes: 0% in stage A_1, 3-7% in stage A_2, 5% in stage B_1, 10-12% in stage B_2, 54-57% in stage C; 10% with Gleason grade ≤5, 70-93% with Gleason grade 9 / 10 Location:peripheral zone (70%), transition zone (20%), central zone (10%) US (21% positive predictive value): √ hypoechoic (61%) / mixed (2%) / hyperechoic (2%) lesion; not detectable isoechoic lesion (35%)√ asymmetric enlargement of gland√ deformed contour of prostate = irregular bulge sign (75% PPV)√ heterogeneous textureSize versus rate of detection: ≤5 mm (36%), 6-10 mm (65%), 11-15 mm (53%), 16-20 mm (84%), 21-25 mm (92%), ≥26 mm (75%) *DDx of hypoechoic lesion:* external sphincter, veins, neurovascular bundle, seminal vesicle, dilated duct, small prostatic cyst, acute prostatitis, benign prostatic hyperplasia, dysplasia, sonographic artifact MR: √ extracapsular extension (90% specific, 15% sensitive):√ obliteration of rectoprostatic angle√ asymmetry of neurovascular bundle*Prognosis:*increase in tumor volume increases probability of capsular penetration, metastasis, histologic dedifferentiation*Mortality:*2.6% for White males, 4.5% for Black males; 34,000 deaths/1992*Screening recommendation* (American Urological Association, American Cancer Society): PSA level measurements + digital rectal exam annually *Rx:*(1)Watchful waiting(2)Radical prostatectomy for disease confined to capsule + life expectancy >15 years(3)Radiation therapy for(a)disease confined to capsule, life expectancy <15 years(b)disease outside capsule, no spread(4)Hormonal therapy (orchiectomy, diethylstilbestrol, leuprolide acetate) for widely metastatic disease(5)Cryosurgery(6)Chemotherapy

Notes:

PRUNE BELLY SYNDROME
=EAGLE-BARRETT SYNDROME=congenital nonhereditary multisystem disorder; almost exclusively in malesTRIAD:1.Absent / markedly hypoplastic abdominal wall musculature ("prune belly")2.Nonobstructed markedly distended redundant ureters ± hydronephrosis and variable degree of renal dysplasia3.Undescended testes (cryptorchidism)*Etiology:* (1)primary mesodermal defect at 7-10 weeks GA(2)massive abdominal distension secondary to massive ureteral dilatation / urine ascites / intestinal perforation with ascites / cystic abdominal masses / megacystis-microcolon-intestinal hypoperistalsis syndrome causes pressure atrophy of abdominal wall muscles; bladder distension interferes with descent of testes *Incidence:*1:35,000 to 1:50,000 livebirths; almost exclusively in males*Groups:* (1)Obstruction of urethra (most commonly urethral atresia)*Associated with:* malrotation (most common anomaly), intestinal atresia, imperforate anus, skeletal abnormalities (meningomyelocele, scoliosis, pectus carinatum /excavatum, arthrogryposis, clubfoot, dislocation of hip, lower limb hemimelia, sacral agenesis, polydactyly), CHD (VSD, pulmonary artery stenosis), Hirschsprung disease, congenital cystic adenomatoid malformation of lung √ bladder wall hypertrophy*Prognosis:*in 20% death within 1 month; in 30% death within 2 years(2)Functional abnormality of bladder emptying (more common) no associated abnormalities√ large floppy urinary bladder√ large urachal remnant*Prognosis:*chronic urinary tract problems ● wrinkled flaccid appearance of hypotonic abdominal wall with bulging flanks (agenesis / hypoplasia of muscles in lower + medial parts of abdominal wall) ● bilateral cryptorchidism ● ± impaired renal function@Bladder√ large distended urinary bladder with bizarre contours√ intramural bladder calcifications√ persistence of urachal remnant ± calcification√ patent bladder neck@Urethra√ elongated + dilated prostatic urethra (absence of prostate)√ dilated prostatic utricle (= small epithelium-lined diverticulum representing the remnant of the fused caudal ends of the müllerian ducts)√ urethral obstruction (stenosis / atresia / dorsal chordae / posterior urethral valves)√ megalourethra(a)complete / fusiform megalourethra (rare)=complete absence / marked deficiency of corpora cavernosa + corpus spongiosum(b)incomplete / scaphoid megalourethra (common)=congenital absence / deficiency of corpus spongiosum@Ureters√ massively dilated tortuous laterally placed ureters√ alternating narrowed + dilated ureteral segments√ vesicoureteral reflux@Kidneys√ asymmetry of renal size + lobulated contours√ no / mild hydronephrosis√ caliceal dilatation ± diverticula√ renal calcifications√ renal dysplasia with cystic dysplastic changes oligohydramnios, pulmonary hypoplasia (in severe cases)*Cx:*respiratory infections (ineffective cough)

Notes:

PYELOCALICEAL DIVERTICULUM
=PYELOGENIC CYST = PERICALICEAL CYST= CALICEAL DIVERTICULUM=uroepithelium-lined pouch extending from a peripheral point of the collecting system into adjacent renal parenchymaTYPE I (calyx): more common; connected to caliceal cup, usually at fornix; bulbous shape; narrow connecting infundibulum of varying length; few millimeters in diameter; in polar region especially upper pole TYPE II (pelvis): interpolar region; communicates directly with pelvis; usually larger and rounder; neck short and not easily identified *Cause:* (1)Developmental origin from ureteral bud remnant (obstruction of peripheral aberrant "minicalyx")(2)Acquired: reflux, infection, rupture of simple cyst / abscess, infundibular achalasia / spasm, hydrocalyx secondary to inflammatory fibrosis of an infundibulum√ formation of single / multiple stones (50%) or milk of calcium (fluid-calcium level)√ opacification may be delayed and remain so forprolonged period√ mass effect on adjacent pelvicaliceal system if large enough*Cx:*recurrent infection*DDx:*ruptured simple nephrogenic cyst, evacuated abscess / hematoma, renal papillary necrosis, medullary sponge kidney, hydrocalyx due to infundibular narrowing from TB / crossing vessel / stone / infiltrating carcinoma

Notes:

PYELONEPHRITIS
=upper urinary tract infection with pelvic + caliceal + parenchymal inflammation Society of Uroradiology recommends to eliminate the terms (acute focal) bacterial nephritis, lobar nephritis, lobar nephronia, preabscess, renal cellulitis, renal phlegmon, renal carbuncle!

Acute Pyelonephritis Emphysematous Pyelitis Emphysematous Pyelonephritis Xanthogranulomatous Pyelonephritis

Notes:

Acute Pyelonephritis =episodic bacterial infection of kidney with acute inflammation, usually involving pyelocaliceal lining + renal parenchyma centrifugally along medullary rays*Etiology:*infected urine from lower tract during adulthood; in 5% anatomic abnormality (obstruction, stone, stasis); (DDx: chronic atrophic pyelonephritis secondary to vesicoureteral reflux in infancy)*Pathway of infection:* (a)ascending bacterial infection usually due to P-fimbriated E. coli (fimbriae facilitate adherence to mucosal surface): initial colonization of ureter in areas of turbulent flow leads to paralysis of ureteral smooth muscle function with dilatation + functional obstruction of collecting system(b)vesicoureteral reflux + pyelotubular backflow: P-fimbriated E. coli not necessary for infection(c)hematogenous spread (12-20%) with Gram-positive cocci*Path:*radiating yellow-white stripes / wedges extending from papillary tip to cortical surface in a patchy distribution + sharply demarcated from adjacent spared parenchyma by 48-72 hours*Histo:*tubulointerstitial nephritis = leukocytic migration from interstitium into lumen of tubules with destruction of tubule cells by released enzymes, bacterial invasion of interstitium by 48-72 hours *Organism:*E. coli > Proteus > Klebsiella, Enterobacter, Pseudomonas*Age:*any; M << F*Prevalence:*1-2% of all pregnant women ● fever, chills, flank pain + tenderness ● leukocytosis ● pyuria, bacteriuria, positive urine culture ● ± microscopic hematuria / bacteremia *Indication for imaging:* (1) diabetes (2) analgesic abuse (3) neuropathic bladder (4) history of urinary tract stones (5) atypical organism (6) poor response to antibiotics (7) frequent recurrences √ normal urogram in 75%!√ smooth normal / enlarged kidney(s), focal >> diffuse involvement of kidney√ delayed opacification of collecting system√ compression of collecting system (edema)√ nonobstructive ureteral dilatation (rare, effect of endotoxins)√ immediate persistent dense nephrogram, rarely striated√ diminished nephrographic density (global / wedge-shaped / patchy)√ nonvisualization of kidney (in severe pyelonephritis, rare)√ "tree-barking" = mucosal striations (rare)CT: √ area of high attenuation on unenhanced scan (= hemorrhagic bacterial nephritis)√ thickening of Gerota fascia + thickened bridging septa / stranding (= perinephric inflammation)√ generalized renal enlargement / focal swelling√ obliteration of renal sinus√ caliceal effacement√ thickening of walls of renal pelvis + calices √ mild dilatation of renal pelvis + ureter√ soft-tissue filling defect in collecting system (= papillary necrosis, inflammatory debris, blood clot)CECT: √ hypoattenuating wedge-shaped area of cortex extending from papilla to renal capsule during nephrographic phase (= lobar segments of hypoperfusion + edema)√ poor corticomedullary differentiation √ streaky linear bands of alternating hyper- and hypoattenuation parallel to axis of tubules + collecting ducts during excretory phase (diminished concentration of contrast material in tubules from ischemia + tubular obstruction by inflammatory cells + debris)√ persistent enhancement on delayed scans in area of earlier diminished enhancement √ contrast material staining of parenchyma on 3-6 hours delayed scan (= functioning renal parenchyma)US: √ majority of kidneys appear normal√ swollen kidney with decreased echogenicity√ loss of central sinus echoes√ wedge-shaped hypo- / isoechoic zones, rarely hyperechoic (due to hemorrhage)√ thickened sonolucent corticomedullary bands√ blurred corticomedullary junctions√ localized increase in size + echogenicity of perinephric fat ± fat within renal sinus√ localized perinephric exudate√ thickening of wall of renal pelvisMR: √ wedge-shaped foci of high signal intensity on contrast-enhanced fast multiplanar IR imagesRenal cortical scintigraphy (Tc-99m DMSA): √ focal areas of diminished uptake (in 90%)*Prognosis:* (1)Quick response to antibiotic treatment will leave no scars(2)Delayed treatment of acute pyelonephritis during first 3 years of life can severely affect renal function later in life: decreased renal function, hypertension (33%), end-stage renal disease (10%)*Cx:*(1)Renal abscess (near-water density lesion without enhancement)(2)Scarring of affected renal lobes often in children + in up to 43% in adults(3)Maternal septic shock (3%)(4)Premature labor (17%)

Notes:

Emphysematous Pyelitis =gas confined to renal pelvis + calices*Organism:*E. coli*Predisposed:*diabetes mellitus (50%); M:F = 1:3*May be associated with:* emphysematous cystitis (rare) ● pyuria√ gas pyelogram outlining pelvicaliceal system√ dilated renal collecting system (frequent)√ ± gas in ureters*DDx:*reflux of gas / air from bladder or urinary diversion

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Notes:

Emphysematous Pyelonephritis =life-threatening acute fulminant necrotizing infection of kidney and perirenal tissues associated with gas formation*Organism:*E. coli (68%), Klebsiella pneumoniae (9%), Proteus mirabilis, Pseudomonas, Enterobacter, Candida, Clostridia (exceptionally rare)*Path:*acute and chronic necrotizing pyelonephritis with multiple cortical abscesses*Mechanism:*pyelonephritis leads to ischemia + low O_2 tension with anaerobic metabolism; facultative anaerobe organisms form CO_2 with fermentation of necrotic tissue / tissue glucose*Predisposed:*immunocompromised patients, esp. diabetics (in 87-97% of cases); ureteral obstruction (in 20-40%)*Average age:*54 years; M:F = 1:2*May be associated with:*XGP ● features of acute severe pyelonephritis (chills, fever, flank pain, lethargy, confusion) not responding to Rx ● positive blood + urine cultures (in majority) ● urosepsis, shock ● fever of unknown origin + NO localizing signs in 18% ● multiple associated medical problems: uncontrolled hyperglycemia, acidosis, dehydration, electrolyte imbalanceLocation:in 5-7% bilateralType I (33%): √ streaky / mottled gas in interstitium of renal parenchyma radiating from medulla to cortex√ crescent of subcapsular / perinephric gas√ NO fluid collection (= no effective immune response)*Prognosis:* 69% mortality Type II (66%): √ bubbly / loculated intrarenal gas (infers presence of abscess)√ renal / perirenal fluid collection√ gas within collecting system (85%)*Prognosis:* 18% mortality√ parenchymal destruction√ absent / decreased contrast excretion (due to compromised renal function)US: √ high-amplitude echoes within renal sinus / renal parenchyma associated with "dirty" shadowing / "comet tail" reverberationsCAVE:(1)kidney may be completely obscured by large amount of gas in perinephric space (DDx: surrounding bowel gas)(2)gas may be confused with renal calculiCT (most reliable + sensitive modality): √ mottled areas of low attenuation extending radially along the pyramids√ extensive involvement of kidney + perinephric space√ air extending through Gerotas fascia into retroperitoneal space√ occasionally gas in renal veinsMR: √ signal void on T1WI + T2WI (DDx: renal calculi, rapidly flowing blood)*Mortality:*60-75% under antibiotic Rx;21-29% after antibiotic Rx + nephrectomy;80% with extension into perirenal space*Rx:*antibiotic therapy + nephrectomy; drainage procedure with coexisting obstruction*DDx:*emphysematous pyelitis (gas in collecting system but not in parenchyma, diabetes in 50%, less grave prognosis)

Notes:

Xanthogranulomatous Pyelonephritis =chronic suppurative granulomatous infection in chronic obstruction (calculus, stricture, carcinoma) originating in medulla*Incidence:*681,000 surgically proven cases of chronic pyelonephritis*Organism:*Proteus mirabilis, E. coli, S. aureus*Path:*replacement of corticomedullary junction with soft yellow nodules; calices filled with pus and debris*Histo:*diffuse infiltration by plasma cells + histiocytes + lipid-laden macrophages (xanthoma cells)*Peak age:*45-65 years; all ages affected, may occur in infants; M:F = 1:3-1:4 ● pyuria(95%) ● flank pain(80%) ● fever(70%) ● palpable mass(50%) ● weight loss(50%) ● microscopic hematuria(50%) ● reversible hepatic dysfunction with elevated liver function tests (50%)◊Symptomatic for 6 months prior to diagnosis in 40%! A.DIFFUSE XGP (83-90%)B.SEGMENTAL / FOCAL XGP (10-17%)=tumefactive form due to obstructed single infundibulum / one moiety of duplex system*DDx:*renal cell carcinoma √ kidney globally enlarged (smooth contour uncommon) / focal renal mass√ contracted pelvis with dilated calices√ totally absent / focally absent nephrogram√ central obstructing calculus: staghorn calculus in 75%√ extension of inflammation into perirenal space, pararenal space, ipsilateral psoas muscle, colon, spleen, diaphragm, posterior abdominal wall, skinRetrograde: √ complete obstruction at ureteropelvic junction / infundibulum / proximal ureter√ contracted renal pelvis, dilated deformed calices + nodular filling defects√ irregular parenchymal masses with cavitationCT: √ low attenuation masses replacing renal parenchymaUS: √ hypoechoic dilated calices with echogenic rim√ hypoechoic masses frequently with low-level internal echoes replacing renal parenchyma√ loss of corticomedullary junction√ parenchymal calcifications are uncommonAngio: √ stretching of segmental / interlobar arteries around large avascular masses√ hypervascularity / blush around periphery of masses in late arterial phase (= granulation tissue)√ venous encasement + occlusion*DDx:*hydronephrosis, avascular tumor*Rx:*nephrectomy

Notes:

PYELOURETERITIS CYSTICA
=hyperplastic transitional epithelial cell collections projecting into ureteral lumen◊Indicative of past / present urinary tract infection!*Cause:*chronic urinary tract irritant (stone / infection)*Histo:*numerous small submucosal epithelial-lined cysts representing cystic degeneration of epithelial cell nests within lamina propria (cell nests of von Brunn) formed by downward proliferation of buds of surface epithelium that have become detached from the mucosa *Organism:*E. coli > M. tuberculosis, Enterococcus, Proteus, schistosomiasis*Predisposed:*diabetics*Age:*6th decade; more prevalent in women ● no specific symptoms; ± hematuriaLocation:bladder >> proximal 1/3 of ureter > ureteropelvic junction; unilateral >> bilateral√ multiple small round smooth lucent defects of 1-3 mm in size; scattered discrete / clustered√ persist unchanged for years in spite of antibiotic therapy*Cx:*increased incidence of transitional cell carcinoma*DDx:*(1)Spreading / multifocal TCC(2)Vascular ureteral notching(3)Multiple blood clots(4)Multiple polyps(5)Allergic urticaria of mucosa(6)Submucosal hemorrhage (eg, anticoagulation)

Notes:

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PYONEPHROSIS
=presence of pus in dilated collecting system secondary to infected hydronephrosis*Path:*purulent exudate composed of sloughed urothelium + inflammatory cells from early formation of microabscesses + necrotizing papillitis*Organism:*most commonly E. coliUS: √ dispersed / dependent internal echoes within dilated pelvicaliceal system√ shifting urine-debris level√ dense peripheral echoes in nondependent location + shadowing (gas from infection)*Cx:*1.XGP2.Renal abscess3.Perinephric abscess4.Fistula to duodenum, colon, pleura

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Notes:

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RADIATION NEPHRITIS

*Histo:*interstitial fibrosis, tubule atrophy, glomerular sclerosis, sclerosis of arteries of all sizes, hyalinization of afferent arterioles, thickening of renal capsuleThreshold dose:2,300 rads over 5 weeks ● clinically resembling chronic glomerulonephritis√ normal / small smooth kidney consistent with radiation field√ parenchymal thickness diminished (globally / focally;related to radiation field)√ diminished nephrographic density

Notes:

REFLUX ATROPHY

*Cause:*increased hydrostatic pressure of pelvicaliceal urine with atrophy of nephrons secondary to long-standing vesicoureteral reflux√ small smooth kidney with loss of parenchymal thickness√ widened collecting system with effaced papillae√ longitudinal striations from redundant mucosa when collecting system is collapsed◊Do NOT confuse with reflux nephropathy!

Notes:

REFLUX NEPHROPATHY

=CHRONIC ATROPHIC PYELONEPHRITIS = ascending bacterial urinary tract infection secondary to reflux of infected urine from lower tract + tubulointerstitial inflammation in childhood (hardly ever endangers adult kidney); most common cause of small scarred kidney*Etiology:*3 essential elements:(1) Infected urine (2) Vesicoureteral reflux (3) Intrarenal reflux *Age:*usually young adults (subclinical diagnosis starting in childhood); M < F ● fever, flank pain, frequency, dysuria ● hypertension, renal failure ● may have no history of significant symptomsSite:predominantly affecting poles of kidneys secondary to presence of compound calyces having distorted papillary ducts of Bellini (= papillae with gaping openings instead of slitlike openings of interpolar papillae)√ normal / small kidney; uni- / bilateral; uni- / multifocal√ focal parenchymal thinning with contour depression in upper / lower pole (more compound papillae in upper pole), scar formation only up to age 4 √ retracted papilla with clubbed calyx subjacent to scar√ contralateral / focal compensatory hypertrophy (= pseudotumor)√ dilated ureters (secondary to reflux) sometimes with linear striations (redundant / edematous mucosa)US: √ focally increased echogenicity within cortex (scar)Angio: √ small tortuous intrarenal arteries, pruning of intrarenal vessels√ vascular stenoses, occlusion, aneurysms√ inhomogeneous nephrographic phaseNUC (Tc-99m glucoheptonate / DMSA with SPECT most sensitive method): √ focal / multifocal photon-deficient areas*Cx:*1.Hypertension2.Obstetric complications3.Renal failure

Notes:

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RENAL / PERIRENAL ABSCESS
=usually complication of renal inflammation with liquefactive necrosis; 2% of all renal masses*Pathway of infection:* (a)ascending (80%): associated with obstruction (UPJ, ureter, calculus)(b)hematogenous (20%): infection from skin, teeth, lung, tonsils (S. aureus), endocarditis, intravenous drug abuse*Organism:*E. coli, Proteus*Predisposed:*diabetics (twice as frequent compared with nondiabetics) ● positive urine culture in 33% ● positive blood culture in 50% ● pyuria, hematuria (absent if abscess isolated within parenchyma)

Renal Abscess Carbuncle Perinephric Abscess

Notes:

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Renal Abscess ● may have negative urine analysis / culture (in up to 20%)IVP: √ focal mass displacing collecting systemCT: √ hypoattenuating focal renal mass with thick irregular enhancing wall / pseudocapsule√ ± presence of gas√ thickened septa + Gerota fascia√ perinephric fat obliteration US: √ slightly hypoechoic (early), hypo- to anechoic (late) mass with irregular margins + increased through-transmission ± septations ± microbubbles of gasNUC (Ga-67 citrate / In-111 leukocytes): √ hot spot*DDx:* cystic renal cell carcinoma

Notes:

Carbuncle =multiple coalescent intrarenal abscesses Term should not be used in radiology reports!

Notes:

Perinephric Abscess *Cause:*acute pyelonephritis / extension of renal abscess through capsule*Predisposed:*diabetics (in 30%), urolithiasis, septic emboli◊14-75% of patients with perinephric abscess have diabetes mellitus!√ loss of psoas margin / obscuration of renal contour√ renal displacement√ focal renal mass√ scoliosis concave to involved side√ respiratory immobility of kidney = renal fixation√ occasionally gas in renal fossa√ unilateral impaired excretion√ pleural effusion

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Notes:

RENAL ADENOMA

◊Small adenoma <3 cm should be considered a renal cell carcinoma of low metastatic potential = borderline renal cell carcinoma!*Incidence:*in 7-15-23% of adults (autopsies); most common cortical lesion; increasing with age (in 10% of patients >80 years of age); increased frequency in tobacco users + patients on long-term dialysis*Age:*usually >30 years; M:F = 3:1Types: (1)Papillary / cystadenoma (38%)(2)Tubular adenoma (38%)(3)Mixed type adenoma (21%)(4)Alveolar adenoma (3%) = precursor of RCC√ solitary in 75%, multiple in 25%√ usually <3 cm in size; subcapsular cortical location√ impossible to differentiate from renal cell carcinoma*Cx:*premalignant / potentially malignant*Prognosis:*average growth rate of 0.4 (range, 0.2-3.5) cm/year; tumors growing <0.25 cm/year rarely metastasize; tumors growing >0.6 cm/year frequently metastasize

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Notes:

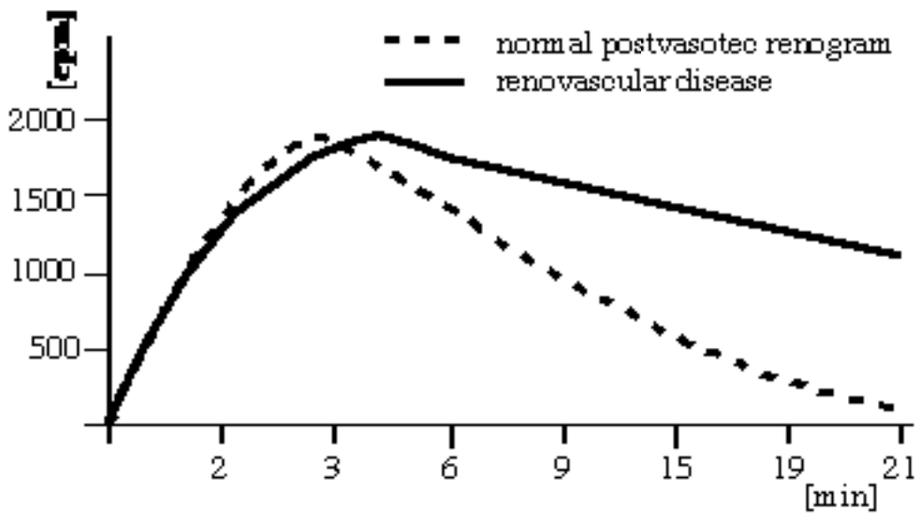
RENAL AGENESIS

Mechanism: (a)formation failure=failure of ureteral bud to form ● hemitrigone = absence of ipsilateral trigone + ureteral orifice(b)induction failure=failure of growing ureteral bud to induce metanephric tissue ● blind-ending ureter A.UNILATERAL RENAL AGENESIS *Incidence:*1:600-1,000 pregnancies; M:F = 1.8:1*Risk of recurrence:*4.5%*Often coexisting with other anomalies:* 1.Genital abnormalities:(a)in male (10-15%): hypoplasia or agenesis of testis / vas deferens, seminal vesicle cyst (Zinner syndrome)(b)in female (25-50%): unicornuate / bicornuate / hypoplastic / absent uterus, absent / aplastic vagina2.Turner syndrome, trisomy, Fanconi anemia, Laurence-Moon-Biedl syndromeLocation:L > R√ visualization of single kidney (DDx: additional kidney in ectopic location)√ absent adrenal gland (11%)√ absent / rudimentary renal vessels√ colon occupies renal fossa√ compensatory contralateral renal hypertrophy (50%) B. BILATERAL RENAL AGENESIS (= Potter syndrome) *Incidence:*1:3,000 to 1:10,000 pregnancies; M:F = 2.5:1*Risk of recurrence:*<1% ● Potters facies = low-set ears, redundant skin, parrot-beaked nose, receding chin◊US-sensitivity is ONLY 69-73% due to decreased visualization from oligohydramnios + discoid-shaped adrenal glands simulating kidneys!√ severe oligohydramnios (after 14 weeks MA)√ bilateral absence of kidneys (after 12 weeks), ureters, renal arteries√ inability to visualize renal arteries by color duplex√ flattened discoid shape of adrenals (due to absence of pressure by kidney)√ inability to visualize urine in fetal bladder (after 13 weeks) = bladder agenesis / hypoplasia; negative furosemide test (20-60 mg IV) not diagnostic (fetuses with severe IUGR may not be capable of diuresis)√ bell-shaped thorax (pulmonary hypoplasia) in mid to late 3rd trimester√ compression deformities of extremities = clubfoot, flexion contractures, joint dislocations (eg, hip)*Prognosis:*stillbirths (24-38%); invariably fatal in the first days of life (pulmonary hypoplasia)*DDx:*functional cause of in utero renal failure (eg, severe IUGR) **Potter Sequence** =hypoplasia of lungs, bowing of legs, broad hands, loose skin, growth retardation associated with long-standing severe oligohydramnios*Cause:*renal agenesis, urethral obstruction, prolonged rupture of membranes, severe IUGR

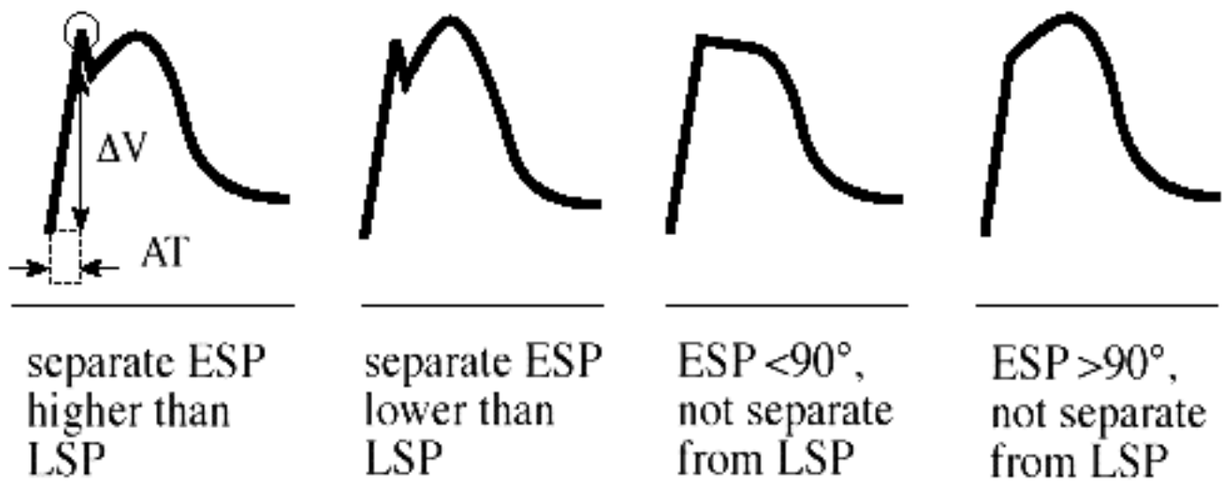
Notes:

RENAL ARTERY STENOSIS

*Prevalence:*1-2% of hypertensive individuals; 4.3 % of autopsies; 10% of hypertensive individuals with coronary artery disease; 25% of patients with hypertension that is difficult to control; in 45% of patients with malignant hypertension; in 45% of patients with peripheral vascular diseaseHemodynamic significance determined by: (a)elevated renin levels in ipsilateral renal vein ≥1.5:1(b)presence of collateral vessels(c)greater than 70% stenosis with poststenotic dilatation(d)transstenotic pressure gradient ≥40 mm Hg(e)decrease in renal size◊15-20% of patients remain hypertensive after restoration of normal renal blood flow (= renal artery stenosis without renovascular hypertension)!*Cause:* 1.Atherosclerosis (60-90%) mostly in proximal 2 cm of main renal artery◊Any of multiple renal arteries (occurring in 14-28% of the population) may be affected!2.Fibromuscular dysplasia (10-30%)3.Others (<10%): thromboembolic disease, arterial dissection, infrarenal aortic aneurysm, arteriovenous fistula, vasculitis (Buerger disease, Takayasu disease, polyarteritis nodosa, postradiation), neurofibromatosis, retroperitoneal fibrosis*Pathophysiology:* decreased perfusion pressure of glomeruli stimulates production of renin in juxtaglomerular apparatus + angiotensin II in kidney; renin converts angiotensinogen into angiotensin I, subsequently converted by angiotensin-converting enzyme (ACE) into angiotensin II which releases aldosterone; aldosterone increases salt + water retention; angiotensin II + aldosterone vasoconstrict vessels (especially intraglomerular efferent arteriole to maintain filtration pressure) *Histo:*tubular atrophy and shrinkage of glomeruli ● abdominal / flank pain ● hematuria ● hypertension ● oliguria, anuria ● low urine sodium concentration Patient selection criteria for screening test: 1.Well-documented recent-onset hypertension with diastolic pressure ≥105 mm Hg2.Patients <25 years of age developing hypertension3.Long-standing well-controlled hypertension becoming refractory to an existing regimen4.Refractory hypertension on an adequate 3-drug regimen (after exclusion of other causes)5.Generalized vascular disease6.Hypertension + abdominal bruit7.Hypertension + elevated serum creatinine (after exclusion of other causes)8.Hypertension treated with ACE inhibitors developing new / worsening of renal failure √ normal / decreased renal size (R 2 cm < L;L 1.5 cm < R) with smooth contour √ vascular calcifications (aneurysm / atherosclerosis)IVP (60% true-positive rate, 22% false-negative rate): √ delayed appearance of contrast material (decreased glomerular filtration)√ increased density of contrast material (increased water reabsorption)√ delayed washout of contrast material (prolonged urine transit time)√ lack of distension of collecting system√ global attenuation of contrast density, urogram may be normal with adequate collateral circulation√ notching of proximal ureter (enlargement of collateral vessels)CT: √ prolongation of cortical nephrographic phase + persistent corticomedullary differentiation√ CT angiography (2-3 mm collimation, pitch ≤1.5-2.0)Angiography: (a)conventional angiography = "gold standard" test(b)intravenous digital subtraction angiography:does not address hemodynamic significance NUC (75-95% sensitive, 80-93% specific): radionuclide renography (preferably with Tc-99m MAG_3) + angiotensin-I converting enzyme (ACE) inhibitor challenge which reduces GFR: ◊Discontinue ACE inhibitor therapy for >24 hours for enalapril + >48 hours for captopril / lisinopril!(a)captopril (Capoten®):Dose:1 mg/kg PO for pediatric patient, 25 or 50 mg PO for adult patientTechnique:radiopharmaceutical injected 60 minutes after ingestion of captopril(b)enalaprilat (Vasotec®):Dose:0.04 mg/kg IV (up to 2.5 mg maximum)Technique: }10 mL fluid/kg body weight PO over 1 hour (to ensure adequate hydration)}5 mCi IV Tc-99m MAG_3 + 20 mg IV furosemide}image acquisition for 22 minutes}postvoid image (or Foley catheter with PVR)}0.04 mg/kg IV enalaprilat (up to a maximum of 2.5 mg) infused over 5 minutes}5 mCi IV Tc-99m MAG3 + 20 mg IV furosemide injected 15 minutes after injection of enalaprilat}image acquisition for another 22 minutes

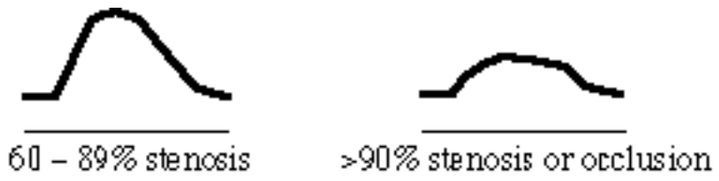


Semiquantitative interpretation of renograms: √ delay in the time to peak activity + elevation of 3rd phase of curve√ residual cortical activity (= activity remaining at 20 minutes expressed as percent of peak) >30% with increase by 10% over baseline following ACEI challenge√ asymmetry of renal uptake <40% of total renal uptake Duplex US: (1)direct signs = measurement at site of stenosis√ peak systolic velocity >150 cm/sec for angles <60° or 180 cm/sec for angles >70° (with many false positives due to suboptimal Doppler angles)√ ratio of peak renal artery velocity to peak aortic center stream velocity >3.5 (for >60% stenosis; 0-91% sensitive, 37-97% specific)√ poststenotic spectral broadening ± flow reversal√ absence of blood flow during diastole (for >50% stenosis)Problems: (a)technically inadequate examination (gas, corpulence, respiratory motion) in 6-49%; usually limited to children + thin adults(b)multiple renal arteries in 16-28%(c)"false" tracings from large collateral vessels / reconstituted segments of main renal artery(d)need to visualize entire length of renal artery(e)transmitted cardiac / aortic pulsations obscure renal artery waveform recordings(2)indirect signs = measurement of distal arterial segments√ tardus-parvus pulse:(a)gradual (= tardus) slope of Doppler waveform during systole = delay in acceleration / pulse rise time of ≥0.07-0.12 sec(b)attenuated (= parvus) Doppler waveform amplitude = decrease in peak systolic velocity to <20-30 cm/sec√ acceleration index = tangential inclination of Doppler waveform in early systole of ≥3 m/sec^2 (single most sensitive screening parameter; 76% sensitive + 95% specific at 20% disease prevalence)√ RI <0.56√ DRI >5% between both kidneys (82% sensitive + 92% specific for stenosis >50%, 100% sensitive + 94% specific for stenosis ≥60%)√ absent early systolic peak (ESP)√ segmental arterial flow detectable with renal artery occlusionFalse-negative:stenosis in accessory renal arteryFalse-positive:coarctation



Renal Artery Waveforms With Normal Early Systolic Peaks
AT = acceleration time; ΔV = velocity difference between early systolic peak velocity and late diastolic velocity; ESP = early systolic peak; LSP = late systolic peak; Acceleration index (AI) = ΔV/AT; 98% PPV for exclusion of renal artery stenosis with a normal spectral tracing from each renal pole

Renal Artery Waveforms with Normal Early Systolic Peaks
AT = acceleration time; DV = velocity difference between early systolic peak velocity and late diastolic velocity; ESP = early systolic peak; LSP = late systolic peak; Acceleration index (AI) = DV/AT; 98% PPV for exclusion of renal artery stenosis with a normal spectral tracing from each renal pole



Tardus-Parvus Pattern

Results for >60% renal artery stenosis: sensitivityspecificityaccuracyAT ≥ 0.07 sec81%95%91%AI < 30 cm/sec^{2}89%86%87%absent ESP92%96%95%

Arteriosclerotic Renal Artery Disease Fibromuscular Dysplasia Of Renal Artery Neurofibromatosis

Notes:

Arteriosclerotic Renal Artery Disease *Incidence:*in up to 6% of hypertensive patients; most common cause of secondary hypertension*Age:*>50 years; M > F*Path:*lesion primarily involving intima ● worsening of preexistent hypertension ● abrupt onset of severe hypertension >180/110 mm Hg ● vascular bruit in 40-50% (present in 20% of hypertensive patients without renal artery stenosis)*Associated with:*severe arteriosclerosis of aorta, cerebral, coronary, peripheral arteriesLocation:main renal artery (93%) + additional stenosis of renal artery branch (7%); bilateral in 31%Ψ eccentric stenosis in proximal 2 cm of renal artery, frequently involving orificeΨ decrease in renal length over time (= high-grade renal artery stenosis with risk for occlusion)*Prognosis:*progression of atherosclerotic lesion (40-45%) to renal atrophy, arterial occlusion, ischemic renal failure*Cx:*azotemia with(a)bilateral renal artery stenoses(b)unilateral renal artery stenosis + poorly functioning contralateral kidneyΔReversible azotemia may be induced by treatment with angiotensin-converting enzyme inhibitors / sodium nitroprusside!*Rx:*(1)Three-step antihypertensive therapy (control of hypertension difficult)(2)Angiotensin-converting enzyme inhibitors (eg, Captopril PO, Enalaprilat IV)(3)Renal artery angioplasty (80% success for nonostial lesion, 25-30% for ostial lesion)(4)Surgical revascularization (80-90% success for any lesion location)

Notes:

Fibromuscular Dysplasia Of Renal Artery *Incidence:*35% of renal artery stenoses; 1,100 patients reported (by 1982) with involvement of renal artery in 60% + extracranial carotid artery in 30%; 25% of all cases of renovascular hypertension*Age:*most common cause of renovascular hypertension in children + young adults <30-40 years; M:F = 1:3*Associated with:*fibromuscular dysplasia of other aortic branches in 1-2%: celiac a., hepatic a., splenic a., mesenteric a., iliac a., internal carotid a. ● hypertension ● progressive renal insufficiencySites:mid and distal main renal artery (79%), renal artery branches (4%), combination (17%); proximal third of main renal artery spared in 98%; bilateral in 2/3; R:L = 4:1 1.INTIMAL FIBROPLASIA (1-2%)*Path:*circumferential / eccentric fibrous tissue between intima + internal elastic lamina*Age:*children + young adults; M:F = 1:1Site:main renal artery + major segmental branches; often bilateral√ narrow annular radiolucent band√ poststenotic fusiform dilatation 2.MEDIAL FIBROPLASIA (60-85%)=medial fibroplasia with microaneurysm*Path:*multiple fibromuscular ridges + severe mural thinning with loss of smooth muscle + internal elastic laminaSite:mid + distal renal artery + branches; usually bilateral√ "string-of-beads" sign = alternating areas of stenoses (weblike constrictions) + aneurysms (which exceed the normal diameter of the artery)√ single focal stenosis 3.MEDIAL HYPERPLASIA (5-15%)*Path:*smooth muscle hyperplasia within arterial mediaSite:main renal artery and branches√ long smooth tubular narrowing 4.PERIMEDIAL FIBROPLASIA (20%)=subadventitial fibroplasia*Path:*fibroplasia of outer 1/2 of media replacing external elastic laminaSite:distal main renal artery√ long irregular stenosis√ beading = NO aneurysm formation (diameter of beads not wider than normal diameter of artery) 5.MEDIAL DISSECTION (5-10%)*Path:*new channel in outer 1/3 of media within external elastic laminaSite:main renal artery + branches√ false channel, aneurysm 6.ADVENTITIAL FIBROPLASIA (<1%)*Path:*adventitial + periarterial proliferation in fibrofatty tissueSite:main renal artery, large branches√ long segmental stenosis *Prognosis:*progression of lesions in 20% causing decline in renal function*Cx:*(1)Giant aneurysm(2)AV fistula between renal artery + vein (in medial fibroplasia)*Rx:*(1)Resection of diseased segment with end-to-end anastomosis(2)Replacement by autogenous vein graft, excision + repair by patch angioplasty(3)Transluminal balloon angioplasty (90% success rate with very low restenosis rate)

Notes:

Neurofibromatosis Hypertension in neurofibromatosis due to: (1)Pheochromocytoma(2)Renal artery stenosis◊Renal artery involvement mainly seen in children!*Types:* (a)mesodermal dysplasia of arterial wall with fibrous transformation (common)(b)narrowing of main renal artery by periarterial neurofibroma (rare)√ saccular funnel-shaped aneurysm involving aorta / main renal artery√ smooth / nodular stenosis (mural / adventitial neurofibroma) in proximal renal artery√ intrarenal aneurysm (rare)*DDx:*fibromuscular dysplasia; congenital renal artery stenosis

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Notes:

RENAL CELL CARCINOMA

=RCC = RENAL ADENOCARCINOMA= HYPERNEPHROMA*Incidence:*80-90% of all renal malignant primaries in adults; 1-3% of all visceral cancers (frequency approximates ovarian cancer, gastric cancer, pancreatic cancer, leukemia)*Age:* 6th-7th decade (generally >40 years); peak age of 55 years; may occur in children beyond age of 7 years; M:F = 2-3:1*Path:*arises from proximal tubular cells; 30% found incidentally with imaging; Tumor growth pattern: papillary (5-15%, best prognosis); trabecular / tubular / cystic / solid (poorer prognosis) *Histo:*(based on cytoplasmic criteria)(a)clear cell = rich in glycogen + lipid content(b)granular cell = intensely eosinophilic due to abundant mitochondria(c)mixed (most frequent type of RCC)(d)sarcomatoid*Predisposed:* (1)Tobacco; phenacetin abuse(2)von Hippel-Lindau disease (10-25%): often small intracystic tumors (hemangioblastoma, retinal angioma, renal cysts)(3)Hemodialysis (in 1.4-2.6%)(4)Acquired cystic disease of uremia (3.3-6.1%; 7 x increased risk)*Robson Staging Classification: Stage:* I:tumor confined to within renal capsule√ sharply defined convex interface with perirenal fatII:extension into perinephric fat but confined to Gerota fascia = renal fascia√ irregular interface between tumor + fatIII A:extension into renal vein or IVCIII B:positive lymph nodesIII C:extension into renal vein + lymph nodesIV A:extension into adjacent organs (other than ipsilateral adrenal)IV B:distant metastasesStaging accuracy:84-91% for CT82-96% for MR poor for US Regional extension:into lymph nodes (9-23%);into main renal vein (21-35%); into IVC (4-10%) Multiple RCC:commonly in von Hippel-Lindau syndrome; bilateral in 1-3% METASTASES ● bone pain, cough, hemoptysis (as initial symptoms of metastatic disease present in 9%)◊28% of patients have clinically apparent multiple distant metastases at presentation!Spread to: lung (55%); lymph nodes (34%); liver (33%); bone (32%); adrenals (19%); contralateral kidney (11%); brain (6%); heart (5%); spleen (5%); bowel (4%); skin (3%); ureter (rare) Incidence of metastatic disease: (a)tumors<3 cm:2.6%(b)tumors3-5 cm:15.4%(c)tumors>5 cm:78.6% ● hematuria (56%), flank pain (36%), weight loss (27%), fever (11-15%) ● classic triad of flank pain + gross hematuria + palpable renal mass (4-9%) ● varicocele (2%) ● normochromic normocytic anemia (28-40%) ● Stauffer syndrome (15%) = nephrogenic hepatopathy= hepatosplenomegaly + abnormal liver function in absence of hepatic metastases (? tumor hepatotoxin) ● Paraneoplastic syndromes: erythrocytosis (2%); hypercalcemia (parathormone, prostaglandin, vitamin D metabolites) √ often lobulated mass, focal bulge in renal contour√ enlargement of affected part of kidney√ calcification (8-18%): usually central + amorphous, peripheral + curvilinear in cystic RCC√ extrinsic compression / displacement / invasion of renal pelvis + calices√ cysts:(a)cystic necrotic tumor (40%)(b)cystadenocarcinoma (2-5%)(c)renal cell carcinoma in wall of cyst (3%)√ tumor growth into renal vein / IVC (30%) IVP: √ diminished function (parenchymal replacement, hydronephrosis)√ absence of contrast excretion (renal vein occlusion)√ pyelotumoral backflow = necrotic part of tumor fills with contrast materialCT: √ mostly inhomogeneous enhancement (due to cystic areas or necrosis)√ ± subcapsular / perinephric hemorrhageUS: √ hyperechoic (50-61%), mostly in small tumors <3 cm (78%), occasionally in large tumors (32%)√ markedly hyperechoic, ie, isoechoic to renal sinus fat, (4-12%) in small tumors (DDx: angiomyolipoma)√ anechoic rim (in 84% of small hyperechoic RCCs), probably due to pseudocapsule of compressed renal tissue (NOT seen in angiomyolipoma)√ isoechoic (30-86%) / hypoechoic (10-12%), mostly in larger tumors√ cystic with increase in acoustic transmission (2-13%) due to extensive liquefaction necrosis (DDx: complicated cyst)√ inhomogeneity due to hemorrhage, necrosis, cystic degenerationMRI (best modality to assess stage III + IV disease): √ low to medium signal intensity on T1WI; hyperintense areas are usually due to hemorrhage√ heterogeneous signal intensity on T2WIAngio: √ typically hypervascular (95%) with puddling of contrast + occasional AV shunting√ enlarged tortuous poorly tapering feeding vessels√ coarse neovascularity + formation of small aneurysms√ parasitization of lumbar, adrenal, subcostal, mesenteric artery branches√ poorly defined tumor margins *Prognosis:* ◊Tumor stage + histologic grade are the most important prognosticators!-5-year survival rates for stages I, II, III, IV are 85-100%, 45-65%, 20-40%, 0-10%;-10-year survival rates for stages I, II, III, IV are 56%, 28%, 20%, 3%-4.4% 3-year survival rate if untreated;-papillary carcinomas have better prognosis than nonpapillary carcinomas!-presence of spindle-shaped cells reduces survival!*Recurrence:*in 11% after 10 years *Rx:*radical nephrectomy (2-5% operative mortality) / parenchyma-conserving procedure dependent on tumor size + stage + grade

Cystic Renal Cell Carcinoma Papillary Renal Cell Carcinoma

Notes:

Cystic Renal Cell Carcinoma A.UNILOCULAR CYSTIC RCC (50%)=extensive necrosis of a previously solid RCC / intrinsic cystic growth of a cystadenocarcinoma fluid-filled mass without criteria of a renal cystB.MULTILOCULAR RCC (30%)=intrinsic multilocular growth impossible to distinguish from multilocular cystic nephromaC.MURAL NODULE IN CYSTIC RCC (20%)(a)asymmetric cystic tumor necrosis(b)tumor arising in wall of preexisting cyst(c)tubular dilatation with secondary cyst formation from tumor obstruction

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Notes:

Papillary Renal Cell Carcinoma *Incidence:*5-15% of all RCC*Age:*40-50 years*Path:*cystic necrosis + degeneration frequent; familial form associated with trisomy 17*Histo:*cells surrounding fronds of fibrovascular stroma; macrophages infiltrating the papillary stalks√ slow growing well-encapsulated tumor√ peripheral calcification frequent√ usually hypovascular√ little / no contrast enhancement√ frequently hypoechoic mass*Prognosis:*favorable (metastasize late)

Notes:

Simple Cortical Renal Cyst Acquired lesion possibly secondary to tubular obstruction; accounts for 62% of all renal masses *Incidence:*in 1-2% of all urograms; in 3-5% of all autopsies*Age:*peak incidence after age 30 years; increasing frequency with age (in 0.22% in pediatric age group, in 50% over age 50)*Path:*low cuboidal / flattened epithelium surrounded by 1-2 mm-thick fibrous wall containing clear / slightly yellow serous fluid *May be associated with:*tuberous sclerosis,von Hippel-Lindau disease, Caroli disease, neurofibromatosis √ large and unifocal when peripheral√ focal attenuation + displacement of collecting system√ focally replaced nephrogram with smooth margin√ "beak / claw sign" = effaced wedge of renal parenchyma√ delicate filamentous often undulating septa (10-15%)√ curvilinear calcification (1%) in wall / septaUS(90-100% accuracy of US & CT):√ spherical / ovoid in shape√ anechoic without internal echoes√ smooth clearly demarcated walls√ acoustic enhancement beyond cystCT: √ near-water-density lesion (<20-25 HU), thin wall, smooth interface with renal parenchyma, no enhancementCystography: √ smooth wall, clear aspirate with low lactic dehydrogenase, no fat content*Cx:*(1)Hemorrhage in 1-11.5%(2)Infection in 2.5%(3)Tumor within cyst in <1%

Notes:

Atypical / Complicated Renal Cyst A.HEMORRHAGIC CYST *Cause:*trauma, varices, bleeding diathesis ● rust-colored puttylike material√ uni- / multilocular cyst separated by thick septa√ thick fibrous ± calcified wall√ fibrin ball inside cyst (rare)CT: √ increased density secondary to acute hemorrhage / high protein contents (= hyperattenuating cyst with approximately 50-90 HU)√ no contrast enhancementMR: √ usually iso- to hyperintense on T1WI (owing to methemoglobin) + hyperintense on T2WI (due to lysis of RBCs)√ variable signal intensities (dependent on amount + acuity of hemorrhage, hemoglobin degradation product, degree of RBC lysis, protein content)√ hematocrit effect (= RBCs settle to cyst bottom)B. INFECTED CYST*Cause:*hematogenous dissemination of bacteria, ascending urinary tract infection*Mean age:*61 years; in 94% females ● history of no response to antibiotic Rx for acute pyelonephritis ● leukocyturiaUS: √ thickened irregular cyst wall (22%)√ internal septations (11%)√ wall calcification (occasionally)√ minute debris either diffusely / fluid-fluid level in dependent portion of cyst√ amorphous solid conglomerates √ round sharply marginated lesion *Dx:*cyst puncture*DDx:*renal abscess, hematoma, renal artery aneurysm, cystic tumor*Rx:*surgery, aspiration, serial follow-up

Notes:

Renal Sinus Cyst =PERIPELVIC / PARAPELVIC CYST = PARAPELVIC LYMPHANGIECTASIA = PARAPELVIC LYMPHATIC CYST=spherical fluid-filled masses intimately attached to renal pelvis without connection to pelvicaliceal system either arising from renal sinus or parenchyma*Incidence:*1.5% (autopsies); 4-6% of all renal cysts*Etiology:* probably ectatic lymphatic channels from lymphatic obstruction; ? posttraumatic extravasation of urine / blood; ? protrusion of parenchymal cysts into sinus; ? mesonephric remnant; ? remnant of wolffian body; ? outpouchings of renal pelvis; ? duplication anomaly *Age:*mostly during 5th-6th decade ● almost always asymptomatic ● pain (from obstructive caliectasis) ● renal vascular hypertension (compression of renal arteries) ● clear straw-colored serous fluid√ soft-tissue density in renal sinus√ focal displacement + smooth effacement of collecting system√ stretching of collecting system when generalized (indistinguishable from sinus lipomatosis)√ rarely curvilinear calcification of cyst wall (4%)US: √ anechoic mass(es) with acoustic enhancement, irregular shape*Cx:*obstructive caliectasis (rarely hydronephrosis)*Rx:*cyst ablation with 95% ethanol if symptomatic*DDx:*hydronephrosis

Notes:

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RENAL DYSGENESIS

=undifferentiated tissue of renal anlage◊Pathologic NOT radiologic diagnosis√ renal vessels usually absent; occasionally small vascular channels

Notes:

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RENAL INFARCTION

Causes: 1.TRAUMA: blunt abdominal trauma, traumatic avulsion of renal artery, surgery2. EMBOLISM: (a)Cardiac: rheumatic heart disease with arrhythmia (atrial fibrillation), myocardial infarction, prosthetic valves, myocardial trauma, left atrial / mural thrombus, myocardial tumors, subacute bacterial endocarditis(b)Catheters: angiographic catheter manipulation, umbilical artery catheter above level of renal arteries3. ARTERIAL THROMBOSIS: arteriosclerosis, thrombangitis obliterans, polyarteritis nodosa, syphilitic cardiovascular disease, aneurysm (aorta / renal artery), sickle cell disease 4. Sudden complete renal vein thrombosis

Acute Renal Infarction Lobar Renal Infarction Chronic Renal Infarction Atheroembolic Renal Disease Arteriosclerotic Renal Disease

Notes:

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Acute Renal Infarction √ normal / large kidney with smooth contour√ normal / expanded parenchymal thickness√ normal / attenuated collecting system, often only opacified by retrograde pyelography√ absent / diminished nephrogram with cortical rim enhancement, rarely striationsUS: √ diminished echogenicity (within <24 hours) √ normal echogenicity (echoes appear within 7 days)NUC (SPECT imaging with Tc-99m DMSA): √ photon-deficient area

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Notes:

Lobar Renal Infarction EARLY SIGNS: √ focal attenuation of collecting system (tissue swelling)√ focally absent nephrogram (triangular with base at cortex)LATE SIGNS: √ normal / small kidney(s) √ focally wasted parenchyma with NORMAL interpapillary line (portion of lobe / whole lobe / several adjacent lobes)CT: √ nonperfused area corresponding to vascular division, cortical rim signUS: √ focally increased echogenicity

Notes:

Chronic Renal Infarction *Path:*all elements of kidney atrophied with replacement by interstitial fibrosis√ normal / small kidney with smooth contour√ globally wasted parenchyma√ diminished / absent contrast material densityUS: √ increased echogenicity (by 17 days)Angio: √ normal intrarenal venous architecture√ late visualization of renal arteries on abdominal aortogram

Notes:

Atheroembolic Renal Disease =dislodgment of multiple atheromatous emboli from the aorta into renal circulation (below level of arcuate arteries)√ normal / small kidneys with smooth contour or shallow depressions√ wasted parenchymal thickness√ diminished density of contrast materialCT: √ patchy nephrographic distributionAngio: √ embolic occlusion

Notes:

Arteriosclerotic Renal Disease =disseminated process involving most of the interlobar + arcuate arteries causing uniform shrinkage of kidney*Age:*generally over 60 yearsAccelerated development in:scleroderma, polyarteritis nodosa, chronic tophaceous gout ● often associated with hypertension (NEPHROSCLEROSIS)√ normal / small kidneys√ smooth contour with random shallow contour depressions (infarctions)√ uniform loss of cortical thickness√ normal / effaced collecting system (fat proliferation)√ increased pelvic radiolucency (vicarious sinus fat proliferation)√ calcification of medium-sized intrarenal arteriesUS: √ increased echogenicity possible√ increased size of renal sinus echoes (fatty replacement) ***Nephrosclerosis*** *Histo:*thickening + hyalinization of afferent arterioles, proliferative endarteritis, necrotizing arteriolitis, necrotizing glomerulitis ● arterial hypertension(a)BENIGN NEPHROSCLEROSIS(b)MALIGNANT NEPHROSCLEROSIS (rapid deterioration of renal function)√ radiographic appearance similar to arteriosclerotic kidney

Notes:

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RENAL LEIOMYOMA
=CAPSULOMA*Prevalence:*5% at autopsy (average size of 5 mm)*Median age:*42 years; M < F*Path:*well-circumscribed lesion with mean size of 12 cm containing hemorrhage (17%) / cystic degeneration (27%)Location:53% subcapsular, 37% capsular, 10% attached to renal pelvis*Associated with:*tuberous sclerosis ● palpable mass (50%), hematuria (20%)√ well-circumscribed exophytic solid lesion ± cleavage plane between tumor and cortex*DDx:*renal leiomyosarcoma, adenocarcinoma

Notes:

RENAL TRANSPLANT

Frequency: 11,000 transplants per year in USA (1994) Complications in 10% ◊Problematic period between 4 days and 3 weeks after surgery! ● hypertension in 50% (from rejection / arterial stenosis)*Prognosis:*organ survival at 2 years in 5% for cadaveric Tx / 88% for living related donor grafts; 7-8 years half-life for cadaveric Tx; 13-24 years half-life for Tx from living related donor

Renal Transplant Scintigram				
	early study (≤24 hours post transplantation)		late study (≥5 days post transplantation)	
	flow	excretion	flow	excretion
Acute tubular necrosis	nl / mildly decreased	decreased	nl / mildly decreased	mildly decreased
Hyperacute rejection	absent	absent		
Acute rejection	decreased	decreased	worsening	worsening
Chronic rejection	decreased	decreased	decreased	decreased

Acute Tubular Necrosis In Renal Transplant Rejection Of Renal Transplant Cyclosporine Nephrotoxicity Urologic Problems With Renal Transplant Vascular Problems With Renal Transplant Gastrointestinal Problems With Renal Transplant Hypertension With Renal Transplant Aseptic Necrosis With Renal Transplant Posttransplant Lymphoproliferative Disease

Notes:

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Acute Tubular Necrosis In Renal Transplant =primary nonfunction within 72 hours of transplantation followed by improvement within a few days to 1 month secondary to ischemia-ATN more frequent in cadaveric than living-related donor transplant (donor hypotension)-ATN greater in transplants with more than one renal artery-ATN related to length of ischemic interval (prolonged organ storage) ● no constitutional symptoms ● elevated urine sodium ● oliguria may begin immediately after transplantation / may be delayed for several daysUS: √ transient enlargement of transplant√ transient increase in resistive indexScintigram: √ normal / slightly decreased transplant perfusion√ decreased + delayed radiopharmaceutical uptake√ delayed / decreased / absent excretion of Tc-99m*DDx:*acute rejection (serial renal studies help to differentiate)

Notes:

Rejection Of Renal Transplant ◊Most common cause of parenchymal failure!◊Rejection occurs in all transplants to some degree!

Causes of Renal Allograft Dysfunction	
Immediate to 1st 48 hours 1. Hyperacute rejection 2. Renal vein thrombosis 3. Discordant size	**Day 2 to day 7** 1. ATN 2. RVT
>1 week post-op 1. Acute rejection 2. ATN	**Delayed** 1. Chronic rejection 2. Drug toxicity 3. Obstruction 4. Infection 5. Extrinsic compression

1.**Hyperacute rejection** (rare)=humeral rejection with preformed circulating antibodies present in recipient at time of transplantation, usually following retransplantation*Path:*thrombosed arterioles + cortical necrosis*Time of onset:*within minutes after transplantation√ complete absence of renal perfusion + renal function on Tc-99m DTPA scan (DDx: complete arterial / venous occlusion)*Rx:*requires immediate reoperation2.**Accelerated acute rejection** =combination of antibody + cell-mediated rejection*Time of onset:*2-5 days after transplantation3.**Acute rejection** =cellular rejection predominantly dependent on cellular immunity*Time of onset:*any time, typically within 5 days to 6 months; peak incidence at 2nd-5th week*Path:* (a)acute interstitial rejection=edema of interstitium with lymphocytic infiltration of capillaries + lymphatics (b)acute vascular rejection (rare)=proliferative endovasculitis + vessel thrombosis ● low urine sodium, increase in serum creatinine ● hypertension ● oliguria ● fever ● tenderness of transplant ● weight gainUS (30-50% negative predictive value): √ increase in renal volume from edema= decreased renal sinus fat with increased cortical thickness (most predictive)√ conspicuous pyramids + decreased cortical echogenicity√ thickening of pelvoinfundibular wall√ diminished echogenicity of renal sinus fatDoppler (higher accuracy than morphologic parameters): √ initially decrease in resistive index(? autoregulatory mechanism)√ increase in resistive index with increasing severity of rejection(a)≤0.70 without any form of rejection (57% negative predictive value)(b)>0.90 (100% positive predictive value, 26% sensitivity)NUC: √ may show decreased renal perfusion + renal function √ initially perfusion may be normal with only function decreased (DDx to ATN may not be possible on single study)√ subsequent exams (1-3 day intervals) demonstrate decreasing renal perfusion√ prolonged excretory phase√ poor and inhomogeneous nephrogramAngio:√ rapid tapering + pruning of interlobar arteries√ multiple stenoses + occlusions√ nonvisualization of interlobular arteries√ prolonged arterial opacification (normally <2 sec)4.**Chronic rejection** =slow relentless progressive process resulting in interstitial scarring + fibrosis*Path:*endothelial proliferation in small arteries + arterioles; glomerular lesions (? recurrence of patients original glomerulonephritis)*Time of onset:*months to years after transplantation√ small kidney√ diminished number of intrarenal vessels√ vascular pruning / stenoses / occlusions

Notes:

Cyclosporine Nephrotoxicity *Action:*impedes rejection process with narrow therapeutic window*Action:*impedes rejection process with narrow therapeutic window
Histo:(a)acutely: damage to tubules, microthrombosis of kidney (secondary to activation of coagulation cascade) (b)chronically: hyaline deposition within arterial walls
√ NO change in renal size / resistive index

Notes:

Urologic Problems With Renal Transplant 1.**Ureteral obstruction** (1-10%) (a)acute:secondary to technical problems (b) late:secondary to ischemia or previous extravasation *Causes:*stricture (most commonly at ureterovesical junction), ureteral kinking, (transient) edema at ureteroneocystostomy, ureteropelvic fibrosis, crossing vessels, blood clot, lymphocele, fungus ball, calculus √ pyelocaliectasis √ normal resistive index strongly argues against obstruction unless ureteral leak is present *DDx:*low-pressure dilatation secondary to denervation + handling of relatively large urine volume (confirmed by Whitaker test)
2.**Urine extravasation** (3-10%) *Causes:* (1)Distal ureteral necrosis secondary to interruption of blood supply (early) / vascular insufficiency due to rejection (late) (2)Leakage from ureteroneocystostomy site (3)Leakage from anterior cystostomy closure site (4)Segmental renal infarction ● high creatinine level in fluid collection *Prognosis:*high morbidity + mortality (death from transplant infection + septicemia)
3.**Pararenal fluid collection** *Incidence:*in up to 50% of transplantations *Cx:*Page kidney (1)Lymphocele (10%) occur weeks to month after transplantation ● does not contain creatinine √ mean diameter of 11 cm √ thick septa (50%) + internal debris *Rx:*sclerotherapy with povidone-iodine; long-term catheter drainage / surgical marsupialization (2)Urinoma √ rarely septated + smaller than lymphoceles (3)Abscess, hematoma ● small hematomas typically resolve spontaneously within a few weeks √ photopenic region with displacement / impression on kidney / urinary bladder
mnemonic:"HAUL" **H**ematoma **A**bscess **U**rinoma **L**ymphocele

Notes:

Vascular Problems With Renal Transplant A.PRERENAL 1.**Renal artery stenosis** (1-12%) ◊Transient elevation of velocities in immediate postoperative period is due to vessel wall edema / arterial spasm! *Time of onset:*within 3 years; cadaver kidney > young donor kidney > living-related donor kidney (a)short-segment stenosis at anastomosis: technical (75%), use of clamp / cannula, trauma, ischemia of donor vessel (b)long-segment stenosis: trauma during allograft harvesting, faulty operative technique, chronic rejection, atherosclerosis, kinking, scar formation ● recent onset of hypertension ● renal insufficiency ● bruit over graft site (occasionally) √ increase in peak systolic velocity >200-210 cm/sec √ 2:1 ratio between peak stenotic and poststenotic velocities √ main renal artery/external iliac artery ratio >3.5 √ gross poststenotic turbulence (supportive evidence) √ dampened signals distal to stenosis √ increase in acceleration time (= pulse rise time) Angio: √ standard test for detection of arterial stenosis *Cx (0.5-2.3%):*hemorrhage, intimal flap, arteriovenous fistula
2.**Renal artery thrombosis** (1-5%) *Cause:*rejection, faulty surgical technique Time of onset:within 1st month *Predisposed:*allografts with disparate vessel size, multiple anastomoses, intramural vessel injury due to faulty handling, rejection ● early sudden onset of anuria √ global absence of perfusion, uptake, excretion √ segmental infarction due to occlusion of polar artery √ hypo- / hyperechoic area ± cortical thickening √ no flow in affected area
3.**Pseudoaneurysm** (in up to 17%) *Cause:*percutaneous biopsy with vascular injury, faulty surgical technique, perivascular infection Location: (a)at anastomotic site: due to suture rupture, anastomotic leakage, vessel wall ischemia (b)mostly of arcuate arteries within allograft: following needle biopsy, mycotic infection √ hypoechoic mass √ mixed arterial + venous pulsations within mass *Prognosis:*mostly spontaneous regression
4.**Arteriovenous fistula** (in 2%) *Cause:*percutaneous biopsy with vascular injury, faulty surgical technique, perivascular infection ● hypertension, hematuria, high-output cardiac failure √ high-velocity low-resistance flow in feeding artery √ arterialization of waveform in draining vein √ turbulence + high-frequency velocity shift √ exaggerated focal color around lesion (= perivascular soft-tissue vibration = bruit)
5.**Renal allograft necrosis** =total lack of perfusion in an area of renal cortex associated with variable degrees of medullary necrosis *Cause:*rejection, surgical ligature, preexistent arterial lesion, severe ATN, prolonged time of warm ischemia Pattern: 1.Small focal necrosis 2.Large isolated area of infarction (segmental arterial occlusion) 3.Outer cortical necrosis 4.Cortical necrosis with large patches 5.Diffuse cortical necrosis 6.Cortical + medullary necrosis 7.Necrosis of whole kidney (occlusion of main renal artery) MR: √ slightly hyperintense (ischemic necrosis) / hypointense (hemorrhagic necrosis) / isointense area on T2WI √ hypointense areas on Gd-DTPA images US: √ hypoechoic (ischemic necrosis) / iso- or hyperechoic (hemorrhagic necrosis) areas √ swollen area (probably cortical edema) √ absence of arterial perfusion by color duplex (not sensitive for small infarcts / superficial cortical necrosis) √ elevated resistive indexes + no / reversed diastolic flow

B.POSTRENAL 1.**Renal / iliac vein thrombosis** (4.2-5%) *Cause:* (a)immediately: injury to epithelium at site of renal vein anastomosis, extrinsic compression by urinoma / hematoma / lymphocele (b)after 1st week: acute rejection, reduced intrarenal arterial flow ● abrupt onset of renal dysfunction ● graft tenderness ● hematuria, proteinuria √ enlargement of transplant √ prolonged arterial transit time without arterial occlusions + arterial spasms √ diminished cortical perfusion √ absent venous flow √ "U-shaped" / plateau-like reversal of diastolic arterial flow √ decreased systolic rise time

HIGH VASCULAR IMPEDANCE OF RENAL TRANSPLANT =pulsatility index (A - B/mean) greater than 1.8 or resistive index (A -B/B) of Doppler signals of 0.75-0.80 indicate a reduction in diastolic flow velocity *Causes:* (a)intrinsic vascular obstruction 1.Acute vascular rejection (later stage) 2.Renal vein obstruction (b)increased intraparenchymal pressure 1.Severe ATN 2.Severe pyelonephritis: CMV, herpes, E. coli, C. albicans 3.Extrarenal compression: large collection, hematoma, discordant size 4.Urinary obstruction (doubted!) 5.Excessive pressure by transducer

Notes:

Gastrointestinal Problems With Renal Transplant *Incidence:*40% 1.Gastrointestinal hemorrhage (a)Upper GI tract bleeding gastric erosions, gastric / duodenal ulcers *Mortality rate:* 2-3 x of normal (b)Lower GI tract bleeding hemorrhoids, pseudomembranous colitis, cecal ulcers, colonic polyps 2.GI tract perforation (3%) *Causes:*spontaneous, antacid impaction, perinephric abscess, diverticular disease Location:colon > small bowel > gastroduodenal *Mortality rate:*approaches 75% (because of delayed diagnosis)

Notes:

Hypertension With Renal Transplant Leading cause of death in renal transplant recipient! *Prevalence:*up to 60% 1 year after transplantation *Cause:* A.TRANSPLANT RELATED 1.Acute transplant rejection 2.Chronic rejection 3.Cyclosporine toxicity 4.Ureteral obstruction 5.Renal artery stenosis (a)accelerated atherosclerosis (b)postsurgical fibrosis at anastomosis B.NOT TRANSPLANT RELATED 1.Renin production of native kidney 2.Original renal disease involving transplant 3.Development of essential hypertension

Notes:

Aseptic Necrosis With Renal Transplant Most common long-term disabling complication; femoral head most common site, bilateral in 59-80% *Frequency:*6-15-29% within 3 years after surgery *Time of onset:*symptoms develop 5-126 (mean 9-19) months after transplantation *Risk factors:* dose + method of glucocorticoid administration, duration + quality of dialysis before transplantation, secondary hyperparathyroidism, allograft dysfunction, liver disease, previous transplantation, iron overload, increased protein catabolism during dialysis *Pathophysiology of corticosteroid therapy:* (1)Fat embolism (fat globules occlude subchondral end arteries) (2)Increase in fat cell volume in closed marrow space (increase in intramedullary pressure leads to diminished perfusion) (3)Osteopenia (increased bone fragility) (4)Reduced sensibility to pain (loss of protection against excessive stress) *Histo:*fragmentation, compression, resorption of dead bone, proliferation of granulation tissue, revascularization, production of new bone ● 40% asymptomatic ● joint pain ● restriction of movement Sites:femoral head, femoral condyles (lateral > medial condyle), humeral head √ subchondral bone resorption √ patchy osteosclerosis √ collapse / fragmentation of bone MR with abbreviated T1WI protocol = test of choice!
see page 37 AVASCULAR NECROSIS

Notes:

Posttransplant Lymphoproliferative Disease =abnormal proliferation of B-cell lymphocytes strongly associated with Epstein-Barr virus infection (in 80%); up to 11% may arise from T-cell lymphocytes *Incidence:* 0.6%after bone marrow transplantation, 1-6%after kidney transplantation (in 20% NHL, especially affecting CNS) 1.8-20%after cardiac transplantation ◊Prevalence of NHL is 35 x greater than in general population! *Cause:*sequela of chronic immunosuppression with limited ability to suppress neoplastic activity Types: 1.Polyclonal B-cell hyperplasia (nearly identical to infectious mononucleosis) 2.Monoclonal non-Hodgkin lymphoma Time of onset:as early as 1 month after transplantation depending on immunosuppressive regimen Location: @Lymph nodes: tonsils, cervical neck nodes @Gastrointestinal tract *Cx:*visceral perforation (frequent) @Thorax √ multiple / solitary well-circumscribed pulmonary nodules ± mediastinal lymphadenopathy (DDx: cryptococcosis, fungus, Kaposi sarcoma) √ patchy airspace consolidation (DDx: edema, infection, rejection) *DDx:*lymphoid hyperplasia (spontaneous resolution) *Rx:*(1)Antiviral agents (controversial) (2)Reduction / cessation of immunosuppressive agents (3)Surgical resection of tumor mass (complete resolution in 63%)

Notes:

RENAL TRAUMA

Classification: 1.Superficial cortical laceration (75-85%) (a)Subcapsular hematoma √ lenticular-shaped area + flattening of subjacent parenchyma (b)Renal contusion √ poorly defined area of low attenuation (c)Small cortical laceration without caliceal disruption *Rx:*observation 2.Complete cortical laceration / fracture communicating with caliceal system (10%) √ extravasation of contrast material √ separation of renal poles (= fracture) *Rx:*clinical judgement required 3.Shattered kidney / injury to the renal vascular pedicle (5%) √ multiple separate renal fragments (= shattered kidney) √ lack of enhancement of part / all of kidney √ ± "rim sign" (= enhancement of renal periphery through intact capsular / collateral vessels) √ extravasation of contrast material *Rx:*surgery *DDx:*respiratory motion artifact (low-attenuation area surrounding kidney)

Notes:

RENAL TUBULAR ACIDOSIS
=clinical syndrome characterized by tubular insufficiency to resorb bicarbonate, excrete hydrogen ion, or both (= nonanion gap metabolic acidosis) ● failure to thrive

Proximal Renal Tubular Acidosis Distal Renal Tubular Acidosis

Notes:

Proximal Renal Tubular Acidosis =TYPE 2 RTA =impaired capacity to absorb HCO_3^- in proximal tubule leads to presence of bicarbonate in urine at lower plasma levels than normal *Pathogenesis:* ? defect in $Na^+/HCO3^-$ cotransport at basolateral membrane; deficit of carbonic anhydrase; parathyroid hormone activates cyclic AMP which inhibits carbonic anhydrase (hypocalcemia of hyperparathyroidism + various types of Fanconi syndrome) ● self-limited acidosis (bicarbonate loss stops once bicarbonate threshold of about 15 mEq/L is reached) ● unimpaired ability to lower urine pH (pH 4.5-7.8 depending on level of plasma bicarbonate) by normal excretion of hydrogen ions ● hypokalemia (due to hyperaldosteronism secondary to decreased proximal resorption of NaCl) ↓ rickets / osteomalacia N.B.:NEVER nephrocalcinosis / nephrolithiasis (due to normal urinary citrate excretion, low urine pH, self-limited less severe acidosis with less calcium release from bone *Dx:*bicarbonate titration test, large requirement of alkali to sustain plasma bicarbonate level at 22 mmol/L *Rx:*administration of alkali ± potassium ± hydrochlorothiazide 1.INFANTILE TYPE OF PRIMARY PROXIMAL RTA *Age:*diagnosed within first 18 months of life; usually male patients ● excessive vomiting in early infancy ● growth retardation (<3rd percentile) ● metabolic hyperchloremic acidosis ● normal quantities of net acid excretion *Prognosis:*transient type with spontaneous remission 2.SECONDARY PROXIMAL RTA =tubular defect of bicarbonate resorption associated with other tubular dysfunction / generalized disease *Cause:* -Fanconi syndrome, cystinosis, Lowe syndrome, hereditary fructose intolerance, glycogen storage disease, galactosemia, tyrosinemia, Wilson disease, Leigh syndrome -1° + 2° hyperparathyroidism, vitamin D deficiency, mineralocorticoid deficiency, osteopetrosis -medullary cystic disease, renal transplantation, vascular accident to kidney in newborn period, multiple myeloma, amyloidosis, nephrotic syndrome, cyanotic CHD, Sjögren syndrome -intoxication with cadmium, outdated tetracycline, methylchromone, 6-mercaptopurine

Notes:

Distal Renal Tubular Acidosis =TYPE 1 RTA (first type discovered) =impaired ability to secrete H^+ in distal tubule despite low levels of plasma bicarbonate (urine cannot be acidified with pH invariably high at >5.5-6.0)
Pathophysiology: primary defect of nonacidification of urine followed by (a)hyperchloremia small constant loss of serum sodium bicarbonate ($NaHCO_3$) without concomitant loss of chloride (NaCl retention) leads to shrinkage of ECF volume (b)chronic severe + progressive acidosis (due to inability to excrete the usual endogenously produced nonvolatile acid) leads to -mobilization of calcium + phosphate from bone (osteomalacia) -growth retardation -hypercalciuria (+ 2° hyperparathyroidism) -loss of phosphate (osteomalacia / rickets) (c)nephrocalcinosis + nephrolithiasis (due to combination of hypercalciuria + elevated urine pH + marked reduction in urinary citrate) (d)potassium wastage with hyperkaliuria + hypokalemia (due to constant small loss of sodium bicarbonate in urine, reduction of ECF space, 2° hyperaldosteronism, increase in sodium-potassium exchange in distal tubule)
*Path:*calcium deposits accompanied by chronic interstitial nephritis with cellular infiltration, tubular atrophy, glomerular sclerosis ● muscle weakness, hyporeflexia, paralysis (due to hypokalemia) ● bone pain (due to osteomalacia) ● polyuria (from defect in urinary concentrating ability as a result of nephrocalcinosis + potassium deficiency) ● low plasma bicarbonate ● hyperchloremic acidosis (from impaired ability to excrete the usual endogenous load of nonvolatile acid) ● alkaline urine (pH >5.0-5.5) ● hypokalemia, loss of sodium ● hypercalciuria (continued mobilization of calcium phosphate from bone due to metabolic acidosis) ● hypocitraturia (increased proximal tubular reabsorption of citrate) *Dx:*acid load test with ammonium chloride (NH_4Cl) *Rx:*administration of mixture of sodium + potassium bicarbonate
*Cx:*interstitial nephritis, chronic renal failure (damage from nephrocalcinosis + secondary pyelonephritis), bone lesions, nephrocalcinosis, nephrolithiasis
1.PERMANENT DISTAL RTA =ADULT TYPE OF PRIMARY DISTAL RTA =BUTLER-ALBRIGHT SYNDROME *Genetics:*mostly sporadic, may be autosomal dominant *Age:*children + adults (usually not diagnosed before age 2); F > M ● vomiting, constipation, polyuria, dehydration ● failure to thrive, growth retardation, anorexia ● polyuria (due to renal concentrating defect) ● potassium loss resulting in flaccid paralysis ● bone pain + pathologic fractures in adolescents + adults (from osteomalacia) ● low serum pH, low bicarbonate concentration ● elevation of chloride ● urinary pH of 6.0-6.5 √ rickets / osteomalacia √ moderately retarded bone age √ medullary nephrocalcinosis / nephrolithiasis (as early as 1 month of age)
2.SECONDARY DISTAL RTA (a)systemic conditions: -starvation, malnutrition, sickle cell disease -primary hyperthyroidism + nephrocalcinosis, 1° hyperparathyroidism + nephrocalcinosis, vitamin D intoxication, idiopathic hypercalcemia, idiopathic hypercalciuria + nephrocalcinosis -amphotericin B nephropathy, toxicity to lithium, toluene sniffing -hepatic cirrhosis, fructose intolerance with nephrocalcinosis, Ehlers-Danlos syndrome, Marfan syndrome, elliptocytosis (b)renal conditions: renal tubular necrosis, renal transplantation, medullary sponge kidney, obstructive uropathy (c)hypergammaglobulinemic states (? autoimmune process): idiopathic hypergammaglobulinemia, chronic active hepatitis, hyperglobulinemic purpura, Sjögren syndrome, cryoglobulinemia, systemic lupus erythematosus, lupoid hepatitis, fibrosing alveolitis
[TRANSIENT DISTAL RENAL TUBULAR ACIDOSIS =INFANTILE TYPE OF PRIMARY DISTAL RTA =LIGHTWOOD SYNDROME =transient self-limited form in infancy (only observed within 1st year of life) with unclear pathophysiology, probably due to vitamin D intoxication]

Notes:

RENAL VEIN THROMBOSIS
*Prevalence:*0.5% (autopsy) *Causes:* A.Intrinsic =thrombotic process begins intrarenally within small intrarenal veins due to acidosis, hemoconcentration, disseminated intravascular coagulation, intrarenal arteriolar constriction reducing venous flow (a)antenatally: abruptio placentae (b)newborns: advanced maternal age, glycosuria in infants of diabetic mothers, dehydration from vomiting, diarrhea, enterocolitis, sepsis, polycythemia, birth trauma, left adrenal hemorrhage, prematurity (c)adults: membranous GN, pyelonephritis, amyloidosis, polyarteritis nodosa, sickle cell anemia, thrombosis of IVC, renal neoplasia (50%), low flow states (CHF, constrictive pericarditis), diabetic nephropathy, lupus nephropathy, sarcoidosis, hypercoagulable states, trauma B.Extrinsic umbilical vein catheterization, thrombosis of IVC with extension into renal vein, malpositioned IVC filter, carcinoma of pancreatic tail invading renal vein (in 75%), pancreatitis, lymphoma, retroperitoneal sarcoma, retroperitoneal fibrosis, metastases to retroperitoneum (bronchogenic carcinoma)
mnemonic:"TEST MAN" **T**hrombophlebitis **E**nterocolitis (dehydration) **S**ickle cell disease, **S**ystemic lupus erythematosus **T**rauma **M**embranous glomerulonephritis **A**myloidosis **N**eoplasm
Radiographic appearance varies with: (1) rapidity of venous occlusion (2) extent of occlusion (3) availability of collateral circulation (4) site of occlusion in relation to collateral pathways *Pathophysiology:*formation of collateral channels develops at 24 hours + peaks at 2 weeks after onset of occlusion Collaterals:ureteral v. to vesicular vv., pericapsular vv. to lumbar vv., azygos v., portal v. on left:in addition gonadal v., adrenal v., inferior phrenic vv.

Acute Renal Vein Thrombosis Subacute Renal Vein Thrombosis Chronic Renal Vein Thrombosis

Notes:

Acute Renal Vein Thrombosis *Path:*hemorrhagic renal infarction from ruptured venules + capillaries without time for effective development of collaterals ● gross hematuria, proteinuria ● asymptomatic / painful flank mass ● consumptive thrombocytopenia ● anuria, hypertension √ smooth enlargement of kidney (edema + hemorrhage) √ initially faint + delayed dense nephrogram √ little / no pyelocaliceal visualization √ focal hemorrhagic infarction + capsular rupture US: √ enlarged kidney of variably altered echotexture √ thrombus within distended renal vein / IVC Doppler-US: √ venous flow present in segmental veins + collateral veins overlying renal hilum mimicking patency of main renal vein √ steady / less pulsatile venous flow compared with contralateral main renal vein √ main renal vein not traceable into IVC on color Doppler √ elevated resistive index >0.70 ± reversed end-diastolic renal arterial flow in native kidney CT: √ prolonged cortical nephrographic phase + persistent corticomedullary differentiation √ thickened renal fascia + perirenal stranding √ retroperitoneal hemorrhage Angio: √ poorly filling cortical arteries √ absent inflow from renal vein into IVC √ thrombus extending into IVC NUC: √ no characteristic pattern on sequential functional study *Cx:*(1) Pulmonary emboli (50%) (2)Severe renal atrophy (may show complete recovery)

Notes:

Subacute Renal Vein Thrombosis =good collateral drainage; impaired function with steady state or recanalization √ enlarged edematous boggy kidney √ slightly diminished / normal nephrographic density (may increase over time) √ compression of collecting system ("spidery calices") √ increased renal cortical echogenicity √ collateral veins allow venous efflux normalizing arterial waveform √ main renal vein appears small due to recanalization

Notes:

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Chronic Renal Vein Thrombosis =indolent stage ● 80-90% asymptomatic ● nephrotic syndrome (proteinuria, hypercholesterolemia, anasarca) √ normal excretory urogram in 25% (with good collateral circulation especially if left side affected) √ notching of collecting system + proximal ureter √ retroperitoneal dilated collaterals √ lacelike intrarenal pattern of calcifications US: √ branching linear calcifications (calcified thrombus) √ small echogenic kidney CT: √ renal vein + IVC thrombus (24%); perirenal collaterals √ prolonged corticomedullary differentiation √ delayed / absent pyelocaliceal opacification + attenuated collecting system √ thickening of Gerota fascia Arteriography: √ enlarged venous collaterals on delayed images

Notes:

RETROCAVAL URETER
=CIRCUMCAVAL URETER = abnormality in embryogenesis of IVC with abnormal persistence of right subcardinal vein ventral to ureter (instead of right supracardinal vein, which is dorsal to right ureter) *Incidence:*0.07%; M:F = 3:1 ● symptoms of right ureteral obstruction √ ureteral course swings medially over pedicle of L3/4, passing behind IVC, and then exiting anteriorly between IVC and aorta returning to its normal position √ varying degrees of hydronephrosis + proximal hydroureteronephrosis

Notes:

RETROPERITONEAL FIBROSIS
=ORMOND DISEASE = CHRONIC PERIAORTITIS *Path:*dense hard fibrous tissue enveloping the retroperitoneum with effects on ureter, lymphatics, great vessels *Causes:* A.PRIMARY RETROPERITONEAL FIBROSIS (2/3) Probably autoimmune disease with antibodies to ceroid (by-product of aortic plaque, which has penetrated into media) leading to systemic vasculitis; *Associated with fibrosis in other organ systems (in 8-15%):* mediastinal fibrosis, Riedel fibrosing thyroiditis, sclerosing cholangitis, fibrotic orbital pseudotumor *Age:*31-60 years (in 70%); M:F = 2:1 *Rx:*responsive to corticoids B.SECONDARY RETROPERITONEAL FIBROSIS (1/3) (1)Drugs (12%): methysergide, b-blocker, phenacetin, hydralazine, ergotamine, methyldopa, amphetamines, LSD (2)Desmoplastic response to malignancy (8%): lymphoma, Hodgkin disease, carcinoid, retroperitoneal metastases (breast, lung, thyroid, GI tract, GU organs) (3)Retroperitoneal fluid collection: from trauma, surgery, infection (4)Aneurysm of aorta / iliac arteries (desmoplastic response) (5)Connective tissue disease: eg, polyarteritis nodosa (6)Radiation therapy *Peak age:*40-60 years; M:F = 2:1 ● weight loss, nausea, malaise ● dull pain in flank, back, abdomen (90%) ● renal insufficiency (50-60%) ● hypertension ● leg edema, fever, hydrocele (10%) ● claudication (occasionally)
Location: plaque typically begins around aortic bifurcation extending cephalad to renal hilum / surrounding kidney; rarely extends below pelvic rim, but may extend caudad to bladder + rectosigmoid IVP Classic TRIAD: (1)ureterectasis above L4/5 (interference with peristalsis) (2)medial deviation of ureters in middle third, typically bilateral (3)gradual tapering of ureter (extrinsic compression) √ usually mild pyelocaliectasis US: √ hypoechoic homogeneous mass in para-aortic region / perinephric space CT: √ periaortic mass of attenuation similar to muscle √ may show contrast enhancement (active inflammation) MR: √ low to medium homogeneous signal intensity on T1WI √ heterogeneous high signal intensity on T2WI (with malignancy / associated inflammatory edema) √ low signal intensity on T2WI (in dense fibrotic plaque) NUC: √ gallium uptake during active inflammation
*DDx:*lymphoma, retroperitoneal adenopathy *Rx:*(1)Withdrawal of possible causative agent (2)Interventional relief of obstruction (3)Corticosteroids

Notes:

RETROPERITONEAL LEIOMYOSARCOMA
*Incidence:*2nd most common primary retroperitoneal malignancy (after liposarcoma) *Origin:* (a)retroperitoneal space without attachment to organs (b)wall of inferior vena cava *Age:*5th-6th decade; M:F = 1:6 ● abdominal mass, pain, weight loss, nausea, vomiting ● abdominal distension, change in defecation habits, leg edema, back / radicular pain, frequency of urination ● hemoperitoneum, GI bleeding, dystocia, paraplegia Metastases: frequently hematogenous, less commonly lymphatic dissemination (a)common sites: liver, lung, brain, peritoneum (b)rare sites: skin, soft tissue, bone, kidney, omentum ◊Distant metastases present at time of diagnosis in 40%
A.EXTRAVASCULAR LEIOMYOSARCOMA (62%) *Path:*extraluminal (= completely extravascular) large tumor with extensive necrosis IVP: √ large soft-tissue mass with (a)displacement of kidney + ureter (b)gas-containing ascending / descending colon √ well-defined fat plane between mass and kidney √ obstruction of kidney (ureteral involvement) √ usually not calcified US: √ solid mass isoechoic to liver / rarely hyperechoic √ complex mass with cystic spaces + irregular walls CT: √ lobulated mass often >10 cm in size √ large cystic areas of tumor necrosis in center of mass √ areas of high attenuation with recent hemorrhage MR: √ intermediate intensity on T1WI with low-intensity areas of necrosis √ inhomogeneous intermediate intensity on T2WI Angio: √ hypervascular tumor with blood supply from lumbar, celiac, mesenteric, renal arteries √ avascular center surrounded by thick hypervascular rind B.INTRAVASCULAR LEIOMYOSARCOMA (6%) *Path:*intraluminal (= completely intravascular) polypoid mass firmly attached to vessel wall Location:between diaphragm + renal veins, may extend along entire length of IVC + into heart √ small solid mass within IVC √ gradually dilatation / obstruction of IVC √ intratumoral vascularity confirmed by Doppler √ irregular enhancement (CT bolus injection) *Cx:* (1)Budd-Chiari syndrome (extension into hepatic veins) (2)Nephrotic syndrome (extension into renal veins) (3)Edema of lower extremities (extension into lower IVC without adequate collateralization) (4)Tumor embolus to lung C.EXTRA- AND INTRAVASCULAR LEIOMYOSARCOMA (33%) √ solid / necrotic extraluminal mass not originating from a retroperitoneal organ with contiguous intravascular enhancing component (PATHOGNOMONIC) D.INTRAMURAL LEIOMYOSARCOMA (extremely rare)
DDx:(1)Liposarcoma (fat content) (2)Malignant fibrous histiocytoma (not as necrotic) (3)Lymphoma (nonnecrotic, tends to envelop IVC + aorta) (4)Primary adrenal tumor (5)IVC thrombus (no luminal enlargement, no neovascularity)
Rx:(1) Complete excision (resectable in 10-75%) (2)Partial resection (reduction in tumor size) (3)Adjuvant chemotherapy / radiotherapy
*Prognosis:*local recurrence in 40-70%; death within 5 years in 80-87% with extraluminal tumors

Notes:

RETROPERITONEAL LIPOSARCOMA
=slow-growing tumor that displaces rather than infiltrates surrounding tissue and rarely metastasizes *Incidence:*2nd most common primary retroperitoneal tumor (after malignant fibrous histiocytoma), 95% of all fatty retroperitoneal tumors *Histo:* rarely arising from lipoma (a)myxoid form (most common): varying degrees of mucinous + fibrous tissue + relatively little lipid =intermediate differentiation √ radiodensity between water + muscle (b)lipogenic form: malignant lipoblasts with large amounts of lipid + scanty myxoid matrix =well-differentiated √ radiodensity of fat (c)pleomorphic type (least common): marked cellular pleomorphism, paucity of lipid + mucin =highly undifferentiated √ radiodensity of muscle *Age:*most commonly 40-60 years; M > F Sites:lower extremity (45%), abdominal cavity + retroperitoneum (14%), trunk (14%), upper extremity (7.6%), head & neck (6.5%), miscellaneous (13.5 %) CT: √ solid pattern: inhomogeneous poorly marginated infiltrating mass with contrast enhancement √ mixed pattern: focal fatty areas (-40 to -20 HU) + areas of higher density (+ 20 HU) √ pseudocystic pattern: water-density mass (averaging of fatty + solid connective-tissue elements) √ calcifications in up to 12% Angio: √ hypovascular without vessel dilatation / capillary staining / laking
*Prognosis:*most radiosensitive of soft-tissue sarcomas; 32% overall 5-year survival
*DDx:*malignant fibrous histiocytoma, leiomyosarcoma, desmoid tumor

Notes:

RHABDOMYOSARCOMA, GENITOURINARY
Frequency: 4-8% of all malignant solid tumors in children <15 years of age (ranking 4th after CNS neoplasm, neuroblastoma, Wilms tumor); 10-25% of all sarcomas; annual incidence of 4.5:1,000,000 white + 1.3:1,000,000 black children *Age:*mean age of 7 years; white:black = 3:1; M:F = 6:4 *Path:*firm fleshy lobulated mass with infiltrative margin / well-defined pseudocapsule; composed of smooth grapelike clusters if intraluminal (= sarcoma botryoides) *Origin:*mesenchyme of the urogenital ridge *Histo (Horn & Enterline):* (a)embryonal (56%) (b) botryoid = "grapelike" (5%) = subtype of embryonal rhabdomyosarcoma (c)alveolar (20%): worst prognosis (d)pleomorphic (1%): mostly in adults DDx:primitive neuroectodermal tumor, extraosseous Ewing sarcoma, synovial cell sarcoma, fibrosarcoma, alveolar soft part sarcoma, hemangiopericytoma, undifferentiated sarcoma, neuroblastoma Metastases:lung, cortical bone, lymph nodes > bone marrow, liver ◊Metastases in 10-20% at time of diagnosis! √ nonspecific imaging features: √ homogeneous echogenicity similar to muscle ± hypoechoic areas (hemorrhage / necrosis) √ hyperemia with high diastolic flow component √ bulky pelvic mass of heterogeneous attenuation √ hypointense on T1WI + hyperintense on T2WI with heterogeneous enhancement √ diffuse tumor vascularity on angio *Prognosis:* (a)14-35% 5-year survival with radical surgery (b)60-90% 3-year survival with chemotherapy added ◊Local recurrence is common!

Bladder-prostate Rhabdomyosarcoma Rhabdomyosarcoma Of Female Genital Tract Paratesticular Rhabdomyosarcoma

Notes:

Bladder-prostate Rhabdomyosarcoma *Age:*in first 3 years of life Location:trigone of urinary bladder / prostate (tumor infiltrating both) ● abdominal pain + distension (from bladder outlet obstruction) ● urinary frequency + dysuria (from urinary tract infection) ● palpable bladder ● hematuria (unusual late manifestation) ● strangury (= painful urge to void without success) √ polyploid intraluminal tumor mass √ elevation of bladder floor with obstruction of bladder neck + large postvoid residual √ ± invasion of periurethral / perivesical tissues √ retroperitoneal lymph node enlargement *DDx:*polyp, hemangioma, ectopic ureterocele, cystitis

Notes:

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Rhabdomyosarcoma Of Female Genital Tract Location:vulva / vagina (infancy), cervix (reproductive years), uterine corpus (postmenopausal) ● vulvar / perineal / vaginal mass ● vaginal bleeding / discharge / protruding grapelike mass *DDx:*polyp, urethral prolapse, hydrometrocolpos, neoplasm

Notes:

Paratesticular Rhabdomyosarcoma *Age:*childhood, 2nd age peak in adolescence Location:spermatic cord, testis, penis, epididymis ● painless scrotal swelling ● palpable nontransilluminating intrascrotal tumor ● bulky abdominal (lymphadenopathy) ● displacement / compression / infiltration of adjacent testis *Prognosis:*73-89% 3-year survival rate *DDx:*hydrocele, epididymitis, testicular neoplasm

Notes:

SCHISTOSOMIASIS
=BILHARZIASIS *Organism:*trematodes of species: S. haematobium (GU tract) >95%; S. mansoni, S. japonicum (GI tract) <5% *Life cycle:* female parasite discharges eggs into vesicular venules; eggs erode bladder mucosa, are excreted with urine + feces, and hatch in fresh water into larval miracidia; larvae invade snail (= intermediate host) of genus Bulinus, Biomphalaria, Oncomelania; resulting daughter sporocytes develop into cercariae and pass into surrounding body of water; penetrate human skin (usually foot) + pass into lymphatics; schistosome settles in portal veins + migrate into pelvic venous plexus *Incidence:*8% of worlds population; 25% in Africa (endemic in South Africa, Egypt, Nigeria, Tanzania, Zimbabwe); endemic in Puerto Rico @Urinary tract ● frequency, urgency, dysuria ● hematuria, albuminuria (most common) ● dull flank pain (from hydronephrosis) ● index of infectious severity = urine egg count Location:lower ureters + bladder √ bladder wall calcifications (in 4-56%): linear / coarse / floccular, beginning at base, parallel to upper aspect of pubic bone, involving all wall layers √ vesical calculi (in 39%), distal ureteral calcification (in 34%), honeycombed calcification of seminal vesicles √ striation of renal pelvis + proximal ureter in 21% (DDx: normal in 3%, other urinary tract infection, vesicoureteric reflux √ ureterectasis (focal egg deposition leads to peristaltic disorganization) √ ureteral strictures in distal third (in 8%, L > R), most commonly in intravesical portion with cobra-head configuration = pseudoureterocele); Makar stricture = focal stricture at L3 √ multiple inflammatory pseudopolyps in ureter secondary to granulomas (= bilharziomas) √ ureteritis cystica √ ureterolithiasis / ureteritis calcinosa (= punctate / linear calcifications) √ vesicoureteral reflux √ polypoid filling defects + mucosal irregularities in urinary bladder (pseudotubercles, papillomas) √ thick-walled fibrotic "flat-topped" bladder with high insertion of ureters √ reduced bladder capacity with significant postvoid residual (fibrotic stage) √ urethral stricture with perineal fistulas *Cx:*Squamous cell carcinoma of bladder *Age:*30-50 years (exposed early in childhood with 20-to-30-year latency period) Location:posterior bladder wall, rarely trigone √ irregular filling defect √ discontinuous calcifications @GI tract √ portal hypertension (ova migrating into portal venous system incite fibrosing granulomatous reaction within presinusoidal portal veins) √ esophageal varices (from portal hypertension) √ polypoid calcifying bowel lesions (from eggs of S. mansoni trapped in bowel wall + inciting granulomatous reaction) @Chest √ enlargement of RV + pulmonary artery + azygos vein (from portal hypertension) √ diffuse granulomatous lung lesions *Rx:*praziquantel

Notes:

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SCROTAL ABSCESS
Etiology: (1)Complication of epididymo-orchitis (often in diabetics), missed testicular torsion, gangrenous tumor, infected hematoma, primary pyogenic orchitis (2)Systemic infection: mumps, smallpox, scarlet fever, influenza, typhoid, syphilis, TB (3)Septic dissemination from: sinusitis, osteomyelitis, cholecystitis, appendicitis NUC: ↓ marked increase in perfusion, hot hemiscrotum with photon-deficient area representing the abscess on Tc-99m pertechnetate scan (DDx: chronic torsion) ↓ increased scrotal uptake with leukocyte imaging US: ↓ hypoechoic / complex fluid collection with low-level echoes (differentiation of intra- from extratesticular abscess location possible) *Cx:*(1)Pyocele (2)Fistulous tract to skin

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Notes:

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SEMINAL VESICLE CYST

1.ACQUIRED SEMINAL VESICLE CYST 2.CONGENITAL SEMINAL VESICLE CYST *Associated with:*anomalies of ipsilateral mesonephric duct: (1)Ectopic insertion of ipsilateral ureter (92%) into bladder neck / posterior prostatic urethra / ejaculatory duct / seminal vesicle (2)Ipsilateral renal dysgenesis (80%) (3)Duplication of collecting system (8%) *Symptomatic age:*21-41 years ● abdominal / flank / pelvic / perineal pain exacerbated by ejaculation ● dysuria, frequent urination ● epididymitis in prepubertal boy ● recurrent urinary tract infection

√ cystic mass posterior to urinary bladder (DDx: müllerian duct cyst) √ dilated ejaculatory duct

Notes:

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SINUS LIPOMATOSIS
=PERIPELVIC LIPOMATOSIS =PELVIC FIBROLIPOMATOSIS =PERIPELVIC FAT PROLIFERATION *Etiology:* (1)Normal increase with aging and obesity (2)Vicarious proliferation of sinus fat with destruction / atrophy of kidney (= replacement lipomatosis) (3)Extravasation of urine leading to proliferation of fatty granulation tissue (4)Normal variant *Age:*6th-7th decade √ kidney may be enlarged √ elongated "spiderlike / trumpetlike" pelvicaliceal system √ infundibula arranged in "spoke-wheel" pattern √ parenchymal thickness diminished with underlying disease √ occasionally focal fat deposit with localized deformity of collecting system Plain film: √ diminished sinus density CT: √ unequivocal fat values US: √ echodense / patchy hypoechoic sinus complex

Notes:

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SQUAMOUS CELL CARCINOMA OF KIDNEY

*Incidence:*15% of all urothelial tumors *Path:*flat ulcerating mass + extensive induration *Associated with:*previous chronic renal infection + calculi (25-60%) √ stricture that may simulate extrinsic cause √ ureteropelvic junction obstruction (common) √ presence of faceted calculi √ thickening of pelvicaliceal wall (with superficial spread over large areas) √ arterial encasement + occlusion + neovascularity √ enlarged pelvic + ureteric arteries √ occlusion of renal vein / branches (41%) *Prognosis:*poor due to early metastases

Notes:

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SUPERNUMERARY KIDNEY
=aberrant division of nephrogenic cord into two metanephric tails (rare) *Associated with:*horseshoe kidney, vaginal atresia, duplicated female urethra, duplicated penis
Location: most commonly on left side of abdomen caudal to normal kidney ↓ supernumerary ureter may insert into ipsilateral kidney / directly into bladder / ectopic site
*Cx:*hydronephrosis, pyonephrosis, pyelonephritis, cysts, calculi, carcinoma, papillary cystadenoma, Wilms tumor

Notes:

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TESTICULAR INFARCTION

*Etiology:*torsion, trauma, leukemia, bacterial endocarditis, polyarteritis nodosa, Henoch-Schönlein purpura √ diffusely hypoechoic small testis √ hyperechoic regions (hemorrhage / fibrosis)

Notes:

TESTICULAR MICROLITHIASIS
*Etiology:*formation of microliths from degenerating cells in the seminiferous tubules + absence of phagocytosis by Sertoli cells *Prevalence:*0.05-0.60% *May be associated with:* Klinefelter syndrome, cryptorchidism, testicular infarcts, granulomas, subfertility, infertility, testicular germ cell tumor (40%), male pseudohermaphroditism, Down syndrome, pulmonary alveolar microlithiasis ● asymptomatic, uncommon incidental finding √ 1- to 2-mm hyperechoic foci scattered throughout the testicular parenchyma (PATHOGNOMONIC) *Cx:*concurrent germ cell tumor in 40% *DDx:*postinflammatory changes, scars, granulomatous changes, benign adenomatoid tumor, hemorrhage with infarction, large-cell calcifying Sertoli cell tumor

Notes:

TESTICULAR RUPTURE

◊Testicular rupture is indication for immediate surgical intervention! *Cause:*scrotal trauma *Salvageability:* 80-90% if surgical repair occurs <72 hours after trauma; 30-55% if surgical repair occurs >72 hours after trauma √ areas of decreased / increased echogenicity (hemorrhage ± necrosis) √ loss of testicular outline √ thickened scrotal wall (= hematoma) √ visualization of fracture plane √ hematocele, may show thickening + calcification of tunica vaginalis if chronic √ uriniferous hydrocele from perforated bulbous urethra √ avascular region on color duplex *Cx:*torsion (due to stimulation of a forceful cremasteric contraction) *DDx:*laceration, contusion, hemorrhage

Notes:

TESTICULAR TORSION

=SPERMATIC CORD TORSION Most common scrotal disorder in children, 20% of acute scrotal pathology *Incidence:*1:160, 10-fold risk in undescended testis compared with normal annual incidence of 1:4,000 males *Etiology:* (1)"Bell and clapper" deformity = high insertion of tunica vaginalis on spermatic cord (2)Abnormally loose mesorchium between testis + epididymis (3)Extravaginal torsion involving testis + tunica vaginalis due to loose attachment of testicular tunics to scrotum during in utero + perinatal period *Peak age:*newborn period + puberty (13-16 years); <20 years in 74-85%; >21 years in 26%; >30 years in 9%

● sudden severe pain in 100% (frequently at night) ● negative urine analysis (98%) ● history of similar episode in same / contralateral testis (42%) ● nausea + vomiting (50%) ● scrotal swelling + tenderness (42%) ● leukocytosis (32%) ● low-grade fever (20%) ● history of trauma / extreme exertion (13%) Location:in 5% bilateral (anomalous suspension of contralateral testis found in 50-80%)

Salvage rate: versus time interval between onset of pain and surgery 80-100%<6 hours 76%6-12 hours 20%12-24 hours near 0%>24 hours spontaneous detorsion in 7% ◊Irreversible ischemic damage in only 3-6 hours! *Cx:*testicular atrophy (in 33-45%)

Acute Testicular Torsion Subacute Testicular Torsion Chronic Testicular Torsion

Notes:

Acute Testicular Torsion ● 70% of patients present within first 6 hours from onset of pain US (80-90% sensitivity): √ normal grey-scale appearance (within 6 hours) √ testicular + epididymal enlargement with decreased echogenicity (within 8-24 hours) √ increase in size of spermatic cord √ scrotal skin thickening √ hydrocele (occasionally) √ loss of spermatic cord Doppler signal (sensitivity 44%, specificity 67%) Color duplex (86% sensitive, 100% specific, 97% accurate): √ absence of testicular + epididymal flow (DDx: global testicular infarction) false-negative:torsion-detorsion sequence, incomplete torsion <360 degrees *Degree of torsion and blood flow:* ● testis usually turns medially up to 1,080 degrees √ diminished blood flow in <180°-torsion at 1 hour √ absent blood flow in any degree of torsion >4 hours √ hyperemia after spontaneous detorsion
NUC (98% accuracy): *Dose:*5-15 mCi Tc-99m pertechnetate *Imaging:*at 2- to 5-second intervals for 1 minute (vascular phase); at 5-minute intervals for 20 minutes (tissue phase) √ decreased perfusion / occasionally normal √ nubbin sign = bump of activity extending medially from iliac artery denoting reactive increased blood flow in spermatic cord with abrupt termination √ rounded cold area replacing testis (requires knowledge of side + location of painful testis)

Notes:

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Subacute Testicular Torsion =MISSED TESTICULAR TORSION ● symptoms present for >24 hours + less than 10 days US: √ enlarged / normal-sized testis with heterogeneous texture √ increased peritesticular flow without parenchymal blood flow NUC: √ normal NUC angiogram / nubbin sign √ "doughnut" sign = decreased testicular activity with rim hyperemia of dartos perfusion MRI: √ enlarged spermatic cord without increase in vascularity √ whirlpool pattern (twisting of spermatic cord) √ torsion knot = low-signal-intensity focus at point of twist (displacement of free protons from epicenter of twist)

Notes:

Chronic Testicular Torsion √ small homogeneously hypoechoic testis √ enlarged echogenic epididymis

Notes:

TESTICULAR TUMOR
Most common neoplasm in males between ages 25-34 years; 1-2% of all cancers in males; 4-6% of all male genitourinary tumors; 1.5% of all childhood malignancies; 4th most common cause of death from malignancy between ages 15-34 years (12%) *Incidence per year:*3-5:100,000 *Peak age:*25-35 years; prior to puberty: yolk sac tumor + teratoma *Risk factors:* (a)Caucasian race, Jewish religion (b)family history of testicular cancer, previous testicular neoplasm (c)testicular maldescent / atrophy (10 x risk); abdominal site affected in 5%, inguinal site affected in 1.25%
● chronic pain, "heaviness" ● acute scrotal pain (10%, from intratumoral hemorrhage) ● enlarging testis, mass ● gynecomastia, virilization Location:mostly unilateral; contralateral tumor develops eventually in 8% *Staging:* StageIlimited to testis + spermatic cord StageIImetastases to lymph nodes below diaphragm II Anonpalpable II Bbulky mass StageIIImetastases to lymph nodes above diaphragm III Aconfined to lymphatic system III Bextranodal metastases
*Metastases:*at presentation in 4-14% to lung, liver, bones, brain, lymph nodes *Tumor activity:*monitored by levels of a-fetoprotein + b-HCG
Color duplex: √ tumor <1.5 cm is hypovascular in 86%, >1.6 cm hypervascular in 95% (DDx: orchitis associated with epididymal hyperemia) √ distortion of vessels
Prognosis:>93% 5-year survival rate for stage I; 85-90% 5-year survival rate for stage II; complete remission under chemotherapy in 65-75%; relapse in 10-20% within 18 months

Germ Cell Tumors (95%) Stromal Cell Tumors = Interstitial Cell Tumors Metastases To Testis (0.06%) Lymphoma / Leukemia Of Testis Burned-out Tumor Of Testis Second Testicular Tumor

Notes:

Germ Cell Tumors (95%) (a)one histologic type in 65% (b)mixed lesion in 35-40% 1.Teratocarcinoma (= teratoma + embryonal cell carcinoma) 2nd most common after seminoma, may occasionally undergo spontaneous regression 2.Embryonal cell carcinoma + seminoma 3.Seminoma + teratoma
mnemonic:"YES CT" **Y**olk sac tumor **E**mbryonal cell carcinoma **S**eminoma **C**horiocarcinoma **T**eratoma
A.**SEMINOMA** (40-50%) Most common tumor in undescended testis *Peak age:* 30-40 years *Spread:*in 25% metastasized on initial presentation, pulmonary metastases develop in 19% ● serum a-fetoprotein usually normal ● b-HCG elevation in 10-15% √ usually uniformly hypoechoic + confined within tunica albuginea √ may be multifocal *Rx:*sensitive to radiation + chemotherapy *Prognosis:*10-year survival rate of 75-85%
B.NONSEMINOMATOUS TUMOR *Age:*20-30 years 1.**Embryonal cell carcinoma** (20-25%) Most common component of mixed testicular tumors; often associated with teratoma *Peak age:*2nd-3rd decade and <2 years *Spread:*most aggressive testicular tumor, visceral metastases ● ± a-fetoprotein elevation √ hypoechoic mass with areas of increased echogenicity + cystic areas (hemorrhage / necrosis) √ may show invasion of tunica albuginea *Prognosis:*30-35% 5-year survival rate
2.**Teratoma** (4-10%) 2nd most common testicular tumor in young boys *Prevalence:*1:1,000,000 *Histo:*consists of elements from more than one germ cell layer (keratin, muscle, bone, cartilage, hair, mucous glands, neural tissue) (a)mature (b)immature *Age:*within first 4 years of life; benign in children; may transform into malignancy in adulthood ● serum a-fetoprotein may be elevated √ mixed echotexture with sonolucent + highly echogenic components (markedly heterogeneous) *Prognosis:*metastases to lymph nodes, bone, liver in 30% within 5 years
3.**Choriocarcinoma** (1-3%) *Peak age:* 20-30 years *Spread:*may rapidly metastasize without evidence of choriocarcinoma in primary lesion, pulmonary metastases develop in 81% ● serum b-HCG always elevated (may produce gynecomastia) √ mixed echotexture (hemorrhage, necrosis, calcifications) √ indistinct margins of pulmonary metastases (due to hemorrhage) *Prognosis:* nearly 0% 5-year survival rate
4.**Yolk sac tumor = endodermal sinus tumor** Equivalent to endodermal sinus tumor of ovary *Age:*predominantly <3 years ● serum a-fetoprotein always elevated √ pulmonary metastases
5.**Epidermoid cyst of testicle** (<1%) ="monodermal dermoid" = KERATIN CYST =benign teratoma with only ectodermal components *Age:*20-40 years; primarily in Whites *Histo:*cyst contains keratin, wall composed of fibrous tissue + lined by squamous epithelium √ sharply circumscribed encapsulated round lesion of 0.5-10.5 cm in diameter √ hyperechoic fibrous cyst wall ± shadowing from calcifications √ hypoechoic cyst contents (= laminated keratin debris) √ may have echogenic center (= calcification of intraluminal content) MRI: √ target appearance with fibrous capsule of low signal intensity on T1WI + T2WI, cyst content of high signal intensity on T1WI + T2WI, central calcification with center of low signal intensity

Notes:

Stromal Cell Tumors = Interstitial Cell Tumors (3% of all testicular tumors, 10-30% during childhood) ● precocious virilism (children) ● gynecomastia (adults) ● loss of libido (adults) ● impotence (adults) *Rx:*conservative resection under ultrasound guidance 1.**Leydig cell tumor** derived from interstitial cells forming the fibrovascular stroma; benign:malignant = 9:1 *Peak age:*3-6 years ● may secrete androgens or estrogens ● gynecomastia (in almost 50%) √ usually hypoechoic nodule 2.**Sertoli cell tumor** derived from Sertoli cells of seminiferous tubules, benign:malignant = 9:1 *Peak age:*1st year of life ● may secrete estrogens √ usually hypoechoic nodule √ punctate calcifications in large-cell calcifying Sertoli cell tumors 3.Gonadoblastoma = primitive gonadal stroma tumor (exceedingly rare) ● dysgenetic gonads + abnormal karyotype

Notes:

Metastases To Testis (0.06%) (a)in adults: prostate > lung > kidney > GI tract, bladder, thyroid, melanoma ◊More common than germ cell tumors in males >50 years of age! (b)in children: neuroblastoma, Wilms tumor, rhabdomyosarcoma √ often multiple and bilateral √ mostly hypoechoic, occasionally echogenic masses

Notes:

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Lymphoma / Leukemia Of Testis *Incidence:*6.7% of all testicular tumors *Lymphoma:*most common testicular tumor in men > age 50; bilateral in 40% *Leukemia:*60-92% incidence of testicular involvement on autopsy, 8-16% on clinical examination during therapy, up to 41% on clinical examination after therapy◊Occult testicular tumor often found in patients in bone marrow remission ("gonadal barrier" to chemotherapy) √ uni- / bilateral diffuse / focal process of decreased echogenicity

Notes:

Burned-out Tumor Of Testis = AZZOPARDI TUMOR = spontaneous regression of testicular malignancy (teratocarcinoma) √ highly echogenic focal lesion ± shadowing (= scarred tumor residue) √ metastases to retroperitoneum, mediastinum, cervical / axillary / supraclavicular lymph nodes, lung, liver

Notes:

Second Testicular Tumor *Risk for second tumor in cryptorchidism:* 15% for inguinal, 30% for abdominal location *Risk for second contralateral tumor:* 500-1,000 x ; bilaterality in 1.1-4.4%; ◊Development interval between 1st + 2nd tumor: 4 months to 25 years ◊Detected in 47% by 2 years; in 60% by 5 years, in 75% by 10 years ◊ Synchronous contralateral tumor in 1-3% *US:*a testicular abnormality is malignant in only 50%!

Notes:

TRANSITIONAL CELL CARCINOMA

Prevalence:85% of all urothelial tumors / primary renal pelvic tumors; 7% of all renal neoplasms *Mean age:*64 years; M:F = 3:1 *Pathogenesis:*chemical carcinogens act locally on epithelium (= field of change), action enhanced by length of contact time (eg, stasis / diverticulum) *Risk factors:* (1)tobacco (2-3 x) (2)aniline dye, benzidine, aromatic amines, azo dyes in textile, rubber, printing, plastic manufacturing (lag time of 10 years) (3)cyclophosphamide therapy (lag time of 6.5 years) (4)analgesic abuse (8 x increase): phenacetin (5)Balkan nephritis (= progressive renal failure + development of bilateral and multiple tumors) (6)recurrent / chronic urinary tract infection *Classification:* (a)exophytic papillary lesion (85%) = frondlike structure with central fibrovascular core lined by epithelial layer -broad based -pedunculated (b)infiltrating: usually higher grade + less common (c)carcinoma in situ *Grade:*usually correlates with stage 1=cells slightly anaplastic 2=intermediate features 3=marked cellular pleomorphism ● frank / microscopic hematuria (72%) ● dull flank pain (22%) ● acute renal colic (due to obstruction) Location:bladder 30-50 x more common than upper urinary tract
SYNCHRONOUS TCC (a)both renal pelves (in 1-2%) (b)both ureters (in 2-9%) (c)bladder-in 24% of primary renal pelvic involvement -in 39% of primary ureteral involvement -in 2% of primary bladder involvement

Renal And Ureteral TCC Bladder TCC

Notes:

Renal And Ureteral TCC *Staging:* TNMAJCCDescription Tis0in situ lesion Ta...noninvasive papillary carcinoma T1Iinvasion of subepithelial connective tissue T2IIconfined to muscularis layer T3IIIinvasion of renal parenchyma / peripelvic soft tissues T4IVextension beyond renal capsule
METACHRONOUS TCC IN UPPER TRACT (a)in 12% of pelvic + ureteral primaries (in 25 months) (b)in 4% of bladder primaries (2/3 within 2 years, up to 20 years later)
@Kidney Site:extrarenal part of renal pelvis > infundibulocaliceal region IVP: √ single / multiple filling defects in renal pelvis (35%) √ "stipple sign" = contrast material trapped in interstices (DDx: blood clot, fungus ball) √ dilated calyx with filling defect (26%) due to partial / complete obstruction of infundibulum √ "phantom calyx" = failure to opacify from obstruction √ ± focal delayed increasingly dense nephrogram √ "oncocalyx" = caliceal distension with tumor √ caliceal amputation (19%) √ absent / decreased excretion with renal atrophy (13%) due to long-standing obstruction of ureteropelvic junction √ hydronephrosis with renal enlargement (6%) due to tumor obstruction of ureteropelvic junction US: √ bulky hypoechoic (similar to renal parenchyma) mass lesion √ splitting / separation of central renal sinus complex √ infiltrative without bulge of renal contour √ ± focal caliceal dilatation CT (52% accuracy due to overstaging): √ sessile filling defect in opacified collecting system √ thickening + induration of pelvicaliceal wall √ central solid mass in renal pelvis expanding centrifugally √ compression of renal sinus fat √ invasion of renal parenchyma (infiltrating growth pattern) with preservation of renal contour √ coarse punctate calcific deposits (0.7-6.7%) may mimic urinary calculi √ variable enhancement of tumor
@Ureter Site:lower 1/3 (70%), mid 1/3 (15%), upper 1/3 (15%) IVP: √ nonfunctioning kidney in advanced tumor (46%) √ hydronephrosis ± hydroureter (34%) √ single / multiple ureteral filling defects (19%) √ irregular narrowing of ureteral lumen Retrograde: √ "champagne glass" / "goblet sign" = focal expansion of ureter around + distal to mass (probably secondary to to-and-fro peristalsis of mass) √ "Bergman sign" = "catheter-coiling sign" = coiling of catheter on retrograde catheterization below the mass CT: √ intraluminal soft-tissue mass √ eccentric / circumferential thickening of ureteral wall *Dx:*cytologic analysis of urine (selective lavage, ureteral urine collection, brush biopsy, ureteroscopy *DDx:*papilloma (benign lesion, fronds lined by normal epithelium)

Notes:

Bladder TCC *Incidence:*5% of all new malignant neoplasms; most common tumor of genitourinary tract; 2% of all cancer deaths in United States *Staging* T 1=A=lesions involving mucosa + submucosa T 2=B1=invasion of superficial muscle layer T 3a=B2=invasion of deep muscular wall T 3b=C=invasion of perivesical fat T 4a=D1=extension to perivesical organs (seminal vesicles, prostate, rectum) T4b=invasion of pelvic / abdominal wall D2=distant metastases Staging accuracy:50% clinically; 32-80% for CT; 73% for MRI Overstaging due to:edema following endoscopy /endoscopic resection, fibrosis from radiation therapy *Histo:*80% low-stage superficial papillary neoplasm, (multifocal in 1/3), becoming invasive in 10-20%; 20% invasive (almost always solitary) Site:lateral wall of bladder, bladder diverticulum (in 0.8-10.8%)
METACHRONOUS TCC OF BLADDER (a)in 23-40% of primary renal TCC after 15-48 months (b)in 20-50% of primary ureteral TCC after 10-24 months
IVP (70% accuracy rate): √ irregular filling defect with broad base and fronds (DDx: rectal gas marginated by Simpsons white line) √ <1% calcified CT / US: √ focal wall thickening √ papillary mass protruding into lumen MR (staging modality of choice): √ TCC isointense to bladder muscle on T1WI + hyperintense on T2WI √ enhancement differentiates between early enhancing mucosa, submucosa, tumor + nonenhancing muscle

Notes:

TUBERCULOSIS
Urogenital tract is the second most common site after lung; almost always affects the kidney first as a hematogenous focus from lung / bone / GI tract *Age:*usually before age 50; M > F ● gross / microscopic hematuria ● "sterile" pyuria ● frequency, urgency, dysuria ● history of previous clinical TB (25%)
@EXTRARENAL SIGNS ON ABDOMINAL PLAIN FILM √ osseous / paraspinous changes of TB (discitis + psoas abscess) √ calcified granulomas in liver, spleen, lymph nodes, adrenals
@RENAL MANIFESTATION ◊Renal TB in 4-8% of patients with pulmonary TB! ◊Radiographic evidence of pulmonary TB in <50% of patients with renal TB (only 5% have active cavitary TB)! Location:unilateral renal involvement in 75% √ displacement of collecting system secondary to tuberculoma (initial infection) √ dystrophic amorphous calcifications in tuberculomas of renal parenchyma (in 25%) √ kidney enlarged (early) / small (late) / normal √ "smudged" papillae = irregularities of surface of papillae √ "moth-eaten" calyx = caliceal erosion (early change) √ irregular tract formations from calyx into papilla √ large irregular cavities with extensive destruction = papillary necrosis √ dilated calices (hydrocalicosis) often with sharply defined circumferential narrowings (infundibular strictures) at one / several sites (most common finding) √ renal calculi (in 10%) √ "putty kidney" = tuberculous pyonephrosis from ureteral stricture √ autonephrectomy = small shrunken scarred nonfunctioning kidney ± dystrophic calcifications √ infection may extend into peri- / pararenal space + psoas
@URETERAL MANIFESTATION Always with evidence of renal involvement as it spreads from kidney Location:either end of ureter (most commonly distal 1/3), usually asymmetric, may be unilateral √ ureteral filling defects (= mucosal granulomas) √ "saw-tooth ureter" = irregular jagged contour secondary to dilatation + multiple small mucosal ulcerations + wall edema (early changes) √ strictures (late changes): "beaded ureter"=alternating areas of strictures + dilatations "corkscrew ureter"=marked tortuosity with strictures + dilatations "pipestem ureter"=rigid aperistaltic short thick and straight ureter √ vesicoureteral reflux through "fixed" patulous orifice √ ureteral calcifications uncommon (usually in distal portion)
@BLADDER MANIFESTATION Infection from renal source causing interstitial cystitis √ thickened bladder wall (= muscle hypertrophy + inflammatory tuberculomas) √ bladder wall ulcerations √ "shrunken bladder" = scarred bladder with diminished capacity √ bladder wall calcifications (rare) *Cx:*fistula / sinus tract
@SEMINAL VESICULAR + EPIDIDYMAL MANIFESTATION Hematogenous infection (NOT ascending) √ calcifications in 10% (diabetes more common cause)
*DDx:*brucellosis, fungal infections (identical picture)

Notes:

UNICALICEAL (UNIPAPILLARY) KIDNEY
*Path:*OLIGOMEGANEPHRONIA = reduced number of nephrons and enlargement of glomeruli *Associated with:*absence of contralateral kidney, other anomalies ● hypertension ● proteinuria ● azotemia

Notes:

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URACHAL ANOMALIES
urachus = median umbilical ligament = thick fibrous cord as the remnant of the allantois (= endodermal outgrowth from yolk sac into stalk) which regresses at 5th month of development
*Cx:*infection (23%), intestinal obstruction, hemorrhage into cyst, peritonitis from rupture, malignant degeneration

Alternating Sinus Patent Urachus Urachal Cyst (30%) Urachal Diverticulum (3%) Urachal Sinus

Notes:

Patent Urachus =fistula between bladder and umbilicus *Incidence:*1:200,000 live births ● urine draining from umbilicus

Notes:

Urachal Cyst (30%) =gradually enlarging cyst due to closure of both ends of urachus *Incidence:*1:5,000 (at autopsy) ● asymptomatic in children unless rupture occurs ● symptomatic in adults due to enlargement / infection √ cystic extraperitoneal mass

Notes:

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Urachal Sinus =urachus patent only at umbilicus *Associated with:*urachal cyst ● umbilical mass / inflammation ± drainage √ thickened tubular structure with echogenic center

Notes:

URACHAL CARCINOMA
=rare tumor arising from the urachus (vestigial remnant of cloaca + allantois) within space of Retzius *Incidence:*0.2-0.34% of all bladder cancers; 20-40% of all primary bladder adenocarcinomas *Histo:* (a)adenocarcinoma (84%) from malignant transformation of columnar metaplasia, in 75% mucin producing (b)TCC (3%), sarcoma, squamous cell carcinoma ◊75% of urachal neoplasms in patients <20 years of age are sarcomas! *Age:*41-70 years; M:F = 3:1 ● suprapubic mass, abdominal pain ● hematuria (71%) ● discharge of blood, pus, mucus from umbilicus ● irritative voiding symptoms ● mucous micturition (25%)
Stage: Icancer limited to urachus IIinvasion limited to urachus III Alocal invasion of bladder III Binvasion of abdominal wall III Cinvasion of peritoneum III Dinvasion of other viscera IV Ametastases to local lymph nodes IV Bdistant metastases
Location:supravesical, midline, anterior (80%), in space of Retzius (bounded by transversalis fascia ventrally + peritoneum dorsally) √ mass anterosuperior to vesical dome with predominantly muscular / extravesical involvement √ invasion of bladder dome (88%) √ low-attenuation mass in 60% (mucin) √ often peripheral psammomatous PATHOGNOMONIC calcifications (70%) √ markedly increased signal intensity on T2WI *Prognosis:*7-16% 5-year survival rate

Notes:

Complete Duplication *Cause:*second ureteral bud arising from mesonephric duct leading to complete ureteral duplication *Prevalence*:0.2% of livebirths; M:F = 1:2; in 15-40% bilateral *Risk of recurrence:*12% in 1st-degree relatives *Embryology:*ureters develop from separate ureteric buds originating from a single Wolffian duct **Weigert-Meyer rule** = lower moiety ureter is incorporated into developing bladder first + ascends during bladder growth + enters bladder at trigone + drains lower pole and interpolar portion; upper moiety ureter remains with wolffian duct longer + passes through bladder wall + inserts inferior and medial to lower moiety ureter below the level of the trigone / into any wolffian duct derivative *Cx:* (1)Vesicoureteral reflux (most commonly) (2)Ectopic ureteral insertion (3)Ectopic ureterocele (4)Ureteropelvic junction obstruction of lower pole
UPPER MOIETY ◊Subject to ureteral obstruction from ectopic ureteral insertion / ectopic ureterocele / aberrant artery crossing! *Associated with:*significant renal dysplasia Site of insertion of ectopic ureter M:suprasphincteric insertion: low in bladder, bladder neck, prostatic urethra, vas deferens, seminal vesicle (seminal vesical cyst), ejaculatory duct ● NO ENURESIS in males as insertion is always above external sphincter ● epididymitis / orchitis in preadolescent male ● urge incontinence (insertion into posterior urethra) F:infrasphincteric insertion: distal urethra, vaginal vestibule, vagina, cervix, uterus, fallopian tube, rectum ● WETTING in upright females if insertion is below external sphincter (common) ● intermittent / constant dribbling LOWER MOIETY ◊Subject to VESICOURETERAL REFLUX due to its shortened ureteral tunnel at bladder insertion *Cx:*lower pole of duplex kidney may atrophy (in 50%) secondary to chronic pyelonephritis = reflux nephropathy (from reflux ± infection) √ clubbed calices underneath focal scars ◊Subject to UPJ OBSTRUCTION
√ two separate echodense renal sinuses + pelves separated by parenchymal bridge √ poor / nonvisualization of upper pole collecting system (delayed films) √ "drooping lily sign" = hydronephrosis + decreased function of obstructed upper pole moiety causing downward displacement of lower pole calices √ lateral displacement of lower pole collecting system + ureter √ "nubbin sign" = scarring, atrophy, and decreased function of lower pole moiety may simulate a renal mass √ tortuous dilated lower pole ureter √ voiding cystogram may show reflux into lower moiety (rare) √ displacement of proximal orifice upward

Notes:

Incomplete / Partial Duplication =branching of single ureteral bud (one ureteral orifice) before reaching metanephric blastema *Prevalence:*in 0.6% of urograms *Associated with:*ureteropelvic junction obstruction of lower renal pole √ bifid ureter (in early branching) √ bifid pelvis (in late branching) √ ureteroureteral reflux = "yo-yo" / "saddle" / "seesaw" peristalsis = urine moves down the cephalad ureter + refluxes up the lower pole ureter and vice versa √ asymmetric dilatation of one ureteral segment √ upper pole ureter may end blindly (seen on retrograde injection only) *Cx:*urinary tract infections

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Notes:

URETEROCELE

=cystic ectasia of subepithelial segment of intravesical ureter *Prevalence:*1:5,000 to 1:12,000 children IVP: √ early filling of bulbous terminal ureter ("cobra head") √ radiolucent halo (= ureteral wall + adjacent bladder urothelium) VCUG: √ round / oval lucent defect near trigone √ effacement with increased bladder distension √ ± eversion during voiding

Simple Ureterocele Ectopic Ureterocele Pseudoureterocele

Notes:

Simple Ureterocele =ORTHOTOPIC URETEROCELE = congenital prolapse of dilated distal ureter + orifice into bladder lumen at the usual location of the trigone, typically seen with single ureter *Presentation:*incidental finding in adults; M:F = 2:3; bilateral in 33% *Cx:*(1)Pyelocaliceal dilatation (2)Prolapse into bladder neck / urethra causing obstruction (rare) (3)Wall thickening secondary to edema from impacted stone / infection

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Ectopic Ureterocele =ureteral bud arising in an abnormal cephalad position from the mesonephric duct and moving caudally resulting in an ureteral orifice distal to trigone within / outside bladder *Incidence:*in 10% bilateral (a)in single nonduplicated system (20%) M:F = 1:1 ● hypoplastic / absent ipsilateral trigone √ poorly visualized / nonvisualized kidney √ small / poorly functioning kidney (b)in upper moiety ureter of duplex kidney (80%) M:F = 1:4-1:8 *Cx:*(1)Bladder outlet obstruction (from ectopic ureterocele prolapsing into bladder neck / urethra) (2)Contralateral ureteral obstruction (if ectopic ureterocele large) (3)Multicystic dysplastic kidney (the further the orifice from normal site of insertion, the more dysplastic the kidney!)

Notes:

Pseudoureterocele =obstruction of an otherwise normal intramural ureter mimicking ureterocele *Cause:* (a)Tumor bladder tumor (most common in adults), invasion by cervical cancer, pheochromocytoma of intravesical ureter (b)Edema from impacted ureteral calculus (most common in children), radiation cystitis, following ureteral instrumentation √ thick, irregular halo in urinary bladder √ "cobra head" / "spring onion" appearance of distal ureter √ NO protrusion of ureter into bladder lumen (oblique views + cystoscopy normal)

Notes:

URETEROPELVIC JUNCTION OBSTRUCTION

Most common cause of fetal / neonatal hydronephrosis *Intrinsic causes:* primarily functional with impaired formation of urine bolus (1)partial replacement of UPJ muscle by collagen (2)abnormal arrangement of junction muscles causing dysmotility (69%) (3)high ureteral insertion (4)mucosal folds in upper ureter (5)eosinophilic ureteritis (6)ischemia *Extrinsic causes:* (1)aberrant vessels to lower pole (2)adventitial bands (3)renal cyst (4)XGP (5)aortic aneurysm *Associated anomalies* (27%): vesicoureteral reflux, bilateral ureteral duplication, bilateral obstructed megaureter, contralateral nonfunctioning kidney, contralateral renal agenesis, meatal stenosis, hypospadia M:F = 5:1 Location:left > right side; bilateral (10-40%) √ large dilated anechoic renal pelvis communicating with calices, no dilatation of ureter IVP: √ sharply defined narrowing at UPJ √ pelvicaliectasis without ureterectasis √ anterior rotation of pelvis √ broad tangential sharply defined extrinsic compression (in arterial crossing) √ longitudinal striae of redundant mucosa (in dehydrated state) √ late changes: unilateral renal enlargement, diminished opacification, wasting of kidney substance OB-US: √ enlargement of renal pelvis + branching infundibula + calices √ anteroposterior diameter of renal pelvis ≥10 mm √ large unilocular fluid collection (severely dilated collecting system) *DDx:* multicystic dysplastic kidney, perinephric urinoma ADDITIONAL TESTS: (1)Diuresis excretory urography (Whitfield): accurate in 85% (2) Diuresis renography (Iodine-131-iodohippurate sodium / Tc-99m-DTPA) (3)Pressure flow urodynamic study (Whitaker) *Rx:* early surgical correction may be needed to preserve renal function

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URETHRAL DIVERTICULUM

*Age:*26-74 years; 6 x more common in black women ● urinary incontinence (9-32-70%) ● asymptomatic (3-20%)

Congenital Urethral Diverticulum Acquired Urethral Diverticulum

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Acquired Urethral Diverticulum *Prevalence:*0.6-6%; M<F *Cause:* (1)obstruction of paraurethral glands with subsequent infection + rupture into urethra (2)trauma: catheterization / childbirth Site:dorsolateral aspect of middle urethra ● vague urinary tract symptoms mimicking chronic / interstitial cystitis, carcinoma in situ of the bladder, detrusor instability ● dyspareunia ● tender cystic swelling protruding from anterior wall of vagina + expulsion of purulent material ● dribbling after voiding ● frequency / urgency (67%), dysuria (45%) ● recurrent urinary tract infections (40%) Voiding cystourethrography (65% accurate): rounded / elongated sac connected to urethra Transrectal US *Cx:*infection, stone formation (in up to 10%), malignant degeneration (5% of all urethral carcinomas) *DDx:*vaginal cyst (Gartner duct cyst, paramesonephric cyst, müllerian duct cyst, epithelial inclusion cyst), ectopic ureterocele, endometrioma, urethral tumor

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URETHRAL TRAUMA

*Incidence:*in 4-17% of pelvic fractures in males, in <1% of pelvic fractures in females *Associated with:*bladder injury in 20% *Types:* I=separation of puboprostatic ligament with craniad displacement of prostate (least common) √ elongated narrowed urethra √ elevation of bladder (displacement by hematoma) II=urethral rupture at prostatomembranous junction above urogenital diaphragm √ contrast extravasation into true pelvis III=rupture of proximal bulbous urethra below the urogenital diaphragm (most common injury) √ contrast extravasation into perineum ± scrotum *Cx:*1.Urethral stricture (38-100%) 2.Impotence (in up to 40%) 3.Incontinence (30%)

Notes:

URINOMA
=uriniferous perirenal pseudocyst secondary to tear in collecting system with continuing renal function *Etiology:* (a)nonobstructive: blunt / penetrating trauma, surgery, infection, calculus erosion (b)obstructive: (1)ureteral obstruction (calculus, surgical ligature, neoplasm) (2)bladder outlet obstruction (posterior urethral valves) ◊ Augmented by sudden diuretic load of urographic contrast material! *Path:*fibroblastic cavity (in 5-12 days), dense connective tissue encapsulation (in 3-6 weeks) √ extravasation of contrast material √ smooth thin-walled cavity (-10 to +30 HU) √ sickle-shaped collection = SUBCAPSULAR urinoma √ cystic mass in perirenal space = **localized perirenal urinoma** (most common) √ cystic mass filling entire perirenal space = **diffuse perirenal urinoma** √ encapsulated expanding intrarenal cystic mass separating renal tissue fragments = **intrarenal urinoma** √ frequently associated with urine ascites *Cx:*retroperitoneal fibrosis, stricture of upper ureter, perinephric abscess ◊Renal dysplasia of affected kidney in almost 100% when detected in utero! *Dx:*aspirated fluid with high urea concentration *DDx:*lymphocele, hematoma, abscess, renal cyst, pancreatic pseudocyst, ascites

Notes:

UROLITHIASIS
Anderson-Carr-Randall theory of renal stone formation: in the presence of abnormally high calcium excretion exceeding lymphatic capacity, microaggregates of calcium (present in the normal kidney) occur in medulla, increase in size, migrate toward caliceal epithelium, and rupture into calices to form calculi *Formation theory:* (a)nucleation theory =crystal / foreign body initiates formation in urine supersaturated with crystallizing salt (b)stone matrix theory =organic matrix of urinary proteins + serum serves as framework for deposition of crystals (c)inhibitor theory =little / no concentration of urinary stone inhibitors (citrate, pyrophosphate, glycosaminoglycan, nephrocalcin, Tamm-Horsfall protein) results in crystal formation *Annual incidence:*1-2:1,000; M:F = 4:1 ◊12% of population develop renal stones by age 70 ◊2-3% of population experience an attack of acute renal colic during their lifetime ◊Patients with acute flank pain have ureteral calculi in 67-95% *Peak age:*onset in 3rd decade *Distribution:* calcium oxalate75% struvite15% uric acid5% calcium phosphate5% cystine1%

	Mineral Composition	*Opacity*
A.	Calcium stones (90%)	
1.	Calcium oxalate monohydrate (= whewellite) + dihydrate (wedellite) (34%) √ small, densely opaque, mamillated (stippled appearance)	+++
2.	Calcium oxalate plus apatite (34%)	+++
3.	Calcium phosphate (= apatite) (5 – 10%) rarely pure (= laminated), occasionally forms in infected alkaline urine	+++
4.	Calcium hydrogen phosphate (= brushite)	+++
5.	Magnesium ammonium phosphate (= struvite) (1%) laminated, result of urea-splitting organisms (usually Proteus), most common constituent of staghorn calculus	++
6.	Struvite plus calcium phosphate (7 – 31%) associated with infection	++
B.	Cystine (3%): mildly opaque	+
C.	Uric acid (5 – 10%): radiolucent	-
D.	Xanthine (extremely rare): nonopaque	-
E.	Matrix (mucoprotein / mucopolysaccharide) (rare): nonopaque	-

Cause: ◊70-80% of patients with first-time stones have a specific metabolic disorder
1.**Hypercalciuria** ● with hypercalcemia (50%): hyperparathyroidism, milk-alkali syndrome, hypervitaminosis D, neoplastic disorders, sarcoidosis, Cushing syndrome ● with normocalcemia (30-60%): obstruction, urinary tract infection, vesical diverticulum, horseshoe kidney, medullary sponge kidney, prolonged immobilization, renal tubular acidosis, idiopathic hypercalciuria (a)absorptive hypercalciuria =increased intestinal absorption of calcium *Cause:*increase in 1,25-dihydroxy-vitamin D levels (50%) (b)renal hypercalciuria =abnormal renal calcium leak *Cause:*diet high in sodium, urinary tract infection (33%) (c)resorptive hypercalciuria =increased bone demineralization secondary to subtle hyperparathyroidism (d)idiopathic 2.**Hyperoxaluria** ◊85% of urinary oxalate is produced endogenously in liver! ◊Oxalic acid is present in many foods but poorly absorbed in healthy individuals resulting in increase in urinary oxalate by only 2-3%! (a)congenital = deficiency of an enzyme leading to accumulation of glycolate + oxylate (b)acquired = increased intake of oxalate / oxalate precursors, excess oxalate absorption from bowel in patients with ileal resection / inflammatory bowel disease ◊Hyperoxaluria has a stronger correlation to severity of stone disease than hypercalciuria!
3.**Hyperuricosuria** ● uric acid lithiasis (15-20%); stones form in acid urine (a)with hyperuricemia: gout (25%) from excessive intake of meat, fish, poultry, myeloproliferative diseases, antimitotic drugs, chemo- / radiation therapy, uricosuric agents, Lesch-Nyhan syndrome (b)with normouricemia: idiopathic; occurrence in acid-concentrated urine (hot climate, ileostomy) *Rx:*raising urinary pH (potassium citrate / sodium bicarbonate)
4.**Cystinuria** (stones form in acid urine) =autosomal recessive disorder in renal tubular reabsorption of cystine, ornithine, lysine, arginine *Age of onset:*after 10 years *Rx:*(1)decreased intake of methionine (2)alkalinization of urine
5.**Xanthinuria** =inherited autosomal recessive deficiency of xanthine oxidase (failure of normal oxidation of purines)
6.Urinary tract infection *Cause:*urea-splitting organisms (Proteus mirabilis, P. vulgaris, Haemophilus influenzae, S. aureus, Ureaplasma urealyticum) + alkaline environment (pH >7.19) may lead to magnesium ammonium phosphate = **struvite stones** *Predisposed:* women (M:F = 1:2), neurogenic bladder, urinary diversion, indwelling catheter, lower-urinary-tract voiding dysfunction √ often branching into staghorn calculi √ most struvite stones are radiopaque, but poorly mineralized matrix stones are not
7.Any condition causing nephrocalcinosis
NONRADIOPAQUE STONES *mnemonic:*"SMUX" **S**truvite (rarely magnesium ammonium phosphate) **M**atrix stone (mucoprotein, mucopolysaccharide) **U**ric acid **X**anthine
CALCULI OFTEN ASSOCIATED WITH INFECTION *mnemonic:*"S and M" **S**truvite (magnesium ammonium phosphate ± calcium phosphate) **M**atrix stone (mucoprotein, mucopolysaccharide)

Acute Obstruction By Ureteric Calculi

Notes:

Acute Obstruction By Ureteric Calculi *see also* ACUTE HYDRONEPHROSIS, page 769 ● renal colic = acute colicky flank pain frequently radiating into pelvis / groin / testis ● hematuria Site:ureteropelvic junction, iliac vessel crossing, ureterovesical junction Plain film:◊60% of calcifications along expected course of ureter on symptomatic side are ureteric stones! ◊Stones may be present in 30% of the time when KUB is negative! IVU: √ hydroureteronephrosis √ displays degree of obstruction US: √ unilateral pelvicaliectasis (up to 35% false-negative, up to 10% false-positive rate) √ resistive index >0.7 in symptomatic kidney √ absent ureteral jet on affected side (may be present with partially obstructing calculus) √ direct visualization of prevesical calculus by transabdominal, transrectal, transvaginal US CT (97% sensitive, 96% specific, 97% accurate): √ calculus within ureter (PATHOGNOMONIC) *DDx:*phlebolith √ all stone compositions readily detectable √ ureteric rim sign (77%) = ureteric edema surrounding impacted small ureteric calculus *DDx:*gonadal vein √ ureterovesical junction edema √ stranding of perinephric / periureteric fat √ perinephric fluid collection √ renal enlargement *Cx:*xanthogranulomatous pyelonephritis *Rx:*(1)hydration (within 3 hours after meal, during strenuous physical activity, at bedtime) maintaining urine output of 2-3 l/day (2)diet: restrict amounts of protein, sodium, calcium (3)drugs: thiazide diuretics (lowers urinary calcium), allopurinol (lowers urate + oxalate excretion) *Prognosis:* (1)Spontaneous passage of ureteral calculi in 93% ◊Most stones <5 mm will eventually pass! (2)Without treatment stone recurrence is 10% at 1 year, 33% at 5 years, 50% at 10 years

Notes:

VARICOCELE
=dilatation + tortuosity of plexus pampiniformis secondary to retrograde flow into internal spermatic vein Components of pampiniform plexus: (a)internal spermatic vein (ventral location) draining testis (b)vein of vas deferens (mediodorsal location) draining epididymis (c)cremasteric vein (laterodorsal location) draining scrotal wall *Etiology:* (1)Incompetent / absent valve at level of left renal vein / IVC on right side (2)Compression of left renal vein by tumor, aberrant renal artery, obstructed renal vein *Incidence:* (a)clinical varicocele: in 10-15% of adult males, in 21-39% of infertile men (b)subclinical varicocele: in 40-75% of infertile men *Theoretical causes for infertility:* (1) increase in local temperature (2) reflux of toxic substances from adrenal gland (countercurrent exchange of norepinephrine from refluxing renal venous blood into testicular arterial blood at the level of the pampiniform plexus) (3) alteration in Leydig cell function (4) hypoxia of germinative tissue due to venous reflux resulting in venous hypertension + stasis ● scrotal pain ● scrotal swelling ● abnormal spermatogram (impaired motility, immature sperm, oligospermia) Location:left side (78%), bilateral (16%), right side (6%)
Bidirectional Doppler sonography (erect with quiet breathing): (1)SHUNT TYPE (86%): insufficient distal valves allow spontaneous + continuous reflux from internal spermatic vein (retrograde flow) into cremasteric vein + vein of vas deferens (where flow is orthograde) via collaterals ● sperm quality diminished ● clinically plexus type (Grade II + III) = medium-sized + large varicoceles √ continuous reflux during Valsalva maneuver (2)STOP TYPE / PRESSURE TYPE (14%): intact intrascrotal valves allow only brief period of reflux from spermatic vein into pampiniform plexus under Valsalva maneuver ● sperm quality normal ● clinically central type (Grade 0 + I) = subclinical + small varicocele √ short phase of initial retrograde flow US: √ diameter of dominant vein in upright position at inguinal canal
*relaxed*during Valsalva
normal 2.2 mm2.7 mm small varicocele2.5-4.0increase of 1.0 mm moderate varicocele4.0-5.0increase of 1.2-1.5 mm large varicocele>5.0increase of >1.5 mm

	relaxed	*during Valsalva*
normal	2.2 mm	2.7 mm
small varicocele	2.5–4.0	increase of 1.0 mm
moderate varicocele	4.0–5.0	increase of 1.2–1.5 mm
large varicocele	>5.0	increase of >1.5 mm

*Dx:*documentation of venous reflux Rx:(1)Ivanissevitch procedure = surgery (2)Transcatheter spermatic vein occlusion

Notes:

VESICOURETERIC REFLUX

A.CONGENITAL REFLUX = PRIMARY REFLUX =incompetence of ureterovesical junction due to abnormal tunneling of distal ureter through bladder wall *Prevalence:*in 9-10% of normal Caucasian babies; in 1.4% of school girls; in 30% of children with a first episode of UTI ● short submucosal ureteral tunnel (normally has a length/width ratio of 4:1) ● large laterally located ureteral orifice Location:uni- / bilateral (frequently involves lower pole ureter in total ureteral duplication) √ renal scars in 22-50% *Prognosis:*disappears in 80% *Cx:*reflux atrophy / nephropathy in 22-50%; end-stage renal disease in 5-15% of adults B.ACQUIRED REFLUX = SECONDARY REFLUX 1.Paraureteric diverticulum = Hutch diverticulum 2.Duplication with ureterocele 3.Cystitis (in 29-50%) 4.Urethral obstruction (urethral valves) 5.Neurogenic bladder 6.Absence of abdominal musculature (prune belly syndrome) *Cx:*renal scarring with UTI (30-60%)

GRADES OF REFLUX (VCUG): GradeI:√ reflux into distal ureters GradeII:√ reflux into collecting system (without caliceal dilatation / blunting) GradeIII:√ all of the above + mild dilatation of pelvis and calices GradeIV:√ all of the above + moderate dilatation (clubbing of calices) GradeV:√ all of the above + severe tortuosity of ureter *Prognosis:* (a)grade I-III resolve with maturation of the ureterovesical junction (b)grade IV-V require surgery to avoid renal scarring + renal impairment + hypertension Renal scarring:>20% chance for grade III-V reflux; 2-3% chance for grade I-II reflux

Radionuclide cystography: ◊Lower radiation dose to gonads than fluoroscopic cystography (5 mrad)! Evaluation of bladder volume at reflux, volume of refluxed urine, residual urine volume, ureteral reflux drainage time (a)indirect: IV injection of Tc-99m DTPA (b)direct: instillation of 1 mCi Tc-99m pertechnetate (more sensitive for reflux during filling phase, which occurs in 20%) US: √ intermittent hydroureteronephrosis = variable size of collecting system √ redundant mucosa causing apparent thickening of renal pelvic wall √ large thin-walled bladder √ midline-to-orifice distance >7-9 mm has high probability of vesicoureteric reflux

Notes:

WILMS TUMOR
=NEPHROBLASTOMA ◊Most common malignant abdominal neoplasm in children 1-8 years old (10%)! ◊3rd most common malignancy in childhood (after leukemia + brain tumors; neuroblastoma more common in infancy)! ◊3rd most common of all renal masses in childhood (after hydronephrosis + multicystic dysplastic kidney)!
*Incidence:*1:10,000 livebirths; 450 cases/year in USA; familial in 1-2%; multifocal in 10%; bilateral in 4.4-9%
*Age:*peak age at 2.5-3 years (range of 3 months to 8 years); rare during first year; 50% before 3 years, 75% before 5 years; 90% before 8 years; rare in adults; M:F = 1:1
*Histo:*arises from undifferentiated metanephric blastema as nephroblastomatosis, recapitulates the developing embryonic kidney (a) aggregates of small blastemal cells (b) neoplastic nodules (c) elongated mesenchymal cells ◊Multilocular cystic nephroma, mesoblastic nephroma, nephroblastomatosis are related to the more favorable types of Wilms tumor!
In 14% associated with: (1)Sporadic aniridia (= severe hypoplasia of iris) (2)Hemihypertrophy: total / segmental / crossed (2.5%); ◊Ipsilateral or contralateral kidney affected ◊Increased incidence of all embryonal tumors (adrenal cortical neoplasms, hepatoblastoma) (3)Beckwith-Wiedemann syndrome = EMG-syndrome (exomphalos, macroglossia, gigantism) + hepatomegaly, hyperglycemia from islet cell hyperplasia (4)Genitourinary disorders (4.4%): (a)Drash syndrome (pseudohermaphroditism, glomerulonephritis, nephrotic syndrome) (b)Renal anomalies (horseshoe kidney, duplex / solitary / fused kidney) (c)Genital anomalies (cryptorchidism, hypospadia, ambiguous genitalia)
Stage: Itumor limited to kidney IIlocal extension into perirenal tissue / renal vessels outside kidney / lymph nodes IIInot totally resectable (peritoneal implants, other than paraaortic nodes involved, invasion of vital structures) IVhematogenous metastases (lung, liver, bone [rare], brain) Vbilateral renal involvement at diagnosis (5-10%)
● palpable abdominal mass (90%) ● hypertension (47-90%) ● abdominal pain (25%) ● fever (15%) ● gross hematuria (7-15%) ● microscopic hematuria (15-20%)
√ large tumor (average size 12 cm) √ sharply marginated with compressed renal tissue = pseudocapsule √ partially cystic = focal hemorrhage and necrosis (71%) √ curvilinear / phlebolithic calcifications in 5% on plain film, in 15% on CT (DDx: regular stippled calcifications in neuroblastoma) √ distorted "clobbered" calices √ tumor may invade IVC / right atrium (4-10%) √ tumor may cross midline √ hypervascular tumor: enlarged tortuous vessels, coarse neovascularity; small arterial aneurysms, vascular lakes √ parasitization of vascular supply US: √ fairly evenly echogenic mass √ ± irregular anechoic areas due to central necrosis + hemorrhage MR: √ hypointense on T1WI, variable on T2WI NUC: √ nonfunctioning kidney (10%) √ hypo- / iso- / hyperperfusion on radionuclide angiogram √ absent tracer accumulation on delayed static images √ displacement of kidney + distortion of collecting system
*Prognosis:*90% survival rate depending on pathologic pattern, age at time of diagnosis, extent of disease

VARIANT:**Cystic partially differentiated nephroblastoma** =combination of MLCN + Wilms tumor elements *Incidence:* ?; M < F √ multiple noncommunicating locules √ polypoid masses within locules

Notes:

WOLMAN DISEASE
=PRIMARY FAMILIAL XANTHOMATOSIS = rare autosomal recessive lipidosis with accumulation of cholesterol esters and triglycerides in visceral foam cells + various tissues (liver, spleen, lymph nodes, adrenal cortex, small bowel) *Etiology:*deficiency of lysosomal acid esterase / acid lipase ● malabsorption in neonatal period: failure to thrive, diarrhea, steatorrhea, vomiting ● delayed growth, diminished muscle mass, abdominal distention √ hepatosplenomegaly √ extensive bilateral punctate calcifications (calcification of fatty-acid soaps) throughout enlarged adrenals (maintaining their normal triangular shape) is DIAGNOSTIC √ enlarged fat-containing lymph nodes √ small bowel wall thickening (due to infiltration of mucosa of small bowel by lipid-filled histiocytes impairing absorption) √ generalized osteoporosis CT & MR:attenuation + signal intensities consistent with deposition of lipids *Dx:*assay of leukocytes / cultured skin fibroblasts *Prognosis:*death occurs within first 6 months of life

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ZELLWEGER SYNDROME
= CEREBROHEPATORENAL SYNDROME autosomal recessive ● muscular hypotonia ● hepatomegaly + jaundice ● craniofacial dysmorphism ● seizures, mental retardation √ brain dysgenesis (lissencephaly, macrogyria, polymicrogyria) √ renal cortical cysts *Prognosis:* death in early infancy

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Notes:

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Level I Obstetric Ultrasound *Indication:*MS-AFP ≥2.5 multiples of mean (MoM) between 14 and 18 weeks MALimited scope of examination to identify frequent causes of MS-AFP elevation in 20-50% of pregnancies: 1.Gestational age ≥2 weeks more advanced than estimated clinically (18%)2.Multiple gestations (10%)3.Unsuspected fetal demise (5%)4.Obvious fetal NTD / abdominal wall defect*Outcome:*no cause identified in 50-80%*Recommendation if level I ultrasound is unrevealing:* (1)amniocentesis for AF-AFP (with normal results in >90%)(2)level II obstetric ultrasound (skipping amniocentesis)

Notes:

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Level II Obstetric Ultrasound *Indication:*AF-AFP ≥2 MoM*Accuracy:*identification of abnormal fetuses in 99%Examination targeted for: 1.Open neural tube defect:anencephaly, encephalocele, open spina bifida, amniotic band syndrome resulting in open neural tube defect2.Closed neural axis anomaly:hydrocephalus, Dandy-Walker malformation3.Abdominal wall defect:gastroschisis, omphalocele, gastropleuroschisis from amniotic band syndrome4.Upper GI obstruction:esophageal atresia ± tracheoesophageal fistula, duodenal obstruction5.Cystic hygroma6.Teratoma:sacrococcygeal, lingual, retropharyngeal7.Renal anomalies:obstructive uropathy, renal agenesis, multicystic dysplastic kidney, congenital Finnish nephrosis◊Risk of fetal chromosomal anomaly is only 0.6-1.1% with normal level II sonogram!

Notes:

First Trimester Bleeding =VAGINAL BLEEDING IN FIRST TRIMESTER*Frequency:*15-25% of all pregnancies, of which 50% terminate in abortionA.INTRAUTERINE CONCEPTUS IDENTIFIED1.Blighted ovum / blighted twin2.Threatened abortion3.Implantation bleed4.Early fetal death5.Gestational trophoblastic disease6.Subchorionic hemorrhageB.NORMAL ENDOMETRIAL CAVITY(a)with b-HCG level >1,800 mIU/mL1.Recent spontaneous abortion2.Ectopic pregnancy(b)with b-HCG level <1,800 mIU/mL1.Very early IUP2.Ectopic pregnancy

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Notes:

Positive b-HCG Without IUP *mnemonic:*"HERE"**H**CG-producing tumor (rare) **E**ctopic pregnancy **R**ecent / incomplete abortion **E**arly intrauterine pregnancy

Notes:

Dilated Cervix 1.Inevitable abortion2.Premature labor=spontaneous onset of palpable, regularly occurring uterine contractions between 20 and 37 weeks MA3.Incompetent cervix=gaping cervix usually develops during 2nd trimester*Predisposed:*cervical trauma (D & C, cauterization), DES exposure in utero with cervical hypoplasia, estrogen medication*Dx:* visualization of fetal parts / amniotic fluid within dilated endocervical canal (stress test: patient standing with bladder empty)*Prognosis:*14th-18th week best time for Rx prior to significant cervical dilatation

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Notes:

Uterus Large For Dates 1.Multiple gestation pregnancy2.Inaccurate menstrual history3.Fibroids4.Polyhydramnios5.Hydatidiform mole6.Fetal macrosomia

Notes:

Empty Gestational Sac 1.Normal early IUP between 5-7 weeks MA2.Blighted ovum*DDx:*Pseudosac of ectopic pregnancy

Notes:

Alpha-fetoprotein =glycoprotein as major circulatory protein of early fetus*Origin:*formed initially by yolk sac + fetal gut (4-8 weeks), later by fetal liverDetectable in (a)fetal serum ● concentration peaks at 14-15 weeks followed by progressive decline(b)amniotic fluid (AF-AFP) secondary to fetal urination, fetal gastrointestinal secretions, transudation across fetal membranes (amnion, placenta), transudation across immature fetal epithelium ● concentration peaks early in 2nd trimester followed by progressive decline(c)maternal circulation (MS-AFP) secondary to leakage from amniotic fluid across the placenta ● levels rise from 7th week, peak at 32nd week, and decline toward end of pregnancy◊Either high / low MS-AFP is associated with 34% of all major congenital defects!

Sample siteApproximate levelPeak(ng/mL)maternal serum3030th-32nd weekamniotic fluid20,000early 2nd trimesterfetal plasma3,000,00014th-15th week

At the end of the 1st trimester AFP is present: in fetal plasmain *milligram* quantitiesin amniotic fluidin *microgram* quantitiesin maternal serumin *nanogram* quantities

Reported in MoM =multiples of mean to standardize interpretation among laboratories

Elevated Alpha-fetoprotein ● screening at 16-18 weeks GA◊Values must be corrected for dates, maternal weight, race, presence of diabetes (diabetes has depressing effect on MS-AFP so that lower levels may be associated with NTDs)(a)Elevation in MATERNAL SERUM (MS-AFP)=defined as ≥2.5 MoM / equivalent to the 5th percentile; 4.5 MoM for multiple gestationsPower of detection at ≥2.5 MoM cutoff: 98% of gastroschisis 90% of anencephalic fetuses 75-80% of open spinal defects 70% of omphaloceles *Incidence:*2-5% screen-positive rate (in 16% normal MS-AFP on retesting); 6-15% of fetuses have some type of major congenital defect; in 1.3 per 1,000 tests fetal anomaly detected◊The higher the AFP elevation the higher the probability of fetal anomalies◊20-38% of women with unexplained high MS-AFP (ie, in absence of fetal abnormality) suffer adverse pregnancy outcomes (premature birth, preeclampsia, 2-4 x IUGR, 10 x perinatal mortality, 10 x placental abruption)!(b)Elevation in AMNIOTIC FLUID (AF-AFP)=defined as ≥2 MoM*Incidence:*<10% of women with elevated MS-AFP and "unrevealing" level I US exam ● determine acteylcholinesterase + karyotype in amniotic fluid◊66% of fetuses of women with elevated AF-AFP levels are normal!◊A targeted level II ultrasound exam will show fetal anomalies in 33%!
Associated with: A.LABORATORY ERRORB.ERRONEOUS DATES (18%): fetus actually older (AFP levels rise 15% per week during 16-18-week window)C.MULTIPLE GESTATIONS (14%)D.FETAL DEMISE (7%) / fetal distress / threatened abortionE.FETAL ANOMALIES (61%)1.Neural tube defects (51%):[anencephaly (30%), myelomeningocele (18%), encephalocele (3%), forebrain malformation] *Prevalence:*1.6 per 1,000 births in USA;6 per 1,000 in Great Britain ◊in 90% as 1st time event!*Risk of recurrence:*3% after one affected child; 6% after 2 affected children2.Ventral wall defects (21%)(gastroschisis, omphalocele): sensitivity of 50% 3.Upper GI obstruction(esophageal / duodenal atresia) 4.Cystic hygroma, teratoma (pharyngeal, sacral)5.Amniotic band syndrome(asymmetric cephalocele, gastropleuroschisis) 6.Renal abnormalities:multicystic dysplastic kidney, renal agenesis, pelviectasis, **congenital Finnish nephrosis** (typically ≥10 MoM + negative amniotic fluid acetylcholinesterase) 7.OligohydramniosF.PLACENTAL LESION1.Infarct2.Chorioangioma3.Peri- and intraplacental hematoma resulting in fetomaternal hemorrhage4.Placental lakes, intervillous thrombosisG.LOW BIRTH WEIGHTH.Normal pregnancy + MATERNAL DISORDER1.Hepatitis2.HepatomaI.Fetal-maternal blood mixing: collection of MS-AFP samples after amniocentesis
mnemonic:"GEM MINER CO"*H0***G**astroschisis **E**sophageal atresia **M**ultiple gestations **M**ole **I**ncorrect menstrual dates **N**eural tube defects **E**rror (laboratory) **R**enal disease in fetus (autosomal recessive polycystic kidney disease, renal dysplasia, obstructive uropathy, congenital Finnish nephrosis) **C**horioangioma **O**mphalocele

Low Alpha-fetoprotein =MS-AFP ≤0.5 / AF-AFP ≤0.72 multiples of the median*Incidence:*3%1.Autosomal trisomy syndromes (trisomy 21, 18, 13)◊20% of trisomy 21 fetuses are found in women with low MS-AFP after adjustment for age!2.Absence of fetal tissues (eg, hydatidiform mole)3.Fetal demise4.Misdated pregnancy5.Normal pregnancy6.Patient not pregnant

Notes:

Use Of Karyotyping *Frequency:*11-35% of fetuses with sonographically identified abnormalities have chromosomal abnormalitiesA.FETAL ANOMALIES1.CNS anomalies: holoprosencephaly (43-59%), Dandy-Walker malformation (29-50%), cerebellar hypoplasia, agenesis of corpus callosum, myelomeningocele (33-50%)2.Cystic hygroma (72%): Turner syndrome3.Omphalocele (30-40%)4.Cardiac malformations5.Nonimmune hydrops6.Duodenal atresia7.Severe early-onset IUGR: trisomy 18, 13, triploidy8.Diaphragmatic hernia9.Bone-echodense bowel (20%): trisomy 21B.MATERNAL RISK FACTORS1.Advanced age2.Low serum a-fetoprotein3.Abnormal triple screen of maternal serum4.History of previous chromosomally abnormal pregnancy (1% risk of recurrence)C.PLANNED INTENSE INTRAUTERINE MANAGEMENT
Fetal anomalies not associated with chromosomal anomalies: 1.Gastroschisis2.Unilateral renal anomaly3.Intestinal obstruction distal to duodenal bulb4.Off-midline unilateral cleft lip5.Fetal teratoma (sacrococcygeal / anterior cervical)6.Isolated single umbilical artery

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Notes:

AMNIOTIC FLUID VOLUME

Production: (a)1st trimester: dialysate of maternal + fetal serum across the noncornified fetal skin(b)2nd + 3rd trimester: fetal urine (600-800 cm^3/day near term), fetal lungs (600-800 cm^3/day near term), amniotic membraneAbsorption: fetal swallowing + GI absorption, fetal lung absorption, clearance by placenta Assessment of amniotic fluid volume by: (1)Subjective assessment ("Gestalt" method):quick + efficient, accounts for GA-related variations in fluid volume, considered the most accurate if performed by experienced operator, operator + interpreter must be identical, no documentation, variations on serial scans difficult to appreciate (2)Depth of largest vertical pocket: simple + quick (used in BPP), pockets >2 cm may be found in crevices between fetal parts with moderately severe oligohydramnios, does not account for GA-related variations(3)Four-quadrant **Amniotic Fluid Index** (AFI): fairly quick, correlates probably better with fluid volume than any single measurement, may not accurately reflect overall fluid volume, may be affected by fetal movement during measurements(4)Planimetric measurement of total intrauterine volume(5)Dye / para-amino hippurate dilution technique:800 cm^3 at 34 weeks, 500 cm^3 >34 weeks

Polyhydramnios Oligohydramnios Intrauterine Membrane In Pregnancy

Notes:

Polyhydramnios =amniotic fluid volume >1500-2000 cm^3 at term*Incidence:*1.1-2-3.5%√ fetus does not fill the AP diameter of uterus√ single largest pocket devoid of fetal parts / cord >8 cm in vertical direction√ AFI ≥20-24 cm*Prognosis:*64% perinatal mortality with severe polyhydramnios*Etiology:* A.IDIOPATHIC (60%)associated with macrosomia in 19-37% *Suggested cause:* (1)increased renal vascular flow(2)bulk flow of water across surface of fetus + umbilical cord + placenta + membranesB.MATERNAL CAUSES (20%)1.Diabetes (5%)2.Isoimmunization (Rh incompatibility)3.Placental tumors: chorioangiomaC.FETAL ANOMALIES (20-63%)(a)gastrointestinal anomalies (6-16%)impairment of fetal swallowing (esophageal atresia in 3%); high intestinal atresias / obstruction of duodenum / proximal small bowel (1.2-1.8%), omphalocele, meconium peritonitis (b)nonimmune hydrops (16%)(c)neural tube defects (9-16%)anencephaly, hydranencephaly, holoprosencephaly, myelomeningocele, ventriculomegaly, agenesis of corpus callosum, encephalocele, microcephaly (d)chest anomalies (12%)diaphragmatic hernia, cystic adenomatoid malformation, tracheal atresia, mediastinal teratoma, primary pulmonary hypoplasia, extralobar sequestration, congenital chylothorax (e)skeletal dysplasias (11%)dwarfism (thanatophoric dysplasia, achondroplasia), kyphoscoliosis, platyspondyly (f)chromosomal abnormalities (9%)trisomy 21, 18, 13 (g)cardiac anomalies (5%)VSD, truncus arteriosus, ectopia cordis, septal rhabdomyoma, arrhythmia (h)genitourinary malformations*Cause:*? hormonally mediated polyuriaunilateral UPJ obstruction, unilateral multicystic dysplastic kidney, mesoblastic nephroma (i)miscellaneous (8%)cystic hygroma, facial tumors, cleft lip / palate, teratoma, amniotic band syndrome, congenital pancreatic cyst◊In polyhydramnios efforts to detect fetal anomalies should be directed at SGA fetuses!
mnemonic:"TARDI"**T**wins **A**nomalies, fetal **R**h incompatibility **D**iabetes **I**diopathic

Notes:

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Oligohydramnios =amniotic fluid volume <500 cm^3 at term single largest pocket devoid of fetal parts / cord ≤1-2 cm in vertical direction AFI ≤5-7 cm*Etiology: mnemonic:*"DRIPP"**D**emise of fetus / **D**rugs (Motrin therapy for tocolysis of preterm labor) **R**enal anomalies, bilateral (= inadequate urine production): renal agenesis / dysgenesis, infantile polycystic kidney disease, prune belly syndrome, posterior urethral valves, urethral atresia, cloacal anomalies 20-fold increase in incidence of fetal anomalies with oligohydramnios!N.B.:bilateral renal obstruction, if combined with intestinal obstruction, may be associated with polyhydramnios**I**UGR (reduced renal perfusion) **P**remature rupture of membranes (most common) **P**ostmaturity

*Cx:*pulmonary hypoplasia, cord compression*Prognosis:*77-100% perinatal mortality with 2nd trimester oligohydramnios

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Notes:

Intrauterine Membrane In Pregnancy A.MEMBRANE OF MATERNAL ORIGIN1.Uterine septum=incomplete resorption of sagittal septum between the fused two müllerian ducts2.Amniotic sheet / shelve=folding of amniochorionic membrane around uterine synechia synechia often thins during uterine stretching + disappears as pregnancy progressesB.MEMBRANE OF FETAL ORIGIN1.Intertwin membrane=apposing membrane of multiple pregnancy2.Amniotic band=rent within amnion3.Chorioamnionic separation=incomplete fusion / hemorrhagic separation of amnion (= inner membrane) and chorion (= outer membrane)4.Subchorionic hemorrhage = chorioamnionic elevation=separation of chorionic membrane from decidua ● implantation bleed of early pregnancy
mnemonic:"STABS"**S**eparation (chorioamnionic) **T**wins (intertwin membrane) **A**bruption **B**ands (amniotic band syndrome) **S**ynechia

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Notes:

Abnormal Placental Size ◊Placental mass tends to reflect fetal mass!A.ENLARGEMENT OF PLACENTA=>5 cm thick in sections obtained at right angles to long axis of placenta(a)maternal disease1.Maternal diabetes (= villous edema)2.Chronic intrauterine infections3.Maternal anemia (= normal histology)4.Alpha-thalassemia(b)fetal disease1.Hemolytic disease of the newborn (= villous edema + hyperplasia)2.Umbilical vein obstruction3.Fetal high-output failure: large chorioangioma, arteriovenous fistula4.Fetal malformation: Beckwith-Wiedemann syndrome, sacrococcygeal teratoma, chromosomal abnormality, fetal hydrops5.Twin-twin transfusion syndrome(c)fetomaternal hemorrhage*mnemonic:*"HAD IT"**H**ydrops **A**bruption **D**iabetes mellitus **I**nfection **T**riploidy B.DECREASE IN PLACENTAL SIZE1.Preeclampsiaassociated with placental infarcts in 33-60% 2.IUGR3.Intrauterine infection4.Chromosomal abnormality

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Notes:

Vascular Spaces Of The Placenta 1.**"Placental cysts"**=large fetal veins located between amnion + chorion anastomosing with umbilical vein√ sluggish blood flow (detectable by real-time observation)2.**Basal veins**=decidual + uterine veins√ lacy appearing network of veins underneath placenta*DDx:* placental abruption3.**Intraplacental venous lakes**√ intraplacental sonolucent spaces√ whirlpool motion pattern of flowing blood

Macroscopic Lesions Of The Placenta 1.**Intervillous thrombosis** (36%)=intraplacental areas of hemorrhage*Etiology:*breaks in villous capillaries with bleeding from fetal vessels√ irregular sonolucent intraplacental lesions (mm to cm range)√ blood flow may be observed within lesionSignificance:fetal-maternal hemorrhage (Rh sensitization, elevated AFP levels)2.**Perivillous fibrin deposition** (22%)=nonlaminated collection of fibrin deposition*Etiology:*thrombosis of intervillous spaceSignificance:none 3.**Septal cyst** (19%)*Etiology:*obstruction of septal venous drainage by edematous villi√ 5-10 mm cyst within septumSignificance:none4.**Placental infarct** (25%)=coagulation necrosis of villi *Etiology:*disorder of maternal vessels, retroplacental hemorrhage√ not visualized unless hemorrhagic√ well-circumscribed mass with hyperechoic / mixed echo patternSignificance:dependent on extent + associated maternal condition5.**Subchorionic fibrin deposition** (20%)=laminated collection of fibrin deposition*Etiology:*thrombosis of maternal blood in subchorionic space√ subchorionic sonolucent areaSignificance:none6.**Massive subchorial thrombus**=BREUS MOLE = PREPLACENTAL HEMORRHAGE

Notes:

Placental Tumor A.TROPHOBLASTIC1.Complete hydatidiform mole2.Partial hydatidiform mole3.Invasive mole4.ChoriocarcinomaB.NONTROPHOBLASTIC1.Chorioangioma (in up to 1% of placentas)2.Teratoma (rare)3.Metastatic lesion (rare): melanoma, breast carcinoma, bronchial carcinoma

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Notes:

Unbalanced Intertwin Transfusion =unbalanced intertwin transfusion through vascular anastomoses between the two circulations of monochorionic twinsA.ACUTE= Twin-embolization syndromeB.CHRONIC= Twin-twin transfusion syndromeC.REVERSE= Acardiac twinning

Notes:

Abnormal Cord Attachment 1.Marginal cord attachment (7%)=battledore placenta (flat wooden paddle used in an early form of badminton) ● no clinical significance2.Velamentous insertion of cord (1%)3.Vasa previa

Notes:

Umbilical Cord Lesions ◊Umbilical cord cysts persisting into 2nd + 3rd trimester are frequently accompanied by fetal anomalies (hernia, intestinal obstruction, urinary tract obstruction, urachal anomalies, omphalocele, cardiac defect, trisomy 18)!
A.DEVELOPMENTAL CORD LESION1.**Umbilical hernia**=protrusion from anterior abdominal wall with normal insertion of umbilical vessels*Predisposed:* Blacks, low-birth-weight infants, trisomy 21, congenital hypothyroidism, Beckwith-Wiedemann syndrome, mucopolysaccharidoses *Prognosis:*spontaneous closure in first 3 years of life2.**Omphalomesenteric duct cyst**√ near fetal end of cord + eccentric in cord3.**Allantoic cyst**=remnant of umbilical vesicle / allantois; usually degenerates by 6 weeks*Histo:*lined by single layer of flattened epithelium√ near fetal end of cord + in center of cord4.**Amniotic inclusion cyst**=amniotic epithelium trapped within umbilical cord5.**Mucoid degeneration of umbilical cord**=**umbilical cord pseudocyst**=liquefaction of Wharton jelly / edema√ focal thickening of Wharton jelly, usually near umbilicus√ usually resolved by 12 weeks MAAssociated commonly with omphalocele 6.**Noncoiled "straight" cord**counterclockwise:clockwise umbilical cords = 7:1 right-handed:left-handed persons = 7:1 *Incidence:*3.7-5%√ absent vascular coiling for entire length of visible cord*At risk for:*intrauterine death (8%), stillbirth, fetal anomalies (24%), prematurity, intrapartum heart rate decelerations, fetal distress, meconium staining
B.ACQUIRED CORD LESION1. **False knot** (a)exaggerated looping of cord vessels causing focal dilatation of cord(b)focal accumulation of Wharton jelly(c)varix of umbilical vessel√ knoblike protrusion / bulge of cord2. **True knot** *Incidence:*1% of pregnancies*Cause:*excessive fetal movements*Predisposed:*long cord, polyhydramnios, small fetus, monoamniotic twins√ local distension / thrombosis of umbilical vein near cord knot resembling an umbilical cyst√ tortuosity of cord at level of knot*Cx:*vascular occlusion + fetal death in uteroOB management:expectant3. **Umbilical cord hematoma** =rupture of the wall of the umbilical vein secondary to mechanical trauma (torsion, loops, knots, traction) / congenital weakness of vessel wall*Incidence:*1:5,505 to 1:12,699 deliveriesLocation:near fetal insertion of umbilical cord (most common)√ hyper- / hypoechoic mass 1-2 cm in size, multiple (in 18%)*Cx:*rupture into amniotic cavity with exsanguination*Prognosis:*52% overall perinatal fetal mortality4. Neoplasm (a)**Angiomyxoma / hemangioma of cord***Incidence:*22 cases in literature*Histo:*multiple vascular channels lined by benign endothelium surrounded by edema + myxomatous degeneration of Wharton jelly*Associated with:*elevated a-fetoprotein levelLocation:more frequently near placental end of umbilical cord√ hyperechoic / multicystic mass within cord√ may be associated with pseudocyst(= localized collection of edema)*Cx:* premature delivery, stillbirth, hydramnios, nonimmune hydrops, massive hemorrhage due to rupture(b)Other tumors: myxosarcoma, dermoid, teratoma5. **Umbilical vein varix** *Incidence:*<4% of all umbilical cord abnormalitiesSite:intraamniotic, intraabdominal√ fusiform dilatation of umbilical vein *Cx:*(1)Thrombosis with subsequent fetal death(2)Partial thrombosis with IUGR*Prognosis:*usually no clinical significance6.Umbilical artery aneurysm

Notes:

FETAL SKELETAL DYSPLASIA
=heterogeneous group of bone growth disorders resulting in abnormal shape + size of the skeleton
More than 200 skeletal dysplasias are known, but only a few are frequent:-thanatophoric dysplasia-osteogenesis imperfecta type II (56%)-achondrogenesis (71%)-heterozygous achondroplasia*Birth prevalence:* 2.3:10,000-7.6:10,000 births for all skeletal dysplasias; 1.5:10,000 births for lethal skeletal dysplasias *Prognosis:* 51% lethal due to hypoplastic lungs:23% stillbirths, 32% death in 1st week of life
Birth Perinatal
*prevalence*deaths

Thanatophoric dysplasia0.69:10,000*1:246Achondroplasia0.37:10,000noneAchondrogenesis, type I0.23:10,000*1:639Achondrogenesis, type II0.25:10,000*Osteogenesis imperf. type II0.18:10,000*1:799Osteogenesis imperf., others0.18:10,000noneAsphyxiating thoracic dysplasia0.14:10,0001:3,196Hypophosphatasia0.10:10,000*Chondrodysplasia punctata, rhizo0.09:10,000*noneCamptomelic dysplasia0.05:10,000*1:3,196Chondroectodermal dysplasia0.05:10,0001:3,196Cleidocranial dysplasia0.05:10,000Diastrophic dysplasia0.02:10,000** = lethal dysplasias
√ shortening of long bones (common characteristic)Femur length >5 mm below 2 standard deviations suggests skeletal dysplasia!√ femur length/foot length ratio <0.9
√ moderate limb shortening of 40-60% of the mean in thanatophoric dysplasia + OI type II√ severe limb shortening of >30% of the mean in achondrogenesis*DDx features:*mineralization, bowing, fractures, number of digits, fetal movement, thoracic measurement, associated anomalies, age of onset*DDx:*constitutionally short limbs, severe IUGR
see also DWARFISM

Fetal Hand Malformation

Notes:

Fetal Hand Malformation ***Polydactyly*** trisomy 13, short-rib-polydactyly syndrome, asphyxiating thoracic dystrophy (Jeune syndrome), Smith-Lemli-Opitz syndrome (a)Postaxial polydactylychondroectodermal dysplasia (Ellis-van Creveld syndrome), Meckel-Gruber syndrome, hydrolethalus syndrome (b)Preaxial polydactylyorofaciodigital syndrome
Syndactyly Apert syndrome, triploidy, Roberts syndrome
Clinodactyly trisomy 21, triploidy
Overlapping Digit trisomy 18
Hitchhikers Thumb diastrophic dysplasia
Flexion Contractures trisomy 13 + 18, fetal akinesia deformation sequence
Limb Reduction congenital varicella, hypoglossia-hyperdactyly syndrome
Amputation amniotic band syndrome

Notes:

FETAL CNS ANOMALIES

*Incidence:*2:1,000 births (United States); 90% as 1st time occurrence*Recurrence:*2-3% after 1st, 6% after 2nd occurrence√ ventricular atrium + cisterna magna are two sensitive anatomic markers for normal brain development!A.HYDROCEPHALUS1.Aqueductal stenosis2.Communicating hydrocephalus3.Dandy-Walker malformation4.Choroid plexus papillomaB.NEURAL TUBE DEFECT*Incidence:*1:500-600 livebirths*Risk of recurrence:*3-4%1.Spina bifida2.Anencephaly3.Acrania4.Encephalocele (8-15%)5.Porencephaly6.Hydranencephaly7.Holoprosencephaly8. Iniencephaly9. Microcephaly10.Agenesis of corpus callosum11.Lissencephaly12.Arachnoid cyst13.Choroid plexus cyst14.Vein of Galen aneurysmC.INTRACRANIAL NEOPLASM1.Teratoma (>50%): benign / malignantLocation: originate from base of skull 2.Glioblastoma3.Astrocytoma

Hypotelorism Hypertelorism Fetal Ventriculomegaly Cystic Intracranial Lesion Abnormal Cisterna Magna

Notes:

Hypotelorism 1.Holoprosencephaly2.Chromosomal abnormalities: trisomy 133.Microcephaly, trigonocephaly4.Maternal phenylketonuria5.Meckel-Gruber syndrome6.Myotonic dystrophy7.Williams syndrome8.Oculodental dysplasia

Notes:

Hypertelorism 1.Median cleft syndrome: cleft lip/palate2.Craniosynostosis:Apert /Crouzon syndrome3.Pena-Shokeir syndrome4.Frontal / ethmoidal, sphenoidal encephalocele5.Dilantin effect

Notes:

Fetal Ventriculomegaly *Cause:* A.Morphologic anomaly (70-80%):1.Spina bifida (30-65%)2.Dandy-Walker malformation3.Encephalocele4.Holoprosencephaly5.Agenesis of corpus callosumB.Abnormal karyotype (10-20%)C.Viral infection20-40% of concurrent anomalies are missed by ultrasound!
"dangling" choroid plexus = choroid hanging from tela choroideawidth of ventricular atrium >10 mm*Prognosis:*21% survival rate; 50% with intellectual impairment; 80% with isolated mild ventriculomegaly (atrial width >10 and ≤15 mm) have normal motor + intellectual function at ≥12 months of age

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Notes:

Cystic Intracranial Lesion *mnemonic:*"CHAP VAN"**C**horoid plexus cyst **H**ydrocephalus, **H**oloprosencephaly, **H**ydranencephaly **A**genesis of corpus callosum + cystic dilatation of 3rd ventricle **P**orencephaly **V**ein of Galen aneurysm **A**rachnoid cyst **N**eoplasm (cystic teratoma)

Notes:

Abnormal Cisterna Magna Normal size between 15 and 25 weeks MA: >2 to <10 mm (usually 4-9 mm) in 94-97% of fetusesA.SMALL CISTERNA MAGNA + "banana sign"1.Chiari II malformation (with myelomeningocele)2.Occipital cephalocele3.Severe hydrocephalusB.LARGE CISTERNA MAGNA1.Megacisterna magna cerebellum + vermis remain intact2.Arachnoid cyst en bloc displacement of cerebellum + vermis3.Cerebellar hypoplasia4.Dandy-Walker syndrome (with vermian agenesis)

Notes:

FETAL NECK ANOMALIES
1.Cervical myelomeningocele2.Occipital cephalocele3.Cystic hygroma4.Teratoma

Nuchal Skin Thickening Macroglossia Micrognathia Maxillary Hypoplasia

Notes:

Nuchal Skin Thickening =NUCHAL SONOLUCENCY / FULLNESS / EDEMA=skin thickening of posterior neck measured between calvarium + dorsal skin margin(a)≥3 mm during 9-13 weeks MA(b)≥6 mm during 14-21 weeks MA◊The smallest measurement should be used!Image plane:axial plane (slightly craniad to that of the BPD measurement) that includes cavum septi pellucidi, cerebellar hemisphere and cisterna magna*Incidence:*among the most common anomaly in 1st trimester + early 2nd trimester*Causes:* A.NORMAL VARIANT (0.06%)B.CHROMOSOMAL DISORDERSTrisomy 21 (in 45-80%), Turner syndrome (45 X0), Noonan syndrome, trisomy 18, XXX syndrome, XYY syndrome, XXXX syndrome, XXXXY syndrome, 18p-syndrome, 13q-syndrome ◊30-40% of fetuses with Down syndrome have nuchal skin thickening!C.NONCHROMOSOMAL DISORDERS1.Multiple pterygium syndrome = Escobar syndrome2.Klippel-Feil syndrome (fusion of cervical vertebrae, CHD, deafness (30%), cleft palate3.Zellweger syndrome = cerebrohepatorenal syndrome (large forehead, flat facies, macrogyria, hepatomegaly, cystic kidney disease, contractures of extremities)4.Robert syndrome5.Cumming syndrome√ larger lymphangiomas with radiating septations are usually found with trisomy 18√ nuchal fullness ≥3 mm during 1st trimester is seen in trisomy 21 / 18 / 13 (30-50% PPV)√ often reverting to normal by 16-18 weeks√ septations within nuchal translucency carries a 20- to 200-fold risk for chromosomal anomalies compared with normal
*Sensitivity:*2-44-75% for detection of trisomy 21*Specificity:*99% for detection of trisomy 21*Positive screen:*1.2-3% in general population (exceeding 0.5% risk of amniocentesis)*False positives:*1-2-8.5%OB-management:thorough sonographic evaluation at 18-20 weeks MA*DDx:*chorioamnionic separation

Notes:

Macroglossia 1.Beckwith-Wiedemann syndrome2.Down syndrome3.Hypothyroidism4.Mental retardation

Notes:

Micrognathia 1.Pierre-Robin syndrome2.Treacher-Collins syndrome3.Goldenhar syndrome (hemifacial microsomia)4.Seckel syndrome (bird-headed dwarfism)5.Multiple pterygium syndrome6.Pena-Shokeir syndrome7.Beckwith-Wiedemann syndrome8.Arthrogryposis9.Skeletal dysplasias10.Trisomy 13, 18, 9 (abnormal karyotype in 25%)*Prognosis:*20% survival

Notes:

Maxillary Hypoplasia 1.Down syndrome2.Drugs (alcohol, dilantin, valproate)3.Apert / Crouzon syndrome4.Achondroplasia5.Cleft lip/palate

Notes:

Pulmonary Hypoplasia *Path:*absolute decrease in lung volume / weight for gestational age*Cause:* 1.Prolonged oligohydramnios (20-25%)2.Skeletal dysplasia (small thorax)3.Intrathoracic mass (lung compression)4.Large hydrothorax (lung compression)5.Neurologic condition (reduced breathing activity)6.Chromosomal abnormality7.CHD with R-sided cardiac obstructing lesion
√ thoracic circumference (TC) <5th percentile for EGA√ declining TC:AC ratio from >0.80 (75% sensitive, 80-90% specific); not applicable for intrathoracic masses

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Notes:

Intrathoracic Mass in order of frequency: 1.Diaphragmatic hernia / eventration2.Cystic adenomatoid malformation3.Bronchopulmonary sequestration4.Bronchogenic cyst with bronchial compression5.Bronchial atresia

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Notes:

Chest Mass

Unilateral Chest Mass 1.Congenital diaphragmatic hernia2.Cystic adenomatoid malformation3.Bronchopulmonary sequestration4.Bronchogenic cyst5.Unilateral bronchial atresia / stenosis
Bilateral Chest Masses 1.Laryngeal / tracheal atresia2.Bilateral cystic adenomatoid malformation3.Bilateral congenital diaphragmatic herniae
Mediastinal Mass 1.Goiter2.Cystic hygroma3.Pericardial teratoma4.Neuroblastoma
Cystic Chest Mass 1.Bronchogenic cyst2.Enteric cyst3.Neurenteric cyst4.Cystic adenomatoid malformation (Type I)5.Congenital diaphragmatic hernia6.Pericardial cyst7.Mediastinal meningocele
Complex Chest Mass 1.Congenital diaphragmatic hernia2.Cystic adenomatoid malformation (Type I, II, III)3.Pulmonary sequestration4.Complex enteric cyst5.Pericardial teratoma
Solid Chest Mass 1.Congenital diaphragmatic hernia (bowel ± liver)2.Cystic adenomatoid malformation type III3.Pulmonary sequestration4.Obstructed lung from bronchial atresia, laryngeal atresia, bronchogenic cyst5.Bronchopulmonary foregut malformation6.Pericardial tumor7.Heterotopic brain tissue
Regressing Fetal Chest Mass 1.Cystic adenomatoid malformation2.Bronchopulmonary sequestration

Notes:

Chest Wall Mass 1.Hemangioma2.Cystic hygroma3.Teratoma4.Hamartoma5.Thoracic myelomeningocele

Notes:

Pleural Effusion 1.Primary idiopathic chylothorax (most common)2.Hydrops fetalis (multiple causes)3.Chromosome anomaly: trisomy 21, 45 XO (mostly)4.Pulmonary lymphangiectasia / cystic hygroma5.Lung mass: cystic adenomatoid malformation, bronchopulmonary sequestration, congenital diaphragmatic hernia, chest wall hamartoma (uncommon)6.Pulmonary vein atresia7.Idiopathic

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Notes:

FETAL CARDIAC ANOMALIES

*Incidence:*1:125 births = 0.8% of population; most common of all congenital malformations (40%)90% occur as isolated multifactorial traits with a recurrence risk of 2-4%10% are associated with multiple birth defectsresponsible for 50% of childhood deaths from congenital malformationsAntenatal sonographic diagnosis to prompt cardiac evaluation: A.ABNORMALITIES IN CARDIAC POSITIONB. CNS1.Hydrocephalus2.Microcephaly3.Agenesis of corpus callosum4.Encephalocele (Meckel-Gruber syndrome)C.GASTROINTESTINAL1.Esophageal atresia2.Duodenal atresia3.Situs abnormalities4.Diaphragmatic herniaD.VENTRAL WALL DEFECT1.Omphalocele2.Ectopia cordisE. RENAL1.Bilateral renal agenesis2.Dysplastic kidneysF.TWINS1.Conjoined twins

Prenatal Risk Factors For Congenital Heart Disease In Utero Detection Of Cardiac Anomalies Structural Cardiac Abnormalities & Fetal Hydrops Fetal Echocardiographic Views

Notes:

Prenatal Risk Factors For Congenital Heart Disease A.FETAL RISK FACTORS1.Symmetric IUGR2.Arrhythmias(a)fixed bradycardia (50%)(b)tachycardia (low risk)(c)irregular: PACs, PVCs (low risk)3.Abnormal fetal karyotype (CHD in Down syndrome in 40%; in Trisomy 18 / 13 in >90%; in Turner syndrome in 35%)4.Extracardiac somatic anomalies by US: omphaloceles (20%), duodenal atresia, hydrocephaly, spina bifida, VACTERL5.Nonimmune hydrops (30-35%)6.Oligo- / polyhydramnios
B.MATERNAL RISK FACTORS1.Maternal heart disease (10%)2.Insulin-dependent diabetes mellitus (4-5%)3.Phenylketonuria (15% if maternal phenylalanine >15%)4.Collagen vascular disease: SLE5.Viral infection: rubella6.Drugs(a)phenytoin (in 2% PS, AS, coarctation, PDA)(b)trimethadione (in 20% transposition, tetralogy, hypoplastic left heart)(c)sex hormones (in 3%)(d)lithium (7%): Ebstein anomaly, tricuspid atresia(e)alcohol (25% of fetal alcohol syndrome): VSD, ASD(f)retinoic acid = isotretinoin (?15%)7.Paternal CHD (risk uncertain)
C.MENDELIAN SYNDROMES1.Tuberous sclerosis2.Ellis-van Creveld syndrome3.Noonan syndrome
D.FAMILIAL RISK FACTORS FOR RECURRENCE OF HEART DISEASE-overall incidence: 6-8:1,000 livebirths-affected sibling: 1-4% (risk doubled)-affected parent: 2.5-4%
In 50% of neonates with CHD there is no identifiable risk factor!*Poor prognostic features:* (1)Intrauterine cardiac failure (hydrops)(2)Severe trisomy (18, 13)(3)Hypoplastic left heart + endocardial fibroelastosis(4)Delivery in center without pediatric cardiology

Notes:

In Utero Detection Of Cardiac Anomalies A.ABNORMAL HEART POSITION1.Diaphragmatic hernia2..Lung anomaly3.Pleural effusion4.Cardiac defectB.CHAMBER ENLARGEMENTRA:LA:1.Tricuspid regurgitation1.Mitral stenosis2.Tricuspid valve dysplasia2.Aortic stenosis3.Ebstein anomalyRV:LV:1.Coarctation1.Aortic stenosis2.Normal in 3rd trimester2.CardiomyopathyC.ABNORMAL FOUR-CHAMBER VIEW1.Septal rhabdomyoma2.Endocardial cushion defect3.Ventricular septal defect4.Ebstein anomaly5.Single ventricleD.VENTRICULAR DISPROPORTION1.Hypoplastic right / left ventricle2.Hypoplastic aortic arch3.Aortic / subaortic stenosis4.Coarctation of aorta5.Ostium primum defectE. INCREASED AORTIC ROOT DIMENSION1.Tetralogy of Fallot2.Truncus arteriosus3.Hypoplastic left ventricle with transpositionF.DECREASED AORTIC ROOT DIMENSION1.Coarctation of aorta2.Hypoplastic left ventricle◊26-80% of serious cardiac anomalies can be detected on four-chamber view!◊Increased sensitivity >20 weeks + by including outflow views!

Notes:

Structural Cardiac Abnormalities & Fetal Hydrops 1.Atrioventricular septal defect + complete heart block2.Hypoplastic left heart3.Critical aortic stenosis4.Cardiac tumor5.Ectopia cordis6.Dilated cardiomyopathy7.Ebstein anomaly8.Pulmonary atresia

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Notes:

Fetal Echocardiographic Views A.FOUR-CHAMBER VIEW1.Position of heart within thorax2.Number of cardiac chambers3.Ventricular proportion4.Integrity of atrial + ventricular septa5.Position + size + excursion of AV valvesB.PARASTERNAL LONG-AXIS VIEW1.Continuity between ventricular septum + anterior aortic wall2.Caliber of aortic outflow tract3.Excursion of aortic valve leafletsC.SHORT-AXIS VIEW OF OUTFLOW TRACTS1.Spatial relationship between aorta + pulmonary artery2.Caliber of aortic + pulmonary outflow tractsD.AORTIC ARCH VIEW

Notes:

FETAL GASTROINTESTINAL ANOMALIES
1.Esophageal atresia ± TE fistula2.Duodenal atresia3.Meconium peritonitis4.Hirschsprung disease5.Choledochal cyst6.Mesenteric cyst

Abdominal Wall Defect Nonvisualization Of Fetal Stomach Double Bubble Sign Dilated Bowel In Fetus Bowel Obstruction In Fetus Hyperechoic Fetal Bowel Intraabdominal Calcifications In Fetus Cystic Mass In Fetal Abdomen Fetal Ascites

Notes:

Abdominal Wall Defect *Prevalence:*1:2,000 pregnancies1.Gastroschisis2.Omphalocele:-upper abdominal wall defect3.Ectopia cordis4.Pentalogy of Cantrell-midabdominal wall defect: classic omphalocele-lower abdominal wall defect5.Bladder exstrophy6.Cloacal exstrophy7.Amniotic band syndrome8.Limb-body wall complex

Notes:

Nonvisualization Of Fetal Stomach ◊Fetal swallowing begins at 11 weeks MA*Incidence:*2% (stomach is visualized in almost all normal fetuses by 14 weeks + in all normal fetuses by 19 weeks)1.Physiologic gastric emptying / intermittent swallowing (repeat scan after 30 minutes)2.Decreased amniotic fluid volume3.CNS abnormalities that impair swallowing4.GI tract abnormalities:(a)congenital diaphragmatic hernia(b)esophageal atresia ± TE fistula◊Nonvisualization of fetal stomach and polyhydramnios in 33% fetuses with esophageal atresia after 24 weeks MA!5.Cleft palate

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Notes:

Double Bubble Sign =fluid filled stomach + proximal duodenum◊A persistently fluid-filled duodenum is always abnormal!1.Duodenal atresia (usually not seen <24 weeks MA)*Cause:*in 30% due to trisomy 212.Duodenal stenosis3.Duodenal web4.Annular pancreas5.Preduodenal portal vein6.Ladd bands7.Malrotation*mnemonic:*"LADS"**L**add bands / malrotation **A**nnular pancreas **D**uodenal atresia **S**tenosis (duodenal)

Notes:

Dilated Bowel In Fetus 1.Meconium ileus◊All newborns with meconium ileus have cystic fibrosis!◊10-15% of newborns with cystic fibrosis present with meconium ileus!2."Apple peel" atresia of small bowel3.Jejunal atresia4.Megacystic-microcolon-intestinal hypoperistalsis syndrome5.Colonic aganglionosis = Hirschsprung disease (may be associated with Down syndrome)6.Anorectal atresia (associated with CNS abnormalities, part of VACTERL complex)

Notes:

Radiology Review Manual
fourth edition

Bowel Obstruction In Fetus *Etiology:*intestinal atresia / stenosis secondary to vascular accident, volvulus, meconium ileus, intussusception after organogenesis*Incidence:*imperforate anus 1:3,000; small bowel 1:5,000; colon 1:20,000*Pathologic types:* Ione / more transverse diaphragmsIIblind-ending loops connected by fibrous stringIIIcomplete separation of blind-ending loopsIVapple-peel atresia of small bowel (occlusion of SMA branch)*Associated with:*GI anomalies in 45% (malrotation, duplication, microcolon, esophageal atresia)√ multiple distended bowel loops >7 mm in diameter√ increased peristalsis√ polyhydramnios (if obstruction above level of mid jejunum; exceptions are esophageal atresia + TE fistula) due to fetal inability to cycle amniotic fluid through gut*Cx:*Meconium peritonitis (50%)*DDx:*(1)Other cystic masses: duodenal atresia, hydronephrosis, ovarian cyst, mesenteric cyst(2)Chronic chloride diarrhea

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Notes:

Hyperechoic Fetal Bowel *Definition:*bowel echogenicity ≥ bone*Incidence:*0.2-0.6% of 2nd trimester fetuses*Cause:*(?) "constipation" in utero due to decreased swallowing, hypoperistalsis, bowel obstruction + increased fluid absorption1.Normal small bowel variant (especially <20 weeks MA) with resolution on follow-up sonogram toward end of 2nd trimester (55-68%)2.Meconium ileus◊Increased abdominal echogenicity is seen in 60-70% of fetuses with cystic fibrosis!3.Meconium peritonitis*Cause:*(a)intestinal atresia with perforation(b)CMV infection4.Chromosomal abnormality (3-25%)(a)Down syndrome (5-14%)(b)Trisomy 13, 18(c)Turner syndrome5.Severe IUGR (16%)*Prognosis:* 5-fold increase in risk for adverse fetal outcome (due to chromosomal abnormality, other anomalies, placental abruption, perinatal death [8-16%], IUGR [67-23%]) ◊30-50% of fetuses with echogenic bowel in 2nd trimester will have poor outcome!*Management:*parental testing for cystic fibrosis, careful fetal anatomic survey, follow-up for growth assessment

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Notes:

Intraabdominal Calcifications In Fetus A.PERITONEAL1.Meconium peritonitis2.Plastic peritonitis associated with hydrometrocolposB.TUMORS1.Hemangioma / hemangioendothelioma2.Hepatoblastoma3.Metastatic neuroblastoma4.Teratoma5.Ovarian dermoidC.CONGENITAL INFECTION1.Toxoplasmosis2.Cytomegalovirus
◊Isolated liver calcifications are relatively frequent + of no clinical significance!

Notes:

Cystic Mass In Fetal Abdomen A.POSTERIOR MID ABDOMEN1.Cysts of renal origin2.Hydroureteronephrosis3.Multicystic dysplastic kidney4.Paranephric collectionB.RIGHT UPPER QUADRANT1.Liver cyst2.Choledochal cystC.LEFT UPPER QUADRANT1.Splenic cystD.ANTERIOR MID ABDOMEN1.Gastrointestinal duplication cyst2.Mesenteric cyst3.Meconium pseudocyst4.Dilated bowel5.Urachal cystE.LOWER ABDOMEN1.Adnexal cyst: follicular cyst (most), corpus luteum cyst, theca lutein cyst, paraovarian cyst, teratoma, cystadenoma*Cx of large cysts:* polyhydramnios, dystocia, torsion, respiratory distress *Prognosis:*60% resolve within first 6 months of life2.Hydrometrocolpos3.Meningocele4.Sacrococcygeal teratoma

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Notes:

Fetal Ascites A.ASCITES + FETAL HYDROPS1.Immune hydrops2.Nonimmune hydropsB.ISOLATED ASCITES1.Urinary ascites2.Meconium peritonitis3.Bowel rupture4.Ruptured ovarian cyst5.Hydrometrocolpos6.Glycogen storage disease

Notes:

FETAL URINARY TRACT ANOMALIES

*Incidence:*0.25%-1% liveborn infants (OB-US);1:100-1:200 neonates (pediatrics)

1.Bilateral renal agenesis2.Infantile polycystic kidney disease3.Adult polycystic kidney disease4.Multicystic dysplastic kidney5.Ureteropelvic junction obstruction6.Megaureter7.Posterior urethral valves8.Prune belly syndrome9.Megacystis-microcolon-intestinal hypoperistalsis syndrome10.Mesoblastic nephroma11.Wilms tumor12.Neuroblastoma*Associated with:*chromosome abnormalities in 12% (74% trisomy, 10% deletion, 9% sex chromosome aneuploidy, 6% triploidy)

- fetal urine production:5 mL/hr at 20 weeks MA;56 mL/hr at 40 weeks MA√ bladder volume:1 mL at 20 weeks MA;36 mL at 40 weeks MA√ filling + emptying of fetal urinary bladder occurs every 10 to 30 (range 7 to 43) minutes√ increased renal parenchymal echogenicity indicates renal abnormality in 80%√ fetal hydronephrosis= AP diameter of renal pelvis >5 mm at 15-20 weeks, ≥8 mm at 20-30 weeks, ≥10 mm at >30 weeks

Notes:

Precocious Puberty =early onset of puberty ● premature thelarche / adrenarche / menses***Isolated*** Premature Adrenarche =pubic hair development due to action of adrenal androgens ● increased levels of adrenal androgens√ prepubertal uterus + ovaries (0.1-1 cm^3)
Isolated Premature Thelarche =breast enlargementmay occur without endocrine abnormalities √ prepubertal uterus + ovaries
Pseudoprecocious Puberty =PSEUDOSEXUAL PRECOCITY = incomplete precocious puberty=pubertal changes occurring independently of the action of pituitary gonadotropins, ie, early development of secondary sex characteristics without ovulation*Cause:*ovarian tumor (eg, granulosa theca-cell tumor, thecoma, choriocarcinoma), ovarian cyst, estrogen-producing adrenal tumor, hypothyroidism, neurofibromatosis, estrogen ingestion ● low gonadotropin levels after LHRH stimulation ● increased estradiol levels√ prepubertal uterus + ovaries√ asymmetric ovarian enlargement (one ovary 2.4-7 cm^3) with macrocysts (>9 mm)
True Precocious Puberty = TRUE ISOSEXUAL PRECOCITY = complete precocious puberty=early development of gonads + secondary sex characteristics with ovulation before 8 years of age*Cause:* (1)Idiopathic activation of hypothalamic-pituitary-gonadal axis (80%)(2)Lesion of pituitary gland / hypothalamus ● increased levels of estrogen ● increased gonadotropin levels after LHRH stimulation ● advanced bone age√ adult-sized ovaries (1.2-12 cm^3)√ dominance of corpus over cervix length

Notes:

Amenorrhea ***Primary Amenorrhea*** =failure to menstruate by 16 years of age*Cause:* A.FEMALE ANATOMIC ANOMALIESB.CONGENITAL DISORDERS OF SEXUAL DIFFERENTIATION(a)pure gonadal dysgenesis√ bilateral dysfunctional / streak gonads(b)mixed gonadal dysgenesis√ testis + streak gonad*Risk:*in 25% development of dysgerminoma / gonadoblastoma in dysgenetic gonads with Y chromosomeC.OVARIAN FAILURE / DYSFUNCTIOND.HYPOTHALAMIC / PITUITARY CAUSES

√ absent / streak gonads + infantile uterus:1.Hypogonadotropic hypogonadism(a)hypothalamic dysfunction: hypothalamic tumor, Kallmann disease (= lack of pulsatile GnRH release), systemic illness, constitutional growth delay, extreme physical / psychological / nutritional stress (cystic fibrosis, sickle cell disease, Crohn disease)(b)pituitary dysfunction: disruption of pituitary stalk from child abuse, head trauma2.Hypergonadotropic hypogonadism=ovarian tissue fails to respond to endogenous gonadotropins(a)abnormal karyotype: Turner syndrome, XY gonadal dysgenesis(b)radiation, chemotherapy, autoimmune disease

√ absent uterus:1.Testicular feminization = male intersex = male pseudohermaphroditism (end-organ insensitivity to testosterone)2.Müllerian dysgenesis (= Mayer-Rokitansky-Küster-Hauser syndrome)√ normal fallopian tubes + ovaries*associated with:* unilateral renal abnormality (50%), skeletal abnormality (12%)

√ small infantile uterus:1.Androgen-producing virilizing tumors of adolescent ovary (usually Sertoli-Leydig cell tumor)√ unilateral adnexal mass2.Turner syndrome3.In utero exposure to diethylstilbestrol

√ normal uterus + unilateral ovarian tumor:1.Estrogen-producing with disruption of menstrual cycle: granulosa cell tumor, thecoma

√ hydrometrocolpos:1.Vaginal membrane / septum

√ bilateral ovarian enlargement:1.Polycystic ovary syndrome (= Stein-Leventhal syndrome): most common cause of secondary amenorrhea

Secondary Amenorrhea 1.Pregnancy: most common cause in girls >9 years of age2.Polycystic ovary syndrome3.Asherman syndrome4.All causes of primary amenorrhea

Notes:

Calcifications Of Female Genital Tract A.UTERUS1.Uterine fibroid2.Arcuate arteriesB.OVARIES1.Dermoid cyst (50%)2.Papillary cystadenoma (psammomatous bodies)3. Cystadenocarcinoma4. Hemangiopericytoma5. Gonadoblastoma6. Chronic ovarian torsion7. Pseudomyxoma peritoneiC.FALLOPIAN TUBES1.Tuberculous salpingitisD.PLACENTAE.LITHOPEDION

Notes:

Free Fluid In Cul-de-sac 1.Follicular rupture2.Ovulation3.Ectopic pregnancy4.S/P culdocentesis5.Ovarian neoplasm6.Pelvic inflammatory disease

Notes:

Frequency Of Pelvic Masses 1.Benign adnexal cyst34%2.Leiomyoma14%3.Cancers14%4.Dermoid13%5.Endometriosis10%6.Pelvic inflammatory disease 8%

Notes:

Cystic Pelvic Masses A.CYSTIC ADNEXAL MASSB.EXTRAADNEXAL CYSTIC MASS1.Peritoneal inclusion cyst2.Mesenteric cyst3.Lymphocele4.Bladder diverticulum5.Ectopic gestation6.Fluid-distended bowel7.Loculated pelvic abscess: appendiceal, diverticular, postoperative

Notes:

Complex Pelvic Mass *mnemonic:*"CHEETAH"**C**ystadenoma / cystadenocarcinoma **H**emorrhagic cyst **E**ndometrioma **E**ctopic pregnancy **T**eratoma (dermoid) **A**bscess (from adjacent appendicitis, etc.) **H**ematoma in pelvis

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Notes:

Solid Pelvic Masses 1.Pedunculated myoma (most common)2.Fibroma3.Adenofibroma4.Thecoma5.Brenner tumor

Notes:

Extrauterine Pelvic Masses 1.Solid adnexal mass2.Metastatic disease3.Lymphoma4.Pelvic kidney5.Rectosigmoid carcinoma6.Prostate carcinoma7.Benign prostatic enlargement8.Bladder carcinoma9.Retroperitoneal tumor10.Intraperitoneal fat11.Vascular mass / malformation12.Hematoma13.Bowel

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Notes:

Adnexal Masses A.CYSTIC1.Physiologic ovarian cyst:-Graafian follicle:at midcycle <25 mm-Corpus luteum:after midcycle <15 mm2.Functional / retention cyst3.Endometrioma4.Tuboovarian abscess5.Dermoid cyst6.Ectopic pregnancy7.Paraovarian cyst8.Serous / mucinous cystadenoma9.Serous / mucinous cystadenocarcinoma10.Hyperstimulation cysts11.Peritoneal inclusion cyst12.Massive ovarian edema13.HydrosalpinxB.SOLID1.Ovarian tumor2.Ovarian torsion3.Oophoritis4.Polycystic ovaries5.Fallopian tube carcinoma(DDx: pedunculated fibroid)

Notes:

Ovarian Tumors ● pressure symptoms: abdominal discomfort, vomiting, flatulence, dyspnea ● acute pain from torsion, hemorrhage ● chronic pain from slowly enlarging mass, impaction, adhesions ● menstrual irregularity*Radiologic guidelines:* ◊Imaging features of ovarian neoplasms virtually never allow a specific diagnosis. Regardless of further differentiation patients always undergo surgery!Signs suggestive of malignancy: √ solid ovarian tumor√ many solid-tissue elements in a complex lesion√ wall thickness >3 mm√ inner wall irregularities√ thick septations >3 mm√ increased echogenicity within a cyst*Age:*13% of neoplasms malignant in premenopause; 45% of neoplasms malignant in postmenopause*Cx:*(1)Torsion (in 10-20%)(2)Rupture (rare)(3)Infection
Classification: A.TUMORS OF SURFACE EPITHELIUM (60%)85-95% of all ovarian cancers (although majority of epithelial tumors are benign) 1.Serous ovarian tumor2.Mucinous ovarian tumor3.Endometrioid tumor4.Cystadenofibroma5.Clear cell adenocarcinoma6. Brenner tumor7.Undifferentiated carcinomaB.GERM CELL TUMORS (30%)40% of germ cell tumors are malignant (a)benign1.Dermoid cyst = mature teratoma (most common)(b)malignantaccount for 75% of ovarian cancers seen in 1st-2nd decade of life; <5% of all ovarian tumors; in order of frequency: 1.Dysgerminoma2.Immature teratoma3.Endodermal sinus tumor4.Embryonal carcinoma5.ChoriocarcinomaC.GONADAL STROMAL TUMORS (5%)(a)Sex cord-mesenchyme tumors1.Granulosa cell tumor2.Theca cell tumor 3.Luteal cell tumor4.Arrhenoblastoma(b)Connective tissue tumor1.Fibroma2.Fibrosarcoma-estrogen-producing tumors: granulosa cell tumor, theca cell tumor = thecoma-androgen-producing tumors: arrhenoblastoma, Sertoli-Leydig cell tumor, clear cell tumorD.SECONDARY OVARIAN TUMORS (5%)Metastases from: pelvic organs, upper GI tract, breast, bronchus, reticuloendothelial tumors, leukemia
Subclassification: adenomaborderlineadenocarcinoma
serous60%15%25%mucinous80%10%10%endometrioidalmost alwaysclear cellalmost alwaysundifferentiatedalways
Terminology: prefix "cyst-":cystic component presentsuffix "-fibroma":>50% fibrous component"tumor of low malignant potential":borderline malignant
Solid Ovarian Tumor 1.Fibroma2.Thecoma3.Granulosa cell tumor4.Sertoli-Leydig cell tumor5.Brenner tumor6.Sarcoma7.Dysgerminoma8.Endodermal sinus tumor9.Teratoma10.Metastasis11.Endometrioma12.Massive ovarian edema

Notes:

Ovarian Cyst ***Image Signature Of Ovarian Cysts*** A.SIMPLE CYST=sharply defined wall; NO internal septations / mural nodulesUS: √ pulsatility index >1.0 / RI >0.4 (unreliable!)MR: √ isointense to urine on T1WI + T2WIB.COMPLEX CYST=does not satisfy criteria for hemorrhagic cysts / endometrioma√ internal septations / mural nodules / internal echoes√ mixed signal intensity, hyperintense on T2WIC.HEMORRHAGIC CYSTUS: √ echogenic mass√ whirled pattern of mixed echogenicity√ "ground-glass" pattern = diffuse low-level echoes√ "fishnet weave" pattern = fine interdigitating septations√ NO color Doppler signalsMR: √ intermediate / high intensity on T1WI√ hyperintense with distinct central area of hypointensity on T2WI
Management Of Ovarian Cyst A.PREMENOPAUSAL1.Unilocular cyst ≤2.5 cm ± hemorrhage*Rx:*no follow-up unless on birth control pills2.Unilocular thin-walled cyst 2.5-6 cm without hemorrhage*Rx:*clinical / sonographic follow-up in 1-2 months ± addition of hormones3.Unilocular cyst 2.5-6 cm with hemorrhage*Rx:*sonographic follow-up in 1 month ± addition of hormones4.Unilocular cyst >6 cm*Rx:*surgeryN.B.:All follow-up scans should take place in the immediate postmenstrual period, when follicular cysts should not be present!
B.POSTMENOPAUSAL1.Unilocular nonseptated thin-walled cyst <3 cm*Incidence:*15-17%√ high resistive index (RI) of >0.7 (resistive index <0.40 is suspect for malignancy!)*Prognosis:*56% decrease in size / disappear; 28% remain unchanged for up to 2 years*DDx:*serous ovarian cyst, peritubal cyst, hydrosalpinx*Rx:*serial follow-up2.Septated cyst / cyst >3 cm / cyst with low RI◊18% of complex cysts are malignant!*Rx:*CA-125 determination + surgical exploration◊Screening of 1300 symptomatic women:-in 2.5% abnormalities on US-in 1.9% benign ovarian tumors-in 0.15% ovarian cancers

Notes:

Postmenopausal Bleeding 1. Endometrial atrophy (most commonly) ● thin atrophic endometrium is prone to superficial ulceration√ in 75% endometrial thickness <4-5 mm√ in 25% endometrial thickness of 6-15 mm2.Endometrial adenomatous hyperplasia√ thickened homogeneous texture3.Endometrial polyp√ cystic endometrial spaces4.Submucosal fibroid5.Endometrial carcinoma (in 7-30%)10% cancer rate with endometrial thickness of 6-15 mm 50% cancer rate with endometrial thickness of >15 mm √ heterogeneous endometrium√ irregular poorly defined endometrial-myometrial interface

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Notes:

Thickened Irregular Endometrium Normal endometrial thickness: <1 cm
1. **Endometrial polyp** =focal hyperplasia of stratum basale; in 20% multiple*Age:*mainly 30-60 years*Histo:*projections of endometrial glands + stroma into uterine cavity(a)hyperplastic polyp resembling endometrial hyperplasia(b)functional polyp resembling surrounding endometrium (least frequent)(c)atrophic polyp√ enlarged cystically dilated glands√ well-defined homogeneous hyperechoic intracavitary mass√ heterogeneous texture suggests infarction, cystic changes, hemorrhageMalignant transformation:in 0.4-3.7%2. **Endometrial hyperplasia** *Age*:peri- / postmenopausal women*Cause:*prolonged endogenous / exogenous unopposed estrogen stimulation√ endometrial thickening >5-6 mm *Types:* (a)glandular-cystic hyperplasia (more common)*Histo:*dilated glands lined by tall columnar / cuboidal epithelium√ small cysts within evenly echogenic endometrium*Prognosis:*NO premalignant condition(b)adenomatous hyperplasia√ endometrium with irregular hypoechoic areas*Prognosis:*precursor of endometrial cancer3.Endometritis4.Primary carcinoma of the endometriumLocation:predominantly in uterine fundus; 24% in isthmic portion)√ irregular heterogeneous endometrium√ mean endometrial thickness of 18.2 mm5.Tamoxifen-related endometrial changes=nonsteroidal antiestrogen may act as partial estrogen agonist with proliferative effects on endometrium6.Metastatic carcinoma:ovary, cervix, fallopian tube, leukemia 7.Hydatidiform mole√ echogenic mass with irregular sonolucent areas8.Incomplete abortion9.Submucosal leiomyoma

Notes:

Fluid Collection Within Endometrial Canal *Types:*blood, mucus, purulent materialA.PREMENOPAUSAL1.Congenital obstructive lesion: imperforate hymen, vaginal septum, vaginal / cervical atresia2.Acquired obstructive lesion: cervical stenosis (following instrumentation / radiation), cervical carcinoma3.Spontaneous hematometra in bleeding disorders4.Pregnancy: intrauterine, ectopic, incomplete abortionB.POSTMENOPAUSAL1.Cervical stenosis2.Pyometrium3.Polyps4.Endometrial / cervical / ovarian cancer

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Notes:

Endometrial Cysts 1.Endometrial cystic atrophy*Histo:*cystically dilated atrophic glands lined by single layer of flattened / low cuboidal epithelium very thin endometrium of <4-5 mm2.Endometrial cystic hyperplasia

Notes:

Diffuse Uterine Enlargement 1.Diffuse leiomyomatosis2.Adenomyosis3.Endometrial carcinoma (15%)

Notes:

Uterine Masses A.BENIGN1.Uterine fibroids (99%)2.Pyometra3.Hemato- / hydrocolpos4.Transient uterine contraction (during pregnancy)5.Bicornuate uterus6.Adenomyosis7.Intrauterine pregnancy8.Lipoleiomyoma (<50 cases in world literature)B.MALIGNANT1.Cervical carcinoma2.Endometrial carcinoma3.Leiomyosarcoma4.Invasive trophoblastic disease

Notes:

Fundic Depression On HSG 1.Bicornuate uterus2.Septate uterus3.Arcuate uterus4.Fundal myoma

Notes:

Vaginal Cyst 1.Gartner duct cyst2.Bartholin gland cyst=female homologue of male Cowper glandsLocation:posterolateral portion of lower vagina3.Paramesonephric / müllerian duct cyst=aberrant remnant of paramesonephric ductLocation:anterior wall of vagina near cervix4.Epithelial inclusion cyst=arise from urogenital sinus*Histo:*lined by transitional epithelium containing thick caseous material

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Notes:

Vaginal Fistula 1.Enterovaginal fistula(a)rectovaginal: incomplete healing of perineal laceration from obstetric trauma, radiation therapy(b)anovaginal: inflammatory bowel disease (10% of patients with Crohn disease)(c)colovaginal: diverticulitis2.Vesicovaginal fistula: hysterectomy, radiation therapy3.Ureterovaginal fistula: vaginal hysterectomy

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Notes:

Vaginal & Paravaginal Neoplasm A.PRIMARY1.Cavernous hemangioma of vulva2.Pedunculated submucosal leiomyoma prolapsed into vagina3.Adenoid cystic carcinoma of Bartholin gland4.Vaginal carcinoma(a)squamous cell carcinoma (90%)(b)adenocarcinoma (3%)5.Rhabdomyosarcoma
B.SECONDARY (80% of all vaginal tumors)direct extension from bladder, rectum, cervix, uterus

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Notes:

GAS IN GENITAL TRACT

A.UTERUS1.Endometritis2.Superinfection of leiomyoma: more common in submucosal leiomyoma (insufficient blood supply)3.Bacterial metabolism of necrotic neoplastic tissue4.Fistula to GI tract: uterine cancer5.Pyometra secondary to obstruction by cervical cancer6.Gas gangrene: due to clostridial infection from septic abortion

B.OVARY1.Superinfected ovarian neoplasm

C.VAGINA1.Vaginitis emphysematosa = nonbacterial self-limiting process mostly occurring during pregnancy characterized by numerous gas-filled spaces in submucosa of vagina + exocervix

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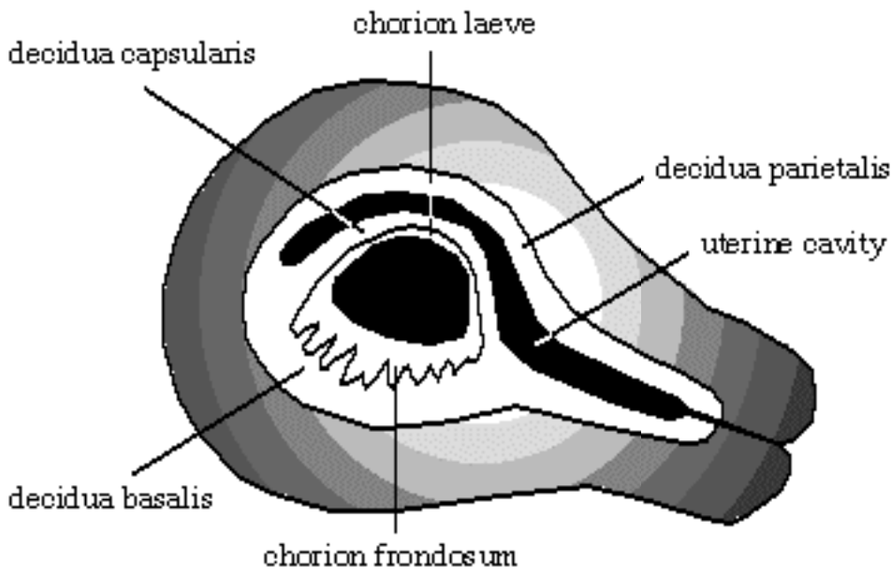
Notes:

HUMAN CHORIONIC GONADOTROPIN
= HCG = glycoprotein elaborated by placental trophoblastic cells beginning the 8th day after conception
A.IMMUNOLOGIC PREGNANCY TEST=indirect agglutination test for HCG in urine; cross-reaction with other hormones / medications possible Becomes positive at 5 weeks MA *Advantages:*readily available, easily + rapidly performed*Disadvantages:*frequently false-positive + false-negative results*Sensitivity:* (a)slide: 400-15,000 mIU/mL (2 min test time)(b)test tube: 1,000-3,000 mIU/mL (2 hours test time)
B.RADIOIMMUNOASSAY (RIA) PREGNANCY TEST=measures beta subunit of HCG in serum with a sensitivity as low as 1-2 mIU/mL◊Serum b-HCG becomes positive at 3 weeks MA / 7-10 days following conception!*Standards:* (1)Second International Standard (SIS)(2)International Reference Preparation (IRP)(3)Third International Standard (TIS)1 mIU/mL (SIS) = 2 mIU/mL (IRP) = 2 mIU/mL (TIS) 1 ng/mL = 5- 6 mIU/mL (SIS)= 10-12 mIU/mL (IRP or TIS)◊Variations of lab values of up to 50% can occur among different laboratories!◊6-15% between-run precision!*Advantages:*specific for HCG, sensitive*Disadvantages:*requires specialized lab + 3-24 hours for completion*Sensitivity:* (a)qualitative: 25-30 mIU/mL (3 hours test time)(b)quantitative: 3-4 mIU/mL (24 hours test time)*Rise:* >66% increase of initial b-HCG level over 48 hours in 86% of NORMAL pregnancies <66% increase of initial b-HCG level over 48 hours in 87% of ECTOPIC pregnancies◊b-HCG levels double every 2-3 days during first 60 days of pregnancy!

"1-7-11 rule": b-HCG (IRP)US landmarksGestational age1,000 mIU/mLgestational sac32 d(<5 weeks)7,200 mIU/mLyolk sac36 d(5 weeks)10,800 mIU/mLembryo + heart motion40 d(<6 weeks)

Notes:

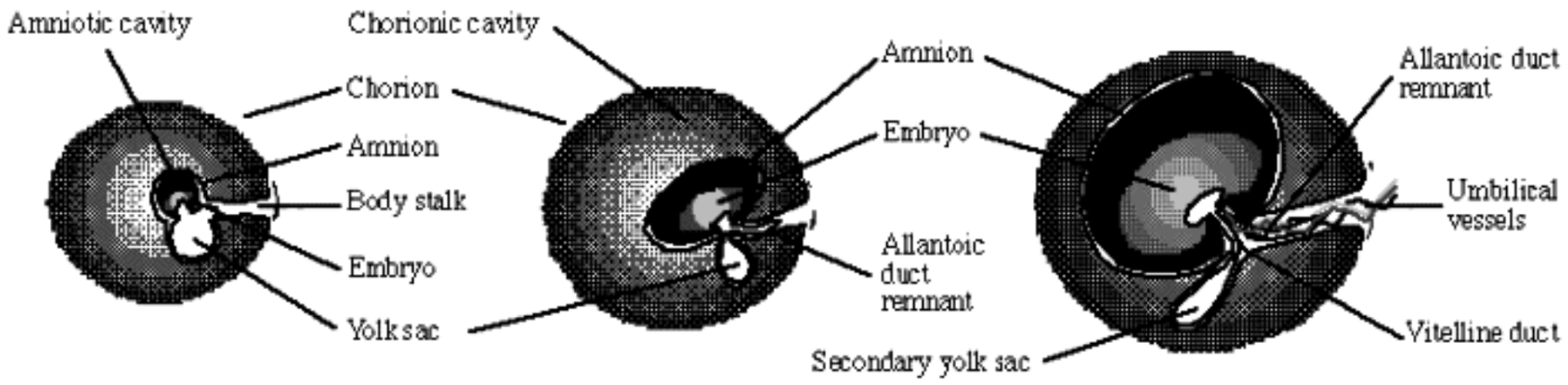
Choriodecidua



Chorion =trophoblast + fetal mesenchyme with villous stems protruding into decidua; provides nutrition for developing embryo(a)chorion frondosum = part adjacent to decidua basalis, forms primordial placenta(b)chorion laeve = smooth portion of chorion with atrophied villi(c)"chorionic plate" = amnionic membrane covering the chorionic plate of the placenta ***Decidua*** (a)decidua basalis = between chorion frondosum + myometrium(b)decidua capsularis = portion protruding into uterine cavity(c)decidua parietalis = decidua vera = portion lining the uterine cavity elsewhere

Notes:

Gestational Sac



Simple double bleb stage
earliest detection at 5 weeks GA, embryo 2 mm in length

Vitelline duct
8 weeks GA

Early coiled umbilical cord
9 weeks GA

Arises from blastocyst which implants into secretory endometrium 6-7 days after ovulation, surrounded by echogenic trophoblast √ intradecidual sign (earliest sign) = intrauterine fluid collection corresponding to gestational sac completely embedded within decidua (48% sensitive, 66% specific, 45% accurate)√ double decidual sac sign (DDS) [most useful at 4-6 weeks GA] = 2 concentric rings (decidua parietalis adjacent to decidua capsularis) surrounding a portion of the gestational sac◊A double decidual sac sign correlates with the presence of pregnancy in 98%√ GS surrounded by endometrial thickening >12 mm√ continuous hyperechoic inner rim >2 mm thick√ spherical / ovoid shape without angulations√ mean sac diameter grows 1.13 (range 0.71-1.75) mm/day***Gestational Sac Size*** linear growth:10 mmby5th week MA60 mmby12th week MAfills chorionic cavity by 11-12 weeks MA ***Visualization Of Gestational Sac*** Earliest visualization:mean sac diameter of 2-3 mmA.GS VISUALIZATION VERSUS b-HCG LEVEL (2nd International Standard):(a)on transabdominal scan:in100%with b-HCG levels of>1,800 IU/L(b)on transvaginal scan:in 20%with b-HCG levels of<500 IU/Lin 80%with b-HCG levels of500-1,000 IU/Lin100%with b-HCG levels of>1,000 IU/L B.GS VISUALIZATION VERSUS MENSTRUAL AGE(a)on transabdominal scan:5.0 ± 1 weeks=5-10 mm5.5 ± 1 weeks=8.5-13 mm6.0 ± 1 weeks=12-17 mm(b)on transvaginal scan:5.0 ± 1 weeks=2 mm5.5 ± 1 weeks=6 mm6.0 ± 1 weeks=11 mm C.GS VISUALIZATION VERSUS VISUALIZATION OF EMBRYO(a)on transabdominal scan100% visualization if gestational sac ≥27 mm (b)on transvaginal scan100% visualization if gestational sac ≥12 mm ◊Transvaginal scan not necessary if on transabdominal scan gestational sac >27 mm without evidence of embryo! *Predictive of miscarriage (in 94%):* "first-trimester oligohydramnios" (misnomer: not diminished size of amnionic cavity but rather chorionic cavity) =mean sac diameter - CRL ≤ 5 mm (with a live embryo at 5.5-9.0 weeks)

Notes:

Yolk Sac =rounded sonolucent structure (outside amniotic cavity) within chorionic sac (= extracoelomic cavity) connected to umbilicus via a narrow stalk; formed by proliferation of endodermal cells at around 4 weeks MA; part of yolk sac is incorporated into fetal gut; the rest persists as a sac connected to the fetus by the vitelline duct*Function:* (a)transfer of nutrients from trophoblast to embryo prior to functioning placental circulatio(b)early formation of blood vessels + blood precursors on sac wall(c)formation of primitive gut(d)source of primordial germ cells Mean size: 1.0 mm by 4.7 weeks MA; 2.0 mm by 5.6 weeks MA; 3.0 mm by 7.1 weeks MA; 4.0 (2.2-5.3) mm by 10 weeks MA; disappears around 12 weeks MA *Earliest visualization:* ↑ at 4-5 weeks MA as one of the "double blebs" on endovaginal scan; in 65% with GS size of ≥8 mm◊Visualization excludes the possibility of an ectopic / anembryonic pregnancy! *Failed pregnancy:* Abnormal pregnancy outcome (using endovaginal technique) generally if (a)yolk sac absent with GS diameter of ≥20 mm (100% specificity + 100% PPV)(b)yolk sac diameter >5.6 mm at <10 weeks MA(c)embryo visualized without demonstrable yolk sac(d)yolk sac shape persistently abnormal

Notes:

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Embryo *Developmental stages:* Preembryonic period:2nd-4th week MATrilaminar embryonic disk:during 5th week MA3 laminae = ectoderm, endoderm, mesoderm Embryonic period:6th-10th week MAphysiologic umbilical herniation:8th-12th week MAFetal period:beginning at 11th week MAAverage growth rate: 0.7 mm per day / 1.5 mm every 2 days; curvilinear growth from 7 mm at 6.3 weeks MA to 50 mm at 12.0 weeks MA *Earliest visualization:* at 5.4 weeks MA at CRL of 1.2 mm on endovaginal scan *Failed pregnancy:* nonvisualization of embryo with mean gestational sac size of ≥18 mm ***Cardiac Activity*** Heart begins to contract at a CRL of 1.5-3 mm = 6th week MA *Earliest visualization (on endovaginal scan):* (a)in 65% of embryos with a CRL of 2-4.9 mm(b)in 100% at ≥ 5 mm CRL = 6.2 weeks*Failed pregnancy:* nonvisualization of cardiac activity with CRL of 2-12 mm means embryonic demise in 94%! ⚱Spontaneous pregnancy loss at <8 weeks gestation occurs in 10-17% of embryos with cardiac activity! *Embryonic Mortality Rate*≤6.2 weeks≤7.0 weeks 11%>100 bpm>120 bpm32%90-99 bpm110-119 bpm64%80-89 bpm100-109 bpm100%<80 bpm<100 bpm

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Notes:

Amnionic Membrane =curvilinear echogenic line within chorionic sac; fills chorionic cavity by 11-12 weeks MA;*Fusion:* -fuses with chorionic membrane at approximately 16 weeks MA to form the chorionic plate-incomplete fusion with chorion frequent (DDx: subchorionic hemorrhage, twin abortion, coexistent with limb-body wall complex)

Notes:

Umbilical Cord *Embryology:* -cord forms between 5th and 12th postmenstrual week with contributions from body stalk, omphalo-mesenteric or vitelline duct, yolk sac, allantois-junction of the amnion with ventral surface of embryo will form umbilicus-midgut undergoes physiologic herniation into the base of the umbilical cord 7-12 postmenstrual weeks-cord grows until end of 2nd trimester: average diameter of 17 mm, length of 50-60 cmAnatomy: -two umbilical arteries = branches of the two internal iliac arteries-one umbilical vein (remains after regression of right umbilical vein in early embryonic period)-Wharton jelly = compressible matrix of cord-spiraling of cord with 0-40 twists established by 9 weeks

Notes:

Placental Grading according to echo appearance of basal zone, chorionic plate, placental substance◊Premature placental calcification is associated with cigarette smoking, hypertension, IUGR!◊Not considered useful because placental grading is imprecise for fetal dating or for fetal lung maturity!GRADE 0√ homogeneous placenta + straight line of chorionic plate*Time:*<30 weeks MAGRADE 1√ undulated chorionic plate + scattered bright placental echoes*Time:*seen at any time during pregnancy; in 40% at term◊in 68% L/S ratio >2.0GRADE 2√ linear bright echoes parallel to basal plate√ confluent stippled echoes within placenta ± indentations of chorionic plate*Time:*rarely seen in gestations <32 weeks MA; seen in 40% at term◊in 87% L/S ratio >2.0GRADE 3√ calcified intercotyledonary septa, often surrounding sonolucent center*Time:*rarely seen in gestations <34 weeks MA; in 15-20% at term◊in 100% L/S ratio >2.0 (= strongly correlated with lung maturity)
PREMATURE PLACENTAL SENESCENCE =grade 3 placenta seen in gestation <34 weeks MA◊in 50% suggestive of maternal hypertension / IUGR

Notes:

Uteroplacental Circulation By 20 weeks MA trophoblast invades maternal vessels and transforms spiral arteries into distended tortuous vessels = uteroplacental arteries *Histo:* (a)in the decidual portion of spiral arteries: proliferating trophoblast from anchoring villi invades lumen of spiral arteries + partially replaces endothelium(b)in the myometrial portion of spiral arteries: disintegration of smooth muscle elements (loss of elastic lamina) leads to easily distensible vascular system of low resistance ***Uterine Blood Volume Flow*** -50 mL/min shortly after conception-500-900 mL/min by termIntervillous blood flow:140 ± 53 mL/min (by Xe-133 washout)***Umbilical Artery Doppler*** Variables of Doppler measurements: site of Doppler (close to placenta preferred), fetal heart rate, fetal breathing, drugs (ritodrine hydrochloride decreases S/D ratio) √ degree of diastolic flow increases as gestation progresses-S/D ratio between 3.3 and 4.3 at 20 weeks-S/D ratio between 1.7 and 2.4 at term√ highly turbulent flow ***IUGR Lesions*** =narrowing of vascular lumen through(a)thrombosis of decidual segments of uteroplacental arteries(b)failure of development of myometrial segments of uteroplacental arteries

Notes:

FETAL MENSURATION

US is more reliable than LMP / physical examination ULTRASOUND MILESTONES: √ gestational sac w/o embryo or yolk sac=5.0 weeks √ gestational sac + yolk sac w/o embryo=5.5 weeks √ heartbeat ± embryo <5 mm=6.0 weeks*Accuracy:* ± 0.5 week

Fetal Age Gestational Sac Early Embryonic Size Crown-rump Length (CRL) Biparietal Diameter (BPD) Cephalic Index (CI) Corrected BPD (cBPD) Abdominal Circumference (AC) Femur Length (FL) Thoracic Circumference (TC) Estimated Fetal Weight (EFW) Appearance Of Epiphyseal Bone Centers CNS Ventricles Diameter Of Cisterna Magna

Notes:

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Fetal Age = GESTATIONAL AGE (GA) = "MENSTRUAL AGE" (MA)=age of pregnancy based on womans regular last menstrual period (LMP) projecting the estimated date of confinement (EDC) at 40 weeks Note the inaccurate clinical usage of "gestational age," which strictly speaking refers to the true age of the pregnancy counting from the day of conception, whereas "menstrual age" refers to the true age of the pregnancy + approximately 2 weeks counting from the first day of the last menstruation! On subsequent scans GA = GA assigned at 1st ultrasound + number of intervening weeks! ACCURACY (95% confidence range): *StageBased on*Accuracy*[weeks]*
1st trimester (5-6 weeks)US milestones±0.5(6-13 weeks)CRL±0.7*2nd trimester* (14-20 weeks)cBPD / HC±1.2BPD / FL±1.4(20-26 weeks)cBPD / HC±1.9BPD / FL±2.1-2.5*3rd trimester* (26-32 weeks)cBPD / HC / FL±3.1-3.4FL±3.1(32-42 weeks)cBPD / HC / FL±3.5-3.8FL±3.5

Notes:

Gestational Sac =average of 3 diameters (craniocaudad, AP, TRV) of anechoic space within sac walls◊used for dating between 6-12 weeks MA (identified as early as 5 weeks MA (on transabdominal scan)*Accuracy:* ± 1 week

Notes:

Early Embryonic Size =length of embryo <25 mm on transvaginal scan <10 weeks MAGestational age (days) = embryonic size (mm) + 42 *Accuracy:*± 3 days

Notes:

Crown-rump Length (CRL)
=length of fetus; useful up to 12 weeks MA (usually identified by 7 weeks MA on transabdominal scan)Rule of thumb:MA (in weeks) = CRL (in cm) + 6

Notes:

Biparietal Diameter (BPD)
=measured from leading edge to leading edge of calvarial table at widest transaxial plane of skull= level of thalami + cavum septi pellucidi + sylvian fissures with middle cerebral arteries ◊Excellent means of estimating GA in 2nd trimester >12 weeks MA*Accuracy*: 2 mm for "between occasion error" ◊Most accurate for dating if combined with HC, AC, FL provided body ratios are normal◊Less reliable for dating in 3rd trimester because of increasing biologic variability!

Notes:

Cephalic Index (CI)
=BPD / OFD; measurements of BPD and occipitofrontal diameter (OFD) are both taken from outer to outer edge of calvarium Confirms appropriate use of BPD if ratio is between 0.70-0.86 (2 SD)

Notes:

Corrected BPD (cBPD)

=BPD and OFD are used to adjust for variations in head shapecBPD = $\sqrt{}$ BPD x OFD ÷ 1.26 **Head Circumference** (HC) Used if ratio of BPD/OFD outside 0.70-0.86 HC= ([BPD + OFD]/2) x p= ([BPD + OFD] x 1.62) x 3.1417 *Accuracy*:slightly less than for BPDHC too large:hydrocephalus, hydranencephalus, intracranial hemorrhage, short limb dystrophies, tumorHC too small:anencephaly, cerebral infarction, synostosis, microcephaly vera

Notes:

Abdominal Circumference (AC)
=measured at level of vascular junction of umbilical vein with left portal vein ("hockey-stick" appearance) where it is equidistant from the lateral walls in a plane perpendicular to long axis of fetus; measured from outer edge to outer edge of soft tissues◊Allows evaluation of head-to-body disproportion◊Better predictor of fetal weight than BPDAC too large:GI tract obstructions, obstructive uropathy, ascites, hepatosplenomegaly, congenital nephrosis, abdominal tumorAC too small:diaphragmatic hernia, omphalocele, gastroschisis, renal agenesis

Notes:

Femur Length (FL)
=measurement of ossified femoral diaphysis*Error:*"flare" at distal end included in measurement (= reflection from cartilaginous condyle)

Notes:

Thoracic Circumference (TC)
=measured in axial plane of chest which includes four-chamber view of heart without inclusion of SQ tissue Linear growth between 16 and 40 weeks similar to ACUseful age-independent parameter:TC:AC >0.80

Notes:

Estimated Fetal Weight (EFW)
based on measurements of head size (BPD / HC), abdominal size (AD / AC), and femur length (FL) *Accuracy:***body part used95% confidence range**abdomen±22%head + abdomen±17-20%head + abdomen + femur±15%

Notes:

Appearance Of Epiphyseal Bone Centers in 95% of all cases -distal femoral epiphysis (DFE): >33 weeks GA-distal femoral epiphysis (DFE) >5 mm: >35 weeks-proximal tibial epiphysis (PTE): >35 weeks GA-proximal humeral epiphysis (PHE): >38 weeks GA

Notes:

CNS Ventricles width of 3rd ventricle:<3.5 mm (any gestational age)

Notes:

Diameter Of Cisterna Magna measured from inner margin of occiput to vermis cerebelli: 2-10 mm DISCORDANT ESTIMATED DATE OF CONFINEMENT (EDC) BY LMP AND BPD: 1.Methodological error in measurement(a)wrong axial section(b)cranial compression (multiple gestation, breech presentation, oligohydramnios, dolichocephaly)2.Erroneous LMPother measurements (AC, FL) correlate with BPD 3.Abnormal head growth(a)BPD less than AC: microcephaly, fetal macrosomia(b)BPD more than AC: intracranial abnormality, asymmetric IUGR

Notes:

Amniotic Fluid Index =sum of vertical depths of largest clear amniotic fluid pockets in the 4 uterine quadrants measured in mm*Method:*patient supine, uterus viewed as 4 equal quadrants, transducer perpendicular to plane of floor + aligned longitudinally with patient's spine*Variation:*3.1% intraobserver, 6.7% interobserverResult: -95th percentile:185 mm at 16 weeks GA,rising to 280 mm at 35 weeks, declining to 190 at 42 weeks-5th percentile:80 mm at 16 weeks GA,rising to 100 mm at 23 weeks,declining to 70 mm at 42 weeks

Notes:

Biophysical Profile (Platt and Manning) = BPP
=in utero Apgar score = assessment of fetal well-beingGestational age at entry:25 weeks MAObservation period:30 (occasionally 60) minutes; ordinarily <8 minutes needed; in 2% full 30 minutes required A.ACUTE BIOPHYSICAL VARIABLES◊Subject to rhythmic variation coincident with sleep-wake cycle!1.Fetal breathing movement (FBM):√ ≥1 episode of chest + abdominal wall movement for a period lasting 30 seconds (time is arbitrary to avoid confusion with general body movements / maternal respiration)stimulated by:glucose, catecholamine, caffeine, prostaglandin synthetase inhibitorsuppressed by:barbiturates, benzodiazepine, labor, hypoxia, asphyxia, prostaglandin $E_2$2.Fetal body movement:√ ≥3 discrete movements of limbs / trunk influenced by:glucose, gestational age, time of day, maternal drugs, intrinsic rhythm, labor3.Fetal toneupper + lower limbs usually fully flexed with head on chest; least sensitive test parameter √ ≥1 episode of opening + closing of hand / extension + flexion of limbB.CHRONIC FETAL CONDITION4.Amniotic fluid volume√ at least one pocket ≥2 cm in vertical diameter in two perpendicular planes◊Avoid inclusion of loops of cord! *Score (for each test):*2 points if normal;0 points if abnormal Results (including NST for a maximum of 10 points):
*Score**Interpretation*Perinatal*mortality*
10asphyxia rare0.0%8+ normal fluidasphyxia rare<0.1%8+ abnormal fluidchronic compromise8.9%6+ normal fluidequivocalvariable6+ abnormal fluidasphyxia probable8.9%4asphyxia highly probable9.1%2asphyxia almost certain12.5%0asphyxia certain60.0%False-negative rate:0.7 per 1,000◊The probability of fetal death within a week of a BPP score of 8/8 is 1 per 1,000!

Notes:

Stress Tests ***Nonstress Test*** (NST) ◊Test needed in less than 5% of cases!√ reactive fetal heart rate tracing (normal) = at least 4 fetal heart accelerations (>15 bpm over baseline lasting >15 seconds) in a 20-minute period subsequent to fetal movement >34 weeks GA√ nonreactive (abnormal) fetal heart rate tracing= absence of acceleration in a continuous 40-minute observation period N.B.:no heart accelerations in immaturity, during sleep cycle, with maternal sedative use*Accuracy*:false-negative rate of 3.2/1000 (if done weekly) or 1.6/1000 (if done biweekly); 50% false-positive rate for neonatal morbidity + 80% for neonatal mortality

Contraction Stress Test (CST) =external monitoring after injection of oxytocin / maternal breast stimulation√ >3 uterine contractions in 10-minute period*Accuracy*:false-negative rate of 0.4/1000; 50% false-positive rate

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Notes:

Amniocentesis *Indication:* (1)Inadequate sonographic fetal anatomic survey due to fetal position / maternal body habitus(2)Equivocal sonographic findings (eg, abnormal posterior fossa but spinal defect not seen)(3)Experienced sonographer not available(4)Nonlethal anomaly detected on Level I sonogram for which karyotype testing is appropriate A.DIAGNOSTIC AMNIOCENTESIS1.Genetic studies: karyotype, DNA analysis, biochemical assay*Timing:*early (11-15 weeks), late (15-18 weeks)2.Neural tube defect: a-fetoprotein, acetylcholinesterase3.Isoimmunization: →-OD 4504.Fetal lung maturity5.Intraamniotic infection6.Confirmation of ruptured membranes B.THERAPEUTIC AMNIOCENTESIS1.Polyhydramnios2.Twin-twin transfusion syndrome*Technique:* √ avoid fetus, placenta, umbilical cord, uterine contraction, fibroid, large uterine vessel√ use continuous ultrasound guidance√ inject 2-5 mL of indigo carmine dye in first sac of twin (colorless fluid assures that second sac has been entered)*Risk:* A.FETAL RISK1.Spontaneous abortion (<1%)2.Amniotic fluid leak3.Chorioamnionitis4.Fetal injury: skin dimple, limb gangrene, porencephalic cyst, hemothorax, spleen laceration, orthopedic abnormality, amniotic band syndromeB.MATERNAL RISK (rare)1.Bowel perforation2.Hemorrhage3.Isoimmunization*Advantage over CVS:* 1.Error rate (<1% versus 2%)2.Culture failure rate (0.6% versus 2.2%)3.Fetal loss rate (0.6-0.8% less)

Notes:

Chorionic villus sampling (CVS)
=aspiration of cells from chorion frondosum for genetic studies (karyotype, DNA analysis, biochemical assay)Transabdominal CVS for rapid karyotyping in 2nd + 3rd trimester = **placental biopsy**
Advantage:>2 weeks earlier results compared with amniocentesis*Timing:*9-11 weeks*Approach:* (a)transcervical route = catheter introduced through cervix into chorion frondosum, easier for posterior placenta; contamination by cervical flora possible;CONTRAINDICATED in cervical infections!(b)transabdominal route = 20- to 22-gauge needle inserted from anterior abdominal wall; easier for anterior / fundal placenta; sterile technique*Chromosome analysis:* (a)direct preparation = analysis of cytotrophoblasts (may have different karyotype than fetus)→analysis can be performed immediately(b)villus culture = cells from central mesenchymal core (same karyotype as fetus)→cultured for several days before analysis*Errors (2%):* 1.Mosaicism = cell line forming cytotrophoblast may develop abnormal karyotype while fetal cell line is normal2.Maternal contamination = cells from maternal decidua may overgrow mesenchymal core cells*Risks:* 1.Spontaneous abortion (1%)2.Perforation of amniotic sac3.Infection4.Teratogenesis: limb reduction defect

Notes:

Cordocentesis =PERCUTANEOUS UMBILICAL BLOOD SAMPLING (PUBS)A.DIAGNOSTIC CORDOCENTESIS1.Hematocrit2.Karyotype3.Immunodeficiency: chronic granulomatous disease, severe combined immunodeficiency4.Coagulopathy: von Willebrand syndrome, factor deficiency5.Platelet disorder: alloimmune / idiopathic thrombocytopenic purpura6.Hemoglobinopathy: sickle cell anemia, thalassemia7.Infection: toxoplasmosis, rubella, varicella, cytomegalovirus, parvovirus8.Hypoxia / acidosis B.THERAPEUTIC CORDOCENTESIS1.Intravascular fetal transfusion (fresh rh-negative CMV-negative leukodepleted irradiated packed cells compatible with mother infused at 10-15 mL/min)2.Direct delivery of medication to fetus *Cx:*1.Chorioamnionitis2.Rupture of membranes3.Umbilical cord hematoma4.Umbilical cord thrombosis5.Bleeding from insertion site6.Fetal bradycardia

Notes:

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MULTIPLE GESTATIONS

*Incidence:*1.2% of all births; in 5-50% clinically undiagnosed at term*Occurrence:* twinsin1:85pregnancies (= 85^{1})tripletsin1:7,600pregnancies (~ 85^{2})quadrupletsin1:729,000pregnancies (~ 85^{3})quintupletsin1:65,610,000pregnancies (~ 85^{4}) ● uterus large for dates ● may have elevated HCG, HPL (human placental lactogen), AFP levels Perinatal morbidity & mortality compared to singletons: twins:up to 5-fold increasetriplets:up to 18-fold increase

Twin pregnancy Amnionicity & Chorionicity

Notes:

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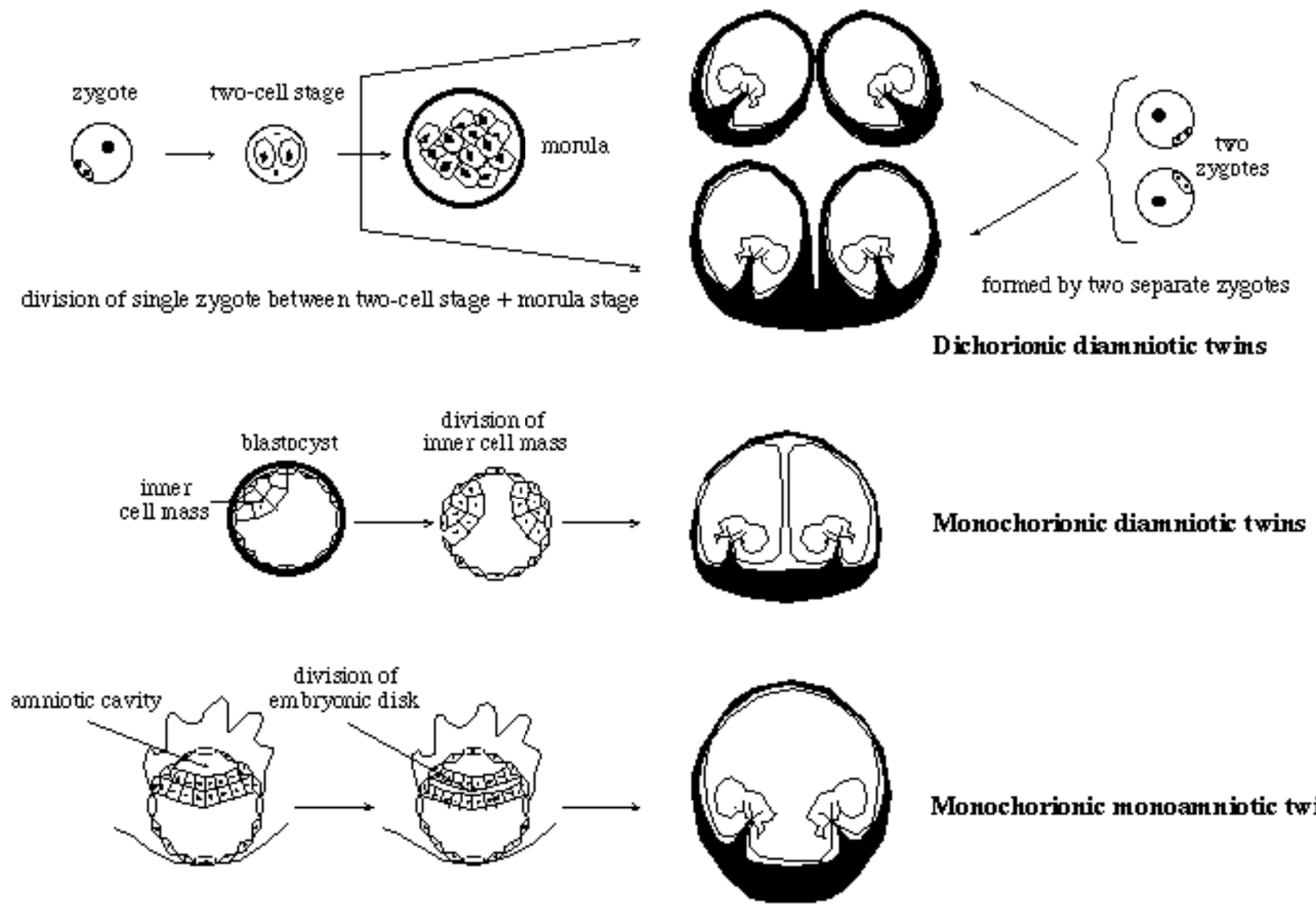
Twin pregnancy



Zygote = fertilized egg 1.MONOZYGOTIC TWINS (1/3)="identical twins"=division of a single fertilized ovum during earliest stages of embryogenesis (chorion differentiates 4 days and amnion 8 days after fertilization)*Incidence:*1:250 birth (constant around the world)*Predisposing factors:* (1)Advanced maternal age(2)in vitro fertilization√ same sex + identical genotype (a)**Dichorionic diamniotic twins** (30%)=separation at two-cell stage (= blastomere) approximately 60 hours / <4 days after fertilization√ 2 separate fused / unfused placentas√ membrane >2 mm due to 2 separate chorionic sacs + 2 separate amniotic sacs (92% accurate for dichorionic diamniotic twins)√ "twin peak" sign = triangular projection of placental tissue insinuated between layers of intertwin membrane (b)**Monochorionic diamniotic twins** (69-80%) (most common)=separation in blastocyst stage between 4th and 7th day after fertilization (chorion already developed and separated from embryo)√ 2 separate amniotic sacs within single chorionic sac◊Common monochorionic placenta has vascular communications in 100%!*Cx:*(1)Twin-twin transfusion syndrome(2)Twin embolization syndrome = DIC in surviving twin from transfer of thromboplastin; 17% morbidity / mortality of survivor after fetal death of twin(3)Acardiac parabiotic twin (c)**Monochorionic monoamniotic twins** (1%)=division of embryonic disk between 8th and 12th day after fertilization (amniotic cavity already developed)√ common amniotic + chorionic sac, no separating membrane√ entanglement of cords (the only definitive positive sonographic sign of monoamnionicity)*Cx:*double perinatal mortality up to 45%(1)Entangled umbilical cord (70%)(2)True knot of cord(3)Conjoined twins (umbilical cord with >3 vessels, shared fetal organs, continuous fetal skin contour)*Prognosis:*40% survival rate (d)**Conjoined twins**
=division more than 13 days after fertilization is usually incomplete; M:F = 3:7*Incidence:*1:50,000 births√ no separating membrane demonstrable (monochorionic, monoamniotic)√ fetuses commonly face each other; most common are thoracopagus + omphalopagus *Cx:*(1)perinatal mortality 2.5 times greater than for dizygotic twins (2)Fetal anomalies 3-7 times higher than in dizygotic twins / singletons (often only affecting one twin): anencephaly, hydrocephalus, holoprosencephaly, cloacal exstrophy, VATER syndrome, sirenomelia, sacrococcygeal teratoma 2.DIZYGOTIC TWINS (2/3)="fraternal twins"(a)fertilization of two ova by two separate spermatozoa during two simultaneous ovulations (occurring either in both ovaries or in one ovary)(b)superfetation = fertilization of two ova by two separate spermatozoa during two subsequent ovulations (frequency unknown)(c)superfecundation = two ova fertilized by two different fathers (very rare)*Incidence:*1:80 to 1:90 births*Predisposing factors:* (1)Advanced maternal age (increased up to age 35): reduced gonadal-hypothalamic feedback with increase of FSH levels(2)Ovulation-inducing agents (multiple pregnancies in 6-17% with clomiphene, in 18- 53% with Pergonal)(3)Maternal history of twinning (3 times as frequent compared with normal population)(4)Increased parity(5)Maternal obesity(6)Race with inherited predisposition for multiple ovulations (Blacks > Whites > Asians)√ different phenotypes; same / opposite sex√ always dichorionic diamniotic

Notes:

Amnionicity & Chorionicity *Embryologic events in monozygotic twins:*
*days after*cleavage results in*fertilizationembryologic eventchorionamnion*1-2cell divisions ⟶ moruladi~di~3-4chorionic differentiation6blastocyst implants inmono~di~endometrium8amnionic differentiationmono~mono~>13division of embryonic diskmono~mono~but conjoined
Rules: ◊Only monozygotic twins can give rise to monochorionic + monoamniotic pregnancies!◊All monoamniotic twins must also be monochorionic!◊All dizygotic twins must be dichorionic + diamniotic!◊77% of all twin pregnancies are dichorionic (ie, all dizygotics [2/3 of all twins] which equals 67% + 30% of all monozygotics [1/3 of all twins] which equals 10%) 1.GESTATIONAL SACS (<10 weeks MA)*Accuracy:*100% in 1st trimester, 80-90% in 2nd trimester√ 2 gestational sacs, each with a live fetus, indicates dichorionic twinning√ single gestational sac with 2 live fetuses indicates monochorionic twins√ single extraembryonic coelom indicates monochorionic twins2.YOLK SAC√ number of yolk sacs = number of amnions3.FETAL GENDER√ different genders (in 25% of twin pregnancies) must be dizygotic twins and thus dichorionic![DDx:testicular feminization demonstrates female external genitalia with a 46,XY karyotype]4.PLACENTAL SITES√ 2 placentas (in 45% of twin pregnancies) indicate dichorionic diamniotic pregnancy√ 1 placenta indicates(a)monochorionic pregnancy(b)dichorionic pregnancy with fused placenta (occurs in 50% of dichorionic twin pregnancies)5.CHORIONIC PEAK√ "twin peak" sign (= triangular projection of placental tissue extending beyond chorionic surface of the placenta + insinuated between layers of intertwin membrane + wider at chorionic surface and tapering to a point some distance inward from surface) indicates dichorionic pregnancy 6.MEMBRANE√ separating membrane confirms diamniotic pregnancy, but does not distinguish between mono- or dichorionic pregnancy√ dichorionic membrane (two layers of chorion + two layers of amnion) is thicker (>2 mm) than monochorionic membrane (two layers of amnion <1 mm): 88-92% accuracy in 1st trimester, 39-83% accuracy in 2nd + 3rd trimester◊All membranes appear to be thin in 3rd trimester!√ absence of membrane suggests a monoamniotic monochorionic twin pregnancy◊Nonvisualization of membrane is not sufficient evidence of monoamnionicity due to technical factors! 7.CORD√ entanglement of cords is the only definitive positive sonographic sign of monoamnionicity√ simultaneous recording of fetal arterial signals at nonsynchronous rates within wide Doppler gate 8.AMNIOGRAPHY√ detection of imbibed intestinal contrast in both twins by CT following single sac contrast injection proves monoamniotic monochorionic twin pregnancy ***Growth Rates Of Twins*** Twins should be scanned every 3-4 weeks >26-28 weeks GA A.Below 30-32 weeks GA√ normal individual twins grow at same rate as singletons√ BPD growth rates similar to singleton fetusesB.Beyond 30-32 weeks GA√ combined weight gain of both twins equals that of a singleton pregnancy (AC of twins < AC of singleton)◊Weight of twin fetus falls below that of singleton when combined weight of twins >4000 g!√ BPD + HC growth may / may not be affected (controversial)√ FL not affected Discordant Growth =weight difference at birth >25%*Cause:*(1)Twin-twin transfusion syndrome(2)IUGR of one fetus√ BPD difference >5 mm (discordant growth in 20-30%)√ discordant HC increases probability of IUGR√ AC is single most sensitive parameter for IUGR√ EFW is most sensitive set of combined parameters for IUGR√ >15% S/D ratio difference of umbilical artery Doppler waveforms between twins
Risks In Multiple Gestations 1.Placental abruption3-fold2.Anemia2.5-fold3.Hypertension2.5-fold4.Congenital anomaly2-3-fold5.Preterm delivery12-fold6.Perinatal mortality4-6-fold◊Risk increases with number of fetuses, monozygosity, monochorionicity *Risk For IUGR* monochorionic-monoamniotic > monochorionic-diamniotic > dichorionic-diamniotic *Risk For Perinatal Mortality:* 1% for singletons, 9% for diamniotic dichorionic twins, 26% for diamniotic monochorionic twins, 50% for monoamniotic monochorionic twins *Prognosis:* (1)Perinatal mortality 5-10 times that of singleton pregnancy (91-124:1,000 births)- 9% for dichorionic diamniotic twins-26% for monochorionic diamniotic twins-50% for monochorionic monoamniotic twins(a)preterm delivery with birth weight <2500 g(b)IUGR (25-32%; 2nd most common cause of perinatal mortality + morbidity)(c)amniotic fluid infection (60%)(d)premature rupture of membranes (11%)(e)twin-twin transfusion syndrome (8%)(f)large placental infarct (8%)(g)placenta previa(h)abruptio placentae(i)preeclampsia(j)cord accidents(k)malpresentations(l)velamentous cord insertion (7-fold increase compared with singleton pregnancy)(2)Fetal death in utero (0.5-6.8%; 3 times as often in monochorionic than in dichorionic gestations)◊50% of twin gestations seen at 10 weeks GA will be singletons at birth!(3)Increased risk of congenital anomalies (23:1,000 births = twice as frequent as in singletons; 3-7 times more frequent in monozygotic twins than in dizygotic twins)

Notes:

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Uterine Size A.NEONATAL UTERUS tubular structure Length of 2.3-4.6 cm (mean 3.4 cm), fundal width of 0.8-2.1 cm (mean 1.2 cm), cervical width of 0.8-2.2 cm (mean 1.4 cm) √ echogenic endometrium + endometrial fluid (in 25%) secondary to maternal hormonal stimulationB.INFANTILE UTERUS*Age:*infancy to 7 years of ageLength of 2.5-3.3 cm, fundal width of 0.4-1.0 cm, cervical width of 0.6-1.0 cm √ cervix occupies 2/3 of uterine lengthC.POSTPUBERTAL UTERUS-nulliparous: 5-8 cm (L); 1.6-3.0 cm (W); 3 cm (D)-multiparous: add 2 cm for multiparous dimensions√ cervix occupies 1/3 of uterine length√ mean uterine volume of 90 cm^3D.POSTMENOPAUSAL UTERUScervix occupies 1/3 of uterine length; 3.5-6.5 cm (L); 1.2-1.8 cm (W); 2 cm (D)

Notes:

Uterine Zonal Anatomy (on T2WI)

Thickness of zones depends on menstrual cycle + hormonal medication A.ENDOMETRIUM√ high signal intensity similar to fatB.JUNCTIONAL ZONE= innermost layer of myometrium*Histo:*compact smooth muscle fibers with 3-fold increase in number + size of nuclei compared with outer myometrium√ low signal intensity (lower water content); seen in 40 -60%, may not be visible in premenarchal + postmenopausal womenC.MYOMETRIUM√ intermediate signal intensity, increases during secretory phase ***Cervical Zones*** (a)Central stripe of high signal intensity on T2WI*Histo:*secretions in endocervical canal + cervical mucosa + plicae palmatae√ arbor vitae / plicae palmatae = irregular branched mucosal pattern of cervical canal(b)Middle layer of low signal intensity continuous with junctional zone of corpus uteri*Histo:*inner zone of fibromuscular stroma with percentage of nuclear area 2.5 times greater than in outer zone(c)Outer layer of intermediate signal intensity*Histo:*outer zone of fibromuscular stroma

Notes:

Endometrium ◊Measurements refer to AP diameter of both apposed endometrial layers (= double thickness) excluding intrauterine fluid1.MENSTRUAL PHASE (usually days 1-5)*Thickness:*1-3 mm√ interrupted thin echogenic line of central interface2.PROLIFERATIVE PHASE (days 6-14)*Thickness:*4-6 mm√ bright echogenic central line (= apposed borders of endometrial canal)√ hypoechoic band (= thickened endometrium)√ surrounded by slightly more echogenic myometrium3.SECRETORY PHASE (days 15-28)*Thickness:*7-14 mm√ bright central line√ markedly echogenic thick endometrium√ thin hypoechoic halo of inner myometrial zone4.POSTMENOPAUSAL*Thickness:*<8 mm thick in 81%; may increase to 15 mm with hormonal replacement (unopposed estrogen, continuous estrogen + progestogen)√ endometrium <5 mm is consistently associated with atrophic inactive endometrium by histology√ Doppler waveforms with resistive index <0.7 suggest malignancy*Rx:*biopsy / DC if endometrial thickness >8 mm

Notes:

Pelvic Spaces 1.Rectouterine pouch = cul-de-sacAnterior boundary:broad ligaments + uterus◊Most dependent portion of pelvis in women!2.Rectovesical recess◊Most dependent portion of pelvis in men!3.Vesicouterine recess4.Inguinal fossalocated between lateral + medial umbilical folds

Notes:

Cervical Length *transabdominal*transvaginal 1st trimester(<14 wks)53 ± 1740 ± 8 mm2nd trimester(14-28 wks)44 ± 1442 ± 10 mm3rd trimester(≥ 28 wks)40 ± 1032 ± 12 mm◊Distended bladder improves visualization but increases cervical length on transabdominal US◊Difference between nulli- and multiparous women 10%! ● physical examination tends to underestimate the true length of the cervix

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Notes:

Pelvic Ligaments

1.Broad ligament*Histo:*2 layers of peritoneumOrigin:uterine peritoneumAttachment:pelvic sidewall-medial superior free edge: formed by fallopian tube -lateral superior free edge: suspensory ligament of ovary-lower margin: cardinal ligamentContents (= parametrium): extraperitoneal connective tissue, smooth muscle, fat, fallopian tube, round ligament, ovarian ligament, uterine + ovarian blood vessels, nerves, lymphatics, mesonephric remnants 2.Round ligament=anterior suspensory ligament of uterus*Histo:*band of fibromuscular tissue + lymphatic channelsOrigin:anterolateral uterine fundus, just below + anterior to ovarian ligamentAttachment:through internal inguinal canal (lateral to deep inferior epigastric vessels) to labia majora 3.Cardinal ligament = transverse cervical ligament = Mackenrodt ligamentOrigin:cervix + upper vaginaAttachment:fascia of obturator internus muscleRelationship: -uterine artery runs along its superior aspect-forms the base of the broad ligament 4.Uterosacral ligamentOrigin:posterolateral cervix + vaginaAttachment:anterior body of sacrum at S2 or S3 5.Ovarian ligament = round ligament of the ovaryOrigin:medial aspect of ovaryAttachment:uterus, just inferior + posterior to fallopian tube + round ligament 6.Suspensory ligament of ovary = infundibulopelvic lig.Origin:anterolateral aspect of ovaryAttachment:connective tissue over psoas muscleContents:ovarian artery + vein 7.Lateral umbilical fold / ligament=reflection of peritoneum over deep inferior epigastric vessels 8.Medial umbilical fold / ligament=reflection of peritoneum over obliterated umbilical arteries 9.Median umbilical ligament=reflection of peritoneum over obliterated urachusOrigin:dome of urinary bladderAttachment:umbilicus

Notes:

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OVARIES
Fixation: fairly mobile with attachments to anterior pelvic wallbybroad ligamentuterine bodybyutero-ovarian ligamentfallopian tubebytubo-ovarian ligamentlateral pelvic wallbyinfundibulopelvic ligament *Embryology:* coelomic (surface) epithelium invaginates into mesenchymal substance (= primary sex cords) and incorporates primordial germ cells, which develop into primordial follicles

Ovarian Size Ovarian Morphology Visualization Of Ovaries Ovarian Cycle Graafian Follicle Ovarian Doppler Signals

Notes:

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Ovarian Size Ovarian volume = length x height x width x 0.523 at birth:1.5 cm (L), 0.25 cm (H), 0.3 cm (W)<2 years:<0.7 cm^3childhood:0.75-0.86 $cm^3$6-11 years:1.19-2.52 cm^3after puberty:2.5-5 cm (L), 0.6-1.5 cm (H),1.5-3 cm (W); 1.8-5.7 cm^3

Notes:

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Ovarian Morphology neonate:√ follicles occasionally fail to involute + undergo growth<8 years:√ solid ovoid structures with homogeneous / finely heterogeneous texture√ up to 70% of ovaries contain cystic follicles (in 95% <9 mm, in 5% >9 mm)

Notes:

Visualization Of Ovaries
after menopause (average onset at age 50): <5 years after menopause:in 78%>10 years after menopause:in 64%-both ovaries:in 85%-one ovary:in 60%following hysterectomy:in 43%

Notes:

Ovarian Cycle 1.FOLLICULAR PHASE = days 1-14 ● a number of immature primordial follicles begin to mature in response to FSH√ multiple small cysts (= stimulated / unstimulated follicles)√ 2-3 follicles in each ovary by day 4, subsequently enlarging to approximately 10 mm√ single "ascendant" / "dominant" follicle (= graafian follicle) appears by day 10, subsequently enlarging to 20-25 mm by day 14√ progressively increasing diastolic flow on the side of maturing follicle2.OVULATORY PHASE = day 14 ● "mittelschmerz" = pain just prior to ovulation (pressure of graafian follicle distending ovarian capsule)√ sudden decrease in follicular size over minutes / hours (= rupture of mature graafian follicle with extrusion of ovum)3.LUTEAL PHASE = days 15-28√ 16-24 mm almost isoechoic cyst with blurred margin + scattered internal echoes (follicular fluid + blood) = corpus luteum of menstruation√ 30- to 40-mm cyst = corpus luteum cyst (fluid collecting in corpus luteum / additional hemorrhage)√ involution + atrophy of corpus luteum on about 24th day of cycle = corpus luteum atreticum

Notes:

Graafian Follicle Size of mature graafian follicle: 17-29 mm√ growth rate 3 mm/day until the last preovulatory 24 hours followed by a sudden increase in diameter√ cumulus = 1-mm mural echogenic focus projecting into antrum of follicle + containing oocyte, followed by ovulation within next 36 hours ***Signs Of Ovulation*** √ development of solid echoes within graafian follicle√ decrease in diameter / sudden collapse of dominant follicle 28-35 hours after LH peak√ "ring" structure within uterine fundus√ free fluid appearing in pouch of Douglas
Signs Of Ovulatory Failure √ development of internal echoes prior to 18 mm size√ continuous cystic enlargement up to 30-40 mm

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Notes:

Ovarian Doppler Signals A.NONFUNCTIONING OVARY√ high-impedance waveform B.FUNCTIONING OVARY-days 1-6:√ high-impedance waveform with RI close to 1.0-days 7-22 = midfollicular to midluteal phase= developing dominant follicle + ovulation + corpus luteal phase: √ continuous diastolic flow with RI close to 0.5-days 23-28 = late luteal phase:√ high-impedance waveform with RI close to 1.0

Notes:

ABORTION
Rate of spontaneous abortions (= miscarriage) ->50% of all fertilized ova (estimate)-31-43% of all implantations (estimate)-10-25% of clinically diagnosed pregnancies-2-4% with normal cardiac activity-decreases with increasing gestational age Majority of pregnancies lost before 7th week MA!*Etiology:*usually due to abnormal karyotype: autosomal trisomy (52%), triploidy (20%), monosomy (15%)

Complete Abortion Incomplete Spontaneous Abortion Inevitable Abortion Missed Abortion Threatened Abortion

Notes:

Complete Abortion ● cervix closed √ thin regular endometrium

Notes:

Incomplete Spontaneous Abortion =RETAINED PRODUCTS OF CONCEPTION=portion of chorionic villi (placental tissue) / trophoblastic tissue (fetal tissue) remaining within uterus ● continued bleeding ● patulous cervixUS (overall accuracy 96%): FindingRetained Products√ gestational sac / collection100%√ sac with dead fetus100%√ endometrium>5 mm thick100%√ endometrium2-5 mm thick 43%√ endometrium<2 mm thick14%*Cx:*endometritis, myometritis, peritonitis, septic shock, diffuse intravascular coagulation (with retention >1 month)*Rx:*suction DC after IV oxytocin

Notes:

Inevitable Abortion =gestational sac with fetus having become detached from implantation site; leading to spontaneous abortion within next few hoursClinical triad: ● bleeding >7 days ● persistent painful uterine contractions ● moderate effacement of cervix ● dilated cervix >3 cm ● rupture of membranes√ sac located low within uterus√ sac surrounded by anechoic zone of blood√ dilated cervix

Notes:

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Missed Abortion =dead conceptus within uterine cavity*Time:*between 8-14 weeks MA ● brownish vaginal discharge ● closed firm cervix√ no cardiac activity in a well-defined embryo with CRL >9 mm (on abdominal scans) / CRL >5 mm (on transvaginal scans)√ gestation not in correspondence with menstrual age√ sac >25 mm in diameter without an embryo (DDx: anembryonic pregnancy)√ sac >20 mm without yolk sac√ crenated irregular / distorted angular sac configuration√ stringlike debris within gestational sac (in 25%)√ discontinuous / irregular / thin (2 mm) choriodecidual reaction√ no double decidual sac√ low sac position√ subchorionic collection*Cx:*coagulopathy secondary to low plasma fibrinogen (after 4 weeks in 2nd trimester pregnancy)*Rx:*suction DC (in 1st trimester);prostaglandin E suppositories (in 2nd trimester)

Notes:

Threatened Abortion =1st trimester bleeding with a live fetus*Incidence:*20-25% of all pregnanciesClinical triad: ● mild bleeding ● cramping ● closed cervix*Prognosis:*50% develop normally; 50% miscarryFactors with a poor prognosis: √ early bradycardia√ large subchorionic hematoma (DDx: implantation bleed)√ relative fetal inactivity

Notes:

ACARDIA
=ACARDIAC MONSTER = TWIN REVERSED ARTERIAL PERFUSION SEQUENCE (TRAP)=rare developmental anomaly of monochorionic twinning in which one twin develops without a functioning heart*Incidence:*1:30,000-35,000 births; in 1% of monozygotic twins*Pathophysiology:* normal twin perfuses acardiac twin through *artery-to-artery* + *vein-to-vein anastomoses* in shared placenta; reversed circulation alters hemodynamic forces which result in abnormal cardiac morphogenesis Spectrum: (1)Holoacardia = no heart at all(2)Pseudoacardia = rudimentary cardiac tissue√ proximity of the two cord insertions on placental surface linked by an arterioarterial anastomosis√ reversed arterial flow in cord toward acardiac twin√ fused placentas√ polyhydramnios A.PUMP TWINat increased risk for fetal demise + preterm labor √ morphologically normal√ cardiac overload signs: hydrops, IUGR, hypertrophy of right ventricle, increased cardiothoracic ratio, hepatosplenomegaly, ascitesB.PERFUSED TWIN = ACARDIAC TWINmonochorial placenta (same gender) with vascular anastomosis sustains life of acardiac monster; wide range of associated abnormalities √ absent / rudimentary heart ("acardius")√ tiny / absent cranium (acephalus)√ small upper torso ± absent / deformed upper extremities√ marked integumentary edema + cystic hygroma*Prognosis:*mortality of 100% for perfused twin, 50% for pump twin (increased with increased size of acardiac twin)*Rx:*laser ablation of umbilical cord to acardiac twin (up to 20-22 weeks)

Notes:

ADENOMYOSIS
=ENDOMETRIOSIS INTERNA=focal / diffuse benign invasion of myometrium by endometrium (heterotopic "endometrial islands") which incite myometrial hyperplasia*Cause:*? uterine trauma (parturition, myomectomy, curettage)*Incidence:*9-31% in hysterectomy specimens*Histo:*endometrial glands (nonfunctioning due to resistance to hormonal stimulation unlike endometriosis) + stroma within myometrium surrounded by hypertrophic smooth muscle*Age:*multiparous women >30 years during menstrual life (later reproductive years)*Associated with:*endometriosis (in 36-40%) ● asymptomatic in 5-70% ● pelvic pain, menorrhagia, dysmenorrhea (abates after menopause) A.FOCAL ADENOMYOSIS = "adenomyoma"√ oval / elongated shape (DDx: leiomyoma is round)√ ill-defined margins (DDx: sharp margin in leiomyoma)√ contiguity with junctional zone (DDx: leiomyomas may occur anywhere in myometrium)B.DIFFUSE ADENOMYOSIS√ smooth uterine enlargement (DDx: diffuse leiomyomatosis) MR (86% sensitive, 86% specific): √ myometrial mass with indistinct margins of primarily low signal intensity on all sequences√ diffuse / focal widening of junctional zone ≥12 mm on T2WI, T2-weighted SE images, contrast-enhanced T1WI images√ central high-intensity spots on T1WI + T2WI (ectopic endometrial tissue / endometrial cyst / hemorrhagic foci) in 50%√ enhancement always less than adjacent myometriumUS (80-86% sensitive, 74-89% specific): √ poorly defined hypoechoic heterogeneous areas within myometrium√ 1-3 mm small myometrial cysts (50%), occasionally with "Swiss cheese" appearance of myometrium√ thickening + asymmetry of anterior and posterior myometrial walls*Cx:*infertility*DDx:*(1)leiomyomas (clinically + sonographically difficult to distinguish)(2)uterine contraction*Rx:*hysterectomy (the only definitive cure)

Notes:

AMNIOTIC BAND SYNDROME
=EARLY AMNION RUPTURE SYNDROME=rupture of the amnion exposing the fetus to the injurious environment of fibrous mesodermic bands that emanate from the chorionic side of the amnion*Prevalence:*1:1,200 - 1:2,000 - 1:15,000 livebirths√ very thin membrane that flaps with fetal movement or attaches to fetus√ abnormal sheet / bands of tissue that attach to the fetus(DDx: uterine synechiae, incomplete amniochorionic fusion, amniochorionic separation due to subchorionic hemorrhage, fibrin deposits, venous lakes, residual sac of blighted twin pregnancy, wisps of umbilical cord)√ restriction of fetal motion secondary to entrapment of fetal parts by bands*Associated with fetal deformities in 77%:* 1.Limb defects (multiple + asymmetric)√ amputation / constriction rings of limbs / digits√ distal syndactyly√ clubbed feet (30%)2.Craniofacial defects=asymmetric nonanatomic defects of skull + brain√ anencephaly√ asymmetric lateral encephalocele√ facial clefting of lip / palate√ asymmetric microphthalmia√ incomplete / absent cranial calcification√ ± attachment of head to uterine wall3.Visceral defects√ gastroschisis ± exteriorization of liver√ omphalocele√ gibbus deformity of spine*DDx:*(1) Chorioamnionic separation(2) Intrauterine synechiae

Notes:

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ANEMBRYONIC PREGNANCY

=BLIGHTED OVUM; may occur as a blighted twin=gestational sac of >2.5 mL with no identifiable embryo √ yolk sac identified without embryo√ empty gestational sac (>6-8 weeks MA)√ gestational sac small / appropriate / large for dates√ lack of growth / decrease in size on serial scans(a)by transabdominal scan:GS usually not visualized before 5-5.5 weeks MA; yolk sac forms at 4 weeks MA when GS is 3 mm; embryo usually visualized by 6 weeks MA √ GS size >20 mm of mean diameter without yolk sac√ GS size >25 mm of mean diameter without embryo√ absence of GS growth documented on repeat scan 7-14 days later(b)by transvaginal scan√ GS size >8 mm of mean diameter without yolk sac√ GS size >16 mm of mean diameter without embryo / cardiac activity*Cx:*first trimester bleeding

Notes:

ARRHENOBLASTOMA

*Age peak:*25-45 years (range 15-66 years)√ solid mass with cystic components (hemorrhage± necrosis)√ unilateral (95%), up to 27 cm in diameter*Cx:*malignant transformation in 22%

Notes:

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ASHERMAN SYNDROME
=association of intrauterine synechiae (= adhesions consisting of fibrous tissue or smooth muscle) with menstrual dysfunction + infertility*Cause:*sequela of endometrial trauma (vigorous instrumentation during dilatation & curettage) usually during postpartum or postabortion period / severe endometritis ● hypomenorrhea / amenorrhea ● habitual abortion / sterilityHSG: √ solitary / multiple filling defects√ bands of tissue traversing endometrial cavity√ irregularity of uterine cavity√ partial / near complete obliteration of uterine cavity (DDx: DES exposure)US: √ thickened endometrium

Notes:

BECKWITH-WIEDEMANN SYNDROME
=EMG SYNDROME (**E**xomphalos = omphalocele, **M**acroglossia, **G**igantism)=common autosomal dominant overgrowth syndrome with reduced penetrance + variable expressivity related to short arm of chromosome 11; sporadic in 85%*Incidence:*1:13,700 to 1:14,300 livebirths; M:F = 1:1 ● neonatal polycythemia√ advanced bone age*Constellation:* (1)Hemihypertrophy13-33%(2)Hyperplastic visceromegaly:57%kidney, liver, spleen, pancreas, clitoris, penis, ovaries, uterus, bladder (3)Abdominal wall defects(a)Omphalocele76%(b)Umbilical hernia49%(c)Diastasis recti33%(4)Macroglossia98%(5)Facial nevus flammeus63%(6)Ear lobe creases and pits66%(7)Prominent eyes with intraorbital creases(8)Infraorbital hypoplasia81%(9)Gastrointestinal malrotation83%(10)Pancreatic islet hyperplasia(11)Cardiac anomalies(12)Natal / postnatal gigantism77% @Adrenal gland*Histo:*adrenocortical cytomegaly, cystic adrenal cortex, hyperplastic adrenal medulla@Kidney*Histo:*disordered lobar arrangement, medullary dysplasia√ nephromegaly√ increased cortical echogenicity (due to glomeruloneogenesis)√ accentuation of corticomedullary definition√ medullary sponge kidney√ pyelocaliceal diverticulaOB-US: √ LGA fetus with growth along 95th percentile√ polyhydramnios (51%)√ thickened placenta√ long umbilical cord*Cx:*(1)Development of malignant tumors (in 10%): Wilms tumor, hepatoblastoma, adrenocortical carcinoma(2)Neonatal hypoglycemia (50-61%)

Notes:

BRENNER TUMOR
= almost always benign ovarian tumor *Incidence:*1.5-2.5%*Histo:*transitional epithelial cells within prominent fibrous connective tissue stroma*Associated with:*mucinous cystadenoma / other epithelial tumor in 20-30%*Peak age:*40-70 years ● may have estrogenic activity√ usually hypoechoic solid homogeneous tumor with well-defined back wall√ mostly 1-2 cm (up to 30 cm) in diameter√ ± extensive calcifications√ bilateral in 5-7%

Notes:

CERVICAL CANCER

6th most common cause of death from cancer in women; 3rd most common gynecologic malignancy; 15,800 new cases + 4,800 deaths in 1996 *Incidence:*12:100,000 women per year*Peak age:*45-55 years*Histo:*squamous cell carcinoma (95%), adeno-carcinoma (5%), unusual clear cell adeno-carcinoma in women exposed to DES in utero*Risk factors:*lower socioeconomic class, Black race, early marriage, increased parity, young onset of sexual relations, multiple sexual partners, positive herpes virus type II titers*FIGO stage:* 0Carcinoma in situ (before invasion)IConfined to cervixIamicroinvasion of stromaIbinvasion confined to cervixIIExtension beyond cervix but not to pelvic wall / lower third of vaginaIIavaginal invasion excluding lower 1/3IIbparametrial involvement excepting pelvic sidewallIIIExtension to pelvic wall / lower third of vaginaIIIainvasion of lower 1/3 of vaginaIIIbparametrial involvement to pelvic wallIVamucosal involvement of bladder / rectumIVbspread to distant organs (paraaortic / inguinal nodes, intraperitoneal metastasis) Significance of tumor size: >4 cm:nodal metastases (80%), local recurrence (40%), distant metastases (28%)<4 cm:nodal metastases (16%), local recurrence (5%), distant metastases (0%) *Spread:*direct extension, lymphatic, hematogenous*Incidence of nodal metastases (77% accuracy for CT, 78% for MR):* 0.3%for stage0, I a16%for stageI b33%for stageII a37%for stageII b ● leukorrhea ± vaginal bleeding (<30%) ● postcoital bleeding / metrorrhagia√ bulky enlargement of cervix (DDx: cervical fibroid)√ fluid-filled uterus (secondary to obstruction)√ signs of parametrial invasion: >4-mm soft-tissue strands extending from cervix into parametria, cardinal / sacrouterine ligaments, irregularity of cervical margins, eccentric parametrial enlargement, obliteration of fat planesMR (76-83% accuracy for staging, 82-92% accuracy for parametrial involvement): √ isointense mass on T1WI√ hyperintense focal bulge / mass on T2WI (DDx: postbiopsy changes, inflammation, nabothian cysts)√ blurring + widening of junctional zone secondary to obstruction of cervical os (retained secretions in uterine cavity)*Prognosis:*local recurrence (usually within 2 years)

Notes:

CHORIOAMNIONIC SEPARATION

(a)normally seen <16 weeks=incomplete fusion of amniotic membrane with chorionic plate(b)abnormal >17 weeks MA =secondary to hemorrhage √ membrane extends over fetal surface + stops at origin of umbilical cord√ elevated membrane thinner than chorionic membrane*Cx:*rupture of amniotic membrane may lead to amniotic band syndrome*DDx:*cystic hygroma (moves with embryo)

Notes:

CHORIOANGIOMA

=benign vascular malformation of proliferating capillaries (= hamartoma)*Incidence:*1:3,500 to 1:20,000 birthsLocation:usually near the umbilical cord insertion site√ well-circumscribed intraplacental mass with complex echo pattern protruding from the fetal surface of the placenta√ polyhydramnios (in 1/3)√ arterial signal on Doppler ultrasound in angiomatous chorioangioma*Cx:*hemorrhage, fetal hydrops, cardiomegaly, congestive heart failure, IUGR, premature labor, fetal demise (with large lesion)

Notes:

CHORIOCARCINOMA
5% of gestational trophoblastic diseases *Age:*child-bearing age*Histo:*biphasic pattern including syncytiotrophoblastic + cytotrophoblastic proliferation without villous structures; extensive necrosis + hemorrhage; early + extensive vascular invasionPreceded by:*mnemonic:*"MEAN"**M**ole (hydatidiform)in 50.0%**E**ctopic pregnancyin 2.5%**A**bortion, spontaneousin 25.0%**N**ormal pregnancyin 22.5% ● continued vaginal bleeding ● continued elevation of HCG after expulsion of molar / normal pregnancy (25%)√ mass enlarging the uterus√ mixed hyperechoic pattern (hemorrhage, necrosis)Spread: (a)hematogenous (usually)(b)lymphatic + direct extension (occasionally)Hemorrhagic + necrotic metastases to lung, vagina, kidney (10-50%), brain √ radiodense pulmonary masses with hazy borders due to hemorrhage√ hyperechoic hepatic foci*Prognosis:*85% cure rate (even with metastases); fatal with spread to kidneys + brain*Rx:*(1)Chemotherapy: methotrexate, actinomycin D ± cyclophosphamide(2)Hysterectomy (if at risk for uterine rupture)*DDx: mnemonic:*"THE CLIP"**T**rue mole **H**ydropic degeneration of placenta **E**ndometrial proliferation **C**oexistent mole and fetus **L**eiomyoma (degenerated) **I**ncomplete abortion **P**roducts of conception (retained)

Notes:

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CLEAR CELL NEOPLASM OF OVARY
=MESONEPHROID TUMOR= almost always invasive carcinoma *Incidence:*5-10% of all ovarian cancers*Histo:*clear cells (cuboidal cells with clear cytoplasm) + hobnail cells (columnar cells with large nuclei projecting into the lumina of glandular elements); similar to clear cell carcinoma of endometrium, cervix, vagina, kidney*Not associated with:*in utero DES exposure (like lesions of the vagina + cervix) ● 75% of patients present with stage I disease ● frequently unilocular cyst + mural nodule*Prognosis:*50% 5-year survival rate

Notes:

CONJOINED TWINS
=incomplete division of embryonic cell mass in monozygotic twins occurring at 13-16 days GA*Incidence:*1:52,000 livebirths; 1:600 twin births;M:F = 3:7 *Types:* A.Inferior conjunction:1.Diprosopustwo faces + one head and body2.Dicephalustwo heads + one body3.Ischiopagusjoined by inferior sacrum and coccyx4.Pygopagus (20%)joined by posterolateral sacrum and coccyx B.Superior conjunction:1.Dipygussingle head, thorax, abdomen + two pelves and four legs2.Syncephalusfacial fusion ± thoracic fusion3.Craniopagus (6%)joined between homologous portions of cranial vault C.Middle conjunction:1.Thoracopagus (18%)between thoracic walls; conjoined hearts (75%)2.Omphalopagus (10%)joined between umbilicus + xiphoid3.Xiphopagusjoined at xiphoid4.Thoracoomphalopagus (28%) D.Incomplete duplication (10%): duplication of only one part of body OB-US (diagnosed as early as 12 weeks GA): √ single placenta without amniotic membrane (monochorionic, monoamniotic = hallmark of monozygotic twinning)√ inseparable fetal bodies + skin contours√ no change in relative position of fetuses√ both fetal heads persistently at same level (fetuses commonly face each other)√ bibreech (more common) / bicephalic presentation (cephalic-breech presentation is most common presentation for omphalopagus)√ polyhydramnios (in almost 50%)√ single umbilical cord with >3 vessels√ backward flexion of cervical spine (in anterior fusion)√ single cardiac motion (shared heart)*Associated malformations:* √ omphalocele√ congenital heart disease*Prognosis:*39% stillborn; 34% die within first days of life

Notes:

CORD PROLAPSE
=prolapse of cord into endocervical canal*Incidence:*0.5% at delivery*Predisposing factors:* nonvertex fetal lie, polyhydramnios, cephalopelvic disproportion, multiple gestation, increased length of umbilical cord *Cx:*cord compression with high perinatal mortalityN.B.:MEDICAL EMERGENCY! Alert obstetrician immediately!*OB-Management::* (1)Patient immediately placed into Trendelenburg / knee-elbow position in radiology department(2)Cesarean section for term infants(3)Expectant management for preterm infants*DDx:***Cord presentation** (= umbilical cord between fetus and internal os)

Notes:

CORPUS LUTEUM CYST

Types: 1.**Corpus luteum of menstruation** formed after rupture of follicle + increasing in size until 22nd day of menstrual cycle √ usually >12-17 mm in size2.**Corpus luteum of pregnancy**

caused by HCG stimulation during pregnancy √ usual size 30-40 mm, may grow up to 15 cm in diameter√ reaches maximum size after 8-10 weeks√ usually resolves before 20 weeks GA (12-15 weeks), occasionally persists past 1st trimester√ thin-walled usually unilateral cyst√ echogenic (organized clot) / sonolucent (resorbed blood)√ low-level internal echoes frequent (= hemorrhage)*Cx:*rupture with intraperitoneal hemorrhage

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Notes:

CYSTADENOFIBROMA
= variant of serous cystadenoma, rarely malignant *Prevalence:*nearly 50% of all benign ovarian cystic serous tumors; bilateral in 6%*Age:*15-65 (mean 31) years ● may produce estrogen excess√ small multilocular cystic tumor√ clusters of short rounded papillary processes

Notes:

DERMOID

=DERMOID CYST = MATURE CYSTIC TERATOMA=congenital tumor containing mature tissues from all 3 germ cell layers with predominance of ectodermal component*Incidence:*5 -11-25% of all ovarian neoplasms; 66% of pediatric ovarian tumors; most common ovarian neoplasm*Origin:*self-fertilization of a single germ cell after the first meiotic division (= random error in meiosis)*Histo:*may contain struma ovarii, carcinoid tumor*Age:*reproductive life (80%); age peak 20-40 years ● abdominal mass (2/3) ● pelvic pressure / pain due to torsion or hemorrhageLocation:bilateral in 8-15-25%√ cystic mass with average diameter of 10 cm√ "dermoid plug" = Rokitansky nodule / protuberance= oval / round solid tissue mass (sebaceous material) of 10-65 mm projecting into cyst lumenPlain film (diagnostic in 40%): √ tooth / bone√ fat density (SPECIFIC)CT: √ round mass of fat floating in interface between two water-density components (93%)√ Rokitansky nodule = dermoid plug (81%), usually single, may be multiple√ fat-fluid level (12%)√ globular calcifications (tooth) / rim of calcification (56%)US (sensitivity 77-87%): √ complex mass containing echogenic components (66%)√ echogenic mass (due to mixture of sebum + hair) with "dirty" acoustic shadowing (= "tip of the iceberg") in a predominantly cystic mass (25-44%) (DDx: stool-filled rectosigmoid)√ predominantly solid mass (10-31%)√ purely cystic tumor (9-15%)√ echogenic focus with acoustic shadowing (due to calcification)MR: √ hyperintense fat within fluid of low signal intensity on T1WI√ hyperintense mass (fat + serous fluid both with high signal intensity) on T2WI√ ± chemical shift artifact (frequency-encoding direction)*Cx:*(1)Malignant degeneration in 1-3% (usually within dermoid plug of tumors >10 cm in diameter in postmenopausal women)(2)Torsion (4-16%)(3)Rupture with chemical peritonitis (rare)(4)Hydronephrosis*Rx:*surgery (to avoid torsion / rupture)

Notes:

DIETHYLSTILBESTROL (DES) EXPOSURE
=first reported transplacental carcinogen@Vagina:adenosis, septa, ridges,clear-cell adenocarcinoma (in 1:1,000 women exposed in utero to DES, by age 35) @Cervix:hypoplasia, stenosis, mucosal displacement, pseudopolyps, hooded / "cockscomb" appearance@Uterus:hypoplasia, bands, contour irregularity, "T- shaped" uterus@Tubes:deformity, irregularity, obstruction

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Notes:

DYSGERMINOMA

=malignant germ cell tumor of ovary homologous to testicular seminoma*Incidence:*0.5-2% of all malignant ovarian tumors*Peak age:*2nd-3rd decade ● no elevation of AFP / HCG (in 5% syncytiotrophoblastic giant cells present, which can elevate HCG levels)Location:usually unilateral; bilateral in 15-17%√ multilobulated solid mass divided by fibrovascular septa√ speckled pattern of calcifications (rare)MR: √ hypo- / isointense septa on T2WI with contrast-enhancement on T1WIUS: √ hyperechoic solid mass, may have areas of hemorrhage + necrosis√ prominent arterial color Doppler flow within septa*Rx:*highly radiosensitive

Notes:

ECLAMPSIA
=occurrence of coma ± pre-, intra-, or postpartum convulsions not related to a coincidental neurologic disorder in a preeclamptic patient*Pathophysiology:*
A.VASOSPASM THEORYoverregulation of cerebral vasoconstrictive response to acute + severe hypertension progresses to vasospasm; prolonged vasospasm causes local ischemia, increased brain capillary permeability, disruption of blood-brain barrier, arteriolar necrosis, leading to cerebral edema + hemorrhage
B.FORCED-DILATATION THEORYwith severe arterial hypertension upper limit of cerebral autoregulation is reached + cerebral vasodilatation starts disrupting the blood-brain barrier and resulting in cerebral edema *Time of onset:*2nd half of pregnancy in primigravida; <20th week GA with trophoblastic disease ● severe throbbing frontal headache ● visual disturbance: scotomata, amaurosis, blurred vision ● retinal / cortical blindness ● hyperreflexia, hemi- / quadriparesis, confusion, coma ● seizures: usually tonic-clonicCT (positive in up to 50%): √ bilateral rather symmetric white matter hypodensities without contrast enhancement√ ± cerebral edema with compression of lateral ventricles√ usually transient + completely reversible cerebral-cortical + basal ganglia hypodensities (= reversible ischemic lesions)√ cerebral infarction in prolonged ischemia√ intracerebral hemorrhage (major cause of mortality in 10-60%)MR: √ transiently increased T2-signal intensity in cerebral cortex + subcortical white matter frequently in watershed areas of posterior hemispheres

Notes:

ECTOPIA CORDIS
=fusion defect of anterior thoracic wall / sternum / septum transversum prior to 9th week of gestationA.THORACIC TYPE (60%)=heart outside thoracic cavity protruding through defect in sternumB.ABDOMIANL TYPE (30%)= heart protruding into abdomen through gap in diaphragm C.THORACOABDOMINAL TYPE (7%)= in pentalogy of Cantrell D.CERVICAL TYPE (3%)= displacement of heart into cervical region *Associated with:* (1)Facial deformities(2)Skeletal deformities(3)Ventral wall defects(4)CNS malformations: meningocele, encephalocele(5)Intracardiac anomalies: tetralogy of Fallot, TGA(6)Amniotic band syndrome*Prognosis:*stillbirth / death within first hours / death within first days of life in most case

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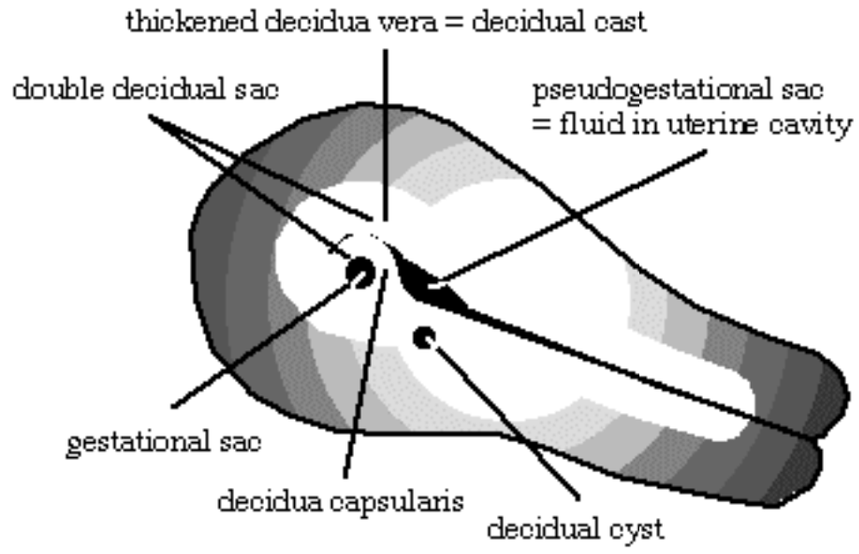
Notes:

ECTOPIC PREGNANCY
=implantation outside the endometrial cavity*Incidence:*1.6:1,000 of all pregnancies (increasing); 9.9:10,000 women annually; 73,700 cases in 1986 in United States*Risk of recurrence:*10-15%*Cause:*delayed transit of the fertilized zygote (formed on day 14 MA) secondary to(a)abnormal angulation of oviduct(b)adhesions or scarring from inflammation(c)slowed tubal transit from ciliary abnormalities*Risk factors:* (1)Previous tubal surgery (tubal ligation / tuboplasty)(2)Previous PID (30-50%): esp. Chlamydia(3)In-vitro fertilization / gamete intrafallopian tube transfer(4)Endometriosis(5)Previous ectopic pregnancy (prevalence up to 1.1%, 10-fold increase in risk, 25% chance of recurrence)(6)Current use of IUD(7)Advanced maternal age◊If the pregnancy cannot be documented as intrauterine, the patient should be considered at risk!*Time of manifestation:*usually by 7th week of MA CLASSIC CLINICAL TRIAD (<50%): ● abnormal vaginal bleeding (75-86%) ● pelvic pain (97%) ● palpable adnexal mass (23-41%) ● secondary amenorrhea (61%) ● cervical motion tenderness ● positive urinary pregnancy test (50%) ● progesterone level <25 mg/mL ● b-HCG does not rise >66% within 48 hours (lower levels + slower rise and decline compared with IUP)◊Most ectopic pregnancies do not exhibit a b-HCG of >6500 mIU/mL (1st IRP) prior to symptomatology!◊A b-HCG level above the discriminatory zone with absence of IUP suggests ectopic pregnancy!**Discriminatory zone of b-HCG** (at which a normal IUP should be visualized): (a)by transabdominal scan:≥6500 mIU/mL (IRP) with 100% sensitivity + 96% specificity(b)by endovaginal scan:≥2000 to 3000 mIU/mL (IRP) *Caveats:*technical quality of exam, multiple gestations, distortion by uterine cavity (leiomyoma), lab error, assay variationLocation: (a)tubal (95%):(1)Ampullary ectopic (75-80%)(2)Isthmic ectopic (10-15%)(3)Fimbrial ectopic (5%)(4)Interstitial ectopic (2-4%)(b)other (5%):(1)Abdominal ectopic(2)Ovarian ectopic (0.5-1%)(3)Interligamentary ectopic(4)Cervical ectopic (0.15%)*Spectrum:* Type 1:unruptured live ectopic + heartbeatType 2:early embryonic demise without rupture / embryonic structures / heartbeatType 3:ruptured ectopic with blood in pelvisType 4:no sonographic signs of ectopic*Dx:*diagnostic laparoscopy (3-4% false negative, 5% false positive) Transvaginal US (6-20% false-negative rate): ◊Detected 1 week sooner than by transvesical US!@Uterus√ absence of intrauterine pregnancy (beyond 6 weeks MA / with ß-HCG level >1,000 mIU/mL [2nd IRP])◊No IUP by transvesical US = ectopic pregnancy in 43-46%◊No IUP by endovaginal US = ectopic pregnancy in 67%√ slight thickening of endometrium√ sloughing of endometrium = decidual cast (21%)√ decidual cast = hyperechoic endometrial thickening (50%) due to hormonal stimulation from ectopic pregnancy √ decidual cyst = 1- to 5-mm cyst at junction of endometrium and myometrium (14%) √ pseudogestational sac = single parietal decidual layer surrounding an anechoic fluid collection in uterine cavity secondary to bleeding (10-20%)√ decidual



endometrium lacks low-impedance blood flow @Adnexa√ "tubal ring" = extrauterine hypoechoic saclike structure (40-68%) 1-3 cm in diameter + surrounded by a 2-4 mm concentric ring√ extrauterine mass of any type (84%)√ solid / complex adnexal mass = clotted blood free in peritoneal cavity / hematosalpinx (36%)√ extrauterine gestational sac without live embryo / yolk sac (35%)√ embryonic heartbeat (6-28%) = PATHOGNOMONIC√ echogenic "tubal mass" (89-100%)√ varying flow pattern depending on viability√ corpus luteum within ovary in >50% on side of ectopic pregnancy (DDx: ectopic pregnancy)@Cul-de-sac√ free fluid (40-83%): echogenic / particulate fluid(= hemoperitoneum) has 93% positive predictive value for ectopic pregnancy*DDx:*anechoic fluid in 10-27% of IUP Doppler-US (low diagnostic impact): √ high-velocity low-impedance flow around extrauterine gestation in 54% (up to 4 kHz shift with 3 MHz transducer, 0.38 ± 0.2 Pourcelot index, RI = 0.18-0.58)√ absence of peritrophoblastic flow after 36 days (<0.8 kHz shift with 3 MHz transducer or <1.3 kHz shift with 5 MHz transducer)*DDx of low-impedance flow:* corpus luteum cyst, tuboovarian abscess, fibroid *Probability of ectopic pregnancy in absence of IUP + clinical symptoms of an ectopic pregnancy with:* normal scan / simple cyst in adnexa5%complex adnexal mass92%tubal ring95%live embryo outside uterus100%*Prognosis:*(1)3.8:10,000 mortality rate (4% of all maternal deaths)(2)Infertility (in 40%)
Dx:(1)Laparoscopy (almost 100% accurate)(2)Culdocentesis (high probability for ectopic with aspiration of nonclotting blood with a hematocrit >15)*Cx:*maternal death in 1:1,000; tubal rupture (10-15%) *DDx:*(1)Hemorrhagic corpus luteum / hematoma(2)Adnexal mass: hydrosalpinx, endometrioma, ovarian cyst(3)Fluid-containing small bowel loop(4)Eccentrically placed GS in bicornuate / retroflexed / fibroid uterus

Abdominal Ectopic (1:6000) Heterotopic Pregnancy Interstitial (Cornual) Ectopic (2-4%)

Notes:

Abdominal Ectopic (1:6000)

◊>25% may be missed sonographically! ● bloating, abdominal pain (fetal movement / peritoneal irritation due to adhesions) ● bleeding, hypotension, shock√ extrauterine location of fetus + placenta√ uterus compressed with visible endometrial cavity line√ absence of uterine wall between gestation + bladder / abdominal wall √ anhydramnios*Cx:*bowel obstruction / perforation; erosion of pregnancy through abdominal wall ***Lithopedion*** ="stone child" = very rare obstetric complication consisting of a dehydrated + calcified demised fetus in an extrauterine pregnancy existing for >3 months without infection *Types:* (1)Lithokelyphosis = fetal membranes calcified(2)Lithokelyphopedion = fetus + membranes calcified(3)True lithopedion = only fetus calcified*Maternal age at discovery:*23-100 years of age; within 4-20 years of fetal demiseLocation:most common in adnexae√ large densely calcified mass in lower abdomen / upper pelvis√ CT scan reveals fetal skeleton*DDx:*uterine fibroid, calcified ovarian malignancy / cyst, sarcoma

Notes:

Heterotopic Pregnancy = ectopic + coexistent intrauterine pregnancy *Incidence:*1:6,800-30,000 pregnancies (higher number of coexisting ectopic with ovulation induction)◊An IUP does not preclude a complete pelvic ultrasound evaluation, although depiction of an IUP virtually excludes the diagnosis of an ectopic pregnancy!

Notes:

Interstitial (Cornual) Ectopic (2-4%)
=ectopic pregnancy with eccentric location in relation to endometrium + close to uterine serosa◊Often rupture late because of greater myometrial distensibility compared with other parts of tube!◊High likelihood of catastrophic hemorrhage + death due to abundant blood supply by both ovarian + uterine arteries!*Increased risk:*previous ipsilateral salpingectomy ● Baart de la Faille sign = broad-based palpable mass extending outward from uterine angle ● Ruge-Simon syndrome = fundus displaced to contralateral side with rotation of uterus + elevation of affected cornu√ eccentric heterogeneous mass in cornual region (66%)√ eccentrically placed gestational sac (25%)√ thinning of myometrial mantle to <5 mm (33%)√ interstitial line sign = thin echogenic line extending directly up to the center of ectopic pregnancy(= endometrial canal / interstitial portion of Fallopian tube) in 92%√ myometrium between sac and uterine cavity√ large vascular channels + peritrophoblastic blood flow√ absence of double decidual sign*Prognosis:*massive bleeding from erosion of uterine arteries + veins (pregnancy survives only 12-16 weeks GA); 2-fold mortality compared with other tubal ectopics*DDx:*pregnancy within horn of bicornuate uterus; hydatidiform mole; degenerating uterine fibroid

Notes:

Early Embryonic Demise / Failing Pregnancy

on endovaginal scan ● b-HCG level <2-3 standard deviations below the mean for given MA / GS size / CRLA.DEFINITE DEMISE√ absence of cardiac activity with CRL of ≥5 mm / ≥6.5 weeks GA (repeat scan in 3 days for confirmation)B.PROBABLY FAILING PREGNANCY√ mean sac diameter of ≥16 mm without embryo√ mean sac size of ≥8 mm without yolk sac (repeat scan in 3 days for confirmation)√ >1,000 mIU/mL(1st IRP) without gestational sac√ >7,200 mIU/mL(1st IRP) without yolk sac√ >10,800 mIU/mL(1st IRP) without embryoC.HIGH RISK OF SUBSEQUENT DEMISE√ severe bradycardia <80 bpm√ small mean gestational sac size (difference between mean sac size and CRL <5 mm is predictive of miscarriage in 94%)D.MODERATELY HIGH RISK OF DEMISE√ bradycardia of 80-90 bpm√ large subchorionic hematoma lifting much of placenta√ yolk sac >6 mm / abnormal shape √ mean gestational sac size too small for good clinical dates√ gestational sac growth ≤ 0.7 mm/day (normal growth rate of 1.13 mm/day determines appropriate time interval for follow-up scan, ie, when sac is expected to be 27 mm)√ sac position in lower uterine segment / cervix√ stringlike / granular debris / fluid-fluid level within gestational sac (= intrasac bleeding)

Notes:

Late Embryonic Demise on endovaginal scan √ wrinkled collapsing amniotic membrane√ irregular distorted shape of gestational sac(DDx: compression by bladder, myoma, contraction) √ absence of double decidual sac = thin (<2 mm) weakly hyperechoic / irregular choriodecidual reaction

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Notes:

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ENDODERMAL SINUS TUMOR OF OVARY
=YOLK SAC TUMOR=rare but highly malignant tumor*Histo:*resembles endodermal sinuses of the rat yolk sac(a)papillary pattern (most common): contains glomerular structures with central vessel + peripheral mantling of epithelial cells (= Schiller-Duval bodies)(b)others: reticular, solid, polyvesicular vitelline-periodic acid-Schiff reaction-a-fetoprotein-positive hyaline globules*Incidence:*<1% of all ovarian carcinomas*Age:*usually adolescence*May be associated with:* teratoma, dermoid cyst, choriocarcinoma ● frequently abdominal enlargement + pain ● elevated serum AFP (common)√ predominantly echogenic solid tumor√ cystic areas (epithelial-lined cysts / cysts of coexisting mature teratoma / hemorrhage / necrosis)√ bilateral in 1%*Rx:*surgery + combination chemotherapy*Prognosis:*poor

Notes:

ENDOMETRIAL CANCER

Most common invasive gynecologic malignancy; 4th most prevalent female cancer in USA women *Incidence:*34,000 new cases per year with 3,000 deaths*Histo:*adenocarcinoma (90-95%), sarcoma (1-3%)*Peak age:*55-62 years; 74% > age 50*Risk factors:*nulliparity, late menopause, unopposed estrogen therapy, polycystic ovaries, obesity, hypertension, diabetes mellitus*FIGO stage:* 0In situI aTumor limited to endometriumI binvasion to less than half of myometriumI cinvasion to more than half of myometriumII aEndocervical glandular involvement onlyII bcervical stromal invasionIII aInvasion of serosa / adnexa / peritoneal metastasesIII bvaginal metastasesIII cmetastases to pelvic / paraaortic lymph nodesIV aInvasion of bladder / bowel mucosaIV bdistant metastases (lung, brain, bone) including intraabdominal / inguinal lymph nodes◊Clinical staging with dilatation & curettage inaccurate in up to 51%!*Histo:* (a)endometrioid carcinoma (75% of all cancers)(b)serous, mucinous, clear cell carcinoma (less common): similar to ovarian counterpart(c)squamous (rare): associated with cervical stenosis, pyometra, chronic inflammation(d)mixed mesodermal tumor: contains elements of epithelial + mesenchymal differentiationLymph node metastases:3% with superficial invasion; 40% with deep invasion ● postmenopausal bleeding without hormonal therapyUS: √ normal-sized / enlarged uterus√ echogenic endometrium >5 mm AP thickness (100% negative predictive value, not very specific)√ inhomogeneous endometrial echotexture with irregular hypoechoic areas√ pulsatility index of <1.5 (DDx: endometritis, benign endometrial polyp)MR (82-92% accuracy for staging, 74-87% accuracy for depth of invasion): √ endometrial cancer has slightly lower signal intensity than endometrium but higher than myometrium on T2WI√ endometrial thickness abnormal if >3 mm (postmenopausal woman) / >10 mm (under estrogen replacement)*DDx:*blood clot, uterine secretions, adenomatous hyperplasia, submucosal leiomyoma√ disruption / absence of junctional zone (myometrial invasion)√ hyperintense areas penetrating into myometrium (deep muscle invasion; 74-87% accuracy)

Notes:

ENDOMETRIOID CARCINOMA OF OVARY

*Incidence:*15% of all ovarian cancers; 2nd most common malignant ovarian neoplasm (after serous adenocarcinoma)*Associated with:*hyperplasia / carcinoma of the uterine endometrium in 20-33%*Histo:*tubular glandular pattern with a pseudostratified epithelium resembling endometrial adenocarcinoma / metastatic colon carcinoma √ solid / complex (= cystic + solid) tumor√ bilateral in 25%*Prognosis:*better than serous / mucinous carcinomas

Notes:

ENDOMETRIOSIS
=encysted functional endometrial epithelium + stroma in an ectopic site outside the uterine cavity / myometrium*Prevalence:*8-10-18% of menstruating women*Etiology:*(1)Peritoneal implantation of endometrial cells via retrograde menstruation through fallopian tubes(2)Metaplastic transformation of peritoneal epithelium into endometrial tissue(3)Traumatic spread (uterine surgery, amniocentesis)*Age:*30-45 years; dependent on normal hormonal stimulation ● infertility◊25% of infertile women have endometriosis◊30-40% of women with endometriosis are infertile ● severe dysmenorrhea, menorrhagia ● chronic pelvic pain (peritoneal adhesions, bleeding) ● dyspareuniaLocation: (a)internal endometriosis (within uterus)=ADENOMYOSIS(b)external endometriosistypical in:ovaries > uterosacral ligaments > pouch of Douglas > uterine serosal surface > fallopian tube > rectosigmoidrare in:urinary bladder wall, umbilicus, bowel wall (20%), laparotomy scar, lungs, pleural space, limbs*Morphologic types:* 1.Discrete pelvic mass◊Multiplicity favors the diagnosis of endometrioma√ typically cystic space = **endometrioma**
="chocolate cyst" up to 20 cm in diameter (usually 2-5 cm)√ anechoic cyst / cyst with "ground-glass" homogeneous low-level echoes (= hemorrhagic debris)√ may contain echogenic material (= clot) appearing as a solid tumor√ may show layering of debris√ smooth walls + acoustic enhancement2.Diffuse form (70%)√ often no detectable abnormality (when lesions small + scattered)√ frequently multiple cysts bilaterally√ thickened wall + loss of definition of borders of pelvic organsMR (90% sensitive, 98% specific with fat suppression + contrast enhancement): √ hyperintense lesions on all pulse sequences in 47%, hypointense on all pulse sequences in 27%√ typically hyperintense on T1WI (similar to fat) + additional hyperintensity (like urine) on T2WI with multiple locules and internal "shading"@ GI tract (5-12-37%) ● change in bowel habits, rectal pain / bleeding*Path:*muscular hypertrophy + fibrosis related to endometriotic deposits in bowel wallLocation:inferior margin of sigmoid colon + anterior wall of rectosigmoid (72%); rectovaginal septum (14%); small intestine (7%); cecum (4%); appendix (3%); occasionally multiple lesions√ single extramucosal mass with crenulated / spiculated mucosal pattern√ polypoid intraluminal mass / annular constricting lesion (rare appearance)CXR: √ catamenial pneumothorax = spontaneous pneumothorax due to endometriosis of diaphragm*Cx:*infertility with involvement of tubes + ovaries (peritubal adhesions causing anatomic distortion, limitation of fimbrial motion, tubal destruction / occlusion)*Dx:*laparoscopy*Rx:*hormonal therapy, surgery*DDx:*hemorrhagic ovarian cyst, dermoid cyst, tubo-ovarian abscess

Notes:

FACIAL CLEFTING
Normal embryology: 1st branchial arch develops into maxillary + mandibular prominences; by 5th week the stomodeum is surrounded by 5 prominences: frontal-nasal, paired maxillary, paired mandibular prominences; nasal pits are formed by invagination of nasal placodes on each side of frontal-nasal prominence; the 2 maxillary prominences grow medially to fuse with the 2 medial nasal prominences forming the upper lip; the lateral nasal prominences form the nasal alae *Incidence:* 0.5:1,000 in blacks; 1:1,000 livebirths in white population; 1.5:1,000 in Asians; 3.6:1,000 in American Indians; 13% of all congenital anomalies; second most common congenital malformation; most common craniofacial malformation *Risk of recurrence:*4% with one affected sibling, 17% with one affected sibling + parent

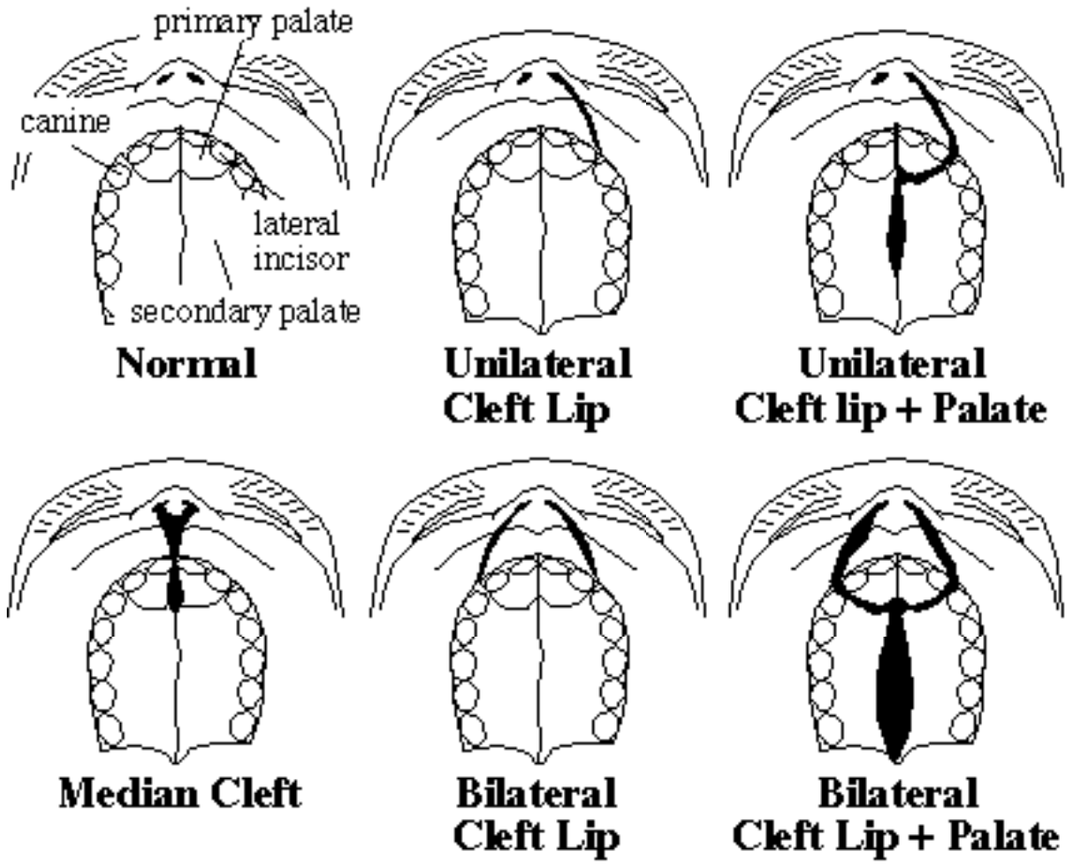
Median Facial Cleft Lateral Facial Cleft

Notes:

Median Facial Cleft =failure of fusion of the 2 medial nasal prominences*Incidence:*rare*Cause:* 1.median cleft face syndrome = frontonasal dysplasia√ brain anomalies rare2.Holoprosencephaly3.Majewski syndrome (short rib, polydactyly, median cleft)

Notes:

Lateral Facial Cleft *Cleft Lip* [25%] *Cause:*lack of fusion of maxillary prominence with medial nasal prominence (= intermaxillary segment) around 7th week MA*Associated with:*anomalies in 20% (most frequently clubfoot); NO chromosomal anomaliesSite:isolated in 8%, bilateral in 20%√ linear echopoor region extending from one side of fetal upper lip into nostril*Prognosis:*excellent
Cleft Lip & Palate [50%] *Cause:*incomplete fusion of lip + primary palate with secondary palate*Associated with:*72 abnormalities in 56-80%: most frequently polydactyly; chromosomal anomalies in 20-33%Location:L > RSite:unilateral in 23%, bilateral in 30%√ linear defect extending through alveolar ridge + hard palate reaching the floor of the nasal cavity / orbit (often deeper + longer cleft than in isolated cleft lip)√ paranasal echogenic mass inferior to nose (= premaxillary protrusion of soft tissue + alveolar process + dental structures) in bilateral cleft lip + palate
Cleft Palate [25%] =lack of fusion of mesenchymal masses of lateral palatine processes around 8th-9th weeks MA*Associated with:*anomalies in 50% (most frequently clubfoot + polydactyly)√ often missed on prenatal sonograms√ small fetal stomach + polyhydramnios (due to impaired fetal swallowing)



Notes:

Premature Atrial Contractions =PAC = most common benign rhythm abnormality√ transient tachycardia √ transient bradycardia (due to atrial bigeminy if every other beat is nonconducted)*Cx:*supraventricular tachycardia (unusual)*Rx*discontinue smoking, alcohol, caffeine*Follow-up:*biweekly auscultation until arrhythmia resolves

Notes:

Supraventricular Tachyarrhythmia *Incidence:*1:25,000; most frequent tachyarrhythmia in children*Etiology:* viral infection, hypoplasia of sinoatrial tract *Pathogenesis:* (1)Automaticity = irritable ectopic focus discharges at high frequency(2)Reentry = electric pulse reentering the atria inciting new discharges*Types:* 1.Supraventricular tachyarrhythmia (SVT)(a)paroxysmal supraventricular tachycardia(b)paroxysmal atrial tachycardia atrial rate of 180-300 bpm + ventricular response of 1:12.Atrial flutter atrial rate of 300-460 bpm + ventricular rate of 60-200 bpm3.Atrial fibrillation atrial rate of 400-700 bpm + ventricular rate of 120-200 bpm*Hemodynamics:* fast ventricular rate results in suboptimal filling of heart chambers + decreased cardiac output, overload of RA, CHF *Associated with:*cardiac anomalies (5-10%):ASD, congenital mitral valve disease, cardiac tumors, WPW syndrome, cardiomyopathy, thyrotoxicosis OB-US: M-mode echocardiography with simultaneous visualization of atrial + ventricular contractions allows inference of atrioventricular activation sequence *Cx:*congestive heart failure + nonimmune hydrops*Rx:*Intrauterine pharmacologic cardioversion (digoxin, verapamil, propranolol, procainamide, quinidine)

Notes:

Atrioventricular Block *Incidence:*1:20,000 livebirths; in 4-9% of all infants with CHD*Etiology:*(1) Immaturity of conduction system(2) Absent connection to AV node (3) Abnormal anatomic position of AV node *Associated with:* (1)Cardiac structural anomalies (45-50%): corrected transposition, univentricular heart, cardiac tumor, cardiomyopathy(2)Maternal connective tissue disease: lupus erythematosus*Types:* 1.First-degree heart block = simple conduction delay√ normal heart rate + rhythm (not reportedly diagnosed in utero)2.Second-degree heart block(a)Mobitz type I=progressive prolongation of PR interval finally leading to the block of one atrial impulse (Luciani-Wenckebach phenomenon)√ a few atrial contractions are not followed by a ventricular contraction(b)Mobitz type II=intermittent conduction with a ventricular rate as a submultiple of the atrial rate (eg, 2:1 / 3:1 block)√ atrial contraction not followed by ventricular contraction in a constant relationship3.Third-degree heart block = complete heart block=complete dissociation of atria + ventricles√ slow atrial + ventricular contractions independent from each other*Cx:*decreased cardiac output + CHF

Notes:

FETAL DEATH IN UTERO

=INTRAUTERINE DEMISE=fetal death during 2nd + 3rd trimestersSpecific signs: √ absent cardiac / somatic motionNonspecific signs seen not before 48 hours after death: √ same / decreased BPD measurement compared with prior exam√ development of dolichocephaly√ "Spalding sign" = overlapping fetal skull bones√ distorted fetus without recognizable structures√ skin edema (epidermolysis) = fetal maceration√ increased amount of echoes in amniotic fluid (= fetal tissue fragments)√ gas in fetal vascular system

"Vanishing Twin" "Fetus Papyraceus"

Notes:

"Vanishing Twin" = disappearance of one twin in utero due to complete resorption / anembryonic pregnancy *Incidence:*13-78% (mean 21%) before 14 weeks GA*Time:*<13 weeks MA√ NO sonographic evidence of twin pregnancy later in pregnancy

Notes:

"Fetus Papyraceus" =compression + mummification of fetus*Time:*in 2nd trimester*Path:*resorption of fluid resulting in paperlike fetal body + compression into adjacent membranes√ compressed mummified fetus plastered against uterine wall*Risk to surviving twin:* A.Dichorionic gestation (minimal risk)(1)Premature labor(2)Obstruction of labor by macerated fetusB.Monochorionic gestation(1)DIC in response to release of thromboplastin from degenerating fetus(a)into maternal circulation(b)into twin fetus through shared circulation (= twin embolization syndrome)

Notes:

Nonimmune Hydrops =excess of total body water evident as extracellular accumulation of fluid in tissues + serous cavities without antibodies against RBC*Incidence:*1:1,500 to 1:4,000 deliveries*Causes:* 1.Cardiac anomalies (40%):(a)structural heart disease (25%): AV septal defect, hypoplastic left heart, rhabdomyoma(b)tachyarrhythmia (15%)2.Hematologic causes: thalassemia, hemolysis, fetal blood loss3.Idiopathic (25-44%)4. Twin-twin transfusion (20%)5. Chromosomal abnormalities (6%): Turner syndrome6.Skeletal dysplasias: achondroplasia, achondrogenesis, osteogenesis imperfecta, thanatophoric dwarfism, asphyxiating thoracic dysplasia7. Renal disease (4%): congenital nephrotic syndrome8.Infections: toxoplasmosis, CMV, syphilis, Coxsackie virus, parvovirus9. Cervical tumors: teratoma10. Chest masses: cystic adenomatoid malformation, extralobar sequestration, mediastinal tumor, rhabdomyoma of heart, diaphragmatic hernia11.Abdominal masses: neuroblastoma, hemangioendothelioma of liver12. Placental tumors: chorioangioma*Prognosis:*46% death in utero; 17% neonatal death

Notes:

Immune Hydrops = ERYTHROBLASTOSIS FETALIS =lysis of fetal RBCs by maternal IgG antibodies*Pathophysiology:* rh-negative women (= no D antigen) may become isoimmunized if exposed to Rh-positive blood (= D allotype present); maternal IgM antibodies develop initially, later IgG antibodies with ability to cross placenta (= transplacental passage) *Prognosis:*(if untreated) 45-50% mild anemia, 25-30% moderate anemia (with neonatal problems only), 20-25% develop hydrops (death in utero / neonatally)*Cause of isoimmunization:* fetomaternal hemorrhage during pregnancy / delivery / spontaneous or elective abortion if fetus is D-positive; fetus has a 50% chance of being rh-negative as 56% of RhD-positive fathers are heterozygous for D antigen *At risk:* Caucasians (15%), Blacks (6%), Orientals (1%); absence of D antigen originates in Basques Determination of extent of disease by: (1)Optical density shift at 450 nm (= delta OD 450) reflects amount of bilirubin in amniotic fluid; reasonably reliable only >25 weeks MA; unreliable in alloimmunization due to Kell antibodies(2)Percutaneous umbilical cord sampling (PUBS) with direct determination of Hct and Hb√ anasarca (= skin edema)√ fetal ascites in 2nd trimester (indicates severe anemia with Hct <15%, Hb <4 g/dL; present in only 66%)√ pleural effusion√ increased diameter of umbilical vein√ subcutaneous edema (skin thickness >5 mm)√ polyhydramnios (75%)√ placentomegaly >6 cm√ pericardial effusion√ hepatosplenomegaly*Prophylaxis:* Rh immune globulin (RhoGAM® = antibody against D antigen) blocks antigen sites on Rh-positive cells in maternal circulation to prevent initiation of maternal antibody production; Rh immune globulin given at 28 weeks to all rh-negative women *OB-Management:* regular monitoring from 18 weeks on when maternal anti-D concentration exceeds 4 IU/mL (severe anemia unlikely if maternal antibodies <15 IU/mL) *Rx:*umbilical vein transfusion during PUBS

Notes:

FOLLICULAR CYST
=unruptured follicle / ruptured follicle that sealed immediately (after continued stimulation) = failure to ovulate / involute; sign of anovulatory cycle*Predisposed:*patients during puberty + menopause;S/P salpingectomy √ thin-walled, unilocular cyst√ size usually >2.5 cm / occasionally up to 10 cm in size√ usually multiple / may be single √ low-level internal echoes / fluid-debris level / septations / predominantly hyperechoic = hemorrhagic cyst (DDx: teratoma, abscess, torsion, malignancy, ectopic pregnancy)*Prognosis:*usually disappears after 1-2 menstrual cycles

Notes:

FUNCTIONAL OVARIAN CYST
Cause: (a)failure of involution of follicle / corpus luteum with changes in the menstrual cycle(b)excessive hormonal stimulation of follicles preventing normal follicular regression (eg, theca-lutein cysts)*Types:* (1)**Follicular cyst** (from preovulatory follicle):may elaborate estrogen, extremely common(2)**Corpus luteum cyst** (from postovulatory follicle):elaborates progesterone causing delayed menstruation / persistent bleeding(3)**Corpus albicans cyst** = from corpus luteum following regression of luteal tissue; no hormone production(4)**Theca lutein cyst**:in hyperstimulated ovary from ovary-stimulating drugs, twins, trophoblastic disease; elaborates estrogen(5)**Surface epithelial inclusion cyst**:common in postmenopausal women*Age:*any; in newborns (influence of maternal estrogen)*Incidence:*3-5-17% in postmenopausal women ● usually asymptomatic ● acute unilateral pelvic pain (from hemorrhage / pressure)√ unilocular smooth-walled cyst√ contents anechoic / with internal debris (from hemorrhage)√ up to 8-10 cm in diameter*Prognosis:*spontaneous regression is common but unpredictable; typically resolve within 2 menstrual cycles (less likely if cyst > 5 cm)*Rx:*(1)hormonal manipulation(2)surgery (absolutely indicated if cyst enlarges)(3)percutaneous aspiration (if chance of malignancy is nil as in infants)*DDx:*cystic teratoma, simple benign epithelial neoplasm, endometrioma in resolution, paraovarian cyst, quiescent hydrosalpinx

Notes:

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GARTNER DUCT CYST
*Frequency:*1-2%*Origin:*remnant of vaginal portion of mesonephric / wolffian duct with incomplete involution + persistent glandular secretion*Histo:*lined by flat cuboidal / columnar epithelium*May be associated with:*complex renal + urogenital malformations(1)Herlyn-Werner-Wunderlich syndrome = ipsilateral renal agenesis + ipsilateral blind vagina(2)Ectopic ureter inserting into Gartner duct cyst ● usually asymptomaticLocation:anterolateral aspect of proximal third of vaginal wall extending into ischiorectal fossa√ well-defined round lesion with fluid contents√ large cysts may displace ureter upward / protrude through introitus*Cx:*dyspareunia; interference with vaginal delivery

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Notes:

GASTROSCHISIS
=paramedian full-thickness abdominal fusion defect usually on right side of umbilical cord; may involve thorax; bowel is nonrotated and lacks secondary fixation to dorsal abdominal wall*Incidence:*1-2:10,000 livebirths (same as omphalocele), sporadic*Cause:*(a)abnormal involution of right umbilical vein resulting in rupture of anterior abdominal wall at area of weakness(b)premature interruption of right omphalo-mesenteric artery (normally persists proximally as superior mesenteric artery) resulting in ischemic damage to abdominal wall*Age of occurrence:*37 days (5 weeks) of embryonic life*Age of detection:*difficult <20 weeks GA*Associated anomalies* (5%): intestinal atresia / stenosis (25%; small size of opening leads to compression or torsion of vessels); ectopia cordis (rare) ● MS-AFP ≥2.5 MoM in 77-100%√ exteriorized bowel = thick-walled edematous freely floating loops outside fetal abdomen (due to lack of peritoneal covering)√ dilated intra- / extraperitoneal bowel√ <2-5 cm paraumbilical defect, usually on right side of cord insertion√ normal insertion of umbilical cord√ no fetal ascites√ polyhydramnios may be present√ liver / spleen may herniate infrequently√ malrotation / nonrotation of bowel*Cx before birth:*(1)Bowel obstruction(2)Peritonitis (exposure of bowel to fetal urine / meconium)(3)Perforation (from peritonitis)(4)Fetal growth restriction (38-77%) secondary to nutritional loss from exposed bowel*Cx after birth:*malrotation, jejunal / ileal atresia (18%), bowel necrosis, necrotizing enterocolitis, hyperalimentation hepatitis, prolonged intestinal motility dysfunction, chronic short-gut syndrome*Mortality rate:*17%Survival rate:87-100% after surgical treatment (during 1st day of life, not influenced by mode of delivery); death from premature delivery / sepsis / bowel ischemia

Notes:

GERM CELL TUMOR OF OVARY

=malignant (except for mature teratoma) ovarian tumors of varying histology*Age:*14 years on average ● pelvic / abdominal pain + mass ● elevated alpha-fetoprotein (60% in immature teratoma; 100% in endodermal sinus tumor) ● elevated b-HCG (30% of endodermal sinus tumors)√ average diameter of 15 cm√ unilateral, rarely bilateral√ calcifications (40%)√ homogeneously solid (3%), predominantly solid (85%), predominantly cystic (12%)

Notes:

GESTATIONAL TROPHOBLASTIC DISEASE
=group of disorders as a result of an aberrant fertilization event arising from trophoblastic elements of the developing blastocyst with invasive tendencyComponents of trophoblast: 1.Cytotrophoblast = stem cell with high mitotic activity2.Syncytiotrophoblast = synthesis of b-HCG3.Intermediate trophoblast = responsible for endometrial invasion + implantation ● increased levels of b-HCG*Incidence:*<1% of all gynecologic malignancies*Associated with:*molar pregnancy (most), post abortion, ectopic pregnancy, term pregnancy*Spectrum:*1.Benign hydatidiform mole (80-90%)2.Invasive mole (5-8-10%)3.Choriocarcinoma (1-2-5%)4.Placental site trophoblastic tumor (rare)*Cytogenesis:* =fertilization of one egg by two sperm = chromosomes completely / predominantly of paternal origin1.Diploid karyotype-46,XX = from fertilization of ovum by two 23,X sperm after loss of maternal haploid chromosomes-46,XY = from fertilization of a chromosomally empty ovum by two different sperm: ● in complete hydatidiform mole (almost 100%), invasive mole (almost 100%), choriocarcinoma (50%)2.Triploid karyotype (69,XXX; 69 XXY; 69,XYY)=fertilization of a normal ovum (23,X) by 2 different sperm thus containing 2/3 paternal chromosomes ● occurs in partial hydatidiform mole*At risk:*maternal age >35 years and <20 years, previous molar gestation, previous spontaneous abortions

Notes:

GRANULOSA CELL TUMOR
Most common hormone-active estrogenic tumor of ovary *Incidence:*1-2-3% of all ovarian neoplasms*Age:*puberty (5%), reproductive age (45%), postmenopausal (50%)*Path:*well-circumscribed, smooth / lobulated solid mass; foci of hemorrhage / cystic degeneration (when tumor gets larger)*Histo:*macro- / microfollicular, alveolar, trabecular, diffuse types ● precocious puberty ● irregular menstruation cycles, menorrhagia, amenorrhea ● abdominal pain, palpable adnexal massLocation: unilateral in 90-95% Dissemination:local extension, spread to peritoneum (similar to cystadenocarcinoma)√ multilocular cyst containing fluid / blood (most frequently)√ size up to 40 cm in diameter√ predominantly hypoechoic mass simulating fibroid√ endometrial glandular hyperplasia*Cx:*(1)Malignant transformation (5-25%)(2)Low-grade endometrial carcinoma (10%)(3)Recurrence (raised serum aromatase + estradiol levels)*Rx:*uni- / bilateral salpingo-oophorectomy ± postoperative chemotherapy*Prognosis:*85% 10-year survival rate

Notes:

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HELLP SYNDROME
=**H**emolysis, **E**levated **L**iver enzymes, **L**ow **P**latelets*Prevalence:*4-12% of patients with severe preeclampsia / eclampsia; higher in White women (24%), with delayed diagnosis of preeclampsia / delayed delivery (57%), in multiparous patients (14%) ● epigastric / RUQ pain (90%) ● nausea + vomiting (45%), occasionally jaundice ● headache (50%) ● demonstrable edema (55%)√ tender hepatomegaly√ fatty infiltration of liver (peak at 35th week)√ subcapsular hematoma of liver + kidney√ hepatic necrosis√ ascites + pleural effusions√ vitreous hemorrhage*Cx:*(1)Perinatal mortality (8-60%)(2)Maternal death (3-24%) from liver rupture, DIC, abruptio placentae, acute renal failure, sepsis

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Notes:

HYDATIDIFORM MOLE
=MOLAR PREGNANCY

Complete / Classic Mole Complete Mole With Coexistent Fetus (1-2%) Invasive Mole Partial Mole

Notes:

Complete / Classic Mole =fertilization of ovum by two 23,X sperm after loss of maternal haploid chromosomes (46,XX) or occasionally fertilization of an "empty egg" (= ovum with no active chromosomal material) by 2 different sperm (46,XY)*Histo:*generalized hydropic swelling of all chorionic villi with prominent acellular space centrally; pronounced trophoblastic proliferation of syncytio- and cytotrophoblast ● severe eclampsia prior to 24 weeks ● uterus too large for dates (in 50%) ● 1st trimester bleeding ● marked elevation of b-HCG with hyperemesis ● passing of grapelike vesicles per vagina ● hyperthyroidism (due to thyroid-stimulating properties of b-HCG) ● anemia (secondary to plasma volume expansion + vaginal bleeding) ● diploid karyotype, almost always paternal XX chromosomes√ hyperechoic to moderately echogenic central uterine mass interspersed with punctate hypoechoic areas√ numerous discrete cystic spaces (= hydropic villi) within a central area of heterogeneous echotexture√ in 25% atypical appearance:√ large hyperechoic areas (blood clot) + areas of cystic degeneration resembling incomplete abortion√ single large central fluid collection with hyperechoic rim mimicking an anembryonic gestation / abortion√ no fetal parts / no chorionic membrane√ bilateral theca lutein cysts (18-37%), which may take 4 months to regress after evacuation of a molar pregnancy*Prognosis:*in 80-85% benign, in 15-20% invasive mole / choriocarcinoma*Rx:*dilatation + suction curettage (curative in 85%)*DDx:*(1)Hydropic degeneration of the placenta (associated with incomplete / missed abortions)(2)Degenerated uterine leiomyoma(3)Incomplete abortion = retained products with hemorrhage(4)Choriocarcinoma

Notes:

Complete Mole With Coexistent Fetus (1-2%)
=molar degeneration of one conceptus of a dizygotic twin pregnancy with same risk of malignant degeneration as in classic mole ● vaginal bleeding in 2nd trimester ● uterus large for dates ● abnormally elevated serum b-HCG ● amniocentesis with normal diploid karyotype excludes diagnosis of partial mole√ normal gestation with placenta + separate typical echogenic material of a hydatidiform mole√ ovarian theca lutein cysts*Prognosis:*fetal survival unlikely due to maternal complications from coexistent mole

Notes:

Invasive Mole =CHORIOADENOMA DESTRUENS*Histo:*excessive trophoblastic proliferation with presence of villous structure + invasion of myometrium*Preexisting condition:*complete / partial hydatidiform mole ● history of previous molar gestation / missed abortion (75%) ● continued uterine bleeding ● persistently elevated b-HCG levels (with failure of b-HCG to return to undetectable levels after treatment of a complete hydatidiform mole)√ hyperechoic tissue with punctate lucencies√ irregular focal hyperechoic region within myometrium√ bilateral theca lutein cysts, 4-8 cm in size√ myometrial invasion occasionally demonstrable*Rx:*chemotherapy, hysterectomy (if at risk for uterine perforation)

Notes:

Partial Mole =areas of molar change alternating with normal villi + fetus with significant congenital anomalies*Histo:*focal proliferations of syncytiotrophoblast; normal villi interspersed with hydropic villi ● triploid karyotype (66% XXY; 33% XXX) due to fertilization of single ovum with 2 sperm ● early onset of preeclampsia√ nearly always coexistent fetus with severe abnormalities√ placenta with numerous cystic spaces*Prognosis:* (1)frequently spontaneous abortion (unrecognized as mole for lack of karyotyping of the abortus)(2)no survival of triploid fetus(3)3% risk of persistent gestational trophoblastic neoplasia

Notes:

HYDRO- / HEMATOMETROCOLPOS

=accumulation of sterile fluid (hydro~) / blood (hemato~) / pus (pyo~) within uterus (~metria) + vagina (~colpos);(a)premenarcheally = secretions + mucus(b)postmenarcheally = blood *Incidence:*1:16,000 female births*Etiology*: A.CONGENITAL OBSTRUCTION(a)persistent urogenital sinus = single exit chamber for bladder + vagina; separate orifice for anus; caused by virilization of female fetus / intersex anomaly / arrest of normal vaginal development*Frequently associated with:* ambiguous genitalia *Age:*newborn period(b)cloacal malformation = single perineal orifice for bladder + vagina + rectum; caused by early embryologic arrest*Frequently associated with:* duplex genital tract *Age:*newborn period(c)imperforate hymen, transverse vaginal septum, segmental vaginal atresia, imperforate cervix, blind horn of bicornuate uterus, Mayer-Rokitansky-Küster-Hauser syndrome (= agenesis of uterus + vagina with active uterine anlage) ● primary amenorrhea = "delayed menarche" ● cyclical abdominal pain ● interlabial mass*Age:*puberty✧Hematometrocolpos / hematocolpos are due to imperforate hymen / transverse vaginal septum✧Hematometra is due to cervical dysgenesis + vaginal agenesis / Mayer-Rokitansky-Küster-Hauser syndrome / obstructed uterine horn*May be associated with:* imperforate anus, hydronephrosis, renal agenesis / dysplasia, polycystic kidneys, duplication of vagina + uterus, sacral hypoplasia, esophageal atresia 2.ACQUIRED OBSTRUCTIONneoplastic obstruction of endocervical canal / vagina, postpartum infection, attempted abortion, cervical stenosis after radiotherapy, postsurgical scarring (eg, dilatation and curettage, traumatic delivery), senile contraction ● vague pelvic discomfort ● pain during defecation / urination ● asymptomatic✓ smooth symmetric enlargement resulting in pear-shaped uterus ± distended vagina✓ varying amounts of low-level internal echoes centrally within uterus continuous with vaginal canal✓ hematosalpinx ± endometriosisOB-US: ✓ cystic / midlevel echogenic retrovesical mass (mucous secretions secondary to stimulation by maternal estrogens during fetal life)✓ cystic mass ± fluid-debris level (distended vagina)✓ bladder often not identified (compression by distended vagina)*DDx:*ovarian cyst, duplication cyst, meconium cyst, mesenteric cyst, rectovesical fistula, anterior meningocele, cystic tumor, trophoblastic disease, degenerating leiomyoma / leiomyosarcoma*Cx:*endometritis, myometritis, parametritis (= pelvic lymphangitis), pelvic abscess, septic pelvic thrombophlebitis, urinary tract infection

Notes:

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IMMATURE TERATOMA OF OVARY
=EMBRYONAL TERATOMA = MALIGNANT TERATOMA = SOLID TERATOMA*Histo:*immature tissue resembling those of the embryo; grade 0-3 reflect amount of immature neuroectodermal tissue*May be associated with:* gliomatosis peritonei = multiple peritoneal implants of mature glial tissue ● elevated AFP levels (50%) ● no elevation of serum HCG levels√ predominantly solid tumor with numerous cysts of varying size√ scattered calcifications (due to invariable association with mature teratoma)

Notes:

INFERTILITY
=failure to conceive after 1 year of unprotected intercourse*Incidence:*affects 10-15% of couples*Etiology:* (a)female factors (55%):Tubal disease (10-20-40%): congenital anomalies, DES exposure, pelvic inflammatory disease, salpingitis isthmica nodosa, endometriosis, postoperative factors, polyp, neoplasm, ectopic pregnancy Uterine factors (2-5%): bicornuate uterus, septate uterus, DES exposure, intrauterine adhesions, endometrial inflammation / infection, uterine neoplasm, complications after pregnancy, leiomyoma Ovulatory disorder (10-20%) Pelvic factors (20-25%) Cervical factors (5-10%) (b)male factors (40%)(c)combination of factors (15-25%)(d)unknown cause (5-10%)Tests: ● history + physical examination ● laboratory tests (mainly hormonal) ● basal body temperature measurement ● postcoital test ● cervical culture ● endometrial biopsy ● sonographic monitoring of ovaries ● sperm agglutination studies ● in vitro mucus penetration test ● laparoscopy + hysteroscopy ● hysterosalpingography

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INTRAUTERINE CONTRACEPTIVE DEVICE

√ double echogenic line with plastic IUD√ reverberation echoes with metal IUD*Types of IUD:* 1.Lippes loop√ 4-5 echogenic dots on SAG view√ horizontal line / dot on TRV view2.Saf-T-coil√ echogenic solid line on SAG view√ series of echoes / dot on TRV view3.Copper 7 / Copper T / Progestasert√ dot in fundus + solid line in corpus on SAG view√ solid line in fundus + dot in corpus on TRV view4.Dalkon shield (no longer produced) *Cx:*pelvic inflammatory disease (2-3-fold risk compared with that of non-IUD users) in 35%; actinomycosis with IUD in place for >6 years

"Lost IUD" IUD & Pregnancy

Notes:

"Lost IUD" =locator device not palpated*Cause:*1.expulsion of IUD2.migration of thread3.detachment of thread4.uterine perforation of IUD◊Abdominal plain film is indicated if IUD not identified by US!

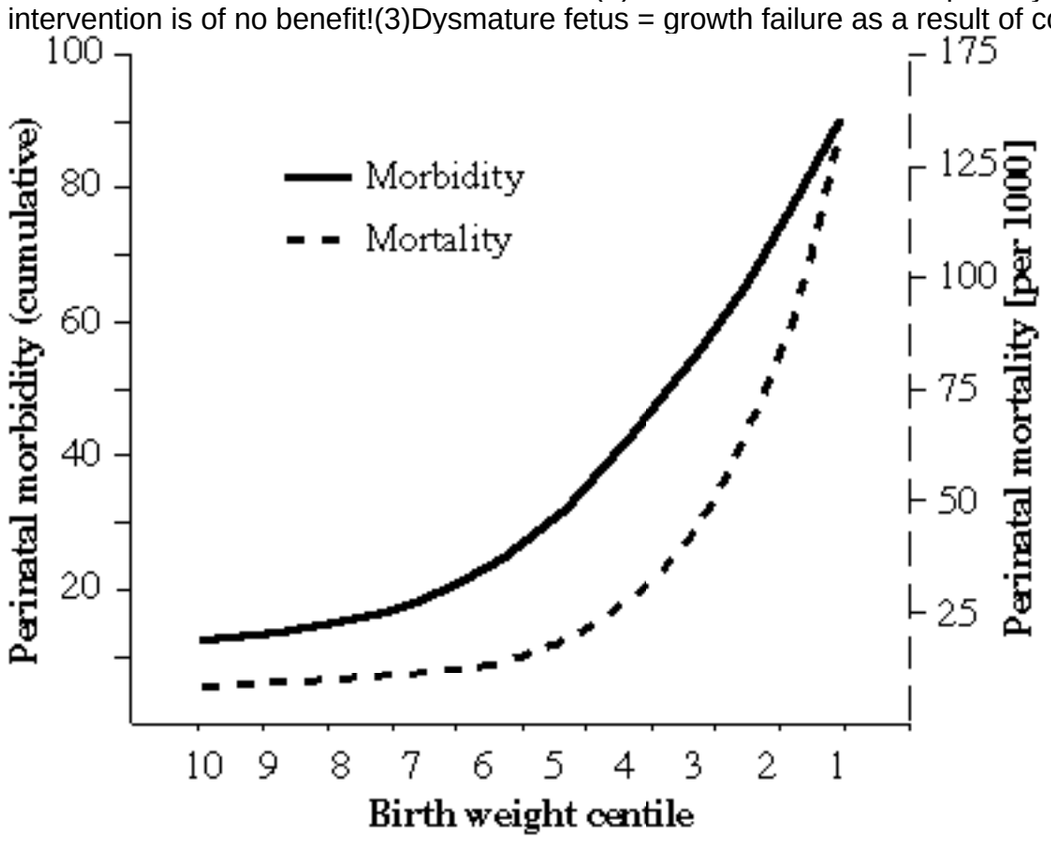
Notes:

IUD & Pregnancy √ IUD may not be visualized after 1st trimester (as uterus grows IUD is drawn into cavity)*Prognosis:*high risk of septic abortion*Rx:*early removal of IUD if string remained in vagina

Notes:

INTRAUTERINE GROWTH RESTRICTION
=FETAL GROWTH RETARDATION=perinate with a weight at/below the 10th percentile for gestational age occurring as a result of a pathologic process inhibiting expression of normal intrinsic growth potentialfor twin pregnancy:discordant weight >25%◊Fetal weight at/below 10th percentile for age will classify 7% of normal fetuses as growth retarded!◊IUGR is primarily an ultrasound diagnosis!*Prevalence:*3-7% of all deliveries; in 12-47% of all twin pregnancies; in 25% of fetuses following birth of a growth-retarded sibling / stillborn*Etiology:* A.UTEROPLACENTAL INSUFFICIENCY (80%)=injury during period of cell hypertrophy resulting in decreased cell size with features of intrauterine starvation + protective cardiac output redistribution reflex ● absence of body fat ● diminished liver and muscle glycogen1.Maternal causes√ asymmetric IUGR / symmetric IUGR (in severe cases)(a)deficient supply of nutrients:cyanotic heart disease, severe anemia (in 10-25% of sickle cell anemia), maternal starvation, life in high altitudes, drugs (anticonvulsants, methotrexate, warfarin), alcohol abuse (dose related), illicit drugs (up to 50% with heroine addiction, 30% with cocaine abuse), uterine anomaly, multiple gestation (in 15-20%) (b)maternal vascular disease resulting in inadequate placental perfusion:nicotine-induced release of catecholamines, preconceptual diabetes, preeclampsia, chronic renal disease collagen vascular disease (SLE) (c)maternal dermographics:maternal age (adolescence / advanced), nulliparous mother, small short habitus, racial influence (Asians) 2.Primary placental causesExtensive placental infarctions, chronic partial separation (abruption), partial mole, Breus mole, chorioangioma, placenta previa, low implantation, placental metastases (breast, melanoma), placentitis (luetic, malaria) *Histo:*reduction in placental villous surface area + in number of capillary vessels√ asymmetric growth failureB.PRIMARY FETAL CAUSES (20%)=injury during the period of cell hyperplasia(= embryogenesis) producing profound reduction in cell number across all cell lines√ symmetric IUGR (globally decreased intrinsic growth)√ normal / increased amniotic fluid volume1.Chromosomal abnormalities (in 2-6%):triploidy, tetraploidy, trisomy 13 + 18 + 21, aneuploidy (Turner syndrome), partial deletion (4-p, 5-p [cri du chat], 13-q), partial trisomy (4-p, 18-p, 10-q, 18-q), unbalanced translocation (chromosomes 4 + 15), balanced translocation (chromosomes 5 + 11) 2.Structural anomalies: congenital heart disease, genitourinary anomalies, CNS anomalies, dwarfism3.Viral infection: rubella (in 40-60%), CMV, varicella (in 40%)◊All fetuses with IUGR need to have a detailed and often repeated search for structural anomalies! ● fundal height as screening test (37-60% true positive, 40-55% false negative; 26-60% false positive)*Sequence of events in fetal hypoxia:* nonreactive CST > absence of fetal breathing > nonreactive NST > diminished fetal movements > absence of fetal movements > absence of fetal tone PHENOTYPES 1.**Pure symmetric IUGR** = decreased-cell-number IUGR = early-insult IUGR = low-profile IUGR=proportionate reduction of all fetal measurements due to(a)intrinsic alteration in growth potential (usually due to chromosomal abnormalities)(b)severe nutritional deprivation overwhelming protective brain-sparing mechanism occurring prior to 26 weeks MA + persisting until delivery√ proportionate decrease in HC and AC maintaining normal HC:AC ratios√ estimated fetal weight <10th percentile for age by middle of 2nd trimester2.**Mixed IUGR** =onset of IUGR during period of mixed hyperplasia / hypertrophy with near normal inherent fetal growth potential but decreased size + impaired function of placenta√ impaired fetal growth ± asymmetry√ abnormal Doppler umbilical artery flow velocity (due to increased placental vascular resistance)√ progressive oligohydramnios3.**Asymmetric IUGR** = decreased-cell-size IUGR=late-onset IUGR = late-flattening IUGR (75%)=disproportionate reduction of fetal measurements due to uteroplacental insufficiency with preferential shunting of blood to fetal brain occurring after 26 weeks GA ◊IUGR usually not detectable before 32-34 weeks GA (time of maximal fetal growth)!Effective time for screening:34 weeks MARoutine surveillance:every 4 weeks beginning at 26 weeks MA √ AC >2 SD below the mean for age = highly suspicious; AC >3 SD below mean for age= diagnostic (AC single most effective fetal parameter for detection of asymmetric IUGR) √ high HC/AC and FL/AC ratios (head size + femur length less affected)√ fetal weight percentile useful for follow-up√ accelerated placental maturity√ decreased amniotic fluid volume√ elevated umbilical artery S/D ratio◊FL/AC ratio + umbilical artery S/D ratio are the only effective techniques to screen for IUGR on a single exam with late prenatal care in 3rd trimester! DIAGNOSTIC ULTRASOUND METHODS ◊An accurate fix on fetal age dictates accuracy of diagnosis of IUGR (early US exam, clinical dates, early physical exam, pregnancy test)!◊Every effort needs to be made to determine the underlying cause for growth failure as it effects management + perinatal morbidity and mortality!1.Fetal morphometric indicesThe three key parameters for diagnosing IUGR are (1) low estimated fetal weight (EFW), (2) low amniotic fluid volume (AFV), (3) maternal hypertension (HBP)!

Sonographic criteria for IUGRPPV [%]NPV [%] advanced placental grade1694elevated FL/AC18-2092-93abnormal UA waveform17-37low total intrauterine volume21-2492-97small BPD21-4492-98slow BPD growth rate3597low EFW4599oligohydramnios5592elevated HC/AC6298 (a)intrafetal proportions√ elevated HC:AC ratio for dysmature IUGR (overall 36% sensitive, 90% specific, 67% PPV, 72% NPV; 93% sensitive in fetus >28 weeks MA with severe dysmature IUGR)◊Early-onset dysmature IUGR not detectable!◊May not be used in anomalous fetuses!(b)rate of growth = growth velocity√ HC, AC, FL measurements allow DDx between erroneous dates + normal small fetus + fetus with intrinsic abnormality√ plot growth curves◊Minimum time interval of 2 weeks necessary!2.Amniotic fluid volume◊Screening for decreased amniotic fluid is of value in the fetus with dysmature IUGR (60-84% sensitive, 79-100% accurate)!√ normal amniotic fluid does not exclude IUGR√ oligohydramnios means dysmature IUGR in a fetus with normal GU tract until proven otherwise (DDx: trisomy 13 + 18)3.Fetal morphologic assessment + fat distribution √ diminished thigh circumference√ absent paraspinal fat pad (posterior neck)√ reduced / absent malar fat pads√ disproportionately small liver size√ increased small bowel echogenicity (= absent omental fat)4.Placental assessment√ increased placental calcium deposition5.Doppler blood flow velocitiesa.Nonstress test (NST)b.Contraction stress test (CST)c.Umbilical artery waveform◊Not useful with unknown dates / for screening!*Physiology:*S/D ratio increases with sampling site closer to fetus + increasing fetal heart rate; S/D ratio decreases with advancing gestational age√ elevated systolic:diastolic ratio (S/D ratio >3.0 beyond 30-34 weeks GA) indicates an increase in vascular resistance within placental circulation√ absent diastolic flow = 50-90% mortality rate√ reverse diastolic flow = impending fetal collapsed.Uterine artery waveform (measured at its point of overlap with external iliac artery)√ S/D ratio >2.6 after 26 weeks GA√ persistence of early diastolic notche.Fetal aortic flow volume (no proven usefulness)√ decrease in blood flow to <185-246 mL/kg/min6.Biophysical profile*Accuracy:*false-negative fetal death rate of 0.645/1000 fetuses within 1 week the last normal BPP; 33% sensitivity, 17% positive predictive value7.Invasive fetal testing: fetal blood analysis for karyotyping, hypoxemia, hypercapnia, acidemia, hypoglycemia, hypertriglyceridemia
*Cx:*increased risk for perinatal asphyxia, meconium aspiration, electrolyte imbalance from metabolic acidosis, polycythemia*Neonatal Cx:*pulmonary hemorrhage + vasoconstriction, persistent fetal circulation, intracranial hemorrhage, bowel ischemia, necrotizing enterocolitis, acute renal failure *Prognosis:*6-8-fold increase in risk for intrapartum death + neonatal death◊20% of all stillborn fetuses are growth retarded! *DDx of fetus small for gestational age (SGA): Definition:*generic clinical term describing a group of perinates at/below the 10th percentile for gestational age without reference to etiology(1)Small normal fetus = constitutionally small fetus(80-85%) ◊No indication for surveillance / intervention!(2)Small abnormal fetus = primary growth failure associated with karyotype anomaly / fetal infection (5-10%)◊Active intervention is of no benefit!(3)Dysmature fetus = growth failure as a result of compromised placental function (10-15%)◊Intensive management is likely of benefit!



Notes:

KRUKENBERG TUMOR
=ovarian tumors from GI tract cancer (colon:stomach = 2:1) now including pancreatic + biliary primaries; 2% of females with gastric cancer develop Krukenberg tumor
Krukenberg tumors antedate the discovery of the primary lesion in up to 20%!*Age:*any age, most common in 5th-6th decade in 80% bilateral hypo- / hyperechoic mass ± cystic degeneration

Notes:

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LIMB-BODY WALL COMPLEX
*Prevalence:*1:10,000 live births*Cause:*? severe form of amniotic band syndrome;? early vascular disruption; ? embryonic dysplasia due to malformation of ectodermal placodes A.EXTERNAL DEFECTS1.Ventral wall anomaly√ large eccentric defectLocation:L:R = 3:1 (DDx: gastroschisis)2Craniofacial defects: anencephaly, cephalocele, facial cleft3.Limb reductions4.Spinal defects: dysraphism, scoliosisB.INTERNAL DEFECTS (in 95%)1.Cardiac defects2.Diaphragmatic absence3.Bowel atresia4.Renal abnormalities: agenesis, hydronephrosis, dysplasia√ persistence of extraembryonic coelom (= separation of amnion + chorion)*Prognosis:*invariably fatal shortly after birth

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Notes:

MACROSOMIA

=FETAL GROWTH ACCELERATION=fetus large for gestational age (LGA) with EFW >90th percentile for age / >4,000 g at term√ AC >3 SD above the mean for age (most reliable measurement)√ estimated fetal weight (EFW) including fetal head, abdomen, femur length >90th percentile (± 15%accuracy)√ low FL:AC ratio√ low HC:AC ratio√ enlarged thigh circumference√ low FL:thigh circumference ratio*Risk:*shoulder dystocia, prolonged labor, meconium spiration

Notes:

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MASSIVE OVARIAN EDEMA
=tumorlike condition with marked enlargement of one / (occasionally) both ovaries due to accumulation of edema fluid in stroma*Age:*6-33 (average 21) years*Cause:* (1)partial / intermittent torsion (obstruction to ovarian lymphatic + venous drainage)(2)ovarian stromal proliferation with enlargement of ovary susceptible to torsion*Histo:*edematous ovarian stroma + extensive fibromatosis surrounding primordial follicles, luteinized cells ● acute / intermittent lower abdominal pain for month ● masculinization (in chronic phase)√ solid / multicystic adnexal mass√ ovarian diameter of 5-40 (mean 11.5) cm*Rx:*oophorectomy / salpingo-oophorectomy / wedge resection with ovarian suspension

Notes:

MAYER-ROKITANSKY-KÜSTER-HAUSER SYNDROME
(1)vaginal agenesis / hypoplasia of proximal + middle segments(2)intact ovaries + fallopian tubes (3)variable anomalies of uterus (agenesis / hypoplasia), urinary tract (renal agenesis, pelvic kidney in 40%), skeletal system*Frequency:*1:4,000-1:5,000*Cause:*lack of müllerian development ● normal external genitalia ● shallow distal vaginal pouch (derived from urogenital sinus) ● amenorrhea ● cyclic pelvic pain (secondary to functioning endometrium within rudimentary uterine tissue)*Rx:*neovaginoplasty

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MUCINOUS OVARIAN TUMOR
*Incidence:*20% of all ovarian tumors; 2nd most common benign epithelial neoplasm of ovary (after serous ovarian adenoma)*Histo:*single layer of nonciliated tall columnar epithelium with clear cytoplasm of high mucin content (similar to endocervix + intestinal epithelium) 80% benign, 10% borderline, 10% malignant*Age:*middle adult life, rare before puberty + after menopause*Cx:*rupture may lead to pseudomyxoma peritonei

Mucinous Cystadenoma Mucinous Cystadenocarcinoma

Notes:

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Mucinous Cystadenoma *Prevalence:*20% of all benign ovarian neoplasms*Age:* 3rd-5th decade of life√ multilocular cyst with numerous thin septa√ cysts frequently have high protein content:√ low-level echoes in cysts√ high attenuation on CT√ hyperintense on T1WI√ usually unilateral, bilateral in 5%

Notes:

Mucinous Cystadenocarcinoma difficult to differentiate from benign variety √ solid tissue areas: thick septa + other soft-tissue elements within septated cyst√ usually unilateral, bilateral in 20%√ capsular infiltration with loss of definition + fixation*Cx:*pseudomyxoma peritonei

Notes:

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NUCHAL CORD
=umbilical cord encircling fetal neck: single loop > two loops (2-3%) > 3 or more loops (<1%)*Incidence:*25% of pregnancies; frequently transient*Associated with:*increased cord length, small fetus, vertex presentation, polyhydramnios ● generally not of clinical significance: no difference in 5-minute Apgar score, no increase in infant mortality√ two adjacent cross sections of cord on longitudinal view of neck (diagnosis facilitated by color Doppler flow)√ indentation of skin by nuchal cord suggests tight loop*Risk:*signs of fetal distress (fetal bradycardia, variable decelerations, depressed 1-minute Apgar score)OB management: 1.Assess fetal well-being (biophysical profile biweekly, NST, fetal growth)2.Vaginal delivery permissible if without evidence of fetal compromise3.Intervention only for signs of fetal distress

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Notes:

OMPHALOCELE
=midline defect of anterior abdominal wall due to failure to form the umbilical ring during 3rd to 4th week of gestation with herniation of intraabdominal contents into base of umbilical cord*Prevalence:*1:4,000 to 1:5,500 pregnancies*Cause:* (a)migration failure of lateral mesodermal body folds√ omphalocele contains liver(b)persistence of primitive body stalk beyond 12th week MA√ omphalocele contains primarily bowel *Age:*earliest detection at 12 weeks menstrual age High incidence of ASSOCIATED ANOMALIES (45-88%): 1.Chromosomal (10-30-58%): trisomy 13, 18, 21, Turner syndrome (13% with liver in omphalocele, 77% with bowel in omphalocele), triploidy2.Genitourinary (40%): bladder exstrophy√OEIS complex = **O**mphalocele + bladder **E**xstrophy + **I**mperforate anus + **S**pinal anomalies3.Cardiac (16-30-47%): VSD, ASD, tetralogy of Fallot, ectopia cordis in pentalogy of Cantrell, DORV4.Neural tube defects (4-39%): holoprosencephaly, encephalocele, cerebellar hypoplasia5.IUGR (20%)6.Beckwith-Wiedemann syndrome (5-10%)7.GI tract:intestinal atresia (vascular compromise); malrotation; abnormal fixation of liver, esophageal atresia, facial cleft, diaphragmatic hernia 8.Limb-body wall deficiency; cystic hygroma ● MS-AFP ≥2.5 in 40-70%√ midline central defect at base of umbilical cord insertion√ defect over entire ventral abdominal wall (mean size 2.5-5 cm)√ widened cord where it joins the skin of the abdomen√ cord inserting at apex of defect√ herniation of abdominal viscera at base of umbilical cord: liver (27%) ± stomach ± bowel√ covering amnioperitoneal membrane (inner layer = peritoneum; outer layer = amnion); may rupture in exceedingly rare cases√ hypoechoic loose mesenchymal tissue (= Wharton jelly) between layers of membrane√ ascites within herniated sac√ polyhydramnios (occasionally oligohydramnios) *mnemonic:*"OMPHALOCele"**O**ther anomalies (common) **M**embrane surrounding viscera **P**erfectly midline **H**eart anomalies **A**scites **L**iver commonly herniated **O** for "zero" bowel complications **C**hromosomal abnormalities (common) *Cx:*(1)Infection, inanition(2)Immaturity (23%)(3)Rupture of hernial sac(4)Intestinal obstruction*Mortality rate:*10% mortality if isolated abnormality; 80% with one / more concurrent malformations; nearly 100% with chromosomal + cardiovascular abnormalities*DDx:*(1)Gastroschisis (usually right-sided defect)(2)Limb-body wall complex (usually left-sided defect)

Pseudo-omphalocele

Notes:

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Pseudo-omphalocele (1)Deformation of fetal abdomen by transducer pressure coupled with an oblique scan orientation may give the appearance of an omphalocele √ obtuse angle between pseudomass and fetal abdominal wall(2)Physiologic herniation of midgut into umbilical cord between 8th and 12th week of gestation√ herniated sac never contains liver√ herniated sac usually <7 mm√ disappears by 12th week GA

Notes:

OMPHALOMESENTERIC DUCT CYST

*Etiology:*persistence + dilatation of a segment of the omphalomesenteric / vitelline duct joining the embryonic midgut and the primary yolk sac, which is formed during the 3rd week and closed by the 16th week of gestation*Histo:*cyst lined by columnar mucin-secreting gastrointestinal epitheliumM:F = 3:5 Location:usually in close proximity to fetus√ umbilical cord cyst up to 6 cm in diameter√ beneath amniotic surface of cord (= eccentric)*Cx:*(1)Compression of umbilical vessels by expanding cyst(2)Erosion of umbilical vein from acid-producing gastric mucosal lining*DDx:*allantoic cyst, umbilical cord hematoma

Notes:

OVARIAN CANCER
8th leading cause of cancer in women; 3rd most common gynecologic malignancy = 25% of all gynecologic malignancies; leading cause of death of all female cancers (60%); 5th leading cause of cancer deaths in women; accounts for 50% of cancer deaths of female genital tract *Etiology*:ovarian surface epithelium proliferates temporarily to repair defect after rupture of ovum which may result in an "inclusion body" / "cystoma"; an error in DNA replication within inclusion body may occur resulting in inactivation / loss of a tumor-suppressor gene*Incidence:*affects 1:2000 women; 50 cases per year per 100,000 women (33 cases per year per 100,000 women > age 50); 26,700 new cases + 14,500 deaths in 1996*Age:*increasing with age; peaking at 55-59 years (80% of cases in women >50 years)*Histo:* A.EPITHELIAL TUMORS (60-70%)(a)serous tumor resembling ciliated columnar cells of the fallopian tubes (50%)(b)endometrioid tumor similar to endometrial adenocarcinoma (15-30%)(c)mucinous tumor similar to endocervical canal epithelium (15%)(d)clear cell carcinoma = mesonephroid tumor (5%)(e)Brenner tumor (2.5%)(f)undifferentiated tumor (<5%)B.GERM CELL TUMORS (15-30%)Most common malignant ovarian neoplasm in girls + young women *Age:*4-27 years(a)mature teratoma (10%) = the only benign variety(b)dysgerminoma (1.9%)(c)immature teratoma (1.3%)(d)endodermal sinus tumor (1%)(e)malignant mixed germ cell tumor (0.7%)(f)choriocarcinoma (0.1%)(g)embryonal carcinoma (0.1%)C.METASTASES (5-10%)D.STROMAL TUMORS (5%) *Size versus risk of malignancy:*<5 cmin3%5-10 cmin10%>10 cmin65%*Increased risk:* nulliparity, early menarche, late menopause, Caucasian race, higher socioeconomic group, positive family history for ovarian cancer (risk factor of 3 with one close relative, risk factor of 30 with two close relatives affected with ovarian cancer), history of breast cancer (risk factor of 2) / early colorectal cancer (risk factor of 3.5) ◊Lifetime risk of ovarian cancer = 1:70 women (1.4%)!*Decreased risk:* pregnancy, use of oral contraceptives, breast-feeding *Stage* (FIGO system) based on staging laparotomy ILimited to ovaryI alimited to one ovaryI blimited to both ovariesI c+ positive peritoneal lavage / ascitesIILimited to pelvisII ainvolvement of uterus / fallopian tubesII bextension to other pelvic tissuesII c+ positive peritoneal lavage / ascitesIIILimited to abdomen = intraabdominal extension outside pelvis / retroperitoneal nodes / extension to small bowel / omentumIVHematogenous disease (liver parenchyma) / spread beyond abdomen◊50-75% of patients have stage III / IV disease at time of diagnosis!*Spread:* (1)direct extension through subperitoneal space (sigmoid mesocolon on left, cecum + distal ileum on right)(2)exfoliation of tumor cells into peritoneal space (often microscopic) with frequent seeding to:-pouch of Douglas-termination of small bowel mesentery-superior aspect of sigmoid-right paracolic gutter-omentum(3)lymphatic spread ● occasional pelvo-abdominal pain ● constipation, urinary frequency ● early satiety ● ascites ● paraneoplastic hypercalcemia ● elevated CA-125 levels (= high-molecular-weight glycoprotein with normal level of <35 units/mL):->35 units/mL in 29% of stage I disease->65 units/mL in 21% of stage I disease◊CA-125 levels elevated in 80% of ovarian cancers (60% of mucinous + 20% of nonmucinous tumors)!◊CA-125 levels elevated in 30% of benign processes (fibroid, pregnancy, menstruation, endometriosis, PID, benign ovarian tumors, cirrhosis)!US: ◊Screening finds adnexal cysts in 1-15% of postmenopausal women; only 3% of ovarian cysts <5 cm are malignant!√ solid / partly solid consistency + papillae√ postmenopausal ovarian volume >9 cm^3 √ low-resistance Doppler waveform (due to lack of muscular layer of arterial wall in neoplasms) with much overlap between benign + malignant tumors:RI <0.40, PI <1.0 *Prediction:*gray-scale US = 99% NPV; presence of internal flow = 49% PPV; abnormal PI/RI = 37-47% PPV√ presence of color flow (malignant vs. benign tumors= 93% vs. 35%) usually within thick wall, septa, papillary projections, solid inhomogeneous areas √ omental / peritoneal masses ("omental cake")√ pseudomyxoma peritonei (with tumor rupture)√ liver metastases√ ascitesBE: √ serosal spiculation / tethering√ annular constriction / complete obstruction*Rx:* stage I:total abdominal hysterectomy (TAH) + bilateral salpingo-oophorectomy (BSO) ± melphalan / intraperitoneal P-32stage >I:TAH/BSO + surgical cytoreduction (debulking) + 6 cycles of chemotherapy (cyclophosphamide + cisplatin)*Prognosis (without change in past 60 years):* 20-40% overall 5-year survival rate, 5-8% for stage IV, 14-30% for stage III, 50% for stage II, 80-90% for stage I *DDx:*tubo-ovarian abscess, dermoid cyst, endometrioma

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OVARIAN FIBROMA /FIBROTHECOMA

*Incidence:*3-4% of all ovarian tumors; bilateral in <10%*Age:*usually menopausal / postmenopausal*Histo:*mesenchymal tumor consisting of intersecting bundles of collagen-producing spindle cells; fibrothecomas also have a small population of theca cells that contain intracellular lipids ● usually asymptomatic ● Meigs syndrome (in only 1%)√ ascites (in 10-15% of tumors >10 cm)√ ± cystic degeneration and edema in larger lesionsUS: √ hypoechoic mass with marked sound attenuationMR: √ low signal intensity on T1WI + T2WI (less than or equal to myometrium)CT: √ well-defined solid homogeneous / slightly heterogeneous mass*DDx:*pedunculated uterine leiomyoma

Notes:

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OVARIAN HYPERSTIMULATION SYNDROME
*Incidence:*severe OHSS in 1.5-6% under Perganol therapy*Etiology:* (1)Induced by HCG therapy with human menopausal gonadotropin (Perganol), occasionally with clomiphene (Clomid)(2)Hydatidiform mole(3)Chorioepithelioma(4)Multiple pregnancies*Path:*enlarged ovaries with multiple follicular cysts, corpora lutea, edematous stroma (fluid shift secondary to increased capillary permeability) ● abdominal pain (100%) + distension (100%) ● nausea (100%), vomiting (36%) ● acute abdomen (17%) ● dyspnea (16%) ● thrombophlebitis (11%) ● marked hemoconcentration ● fainting (11%) ● blurred vision (5%) ● anasarca (5%) ● hydrothorax ● enhanced fertility√ ovary >5 cm in longest dimension containing large geometrically packed follicles√ ovarian cyst >10 cm (100%): usually disappear after 20-40 days√ ascites (33%)√ pleural effusion (5%)√ hydroureter (11%)*Cx:*(related to volume depletion)(1)Hypovolemia + hemoconcentration(2)Oliguria, electrolyte imbalance, azotemia(3)Death from intraabdominal hemorrhage / thromboembolic event

Notes:

OVARIAN VEIN THROMBOSIS

Etiology:

(1)Bacterial seeding from puerperal endometritis with secondary thrombosis (pregnancy + puerperium are hypercoagulable states)=**puerperal ovarian vein thrombophlebitis**

(2)Pelvic inflammatory disease(3)Gynecologic surgery(4)Malignant tumors(5)Chemotherapy*Incidence:*1:600-1:2,000 deliveries ● presents on 2nd / 3rd postpartum day ● lower abdominal / flank pain (>90%) ● palpable ropelike tender abdominal mass (50%) ● fever if diagnosis delayedLocation:right ovarian vein (80%), bilateral (14%), left ovarian vein (6%)CT: √ tubular structure in location of ovarian vein with low-density center + peripheral enhancement*Cx:*IVC thrombosis; pulmonary embolism (25%); septicemia; metastatic abscess formation*Mortality:*5%*Rx:*IV antibiotics + heparin; ligation of involved vessel at most proximal point of thrombosis after failure to improve after 3-5 days*DDx:*appendicitis, broad-ligament phlegmon / hematoma, torsion of ovarian cyst, urolithiasis, pyelonephritis, degenerated pedunculated leiomyoma, pelvic cellulitis, pelvic / abdominal abscess

Notes:

PARAOVARIAN CYST
=vestigial remnant of Wolffian duct in mesosalpinx*Frequency:*10% of all pelvic masses*Embryology:* Wolffian body (= mesonephros) consists of (a)mesonephric duct (= Wolffian duct)in female degenerates into vestigial structures of epithelial-lined cysts (= canals / duct of Gartner) Location:at lateral edge of uterus and vagina extending from broad ligament to vestibule of vagina(b)mesonephric tubulesin female degenerates into vestigial structures of 1.EPOÖPHORON (at lateral part of Fallopian tube)2.PAROÖPHORON: (at medial part of Fallopian tube)Location:between the tube and hilum of the ovary within the two peritoneal layers of broad ligament 1.**Gartner duct cyst**: inclusion cyst; lateral to vagina + uterine wall2.**Paroöphoron**: medial location between tube + hilum of ovary3.**Epoöphoron**: lateral location between tube + hilum of ovary4.**Hydatids of Morgagni** (= appendices vesiculosae): most lateral + outer end of Gartner duct√ ≥1 vesicle(s) attached to fringes of tube + filled with clear serous fluid √ thin-walled unilocular cyst, up to 18 cm in diameter√ may arise out of pelvis (if pedunculated + mobile)√ ± low-level internal echoes (from hemorrhage)*DDx:*functional cyst, cystic teratoma, benign epithelial neoplasm

Notes:

PELVIC INFLAMMATORY DISEASE
=acute clinical syndrome associated with ascending spread of microorganisms ("canalicular spread") from vagina / cervix to uterus, fallopian tubes, and adjacent pelvic structures, not related to surgery / pregnancy*Incidence:*10% of women in reproductive age (17% in Blacks); 1 million American women/year*Risk factors:*early age at sexual debut, multiple sexual partners, history of sexual transmitted disease, douching*Predisposed:*formerly married > married > never married; intrauterine contraceptive device (1.5-4-fold increase in risk)*Etiology:*(a)bilateral: venereal disease, IUD, S/P abortion(b)unilateral = nongynecologic: rupture of appendix, diverticulum, S/P pelvic surgery*Organisms:* (1)Chlamydia trachomatisChlamydia trachomatis + Neisseria gonorrhea (>50% with high prevalence of coinfection) damage protective barrier of endocervical canal with spread to tubes (30-50%) producing fibrosis + adhesions(2)Aerobes: Streptococcus, Escherichia coli, Haemophilus influenzae(3)Anaerobes: Bacteroides, Peptostreptococcus, Peptococcus(4)Mycobacterium tuberculosis (hematogenous)(5)Actinomycosis in IUD users(6)Herpesvirus hominis type 2, Mycoplasma*May be associated with:* **Fitz-Hugh-Curtis syndrome**
(= gonorrheic perihepatitis) ● usually bilateral lower abdominal pain (due to peritoneal irritation) ● abnormal vaginal discharge / uterine bleeding ● dysuria, dyspareunia, nausea, vomiting ● fever, leukocytosis, elevated ESR ● lower abdominal + adnexal + cervical motion tenderness 1.**Endometritis**
√ endometrial prominence√ small amount of fluid within uterine lumen√ gas reflection within uterine cavity (most specific)√ pain over uterus2.**Salpingitis**
not depicted by imaging techniques ● often beginning during / immediately after menstruation (due to less effective barrier of mucus at cervix)*Salpingitis isthmica nodosa* unknown etiology, commonly associated with pelvic inflammatory disease, infertility, ectopic pregnancy ● nodular thickening of isthmic portion of tube√ tubal irregularity + multiple diverticula / tubal obstruction on HSG3.**Hydro- / pyosalpinx**
=continued secretion of tubal epithelium into lumen of a fallopian tube obstructed at two sites*Cause:*infection, endometriosis, adhesions, microtubal surgeryLocation:ampullary / infundibular portion of tube√ undulating / folded tubular structure in extraovarian location filled with sterile fluid / debris / pus√ short linear echoes protruding into lumen (= tall ramified mucosal folds)√ longitudinal folds in ampullary portionHSG: √ absence of peritoneal spill*Cx:*tubal torsion*DDx:*dilated uterine / ovarian vein, developing follicle4.**Tubo-ovarian abscess**
*Cause:*sexually transmitted disease, IUD (20%), diverticulitis, appendicitis, pelvic surgery, gynecologic malignancy*Organism:*anaerobic bacteria become dominant
Location:usually in posterior cul-de-sac extending bilaterally√ multilocular complex mass often with debris, septations, irregular thick wall√ may contain fluid-fluid levels or gas *Dx:*clinically, laparoscope√Imaging employed only to differentiate between medical + surgical condition!*Cx:*1.Infertility due to tubal occlusion (25%): 8% after single episode, 20% after 2 episodes, 40% after ≥3 episodes of PID2.Ectopic pregnancy (6 x as frequent)3.Chronic pelvic pain (from pelvic adhesions)*DDx:*acute appendicitis, endometriosis, hematoma of corpus luteum, ectopic pregnancy, paraovarian cyst

Notes:

PENA-SHOKEIR PHENOTYPE
=autosomal recessive syndrome (45% sporadic, 55% familial) characterized by fetal akinesia*Cause:*decreased / absent fetal motion secondary to abnormalities of fetal muscle / nerves / connective tissue ("fetal akinesia deformation sequence")*Time of first detection:* 16-18 weeks MA @Spine:scoliosis, kyphosis, lordosis@Thorax: pulmonary hypoplasia, cardiac anomalies@Kidney:renal dysplasia@Limbs:limited movement, knee + hip ankylosis (arthrogryposis), abnormal shape + position, demineralization, camptodactyly, clubfeet√ craniofacial anomalies√ polyhydramnios√ IUGR√ short umbilical cord*Prognosis:*still birth*DDx:*multiple pterygium syndrome, Neu-Laxova syndrome, restrictive dermopathy, Larsen syndrome, trisomies 13 + 18

Notes:

PENTALOGY OF CANTRELL
=sporadic very rare abnormality*Cause:*failure of lateral body folds to fuse in the thoracic region with variable extension inferiorly1.Omphalocele + defect of lower sternum2.Ectopia cordis3.Deficiency of anterior diaphragm (herniation of intraabdominal organs into thoracic cavity is rare)4.Deficiency of diaphragmatic pericardium5.Cardiovascular malformation: atrioventricular septal defect (50%), VSD (18%), tetralogy of Fallot (11%)*Associated with:*trisomies exteriorization of heart*Prognosis:*death within a few days after birth

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Notes:

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PERITONEAL INCLUSION CYST
=PERITONEAL PSEUDOCYST = ENTRAPPED OVARIAN CYST*Cause:* from previous abdominal surgery (time delay of 6 months to 20 years) / trauma / pelvic inflammatory disease / endometriosis*Pathogenesis:*extensive pelvic adhesions result in impaired peritoneal clearing of fluid normally produced by an active ovary*Path:*cyst adherent to surface of ovary*Histo:*cyst lined by hyperplastic mesothelial cells + fibroglandular tissue with chronic inflammation*Dx:* single / multiloculated cyst contiguous with ovary*Cx:*infertility*Rx:*surgery (30-50% risk of recurrence)*DDx:*paraovarian cyst (ovoid cyst outside ovary), hydrosalpinx (visible folds, located outside ovary), ovarian neoplasm, lymphangioma

Notes:

PLACENTA ACCRETA
=underdeveloped decidualization with chorionic villi growing into myometrium*Incidence:*1:2,500-7,000 deliveries; in 5% of placenta previa patients*Risk of placenta accreta vs. cesarean section:* in 10% of placenta previa; in 24% of placenta previa + 1 cesarean section; in 48% of placenta previa + 2 cesarean sections; in 67% of placenta previa + 4 cesarean sections *Predisposed:*areas of uterine scarring with deficient decidua: previous dilatation + curettage, endometritis, submucous leiomyoma, Asherman syndrome, manual removal of placenta, adenomyosis, increasing parity*Associated with:* placenta previa (20%)*Types:* 1.PLACENTA ACCRETA = chorionic villi in direct contact with myometrium2.PLACENTA INCRETA = villi invade myometrium3.PLACENTA PERCRETA = villi penetrate through uterine serosaUS (78% sensitive, 94% specific): √ thinning to <1 mm / absence of hypoechoic myometrial zone between placenta + echodense uterine serosa / posterior bladder wall[retroplacental hypoechoic zone of decidua + myometrium + dilated periuterine venous channels measures 9.5 mm thick >18 weeks GA) √ thinning / irregularity / focal disruption of linear hyperechoic boundary echo (= uterine serosa-bladder wall interface)√ focal masslike elevations / extensions of echogenic placental tissue beyond uterine serosa√ >6 irregular intraplacental lacunae (= vascular spaces)*Cx:*(1)Retention of placental tissue(2)Life-threatening hemorrhage in 3rd stage of labor necessitating emergent hysterectomy(3)Persistent postpartum bleeding(4)Maternal death

Notes:

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PLACENTA EXTRACHORIALIS
= chorionic plate smaller than basal plate; ie, the transition of membranous to villous chorion occurs at a distance from the placental edge that is smaller than the basal plate radius A.CIRCUMMARGINATE PLACENTA*Incidence:*up to 20% of placentas ● No clinical significance ● placental margin not deformedB.CIRCUMVALLATE PLACENTA=attachment of fetal membranes form a folded thickened ring with underlying fibrin + often hemorrhage*Incidence:*1-2% of pregnancies*Cx:*premature labor, threatened abortion, increased perinatal mortality, marginal hemorrhage

Notes:

PLACENTAL ABRUPTION

=ABRUPTIO PLACENTAE=premature separation of placenta from the myometrium secondary to maternal hemorrhage into decidua basalis between 20th week and birth*Incidence:*0.5-1.3% of gestations*Risk factors:mnemonic:*"VASCULAR"**V**ascular disease + hypertension **A**bruption (previous history) **S**moking **C**ocaine **U**nknown (idiopathic) **L**eiomyoma **A**nomaly (fetal malformation) **R**eckless driving (trauma) *Associated with:* intraplacental infarction / hematoma ● vaginal bleeding (80%): bright red (acute), brownish-red (chronic) ● abdominal pain (50%) ● consumptive coagulopathy = DIC (30%) ● uterine rigidity (15%)Site: (a)marginal (most common site)low-pressure bleed due to tears of marginal veins; associated with cigarette smoking (b)retroplacentalhigh-pressure bleed due to rupture of spiral arteries; associated with hypertension + vascular disease √ hyperechoic / isoechoic hematoma (initially difficult to distinguish from placenta)√ hypoechoic / complex collection between uterine wall + placenta in 50% within 1 week (hematoma / placental infarction)√ anechoic collection within 2 weeks√ separation / rounding of placental margin √ abnormally thick + heterogenous placenta (if blood isoechoic)√ elevation of chorioamnionic membrane(DDx: incomplete chorioamnionic fusion during 2nd trimester, blighted twin) *Prognosis:* (1)Only large hematomas (occupying >30-40% of the maternal surface) result in fetal hypoxia(2)Abruptions with contained hematoma have worse prognosis(3)Responsible for up to 15-25% of all perinatal deaths(4)Normal term deliveries in 27% of hematomas detected >20 weeks GA(5)Normal delivery in 80% of intrauterine hematomas detected <20 weeks GA*Cx:*(1)Perinatal mortality (20-60%), up to 15-25% of all perinatal deaths(2)Fetal distress / demise (15-27%)(3)Premature labor + premature delivery (23-52%) (3-fold increase)(4)Threatened abortion during first 20 weeks(5)Infant small-for-gestational age (6-7%)*DDx:* (1)Normal draining basal veins(2)Normal uterine tissue(3)Retroplacental myoma(4)Focal contraction(5)Chorioangioma(6)Coexistent mole

Notes:

PLACENTAL HEMORRHAGE
Location:subchorionic, subamniotic, marginal, retroplacental

Preplacental Hemorrhage Retroplacental Hemorrhage

Notes:

Preplacental Hemorrhage =BREUS MOLE = SUBCHORIAL HEMORRHAGE=variant of placental abruption with progressive slow intracotyledonary bleeding*Incidence:* in 4% of all placental abruptions*Etiology:*massive pooling + stasis due to extensive venous obstruction*Time of onset:*18 weeks MA√ total loss of normal placental architecture√ gelatinous character of placenta elicited by fetal movement / abdominal jostling√ severe symmetric IUGR*Risk for fetal demise:* 67% overall; 100% for hematomas >60 mL

Notes:

Retroplacental Hemorrhage =accumulation of blood behind placenta, which may dissect into placenta / myometrium secondary to rupture of spiral arteries*Incidence:*4.5%; 16% of all placental abruptions ● external bleeding√ thickened heterogeneous appearing placenta (hematoma of similar echogenicity as placenta)√ rounded placental margins + intraplacental sonolucencies*Cx:*(1)Precipitous delivery(2)Coagulopathy(3)Fetal demise (accounts for 15-25% of all perinatal deaths); risk for fetal demise with hematomas >60 mL: 6% before 20 weeks GA; 29% after 20 weeks GA

Notes:

PLACENTA MEMBRANACEA
=presence of well-vascularized placental villi in the peripheral membranes*Cause:*? endometritis, endometrial hyperplasia, extensive vascularization of decidua capsularis, previous endometrial damage by curettage ● repeated vaginal bleeding extending into 2nd trimester + abortion at 20-30 weeks ● postpartum hemorrhage√ thickened outline over whole gestational sac (0.2-3.0 cm)√ may show additional distinct disk of placenta

Notes:

PLACENTA PREVIA

=abnormally low implantation of ovum with the placenta covering all / part of internal cervical os*Incidence:*0.5% of all deliveries; in 7-11% of women with 2nd + 3rd trimester vaginal bleeding; in 0.26% with unscarred uterus*Risk for placenta previa vs. cesarean section:* 0.65% after 1 section, 1.8% after 2 sections, 3% after 3 sections, 10% after 4 sections *Cause:*defective decidual vascularization in areas of endometrial scarring causing compensatory placental thinning; placenta occupies a greater surface of the uterus with increased probability for encroachment upon internal os*Predisposed:* (1)Previous uterine incision (cesarean section, myomectomy)(2)Older women(3)Multiparous women *Types on clinical examination:* 1.Central / total previa (1/3) = complete covering of internal os2.Partial previa = internal os partially covered by placenta3.Low-lying placenta = low placental edge without extension over internal os; palpable by examining finger painless vaginal bleeding in 93% (usually 3rd trimester / as early as 20 weeks)3-5% of all pregnancies are complicated by 3rd trimester bleeding; of these 7-11% are due to placenta previa! US - FALSE POSITIVES (5-7%): 1.Placental "migration" / rotation=differential growth rates between lower uterine segment + placenta63-93% will have normal implantation at term!-conversion to normal position: anterior wall > posterior wall of uterus-NO conversion if placenta attaches to both posterior + anterior walls2.Overfilled urinary bladderbladder-induced compression leads to apposition of the lower anterior + posterior uterine walls (cervical length >3.5-4 cm) simulating a placenta previa 3.Focal myometrial contraction (myometrial thickness>1.5 cm) in the region of the lower uterine segment *mnemonic:*"ABCD and F"**A**bruption (may mimic placenta previa) **B**ladder (must be empty) **C**ontraction (may have to wait 15-20 minutes) **D**ates (be wary in 1st half of pregnancy) **F**ibroid US - FALSE NEGATIVES (2%): 1.Obscuring fetal headremedied by Trendelenburg position / gentle upward traction on fetal head 2.Lateral position of placenta previa; remedied by obtaining oblique scans3.Blood in region of internal os mistaken for amniotic fluid *Cx:*(secondary to premature detachment of placenta from lower uterine segment)(1)Maternal hemorrhage (blood from intervillous space)(2)Premature delivery(3)IUGR(4)Perinatal death (5%)*Rx:*precludes vaginal delivery + pelvic examination

Notes:

PLACENTAL SITE TROPHOBLASTIC DISEASE
=very rare neoplasm (? type of choriocarcinoma)*Path:*microscopic tumor / diffuse nodular replacement of myometrium*Histo:*proliferation of predominantly intermediate trophoblasts but no syncytio- or cytotrophoblasts ● abnormal bleeding / amenorrhea ● low b-HCG levels (due to lack of syncytiotrophoblastic proliferation)√ cystic / solid lesions ± central component√ myometrium usually invaded*Prognosis:*benign / highly malignant course*Rx:*hysterectomy

Notes:

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POSTMATURITY SYNDROME

=inability of aging placenta to support demands of fetus *Incidence:*in 15% of all postterm gravidas ● meconium-stained amniotic fluid√ grade 3 placenta (in 85%), grade 2 (in 15%), grade 1 (in 0%)√ decreased subcutaneous fat + wrinkling of skin√ long fingernails√ decreased vernix*Cx:*meconium aspiration, perinatal asphyxia, thermal instability

Postterm Fetus

Notes:

Postterm Fetus =fetus undelivered by 42nd week MA*Incidence:*7- 12% of all pregnancies*Risk of perinatal mortality:* 2-fold at 43 weeks MA, 4- to 6-fold at 44 weeks MA

Notes:

PREECLAMPSIA

=TOXEMIA OF PREGNANCY*Incidence:*5% of pregnancies, typically during 3rd trimesterClinical triad: ● pregnancy-induced / -aggravated hypertension ● proteinuria ● peripheral edema + weight gain*Histo:*blunted invasion of vasa media of spiral arterioles + focal vasculitis + atheromatous degeneration + fibrin deposits in intima of maternal placental arterioles√ heavy calcium deposition (in areas of placental degeneration)√ IUGR (6% with late-onset preeclampsia, 18% with early-onset preeclampsia)*Cx:* @CNS@Liver: hematoma, infarction@Kidney ECLAMPSIA ● convulsions + coma

Notes:

PREMATURE RUPTURE OF MEMBRANES
=spontaneous rupture of chorioamnionic membranes before the onset of labor*Types:* (a)Preterm premature rupture of membranes (PPROM) <37 weeks GA(b)Term premature rupture of membranes (TPROM) >37 weeks GA*Incidence:*overall 2.1-17.1%; PPROM 0.9-4.4%; in 29% of all preterm deliveries; in 18% of all term deliveries*Risk of recurrence:*21% of women with PPROM*Cause:*? infection of membranes*Cx:* (a)TPROM:->24 hours may result in intrapartum fever->72 hours may result in chorioamnionitis + still-birth(b)PPROM: respiratory distress syndrome (9-43%), neonatal sepsis (2-19%)

Notes:

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PRIMARY OVARIAN CHORIOCARCINOMA

=NONGESTATIONAL CHORIOCARCINOMA*Incidence:*extremely rare; 50 cases in world literature*Age:*<20 years ● elevated serum HCG√ predominantly solid tumor with areas of hemorrhage + necrosis*DDx:*metastasis to ovary from gestational choriocarcinoma (reproductive age)

Notes:

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SECKEL SYNDROME
=BIRD-HEADED DWARFISM=rare autosomal recessive disorder (44 cases) ● proportionate postnatal short stature ● characteristic stance: slight flexion of hips and knees ● mental retardation ● simian crease ● cryptorchidism@Skull√ severe microcephaly√ receding forehead, large beaked nose, micrognathia@Skeleton√ dislocation of radial head + hypoplasia of proximal end of radius√ absence of phalangeal epiphysis√ clinodactyly of 5th digit√ gap between 1st and 2nd toe√ hip dislocation√ hypoplasia of proximal fibula√ 11 pairs of ribsOB-US: √ severe IUGR√ oligohydramnios√ decreased bone length (femur, tibia, fibula)√ decreased AC, HC

Notes:

SEROUS OVARIAN TUMOR

*Incidence:*30% of ovarian tumors*Histo:*lined by tall columnar epithelial cells (like fallopian tubes), filled with serous fluid, psammoma bodies (= microscopic calcifications) in 30%;60% benign, 15% borderline, 25% malignant *Age:*20-50 years (malignant variety later)1.**Serous cystadenoma**

second most common benign tumor of the ovary (after dermoid cyst); 20% of all benign ovarian neoplasms √ uni- / multilocular thin-walled cyst up to 20 cm in diameter √ only small amount of solid tissue: occasional septum / mural nodule√ bilateral in 7-20-30%2.**Serous cystadenocarcinoma**

=60-80% of all ovarian carcinomas√ cyst with large amount of solid tissue: papillomatous excrescences within cyst (= papillary serous carcinoma)√ may have calcifications√ bilateral in 50-70%√ loss of capsular definition + tumor fixation√ ascites secondary to peritoneal surface implantation√ lymph node enlargement (periaortic, mediastinal, supraclavicular)

Notes:

SERTOLI-LEYDIG CELL TUMOR OF OVARY

*Origin:*from hilar cells of ovary*Incidence:*<0.5%*Age:* any age; most common in 2nd-3rd decade ● androgenic√ hypoechoic mass simulating fibroid√ may have cystic / hemorrhagic degeneration

Notes:

SINGLE UMBILICAL ARTERY

Etiology:

(1) Primary agenesis of one umbilical artery (usually first appears in 5th menstrual week)(2)Secondary atrophy / atresia of one umbilical artery(3) Persistence of original single allantoic artery of the body stalk*Incidence:*0.2-1% of singleton births; 5% in dizygotic twins; 2.5% in abortuses; increased incidence in trisomy D / E, diabetic mothers, White patients, spontaneous abortions *Associated with:* (a)Congenital anomalies (21%):1.CHD (most frequent): VSD, conotruncal anomalies2.Abdomen: ventral wall defect, diaphragmatic hernia3.CNS: hydrocephalus, holoprosencephaly, spina bifida4.GU: hydronephrosis, dysplastic kidney5.Esophageal atresia, cystic hygroma, cleft lip6.Polydactyly, syndactyly(b)IUGR(c)Premature delivery(d)Perinatal mortality (20%): stillbirth (66%)(e)Marginal (18%) / velamentous (9%) insertion of umbilical cord(f)Chromosomal anomalies (67%): trisomy 18 > trisomy 13 > Turner syndrome > triploidy Site:left artery slightly more often absent than right√ axial view of cord shows 2 vessels√ single umbilical artery nearly as large as umbilical vein (umbilical vein-to-umbilical artery ratio < 2)√ incurvation of distal aorta toward common iliac artery on the side of patent umbilical artery√ ipsilateral hypoplastic common iliac artery√ absence of abdominal portion of umbilical artery on ipsilateral side of missing umbilical artery√ color flow imaging permits earlier (15-16 weeks) + more confident diagnosis*Prognosis:* (1)4-fold increase in perinatal mortality (14%) with concurrent major abnormality(2)Isolated single umbilical artery does not affect clinical outcome*DDx:* (1)normal variant = two arteries at fetal end may fuse near placental end into single umbilical artery (umbilical arteries normally unite with allantoic artery near placental insertion)(2)arterial convergence of 2 into 1 umbilical a.

Notes:

STEIN-LEVENTHAL SYNDROME
=POLYCYSTIC OVARY SYNDROME*Incidence:*2.5% of all women*Etiology:*deficient aromatase activity (catalyst for conversion of androgen into estrogen) results in androgen excess; exaggerated pulsatile release of LH stimulates continued ovarian androgen secretion at the expense of estradiol; reduction of local estrogen impairs FSH activity; this results in accumulation of small- + medium-sized atretic follicles without final maturation into graafian follicles*Path:*pearly white ovaries with multiple cysts below the capsule, which are lined by a hyperplastic theca interna layer showing pronounced luteinization; granulosa cells are absent / degenerating; corpora lutea are absent *Age:*late 2nd decade*Associated with:*Cushing syndrome, basophilic pituitary adenoma, postpill amenorrhea, virilizing ovarian / adrenal tumor ● reduced infertility / sterility ● mild facial / severe generalized hirsutism ● obesity ● secondary amenorrhea (most common cause) ● menstrual irregularities / oligomenorrhea ● cystic acne ● cephalic hair loss ● periodic abdominal discomfort ● elevated LH levels without LH surge + normal / decreased FSH = increased LH/FSH ratio ● elevated androstenedione / testosterone levels ● elevated estrone / estradiol√ bilaterally enlarged ovaries >15 cm^3 (70%)√ normal ovarian size (in 30%),polycystic ovaries have a volume of 6-30 cm^3√ excessive number of developing follicles√ multiple (more than 5) small cysts of 5-8 mm in subcapsular location (40%)√ hypoechoic ovaries (25%)√ isoechoic ovaries (5%)*Cx:*endometrial cancer <40 years of age (due to unopposed chronic estrogen stimulation)*DDx:*ovaries in congenital adrenal hyperplasia, normal ovaries*Rx:*(1)Ovulation induction with clomiphene (Clomid) / menotropins (Perganol)(2)Wedge resection (transient effect only)

Notes:

STUCK TWIN

=one twin with IUGR residing within an oligo- / anhydramniotic sac of a diamniotic twin pregnancy√ amnion invisible secondary to close contact with fetal parts√ fetus fixed relative to the uterine wall without change during shift in maternal position√ diminished / absent active fetal motion√ absence of intermingling of fetal parts between twins*Prognosis:*fetal death in utero **SUCCENTURIATE LOBE OF PLACENTA** =ACCESSORY LOBE = separate mass of chorionic villi connected to main placenta by vessels within membrane*Cause:*placental villi atrophy in area of inadequate blood supply + proliferate in two opposite directions (trophotropism) with fetal vessels remaining at the site of villous atrophy*Incidence:*0.14-3%*Cx:*(1)Retained in utero with postpartum hemorrhage(2)Placenta previa with intrapartum hemorrhage(3)Vasa previa = succenturiate vessels traversing internal os, which may rupture resulting in fetal blood loss

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Notes:

SUBCHORIONIC HEMORRHAGE
=separation of chorionic membrane from decidua with accumulation of blood in subchorionic space (placental membranes are more easily stripped from myometrium than from placenta)*Incidence:*81% of all placental abruptions; in 91% before 20 weeks MA ● may lead to vaginal hemorrhage after dissection through decidua (18% of all causes of 1st-trimester bleeding)√ detached placental margin from adjacent myometrium (60%)√ hematoma contiguous with placental margin (100%)√ predominant hemorrhage often separate from placenta, even on opposite side of placenta*Prognosis:*worsens with (1) increased maternal age (2) earlier gestational age (3) size of hematoma; 9% overall miscarriage rate; risk of fetal demise doubles once hematoma reaches 2/3 of circumference of chorion

Notes:

TERATOMA OF NECK

=germ cell tumor of neck (oropharynx, tongue)↓ polyhydramnios in 30% (from esophageal obstruction)↓ complex mass in cervical region*Cx:*airway obstruction*DDx:*cystic hygroma, goiter, branchial cleft cyst, cervical meningocele, neuroblastoma of neck, hemangioma of neck

Notes:

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TERATOMA OF OVARY

=immature derivatives of all 3 germ cell layers*Incidence:* rare*Age:*childhood / adolescence√ cystic / complex mass (most frequently)√ usually large solid mass with internal echoes

Notes:

THECA CELL TUMOR OF OVARY

=THECOMA*Incidence:*1-2% of all ovarian neoplasms*Age:*>30 years (30%), postmenopausal (70%) ● estrogenic√ hypoechoic mass with sound attenuation√ unilateral

Notes:

THECA LUTEIN CYST
=form of ovarian hyperstimulation ● associated with abnormally high levels of b-HCG secondary to(a)multiple gestations (b)gestational trophoblastic disease (in 40%)(c)fetal hydrops(d)pharmacologic stimulation with b-HCG(e)normal pregnancy (uncommon)√ multiloculated cysts, often bilateral√ ovaries several cm in size√ involution within a few months after source of gonadotropin removed

Notes:

TORSION OF OVARY
=result of rotation of ovary on its axis producing arterial, venous, and lymphatic stasis *Age:*usually affects prepubertal girls, may occur prenatally, increased risk during pregnancy*Cause:* (1)Enlarged ovary (large cyst / tumor, paraovarian cyst)(2)Hypermobility of adnexa (more frequent in younger children + during pregnancy), excessively long mesosalpinx, tubal spasm ● severe lower abdominal pain, nausea, vomiting, fever ● palpable mass in 50%Location:R:L = 3:1US: √ markedly enlarged hypo- / hyperechoic midline mass√ multiple peripheral cysts (= transudation of fluid into follicles) measuring 8-12 mm in diameter (64-74%)√ good sound transmission (vascular engorgement + stromal edema)√ free fluid in cul-de-sac (32%)√ absence of Doppler waveforms (not always reliable)√ ± complex mass (if secondary to cyst / tumor)CT + MR: √ deviation of uterus to side of torsion√ engorgement of blood vessels on side of torsion√ small amount of ascites√ obliteration of fat planes around torsed ovary√ lack of enhancement*Prognosis:*spontaneous detorsion is common (history of prior similar episodes)

Notes:

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TRIPLOIDY

=69 chromosomes*Incidence:*1% of conceptions; 0.04% of 20-week fetusesNO obvious pattern!√ early severe asymmetric IUGR (MOST PROMINENT FEATURE); cephalocorporal disproportion√ oligohydramnios√ large hydropic placenta with scattered vesicular spaces (partial hydatidiform mole)√ congenital heart disease: ASD, VSD√ brain anomalies: hydrocephalus, holoprosencephaly, neural tube defect√ cleft lip / palate√ syndactyly of fingers√ omphalocele√ renal abnormalities*Prognosis:*most ending in spontaneous abortion

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Notes:

TRISOMY 13
=PATAU SYNDROME*Incidence:*1:5,000 births@OB:severe IUGR, hydramnios@CNS:alobar holoprosencephaly, posterior encephalocele, neural tube defect@Face:midline labial cleft, proboscis, hypotelorism, cyclopia, anophthalmia@Skeleton:postaxial polydactyly, rocker bottom foot@Heart:(CHD in 90%) VSD, echogenic chordae tendineae, hypoplastic ventricle, tetralogy of Fallot, transposition@Kidney:polycystic kidney, horseshoe kidney@GI:omphalocele (occasionally)*Prognosis:*few infants live more than a few days / hours*DDx:*Meckel-Gruber syndrome

Notes:

TRISOMY 18

=EDWARD SYNDROME*Incidence:*3:10,000 births ● triple-marker screening test: ● decreased maternal alpha-fetoprotein ● decreased HCG (DDx: increased in own syndrome) ● decreased estriol@OB:severe symmetric IUGR (28% <24 weeks MA), single umbilical artery (30%), polyhydramnios (occasionally)@Face:micrognathia, hypotelorism, facial cleft (10-40%)@Head:strawberry-shaped head (50%), cystic hygroma@CNS:holoprosencephaly, choroid plexus cyst (30-75%), small cerebellum with prominent cisterna magna, myelomeningocele@Hand:clenched hand with overlapping of index finger (>60%, HIGHLY CHARACTERISTIC)@Arm:shortened radial ray, clubbed forearm@Foot:clubbed foot, rocker-bottom foot@Heart:(CHD in 90%) VSD, complete AV canal, DORV@GI:diaphragmatic hernia, omphalocele (30-40%), TE fistula@Kidney:polycystic kidney, horseshoe kidney, UPJ obstruction*Prognosis:*usually delivered by emergency cesarean section due to IUGR + fetal distress, if not detected prenatally

Notes:

TWIN EMBOLIZATION SYNDROME

=rare complication of monochorionic pregnancy following the death of one twin whose blood pressure falls to zero*Pathophysiology:* 1.Acute reversal of transfusion to co-twin at time of intrauterine demise of one twin with ischemic changes in survivor2.Embolization of thromboplastin-enriched blood / detritus from the dead to the living twin through vascular anastomoses in placenta*Embolized organs:*CNS (72%), GI tract (19%), kidneys (15%), lungs ventriculomegaly, cortical atrophy, porencephalic cyst, cystic encephalomalacia within 2 weeks of death of co-twin

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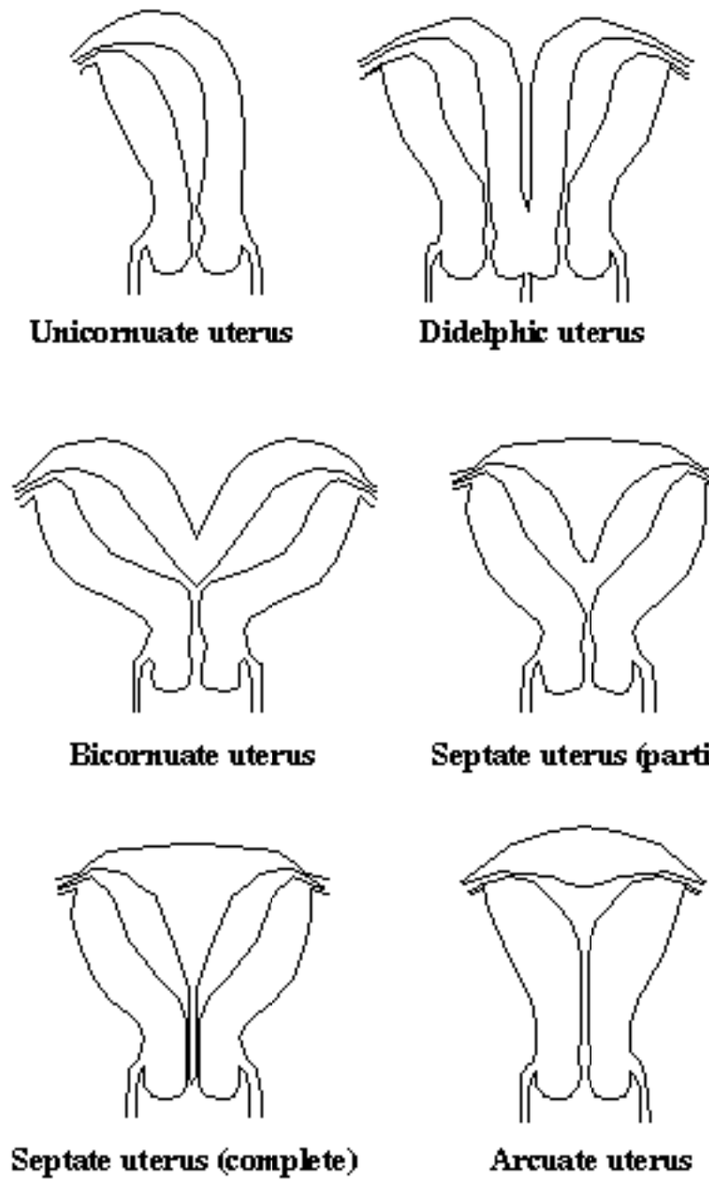
Notes:

TWIN-TWIN TRANSFUSION SYNDROME
=FETO-FETAL TRANSFUSION SYNDROME=MONOVULAR TWIN TRANSFUSION= INTRAUTERINE PARABIOTIC SYNDROME = complication of monozygotic twinning with one placenta or one fused placenta of mono- / dizygotic twins *Incidence:*5-18% of twin pregnancies; 5-15% of monozygotic multiple pregnancies; 15-30% of monochorionic twin gestations*Cause:*unbalanced intrauterine shunting of blood through shared placental vessels*Time of onset:*2nd trimester with discordant amniotic fluid volumes*Path:*large communication between arterial circulation of one twin and venous circulation of the other twin through *arteriovenous* shunt (= common villous district) deep within placenta√ discrepant amniotic fluid volume (75%)√ discordant BPD by >5 mm (57%)√ discordant estimated fetal weight >25% (67-100%) A.DONOR TWIN=twin that transfuses the recipient twin + remains itself underperfused ● anemia + hypovolemia ● high output cardiac failure + hydrops (rare)√ oligohydramnios (75-80%) / "stuck twin" = severe oligohydramnios (60%) from oliguria√ intrauterine growth restriction (common) diagnosed by discordant EFW of >25%√ morphologically normalB.RECIPIENT TWIN ● polycythemia (higher hemoglobin) ● plethora = hypervolemia (volume overload)√ polyhydramnios (70-75%) from increased fetal urination√ fetal hydrops (10-25%): pericardial + pleural effusions, ascites, skin thickening√ organomegaly√ fetus papyraceus = macerated dead fetus√ velamentous cord insertion (64%) *Prognosis:*80-100% perinatal mortality if presenting <28 weeks MA and left untreated*Cx:*amniorrhexis, preterm labor*Rx:*elective termination, volume-reduction amniocentesis of polyhydramniotic sac (decreasing mortality rates to 34%), selective feticide, laser ablation of vascular anastomoses*DDx:*IUGR of one dizygotic twin (two separate placentas, two different sexes)

Notes:

UTERINE ANOMALIES
=anomalies of fusion of paramesonephric duct(= müllerian duct) completed by 18th week of fetal life *Incidence:*0.1-3%◊Uterine anomalies are found in 9% of women with infertility / repeated spontaneous abortions!◊25% of women with uterine abnormalities have fertility problems!*Associated with:*urinary tract anomalies in 20-50%;



possibly increased familial occurrence of limb reduction

Classification: (classes in parenthesis refer to the classification of the American Fertility Society) A.ARRESTED MÜLLERIAN DUCT DEVELOPMENT1.bilateral: **Uterine agenesis / hypoplasia** (class I)*Incidence:*1:5,000*Often associated with:*vaginal agenesis / hypoplasia*Age of detection:*menarche√ small uterus with small endometrial canal√ poor zonal differentiation + abnormal T2-hypointense myometrium2.unilateral: **Unicornuate uterus** = Uterus unicornis unicollis (class II)(a)with contralateral rudimentary horn(b)without rudimentary horn*Incidence:*3-6-13% of uterine anomalies*May be associated with:*ipsilateral renal agenesis ● infertility in 5-20% ● ? pregnancy wastage √ reduced uterine volume√ asymmetric ellipsoidal uterine configuration√ rudimentary horn may contain endometrium + may communicate with main uterine cavity√ solitary fusiform "banana-shaped" uterine cavity with lateral deviation within pelvis terminating in a single fallopian tube on HSG*Cx:*cryptomenorrhea within endometrium-containing rudimentary horn that does not communicate with endometrium cavityB.TOTAL / PARTIAL FAILURE OF MÜLLERIAN DUCT FUSION(75% of uterine anomalies) 1.**Uterus didelphys** (class III)=complete duplication with 2 vaginas + 2 cervices + 2 uterine horns*May be associated with:*renal agenesis ● usually asymptomatic√ two widely spaced uterine corpora, each with a single fallopian tube√ separate divergent uterine horns√ large fundal cleft√ cervical duplication√ horizontal septum of upper vagina (ipsilateral to renal agenesis)√ opacification of single deviated horn on HSG *Cx:*unilateral hydro- / hematocolpos (if transverse vaginal septum present) with reflux endometriosis*Rx:*surgery is rarely performed2.**Bicornuate uterus** = uterus bicornis (class IV)=lack of fusion of corpus(a)bicornis bicollis = complete with division down to internal os(b)bicornis unicollis = partial√ concave / heart-shaped external fundal contour due to a large fundal cleft >1-2 cm deep√ separation of uterine horns√ intercornual angle of >75-105° (demonstrated on luteal-phase US in conjunction with HSG)√ intercornual distance (= distance between maximum lateral extent of hyperintense endometrium on transaxial image) >4 cm√ divider between cornua comprised of myometrium / fibrous tissue / both√ fusiform shape of each uterine horn with lateral convex margins√ discrepancy in size of the 2 uterine horns√ elongation + widening of cervical canal + isthmusLaparoscopy:typical external fundal indentation*Cx:*repeated spontaneous abortions (frequently in 2nd-3rd trimester), premature rupture of membranes, premature labor, persistent, SGA infant, malpresentations (transverse lie)*Rx:*transabdominal surgery to fuse uterine horns (abdominal metroplasty)C.NONRESORPTION OF SAGITTAL UTERINE SEPTUM1.**Septate uterus** (class V)Most common anomaly (almost 50%) associated with reproductive failure in 67% *Path:*septum may be composed of fibrous tissue (low-signal intensity), myometrium (intermediate-signal intensity), or both√ convex / flat / minimally indented (≤1 cm) external fundal contour√ distal portion of septum hypoechoic to myometrium (= fibrous tissue)√ acute angle of <75° between uterine cavities√ duplication of uterine horns on HSG (DDx to bicornuate uterus unreliable) √ endometrial canals completely separated by tissue isoechoic to myometrium extending into endocervical canal*Types:* (a)Uterus septus=complete septum extending to internal os(b)Uterus subseptus=partial septum involving endometrial canal*Cx:*90% abortion rate (poor septal vascularity)*Rx:*hysteroscopic metroplasty (= excision of septum) 2.**Uterus arcuatus** (class VI)Most common anomaly unassociated with reproductive failure √ NO division of uterine horns√ normal fundal contour√ smooth indentation of fundal endometrial canal√ increased transverse diameter of uterine cavity√ single uterine canal with saddle-shaped fundus on HSGD.INADEQUATE HORMONAL STIMULATION DURING FETAL DEVELOPMENT= DES (= diethylstilbestrol) -related abnormalities (class VII) ● synthetic hormone used in 1950s + 1960s to prevent miscarriage ● may cause abnormal uterine morphology (with decreased fertility) ● increased risk of vaginal malignancy1.**Uterine hypoplasia**
associated with diethylstilbestrol (DES) exposure in utero √ mean uterine volume = 50 $cm^3$2.**T-shaped uterus**
encountered in 15% of women exposed to DES (diethylstilbestrol) in utero √ low uterine volume√ uterine fundus thinner than cervix √ greater width than depth of corpus + fundus over cervix√ T-shaped lumen on hysterosalpingogram

Notes:

UTERINE LEIOMYOMA

=FIBROID = benign overgrowth of smooth muscle + connective tissue; commonest cause for uterine enlargement after pregnancy*Histo:*monoclonal proliferation of smooth muscle cells (NOT myometrial hyperplasia)*Hormonal dependency:* 1.Growth during pregnancy in 15-32% by a mean volume of 12 ± 6% within the 1st trimester (NOT during remainder of pregnancy)◊The larger the myoma, the greater the likelihood of growth!2.Shrinkage in puerperium + after menopause *Incidence:*in 20-25-50% of women > age of 30 years; black:white women = 3:1 - 9:1*Age:*usually >30 years ● asymptomatic in 70-75% ● palpable mass ● pelvic pressure / pain (torsion, infarction, necrosis) ● hypermenorrhea (= heavy prolonged periods)Location:mostly in fundus + corpus; in 3% in cervix1.Intramural (within confines of uterine outline) in 95%2.Subserosal = exophytic(a)parasitic fibroid = subserosal fibroid, which has become detached secondary to circulatory occlusion of vessels in pedicle; revitalized through omental / mesenteric blood supply(b)intraligamentous fibroid (eg, within broad ligament)3.Submucosal(a)fibroid polyp = partial / complete extrusion of pedunculated submucosal fibroid through cervical canal √ uterine enlargement√ lobulated / nodular distortion of uterine outline (subserosal leiomyoma) + indentation of urinary bladder√ distortion / obliteration of the contour of the uterine cavity (submucosal leiomyoma)√ intramural soft-tissue mass (most frequent), usually multiple, solitary in 2%√ speckled / ringlike / popcorn calcificationUS (60% sensitivity, 99% specificity, 87% accuracy): √ hypoechoic solid concentric mass (<33%) (= muscle component prevails)√ echogenic attenuating mass (= dense fibrosis prevails)√ sharp discrete refractory shadows (from borders between fibrous tissue and smooth muscle, margins of leiomyoma with normal myometrium, edges of whorls, bundles of smooth muscle)√ anechoic features (secondary to internal degeneration: atrophic, hyaline, cystic, myxomatous, lipomatous, calcareous, carneous, necrobiotic, hemorrhagic, proteolytic degeneration)CT: √ hypo- / iso- / hyperdense mass containing mixed hyperechoic areasMR (86-92% sensitivity, 100% specificity, 97% accuracy; desirable for planning myomectomy): √ sharply marginated homogeneous focal area of low / intermediate signal intensity on T1WI + T2WI√ occasionally inhomogeneous high signal intensity on T2WI (from hemorrhage / hyaline degeneration or in highly cellular leiomyoma or leiomyoma with edema)√ hyperintense rim in 33% (dilated lymphatics / veins / edema)√ enhancement pattern (usually later than myometrium): 65% hypointense, 23% isointense, 12% hyperintense to myometriumHysterosalpingography (9% sensitivity, 97% specificity, 76% accuracy) *Cx:* (1)Infertility in 35%(a) narrowing of isthmic portion of tube (b) impingement on endometrium interfering with implantation; infertility rates highest for submucosal leiomyomas (2)Complications in pregnancysignificantly increased for myomas >200 cm^3 (a)Increased frequency of spontaneous abortions(b)Increased frequency of IUGR(c)Preterm labor in 7% + premature rupture of membranes(d)Uterine dyskinesia, uterine inertia during labor(e)Dystocia, obstruction of birth canal during vaginal delivery (if near internal os)(f)Postpartum hemorrhage(3)Hydroureteronephrosis(4)Malignant transformation (in 0.2%)*Rx:*surgery for: pain, menorrhagia, visceral compression◊Submucosal leiomyomas may be treated with hysteroscopic myomectomy*DDx of necrotic leiomyoma:* (1)Ovarian mass (ovarian cyst, hemorrhagic cyst, endometrioma, cystic dermoid, cystadenoma, malignancy)(2)Ectopic interstitial pregnancy(3)Intrauterine gestational sac(4)Intrauterine fluid collection(5)Hydatidiform mole(6)Myometrial contraction (lasts for 15-30 minutes)(7)Cervical tumor(8)Hematoma of broad ligament*DDx of pedunculated subserosal leiomyoma:* ovary: use transvaginal US / MR to identify follicles!

Notes:

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UTERINE RUPTURE IN PREGNANCY
=disruption of all layers surrounding the fetus (membranes, decidua, myometrium, serosa)*Prevalence:*3-5% for classic cesarean sections; 1-2% for lower segment operations*Classification:* 1.Spontaneous rupture during labor2.Traumatic rupture during delivery3.Rupture due to myometrial scars / disease*Predisposed:* previous uterine surgery, previously excessively long / difficult labor Location:(a)corpus with rupture before onset of labor(b)lower uterine segment during labor, L > R*Cx:*hypofibrinogenemia (triggered by excessive blood loss, trauma, amniotic fluid embolism)*Mortality:*2-20% maternal mortality; 10-25% fetal mortality*DDx:*Uterine dehiscence = rupture of only myometrium

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Notes:

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UTERINE TRAUMA DURING PREGNANCY

*Incidence:*6-7% (70% due to motor vehicle accident)1.Placental abruption2.Fetal injury (eg, cerebral injury)3.Fetal death

Notes:

VAGINAL AGENESIS

2nd most common cause of primary amenorrhea *Incidence:*1:4,000-5,000 women ● cyclic abdominal pain*May be associated with:* (1)Uterine + partial tubal agenesis (90%)(2)Unilateral renal agenesis / ectopia (34%)(3)Skeletal malformations (12%)(4)McKusick-Kaufman syndrome (hydrometrocolpos + polydactyly + heart defects)(5)Ellis-van Creveld syndrome

---

Notes:

VASA PREVIA
=rare type of velamentous cord insertion in which umbilical vessels cross the internal os(a)vessels connecting separate succenturiate lobe to main portion of placenta(b)cord vessels of velamentous (membranous) cord insertion from low-lying placenta(c)aberrant chorionic vessels in association with marginal cord insertion from low lying placenta*Cx:*(1)Bleeding from torn fetal vessels(2)Cord compression by presenting part during labor(3)Cord prolapse*Risk:*50-100% fetal mortality

Notes:

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VELAMENTOUS CORD INSERTION
=umbilical cord insertion into membranes before entering placenta = attachment of cord to chorion laeve*Incidence:*0.09 to 1.8%*Associated with:* (a)multiple gestation, uterine anomaly, IUD(b)congenital anomalies (in 5.9-8.5%): asymmetric head shape, spina bifida, esophageal atresia, obstructive uropathy, VSD, cleft palate*Cx:*(1)IUGR(2)Preterm labor*Risk:*(1)Cord compression(2)Rupture of cord with traction during delivery

Notes:

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Table of dose, energy, half-life, radiation dose

TABLE OF DOSE, ENERGY, HALF-LIFE, RADIATION DOSE

PEDIATRIC DOSE

RADIATION DOSE

Quality control

QUALITY CONTROL

RADIOPHARMACEUTICALS

- Radionuclide Impurity
- Radiochemical Impurity
- Chemical Impurity
- Pyrogen Testing

CALIBRATORS

- Constancy = Precision
- Linearity
- Accuracy
- Geometry

SCINTILLATION CAMERA

- Spatial Resolution / Linearity

SOURCES OF ARTIFACTS

SPECT QUALITY CONTROL

- Uniformity
- Center Of Rotation (COR)
- Sources Of Artifacts

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Positron emission tomography

POSITRON EMISSION TOMOGRAPHY

PET imaging in oncology

Immunoscintigraphy

IMMUNOSCINTIGRAPHY

Gallium scintigraphy

GALLIUM-67 CITRATE

- Binding Sites
- Uptake
- Excretion
- Time Of Imaging
- Normal Variants Of Ga-67 Uptake
- Indications
- Gallium In Bone Imaging
- Gallium In Tumor Imaging
- Gallium In Lung Imaging
- Gallium In Renal Imaging
- Gallium Imaging In Lymphoma
- Gallium Imaging In Malignant Melanoma

AGENTS FOR INFLAMMATION

Bone scintigraphy

BONE AGENTS

BONE MARROW AGENTS

- Pediatric Indications For Bone Scan
- Superscan
- Hot Bone Lesions
- Photon-deficient Bone Lesion
- Benign Bone Lesions
- Soft-tissue Uptake
- Incidental Urinary Tract Abnormalities

Brain scintigraphy

RADIONUCLIDE ANGIOGRAPHY

- Ceretec Brain Imaging
- I-123 Spectamine Brain Imaging

POSITRON EMISSION TOMOGRAPHY

RADIONUCLIDE CISTERNOGRAPHY

- CSF Leak Study
- Hydrocephalus

Thyroid and parathyroid scintigraphy

THYROID SCINTIGRAPHY

- Tc-99m Pertechnetate
- Iodine-123
- Iodine-131
- Iodine Fluorescence Imaging
- Thyroid Uptake Measurements

PARATHYROID SCINTIGRAPHY

- Technetium-thallium Subtraction Imaging
- Technetium-99m Sestamibi

Lung scintigraphy

PERFUSION AGENTS

- Tc-99m Macroaggregated Albumin (MAA)
- Tc-99m Human Albumin Microspheres

VENTILATION AGENTS

- Xenon-133
- Xenon-127
- Krypton-81m
- Tc-99m DTPA Aerosol
- Carbon Dioxide Tracer

TUMOR IMAGING

- Positron Emission Tomography

QUANTITATIVE LUNG PERFUSION IMAGING

- Unilateral Lung Perfusion
- Perfusion Defects

PULMONARY THROMBOEMBOLISM

Heart scintigraphy

CARDIAC IMAGING CHOICES

LEFT VENTRICULAR ANATOMY AND PROJECTIONS

EJECTION FRACTION

BLOOD POOL AGENTS

- Tc-99m DTPA / Tc-99m Sulphur Colloid
- Tc-99m-labeled RBCs
- Tc-99m HSA

MYOCARDIAL PERFUSION IMAGING AGENTS

- Potassium-43
- Thallium-201 Chloride
- Tc-99m MIBI (Sestamibi)
- Tc-99m Teboroxime
- Tc-99m Tetrofosmin
- Positron Emission Tomography

STRESS TEST

- Physical Stress Test
- Pharmacological Stress Test

VENTRICULAR FUNCTION

- First-pass Ventriculography
- Equilibrium Images
- Gated Blood Pool Imaging

INFARCT-AVID IMAGING

- Tc-99m Pyrophosphate
- Tc-99m Antimyosin Fab Fragments

NONAVID INFARCT IMAGING

MYOCARDIAL ISCHEMIA

INTRACARDIAC SHUNTS

Liver and gastrointestinal tract scintigraphy

Renal and adrenal scintigraphy

RENAL AGENTS

- Tc-99m DTPA
- [Tc-99m Glucoheptonate]
- Tc-99m DMSA
- [I-131 OIH]
- Tc-99m Mercaptoacetyltriglycine (MAG3)
- Enalaprilat-enhanced Renography
- Cold Defect On Renal Scan

DIFFERENTIAL RENAL FUNCTION

RADIONUCLIDE CYSTOGRAM

ADRENAL SCINTIGRAPHY

- I-131 Metaiodobenzylguanidine (MIBG)
- I-123 Metaiodobenzylguanidine
- Iodocholesterol

Statistics

STATISTICS

- Sensitivity
- Specificity
- Accuracy
- Positive Predictive Value
- Negative Predictive Value
- False-positive Ratio
- False-negative Ratio
- Disease Prevalence

BAYESS THEOREM

RECEIVER OPERATING CHARACTERISTICS (ROC)

KAPPA (&K;)

CONFIDENCE LIMIT

CLINICAL EPIDEMIOLOGY

- Screening Techniques
- Self-selection
- Randomized Trials
- Case-control Studies

Calculation of odds ratio = ad / bc :

Water-soluble contrast media

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BONE SCLEROSIS

Diffuse Osteosclerosis Constitutional Sclerosing Bone Disease Solitary Osteosclerotic Lesion Multiple Osteosclerotic Lesions Dense Metaphyseal Bands Bone-within-bone Appearance

Notes:

INTRAOSSEOUS LESION

Bubbly Bone Lesion Nonexpansile Unilocular Well-demarcated Bone Defect Nonexpansile Multilocular Well-demarcated Bone Defect Expansile Unilocular Well-demarcated Osteolysis Poorly Demarcated Osteolytic Lesion Without Periosteal Reaction Poorly Demarcated Osteolytic Lesion With Periosteal Reaction Mixed Sclerotic And Lytic Lesion Trabeculated Bone Lesion Lytic Bone Lesion Surrounded By Marked Sclerosis mnemonic:"BOOST" Multiple Lytic Lesions Lytic Bone Lesion In Patient <30 Years Of Age Lytic Bone Lesion On Both Sides Of Joint

Notes:

Syringomyelia =ACQUIRED / SECONDARY SYRINGOHYDROMYELIA=any cavity within substance of spinal cord which may communicate with the central canal, usually extending over several vertebral segments*Histo:*not lined by ependymal tissue*Pathophysiology:*interrupted flow of CSF through the perivascular spaces of cord between subarachnoid space + central canal*Cause:* 1.Posttraumatic syringomyelia*Incidence:*in 3.2% after spinal cord injuryLocation:68% in thoracic cord 0.5-40 cm (average 6 cm) in length syrinx may be septated (parallel areas of cavitation) on transverse T1WI loss of sharp cord-CSF interface (obliteration of arachnoid space by adhesions) in 44% associated with arachnoid loculations (extramedullary arachnoid cysts) at upper aspect of syrinx2.Postinflammatory syringomyeliasubarachnoid hemorrhage, arachnoid adhesions, S/P surgery, infection (tuberculosis, syphilis) 3.Tumor-associated syringomyeliaspinal cord tumors, herniated disk; secondary to circulatory disturbance + thoracic spinal cord atrophy 4.Vascular insufficiency

Notes:

BONE OVERGROWTH

Bone Overdevelopment Erlenmeyer Flask Deformity

Notes:

JOINTS

Notes:

EPIPHYSIS

Epiphyseal / Apophyseal lesion Stippled Epiphyses Epiphyseal Overgrowth Ring Epiphysis Epiphyseolysis

Notes:

TRAUMA

Childhood Fractures Pseudarthrosis In Long Bones Exuberant callusExuberant Callus Formation

Notes:

RIBS

Rib Lesions Rib Notching On Inferior Margin Rib Notching On Superior Margin Ribbon Ribs Bulbous Enlargement Of Costochondral Junction Wide Ribs Expansile Rib Lesion Short Ribs Dense Ribs Hyperlucent Ribs

Notes:

CLAVICLE

Absence Of Outer End Of Clavicle Penciled Distal End Of Clavicle Destruction Of Medial End Of Clavicle

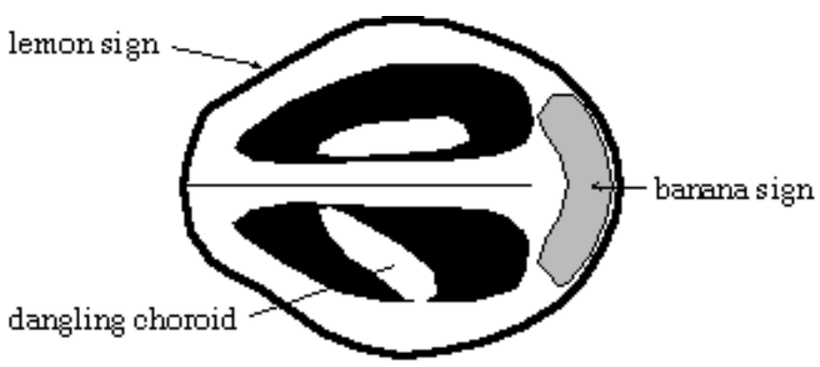
Notes:

WRIST & HAND

Carpal Angle Metacarpal Sign Lucent Lesion In Finger Resorption Of Terminal Tufts Acroosteolysis Fingertip Calcifications Syndactyly Polydactyly Clinodactyly Brachydactyly

Notes:

MYELOMENINGOCELE
=sac covered by leptomeninges containing CSF + variable amount of neural tissue; herniated through a defect in the posterior / anterior elements of spine*Incidence:*1:1,000-2,000 births (in Great Britain 1:200 births); twice as common in infants of mothers >35 years of age; Caucasians > Blacks > Orientals; most common congenital anomaly of CNS*Etiology:*localized defect of closure of caudal neuropore (usually closed by 28 days) ● positive family history in 10% ● neural placode = reddish neural tissue in the middle of back made up of open spinal cord ● normal skin / cutaneous abnormality: pigmented nevus, abnormal distribution of hair, skin dimple, angioma, lipoma ● MS-AFP (≥ 2.5 S.D. over mean) permits detection in 80% (positive predictive value of 2-5%) if defect not covered by full skin thickness*Recurrence rate:*3-7% chance of NTD with previously affected sibling / in fetus of affected parent*Associated with:* (1)Hydrocephalus (70-90%): requiring ventriculoperitoneal shunt in 90%◊25% of patients with hydrocephalus have spina bifida!(2)Chiari II malformation (100%)(3)Congenital / acquired kyphoscoliosis (90%)(4)Vertebral anomalies (vertebral body fusion, hemivertebrae, cleft vertebrae, butterfly vertebrae)(5)Diastematomyelia (31-46%): spinal cord split above (31%), below (25%), at the same level (22%) as the myelomeningocele(6)Duplication of central canal (5%) cephalic to + at level of placode(7)**Hemimyelocele** (10%) = two hemicords in separate dural tubes separated by fibrous / bony spur: one hemicord with myelomeningocele on one side of midline, one hemicord normal / with smaller myelomeningocele at a lower level ● impaired neurological function on side of hemimyelocele(8)Hydromyelia (29-77%) depending on efficacy of hydrocephalus treatment(9)Chromosomal anomalies (10-17%): trisomy 18,trisomy 13, triploidy, unbalanced translocation◊In 20% no detectable associated anomalies!Location: (a)**dorsal meningocele**: lumbosacral (70% below L2), suboccipital(b)**anterior sacral meningocele** = prolapse through anterior sacral bony defect; occasionally associated with neurofibromatosis type 1, Marfan syndrome, partial sacral agenesis, imperforate anus, anal stenosis, tethered spinal cord, GU tract / colonic anomalies; M:F = 1:4(c)**lateral thoracic meningocele** through enlarged intervertebral foramen into extrapleural aspect of thorax; right > left side, in 10% bilateral; often associated with neurofibromatosis (85%) + sharply angled scoliosis convex to meningocele√ expanded spinal canal√ erosion of posterior surface of vertebral body√ thinning of neural arch√ enlarged neural foramen(d)**lateral lumbar meningocele** through enlarged neural foramina into subcutaneous tissue / retroperitoneum; often associated with neurofibromatosis / Marfan syndrome√ expanded spinal canal√ erosion of posterior surface of vertebral body√ thinning of neural arch√ enlarged neural foramen(e)**traumatic meningocele** = avulsion of spinal nerve roots secondary to tear in meningeal root sheath; in C-spine after brachial plexus injury (most commonly) √ small irregular arachnoid diverticulum with extension outside the spinal canal(f)**cranial meningocele** = encephalocele OB-US: detection rate of 85-90%; sensitivity dependent on GA (fetal spine may be adequately visualized after 16-20 weeks GA); false-negative rate of 24% √ spinal level estimated by counting up from last sacral ossification center = S4 in 2nd trimester + S5 in 3rd trimester (79% accuracy for ± spinal level)√ may have clubfoot / rocker-bottom foot√ polyhydramnios @Spine:√ loss of dorsal epidermal integrity√ soft-tissue mass protruding posteriorly + visualization of sac√ widening of lumbar spine with fusiform enlargement of spinal canal√ splaying (= divergent position) of ossification centers of laminae with cup- / wedge-shaped pattern (in transverse plane = most important section for diagnosis)√ absence of posterior line = posterior vertebral elements (in sagittal plane)√ gross irregularity in parallelism of lines representing laminae of vertebrae (in coronal plane) √ anomalies of segmentation / hemivertebrae (33%) with short-radius kyphoscoliosis√ tethered cord (with lumbar / lumbosacral myelomeningocele)@ Head:



√ "lemon sign" = concave / linear frontal contour abnormality located at coronal suture associated with nonskin covered myelomeningocele (in 98% of fetuses ≤24 weeks + 13% of fetuses >24 weeks; positive predictive value 81-84%, in 0.7-1.3% of normal fetuses)√ "banana sign" = obliteration of cisterna magna with cerebellum wrapped around posterior brainstem secondary to downward traction of spinal cord in Arnold-Chiari malformation type II (in 96% of fetuses ≤24 weeks + in 91% of fetuses >24 weeks) √ nonvisualization of cerebellum√ effaced cisterna magna (100% sensitivity)◊the normal cisterna magna is 3-10 mm deep and usually visualized in 97% at 15-25 weeks GA√ BPD <5th percentile during 2nd trimester (65-79% sensitivity)√ HC <5th percentile (35% sensitivity)√ ventriculomegaly (40-90%) with choroid plexus incompletely filling the ventricles (54-63% sensitivity) = "dangling" choroid on dependent side; in 44% of myelomeningoceles <24 weeks GA; in 94% of myelomeningoceles during 3rd trimesterPlain films: √ bony defect in neural arch√ deformity + failure of fusion of lamina√ absent spinous process√ widened interpedicular distance√ widened spinal canal*Rx:*(1)Possibly elective cesarean section at 36-38 weeks GA (may decrease risk of contaminating / rupturing the meningomyelocele sac)(2)Repair within 48 hours*Postoperative complications:* (1)Postoperative tethering of spinal cord by placode / scar(2)Constricting dural ring(3)Cord compression by lipoma / dermoid / epidermoid cyst(4)Ischemia from vascular compromise(5)Syringohydromyelia*Prognosis:* (1)Mortality 15% by age 10 years(2)Intelligence: IQ <80 (27%); IQ >100 (27%); learning disability (50%)(3)Urinary incontinence: 85% achieve social continence (scheduled intermittent catheterization)(4)Motor function: some deficit (100%); improvement after repair (37%)(5)Hindbrain dysfunction associated with Chiari II malformation (32%)(6)Ventriculitis: 7% in initial repair within 48 hours, more common in delayed repair >48 hours

Notes:

HIP

Snapping Hip Syndrome Protrusio Acetabuli Pain With Hip Prosthesis Evaluation Of Total Hip Arthroplasty Tibiotalar Slanting

Notes:

Uptake at 24 hours:most intense in RES, liver, spleen (4%), bone marrow (lumbar spine, sacroiliac joints), bowel wall (chiefly colonic activity on delayed images), renal cortex, nasal mucosa, lacrimal + salivary glands, blood pool (20%), lung (<3% = equivalent to background activity), breastsat 72 hours:activity in liver, skeleton, colon, nasal mucosa, occiput; kidney activity no longer detectable; lacrimal + salivary glands may still be prominent

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Notes:

FOOT

Abnormal Foot Positions Clubfoot = Talipes Equinovarus Rocker-bottom Foot = Vertical Talus Heel Pad Thickening

Notes:

SOFT TISSUES

Histologic Classification Of Soft-tissue Lesions Fat-containing Soft-tissue Masses Muscle Hyperintensity On STIR Images Extraskeletal Osseous + Cartilaginous Tumors Soft-tissue Calcification Interstitial Calcinosis Soft-tissue Ossification Connective Tissue Disease

Notes:

FIXATION DEVICES

Internal Fixation Devices External Fixation Devices Intramedullary Fixation Devices

Notes:

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BONE MINERALS

Calcium Phosphorus

Notes:

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Excretion (a)via GI tract (10-20%)hepatobiliary pathway + colonic mucosal excretion: enemas + laxatives promote clearing of bowel activity (b)via urinary tract (10-20% within 24 hours)no activity in kidneys + urinary bladder after 24 hours (c)via various body fluidseg, human milk (mandates to stop nursing for 2 weeks)

Notes:

HORMONES

Parathormone Vitamin D Metabolism Calcitonin

Notes:

ACHONDROPLASIA

Heterozygous Achondroplasia Homozygous Achondroplasia

Notes:

SPINAL STENOSIS
= encroachment on central spinal canal, lateral recess, or neuroforamen by bone / soft tissue *Cause:* A.Congenitally short pedicles(a)idiopathic(b)developmental: Down syndrome, achondroplasia, hypochondroplasia, Morquio diseaseB.Acquired:1.Hypertrophy of ligamentum flavum = buckling of ligament secondary to joint slippage in facet joint osteoarthritis (most common)2.Facet joint hypertrophy3.Degenerated bulging disk4.Spondylosis, spondylolisthesis5.Surgical fusion6.Fracture7.Ossification of posterior longitudinal ligament8.Paget disease9.Epidural lipomatosis*Age:*middle-aged for congenital cause / elderly during 6th-8th decade for acquired cause; M > FLocation:generally involves lumbar spinal canal; cervical spinal canal may be similarly affected√ distorted shape of thecal sac√ obliteration of epidural fat√ narrowing of cervical canal <13 mm, of lumbar canal<16 mm (AP diameter)√ interpedicular distance <25 mm◊Measurements are not a valid indicator of disease! ***Lumbar Spinal Stenosis*** *Cause:* 1.Achondroplasia:√ narrowed interpediculate distance progressive toward lumbar spine2.Paget disease: bony overgrowth3.Spondylolisthesis4.Operative posterior spinal fusion5.Herniated disk6.Metastasis to vertebrae7.Developmental / congenital*Age:*presentation between 30-50 years of age ● low back pain ● "neurogenic claudication" = bilateral lower extremity pain, numbness, weakness worse during walking / standing + relieved in supine position and flexion ● cauda equina syndrome: paraparesis, incontinence, sensory findings in saddlelike pattern, areflexia√ sagittal diameter of spinal canal <12 mm (normal range in adults: 15-23 mm)√ dural sac area <100 mm^2 √ diminished amount of CSF + crowding of nerve roots√ unusual small quantity of contrast material to fill thecal sac√ anteroposterior + interpediculate diameter spinal canal constricted√ hourglass configuration of thecal sac (SAG view)√ triangular / trefoil shape of thecal sac (AXIAL view)√ redundant serpiginous nerve roots above + below stenosis√ may appear as spinal block in hyperextended neck on AP views√ thickened articular process, pedicles, laminae, ligaments√ bulging disks

Notes:

Sensitivity =ability to detect disease=probability of having an abnormal test given disease=number of correct positive tests / number with disease=true positive ratio = TP / (TP + FN) = TP / D+ ● D+ column in decision matrix◊independent of prevalence

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Notes:

Specificity =ability to identify absence of disease=probability of having a negative test given no disease=number of correct negative tests / number without disease=true negative ratio = TN / (TN + FP) = TN / D- ● D- column in decision matrix◊independent of prevalence

Notes:

DISLOCATION

Hip Dislocation Patellar Dislocation Shoulder Dislocation Wrist Dislocation

Notes:

Positive Predictive Value =positive test accuracy=likelihood that a positive test result actually identifies presence of disease=number of correct positive tests / number of positive tests=TP / (TP + FP) = TP / T+ ● T+ row in decision matrix◊dependent on prevalence◊PPV increases with increasing prevalence for given sensitivity + specificity◊PPV increases with increasing specificity for given prevalence

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Notes:

SPONDYLOLISTHESIS
=forward displacement of one vertebra over another*Incidence:*4% of general populationLocation:L5/S1 or L4/L5Grades I-IV (Meyerding method): each grade equals 1/4 anterior subluxation of superior on inferior vertebral body

Isthmic Spondylolisthesis = open-arch type Degenerative Spondylolisthesis = closed-arch type

Notes:

FIBROUS HISTIOCYTOMA

Benign fibrous histiocytoma Atypical Benign Fibrous Histiocytoma Malignant Fibrous Histiocytoma

Notes:

Geometry =to assure that measurement is not dependent upon location of tracer within ionization chamber, usually done by manufacturer*Test frequency:*at installation / after factory repair / recalibration*Method:*0.5 mL of Tc-99m (activity 25 mCi) is measured in a 3-mL syringe; syringe contents are then diluted with water to 1.0 mL, 1.5 mL, and 2.0 mL and each level remeasured; test is repeated with a 10-mL glass vial

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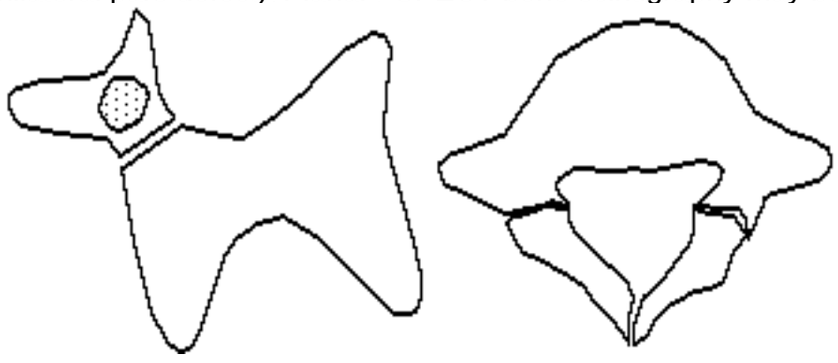
Notes:

SPONDYLOLYSIS
=pars interarticularis defect between superior + inferior articulating processes as the weakest portion of spinal unit*Incidence:*3-7% of population; in 30-70% other family members afflicted*Age:*early childhood; M:F = 3:1; Whites:Blacks = 3:1*Cause:* (a)pseudarthrosis following stress (fatigue) fracture of pars (in most) from repetitive minor trauma; common in gymnastics (30%), diving, contact sports (football, soccer, hockey, lacrosse)(b)hereditary hypoplasia of pars leads to insufficiency fracture; eg, pars defect in 34% of Eskimos(c)secondary spondylolysis: neoplasm, osteomyelitis, Paget disease, osteomalacia, osteogenesis imperfecta(d)congenital malformation: frequently associated with spina bifida occulta of S1, dorsally wedge-shaped body of L5, hypoplasia of L5; HOWEVER: no pars defects have been identified in fetal cadavers ● symptomatic in 50% (if associated with degenerative disk disease / spondylolisthesis)Location:L5 (67-95%); L4 (15-30%); L3 (1-2%);in 75% bilateralPlain film: √ radiolucent band ± sclerotic margin resembling the collar of the "Scottie dog" (on oblique view)√ may be associated with spondylolisthesis√ subluxation of involved vertebra (if pars defect bilateral)√ Wilkinson syndrome = reactive sclerosis + bony hypertrophy of contralateral pedicle + lamina (produced by stress changes related to weakening of neural arch in unilateral pars defect)◊Planar / SPECT bone scintigraphy may be useful!CT: √ pars defect located 10-15 mm above disk space√



inner contour of spinal canal interrupted

Spondylolysis oblique radiograph of L5CT scan through mid-vertebral body

Spondylolysis of Cervical Spine

Notes:

Linearity =accurate measurement over large range of activity levels*Test frequency:*4 x per year*Method:*1 mCi source activity is measured every 4 hours for 10 / more measurements (down to 10-100 µCi)*Evaluation:*measurements must fall within ± 5% of the calculated physical decay curve

Notes:

ANGIOGRAPHY Burning sensation: (a)intense with concentration of 60-76% HOCM(b)reduced with concentration of ≤30% HOCM / LOCM♦Overall incidence of adverse allergic-type reactions is (for unknown reasons) much less with intra-arterial than with intravenous use of contrast media!

Notes:

CHORDOMA

◊Chordoma is the most common primary malignant tumor of the spine in adults excluding lymphoproliferative neoplasms!*Prevalence:*1:2,000,000; 1-2-4% of all primary malignant neoplasms of bone; 1% of all intracranial tumors*Etiology:*originates from embryonic remnants of notochord / ectopic cordal foci (notochord appears between 4th and 7th week of embryonic development, extends from Rathke pouch to coccyx and forms nucleus pulposus)*Age:*30-70 years (peak age in 6th decade);M:F = 2:1; highly malignant in children*Path:*lobulated tumor contained within pseudocapsule*Histo:* (1)typical chordoma: cords + clusters of large bubblelike vacuolated (physaliferous) cells containing intracytoplasmic mucous droplets; abundant extracellular mucus deposition + areas of hemorrhage(2)chondroid chordoma: cartilage instead of mucinous extracellular matrixLocation:(a) 50% in sacrum (b) 35% in skull base(c) 15% spinal axis (d) other sites (5%) in mandible, maxilla, scapula√ enhancement after contrast administrationCT: √ low-attenuation within soft-tissue mass (due to myxoid-type tissue)√ higher attenuation fibrous pseudocapsuleMR (modality of choice): √ heterogeneous low to intermediate intensity on T1WI, occasionally hyperintense (due to high protein content)√ very high signal intensity on T2WI (similar to nucleus pulposus with high water content)NUC: √ cold lesion on bone scan√ no uptake on gallium scanMetastases (in 5-43%) to:liver, lung, regional lymph nodes, peritoneum, skin (late), heart*Prognosis:*almost 100% recurrence rate despite radical surgery

Sacrococcygeal Chordoma (50-70%) Spheno-occipital Chordoma (15-35%) Vertebral / Spinal Chordoma (15-20%)

Notes:

Superscan A.Metabolic1.Renal osteodystrophy2.Osteomalacia√ randomly distributed focal sites of intense activity = Looser zones = pseudofractures= Milkman fractures (most characteristic)3.Hyperparathyroidism√ focal intense uptake corresponds to site of brown tumors4.Hyperthyroidismrate of bone resorption more increased than rate of formation (= decrease in bone mass) ● hypercalcemia (occasionally) ● elevated alkaline phosphatase√ NOT visible on radiographs√ susceptible to fractureB.Widespread bone lesions1.Diffuse skeletal metastases (most frequent) from prostate, breast, multiple myeloma, lymphoma, lung, bladder, colon, stomach2.Myelofibrosis / myelosclerosis3.Aplastic anemia, leukemia4.Waldenström macroglobulinemia5.Systemic mastocytosis6.Widespread Paget disease √ diffusely increased activity in bones: particularly prominent in axial skeleton, calvarium, mandible, costochondral junctions (= "rosary beading"), sternum (= "tie sternum"), long bones√ increased metaphyseal + periarticular activity√ increased bone-to-soft-tissue ratio√ "absent kidney sign" = little / no activity in kidneys but good visualization of urinary bladder√ femoral cortices become visibleCAVE:scan may be interpreted as normal, particularly in patients with poor renal function!

Notes:

SCHEUERMANN DISEASE
=SPINAL OSTEOCHONDROSIS = KYPHOSIS DORSALIS JUVENILIS = VERTEBRAL EPIPHYSITIS=disorder consisting of vertebral wedging + endplate irregularity + narrowing of intervertebral disk space*Incidence:*in 31% of male + 21% of female patients with back pain*Age:*onset at pubertyLocation:lower thoracic / upper lumbar vertebrae; in mild cases limited to 3-4 vertebral bodies√ anterior wedging of vertebral body of >5°√ increased anteroposterior diameter of vertebral body√ slight narrowing of disk space√ kyphosis of >40°/ loss of lordosis; scoliosis√ Schmorl nodes (intravertebral herniation of nucleus pulposus into vertebral body) = depression in contour of endplate in posterior half of vertebral body; found in up to 30% of adolescents + young adults√ flattened area in superior surface of epiphyseal ring anteriorly = avulsion fracture of ring apophysis due to migration of nucleus pulposus through weak point between ring apophysis + vertebral endplate (fusion of ring apophysis usually occurs at about 18 years of age)√ detached epiphyseal ring anteriorly*DDx:*(1)Developmental notching of anterior vertebrae (NO wedging or Schmorl nodes)(2)Osteochondrodystrophy (earlier in life, extremities show same changes)

Notes:

Accuracy *Test frequency:*annually*Method:*measurements of three different activity standards whose amount is certified by the National Bureau of Standards (NBS); standard values are decayed mathematically to calibrator dateCo-57:123 keV, half-life of 270 daysBa-133:354 keV, half-life of 7.2 yearsCs-137:662 keV, half-life of 30 years*Evaluation:*measurements must fall within expected range

Notes:

Negative Predictive Value =negative test accuracy=likelihood that a negative test result actually identifies absence of disease=number of correct negative tests / number of negative tests=TN / (TN + FN) = TN / T- ● T- row in decision matrix◊dependent on prevalence◊NPV increases with decreasing prevalence for given sensitivity + specificity◊NPV increases with increasing sensitivity for given prevalence

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Notes:

Mixed Bone Metastases breast, prostate, lymphoma

Notes:

Expansile / Bubbly Bone Metastases kidney, thyroid

Notes:

Permeative Bone Metastases Burkitt lymphoma, Mycosis fungoides

Notes:

Bone Metastases With "Sunburst" Periosteal Reaction (infrequent) prostatic carcinoma, retinoblastoma, neuroblastoma (skull), GI tract

Notes:

Bone Metastases With Soft-tissue Mass thyroid, kidney

Notes:

OSTEOMYELITIS

Acute Osteomyelitis Chronic Osteomyelitis Brodie Abscess Epidermoid Carcinoma

Notes:

Uniformity 1.64 x 64 word matrix = 30 million count flood with collimator, orientation and magnification same as patient study2.Co-57 sheet source with <1% uniformity variance is necessary3.128 x 128 word matrix = 120 million count flood with collimator, orientation, and magnification same as patient study*Frequency of quality control*:weekly

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Notes:

MYOCARDIAL ISCHEMIA
can be assessed (a)directly with stress Tl-201 imaging(b)indirectly with gated blood pool imaging (wall motion, ejection fraction) LOCATION OF PERFUSION DEFECTS (1)Right coronary artery (RCA)best seen on left LAT / AP projections √ inferior + posteroseptal segments(2)Circumflex branch of left coronary artery (LCX)best seen on LAO projection √ posterolateral segment(3)Anterior descending branch of left coronary artery (LAD)√ anteroseptal, anterior, anterolateral segmentsN.B.:decreased activity in apical + posterior segments is not reliably correlated with disease of any vessel!

Notes:

SPONDYLOEPIPHYSEAL DYSPLASIA

Spondyloepiphyseal Dysplasia Congenita Spondyloepiphyseal Dysplasia Tarda

Notes:

KLIPPEL-FEIL SYNDROME
=BREVICOLLIS=synostosis of two / more cervical segments*May be associated with:* platybasia, syringomyelia, encephalocele, facial + cranial asymmetry, Sprengel deformity (25-40%), syndactyly, clubbed foot, hypoplastic lumbar vertebrae; renal anomalies in 50% (agenesis, dysgenesis, malrotation, duplication, renal ectopia); congenital heart disease in 5% (atrial septal defect, coarctation) ● clinical triad of(1)short neck(2)restriction of cervical motion(3)low posterior hairline ● deafness (30%) ● webbed neckLocation:cervical spine√ fusion of vertebral bodies and posterior elements√ ± hemivertebrae√ may have cervicothoracic / cervical / atlanto-occipital fusion√ torticollis√ scoliosis√ rib fusion√ Sprengel deformity (25-40%) = elevation + medial rotation of scapula (may be related to presence of anomalous omovertebral bone)√ ear anomalies: absent auditory canal, microtia, deformed ossicles, underdevelopment of bony labyrinth

Notes:

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ARACHNOIDITIS

*Etiology:*back surgery, hemorrhage, trauma, Pantopaque (inflammatory effect potentiated by blood), idiopathic*Associated with:*syrinxMyelo: √ blunting of nerve root sleeves√ blocked nerve roots without cord displacement (2/3)√ streaking + clumping of contrastCT: √ fusion / clumping of nerve roots√ featureless empty-looking sac with roots adherent to wall (final stage)

Notes:

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ARACHNOID CYST OF SPINE

Location:dorsal to cord in thoracic regionSite: (a)extradural cyst secondary to congenital / acquired dural defect(b)intradural secondary to congenital deficiency within arachnoid (= true arachnoid cyst) / adhesion from prior infection or trauma (= arachnoid loculation)√ oval sharply demarcated extramedullary mass√ immediate / delayed contrast filling depending upon size of opening between cyst + subarachnoid space√ local displacement + compression of spinal cord√ higher signal intensity than CSF (from relative lack of CSF pulsations)

Notes:

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ARACHNOID DIVERTICULUM
=widening of root sheath with arachnoid space occupying >50% of total transverse diameter of root + sheath together*Cause:*? congenital / traumatic, arachnoiditis, infection*Pathogenesis:*hydrostatic pressure of CSF√ scalloping of posterior margins of vertebral bodies√ myelographic contrast material fills diverticula

Notes:

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ARTERIOVENOUS MALFORMATION OF SPINAL CORD

Classification: 1.True intramedullary AVM=nidus of abnormal intermediary arteriovenous structure with multiple shunts*Age:*2nd-3rd decade*Cx:*subarachnoid hemorrhage, paraplegia*Prognosis:*poor (especially in midthoracic location)2.Intradural arteriovenous fistula=single shunt between one / several medullary arteries + single perimedullary vein3.Dural arteriovenous fistula=single shunt between meningeal arteries + intradural vein4.Metameric angiomatosis

Notes:

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ATLANTOAXIAL ROTARY FIXATION

● history of insignificant cervical spine trauma / upper respiratory tract infection ● limited painful neck motion ● head held in "cock-robin" position + inability to turn head √ atlanto-odontoid asymmetry (open mouth odontoid view):√ decrease in atlanto-odontoid space + widening of lateral mass on side ipsilateral to rotation √ increase in atlanto-odontoid space + narrowing of lateral mass on side contralateral to rotation√ atlantoaxial asymmetry remains constant with head turned into neutral position*Types:* I<3 mm anterior displacement of atlas on axisII3-5 mm anterior displacementIII>5 mm anterior displacementIVposterior displacement of atlas on axis*DDx:*torticollis (atlantoaxial symmetry reverts to normal with head turned into neutral position)

Notes:

BRACHIAL PLEXUS INJURY

1.Erb-Duchenne: adduction injury affecting C5/6 (downward displacement of shoulder)2.Klumpke: abduction injury at C7, C8, T1 (arm stretched over head)√ pouchlike root sleeve at site of avulsion√ asymmetrical nerve roots√ contrast extravasation collecting in axilla√ metrizamide in neural foramina (CT myelography)

Notes:

CAUDAL REGRESSION SYNDROME
=midline closure defect of neural tube with a spectrum of anomalies*Etiology:*disturbance of caudal mesoderm <4th week of gestation from toxic / infectious / ischemic insult*Incidence:*1:7,500 births; 0.005-0.01%*Predisposed:*infants of diabetic mothers (16-22%)◊NOT associated with VATER syndrome!A.Musculoskeletal anomalies@Lower extremity ● symptoms from minor muscle weakness to complete sensorimotor paralysis of both lower extremities√ hip dislocation√ foot deformities √ hypoplasia of extremities@Lumbosacral spine√ spina bifida (myelomeningocele often not in combination with hydrocephalus)√ total / partial sacral agenesis√ total / partial agenesis of lumbosacral spine√ fusion of caudal-most 2 or 3 vertebrae√ narrowing of spinal canal rostral to last intact vertebra√ characteristic wedge-shaped cord terminus (hypoplasia of distal spinal cord)√ spinal cord may be tethered ± associated lipoma√ ± dural sac stenosis with high termination√ ± spinal cord lipoma, teratoma, cauda equina cystB.Genitourinary anomalies ● neurogenic bladder (if >2 segments are missing) ● malformed external genitalia ● lack of bowel control√ ± bilateral renal aplasia with pulmonary hypoplasia + Potter facies√ anal atresia OB-US: ● normal / imperforate anus√ normal / mildly dilated urinary system√ normal / increased amniotic fluid√ 2 umbilical arteries√ 2 hypoplastic nonfused lower extremities√ sacral agenesis, absent vertebrae from lower thoracic / upper lumbar spine caudally

Sirenomelia

Notes:

Sirenomelia =recently considered a distinct separate entity from caudal regression syndrome◊NOT associated with maternal diabetes mellitus! ● Potter facies ● absence of anus ● absent genitalia√ bilateral renal agenesis / dysgenesis (lethal)√ marked oligohydramnios√ single aberrant umbilical artery√ single / fused lower extremity√ sacral agenesis, absent pelvis, lumbosacral "tail", lumbar rachischisis*Prognosis:*incompatible with life

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Notes:

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Sacrococcygeal Chordoma (50-70%) 40% of all sacral tumors *Peak age:*40-60 years; M:F = 2:1 ● low back pain (70%) ● constipation / fecal incontinence ● rectal bleeding (42%) ● sciatica ● frequency, urgency, straining on micturition ● sacral mass (17%)Location:esp. in 4th + 5th sacral segment√ presacral mass with average size of 10 cm extending superiorly + inferiorly; rarely posterior location√ displacement of rectum + bladder√ solid tumor with cystic areas (in 50%)√ osteolytic midline mass in sacrum + coccyx√ amorphous peripheral calcifications (15-89%)√ secondary bone sclerosis in tumor periphery (50%)√ honeycomb pattern with trabeculations (10-15%)√ may cross sacroiliac joint*Prognosis:*8-10 years average survival; 66% 5-year survival rate (adulthood)*DDx:*Giant cell tumor, plasmacytoma, lymphoma, metastatic adenocarcinoma, aneurysmal bone cyst, atypical hemangioma, chondrosarcoma, osteomyelitis, ependymoma

Notes:

Spheno-occipital Chordoma (15-35%) *Age:*younger patient (peak age of 20-40 years);M:F - 1:1 ● orbitofrontal headache ● visual disturbances, ptosis ● 6th nerve palsy / paraplegiaLocation:clivus, spheno-occipital synchondrosis√ bone destruction (in 90%): clivus > sella > petrous bone > orbit > floor of middle cranial fossa > jugular fossa > atlas > foramen magnum√ reactive bone sclerosis (rare)√ calcifications / bone fragments (20-70%)√ soft-tissue extension into nasopharynx (common), into sphenoid + ethmoid sinuses (occasionally), may reach nasal cavity + maxillary antrum√ variable degree of enhancementMR: √ large intraosseous mass extending into prepontine cistern, sphenoid sinus, middle cranial fossa, nasopharynx√ posterior displacement of brainstem√ usually isointense to brain / occasionally inhomogeneously hyperintense on T1WI√ hyperintense on T2WI*Prognosis:*4-5 years average survival*DDx:*meningioma, metastasis, plasmacytoma, giant cell tumor, sphenoid sinus cyst, nasopharyngeal carcinoma, chondrosarcoma

Notes:

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Vertebral / Spinal Chordoma (15-20%) more aggressive than sacral / cranial chordomas *Age:*younger patient; M:F = 2:1 ● low back pain + radiculopathyLocation:cervical (8% - particularly C2), thoracic spine (4%), lumbar spine (3%)√ solitary midline spinal mass√ tumor calcification in 30%√ sclerosis / "ivory vertebra" in 43-62%√ total destruction of vertebra, initially unaccompanied by collapse√ variable extension into spinal canal√ violates disk space to involve adjacent bodies (10-14%) simulating infection√ anterior soft-tissue mass*Cx:*complete spinal block*Prognosis:*4-5 years average survival*DDx:*Metastasis, primary bone tumor, primary soft-tissue tumor, neuroma, meningioma

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Notes:

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CSF FISTULA

Cause: (1)Trauma to skull base (most commonly)◊2% of all head injuries develop CSF fistula(2)Tumor: especially those arising from pituitary gland(3)Congenital anomalies: encephalocele ● traumatic leak: usually unilateral; onset within 48 hours after trauma, usually scanty; resolve in 1 week ● nontraumatic leak: profuse flow; may persist for years ● anosmia (in 78% of trauma cases)Location:fractures through frontoethmoidal complex + middle cranial fossa (most commonly)♈ high-resolution thin-section CT in coronal plane followed by rescanning after low-dose intrathecal contrast material instilled into lumbar subarachnoid space*Cx:*infection (in 25-50% of untreated cases)

Notes:

DEGENERATIVE DISK DISEASE

◊Therapeutic decision-making should be based on clinical assessment alone!◊There are no prognostic indicators on images in patients with acute lumbar radiculopathy!35% of individuals without back trouble have abnormal findings (HNP, disk bulging, facet degeneration, spinal stenosis) ◊Imaging tests are only justified in patients for whom surgery is considered! *Pathophysiology:* loss of disk height leads to malalignment (= rostrocaudal subluxation) of facet joints causing spine instability with arthritis, capsular hypertrophy, hypertrophy of posterior ligaments, facet fracture Plain film: √ narrowing of disk space√ disk calcification√ vacuum disk phenomenon = radiolucent interspace accumulation of nitrogen gas at sites of negative pressure√ intervertebral osteochondrosis = loss of disk space height + bone sclerosis of adjacent vertebral bodies√ cartilaginous nodes = intraosseous disk herniation√ spondylosis deformans = endplate osteophytosis secondary to anterolateral disk displacement resulting in traction osteophytes at sites of osseous attachment of annulus fibrosus fibers of SharpeyMyelography: √ delineation of thecal sac, spinal cord, exiting nerve rootsCT (accuracy >90%): √ facet joint disease (marginal sclerosis, joint narrowing, cyst formation, bony overgrowth)MR: √ endplate changes (Modic & DeRoos):(a)Type I(4%) with decreased signal on T1WI + increased signal on T2WI (= vascularized fibrous tissue), contrast-enhancement of marrow(b)Type II(16%) with increased signal on T1WI + isointensity on T2WI (= local fatty replacement of marrow)(c)Type IIIwith decreased signal on T1WI + T2WI (= advanced sclerosis)NUC: SPECT imaging of vertebrae can aid in localizing increased uptake to vertebral bodies, posterior elements, etc. √ eccentrically placed increased uptake on either side of an intervertebral space (osteophytes, discogenic sclerosis) *Sequelae:*(1) disk bulging (2) disk herniation (3) spinal stenosis (4) facet joint disease TERMINOLOGY: 1.Disk bulge=concentric smooth circumferential expansion of softened disk material beyond the confines of endplates2.Disk protrusion=focal protrusion of disk material maintaining broad base with parent disk due to focally weakened / ruptured annulus but intact posterior longitudinal ligament3.Disk extrusion=prominent focal extrusion of disk material with only an isthmus of connection to parent disk due to(a)ruptured annulus + intact posterior longitudinal ligament(b)ruptured annulus + ruptured posterior longitudinal ligament4.Free fragment=frank separation of disk material from parent disk5.Free fragment migration=separated disk material travels above / below intervertebral disk space

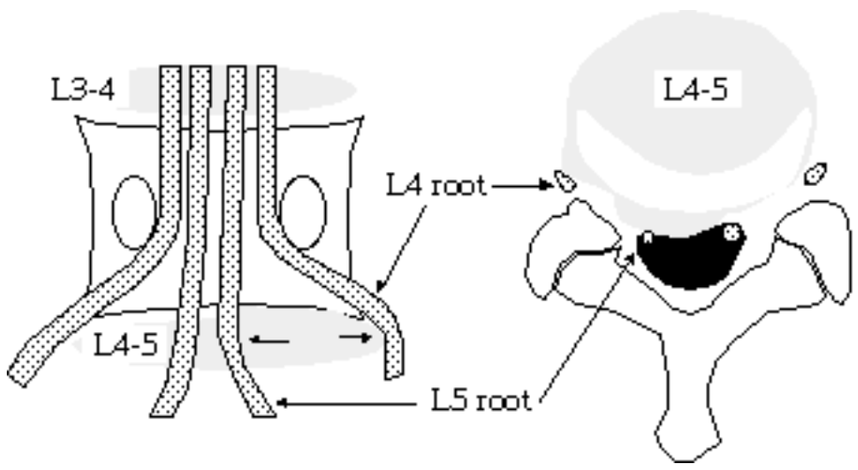
Bulging Disk Herniation of Nucleus Pulposus Free Fragment Herniation Cervical Disk Herniation

Notes:

Bulging Disk =broad-based disk extension outward in all directions with intact but weakened annulus fibrosus + posterior longitudinal ligament*Age:*common finding in individuals >40 years of ageLocation:lumbar, cervical spine√ rounded symmetric defect localized to disk space level√ concave anterior margin of thecal sacMR: √ nucleus pulposus hypointense on T1WI + hyperintense on T2WI (water loss through degeneration)

Notes:

Herniation of Nucleus Pulposus =HNP = focal protrusion of disk material beyond margins of adjacent vertebral endplates secondary to rupture of annulus fibrosus confined within posterior longitudinal ligament◊21% of asymptomatic population has disk herniation! ● local somatic spinal pain = sharp / aching, deep, localized ● centrifugal radiating pain = sharp, well-circumscribed, superficial, "electric," confined to dermatome ● centrifugal referred pain = dull, ill-defined, deep or superficial, aching or boring, confined to somatome(= dermatome + myotome + sclerotome) Location:L4/5 (35%) > L5/S1 (27%) > L3/4 (19%) > L2/3 (14%) > L1/2 (5%); thoracic spine affected in 3:1,000 disk operations(a)posterolateral (49%) = weakest point along posterolateral margin of disk at lateral recess of spinal canal (posterior longitudinal ligament tightly adherent to posterior margins of disk)(b)posterocentral (8%)(c)bilateral (on both sides of posterior ligament)(d)lateral / foraminal (<10%)(e)intraosseous / vertical = Schmorl node (14%)(f)extraforaminal = anterior (commonly overlooked) (29%)Myelography: √ sharply angular indentation on lateral aspect of thecal sac with extension above or below level of disk space (ipsilateral oblique projection best view)√ asymmetry of posterior disk margin√ double contour secondary to superimposed normal + abnormal side (horizontal beam lateral view)√ narrowing of intervertebral disk space (most commonly a sign of disk degeneration)√ deviation of nerve root / root sleeve√ enlargement of nerve root secondary to edema ("trumpet sign")√ amputated / truncated nerve root (nonfilling of root sleeve)MR: √ herniated disk material of low signal intensity displaces the posterior longitudinal ligament and epidural fat of relative high signal intensity on T1WI*Cx:*spinal stenosis*Prognosis:* conservative therapy reduces size of herniation by 0-50%in 11% of patients,50-75%in 36% of patients,75-100%in 46% of patients(secondary to growth of granulation tissue)



Lateral Disk Herniation Nerve compression usually occurs posterolaterally (here at L4-5); therefore an atypical lateral compression (here of L4 root) directs surgery to the wrong more cephalad level (L3-4 disk)

Notes:

Free Fragment Herniation =DISK SEQUESTRATION=complete separation of disk material with rupture through posterior longitudinal ligament into epidural space Missed free fragments are a common cause of failed back surgery! migration superiorly / inferiorly away from disk space with compression of nerve root above / below level of disk herniation disk material noted >9 mm away from intervertebral disk space soft-tissue density with higher value than thecal sac*DDx:*(1)Postoperative scarring (retraction of thecal sac to side of surgery)(2)Epidural abscess(3)Epidural tumor(4)Conjoined nerve root (2 nerve roots arising from thecal sac simultaneously representing mass in ventrolateral aspect of spinal canal; normal variant in 1-3% of population)(5)Tarlov cyst (dilated nerve root sleeve)

Notes:

Cervical Disk Herniation *Peak age:*3rd-4th decade ● neck stiffness, muscle splinting ● dermatomic sensory loss ● weakness + muscle atrophy ● reflex lossSites:C6-7 (69%); C5-6 (19%); C7-T1 (10%);C4-5 (2%)*Sequelae:*(1)compression of exiting nerve roots(2)cord compression (spinal stenosis + massive disk rupture)

Notes:

DERMOID OF SPINE
=uni- / multilocular cystic tumor lined by squamous epithelium containing skin appendages (hair follicles, sweat glands, sebaceous glands)*Cause:* (a)congenital dermal rest / focal expansion of dermal sinus(b)acquired from implantation of viable dermal tissue (by spinal needle without trocar)*Incidence:*1% of spinal cord tumors*Age at presentation:*<20 years; M:F = 1:1*May be associated with:*dermal sinus (in 20%) ● slowly progressive myelopathy ● acute onset of chemical meningitis (secondary to rupture of inflammatory cholesterol crystals from cyst into CSF) Location:lumbosacral (60%), cauda equina (20%)Site:extramedullary (60%), intramedullary (40%)√ almost always complete spinal block on myelography√ intensity of fat√ occasionally hypointense on T1WI + hypodense on CT (secretions from sweat glands within tumor)√ NO contrast enhancement√ CT myelography facilitates detection

Notes:

DIASTEMATOMYELIA
=SPLIT CORD = MyeloschisisMYELOSCHISIS=sagittal division of spinal cord into two hemicords, each of which contains a central canal, one dorsal horn + one ventral horn*Etiology:*congenital malformation as a result of split notochord; M:F = 1:3 *Path:* (a)2 hemicords each covered by layer of pia within single subarachnoid space + dural sac (60%); not accompanied by bony spur / fibrous band(b)2 hemicords each with its own pial, subarachnoidal + dural sheath (40%); accompanied by fibrous band (in 25%), cartilaginous / bony spurs (in 75%)*Associated with:*myelomeningocele ● hypertrichosis, nevus, lipoma, dimple, hemangioma overlying the spine (26-81%) ● clubfoot (50%) ● muscle wasting, ankle weakness in one legLocation:lower thoracic / upper lumbar > upper thoracic > cervical spine√ congenital scoliosis (50-75%)√5% of patients with congenital scoliosis have diastematomyelia√ spina bifida over multiple levels√ anteroposterior narrowing of vertebral bodies√ widening of interpediculate distance√ narrowed disk space with hemivertebra, butterfly vertebra, block vertebra√ fusion + thickening of adjacent laminae (90%)(a)fusion to ipsilateral lamina at adjacent levels(b)diagonal fusion to contralateral adjacent lamina= intersegmental laminar fusion√ bony spur through center of spinal canal arising from posterior aspect of centra (<50%)√ thickened filum terminale >2 mm (>50%)√ tethered cord (>50%)√ low conus medullaris below L2 level (>75%)√ the 2 hemicords usually reunite caudal to cleft√ defect in thecal sac on myelogram*Cx:*progressive spinal cord dysfunction

Notes:

DISCITIS

most common pediatric spine problem *Etiology:*
(1)Bloodborne bacterial invasion of vertebrae infecting disk via communicating vessels through endplate(2)invasive procedure: surgery, discography, myelography, chemonucleolysis*Agents:* (a)pyogenic: Staphylococcus aureus (by far most frequent), Gram-negative rods (in IV drug abusers / immunocompromised patients)(b)nonpyogenic: tuberculosis, coccidioidomycosis*Pathogenesis:*infection starts in disk (still vascularized in children) / in anterior inferior corner of vertebral body (in adults) with spread across disk to adjacent vertebral endplate*Age peak:*6 months to 4 years and 10-14 years; average age of 6 years at presentation ● over 2-4 weeks gradually progressing irritability, malaise, fever ● back / referred hip pain, limp ● refusal to bear weightLocation:L3/4, L4/5, unusual above T9; usually involvement of one disk space (occasionally 2)Plain film (positive 2-4 weeks after onset of symptoms): √ decrease in disk space height (earliest sign)= intraosseous herniation of nucleus pulposus into vertebral body through weakened endplate √ indistinctness of adjacent endplates with destruction√ endplate sclerosis (during healing phase beginning anywhere from 8 weeks to 8 months after onset)√ bone fusion (after 6 months to 2 years)CT: √ paravertebral inflammatory mass√ epidural soft-tissue extension with deformity of thecal sacMR (preferred modality; 93% sensitive, 97% specific, 95% accurate): √ decreased marrow intensity on T1WI in two contiguous vertebrae√ in early stage preserved disk height with variable intensity on T2WI (often increased)√ in later stages loss of disk height with increased intensity on T2WI NUC (41% sensitive, 93% specific, 68% accurate on Tc-99m MDP + Tc-99m WBC scans): √ positive before radiographs√ increased uptake in disk space + contiguous vertebra√ bone scan usually positive in adjacent vertebrae (until age 20) secondary to vascular supply via endplates; may be negative after age 20*Cx:*kyphosis*Rx:*immobilization in body cast for ~4 weeks*DDx:*osteomyelitis of vertebra

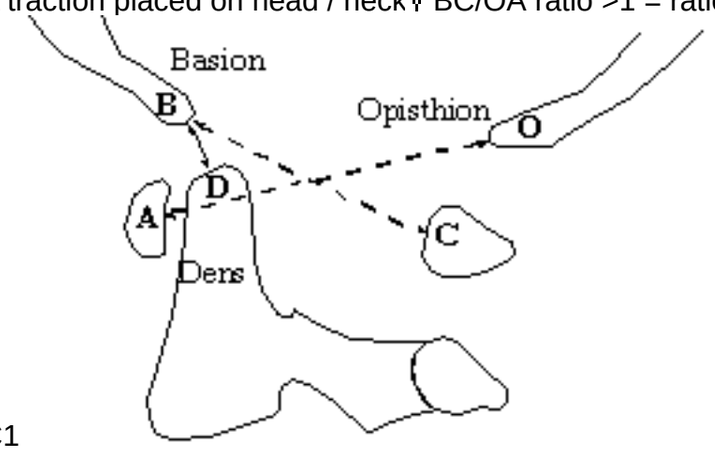
Postoperative Discitis

Notes:

Postoperative Discitis *Frequency:*0.75-2.8%*Organism:*Staphylococcus aureus; many times no organism recovered ● severe recurrent back pain 7-28 days after surgeryaccompanied by decreased back motion, muscle spasm, positive straight leg raising test ● fever (33%) ● wound infection (8%) ● persistently elevated / increasing ESRMR: √ decreased signal intensity within disk + adjacent vertebral body marrow on T1WI√ increased signal intensity in disk + adjacent marrow on T2WI often with obliteration of intranuclear cleft√ contrast-enhancement of vertebral bone marrow ± disk space*DDx:*degenerative disk disease type I (no gadolinium-enhancement of disk)

Notes:

Atlanto-occipital Dislocation=ATLANTO-OCCIPITAL DISTRACTION INJURY=disruption of tectorial membrane + paired alar ligaments*Cause:*rapid deceleration with either hyperextension or hyperflexion*Age:*childhood (due to larger size of head relative to body, increased laxity of ligaments, horizontally oriented occipito-atlanto-axial joint, hypoplastic occipital condyles) ● neurologic symptoms: range from respiratory arrest with quadriplegia to normal neurologic exam ● discomfort, stiffness√ retropharyngeal swelling (80%)√ dens-basion distance (BD) >12.5 mm without traction placed on head / neck√ BC/OA ratio >1 = ratio of distance between basion + posterior arch of C1 divided by distance between opisthion + anterior arch of C1



CT: √ blood in region of tectorial membrane + alar ligaments*Cx:*injury to caudal cranial nerves, upper 3 cervical nerves, brainstem, upper part of spinal cord

Notes:

DORSAL DERMAL SINUS
=epithelium-lined dural tube extending from skin surface to intracanalicular space + frequently communicating with CNS / its coverings*Cause:*focal area of incomplete separation of cutaneous ectoderm from neural ectoderm during neurulation*Age:*encountered in early childhood-3rd decade;M:F = 1:1 ● midline dimple / pinpoint ostium ● hyperpigmented patch / hairy nevus / capillary angiomaLocation:lumbosacral (60%), occipital (25%), thoracic (10%), cervical (2%), sacrococcygeal (1%), ventral (8%)CT myelography (best modality to define intraspinal anatomy): √ groove in upper surface of spinous process + lamina of vertebra√ hypoplastic spinous process√ single bifid spinous process√ focal multilevel spina bifida√ laminar defect√ dorsal tenting of dura + arachnoid√ sinus may terminate in conus medullaris / filum terminale / nerve root / fibrous nodule on dorsal aspect of cord / dermoid / epidermoid√ nerve roots bound down to capsule of dermoid / epidermoid cyst√ displacement / compression of cord by extramedullary dermoids / epidermoids√ expansion of cord by intramedullary dermoids / epidermoids√ clumping of nerve roots from adhesive arachnoiditis◊50% of dorsal dermal sinuses end in dermoid / epidermoid cysts!◊20-30% of dermoid cysts / dermoid tumors are associated with dermal sinus tracts!*Cx:*(1)Meningitis (bacterial / chemical)(2)Subcutaneous / epidural / subdural / subarachnoid / subpial abscess (bacterial ascent)◊Dermal sinus accounts for up to 3% of spinal cord abscesses!(3)Compression of neural structures

Notes:

EPIDERMOID OF SPINE

=cystic tumor lined by a membrane composed of epidermal elements of skin*Cause:* (a)congenital dermal rest / focal expansion of dermal sinus(b)acquired from implantation of viable epidermal tissue (by spinal needle without trocar)*Incidence:*1% of spinal cord tumors*Age at presentation:*3rd-5th decade; M > F*May be associated with:*dermal sinus ● slowly progressive myelopathy ● acute onset of chemical meningitis (secondary to rupture of inflammatory cholesterol crystals from cyst into CSF)Location:upper thoracic (17%), lower thoracic (26%), lumbosacral (22%), cauda equina (35%)Site:extramedullary (60%), intramedullary (40%)√ almost always complete spinal block on myelography√ displacement of spinal cord / nerve roots√ small tumors isointense to CSF√ NO contrast enhancement√ CT myelography facilitates detection

Notes:

EPIDURAL HEMATOMA OF SPINE

Etiology:(1) vertebral fracture / dislocation (2) traumatic lumbar puncture (3) hypertension (4) AVM (5) vertebral hemangioma (6) bleeding diathesis / anticoagulation / hemophilia (7) idiopathic (45%)*Peak age:*40-50 years ● acute radicular pain ● paraplegiaLocation:thoracic spine (most common)√ compression of posterior aspect of cord√ high attenuation lesion on CT√ iso- / slightly hypointense lesion on T1WI with marked increase in intensity on T2WI

Notes:

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FRACTURES OF SKULL

1.Linear fracture (most common type)√ deeply black sharply defined line*DDx:*(1)vascular groove, esp. temporal artery (gray line, slightly sclerotic margin, branching like a tree, typical location (temporal artery projects behind dorsum sellae)(2)suture2.Depressed fracture ● often palpable√ bone-on-bone density*Rx:*surgery indicated if depression >3-5 mm (due to arachnoid tear / brain injury)N.B.:CT / MR mandatory to assess extent of underlying brain injury3.Skull-base fracture

LeFort Fracture Sphenoid Bone Fracture Zygomaticomaxillary Fracture Blowout Fracture

Notes:

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LeFort Fracture =all LeFort fractures involve pterygoid processA.LeFort I = Transverse maxillary fracturecaused by blow to premaxilla Fracture line:(a) alveolar ridge(b) lateral aperture of nose(c) inferior wall of maxillary sinus√ detachment of alveolar process of maxillaB.LeFort II = "Pyramidal fracture"Fracture line:arch through(a) posterior alveolar ridge(b) medial orbital rim(c) across nasal bones√ separation of midportion of faceC.LeFort III = "craniofacial disjunction"Fracture line:horizontal course through(a) nasofrontal suture(b) maxillo-frontal suture(c) orbital wall(d) zygomatic arch√ separation of entire face from base of skull

Notes:

Sphenoid Bone Fracture *Incidence:*involved in 15% of skull-base fractures ● CSF rhinorrhea / otorrhea ● hematotympanum ● battle sign = mastoid region ecchymosis ● raccoon eyes = periorbital ecchymosis ● 7th / 8th nerve palsy ● muscular dysfunction: problems with ocular motility, mastication, speech, swallowing, eustachian tube function√ air-fluid level in sinuses + mastoid√ axial thin-slice high-resolution CT for best delineation of fractures√ water-soluble intrathecal contrast material for CSF fistula

Notes:

Zygomaticomaxillary Fracture ="TRIPOD" FRACTURE = MALAR / ZYGOMATIC COMPLEX FRACTURE*Cause:*direct blow to malar eminence ● loss of sensibility of face below orbit ● deficient mastication ● double vision / ophthalmoplegia ● facial deformityFracture line: (a) lateral wall of maxillary sinus (b) orbital rim close to infraorbital foramen (c) floor of orbit (d) zygomatico-frontal suture / zygomatic arch

Notes:

Blowout Fracture =isolated fracture of orbital floor*Cause:*sudden direct blow to globe with increase in intraorbital pressure transmitted to the weak orbital floor, often associated with fracture of the thin lamina papyracea ● diplopia on upward gaze (entrapment of inferior rectus + inferior oblique muscles) ● enophthalmos ● facial anesthesia√ soft-tissue mass extending into maxillary sinus√ complete opacification of maxillary sinus (edema + hemorrhage)√ depression of orbital floor√ posttraumatic atrophy of orbital fat leads to enophthalmos

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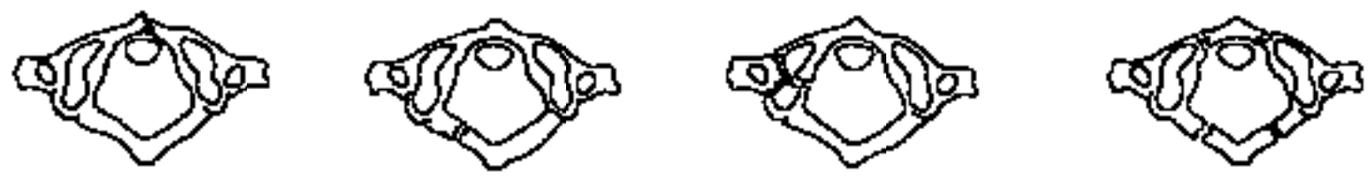
Notes:

FRACTURES OF CERVICAL SPINE

*Frequency:*C2, C6 > C5, C7 > C3, C4 > C1Location: (a)upper cervical spine = C1/2 (19-25%):atlas (4%), odontoid (6%)(b)lower cervical spine = C3-7 (75-81%)(c)multiple noncontiguous spine fractures (15-20%)Site:vertebral arch (50%), vertebral body (30%), intervertebral disk (25%), posterior ligaments (16%), dens (14%), locked facets (12%), anterior ligament (2%)*Associated with:*thoracic / lumbar spine fracture in 5-15%N.B.:Plain radiography misses 20-30% of cervical spine injuries!Most missed fractures involve C1 (8%), C2 (34%), C4 (12%), C6-7 (14%), occipital condyles !



Atlas Fractures

Axis Fractures

Dens Fractures

A.HYPERFLEXION INJURY (46-79%)1.Odontoid fracture2.Simple wedge fracture (stable)3.Teardrop fracture: most severe + unstable injury of C-spine4.Anterior subluxation5.Bilateral locked facets (unstable)6.Anterior disk space narrowing7.Widened interspinous distance8.Spinous process fracture = clay shovelers fracture=sudden load on flexed spine with avulsion fracture of C6 / C7 / T1 (stable) B.HYPEREXTENSION INJURY (20-38%)1.Anteriorly widened disk space2.Prevertebral swelling3.**Teardrop fracture** = avulsion of anteroinferior corner by anterior ligament (unstable) typically at C24.Neural arch fracture of C1 (stable = anterior ring + transverse ligament intact)5.Subluxation (anterior / posterior)6.**Hangmans fracture** = bilateral neural arch fracture of C2 (unstable)√ prevertebral soft-tissue swelling√ anterior subluxation of C2 on C3√ avulsion of anteroinferior corner of C2 (rupture of anterior longitudinal ligament) C.FLEXION-ROTATION INJURY (12%)1.Unilateral locked facets (oblique views!, stable) D.VERTICAL COMPRESSION (4%)1.**Jefferson fracture** = comminuted fracture of ring of C1 (unstable)√ lateral displacement of lateral massa (self-decompressing)(*DDx:* Pseudo-Jefferson fracture = lateral offset of lateral masses of atlas without fracture in fusion anomalies of anterior / posterior arches of C1, in children as lateral masses of atlas ossify earlier than C2) 2.Burst fracture = intervertebral disk driven into vertebral body below (stable)√ several fragments, fragment from posterior superior margin often in spinal canal E.LATERAL FLEXION / SHEARING (4-6%)1.Uncinate fracture2.Isolated pillar fracture3.Transverse process fracture4.Lateral vertebral compression

Significant signs of cervical vertebral trauma Atlas Fracture Axis Fracture

Notes:

Significant signs of cervical vertebral trauma (a)most reliable + specific√ widening of interspinous space (43%)√ widening of facet joint (39%)√ displacement of prevertebral fat stripe (18%)(b)reliable but nonspecific√ wide retropharyngeal space >7 mm (31%)(DDx: mediastinal hemorrhage of other cause, crying in children, S/P difficult intubation) (c)nonspecific√ loss of lordosis (63%)√ anterolisthesis / retrolisthesis (36%)√ kyphotic angulation (21%)√ tracheal deviation (13%)√ disk space: narrow (24%), wide (8%)

Notes:

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Atlas **Fracture** *Incidence:*4% of cervical spine injuriesSite:posterior arch, anterior arch, massa lateralis, Jefferson fracture*Associated with:*fractures of C7 (25%), C2 pedicle (15%), extraspinal fractures (58%)

Notes:

Axis Fracture *Incidence:*6% of cervical spine injuries*Associated with:*fractures of C1 in 8%Type I=avulsion of tip of odontoid (5-8%)√ difficult to detectType II=fracture through base of dens (54-67%)*Cx:*nonunionType III=subdental fracture (30-33%)*Prognosis:* good*DDx:*os odontoideum, ossiculum terminale, hypoplasia of dens, aplasia of dens

Notes:

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FRACTURES OF THORACOLUMBAR SPINE

40% of all vertebral fractures that cause neurologic deficit, mostly complex (body + posterior elements involved)Location:2/3 at thoracolumbar junction√ diastasis of apophyseal joints√ disruption of interspinal ligament√ retropulsion of body fragments into spinal canal√ "burst" fragments at superior surface of body

Fracture of Upper Thoracic Spine (T1 to T10) Fracture of Thoracolumbar Junction (T11 to L2) Chance Fracture

Notes:

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Fracture of Upper Thoracic Spine (T1 to T10) *Types:* 1.compression / axial loading fracture (most common)√ wedging of vertebral body√ retropulsion of bone fragments√ posttraumatic disk herniation2.burst fracture√ associated fracture of posterior neural arch√ comminuted retropulsed bone fragments3.sagittal slice fracture √ vertebra above telescopes into vertebra below, displacing it laterally4.anterior / posterior dislocation√ torn anterior / posterior longitudinal ligament√ facet dislocation√ Relatively stable fractures due to rib cage + strong costovertebral ligaments + more horizontal orientation of facet joints! Signs of spinal instability: =inability to maintain normal associations between vertebral segments while under physiologic load√ displaced vertebra√ widening of interspinous / interlaminar distance√ facet dislocation √ disruption of posterior vertebral body line

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Notes:

Fracture of Thoracolumbar Junction (T11 to L2) =area of transition between a stiff + mobile segment of the spine ● neurologic deficit (in up to 40%)*Classification based on injury to the middle column:* (1)Hyperflexion injury (most common)=compression of anterior column + distraction of posterior spinal elements(a)hyperflexion-compression fracture√ loss of height of vertebral body anteriorly + laterally√ focal kyphosis / scoliosis√ fracture of anterosuperior end plate(b)flexion-rotation injury (unusual)◊Very unstable! ● catastrophic neurologic sequela: paraplegia√ subluxation / dislocation√ widening of interspinous distance√ fractures of lamina, transverse process, facets, adjacent ribs(c)shearing fracture-dislocation=damage of all 3 columns secondary to horizontally impacting force(d)flexion-distraction injury: Chance fracture2.Hyperextension injury (extremely uncommon)√ widened disc space anteriorly√ posterior subluxation√ vertebral anterior superior corner avulsion√ posterior arch fracture3.Axial compression fracture◊Unstable!√ burst fracture with herniation of intervertebral disc through end plates + comminution of vertebral body√ marked anterior vertebral body wedging√ retropulsed bone fragment√ increase in interpediculate distance√ ± vertical fracture through vertebral body, pedicle, lamina

Notes:

Chance Fracture =SEATBELT FRACTURE*Mechanism:*shearing flexion-distraction injury (lap-type seatbelt injury in back-seat passengers) ● neurologic deficit infrequent (20%)Location:L2 or L3√ horizontal splitting of spinous process, pedicles, laminae + superior portion of vertebral body√ disruption of ligaments√ distraction of intervertebral disc + facet joints◊Fracture often unstable!*Often associated with:* (1)bone injuryrib fractures along the course of diagonal strap; sternal fractures; clavicular fractures(2)soft-tissue injurytransverse tear of rectus abdominis muscle; anterior peritoneal tear; diaphragmatic rupture(3)vascular injurymesenteric vascular tear; transection of common carotid artery; injury to internal carotid artery, subclavian artery, superior vena cava; thoracic aortic tear; abdominal aortic transection(4)visceral injuryperforation of jejunum + ileum > large intestine > duodenum (free intraperitoneal fluid in 100%, mesenteric infiltration in 88%, thickened bowel wall in 75%, extraluminal air in 56%); laceration / rupture of liver, spleen, kidneys, pancreas, distended urinary bladder; uterine injury ***Chance Equivalent*** =purely ligamentous disruption leading to lumbar subluxation / dislocation√ mild widening of posterior aspect of affected disk space√ widened facet joints√ splaying of spinous processes = "empty hole sign" on AP view

Notes:

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GLIOMA OF SPINAL CORD

*Often associated with:*syrinx1.Ependymoma (60-70%)Location:lower spinal cord, conus medullaris, filum terminale; extends over several vertebral segments√ well-demarcated / diffusely infiltrating tumor√ occupies whole width of spinal cord√ focal mass with areas of extensive cystic degeneration, hemorrhage, and calcification√ erosion of vertebral body (uncommon)MR: √ intense homogeneous sharply marginated focal enhancement on Gd-enhanced MR√ hypointense tumor margin on T1WI + T2WI2.Astrocytoma (30%)*Histo:*low-grade astrocytoma I and II (75%), high-grade astrocytoma III and IV (8%)Location:cervical + thoracic spine; often extending into lower brainstem√ usually homogeneous extensive cord tumor with widening of spinal cord√ eccentric location within spinal cord√ dilated veins on surface of cord√ mass may be cystic with water-soluble myelographic contrast entering cystic space on delayed CT images√ patchy irregular Gd enhancement on MR

Notes:

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HEMANGIOBLASTOMA OF SPINE
=ANGIOBLASTOMA = ANGIORETICULOMA*Incidence:*2% of all spinal cord tumors; mostly sporadic*Associated with:* von Hippel-Lindau disease (in 1/3)*Age:*middle age; M:F = 1:1Location:intramedullary (75%), radicular (20%), intradural extramedullary (5%); solitary in >90%; mostly in cervicothoracic spine√ increased interpediculate distance (mass effect)√ expanded cord√ intratumoral cystic component (50-60%)√ large draining veins form sinuous mass along posterior aspect of cord√ densely staining tumor nodule√ frequently accompanies syrinxMR: √ well-demarcated Gd-enhancing mass√ curvilinear area of signal void*Cx:*intramedullary hemorrhage

Notes:

KÜMMELL DISEASE
= intravertebral vacuum phenomenon *Cause:*1.Osteonecrosis2.Weeks to months following acute fracture*Pathophysiology:*likely to represent gaseous release into bony clefts within a nonhealed fracture underneath endplate*Age:*>50 yearsLocation:most commonly at thoracolumbar junction↑ gas collection increasing with extension + traction, decreasing with flexion

Notes:

LEPTOMENINGEAL CYST

="Growing" fracture*Incidence:*1% of all pediatric skull fractures*Pathogenesis:*skull fracture with dural tear leads to arachnoid herniation into dural defect; CSF pulsations produce fracture diastasis + erosion of bone margins (apparent 2-3 months after injury)*Age:*usually <3 years√ skull defect with indistinct scalloped margins√ CSF-density cyst adjacent to / in skull, may contain cerebral tissueMR: √ cyst isointense with CSF + communicating with subarachnoid space√ area of encephalomalacia underlying fracture (frequent)√ intracranial tissue extending between edges of bone

Notes:

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LIPOMA OF SPINE
=partially encapsulated mass of fat + connective tissue with connection to leptomeninges / spinal cord Types: (a)intradural lipoma (4%)(b)lipomyelomeningocele (84%)(c)fibrolipoma of filum terminale (12%)Intradural lipomas + lipomyelomeningoceles represent 35% of skin-covered lumbosacral masses + 20-50% of occult spinal dysraphism!

Intradural Lipoma Lipomyelomeningocele Fibrolipoma of Filum Terminale

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Notes:

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Intradural Lipoma =subpial juxtamedullary mass totally enclosed in intact dural sac*Incidence:*<1% of primary intraspinal tumors*Age peaks:*first 5 years of life (24%), 2nd + 3rd decade (55%), 5th decade (16%) ● slow ascending mono- / paraparesis, spasticity, cutaneous sensory loss, defective deep sensation (with cervical + thoracic intradural lipoma) ● flaccid paralysis of legs, sphincter dysfunction (with lumbosacral intradural lipoma) ● overlying skin most often normal ● elevation of protein in CSF (30%)Location:cervical (12%) / cervicothoracic (24%) / thoracic (30%); dorsal aspect of cord (75%), lateral / anterolateral (25%)√ spinal cord open in midline dorsally√ lipoma in opening between lips of placode√ exophytic component at upper / lower pole of lipoma√ syringohydromyelia (2%)√ focal enlargement of spinal canal ± adjacent neural foramina√ narrow localized spina bifida

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Notes:

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Lipomyelomeningocele =lipoma tightly attached to exposed dorsal surface of neural placode blending with subcutaneous fat*Incidence:*20% of skin-covered lumbosacral masses; up to 50% of occult spinal dysraphism*Age:*typically <6 months of age; M < F ● semifluctuant lumbosacral mass with overlying skin intact ● sensory loss in sacral dermatomes, motor loss, bladder dysfunction ● foot deformities, leg painLocation:lumbosacral; longitudinal extension over entire length of spinal canal (in 7%)√ lipoma may enter central canal and extend rostrally(= "intradural intramedullary lipoma")√ lipoma may extend upward within spinal canal external to dura (= "epidural lipoma")√ tethered cord√ large spinal canal√ erosion of vertebral body + pedicles√ posterior scalloping (50%)√ focal spina bifida√ segmental anomalies / butterfly vertebra (up to 43%)√ confluent sacral foramina / partial sacral agenesis (up to 50%)

Notes:

Fibrolipoma of Filum Terminale *Incidence:*6% of autopsies ● asymptomaticLocation:intradural filum, extradural filum, involvement of both portions*Prognosis:*potential for development of symptoms of tethered cord

Notes:

LÜCKENSCHÄDEL
=CRANIOLACUNIA = LACUNAR SKULL = mesenchymal dysplasia of calvarial ossification (developmental disturbance)*Age:*present at birth*Associated with:*(1) meningocele / myelomeningocele (2) encephalocele (3) spina bifida(4) cleft palate (5) Arnold-Chiari II malformation ● normal intracranial pressureLocation:particularly upper parietal area√ honeycombed appearance about 2 cm in diameter (thinning of diploic space)√ premature closure of sutures (turricephaly / scaphocephaly)*Prognosis:*spontaneous regression within first 6 months of life*DDx:*(1)Convolutional impressions = "digital" markings (visible at 2 years, maximally apparent at 4 years, disappear by 8 years of age)(2)"Beaten brass" = "hammered silver" appearance of increased intracranial pressure

Notes:

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MENINGIOMA OF SPINE
*Incidence:*25-45% of all spine tumors; 2-3% of pediatric spinal tumors; 12% of all meningiomas*Age:*>40 years + female (80%)Location:thoracic region (82%); cervical spine on anterior cord surface near foramen magnum (2nd most common location); 90% on lateral aspectSite:intradural extramedullary (50%); entirely epidural; intradural + epidural ● spinal cord / nerve root compression√ bone erosion in <10%√ scalloping of posterior aspect of vertebral body√ widening of interpedicular distance√ enlargement of intervertebral foramen√ may calcify (not as readily as intracranial meningioma)CT: √ solid smoothly marginated mass isodense to skeletal muscle√ marked enhancementMR: √ isointense to grey matter on T1WI + T2WI√ rapid + dense enhancement after Gd-DTPA

Notes:

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METASTASES TO SPINE

Source: (a)Metastatic tumors: breast, prostate, lung, kidney, lymphoma, malignant melanoma(b)Primary tumor: multiple myeloma*Pathogenesis:*hematogenous spread to vertebral bodies (bones with greatest vascularity)MR: √ patchy multifocal relatively well defined lesions√ diminished signal on T1WI + increased signal on T2WI (except for blastic metastases with diminished T1 + T2 signals)*DDx:*(1)Infection (centered around disk space)(2)Primary vertebral tumor (rare in older patients, almost always benign in patients <21 years of age)

Notes:

METASTASES TO SPINAL CORD
Metastases from Outside CNS (a)with subarachnoid hemorrhage: malignant melanoma, choriocarcinoma, hypernephroma, bronchogenic carcinoma(b)others:breast, lymphoma√ predominantly dorsal location√ single / multiple nodules√ thickening of meninges√ matted nerve roots

CSF Seeding of Intracranial Neoplasms

Notes:

CSF Seeding of Intracranial Neoplasms *Age:*occurs more frequently in pediatric age group than in adults*CNS-tumors causing drop metastases:* 1.Medulloblastoma: up to 33%2.Ependymoma: after local recurrence, more common in infra- than supratentorial ependymomas3.Anaplastic glioma4.Germinoma5.Pineoblastoma, pineocytomaLess common:malignant choroid plexus papilloma, angioblastic meningioma *mnemonic:*"MEGO TP"**M**edulloblastoma **E**pendymoma **G**lioblastoma multiforme **O**ligodendroglioma **T**eratoma **P**ineoblastoma Location:lumbosacral + dorsal thoracic spine√ thickened + nodular nerve roots√ nodular + irregularly narrowed thecal sac√ enlarged cord (from coating of outer wall of spinal cord)√ Gd-DTPA enhancement

Notes:

MYELOCYSTOCELE
=SYRINGOCELE=hydromyelic spinal cord + arachnoid herniated through posterior spina bifida; least common form of spinal dysraphism*May be associated with:*GI tract anomalies, GU tract anomalies ● cystic skin-covered mass over spine ● cloacal exstrophy (frequent)Location:lower spine > cervical > thoracic spine√ direct continuity of meningocele with subarachnoid space√ cyst communicating with widened central canal of spinal cord typically posteriorly + inferiorly to meningocele√ lordosis, scoliosis, partial sacral agenesis (common)

Notes:

NEURENTERIC CYST
=incomplete separation of foregut and notochord with persistence of canal of Kovalevski between yolk sac + notochord; cyst connected to meninges through midline defect*Incidence:*rarest of bronchopulmonary foregut malformations (pulmonary sequestration, bronchogenic cyst, enteric cyst)*Associated with:*neurofibromatosis; meningocele; spinal malformation (stalk connects cyst and neural canal; usually no stalk between cyst and esophagus)Location:anterior to spinal canal on mesenteric side of gut√ posterior mediastinal mass√ air-fluid level (if communicating with GI-tract through diaphragmatic defect)√ spinal dysraphism at the same level:√ midline cleft in centra (accommodates stalk)√ anterior / posterior spina bifida√ vertebral body anomalies: absent vertebra, butterfly vertebra, hemivertebra, scoliosis√ diastematomyelia√ thoracic myelomeningocele

Notes:

OSSIFYING FIBROMA
*Peak incidence:*first 2 decades of life*Histo:*areas of osseous tissue intermixed with a highly cellular fibrous tissueSites:maxilla > frontal > ethmoid bone > mandible (rarely seen elsewhere)√ areas of increased + decreased attenuation√ intact inner + outer table√ slow-growing expansile lesion√ usually unilateral + monostotic*DDx:*may be impossible to differentiate from fibrous dysplasia

Notes:

OSTEOMYELITIS OF VERTEBRA

*Incidence:*2-10% of all cases of osteomyelitis*Causes:* (1)direct penetrating trauma (most common); following surgical removal of nucleus pulposus(2)hematogenous: associated with urinary tract infections / following GU surgery / instrumentation; diabetes mellitus; drug abuse*Pathophysiology:*infection begins in low-flow end-vascular arcades adjacent to subchondral plate*Organism:*Staphylococcus aureus, Salmonella*Peak age:*5th-7th decade ● pain in back, neck, chest, abdomen, flank, hip ● neurologic deficit ● fever (most common presenting symptom), leukocytosis ● increased erythrocyte sedimentation rate ● positive blood / urine culture√ disk space narrowing (earliest radiographic sign)√ demineralization of adjacent vertebral endplates√ bulging of paraspinal lines√ tracer uptake in adjacent portions of two vertebral bodies√ decreased marrow signal on T1WI√ iso- / hyperintense marrow signal on T2WI*Cx:*secondary infection of intervertebral disk is frequent*Rx:*>4 weeks course of IV antibiotics*DDx:*discitis

Notes:

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PERINEURAL SACRAL CYST

=TARLOV CYST = cyst arising from posterior rootlets (S2 + S3 most common) = dilated nerve-root sleeve as normal variant√ sacral erosion√ may communicate with thecal sac

Notes:

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SACRAL AGENESIS
=CAUDAL REGRESSION SYNDROME = midline closure defect of neural tube*Incidence:*0.005-0.01%*Predisposed:*infants of diabetic mothers (16%)*Associated with:* (1)musculoskeletal anomalies: hip dislocation, foot deformities, hypoplasia of extremities(2)lack of bladder / bowel control(3)spina bifida (myelomeningocele often not in combination with hydrocephalus) NOT associated with VATER syndrome√ sacral agenesis√ ± dural sac stenosis with high termination√ ± tethered cord with associated lipoma, teratoma, cauda equina cyst*Cx:*neurogenic bladder (if >2 segments are missing)

Notes:

SACROCOCCYGEAL TERATOMA

*Incidence:*1:40,000 livebirths; Type I + II (80%); most common congenital solid tumor in the newborn; M:F = 1:4*Pathogenesis:* (1)growth of residual primitive pluripotential cells derived from the primitive streak + knot (Hensen node) of very early embryonic development(2)attempt at twinning ● increased prevalence of twins in family*Histo:* (1)Mature teratoma (55-75%) with elements from glia, bowel, pancreas, bronchial mucosa, skin appendages, striated + smooth muscle, bowel loops, bone components (metacarpal bones + digits), well-formed teeth, choroid plexus structures (production of CSF)◊MATURE TERATOMA = benign tumor composed of tissues foreign to the anatomic site in which they arise, usually containing tissues from at least 2 germ cell layers(2)Immature teratoma (11-28%): admixed with primitive neuroepithelial / renal tissue◊IMMATURE TERATOMA = benign teratoma with embryonic elements(3)Malignant germ cell tumor(a)mixed malignant teratoma (7-17%): elements of endodermal sinus tumor (= yolk sac tumor) + either form of teratoma(b)pure endodermal sinus tumor (rare)(c)seminoma (dysgerminoma), embryonal carcinoma, choriocarcinoma (extremely rare)Metastases to: lung, bone, lymph nodes (inguinal, retroperitoneal), liver, brain *Age:*50-70% during first few days of life; 80% by 6 months of age; <10% >2 years of age; M:F = 1:4 *Classification (Altman):* Type Ipredominantly external lesion covered by skin with only minimal presacral component (47%)Type IIpredominantly external tumor with significant presacral component (35%)Type IIIpredominantly sacral component + external extension (8%)Type IVpresacral tumor with no external component (10%) *Associated with:*other congenital anomalies (in 18%):(1)musculoskeletal (5-16%): spinal dysraphism, sacral agenesis, dislocation of hip(2)renal anomalies: hydronephrosis, renal cystic dysplasia, Potter syndrome(3)GI tract: imperforate anus, gastroschisis, constipation(4)fetal hydrops (due to high-output cardiac failure)(5)placentomegaly (due to fetal hydrops)(6)curvilinear sacrococcygeal defect (rare autosomal dominant inheritance with equal sex incidence, low malignant potential, absence of calcifications) + anorectal stenosis / atresia, vesicoureteral reflux ● AFP elevated with mixed malignant teratoma + endodermal sinus tumor (CAVE: fetal + newborn serum contains AFP which reaches adult levels not until about 8 months of age) ● premature labor (due to polyhydramnios + large mass) ● uterus large for dates ● radicular pain, constipation, urinary frequency / incontinencePlain film: √ amorphous, punctate, spiculated calcifications, possibly resembling bone (36-50%); suggestive of benign tumor√ soft-tissue mass in pelvis protruding anteriorly + inferiorlyBE: √ anterosuperior displacement of rectum√ luminal constrictionIVP: √ displacement of bladder anterosuperiorly√ development of bladder neck obstructionMyelography: √ intraspinal component may be presentAngio: √ neovascularity (arterial supply by middle + lateral sacral + gluteal branches of internal iliac artery, branches of profunda femoris artery)√ enlargement of feeding vessels√ arterial encasement√ arteriovenous shunting√ early venous filling with serpiginous dilated tumor veinsUS / CT: √ solid (25%) / mixed (60%) / cystic (15%) sacral mass√ 1-30 cm (average size of 8 cm) in diameter√ polyhydramnios (2/3)√ oligohydramnios, fetal hydronephrosis, fetal hydrops with ascites, pleural effusions, skin edema, placentomegaly are poor prognostic factorsMR: √ lobulated + sharply demarcated tumor extremely heterogeneous on T1WI as a result of high signal from fat, intermediate signal from soft tissue, low signal from calcium√ best modality to detect spinal canal invasion *Prognosis:*prevalence of malignant germ cell tumors increases with patient's age◊predominantly fatty tissue tumors are usually benign◊hemorrhage / necrosis is suggestive of malignancy◊cystic lesions are less likely malignant◊sacral destruction indicates malignancy◊patients >2 months of age have a malignant tumor with a 50-90% probability*Cx:*(1)dystocia in 6-13%(2)massive intratumoral hemorrhage(3)fetal death in utero / stillbirth*Rx:*1.Complete tumor resection + coccygectomy + reconstruction of pelvic floor: up to 37% recurrence rate, esp. without coccygectomy2.Multiagent chemotherapy (in malignancy) with long-term survival rate of 50%*DDx:*1.Myelomeningocele (superior to sacrococcygeal region, not septated, axial bone changes)2.Rectal duplication, anterior meningocele (purely cystic)3.Hemangioma, lymphangioma, lipomeningocele, lipoma, epidermal cyst, chordoma, sarcoma, ependymoma, neuroblastoma

Notes:

SPLIT NOTOCHORD SYNDROME
=spectrum of anomalies with persistent connection between gut + dorsal ectoderm*Etiology:*failure of complete separation of ectoderm from endoderm with subsequent splitting of notochord and mesoderm around the adhesion about 3rd week of gestation√ fistula / isolated diverticula / duplication / cyst / fibrous cord / sinus along the tract*Types:* 1.**Dorsal enteric fistula** = fistula between intestinal cavity + dorsal midline skin traversing prevertebral soft tissue, vertebral body, spinal canal, posterior elements of spine ● bowel ostium / exposed pad of mucous membrane in dorsal midline in newborn ● opening passes meconium + feces√ dorsal bowel hernia into a skin- / membrane-covered dorsal sac after passing through a combined anterior + posterior spina bifida2.**Dorsal enteric sinus**=blind remnant of posterior part of tract with midline opening to dorsal external skin surface3.**Dorsal enteric enterogenous cyst**=prevertebral / postvertebral / intraspinal enteric-lined cyst derived from intermediate part of tractIntraspinal enteric cyst *Age at presentation:*20-40 years ● intermittent local / radicular pain worsened by elevation of intraspinal pressureLocation:intraspinal in lower cervical / upper thoracic region√ enlarged spinal canal at site of cyst√ hemivertebrae, segmentation defect, partial fusion, scoliosis in region of cyst4.**Dorsal enteric diverticulum**=tubular / spherical diverticulum arising from dorsal mesenteric border of bowel as a persistent portion of tract between gut + vertebral column5.**Dorsal enteric cyst**=involution of portion of diverticulum near gut ● mass in abdomen / mediastinum (due to bowel rotation)

Notes:

Isthmic Spondylolisthesis = open-arch type *Cause:*usually bilateral spondylolysis=separation of anterior part (vertebral body, pedicles, transverse processes, superior articular facet) from posterior part (inferior facet, laminae, dorsal spinous process)*Age:*often <45 years ● symptomatic if intervertebral disk + posterosuperior aspect of vertebral body encroaches on superior portion of neuroforamen√ elongation of spinal canal in anteroposterior diameter√ bilobed configuration of neuroforamen√ ratio of maximum anteroposterior diameter of spinal canal at any level divided by diameter at L1 >1.25

Notes:

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Degenerative Spondylolisthesis = closed-arch type =PSEUDOSPONDYLOLISTHESIS*Cause:*degenerative / inflammatory joint disease (eg, rheumatoid arthritis)*Pathophysiology:*excess motion of facet joints*Age:*usually >60 years ● commonly symptomatic√ narrowing of spinal canal√ hypertrophy of facet joints√ ratio of maximum anteroposterior diameter of spinal canal at any level divided by diameter at L1 <1.25

Notes:

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Spondylolysis of Cervical Spine =progressive degeneration of intervertebral disks leading to proliferative changes of bone + meninges; more common than disk herniation as a cause for cervical radiculopathy *Incidence:*5-10% at age 20-30; >50% at age 45;>90% by age 60 ● spastic gait disorder ● neck painLocation:C4-5, C5-6, C6-7 (greater normal cervical motion at these levels)*Sequelae:*(a)direct compression of spinal cord(b)neural foraminal stenosis(c)ischemia due to vascular compromise(d)repeated trauma from normal flexion / extension*DDx of myelopathy:* rheumatoid arthritis, congenital anomalies of craniocervical junction, intradural extramedullary tumor, spine metastases, cervical spinal cord tumor, arteriovenous malformation, amyotrophic lateral sclerosis, multiple sclerosis, neurosyphilis

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Notes:

SYRINGOHYDROMYELIA
=SYRINGOMYELIA = SYRINX (used in a general manner reflecting difficulty in classification)=longitudinally oriented CSF-filled cavities + gliosis within spinal cord frequently involving both parenchyma + central canal*Age:*primarily childhood / early adult life ● loss of sensation to pain + temperature (interruption of spinothalamic tracts) ● trophic changes [skin lesions; Charcot joints in 25% (shoulder, elbow, wrist)] ● muscle weakness (anterior horn cell involvement) ● spasticity, hyperreflexia (upper motor neuron involvement) ● abnormal plantar reflexes (pyramidal tract involvement)Location:predominantly lower end of cervical cord; extension into brainstem (= syringobulbia)CT: √ distinct area of decreased attenuation within spinal cord (100%)√ swollen / normal-sized / atrophic cord√ no contrast enhancement√ flattened vertebral border (rare) with increased transverse diameter of cord√ change in shape + size of cord with change in position (rare)√ filling of syringohydromyelia with intrathecal contrast(a)early filling via direct communication with subarachnoid space(b)late filling after 4-8 hours (80-90%) secondary to permeation of contrast materialMyelography: √ enlarged cord (DDx: intramedullary tumor)√ "collapsing cord sign" = collapsing of cord with gas myelography as fluid content moves caudad in the erect position (rare)MR: √ cystic area of low signal intensity on T1WI, increased intensity on T2WI√ presence of CSF flow-void (= low signal on T2WI) within cavity from pulsations√ beaded cavity from multiple incomplete septations√ cord enlargement

Hydromyelia Syringomyelia Reactive Cyst

Notes:

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Hydromyelia =PRIMARY SYRINGOHYDROMYELIA =CONGENITAL SYRINGOHYDROMYELIA=dilatation of persistent central canal of spinal cord (in 70-80% obliterated) which communicates with 4th ventricle (= communicating syringomyelia)*Histo:*lined by ependymal tissue *Associated with:* (1)Chiari malformation in 20-70% metameric haustrations within syrinx on sagittal T1WI(2)Spinal dysraphism(3)Myelocele(4)Dandy-Walker syndrome(5)Diastematomyelia(6)Scoliosis in 48-87%(7)Klippel-Feil syndrome (8)Spinal segmentation defects(9)Tethered cord (in up to 25%)

Notes:

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Reactive Cyst =POSTTRAUMATIC SPINAL CORD CYST= CSF-filled cyst adjacent to level of trauma; usually single (75%) ● late deterioration in patients with spinal cord injury (not related to severity of original injury)*Rx:*shunting leads to clinical improvement

Notes:

TETHERED CORD
=TIGHT FILUM TERMINALE SYNDROME = LOW CONUS MEDULLARIS =abnormally short + thickened filum terminale with low position of conus medullaris*Etiology:*failure of ascent of conus (normal location of tip of conus medullaris: L 4/5 at 16 weeks of gestation, L 2/3 at birth, L1/2 >3 months of age)*Pathophysiology:*mechanical + metabolic + vascular insults with stretching of cord*Age at presentation:*5-15 years (in years of growth spurt); M:F = 2:3*Associated with:*lipoma in 29-78%, diastematomyelia, imperforate anus ● dorsal nevus, dermal sinus tract, hair patch (50%) ● bowel + bladder dysfunction in childhood ● spastic gait with muscle stiffness ● lower extremity weakness + muscle atrophy ● asymmetric hyporeflexia + fasciculations ● orthopedic anomalies: scoliosis, pes cavus, tight Achilles tendon ● hypalgesia, dysesthesia ● paraplegia, paraparesis ● radiculopathy (adults) ● hyperactive deep tendon reflexes ● extensor plantar responses ● anal / perineal pain (in adults) ● back pain (particularly with exertion)√ lumbar spina bifida occulta with interpedicular widening√ scoliosis (20%)√ diameter of filum terminale >2 mm at L5-S1 level (55%), small fibrolipoma within thickened filum (23%), small filar cyst (3%), spinal cord ending in a small lipoma (13%)√ posteriorly located tethered conus medullaris + filum terminale (supine views)√ conus medullaris below level of L2 by age 12 (86%)√ abnormal lateral course of nerve roots (>15° angle relative to spinal cord)√ widened triangular thecal sac tented posteriorly (thecal sac pulled posteriorly by filum)MR: √ prolonged T1 relaxation in center of spinal cord on T1WI in 25% (? myelomalacia / mild hydromyelia)*Rx:*decompressive laminectomy / partial removal of lipoma ± freeing of cord

Notes:

TERATOMA OF SPINE
=neoplasm containing tissue belonging to all 3 germinal layers at sites where these tissues do not normally occur *Incidence:*0.15% (excluding sacrococcygeal teratoma)*Age:*all ages; M:F = 1:1*Path:*solid, thin- / thick-walled partially / wholly cystic with clear / milky / dark cyst fluid, uni- / multilocular, presence of bone / cartilageLocation:intra- / extramedullary√ complete block at myelography√ syringomyelia above level of tumor√ spinal canal may be focally widened

Notes:

MANDIBLE & MAXILLA

Mandibular Hypoplasia = Micrognathia Destruction Of Temporomandibular Joint Radiolucent Lesion Of Mandible Tooth Mass

Notes:

SKULL

Sutural Abnormalities Wormian Bones Increased Skull Thickness Abnormally Thin Skull Osteolytic Lesion Of Skull Lytic Area In Bone Flap Button Sequestrum Absent Greater Sphenoid Wing Absence Of Innominate Line Widened Superior Orbital Fissure Tumors Of The Central Skull Base

Notes:

CRANIOVERTEBRAL JUNCTION

Craniovertebral Junction Anomaly Platybasia

Notes:

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ATLAS AND AXIS

Atlas Anomalies Axis Anomalies Atlantoaxial Subluxation

Notes:

VERTEBRAL BODY

Small Vertebral Body Enlarged Vertebral Body Enlarged Vertebral Foramen Cervical Spine Fusion Vertebral Border Abnormality Bony Projections From Vertebra Vertebral Endplate Abnormality Bullet-shaped Vertebral Body Bone-within-bone Vertebra Ivory Vertebra

Notes:

TUMORS OF VERTEBRA

Expansile Lesion Of Vertebrae Bone Tumors Favoring Vertebral Bodies Primary Tumor Of Posterior Elements

Notes:

INTERVERTEBRAL DISK

Vacuum Phenomenon In Intervertebral Disk Space Intervertebral Disk Calcification Intervertebral Disk Ossification Schmorl Node

Notes:

SPINAL CORD

Intramedullary Lesion Intradural Extramedullary Mass Epidural Extramedullary Lesion Tumors Of Nerve Roots And Nerve Sheaths Cord Lesions Cord Atrophy Delayed Uptake Of Water-Soluble Contrast In Cord lesion Extra-arachnoid Myelography

Notes:

SACRUM

Destructive Sacral Lesion

Notes:

Indications A.InfectionGallium has been largely replaced with WBC imaging but can be used in chronic infection 1.Inflamed / infarcted bowel (eg, Crohn disease) *DDx:*normal bowel excretions (must be cleared by enema; bowel pathology shows persistent activity)2. Diffuse lung uptake sarcoidosis, diffuse infections (TB, CMV, PCP), lymphangitic metastases, pneumoconioses (asbestosis, silicosis), diffuse interstitial fibrosis (UIP), drug-induced pneumonitis (bleomycin, cyclophosphamide, busulfan), acute radiation pneumonitis, recent lymphangiographic contrast 3.Lymph node involvementsarcoidosis, TB, MAI, Hodgkin disease *DDx:*NOT seen in Kaposi sarcoma, a useful distinction in AIDS patients with hilar nodesB.TumorNeoplastic uptake is variable; prominent uptake is usually seen in: 1.Non-Hodgkin lymphoma (especially Burkitt)2.Hodgkin disease3.Hepatoma4.MelanomaUseful in: -detection of tumor recurrence-DDx of focal cold liver lesions on Tc-99m sulfur colloid scan ***No Ga-67 Uptake*** most benign neoplasms; hemangioma; cirrhosis; cystic disease of the breast, liver, thyroid; reactive lymphadenopathy; inactive granulomatous disease

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Notes:

BRAIN ATROPHY

Cerebral Atrophy Cerebellar Atrophy

Notes:

EXTRA-AXIAL LESIONS

Extra-axial Tumor Leptomeningeal Disease Pericerebral Fluid Collection In Childhood

Notes:

VENTRICLES

Ventriculomegaly Colpocephaly Intraventricular tumor

Notes:

PERIVENTRICULAR REGION

Periventricular Hypodensity Enhancing Ventricular Margins Periventricular Calcifications In A Child Periventricular T2WI-hyperintense Lesions

Notes:

HYPODENSE BRAIN LESIONS

Diffusely Swollen Hemispheres Edema Of Brain Brain Herniation Cholesterol-containing CNS Lesions Cyst With A Mural Nodule Midline Cyst Posterior Fossa Cystic Malformation Suprasellar Low-density Lesion With Hydrocephalus Mesencephalic Low-density Lesion Intracranial Pneumocephalus

Notes:

HYPERDENSE INTRACRANIAL LESIONS

Intracranial Calcifications Increased Density Of Falx Intraparenchymal Hemorrhage Dense Cerebral Mass Dense Lesion Near Foramen Of Monro

Notes:

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BRAIN MASSES

Notes:

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ENHANCING BRAIN LESIONS

Gyral Enhancement Ring-enhancing Lesion Of Brain Dense And Enhancing Lesions Multifocal Enhancing Lesions Innumerable Small Enhancing Cerebral Nodules Enhancing Lesion In Internal Auditory Canal

Notes:

VASCULAR DISEASE

Classification Of Vascular CNS Anomalies Occlusive Vascular Disease Displacement Of Vessels

Notes:

BASAL GANGLIA

Bilateral Basal Ganglia Lesions In Childhood Low-attenuation Lesion In Basal Ganglia Basal Ganglia Calcification Linear Echogenic Foci In Thalamus + Basal Ganglia

Notes:

RESPIRATORY DISTRESS

● wheezing (inconsequential) ● bronchoconstriction (life-threatening) ● laryngeal edema (life-threatening)A.MILD□50 mg diphenhydramine□0.3 mL epinephrine (1:1000) SQmay repeat after 15 min up to 1 mL □supplemental oxygen at 2-3 L/minif persistent, □metaproterenol / terbutaline in metered-dose inhalerB.SEVERE(add to the above) □250 mg aminophylline IV over 15-30 min with care*Cx:*hypotension, cardiac arrhythmia□200-400 mg hydrocortisone IVif unsuccessful, may require intubation if anxiety exacerbates bronchospasm, sedation with 5-10 mg Demerol IV

Notes:

SELLA

Destruction Of Sella J-shaped Sella Enlarged Sella Intrasellar Mass Hypointense Lesion Of Sella Parasellar Mass Suprasellar Mass Enhancing Supra- and Intrasellar Mass Perisellar Vascular Lesion

Notes:

PINEAL GLAND

Classification Of Pineal Gland Tumors Intensely Enhancing Mass In Pineal Region

Notes:

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EMBRYOLOGY

Neurulation Brain Growth Neuronal Migration

Notes:

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CEREBRAL VESSELS

Notes:

CEREBELLAR VESSELS

Vertebral Artery Anterior Inferior Cerebellar Artery Posterior Inferior Cerebellar Artery Superior Cerebellar Artery

Notes:

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ABSCESS OF BRAIN

Pyogenic Abscess Granulomatous Abscess

Notes:

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CHIARI MALFORMATION

Chiari I Malformation (adulthood) Chiari II Malformation (childhood) Chiari III Malformation Chiari IV Malformation

Notes:

EMPYEMA OF BRAIN

Subdural Empyema Epidural Empyema

Notes:

Tc-99m DTPA =Tc-99m diethylenetriamine pentaacetic acid=agent of choice for assessment of(1)Perfusion(2)Glomerular filtration = relative GFR(3)Obstructive uropathy(4)Vesicoureteral reflux *Pharmacokinetics:* chelating agent; 5-10% bound to plasma protein; extracted with 20% efficiency on each pass through kidney (= filtration fraction); excreted exclusively by glomerular filtration (similar to inulin) without reabsorption / tubular excretion / metabolism Time-activity behavior: -abdominal aorta (15-20 seconds)-kidneys + spleen (17-24 seconds); liver appears later because of portal venous supply-renal cortical activity (2-4 minutes): mean transit time of 3.0 ± 0.5 minutes; static images of cortex taken at 3-5 minutes-renal pelvic activity (3-5 minutes): peak at 10 minutes; asymmetric clearance of renal pelvis in 50%; accelerated by furosemide *Biologic half-life:*20 minutes*Dose:*10-20 mCi*Radiation dose:*0.85 rads/mCi for renal cortex; 0.6 rads/mCi for kidney; 0.5 rads/mCi for bladder; 0.15 rads/mCi for gonads; 0.15 rads/mCi for whole body Adjunct: Lasix administration (20-40 mg IV) 20 minutes into exam allows assessment of renal pelvic clearance with accuracy equal to Whitaker test (DDx of obstructed from dilated but nonobstructed pelvicalyceal system)

Notes:

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GANGLION CELL TUMOR

Gangliocytoma Ganglioglioma

Notes:

Hydrocephalus A.Normal-pressure hydrocephalus√ reversal of normal CSF flow dynamic = tracer moves from basal cisterns into 4th, 3rd, and lateral ventricles√ loss of w signB.Obstructive hydrocephalus√ delay (up to 48 hours) for tracer to surround convexities + reach arachnoid villi√ positive w sign

Notes:

NEONATAL INTRACRANIAL HEMORRHAGE

Germinal Matrix Bleed Choroid Plexus Hemorrhage Intracerebellar Hemorrhage Intraventricular Hemorrhage Periventricular Leukoencephalopathy

Notes:

Soft-tissue Uptake A.Physiologic 1.Breast2.Kidney: accentuated uptake with dehydration, antineoplastic drugs, gentamicin3.Bowel: surgical diversion of urinary tract B.Faulty preparation with radiochemical impurity(a)free pertechnetate (TcO^{4-})*Cause:*introduction of air into the reaction vial^{99m}Tc activity in mouth (saliva), salivary glands, thyroid, stomach (mucus-producing cells), GI tract (direct secretion + intestinal transport from gastric juices), choroid plexus(b)Tc-99m MDP colloid*Cause:*excess aluminum ions in generator eluate / patient ingestion of antacids; hydrolysis of stannous chloride to stannous hydroxide, excess hydrolized technetium^{99m}Tc diffuse activity in liver + spleen C.Neoplastic conditions(a)Benign tumor1.Tumoral calcinosis2.Myositis ossificans(b)Primary malignant neoplasm1.Extraskeletal osteosarcoma / soft-tissue sarcoma: bone forming2.Neuroblastoma (35-74%): calcifying tumor3.Breast carcinoma4.Meningioma5.Bronchogenic carcinoma (rare)6.Pericardial tumor(c)Metastases with extraosseous activity1.to liver: mucinous carcinoma of colon, breast carcinoma, lung cancer, osteosarcoma*mnemonic:*"LE COMBO"**L**ung cancer **E**sophageal carcinoma **C**olon carcinoma **O**at cell carcinoma **M**elanoma **B**reast carcinoma **O**steogenic sarcoma 2.to lung: 20-40% of osteosarcoma metastatic to lung demonstrate Tc-99m MDP uptake3.Malignant pleural effusion, ascites, pericardial effusion D. Inflammation1.Inflammatory process (abscess, pyogenic / fungal infection):(a)adsorption onto calcium deposits(b)binding to denatured proteins, iron deposits, immature collagen(c)hyperemia2.Crystalline arthropathy (eg, gout)3.Dermatomyositis, scleroderma4.Radiation: eg, radiation pneumonitis5.Necrotizing enterocolitis6.Diffuse pericarditis7.Bursitis8.Pneumonia E.Trauma1.Healing soft-tissue wounds2.Rhabdomyolysis: crush injury, surgical trauma, electrical burns, frostbite, severe exercise, alcohol abuse3.Intramuscular injection sites:especially Imferon (= iron dextran) injections with resultant chemisorption; meperidine4.Ischemic bowel infarction (late uptake)5.Hematoma: soft tissue, subdural6.Heterotopic ossification7.Myocardial contusion, defibrillation, unstable angina pectoris8.Lymphedema F.Metabolic1.Hypercalcemia (eg, hyperparathyroidism):(a)uptake enhanced by alkaline environment in stomach (gastric mucosa), lung (alveolar walls), kidneys (renal tubules)(b)uptake with severe disease in myocardium, spleen, diaphragm, thyroid, skeletal muscle2.Diffuse interstitial pulmonary calcifications: hyperparathyroidism, mitral stenosis3.Amyloid deposits G.Ischemia with dystrophic soft-tissue calcifications=necrosis with dystrophic calcification@Spleen: infarct (sickle cell anemia in 50%), microcalcification secondary to lymphoma, thalassemia major, hemosiderosis, glucose-6-phosphate-dehydrogenase deficiency@Liver: massive hepatic necrosis@Heart: transmural myocardial infarction, valvular calcification, amyloid deposition@Muscle: traumatic / ischemic skeletal muscle injury@Brain: cerebral infarction (damage of blood-brain barrier)@Kidney: nephrocalcinosis@Vessels: calcified wall, calcified thrombus ***Abnormal Uptake Within Kidneys*** 1.Effect of chemotherapeutic drugs:bleomycin, cyclophosphamide, doxorubicin, mitomycin C, 6-mercaptopurine2.S/P radiation therapy3.Metastatic calcification4.Pyelonephritis5.Acute tubular necrosis6.Iron overload7.Multiple myeloma8.Renal vein thrombosis9.Ureteral obstruction ***Abnormal Uptake Within Breast*** 1.Breast carcinoma2.Prosthesis3.Drug-induced ***Abnormal Uptake In Ascitic, Pleural, Pericardial Effusion*** 1.Uremic renal disease2.Infection3.Malignant effusion

Notes:

ORBIT

Spectrum Of Orbital Disorders Intraconal Lesion Extraconal Lesion Orbital Mass In Childhood Mass In Superolateral Quadrant Of Orbit Extraocular Muscle Enlargement

Notes:

GLOBE

Spectrum Of Ocular Disorders Microphthalmia Macrophthalmia Ocular Lesion Vitreous Hemorrhage Dense Vitreous In Pediatric Age Group Retinal Detachment Choroidal Detachment Leukokoria

Notes:

OPTIC NERVE

Optic Nerve Enlargement

Notes:

LACRIMAL GLAND

Lacrimal Gland Lesion Lacrimal Gland Enlargement

Notes:

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ORBITAL CONNECTIONS

Superior Orbital Fissure Inferior Orbital Fissure Optic Canal

Notes:

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ENDOPHTHALMITIS

Infectious Endophthalmitis Sclerosing Endophthalmitis

Notes:

EAR

Hearing Deficit Pulsatile Tinnitus ± Vascular Tympanic Membrane Temporal Bone Sclerosis External Ear Masses Middle Ear Masses Inner Ear Masses

Notes:

SINUSES

Opacification Of Maxillary Sinus Paranasal Sinus Masses Granulomatous Lesions Of Sinuses Hyperdense Sinus Secretions Opacified Sinus & Expansion / Destruction

Notes:

NOSE

Nasal Vault Masses Mass In Nasopharynx

Notes:

PHARYNX

Parapharyngeal Space Mass Pharyngeal Mucosal Space Mass Masticator Space Mass Carotid Space Mass Retropharyngeal Space Mass Prevertebral Space Mass

Notes:

AIRWAYS

Inspiratory Stridor In Children Airway Obstruction In Children Tracheal Tumor

Notes:

LARYNX

Vocal Cord Paralysis Epiglottic Enlargement Aryepiglottic Cyst

Notes:

NECK

Solid Neck Masses In Childhood Lymph Node Enlargement Of Neck Congenital Cystic Lesions Of Neck Branchial Fistula Air-containing Masses Of Neck

Notes:

PAROTID GLAND

Parotid Gland Enlargement Multiple Lesions Of Parotid Gland

Notes:

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THYROID

Congenital Dyshormonogenesis Hyperthyroidism Decreased / No Uptake Of Radiotracer Increased Uptake Of Radiotracer Prominent Pyramidal Lobe Thyroid Calcifications Cystic Areas In Thyroid Thyroid Nodule Discordant Thyroid Nodule Hot Thyroid Nodule Cold Thyroid Nodule

Notes:

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Tc-99m Pertechnetate *Physical decay:*10 mCi Tc-99m decays to 2.7 x 10^{-7} mCi Tc-99*Physical half-life:*2 x 10^{5} years*Biologic half-life:*6 hours*Decay:*by photon emission of 140 keV *Quality control:* (1)<0.1% Mo-99 (= 1 µCi/mCi), maximum of Mo-99 at 5 µCi(2)<0.5 mg aluminum/10 mCi Tc-99m(3)<0.01% radionuclide impurities *Administration:*oral / IV*Dose:*3-5 mCi administered IV 20 minutes prior to imaging (100-300 mrad/mCi) *Pharmacokinetics:* Uptake:in thyroid, salivary glands, gastric mucosa, choroid plexusExcretion:mostly in feces, some in urine *Uptake in thyroid:* 0.5-3.7% at 20 minutes (time of maximum uptake) assessment of trapping function only; NO organification; may be almost completely discharged by perchlorate Comparison to iodine: (a)target-to-background ratio less favorable than with iodine(b)greater photon flux than iodine = detectability of small thyroid lesions (>8 mm) is improved(c)lesions with pertechnetate-iodine discordance (= hot on Tc-99m pertechnetate + cold on radioiodine) are very rare + due to Tc-99m-avid cancer *Imaging:* (a)Collimator: usually with pinhole collimator for image magnification (5-mm hole)(b)Distance: selected so that organ makes up 2/3 of field of view; significant distortion of organ periphery occurs if detector too close(c)Counts: 200,000-300,000 counts are usually acquired within 5 minutes after a dose of 5-10 mCi of Tc-99m pertechnetate(d) Image must include markers for scale + anatomic landmarks + palpatory findings

Notes:

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Deep spaces of suprahyoid head & neck

Pharyngeal mucosal space Parapharyngeal space Retropharyngeal space Prevertebral space Carotid space Parotid space

Notes:

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FRACTURE OF TEMPORAL BONE

Longitudinal Fracture Of Temporal Bone (75%) Transverse Fracture Of Temporal Bone (25%)

Notes:

GOITER

Adenomatous Goiter Diffuse Goiter Iodine-deficiency Goiter Toxic Nodular Goiter Intrathoracic Goiter

Notes:

OTIC CAPSULE DYSPLASIA

Cochlear Aplasia Single-cavity Cochlea Insufficient Cochlear Turns Anomalies Of Membranous Labyrinth Small Internal Auditory Canal Large Vestibule Large Vestibular Aqueduct

Notes:

THYROID ADENOMA

Adenomatous Nodule (42-77%) Follicular Adenoma (15-40%)

Notes:

THYROIDITIS

Hashimoto Thyroiditis DeQuervain Thyroiditis Painless Thyroiditis Acute Suppurative Thyroiditis

Notes:

DENSE LUNG LESION

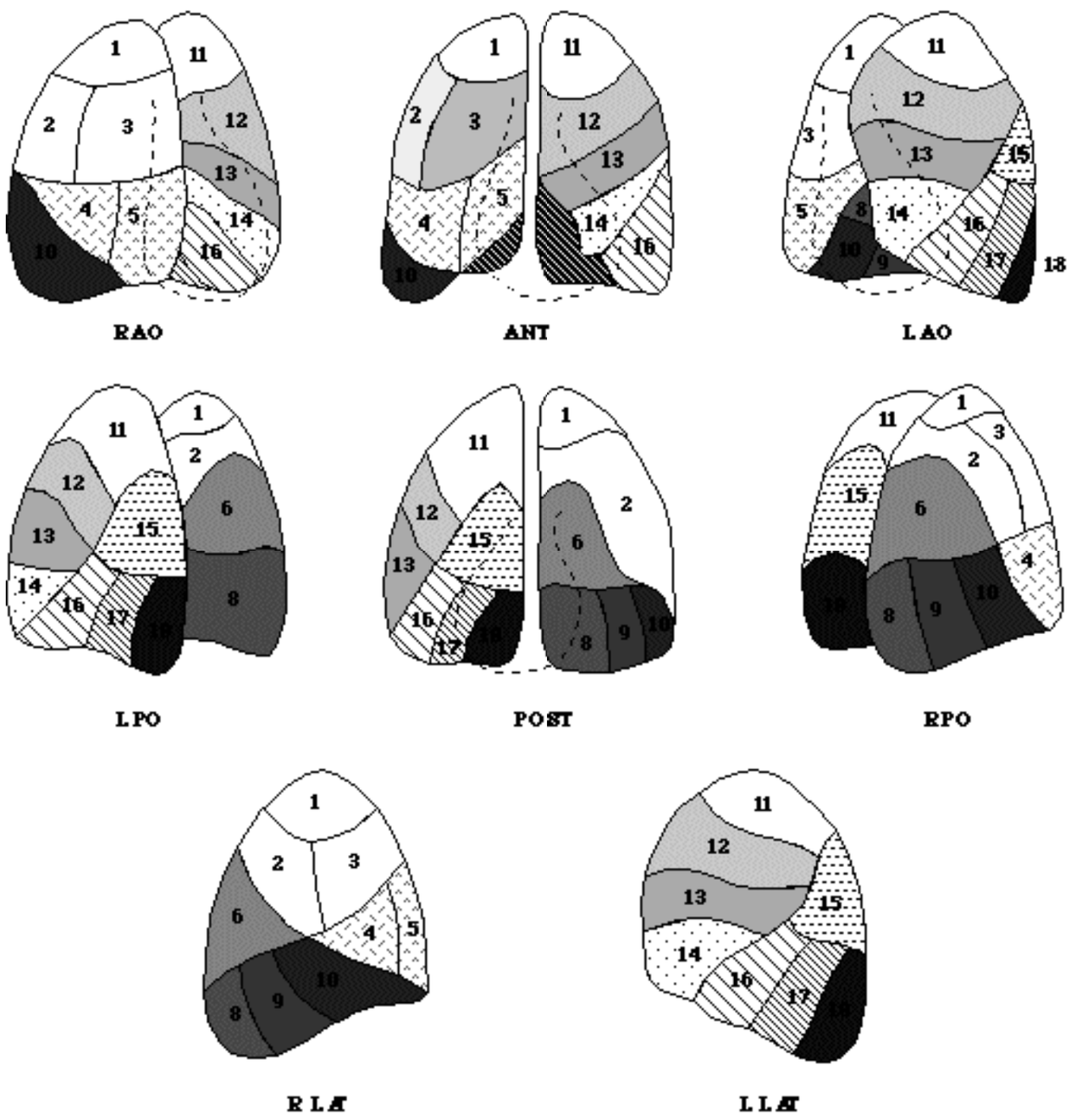
Ground-glass Attenuation Opacification Of Hemithorax Atelectasis Multifocal Ill-defined Densities Diffuse Infiltrates In Immunocompromised Cancer Patient Chronic Infiltrates Ill-defined Opacities With Holes Perihilar "Bat-wing" Infiltrates Peripheral "Reverse Bat-wing" Infiltrates Recurrent Fleeting Infiltrates Tubular Density

Notes:

PULMONARY THROMBOEMBOLISM

Interpretation Criteria for V/Q Lung Scans

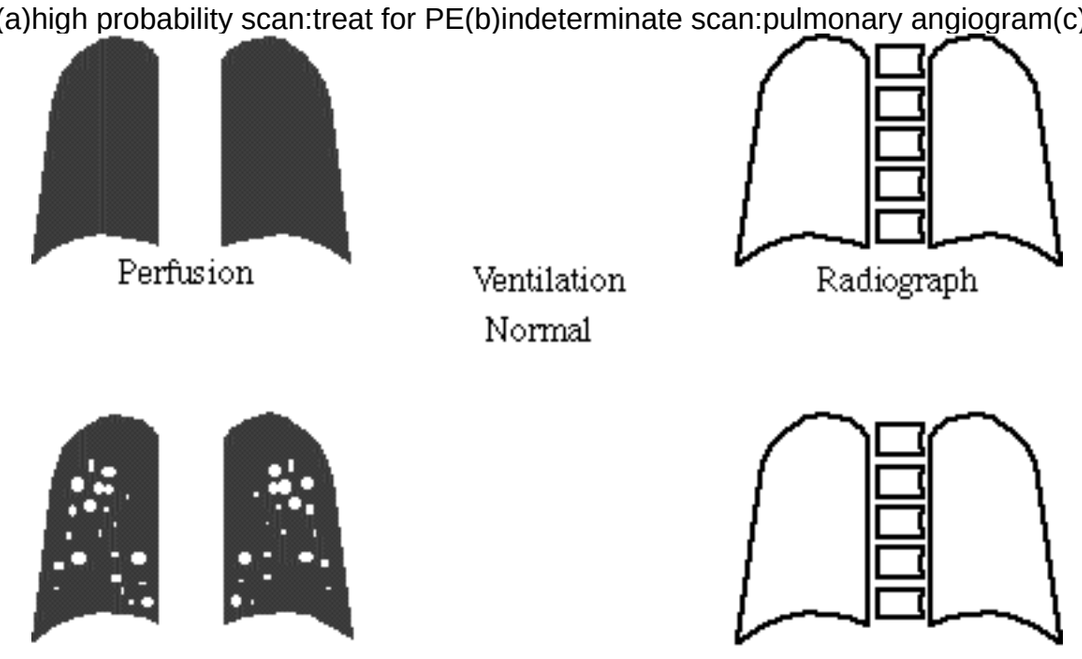
Probability of PE	Biello criteria	PIOPED criteria
Normal	√ normal perfusion	√ normal perfusion
Low (~10%)	√ small (<25% segment) V/Q mismatches √ focal V/Q matches without corresponding CXR abnormality √ perfusion defects substantially smaller than CXR abnormality	√ small perfusion defects regardless of number / ventilation scan finding / CXR finding √ perfusion defect substantially smaller than CXR abnormality; ventilation findings irrelevant √ V/Q match in ≤50% of one lung / ≤75% of upper / mid / lower lung zone; CXR normal / nearly normal √ single moderate perfusion defect with normal CXR; ventilation findings irrelevant √ nonsegmental perfusion defects
Indeterminate (30 – 40%)	√ severe COPD with perfusion defects √ perfusion defect + CXR opacity of same size √ single moderate V/Q mismatch without corresponding CXR abnormality	√ 1 large (segmental) ± 1 moderate (subsegmental) V/Q mismatch √ 1–3 moderate (subsegmental) V/Q mismatches √ 1 matched V/Q with normal CXR
High (~90%)	√ perfusion defects substantially larger than CXR abnormalities √ ≥2 moderate (25–90% segment) / ≥2 large (>90% segment) V/Q mismatches; no corresponding CXR abnormality	√ ≥2 large (segmental) perfusion defects; ventilation scan + CXR findings normal √ >2 large (segmental) perfusion defects substantially larger than matching ventilation / CXR abnormality √ ≥2 moderate (subsegemtal) + 1 large (segmental) perfusion defect; ventilation + CXR findings normal √ ≥4 moderate (subsegmental) perfusion defects; ventilation + CXR findings normal



Lung Segments

RUL RML RLL LUL LLL
1 apical 4 lateral 6 superior 11 apicoposterior 15 superior
2 posterior 5 medial 7 mediobasal 12 anterior 16 anteromedial basal
3 anterior 8 posterobasal 13 superior lingual 17 laterobasal
9 laterobasal 14 inferior lingual 18 posterobasal
10 anterobasal Segmental defect =involves >75% of a known bronchopulmonary segmentSubsegmental defect=involves 25-75% of a known bronchopulmonary segmentV/Q match=abnormal ventilation in region of perfusion defectV/Q mismatch=normal ventilation / normal CXR in region of perfusion defect or perfusion defect larger than ventilation defect / CXR abnormalityPerfusion images will detect: (a)90% of emboli that completely occlude a vessel >1 mm in diameter(b)90% of surface perfusion defects that are larger than 2 x 2 cm(c)26% of emboli that partially occlude a vessel ● A history of prior PE decreases probability of acute embolism because

small V/Q mismatches never resolve!*Therapeutic implications:* (a)high probability scan:treat for PE(b)indeterminate scan:pulmonary angiogram(c)low probability



scan:consider other diagnosis, unless clinical suspicion very high
PIOPED (Prospective Investigation of Pulmonary Embolism Diagnosis) study results:
Probability of PEin angiogrampositive in high13%88%intermediate39%33%low34%16%normal14%9%*Indications for pulmonary angiography:* 1.Embolectomy is a therapeutic option2.Indeterminate V/Q scan with high clinical suspicion + risky anticoagulation therapy3.Specific diagnosis necessary for proper management (vasculitis, drug induced, lung cancer with predominant vascular involvement) *Overall accuracy:* 68% for perfusion scan only, 84% for ventilation-perfusion scan ◊100% sensitivity in detection of PE is due to the occurrence of multiple emboli (usually >6-8), at least one of which causes a perfusion defect!◊A normal perfusion scan virtually excludes PE!◊In an individual <45 years of age a subsegmental perfusion defect + pleuritic chest pain in the same region is indicative of pulmonary embolism in 77%!(DDx: idiopathic / viral pleurisy) ◊73-82% of patients have equivocal perfusion scans (ie, low and intermediate probability)!◊Interobserver variability for intermediate- and low-probability scans is 30%!False-positive scans:nonthrombotic emboli, IV drug abuse, vasculitis, redistribution of flow, acute asthma (due to mucous plugging)False-negative scans:saddle embolus √ associated with normal ventilation scan in >90%√ "stripe sign" = rim of preserved peripheral activity to a perfusion defect usually indicates(a)nonembolic cause(b)old / resolving pulmonary embolism
Correlation with CXR: *CXR category*nondiagnostic V/Q scan
no acute abnormality12%linear atelectasis12%pulmonary edema12%pleural effusion36%parenchymal consolidation82% √ focal lung opacity + not ventilated + not perfused = "indeterminate scan"*Cause:*pneumonia, pulmonary embolism with infarction, segmental atelectasis√ perfusion defect larger than CXR opacity= high probability for PE √ perfusion defect substantially smaller than CXR opacity = low probability for PE√ perfusion defect of comparable size= intermediate probability √ focal lung opacity (not changed >1 week) + not ventilated + not perfused = low probability for PE◊When there is lung opacity, evaluate well-aerated areas for perfusion defects!◊COPD does not diminish usefulness of V/Q scan, but does increase likelihood of an indeterminate result!◊75% of patients with pulmonary edema + without pulmonary embolism have a normal perfusion scan! Influence of clinical estimate:

V/Q scanClinical probabilityPE present
high-probability>80%96%low-probability<20%4%indeterminateDVT present93% Influence of cardiopulmonary disease (CPD):
V/Q probabilitynormal CXRno prior CPDany prior CPDCOPD

high67%93%83%100% intermediate24%39%26%22%low17%15%14%6%near normal3%4%4%0%

Notes:

Gastroesophageal Reflux 89% correlation with acid reflux test *Cause:* (1)Decreased pressure of lower esophageal sphincter(a)transient-complete relaxation of LES(b)low resting pressure of LES(2)Transient increase in intra-abdominal pressure(3)Short intra-abdominal esophageal segment*Age of population:*usually 6-9 months, up to 2 years ● poor weight gain ● vomiting, aspiration, choking ● asthmatic episodes, stridor, apnea *Detection:*upper GI examination with barium, distal esophageal sphincter pressure measurements, 24-hour pH probe measurement in distal esophagus (gold standard), radionuclide examination *Preparation:*4 hours / overnight fasting; abdominal sphygmomanometer (for adults) *Dose:*0.5-1.0 mCi Tc-99m sulfur colloid in 300 mL of acidified orange juice (150 mL juice + 150 mL 0.1 N hydrochloric acid) followed by "cold" acidified orange juice *Imaging:*at 30-60-second intervals for 30-60 minutes, images taken in supine position from anterior; sphygmomanometer inflated at 20, 40, 60, 80, 100 mm Hg *Interpretation:* Reflux (in %) = ([esophageal counts - background] / gastric counts) x 100 √ up to 3% magnitude reflux is normal√ evidence of pulmonary aspiration (valuable in pediatric age group) *Cx:*reflux esophagitis secondary to(a)delayed clearance time of esophageal acid load: tertiary / repetitive esophageal contractions, supine position of refluxor, aspiration of saliva, stimulation of salivary flow, stretched phrenoesophageal membrane in hiatal hernia(b)delayed gastric emptying: increased intragastric pressure (gastric outlet obstruction), viral gastropathy, diabetes *Prognosis:* (1)Self-limiting process with spontaneous resolution by end of infancy (in majority of patients)(2)Persistent symptoms until age 4 (1/3 of patients)(3)Death from inanition / recurrent pneumonia (5%)(4)Cause of recurrent respiratory infections, asthma, failure to thrive, esophagitis, esophageal stricture, chronic blood loss, sudden infant death syndrome (SIDS)*Rx:* (1)Conservative therapy:avoidance of food + drugs that decrease pressure in LES, elevation of head during sleep, acid neutralization, cimetidine / ranitidine (reduction of acid production), metoclopramide / domperidone (increase sphincter pressure + promote gastric emptying) (2)Antireflux surgery

Notes:

PULMONARY MASS

Differential-diagnostic Features Of Lung Masses Benign Lung Tumor Solitary Nodule / Mass Large Pulmonary Mass Cavitating Lung Nodule Shaggy Pulmonary Nodule Hemorrhagic Pulmonary Nodule Multiple Nodules And Masses Pneumoconiosis Classification Pleura-based Lung Nodule Focal Area Of Ground-glass Attenuation Intrathoracic Mass Of Low Attenuation

Notes:

PULMONARY CALCIFICATIONS

Multiple Pulmonary Calcifications Calcified Pulmonary Nodules

Notes:

LUCENT LUNG LESIONS

Hyperlucent Lung Localized Lucent Lung Defect Multiple Lucent Lung Lesions Pulmonary Cyst Multiple Thin-walled Cavities

Notes:

MEDIASTINUM

Mediastinal Shift Pneumomediastinum Mediastinal Fat Acute Mediastinal Widening Mediastinal Mass Low-attenuation Mediastinal Mass Mediastinal Cysts Hilar Mass Eggshell Calcification Of Nodes Enlargement Of Azygos Vein

Notes:

THYMUS

Thymic Mass Diffuse Thymic Enlargement

Notes:

TRACHEA & BRONCHI

Tracheal Tumor Endobronchial Tumor Bronchial Obstruction Mucoid Impaction Signet-ring Sign HRCT Classification Of Bronchiolar Disease Bronchial Wall Thickening Broncholithiasis

Notes:

PLEURA

Pneumothorax Pleural Effusion Hemothorax Solitary Pleural Mass Multiple Pleural Densities Pleural Thickening Apical Cap Pleural Calcification

Notes:

DIAPHRAGM

Bilateral Diaphragmatic Elevation Unilateral Diaphragmatic Elevation

Notes:

CHEST WALL

Chest Wall Lesions Lung Disease With Chest Wall Extension Malignant Tumors Of Chest Wall In Children Pancoast Syndrome

Notes:

NEONATAL LUNG DISEASE

Mediastinal Shift & Abnormal Aeration Reticulogranular Densities In Neonate Hyperinflation In Newborn Hyperinflation In Child

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AIRWAYS

Embryology Of Airways Airway Acinus Primary Pulmonary Lobule Secondary Pulmonary Lobule Surfactant

Notes:

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LUNG FUNCTION

Notes:

ANAPHYLACTOID REACTION

● tachycardia (pulse >100) ● hypotension (systolic blood pressure <80 mm Hg) ● dizziness, diaphoresis ● loss of consciousnessA.MILD□volume expander IV□ 0.2-0.4 mL epinephrine (1:1000) SQB.SEVERE□volume expander IV□1 mL epinephrine (1:10,000) IV up to 3 mL over 5 min (rate of 0.1 mL/min = 10 µg/min)□ oxygen□EKG and central pressure monitor□500 mg hydrocortisone IV□50 mg diphenhydramineif hypotension persists, 5-10 mg/kg/min dopamine IV code team + intensive care unit

Notes:

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DIAPHRAGMATIC HERNIA

Congenital Diaphragmatic Hernia Traumatic Diaphragmatic Hernia

Notes:

Perfusion Defects A.VASCULAR DISEASE(a)Acute / previous pulmonary embolus1.Pulmonary thromboembolic disease2.Fat embolism√ nonsegmental perfusion defect3.Air embolism√ characteristic decortication appearance in uppermost portion on perfusion scintigraphy4.Embolus of tumor / cotton wool / balloon for occlusion of AVM / obstruction by Swan-Ganz catheter, other foreign body5.Dirofilaria immitis (dog heartworm): clumps of heartworms break off cardiac wall + embolize pulmonary arterial tree6.Sickle cell disease (b)Vasculitis1.Collagen vascular disease: sarcoidosis2.IV drug abuse3.Previous radiation therapy:√ defect localized to radiation port4.Tuberculosis (c)Vascular compression1.Bronchogenic carcinoma:√ perfusion defect depending on tumor size + location2.Lymphoma / lymph node enlargement3.Pulmonary artery sarcoma4.Fibrosing mediastinitis due to histoplasmosis5.Idiopathic pulmonary fibrosis:√ small subsegmental defects in both lungs6.Aortic aneurysm (large saccular / dissecting)7.Intrathoracic stomach (d)Altered pulmonary circulation1.Absence / hypoplasia of pulmonary artery2.Peripheral pulmonary artery stenosis3.Bronchopulmonary sequestration4.Primary pulmonary hypertension√ upward redistribution + large hilar defects√ multiple small peripheral perfusion defects5.Pulmonary venoocclusive disease6.Mitral valve disease√ predilection for right middle lobe + superior segments of lower lobes7.Congestive heart failure√ diffuse nonsegmental VQ mismatch√ enlargement of cardiac silhouette + perihilar regions√ reversed distribution: more activity anteriorly than posteriorly√ accentuation of fissures√ flattening of posterior margins of lung (lateral view)√ pleural effusionB.AIRWAY DISEASE◊Nearly all pulmonary disease produces decreased pulmonary blood flow to affected lung zones!1.Asthma, chronic bronchitis, bronchospasm, mucous plugging2.Bronchiectasis (bronchiolar destruction)3.Emphysema (bulla / cyst)4.Pneumonia / lung abscess5.Lymphangitic carcinomatosis√ perfusion defects in area of hypoxia (reflex vasoconstriction)√ abnormal ventilation to a similar / more severe degree√ mostly nonanatomic multiple defects (in 20%)

Notes:

IDIOPATHIC INTERSTITIAL PNEUMONIA

Acute Interstitial Pneumonia Subacute Interstitial Pneumonia Chronic Interstitial Pneumonia

Notes:

MESOTHELIOMA

Benign Mesothelioma Malignant Mesothelioma

Notes:

RADIATION DOSE
Critical organ rad/mCi

I-131Thyroid1,000 I-125Thyroid900 In-111 oxine WBCSpleen26 I-123Thyroid15 In-111 DTPASpinal cord12 Tl-201Kidney1.5 Ga-67 citrateColon1.0 Tc-99m MAALung0.4 Tc-99m albumin microspheresLung0.4 Tc-99m DISIDALarge bowel0.39 Tc-99m sulfur colloidLiver0.33 Yc-99m pertechnetateIntestine0.3 Thyroid0.15 Tc-99m glucoheptonateKidney0.2 Tc-99m pertechnetate (+ perchlorate)Colon0.2 Tc-99m pyrophosphateBladder0.13 Tc-99m phosphateBladder0.13 Tc-99m DTPABladder0.12 Tc-99m-tagged RBCsSpleen0.11 Tc-99m albuminBlood0.015 Xe-133Trachea

Notes:

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BREAST DENSITY

Asymmetric Breast Density Diffuse Increase In Breast Density

Notes:

OVAL-SHAPED BREAST LESION

Mammographic Evaluation Of Breast Masses Well-circumscribed Breast Mass Fat-containing Breast Lesion Breast Lesion With Halo Sign Stellate / Spiculated Breast Lesion Tumor-mimicking Lesions Solid Breast Lesion By Ultrasound

Notes:

NIPPLE and SKIN

Nipple Retraction Nipple Discharge Secretory Disease Skin Thickening Of Breast Axillary Lymphadenopathy

Notes:

REPORTS

Breast Imaging Reporting And Data System (BIRD) Lexicon Descriptors For Reporting (ACR)

Notes:

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BREAST ANATOMY

Lobes Terminal Duct Lobular Unit (TDLU) Components Of Normal Breast Parenchyma Parenchymal Breast Pattern (László Tabár)

Notes:

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QUALITY CONTROL

- Quality control logs should be kept for 3 years!

Notes:

False-negative Ratio =proportion of diseased patients with a normal test result ● D+ column in decision matrix=FN / (TP + FN) = FN / D+=1 - sensitivity = (TP + FN - TP) / (TP + FN)

Notes:

MASTITIS

Puerperal Mastitis Nonpuerperal Mastitis Granulomatous Mastitis

Notes:

CONGENITAL HEART DISEASE

Classification Of CHD Incidence Of CHD In Liveborn Infants CHD With Relatively Long Life Juxtaposition Of Atrial Appendages Continuous Heart Murmur Congestive Heart Failure & Cardiomegaly Congenital Cardiomyopathy Neonatal Cardiac Failure Syndromes With CHD

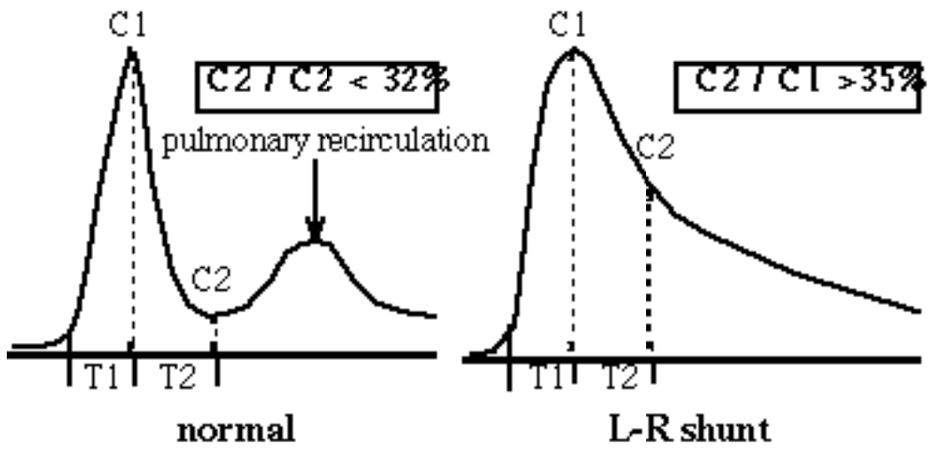
Notes:

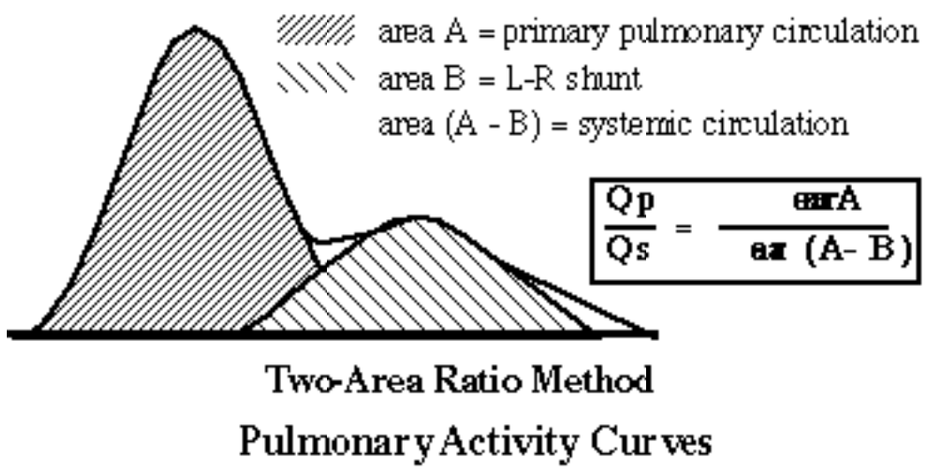
SHUNT EVALUATION

Evaluation Of L-to-R Shunts Abnormal Heart Chamber Dimensions Cardiomegaly In Newborn

Notes:

INTRACARDIAC SHUNTS
Blood-pool agents administered by peripheral IV injection: Tc-99m pertechnetate, DTPA, sulfur colloid, macroaggregated albumin, labeled RBCs *Method:* C2/C1-method measures hemodynamic significance of a shunt; raw data obtained from pulmonary activity curve (gamma variate method, Q_p:Q_s ratio = two-area ratio method, count method); accuracy depends on the shape of the input bolus (single peak of <2 seconds duration); measuring C1, C2, T1, T2





A.NormalC2/C1 is <32%B.L-R shunt*Indication:*ASD, VSD, AV canal, aortopulmonic window, rupture of sinus of Valsalva aneurysm√ C2/C1 >35% (area A = primary pulmonary circulation; area B = L-R shunt; area (A - B) = systemic circulation; Q_P / Q_S = area A / area (A - B) >1.2)C.R-L shunt*Indication:*Tetralogy of Fallot, transposition, truncus, Ebstein anomaly√ early arrival of tracer in left side of heart + aorta (first-pass method) prior to arrival of activity from lungs to LV√ quantification possible only by registration of sum of activity of trapped macroaggregate / microspheres in brain + kidneys *Causes of abnormal nonshunt-related activity:* (1)Radiopharmaceutical breakdown√ free pertechnetate activity in salivary glands, gastric mucosa, thyroid, kidney(2)Hepatic cirrhosisabnormal pulmonary vascular channels bypassing the lung (in 10-70%) (3)Pulmonary AVM

Notes:

ACYANOTIC HEART DISEASE

Increased Pulmonary Blood Flow Without Cyanosis Normal Pulmonary Blood Flow Without Cyanosis

Notes:

PULMONARY VASCULARITY

Increased Pulmonary Vasculature Decreased Pulmonary Vascularity Normal Pulmonary Vascularity & Normal-sized Heart Pulmonary Arterial Hypertension Cor Pulmonale Pulmonary Venous Hypertension Pulmonary Artery-Bronchus Ratios

Notes:

AORTA

Enlarged Aorta Aortic Wall Thickening Double Aortic Arch Right Aortic Arch Left Aortic aArch Bovine Aortic Arch Cervical Aortic Arch Vascular Rings Aortic Stenosis Abnormal Left Ventricular Outflow Tract

Notes:

PULMONARY ARTERY

Invisible Main Pulmonary Artery Unequal Pulmonary Blood Flow Dilatation Of Pulmonary Trunk

Notes:

PERICARDIUM

Pericardial Effusion Pneumopericardium

Notes:

VENA CAVA

Vena cava anomalies IVC Obstruction

Notes:

SURGERY

Surgical Procedures Postoperative Thoracic Deformity Heart Valve Prosthesis

Notes:

STRESS TEST
Rationale: increased heart rate will unveil insufficient regional perfusion secondary to coronary artery disease

Physical Stress Test Pharmacological Stress Test

Notes:

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CARDIOVASCULAR ANATOMY AND ECHOCARDIOGRAPHY

Normal Blood Pressures Development of Major Blood Vessels Right Ventricle Viewed from Front Sweep of Transducer From Aorta Toward Apex

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AORTIC ISTHMUS VARIANTS

Aortic Isthmus Aortic Spindle Ductus Diverticulum

Notes:

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CORONARY ARTERIES

Coronary Artery Collaterals Coronary Artery Dominance Coronary Arteriography

Notes:

EJECTION FRACTION
Ejection fraction (EF) = stroke volume (SV) divided by end-diastolic volume (EDV) stroke volume =end-diastolic volume (EDV) minus end-systolic volume (ESV) **EF = [EDV - ESV] / [EDV]**
= [ED_{counts} - ES_{counts}] / [ED_{counts} - BKG_{counts}] sensitive indicator of left ventricular function *Accuracy in detection of coronary artery disease:* (a)Exercise EF:87% sensitivity; 92% specificity(b)Exercise ECG:60% sensitivity; 81% specificity *Interpretation:* @Left ventricleMean normal value= 67 ± 8% (increase under stress normally >5-7%)Probably abnormal≤55%Definitely abnormal<50%Peak exercise LVEF is an independent predictor of coronary artery disease@Right ventriclemean normal value>45%(RV ejection fraction is smaller than for LV because RV has greater EDV than LV but the same stroke volume) False-positive with(a) inadequate exercise(b) recent ingestion of meal √ EF unchanged / decreased in coronary artery disease√ new regional wall motion abnormality under exercise in coronary artery disease√ correlates well with clinical severity of myocardial infarctionShortcoming: poor study in patients with atrial fibrillation because of inability to achieve adequate cardiac gating (exercise MUGA can yield more sensitive assessment of coronary artery disease)

Notes:

VENOUS SYSTEM OF LOWER EXTREMITY

Deep Veins Of Lower Extremity Superficial Veins Of Lower Extremity Communicating = Perforating Veins Doppler Waveforms of Hepatic Veins

Notes:

Tc-99m Pyrophosphate *Pathophysiology in MYOCARDIAL INFARCTION:* Pyrophosphate is taken up by myocardial necrosis through complexation with calcium deposits >10-12 hours post infarction -requires presence of residual collateral blood flow-30-40% maximum accumulation in hypoxic cells with a 60-70% reduction in blood flow (greater levels of occlusion reduce uptake)*Uptake post infarction:* -earliest uptake by 6-12-24 hours;-peak uptake by 48-72 hours;-persistent uptake seen up to 5-7 days with return to normal by 10-14 days *Sensitivity:*90% for transmural infarction, 40-50% for subendocardial (nontransmural) infarction*Specificity:*as low as 64% *Dose:*15-20 mCi IV (minimal count requirement of 500,000/view)*Imaging:*at 3-6 hours (60% absorbed by skeleton within 3 hours) *Indications:* 1.Lost enzyme pattern = patient admitted 24-48 hours after infarction2.Equivocal ECG + atypical angina:(a)left ventricular bundle branch block(b)left ventricular hypertrophy(c)impossibility to perform stress test(d)patient on digitalis3.ST depression without symptoms4.Equivocal enzyme pattern + equivocal symptoms5.S/P cardiac surgery (perioperative infarction in 10%, enzymes routinely elevated, ECG always abnormal), requires preoperative baseline study as 40% are preoperatively abnormal6.For detection of right ventricular infarction NOT HELPFUL: 1.In differentiating multiple- from single-vessel disease2.Typical angina3.Normal ECG stress test + NO symptoms *Scan interpretation:* [Grade 2+ and above are positive] Grade 0no activityGrade 1+faint uptakeGrade 2+slightly less than sternum, equal to ribsGrade 3+equal to sternumGrade 4+greater than sternum √ "doughnut" pattern = central cold defect (necrosis in large infarct) usually in cases of large anterior + anterolateral wall infarctions√ uptake in inferior wall extending behind sternum (anterior projection) suggests RV infarction√SPECT imaging improves sensitivity (eliminates rib overlap)√ diffuse uptake can be seen in angina, cardiomyopathy, subendocardial infarct, pericarditis and normal blood pool (normal blood pool can be eliminated with delayed imaging) FALSE POSITIVES (10%) A.Cardiac causes1.Recent injury: myocardial contusion, resuscitation, cardioversion, radiation injury, adriamycin cardiotoxicity, myocarditis, acute pericarditis2.Previous injury: left ventricular aneurysm, mural thrombus, unstable angina, previous infarct with persistent uptake3.Calcified heart valves / coronaries (rare) / chronic pericarditis4.Cardiomyopathy: eg, amyloidosisB.Extracardiac causes:1.Soft-tissue uptake: breast tumor / inflammation, chest wall injury, paddle burns from cardioversion, surgical drain, lung tumor2.Osseous: calcified costal cartilage (most common), lesions in rib / sternum3.Increased blood pool activity secondary to renal dysfunction / poor labeling technique (improvement on delayed images) *mnemonic:*"SCUBA"**S**ubendocardial infarction (extensive) **C**ardiomyopathy / myocarditis **U**nstable angina **B**lood pool activity **A**myloidosis FALSE NEGATIVES (5%) Myocardial metastasis PERSISTENTLY POSITIVE SCAN (>2 weeks) =ongoing myocardial necrosis indicating poor prognosis, may continue on to cardiac aneurysm, repeat infarction, cardiac death-in 77% of persistent / unstable angina pectoris-in 41% of compensated congestive heart failure-in 51% of ECG evidence of ventricular dyssynergy *Prognosis:*the larger the area, the worse the mortality + morbidity

Notes:

ANOMALOUS PULMONARY VENOUS RETURN

Total Anomalous Pulmonary Venous Return Partial Anomalous Pulmonary Venous Return = PAPVR

Notes:

CARDIOMYOPATHY

Congestive Cardiomyopathy Hypertrophic Cardiomyopathy Restrictive Cardiomyopathy

Notes:

VENOGRAPHY (1)Foot / calf discomfort or pressure or burning(a)~24% with 60% HOCM(b)~5% with 40% HOCM / 300 mg I/mL LOCM◊The addition of 10-40 mg lidocaine/50 mL of contrast media decreases patient discomfort!(2)Postphlebography deep vein thrombosis(a)26-48% with 60% HOCM(b)0-9% with dilute HOCM / LOCM◊Infusion of 150-200 mL of 5% dextrose in water / 5% dextrose in 0.45% saline / heparinized saline through injection site immediately after examination reduces likelihood of DVT!

Notes:

Disease Prevalence =proportion of diseased subjects to total population=(TP + FN) / (TP + TN + FP + FN) = D+ / total◊Sensitivity + specificity are independent of prevalence◊Affects predictive values + accuracy of a test result *Example:*
Test A: **90%** sensitivity + ***90%*** specificityGOLD STANDARD Tnormalabnormalsubtotal Enormal 90 10 100 Sabnormal10 90 100 T

---

subtotal100100200 NPV = 90% PPV = 90%
Test B: prevalence of **10%**, 90% sensitivity + specificityGOLD STANDARD Tnormalabnormalsubtotal Enormal 1622 164 Sabnormal18 18 36 T

---

subtotal18020 200 NPV = 99% PPV = 50%
Test C: prevalence of **90%**, 90% sensitivity + specificityGOLD STANDARD Tnormalabnormalsubtotal Enormal 18 18 36 Sabnormal2 162164 T

---

subtotal20 180200 NPV = 50% PPV = 99%

Notes:

TRANSPOSITION OF GREAT ARTERIES

Complete Transposition oOf Great Arteries Corrected Transposition Of Great Arteries

Notes:

LIVER

Notes:

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GALLBLADDER

Nonvisualization Of Gallbladder On OCG Nonvisualization Of Gallbladder On US High-density Bile Displaced Gallbladder Alteration In Gallbladder Size Diffuse Gallbladder Wall Thickening Focal Gallbladder Wall Thickening Mobile Intraluminal Mass In Gallbladder Comet-tail Artifact In Liver And Gallbladder Echogenic Fat In Hepatoduodenal Ligament

Notes:

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BILE DUCTS

Gas In Biliary Tree Obstructive Jaundice In Adult Neonatal Obstructive Jaundice Large Nonobstructed CBD Filling Defect In Bile Ducts Bile Duct Narrowing Papillary Stenosis Periampullary Tumor Double-duct Sign Congenital Biliary Cysts

Notes:

PANCREAS

Congenital Pancreatic Anomalies Pancreatic Calcification Fatty Replacement & Atrophy Of Pancreas Pancreatic Mass Pancreatic Neoplasm Hypervascular Pancreatic Tumors Pancreatic Cyst Hyperamylasemia

Notes:

SPLEEN

Notes:

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LIVER

Portal Venous Anatomy Functional Segmental Liver Anatomy Hepatic Arterial Anatomy (Michels classification) Hepatic Fissures Size Of Liver Normal Hemodynamics Parameter Of Liver Liver Tests

Notes:

BILE DUCTS

Normal Size Of Bile Ducts Bile Duct Variants Pancreaticobiliary Junction Variants

Notes:

Congenital Gallbladder Anomalies

Agenesis Of Gallbladder Hypoplastic Gallbladder Septations Of Gallbladder Gallbladder Ectopia

Notes:

PANCREAS

Pancreatic Development & Anatomy

Notes:

CHOLANGIOCARCINOMA

Intrahepatic Cholangiocarcinoma Extrahepatic Cholangiocarcinoma

Notes:

CHOLANGITIS

Notes:

CHOLECYSTITIS

Notes:

ECHINOCOCCAL DISEASE

Echinococcus Granulosus Echinococcus Multilocularis

Notes:

HEPATIC HEMANGIOMA

Cavernous hemangioma of liver Infantile Hemangioendothelioma Of Liver

Notes:

HEPATITIS

Acute Hepatitis Chronic Hepatitis

Notes:

Gastrointestinal Bleeding Detection depends on: (1)Rate of hemorrhage (≥ 0.05 mL/min); NUC more sensitive than angiogram(2)Continuous versus intermittent bleeding (most GI hemorrhages are intermittent)(3)Site of hemorrhage(4)Characteristics of radionuclide agentANGIOGRAPHY: detection requires a bleeding rate of approximately 0.5 mL/min; 63% sensitivity for upper GI bleed; 39% sensitivity for lower GI bleed ***Tc-99m Sulfur Colloid*** *Indication:*bleeding must be active at time of tracer administration; length of active imaging can be increased by fractionating dose-Disappearance half-life of 2.5-3.5 minutes (rapidly cleared from blood by RES + low background activity)-Active bleeding sites detected with rates as low as 0.05-0.1 mL/min-Not useful for upper GI bleeding (interference from high activity in liver + spleen) or bleeding near hepatic / splenic flexure*Dose:*10 mCi (370 MBq) *Imaging:* every image should be for 500,000-1,000,000 counts with oblique + lateral images as necessary (a)every 5 seconds for 1 minute ("flow study"= radionuclide angiogram) (b)60-second images at 2, 5, 10, 15, 20, 30, 40, 60 minutes; study terminated if no abnormality up to 30 minutes(c)delayed images at 2, 4, 6, 12 hours √ extravasation of tracer seen in active bleeding*Specificity:*almost 100% (rare false-positives due to ectopic RES tissue)*False positives:*transplanted kidney, ectopic splenic tissue, modified marrow uptake, male genitalia, arterial graft, aortic aneurysm ***Tc-99m-labeled RBCs*** ***(In Vivtro Labeling Preferred)*** *Indications:*acute / intermittent bleeding (0.35 mL/min)-Remains in vascular system for prolonged period-Liver + spleen activity are low allowing detection of upper GI tract hemorrhage-Low target-to-background ratio (high activity in great vessels, liver, spleen, kidneys, stomach, colon; probably related to free pertechnetate fraction) *Dose:*10-20 mCi*Imaging:*
(a)every 2 seconds for 64 seconds(b)static images for 500,000-1,000,000 counts at 2, 5, and every consecutive 5 minutes up to 30 minutes + every 10 minutes until 90 minutes(c)delayed images at 2, 4, 6, 12 hours up to 36 hours *Localization of bleeding site:* may be difficult secondary to rapid transit time (reduced bowel motility with 1 mg glucagon IV) or too widely spaced time intervals; overall 83% correlation with angiography √ increase in tracer accumulation over time in abnormal location√ bleeding site conforms to bowel anatomy√ change in appearance with time consistent with bowel peristalsis *Sensitivity:* in 83-93% correctly identified bleeding site (50-85% within 1st hour, may become positive in 33% only after 12-24 hours); collection as small as 5 mL may be detected; superior to sulfur colloid -50% sensitivity for blood loss <500 mL/24 hours->90% sensitivity for blood loss >500 mL/24 hours*False positives (5%):* physiologic uptake in stomach + intestine, renal pelvis uptake, hepatic hemangioma, varices, inflammation, isolated vascular process (AVM, venous / arterial graft) *False negatives:* 9% for bleeding of <500 mL/24 hours ***Tc-99m Pertechnetate*** *Indication:*bleeding from functioning gastric mucosa in Meckel diverticulum / intestinal duplication; consider in adults up to age 25; independent of bleeding rate*Pathophysiology:*tracer accumulation in mucus-secreting cells◊Avoid barium GI studies + endoscopy + irritating bowel preparation prior to study!*Dose:*5-10 mCi (185-370 MBq)*Imaging:* (a)radionuclide angiogram 2-3 seconds/frame for 1st minute(b)sequential 5-minute images up to 20 minutes with 500,000-1,000,000 counts per image*Sensitivity:*>80%enhanced by -fasting for 3-6 hours to reduce gastric secretions passing through bowel-nasogastric tube suction to remove gastric secretions-premedication with pentagastrin (6 µg/kg SC 15 minutes before study) to stimulate gastric secretion of pertechnetate-premedication with cimetidine (300 mg qid x 48 hours) to reduce release of pertechnetate from mucosa-voiding just prior to injection *False positives:* Barrett esophagus, duodenal ulcer, ulcerative colitis, Crohn disease, enteric duplication, small bowel, hemangioma, AV malformation, aneurysm, volvulus, intussusception, urinary obstruction, uterine blush *False negatives:* ulcerated epithelium

Notes:

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ABNORMAL INTRA-ABDOMINAL AIR

Abnormal Air Collection Pneumoperitoneum Pseudopneumoperitoneum Pneumoretroperitoneum Pneumatosis Intestinalis Soap-bubble Appearance In Abdomen Of Neonate

Notes:

ABDOMINAL CALCIFICATIONS & OPACITIES

Opaque Material In Bowel Diffuse Abdominal Calcifications Focal Alimentary Tract Calcifications Abdominal Wall Calcifications Abdominal Vascular Calcifications

Notes:

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ABNORMAL INTRA-ABDOMINAL FLUID

Ascites Fluid Collections Intra-abdominal Cyst In Childhood

Notes:

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ESOPHAGUS

Esophageal Contractions Abnormal Esophageal Peristalsis Diffuse Esophageal Dilatation Air Esophagogram Abnormal Esophageal Folds Esophageal Inflammation Esophageal Ulceration Double-barrel Esophagus Esophageal Diverticulum Tracheobronchoesophageal Fistula Long Smooth Esophageal Narrowing Focal Esophageal Narrowing Esophageal Filling Defect Esophageal Mucosal Nodules / Plaques Extrinsic Esophageal Impression

Notes:

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STOMACH

Notes:

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DUODENUM

Extrinsic Pressure Effect On Duodenum Thickened Duodenal Folds Duodenal Filling Defect Duodenal Tumor Enlargement Of Papilla Of Vater Duodenal Narrowing Dilated Duodenum Postbulbar Ulceration

Notes:

SMALL BOWEL

Small Bowel Diverticula Small Bowel Ulcer Separation Of Bowel Loops Normal Small Bowel Folds & Diarrhea Dilated Small Bowel & Normal Folds Abnormal Small Bowel Folds Atrophy Of Folds Ribbonlike Bowel Delayed Small Bowel Transit Multiple Stenotic Lesions Of Small Bowel Small Bowel Filling Defects Small Bowel Tumors

Notes:

CECUM

Ileocecal Valve Abnormalities Coned Cecum Cecal Filling Defect

Notes:

COLON

Colon Cutoff Sign Colonic Thumbprinting Colonic Urticaria Pattern Colonic Ulcers Multiple Bulls-eye Lesions Of Bowel Wall Double-tracking Of Colon Colonic Narrowing Colonic Filling Defects Colonic Polyp

Notes:

RECTUM and ANUS

Rectal Narrowing Enlarged Presacral Space Lesions Of Ischiorectal Fossa

Notes:

PERITONEUM

Peritoneal Mass

Notes:

MESENTERY & OMENTUM

Omental Mass Mesenteric Mass Mesenteric / Omental Cysts Umbilical Tumor

Notes:

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ABDOMINAL LYMPADENOPATHY

Regional Patterns Of Lymphadenopathy Enlarged Lymph Node With Low-density Center

Notes:

ENTEROPATHY

Protein-losing Enteropathy Malabsorption

Notes:

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GASTROINTESTINAL HORMONES

Cholecystokinin Gastrin Glucagon Secretin

Notes:

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ESOPHAGUS

Lower Esophageal Anatomy Muscular Rings Of Esophagus

Notes:

STOMACH

Gastric Cells Effect Of Bilateral Vagotomy Pylorus

Notes:

SMALL BOWEL

Duodenal Segments Small Bowel Folds Normal Bowel Caliber Small Bowel Peristalsis

Notes:

INTESTINAL FUNCTION

Intestinal Gas Intestinal Fluid Defecography / Evacuation Proctography

Notes:

PERITONEUM

Peritoneal Spaces

Notes:

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BLUNT ABDOMINAL TRAUMA
CT is imaging method of choice for evaluation of stable patients

Hemoperitoneum Hypovolemia Blunt Trauma To Spleen Blunt Trauma To Liver (20%) Blunt Trauma To Gallbladder (2%) Blunt Trauma To GI Tract (5%) Blunt Trauma To Pancreas (3%) Blunt Trauma To Kidney Blunt Trauma To Ureteropelvic junction (rare) Blunt Trauma To Bladder

Notes:

ESOPHAGITIS

Acute Esophagitis Candida Esophagitis Caustic Esophagitis Chronic Esophagitis Cytomegalovirus Esophagitis Drug-induced Esophagitis Herpes Esophagitis Human Immunodeficiency Virus Esophagitis Reflux Esophagitis Viral esophagitis

Notes:

Gastric Emptying *Dose:*0.5-1 mCi(a)Tc-99m sulfur colloid cooked with egg white / liver pâté as solid food(b)In-111 DTPA for simultaneous measurement of liquid phase*Imaging:*1-minute anterior abdominal images obtained at 0, 10, 30, 60, 90 minutes in erect position if dual-head camera available; anterior and posterior imaging performed with geometric mean activity calculated*Pharmacokinetics:* 79% tracer activity in stomach for solid phase at 10 minutes; 65% at 30 minutes; 33% at 60 minutes; 10% at 90 minutes *Normal result:*50% of activity in stomach at time zero; should empty by 60 ± 30 minutes√ acutely delayed emptying in stress (pain, cold), drugs (morphine, anticholinergics, levo-dopa, nicotine, b-adrenergic antagonists), postoperative ileus, acute viral gastroenteritis, hyperglycemia, hypokalemia√ chronically delayed gastric emptying in gastric outlet obstruction, postvagotomy, gastric ulcer, chronic idiopathic intestinal pseudoobstruction, GE reflux, progressive systemic sclerosis, dermatomyositis, spinal cord injury, myotonia dystrophica, familial dysautonomia, anorexia nervosa, hypothyroidism, diabetes mellitus, amyloidosis, uremia√ abnormally rapid gastric emptying in gastric surgery, ZE syndrome, duodenal ulcer disease, malabsorption (pancreatic exocrine insufficiency / celiac sprue)

Notes:

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GASTRIC ULCER

Benign Gastric Ulcer Malignant Gastric Ulcer

Notes:

GASTRITIS

Corrosive Gastritis Emphysematous Gastritis Erosive Gastritis Phlegmonous Gastritis

Notes:

HERNIA

External Hernia Internal Hernia Hiatal Hernia Umbilical Hernia

Notes:

LEIOMYOSARCOMA

Leiomyosarcoma Of Small Bowel Leiomyosarcoma Of Stomach Carney Syndrome

Notes:

KIDNEY

Notes:

EXCRETORY UROGRAPHY Clearance:>99% of contrast material eliminated through kidney (<1% through liver, bile, small and large intestines, sweat, tears, saliva); vicarious excretion with renal insult / failure (may be unilateral as in obstructive uropathy)Halftime:1-2 hours (doubled in dialysis patients)Concentration:60% by weight(a)Sodium-containing HOCM↓ less distension of collecting system (b)Meglumine-only HOCM↓ improved distension of collecting system (due to decreased tubular resorption of water)(c)LOCM↓ denser nephrogram + slightly denser pyelogram than HOCM (due to higher tubular concentration)

Notes:

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RENAL MASS

Bilateral Renal Masses Renal Mass In Neonate Renal Mass In Older Child Growth Pattern Of Renal Tumors In Adults Local Bulge In Renal Contour Unilateral Renal Mass Avascular Mass In Kidney Hyperechoic Renal Nodule Hyperattenuating Renal Mass On NECT Low-density Retroperitoneal Mass Focal Area Of Increased Renal Echogenicity Fat-containing Renal Mass Renal Sinus Mass Renal Pseudotumor

Notes:

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RENAL CYSTIC DISEASE

Potter Classification Renal Cystic Disease Syndromes With Multiple Cortical Renal Cysts Multiloculated Renal Mass

Notes:

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ABNORMAL NEPHROGRAM

Notes:

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COLLECTING SYSTEM

Spontaneous Urinary Contrast Extravasation Widened Collecting System & Ureter Caliceal Abnormalities Filling Defect In Collecting System Effaced Collecting System

Notes:

RENAL CALCIFICATION

Retroperitoneal Calcification Calcified Renal Mass Nephrocalcinosis

Notes:

RENOVASCULAR DISEASE

Renovascular Hypertension Renal Aneurysm Spontaneous Renal Hemorrhage Renal Doppler

Notes:

URETER

Ureteral Deviation Megaureter Ureteral Stricture Ureteral Filling Defect

Notes:

ADRENAL GLAND

Adrenal Medullary Disease Adrenal Cortical Disease Bilateral Large Adrenals Unilateral Adrenal Mass Cystic Adrenal Mass Adrenal Calcification

Notes:

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URINARY BLADDER

Bilateral Narrowing Of Urinary Bladder Small Bladder Capacity Bladder Wall Thickening Urinary Bladder Wall Masses Bladder Tumor Bladder Wall Calcification Masses Extrinsic To Urinary Bladder

Notes:

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MALE GENITAL TRACT

Acutely Symptomatic Scrotum Scrotal Wall Thickening Scrotal Gas Scrotal Mass Calcification Of Male Genital Tract Cystic Lesions Of Testis Epididymal Enlargement With Hypoechoic Foci Cystic Lesions Of Epididymis

Notes:

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PROSTATE and URETHRA

Seminal Vesicle Cyst Large Utricle Prostatic Cysts Hypoechoic Lesion Of Prostate Cowper (Bulbourethral) Gland Lesions Urethral Tumors

Notes:

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RENAL ANATOMY

Adult Kidney Renal Size (in cm) Renal Echogenicity Renal Vascular Anatomy Perirenal Compartments

Notes:

RENAL HORMONES

Antidiuretic Hormone (ADH) Renin-aldosterone Mechanism

Notes:

DEVELOPMENTAL RENAL ANOMALIES

Numerary Renal Anomaly Renal Underdevelopment Renal Ectopia

Notes:

ANATOMY OF URETHRA

Male Urethra Female Urethra

Notes:

Iodocholesterol *Agent:*I-131 6-beta-iodomethyl-19-norcholesterol (NP-59); NO FDA approval (available as investigational new drug)*Indications:*adrenocortical imaging(1)ACTH-independent Cushing syndrome (adenoma, cortical nodular hyperplasia)(2)Adrenocortical carcinoma spectrum from nonfunctioning to functioning(3)Primary aldosteronism (adenoma, bilateral adrenal hyperplasia) improved scintigraphic discrimination requires dexamethasone suppression before + during imaging(4)Hyperandrogenism (adrenal adenoma, zona reticularis hyperplasia, polycystic ovary disease, ovarian stromal hyperplasia, androgen-secreting ovarian neoplasm)(5)Incidentaloma (= adrenal mass) localization to side of CT-depicted adrenal mass (= concordant uptake) suggests hyperfunctioning adenoma markedly diminished / absent uptake(= discordant uptake) or symmetric uptake(= nonlateralization) suggests space-occupying mass (eg, cyst) / malignant adrenal mass *Pharmacokinetics:* NP-59 is incorporated into low-density lipoproteins (LDL), circulates to adrenal cortex, absorbed from LDL complex by low-density lipoprotein receptors, esterified in adrenal cortex; adrenocortical uptake affected by adrenocortical secretagogues (corticotropin, angiotensin II); Enterohepatic excretion may obscure adrenals (prior laxative administration beneficial) *Dose:*1 mCi (37 MBq) with slow IV injectionRadiation dose:26 rad/mCi for adrenals, 8.0 rad/mCi for ovaries, 2.4 rad/mCi for liver, 2.3 rad/mCi for testes, 1.2 rad/mCi for whole body *Method:*Lugol solution administered orally (50 mg of iodine per day) for 4-5 days starting the day before injection (to block thyroid uptake of free iodine); mild laxative administered to decrease bowel activity *Imaging:* (a)5-7-day interval between injection + imaging;(b)3-5-day interval between injection + imaging in case of dexamethasone suppression (1 mg four times daily for 7 days prior to and throughout 4-5 days of postinjection imaging interval)

Notes:

Binding Sites (a)fluid spaces1.Transferrin, haptoglobin, albumin, globulins in blood serum2.Interstitial fluid space (increased capillary permeability and hyperemia in inflammation + tumor)3.Lactoferrin in tissue(b)cellular binding1.Viable PMNs incorporate 10% of Ga-67 (bound to lactoferrin in intracytoplasmic granules)2.Nonviable PMNs + their protein exudate (iron-binding proteins are deposited at sites of inflammation; these remove iron from the extracellular space; iron is no longer available for bacterial growth)3.Lymphocytes have lactoferrin-binding surface receptors4.Phagocytic macrophages engulf protein-iron complexes5.Bacteria + fungi (siderophores = lysosomes) have iron-transporting protein mechanism6.Tumor cell-associated transferrin receptor + transportation into cells (lymphocytes bind Ga-67 less avidly than PMNs; RBCs do not bind Ga-67)*mnemonic:*"LFTS"**L**actoferrin (WBCs) **F**erritin **T**ransferrin **S**iderophores (bacteria)

Notes:

EPIDIDYMITIS

Notes:

Tc-99m DMSA =Tc-99m dimercaptosuccinic acid=suitable for imaging of functioning cortical mass: pseudotumor versus lesion*Pharmacokinetics:* high protein-binding + slow plasma clearance; 4% extracted per renal passage; 4-8% glomerular filtration within 1 hour and 30% by 14 hours; 50% of dose accumulates in proximal + distal renal tubular cells by 3 hour (= cortical agent) *Imaging:*after 1- **3**-24 hours (optimal at 34 hours); improved sensitivity to structural defects with SPECT*Biologic half-life:*>30 hours*Dose:*5-10 mCi*Radiation dose:*0.014 rads/mCi for gonads; 0.015 rads/mCi for whole body

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Notes:

POLYCYSTIC KIDNEY DISEASE

Autosomal Dominant Polycystic Kidney Disease Autosomal Recessive Polycystic Kidney Disease

Notes:

RENAL CYST

Simple Cortical Renal Cyst Atypical / Complicated Renal Cyst Renal Sinus Cyst

Notes:

Alternating Sinus =cystic dilatation of urachus periodically emptying into bladder / umbilicus

Notes:

Urachal Diverticulum (3%) =urachus communicates only with bladder dome

Notes:

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URETERAL DUPLICATION
=RENAL DUPLICATION

Complete Duplication Incomplete / Partial Duplication

Notes:

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Iodine-131 *Indication:*thyroid uptake study, thyroid imaging, treatment of hyperthyroidism, treatment of functioning thyroid cancer, imaging of functioning metastases*Production:*by fission decay*Physical half-life*:8.05 days (allows storing for long periods) *Decay:*principal gamma energy of 364 keV (82% abundance) + significant beta decay fraction of a mean energy of 192 keV (92% abundance)*Dose:*30-50 µCi (1.2 rad/µCi = 50 rad for thyroid) *Radiation dose:* (90% from beta decay, 10% from gamma radiation) 0.6 mrad/mCi for whole body; 1.2 mrad/µCi for thyroid (critical organ) *Pharmacokinetics:*identical to I-123*Disadvantages:* (a)Too energetic for gamma camera, well suited for rectilinear scanner with limited resolution(b)High radiation dose prohibits use for diagnostic purposes(c)Ectopic thyroid tissue just as well detectable with I-123 or Tc-99m pertechnetate

---

Notes:

Congenital Urethral Diverticulum *Cause:*ectopic cloacal epithelium; M>F

Notes:

GENERAL OBSTETRICS

Level I Obstetric Ultrasound Level II Obstetric Ultrasound First Trimester Bleeding Positive &b;-HCG Without IUP Dilated Cervix Uterus Large For Dates Empty Gestational Sac Alpha-fetoprotein Use Of Karyotyping

Notes:

PLACENTA

Abnormal Placental Size Vascular Spaces Of The Placenta Placental Tumor Unbalanced Intertwin Transfusion

Notes:

UMBILICAL CORD

Abnormal Cord Attachment Umbilical Cord Lesions

Notes:

FETAL CHEST ANOMALIES

Pulmonary Hypoplasia Intrathoracic Mass Chest Mass Chest Wall Mass Pleural Effusion

Notes:

GYNECOLOGY

Precocious Puberty Amenorrhea Calcifications Of Female Genital Tract Free Fluid In Cul-de-sac

Notes:

PELVIC MASS

Frequency Of Pelvic Masses Cystic Pelvic Masses Complex Pelvic Mass Solid Pelvic Masses Extrauterine Pelvic Masses

Notes:

ADNEXA

Adnexal Masses Ovarian Tumors Ovarian Cyst

Notes:

UTERUS

Postmenopausal Bleeding Thickened Irregular Endometrium Fluid Collection Within Endometrial Canal Endometrial Cysts Diffuse Uterine Enlargement Uterine Masses Fundic Depression On HSG

Notes:

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VAGINA

Vaginal Cyst Vaginal Fistula Vaginal & Paravaginal Neoplasm

Notes:

ANATOMY OF GESTATION

Choriodecidua Gestational Sac Yolk Sac Embryo Amnionic Membrane Umbilical Cord Placental Grading Uteroplacental Circulation

Notes:

ASSESSMENT OF FETAL WELL-BEING

Amniotic Fluid Index Biophysical Profile (Platt and Manning) = BPP Stress Tests

Notes:

INVASIVE FETAL ASSESSMENT

Amniocentesis Chorionic villus sampling (CVS) Cordocentesis

Notes:

UTERUS

Uterine Size Uterine Zonal Anatomy (on T2WI) Endometrium Pelvic Spaces Cervical Length Pelvic Ligaments

Notes:

EMBRYONIC DEMISE
Incidence: 20-71% loss rate of one twin <10 weeks

Early Embryonic Demise / Failing Pregnancy Late Embryonic Demise

Notes:

FETAL CARDIAC DYSRHYTHMIAS
normal heart rate: 120-160 bpm

Premature Atrial Contractions Supraventricular Tachyarrhythmia Atrioventricular Block

Notes:

FETAL HYDROPS

Nonimmune Hydrops Immune Hydrops

Notes:

TABLE OF DOSE, ENERGY, HALF-LIFE, RADIATION DOSE

OrganPharmaceuticalDosekeV$T_{1/2}$ phys$T_{1/2}$ bio

BrainTc-99m pertechnetate10 - 105 mCi1406 h Tc-99m DTPA10 mCi1406 h Tc-99m glucoheptonate10 mCi1406 h Tc-99m Ceretec 20 mCi140 6 h I-123 Spectamine3 - 6 mCi159 13.6 h CSFIn-111 DTPA500 µCi173,2472.8 d Tc-99m DTPA1 mCi1406 h CardiacTl-2011 - 2 mCi**72**,135,16773 h Tc-99m pyrophosphate15 mCi1406 h Tc-99m pertechnetate15 - 25 mCi1406 h Tc-99m-labeled RBCs10 - 20 mCi1406 h Tc-99m sestamibi25 mCi1406 h Tc-99m teboroxime30 mCi1406 h LiverTc-99m sulfur colloid3 - 5 mCi1406 h Tc-99m DISIDA4 - 5 mCi1406 h LungXe-1275 -10 mCi172,203,37536.4 d13 s Xe-13310 - 20 mCi81,1615.3 d20 s Kr-81m20 mCi176,188,19013 s Tc-99m MAA aerosol3 mCi1406 h8 h KidneyTc-99m DTPA15 - 20 mCi1406 h Tc-99m DMSA2 - 5 mCi1406 h Tc-99m glucoheptonate15 - 20 mCi1406 h Tc-99m mercaptoacetyltriglycine10 mCi1406 h I-131 Hippuran250 µCi365*8 d18 min I-123 Hippuran 1 mCi15913.2 h ThyroidTc-99m pertechnetate5 - 10 mCi1406 h I-12350 - 200 µCi15913.2 h I-12530 - 100 µCi27,3560 d I-13130 - 100 µCi365*8 d TestesTc-99m pertechnetate10 mCi1406 h Gastric mucosa Tc-99m pertechnetate50 µCi / kg1406 h GalliumGa-67 citrate3 - 5 mCi88,185,300,3883.3 d WBCIn-111 oxine550 µCi173,2472.8 d Tc-99m Ceretec10 - 20 mCi1406 h

*mnemonic:** = as many days as in a year

Notes:

PEDIATRIC DOSE Actual doses for pediatric patients may vary in different institutions based on empirical data. As rough guidelines use: 1.Clarks rule (body weight):$Dose_{Ped}$=body weight [in lbs] / 150 x $Dose_{Adult}$ 2.Youngs rule (child up to age 12):$Dose_{Ped}$=Age of child / (Age of child + 12) x $Dose_{Adult}$ 3.Surface area:$Dose_{Ped}$=(weight [in kg] $^{0.7}$ / 11) / 1.73 x $Dose_{Adult}$

Notes:

Radionuclide Impurity =amount (µCi) of radiocontaminant per amount (µCi/mCi) of desired radionuclide ***Mo-99 Breakthrough Test:*** (a)allowable contamination of 1:1,000 (= 0.15 µCi Mo-99 per 1 mCi of Tc-99m)(b)<5 µCi Mo-99 per administered dose (NRC dropped this requirement, but nonagreement states may still require this)Measured after lead shielding of vial (filters 140 keV but permits 452 keV of Mo-99 to pass through) Effect of impurity: increased radiation dose, poor image quality

Notes:

Radiochemical Impurity Precise registration of different compounds of Tc-99m, eg -hydrolyzed reduced technetium [$TcO(OH)_2 \bullet H_2O$]-free pertechnetate [$TcO^4]^{-1}$can be monitored by paper chromatography Effect of impurity with hydrolyzed reduced Tc: RES uptake, poor image quality, increased radiation dose

Notes:

Chemical Impurity Chemicals from elution process are restricted in their amount: Tc-99m:<10 µg Al per 1 mL eluate if radionuclide from fission generator;<20 µg Al per 1 mL eluate if radionuclide from neutron bombardment ***Aluminum Ion Breakthrough Test:*** One drop of generator eluate placed on one end of special test paper containing aluminum reagent; equal-sized drop of a standard solution of Al^{3+} (10 ppm) is placed on other end of strip; if color at center of drop eluate is lighter than that of standard solution, the eluate has passed the colorimetric test Effect of impurity: degradation of image quality

Notes:

Pyrogen Testing *USP XX Test* Monitor rectal temperature of 3 suitable rabbits after injection of material through ear vein Acceptable results:no rabbit shows a rise of >0.6°C; total rise of all three <1.4°C ***Limulus Amoebocyte Lysate Test*** (LAL) Highly specific for Gram-negative bacterial endotoxins, sensitivity 10 x greater than USP XX test Amoebocyte = primitive blood cell of horseshoe crab (Limulus polyphebus); lysate formed by hydrolysis of amoebocyte Positive result:in the presence of minute amounts of endotoxin LAL forms an opaque gel; response to other pyrogens (particulate contaminations, chemicals) doubtful

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Notes:

Constancy = Precision
=reproducibility over time*Test frequency:*daily*Method:*measurement of a long-lived source, usually a Cs-137 standard*Evaluation:*measurement must fall within ± 5% of the calculated activity

Notes:

Field Uniformity =ability of camera to reproduce a uniform radioactive distribution = variability of observed count density with a homogeneous flux(a)Integral uniformity=maximum deviation(b)Differential uniformity=maximum rate of change over a specified distance (5 pixels) *Causes for nonuniformity:* (1)High kilovoltage drift of photomultiplier (PM) tubes(2)Physical damage to collimator(3)Improper photopeak setting(4)Contamination *Frequency of quality control:*daily A.INTRINSIC FIELD UNIFORMITY TEST (without collimator) 1.Remove collimator + replace with lead ring (to eliminate edge packing)2.Place a point source at a distance of at least 5 crystal diameters from detector (4-5 feet for small, 7-9 feet for large crystals)3.Point source contains 200-400 µCi of Tc-99m for minimal personnel exposure (avoid contamination of crystal)4.Set count rate below limit of instrument (<30,000 counts)5.Adjust the pulse height selector to normal window settings by centering at 140 keV with a window of 15% (for Tc-99m studies only)6.Use the same photographic device7.Acquire 1.25 million counts for a 10" field of view, 2.5 million counts for a 15" field of view8.Register counts, time, CRT intensity, analyzer settings, initials of controller B.EXTRINSIC FIELD UNIFORMITY TEST(with collimator) 1.Collimator is kept in place◊Only 1 of 2,000 gamma rays that reach the collimator are transmitted to the sodium iodide crystal!2.Sheet source / flood of 2-10 mCi activity is placed on collimator(a)fillable floods: mix thoroughly, avoid air bubbles, check for flat surface(b)nonfillable: commercially available Co-57 source3.Other steps as described above Evaluation: (1)Compare uncorrected with corrected images. Note acquisition time!(2)Store correction flood(3)Rerecord image with corrected flood + check for uniformity

Notes:

Spatial Resolution / Linearity A.SPATIAL RESOLUTION=parameter of scintillation camera that characterizes its ability to accurately determine the original location of a gamma ray on an X,Y plane; measured in both X and Y directions; expressed as full width at half maximum (FWHM) of the line spread function in mm(a)intrinsic spatial resolution(b)system spatial resolutionB.INTRINSIC SPATIAL LINEARITY=parameter of a scintillation camera that characterizes the amount of positional distortion caused by the camera with respect to incident gamma events entering the detector(a)differential linearity = standard deviation of line spread function peak separation (in mm)(b)absolute linearity = maximum amount of spatial displacement (in mm) *Frequency of quality control:*every week 1.Mask detector to collimated field of view (lead ring)2.Lead phantom is attached to front of crystal(a)Four-quadrant bar pattern (3 pictures each after 90° rotation to test entire crystal)(b)Parallel-line equal-spacing (PLES) bar pattern [2 pictures](c)Smith orthogonal hole test pattern (OHP) [1 picture only](d)Hine-Duley phantom [2 pictures]3.Set symmetric analyzer window to width normally used4.Place a point source (1-3 mCi) at a fixed distance of at least 5 crystal diameters from detector on central axis (remove all sources from immediate area so that background count rate is low)5.Acquire 1.25 million counts for a small field, 2.5 million counts for a large field on the same media used for clinical studies6.Record counts, time, CRT intensity, analyzer setting, initials of controller(All new cameras are equipped with a spatial distortion correction circuit) *Evaluation:* Visual assessment of (1)Spatial resolution over entire field(2)Linearity ***Intrinsic Energy Resolution*** =ability to distinguish between primary gamma events and scattered events; performed without collimator; expressed as ratio of photopeak FWHM to photopeak energy (in %) ***CRT-output / Photographic Device*** (1)Check for dirt, scratches, burnt spots on CRT face plates(2)Adjust grey scale + contrast settings to suit film

Notes:

SOURCES OF ARTIFACTS

A. Attenuator between source and detector Materials: cable, lead marker, solder dropped into collimator during repair, belt buckle / watch / key on patient, defective collimator(a)at time of correction flood procedure:√ hot spot(b)after correction flood procedure:√ cold spot B. Cracked crystal √ white band with hot edges C.PMT failure + loss of optical coupling between PMT and crystal√ cold defectD. Problems during film exposure + processing 1.Double exposed film2.Light leak in multiformat camera3.Water lines from film processing4.Frozen shutter:√ part of film cut off5.Variations in film processingE.Improper window setting1.Photopeak window set too high:√ hot tubes2.Photopeak window set too low:√ cold tubesF. Administration of wrong isotope√ atypically imaged organsG. Excessive amounts of free Tc-99m pertechnetate √ too much uptake in choroid plexus, salivary glands, thyroid, stomachH. Faulty Injection Techniqueeg, inadvertently labeled blood clot in syringe leading to iatrogenic pulmonary emboli I. Contamination with radiotracer on patient's skin, stretcher, collimator, crystal J.CRT problems1.Burnt spot on CRT phosphor2.Dirty / scratched CRT face plates

Notes:

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SPECT QUALITY CONTROL
=SINGLE PHOTON EMISSION COMPUTED TOMOGRAPHY=gamma cameras rotating about a pallet supporting the patient obtain 60-120 views over 180° / 360° rotation with typically a field of view of 40-50 cm across the patient and 30-40 cm in axial directionSpatial resolution:~8 mm for high-count study

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Uniformity Center Of Rotation (COR) Sources Of Artifacts

Notes:

LIPPINCOTT WILLIAMS & WILKINS
A Wolters Kluwer Company

Center Of Rotation (COR)
1.Tc-99m-filled line source (5-8 mCi) positioned 3-5 cm off the center of rotation while keeping scanning palette out of field of view2.Direction of rotation to be the same as patient study3.Number of steps (32, 64, or 128) to be the same as in patient study4.Time per step such that at least 100K counts are acquired5.COR must be done with same collimator, orientation, and magnification as patient study*Frequency of quality control*:weekly

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Notes:

Sources Of Artifacts 1.Scanning palette in field of view2.Collimator shifting + rotation on camera face3.Noncircular orbit of camera head4.PM tube failure5.PM tube uncoupling6.Cracked crystal7.Improper peaking of camera

Notes:

POSITRON EMISSION TOMOGRAPHY

= PET = technique that permits noninvasive in vivo examination of metabolism, blood flow, electrical activity, neurochemistry *Concept:* measurement of distribution of a biocompound as a function of time after radiolabeling and injection into patient *Labeling:* PET compounds are radiolabeled with positron-emitting radionuclides *Physics:* positron matter-antimatter annihilation reaction with an electron results in formation of annihilation photons, which are emitted in exactly opposite directions (511 keV each); detected by coincidence circuitry through simultaneous arrival at detectors (bismuth germanate-68) on opposite sides of the patient (= electronic collimation through coincidence circuit); lead collimators not necessary (= advantages in resolution + sensitivity over SPECT); spatial reconstruction similar to transmission CT *Radionuclide production:* in nuclide generator / particle accelerator (positive / negative ion cyclotron; linear accelerator) Expected amount of radionuclide: 500-2,000 mCi Generator characteristics: beam energy (radionuclide production rate increases monotonically with beam energy), beam current (production rate directly proportional to beam current), accelerated particle, shielding requirement, size, cost *Radiopharmaceutical production:* (1)Initialize accelerator, setup(2)Irradiation(3)Synthesis(4)Sterility test, compounding *Sensitivity:* =fraction of radioactive decays within the patient that are detected by the scanner as true events (measured in counts per second per microcurie per milliliter) 30-100 times more sensitive than SPECT (due to electronic collimation as opposed to lead collimation)! *Resolution:* =resolving power = smallest side-by-side objects that can be distinguished as separate objects in images with an infinite number of counts (measured in mm);determined by -distance a positron travels before annihilation occurs (usually 0.5-2 mm depending on energy)-angle variation from 180° (±5° = 0.5 mm)-physical size of detector (1-3 mm) Typical spatial resolution: 4-7 mm *Measurement of radioactivity distribution:* Pixel values proportional to radioactivity per volume Unit:mg of glucose per minute per 100 g tissue*Imaging time:*1-10 min*Organ-specific concentration:* (a)heart, brain: contain little glucose-6-phosphatase resulting in high concentrations of F-18 fluorodeoxyglucose-metabolic rate of glucose is proportional to phosphorylation rate of FDG(b)liver: abundance of glucose-6-phosphatase + low levels of hexokinase resulting in rapid clearing of FDG(c)neoplasm: enhanced glycolysis with increased activity of hexokinase + other enzymes

Isotope		Use	Half-life (min)	Average Positron Energy (keV)	Typical Reaction	Yield at 10 MeV (mCi/μA EOSB)
rubidium	Rb-82		1.23	1,409	Sr/Rb generator	...
fluorine	F-18	glucose metabolism	109	242	O-18(p,n)F-18	120
oxygen	O-15	O_2, H_2O, CO_2, CO	2.1	735	N-15(p,n)O-15	70
nitrogen	N-13	perfusion of NH_3	10	491	C-13(p,n)N-13	110
carbon	C-11	carbon metabolism	20.3	385	N-14(p,α)C-11	85

p = proton injected; n = neutron ejected; α = alpha particle; EOSB = end of saturated bombardment (infinitely long irradiation at which time the numbers of radionuclides produced equals the number of radionuclides that are decaying) per microampere of beam current (= number of particles per second emerging from accelerator and impinging on target material)

PET imaging in oncology

Notes:

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PET imaging in oncology *Pathophysiology:* serum glucose competes with FDG for entry into tumor cells; malignant cells have a high rate of glycolysis 1.Lung cancer √ tumor uptake > mediastinal uptake of FDG (94-97% sensitive, 87-89% specific, 92% accurate)√ FDG can differentiate adrenal "incidentaloma" from metastasis2.Breast cancer3.Colon cancer recurrence4.Lymph node metastases from head and neck cancer (91% sensitive, 88% specific)5.Brain tumor:(a)necrosis versus residual / recurrent tumor√ decreased FDG uptake in necrosis(b)response to chemo- / radiation therapy(c)prediction of patients average survival in pediatric primary brain tumors:≤6 monthsif FDG uptake ≥ gray matter1-2 yearsif FDG uptake > white matter2.5 yearsif FDG uptake = white matter3 yearsif FDG uptake < gray matter6.Pancreatic cancer (96% sensitive + specific)7.Lymphoma staging with whole-body scan

Notes:

IMMUNOSCINTIGRAPHY =imaging with monoclonal antibodies [= homogeneous antibody population directed against a single antigen (eg, cancer cell)], which are labeled with a radiotracer *Hybridoma technique:* antibody-producing B lymphocytes are extracted from the spleen of mice that were immunized with a specific type of cancer cell; B lymphocytes are fused with immortal myeloma cells (= hybridoma)
Agents: Indium-111 satumomab pendetide = indium-111 CYT-103 (OncoScint® CR/OV) = murine monoclonal antibody product derived by site-specific radiolabeling of the antibody B27.3-GYK-DTPA conjugate with indium-111 *Use:*detection + staging of colorectal + ovarian cancers *Dose:*1 mg of antibody radiolabeled with 5 mCi of indium-111 injected IV
*Biodistribution:*liver, spleen, bone marrow, salivary glands, male genitalia, blood pool, kidneys, bladder *Imaging:*2 sets of images 2-5 days post injection + 48 hours apart

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Notes:

GALLIUM-67 CITRATE

Ga-67 acts as an analogue of ferric ion; used as gallium citrate (water-soluble form) *Production:*bombardment of zinc targets (Zn-67, Zn-68) with protons (cyclotron); virtually carrier-free after separation process*Decay:*by electron capture to ground state of Zn-67*Energy levels:* (a) used:93 keV (38%), 184 keV (24%), 300 keV (16%)(b) unused:91 keV (2%), 206 keV (2%), 388 keV (8%) *Physical half-life:*3.3 d (= 78 hours)*Biologic half-life:*2-3 weeks*Adult dose:*3-6 mCi or 50 µCi/kg *Radiation dose:* 0.3 rads/mCi for whole body; 0.9 rads/mCi for distal colon (= critical organ); 0.58 rads/mCi for red marrow; 0.56 rads/mCi for proximal colon; 0.46 rads/mCi for liver; 0.41 rads/mCi for kidney; 0.24 rads/mCi for gonads *Physiology:* Ga-67 is bound to iron-binding sites of various proteins (strongest bond with transferrin in plasma, lactoferrin in tissue); multiexponential + slow plasma disappearance; competitive iron administration (Fe-citrate) enhances target-to-background ratio by increasing Ga-67 excretion

Binding Sites Uptake Excretion Time Of Imaging Normal Variants Of Ga-67 Uptake Indications Gallium In Bone Imaging Gallium In Tumor Imaging Gallium In Lung Imaging Gallium In Renal Imaging Gallium Imaging In Lymphoma Gallium Imaging In Malignant Melanoma

Notes:

Time Of Imaging usually 6, 24, 48, 72 hours◊Best target-to-background ratio generally at 72 hours◊Optimal target-to-background ratio at 6-24 hours for abscess◊Optimal target-to-background ratio at 24-48 hours for tumor ***Degrading Factors Of Imaging*** √ lesions <2 cm are not detectable√ photon scatter within overlying tissues√ physiologic high activity of liver, spleen, bones, kidney, GI tract may obscure lesion

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Notes:

Normal Variants Of Ga-67 Uptake 1.Breasts: increased uptake under stimulus of menarche, estrogens, pregnancy, lactation, phenothiazine medication2.Liver: suppressed uptake by chemotherapeutic agents / high levels of circulating iron / irradiation / severe acute liver disease3.Lung: prominent uptake after lymphangiography4.Spleen: increased uptake in splenomegaly5.Thymus: uptake in children6.Salivary glands: uptake within first 6 months after radiation therapy to neck (may persist for years)7.Epiphyseal plates in children8.Previous steroid therapy, chemotherapy, and radiation therapy may decrease Ga uptake

Notes:

Gallium In Bone Imaging Increased activity in: 1.Active osteomyelitis (90% sensitivity is higher than for Tc-99m MDP)2.Sarcoma3.Cellulitis (bone scan followed by gallium scan)4.Septic arthritis, rheumatoid arthritis5.Paget disease6.Metastases (65% sensitivity, less than for bone agents)

Notes:

Gallium In Tumor Imaging Particularly useful in evaluating extent of known tumor disease + in detection of tumor recurrence A.USEFUL CATEGORY1.Lymphoma(a)Hodgkin disease: 74-88% sensitivity(b)NHL: sensitivity varies-histiocytic form: 85-90% sensitivity-lymphocytic well-diff.: 55-70% sensitivity95% sensitivity for mediastinal disease, 80% sensitivity for cervical + superficial lesions; poor sensitivity below diaphragm 2.Burkitt lymphoma: almost 100% sensitivity3.Rhabdomyosarcoma: >95% sensitivity4.Hepatoma: 85-95% sensitivity5.Melanoma: 69-79% sensitivity B.POSSIBLY USEFUL1.NHL: good for large + mediastinal lesions2.Nodal metastases from seminoma + embryonal cell carcinoma: 87% sensitivity3.Non-small cell lung cancer: 85% sensitivity for primary of any histologic type, 90% probability for uptake in mediastinal nodes, 67% probability for uptake in normal mediastinal nodes, 90% probability for uptake in extrathoracic metastases C.NOT USEFULhead & neck tumors, GI tumors (especially adenocarcinomas; 35-40% sensitivity), breast tumor (52-65% sensitivity), gynecologic tumors (<26% sensitivity), pediatric tumors

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Notes:

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Gallium In Lung Imaging ◊Scans obtained at 48 hours, because 50% of normals show activity at 24 hours A.FOCAL UPTAKE1.Primary pulmonary malignancy (>90% sensitivity)2.Benign disorders: granuloma, abscess, pneumonia, silicosisB.MULTIFOCAL / DIFFUSE UPTAKE(a)Infection1.Tuberculosis√ intense uptake in active lesions (97%)= parameter of activity √ diffuse uptake in miliary TB + rapidly progressive TB pneumonia2.Pneumocystis carinii√ increased uptake at time when physical signs, symptoms, and roentgenographic changes are unimpressive3.Cytomegalovirus(b)Inflammation1.Sarcoidosis70% sensitivity for active parenchymal disease, 94% sensitivity for hilar adenopathy =indicator of therapeutic response to steroids2.Interstitial lung diseasepneumoconiosis, idiopathic pulmonary fibrosis, lymphangitic carcinomatosis 3.Exudative stage of radiation pneumonitis(c)Drugs1.Bleomycin toxicity2.Amiodarone(d)Contrast lymphangiography (in 50%)C.GALLIUM UPTAKE + NORMAL CHEST FILM1.Pulmonary drug toxicity2.Tumor infiltration3.Sarcoidosis4.Pneumocystis carinii

Notes:

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Gallium In Renal Imaging Abnormal uptake on delayed images at 48-72 hours A.Renal tumor1.Primary renal tumor (variable uptake)2.Lymphoma / leukemia3.Metastases (eg, melanoma)B.Renal inflammation1.Acute pyelonephritis (88% sensitivity):√ diffuse / focal uptake 2.Lobar nephronia3.Renal abscessC.Others1.Collagen-vascular disease, vasculitis, Wegener granulomatosis2.Amyloidosis, hemochromatosis3.Hepatic failure4.Administration of antineoplastic drugsD.Transplant1.Acute / chronic rejection2.Acute tubular necrosisE.Urinary bladder1.Cystitis2.Tumor *mnemonic:*"CHANT An OLD PSALM"**C**hemotherapy **H**emochromatosis, **H**epatorenal failure **A**cute tubular necrosis, **A**cute lobar nephronia **N**eoplasm **T**ransfusion, **T**uberous sclerosis **A**bscess **O**bstruction **L**ymphoma **D**rugs (Fe, drugs causing ATN) **P**yelonephritis, **P**olyarteritis nodosa **S**arcoidosis **A**myloidosis, **A**llograft **L**eukemia **M**etastasis, **M**yeloma

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Notes:

Gallium Imaging In Lymphoma A.Hodgkin disease50-70% average sensitivity dependent on size, location, technique B.Non-Hodgkin lymphoma30% sensitivity for lymphocytic subtype, 70% sensitivity for histiocytic subtype *Sensitivity:* 90% for mediastinal nodes 80% for neck nodes 48% for periaortic nodes 47% for iliac nodes 36% for axillary nodes

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Notes:

Gallium Imaging In Malignant Melanoma *Types:* 1.Lentigo maligna: low invasiveness, low metastatic potential2.Superficial spreading melanoma: intermediate prognosis3.Nodular melanoma: most lethal *Prognosis (level of invasion versus 5-year survival):* LevelI(in situ)100%LevelII(within papillary dermis)100%LevelIII(extending to reticular dermis)88%LevelIV(invading reticular dermis)66%LevelV(subcutaneous infiltration)15% Ga-67: >50% sensitivity for primary + metastatic sites; detectability versus tumor size: 73% sensitivity >2 cm; 17% sensitivity <2 cm Bone, brain, liver scintigraphy: show very low yield in detecting metastases at time of preoperative assessment and are not indicated

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Notes:

AGENTS FOR INFLAMMATION
1.**Ga-67 citrate**
overall 58-100% sensitivity; 75-100% specificity (lower for abdominal inflammation because of problematic abdominal activity) *Indication:* chronic + nonpyogenic inflammation, pulmonary infection + lymphadenitis with HIV-positivity, granulomatous disease (eg, sarcoidosis) *Pathophysiology:* leakage of protein-bound Ga-67 into extracellular space secondary to hyperemia + increased capillary permeability; Ga-67 is preferentially bound to nonviable PMNs + macrophages 1.Leukocyte incorporation (rich in lactoferrin)2.Bacterial uptake (iron-chelating siderophores)3.Inflammatory tissue stimulates lactoferrin production GALLIUM IN CHRONIC ABDOMINAL INFLAMMATION 67% sensitivity, 64% specificity, 13% false-negative rate, 5% false-positive rate *Dose:*5 mCi*Imaging:*routine at 48-72 hours (after clearance of high background activity); optional at 6-24 hours (prior to renal + gastrointestinal excretion); delayed images as needed√ diffuse uptake in peritonitis√ localized uptake in acute pyogenic abscess, phlegmon, acute cholecystitis, acute pancreatitis, acute gastritis, diverticulitis, inflammatory bowel disease, surgical wound, pyelonephritis, perinephric abscess 2.**In-111-labeled WBC**
=In-111-oxine-labeled autologous leukocytes with 80% sensitivity; 97% specificity, 91% accuracy (superior to Ga-67 citrate); no activity in intestinal contents / urine*Indication:* occult sepsis, acute pyogenic infection, abdominal + renal abscess, inflammatory bowel disease, nonpulmonary infection with HIV positivity, prosthetic graft infection (bone / cardiovascular graft), acute + chronic + complicated bone / joint infection *Technique:* harvesting of cells followed by separation from RBCs and platelets + washing off plasma proteins; chelating agents (oxine = 8-hydroxyquinoline / tropolone) used for labeling; lipophilic oxine-indium complex penetrates cell membrane of white cells; intracellular proteins scavenge the indium from oxine; oxine diffuses out from cell; requires 2 hours of preparation time Recovery rate:30% at 1-4 hours after injection*Limitations:*19 gauge IV access, leukopenia, impaired chemotaxis, abnormal WBCs, children*Dose:*0.5 mCi*Half-life:* 67 hoursUseful photopeaks: 173 keV (89%), 247 keV (94%) *Radiation dose:* 13-18 rad/mCi for spleen; 3.8 rad/mCi for liver; 0.65 rad/mCi for red marrow; 0.45 rad/mCi for whole body; 0.29 rad/mCi for testes; 0.14 rad/mCi for ovaries (compared with Ga-67 higher dose to spleen, but lower dose to all other organs) *Biodistribution:*spleen, liver, bone marrow; blood clearance halftime of 6-7 hours*Imaging:* best at 18-24 hours following injection of cell preparation; optional at 2-6 hours (eg, in inflammatory bowel disease); delayed images as needed; bone marrow uptake provides useful landmarks √ focal activity greater than in spleen is typical for abscess (comparison based on liver, spleen, bone marrow activity)√ activity equal to liver (significant inflammatory focus)√ abdominal activity is always abnormal *False positives:* @Chest: CHF, RDS, embolized cells, cystic fibrosis@Abdomen: accessory spleen, colonic accumulation, renal transplant rejection, GI hemorrhage, vasculitis, ischemic bowel disease, following CPR, uremia, postradiation therapy, Wegener granulomatosis, ALL@Miscellaneous: IM injection, histiocytic lymphoma, cerebral infarction, arthritis, skeletal metastases, thrombophlebitis, hematoma, hip prosthesis, cecal carcinoma, postsurgical pseudoaneurysm, necrotic tumors that harvest WBCs*False negatives:* chronic infection, aortofemoral graft, LUQ abscess, infected pelvic hematoma, splenic abscess, hepatic abscess (occasionally)

3.**Tc-99m-labeled WBC**
*Optimal use:*osteomyelitis in extremities *Advantages over In-111 WBC imaging:* (a)improved photon flux(b)earlier imaging *Disadvantages:* (1)Biliary excretion leads to bowel activity, which may obscure abdominal abscess if not imaged early(2)Heart and blood pool may obscure disease(3)Nonspecific accumulation in lung *Technique:* Tc-99m Ceretec binds with autologous WBCs and is reinjected *Imaging:* 30 minutes (optimum for use in abdomen), 60 minutes, 3-4 hours, 24 hours (optional) *False positives:* may be due to unusual marrow distribution, correlation with bone marrow (sulfur colloid) scan may be necessary

Notes:

BONE AGENTS
A.POLYPHOSPHATES = LINEAR PHOSPHATES= CONDENSED PHOSPHATES First agents described; contain up to 46 phosphate residues; simplest form contains 2 phosphates = pyrophosphate (PYP) B.DIPHOSPHONATESOrganic analogs of pyrophosphate characterized by P-C-P bond; chemically more stable; not susceptible to hydrolysis in vivo; most widely used agents: 1.ethylene hydroxydiphosphonate (EHDP)= ethane-1-hydroxy-1,1-diphosphonate 2.methylene diphosphonate (MDP)C. IMIDODIPHOSPHONATES (IDP) Characterized by P-N-P bond *Indications:* 1.Imaging of bone, myocardial / cerebral infarct, ectopic calcifications, some tumors (neuroblastoma)2.Rx for Paget disease, myositis ossificans progressiva, calcinosis universalis (inhibits formation + dissolution of hydroxyapatite crystals) *Usual dose:*20 mCi (740 MBq)*Radiation dose:*0.13 rad/mCi for bladder (critical organ), 0.04 rad/mCi for bone, 0.01 rad/mCi for whole body *Imaging:* @Bone: 2-3 hours post injection◊ Fractures may not show positive uptake until 3-10 days depending on age of patient@Myocardium: 90-120 minutes post injection◊Ideal imaging time is 1-3 days post infarction *Labeling:*Tc (VII) is eluted as a pertechnetate ion; chemical reduction with Sn (II) chloride; chelated into a complex of Tc-99m (IV)-tin-phosphate *Quality Control:* (1)<10% Tc-99m tin colloid / free Tc-99m pertechnetate (a good preparation is 95% bound)(2)Agent should not be used prior to 30 minutes after preparation(3)Avoid injection of air in preparation of multidose vials (oxidation results in poor Tc bond)(4)Kit life is 4-5 hours after preparation *Uptake:* (a)rapid distribution into ECF (78% of injected dose with biologic half-life of 2.4 minutes) directly related to blood flow + vascularity; blood clearance rate determines ECF (= background) activity (at 4 hours 1% for diphosphonates, 5% for pyrophosphate / polyphosphate secondary to greater degree of protein binding)(b)chemisorbs on hydroxyapatite crystals in bone + in calcium crystals in mitochondria; MDP concentration at 3 hours is directly proportional to calcium contents of tissues (14-24% calcium in bone, 0.005% calcium in muscle); 50-60% (58% for MDP, 48% for EHDP, 47% for PYP) are localized in bone by approx. 3 hours depending on blood flow + osteoblastic activity; 2-10% of the dose are present within soft tissues; myocardial uptake depends on at least some revascularization of infarcted muscle *Excretion:* via urinary tract by 6 hours in 68% of MDP/EHDP, in 50% of PYP, in 46% of polyphosphates◊Forcing fluids + frequent voiding reduces radiation dose to bladder! THREE-PHASE BONE SCANNING over area of interest 1.Rapid sequence flow study (2-5 seconds/frame)= early arterial flow = 1st phase 2.Immediate postflow images (1 million counts for central body + 0.5 million counts for extremities)= blood pool = 2nd phase 3.Delayed images (0.5-1.0 million counts) between3-4 hours following injection = 3rd phase

Notes:

BONE MARROW AGENTS
for assessment of hematopoiesis / phagocytosis by RES 1.Tc-99m sulfur colloid (10% uptake in bone marrow) 2.In-111 chloride 3.Tc-99m MMAA=mini-microaggregated albumin colloid for liver, spleen, hematopoietic marrow*Particle size:*30-100 microns*Dose:*10 mCi*Marrow dose:*0.55 radMarrow accumulation at 1 hour: 6 x higher than for sulfur colloid 3 x higher than for antimony-sulfur colloid *Indications:* (a)expansion of hematopoietically active bone marrow1.Hematologic disorders to reveal presence of peripheral expansion of functional marrow(b) focal defect due to displacement by infiltrating disease1.Marrow replacement disorders: eg, Gaucher disease2.Bone infarction: eg, sickle cell anemia (DDx from osteomyelitis)3.Avascular necrosis in children

Pediatric Indications For Bone Scan Superscan Hot Bone Lesions Photon-deficient Bone Lesion Benign Bone Lesions Soft-tissue Uptake Incidental Urinary Tract Abnormalities

Notes:

Pediatric Indications For Bone Scan A.Back pain1.Discitis2.Pars interarticularis defect: SPECT imaging adds sensitivity3.Osteoid osteoma: can be used intraoperatively to assure removal of nidus4.Sacroiliac infectionB.Nonaccidental trauma

Notes:

Hot Bone Lesions *mnemonic:*"NATI MAN"**N**eoplasm **A**rthropathy **T**rauma **I**nfection **M**etastasis **A**septic **N**ecrosis **Long Segmental Diaphyseal Uptake**
A.BILATERALLY SYMMETRIC1.Hypertrophic pulmonary osteoarthropathy2.Thigh / shin splints = mechanical enthesopathy3.Ribbing disease4.Engelmann disease = progressive diaphyseal dysplasiaB.UNILATERAL1.Inadvertent arterial injection2.Melorheostosis3.Chronic venous stasis4.Osteogenesis imperfecta5.Vitamin A toxicity6.Osteomyelitis7.Paget disease8.Fibrous dysplasia

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Notes:

Photon-deficient Bone Lesion =decreased radiotracer uptake A.Interruption in local bone blood flow=vessel trauma or vascular obstruction by thrombus / tumor1.Early osteomyelitis2.Radiation therapy3.Posttraumatic aseptic necrosis4.Sickle cell crisisB.Replacement of bone by destructive process1.Metastases (most common cause): central axis skeleton > extremity, most commonly in carcinoma of kidney + lung + breast + multiple myeloma 2.Primary bone tumor (exceptional) *mnemonic:*"HM RANT"**H**istiocytosis X **M**ultiple myeloma **R**enal cell carcinoma **A**naplastic tumors (reticulum cell sarcoma) **N**euroblastoma **T**hyroid carcinoma

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Notes:

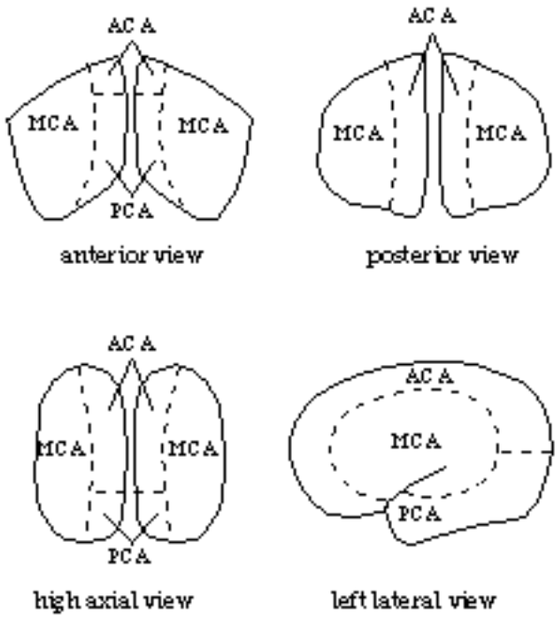
Benign Bone Lesions A.NO TRACER UPTAKE1.Bone island2.Osteopoikilosis3.Osteopathia striata4.Fibrous cortical defect5.Nonossifying fibromaB.INCREASED TRACER UPTAKE1.Fibrous dysplasia2.Paget disease3.Eosinophilic granuloma4.Melorheostosis5.Osteoid osteoma6.Enchondroma7.Exostosis

Notes:

Incidental Urinary Tract Abnormalities >50% of injected dose of Tc-99m MDP is excreted by 3 hours A.Bilateral diffuse increased uptake=uptake greater than that of lumbar spine(a)excess tissue calcium1.Hyperparathyroidism2.Hypercalcemia3.Osteosarcoma metastatic to kidney(b)tissue damage1.Drug-induced nephrotoxicity(a)Chemotherapy (eg, cyclophosphamide, vincristine, doxorubicin, bleomycin, mitomycin-C, S-6-mercaptopurine, mitoxantrone)(b)aminoglycosides(c)amphotericin B2.Radiation therapy3.Necrotic renal cell carcinoma (rare)4.Renal metastasis (rare)5.Acute pyelonephritis6.Acute tubular necrosis7.Multiple myeloma(c)iron overload1.Sickle cell anemia2.Thalassemia major*mnemonic:*"RICH CON"**R**adiation therapy to kidney **I**ron overload **C**hemotherapy (cytoxan, vincristine, doxorubicin) **H**yperparathyroidism **C**alcification (metastatic), **C**arcinoma **O**bstruction (urinary) **N**ephritis, **N**ormal variant B.Bilateral decreased renal uptake(a)loss of renal function1.Endstage renal disease(b)increased osteoblastic activity (= superscan) C.Focally decreased renal uptake(a)space-occupying lesion replacing normal renal parenchyma1.Abscess2.Cyst3.Primary / metastatic renal neoplasm(b)Scar1.Infarct2.Chronic pyelonephritis3.Partial nephrectomy D.Uni- / bilateral focally increased GU uptake(a)urine accumulation1.normal upper pole calices (supine position)2.Urinary tract diversion / ileal conduit3.Urinoma E.Change in location of kidney1.Congenital anomaly (eg, pelvic kidney)

Notes:

RADIONUCLIDE ANGIOGRAPHY



Mechanism of accumulation: disruption of blood-brain barrier *Agents:* A. Tc-99m glucoheptonate 15-20 mCi bolus injection in <2 mL saline; 30 flow images of 2 seconds duration; static image of 1 million counts after 4 hours; delayed image after 24 hours (higher target-to-background ratio than DTPA) B. Tc-99m DTPA C. Thallium-201: best predictor for tumor burden √ increased perfusion in1.Primary / metastatic brain tumor2.AVM, large aneurysm, tumor shunting3.Luxury perfusion after infarction4.Infections (eg, herpes simplex encephalitis)5.Extracranial lesions: bone metastasis, fibrous dysplasia, Paget disease, eosinophilic granuloma, fractures, burr holes, craniotomy defects√ asymmetric decreased perfusion in acute / chronic cerebrovascular disease + mass lesions (tumor, hemorrhage, subdural hematoma)√ "flip-flop" phenomenon (= decreased perfusion in arterial phase, equalization of activity in capillary phase, increased activity in venous phase) in CVA secondary to late arrival of blood via collaterals + slow washout√ bilateral absent flow in brain death

Ceretec Brain Imaging I-123 Spectamine Brain Imaging

Notes:

Ceretec Brain Imaging *Pharmacokinetics:* lipophilic radiopharmaceutical distributing across a functioning blood-brain barrier proportional to cerebral blood flow; no redistribution *Indication:* acute cerebral infarct imaging before evidence of CT / MRI pathology; positive findings within 1 hour of event

Notes:

I-123 Spectamine Brain Imaging *Pharmacokinetics:* initially distributes proportional to regional cerebral blood flow with increased flow to basal ganglia and cerebellum; homogeneous uptake in gray matter; decreased activity in white matter; redistribution over time √ activity in an area of initial deficit on reimaging (after 4 hours) implies improved prognosis***Seizures*** Abnormal cerebral radionuclide angiography within 1 week of seizure activity even without underlying organic lesion *Etiology:* (1)35% cerebral tumors (meningioma in 34%, metastases in 17%)(2)Cerebral vascular disease (more common in age >50 years)(3)Trauma, inflammation, CNS effects of systemic disease √ transient hyperperfusion of involved hemisphere ***Brain Tumor*** Good correlation between hyperperfusion and enlarged supplying vessels *Etiology:* (1)Meningioma (increased activity in 60-80%);(2)Metastases (increased activity in 11-23%);(3)Vascular metastases: thyroid, renal cell, melanoma, anaplastic tumors from lung / breast ***Cerebral Death*** Increased intracranial pressure results in markedly decreased cerebral perfusion, thrombosis, total cerebral infarction *Path:*severe brain edema, diffuse liquefactive necrosis √ carotid arteries visualized (= confirmation of good bolus) √ activity stops abruptly at the skull base √ sagittal sinus not visualized √ activity in arteries of face + scalp with "hot nose" sign*DDx by EEG:* severe barbiturate intoxication may produce a flat EEG response in the absence of brain death ***Arterial Stenosis*** ◊Radionuclide angiography of limited value!(1)Complete occlusion / >80% stenosis of ICA:53-80% sensitivity (2)50-80% stenosis of ICA: 50% sensitivity(3)<50% stenosis of ICA: 10% sensitivity Problematic lesions: (1)Bilaterally similar degree of stenosis(2)Occlusion of MCA + unilateral ACA(3)Vertebrobasilar occlusive disease (20% sensitivity)

Notes:

POSITRON EMISSION TOMOGRAPHY

A.REGIONAL CEREBRAL BLOOD FLOW(a)breathing of carbon monoxide (C-11 and O-15), which concentrates in RBCs(b)Xe-133 inhalation / injection into ICA / IV injection after dissolution in saline: volume distribution is in the water space of the brain; no correction for recirculation necessary because all Xe is exhaled during lung passage, but correction for scalp + calvarial activity is required (for inhalation method)√ washout rate of grey matter:white matter = 4-5:1B.GLUCOSE METABOLISMfor measurements of metabolic rate + mapping of functional activity (a)C-11 glucose: rapid uptake, metabolization, and excretion by brain(b)F-18 fluorodeoxyglucose (FDG): diffuses across blood-brain barrier + competes with glucose for phosphorylation by hexokinase, which traps FDG-6-phosphate within mitochondria; FDG-6-phosphate cannot enter most metabolic pathways (eg, glycolysis, storage as glycogen) and accumulates proportional to intracellular glycolytic activity; FDG-6-phosphate is dephosphorylated slowly by glucose-6-phosphatase and then escapes cell*Indications:* 1.Focal epilepsy prior to seizure surgery√ interictal decreased uptake of FDG of >20% at seizure focus (70% sensitivity, 90% for temporal lobe hypometabolism)√ hypermetabolism within 30 minutes of seizure √ measurement of opiate receptor density with C-11-labeled carfentanil (= high-affinity opium agonist) uptake by μ receptors (found in thalamus, striatum, periaqueductal gray matter, amygdala), which mediate analgesia and respiratory depression2.Alzheimer disease ● clinical diagnosis false positive in 35%√ bilateral temporoparietal hypoperfusion + hypometabolism resulting in decreased FDG uptake (92-100% sensitive)√ sparing of sensory and motor cortex + basal ganglia + thalamus*DDx:*frontal lobe dementia, primary progressive aphasia without dementia, normal-pressure hydrocephalus, multi-infarct dementia3.Parkinson disease=deficient presynaptic terminals with normal postsynaptic dopaminergic receptors ● clinical diagnosis in 50-70% accurate*DDx:*drug-induced chorea, Huntington disease, tardive dyskinesia, progressive supranuclear palsy, Shy-Drager syndrome, striatonigral degeneration, alcohol-related cerebellar dysfunction, olivopontocerebellar atrophy4.Huntington disease, senile chorea√ hypometabolism of basal ganglia 5.Schizophrenia√ abnormally reduced glucose activity in frontal lobes√ dopamine receptors in caudate / putamen elevated to 3 x that of normal levels6.Stroke, cerebral vasospasm√ disassociated oxygen metabolism + brain blood flow

Notes:

RADIONUCLIDE CISTERNOGRAPHY

Indications: 1.Suspected normal pressure hydrocephalus2.Occult CSF rhinorrhea / otorrhea3.Ventricular shunt4Porencephalic cyst, leptomeningeal cyst, posterior fossa cyst*Technique:* 1.Measurement of spinal subarachnoid pressure2.Sample of CSF for analysis3.Subarachnoid injection of radiotracerNormal study (completed within 48 hours): symmetric activity sequentially from basal cisterns, up the sylvian fissures + anterior commissure, eventual ascent over cortices with parasagittal concentration √ image lumbar region immediately after injection to assure subarachnoid injection √ activity in basal cistern by 2-4 hours √ activity at vertex by 24-48 hours √ no / minimal lateral ventricular activity (may be transient in older patients) *Agents:* 1.**Indium-111 DTPA** *Physical half-life:*2.8 days*Gamma photons:*173 keV (90%), 247 keV (94%) detected with dual pulse height analyzer*Dose:*250-500 µCi*Radiation dose:*9 rads/500 µCi for brain + spinal cord (in normal patients)*Imaging:*at 10-minute intervals / 500,000 counts up to 4-6 hours; repeat scans at 24, 48, 72 hours 2.**Technetium-99m DTPA**
Not entirely suitable for imaging up to 48-72 hours; DTPA tends to have faster flow rate than CSF; used for shunt evaluation + CSF leak study since leak increases CSF flow *Dose:*4-10 mCi*Radiation dose:*4 rads for brain + spinal cord 3.**Iodine-131 serum albumin** (RISA)prototype agent; beta emitter *Physical half-life:*8 days; high radiation dose of 7.1 rads/100 µCi; no longer used secondary to pyrogenic reactions 4.**Ytterbium-169 DTPA**
*Physical half-life:*32 days*Gamma decay:*63 keV; 177 keV (17%); 198 keV (25%); 308 keV;dual pulse height analyzer set for 177 + 198 keV*Dose:*500 µCi*Radiation dose:*9 rads/500 mCi for brain + spinal cord (in normal patients)

CSF Leak Study Hydrocephalus

Notes:

CSF Leak Study *Purpose:*localization of origin of CSF leak in patient with CSF rhinorrhea / otorrhea*Causes of dural fistula:* (a)traumatic:in 30% of basilar skull fractures(b)nontraumatic:brain, pituitary and skull tumors; skull infections; congenital defectsLocation of dural fistula: cribriform plate > ethmoid cells > frontal sinus *Method:* 1.Weigh cotton pledgets2.Pledgets placed by ENT surgeon in the anterior and posterior turbinates bilaterally3.Radiopharmaceutical injected intrathecally via lumbar puncture; immediate postinjection view of lumbar region to assure intrathecal placement4.Pledgets removed and weighed 4-6 hours after lumbar injection5.Pledget activity counted + indexed to weight6.Results compared with 0.5-mL serum specimens drawn at the time of pledget removal7.Pledget to serum count ratio of >1.5 is evidence of CSF leak8.With active leak patient should be placed in various positions with various maneuvers to accentuate leak

Notes:

THYROID SCINTIGRAPHY

A.SUPPRESSION SCAN=to define autonomy of a nodule√ suppression of a hot nodule following T_3/T_4 administration is proof that autonomy does not existB.STIMULATION SCAN=to demonstrate thyroid tissue suppressed by hyperfunctioning nodule√ administration of TSH documents functioning thyroid tissue (rarely done)C.PERCHLORATE WASHOUT TEST=to demonstrate organification defect√ repeat measurement of radioiodine uptake following oral potassium perchlorate shows lower values if organification defect present

Tc-99m Pertechnetate Iodine-123 Iodine-131 Iodine Fluorescence Imaging Thyroid Uptake Measurements

Notes:

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Iodine-123 ◊Agent of choice for thyroid imaging!*Production:* in accelerator; contamination with I-124 dependent on source (Te-122 in ~ 5%, Xe-123 in ~ 0.5%); contamination with I-125 increases with time elapsed after production *Physical half-life:*13.3 hours*Decay:*by electron capture with photon emission at 159 keV (83% abundance) + x-ray of 28 keV (87% abundance)*Dose:*200-400 µCi orally 24 hours prior to imaging (radiation dose of 7.5 mrads/µCi)*Uptake:*iodine readily absorbed from GI tract (10-30% by 24 hours), distributed primarily in extracellular fluid spaces; trapped + organified by thyroid gland; trapped by stomach + salivary glands*Excretion:*via kidneys in 35-75% during first 24 hours + GI tract *Disadvantages* compared with Tc-99m pertechnetate: (1)More expensive(2)Less available(3)More time-consuming(4)Higher dose to thyroid (but less to whole body)

Notes:

Iodine Fluorescence Imaging *Technique:* collimated beam of 60 keV gamma photons from an Am-241 source is directed at thyroid, which results in production of K-characteristic x-rays of 28.5 keV; x-rays are detected by semiconductor detector *Advantages:* (1)No interference with flooded iodine pool / thyroid medication(2)Measures total iodine content(3)Low radiation exposure (15 mrad) acceptable for children + pregnant women*Disadvantage:*dedicated equipment necessary

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Notes:

Thyroid Uptake Measurements *Agents:*I-123 / I-131 (easier to use), Tc-99m pertechnetate (requires calibration)*Uptake:* measurements at both 4 and 24 hours prevent missing the occasional rapid-turnover hyperthyroid patient returning to normal by 24 hours; uptake values distinguish different causes of hyperthyroidism (a)normal:>25% at 4 hours, >35% at 24 hours(b)increased:in Graves disease(c)decreased:in subacute thyroiditisN.B.:Uptake values do not diagnose hyperthyroidism, which is done with laboratory values (T_4, T_3, TSH) and clinical history

Notes:

Technetium-thallium Subtraction Imaging *Sensitivity:*72-92% (depending on size, smallest adenoma was 60 mg)*Specificity:*43% (benign thyroid adenomas, carcinomas, lymph nodes also concentrate thallium) *Method:* (1)IV injection of 1-3.5 mCi Tl-201 chloride; images recorded for 15 minutes with 2-mm pinhole collimator √ concentrates in normal thyroid + enlarged parathyroid glands (extraction proportional to regional blood flow + tissue cellularity)(2)IV injection of 1-10 mCi Tc-99m pertechnetate; images recorded at 1-minute intervals for 20 minutes√ pertechnetate concentrates only in thyroid(3)Computerized subtraction√ focal / multifocal excess Tl-201*Limitations:* (1)unfavorable dosimetry + poor quality images of Tl-201 (up to 3.5 mCi, 80 keV photons)(2)prolonged patient immobilization (motion artifact)(3)processing artifacts (eg, over- / undersubtraction)(4)poor Tc-99m thyroid uptake from interfering medications / recent iodinated contrast media(5)parathyroid pathology may be mimicked by coexisting thyroid disease (eg, nonfunctioning adenoma, multinodular goiter)*Indication:* Localization of one / more parathyroid adenoma (hyperplasia not visualized), may be more sensitive than CT / MRI in detection of ectopic mediastinal parathyroid tissue and in postoperative context

Notes:

Technetium-99m Sestamibi =Tc-99m MIBI*Sensitivity:*88-100% (smallest adenoma weighed 150 mg); 91% for early SPECT imaging◊For unknown reasons even large tumors (2 g) may not accumulate sufficient MIBI for detection!*Pharmacokinetics:* MIBI localizes in myocardium + mitochondria-rich tumors proportional to regional blood flow + cellular metabolic activity; MIBI washes out of thyroid quickly, but is retained in abnormal parathyroids (= need for dual-phase study) *Method:* 1.IV injection of 20-25 mCi Tc-99m MIBI2.10-30 minutes after injection anterior cervicothoracic images (5 minutes/view) with large-field-of-view camera equipped with low-energy high-resolution parallel-hole collimator3.Repeat set of images at 2-4 hours post injection (10 minutes/view)4.Adjunctive imaging with thyroid-selective agent for computer-aided subtractions is optional*Advantages (over thallium):* A.Physical properties:-optimal gamma emission (140 keV)-abundant photons (high dose of 20 mCi)-favorable dosimetry-high parathyroid-to-thyroid ratio-unaffected by medications / iodinated contrastB.Technical features:-Single readily available radiopharmaceutical-Simple protocol of early + delayed images-No prolonged patient immobilization-No subtraction study / computer processing-SPECT / multiple projections possibleC.Scan interpretation-sharp images-clear visualization of abnormal parathyroid glands-ectopic sites surveyed

Notes:

Tc-99m Macroaggregated Albumin (MAA) Preparation: human serum albumin (HSA) is heat-denatured + pH adjusted; added stannous chloride precipitates albumin into tin-containing macroaggregates; lyophilization prolongs stability; added Tc-99m pertechnetate is reduced by $SnCl_2$ and tagged onto the MAA particles *Quality control (USP guidelines):* (1)90% of particles should have a diameter between 10-90 µm(2)No particle should exceed 150 µ(3)Should be at least 90% pure (by ascending chromatography)(4)A batch of Tc-99m MAA should not be used >8 hours after preparation(5)Preparation should not be backflushed with blood into syringe, causes "hot spots" on lungs*Physical half-life:*6 hours*Biologic half-life:*6 hours*Dose:*approximately 2-4-6 mCi + 0.14 µg/kg albumin which corresponds to >60,000 particles (recommended number particles is 200,000-500,000 particles for even spatial distribution + good image quality) }IV injection in supine position to give an even distribution between base + apex of lung (ventral to posterior gradient persists)}imaging in upright position to allow maximum expansion of lung, especially at lung basesN.B.:reduce number of particles to 80,000 in(a)critically ill patients with severe COPD, on mechanical ventilator support, documented pulmonary arterial hypertension, significant left-to-right cardiac shunts need reduction in number of particles but not tagged activity!(b)children up to age 5 need reduction in number of particles + tagged activity! *Radiation dose (rads/mCi):* 0.013 for whole body, 0.25 for lung (critical organ), 0.01 for gonads PHYSIOLOGY 90% of MAA particles act as microemboli and will be trapped in lung capillaries on first pass; there are an estimated 600 million pulmonary arterioles small enough to trap the particles; the effect is insignificant physiologically as only 500,000 particles are injected per study; 0.22% of capillaries become occluded (= 2 of 1000); protein is lysed within 6-8 hours and taken up by RES; particles <1 µ are phagocytized by RES in liver + spleen IMAGING Large-field-of-view scintillation camera + parallel-hole low-energy collimator with identical recording times for corresponding views Views:anterior, posterior, lateral, posterior oblique (additional information in 50% due to segmental delineation of basal segments and separation of both lungs), anterior oblique (additional information in 15%); oblique views reduce equivocal findings from 30% to 15%

Notes:

Tc-99m Human Albumin Microspheres *Particle size:*20-30 µ*Biologic half-life:*8 hours

Notes:

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VENTILATION AGENTS
Xe-133, Xe-127, Xe-125, Kr-81m, N-13, O_2-15, CO_2-11, CO-11, radioactive aerosol (Tc-99m-DTPA, Tc-99m-PYP, Tc-99m-labeled ultrafine dry dispersion of carbon "soot")

Xenon-133 Xenon-127 Krypton-81m Tc-99m DTPA Aerosol Carbon Dioxide Tracer

Notes:

Xenon-133 Fission product of U-235 *Decay:*to stable Cs-133 under emission of beta particle (374 keV), gamma ray (81 keV), x-ray (31 keV); beta-component responsible for high radiation dose of 1 rad to lung) *Physical half-life:*5.2 days*Biologic half-life:* 2-3 minutes*Physical properties:*highly soluble in oil + grease, absorbed by plastic syringe *Administration:*injection into mouth piece of a disposable breathing unit at the beginning of a maximal inspiration*Dose:*15-20 mCi TECHNIQUE Ventilation study preferably done before perfusion scan to avoid interference with higher-energy Tc-99m (Compton scatter from Tc-99m into lower Xe-133 photopeak); [may be feasible after perfusion scan if dose of Tc-99m MAA is kept below 2 mCi + concentration of Xe-133 is above 10 mCi/l of air and if Xe-133 acquisition times for washing, equilibrium, washout images are kept to about 30 seconds] Posterior imaging routine, ideally in upright position Phase 1 = single-breath image: =inhalation of 10-20 mCi Xe-133 to vital capacity with breath-holding over 10-15-20 seconds (65% sensitivity for abnormalities)√ cold spot is abnormalPhase 2 = equilibrium phase: =tidal breathing = closed-loop rebreathing of Xe-133 + oxygen for 3-4-5 minutes for tracer to enter poorly ventilated areas; also functions as internal control for air leaks; posterior oblique images + posterior images are obtained to improve correlation with perfusion scan.√ activity distribution corresponds to aerated lungPhase 3 = washout phase: =clearance phase after readjusting intake valves of spirometer permitting patient to inhale ambient air and to exhale Xe-133 into shielded charcoal trap; washout phase should last >5 min}images taken at 30-60 sec intervals for >5 min√ rapid clearance within 90 seconds with slight retention in upper zones is normal√ tracer retention (hot spot) at 3 minutes reveals areas of air-trapping √ poor image quality secondary to significant scatter√ abnormal scan:(a)delayed washing (initial 30 seconds of tidal breathing)(b)tracer accumulation on equilibrium views (partial obstruction with collateral air drift + diffusion into affected area via bloodstream)(c)delayed washout = retention >3 minutes(d)tracer retention in regions not seen on initial single-breath view (from collateral airdrift into abnormal lung zones)

Notes:

Xenon-127 Cyclotron-produced with high cost *Physical half-life:*36.4 days*Photon energies:*172 keV (22%), 203 keV (65%)*Advantages:* (1)High photon energy allows ventilation study following perfusion study(2)Decreased radiation dose (0.3 rad)(3)Storage capability because of long physical half-life

Notes:

Krypton-81m insoluble inert gas; eluted from Rb-81 generator (half-life of 4.7 hours); decays to Kr-81 by isomeric transition *Physical half-life:*13 seconds*Biologic half-life:* <1 minute*Principal photon energy:*190 keV (65% abundance)*Advantages:* (1)Higher photon energy than Tc-99m so that ventilation scan can be performed following perfusion study(2)Each ventilation scan can be matched to perfusion scan without moving patient(3)Can be used in patients on respirator (no contamination due to short half-life)(4)Low radiation dose (during continuous inhalation for 6-8 views 100 mrad are delivered)*Disadvantages:* (1)High cost(2)Limited availability (generator good only for one day, so weekend availability may not be possible(3)No washout images possible due to short half-life(4)Decreased resolution due to septal penetration with low-energy collimators √ lack of activity = abnormal area (tracer activity is proportional to regional distribution of tidal volume because of short biologic half-life, washout phase not available)

Notes:

Tc-99m DTPA Aerosol =Tc-99m diethylenetriaminepentaacetic radioaerosol=UltraVent®*Biological half-life:*55 min*Administration:*delivery through a nebulizer during inspiration*Dose:*30-45 mCi in 2-3 mL of saline at a nebulizer flow rate of 8-10 L/minPHYSIOLOGY radioaerosols are small particles that become impacted in central airways, sediment in more distal airways, experience random contact with alveolar walls during diffusion in alveoli; crosses respiratory epithelium with rapid removal by bloodstream◊Less physiologic indicator of ventilation + subject to nebulization technique◊Erect position preferable for basilar perfusion defects (dependent lung region receives more ventilation + radiotracer)

Notes:

Carbon Dioxide Tracer O-15-labeled carbon dioxide *Physical half-life:*2 minutes (requires on-site cyclotron)PHYSIOLOGY: inhalation of carbon dioxide; rapid diffusion across alveolar-capillary membrane; clearance from lung within seconds √ cold spot due to failure of tracer entry into airway= airway disease√ hot spot due to delayed / absent tracer clearance= perfusion defect (87% sensitivity, 92% specificity)*Indications:* 1.Emboli can be detected in preexisting cardiopulmonary disease2.Equivocal / indeterminate V/Q studies

Notes:

Positron Emission Tomography *Dose:*10 mCi FDG*Technique:* }patient fasts for 4 hours◊Elevated serum glucose may cause a decrease in FDG uptake!}imaging 30-60 minutes after IV injection in 30-45 image planes (15 cm axial field of view; resolution of 5 mm)}calculation of standardized uptake ratio (SUR) in region of interest (ROI) = mean activity in ROI [mCi/mL] divided by injected dose [mCi]◊SUR >2.5 indicates malignant disease*Indications:* (1)Focal pulmonary abnormalityaccurate differentiation of benign and malignant lesions as small as 1 cm √ low FDG uptake = benign√ increased FDG uptake = cancer, active TB, histoplasmosis, rheumatoid nodule(2)Staging lung cancer◊Occult metastases detected in up to 40% of cases!(a)Intrathoracic lymph nodes√ lymph node with short-axis diameter > 1 cm by CT + not FDG avid = 100% NPV√ small lymph node by CT + intense FDG uptake = 100% PPV(b)adrenal metastasis: 100% sensitive, 80% specific(3)Recurrent disease√ increased FDG uptake at sites of residual radiographic abnormality >8 weeks after completion of therapy

Notes:

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QUANTITATIVE LUNG PERFUSION IMAGING
Indication: determination of postresection pulmonary function when combined with pulmonary function testing (FEV_1) *Technique:* 1.Acquire posterior and anterior perfusion (MAA) image and calculate geometric mean2.Separate into right + left and into 2 equal lung zones from top to bottom, which yields 4 segments (upper left, bottom right, etc)*Result:* activity in each segment is compared with total activity, which yields % perfusion to each lung field

Unilateral Lung Perfusion Perfusion Defects

Notes:

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Unilateral Lung Perfusion *Incidence:*2% A.PULMONARY EMBOLISM (23%)B.AIRWAY DISEASE(a)Unilateral pleural / parenchymal disease (23%)(b)Bronchial obstruction1.Bronchogenic carcinoma (23%)2.Bronchial adenoma3.Aspirated endobronchial foreign bodyC.CONGENITAL HEART DISEASE (15%)D.ARTERIAL DISEASE1.Swyer-James syndrome (8%)2.Congenital pulmonary artery hypoplasia / stenosis3.Shunt procedure to pulmonary artery (eg, Blalock-Taussig)E.ABSENT LUNG1.Pneumonectomy (8%)2.Unilateral pulmonary agenesis *mnemonic:*"SAFE POEM"**S**wyer-James syndrome **A**genesis (pulmonary) **F**ibrosis (mediastinal) **E**ffusion (pleural) **P**neumonectomy, **P**neumothorax **O**bstruction by tumor **E**mbolus (pulmonary) **M**ucous plug

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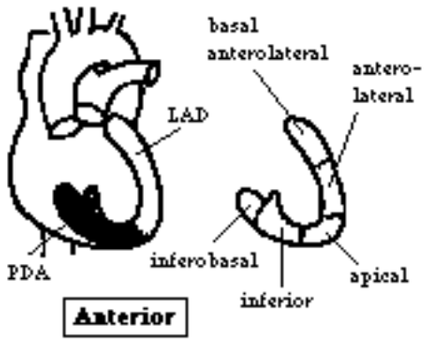
Notes:

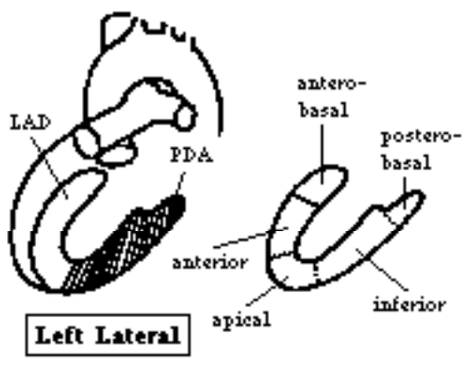
CARDIAC IMAGING CHOICES
1.PLANAR imaging2.SPECT imagingimproves object contrast by removing overlying tissues 3.QUANTITATIVE analysis=circumferential profiles = plotting of average counts along equally spaced radii emanating from center of LV makes interpretation more objective + reproducible

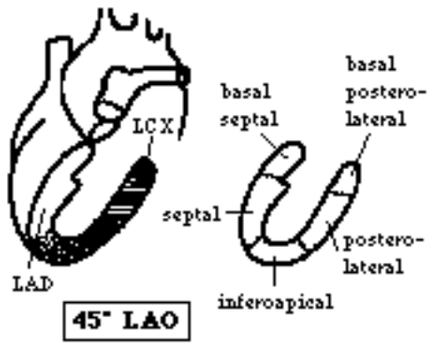
Notes:

LEFT VENTRICULAR ANATOMY AND PROJECTIONS

A.AP√ displays anterolateral wall, apex, inferior wall√ decreased activity at apex of LV due to thinning in 50%B.LEFT LATERALdisplays inferior wall, anterior wall C.LAO 40° / LAO 70° ◊Most often used projection; for all exercise studies√ displays interventricular septum, posterior wall, inferior wall√ best projection to separate right + left ventricles√ best projection to evaluate septal + posterior LV wall motionD.RAO 45°√ displays anterior + inferior ventricular wall√ useful during 1st-pass studies with temporal separation of ventriclesE.LPO 45° (rarely used)10° caudal tilt minimizes LA contamination of LV region √ displays anterior + inferior ventricular wall√ preferred over RAO 45° because LV is closer to cameraF.Angled LAO (slant-hole collimator / caudal tilt)√ separates ventricular from atrial activity√ highlights apical dyskinesis







Notes:

Tc-99m DTPA / Tc-99m Sulphur Colloid preferred for cardiac first-pass studies as they allow multiple studies with little residual from any preceding study

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Notes:

Tc-99m-labeled RBCs = agent of choice because of good heart-to-lung ratio *Technique:* (1)IN VITRO LABELING}50 mL drawn blood incubated with Tc-99m reduced by stannous ion; RBCs washed and reinjected◊Recently developed labeling kit allows excellent in vivtro labeling with only 3 mL of blood and is no longer time-consuming and expensive(2)IN VIVO LABELING}IV injection of stannous pyrophosphate (1 vial PYP diluted with 2 mL sterile saline = 15 mg sodium pyrophosphate containing 3.4 mg anhydrous stannous chloride)}15-30 minutes later injection of Tc-99m pertechnetate (+7), which binds to "pretinned" RBCs (reduction to Tc-99m [+4])◊Least time-consuming + easiest method!◊Worst labeling efficiency (30% not tagged to RBCs + excreted in urine)!(3)IN VIVTRO LABELING= MODIFIED IN VIVO METHOD }10 minutes after IV injection of 1 mg stannous pyrophosphate 10 mL of blood are drawn + incubated with Tc-99m pertechnetate for 10-20 minutes with small amount of heparin added + reinjected (3-way stopcock technique)◊Preferred method because of high labeling efficiency with little free pertechnetate!N.B.:poor tagging in(a)heparinized patient(b)injection through IV line (adherence to wall)(c)syringe flushed with dextrose instead of saline*Dose:*15-30 mCi (larger dose required for stress MUGA + obese patients);for children: 200 µCi/kg (minimum dose of 2-3 mCi)*Radiation dose:*1.5 rad for heart, 1.0 rad for blood, 0.4 rad for whole body

Notes:

Tc-99m HSA HSA = human serum albumin *Indication:*drug interference with RBC labeling (eg, heparinized patient)*Physiology:*(a)albumin slowly equilibrates throughout extracellular space(b)poorer heart-to-lung ratio than with labeled RBCs

Notes:

Thallium-201 Chloride =cation produced in cyclotron from stable Tl-203=image agent of choice to assess myocardial viability*Cyclotron:*by (p,3n) reaction to radioactive Pb-201 (half-life of 9.4 hours) which decays by electron capture to Tl-201*Decay:*by electron capture to Hg-201*Energy spectrum:*69-83 keV of Hg-K x-rays (98% abundance); 135 keV (2%) + 167 keV (8%) gamma photons*Physical half-life:*74 hours*Biologic half-life:* 10 ± 2.5 days*Dose:*1.5-3-4 mCi (the larger dose for SPECT)*Radiation dose:* 3 rad for kidneys (critical organ) (1.2 rad/mCi); 1.2 rad for gonads (0.6 rad/mCi); 0.7 rad for heart + marrow (0.34 rad/mCi); 0.5 rad for whole body (0.24 rad/mCi) *Quality control:*should contain <0.25% Pb-203, <0.5% Tl-202 (439 keV)*Indications:* 1.Acute myocardial infarction2.Coronary artery diseaseparticularly useful over ECG in: (a)conduction disturbances (eg, bundle branch block, preexitation syndrome)(b)previous infarction(c)under drug influence (eg, digitalis)(d)left ventricular hypertrophy(e)hyperventilation(f)ST depression without symptoms(g)if stress ECG impossible to obtain *Thallium uptake & distribution* intracellular uptake via Na/K-ATPase analogue to ionic potassium, but less readily released from cells than potassium; distribution is proportional to regional blood flow; uptake depends on quality of regional perfusion + integrity of sodium-potassium pump @ Blood pool <5% remain in blood pool 15 minutes post injection @Myocardiumuptake depends on (a) myocardial perfusion (b) myocardial mass (c) myocardial cellular integrity First-pass extraction efficiency is 88%!REMEMBER:90% in 90 seconds!-4% of total dose localizes in myocardium at rest (myocardial blood flow = 4% of cardiac output)-peak myocardial activity occurs at 5-15 minutes after injection-uptake can be increased to 8-10% with dipyridamole stress-clearance from myocardium is proportional to regional perfusion@ Skeletal muscle + splanchnicus: first-pass extraction efficiency is 65% -accumulate 40% of injected dose-4-6 hours fast + exercise decreases flow to splanchnicus and increases cardiac uptake@ Lung: 10% of total dose localizes in lung -augmented pulmonary extraction with left ventricular dysfunction, bronchogenic carcinoma, lymphoma of lung <5% activity over lung is normal heart-to-lung ratio decreased with triple-vessel disease@ Kidney: accumulates 4% of injected dose -excretion of 4-8% within 24 hours@ Thyroid: increased uptake >1% in Graves disease + thyroid carcinoma@ Brain: uptake only if blood-brain barrier disrupted *Technique:* A.Single dose method}3 mCi injected at peak exercise for exercise image immediately + rest image 3 hours laterB.Split dose method}2 mCi injected for exercise image}1 mCi reinjected at rest after 3 hours with rest image taken 30 minutes laterC.Booster reinjection technique}reinjection of thallium followed by imaging after 18-24-72 hours augments blood concentration of isotope=late reversibility provides evidence of regional myocardial ischemia + viability not appreciated even on very delayed (24-72 hours) redistribution images; predicts scintigraphic improvement post intervention Reasoning:50% of irreversible persistent defects improve significantly after booster reinjection*Imaging:* 1.EXERCISE IMAGE=stress thallium image=map of regional perfusion obtained within minutes after injection at peak exercise; initial distribution proportional to myocardial blood flow, arterial concentration of radioisotope, and muscle mass; 300,000-400,000 counts / view (approximately 5-8 minutes sampling time), should be completed by 30 minutes2.REDISTRIBUTION IMAGE=equilibrium between tracer uptake and efflux dependent on blood flow + mass of viable tissue + concentration gradients=map of ischemic viable myocardium obtained at rest after 2-3-6 hours; washout half-life from myocardium is 54 minutes3.DELAYED IMAGE (optional)=viability study at 24 hours

INTERPRETATION OF STRESS THALLIUM IMAGES

Immediate ImageDelayed ImageDiagnosis
normalnormalnormaldefectfill-inexertional ischemiadefectpersistentmyocardial scardefectpartial fill-inscar + ischemia / persistent ischemia

1.Initial phase = first-pass extraction temporary defect accentuated by exercise defect >15% of ventricular surface suggests >50% stenosis of coronary artery right heart well seen during stress test, tachycardia, volume / pressure overload2.Redistribution phase (on 2-4-hour images) washout in normal areas slow continued accumulation of tracer for areas of greatly reduced perfusion increased uptake in viable ischemic zones permanent defect = nonviable myocardium as in myocardial infarction / fibrosis increased lung activity (= >50% of myocardial count) indicative of (a)left ventricular failure due to severe LCA disease / myocardial infarction(b)pulmonary venous hypertension due to cardiomyopathy / mitral valve disease right heart faintly visualized during rest (15% of perfusion to right side); increased activity in RV due to(a)increase in ventricular systolic pressure(b)increase in mean pulmonary artery pressure(c)increase in total pulmonary vascular resistance *Sensitivity:*overall 82-84% for stress Tl-201(60-62% for exercise ECG)(a)increased with:(1)severity of stenosis (86% + 67% sensitive with stenosis >75% + <75%)(2)greater number of involved arteries(3)stenosis of left main > LAD > RCA > LXC(4)prior infarction(5)high work load during exercise testing in patients with single-vessel disease(b)decreased with:(1)presence of collateral(2)beta blockers(3)time delay for poststress images*Specificity:*overall 91-94% for stress Tl-201(81-83% for exercise ECG) *False-positive thallium test (37-58%):* A.Infiltrating myocardial disease1.Sarcoidosis2.AmyloidosisB.Cardiac dysfunction1.Cardiomyopathy2.IHSS3.Valvular aortic stenosis4.Mitral valve prolapse (rare)C.Decreased cardiac perfusion other than myocardial infarction1.Cardiac contusion2.Myocardial fibrosis3.Coronary artery spasm(severe unstable angina may cause defect after stress + on redistribution images, but will be normal at rest!) D.Normal variant1.Apical myocardial thinning2.Attenuation due to diaphragm, breast, implant, pacemaker*mnemonic:*"IM SIC"**I**diopathic hypertrophic subaortic stenosis **M**yocardial infarct without coronary artery disease **S**carring, **S**pasm, **S**arcoidosis **I**nfiltrative / metastatic lesion **C**ardiomyopathy *False-negative thallium test:* 1.Under influence of beta-blocker (eg, propranolol)2."Balanced ischemia" = symmetric 3-vessel disease3.Insignificant obstruction4.Inadequate stress5.Failure to perform delayed imaging6.Poor technique*mnemonic:*"3NMRS COR"**3**-vessel disease (rare) **N**oncritical stenosis **M**edications interfering **R**ight coronary lesion (isolated) **S**ubmaximal exercise **C**ollateral (coronary) blood vessels **O**verestimation of stenosis on angiography **R**edistribution (early / delayed) *Advantages compared to Tc-99m compounds:* (1)higher total accumulation in myocardium(2)provides redistribution information*Disadvantages:* (1)low energy x-rays result in poor resolution (improved with SPECT)(2)dose is limited by its long half life(3)half-value thickness of 3 cm results in less avid appearing myocardium: inferior wall (deeper part of myocardium) / anterolateral wall (overlain by breast)(4)imaging must be completed by 45 minutes post injection or redistribution occurs

Notes:

Tc-99m MIBI (Sestamibi) =cationic lipophilic isonitrile complex which associates with myocyte mitochondria*Pharmacokinetics:* -relatively rapid clearance from circulation (40% first pass extraction)-high myocardial accumulation (4%) with nonlinear uptake proportional to regional perfusion (fall-off in extraction at higher rates of flow)-slow washout with long retention time in myocardium with little recirculation-significant hepatic activity*Excretion:*through biliary tree (give milk after injection and before imaging to decrease GB activity)*Dose:*25 mCi (Cardiolite®)*Imaging:*optimum images 1 hour after injection (may be imaged up until 3 hours) *Technique:*separate injections for stress and rest studies because of slow washoutA.1-day protocolImproved detection of reversibility compared with stress-rest protocol }rest images 60-90 minutes after injection of 8 mCi Tc-99m sestamibi}wait 0-4 hours}stress patient followed by injection of 25 mCi Tc-99m sestamibi at peak stress (increased myocardial blood flow means increased myocardial uptake)}image 30-60 minutes later (optimum imaging time of stress-induced defects)B.2-day protocol (stress-rest protocol):}stress images on 1st day: Tc-99m sestamibi given at peak stress; imaging after 30-60 minutes delay to allow liver activity to decrease}repeat on 2nd day if stress views abnormal *Advantages over thallium:* (1)Low radiation dose related to shorter half-life allowing larger doses with less patient radiation(2)excellent imaging characteristics due to(a)improved photon flux which means faster imaging + allows cardiac gating(b)higher photon energy means less attenuation artifact from breast tissue / diaphragm + less scatter(3)NO redistribution(4)Temporal separation of injection and imaging allows injection during acute myocardial infarct when patient may not be stable for imaging; after stabilization + intervention (angioplasty / urokinase) imaging can demonstrate the pre-intervention defect(5)low cost(6)easy availability(7)flexible scheduling(8)increased patient throughput*Disadvantage:*less well suited to assess viability

Notes:

Tc-99m Teboroxime =neutral boronic acid oxime complex*Pharmacokinetics:* -very rapid clearance time from circulation (rapid uptake by myocardium with high extraction efficiency)-distribution proportional to cardiac blood flow EVEN at high blood flow levels (sestamibi + thallium plateau at high levels of flow)-biexponential washout from myocardium-high background from lung + liver*Dose:*25-30 mCi (Cardiotec®)*Imaging:*must begin immediately post injection due to rapid washout; rest image can immediately follow stress image

Notes:

Tc-99m Tetrofosmin =diphosphine complex*Related compounds:*Q12 (furifosmin), Q3*Pharmacokinetics:* -lower first-pass extraction and accumulation than thallium -slow myocardial washout-rapid background clearance

Notes:

Positron Emission Tomography *Perfusion agents:*N-13 ammonia, O-15 water,Rb-82 (available from a strontium generator)*Metabolic agents:*Fluorine-18-deoxyglucose (glycolysis), carbon-11-palmitate (beta-oxidation), carbon-11-acetate (tricarboxylic acid cycle)*Pathophysiology:* in myocardial ischemia glycolysis (utilization of glucose) increases while mitochondrial b-oxidation of fatty acids decreases! *Sensitivity:*>95%√ mismatched defect (= decreased perfusion but enhanced metabolism indicated by FDG uptake) indicates viable myocardium (= dysfunctional myocardium salvageable by revascularization procedure)√ matched defect (= flow + FDG accumulation both decreased) indicate nonviable myocardium√80-90% of matching defects do not improve after bypass√ 11-C-acetate superior to FDG (accurately reflects overall oxidation metabolism, not influenced by myocardial substrate utilization)*Comparison with thallium:* accuracy for fixed lesions similar; higher for reversible ischemia

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Notes:

Physical Stress Test }exercise in erect position (peak heart rate lower if supine) on treadmill or bicycle; isometric handgrip exercise raises blood pressure less (but adequate for evaluation)}starting point of workload selected according to preliminary exercise results (at an average of 200 kilowatt pounds)}workload increments by 200 kilowatt pounds up to 85% of predicted maximum heart rate (= 220 - age) / exercise limited by symptoms of chest pain, dyspnea, fatigue, arrhythmia, ischemic ECG (cardiologist with crash cart should be available) *End points for discontinuing exercise:* A.Symptoms:chest pain, dyspnea, fatigue, leg cramps, dizzinessB.Signs:fall in BP >10 mm Hg below previous stage, ventricular tachycardia, run of 3 successive ventricular premature beatsProblems with exercise imaging: (1)Sensitivity to detect ischemic lesions decreases with suboptimal exercise (in particular for older population)(2)Higher false-positive tests in women (artifacts from overlying breast tissue)(3)Propranolol (beta blocker) interferes with stress test, should be discontinued 24-48 hours prior to testing

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Notes:

Pharmacological Stress Test *Advantage:* (1)Reproducibility(2)Independent from patient motivation(3)Freedom from patient infirmities, eg, severe peripheral vascular disease, arthritis, pain*Drug:* A.VasodilatorsAction:binding to A2 receptors affects the intracellular cyclic AMP, GMP, and calcium levels resulting in coronary hyperemiaN.B.:Discontinue use of caffeine, tea, chocolate, cola drinks for 24 hours prior to test(1)IV infusion of 0.15 mg/kg/min dipyridamole(= Persantine®) for 4 minutes causing 3-5-fold increase in coronary artery blood flowDrug action:30 minutesSide effects:flushing, nausea, bronchospasm (reversible with aminophylline)◊ Prolonged supervision after test necessary}radiotracer injection 3-5 minutes later(2)IV infusion of 140 µg/kg/min adenosine(= Adenocard®, Adenoscan®) for 6 minutes Drug action:2-3 minutes (half life of 15 sec)Side effects:flushing, nausea, AV block, bronchospasm◊Supervision after test not needed}radiotracer injection during 3rd minute*Contraindication:*significant pulmonary disease requiring use of inhalersB.InotropesAction:beta-1 agonist increasing myocardial contractility thus oxygen demandCandidates:patients with COPD, asthma, allergy to vasodilators(1)IV infusion of 5 µg/kg/min dobutamine for 5 minutes, increased in steps of 5 µg/kg every 5 minutes to a maximum infusion rate of 30-40 µg/kg/min titrated to patients response}radiotracer injected at onset of significant symptoms / ECG changes / achievement of maximal rate of infusion or heart rate}infusion maintained for an additional 2 minutes with dose adjusted to patients condition(2)IV infusion of arbutamine with its own computerized delivery system titrating dose rate automatically*Contraindication:*severe hypertension, atrial flutter / fibrillation Applied to: 1.THALLIUM IMAGING (redistribution images after stress test):}injection of 1.5-2 mCi of Tl-201 during peak exercise, continuation of exercise for additional 60 seconds before imaging commencesClues for stress images: √ RV myocardium well visualized√ little pulmonary background activity√ little activity in liver, stomach, spleen√ distribution more uniform after stress than during rest◊Degree of liver uptake useful as direct measure of level of exercise!Sources of technical errors: *mnemonic:*"ABCDE PS"**A**ttenuation from overlying breast / diaphragm **B**ackground oversubtraction **C**amera field nonuniformity **D**rugs, **D**elayed (excessively) imaging, **D**ose infiltration **E**ating / **E**xercising between stress + delayed images **P**ositioning variation between stress + delayed images **S**ubmaximal exercise 2.GATED BLOOD POOL IMAGING (response of EF)√ increase in ejection fraction from 63-93% in normals√ increase in ventricular wall motion (anterolateral > posterolateral > septal)

Notes:

First-pass Ventriculography =FIRST TRANSIT = recording of initial transit time of an intravenously administered tight Tc-99m bolus through heart + lungs; limited number of cardiac cycles available for interpretation; additional projections / serial studies require additional bolus injection*Accuracy:*good correlation with contrast ventriculography*Agents:*pertechnetate, pyrophosphate, albumin, DTPA, sulfur colloid (almost any Tc-99m-labeled compound except lung scanning particles), Tc-99m-labeled autologous RBCs (most commonly)*Indication:* (1)Only 15 seconds of patient cooperation required(2)Calculation of cardiac output + ejection fraction (RBCs)(3)Subsequent first-pass studies within 15-20 minutes of initial study possible (DTPA)(4)Separate assessment of individual cardiac chambers in RAO projection (temporal separation without overlying atria, pulmonary artery, aortic outflow tract), eg, for right ventricular EF and intracardiac shunts*Technique:* }cannulation of antecubital vein with ≥20 ga needle attached to 3-way stopcock and two syringes:}syringe 1 contains ≤1 mL of radiotracer}syringe 2 contains a saline flush (10-20 mL)}injection of radiotracer is followed by a strong flush of saline*Gating:* Improved images obtained by selection of time interval corresponding only to RV passage of bolus averaged over several (3-5) individual beats; gating may be done intrinsically or with ECG guidance *Imaging:* Region of interest (ROI) over RV silhouette in RAO projection; background activity taken over horseshoe-shaped ventricular wall; counts in ROI displayed as function of time; 25 frames/second for 20-30 seconds Normal passage of bolus:SVC, RA, RV, lungs, LA, LV, aortaR-to-L shunt:tracer appears in left side of heart before passage through lungsEvaluation of: 1.Obstruction in SVC region2.Reflux from RA into IVC / jugular vein3.Stenosis in pulmonary outflow tract4.R-L shunt5.Contractility of RV6.Sequential beating of RA and RV7.Ejection fraction of RV and LV

Notes:

Equilibrium Images ="blood pool" radionuclide angiography*Agents:*Tc-99m-labeled autologous RBCs (most commonly) / human serum albumin*Imaging:*after thorough mixing of radiotracer throughout vascular space}acquisition of images during selected portions of cardiac cycle triggered by R-wave; each image is composed of >200,000 counts (2-10 minutes) obtained over 500-1,000 beats after equilibrium has been reached; high-quality images can be obtained in different projections}gated acquisition from 16-32 equal subdivisions of the R-R cycle (electronic bins) allows display of synchronized cinematic images (assembled to composite single-image sequence) of an "average" cardiac cycle√ may be displayed as time activity curves reflecting changes in ventricular counts throughout R-R interval-measured functional indices: preejection period (PEP), left ventricular ejection time (LVET), left ventricular fast filling time ($LVFT_1$), left ventricular slow filling time ($LVFT_2$), PEP/LVET ratio, rate of ejection + filling of LV}at rest: count density 200-250 counts/pixel requires generally 7-10 minutes acquisition time for 200,000-250,000 counts/frame}during exercise: 100,000-150,000 counts/frame requires an acquisition time of 2 minutes Evaluation of: 1.LV ejection fraction2.Regional wall motion3.Valvular regurgitation *Interpretation:* 1.Heart failure: decreased EF, prolongation of PEP, shortening of LVET, decreased rate of ejection2.Hypertensive heart: normal systolic indices, normal EF, prolonged $LVT_1$3.Hypothyroidism: prolonged PEP, normal EF4.Aortic stenosis: mild reduction of EF, prolonged LV emptying time, decreased rate of ejection, normal rate of filling√ area of decreased periventricular uptake secondary to(a)pleural effusion >100 mL (b)ventricular hypertrophy

Notes:

Gated Blood Pool Imaging =MULTIPLE GATED ACQUISITION (MUGA)Recording of: (1)Ejection fraction (EF) of left ventricle before + after exercise (>6 million counts, 32 frames)(2)Regional wall motion of ventricular chambers (>4.5 million counts, 24 frames)(a)at rest:myocardial infarction, aneurysm, contusion(b)during exercise:ischemic dyskinesia (detectable in 63%)(3)Regurgitant indexProjection: (a)best septal view (usually LAO 45°) for EF; often requires some cephalad tilting of detector head(b)two additional views for evaluation of wall motion (usually anterior + left lateral views)*Imaging:* Physiologic trigger provided by R-R interval of ECG ("bad beat" rejection program desirable) (a)gated images obtained for 5 minutes(b)2-minute image acquisition time for each stage of exercise *PROs:*(1)Higher information density than 1st-pass method(2)Assessment of pharmacologic effect possible(3)"Bad beat" rejection possible*CONs:*(1)Significant background activity(2)Inability to monitor individual chambers in other than LAO 45° projection(3)Plane of AV valve difficult to identify*Radiation dose:*1.5 rad for heart; 1.0 rad for blood; 0.4 rad for whole body

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Notes:

INFARCT-AVID IMAGING
=hot spot imaging*Agent:*Tc-99m pyrophosphate (standard), Hg-203 chlormerodrin, Tc-99m tetracycline, Tc-99m glucoheptonate, F-18 sodium fluoride, Indium-111 antimyosin (murine monoclonal antibodies to myosin), Tc-99m antimyosin Fab fragment

Tc-99m Pyrophosphate Tc-99m Antimyosin Fab Fragments

Notes:

Tc-99m Antimyosin Fab Fragments =specific marker for myocyte damage=Fab fragments of an antibody raised against water-insoluble heavy chains of cardiac myosin that are exposed due to necrosis*Sensitivity*:95% uptake ONLY in acute infarct with decreasing intensity as the infarct heals

Notes:

NONAVID INFARCT IMAGING

=Cold spot imaging=myocardial perfusion study for acute myocardial infarct*Agent:*Tl-201 (at rest)*Sensitivity after onset of symptoms:* 96% within 6-12 hours, 79% after 48 hours, 59% in remote infarction; sensitivity for SPECT (seven pinhole tomography) 94% > planar scintigraphy 75% √ fixed permanent defect in acute infarction√ fixed permanent defect at rest + on stress thallium + redistribution images in old infarction√ "cold defect" at rest may represent transient ischemia in unstable anginaN.B.:Tl-201 cannot distinguish between recent + remote infarction!

Notes:

BILIARY SCINTIGRAPHY
Tc-99m IDA analogs =Tc-99m acetanilide iminodiacetic acid analogs (IDA) Dependent on the substances lipophility, there is a trade-off between renal excretion + hepatic uptake (BIDA is the most lipophilic, HIDA the least lipophilic) 1.HIDA (2,6-dimethyl derivative): [H = hepatic] bilirubin threshold of <18 mg/dL; 15% renal excretion2.BIDA (parabutyl derivative): bilirubin threshold of <20 mg/dL3.PIPIDA (paraisopropyl derivative): 2% renal excretion4.DIDA (diethyl derivative)5.DISIDA (diisopropyl derivative) = Disida®, Disofen®, Hepatolite®: bilirubin threshold of <30 mg/dL6.TMB-IDA (m-bromotrimethyl IDA) = Mebrofenin®, Choletec®: $T_{1/2}$ uptake is 6 minutes, $T_{1/2}$ excretion is 14 minutes in normals *Quality control:*the final compound should contain- 90-100% Tc-99m IDA-<10% Tc-99m tin colloid-<10% Tc-99m sodium pertechnetate *Pharmacokinetics:* @Bloodstreamtracer bound predominantly to albumin, which decreases renal excretion (renal excretion seen in most normals); dissociation of albumin + Tc-99m-IDA takes place at space of Disse @Liverpeak liver activity 5-10 minutes post injection = hepatic phase; 85% extracted by hepatocytes; tracer enters anion pathway of bilirubin ◊Delayed liver uptake implies hepatocyte dysfunction / CHF (less likely)◊Look for liver lesions on early images@Bilesecretion by hepatocytes without conjugation; CBD + cystic duct visualized within 15 minutes (not always visualized in normals); GB visualized by 20 minutes ◊Activity in right paracolic gutter / intraperitoneal space implies postoperative bile leak@Bowelexcretion into duodenum by 30 minutes; bowel visualized within 1 hour; no enterohepatic recirculation *Dose:*3-7 mCi for adults (higher dose may be needed for high bilirubin level + for tracer with lower bilirubin threshold)*Radiation dose:*2 rad for upper large bowel; 0.55 rad for gallbladder; 3 rad/mCi for small bowel; 0.01 rad/mCi for whole body *Patient preparation:* 1.Narcotics (opiates) + sedatives increase tone of sphincter of Oddi and are stopped 6-12 hours before exam2.Fasting of at least 2-4 hours but <24 hours3.Cholecystokinin-C-terminal octapeptide= Sincalide (slow IV injection of 0.02 µg/kg Kinevac®) may be used to empty gallbladder about 30 minutes before tracer injection in patients on prolonged fasting (gallbladder atony + retained bile and sludge secondary to absence of endogenously produced CCK) Useful in:(a)patient fasting >24 hours / on total parenteral nutrition(b)acalculous cholecystitisSide effect:increase in biliary-to-bowel transit time *Equipment:* Large field-of-view scintillation camera fitted with LEAP collimator; spectrometer set at 140 keV with 20% window Computer software for deconvolutional analysis allows determination of percent of hepatic arterial and percent of portal venous blood flow to liver (helpful in assessment of liver transplants) *Imaging:*
at 5-10-minute intervals for 60 minutes; if gallbladder not visualized for at least up to 4 hours; RLAT, RAO, LAO projections to confirm gallbladder position ◊Look for enterogastric reflux as a cause of biliary gastritis! *IV morphine sulfate* (0.04 µg/kg): contracts sphincter of Oddi + raises intrabiliary pressure with retrograde filling of gallbladder; maximal effect 5 minutes post injection; shortens study time to 1 hour in cases of nonvisualization of gallbladder when injected 30-40 minutes into study; increases accuracy from 88% to 98% and specificity from 83% to 100% *Normals:* gallbladder appearance within 60 minutes (90% within 30 minutes); gallbladder visualization within 30 minutes after administration of morphine; small bowel activity within 90 minutes (80% within 60 minutes) ***Gallbladder Ejection Fraction*** (GBEF) GBEF = $[GB_{initial} - GB_{post}] \div GB_{initial}$ *Indication:* (1)to increase sensitivity of study for acute (acalculous) cholecystitis(2)in patients with atypical GB pain and no cholelithiasis *Technique:* 1.Select ROI about GB2.Administer Sincalide in a dose of 0.02 µg/kg body weight IV over 30 minutes (with infusion pump)*Normal result:*>30% GBEF
False-positive DISIDA Scan *mnemonic:*"F2C PAL"**F**ood (recent meal) **F**asting (prolonged) **C**ystic duct cholangiocarcinoma **P**ancreatitis **A**lcoholism **L**iver dysfunction
False-negative DISIDA Scan *mnemonic:*"ADA"**A**calculous cholecystitis **D**uodenal diverticulum simulating GB **A**ccessory cystic duct

Notes:

LIVER SCINTIGRAPHY

Technetium-99m Sulfur Colloid =LIVER-SPLEEN SCAN*Indications:*liver, spleen, bone marrow, acute rejection in renal transplant, lower GI bleeding, gastric emptying*Preparation:* Tc-99m pertechnetate and sodium trisulfate are heated in a water bath (95 ± 5°C) for 10 ± 2 minutes; sulfur atoms aggregate to form a "colloid" (average particle size 0.1-1 µ with a range of 0.001-1 µ; true colloid has a particle size of 0.001-0.5 µ); gelatin is added to prevent further growth of particles *Quality control:* (a)>92% remain at origin of ascending chromatography(b)upper limit for particle size is 1 µ-Usual cause for poor preparation is excessive / prolonged heating or a pH >7-Preparation should not be used >6 hours (agglomeration of particles with aging) *Dose:*usually 3-6 mCi (8 mCi for SPECT)*Radiation dose:*0.3 rad/mCi for liver (critical organ); 0.02 rad/mCi for whole body; 0.025 rad/mCi for bone marrow*Imaging:*15-30 minutes post IV injection *Pharmacokinetics:* accumulation in liver (85%), spleen (10%), bone marrow (5%); lung localization is rare (presumably secondary to circulating endotoxins + macrophage infiltration) A.RETICULOENDOTHELIAL LOCALIZATION√ colloid shift away from liver in diffuse hepatic dysfunction / decreased hepatic perfusion√ increased bone marrow activity in hemolytic anemia√ increased splenic activity in hypersplenism of splenomegaly / cancer / systemic illnessB.BONE MARROW LOCALIZATIONHematopoietic system extends into long bones in children; recedes to axial skeleton, femora, and humeri with age◊Bone marrow distribution cannot be used to determine sites of erythropoiesis!C.ABSCESS LOCALIZATIONSulfur colloid phagocytized by PMNs + monocytes *Labeling:* (a)in vivo:small labeling yield(b)in vitro:40% labeling efficiency, but difficult + time-consuming preparation ***Colloid Shift*** A.Hepatic dysfunction1.Cirrhosis2.Hepatitis3.Chronic passive congestionB.Augmented perfusion of spleen + bone marrow1.Hematopoietic disorders2.Long-term corticosteroid therapy ***Focal Hot Liver Lesion*** 1.IVC / SVC obstruction√ increased perfusion of quadrate lobe located at posterior aspect of medial segment left hepatic lobe (collateral pathway via umbilical vein)2.Budd-Chiari syndrome√ "increased" perfusion of caudate lobe (actually decrease of activity elsewhere in liver)3.FNH (varying amount of Kupffer cells)√ hot / cold / isoactive with surrounding parenchyma4.Regenerating nodules of cirrhosis ***Defects In Porta Hepatis*** 1.Normal variant (thinning of hepatic tissue overlying portal veins + gallbladder)2.Biliary causes: dilatation of bile ducts, gallbladder hydrops3.Enlarged portal lymph nodes4.Metastases5.Hepatic cyst6.Hepatic parenchymal disease (pseudotumor)7.Hepatic compression by adjacent extrinsic mass8.Postsurgical changes following cholecystectomy ***Focal Liver Defects*** A.Neoplastic(a)primary liver tumor: hepatoma, hemangioma, hepatic adenoma, FNH(b)metastases: 85% sensitivity, 75-80% specificity (for lesion >1-2 cm)B.Infectious disease / abscessC.Benign cystD.TraumaE.Pseudotumor = normal variant*mnemonic:*"LCHAIM**L**ymphoma **C**yst **H**ematoma **A**bscess **I**nfarct **M**etastasis ***Mottled Hepatic Uptake*** 1.Cirrhosis2.Acute hepatitis3.Lymphoma4.Amyloidosis5.Granulomatous disease(sarcoid, fungal, viral, parasitic) 6.Chemo- / radiation therapy

Notes:

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SPLENIC SCINTIGRAPHY

1.Tc-99m sulfur colloid: 3-5 mCi 2.Tc-99m heat-denatured erythrocytes*Indication:* (1) Splenic trauma (2) Accessory + ectopic spleen *Technique:* 20-30 minutes after injection of pyrophosphate IV 15-20 mL of blood are drawn + incubated with 2 mCi of pertechnetate; blood is heated to 49.5°C for 35 minutes and reinjected ⚱ Fragmentation of RBCs from overheating increases hepatic uptake!*Imaging:*20 minutes post injection

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Notes:

Radionuclide Esophagogram *Preparation:*4-12 hours fasting; imaging in supine / erect position*Dose:*250-500 µCi Tc-99m sulfur colloid in 10 mL of water taken through straw*Imaging:*when swallowing begins√ normal transit time: 15 seconds with 3 distinct sequential peaks progressing aborally√ prolonged transit time: achalasia, progressive systemic sclerosis, diffuse esophageal spasm, nonspecific motor disorders, "nutcracker" esophagus, Zenker diverticulum, esophageal stricture + obstruction*Difficult interpretation in:*hiatal hernia, GE reflux, Nissen fundoplication

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Notes:

Levine / Denver Shunt Patency *Technique:* sterile injection of 0.5-1 mCi Tc-99m MAA / sulfur colloid via paracentesis *Imaging:* over abdomen (or chest) to detect uptake in liver (or lung), which confirms patency

Notes:

RENAL AGENTS

Renal Scintigraphic Agents			
MORPHOLOGIC AGENTS			
Tc-99m GHA	5 mCi	proximal tubular uptake + glomerular filtration	collecting system visualized on delayed images
Tc-99m DMSA	2–5 mCi	proximal + distal tubular uptake	limited availability, relatively high radiation dose, collecting system not visualized on delayed images
FUNCTIONAL AGENTS			
I-131 OIH	200–400 μCi	80% secreted, 20% filtered	routinely used for ERPF measurement, analog of PAH, highest renal extraction fraction, poor image detail, high radiation dose, requires high-energy collimator
Tc-99m DTPA	10–15 mCi	nearly 100% filtered	GFR calculation, delayed time-to-peak with slow clearance
Tc-99m MAG_3	2–10 mCi	99% secreted	ERPF estimate, good cortical detail, high target-to background ratio

1.Agents for renal function:Tc-99m DTPA,I-131 Hippuran2.Renal cortical agent:Tc-99m DMSA3.Renal combination agent:Tc-99m glucoheptonate

Tc-99m DTPA [Tc-99m Glucoheptonate] Tc-99m DMSA [I-131 OIH] Tc-99m Mercaptoacetyltriglycine (MAG3) Enalaprilat-enhanced Renography Cold Defect On Renal Scan

Notes:

[Tc-99m Glucoheptonate] largely replaced by Tc-99m MAG3 *Pharmacokinetics:* rapid plasma clearance + urinary excretion with excellent definition of pelvicalyceal system during 1st hour; extracted by (a) glomerular filtration and (b) tubular excretion (30-45% within 1st hour); 5-15% of dose accumulates in tubular cells by 1 hour, 15-25% by 3 hours; cortical accumulation remains for 24 hours Imaging: (a)collecting system within first 30 minutes(b)renal parenchyma after 1-2 hours (interfering activity in collecting system) *Biologic half-life:*2 hours*Dose:*15 (range 10-20) mCi *Radiation dose*:0.17 rads/mCi for kidney; 0.008 rads/mCi for whole body; 0.015 rads/mCi for gonads

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Notes:

[I-131 OIH] largely replaced by Tc-99m MAG3 =I-131 orthoiodohippurate (Hippuran®)=good for evaluation of renal tubular function / effective renal plasma flow; agent with highest extraction ratio without binding to renal parenchyma; visualizes kidney even in severe renal failure*Pharmacokinetics:* 80% secreted by proximal tubules; 20% filtered by glomeruli; maximal renal concentration within 5 minutes; normal transit time of 2-3 minutes; approximately 2% free iodine◊Lugols solution is administered to protect thyroid*Imaging:* in 15-60-second intervals for 20 minutes; renal uptake determined from images obtained by 1-2 minutes (patient in supine position for equidistance of kidneys to camera) *Biologic half-life:*10 minutes (with normal renal function)*Dose:*200 (range 150-300) µCi *Radiation dose:*0.06 rads/200 µCi for bladder; 0.02 rads/200 µCi for kidney; 0.02 rads/200 µCi for whole body; 0.02 rads/200 µCi for gonads

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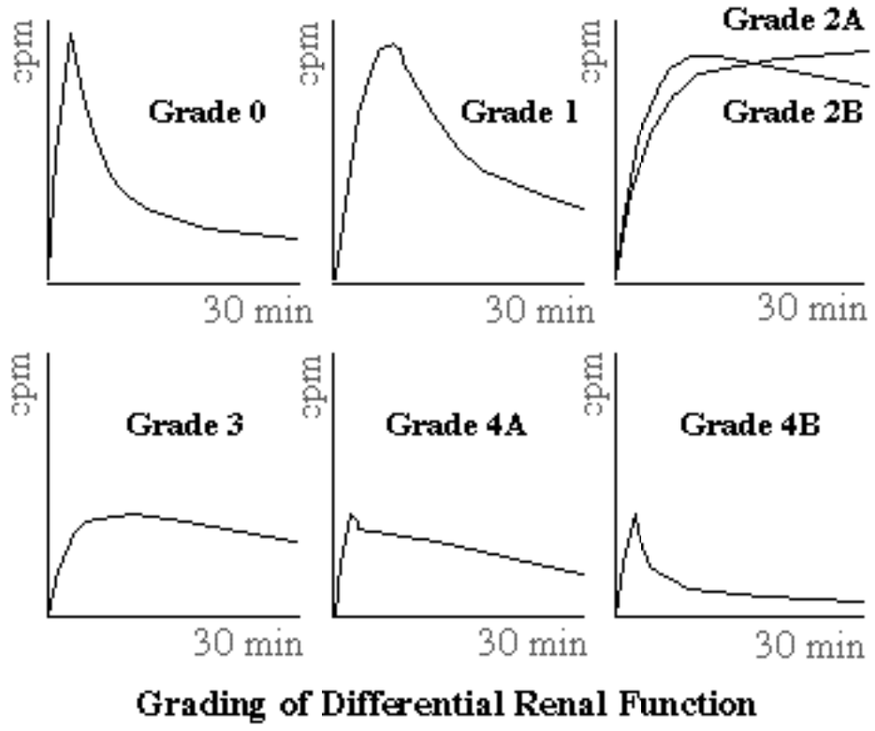
Notes:

Tc-99m Mercaptoacetyltriglycine (MAG_3)

=renal plasma flow agent similar to OIH but with imaging benefits of Tc-99m label (improved dosimetry)*Pharmacokinetics:* correlates with renal plasma flow; clearance is less than Hippuran *Dose:*10 mCiEvaluation: true renal plasma flow = MAG_3 flow (obtained off renogram curve) multiplied by a constant (varies between 1.4 and 1.8)

Notes:

Enalaprilat-enhanced Renography =screening for renovascular hypertension with angiotensin-converting enzyme inhibitor (ACEI)*Pharmacology:* the affected kidney responds to decreased arteriolar flow by releasing angiotensin II (= extremely potent vasoconstrictor acting on the efferent renal arteriole to increase filtration pressure); ACE inhibitors (eg, captopril, enalapril) block the angiotensin-converting enzyme Protocol: 1.Blood pressure checked (to prevent testing-4 d excessively hypertensive patients)2.Stop antihypertensive medications-9 hrs overnight (except for b-blockers)3.Fasting (liquids acceptable)-4 hrs4.Bladder catheterization to monitor -40 minurinary output5.1/2 normal saline IV drip at 75 mL/hr-30 min6.Lasix (= furosemide) IV-5 min20 mg if serum creatinine <1.5 mg/dL,40 mg if serum creatinine >1.5 mg/dL,60 mg if serum creatinine >3.0 mg/dL(not to exceed 1.0 mg/kg)7.2.5 mCi Tc-99m MAG_3 IV for baseline0 minstudy(a)flow phase with 1 sec/frame for60 frames(b)tracer kinetic (dynamic) phasewith 15 sec/frame for 120 frames8.Rehydration with 1/2 normal salinekeeping a 250-300 mL negative+30 minfluid balance 9.0.04 mg/kg Enalaprilat IV with blood +105 minpressure + heart rate checksq 5 minutes10.Repeat Lasix (= furosemide) IV+115 min(step 6)11.7.5 mCi Tc-99m MAG_3 IV for +120 minEnalaprilat-enhanced study12.10 mCi Tc-99m MAG_3 IV single post-Enalaprilat study for patients already onACEI therapy



Grading of Differential Renal Function

√ change from baseline grade 0 / 1 by >1 grade=high probability for renal artery stenosis√ abnormal baseline curve without change= indeterminate for renovascular hypertension √ functional improvement following ACEI challenge= low probability for renovascular hypertension

Notes:

Cold Defect On Renal Scan *mnemonic:*"CHAT SIN"**C**yst **H**ematoma **A**bscess **T**umor **S**car **I**nfarct **N**eoplasm

Notes:

Radiology Review Manual
fourth edition

DIFFERENTIAL RENAL FUNCTION

Agents: (1)Tc-99m DTPA:measurements prior to excretion within first 1-3 minutes; images taken at 1.5-second intervals for 30 seconds followed by serial images for next 30 minutes (2)I-131 Hippuran:measurements prior to excretion within first 1-2 minutes Evaluation:generation of time-activity curves√ upslope (= accretion phase) √ peak activity (maximal uptake phase) √ downslope (excretion phase)
√ increased hepatic + soft-tissue uptake with impaired renal function√ measurements usually not significantly affected with differences in renal depth√ measurements are accurate in renal obstruction if obtained within 1-3 minutes√ prediction about functional recovery not possible following surgical relief of obstruction

Notes:

RADIONUCLIDE CYSTOGRAM
Technique: Infusion of 0.5-1 mCi Tc-99m pertechnetate-saline mixture into bladder *Imaging:* posterior upright views throughout filling and voiding phases; review on cinematic loop helpful; residual bladder volume can be calculated *Advantage:* lower radiation dose to child than comparable contrast study

Notes:

ADRENAL SCINTIGRAPHY

A.ADRENOCORTICAL IMAGING AGENTS1.NP-592.Selenium-75 6-b-selenomethylnorcholesterol (Scintadrin®)B.SYMPATHOADRENAL IMAGING AGENTS1.I-131 / I-123 metaiodobenzylguanidine (MIBG)

I-131 Metaiodobenzylguanidine (MIBG) I-123 Metaiodobenzylguanidine Iodocholesterol

Notes:

I-131 Metaiodobenzylguanidine (MIBG) *Indications:* APUDomas = tumors of neural crest origin (C cells of thyroid, melanocytes of skin, chromaffin cells of adrenal medulla, pancreatic cells, Kulchitsky cells), which share the presence of neurosecretory granules capable of accumulating I-131 MIBG (1)Pheochromocytoma (80-90% sensitivity, >90% specificity); tumors as small as 0.2 g have been detected(2)Neuroblastoma, carcinoid, medullary thyroid carcinoma, nonfunctioning retroperitoneal neuroendocrine tumor, middle mediastinal paraganglioma, adrenal metastasis of choriocarcinoma, Merkel (skin) tumor *Pharmacokinetics:* Chemically similar to norepinephrine, which is synthesized by adrenergic neurons + cells of the adrenal medulla; localizes in storage granules of adrenergic tissue by means of energy- and sodium-dependent uptake mechanism; not metabolized to any appreciable extent; Normal activity is seen in liver, spleen, bladder, salivary glands, myocardium, lungs; 85% of injected dose is excreted unchanged by kidneys *Method:* Lugol solution administered orally (50 mg of iodine per day) for 4-5 days starting the day before injection (to block thyroid uptake of free iodine) *Dose:*0.4 mCi (14.8 MBq) or maximally 0.5 mCi/1.73 square meters of body surface MIBG*Radiation dose:*35 rad/mCi for adrenal medulla, 1.0 rad/mCi for ovaries, 0.4 rad/mCi for liver, 0.22 rad/mCi for whole body*Imaging:*24, 48, (72) hours after injection*False-negative scan:* uptake blocked by reserpine, imipramine, other tricyclic depressants, amphetamine-like drugs

Notes:

I-123 Metaiodobenzylguanidine also allows SPECT imaging *Dose:*10 mCi*Radiation dose:*2.76 rad/mCi for adrenals, 0.07 rad/mCi for ovaries, 0.05 rad/mCi for liver, 0.02 rad/mCi for whole body*Imaging:*at 6 and 24 hours

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Notes:

STATISTICS

Incidence=number of diseased people per 100,000 population per year **Prevalence**=number of existing cases per 100,000 population at a target date
Mortality=number of deaths per 100,000 population per year **Fatality**=number of deaths per number of diseased
Decision Matrix: GOLD STANDARD Tnormalabnormalsubtotal EnormalTNFNT-NPV SabnormalFPTPT+PPV T

subtotalD-D+total specsensacc
TP=test positive in diseased subjectFP=test positive in nondiseased subjectFN=test negative in diseased subjectTN=test negative in nondiseased subjectT+=abnormal test resultT-=normal test resultD+=diseased subjectsD-=nondiseased subjects

Sensitivity Specificity Accuracy Positive Predictive Value Negative Predictive Value False-positive Ratio False-negative Ratio Disease Prevalence

Notes:

Accuracy =number of correct results in all tests=number of correct tests / total number of tests=(TP + TN) / (TP + TN + FP + FN) = (TP + TN) / total◊depends much on the proportion of diseased + nondiseased subjects in studied population◊Not valuable for comparison of tests*Example:*same test accuracy of 90% for two tests A and B
Test A GOLD STANDARD Tnormalabnormalsubtotal Enormal 90 10 100 Sabnormal10 90 100 T

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subtotal100100200
Test B GOLD STANDARD Tnormalabnormalsubtotal Enormal 17020 190 Sabnormal0 10 10 T

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subtotal17030 200

Notes:

False-positive Ratio =proportion of nondiseased patients with an abnormal test result ● D- column in decision matrix=FP / (FP + TN) = FP / D-=1 - specificity = (TN + FP - TN) / (TN + FP)

Notes:

BAYESS THEOREM
=the predictive accuracy of any test outcome that is less than a perfect diagnostic test is influenced by(a)pretest likelihood of disease(b)criteria used to define a test result

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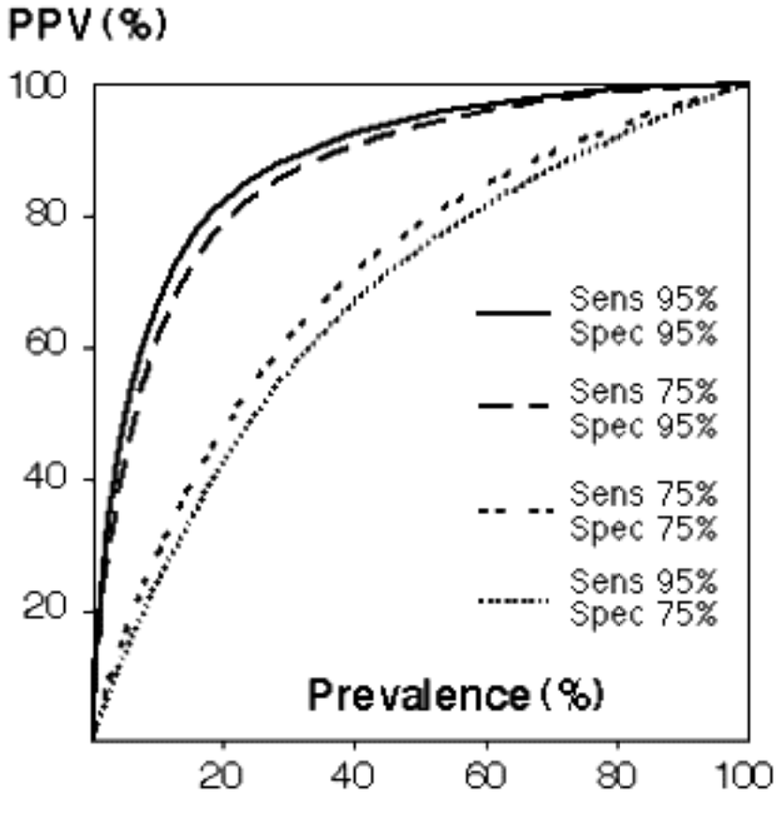
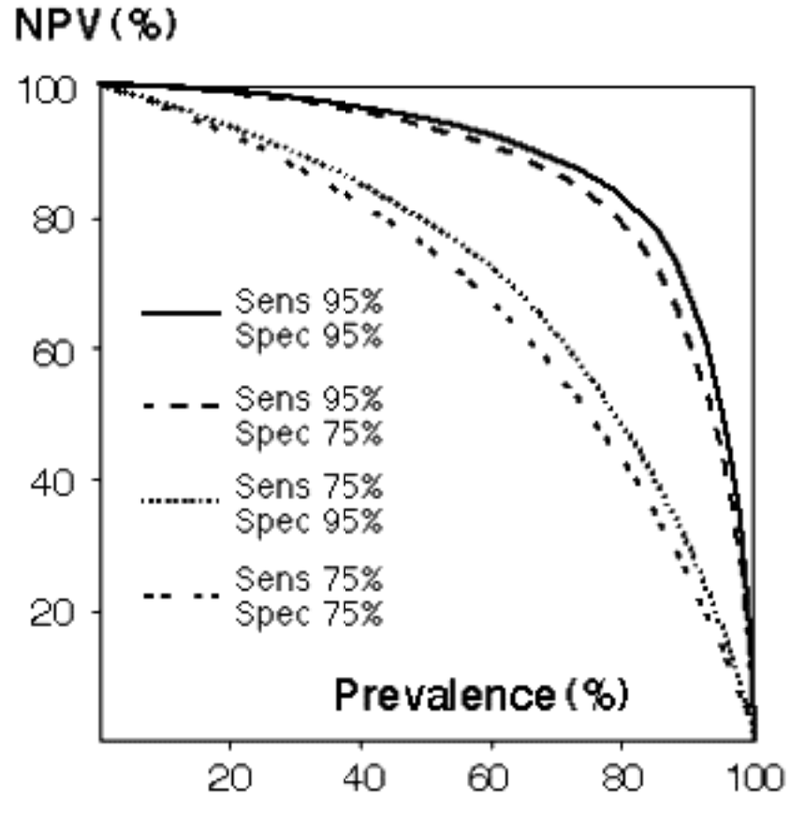
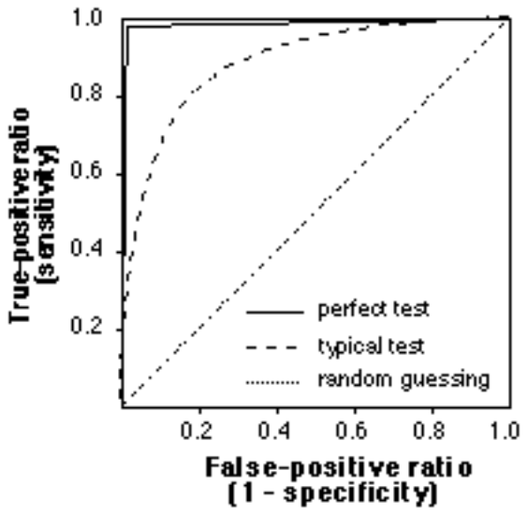
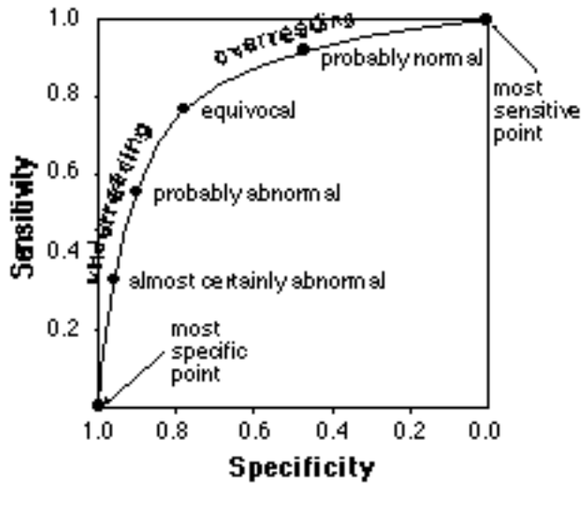
Notes:

RECEIVER OPERATING CHARACTERISTICS (ROC)
=degree of discrimination between diseased + nondiseased patients using varying diagnostic criteria instead of a single value for the TP + TN fraction=curvilinear graph generated by plotting TP ratio as a function of FP ratio for a number of different diagnostic criteria (ranging from definitely normal to definitely abnormal)Y-axis:true-positive ratio = sensitivityX-axis:false-positive ratio = 1 - specificity;reversing the values on the X-axis results in an identical "sensitivity-specificity curve" *Use:*variations in diagnostic criteria are reported as a continuum of responses ranging from definitely abnormal to equivocal to definitely normal due to subjectivity + bias of individual radiologist◊A minimum of 4-5 data points of diagnostic criteria are needed! *Difficulty:*subjective evaluation of image features; subjective diagnostic interpretation; data must be ordinal (= discrete rating scale from definitely negative to definitely positive) *Interpretation:* ◊Increase in sensitivity leads to decrease in specificity!◊Increase in specificity leads to decrease in sensitivity!◊The most sensitive point is the point with the highest TP ratio-equivalent to "overreading" by using less stringent diagnostic criteria (all findings read as abnormal)◊The most specific point is the point with the lowest FP ratio-equivalent to "underreading" by using more strict diagnostic criteria (all findings read as normal)◊The ROC curve closest to the Y-axis represents the best diagnostic test◊Does not consider disease prevalence in the population









Notes:

KAPPA (K)
measures concordance between test results and gold standard◊Analogous to Pearson correlation coefficient (r) for continuous data!

GOLD STANDARD

T		$M_2M'_2$	$M_3M'_3$	$M_4M'_4$	M_1
E	$M_2M'_1$		$M_2M'_3$	$M_2M'_4$	M_2
S	$M_3M'_1$	$M_3M'_2$		$M_3M'_4$	M_3
T	$M_4M'_1$	$M_4M'_2$	$M_4M'_3$		M_4
	M'_1	M'_2	M'_3	M'_4	N

$$P_o = \frac{\Sigma_1^4 A}{N} \qquad P_c = \frac{\Sigma_1^4 MM'}{N^2}$$

$$\kappa = \frac{P_o - P_c}{1 - P_c}$$

GOLD STANDARD

T	18	3	0	0	21
E	2	20	5	2	29
S	1	4	20	3	28
T	0	0	5	17	22
	21	27	30	22	100

*Example:*K = 0.743 GOLD STANDARD T1830021 E2205229 S1420328 T0051722 21273022100 *Predictive value of K:* 0.00 - 0.20little or none0.20 - 0.40slight0.40 - 0.60group0.60 - 0.80some individual0.80 - 1.0individual

Notes:

CONFIDENCE LIMIT
=degree of certainty that the proportion calculated from a sample of a particular size lies within a specific range (binomial theorem)◊Analogous to the mean ± 2 SD

Notes:

CLINICAL EPIDEMIOLOGY
=application of epidemiologic principles + methods to problems encountered in clinical medicine with the purpose to develop + apply methods of clinical observation that will lead to valid clinical conclusionsEpidemiology = branch of medical science dealing with incidence, distribution, determinants in control of disease within a defined population

Screening Techniques Self-selection Randomized Trials Case-control Studies Calculation of odds ratio = ad / bc :

Notes:

Screening Techniques *Principle question:*can early detection influence the natural history of the disease in a positive manner?*Outcome measure:*early detection + effective therapy should reduce morbidity + mortality, ie, increase survival rates (observational study)!*Biases:* **Lead time** = interval between disease detection at screening + the usual time of clinical manifestation; early diagnosis always appears to improve survival by at least this interval, even when treatment is ineffective **Length time** = differences in growth rates of tumors: (a)slow-growing tumors exist for a long time before manifestation thus enhancing the opportunity for detection(b)fast-growing tumors exist for a short time before manifestation thus providing less opportunity for detection at screening "interval cancers" = clinically detected between scheduled screening exams are likely fast-growing tumors; patients with tumors detected by means of screening tests will have a better prognosis than those with interval cancers

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Notes:

Self-selection
= decision to participate in screening program; usually made by patients better educated + more knowledgeable + more health-conscious; mortality rates from noncancerous causes can be expected to be lower than in general population

Overdiagnosis = detection of lesions of questionable malignancy, eg, in-situ cancers, which might never have been diagnosed without screening + have an excellent prognosis

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Notes:

Randomized Trials *Design:*two arms consisting of (a) study group(b) control group with patients assigned to each arm on randomized basis *Endpoint:*difference in mortality rates of both groups*Power:*study must be of sufficient size + duration to detect a difference, if one exists; analogous to sensitivity of a diagnostic test *Impact on effective size of groups:* **Compliance** = proportion of women allocated to screening arm of trial who undergo screening **Contamination** = proportion of women allocated to control group of trial who do undergo screening

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Notes:

Case-control Studies Retrospective inquiry which is less expensive, takes less time, is easier to perform: (a)determine the number of women who died from breast cancer(b)chose same number of women of comparable age who have not died from breast cancer(c)ascertain the number of women who were screened + who were not screened in both arms

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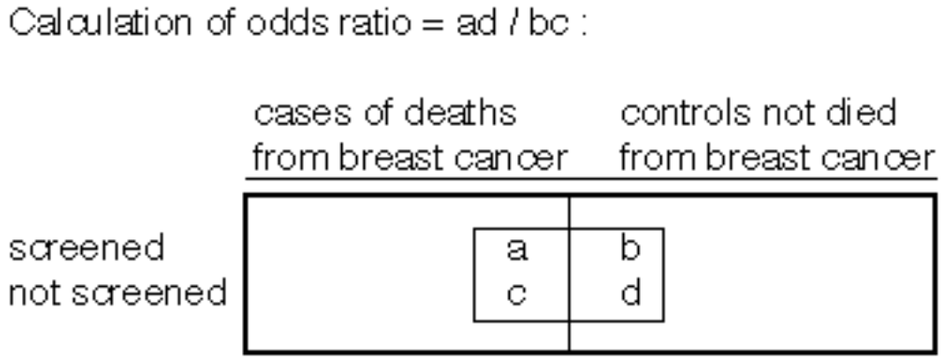
Notes:

Calculation of odds ratio = ad / bc :



Notes:

WATER-SOLUBLE CONTRAST MEDIA
Ionic=dissociation in waterNonionic=soluble in water (hydrophilic); no dissociation in solutionIodine-to-particle ratio =quotient of iodine atoms (attenuation of x rays) and number of particles (osmotoxic effect)ratio 1.5 agents=high-osmolar contrast media (HOCM)ratio 3.0 agents=low-osmolar contrast media (LOCM)ratio 6.0 agents=isotonic contrast media (IOCM)

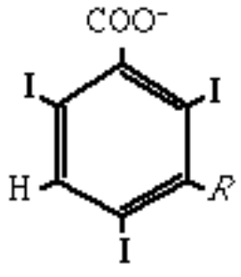
Physicochemical Properties of Commonly Used Radiographic Contrast Media

Contrast Media	*Compound*	*mOsm/kg H₂O*	*Viscosity (cp) at 37°C*	*Iodine mg/mL*
Ionic monomers				
Renografin®-60 (Squibb)	Na-meglumine diatrizoate	1420	4	292
Hypaque®-60 (Sanofi Winthrop)	Na-meglumine diatrizoate	1415	4	282
Conray®-60 (Mallinckrodt)	Meglumine-iothalamate	1500	4	282
Ionic dimers				
Hexabrix® (Mallinckrodt)	Na-meglumine ioxaglate	600	7.5	320
Nonionic monomers				
Omnipaque®300 (Sanofi Winthrop)	iohexol	672	6.3	300
Isovue®300 (Squibb)	iopamidol	616	4.7	300
Optiray®320 (Mallinckrodt)	ioversol	702	5.8	320
Nonionic dimers				
Isovol®300 (Schering AG)	iotrolan	~310	9.1	300

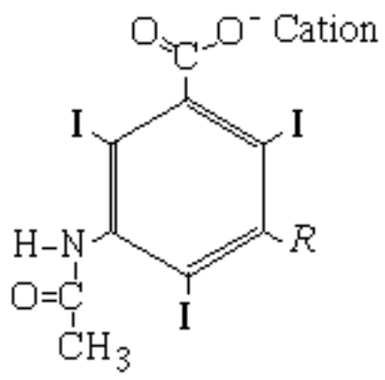
Δ Osmolality of human serum is 290 mOsm/kg!
Δ The higher the number of hydroxyl groups, the larger the size + the higher the viscosity + the higher the hydrophilicity! This decreases protein- and tissue-binding properties making the compound biologically more inert!

Notes:

IONIC MONOMERS
=monoacidic salts composed of benzoic acid derivatives, with 3 hydrogen atoms replaced by iodine atoms + 3 hydrogen atoms replaced by simple amide chainsin solution: strong organic acid completely dissociated (ionized) into negatively charged ions / anions Conjugated cations: (1)sodium(2)methylglucamine (meglumine)(3)combination of aboveIodine concentration:up to 400 mg/mLIodine-to-particle ratio: 3:2 or 1.5:1Osmolality:1400-2100 mOsm/kg = HOCM



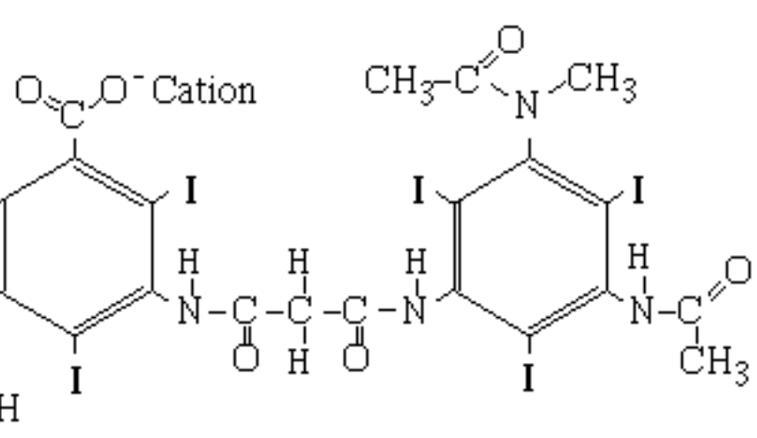
Acetrizoate
The parent triiodinated contrast medium in first clinical use; the benzene ring is attached to a carboxyl (COO-) group at the 1-carbon position and conjugated with sodium / meglumine



Diatrizoate
The unsubstituted hydrogen of acetrizoate has been exchanged for another acetamido unit leading to higher biologic tolerance through higher degree of protein binding

Notes:

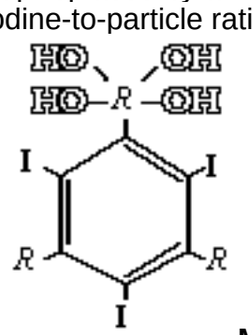
IONIC DIMERS
Construction: 2 iodinated benzene rings containing 6 iodine atoms, one of which contains an ionizing carboxyl group; benzene rings are connected by a common amide side chain Conjugation with:sodium + meglumineCompound:ioxaglate (the only available)Iodine concentration:320 mg/mLIodine-to-particle ratio:6:2 or 3:1Osmolality:600 mOsm/kg = LOCM



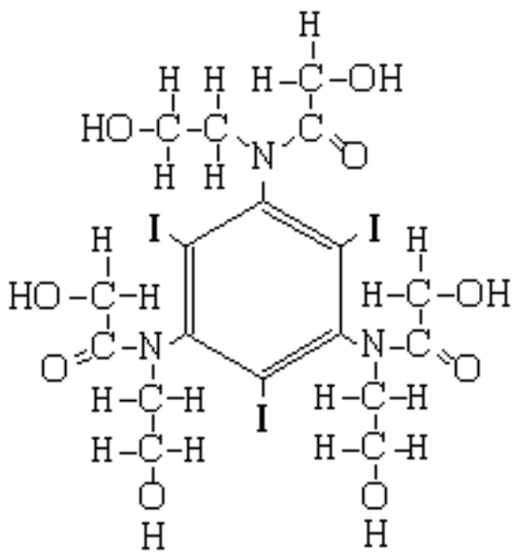
Ioxaglate (Hexabrix®)
Sodium + meglumine are conjugated with the carboxyl group.

Notes:

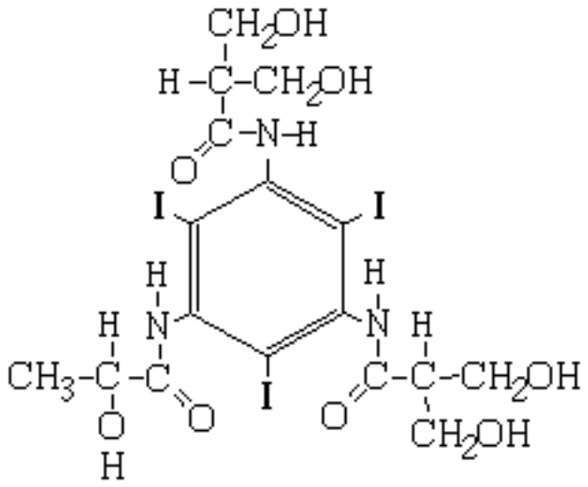
NONIONIC MONOMERS
Construction: benzoic acid carboxyl group replaced by amide; side chains have been modified by adding 4-6 hydroxyl (OH) groups which allows solubility in water
Iodine concentration:up to 350 mg/mLIodine-to-particle ratio:3:1Compounds:iohexol, iopamidol, ioversol, iopental, iopromide (Ultravist®), iobitridol (Xenetix®), ioxilan



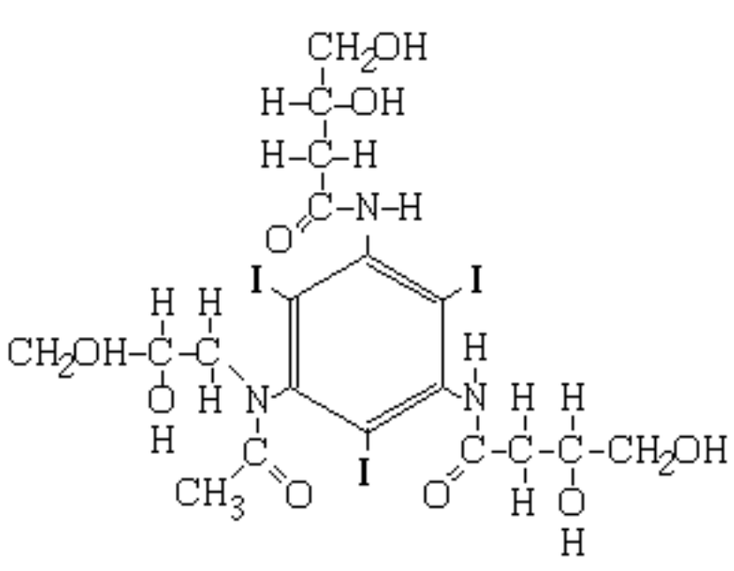
(Oxilan®)Osmolality:616-796 mOsm/kg **Metrizamide** The first compound with 4 hydroxyl groups positioned at one end of the molecule on the glucosamide moiety.



Iohexol (Omnipaque®)
contains 6 hydroxyl (OH) groups more evenly distributed around the molecule improving subarachnoid toxicity.



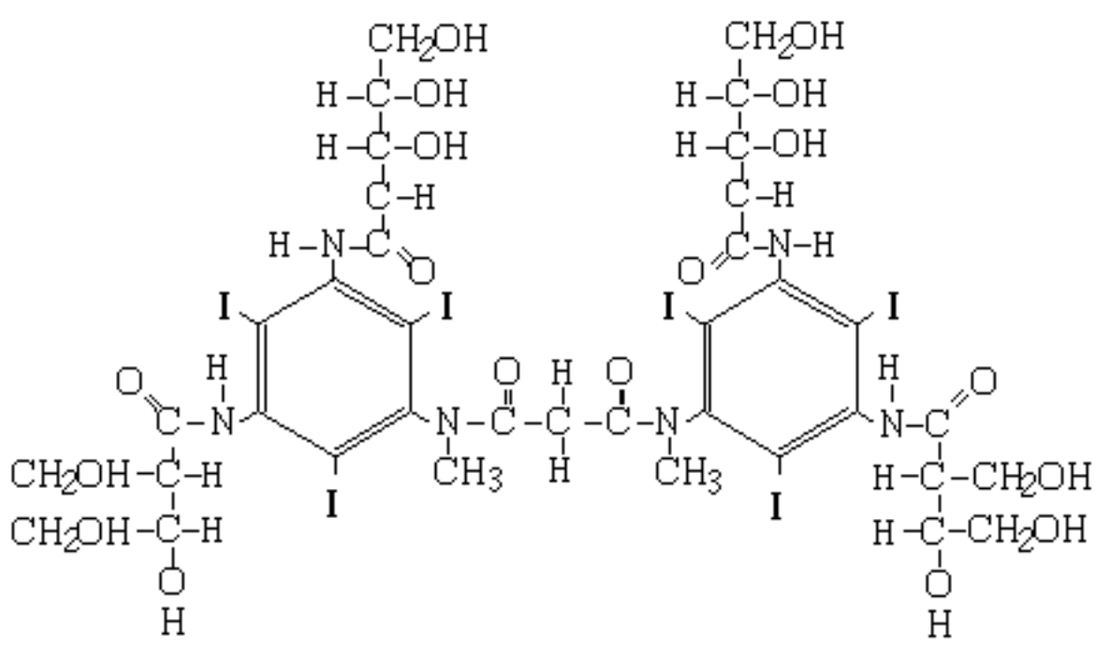
Iopamidol (Isovue®)
This nonionic monomer contains 5 hydroxyl (OH) groups.



Ioversol (Optiray®)
This nonionic monomer contains 6 hydroxyl (OH) groups.

Notes:

NONIONIC DIMERS
Construction: contain up to 12 hydroxyl groups to eliminate ionicity, increase hydrophilicity, lower osmotoxicity, and increase iodine atoms per molecule
Compounds:iodecol, iotrolan (Isovist®), iodixanol (Visipaque®)Iodine-to-particle ratio: 6:1Osmolality:hypo- / isoosmolar



Iotrolan (Iotrol®)
This nonionic dimer contains 12 hydroxyl (OH) groups.

Notes:

ADVERSE CONTRAST REACTIONS

A.Nonidiosyncratic (= dose-related) reactions*Cause:*direct chemotoxic / hyperosmolar effect ● nausea, vomiting ● cardiac arrhythmia ● renal failure ● pulmonary edema ● cardiovascular collapse B.Idiosyncratic (= anaphylactoid) reactions*Cause:*unknown ● hives, itching ● facial / laryngeal edema ● bronchospasm, respiratory collapse ● circulatory collapse C.Delayed reactions ● erythematous rashes, pruritus ● fever, chills, flulike symptoms ● joint pain ● loss of appetite, taste disturbance ● headache, fatigue, depression ● abdominal pain, constipation, diarrhea **Risk Factors and Incidence of Adverse Reactions for High- and Low-Osmolality Contrast Media**

Type of ReactionHOCMLOCM(%)(%)Overall incidence Australia (Palmer et al.)3.801.20United States (Wolf et al.)4.200.70Japan (Katayama et al.)12.703.10Severe adverse reactions0.220.04Severe allergies to drugs, foods, etc.23.406.90Asthma19.707.80Repeat reaction to contrast media16-444.1-11.2Significant underlying medical conditions (a)renal disease(b)cardiac disease(c)blood dyscrasias(d)pheochromocytoma ◊Approximately 20-40% of population are at increased risk for adverse reaction to contrast media!◊Mortality rates from contrast reactions are too small for both HOCM + LOCM to be statistically significant!

USEFUL MEDICATIONS: DERMAL CONTRAST REACTION RESPIRATORY DISTRESS ANAPHYLACTOID REACTION VASOVAGAL REACTION TREATMENT OF PREMEDICATED PATIENTS STEROID PREMEDICATION PROTOCOL

Notes:

USEFUL MEDICATIONS:

1.Alpha- and b-adrenergic agentAction:vasoconstriction, increased cardiac output 1 mL glass vials of epinephrine 1:1000 prepackaged 10-mL syringes of epinephrine 1:10,000*Cx:*arrhythmia, myocardial ischemia, nausea, vomiting, tremulousness, headache2.Atropine*Cx:*angina, myocardial infarction3.Metered-dose inhalers of b-adrenergic bronchodilators metaproterenol terbutaline4.H_2 antagonists = antihistamines diphenhydramine hydroxyzine5.Aminophylline 250 mg in 10 mL of 5% dextrose6.Sedatives Demerol*Cx:*respiratory depression7.Volume expander crystalloid solution as 0.9% saline hydroxylethyl starch (high-molecular-weight colloid)8.Dopamine9.Oxygen (administered by nasal prongs / mask)

Notes:

DERMAL CONTRAST REACTION

● hives = urticaria ● itching = pruritus ● flushing ● facial angioedema (= nonpruritic SQ edema of eyelid / peroral)A.MILDNone (scattered hives do not require treatment!) B.IRRITATING○50 mg diphenhydramine PO / IMor ○25 mg hydroxyzine PO / IMC.SEVERE URTICARIA○diphenhydramine / hydroxyzine○0.3 mL epinephrine (1:1000) SQ○IV line started + kept open (with normal saline / Ringers lactate)

Notes:

VASOVAGAL REACTION

● sinus bradycardia (pulse <60) + hypotension (systolic blood pressure <80 mmHg) ● dizziness, diaphoresis ● loss of consciousness ▫ deflate balloon + remove tip (if during BE) ▫ Trendelenburg position + leg elevation ▫ rapid IV infusion of 0.9% saline / volume expander if symptoms persist, add: ▫ 0.5-0.7 mg atropine IV every 5 min up to 2-3 mg

Notes:

TREATMENT OF PREMEDICATED PATIENTS

A.Patient on b-blockerif response to epinephrine inadequate ▫1-5 mg glucagon IV + subsequent slow drip of 5 mg glucagon over 60 minB.Patient on calcium channel blocker (eg, nifedipine, nicardipine)▫calcium IVC.Excessive vasoconstriction on epinephrine IV▫3 mg/kg/min of reconstituted sodium nitroprusside (50 mg in 500-1000 mL of 5% dextrose wrapped in metal foil during use to protect solution from light)

Notes:

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STEROID PREMEDICATION PROTOCOL

32 mg methylprednisolone PO 12 and 2 hoursprior to IV contrast administration*Indication:*previous respiratory adverse contrast reaction, history of significant allergies / severe asthmaCaution in patients with:active tuberculosis, diabetes mellitus, peptic ulcer disease

Notes:

Nonoliguric Transient Renal Dysfunction =transient decline of renal function ● serum creatinine level peaks on days 3-5 ● serum creatinine returns to baseline values within 14-21 days ● fractional excretion of sodium <0.01 (DISTINCTIVE CHARACTERISTIC compared with other causes)

Notes:

Acute Renal Failure =sudden + rapid deterioration of renal function=increase in serum creatinine of >25% or to >2 mg/dL within 2 days of receiving contrast material*Frequency:*1-30%; 3rd most common cause of in-hospital renal failure after hypotension and surgery*Risk factors:* 1.Preexisting renal insufficiency (serum creatinine >1.5 mg/dL)2.Diabetes mellitus (possibly related to dehydration / hyperuricemia)3.Dehydration4.Cardiovascular disease5.Use of diuretics6.Advanced age >70 years7.Multiple myeloma (in dehydrated patients)8.Hypertension9.Hyperuricemia / uricosuria*Highest risk:*diabetics with renal insufficiency (ratio 3 nonionic LOCM appear to be 50% less nephrotoxic than ratio 1.5 ionic HOCM)CAVE: ◊Small decreases in renal function may greatly exacerbate the mortality caused by the underlying condition!◊Metformin (Glucophage®) should be discontinued for 48 hours after contrast medium administration (accumulation of metformin may result in lactic acidosis which is fatal in 50%)!*Proposed mechanisms:* 1.Vasoconstriction(a)increase in intrarenal pressure induced by hypertonicity(b)intrarenal smooth muscle contraction in response to hypertonic substances2.RBC aggregation in medullary circulation3.Direct tubular cell injury *Potential antidotes:* Hydration (0.45% saline at 100 mL/h) 12 hours before + 12 hours after angiography √ immediate dense nephrogram persisting for up to 24 hours (in 75%)√ gradually increasing dense nephrogram resembling bilateral acute ureteral obstruction (in 25%)√ bilaterally enlarged smooth kidneys√ poor opacification of urine-conducting structures√ effacement of collecting system (interstitial edema)*Cx:*34% mortality (0.4% of all patients)*Rx:*0.1% require renal replacement therapy

Notes:

Notes:

CALIBRATORS

Constancy = Precision Linearity Accuracy Geometry

Notes:

DISLOCATION

Atlanto-occipital Dislocation=ATLANTO-OCCIPITAL DISTRACTION INJURY

Notes:

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GASTROINTESTINAL SCINTIGRAPHY

Radionuclide Esophagogram Gastroesophageal Reflux Gastric Emptying Gastrointestinal Bleeding Levine / Denver Shunt Patency

Notes:

RADIOPHARMACEUTICALS

Radionuclide Impurity Radiochemical Impurity Chemical Impurity Pyrogen Testing

Notes:

SCINTILLATION CAMERA

Spatial Resolution / Linearity

Notes:

PARATHYROID SCINTIGRAPHY

Technetium-thallium Subtraction Imaging Technetium-99m Sestamibi

Notes:

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PERFUSION AGENTS

Tc-99m Macroaggregated Albumin (MAA) Tc-99m Human Albumin Microspheres

Notes:

TUMOR IMAGING

Positron Emission Tomography

Notes:

BLOOD POOL AGENTS

Tc-99m DTPA / Tc-99m Sulphur Colloid Tc-99m-labeled RBCs Tc-99m HSA

Notes:

MYOCARDIAL PERFUSION IMAGING AGENTS

Potassium-43 Thallium-201 Chloride Tc-99m MIBI (Sestamibi) Tc-99m Teboroxime Tc-99m Tetrofosmin Positron Emission Tomography

Notes:

VENTRICULAR FUNCTION

First-pass Ventriculography Equilibrium Images Gated Blood Pool Imaging

Notes:

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NEPHROTOXICITY

Nonoliguric Transient Renal Dysfunction Acute Renal Failure

Notes:

LIPPINCOTT WILLIAMS & WILKINS
A Wolters Kluwer Company

Potassium-43 Not suitable for clinical use because of its high energy

Notes:

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