JOHN E TURRENTINE

CLINICAL PROTOCOLS in OBSTETRICS and GYNECOLOGY

THIRD EDITION

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Foreword

What a pleasure it would have been in 'my day' to have this type of book to utilize before taking the Boards. Also, there were many times during my practice when I needed a fast refresher on symptoms presented that were not commonplace for my practice. Regardless of the length or the volume of any one doctor's practice, there are always questions that demand confirmation on one's memory of those details. This book can easily fulfill each of those needs.

This is an excellent compilation of the up-to-date knowledge on almost any topic within Obstetrics and Gynecology. It could be used for studying for medical school exams and written and oral boards, for research on subjects that do not occur in a particular practice very often, and for a complete and very user-friendly resource for any Obstetric and Gynecology library. It would be particularly valuable within a residency program for rapid access to details of a certain diagnosis. The book is organized in a simple, yet in-depth way to find the pertinent information desired – topic, subjects within that topic – symptoms, diagnosis, treatments – and then the details and statistics. Looking up a topic is as logical and easy as using a dictionary.

During the time that John and I practiced together, he was well-trained in surgery and fully knowledgeable and enthusiastic about specifics and diagnostic details in the Obstetrics and Gynecologic field, always researching the most up-to-date study results and protocols. He taught medical field students at Dalton State College and did excellent, in-depth presentations for various pharmaceutical companies. His expertise in diagnosing, surgery, research and teaching provided an ideal basis for creating this excellent resource for Obstetrics and Gynecology students, residents and physicians. It is a pleasure watching John use these talents to benefit others who follow in the field.

Sidney L Sellers MD
Obstetrics and Gynecology
Dalton, GA, USA
Practiced 1957 – 2006 at the
Emory School of Medicine,
Atlanta, GA, USA

About the Author

Dr John E Turrentine is a Clinical Professor of Obstetrics and Gynecology for the Medical College of Georgia and Director/Instructor for the Dalton State College of Surgical Technology. He is an avid lecturer and instructor for minimally invasive surgeries, especially in the area of total laparoscopic hysterectomy, female urinary incontinent and prolapse procedures, and most significantly is the innovator and expert on MIVH (minimally invasive vaginal hysterectomy). Having been in Ob/Gyn practice for over 25 years, Dr Turrentine teaches other physicians how to pass the ACOG Board Certifying Exams.

Dr Turrentine received his Medical Doctorate from the Medical College of Georgia and is a Doctor of Ministry from the Trinity Theological Seminary. He is a Board Certified Diplomat and Fellow of the American Board of Obstetrics and Gynecology. He is an Ordained Minister through FCF (Faith Christian Fellowship). He has served on multiple boards, including chairmanship positions for the ACOG Satellite Symposium, Young Life, and the Appalachian Women's Enrichment Center for the Pregnancy Crisis Centers throughout North Carolina, Tennessee, and North Georgia. He has been featured on MSNBC, PAX TV, MCG Alumni Magazine, and other TV, Newspaper, and Magazines.

Dr Turrentine is recognized worldwide for his previous books, *Clinical Protocols in Obstetrics and Gynecology* (The TAN Book) 1st and 2nd editions, *Surgical Transcriptions in Obstetrics and Gynecology* and *Surgical Transcriptions and Pearls in Obstetrics and Gynecology*.

Dr Turrentine's primary love is his family. His family includes a supportive wife, a son in medical school, another son in pre-law, a daughter studying for a horticultural degree, and another daughter still at home. His other interests include teaching history, ethics, philosophy, swimming, running, and hiking. He holds a private pilot certificate, including seaplane rating, and is a current SSI and PADI Master Diver and Divemaster.

Introduction

This book is the most up-to-date Ob/Gyn textbook compiled to help anyone both pass the ACOG Written or Oral Board Examinations and also use as a reference while practicing Ob/Gyn. It is in simple alphabetical order so it is easy to find solutions to everyday problems. Every effort was made to list the main topics on the left-hand side of the page. The "meat" of the matter or subject has been listed in the middle, including etiologies, symptoms, diagnoses, and treatment modes. To the far right, whenever possible, answers to percentages or minutia have been listed, so this book makes an excellent study guide.

The same and sometimes improved flow charts and pictures are included that made the TAN book a best-selling medical textbook. These all make for a quick reference and study guide. If you know this book, you WILL pass your certification exam. You will also practice excellent Ob/Gyn.

Notice to readers

Our knowledge in clinical sciences is constantly changing. As new information becomes available, changes in treatment and in the use of drugs become necessary. The author and publisher of this volume have taken care to make certain that the doses of drugs and schedules of treatment are correct and compatible with the standards generally accepted at the time of publication. The reader is advised to consult carefully the instruction and information material included in the package insert of each drug or therapeutic agent before administration. This advice is especially important when using new or infrequently used drugs.

ABDOMINAL PREGNANCY

Incidence is: 1/7000
Signs: amenorrhea, abdominal pain, poor response to oxytocin

ABDOMINAL SACRAL COLPOPEXY

See also Prolapse (POP)
Success rate 90%
Have these available in OR – sterile thumbtacks and/or bone wax
Complications – hemorrhage, enterocele, mesh erosion
Identify ureter, especially on right
Retract rectosigmoid colon laterally
Vascular plexus – on sacral periosteum BLEEDS
RETROPERITONEALIZE FASCIA LATA (or Mersilene, Marlex, etc.)
Use 'straight-in' sacral colpopexy kit with 'Y' sling graft by American Medical (1 800 253-4267)
To see how this procedure is done, refer to Turrentine J. *Surgical Transcriptions and Pearls of Obstetrics and Gynecology*, 2nd edn. London: Informa Healthcare, 2006.

ABDOMINAL WALL

Layers

Skin

Subcutaneous fat
Camper's fascia (superficial fascia)
Scarpa's fascia (deep fascia)
Anterior rectus sheath (fascial muscle cover)
Preperitoneal fat
Peritoneum

ABORTIONS

Therapeutic

Mifepristone (RU486) approved in the USA for voluntary termination of IUP of up to 7 weeks (49 days from LMP)

Method
Day 1 Counseling, especially about 5% failure rate and possible need for surgical intervention. Malformations if continued pregnancy after failure. Patient to sign PATIENT AGREEMENT and/or CONSENT. Know or review contraindications. Then, 600 mg (three tablets of 200 mg each) given as single oral dose. This administration should be witnessed and done while in office.
Day 3 Misoprostol 400 µg (two tablets of 200 µg each) given as single oral dose (unless abortion has occurred and been documented by exam and ultrasound). Patient usually given something for cramping
Day 14 Post-treatment follow-up (persistent or enlarging sac requires surgery for removal)

Medical abortion **(if RU486 not available)** Misoprostol 800 µg

If uncertain about location give misoprostol 5 days after Mtx 1 mg/kg

Ectopic Mtx alone IUP Cytotec (misoprostol 800 µg) alone or Cytotec 800 µg then mifepristone 600 mg (RU486) 36–48 h later or as described above

Misoprostol 400 µg every 6 h for ≤ 48 h appears to be an effective regimen for second-trimester pregnancy termination, resulting in a shortened delivery time. (Dickinson JE, Evans SF. Optimization of intravaginal misoprostol dosing schedules in second-trimester pregnancy termination. *Am J Obstet Gynecol* 2002;186:470–4)

Surgical abortion (discouraged if < 6 weeks – increased risk of incomplete evacuation, ectopic)
Difficulty with cannula? Use laminaria, Cytotec or rotation of tip of dilator
Labs – Rh p.r.n., Hct, pregnancy test, STD?, Paps
Anesthesia
(1) Give Lortab® 5 or Percocet® 5 AND Xanax® 0.5 mg p.o. 30 min prior
(2) Give Valium® 10 mg with lidocaine 20 mg IV through butterfly and Nubain® 10 mg IV just prior to start of procedure

Paracervical 7, 9, 11, 1, 3, 5 o'clock or simply inject 6 cc of 1% lidocaine at 5 and 7 o'clock
Local increases postabortal fever
General increases death, perforation, bleeding and aspiration
Diprivan® is all that is usually needed if patient desires sleep
Selection of cannula #8 for 8 weeks; straight for decreased pain, curved for ante- or retroflexed uterus
Postop meds
RhoGAM < 12 weeks; MICRhoGAM (50 µg) > 12 weeks full dose
Doxycycline 100 mg p.o. b.i.d. for a few days postoperatively
Methergine® not needed unless > 10 weeks' gestation
NSAIDs for postop discomfort

Types of abortion

Threatened abortion — Bleeding, os closed

Inevitable abortion — Bleeding, os open, no POC passed

Incomplete abortion — Bleeding, os open, some POC visualized

Complete abortion — Bleeding, os closed, all POC extruded

Missed abortion — No viable fetus, no bleeding, no symptoms
Surgical evacuation of the uterus via D&C is not obligatory for first-trimester missed abortion (Wood SL, Brain PH. Medical management of missed abortion: a randomized clinical trial. *Obstet Gynecol* 2002;99:563–6)

Therapeutic abortion — Elective termination

Septic abortion — Any SAB or TAB with intrauterine infection

Usually due to clostridial sepsis
Presents with tachycardia and FEVER
Hematuria and shock develop rapidly
Dxn; H&P, cultures by endobiopsy or evacuation +++gm + rods on Gm stain.
Check serum pregnancy test
Rx:

(1) High dose ab – PCN
(2) Empty uterus – first-trimester vacuum
second-trimester D&E with US or use PGE
(3) Laparotomy p.r.n.
(4) Hysterectomy with BSO (if hemolysis or systemic)
(5) Hyperbaric oxygenation p.r.n.
(6) Supportive care – ICU – cardiovascular support to restore B/P treat ARDS (ventilation if O_2 < 90%)

Spontaneous abortion

Clinically recognized	10–15%
Lost in first or early second trimester	15–20%
Lost prior to menses	50–75%
Likelihood of fetomaternal hemorrhage after spontaneous abortion	3–4%
Percent of spontaneous first-trimester abortions that show chromosomal abnormalities	50–70%
Percent of spontaneous second-trimester abortions that show chromosomal abnormalities	30%
Percent of stillbirths that show chromosomal abnormalities	3%
Autosomal trisomy is detected in abnormal abortuses	50–60%
Trisomy 16 is most common of autosomal trisomies	
Turner's (XO) is most common single entity detected	20–25%
Polyploid (usually triploid)	44%
Most likely cause of spontaneous abortions is embryonic aneuploidy if prior to	6th week
Most likely cause of spontaneous abortions is lupus anticoagulant syndrome if after	11th week
Rate of pregnancy loss after detecting a live embryo in the first trimester is	< 5%
Risk of subsequent pregnancy ending in a spontaneous or spontaneous recurrent abortion:	
No previous abortion	10.7%
One previous abortion	15.9%

Two previous abortions	25.1%
Three previous abortions	45%
Four previous abortions	54.3%
Overall	11.3%
What % of elective abortions are second-trimester abortions?	10%
What is the appropriate vacuum for evacuating an incomplete abortion in the first trimester?	40 mmHg
To undertake an elective abortion at 10 *menstrual weeks'* gestation, correct cannula size is	8 mm
What period of time does one have to give RhoGAM immunoglobulin (RHIG) prophylaxis if not given within 72 h of delivery or abortion?	28 days
Incidence of vaginal bleeding in first trimester	20%
Risk of miscarriage in patient with first-trimester bleeding	1/2 to 2/3
FHR per US – incidence of spontaneous abortion with first-trimester bleeding is only	10%
US with no FHR is indicative of fetal demise if sac is	> 1.2 cm
Risk of combined IUP and ectopic is	1/8000–1/30 000
RhoGAM < 12 weeks' gestation	MICRhoGAM® (50 µg)
> 12 weeks' gestation	Full dose RhoGAM®

Most likely organisms to cause postabortal endometritis are *Neisseria gonorrheae, Chlamydia* and *Streptococcus*
Treat endometritis with doxycycline, ofloxacin and/or ceftriaxone

Habitual abortions

Causes	*Diagnosis*	*Treatment*
Immunologic	APTT, lupus, VDRL, antiphos abs	Heparin, ASA, prednisone
Microbiologic	Cervical and endometrial cultures	Tetracycline, emycin
Endocrinologic	Endo Bx, TSH, prolactin, midcycle progesterone, BBT charting	Clomid®, progesterone, thyroid, bromocriptine
Genetics	Karyotype	Genetic counseling, donor insemination, IVF
Anatomic	HSG, laparoscopy, hysteroscopy	Septum, cerclage, lyse synechia, myomectomy, metroplasty, tuboplasty, IVF
Metabolic	As indicated	As indicated
Environmental	Tobacco, EtOH abuse	Eliminate consumption or exposure

Common genetic causes of RPW (recurrent pregnancy wastage)

Aneuploidy
Chromosomal translocation – most common structural abnormality

CPM (confined placental mosaics)	1–2%

Carriers of factors Leiden – increased risk of venous thromboembolism

Anatomic anomalies of RPW

Unicornuate uterus – rate of spontaneous pregnancy loss is	51%
Uterine didelphys – rate of spontaneous pregnancy loss is	40%
Bicornuate uterus – rate of spontaneous pregnancy loss is	30%
Septate uterus – rate of spontaneous pregnancy loss is	65%
Resection of the septum results in the successful delivery rate of	86%
Asherman's syndrome – pregnancy rates of untreated is	45%
Hysteroscopic resection of Asherman's – rate of conception is	84%

Endocrine factors of RPW

Luteal phase defect
Uncontrolled diabetes
Thyroid disease
Hyperprolactinemia
Hyperandrogenemia

Immunologic factors of RPW

Autoimmunity

Antiphospholipid antibodies – implicated in Increased platelets aggregation, decreased endogenous anticoagulant activity, increased thrombosis and vasoconstriction resulting from immunoglobin binding to both platelet and endothelial membrane phospholipid. Screen patients with RPW by drawing – APTT, kaolin clotting time, lupus anticoagulant and cardiolipin ab. 10–16%

Treat with heparin and low-dose aspirin… pregnancy achieved in 70%

Alloimmunity

Refers to all causes of pregnancy loss related to an abnormal maternal immune response to antigens on placental or fetal tissues. Suggested that couples with RPW have sharing of HLA (human leukocyte antigens), a condition that would not allow the mother to make blocking antibodies. Treatment – IV immune globulin ??

Partial birth abortion > 16 weeks – 5.5%

May be the best or most appropriate procedure to save the life or preserve the health of the patient
Must have ALL four elements in sequence:

(1) Deliberate dilatation of cervix, usually > sequence of days
(2) Instrumental conversion of fetus to footling breech
(3) Breech extraction of body except the head, AND
(4) Partial evacuation of the intracranial contents of a living fetus to effect vaginal delivery of a dead but otherwise intact fetus

Incomplete and/or recurrent abortion

< 12 weeks

H&H, WBC, Group & Rh
Fibrinogen and platelets
D&E
D/c 6–8 h postop if stable with minimal bleeding
F/u 2 weeks

13–28 weeks

Offer watchful expectancy at least x 3 weeks (> 4 weeks 25–40% DIC) ***or*** PGE_2; (D&E okay if experienced)
CBC, fibrinogen, platelets, Group & Rh
Type & screen
NPO night before
Repeat PGE_2 q. 4 h
D5½ NS
Demerol® 25 mg IV q. 3 h p.r.n.
Phenergan® 25 mg IV q. 4 h or Zofran 8 mg subling p.r.n. nausea
6 h postop – H&H, fibrinogen level
If USS – d/c x 24 h – RTO in 2 weeks

> 28 weeks

CBC w/ platelets, Group and Rh, fibrinogen, Type & cross 2 units;
D5½
Pitocin® or Cytotec ***or*** with PGE_2 prior to Pitocin
US q. h
Stillbirth protocol (photos, opportunity to view and hold)
Request autopsy
Hct & fibrinogen
If USS – d/c x 24 h – RTO x 2 weeks

Recurrent pregnancy loss – sample form

Name _____

	Normal	*Significant results*
Genetic		
Karyotype partners		
Genetics on POC		
Anatomic		
Hysterosalpingography		
Laparoscopy		
Hysteroscopy		
Endocrinologic		
Basal body temperature		
Endometrial biopsy		
Mid-luteal progesterone		
TSH		
Prolactin		
Immunologic		
Lupus anticoagulant		
ANA		
Anticardiolipin antibodies		
VDRL		
APTT		
APA		
APLA (antiphospholipids)		
Infectious		
Mycoplasma hominis		
Ureaplasma urealyticum		
Toxoplasma gondii		
Listeria		
Chlamydia		
GBBS		
Titers for:		
HSV		
CMV		
Toxoplasmosis		
Metabolic		
Panel I		
Toxins		
Nicotine		
Drugs		
EtOH		

This form may be used in the patient's chart

ABRUPTIONS

	Separation of normally implanted placenta, usually after 20 weeks, initiated by bleeding into decidua basalis.	
	Incidence of occurrence is	(@1%) or 1/120
	Fatal to fetus	1/420
Etiology	Caused by increased B/P	50%
	Other causes (cigarettes, cocaine, trauma, short cords, rapid decompression of uterus)	50%
	Mortality rate increases by how much with each cigarette ppd?	40%
	Trauma abruption evolves within	24 h
	Usually asymptomatic for 4–6 h then symptoms	> 24 h
	Cocaine use increases abruption rate by	10%
	Consider physical abuse, which is prevalent during pregnancy	@ 8%
	Hypertensive disorders and history of prior abruptions	
Symptoms	PAINFUL VAGINAL BLEEDING. Fetal distress, abdominal pain, increased uterine tenderness. Darker blood with rigid, sudden, severe sharp pain @ abdomen. 'Tearing or burning'	
	Vaginal bleeding most common presenting sign	78–84%
	Uterine tenderness or back pain (second most common)	62–66%
	Tachysystole	17%
	Uterine hypertonus	17%
Management	Oxygen and crystalloids. C-SECTION if severe	
	Watch for DIC. Obtain FSP every 4 h, replace with FFP or cryo if fibrinogen < 100 mg per 100 ml. Replace platelets if < 50 000	
	Risk of recurrence of abruptio placentae is	5–16%
	Recurrence risk of abruptio placentae rises to what % after two previous abruptions?	25%
	• DIC occurs during abruption what % of time?	10%
	Blood clotting time is	> 8 min
	Most sensitive lab is	FDP
	Delivery is ultimate treatment, blood products seldom needed, DIC persists @ how many hours?	12
	C-section with DIC – replace clotting factors if *platelets	< 30 000
	or *fibrinogen	< 100
	To correct fibrinogen, give cryoprecipitate – how many bags?	15–20
	Platelets – one bag increases platelets @	10 000
	Prevent hypovolemia (maternal death – ischemic damage to kidneys. Sheehan's syndrome – anterior pituitary necrosis)	

Abruptio placentae – summary

Diagnosis

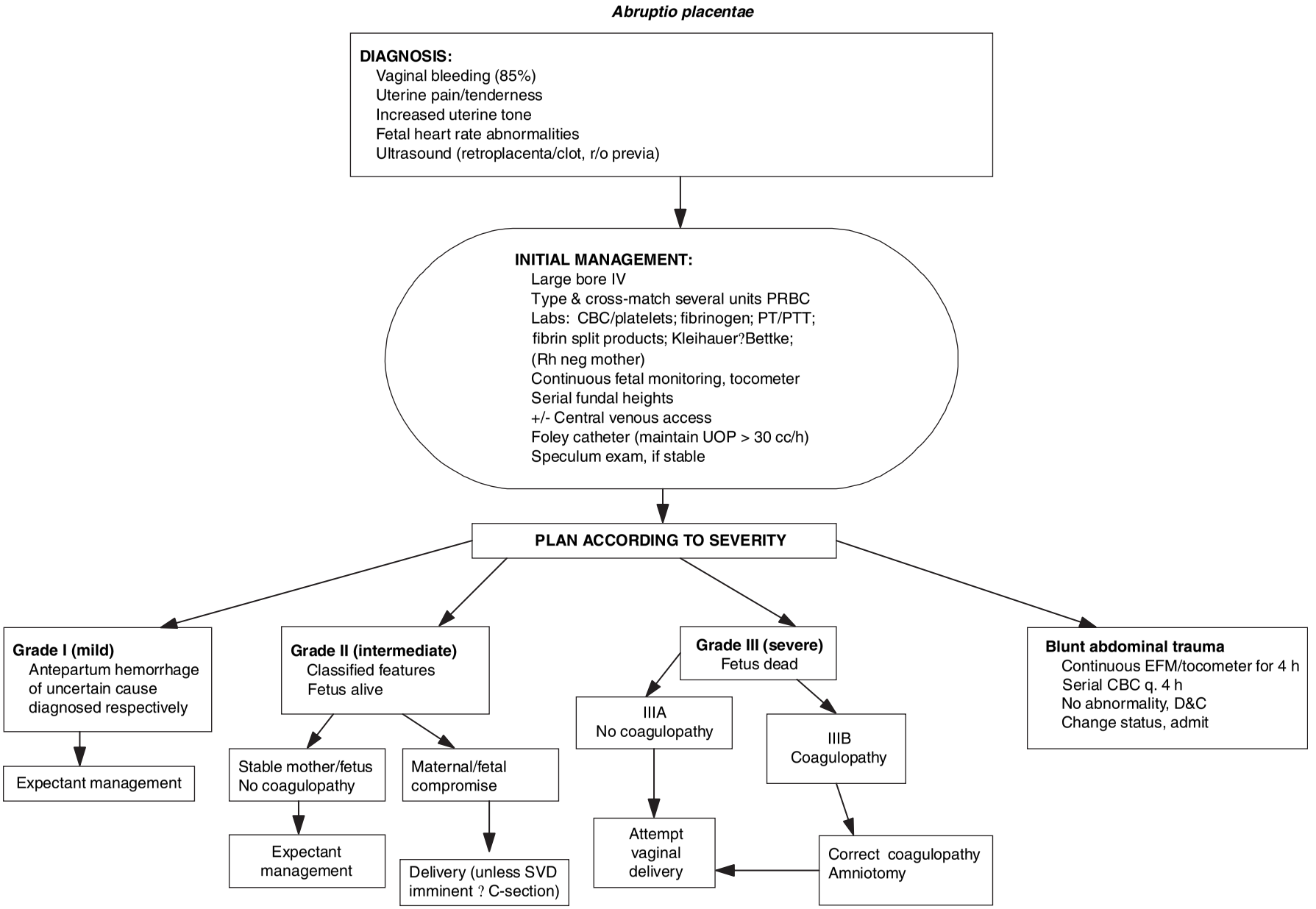
(1) *Clinical symptoms*
Fetal tachycardia/IUFD
Virchow's triad
uterine pain – focal or generalized
increased tone
vaginal bleeding (85%) – 15% concealed
(2) *Imaging* (ultrasound)
Helpful in concealed abruption – sonolucent retroplacental area
Locate placenta (i.e. r/o previa)

Management

(1) Large bore IV (16 or 18 gauge)
– crystalloid – (LR, D5NS)
– can be used for blood transfusion
(2) Type and cross–match 2–4 units PRBC
(3) Labs: CBC w/ platelets; coagulation profile (fibrinogen, PT, PTT, fibrinogen split products); repeat q. 2–3 h
(4) Continuous EFM, tocometer
(5) Measure serial FH (especially concealed abruption)
(6) Consider central venous access (especially when impending or actual shock suspected)
(7) Strict I&Os (UOP > 30 cc/h)
(8) Determine extent of fetal–maternal hemorrhage (i.e. Kleihauer–Bettke)
Rh neg mother – additional RhoGAM (vial > 30 ml)
(9) If stable, spec exam

Plan

(1) Delivery (when possible)
– low threshold for Cesarean section (fetal/maternal indication)
– if rapid vaginal delivery expected, attempt (or fetus dead)
(2) Expectant management
– patient/fetus stable
– no coagulopathy
(3) Correct coagulopathy
– PRBC
– FFP
– cryo precipitate
– platelets
(4) Correct hypovolemia/restore adequate circulation
– rapid infusion crystalloid/cross-matched blood (O neg in emergency)
– maintain Hct > 30%
(5) Avoid incision or episiotomy if possible
– careful hemostasis intrapartum/intra op
(6) Postpartum
– monitor resolution of coagulopathy
– correct anemia, fluid/electrolyte imbalance
– monitor incision/episiotomy site (r/o hematoma)
– strict I&Os


Abruptio placentae
DIAGNOSIS:
Vaginal bleeding (85%)
Uterine pain/tenderness
Increased uterine tone
Fetal heart rate abnormalities
Ultrasound (retroplacenta/clot, r/o previa)
INITIAL MANAGEMENT:
Large bore IV
Type & cross-match several units PRBC
Labs: CBC/platelets; fibrinogen; PT/PTT;
fibrin split products; Kleihauer?Bettke;
(Rh neg mother)
Continuous fetal monitoring, tocometer
Serial fundal heights
+/- Central venous access
Foley catheter (maintain UOP > 30 cc/h)
Speculum exam, if stable
PLAN ACCORDING TO SEVERITY
Grade I (mild)
Antepartum hemorrhage
of uncertain cause
diagnosed respectively
Expectant management
Grade II (intermediate)
Classified features
Fetus alive
Stable mother/fetus
No coagulopathy
Expectant
management
Maternal/fetal
compromise
Delivery (unless SVD
imminent ? C-section)
Grade III (severe)
Fetus dead
IIIA
No coagulopathy
Attempt
vaginal
delivery
IIIB
Coagulopathy
Correct coagulopathy
Amniotomy
Blunt abdominal trauma
Continuous EFM/tocometer for 4 h
Serial CBC q. 4 h
No abnormality, D&C
Change status, admit

ACCRETA

Absence of Nitabuch's layer with invasion of placenta into or through placenta

Management options

(1) If diagnosis is made prior to delivery – have 4 units PRBCs and anesthesiologist at delivery
(2) Hysterectomy – if preservation of uterus not important and/or bleeding is excessive
(3) Oversew defect and treat with Pitocin and an antibiotic
(4) Resection and uterine repair
(5) Leave placenta *in situ* with curettage and cut umbilical cord as short as possible

with placenta previa

(6) Bilateral uterine artery ligation
(7) Internal iliac artery ligation
(8) Pack lower uterine segment × 12 h
(9) Methotrexate – no consensus
(10) Hysterectomy

Incidence

Accreta, increta and percreta	1 : 7000
Accreta, increta, percreta	78%, 17%, 5%
Incidence of accreta in patients who have had previous C-section and previa	25%
Incidence of accreta in patients who have had two previous C-sections and previa	50%
Incidence of accreta in patients who have had multiple previous C-sections	60–65%
What % of pts with placenta previa/accreta will have to have Cesarean hysterectomy	66%

Tocolytics

$MgSO_4$ is agent of choice if placenta previa associated with accreta. Not β-mimetics due to associated tachycardia and decreased blood pressure

ACUTE TUBULAR NECROSIS

Acute blood loss is most common cause of renal failure in Ob
U/A – shows renal tubule cells and red cell casts

Urine Na^+	> 40 mg/l
Urine to plasma ratio	< 3 to 1

Acute cortical necrosis is an end-stage condition following 2–3 weeks of renal failure

ADAPTATIONS IN PREGNANCY

Uterus

Hypertrophy and dilatation 70 g → 1100 g
10 ml → 5 l

Putrescine polyamines that increase @ 13–14 weeks' gestation
Diamine oxidase activity increases @ 13–14 weeks' gestation 1000-fold increase
Catecholamines decrease in placental perfusion (epinephrine and norepinephrine)
Nitric oxide (EDRF) potent vasodilator (97% umbilical vein, 7% umbilical artery)

Cervix

12 × decreased mechanical strength
Hegar's sign – softening of the neck of the cervix

Vagina

Increased thickness of mucosa, loosening of connective tissue, hypertrophy of vaginal muscle, small intermediate cells (navicular cells) and vesicular nuclei without cytoplasm. Increased lactic acid from glycogen 3.5–6

Chadwick sign – violet color of vagina (hyperemia)

Ovaries and fallopian tubes

(1) Relaxin – (A + B chains) $H_1 + H_2$ on chromosome 7
  (a) Concentration – maternal serum = 1000 mg/l
  (b) amniotic fluid = 9 mg/l
  (c) Separation of symphysis
(2) Luteoma – large acidophilic cells of solid tumor
  Female virilized but usually placenta protects by converting to estrogens

(3) Hyperreactio luteinalis – as above but cystic not solid
Usually bilateral and very increased levels of hCG
Fallopian tubes – mucosa is flattened

Abdominal wall and skin

Striae gravidarum – separation of skin with scarring
Diastasis recti – separation of rectus muscle
Chloasma – mask of pregnancy (β-endorphins and α-MSH produced in pituitary) increased
Angioma (vascular spiders) 2/3 white and 10% black
Palmar erythema 2/3 white and 1/3 black
Hyperestrogenemia of pregnancy

Urinary system

Kidney increases in size, GFR increases by 50%
RPF increases, glucosuria, amino acids and water soluble vitamins are increasingly lost. Hydronephrosis and hydroureter common due to compression on left by sigmoid and dextrorotation of the uterus on the right along with right ovarian complex. Progesterone influences the enlarged ureter too

Gastrointestinal tract

Motilin decreases. Pyrosis (heartburn) increases. Epulis (focal swelling of the gums), prolonged gastric-emptying time. Increased hemorrhoids

Liver and gallbladder

Increased alkaline phosphatase (× 2). Leucine amino peptidase increases.
Plasma albumin decreases. Cholinesterase decreases
Gallbladder is sluggish → increased incidence of stones

Eyes

Corneal sensitivity decreases (increasing thickness)
Intraocular pressure decreases (increased PIH)
Findings = Krukenberg spindles

Endocrine

Pituitary enlarges
GH increases from 10 to 28 weeks' gestation. Peaks at 14–15 weeks
Prolactin increases x 10 (amniotic prolactin source = decidua)
TRH and serotonin increases prolactin
PIF (dopamine) inhibits prolactin
Prolactin peaks at (fetal plasma) 35 weeks' gestation
(amniotic fluid) 20–26 weeks' gestation
β-lipotropin → α-lipotropin and β-endorphin (increases)

Thyroid gland

Increases	*Decreases*
TBG	Parathyroid decreases then increases
T_4	DHEA-S
T_3	Cortisol
Free T_4 increases then decreases	
Androstenedione	
Testosterone	
Free cortisol	

No change in TRH and DOC variable

Metabolic changes

Daily caloric intake of a pregnant women is increased by 300–400 kcal over that of a non-pregnant female
Water retention 6.5 liters
3.5 liters associated with fetus, placenta and amniotic fluid
3 liters associated with increased maternal blood volume
Protein – active nitrogen use only 25%
Carbohydrate – HPL stimulates synthesis and secretion of insulin in the islet cells. Progesterone increases basal insulin concentration.
Estradiol → hyperinsulinism
Fat metabolism – LDL peaks at 36 weeks. HDL peaks at 25 weeks.
Progesterone acts as lipostat in hypothalamus to reset
Minerals – Fe^+ requirements increase
Ca^+ and Mg^+ decrease
Cu increases then decreases
Acid–base – hyperventilation → respiratory alkalosis. Oxygen curve shifts to left. Bohr effect → stimulates increase of 2,3 diphospho-glycerate in maternal RBCs → O_2 curve back to right → O_2 to fetus
Electrolytes – Na^+ and K^+ decrease. GFR increases. Progesterone counteracts Na^+ and K^+ effects of aldosterone

Hematologic changes

(1) Blood volume increases by 40–45%
More plasma than RBCs. Nevertheless, volume of circulatory RBCs increase @ 33% (@450 ml).
Reticulocyte count increases > 20 weeks
(2) Atrial natriuretic peptides (ANP) – increase renal blood flow and GFR decreases renin secretion → decreases basal release of aldosterone from zona glomerulus
(3) Brain natriuretic peptide (BNP) – more potent than ANP as vasodilator. Secreted in large amounts by human amnion cells

Hemoglobin and hematocrit decreases
White blood cells count decreases. However, human antibodies decrease, therefore WBCs increase

Blood loss: vaginal delivery 500–600 ml
C-section 1000 ml

Increased	*Decreased*
Fibrinogen (50%)	Factors 11 and 13
Sed rate	Platelets (moderate)
Factors 7, 8, 9, 10	Free protein S
Factor 2 (slightly)	Plasmin
Plasminogen	

No change in total protein S, C and antithrombin III

Iron metabolism

(1) Fe^{+} needed to increase 450 ml RBCs in normal pregnancy 500 mg
(2) Total Fe^{+} antepartum needed 1000 mg
(3) Amount of Fe^{+} excreted every day in absence of bleeding 0.5–1 mg
(4) Fe^{+} content of normal fetus 300 mg
(5) Amount Fe^{+} in 1 ml normal RBC 1.1 mg
(6) Total Fe^{+} requirements 750–800 mg
Placenta and fetus 300 mg
Increased RBCs 500 mg
(7) Amount of Fe^{+} needed to meet normal demands of fetus AND increase maternal volume 800 mg
(8) Amount of Fe^{+} lost in menses 25 mg
(9) Fe^{+} in 325 mg tablet = 65 element Fe^{+} 10–20% absorbed, therefore 7–12 mg absorbed daily

Cardiovascular changes

Increased	*Decreased*
Resting pulse (17%)	Arterial pressure
Stroke volume	Vascular resistance
Cardiac output (43%)	Systemic (–21%)
Renin	Pulmonary (–34%)
Angiotensin II	
Aldosterone	

Systolic murmur is noted in what % of pregnant patients? 90%
Diastolic (soft) murmur is noted transiently in what %? 20%
Continuous murmur arise in breast vasculature in what %? 10%

Respiratory changes

See Respiratory changes

ADD-BACK THERAPY

Premarin every day or MPA daily on days 1–14
This can be started @ 3 months after the start of GnRH analogs
Affords symptomatic relief and prevents bone loss
NOT TO BE STARTED at same time – efficacy to decrease fibroid is impaired

ADENOCARCINOMA

Uterus

Most common gyn cancer – 4th most common cancer in females
Endometrioid adenocarcinoma (65%) = most frequent histology
OCPs and smoking decrease risk. Tamoxifen increases thickness, polyps and risk of cancer
Black females have increased aggressive histological types (clear cell)

Treatment

Stage I, grade I = TAHBSO with cytology
Deep > 1/3 or grades 2 + 3 = add pelvic + periaortic node dissection
If deep invasion, grade 3, + nodes, extension to cervix, + surgical margins or extrauterine disease = add radiation

Cervix

10% of cervical cancer
Occult lesions
Multifocal/skip lesions
More aggressive than squamous cell carcinoma (90%, begins in T-zone, not multifocal, keratin pearls)

ADENOMYOSIS

Definition

Endometrial glands and stoma invading myometrium by one of the following:

Low-power field	1
High-power field	2
Depth of	3 mm

ADHESIONS

Preventative measures

BEST – gentle handling of tissues, minimize number and extent of incisions, strive for absolute hemostasis, and use small, nonreactive suture

Antibiotics – Cephalosporins and tetracyclines (lavage). Some evidence may be of benefit
Heparin – Controversial
Crystalloid solutions – Normal saline or Ringer's lactate. Unproven. Some animal studies suggest there is an increased adhesion formation
Steroids – Dexamethasone. Possibly decreases inflammatory response, but unproven
Polysaccharide polymer – Dextran 70 (Hyskon) Controversial. 200 ml placed in posterior cul-de-sac or around surfaces. Risks are abdominal bloating, anaphylaxis, liver function abnormalities, wound separation, or rare DIC

Barrier agents
Absorbables (require hemostasis)
INTERCEDE (oxidized regenerated cellulose) 2 x more effective as microsurgery alone
SEPRAFILM (Hyaluronate–carboxymethylcellulose)
Non-absorbables
GORTEX (expanded polytetrafluoroethylene) – must be removed
PRECLUDE (polytetrafluoroethylene). Particularly useful for patients undergoing myomectomy
SHELHIGH NO-REACT (pericardial patch)
Fluid
SEPRACOAT (hyaluronic acid-coat) Limited data on efficacy in myomectomies
INTERGEL™ (dilute solution of hyaluronic acid). Decreases extent + severity of *de novo* adhesions when applied over the serosal surfaces. Withdrawn from market for reports of postoperative pain and complications

Myomectomy

Posterior uterus	94%
Fundal/anterior	56%

Hysterectomy

Bowel obstructions	1.6%

ADNEXAL MASS IN PREGNANCY

Incidence 0.5–2.2%
Most common is leiomyoma
Most common in first trimester is corpus luteum
Most common neoplastic lesion is benign cystic teratoma or cystadenomas
Second most common malignancy in pregnancy is ovarian 1/7500
Common adnexal tumors found during pregnancy:
- Corpus luteum
- Benign neoplasm
- Benign cystic teratoma 27%
- Benign cystadenoma 33%
- Uterine leiomyoma 1.5%
- Malignancy (10% of adnexal tumors that persist during pregnancy)

Diagnosis
US (MRI if equivocal)
The serum CA-125 level is typically elevated during the 1st trimester, but may be useful for assessment later in pregnancy

Treatment
Surgery 16–20 weeks is ideal
During the first and second trimesters, laparoscopy is as safe as laparotomy. However, in general, if malignancy is suspected, a vertical incision is preferred

Risks
Fetal loss, PTD and infection
< 5 cm @50%
Some ovarian cancers may present acutely, such as a rapidly growing germ-cell tumor or a ruptured and hemorrhaging granulosa-cell tumor

ADOLESCENT DEPRESSION

Third leading cause of death
Male > female
Drug ingestion most frequent method
Firearms – most common method of *completed* suicide among young

Symptoms
Depressed mood
Diminished interest or pleasure
Decreased ability to concentrate or think
May present with symptoms of hyperactivity
May present with symptoms of repeated accidents or injuries

Risk factors for adolescent suicide
Presence of mental disorder
Family history of suicide
Gay or lesbian youth
Very high-achieving adolescents

ADULT RESPIRATORY DISTRESS SYNDROME

Moderate to severe hypoxemia
Diffuse alveolar infiltrates in absence of pulmonary infection
Etiology: diffuse alveolar injury
Diagnosis: pulmonary artery catheter
Treatment: treat underlying cause
ARDS – risk associated with sepsis, mortality = @ 50%

SIRS
Systemic inflammatory response syndrome
Diagnosis: B/P systolic < 60 mmHg
Urine output < 30 ml/h
Treatment: O_2, circulatory volume
Check CBC, lytes, ABG, BUN, creatinine, U/A, PT, PTT, fibrinogen, CXR
Vasopressor treatment
Start antibiotics
Abscess? If detected – drain
SIRS = 25–50% mortality

Physiology of ARDS

Increased airway pressure with 'stiff lungs'
Increased capillary permeability
Ventilation–perfusion mismatching
Decreased lung compliance
Decreased pulmonary capillary wedge pressure (hydrostatic)
Decreased residual capacity
Arterial pO_2 < 50–60 despite O_2 concentration of > 60%

AGCUS

Rate	0.2–0.5%

Atypical glandular cells of undetermined significance

Management

Colposcopy with biopsy and ECC
Conization if suspect preinvasive or invasive adenocarcinoma
In any woman with AGCUS, do a colposcopy, endocervical evaluation, directed biopsy, and pelvic exam
If endometrial cells are suspected or if risk factors are present → do endometrial biopsy, D&C or hysteroscopy (always if > 35 years old)
If no abnormalities are noted on D&C, suspect extrauterine sites such as ovary, fallopian tube, GI tract and breast
Knowledge of glandular disease of the cervix remains far behind its squamous counterpart
Conditions known to mimic ACIS on Pap smears: lower uterine segment sampling, tubal metaplasia, polyps and endometriosis
Conservative management of ACIS (adenocarcinoma *in situ* of the cervix)
The distance from the closest ACIS lesion to the endocervical margin should be > 10 mm
FIGO stage IA1 disease best describes microinvasive adenoma of cervix
There have been no published reports of lymph node metastases in IA1
AGCUS report on a ThinPrep® specimen indicates a significant risk for invasive cancer or other serious pathology
What chance does a woman have of invasive cancer somewhere if she has a report of AGCUS? 10%

- Practitioners generally under manage patients with atypical glandular cells of undetermined significance and over manage patients with atypical squamous cells of undetermined significance.

(Smith-McCune K, Mancuso V, Constant T, *et al*. Management of women with atypical Papinicolaou tests of undetermined significance by board-certified gynecologists: discrepancies with published guidelines. *Am J Obstet Gynecol* 2001;185:551–6)

AGE AND ASSOCIATED INFERTILITY

> 34	11%
> 40	33%
> 45	87%

ALCOHOL

Diet

Distilled whiskey 1–1½ oz	100 calories
Beer (regular) 12 oz	150 calories
Beer (light) 12 oz	100 calories
Wine (dry) 4 oz	90 calories

Screening methods

Cut	***T***olerance
Annoyed	***A***nnoyed
Guilty	***C***ut
Eye opener	***E***ye opener

ALLERGIC REACTION

First-line therapy	Urticaria, bronchospasm	epinephrine 1 : 1000 SC 0.5 cc
	Respiratory distress, systolic > 80	Epi 1 : 1000 SC or IM 0.5 cc
	Laryngeal edema, respiratory failure	Epi 1 : 10000 IV
Second-line therapy		Benadryl® 25–75 mg p.o. q. 6 h x 3–5 days
		prednisone 40–60 mg p.o. q. daily x 3–5 days

ALOPECIA

Types	Kerion – any fungus (esp *M. canis*) severe inflammatory reaction	
	Trichotillomania – act of removing one's own hair by manipulation	
	Alopecia areata – rapid asymptomatic loss of hair	
	Telogen effluvium – associated with weight loss, stress	
	Anovulation with PCO – most common cause hyperandrogenism demonstrated in	40%
Treatment	Approved dosage of finasteride 1 mg/day may not be enough for male androgenetic alopecia. In one Italian study (Iorizzo M, Vincenzi C, Voudouris S, Piraccini BM, Tosti A. Finasteride treatment of female pattern hair loss. *Arch Dermatol* 2006; 142: 298–302) 2.5 mg/day was well tolerated	

ALPHA-FETOPROTEIN

Fetal AFP – produced sequentially by fetal yolk sac, GI tract and liver
Reaches peak concentration at end of first trimester. Abrupt decrease in AFP production at 30 weeks
MSAFP – continues to increase with fetal levels that decrease
Mechanism of transfer – 2/3 transplacental, 1/3 amniotic

5-ALPHA-REDUCTASE DEFICIENCY

This enzyme is needed to convert testosterone to dihydrotestosterone, which is required for the development of penis and scrotum
These children are usually raised as females, some even father children. There are normal male levels of testosterone and estrogen but no breast development

ALTERNATIVE MEDICINES

Approximately half women in the USA and Canada use alternative medicines
Women use this % botanicals for menopausal symptoms 10–15%

St John's wort (Hypericum perforatum)	Depressive disorders (major depressions cannot be treated)
Valerian	Sleep disorders
Ginkgo biloba	Circulatory disorders
Echinacea	URIs
Garlic	Hypercholesterolemia
Classes of phytoestrogens	Isoflavones
	Lignans
	Coumestans
	Soy
	Lentils, legumes, garbonzo beans
	Flaxseed
	Cereals and fruits
	Red clover
	Bean sprouts, sunflower seeds

ALZHEIMER'S

Risks

Age
FMH
Genetics — chromosomes 1, 14, 21
APP — chromosome 19
Head trauma
Female

Protective

Estrogen
Increased educational level
Anti-inflammatory
Antioxidant use

Diagnosis

Mini cognitive tests
'Clock test' – have patient attempt to draw a picture of the face of a clock. The times will usually be very unusual
Others include the 'two word test' where a patient is told two to three words at beginning of brief conversation then asked to recall them
The 'backward count test' is another test in which patients are requested to count backward or backward by sevens, etc.
These patients are very skilled at turning the situation around and not answering some of these tests

Treatment

Cholinergics
Tacrine q.i.d. – increases liver enzymes
Zoloft – increases serotonin
Aricept® (donepezil HCl) 5 mg/day × 1 month then 10 mg/day × 2–3 months orally at night. This inhibits acetylcholinesterase, which is one of the enzymes that cause the breakdown of cholinesterase
Reminyl® 16 and 24 mg daily taken as 8 mg or 12 mg tablets b.i.d. with a full meal. This inhibits acetylcholinesterase and nicotinic receptors
Exelon® 1.5, 3, 4.5 and 6 mg dosages to be taken b.i.d. beginning with smaller dosages and titrating up. This drug inhibits both acetylcholinesterase and butylcholinesterase
Use one of these then reassess changes in behavior, cognition after a few months of therapy
Estrogen should be used to PREVENT rather than treat Alzheimer's

AMBIGUOUS GENITALIA

Fusion of labial folds and absence of palpable testes.
Incidence is — 1/5000–1/15 000
Determinants of sex rearing is fertility potential of phallus/responsiveness — increased in Alaskan Yupik Eskimo
Delay sex assignment until diagnosis ('not developed yet')
Technically – construction of female genitalia is easier.
Reassignment of sex can be made up to 18 months

Diagnosis

H&P
MRI – uterus + cervix? ovaries? undescended testes? urethra, vagina
Labs – karyotype, lytes, 17-OHP, androgens (testosterone, DHEA), 11-deoxycortisol, 11-deoxycorticosterone

Three types

Salt-wasting – 66% with virilizing adrenal hyperplasia
Symptoms: failure to thrive, apathy, vomiting, hyponatremia, hyperkalemia, acidosis
Non-saltwasting (virilizing)
Late-onset – seen after adolescence, menstrual irregularities, infertility
Most common cause is congenital adrenal hyperplasia (female pseudohermaphroditism or congenital virilizing adrenal hyperplasia).
NEED RAPID DIAGNOSIS TO SAVE INFANT — 7 days
If left untreated – progressive virilization, metabolic disorders (salt-wasting, increased B/P, hypoglycemic)
MOST FREQUENT CAUSE OF ENDOCRINE NEONATAL DEATH AND SEX AMBIGUITY

(1) Congenital adrenal hyperplasia
  (a) 21-Hydroxylase deficiency (most common CAH) — 90%
    Increased serum (50–400-fold) — 17-OHP

Located on chromosome 6
Most common autosomal recessive trait
Rx with glucocorticoids
Prenatal Rx: dexamethasone (if karyotypes OK – stop)
Newborn Rx: cortisol 12–18 mg/m^2
Abnormal 17-OHP @ 48 h after birth is 3500–40 000 ng/dl (50–400 x)

(b) 11β-Hydroxylase deficiency 5–8%
Increased serum 17-OHP and increased 11-deoxycorticosterone
Hypertensive in what % of cases? 66%
Form of non-salt-wasting CAH
Located on chromosome 8
Karyotype for A + B are 46XX

(c) 3β-Hydroxysteroid dehydrogenase deficiency Rare
Increased 17-OHP but can be normal
Karyotype is 46XY

(2 Male pseudohermaphroditism – rare enzyme disorders
5α-reductase deficiency 46XY

(3) True hermaphroditism

(4) Gonadal dysgenesis

#3 and 4 have normal androgens and 17-OHP
Laparotomy, gonadal biopsy or gonadectomy needed to confirm
Laparoscopy INADEQUATE because gonads are possibly small and hidden in inguinal canal

Ambiguous genitalia – Summary

Definition

Anatomic modification of the external genitalia making specific determination of gender difficult

Evaluation

The prime diagnosis, until ruled out, is congenital adrenal hyperplasia, because this is the only condition that is life-threatening

Differential diagnosis (four categories)

(1) Female pseudohermaphroditism
(2) Male psuedohermaphroditism
(3) True hermaphroditism
(4) Gonadal dysgenesis

Diagnostic work-up

(1) History and physical
Are gonads palpable? (Most important part of the exam)
Phallus length and diameter?
Position of the urethral meatus?
Degree of labioscrotal fold fusion?
Is there a vagina, vaginal pouch or urogenital sinus?
(2) Pelvic ultrasound or MRI
(3) Blood for karyotype analysis, serum electrolytes androgens (androstenedione, testosterone, DHEA, DHEAS), 17-OHP, 11-deoxycorticosterone and 11-deoxycortisol
(4) In selected cases – laparotomy, gonadal biopsy and/or gonadectomy (laparoscopic evaluation is inadequate)

Laboratory findings

(1) Female pseudohermaphroditism (genetic females with excess androgen) in the absence of maternal androgen excess – three forms of congenital virilizing adrenal hyperplasia:
(a) 21-Hydroxylase deficiency – elevated serum 17-OHP
This is the most common form of congenital adrenal hyperplasia (90%), the most frequent cause of sexual ambiguity and the most frequent endocrine cause of neonatal death
(b) 11β-hydroxylase deficiency – elevated serum 11-deoxycorticosterone and 11-deoxycortisol
(c) 3β-hydroxysteroid dehydrogenase deficiency – elevated 17-hydroxypregnenolone and dehydroepiandrosterone
(2) Male pseudohermaphroditism – the result of rare enzyme disorders
(3) True hermaphrodite or gonadal dysgenesis – normal androgens, normal 17-OHP
Laparotomy, gonadal biopsy and/or gonadectomy is needed to confirm the diagnosis

Treatment

It is better to delay sex assignment, than to reverse it at a later date. Tell the parents that the genitals are unfinished, rather than abnormal
The sex assignment depends on whether the phallus can develop into a functional penis. The construction of female genitalia is technically easier
If reassignment of sex is necessary, it can usually be made safely up to 18 months of age

AMENORRHEA

Mean age of menarche is how old? 12.8 years

Definition

Absence of menstruation for 3 or more months in female with past menses or absence of menarche by age of 16 years in female who has never menstruated
No period by what age in the absence of secondary sex characteristics? 14
No period by what age regardless of presence of secondary sex characteristics? 16
Causes include anatomic, ovarian failure or endocrine imbalance

Causes

(1) Central hypothalamic–pituitary deficiency in gonadotropic production
No breast development due to decreased production of E_2
Normal external and internal genitals. Draw FSH
Ovarian failure if FSH increased and over 40 mIU/ml
Central defect (hypothalamic–pituitary) if decreased FSH
GnRH stimulation – LH (increased) indicates hypothalamic such as isolated gonadotropin deficiency
LH with no change indicates pituitary such as pituitary adenoma

(2) Androgen insensitivity (46XY)
Breasts are present (aromatization of androgens to estrogens)
Absence of Müllerian structures (no uterus, cervix, tube, upper vagina)
Testosterone and LH elevated. Draw serum testosterone
Incomplete androgen insensitivity (46XY) (testicular feminization)
Usually associated with ambiguous genitalia, minimal breast development and minimal pubic hair
– Müllerian agenesis (46XX) (Mayer–Rokitansky–Kuster–Hauser syndrome)
Breasts are present
Absence of Müllerian structures
Mild – incomplete fusion of Müllerian cyst with urogenital sinus vaginal transverse septum
Complete – no uterus, cervix, fallopian tube or upper vagina
Testosterone level normal. Pubic hair usually normal
Check IVP as incidence of coexisting renal anomaly is 50%
Vertebral anomalies, cardiac and congenital anomalies increased
Vertebral anomalies are usually increased @ 12%
M-R-K-H is the second most common cause of amenorrhea

(3) Gonadal dysgenesis (45XO, 46XX or 46XY)
Usually due to random chromosomal disorder
Can be due to deletion of all or part of an X chromosome
Sometimes a genetic defect, rarely 17α-hydroxylate deficiency
DO NOT DEVELOP OVARIES – instead gonadal streaks
Most common cause of primary amenorrhea 50%
– Turner's (45XO). Resulting from abnormal karyotype
No breasts, shield chest, web neck, short stature
Coarctation of aorta or bicuspid aortic valve
Increased risk of renal anomalies especially horseshoe kidneys
– Swyer (46XY). Gonadal failure in early fetal development
Absence of both testosterone and MIF
No breasts (deficiency in estrogen)
External and internal genitalia
Remove any gonad if there is the presence of Y chromosome after the age of 18 due to increased incidence of malignancy by 25%

(4) Differential; Pregnancy, iatrogenic, ovarian failure, autoimmune disease, PCO, hyperprolactinemia, chronic disease, anovulation, menopause, Asherman syndrome, radiation/chemotherapy, anorexia/stress, tumor – pituitary/hypothalamic, developmental/genetic

Pearls

Asherman syndrome may result from pregnancy endometritis
Testicular feminization is characterized by breast development, decreased pubic and axillary hair and blind or absent vagina
What two labs should be obtained in evaluation of hirsutism and virilization? Testosterone
DHEA-S

Kallmann syndrome is associated with primary amenorrhea, anosmia and color blindness
Increased secretion of what can cause increased production of prolactin levels? TRH
Prolactin levels should be drawn in relaxed fasting state
Prolactin levels are increased by sleep and food ingestion
Visual-field and extensive pituitary function testing are indicated whenever a pituitary neoplasm is 10 mm
LH/FSH ratio aid in diagnosis of PCO if ratio > 2.5
McIndoe procedure = vaginoplasty for adolescents who have no vagina
Frank procedure = use of vaginal dilator or increase in size using dilator
Hypoestrogenic females have to ingest increased amounts of calcium to achieve a + calcium balance
Exercise-induced amenorrhea is hypothalamic. FSH level is normal to low
Concern is osteoporosis – give estrogen

Amenorrhea – Summary

Definition (in absence of pregnancy)

(1) No menses by age 14 in absence of secondary sexual characteristics or
(2) No menses by age 16 regardless of presence of secondary sexual characteristics or
(3) Three normal cycle intervals without menses or 6 months of amenorrhea in previously menstruating women

Compartmentalization of evaluation

Compartment I:	Outflow tract/endometrium
Compartment II:	Ovary
Compartment III:	Anterior pituitary
Compartment IV:	CNS (hypothalamus)

Evaluation

History & physical
R/o pregnancy
Therapeutic/laboratory investigation
TSH – r/o hypothyroidism
Prolactin – If greater than 100 mg/ml, MRI
Progestin challenge (progesterone in oil 200 mg IM or medroxyprogesterone acetate 10 mg p.o. q.d. x 5 days)
If + withdrawal bleed, diagnostic of anovulation
If – withdrawal bleed, investigate Compartment I

Compartment I

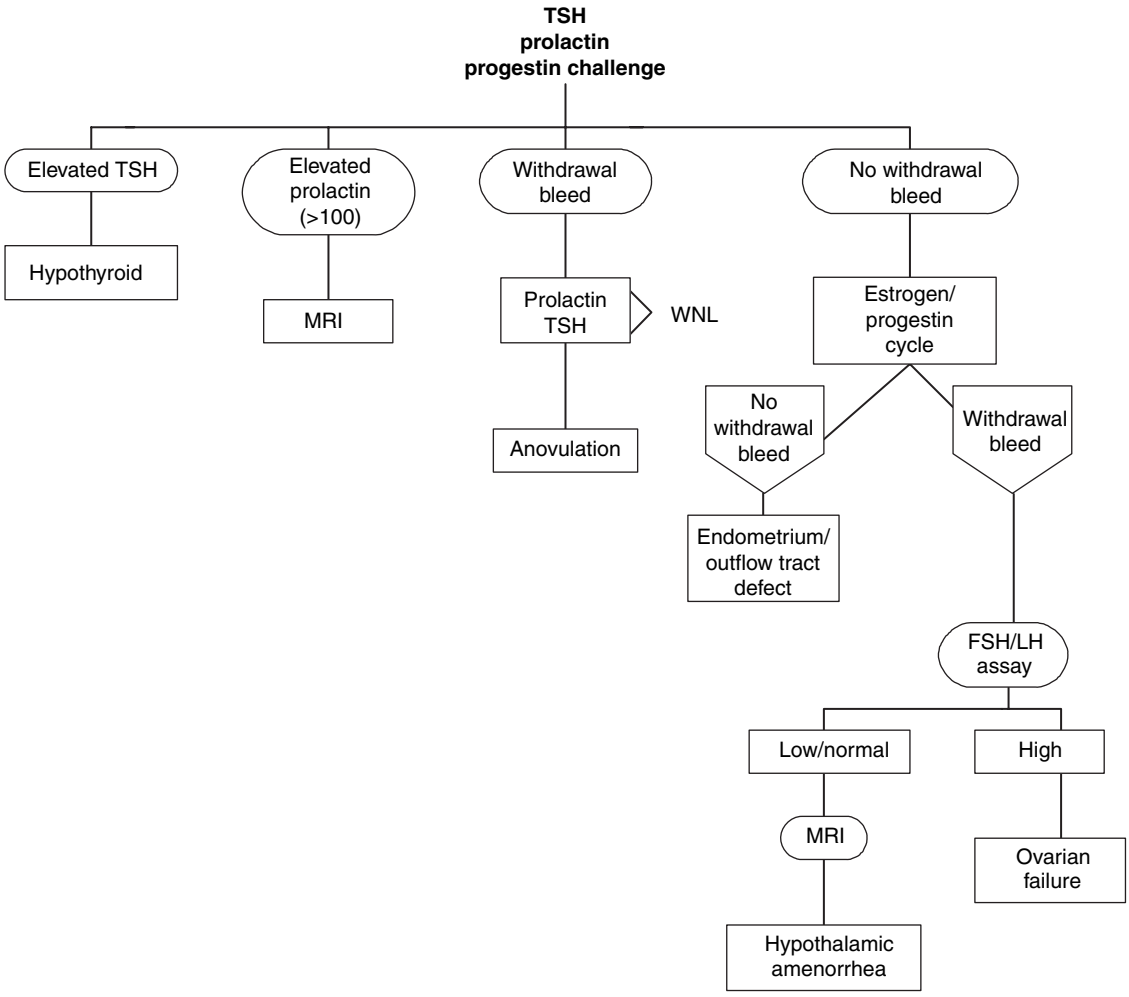
Estrogen/progestin cycle (1.25 mg conjugated estrogen q.d. x 21 days plus medroxyprogesterone acetate 10 mg q.d. for the last 5 days

Negative (–) withdrawal bleed	– defect in endometrium or outflow tract
Positive (+) withdrawal bleed	– investigate compartments II and IV

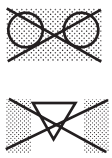
Compartments II, III and IV

FSH/LH assay – At least 2 weeks after estrogen/progestin

Low/normal – MRI	Hypothalamic amenorrhea
High	Ovarian failure


TSH
prolactin
progestin challenge
Elevated TSH
Hypothyroid
Elevated prolactin (>100)
MRI
Withdrawal bleed
Prolactin TSH
WNL
Anovulation
No withdrawal bleed
Estrogen/ progestin cycle
No withdrawal bleed
Withdrawal bleed
Endometrium/ outflow tract defect
FSH/LH assay
Low/normal
High
MRI
Ovarian failure
Hypothalamic amenorrhea

NO MENSES BY AGE 14 YEARS, AND NO SECONDARY SEX CHARACTERISTICS
NO MENSES BY AGE 16 YEARS, WITH SECONDARY SEX CHARACTERISTICS

Patient type	*Presumptions*	*Distinguishing tests*
Breasts absent; uterus present 	Lack of breasts indicates estrogen is not being produced by the gonads because of hypothalamic–pituitary failure, lack of ovarian follicles or lack of two active X chromosomes Presence of uterus indicates Y chromosome is not present	FSH level identifies if estrogen lack is caused by ovarian failure (high FSH)* or hypothalamic–pituitary failure (low FSH) GnRH stimulation identifies whether the hypothalamus or pituitary has failed: • Hypothalamic failure (LH rises) • Pituitary failure (lack of LH response)
Breasts present; uterus absent 	Presence of breasts indicates estrogen was or is being produced by the gonads Absence of uterus indicates either of the following: • Müllerian agenesis is present in an otherwise normal female (Mayer–Rokitansky) • The patient has a Y chromosome (androgen insensitivity)	Testosterone level suggests if the patient is: • 46XX with Müllerian agenesis (female levels) • 46XY with androgen insensitivity (male levels) Karyotyping confirms genetic sex is male with lack of androgen receptors. The gonads should be removed to prevent malignant transformation
Breasts absent; uterus absent 	Lack of breasts indicates estrogen is not being produced by the gonads because of gonadal agenesis, agonadism or rare gonadal enzyme deficiencies Absence of uterus indicates the patient has a Y chromosome with testes that produced MIF at one time Presence of female external genitalia indicates no testes were present to produce testosterone when the external genitalia formed	Karyotyping of 46XY, an elevated gonadotropin level and a testosterone level in the female range confirms gonadal agenesis of agonadism Gonadal biopsy is needed to diagnose rare enzyme deficiencies
Breasts present; uterus present 	Presence of breasts indicates estrogen was or is being produced by the gonads Presence of uterus indicates Y chromosome is not present	These patients should be worked up with β-hCG, TSH level, prolactin level, progesterone challenge test

*High FSH: 99.00%, ovarian failure; 0.99%, 17-hydroxylase deficiency (46XX); 0.01%, oat cell CA of lung

AMNIOCENTESIS

Definition

Amniocentesis is prenatal diagnostic testing of the amniotic fluid

Genetic amniocentesis

Gestation of 16–18 weeks is the optimal time for genetic amniocentesis for the following indications:

(1) Maternal age of 35 or older at EDC
(2) Parental translocation carriers prior infant
(3) Family history of neural tube defect
(4) Paternal age of 55 years or older
(5) Mother known to be carrier of X-linked disorder
(6) History of habitual abortion
(7) Risk for prenatally diagnosable biochemical/genetic disorder
(8) Maternal serum fetoprotein abnormal

Non-genetic amniocentesis

During the second and third half of pregnancy for the following indications:

(1) Fetal lung maturity
(2) Rh iso-immunization
(3) Meconium
(4) Postdatism
(5) Amnionitis – for Gram stain and culture

Procedure

(1) Genetic counseling and informed patient consent must precede amniocentesis
(2) The amniocentesis is to be performed by a physician
(3) The amniocentesis is performed under aseptic technique. Select the site for transabdominal insertion of spinal needle (22 or 20 gauge) by ultrasonographic determination of placenta site, fetal position and the presence of a suitable pool of amniotic fluid. Avoid the placenta and fetus. All amniocentesis procedures are to be performed under sonographic guidance. The tap should be done, if possible, at midline to avoid major vessels
(4) When an inadequate specimen is retrieved or the fluid is very bloody, a second needle insertion may be necessary. More than two needle insertions should be avoided. The first few drops of fluid should be discarded in order to minimize the risk of contamination by maternal cells in the needle pathway
(5) In case of twins, one sac should be tapped, the fluid collected and 0.5 cc violet gentian should be inside the same sac. A second needle and another site should be used for the second tap. Clear fluid should be obtained
(6) Patients are released after a brief period of observation and ultrasound documentation of fetal viability
(7) Instructions should be given to patient about resting for the remainder of the day and notify in case of fever, contractions or bleeding

AMNIOINFUSION

Bolus	800 ml
At rate of	10–15 ml/min
Until non-reassuring FHR abates then +	250 ml
Repeat if fluid loss, positional change, patient has Valsalva or FHR decreases again	
Continuous method: loading dose of	10 ml/min x 1 h
Then maintenance dose per infusion pump at	3 ml/min

AMNIONITIS

Definition

Amnionitis is a clinically defined infectious disease process involving the intrauterine contents during pregnancy. Synonymous terms include chorioamnionitis ('chorio'), intra-amniotic infection and amniotic fluid infection
For the most part, amnionitis is a bacterially mediated event, although other types of pathogens – such as mycoplasmas and viruses–have been implicated as causative agents

Pathogenesis

The most common route for infection involves the passage of micro-organisms from the lower genital tract in an ascending fashion.
In a majority of cases, this follows either spontaneous or artificial rupture of the fetal membranes
A less common route for transmission involves the hematogenous spread of a maternally derived organism via transplacental passage. The exact mechanism by which this occurs has yet to be clearly defined
Organisms such as Group B streptococci and *Escherichia coli* are overly represented in amnionitis cases, in particular those associated with bacteremia. Organisms such as *Gardnerella vaginalis*, *Fusobacterium* and *Bacteroides bivius* are not uncommonly seen in this disease process

Diagnosis

Criteria for the diagnosis of amnionitis include fever, maternal tachycardia, fetal tachycardia, uterine tenderness, foul-smelling amniotic fluid and maternal leukocytosis. From a practical standpoint, fever is the primary clinical feature needed to establish the diagnosis of amnionitis
The only laboratory studies that help support the diagnosis of amnionitis involve sampling of the amniotic fluid. Although culture is the gold standard for confirming the diagnosis, it is not particularly useful in the acute setting. A positive Gram stain (defined as the identification of any bacteria in an uncentrifuged amniotic fluid sample using high-power magnification) correlates relatively well with subsequent culture positivity

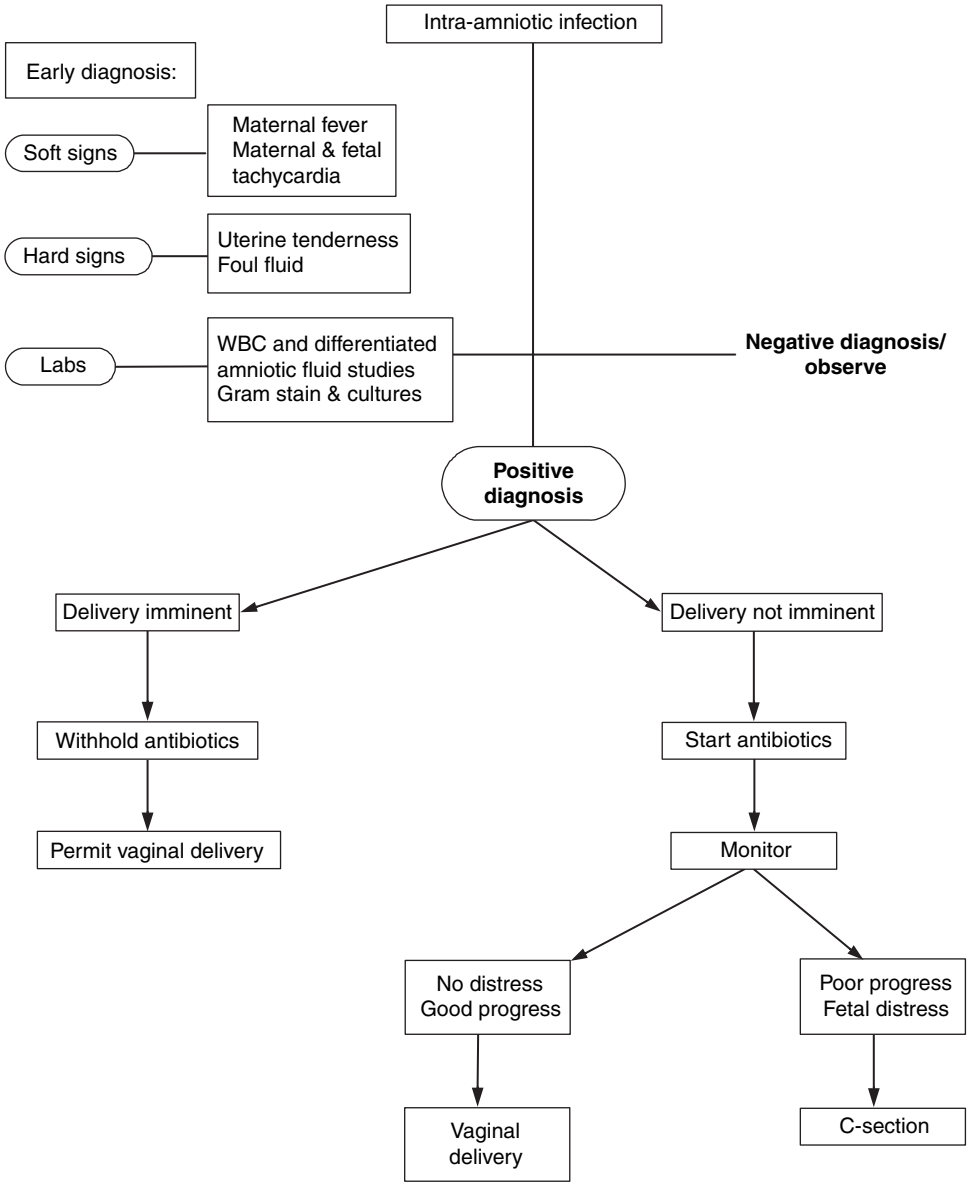
Therapy

When clinical amnionitis is diagnosed, basic goals of therapy are:
(1) To initiate the labor and delivery process regardless of the gestational age
(2) To attempt identification of the pathogens involved in the infectious disease process
(3) To initiate empiric antibiotic therapy
(4) To carefully monitor uterine and fetal heart rate activity

Given the mixed polymicrobial infectious disease process, broad-spectrum parenteral antimicrobial therapy is indicated. This typically includes:
(a) Ampicillin, 1–2 g every 6 h
(b) Gentamicin given in loading and maintenance doses according to the patient's weight and
(c) Clindamycin 900 mg every 8 h (or metronidazole)

In patients with mild to moderate infections use of any of the monotherapies seems appropriate (especially second- and third-generation cephalosporins and penicillins)
Unless delivery is imminent, it is recommended that antibiotic therapy be initiated during the intrapartum interval. Although this may hamper the neonate's evaluation with regard to sepsis, data clearly indicate an improvement in maternal and neonatal outcome when therapy is initiated early
To reduce puerperal morbidity, the vaginal route is clearly preferable for the mother. For the fetus, vaginal delivery is preferred only if it is expeditious and atraumatic
Patients with amnionitis usually have a prompt clinical response to delivery and antibiotics therapy. Assuming the patient has a rapid response to initial therapy, it would seem appropriate to discontinue antibiotics after the patient has been afebrile for 24 h, has return of bowel function and does not demonstrate unusual uterine tenderness. If fever persists after delivery, the patient should be evaluated for other foci of infection or for associated non-infectious complications, such as septic pelvic thrombophlebitis

Diagnosis and management of amnionitis



Pearls

	Incidence at term	1–5%
	Increased infant mortality in term infants	1–4%
	Incidence at preterm	25%
	Increased infant mortality in preterm infants	15%
Etiology	Ascending infection (bacteroides, *E. coli*, anaerobic streptococcus, GBBS)	
	Increased risk – low SE status, young, nulliparous, multiple exams, extended duration of labor and ROM	
	Differential diagnosis – URI, bronchitis, pneumonia, pyelonephritis, appendicitis	
Diagnosis	Maternal fever, maternal + fetal tachycardia, uterine tenderness, purulent amniotic fluid	
	Be cautious about elevated WBC or elevated concentration of C-reactive protein	
	Amniocentesis, Gram stain, BPP	
	Blood cultures are positive	5–10% (one source 28%)
	Amniotic fluid glucose	≤ 10–15%
	Amniotic fluid interleukin	≥ 7.9 ng/ml
	Amniotic fluid leukocyte esterase	≥ 1 + reaction
	Maternal WBC elevated with leukocytes	≥ 15 000
Dysfunctional labor	Patients who require Pitocin	75%
	Patients who require C-section	34–40%
	FHR abnormalities observed (tachycardia + decreased variability)	75%
Treatment	*Benefits of early treatment*	
	Decreased frequency of neonatal bacteremia	
	Decreased duration of maternal fever and hospitalization	
	(1) Ampicillin 2 g q. 6 h with gentamicin 1.5 mg/kg q. 8 h	
	(2) Penicillin 5 million units q. 6 h with gentamicin 1.5 mg/kg q. 8 h	
	(3) If C-section, add clindamycin 900 mg q. 8 h or Flagyl® 500 mg q. 8 h	
	No oral antibiotics needed but continue antibiotic therapy until patient is afebrile without symptoms for how many hours?	24 h
	(May give oral antibiotic therapy for documented staph or following vaginal delivery with rapid defervescence of symptoms)	
	Definitely treat + culture or PPROM	

AMNIOTIC EMBOLISM

	Incidence	1/20 000
	Comprises what % of maternal deaths?	10% or 5th leading cause of death.
	Maternal mortality is	Ranges from 26 to 90%
	Maternasl neurological deficit is	24%
	If intrapartum, fetal mortality rate is	61%
Diagnosis	Acute hypoxia, hypotension or cardiac arrest, coagulopathy	
	Clinical presentation similar to anaphylaxis and septic shock	
Treatment	Expedient diagnosis and treatment. CVP, intubation, treatment of DIC	

Amniotic fluid embolism – Summary

Sudden signs

(1) Agitation
(2) Dyspnea
(3) Anxiety
(4) Respiratory arrest

During labor, delivery or postpartum

Differential diagnosis

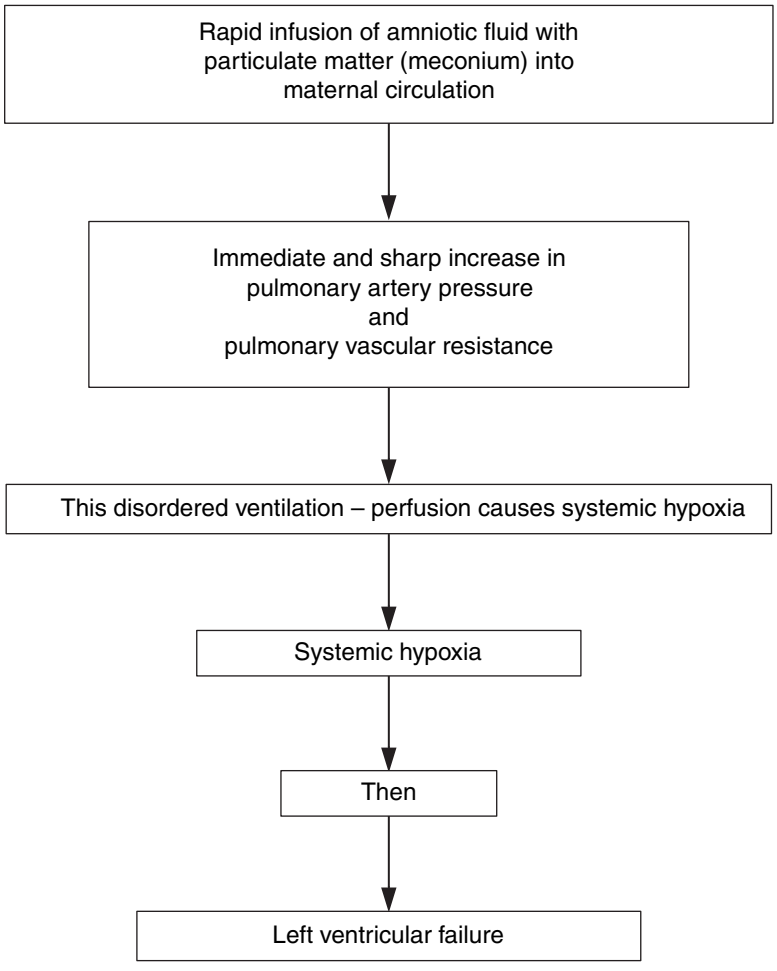
(1) Acute pulmonary edema
(2) Pulmonary emboli from the peripheral venous circulation
(3) Cardiac arrhythmias (MI)
(4) Uterine rupture or anesthesia complications can mimic

During resuscitative efforts – obtain blood from the pulmonary artery via central lines. Look for fetal squames (Attwood stain) and mucin (Giemsa stain). This will confirm the diagnosis in patients who survive

Management

(1) Endotracheal intubation
(2) ABGs (monitor for blood gases to maintain O_2 flow rates)
(3) CPR p.r.n.
(4) Digoxin or dopamine (in second phase of disorder for left ventricular failure)
(5) Swan–Ganz (triple-lumen pulmonary artery) catheter (Obtain special stains during placement)
(6) ICU (if patient survives – meticulous attention to cardiac and renal function and fluid balance)
(7) Pay attention to blood loss (PTT, plts, FDP, fibrinogen)
(8) FFP and/or plts for D/C

Amniotic fluid embolism



Thromboplastin-rich amniotic fluid triggers the intrinsic clotting system with rapid defibrination and hemorrhage (DIC), which aggravates an already complex cardiovascular picture

ANATOMY

Important points of anatomy to remember:

Arterial and venous supply

(1) External iliac artery → inferior epigastric artery
(2) Ovarian veins
Right ovarian vein drains into inferior vena cava
Left ovarian vein drains into left renal vein
(3) Appendiceal artery → ileocolic branch of the superior mesentery artery
(4) Uterine vein → internal iliac veins
(5) Abdominal aorta → ovarian arteries
(6) Inferior epigastric → injury can occur with Maylard incision
(7) Gastroepiploic arteries → ligate during omentectomy
(8) Collateral circulation after hypogastric ligation is via→ lateral and medial circumferential femoral arteries and middle sacral arteries

Branches of hypogastric

Posterior division	*Anterior division*
Iliolumbar	Internal iliac (hypogastric)
Lateral sacral	Obturator
Superior gluteal	Umbilical → superior vesical
	Uterine → vaginal
	Middle rectal → inferior rectal
	Inferior gluteal
	Internal pudendal

Blood supply to vagina

Upper 1/3 → cervicovaginal branch of uterine
Middle 1/3 → inferior vesical arteries
Lower 1/3 → middle rectal and internal pudendal

Blood supply to perineum

Internal pudendal artery → Inferior rectal and posterior labial

Blood supply to uterus

Anterior branch of hypogastric → uterine artery
Uterine vein → internal iliac vein

Lymphatics

Lymphatics of upper vagina drain to iliacs and obturators
Lymphatics of middle vagina drain to internal iliacs
Lymphatics of lower 1/3 of vagina drains to inguinal (femoral) nodes
Lymphatics of the uterus
Lower uterine segment and cervix drain to iliacs and hypogastrics
Upper segment and corpus drain to internal iliacs, hypogastrics, ovarian and periaortics
(Endometrial cancer → mets to inguinal nodes → round ligament)
Lymphatics of the ovaries
Drain to pericaval and periaortics

ANEMIA

Anemia is a common medical problem in women, more frequently than in men, due to blood loss from menstruation and childbirth and as a result of certain problems that occur more often in women, such as collagen vascular disease. Finding the precise etiology is necessary to appropriately manage the anemia

Symptoms

Fatigue, tachycardia, palpitations, and dyspnea on exertion. History taking should include a detailed menstrual calendar that records frequency, duration of flow, and the presence or absence of clots. GI symptoms like heartburn or dark tarry stools should also be noted

Diagnosis

Confirming the diagnosis is essentially the first step. Measure hemoglobin and hematocrit in venous blood with a CBC and classify the anemia according to the diminished production or increased destruction of red blood cells. An alternative approach is to measure MCV and categorize the anemia into microcytic, normocytic, or macrocytic subtypes. Iron deficiency anemia, thalassemias, and anemia of chronic disease are associated with low MCV (<80 fl), while pernicious anemia is associated with a high MCV (>100 fl).

Differential

Iron deficiency anemia – most common anemia found in women.
Serum ferritin is diagnostic if found to be low

Treatment

Oral iron supplements (ferrous gluconate better tolerated than ferrous sulfate; sometimes recommended to take with citrus juice for vitamin C on an empty stomach for better absorption. Although this oral therapy may take 8–9 months to restore iron, IV iron is recommended only for patients refractory to oral iron therapy)

Hypochromic microcytic anemia (MCV < 80 fl)

(1) Iron (Fe+) deficiency anemia (most common)
Decreased iron
Causes:
(a) Dietary deficiency (uncommon in U.S.)
(b) Decreased iron absorption (pernicious anemia, gastric surgery, or removal of terminal ileum)
(c) Pregnancy
(d) Lactation
(e) Blood loss (GI loss, menstruation)
(f) Iron sequestration (pulmonary hemosiderosis)
Symptoms: fatigue
Treatment: elemental Fe+ 200 mg daily

(2) Anemia of chronic disease (especially in elderly women)

(3) Sideroblastic anemia
(a) Congenital
(b) Lead (c) Alcohol (d) Drugs

(4) Copper deficiency

(5) Zinc poisoning (rare)

(6) Thalassemias
α-Thalassemia either asymptomatic or clinically silent. Trait will show mild microcytic anemia. Hgb H disease will show intraerythrocytic inclusions and hydrops fetalis will show Bart's B4Hgb precipitations

Thalassemia major and minor

(a) β-Thalassemia major (Cooley anemia) Increased Hgb A_2, hypochromic microcytosis. Females who survive are usually sterile (80% of untreated children die in first 5 years of life.) Treatments are repeated transfusions, splenectomy, and iron chelation with deferoxamine. Therapies under investigation are bone marrow transplant and gene therapy

(b) β-Thalassemia minor
Hgb A_2 > 3.5%, Hgb F > 2%. Hypochromic microcytosis
Anemia is mild
Treatment: Fe+ 60 mg and folic acid 1 mg daily. Constant monitoring and transfusion as needed

(c) Spherocytosis
Hemolysis and corresponding anemia dependent upon intact spleen

Normocytic anemia (normal MCV, 80 $\rightarrow$100 fl)

(1) Acute blood loss (most common)

(2) Early iron deficiency anemia

(3) Anemia of chronic disease (infection, HIV, inflammation, malignancy)

(4) Bone marrow suppression (bone marrow invasion, acquired pure red cell aplasia, aplastic anemia/myelofibrosis)

(5) Autoimmune hemolytic anemia (erythroblastosis fetalis, transfusion reaction, collagen vascular disease, hemolytic uremic syndrome/thrombocytopenic purpura)

(6) Chronic renal disease (decreased erythropoietin)

(7) Endocrine disorder (hypothyroidism or hypopituitarism)

(8) Spherocytosis (hereditary anomaly of the red cell membrane)

(9) Paroxysmal nocturnal hemoglobinuria

Megaloblastic anemia (macrocytic anemia: MCV > 100 fl)

(1) Folic acid deficiency – pernicious anemia of pregnancy, macrocytosis, increased MCV, hypersegmentation of neutrophils. Incidence is increased with ethanol ingestion (ETHANOL ABUSE)
Symptoms: nausea, vomiting and anorexia
Treatment: (1) Folic acid 1 mg daily
(2) Vitamin B_{12} IM 1000 mg weekly for 4 weeks then monthly for life. Oral supplements of B_{12} can be used after the first month of injections, if absorption is not a problem

(2) Vitamin B_{12} deficiency – rare
Takes years to deplete vitamin B_{12}
Incidence increased with gastric resection, Crohn's disease
Symptoms: neurological (posterior lateral column)
(3) Myelodysplastic syndromes
(4) Acute myeloid leukemia
(5) Reticulocytosis
Hemolytic anemia, response to blood loss, or response to appropriate therapy
(6) Drug-induced anemia
(7) Liver disease/severe hypothyroidism

ANESTHESIA

Predisposition to difficult intubation
(1) Full dentition (protuberant teeth)
(2) Breast enlargement
(3) Abnormal neck (enlarged thyroid, arthritis or neck facial edema)
(4) Receding mandible (or small mandible)
(5) Protruding maxillary incisors
(6) Marked obesity
(7) Asthma (or serious medical or Ob conditions)
(8) History of problems with anesthetics
(9) Thick tongue

Pregnancy
DO NOT
(1) Use depolarizing muscle relaxants prior to administering non-polarizing agents for muscle relaxation
(2) Extubate in upright position
% of patients who report awareness during general ob anesthesia 20%

Anesthesia criteria
Feasible to administer epidural earlier; however, some studies support postponing epidural until cervix is 4–5 cm. One could use some Sublimaze® (fentanyl) or other narcotic until the initiation of active labor. Use terms as 'reasonable', 'apparently slow labor'

ANGIOTENSIN-CONVERTING ENZYME INHIBITORS (ACE INHIBITORS)

Cause
Oligohydramnios
Fetal and neonatal death
Renal failure
Fetal hypocalvaria

ANOREXIA NERVOSA

Refusal to maintain body weight at or above minimal normal weight for age and height < 85%
Intense fear of gaining weight or becoming fat
Denial of body weight or shape for self-evaluation
Amenorrhea (at least three cycles)

Symptoms
Dry skin
Yellow palms
Hypothermia
Bradycardia
Hypotension

Labs
Increased cortisol
Decreased T_3 and T_4

Treatment
Force feed
Psychotherapy
HRT (prevent osteoporosis)

ANOVULATION (CHRONIC) (With no evidence of hyperandrogenism)

Labs	FSH, TSH, DHEA-S, prolactin, endogenous estrogens
	Serum progesterone levels compatible with presumptive ovulation = 3–5 ng/ml

ANTIBIOTICS

	If allergic to PCN or cephalosporins, give: clindamycin, doxycycline or metronidazole
Bacterial endocarditis prophylaxis	Ampicillin 2 g IV or IM plus gentamicin 1.5 mg/kg @ 30 min prior to procedure then amoxicillin 1.5 g p.o. 6 h after initial dose or repeat ampicillin/gentamicin IV/IM dose If allergic to PCN – give vancomycin 1 g IV slowly over 1 h prior to surgery with gentamicin then repeat 8 h later
Categories	Most antibiotics are category B Gentamicin and fluoroquinolones are category C Nitrofurantoin is category B Sulfonamides are category B and D (avoid at term) Tetracyclines and streptomycin are category D
Pearl	Give second dose of antibiotic if (1) Blood loss > 1500 ml (ab concentration is decreased) (2) Procedure > 3 h (renal excretion will decrease effective ab) Scrubs also decrease bacterial concentration for 30–120 min

ANTIPHOSPHOLIPID SYNDROME

False + RPR (STS)	75%
+ test for lupus anticoagulant	90%
+ test for anticardiolipin (IgG and IgM)	90%

One or more of the following:
- Arterial or venous thrombosis
- Connective tissue disease
- Autoimmune thrombocytopenia
- Unexplained pregnancy loss beyond first trimester

SLE is usually not associated with thrombotic events or second-trimester losses but + test for syphilis can be present

CAPS – Catastrophic antiphospholipid syndrome (Asherson's syndrome)
Tissue necrosis of the extremities is a hallmark of CAPS.

Figure 1 Tissue necrosis of the extremities is a hallmark of CAPS (catastrophic antiphospholipid syndrome, Asherson's syndrome)

Labs	Lupus anticoagulant and anticardiolipin antibodies
Indications to test	Unexplained fetal death or stillborn Recurrent pregnancy loss (3 or > sp abs or ? second- or third-trimester fetal death Severe PIH < 34 weeks Severe fetal growth restriction or evidence of uteroplacental insufficiency Medical: non-traumatic thrombosis, stroke or TIA. Autoimmune thrombocytopenia, SLE, hemolytic anemia. False-positive serology for syphilis
Diagnostic	Medium to high + anticardiolipin antibodies of IgG isotype
Treatment	Heparin 15 000–20 000 U unfractionated t.i.d. Low dose ASA, calcium carbonate 1500 mg, vitamin D and exercise. Close OB care

AORTIC STENOSIS

Most commonly a result of rheumatic heart disease
AVOID decrease cardiac output (angina, MI, syncope and SD).
Increased heart rate. Decrease in intravascular volume.
PULMONARY ARTERY CATHETER INDICATED (18 mmHg)

APGAR SCORE

Sign	*0 Points*	*1 Point*	*2 Points*
Heart rate	Absent	< 100	> 100
Respiratory effort	Absent	Slow, irregular	Good, crying
Muscle tone	Limp	Some flexion	Active motion
Reflex irritability	No response	Grimace	Cough or sneeze
Color	Blue–white	Body pink, extremities blue	Completely pink

Acronym: APGAR = activity, pulse, grimace, appearance, respirations

APPENDECTOMY

Diagnosis	Helical CT is an accurate, non-invasive technique for the diagnosis of acute appendicitis in pregnancy. However, the misdiagnosis of appendicitis has not changed following the introduction of CT, ultrasound and laparoscopy. The frequency of appendiceal perforation has also not decreased with the introduction of these technologies (Flum DR, Morris A, Koepsell T, Dellinger EP. Has misdiagnosis of appendicitis decreased over time? A population-based analysis. *J Am Med Assoc* 2001;286:1748–53)
Pregnancy	Rupture of the appendix occurs 2–3 times more often in pregnancy because of delayed diagnosis. Treatment in pregnancy 1st Trimester – laparoscopic appendectomy IV antibiotics if there is any perforation, peritonitis, or abscess formation Tocolysis is unnecessary in uncomplicated appendicitis, but may be indicated if the patient goes into labor after surgery 3rd Trimester – if there is perforation or peritonitis, a C-section is indicated
Incidental/non-emergent indications	(1) Female 10–30 years of age (2) Female with exploratory surgery for unexplained pain (3) Female with exploratory surgery for RLQ pain (4) Female who is mentally handicapped
Contraindications	(1) Female with Crohn's disease (2) Female with inaccessible appendix (3) Presence of grafts or material (4) Prior history of radiation (5) Unstable medical condition

ARREST OF DILATATION

Criteria for arrest disorder
(1) Latent phase complete 4 cm
(2) Uterine contractions without contractions at 200 MV units for 2 h

Evaluate 3 Ps
(1) **P**owers – 3–5 contractions in a 10-min window
(2) **P**assenger – fetal weight, position and attitude
(3) **P**assage – bony pelvis (OP with narrow pelvis best delivered without rotation)

See also Protraction disorder

ARTHRITIS

Chronic arthritis

Rheumatoid
Insidious onset over weeks to months. Small joints of hand (usually NOT distal). Wrists, elbows, shoulders. MORNING STIFFNESS + rheumatoid factor

Osteoarthritis
Common in females over what age? 55 years
Distal interphalangeal joints of hands. Weight-bearing joints (HIPS + KNEES).
NOT associated with morning stiffness. No lab abnormalities with joint fluid non-inflammatory

Acute polyarticular

Systemic lupus erythematosus
Butterfly rash, photosensitivity, fever, fatigue and arthritis

Acute monoarticular

Disseminated gonococcal infection
Young sexually active female. Most common cause of septic arthritis in the USA. Associated with pustular or papular dermatitis. KNEE, elbow, ankle, small joints

Gout
Precipitates attack: trauma, surgery, EtOH abuse, medical illness
What lab level is elevated in prevalence? Serum uric acid
What can be found in the synovial fluid? Urate crystals

ARTIFICIAL INSEMINATION CUMULATIVE PREGNANCY RATE

After six cycles AID 40–50%

ASCUS

What % of Paps? 5%
HGSIL or invasion 15–20%

What causes ASCUS?

Changes usually due to HPV-koilocytosis (perinuclear halo seen)
Which viral protein of HPV disables p53? E6 + E7

Diagnosis

Colpo with ECC and biopsy

When should LEEP or cone be carried out?

(1) Biopsy does not explain abnormal cells?
(2) ECC has CIN
(3) Microinvasion is seen on biopsy
(4) Abnormal cytology with no visible colposcopic lesion
(5) Atypical epithelial extension to endocervical canal

LGSIL and HGSIL = not reason to cone. Histologic reasons needed to cone
Remember adenoca = ECC if bleeding or unexplained
ASCUS with repeat Pap q. 3–6 months. Repair process due to trauma or to infection is a usual cause
ASCUS can be associated with atrophy in elderly patient – treat with estrogen therapy then repeat Pap

Staging of cervical cancer

Examination and palpation (cervix, vagina, parametrium, side walls)
Examination of supraclavicular nodes and upper abdomen
CXR, IVP, cysto, flexible sigmoidoscopy or BE. NO CT, MRI or lymphangiogram

ASHERMAN'S SYNDROME

Associated with	Curettage after term pregnancy Therapeutic and spontaneous abortion Myomectomy Habitual abortion Hypomenorrhea
Diagnosis	Filling defects on HSG

ASHERSON'S SYNDROME

Associated with	A rapidly progressive variant of the antiphospholipid syndrome CAPS → catastrophic antiphospholipid syndrome 60% of the time, something triggers it Most common trigger is infection (22%). Trauma is 14%
Clinical manifestations	Vary widely

ASSAULT

Assault occurs in this	% of the victim's home	50%
	% of victim's home more than once	50%
	% of ALL females who have had an attempted or actual assault	44%
	% of assaults that are DATE RAPE	25–85%
	Estimated % of rapes that are unreported in the USA	90%
	Percent of all US couples who will experience one incident of violence	40%
	What % of sexual assault victims report to ER within 72 h?	15–20%
	If > 72 h, forensic evidence not collected but remainder of exam is the same	
	This % of victims will have somatic complaints	80%
	History (mens, Ob, contraception, date of last sex) ***I***nformed consent (seek support person) ***D***ocument (timing, nature, weapons, substances, information) ***F***orensic (evidence of clothes, blood type for DNA, saliva, hair, fingernails) ***E***mergency contraception ***A***ntibiotics ***L***ab (GC, *Chlamydia*, Trich)	
Work-up of sexual assault victim	(1) Culture for GC and chlamydia. Culture and wet mount for BV, Trich and candidiasis. Serum for serology analysis if test positive (2) *Pregnancy prevention* Ovral® two tabs 12 h apart (3) *Prophylaxis* Hepatitis B vaccine, ceftriaxone 125 mg IM, doxycycline 100 mg b.i.d. x 7 days, metronidazole 2 g p.o. (no consensus @ HIV prophylaxis) (4) *Follow-up* 2 weeks – cultures for GC and chlamydia – not needed if treated 12 weeks – Serology *T. pallidum*, exam for infection Hep B virus – not needed if vaccine given 6 months – HIV (repeat test at 6 months)	

ASTHMA

Tidal volume = air going in and out. This increases	40%
Minute ventilation = how much air is going in and out in 1 min (RR x TV) increases	50%
There is a decrease in pCO_2 in pregnancy. Severe if CO_2 is	36 mmHg
In pregnancy, there is a decrease in pCO_2 to average of	30 mmHg
There is an average increase in pO_2 to average of	106 mmHg
Forced expiratory volume (FEV_1) and peak expiratory flow rate (PEFR) are both	unchanged

Management

Determine first whether the asthma is mild, moderate or severe and also try to eliminate the exacerbating factor. For instance, in pregnancy, often times the patient has esophageal reflux that can be the precipitating factor. If so, Zantac® 150 mg p.o. b.i.d. can be given

If there is severe distress or there is poor response to outpatient treatment: Do H&P, PEF or FEV_1, oxygen saturation, and fetal assessment with continuous electronic fetal monitoring and/or biophysical profile

Treatment

The author's choices for outpatient management of asthma during pregnancy are

(1) *Leukotriene* modifiers montelukast (Singulair®)10 mg p.o. q. day or zafirlukast (Accolate) and
(2) Budesonide (category B Rhinocort or Pulmicort®) two puffs b.i.d. (dry cortisol powder inhaler) or
(3) Short-acting β_2 agonist metered-dose inhaler; 2–4 puffs every 20 minutes, up to 3 times.
(4) Oral corticosteroid: 40–60 mg/day for 3–10 days.
(5) Ipratropium metered-dose inhaler; 4–8 puffs as needed.

More detailed treatment

Mild – terbutaline (category B), albuterol (category C)
Moderate – beclomethasone (category C)
cromolyn (category B) useful in exercise-induced asthma
Severe – theophylline (category C) decreased clearance so decrease dose. Aim for serum levels of 8–12 µg/ml

Nebulized albuterol: 2.5–5.0 mg every 20 minutes for 3 doses, then 2.5–10 mg every 1–4 hours as needed
Nebulized ipratropium: 0.5 mg every 30 minutes for 3 doses, then every 2–4 hours as needed
Oxygen
Systemic corticosteroid: 120–180 mg/day in 3–4 divided doses for 48 hours, then 60–80 mg/day until PEF = 70%
Consider intravenous aminophylline: 6 mg/kg loading dose, 0.5 mg/kg per hour initial maintenance; keep theophylline level between 8 and 12 ug/ml
Consider 0.25 mg subcutaneous terbutaline or magnesium sulfate if no response to therapy
See also Respiratory disorders

ATELECTASIS

Most common cause of postop fever
Fever, tachypnea and tachycardia develop within first 72 h after surgery
Exam demonstrates:
(1) Decreased breath sounds
(2) Moist inspiratory rales
(3) Increased productive cough
(4) Increased WBCs
(5) Patchy infiltrate on CXR
Usually resolves by 3rd–5th postop day

AUGMENTATION OF LABOR

Pitocin 1 mU/min IV then increase by 1 mU/min IV q. 30 min

PGE_2

Dinoprostone (FDA approved)
Prepidil® gel q. 8 h
Cervidil® tampon @12–15 h prior to induction

PGE_1

Misoprostol (not FDA approved but used for > 10 years)
Cytotec® 25 µg q. 3 h
50 µg q. 6 h
Prostin® suppositories 2.5 µg

Dilatories
Laminaria

Amniotomy

Pitocin

Contractions 25–75 mmHg amplitude
95–395 MV units
Hyperstim > five contractions in 10 min or contractions of normal duration within 1 min of each
Treat hyperstim with terbutaline 0.25 mg IV or $MgSO_4$ 4 g in 10–20% dilution

AZT

Start in an HIV-infected patient if CD4 count is	< 500/mm
Prophylaxis for *Pneumocystis carinii* if CD4 count	< 200/mm
This CD4 lymphocyte count would necessitate AZT therapy in pregnancy	100/mm
Risk of perinatal transmission of HIV infection is approximately	25–40%
AZT therapy decreases risk of prenatal transmission by	66%

Prenatal care, AZT therapy, AND C-section reduce transmission rate of AIDS the most

BACK DOWN LIE

Do low vertical incision

BACK LABOR

See Sterile water papules

BACK UP LIE

May do low transverse incision

BACTERIAL VAGINITIS

See also section on Vulvovaginosis.
Associated with PTD (preterm delivery)
Screen for BV in patients at *high* risk for PTL

Treatment of choice is ORAL metronidazole (better than vaginal)
Other treatments include Metrogel vaginal gel at night for 5 nights, Relagard at night for 5 nights, or clindamycin gel (Clindesse) once

Treatment not to begin prior to first trimester
Rescreening or re-treatment of persistent BV not clear. Routine screening NOT endorsed

Twice weekly metronidazole can keep recurrent BV in check but one must be vigilant for candidiasis and consider suppressive therapy for it as well as BV

BV recurs in up to 30% of women within 3 months, and greatly disrupts well-being. High-dose treatment (and possibly condoms) improve cure rate

BARTHOLIN'S GLAND

Obstruction of Bartholin's duct with pain, tenderness and increase in size
Abscess – GC, *E. coli*, *Proteus*, vaginal flora usually anaerobes

Treatment

Asymptomatic	None
Symptomatic	I&D
	Word catheter
	Marsupialization
	Excision

- Postmenopausal patient may present with malignancy so definitely excise

Squamous cell, transitional cell and ADENOID CYSTIC carcinoma – wide local excision

Local recurrences common
Deeply invasive but no nodes are involved. Risk of excision in increased bleeding, scar formation, cellulitis, etc.

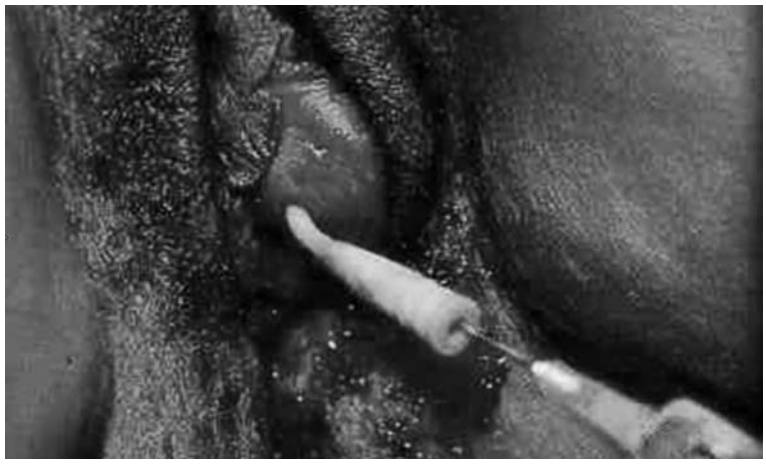


Figure 2 Treatment of Bartholin's gland cyst with Word catheter

BASAL CELL CARCINOMA OF VULVA

	What % of vulvar cancers? 2% Usually of labia majora. 'Rodent ulcers' – central ulceration. Peripheral rolled edges
Diagnosis	Histology shows peripheral palisading of tumor cells
Treatment	Local excision with clear margins. If recurrence = wide local excision

BEHÇET'S DISEASE

Autoimmune process

(1) Ulcerative on anogenital area
(2) Ulcer of buccal membrane
(3) Eye involvement with neuro consequence

BELL'S PALSY

Occur in what % of pregnancy?	3 x more common in pregnancy
Third trimester	75%
First and second trimester	15%
Postpartum	10%

Isolated 7th facial cranial nerve palsy

Symptoms	(1) Acute onset of pain in ear (2) Right- or left-sided facial tightness and pain (3) Inability to close eye (4) Metallic taste in mouth
Etiology	Exposure to cold, hypercoagulability of pregnancy, hormone changes, fluid retention (mechanical compression or blood supply to nerve is compromised)
Treatment	Supportive
Prognosis	Good, usually spontaneously resolves, rapid

BIOPHYSICAL PROFILE

***G**ross body movements*	At least three discrete moves in 30 min
***R**ate (NST)*	At least two accelerations > 15 BPM of 15 s duration in 30 min
***A**mniotic fluid*	At least one pocket measuring 2 cm in two perpendicular planes
***B**reathing movements*	At least one episode > 30 s in 30 min
***T**one*	At least one episode of active extension in 30-min period
	Intervene if 6 or <

BISHOP'S SCORE

System to evaluate cervical induction

Features	*0 Points*	*1 Point*	*2 Points*	*3 Points*
Dilatation (cm)	0	1–2	3–4	5–6
Effacement (%)	0–30	40–50	60–70	80
Station	–3	–2	–1, 0	+1, +2
Consistency	Firm	Medium	Soft	—
Position	Posterior	Mid	Anterior	—

Inducible if score is > 5

BLEEDING

Amenorrhea — Absent menstrual flow > 90 days

Menorrhagia — Excessive bleeding at the time of menses < 60 ml in 29% is normal
However, 49% of women who complain of heavy periods have flow <80 ml and 27.7% who report normal flow lose > 80 ml of blood

Metrorrhagia — Bleeding occurring irregularly between menses

Menometrorrhagia — Prolongation of the menstrual flow associated with irregular intermenstrual bleeding

Postmenopausal — Bleeding that occurs when after the onset of menopause? 1 year after

Etiologies of bleeding — DUB, pregnancy complications, organic pelvic lesions and extragenital problems (coagulopathies, endocrinopathies, iatrogenic)

Prepubertal bleeding
Bleeding prior to what age is abnormal? 9 years
Cause = infection, foreign body, trauma, prolapse of urethra, neoplasm
DES, OCPs, family history of dyscrasia? precocity?
Ages 20–40, DUB (anovulatory bleeding) is responsible for < 20%
Age > 40, DUB becomes common

Perimenarchal bleeding (adolescence)
What % of excessive abnormal uterine bleeding is due to coagulation defects? 20%
If hemoglobin is < 10 g/dl, risk of coagulopathy is 45%
Most common coagulopathy in adolescence is von Willebrand's + ITP
Less frequent coagulopathies are leukemia, sepsis, + hypersplenism
Suspect coagulopathy if onset of heavy bleeding begins at menarche

Diagnosis
(1) Use narrow blade speculum, one-finger digital exam or rectoabdominal exam p.r.n. and/or do ultrasound if suspect mass. Do Pap and cervical cultures if suspect sexual activity
(2) Labs
CBC, PT, PTT, bleeding time, platelet count
Pregnancy test, TSH, prolactin level

Treatment
OCPs – Ethinylestradiol 30 µg and desogestrel 15 mg or
Ovral (OCPs reduce mean menstrual blood loss from 60.2 ml to 36.5 ml after 3 months)
Provera® 10 mg/day x 10 days or on cycle days 16–25, or for profuse bleed give Premarin® IV 25 mg q. 4 h up to three doses to stop active bleed. If hormone therapy fails to slow bleeding, do hysteroscopy to rule out polyps, submucous myomas or an A–V malformation
Norethindrone 5 mg 3 times daily on cycle days 5–26
Femhrt (norethindrone acetate 1 mg, with ethinylestradiol 5 µg) – give twice-daily or in more extreme cases, 3 times daily
NSAIDs – Naproxen 500 mg every 12 hours starting at onset of period. Relieves dysmenorrhea too
IUD (progestin-releasing) – menstrual blood loss decreased 74–97% 6–12 months after insertion of the progestin-releasing intrauterine system

Reproductive age bleeding

OVULATORY BLEEDING

(1) Midcycle bleeding: Premarin 1.25–2.5 mg 3 days prior to 2 days after ovulation
(2) Premenstrual bleeding: Dxn with endometrial biopsy
If trying to conceive — Clomid
If not trying to conceive — OCPs

ANOVULATORY BLEEDING
Dxn with endometrial biopsy and ultrasound
Treatment is same as #2 for ovulatory bleeding

Postmenopausal bleeding

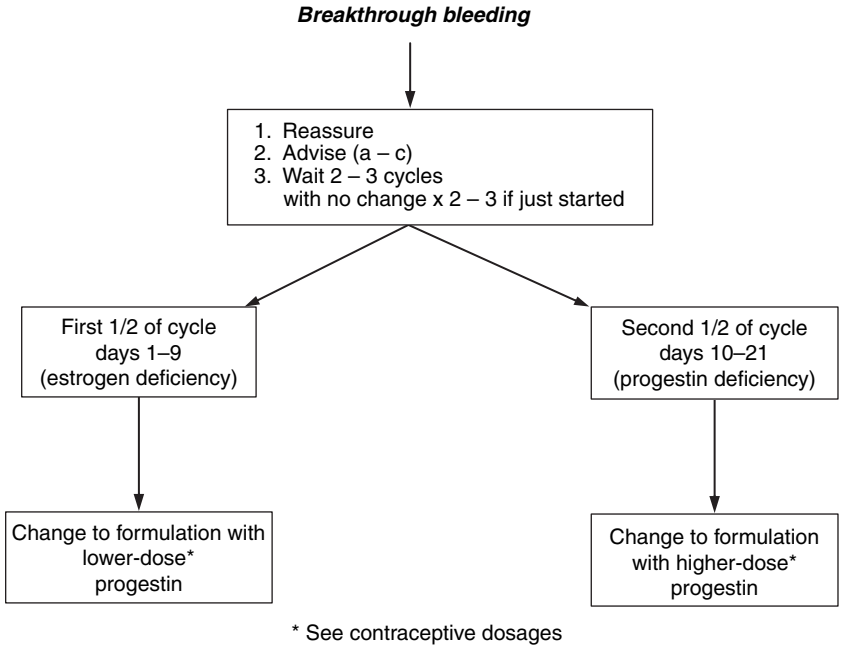
See Postmenopausal bleeding

Postcoital bleeding

Occurs in up to 45% patients using OCPs (with increased vascularity and fragility of cervix, hyperplasia of glands and trauma)
Cervical infection BV 10%, Trich 3% and yeast 2%
Uncommon causes: *T. pallidum*, *H. ducreyi*, *M. tuberculosis*
Increased frequency observed in midcycle and late in secretory phase of an ovulatory cycle

Breakthrough bleeding

Complications for women on OCPs
(a) More likely to occur in smokers than non-smokers
(b) Taking OCPs at same time each day minimizes BTB
(c) Pelvic infections may also cause BTB. Evaluate p.r.n.



* See contraceptive dosages

BLIGHTED OVUM

	Ultrasound findings of a sac without fetal cardiac activity	> 1.2 cm
	Dropping hCG levels	
	Spontaneous abortions that are clinically recognized	10–15%
	Pregnancies lost in first or early second trimester	15–20%
	Pregnancies lost prior to missed menses	50–75%
Medical abortion	Misoprostol 800 µg or if uncertain about location of pregnancy give methotrexate 1 mg/kg IM then give misoprostol	5 days later
Surgical	Paracervical at	7, 9, 11, 1, 3, 5
	Labs	Rh, Hct, preg test STDs? Paps?
	Use cannula of correct size, if 8 weeks =	# 8
	Straight cannula	less pain
	Curved cannula – use for ante- or retroflexed uterus	

Difficulty with stenotic cervix? Try Laminaria, Cytotec, rotation of tip of dilator

Postop: give doxycycline, Methergine®, NSAIDs
If Rh negative and < 12 weeks give MICRhoGAM 50 µg
> 12 weeks give full dose 300 µg

BLOOD PRODUCTS

What % of blood products are administered to patients at or near the time of surgery? 60%
In Ob/Gyn, the transfusion rate has been reported between 0.16% and 8.6% with higher rate for TAH

Whole blood

Advantageous during massive hemorrhage as it is cost effective + decreases infection risk. Disadvantage is that there is a decrease in number of platelets within hours of preparation followed by a rapid depletion of factors V and VIII within 1–14 days

PRBCs

Choice for hemorrhagic shock. O_2 carrying capacity usually met with 7 g/dl Hgb or 21% Hct
DO NOT GIVE if Hgb > 8 g/dl. 21–35 day shelf life. 4 to 1 ratio when blood loss is > 25% of blood volume
For each unit of PRBCs transfused, the Hgb increases 1 g/dl
The Hct increases by 2–3%
Consider giving if < 10 g/dl in patient receiving radiation as response is better due to oxygen to tissue, resulting in free radical formation
Clinical criteria to decide to transfuse should be:

Tachycardia and dizziness
Duration and cause of anemia
Intravascular volume
Extent of the operation or trauma
Probability of additional blood loss
Presence of coexisting conditions such as coronary artery disease, pulmonary insufficiency, cerebrovascular disease and peripheral vascular disease

FFP

Plasma, factors 2, 5, 7, 8, 9, 12, 13 and 500 mg of fibrinogen in 200–250 ml bags

Give in DIC. 4 to 1 ratio when blood loss is > 25% blood volume
For every unit of FFP given, the clotting factor levels rise by 3%

Cryo

80 U/ml of 8, 13, von Willebrand factor, 200–300 mg fibrinogen, fibronectin

Give if: hypofibrinogenemia (usually DIC)
Von Willebrand's disease (prefer factor VIII)
or hemophilia A (prefer VIII:C)

Platelets

Usually needed when massive transfusions (> 10 U PRBCs in 24 h) are given or if pre-op platelet count < 50 000 (10–20 000 count usually @ with spontaneous bleeding). Give if < 20 000. 1 unit of platelets increases platelet count 5000–10 000

Give RhoGAM (300 µg) for every 3 units of platelets transfused (If Rh-negative woman given Rh-positive platelets)

Crystalloids (RL or NS)

STAT resuscitation 3 to 1 rule (300 ml crystalloids per 100 ml blood/plasma volume lost)
Stored blood has decreased pH but acidosis most likely associated with persistent shock
Transfusion of large amounts of blood – alkalosis due to metabolism of citrate to bicarbonate
After transfusion of 10 or > units of PRBCs, crossmatching no longer accurate

Large volume transfusion results in hypokalemia
Check Ca^{+} levels frequently with transfusion of stored blood due to citrate in preservative (binds to Ca^{+})
Do coagulation studies after 5–10 units of transfused blood
Coagulation studies include PT, PTT, platelet count and fibrinogen level
Give platelets and/or coagulation factors ONLY if evidence of deficiency
No correlation with volume of blood given and abnormal coagulation
Increased PT and PTT associated with ongoing hemorrhage – treat with 2 units FFP

Summary chart of blood components

Component	*Major indications*	*Action*	*Not indicated for*	*Special precautions*	*Hazards**	*Rate of infusion*
Whole blood	Symptomatic anemia with large volume deficit	Restoration of oxygen-carrying capacity, restoration of blood volume	Condition responsive to specific component	Must be ABO-identical Labile coagulation factors deteriorate within 24 h after collection	Infectious diseases, septic/toxic, allergic, febrile reactions, circulatory overload, GVHD	For massive loss, as fast as patient can tolerate
Red blood cells; red blood cells; (adenine–saline added)	Symptomatic anemia	Restoration of oxygen-carrying capacity	Pharmacologically treatable anemia Coagulation deficiency	Must be ABO-compatible	Infectious diseases; septic/toxic, allergic, febrile reactions; GVHD	As fast as patient can tolerate but less than 4 h
Red blood cells, leukocytes reduced	Symptomatic anemia, febrile reactions from leukocyte antibodies	Restoration of oxygen-carrying capacity	Pharmacologically treatable anemia Coagulation deficiency	Must be ABO-compatible	Infectious diseases, septic/toxic, allergic reactions (unless plasma also removed, e.g. by washing); GVHD	As fast as patient can tolerate but less than 4 h
Fresh frozen plasma	Deficit of labile and stable plasma coagulation factors and TTP	Source of labile and non-labile plasma factors	Condition responsive to volume replacement	Should be ABO-compatible	Infectious diseases, allergic reactions; circulatory overload	Less than 4 h
Liquid plasma; plasma and thawed plasma	Deficit of stable coagulation factors	Source of non-labile factors	Deficit of labile coagulation factors or volume replacement	Should be ABO-compatible	Infectious diseases allergic reactions	Less than 4 h
Cryoprecipitated AHF	Hemophilia A‡ von Willebrand's disease‡ Hypofibrinogenemia Factor XIII deficiency	Provides factor VIII, fibrinogen, vWF, factor XIII	Deficit of any plasma protein other than those enriched in cryoprecipitated AHF	Frequent repeat doses may be necessary	Infectious diseases; allergic reactions	Less than 4 h
Platelets; platelets, pheresis†	Bleeding from thrombocytopenia or platelet function abnormality	Improves hemostasis	Plasma coagulation deficits and some conditions with rapid platelet destruction (e.g. ITP)	Should not use some microaggregate filters (check manufacturer's instructions)	Infectious diseases; septic/toxic, allergic, febrile reactions; GVHD	Less than 4 h
Granulocytes, pheresis	Neutropenia with infection	Provides granulocytes	Infection responsive to antibiotics	Must be ABO-compatible, do not use depth-type microaggregate filters	Infectious diseases; allergic reactions; febrile reactions; GVHD	One unit over 2-4-h period – closely observe for reactions

*For all cellular components there is a risk the recipient may become alloimmunized

†Red blood cells and platelets may be processed in a manner that yields leukocyte-reduced components for which the main indications are prevention of febrile, non-hemolytic transfusion and prevention of leukocyte alloimmunization Risks are the same as for standard components except for reduced risk of febrile reactions

‡When virus-inactivated concentrates are not available

BLOOD TRANSFUSION RISKS

Approximately how many patients in the USA receive the wrong unit of blood each year?		1000
Immunologic risks (mild fever, chills, urticaria)		1/50–100
Hemolytic transfusion reactions		1/6000
Fatal hemolytic transfusion reactions		1/100 000
Hepatitis C	80–90%	1/3300
Hepatitis B	10%	1/50 000–1/200 000
CMV	3–5%	
Epstein–Barr virus	1–3%	
HIV	< 1%	1/150 000–1/1 000 000 (1/676 000)
Risk of a woman becoming infected with HIV after transfusion with a unit of allogenic blood		1/680 000
Risk of HIV infection after percutaneous exposure to HIV infected blood		0.3%
Risk of developing AIDS after a needle-stick exposure from a known sero + patient is		1/250
Febrile non-hemolytic reactions (temp increase of 1°C during or after transfusion)		1/200
Urticarial reaction (wheezing, urticaria and pruritis with IgE and IgG abs reacting with donor antigen)		1/300

This reaction can be avoided with pre- and postmed antihistamines
This is only type transfusion reaction that can be resumed

BODY MASS INDEX

Calculated by dividing subject's wt (kg) by ht (m^2)

Overweight if BMI	> 26 kg/m^2
Obese if BMI	> 29 kg/m^2

Obese patients may spontaneously ovulate if they lose as little as 10% of their body weight

BOWEL PREP

(1) Chilled Golytely p.o. (polyethylene glycol electrolyte solution) given the day prior to surgery at rate of 1 liter/h (no more than 4 liters of solution or > than 4 h) until rectal effluent is clear
(2) Pre-op antibiotic: cefotetan 1 g, cefoxitin 2 g or ceftizoxime 1 g versus ampicillin/sulbactam 3 g IV (Unasyn®)
(3) May choose to use magnesium citrate in place of Golytely

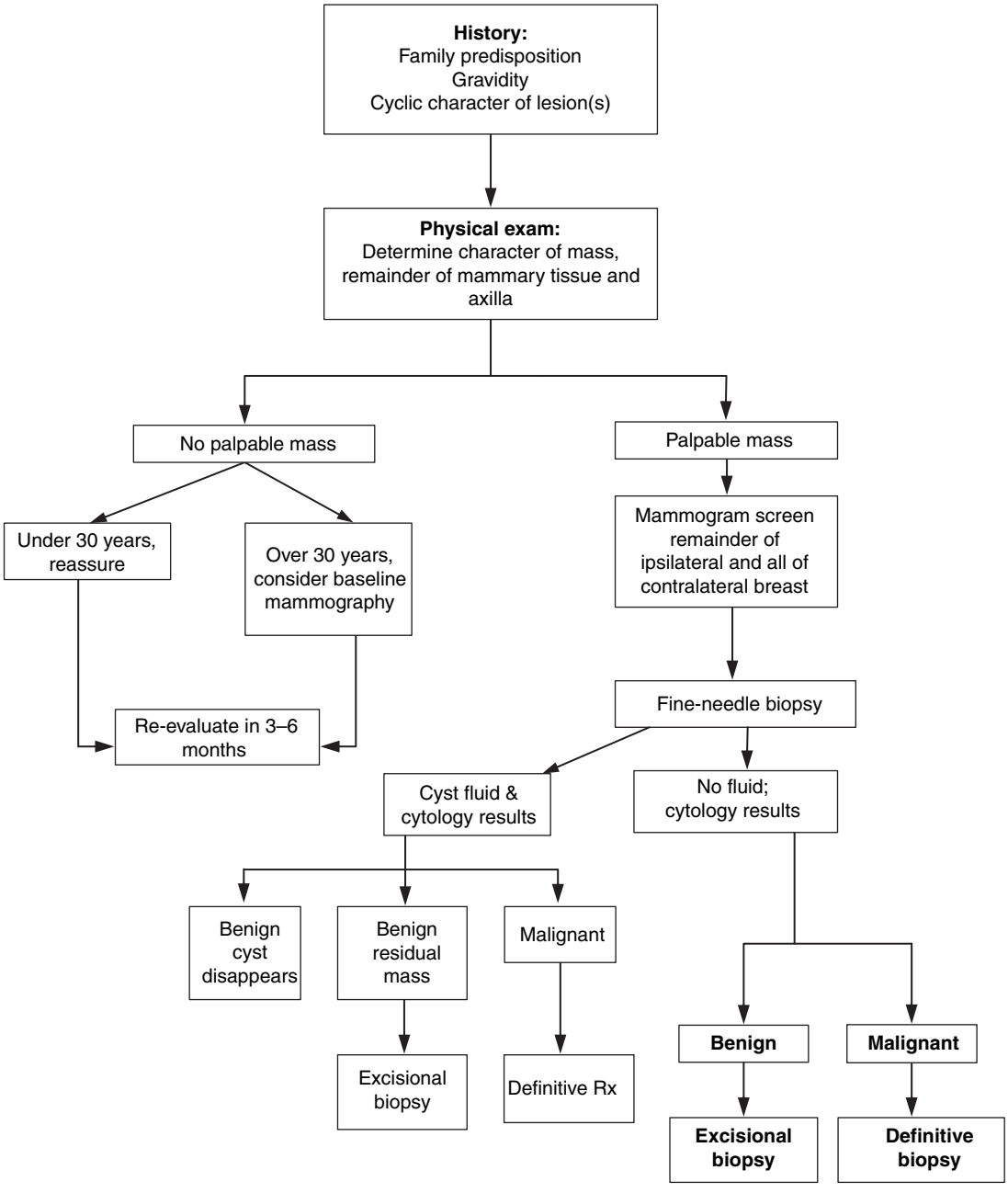
BREAST

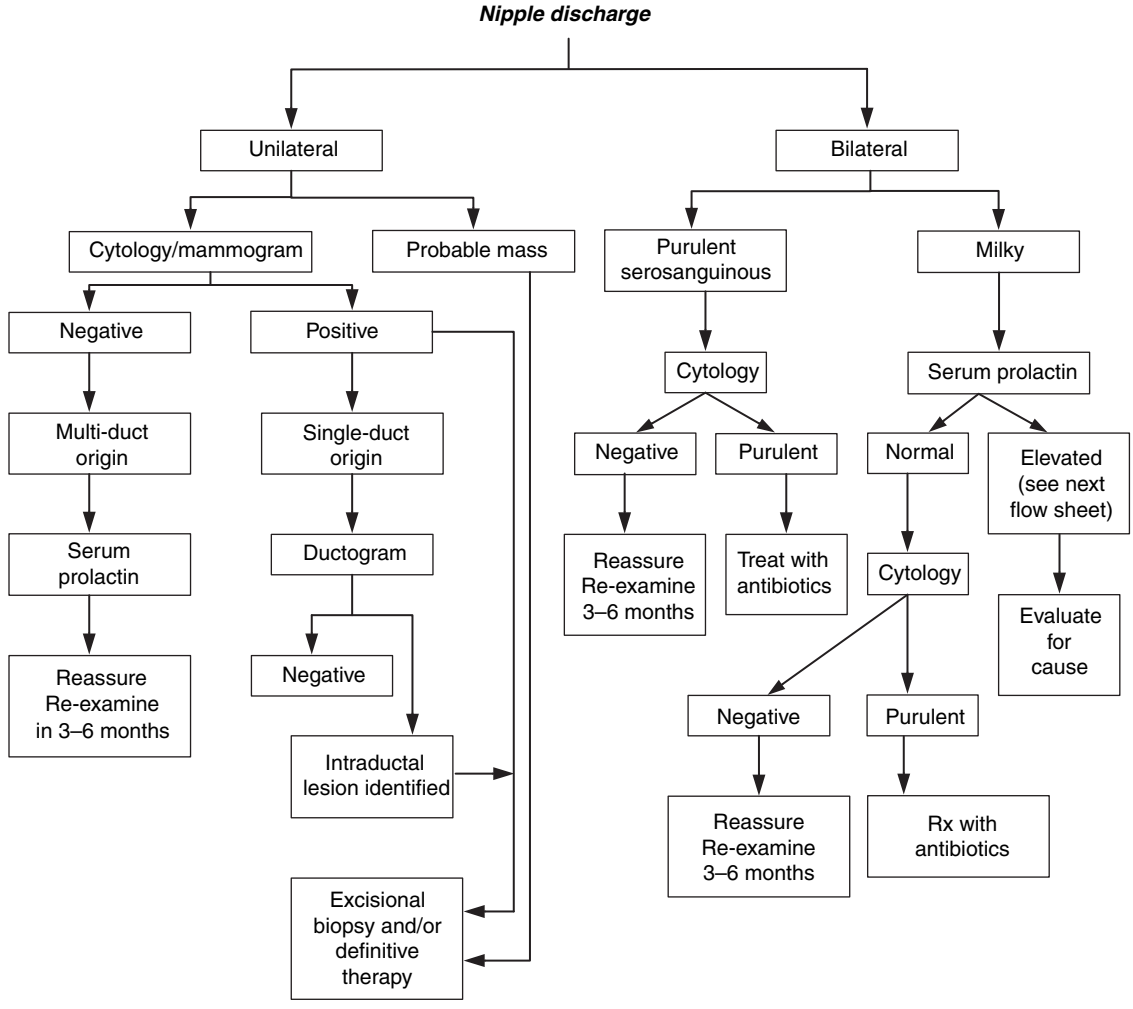
Types of nipple discharge

Color	*Other names*	*Most common cause*	*Frequency*	*% Caused by cancer*
Milky	Galactorrhea	Physiologic Breast-feeding Pregnancy Postpartum Prolactin excess Pituitary adenomas		Unknown
Multicolored	Sticky, green yellow, serous	Ductal ectasia		Rare
Purulent	Infected	Bacterial infection		Rare
Clear	Watery	Ductal carcinoma	2.2%	33.3–45%
Yellow	Serous	Fibrocystic disease	41.1%	5.9%
Pink	Serosanguinous	Fibrocystic disease Ductal papillomas	31.8%	12.9%
Bloody	Sanguinous	Fibrocystic disease Ductal papillomas	24.9%	27%

Breast mass

Diagnosis and treatment

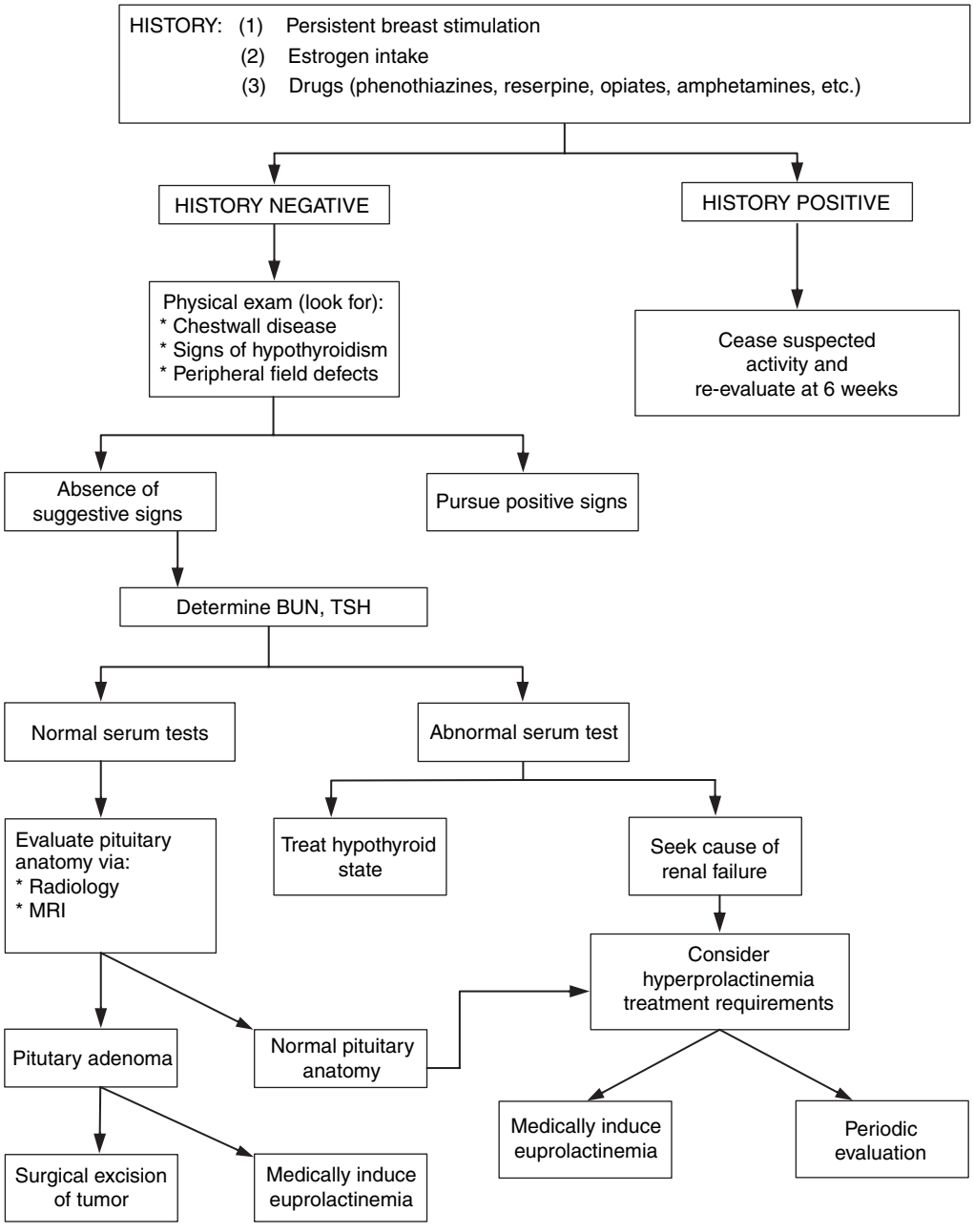



Nipple discharge
Unilateral
Bilateral
Cytology/mammogram
Probable mass
Purulent serosanguinous
Milky
Negative
Positive
Cytology
Serum prolactin
Multi-duct origin
Single-duct origin
Negative
Purulent
Normal
Elevated (see next flow sheet)
Serum prolactin
Ductogram
Reassure Re-examine 3–6 months
Treat with antibiotics
Cytology
Evaluate for cause
Reassure Re-examine in 3–6 months
Negative
Intraductal lesion identified
Negative
Purulent
Excisional biopsy and/or definitive therapy
Reassure Re-examine 3–6 months
Rx with antibiotics

Nipple discharge

Hyperprolactinemia

Repeat serum assay to rule out physiologic causes of prolactin secretion



Puerperal mastitis (three categories)

(1) *Milk stasis* – incomplete breast emptying causing engorgement and pain
(2) *Non-infectious inflammation* – arises when milk stasis is persistent and severe leading to edema, erythema, pain and tenderness
(3) *Acute mastitis* – final step in progression of disease characterized by: edema, erythema, pain, myalgias, chills, fever, tenderness

Predisposing factors

(1) Failure to empty breast adequately, most common
(2) Fissuring of nipples and bacterial inoculum from infant's mouth/mother's skin
(3) Incorrect preparation/care of nipples
(4) Improper positioning of infant for nursing
(5) Lowered maternal immune defenses

Diagnosis (acute puerperal mastitis)

(1) Symptoms: malaise, myalgias, fever, chills, pain
(2) Signs: edema, erythema, temp > 37.8°C (100°F), breast tenderness
(3) Milk cultures (discard first 3 cc)
  (a) Milk stasis: < 10^6 leukocytes/cc; < 10^3 bacteria/cc
  (b) Non-infectious inflammation: > 10^6 leukocytes/cc, < 10^3 bacteria/cc
  (c) Infectious mastitis: > 10^6 leukocytes/cc, > 10^3 bacteria/cc

Treatment

(1) Adequate milk emptying (continued nursing, infection extraductal)
(2) Moist heat
(3) Adequate hydration
(4) NSAIDs (ibuprofen, Naprosyn®, etc.)
(5) Empiric antibiotics:
  (a) Dicloxacillin 500 mg p.o. q. 6 h
  (b) Ampicillin 500 mg p.o. q. 6 h
  (c) Erythromycin 500 mg p.o. q. 6 h
  (d) Cefalexin 500 mg p.o. q. 6 h

Sequelae

(1) Persistent infection/breast abscess
(2) Increased risk if nursing discontinued
(3) Once diagnosed:
  (a) Cease nursing on infected side
  (b) Initiate IV antibiotics
    – Ancef® 1 g IV q. 6–8 h or
    – ampicillin 1 g IV q. 6 h
      AND
    – clindamycin 900 mg IV q. 8 h or
    – metronidazole 500 mg IV q. 6 h
      OR
    – Unasyn® 1.5–3.0 g IV q. 6 h
(4) Absence of favorable response within 48–72 h requires surgical I&D

Non-puerperal mastitis

Characterized as partial blockage of ducts by keratotic debris and squamous metaplasia

Predisposing factors

(1) Manipulation of breast (mammogram)
(2) Oral stimulation
(3) Adjacent cutaneous infection

Diagnosis

(1) *Acute* – pain, fever, edema, erythema, firm subareolar mass
(2) *Subacute* – similar presentation with tender, fluctuant mass
(3) *Chronic* – follows multiple, recurrent infections (sinus tracts, suppuration, fluctuant mass, pain and edema)

Treatment

(1) Acute – penicillinase-resistant penicillin plus metronidazole or broad-spectrum antibiotic (fluoroquinolone)
(2) *Subacute* – surgical I&D, broad-spectrum antibiotics

BREAST ABSCESS

	Suspect if failure of mastitis to respond to initial treatment within 18–72 h and/or development of palpable mass	
Diagnosis	Ultrasound and aspiration of exudate – culture and Gram stain	
Treatment	I&D (under general)	dependent area
		incision to follow circumareolar
		multiple with several incisions and dissect loculations
		leave open to heal by secondary intention
		frequent dressings with antibiotics
		TSS reported – breast disfigurement possible

BREAST BIOPSY (OPEN)

Required if:
- Bloody fluid on cyst aspiration
- Recurrence of cyst after three aspirations
- Bloody nipple discharge
- Nipple ulceration or persistent crusting
- Skin edema and erythema suspicious of inflammatory breast cancer

BREAST CANCERS

Established risk factors

High > 4	Older age (65–69 vs 30–34)	17 x
	Strong FMH (premenopausal first-degree relative or bilateral)	9 x
	Country of birth (North America or Northern Europe)	
	Personal history (*in situ* or invasive)	
	Biopsy showing proliferative lesion with atypia	
Moderate 2–4	Ductal hyperplasia/sclerosing adenosis	1.5–2 x
	Atypical ductal hyperplasia/lobular hyperplasia	4–5 x
	Lobular carcinoma *in situ*	8–11 x
	Nulliparity or late age at first birth	
	Upper class	
	Obesity	
	Primary relative with history of breast cancer (mother or sister)	3 x
Low 1.1–1.9	Early menarche, late menopause, history of breast cancer in one breast, complex fibroadenoma	1.5–4 x
	Moderate EtOH intake	
Gail model risk factor for women > 35	Tamoxifen should be started if (D/C ERT or HRT x 3 months prior to starting tamoxifen)	> 1.67
	Tamoxifen, when given to women with increased breast cancer, decreased the rate of breast cancer by	49%
	but increased the rate of endometrial cancer by	2 x
	Nolvadex (AstraZeneca's original brand name Tamoxifen) has been discontinued due to the wide availability of generic tamoxifen after June 30, 2006	
Key points	Leading cause of death in the USA for women at age 65 years or > is diseases of the heart (NOT breast cancer)	
	Cumulative absolute risk of death due to coronary heart disease in a woman 50–94 is approximately	30%
	Cumulative absolute risk of death due to breast cancer in a woman who is 50–94 is approximately	3%
	Four essentials of good breast care: (1) Clinical breast exam (2) Screening mammogram (3) Diagnostic mammogram (when abnormalities are present) (4) Tissue diagnosis (if abnormality does not resolve by follow-up breast exam or imaging studies) Communication and documentation are other imperatives	
	Recommendations for postmenopausal use of unopposed estrogen: (1) Assess mammographic density before and after initiation of ET. If density increases, stop therapy or reduce the dosage and repeat mammogram in 3–6 months	

(2) Measure high-sensitivity serum estradiol in women at high risk. Values in excess of 10 pg/l may reflect an increased risk of breast cancer in untreated women – although no particular level of concern has been definitively identified
(3) Individualize dose and length of therapy according to age and indication. (Arbitrary restriction of estrogen therapy to 5 years is not biologically rational or clinically justifiable.)

Breast cancer that develops in a woman using HT compared with breast cancer in a non-HT user is more likely to:

be diagnosed earlier
have a more favorable prognosis
be more well-differentiated

Progestogen effects on breast tissue include:

promotion of the growth of lobules in the breast alveoli
association with an increase in breast tumors in beagle dogs
biphasic effect of stimulation then inhibition with long-term use

Greatest relative risk for development of breast cancer with HT is when estrogen plus progesterone is used in a cyclic fashion

Women receiving estrogen plus testosterone therapy had a 17.2% increased risk of breast cancer per year of use. There was a 2.5-fold increased risk of breast cancer in current users of estrogen plus testosterone therapies compared with women who never used postmenopausal hormone therapy. The risk of breast cancer associated with current use of estrogen plus testosterone therapy was significantly greater compared with estrogen-alone (P=0.007) therapy and marginally (P=0.11) greater than estrogen plus progesterone therapy (Tamimi RM, *et al, Arch Intern Med* 2006: 166: 1483–9)

Key studies

Multicenter Breast Cancer Prevention Trial	Showed tamoxifen decreased breast cancer incidence to high-risk women by 49% But increased rate of endometrial cancer by 2 x
STAR study	Head-to-head study between tamoxifen and raloxifene. Clinical trial results indicate that raloxifene has no effect on the risk of coronary heart disease and is equivalent to tamoxifen in reducing the risk of invasive breast cancer. Neither drug increases the risk of strokes. It is estimated that both these drugs reduce breast cancer by about 50% It should also be noted that there were no statistically significant differences in the tamoxifen and raloxifene except that there was a statistically significant difference in uterine hyperplasia both with and without atypia in women in the tamoxifen group compared with those in the raloxifene group. STAR P-2 Trial – Vogel VG, *et al. JAMA* 2006; 295:2727–41.
RUTH study	The known favorable impact of raloxifene on the cholesterol-lipid profile was not robust enough to prevent coronary events
STARE study	Theoretical study suggested by Sarah Berga, MD after the report by O'Meara ES, *et al*. in *J Natl Cancer Inst* 2001;93:754–61 showed that women with estrogen receptor-positive breast cancer who decided to take HRT had lower overall mortalities, decreased risk of dementia and lower risk of breast cancer recurrence. This study would include estrogens in a head-to- head study with the 'antiestrogens' (tamoxifen and raloxifene) % of American women who will develop breast cancer sometime in their lives 12% How much more likely is a US woman to die from cardiovascular disease than breast cancer? 14 x
USC study	Highest odds ratio risk of breast cancer was associated with CYCLIC HRT
Nachtigall study	Data showed overall incidence of breast cancer in HRT users vs non-users was 0 vs 11.5%
Iowa Women's Health study	Found that breast cancer that *did* develop in HRT users was associated with favorable histological findings

Stallard study

Showed that non-users of HRT compared with HRT users were more likely to develop breast cancers that were ductal carcinoma *in situ*

Lobular carcinoma is associated with a better prognosis than ductal carcinoma

ATAC trial

More than 9000 women in 380 sites in 23 countries were enrolled in the Arimidex, Tamoxifen, Alone or in Combination. Compared with Tamoxifen, anastrozole increased disease-free survival by 14%

Other trials that favor aromatase Inhibitors over tamoxifen

The BIG trial, ITA trial, IES trial, and MA-17 trial. These trials suggest that one might consider 5 years of AI alone or sequential therapy with 2 to 3 years of tamoxifen followed by an AI for 2–5 years. Also consider giving AIs for a minimum of 2½ years to women who finish 5 years of tamoxifen

Based on Collaborative Group

Meta-analytic data, attributable risk of dying from breast cancer in women who started ERT at age 50 is 0.67%
For screening younger women at risk for breast cancer (especially premenopausal women with a hereditary risk of breast cancer), MRI may be a more accurate imaging technique than mammography (Stoutjesdijk MJ, Boetes C, Jager GJ, *et al*. MRI and mammography in women with a hereditary risk of breast cancer. *J Natl Cancer Inst* 2001;93:1095–102)
% that can be explained by risk factors 30–50%
Females with breast cancer who have no risk factors 80%
Invasive breast cancers who can be eliminated by prophylactic mastectomy 90%

BRCA1 lifetime risk of breast cancer is 45–80%
BRCA1 lifetime risk of ovarian cancer is 50%
BRCA2 lifetime risk of breast cancer is 85%
BRCA2 lifetime risk of ovarian cancer is 16%
BRCA2 lifetime risk of male breast cancer is 6%

No consensus on association of HRT with increased risk of breast cancer with postmenopausal women. Progestin does NOT protect. +FMH of breast cancer does NOT increase risk in HRT users
History of benign breast disease does NOT increase risk in HRT users
No data to support an increased risk of breast cancer recurrence or reduction in survival rate after admission or readmission of HRT

What % of women with breast cancer in pregnancy have positive lymph nodes? 50–80%
Axillary lymph node dissection is not recommended for DCIS as < 1% of patients have axillary node involvement when no evidence of microinvasion is present
LCIS has what amount of nodal involvement? None
LCIS is premenopausal and findings on physical, mammo and nodes are Negative
DCIS is pre- and postmenopausal, physical findings include mass, nipple discharge, mass or microcalcifications

Mammographic signs

Mammographic signs of malignancy

> 5 clustered ductal microcalcifications
Irregular stellate mass < 40%
Subtle signs (interval change)
Dilated duct or asymmetry (focal), get spot compression films

Biopsy if palpable
If non-palpable, get mammography-guided needle aspiration or core bx
Ultrasound for evaluation of cystic nature

Histology of fibroadenoma = benign ductal cells (staghorn)
normal stroma 'naked' or 'nude' bipolar nuclei
Tissue diagnosis is necessary for definitive diagnosis of invasion or to confirm *in situ* cancer

Therapies

Tamoxifen

What % of ER+ tumors respond to tamoxifen? 50%
What % of patients with metastatic breast cancer will have tumor regression in response to tamoxifen? 30%
Tamoxifen's metabolites include *N*-dimethyltamoxifen and 4-hydroxytamoxifen
N-dimethyltamoxifen has half-life that is how much that of tamoxifen? 2 x
4-Hydroxytamoxifen has a short half-life but has a binding affinity to the ER how much greater than estradiol? 20–30 x
How long should a woman stay on tamoxifen once she is taking tamoxifen? 5 years
Bisphonates appear to increase BMD – not inactivated by osteoclasts and appear to inhibit the adhesion of breast cancer cells to bone matrix
Tamoxifen (Nolvadex®) is a non-steroidal with potent antiestrogenic properties – its ACTION is to compete with circulating estrogens by binding to estrogen receptors
Metastatic breast cancer and adjuvant treatment of breast cancer (especially with NEG nodes and + ER)
Prophylaxis = multiple primary relatives with breast cancer, osteoporosis or history of lobular CIS of breast
BMD less with premenopausal women but increased with postmenopausal women
Changes: decreases LDL and total cholesterol but no effect on HDL @ 5 years after therapy

- Decreases cardiac events but slight increase in thromboembolic events. Use with caution in patients with history of stroke
- Can cause endometrial changes, eye changes (cataracts)

Doses: 10 mg and 20 mg
Recommendations for follow-up of patients on tamoxifen:
Annual gyn exam
Endo biopsy if abnormal bleeding, bloody discharge or spotting
Hysterectomy if atypical endometrial hyperplasia or cancer
TVUS (sonohysterography p.r.n.) – if endometrial biopsy not diagnostic
Megace® – endohyperplasia without atypia with follow-up endo bx

Chemo is used more commonly in premenopausal women because they are more likely to develop ER-negative tumors compared to postmenopausal women in this ratio 50% vs 75%
Some trials have shown, however, that tamoxifen is very active agent in premenopausal women with metastatic disease with an average overall response rate of 31%
Amenorrhea occurs in what % of premenopausal patients on tamoxifen? 1/3

Other hormonal therapies

Other hormonal agents used include aminoglutethimide (AG), anastrazole and progestins
Anastrozole – selective aromatase inhibitor (1 mg/day) response 30%
Megestrol acetate 160 mg/day

Chemotherapy

Chemotherapy is considered palliative. Standard regimens are:
5-fluorouracil (5-FU), doxorubicin and cyclophosphamide FAC
Cyclophosphamide, methotrexate and 5-FU CMF
Radiation treatment decreases recurrence risk by 1/2
Axillary dissection – modest improvement if any. Used for staging
Chemotherapy – the higher the risk – the higher the gain
Low risk reduce recurrence from 10–5%
High risk 50–25%

Biological therapies

Trastuzumab (Herceptin) cuts the risk of recurrence in half. 52% drop
This works best in cancers that overexpress the protein ErbB-2 also and better known as→ HER2/neu

Stages	< 2 cm	I
	> 2 cm or tumor with + lymph nodes	II
	Inflammatory, skin nodules or dimpling, locally advanced	III
	Metastatic to other areas from breasts	IV
N	Lymph nodes?	
Grade	Estrogen receptor status, necrosis, cytology, calcification, coarse or fine	
	Most common type of breast malignancy is infiltrating ductal carcinoma	

BREAST CYSTS/LESIONS

(1) Breast lesion < 2 cm with typical appearance of fibroadenoma = expectant management
(2) Simple cysts – fine-needle aspiration
(3) Clinically or radiographically suspicious – excisional biopsy

CANCER MOST COMMONLY FOUND IN UPPER OUTER QUADRANT

Aspiration technique

(1) Hold with two fingers
(2) Aspirated with 20 or 22 gauge needle
(3) If fluid is straw-colored, green-brown or green – reassure and follow-up in 2 weeks

BREASTFEEDING

Advantages	Decreases infant otitis, diarrhea, bacterial meningitis and maternal breast cancer	
Disadvantages	Mother needs to stay close, occasional mastitis, usually unfriendly work and societal environment. Four times more likely to have dyspareunia (*Am J Obstet Gynecol* 2001;184:881–90)	
	Patients leaving hospital breastfeeding	62%
	Goal of US Public Health to have patients leaving hospital	75%
Contraindicated	Lithium, methotrexate, Ergotrate©, bromocriptine, cocaine	
Mastitis	Take antibiotics and continue to breastfeed	

BRCA1

Increases lifetime risk of breast cancer by	45–80%
Increases lifetime risk of ovarian cancer by	45–50%

BRCA2

Increases breast cancer risk by	80%
Increases ovarian cancer risk by	16%
Increases male breast cancer by	6%

Evaluation of women with *BRCA1* and *BRCA2:*

- Monthly SBE to begin at 18–21 years old
- Annual or semi-annual exams to begin at 25 years old
- Annual mammography should begin at 25–35 years old
- *BRCA1* should also have Ca-125 and TVUS semi-annually at age 25
- Hysterectomy with BSO = ? insufficient evidence

BRCA1 and *BRCA2* = associated with breast ovarian cancer on chromosome 17q

BREECH

	Incidence of cord prolapse	4–7%
	Complete	5–10%
	Single footling breech	25%
	Frank breech (may be good candidate for vaginal delivery)	0.4%
	Congenital anomalies increased from	2.4–6.3%
	Risk of hyperextended head in a breech presentation	5%
	Frequency of breech presentation at term	3–4%
	In attempted vaginal delivery of breech-presenting fetuses, adequate progress of labor in multiparas =	1.5 cm/h
Risk factors	Prematurity Congenital anomalies Fetal neuromuscular disorder	
External cephalic version (ECV)	What proportion of ECVs lead to complications requiring stat delivery?	1–2%
	Risk factors for failure to ECV include: maternal obesity, frank breech presentation, primiparity	
	Contraindications to ECV include: gestation < 36 weeks, multiple gestation, oligohydramnios	
	C-section mothers with breech-presenting fetuses whose mothers have: contraindications to labor, inadequate X-ray pelvimetry findings, inadequate progress during labor	
Computed tomography pelvimetry	A recommended diagnostic modality to determine whether a TOL is appropriate in breech-presenting fetuses	
Forceps (Pipers, Elliot or Simpson)	Traction is not generally required or desirable. The goal of forceps assistance is to maintain flexion as long as possible during delivery of the aftercoming head	
• *Criteria for breech*	Well-flexed head Frank breech Zatuchni–Andros score (scored by parity, gestational age, EFW, dilatation, station, previous breech)	> 5
Mauriceau–Smellie–Veit maneuver	Most useful in rotating aftercoming head to AP	

BROMOCRIPTINE

	Pregnancy rate	80%
	Hyperprolactinemia causes approximately what % of ovulation disturbance?	15%
Side-effects	Nausea, nasal stuffiness, headache, orthostatic hypotension Decrease side-effects by gradual decreases in dosages	
Initial dose	1.25 mg/day p.o. then increase weekly in 1.25 mg increments	
What is it?	Semisynthetic ergot alkaloid with dopamine receptors	

BRONCHITIS

	Affects what % of adults?	25%
	Smoking is usually associated with what % of pts with chronic bronchitis?	90%
	Often viral. If bacterial *Hemophilus*, *Streptococcus*, *Moraxella*	
Treatment of choice	Cephalosporins Cefixime (Suprax®) 400 mg daily x 7–10 days Cefuroxime (Ceftin®) 250 mg b.i.d. x 7–10 days Loracarbef (Lorabid®) 400 mg b.i.d. x 7–10 days	

BROW PRESENTATION

Delivery CANNOT take place if brow persists

Etiology
Unstable
Usually converts to face or OP

Diagnosis
Abdominal palpation or vaginal exam

Prognosis
Poor unless birth canal is huge

Treatment
If progressing without any distress and no unduly vigorous contractions – no interference is necessary

BULIMIA

Enlarged parotids, erosion of dental enamel, hypotension, arrhythmias
See also Anorexia nervosa

BURNS IN PREGNANCY

Major burn defined as what % of surface area?	10%
Maternal mortality is 25% if surface area is	50%
Maternal mortality is 100% if surface area is	80%
Gestational age does not influence survival	
Stillbirth is 75% if burn is what % of surface area?	30%
Largest burn survived by mother AND fetus was	58%
Treatment is standard burn therapy with good prognosis for burn	< 30%
Consider delivery if burn is	> 50%

CANAVAN DISEASE

Developmental delay, macrocephaly, hypotonia and poor head control
Most children with Canavan disease will die in first decade of life
More prevalent among individuals of Eastern European Jewish (Ashkenazi) background
Caused by deficiency of the enzyme aspartoacylase, which leads to increased excretion of its substrate N-acetylaspartic acid (NAA)

CANCER

Most common cancer is	skin
Most common *gyn* cancer is	breast
Most common cause of cancer deaths	lung
Most common cause of *gyn* cancer *deaths* is ovarian (according to *Oncology Prolog* 2000)	5%
breast (according to Appleton and Lange)	15%
What % of women will develop breast cancer in their lifetime?	1 in 8
What % of women will develop ovarian cancer in their lifetime?	1 in 70
Endometrial cancer is the most common gyn cancer in women of 45 years of age	
The incidence of colon cancer increases with age	
Ovarian cancer is fourth leading cause of cancer death in women and according to the calculations from the Surveillance, Epidemiology and End Results (SEER) risk is	1/58

Ovarian cancer risk factors
Early menarche, late menopause, nulliparity or low parity, first term pregnancy after age 30 and frequent use of ovulation-inducing drugs

Breast cancer risk factors
Early menarche, late menopause, nulliparity and more than 30 years of age at first live birth

Gail model
Key risk factors to estimate individual risk for breast cancer include:
present age, age at menarche, age at first live birth, number of first-degree relatives with breast cancer and number of previous breast biopsies

Colon cancer	Lifetime risk for men and women in the USA is	6%
Uterine cancer risk factors	Obesity, chronic anovulation, diabetes and hereditary non-polyposis colorectal cancer	

CANCER AND GENETICS

Chromosome 17q associated with breast–ovarian cancer	*BRCA1* and *BRCA2*
Not associated with breast cancer	Lynch I syndrome
Increased risk of proximal colon cancer, stomach cancer, small bowel cancer, bile duct cancer and urinary tract cancers. Females at risk for endometrial and epithelial ovarian cancer	Lynch II syndrome
Rhabdomyosarcomas and osteosarcomas in children. Breast cancer and other tumors in mothers	
Germline mutation in *p53* tumor suppressor gene on chromosome 17q	Li–Fraumeni syndrome
Ovarian cancer cases develop as a result of an inherited abnormality in *BRCA1* and 2 gene products in approximately this % of patients	5–10%

CANCER AND PREGNANCY

Cervical (most common)		0.5%
Ovarian		1/8000–1/14 000
Colorectal		1/13 000
Breast		
Lymphoma		1/6000
Leukemia		1/75 000
Melanoma	0.14–2.8 (per 1000 births)	6–12/100 000
CIN in pregnancy	Study of 95 000 deliveries	SIL 0.14%
Cancer		0.7%

In pregnancy, there is eversion of cervix, therefore T-Z easily visible
Treatment = repeat colpo and Pap each trimester (according to *Prolog Gyn Oncology and Surgery*, 4th edn. Washington, DC: ACOG)

Cervical cancer in pregnancy

Most common cancer that occurs in pregnancy

Treatment: IA1 simple hysterectomy
>IA1 prior to 20 weeks do radical hyst with fetus *in situ*
After 20 weeks do hysterotomy for evacuation of fetus then radical hyst with pelvic lymphadenectomy

Ovarian cancer in pregnancy

Second most frequent gyn malignancy complicating pregnancy
Usually epithelial or germ cell
Characteristics on ultrasound

	> 8 cm
	internal complexities
	septations
	excrescences
	papillations
	ascites

CA-125 levels – usually falsely elevated in first trimester
Normally < 35 during second and third trimesters
AFP levels – normally elevated in pregnancies (100 x increase with an endodermal sinus tumor)
LDH and β-hCG – normally elevated in pregnancies, not useful markers
Chemo for germ cell tumors – bleomycin, etoposide, cisplatin, vinblastine
Chemo for epithelial tumors – cyclophosphamide and platinum compounds

Colorectal cancer in pregnancy

Third most common cancer in pregnancy and females in general
Tends to be more aggressive in pregnancy
Delay in diagnosis is common secondary to the pregnancy
Young patients may have a genetic predisposition
AVOID barium enema (0.82–1.14 cGy) in pregnancy

Breast cancer in pregnancy

Diagnosis: Ultrasound sensitivity is 93%
Mammography has a false-negative rate in pregnancy of 25%
Treatment: Treat malignancy and allow pregnancy to proceed.
Stage I + II – modified radical mastectomy with radiation exp to fetus.
Lumpectomy with postpartum radiation if diagnosis is in third trimester.
Stage III + IV – 5-year survival rate is only 10%
Advise: Postpone pregnancy for 2 years after initial diagnosis (the majority of recurrences occur within the first 2 years after dxn)

Lymphoma in pregnancy

Initial presentation is PAINLESS
Supradiaphragmatic lymphadenopathy
History: Weight loss > 10% in 6-month prior to diagnosis.
Unexplained fever > 38°C. Drenching night sweats
Treatment: IA + IIA – radiation Rx (delay until after delivery)
IB, IIB, III and IV – chemo and radiation

Leukemia in pregnancy

Extremely challenging and difficult. Most common cause of death is hemorrhage and infection
AML (acute myelogenous leukemia) – treat with cytarabine
ALL (acute lymphoblastic leukemia) – treat with vincristine and prednisone
ALL is associated with PTB, IUGR and stillbirths

Malignant melanoma in pregnancy

Treatment: stage I + II – wide local excision
Stage III – surgical excision (25–50% cure)
Metastasis:
(a) Isolated limb perfusion (with melphalan, interferon or TNF)
(b) Regional or systemic chemotherapy
(c) Radical therapy
(d) Intralesional immunotherapy
(e) Electropuration

Thyroid cancer in pregnancy

Diagnosis: Aspiration
Consider biopsy for (1) Cyst > 4 cm
(2) Complex cystic/solid component
(3) Recurrences after three aspirations
Treatment: (depends on histology)
(1) Most are well differentiated with good prognosis. Surgical resection is primary therapy – can delay until postpartum. AVOID 131-iodine – causes fetal hypothyroidism + cretinism
(2) Medullary. Stat total thyroidectomy
(3) Anaplastic – most aggressive

CANCER DEATHS

Accounts for what % of deaths in women?

Lung/bronchus	25%
Breasts	16%
Colon/rectum	11%
Pancreas	6%
Ovary	5%

CARDIOVASCULAR DEATHS ARE MUCH MORE COMMON THAN CANCER IN WOMEN

CARDIAC

Proportion of normal cardiac output that is generated by closed chest compressions during CPR	30%
Normal cardiac ejection fraction is	66%

Target heart rates

Non-pregnant	(220 – age) x 0.6–0.8
Pregnant	(220 – age) x 0.7

Clinical factors independently associated with perioperative cardiac complications

History	*Points*
Age > 70 years	5
MI in previous 6 months	10

Physical exam

S3 gallop or JVD	11
Important valvular aortic stenosis	3

Electrocardiogram

Rhythm other than sinus or PACs on last preoperative ECG	7
> 5 PVCs/min documented at any time before operation	7

General status

PaO_2 < 60 or $PaCO_2$ > 50 mmHg, K < 3.0 or HCO_3 < 20 mEq/l, BUN > 50 or Cr > 3.0 mg/dl, abn SGOT, signs of chronic liver disease or patient bed-ridden from non-cardiac disease	3

Site of operation

Intraperitoneal, intrathoracic or aortic operation	3
Emergency operation	4

General

Heart disease is the single leading cause of death among women. It kills more women than all the gynecological cancers combined!

What % of women die within 1 year of a recognized heart attack? 42%

The reason more women die than men is that women are less likely than men to receive timely, lifesaving diagnosis and therapy (stress testing, caths, anticlot agents and even simple lipid analysis!). Women tend to be worked-up for gallbladder or GERD rather than possible MIs like men

Women are generally older than men when they are diagnosed with CAD. This is probably because estrogen helps prevent plaque formation. Estrogen tends to elevate HDL and decrease LDL and triglycerides. This does not mean women do not have CAD, only that it is looked for at a much older age. If it was searched for, it could be found

Diagnosis

This should be the same as in men with the exception of the 'ultrafast CT and EBT' which do not detect calcification in women prior to age 40 at the same rate that they detect it in men (again, probably due to the level of estrogen). However, if she has increased risk factors, she should be screened more vigorously

Lipid screening (total cholesterol, LDL, HDL and triglycerides) should be evaluated routinely after age 20. If there is strong family history of premature CAD then she should be considered for advanced lipid testing (genetics and photyping) at least once for a screen of known increased risk factors such as Lp(a), homocysteine, small dense LDL or a low HDL_{2B}

Lipid parameters

(1) LDL-C < 100 mg/dl
(2) HDL-C > 40 mg/dl
(3) TG < 150 mg/dl

Metabolic syndrome (syndrome X)

(1) Insulin resistance (precedes type II diabetes in the majority of patients. Impaired insulin activity in the liver
(2) Hyperlipidemia
(3) Hypertension
(4) Abdominal obesity

In the metabolic syndrome, CHD risk approaches that of Type II DM
Define those at risk:

Male	> 40 inch waist circumference
Female	> 35 inch waist circumference
Triglycerides	≥ 150 mg/dl
Male HDL-C	< 40 mg/dl
Female HDL-C	< 50 mg/dl
B/P	130/≥ 85 mmHg
Glucose	≥ 110 mg/dl

Cigarette smoking, family history of heart disease or increased B/P or on B/P medicine

Metabolic syndrome increases in postmenopausal women
Metabolic syndrome is listed in ICD-9 New 277.7 through 277.79

Deadly → insulin resistance increased TG, SDLDL, proinflammatory state and hypercoagulability
Watch for multiple subtle risk factors

Screening for hypertension: untreated hypertension, even high normal blood pressures, increases risk 2–3 x more in women than it does in men

It is important for physicians to remember that when ordering stress testing, especially stress imaging (nuclear as well as echo) that these tests are more likely to be underread in women than in men, secondary to breast attenuation

Ibuprofen antagonizes the cardioprotective platelet inhibition that is induced by aspirin

CARDIAC ANOMALIES

Most common at birth is	VSD then PS then PDA
Most common in adults is	MVP then ASD
Eisenmenger syndrome	Right to left shunting 30–50% mortality rate Causes right ventricular failure
Pulmonary embolism	
Most reliable symptom is	dyspnea
Most reliable sign is	tachypnea
Get CXR, ventilation–perfusion scan	
Treat with heparin, reverse if necessary with protamine sulfate	
Mortality rate is	30%

CARDIAC DEFECTS

Management of cardiac valve defects in gravidas

Lesion	*Pathophysiology*	*Maternal complications*	*Key to therapy*	*Endocarditis prophylaxis**
Mitral stenosis	Limited left ventricular filling	Arrhythmia, pulmonary congestion	Optimize preload; avoid hypotension and tachycardia	Recommended
Mitral insufficiency	Atrial regurgitation	Limited cardiac output, arrhythmia, pulmonary congestion	Avoid hypotension and tachycardia	Optional
Aortic stenosis	Obstructed left ventricular outflow	Fixed cardiac output, compromised blood supply to coronary and cerebral arteries	Maintain cardiac output; reduce afterload; avoid hypotension and tachycardia	Recommended
Aortic insufficiency	Regurgitant cardiac output	Limited cardiac output, congestive heart failure	Avoid volume overload; reduce afterload	Recommended

Management of structural cardiac defects in gravidas

Lesion	*Pathophysiology*	*Maternal complications*	*Key to therapy*	*Endocarditis prophylaxis**
Atrial septal defect	Bidirectional atrial flow	Arrhythmia	Avoid volume overload	Recommended
Ventricular septal defect	Left-to-right shunt	Right ventricular overload	Avoid volume overload	No
Patent ductus arteriosus	Left-to-right shunt	Increased pulmonary flow	Avoid volume overload	Recommended
Eisenmenger syndrome	Pulmonary hypertension with bidirectional shunting	Congestive heart failure, hypoxia, sudden death	Recommended termination of pregnancy; supply continuous oxygen; avoid hypotension	Recommended
Tetralogy of Fallot	Ventricular septal defect, overriding aorta, pulmonary stenosis and right-to-left shunt	Congestive heart failure, hypoxia	Maintain preload delivery with limited afterload reduction; provide oxygen	Recommended
Coarctation of the aorta	Obstructed cardiac output	Limited cardiac output, congestive heart failure, aortic dissection or rupture	Reduce afterload; avoid volume overload	Recommended

Management of developmental cardiac valve defects in gravidas

Lesion	*Pathophysiology*	*Maternal complications*	*Key to therapy*	*Endocarditis prophylaxis**
Idiopathic hypertrophic subaortic	Obstructed outflow from left ventricle	Fixed cardiac output, congestive heart failure	Obstruction improves with volume expansion; avoid hypotension and tachycardia	Recommended
Marfan syndrome	Aortic regurgitation with aneurysm formation at the aortic root	Aortic dissection or rupture, marginal cardiac output, congestive heart failure secondary to regurgitation	Maintain cardiac output; avoid volume overload; prescribe beta-blockers	Recommended

*Endocarditis prophylaxis: ampicillin 2 g IV, then 1 g q. 4–6 h while in labor; gentamicin 1.5 mg/kg IV then repeated 8 h later

CARDIAC DISEASE IN PREGNANCY

New York Heart Association Classification	Class I	Asymptomatic
	Class II	Symptoms with greater than normal activity
	Class III	Symptoms with normal activity
	Class IV	Symptoms at bed-rest
NYHA (continued)	Group I (ASD, VSD, PDA, pulmonic/tricuspid disease, corrected tetralogy of Fallot, porcine valve, mitral stenosis – NYHA Class I + II)	
	Mortality	< 1%
	Obstetric risk of CHF	< 10%
	Group II (mitral stenosis with atrial fib, artificial valve, mitral stenosis –NYHA Class III + IV, aortic stenosis, coarctation of aorta –uncomplicated, uncorrected tetralogy of Fallot, Marfan syndrome with normal aorta and previous MI)	
	Mortality	5–15%
	Obstetric risk of CHF	80%
	Group III (Eisenmenger syndrome, Marfan syndrome with aortic root involvement, coarctation of aorta – complicated with diam > 4 cm, pulmonary hypertension)	
	Mortality	25–50%
	Obstetric risk of CHF	100%
Mitral valve prolapse	Most common congenital heart defect in young women Rarely affects maternal or fetal outcome	
Mitral stenosis	Most common rheumatic valvular lesion seen in pregnancy	

	10 years may lapse before the patient experiences symptoms of decreased cardiac output. Mild-to-moderate pulmonary congestion occurs at a pulmonary capillary wedge pressure of 18–25 mmHg Frank pulmonary edema appears at a wedge pressure of > 30 mmHg Great stress on cardiovascular system because of fixed cardiac output 20% of patients become symptomatic by 20 weeks' gestation Affected patients should limit their physical activity If volume overload is present, they should be diuresed carefully Arrhythmias (especially atrial fibrillation) should be controlled. If mural thrombi are present, anticoagulation is required with heparin C-section should be performed ONLY for OB indications. Swan–Ganz should be used if significant heart disease exists. Labor in left lateral position and receive supplemental oxygen. Avoid hypotension if epidural is administered. Use verapamil or digoxin to slow ventricular contraction rate if an atrial arrhythmia is present
Mitral regurgitation	May occur in patients with a history of (1) Rheumatic fever (2) Endocarditis (3) Idiopathic hypertrophic subaortic stenosis or (4) Mitral valve prolapse (most common) Decrescendo murmur is detected but is usually diminished in gravid Usually tolerated but may present with LHF (fatigue and dyspnea) Atrial enlargement and fibrillation may develop – might need CVP Epidural anesthesia is recommended (pain may lead to increased B/P and afterload, causing pulmonary vascular congestion) Patients with history of RF require either 1.2 million units of penicillin G q. month or daily oral penicillin or erythromycin throughout pregnancy
Aortic stenosis	Rarely seen in pregnancy Result of late complication of rheumatic fever Usually not symptomatic until 5th or 6th decade of life *Symptoms:* Angina and syncope upon exertion. 50% mortality rate in 5 years after symptoms appear During pregnancy, mortality for patients may be as high as 17% Sudden death from hypotension may occur Great care must be taken to prevent hypotension and tachycardia caused by blood loss, regional anesthesia or other medications Hydrate and place in left lateral position and use Swan–Ganz catheter Give antibiotic prophylaxis
Aortic regurgitation	Late complication of RF that appears 10 years after acute episode. Seen with Marfan syndrome or congenital bifid aortic valves. High-pitched, blowing murmur. Complete childbearing prior to symptoms or if LHF – repair before Target heart rate to be maintained should be 80–100 beats/min
Arrhythmias with cardiac disease	Best left untreated – ablate if serious and life-threatening. Artificial pacing and electrical defibrillation should not affect fetus
Ischemic heart disease	Most occur during third trimester 67% If MI occurs before 24 weeks' gestation, pregnancy should be ended. If delivery occurs within 2 weeks of acute event, the maternal mortality reaches 50% Management is same as non-pregnant (ICU, oxygen, analgesia). C-section only for obstetric indications. Epidural is safe Most common arrhythmia in pregnant female is PATs

Lethal dysrhythmia protocols

Ventricular fibrillation

Defibrillate at 200–300 J. Repeat if ineffective

Intubate and ventilate with oxygen. Give epinephrine, 0.5–1.0 mg IV. Repeat every 5 min. Give sodium bicarbonate, 1 mEq/kg (75–100 mg). Repeat with half the dose every 10 min as needed

Defibrillate at 360 J; repeat

Give bretylium tosilate (Bretylol®), 5 mg/kg IV (350–500 mg)

Defibrillate at 360 J; repeat

Give bretylium, 10 mg/kg IV (750–1000 mg)

Defibrillate at 360 J; repeat

After the maximum dose of bretylium or as an alternative, one may give lidocaine hydrochloride (Xylocaine®) or procainamide hydrochloride (Pronestyl®) as an adjunct to defibrillation

Give 1 mg/kg of lidocaine as an initial bolus and follow after 10 min by 0.5 mg/kg. This may be repeated until a total dose of 225 mg is reached and followed by maintenance infusion at 2–4 mg/min

Give 100 mg of procainamide over 5 min, repeated every 5 min. Stop bolus dosage on noting hypotension, suppression of dysrhythmia, a 50% increase in width of the QRS complex or on reaching a total dose of 1 g

Maintenance is 1–4 mg/min

Asystole

Intubate and ventilate with oxygen. Give epinephrine, 0.5–1.0 mg IV. Repeat every 5 min. Give sodium bicarbonate, 1 mEq/kg (75–100 mg). Repeat with half the dose every 10 min as needed

Give atropine 1.0 mg IV

Give calcium chloride 10% solution, 5 ml IV. Repeat every 10 min

Give isoproterenol (Isuprel) infusion, 2–20 mg/min

Arrange for pacemaker placement

Electromechanical dissociation

Intubate and ventilate with oxygen. Give epinephrine 0.5–1.0 mg IV. Repeat every 5 min. Give sodium bicarbonate, 1 mEq/kg (75–100 mEq). Repeat half the dose every 10 min as needed

Give calcium chloride, 10% solution IV 5 ml. Repeat every 10 min

Give isoproterenol infusion, 2–20 mg/min

Consider hypovolemia, tension pneumothorax and cardiac tamponade as possible causes and treat appropriately

Congenital heart disease	Incidence is 4–8/1000 Percentage of women with congenital heart disease who are pregnant who will deliver infants with same 50% L to R shunts usually corrected during childhood – if the defect has been corrected, the outcome is usually good. If not, pregnancy only slightly increases degree of shunting. If pulmonary hypertension has caused reversal of the shunt, the outcome of the pregnancy is dismal
(A) Atrial septal defects	Most common congenital heart lesions in adults. Usually exhibit pulmonary ejection murmur and a second heart sound that is split in both the inspiratory and expiratory phases. Usually well tolerated unless associated with pulmonary hypertension. (Atrial fib, pulmonary htn and HF usually do not arise until 5th decade.) For patients without complications, no special rx is necessary. Complicated patients need monitoring by both Ob and cardiologist. Prolonged bed-rest, invasive cardiac monitoring, treatment p.r.n.
(B) Ventricular septal defects	Usually close spontaneously or are corrected surgically in childhood. Rarely, uncorrected lesions lead to significant L to R shunts with PH. Epidural anesthesia and Swan–Ganz catheter are recommended. Fetal echo recommended. Incidence in offspring of VSDs is 4%
(C) Patent ductus arteriosus	Usually tolerated well in pregnancy unless pulmonary hypertension Pregnancy is not recommended for patients with large patent ductus
(D) Tetralogy of Fallot	(1) R ventricular outflow tract obstruction (2) VSD (3) Overriding aorta R to L shunt and cyanosis. If uncorrected, pt rarely lives past childhood If pregnancy does occur, incidence of HF is 40% Monitor patient for left heart failure. Monitor fetus for IUGR Counsel pt. Maternal cyanosis is associated with spontaneous abortion and preterm birth. Invasive cardiac monitoring is appropriate during labor. Use extreme caution with spinal or epidural anesthesia due to decreased B/P. Better choice of anesthesia includes systemic inhalation agents and local anesthetic
(E) Coarctation of aorta	Associated with other cardiac lesions as well as berry aneurysms. Characterized by a fixed cardiac output. Prevent *hypotension*. Newborn should be evaluated carefully as infants display cardiac lesions @ 2%
(F) Ebstein's anomaly	Congenital malformation of the tricuspid valve in which the right ventricle must act as both an atrium and ventricle. Ideally, surgical correction should be performed prior to pregnancy
Adult cardiac conditions that may worsen in pregnancy	
(A) Eisenmenger syndrome	When L to R shunt causes pulmonary arterial obliteration and pulmonary hypertension, eventually causing a R to L shunt Maternal mortality rate 50% Fetal mortality rate (if cyanosis is present) of more than 50% IUGR exhibited in this % of fetuses 30% Advise termination of pregnancy If pregnancy is continued, monitor postpartum with Swan–Ganz Avoid hypovolemia Postpartum death most often occurs within 1 week after delivery (sometimes 4–6 weeks after delivery)
(B) Marfan syndrome	Autosomal dominant disorder of the fibrillin gene – characterized by weakness of the connective tissues. Genetic counseling recommended. If aortic root is < 4 cm, risk is similar to general population. If aortic root is > 4 cm, risk of complications is significantly increased. Hypertension to be avoided – manage with β-blockers (second trimester +>). Epidural anesthesia during labor is considered safe

(C) Idiopathic hypertrophic subaortic stenosis	Autosomal dominant disorder. L ventricular outflow tract obstruction secondary to a hypertrophic interventricular septum. Genetic counseling is advised for affected patients *Treatment in labor:* (1) Inotropic agents should be avoided – may exacerbate the obstruction (2) Labor in left lateral decubitus position (3) Avoid/limit medications that decrease systemic vascular resistance (4) Monitor cardiac rhythm and treat tachycardia promptly (5) Second stage of labor should be curtailed by forceps or vacuum to avoid Valsalva's maneuver

CARDINAL MOVEMENTS OF LABOR

	Every **D**arn **F**ool **I**n **E**gypt **E**ats **E**lephants Engagement, Descent, Flexion, Internal rotation, Extension, External rotation, Expulsion
Cardiomyopathy (peripartum)	Maternal mortality rate 50% Develops in the last month of pregnancy or first 6 months postpartum without any obvious etiology Most common onset is during how many months postpartum? 3 months
Risk factors	Multiparity, AMA, multiple gestations and pre-eclampsia or eclampsia, etc.
Management	Bed-rest, sodium restriction, diuretics, inotropics and/or anticoagulants. Heart transplant if disease advanced
Labor	Monitor during and for at least 24 h postpartum. Give hydralazine, furosemide and/or digoxin and dopamine if necessary. Supplemental oxygen
Delivery	Epidural for pain control, curtain second stage with forceps or vacuum. C-section for OB indications
Incidence	(Increased risk with obesity, AMA and increased B/P, anemia, infection too) 1/4000 Symptoms are that of CHF (dyspnea, orthopnea, cough, palpitations, chest and abdominal pains) Treatment is that for CHF (digitalis, diuretics, anticoagulate as increased incidence of pulmonary problems. Heart transplant for end-stage heart failure). Future pregnancy if normal cardiac size and function, otherwise CONTRAINDICATED

CARDIOMYOPATHY

Cardiomyopathy is rare in patients who are at or near term, but it can be deadly. The prognosis for these women is really bad, with up to 85% dying by 5 years. Almost half of these deaths will occur within the first 6 months post partum
Consider the possibility in any woman who is pregnant or who has recently delivered and complains of swelling and trouble breathing

(1) Do a careful evaluation for cardiomyopathy in any pregnant patient who complains of shortness of breath, leg edema, or cardiac symptoms
(2) When the diagnosis is peripartum cardiomyopathy, use diuretics to reduce cardiac preload, vasodilators to reduce cardiac afterload, and inotropic agents to improve cardiac contractility
(3) Be cautious about diuresis in pregnant patients, though, because it decreases uterine perfusion
(4) For a woman with cardiomyopathy, vaginal delivery is preferable to C/S, and counseling on avoiding future pregnancies is imperative because of the high risk of cardiac complications

Diagnosis

Classic:
(1) Development of cardiac failure in last month of within 5 months postpartum

(2) Absence of an identifiable cause for cardiac failure
(3) Absence of recognizable heart disease before last month of pregnancy
(4) Additional:
Left ventricular systolic dysfunction demonstrated by classic echocardiographic criteria: ejection fraction < 45%, shortening fraction < 30%, left ventricular end-diastolic dimension > 2.7 cm/m^2

Signs and symptoms

Dyspnea, orthopnea, fatigue, edema, hypoxia. Fever uncommon

Lab results

CBC → normal
Liver function tests→ may be elevated if right heart failure involved
B-type natriuretic peptide→ elevated
Troponin, creatine kinase → normal unless ischemia
Creatinine → may be elevated
EKG→ Nonspecific changes. May reveal atrial fibrillation
Chest xray→ Cardiomegaly, pulmonary edema. Pleural effusions uncommon
Echocardiogram → ejection fraction < 45%
→ shortening fraction < 30%
→ dialated left ventricle

Treatment

Hospitalize, oxygen, IV access, pulse oximetry, fluid restriction, low-sodium diet
If still pregnant→obtain obstetric ultrasound, monitor fetal heart rate, give steroids for fetal lung maturity, once cardiac function is maximized – evaluate for delivery, minimize cardiac work during labor and delivery, use regional anesthesia to reduce preload and afterload, reserve C/S for usual indications, and start ACE inhibitor after delivery

Initiate medical therapy (goal SBP < 110 mmHg), improved symptoms.
Diuresis (i.e., furosemide)

Afterload reduction;
If pregnant→ Calcium channel blocker (e.g., amiodipine) or nitroglycerin (e.g., Isordil) or hydralazine
If delivered→ ACE inhibitor (e.g., enalapril, captopril)

Improve contractility with digoxin and/or dobutamine

If symptoms persist→ low dose beta blocker (e.g., metoprolol)

Anticoagulation for atrial fibrillation or intramural thrombus
Consider anticoagulation prophylaxis if pregnant or recently postpartum without above indications:
If pregnant→ Unfractionated heparin
If delivered→ Low-molecular-weight heparin, unfractionated heparin, or warfarin

Refractory pulmonary edema → add positive airway pressure
May require intubation

Counseling

Women with a history of PPCM and evidence of incomplete left ventricular recovery should be counseled to avoid pregnancy

CARPAL TUNNEL SYNDROME

Diagnosis

+ Tinel's sign – tap over carpal ligament produces tingling in fingers
+ Phalen's test – wrist flexion causing pain, numbness or tingling within 60 s is + in 60% of patients

Treatment

Non-pregnant or pregnant: splinting, vitamin B_6 100 mg daily, local steroid injection (1 ml or 40 mg) of Depo-Medrol with 1 ml of 1% lidocaine without epinephrine. Surgery only if necessary

Associated conditions and causes

Trauma-related structural changes	*Systemic diseases*	*Hormonal changes*	*Tumors/ neoplasms*	*Anomalous anatomic structures*	*Mechanical overuse*	*Infections*
Distal radius fracture	Rheumatoid conditions: arthritis, gout, cervical atrophy, intercarpal arteritis, tenosynovitis, bursitis, fibromyositis	Pregnancy	Lipoma	Aberrant muscles (e.g. lumbrical, palmaris longus, palmaris profundus)	Vibrating machinery	Tuberculosis (and other mycobacterial infections)
Lunate/ perilunate dislocations	Diabetes mellitus	Acromegaly	Ganglion	Median artery thrombosis	Prolonged hammering	Pyogenic infections
Post-traumatic arthritis/ osteophytes	Thyroid imbalance (especially hypothyroid)	Menopause	Multiple myeloma	Enlarged persistent median artery	Prolonged typing	Leprosy
Edema	Amyloidosis	Oral contraceptive use	Vascular tumors	Hypertrophy of palmaris longus muscle		
Hemorrhage/ hematoma`	Hemophilia	Systemic steroid use		Arteriovenous fistulas (hemodialysis)		
Burns	Alcoholism/ cirrhosis					
Colles' fracture	Raynaud's phenomenon, Paget's disease, obesity, syphilis, acromegaly, Cushing's disease, sarcoidosis, systemic lupus erythematosus, polymyositis, scleroderma, pernicious anemia, adiposita dolorosa, purpura simplex					

CELLULITIS

Causes	Most common cause is	uterine infection
Predisposed	Affluent females with C-section	13%
	Indigent females with C-section	27%
	Vaginal delivery	1–3%
	Vaginal delivery with increased risk	6%
	Anaerobic etiology in C-section	80%
Risk factors	(1) Duration of labor	
	(2) Duration of ROM	#1 + 2 most common
	(3) Multiple cervical exams	
	(4) Internal fetal monitoring	
	(5) Lower socioeconomic status	
	(6) Colonization of lower genital tract (GBBS, BV, *Chlamydia*, *Mycoplasma*)	
	(7) Abdominal twin delivery	3 x increased
Microbiology	*Anaerobes*	45%
	Peptococcus	
	Peptostreptococcus	
	Aerobes	
	Enterococcus	14%
	Gram negative bacteria (*E. coli*, etc.)	9%
	Group A, B, D	8%
	Staphylococcus aureus	
	Others	
	Mycoplasma, Ureaplasma, Chlamydia	
	80% cases of infection after C-section are anaerobic	
Pathogenesis	Bacterial contamination from vaginal flora – metritis	
Diagnosis	FEVER, uterine tenderness, purulent or foul-smelling lochia	
	Labs – WBC usually	15 000–30 000/μl
	Blood culture most helpful	
Therapy	Clindamycin–gentamicin is curative	85–95%
	Others: cefoxitin, piperacillin, cefotetan	
	Treat until afebrile for	24–48 h
	Further treatment on outpatient basis usually not necessary	
	Preference: cefotetan 2 g	q. 12 h
	or ampicillin/sulbactam 3 g	q. 6 h
	or clindamycin 900 mg	q. 8 h
	with gentamicin 2 mg/kg then 1–1.5 mg/kg or 5–7 mg/kg once daily	q. 8 h
Persistent fever	Abscess? Resistant organisms? Wound infection? Infection at other sites? Septic thrombophlebitis?	
	What % of metritis respond within 48–72 h to ab regimens?	90%
	(1) Abscess – drain	
	(2) Resistant organisms – switch antibiotics	
	(3) Wound infection – I&D, debridement and antibiotic therapy	
	(4) Infection at other sites	
	(5) Septic thrombophlebitis – antibiotics with full heparinization	
	Prophylaxis: Antibiotic to patients undergoing non-elective C-section. Use short course of 1–3 doses and initiate after cord clamping. Choices: ampicillin, cephalothin, cefazolin	

CERCLAGE

Etiology of cervical incompetence

Obscure. Risks are increased with cervical trauma or DES exposure

Diagnosis

Characterized by painless dilatation of the cervix in the second (or early third) trimester. May be associated with bulging membranes and eventually, rupture of membranes followed by expulsion of a premature fetus

Evaluation

Use vaginal ultrasound to see if there is cervical shortening < 2.5 cm
Cervical length is usually > 3 cm. Increased preterm delivery history
If cervix ≥ 3 cm, the risk of PTD is 5%
If cervix is <2 cm, the risk of PTD is 77%
If the cervix is <3 cm, the patient may benefit from steroids + transfer
Amniocentesis? Gram stain for aerobic and anaerobic bacteria, mycoplasmas, WBC count + glucose. May not want cerclage or tocolysis
One study (Sakai M, Shiozaki A, Takata M, *et al.* Evaluation of effectiveness of prophylactic cerclage of a short cervix according to interleukin-8 in cervical mucus. *Am J Obstet Gynecol* 2006: 194:14–19) suggests that doing a cerclage in a patient with a positive IL-8 could be harmful but does show that doing a cerclage in a patient with a negative IL-8 could be helpful

Observational studies

Health – patients with a short cervix undergoing cerclage had a 10 x reduction in the rate of preterm birth
Two other studies did not find cerclage beneficial (Berghella V, Haas S, Chervonera I, *et al*. Patients with prior second-trimester loss: prophylactic cerclage or serial transvaginal sonograms? *Am J Obstet Gynecol* 2002;187:747–51; Berghella V, Daly SF, Tolosa JE, *et al*. Prediction of preterm delivery with transvaginal ultrasonography of the cervix in patients with high-risk pregnancies: does cerclage prevent prematurity? *Am J Obstet Gynecol* 1999;181:809–15; Hassan SS, Romero R, Maymon E, *et al*. Does cerclage prevent preterm delivery in patients with a short cervix? *Am J Obstet Gynecol* 2001;184:1325–9)
Doing a cerclage in the face of a positive cervical mucous IL-8 value resulted in the highest PTB rate before 37 weeks at 78%, and a much shorter procedure-to-delivery interval. (Sakai *et al*, *Loc cit*)

Randomized studies

(1) CIPRACT (Cervical Incompetence Prevention Randomized Cerclage Trial) enrolled patients only at risk for PTD + results similar (Althuis S, Dekker G, Hummel P, *et al*. Cervical Impotence Prevention Randomized Cerclage Trial (CIPRAT): effect of therapeutic cerclage with bed rest vs. bed rest only on cervical length. *Ultrasound Obstet Gynecol* 2002;20:163–7)
(2) Rust Trial – no difference in cerclage and control group (Rust OA, Atlas RO, Jones KJ, *et al*. A randomized trial of cerclage versus no cerclage among patients with ultrasonographically detected second-trimester preterm dilatation of the internal os. *Am J Obstet Gynecol* 2000;183:830–5)
(3) Fetal Medicine Foundation of UK – no difference in patients with short cervix and no risk factors

Contraindications to cerclage

(1) Bleeding
(2) Contractions
(3) Rupture of membranes
(4) Chorioamnionitis
(5) Dilatation > 4 cm
(6) Polyhydramnios
(7) Fetal anomaly

Timing of cerclage

Delay until about 14 weeks EGA (past SABs timing) 14–26 weeks

Pre-op evaluation

(1) Ultrasound (r/o major anomalies, confirm viability)
(2) Screen for: GC, *Chlamydia* and Group B Streptococcus. (treat + cultures)
(3) No coitus at least 1 week prior and 1 week after cerclage

Types of cerclage

McDonald – procedure of choice
(1) Purse-string technique using 5 mm Merselene band
(2) 4–5 'bites' at level of internal os (encircle cervix)
(3) Knot placed anteriorly (facilitates removal)

Shirodkar – more difficult
(1) Used with previous McDonald failures
(2) Submucosal placement (bladder mobilized cephalad)
(3) More closely approaches level of internal os

Modified Shirodkar
Anterior to posterior bilaterally – tying posteriorly and burying the anterior knot at 12 o'clock

Transabdominal – suture at internal os during laparotomy. Use for:
(1) Traumatic cervix
(2) Congenital shortening
(3) Previous failed vaginal cerclage
(4) Advanced cervical effacement

Emergency procedures
(1) Elevation of bulging membranes
  – overfill bladder with 1000 cc saline
  – Trendelenberg (with or without Foley displacement)
  – use sponge stick with condom cover
(2) Saskatchewan procedure – sutures tied across external os
(3) Wurm procedure – 2 to 10 to 8 to 4 and tie at 3 then 1 to 5 to 7 to 11 and tie at 12

These and other cerclage procedures are described in detail in Turrentine JE. *Surgical Transcription in Obstetrics and Gynecology*, 1st and 2nd edns. Carnforth, UK: Parthenon Publishing, 1994. London: Informa Healthcare, 2006.

Complications

(1) Fetal loss – rate is 2 4%
(2) Infection (this is decreased if done prior to 18 weeks' gestation)
(3) Rupture of membranes
(4) Chorioamnionitis (has been documented as high as 60%)
(5) Preterm delivery (26%)
(6) Cervical lacerations (3–13%)
(7) Cervical dystocia secondary to scarring (5%)

Management

Infection – cut cerclage and induce

Rupture of membranes – if @ 48 h after cerclage there is an increased risk of fetal or maternal infection if left in place. Release suture if delivery is imminent!

Placement of an early cerclage does not result in improved outcomes over serial early transvaginal ultrasound of the cervix (Kelly S, Pollock M, Maas B, *et al*. Early transvaginal ultrasonography versus early cerclage in women with an unclear history of incompetent cervix. *Am J Obstet Gynecol* 2001;184:1097–9)

CEREBRAL PALSY

(1) Rate per births 1–2 per 1000
(2) No evidence that asphyxia causes CP
(3) ALL of the following MUST be present to link CP with birth:
  (a) Umbilical artery pH < 7
  (b) Apgar score 0–3 for > 5 min
  (c) Neonatal neurological sequelae (seizure, coma, hypotonia)
  (d) Multiple organ system dysfunction
  Cardiovascular, pulmonary, renal, hematologic, gastrointestinal

Despite the above evidence that usually does not link CP with birth, most medical–legal cases of CP are settled out of court because of the fear of large jury awards (over what the doctor's insurance covers) against the doctor even though the doctor is not to blame for the condition

CERVICAL CANCER

CIN statistics

CIN I to cancer	1%
CIN II to cancer	15
CIN III – Mean age	28 years old

	ASCUS – Approximately what % of Paps?	5
	Normal cervix to CIN III in years	4.5 years
	LEEP – Complication of bleeding from cervix	5%
	Complication of stenosis of cervix	1%
	Conization depth if cervix is 2 cm is	20 mm
CIS of cervix	*See* Oncology (Cervix)	
	Facts about preinvasive cancer of the cervix:	
	(1) Mean age of CIS to cancer	15 years
	(2) CIN in the USA	600 000 cases
	(3) HPV– potentially dangerous	16, 18, 31, 33, 35
	(4) Frequency of Pap smears if patient has HIV is	every 6 months
	(5) CIN I progresses to cancer	1%
	CIN II progresses to cancer	15%
	CIN III – mean age is	28 years old
	Transition time from normal Pap to CIN III is	4.5 years
	(6) ASCUS comprises which % of Pap smears?	5%
	(7) LEEP – complications include bleeding 5% of the time and stenosis of cervix 1% of the time	
	For information on staging, nodes and treatment, *see* Oncology (cervix)	
Incidence	1/6 of all genital cancers; whites: 15/100 000; blacks: 34/100 000	
Risk factors	(1) Early age of coitus (2) Multiple male sexual partners (3) Smoking (smoking increases incidence by 3.5 times) (4) HPV (5) HIV	
Prevention	HPV vaccine is now available and although it will take decades to see cervical cancer rates drop, we will soon see fewer CIN 2/3 lesions once HPV 16/18 vaccination is routine. The quadrivalent vaccine also targets HPV types 6 and 11, which cause 90% of genital warts. Primary target for the vaccine will be children prior to sexual activity. Women who are already sexually active can also receive the vaccine. Merck has Gardasil Quadrivalent Vaccine providing 100% protection against persistent HPV 6, 11,16, 18 and HPV 16/18-related CIN. Girls and women between ages 9 and 26 should be recommended for vaccination – ideally before the onset of sexual activity, but women who are already active should also be vaccinated because they may not have been exposed to all the HPV types that the vaccine protects. The vaccine is category B and therefore considered "safe" near the time of conception. GlaxoSmithKline expects to put Cervarix Bivalent HPV 16, 18 vaccine on the market sometime in 2007	
Screening	(1) Annual Paps to begin after first sexual activity and/or after age 18 (2) Test every 1–2 years until age 30. (Some recommend that after three normal Paps, screening can be every 2–3 years after age 30 as the squamous metaplasia area – the substrate for neoplasia – is diminished in most women in their 30s.) However, if an older woman's sexual practices change, consider restarting more frequent screening (3) Consider discontinuing Pap tests after age 65–70 in well-screened women with no history of significant dysplasia. Evidence does not support a specific age to stop screening. Again, restart screening if sexual practices change to more frequency (4) Consider discontinuing Pap testing in women whose uterus and cervix have been removed and who have no history of high-grade cervical dysplasia or cancer. (However, consider screening vaginal cuff and walls for vaginal dysplasia every 1 to 2 years.)	

(5) Continue annual Pap testing in women with a history of cervical cancer *in utero* exposure to diethylstilbestrol (DES), or who are immunocompromised
(6) Screening will continue long after the advent of multivalent HPV vaccines to prevent the 30% of cancers linked to high-risk HPV types that are not in the vaccine, to protect the unvaccinated, and to protect the previously HPV-infected
(7) HPV 16/18 testing may permit less aggressive management of women with other high-risk HPV infections. A single positive test for HPV 16/18 is twice as likely as an LSIL Pap to identify women at high risk for CIN 3+. Based on data obtained since the 2001 Consensus Conference, it now appears reasonable to incorporate knowledge of a woman's HPV status in management of atypical glandular cell (AGC) cytological abnormalities. Current data clearly indicate that women with ASCUS who are HPV DNA-positive and women with LSIL have the same risk of having high-grade disease and should therefore be managed identically. **When cytology is negative and HPV is positive, repeat both tests in 6–12 months**

Etiology

E6 and E7 viral proteins produced by HPV disables p53 and Rb host

Diagnosis

(1) Biopsy
(2) Colpo with biopsy
(3) LOOP (LEEP) or cone if:
(a) Bx does not explain abnl cells; (b) Atypical epithelium extends to endocervical canal; (c) Abnl cytology with no visible colposcopic lesion; (d) Microinvasion of bx; or (e) ECC demonstrates CIN

Presentation

(1) Abnormal vaginal bleeding
(2) Discharge
(3) Postcoital bleeding
(4) Prolonged menses

Advanced disease:
(1) Pelvic and sciatic pain
(2) Leg edema
(3) Voiding difficulties

Clinical staging

(1) Physical (inspection and palpation of cervix, vagina, parametrium and pelvic side-walls. Check supraclavicular node region and upper abdominal region
(2) CXR (not CT or MRI)
(3) Colpo
(4) Cystoscopy
(5) IVP
(6) Flexible sigmoidoscopy or proctoscopy and/or BE

Stage patient while she is under anesthesia – stage cannot be changed. *See* staging under Oncology (Cervix)
However – VERY IMPORTANT TO REMEMBER:
Microinvasive cancer of cervix
Stage IA – identified only microscopically (all stages over this are gross lesions even with superficial invasion)
Maximum depth = 5.0 mm. No wider than 7.0 mm
IA1 – stromal invasion to 3 mm. No wider than 7.0 mm
IA2 – stromal invasion of 3–5 mm. No wider than 7.0 mm
Vascular space involvement (either venous or lymphatic) does *NOT* alter staging
See All Stages of Cervical Cancer in *Clinical Protocols in Obstetrics and Gynecology (The TAN Book),* Turrentine JE, Aviles M, Novak JS. Carnforth, UK: Parthenon Publishing, 2000:141

Treatment of early lesions

Remember, Stage IA1 and sometimes IA2 – Conization if margins are free and no lymph-vascular space invasion then simple hysterectomy (lymphectomy not recommended as no metastasis). However, most IA2 (> 3 mm) are treated with radical hysterectomies with pelvic lymphadenectomies (3% node +)

Treatment

Surgery or radiation

Radiation if > Stage I and IIA
Controversial if Stage IB and Stage IIA
Surgery for young patients (ovarian and vaginal function preserved)
• Radical hysterectomy: remove uterus, upper 25% of vagina, uterosacral and uterovesical ligaments, bilateral parametrium and pelvic dissection of ureteral, obturator, hypogastric and ileac nodes

Summary of treatment

(1) CIN
   (a) LEEP; (b) CO_2 laser; (c) Cryo; (d) Electrocautery (perform a–d when invasive cancer excluded and cone not indicated)
   Must see the entire abnl epithelium and endocx free of lesion and cytology, colposcopy and histology, all must correlate
   Follow-up with repeat Pap 6 months after treatment
(2) Stage IA Cx ca – hysterectomy (patients with lesions <3 mm which is stage IA1 then cone is okay)
(3) Stage IB, IB1, IIA – radical hyst with bilat pelvic lymph nodes or radiation
(4) Stage IIB, IIIA, IIIB, IVA – radiation treatment, some radiation potentiator hydroxyurea, multichemotherapy and surgery
(5) Recurrent – pelvic exenteration
(6) If can NOT handle radiation or surgery – chemo (cisplatin alone)
(7) Pregnant
   (a) Fetus not viable – treat as non-pregnant; (b) Second trimester – consider termination; (c) Later – consider survival of fetus versus risk to wait

CERVICAL RIPENING

• Use of transcervical Foley catheter for preinduction cervical ripening is both as safe and efficacious in the outpatient setting as it is in the inpatient setting (Sciscione AC, Muench M, Pollock M, *et al*. Transcervical Foley catheter for preinduction cervical ripening in an outpatient versus inpatient setting. *Obstet Gynecol* 2001;98:751–6)

Cost differences

Misoprostol (Cytotec) Tablet	$0.36–1.20 for 100 μg tablet
Dinoprostone (Prepidil) Gel Kit	$65.00–75.00
Dinoprostone (Cervidil) Vaginal Insert	$165.00

Sublingual misoprostol

More effective than oral misoprostol for cervical ripening according to Shetty A, Danielian P, Templeton A. Sublingual misoprostol for the induction of labor at term. *Am J Obstet Gynecol* 2002;186:72–6
Buccal administration of misoprostol is an acceptable alternative, yielding rapid onset of action and avoiding repeated vaginal exams according to Carlan SJ, Blust D, O'Brien WF. Buccal versus intravaginal misoprostol administration for cervical ripening. *Am J Obstet Gynecol* 2002;186:229–33
Doses used in study: buccal route – q. 6 h × 6 doses, first two doses were 200 μg tablet then 300 μg for last four doses compared to vaginal route – q. 6 h × 6 doses, first two doses were 50 μg tablet increased to 100 μg tablet for the last four doses

CERVICITIS

Coinfection rate is as high as 60%
Treat both *N. gonorrhoeae* and *C. trachomatis*

Treatments

(1) Ceftriaxone 125 mg IM plus doxycycline 100 mg p.o. b.i.d. × 7 days
(2) Ofloxacin 400 mg p.o. plus doxycycline 100 mg p.o. b.i.d. × 7 days
or
(3) Ceftriaxone 125 mg IM plus azithromycin 1 g orally

CERVIDIL

Incidence of hyperstimulation	5%
Hyperstimulation usually occurs after placement within	1.5–9.5 h

ACOG recommends uterine monitoring continuously electronically for as long as device is in place and for 15 min after removal (ACOG Technical Bulletin No. 209, October 1998)

Cervidil (Dinoprostone, 10 mg) is for hospital use only and should be opened only at the tear mark and *never* with scissors or a sharp object as it may compromise or cut the pouch that serves as the retrieval system for the polymeric slab. Make sure slab is obtained

CHANCROID

Symptoms and appearance	Acute painful ulcer of vulva with ragged edges – solitary or multiple
Cause	*Hemophilus ducreyi*
Diagnosis	Gram stain shows classic streptobacillary chains – 'school of fish'
Treatment	TMP–SMX or erythromycin or Rocephin®

CHANGES IN PREGNANCY

See Adaptations in pregnancy

CHEMOTHERAPY

S phase (DNA synthesis)

Alkylating agents	Interact with DNA:	
	Cyclophosphamide	Hemorrhagic cystitis
	Chlorambucil, cyclophosphamide, melphalan	Leukemia
	Ifosfamide	Coma
Antitumor antibiotics	Directly attack DNA, producing breaks + interfering with DNA synthesis:	
	Actinomycin D	
	Doxorubicin	
	Bleomycin	Pulmonary fibrosis
Antimetabolites	Interference with DNA and RNA synthesis:	
	5-FU (radiation sensitizer)	Cerebellar ataxia
	Methotrexate	Increase bone marrow toxicity
Platinum compounds	Varied action (interfere with no single mode of action), sometimes bind to DNA:	
	Cisplatin	Renal toxicity, deafness
	Carboplatin	N&V, myelosuppression
Topoisomerase II inhibitors	Inhibit the enzyme topoisomerase II and cause double-stranded DNA breaks	
	Etoposide (VP-16):	

M phase (mitosis)

	Most sensitive to radiation during this stage of cell kinetic cycle	
Vinca alkaloids and taranes	Arrest mitosis with toxic destruction of mitotic spindle:	
	Vinblastine	Increased bone marrow toxicity
	Vincristine	Increased neurotoxicity
	Paclitaxel	Arrhythmias

Specific reactions

(1) Bone marrow toxicity
Doxorubicin, vinblastine, methotrexate, carboplatin
(2) Pulmonary fibrosis
Paclitaxel, bleomycin
(3) Alopecia
Ifosfamide, 5-FU, doxorubicin, methotrexate
(4) Severe inflammatory/ulcerative reactions
Doxorubicin, mitomycin C, actinomycin D
(5) Cardiotoxic

Doxorubicin
- Meticulous dental hygiene should be practiced during and after antineoplastic therapy to modify complications of oral stomatitis

CHEMORADIATION

New treatment for cervical cancer
Examples of radiation sensitizers are — 5-Fluorouracil, Cisplatin, Mitomycin C, Hydroxyurea

Benefits to administering chemotherapy concurrently with radiotherapy are:
(1) Cell cycle synchronization
(2) Decreased risk of cross-resistance
(3) No delay in therapeutic modalities
(4) Decreased oxygen-depleted fractions

Disadvantages include
(1) Unknown long-term complications
(2) Potential for increased side-effects

Types of chemoradiation
(1) Neoadjuvant – chemo given for variable # of cycles prior to definitive treatment
(2) Concurrent – chemo and radiation are administered simultaneously. Most effective in primary treatment for cervical cancer
(3) Adjuvant – definitive treatment (radiation) is followed by chemo

Chemoradiation has decreased risk of disease recurrence for patients with advanced-stage cervical cancer by approximately — 30–50%

CHLAMYDIA

Frequently asymptomatic
Rate of perinatal transmission — 60–70%
Treatment is azithromycin p.o. — 1 g

Screening
Do not use wooden shafts – preservatives are toxic to *C. trachomatis.*
Sensitivity of ligase chain reaction assay from first-stream urine catch is approximately 95%
Advantages of ligase chain reaction:
(1) Improved sensitivity
(2) Less resource intensive than pelvic so better for widespread use
(3) More comfortable than pelvic thus increasing compliance + use
(4) *N. gonorrhoeae* can be obtained from same urine specimen

Classification of Chlamydia
A, B, Ba and C — Blinding trachoma
D through K — NGU, PID, cervicitis, epididymitis, proctitis and conjunctivitis
L1 through L3 — LGV
Diagnosed by microimmunofluorescence
Treated with doxycycline, tetracycline, sulfa or chloramphenicol

CHOLECYSTITIS

Increased risks
Rapid weight loss
OCPs — 2 x increase
Lipid-lowering medications — 33%
Increase liver enzymes (AST + ALT)

Anatomy

Gold standard
Endoscopic retrograde cholangiopancreatography — ERCP
To test for stones in the common bile duct

Cholangitis and acute pancreatitis can be life-threatening but what % of patients remain asymptomatic for years (but at any time can develop a crisis)? 8–15%

CHOLESTEROL

Facts

35% of heart attacks occur even when cholesterol levels are 150–200 mg/dl
Triglyceride levels are dangerous when above 150 mg/dl
Estrogen increases HDL, VLDL and triglycerides
Progestins increase (*Prolog* 4th edn RepEndo + Infert. Washington, DC: ACOG) LDL

Dietary fiber

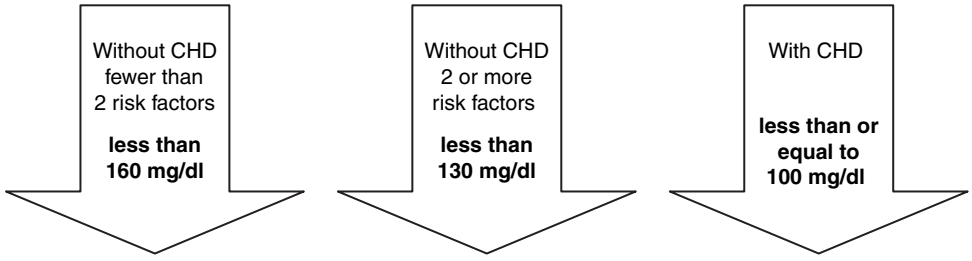
Dietary measures, such as addition of soluble fiber and substitution of soy protein for meat and dairy products, can help patients achieve lower cholesterol levels. (Each 1% reduction in serum cholesterol can reduce heart disease mortality by 2%.) *Alternative Med Alert* December 2001

Treatment outline

See following protocols

National Cholesterol Education Program – Guidelines and Goals for your Patients at Risk

When diet and exercise are not enough to lower cholesterol, the NCEP recommends lowering LDL-cholesterol following the guidelines below. Using these levels and risk factors as guidelines, medications such as atorvastatin (Lipitor), fluvastatin, pravastatin, simvastatin or lovastatin may be started



NCEP recommends lowering LDL-C further than these goals if possible

NCEP recommends lowering LDL-C further than these goals if possible

Identifying patients with CHD risk factors

Family history of early CHD

Any parent or sibling with CHD (younger than 55 years if male and younger than 65 years if female)

Age

Male ≥ 45 years; female ≥ 55 years or premature menopause without estrogen replacement therapy

– Men in their forties are four times more likely to die from CHD than women of the same age. After menopause, the incidence of CHD increases progressively in women until ultimately as many women as men die of CHD

Hypertension

Blood pressure ≥ 140/90 mmHg or on antihypertensive medication

– Because it is difficult to determine how long blood pressure has been controlled versus uncontrolled, even patients undergoing treatment are considered to be at risk

Current smoker

Smoking cessation is one of the most effective ways to reduce the risk of CHD and other atherosclerotic diseases

Diabetes mellitus

In men, diabetes triples the risk of CHD; in women, the increase in risk may be even greater

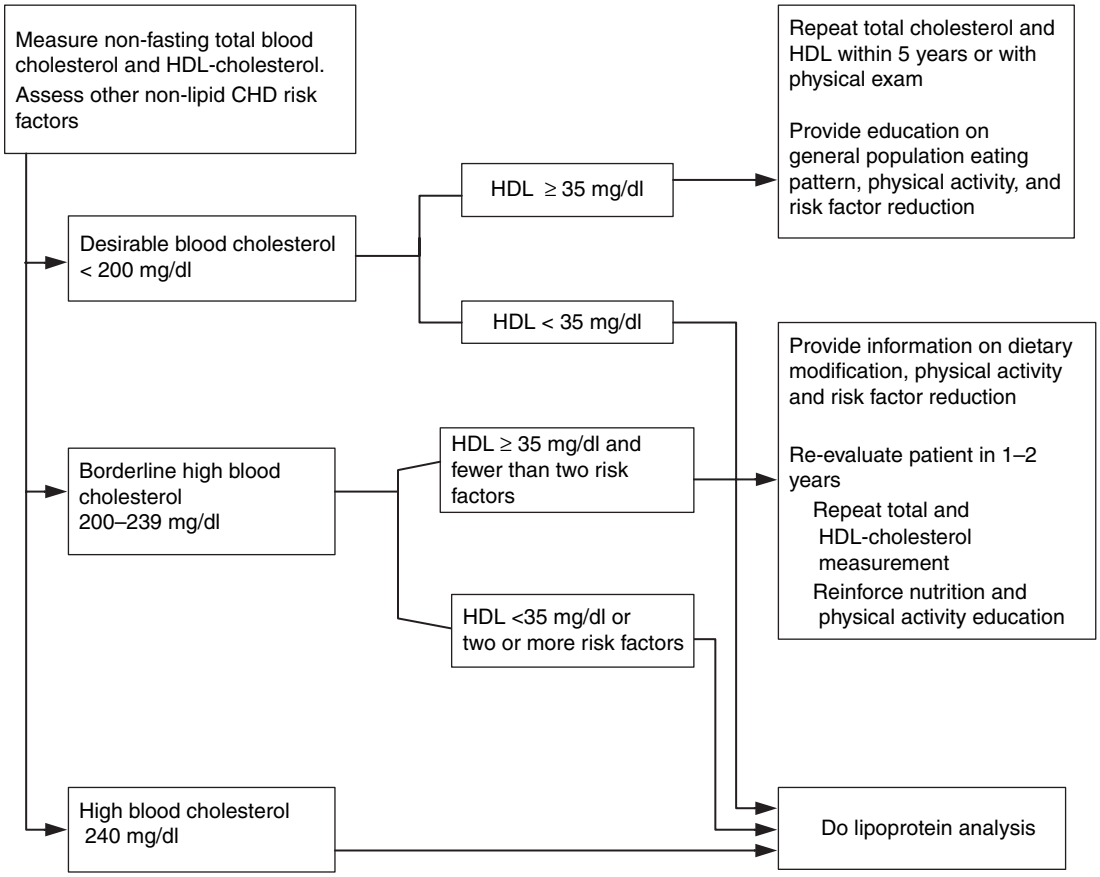
Low HDL-cholesterol (<3.5 mg/dl)

Evidence shows that for every 1-mg/dl decrease in HDL-C, the risk of CHD is increased by 2–3%. In the Framingham study, a 10-mg/dl decrease in HDL-C correlated to a 50% increase in coronary risk among women

If HDL-C is ≥ 60 mg/dl, subtract one risk factor

Indicated as an adjunct to diet to reduce elevated total cholesterol (TC), LDL-C, apoB and TG levels in patients with primary hypercholesterolemia (heterozygous familial and non-familial) and mixed dyslipidemia

Primary prevention in adults without evidence of CHD: initial classification based on total cholesterol and HDL-cholesterol



CHD risk factors

Positive

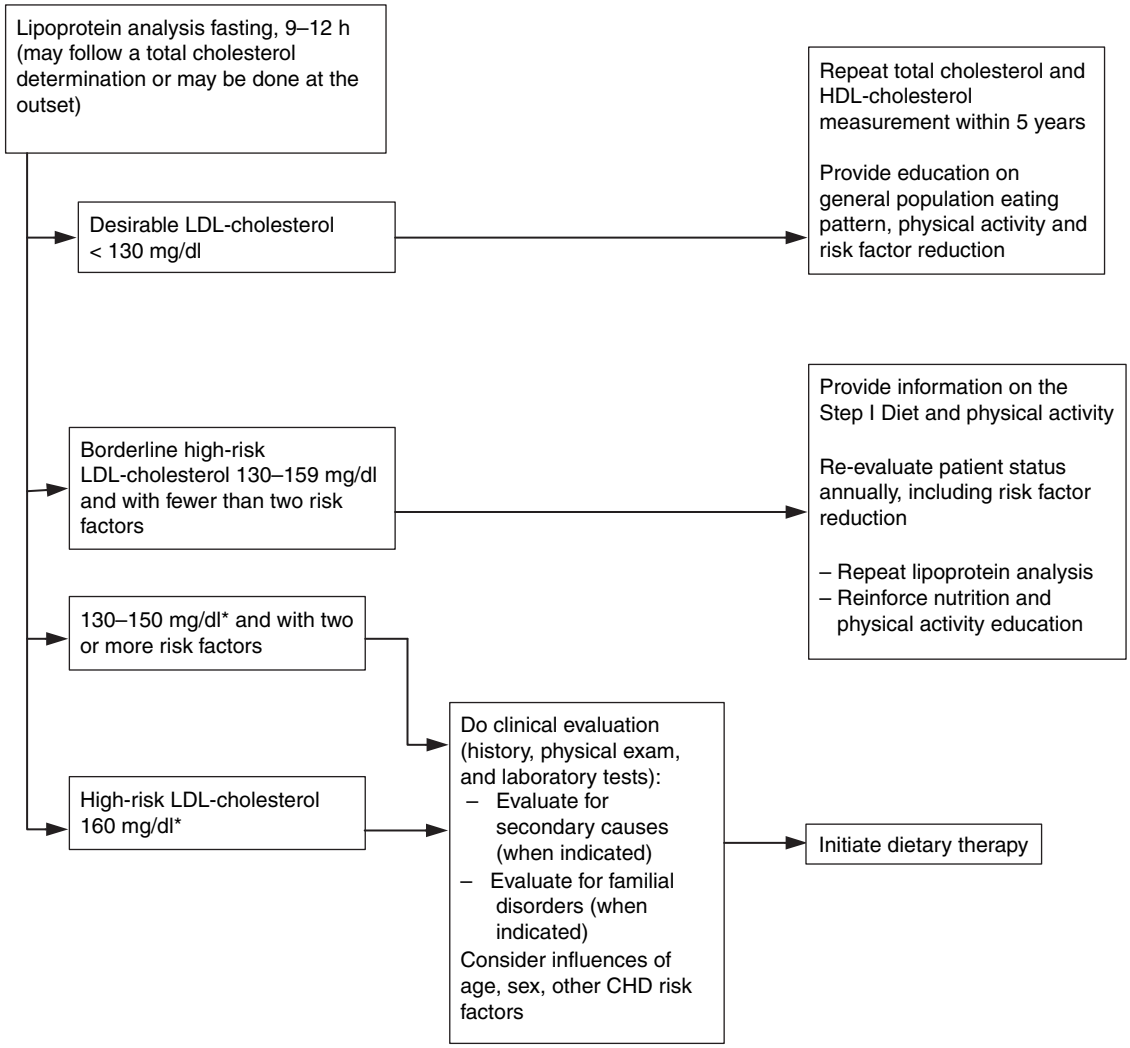
Age: Male ≥ 45 years
Female 55 years or premature menopause without estrogen replacement therapy

Family history of premature CHD
Smoking
Hypertension
HDL-cholesterol <35 mg/dl
Diabetes

Negative

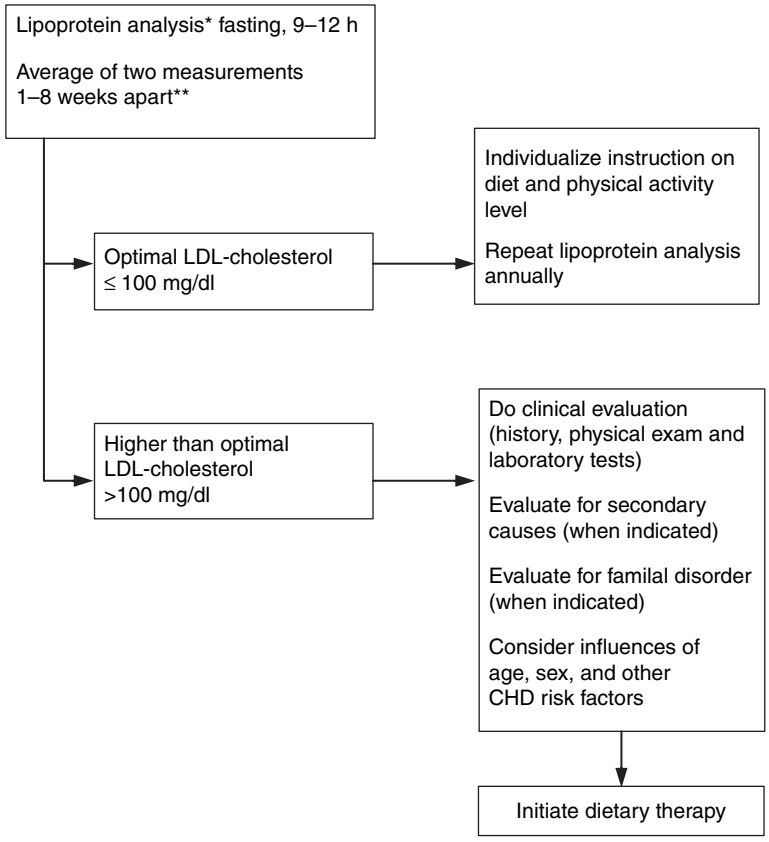
HDL-cholesterol ≥ 60 mg/dl

Primary prevention in adults without evidence of CHD: subsequent classification based on LDL-cholesterol



*On the basis of the average of two determinations. If the first two LDL-cholesterol tests differ by more than 30 mg/dl, a third test should be obtained within 1–8 weeks and the average value of three tests used

Secondary prevention in adults with evidence of CHD: classification based on LDL-cholesterol



* Lipoprotein analysis should be performed when the patient is not in the recovery phase from an acute coronary medical event that would lower their usual LDL-cholesterol level

** If the first two LDL-cholesterol tests differ by more than 30 mg/dl, a third test should be obtained within 1–8 weeks and the average value of the three tests used

CHORANGIOSIS

Poorly defined	LPF 10 villi each with 10 or > vascular channels in Non-infarcted and non-ischemic zones of at least Different placental areas. Not congestion	10 or > 3
Not common	Among 1350 placentas	5.5%
Ominous connotation	Associated with high frequency in stillbirths and many perinatal circumstances that suggest long-standing hypoxia. More commonly observed in the placentas of babies who develop cerebral palsy	

CHORIOAMNIONITIS

See Amnionitis

CHORIONIC VILLUS SAMPLING (CVS)

Can be performed	(1) Transcervical, transabdominal or transvaginal (2) Relatively safe at 10–12 weeks' gestation	
CANNOT be performed	(1) < 10 weeks' gestation (rather DO NOT perform prior to 10 weeks) (2) For diagnosis of NTD (neural tube defect) or fragile X	
Determines	Chromosomal, enzymatic and DNA status of fetus	
Requires	(1) Genetic counseling (2) Experienced operator (3) Experienced lab (in processing villi specimens and interpreting the results)	
Counsel patient about	(1) Increased risk of transverse digital defects (2) Association with oromandibular-limb hypogenesis syndrome (3) Increase incidence of fetal losses more so than amnio	1 in 300
Greatest risk	Damage as result of placental bleeding is especially great Transcervical CVS may increase the risk of pregnancy loss when the placenta is near the cervix. Limb reductions are secondary to hypovolemia and ischemia Oromandibular hypogenesis is associated with severe transverse limb reductions (1/200 000 live births) almost exclusively occur (*Ob Prolog* 4th edn. Washington, DC: ACOG)	< 9 weeks < 9 weeks

CHRONIC PELVIC PAIN

What is the estimated incidence of CPP in women of reproductive age? 15%

What is the *definition* of CPP? Pelvic pain that lasts longer than 6 months

What is the chance that the cause of CPP is endometriosis once anatomic, GI, and genitourinary (PUF or IC) causes are ruled out? 80%

Diagnosis

(A) Have patients bring a written pattern of symptoms

(B) Have patients prepare monthly symptom calendars, illness progression timelines, and temperature charts like ovulation and bring them to office

(C) Encourage patients to be honest about their symptoms and to not feel shy about mentioning painful intercourse or problems with bowel movements or urination

(D) Establish trust. It may be necessary to ask difficult questions about domestic violence, physical or sexual abuse, or psychological conditions that can be fueled by chronic pain

(E) Ask about possible complaints of dysmenorrheal, dyspareunia, heavy or irregular bleeding, infertility, painful defecation or urination, lower back pain, or pain that radiates down one or both legs – particularly during

menstrual periods. (Endometriotic pain can be either cyclic or noncyclic)

(F) *Differential diagnosis*
  (1) Genitourinary – ruptured ovarian cyst, ectopic pregnancy, IC/ painful bladder syndrome, infarcted leiomyoma, symptomatic adenexal cysts, adenomyosis, primary dysmenorrheal, urethral syndrome, recurrent cystitis, urolithiasis
  (2) Gastrointestinal – appendicitis, IBS (irritable bowel syndrome), celiac disease, inflammatory bowel disease
  (3) Neurologic: central or peripheral sensitization (persistent pain following PID or infectious colitis), postoperative abdominal or vaginal wall neuromas, pudendal neuralgia, symptomatic intra-abdominal adhesions
  (4) Musculoskeletal disorders: trigger points, pelvic floor pain syndromes, pelvic girdle dysfunction, symphyseal separation, sacroiliac joint dysfunction
  (5) Cognitive–psychological issues: somatization, catastrophizing and/or domestic violence
  (6) Immunologic: endometriosis, pelvic congestion syndrome

Workup: If the patient has suffered CPP of at least 3–6 months' duration, and has been unresponsive to a trial of nonsteroidal anti-inflammatory drugs (NSAIDs) and/or oral contraceptives, a diagnosis of endometriosis should be suspected

Physical: Thorough rectal examination and pelvic examination of the uterus, ovaries, fallopian tubes, and cervix are essential. If possible, the exam should be performed during early menses when endometrial lesions are likely to be at their largest and most tender. During the rectal exam, test for focal tenderness at the uterosacral and cardinal ligaments and rectovaginal septum. Focal tenderness is associated with a 97% chance that a lesion exists in the area that will be visible during laparoscopy and a 66% chance that the lesion is related to endometriosis. Look for adnexal and uterine tenderness, retroflection of the uterus, limited uterine mobility, pelvic masses, and uterosacral ligaments that may be indurated or nodular. The rectovaginal examination should focus on uterosacral, cul-de-sac, and septal nodules.

*Carnett's test – to differentiate abdominal wall pain from deeper visceral pain, have the patient lie in supine position with her legs flexed at the knees. Have her perform a modified abdominal crunch, engaging the rectus abdominis while coming 2–4 inches off the table. Comparison of pain with and without contraction of these muscles may help locate the source of the pain, as, with the muscles engaged, the viscera are shielded from an examiner's hand. Women whose pain diminishes with the abdominal wall engaged during Carnett's test are likely to have a visceral or intra-abdominal cause responsible for their pelvic pain

Imaging studies: Pelvic ultrasound can detect ovarian endometriomas and, when performed transrectally, has been able to diagnose rectovaginal endometriosis. Neither ultrasound nor MRI can detect peritoneal endometrial implants (Takahashi K, Okada M, Okada S, *et al*. Studies on the detection of small endometrial implants by MRI using a fat saturation technique. *Gynecol Obstet Invest* 1996; 41: 203–6*).* If ultrasound shows any abnormality, a laparoscopy should be recommended

Laparoscopy: While some patients may insist on laparoscopic confirmation, the procedure provides a relatively definitive diagnosis rate of 43–45%. (Winkel C. Evaluation and management of women with endometriosis. *Obstet Gynecol* 2003; 102: 397–408 and Walter AJ, Hentz JG, Magtibay PM, *et al*. Endometriosis: Correlation between histologic and visual findings at laparoscopy. *Am J Obstet Gynecol* 2001; 184:1407–13.) The patient should be informed that established practice today is to treat the condition empirically

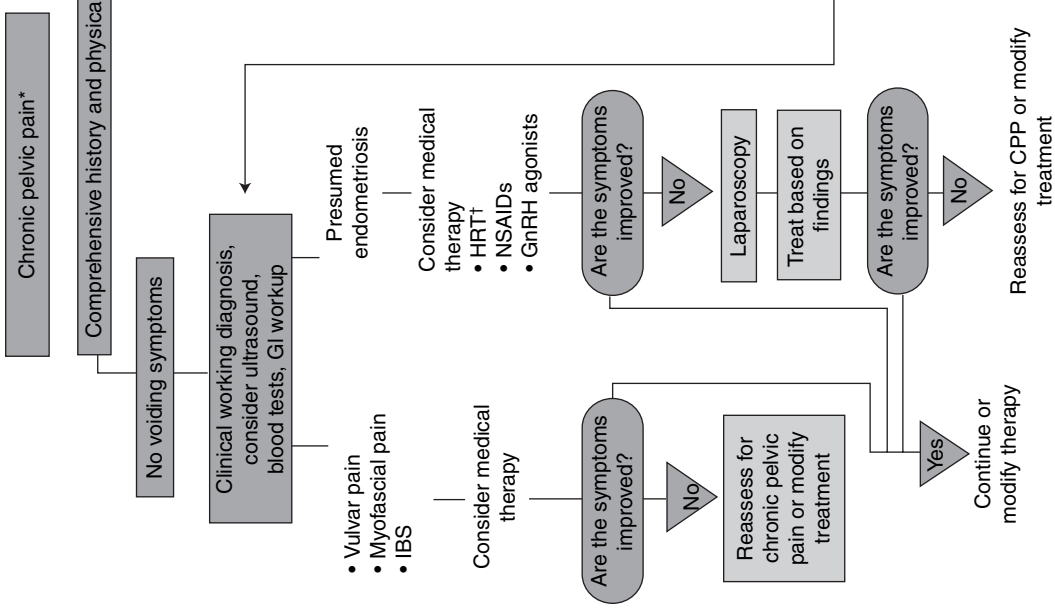
without surgical diagnostic confirmation due to its limitations. (American College of Obstetricians and Gynecologists Commettee on Practice Bulletins–Gynecology. ACOG Practice Bulletin No. 51: chronic pelvic pain. *Obstet Gynecol* 2004; 103:589–605). In addition, successful diagnosis is closely linked to surgical expertise. In one study (Howard FM. The role of laparoscopy in chronic pelvic pain; promises and pitfalls. *Obstet Gynecol Surv* 1993; 48:357–87) endometriosis was detected in only 28% of patients, whereas experienced laparoscopists found the condition in 70% of their cases. (Koninckx PR, Meuleman C, Demeyere S, *et al*. Suggestive evidence that pelvic endometriosis is a progressive disease, whereas deeply infliltrating endometriosis is associated with pelvic pain. *Fertil Steril* 1991; 55:759–65.)
Treatment: A finding of no focal tenderness in a patient with CPP suggests that the disease is in its early stages and infertility is not yet an issue. In this case, it is appropriate to inform the patient that it is safe and effective to empirically treat her for endometriosis even in the absence of surgical confirmation. Hysterectomy is considered the only cure for endometriosis; however, it is clearly a last resort. Treatment generally progresses from simple pain relievers, to oral contraceptives, to medications that mimic pregnancy (GnRH agonists, i.e., Lupron, Zolodex, Synarel), continuous androgens (Danazol), or continuous progesterones (DMPA). Add-back therapy such as norethindrone acetate 5 mg daily can reduce the hypoestrogenic side effects of GnRH agonists. These treatments can also be used to inhibit recurrence following laparoscopic surgery to remove growths and lesions. Progestins are effective for a variety of pelvic pain syndromes, including pelvic congestion syndrome, and endometriosis-related pain. Short-acting agents like norethindrone can reduce severe menstrual pain. Many patients who do not respond to GnRH agonists have underlying neuromuscular dysfunction that responds to physical therapy or agents used for neurologic pain (tricyclic antidepressants such as nortryptiline or imipramine 10–25 mg at night increased every 4–7 days by same dose to 100–150 mg at night and antiepileptics such as gabapentin 100–300 mg at night tapered every 4–7 days up to 900–1200 mg t.i.d.). Be familiar with possible side effects of these drugs. Research suggests that IBS symptoms improve with either cognitive behavioral therapy or tricyclic antidepressants

Perineal pain syndromes, classically described as *pudendal neuralgia,* may respond to physical manipulation and strengthening exercises designed to relieve tension on peripheral pelvic nerves

Local areas of abdominal wall allodynia, particularly in an old abdominal incision scar, may respond dramatically to local anesthetic injections

What percentage of chronic pain patients have sleep disorders? 75%
These sleep disorders stem from their chronic disease. Targeting this sleep deprivation may be critical to restoring a normal quality of life, and in turn, appropriate coping with a chronic pain condition

Algorithm for management of chronic pelvic pain



CIRCUMCISION

Types (male/infant)	(1) Gomco (2) Mogen (3) Plasti-bell
Pain control	The Mogen clamp technique appears to be associated with less crying and grimacing than the use of the Gomco clamp and the same also seems to be true in comparing the sucrose pacifier with the water pacifier (Kaufman GE, Cimo S, Miller LW, Blass EM. An evaluation of the effects of sucrose on neonatal pain with two commonly used circumcision methods. *Am J Obstet Gynecol* 2002;186:564–8)
Risk of cervical cancer	The risk of cervical cancer appears diminished in a woman whose sexual partner has been circumcised (Castellsague X, Bosch FX, Munoz N, *et al*. Male circumcision, penile HPV infection and cervical cancer in female partners. *N Engl J Med* 2002;346:1105–12)
Other risks	Clinical studies also indicate a *reduced* risk in circumcised males of UTIs, penile cancer, penile inflammation, and transmission of some sexually transmitted infectious disease
	UTI rate (circumcised vs uncircumcised)= 1.9 vs 7.0 per 1000 boys
	Despite the increased risks in uncircumcised boys, vocal groups against circumcision complain of the risk of pain, bleeding, local infection and the possibility of long-term emotional harm along with the inability of the newborn to give consent
	Percent of circumcision in U.S. from 1997 to 2000 61%
	Research is being conducted to investigate whether or not circumcision reduces the ability of HIV-infected men to transmit the virus. It is thought that the procedure may reduce the incidence
FGM	Female genital mutilation is discouraged by WHO and other agencies. The highest known prevalence is in Africa. The harmful effects of FGM include hemorrhage, difficult labor/childbirth, genital tears, infections and scar/keloid formation
Types to be familiar with	Type 1 – partial or total excision of the clitoris
	Type 2 – excision of the clitoris and labia minora
	Type 3 – excision of part or all of the external genitalia and stitching/narrowing of the vaginal opening (infibulation)
	Type 4 – the unclassified type and refers to any other mutilation performed on the external genitalia such as gishiri cut or piercing of any part of the external genitalia

CIRCUMVALLATE PLACENTA

	Incidence 1–2%

CLITOROMEGALY

	Normal clitoris of newborn often appears large, so examine in supine position with thighs flexed against abdomen
Clitoral index	Normal ≤ 6 mm^2 Clitoromegaly > 6 mm^2
Possible causes	CAH – 21-hydroxylase deficiency (an autosomal recessive trait) 95% Has three forms: (1) Simple virilizing (2) Salt-wasting form (severe) (3) Non-classic or late-onset form (virilization) True hermaphroditism Teratogenic agents ingested during pregnancy Maternal androgen-secreting tumor
Diagnosis	Check for elevated urinary 17-ketosteroids, plasma DHEA, 17-OHP
Treatment	Treat salt-wasting form of CAH – corticosteroids, mineralocorticoids and NaCl

CLOMIPHENE

Treats oligo-ovulation. How does it work? It is a SERM of the triphenylethylene group. It binds to estrogen receptors as a strong antiestrogen and increases LH because the brain receptors read there is too little estrogen. It is also non-steroidal, crosses all cell membranes and affects cervical mucus adversely. (Raloxifene is a cousin but in the benzathiophene family of SERMs)

Clomid® or Serophene® is an estrogen-receptor antagonist

Induces ovulation in what % of patients taking it? 70–80%

Pregnancy rate with Clomid is 40%

Incidence of twins with Clomid is 5–10%

DHEA-S levels should be drawn if no ovulation with Clomid dose of over 150 mg

Follicle diameter with Clomid treatment should be 20 mm or >

Side-effects: Hot flushes, headaches and nausea, mood alterations, visual changes

Works better (Clomid in combination):

(1) If patient's BMI is optimized (preferably <27 kg/m_2)
(2) If there is insulin resistance, combine with insulin sensitizers such as metformin 500 mg q. daily × 1 week, then b.i.d. × 1 week, then t.i.d. × 1 week or 850 mg b.i.d. for better compliance
(3) If DHEA-S level is > 2 μg /ml, give dexamethasone 0.5 mg daily on cycle days 5 through 9 or days 3–7
(4) If DHEA-S level is < 2 μg /ml, consider 2 months of OC therapy followed by Clomid therapy

COITAL CEPHALALGIA

Headache that occurs during or soon after sexual intercourse

Benign coital cephalalgia

Types:

(1) Muscle contraction type
(2) Vascular type
(3) Low CSF pressure type

Most are vascular types. Rule out subarachnoid hemorrhage and/or aneurysm with CT possible LP, arteriograms p.r.n.

Treatment: Propranolol 80 mg LA capsules 120 mg LA caps p.r.n.
Bellergal®-SR

Caution: Decreased libido or depression

COLON CANCER

Occurs in what % patients with no known risk 75%

Digital rectal exam on females should be performed after the age of 50

Fecal occult blood testing after the age of 50 reduces colon cancer by 25%

For 3 days prior to guaiac testing – avoid aspirin > 325 mg/day
NSAIDs and vitamin C
Red meat, poultry, fish, raw vegetables

If test is + for blood then do Full colonoscopy or
Barium enema with flexible sigmoidoscopy

Sigmoidoscopy every 3–5 years after the age of 50 reduces colon cancer by 30%

Percent of patients with colorectal cancer who will have relapse 50%

Second cause of cancer death

Most common symptom Bleeding

Other symptoms:

Early symptoms
Change in bowel habits
Constipation/diarrhea
Mucus (sometimes mixed with blood)
Tenesmus (ineffectual attempt to defecate often associated with painful spasms of anus)

Late symptoms
Low-back and rectal pain
Dyspepsia
Flatulent distention
Borborygmi
Palpable abdominal mass
Weight loss or weakness

COLPOSCOPY

	• GREEN FILTER and white light	Always use both
	To rule out invasive cancer	Biopsy
Keratosis	White epithelium prior to application of 3–5% acetic acid. HPV is most common cause. Other causes are keratinizing CIN or cancer, chronic trauma (diaphragms, tampons, pessaries), radiorx	
Aceto-white epithelium	Turns white after application of 3–5% acetic acid. Dysplastic cells most affected (large nuclei with increase protein that coagulates)	
Punctation	Dilated capillaries terminating on surface as dots – CIN	
Mosaicism	Terminating capillaries around blocks of A–W epithelium (tile) – CIN	
Atypical vessels	Often associated with invasive cancer. Usually postcoital bleeding	
	CIN I regression	60–80%
	CIN III regression	30%
	White lesions with vessels on top indicate what until proven otherwise?	CIS or microinvasion
	Do not forget to examine vulva and vagina along with the cervix during colposcopy!!	

CONDOMS

Adolescent use rising
Highest rates of GC and *Chlamydia* is in 15–19-year-old age group
Condom use is not consistent
Teens more likely to use condoms if use of condom discussed with clinician

CONFIDENTIALITY

Violation may be necessary if

(1) High probability of harm to third party
(2) Potential harm is a serious one
(3) Information can be used to prevent harm
(4) Greater good will result from breaking confidentiality than from maintaining it

CONDYLOMA ACUMINATA

Incubation	Long	1–8 months
Anatomic distribution	Cervix	70%
	Vulva	25%
	Anus	20%
	Vagina	10%
Predisposed	Diabetes, pregnancy, local trauma, immunocompromised	
Causation	HPV	DNA virus, most common viral STD
	Highly contagious	25–65%
	> 70 subtypes	21 subtypes involved in genital infections
		#16 and #18 are associated with pre-malignant and malignant lesions
		#6 and #11 are associated with benign lesions
Diagnosis	Koilocyte is characteristic cell seen on Pap smear	
	Koilocytosis is associated with atypia and dysplasia	
	Perinuclear halo is diagnostic of koilocyte	
	Colpo if koilocytosis is present	
Treatment	< 2–3 cm	85% TCA, condylox, podophyllin or Aldara
	> 2–3 cm	Electrocautery, cryo or laser
Other subtypes	16, 18, 31, 35, 39, 45, 51, 53, 56, 58	

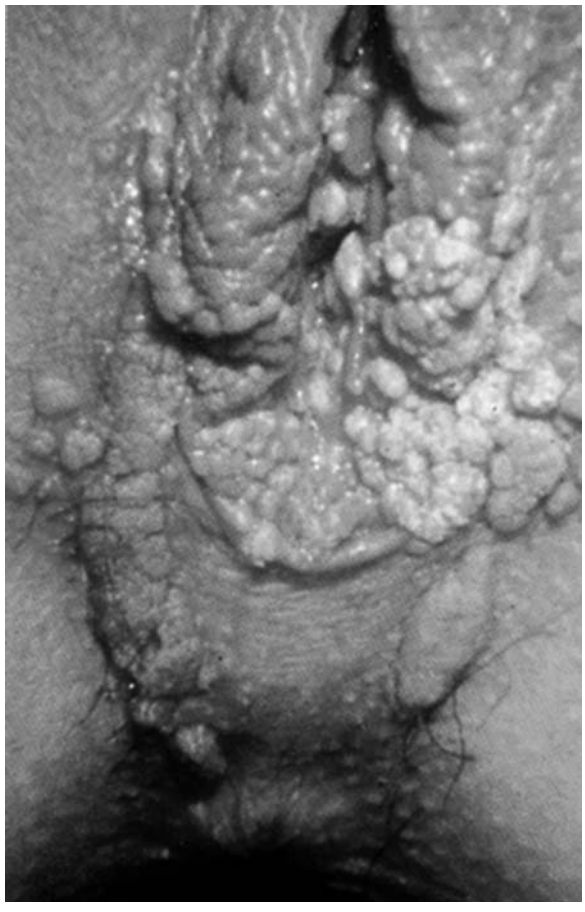


Figure 3 Human papillomavirus causing condyloma acuminata

CONFINED PLACENTAL MOSAICISM (CPM)

Found in this % of CVS specimens	1–2%
The suspected mechanism is that in CPM there is rampant growth of the placenta and the fetus increases the probability of random errors in cell replication	

CONIZATION

Indications

(1) Intraepithelial lesion or microinvasive cancer is present in ECC
(2) Cytology abnormality not consistent with tissue diagnosis
(3) Entire transformational zone is not visible
(4) Microinvasive cancer is diagnosed by directed biopsy
(5) Cytologic or biopsy evidence of premalignant or malignant *glandular* epithelium is detected
(6) No lesion is visible colposcopically

CONTRACEPTION

How many pregnancies are unintended?	2/3
For 'morning after' pill and patients on antiepileptic drugs use	50 μg
For non-contraceptive benefits and contraception use	30–35 μg

Benefits of oral contraceptives

Skin	Improvement of acne	Triphasic norgestimate
	Increased oiliness	Levonorgestrel and norgestrel
BMD (bone mineral density)	+ effect with OCPs and maintaining BMD	
	If no bleeding – no estrogen (anorexia nervosa, exercise-induced amenorrhea, gonadal dysgenesis, early oophorectomy, premature ovarian failure, chemo/radiation, hyperprolactinemia)	

Calcium supplementation especially in teenagers and women in their twenties

Protective effects

Helps prevent ovarian cysts, benign breast disease, including fibrocystic changes and fibroadenomas

Helps prevent pain associated with endometriosis, menorrhagia, polycystic ovary syndrome, and pelvic inflammatory disease

Protection against ovarian, endometrial, and possibly colorectal cancer

Endometrial cancer

Duration of use important. The longer the use, the greater is the reduction in risk of cancer

Ovarian cancer

OCPs prevent ovulation and decrease risk of ovarian cancer by 40–80%
The longer duration, the better. Protection continues after OCPs are discontinued for 20 years

Smokers

Do not give estrogen in smokers > 35! Estrogen increases thromboembolic episodes, especially in women over 35 years old.

Perimenopause

Maintain OCPs if non-smoker to gain benefits

Percent of pregnancies per patient use

Method	*%*
Postcoital douche	80
Rhythm	40
Condom	15–25
Condom and spermicide	5–15
OCPs (oral contraceptive pills)	5–15
IUDs	3–10

Concerns prior to starting oral contraceptives

Suggested screening examination

Blood pressure measurement
Breast, abdominal and pelvic examination
Pap test
Complete blood count
Urinalysis
In case of family history of vascular disease: lipid panel
In case of family history of diabetes: 2-h postprandial blood glucose test; if elevated, perform glucose tolerance test
In case of patient history of liver disease: liver panel

Contraindications and precautions to the use of oral contraceptives

Contraindications

Oral contraceptives should not be used by women who currently have the following conditions:

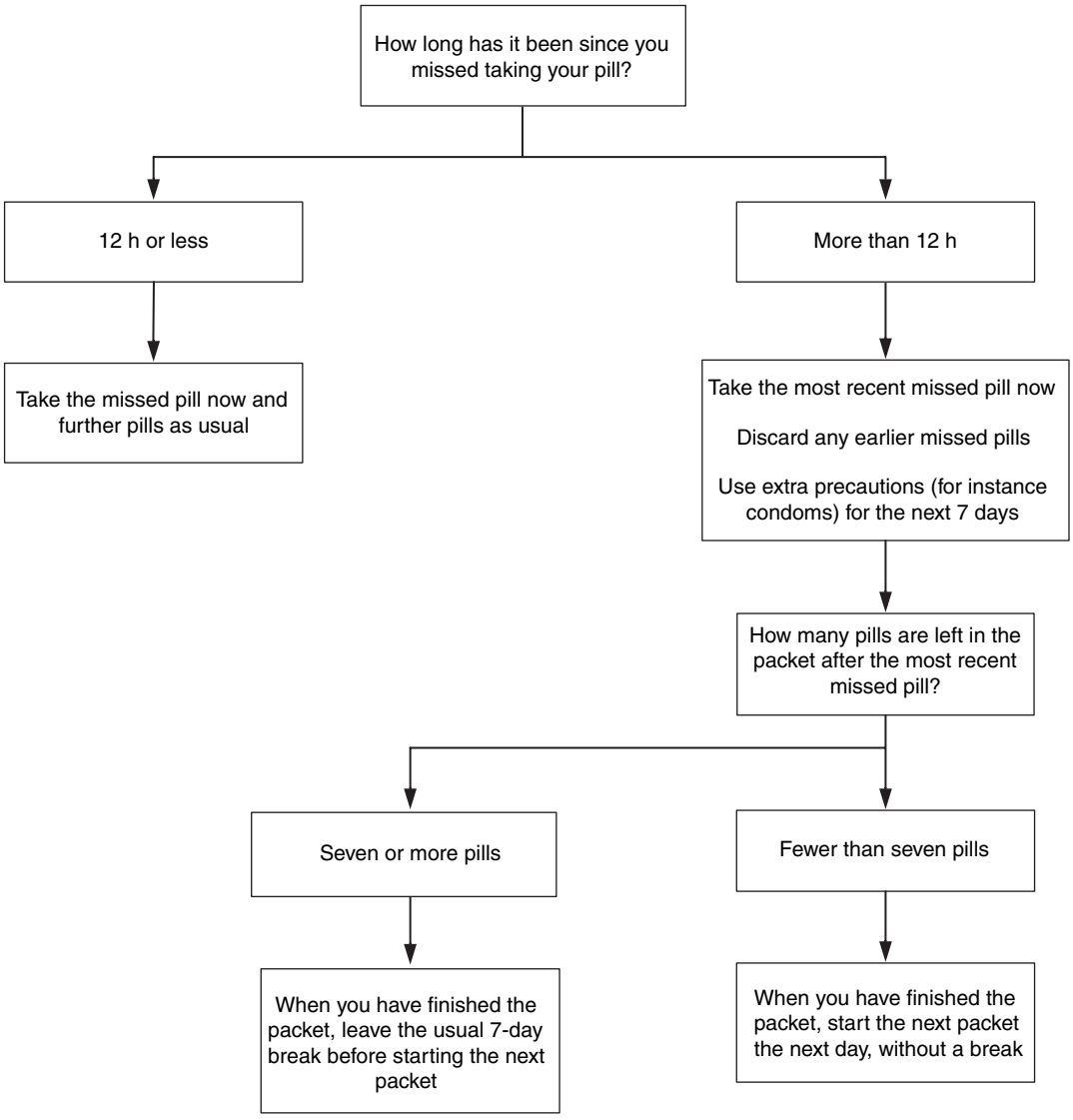
- Thrombophlebitis or thromboembolic disorders
- A history of deep vein thrombophlebitis or thromboembolic disorders
- Cerebral vascular or coronary artery disease
- Known or suspected carcinoma of the breast
- Carcinoma of the endometrium or other known or suspected estrogen-dependent neoplasia
- Undiagnosed abnormal genital bleeding
- Cholestatic jaundice of pregnancy or jaundice with prior OC use
- Hepatic adenomas or carcinomas
- Known or suspected pregnancy

Precautions

Women with the following conditions who take oral contraceptives should be monitored with particular care:

- Breast nodules or a strong family history of breast cancer
- Hyperlipidemia
- Impaired liver function
- Conditions that may be aggravated by fluid retention
- History of depression
- Visual changes or changes in lens tolerance in a woman with contact lenses (should be assessed by an ophthalmologist)

Missed pills – preventing pregnancy in women who miss one or more oral contraceptive



Oral contraceptive types and dosages

Low-dose monophasics

Alesse (Wyeth-Ayerst) 21 or 28 day
- 0.1 mg levonorgestrel
- 0.02 mg ethinylestradiol

Brevicon (Searle) 21 or 28 day
- 0.5 mg norethindrone
- 0.035 mg ethinylestradiol

Demulen 1/35 (Searle) 21 or 28 day
- 1 mg ethynodiol diacetate
- 35 µg ethinylestradiol

Desogen (Organon) 28 day
- 0.15 mg desogestrel
- 0.03 mg ethinylestradiol

Levlen (Berlex) 21 or 28 day
- 0.15 mg levonorgestrel
- 0.03 mg ethinylestradiol

Loestrin 1/20 (Parke-Davis) 21 day
- 1 mg norethindrone acetate
- 20 µg ethinylestradiol

Loestrin 1.5/30 (Parke-Davis) 21 day
- 1.5 mg norethindrone acetate
- 30 µg ethinylestradiol

Loestrin Fe 1/20 (Parke-Davis) 28 day
- 1 mg norethindrone acetate
- 20 µg ethinylestradiol
- 7 pills 75 mg ferrous fumarate

Loestrin Fe 1.5/30 (Parke-Davis) 28 day
- 1.5 mg norethindrone acetate
- 30 µg ethinylestradiol
- 7 pills 75 mg ferrous fumarate

Lo-Ovral (Wyeth-Ayerst) 21 or 28 day
- 0.3 mg norgestrel
- 0.03 mg ethinylestradiol

Mircette (Organon) 28 day
- 20 µg ethinylestradiol
- 150 µg desogestrel (days 1–21)
- Placebo (days 22–23)
- 10 µg ethinylestradiol (days 24–28)

Modicon (Ortho) 21 or 28 day
- 0.5 mg norethindrone
- 0.035 mg ethinylestradiol

Nelova 1/35E (Warner-Chilcott)
- 1 mg norethindrone
- 35 µg ethinylestradiol

Nelova 0.5/35E (Warner-Chilcott)
- 0.5 mg norethindrone
- 35 µg ethinylestradiol

Nordette (Wyeth-Ayerst) 21 or 28 day
- 0.15 mg levonorgestrel
- 0.03 mg ethinylestradiol

Norethin 1/35E (Roberts) 28 day
- 1 mg norethindrone
- 35 µg ethinylestradiol

Norinyl 1+35 (Searle) 21 or 28 day
- 1 mg norethindrone
- 0.035 mg ethinylestradiol

Ortho-Cept (Ortho) 21 or 28 day
- 0.15 mg desogestrel
- 0.03 mg ethinylestradiol

Ortho-Cyclen (Ortho) 21 or 28 day
- 0.250 mg norgestimate
- 0.035 mg ethinylestradiol

Ortho-Novum 1/35 (Ortho) 21 or 28 day
- 1 mg norethindrone
- 0.035 mg ethinylestradiol

Ovcon 35 (Bristol-Myers Squibb) 21 or 28 day
- 0.4 mg norethindrone
- 0.035 mg ethinylestradiol

Yasmin (Berlex) 28 day
- 30 µg ethinylestradiol
- 3 µg drospirenone

Yaz (Berlex) 24/4 – day dosing.
- 0.02 mg ethinylestradiol
- 3 mg drospirenone

Triphasics

Cyclessa (Organon) 28 day
- 7 days: 0.100 mg desogestrel, 0.025 mg ethinylestradiol
- 7 days: 0.125 mg desogestrel, 0.025 mg ethinylestradiol
- 7 days: 0.150 mg desogestrel, 0.025 mg ethinylestradiol

Ortho-Novum 7/7/7 (Ortho) 21 or 28 day
- 7 days: 0.5 mg norethindrone, 0.035 mg ethinylestradiol
- 7 days: 0.75 mg norethindrone, 0.035 mg ethinylestradiol
- 7 days: 1 mg norethindrone, 0.035 mg ethinylestradiol

Tri-Levlen (Berlex) 21 or 28 day
- 6 days: 0.050 mg levonorgestrel, 0.030 mg ethinylestradiol
- 5 days: 0.075 mg levonorgestrel, 0.040 mg ethinylestradiol
- 10 days: 0.125 mg levonorgestrel, 0.030 mg ethinylestradiol

Tri-Cyclen (Ortho) 21 or 28 day
- 7 days: 0.180 mg norgestimate, 0.35 mg ethinylestradiol
- 7 days: 0.215 mg norgestimate, 0.035 mg ethinylestradiol
- 7 days: 0.250 mg norgestimate, 0.035 mg ethinylestradiol

Tri-Cyclen Lo (Ortho)
- 7 days: 0.180 mg norgestimate, 0.025 mg ethinylestradiol
- 7 days: 0.215 mg norgestimate, 0.025 mg ethinylestradiol
- 7 days: 0.250 mg norgestimate, 0.025 mg ethinylestradiol

Tri-Norinyl (Searle) 21 or 28 day
- 7 days: 0.5 mg norethindrone, 0.035 mg ethinylestradiol
- 9 days: 1 mg norethindrone, 0.035 mg ethinylestradiol
- 5 days: 0.5 mg norethindrone, 0.035 mg ethinylestradiol

Triphasil (Wyeth-Ayerst) 21 or 28 day
- 6 days: 0.050 mg levonorgestrel, 0.030 mg ethinylestradiol
- 5 days: 0.075 mg levonorgestrel, 0.040 mg ethinylestradiol
- 10 days: 0.125 mg levonorgestrel, 0.030 mg ethinylestradiol

Biphasics

Jenest (Organon) 28 day
- 7 days: 0.5 mg norethindrone
  0.035 mg ethinylestradiol
- 14 days: 1 mg norethindrone
  0.035 mg ethinylestradiol

Ortho-Novum 10/11 (Ortho) 21 or 28 day
- 10 days: 0.5 mg norethindrone
  0.035 mg ethinylestradiol
- 11 days: 1 mg norethindrone
  0.035 mg ethinylestradiol

Graduated estrophasics

Estrostep 21 (Warner Chilcott) 21 day
- 5 days: 1 mg norethindrone acetate
  0.02 mg ethinylestradiol
- 7 days: 1 mg norethindrone acetate
  0.03 mg ethinylestradiol
- 9 days: 1 mg norethindrone acetate
  0.035 mg ethinylestradiol

Estrostep FE (Warner Chilcott) 28 day
- 5 days: 1 mg norethindrone acetate
  0.02 mg ethinylestradiol
- 7 days: 1 mg norethindrone acetate
  0.03 mg ethinylestradiol
- 9 days: 1 mg norethindrone acetate
  0.035 mg ethinylestradiol
- 7 days: 75 mg ferrous fumarate

Extended-cycle (Menstruation only, 4 times per year)

Seasonique (Duramed)
- 84 days: 0.15 mg levonorgestrel
  0.03 mg ethinylestradiol
- 7 days of tabs with 0.01 mg ethinylestradiol.

Seasonale (Duramed – considering selling)
- 84 days: 0.15 mg levonorgestrel
  0.03 mg ethinylestradiol
- 7 day break prior to next 3-month cycle.

Progestin-only

Micronor (Ortho) 28 day
- 0.35 mg norethindrone

Nor-QD (Searle) 42 day
- 0.35 mg norethindrone

Ovrette (Wyeth-Ayerst) 28 day
- 0.075 mg norgestrel

Adapted from Caufield KA. Controlling fertility (updates by Dr John Turrentine). In Youngkin EQ, Davis MSD, eds. *Women's Health: A Primary Care Clinical Guide*. Norwalk, Connecticut: Appleton and Lange, 1994:112–14; and *Physician's Desk Reference*, 51st edn. Montvale, NJ: Medical Economics Books, 1997

Patch

There is better compliance with the use of the contraceptive patch than with OCPs, but the contraceptive efficacy and cycle control are similar in both methods. (Audet MC, Moreau M, Koltun WD, *et al*. Evaluation of contraceptive efficacy and cycle control of a transdermal contraceptive patch vs an oral contraceptive: a randomized controlled trial. *JAMA* 2001;285:2347–54). Breast discomfort is slightly higher in the first two cycles with the patch than the pill. Dysmenorrhea was also more frequent with the patch, but the difference was not statistically significant. There may be a slightly increased risk of thromboembolic episodes with the patch compared with the oral contraceptive methods because a patient will be exposed to about 60% more estrogen using the patch (*Ortho-Evra)* than using a typical birth control pill containing 35 μg of estrogen.

Ortho Evra™ (Ortho-McNeil Pharmaceuticals) = 150 μg norelgestromin and 20 μg ethinylestradiol
This patch is worn weekly then discarded with a patch-free interval
99% effective

IUDs

LNG-IUS (Levonorgestrel-Releasing Intrauterine System) →Mirena

Mirena® (contains levonorgestrel) should be replaced every 5 years — 99% effective

Paraguard® CU T380 can be left in for 8–10 years — 98.5% effective

Progestasert® must be replaced yearly

Injections

DepoProvera (150 mg IM every 3 months) — 99% effective
Lunelle® (IM every month) — 99% effective

Rings

NuvaRing® (Organon Inc., West Orange, NJ) — 98% effective
120 μg etonogestrel and 15 μg ethinylestradiol
Wear for 3 weeks, then discard and have 1-week interval free
Efficacy compared to OCs particularly Triphasil®

Barrier methods

Diaphragms — 94% effective when used with spermicide
Cervical caps — 91% effective if never pregnant, but only 74% after parous
Sponge – frequent side-effect is 'vaginitis' in — 15%
Male condoms — 97% effective with perfect use
Female condoms — 95% effective with perfect use
Spermicides
VCF vaginal contraceptive film (thin square of dissolving Nonoxynol-9)
As effective as any barrier spermicide

Implants (Norplant®) LNG

Failure rate in first year	0.2%
Failure rate in fifth year	1.1%
Irregular bleeding	30–50%
Weight gain in some of about	20–25 lb
Use in U.S. women (<1 million)	1%
Six capsules each with levonorgestrel	36 mg
Total levonorgestrel	216 mg
5-year contraception – subtherapeutic within ? days removed?	3
Ovulation after removal resumes ? weeks?	2–4

(Implanon®) ENG

Single rod containing containing — 68 mg of etonogestrel (ENG)
3-year contraception — mixed with ethylene vinyl acetate
Training offered – call 1-877-IMPLANON

Congenital anomalies with contraception

No evidence regarding OCPs, IUD or spermicides

Permanent methods

Tubal sterilization – Methods include types of ligation, excision, falope rings, Hulka clips, laparoscopy and vaginal. These topics are covered in full in Turrentine JE. *Surgical Transcriptions in Obstetrics and Gynecology*, 1st and 2nd edns. Carnforth, UK: Parthenon Publishing, 1996. London: Informa Healthcare, 2006.

Transcervical sterilization – Essure. A microinsertable device and catheter delivery system for minimally invasive transcervical tubal access. This consists of a nickel titanium alloy outer coil and polyethylene terephthalate fibers. The PET fibers are a mesh between the inner and outer coils of the device. The device promotes tissue growth in the fallopian tubes, which over a 3-month period, provides tubal occlusion. Other contraception must be used for 3 months after the procedure is performed

Contraceptive chart

		HOW EFFECTIVE IS THIS METHOD?	HOW MANY OPTIONS ARE AVAILABLE?	HOW OFTEN IS IT USED?	ARE THERE INTERRUPTIONS WITH THIS METHOD?	PREGNANCY AFTER USE CAN OCCUR
HORMONAL CONTRACEPTIVES	**The Patch**	99% effective	There is only 1 contraceptive patch	The Patch is applied once a week for 3 weeks. During Week 4, no patch is used	There are no interruptions with this method	Once stopped, it may take a few cycles before you can become pregnant
	Oral contraceptive (The Pill)	99% effective	There are a variety of pills available in different doses	You should take your pill every day, at approximately the same time each day	There are no interruptions with this method	Once stopped, it may take a few cycles before you can become pregnant
	Contraceptive injections	99% effective	There are 2 options currently available; a monthly injection and an injection that is given every 3 months	You receive an injection either monthly or every 3 months	There are no interruptions with this method	Ovulation may be delayed up to a year
	Progestin-releasing intrauterine device (IUD)	99% effective	There is 1 hormone-releasing IUD currently available	The suggested length of use is 5 years or less	There are no interruptions with this method	Once removed, fertility can return within a year
	Vaginal ring	99% effective	There is only 1 vaginal ring	Each month, the vaginal ring is inserted into the vagina and left in place for 3 weeks. During Week 4, you do not wear the ring	There are no interruptions with this method	Once stopped, it may take a few cycles before you can become pregnant
NON-HORMONAL CONTRACEPTIVES	**Male condom**	97% effective	There are a variety of styles, sizes, colors, materials and textures	A new one must be used every time you have sex	Must be applied when the penis is erect. May cause a slight interruption before sex	Without this device, there is no protection against pregnancy
	Female condom	95% effective	There is 1 female condom currently available	A new one must be used every time you have sex	A female condom can be inserted up to 8 hours before sex	Without this device, there is no protection against pregnancy
	Intrauterine device	99% effective	There is 1 copper-T IUD currently available	Once inserted in the uterus, it can be left in place for up to 10 years	There are no interruptions	Once removed, fertility can return within about 1 month
	Spermicides	94% effective – use with a vaginal barrier increases effectiveness	There are a variety of spermicides available in foams, jellies, creams and vaginal suppositories	Must be used every time you have sex	Must be inserted no more than 1 hour before sex	Without this device, there is no protection against pregnancy
VAGINAL BARRIERS	**Diaphragm**	94% effective	There are a variety of sizes available	Must be used every time you have sex (and fresh spermicide must be applied each time)	The diaphragm can be inserted 6 to 8 hours before sex	Without this device, there is no protection against pregnancy
	Cervical cap	84% effective in women who have had a child (91% in those who have not)	There are a variety of sizes available	Must be used every time you have sex (and spermicide must be applied when inserted)	The cervical cap provides continuous protection for up to 48 hours	Without this device, there is no protection against pregnancy
PERMANENT METHODS	**Surgical sterilization**	Greater than 99% effective	For women, there is a tubal ligation (having your tubes 'tied'); for men, there is a vasectomy	These procedures are permanent and irreversible	There are no interruptions with this method	You will no longer be able to get pregnant

CORD PROLAPSE

Incidence	1/200 or 0.1–0.5%
Percent of cord prolapses associated with breech	50%
Incidence in compound presentations	20%
Found in vagina	45%
Found at introitus	39%
Found along presenting part	11%
Found between legs with breech	4%

Perinatal mortality

2–8% (one source states over 20%)

Predisposing factors

(1) Most frequent causes
  (a) Abnormal presentation
  (b) Fetal hypotension (with abruptio)
  (c) Multiparity
  (d) Multiple gestation
  (e) Prematurity
(2) Less common factors
  (a) Contracted pelvis
  (b) Extended cord length
  (c) Obstetric manipulations
  (d) Polyhydramnios
  (e) Premature rupture of membranes
  (f) Rupture of membranes before engagement (spont. or art.)

Diagnosis

(1) Palpable cord on vaginal exam
(2) Observed cord protruding onto vulva
(3) FHR pattern suggesting cord compression
  (a) Prolonged, severe, variable decelerations
  (b) Bradycardia
  (c) Ultrasound (may diagnose high-risk cases prior to distress)

Treatment

URGENT DELIVERY TO AVOID ASPHYXIA AND DEATH
Preparation for surgery:
(1) Push presenting part cephalad
(2) Knee–chest position or Trendelenburg
(3) Replace cord into uterine cavity and cephalad to presenting part
(4) Fill bladder with 500–700 ml of saline
(5) Give oxygen!!!
(6) Consider giving a tocolytic agent (terbutaline) IV

Management

(1) Place mother in Trendelenburg or knee–chest position
(2) Elevate presenting fetal part
(3) Administer oxygen to mother
(4) Swiftly order preparations for C-section
(5) If preparations are prolonged:
  (a) Distend bladder (500–700 ml NS thru cath)
  (b) Administer a tocolytic agent (terbutaline) IV
  These steps will serve to elevate presenting part and decrease or stop uterine contractions both allowing better perfusion

Comparative modes of delivery (% perinatal mortality)

SVD – 35.5%; LFD – 0%; MFD – 33.3%; VE – 33.3%; assisted breech extraction – 25%; total breech extraction or version and extraction – 10%; total of these – 26.9%, compared to C-section – 3.4%

CORTISOL TEST

Rule out Addison's

'Humpback', decreased K^+, Cl, GTT, eosinophils and WBCs

The test

Give 1 mg Decadron® at 11 pm then draw serum cortisol at 8 am
Normal result should be <5 mg

CRITICAL CARE ESSENTIALS

Wedge and urine decreased with pulse elevated and H&H ok =	increased vol
Wedge increased, urine decreased, lungs X, H&H ok =	Lasix®
Wedge and urine decreased with pulse elevated and H&H decreased =	give blood

CUSHING'S SYNDROME

Symptoms or findings	Obesity	95%
	Moonface and molar rash	95%
	Hypertension	85%
	Glucose intolerance	80%
	Menstrual/sexual dysfunction	75%
	Hirsutism and acne	72%
	Striae	67%
	Weakness	65%
	Osteoporosis	55%
	Easy bruisability	55%
	Depression	80%
	Edema of legs	40%
	Overnight dexamethasone suppression test given at 11 pm and tested at 8 am the following morning is:	
	+ if fails to suppress plasma cortisol under	5 μg /day
	+ if urinary cortisol	> 100 μg/day
	Almost virtually diagnostic of Cushing's in a non-pregnant female if value is	> 250 μg/day

CYSTIC FIBROSIS

	Chronic pulmonary and exocrine pancreatic disease	
	Almost all men with CF have bilateral absence of vas deferens	
	Another disorder – congenital bilateral absence of the vas deferens (CBAVD) is found in 1% of infertile men and in higher % of those presenting with azoospermia. CBAVD is milder form of CF. No fructose in sperm when obstruction of vas is present	
	CF is most common lethal autosomal recessive disease found in people of N. European descent	
	Carrier frequency is about	4–5% or 1/22–25
	Disease frequency is	1/2500
		1/1600 in N. European descent
Clinical manifestations of CF	Meconium ileus	
	Chronic obstructive pulmonary disease leading to bronchiectasis and respiratory failure	
Life expectancy	Patients can be expected to live to age 26 or longer	
	CF gene found on chromosome number	7, locus 31
	A fragment of this gene on chromosome 7 encodes a protein called cystic fibrosis transmembrane conductance regulator (CFTR) that assists in transport of chloride ions to maintain hydration in epithelial-lined lumina	
	If CFTR is dysfunctional, secretions in pulmonary small airways and in the pancreatic ducts become tenacious and obstruct those structures – lung + pancreatic problems	
	OFFER TESTING ONLY TO PATIENTS AND COUPLES WITH FMH OF CF. Get informed consent!	
	Mutations in CF patients @ deletion of three base pair resulting in the loss of phenylalanine residue	
	Amniotic fluid cells or CVS may be used prenatally	
Increased F508	Testing for increased F508 deletion is imperfect – detects only half to two-thirds carrier couples	

How many gene mutations can cause CF? (Most common is increased F508) 400–800
Most labs screen for how many of the most common genes that cause CF? 32
Negative test decreases risk to 1 in 246 or 90%
If husband carrier is unknown, pt with CF has estimated risk of having child with CF of 1/50

Screening

Screening for only one partner; voluntary, informed consent required, education and counseling, quality control of lab and equal access to testing should be available
Population-based screening should NOT be recommended with NEGATIVE FMH

Congenital bilateral absence of vas deferens (CBAVD)

Most men nevertheless have caput epididymis – microsurgical sperm aspiration is possible
Then intracytoplasmic sperm injection (dilution with glycerol 10%)
Genetic basis of disorders requires proper testing and counseling take place and parents-to-be are very informed of genetic risk to offspring

CYSTIC HYGROMA

Congenital malformation of lymphatic system usually seen in nuchal region uncommon. Can be associated with chromosomal anomalies or fetal hydrops

First trimester

Aneuploidies

Second trimester

XO (monosomy) most common

Diagnosis

Ultrasound and physical exam

Prognosis

If nl karyotypes without septations and spontaneously resolves = good
Those with septated lesions – increased risk of abnormal karotype with decrease in survival rate

CYSTOCELE

Rupture of pubovesicle cervical fascia (central defect of endopelvic fascia)
Anterior repair corrects anterior midline vaginal defect. *See* Prolapse (POP)
Repaired by dissection to lateral vaginal wall until defect demonstrated

CYSTS

Features of benign versus malignant

Cystic	Ascites
Unilocular	Solitary thick septa
<10 cm	Papillations
Unilateral	Matted bowel
Regular borders	Irregular borders

Most common cysts or masses found in reproductive age women:

Pelvic mass	Pregnancy
Pelvic neoplasm	Fibroid
Ovarian mass	Functional cysts
Ovarian neoplasm	Dermoid

Ovarian cancer is unlikely to cause sudden pain

In a *premenopausal* woman presumed to have a benign cyst, surgery for pain or failure to resolve should conserve the ovary if at all possible

In *postmenopausal* women, repeat sonograms and observation are justified unless CA-125 is elevated or the cyst's size or complexity increases. When surgery is necessary in postmenopausal women, remove the entire ovary for complete pathologic analysis

DAYS TO REMEMBER

Morula	2–3 days after fertilization
Blastocyst	4–5 days after fertilization
Fertilized ovum reaches uterus in	5–6 days
Implantation	6–7 days
Trophoblastic venous sinuses form	9–11 days
Cardiovascular system begins to form	21 days
Earliest morphological indicator of sex appears	8–9 weeks
Oogenesis begins	11–12 weeks

DEEP VEIN THROMBOSIS

See DVT

DEHYDRATION

Guidelines for the athlete

Effects of dehydration	*What to drink during exercise*
Dehydration can affect an athlete's performance in less than an hour of exercise – sooner if the athlete begins the session dehydrated	If exercise lasts more than 45 min or is intense, a sports drink should be consumed during exercise
Dehydration of just 1–2% of body weight (only 1.5–3 lb for a 150-lb athlete) can negatively influence performance	A 6–8% carbohydrate (CHO) solution maintains optimal carbohydrate metabolism
Dehydration of greater than 3% of body weight increases an athlete's risk of heat illness (heat cramps, exhaustion or stroke)	During events when fluid loss is of primary concern a beverage with less than 7% CHO is recommended
	Fluids with salt (NaCl) are beneficial for increasing thirst and voluntary fluid intake as well as offsetting losses

Recognition of the basic signs of dehydration

Thirst, irritability, fatigue, muscle cramps, loss of performance, vomiting

Recommended guidelines

(1) Before exercise: drink at least 17–20 oz of Gatorade 2–3 h before the activity starts
(2) During exercise: drink 28–40 oz of Gatorade per hour of play (at least 7–10 oz every 10–15 min or amount equal to sweat and urine loss)
(3) After exercise = drink at least 20 oz of Gatorade per pound of weight loss within 2 h to help rehydration

• Gatorade© Thirst Quencher contains a 6% carbohydrate solution (14 g CHO/8 oz)

DELIVERY DESCRIPTION

Include
Infant info: viability, weight, sex, Apgars, presentation, position
Maternal info: episiotomy? extension? repair description, EBL
Anesthesia, laceration of cervix, vagina or vulva
Other: placental and cord description

DEPO-LUPRON

	Leuprolide acetate – GnRH agonist	
Action	Endometriosis treat for	6 months
	Fibroids (uterus shrinks more than fibroid) treat for	3 months
	Initial stimulation followed by suppression of pituitary gonadotropins	
Contraindications	Undiagnosed vaginal bleeding, pregnancy and breastfeeding	
Side-effects	Amenorrhea after two doses	98%
	Bone loss of what % after first 6 months of treatment?	5%
	Flare response after first dose seen in	3 weeks
	(Hot flashes, palpitations, syncope, menopausal symptoms)	
	Anaphylaxis possible – treat with epinephrine 1 : 1000	0.5 cc SC
Birth control	Needed for first	2 months
	Category	X
Dose	IM every month for 3–6 months	3.75 mg

DEPO-PROVERA® (DMPA)

Medroxyprogesterone acetate
150 mg IM every 3 months for contraception Increase dose for other
104 mg subq every 12 to 14 weeks (thigh or abdomen)
(Both IM and subcutaneous DMPA cause decrease in BMD after 1 to 2 years of treatment)

Resumption of ovulation	7–9 months
Mechanism of action	Blocks LH surge Thickens mucus Alters endometrium
Treatment of bleeding	Ibuprofen 800 mg t.i.d. × 5 days or ethinylestradiol 20 μg or Premarin 1.25 mg × 10–21 days

Action	Contraception and others Inhibits gonadotropins – prevents ovulation. Thins endometrium Helps relieve endometriosis-associated pain	
Side-effects	Irregular bleeding, weight changes, breast tenderness, acne, hair loss, galactorrhea, eventual amenorrhea to 55% after 1 year, loss of bone density with long-term therapy	
Birth control	Effective within	24 h
	Category	X
Dose	IM every 3 months	150 mg
	Subq every 12–14 weeks	104 mg
	Resumption of ovulation after DMPA	7–9 months
	What % patients conceive within 1 year?	70%

DEPRESSION

See also Postpartum depression

Symptoms

(1) *Emotions*
Sadness, hopelessness, restlessness, irritability, loss of interest, trouble concentrating, trouble making simple decisions, guilt, or thoughts of death or suicide
(2) *Headaches*
Bothered by headaches that cannot be explained by other conditions
(3) *Sleep*
Sleeping too much, or not enough, sleep problems affecting patient's life
(4) *Fatigue and decreased energy*
(5) *Stomach aches*
(6) *Weight*
Losing or gaining weight recently without trying
(7) *Aches & pains*
(8) *Stress & tension*

Treatment

SSRIs Start with SSRIs. If the patient becomes pregnant, there is a slight risk of persistent PPHN (pulmonary hypertension of the newborn). However, The risk is small (6–12 cases of PPHN per 1000 births, or 0.6–1.2%). On the other hand, if the drugs are discontinued, there is a serious likelihood depression will recur, which poses other fetal and maternal risks. Weigh the risks, and tell the gravida treated with an SSRI that 99% of infants deliver without PPHN

Activating
Citalopram (Celexa), escitalopram (Lexapro), fluoxetine (Prozac or Serafem), or sertraline (Zoloft)

Sedating
Fluvoxamine (Luvox) or paroxetine (Paxil or Paxil-CR)

Other antidepressants

Buproprion	Wellbutrin or Wellbutrin-SR (class B and dopamine-activating)
Mirtazapine	Remeron or Remeron SolTab (complex, sedating)
Venlafaxine	Effexor or Effexor-XR (serotonin, norepinephrine, mildly sedating)
Duloxetine	Cymbalta (duloxetine HCl) is also indicated for the treatment of major depressive disorder (MDD). Immediate switching from an SSRI is well tolerated
EMSAM	Selegilline transdermal system is the first transdermal patch for the treatment of MDD. It is a monoamine oxidase inhibitor (MAOI). Avoid foods high in tyramine (aged cheese and tap beer) to reduce risk of hypertensive crisis especially with 9 mg/24 h patch and 12 mg/24 h patch. Start with 6 mg/24 h patch daily without tyramine dietary restriction then if there is a need for an increase dose, do dietary restriction of tyrosine

Suicide National Hotline 1 800 784-2433

DERMATOLOGIC CONDITIONS COMMON TO OB/GYN

Acne

Treatment

(1) Mild
   (a) Benzoyl peroxide (PanOxyl®, Benzagel® or Desquam X®)
   (b) Cleocin T® solution 30 or 60 ml
   (c) Erythromycin base (Staticin®, 60 ml, Eryderm® 60 ml, T-stat® pads)

(2) Moderate
   (a) Tretinoin (Retin-A®) q. h on dry face or q.o.d.
   Use 0.025% cream or 0.01% gel for fair complexion
   Use 0.05% cream or 0.025% gel for others
   Cream preferred for dry skin; gel for oily skin
   (b) Benzoyl peroxide at different time than Retin-A
   (c) Tazorotene topical gel 0.1% (Tazorac®) Use q. h on clean face

(3) Severe
   (a) Tetracycline 500 mg b.i.d. on empty stomach. Decrease dose to 250–500 mg daily after lesions clear or increase dose after 4–6 weeks to 2 g daily for several weeks if lesions have not subsided
   (b) Erythromycin 1 g daily effective
   (c) Doxycycline hyclate or monohydrate (100–150 mg b.i.d.) The monohydrate causes less GI side-effects but is more expensive
   (d) Minocycline 100 mg b.i.d. (more expensive than TCN or Dox but most effective antibiotic in its class)
   (e) Ampicillin if pregnant

• WARN patients about decreased effectiveness of OCs and dangers of sunlight (especially with estrogens, prednisones, spironolactone)

Oral contraceptive therapies that are approved by FDA for acne:

(1)	Ortho Tri-Cyclen®	35 μg of estrogen
(2)	Estrostep®	20 μg/30 μg/35 μg of ethinylestradiol
(3)	Alesse®	20 μg ethinylestradiol

Rosacea

Diagnosis
Red flush of central face, neck and nose
Rule out malignant carcinoid, lupus; basal cell carcinoma if rhinophyma present
Treatment
Avoid hot food and drinks
Tetracycline 500 mg p.o. b.i.d.
Metrogel 0.75% × 9 weeks

Hidradenitis

Diagnosis
Large cysts and/or abscesses, in axilla, under breasts, groin, buttocks, anogenital region and/or thighs
Treatment
Antibiotics, prednisone, Accutane®, surgery

Melasma

Diagnosis
Brown, macular facial pigmentation – increased with sun exposure (develops in 5–70% pregnancies)
Common with OCs (5–34%)
Treatment
Melanex® 3% or Solaquin forte® 4% b.i.d.
Retin-A cream if not pregnant
Avoid sun or use sun blocks
Chemical peels

Cowden's disease

Diagnosis
Flesh, pink or brown papules at midfacial, perioral, lips and/or ears
Punctate keratoses of the palms and soles
Mutations in the *PTEN* gene on chromosome 10
Breast cancer can occur in up to 30% of women with Cowden's disease
Treatment
Assess for breast cancer (20–30%) – often bilateral
Prophylactic mastectomy advocated
Thyroid cancer is present in 8%

Alopecia

Diagnosis
Drugs, secondary syphilis ('moth-eaten' appearance),
Tinea capitis or androgenetic etiology – androgen tumor?
Treatment
Determine etiology and treat accordingly

Seborrheic keratosis

Diagnosis
Barely elevated small papules
Treatment
Electrodessicate, shave excision or liquid nitrogen

Fungus of toenails or fingernails

Diagnosis
Establish diagnosis with KOH prep
Fax 216 844-1076 for Derm Pak
Treatment
Diflucan® 150 mg weekly
Lamisil® 250 mg daily p.o. continuous (6 weeks for fingernails and 12 weeks for toenails)
Sporanox® (itraconazole) 200 mg/day. Pulse dose with 400 mg/daily for the first weeks of each month for 16 weeks

Psoriasis

Diagnosis
Rich red hue, smooth plaque; genetic 1–3% – can occur at site of trauma
Scaly, red follicular papules merge to form large, bright plaques
Avoid lithium, β-blockers, antimalarials and systemic steroids

Treatment
Betamethasone dipropionate (Diprolene®, Alphatrex®)
Anthra-Derm® 0.1, 0.25, 0.5, 1% ointment 1.5 oz, 42.5 g tubes
Drithocreme® HP 1% cream, 50 g tube
PsoriGel® 7.5% coal tar solution; 1% alcohol gel 4 oz
Topical steroids (pulse dosing – 2 weeks of medication and 1 week of lab only with plastic occlusion very effective) for psoriasis on < 20% of body
For more than 20% of body – consider dermatology referral for UVB/tar, PUVA, methotrexate, hydrea, etretinate, etc.

Lichen sclerosus

Diagnosis
White 'cigarette-paper plaque-like' lesions. Biopsy necessary to rule out squamous carcinoma
Treatment
Clobetasol (Temovate®) 0.05% cream or ointment (30 g) b.i.d. applications for 10–14 days then taper to twice weekly. Monitor these patients closely every 6 months for squamous cancer

Pregnancy-associated rashes

Most common in primigravida

PUPP (Polymorphological Urticarial Papules of Pregnancy)

No risk to mother and infant
Seldom recurs. Usually resolves 2 weeks postpartum
Usually > 28 weeks
Increased with multiple births or increased weight gain
Extreme pruritus starts on abdomen in striae
Treatment
(1) Aveeno baths
(2) Cool compresses
(3) Benadryl 25 mg p.o. q. 4–6 h
(4) Prednisone, phototherapy – deliver baby

Herpes gestationis (pemphigoid gestationalis)

Rare (1 : 50 000 pregnancies)
Risk is unclear to fetus (reports of increased PTD and SGA and transient neonatal lesions and occasionally associated with Graves' disease)
Often recurs. Usually occurs earlier but usually in second or third trimester
C3 complement. Increased HLA-DR3 and HLA-DR4
Not associated with herpes virus despite name. Eruptions usually start periumbilical. Urticarial plaques with tense vesicles or bullae. Has been associated with trophoblastic disease
Treatment
Systemic and topical steroids

Pruritus gravidarum

Most common (1–2%)
Risk is increased in regards to infant mortality and prematurity
Usually recurs
Associated with cholestasis (itching associated with bile acids)
Intense itching during pregnancy (usually more intense on extremities than trunk)

Impetigo herpetiformis

Rare
Risk is increased (systemic symptoms with decreased Ca^{+} and also decreased parathyroid)
Sepsis can occur

DERMOIDS

Most common – neoplastic ovarian lesion in females of reproductive age

Bilateral	15–25%
Malignant (usually squamous)	< 2%
Torsion (most frequent complication)	16%

Treatment of torsion: untwist or cystectomy (pseudoencapsulation)
Avoid spillage (chemical peritonitis)
Struma ovarii (% ovarian teratoma) 2–3%
(*See* Struma ovarii for more details)

DETROL

Tolterodine tartrate – potent antimuscarinic Category C

Indications Overactive bladder, symptoms of DI – frequency and urgency, increased residual urine, decreased detrussor pressure

Contraindications Narrow angle glaucoma, urinary and gas retention

Caution If used with emycins or ketoconazole (cytochrome P450 pathway). Consider decreasing dose

Usual dose 2 mg p.o. b.i.d. or 4 mg LA daily

DIABETES AND PREGNANCY

Background

(1) 2–3% pregnancies affected
(2) 90% of this 2 3% represent GDM
(3) 50% of women who develop GDM will develop overt DM within 20 years
(4) Women with overt DM who conceive have a 10-fold increase in maternal mortality and perinatal mortality of 4%

Classification

A1 Diet-controlled GDM
A2 GDM complicated by insulin use, hypertension, polyhydramnios, macrosomia or prior stillbirth
B Overt; onset > age 20 and duration <10 years
C DM overt; onset age 10–19 or duration 10–19 years
D Juvenile onset or duration of 20 years or more
F Associated with nephropathy
R Associated with retinopathy
T Renal transplant patients

Major malformations

(1) Increased × 4
(2) Risks increased by 30% between 5–9 weeks (embryogenesis)
(3) Spontaneous abortions increased by 35%
(4) Common: CNS, cardiac, renal, retinal
(5) Uncommon: caudal regression syndrome

Goals

Mean:	90–105
Fasting:	60–90
Preprandial:	80–95
Postprandial:	< 120

Screening

(1) HbA_{1C}: Preconception level (normal–similar to non-DM women)
Used to assess anomaly risk and to provide a goal for the woman aspiring to improve her chances of a good outcome
(2) Screen all women over 25 years of age at 24–28 weeks
(3) Screen early in pregnancy (first visit) and 24–28 weeks those women with:
  (a) Family history of DM
  (b) Prior infant with cardiac anomaly
  (c) History of stillbirth
  (d) History of repeated pregnancy loss
  (e) Previous child > 4000 g

Diagnosis

(1) If 1 h 50-g p.o. glucose challenge test is >140 mg/dl
(2) 3 h GTT of 100-g p.o. glucose after 3 days of adequate carb. intake. Two abnormal values are necessary to make diagnosis of GDM

Time	*Glucose level (mg/dl)*	
	WHO	*Carpenter & Coustan*
Fasting	<105	<95
1 h	<190	<180
2 h	<165	<155
3 h	<145	<140

(3) If 1 h 50-g test is greater

Management

(1) Goals: maintain FBS of 60–80 mg/dl; 2 h pp levels of 60–100 mg/dl
(2) Diet: 2200–2400 kcal for women of normal weight
(3) Recommend 20–30 minutes of exercise 3–4 times weekly. If women are willing and able, exercise can improve postprandial blood glucose levels and insulin sensitivity
(4) Insulin: abdominal to achieve consistency and rotation and at perpendicular to skin to prevent intradermal injection rather than SC

NPH alone	*NPH + REG*	
a.m. 2/3	a.m. 2/3 – 2/3 NPH	1/3 REG
p.m. 1/3	p.m. 1/3 – 1/2 NPH (q.h.s.)	1/2 REG (AC)

Surveillance

(1) *Patient diary* (charting of glucose levels, insulin dosage and date/time)
(2) *Type A1*: no amnio; delivery by 40 weeks
(3) *Type A2, B, C*: twice weekly NSTs > 34 weeks; delivery at 38 weeks if glucose levels abnl and PG present
(4) *Type D, F, R*: twice weekly NSTs from 28–30 weeks; delivery at 36 weeks if abnl glucose levels and PG present
(5) *Ultrasound* (fetal anatomy) with echocardiogram (serious consideration) 18–20 weeks

Pre-term labor

(1) $MgSO_4$ or calcium channel blocker. (Avoid terbutaline if possible – tendency to cause hyperglycemia)
(2) Corticosteroids (for lung maturity, but know that hyperglycemia will probably result)

Labor & delivery

(1) Insulin (regular insulin 50 units in 500 ml NS)
Shake well; run out 50 ml waste to ensure absorption of surfaces
Continuous pump rate of 0.5 units/h or > with increments of 0.5–1 unit/h to obtain necessary glucose levels
(2) D5LR
(3) Bedside glucose values every hour with finger stick test strips
(4) Adjust infusion p.r.n. to maintain glucose levels 100 130 mg/dl

Diabetes-in-pregnancy program protocol

Class A and A/B

(1) Glucose determination weekly
(2) Biweekly visits until 34 weeks, then weekly
(3) Ultrasound examination every month
(4) Non-stress test at 34 weeks, then weekly
(5) HbA_{1C} not necessary
(6) No 24-h urine, ophthalmologic evaluation or fetal ECG necessary
(7) Daily fetal movement counts

Class B and C

(1) Daily home glucose monitoring
(2) Biweekly visits until 34 weeks, then weekly
(3) Ultrasound: dating at 20 weeks (profile and echocardiogram), then monthly
(4) HbA_{1C} monthly

(5) Non-stress test at 33 weeks, then weekly
(6) Ophthalmologic evaluation, follow-up according to findings
(7) 24-h urine initially and in each trimester
(8) Daily fetal movement counts

Class D to H: Above, plus the following: ECG initially, uric acid, liver function tests, fibrinogen and fibrin split products in each trimester

Delivery time: Class A and B: <42 weeks' gestation
Class C to H: at term gestation or pulmonic maturity (weekly amniocentesis starting at 38.5 weeks)

Labor:
(1) Blood glucose to be maintained at <100 mg/dl
(2) Intravenous: D5½ NS solution and 10 units of regular insulin ml/h – 1 unit insulin/h
(3) D5½ NS solution piggy-backed to insulin-carry solution to adjust glycemia
(4) Hourly finger-stick blood glucose determinations

Diabetes screening

50-g oral glucose load, between 24 and 28 weeks' gestation, without regard to time of day or prandial state; venous plasma glucose measured 1 h later
If value ≥140 mg/dl – schedule 3-h GTT

3-h GTT: 100-g oral glucose load in a.m. > fast of 8 h
Patient should remain seated and not smoke throughout testing

FBS	≥105
1 h	≥190
2 h	≥165
3 h	≥145

If screen is normal, no further dipstick required after 24 weeks' gestation (for glucosuria) (Gribble RK, Meier PR, Berg RL. The value of urine screening for glucose at each prenatal visit. *Obstet Gynecol* 1995; 86:405–10)

Abnl GTT (class A + B) [if 3-h GTT abnl]:
Glucose weekly
Biweekly visits until 34 weeks, then weekly
Ultrasound every month
NST at 34 weeks, then weekly
Daily fetal movement counts

Antepartum surveillance of the diabetic pregnancy

Test	*When to initiate*
Maternal assessment of fetal activity	28 weeks
Non-stress test	Weekly beginning at 28 weeks; twice weekly beginning at 34 weeks
Contraction stress test	Any time a non-reactive non-stress test is obtained
Biophysical profile	In conjunction with contraction stress test
Non-stress test	
Fetal body movements	
Fetal breathing	
Fetal tone	
Volume of amniotic fluid	
Hemoglobin A1C levels	Early in gestation or upon presentation for prenatal care, also at any time maternal compliance is questioned
Ultrasound	Every 4–6 weeks (to screen for fetal macrosomia, fetal size and determination of the best route for delivery)

Patient-monitored capillary blood glucose goals during pregnancy in diabetic women

Specimen	*Blood glucose (mg/dl)*
Fasting	60–90 (3.3–5.0 mM)
Pre-meal	60–105 (3.3–5.8 mM)
Postprandial 1 h	100–120 (5.5–6.7 mM)
0200–0600	60–120 (3.3–6.7 mM)

Risk factors for gestational diabetes

- Age 30 or older
- Obesity
- Hypertension
- Glycosuria during the current pregnancy
- Prior delivery of an infant with birth weight > 9 lb
- Prior stillbirth
- One or more family members with diabetes mellitus

Screening and diagnostic criteria for gestational diabetes mellitus

Screening

All pregnant women without a diagnosis of gestational diabetes prior to 24 weeks
50-g oral glucose load, between 24 and 28 weeks' gestation, without regard to time of day or prandial state
Venous plasma glucose measured 1 h later and
Value of ≥140 mg/dl (7.8 mmol/l in venous plasma indicates need for 3-h glucose test)

Diagnosis

100-g oral glucose load, administered in morning after overnight fast of 8–14 h and after at least 3 days of unrestricted diet (≥150 g carbohydrate) and physical activity
Venous plasma glucose is measured at fasting, 1, 2 and 3 h after glucose load (subject should remain seated and not smoke throughout test and
Two or more of the following venous plasma concentrations must be met or exceeded for positive diagnosis:

Fasting	105 mg/dl (5.8 mmol/l)
1 h	190 mg/dl (10.6 mmol/l)
2 h	165 mg/dl (9.2 mmol/l)
3 h	145 mg/dl (8.1 mmol/l)

American College of Obstetricians & Gynecologists (1994) criteria for diagnosis of gestational diabetes using 100 g glucose taken orally – gestational diabetes is diagnosed when any two values are met or exceeded

	Plasma glucose (mg/dl)	
Timing of measurement	*National Diabetes Data Group (1979)*	*Carpenter & Coustan (1982)*
Fasting	<105	<95
1 h	<190	<180
2 h	<165	<155
3 h	<145	<140

Classification of diabetes complicating pregnancy

Class	*Onset*	*Fasting plasma glucose*	*2-h Postprandial glucose*	*Therapy*
A1	Gestational	<105 mg/dl	<120 mg/dl	Diet
A2	Gestational	>105 mg/dl	>120 mg/dl	Insulin

Class	*Age/onset*	*Duration (years)*	*Vascular disease*	*Therapy*
B	Over 20	<10	None	Insulin
C	10–19	10–19	None	Insulin
D	Before 10	>20	Benign retinopathy	Insulin
F	Any	Any	Nephropathy*	Insulin
R	Any	Any	Proliferative retinopathy	Insulin
H	Any	Any	Heart	Insulin

*When diagnosed during pregnancy: 500 mg or more proteinuria per 24 h measured before 20 weeks' gestation

Classification of diabetes in pregnancy

Pre-gestational diabetes

Class	*Age of onset (years)*	*Duration (years)*	*Vascular disease*	*Therapy*
A	Any	Any	No	Diet only
B	> 20	<10	No	Insulin
C	10–19	10–19	No	nsulin
D	Before 10	> 20	Benign retinopathy	Insulin
F	Any	Any	Nephropathy	Insulin
R	Any	Any	Proliferative retinopathy	Insulin
H	Any	Any	Heart disease	Insulin

Gestational diabetes

Class	*Fasting glucose level*	*Postprandial glucose level*
A1	<105 mg/dl and	<120 mg/dl
A2	>105 mg/dl and/or	>120 mg/dl

Gestational diabetes

Screening

(50-g glucose, check glucose 1 h later; if > 135, 3-h GTT)

(1) First visit and at 28 weeks for patients with one of the following risk factors:
  (a) Family hx of DM (*H/o repeated pregnancy loss)
  (b) > 25% above IBW (*Previous child > 4000 g)
(2) All other OB patients: 24–28 weeks

Diagnosis

(1) All patients with abnl 1 h ***or*** random glucose >135
(2) 3-h GTT
  (a) Nl activity
  (b) No intercurrent illness
  (c) Adequate diet for 3 days prior to test
  (d) Fasting glucose – 100 g glucose – blood glucose at 1 h, 2 h, 3 h

ABNORMAL VALUES*:	FBS	> 105	
	1-h	> 190	*Two or more = GDM
	2-h	> 165	
	3-h	> 145	

Management

(1) Diet modification
  (a) Consult nutritionist ***or***
  (b) 36 kcal/kg or 15 kcal/lb (IBW) + 100 kcal/trimester
  (c) Diet composition: 40–50% CHO, 12–20% protein, 30–35% fat
(2) Glucose monitoring (Pt diary)
  (a) FBS <105, 2-h postprandial <120 q.d.
  (b) If either consistently abnl, insulin

Insulin

Anticipated requirements:

EGA	6–18 weeks	0.7 U/kg	Type I & II DM (pre-existing)
	18–26 weeks	0.8 U/kg	Type I & II DM (pre-existing)
	26–36 weeks	0.9 U/kg	
	36–40 weeks	1.0 U/kg	

Initiate therapy at ½ above doses

Distribution

NPH alone	*NPH + REG*	
a.m. 2/3	a.m. 2/3 – 2/3 NPH	1/3 REG
p.m. 1/3	p.m. 1/3 – 1/2 NPH (q.h.s.)	1/2 REG (AC)

Change only one insulin dose per week

Pt diary AC & HS (8, 12, 17, 22)

Maternal/fetal surveillance

Type I/II IDDM

Mother (at intake)

– thyroid panel, 24-h urine protein/Cr Cl/BUN/Ophtho consult/HgA, C (then q. 6 weeks)

Fetus	– NST protocol		
	– mother with vascular disease	30–33 weeks	q. week
		34–36 weeks	3 × per week
		36+ weeks	q. day
	– no vascular disease	32–35 weeks	q. week
		36–37 weeks	3 × per week
		37+ weeks	q. day

Gestational DM

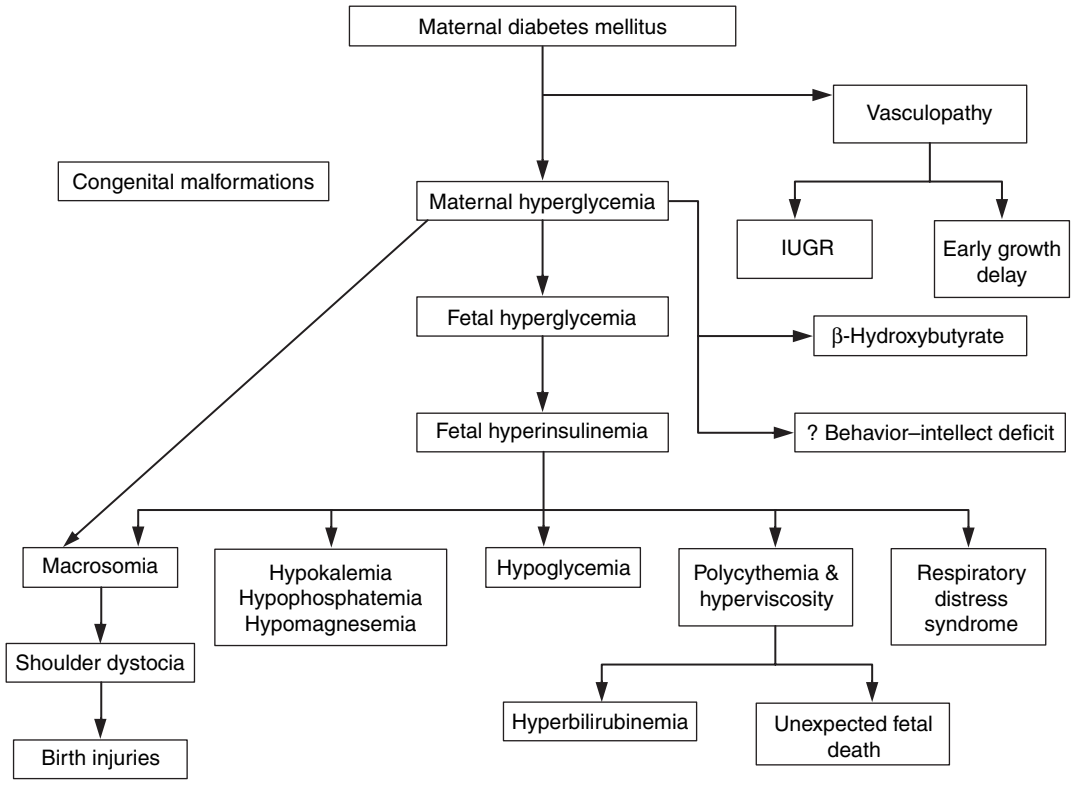
Maternal – HA, C q. 6 weeks

Fetus – NST protocol

– diet-controlled	38+ weeks	q. week
– insulin requiring	32–35 weeks	q. week
(same as above; no vasc dx)	36–37 weeks	3 × per week
	37+ weeks	q. day

Consider AOC after 36 weeks for FLM in insulin-requiring

Problems when diabetes is a factor



Diabetes – Study summary

Gestational	A1 FBS	< 105 mg/dl
	A1 2-h PP	< 120 mg/dl
	A2 FBS	> 105 mg/dl
	A2 2-h PP	> 120 mg/dl
B	Age	> 20 years old
	Duration	< 10 years
C	Age	10–19 years old
	Duration	10–19 years
D	Age	< 10 years
	Duration	> 20 years
F	Nephropathy	
R	Retinopathy	
H	Heart	
Screen	FBS × 2	140
	3-h GTT	105, 190, 165, 145
Diet percentages	Example: (30 kcal/kg so 70 kg × 30 = 2100 ADA diet How much fat in an 1800 g diet? 30% of 1800 = @600 Divide 600 by 9 to get @ 60 g of fat	
	Carbohydrates	50%
	Fats	30%
	Proteins	20%
Gram/Cal formula	Carbohydrate	4
	Fat	9
	Protein	4
Insulin dosage calculation	Example: 80 kg in third trimester = 80 × 0.9 = 72 units of insulin	
	First trimester with Wt in kg x	0.5
	Second trimester with Wt in kg x	0.7
	Third trimester with Wt in kg x	0.9
	then a.m. NPH to Reg (also total a.m. to p.m. is also)	2/3 to 1/3
	then p.m. NPH to Reg	½ to ½
Insulin reference guide	*Rapid acting* Humalog® (lispro): onset is within 15 min, peaks 0.5–1.5 h, lasts 4–6 h Novolog® (aspart): onset is within 15 min, peaks 1–3 h, lasts 4–6 h *Short acting* (regular)(R): onset is within ½ to 1 h, peaks 2–3 h, lasts 6–8 h *Intermediate acting* NPH (N): onset is 2–4 h, peaks 6–10 h, lasts 14–18 h Lente®: onset is 3–4 h, peaks 6–12 h, lasts 16–20 h *Long acting* Ultralente®: onset is 6–10 h, peaks 10–16 h, lasts 20–24 h Glargine (Lantus®): onset is 2 h, peakless, lasts 24 h *Fixed combination of N & R* 70/30 = 70% N & 30% R: onset is ½ to 1 h, dual peak, lasts 14–18 h 50/50 = 50% N & 50% R: onset is ½ to 1 h, dual peak, lasts 14–18 h	
Pregestational diabetes	FBS	60–90 mg/dl
	Before meals	60–105 mg/dl
	After meals (1 h)	130–140 mg/dl
	(2 h)	120 mg/dl
	2 a.m. to 6 a.m.	60–90 mg/dl
Gestational diabetes	FBS criteria for A1 diabetic	< 105 mg/dl
	FBS criteria for A2 diabetic	> 105 mg/dl
	2-h postprandial criteria for A1 diabetic	< 120 mg/dl
	2-h postprandial criteria for A2 diabetic	> 120 mg/dl
	The best measure of overall metabolic control during the pre-conception period is	$HgbA_{1C}$
	$HgbA_{1C}$: most significant risk of malformations	> 10%
	$HgbA_{1C}$: risk of malformation in an insulin-dependent diabetic pregnancy with value > 8.5%	22%
	Threshold for being considered under good control	6%
	EFW to perform C-section on diabetic	4250–4500 g or >

Daily diabetic thresholds
FBS 60–90 mg/dl
2-h PP < 120 mg/dl
Diet 2200–2400 or 1800–2000 cal (Ob Prolog 4th edn)
Ultrasound + MSAFP @ 18–20 weeks
MSAFP can be lower in diabetics
BPP, NST, CST third trimester
Maintain euglycemia – plan normal delivery but > 4500 g, plan C-section

Intrapartum management of diabetes

Maintain euglycemia by holding a.m. insulin, giving dextrose IV and giving short-acting insulin by checking glucose values every 1–2 h
Pre-term treatment: use mag sulfate (not β-sympathomimetic)

Diabetic ketoacidosis

Treated by giving IV insulin bolus of 10–20 U reg
Then giving 5–10 U/h
Run IVFs of NS 1 liter/h × 2 h then add 5% glucose in water after glucose < 250 mg/dl
Do not give bicarb unless pH is < 7.0–7.1
Diabetic ketoacidosis can result in pregnancy losses as high as 50%
Ketones produced are β-OH-butyrate and acetoacetate. Total deficits in DKA are 3–6 liters

Treatment of gestational diabetes (A1)

Diet of 1800–2000 cal
Calculated for @ 30 kcal/kg ideal body weight
Start insulin if, despite dietary restrictions, the patient has persistent values of:
FBS > 105 mg/dl
1-h postprandial > 140 mg/dl
2-h postprandial > 120 mg/dl

Postpartum evaluation

2–3% of *all* pregnancies complicated by diabetes
Gestational diabetes mellitus (GDM) comprise 90% of this 2–3%
Type I (insulin-dependent), Type II (non-insulin dependent)
Evaluate postpartum:

Time tested	*No DM*	*IMP glucose intolerance*	*Diabetes mellitus*
FBS	< 115	< 140	> 140
½, 1, 1½ h	All < 200	1 value > 200	1 value > 200
2 h	< 140	140–199	> 200

*Above values based on a 2-h, 75-g oral GTT
*FBS determinations of ≥ 140 on two occasions establish the diagnosis

Complications of insulin dependent diabetes mellitus

Diabetic nephropathy defined by what amount of albumin/24 h specimen? > 500 mg
Normal albumin excretion rate < 15–20 μg /min
Albumin excretion rate that is STRONGLY associated with eventual development of nephropathy > 30 μg/min
In term infant, hypoglycemia is defined as blood sugar below what level on two occasions during the first 72 h of life? 30 mg/dl
An infant of diabetic mother is lethargic. Glucose is 40%. Central stick is ordered. At what level of Hct would one consider a partial exchange transfusion due to polycythemia? 65%
What % of macrosomic infants (> 4000 g) of diabetic mothers have shoulder dystocia 30%
What % of macrosomic infants (> 4500 g) of diabetic mother have shoulder dystocia 50%
Type I diabetes is associated with chromosome 21
What is the risk for congenital anomalies in infants born to mothers with Type I diabetes? 6–12%
Which congenital anomaly is most common in infants born to diabetic mothers relative to those born to mothers in the general population? Caudal agenesis

Prevalence of diabetes (diagnosed and undiagnosed) in adults in the USA is what %? 0.6%

Background retinopathy not a contraindication to pregnancy. However, background retinopathy progresses to proliferative retinopathy in what % of patients? 16%
In women with Type I diabetes, the risk for progression of proliferative retinopathy is increased by proteinuria, hypertension and pregnancy
This needs aggressive treatment with possible termination of pregnancy becoming necessary

Rosiglitazone (Avandia or if in combination with metformin→Avandamet and if with glimerpiride→Avandaryl) that is used in treatment of type 2 diabetes has been shown to have a fracture rate of 2.74 per 100 patient-years, significantly higher than the rates in two other treatment groups

DIETHYLSTILBESTROL (DES) SYNDROME

Associated with Vaginal adenosis
Ectropion cervix
Cockscomb cervix
Clear cell carcinoma
Hypoplastic uterine cavity

Increased risks of clear cell vaginal adenocarcinoma 1/1000
Repeat Paps of cervix AND vaginal cytology
Associated with abnormalities of the cervix, uterus and upper vagina
Increases risks for Spontaneous abortion
Preterm cervical effacement Preterm labor
Ectopic pregnancy
Increased breast cancer (1.3 relative risk) slight

DES-exposed patients

Adenosis 90% if < 8 weeks when exposed
10% if > 16 weeks when exposed
Adenosis found in proximal 1/3 of anterior vagina
Clear cell adenocarcinoma – anterior upper 1/3 of vagina or on posterior ectocervix
Reddish or induration. Vaginal discharge or bleeding
If Paps or colpo are normal then still do annual palpation and Pap of vagina. Also look for collars, hoods, septa, cockscombs, incompetence, uterine or fallopian tube defects

Remember:
Adenosis occurs if DES given < 8 weeks' gestation 90%
Adenosis occurs if DES given > 16 weeks' gestation 10%
Clear cell adenocarcinoma of vagina or cervix in a DES-exposed patient has risk of development 1 : 1000
Lesions usually occur along posterior 1/3 of anterior vagina or on posterior ectocervix

DISCHARGE

Stable vital signs
No evidence of untreated infections
Adequate oral intake
Satisfactory bowel and urinary tract function

DISSEMINATED INTRAVASCULAR COAGULATION (DIC)

Treatment

Platelets If platelet count < 50 000
Packs of 6–10 units. One unit increases plt ct 5000–10 000
FFP (4 : 1 is ratio of PRC to FFP) Give if hypovolemia
Cryoprecipitate Give if hypofibrinogenemic
4 g fibrinogen 15–20 u @ 100 mg/dl

See Blood products

DNA VIRUSES

DNA viruses to remember	Hepatitis B HPV Herpes Varicella

DOMESTIC VIOLENCE HOTLINE

1-800-799-SAFE	
Childhood abuse increases risk factor	Repeat cycles

DOUBLE BUBBLE SIGN

	Seen with duodenal atresia – fetal small bowel obstruction causing dilatation proximal to obstruction
	Double bubble is seen with Down syndrome on ultrasound in what % of patients? 30%
	Usually normal appearing in second trimester and seen most often in third trimester Seen @ 1/10 000
Some ultrasonic findings	(1) Duodenal atresia (classic 'double bubble') (2) Cardiac defects – e.g. endocardial cushion defects (3) Cystic hygroma – septated areas @ neck

DOWN SYNDROME

	Major sonographic findings with Down syndrome occur at 14–24 weeks in 25% cases
	Structural defect has the largest likelihood ratio for detecting Down syndrome
	A 'normal' genetic sonogram cannot reduce the age-related risk of Down syndrome below 1/270
	once a subject has attained the maternal age of 35
Ultrasonic findings	(1) Hyperechoic bowel (2) Increased nuchal skinfold measurement (≥ 6 mm) (3) Complex heart disease
Recommendations	ACOG Practice Bulletin No. 77 (*Obstet Gynecol* 2007; 109: 217–27) endorses routinely offering first-trimester screening for fetal chromosomal abnormalities to all pregnant women, not just those over 35 years of age. First-trimester screening using nuchal translucency and biochemical markers results in higher detection rates than second-trimester maternal serum triple screen and is comparable to or better than the quadruple screen at equivalent false-positive rates
	A nuchal translucency value of 3–4 mm or more warrants immediate chorionic villus sampling

DRUG CATEGORIES

Controlled studies in female failed to demonstrate any risks	A
Have not demonstrated any risks but there are no controlled studies	B
Revealed adverse effects but no controlled studies. Give only if benefit justifies risks	C
Positive evidence of *human* fetal risk. Use may be acceptable despite risks	D
Studies demonstrate fetal abnormalities. Drugs in this category are contraindicated in pregnancy	X

DUBLIN PROTOCOL

Active management of labor (derived from Dublin, Ireland). Lowers C-section rate to 4.8%

(1) Patient education
(2) Strict criteria for:
  (a) Diagnosis of labor
    Regular painful contractions AND one of:
    Passage of mucus plug
    Complete effacement or spontaneous ROM
  (b) Determination of abnormal labor > 1.2 or > 1.5 cm/h
  (c) Interpretation of fetal compromise
(3) High-dose Pitocin protocol
  6 mu/ml increased by 6 mu/ml every 15 min to max out at 40
(4) Personal nurse in labor
(5) Peer review of all operative deliveries

• AROM @ 1 h after admission if not already ruptured
• Pitocin if dilating less than 1 cm per h

DVT

TVH	7%
TAH	14%
Radical hysterectomy	25%
% of DVT that occurs postpartum	75%
Pregnancy presents an increased risk above baseline of	6–7%
Risk of DVT in 28-year-old patient not using contraception	10/100 000 women
Risk of DVT in a 49-year-old patient on HRT	30/100 000
Risk of DVT in a 32-year-old patient with uncomplicated 24 weeks IUP	60/100 000
Overall risk is greatest during postop period after major surgery – risk rises	10 ×
Age increases risk of DVTs	
Antepartum DVT that results in PE if left untreated	25%
If antepartum DVT treated then incidence of PE drops to	4.5%
Clotting factors responsible for thromboembolic episodes:	
Factor VIII increase	25%
Fibrinogen increase	4 ×
Leiden V factor (homozyg 80–90 ×)	20%
Homocysteine (folate, B_6, B_{12} decrease this)	10%
Protein 20280	6%
Protein C deficiency (1.7)	*3%
Protein S deficiency (6.6)	*1–2%
Postpartum development of pulmonary embolism is relatively uncommon with an incidence of	1 : 5000
Symptoms How many patients are asymptomatic?	50%
Of the patients who are symptomatic:	
Induration of calf muscles	68%
Minimal edema	52%
Calf tenderness	25%
Difference in leg diameter > 1 cm	11%
Homan's sign	10%
Differential a DVT from a postmortem clot at autopsy	
The DVT looks like candy cane if came up from leg	Lines of Zahn
The postmortem clot is simply similar to jello	

Prophylaxis in gyn patients for LMWH

Condition	*Prophylaxis*
Low-to-moderate risk	
Surgery for benign disease Age > 40 and < 60 years	Dalteparin 2500 U 2 h preop, then daily postop Enoxaparin (Lovenox®), 20 mg deep SC, 30 min to 2 h preop, then daily postop

Continued

continued

Obesity	
Venous stasis changes	
Smoking history	
High risk	
History of DVT	Dalteparin 5000 U 10–12 h preop, then daily postop; or 2500 U 2 h preop, 2500 U 12 h postop, then 5000 U daily thereafter
Diagnosis of cancer	
Radical surgery	Enoxaparin (Lovenox®) 40 mg 2 h preop, then daily postop
Very high risk	
Exenteration	LMWH (low-to-moderate risk doses) plus external pneumatic compression
Radical vulvectomy	
Two of the following three risk factors:	
(1) Age > 60 years	
(2) History of DVT	
(3) Diagnosis of cancer	

Treatment	Enoxaparin (Lovenox®) 1 mg/kg SC q. 12 h (maximum dosage of 150 mg q. 12 h)	
	or enoxaparin (Lovenox®) 1.5 mg/kg SC q. 24 h (maximum dose of 150 mg, if dose > 150 mg, use q. 12 h dosing)	
	Dosing cautions: Low-weight patients (< 45 kg) – adjustment may be needed, specific guidelines are not available Renal failure – enoxaparin is primarily excreted through the urine Apparent clearance values have been reported as 30% lower in patients with CrCl < 30 ml/min. Adjustments should be considered but specific guidelines are not currently available. For patients with CrCl < 30 ml/min, unfractionated heparin may be best choice	
	Heparin to Lovenox® Wait 4 h after the last dose of heparin to start Lovenox	
	Lovenox to heparin Wait 12 h after the last dose of Lovenox to start heparin	
Prophylaxis in pregnancy	LMW heparin (enoxaparin 1 mg/kg q. 12 h)	30 mg b.i.d.
	Unfractionated heparin (keep Aptt 1.5–2.5)	5000 mg SC b.i.d.
	Increase to this in second trimester	7500 mg SC b.i.d.

Condition	*Prophylaxis*
Low-to-moderate risk	
Antiphospholipid antibody syndrome with a history of stillbirth or recurrent pregnancy loss but no TE	Dalteparin, 100 U/kg every 12 h Enoxaparin, 30 mg every 12 h prior to 28 weeks: 40 mg every 12 h after 28 weeks
Protein S deficiency Protein C deficiency History of TE not associated with pregnancy Heterozygosity for factor V Leiden or prothrombin 20210 and a history of TE	
High risk	
Antiphospholipid antibody syndrome with a history of TE	Dalteparin 200 U/kg every 12 h
Antithrombin III deficiency History of TE in pregnancy of puerperium Homozygosity for factor V Leiden or prothrombin 20210	Enoxaparin 1 mg/kg every 12 h
DVT or PE	Enoxaparin (Lovenox®) 1.5 mg/kg q. daily SC

Prophylaxis is cost-effective. Graded elastic stockings with intermittent pneumatic compression devices – most cost-effective and efficient method of prevention

DYSFUNCTIONAL UTERINE BLEEDING

Diagnosis of exclusion

Eliminate organic lesions, coagulation disorders

(1) Anovulatory – postmenarchal secondary to continuous E_2 production with corpus luteum formation and progesterone production → proliferative endometrium
(2) Ovulatory – after adolescent and < perimenopausal

Labs:
(1) (a) CBC; (b) TSH; (c) Serum prolactin; (d) Androgens such as androstenedione, testosterone, DHEA-S; (e) FSH + LH
(2) Endometrial biopsy (if premenopausal)
(3) Hysteroscopy

Treatment

(1) Conservative
(2) Progestins
(3) Low-dose OCPs
(4) IV estrogens with p.o. estrogens
(5) Prostaglandin synthesis inhibitors (naproxen, Cox II inhibitors, ibuprofen) before bleeding
(6) Ablation/hysteroscopy or hysterectomy

DUB with profound anemia

(1) IV conjugated equine estrogens 25 mg every 4 h × 3 doses, then premarin 2.5 mg daily × 3 weeks or
(2) OCP tapered regimen:
4 pills × 4 days then
2 pills × 2 days then
1 pill daily to complete a 21-day pill pack

Nausea and vomiting may interfere with absorption and compliance; therefore Zofran® 8 mg ODT (oral disintegrating tablets) every 6 h can be given if necessary. These do not cause somnolence like Phenergan®, Tigan®, etc. therefore do not interfere with work

DYSMENORRHEA

Painful menstrual cramps

Leukotriene increases myometrial contractions F_2

Primary dysmenorrhea

No apparent pathology

Etiology
Increased production and release of prostaglandins which cause increased contractions and uterine activity thus decreased uterine blood flow thus ischemia and pain (occurs only in females with ovulatory cycles)

Examination
Normal, menses regular, duration of pain 2–3 days

Treatment
(1) NSAIDs – block prostaglandin synthetase thus block production of prostaglandins (70–90% success)
a) Ibuprofen 800 to 1200 mg initial then 800 mg every 6 hours.
b) Naproxen sodium (Anaprox, Naproxyn) 250 to 500 mg initially then 250 mg every 6 hours.
(2) Mefenamic acid (Ponstel® 250–500 mg then 250 mg every 6 h)
(3) Cox II inhibitor although because of the growing concerns about the adverse affects of COX-2 inhibitors, older NSAIDs are probably preferred over the newer COX-2 inhibitors.
(4) OCPs (oral, transdermal, or intravaginal) or
(5) Combination of NSAIDS and OCPs
(6) Heat (ThermaCare): one study found topical heat was similar or superior to oral ibuprofen. (Research suggests that heat and ibuprofen would work well together but seem to have a similar mechanism of action.)

Secondary dysmenorrhea

Pathology

Etiology or causes of secondary dysmenorrhea
Endometriosis
Submucous leiomyoma
IUD
Adenomyosis
Adhesions
Malformations

Examination
May be abnormal, > menarche, usually irregular menses, usually anovulatory
DIAGNOSTIC LAPAROSCOPY NEEDED

Treatment
Depends on the cause

Dysmenorrhea incidence in US females	5%
Most common in females between	20–24 years old

DYSPNEA IN PREGNANCY

Caused by increased levels of estrogen and progesterone

Occurs in what % of pregnant pts?	76%
By 20 weeks' gestation	50%
By 30 weeks' gestation	76%
Physiological – occurs in how many pregnancies?	3/4

Causes
Increased levels of estrogen and progesterone. Airway conductance and lung compliance are increased due to progesterone induced bronchial smooth muscle relaxation → increases tidal volume and decreases residual capacity → increases minute ventilation → second trimester →increases tidal volume by 40% → mild alkalosis→ increases respiratory rate by 10–15%
'Perception of shortness of breath'

DYSTOCIA

Criteria that must be met prior to arrest disorder being diagnosed:

(1) Latent phase complete	4 cm
(2) Uterine contraction pattern × 2 h without cervical change in Montevideo units	200 MV units
Nulliparous patient with prolonged latent phase	> 20 h
Nulliparous patient with prolonged second stage	> 2 h
Nulliparous patient with prolonged second stage with epidural in place	> 3 h
Multiparous patient with prolonged latent phase	> 14 h
Multiparous patient with prolonged second stage	> 1 h
Multiparous patient with prolonged second stage with epidural in place	> 2 h

Arrest disorder = complete cessation of progress
Protraction disorder = slower than normal labor:

Nulligravid patient	< 1.2 cm per h
Multigravid patient	< 1.5 cm per h

Evaluate the '3 Ps'

(1) Powers – uterine contractility

How many contractions should there be in a 10-min window?	3–5
What % of patients require over 200–224 MV units?	91%
What % of patients require ≥ 300 MV units?	40%

(2) Passenger – the fetus
Evaluate the weight, position and attitude
(3) Passage – bony pelvis
Deeply engaged head with OP and narrow maternal pubic arch is best delivered without rotation

Predicting shoulder dystocia
Difficult (Prevention is impossible)

Mid-pelvic delivery, prolonged second stage or macrosomia
increased risk 23%
Macrosomia (4.5 kg) and/or diabetes increased risk 50%
Patient should be fully appraised of options. Remember, 1000s of C-sections with all the risks, complications (ileus, hemorrhage, PE), morbidity and mortality are needed to prevent just a few dystocia cases

Risks

Women with gestational diabetes and/or a macrosomic fetus are at highest risk for shoulder dystocia

		Weight (g)	*% Dystocia w/out +*	*with Diabetes*
(1)	> 8#	< 3000	0.2	
		3000–3499	0.8	3.7
		3500–3999	2.3–2.9	
		4000–4499	8.6–10.3	23.1
		> 4500	24–35.7	50

(2) Antepartum
  (a) Birth weight
  (b) Fundal height
  (c) Maternal diabetes
  (d) Maternal pre-pregnancy weight; maternal wt gain during pregnancy
  (e) Post-term pregnancy (> 42 weeks)
  (f) Prior delivery with shoulder dystocia
  (g) Wt of largest previous infant (> 4500 g)
(3) Intrapartum
  (a) Prolonged second stage
  (b) Prolonged second stage plus mid-pelvic delivery
  (c) Prolonged decel phase

Management

Maneuvers of shoulder dystocia
(Initially call for help if available)

(1) McRobert's
(2) Suprapubic pressure
(3) Wood's maneuver – corkscrew
  Post-shoulder 180 degrees. Body not head – not independent of body.
  (Variant: Rubens maneuver where Ob pushes on posterior aspect of the posterior shoulder causing shoulder abduction)
(4) Mazzanti maneuver
  Delivery of post-shoulder; trace humerus to elbow, flex elbow so forearm is first
  delivered across chest and out
(5) Fractures
  (a) Clavicle
  (b) Humerus
(6) Extended episiotomy – 4th degree procto episiotomy

NEVER APPLY EXCESSIVE TRACTION

(7) Zavanelli maneuver
  Cephalic replacement as initial maneuver rather than last resort if difficulty encountered especially to those who are inexperienced in dystocia treatment
  (O'Leary JA. Cephalic replacement for shoulder dystocia: present status and future role of the Zavanelli maneuver. *Obstet Gynecol* 1993; 82: 847–50)
  Some sources say try to avoid at all cost secondary to increase neurological injuries and decreased experience (@ 40 known documented cases)
(8) Mueller–Hillis maneuver
  (Thorp JM Jr, Pahel-Short L, Bowes WA Jr. The Mueller–Hillis maneuver: Can it be used to predict dystocia? *Obstet Gynecol* 1993; 82: 519–22.)
  Applying fundal pressure to see if head moves down in pelvis – no longer recommended
(9) Gaskin or "all fours" maneuver – advocated by midwives but sometimes this takes longer than 4 to 6 minutes available especially if an epidural is in place

Brachial plexus injury

Erb's $C_{5–6}$
Klumpke's $C_8–T_1$
Some are spontaneous

Some are encountered without chart documentation
3–5% < 4.5 kg
15–30% > 4.5 kg
80% resolve in 1 year. The remainder usually show partial recovery without surgery – others unfortunately –
Asphyxia: 20% noted in surviving infants of dystocia. 5–10 min from time cord compressed
Fractured humerus or clavicle – minor – resolve

Shoulder dystocia – *See* Shoulder dystocia

EATING DISORDERS

Anorexia	*Bulimia*
Hypotension	Hypotension
Dry skin with lanugo	Enlarged parotids
Yellow palms	Erosion of tooth enamel
Bradycardia	Cardiac arrhythmias
Hypothermia	

• Normal weight bulimic patients generally do not suffer from osteoporosis whereas anorexia nervosa patients, particularly if associated with binge eating and purging, are at very high risk for osteopenia and osteoporosis

ECTOPIC PREGNANCY

Incidence	1/100
Incidence in blacks and Hispanics increased	1.6 ×
What % of ectopics cause all maternal deaths?	15%
Number of annual deaths related to ectopic pregnancy	25–50
Risk of recurrence of ectopic (two sources)	7–13%
	10–25%
Risk of IUP following ectopic	50–80%
Risk of spontaneous abortion same as general population	
Symptoms (abdominal pain/amenorrhea)	90–100%/77–95%
hCG fails to increase in 48 h by	50–66%
hCG doubles normally with ectopics what % of time	20%
Progesterone less than what excludes viable IUP	5 ng/ml
Progesterone less than 15 ng/ml observed in tubal pregnancies	81%
Progesterone less than 15 ng/ml observed in abnormal pregs	93%
Progesterone less than 15 ng/ml CAN be seen in % normal	11%
Progesterone level that is highly suggestive of viable preg	> 25 ng/ml
Culdocentesis + only if ectopic ruptures which is only	> 20%
Laparoscopy misses diagnosis as it is too small in	2–4%
Transvaginal ultrasound Second International Standard of hCG	500 mIU/ml
Abdominal ultrasound Second International Standard of hCG	6500 mIU/ml
Sac can normally be seen with TV ultrasound when hCG	1000–2000mIU/ml

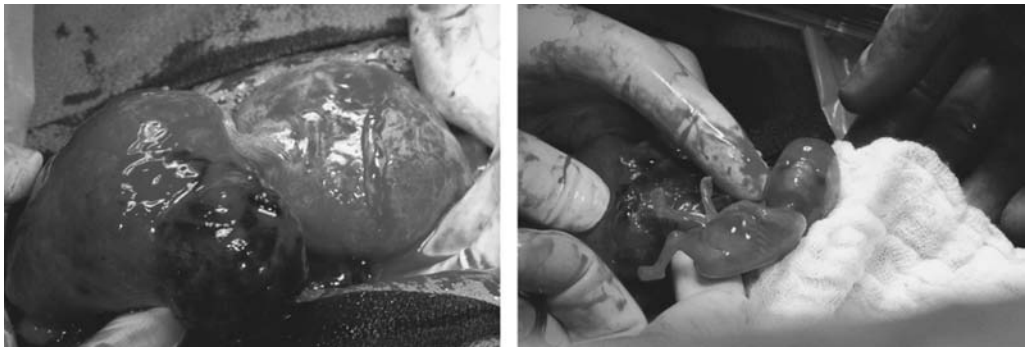


Figure 4 Tubal ectopic pregnancy. (a) Large ruptured fallopian tube from ectopic pregnancy; (b) fetus extruded from tube in patient who presented to ER with shock

Location of pathology	Oviduct	97.7%
	Ampullary portion	81%
	Isthmus	12%
	Fimbria	5%
	Interstitial	2%
	Cornual (development in rudimentary horn of bicornuate uterus). Very rare, causes symptom to develop later, difficult to diagnosis, causes massive hemorrhage	
	Abdominal	1.4%
	Ovarian or cervical (*See* Spiegelberg's criteria)	< 1%
	Ectopics are EXTRALUMINAL – that is why a salpingostomy and not an otomy can be done	
	Ruptures occur into ANTIMESENTERIC side because the ectopic outgrows its blood supply	
Risks	Previous PID, tubal surgery, ectopic, IUD, smoking or progestin-only OCPs	
Signs and symptoms	CLASSIC TRIAD (especially if ruptured)	
	(1) Pain (2) Amenorrhea (3) Vaginal spotting	
Diagnosis	H&P, serial hCG levels, decreased progesterone levels, decreasing estradiol levels, increasing MSAFP, C-reactive protein and Ca-125 levels, vaginal US, curettage, culdocentesis (+ if ruptured but < 20% will not be +), laparoscopy (misses 2–4% due to being small plus risk and expense is increased)	
	If hCG fails to increase by at least	66% or >
	in 2 days → a non-viable pregnancy or an ectopic pregnancy should be assumed	
	hCG < 50% increase in 48 h → abnormal pregnancy	
	Abnormal pregnancy, then hCG rises or falls very slowly and NO POC or pathology → strongly suspect ectopic	
	Serum progesterone < 5 ng/ml	abnormal pregnancy
	> 25 ng/m	lviable pregnancy
	< 15 ng/ml	most ectopics
	Ultrasound diagnosis – sac can normally be seen with TVUS when hCG level is between 1000 and 2000 mIU/ml (first and second IRP)	
Non-surgical management	Single-dose methotrexate	50 mg/m^2 IM
	Single dose and multi-dose regimens for methotrexate treatment of ectopic pregnancy are equally efficacious	
Inclusion criteria	hCG rising, hemodynamically stable	
	Transvaginal sono → unruptured ectopic	
	Ectopic mass < 3.5 cm	
	Patient desires future fertility	
Exclusion criteria	Declining hCG after D&C	
	Mass > 3.5 cm	
	Hemodynamically unstable	
	Desires sterilization	
	Previous sterilization	
	Abnormal CBC or SGOT (WBC < 3000/mm^3 or APT > 50 IU/l)	
	Active pulmonary disease	
	Patient non-compliance	
	Free fluid and pelvic pain (ruptured ectopic)	
Patient instructions	No alcohol, intercourse, vitamins or folic acid and use contraception	
Protocol and follow-up	Day 0	hCG, D&C (?), CBC with diff, SGOT, creatinine, Rh
	Day 1	hCG and give methotrexate
	Day 4	hCG
	Day 7	hCG
	On day 7, hCG should be 15% less than day 4. If not, repeat mtx	
	If cardiac activity on vaginal US, repeat US every other day until no cardiac activity	

Pearls

Usually there is increased pain post mtx injection. If pain is increased, check hematocrit. If lower than previous mtx, do US for increased fluid in cul-de-sac

Most hCG titers on day 4 are greater than day 1 – be patient

Mean time to resolution for hCG to be < 5 is	35–40 days
Failure rate	5.8%
Failure rate if cardiac activity seen	14.3%
Tubal patency rate post-resolution by HSG	83%

Recurrent ectopic rate is less than with linear salpingotomy

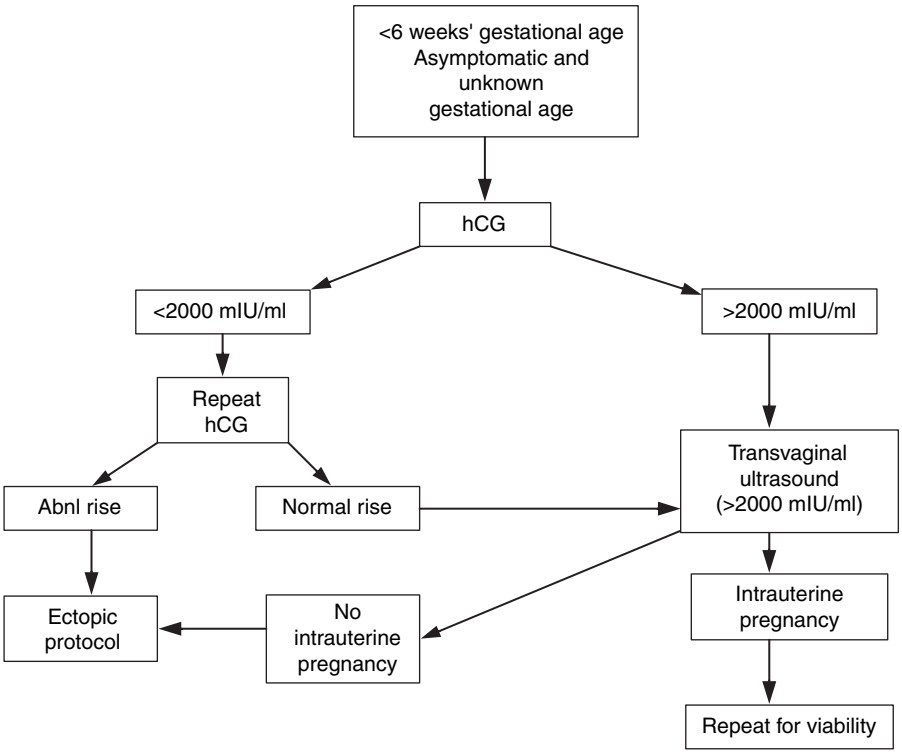
• hCG levels in ectopic pregnancy can be up, down, and all around. The diagnosis of ectopic pregnancy must be made on a combination of laboratory or sonographic and clinical findings. According to one study, approximately the same number of women with ectopic pregnancy experienced an increase in hCG values as did those who experienced a decrease in hCG values. The pattern of hCG measurement for ectopic pregnancy cannot be characterized by a single predictive curve. In 29% of patients with ectopic pregnancy, the hCG profile mimics either an intrauterine pregnancy that is viable or a complete spontaneous abortion (Silva C, Sammel MD, Zhou L, *et al*. Human chorioniogonadotropin profile for women with ectopic pregnancy. *Obstet Gynecol* 2006; 107: 605–10)

Protocols

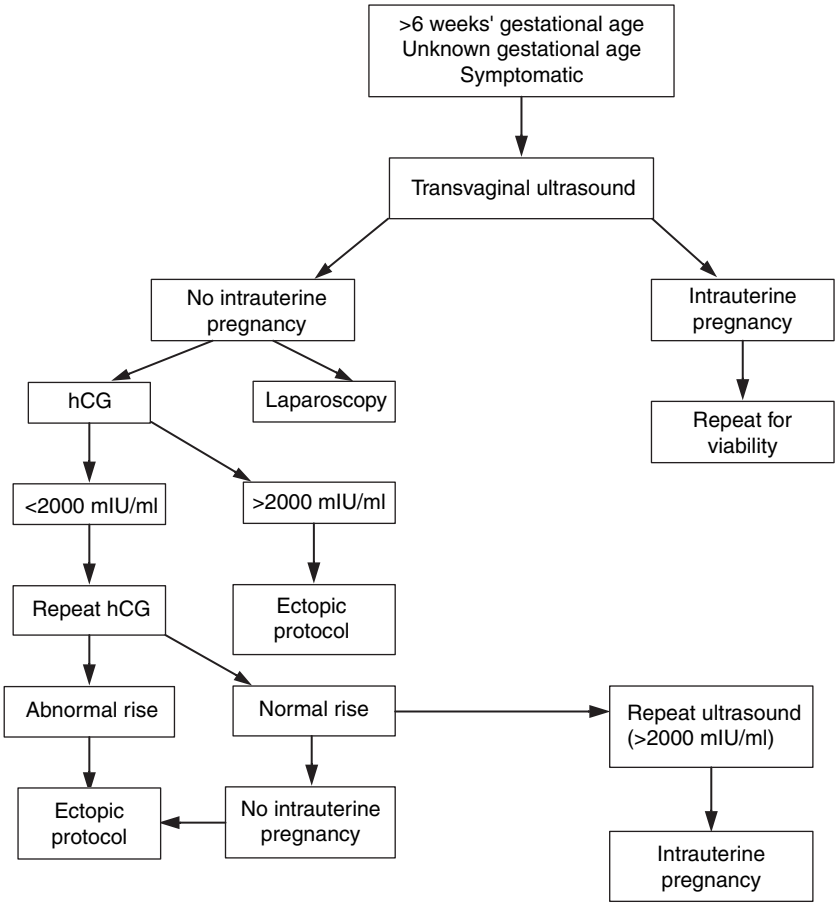
Methotrexate therapy for persistent ectopic pregnancy

Route	*Dosage*	*Success number (%)*
Oral	10 mg p.o. q.d. × 5 days	1/1 (100%)
		2/2 (100%)
		1/1 (100%)
	5 mg p.o. q.d. × 5 days	1/1 (100%)
	5–10 mg p.o. q.d. × 5–7 days	14/15 (94%)
IV	100 mg/m^2 IV bolus over 1 h, then 200 mg/m^2 IV over 12 h, leucovorin 10 mg/m^2 p.o. q. 12 h × 4 doses	3/3 (100%)
IM	1 mg/kg IM q.o.d. × 3 doses Alternating leucovorin 0.1 mg/kg IM q.o.d. × 3 doses	1/1 (100%)
	50 mg/m^2 IM × 1 dose No leucovorin	19/19 (100%)
Local	Not evaluated	

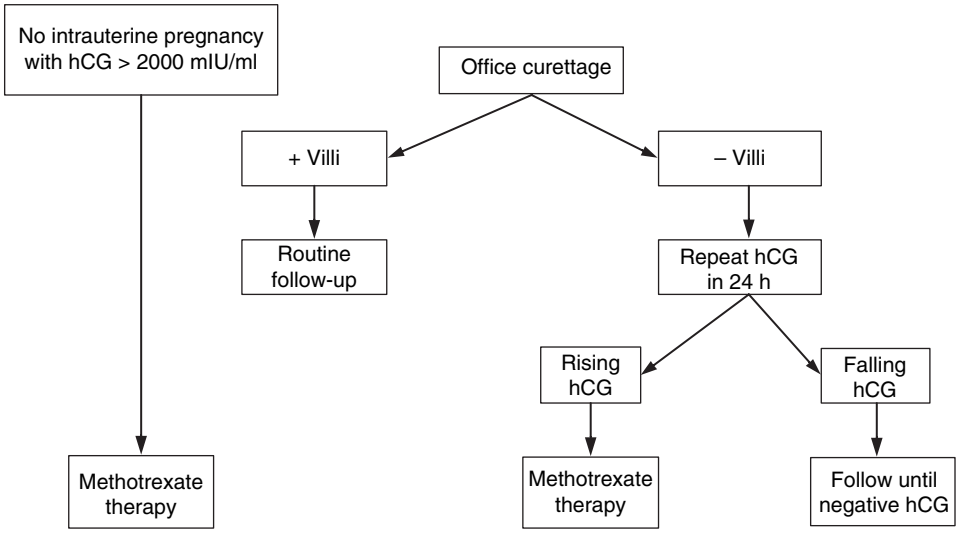
Patient at risk (before 6 weeks)



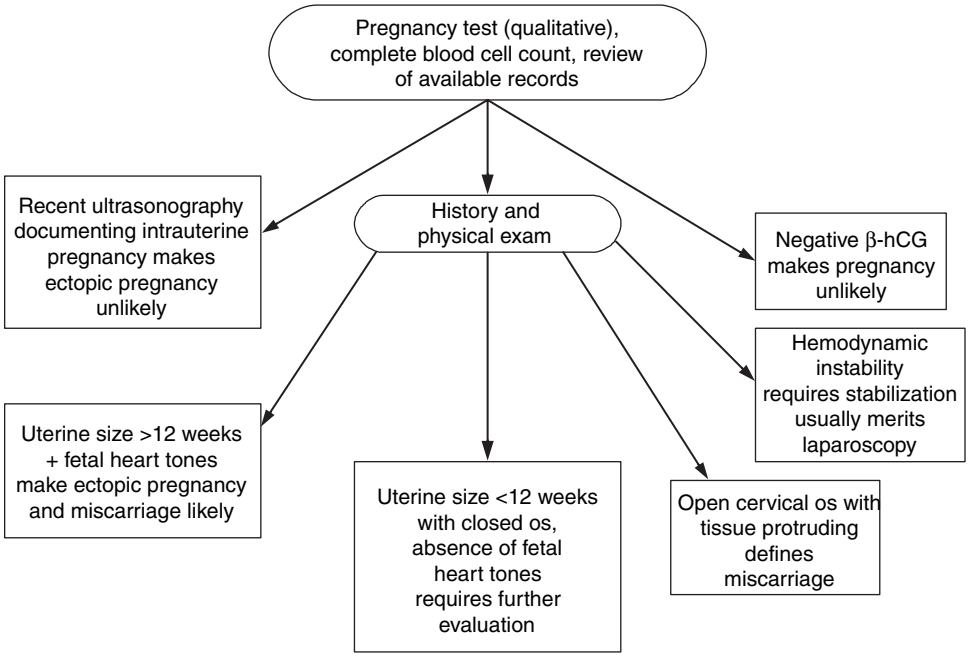
Patient at risk (after 6 weeks)



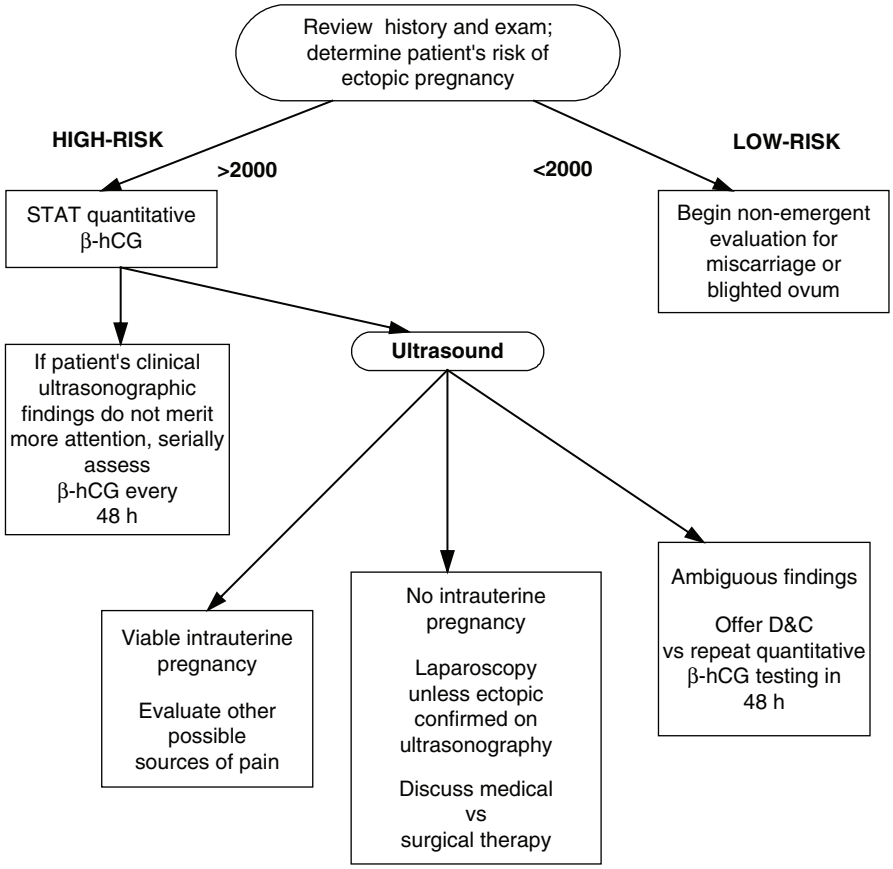
Ectopic protocol



Female patient with lower abdominal pain or abnormal bleeding



Pregnant patient with lower abdominal pain or abnormal bleeding, uterine size <12 weeks or no fetal heart tones, cervical os closed, hemodynamically stable



Risk factors include history of STDs or PID, ectopic pregnancy, pelvic surgery, or prior IUD use; signs of peritoneal irritation (e.g. guarding, rebound); hemodynamic instability; and unreliable or non-compliant patient

Treatment of ectopic with methotrexate is successful if sac diameter < 3.5 cm and NO cardiac act 90–95%
Post-treatment tubal patency (with mtx) 71%
% patients achieve pregnancy after mtx therapy 80%
% recurrence rate after treatment 13%
Persistent ectopic 5%
Diagnose persistent ectopic with post rx β-hCG plateau or increase

Surgical management

Laparoscopy versus laparotomy?
Linear salpingostomy (not sutured) or salpingotomy (sutured)
Salpingectomy (removal of tube) or segmental resection (portion of tube) for reanastomosis later
Milking tube especially if near fimbria (increases recurrence)
Direct injection of mtx (especially interstitial or cornual ectopics < 3 cm)
Corneal resection

- Segmental salpingectomy is the most popular method. Tubal incisions are ANTIMESENTERIC

Bilateral salpingectomy if patient develops ectopic pregnancy after tubal sterilization
Definitive surgical therapy more effective than methotrexate for ectopic pregnancy. The overall success rate of laparoscopy for ectopic pregnancy is around 90% and for methotrexate around 79% according to Lewis-Bliehall C, Rogers RG, Kammerer-Doak DN, *et al*. Medical vs surgical treatment of ectopic pregnancy. *J Reprod Med* 2001;46:983–8

EISENMENGER SYNDROME

VSD with left to right shunting → pulmonary hypertension → bidirectional or right to left shunting → right ventricular hypertrophy
AVOID DECREASES IN B/P (blood loss, regional anesthesia or syncope)

EMBOLUS

% of all deaths after gyn surgery 40%
Second leading cause of death after legal abortion
Leading cause of death in patients with uterine or cervical cancer

Diagnosis

Symptoms are TACHYPNEA, SOB, TACHYCARDIA, CHEST PAIN, HEMOPTYSIS
CXR, EKG, ABG → if abnormal get ventilation–perfusion lung scan. If ventilation–perfusion scan is 'indeterminate' get pulmonary arteriography

Treatment

STAT anticoagulation therapy using heparin IV until aptt is 1.5–2 × nl × 5 days then warfarin (Coumadin®) therapy × 3 with pt 2–2.5 × nl

EMBRYOLOGY

Male sexual differentiation

Undifferentiated gonad → (Y chromosome with TDF (SRY)) →
Embryonic testes → Sertolli cells → MIF → Müllerian regression
Embryonic testes → Leydig cells → testosterone → Wolffian ducts + DHT
Wolffian ducts → vas deferens, seminal vesicle and epididymis
DHT → penis, scrotum and prostate

Intersexuality

See page 22

Important embryological structures and eventual developmental organs to remember

Embryological structure	*Male structure*	*Female structure*
Gubernaculum	Gubernaculum testes	Round ligament
		Ovarian ligament
Mesonephric (Wolffian)	Epididymis	Gartner's duct
	Ductus deferens	
	Ureter	Ureter
Mesonephric (Wolffian) duct	Epididymis	Gartner's duct
	Vas deferens	Duct of epoophoron
	Appendix of epididymis	Appendix of vesiculosa
	Ureter, pelvis, calyces and collecting tubules of kidneys	Ureter, pelvis, calyces and collecting tubules of kidneys
Paramesonephric (Müllerian) duct	Appendix of testis	Uterine tube, uterus, cervix and upper 2/3 of vaginal wall
Urogenital sinus	Bladder and urethra	Bladder and urethra
	Prostate gland	Greater vestibular gland and lower 1/3 of vagina
Sinus tubercle		Hymen
Phallus	Glans penis	Glans clitoris
Urogenital folds	Ventral penis	Labia minora
Labioscrotal swellings	Scrotum	Labia majora

Results of abnormal embryological development:
Transverse vaginal septum – sinovaginal bulb fails to canalize. Includes fusion of Müllerian duct and urogenital sinus
Absence of uterus – paramesonephric duct does not develop
Uterus didelphys – paramesonephric duct does not fuse
Longitudinal vaginal septum – failure of fusion of lower Müllerian ducts. 'Double barrel vagina'

EMERGENCY CONTRACEPTION

	Effectiveness	75% reduction
	In other words, in a single act of unprotected coitus, a woman has an 8/100 chance of becoming pregnant. It is reduced to 2/100 with EC which is a chance of avoiding pregnancy of	98%
	Plan B provides effectiveness of	88% reduction
	which is a chance of avoiding pregnancy of	99%
	The use of an IUD provides effectiveness of	99.9% reduction
Yuzpe method is	Ethinylestradiol	100 µg
	Levonorgestrel	0.5 mg
	Initiate within first	72 h
	May benefit up to first 120 h but decreased efficacy >	
	Midcycle coitus results in pregnancy	8/100
	If emergency contraception used, this is reduced to	2/100
	Use antiemetic @ how soon prior to OCPs?	30 min to 1 h
	Zofran 8 mg ODT is an excellent option as an antiemetic	
Options of Yuzpe	Ovral® (as above) two doses	12 h apart
	Lo-Ovral®, Nordette®, Levlen®, Triphasil®, Tri-Levlen®, 4 doses	q. 12 h × 2
	Low-dose (20 µg) 5 doses (or pills)	q. 12 h apart × 2
Dedicated marketed products		
Examples	Preven™	Same dosing as Yuzpe method
	Plan B	0.75 mg pill of levonorgestrel within 72 h q. 12 h × 2
	Plan B	1 pill ASAP and 1 pill 12 h later www.go2planB.com or 1-800-330-1271
	Preven	1 blue pill ASAP and 2 blue pills 12 h later 0.25 mg levonorgestrel 0.05 mg ethinylestradiol (EE)

Oral contraceptives (Yuzpe)

Ovral	2 white pills ASAP and 2 white pills 12 h later
Lo-Ovral	4 white pills ASAP and 4 white pills 12 h later
Levlen	4 white pills ASAP and 4 white pills 12 h later
Levora®	4 white pills ASAP and 4 white pills 12 h later
Nordette	4 light orange pills ASAP and 4 light orange 12 h later
Triphasil or Tri-Levlen	4 yellow pills ASAP and 4 yellow pills 12 h later
Trivora®	4 pink pills ASAP and 4 pink pills 12 h later
Alesse	5 pink pills ASAP and 5 pink pills 12 h later
Levlite®	5 pink pills ASAP and 5 pink pills 12 h later

Other options

Estrone b.i.d. × 5 days	5 mg
Danazol 400–600 mg STAT	12 h apart
Cu IUD (Copper-T 380A)	Within 5–7 days except in rape or STD 99% effective
Mifepristone (RU 486)	Not approved by FDA at this time

Mechanism of action of EC

EC may inhibit ovulation

Other mechanisms that contribute to the effectiveness of EC include:

- Thickening of cervical mucus to trap sperm
- Preventing fertilization of the egg by the sperm
- Interfering with tubal transport
- Preventing the zygote from implanting in the uterus
- Altering endometrial receptivity
- Interfering with the luteinizing hormone surge
- Inhibiting the function of the corpus luteum

ENDOCARDITIS

Prophylaxis

Ampicillin IV or IM @ 30 min prior to procedure	2 g
With gentamicin IV or IM	1.5 mg/kg
Then give amoxicillin p.o. 6 h later after initial dose	1.5 g
or Amp/Gen IV or IM dose again 8 h later	
For penicillin allergy – give vancomycin IV slowly over 1 h	1 g

ENDOCRINOLOGY

Conditions to differentiate	*Characteristics of conditions*
(1) Congenital adrenal hyperplasia (female pseudohermaphrodite) also known as congenital virilizing adrenal hyperplasia	Absence of palpable testes; fusion of labial folds; diagnosis: increase serum 17-OH progesterone; most common autosomal recessive trait chromosome 6
(2) Androgen insensitivity, (male pseudohermaphrodite), (complete testicular feminization)	Female phenotype despite male genotype (XY chromosomes); descent of testes – normal but abnormal position (remove at puberty); blind vaginal pouch; absent uterus and tubes; testosterone – normal or elevated. LH elevated, estrogen levels elevated, FSH normal or elevated; MIF (Müllerian inhibiting factor) present; diagnosis likely: breast development, primary amenorrhea, short vagina (blind pouch), absent uterus and cervix, scanty or absent pubic and axillary hair. (Some secondary sexual characteristics – breast development from aromatization of androgen to estrogen) • Remember: absence of uterus occurs only in two conditions – androgen insensitivity and Müllerian agenesis (Mayer–Rokitansky–Kustner–Hauser syndrome)
(3) Incomplete androgen insensitivity	Men with infertility – incidence can reach 40% due to azoospermia or oligospermia; Reifenstein syndrome – phallus large enough to assign sex as male at birth despite hypospadias, ambiguous genitalia; at puberty gynecomastia occurs but is minimal, karyotype is male (XY) – distinguishes it from other feminization syndromes (Klinefelter's)
(4) Gonadal dysgenesis	Genotype 45XO, 46XX or 46XY; usually do not develop ovaries, instead gonadal streaks; most common cause of primary amenorrhea – 50% due to random chromosomal disorder, deletion of all or part of X chromosome, genetic defect, rarely 17α-hydroxylase deficiency
(5) Swyer syndrome (bilateral dysgenesis of the testes)	Female phenotype but male genotype (46XY); primary amenorrhea; absence of secondary sexual characteristics; lack of production of MIF, testosterone and estrogen; adrenals produce androgens to explain hirsutism; estrogen and progestin therapy supports female secondary sexual development; Y-band areas where testes failed to develop need to be REMOVED
(6) Klinefelter syndrome	47XXY; non-disjunctional event at sex chromosomes occurring secondary to error in oogenesis or spermatogenesis; tall with azoospermia; gynecomastia in 1/3 of these patients; primary infertility
(7) Karyotype XYY	May appear normal male but TALL; aggressive personalities; fertile but female partners may have repeated pregnancy losses
(8) Perrault syndrome	Combination of XX gonadal dysgenesis and neurosensory deafness
(9) Turner syndrome	Female phenotype – 1/2500 live births; XO genotype – (part or all of one X chromosome missing – 45X, 45X/46XY, 45X/46X;Xq); cystic hygroma; gonadal dysgenesis with increase in FSH and LH; short stature and webbed neck; cardiac lesion – coarctation of aorta; know Turner stigmata – sexually immature female, short stature, web neck (cystic hygroma), wide spaced nipples, shield chest, streak gonads, coarctation of the aorta, renal anomalies, trouble hearing, high arched palate, normal IQ, low posterior hairline and pigmented nevi. What % abort in first trimester? 97%
(10) Noonan syndrome (male Turner)	Phenotype – appears like 'Turner' except genotype is XY and cardiac lesion is pulmonic stenosis instead of coarctation of aorta; fertile; autosomal dominant with variable expression

Continued

continued

(11) Kallman syndrome	Phenotype (5–7 times more frequent in males than females); genotype XX; insufficient pulsatile secretion of GnRH; low to absent FSH, LH (responds to gonadotropins but NOT Clomid); anosmia or hyposmia (inability or decreased ability to smell); amenorrhea; normal height for age; infantile sexual development (minimal or absent pubertal develop); three modes of transmission: (1) X-linked (most common) short arm of X; (2) autosomal dominant; or (3) autosomal recessive
(12) Mayer–Rokitansky–Kuster–Hauser syndrome (Müllerian agenesis)	Phenotype female; genotype female (XX); presents with primary amenorrhea (second most common cause) – 15%; absent uterus; normal secondary sexual characteristics; obtain IVP – incidence of coexisting renal anomalies is 50%; there is also an increased incidence of vertebral anomalies of 10%
(13) McCune–Albright syndrome	Triad of: (1) Café-au-lait spots; (2) fibrous dysplasia; and (3) cysts of skull and long bones. Isosexual precocious puberty – 40%; diagnose in neonatal period so treatment can be given so normal puberty ensues (testolactone); GnRH-independent precocious puberty; if left untreated → develops heterosexual precocious puberty from the adrenal androgens (most common cause, however, of pseudo-precocious puberty is an estrogen-secreting ovarian tumor)

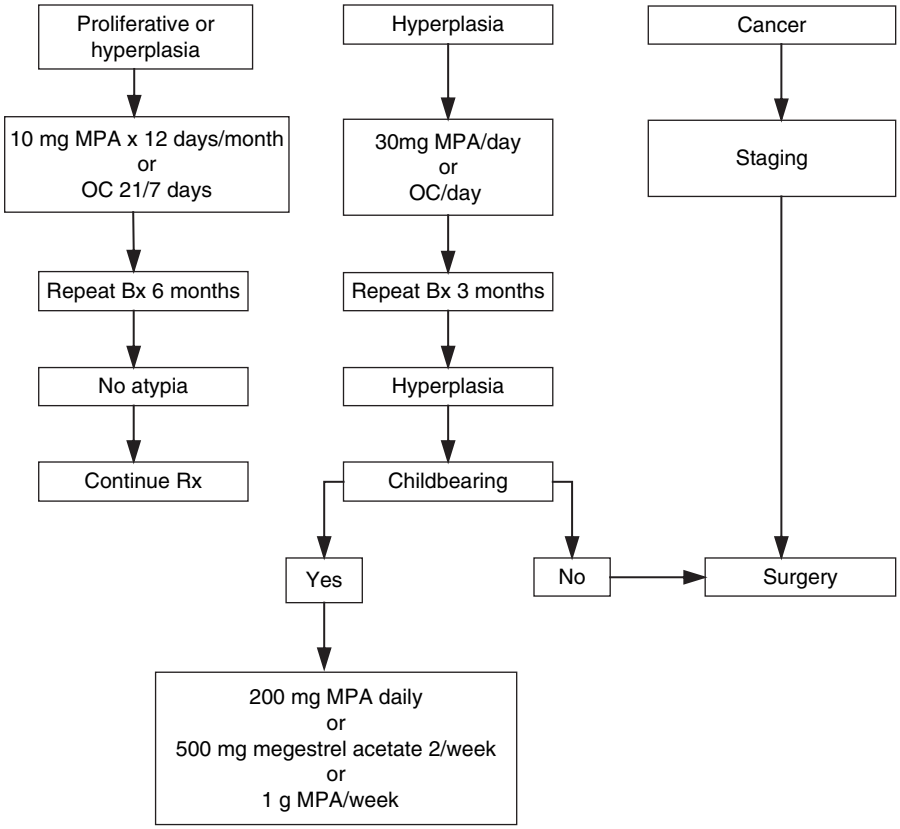
Pituitary control	Norepinephrine and dopamine → arcuate nucleus → GnRH pulses → anterior pituitary
	Dopamine → → → → → ··· → anterior pituitary
	Anterior pituitary produces FSH, LH, prolactin, TSH, GH, MSH, ACTH
	Posterior pituitary produces antidiuretic hormone, oxytocin and vasopressin

ENDOMETRIAL ABLATION

YAG laser (penetrates highly vascular areas). Successful	90%
Loop resectoscope – favor using which cutting loop?	8 mm
Why? Removes tissue in 1 cut – decreases risk of perforation	
Rollerball or barrel – blended current used, difficult to visualize cavity, can be done with irregular uterus	
Balloon therapy heated to over	60°C
Not effective if endo cavity	> 10 cm
ThermaChoice (Ethicon, Inc., Somerville, NJ)	
Cryoblation therapy – 5 mm probe used with ultrasound monitoring to help provide safe margin between cryozone and uterine serosal surface. Ablates with temperatures below –100°C *Advantages:* (1) Does not require painful distention of cavity (2) No danger of harmful fluid imbalance CryoGen (San Diego, CA)	
Scarring will occur by definition. Therefore, cornual hematometra or postablation tubal sterilization syndrome is not uncommon in patients having undergone endometrial ablation. (McCausland AM, McCausland VM. Frequency of symptomatic cornual hematometra and postablation tubal sterilization syndrome after total rollerball endometrial ablation: a 10-year follow-up. *Am J Obstet Gynecol* 2002;186:1274–83)	

ENDOMETRIAL BIOPSY

Interpreting the endometrial biopsy



	Atypia increases risk toward endometrial cancer	
Penny, Nickel, Dime, Quarter	'Penny' represents simple hyperplasia. Progression risk to cancer	1%
	'Nickel' represents complex hyperplasia without atypia	5%
	'Dime' represents simple hyperplasia with atypia	10%
	'Quarter' represents complex hyperplasia with atypia	25–30%
Most important in management of endometrial hyperplasia without atypia	(1) Patient age (2) Histological pattern of hyperplasia	
	Most frequent symptom of hyperplasia – abnormal vaginal bleeding	
Diagnosis	ENDOMETRIAL BIOPSY → if not diagnostic → hysteroscopy Most common with perimenopausal years	
Treatment	Hyperplasia	10 mg MPA × 12 days/month or OC 21/7 days 30 mg MPA/day or OC/day 200 mg MPA daily or 500 mg megestrel acetate 2/week or 1 g MPA/week
	If ≥ 40 years of age: Progestin with endometrial biopsy follow-up in 3–6 months or D&C + or – hysteroscopy (f/up pt < 50 years old) or hysterectomy (with indication)	
	If ≤ 40 years of age: D&C and/or hysteroscopy → f/up with endometrial biopsy in 3–6 months or progestin 10 mg daily × 10 days, OCPs, ovulation induction	

ENDOMETRIAL CANCER VS ATYPICAL HYPERPLASIA

Severe atypical hyperplasia (not CIS as terminology)
Anaplasia, irregularly shaped nuclei and LACK OF STROMAL INVASION. ATYPIA – most important predictor of malignant potential

Pathological diagnosis

Atypical hyperplasia – nuclear enlargement with clearing of center of nucleus with increased chromatin peripherally

Complex – marked crowding of glands but some stroma remains
Well-differentiated cancer – back to back glands with no stroma
UTERINE BLEEDING

Most important factors

(1) Patient age
(2) Histological pattern

Treatment

(1)	Moderate to severe atypical hyperplasia	Hysterectomy
(2)	Lesser lesions	Progestins
(3)	Postmenopausal	TAH with BS&O
(4)	Stage I, grade 1 carcinoma	TAH with BS&O

ACOG Practice Bulletin No. 65 (*Obstet Gynecol* 2006; 107: 952) states that "Most women with endometrial cancer should undergo systemic surgical staging, including pelvic washings, bilateral pelvic and paraaortic lymphadenectomy, and complete resection of all disease. Exceptions to this include young or perimenopausal women with grade 1 endometrioid adenocarcinoma associated with atypical endometrial hyperplasia and those at increased risk of mortality secondary to comorbidities."

ENDOMETRIAL CARCINOMA

> 1/2 myometrial invasion – what stage? invasive cancer
Treatment for invasive cancer is TAHBSO, nodes and irradiation

Radiation

Radiation to upper vagina (pTAHBSO nodes)	6000–7000 cGy
Radiation to whole pelvis (pTAHBSO nodes)	4500–5000 cGy

Give if grade I tumor with deep (> ½ thickness of myometrium)
Give if grade II tumor with superficial myometrial invasion (< ½)
Give if grade III tumor with any degree of myometrial invasion
Give if any grade tumor with lymph–vascular space or cx invasion

See Oncology for FIGO staging of Endometrial (Uterine) Cancer

Tumor grade and tumor depth

MOST IMPORTANT factors of lymph node metastasis

Grade

Stage I	3%
Stage II	9%
Stage III	18%

Depth

< ½	< 5%
≥ ½	25%

Local spread, tumor size and lymph–vascular spread also increase the risk of nodal metastasis
According to Harai Y, Takeshima N, Kato T, *et al*. Malignant potential of peritoneal cytology in endometrial cancer.*Obstet Gynecol* 2001;97:725–8, endometrial cancer cells found in peritoneal cavity usually disappear within short period of time and seem to have a low malignant potential. However, they theorize that only malignant cells from special cases, such as adnexal metastasis, may be capable of independent growth and are possibly associated with intraperitoneal recurrence. As above – depth, grade, + node status are much more important in determining outcome

What % of nodal mets have enlarged nodes? < 10%
So palpation is not adequate to decide @ lymphadenectomy

Mixed mesodermal tumors are what % of uterine malignancies?	1–2%
If spread beyond uterus there is only this survival rate	25%
If confined to uterus, the pelvic lymph node met rate is	15–20%

Radiation controversial (local rec but not overall)
Chemotherapy investigational (advanced or recurrent disease)

Low-grade stromal sarcoma	Rubbery, worm-like, yellowish-gray cord type tumors with minimal atypia with < 10 and usually < 5 mitosis per	10 HPF
	Late recurrences usually local in 5–25 years at rate of	50%
Treatment	TAHBSO or TLHBSO, postop radiation and progestins with megestrol acetate orally at a dose of	160 mg/day
	Women who undergo TLH, BSO, and staging typically are discharged the morning after surgery. Most patients return to normal daily activities within 2 weeks of the procedure. Post-op therapy is dependent on tumor grade, invasion, and/or metastasis. Metastasis may warrant whole-pelvic radiotherapy	
Risk factors for endometrial carcinoma	Nulliparous	2–3 ×
	Diabetes	1.3–2.8 ×
	Menopause > 52 years old	2–3 ×
	Overweight 21–50 lb	3 ×
	Overweight 50 lb	10 ×
	Unopposed estrogen	8 ×
	Chance that an office biopsy for endometrial cancer will underestimate tumor grade	20%
	Chance that an office Pap smear will detect endometrial cancer	40–50%
	One year of contraception will decrease a woman's risk of endometrial cancer (40–55) by	50%
	The presence of pyometra should be an alert to the high probability of malignancy	
	Shortest distance from serosa to tumor of endometrial cancer for which no treatment is needed	10 mm

ENDOMETRIAL POLYPS

Most asymptomatic	
What % patients with abnl bleeding will have polyps?	25%
Polyps that undergo malignant transformation	0.5%
Endometrial polyps that are solitary	80%
Endometrial polyps that are multiple	20%
What chromosome is common in *stromal* cell of polyp?	6p21
Curettage removes what % of polyps?	25%
So hysteroscopy is best therapy for polyps	

ENDOMETRIOSIS

Theories of etiology

(1) Sampson – retrograde flow

(2) Halbane – hematogenous/lymphatic

(3) Meyer – coelomic metaplasia

(4) Dmowski – decreased cellular immunity, embryonic cell rest

• Most likely mode of etiology is the decreased capacity of peritoneal macrophages to induce cytolysis of ectopic endometrial cells

Incidence

Endometriosis is thought to affect what percentage of all US women?	10%
Among symptomatic women the incidence of endometriosis increases dramatically according to the complaint;	
(1) Patients with infertility	30%
(2) Patients undergoing laparoscopy	45%
(3) Patients with chronic pevic pain	97%

Peritoneal fluid factors

(1) Increased peritoneal fluid in luteal phase

(2) Increased concentration in peritoneal fluid of:
- (a) Prostaglandins
- (b) Interleukins
- (c) Tumor necrosis factor-α
- (d) Transforming growth factor-β
- (e) Monocytic chemotactic protein-1

(3) Increased numbers of activated macrophages and natural killer cells

(4) Decreased concentration of interferon-γ

Suspect diagnosis

If subfertility, dysmenorrhea, dyspareunia, chronic pelvic pain

May be asymptomatic

Diagnosis

- Histology of laparoscopic biopsy. Insufficient data to support use of measurement of any peritoneal fluid growth factor, cytokine or angiogenic factor. Ultrasound is helpful in evaluation of endometriomas

Typical lesions of endometriosis are histologically negative in what % of cases? 24%

Histological biopsy:

Presence of

(1) Endometrial glands and/or
(2) Stroma
(3) Epithelium
(4) Hemosiderin-laden macrophages

In dysmenorrheic women with endometriosis, pain typically begins several days premenstrually and ends before menses is complete

Ultrasonography may reveal endometriomas as a "ground-glass" appearance

Laparoscopy has confirmed endomeriosis in patients diagnosed clinically on the basis of moderate to severe dysmenorrheal, suspicious physical/ultrasonographic pelvic findings,and an evaluation for infection including a sed rate and cervical cultures in what percent of patients? 78–87%

MRI has also been compared with laparoscopy for diagnostic accuracy

Staging

Revised American Society for Reproductive Medicine Chart ASRM

Classification based on

Appearance, size and depth
Presence and extent and type adnexal lesions
Degree of cul-de-sac obliteration
Endometriosis is found in what % of pts with pelvic pain, dysmenorrhea and/or dyspareunia? 10–15%
Endometriosis is found in what % of pts with infertility? 30–40%
Fulguration will decrease pain but ? concerning increasing fertility
Danazol and GnRH agonist decrease implants as much as 70–80%

Treatments

Medical

(1) First line : OCPs and/or NSAIDS
(2) Second line:
Continuous progestins (DMPA) – decidualization of endometriotic implants.
Continuous androgens (Danazol) – atrophy of implants
GnRH (Lupron®, Synarel®, Zoladex®) – decreases ovarian estradiol production so decreases growth of implants
Levonorgestrel-releasing intrauterine system
GnRH with add-back therapy – Premarin, Provera, Prempro™, norethindrone acetate or low-dose OCPs. Consider biphosphonates but bone loss due to GnRH agonist anologs may be mitigated by concomitant add-back estrogen therapy

Surgical

Cautery/laser of implants
Uterine suspension (especially if uterus is retroverted or if there is extensive disease in the cul-de sac)
Oophorectomy (even 1/10th ovary can preserve function and fertility)
Postop GnRH analog rx (good for superficial but much less for larger lesions)
Presacral neurectomy and/or uterosacral resection (good for midline pain relief @ 6–12 months but no evidence fertility is increased and possible complications include injury to middle sacral vessels, ureter or postop transient bladder dysfunction). Removal of the superior hypogastric plexus (presacral neurectomy) has not proved to be more effective in controlling pelvic pain than conservative surgery that only destroys endometrial implants; therefore presacral neurectomy is no longer advised, according to Candiani GB, *et al*. Presacral neurectomy for the treatment of pelvic pain associated with endometriosis: a controlled study. *Am J Obstet Gynecol*. 1992; 167: 100–3.
Hysterectomy with unilateral vs BS&O (start postop HRT immediately with estrogen AND progestin)

Mild – no evidence that surg vs med vs expect rx increases fertility
Moderate – pregnancy is successful after surgery rx in 60%
Severe – pregnancy is successful after surgery rx in only 35%
Highest pregnancy rate during first year after surgery (no HRT) < 20%
Recurrence rate after surgery (if it does – limited success rate) 44%

Rectovaginal nodules in excess of 3 cm have a high association – 11% with ureteral compromise in patients with endometriosis so an IVP would be mandated in nodules larger than this diameter. (Donnez J, Nisolle M, Squifflet J. Ureteral endometriosis: a complication of rectovaginal endometriotic (adenomyotic) nodules. *Fertil Steril* 2002; 77:32–7)

Pain relief

GnRH × 3 months or danazol × 6 months is effective
Continued treatment: add-back therapy with low-dose OCPs and consider biphosphonates
OCPs, MPA, Lupron (even without surgery and proof of diagnosis)
ERT okay after hysterectomy/BS&O
Severe endometriosis – medical therapy may not be sufficient
Expectant management is okay if patient is asymptomatic
Endometriosis may regress

ENDOMETRITIS

Reliable indicators of diagnosis are uterine tenderness and temperature > 38°C
Blood cultures are + < 7%
C-section with BV increases risk of endometritis 5 ×
Vaginal delivery with presence of meconium can be independent factor in development of endometritis. (Jazayeri A, Jazayeri MK, Sahinler M, *et al*. Is meconium passage a risk factor for maternal infection in term pregnancies? *Obstet Gynecol* 2002;99:548–52)

Bacteria

Bacteroides, Streptococcus and *E. coli*
Gentamicin/clindamycin NOT effective against *S. fecalis*
There is a rising prevalence of resistant bacteria to β-lactam/β-lactamase inhibitor combinations → Enterococcus

Treatment

Unasyn (ampicillin/sulbactam) 3 g stat then 1.5 q. 6 h or cefotetan 2 g and other regimens:

Regimen #1:	Clindamycin 500 mg IV q. 8 h
	Gentamicin 1.5 mg/kg IV q. 8 h
Regimen #2:	Clindamycin 900 mg IV q. 8 h
	Aztreonam 1–2 g IV q. 8 h
Regimen #3:	Metronidazole 500 mg IV q. 6 h
	Penicillin 5 mu IV q. 6 h
	Ampicillin 2 g IV q. 6 h
	Mefoxin 2 g IV q. 6 h
	Unasyn 3 g IV stat then 1.5 g IV q. 6 h

Outpatient endometritis

Symptoms

Intermenstrual bleeding

Bleeding at inappropriate times while taking OCPs. Recent onset of dyspareunia. Vague, crampy lower abdominal pain

Treatment

Ceftriaxone 250 mg IM plus doxycycline 100 mg b.i.d. × 14 days or ofloxacin 400 mg p.o. b.i.d. × 14 days plus either clindamycin 450 mg p.o. q.i.d. × 14 days or metronidazole 500 mg p.o. b.i.d. × 14 days

ENGAGEMENT

BPD at level of plane of inlet or presenting part at ischial spines
Inlet is limiting factor of pelvis 10–11 cm

ENTEROCELE REPAIRS

Moscowitz	Superficial bites to *encircle* the pouch of Douglas (cul-de-sac)
Halbane	Posterior vaginal wall to anterior rectal wall creates shelf and obliterates cul-de-sac
Vaginal	Enterocele sac dissected free. Suture placed @ neck above levator hiatus. Additional bites (purse string) above initial suture incorporating uterosacral ligaments

ENZYME DISORDERS

Important deficiencies to remember:

Deficiency of 21-hydroxylase leads to elevation of	progesterone
Deficiency of 17-hydroxylase leads to elevation of	pregnenolone
Deficiency of aromatase leads to elevation of	androstenedione

EPIDEMIOLOGY

Randomized clinical trial	Greatest scientific value – the Gold Standard
Observational studies	
Cohort study	Observational study of large numbers over a long period
Case–control study	Highly efficient but prone to numerous bias
Cross-sectional study	
Case-series report	
Case report	
Sensitivity	The proportion of truly diseased persons in the screened population who are identified by the screening test
Specificity	The proportion of truly non-diseased persons who are so identified by the screening test

EPIDURAL

Hypotension is most common complication. Dural puncture (PDPH) complicates	1%

Definition of maternal decrease in B/P with epidural is loss of sympathetic tone with dilatation of resistance and capacitance vessels. Maintain B/P with vasoconstriction of *upper* body

Definition is decrease of arterial mean B/P of ? less than pre-epidural	20–30 mmHg
or systolic B/P less than	100 mmHg
Epidural needle passes through the skin, supraspinous ligament, intraspinous ligament and ligamentum flavum. The epidural space is between the flavum and the dura. Onset takes	8–20 min

- Would you place an epidural in a patient who is less than 4 cm dilated? Some studies seemingly show that placing an epidural too early can slow or even arrest the labor. However, more recent studies contradict this practice

It is certainly feasible and reasonable to administer an epidural earlier if narcotics are not relieving the patient's pain. Some patients seem to even benefit from the relaxation and relief of pain that is provided by the epidural in such a way that the labor will sometimes progress more quickly

Dr Cynthia Wong of Northwestern University was lead author on a major study showing no benefit of delaying epidural analgesia in women with spontaneous labor (Wong CA, Scavone BM, Peaceman AM,

et al. The risk of cesarean delivery with neuraxial analgesia given early versus late in labor. *N Engl J Med* 2005; 352: 655–65)
Present recommendations to placing an epidural in a laboring patient is that there should no longer be an arbitrary degree of cervical dilation before such a decision is made.

Although persistent posterior occiput position results in certain intrapartum complications, its incidence is not increased by the use of epidural analgesia. (Fitzpatrick M, McQuillan K, O'Herlihy C. Influence of persistent occiput posterior position on delivery outcome *Obstet Gynecol* 2001;98:1027–31)

Dosages

(1) Marcaine® 0.75% (bupivacaine) 20 cc
Normal saline 30 cc
Fentanyl 250 µg/5 ml (Sublimaze) 10 c
60 cc

Use test dose of 3 cc (epinephrine) solution. Then top off dose 3–6 cc followed by 6 cc/h rate. Use this formula for expected long labors and less motor blockade

(2) Lidocaine 2% with Epi (1 : 200 000) 20 cc
8.4% Sodium bicarbonate (50 ml vial) 2 cc
Fentanyl 100 µg/2 ml (Sublimaze) 2 cc
24 cc

Use this solution for more of a sensory blockade for postpartum tubals, C-sections and expected operative deliveries. Same dosages as above except C-sections then inject 3 cc test dose, slow 12 cc bolus and add 5 more cc if T10 level not reached after few minutes

(3) Xylocaine 2% (10 ml vial × 2) 20 cc
8.4% Sodium bicarbonate (50 ml vial) 2 cc
Fentanyl 100 µg/2 ml (Sublimaze) 2 cc
24 cc

Use this formula in same type situations as in #2, but with the exception of patient having PIH or condition that contradicts the use of epinephrine

Combined spinal/epidural (CSE) anesthesia because of the advent of pencil-point spinal needles, as compared with 'traditional' epidural, has reduced the incidence of postdural puncture headache and allowed the 'walking epidural'. However, some patients have complained of less pain relief and also some increased respiratory depression. Protocols for either should be established

Back labor relief

See Sterile water papules

Epidural analgesia – potential complications

Complication	*Incidence*	*Presumed etiology, signs or symptoms*	*Treatment*
Maternal hypotension (most common)	Approx. 22%	Sympathetic blockade leading to vasodilatation, vascular pooling, diminished venous return	Volume preload with 500–1000 ml or balanced salt solution; left uterine displacement; avoid supine position; ephedrine if additional therapy required
Maternal central nervous (high spinal)	0.06%	Unintentional subarachnoid injection of local anesthetic; severe hypotension, profound bradycardia, respiratory compromise	Support airway; intubate; 100% O_2; intravenous fluids; vasopressors
Maternal central nervous system toxicity	0.03–0.5%	Intravascular injection of local anesthetic leading to slurred speech, dizziness, tinnitus, metallic taste, oral paresthesias, syncope, seizures, coma, potential cardiopulmonary arrest (bupivacaine cardiotoxicity)	Oxygenate; intubate if necessary; treat seizures with thiopental or diazepam; uterine displacement; IV fluids; vasopressors; CPR if needed
Maternal temperature elevation	Directly proportional to duration of epidural	Sympathetic nervous system blockade thought to inhibit heat loss	Antibiotics if intrauterine infection suspected (temp. usually $\geq 38^{\circ}C$)
Post-dural puncture headache	1–2%	Loss of cerebrospinal fluid (CSF) through puncture site with decreased CSF pressure. Onset several hours to days after puncture; headache on sitting or standing; relief when horizontal	Conservative: rest in horizontal position; hydrate; analgesics; caffeine; if no relief, epidural blood patch
Transient rise in fetal transcutaneous pCO_2	Unknown	May be associated with either decreased uteroplacental perfusion or maternal hyperventilation due to pain, anxiety	None necessary
Fetal abnormal heart rate pattern	Dependent on hypotension, maternal position, contraction pattern	Fall in maternal blood pressure leading to uteroplacental insufficiency; exacerbated by aortocaval compression or uterine hyperstimulation	Correction of hypotension by hydration; left uterine displacement; avoid supine position; avoid uterine hyperstimulation

Epidural analgesia – some neurological complications reported with regional analgesia

Cause		*Lesion or event*	*Sequelae*
Needle trauma during spinal or epidural		Nerve root lesion	Numbness and paresthesia
Vertebral stenosis		Nerve root lesion	
Epidural hematoma, abscess or tumor		Space-occupying lesion Nerve root lesion	
Coagulopathy		Space-occupying lesion Nerve root lesion	
Vasculitis		Cord ischemia	Paralysis, anterior spinal artery syndrome
Hypotension Epinephrine			
Infection		Chronic arachnoiditis	Low back pain, cauda equina syndrome
Error			
	Wrong agent	Pentothal, phenol, iodine-containing skin disinfectant, radiographic contrast media, detergents	
	Wrong solution	Low pH or hypo-osmotic solution, preservatives and antioxidants from multidose vials resulting in cord ischemia or arachnoiditis	
	Wrong volume	Total spinal, spinal fluid leakage and displacement	Reversible total paralysis, headache, diplopia from paresis of IVth and VIth nerves

EPILEPSY

Incidence of serious malformations (attempt monotherapy)	3%
Decrease risk of NTD with how much folic acid?	4 mg
May discontinue antiepileptic drugs before or during pregnancy if seizures have been well controlled	2 years
Serious malformations are increased with one AED (anti-epileptic drug)	3%
With two AEDs	5%
With three AEDs	10%
With four AEDs	20%

Management

Preconception

(1) Folic acid 4 mg daily at least 1 month prior to conception
(2) Compliance in taking AED must be stressed. Inform that there is 2.4 × or more risk of congenital malformations but worse outcome if seizure occurs causing hypoxia. (Most epileptic women do have normal babies)
(3) Try to decrease the number of AEDs

Antenatal

(1) Obtain AED levels every 3–4 weeks or more frequent if seizures, drug toxicity or toxicity develop
(2) Raise doses if necessary to maintain effective anticonvulsant activity (use 30 mg rather than 100 mg phenytoin capsules)
(3) If seizure control is not maintained and anticonvulsant dose has been increased until toxic effects are apparent, add additional anticonvulsant medication. Prescribe folic acid 1 mg and follow CBC. (Folic acid deficiency is common)

Early management

Treat nausea and vomiting early so patient will be able to take AED

Second trimester

(1) MSAFP; (2) Level II ultrasound; (3) Review seizure frequency;
(4) Consider amniocentesis

Third trimester

(1) Begin NST every week at 34 weeks' gestation
(2) Begin daily fetal kick count movements
(3) Vitamin K therapy to begin at 32 weeks' gestation – 10 mg p.o. daily at 32–36 weeks' gestation, then 20 mg p.o. daily at 36 weeks until delivery; 10 mg IV during labor

Postpartum

(1) Decreased AEDs
(2) Check patient every 3–4 weeks after delivery
(3) Breastfeeding is not contraindicated while mother is on AEDs

EPISIOTOMY

If it does not heal think about what disease? Crohn's

Determining episiotomy degree – vaginal wall/mucosa and skin of perineum — First degree
Superficial transverse perineal muscle — Second degree
Rectal sphincter — Third degree
Rectal mucosa — Fourth degree

ACOG Practice Bulletin No. 71 (*Obstet Gynecol* 2006; 107: 956–62) recommends that episiotomy be restricted as much as possible

Evidence supports that restricted use of episiotomy is preferable to routine use
Median episiotomy is associated with higher rates of injury to the anal sphincter and rectum than is mediolateral episiotomy, whereas mediolateral episiotomy may be preferable to median episiotomy in selected cases. Routine episiotomy does not prevent pelvic floor damage leading to incontinence
Routine episiotomy is no longer indicated and should be used only in selective cases (Goldberg J, Holtz D, Hyslop J, *et al*. Has the use of routine episiotomy decreased? Examination of episiotomy rates from 1983 to 2000. *Obstet Gynecol* 2002;99:395–400)

Prenatal perineal massage after 34 weeks of pregnancy reduces the likelihood of episiotomy by 15%

Perineal massage also reduced reported pain after childbirth and also had a reduction in the incidence of trauma by 9%

ERYTHROBLASTOSIS FETALIS

If pregnancy complicated by Rh, ab titers should be determined at prenatal visit — First
20 weeks
and thereafter every — 4 weeks

No intervention if albumin titer is < 1 : 16
or indirect antiglobulin titer is < 1 : 32
If pt has had prior affected pregnancy, no ab titers needed and amnio or percutaneous umbilical cord sampling to be done q. 4–8 weeks earlier than prior gestational age that sign morbidity occurred

Amnio every 3–4 weeks if in Zone — I
Amnio every 1–4 weeks if in Zone — II
Severe hemolytic disease if in Zone — III
Zone III – high probability of fetal death in how many days? 7–10

Antibodies that produce E. fetalis: Duffy, Kell, Kidd and Lutheran ('dies and kills'), Lewis – does not cause E. fetalis ('Lewis lives')

ESOPHAGEAL CANDIDIASIS

+HIV, CD4 count < 200/mm^3, CD4% < 14 (active AIDS)
Treat with Retrovir® (zidovudine) and ketoconazole p.o. b.i.d. 200 mg
or Diflucan® to prevent systemic disease

ESSURE

Essure is a relatively new hysteroscopic sterilization method replacing the silastic plugs used in the past. The innermost layer are fibers that elicit a benign localized tissue in growth that occludes the tubal lumen. The patient must not be allergic to nickel, as the outer coil is made of a nickel–titanium alloy (nitinol). Some patients, especially if immunosuppressed, may take up to 3 months for their tubes to occlude.

Helpful hints;

(1) Use contraception for 3 months prior, to thin endometrium
(2) Schedule procedure within the first 2 weeks of cycle
(3) Patients should take NSAID @ 1 hour prior to procedure
(4) IV sedation (ketorolac 30 mg) given for procedure but paracervical block in office works just as well
(5) Flush the uterus as necessary for clots or debris and aspirate
(6) Confirm visibility of both ostia and start with tube that appears most difficult
(7) Use 2–3 liters of warmed saline to enhance uterine dilation and tubal canulation. Avoid uterine overdistention
(8) Inadequate uterine distention due to patulous cervix can be overcome by using another tenaculum or twisting cervix 45 degrees
(9) The microinsert is inserted into the tubal ostia at the level of the black marker
(10) The delivery catheter is retracted. The notch at the opening of the tubal ostia shows correct placement
(11) The delivery wire is retracted from the microinsert

ESTRING

Soft, flexible, silicone ring insert placed into upper part of vagina lasting	3 months
Releases how much estrogen?	7.5 µg/24 h
Improve vaginal and urinary symptoms and mucosal appearance without provoking bleeding	

ESTROGEN

Measured in picograms/ml	pg/ml
Premarin 1.25 = Estrace® 2 mg = ethinylestradiol 20 µg	

ESTROGEN REPLACEMENT THERAPY

	% of postmenopausal women using ERT	16–20%
	What % of these women discontinue ERT use after 1 year?	50%
	Unopposed ERT leads to increased incidence of endometrial hyperplasia of	4–8 ×
Transdermals	% of women using transdermals experience adverse skin reactions:	
	ETOH reservoir (Estraderm patch)	17%
	Adhesive hormone matrix (Alora®, Climara®, Vivelle®, etc.)	5–8%
Natural isoflavonic phytoestrogen	Found in high concentrations in soy products. Exert only minimal to no influence on plasma leptin concentrations. Minimal to no influence on endometrial or vaginal epithelial changes. Minimal amelioration of vasomotor symptoms (Phipps WR, Wangen KE, Duncan AM, *et al*. Lack of effect of isoflavonic phytoestrogen intake on leptin concentrations in premenopausal and postmenopausal women. *Fertil Steril* 2001;75:1059–64)	

Hip fractures	ERT reduces fractures as compared to no HRT by	50%
Lipid profile	Improves (increases HDL, decreases LDL, affects endothelial vasculature)	
Others	Decreases osteoporosis, colon cancer and possibly Alzheimer's. There may be slightly increased risks during first 12–24 months in patients who already have heart disease. (HERS trial)	
ERT and endometrial cancer	In absence of estrogen replacement therapy	
	Well diff endometrioid type with superf inv – risk of persistence	5%
	Mod diff (up to half myometrial inv) renders risk of	10–15%
	Poorly diff (> ½ myometrial inv) renders risk of	40–50%
	Non-endometrioid type increases risk well over	50%
`	If FREE of tumor → ERT cannot result in recurrence. If estrogen-dependent neoplasm is present → it will eventually recur. If estrogen-dependent neoplasm is present → ERT may result in earlier recurrence So, assess risks, discuss information with patient including the alternatives, risks and benefits. Use ERT if appropriate after cancer	
ERT and breast cancer	No data to indicate increased risk of recurrent breast cancer in postmenopausal women receiving ERT. Consider ERT but use with caution. Weigh possible benefits. Consult patient's oncologist Extensive randomized, prospective trials are needed. NIH study demonstrated that there were actually fewer breast cancer recurrences when ERT was utilized versus when not used. WHI study demonstrated a possible increase in breast cancer with long-term use	

ETHICS

Terms to be familiar with

Autonomy – self-rule
Beneficence – promote well-being and avoid doing harm
Justice – treat equally
Informed consent – adequate disclosure. Nature of intervention, risks versus benefits, alternatives with risks vs benefits
Honesty – complete and truthful information
Confidentiality – duty to respect patient's privacy. Duty to maintain confidentiality takes precedence over other obligations

EVISCERATION

	Frequency of fascial dehiscence is between	0.3–3%
	Occurs more commonly with vertical than with transverse incisions	
	Mortality rate associated with evisceration is	10–35%
	Usually occurs on what days postop?	5–14
Abdominal evisceration	*Treatment*	
	Sterile moist towels, abdominal binder and narcotic cough suppressant → to OR → explore and resect any compromised gut → #1 PDS to close fascia → excise skin and subcutaneous fat → irrigate → close skin primarily → NG tube → TPN	
Vaginal evisceration	Rare	
	Treatment	
	Warm, moist diaper → to OR → withdraw viscera → inspect for necrosis and pelvic supporting tissues → suspend → prevent enterocele	

EXAMINATION SCHEDULES

Age (years)	*Complete physical**	*Screening examinations*	*Screening tests*	*Immunizations*
13–15	Initial visit – need not include pelvic	Blood pressure, weight, body image	Informational and introductory	HPV vaccine if not already done 8–12
20–39	Every 5 years	Blood pressure annually	Pap smear every 1–3 years**	Diphtheria and tetanus every 10 years
		Breast every 1–3 years	Serum cholesterol every 5 years	Rubella once if necessary
		Pelvic every 1–3 years	Rubella titer at age 20	
			Mammography at age 35	
40–49	Every 3 years	Blood pressure annually	Pap smear every 1–3 years	Diphtheria and tetanus every 10 years
		Breast annually	Mammography every year	
		Pelvic annually	Occult blood every year	
			Serum cholesterol every 5 years	
			Tonometry	
50–69	Every 2 years	Blood pressure annually	Mammography annually	Influenza annually
		Breast annually	Occult blood annually	Pneumococcal vaccine at 65
		Pelvic annually	Pap smear every 3 years	Diphtheria and tetanus every 10 years
		Proctosigmoid-oscopy every 3 years	Serum cholesterol every 5 years	
			Tonometry every 2 years	
70 & up	Annually	Proctosigmoid-oscopy every 3 years	Mammography annually	Influenza annually
			Occult blood annually	
			Tonometry annually	

*Includes health risk and hearing assessment with education about exercise, nutrition, stress management, smoking, alcohol and drug abuse, seat belt use, repeated excessive exposure to the sun and osteoporosis
**After two consecutive negative results

EXERCISE DURING PREGNANCY

Major questions

(1) Increase in non-working tissues produces vasoconstriction. (Does pregnant uterus have same vasomotor mechanism?)

(2) Increased body temperature – shift of blood volume from non-working tissues (splanchnic + renal) to working tissues (muscle + skin)

Recommendations

(1) Mild to moderate exercise does not have to be curtailed during pregnancy

(2) Avoid hyperthermia (not in excess of 102°F). Fetus is at 1°C warmer than mother. Avoid more than 10 min in sauna or hot tub

(3) Avoid difficult activities (skiing or horseback riding) that require increased coordination secondary to increased production of relaxins that cause increases in uncoordination and increases the chance of accident

(4) Decrease overall performance to about 50% of non-pregnant levels in third trimester

(5) Non-weight bearing exercise can be maintained at higher levels throughout pregnancy

(6) Avoid supine position during exercise after first trimester. (Decreases cardiac output in that position)

(7) Maintain adequate carbohydrate diet – avoid hypoglycemia

(8) Increase heat dissipation (appropriate hydration, clothing and avoidance of adverse environmental conditions)

Relative contraindications to exercise

(1) Incompetent cervix

(2) Twins after 24 weeks' gestation
Multiple gestation > 24 weeks or when fundal height is term

(3) History of PTL

(4) Known placenta previa after second trimester or if bleeding at any trimester

(5) PROM

(6) History of PIH

(7) Essential hypertension

(8) Certain cardiac diseases

(9) History of IUGR

(10) Cardiac arrhythmia

(11) Asthma or COPD

(12) Type II diabetes mellitus

(13) Breech presentation during third trimester

(14) Previous sedentary lifestyle

(15) Underweight

(16) Obesity

(17) Iron-deficiency anemia

(18) Recurrent spontaneous abortion of unknown origin (first trimester)

Heart rate of pregnant woman should not exceed	140 b.p.m
Target heart rate formula:	
Non-pregnant	(220 – age) × 0.8
Pregnant	(220 – age) × 0.7

Exercise programs

(1) Squatting positions – decrease incidence of forceps and shorten secondary stage labor
(2) Pelvic floor exercises – may benefit postpartum for muscles to return to the pre-pregnancy condition
(3) Toning exercises – helps maintain proper posture and prevents lower back pain
(4) Semi-recumbent/sitting – not supine exercises to avoid aortocaval compression syndrome
(5) Recreational and sports activities – okay, but orthopedic risk
(6) Jogging – do not *initiate* after pregnancy. Limit to about 2 miles per day to prevent hyperthermia and dehydration. 4–6 mile brisk walk. Pay attention to terrain and wear shoes with proper support
(7) Aerobics – consistent with jogging recommendations
    (a) Programs should have a scientific basis
    (b) Avoid overextension + exercises on back
    (c) Avoid hard surfaces and limit reps to 10
    (d) Warm-up and cool-down should be done gradually

BICYCLING

(1) Program can be started during pregnancy
(2) Stationary cycle is preferable to standard bicycling because of weight and balance changes during pregnancy
(3) Bicycling should be avoided out of doors during high temperatures and high pollution levels

SWIMMING – may be the best

(1) Respiratory changes may make swimming difficult in late pregnancy
(2) Calisthenic exercise in water is encouraged for maintenance of strength and flexibility
(3) Avoid water that is too cold or too hot
(4) Jacuzzi temps > 38.5°C should be avoided

SCUBA DIVING – **avoid**

Fetus may be at greater risk than mother (decompression sickness, hyperoxia, hypoxia, hypercapnia and asphyxia)

MUSCULAR STRENGTH & ENDURANCE – increases chance of transient hypertension (Valsalva maneuver)

(1) Training with light weights can cautiously continue in pregnancy
(2) Avoid heavy resistance on weight machines
(3) Avoid use of heavy free weights. Use close spotter for light free weights
(4) Avoid Valsalva maneuver – use proper breathing

CONTACT SPORTS – avoid after first trimester

Main points to remember

(1) Can continue regular exercise (at least 3 times per week)
(2) Avoid exercise in the supine position after the first trimester
(3) Modify intensity according to maternal symptoms
(4) Avoid even mild abdominal trauma
(5) Ensure adequate diet (normal pregnancy requires 300 kcal/day)
(6) Augment heat dissipation with adequate hydration
(7) Prepregnancy exercise routines should be resumed gradually
(8) AVOID → hyperthermia (102°F) such as hot tubs and saunas. Difficult activities that require coordination. SCUBA
(9) Check contraindications if high-risk or abnormal pregnancy

EXTERNAL CEPHALIC VERSION

Usually successful in what % of patients? 65–70%
Increased success rate with terbutaline especially with nulliparas up to 27–52%
SCORING SYSTEM (using parity, dilatation, EFW, placenta position and station)

	Score		
Factors	*0*	*1*	*2*
Parity	0	1	2
Dilatation	≥ 3 cm	1–2 cm	0 cm
Estimated weight	< 2500 g	2500–3500 g	> 3500 g
Placenta	Anterior	Posterior	Lat/fundal
Station	≤ –1	–2	≥ –3

Not a suitable candidate if ≤ 4
Ideal candidate if score is ≥ 8

• *Criteria for ECV*
Completion of 36 weeks
Prefer terbutaline especially with nulligravids
An ultrasound for presentation before and after
Reactive NST or BPP
Rh therapy if needed
Scoring system (parity, dilatation, EFW, placenta, station) ≥ 8
INFORMED CONSENT

Contraindications

Multiple pregnancy
IUGR
Third-trimester bleeding
Abnormal AFV
Uterine malformation
Placenta previa
Uncontrolled hypertension
Maternal cardiac condition
PIH
Non-reassuring FHR
Major fetal anomaly

Decrease success rate if

Low AFV, obesity, anterior placental location, cervical dilatation, low breech into pelvis, anterior or posterior position of fetal spine

External version of breech presentation or transverse lie

The following patients should be excluded from consideration for external version of breech presentation:

(1) Any patient in whom a tocolysis is contraindicated
(2) Any patient in whom there is a high index of suspicion for utero placenta insufficiency and fetal distress
(3) Premature labor, PROM or very dilated cervix
(4) Multiple gestation
(5) Third-trimester bleed, suspected abruption, placenta previa
(6) Gestational age less than 36 weeks or estimated fetal weight greater than 3800 g
(7) Previous uterine surgery

Protocol

(1) The risk/benefit should be discussed with the patient in advance. The patient should be aware of the risk of transient fetal bradycardia during the procedure and the occasional (less than 5%) need for urgent Cesarean. A routine hospital consent form will be signed at time of version
(2) The patient's prenatal records, including lab work should be in Labor & Delivery
(3) The Labor & Delivery staff and the OB Anesthesia staff should be notified of the date and time of the attempted version and enough staff should be available at the time of the version, if a Cesarean becomes necessary
(4) The patient should be NPO after midnight
(5) On arrival to Labor & Delivery, a sonogram should be performed to determine:
    (a) Fetal position and type of breech
    (b) Estimated fetal weight
    (c) Head extension and nuchal cord if possible
    (d) Anomalies if possible
    (e) Placenta location
    (f) Amniotic fluid volume

If contraindications to version are determined the procedure should be canceled

(6) A non-stress test should be performed and evaluated prior to the procedure
(7) A deep-vein open IV should be inserted and a type and hold drawn
(8) A tocolysis (terbutaline or $MgSO_4$) may be started at the lowest dose, as per protocol (see individual tocolysis protocols). Tocolysis may not be necessary in some patients
(9) The version should be attempted as soon as the tocolytic is effective if infusion. This may be 5 min for subcutaneous terbutaline or 30 min for $MgSO_4$
(10) The version should be done with an assistant who can provide intermittent fetal heart rate monitoring and sonograph during the procedure
(11) After the attempted version, continue fetal monitoring for 1 h. The patient needs a reactive NST prior to discharge
(12) If the patient is Rh negative, a Kleihauer-Betke test should be drawn and the appropriate RhoGAM should be administered prior to discharge

FACE PRESENTATION

Chin (mentum)	Mentum posterior – labor is impeded. MP can convert spontaneously to anterior even late in labor
Etiology	Enlargement of neck (fetal goiter), coils of cord @ the neck may cause extension, anencephalic fetus, pelvic contraction increased, macrosomia increased, pendulous abdomen in multiparas (permits fetal back to sag promoting cervical extension)
Diagnosis	Vaginal exam X-ray sometimes needed as can be confused with breech
Treatment	May attempt delivery of mentum anterior, however C-section is frequently preferred due to association with pelvic contraction C-SECTION mentum posteriors – rotations are obsolete

FATTY LIVER OF PREGNANCY

Serum bilirubin usually	< 10
Aminotransferases elevated but usually	< 300
HALLMARKS with symptoms are glucose	< 50 mg/dl
and WBCs often above	30 000

Symptoms: anorexia, headache, fatigue, jaundice, vomiting or abdominal pain (especially RUQ or diffuse to back)
Labs: S. bilirubin usually < 10 mg/dl unless hemolysis or renal fails. PT and PTT prolonged. Aminotransferases increased but usually < 300 u/l. Glucose < 50 mg/dl and WBCs > 30 000

FECAL INCONTINENCE

'Dovetail sign' with decreased sphincter tone

Patients with third- or fourth-degree tears – what % experience urgency and incontinence?	50%
What % have pudendal neuropathy?	60%
If there is pudendal neuropathy, anal sphincteroplasty is success	80%

Diagnosis: ? Transanal ultrasonography → pudendal nerve motor latency testing… → if + and no external anal defect → treat with biofeedback or dynamic anal graciloplasty p.r.n. If negative and pudendal neuropathy → then do an anal sphincteroplasty

FERTILIZATION/GROWTH

Morula	2–3 days
Blastocyst	4–5 days
Fertilized ovum reaches uterus in	5–6 days
Implantation	6–7 days
Trophoblastic venous sinuses form	9–11 days
Cardiovascular system	21 days
Earliest morph indicator of sex appear	8–9 weeks
Oogenesis begins	11–12 weeks

FETAL ALCOHOL SYNDROME

Incidence	1/1000

Diagnosis (diagnose if two of the following are present and suspect if one is present):
(1) Growth restriction
(2) Facial abnormalities (low-set ears, thin upper lip, midfacial hypoplasia)
(3) CNS impairment (microcephaly, ADD, MR)
(4) Other physical defects:
Cardiac – VSD (most common), increased ASD, abnormality of GV, tetralogy of Fallot

FETAL ASSESSMENT

When to start	Start at-risk patients	32–34 weeks
How frequent	Start high-risk patients	26–28 weeks
	At risk	weekly
	At high risk	biweekly
How reassuring	Very reassuring in that NSTs neg predictive value	99.8%
	Neg predictive value for CST, BPP and mod. BPP	99.9%
Kick counts	Fetal kick counts – how many movements in 2 h is normal?	10
NST	NST – 2 or > FHR accelerations (15 b.p.m. + lasts 15 s) in period of	20 min
	Non-reactive NST – lacks suff FHR accels in > how many min?	40 min
	24–28 weeks' gestation – may not be reactive in this %	50%
	28–32 weeks' gestation – may not be reactive in this %	15%
CST	OCT or contraction stress test = three 40 s contractions in	10 min
	POSITIVE contraction stress test = decels with ? contractions	50% or >
BPP	Modified BPP (normal) = reactive NST and AFI	> 5 cm
	(abnormal) = non-reactive NST and AFI	< 5 cm
UADV	Umbilical artery Doppler velocimetry	
	Normal	high velocity diastolic flow
	Extreme IUGR	absent or even reversed flow
	This test is good for IUGR, post-term gestation, DM, SLE, antiphospholipid syndrome	
	In a high-risk obstetric population undergoing antepartum fetal testing, perinatal mortality rate is	12/1000

• *FHR monitoring criteria*

No differences have been seen in patients who were monitored electronically vs intermittent Doppler auscultation. Depends on the standard of care for the community

Fetal bradycardias and prolonged decelerations are 2 distinct entities; the first usually does not warrant immediate intervention

Fetal scalp stimulation to assess fetal status should be done during periods of FHR baseline

FETAL CIRCULATION

Umbilical vein → ductus venosus
Portal vein (right and left) → join at this point level for abdominal circumference measurement
Inferior vena cava → to right atrium
Foramen ovale → to left atrium
Left and right ventricles → ductus arteriosus to aorta (indomethacin can cause premature closure)
Hypogastric arteries → to umbilical arteries and become ligamentum teres

FETAL DEMISE

Incidence	Fetal deaths per what definition of total births?	1000
	Reporting requirements vary. Most states	20 weeks or >
Diagnosis	Ultrasound (sometimes FRH is interpreted as mom's rate)	
Follow for DIC	DIC = ¼ females with dead fetus develop this after 4 weeks	
	Fibrinogen to be measured weekly (normal)	450 mg/dl
	DIC	< 100 mg/dl
Delivery	PGE_2 20 mg every 4 h with Zofran® (ondansetron) 8 mg every 6 h or Phenergan 25 mg every 6 h	
	Use laminaria or Pitocin p.r.n. Electrolytes every 24 h	

Cause

Request autopsy, placental culture/stains (*Listeria*), karyotype (use blood, skin, fascia, patellar tendon, amnio), Kleihauer–Betke, VDRL, antibody screen, tox titers, CMV, lupus, glucose, thyroid, viral cultures, bacterial cultures. Obtain photographs and X-rays

Labs

Cause and tests to rule out

- Drug use – get blood and urine for toxicology
- Abruption – Kleihauer–Betke stain
- Diabetes – $HgbA_{1C}$, glucose
- VDRL – to rule out syphilis
- Viral titers – Rubella, parvovirus
- Rh and antibody screen
- Lupus anticoagulant, anticardiolipin antibody

'DAD VV tails real low'

FETAL FIBRONECTIN TEST

Criteria

(1)	Amniotic membranes intact	
(2)	Cervical dilatation is minimal	< 3 cm
(3)	Sampling must be between 24 weeks, 0 days and 34 weeks, 6 days	
Negative tests help rule out imminent delivery within		2 weeks
Not for general OB population. Results from lab must be timely		

FETAL WEIGHT

Estimated	
20 weeks	500 g
28 weeks	1000 g
32 weeks	1600 g
36 weeks	2500 g
40 weeks	3500 g

FEVER (POSTOP)

Definition

≥ 38°C (100.4°F) × 2 @ 6 h apart > 24 h after surgery or 38.7°C (101.5°F) anytime

Infection	20%
Non-infection (90% microatelectasis)	80%

Increased risk

Increased risk of postop fever if EBL	> 1500 cc
or operating time is over	2 h
Thickness of SC is highest risk factor for wound infection	
If patient is over 200 lb, risk of infection increases	8 ×

Causes

Remember the 5 Ws

(1) Atelectasis – most common cause of fever
Diagnosis – presents with fever, tachypnea, tachycardia
Treatment – spontaneously resolves (usually) by 3–5 days

***W**ind*

Incentive spirometry. If does not clear: chest PT, IPPB, aerosol or intermittent/continuous and airway pressure

(2) Pneumonia – commonly associated with atelectasis
Infection usually begins in the collapsed area of lungs
Diagnosis – presents with high fever, cough, dyspnea, tachypnea, increased sputum production and purulent, coarse rales, toxic
Treatment – same as atelectasis PLUS parental antibiotics. Initial choice of antibiotic based on Gram stain then sputum cultures

***W**ound*

(3) Wound infection – after hysterectomy the incidence is 5%
90% of this 5% is within the first 2 weeks after hyst. The incidence is 8 times more frequent if the patient is > 200 #

Diagnosis – presents with fever on 5–10th day, tachycardia, increases tenderness and pain
Two RARE types – VERY VIRULENT (can produce toxicity in the first 48 h). These are (i) *Clostridium* and (ii) acute β-hemolytic streptococcus
Treatment – (i) Open and drain, (ii) Gram stain, aerobic and anaerobic cultures, (iii) pack to debride and irrigate, (iv) antibiotics if peripheral cellulitis, (v) delayed closure after afebrile and granulation and (vi) prophylactic antibiotics for high-risk or other situations

W*alk her!*

(4) Phlebitis – superficial thrombophlebitis is commonly associated with IV catheter
Diagnosis – presents with superficial tenderness along course of veins and develops painful, erythematous induration with or without fever
Treatment – IV caths should be replaced every 48 h
(i) If phlebitis occurs – remove catheter, (ii) rest, elevation and local heat, (iii) moderate to severe cases – treat with NSAIDs or rarely with therapeutic doses of IV heparin and antibiotics

(5) DVT 15% incidence
Usually begins during surgery so prevent rather than treat.
Three key predisposing factors are (i) increased coagulation factors, (ii) damage to vessel wall, (iii) venous stasis
Diagnosis – 50% are asymptomatic. The 50% symptomatic patients:

(a) Induration of calf muscles	68%
(b) Minimal edema	50%
(c) Calf tenderness	25%
(d) Difference in leg diameter	11%
(e) Homan's sign	10%

(f) Doppler ultrasound (venography is the Gold Standard)
Treatment – Heparin 5000–10 000 IU IV then 1000–1500 IU until ptt is 1.5–2.5 × out. For 5–7 days then Coumadin 15 mg daily for 3 months after 48 h of heparin. *Also see* treatment with enoxaparin (Lovenox) under DVT

Etiology

Hereditary deficiencies:

(a) Factor VIII deficiency	25%
(b) Factor V Leiden	20%
(c) Homocysteine	10%
(d) Protein 20280	6%
(e) Protein C	3%
(f) Protein S	1–2%
(g) Antithrombin III	

DVT risk in perspective (chance of DVT):

28-year-old female not on OCPs	10/100 000
49-year-old female on HRT	30/100 000
32-year-old pregnant female at 24 weeks' gestation	60/100 000

(6) Septic pelvic thrombophlebitis – occurs in procedures 0.1–0.5%
Diagnosis – exclusion of others. When fever is not responding to appropriate antibiotic treatment and there is no abscess or hematoma
Sometimes presents with fever, tachycardia, GI distention or unilateral abdominal pain
Treatment – heparin 7–10 days. Long-term treatment not needed unless septic pulmonary embolus occurred during hospitalization

***W**ater*

(7) UTI – most commonly acquired in hospital patient from Foley cath
Atonic bladder is more prone (with or without catheter)
Increased risk – (i) older female, (ii) diabetics (3 × risk), (iii) length of time catheter is left in place
Diagnosis – presents with frequency, mild dysuria if lower UTI presents with high fever, chills, flank pain if higher

Treatment – antibiotic therapy. If symptoms persist after ab rx, check IVP to rule out ureteral obstruction
Treat with ab for 10 days if symptoms exist with Foley in place, not just single dose or 3-day therapy

***W**onder drugs*

(8) Drug fever – reaction to a drug can cause fever. Discontinue the suspected drug

Remember the Ws again

Wind (atelectasis)	Postop day 1–3
(pneumonia)	Postop day 3–7 or >
Wound (*Streptococcus* or *Clostridium*)	Postop day 1–2
(other bacteria)	Postop day 5–7 or >
(ovarian abscess)	Postop 1 week or >
(cuff cellulitis)	Postop day 4–6
Walk (phlebitis)	Postop day 3–5
(DVT)	Postop day 3–7 or >
Water (UTI)	Postop day 3–7 or >
(ureteral obstruction)	Postop 1 week or >

Wonder drugs (drug-induced fever). Anytime while on any drug(s)

Consider Ob situations too!

Womb (endometritis)

Weaning (breast engorgement, mastitis, breast abscess)

FIBROCYSTIC BREAST

Occurs in what % of females age of < 21	10%
What % of cytologic specimens of breast fluid show evidence of malignancy?	0.1–1%

Most common benign condition of the breast
Histology – cystic and epithelial proliferation and stromal fibrosis. If associated with atypia → associated with increased incidence of cancer

FIBROIDS

Symptoms of bleeding vs pain/pressure	33% vs 33%

Symptoms depend on #, size and location. (Frequency, urgency, rectal pressure, infertility, enlarging midline mass?)

What % of fibroids are symptomatic?	20–40%
Uterine sarcoma noted in only	0.1%

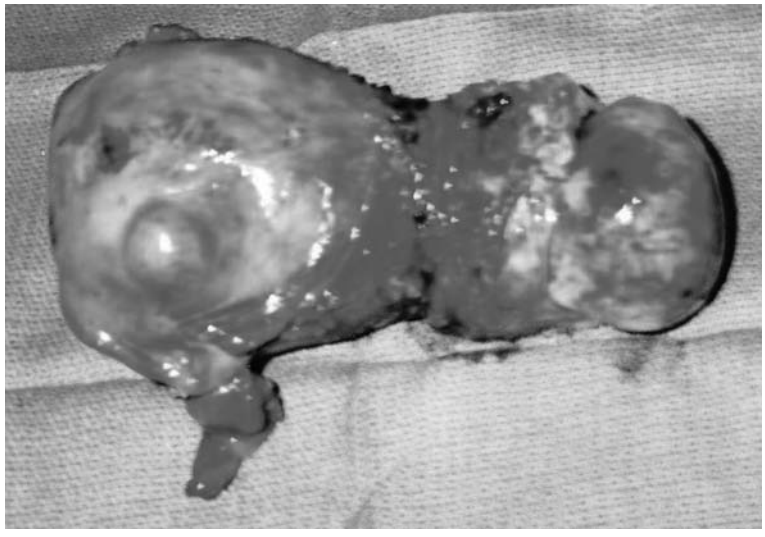


Figure 5 The patient presented with heavy bleeding, anemia and a large mass protruding into the vagina. Note the large fibroid coming through the dilated cervical opening of the uterus

Treatment

Expectant

(1) Expectant especially if uterus ≤ 12 cm with slow growth (< 6 cm in size in 1 year) or no growth

Hormonal

(2) Hormone therapy – Lupron, DMPA, danazol, RU486

(A) Advantages
- (a) Perimenopausal – often avoids hysterectomy
- (b) Shrink to allow better surgery mode
- (c) Decreases blood loss (100–150 cc)
- (d) Correct anemia decreasing need for transfusion
- (e) Atrophy endometrium for hysteroscopic ablation

(B) Disadvantages
- (a) Can cause degeneration – 'piece meal' myoma
- (b) Hypoestrogenic side-effects
- (c) Expense
- (d) Need for injections

Myomectomy

(3) Myomectomy – used especially for retaining fertility. Recurrence rate with myomectomy is 20%

Destructive techniques

(4) Laparoscopic myolysis – electrothermy, laser coagulation or cryo (holes drilled)

(5) Laparoscopic cryomyolysis – one hole is drilled into center of fibroid to form ice ball ('her/option' cryoblation system by CryoGen, San Diego, CA)
Both myolysis and cryomyolysis respond better if treated preoperatively with GnRH agonist

UAE

(6) Uterine arterial embolization

Hysterectomy

(7) Hysterectomy

Most hysterectomies should be performed vaginally even for fibroids. The advantages for the patients more than outweigh the risks and disadvantages. (MIVH developed by the author is the newest method)
See section on Hysterectomy comparing TVH to TAH

Comparison of hysterectomy to myomectomy

Advantage of hysterectomy over myomectomy

(1) Less blood loss

(2) Decrease chance of recurrence (within 20 years of a myomectomy, 25% subsequently have hysterectomy for recurrences)

(3) Postop complications much less wound infection 2%
bleeding 2%

Advantages of myomectomy over hysterectomy

(1) Preservation of reproductive capacity

(2) Lack of possible negative psychological effects from uterine loss

Myomectomy for infertility

Fibroid in association with recurrent second-trimester pregnancy loss. Location (submucous) is more significant than size of fibroid

Theories

(1) Thinning of endometrium so implantation is in poor site

(2) Rapid growth (increased hormones of pregnancy) → compromises blood supply → necrosis ('red degeneration') → uterus contracts

(3) Encroachment of fibroids upon fetal space to develop

Surgery

Reserved for repetitive second-trimester spontaneous abortions with female whose abortuses were normal (pheno + karyotype) and viability is > 9–10 weeks' gestation

Recurrence rate of myomectomy for infertility is 20–25%

'Red' degeneration

Myomas during pregnancy or puerperium occasionally undergo 'red' or 'carneous' degeneration that is caused by a hemorrhagic infarction

Signs and symptoms

(1) Focal pain

(2) Tenderness to palpation

(3) Occasional low grade fever

(4) Moderate leukocytosis common

(5) Peritoneal 'rub' – develops secondary to inflammation of parietal peritoneum overlies infarcted myoma

Differential

(1) Appendicitis
(2) Placental abruption
(3) Ureteral stone
(4) Pyelonephritis

Treatment

(1) Analgesia (i.e. codeine)
(2) Usually spontaneously abates within a few days

FISTULA

Etiology

Ob/Gyn surgery or radiation therapy. Most common cause Ob trauma

Usually due to episiotomy	3rd or 4th degree
Location of fistula after hysterectomy or enterocele repair	upper 1/3
Location of fistula after posterior colporrhaphy	lower 1/3
Genito *urinary* fistula incidence after radical hysterectomy is	1–2%

Symptoms

Usually 7–14 days postop there is rectal passage of blood clots or gas/fecal material from vagina. Causes emotional distress in patient

Diagnosis

Define defect with small metal probe or catheter in rectum with methylene blue and tampon in vagina

What % of fistula spontaneously close?	25–50%
Obstipate	25% heal spontaneously

Treatment

Mechanical bowel prep

Golytely	1 liter/h till clear or
Phosphate soda	4 oz × 2 @ 4 h apart prior

Antibiotic prep

Cefotetan 2 g or Unasyn 3 g

Surgical technique – wide mobilization, adequate blood supply, minimal to no pressure

Postop: low residue diet, decrease fiber, stool softener and avoid coitus

Critical surgical principles for fistula repair

(1) Wide mobilization
(2) Excision of entire tract
(3) Meticulous closure of rectal orifice
(4) Reapproximation of broad tissue surface to broad tissue surface WITHOUT surface tension

Choices of fistula repair

(1) Use of Martiius graft
(2) Transverse transperitoneal repair
(3) Development and advancement of a rectal flap
(4) Development and advancement of a vaginal flap
(5) Interposition of the levator muscle and fascia of Colles between the vaginal and rectal tissues

FITZ-HUGH–CURTIS SYNDROME

Perihepatic inflammation and adhesions that develop in 10–15% females with acute PID from transperitoneal or vasc dis of GC or Chl

Signs and symptoms

(1) RUQ pain
(2) Pleuritic pain
(3) Tenderness with liver palpation
(4) Usually preceded by PID

Differential

(1) Acute cholecystitis
(2) Acute appendectomy
(3) Ectopic pregnancy
(4) Pneumonia
(5) Adnexal torsion

Diagnosis
(1) H&P
(2) Laparoscopy (most accurate) – especially with patients of uncertain diagnosis or patients not responding to treatment
(3) Endometrial biopsy – 90% correlation
(4) Ultrasound – limited value
(5) Culdocentesis – questionable
(6) Laboratory – lacks evidence to support diagnosis

FLU

See Influenza

FORCEPS

Classifications

• *Outlet*
(1) Fetal head is at +3 station and rotation does not exceed 45°
(2) Scalp is visible at the introitus without separating the labia
(3) Fetal skull has reached the pelvic floor
(4) Sagittal suture is in AP diameter or R or L OA or OP
(5) Fetal head is at or on perineum
• *LFD*
(1) Fetal skull at or > +2 cm station and rotation may exceed 45°
(2) Rotations of 45° or < (L or R OA to OA or Lor R OP to OP)
(3) Fetal skull not on pelvic floor
• *MFD*
Station > 2 + but head engaged

Indications for operative vaginal delivery

• The following apply when the fetal head is engaged and the cervix fully dilated
(1) Prolonged second stage
(2) Suspicion of immediate or potential fetal compromise (fetal bradycardia)
(3) Shortening of the second stage for maternal benefit (maternal cardiac condition)
(4) Maternal exhaustion

Types of forceps

Simpson's – best suited for a molded head
Tucker–McLane's – solid blades, prefer for unmolded outlet
Elliot's – preferable for the unmolded head
Keiland's – for rotation OP to OA (Scanzoni maneuver)
Piper's – for breech delivery
Laufe's – short, outlet deliveries

Indications (examples)

Maternal exhaustion
Prolonged second stage
Maternal cardiac condition
Fetal bradycardia

Requirements for safe forceps

Complete cervical dilatation
Empty bladder
Rupture of membranes
Presenting part at or below ischial spines
Adequate pain relief
Likelihood of success

Complications

(1) Retinal hemorrhage
More common after instrumental delivery. Appears to be transient
(2) Subgaleal hematoma
(a) Bleeding beneath the aponeurosis of the scalp. (Most serious complication of VE rather than forceps. 5–10/1000 VE) 89% of subgaleal hematomae were delivered by VE – 3% died
(b) Baby may show signs of overt shock, hypotension, tachycardia and a drop in hematocrit. Treatment includes careful monitoring, use of a pressure bandage to the scalp and early transfusion

(3) Caput
Effusion of serum that overlies the periosteum that resolves
(4) Cephalohematoma
Collections of blood that accumulate under the periosteum of skull bones, usually parietal. 10–25% are associated with skull fracture
(5) Intracranial hemorrhage
Intracranial hemorrhage in the term newborn infant is more common than was previously realized
(6) Skull fractures
True incidence of skull fracture at the time of VE may be higher than appreciated, for unless neonates demonstrate abnormal neuro behavior, they do not routinely undergo skull films

Maternal complications-
Only 4% of women who undergo instrumental delivery sustain anal sphincter injury, but up to 50 % of women with 3rd-degree perineal tears have had instrumental delivery
Strongly consider right mediolateral rather than midline episiotomy because the midline is associated with serious risk of anal sphincter injury with potential long-term consequences

Forceps versus vacuum

Retinal hemorrhage more common with VE than forceps (38% vs 17%)
Cephalohematoma more common with VE than forceps (9% vs 3%)
Facial bruise, abrasion and nerve palsies more common with forceps
Maternal injury is more likely with forceps, fetal injury is more likely with vacuum

Avoid forceps if

(1) Proper application is not possible
(2) The case is risky

Key

(1) Remember that rotations can also be done manually
(2) Proper placement!
(3) Documentation (indication, instrument used, station, position, degree of asynclitism of fetal head when forceps initiated, any rotation that was required, anesthesia, EBL, specifics of laceration and/or episiotomy, infant Apgar scores and cord gases. Write a pre-op and post-op detailed note)
(4) Consider Ob history (Hx of malpresentation, persistent OP – possible anthropoid pelvis or obesity, excessive weight gain, and glucose intolerance – all warning signs of LGA infant)
(5) Informed consent – written recommended like for C-section.
(6) Abdominal exam is critical – EFW, OA, and engaged fetal head?
(7) Keep molding in mind – traction + molding may increase risk of intracranial injury
(8) Be aware of fetal head position throughout labor
(9) Have a valid indication (see above indications)
(10) Avoid sequential use of instruments (VE-LFD-VE) if at all possible
(11) Have a clear endpoint and exit strategy (failed forceps to C/S is okay and some Ob Departments have set up guidelines for the number of forcep and/or vacuum extraction attempts
(12) Handle bad outcomes with compassion

• Spontaneous vaginal delivery is more likely after previous instrumental delivery than after cesarean section

FRAGILE X

Most common inherited form of mental retardation

More common in males occurring	1 : 1500
Incidence in females	1 : 2500
Triple repeat (cystosine–guanine–guanine). Full mutation	> 200

All sons who inherit an expanded, full mutation will have fragile X features. In daughters, prognostication is limited

Gene

FMR-1 (long q arm of X): Premutation	50–100 repeats
Mutation	> 200 repeats

Symptoms

Autistic behaviors. Macroorchidism in adult males. Narrow face with a large jaw. Speech and language problems. Becomes more noticeable with age. Mental retardation ranges from borderline to severe – most are moderate

CVS

Not reliable
Transmission depends on:
(1) Sex of parent
(2) Number of cytosine–guanine–guanine repeats in the parental gene

Diagnosis

(1) DNA-based molecular tests
  (a) Southern blot analysis
  (b) Polymerase chain reaction
(2) Test for fragile X if developmental delay or MR of ? etiology
(3) Test if family history of fragile X or family history of MR
(4) Offer amniocentesis to known carriers
(5) Test women with elevated FSH, especially with family history of premature ovarian failure, fragile X syndrome, or relative of either sex with undiagnosed mental retardation, late-onset tremor or ataxia, or movement disorders

Prenatal fragile X diagnosis can be obtained using DNA from amniocytes or CVS. Screening for fragile X would cost from $99 to $300.00 per test

FRANK PROCEDURE

Procedure which is utilized to take years of pressing to form new vagina using dilator

GALACTORRHEA

Prolactin level – if up, get TSH – if up, check T_3 and T_4
If normal TSH and prolactin and regular menses no further test
If abnormal menses – get AP and lat coned down view of sella turcica – if abnormal, get MRI
If prolactin elevated and patient has headache or visual disturbances, get MRI
If prolactin > 100 ng/ml, get MRI
Treatment: If estrogen levels (E_2) are decreased under this value, one should give estrogen to prevent osteoporosis 40 pg/ml
If patient has macroadenoma with increased prolactin, bromocriptine is to be given
Empty sella syndrome (incompleteness of the sellar diaphragm) is seen in what % autopsies? 5%
Empty sella syndrome is seen in what % of patients with amenorrhea and galactorrhea? 4–16%

Causes	*Medications*
Thyroidism (hypo) 3–5%	**H**ormones (estrogens, OCPs, TRH)
Adenoma	**A**ntihypertensives (α-methyl dopa)
Kidney disease	**D**opamine
Medications	**P**sychotropics (phenothiazines)
Hypothalamic	**A**ntiemetics or anesthetics
Acromegaly (pit tumor, Cushings)	**L**-Dopa
Trauma (thoracic operation, herpes zoster or chest trauma)	

Lactation syndrome varients causing galactorrhea:
Forbes–Albright – association of galactorrhea with intrasellar tumor
Chiari and Frommel – antecedent pregnancy with inappropriate persistent galactorrhea
Argonz and del Castillo – inappropriate galactorrhea in absence of previous pregnancy

GALLBLADDER DYSFUNCTION

Oral contraceptive therapy increase risk	2 ×
Hypolipidemic medicines increase risk	33%
This % of patients may have gallstones in common bile duct for years asymptomatically	8–15%
This % of pregnant patients (in study of GB disease) scanned by ultrasound have evidence of stones	96%
Gold Standard to test for stones in common bile duct	ERCP (endoscopic retrograde cholangiopancreatography)

GASTROSCHISIS VERSUS OMPHALOCELE

Gastroschisis	Incidence is	1 : 10 000
	ISOLATED ANOMALY. Defect in abdominal wall is to the RIGHT of the umbilicus	
	Preterm labor complicates	> 50%
	Vaginal delivery is okay (C-section does not improve chances)	
	Survival is approximately	90%
Omphalocele	Incidence is	1 : 4000
	Associated with OTHER ANOMALIES	70% of the time
	Insertion of cord into sac with membrane – CENTRAL	
	Preterm labor also complicates	> 50%
	Mortality is approximately	60%
	If contains only bowel → 87% have abnormal karyotype	
	If contains only liver → 9% have abnormal karyotype	

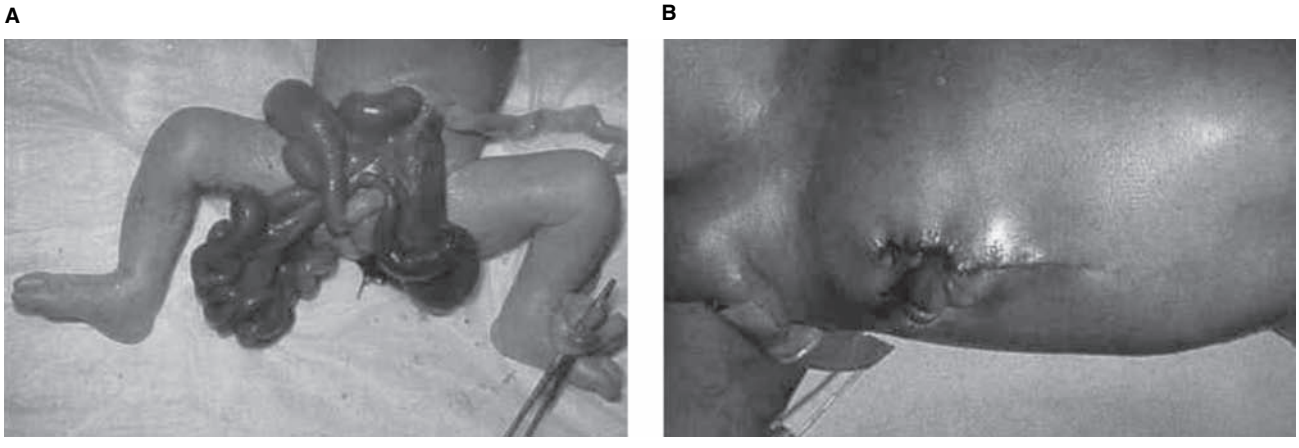


Figure 6 Gastroschisis. (a) This infant delivered prematurely while patient was undergoing abruption and hemorrhage. Note the defect in the abdominal wall is to the right of the umbilicus. In omphalocele, the defect is central. (b) The same infant after repair

GENETICS

This % of chromosomal abnormalities are lost (first trimester) spontaneously 50–60%

Triploidy – common findings in miscarriage. Dispermy leads to partial moles. Digyny leads to small, malformed fetus, PIH

Chromosomal errors

Rate of recurrence 1%

Autosomal trisomies (detected in 50–60% of abnl abs)

(1) Down's – most common, accounts for @ newborns 1/650

Down's arise by non-disjunction in first or second meiotic division in ovum (increased frequency with AMA) 90%

% of sperm have wrong # of chromosomes as well – perhaps swim poorly or fail to fertilize well (no age effect is apparent) 5–10%

(2) Edward's – 90% die before age 1. Choroid plexus better marker here than for +21. Incidence is 1/5000

(3) Patau's – clefting, polydactyly, CNS anomalies 1/10 000

Sex chromosomal abnormalities

(1) Monosomy X – Turner's syndrome

What % end in first- or second-trimester losses? 90%

Posterior nuchal SEPTATED cystic hygromas @

First trimester 50%

Second trimester 85%

Stigmata of Turner's 45X

Most common single entity found in spontaneous abs 1/2500

Webbed neck probably due to cystic hygroma pigmented nevi

Low posterior hairline

Normal IQ but seem MR due to hearing deficit. Coarctation of aorta. Increase carrying angle. Short metacarpals

Shield chest with wide spread nipples. Streak gonads. Renal agenesis

XY – Noonan's syndrome

	'Male Turner's' cardiac lesion is pulmonic stenosis. Phenotype like Turner's	
	(2) Klinefelter's syndrome	XXY
	Tall, sterile males with small, firm testes. IQ down @	20
	(3) XXX, XYY (paternal origin of extra chromosome almost as frequent as maternal.) IQ drop compared to sibs @	20
Chromosomal rearrangements	*Translocations*	
	About how many humans have a translocation?	1/250
	(1) Reciprocal – occur by breaks and repairs. Can involve any part of any chromosome. Effects are variable. A 'general' figure for liveborn outcome	8–10%
	(2) Simple – addition of one part of chromosome to another non-homologous chromosome	
	(3) Robertsonian – occur by centromeric fusion of acrocentric chromosomes	
	Which Robertsonian translocation is the commonest translocation in man?	13/15
	The 13/15 translocation may lead to increased risk of sp ab, it is usually unrecognized, but actual risk for abnormal liveborn offspring is	< 2%
FISH (fluorescent in situ hybridization)	FISH probes are pieces of DNA that anneal with identical pieces of DNA in the cell. What % abnormalities are prenatally detected?	80%
	Microdeletions may not be visible on standard cytologic analysis so FISH microdeletion probes are used with abnormal US +/or fam hx	
	Examples of microdeletion disorders:	
	Prader–Willi (hypotonia, obesity, MR)	
	Angelmann (seizures, ataxia, MR)	
	Williams (elfin facies, cardiac anomalies, MR)	
	DiGeorge (immune deficiency, hypoparathyroidism, cardiac anomaly)	
	Smith Magenis (autistic mannerisms, MR, unusual behaviors), etc.	
Telomere probes	Detects ends of chromosomes, will identify smaller rearrangements both balanced and unbalanced. A new tool just becoming available	
Location of chromosomal abnormalities of these common disorders	Blood type (CDE antigens)	1
	Wolf syndrome (deletion)	4
	Cri du chat (deletion)	5
	21-hydroxylase deficiency	6
	Cystic fibrosis	7
	11β-hydroxylase deficiency	8
	Paracentric inversion (1–3%)	9
	Patau's syndrome	13
	Robertsonian translocations	(13–15) and (21, 22), 14q, 21q
	Prader–Willi	15q12
	Angelman	15q12
	Most common trisomy detected in spontaneous abortions	16
	Edwards' syndrome	18
	Down's syndrome	21
	Type I diabetes	21
	DiGeorge's syndrome (microdeletion)	22
Autosomal dominant	Rate of recurrence	50%
	Neurofibromatosis	
	Marfan's	
	Huntington's chorea	
	Polycystic kidneys with adult onset	
	Von Willebrand's disease	
	Sipple's syndrome (multiendocrine neoplasia type II)	
	% risk of a new dominantly transmissible disorder if Dad > 50 is	1–2%
X-linked	Rate of recurrence	50%
	Testicular feminization	
	Hemophilia A	deficiency factor 8 (1/10 000)
	Placental sulfatase deficiency	
	Muscular dystrophy	1/3300
	Diabetes insipidus	
	There are sex-linked dominants (Duchenne muscular dystrophy and hemophilia A and B)	

	There are sex-linked recessives (worse in males – Goltz syndrome, incontinentia pigmentii and those that occur almost exclusively in females – Rett, Aicardi syndromes)	
	Fragile X More common in females than males in ratio of Most common hereditary form of MR. Due to triplet repeat mutation	2 : 1
Autosomal recessive		25%
	Thalassemia, sickle cell anemia Congenital adrenal hyperplasia PKU Galactosemia Metabolic disorders (Tay–Sachs) Cystic fibrosis – deletion of phenylalanine at position 508	
Chromosome groups	1, 2, 3	A
	4, 5	B
	6, 7, 8, 9, 10, 11, 12	C
	13, 14, 15	D
	16, 17, 18	E
	19, 20	F
	21, 22	G
	Sex chromosomes	
Imprinting	Process of turning off one copy of a gene (mat or pat) during RNA transcription Many imprinted genes have to do with growth: Beckwith–Wiedemann syndrome characterized by organomegaly, omphalocele and risk of abdominal tumors. Make too much	IGF_2
	Prader–Willi is microdeletion with imprinted	*SNRPN*
	From mother or maternal uniparental disomy (2 copies of mother's chromosome 15) and inactivated	*SNRPN*
	UPD – unexplained IUGR, neonatal diabetes	
Multifactorial inheritance	Offspring of affected individuals recurrence risk is	2–8%
	Examples: spina bifida, clefting, pyloric stenosis, clubfoot, etc.	
Cancer genetics	Oncogenes serve as stimuli to cell division such as HPV and genes associated with aggressive tumor growth such as HER2/*neu* and estrogen receptors	
	Tumor suppressors function to be certain that DNA transcription is correct before cell is allowed to divide. Tumor suppressors are sometimes hereditary (breast + ovary) such as	*BRCA1*
	and	*BRCA2*
	and some endometrial cancer is hereditary such as	*HNPCC*
	The *most* common *HNPCC*-associated cancer is endometrial cancer Hereditary breast–ovarian cancer risk assessment is *best* done using the Frank model	
	The lifetime risk of developing colorectal cancer or breast cancer if your patient has inherited a clinically significant *HNPCC* gene mutation or a *BRCA1/2* gene mutation is	50%
	When evaluating an asymptomatic woman for *HNPCC*, an appropriate first step in gene mutation testing would be to test colorectal tissue from an affected family member for microsatellite instability	
	The common founder mutation gene test is *most* useful in Ashkenazi Jewish men and women	
	The empiric risk for a fetus with a balanced translocation, to have anomalies or develop mental delay	10%
	MHC (major histocompatibility complex) in humans is located on what chromosome?	7
	Occasionally, carriers will display some symptoms. An example is with cystic fibrosis and CBAVD (congenital bilateral absence of the vas deferens)	

Polymorphism is a change in the genetic code that is not expected to significantly change the size or function of the resulting protein

Ultrasound findings in regards to genetics:

(1) The likelihood of a karyotypic abnormality after detecting
    1 structural abnormality is 14%
    5 70%
    7 82%
(2) The all-or-none scoring system for a genetic sonogram evaluates all the following:
    (a) Thickened nuchal fold
    (b) Pyelectasis
    (c) Echogenic intracardiac focus
    (d) Choroid plexus cysts
(3) A normal second-trimester ultrasound examination reduces the likelihood of the fetus being karyotypically abnormal by approximately 50%

GENITAL MUTILATION

(1) Usually performed prior to adolescence
(2) Removal of clitoral prepuce, clitoris, labia minora and occasionally much of the labia majora
(3) Infibulation of the vagina
(4) No scientific basis for the procedure

GENITAL ULCERS

Herpes	HSV – painful vesicles intranucleated giant cells – Rx acyclovir Incubation period 2–7 days
Syphilis	*T. pallidum* – painless – darkfield microscopy – Rx penicillin Incubation period 2–4 weeks
Chanchroid	*H. ducreyi* – very painful, Gram stain (school of fish) – Rx rocephin/Emycin 1–14 days
Granuloma inguinale	*Calymmatobacterium granulomatis*, PL ulcer, Donovan bodies, TCN 1–4 weeks
LGV	*Chlamydia trachomatis* – tender lymph nodes, culture/compfix, Rx doxycycline 3 days to 6 weeks
Causal organisms and important points	Granuloma inguinale *Calymmatobacterium granulomatis* Look for 'Donovan bodies' – look like 'safety pins'
	Chanchroid *Hemophilus ducreyi* Look for 'school of fish pattern' – the classic streptobacillus chains
	Lymphogranuloma venereum *Chlamydia trachomatis* Serotypes L1, L2, L3. Look for multiple fissures of perineum and rectum

GESTATIONAL TROPHOBLASTIC DISEASE

Hydatidiform moles	1/1000–1/1200
	Partial mole 69XXX or 69XXY Usually small for dates. Focal trophoblastic proliferation. < 5–10% of postmolar GTD
	Complete mole 46XX or 46XY Fetus is absent. Diffuse villous edema. Frequent medical complications. Theca lutein cysts are present in 15–20%
	Invasive moles Rarely metastasize. Treated with chemotherapy
	Invasive moles follow partial moles in @4–11%
	Invasive moles or choriocarcinoma follow complete moles @ 25%

Choriocarcinoma 1/20 000–1/40 000

- Develop from term pregnancies 50%
- Develop after molar pregnancies 25%
- Develop after other gestations 25%

Gestational choriocarcinoma

Develop early systemic hematogenous metastasis. Chemotherapy is indicated. Syncytiotrophoblast and cytotrophoblast elements. Pure epithelial neoplasm

Placental site tumors

Secrete amounts of β-hCG that are small in relation to tumor volume
NOT SENSITIVE TO CHEMOTHERAPY
HYSTERECTOMY is treatment of choice

CXR ?

What % of pts with a negative CXR with GTD will have metastasis on CT scan? 40%

Symptoms

Bleeding between 6–16 weeks' gestation	80–90%
Large for dates	50%
Small for dates	25%
Theca lutein cysts	15%
Hyperemesis	8%
Hyperthyroidism	1%
Incidence of PIH (first or second trimester)	1%

Diagnosis

(1) Complete mole
  (a) hCG > 100 000
  (b) Ultrasound → 'snowstorm' appearance
(2) Partial mole
  (a) Histology after D&C
  (b) Pre-evacuation hCG increased but not as high as in the complete

Incidence of spontaneous remission after evacuation of molar pregnancy is 80%

Average time (acc to DiSaia) until undetectable levels of hCG after evacuation of molar pregnancy 7
hCG levels usually above 100 000 mIU/ml

Treatment

Labs

(1) Clotting function studies
(2) Blood type and antibody screen
(3) Renal and liver function studies
(4) Determination of β-hCG level
(5) CXR (CT might be considered) and ultrasound
(6) CBC with platelets

Evacuate uterus. Use suction D&E device with 12–14 mm suction cannula. Pitocin to be started after starting the evacuation and continue for several hours. If the uterus is > 16 weeks size – have 2 units of PRBCs available. Pulmonary complications are frequent with enlarged uteri. Replace blood or IV loss p.r.n. Give RhD p.r.n.

Methotrexate – treat non-metastatic GTD. Methotrexate is excreted by kidneys – get pretreatment creatinine. Hysterectomy – will shorten and decrease amount of chemo but does not improve high-risk metastatic disease

Treatment for hydatidiform mole is usually curative in 80%
What size suction cannula should be used during a suction D&E for hydatidiform mole? 12–14 mm
Pitocin should be used after start of evacuation and continued for several hours. If the uterus is over 16 weeks, how many units of PRBCs should be available? 2 units

hCG levels should be obtained every 1–2 weeks
Until results are negative for how many negative determinations? 3
Then hCG levels should be followed every 3 months
For how many months? 6–12

GnRH ANALOGS

Decapeptide produced in arcuate nucleus in the median eminence of hypothalamus inhibited by dopamine
Substitutions at position 6
Secreted in pulsatile fashion by hypothalamus
Serum half-life 2–8 min

GRANULAR CELL TUMOR

Misnomer because actually is schwannoma, arising from a nerve sheath. Lesions can occur or may recur anywhere but rarely are truly malignant. Sometimes called myoblastoma

GRANULOMA INGUINALE

Chronic PAINLESS nodule of vulva that progresses to a beefy red ulcer. Uncommon in the USA but with increased frequency in the Caribbean

Cause — *Calymmatobacterium granulomatis*

Diagnosis — Donovan bodies (deep-staining bacteria with a bipolar appearance resembling a safety pin)

Treatment — Oral tetracycline

GROUP B STREPTOCOCCUS

Percent of pregnancies that are colonized with GBS in vagina or rectum 20%
Fatality rate in infants infected with GBS 5–20%

ACOG — Selective prophylaxis to all at risk regardless of culture status

Risk factors
(1) Temperature > 100.4°F
(2) Rupture of membranes > 18 h
(3) Premature infant < 37 weeks
(4) Chorioamnionitis
(5) Intra-amniotic infection
(6) GBBS bacteriuria in pregnancy
(7) Previously affected infant

CDC — Screen at 35–37 weeks' gestation and treat all + results and increased risk patients

AAP — Screen at 26 weeks' gestation and treat all + results and increased risk patients

Current Recommendations

(1) All pregnant women should be screened at 35–37 weeks' gestation for vaginal and rectal colonization

(2) Screen each pregnancy to determine need for prophylaxis. Prior colonization in a previous pregnancy is *not* an indication for treatment in a future pregnancy.

(3) Exception – women with prior GBS infected neonate or with GBS colonized urine in the current pregnancy should receive intrapartum prophylaxis

(4) GBS colonized women who have a planned C-section prior to rupture of membranes or onset of labor do not need intrapartum prophylaxis. If prophylaxis is given, it is best at time of incision rather than at least 4 hours prior to delivery

(5) If no screening obtained and patient is < 37 weeks, rupture of membranes > 18 hours, or temperature ≥ 100.4°F (38°C), then intrapartum prophylaxis is indicated

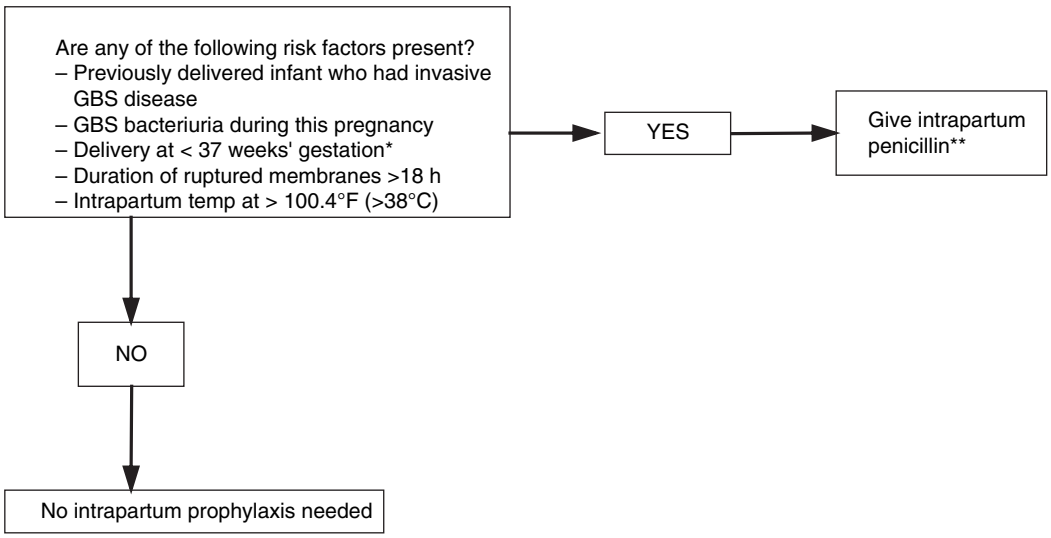
Treatment

Penicillin G 5 million units IV, then 2.5 million units IV every 4 h or ampicillin 2 g IV, then 1 g IV every 4 h. If the patient is allergic to penicillin and not at high risk for anaphylaxis, the drug of choice is cefazolin. If allergic to penicillin and high risk for anaphylaxis, obtain GBS culture with erythromycin and clindamycin sensitivity testing and if sensitive give clindamycin 900 mg IV every 8 h. If the culture is not sensitive to either antibiotic or sensitivity testing is not feasible, then the drug of choice is vancomycin. To date, there are no reported GBS penicillin-resistant strains; however, there has been a notable increase of *in vitro* GBS resistance to clindamycin and erythromycin

GROWTH HORMONE

(1) Decreases glucose tolerance
(2) Increases incidence of carpal tunnel syndrome
(3) Increases exercise capacity
(4) Decreases body fat
(5) Increases muscle mass

American College of Obstetrics and Gynecology algorithm for prevention of early-onset group B streptococcal disease in neonates, using risk factors



*If membrane ruptured at < 37 weeks' gestation, and the mother has not begun labor, collect group B streptococcal culture and either (a) administer antibiotics until cultures are completed and the results are negative or (b) begin antibiotics only when positive cultures are available

**Broader-spectrum antibiotics may be considered at the physician's discretion, based on clinical indications

Group B beta streptococcus - Summary

Group B Beta Strep is responsible for causing 15 000 cases of neonatal sepsis. The number of early-onset disease cases decreases with intrapartum prophylaxis for GBBS carriers. Although prophylaxis is widely accepted, there is still debate as to the best strategy for identifying women who are carriers. Also, cost-effective – necessity of antepartum screening has come under question

Asymptomatic colonization GBBS is present in 15–40% of pregnant women. (Variation in colonization rate 2 degrees to ethnicity, geographic location, number sites cultured as well as methods of culture)

African-Americans > 21%; Hispanics 20.9%; Whites 13.7%;
Hisp (Caribbean descent) 28%; (Mexican descent) 9.2%;
Diabetics 2 x higher – 20 vs 10%

Isolation rates highest from introitus, rectum, cervix

Supports concept gastrointestinal tract as reservoir

Vertical transmission occurs 40–73% of infants of colonized women

For every 100 colonized women, only 1 infant will develop GBBS

Overall, attack rate ranges from 1 to 3 per 1000 live births

Risk factors for neonatal GBBS

Preterm labor
Preterm, PROM
ROM > 18 h prior to delivery
Intrapartum fever
Hx of infant with GBBS infection

Clinical manifestations (most common) of early-onset GBBS < *c.*7 days of life (late onset > *c.*7 days of life). Early onset accounts for 66% of all neonatal infection. Mortality rate 25–33%

(1) Septicemia
(2) Pneumonia
(3) Meningitis

Late onset presents with meningitis in 85% (nosocomial/cross. colon)
Mortality 15–20% but survivors – 25–50% neurologic sequelae

Culture

Gold standard 'Todd-Hewitt' Broth. Because cultures take 48 h, rapid GBBS antigen using coagulation, latex-particle agglutination and enzyme immunoassay. 1–2 h but has low sensitivity in lightly colonized patients, and could thus potentially fail to identify many patients who should receive prophylaxis

HALBANE PROCEDURE

Obliterates cul-de-sac
Approximates posterior vaginal wall to anterior rectal wall
Isolates from intra-abdominal pressure
Provides peritoneal shelf that deflects pressure from this dependent portion of female pelvis

HEADACHE

Chronic migraine is a frequent headache disorder that affects 2–3% of the general population

Features of primary headaches

	Migraine headache	*Tension-type headache*	*Cluster headache*
Aggravating or triggering factors,	Alcohol, chocolate, other foods, altered sleep, change in weather, menstruation, physical activity	Emotional stress, rebound effect of overuse of analgesics (mostly unknown)	Alcohol (mostly unknown)
Ameliorating factors	Dark, quiet, rest	Hot or cold compresses	Physical activity
Associated symptoms			
Nausea	Usual	Slight and uncommon	Rare
Phonophobia	Usual	Slight and uncommon	Rare
Photophobia	Usual	Slight and uncommon	Rare
Nasal congestion/ rhinorrhea	Rare	Not present	Usual
Red/tearing eyes	Rare	Not present	Usual
Ptosis/miosis	Not present	Not present	Often
Aura	Occasional	Not present	Not present
Characteristics			
Type of pain	Throbbing	Steady ache	Boring
Location of pain	Unilateral	Bilateral	One orbit
Intensity of pain	Moderate-to-severe	Slight-to-moderate	Excruciating
Duration	4 h to 3 days	Hours, weeks, months	30 min to 3 h
Frequency	2/week to 2/year	Daily to 2/year	Daily for weeks or months
Family history	Usual	Occasional	Rare
Gender	F > M	F > M	M > F

Sample questionnaire

Patient: _____ **Date:** _____

(Please answer all questions below – YES or NO – with a check mark)

PAST HEADACHES

(Patient may have more than one type of headache or mixed headaches)

YES	NO		
___	___	1.	Do you have an idea of what may be causing your headache? (Whiplash, diabetes, high blood pressure, eye strain, etc.)
___	___	2.	Did this same type of headache ever occur before?
___	___	3.	Do you have more than one type of headache?
___	___	4.	Is the headache pain so intense that sometimes it becomes unbearable?

TENSION HEADACHES

(Muscle contraction headache)

Head pain, tension, and muscle contractions of head, neck or shoulders

___	___	5.	Do your headaches occur during stressful tension or nervousness at home, at work or during social occasions?
___	___	6.	Do your neck, shoulder muscles or head junction feel tight and painful during the headache?
___	___	7.	Is your headache pain dull and steady, like an intense constant pressure?
___	___	8.	Does your headache feel like a tight band around the head?
___	___	9.	Do you usually have one (1) or more headaches per week?
___	___	10.	Do your headaches occur during the day?
___	___	11.	Does mother, father or any blood relative have similar headaches?
___	___	12.	Does exertion (lifting, running, straining, sex) affect your headache?
___	___	13.	Does nausea and/or vomiting occur before or during your headache?

MIGRAINE HEADACHES

(Common or Classic)

Usually women. Relieved by parenteral ergotamine confirms diagnosis

___	___	14.	Do you have any changes in vision (flashing lights, sensitivity to light, spots, blurred vision, etc.) before or during your headache?
___	___	15.	Does your headache usually start on one side of the head?
___	___	16.	Does your headache throb and pulsate or feel like it's pounding?
___	___	17.	Do your headaches usually occur during the night or upon awakening?
___	___	18.	Do your headaches usually occur during weekends and holidays?
___	___	19.	(Females only) Is your headache associated with your menstrual period?

CLUSTER HEADACHES

Usually men. 3 or more headaches per day for 4–8 weeks

___	___	20.	Do you have watering of the eye on the affected side of the headache?
___	___	21.	Do alcoholic drinks cause or aggravate your headaches?

ORGANIC ORIGIN

Allergy, sinus infection, aneurysm, brain tumor, etc.

___	___	22.	Does chocolate, cheese, milk, nuts, Chinese food or any other food cause or worsen your headaches?
___	___	23.	Do you have any hearing problems – noise, drainage or stuffiness in either ear?
___	___	24.	Have you noticed any paralysis, muscle weakness, numbness, swallowing problems or speech changes during your headaches?
___	___	25.	Do you have any facial pain, aching jaws, stuffiness or congested sinuses along with your headache?
___	___	26.	Has it been over eighteen (18) months since you last visited a dentist?

PREVIOUS TESTS & MEDICATIONS

___	___	27.	Have you had tests of headache? (X-ray, brain scan, injections, etc.)
___	___	28.	Have you used any previous headache medication? List all medications on the back of this form

Differentiate Tension, migraine, cluster and organic headaches

Treatment options

Migraines
(1) Naproxen 500 mg p.o. daily
(2) Metoclopramide 10 mg p.o. daily
(3) Butorphanol NS 1 mg
(4) Sumatriptan (Imitrex) 6 mg SC or 25 and 50 mg p.o. or 5, 10, and 20 mg NS
(5) Naratriptan hydrochloride (Amerge®) 2.5 or 5 mg tablet p.o. q. 4 h (max. dose 2 tablets in 24 h)

Migraine prophylaxis
(1) Propranolol 20 mg p.o. t.i.d.
(2) Verapamil 80 mg p.o. t.i.d.
(3) Methylergonovine 0.2 mg p.o. t.i.d.
(4) Naproxen 250 mg p.o. t.i.d.
(5) Divalproex Na^+ 250 mg p.o. b.i.d.
(6) Amitriptyline 10–25 mg p.o. q.d.
(7) Methysergide 2 mg p.o. b.i.d.
(8) Melatonin 3 mg @ 30 minutes prior to bedtime (esp. if HA related to delayed sleep phase syndrome)

Clusters
(1) Sumatriptan 6 mg SC
(2) Ergotamine mg sublingual

Cluster prophylaxis
(1) Verapamil 80 mg p.o. t.i.d.
(2) Ergotamine tartrate with caffeine 100 mg – 1 mg p.o. b.i.d.

Chronic tension headache prophylaxis
(1) Amitriptyline 25 mg p.o. q.d.
(2) Divalproex Na^+ 250 mg p.o. b.i.d.
(3) Dihydroergotamine 0.5 mg t.i.d. IV

Menstrual migraines

(1) Women experience migraines approximately 3 times more likely than their male counterparts
(2) What proportion of females experience *true* menstrual migraine, as opposed to menstrually associated migraine (MAM)? 7–14%

Falling estrogen levels reduce endogenous endorphin activity, raising sensitivity to pain

Menstrual migraines in OC users most likely occur during the placebo days of the pill cycle. Triptan with the lowest initial response rate but the drug most likely to stave off migraine recurrences because of its long half-life is naratriptan

Maximum recommended daily dose of sumatriptan tablets is 200 mg

In OC users experiencing recurrent migraine, the optimal Pill formula is norethindrone/ethinylestradiol 20 mg. OC users requiring add-back estrogen during the placebo week are advised to take:
conjugated equine estrogens
esterified estrogens
oral or transdermal 17β-estradiol

In severe and refractory MAM, which hormonal treatment is recommended? GnRH agonists

In menstrual migraineurs with comorbid hypertension, which prophylactic regimen might be particularly useful? β-blockers

Strategies to try in management of chronic daily headache and MAM:
(1) Switching to a lower-dose OC
(2) Instituting a 2-week course of a triptan
(3) Adding ice packs, massage and stress-reduction techniques

HEARTBURN (GRAVID CONSIDERATIONS)

Incidence in pregnancy There is a wide range of incidence 10–80%

Symptoms Indigestion, epigastric pain, dysphagia, water brash (hypersalivation), anorexia, nausea, vomiting and rarely pulmonary symptoms

Complications Esophagitis, bleeding, strictures (rare)

Helpful signs to diagnose

History:
Unable to lie down, forced to sleep upright
Exacerbates: fatty foods, caffeine, chocolate, natural mint, onions, garlic

Differential diagnosis

PUD, gastritis, gallstones, constipation, pancreatitis, fatty liver of pregnancy and pre-eclampsia

Consider these labs

Liver function tests, amylase, urinalysis

Treatment steps

(1) Avoid food/beverage 3 h prior to bed
(2) Avoid ETOH and smoking (decreased LES tone)
(3) Eat smaller and more frequent meals
(4) Avoid foods that exacerbate symptoms
(5) High-protein and calcium-rich food may increase LES pressure and improve symptoms

Medications

Antacids 50% pregnant women take antacids

Liquids have greater gastric acid neutralizing capacity
Tablets (according to one study) increased esophageal pH improved relief reflux

Aluminum hydroxide (Mylanta®, Amphojel®, etc.)	Pregnancy class B1
Magnesium hydroxide (Maalox®, Riopan®, etc.)	B1
Calcium carbonate (Tums®, Rolaids®, Alka-Mints®)	B1
H2 blockers neutralize gastric pH and decrease gastric volume	
Cimetidine (Tagamet®) 400 mg q.i.d. or 800 mg b.i.d.	B2
Ranitidine (Zantac®) or famotidine (Pepcid®) 150 mg b.i.d./10 mg b.i.d.	B1
Nizatidine (Axid®)	C
Sucralfate coats mucosa and there is possible aluminum absorption	B1
Motility agents increase gastric emptying and LES pressure	
Metoclopramide (Reglan®) 10–15 mg q.i.d., 30 min prior to meals +hs	B
Cisapride (Propulsid®) 10 mg q.i.d., 15 min prior to meals and hs	C
Proton pump inhibitors suppress gastric acid secretion	
Omeprazole (Prilosec®)	C
Lansoprazole (Prevacid®) 20 mg q. daily	C
Pantoprazole (Protonix®) 40 mg q. daily	B
Esomeprazole (Nexium®)	C

Precautions

Gaviscon®, an antacid, with increased dosages can be associated with silicaceous nephrolithiasis, hypotonia, respiratory distress and cardiovascular problems in the fetus

Cimetidine has an increased effect of theophylline, warfarin, Dilantin® and lidocaine. It is used as a pre-op medication to prevent gastric acid aspiration (Mendelssohn's syndrome). It is slow to cross the placenta
It has antiandrogenic effects (800 mg b.i.d. or 400 mg q.i.d.)
Carafate® – dose 1 g q.i.d. Coats ulcer crater and promotes healing. As effective as H_2 blocker in relieving gastroesophageal reflux but it is costly. It also has bioavailable aluminum and has been associated with fetal death, abnormal skeletal growth and impaired hearing and memory in treated offspring of rats
Metoclopramide can also be used as a pre-op to increase gastric emptying, decrease emesis and increase lactation. Side-effects can include anxiety, insomnia, hallucinations and dystonic symptoms
Cisapride can be used to treat patients with nocturnal heartburn due to reflux. It releases endogenous acetylcholine and stimulates gas motility. Use only if potential benefits justify potential risks
Proton pump inhibitors help heal erosive esophagitis

If symptoms persist

Further testing needs to be done to rule out bleeding, esophageal strictures, Barrett's esophagus, pre-cancer or cancerous conditions
Endoscopy can be performed using Demerol, Versed®, Valium or lidocaine 10% spray. Try to avoid barium studies with fluoroscopy

hCG

Human chorionic gonadotropin. The syncytiotrophoblast is responsible for the production of hCG

Level of hCG to see a SAC using a vaginal probe should be	1500
Level of hCG to see a SAC using an abdominal probe should be	6000
Level of hCG to see FETAL ACTIVITY using either probe should be	10 000

Phantom hCG phenomenon – false high levels of LH or a substance in blood that interferes with the hCG immunoassay. These substances may represent heterophilic antibodies, human antimouse antibodies (HAMA) or other antibodies to rabbit, goat or sheep immunoglobulin, non-specific protein binding or hCG-like substances. Because these are large glycoproteins, they are not excreted in urine. If phantom hCG is suspected – before treating an ectopic or gestational trophoblastic disease, consider performing both a urinary hCG assay and another type of serum hCG assay in a reference lab prior to initiating therapy, thereby avoiding a potentially disastrous situation for the patient and risk of liability for doctor

HELLP

Diagnosis and definition

Hemolysis	Abnormal peripheral smear	
	Bilirubin	1.2 mg/dl
Elevated liver enzymes	SGOT	> 72 IU/l
	Lactate dehydrogenase (LDH)	> 600 IU/l
Low platelets	Platelet count	< 100 000

Assessment and stabilization

(1) If DIC is present, correct coagulopathy
(2) Provide antiseizure prophylaxis with magnesium sulfate
(3) Treat severe hypertension
(4) Transfer to tertiary care center if appropriate
(5) Perform computer tomography or ultrasound of the abdomen if subcapsular hematoma of the liver is suspected

Evaluate fetal well-being
Evaluate fetal lung maturity if < 35 weeks' gestation
(1) If mature, induce delivery
(2) If immature, give steroids, then allow for delivery
(3) Deliver if abnormal fetal assessment
(4) Deliver if progressive deterioration in maternal condition

HEMATURIA

Definition

Presence of blood in urine (isolated hematuria) produced by bleeding in the urinary tract from urethra to renal pelvis

Total hematuria: occurs evenly throughout voiding (blood mixed fully with urine); suggests bleeding source proximal to bladder
Initial/completion hematuria: occurs at beginning or end of micturition; suggests bladder or urethral origin

Causes

(1) Urinary calculi
(2) Benign/malignant neoplasm
(3) Infection
(4) Tuberculosis
(5) Trauma
(6) Renal disease

Radiologic studies

(1) IVP, renal ultrasound – evaluate for hydronephrosis, renal/ureteral stones
(2) CT, renal arteriography – sometimes necessary to disclose certain lesions (cysts, tumors)
(3) Retrograde pyelography – when IVP not possible (Cr >1.5)

Diagnostic procedures

(1) Cystoscopy – refer to Urology
(2) Renal biopsy – refer to Nephrology

Diagnosis of hematuria

Urinalysis (midstream)
RBCs → Cath excludes vaginal or uterine bleeding
RBCs → urine culture (most frequent cause of hematuria after age of 20 is acute UTI)
RBCs → plus do second a.m. urine specimen for cytologic analysis to rule out precancerous condition
Rule out Stone, Hematologic, Infectious and/or Trauma as etiology

HEMOGLOBINOPATHIES

Genetic screening

(1) Electrophoresis – appropriate initial lab test
(2) Solubility testing – valuable test for rapid diagnosis of sickle cell disease
(3) MCV – recommended for patients at increased risk for thalassemia
(4) When screening indicated – both partners should have red cell indices and Hgb electrophoresis as primary tests

If MCV decreased → increased risk for α- or β-thalassemia

If MCV < normal:
Fe^{+} deficiency absent, then do DNA testing
Electrophoresis absent for β-thalassemia
DNA testing will look for α-globin gene deletions
Remember, hemoglobin electrophoresis is the primary screen

HEMORRHAGE IN OBSTETRICS

Postpartum hemorrhage

Definition

Loss of > 500 cc of blood during delivery
Underdiagnosed (~40% lose > 500 cc/5% lose >1000 cc)
Early – within 24 h after delivery
Late – 24 h to 6 weeks after delivery

Etiology

Uterine vs. extrauterine

Uterine

(1) Atony – over-distension (hydramnios, multiple gestation), temporal (rapid/prolonged labor), macrosomia, high parity, chorioamnionitis, tocolytics ($MgSO_4$, terbutaline), prolonged oxytocin administration, halothane anesthesia
(2) Rupture – previous uterine surgery, internal podalic version, breech extraction, obstructed labor (esp. high parity/multi-gestational), abnormal fetal presentation, mid-forceps rotations
(3) Inversion – complete vs incomplete

Extrauterine

(1) Trauma – (cervical/vaginal and/or rectal lacerations), forceps, macrosomia, precipitous labor, episiotomy
(2) Hematoma – vulvar (subacute volume loss/pain), vaginal (severe rectal pressure), retroperitoneal (least common, but most dangerous/no warning signs)
(3) Retained placental fragments – accreta, increta, percreta, abnormalities (succenturiate lobe)
(4) Coagulopathy – obstetric conditions (abruption, amniotic fluid embolism, pre-eclampsia, retained dead fetus). Medical conditions (acquired/inherited coag disorders, autoimmune thrombocytopenia, anti-coagulant use)

What % of maternal deaths are due to hemorrhage?	1/8
What % of blood volume is noted by 30 weeks' gestation?	40%
How many milliliters of blood per minute flows at term?	600 ml
What drop in hematocrit defines hemorrhage for vaginal delivery?	500 ml or 10% drop
If the parity is > 7, there is how many times the risk of uterine rupture?	20 x

Transfuse if the EBL is > 1000–1500 ml

Classification of hemorrhage

Hemorrhage class	*Acute blood loss*	*% lost*	*Response*
1	900 ml	15	Asymptomatic
2	1200–1500 ml	20–25	Tachycardia
3	1800–2100 ml	30–35	Hypotension
4	>2400 ml	40	Shock

Class 3 also has worsening tachycardia with cool extremities while class 4 may cause oliguria/anuria

Postpartum blood loss is often clinically underestimated by 30–50%
Early bleed → atony, retained POC, lacerations
Late bleed → subinvolution, retained POC, endomyometritis
Think coagulation defects if abruption, fetal demise, PIH, AFE, sepsis
Urine output – most accurate method of determining volume depletion

Treatment

Uterine massage or compression→

Pitocin (oxytocin) 20 mIU in 1000 ml IV →

Methergine (methylergonvine) 0.2 mg IM or IV→

Hemabate® (15 methylprostaglandin $F_{2\alpha}$) IM or intramyometrially in dose of 250 µg) every 15–90 minutes IM or IU or $PGF_{2\alpha}$

Dinoprostone (PGE_2) 20 mg PR q 2 hours→

Cytotech (misoprostol) 600–1000 µg PR or PO single dose

Angiographic uterine arterial embolization successful 80–95%

Activated factor VII (rF-VIIa) works well in severe postpartum hemorrhage when other interventions fall short
Give 3 doses of rF-VIIa (200 µg/kg after initial 8 units of RBCs infused then 100 µg/kg 1 hour later and 100 µg/kg 3 hours later
Use rG-VIIa judiciously … the cost is $1400/g

Uterine artery ligation successful 80–92%
Hypogastric (int iliac) artery ligation is successful 50% or 90%*
*Ligation of ascending branch of uterine artery controls 90% of patients with pelvic bleeding according to *Prolog*

Blood component therapy

Component	*Contents*	*Volume*	*Anticipated effect (per unit)*
PRBCs	RBC, WBC, plasma	300 ml	Increase Hgb by 1 g/dl
Platelets	Platelets +	50 ml	Increase plt ct by 7500
FFP	Fibrinogen, AT III,	250 ml	Increase fib by 10 mg/dl
Cryo	Fib, factor VIII, von Willebrand factor, factor XIII	40 ml	Increase fib by 10 mg/dl

Hysterectomy

PRBCs (250-ml units). Each unit increases Hct by @3%
Risk of transfusion per 1 unit of PRBCs:
HIV 1/150 000–1/1 000 000
Hepatitis B 1/50 000
Hepatitis C 1/3300
Fatal reactions 1/100 000 per unit
FFP (250-ml units). Each unit increases fibrinogen by @ 25 mg/dl
Use in massive hemorrhage with DIC or if levels of fibrinogen are < 100 mg/dl
Cryoprecipitate (15-ml units). Give if HYPOFIBRINOGENEMIC. Has fibrin, Von Willebrand factor, 8, 13
Platelets – usually 6–10 units are used at a time. Each unit increases plt count by @5–10 000 plts
Consider platelet transfusion for surgery if plt count is < 50 000
For SVD, platelets need to be > 20 000

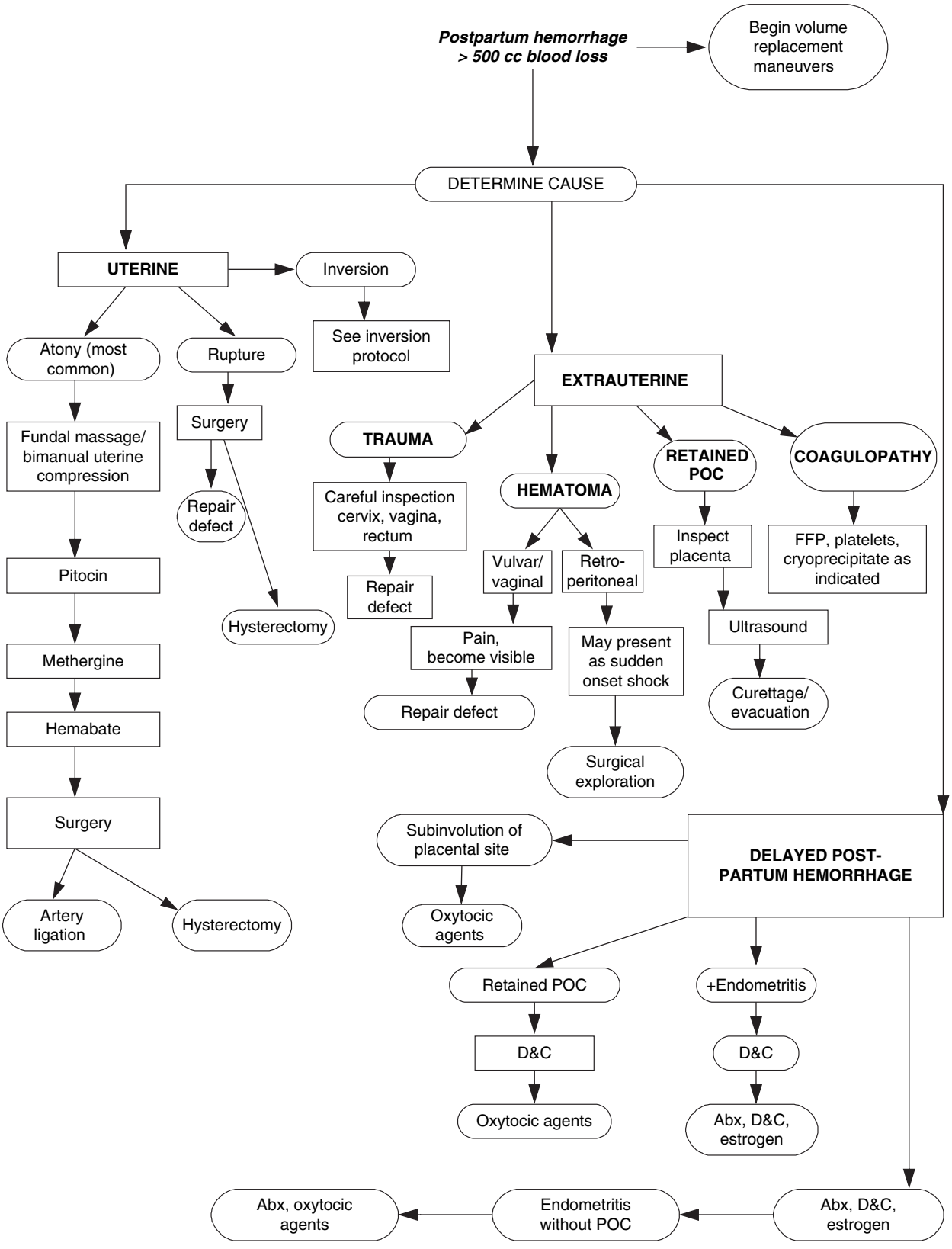
There is a 44-fold increase in maternal death from obstetric hemorrhage in Jehovah's Witness patients. (Singla AK, Lapinski RH, Berkowitz RL, *et al*. Are women who are Jehovah's Witnesses at risk of maternal death? *Am J Obstet Gynecol* 2001;185:893–5)

Delayed postpartum hemorrhage

> 24 h postpartum

Etiology Subinvolution of placental site, retained POC, endometritis

Management Pitocin, Methergine, Hemabate, antibiotics (endometritis), r/o coagulopathy, curettage (if necessary), may attempt angiographic embolization prior to surgery/hysterectomy


Postpartum hemorrhage
> 500 cc blood loss
Begin volume replacement maneuvers
DETERMINE CAUSE
UTERINE
Inversion
See inversion protocol
Atony (most common)
Rupture
Fundal massage/ bimanual uterine compression
Surgery
Repair defect
Hysterectomy
Pitocin
Methergine
Hemabate
Surgery
Artery ligation
Hysterectomy
EXTRAUTERINE
TRAUMA
Careful inspection cervix, vagina, rectum
Repair defect
HEMATOMA
Vulvar/ vaginal
Retro-peritoneal
Pain, become visible
Repair defect
May present as sudden onset shock
Surgical exploration
RETAINED POC
Inspect placenta
Ultrasound
Curettage/ evacuation
COAGULOPATHY
FFP, platelets, cryoprecipitate as indicated
DELAYED POST-PARTUM HEMORRHAGE
Subinvolution of placental site
Oxytocic agents
Retained POC
D&C
Oxytocic agents
+Endometritis
D&C
Abx, D&C, estrogen
Abx, D&C, estrogen
Endometritis without POC
Abx, oxytocic agents

HEPARIN (LOW MOLECULAR WEIGHT)

Easier administration, less need for lab monitoring, less risk of hem, but more costly by 4–6 x. Can be given SC once or twice daily without monitoring. Has longer half-life than unfractionated heparin. Inadequate info to recommend for pregnant women with mechanical heart valves. Like unfractionated heparin there is no greater risk of bone demineralization. Does not cross placenta

Advantages

(1) Longer half-life
(2) More predictable dose–response relationship
(3) Decreased risk of thrombocytopenia
(4) Decreased risk of hemorrhagic complications

Disadvantages

(1) 4–6 x more costly
(2) Ease of administration – less monitoring
(3) Inadequate information to use with pregnant females with mechanical heart valves

Other points

(1) Does not cross placenta (like unfractionated heparin)
(2) Can be given SC once or twice daily without monitoring
(3) More predictable dose–response relationship
(4) Can be continued throughout L&D or C-section. PTT and PT are not helpful – should not be obtained

HEPATITIS

A 1/3
RNA, fecal–oral, IgM, give vaccine + immunoglobulin to sex + household contacts. Vaccine contraindicated with other live viruses
Perinatal transmission does not occur. Chronic carrier state does not exist

B 40–45%
DNA, parental, perinatal, sexual; Hep B surface Ag (HbeAg increased viral load) HBIG + vaccine
Perinatal transmission with + HBsAg 10–20%
Perinatal transmission with + HbeAg and HbsAg 90%

C 10–20%
RNA, post-transfusion (90%). Most common blood-borne infection in US – Anti-C ab, no vaccine available
Perinatal transmission 10–44%

Diagnosis

Liver Bx Rx: Ribitron 8 a.m. and 3 p.m. Interferon A injection 3 x per week
• Women aged 30–40 are the highest of any age range to contract HCV at 3%
• Target women given blood transfusions prior to 1
BREASTFEEDING – may ? increase risk of transmission to baby/major factor is viral load at birth
Transdermal patch (Alora)

For ERT with *liver disease*	Category X
0.05 mg/day	1.5 mg estradiol
0.75 mg/day	2.3 mg estradiol
0.1 mg/day	3 mg estradiol

Apply to abdomen, hip or buttock twice weekly
Do not use – undiagnosed bleeding, known or suspected pregnancy, known or suspected breast cancer, estrogen-dependent neoplasia, thromboembolic disorder, allergy

D
Coinfection (acute hep B + D)
Superinfection (chronic hep B with acute hep D)

E
Rare in the USA. Similar to A

G
Associated with chronic viremia > 10 years with chronic B or C

Hepatitis B in pregnancy

DNA virus (Dane particle)
3 principal antigens (HbsAg, HbcAg, HbeAg)
Acute infection 1–2/1000 pregnancies
Chronic infection 5–15/1000 pregnancies
Transmitted parenterally or by sexual contact
Risk factors
- – History of IV drug abuse
- – History of sexually transmitted disease
- – Multiple sexual partners
- – Health-care/public safety career
- – Household hepatitis B carrier
- – Work/treatment in hemodialysis
- – Bleeding disorder (recipient of blood products)

Acute infection mortality = 1% (85–90% complete resolution)
Chronic infection in 10–15% (15–30% active viral DNA replication)
Perinatal transmission 10–20% of HepBsAg seropositive
(90% if mother HbsAg and HbeAg positive)

Clinical manifestation

Symptoms:	malaise, fatigue, anorexia, nausea, RUQ/epigastric pain
Signs:	jaundice, upper abdominal tenderness, hepatomegaly, dark urine, alcoholic stool (fulminant hepatitis = coagulopathy, encephalopathy)

Diagnosis

Laboratory tests – marked increase ALT, AST, serum bilirubin (severe hepatitis = coagulation abnormalities, hyperammonemia)
Liver biopsy – rarely indicated

Specific serology

Hepatitis virus	*Acute*	*Chronic*
A	Hep A IgM ab	None
B	HBsAg	HBsAg
	HBeAg (high infectivity)	
	HBcAg IgM ab	
C	Hep C ab	Persistent hepatic dysfunction
D	Hep D Ag	Hep D Ag
	Hep D IgM ab	Hep D IgG ab

Management

Supportive

Hospitalization for severe cases (encephalopathy, coagulopathy, etc.)
Mild to moderate illness may be managed as out-patient
- – reduce activity
- – avoid upper abdominal trauma
- – maintain nutrition/hydration
- – avoid intimate contact with household or sexual partners until immunoprophylaxis initiated

Specific immunotherapy

Hepatitis A	*Vaccine*	– investigative trials
	Immunoglobulin	– pre/post exposure prophylaxis for travel to endemic areas (safe in pregnancy)
Hepatitis B		– cannot alter natural course once patient is clinically ill
	Vaccination	– indicated for women with risk factors – susceptible pregnant patients targeted for vaccine
	Immunoglobulin	– exposure to Hep B prior to vaccination exposure via sexual contact – single dose HBIG within 14 days exposure via injury (needle stick, etc.) – immediate dose followed by second dose 1 month later
	Passive/active immunization especially important in pregnant pts (reduces perinatal transmission 85–95%)	
Neonatal immunoprophylaxis		
	Vaccination recommended for all newborns (CDC) 1st vaccination = birth to 12 h 2nd vaccination = 1 month 3rd vaccination = 6 months	
	Immunoglobulin	– indicated for newborns of HbsAg positive or unknown status mother – HBIG 0.5 ml IM = birth to 12 h (Hep B screening recommended for all pregnant women)
Hepatitis C/D	No antiviral agents available (Measures to prevent Hep B effective in preventing Hep D)	

HEREDITARY NON-POLYPOSIS COLORECTAL CANCER

Accounts for about what % of all colorectal malignancies and is most commonly associated with endometrial cancer?	5%
This is the most heritable colorectal cancer. Normal lifetime risk of developing colorectal cancer is	2%
If the same 70-year-old woman has an HNPCC-associated mutation	82%
Normal 70-year-old woman lifetime risk of developing various cancers vs one with an HNPCC mutation:	
Endometrium	1.5% vs 60%
Ovary	1% vs 12%
Urinary tract	< 1% vs 4%
Small intestine	< 1% vs 5%
Biliary tract	< 1% vs 2%
Stomach	< 1% vs 13%
Brain	< 1% vs 3.7%

Use early screening such as colonoscopy, pelvic ultrasound, endometrial biopsy and serum CA-125. Patients must meet ALL of the Amsterdam criteria II or any ONE of the Bethesda criteria-modified. (For criteria, refer to source chart in Powell MA, Mutch DG. *Contemporary OB/GYN*, Dec 1, 2001: 86)

HERPES

HSV-1 orolabial	DNA virus	15–20% genital
HSV-2 genital	DNA virus	80–85% genital
Three types of infections	(1) Primary (2) Initial, non-primary (3) Recurrent infection	

Infection in pregnancy

Most infections in pregnancy are recurrent with prevalence of 1%
Rare for virus to cross placenta → can infect fetus across birth canal
Greatest risk occurs during primary maternal infection 40%

Diagnosis

History and physical (NOT cultures) are most commonly used
Culture of lesion is Gold Standard. (Best not to tell patient a definitive diagnosis unless certain prior to having definitive culture results.) Can use Tzanck test to look for 'Giant Cells'. Can use *POCkit HSV 2 Rapid Test* if physician's office is classified by CLIA as 'Moderately Complex Lab' (Call 877-776-2548)

Classic signs and symptoms
Other common signs ands symptoms

(1) Painful or pruritic vesicles clustered on the labia and/or buttocks
(2) Dysuria
(3) Tender inguinal lymph nodes
(4) Cervical ulcerations

Tzanck smear

Rapid and inexpensive test
(1) Scrape opened vesicle on slide
(2) Giemsa, Sedi or Wright's stain is applied
(3) Characteristic cytopathology:
  (a) Multinucleated giant cells
  (b) Atypical keratinocytes
  (c) 'Ground glass' cytoplasm

Viral HSVII culture

Obtain when vesicle is wet. 90% (only 25–30% recovery with crusted lesions)
• Also test for syphilis, GC/*Chlamydia*, bacterial vaginosis and *Trichomonas*

Educate patient

(1) Warn about spread
(2) Advise use of condoms
(3) Recurrences
(4) Advise about danger of perinatal transmission, asymptomatic shedding especially increased with prolonged first-degree outbreak and frequent symptomatic recurrences

Treatment in pregnancy

(1) Overt lesions regardless of time since ROM C-section
(2) If asymptomatic with *no* prodromal symptoms and/or *no* vesicule lesions vaginal delivery
(3) Treat maternal life-threatening HSV with IV acyclovir
• Primary HSV in pregnancy → antiviral therapy
• Patient with primary HSV with ACTIVE LESIONS → C-section
• Patient > 36 weeks with primary HSV → antiviral therapy
• Patient with recurrent HSV with active lesions or symptoms → C-section
• PTL or PROM with active HSV → expectant management
• Patient > 36 weeks with increased risk of recurrent HSV → antiviral therapy
• No active lesions or symptoms during labor → vaginal delivery

General antiviral treatment

Primary infection	acyclovir 400 mg orally t.i.d. for 7–10 days
Recurrent infection	acyclovir 400 mg t.i.d. for 5 days or 800 mg b.i.d. for 5 days or Valtrex® 500 mg orally b.i.d.
	Famciclovir 1000 mg b.i.d. for 1 day initiated within 6 hours of symptoms
Frequent recurrences	acyclovir 400 mg orally for 6 years
Herpes zoster	Valtrex 1 g orally t.i.d. or acyclovir 800 mg orally 5 times per day or Famvir® 500 mg orally t.i.d.
Suppression of HSV II	Valtrex 500 mg to 1 g daily acyclovir 400 mg orally b.i.d.

Important points to remember

Primary infection – viral shedding occurs for	2–3 weeks
Recurrent attacks – viral shedding occurs for	5–6 days
Virus stays dormant in – dorsal root ganglia of	S2, S3 and S4
What % of partners will contract the disease?	75%
What % HSV II is genital?	80–85%
Greatest risk to fetus occurs during PRIMARY maternal infection	40%
Risk if mother has recurrent infection (if overt lesion present with vaginal delivery) is	< 1%

There seems to be a protective effect of passive acquired maternal antibodies with lower viral inoculum associated with asymptomatic infection
HSV is acquired by what % of seronegative women during pregnancy? 2%
HSV with PPROM (rupture of membranes < 37 weeks' gestation)
Risk of neonatal HSV is 19%
Therefore, give acyclovir prophylaxis to patient with history
PPROM < 30–32 weeks' gestation and stable – treat expectantly.
75% of patients with PPROM, regardless of management, deliver @ within 1 week
Patients with PPROM – intra-amniotic infection occurs in 13–60%
Postpartum infection occurs in 2–13%
Incidence of infection increases with decreased gestational age at time of membrane rupture

General treatment

Primary infection	acyclovir 400 mg t.i.d. 7–10 days
Recurrent infection	acyclovir 400 mg t.i.d. 5 days or 800 mg 5 days
Frequent recurrences	acyclovir 400 mg b.i.d. for 6 years

HIDRADENOMA PAPILLIFERUM

Benign sweat gland tumors arising from labia minora and majora
Small firm tumors with a pointed center. Microscopic – can be mistaken for adenocarcinoma
Treatment – surgical excision is treatment necessary for both diagnosis and cure

HIGH-GRADE SIL

Atypical nuclei take up the majority of the cell. Cells are smaller than epithelial cells
Compared to LGSIL – koilocytosis with perinuclear clear areas. HPV.
Coarsening of the chromatin or squamous cell cancer of cervix – keratin pearls, not multifocal originates at T-zone and makes up what % of cancer of the cervix? 90%
Vs adenocarcinoma of cervix – multifocal/skip lesions, occult lesions, more aggressive than squamous cell cancer and makes up this % 10%

HIRSUTISM

Suspect ovarian tumor if testosterone level is	> 200 ng/dl
If testosterone ≤ 200 ng/dl, draw DHEA	
Treat symptoms if DHEA	< 500 µg/dl
Exclude CAH if DHEA	500–700 µg /dl
Obtain MRI to rule out adrenal tumor if DHEA	> 700 µg /dl
Exceptions to algorithm	
Cushing's syndrome 11 p.m. give dexamethasone	1 mg
Then at 8 a.m. draw serum cortisol – should be	< 5 µg/ml
21-Hydroxylase deficiency 8 a.m. 17-OHP, value should be	> 4 ng/ml
Hyperprolactinemia – draw serum prolactin	

Spironolactone is effective in stabilizing what % of unwanted hair growth? 80%

Etiology

Anovulation, excess androgen production by ovaries and adrenal (least common)

Prevalence of causes of hirsutism

PCOS	70–85%
Idiopathic hirsutism	5–15%
21-Hydroxylase deficient non-classic adrenal hyperplasia	1–8%
HAIRAN (hyperandrogenic insulin resistant acanthosis nigricans) syndrome	3–4%
Ovarian androgen-secreting tumors	0.3–0.1%
Drug-induced	0.5–1.0%

Differential

Drug induced, intersex (amb gent), ovary (PCO, tumors), adrenal (tumors, Cushings, CAH), peripheral (idiopathic) or pregnancy (luteoma, etc.)

Treatment

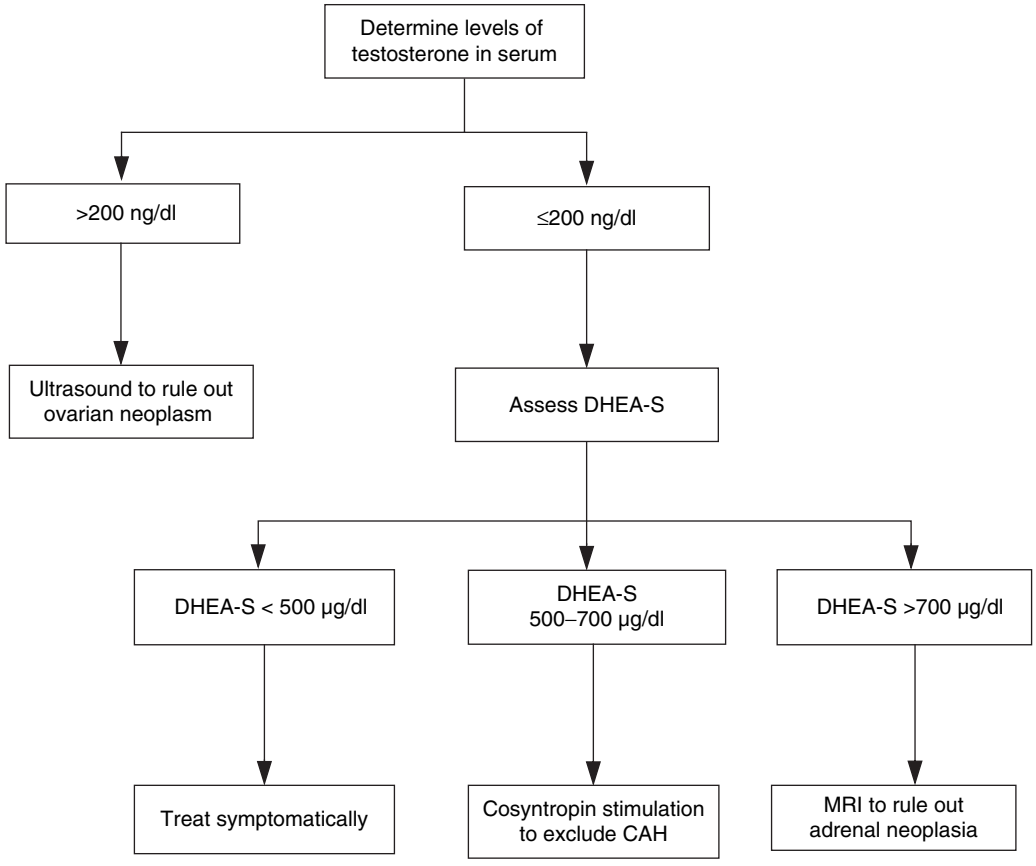
Increased testosterone — OCPs
Increased DHEA-S (< 5 µg /ml) — OCPs
Increased DHEA-S (> 5 µg /ml) — dexamethasone 0.25–0.5 mg p.o. hs
Increased testosterone + increased DHEA-S (7) — OCPs + Dex
Increased 3α-androstenediol glucuronide – spironolactone 100–200 mg p.o. daily

Ovarian – OCPs, progestins (DMPA), GnRH agonist (Lupron), Antiandrogenism (cyproterone acetate – Diane®), spironolactone, ketoconazole, corticosteroids
Peripheral – cyproterone acetate, spironolactone 100–200 mg daily, progesterone (topical), OCPs and 5α-reductase inhibitors

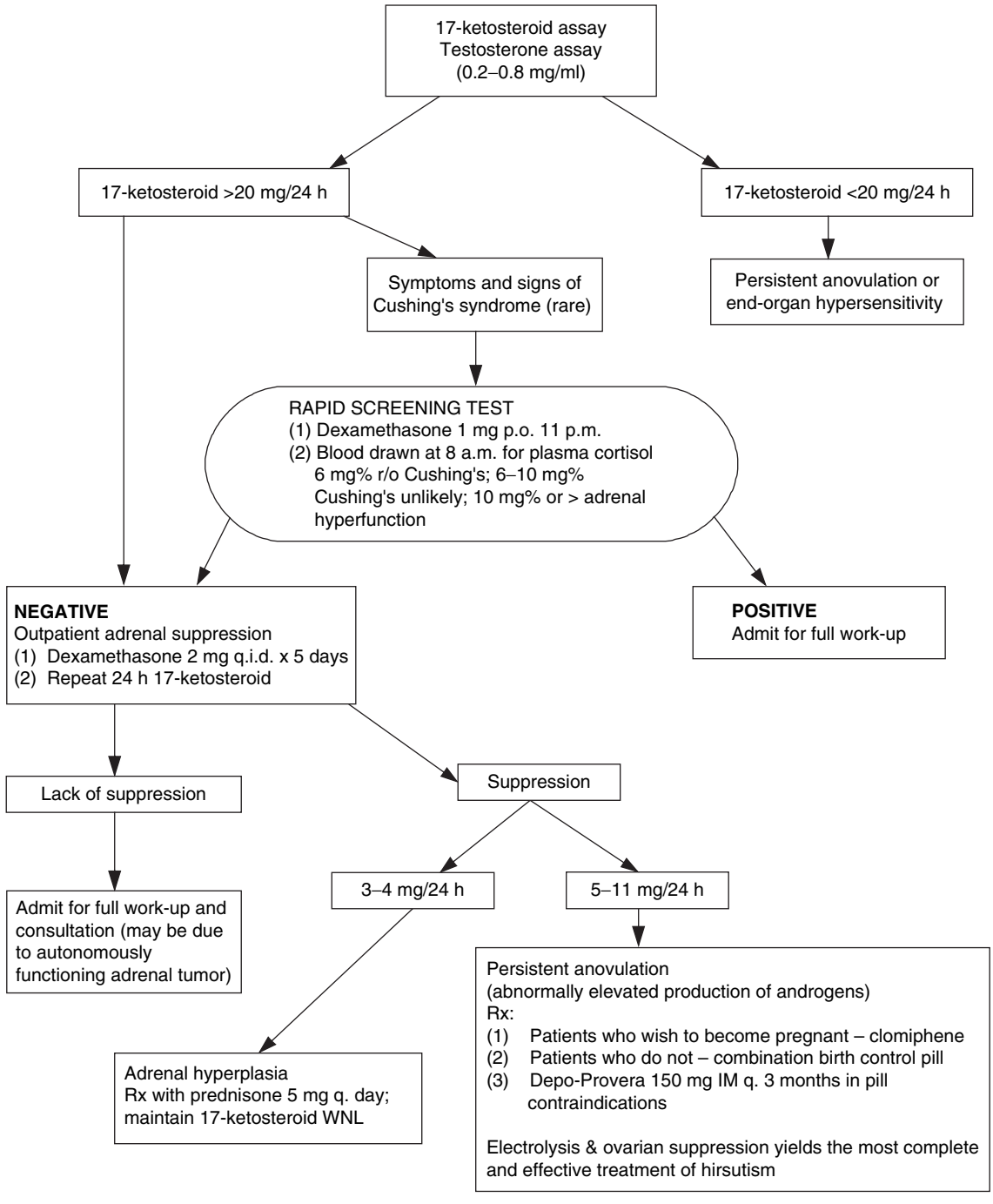
How some treatments work

OCPs – progestin decreases LH, estrogen increases SHBG.
Progestins of some OCPs decrease peripheral 5α-reductase activity in skin. There is also some increased metabolic clearance of testosterone by hepatic enzymes
May improve with additional treatment using antiandrogens or 5α-reductase inhibitors
Spironolactone (Aldactone®) 50–200 mg daily reduces 5α-reductase
Flutamide (Eulexin®) 250 mg one t.i.d. (antiandrogen)
Finasteride (Proscar®) 5–7.5 mg daily reduces 5α-reductase
Eflornithine HCl cream 13.9% may be useful only for the removal of unwanted facial hair
After 6 months, if still hirsute – electrolysis, shaving, waxing, laser depilation

Laboratory findings



Hirsutism protocol



HIV

Physician's responsibilities in regard to HIV

(1) Offer voluntary and confidential HIV testing to *all* women
(2) Individual female reproductive choices should be respected regardless of her HIV status
(3) A physician may breech confidence if it is clear that there is a high probability of harm to the uninformed individual
(4) HIV-positive patients are entitled to same privacy as other patients
(5) Postpartum mother may refuse to inform pediatrician

Important points about HIV

Scheduled C-section to decrease vertical transmission of HIV whether or not the patient is receiving ZDV therapy
Risk of vertical transmission without ZDV therapy is 25%
Scheduled C-section at 38 weeks' gestation is recommended for HIV-infected mother to decrease likelihood of onset of labor or ROM before labor
Amniocentesis to determine fetal maturity should be AVOIDED

Treatment of HIV in pregnancy

Antepartum treatment is zidovudine 5 x per day x 15 weeks with each dose 100 mg
Intrapartum treatment is zidovudine loading dose over 1 h IV at dose of 2 mg/kg
Then until delivery give 1 mg/kg/h
Neonatal treatment is zidovudine p.o. q. 6 h x first 6 weeks starting 8–12 h postpartum at dose 2 mg/kg
Avoid breastfeeding

Perinatal transmission of untreated HIV + female is 30%
Treated HIV + female with AZT is 8–10%
Treated HIV + female with AZT and Ob care 4–5%
Treated HIV + female with AZT and C-section 1–2%
How long does it take for antibodies to the HIV virus to develop? 6–12 months

hMG

Hyperstimulation syndrome incidence is 1%
In order to trigger ovulation, the leading follicle should be how many mm in diameter? 16–20 mm
The follicle typically enlarges how many mm/day allowing anticipation of ovulation? 2–3 mm
The egg is capable of being fertilized for how many hours? 24 h
Probability of fertile couple becoming pregnant within one menstrual cycle is 25%
hMG can be Pergonal® LH + FSH or Metrodin® FSH
NO PELVIC or ABDOMINAL EXAMS should be done if hyperstimulation is suspected

HOMOCYSTEINEMIA

Hyperhomocysteinemia has been linked to osteoporosis, Alzheimer's disease, vascular dementia, and decreased cognitive function

At present, routine supplementation of folic acid, pyridoxine, and cobalamin is safe and may potentially reduce osteoporotic fractures and CVD events

Homocysteine levels and mortality

Homocysteine level, μmol/l	Mortality odds ratio
9–14.9	1.9
15–19.9	2.8
≥20	4.5

HORMONES

Polypeptide hormones	Prolactin, GH (growth hormone) and HPL (human chorionic somatomammotropin)
Glycoprotein hormones	Thyrotropin (TSH), FSH, LH, hCG
Hormones produced by the corpus luteum	Progesterone, estrogen, inhibin and relaxin
Know the functions of these hormones	Inhibin (1) Suppresses FSH release (2) Produced by gonadotropin-dependent granulosa cells Activin – stimulates FSH release Relaxin (1) Modulates function of corpus luteum (2) Makes the uterus quiescent Follistatin – also suppresses FSH release
Equivalents of hormones	Conjugated estrogen (0.525 mg) – ethinylestradiol (0.005–0.010 mg) Premarin 0.625 mg – Ogen® (estropipate) 1.2 mg

HORMONES AND HORMONE REPLACEMENT THERAPY

BONE FRACTURE

- What % of bone is lost at the spine using Lupron (mostly recovered after discontinuance)? 5%
- What % of bone is lost using DMPA? 8%
- What % bone is lost after the first 3–5 years after menopause? 20–25%

- Estrogen replacement therapy has been proven to increase bone density > 49%

BREAST CANCER

- 1/25 women die of breast cancer – what is the ratio of women who die from CAD? ½

  In regard to the WHI study and breast cancer, there was a slight increase noted at year 4, with a trend toward a later decline in the number of cases. In addition, the confidence intervals for the hazard ratios for breast cancer crossed 1 in both the unadjusted and adjusted analyses and are therefore not considered valid. No increased risk of breast cancer *in situ* was apparent. Breast cancer risk seems to increase with EPT use beyond 5 years

  In regard to recurrent breast cancer and the use of HRT, multiple retrospective studies have not demonstrated an increase in the risk of breast cancer recurrence with the use of HT. Some studies, however, have shown that by increasing mammographic density, HT reduces the sensitivity of mammography

  Top 3 risk factors for breast cancer: obesity, no daily exercise, and more than 2 alcoholic drinks daily

CORONARY ARTERY DISEASE

- The death rate from CAD is 50%

  The latest data support the idea that among recently menopausal women, estrogen treatment does not increase, and may decrease, the risk of cardiovascular disease. The WHI showed that over 6.8 years of follow-up, there was a trend for reduced myocardial infarction and death from coronary disease if women were on ERT (0.625 mg of equine estrogen daily). There was also a statistically significant decrease in coronary artery bypass surgery or percutaneous coronary artery intervention

- HRT, in some studies, reduces the rate of MI by 1/3 to 1/2
- The use of HRT at the time of MI was associated with approximately a 35% reduction in mortality. (Shlipak MG, Angeja BG, Go AS, *et al*. Hormone therapy and in-hospital survival after myocardial infarction in postmenopausal women. *Circulation* 2001;104: 2300–4)
- However, recent research from WHI indicates that there may be a slightly increased risk of heart attack and stroke with the use of HRT (see summary chart of WHI below). Investigators recommended that CEE/MPA not be initiated or continued for the primary prevention of CHD
- Estrogen alone may be beneficial for the cardiovascular system, whereas adding MPA may increase risks

As expected, higher daily estrogen metabolite levels were associated with a favorable CVD risk profile in the subcohort analysis from SWAN

- Women who began HRT within 5 years of menopause had less heart disease than women who started HRT more than 5 years after menopause

STROKE Among hysterectomized women in the WHI, the main risk of estrogen treatment was a small increase in the risk of stroke

HOT FLASHES Estrogen treatment is clearly effective in reducing vasomotor symptoms

ROUTES OF ADMINISTRATION

- Nonoral routes of administration of ET/EPT may offer advantages and disadvantages, but the long-term risk–benefit ratio has not been demonstrated.

OTHER FACTS

- What % of women receive HRT after menopause? 16%
- What % of women discontinue the HRT after 1 year of use? 50%
- Normal range for serum estradiol concentration in postmenopausal pt not on HRT is 10–20 pg/ml

	10 000 women/year taking placebo	*10 000 women/year taking combination HRT*	*Difference per year*
Breast cancer	30	38	8 more women with breast cancer
Heart attacks	30	37	7 more women with heart attacks
Strokes	21	29	8 more women with strokes
Blood clots	16	34	18 more women with blood clots
Colorectal cancer*	16	10	6 fewer women with colorectal cancer
Hip fractures	15	10	5 fewer women with hip fractures

*HRT is not indicated for the prevention or treatment of colorectal cancer or hip fractures

Menopause with ERT or HRT

Estrogen replacement therapies (ACOG still recommends the use of ERT or HRT for short-term relief of menopausal symptoms)

Active ingredients	*Brand name*	*Strengths*	*Manufacturer**	*Minimum dosage/day***
ESTROGENS				
17β-Estradiol				
Oral	Estrace	0.5, 1, and 2 mg	Westwood-Squibb	0.5 mg
	Femtrace	0.45, 0.9, and 1.8 mg	Warner Chilcott	0.45 mg
	Gynodiol	0.5, 1, 1.5, and 2 mg	Novavax	
Transdermal patches	Estraderm	0.05, 0.1 mg(1)	Novartis	0.05 mg
	Esclim	0.025, 0.0375, 0.05, 0.075, 0.1 mg	Novartis	0.025 mg
	Menorest(3)	0.0375, 0.05, 0.075 mg(1)	Rhone	0.0375 mg
	FemPatch	0.05, 0.1 mg(1)	Parke-Davis	0.05 mg
	Climara	0.025, 0.0375, 0.05, 0.06, 0.075, 0.1 mg(2)	Berlex	0.025 mg
	Alora	0.025, 0.05, 0.75, 0.1 mg (2x wk)	Watson	0.025 mg
	Menostar	14 μg (ounce per week)	Berlex	14 μg
	Vivelle-Dot	0.025, 0.0375, 0.05, 0.075, and 1 mg	Novogyne	0.025 mg
Transdermal gel	EstroGel	0.035 (0.75 mg x 1 per day) 1 arm	Solvay	0.06 mg
Transdermal	Estrasorb	0.05 mg (two pouches) calf & thigh	Espirt→ Novavax	0.05 mg
Estropipate	Ogen	0.625, 1.25, 2.5 mg	Pfizer	0.625 mg
	Ortho-Est	0.625, 1.25 mg	Ortho	0.625 mg
Esterified estrogens	Estratab	0.3, 0.625, 1.25, 2.5 mg	Solvay	0.3 mg
	Menest	0.3, 0.625, 1.25, 2.5 mg	King	0.3 mg
Synthetic conjugated estrogens	Cenestin	0.3, 0.45, 0.625, 0.9, 1.25 mg	Duramed	0.3 mg
	Enjuvia	0.3, 0.45, 0.625, 0.9, 1.25 mg	Duramed	
	Premarin (CEE)	0.3, 0.45, 0.625, 0.9, 1.25, 2.5 mg	Wyeth-Ayerst	0.3 mg
Ethinylestradiol	Estinyl	0.02, 0.05, 0.5 mg	Schering	
Vaginal rings	Estring	2 mg (Replace q. 3 months)	Pfizer	7.5 μg/24 h
	FemRing	0.05 and 0.1 mg (Replace q. 3 mos)	Warner Chilcott	0.05 mg
Vaginal tablets	Vagifem	25 μg estradiol q. d × 2 wks then twice weekly thereafter	Novo Nordisk	25 μg
Vaginal creams	Premarin Vag.	0.01% (0.625 mg/g)	Wyeth	0.01%
	Estrace Vag.	0.01% micronized estradiol	Warner Chilcott	0.01%
	Ogen Vaginal	1.5 mg/g	Pfizer	—
Intramuscular injections				
	Depo-Estradiol	5 mg/ml estradiol cypionate	Pfizer	1–5 mg IM q 3–4 weeks
	Delestrogen	10, 20, 40 mg/ml estradiol valerate	King	10–20 mg IM q. 4 weeks.
Chlorotrianisene	Tace	12 mg	Hoechst Marion Roussel	12 mg
Estradiol pellets	N/A	25 mg	Pharmacy Center	Can vary
PROGESTOGENS				
Progestins				
Medroxyprogesterone acetate (MPA)	Provera	2.5, 5, 10 mg	Pfizer	2.5–5 mg for continuous combined, 5–10 mg for sequential
	Cycrin	2.5, 5, 10 mg	ESI Lederle	2.5–5 mg for continuous combined, 5–10 mg for sequential
	Amen	10 mg	Carnick	10 mg for sequential

Norethindrone	Norlutin	5 mg	Parke-Davis	2.5 mg
Norethindrone acetate	Norlutate Aygestin	5 mg 5 mg (2.5–10 mg × 5–10 d)	Parke-Davis Duramed	2.5 mg
Progesterones				
Micronized	N/A	50, 100, 200 mg	Compounding companies	50–100 mg for continuous combined, 200 mg for sequential
	Prometrium	100 and 200 mg (200 × 12–14 d)	Solvay	100 mg
TESTOSTERONES				
Testosterone pellets	N/A	75 mg	Barter Pharm. Co.	
Testosterone cypionate				
Estradiol cypionate IM	Depo-Testadiol	1 mg q. wk	Upjohn	
Testosterone buccal system mucoadhesive	Stiant	Apply to gum above incisor every other day to every day **This is an off-label use*	Columbia	

(Continued)

WHI preliminary findings for estrogen alone – as reported by the NIH

Outcomes	*Reported changes vs placebo after nearly 7 years*
CHD	No increased or decreased overall risk
Breast cancer	No increased risk
Stroke	Increased risk
Hip fractures	Decreased risk
Probable dementia and mild cognitive impairment	Trend toward increased risk

BIOIDENTICAL HORMONES

The position statement of the North American Menopause Society (March 2007) is that "the scientific evidence for these preparations was also reviewed and it was concluded that in the absence of safety and efficacy data for any specific preparation, the generalized risk–benefit ratio data of commercially available ET/EPT products apply equally to this group of compounded therapies. Moreover, the Panel recommended caution in use of these products in the absence of regulatory oversight of quality, purity, and batch-to-batch consistency of consistency of ingredients."

Hormone replacement therapies (continued)

Active ingredients	*Brand name*	*Strengths*	*Manufacturer**	*Minimum dosage/day***
COMBINED PRODUCTS				
	Activella	1 mg estradiol 0.5 mg norethindrone acetate	Parke-Davis	1 tablet
	Estratest FS & HS	0.625 mg esterified estrogens and 1.25 mg methyltestosterone (MT); 1.25 mg esterified estrogens and 2.5 mg MT	Solvay	1 tablet 1 tablet
	Angeliq	1 mg estradiol and 0.5 mg drospirenone	Berlex	1 tablet
	Femhrt	5 µg ethinylestradiol and 1 mg norethindrone acetate; 2.5 µg ethinylestradiol and 0.5 mg norethindrone acetate	Warner Chilcott	1 tablet 1 tablet
	Prefest	1 mg estradiol q.d. × 3 d then 1 mg/ 0.09 mg norgestimate q.d. x 3 d	Duramed	1 tablet
	Premphase	0.625 mg CEE, w/ 5 mg MPA in last 14 tablets	Wyeth-Ayerst	1 tablet
	Prempro	0.3 mg CEE and 1.5 mg MPA	Wyeth-Ayerst	1 tablet
	Prempro	0.45 mg CEE and 1.5 mg MPA	Wyeth-Ayerst	1 tablet
	Prempro	0.625 mg CEE and 2.5 mg MPA	Wyeth-Ayerst	1 tablet
	Prempro	0.625 mg CEE and 5 mg MPA	Wyeth-Ayerst	1 tablet
Transdermal patch	CombiPatch[1]	Estradiol 0.05 mg daily norethindrone acetate 0.14 and 0.25 mg (9 mm) 0.14 mg norethindrone q.d. (16 mm) 0.25 mg norethindrone q.d.	Novogyne	Twice weekly
	Climara Pro Patch	0.45 mg ethinylestradiol and 0.015 mg of levonorgestrel	Berlex	Once weekly
Vaginal ring	NuvaRing	0.120 mg etonogestrel 0.015 mg ethinylestradiol	Organon	Intravaginal ring removed at 3 weeks & 1 week break
ALTERNATIVES				
Raloxifene	Evista	60 mg	Lilly	60 mg

KEY:
[1]Change patch twice weekly
[2]Change patch once weekly
[3]Not available in the USA
*Sample listing; others available
**Some minimum dosages not available

Menopause without ERT – contraindications to ERT

Absolute contraindications	Current breast cancer
	Current endometrial cancer
	Acute DVT or evolving thromboembolic event
	Undiagnosed vaginal bleeding
Relative contraindications	History of breast cancer
	History of endometrial cancer
	History of DVT
	Chronic liver disease
	Endometriosis
	History of CVA or recent MI
	Pancreatic disease
	Fibrocystic breast disease
	Large fibroid uterus
	Familial hyperlipidemia
	Hepatic porphyria
	Hypertension aggravated by estrogen
	Migraines aggravated by estrogen

Risk factors for osteoporosis

Non-modifiable risk factors	Female sex
	Age > 65 years
	Caucasian or Oriental race
	Premature menopause (spontaneous or surgical)
	History of an atraumatic fracture
	Loss of height of >1 inch
	Family history of osteoporosis
	Chronic steroid therapy
	Coexisting medical conditions
	Hyperparathyroidism
	Hyperthyroidism
	Malignancies (e.g. myeloma)
	Cushing's syndrome
Modifiable risk factors	History of smoking
	Reduced weight for height
	Excessive alcohol consumption
	Excessive caffeine consumption
	Lack of exercise
	Diet deficient in calcium
	Diet deficient in vitamin D
	High-protein diet
	Medications
	Prolonged heparin therapy
	Chronic steroid therapy

Treatment of vasomotor symptoms without estrogen

Treatment	*Dosage/route of administration*	*Efficacy (vs. placebo)*
Steroid hormones		
Progestins		
Depomedroxyprogesterone	150 mg IMI q. 3 months	Effective
Medroxyprogesterone	20 (10–80) mg p.o. q.d.	Effective
Megesterol acetate	20 mg p.o. b.i.d.	Effective
Androgens		
4-Hydroxyandrostenedione	250–500 mg IMI q. 1–2 weeks	Possibly effective
Danazol	100 mg p.o. q.d.	Possibly effective
Synthetic steroids		
Org-OD-14 (Tibolone)	2.5 mg p.o. q.d.	Probably effective
Non-steroidal medications		
Clonidine	0.05–0.15 mg p.o. or transdermal 200 µg q.d.	Probably effective
α-Methyldopa	250–500 mg p.o. b.i.d.	Probably effective
Bellergal-Retard	Variable	Insufficient data
β-Blockers	Variable	Not effective
Clomiphene citrate	50–150 mg p.o. q.d.	Not effective
Naloxone	22 µg/min IV	Not effective
Lofexidine	0.1–0.6 mg p.o. b.i.d.	Possibly effective
Veralipride	100 mg p.o. q.d.	Possibly effective
Environmental alteration		
Layered clothing	_____	No clinical data
Moderate exercise	No clear indication of amount needed	Data support effectiveness
Avoidance of caffeine	_____	No clinical data
Avoidance of spicy foods	_____	No clinical data
Antidepressants		
Paxil, Celexa, Prozac, Effexor	Varied doses	Reduces hot flashes
Natural remedies		
Vitamin B, C or E	Variable	No clinical data
Zinc	Variable	No clinical data
Bee pollen	_____	No clinical data
Black Cohosh (Remifemin)	80 mg/d	Probably effective
Ginseng tea	_____	No clinical data
Fenugreek	_____	No clinical data
Gotu kola	_____	No clinical data
Red clover (Promensil or Rimostil)	40–160 mg daily PO	Data do not support use
Wild yam root	_____	No clinical data

- It is important to distinguish Remifemin from Remifemin Plus, which contains St John's wort as an additional product component. It also is important to distinguish between black cohosh and blue cohosh. Blue cohosh is a completely different botanical (*Caulophyllum thalictroides*), used in the past for labor induction and augmentation, and has considerable adverse and toxic potential (abortifacient, teratogenicity, coronary artery constriction, etc.)
- The result of a recent clinical trial suggest that the combination of black cohosh and St John's wort may be useful in treating both vasomotor symptoms associated with menopause as well as depression. (Uebelhack R, *et al*. Black cohosh and St John's wort for climacteric complaints: a randomized trial. *Obstet Gynecol* 2006; 107: 247–55)
- Whenever possible, use the specific brand of botanical agent studied in clinical trials
- Avoid black cohosh and soy products in women with contraindications to estrogen
- Initial data on red clover were promising, but the more rigorous meta-analysis does not support its use
- Speroff points out that black cohosh is not estrogenic, and black cohosh has no effect on menopausal symptoms. He states that, thus far, all phytoestrogen products (including soy and red clover extracts) are proving to be no different than placebo for treating hot flushes. Estrogen products continue to be the most efficacious for this purpose. The serotonin uptake inhibitor class of antidepressants is next most effective

Treatment of psychosexual issues without estrogen

Treatment	*Dosage/route of administration*	*Efficacy*
Pharmacologic treatment		
Androgens		
Methyltestosterone	2.5 mg p.o. q.d.	Possibly effective
Androgel 1% (metered dose)	1.25 mg applied daily to one arm	Possibly effective
Natural remedies		
Vitamin C	500 mg p.o. q.d./b.i.d.	No clinical data
Tryptophan	Variable	Possibly useful
Inositol	100 mg p.o. q.d.	No clinical data
Vitamin B_6	50–100 mg p.o. q.d.	No clinical data
Magnesium	Variable	No clinical data
Behavioral and/or psychological interventions	_____	Probably ineffective

Treatment of urogenital atrophy without estrogen

Prevention	*Dosage/route of administration*	*Efficacy*
Continued sexual activity	_____	Effective
Lubrication		
Water-based lubricants	_____	Useful
Petroleum-based lubricants	_____	Useful
Vegetable oils	_____	Useful
Polycarbophil	_____	Useful
Douching with yogurt	_____	Probably ineffective
Pharmacologic therapy		
Tamoxifen	20–40 mg orally daily	Probably ineffective

Prevention and treatment of cardiovascular disease without estrogen

	Dosage/route of administration	*Efficacy*
Prevention		
Environmental modification		
Smoking cessation	_____	Effective
Moderate physical exercise	_____	Effective
Control cholesterol	_____	Effective
Control hypertension	_____	Effective
Control diabetes	_____	Effective
Control weight	_____	Effective
Pharmacological treatment		
Aspirin	81–325 mg p.o. daily	Effective
Moderate alcohol consumption	Variable	Effective
Progesterone	10–15 g per day	Possibly effective
HMG-CoA reductase inhibitors	Variable	Possibly effective
Niacin	1–2 g p.o. t.i.d.	Possibly effective
Bile resins	Variable	Possibly effective
Natural remedies		
Antioxidant vitamins (vitamin E, vitamin C,β–carotene)	Variable	No clinical data
Treatment		
Pharmacological treatment		
Aspirin	81–325 mg p.o. q.d.	Effective
β-Blockers	Variable	Effective long-term
Calcium-channel blockers	Variable	Possibly effective
ACE inhibitors	Variable	Possibly effective
SERM (Evista)	60 mg p.o. q.d.	Effective
Surgery		
Conservative (angioplasty)	_____	Effective
Radical (coronary bypass, transplantation)	_____	Effective

Prevention of osteoporosis without estrogen

	Dosage/route of administration	*Efficacy*
Prevention		
Screening		
Bone mass index	_____	Useful
Environmental modification		
Smoking cessation	_____	Effective
Avoid alcohol excess	_____	Probably effective
Moderate exercise	_____	Effective
Dietary modification		
Vitamin D	800 IU p.o. q.d.	Effective
Pharmacologic treatment		
Agents that retard bone resorption		
Calcium	500–800 mg p.o. q.d. (elemental calcium)	Effective
Calcitonin	50 IU p.o. q.i.d.	Effective
Calcitriol	0.5–1 µg p.o. q.d.	Probably effective
Agents that promote bone formation		
Sodium fluoride	50–75 mg p.o. q.d.	Effective
Human parathyroid hormone		
Teriparatide (Forteo – Lilly)	20–40 µg SC q.d.	Effective
Anabolic steroids	Variable	Probably effective
Pharmacologic treatment		
Agents that retard bone resorption		
Calcium	1500 mg p.o. q.d. (elemental calcium)	Effective
Salmon calcitonin	50 U q.d./q.i.d. IV or intranasal (100 IU b.i.d.)	Possibly effective
Vitamin D analogs		
Calcitriol	0.25 µg p.o. q.d.	Possibly effective
Ergocalciferol	800 IU p.o. q.d.	Possibly effective
Cholecalciferol	800 IU (20 µg) p.o. q.d.	Possibly effective
Bisphosphonates		
Etidronate	200–400 mg p.o. q.d. x 2 weeks	
	12 weeks off	Effective
Alendronate (Fosamax – Merck)	5–20 mg p.o. q.d. or 70 mg weekly	Effective
Tiludronate	Not determined	Under investigation
Residronate (Actonel – Proctor&Gamble)	5 mg p.o. daily or 35 mg weekly	Effective
Pamidronate	Not determined	Under investigation
Ibandronate (Boniva – Roche)	150 mg p.o. monthly	Effective
Progesterone	Variable	Possibly effective
Thiazide diuretics	Variable	Probably ineffective
Tamoxifen (Nolvadex)	20–40 mg p.o. q.d. (10 mg b.i.d.)	Probably ineffective
Agents that promote bone formation		
Sodium fluoride	50–75 mg p.o. q.d.	Effective
Parathyroid hormone	40 µg SC q.d.	Probably effective
Growth factors	Variable	Probably ineffective
Anabolic steroids	Variable	Probably ineffective
Potassium bicarbonate	60–120 mmol p.o. q.d.	Possibly effective
Selective estrogen receptor modulators (SERM)		
Raloxifene (Evista)	60 mg p.o. q.d.	Effective
Tamoxifen (Nolvadex)	20–40 mg p.o. q.d. (10 mg b.i.d.)	Probably ineffective

HOT FLUSHES

Hot flushes are controlled without ERT using Megace® in dosage of 10–40 mg/day
Transdermal clonidine (Catapres®) is effective in < 50%
Bellergal-S is option but try Megace or Catapres first
Hot flushes can be caused by or associated with:
(1) Menopause
(2) Carcinoid tumors
(3) Systemic mastocytosis
(4) Medullary cancer of the thyroid
(5) Medications:
Tricyclic antidepressants
Monoamine oxidase inhibitors
Calcium channel blockers
Serotonin uptake inhibitors
(6) Idiopathic flushing
(7) Idiopathic anaphylaxis
(8) Pheochromocytoma
(9) Hyperthyroidism
(10) Acromegaly

Rule out hyperthyroidism prior to starting HRT or ERT for perimenopause. If normal TSH and symptoms continue – consider ruling out pheochromocytoma if patient's B/P is elevated. (*See* Pheochromocytoma)

• Avoid black cohosh and soy products in women with contraindications to estrogens

HUAM (HOME UTERINE ACTIVITY MONITORING)

Prevention of prematurity is controversial. Cervical dilatation alone as an appropriate endpoint for approval of this technology. Available data do not support the effectiveness of HUAM for prevention of preterm labor

HUMAN PAPILLOMAVIRUS

High-risk HPV types	16, 18, 45, 46
Low-risk HPV types	6, 11, 42, 43, 44
The average annual incidence of HPV infection in college women is	14%
The median duration of HPV infection is	8 months

HYDATIDIFORM MOLE

See Gestational trophoblastic disease

HYDROPS

Causes

(1) Immune response to hemolytic disease. What % of hydrops?	13%
(2) Non-immune response to:	
Intrinsic factors	64%
Cystic hygroma	41%
Heart anomalies	27%
Arrhythmia – multiple malformations	21%
Sacrococcygeal teratoma	4%
Twin–twin transfusion	4%
Placental anomaly	2%
Idiopathic factors	22%

Diagnosis	Ultrasound, maternal blood analysis (Hgb electrophoresis, K-B, IC, serologies for syphilis, toxoplasmosis, CMV, TUBS, parvo) or cordocentesis
Treatment	Depends on the cause (see above)
Complications	Increased maternal PIH, PTL (50%) due to hydramnios and postpartum hemorrhage due to uterine overdistention and/or retained placenta

HYDROSALPINGES

Watery sterile fluid in fallopian tube → end stage of pyosalpinx
PID – main cause of tubal infertility and ectopic pregnancy

Incidence of tubal infertility after	one PID	12%
	two PID	23%
	three PID	54%
Risk of ectopic after PID increases		6–7 x
With bilateral hydrosalpinx, IUP is slim at most only about		12%

Diagnosis	HSG (hysterosalpingogram) If suspect PID, get sed rate → if elevated → treat with doxycycline 200 mg then 100 mg b.i.d. for 5 days and postpone HSG until sed rate is normal. Water-soluble dye – risk of infection < 1% but 11% with dilated tubes will develop PID from HSG. If tubes are dilated, also give doxycyline as above after the HSG
	Conception rate within 1 year after using water-soluble agent 27% Use oil dye if there is no history of suspected PID as this causes less spasm and increases the conception rate after HSG
	Conception rate within 1 year after using oil-based agent 41% Delayed film – crucial to differentiate normal spill from dye that is just distributed through the pelvis Refer for *in vitro* fertilization if large hydrosalpinges are seen. Distal obstruction is more commonly seen
Treatment	Best to remove bilateral hydrosalpinx as it will reduce fluid and ectopic rate is @ 15%
	Ovaries are not to be disturbed and patient to be referred for IVF–ET
Prognosis	(After tubal reconstruction) – depends on the damage. If damage is extensive, the chance of conception after tubal reconstruction is almost nil → refer for IVF

HYGROMAS

Cystic hygromas are a malformation of the lymphatic system (occurs in late 6th gestational week)
First trimester – consider aneuploidies
Second and third trimester – monosomy XO is common
Check karyotype – If normal, the prognosis is good
Check for septations – if septations present, prognosis is decreased
With abnormal karyotype AND septations → this is worse prognosis

HYPEREMESIS

Nausea and vomiting to extent of weight loss, dehydration, ketosis and electrolyte imbalance

Incidence	@ what % of women with nausea and vomiting develop hyperemesis gravidarum?	1.3%
	Nausea + vomiting	50%
	Nausea only	25%
	Neither	25%

Definition of hyperemesis gravidarum	Persistent vomiting, weight loss > 5%, ketonuria, electrolyte abnormalities, dehydration (increased specific gravity), usually requires hospitalization

When does it occur?	Most of the time, the majority between 4–7 weeks' gestation Usually this % is over by 16 weeks' gestation 90%
Etiology	Vomiting center in medulla is thought to be affected – by unknown. Hormones? Vitamin deficiency? Psychological influences? GI dysmotility of pregnancy? *Helicobacter pylori* factor? (80% vs 50% *H. pylori* present in N+V of pregnancy patients)
Differential diagnosis	Gastroenteritis, hepatitis, cholelithiasis, pancreatitis, pyelonephritis, appendicitis, peptic ulcer disease, multiple pregnancies, hydatidiform mole
Labs	CBC, U/A, lytes, LFTs, amylase, TSH Test for ketones while NPO after every void, I&Os, weight Specific gravity (concentrated) 1.020–1.030
Ketones	Acetone, acetoacetate and β-OH butyrate
Management	(1) First try Increase protein and decrease carbohydrate and fatty foods in diet. Vitamin B_6 25 mg t.i.d. (50% stop vomiting). Severe nausea is reduced to mild to moderate nausea. Premesis® Rx is a prescription tablet containing vitamin B_6 75 mg so it can be given once per day. It also contains vitamin B_{12} (12 μg), folic acid (1 mg) and calcium carbonate (200 mg) (2) Second try B_6 and doxylamine (similar to Bendectin®) – vitamin B_6 50 mg tablet ½ tablet p.o. t.i.d. with doxylamine (Unisom®) 25 mg one tablet p.o. q. h and/or ½ tablet in a.m. and ½ tablet in p.m. (3) Third try And/or add CAM (complementary alternative medicine) Ginger (ginger capsules 250 mg t.i.d. to q.i.d.). Acupressure (wristbands available) Most popular acupoint for nausea and vomiting is Neiguan (P6) point located two cun (approximately three finger-breadths) below the distal wrist crease on the anterior surface (palmar side) of the wrist. Acupressure may be more successful than acupuncture for the indication of mild to moderate nausea and vomiting during early pregnancy. Increasing the frequency of treatments may reduce the frequency and severity of vomiting
If still uncomfortable	Add doxylamine succinate (Unisom®) p.o. 12.5–25 mg daily. Diagnose and treat any *Helicobacter pylori* infection
Intake and weight *IVFs*	Review at each visit D5NS 250 cc/h x 4 h then 150 cc/h Give KCl, MVT, folic acid and/or vitamin B_6 p.r.n. Total parental nutrition p.r.n. Refer to CNSD (Cert. Nutritional Support Dietician)
Diet	Day #1 – NPO, day #2 – clear, day #3 – low fat bland 3 x/day + three snacks
Drug therapy	• Ondansetron HCl (Zofran) 32 mg/50 ml premixed bag – best therapy, first choice – category B. Does not cause sedation. Patient can carry out routine activities. Disadvantage is the cost – it is expensive. Zofran also available in tablet and oral disintegrating tablets forms – 4 mg, 8 mg, 24 mg tablets; 4 mg, 8 mg ODT (strawberry flavor) • Anticholinergic (scopolamine) • Antihistamines (diphenhydramine/Benadryl) Category B • Serotonin (5-HT3) antagonist (Zofran and others) Category B • Benzamides (metoclopramide/Reglan) Category B • Promethazine (Phenergan) Category C • Phenothiazines (Compazine®) Category D • Butyrophenones (droperidol) – has Black Box Warning now (2001); has caused arrhythmias
Doses (most common therapies)	Phenergan 25 mg IV or suppository q. 6 h In doses of 50 mg → 50% patients sleep so titrate doses 12.5 mg →

25 mg → 50 mg
Zofran 32 mg IV @ 15 min then 0.15 mg/kg IV q. 4–8 h x 3 dose
Zofran 8 mg ODT or p.o. t.i.d. x 14 days → 0% sleepers
Reglan 10 mg IV or p.o. t.i.d. ½ hour prior to meals and hs
Benadryl 50 mg IV @ 30 min then q. 6 h

Surgical therapy Nasogastric, gastrostomy or jejunostomy feedings

HYPERLIPIDEMIA

Treatment

(1) *Increased cholesterol*
Cholestyramine, colestipol, niacin, atorvastatin, lovastatin, pravastatin, simvastatin
(2) *Increased triglycerides*
Gemfibrozil, niacin
(3) *Combined hyperlipids*
Niacin, atorvastatin, lovastatin, pravastatin, omega-3 fatty acid, vitamin E and vitamin C

HYPERPLASIA

Diagnosis Endometrial biopsy

Histology Nuclear enlargement, hyperchromasia, irregularity of nuclei, significant crowding but with some intervening stroma
Most important prognosticator of malignant potential ATYPIA

Treatment

If patient over 40 – HYSTERECTOMY is treatment of choice but if patient at increased risk – progestins x 3–6 months with repeat endometrial biopsies or D&C and/or hysteroscopy. Hysterectomy if indicated

If patient under 40 – progestin 10 mg daily x 10 days or Provera 20 mg daily day 16–25 or DMPA 200 mg IM q. 2 months x 3 doses or OCP or ovulation induction with FOLLOW-UP in 3 months
Endometrial evaluation 3–6 month follow-up is effective 62%
Discuss risks and informed consent @ hysterectomy p.r.n.

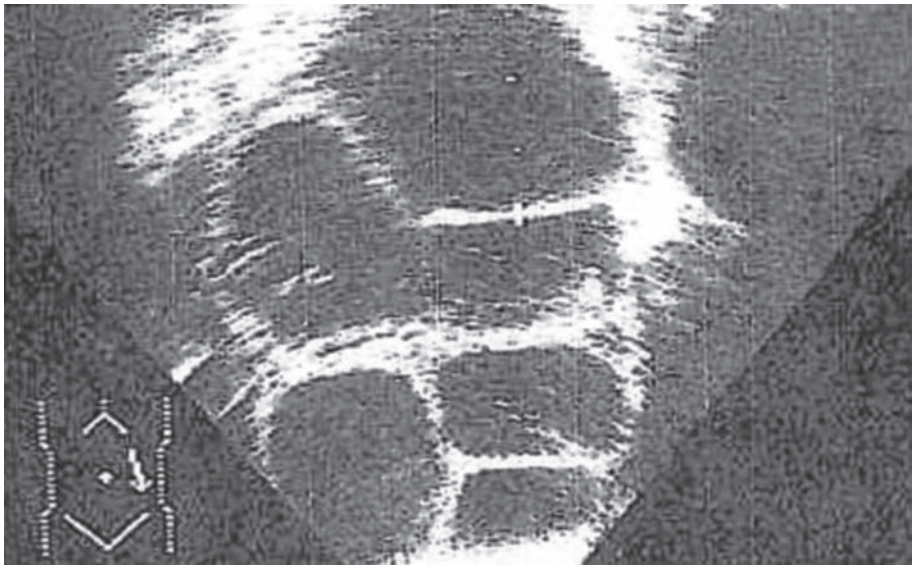


Figure 7 Ultrasound of hyperstimulation syndrome

HYPERSTIMULATION SYNDROME

NO PELVIC OR ABDOMINAL EXAMS – incidence 1%

Symptoms

Mild – abdominal distention/discomfort. Nausea, vomiting, diarrhea. Ovaries enlarge to 5–12 cm. Bloating, decreased appetite
Moderate – features of mild OHSS plus US evidence of ascites
Severe – features of moderate OHSS plus evidence of ascites and/or hydrothorax or difficulty in breathing. All these plus change in blood volume, hemoconcentration, coagulation abnormalities and diminished renal perfusion and function

Course of disease

Symptoms typically start 24–48 h and peak around 7 days following ovulation or follicular aspiration. Resolves 10–14 days

Risk factors

Young age (< 35 years), low body weight, hCG luteal supplementation, COH associated with GnRH agonist protocols, recently established pregnancy, high serum estradiol (E_2), rapidly increasing E_2 levels, multiple follicles, number of oocytes retrieved and findings consistent with polycystic ovaries (PCO) such as 'necklace' sign on US
Most widely accepted risk factors: young age, PCO and history of severe OHSS

Prevention

Recognition of high-risk profile of patient (most important).
Monitoring serum E_2 level and/or follicular response via ultrasound. Follicular puncture and aspiration. Use of intravenous albumin to enhance intravascular oncotic pressure. Using GnRH agonist instead of using hCG may help prevent OHSS. Use of progestogens for luteal phase support instead of hCG may help. Cryopreservation of all embryos from an IVF cycle precludes pregnancy and may shorten a patient's course of OHSS

Management

May or may not need to hospitalize. Urine output less than 1 liter/day or a 24-h fluid imbalance of more than 1 liter may necessitate hospitalization for closer observation

Hospitalize if:
(1) Symptoms of nausea, abdominal pain, vomiting or diarrhea cause intolerance of food or liquid
(2) Examination reveals hypotension, decreased breath sounds, tense abdomen or other signs of ascites, 'peritoneal' signs
(3) Abnormal blood tests:
  (a) Hematocrit > 48%
  (b) Sodium level < 135 mEq/l
  (c) Potassium level > 5.0 mEq/l
  (d) Creatinine level > 1.2 mg/dl
(4) Ultrasound findings – presence of fluid pockets between loops of bowel when patient is lying supine

Fluid management:
(1) Normal saline is fluid of choice
(2) Diuretics contraindicated for low urine output
(3) I&Os q. 2–4 h
(4) Consider albumin and/or dopamine with central lines p.r.n. severe

Thrombosis prevention (consider)
(1) Subq heparin
(2) Pneumatic compression hoses if patient confined to bed

Ascites management – paracentesis p.r.n.

HYPERTENSION IN PREGNANCY

Second leading cause of maternal mortality. (Second only to embolism)

Chronic hypertension – antiphospholipid syndrome increases risk of development of PIH
Patients with homozygous genes for angiotensinogen gene T23T have an increased risk of development of PIH compared to patients with chronic hypertension

Important points to remember in treatment of chronic hypertension:
(1) α–methyl DOPA (Aldomet) – first line
(2) Labetalol and Atenolol – second line of therapy and alternative
(3) β-blockers – monitor for IUGR
(4) ACE inhibitors – renal dysplasia, renal failures, oligohydramnios, fetal growth restriction, hypocalvaria and death can occur

PIH complicates what % of pregnancies in the USA?	6–8%
PIH directly caused what % of maternal deaths?	15%
PIH develops prior to what week gestation?	20th

HYPERTHYROIDISM

	Graves' disease makes up this % of hyperthyroidism cases	85%
Symptoms	Nervousness, palpitations, heat intolerance, goiter, weight loss or inability to gain weight. In pregnancy, most commonly → persistent tachycardia and lack of weight gain	
Diagnosis	Increased FT_4 or free thyroxine index and decreased TSH	
Treatment	Tapazole® p.o. b.i.d. in dose of	10–20 mg
	or PTU p.o. t.i.d. in dose of	100–150 mg
	add propranolol q. 6–8 h p.r.n. in dose of	10–40 mg
	When FT_4 index improves, decrease antithyroid drug to half dose then until tapazole is 15 mg or PTU is 50 mg daily. Goal is to keep FT_4 index in upper 1/3 normal until 30–34 weeks' gestation, then if euthyroid, then d/c until delivery. Double last total daily amount p.r.n.	
	• Thyroid stimulating antibodies can cross placenta and cause neonatal Graves'	

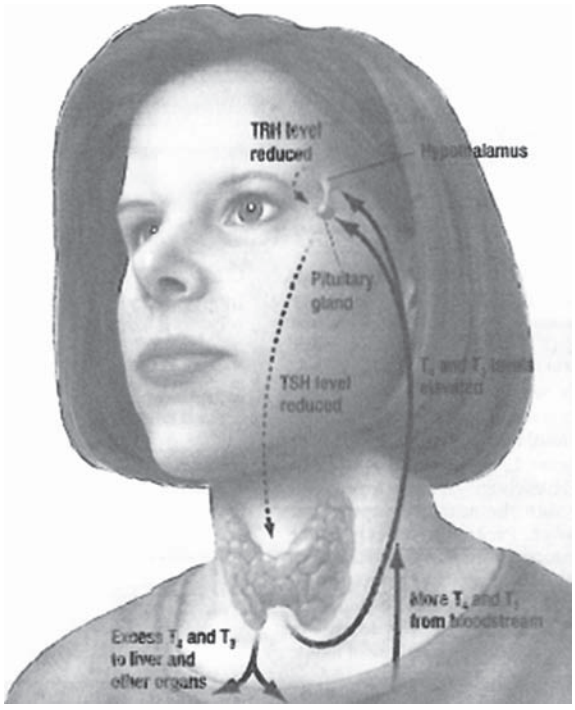


Figure 8 Cycle of thyroid hormones in blood stream

HYPOGASTRIC ARTERY LIGATION

Right angle or Mixter clamp is passed *lateral to medial* beneath – hugging its surface. This protects the hypogastric vein. Doubly ligate – DO NOT transect. Palpate femoral pulses and identify ureter before + after. Angiographic arterial embolization is an alternative using Gelfoam as small pledgets. MAST suits while waiting p.r.n.

HYPOTHYROIDISM

Symptoms

Tiredness, lethargy, constipation, cold intolerance, menorrhagia and infertility
More advanced symptoms – drowsiness, decrease in intellect and motor activity, hair loss, brittle nails, husky voice, weight gain, stiffness and tingling of the fingers, dry skin. In pregnancy, increase in spontaneous abortions and PIH

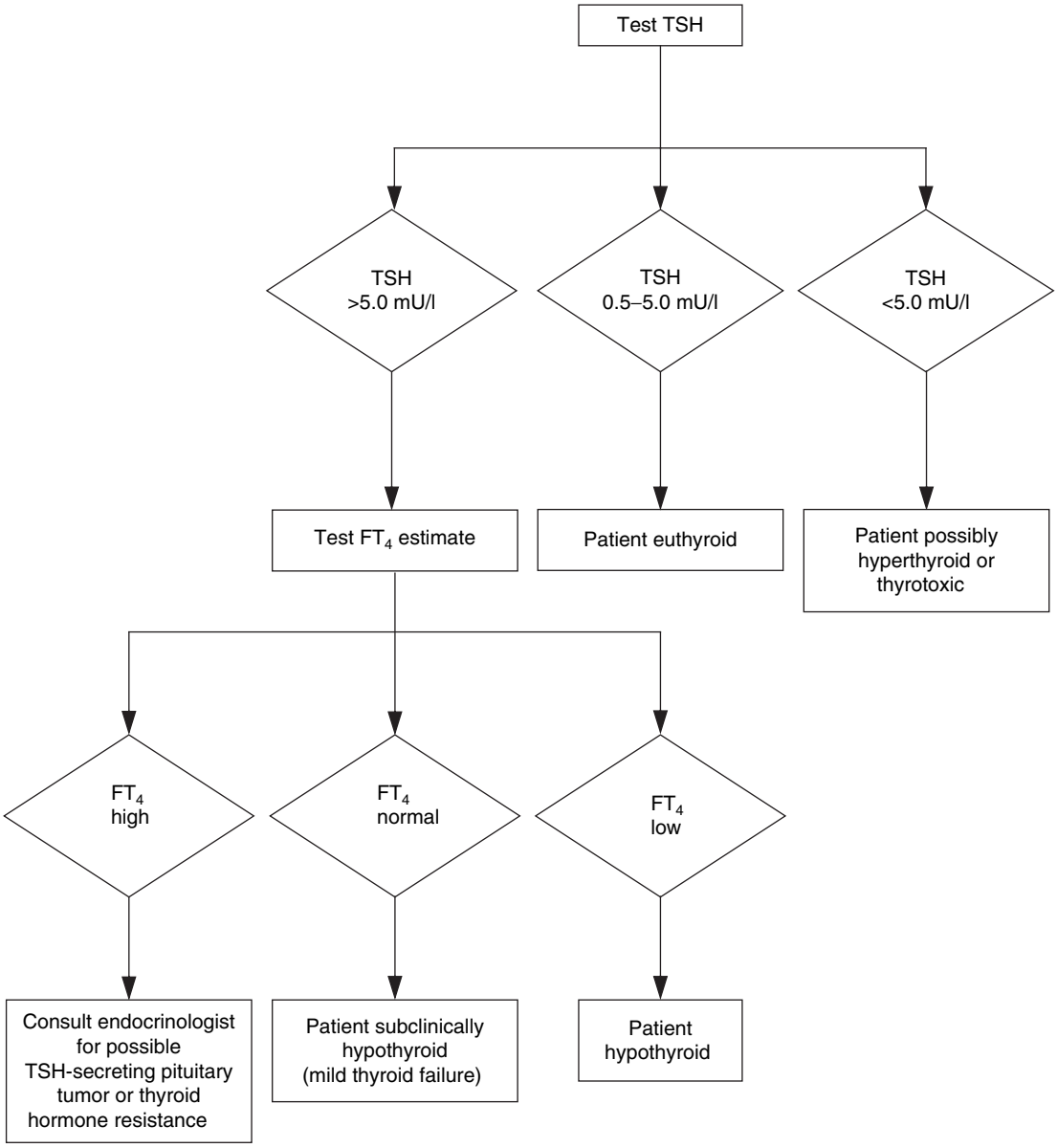
Diagnosis

Increased TSH, decreased serum thyroxine

Treatment

L-Thyroxine (1.6–2 mg/kg of ideal body weight) – (0.075–0.15 mg/day)
If patient is taking thyroid replacement at time of initial visit for pregnancy, check TSH. 50% of pregnant patients will need an increase in dosage
If TSH is elevated, increase L-thyroxine by 50 mg and repeat TSH in 4–6 weeks
If TSH is normal, repeat TSH at 22–28 weeks' gestation. After delivery, return to pre-pregnancy L-thyroxine dose. Start newly diagnosed hypothyroid patients with full replacement dose. Repeat TSH every 4 weeks and adjust the amount of L-thyroxine to keep the serum TSH within normal limits
In pregnancy, what % will need an increased dose? 50%

Diagnosis algorithm: primary hypothyroidism



HYPOTHALAMIC PEPTIDES

GnRH, CRF, GHRF, SS

HYPOTHERMIA

Can lead to

Cardiac dysrhythmias
Impaired anaerobic metabolism
A shift to LEFT in the oxygen–hemoglobin dissociation curve (decreased oxygen release)
Increased intracellular potassium release
Delayed drug metabolism

HYPOXIA AND ASPHYXIA

(1) The term asphyxia should be reserved for clinical context of damaging acidemia, hypoxia and metabolic acidosis
(2) Persistent Apgar score of 0–3 for longer than 5 min suggests hypoxic damage
(3) Hypotonia and GI dysfunction suggests hypoxic damage
(4) Profound metabolic or mixed acidemia (pH < 7.00) on an umbilical cord artery blood sample

HYSTERECTOMY

What % hysterectomies in the USA are abdominal?	70%
What % are performed to treat myomas?	30%
Incidence of postop wound infections	2%
Incidence of postop bleeding	2%

Expert gyn surgeons use the vaginal route for more than 90% hysterectomies

Types of hysterectomies from most invasive to less invasive:

RTAH	radical total abdominal hysterectomy
TAH	total abdominal hysterectomy
HALS	hand-assisted laparoscopic surgery (hysterectomy)
LAVH	laparoscopic-assisted vaginal hysterectomy
TLH	total laparoscopic hysterectomy
TSH	total supracervical hysterectomy
TVH	total vaginal hysterectomy
MIVH	minimally invasive vaginal hysterectomy

The pendulum for doing minimally invasive surgery is swinging back to pelvic and vaginal surgery. Initially vaginal surgery was performed because abdominal surgery could not be done. Infections and the development of antibiotics influenced surgical gynecologists to do more abdominal surgeries. Now to avoid filling the abdomen with CO_2 gas and going through seven layers of tissue with one or multiple incisions, the MIVH has been developed to decrease pain and speed recovery. Entering a thin layer of tissue anterior and posterior to the uterus, the MIVH was developed and can be seen in the following step-by-step photographs (Figures 9–16). The author has carried out thousands of MIVHs with operating room times from 5 to 20 min and minimal complications, despite obesity, fibroids, or previous surgeries. Uteri larger than 1400 g have been removed vaginally via morcellation techniques. No indwelling catheter is required for MIVH and there is never a need for a “pre-op laparoscopic peek”. Preliminary laparoscopy can frighten even a highly skilled surgeon away from what more than likely would be a much easier than imagined vaginal procedure, not to mention the increased risk of placing a trocar into the abdomen. **Do more TVHs or MIVHs!!!!!** OB/Gyn surgeons should be performing many more vaginal compared to abdominal or laparoscopic-assisted hysterectomies!!!!!

(1) If the bony pubic arch is adequate, the cervix mobile at its own level and the uterus is freely movable – the uterus should be able to be removed vaginally regardless of size or complex pathology
(2) The benefits of a vaginal hysterectomy include a quicker return to normal activity, less pain and lower costs compared to abdominal hysterectomy
(3) Remember to minimize the size of myomas either by giving GnRH agonists preoperatively or by performing bivalving, lash or coring type incisions to place traction and invert the uterus

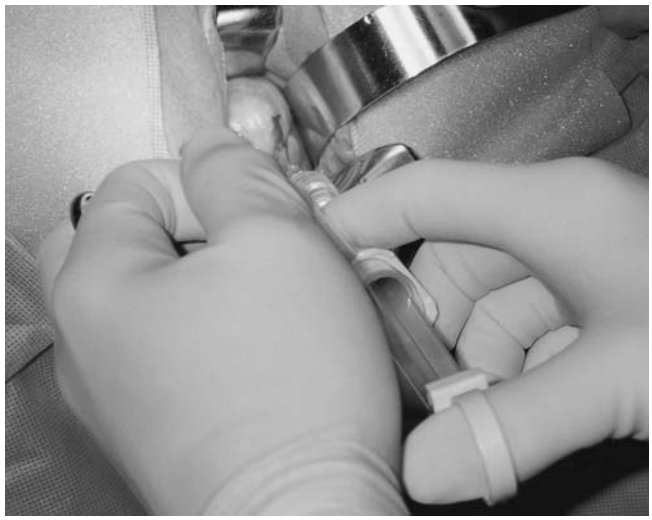


Figure 9 MIVH – injection of Marcaine with 50% epinephrine into the cervical mucosa decreases bleeding and helps develop a plane of dissection

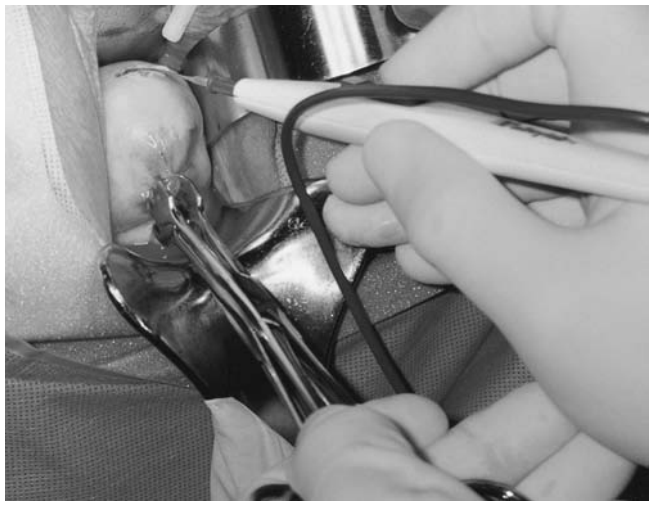


Figure 10 MIVH – cutting with a 90° angle *Bovie*, a small anterior and posterior incision into the cervical mucosa

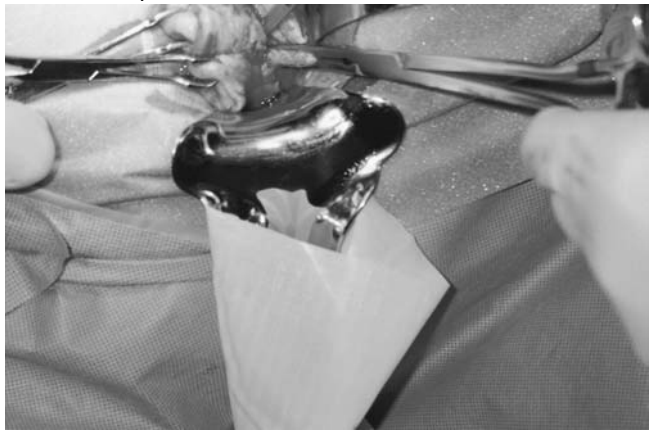
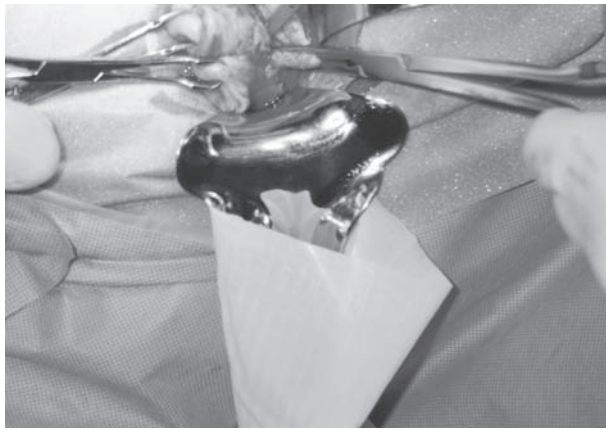


Figure 11 MIVH – after entering the anterior and posterior peritoneum, the regular weighted retractor is removed and replaced by the duckbill weighted retractor for excellent exposure

(a)



(b)

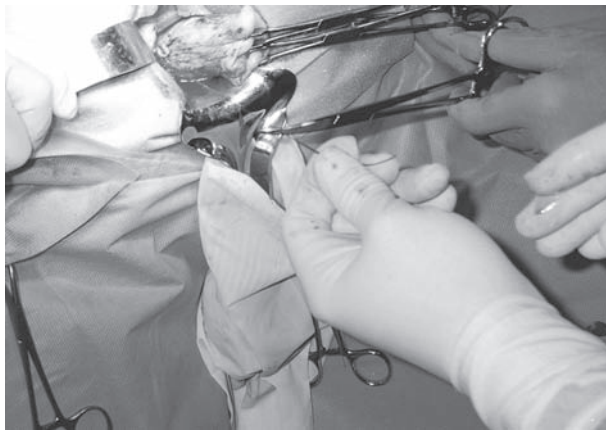


Figure 12 MIVH – the cardinal and portions of the uterosacral ligaments are bilaterally clamped and doubly ligated with 0-Vicryl suture then tagged for later plication

Figure 13 The remainder of the uterine vessels, broad ligaments, utero-ovarian ligaments, round ligaments, and infundibulopelvic ligaments (if adnexa are to be removed) are stapled with stapling device (Ethicon Endo-Surgery, Inc., ETS Compact-Flex45 Articulating Linear Cutter). The stapler remains the best option available due to necessary use of wet sponges required to protect the vagina if the Gyrus or other cauterizing clamps are used. However, the Harmonic Ace is still under investigation as an excellent alternative because of its minimal lateral spread statistics.

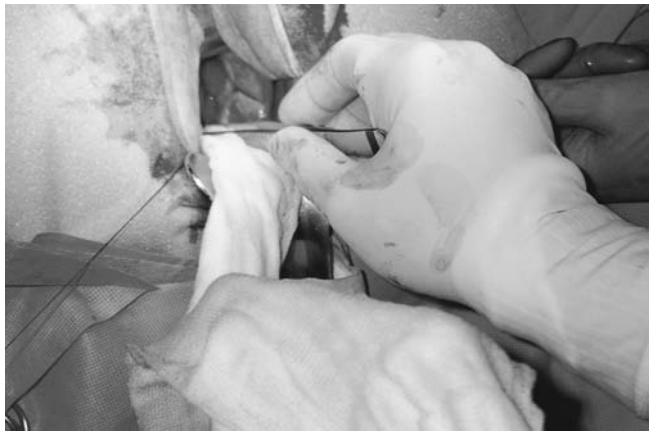


Figure 14 The viscera are displaced with the wet "JET" pack (McNeil – Catalog # 40120) for better exposure to perform appendectomy, visualize the peritoneum better, and/or check for any possible bleeding

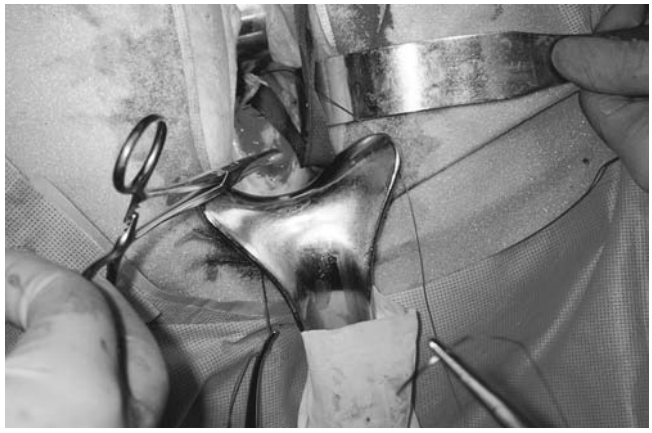


Figure 15 The "JET" pack is tagged with an Allis clamp so that it is never possible to lose the only packing ever used in this procedure

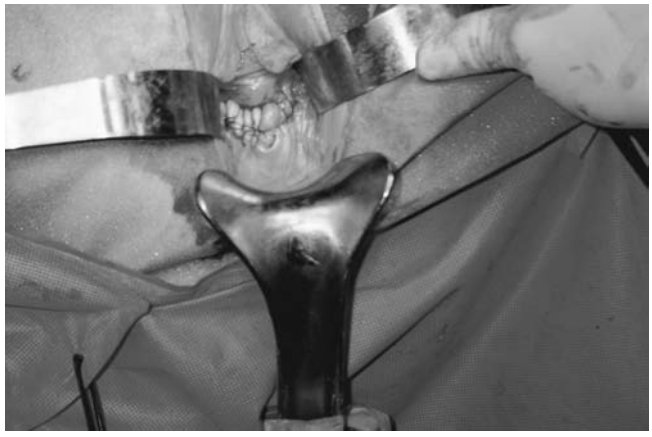


Figure 16 The peritoneum is reapproximated in a "purse string" fashion, ligaments are plicated, and vaginal mucosa is closed. No catheter or vaginal packing is left in place

Criteria for hysterectomy

Palpable myoma or concern to patient
Excessive anemia > 8 days of anemia
Discomfort
Rule out *C*ervical cancer, *A*nemia, *M*edical diseases and *E*ndometrial cancer prior to surgery

Emergency hysterectomies

Rare with exception of hemorrhage, torsion, or accreta. Occasionally prolapsed fibroids can be snared vaginally and removed, especially if there is a small-diameter pedicle

Method of estimating weight of uterus

The most accurate method of predicting uterine weight preoperatively is the bimanual assessment. However, one can also predict weight by doing an ultrasound and measuring:
width x AP x length = fundus
fundus x 0.52 = estimated weight of uterus in grams
(60–120 g is average size of uterus; it is estimated that the average Ob/Gyn can remove a 280 g uterus through the vagina)
Normal uterine weight 60–90 g
Myometrial hyperplasia > 120 g

Comparison of TVH to TAH

TVH	*TAH*
Decreased bowel manipulation	Adnexal pathology
Correct vaginal relaxation at same time	TOA
Decreased compromise to pulmonary system	Severe endometriosis
Avoid incisional complication	Need to explore
Decreased pain and adhesions	
Increased ambulation	

Supracervical hysterectomy

Subsequent trachelectomy is common occurrence (> 20%) following elective laparoscopic supracervical hysterectomy. (Okara EO, Jarnes KD, Sutton C. Long-term outcome following laparoscopic supracervical hysterectomy. *Br J Obstet Gynaecol* 2001;108:1017–20)

Vaginal hysterectomy after C-section

Rates of bladder injury were not statistically different: 1.86% vs 0.89%, according to cumulative data from 4 studies published between 1980 and 2003 (Agostini A, Vejux N, Colette E, *et al*. Risk of bladder injury during vaginal hysterectomy in women with a previous cesarean section. *J Reprod Med* 2005; 50: 940–2). Therefore a previously scarred bladder flap does not necessarily mandate an abdominal approach – only closer attention to how the bladder is advanced. The author actually believes that the abdominal approach increases the risk of bladder injury after C-section due to the technique used to develop the bladder flap from above during C-section. This is probably why such minimal complications to the bladder occur during MIVH

Laparoscopic vs vaginal hysterectomy

The rate of complications with laparoscopic/LAVH oophorectomy is approximately 2x higher than that of vaginal hysterectomy and prophylactic oophorectomy. (Agostini A, Vejux N, Bretelle F, *et al*. Value of laparoscopic assistance for vaginal hysterectomy with prophylactic bilateral oophorectomy. *Am J Obstet Gynecol* 2006; 194: 351–4.) ***Ureteral injury is less common with vaginal than with abdominal or laparoscopic routes!*** Most bladder injuries during vaginal surgery occur above the trigone and can be repaired by the gynecologic surgeon. (*see* Wound closure)

Always manage the vaginal cuff regardless of hysterectomy type

McCall culdoplasty at all three levels of support (proximal, lateral, and distal). Internal McCall sutures allows placement of 3 rows of sutures across the cul-de-sac from one uterosacral ligament to the other. External McCall sutures allow for 3 additional rows of absorbable sutures incorporated in the vaginal epithelium and uterosacral ligaments to move the vaginal cuff superiorly

Suspensory sutures can also be performed with high uterosacral attachment so that the uterosacrals need not be brought together

To avoid complications

It is essential that the uterine vessels be ligated before morcellation begins

Avoid blunt dissection in women with a history of pelvic surgery

Perform cystoscopy after all hysterectomies, especially those that became more difficult during surgery. Ensuring hemostasis and the integrity of the bladder and ureteral patency is of utmost importance

The Harmonic Ace is a scalpel that uses ultrasonic energy to cut, dissect, and coagulate tissue. Ethicon Endo-Surgery, Inc. developed this instrument. The author uses and recommends this during TLH because of the minimal amount of lateral spread and temperature differences – thus less damage to soft tissue. This has been compared to electrocautery, Kleppinger Bipolar, Gyrus ACMI, LigaSure, and CO_2 laser. The value of Lyons and PKS Plasma Trissector vessel sealing and thermal spread is unknown at the time of this publication

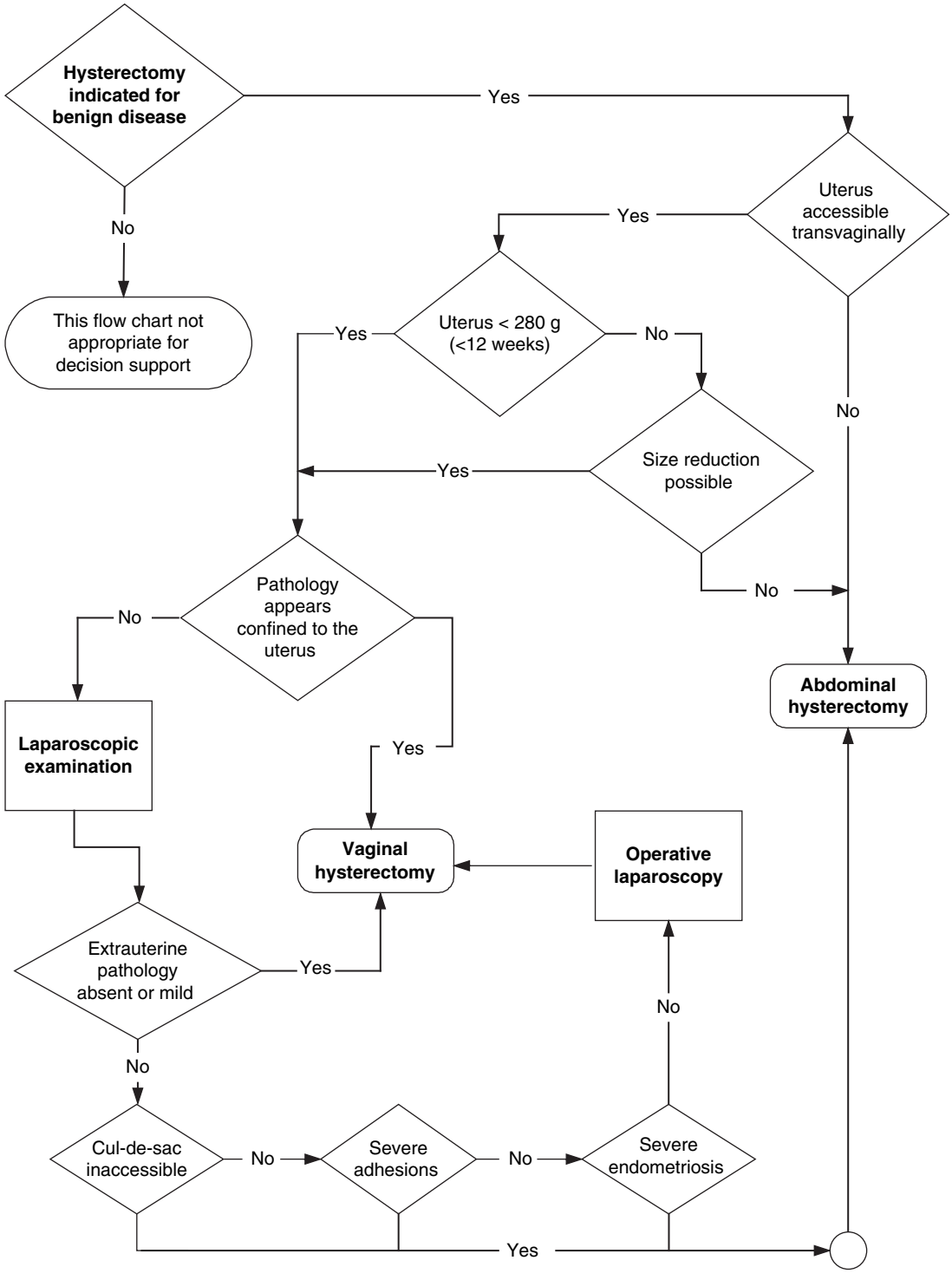
Spare ovaries?

Approximately ½ of all women over 40 will die of heart disease, while fewer than 1% will die of ovarian cancer

Hysterectomy itself appears to reduce the risk of ovarian cancer by 5% to 15%

Oophorectomy does not provide a survival benefit over ovarian conservation. It would certainly be acceptable, however, to remove ovaries of a woman over 45–46 years old who is lean, normotensive, with a favorable lipid profile, but fears the possibility of ovarian cancer. Whether this is right or wrong, as long as the patient is informed that this may not even rule out ever having the cancer, it is acceptable for the patient's peace of mind

Determining the route of hysterectomy



HYSTEROSALPINGOGRAM

Consider PID prior to HSG
If suspect PID, get sed rate – if elevated, give doxycycline 200 mg, then 100 mg b.i.d. x 5 days and postpone till sed rate normal

When to do HSG

Perform early in proliferative phase of cycle AFTER cessation of menstrual flow but preferably prior to ovulation — 6–10th day

Risk of infection — < 1%
Risk of infection with dilated tubes — 11%
If tubes are noted to be dilated, give doxycycline 200 mg then 100 mg b.i.d. after HSG. If no history of suspected PID, use oil dye as it decreases spasm and increases conception up to — 41%

Risk of dye embolization and/or salpingitis — 1–2%
Conception rate within 1 year after H_2O solution agent is — 27%
Laparoscopy with chromotubation is indicated if HSG is contraindicated or results are abnormal

HYSTEROSCOPY

CO_2 is agent of choice for *diagnostic.* NOT operative and limited to
how many ml per min? — 40–60
At pressure of ? mmHg — 100
When distending the uterus with CO_2 gas, care should be taken not to exceed intrauterine pressure of — 150 mmHg

CO_2 gas is not practical for operative procedures because it does not allow for clearing debris and should never be used for operative hysteroscopic procedures because of the high risk of CO_2 embolism. Air embolism can be detected by a machine-like murmur over the precordium that can often be auscultated

With suspicion of an air embolism, these actions should be taken:
(1) Hysteroscope should be immediately removed
(2) Vagina should be occluded with a wet sponge
(3) The patient should be turned to the left side
(4) The patient should be transferred to an intensive care unit

Normal saline and lactated Ringer's are useful for diagnostic but NOT operative
Nd : YAG – poor cutter but excellent COAGULATOR
Argon + KTP used for excellent CUTTING action
Dextran can cause DIC and PULMONARY EDEMA so do not use more than — 500 ml

1.5% Glycine can cause hyponatremia and hyperammonemia. Decreased visual acuity, cerebral edema and intracranial volume expansion with herniation
Half-life — 85 min

3% Sorbitol can cause hyponatremia and cerebral edema. Mannitol 0.54% added to decrease risk of fluid overload. Half-life is — 35 min
Sorbitol is metabolized to fructose and glucose

Hyskon® (32% dextran 70) is RAPIDLY ABSORBED. It is a volume expander of — 10 : 1
200 ml absorbed displaces how much blood volume? — 2 liters
ACUTE PULMONARY EDEMA
"CARAMELIZES INSTRUMENTS"
Dextran molecules can produce DIC
+ are too big to diuresis therefore needs PLASMAPHORESIS
(also interferes with immune blood tests)
Molecular weight of Hyskon is — 70 000 daltons
Anaphylaxis occurs in — 1/10 000
Infusion should not exceed — 300 ml
Patient absorption of Hyskon should not exceed — 250 ml

D5 in H_2O can cause profound hyponatremia @ lethargy, confusion, pulmonary edema. Na + Cl (0.9%) or RL not associated with electrolyte imbalance or metabolic disturbance. Fluid overload can be quickly reversed. Rollerball endometrial ablation is an effective treatment for menorrhagia; younger women < 35 who undergo ablation have an increased risk of subsequent hysterectomy compared to women > 45 (Dutton C, Ackerson L, Phelps-Sandall B. Outcomes after rollerball endometrial ablation for menorrhagia. *Obstet Gynecol* 2001;98:35–9)

Key points

Preoperative treatment with a GnRH agonist increases the odds of operative complications by a factor of 4–7

Ultrasound guidance may improve outcomes in selected hysteroscopic procedures.

Preop vaginal misoprostol (Cytotec), a synthetic prostaglandin E_1 analog, prior to hysteroscopy, can significantly reduce the necessity for cervical dilation and minimizes cervical complications and operative time

To see all the different hysteroscopic procedures and how these are done (including ablative surgeries) → refer to Turrentine JE. *Surgical Transcriptions and Pearls in Obstetrics and Gynecology*, 2nd edn. London: Informa Healthcare, 2006

ILEUS

Functional symptoms

Steady, mild abdominal pain. Silent abdomen with absent bowel sounds, abdominal distention, tympany and absence of flatus. N&V < 24 h

Flat plate

Supine, erect and lateral X-rays show gas in small and large bowel

Diagnosis

PO Gastrograffin (stimulates peristalsis, passage of stools in hours and not toxic like barium if spills during possible surgery)

Treatment

GI rest and time, IVFs, lytes, WBCs, Mylicon® decreases surface tension. NG tube only p.r.n. USUALLY SELF-LIMITING

Risk of ileus increases if

(1) Contamination by pus or blood
(2) Extensive handling of tissue
(3) Obesity
(4) Ice cubes, gum chewing or carbonated beverages
(5) Preop immobility
(6) Prolonged use of narcotics
(7) Retroperitoneal surgery
(8) Removal of large peritoneal adhesions during surgery

IMMUNIZATIONS

General principles

There are four types of immunologic therapy:

(1) Inactivated vaccines: hepatitis B, influenza and pneumococcus
(2) Live-attenuated vaccines: measles, mumps, rubella and polio
(3) Toxoids: tetanus, diphtheria
(4) Immunoglobulins: hepatitis B, rabies, tetanus, varicella, hepatitis A and measles

Childhood immunizations cause most women to be immune to measles, mumps, rubella, tetanus, diphtheria and polio by child-bearing age

Vaccinate according to age group and risk factors

Age 13–18
Tetanus–diphtheria booster (age 14–16 x 1)
At-risk groups:
(1) Child-bearing age and no evidence of immunity – MMR
(2) Blood products, household/sexual contacts of hepatitis B carriers, multiple sexual partners in past 6 months – hepatitis B vaccine

Age 19–65
Tetanus–diphtheria booster (every 10 years)
Influenza vaccine (every year starting at age 55)
At-risk groups:
(1) Child-bearing age and no evidence of immunity – MMR
(2) IV drug users, blood product recipients, health-care workers, household/sexual contacts of Hep B carriers, multiple sexual partners in past 6 months – Hep B vaccine
(3) Chronic cardiopulmonary disease, metabolic diseases, diabetes, hemoglobinopathies, immunosuppression, renal dysfunction – influenza vaccine annually
(4) Conditions prone to pneumococcal infection (i.e. immunosuppression), chronic cardiopulmonary disease, sickle cell disease, renal disease, status postsplenectomy, diabetes, alcoholism, cirrhosis – Pneumovax

Age 65 and >
Tetanus–diphtheria booster (every 10 years)
Influenza vaccine annually
Pneumovax (once)
At-risk groups:
Exposure to blood products, household/sexual contacts with chronic Hep B carriers – Hep B vaccine

Immunizations in pregnancy

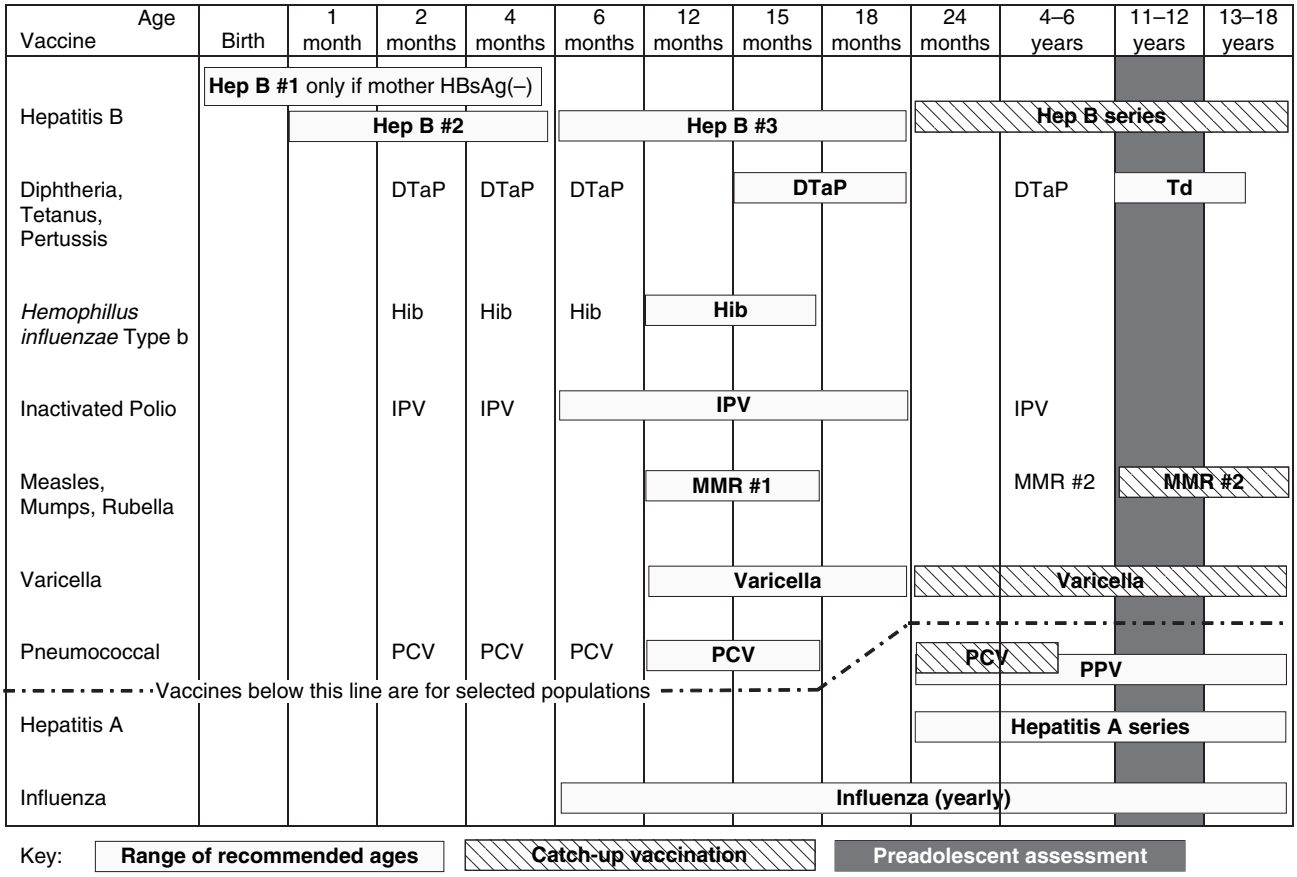
Theoretical concern of congenital infection by live vaccines during pregnancy although there have been no reported cases
MUST weigh several factors: risk of exposure, maternal risk, fetal risk and risk from vaccine/toxoid
Rule of thumb → no live vaccines unless:
(1) Susceptibility/exposure probable
(2) Disease threat to woman/fetus – vaccine risk
Only routinely administered immunizations during pregnancy:
(1) Tetanus–diphtheria toxoids
(2) At-risk group for Hep B virus (see above)
MMR → Give 3 months before pregnancy or stat postpartum
Polio/yellow fever vaccine → when traveling to endemic area
Immune globulins:
(1) After exposure to measles, Hep A, B, tetanus, chickenpox or rabies
(2) VZIG for newborns of mother who develop chickenpox 5 days before, until 2 days after delivery
(3) All women without a history of chickenpox should be passively immunized with VZIG within 96 h of an exposure to chickenpox

• *See also* Vaccines

Specific indications for vaccines and immune globulins during pregnancy

Immunizing agent	*Indications*
Vaccines	
Live virus	
Poliomyelitis (Sabin)	Immediate protection against poliomyelitis for previously unimmunized individuals
Yellow fever	Travel to endemic areas
Measles	Contraindicated
Mumps	Contraindicated
Rubella	Contraindicated
Live bacteria	
Tularemia	Rabbit handlers, laboratory workers
Bacille Calmette–Guérin	Not recommended
Killed virus	
Hepatitis B	Pre- and postexposure prophylaxis for individuals at high risk
Influenza	Chronic cardiopulmonary or renal disease; diabetes mellitus
Poliomyelitis (Salk)	Travel to epidemic areas; laboratory workers
Rabies	Exposure to potentially rabid animals
Killed bacteria	
Cholera	Entry requirement for some countries
Meningococcus	Epidemic meningococcal-non-B disease
Plague	Laboratory workers; travel to areas with human disease
Pneumococcus	Cardiopulmonary disease, splenectomy, alcoholism, Hodgkin's
Typhoid	Household contact with chronic carrier; travel to endemic areas
Pertussis	Not recommended
Toxoids	
Anthrax	Laboratory workers; handlers of furs and animal hides
Tetanus–diphtheria	Primary immunization; booster
Immune globulins	
Pooled human	
Hepatitis A	Pre- and postexposure prophylaxis
Measles	Postexposure prophylaxis
Hyperimmune	
Hepatitis B	Postexposure prophylaxis
Rabies	Postexposure prophylaxis
Tetanus	Postexposure prophylaxis
Varicella zoster	Postexposure prophylaxis
Horse serum	
Botulism	Treatment of infection
Diphtheria	Treatment of infection

Recommended childhood immunization schedule USA, 2002



This schedule indicates the recommended ages for routine administration of currently licensed childhood vaccines, as of December 1, 2001, for children through age 18 years. Any dose not given at the recommended age should be given at any subsequent visit when indicated and feasible. ▨ Indicates age groups that warrant special effort to administer those vaccines not previously given. Additional vaccines may be licensed and recommended during the year. Licensed combination vaccines may be used whenever any components of the combination are indicated and the vaccine's other components are not contraindicated. Providers should consult the manufacturers' package inserts for detailed recommendations. Approved by the Advisory Committee on Immunization Practices. Atlanta, GA: Centers for Disease Control and Prevention, US Department of Health and Human Services, 2002

IMPERFORATE HYMEN

	Cruciate incision should be made (like an X) from 10 to 4 and 2 to 8 o'clock This occurs at the junction of the sinovaginal bulbs with the urogenital sinus
Diagnosis	History
	Physical – blue bulging membrane at introitus Ultrasound – confirms distended vagina
Management	Excise CENTRALLY using an X incision. Avoid needling because this increases risk of infection. Avoid probing or curetting uterus as this increases risk of perforation
Differentiate	From transverse vaginal septum. Hematometria but bulging membrane not visible → identify separate hymenal ring. (Failed fusion and canalization of urogenital sinus and Müllerian duct derivative)

INCISIONS

Midline	*Advantages*	*Disadvantages*
	More rapid entry Better exposure	Weaker Less cosmetic

Transverse	Greater strength More cosmetic	Less rapid entry Reduced exposure
Risk factors for disruption	Infection, obesity, diabetes, emphysema or chronic bronchitis, ileus, malignancy, ascites, irradiation, chemotherapy, corticosteroid therapy	

INCONTINENCE

'DIAPERS'	Drugs, Infection, Atrophic vaginitis, Psychological factors, Endocrine, Restricted mobility, Stool impaction. (Mnemonic for urinary and fecal incontinence)
Urinary incontinence	*See* Urinary incontinence

INCREASES IN GRAVID

In pregnancy, renal clearance is increased by	40%
This means aminoglycosides get out quicker. However, there is decreased renal clearance with theophylline. Keep at a low level of @	8–12 mg/dl
GFR increases by	50%
Serum creatinine values decrease from 0.7 mg/dl to	0.5 mg/dl
BUN values decrease from 12 mg/dl to	8 mg/dl
RENAL INSUFFICIENCY if serum creatinine is	0.9 mg/dl
BUN is	14 mg/dl

INDUCTION

Most patients attain normal progression of labor with how many Montevideo units of uterine activity?	150–350 MV units
Low-dose Pitocin starting dose	0.5–2 mU
Increase dose by 1–2 mU/min in an interval of	15, 30 or 40 min
High-dose Pitocin starting dose	6 mU
Increase dose by 1, 3, or 6 mU/min in an interval of	15, 20–40 min

Reduce the risks of pitocin

(1) Start with a written note (include indication and patient consent)
(2) Conduct a comprehensive consent process (give patient options)
(3) Describe both uterine and fetal responses (reassuring?)
(4) Discontinue oxytocin when the uterus overreacts
(5) Adjust oxytocin to reflect changes in labor patterns
(6) Consider including a labor curve

Methods of cervical ripening

(1) *Pharmaceutical methods*
Intracervical PGE_2
Intravaginal PGE_2 gel
Intravaginal PGE_2 controlled-release insert
Intravaginal misoprostol
(2) *Mechanical methods*
Osmotic dilators (not associated with hyperstimulation)
No uterine or fetal monitoring required
Balloon catheter ripening
Intracervical laminaria
(3) *Membrane stripping (also known as sweeping)*
Decreases the incidence of postdates pregnancy when done weekly beginning at 38 weeks' gestation. Membrane sweeping at the initiation of labor induction also increased the spontaneous vaginal delivery rate, reduced oxytocic drug use, shortened induction to delivery interval, and improved patient satisfaction. (Tan PC, Jacob R, Omar SZ. Membrane sweeping at initiation of formal labor induction: a randomized controlled trial. *Obstet Gynecol* 2006;107:569–77)

Dose of terbutaline used to treat uterine hyperstimulation caused by cervical ripening agents is 0.25 mg SC

How does Pitocin work? It causes contractions by causing the release of calcium from the sarcoplasmic reticulum

How does terbutaline work? The mechanism of action is to increase intracellular cyclic AMP (adenosine monophosphate)

INFECTION

Infection	*Treatment*	
UTI	TMP/SMX or nitrofurantoin x 3 days	
PTL	None recommended Assess for need for GBS Rx	
PPROM	Ampicillin and macrolide x 7 days	
Chorioamnionitis	Ampicillin and gentamicin	Clindamycin for pen allergy
PP endometritis	Clindamycin and gent EID *or* ampicillin/sulbactam	
C/S (prophylaxis)	First-gen. cephalosporin	Clindamycin for pen allergy
Endocarditis prophylaxis	Ampicillin and gentamicin	30 min prior to delivery and 6 h postdelivery

BV found in what % of patients? 10–25%
What % of BV infections are asymptomatic? 50%

INFERTILITY

Definition

Inability to conceive after how many months for a couple that has never conceived? 12

Inability to conceive after how many months for a couple with a prior conception? 6

Bullet work-up

(1) Very thorough history and physical
(2) Initial diagnostic tests
  (a) Semen analysis
  (b) Basal body cell temperature charting x 3 months
  (c) Consider HSG and postcoital test

More specific work-up

(1) Male factor

  Urological exam, serum testosterone, FSH, testicular biopsy, serum fructose, diabetes screen, sperm penetration assay

  Treatment

  Hypogonadism → hMG or GnRH
  Varicocele → surgical ligation
  Decreased penetration/oligospermia – IVF, ICSI

(2) Female pelvic factor
  (a) Tubal disorders – HSG, laparoscopy
    Treatment → tuboplasty/lysis of adhesions (scope), IVF
  (b) Uterine disorders – HSG, ultrasound
    Treatment → hysteroscopic lysis of adhesions/metroplasty/septoplasty (followed by antibiotic and high-dose estrogen), myomectomy
  (c) Endometriosis – laparoscopy
    Treatment → laparoscopic fulguration (moderate/severe) and/or GnRH agonist, OCs, danazol, Synarel®, etc.
(3) Ovulatory factor
  FSH/lH, prolactin, thyroid profile, progesterone challenge, endometrial biopsy (luteal phase), serum progesterone, MRI
  Treatment → hyperprolactinemia – bromocriptine (Parlodel®)
  thyroid disorder – treat appropriately

Positive progesterone challenge – Clomid with or without dexamethasone 0.5 mg and/or hCG. If Clomid fails, consider Pergonal or referral. Luteal phase defect – progesterone suppositories 25 mg intravaginally b.i.d. or 250 mg IM weekly

(4) Cervical factor
Check for spinnbarkeit, ferning and os patency midcycle
Treatment → absent/poor quality mucus → low-dose estrogen or IUI (intrauterine insemination)
Culture for ureaplasma/*Chlamydia*
If + culture → give antibiotics (Zithromax 1 g once or doxycycline 250–500 mg b.i.d. x 10 days

Fecundity

After 3 months	57%
After 6 months	72%
After 12 months	85%
After 2 years	93%

Never tell a couple that they will not be able to conceive

What % of azoospermic males can conceive? (Probably unable to see all of sperm microscopically)	0.3%
What is the pregnancy rate of females with primary ovarian failure?	4–5%

Primary ovarian failure

Day 3 FSH – normal result is	5–7 mIU/ml
If patient in her 20s with this result, ovaries @ to fail	> 8
If patient in her 30s with this result, she has had it	> 8
Very rare to be able to conceive with this result FSH	> 15
This % 20-year-olds are found to fail to generate embryos in IVF during the recruiting for IVF program	5%

Endometriosis

Stage I and II – treated or untreated demonstrates no difference
Stage III and IV – surgical treatment is efficacious
IVF best treatment for tubal disease

What proportion of couples experience infertility?	15%

Male factor

There is an upper limit to sperm count	250 000 000 million/ml
What percent of infertile men consume very little fruits and vegetables?	83%

There were only 40% of the fertile men in the low fruit and vegetable study. Antioxidants in broccoli, oranges, tomatoes, peppers, and leafy greens seem to be the ingredients that energize sperm

What is the average amount of semen expelled with each ejaculation?	1 tsp
Sexual dysfunction as the cause of male infertility is	< 5%
Endocrine problems as the cause of male infertility is	< 3%
Retrograde ejaculation as the cause of male infertility is	< 0.5 ml

Treatment for abnormal semen analysis:

Clomid and IUI successful	5%

IVF with embryo transfer cheaper than injectables with IUI. Treatment of azoospermia – TASA (testicular aspiration). Rule out deletion of part of Y chromosome – donor sperm?

Causes of infertility	*Work-up and labs to determine cause*	*Incidence*
Male factor	Semen analysis (2–6 cc, liquefies, ≥ 20 mil, 50% mot, 60% mor)	35%
Female pelvic factor	HSG	25%
Ovulatory factor	LH/FSH, BBT, Bx, TSH, prolactin	20%
Cervical factor	Postcoital test	10%

Prior to semen analysis, there must be a period of abstinence of	48 h
The semen sample must be carried in for analysis within	2 h
If semen analysis is abnormal, two additional samples must be obtained how many weeks apart?	2

Obtain FSH if semen sample demonstrates oligospermia. If testes small + serum FSH > twice normal then suspect primary testicular failure. In hypogonadotropic hypogonadism, the FSH, LH + test all LOW

Proxeed is a nutritional supplement that has been shown to sometimes improve the quality of sperm after 2–3 months of use. Dosage is one packet in @ 4 ounces of fluid in the morning and evening

Ovulation is assessed with BBT, progesterone and endometrial biopsy
How many days after estimated ovulation should the serum progesterone be measured? 7 days
Low progesterone level may be consistent with normal ovulation

Side-effects of infertility meds

(1) Gonadotropins – hyperstimulation syndrome, local injection-associated effects
(2) Bromocriptine – GI irritation, orthostatic hypotension, headache, nasal congestion
(3) GnRH – local injection-related effects
(4) Clomiphene citrate – hot flashes, visual symptoms, nausea

Effects of drugs or toxins on male infertility

(1) Sulfa drugs – impaired spermatogenesis
(2) Narcotics – decreased libido
(3) Phenytoin – ejaculatory dysfunction
(4) Diethylstilbestrol – testicular atrophy
(5) Radiotherapy – germ cell depletion

Intrauterine insemination/timing of ovulation induction

Prerequisites

(1) Abnormal semen analysis

Normal semen: sperm count > 20 million/ml
motility at least 50%
morphology at least 30%
leukocytes <1 million/ml

(2) Male should also have history/physical, endocrine work-up p.r.n., antisperm ab testing p.r.n., sperm function testing or radiologic evaluation p.r.n.
(3) Female should have HSG, GC/*Chlamydia* cultures, postcoital test, and late luteal phase endometrial biopsy, laparoscopy and hysteroscopy may be indicated in some
(4) HIV & hepatitis tests should be considered initially for both partners

Timing

Prior to ovulation or at the time of (*not* after ovulation)
LH kits, ultrasound, cervical mucus for Spinnbarkeit/ferning
Ovulation usually occurs 24–36 h after the LH surge
hCG 10 000 units – ovulation occurs 34–36 h after injection

Seminal wash

Mix with physiologic buffer (Hamm's) – centrifuge on low – pellet – resuspend by diluent. Use 0.5 ml for IUI (not too much volume *in* uterus). Best for patients with hypospadias, retrograde ejaculation or pure oligospermia

(1) *Clomid:* 50 mg day 5–9 or day 3–7 (recruitment of more follicles)
Confirm ovulation with LH kit, BBT, US endo Bx or mid-luteal phase serum progesterone
Increase by 50 mg/d p.r.n. subsequent cycle p.r.n.
(2) *hCG:* to be given 7 days after the last dose of CC or when ultrasound reveals a follicle of 22–24 mm diameter. No more than six ovulatory cycles recommended
(3) *Human menopausal gonadotropins*
Prior to giving hMG, US to rule out ovarian cysts; serum estradiol (E_2) on 3rd day of cycle
If no cysts >10 mm and E_2 concentration < 50 pg/ml, start hMG 150 IU/d on day 3
Check E_2 levels on day 4, 6, and 8
Levels should be 100–200 (day 4), 400–600 (day 6), and 800–1200 pg/ml (day 8). Dosage adjusted accordingly
hCG 10 000 U IM when a single follicle or multiple follicles have reached 17 mm or > in diameter
hCG 5000 U IM or none given if concern about hyperstimulation

IUI performed at 34–36 h after hCG injection. Progesterone 250 mg IM or 25 mg supp b.i.d. started on day of IUI. Serum β-hCG level is obtained 16–18 days after IUI. IUIs timed at 18 and 42 h after hCG are superior to a single insemination

IUI

Insemi-Cath (Cook OB/GYN) on TB syringe or Mini Space IUI-Cath (1-800-441-1973)
0.2 ml of air then 0.5 ml (to fundus)
No lubricants
Clean cervix with normal saline
Delay injection until no cramping
Pt to lie for 15 min after IUI (? Effectiveness)

Complications

Spontaneous abortion – 26% vs 10–15%
Ectopic – increased to 8% vs 1%
Multiple pregnancies (with hMG 25–30%)
(with CC 5–10%)
Ovarian hyperstimulation – 1% (especially if $E_2 > 2000$)
IUI with hMG significantly increases pregnancy rates especially with men with impaired semen parameters
Refer to IVF or IVF with intracytoplasmic sperm injection if sperm counts < 5 million/ml or linear progressive motility < 20%

INFLUENZA

Symptoms

Fever, myalgias, headache, malaise

Diagnosis

Directigen™ FLU A and QuickVue® influenza tests are rapid enzyme immunoassay test kits

Reverse transcriptase polymerase chain reaction methods available

Treatment

Zanamivir (Relenza®) Category B is given 12 h apart for 5 days
in dose of Two puffs b.i.d.
Oseltamivir (Tamiflu®) Category C is given with 75 mg tablets p.o. for 5 days
in dose of One tablet b.i.d.

Vaccines

Amantadine and rimantadine. On January 14, 2006, the CDC recommended that clinicians *NOT* prescribe these to treat or prevent influenza during the 2005–2006 flu season. 91% of the strains of virus in the U.S. are resistant to these drugs. These also CANNOT BE GIVEN if patient allergic to eggs. Antibodies develop in 14 days when using flu vaccine. Side-effects to amantadine are dizziness, nausea and insomnia. Pregnant women are more susceptible in third trimester

INTERSEXUALITY

Male pseudohermaphrodite

Gonads are testes

Female pseudohermaphrodite

Gonads are ovaries

True hermaphrodite

Individual with both testicular and ovarian tissue

TDF

Testis-determining factor is a product of a gene located on the short arm of the Y chromosome termed *SRY*

SRY

Sex-determining region of the Y

TDF on the *SRY* gene is responsible for testicular development

Transvestism/transsexualism

These two terms are used only when there is a discrepancy between the two criteria of sex of rearing and the gender role. The sex of rearing is the assigned sex while the gender role is the adopted sex of an individual

Sex identification

(1) Genetic sex
(2) Gonadal sex
(3) Morphology of the external genitalia
(4) Morphology of the genital ducts
(5) Hormonal status
(6) Sex of rearing
(7) Gender role

Male pseudohermaphrodite

Lack of Müllerian duct regression, mixed gonadal dysgenesis, deficient testosterone production, deficient dihydrotestosterone production, exposure to anti-androgenic substances, complete androgen insensitivity (testicular feminization), incomplete androgen insensitivity

Female pseudohermaphrodite

Congenital adrenal hyperplasia, 21-hydroxylase deficiency, increased DHEA and 17-OH prog, 11β-hydroxylase deficiency, exogenous androgen intake, endogenous androgen effect

True hermaphroditism

Testicular tissue should be removed before puberty and the external genitalia reconstructed along female lines. The majority of true hermaphrodites have a chromosome constitution of 46XX

The rest include 46XY, XY/XYY mosaics or XX/XY chimeras

Distinguishing tests

See page 22

INTERSTITIAL CYSTITIS

Symptoms

Urgency/frequency and/or pelvic pain. Consider flares that correlate with specific events in a patient's life, especially associated with sexual intercourse (adult women during or after coitus, younger 17–18 year old women – symptoms occur a day or two after intercourse). Flares also occur sometimes the week prior to menses, foods high in potassium, and during stress

Urinary frequency is present in what % of patients? 90%

Etiology

The mucous surface coating of the bladder contains glycosaminoglycans (GAGs), which normally act to regulate permeability. If the GAG layer is defective, as appears to be the case in IC, toxic substances in the urine can penetrate the urothelium and gain access to the deeper layers of the bladder, inducing a tissue reaction. Potassium is likely the primary toxin, leaking through the epithelium, thus activating the nerves and directly depolarizing them. Mast cells are also close to nerves and could also become activated, releasing histamine, which causes local pain and irritation to tissues → irritation causes release of substance P, which stimulates mast cells further and creates a bigger leak in the uroepithelium, releasing more K^+

Diagnosis

Must have at least one of these symptoms → (1) frequency or (2) pain linked to bladder AND must have the findings of at least one of these (1) Classic Hunner's Ulcer or (2) Diffuse glomerulations x 3 quadrants

Identify the disorder using the Pelvic Pain and Urgency/Frequency (PUF) Patient Symptom Scale questionnaire. A high PUF score is one reason to suspect IC. Intravesical potassium sensitivity test can be used to determine whether patients with chronic pelvic pain have a urological component to their etiology of pain in 85% of patients with + testing. (Parsons CL, Bullen M, Kahn BS, *et al*. Gynecologic presentation of interstitial cystitis as detected by intravesical potassium sensitivity. *Obstet Gynecol* 2001;98:127–32)

Exclude other causes. Decrease intake of carbonated drinks. The urethral syndrome may represent an earlier phase of IC in which the patient is not continuously symptomatic except the intensity and duration of the pain are greater in IC

Algorithm for diagnosis CPP bladder origin/IC

CPP with urgency/frequency → physical exam = tender bladder + PUF questionnaire score ≥ 8 + negative urinalysis → consider CPP of bladder origin (interstitial cystitis). Potassium test optional to help confirm diagnosis. If PUF score is > 10 to 12 → there is an 80% chance that the potassium sensitivity test will be positive

PUF questionaire

Pelvic Pain and Urgency / Frequency
Patient Symptom Scale

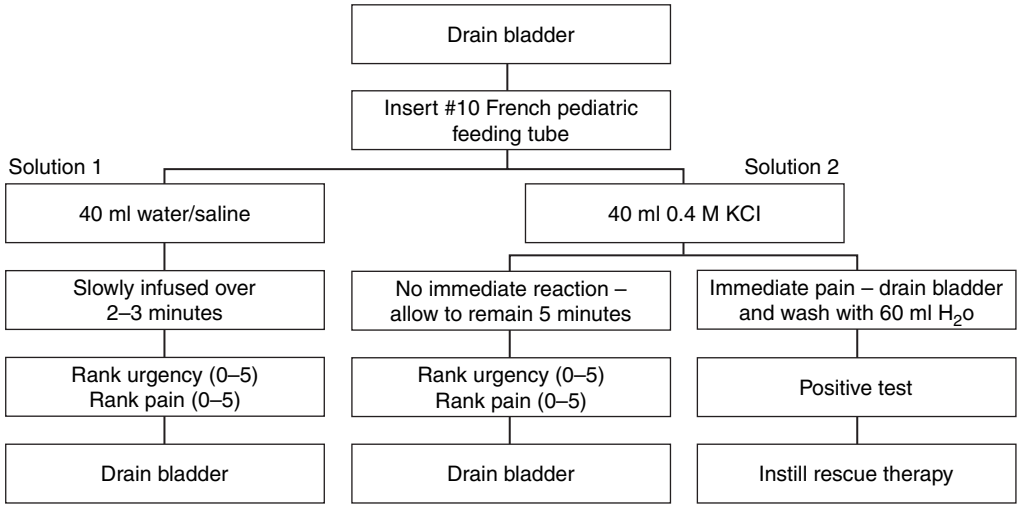
Please circle the answer that best describes how you feel for each question.

	0	1	2	3	4	SYMPTOM SCORE	BOTHER SCORE
1 How many times do you go to the bathroom during the day?	3–6	7–10	11–14	15–19	20+		
2 a. How many times do you go to the bathroom at night?	0	1	2	3	4+		
b. If you get up at night to go to the bathroom, does it bother you?	Never	Occasionally	Usually	Always			
3 Are you currently sexually active? YES ____ NO ____							
4 **a. If you are sexually active,** do you now or have you ever had pain or symptoms during or after sexual intercourse?	Never	Occasionally	Usually	Always			
b. If you have pain, does it make you avoid sexual intercourse?	Never	Occasionally	Usually	Always			
5 Do you have pain associated with your bladder or in your pelvis (vagina, labia, lower abdomen, urethra, perineum)?	Never	Occasionally	Usually	Always			
6 Do you still have urgency after you go to the bathroom?	Never	Occasionally	Usually	Always			
7 a. If you have pain, is it usually		Mild	Moderate	Severe			
b. Does your pain bother you?	Never	Occasionally	Usually	Always			
8 a. If you have urgency, is it usually		Mild	Moderate	Severe			
b. Does your urgency bother you?	Never	Occasionally	Usually	Always			

SYMPTOM SCORE (1, 2a, 4a, 5, 6, 7a, 8a)	
BOTHER SCORE (2b, 4b, 7b, 8b)	
TOTAL SCORE (Symptom Score + Bother Score) =	

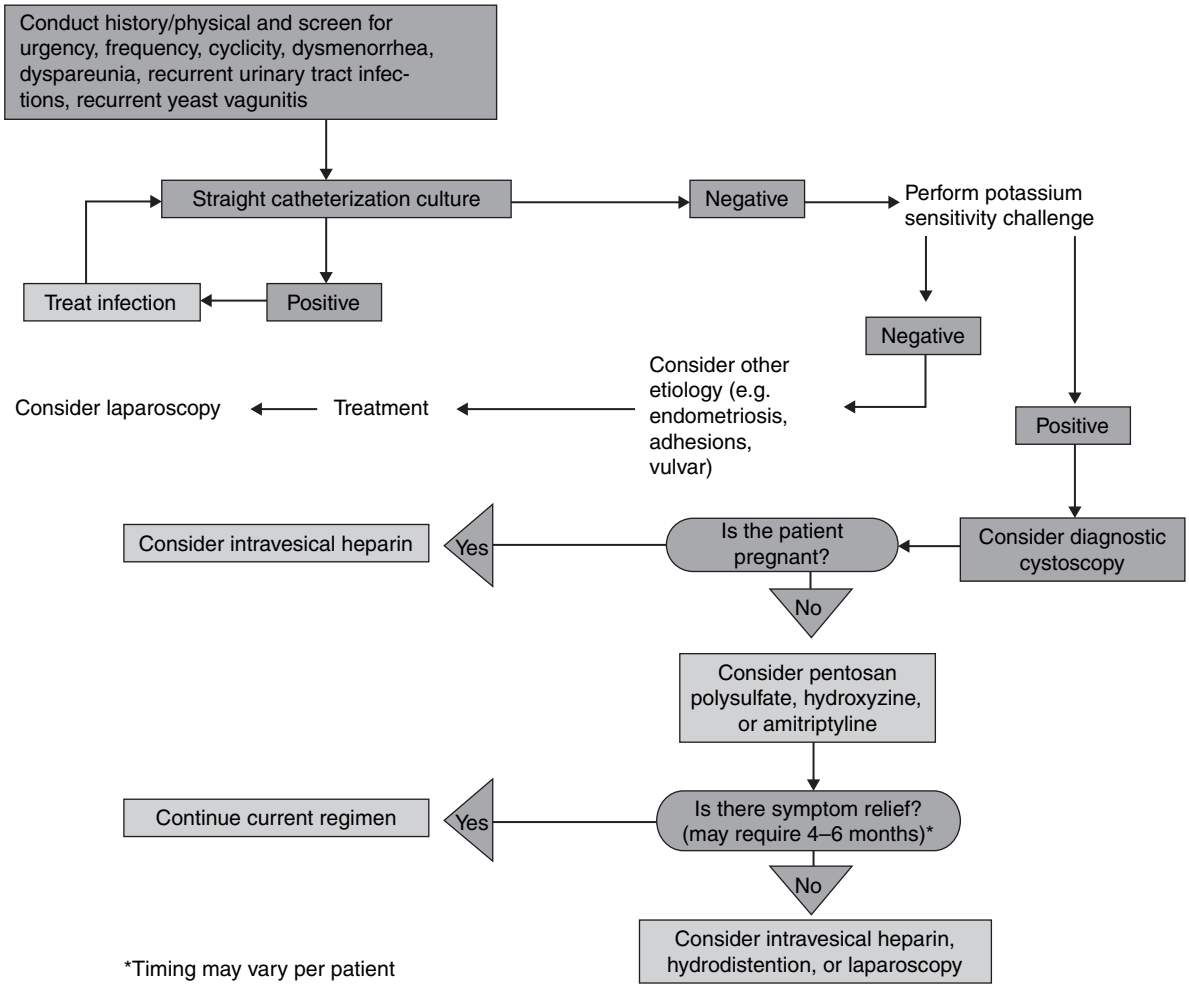
*Total score range is from 1 to 35. A total score of 10–14 = 75% likelihood of positive PST; 15–19 = 79%; 20+ = 94%

Potassium sensitivity test



Parsons CL *et al. J Urol* 1998;159:1862–67

Algorithm for management of interstitial cystitis



*Timing may vary per patient

Treatment

Elmiron® (pentosan polysulfate sodium) is only oral medicine approved by FDA to treat interstitial cystitis. This corrects the defect in the mucosal GAG layer. Atarax, Elavil, or Prozac are helpful medical therapies

Pentosan polysulfate sodium: mechanism of action
(1) GAG replacement
(2) Mast cell inhibition
  (a) Immunologic
  (b) Neurogenic
  (c) Other
(3) May modulate C-fiber sensory nerves–Inhibits calcium channels

Treat IC with Elmiron 100 mg b.i.d. to t.i.d. for 3–4 months then repeat PUF. One can increase to 600 mg b.i.d. with no liver function studies needed. There are no known drug interactions and Elmiron is category B

Dietary guidelines for interstitial cystitis

Food category	*Permitted foods*	*Foods to avoid or use cautiously*
Fruits	Blueberries, melons other than cantaloupe, and pears	All other fruits and juices made from them
Vegetables	Potatoes, homegrown tomatoes, and vegetables other than those listed on the right	Fave beans, lima beans, onions, rhubarb, tofu, and store-bought tomatoes
Milk/dairy	White chocolate, cottage cheese, American cheese, milk	Aged cheeses, sour cream, eggs, yogurt, chocolate
Carbohydrates/grains	Pasta, rice, and breads other than those listed on the right	Rye and sourdough breads
Meats/fish	Poultry, fish, and meats other than those listed on the right	Aged, canned, cured, processed and smoked meats and fish; anchovies; caviar; chicken livers; corned beef; and meats that contain nitrates or nitrites
Nuts	Almonds, cashews, and pine nuts	Most other nuts
Beverages	Bottled or spring water; decaffeinated, acid-free coffee and tea; some herbal teas	Alcoholic beverages; beer and wine; carbonated drinks; coffee, tea, and cranberry juice
Seasonings	Garlic and seasonings other than those listed on the right	Mayonnaise, miso, spicy foods (especially Chinese, Mexican, Indian, and Thai foods)
Preservatives		Benzol alcohol, citric acid, monosodium glutamate, aspartame, saccharin, and foods containg preservatives, artificial ingredients/colors

Multimedical therapies:
Elavil 25 mg every night
Atarax 25 mg hs or 25 mg every AM and q.hs
Ditropan XR 5–10 mg orally every day
Prozac 10–20 mg orally daily

Intravesical therapy (use # 10 French Ped tube & instill into bladder)
Anesthetic solution → 4–6 mg/kg 5% lidocaine and equal volume 8.4% $NaHCO_3$
Treatment solution → Elmiron 100 mg or 10–20 K heparin in 20 cc 1% lidocaine and 3 cc 8.4% $NaHCO_3$

INTERVAL DELIVERY

Definition

A long delay between delivery of fetuses in a multiple gestation

Contraindications

Abruption, fetal distress, chorioamnionitis, labor refractory to tocolysis

Informed consent

Discuss financial burden, prematurity, chances of success and neonatal morbidity

Work-up

CBC, US, evaluate symptoms of labor, consider amnio for maturity, NO digital exams, DO NOT remove placenta, obtain pt, ptt, platelet count, fibrinogen and FDP

INTRAUTERINE DEVICES (IUDs)

Insertion of IUD how many days prior to ovulation time decreases inflammatory reaction? 2 days

Single doxycycline 200 mg dose. Decreased unscheduled postinsertion visits for pain, discharge and bleeding. Did not decrease infection rate.

Some prefer to insert during menses so cramping associated with insertion becomes less noticeable and so that the physician can be more confident that the patient is not pregnant.

Women having abortions are often motivated to begin effective contraception, and IUD or LNG-IUS are excellent choices. These are also excellent choices for overweight women in that these are not contraindicated in association with obesity

Mechanism

Prevent fertilization of egg in fallopian tube by reducing number of sperm in tube and possibly by an additional effect of copper on fertility of the egg. The progestin-containing IUDs act mainly by rendering the endometrium atrophic thereby interfering with the implantation of fertilized egg. Also, in what % of women using progestin-type IUDs for at least a year, is ovulation prevented? 50%

Avoid inserting IUDs

In patients with multiple sexual partners or in patients who have an STD

Contraindications

Pregnancy, pelvic malignancy, PID, hyperbilirubinemia (due to Wilson's disease – applies only to those IUDs containing copper)

Relative contraindications

History of ectopic, PID, severe dysmenorrhea, sickle cell anemia, congenital anomalies of the genital tract and valvular heart disease

Possible complications

Expulsion, perforation, dysmenorrhea and pregnancy – ectopic or IUP

Treatment of pregnancy complication – rule out ectopic (2–3% chance), remove IUD ASAP, advise about spontaneous abortion, septic abortion, preterm labor, offer pregnancy termination

Technique

(1) Analgesics and an antibiotic
(2) Antiseptic to cervix and paracervical placed at 5 and 7 o'clock
(3) Sound if < 6 or > 9 cm (do not insert)
(4) Patient education – check string and have patient return to office after menses

Teach patient to check tail of IUD

Absence or longer tail may mean IUD has been expelled or is in process of being expelled. Pregnancy needs to be ruled out. If the tail is absent or shorter than usual, this could be due to the IUD having migrated or perforated the uterine wall

(5) OCPs or some other birth control method for the first month after insertion. Some use second method during ovulation every month
(6) Antibiotic given 1 h prior to insertion → doxycycline 200 mg

Types of IUDs

(1) ParaGard® T 380A is an IUD with copper wire wrapped around it

Approved for how many years of use? 10 years
Effective for at least how many years? 12 years
First-year failure rate in typical use (%) 0.8

Ortho-McNeil offers IUDs free of charge to financially disadvantaged patients if the provider will insert the IUD free of charge

A levonorgestrel intrauterine system can protect against endometrial hyperplasia and endometrial proliferation

(2) Progestasert® is an IUD that releases progesterone at rate of 65 µg /day

Approved for how long of use in United States? 1 year
Approved for use how long in France? 18 months

(3) Mirena (Berlex) is an IUD that releases levonorgestrel at	20 µg /day
Approved for how many years of use?	5 years
Effective for at least how many years?	7 years
First-year failure rate in typical use (%)	0.1
Berlex information line is 1-866-647-3646	

INTRAUTERINE GROWTH RESTRICTION

Small for gestational age (IUGR/SGA)

What % of pregnancies?	7–10%
Type I symmetric (viral infection, chromosomal or congenital anomaly). Restricted AC and HC	
Type I IUGR begins early in gestation, entire fetus proportionally small all	20% abnormal
Ponderal index is	Normal
Head, abdominal circumference, length and weight all	< 10%
Type II asymmetric (increased B/P, DM, PIH, plac abnl, renal disease, multiple gestation). Restricted – AC only	
Type II IUGR begins later in gestation, preserving the head and femur	80%
Decrease in abdominal circumference only	
Intermediate type probably occurs in middle phase of growth (less common than type I+II)	
Intermediate type may result from lupus, nephritis, vasculitis and is increased in diabetic mothers	
FL/AC normal ratio	0.22
Abnormal ratio	0.23
Effective for asymmetric IUGR	
What is the single parameter that is most predictive of IUGR with accurate knowledge of dates?	AC

Ponderal index

Closely related to perinatal morbidity than is birth-weight percentile	
The perinatal mortality is higher in IUGR compared to normal pregnancies by	5–10 times normal

Etiology of IUGR

Maternal	*Placental*	*Fetal*
Pre-eclampsia	Abnormal presentation	Chromosomal abnormalities
Chronic hypertension	Chronic villitis	Multifactorial defects
Chronic renal disease	Placenta infarcts	Infections
Connective tissue disorder	Placenta hemangiomas	Multifetal pregnancies
Diabetes with vascular lesions	Placenta previa	
Sickle cell anemia	Circumvallate placenta	
Cardiac disease Class III or IV		
Severe malnutrition		
Smoking		
Alcohol ingestion		
Infection		

Screening and diagnosis of IUGR

Antepartum screening methods to detect IUGR fetus include:
(1) Careful serial measurement of uterine fundal height
(2) Progressive weight gain of the month

(3) Growth profile by ultrasound scanning
  (a) Progressive growth of biparietal diameter, fetal limb length, head circumference
  (b) Amniotic fluid volume
  (Oligohydramnios is a common finding in IUGR) 90% of cases may be the earliest sign detected on ultrasound
  (c) Head to abdominal circumference
  HC/AC ratio in a normal growing fetus is:
  1 > before 32 weeks
  1 = at 32 to 34 weeks
  1 < after 34 weeks
  In fetus affected by asymmetric growth restriction, the HC remains larger than that of the body. The HC/AC ratio is then elevated
  (d) Femur to abdomen ratio
  Femur length is minimally affected by fetal growth impairment
  Abdominal circumference which is the most affected measurement
  FL/AC remains constant after 20 weeks. FL/AC is 22 at all gestational ages from 21 weeks to term
  FL/AC ratio greater than 23.5 suggests IUGR
  (e) Doppler wave form analysis. S/D ratio of umbilical artery.
  The development of Doppler ultrasound has provided the obstetrician with a new tool for the assessment of IUGR fetus
  The researchers found that the negative predictive value of a normal S/D ratio (normal S/D < 3) was 95%
  The positive predictive value of an S/D ratio greater than 3.0 was 49%
  An exciting possibility of Doppler examination is that it may be useful in making the critical distinction between the fetus that is small and healthy (SGA) and the one that is truly growth retarded. The majority of SGA babies have normal S/D ratio
  Reversed end diastolic flow in the umbilical artery reflects severe fetal compromise and is an ominous finding. It is associated with 50–64% mortality rate, so delivery of the fetus is recommended when reversed end diastolic flow is detected

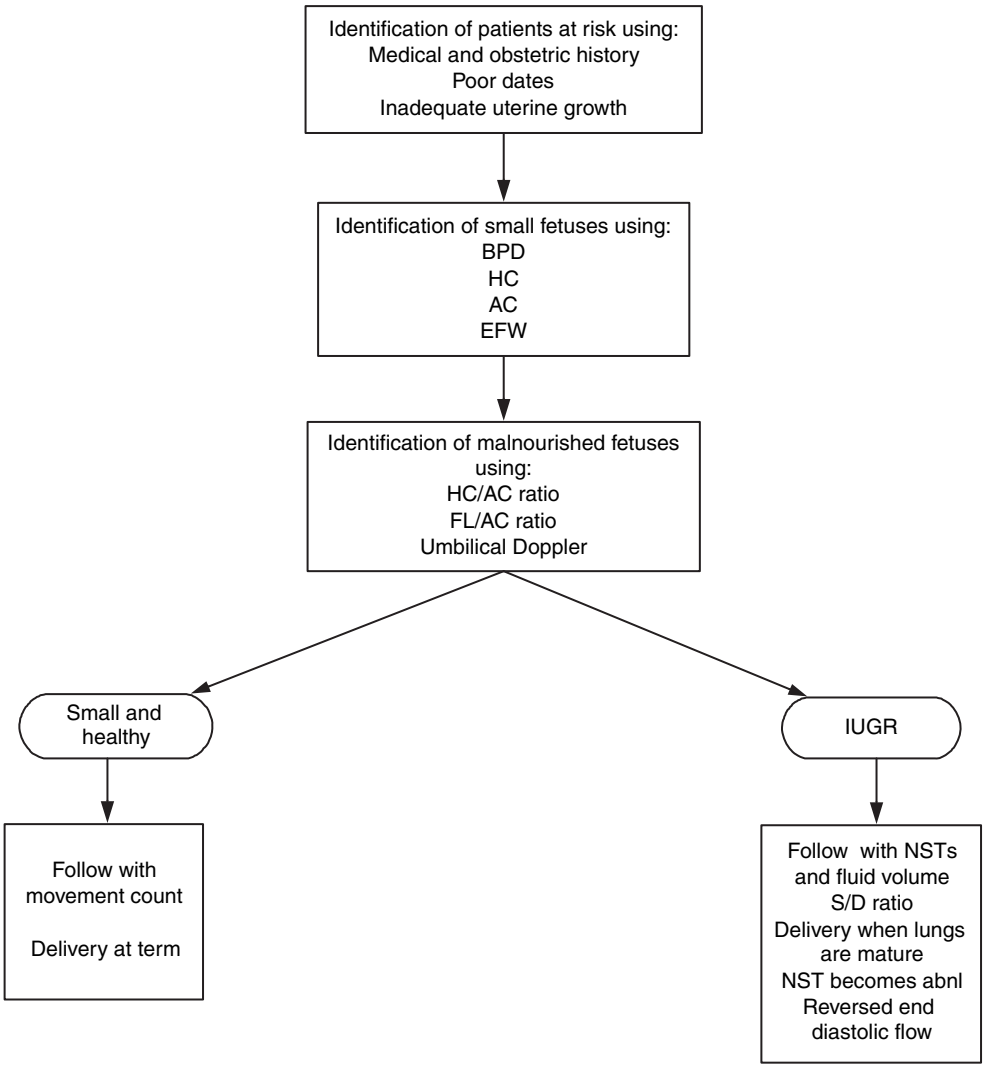
Management

Remote from term

(1) Intervention to improve intrauterine environment
(2) Avoid smoking, alcohol, drugs
(3) Control maternal disease
(4) Adequate maternal nutrition
(5) Decrease physical activity (bed rest)
(6) Fetal surveillance
  (a) NST
  Depending on the clinical circumstance the frequency of NST testing varies from once every week to every day
  Daily NSTs are indicated for patients with severe IUGR and with S/D ratios above 6
  (b) Contraction stress test (CST)
  (c) Biophysical profile (BPP)
  – (b) and (c) may be used to follow abnormal NSTs
  – delivery of the baby is the best management when the back-up test suggests fetal compromise
  (d) Ultrasound every 2–3 weeks for internal growth
  (e) Fluid volume
  This evaluation should be performed every week and the frequency of NST testing should be increased if the amount of fluid decreases. Delivery may be indicated if severe oligohydramnios develops

Close to term or during delivery

(1) Close monitoring during labor
(2) Because of the high incidence of intrapartum asphyxia, labor and delivery in IUGR babies should be managed aggressively. The liberal use of Cesarean section is advised
(3) Direct fetal monitoring using scalp electrode and uterine pressure catheter should be initiated as early as possible
(4) Amnioinfusion should be performed early in labor if the amniotic fluid volume is decreased
(5) Even mild signs of distress should be followed with scalp stimulation or fetal scalp pH sampling
(6) The second stage of labor, with its well-known tendency toward low pH values, should be kept to a minimum (use of forceps or vacuum when the vertex is well below plus 2 station)
(7) The best choice for pain relief during labor is epidural anesthesia
(8) The placenta of an IUGR baby needs careful examination by a competent placental pathologist
(9) Pediatrician should be present at the time of delivery


Identification of patients at risk using:
Medical and obstetric history
Poor dates
Inadequate uterine growth
Identification of small fetuses using:
BPD
HC
AC
EFW
Identification of malnourished fetuses using:
HC/AC ratio
FL/AC ratio
Umbilical Doppler
Small and healthy
IUGR
Follow with movement count
Delivery at term
Follow with NSTs and fluid volume
S/D ratio
Delivery when lungs are mature
NST becomes abnl
Reversed end diastolic flow

INTRAVENOUS FLUIDS

NS	0.9 NaCl
½ NS	0.45 NaCl
D5NS – How much dextrose does it contain in 1 liter?	5% or 50 g
RL	

IN VITRO FERTILIZATION

Pronuclei (of sperm and egg) fuse to form zygote (haploid 23 to diploid 46) on day	1
Zygote goes from 2 cells to 4 cells (blastomeres) on day	2
The blastomeres become a compact 16-cell morula on day	3
Syngamy is the last stage of the mingling process in which morula becomes blastocysts on days	4–5
The trophectoderm (outer cell of blastocysts) invades uterine wall between days	5–7
Embryogenesis begins between days	15–16
Pre-embryonic stage lasts until what day after fertilization?	14
Fertilized ovum reaches uterus in	5–7 days
The most widely used method of embryo biopsy is cleavage-stage biopsy. The primary reason patients stated they wanted PGD (prenatal genetic diagnosis) was their objection to termination of pregnancy (in what %)	60%
Blastomere biopsy results in a hole which is ? % bigger that probably contributes to increased reduction in implantation rates?	50–100%

IRON

Effects on iron and TIBC	Inflammatory disease	Decreased iron, decreased TIBC
	Aspirin	Decreased iron, increased TIBC
	Fe^{+} deficiency	Decreased iron, increased TIBC

IRRIGATION

Intraperitoneal irrigation with antibiotics is not recommended for use during infertility-related surgery because it may be associated with crystallization and adhesion formation

IRRITABLE BOWEL SYNDROME

Diagnosis

At least 3 months of continuous or recurrent symptoms of:

(1) Abdominal pain or discomfort that is:
  (a) Relieved with defecation and/or
  (b) Associated with a change in frequency of stool and/or
  (c) Associated with a change in consistency of stool and

(2) Two or more of the following on > 25% of occasions or days:
  (a) Altered stool frequency (> 3 bowel movements/day or < 3 bowel movements/week)
  (b) Altered stool form (lumpy/hard or loose/watery)
  (c) Altered stool passage (straining, urgency or feeling of incomplete evacuation)
  (d) Passage of mucus
  (e) Bloating or feeling of abdominal distension

Labs: < 50 CBC, LFTs, electrolytes, hemocult, consider sigmoidoscopy
> 50 CBC, LFTs, electrolytes, colonoscopy or air-contrast barium enema with sigmoidoscopy

Differential

Malabsorptive conditions (sprue), dietary factors (lactose intolerance, excessive caffeine intake), infection (*Giardia lamblia*), inflammatory

bowel disease (Crohn's disease, ulcerative colitis), psychological disorders (somatization, depression), miscellaneous (endometriosis)

Treatment

Lotronex® (alosetron) – IBS especially if prominent symptom is diarrhea. Does not work in men. It is a selective 5-HT3 receptor antagonist. Dose is 1 mg p.o. b.i.d. Lotronex should be used only short term. Risk of intestinal obstruction should be explained
Zelnorm® (tegaserod) – IBS especially if prominent symptom is constipation. Dose is 6 mg p.o. b.i.d.

ISOLATED GONADOTROPIN DEFICIENCY

Not result of isolated FSH or LH but a failure of these GnRH neurons to migrate successfully from the nasal region of the developing brain. Frequently associated with IGD is anosmia – Kallmann's syndrome (disease in male but has been referred to in female with anosmia *and* IGD → less appropriate)

KEGEL EXERCISES

Pelvic muscle exercises
Type I (aerobic) and Type II (anaerobic) muscle types are trained to hypertrophy so symptoms improve in 2–3 months
Type I (slow-twitch) – aerobic oxidative metabolism to support
Type II (fast-twitch) – anaerobic glycolytic metabolism to support
Kegels are less effective in postmenopausal females than premenopausal females
66% Incidence of reduction during 16 week PME protocol, 2 months or longer
Written or verbal instructions usually inadequate
Errors in technique – contraction of auxiliary muscles (gluteal, thigh) and most seriously – a Valsalva or straining down effort
Audio tape: HELP for Incontinent People (1-800-252-3337)
30–80 per day with 10 s relaxation recommended between contractions
Quick 'flick,' 'pull', 'squeeze', 'tighten', 'lift', 'clench', 'contract'
Cones can be purchased to help train pelvic floor
FOLLOW-UP: Absence of gluteal or thigh contractions. Digital palpation of pelvic floor during PME – descent of clitoris and inward/up motion of anus – lifting of exam finger by three layers of perivaginal muscle layers

Patient guide to prevention or treatment of urinary incontinence

Q **What is pelvic muscle exercise?**
A Pelvic muscle exercise (also called Kegel exercise) is the tightening and relaxing of the muscles that support the uterus, bladder and other pelvic organs. Strong pelvic muscles can help prevent accidental urine leakage

Q **Why should I do pelvic muscle exercise?**
A Regular pelvic muscle exercise makes these muscles stronger. Women who have a problem with urine leakage have been able to eliminate or greatly improve this problem just by doing pelvic muscle exercise every day

Q **How do I do pelvic muscle exercise?**
A The feeling you should have when you are doing pelvic muscle exercise is that all the pelvic muscles are drawing inward and upward. A good way to learn the exercise is to pretend that you are trying to avoid the embarrassing passing of intestinal gas. Think about the muscles that tighten (or contract) to keep the gas from escaping. Bring that same tightening forward to the muscles around your vagina and move the contraction up to the higher levels of your pelvis. There are three layers of muscle to tighten and you can feel them as you move the contraction up to the highest level

Continued

Continued

Important tips

(1) Each contraction should be as hard or intense as you can make it without tightening your thigh or buttock muscles

(2) Work up to holding each contraction for 2 s, then for 4, 6, 8 and 10 s as your muscles become stronger

(3) Rest for at least 10 s (longer if you need to) between each contraction, so that each one is as hard as you can make it

(4) Each contraction should reach the highest level of your pelvis; you will feel the pulling up and in over the three distinct layers of muscle

Q **How often should I do these exercises and how many should I do?**

A If you have some problem with urine leakage, we recommend 30 contractions each day. You can expect to see some improvement after doing regular pelvic muscle exercise for about 6–8 weeks, so don't be discouraged if you don't notice results right away. Remembering to contract the muscles prior to coughing, blowing your nose or sneezing will help you avoid leakage. This technique can also help to control sudden urges to urinate

Q **Are there any mistakes to avoid with pelvic muscle exercise?**

A The most serious mistake is to strain down instead of drawing the muscles up and in. Trying this will show you what NOT to do: take a breath, hold it and push down with your abdomen. You can feel a pushing out around your vagina. It is very important to avoid this straining down. To keep from straining down while you do pelvic muscle exercise, exhale gently and keep your mouth open each time you tighten the pelvic muscles. You can also keep your hands on your abdomen while you tighten your pelvic muscles. If you feel your stomach pushing out against your hands, you are straining down. Do not continue with pelvic muscle exercise until you check with your physician to learn how to do it properly

Avoid tightening thigh and buttock muscles. This takes away from the effectiveness of your pelvic muscle exercise. If it seems impossible not to tighten the thigh and buttock muscles, concentrate first on full relaxation and then try gentle 'flicks' of the pelvic muscles; for example, 'flick, relax, flick, relax'. After gaining confidence, try a second flick on top of the first and then a third – 'flick flick, flick, relax' – working the muscles to higher layers with each flick

Keys to success

It is a challenge to work any new health habit into your everyday life. Here are some things that other women have found helpful in making pelvic muscle exercise a regular part of their self-care:

(1) Think about your usual day and pick a time (about 15 min) when you will be able to do your pelvic muscle exercise every day. Maybe when you first wake up is a good time or maybe afternoon or evening is better

(2) Decide on a way to remind yourself to do pelvic muscle exercise. You might put a note on your bathroom mirror or plan to do your exercises during a TV program that you watch every day. Just think of something that happens every day that will remind you to do it

(3) Reward yourself for exercising each time you do it. You might get some special small candies and treat yourself to one each day that you remember to do pelvic muscle exercise. Or you could draw a small flower on your calendar to mark each day you exercise and get yourself a real bouquet of flowers when you have drawn 10 flowers. Any small reward that you know will keep you working on this new habit is fine

(4) Monitor your progress, especially if you have a problem with urine leakage. You might want to keep a daily diary of whether you have had an accident and how many times it happened. Over the weeks, you will be able to measure your own progress. Another way is to see whether you can slow or stop your urine stream when you are going to the bathroom. We recommend that you try this no more than once a week. As your pelvic muscles get stronger, you will be able to stop the stream more quickly

Good luck on your program of pelvic muscle exercise! Please call your physician if you have any questions about this program to strengthen your pelvic muscles

LABIAL AGGLUTINATION

Prevalence in white females 20%
Prevalence in black and Hispanic females < 5%

Etiology

(1) Inflammatory reaction – anestrogenic state
(2) Recurrent UTI
(3) *E. coli* – primary bacteria
(4) Can be sexual abuse
(5) History of new occurrence → differential diagnosis to rule out:
  (a) Müllerian agenesis
  (b) Androgen insensitivity syndrome
  (c) Hermaphroditism
  (d) Ambiguous genitalia of adrenogenital syndrome
  (e) Imperforate hymen

Treatment

Asymptomatic – leave alone → usually hormonal + pH changes cause spontaneous resolution
Symptomatic:
(1) Perineal hygiene (Sitz baths)
(2) Treatment consists of estrogen cream b.i.d. for 7–10 days
  Separation usually occurs in 1–4 weeks
(3) Do not forcefully separate
(4) Do not surgically incise – adhesions will form if incised
(5) If recurs – treat only if symptomatic

LABIAL CYSTS – BARTHOLIN'S CYST

Etiology – obstruction of duct due to inflammation, trauma or cancer
Symptoms – rapidly develop in 2–4 days
Symptoms consist of pain, dyspareunia and pain with ambulation
Signs – mass, erythema, tenderness, edema, cellulitis

Treatment

(1) Asymptomatic < 40 years of age — No treatment
(2) Asymptomatic > 40 years of age — Excise
(3) Acute adenitis or abscess — Antibiotics and Sitz baths
(4) Symptomatic — Develop fistulous tract
  (a) Word catheter – place under mucus epithelium, use saline not air
  (b) Marsupialization – mucus epithelium to be sutured back to open
  (c) Excision – if persistent deep infection or multiple recurrences or to rule out adenocarcinoma in age > 40
  Caution: branches of pudendal artery to be avoided so as to avoid postop vulvar hematoma

Excision if recurrent, multiple recurrence or patient age > 40

LABOR

Cardinal movements

Engagement, descent, flexion, internal rotation, extension, external rotation, expulsion

Montevideo units

Labor 80–120 MV units
Three contractions in 10 min each of 50 mmHg intensity 150 MV units
Contractions palpable only after intensity reaches > 10 mmHg
Prior to C-section for dystocia, uterine activity should reach at least 200–275 MV units
It takes 30–40 min for the full effect of an increase in oxytocin dosage to be evident

LABOR MEDICATIONS

IVFs

RL – better for pre-major conduction anesthesia
(Infusing any solution containing dextrose at high rates may result in osmotic diuresis and consequent dehydration)

Pain

Sublimaze (fentanyl) 1–2 cc IV q. 1–2 h p.r.n. pain
Demerol 50–100 mg, Phenergan 15–50 mg = IM every 3–4 h or dec dosages IV
Stadol 1 mg IV or 1–2 mg IM every 4 h
(Do not use if delivery anticipated within 4 h)
Nubain® 5–20 mg IV or 10–20 mg IM every 2–4 h
(Maximum daily dose is 160 mg)

Sedation

Nembutal® 100–200 mg p.o.
Seconal® 100–200 mg IM
Demerol 100 mg IM
Morphine 10 mg IM

Antacid

30 ml of 0.3 M sodium citrate with citric acid (Bicitra®) before anticipated general anesthesia to protect against aspiration pneumonitis

Hypertension

Apresoline® 5–10 mg IV bolus every 15–20 min until diastolic 90–100
Normodyne® 20, 40, 80 mg then 80 mg thereafter at 10-min intervals (max of 300 mg)
Adalat®, Procardia® 10–20 mg p.o. every 20–30 min

Ripening/augmentation

Prepidil Gel intracervical every 6–8 h no more than 3 in 24 h
Prostin 2.5 mg suppository intravaginal every 3–6 h
Cervidil tampon intravaginal 12–15 h prior to induction
Cytotec 25 µg intravaginal every 3–6 h
Pitocin 0.5–1 mIU/min with inc to 1–2 mIU/min at 30–60-min intervals
2 mIU/min with inc to 2 mIU/min at 30–60-min intervals
6 mU/min with inc to 6 mU/min at 20-min intervals

Induction

'DrT' Pitocin protocol

Augment:	1 mu/min IV then inc by 1 mu/min IV every 30 min
Active I:	6 mu/min IV and inc by 1 mu/min every 30 min
Active II:	6 mu/min IV and inc by 6 mu/min every 15 min

36 mu/min is maximum unless ordered to increase by MD

Diabetic management

Regular insulin 50 units in 500 ml of NS
Shake well – run out 50 ml waste to ensure absorption of surfaces
Continuous pump rate of 0.5 units/h or > with increments of 0.5–1 unit/h to obtain necessary glucose levels
Patient should also receive D5LR ml/h to avoid starvation during labor
Check glucose values every hour with finger stick test strips
Adjust infusion p.r.n.

LAPAROSCOPY

Skill at open laparotomy does not necessarily transfer to skill at minimally invasive techniques. (Figert PL, Park AE, Witzke DB, *et al*. Transfer of training in acquiring laparoscopic skills. *J Am Coll Surg* 2001; 193:533–7)

Main reasons laparoscopists get sued

(1) Inexperience
(2) Defects in eye–hand coordination
(3) Ignorance of three-dimensional anatomy
(4) Equipment and technique failure
(5) Infection
(6) Improper patient selection
(7) Repositioning patients during surgery
(8) Failure to plan and be prepared for foreseeable complications

Complications

Surgeons in solo practice or those with a variable surgical assistant were how much more likely to have a complication? 7.74 and 4.8 ×

(1) Verres needle stick into stomach 0.03%
Rx with observation if no leakage. If leakage, bleeding or suspect posterior injury then 2-layer closure, NG tube and H_2 blockers

(2) Perforation of small or large bowel @ 1%
Incidence of injury to small bowel not really known. Incidence of laparoscopic bowel perforation and abrasion is 0.2% and 0.6%, respectively
What % of injuries are not recognized at time of surgery? 69%
What % of injuries require laparotomy? 80%
What are some of the first presenting signs and symptoms of an unrecognized laparoscopic bowel injury?
(a) Persistent focal pain in a trocar site
(b) Abdominal distention, diarrhea, leukopenia
(c) Free air not reliable due to retention of CO_2 under diaphragm (@ 40% patients will have more than 2 cm of free air at 24 h)
Repair – majority of trocar punctures require suture reapproximation
– needle punctures usually can be managed conservatively and do not require any treatment
– burn injuries require resection of 1–2 cm of viable tissue around injury site to ensure undamaged tissue

(3) Perforation of vessel 2%
Most common site of vascular injury is the inferior epigastrics
Avoid epigastrics by placing trocar lateral to the rectus muscles and/or transillumination of the lower abdominal wall
CT scans found that lateral trocar should be placed 8 cm from the midline and at least 5 cm above the symphysis to minimize risk of vessel injury

(4) Burn injuries
(a) Direct coupling – monopolar electrosurgery injury due to conductive injury touch with other structures
(b) Capacitative coupling – if 'return' to dispersive electrode is blocked by insulation (*avoid hybrids*)
(c) Insulation defect – type of direct coupling injury
(d) Dispersive electrode injury – when pad becomes partially detached → increases current density → skin burn

(5) Uterine perforation 0.5%

(6) Subcutaneous emphysema 1%

(7) Bladder injury < 1%
(a) Minimize this by making certain the bladder is empty and that placement of secondary trocars are under direct visualization
(b) If recognized – suture at time of surgery
(c) If unrecognized – patient usually presents with urinary ascites, abdominal pain and distension with fever, chills, oliguria, nausea and vomiting. BUN and creatinine will be elevated and patients will respond to aggressive hydration and bladder drainage. Cystoscopy is rarely indicated and these type of injuries will heal spontaneously and do not require surgical repair

At laparoscopy with usual insufflation flow rate of gas of 1 liter/min, abd pressure should not be > 20 mmHg

Air embolus at time of laparoscopy is from Trendelenberg position, not laparoscopy

Delayed recognition of bowel injury is an independent predictor of death from laparoscopic entry injuries

Newer instrumentation – Endoscopic Floating Ball (TissueLink Medical, Dover, NH)
Laparoscopic 'Plasma' Forceps and 'Plasma' Dissector (Gyrus Medical Inc., Maple Grove, MN)
Capio Suture Capturing Device (Boston Scientific, Urology/Gynecology, Natick, MA)
LigaSure Atlas (Valleylab, Boulder, CO) for sealing + cutting
LigaSure Lap (Valleylab) – 5 mm sealer single use replaces Klepenger

LATE DECELERATIONS

Exhibited prior to acidemia. Variability of baseline disappears as acidemia develops

Lates can occur in

Maternal hypotension (epidural)
Increased uterine activity (oxytocin stimulation)
Placental dysfunction (maternal hypertension, DM, collagen-vascular disorders, abruption)

Action for concern

Observe labor if pH	> 7.25
Repeat pH within 30 min if pH	7.2–7.25
Recheck STAT on way to OR if pH	< 7.20

If low pH again – C-section

Treatment

D/C Pitocin. Give terbutaline. Turn patient on her side. Increase IVFs. Give O_2. R/O prolapsed cord. Correct any decreased B/P due to epidural

Why bradycardia with hypoxia?

Hypoxia affects the chemo + baroreceptors, which triggers a cascade of events that cause vagal stimulation which ends in bradycardia and is eventually directly hypoxic to the fetal cardiac muscle
No single fetal heart rate is associated with neurological injury

Pearls for managing

(1) Reduce aorto-caval and/or cord compression = change patient positioning
(2) Restore intravascular volume = administer intravenous fluid bolus
(3) Reduce uterine activity = d/c oxytocin drip and give tocolytic Rx
(4) Enhance oxygen delivery to fetus = give supplemental oxygen
(5) Resolve hypotension = administer vasopressor therapy (ephedrine)
(6) Resolve oligohydramnios and cord compression = perform transcervical amnioinfusion

Prolonged decelerations

Fetal bradycardias and prolonged decelerations are 2 distinct entities; the first usually does not warrant immediate intervention

LEEP

LEEP = LLETZ = LETZ

Important points to remember

(1) Long-term sequelae of LEEP can be cervical stenosis and cervical incompetence
(2) Complications of LEEP include infection and bleeding
(3) Most cases of ASCUS regress spontaneously and therefore do not require LEEP
(4) Use caution when performing LEEP in the luteal phase. The luteal phase of the cycle is characterized by increased vascularity of the endometrium and underlying tissue, which may account for more bleeding than procedures done in the follicular phase

LEGAL TERMS

Important terms to remember

Fiduciary beneficence – obligation to act for the benefit of the patient
Patient autonomy – right of patient to make informed decisions regarding her health care
Justice – extent to which an adequate level of health care is made equally available and accessible to all

LEIOMYOMAS

Chromosomes	1, 6, 7, 12, and 14
Bleeding is reason for what % of hysterectomies?	33%
Pain and pressure is reason for this % of hysterectomies	33%
This % of hysterectomies are done for myomas	30%

Most common pelvic tumor in females. Seen in what % of reproductive women? 20%
Most frequent non-random cytogenic abnormality del (7)(q21)
Labor – asymptomatic if fibroid < 3 cm
can cause PTL, pain, abruption, C-section if fibroid > 3 cm
can cause obstructed labor if fibroid > 6 cm
Degeneration of fibroid – *See* Red degeneration

LEIOMYOSARCOMA

How many mitoses per 10 HPF should be seen to diagnose leiomyosarcoma? > 10
How many mitoses per 10 HPF with moderate to severe cellular atypia should be seen? 5–10
What % recur when > 10 mitoses are seen with 10 HPF? 50–75%
What % recur when 5–10 mitoses are seen per 10 HPF? 33%
Doxorubicin chemo for extrauterine or distant metastases if these are found. Follow-up exams should be done at 3-month intervals

LEMON SIGN

US finding for NTD which can be decreased by routine folic acid given 3 months prior in dose of 0.4 mg
Give this dose after an occurrence or recurrence 4 mg
The specific image seen on US is 'frontal notching'
The banana sign is associated with cerebellar changes

LICHEN SCLEROSUS

The treatment for lichen sclerosus is clobetasol (Temovate®) b.i.d. × 2–3 weeks then q. hs till gone 0.05%
Some prefer testosterone 2% cream between courses of clobetasol
This % of patients with lichen sclerosus develop squamous cell hyperplasia 27–35%
This % of patients with lichen sclerosus develop intraepithelial neoplasia 5%
This % of patients with lichen sclerosus develop vulvar cancer 4%

Etiology
Unknown

Histology
White, thinning epithelium, loss of rete pegs, dermal homogenization

Symptoms
Itching, dyspareunia, subepithelial hemorrhages
Dyspareunia and decreased coitus found in 80%
• Most often in PMP patients but can occur in extragenital sites and in children – can be confused with child abuse
What % of lichen sclerosus cases occur in children? 15%

Treatment
Temovate 0.05% × 2–3 weeks then q. h till gone
2% testosterone propionate in white petroleum is optional during treatment with clobetasol
For persistent vulvar pruritis – intradermal injection of steroids and subcutaneous injection of absolute alcohol. Massage evenly

Potential side-effects
Intense vulvar itching, urinary retention
There is no good evidence that women with lichen sclerosus face a higher risk for vulvar carcinoma

LI–FRAUMENI SYNDROME

Mutation of *p53* tumor-suppressor gene on chromosome 17
Autosomal dominant malignancy that occurs by age 30

Breast, adrenocortical, brain ca, soft tissue and osteogenic sarcomas.
Leukemias. Rhabdomyosarcomas + osteosarcomas in children.
Breast and other tumors in their mothers

LUNG MATURITY

Development of fetal lungs

Glandular period (pulmonary tree)	3–16 weeks
Canalicular period (bronchioles)	16–24 weeks
Terminal sac period (alveolar)	
Amniocentesis is not warranted for pulmonary maturity prior to	33 weeks

Indirect methods

Can be performed to determine elective delivery such as:

(1) FHTs documented by non-electronic fetoscope by	20 weeks
(2) FHTs documented by Doppler for	30 weeks
(3) + hCG by a reliable lab for	36 weeks
(4) US measurement of C–R length ≥ 39 weeks done	6–11 weeks
(5) US measurement supporting clinical age of 39 weeks done	12–20 weeks

Direct tests

To determine fetal maturity are derived testing products of type II pneumocytes:

L/S (centrifuge + freeze)	2–3.5
Affected by blood and meconium	
PG	present
FSI (meconium and blood interferes with silicon tube)	≥ 47–48
Fluorescence polarization	> 55
OD at 650 mm	≥ 0.15
Lamellar body counts @	30 000–50 000
Ultrasound BPD/FL	9.2/7.3

LUPRON

See Depo-Lupron

LUPUS

During the crisis, the complement levels are decreased
Suspect female with recurrent DVT, cerebrovascular stroke or coronary thrombosis. Suspect if labs show false + VDRL, abnormal ptt or decreased platelets

LUTEAL CYSTS

Follicular

Most common

Frequently multiple	2 mm–1.5 cm
Most are asymptomatic. Transient tenderness if symptomatic	
Normal follicle becomes follicular if diameter	≥ 2.5–3 cm

Etiology

(1) Dominant follicle fails to rupture or
(2) Immature follicle fails to undergo atresia

Treatment

(1) Conservative – observe. Most reabsorb or silently rupture @ 4–8 weeks
(2) Surgery if
  (a) Mass prior to puberty or after menopause
  (b) Solid mass at any age
  (c) Cystic mass > 8 cm
  (d) Cystic mass 5–8 cm for over 8 weeks duration
(3) Transvaginal ultrasound – differentiate simple from complex cyst, serial measurements

• Surgery is cystectomy (usually laparoscopic)

Recurrence rate after laparoscopy is @	2%

Tissue sample p.r.n. – not cytology of fluid

Corpus luteum

	Most asymptomatic	3–4 cm
	Unilateral lower abdominal pain usually R-sided	66%
	Delayed menses. Intra-abdominal bleeding (minimal to massive) possible	
	Holban's classic syndrome – delay in normal period – spotting and unilateral pain – small, tender adnexal mass	
	Most rupture @ days	20–26
Differential diagnosis	(1) Ruptured corpus luteum (2) Ectopic (3) Ruptured endometrium (4) Torsion	
Diagnosis	hCG, vaginal ultrasound, culdocentesis	
Treatment	(1) Conservative – observe if unruptured or ruptures with small amount of fluid with minimum to moderate pain (2) Surgery – (cystectomy – operation of choice) if ruptured with hemoperitoneum or severe pain	
	Rate of recurrence after laparoscopy is @	14%

Theca lutein cysts

	Least common, most asymptomatic Bilateral, almost always large and massive	
Etiology	Prolonged stimulation, arise from increased ovarian sensitivity	
	Associated with:	
	(1) Molar pregnancy	50%
	(2) Choriocarcinoma	10%
	(3) Third-trimester conditions (twins, diabetes, Rh sensitivity)	
	(4) Infertility, ovulation-induction meds	
	(5) Hypothyroidism (rare)	
Symptoms	Vague symptoms – ascites, increased abdominal girth	
	Torsion and bleeding rare @	1%
Diagnosis	Palpation – ultrasound confirms	
Treatment	Observation – usually regresses. Handle delicately – do not drain – risk hemorrhage	

LUTEAL PHASE DEFICIENCY

Diagnosed with two endometrial biopsies late in cycle @ how many days prior to menses?	2
The biopsy is usually out of phase with stromal edema, fully secretory glands with secretions in lumen and no decidual formation yet at @ day #	22
Diagnosis suspected if increase in BBT lasting	10 days or <
Endo bx histology out of phase by	> 2 days
Should be documented in two successive cycles for diagnostic validity	

LYNCH I SYNDROME

Hereditary colon cancer – not associated with breast or ovarian cancers

LYNCH II SYNDROME

HNPCC syndrome – hereditary non-polyposis colon cancer

All family members at increased risk for proximal colon cancer, cancers of stomach, urinary tract, small bowel and bile duct. Females at increased risk of endometrial and epithelial ovarian cancer

Criteria

(1) Individual should be diagnosed < 30 years old
(2) Successive generations are affected
(3) Relatives with histologically verified colorectal cancer

What % of HNPCC families do not meet the Amsterdam criteria? 20%

Cause of Lynch II syndrome – germline mutations in the 'mismatch repair' genes *hMSH1*, *hMLH1*, *hMSH2* and *hMLH2*

Management

Colonoscopy to cecum q. 1–3 years beginning at age 20–25 years
Annual screening for endometrial cancer with transvaginal US and endometrial biopsy to begin at age 25–35 years
Prophylactic colectomy and TAHBSO – controversial

MACROSOMIA

Definition

Birth weight greater than 4000 g
What % of pregnancies have over 4000 g infants? 10–14%
What % of pregnancies have over 4500 g infants? 2.5%
What is the breakdown of races having macrosomic infants? W/B/A 12%/4%/5%
How much weight does an average human fetus near term gain per week? 250 g
Fetal weight CANNOT BE ACCURATELY determined! See below

Ultrasound predictability > 4000 g has an average predictability error > 300–400 g
All methods of predictability have comparable sensitivities of no more than 60%
Therefore, PREDICTABILITY OF SHOULDER DYSTOCIA IS LIMITED
Shoulder dystocia occurs in 5% of deliveries in gravidas whose infants weigh 4000–4250 g

Risks of macrosomia

Increase with maternal diabetes, post-term pregnancies, pregnancy weight gain > 35 #, abnormal GTT (esp. 1 h), obesity, maternal height > 5 ft 3 in, AMA > 35 years old, multiparity, male fetus, white race
The finding of an extremely low MSAFP level should alert the physician to possible fetal macrosomia. (Baschat AA, Harman CR, Farid G, *et al*. Very low second-trimester MSAFP: association with high birth weight. *Obstet Gynecol* 2002;99:531–6)

Fetal weight is usually decreased with chronic maternal hypertension, PIH, maternal tobacco abuse, increased altitudes

MAGNESIUM SULFATE

Therapeutic mag levels 4–7 mEq/l
Loss of patellar reflex 8–10 m Eq/l
Respiratory depression 10–12 mEq/l
Respiratory arrest ≥ 12 mEq/l
Cardiac arrest 30–35 mEq/l

Magnesium sulfate is contraindicated with myasthenia gravis, renal disease and hypocalcemia

MAMMOGRAPHY

Detects microcalcification of tumor @ what size? 1 mm (0.1 cm)
Tumor has grown how many years prior to detection by mammogram? 6–8 years

Mammography can still detect lesion @ how many years prior to clinical exam?	2
What % of improvement in breast cancer survival has mammography provided?	30%
Mortality is decreased by how much if female screened annually after age 40?	½
What is the false-negative rate for diagnosing malignancy with mammography?	10–20%

MANEUVERS

Brandt	Prevents inversion of uterus
Woods	'Corkscrew' to extract impacted shoulder
Marceau	Delivery of after-coming head with fingers in maxilla
Pinard	Delivery of lower extremities with Frank breech (knee from midline)
Scanzoni	Rotation of head with forceps with a pelvic curve
Ritgen	Extension of head with elevation of chin via rectum
McRobert's	Flexion of hips to disimpact shoulder
Prague	Managing persistence of the fetal spine directed toward the maternal spine (sacrum) by using two fingers to grasp shoulders of back down fetus while other hand draws feet over mother's abdomen
Bracht	Breech allowed to deliver spontaneously then body held against maternal symphysis

MASTALGIA

Treated with GnRH, bromocriptine, danazol, oil of evening primrose, tamoxifen. Switch from MPA to micronized progesterone or norethindrone. Ultrasound or appropriate biopsy if continues

Mastalgia (mastodynia) is confined to the breast tissue and may be cyclic or non-cyclic and diffuse or localized. All women presenting with mastalgia deserve a complete evaluation including: breast-oriented history, complete breast examination, mammography (if over age 25) and fine-needle aspiration of any palpable dominant breast mass

If no significant abnormality is discovered, the patient can be reassured that there is no evidence of breast cancer and that her symptoms are common to many women – probably physiologic (end-organ sensitivity)

Fewer than 15% of women with breast cancer present with pain as a chief complaint. Breast cancer pain is usually localized, non-cyclic and associated with a palpable mass

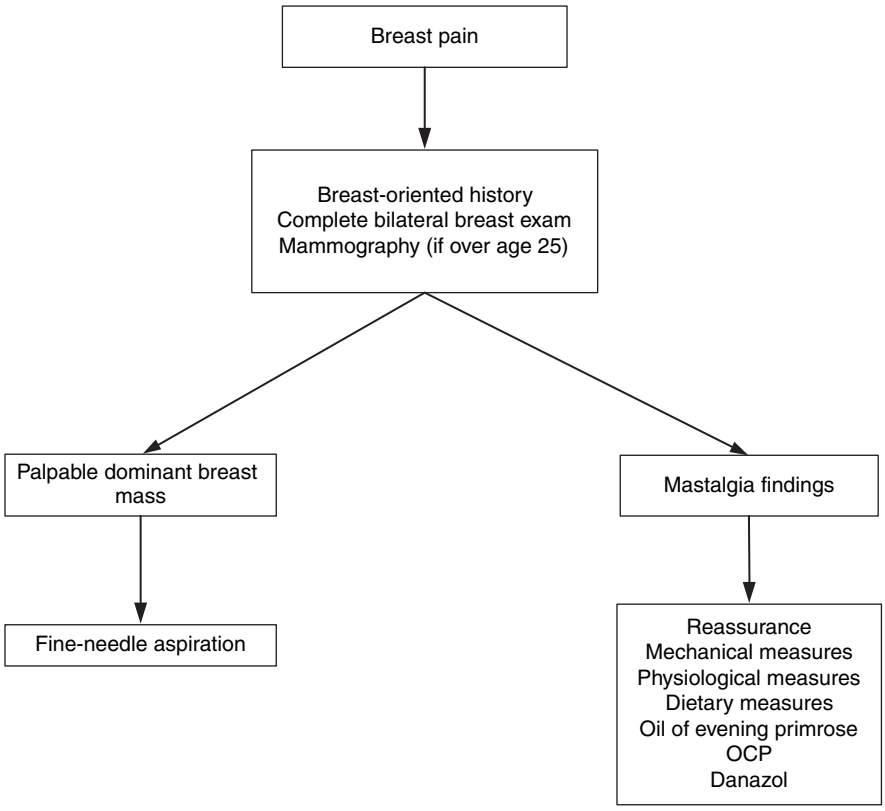
Treatment

More than 75% of women presenting with mastalgia, after complete breast evaluation, will be satisfied with, and appropriately treated by reassurance. If further therapy is required, it should be tried in a step-wise fashion starting with:

(1) *Mechanical measures:* changing to a brassiere with good support, no wires and no pressure points; heating pad or hot towels; massage
(2) *Physiological measures:* ventilation of any acute stress caused by exposure to breast cancer patients or information
(3) *Dietary measures:* weight reduction if obese; premenstrual salt restriction
(4) *Pharmacologic measures:*
    (a) 1/35 monophasic oral contraceptive therapy
    (b) Danazol – 100 mg twice a day until the mastalgia is controlled. In menstruating women, danazol treatment should begin during menstruation to avoid the possibility of pregnancy, a contraindication to danazol. The dose can be increased incrementally up to 400 mg a day
    (c) Oil of evening primrose

After the breast symptoms have been controlled for at least a month, the dose of danazol can often be reduced incrementally to as low as 50 mg per day. The patient should be maintained on the lowest effective dose for at least 6 months. As many as 50% of women will experience return of their mastalgia within 6 months after cessation of therapy. In these cases, danazol therapy can be repeated

Effective non-hormonal contraception should be practiced during danazol treatment. Side-effects of treatment include: irregular menstrual bleeding and masculinization



MASTECTOMY

Segmental (lumpectomy), simple, modified radical, radical and extended radical
Segmental – excision of a quadrant or lumpectomy
Simple – removal of entire breast but leaves nodes
Modified radical – en block removal of breast, pectoralis major FASCIA and axillary lymph nodes
Radical – en block removal of breast, pectoralis major muscles and axillary lymph nodes
Extended radical – radical including removal of INTERNAL MAMMARY nodes

MATERNAL–FETAL RELATIONSHIP

- Pregnant patient may refuse a diagnostic procedure, medical therapy or surgical procedure intended to enhance or preserve fetal well-being
- If patient refuses, the obstetrician may request involvement of the court
- Obstetrician should involve ethical principle of beneficence

MATURATION INDEX

Superficial ('wafer-thin' cytoplasm) cells – estrogen +
Parabasal (thick cytoplasm) cells demonstrate decreased estrogen –

Ovulation has 40% intermediate cells and with increase ERT, the superficial cells make up	60%
Postpartum and postmenopausal periods have no superficial cells but parabasal cells comprise	100%

McENDOE PROCEDURE

Operation to create a vagina. Dissect where vagina should be. Skin graft placed on foam rubber mold, 'inside of graft on outside'. One week later, pull form. Need to keep foam rubber form in for 3–4 h per day as skin can retract. Must continue for rest of patient's life

MEASLES

Rubeola – sixth to seventh largest killer among infectious diseases

Pathology

Fomites, fever, URI, red spots with bluish to white centers on buccal mucosa (Koplik's spots). 1–2 days after the above 2-week incubation period – RASH begins on head and descends to trunk and limbs

Diagnosis

Multinucleated giant cells. IgM antibodies in acute serum taken 2–3 days after the onset of rash. IgG antibodies in later samples. PCR (polymerase chain reaction)

Virology

RNA virus – morbillivirus

Complications

Can exacerbate tuberculosis

Diarrhea	9%
Bacterial or viral otitis and pneumonitis	7%
Encephalitis	20–40%
Mortality	20%

Subacute sclerosing panencephalitis (SSPE) can occur 5–7 years after. Personality change, intellectual decline, deterioration, seizures, death. Rate is 0.5–2 per 100 000 cases

Pregnancy

Maternal mortality – 3 × that of non-pregnant patient. High rate of fetal loss and prematurity

Vaccination
Contraindicated in pregnancy or those with egg allergy
What % of those vaccinated develop a temp of 103°F or higher ? 45%
What day after vaccination does this % usually begin temps? 42 days

Management in pregnancy
Children born to mothers who have measles in last week of gestation or first week postpartum, should be treated with immune globulin 0.25 ml/kg IM

MEDICINES COMMONLY PRESCRIBED

Broad-spectrum antibiotics
Augmentin® 250 mg, 500 mg or 875 mg — One tablet p.o. q. 8–12 h
Amoxil® 250 mg, 500 mg or 875 mg
- ENT — 500 mg q. 12 h or 875 mg q. 12 h
- Lower URI — 875 mg q. 12 h or 500 mg q. 8 h
- Skin infection — 500 mg q. 12 h or 875 mg q. 12 h
- UTI — 500 mg q. 12 h or 875 mg q. 12 h
- GC, acute urethritis — 3 g as single oral dose

Cardiac prophylaxis
Ampicillin 2 g IV @ 30 min to 1 h prior to surgery or labor, then 1 g q. 4–6 h while in labor or 1 dose 6 h postop with gentamicin 1.5 mg/kg IV preop then repeated 8 h later

Genital
Bacterial vaginosis
Metronidazole — 500 mg p.o. b.i.d.
Metrogel® — Apply 5 g hs × 5 nights
Clindamycin 2% cream — 5 g intravaginally × 7 nights

Candidiasis

Agent	*Brand name*	*Dosage*
Butoconazole 2% cream	Femstat®	5 g intravaginally × 3 days
Clotrimazole 1% cream	Gyne-Lotrimin®	5 g intravaginally × 7–14 days
	Mycelex®-7	5 g intravaginally × 7–14 days
Clotrimazole vaginal tablets	Gyne-Lotrimin® vaginal inserts	One 100 mg insert × 7 days
	Mycelex-7 vaginal inserts	One 100 mg insert × 7 days
	Mycelex-G vaginal tablets	One 500 mg tablet
Fluconazole oral tablets	Diflucan® tablets	One 150 mg tablet
Miconazole 2% cream	Monistat® 7	5 g intravaginally for 7 days
Miconazole suppositories	Monistat 7	One 100 mg suppository × 7 days
	Monistat 3	One 200 mg suppository × 3 days
Terconazole 0.4% cream	Terazol® 7	5 g intravaginally × 7 days
Terconazole 0.8% cream	Terazol 3	5 g intravaginally × 3 days
Terconazole suppositories	Terazol 3	One 80 mg suppository × 3 days
Tioconazole 6.5% vaginal	Vagistat®-1	5 g intravaginally once

Trichomonas – metronidazole — 1 g p.o. once

Lower respiratory tract
Levaquin® — 500 mg p.o. daily
Zithromax® — 500 mg p.o., then 250 mg p.o. daily × 4 days

Skin and skin structures
Doxycycline — 100 mg p.o. q. 12 h
Tetracycline — 250 mg and 500 mg tablets (1 g in 2–4 daily divided doses then 125–500 mg daily after improvement)

Traveler's diarrhea
Prevention – TMP 160 mg/SMX 800 mg (Septra® DS) — 1 tab p.o. daily or doxycycline 100 mg p.o. b.i.d. then daily
Treatment – Cipro — 500 mg p.o. b.i.d. and Loperamide two tablets then one tab after each loose stool not > 8/day and HYDRATION

Upper respiratory tract
Amoxicillin — 500 mg p.o. t.i.d. × 3 days for first-line therapy of sinusitis
Ceftin — 250 mg p.o. b.i.d.
Augmentin — 875 mg p.o. b.i.d.
Biaxin® — 500 mg p.o. b.i.d.
Levaquin — 500 mg p.o. q. daily
Vantin® — 100–200 mg p.o. b.i.d.

Urinary tract infections	Septra DS (TMP/SMX DS)	One tablet p.o. b.i.d. × 3 days
	Macrobid® 100 mg	One tablet p.o. b.i.d. × 3 days
	For recurrent cystitis – treat with quinolones	
	Levaquin	500 mg p.o. daily or
	Tequin®	400 mg p.o. daily
Coughs/colds	*Flu*	
	Tamiflu	75 mg 1 capsule b.i.d. for 5 days (Category C)
	Relenza®	Two puffs b.i.d. for 5 days (Category B)
Gastrointestinal	*Constipation*	
	MiraLax® (polyethylene glycol 3350) or (PEG 3350)	
	Ducolax® tablets and/or suppositories	2 tablets and 1 suppository
	Diarrhea	
	Lomotil® or Imodium®	1–2 tablets initially followed by one tablet every 6 h or after each loose stool
	GERD or heartburn – *See* Heartburn of pregnancy	
	Irritable bowel syndrome – *See* Irritable bowel syndrome	
Ophthalmologicals	Neosporin® ophthalmological solution 10 cc	1–2 gtts q. 2–3 h
	Opthaine® (local anesthesia)	b.i.d. to q.i.d.
Otics	Auralgan® otic solution ½ ounce	1 gtts. q. 1–2 h till no pain
	Debrox® 1 ounce	5–10 gtts b.i.d. × 3–4 days
Pain relief	Morphine	8–10 mg IM or 2 mg IV then MS continue 30 or 60 mg tablets p.o. q. 12 h
	(Good for fractures, postop pain, etc.)	
	Ketoralac (Toradol®)	30–60 mg IM then 15–30 mg p.o. q. 6 h
	(Excellent for kidney stones, postop pain, watch for bleeding)	
	Meperidine (Demerol)	25–100 mg IM or IV then Mepergan® fortis p.o. q. 3–4 h
	(Caution with patients > 60 years old)	
	Sublimaze® (fentanyl)	1–2 cc IV or transdermal 1–2 cc/h
	(Very good for short acting, labor or postop outpatient)	
	Oxycodone (Percocet 5, Lorcet Plus, etc.)	One tablet p.o. q. 4 h
	Dilaudid	1, 2, 3 or 4 mg IM, p.o., IV or rectal suppositories
	B & O Suppositories – available in 16-A and 15-A suppositories	
	(The 16-A is the most commonly used and consists of 60 mg of opium and 16 mg of belladonna extract. This works very well to relieve rectal pain such as hemorrhoidectomies, post. repair etc.)	
Sedation/sleep	Ambien®	10 mg p.o. q. hs
	Sonata®	10 mg p.o. q. hs
	Placidyl® (chloral hydrate)	500 mg p.o. hs
	Morphine	15 mg
	Nembutal	100–200 mg
	Scopolamine (rarely used anymore)	gr1/100, second dose 1/200
Stat 'disaster' mix	Isoprel 1 cc	
	Atropine 2 cc (0.8 mg)	
	Neosynephrine 1 cc (10 mg)	
	Put all of the above into 20 cc sterile water → give 5 cc STAT IV for unknown type of cardiac arrest. This is a last resort – otherwise do not attempt using this mixture	
Urinary incontinence	Detrol LA	4 mg p.o. daily or 2 mg p.o. b.i.d.
	Ditropan XL	10 mg p.o. daily or 5 mg p.o. b.i.d.
	These meds and more are for detrusor incontinence. *See* Urinary incontinence	

MEIGS' SYNDROME

Fibroma, ascites and hydrothorax	
Fibroma is a benign, solid, ovarian neoplasm that comprises what % of benign neoplasms?	5%
What % are malignant?	1%

What % are unilateral? 90%
What % of fibromas present with Meigs' syndrome? < 5%
What % have ascites if the tumor is over 6 cm in size? 50%
What % of hydrothorax is found in the right pleural space? 75%
Incidence of ascites is directly proportional to the size of the tumor

Remember, most patients with preop ascites and solid tumor have ovarian cancer!

MELANOMA

Melanoma of the vulva

Of all primary vulvar malignancies, represents 2–4%
Second most common vulva malignancy
Staging: Clark's levels
Chung
Breslow

Histologic types

Superficial spreading melanoma — most common type
Lentigo malignant melanoma — flat freckle
Nodular melanoma – tends to penetrate deeply and metastasize widely. Most aggressive

Diagnosis

Excise or biopsy unless present and unchanged for some years. Take from center of lesion – no evidence that biopsy 'spreads tumor'

Treatment

< 1 mm invasion – radical local excision
> 1 mm invasion – en block resection and regional lymph nodes
Treatment is radical wide excision with how much lateral margins? 2 cm

Prognosis

Poor but behavior is unpredictable
5-year survival rate 20–50%
≤ 1 mm have excellent prognosis but as depth increases so does mortality
Prognosis is so poor for patients with + lymph nodes that there does not appear to be any value in performing lymphadenectomy. Chemo and immunotherapy for vulvar melanoma have been disappointing
Clark's levels (five) – deeper the worse prognosis:
(1) Intraepidermal
(2) Papillary dermis
(3) Fills papillary dermis
(4) Reticular dermis
(5) Enters fat layer

Melanoma of cervix

Overall prognosis is poor. 5-year survival rate usually < 50%
Only 26 published cases of primary cervical melanoma
How many cases of female genital melanoma at Duke? 43
Vulva – vaginal – cervical 30 – 9 – 4

Diagnosis

Biopsy/LEEP
+S-100, HMB 45, melanoma antigen

Treatment

Radical hysterectomy, partial vaginectomy, pelvic and para-aortic lymph node dissection. Radiation. Chemotherapy (Melphalon, interferon, TNF). Immunotherapy (intralesional high-dose interferon)

MENOPAUSE

Definition

Cessation of menses for minimum of 6 months due to inadequate follicular development and waning estrogen production

Osteoporosis

Bone loss in first 5–7 years after menopause is 20%
Hip fractures/year in women 250 000
This % of white women fracture their hips 33%
This % of black women fracture their hips 25%

	This % of white women fracture their spines	25%
	What % of hip fracture patients die in first 3–4 months?	16%
	HRT increases spine BMD in compliant patients by	3.5–5%
	Continuous regimen better than ERT alone or cyclic therapy	
	HRT decreases fracture risk by	50%
	Bisphosphonates increase BMD by	6%
	Decrease fracture risk by	50%
	SERM increased BMD by	1–2%
	Decrease vertebral fracture risk by	40%
	Fluoride increases BMD by	10%
Cardiovascular	ERT/HRT reduce risk of future events and death by	50–90%
	How? Lipid-dependent and -independent mechanisms	
	New data from WHI (Women's Health Initiative) showed that combined estrogen and MPA (medroxyprogesterone acetate) may increase the risks of heart attacks, stroke, breast cancer and blood clots, but still reduce colorectal cancer and hip fractures	
Alzheimer's	Reduction in relative risk for AD in women who used ERT by	40–60%
	How? Stimulates axonal regeneration and production of neurotransmitters (acetylcholine and serotonin), protects neurons from amyloid toxicity and increases cerebral blood flow	
Other benefits of ERT or HRT	Dermatologic, improved sleep patterns, + effect on urinary incontinence, reduced risk of hip fractures and protective against colon cancer	
Risks of HRT	Relative risks (first pregnancy after 30 years to delayed menopause for risk of breast cancer)	1.48–1.36
	Current use of HRT and risk for breast cancer	1.12
	See Hormones and hormone replacement therapy	

MEN (MULTIPLE ENDOCRINE NEOPLASIA)

	MEN I – autosomal dominant (parathyroid, anterior pituitary, pancreatic islet tumors). Chromosome	11q
	MEN II – (medullary thyroid cancer, pheochromocytoma and parathyroid/adenomas). Chromosome	10q
	Medullary thyroid	95%
	Pheochromocytoma	50%
	Parathyroid and adenomas	only 15–30%
	DNA diagnosis at age 6 – prophylactic thyroidectomy	

MENSTRUAL CYCLE

Proliferative (follicular)	Growth. Pseudostratified epithelial nuclei (late)	
Secretory (luteal)	Subnuclear vacuoles around day	16
	Stromal edema seen about day	21
	Spiral arterioles seen about day	23
	Leukocyte infiltration seen by day	27
What causes menstruation?	Sloughing of the endometrium as a result of withdrawal of the requisite hormonal support (estrogen and progesterone)	
	Normal volume	< 80 ml
	Average	30 ml
	Normal interval	28 days
	Normal length	2–7 days

MENSTRUAL MIGRAINE

See Headache

METROMENORRHAGIA

Definition	Blood loss over	80 cc
	or over	7 days
	Normal menstrual blood loss is between	30–35 cc

MIGRAINE HEADACHE

Headache lasting 4–72 h, unilateral, pulsating, N&V, photophobia

Treatment	Somatotropins	
	Example – Imitrex 20 mg NS, 6 mg SC or 25–50 mg p.o.	
	Amerge 2.5 mg p.o. (can be repeated in 4 h). Category C	
	Zomig 2.5 mg p.o.	
Prophylaxis	Propanolol 40–240 mg /day	20 mg t.i.d.
	Atenolol 50–120 mg/day	
	Fluoxetine 10–80 mg/day	
	Amitriptyline 10–25 mg q. daily	
	Verapamil HCl (240–720 mg/day)	80 mg p.o. t.i.d.
	See also Headache	

MISOPROSTOL (CYTOTEC®)

PGE_1 – more stable and less expensive than other PGs

Dosage q. 3 h	25 µg
Dosage associated with increased rate of uterine tachysystole	50 µg

Currently not approved (by FDA) for induction of labor. Administered in tablet form into posterior fornix. It is FDA-approved for use in peptic ulcer disease. ACOG does not recommend for cervical ripening in patients who have had prior C-section or major uterine surgery

MOLLUSCUM CONTAGIOSUM

Pox Virus – 'Volcanos'
Dxn: Wright's or Giemsa stain

Rx: Local injection, evacuate casious material, curette-rx base	85% TCA

MONDOR DISEASE

Superficial thrombophlebitis, acute pain with erythema usually in upper lateral portion of the breast. Diagnosis is made on characteristic linear, tender, erythematous mass. Superficial thrombophlebitis in the thoracoepigastric vein which drains the upper outer quadrant of the breast

MONITORING

	Percent of patients receiving epidurals that demonstrate uterine hypertonia with decels	9–12%
BTB variability (long-term)	Absent	0–2 BPM
	Minimal	3–5 BPM
	Average	6–10 BPM
	Moderate	10–25 BPM
	Marked	> 25 BPM

Average and moderate beat-to-beat variability is seen in normal healthy fetuses
Short-term variability can only be determined by internal monitoring

Ob patients with NO risks	First-stage labor evaluate and record FHR every	30 min
	Second-stage labor evaluate and record FHR every	15 min

Ob patients with increased risks	First-stage labor evaluate and record FHR every	15 min
	Second-stage labor evaluate and record FHR every	5 min
Ob patients with increased risks and continuous monitoring	First-stage labor evaluate and record FHR every	15 min
	Second-stage labor evaluate and record FHR every	5 min
Amnioinfusion	Room temperature, normal saline	
	Bolus	800 ml
	at rate of	10–15 ml/min
	Continue × 1 h at rate of	10 ml/min
	followed by maintenance dose of	3 ml/min
Epidurals	Following the administration of an epidural, one might see decreased beat-to-beat variability, late decelerations or a combination of these	
	Approximately what % of patients receiving epidurals demonstrate uterine hypertonia with resultant decelerations?	9.9–12.5%
FHR at term gestation	Usually ranges from	120–160 BPM
Periodic changes in FHR	Are common in labor. These changes occur in response to contractions or fetal movement and include accelerations and decelerations	
Non-reassuring pattern	Degree to which decels are non-reassuring depends on their depth and duration but most importantly the frequency and progression of recurrence	
Consider fetal scalp electrode	If decelerations are persistent and progressively worsening (those considered non-reassuring)	
Not a substitute for informed clinical judgment	Intrapartum fetal assessment by FHR monitoring is only one parameter of fetal well-being. It involves evaluation of the pattern as well as the rate, but it is not a substitute for informed clinical judgment	
Prolonged deceleration	FHR levels below the baseline lasting	60–90 s
Fetal baroreceptor regulatory mechanism	Cord compression/cord prolapse – activates FBRM causing stimulation of vagal center which is part of parasympathetic nervous system	
Autonomic nervous system	Parasympathetic – decreases heart rate Sympathetic – increases heart rate	
Bradycardia	< 120 BPM lasting 10 min	
Early decelerations	Head compression – stimulation of vagus nerve	
	Uniform – 'upside down contraction' – rarely	< 110 BPM
	Occurs during vaginal exams, pushing, vertex, after ROM, CPD	
Variable decelerations	Cord occlusion, nuchal cord, late labor, compression, prolapse. No consistent shape – may assume any shape. Variable onset and offset depth and duration. Not associated with acidosis unless severe Mild – < 30 s duration or not less than 80 BPM Severe – < 70 BPM and > 60 s duration Atypical – fetal hypoxia, biphasic, decreased variability, continues with lower baseline	
Late decelerations	Uteroplacental insufficiency Usually occurs with: (1) Blood disorders (anemia, SSD, Rh isoimmunization) (2) Bleeding disorders (abruption, placenta previa) (3) Hypertensive disorders (PIH, chronic hypertension) (4) Placental dysfunction (post-term, IUGR, etc.) (5) Disorders of blood vessels (DM, ASHD) (6) Hypotensive (supine hypotensive syndrome, dehydration, anesthesia) (7) Uterine hyperstimulation (Pitocin augmentation or induction) (8) Cardiac disease	

External fetal monitoring

Advantages	*Disadvantages*
Anytime	Difficult to read if obese
Convenient	Sometimes many artifacts
Non-invasive	FHR can be lost if positional change
Minimal training	Patient on back but no other
All time-fetoscope not needed	FHR *variability* NOT available
Frequency of contractions	No info on quality or quantity of contractions
Changes in FHR detected	Baseline tone of uterus cannot be determined
No fetal or maternal comp	

Internal fetal monitoring

Advantages	*Disadvantages*
Allows patient to move	Requires some dilatation and ROM
Accurate measurement of contractions	Requires skills of examiner
FHR variability can be assessed	Uncomfortable application
Reveals baseline tonus of uterine contraction	Requires sterile, disposable equipment
Cultures can be obtained	If fetus low – IUPC difficult to place
No artifacts (unless there is a dead fetus – make sure pulses are different)	Increased maternal/fetal morbidity

MUCINOUS CYSTADENOMA

Huge, bluish-white-gray, translucent usually no significance *bilateral* cysts. Interior – many discrete septa. Serous cyst are bilateral only 10%
Micro – tall epithelium with basal nuclei + goblet cells
Scopes are contraindicated if suspicious for malignancy. Always schedule for possible laparotomy. Treatment depends on age, exam and ultrasound
What % of all ovarian neoplasms? 20%
What % of ovarian cancers? 8%

MUCUS

Poor quality is thick and tenacious. 'Shaking' pattern in non-progressively motile sperm – sperm ABs
Rx of poor mucus includes low-dose estrogen and/or robitussin x 1 week

MÜLLERIAN AGENESIS

What % are associated with urinary tract abnormalities? 50%
See Amenorrhea

MYASTHENIA GRAVIS

What is the incidence in pregnancy? 1/20 000
Deficiency of nicotinic postsynaptic acetylcholine receptor protein at motor end-plate of skeletal muscle usually decreased @ 25%

Symptoms of diplopia, dysphagia and weakness (ptosis and diplopia in most patients). Extremities, diaphragm and neck extensors may be affected. Generalized weakness found in 85%

What % of patients have spontaneous remission during first 2 years? 25%

In pregnancy, 41% have exacerbations, 29% have remissions, 32% have no change

Treatment

Anticholinesterase agents, pyridostigmine (Mestinon®) usually 60 mg every 4–6 h
Neostigmine (15 mg oral dose = 60 mg dose of pyridostigmine)
Timespan is a sustained-release form of pyridostigmine used to get through a night without meds or at party without droopy eyelids or slurred speech
CellCept® 500 mg 2 h after or 1 h prior to meals

Steroids × 2 weeks then decrease dosing. If no improvement after 4 weeks – plasmapheresis
Corticotropin (corticosteroids, azathioprine, cyclosporine) for crisis.
Other treatments include azathioprine (antimetabolite) and plasmapheresis

Must treat any infection (including UTI) because these may predispose to exacerbations. If gravid patient in labor, be ready to perform operative assisted vaginal delivery as patient may tire easily in the second stage. Full respiratory support should be available.
A 0.5-mg IV or 1.5-mg SC dose of neostigmine is equivalent to a 15-mg oral dose of neostigmine or a 60-mg oral dose of pyridostigmine
Vaginal delivery is preferred and C-section should be reserved for Ob indications

Regional anesthesia is the ideal choice for vaginal and C-section delivery. Avoid ester-type local anesthetics (such as tetracaine and chloroprocaine) as the metabolism depends on plasma cholinesterase, which is diminished. If there is significant respiratory compromise or bulbar involvement, general endotracheal anesthesia is recommended as airway management, oxygenation and control of secretions are made easy. Atracurium, mivacurium, propofol and/or sodium thiopental may be used to induce anesthesia
SICU should be considered for postop with frequent AB gases and airway secretion management
MAG SULFATE CONTRAINDICATED
Other drugs that exacerbate MG are aminoglycosides, polymyxins, tetracycline; β-adrenergic drugs; succinylcholine or curare: narcotic analgesics, sedatives, tranquilizers; lithium; quinidine, quinine, quinacrine; tetracaine or chloroprocaine

Neonatal myasthenia gravis (NMG)

A transient condition usually begins 24–72 h after birth
Develops in what % of myasthenic mothers? 10–15%
Most common clinical features of NMG are feeding difficulties and generalized hypotonia

MYOCARDIAL INFARCTION

Anesthesia-associated fatalities account for 10–30%
GREATEST RISK not immediately postop but postop days 3 and 4
EKG – ST changes – creatinine phosphokinase if EKG changes

MYOMAS

Most common pelvic tumor in females. What % reproductive women? 20%
Most affected chromosomes 1, 6, 7, 12 and 14
Most frequent non-random cytogenic abnormality del 7 q21
Monoclonal – contain non-random cytogenic abnormalities in what % of cases? 45%

Histology

Overproduce collagen which causes pale color and firmness
% hyalinized 60%
% with areas of hemorrhage 11%
% with calcium 10%
% that undergo cystic degeneration 4%
Most have < 3 mitoses per 10 HPF

> 5 with cellular AND atypical nuclei per HPF or > 10 per 10 HPF = leiomyosarcomas

By suppression of estrogen and progestogen receptors in premenopausal females, volume decreases by 50%

Depends on myoma size, number and location
Affects – abruption, PTL, C-section, retained placenta, PPH, fetal malposition

This size myoma does not significantly affect pregnancy < 3 cm
This size myoma can cause increase PTL, pain, placental abruption and necessitate C-section > 3 cm
This size myoma increases the risk of obstructed labor > 6 cm

Types

(1) Submucosal
(2) Intramural
(3) Subserosal
See Figure 5 on page 152

Symptoms

Most asymptomatic. Depends on size, number and location
• Abnormal bleeding 33%
• Pelvic pressure or pain 33%
Rectal symptoms. Infertility. Recurrent pregnancy loss. Enlarged midline mass

Diagnosis

Absolute – removal at time of pathology
Presumed – pelvic US, HSG, saline infusion sonography, hysteroscopy and/or MRI

Treatment

(1) Expectant management
(2) Hormone therapy
Advantages – shrinks, decreases EBL, corrects anemia, atrophic endometrium
Disadvantages – delays final diagnosis, expense, bone loss, vaginal hemorrhage in 3%
(3) Surgery
MIVH, TVH, LSH, LAVH, HALS, TSLH, TAH, myomectomy, hysteroscopy (Versapoint 4-mm wire loop), laparoscopy or bilateral oophorectomy?
Key points to hysteroscopic myomectomy include:
(a) The goal of hysteroscopic myomectomy is complete removal of the fibroid without trauma to normal uterine tissue
(b) Patients with Type 0 and Type I fibroids often require only 1 surgery; patients with Type II fibroids should be advised that 2 surgeries may be needed to remove the entire fibroid
(c) Adjuvant preoperative hormonal therapy facilitates surgical scheduling, helps prevent further blood loss in patients already suffering from anemia, and reduces distention media intravasation
(4) Uterine arterial embolization
According to Spies and colleagues (Spies JB, Ascher SA, Roth AR, *et al*. Uterine artery embolization for leiomyomata. *Obstet Gynecol* 2001;98:29–34):
(a) Complaints of menorrhagia improved for more than 80% of 85% of the 200 consecutive patients in the study that bled prior
(b) Uterine volume was reduced by 27% at 3 months and 38% at 12 months following the procedure
(c) The dominant fibroid volume was reduced by 44% at 3 months and 58% at 12 months
Pain remains biggest postop problem especially during the first 24 h is NOT trivial. What is needed is a prospective, randomized trial comparing uterine artery embolization to hysterectomy for treatment of uterine leiomyomas

Hysterectomy vs myomectomy

Hysterectomy –
decreased recurrence

decreased blood loss	
infection/bleeding complications	2%
Myomectomy –	
recurrence	50%
increase in blood loss	
adhesion rate higher	
Hysterectomy is performed in a ratio to myomectomy	10 : 1
The recurrence rate after myomectomy is	50%
Myomectomy adhesions are common (posterior/anterior uterus)	94%/56%

Myomectomy increases risk of uterine rupture
Avoid myomectomy during pregnancy if at all possible due to increased bleeding

NAEGELE'S RULE

First day of last period –	subtract	3 months
	add	7 days

Calculation of EDD

NECROTIZING FASCIITIS

High mortality rate of	12–60%

Rapid progression with vessel thrombosis → tissue necrosis
Bacteria – staphylococci, streptococci, *Clostridium*, hemolytic streptococci
Predisposing conditions – diabetes, immune suppression, radiation, fever, tach, devascularized skin, hem bullous inflammation + edema

Evaluate first 24 h every	3 h

Treatment

Aggressive excision, debridement, pack wounds, do not close, antibiotics
Circulation and tissue oxygenation – hyperbaric O_2 p.r.n.

NEEDLESTICK

Injury rate in gyn surgery	2–10%
Chance of HIV conversion after HOLLOW needle (not solid)	0.3–0.4%

Double gloving decreases incidence. Prophylaxis with ZDV is optional

Check HIV status baseline then	6 weeks, 3 months and 6 months

NEONATAL LUPUS ERYTHEMATOSIS

Skin lesions seen	50%
Congenital heart block	1/20 000 births
Skin lesions and heart block	10%

Liver disease and thrombocytopenia
Heart block and skin lesions associated with Sjögren's syndrome

A antibodies	anti-SSA or Ro
and syndrome B antibodies	anti-SSBs or La

Affect cardiac system – complete heart block – external pacing

needed for	AV node
Some need pacing also for	SA node

NERVE INJURIES

Femoral

Too much flexion on retractor or flexion of thighs too far back against abdomen at TVH or increased risk of injury in thin patient with low Pfannenstiel incision using self-retaining retractors by lateral blades during TAH
Symptoms – difficulty climbing stairs, inability to flex thigh, decrease of sensation on the inner thigh, anterior hip pain, decrease knee jerk. Unable to lift or support foot

	Best prevention – placing a lap pad between the abdominal wall and the retractor *Femoral nerve innervates* – quadriceps, sartorius, iliacus, pectineus *Most effective therapy* – tincture of time (1–24 weeks functional)
Lateral femoral cutaneous	Too much retractor pressure No motor weakness – lateral aspect of thigh is numb
Genitofemoral nerve	Found on belly of psoas muscle. Sensory to anterior vulva and anterior thigh
Obturator nerve	Loss of adduction. Numbness over medial aspect of thigh but no muscle weakness
Sciatic nerve	If knees too far out to sides in lithotomy position Leg numbness, difficulty with walking, decreased sensation to posterior and lateral surfaces of leg and foot, inability to dorsiflex the foot
Peroneal nerve	Damage during normal SVD in lithotomy. Causes footdrop
Pudendal	Injury most likely during SSLF. Numbness to vulva. Loss of urinary or fecal continence

NEURAL TUBE DEFECTS

Types	Spina bifida Anencephaly Encephalocele	
	Meningocele – opening in lumbosacral vertebrae Meningomyelocele – meningocele that contains neural element Meningoencephalocele – defect in skull in which part of brain protrudes into sac Anencephaly – failure of closure of cranial end of neural tube Encephalocele – extrusion of brain tissue through skull defect, generally covered by overlying skin	
Etiology	Multifactorial but there is an abnormal gene that is a variation of the gene that produces the enzyme, 5,10-methylenetetrahydrofolate reductase that is critical for folate use	
	Folic acid decreases incidence by	50–70%
	No identifiable risks noted in what % of patients?	90%
	Neural tube normally closes	3rd + 4th week
	Second most common fetal malformation behind heart defects	
	Most NTDs are multifactorial	85%
	Incidence in the USA	1/1000
	Hydrocephalus is present in what % of NTDs?	80%
	Increased AFP and AChE (acetylcholinesterase) suggest open fetal defect	99%
	Increased AFP and normal AchE suggest fetal defect other than NTD. AchE is found in blood cells, muscles, nerves	
	Increased MSAFP and normal US are at risk for LBW, fetal death and oligohydramnios	
	MSAFP detects what % of open NTDs?	80%
	MSAFP detects what % of all open NTDs?	80–85%
	MSAFP is elevated in what % of black females?	10%
	MSAFP is elevated in what % of insulin-dependent DM?	15%
	MSAFP doubles in twins. Most common reason for false-positive or elevated MSAFP is underestimation of gestational age	
Ultrasound findings	BPDs smaller with spina bifida, scalloping of frontal bones, banana sign, lemon sign, downward displacement of cerebellum, varying degrees of ventriculomegaly	
	US will have some type of cranial anomaly (‘lemon sign’ – frontal notching or ‘banana sign’ – cerebellar changes, small BPD, ventriculomegaly, ‘Arnold–Chiari malformation’ – obliteration of cisterna magna) in what % of cases of NTD?	99%

	Risk of chromosomal anomaly in pt < 35 with decreased MSAFP and normal US	< 1%
	Risk of NTD if affected parent	5%
	Risk of NTD if one previously affected child	2%
	Risk of NTD if two previously affected children	6%
	Risk of NTD if positive family history	1%
Diagnosis	MSAFP, ultrasound, amniocentesis, targeted ultrasound and amnio	
Prevention	0.4% folic acid daily 3 months prior through first trimester which reduces incidence by	> 70%
	How much folic acid is given to a patient with a previously affected child or patient with epilepsy?	4 mg
	What is the daily folic acid requirement or recommendation for twin pregnancy?	1 mg
Treatment	Open spina bifida – atraumatic, unlabored C-section with intact membranes	
	What % of patients with NTDs have no identifiable risks?	90%
	Parents with previously born child with NTD have what chance to deliver a subsequent child?	10 ×
	The recurrence rate is	1–5%

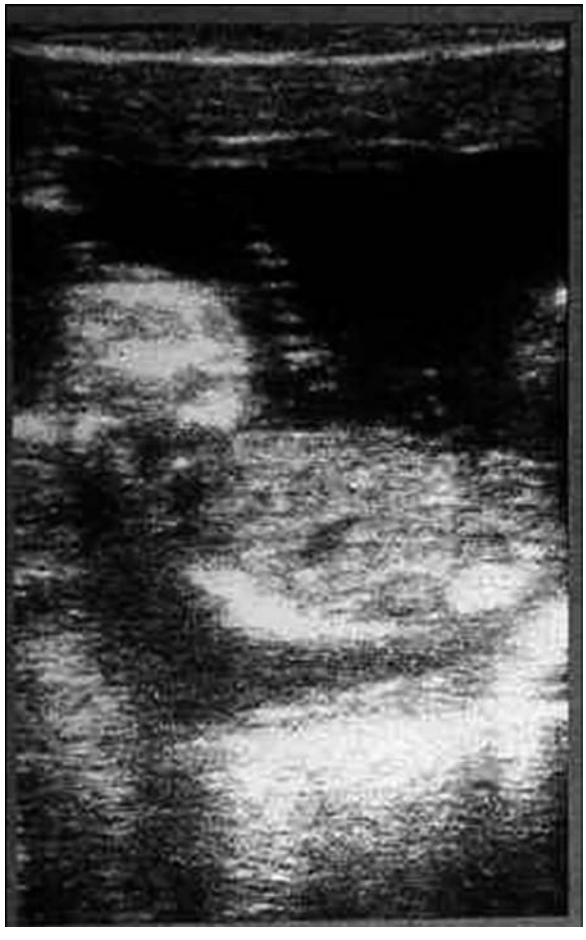


Figure 17 Ultrasound of an anencephalic fetus

NEUROLOGICAL (NEONATAL) SEQUELAE

Evidence includes – seizures, coma and hypotonia AND one or > of the following:

Cardiovascular
Gastrointestinal
Hematologic
Pulmonary
Renal system dysfunction

NEURO-OBSTETRICS

	Early labor	T11 and T12
	Late stage of labor	T10, T11, T12, L1
Pseudotumor cerebri	Increase or decrease in CSF. Increased risk to obese pts and pts who have recently gained weight. Symptoms: H.A. diplopia, visual disturbances, papilledema. First trimester or first month postpartum	
	Most common symptom is headache	95%
	Then diplopia with blurred vision	75%
	Commonly found in obese or those who recently gained weight	
	Complicates pregnancy	1/1000
	This is the recurrence rate	30%
	Rx: Repetitive lumbar punctures to PREVENT BLINDNESS. Also acetazolamide, furosemide and dexamethasone	
Carpal tunnel syndrome	Experienced by this % of pregnant women	25%
	Bilateral	80%
	Treat with splint p.r.n. Patients require surgery only	10%
Spinal cord injuries	Autonomic hyperreflexia	T5–6
	Cough reflex impaired – check pulmonary function	T10
	Painless labor with injury above	T12
Chorea gravidarum	Occurs in association with rheumatic fever what % of time?	2/3
	Recurrence common, mostly primagravidas	
	This % of lupus patients demonstrate chorea	2%
Bell's palsy		
	Isolated facial nerve palsy involving cranial nerve	7
	Symptoms:	
	(1) Acute onset of pain in ear	
	(2) R- or L-sided facial pain or tightness	
	(3) Inability to close eye	
	(4) Metallic taste in mouth	
	How much more common in pregnancy?	3 ×
	Occurs in third trimester	75%
	Occurs in first and second trimester	15%
	Occurs postpartum	10%
	Etiology – exposure to cold, fluid retention, hormone changes or hypercoagulability	
	Treatment – symptomatic to provide prevention of corneal abrasion, as the eyelid does not close. The use of steroids is controversial.	
	Most cases resolve spontaneously	
	Prognosis – good. Usually spontaneously resolves within weeks to months	90%

NIPPLE DISCHARGE

Milky galactorrhea	Physiologic, breastfeeding, pregnancy, postpartum, prolactin excess or pituitary adenomas
	Multicolored, sticky, green-yellow, serous – ductal ectasia
	Purulent, infected – bacterial infection
	Clear, watery – ductal carcinoma
	Yellow, serous – fibrocystic disease
	Pink, serosanguinous – fibrocystic disease or ductal papilloma
	Bloody, sanguinous – fibrocystic disease or ductal papilloma
	See also Breast

NORPLANT®

What % USA women use these?	1%
Six capsules are used each containing how much levonorgestrel?	36 mg
The total dose of levonorgestrel is	216 mg
It provides contraception for how many years?	5
Subtherapeutic doses after removal within how many days?	3
Ovulation resumes after removal within	2–4 weeks

	Pregnancy rate for first year is	0.09%
	Five-year cumulative pregnancy rate is	1.1%
	The failure rate increases to what % in patients taking phenytoin, phenobarb, carbamazepine?	20%
Mechanism of action	Initially ovulation is suppressed in what % (then over time ovulation resumes) but cervical mucus is thick (impenetrable) then endometrial atrophy	80%
Release rate	Per day for first 9 months	85 µg
	Per day for next 18 months	50 µg
	Per day for 60 months	35 µg
Side-effects	Spotting, irregular bleeding or both	50%
	Amenorrhea or oligomenorrhea	20%
	Regular withdrawal bleeding but decreased flow	25%
	Other possible side-effects include headache (r/o papilledema), weight change, acne, mood change, vaginal dryness, change in libido, dyspareunia, mastalgia, risk of ectopic	
Contraindications to long-acting progestins	Active thrombophlebitis or thromboembolic disorders, unexplained abnormal genital bleeding, pregnancy, active liver disease, breast malignancy, allergy	
	Norplant (in particular) – patient with idiopathic intracranial hypertension (r/o headache)	
	DMPA (in particular) – patient with cardiovascular accidents	
	Relative contraindications to all hormonal methods – use of rifampicin and griseofulvin	
Other implants	Implanon	A single rod implant
	Approved by FDA for a duration of	3 years
	Documented duration of use	16 years

NUTRITION IN PREGNANCY

	Recommended carbohydrates	60%
	Recommended protein	20%
	Recommended fat	20%
Food sources for necessary vitamins in pregnancys	Vitamin A – dark yellow vegetables, milk	
	Vitamin C – strawberries, broccoli, tomatoes	
	Vitamin D – fortified milk, fish liver oil	
	Vitamin E – vegetable oils, wheat germ	
	Folic acid – orange juice, liver, legumes, nuts	
Recommend omega-3	Children of mothers who had taken cod liver oil during pregnancy scored higher on the mental processing sections of the K-ABC than did children whose mothers were in the corn oil group. Intelligence test scores and visual acuity/functioning are improved with omega-3 fatty acids. Therefore, studies prompt providers to recommend omega-3 supplementation during both the latter half of pregnancy and breast-feeding. Consumption of refined fish oil and "safe" oily fish is similarly associated with increased DHA levels during pregnancy and breast-feeding	
Calcium helps prevent PIH	New studies confirm that calcium consumption of @ 1500 mg daily during pregnancy: (1) can reduce risks of hypertensive complications including preeclampsia in women with low calcium intake and (2) may reduce the risks of young mothers giving birth prematurely. Milk	
Calcium to prevent PTB	provides about 70% of the calcium for most Americans, but other sources include fortified orange juice, cereals, tofu, soy products, green leafy vegetables, as well as fish (especially sardines and salmon). Calcium tablets are also available	
Folate to prevent NTDs	Folate supplementation in pill form needs to be started preferably before conception to be marginally effective. It takes 3 months to achieve steady state folate levels using vitamin supplementation. Spinal cord completes fusing at 8–9 weeks' gestation, so starting folic acid at the first prenatal visit will not reliably prevent NTDs	

Mercury and seafood warning	Pregnant women are advised to avoid the most contaminated species (tilefish, swordfish, king mackerel, and shark) and to limit the consumption of other fish to no more than 12 oz/wk of species with low mercury concentration and 6 oz/wk if the mercury content in a species is not known. Mercury may cause neurological problems in developing fetus	

OBESITY

	Defined as BMI equal to or over	30
	BMI = Wt (kg) / Ht (m^2)	
	Overweight	25.1–29.9 kg/m^2
	Ideal	19–25 kg/m^2
Incidence	Women overweight (BMI 25–29.9)	33%
	Women obese (BMI ≥ 30)	16%
	Of all adult Americans, those who are overweight or obese	67%
Risks	1-point increment in BMI increases risk of heart failure by	7%
	Being overweight in women, increases risk of heart failure by	50%
	Obesity increases risk of heart failure by	90%
	Overweight and obesity is associated with increased diabetes, hypertension, coronary heart disease and left ventricular hypertrophy	
Treatment	Diet and exercise	
	Impact of exercise is less in women compared to men by how much?	30–40%
	Why? Because women have a lower resting metabolic rate due to:	
	(1) Smaller surface area	
	(2) Smaller body mass	
	(3) Greater % of body fat	
	Xenical® 120 mg – blocks what % of fat reabsorption?	30%
DO NOT USE	Dexfenfluramine (Redux) and fenfluramine (Pondimin) – FDA withdrew from market	
	Fen-phen (fenfluramine + phenteramine) – FDA never approved	
	Caused valvular heart disease and pulmonary hypertension	
	Phenteramine (alone) – remains available, not associated with serious side-effects	
	Effexor® XL (SSNRI) 75 mg p.o. daily for depression. Potent inhibitor of serotonin and norepinephrine on postsynaptic receptor sites (blocks reuptake)	
Surgical considerations	Adding a closed drain did not improve outcome beyond that achieved by subcutaneous closure. In obese women having a C-section, closure of the subcutaneous layer reduces risk of wound complications such as seroma, hematoma, incisional abscess, and fascial dehiscence. Drains should not be used in high risk women having cesarean delivery	

OBSTRUCTION (BOWEL)

	Adhesions are the most common cause of obstruction: SBO	80%
	Colon	20%
	Previous Gyn surgery is most common cause of SBO in women – after benign surgery	2/1000
	After radical surgery what % develop obstruction?	8%
Symptoms	Intermittent pain mixed with pain-free intervals. Periods of intense cramping. Borborygmi – high pitched metallic sound. Usually presents between 5th and 7th day postop. Vomiting with abdominal distension. Profuse NG drainage	
Flat plate	Air fluid levels like 'stepladder'. Gas proximal to obstruction	
Treatment	Expectant therapy is successful in	60%
	Decompress with NG or Miller-Abbott tube. IVFs, serial WBCs and X-rays	
	Surgery p.r.n.	

Major cause of morbidity and mortality

Delay in diagnosis causing peritoneal irritation, fever, increased WBCs, increased sepsis and increased distention

Ileus vs obstruction

Know the difference!

Adynamic ileus	*Bowel obstruction*
Small and large bowel distended in *proportion* to each other	Small bowel obstruction with dilated small bowel *proximal to site* of the obstruction
Gas scattered throughout the GI tract	*Air fluid levels are common,* at different levels in the bowel with a *"stepladder"* appearance
Air fluid levels in small bowel are rare, but if present are at the same levels	

OCCUPATIONAL HAZARDS TO PREGNANCY

Stressors during pregnancy

(1) Standing more than 3 h – increase in prematurity; no effect on birth weight
(2) Lifting more than 12 kg – no studies show any effect on birth weight or PTL
(3) Strenuous work – most studies show no effect on birth weight or PTL

Physical agents

(1) *Heat*
≥ 38.9°C Increases the rate of spontaneous abortions or birth defects (mostly neural tube)
Women with early hyperthermic episodes – counseled and AFP + US studies
(2) *Radiation*
Preimplantation "All or None" phenomenon
Greatest effect during late first and early second trimester
< 5 rads – no intervention recommended
> 5 rads – counsel; offer sonogram screen for microcephaly
(3) *Video display terminals*
No known effect. Increased CTS – place keypad
(4) *Chemicals*
See chart below regarding "Developmentally toxic exposures in humans". If necessary, contact CDC in Atlanta, GA (404) 639-3311
(5) *Hairstylists*
Minimize by use of gloves. Dermatitis. Mutagenic but not teratogenic. Minimize exposure in first trimester
(6) *Painters/artists*
Lead salts are of concern associated with increased spontaneous abortions, infant cognitive impairment, stillbirth rates in humans, CNS abnormalities. Women at risk should be monitored prior to conception. Lead concentration >10 mg/ml – remove from exposure and consider chelation before pregnancy. No consensus how to manage after pregnancy (increased lead from bone stores and the chelating agent calcium edetate may be developmentally toxic, probably decreased zinc stores)
(7) *Solvent workers*
Ethylene glycol, toluene or gasoline, etc. similar to EtOH syndrome
An excess of MR, hypotonia, microcephaly
(8) *Pesticide workers*
Carbaryl and pentachlorophenol. Animal studies demonstrate impaired reproductive success or cause skeletal and body wall defects

If ethylene glycol, toluene, gasoline, carbaryl or pentachlorophenol are suspected, blood or urine levels along with liver function tests can be obtained and, if abnormal, increased fetal monitoring of fetal development is recommended

Developmentally toxic exposures in humans

Aminopterin	Lead
Androgens	Lithium
Angiotensin-converting	Methimazole
enzyme inhibitors	Methyl mercury
Carbamazepine	Parvovirus B19
Cigarette smoking	Penicillamine
Cocaine	Phenytoin
Coumarin anticoagulants	Radioiodine
Cytomegalovirus	Rubella
Diethylstilbestrol	Syphilis
Ethanol (≥1 drink/day)	Tetracycline
Etretinate	Thalidomide
Hyperthermia	Toxoplasmosis
Iodides	Trimethadione
Ionizing radiation (>10 rads)	Valproic acid
Isotretinoin	Varicella

OLIGOHYDRAMNIOS

Oligohydramnios is defined as an AFI of ≤ 5 cm
Dysmaturity syndrome – post-term gestational assessment with thick meconium, deep decels
AFI marginal = 13 × inc perinatal mortality 57/1000
Severe oligohydramnios = 47 × increases perinatal mortality 188/1000
Second-trimester oligohydramnios 43%
W/ lethal pulmonary hypoplasia 33%
Anhydramnios (no fluid) 88% lethal outcomes
Severe, long-standing oligohydramnios inhibits lung growth and promotes limb defects (club foot, arm contractures)

Principal diagnosis with oligohydramnios

(1) PROM
(2) Placental insufficiency
  (a) Chronic abruption
  (b) Maternal hypertension
  (c) Placental crowding in multiple gestation
  (d) Autoimmune disease (lupus, antiphospholipid syndrome)
(3) Urinary tract anomaly
  (a) Polycystic or multicystic dysplastic kidneys
  (b) Renal agenesis
  (c) Ureteral or urethral obstruction
(1) Try to r/o ROM
(2) US fetal renal systems – do amnio if cystic kidneys and renal pelvic condition (assess with trisomy 21 + 18)
(3) R/o IUGR – abd circ legs behind head
  High vas resistance or uterine Doppler studies corroborate oligo due to placental insufficiency
  Hospitalize if diagnosed
  26–32 weeks – amnio – mature? – deliver
(4) Consider pulmonary hypoplasia (lung area ratio should be > 66%)

Diagnostic adjuncts

Amnioinfusion – infection
Dye infusion to r/o membranes
Furosemide test to visualize fetal bladder

Management

Continual antepartum testing
Inc rates of meconium; fetal distress and C-section
Intrapartum amnioinfusion – improved but over-distended uterus
Maternal hydration – effective
Amniotic fluid volume normally diminishes *after 35 weeks'* gestation
Post-term patients are *5 times* more likely to develop oligohydramnios in 3–4 days after a normal AFI, as compared to term patients
Therefore, post-term patients should have *semi*-weekly amniotic fluid volume assessment, with pockets < 3 cm being considered normal

ONCOLOGY

Cervix	CIS	0
	Confined to cervix	I
	Microscopic	IA
	No deeper than 3 mm, no wider than 7 mm (CKC or hyst ok)	IA1
	3–5 mm depth or ≤ 5 mm depth ≤ 7 mm horizontal (radical hysterectomy or radiation)	IA2
	Lesion > IA2	IB
	No larger than 4 cm	IB1
	Larger than 4 cm	IB2
	Upper vagina but not lower 1/3	II
	No parametrial involvement	IIA
	Parametrial involved	IIB
	Lower 1/3 of vagina	III
	No extension to pelvic wall	IIIA
	Extension to pelvic wall or/and hydronephrosis	IIIB
	Beyond true pelvis or mucosa of bladder or rectum	IV
	Spread to adjacent organs	IVA
	Distant spread	IVB
Cervical lymph nodes	Parametrial, paracervical (ureteral), obturator, hypogastric, external iliac, sacral nodes	
	Distant secondary group: common iliac, inguinal, para-aortic	
	Nodes are positive in Stage I	20%
	Nodes are positive in Stage II	40%
	Nodes are positive in Stage III	50%
	Stage I have + para-aortic nodes	6%
Cervical nerve supply	Includes sympathetics merging at Frankenhauser's plexus and S2, S3, S4	
	Treatment	
	IA1 – CKC or simple hysterectomy is okay	
	IA2 – IIA Radical hysterectomy with bilateral pelvic and periaortic lymphadenectomy. Radical hyst includes supporting ligaments of uterus and upper 25% of the vagina. Lymph node dissection includes ureteral, obturator, hypogastrics and iliacs	
	IIB–IVB radiation	4000 WP 6000/h brachytherapy
	Chemotherapy	Cisplatin
Fallopian tube	Similar to ovarian staging	
Ovary	Limited to ovaries	I
	One ovary	IA
	Two ovaries	IB
	One or two ovaries but with ascites, ruptured capsule and/or tumor on external surfaces	IC
	Pelvic extension	II
	To uterus and/or tubes	IIA
	To other pelvic structures	IIB
	IIA or IIB with ascites, ruptured capsule or tumor on external surfaces	IIC
	Positive nodes and/or implants outside pelvis	III
	Negative nodes but microseeding	IIIA
	Negative nodes but with seeding < 2 cm	IIIB
	Positive nodes and/or seeds > 2 cm or retroperitoneal /ing nodes	IIIC
	Distant metastasis	IV
	Diagnosis of ovarian cancer remains elusive. CA-125 and/or transvaginal ultrasound found a large number of false-positive women; therefore these modes are not recommended for routine screening but may be helpful during regular screening in women with strong family histories of ovarian or breast cancers. BE AWARE of complaints of abdominal pain and swelling that may mimic digestive problems. (Ova Check is a simple blood test that is easy to perform and highly effective in identifying women with ovarian cancer, but is not yet approved by FDA and some scientists question the design and results of the original studies.) Basically THERE IS NO RECOMMENDED TEST FOR DIAGNOSIS OF OVARIAN CANCER	

There are, however, four serum protein markers that Mor and colleagues at Yale University identified that, when used together, achieved a sensitivity, specificity, and positive predictive value of 95%, with a negative predictive value of 94%. The markers are leptin, prolactin, osteopontin, and insulin-like growth factor-II. They successfully detected 23 of 24 patients with stage I and II disease. These markers, however, have not yet met the stringent requirements for population-based screening

Epithelial tumors 80–85%

Serous (CA-125, psammoma bodies, ciliated tubal epithelium)
Malignant 20%
Mucinous (CEA + 40%, *Pseudomyxoma peritonei*, colum) 15%
Endometrioid (CA-125, pseudoxanthoma cells, glands). Malignant 95%
Clear cell (CA-125, Hobnail cells, mesonephric tissue) 98%
Brenner's (Walthard cell rests, transitional epithelium) 2%

Germ cell tumors 10–15%

Teratoma mature (Rokitansky prominence)
Most common neoplastic ovarian lesion of female reproductive age
Bilateral 25%
Strumo ovarii (monodermal teratoma) % teratomas 2–3%
What % strumo ovarii develop thyrotoxicosis? < 5%
Strumo carcinoid – rare, usually unilateral
Malignant transformation rare with mature teratoma (usually squamous)
Most frequent complication is TORSION occurring what % 16%
If torsion is recent – untwist and perform cystectomy
Teratoma immature (AFP, CA-125). Malignant 100%
Neural rosette is used to grade these tumors
Dysgerminoma ("bacon & eggs" tumor, LDH, radiation sensitive, fibrous septae + lymphocytes, most common malignant ovarian tumor in pregnancy, "polka-dots"). Malignant 100%
Gonadoblastoma (CALCIFICATION is extensive, frequent germ cells with pale cytoplasm). Malignant only if dysgerminoma elements are present ?
Endodermal sinus tumor (AFP; Schiller–Duval bodies, which are blood vessels surrounded by tumor cells within a space surrounded by more tumor cells). Malignant 100%
Embryonal tumor (AFP AND hCG, syncytiotrophoblastic cells) 100%
Non-gestational choriocarcinoma (hCG)

Germ cell tumor markers

Neoplasm	*Marker*				
	AFP	*CA-125*	*hCG*	*LDH*	*CEA*
Endodermal sinus tumor	Increased	Usually up	Maybe up	Usually up	Maybe up
Immature teratoma	Maybe up	Maybe up	Maybe up	Maybe up	Maybe up
Dysgerminoma	0	Rarely up	Rarely up	Usually up	0
Choriocarcinoma	0	0	Increased	0	0

Treatment of germ cell tumors

VAC (vincristine, actinomycin D, cyclophosphamide)
VBP (vinblastine, bleomycin, cisplatin)
Stage IA1 germ cell tumors are cured by surgery alone 100%
Stage II, III, IV endodermal and embryonal cell tumors = bleomycin, etoposide + cisplatin × 3 doses
Percent recur if no postop Rx is given = 85%
Bleomycin, etoposide and cisplatin cure what % of cases? 95%

Treatment of germ cell tumor in teenager

(1) Most are unilateral (except dysgerminoma = @ 10–15% bilateral)
(2) 85% patients with endodermal sinus (yolk sac) will die (even stage I) IF NO postop treatment is given so GIVE 3 cycles of bleomycin, etoposide and cisplatin. This will cure > 95%
(3) Toxicities

Bleomycin – pulmonary fibrosis, skin hyperpigmentation
Cisplatin – ototoxicity, neuro and nephrotoxicity, Raynaud's phenomenon and ischemic heart disease

Gonadal–stromal tumors		3–5%
	Granulosa cell tumor (Call–Exner bodies – degenerative spaces filled with eosinophilic and cellular debris, inhibin, estrogen production – associated with acute hemorrhage, incomplete precocious puberty, 'coffee beans' – nuclear grooves). Malignant	< 5%
	Unilateral	95%
	Stage I at diagnosis	90%
	Chemotherapy: actinomycin D, 5-FU, cyclophosphamide	
	Fibrothecoma (seen with MEIGS' SYNDROME – ovarian fibroma, ascites, hydrothorax usually on right but rare in < 5% fibroma "Nats" – looks like fibroid, vacuolated spindle cells). Malignant	< 5%
	Meigs' – associated with ascites directly proportional to size of tumor > 6 cm	50%
	Usually unilateral tumor	90%
	Sxs: pressure and abdominal enlargement. Rx: remove tumor. Thecoma element can produce estrogen. Fat stain shows abundant lipid material	
	Sertoli–Leydig cell tumor (crystals of Reinke) Testosterone production can cause heterosexual precocious puberty. (Androblastomas, arrhenoblastomas. Tubular pattern micro). Cystic + hem degen. Ca^{+} can be present. Malignant	< 5%
	Chemo Rx; VAC (vincristine, actinomycin D, cyclophosphamide)	
	Lipid cell tumor (testosterone production). Malignant	30%
	Gynandroblastoma (testosterone production). Malignant	100%
Metastatic tumors	Krukenberg tumor SIGNET-RING CELLS, these tumors usually originate from GI tract or breast most often, bilaterality is a clue that tumor may be metastatic. Glary appearance due to mucin	
	Small cell carcinoma of the ovary – associated with hypercalcemia	
Borderline ovarian tumor	(Low malignant potential) (1) Epithelial proliferation but no evidence of stromal invasion (2) Extraovarian implants present in 30% of patients! (3) 1/3 patients with Stage I or II ovarian cancer will have more advanced, so STAGE (4) Less than 10% with lymphatic mets have enlarged nodes (5) Treat conservatively: (a) Omentectomy (b) Peritoneal biopsies (c) Selected pelvic and para-aortic lymph node biopsies	
Molecular targeted therapy	Oregovomab (OvaRex) targets CA-125 in ovarian cancers. This is a monoclonal antibody-based treatment. (CA-125 is expressed on the surface of more than 80% of epithelial ovarian cancers)	
"Second-look" lap for ovarian carcinoma	Advantages: (1) 50% patients after chemo will have advanced disease at second-look surgery (2) Opportunity to resect (controversial). Theoretically – reduces residual tumor Disadvantages: (1) Major surgery (most common complication – prolonged ileus) (2) Investigational procedure	
Uterus	Confined to uterus	I
	Endometrium	IA
	< 1/2 myometrium	IB
	> 1/2 myometrium	IC
	Spread to cervix	II
	Endocervix glandular involvement only	IIA
	Cervical stromal invasion	IIB
	Invades serosa and/or adnexa and/or + peritoneal cytology	IIIA
	Vaginal metastasis	IIIB
	Lymph nodes (mets in pelvic or para-aortics)	IIIC
	Bladder and/or bowel mucosa	IVA
	Distant metastasis (including intra-abdominal and/or ing nodes)	IVB
	Treatment of grade I endometrial carcinoma is TAHBSO with cytology (cytology is + what % ABC)	15%

	Treatment of grade II endometrial carcinoma is TAHBSO with lymphadenopathy	
	Add radiation therapy according to grade and depth of invasion	
	Invasive cancer – treatment is TAHBSO nodes and radiation	
	Upper vagina	6000
	Whole pelvis	5000
	Risk of nodal involvement if invasion < ½ is	< 5%
	Risk of nodal Involvement if invasion > ½ is	25%
	Blood supply to uterus is from hypogastric to uterine artery. Ovarian artery also supplies some. Uterine vein empties into internal iliac vein. Collateral circulation to pelvis after hypogastric ligation is by lat and medial circumflex femoral artery and middle sacral artery.	
	Diagnosis is highly suspicious if patient is having abnormal bleeding, especially if obese, hypertensive, diabetic and/or has thickened endometrial stripe per transvaginal ultrasound. Confirmation is made with endometrial biopsy and/or D&C	
Lymphatics	Lower uterine segment and cervix drain to iliacs and hypogastrics	
	Corpus drains to internal iliacs, hypogastrics, ovarian and para-aortics	
	Nerve innervation to uterus is hypogastric plexus by sympathetics merging at Frankenhauser's complex	
	Uterus (sympathetics of)	T11–T12
	Cervix and upper vagina	S2–S4
	Perineum	Pudendal
	Endometrial biopsy is the preferred method of diagnosis of endometrial cancer	
Stromal tumors of uteri	*See* Stromal sarcoma	
Vagina	CIS	0
	Treatment is surgery (laser vap or radiotherapy)	
	Vaginal wall	I
	Treatment is radical surgery. (Hyst, vaginectomy and pelvic lymph)	
	Subvaginal	II
	Extension to wall (including pubic bone)	III
	Treatment of II and III is radiotherapy	
	Beyond pelvis	IV
	Bladder and rectum	IVA
	Distant metastasis	IVB
	Treatment is exenteration with Ext 5000 rads to whole pelvis and interstitial implants	
	Blood supply is from internal iliac artery to vaginal artery with the following branches:	
	Cervicovaginal branch of uterine to	upper 1/3
	Inferior vesical arteries to	middle 1/3
	Middle rectal and internal pudendal to	lower 1/3
	Lymphatics from vagina:	
	Iliacs, obturators drain	upper 1/3
	Internal iliacs (hypogastrics) drain	middle 1/3
	Inguinal (femoral) drain	lower 1/3
	Nerves to vagina	
	Pudendal (more sensitive)	lower 1/3
	Work-up for vaginal cancer includes CXR, IVP, cystoscopy and proctoscopy	
	VAIN – mostly upper 1/3 and multifocal (treatment is local excision, 5-FU or laser) to what size?	1.5 mm
	Adenosis – Mat DES prior to 18th week of gestation – treat with laser or	5-FU
Vulva	CIS	0
	Vulva or perineum < 2 cm	I
	Vulva or perineum > 2 cm	II
	Spread to urethra, vagina and/or anus	
	Unilateral regional lymph node spread	III

Urethra, bladder mucosa, rectal mucosa, pelvic bone and/or bilateral nodal metastasis IVA
Distant metastasis including pelvic lymph nodes IVB
Blood supply is from pudendal artery. Internal pudendal artery to perineum. Inferior rectal and posterior labial arteries are branches
Lymphatics
Inguinal (femoral or sentinel nodes)
If lesion is < 2 cm the % + nodes is 15%
If lesion is > 2 cm the % + nodes is 38%

Where is Cloquet's node located?
Answer: In the femoral triangle medial to the femoral vein
What are the borders of the femoral triangle?
Answer: inguinal ligament, pectineus m and iliopsoas m
Nerves to vulva
Pudendal nerve mediates along S2, S3, S4
Complex arrangement of Meissner's corpuscles most dense at clitoris

Where does the femoral nerve lie in relationship to the femoral triangle?
Answer: Outside the triangle. The artery and vein lie inside it NAV

ORAL CONTRACEPTIVE PILLS

What % pts switched brands due to BTB? 33%
What increased % for BTB is found in smokers taking OCPs? 47%
Estimated OC dose that eliminated excess risk of MI 3–35 μg
Pregnant women have what chance of thromboembolism? 10/1 million
OCPs protect against
(1) Benign breast disease
(2) Fe^{+} deficiency anemia
(3) Ovarian cysts
Treatment for osteopenia in reproductive females – OCPs and calcium
Treatment for acne – triphasic norgestimate and ethinylestradiol
Treatment to decrease menstrual blood loss, duration of menstruation and dysmenorrhea – 30 μg ethinylestradiol
OCPs and symptomatology and management
(1) If patient with acne on OCs – decrease progestin
(2) Hyperplasia or bleeding – increase progestin
(3) Severe acne – CPA 2 mg (Diane® 35)
(4) Chloasma – decrease estrogen (but avoid BTB) and avoid UV light
(5) Mood swings – progestin-only injectables
(6) Early-cycle bleeding – increase estrogen
(7) Amenorrhea – increase estrogen

See Contraception

ORTHOSTATIC INTOLERANCE

(1) Most common disorder of B/P regulation after essential hypertension
(2) Characterized by orthostatic tachycardia (> 30 BPM increase heart rate on standing), is also frequently characterized by light-headedness, dizziness, palpitations, exercise intolerance, near-syncope, occasionally syncope and orthostatic tachycardia, but unusually with sustained orthostatic hypotension
(3) May also be associated with MVP, chronic fatigue syndrome, primary hypovolemia, lower body venous pooling, decreased plasma volumes, prolonged weightlessness or inappropriate sinus tachycardia

OSTEOPOROSIS

Associated with accelerated bone loss in postmenopausal women primarily from trabecular bone Type I

Associated with slow progressive loss in men and women leading to hip and vertebral fractures — Type II

Lifestyles that influence bone mass:
(1) Cigarette smoking or excessive use of alcohol
(2) Hormones
(3) Medications (glucocorticoids, anticonvulsants, heparin, thyroxine)
(4) Diseases – Cushing's, hyperthyroidism, anorexia, amenorrhea,
(5) Nutrition – vitamin D + Ca^{+}
(6) FMH

No guidelines for screening (BMD testing) but might offer to women who:

(1) Refuse or decline HRT
(2) Are aged 65 and older
(3) Have risk factors (other than being white, postmenopausal, and female) such as being on long-term medications such as corticosteroids, lithium, GnRH agonists, anticonvulsants, tamoxifen, TPN, DEPO, or diseases such as COPD, eating disorders, spinal cord transaction, thalassemia, weight loss, MS, multiple myeloma, s/p gastrectomy, etc.
(4) Have suffered a fracture to confirm diagnosis and determine severity of disease.

Diagnose with DXA

Bone mass values measured in — g/cm^2
Sex and age-matched reference population — Z-score
Bone mass relative to mean peak bone mass — T-score
If a T-score is between 0.0 and –0.9 then bone mass is — normal to low normal
If a T-score is between –1.0 and –1.4 then bone mass is — 10–15% below normal and the risk of spine and hip fracture is 2.3 and 2.6 times greater
If a T-score is between –1.5 and –1.9 then bone mass is — 15–20% below normal and the risk of spine and hip fracture is 3 and 4 times greater
If a T-score is between –2.0 and –2.4 then bone mass is — 20–25% (osteoporotic) and the risk of spine and hip fracture is 5 and 7 times greater
If a T-score is –2.5 or lower then bone mass is **more than 25%** below and risk of spine and hip fracture is 8 and 11 times greater
These T scores are compared with a healthy young adult female with a T-score of 0.0
Osteoporosis is standard deviation of BMD — ≥ 2.5
Osteopenia is standard deviation of BMD between — 1–2.5
Two basic characteristics of osteoporosis: reduced bone mineral density (BMD) and poor bone quality
For every decrease of 1 SD in lumbar-spine BMD, the risk of vertebral fracture is approximately doubled

DXA – dual energy X-ray absorptiometry (measures spine, hip, or total body)

QCT – quantitative computed tomography (measures spine)

DPA – dual photon absorptiometry (measures spine, hip, or total body)

Pearls of peripheral measurement

pDAX – peripheral dual energy X-ray absorptiometry (measures wrist, heel, or finger)

SXA – single energy X-ray absorptiometry (measures wrist or heel)

QUS (quantitative ultrasound) uses US to measure density at heel, lower leg, or patella

pQCT – peripheral quantitative computed tomography (measures wrist)

RA – radiographic absorptiometry (X-ray of hand; BMD compared to metal wedge)

SPA – single photon aborptiometry (measures wrist)

Peripheral bone mineral density can be used to assess fracture risk, with one exception (hip fracture risk), which is best assessed with direct

measurements of hip density – hence the reluctance to promote the machines that measure peripheral bone density. However, peripheral machines do a good job, with the method that uses a finger doing the best (due to the ability to immobilize a finger in a standard fashion, minimizing variability). Peripheral measurements have a predictive value very similar to that of central measurements. Finding a low BD by any method indicates a high risk of fracture within the following year. A fracture means osteoporosis unless ruled otherwise. (Siris ES, Miller PD, Barrett-Connor E, *et al*. Identification and fracture outcomes of undiagnosed low bone mineral density in postmenopausal women: results from the National Osteoporosis Risk Assessment. *JAMA* 2001;286:2815–22)

Other methods of diagnosis

Pearls

Cortical (outer shell) bone makes up what % of bone?	75%
Trabecular (spongy, inner) bone makes up what % of bone? (vert + pelvis)	25%
Peak bone mass peaks at age	30
What % bone is lost after age 30 per year?	0.4%
After menopause how much cortical bone is lost per year?	2%
After menopause how much trabecular bone is lost per year?	5%

Prevention of bone loss

Fluoride increases BM but not architecture
What increases both BM and architecture but not preventive? PTH
Alendronate (Fosamax®) – 5 mg daily to prevent and 10 mg daily or 70 mg weekly to treat
Risedronate (Actonel®) – 5 mg daily or 30 mg (Paget's dose) per week now available in 35 mg per week dose and FDA-approved
Ibandronate (Boniva) – 150 mg per month.
In the MOBILE study, the 150-mg monthly dose of ibandronate was superior to daily use in terms of lumbar spine bone density
Raloxifen (Evista®) 60 mg orally daily
Raloxifen has no time limit
Tamoxifen (Nolvadex®) – presently used in breast cancer prophylaxis
Estrogens, exercise, calcium, vitamins
ERT reduces lifetime fracture risk by more than half
HRT's greatest benefit is obtained if started shortly after, menopause

(1) Reduces Colles' fractures by @ 50%
(2) Reduces incidence of vertebral deformities by @ 90%
(3) In low BMD of forearm, bone loss was slowed by exercise alone or in combination with calcium but ONLY with combined use of HRT and exercise was bone loss reversed and bone mass increased

Bone density does not necessarily define the whole story in prevention of fractures in that raloxifene produces a smaller increase in vertebral bone density compared to estrogen and alendronate, yet the three agents are associated with essentially identical reductions in vertebral fractures. Studies of combined therapies not yet available at time of this publication

Treatment of osteoporosis

Alendronate (Fosamax) – 10 mg daily or 70 mg per week: 50% reduction in all
Risedronate (Actonel) – 35 mg orally per week dose:
Vertebral >40%, other >30% reduction
Ibandronate sodium (Boniva) – 150 mg orally every month
Calcitonin (Miacalcin®) NS – 200 mg nasally per day. May have anesthetic properties for fractures and be useful in nursing homes where patients are bedridden (cannot sit up to take bisphosphonates):
21–54% reduction in vertebral fractures only
Raloxifene (Evista) – 60 mg daily. Vertebral: 40% reduction

Basic Four

(1) *HRT or ERT* (estrogen replacement therapy) 30–40% reduction
(2) Calcium 1200–1500 mg daily (500 mg t.i.d. or 600 mg b.i.d.) 30% reduction
(3) Vitamin D 400–800 mg daily. Probably as effective or better than calcium

(4) Exercise:
- weight bearing
- stimulates osteoblasts to form new bone
- maintains bone mass
- increases strength and coordination

As to when to intervene, one should rely on a constellation of factors, not just numerical bone-density value. To prevent fractures, one cannot simply wait until women have osteoporosis to treat them. For instance, the absolute fracture risk of a 50-year-old woman with a T score of –3 is exactly the same as that of an 80-year -old woman with a T score of –1

For treatment of advanced osteoporosis

Human parathyroid hormone

→Teriparatide (Forteo) 20 µg SC daily for 18–24 months

Parathyroid hormone injections have significant effects on fracture risks in osteoporotic patients (Neer RM, Arnaud CD, Zanchetta JR, *et al*. Effect of parathyroid hormone (1-34) on fractures and bone mineral density in postmenopausal women with osteoporosis. *N Engl J Med* 2001;344:1434–41) 65% reduction in vertebral and 54% in other fractures

Menopausal women on ERT need how much calcium per day? 1000–1200 mg

Menopausal women not on ERT need how much calcium per day? 1500 mg

Adolescents need how much calcium per day? 1200–1500 mg

Calcium citrate is more soluble and better absorbed than calcium carbonate. Calcium citrate should be recommended to patients who are taking H_2-blockers or proton pump inhibitors, as well as elderly patients with occult achlorhydria who are unable to absorb calcium from calcium carbonate supplements.

Human monoclonal antibody

→Denosumab 6, 14, or 30 mg SC q. 3 months or 14, 60, 100, or 210 mg SC q. 6 months increases BMD and decreases bone resorption in postmenopausal women with documented low bone mass. Denosumad is a new and highly specific, fully human antibody against receptor activator of NF-kappaB ligand (RANKL). RANKL acts as an endogenous activator of osteoclastogenenesis and osteoclast activity and its inhibitor, osteoprotegerin (OPG). According to one study, of asymptomatic conditions requiring preventive treatment, osteoporosis has one of the poorest adherence rates. Compliance with treatment over a long period of time is the single most important factor in osteoporosis prevention. The advantage of denosumab is not only its comparative increase in bone mineral density over alendronate and others, but also its ease of compliance since it is given by SC injections at 3- to 6-month intervals. (McClung MR, Lewiecki EM, Cohen SB, *et al*. Denosumab in postmenopausal women with low bone mineral density. *N Engl J Med* 2006; 354: 821–31)

Strontium ranelate

Strontium ranelate is a new therapy proposed for the treatment of osteoporosis. It has anabolic and antiresorptive qualities and acts by increasing collagen and no-collagen protein synthesis, inhibiting osteoclast differentiation, reducing osteoclast function, and enhancing pre-osteoblast differentiation. Studies in postmenopausal women show that it is effective in treating and preventing osteoporosis

Other treatments include calcitriol, other bisphosphonates (etidronate, pamidronate, tiludronate, zoledronic acid), sodium fluoride, and tibolone

Combination therapies

Alendronate and estrogen or alendronate plus a SERM have been shown to be superior than either single agent. The same has been shown when treatments are combined with testosterone

Androgens to reduce risk of fracture

Adding an androgen to ERT or HRT may offer greater skeletal benefit than estrogen alone. Androgens decrease SHBG (one of the independent factors for increased risk of osteoporosis), i.e. low levels of E_2 or DHEA-S or high levels of SHBG or PTH

Low androgen concentrations in premenopausal women have been linked with bone loss

	Estratest® vs CEE showed similar decreases in urinary excretion of bone resorption markers (deoxypyridinoline, pyridinoline, hydroxyprolene) The CEE showed decreases in the serum markers of bone formation. Estratest showed increases in all bone-formation markers. Levels of SHBG increased with CEE alone and decreased with E/A (Estratest). E/A therapy was associated with a significant increase in spinal BMD as compared with baseline (Watts NB, Notelovitz M, Timmons MC, *et al*. Comparison of oral estrogens and estrogens plus androgen on bone mineral density, menopausal symptoms and lipid-lipoprotein profiles in surgical menopause. *Obstet Gynecol* 1995;85:529–37)	
	E_2 implants, 50 mg alone vs E_2 50 mg plus testosterone 50 mg, were administered three times yearly. BMD (total body, vertebra and hip) increased earlier and to a greater degree in the E/A group	
	Micronized progesterone and medroxyprogesterone acetate did not add to the bone-protecting effects of ERT, although norethindrone acetate (an androgenic progestin) has been shown to have an additive effect in BMD when compared with those treated with ERT alone	
Androgen-deficiency syndrome	Symptoms can include decreased libido (less desire, less frequency, and less sexual pleasure), bone loss, fatigue and lack of well-being Androgen levels can decrease by almost half during menopause and when a woman's ovaries are surgically removed androgen loss is much more dramatic with testosterone levels dropping	80%
Statins	Associated with a 71% significant reduction in fracture risk	
Soy products	Basic research indicates that dietary soy products may have definitive effects in protecting estrogen-deficient animals from the development of osteoporosis and osteopenia. However, there is little clear evidence that these products will work in treatment of already established osteopenia in humans. It should not be detrimental to increase the consumption of these products in the Western diet. The American Heart Association recommends 20–50 g/d of soy protein. Isoflavone supplements should contain about 50 mg/d and should not exceed 100 mg/d. IP is administered as 200 mg t.i.d., and if used should be combined with both calcium and vitamin D supplements.	
	• Parathyroid hormone analog builds new bone. Estrogen, bisphosphonates, and SERMs retard resorption	
Trials	MORE (Multiple Outcomes of Raloxifene Evaluation) trial – showed the prevalence of fractures (not rate) is far greater with osteopenia	
	Rotterdam trial – 12% of nonvertebral fractures were in women with normal BMD	
	NORA (National Osteoporosis Risk Assessment) trial – of postmenopausal women who suffered a new fracture within 1 year, 82% had osteopenia.	
Four top predictors of fracture	The four top predictors of fracture within 1 year are: (1) Previous fracture, regardless of T score (2) T score worse than –1.8 (3) Poor health (4) Poor mobilitiy	

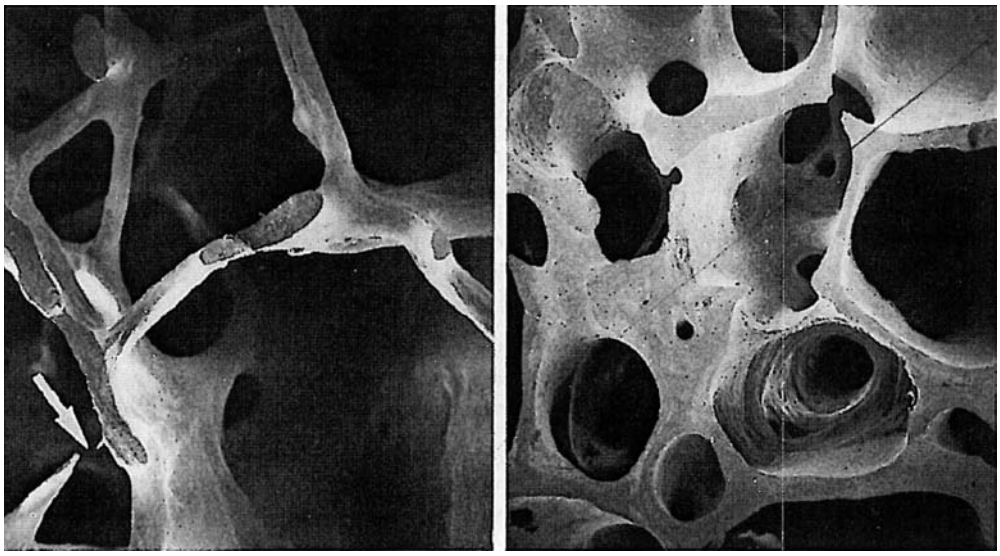
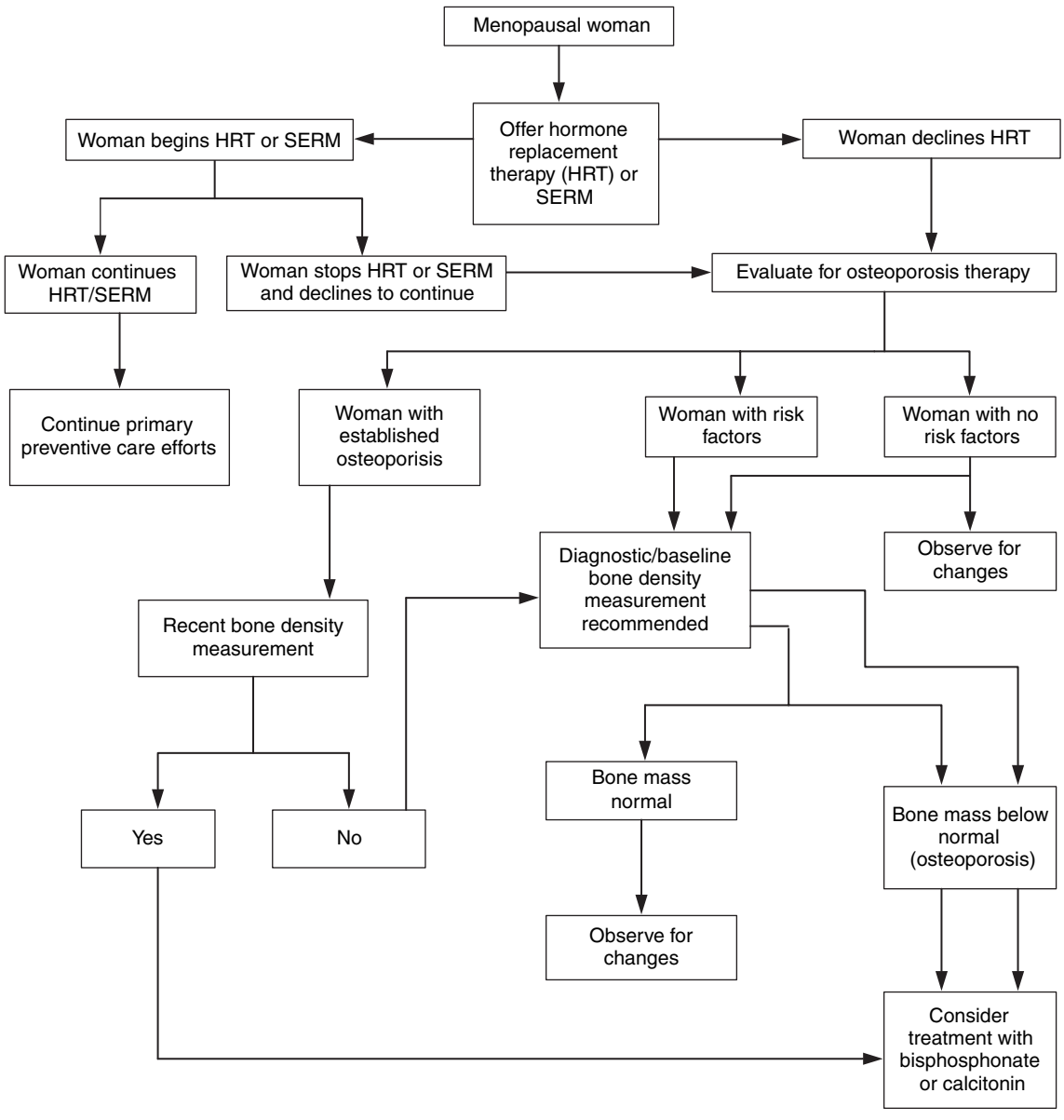


Figure 18 Osteoporotic vs normal bone with arrow pointing to fracture in the osteoporotic bone

Osteoporosis treatment algorithm



OVARY

Blood supply

Ovarian artery (branch of aorta). Left ovarian vein drains to left RENAL vein. Right ovarian vein drains to inferior vena cava

Lymphatics

Para-aortic nodes

Nerve supply

Sympathetic plexus

Embryology and physiology of ovary

Theca cells are involved with androstenedione production and are responsive to	LH
Granulosa cells synthesize estrogen and are responsive to	FSH
Germ cells multiply to form how many oogonia by 16–20 weeks?	6–7 million
How many oocytes are present at birth?	2 million
Adult ovary contains @ how many follicular units?	300 000
About how many will reach full maturation and ovulate?	300–400
Inhibin and follistatin secreted by granulosa cells suppress	FSH
Activin augments	FSH
Ovulation occurs how many hours after LH surge?	34–36
Ovulation occurs how many hours after LH peak?	10–12
Ovulation occurs how many hours after estradiol peak?	24–36
What dose of radiation to the ovary would have no effect? (in rads)	≤ 60
What dose in rads to the ovary would result in 100% sterility?	800
Genes known to be expressed exclusively in oocytes	*ZP3*

Treatment of choice for BORDERLINE ovarian cancer in infertility patients is cystectomy. Borderline tumors demonstrate epithelial proliferation but no evidence of stromal invasion

Extraovarian implants are present in borderline tumors in what %?	30%
Recurrence of borderline tumor (not associated with disease spread elsewhere) is	5–10%
What % of borderline tumors make up epithelial tumors?	15%

Unilateral S&O is option for stage IA and residual disease

TAHBSO is treatment for perimenopausal and postmenopausal patients

Intraop: Bxs, washings, partial omentectomy, lymphadectomy

Postop: semiannual follow-up

Prophylactic oophorectomy

Cancer prevention

Risk of cancer	1/70
According to SEER, lifetime risk of ovarian cancer is	1/58 1.71%
If 300 000 oophorectomies were done, how many cases of ovarian cancer could be prevented	1000

Prophylactic oophorectomy cannot prevent the development of peritoneal carcinomatosis. There is essentially no screening method for ovarian cancer. There are some useful adjuncts to screening high-risk patients such as:

(1) Vaginal ultrasound (three-dimensional US possibly better)
(2) Serum CA-125
(3) LPA (plasma lysophosphatidic acid) detected 9 or 10 patients with stage I ovarian cancers and in all with higher stages

Oral contraceptives decrease risk of ovarian cancer (when taken for 5 years or more) by	50%

Why take ovaries (at time of hysterectomy)?

(1) Cancer risk
(2) Family history of epithelial ovarian cancer
(3) Family member or friend had reoperation
(4) 5–20% patients have reoperation for pathology involving ovaries
(5) Patient's desire to have ovaries removed

Individualize, HRT compliance important, genetic risk

What % of endodermal or embryonal tumors will recur if no postop treatment is given?	85%
Germ cell tumors IA1 are cured with surgery alone in what %?	100%
If stage II, III or IV – chemotherapy should be bleomycin, etoposide and cisplatin × how many doses?	3

The chemo Rx of bleomycin, etoposide and cisplatin cures what % of cases?	95%
What % of granulosa cell tumors are stage I at time of diagnosis?	90%
What % of granulosa cell tumors are unilateral?	95%
"Second-look" lap for ovarian cancer – advantage is that this % of patients will have advanced disease at second-look surgery	50%
The other advantage is there is the opportunity to resect (controversial) and theoretically reduce any residual tumor. The disadvantage is major surgery/prolonged ileus (investigational procedure)	
Suboptimal disease (IIIC) is residual disease > 1–2 cm	
Suboptimal disease has a poor prognosis of 5-year survival rates @	10–15%
Treat with Taxol® (paclitaxel) and Platinol® (cisplatin). Median survival is	> 3 years
Whole radiation treatment only for patients with no gross residual disease	
Lifetime current risk of ovarian cancer associated with *BRCA1* germline mutation is	30%
Lifetime current risk of ovarian cancer associated with *BRCA2* germline mutation is	10%
Oral contraceptive use may reduce a woman's risk of ovarian cancer as much as	50%
Risk of primary peritoneal cancer after prophylactic oophorectomy in increased risk patients is	2–15%

PAGET'S DISEASE OF VULVA

Symptoms	Pruritis, soreness, superficial, red to pink, velvety, eczematoid lesion	
Diagnosis	Keyes punch 3–5 mm	
Histology	"Percolating to the surface"	
	Eosinophilic Paget's cells at epithelium	
Treatment	Wide excision	> 2 cm
	Frozen section	
	Radical bilateral inguinal–femoral lymphadenectomy p.r.n.	
	Milk line lesions comprise what % of lesions?	15%
	Adenocarcinoma or squamous carcinoma is present in what % of Paget's?	5–20%
	Wide local treatment with how many cm beyond margin?	1–2
	If underlying adenocarcinoma, rad vulvectomy or hemivulvectomy with femoral–inguinal lymphadenectomy	
	IV fluorescein allows visualization of margins:	
	Pos predictive value	97.4%
	Neg predictive value	99.9%
	Survival rate in all	90%
	Paget's disease of the vulva often recurs, especially when the initial lesion is large.	

PAIN

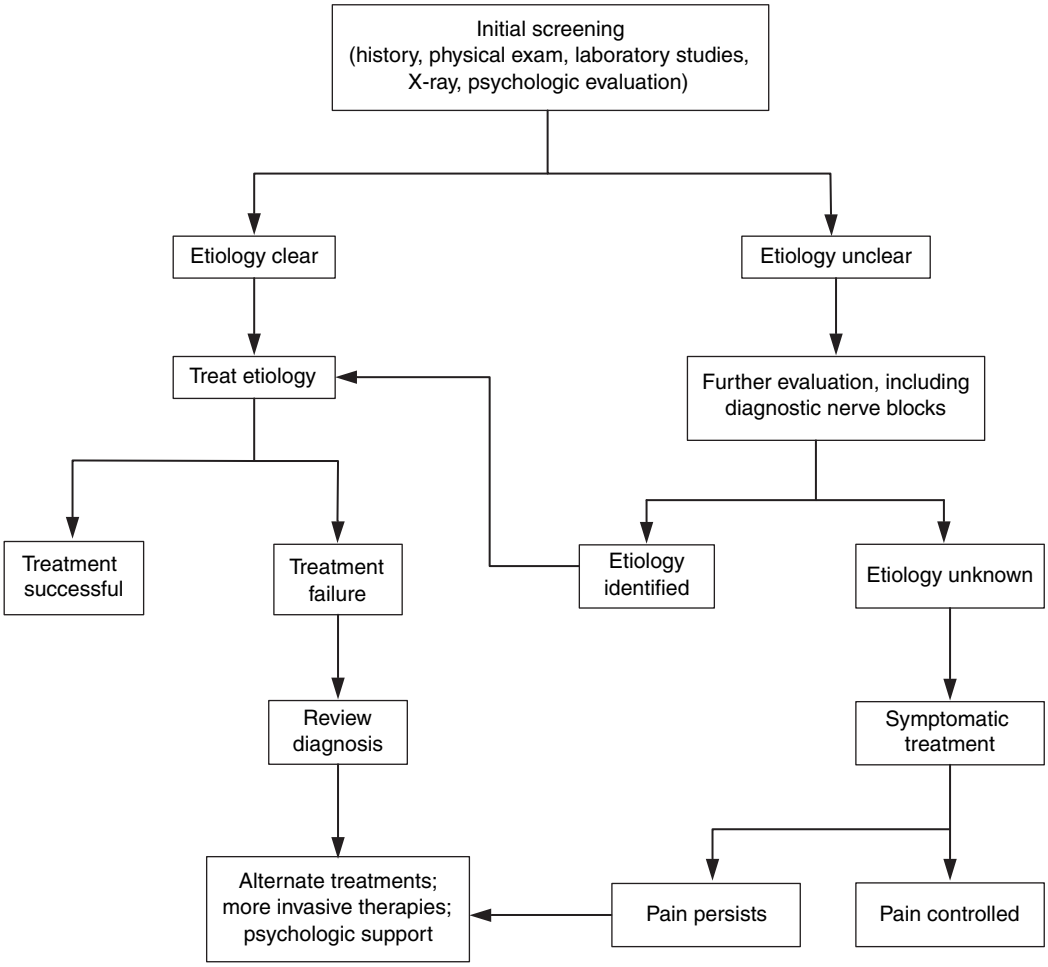
Chronic pelvic pain	Best relief of pain associated with endometriosis is GnRH – % of patients with pain relief?	75–90%
	Progestin (Provera) – reported to be as effective as GnRH agonist but comparison has not been done. OCPs – (continuous fusion) but not as good as GnRH. Adhesiolysis and/or cervical dilatation – neither shown to help	
	History of sexual abuse seen in what % of chronic pelvic pain?	50%
	Laparoscopy reveals this % of pelvic abnormalities incidentally	60–80%
	What % of these patients are undergoing laparoscopy for sterilization?	30%
Pelvic pain differential	PID/infection, dysmenorrhea, ovarian cyst (rupture), adenomyosis, endometriosis, appendicitis, cystitis, diverticulitis, mesenteric adenitis, kidney/bladder stone, pelvic adhesions, leiomyomata, ectopic pregnancy, torsion, tubal syndrome	

(after tubal), hydrosalpinx, lower lobe lung process, pyelonephritis, Meckel's diverticulum, viral bacterial GI syndrome, irritable bowel syndrome (IBS), Crohn's disease, ulcerative colitis and/or psychosomatic

Pain medication

(1) Morphine – best, well known, inexpensive
(2) Ketorolac (Toradol®) – NSAIDs, GI ulceration, bleeding — 5 days
(3) Meperidine (Demerol) – Short duration — 2.5–3.5 h
Metabolite (normeperidine). CNS stimulation (dysphoria, agitation, seizures). Avoid in elderly
(4) Methadone – more expensive than morphine sulfate, longest acting. Reserve for those who cannot tolerate morphine
(5) Sublimaze® (fentanyl) – short acting. Patches require additional dosing and can cause increased respiratory depression
(6) Oxycodone – oral dosing only. Requires frequent dosing. Caution to avoid acetaminophen toxicity
(7) PCA (patient controlled anesthesia) – better than IM dosing. Increased expense. Associated with increased urinary retention

Chronic pain management



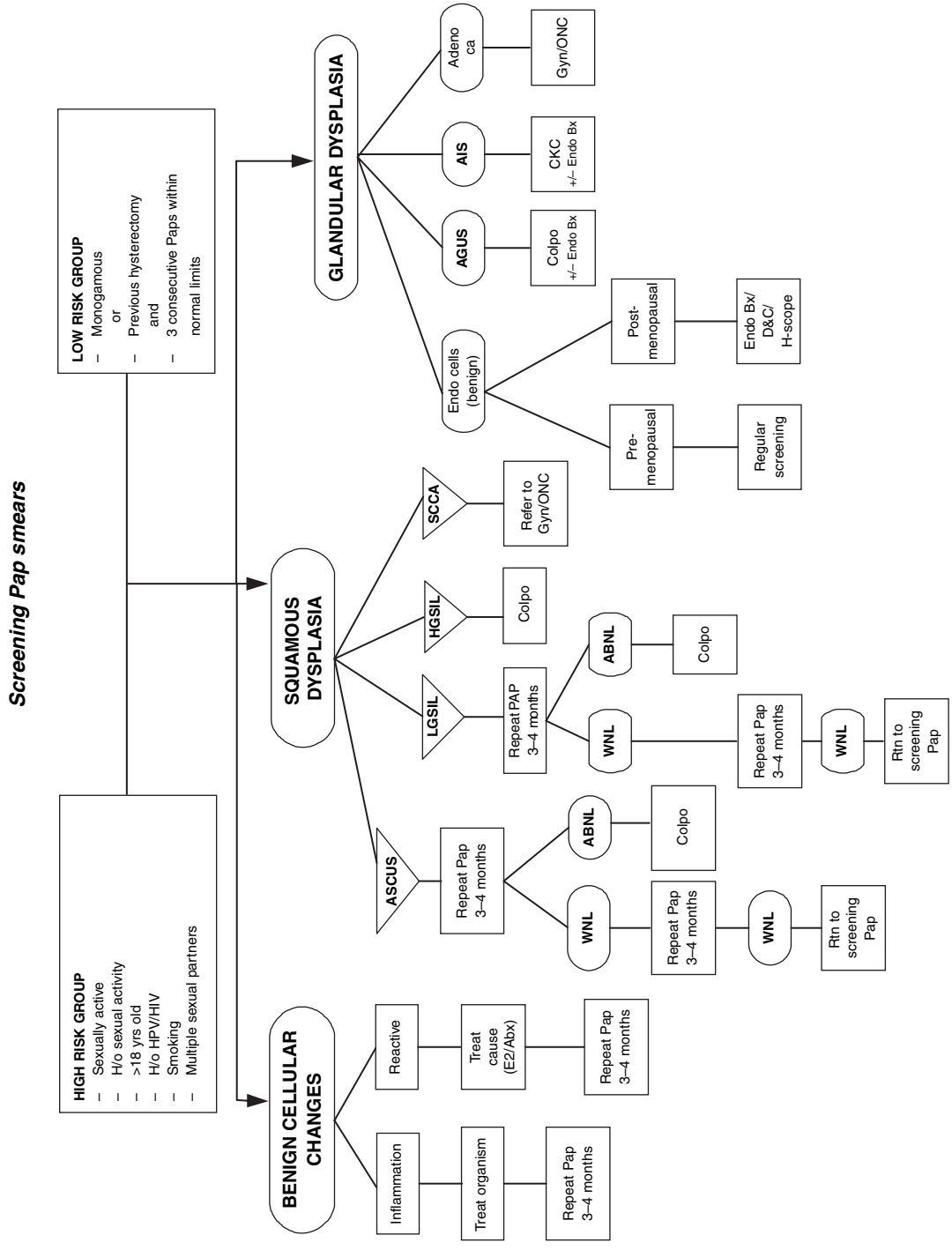
PAP SMEAR

Introduction of Pap smear was by Papanicolaou and Traut in 1943
Since 1947, the incidence of cervical cancer went from 34 per 100 000 to 7.7 per 100 000 in 1996

How to perform a Pap smear

Collect before bimanual exam. Collect before testing for STDs.
Ectocervix is scraped using spatula and rotated 360° two times.
Ectocervix is obtained before the endocervical brush is used.
Do not use lubricants during collection of specimen
Upper two-thirds of epithelium show some evidence of change but lower third lacks evidence CIN I
Abnormal changes involve the lower two-thirds of the epithelium CIN II
Full thickness and mitotic figures CIN III
Early stromal invasion of small foci less than ? from basement epithelium invading BM < 1 mm
Microinvasive carcinoma involves what depth/what horizontal spread? ≤ 3 mm/≤ 7 mm
Occult invasive carcinoma is what depth of invasion? > 500 mm^3 (5 mm)
Mortality from cervical cancer has decreased by what % since Paps introduced? 70–90%
False-negative rate for single Pap test is 10–25%
What % LGSIL spontaneously resolve? 60%
What % LGSIL progress to HGSIL? 15%
Evaluate high-risk premenopausal patient with ASCUS on a Pap smear with colposcopy
What % of women with ASCUS have a high-grade dysplastic process? 5–13%
High-risk patients requiring frequent Pap screening:
Females with multiple partners
Females who began intercourse at an early age
Females whose male partners have had sexual partners with cervical cancer
Smokers and abuses of other substances including alcohol
Management for a patient with a high-grade lesion that involves the endocervical canal is CKC or LEEP
Manage AGUS that is highly suggestive of an endocervical lesion on Pap with CKC or LEEP
THIN PREP is monolayer prep approved by FDA that increases sensitivity of Pap due to increased diagnosis of LGSIL and residual fluid can be saved without calling pt back but cost how much more? $35
Washes debris $15–20 more than conventional Pap
PAPNET – normals are rescreened and computer selects how many of the most abnl cells? 128
These cells are re-examined and original slide re-examined p.r.n.
Cost is how much more? $45–50
Paps prepared in the normal manner
Paps read as NORMAL are RESCREENED. Re-examined by cytotechnologist or pathologist. The original slide is re-examined if necessary. Disadvantage = + 3–7 more days to get result and more expensive
AUTOPAP – conventional Pap is reviewed by a computer program. (Identifies 5 × more false-negatives) Adds cost of $45–50
Endocervical cells *absent* on Pap. If no risk (with 3 nl Paps + normal with only absence) then repeat 12 months
If risk factors present, repeat Pap at patient's convenience

Screening Pap smears



Pap test frequency

Begin Paps with onset of sexual activity and continue every or more frequent with increased risks	3 years
After hysterectomy for bleeding with mild dysplasia	q. 12 months
After hyst for severe cervical dysplasia after cone	q. 6 months
Pelvic pain with three consecutive normal Paps	None indicated
Hyperspectral diagnostic imaging of the cervix, using UV light generated by a mercury vapor lamp, is able to discriminate CIN from normal tissues (but with difficulty differentiating squamous epithelium from squamous metaplasia) in a matter of how many seconds?	12

Local excision of CIN after Pap and colposcopy evaluation

(1) Cryo – nitrous or CO_2. Double-freeze technique helpful – extends 4–5 mm beyond edge of probe
(2) CO_2 laser vaporization – Depth 7 mm effective for 99%. Power density $\geq$ 1000 Watts/cm^2
(3) LEEP – Depth 7–8 mm. Extend 4–5 mm beyond affected area

• If a woman has an ASCUS finding on cytology but is HPV-negative, rescreening is preferable over immediate colposcopy

PARASITES

See chart on next page

PARASITIC INFECTIONS IN PREGNANCY

Infection	*Organism*	*Symptoms*	*Route of infection*	*Diagnoses*	*Pregnancy effects*	*Placental trans*	*Drug of choice (FDA)*	*Alternate drug*	*Drug (if not pregnant)*
Giardiasis	*Giardia lamblia*	Watery, bulky diarrhea; abd pain; flatulence; nausea, wt loss; malaise	Fecal–oral	Trophozoites in stool	Secondary maternal disease	None	Humatin® (paromomycin) 30 mg/kg/day in 3 doses for 5–10 days (B)	Flagyl (metronidazole) 250 mg t.i.d. ×5 days (B) (last two trimesters)	Atabrine HCl (quinacrine) 100 mg/day ×5 days (C)
Pinworms	*Enterobius vermicularis*	Intense perineal and anal itching particularly at night	Auto-inoculation	Demonstration of worms on adhesive tape	None known	None	Antiminth®, Combantrin® (pyrantel pamoate) 10 mg/kg – max 1 g (base) after 1st trimester. Repeat dose 2 weeks later Clothing/bedding to be washed in hot water and chlorine bleach		Vermox® (mebendazole) Equizole® Mintezol® Thibenzole® (thiabendazole)
Hookworms	*Ancylostoma duodenale Necator americanus*	Anemia	Skin penetration of larvae from soil	Eggs in fecal smears	Secondary to maternal anemia	None	Iron Pyrantel 11 mg/kg × 3 days		
Amebiasis	*Entamoeba histolytica*	Asymptomatic or 10–50%; Sxs of colicky lower abd pain	Fecal–oral	*E. histolytica* in stool or sigmoidoscopy	Secondary to maternal disease	None	Humatin 30 mg/kg/day in 3 doses ×7 days. Give Flagyl 750 mg p.o. t.i.d. × 10 days then Humatin for severe infections	If severe, give dehydroemetine 1.5 mg/kg/day ×5 days CDC (404) 6393670	Iodoquinol or emetine
Malaria	*Plasmodium ovale vivax* Non-resistant *P. falciparum* Chloroquine-resistant *P. falciparum*	High fever/chills, abd pain, nausea vomiting, delirium	Anopheline mosquito	*Plasmodium* parasites in stained peripheral blood smears	Secondary to maternal disease	1–4% congenital malaria documented	Aralen® (chloroquine) 1 g then 500 mg at 6, 24, and 48 h then weekly till after delivery; then primaquine 15 mg daily × 14 days postpartum (screen for G-6PD)	Parental quinine gluconate for life-threatening infections *P. falciparum* Rx for resistant *P. falciparum* is quinine 650 mg. t.i.d. ×3–7 days plus pyrimethamine/ sulfadoxine, 3 tabs on day 3 of tx	Larium® (mefloquine)
Pediculosis pubis (Crab louse)	*Phthirius pubis*	Pruritus and itching	Close contact usually sexual	Visualization of adult lice or nits (eggs) under magnification		None	Permethrin 1% cream applied × 10 min then washed off	Pyrethrins Piperonyl butoxide ×10 min then washed off	Lindane 1% ×4 min then wash

PARVOVIRUS

	Incidence 1/400 pregnancies
	Maternal parvovirus is transmitted to the fetus in @ what % of cases? 30%
	Fifth disease (erythema infectiosum) is caused by parvovirus B19, a single-stranded DNA virus. It was called fifth disease because it was 5th pink-red rash – following scarlet fever, measles, rubella and roseola – to be described by physicians. 'Slapped cheek' is seen
	Spontaneous abortion may result from maternal infection in the first trimester 10%
	Parvovirus diagnosed in the late second and third trimesters carries a risk of stillbirth and hydrops fetalis
	Non-immune hydrops is caused from the anemia caused by the virus
Diagnosis	The ELISA and Western blot analysis appear to be the most reliable methods for detecting IgG and IgM antibodies in maternal serum
	What % of adults are immune? (Have IgG antibodies) 50%
	If IgG and IgM are both negative, repeat titers in 3–4 weeks
Management	Weekly ultrasound examinations for 8–10 weeks after diagnosing parvovirus in the gravida. If hydrops seen on ultrasound, cordocentesis (PUBS) is done
	Complication rate of PUBS is 1%
	Blood is sent to lab for MCV, Hct, leukocyte and platelet count
	When intrauterine RBC transfusion is performed in presence of hydrops and anemia, fetal survival rate ranges from 60–80%
	Without treatment, rate drops to 15–30%

PATERNAL AGE

	Predisposes fetus to mutations associated with mutations in X-linked genes through carrier daughter ("grandfather effect")
	Hemophilia A or Duchenne muscular dystrophy or predisposes fetus to mutations in autosomal dominant diseases
	Increased risk rises exponentially instead of linearly
Examples	Neurofibromatosis, achondroplasia, Apert syndrome or Marfan syndrome

PEAKS

What week gestation does fetal AFP peak?	15
What week gestation does maternal AFP peak?	30
What week gestation does maternal hCG peak?	10–12

PEDIATRIC DISCHARGE

	What % of pediatric discharge is non-specific? 75%
	What % of cultures will identify organism? 25%
	Think in descending order of incidence: Infection Foreign body Tumor
	Usually no need for anesthesia – use Huffman vagiscope or test tube with otoscope but not otoscope alone
	Use with care if nasal speculum is used
Why susceptible?	Exposed to more bacteria
	Lack estrogen
	Neutral pH
	Poor perineal hygiene
	Lacks glycogen, lactobacilli and sufficient antibodies
	Scratch–itch cycle
Symptoms	Pain, pruritis, irritation, dysuria

Differential diagnosis	Foreign body (especially if bloody discharge present)
	Pin worms (especially if primary symptom is itching at night) Ectopic ureter Child abuse
Diagnosis	KNEE CHEST POSITION Remove any foreign body with small female urethral swab or irrigate. Local trauma most common cause. Others – infectious, neoplastic, hormonally mediated, etc.
Treatment	Improve hygiene, Sitz baths, clean, D/C bubble baths and soft soap, apply 0.5% hydrocortisone if intense itching. Use Vermox for pinworms. Irrigation or removal of foreign body. Estrogen to vulva – not vagina. Antibiotic p.r.n. for 10–14 days

PEDIATRIC GYNECOLOGY

Congenital uteri	*Uterine unicollis* Rudimentary horn Bleeding, pain. Remove blind horn *Uterine didelphis* Slender cavities – Jones/Tompkins *Septate uterus* Hysteroscopic dissection Blind vagina (Müllerian remnant) Lateral to vagina – open vaginally *Arcuate uterus* Exposed to estrogen *in utero* *Imperforate hymen* Cruciate incision (10 to 4; 2 to 8)
Congenital absence of vagina	*See* Frank and McEndoe procedures
Ambiguous genitalia	Reassure – "genitals not developed yet". Rule out CAH – inability to produce cortisol. Get buccal smear and order karyotype. Raise as male if functioning phallus and Y chromosome, otherwise it is easier to surgically repair and raise as a female
	See Ambiguous genitalia
Fusion of labia	Atrophic – thin black line. Treat with estrogen cream b.i.d. 4–6 weeks
Abnormal bleeding	Vaginitis Trauma UTI or GI track Hormone activity Tumor (granulosa cell tumor – precocious puberty – nuclear grooves) Condyloma – sexual abuse Hymenal tags Sarcoma botryoides (rhabdomyosarcoma) Clear-cell adenocarcinoma Urethral prolapse – treat with estrogen Vitiligo – chronic irritation

PELVIC EXAMINATION

Vulva	Mons, labia majora and minus – scars or lesions?
	Clitoris – cylindrical? Body and glans of normal size and/or shape? Hymen – annular, septate, cribiform? Absent with porous introitus?
Vaginal orifice	Patulous with adequate rugae? No lesions of the urethral meatus?
	Skene's or Bartholin's ducts?
Uterus	Position, size, shape, mobile, tender?
Adnexa	Enlarged, mobile, tender?

PELVIC INFLAMMATORY DISEASE (PID)

	Lower abdominal pain is present in what % patients with PID?	90%
	Mucopurulent cervical discharge?	75%
	Sed rate > 15 mm/h	75%
	WBC > 10 000	50%
Diagnosis	Abdominal, cervical, AND adnexal pain plus one of the following:	
	Temperature	> 100.4°F
	WBC	> 10 500
	Sed rate	> 15
	Mass	
	Cul-de-sac evidence of WBCs or bacteria. Evidence of GC or *Chlamydia*	
Treatment	*Outpatient*	
	Ceftriaxone (Rocephin)	250 mg IM
	Doxycycline p.o. b.i.d. × 14 days	100 mg
	or	
	Cefoxitin 2 g IM plus probenecid 1 g orally	
	Doxycycline 100 mg b.i.d. ×14 days	
	or	
	Ofloxacin p.o. b.i.d. × 14 days	400 mg
	Metronidazole p.o. b.i.d. × 14 days	500 mg
	Inpatient	
	Cefotetan (Cefotan) IV q. 12 h	2 g
	Doxycycline IV q. 12 h	100 mg
	or	
	Cefoxitin 2 g IV q. 6 h	
	Doxycycline 100 mg IV or PO × 72 h then oral doxycycline 100 mg twice daily for a 14-day course	
	or	
	Clindamycin IV q. 8 h	900 mg
	Gentamicin IV q. 8 h	2 mg/kg then 1.5 mg
	or daily in dose of	5–7.5 mg/kg
	Treatment failures for outpatient therapy	10–20%
	Treatment failures for inpatient therapy	5–10%
	Re-evaluate patients getting outpatient therapy in	48–72 h
	Hospitalize:	
	(1) Outpt rx not improved after 48–72 h	
	(2) Adolescents	
	(3) Adnexal or pelvic abscesses	
	(4) Diagnosis of PID in question	
	(5 Pregnancy patients with acute PID	
	See also Sexually transmitted diseases	

PELVIC MASS

	Best screening is regular exams	
	Patient's AGE is MOST IMPORTANT factor for determining potential for malignancy	
	Premenarchal and postmenopausal – both highly abnormal ages to find a mass	
	Reproductive age – most masses occur in this age; most are benign	
Tumor markers	Germ cell tumors	AFP + hCG
	CEA is elevated in what % of ovarian cancer especially mucinous but also in PUD, diverticulitis, bronchitis and cigarette smokers?	40%
	Immunodiagnostic for serous tumors	CA-125
Ultrasound findings of malignancy	Multiloculated with septations. Irregular border with papillations. Internal complexities rather than clarity	
	Size	
	Ascites present??	> 8 cm

Cysts and masses

Benign tumors	*Malignant tumors*
Smooth walled	Irregular border
Cystic	Solid or semisolid
Mobile	Fixed
Unilateral	Bilateral (increase risk 2.6 ×)
< 8 cm	> 8 cm

Associated with nodules in cul-de-sac or associated with ascites

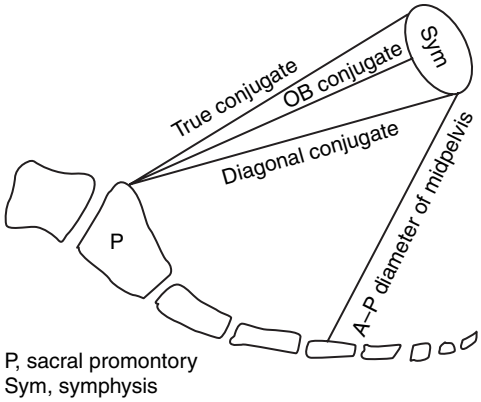
Surgical evaluation

Ovarian cystic lesion > 5 cm after 6–8 weeks without regression. Any solid ovarian lesion. Any ovarian lesion with papillary vegetation on cyst wall. Any adenexal mass > 10 cm. Ascites. Palpable mass in premenarchal or postmenopausal patient. Suspected torsion or rupture

Colonoscopy, IVP

PELVIC MEASUREMENTS

Inlet	Diagonal conjugate must be	≥ 11.5 cm
	Obstetric conjugate must be	≥ 10 cm
Midpelvis	Interspinous diameter	10 cm
	AP diameter	11.5 cm
Outlet	AP diameter	9.5–11.5 cm
	Transverse diameter	11 cm
	Posterior sagittal diameter	7.5 cm
	Biischial diameter (fist)	8 cm
Caldwell + Maloy	Gynecoid pelvis in what % females?	50%
	Oval, round, arch wide	
	Android (worse)	1/3
	Spine prominent, sidewalls converge	
	Anthropoid (OP is common). What % females?	1/4
	Blacks	1/2
	Whites	1/4
	AP diameter greater than long transverse	
	Platypelloid (OT is common). What % females?	3%
	Short AP, wide transverse	



P, sacral promontory
Sym, symphysis

Figure 19 Pelvic conjugates

PELVIC MUSCLE EXERCISE (PME)

See Kegel exercises

The Colpexin sphere, an intravaginal device for women with advanced genital prolapse that supports the prolapse above the levator musculature and helps patients strengthen their pelvic floor muscles, can also serve as a test to objectively assess pelvic floor muscle contractility and strength

PELVIC PAIN *See* Chronic Pelvic Pain.

PENTALOGY OF CANTRELL

Omphalocele
Lower sternal defect
Anterior diaphragm defect
Deficiency of diaphragmatic pericardium
Intracardiac abnormality

PERIMENOPAUSE

Diagnosis of exclusion. Draw TSH. Rule out thyroid disease. Inhibin levels may be helpful as these decrease as perimenopause is initiated. FSH and estradiol levels not helpful. Increased FSH level on cycle day 3 indicates poor prognosis for pregnancy. Inhibin which is made in granulosa cells of ovary suppresses pituitary FSH

Luteal phase inhibin responsible for early recruitment of dominate follicle for next cycle is inhibin A
Follicular phase inhibin may explain the short follicular phase in the perimenopausal pt – inhibin B

Hormone profile of perimenopausal woman

Increased FSH and LH, fluctuating and decreasing E_2 levels and decreased progesterone androstenedione and testosterone
Perimenopause is defined clinically by menstrual irregularities especially shortening of the menstrual cycles
Anovulation and bleeding are key symptoms
Progesterone measured @ 1 week prior to menses = diagnosis of anovulation if serum level < 300 ng/dl
Anovulation with DUB @ with proliferative or hyperplastic endometrium (no atypia) = MPA 5–10 mg × first 10 days of each month × several months
Follow-up aspiration curettage after 3–4 months on MPA. If histological regression not seen, do D&C
If hyperplasia with atypia is noted – hysterectomy is treatment of choice due to high risk of invasive ca
Progestins are not believed to be associated with an increased risk of VTE
Hot flushes – incidence of premenopausal flushes 10–25%
Other causes of hot flushes: cancer, carcinoid, leukemia, pheochromocytoma, psychosomatic, stress, thyroid disease
Cause – ? originates in hypothalamus – declining estrogen.
FSH, TSH, estradiol

Treatment

Estrogen, selective serotonin reuptake inhibitors are very effective
OCPs – 20 µg formulation has no significant impact on the measurements of clotting factors, even to smokers. Benefits also include decreased endometrial cancer, ovarian cancer, endometriosis, fibroids, benign breast disease, rheumatoid arthritis, ovarian cysts and increased bone density, regular menses, protection against atherosclerosis (possibly)

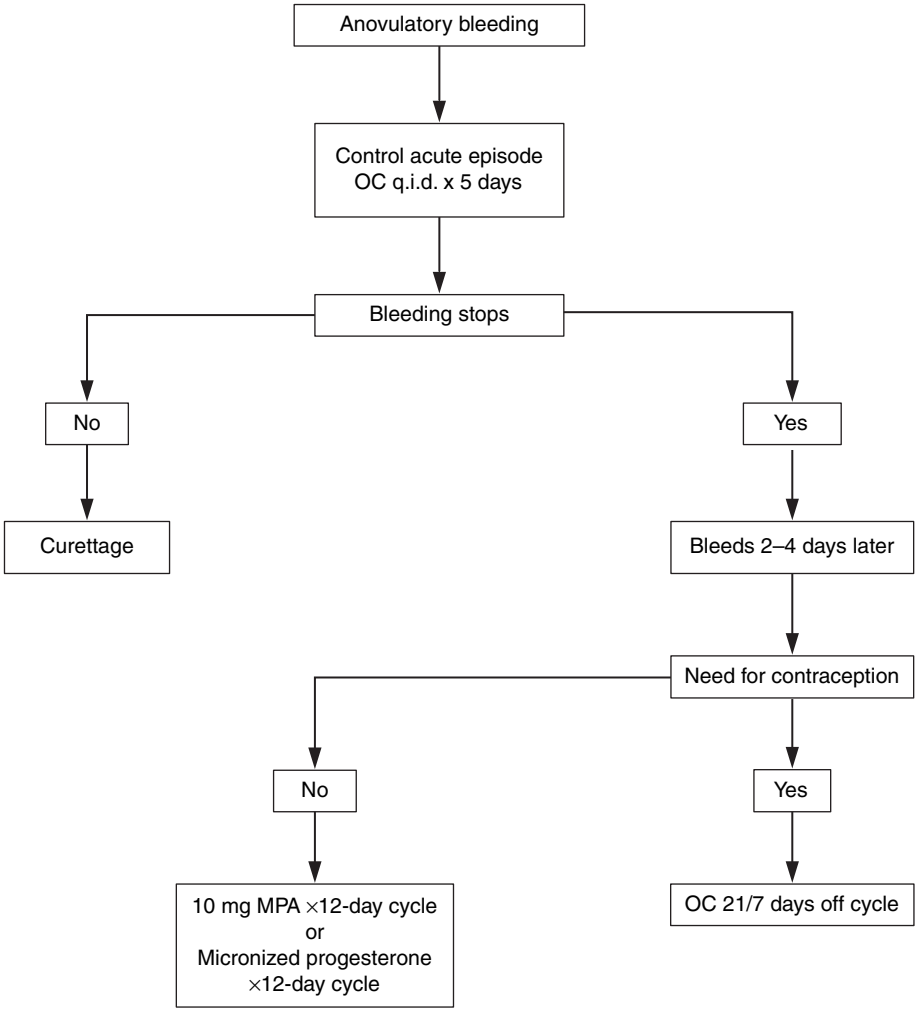
Conventional HRT is not the best option for perimenopausal women because it may not suppress ovulation, and therefore provide neither contraceptive benefit nor control of menstrual irregularity. Women should be counseled to use contraception until after the onset of menopause to prevent unwanted pregnancy

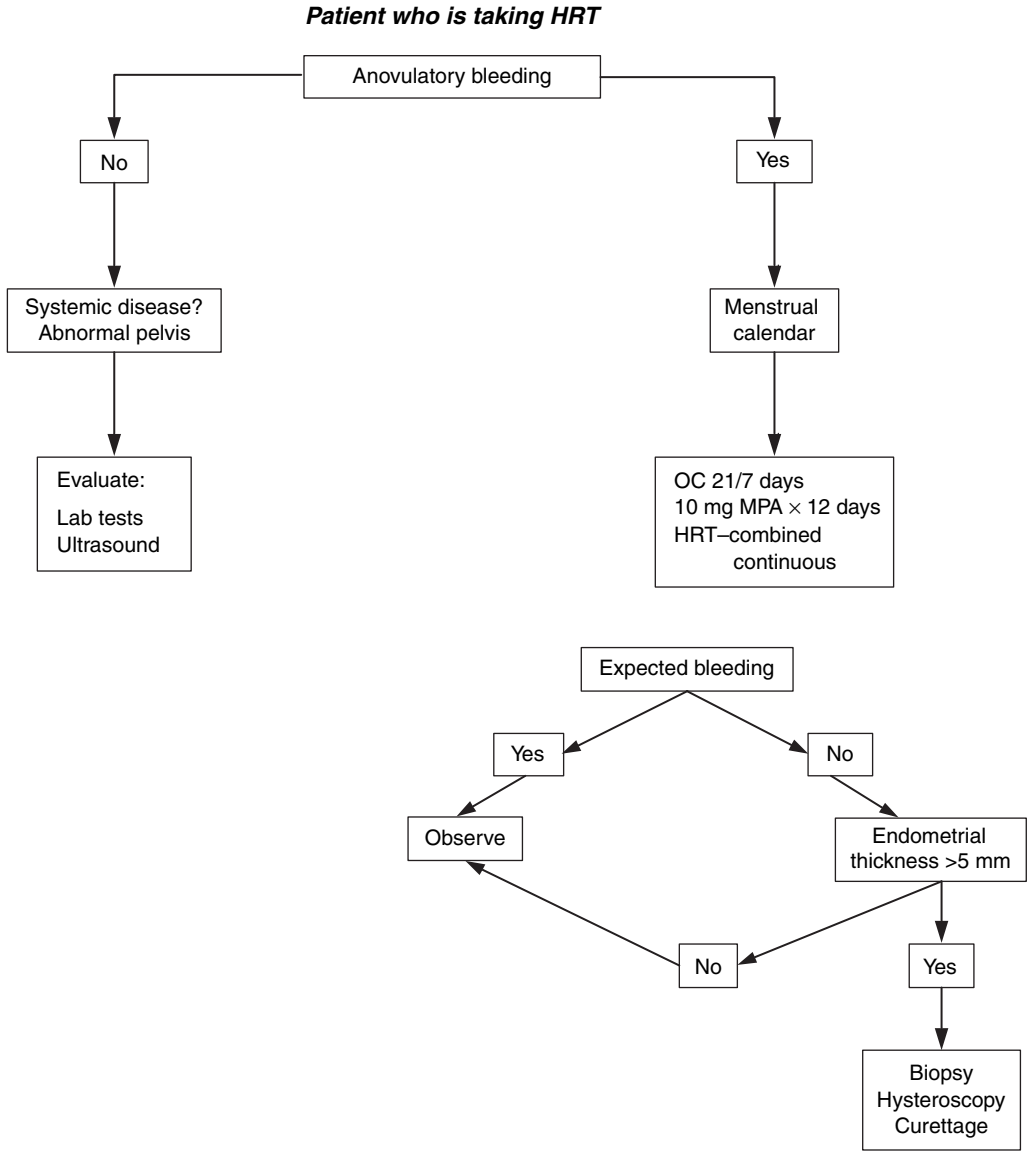
When to change from OCP to postmenopausal HRT:
Begin FSH level at age 50 (6–7th day of pill-free week – Friday)

When FSH > 20 IU/l, it is time to change (2 weeks pill free more accurate but not practical – some empirically change after age 50) or one can switch from OCs to HRT = when there is an increased FSH and/or decreased E_2 levels after an off-pill interval of 2 weeks

Average age of onset	45.1–47.5 years
Age of onset for 95% of women	39–51 years
Average duration	5 years
Duration for 95% of women	2–8 years

When patient is no longer ovulating





PERINATAL DEFINED

Perinatal period is from 20–22 weeks' gestation through how many completed days of life?	28
Neonatal period is from birth to how many days of completed life?	28
Early neonatal is first	7 days
Late neonatal period is days 8 through	28
Death is expressed in hours if infant under	24 h
Death is expressed in completed days of life if infant over	24 h
Live birth is one that shows ANY evidence of life at delivery	
Fetal death (formerly 'stillbirth') shows NO sign of life at delivery	
USA	500g
International	1000 g
Threshold of viability (23–25 weeks' gestation or < 750 g)	
23 weeks or 500–600 g survival rate	15–20%
24 weeks or 600–700 g survival rate	41–54%
25 weeks or 700–800 g survival rate	59–65%
Most common serious morbidity is RDS in infants under	750 g
What % children < 750 g experience moderate to severe disability including blindness + CP	50%
C-section for extremely premature infants is beneficial	

PERIURETHRAL INJECTIONS

Use with ISD and decreased mobility of urethra and/or higher risk for surgical procedures

Collagen – requires allergy testing and is not permanent
Durasphere – microscopic carbon beads, thick substance.
No allergy testing required, permanent and can be seen radiologically
Procedures are performed cystoscopically, require a PIN number to order and bulking agents are injected at urethrovesical junction

PESSARIES

Indications

(1) Temporary or delay measure until surgery for pelvic prolapse
(2) Use preoperatively to help heal erosions
(3) Use for young women with prolapse to defer surgery until after childbearing is complete (maintenance of childbearing ability)
(4) Diagnostic aid to clarify if pelvic or back discomfort are symptoms of pelvic prolapse
(5) Unmask latent stress urinary incontinence (following insertion of pessary, if new onset or worsening of SUI, suspect ISD)
(6) Avoidance of surgery (high-risk, failed previous procedure)
(7) Interim or permanent symptom control
(8) Patient preference for conservative management

Selection

Advanced prolapse with large genital hiatus	Gellhorn
Moderate cystoceles	Rings with support
Vault prolapse (first choice)	Ring with and without support
Rigid pessaries	No longer recommended
Cystoceles and rectoceles	Gehrung
Retroversion of uterus	Lever pessaries (Hodge and Smith)
Close follow-up needed due to possible severe vaginitis	Cube

Fitting

Most common sizes are 3 to 5
Similar to diaphragm fitting
Fit, have patient bear down on table, ambulate and sit on toilet in attempt to expel – if expels – too small. If it is uncomfortable or if there is urinary obstruction, the pessary is too large. If vaginal atrophy present, use topical estrogen, antibacterial cream or an estrogen ring above it

Patient follow-up

After initial fitting, patient should return in	1–2 weeks
If patient cannot remove and clean her own pessary	q. 2–3 months
Follow-up visits can be increased to	q. 6 months

Remember to follow-up cubes much more frequently

Contraindications

Severe erosions
Active vaginitis
Pelvic inflammatory disease
Non-compliant patient. (Severe complications can include vesicovaginal or rectovaginal fistula or impacted pessaries in neglected cases)

Anti-incontinence devices

See Urinary incontinence

PEYRONIE'S DISEASE

Treatment

Vitamin E 1000 mg daily
Verapamil 80 mg tablet daily
Isoptin®? Cream?
Radiation therapy
Surgery
Potaba® pills (6 pills 4 times per day)

PHEOCHROMOCYTOMA

Although a rare cause of hypertension and/or hot flushes, pheochromocytoma is ultimately correctable

Diagnosis

P-MET (plasma metanephrines) should be the first test of choice
Highest sensitivity tests are:
P-FMET (plasma-free metanephrines) and U-FMET (urinary fractionated metanephrines)

Highest specificity tests are:
U-VMA (urinary vanillylmandelic acid) and U-TMET (urinary total metanephrines)

PHYLLODES TUMOR

What % of phyllodes tumors contain some characteristics of malignant process? 10%

Diagnosis — Stromal proliferation with cellularity of connective tissue

Treatment — Total wide excision with wide margin of healthy tissue

PITOCIN

How to mix and calculate milliunits of Pitocin
10 U in 1 liter D5W
10 U in 1000 or
10 000 mu in 1000 or
10 milliunit per 1 cc or
1 milliunit per 0.1 cc to be given over 60 min or
6 cc per min
or to be given at rate ?
(Start at 1–2 mu/min) and/or 6 mu/min (Dublin)
Double at increased flow rate by 0.5–6 q. 15, 20 or 30 min until good labor pattern seen. STOP STAT for severe decels or tet contractions > 60 s

How does Pitocin work? — It has properties identical to oxytocin of the posterior lobe of the pituitary. It has selective action on smooth muscle of the uterus stimulating, increasing the frequency or raising the tone of the contractions of these muscles by causing the release of calcium from the sarcoplasmic reticulum. Pitocin is category A

PITUITARY MACROADENOMA WITH HYPERPROLACTINEMIA

Cabergoline (Dostinex®) is long-acting dopamine agonist with long half-life, fewer side-effects than Parlodel, dose is what weekly? 1–2 ×
Transphenoidal resection – recurrence rate for hyperprolactinemia can be as high as 80%
Within how many years of surgery? 3

PLACENTA ACCRETA

Incidence
Accreta, increta, percreta 78%, 17%, 5%
After one C-section and previa 23%
After multiple C-sections and previa 62%
Placenta accreta is the most common indication for peripartum hysterectomy, and likely results from the increase in Cesarean deliveries and uterine curettages. (Kastner ES, Figueroa R. Emergency peripartum hysterectomy: experience at a community teaching hospital. *Obstet Gynecol* 2002;99:971–5)

Types
Accreta – attached to myometrium
Increta – invades myometrium
Percreta – penetrates myometrium

Treatment
Five procedures:
(1) Hysterectomy (most common treatment)
(2) Remove and oversew giving Pitocin and liberal use of antibiotics
(3) Localized resection
(4) Curettage, leaving *in situ*
(5) Methotrexate treatment

PLACENTA PREVIA

Definition

A placenta previa is a placenta implanted on the lower uterine segment that prevents descent of the fetus. The degree to which the internal cervical os is covered by the placenta determines whether a placenta previa is classified as marginal, partial or complete:

(1) Complete: implantation of the placenta across the cervical os
(2) Partial (incomplete): placenta covers part of internal os (or, for incomplete, the placental edge is within 2 cm of internal os but does not cover the os)
(3) Marginal (low-lying): placenta just reaches the edge of the internal os (or, for low-lying, the distance from the internal cervical os to the placental edge is between 2 and 3.5 cm)

Incidence

Per pregnancies	(@ 0.4%) or 1/200
Total/partial	30%/30%
What % persists until term?	10%
Placenta previa is known to have caused what % of death between 1979 and 2002?	7%
If asymptomatic until midtrimester, resolution may occur in	75%
If symptomatic until 24–36 weeks, resolution occurs in only	15%
Increased risk with:	
(1) Multips/AMA > 35	1/100
> 40	1/50
(2) Prior C-sections	
> 1 C-section	1%
> 2 C-sections	2%
> 3 C-sections	4%
(3) Defective decidualization	
Smokers	2 × increase
Cocaine abusers	
(4) Large placentas	
(5) Cretas associated	
What % of placenta previas also have cretas?	5%
Increased risk of creta if PP and prior C-section of	25%
What incidence of fetal congenital malformations are associated with placenta praevia?	2-fold
Fetal growth restriction (according to Varma TR. *Fetal* growth and placental function in patients with placenta previa. *J Obstet Gynaecol Br Commonw* 1973;80:311–15) is also increased with previa to	16%
Maternal mortality is	< 1%
Perinatal mortality is	< 10%
Incidence of PTD with placenta previa	50%
How much blood loss can occur before most patients become hemodynamically unstable?	25% or 1500 ml

Etiology

Zygote implants low

Symptoms

PAINLESS BLEEDING

Diagnosis

Ultrasound diagnosis per abdominal/transvaginal 70%/97%

Transvaginal ultrasound is superior to abdominal and concern for disruption of the placenta with the vaginal probe is unfounded according to multiple studies

Accurate diagnosis may be difficult if the uterus is contracting during US imaging

Also look for placenta accreta. In women with placenta previa, the risk of placenta accreta was 67% after 4 prior cesarean deliveries. Characteristics that are suggestive of placenta accreta include:

(1) Absence of the normal hypoechoic myometrial zone

(2) Presence of multiple lakes scattered throughout the placenta, creating a “Swiss cheese” appearance

(3) Focal disruption of the uterine serosa bladder wall

It is difficult prior to delivery to diagnose accreta, but color flow/power Doppler imaging with 2- and 3-dimensional techniques and MRI help improve the chances of diagnosis prior to delivery, but never a guarantee

"Migration" can occur in lies that are close 90%
In mild bleeding with marginal – head compression decreases bleed
Transfusion rate with placenta previa is 30%
Mean gestational age at the time of the first episode of bleeding is 29–32 wks
Some asymptomatic cases resolve, so do monthly evaluations with U/S

Differential

Bleeding complicates what % of pregnancies? 6%
Placenta previa comprises 7%
Placental abruption comprises 13%
Other causes (PTL, coitus, etc.) 80%
Marginal placenta previa lies within how many centimeters of the os? 2–3 cm
HALLMARK of PP is sudden onset of painless bleeding. How many asymptomatic till labor? 10%

Treatment

Restrict activity only after 30 weeks
Transfuse to a hematocrit of at least 30% in women actively bleeding
Maintain intensive observation, insert large-bore IV cath, CVP if unstable
$MgSO_4$ is agent of choice for tocolysis

Stat C-section:
Patient at term, in labor or with excessive bleeding regardless of age
Incision is usually transverse, but vertical p.r.n. if worried about association with fetal bleeding with anterior placenta previa
Risk of creta with anterior placenta is 4%
Risk of creta with anterior placenta and history of C-section 16–25%
If there is strong evidence of accrete or percreta at the time of delivery, leave the placenta in situ and perform hysterectomy

Outpatient treatment

If patient has not bled for 72 h, an acceptable alternative IF:
(1) Patient is reliable and compliant with medical advice
(2) Has adequate transportation to hospital
(3) Has the ability to access emergency services from home
(4) Lives within a reasonable distance from the hospital
(5) Rehospitalize women with recurrent vaginal bleeding during outpatient management.

Management

(1) Do not perform a pelvic examination until ultrasound report is available
(2) If a previa has been ruled out, the following steps should be taken:
    (a) Do speculum exam to rule out causes of bleeding such as cervicitis, polyps or cervical lesions
    (b) Look for other placental abnormalities such as placenta abruption
(3) If a placenta previa is diagnosed in second trimester, the following steps should be taken:
    (a) Start intravenous infusion of fluid with 18-gauge needle
    (b) Obtain a coagulation profile
    (c) Evaluate fetal viability, advanced labor or uncontrollable hemorrhage
        – If ultrasound examination shows no heart activity, consider termination. If fetus is alive, manage conservatively (if bleeding is mild to moderate)
        – If advanced labor or uncontrollable bleeding is present, proceed with C-section
(4) Do not do a double set-up examination unless ready to commit to delivery
(5) Use of tocolytic agents:
    (a) Use only when the uterus is contracting and/or vaginal bleeding is not sufficient to cause maternal hypotension
    (b) Do not use if blood replacement would be unable to keep up with blood loss or the patient is in active labor
(6) Provide patient with risk, benefits and alternatives regarding increased incidence of intrauterine growth restriction, need for adequate nutrition and cessation of smoking

(7) Repeat ultrasound examination at 35–36 weeks

If placenta previa is diagnosed at 35–36 weeks, the following steps should be taken:

(1) Complete previa
   (a) Determine fetal lung maturity (PG or L/S ratio) via ultrasound-guided amniocentesis
   (b) If fetal lungs mature, delivery by C-section
   (c) If fetal lungs immature, monitor weekly for maturity, then do C-section
(2) Marginal or partial previa
   (a) Do amniocentesis as above
   (b) If fetal lungs mature, consider two possible causes:
      – Double set-up when ready to commit to delivery
      – Follow with serial ultrasound to see whether placenta moves upward, as long as there is no further bleeding
   (c) If no longer a placenta previa on ultrasound, treat as a normal pregnancy

PLACENTAL SITE TUMORS

	Locally invasive secondary to cytotrophoblastic cells of placenta
Incidence	PST are rare but may be found after abortion, mole or normal IUP
Diagnosis	Increase levels of HPL and hCG
Treatment	Hysterectomy Not susceptible to chemotherapy

PLACENTAL TRANSPORT

Xenobiotics	Drugs and other chemicals	Simple diffusion
Glucose	(Down concentration gradient)	Facilitated diffusion
Amino acids	(Against concentration gradient)	Active transport
Active transport	Amino acids, calcium, phosphorus, iron	
Simple diffusion	Glucose, CO_2	
Facilitated diffusion	Glucose	
Endocytosis	IgG	
Does NOT cross placenta	TSH, IgM, T_3, T_4, thyroxine, insulin, prednisone	(TITT TIP)
CROSSES placenta	Propylthiouracil, iodine, TRH, LATS (long-acting thyroid stimulator), IgG and propranolol Remember the mnemonic	(PIT LIP)

POLYCYSTIC OVARY SYNDROME

Definition	An endocrine dysfunction in reproductive age women presenting with two or more of the following symptoms: (1) Menstrual dysfunction (2) Androgen excess (3) Polycystic ovaries (4) Insulin resistance (5) Infertility (6) Obesity
Suspect PCO	In any reproductive-aged woman presenting with menstrual irregularities combined with hirsutism, infertility, obesity or insulin resistance
Pathophysiology	Major endocrine manifestations are: (1) Chronic anovulation – occurs as result of ovarian dysfunction secondary to one or more of following: (a) Increased LH stimulation of the theca–stromal cell complex (b) Resulting increased ovarian androgen production interfering with normal follicular maturation

(c) Effect of peripheral insulin stimulation of thecal and stromal cells of the ovary
(d) Effect of increased somatotropin such as growth hormone and insulin-like growth factor-I (IGF-I) on gonadotropin stimulation of the ovary

(2) Hyperandrogenism – increased androgen production primarily from the ovary but also from the adrenal glands (this results from chronic LH stimulation of the theca and stromal compartments)
(3) Elevated LH – result of an increased LH pulse frequency of GnRH pulses from hypothalamus
(4) Hyperinsulinemia – like NIDDM with peripheral insulin resistance and pancreatic β-cell dysfunction resulting in altered glucose transporter systems resulting in defective insulin signaling mechanism

Incidence

Prevalence rate in reproductive-aged women is @ 5–10%

Genetics

Studies suggest an altered regulation of expression of the insulin gene or an inheritance as an autosomal dominant disorder with reduced penetration

Clinical symptoms

(1) Presenting symptoms
(a) Abnormal uterine bleeding – oligomenorrhea/amenorrhea
Oligomenorrhea 85–90%
Amenorrhea 30–40%
(b) Increased body hair – face, chest, abdomen (slow process) 80%
(c) Infertility 40%
(d) Obesity – upper-half body obesity 50%
(e) Polycystic ovaries – some women with true PCO do not have polycystic ovaries and some normal women have them

(2) Physical examination findings
(a) Hirsutism
(b) Obesity
(c) Polycystic ovaries observed with pelvic ultrasound

Diagnosis (lab testing)

(1) To rule out other pathologies or conditions

(a) Urine hCG (to rule out pregnancy)	CPT 81025
(b) TSH (to rule out hypothyroidism)	CPT 84443
(c) Prolactin (to rule out hyperprolactinemia)	CPT 84146
(d) 17-Hydroxyprogesterone (to rule out late-onset adrenal hyperplasia)	CPT 83498
(e) Pelvic ultrasound (to rule out ovarian tumors)	CPT 76856

(2) To substantiate changes compatible with PCOS

(a) LH	CPT 83002
(b) FSH	CPT 83001
(c) Testosterone, total	CPT 84403
(d) DHEA-S	CPT 82627
(e) Glucose (fasting)	CPT 82947
(f) Insulin, total (fasting)	CPT 83525

A fasting glucose to insulin ratio of < 4.5 is diagnostic of peripheral insulin resistance

Pearls

- Pulsatility of LH secretion can generally be overcome by drawing two samples at half-hour intervals and either combining the samples or averaging the individual results
- Free serum T may be more sensitive test; however, the clinical variance and increased cost make this assay less suitable than total
- DHEA-S is exclusively an androgen of adrenal origin and is reported to be elevated in over 50% of women with PCOS
- US finding of ten echo-free cysts from 2–8 mm size or an ovarian volume > 5.5 cm^2 is compatible with diagnosis of PCOS

Management

Must be directed toward several areas of care rather than just one. Direct care toward the woman's presenting complaint and concerns and the prevention of known major long-term complications of PCOS. *See* Treatment below

Diagnosis with hyperandrogenism

Increase in total or free testosterone and oligo-ovulation
Oligo-ovulation defined as cycle duration > 35 days
or less than how many cycles per year? 8

Differential

Exclude increased prolactin, thyroid dysfunction and/or androgen-secreting tumors, etc.
(Ovarian – Sertoli–Leydig or adrenal with Cushing's), late onset 21-OH deficiency – sexual ambiguity?)
Iatrogenic? Rapid progressive hirsutism/virilization – ovarian or adrenal tumor. Family history of androgen excess, short statue, mild virilization – suspect late-onset 21-hydroxylase deficiency

Features of polycystic ovarian syndrome are oligo-ovulation	> 90%
Hirsutism	75%
Polycystic ovaries	75%
Decreased SHBG	70%
Increased free testosterone	55%
LH/FSH ratio > 3	45%
Hyperprolactinemia	20%

Metabolic abnormalities of PCO

Insulin resistance, hyperlipidemia, increased free fatty acids, non-insulin-dependent diabetes mellitus, android obesity

Diabetes evaluation (WHO)

Normal FBS	< 115 mg/dl
2 h	< 140 mg/dl
Impaired GT FBS	< 140 mg/dl
Impaired GT 2 h	140–199 mg/dl
Diabetic FBS	> 139 mg/dl x 2
Diabetic 2 h	> 199 mg/dl

Waist/hip ratio

Waist measurement = smallest circumference between rib cage and iliac crest
Hip measurement = largest circumference between the waist and thighs

Android obesity	> 0.85
Gynoid obesity	< 0.75

More theories of genetics

What % of sisters develop PCOS?	50%
Paternal transmission	80%
Maternal transmission	35%

Suggest X-linked dominant or autosomal dominant transmission

Insulin resistance

(Increased abdominal circumference, acanthosis, hirsutism, etc.) can be treated with metformin in doses of 500–850 mg t.i.d.
Hyperinsulinemia is not diabetes but up to what % of PCOS patients will develop NIDDM? 40%
Must have glucose intolerance, can eventually develop insulin deficiency diabetes Type I
Patient needs to only lose what % weight to show marked improvement in insulin androgens and glucose levels? 5–7%

Ovarian anatomy of PCOS

Multiple immature follicles and theca cell hyperplasia
"Pearl necklace". Not all with PCOS have US findings
What % of normal women will have typical US of PCOS? 25%
What % of normal women on OCPs will have US findings typical of PCOS? 14%

PCOS labs

LH/FSH ≥ 3 : 1
Prolactin, TSH and T_4
If hirsutism + acne draw free testosterone, DHEA-S – increased but < 2 ng/ml and 8 ng/ml
If not withdrawing with a progesterone then draw estrogen level
If Ashkenazi Jew, then rule out 21-hydroxylase deficiency with 17-OHP
Check FBS and 2-h glucose. HbA1C should be < 7%

Treatment

(1) Obesity (diet and exercise)
BMI of this % restores regular menses and fertility 27%
BMI = body weight (kg) x height (m) squared
Recommend dietary intervention when ideal body weight > 20% as there is a statistically significant increased mortality
A BMI that similarly warrants intervention is that of 27.3
(Nutrition and maintenance of appropriate weight. In: Seltzer VL, Pearce WH, eds. Women's Primary Health Care. New York: McGraw-Hill, 1995:53)

MAIR-AN syndrome = extreme manifestation of hyperandrogenism and hyperinsulinism (rare). Triad is hyperandrogenism, insulin resistance and acanthosis nigricans

(2) Androgen excess (and excess body hair)
Finasteride and flutamide are teratogenic
Finasteride is a 5α-reductase inhibitor (Proscar®) 5 mg per day
Flutamide is an androgen-receptor competitor (Eulexin®) 50 mg b.i.d.

Spironolactone is an androgen-receptor competitor 50–100 mg b.i.d.
Diane (cyproterone acetate) dose is 100 mg/day
Give dexamethasone 0.25–0.5 mg hs with OCP if DHEA > 4 mg/ml
Give GnRH analogs with OCPs if other options fail
May combine electrolysis with medical treatment

(3) Hyperinsulinism
Metformin (glucophage) 500 mg t.i.d. to q.i.d.
Metformin is best tolerated if started as lower dose such as 500 mg or 850 mg daily with slow increase over several weeks to 1500–2000 mg dose. There is a remote risk of lactic acidosis so a renal function test is good idea prior to starting meformin so get serum creatinine
Rosiglitazone (also good for hyperinsulinism) 4 mg daily or 2 mg b.i.d. or 4 mg b.i.d.
Rosiglitazone is similar to troglitazone, which has caused hepatotoxicity; therefore get liver function test prior to starting it and every 2 months for 1 year and periodically thereafter (specifically alanine aminotransferase (ALT))
Glumetza – once daily. This is a metformin HCl extended release tablet. It eventually releases 2000 mg metformin daily.

Ovulation and subsequent pregnancy rates can be enhanced by administration of metformin in patients with PCOS and increased insulin resistance. (Heard MJ, Pierce A, Carson SA, *et al*. Pregnancies following use of metformin for ovulation induction in patients with PCOS. Fertil Steril 2002;77:669–73)

However, according to Moll et al. (*BMJ* 2006; 332:1485), there is no difference in ovulation rates between clomiphene citrate alone and clomiphene citrate and metformin. Therefore it might be wise to try clomiphene citrate alone in women with PCO then if no success – add multiple therapies

(4) Cardiovascular (diet, exercise, insulin p.r.n., anticholesterol drugs)
Ideal target for weight loss has been to approach level @ 15% of ideal body weight corrected for height and age

(5) Infertility (Clomid, FSH, Clomid + metformin, weight loss)
Laser drilling if Clomid fails – ovary cycles postop @ 90%
Clomid or Serophene® 50 mg x 5 days starting on cycle day 3 or 5
Rule out other causes such as obstructed fallopian tubes, abnormal semen analysis and presence of pelvic adhesive disease

(6) Menstrual disorders (progesterone withdrawal, OCPs, etc.)
Duration more important than dose – minimum Provera is 2.5 x 12
There is an increased risk of endometrial hyperplasia and cancer. Use cyclic or continuous progestins or use oral contraceptives
(a) Provera (MPA) – 5–10 mg daily for 10–14 days per month
(b) Aygestin (norethindrone acetate) – 5–10 mg daily x 10–14 days
(c) Micronor (norethindrone) – 0.35 mg daily throughout the month
(d) Depo-Provera (MPA) – 150 mg IM every 3 months
(e) Lupron (GnRHa) – 3.75 mg IM monthly or 11.25 mg IM every 3 months

This method suppresses unopposed ovarian estrogen as well as reducing androgen production. In cases of severe abnormal bleeding, this approach may be useful for extended periods of time along with the use of hormone add-back therapy
Remember, long-term use of GnRHa alone is associated with:
- Loss of trabecular bone
- Defects in CNS such as memory loss and defects in thought processing
- Cardiovascular defects including heart attacks
- Quality of life issues such as hot flushes, mood changes and sleep disturbances

Add-back therapy`

Add-back therapies can include:
(1) PremPro – 0.625/2.5 mg or 0.625/5 mg daily or
(2) FemHrt – 1 mg/5 μg daily or
(3) Progestins already listed above
Remember that Lupron is not considered the standard regime for PCOS because of cost. It is reserved for those who do not respond to usual therapies

Key points

Women with PCOS and insulin resistance are at increased risk for impaired glucose tolerance or diabetes. Hypoglycemic agents can reduce circulating androgen levels, increase sex hormone binding globulin, facilitate weight loss, and induce ovulation

Take steps to enhance or induce ovulation. Even women who do not desire fertility stand to gain, because chronic anovulation increases the risk of endometrial cancer

Address hirsutism and other hyperandrogenic effects. Treatment of hirsutism is best approached with a combination of medical and mechanical means. Counsel patients that response is likely to be slow and subtle

POLYHYDRAMNIOS

Associated with diabetes	25%
Associated with congenital malformations	20%
Associated with twins	8%

Definition

Significantly increased risks that pregnancy will be complicated by:
(1) Maternal diabetes
(2) Fetal anomaly
(3) PTL
(4) PROM
(5) Multiple pregnancies

Acute

Late second or third trimester; poor prognosis; 7:12 perinatal deaths

Chronic

Slow, early onset; better prognosis. Linked to maternal glucose intolerance, macrosomia, fetal anomalies

Physiology

Placenta then fetal urine excretion produces fluid. Fetal small bowel/diffusion through amnion/chorion absorbs. Most polyhydramnios is thought to be due to increased fetal urine production

Diagnosis

`

Dye methods (inject/draw out dilution) 8% accurate

TIUV

AFI – each quadrant, largest pocket measured in vertical axis. Sum of largest pockets in all four quadrants is AFI. 95th percent of amniotic fluid index during the third trimester is 25 cm

(1) Observe weight gain
(2) Compare fundal height changes
(3) Palpate abdomen
(4) Perform ballottement for fetal parts
(5) Perform ultrasound – confirm polyhydramnios, detect multiple gestation and obvious structural congenital malformation
    (a) BPD ventricle-to-hemisphere ratio
    HC vertebral column
    (b) Evaluate heart and chest cavity
    (c) Examine abdomen for ascites, abdominal masses, gastrointestinal atresia, abdominal wall masses, ompholocele or gastroschisis
    (d) Urinary system (kidneys, ureters and bladder filling)
    (e) Evaluate skeletal system
    (f) Evaluate placenta
(6) Do 3-h GTT
(7) Coombs' test (screen for irregular antibodies with indirect antiglobulin test)
(8) Amnio for karyotype analysis

Etiology

Idiopathic	60%
Diabetes mellitus	19%
Multiple gestation (twin–twin syndrome)	7.5%
Blood group incompatibility	5%
Congenital malformation	8.5%

Management

38 weeks – PG/LS ratio
Bed-rest
High-protein diet
Monitor serum proteins and use amnio to aspirate for SOB
Watch for CHF or IUGR
Sedation
No diuretics – little effect of TV of AF, may be harmful
Indomethacin (investigational) 50–100 mg p.o. t.i.d.–q.i.d.
Decreased AF production by decreasing fetal urine production
Dis: Premature closure of ductus arteriosus
Fetal pulmonary hypertension
Tricuspid insufficiency

Steps in delivery

(1) Obtain fetal maturity studies – ultrasound, BPD, FL, head and abdominal circumferences, fetal lung maturity studies
(2) Before induction – amnio p.r.n. to dec AF
(3) Type and screen mother's blood
(4) Baseline coagulation studies: platelets, CBC, fibrinogen
(5) Controlled amniotomy with slow release of AF
(6) Observe for placental separation
(7) Observe for postpartum hemorrhage

Acute polyhydramnios (24–27 weeks)

(1) Erythroblastosis fetalis ? Rx
(2) Congenital malformations – term of pregnancy
(3) If no cause – therapeutic amniocentesis
500–1000 ml of AF

Tocolytics – $MgSO_4$ (3–5 g bolus over 30 min and then 2–5 g/h concentration 4–8 mg/dl), Brethine®, NSAIDs
(indomethacin 50–100 mg p.o. t.i.d.–q.i.d.)

Locate placenta

Hypoproteinemia – will develop; increase protein diet

Albumin IV p.r.n.

No diuretics

Antibiotics are contraindicated – can conceal early amnionitis

POLYMYALGIA RHEUMATICA

How much more common in women?	2 x
Usually how often > age of 50?	1/750
Lasts	2–7 years
Morning stiffness longer than	30 min

Increased sed rate, fever, weight loss, fatigue, depression. Similar to Lyme disease but sero test is negative
Treatment is prednisone 15 mg daily using sed rate as a guide

POLYPS

What % of polyps do not respond to progesterone?	66%
What % undergo histologic change?	33%
What % undergo malignant change?	0.5%
What % of women who show abnormal bleeding have polyps?	25%
What % of polyps are solitary?	80%
What % are multiple?	20%

The chromosome in stromal cells of polyps is	6p21
Hysteroscopy is the treatment of choice because curettage removes only this % of polyps	25%

POST-COITAL TEST

Perform after how many hours abstinence?	48
Examine cervical mucus within how many hours after coitus?	2–8
Examine mucus how many hours prior to estimated ovulation in order to assess optimal mucus?	24–48
Normal findings are to see how many progressively motile sperm per HPF in clear, acellular mucus?	5–10
How long should the spinnbarkeit be?	> 8 cm
Failure to penetrate at least what % of the hamster ova of a sperm penetration assay suggests an impairment of male fertility?	10%

POST-DATE PREGNANCY

Definition

True incidence difficult to ascertain as most early studies relied on menstrual dating	
Pregnancy exceeding 42 weeks post-FDLMP or past how many days past FDLMP?	294
Incidence originally	4–14%
So incidence with early menstrual dating of	7.6%
Decreased to what % by early ultrasound to	2.6%
Further decreased to what % when both ultrasound *AND* menstrual dating included for diagnosis	1.1%

Incidence

	9%
Meconium stain	25%
Macrosomia	20%
Dysmaturity	20%
Oligohydramnios	15% (AFI ≤ 5 cm)
Perinatal mortality rates double by how many weeks?	43
Increases 4–6-fold by how many weeks?	44

Complications

(1) Postmaturity
- Placenta maximally developed at 37 weeks
- May decrease in surface area/function after 37 weeks
- Increased IUFD rates after 42 weeks

(2) Meconium
- 25–30% of pregnancies ≥ 42 weeks
- Tends to be thicker secondary decreased AFV
- Increased risk of meconium aspiration syndrome

(3) Oligohydramnios
- Peak AFV @ 37 weeks (~1000 cc)
- Decreases to average 250 cc by 42 weeks
- Increased incidence of cord compression/acute hypoxia

(4) Macrosomia
- > 4500 g, occurs in 2.5–10% at ≥ 42 weeks
- Increased risk of maternal/fetal trauma
- Increased risk of shoulder dystocia

Fetal complications

Meconium stain incidence with post-dates	25–30%
Macrosomia	20%
Dysmaturity	20%
Oligohydramnios (AFI ≤ 5 cm)	15%
Peak AFV at how many weeks?	36
Declines to what volume by 40 weeks?	800 cc

Maternal complications

C-section rate due to macrosomia and fetal distress increases to how many times normal rate?	3–4 x

Diagnosis

Most commonly an error in dating – Naegele's rule or quickening	16–20 weeks
Fetal stethoscope	18–20 weeks
At 20 weeks, the fundus should be at the umbilicus at	20 cm
Early exam should be consistent with dates	

Correct assessment of gestational age
Accuracy indirectly proportional to gestational age at time of assessment ('the earlier, the better')
Document:
(1) Regularity, length, date of last menses
(2) Uterine size: first trimester/20 weeks at umbilicus
(3) Date of first fetal movement (quickening) 16–20 weeks
(4) Fetal heart rate detection (Doppler) 10–12 weeks
(5) US dating: first trimester – CRL (error ± 3–5 days)
Second trimester – BPD, HC, FL (error ± 7–10 days)
Dating: – known LMP most accurate – Naegele's rule
First day of LMP – 3 months + 7 days = EDC
Known date of conception – using pregnancy wheel at 2 weeks
If LMP unsure – early US

Etiology

Multifactorial neuronal and hormonal processes including fetal brain, pituitary gland, adrenal gland hypoplasia, placenta, fetal membranes/decidua which is rich in $PGF_{2\alpha}$
Amnion rich in PGE_2
Chorion rich in 15-hydroxyprostaglandin dehydrogenase or PGDH
Can be secondary to anencephaly, fetal adrenal hypoplasia, abdominal pregnancy, placental sulfatase deficiency (all of these have decreased estrogen production)

Treatment

Induce if Bishop score	> 7
Otherwise NSTs, BPP or modified BPP (NST + AFI)	semiweekly
NST has false-negative rate with weekly testing	3.2/1000
NST has false-negative rate with twice weekly testing	1.9/1000
CST has false-negative rate	0.71/1000
AFI oligo	< 5
borderline	5–8
normal	> 8
Antenatal surveillance should begin by	42 weeks

No single protocol appears superior. No evidence that monitoring improves outcome. Unknown whether expectant rx vs induction is better although recent research indicates that morbidity and mortality associated with expectant management is greater than previously appreciated
ACOG guidelines – induce low-risk pregnancy at 43rd week

Surveillance strategy

(1) NST/AFI
Reactive + AFI > 5 = continue surveillance
Reactive + AFI < 5 = biophysical profile or cervical ripening
Non-reactive and/or significant decelerations – cervical ripening
(2) Biophysical profile (optional)
> 6 – continue surveillance
≤ 6 – cervical ripening

Cervical ripening

(1) Prostaglandin E_2 gel 0.5 mg vs. vaginal suppository 2.5 mg q. 4 (or Cytotec 50 μg/25 μg)
Check cervix before each dose:
Favorable – labor induction/augmentation
Unfavorable – repeat prostaglandin application
(2) Oxytocin – low-dose cervical ripening at l–2 mu/mtn

Intrapartum management

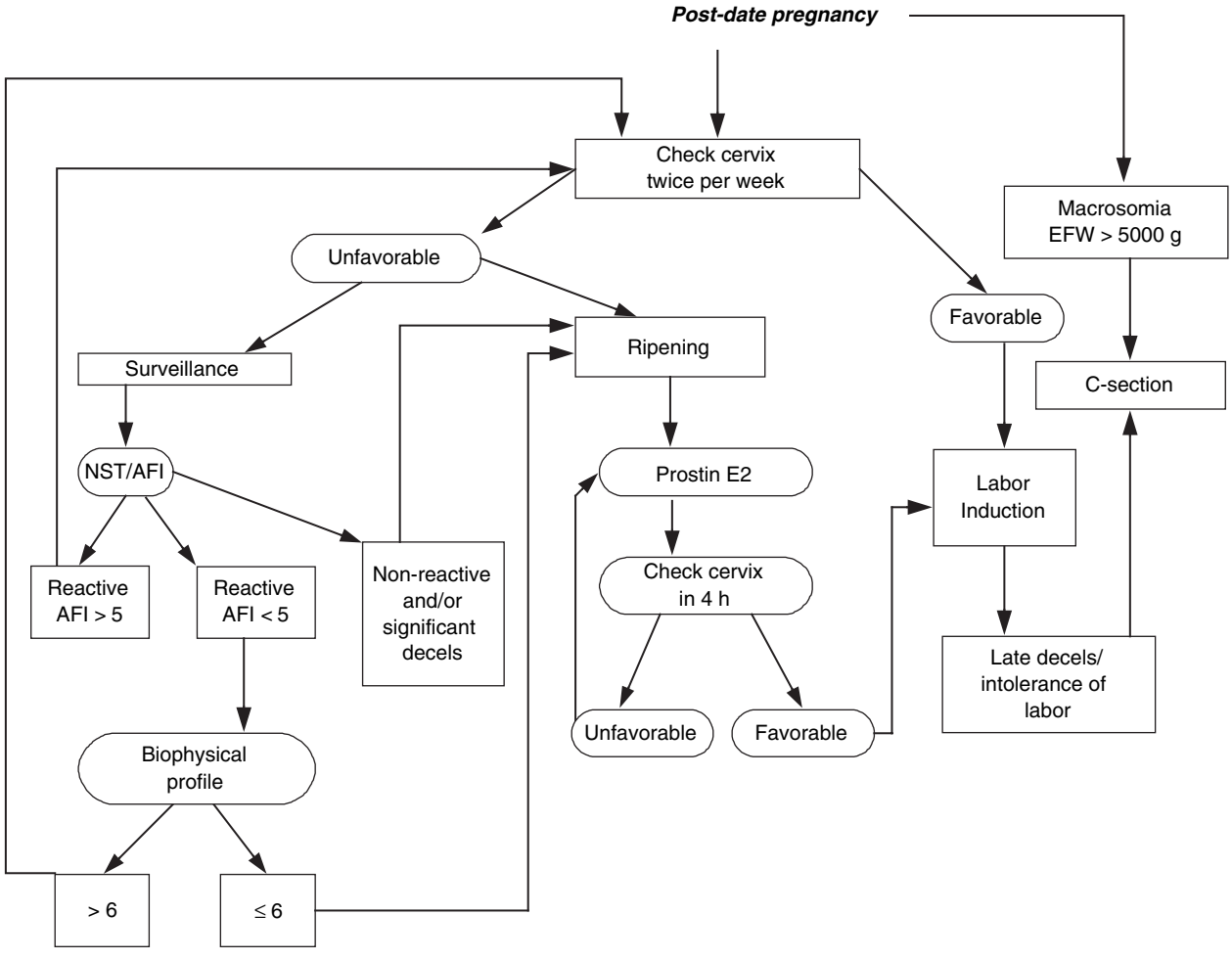
(1) Continuous EFM
Persistent late decels/fetal intolerance of labor – Cesarean delivery
Frequent variables – consider amnioinfusion
(2) Suspect macrosomia
Avoid midpelvic operative delivery
EFW > 5000 g – consider C-section
(3) Determine presence of meconium
Consider amnioinfusion
Aggressive suctioning of infant on delivery of head (wall suction)

Prolonged gestation

Percent shoulder dystocia:	
Normal pt with 4000 g infant	10%
Diabetic pt with 4000 g infant	20%
Normal pt with 4500 g infant	25%
Diabetic pt with 4500 g infant	50%

Shoulder dystocia can cause Erbs palsy which is injury to nerve roots	C5–6
or Klumpke's palsy which involves	C8–T1
Shoulder dystocia is defined as a delay in the delivery of the body after delivery of the head	> 60 s
How many seconds do you have to deliver the body after the head without compromise?	150 s
That is how many minutes?	2½

- *Post-term gestation criteria*
  It would be reasonable to follow after 42 weeks with BPP or other
  However, one could justify induction as a reasonable alternative if the cervix was favorable or there were other mitigating circumstances



POSTMENOPAUSAL BLEEDING

Endometrial atrophy	60–80%
Endometrial polyps	2–12%
Estrogen therapy (unopposed)	8 x increased incidence
Endometrial hyperplasia	5–10%
(Obesity, exogenous estrogen, estrogen-secreting tumor (ovary))	
Endometrial carcinoma	10%
ENDOMETRIAL BIOPSY !!!!!!!	90–98% accurate
Using transvaginal ultrasound, endometrial thickness on both sides would be rare to be under what measurement with endometrial hyperplasia?	< 4 mm
How accurate are Pap smears in diagnosing endometrial cancer?	30–50%

POSTMENOPAUSAL MASS

Incidence of malignant ovarian neoplasm	10%
Low-risk patients can be treated with laparoscopy – what size mass would define low risk?	≤ 5 cm

Otherwise plan laparotomy with vertical incision, washings, exploration and oophorectomy

POSTOPERATIVE NAUSEA

- Zofran 8 mg ODT or 4 mg IV pre-op, in Recovery Room, then q. 6 h
- Treatment with what amount of oxygen will decrease postop nausea? 80%

What % does 80% compared to 30% O_2 decrease postop nausea in the first 24 h? 17–30%

(Greif R, Laciny S, Rapf B, *et al*. Supplemental oxygen reduces the incidence of postoperative nausea and vomiting. *Anesthesiology* 1999;91:1246–52)

POSTPARTUM DEPRESSION

See also Psychiatric

Symptoms

Dysphoric mood
Loss of interest in usually pleasurable activities
Difficulty concentrating or making decisions
Psychomotor agitation or restriction
Fatigue
Changes in appetite or sleep
Recurrent thoughts of death/suicide
Feelings of worthlessness or guilt, especially failure at motherhood
Excessive anxiety over child's health

Dose ranges and side-effect profiles of antidepressants commonly used to treat postpartum depression

Drug	Therapeutic range (mg/day)	Side-effects[1]			
		Anticholinergic[2]	*Orthostatic hypotension*	*Arrhythmia*	*Weight gain (> 6 kg)*
TRICYCLICS					
Amitriptyline (Elavil)	75–300	4+	4+	3+	4+
Desipramine (Norpramin)	75–300	1+	2+	2+	1+
Imipramine (Tofranil)	75–300	3+	4+	3+	3+
Nortriptyline (Pamelor)	40–200	1+	2+	2+	1+
SSRIs					
Fluoxetine (Prozac or Sarafem)	10–40	0	0	0	0
Paroxetine (Paxil)	20–50	0	0	0	0
Sertraline (Zoloft)	50–150	0	0	0	0
Citalopram (Celexa)	20–40	+	+	+	+/–
Escitalopram (Lexapro)	10–20	+	+	+	+/–
SSNRIs					
Venlafaxine (Effexor)	75–150	0	0	+	0
"NATURAL" REMEDIES					
St John's Wort (Hypericum perforatum)	300–1450	2+	0	0	0

[1]0 = Absent or rare, 4+ = relatively common
[2]Dry mouth, blurred vision, urinary hesitancy, constipation, drowsiness
St John's Wort can also cause skin reactions, including photosensitization, rash, and itching; gastrointestinal problems; fatigue; restlessness, headaches; dizziness; and dry mouth (as indicated by the +). St John's Wort has been shown to be effective in short-term mild depression but further studies are needed in regard to more serious long - term and severe depression

POSTPARTUM HEMORRHAGE

If Pitocin and methergine do not arrest PPH, then Hemabate® (15-methyl-$PGF_{2\alpha}$) can be dosed 0.25 mg q. 1–2 h

(1) Early – within first 24 h after delivery
  – Uterine atony – caused by overdistention, protracted labor, macrosomia, increased parity, chorioamnionitis
  – Retained placental fragments
  – Lacerations, uterine inversion, uterine rupture, coagulopathy
(2) Late – after the first 24 h but prior to 6 weeks postpartum
  – Subinvolution, infection, retained products of conception

Management

(1) Determine etiology
(2) Volume replacement
(3) Vital signs and urinary output
(4) Check labs to rule out coagulopathy (PT, PTT, platelet count, fibrinogen level)

Treat cause with

(1) Medical treatment
  (a) Pitocin, methergine, Hemabate (0.25 mg q. 15–60 min p.r.n.)
  (b) Antibiotics
  (c) RL 3 : 1 (1–2 large bore IV lines)
  (d) Whole blood, PRBCs, FFP, platelets, cryo
(2) Surgical treatment
  (a) Ligation of uterine artery
  (b) Hypogastric artery ligation
  (c) Hysterectomy – this is quickest and safest
  (d) Curettage (especially for late bleeding)
(3) Invasive radiology – if time, uterine artery embolization

PRECIPITATE LABOR

	Incidence		2% births in the USA
Definition	Labor to delivery in less than		3 h
	Short labor defined –	for nulliparous is dilation	≥ 5 cm/h
		for multiparous is dilation	≥ 10 cm/h
Associated with	Short labor is associated with abruption @		20%
	Also associated with meconium, postpartum hemorrhage, cocaine abuse and low Apgars		
Etiology	(1) Decrease resistance of soft parts of birth canal (2) Strong uterine and abdominal contractions (3) Absence of painful sensations (rare)		
Effects	*Maternal* (1) Uterine rupture (2) Lacerations (3) AFE (4) Hemorrhage		
	Fetal (1) Decreased uterine blood flow and fetal oxygen (2) Head trauma (3) Increased meconium (4) Decreased Apgar scores		
Treatment	Stop any oxytoxic agents		

PRECOCIOUS PUBERTY

Definition	Signs of secondary sexual maturation at an age 3 standard deviations below the mean for that population. In North America, this would be secondary sex characteristics before age 8 or menarche before age 9
Evaluation	The two primary concerns of the parents are: (1) The social stigma ('different from peers') (2) Decreased height due to premature closure of epiphyseal growth centers Subdivided into two classifications: (1) GnRH-dependent (complete, true, isosexual, central) – premature maturation of the hypothalamic–pituitary–ovarian axis. Usually the etiology is unknown. Is the most common (2) GnRH-independent (incomplete, pseudo, isosexual or heterosexual, peripheral) – independent of hypothalamic–pituitary control. The most common cause is an estrogen-secreting ovarian tumor (60% are granulosa cell tumors). McCune–Albright syndrome is a rare triad of café-au-lait spots, fibrous dysplasia and cysts of the skull and long bones
Differential diagnosis	75% of precocity in girls is idiopathic Is very important to rule out a serious disease in the CNS, ovary and adrenal gland
Diagnostic work-up	History and physical – must rule out life-threatening neoplasms of the ovary, adrenal and CNS Record height, weight and Tanner stages Brain imaging studies (CT and/or MRI) Serum estradiol, FSH, LH, TSH, triiodothyronine, thyroxine, prolactin, testosterone, DHEA or DHEA-S, hCG Bone age by hand-wrist films every 6 months to establish the rate of skeletal maturation Abdominal ultrasound and/or CT to evaluate ovarian, uterine or adrenal gland enlargement

Laboratory findings in disorders producing precocious puberty

	Gonadal size	*Basal FSH/LH*	*Estradiol or testosterone*	*DHEA-S*	*GnRH response*
Idiopathic	Increased	Increased	Increased	Increased	Pubertal
Cerebral	Increased	Increased	Increased	Increased	Pubertal
Gonadal	Unilater. incr.	Decreased	Increased	Increased	Flat
Albright	Increased	Decreased	Increased	Increased	Flat
Adrenal	Small	Decreased	Increased	Increased	Flat

From Speroff L, Glass RH, Kasc NG. *Clinical Gynecologic Endocrinology and Infertility*, 5th edn. Baltimore: Williams & Wilkins, 1994:375

Treatment

Depends on the cause, extent and progression of precocious signs and whether the cause can be removed operatively

Definitely treat:
(1) Girls with menarche before age 8
(2) Progressive thelarche and pubarche
(3) Bone age over 2 years greater than their chronologic age

The drug of choice for GnRH-dependent precocious puberty is GnRH agonists
Maintain therapy until the median age of puberty. The drug of choice for McCune–Albright syndrome is testolactone. Both child and her family need intensive counseling

PREGNANCY

Presumptive evidence

Nausea with or without vomiting
Urinary symptoms
Fatigue
Perception of fetal movement
Signs – cessation of menses, cervical mucus (ferning), breast changes, Chadwicks's sign (bluish vagina), skin pigmentation

Probable evidence

Enlargement of abdomen
Change in size, shape and consistency of uterus
Changes in the cervix (Hegar's sign – softening of the cervix)
Braxton Hick's contractions
Ballottement
Outlining the fetus
Pregnancy test positive

Positive signs of pregnancy

+ Fetal heart rate
+ Fetal movements per examiner
+ Ultrasound recognition of pregnancy
+ X-ray of fetus

Increases in pregnancy (partial list)

Fibrinogen increases	50%
GFR increases	50%
O_2 consumption increases	25%
Total thyroxin concentration	
Thyroid-binding globulin concentration	

No change

TSH
Free thyroxine

Decreases

DHEA-S, motilin, factors 11 and 13, H&H (< 11 abnl), cortisol, platelets, arterial pressure and vascular resistance

PREGNANCY-INDUCED HYPERTENSION (PIH)

	Retention of blood vessel wall musculature (due to failure of secondary wave of invasion of cytotrophoblasts into myometrial portion of spiral arteries) → reduced uterine-placental perfusion	
	(1) Increased ratio of serum thromboxane to prostacyclin	
	(2) Increased serum concentration of endothelin	
	(3) Increased serum concentration of glutathione	
Severe disease is defined by	B/P systolic	> 160
	diastolic	> 110
	Proteinuria	> 5 g/24 h
	Oliguria	< 500 ml/24 h
	Pulmonary edema and microangiopathic hemolysis. Acute onset of renal failure. Increase in serum creatinine. Grand mal seizures. Eclampsia. HELLP syndrome	
	Thrombocytopenia (most frequent)	< 100 000/ml
	Symptoms suggesting end-organ involvement – visual disturbances, headaches or RUQ painIUGR or oligohydramnios	
	PIH with DIC is diagnosed with thrombocytopenia	< 100 000
	Low fibrinogen levels	< 300 mg/dl
	Fibrin split products	> 40 mg/ml
	HELLP syndrome is usually antecedent to DIC in what % of abruption or hemorrhage?	21–38%
	What % subcapsular liver hematomas?	100%
	Patients with PIH DO NOT have blunted pressor response to infused angiotensin	II
	Plasma volume contraction depending on severity and duration caused the hematocrit to	increase
	Intravascular volume does what if PIH present despite increase total body water?	Decreases
	Antithrombin III levels are what in PIH?	Decreased
	B/P is WNL initially with HELLP syndrome in what % patients?	10–20%
	Increased liver enzymes are probably secondary to vasospasm and ischemia of PIH	
	Does hyperreflexia correlate with the severity of PIH?	No
Risk factors for PIH	Chronic renal disease	20 : 1
	Angiotensinogen gene *T235* (homozygous)	20 : 1
	Chronic hypertension	10 : 1
	Antiphospholipid syndrome	10 : 1
	Twin gestation	4 : 1
	Angiotensinogen gene *T235* (heterozygous)	4 : 1
	Nulliparity	3 : 1
	Age > 40 years	3 : 1
	Diabetes	2 : 1
	Black race	1.5 : 1
Treatment	Magnesium sulfate levels – therapeutic levels	4–7 mEq/l
	Loss of patellar reflex	8–10 mEq/l
	Respiratory depression	10–12 mEq/l
	Respiratory arrest	≥ 12 mEq/l
	Cardiac arrest	> 25 mEq/l
	Loading dose of $MgSO_4$ is how many grams? (60 ml of 10% $MgSO_4$ in RL or D5NS)	4–6 g
	Over how many minutes?	15–20 min
	IM loading dose is	10 g
	Maintenance dose is	2 g/h
	IM maintenance dose is	5 g q. 4 h
	(40 g $MgSO_4$ to 1 liter of D5 0.9 NS or RL at 50 ml/h)	
	Avoid $MgSO_4$ intoxication – ensure prior to each dose that:	
	(1) Urine flow is at least 100 ml/4 h	
	(2) Patellar reflex is present	
	(3) No respiratory depression	
	Alternative to $MgSO_4$ is phenytoin in loading dose of	1000 mg

Then give (10 h later) 500 mg
How many hours later? 10
While on $MgSO_4$, deep tendon reflexes and vital signs should be checked hourly
Intake and output should be checked every 2–4 h
I&Os should be at least > 100 ml/4 h
To reverse toxic effects of $MgSO_4$, give Ca^+ gluconate 1 g
(Ca^+ gluconate 10 ml of 10%) slow IV over 2–3 min
Provide O_2, intubation and mechanical ventilation

Key points;
(1) Give $MgSO_4$ at the time of diagnosis to all preeclamptic patients who are to be delivered
(2) Administration of $MgSO_4$ for new-onset hypertension and preeclampsia remote from term is controversial
(3) Even with therapeutic serum concentrations of magnesium, convulsions are possible
(4) $MgSO_4$ should be administered for 24 h after delivery or after the last postpartum seizure
(5) Safe administration requires vigilant monitoring of reflexes, respiratory status, and urine output

CONTRAINDICATIONS to $MgSO_4$:
Myasthenia gravis
Hypocalcemia
Renal or heart disease
Ca^+ channel blockers
Known allergies

If $MgSO_4$ is unsuccessful in treatment of eclampsia, give sodium amobarbital IV in dose of 250 mg and continue for 24 h after delivery – limiting IVFs
Hydralazine HCl IV is given in bolus of what dosage every 20 min p.r.n. increased B/P? 5–10 mg
Labetalol IV every 10 min can be given as an alternative in dosage of 20 mg
To a maximum dose of 300 mg
Instead of hydralazine HCl
ASA is option for high-risk patients – not normotensive patients in dose of 60–80 mg
Ca gluconate 2 g/day possibly decreases risk of PIH by 3 x
Vaginal delivery is generally preferable to C-section (even in patients with manifestations of severe disease)
Epidural anesthesia (pre-load) – had no thrombocytopenia
Improvement in intervillous flow
Ca^+ channel blockers can be used in POSTPARTUM
Most frequent finding for criteria for severe PIH is thrombocytopenia with platelets < 100 000/µl
Thromboxanes – cause further platelet activation, aggregation and vasospasm
Prostacyclins – produced along endothelial cells lining blood vessels, prevent platelet aggregation and dilate blood vessels
In PIH → there is increased thromboxane to decreased prostacyclin
ASA (81 mg) – disables platelet thromboxane-producing machinery for 1 week lifespan of platelet

PREGNANCY TUMOR

Represents benign outgrowth of palatal plate
Recurrence is common
Massive hemorrhage can occur secondary to trauma of this tumor
Surgical removal is treatment of choice for this condition

PREMARIN

Name some of the ingredients of premarin:
Estrone, 17α-estradiol,

Equilin, 17α-dihydroequilin,
Equilenin, 17α-dihydroequilinin

PREMATURE OVARIAN FAILURE

Definition

Menopause is considered premature if it occurs before age 40
Normal menopause occurs (on the average) at about the age of 51
POF is when a woman has stopped menstruating for 3 months or more and has high levels of FSH and LH and low levels of estrogen

Diagnosis

Diagnostic rise in FSH to > 40 mIU/ml
new assays >30 mIU/ml

Causes

(1) Autoimmune disease (lupus, RA, liver conditions) 20–55%
(2) Autoimmune adrenal disease (Addison's) 10–20%
LIFE-THREATENING IF NOT TREATED
(3) Hypothyroidism, IDDM, ITP, galactosemia, X-chromosomal abnormalities (Turner's syndrome), radiation or chemotherapy
(4) Surgical menopause without hormonal replacement (this is usually very severe)

Risks

(1) Coronary heart disease (CHD) 2.2 times more likely to develop
(2) Osteoporosis
(3) Sexual dysfunction

Treatmen

Consider genetic evaluation if POF < age of 40 (not related to surgery)
Hormone replacement therapy – possibly androgen supplementation

PREMATURE RUPTURE OF MEMBRANES (PROM)

What % of patients begin labor with PROM within 24 h? 90%
What % of pts with PPROM (< 37 weeks) begin labor within 24 h? 70%
within 72 h? 90%

Etiology

Infection? Weakening of membranes?

Diagnosis and management
(1) Sterile speculum examination (rule out prolapsed cord)
  (a) pH (nitrazine is blue – alkaline) and check for *fern*ing
  (b) Cervical check for dilation and/or fluid coming from os
  (c) Obtain cultures (streptococcus, *Chlamydia*, BV, and/or gonorrhea)
  (d) AVOID DIGITAL EXAMINATION
(2) History alone – correct diagnosis PROM > 90%
(3) Ultrasound
  (a) Sometimes confirms diagnosis
  (b) Confirms presenting part
  (c) Assesses gestational age
(4) Monitor
  (a) Rule out or in labor
  (b) Evaluate possible non-reassuring fetal status

Expectant management

In hospitalization
– Deliver if develops chorioamnionitis
– Deliver if non-reassuring fetal status
– Deliver if PTL or check pulmonary maturity? Induce p.r.n.

Evaluation of chorioamnionitis

(1) Clinical symptoms – vital signs, uterine tenderness, odor of lochia
(2) Lab tests – increased WBC, increased C-reactive protein concentration → interpret labs with caution
(3) Amniocentesis – Gram stain, culture of fluid
(4) Ultrasound – fetal age, fetal lie, presence or absence of oligo
(5) BPP – perform daily and if non-reassuring → increase risk infection

Treatment of chorioamnionitis
(1) If + cultures, treat with antibiotics
(2) Prophylaxis with antibiotics if PPROM (prolongs pregnancy and decreases morbidity < 25 weeks with 40% survival)
(3) Fetal monitoring continues then daily, transfer p.r.n.

Treatment

Delivery is the treatment of choice for PROM when gestation is > 36 weeks
Prolongation of pregnancy if no labor, infection or cord compression per FHR is treatment for 26–35 weeks
If the pregnancy is less than 25 weeks with PROM, the survival is only 40%
Steroids should be given if gestation < 32 weeks
Steroids can be given with PROM up to 34 weeks
Antibiotics to improve perinatal outcome should be given if protocols to be pursued < 35 weeks
Steroids given when < 32 weeks' gestation to reduce RDS, IVH, NE and death

Manage conservatively < 30–32 weeks if no maternal or fetal contraindications exist
- Tocolysis okay to permit steroids and antibiotics time

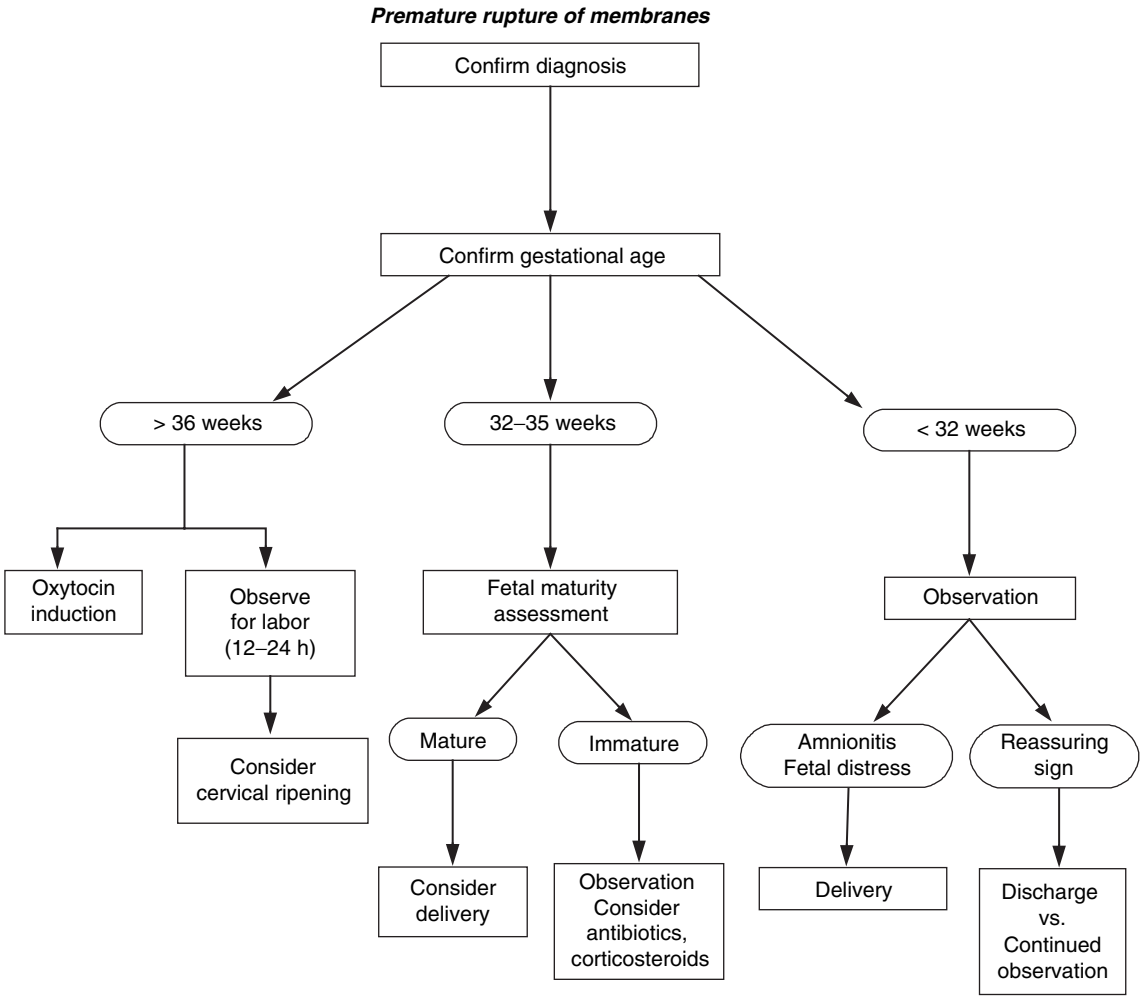
If the gestational age is less than 32 weeks and the mother and the fetus are stable, expectant management is appropriate. However, if the fetal presentation is unstable or the fetal heart rate tracing is worrisome, the patient should be delivered

AVOID DIGITAL EXAMS

Fetal fibronectin (FFN) normally increases < 20 weeks in cervicovaginal secretion = + if > 50 mg/dl after 23–24 weeks
False positives if sexual activity within 24 h, recent cervical exam and/or vaginal bleeding
FFN compares with transvaginal US assessment of cervical length

Preterm PROM

Review of work-up
Monitor and ultrasound (oligo? lie? position? presentation?)
Sterile speculum exam
Nitrazine for pH
Ferning and wet mount for BV
Culture for GBBS, GC and *Chlamydia*
AVOID DIGITAL EXAMS


Premature rupture of membranes
Confirm diagnosis
Confirm gestational age
> 36 weeks
32–35 weeks
< 32 weeks
Oxytocin induction
Observe for labor (12–24 h)
Consider cervical ripening
Fetal maturity assessment
Mature
Immature
Consider delivery
Observation Consider antibiotics, corticosteroids
Observation
Amnionitis Fetal distress
Reassuring sign
Delivery
Discharge vs. Continued observation

PREMENSTRUAL SYNDROME (PMS)

PMS may be defined as the cy=clic recurrence, in the luteal phase of the menstrual cycle, of a combination of distressing physical, psychological and/or behavioral changes of sufficient severity to result in deterioration of interpersonal relationships and/or interference with normal activities

The symptoms of PMS must appear during the luteal phase, which begins with ovulation, and diminish greatly or disappear with the onset of menstruation or shortly afterward. A woman who has symptoms throughout the cycle does not have PMS

Diagnosis

The diagnosis of PMS is made with at least 2 months of documented ovulation and concurrent record-keeping of symptomatology disrupting lifestyle during the luteal phase. The symptom diary should be recorded daily throughout the month

Ask the patient to list three to five symptoms that bother her the most and enter these on a daily symptom checklist for PMS. Have the patient track these symptoms for two menstrual cycles and bring the checklist back to you

PMS symptoms

Tension	Hypoglycemic episodes
Anxiety	Increased appetite
Mood swings	Headaches
Irritability	Sweet cravings
Depression	Weight gain
Confusion	Abdominal bloating
Crying	Breast tenderness
Forgetfulness	Swelling of extremities

Evaluation for PMS should include a history, physical exam and possibly laboratory studies. The history should elicit risk factors that correlate with PMS, sources of stress, medical or psychiatric problems and physical substance/alcohol or sexual abuse.

The differential diagnosis of PMS includes molimina, situational stress disorders and chronic affective disorders

Molimina are the symptoms that women ordinarily experience premenstrually. They are the same as PMS symptoms, but are experienced to a lesser degree and allow women to continue their normal functions

Situational stress disorders result from major life changes such as divorce or a new job. The possibility of such stressors should be elicited in the history

The main feature distinguishing PMS from chronic affective disorders is the follicular phase. Symptoms may be exacerbated premenstrually, but these patients have some level of dysfunction throughout the entire cycle

The primary difference between the ACOG guidelines for PMS and the PMDD criteria is the number of symptoms required for the diagnosis. PMDD requires at least five of these symptoms, while the PMS guidelines do not require a specific number or class of symptoms. APA symptoms of PMDD (five or more at severe level premenstrually – one must be a CORE* symptom:

Markedly depressed mood*	Lethargy, fatigue
Marked anxiety/tension*	Appetite change/food cravings
Marked affective lability*	Sleep disturbance
Decreased interest in usual activities*	Feeling overwhelmed
Difficulty concentrating	Physical symptoms (breast tenderness, bloating, headache)
Marked anger/irritability	

What percentage of women meet the criteria for PMDD? 5%

What percentage of women significantly experience PMS but do not meet the criteria for PMDD? 20–25%

	The examiner should be looking for organic disease associated with PMS-type symptoms. These may include galactorrhea associated with hyperprolactinemia or pelvic pathology like ovarian cyst, endometriosis, leiomyomata or pelvic inflammatory disease associated with pelvic pain and distention. Most commonly the physical exam is normal
	There is no laboratory test to identify premenstrual syndrome or premenstrual dysporic disorder. Some lab tests might be helpful, however, such as thyroid tests that would rule out the cause of fatigue or other similar symptoms. Experience indicates that patient education and support, stress reduction, a healthy diet, regular exercise and vitamin supplementations help many women both to understand and to feel more in control of their symptoms
Drug therapies	State-of-the-art treatment for PMS is with selective serotonin reuptake inhibitors (SSRIs). When these do not work, an anxiolytic is usually next choice. A GnRH agonist may be used to suppress the menstrual cycle when symptoms are severe and respond to no other therapy.
	What percentage of women with severe PMS or PMDD respond to serotonergic anidepressants? 50–60%
	SSRIs are the first-line treatment at present. These can be administered daily or in the symptomatic luteal phase. If SSRIs are discontinued, symptoms may return swiftly
SSRIs	Start with half the recommended dosage for depression in order to reduce side-effects. If the patient tolerates this well, increase the dosage to 50 mg daily. See actual dosages below in summary
	Hold this dosage for one or two menstrual cycles to determine the degree of efficacy
	Regarding the patient who conceives→ Chambers and colleagues observed a 6.1-fold increased risk of persistent pulmonary hypertension in newborns whose mothers had received SSRIs after 20 weeks' gestation
Anxiolytics	If an SSRI does not work, alprazolam (Xanax®) is usually the next choice
	Start with 0.25 mg t.i.d. and increase as needed to control symptoms to a total of 1–1.25 mg/day
	Buspirone HCl 10 mg (Buspar®) p.o. t.i.d., taken throughout the cycle, may be effective
GnRH agonist	GnRH agonist can relieve the symptoms of PMS by producing a medical oophorectomy in patients for whom no other treatments work

Premenstrual syndrome (Patient's guide)

Coping with PMS

For women who suspect premenstrual syndrome (or who don't, but find that on some days things just don't go right!), there are several ways of coping that do not require a doctor's prescription

Chart your symptoms

Finding out whether or not you are a victim of PMS can make you feel better. If you are a victim, knowing that you are not alone and understanding that the disorder is biochemical and not psychological can provide enormous relief

Talk about it

Talk about it with your husband, family and even your employer – if necessary. You need their sympathy and support rather than having them tell you to "pull yourself together" – which is exactly what someone with PMS cannot do. Your children also need to understand what PMS entails

Eat frequently and properly

Good nutrition, with reduced fats and increased complex carbohydrates, is important throughout your cycle, but even more so after ovulation, when it is especially vital to keep blood sugar on an even keel. The change in hormone levels then alters your biochemistry, making you more susceptible to low-blood-sugar reactions – such as irritability, migraines, panic, tears, angry outbursts. Never go for more than 4–5 h without food; a snack at bedtime may help, too

Exercise regularly

Half-hour aerobic workouts, in which you increase your pulse and work up a sweat, are good mood elevators. You should exercise three times a week all month

Cut down on salt

Since salt holds water, reducing salt intake should reduce bloating. In addition, do not add salt to your food. Cook with less and avoid high-sodium foods

Take vitamin supplements

The "B" vitamins – especially B_6 – are known to reduce bloating and have an antidepressant effect and they seem to help control carbohydrate cravings. Suggested beginning daily dosage is a B-complex, containing 50 mg of B_6 daily, building up to 200–500 mg in a few months. Since B vitamins are water soluble, you will excrete what you don't need

Add bran to your diet

Some women become constipated during the premenstrual time and for the first few days of their period. Bran will bind water to itself and aid elimination. Be careful with alcohol. It may take only half your normal amount to make you merry!

Cut down on caffeine

This includes not only coffee but tea, cola, diet sodas and chocolate as well. A group of substances (xanthines) in these encourages breast cysts: women who reduce intake of xanthines may find breasts are less tender during the premenstrual stage

Reduce stress if possible

Take things easy just before your period. If you're working, try to schedule important meetings and deadlines for another time of the month; at home, do not plan a dinner party or invite your cousin and her four kids for the weekend. Set aside some time to nap, listen to music, read or go for a walk

Make love

Many women find that orgasm helps reduce pent-up tension. While masturbating may not be as good as sex with a loving partner, it also reduces pelvic congestion

The above are general recommendations for patients with symptoms of PMS. The actual PMS work-up can become quite complicated. SSRIs can be very helpful in this syndrome

Summary

No lab test to diagnose

Diagnose with true cyclicity of symptoms and exclusion of other medical and psychiatric disorders

Patients with PMS are symptom-free from what day of the cycle to what day of the cycle?	4–12
What % of females with PMS have an underlying psychiatric disorder?	50–60%
PMS occurs in what % of females?	20–40%
Average premenstrual weight gain is	¼ pound

Treatment of PMS consists of regular exercise and:

Fluoxetine	20 mg daily or
Sertraline HCl	50 mg daily or
Paroxetine HCl	10 mg daily or
Paroxetine-CR	12.5 or 25 mg daily or
Citalopram	20 mg daily or
Venlafaxine	25 mg b.i.d.

Uncontrolled studies have shown caffeine + chocolate elimination to help mastalgia. B_6 increased to	200 mg

New formulations of OCPs sometimes help alleviate symptoms

Diuretic therapy or alprazolam (monitor) only if very necessary

PRE-OP LABS

Guidelines

The following labwork is required for ALL PATIENTS coming to the OR for surgery:

Age (years)	*Men*	*Women*
Under 40	None	Hgb or Hct
40–59	EKG BUN/glucose	EKG BUN/glucose Hgb or Hct
Over 60	EKG CXR BUN/glucose Hgb or Hct	EKG CXR BUN/glucose Hgb or Hct

Special notes

CXR is good for 1 year assuming no interval change in health

EKG is good for 6 months assuming no interval change in health

Blood work is good for 1 month assuming no interval change in health

These guidelines assume that the patient is otherwise in good health. Complicating factors (i.e. diabetes, hypertension, COPD) should obviously be considered for need to expand on these guidelines

PRE-TERM BIRTH

Diagnosis

Gestational age between	20–37 weeks
Cervical CHANGE needed to treat if cervix	< 2 cm or < 80% effaced
If cervix is	> 2 cm or > 80%
Treat if 4 contractions noted every	20 min
or 8 contractions noted every	60 min

Biochemical markers

FDA-approved markers:

(1) Fetal fibronectin (FFN)
(2) Salivary estriol

How FFN works

FFN normally increases, 20 weeks' gestation in cervicovaginal secretion. + if 50 mg/dl after 23–24 weeks. FFN compares with transvaginal US assessment of cervical length. There can be a false positive if sexual activity within 24 h, recent cervical exam and vaginal bleeding

Draw back to salivary estriol – must wait after eating, chewing or smoking

Investigational markers:
(1) Corticotropin-releasing hormone (CRH)
(2) hCG
(3) Prolactin
(4) Cytokines
(5) Interleukin-1α
(6) Interleukin-6
(7) Interleukin-8

Morbidities associated with pre-term birth

Anemia, apnea, cerebral palsy, infections resulting from immature immune system, intraventricular hemorrhage, jaundice, mental retardation and learning disabilities, necrotizing enterocolitis, neonatal death, periventricular leukomalacia, respiratory distress syndrome, and retinopathy

Treatment

Lateral bed-rest, hydration and/or IVFs (why hydrate?)
ADH theory
Flood gate + similar structure theories from posterior pituitary
External monitor
Sterile speculum exam – R/o PROM with nitrazine + ferning
Culture for GBBS, BV, GC and *Chlamydia*
Ultrasound – EGA, EFW + position, AFV, placenta, anomalies
Labs – CBC, U/A + culture, lytes, glucose, creatinine

Drug therapies and possible complications

- β-Sympathomimetics (ritodrine or terbutaline) 0.25 mg SC q. 6 h
  Pulmonary edema
  Hypokalemia
  Hyperglycemia
- $MgSO_4$ – neuromuscular blocking agent 4–6 g x 20 min then 2–3 g
  Contraindicated with MG
  Care with renal failure, hypocalcemia
- Indomethacin (NSAID) – blocks PG production
  Increase in IVH, necrotizing enterocolitis, closure of ductus, oligo
- Nifedipine – antihypertensive
  Associated with increase in stroke and/or MI hypotension
  Chance of having PTD if patient has already had PTD 20–40%
- 17P – 17α-hydroxyprogesterone caproate (HCP) 250 mg/ml

Administration of weekly injections until 36 weeks' gestation reduced the rate of pre-term birth at:

Earlier than 37 weeks	32% reduction
Earlier than 35 weeks	31% reduction
Earlier than 32 weeks	39% reduction

Infants born weighing less than 2500 g and the admission rate of infants to NICU were also reduced irrespective of mother's race, number of previous pre-term births, or gestational age of previous preterm births

References regarding 17-P

Johnson JWC. Progestins in pregnancy. In: Niebyl JR, ed. *Drugs used in Pregnancy*. Philadelphia: Lea & Febiger; 1988:109–16
Schardein JL. Congenital abnormalities and hormones during pregnancy: a clinical review. *Teratology* 1980; 22:251–70

Pre-term labor – Summary

Diagnostic criteria for pre-term labor

(1) Gestational age between 20 and 37 weeks
(2) If the cervix is less than 2 cm dilated and less than 80% effaced, then cervical change is required to initiate tocolytic therapy
(3) If the cervix is already > 2 cm or > 80% effaced, then therapy may be initiated when contractions occur with a frequency of four every 20 min or eight every 60 min, despite lateral bed-rest and intravenous hydration

Evaluation of pre-term labor

The initial evaluation of the patient with possible PTL has the following goals:
(1) Confirm the diagnosis of PTL
(2) Identify contraindications to tocolytic
(3) Select the most appropriate tocolytic

A "step-by-step" approach

(1) Lateral bed-rest and intravenous fluids constitute care while a thorough obstetric and medical diagnosis is obtained
(2) An external monitor assesses contractions and fetal well-being
(3) A sterile speculum examination should be done to exclude PROM and cultures of vaginal group B streptococci, cervical gonorrhea, and *Chlamydia* should be obtained
(4) Ultrasound should be performed to confirm gestational age; to assess amniotic fluid volume; and fetal weight and position; to locate placenta and to rule out anomalies
(5) Lab work should include a complete blood count and differential, urinalysis and culture, electrolytes and glucose and creatinine levels

Contraindications to tocolysis

Maternal	*Fetal*
Hypertension	Fetal demise or lethal anomaly
Cardiac disease	Amnionitis
Bleeding–abruption	Fetal distress
Hyperthyroidism	IUGR
	Gestational age > 37 weeks
	Birth weight > 2500 g
	Cervical

Contraindications for specific tocolytic agents

β-*mimetic agents*

Maternal cardiac rhythm disturbance or other cardiac disease

Poorly controlled diabetes, thyrotoxicosis or hypertension

Magnesium sulfate
- Hypocalcemia
- Myasthenia gravis
- Renal failure

Nifedipine
- Maternal liver disease

Indomethacin
- Asthma
- Coronary artery disease
- Gastrointestinal bleeding (active or past hx)
- Oligohydramnios
- Renal failure
- Suspected fetal cardiac or renal anomaly

Potential complications of tocolytic agents

β-**adrenergic agents**
- Hyperglycemia
- Hypokalemia
- Hypotension
- Pulmonary edema
- Cardiac insufficiency
- Myocardial ischemia
- Maternal death

Indomethacin
- Hepatitis
- Renal failure
- GI bleeding

Magnesium sulfate
- Pulmonary edema
- Respiratory depression
- Cardiac arrest
- Maternal tetany
- Profound muscular paralysis
- Profound hypotension

Nifedipine
- Transient hypertension

Tocolytic therapy with magnesium sulfate

Procedure

(1) Loading
4–6 g of magnesium sulfate (10% solution) IV slowly over 20-min period
(2) Maintenance
Add 40 g of 50% magnesium sulfate solution to 920 cc D5W. Infuse at 2 g/h (50 cc/h). Infusion rate may be increased to 3 g/h if uterine activity has not subsided in 30 min. Magnesium levels should be obtained before and after loading dose and at 2, 6 and 12 h during maintenance therapy. Therapeutic level 5–8 ng/l
(3) Monitor
(a) Deep reflexes
(b) Respiration every 12 min
(c) Urinary output 25 cc or more per hour
(d) If deep reflexes are absent, discontinue immediately. Obtain magnesium sulfate level every 4 h

Calcium gluconate (1 g IV) is the antidote and must be available in labor rooms (10 ml 10% sol IV @ 3 min)

Magnesium sulfate infusion should be continued for a minimum of 10–12 h, after cessation of uterine activity. Strict intake and output charting should be maintained as magnesium sulfate does have cardiovascular side-effects

Terbutaline sulfate therapy (Brethine)

Dosage and administration

Subcutaneous

(1) Use a 25-gauge subcutaneous needle or 1-cc tuberculin syringe and obtain a 1-mg terbutaline sulfate
(2) Administer terbutaline sulfate 0.25 SC initially and repeat every 30 min for a total of five doses (1.25 mg) as long as the maternal pulse rate is less than 120 BPM. **Notify physician if heart rate is over 120 BPM**
(3) If premature labor has not been effectively arrested following initial therapy, terbutaline should be discontinued
(4) If premature labor has been effectively arrested, begin maintenance subcutaneous dose as follows

Most patients

(1) Terbutaline 0.25 mg SC every 6 h for 24 h
(2) Maternal BP and pulse taken and recorded prior to each dose
(3) Do not administer drug if maternal pulse is more than 120 BPM and notify physician
(4) Should uterine activity persist during the maintenance therapy, administer a start dose (0.25 mg) and increase dose scheduled to 0.25 mg every 4 h for the remainder of 48-h period as per physician's order. Should this regime become ineffective, therapy should be discontinued

Certain patients

Certain patients may be candidates for "treat and release" therapy. These will be patients with mild, poorly felt contractions without cervical changes who may be deemed to be in questionable or mild premature labor and whose contractions ceased within 6 h of initial therapy

Calcium channel blocking therapy (nifedipine)
Not recommended due to potential maternal hypotension, thus increased uteroplacental perfusion

Prostaglandin inhibitor therapy (sulindac or indomethacin)
Not recommended due to associated neonatal morbidity

Adjunctive therapy

Corticosteroids should be considered for the induction of fetal lung maturity. All women between 24 and 34 weeks of pregnancy at risk for pre-term delivery are candidates for antenatal corticosteroid therapy

Betamethazone 12 mg IM in two doses 24 h apart ***or***
Dexamethazone 5–6 mg q. 12 h for total of up to four doses

PREVENTIVE CARE

Tetanus–diphtheria booster is needed once every	11–16 years
Cholesterol testing begins at age	45
Cholesterol testing is then repeated every	5 years
Fecal occult blood testing should begin at age	50
Tetanus booster is required after age 19 every	10 years
Leading cause of death for females is ACCIDENTS between ages	19–39
Leading cause of death for females is CANCER between ages	40–64
Leading cause of death for females is HEART DISEASE after age	65
Influenza vaccine should be given annually to women after age	50
Pap tests should start when a woman is sexually active or at age	18

PROGESTERONE

Principal source during pregnancy is the placenta
Corpus luteum
produces at rate of 22–43 mg/day
Progesterone responsible for:
(1) Prepares endometrium for implantation (proliferative to secretory)
(2) Inhibits contractions of uterus
(3) Increases viscosity of cervical mucus
(4) Stimulates breast glandular development
(5) Raises basal body temperature

Progestin potency

MPA	10 mg
Is = to how much micronized progesterone?	200 mg
How much progesterone vaginal suspension?	90 mg
Norethindrone?	
Medroxyprogesterone	C17
Testosterone derivatives (norethindrone, norgestrel – levonorgestrel)	C19

"Natural" Progesterones

Derived from diosgenin in soybeans (*Glycine max*) or an inedible Mexican wild yam (*Dioscorea villosa*). Diosgenin is not converted to progesterone in human body so oral or topical wild yam preparations would be expected to be ineffective for hormonal purposes

IM	25 mg/day
Local (OTC)	30 mg/day
Loses potency due to 5α-reductase activity so give	60 mg/day

Transdermal (Pro Femme, Pro Gest and other creams)
Should not be used as the progestin component of HRT. This does not preserve bone
Rectal and vaginal 100 mg/day

Micronized progesterone

(Allows form of extremely small particles of progesterone to be absorbed) < 10 μm
Prometrium 100–200 mg/daily

Crinone

(Vaginal gel with bioadhesive that induces secretory endometrium despite low serum levels)
Dosage is every other day delivering 45 mg/day
Half-life of progesterone is 30 min
Salivary measurements of progesterone do not reflect serum levels and should not be trusted

Progesterone gels

Prochieve 4%	Indicated for women with secondary amenorrhea Dose is every other day up to six total doses
Prochieve 8%	Indicated for progesterone supplementation in assisted reproductive technology (ART) Dosage is once daily for supplemention and twice daily for ovarian failure

Side-effects

(1) Attenuates lipid profile with estrogen but due to direct vasoconstricting effect, progestins potentially may negate the protective actions of estrogens
(2) Lethargy

(3) Weight gain
(4) Fluid retention
(5) Breast tenderness
(6) Sedation and mood changes are probably the result of progesterone metabolites that bind to GABA receptors in the CNS

PROLAPSE (POP)

Prolapse is not relaxation or attenuation. It is actually specific breaks in specific fascia and fibers. Goal is to optimize the surgical repair by individualizing and identifying primary site of damage that can be repaired. Look for vault prolapse in any woman who has an advanced degree of vaginal prolapse. The goals of surgery are to normalize support of all anatomic compartments, alleviate clinical symptoms, and optimize sexual, bowel, and bladder function

Three principal sources of damage:
(1) Weakness associated with neuropathy – congenital (spina bifida) or acquired (disc herniation)

Patients with abnormal spinal curvatures were 3.2 × more likely to have pelvic organ prolapse than patients with a normal curvature (Mattox TF, Lucente V, McIntyre P, *et al*. Abnormal spinal curvature and its relationship to pelvic organ prolapse. *Am J Obstet Gynecol* 2000;183:1381–4)

(2) Trauma (ob origin)
(3) Aging (decreased estrogen – loss of collagen + supporting tissue)

Symptoms

Anterior (urgency, frequency, urinary incontinence, voiding dysfunction)
Posterior (difficulty with defecation, pelvic pressure, bearing down sensation, sacral backache)
Protrusion from vagina, coital difficulty

Some patients have no urinary incontinent symptoms, despite severe prolapse, but some patients will have these symptoms after the prolapse is corrected; therefore, it is recommended that a Burch or midurethral sling be done at the same time of prolapse correction

Grades of descent

Between normal position and ischial spines	I
Between ischial spines and hymen	II
At hymen	III
Through hymen	IV

Describe grade of descent with and without prolapse

Evaluate

Pelvic diaphragm, endopelvic fascial support, vaginal apex, anterior/posterior walls and perineum

What % of females with POP are affected enough to need surgical therapy?	10–15%
What % after vaginal surgery will need reoperation?	33%
What % after abdominal surgery will need reoperation?	16%

Procidentia is total uterine prolapse with eversion of entire vagina
Lithotomy – adhesions? tumors? lesions? cytology + biopsy? ureters? down?

Ureters are obstructed in what % with procidentia?	92%

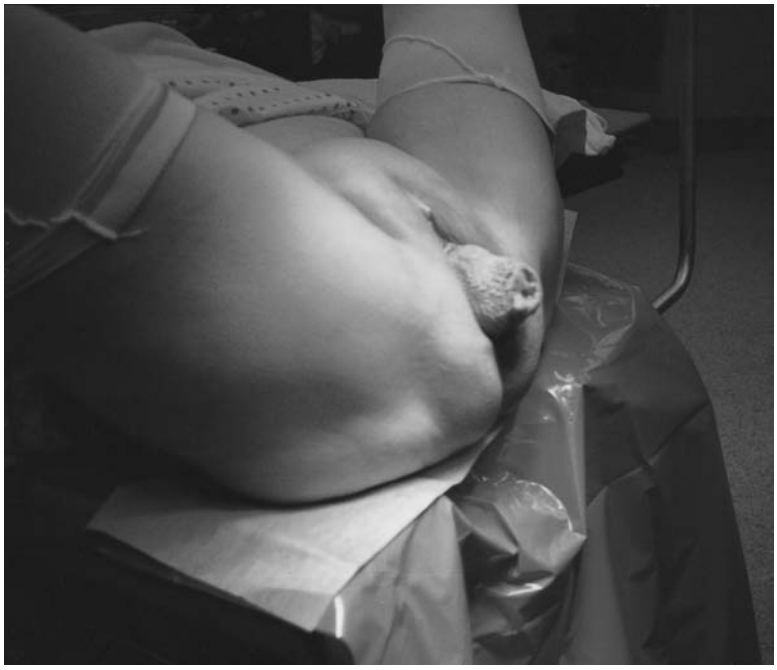


Figure 20 Uterine prolapse (complete) before surgery

Evaluate for enterocele with patient in standing position with maximum Valsalva maneuver
It is not cost-effective to use urodynamic testing as opposed to a basic office evaluation of incontinence when evaluating patients with known prolapse and symptoms of SUI (Weber AM, Walters MD. Cost-effectiveness of urodynamic testing before surgery for women with pelvic organ prolapse and stress urinary incontinence. *Am J Obstet Gynecol* 2000;183:1338–46)

Endopelvic fascia

Fibromuscular network

(1) Cardinal and uterosacral ligaments
Tenaculum to right and left. Is cervix to introitus?

(2) Pubocervical fascia
Paravaginal defects very palpable (lateral along the "white line" of the arcus tendineus) – correct with PARAVAGINAL repair
Endopelvic fascia and bilateral sup ant vaginal sulcus is sutured to arcus tendineus fascia with interrupted permanent sutures (with GSI – repair with Burch for retropubic urethropexy)

Central (midline) defect – correct with anterior repair
CYSTOCELE is attenuation and/or rupture of the pubocervical fascia. May also have vaginal vault prolapse with failure of vaginal support structures – cardinal and uterosacral ligaments. An ANTERIOR COLPORRHAPHY corrects anterior midline vaginal endopelvic defects. It is effective, quicker recuperation, there is no neuromusculature compromise of the pelvic diaphragm but should not be performed as primary procedure for incontinence as it has failure rate of @ 50%

Transverse defect (alone) – no SUI. Postvoid residual check

(3) Rectovaginal (Denonvillier's) fascia
Rectocele – caused by inferior separation of rectovaginal fascia. Perineal descent due to pudendal nerve injury

Diagnosis: push lower two-thirds of vagina down with cottonball stick then look for bulge in front. Repair by POSTERIOR COLPORRHAPHY. Involves:
(a) Plication of levator ani muscles in midline
(b) Narrowing of vaginal caliber
(c) Perineorrhaphy to close genital hiatus

Risks:
(a) Dyspareunia
(b) Decreased defecatory function
Enterocele – evaluate by pushing down on perineum while lifting cervix or having patient stand and performing maximal Valsalva maneuver.
Prevent and correct with obliteration of the cul-de-sac

Pelvic diaphragm

Fibromuscular connective tissue backed up by striated muscle with its fascial covering. Serves as back-up support to endopelvic fascia and as principal support during increased intra-abdominal pressure

(1) Levator group – test with Kegel's and neuro exam
Iliococcygeus
Pubococcygeus – fibers of Luschka broken with paravaginal defect. Most significant
Coccygeus

Urogenital diaphragm

Superficial and anterior to pelvic diaphragm and aids in closure of the vagina, urethra and rectum

(1) Transverse perinei muscles
(2) Intrinsic muscles of the perineum
Bulbocavernosus m
Superficial transversus m
External and internal anal sphincters m
Perineorrhaphy

Treatment options

Minimal – medical therapy
Pessaries (rings, donuts, Gehrung, Gelhorn, cubes only short term), Kegel's, estrogen × 30 days

Surgical therapy
MIVH or TVH with A&P repair followed by a procedure to restore vault support and preserve vaginal coital function (McCall's culdoplasty – uterosacral ligament suspension – better vaginal depth and normal alignment)
Cystoscopy is essential with uterosacral ligament suspension – ureteral injury rate is as high as 11%
LAVH or TAH with A&P repair if PID/endometriosis or unable to accomplish vaginally
Grafts may be used as necessary for A&P repairs

Good uterine support
MANCHESTER–FOTHERGILL
A&P with amputation of the cervix

Older female without sexual activity
LeFORT partial colpocleisis
A&P with vaginal walls sutured together

Vaginal vault suspensions
Consider when symptomatic prolapse of vaginal apex. Most reconstructive cases can be performed transvaginally. If the apex is repaired up solidly, the surgeon is usually home free. Remember that any operation that alters the vaginal axis will seriously weaken the vagina opposite the distorted axis
Vaginal approach
(1) Uterosacral suspension – attach uterosacral and cardinal ligaments to pararectal fascia. Better vaginal depth with normal alignment
Caution: ureters in close proximity of the uterosacral ligaments

- The ureters are how close at the level of the cervix in cadavers? 1.4 cm

(2) Sacrospinous ligament fixation – suspend vaginal vault @ 2 cm medial and anterior to ischial spine with native vaginal tissue. Use with functional pelvic diaphragm + good endopelvic fascia. Use Miya hook or Capio Suture Capturing Device (Boston Scientific, Urology/Gynecology, Natick, MA) and pulley stitch to attach to sacrospinous/coccygeal ligament. If sexual function is critical to the patient, a sacrocolpopexy should be the primary surgical option
Not anatomic, but success rate is 83–97%

Can predispose to recurrent anterior wall prolapse, vaginal shortening, sexual dysfunction, pain and hemorrhage. *Caution*: pudendal, sciatic and gluteal nerve entrapment. Pudendal artery

Iliococcygeus and sacrospinous fixation offer equally effective results for the treatment of vaginal vault prolapse with similar rates of postoperative cystocele, buttocks pain and bleeding requiring transfusion (Maker CF, Murray CJ, Carey MP, *et al*. Iliococcygeus or sacrospinous fixation for vaginal vault prolapse. *Obstet Gynecol* 2001;98:40–4) Bilateral sacrospinous fixation avoids lateral vaginal deviation

If voiding improves when prolapse is reduced, the prolapse is probably causing urethral obstruction. Before repairing an advanced degree of prolapse, identify any urethral obstruction or occult sphincteric incontinence

(3) TVTs or TOTs (transobturator) slings – usually included if there is any urinary incontinence involvement. Anterior compartment prolapse is more likely with a concomitant anti-incontinence procedure. Until trials are done, how the kits with synthetic mesh compare with conventional repairs will not be known. Various kits used today include:

(a) Perigee (transobturator anterior prolapse repair system) – treats all types of anterior vaginal wall defects – central, lateral, proximal, and distal – with a standardized, repeatable approach

(b) Apogee (vaginal vault prolapse repair system) – treats apical and posterior prolapse with graft augmentation options that accommodate individual patient pathologies

(c) Straight-In Sacral Colpospexy System is designed to treat vaginal vault prolapse by suspending the vaginal apex from the sacrum with a tension-free sling

(d) Monarc TOC Series is a hammock-shaped mid-urethral sling designed to mimic patient anatomy and restore normal pubo-urethral support

(e) SPARC is a minimally invasive sling system that utilizes a suprapubic approach and polypropylene mesh to create a U-shaped sub-urethral support under the urethra during increased abdominal pressure

(f) BioArc is a hybrid sling system that pairs a suburethral biologic graft material called InteXen LP with AMS-proven polypropylene mesh for lateral support. Currently, it is the only sling offering a synthetic/biologic combination. BioArc is a unique option for physicians who prefer biologics or for those patients who may be at high risk for complications with synthetic grafts

(g) In-Fast Ultra is a device that allows minimally invasive, transvaginal sling placement for proximal urethral support. A concomitant repair surgery also can be performed at the same time

(h) Acticon Neosphincter treats severe fecal incontinence due to neurogenic, congenital or traumatic causes when more conservative treatments have failed

(i) Posterior slings – minimally invasive treatment of vaginal prolapse via small bilateral incisions made @ 2 cm lateral and posterior to the rectum, through the levator muscle, and threading the graft behind the vaginal apex and parallel to the vagina on both sides, pulling the apex back into the pelvis.

- **This is an excellent option to retain sexual function, and is often replacing the sacrocolpopexy in popularity due to its safety, simplicity, and minimal invasiveness**

Abdominal approach

Transabdominal sacral colpopexy – suspend vaginal vault to S3–4 vertebral bodies just below the sacral promontory. Use with attenuated endopelvic fascia, compromised pelvic floor and severe ongoing physical stress.
Consistent cure rate is > 90%
Sacral colpopexy vault suspension technique has best longevity

Complications:
(1) Hemorrhage – keep bone wax and thumb tacks
(2) Vaginal mesh erosion within 5–9 years out 3.3%
(3) Enterocele formation behind graft – prevent with concurrent Halban culdoplasty

AVOID MIDDLE SACRAL ARTERY
KEEP STERILE THUMB TACKS and BONE WAX available at all times
The Straight-In System is designed to treat vaginal vault prolapse via the laparoscopic or abdominal approach. Pre-configured IntePro Y-graft with large pores encourage tissue ingrowth. Titanium screws are used with the Straight-In Powered Inserter for direct access to the sacrum and precise screw placement. A vaginal distender is also available for full mobilization of the vaginal apex

Preoperative low-dose estrogen cream is crucial in most postmenopausal women regardless of the planned type of corrective prolapse procedures

Place multiple sutures (include posterior vaginal wall) to obliterate the cul-de-sac and prevent enterocele

PROSTAGLANDINS

Prostacyclin is a potent vasodilator that decreases platelet aggregation – What prostaglandin? PGI_2
This is the principal prostanoid synthesized in the endothelial cells of blood vessel walls. Thromboxane is a potent vasoconstrictor that increases platelet aggregation (prostanoids in PIH). There is an increase in thromboxane-to-prostacyclin ratio during PIH
Which prostaglandin increases the synthesis of non-collagenous proteins and hyaluronic acid?
Which induces the production of the cytokine interleukin-1? PGE_2
This "inflammatory process", with its increased enzyme activity allows for the loosening, separation and splitting of collagen fibers of cervix
How much aspirin does it take to disable platelets–thromboxane production machinery for 1 week lifespan of platelet? (Only pts at risk should be treated, such as rec PIH, IUGR, Hx of fetal demise) 81 mg per day

PROSTAGLANDIN CERVICAL RIPENING

Increases Bishop's score
Decreases total number of hours of labor
Decreases total dose of oxytocin required
Increases incidence of spontaneous labor
Does not affect C-section rate in any of the studies
How prostaglandin ripening works – causes cervical connective tissues to exhibit increased remodeling, involving altered proteoglycan metabolism (believed to cause a breakdown of the collagen matrix), increasing smooth muscle fiber alignment and hypertrophy and changes in glycosaminoglycans. During this process, PGE_2 increases synthesis of non-collagenous proteins and hyaluronic acid, which may induce the production of the cytokine interleukin-1. This "inflammatory process" with increased enzyme activity allows for loosening, separation and splitting of collagen fibrils

PROTRACTION DISORDER

Criteria		
	Nulligravid – cervical dilatation	< 1.2 cm/h
	Prolonged latent phase	20 h
	Prolonged second stage	> 2 h
	Prolonged second stage with epidural	> 3 h
	Multipara – cervical dilatation	< 1.5 /h
	Prolonged latent phase	14 h
	Prolonged second stage	1 h
	Prolonged second stage with epidural	2 h

See also Arrest of dilatation

PSYCHIATRIC

What % pregnant women experience depression? 10%
What % pregnancies treated with lithium in first trimester result in Ebstein's anomaly? 0.1%
Treatment for initial onset of late-life depression should receive therapy AFTER recovery for 6 months
Older women with recurrent depression should receive therapy indefinitely
Treat depression in pregnancy with SSRIs if:
(1) Nutrition compromised
(2) Patient only sleeping 2–3 h per night
(3) Suicidal ideation
(4) Use of EtOH to self-medicate
Refer if suicidal *intent*, delusions and/or hallucinations, history of poorly controlled bipolar illness requiring multiple medicines or concurrent substance abuse

Common psychiatric disorders in women	*Treatments*
Panic disorder	SSRIs
Depression	SSRIs
Adjustment disorder	
Anxiety and depression	
Self-limited @ life stressors	
Hypochondriacal	
Somatic symptoms	70% in general population
Somatic disorder	(Exc females 0.2–2%)

Benzodiazepines – relatively contraindicated in late pregnancy due to neonatal syndromes and withdrawal
Anticonvulsants – concern is for neural tube defects and possible craniofacial defects

Typical psychological changes of pregnancy

(1) First trimester – concerns with body image that threaten self-esteem and sexuality. Feelings of vulnerability
(2) Second trimester – process of attachment to baby, fondness – concerns about baby not being normal
(3) Third trimester – embodies task of separation from fetus

Aspects of pregnancy → developmental crisis involving adaptive tests for both partners

Postpartum blues → "transient"

Postpartum depression → major depression @ 2 weeks after delivery; 30–50% risk of recurrence with each subsequent pregnancy

Postpartum psychosis → florid affective episodes with hallucinations, emotional lability, which occurs 2 weeks after delivery

St John's wort

The efficacy of St John's wort for a major depressive disorder was unsupported in a recent trial and may even be detrimental to the patient's overall mental health (*Hypericum* Depression Trial Study Group. Effect of *Hypericum perforatum* (St. John's wort) in major depressive disorder: a randomized controlled trial. *JAMA* 2002;287:1807–14)

PUBARCHE

Early hair over vulva or axilla? Premature pubarche if seen in female under what age? 8 years
If seen in male under what age? 9 years
Measure DHEA-S, testosterone, bone measurement of hand and wrist
Think adrenal hyperplasia or androgen-secreting tumor

PUBIC PARASITES

Crab louse → pediculosis pubis → *Phthirus pubis*
Itch mite → scabies → *Sarcoptes scabiei*
Symptoms → constant itching or increased symptoms of itching at night

Diagnosis → visualization of nits (eggs), parasites, excretions
Treatment → Nix cream (permethrin) or Kwell (lindane)

PULMONARY ARTERY CATHETER

Normal ranges

Right atrium or pulmonary artery, wedged mean		5–10 mmHg
Right ventricle,	systolic	15–20 mmHg
	diastolic	0–8 mmHg
Pulmonary artery, systolic		15–20 mmHg
	diastolic	8–15 mmHg

PULMONARY EMBOLISM

Diagnosis

CXR (normal or Westermark's sign or Hampton's hump). Also can see an enlarged descending pulmonary artery. EKG (sinus tach, new onset atrial fib or flutter). Also can see S wave in lead 1. Q wave in lead V3
ABG
Ventilation–perfusion (VQ) lung scan positive 90%

Treatment

Stat heparin (*see* Heparin therapy under Deep vein thrombosis)

PUPILS AND DRUGS

Amphetamine and cocaine → dilated
Heroin and morphine → pinpoint
Marijuana → injected conjunctivae

PUPP

Not associated with adverse perinatal outcome or recurrences.
Rx with benadryl. Incidence is 0.5%
(*See* Dermatologic conditions)

PYELONEPHRITIS IN PREGNANCY

Incidence in pregnancy 1–2%
Most common bacteria (*E.coli/Klebsiella–Enterobacter/Proteus*) 77%/15%/4%
Risk factors for ARDS are increased tocolytics and hydration
Treat with cephalosporin (Mezlin®, Pipracil®, Zosyn®) These are category B
Rocephin 1–2 g/day

PYROSIS

Heartburn in pregnancy is noted in what % of pregnancies? 70%

Etiology

(1) Decreased resting pressure of esophageal sphincter
(2) Increased acidity of gastric juice
(3) Progesterone decrease tone and propulsive motility of esophagus
(4) Increased size of uterus → increases abdominal–thoracic pressure
(5) Decreased motilin levels (hormone that stimulates smooth muscle of gut)

Treatment

(1) Elevate head of bed
(2) Bland, fat-free foods in small amounts
(3) Bicitra or Milk of Magnesia → Tagamet or Zantac (Category B). Reglan has not been found to be beneficial

RADIATION

There are NO anomalies, growth restrictions or abortions in pregnancy if radiation exposure is < 5 rads

First trimester – ALL OR NONE EFFECT
Organogenesis – estimated dose of 100 cGy will result in what % offspring with anomalies at birth? 100%
CNS effects may occur when radiated up to how many weeks? 25 weeks
Fetal stage: doses above what rad dose may result in growth retardation? 50 cGy
The upper limit of radiation exposure in pregnancy is CONTROVERSIAL. *Ob* Prolog states that this amount is safe in pregnancy < 1 cGy (1000 mrad)

Nomenclature

Rad – radiation absorbed dose
1 rad = 100 erg/g
1 Gray = 100 rad or 1 joule/kg
1 cGy = 1 rad
Radiosensitivity for most gyn tumors regarding microscopic disease is rad cure of 4000–5000
In @ what % of cases? 80–90%
2 cm lateral and 2 cm superior to cervical os (point at which the uterine artery goes above ureter) Point A
Central control 7500–8000 cGy
3 cm lateral to point A (pelvic wall). Lymph nodes up along pelvic wall Point B
Side walls Usually 5500 rad + or – 1000
Greatest exposure risk is between what gestational weeks? 8–15 weeks

Carcinogenesis

What % of children will develop childhood leukemia if exposed to ionizing radiation? 1/2000
What % of children will develop a malignancy from exposure to X-ray? 1/1000
Ultrasound should be limited to how many mW/cm^2? 94
US and MRI *not* associated with any adverse fetal effects but MRI should be avoided in ? trimester? 1st
Iodine is CONTRAINDICATED in pregnancy so instead of using ^{131}I, use ^{99m}Tc with < 0.5 rad
Pregnancy should not prevent X-ray procedures. (US and/or MRI to be used rather than X-rays when possible)
How much radiation is utilized for these procedures?

CXR	0.02–0.07 mrad
Mammo	7–20 mrad
Abdominal film	100 mrad
CT pelvimetry	250 mrad
CT of head or chest	< 1 rad
IVP	> 1 rad
BE or SB series	2–4 rads
CT of abdomen and lumbar spine	3.5 rads

Maximal amount of tolerated radiation in cancer therapy is remembered with mnemonic,
"A Small Rat Ran By" V – see below:

Abdomen	2500 rads
Small bowel	4000 rads
Rectum	5000 rads
Rectovaginal septum	6000 rads
Bladder	7000 rads
Vagina	7000 rads
Cervix	12 000 rads

RALOXIFENE (EVISTA)

Decreases vertebral fractures by 30–50%
Unlike tamoxifen in that it has no apparent trophic effect on the endometrium. Has NOT been shown to have a definitive, + effect on cognitive function in postmenopausal women. Raloxifene shows no cardioprotective properties in 4-year MORE study (according to Barrett-Connor E, Grady D, Sashegyi A, *et al*. Raloxifene and cardiovascular events in osteoporotic postmenopausal women: four-year results from the MORE (Multiple Outcomes of Raloxifene Evaluation) randomized trial. *J Am Med Assoc* 2002;287:847–57). Decreases LDL, no change in HDL or triglycerides. Increases risk of venous thromboembolic phenomena

RECTOCELE

Separation of rectovaginal fascia
Posterior repair corrects rectovaginal fascial defect

RECTOVAGINAL FISTULA

What % heal spontaneously? 25%
See also Fistula

RECURRENCE RISK

Obstetric complications	*General risk (%)*	*Risk of recurrence*
Gestational diabetes	1–3	46%
Placental abruption	0.5–3	5–100 x baseline
Placenta previa	0.3–0.5	6–8 x baseline
Spontaneous PTB	3	7–64%
Pre-eclampsia	5.6	7.5%
HELLP syndrome	0.2–0.7	4–38%
Stillbirth	0.8	7.3–8.4%
Shoulder dystocia	0.5–1	2–16 x baseline

RED DEGENERATION

Myomas that undergo 'red' or 'carneous' degeneration by hemorrhagic infarction. Hyaline and cystic degeneration (liquefaction) can be confused with cyst. These are other types of fibroid degeneration
Percentage of types of degeneration – hyaline/red/calcium/cystic 60%/11%/10%/4%
See Myomas

Symptoms Focal pain, tenderness to palpation, occasionally low fever or increased WBC
Differential Appendicitis, pyelo, abruption, stone
Treatment Rest, codeine – usually resolves spontaneously

RESPIRATORY CHANGES IN PREGNANCY

Increased	*Decreased*	*No change*
Tidal volume	Functional residual capacity	Arterial PaO_2
Minute ventilatory volume	Residual volume	Respiratory rate
Minute oxygen uptake	Total pulmonary resistance	Maximum breathing capacity
Airway conductance and closing volume	pCO_2	Forced or timed vital capacity
Oxygen consumption		

RESPIRATORY DISORDERS

Asthma

What % of pregnancies? 4%
Associated with increase in PIH, hyperemesis and hemorrhage.
Increase in IUGR, PTD, LBW, neonatal hypoxia

Manage with
(1) Baseline spirometry
(2) Peak expiratory flow daily – maintain 80% goal – treat p.r.n.
(3) Early ultrasound, fetal kick count surveillance, NST/BPP p.r.n.
(4) EFM during exacerbation – maintain SaO_2 at 95% or >

Management with asthma exacerbations
(1) Rest, O_2, hydration, β_2-agonist therapy, EFM
(2) Hydrocortisone 100 mg IV to decrease risk of inflammatory-mediated response 6–8 h later
(3) Oral steroids 1–2 weeks pulsed course p.m. if inhaler not option
(4) Identify asthma "triggers" → 75–80% have positive skin test
(5) Continue "allergy shots" in pregnancy if already been diagnosed
(6) Give annual influenza vaccine to pregnant patients if no egg allergy
(7) Do not avoid physical activity
(8) Cromolyn Na^+ inhaler regularly (mast cell stabilizer – prevents histamine release)

(9) β_2-agonist b.i.d. to q.i.d. inhaler
(10) Oral prednisone/prednisolone p.r.n. (11β-ol-dehydrogenase metabolizes in placenta)
(11) 1-h 50 g glucose tolerance test at 27–30 weeks secondary to increased risk of gestational DM
(12) Severe exacerbation – inhaled nebulized β-agonists
Terbutaline p.r.n.
PTL – $MgSO_4$ prescription of choice
Terbutaline requires increased dosing (ASA, NSAIDs, ibuprofen, indomethacin → 11% have hypersensitivities to these)

Management of labor
(1) Continue regularly scheduled medicines (except oral steroids)
(2) If moderate to severe, check peak flow volume on admission then repeat every 12 h as needed
(3) Maintain adequate hydration
(4) Provide adequate analgesia
(5) Avoid methergine and prostaglandin $F_{2\alpha}$ (Hemabate) → these are bronchoconstrictors. Use Pitocin or prostaglandin E_2 as needed
(6) Hydrocortisone 100 mg (or equivalent) IV every 8 h until 24 h postpartum if patient has a history of oral steroid use at least 2 weeks within previous 6 months or for those who have frequent exacerbations. This provides adrenal support and helps prevent exacerbations due to labor

Epistaxis

Treatment
(1) Intranasal saline spray 5–6 times daily
(2) Eucerin®/aloe b.i.d. in a.m. and p.m.
(3) Pinch nostrils and sit forward for 10 min

Rhinitis

Diagnostic character of mucus
(1) Copious clear secretions — Allergic
(2) Yellowish/greenish discharge — Infection

Common causes and treatments of nasal congestion during pregnancy:
(1) Allergic rhinitis (most common)
Changes in cortisol levels
Treatment – beclomethasone, topical cromolyn, Sudafed®
(not if hypertensive)
(2) Acute or chronic maxillary sinusitis
Tender frontal sinuses, yellowish/greenish discharge, X-ray p.r.n.
Treatment: amoxicillin 500 mg t.i.d. × 3 weeks
Erythromycin if allergic
Sudafed 60 mg b.i.d. or 30 mg q.i.d.
Vantin® 200 mg p.o. q. 12 h x 10 days as alternative
(3) Nasal polyposis
Steroid burst will sometimes shrink polyps, but not recommended
Treatment: Usually delay until after pregnancy is necessary
(4) Rhinitis medicamentosa (rebound rhinitis)
Occurs secondary to excessive use of over-the-counter decongestant nasal sprays. Sometimes it is necessary to use oxymetazoline (topical vasoconstrictor) to facilitate evaluation in patients who are not hypertensive
Treatment: discontinue spray or drops. Give p.o. decongestants or intranasal corticosteroids

RETROGRADE EJACULATION

Associated with urinary tract surgery (prostatectomy or surgery of bladder neck as child)
Diabetes
Spinal cord injuries

Diagnosis — Postejaculate urine sample

Treatment — α-Adrenergics or insemination with semen from bladder

Rh

RhD-negative woman (who is not RhD alloimmunized) should receive anti-D immune globulin (RhoGAM)
- @ 28 weeks unless father of baby also RhD negative
- Within 72 h after delivery of RhD + infant

- After first-trimester pregnancy loss
- After invasive procedures (CVS, amnio, fetal blood sample)

Also consider giving RhoGAM if patient experiences:

- Threatened abortion
- External cephalic version
- Second- or third-trimester antenatal bleeding
- Abdominal trauma

Rh antigens are CDEce – there is no little d	
What % Caucasians are Rh negative?	15%
What % Asians and North American Indians are Rh negative?	5%
What % of the Basque population are Rh negative?	95%
The most common cause of Rh D alloimmunization is fetomaternal hemorrhage in what % cases?	90%
Antenatal fetomaternal hemorrhage occurs in what %?	10%
The dose of Rh anti-D globulin (RhoGAM) is	300 µg
This dose prevents RhD alloimmunization up to how many ml of RhD and blood?	30 ml
How many fetal cells?	15 ml
If RhoGAM is forgotten during the postpartum stay, it can be given up to how many days?	28
If patient is 'weak D positive' then the patient does not need RhoGAM because she is positive BUT if she is postpartum, investigate possible fetomaternal hemorrhage	

Rh isoimmunization

D immunoglobulin administration to potentially susceptible candidates greatly reduces their chances of developing D isoimmunization and subsequent fetal morbidity/mortality of Rh hemolytic disease

Prenatal testing

Determine maternal ABO and Rh type with prenatal profile at initial visit

Rh negative (not isoimmunized) women should have repeat D antibody determination at 28–29 weeks' EGA

If negative, prophylactic D immunoglobulin (RhoGAM)
If positive, manage as D-sensitized

Presence of D-u (variant of D antigen) most often indicates maternal carriage of D-u antigen (considered Rh positive)

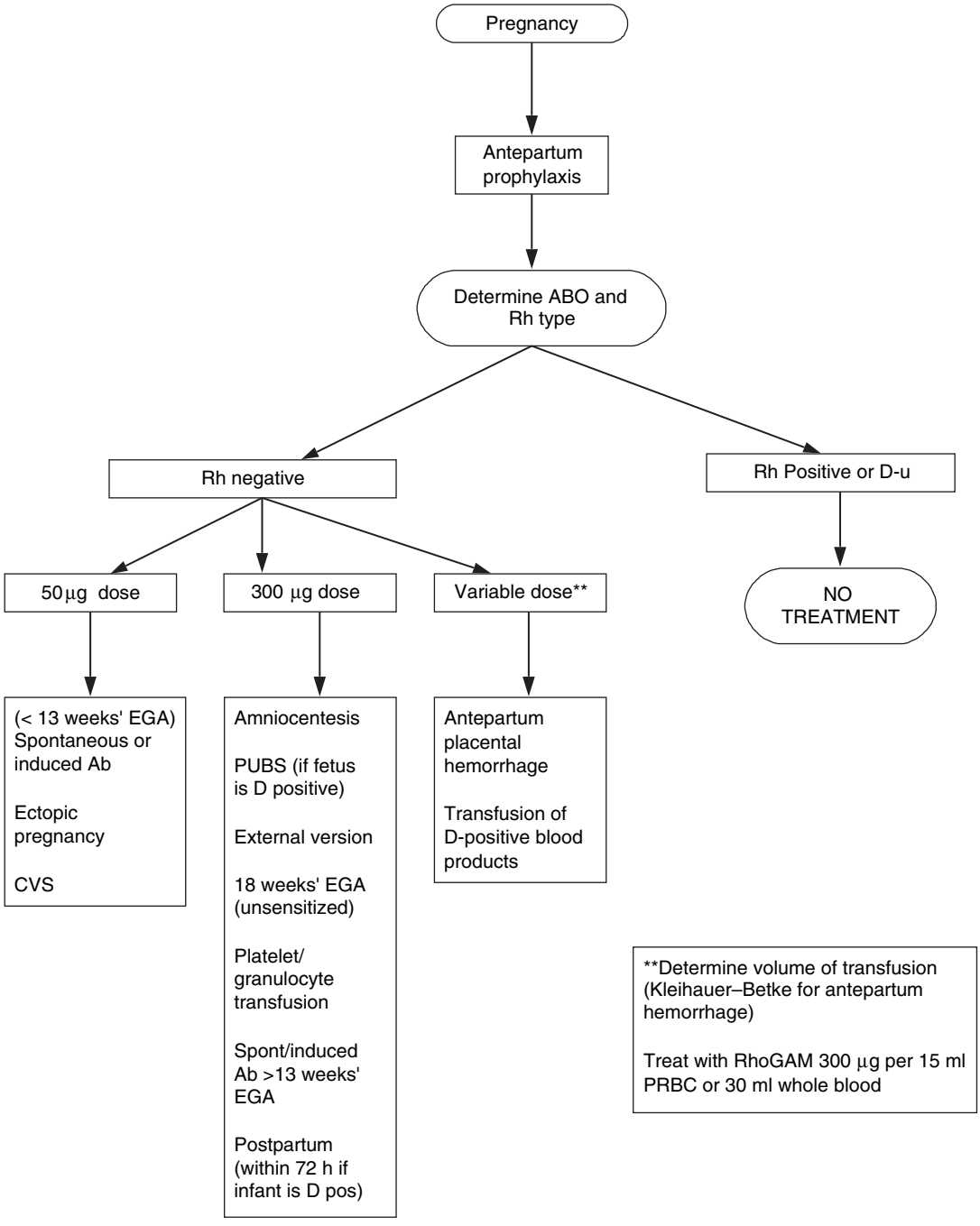
Prophylactic administration (used only in unsensitized Rh women)

(1) Abortion (induced or spontaneous) and ectopic pregnancy
   (a) Up to 13 weeks' EGA – 50 µg D immunoglobulin
   (b) After 13 weeks' EGA – full dose (300 µg D immunoglobulin)
(2) Amniocentesis
   300 µg dose in first, second or third trimester. Follow with routine antepartum/postpartum prophylaxis. If delivery anticipated within 48 h, RhoGAM may be held until postpartum
(3) Chorionic villus sampling
   50 µg dose D immunoglobulin
(4) Percutaneous umbilical cord blood sampling
   In D-negative women, analyze fetal blood. If D-positive, give 300 µg dose
(5) External version
   May precipitate fetal/maternal bleeding – 300 µg dose

Special situations

(1) Antepartum placental hemorrhage
   (a) Kleihauer–Bettke to estimate volume of fetal–maternal transfusion
   (b) 300 µg dose protects against 30 ml fetal blood (15 ml fetal RBC)
   (c) May test 48–72 h after RhoGAM dose for adequate treatment (excess D immunoglobulin = adequate treatment)
(2) Postpartum/postabortal sterilization
   Controversial, but low risk of sensitization, probably precludes this group from treatment
(3) Administration of blood/blood products
   (a) Use of D-positive PRBC/platelets/granulocytes may cause sensitization
   (b) With D-positive PRBC – 300 µg per 15 ml PRBC (administered in six divided doses q. 12 h x 72 h)
   (c) Platelets/granulocytes – single vial (300 µg) adequate

Prevention of Rh isoimmunization



RHABDOMYOSARCOMA

Most common soft tissue sarcoma of childhood. This makes up what % of malignant disease in children? 4–8%

Diagnosis: Biopsy. Poorly differentiated round or spindle-shaped cells. Electron microscopy – striated muscle fibers

Staging:
I = localized
II = regional with involved nodes
III = incomplete resection or biopsy with gross residual disease
IV = distant metastasis

Treatment: Chemotherapy (VAC) with subsequent limited surgery or radiation

RITGEN MANEUVER

Operator extends head via fetal chin through maternal rectum. Quickens delivery but presents greater fetal head diameter to maternal vulva so leads to more frequent episiotomy or vaginal lacerations

RNA VIRUS

Includes HIV, rubella, rubeola and hepatitis types A, C, D, E

ROBOTIC SURGERY

Types da Vinci (all systems below are similar to that incorporated in da Vinci)
Zeus MicroWrist
Voice-directed HERMES control system
AESOP robotic endoscope positioner

Disadvantages High price tag ($1.5 million for new, 4-arm da Vinci)

Advantages Computer interface that erases any tremor of surgeon's hands
Console that surgeon sits away from peripheral distractions
Virtual sense of being within the pelvic cavity
Easy movements and unparalleled visualization
Actually easier to learn than laparoscopic surgery (intuitive)
Robot responds directly to the directions of the surgeon's fingers
Robot's articulating arms are flexible compared with the "rigidity" of scopes

Gyn procedures that can performed by robot
Burch colposuspension
Dermoid cyst removal
Endometrial ablation
Hysterectomy
Laparoscopically assisted vagainal hysterectomy
Myomectomy
Oophorectomy
Oophorocystectomy
Ovarian cystectomy
Ovarian transposition
Removal of fibroids
Salpingectomy
Tubal ligation
Tubal reanastomosis
Tuboplasty
Vaginal prolapse repair

RU 486 (MIFEPRISTONE)

Is as effective as high-dose OCP for postcoital contraception. RU 486 in a dose of 600 mg will terminate pregnancies what %? 80%

Prolonged administration results in anovulation

SACROSPINOUS LIGAMENT FIXATION

Rectovaginal space/rectal pillar dissected
Sacrospinous ligament/coccygeus space
Miya hook ligature carrier 2 prolene
Place 2–3 cm medial to ischial spine
AVOID PUDENDAL VESSELS AND PUDENDAL NERVE

See Prolapse (POP)

This procedure is fully explained in Turrentine JE. *Surgical Transcriptions and Pearls in Obstetrics and Gynecology*, 2nd edn. London: Informa Healthcare, 2006

SARCOMA

Make up what % of uterine tumors?	3%
Mixed mesodermal tumors are the most common and this % is found outside uterus at time of dxn	60%

Endolymphatic stromal myosis – low grade – surgery only – may recur after LONG INTERVAL
Adenosarcoma – low malignant potential – TAHBSO with selective nodes – Rad + Chemo don't help

SCORING SYSTEMS

Apgar scoring of newborns
Bishop's pelvic score for induction
Vaginal atrophic index (VAI) for scoring atrophic vaginitis
VBAC scoring system (Flamm–Geiger) to evaluate likelihood of successful trial of labor
Zatuchni–Andros breech score to evaluate likelihood of avoiding problems during breech delivery

Apgar scoring of newborns

Sign	*0 Points*	*1 Point*	*2 Points*
Heart rate	Absent	Under 100	Over 100
Respiratory effort	Absent	Slow, irregular	Good, crying
Muscle tone	Limp	Some flexion	Active motion of extremities
Reflex irritability: response to catheter	No response	Grimace	Cough or sneeze, cry in nostril
Color	Blue–white	Body pink, extremities blue	Completely pink

Bishop's pelvic score

Features	*0 Points*	*1 Point*	*2 Points*	*3 Points*
Dilatation (cm)	0	1–2	3–4	5–6
Effacement (%)	0–30	40–50	60–79	80
Station	–3	–2	–1, 0	+1, +2
Consistency	Firm	Medium	Soft	—
Position	Posterior	Mid	Anterior	—

Vaginal atrophy index (VAI)

	1 Point	*2 Points*	*3 Points*
Skin elast + turgor	Poor	Fair	Excellent
Pubic hair	Sparse	Normal	> Normal
Labia	Dry atrophy	Full	> Full
Introitus	<1 Fg br	1 Fg br	2 Fg br
Vaginal mucosa	Thin/friable	Sm	Rugated
Vaginal depth	Short	Normal	At least normal

VBAC scoring system (Flamm–Geiger)

	Points
< 40 years of age	2
Vaginal delivery before and after their C-section	4
Vaginal birth after the first C-section	2
Vaginal birth before their Cesarean birth	1
No vaginal delivery	0
First C-section done for reason other than FTP	1
Cervix > 75% on admission	2
Cervix 25–75% on admission	1
Cervix < 25% on admission	0
Cervix dilated ≥ 4 cm on admission	1

Points	*Likelihood of successful TOL (%)*
0–2	49.1
3	59.9
4	66.7
5	77.0
6	88.6
7	92.6
8–10	94.9

Zatuchni–Andros breech score

	0 Points	*1 Point*	*2 Points*
Parity	Primagrav.	Multip.	>
Gestational age (weeks)	39 or >	38	37 or <
EFW	> 8#	7–8#	< 7#
Prev. breech	0	1	2 or >
Cx dil (cm)	2	3	4 or >
Station	–3 or higher	–2	–1 or lower

Total score of 5 or > indicates no difficulty in delivery of breech per vagina

SCREENING

Routine screening

Periodic H&P
Mammogram yearly > age 40 (definitely after age 50). Baseline > 35
Fecal blood test > age 50
Annual Pap (until at least three normal Pap smears)
Cholesterol every 5 years
Flexible sigmoidoscopy every 3–5 years after age 50
Annual flu shot after age 55

Tetanus–diphtheria every 10 years
Pneumococcal vaccine once at age 65
Plus if patient is obese then get fasting glucose test and if she is a smoker then get lipid profile

Lifestyle review

Smoking, alcohol, exercise, sexual behavior or risks, use of non-conventional therapies

If menopausal, discuss osteoporosis prophylaxis, screening if at risk and treatment p.r.n.

SELECTIVE ESTROGEN RECEPTOR MODULATORS (SERMs)

Triphenylethylenes
Clomiphene (Clomid or Serophene)
Tamoxifen (Novadex). Decreases breast cancer by 50%
Toremifene (Fareston). Well suited for PMP with metastatic breast cancer
Benzothiophene
Raloxifene (Evista)
(1) Decreases vertebral fractures by 30–50%
(2) Decreases C-reactive protein, lipoprotein and homocysteine
(3) Useful with females with history of breast cancer
(4) Increases vasomotor instability

STAR study – tamoxifen versus raloxifene (head-to-head study)

SEPSIS

Definitions

Early sepsis ("warm shock")
Systemic response to infection (temp > 38°C, tachycardia, tachypnea, $PaCO_2$ < 32 mmHg, WBC >12 000 or < 4000 or >10% bands [i.e. left shift])

Late/severe sepsis ("cold shock")
Characterized by hypoperfusion, hypotension organ dysfunction (peripheral cyanosis, cold extremities, lactic acidosis, oliguria, MS changes)

Septic shock
Sepsis accompanied by hypotension unresponsive to fluid resuscitation often requiring inotropic or vasopressor agents

Multiple organ system failure
Altered organ function such that homeostasis is not maintained without intervention

Diagnosis

Clinical manifestations
(1) Cardiovascular
  (a) Vasodilatation/increased vascular permeability – *hypotension*
  (b) Myocardial depression – *cardiac dysfunction*
(2) Pulmonary
  (a) Vascular permeability/endothelial damage – *hypoxemia/ARDS*
(3) Renal
  (a) Hypotension/vasoconstriction – *oliguria*
  (b) Prolonged cortical hypoxia – *ATN*
  (c) Immune-mediated damage – *interstitial nephritis*
(4) Hematologic
  (a) Endotoxin activation of coagulation cascade – *DIC*
  (b) Demargination/immune response – *leukocytosis*
(5) Neurologic
  (a) Decreased cerebral blood flow/hypoxia – *altered mental status*
(6) Homeostatic
  (a) Endotoxin/TNF effect on hypothalamus – *fever*

Laboratory investigations
(1) CBC and platelets with differential
(2) Electrolytes
(3) Arterial blood gases
(4) BUN/Cr

(5) Urinalysis
(6) Coagulation studies (PT/PTT, fibrinogen)
(7) Serum lactate
(8) Cultures – blood, urine, other suspicious sites (endometrium, amniotic fluid, wound/episiotomy, sputum/drains)
(9) Radiologic studies – CXR +/– CT, MRI or abdominal X-ray

Principles of management

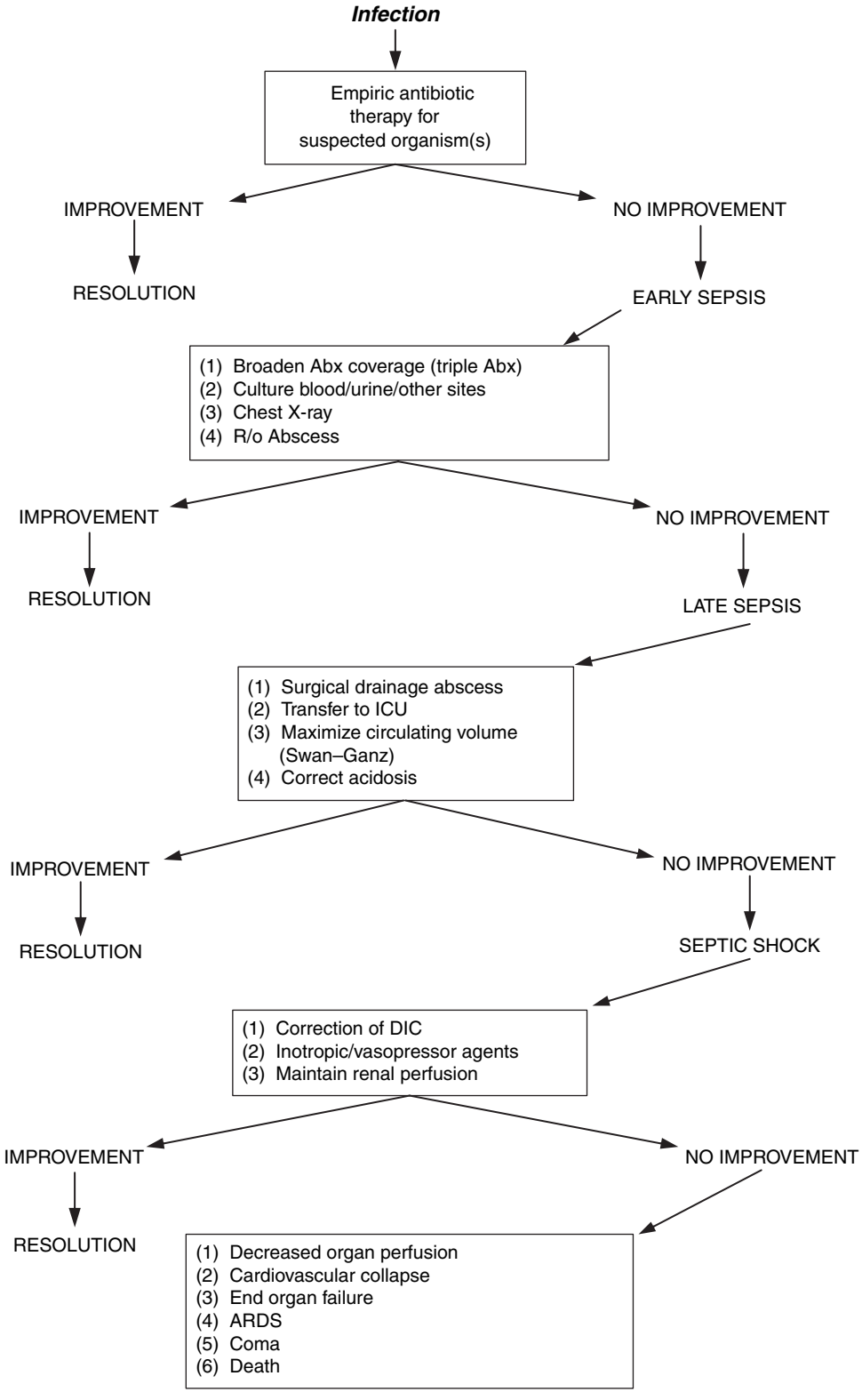
Early (simple sepsis)
(1) Maintain adequate oxygenation (supplemental O2)
(2) Maintain adequate circulating volume (IV fluids)
(3) Obtain appropriate laboratory data
(4) Initiate appropriate antibiotics (broad-spectrum)

Late (severe sepsis/shock)
(5) Transfer to Intensive Care (Swan–Ganz catheter)
(6) Surgical removal/drainage of abscess or infected tissue
(7) Tailor antibiotic coverage to culture results
(8) Institute inotropic/vasopressor agents

Antibiotic regimens

(1) Ampicillin 2 g IV q. 6 h + gentamicin (load: 2 mg/kg, maintenance: 1.5 mg/kg IV q. 8 h) + clindamycin 900 mg IV q. 8 h
(2) 3rd generation cephalosporin (cefotaxime 2.0 g IV q. 4 h or ceftriaxone 2.0 g IV q. 12 h or ceftazidime 2.0 g IV q. 8 h) + gentamicin (dose as in #1)
(3) Ticarcillin/clavulanate 6.2 g IV q. 6 h or piperacillin/tazobactam 6.75 g IV q. 6 h + gentamicin (dose as in #1)
(4) Cefoxitin 2.0 g IV q. 8 h + gentamicin (dose as in #1)

(PCN/cephalosporin allergic):
(5) Imipenem 500 mg IV q. 6 h
(6) Aztreonam 2.0 g IV q. 6 h + gentamicin (dose as in #1) + clindamycin 900 mg IV q. 8 h


Infection
Empiric antibiotic therapy for suspected organism(s)
IMPROVEMENT
RESOLUTION
NO IMPROVEMENT
EARLY SEPSIS
(1) Broaden Abx coverage (triple Abx)
(2) Culture blood/urine/other sites
(3) Chest X-ray
(4) R/o Abscess
IMPROVEMENT
RESOLUTION
NO IMPROVEMENT
LATE SEPSIS
(1) Surgical drainage abscess
(2) Transfer to ICU
(3) Maximize circulating volume (Swan–Ganz)
(4) Correct acidosis
IMPROVEMENT
RESOLUTION
NO IMPROVEMENT
SEPTIC SHOCK
(1) Correction of DIC
(2) Inotropic/vasopressor agents
(3) Maintain renal perfusion
IMPROVEMENT
RESOLUTION
NO IMPROVEMENT
(1) Decreased organ perfusion
(2) Cardiovascular collapse
(3) End organ failure
(4) ARDS
(5) Coma
(6) Death

SEPTIC SHOCK (SIRS – SYSTEMIC INFLAMMATORY RESPONSE)

Associated with a mortality in ICU of	20–50%
If ARDS develops, there is a mortality rate of	50%

SEQUENCES

Innermost	*Outermost*
Zona pellucida – granulosa	Theca interna
Responsive to FSH	Responsive to LH
Synthesizes estrogen	Synthesizes androstenedione products
Medulla cortex	Germinal epithelium

Estrogens in sequence of decreasing potency:
Estradiol, estrone, estriol

Most frequent sites of osteoporotic fractures:
Vertebra, distal radius, femoral neck

SEXUAL ASSAULT

See Assault

SEXUAL DYSFUNCTION

Definition

Sexual dysfunction is a chronic disturbance in the sexual response cycle

The overall prevalence rate of female sexual dysfunction has been reported as high as	43%
Compared to male sexual dysfunction at rate of	31%
What % of married women believe that a satisfying sex life is important?	84%

Detect with abbreviated interview

(1) Sexually active?
(2) Pain with sex?
(3) Problems or questions?

- "Is contraception an issue for you?"
- "Please tell me the nature of your sexual practices, whether by yourself or with others. I am asking because some sexual practices play a role in decisions @ diagnosis and treatment – I am not asking to pass judgment on anything you do."

Types of sexual dysfunction

(1) Disorders of desire or libido (low sexual desire)
  (a) Hypoactive sexual desire disorder (HSDD)
    Hormone deficiencies or neuropsychiatric disorders
  (b) Sexual aversion disorder (SAD)
    Childhood or painful sexual abuse or experiences
    Feelings of shame and guilt.
(2) Disorders of arousal (inability to attain or maintain physical response to sexual arousal)
  Causes can be:
  (a) Diabetes
  (b) Arteriosclerosis
  (c) Medications, e.g. many blood pressure and psychiatric drugs
(3) Orgasmic disorders (inability or delayed orgasm)
  Physical causes can be surgery, hormone deficiency, or medications such as antidepressants
(4) Pain disorders
  (a) Dyspareunia (pain during coitus)
    Insufficient vaginal lubrication, inflammation, endometriosis, or vaginal/pelvic infections
  (b) Vaginismus (spasm of vaginal muscles during coitus)
    Vaginal scarring (previous injuries, surgeries, childbirth), vaginal irritation or inflammation (douches, spermicides, latex condoms) or vaginal infection
  (c) Non-coital sexual pain

(5) Psychological causes
Sexual guilt, grief, trauma, depression, interpersonal conflict with a sexual partner

Symptoms

(1) HSDD – deficiency or absence of sexual fantasies or desire
SAD – phobic aversion to and avoidance of sexual contact with partner
(2) Inability to attain or maintain sexual excitement
(3) Primary orgasmic disorder – the patient has never experienced an orgasm
Secondary orgasmic disorder – the patient has recently become anorgasmic
(4) (a) Genital pain with intercourse
(b) Involuntary spasm of the muscles comprising the outer third of the vagina
(c) Genital pain with non-coital sexual stimulation

Possible CAUSES

(1) Side-effects of SSRIs (serotonin reuptake inhibitors)
(2) Tricyclic antidepressants
(3) Antihypertensives
(4) Benzodiazepines
(5) Adrenal insufficiency
(6) Relationship problems
(7) Pelvic organ prolapse (POP) is likely to result in sexual dysfunction (Barber MD, Visco AG, Wyman JF, *et al*. Sexual function in women with urinary incontinence and pelvic organ prolapse. *Obstet Gynecol* 2002;99:281–9) as compared to urinary incontinence, which is less likely to result in sexual inactivity than POP
(8) Cimetidine
(9) Bromocriptine
(10) Spironolactone
(11) Tamoxifen
(12) Cancer
(13) Ovaries in the cul-de-sac
(14) Pelvic infection, fibroids, endometriosis
(15) Hypoestrogenism
(16) Chronic diseases
(17) Other conditions (pregnancy, lactation, menopause)

Treatment (depends on etiology)

(1) HSDD – trial of testosterone especially in menopausal women to increase libido and clitoral sensitivity. EROS – CTD is a “clitoris pump” that is FDA approved; it uses a suction cup/hand-help vacuum device to increase blood flow to the clitoris. ERT sometimes increases libido, improves clitoral sensation, and decreases pain during intercourse for women in menopause. Topical creams and Femring or Estrings can also help with vaginal irritation, pain, or dryness
SAD – refer for counseling
(2) Treat underlying physical disorder. Consider sildenafil, local vasodilating agents and appropriate estrogen replacement. Refer for counseling if necessary. There is a nasal spray that looks promising for the treatment of female sexual arousal disorder called bremelanotide (PT-141). It directly stimulates the brain’s sexual control center. Women who have used it in clinical trials report feeling “genital warmth, tingling and throbbing,” as well as “a strong desire to have sex.” It is not yet approved by the FDA as of this publication
(3) Correct underlying pharmacologic problem and/or refer for sexual or psychological counseling and look for OTC and herbal supplements as possible etiological agents
(4) Correct underlying perineal trauma (eliminate soaps and harsh chemicals) and medical conditions (infection and endometriosis). Vaginal dilators can be inserted into the vagina for 15 minutes, twice daily, to treat vaginismus. Kegel exercises and techniques to relax the vaginal muscles and relieve orgasmic disorders and vaginismus. *See* Pelvic (Kegel) exercises

(5) Try physical therapy (pelvic-floor biofeedback). Refer for sexual and/or psychological counseling. Search for history of abuse or molestation in these women. Pelvic pain is often multifactorial

Alternative therapies

(1) DHEA 50 mg/day for 12 months (Baulieu EE, Thomas G, Legrain S, *et al*. Dehydroepiandrosterone (DHEA), DHEA sulfate and aging: contribution of the DHEAge Study to a sociobiomedical issue. *Proc Natl Acad Sci USA* 2000;97:4279–84). Increases libido and sexual satisfaction but can increase androgens, decrease HDL, SHBG and can be correlated with increased CHD. More study needed for this along with *Ginkgo biloba*, yohimbine and arginine

(2) EROS–CTD
The EROS clitoral therapy device (EROS–CTD) is the first FDA-approved device for female sexual dysfunction
The EROS–CTD patients reported what % improvement of:

Increased clitoral sensation?	90%
Greater vaginal lubrication?	80%
Improved ability to have an orgasm	55%
Higher overall sexual satisfaction	80%

SEXUAL RESPONSE

Excitement	Vagina lubricates, lengthens + distends, tension increases, skin flushes, breasts engorge	
Plateau	Vagina decreases in diameter by	50%
	Vaginal inner two-thirds distends, clitoris retracts, systolic B/P increases, breasts AND areolas engorge	
Orgasm	Vagina contracts strongly at how many second intervals?	0.8 s
	How many times does it contract?	5–10 ×
	Cervix dilates, hyperventilation, tachycardia at rate of	110–180 BPM
Resolution	Returns to normal	

SEXUALLY TRANSMITTED DISEASES

Genital ulcers

Feature	*Syphilis*	*Herpes*	*Chancroid*	*LGV*	*Granuloma inguinale*
Incubation	2–4 weeks	2–7 days	1–14 days	3 days–6 weeks	1–4 weeks
Pain	Rare	Common	VERY tender	Varies	Uncommon
Lymph nodes	Firm, NT	Firm, NT	Tender, Sup	Tender	Pseudoadenopathy
	Bilat	Bilat	Usu unilat	Sup, loc	
Characteristics	*T. pallidum*	Resides dorsal root ganglia	*Hemophilusi ducreyi*	*Chlamydia trachomatis*	*Calymmatobacterium granulomatis*
Diagnosis	Dark field microscopy	Cultures WBA	Gram stain "School of fish" culture	Complement fixation or culture Multiple fissures of perineum/ rectum	Find Donovan bodies
Treatment	Penicillin B 2.4 million u	Acyclovir	Rocephin or erythromycin	Doxycycline	Tetracycline

Pelvic inflammatory disease (PID)

Risk factors for PID	Age 14–24 (One-third of U.S. girls are sexually active by age 15)
	Sexually active
	Multiple sex partners

New sex partner
Hx of STD
Hx of PID
Use of an IUD for contraception
Nulliparity
Onset of pain during or within 1 week of menses
Cigarette, alcohol or illicit drug use
Pelvic instrumentation

Criteria for clinical diagnosis of PID

Minimum criteria for clinical Dx (all three must be present)
Lower abdominal tenderness
Bilateral adnexal tenderness
Cervical motion tenderness

Additional criteria useful in Dx (one or more necessary for dx)
Oral temp > 101°F (> 38.3°C)
Abnormal cervical or vaginal discharge
Elevated ESR or C-reactive protein
WBC > 10 500
Evidence of cervical infection with *Neisseria gonorrhoeae* or *Chlamydia trachomatis*
Tubo-ovarian abscess on sonography or radiologic test
Laparoscopic abnormalities consistent with PID
Histopathologic evidence on endometrial biopsy

CDC criteria for hospital admission
Adolescent patient
Concurrent HIV infection
Dx of PID uncertain
Failure of outpatient treatment
Inability of patient to follow or tolerate outpatient regimen
Inability to exclude surgical emergency
Pregnancy
Severe illness or nausea and vomiting
Suspected pelvic abscess
Uncertainty about clinical f/u within 24 h of starting antibiotic tx
All nulliparous women

Inpatient treatment guidelines

Regimen A
Cefoxitin sodium (Mefoxin), 2 g IV q. 6 h, or
Cefotetan disodium (Cefotan), 2 g IV q. 12 h, plus
Doxycycline 100 mg IV (Vibramycin IV) q. 12 h
– Continue this regimen for at least 48 h after clinical improvement
– After discharge, the patient continues doxycycline 100 mg p.o. b.i.d. for a total of 14 days

Regimen B
Clindamycin 900 mg IV q. 8 h, plus
Gentamicin in IV or IM (loading dose of 2 mg/kg of body weight followed by a maintenance dose of 1.5 mg/kg q. 8 h
– Continue this regimen for at least 48 h after clinical improvement
– After discharge, the pt is given doxycycline 100 mg p.o. b.i.d. or clindamycin 450 mg p.o. q.i.d. for 14 days

Outpatient treatment guidelines

Regimen A
Cefoxitin 2 g IM; plus probenecid (Benemid), 1 g p.o. concurrently, or
Ceftriaxone (Rocephin), 250 mg IM, plus
Doxycycline 100 mg p.o. b.i.d. for 14 days or Zithromax 1 g p.o.

Regimen B
Ofloxacin 400 mg p.o. b.i.d. for 14 days, plus
Clindamycin 450 mg p.o. q.i.d. for 14 days, or
Metronidazole 500 mg p.o. b.i.d. for 14 days

SHEEHAN'S SYNDROME

Postpartum pituitary necrosis due to postpartum hemorrhage

Symptoms

Amenorrhea, fatigue, galactorrhea, decrease axillary and pubic hair

Diagnosis

Draw CORTISOL level STAT!

Treatment

Hydrocortisone IV or 100 mg
Dexamethasone IM (does not interfere with cortisol assay)
Also give:

Cortisone acetate (Cortone®)	25 mg/day
or prednisone	5 mg/day
Fludrocortisone (Florinef®) for mineralocorticoid replacement	0.1 mg/day

Levothyroxine? GH? estrogen/progesterone?
FSH/LH if patient wants pregnancy

SHOCK

Normal CVP is 4 + or –2

Central monitoring

(1) Peripheral artery
(2) CVP – R internal jugular vein
(3) Swan–Ganz (pulmonary artery cath)

Most common cause of shock = blood volume deficiency

CVP decrease with	*CVP increase with*
Sepsis	Right ventricular failure
Shock	Cardiac tamponade
Anaphylaxis	Pulmonary embolus
Inadequate vascular volume	Fluid overload

Types of shock

Hypovolemic (hemorrhagic)
Distributive (septic)
Cardiogenic shock
Extracardiac obstructive shock

Order

Oxygenate, restoration of circulatory volume, drug therapy, evaluation and remedy basic problem

SHOULDER DYSTOCIA

Planned C-section may be reasonable for diabetic pt with EFW between	4250–4500 g
Less than what % of all deliveries complicated by SD will result in permanent brachial plexus injury?	10%

It appears that intrauterine brachial plexus palsy (BPP) not associated with shoulder dystocia is almost always temporary, whereas almost all permanent BPP is associated with shoulder dystocia (Gurewitsch ED, Johnson E, Hamzehzadeh S, Allen RH. Risk factors for brachial plexus injury with and without shoulder dystocia. *Am J Obstet Gynecol* 2006; 194: 486–92)

Macrosomia infants > 4500 g comprise only this % of pregnancies	0.4%
If the patient is diabetic, obese and post-term, the risk for macrosomia is	5–15%
Define shoulder dystocia – delivery of shoulder after delivery of head that exceeds	60 s
How much time does one have to deliver the shoulders after the head before hypoxia sets in?	2½ min or 150 s

Treatment plan

(1) Call for help
(2) Gentle traction
(3) Drain bladder p.r.n.
(4) Suprapubic pressure
(5) McRobert's maneuver
(6) Episiotomy or extension of episiotomy
(7) Wood's maneuver (corkscrew)
(8) Mazanti maneuver (delivery of posterior arm)
(9) Fracture outer third of clavicle–mid humerus p.r.n.
(10) Zavanelli maneuver followed by C-section

Important tips

(1) Most shoulder dystocia cannot be prevented
(2) Risk is increased with obesity and diabetes
(3) Erb's palsy is caused by stretching of C5–C6
(4) Klumpke's palsy is caused by stretching of C8–T1
(5) C-sections on all macrosomic fetuses is NOT appropriate
(6) Elective C-sections are reasonable for diabetics with EFW > 4250 g
(7) Injuries are common with SD but only what % are permanent? 10%
(8) Ultrasound measurements have limited accuracy
(9) AVOID EXCESSIVE TRACTION
(10) Shoulder dystocia is the most prominent risk factor for brachial plexus palsy in the setting of vacuum extraction

SICKLE CELL DISEASE

Autosomal recessive–common types SS, SC, Sb thalassemia
Cause for HgbS is single substitution of VALINE for GLUTAMIC ACID
RBCs that normally have half-life of 120 days now only 5–10 days
The RBCs become sickle-shaped sludge in small blood vessels – ischemia + infarction – pain
Sickling triggered by decreased oxygen tension and acidosis

Incidence

How many African-Americans have the trait? 1 in 12
If two parents have trait, what is chance that child will have SC disease? 25%
How many African-Americans have the disease? 1/600
Hgb C in African-American is present in 1/40–1/50

Diagnosed by

Hemoglobin electrophoresis

Increased risks for crisis

Pyelo and decreased urine concentration
Pulmonary infarction
Infection (spleen)
Cholelithiasis with increased stones
Poor perinatal outcome
Spontaneous abortion, stillbirth, pre-term birth or IUGR

During pregnancy

Screen for UTIs frequently
Pneumococcus vaccine early in life
Check iron levels
Give folic acid
Check B/P often secondary to increased risk of PIH
Serial ultrasounds
Serial NSTs
Vaginal deliveries preferable

Treatment

Analgesia, oxygen and hydration. Transfusion if necessary

SINUSITIS

Acute < 1 month
Subacute 1–3 months
Chronic > 3 months

Sinusitis is the fifth most common diagnosis for which clinicians prescribe antibiotics in the ambulatory setting. First-line antibiotics (defined as amoxicillin, TMP–SMX and erythromycin) had identical success rates compared to second-line treatments (90.1% vs 90.8%) and relapse rates were similarly indistinguishable (3.3% vs 3.5%). However, mean cost between first-line Rx ($68.98) vs second-line Rx ($135.17) were about half the cost. It is recommended that first-line therapies be used first. (Piccirillo JF, Mager DE, Frise ME, *et al*. Impact of first-line antibiotics for the treatment of acute uncomplicated sinusitis. *JAMA* 2001;286:1849–56)

Sinuses MOST involved

Maxillary, anterior ethmoidal, frontal

Symptoms

Cough, nasal discharge, bad breath, facial pain, low-grade fever

Diagnosis

Secretions, CT

Pathology

Streptococci, *Hemophilus*, *Moraxella*

Treatment
Ampicillin, amoxicillin, TMP–SMX, erythromycin
Use antibiotic with β-lactamase activity if necessary after 14 days

SINUSOIDAL HEART RATE

CNS, absence of autonomic nervous system control over heart, high output failure or tissue hypoxia of the fetal heart

SKIN CANCERS

Basal cell, squamous cell, superficial spreading melanoma and acral-lentiginous melanoma
Familiarize oneself with gross appearances of these

Basal cell
Most common skin cancer of light-complexioned people
Ring with central depression. Larger lesions have rolled border. Often found on face, especially the nose

Squamous cell
Usually found on hands and/or limbs. Increased incidence in black patients. Dull, red and crusted

Superficial spreading melanoma
Most common type of melanoma

Acral-lentiginous melanoma
Irregular black macule. Often found on toes. Increased incidence in Asians, Blacks, Hispanics and Indians

SLING PROCEDURE

Indication for treatment
ISD (intrinsic sphincter deficiency) with urethral hypermobility and SUI (stress urinary incontinence)
(Burch is for defect in endopelvic fascia)

Technique for sling
Various grafts
There are 30 different synthetic midurethral slings on the market. Some of the better known are below:

(a) INFLUENCE FASCIAL ALLOGRAFT or
TUTOPLAST are human freeze-dried/solvent-dehydrated fascia lata

(b) REPLIFORM (Lifecell Corporation, Woodlands, TX; distributed by Boston Scientific, Urology/Gynecology, Natick, MA) or ALLODERM are decellularized human cadaveric dermis
STRATASIS (porcine small intestinal submucosa)

(c) PELVICOL, IN-FIRST ULTRA (secured with bone anchors), INTEXENE (acellular collagen matrix–porcine dermal xenografts)
The author soaks his graft in an antibiotic solution for 20 min prior to use. Relatively little info is available to support or discourage the use of xenograft materials in sling procedures

(d) PELVISOFT (acellular collagen matrix) More porous, less stiff and softer to use than PELVICOL.

(e) Multifilament and small-pore mesh products such as OBTAPE, TVT-O, MONARC, IVS all have erosion rates of 1.8–17%. A consensus may be emerging that the safest synthetic material is monofilament polypropylene with pore size larger than 70 μm

(f) Avoid cadaveric fascia, as more complications and re-operations occurred with this compared to autologous rectus fascial slings (Howden NS, Zyczynski HM, Moalli PA, *et al*. Comparison of autologous rectus fascia and cadaveric fascia in pubovaginal sling continence outcomes. *Am J Obstet Gynecol* 2006; 194: 1444-9

Synthetic grafts tend to have slightly higher success rates; biologic grafts tend to be better tolerated

Various methods

(a) BONE SCREW
Dissect perivesical and periurethral fascia. After perivesical fascia are mobilized, then the BONE of the pubic rami is cleaned with sponge. Titanium (Precision Speed Tack or Precision Twist, Boston Scientific, Urology/Gynecology, Natick, MA) bone screws with #1 Prolene drilled into bone of posterior pubic rami. Prolene is threaded through #18 gauge needle and graft. #2–0 Vicryl placed into suture graft then # 0 Vicryl on Uro-6 needle Gortex sutured at angle

(b) TVT (tension-free vaginal tape) Graph is placed under middle third of urethra to elevate via various needle-threading kits that are available. This technique is done behind the vaginal mucosa

GYNECARE TVT has the most evidence and longest follow-up available in the literature. The company markets all 3 approaches, including vaginal, abdominal ("top-down"), and obturator. (In a retrospective case series by Gandhi *et al* and a randomized trial by Lord *et al*, Gynecare TVT had better continence outcomes compared with SPARC)

(c) TOT (tension-free obturator tape)
Graft is placed under urethra via the obturator canal. The outside-in technique results in the mesh being placed farther from the obturator canal and closer to the ischiopubic ramus, theoretically reducing the risk of neurovascular injury

Anterior or posterior repairs can be done in conjunction with any of these (a, b, or c)

Suprapubic catheter is an excellent choice to be used if urinary retention is anticipated postoperatively in any of these procedures. Cystoscopy should be done during and after these procedures to check for inadvertent bladder injury. If perforation with trocar occurs, check ureteral orifices for efflux from both. Perforations to the bladder dome, anterior or lateral bladder neck usually heal spontaneously and require no extended bladder drainage. **To understand how to perform these methods in detail**, *see* Turrentine JE, *Surgical Transcriptions and Pearls of Obstetrics and Gynecology*, 2nd edn. London: Informa Healthcare, 2006

Midurethral slings: In regards to whether retropubic or transobturator slings are better, randomized trials will be out by 2008 and 2009 to determine both objective and subjective treatment success

Experience and anesthesia

Both general anesthesia and the inexperience of the surgeon with the TVT procedure have negative effects on outcome. Schraffordt Koops SE, Bisseling TM, van Brummen HJ, Heintz APM, and Vervest HAM (What Determines a Successful Tension-Free vaginal tape? A prospective multicenter cohort study: Results from The Netherlands TVT database. *Am J Obstet Gynecol* 2006; 194: 65–74) believe that only experienced surgeons should perform TVT procedures. The success rate for experienced surgeons was 72.4% at the 2-year interval, compared to the 61.7% for surgeons during their first 10 procedures. The article contains very extensive tables worthy of review by any surgeon who is performing this TVT. The negative influence of general anesthesia on success of the TVT procedure was not explained

SMOKING

What % of reproductive women smoke?	30%
What % of ALL CANCERS are secondary to smoking?	30%
What % of cardiac deaths in women under the age of 65 years old are secondary to smoking?	55%
Quitters usually relapse within what amount of time?	1 week
Women most often begin to smoke during what years of age?	11–14

SPERM OR SEMEN (ABNORMAL)

Hypospermia or oligospermia	< 2 ml volume
Hyperspermia	> 6 ml volume
Aspermia	Absence of semen
Azoospermia	Absence of sperm in semen
Oligozoospermia	< 20 million sperm/ml
Polyzoospermia	> 250 million sperm/ml
Asthenozoospermia	< 50% of sperm with forward progression
Teratozoospermia	> 60% abnormal sperm

Environmental toxins

Sulfasalazine – sperm count and motility decreased
Chemotherapy – count decreases with germ cell destruction – FSH increase
Alcohol – inhibits Leydig cell biosynthesis. Testosterone – decrease
Chemical toxicants (DBCP, metals, lead, cadmium, mercury)
Toxic to all parts of testes. Pesticides cause azoospermia, oligospermia and decreased FSH, LH, low to normal testosterone, normal estradiol

Sperm antibodies

(1) 3–7% of men presenting for fertility evaluation have significant titers of sperm antibodies that are responsible for their infertility
(2) Approximately half men develop sperm antibodies in serum after a vasectomy
(3) A "shaking" pattern in non-progressively motile sperm suggests presence of sperm antibodies in either partner

SPERM ANTIBODIES

What % of males presenting for fertility evaluation have significant titers of sperm antibodies that are responsible for their infertility?	3–7%
What % of men develop sperm antibodies in their semen after a vasectomy?	@1/2

A "shaking" pattern in non-progressively motile sperm suggests presence of sperm antibodies in either partner

SPIEGELBERG'S CRITERIA FOR OVARIAN PREGNANCY

(1) Tube + fimbria must be intact
(2) Gestational sac must occupy normal ovarian position
(3) Sac must be connected to uterus by utero-ovarian ligament
(4) Ovarian tissue must be identified histologically in the wall of the gestational sac

SPINAL CORD INJURY

• Fertility NOT affected but common problems include:	
UTI	80%
Anemia	63%
Pressure sores	26%
• Patient with spinal cord transection at what segment may have painless labor?	>T10
• Anesthesia should be used to prevent autonomic dysreflexia (blocks stimuli arising from organs)	
• Vaginal delivery can be expected	
• Spinal cord injuries occur in ages 15–25 in what % of the time?	50%
• What % of this age group of SCIs are female?	15%
• If SCI is above or at T5, what % patients are subject to AUTONOMIC DYSREFLEXIA?	85%

• Stimulus is unmodified by supraspinal centers thus catacholamine release – vasoconstriction

Symptoms

- Increased B/P associated with HA, bradycardia, arrhythmia, sweating, nasal congestion, resp distress, fetal hypoxia. AVOID stimulation of vagina, bladder or bowel. GIVE EPIDURAL

STD TREATMENT

Chlamydia	Azithromycin p.o. as one dose	1 g
	Amoxicillin p.o. t.i.d. × 7 days	500 mg
Gonorrhea	Ceftriaxone IM in single dose	125 mg
	Cefixime orally as single dose	400 mg
	Spectinomycin IM in single dose	2 g

STERILE WATER PAPULES

Intradermal/intracutaneous water injection for relief of back pain in labor, whiplash, renal pain. Success rate is 89%

How many women suffer from severe low-back pain in labor? 1/3
What predicts back pain in labor? History of back pain during menses and/or during pregnancy
Incidence of fetus entering the pelvis in an OP position is up to 30%
Persistent posterior positions occur in approximately what % of all labors? 5%
ROP is estimated to be how many times more common than LOP? 5 ×

Technique

Locate four specific sites lateral to sacrum and below iliac crest. (Many women have an indentation on their sacrum at this point.)
Mark with pen – next two sites are 2–3 cm below and 1–2 cm medial.
Inject 0.1–0.15 ml intradermally. *Warn patient in advance* about sting of injection site that lasts about 30 s
Relief is within about 2 min after injection
Effect of injection lasts from 1–3 h and may be repeated
Best to have two people administering injections during contraction

Mechanisms of action of SWP

(1) Cause distention of skin, stimulating nociceptors and mechanoreceptors. Stimulates fast-conducting A fibers as in gate control theory
(2) "Counterirritation" theory as with TENS
(3) Release of β-endorphins

STERILIZATION METHODS

Vaginal colpotomy

Minimally invasive with small incision made under cervix into cul-de-sac. Small valentine to lift uterus, long allis clamps, jet pack, plain ties for tubes, and suture for closure of vaginal colpotomy

Abdominal mini-lap or at time of C-section

Irving – tube is buried in broad ligament
Pomeroy – tube is brought up into a loop, tied with plain suture, and loop is cut
Parkland – wide piece of tube is cut and tied in two separate sections
Madlener – tube is brought up into a loop and tied with suture
Kroener – fimbriectomy

Laparoscopy

Tubes are divided, cauterized, clipped (Hulka), or banded (Falope Rings)

Hysteroscopy

Essure – a microinsert of flexible stainless steel inner coil and outer coil of nickel titanium alloy (nitinol), and an innermost layer of polyethylene terephthalate (PET) fibers. These fibers gradually elicit a benign localized tissue in growth that occludes the tubal lumen. (*See also* Essure; also Turrentine JE. *Surgical Transcriptions and Pearls of Obstetrics and Gynecology.* London: Informa Healthcare, 2006)

STEROIDS

Betamethasone 12 mg IM × 2 doses	24 h apart
Dexamethasone 6 mg IM × 4 doses	12 h apart
Beta and dexamethasone are alike in structure, placental transport, with little or no mineralocorticoid activity and half-life of how many hours?	72 h

Repeat doses?

Markedly reduce maternal basal cortisol levels – could cause maternal adrenal suppression, an effect that could be of concern during the stress of labor and delivery. Repeat doses should only be used in those pregnancies at the *highest risk for pre-term delivery*

- National Institute of Child Health and Human Development convened a Consensus Panel in August 2000 – concerned about adverse affects on neurological development and growth without clear evidence of benefit. Panel concluded that use of repeated steroids should now only be used in research studies
- There is no improvement in neonatal morbidity with weekly administration of antenatal corticosteroids compared to a single course of corticosteroids. (Guinn DA, Atkinson MW, Sullivan L, *et al*. Single vs weekly courses of antenatal corticosteroids for women at risk of preterm delivery: a randomized controlled trial. *J Am Med Assoc* 2001;286:1581–7)

What weeks of gestation is it best to administer steroids?	24–34 weeks
Decreased RDS born at	29–34 weeks
Decreased severity of RDS	24–28 weeks
Decreased incidence of IVH	24–28 weeks
Give with ROM if no chorioamnionitis	< 30–32 weeks
Increased risk unless delivery imminent or danger to mother	< 34 weeks

Which steroid is better?

One randomized, double-blind study presented at the annual Meeting of the Society for Maternal–Fetal Medicine showed that prenatal dexamethasone is superior to betamethasone in its reduction of two major neonatal morbidity and mortality outcomes – intraventricular hemorrhage and periventricular leukomalacia

STILLBIRTHS

H&P, photography if possible, mat TORCH, obtain placenta, membranes and cord of at least	3 ml
Analysis of bile, vitreous humor and urine. Tissue – get how much skin?	1 cm^2

Place in sterile NS or medium at room temperature. Autopsy if possible – reach consensus

STROMAL SARCOMA

Most common preoperative diagnosis in patients with LGSS is myomata uteri

In patients with LGSS, extrauterine tumor is present in approximately how many cases?	1/3

Among patients with LGSS, a higher recurrence rate is reported in patients with residual ovarian tumor

LGSS histology includes

Proliferation of uniform, benign-appearing, stromal cells
Whorling pattern around tumor vessels
Mitoses
Infiltrative margins

Most effective means of prolonging the progression-free interval among patients with advanced LGSS is postoperative progestational therapy

STRUMA OVARII

What % of teratomas?	2–3%
Usually measure less than what diameter?	10 cm

Thyrotoxicosis develops in < 5%
If metastasis present, treat with ^{131}I

Carcinoids (histologically resemble GI tract, unilateral, ovarian teratoma) % true carcinoid 30%
Metabolite of serotonin can be measured in urine 5-HIAA

STUCK TWIN SYNDROME

Severe form of TTTS with absence of amniotic fluid in donor's sac
Membrane cannot be visualized because it is so closely wrapped against the donor twin. Rule out monoamniotic twin gestation – difficult to do this sometimes

SUBTOTAL (SUPRACERVICAL) HYSTERECTOMY

Usually this is done only in last resort when concerned with:
(1) Increased blood loss
(2) Anatomic distortion
(3) Injury to pelvic floor
(4) Precarious condition of patient
Most common reason to leave cervix – limit surgical risk

Disadvantages	*Advantages*
Cervix can become inflamed	Avoidance of injury to pelvis
Cervix can cause discharge	Limits surgical risks
Mucocele can form	Decreased injury to urethra, bladder, etc.
Can become precancer	Preservation of sexual function
Can develop cervical cancer	Absence of granulation tissue
Need for continued Paps	Decreases infectious morbidity

SUCCENTURIATE PLACENTA

One or more accessory lobes distant from main placenta
HEMORRHAGE! Incidence of succenturiate placenta 3%

SUTURE

	Tensile strength and degree of inflammatory response	*Dissolves*
Natural fibers		
Plain catgut*	(00 size) 7 lb, losing half strength in 4–6 days; high	70 days
Chromic catgut	(00 size) 8 lb, losing half strength in 10–14 days; high	90 days
Synthetic fibers		
Polyglycolic acid and coated polyglactin-910	(00 size) 9.6 lb, losing half strength in 21 days; low	60–90 days
Pretreated coated polyglactin-910**	(00 size) 9.9 lb, losing half strength in 5 days; low	42 days
Dexon	Losing half strength in 14 days	
Maxon	Losing half strength in 21 days	
PDS***	Losing half strength in 42 days	

*Suggested for Tubal ligation; **Suggested for episiotomy repair; ***Suggested for vertical abdominal fascial closure

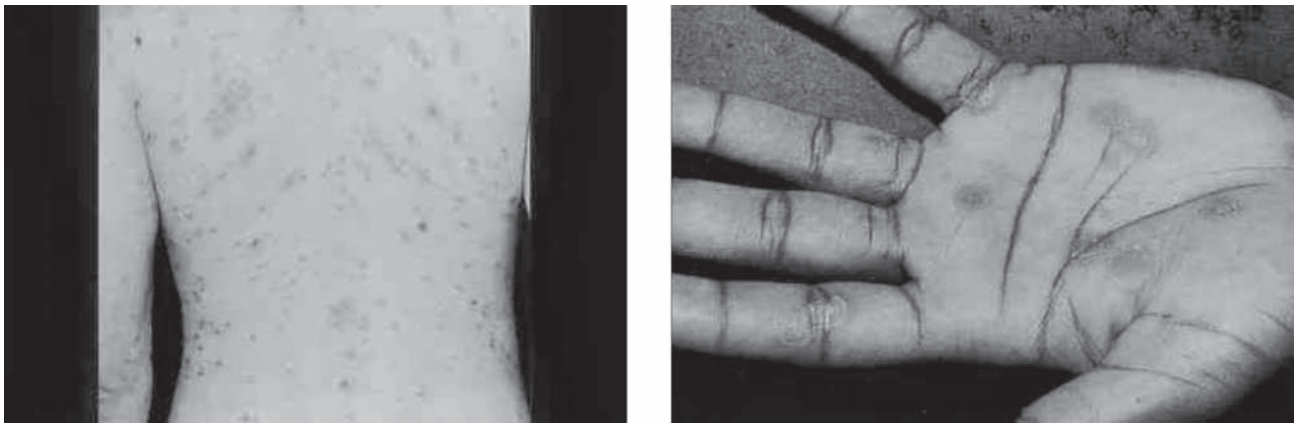
SYPHILIS

- Hard chancre in primary syphilis can be seen within how many weeks of exposure? 3 weeks
- Condyloma latum and/or rash in secondary syphilis can be seen @ 6 weeks to 6 months

- Positive serology is present between 4–6 weeks
- Latent stage or tertiary syphilis is seen between 2–20 years
- What % of patients develop CNS, cardiac and muscle abnormalities? 33%
  Gummas – skin + bone. Optic atrophy and aneurysms

Treatment

- For primary and secondary syphilis – benzathine PCN IM × 1 dose 2.4 million units
- A second dose is given a week later if pt is pregnant to prevent congenital syphilis in 98%
- For tertiary syphilis – benzathine PCN IM × 3 doses weekly for a total dose of 7.2 million units
- Weekly doses would be 2.4 million units
- Alternate dosing for penicillin allergies:
  Doxycycline b.i.d. for 2 weeks 100 mg
  Tetracycline q.i.d. for 2 weeks 500 mg
- If syphilis duration >1 year, give doxycycline or tetracycline for 4 weeks
- If pregnant, desensitization needed to give PCN
- For neurosyphilis – daily aqueous crystalline PCN G in doses of 12–24 million units
  Or how much every 4 h × 10–14 days 2–4 million units
  Or how much procaine PCN IM daily × 10–14 days 2.4 million units
  Plus PROBENECID q.i.d. × 10–14 days 500 mg



(a) (b)

Figure 21 Characteristic rash of secondary syphilis: (a) on back; (b) palmar rash

TAMOXIFEN

- Non-steroidal with potent antiestrogen properties
- TRIPHENYLETHYLENE
- Competes with circulating estrogens or binding to estrogen receptors
- Used in treatment for metastatic breast cancer and adjuvant treatment of breast cancer especially with negative nodes and + estrogen receptors

Prophylaxis

- Multiple primary relatives with breast cancer, history of lobular CIS of breast or osteoporosis to increase BMD
- Prevention trial showed what % decrease in occurrence of primary disease in high-risk patient? 49%
- GAIL model defines high risk as 35 years or > with 5-year predicted risk of breast cancer of 1.67%
- Decreases LDL, increases BMD, no effect on HDL and increases endometrial cancer by 2 ×
- Decreases cardiac events but may slightly increase thromboembolic events. Optic changes including cataracts

Decreases vertebral fractures by 48%

Endo Bx if patient experiences bleeding. What endometrial thickness measurement in a postmenopausal woman correlates with atrophic histological changes? ≤ 4–5 mm

TANNER STAGING

Prepubertal		Stage I
9.8 years	Small mound – sparse pubic hair by 10.5 years	Stage II
11.2 years	Enlargement but no sep of breast and areola. Dark, coarse on mons 11.4 years	Stage III
12.1 years	Mound of areola. Adult but lim to mons 12.1 years	Stage IV
	Recessed areola 14.6 years. Adult spread dist 13.7 years	Stage V

TAY–SACHS

Autosomal recessive

Lysosomal storage disease in which GM2 gangliosides accumulate throughout body

Frequency of Tay–Sach carriers in Jews of East European descent (Ashkenazi) is @ 1/30

People of French-Canadian and Cajun descent also have greater carrier frequency than general population

TEMPERATURE CONVERSIONS

Centigrade to Fahrenheit –	Multiply by	1.8
	and add	32
Fahrenheit to Centigrade –	Subtract	32
	and multiply by	0.555

TESTOSTERONE

- Normal reproductive range is 20–80 ng/dl
- There is a powerful placebo response that patients experience when placed on testosterone. Data thus far indicate that only superphysiological testosterone can produce sexuality and psychological effects. However, a study adapted from Davis and colleagues (Davis SR, McCloud P, Strauss BJ, *et al*. Testosterone enhances estradiol's effects on postmenopausal bone density and sexuality. *Maturitas* 1995;21:227–36) demonstrated that an estrogen (esterified estrogen 1.25 mg) in combination with methyltestosterone 2.5 mg improved sexuality score statistics better than the same dose of estrogen alone. This added testosterone improved all elements of the

score including libido, activity, satisfaction, pleasure, fantasy orgasm and relevancy

- Testosterone, methyltestosterone and NETA (norethindrone acetate) all decrease SHBG thus increasing free bioavailable testosterone and estrogen
- If symptomatic relief of hot flushes is not achieved after 4 weeks, usually an estrogen/androgen therapy can resolve the problems prior to increasing estrogen dose. Switching to a transdermal estrogen can also avoid the protein-binding properties of SHBG
- Androgen-deficiency syndrome is usually treated by adding testosterone to hormone regimen. This mainly increases bone density but also increases the bioavailability of estrogen because of the decrease in SHBG. Osteoporosis with failure to respond to postmenopausal estrogen therapy or a patient with low turnover osteoporosis should be switched to an estrogen/androgen combination therapy

Available	Intramuscular	250 mg/cc for men 125 mg/cc for women
	Sublingual tablets	10 mg, 25 mg, 50 mg for men 0.625 mg, 1.25 mg, 2.5 mg for women (College Pharmacy at 800 888-9358)
	Subcutaneous pellets	75 mg
	Oral (methyltestosterone in combo with premarin) (Half strength or full strength) by Solvay	Estratest

THELARCHE

Normal	LH and FSH	3–6 mIU/ml
	Estradiol	< 20 pg/ml
	Bone age	+ or – 1
	Ultrasound of ovaries	1 × 1 × 1
Gonadal dysgenesis	LH and FSH	up to 10 + 14 mIU/ml
	Estradiol	< 20 pg/ml
	Bone age	1.5
	Ultrasound of ovaries	not visualized
Isosexual precocious puberty	LH and FSH	9 + 8 mIU/ml
	Estradiol	up to over 42 pg/ml
	Bone age	up to over 4.5
	Ultrasound of ovaries	2 × 2 × 2
Premature thelarche	LH and FSH	3 + 4 mIU/ml
	Estradiol	< 20 pg/ml
	Bone age	1.5
	Ultrasound of ovaries	1.5 × 1.5 × 1
Precocious pseudopuberty	LH and FSH	< 3 + < 3 mIU/ml
	Estradiol	> 77 pg/ml
	Bone age	3
	Ultrasound of ovaries	unilateral enlargement 2 × 3 × 4

THROMBOCYTOPENIA

Neonatal alloimmune should be treated with	IVIG
Epidural is safe in patients with platelet counts	> 100 000
Mild maternal thrombocytopenia	≥ 70 000
In asymptomatic female is usually benign gestation thrombocytopenia – Rx with routine periodic repeat platelet counts.	
Platelets that are this rarely require therapy	≥ 50 000
Normal non-pregnant platelet count is	150 000–400 000
Normal	> 150 000
Mild thrombocytopenia	100 000–150 000

Moderate thrombocytopenia	50 000–100 000
Severe thrombocytopenia	< 50 000
Significant spontaneous bleeding	< 10 000
Excessive bleeding is associated with trauma or surgery is common	< 50 000
Gestational thrombocytopenia is found in what percent of pregnancies?	5%
Normally, gestational thrombocytopenia does not typically cause maternal, fetal, or neonatal complications	

Treatment

Treat with prednisone	1–2 mg/kg/day
For how many weeks and tapered over this period?	1–3 weeks
Give IVIG if platelet level	< 10 000
Or if platelets this low and bleeding	< 30 000
Splenectomy results in a complete remission of what % patients?	66%
Immunize for pneumococcus, *H. influenzae* and meningococcus	
Platelet infusion (10 000/μl per unit) to be given p.r.n. to control life-threatening hemorrhage or prep for surgery. Usually this many units are needed	6–10 units

Rule out

Preeclampsia and HELLP
HIV (In 10 % of HIV patients, thrombocytopenia is the first clinical finding, although it can present at any time later.

Work-up

CBC and peripheral smear – rule out drugs or other medical disorders then if > this level, probably gestational	70 000
if < this level, probably ITP	70 000
if < this level, most certainly ITP	50 000
Rule out DIC, PIH, TTP, hemolytic uremic syndrome, acute fatty liver if in what trimester?	Third

THROMBOPHILIAS

Inherited thrombophilias

(1) Coagulation inhibitors
   (a) Antithrombin III deficiency – binds with all serine protease coag factors except factor VII. Affects 0.02–0.2% of general population and confers risk of thromboembolism as high as 40% during pregnancy
   (b) Protein C and protein S deficiencies – inadequate levels result in increased fibrin production – clot. Protein C deficiency affects 0.2–0.5% of general population. Risk with FMH of thrombosis in pregnancy is 3–10% and 7–19% postpartum
   Protein C deficiency in general population is 0.08%. Risk of thrombosis in patients with + family history is 0–6% during pregnancy and 7–22% during postpartum

(2) Thrombophilias secondary to identifiable genemutations
   (a) Factor V Leiden mutation [amino acid substitution at position 506 (arginine → glutamine) results in loss of protein C cleavage site in factor V and accounts for high incidence of DVT]
   Prevalence in white population is 6–11% and approximately 1% in blacks. In patients with + family history of DVT, the risk in pregnancy for thrombosis is 10–14% and 19% in postpartum
   (b) Prothrombin gene mutation – (factor II or prothrombin stimulates coagulation by positive feedback loops and promotes anticoagulation via protein C pathway). The G20210A prothrombin gene mutation is associated with elevated levels of plasma prothrombin resulting in increased levels of fibrin and increased risk of thrombosis. Prevalence in general population is 2–6%

(c) Hyperhomocystinemia – established independent risk factor mostly caused by homozygosity for methylene-tetrahydrofolate reductase (MTHFR). 1–11% prevalence in general population
FOLIC ACID SUPPLEMENTS DECREASE HOMOCYSTEINE LEVELS

Pregnancy complications (other than DVTs, PE, and cerebral vein thrombosis) also include severe or recurrent PIH, abruption, IUGR and second- or third-trimester pregnancy losses, and stroke

Screening tests for recurrent histories of above include:

Factor V Leiden mutation
Prothrombin mutation
MTHFR mutation
Antithrombin III antigen activity levels
Protein C antigen activity levels
Protein S antigen activity levels (free and total)
(Protein C and S are not reliable tests *during pregnancy.* In addition, during extensive DVT or treatment with an anticoagulant – antithrombin III, protein C and S are also not reliable in that there are low levels)

Screen women with a prior adverse pregnancy outcome for thrombophilia; without treatment, their risk of another adverse outcome ranges from	66–83%
The risk of VTE during pregnancy and postpartum for women who have antithrombin deficiency and a history of VTE is roughly	40%

Acquired thrombophilias

(1) Antiphospholipid antibody syndrome
  (a) Lupus anticoagulant
  (b) Anticardiolipin antibodies
  (c) Activated protein C resistance
(2) Hyperhomocysteinemia

Screening and treatment for thrombophilia remain experimental in these women

THROMBOPHLEBITIS

Incidence	Vaginal delivery	1/9000
	C-section	1/800
	Ovarian vein thrombosis	Increased on right more than left

Treatment

Heparin. However, antibiotics alone were shown to be as good as antibiotics and heparin for treatment of septic thrombophlebitis (Brown CE, Stettler RW, Twickler D, *et al*. Puerperal septic pelvic thrombophlebitis: incidence and response to heparin therapy. *Am J Obstet Gynecol* 1999;181:143–8)

THROMBOSIS

Superficial thrombophlebitis most likely can be cleared up by changing IV site if placement	> 48 h
DVT begins during surgery. What % originate in the leg?	75%
Induration of calf	68%
Minimal edema	52%
Calf tenderness	25%
Positive Homan's sign	10%
Treat septic thrombophlebitis with heparin for how many days?	7–10
What % of patients with a thromboembolic phenomenon have tachypnea?	90%

Thrombosis factors	Factor VIII	25%
	Leiden V	20%
	Homocysteine	10%
	Protein 20280	6%
	Protein C deficiency	3%
	Protein S deficiency	1–3%
	Triglycerides under this level – considered normal	150 mg/dl
	Myocardial infarctions in what % are found with triglyceride levels of 150–200?	35%
	Septic pelvic thrombophlebitis are found in this ratio of vaginal deliveries	1/9000
	Found in this fraction of C-section deliveries	1/800
	Ovarian vein thrombosis is found more on right or left?	Right
	According to Brown and colleagues (Brown CE, Stetter RW, Twickler D, *et al*. Puerperal septic pelvic thrombophlebitis: incidence and response to heparin therapy. *Am J Obstet Gynecol* 1999;181:143–8), antibiotics alone are as good as antibiotics with heparin for the treatment of SPT	
Other risk factors	Major gyn surgery, age > 40 years, malignancy, previous venous thrombosis (DVT or PE), obesity, immobility, pregnancy and the post partum period, oral contraceptives, hormones, tamoxifen, varicose veins, prolonged surgical procedure, radical vulvectomy, pelvic exenteration, inguinal–femoral lympadenectomy, and/or as above – inherited or acquired thrombophila (Factor V Leiden, etc.)	
Virchow's triad	Stasis, hypercoagulability, vessel wall abnormality	
Diagnosis of DVT	Swelling of calf or thigh (unilateral) Pain or tenderness US (venous Doppler)	
Diagnosis of PE	Dyspnea, tachypnea, tachycardia, and shortness of breath. Pleuritic chest pain, hemoptysis, fever, panic, cyanosis, diaphoresis, friction rub or changes in heart sounds	
	ABG (PaO_2)	< 85 mmHg
	EKG – tachycardia, right axis shift	
	CXR – atelectasis? pleural effusion? increased diaphragm?	
	Lung scan	
	What percent of pulmonary emboli show no signs or symptoms of thrombosis in the lower extremities?	80%
Treatment	Immediate heparin for 5–10 days. Monitor with APTT then subcutaneous heparin every 24 h in two divided doses for remainder of pregnancy. APTT levels should be obtained 6 h after subcutaneous dose	
	Interventions for pulmonary embolism include:	
	STAT anticoagulant therapy, respiratory support, embolectomy, pulmonary artery catheterization, and vena cava interruption	
	Heparin and warfarin Rx should overlap × 4 days. (Warfarin can be started postpartum and thromboembolic episodes should be treated for at least 3 months	

Heparin dosing guidelines

(1) Obtain patient's weight in kg = _____

(2) Calculate bolus dose 80 units/kg = _____units IV

(3) Standard heparin infusion is 10 000 units of heparin in 250 ml D5W
IV heparin maintenance dose 15–25 U/kg/h = _____ units

(4) Warfarin _____ mg. Begin day 1–3 heparin therapy (if postpartum)

Weight	*Loading dose*	*Maintenance dose*
≤149 lb (≤70 kg)	5 000 units	1000 units/h (25 ml/h)
150–200 lb (71–90 kg)	7 500 units	1400 units/h (35 ml/h)
≥ 201 lb (≥91 kg)	10 000 units	1800 units/h (45 ml/h)

Dose adjustments

APTT	*Rate change (ml/h) (ml/h)*	*Dose change*
< 36 s	+5	+200 U, 5000 U bolus
36–44 s	+3	+120 U, no bolus
45–73 s	0	none
74–90 s	–3	–120 U, stop heparin × 1 h
> 90 s	–3	–120 U, stop heparin × 1 h

Prophylactic heparin dosages
First and second trimester 5000–7500 units SC b.i.d.
Third trimester 10 000 units SC b.i.d.
or
Monthly US Doppler studies of lower extremities

Labor
D/c heparin infusion 6 h prior to anticipated delivery. Subcutaneous heparin may be withheld at onset of labor
Protamine reversal for APTT >1–1½ × control

Epidural contraindicated

Heparin infusion can be restarted when hemostasis is achieved (usually 2 h after delivery)

Prophylactic regimens for venous thromboembolism

Low-dose unfractionated heparin

Medical illness: 5000 U subcutaneously every 12 h

General surgery: 5000 U subcutaneously every 8–12 h, starting 1–2 hours preoperatively

Low-molecular-weight heparin/heparinoids
Moderate risk:

Enoxaparin, 20 mg subcutaneously 1–2 h preoperatively and once a day postoperatively

Dalteparin, 2500 U subcutaneously 1–2 h preoperatively and once a day postoperatively

High risk:

Enoxaparin, 40 mg subcutaneously > 2 h preoperatively and once a day postoperatively

Dalteparin, 5000 U subcutaneously > 2 h preoperatively and once a day postoperatively

Pneumatic compression

Postop VTE declines 3-fold with eternal pneumatic compression during surgery and for 5 days post op

- LMWH and external pneumatic compression are considered the best choices for prophylaxis in high risk patients

Complications

Hemorrhage 5–10%
Thrombocytopenia 3% (monitor platelets first 3 weeks Rx – d/c if platelets <100 000)
Osteoporosis – (supplemental vit D rec for long-term Rx)
Increased liver enzymes

Reverse

Protamine sulfate 1 mg/100 units of heparin
(do not exceed 100 mg)

THYROID DISEASE

Irregular cycles, hot flushes, fatigue, constipation, dry skin, hair loss, weight gain, increased cholesterol; do TSH

What % females have subclinical thyroid dysfunction?	8%
What % will develop overt disease?	2–5% per year
What % will develop disease who have subclinical thyroid dysfunction and antithyroid antibodies?	5–7%

Hyperthyroidism

What % of pregnancies are complicated by hyperthyroidism and thyroid storm?	0.2%

Suspect hyperthyroidism in pregnancy if the patient has:

(A) Tremor or nervousness
(B) Frequent stools
(C) Excessive sweating

Mild hyperthyroidism mimics symptoms of normal pregnancy, and can present as fatigue, increased appetite, vomiting, palpitations, tachycardia, heat intolerance, increased urinary frequency, insomnia, and emotional lability

Hyperthyroidism is less common, occurring in less than	1%
Most common cause in the USA is	**Graves' disease**

Other causes

Solitary toxic adenoma, subacute granulomatous thyroiditis (deQuervain's), and, if pregnant → hyperemesis gravidarum, trophoblastic disease, exogenous thyroid hormone

Affects of untreated hyperthyroidism

Fetal → spontaneous abortion, prematurity, low birthweight, and/or fetal/neonatal thyrotoxicosis

Maternal → preeclampsia, maternal heart failure, infection, anemia, and/or thyroid storm

Common precipitants of thyroid storm

Acute surgical emergency, induction of anesthesia, diabetic ketoacidosis, pulmonary embolism, noncompliance with antithyroid medications, myocardial infarction, infection, hypertension or preeclampsia, L & D, and severe anemia

Symptoms

Tachycardia, atrial fib, tremor, muscle weakness, increased reflexes, myalgia, low-grade fever, sore throat and dysphagia

Diagnosis

Decreased TSH, elevation of free T_4. (If T_4 is normal, measure T_3, as this is 5% cases)

Treatment

Radioiodine therapy, antithyroid medication (propylthiouracil or methimazole) and (rarely) thyroidectomy. β-Blockers are also helpful in treating symptoms, but use propanolol with caution because it has a tendency to increase pulmonary diastolic pressure, and cardiac failure is a frequent presentation of thyroid storm. Propylthiouracil and methimazole alone can reduce the T_3 concentration by 75%

Glucocorticoids should be started as soon as thyroid storm is diagnosed.

Therapy is basically designed to:

(A) Reduce the synthesis and release of thyroid hormone

(B) Remove thyroid hormone from the circulation and increase the concentration of TBG

(C) Block the peripheral conversion of T_4 to T_3

(D) Block the peripheral actions of thyroid hormone

(E) Treat the complications of thyroid storm and provide support

(F) Identify and treat potential precipitating conditions

Supportive care for patient in thyroid storm

(A) IV fluids and electrolytes

(B) Cardiac monitoring

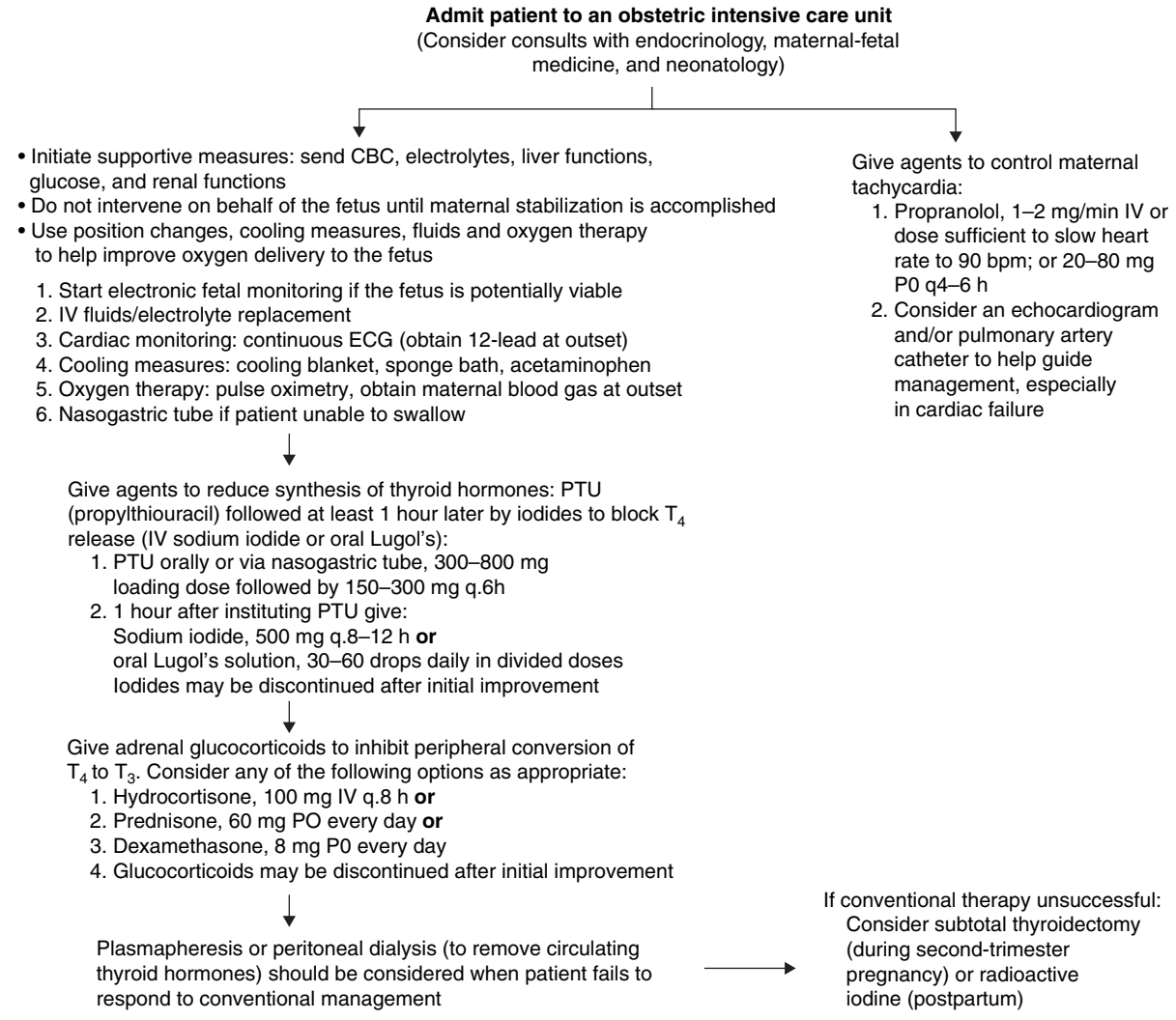
(C) Consideration of pulmonary artery catheterization (central hemodynamic monitoring to guide beta-blocker therapy during hyperdynamic cardiac failure)

(D) Cooling measures: blanket sponge bath, acetaminophen, avoid salicylates (risk of increased T_4)

(E) Oxygen therapy (consider arterial line to follow serial blood gases)

(F) Nasogastric tube if patient is unable to swallow (may be only avenue for propylthiouracil administration)

Algorithm for management of tyroid serum



Hypothyroidism

	Very common in women. Most frequent causes in the USA are Hashimoto's thyroiditis and chronic lymphocytic thyroiditis
	Antithyroglobulin antibodies reported in @ 60%
	Antithyroid peroxidase antibodies in @ 90%
Symptoms	Fatigue, cold intolerance, constipation, weight gain, muscle cramps, hair loss, depression, cognitive dysfunction, menstrual irregularities and infertility
Diagnosis	Elevated TSH, decreased T_4
Treatment	Levothyroxine – average replacement dose in adults is 1.6 µg/kg of body weight (usually 75–100 µg daily). Dose in elderly patients should be less – typically 12.5–25 µg daily
	Minimum of 6–8 weeks is necessary between changes in dosage. Improvement in symptoms can usually be seen within 2–3 weeks
	Hypothalamus → TRH then pituitary → TSH – pregnancy
	hCG increases thyroid volume by 10–20%
	Also increases thyroxine or T_4
	Placenta increases E_2 – increased TBG 2–3 ×
	Recommendations (American Thyroid Association) – age 35 and q. 5 years thereafter TSH
	If suspect central (secondary) hypothyroidism, draw FT_4
Miscarriage	Draw TSH and anti-thyroid peroxidase antibodies (TPO)
Primary hypothyroidism	Increased TSH, decreased free T_4
Central hypothyroidism	Decreased or normal TSH, decreased free T_4

Primary hyperthyroidism — Decreased TSH, increased FT_4, T_3

Central hyperthyroidism — Increased or normal TSH, increased free T_4, increased T_3

Pregnancy

Check TSH and FT_4 level every 4–6 weeks. Levothyroxine dose usually needs to be increased by 50%

Hyperemesis gravidarum is associated with higher free T_4, total T_3 and lower TSH levels

hCG levels > 10 000 IU/l may be associated with biochemical hyperthyroidism

> 30 000 IU/l may be associated with clinical hyperthyroidism

(Therefore, sudden development of hyperthyroidism in first trimester should raise the question of a molar pregnancy)

TIMED TESTS

FSH evaluation	Day 3
Hysterosalpingogram	Day 8
Postcoital test	Day 14
Serum progesterone level for ovulation evaluation	Day 21
Endometrial biopsy for infertility evaluation	Day 26

TINNITUS IN PREGNANCY

Tinnitis is the perception of sound (ringing, whooshing, buzzing, or pulsing) in the ears or head when no eternal source is present. This condition has a reported lifetime prevalence of 33%

BEWARE of tinnitus in pregnancy

	Tinnitus	*Hearing loss*	*Vertigo*	*Hypertension*	*Cranial nerve VII involvement*
Acoustic neuroma	+	+			+
Otosclerosis	+	+			
Menière's disease	+	+	+		
Preeclampsia	+			+	
Benign tinnitus of pregnancy	+				

Pearls

Tinnitus is twice as common in pregnancy and occurs in what % of pregnant women? 25%

Indications for otolaryngology referral include hearing loss, vertigo, and facial weakness

Always evaluate for signs/symptoms of preeclampsia in gravid women with tinnitus

TORCH

Toxoplasmosis, other viruses, rubella, cytomegalovirus and herpes simplex virus

Toxoplasmosis is seen in what % of females exposed by undercooked meat or cat feces? 1/3

Toxoplasmosis

Avoid contaminated meat, unwashed fruit/vegetables, unpasteurized cheese, cat feces

What % live births are affected? 0.1–0.6%

US demonstrates dilated ventricles, pericardial effusions, echogenic bowel, calcifications in brain

Rate of transmission for first trimester is 25%
second trimester is 54%
third trimester is 75%

Spiramycin, pyrimathamine, sulfadiazine will not decrease transmission but can decrease sequelae

Rubella	Communicable in	< 7 days
	Rash	> 4 days
	If exposed at less than 11 weeks' gestation, risk to infant is	90%
	11–12 weeks	33%
	13–14 weeks	11%
	> 16 weeks	0%
Cytomegalovirus	What % mothers already infected?	80%
	Microcalcifications, microcephaly, MR, etc.	
	MR	55%
	IUGR and microcephaly	40%
	Decreased platelets	70%
	Most common congenital infection in the USA	
	What % of newborns each year are infected?	1%
	Maternal infection usually asymptomatic	
	What % higher SES vs lower SES are susceptible?	45% vs 15%
	How many seroconvert during pregnancies?	1–4%
	What % of these transmit CMV to fetus?	40–50%
	Most common cause of deafness in the USA	
	Rates of transmission throughout pregnancy	40%
	Amniocentesis provides the best technique for prenatal diagnosis	
Herpes	What % of pregnancies have + titers?	0.2–7.4%
	What % of pregnancies shed virus at time of delivery?	0.1–0.4%
	HSV-I to HSV-II found in genital area	15–20% to 80–85%
	Greatest risk to fetus is during primary infection at SVD	40%
	During recurrent infection, infants only are infected	≤ 1%
Varicella-Zoster (Chickenpox)	Greatest risk on congenital varicella is between	13–20 weeks
	No clinical risks after	20 weeks
	VZIG needs to be given within how many hours?	96
	The dosage of VZIG is	125 units per 10 kg IM
	Immunoglobulin and Varivax® not recommended during pregnancy	
	The dose of Varivax between ages 12 months and 12 years is	0.5 ml (1 dose)

TORSION

	Most common size of benign mass that undergoes torsion is	8–12 cm
	Torsion of a malignant tumor is rare	
	The ratio of torsion on the right as compared to torsion on the left is	3 to 1
Symptoms	Acute pain and palpable tenderness	90%
	Nausea and vomiting	66%
	Low fever and increased WBCs (due to hypoxia and necrosis)	
Treatment	Laparoscopy	75%
	Untwist but if vascular compromise do salpingoophorectomy	

The pendulum of therapy actually swings back and forth for ovarian torsion→ on one hand, conservative laparoscopy is recommended, but because of the fear of thrombus formation in the ovarian vein, thromboembolism could occur, but since this is a minimal risk, untwisting the adnexa became the standard management. Recently, adnexa-sparing laparoscopic procedures for ovarian torsion have been shown to predispose to recurrence of torsion (Pansky M, Smorgick N, Herman A, Schneider D, Halperin R. Torsion of normal adnexa in postmenarchal women and risk of recurrence. *Obstet Gynecol* 2007; 109:e355–9), but these data are sparse and therefore conservative therapy is still recommended at this time to spare a healthy and normal appearing ovary

TRANSVERSE LIE

Back up	Do low transverse (or vertical if concerned about baby turning)	
Back down	Do vertical incision	
	Incidence	1/360

Etiology	Multiparity, pre-term fetus, previa, abnormal uterus, increased amniotic fluid, contracted pelvis Women with four or more deliveries have 10 × incidence
Diagnosis	Inspection and palpation of abdomen
Course of labor	Retraction ring develops – situation becomes neglected transverse lie If fetus small (< 800 g) and pelvis large, SVD is possible. Fetus delivers doubled up upon itself as conduplicato corpore. Uterus can and does usually RUPTURE
Management	External version worthwhile ONLY prior to or with early labor if no other contraindications are present. (Hold head in pelvis for several contractions) Vertical incision C-section is likely necessary since neither the head nor the feet are in the pelvis for extraction. (*See* Back up/Back down)

TRAUMA

Immediate care	MAINTAIN AIRWAY, deflect uterus to left, circulating volume. Exam, control hemorrhage, identify + stabilize serious injuries. Pelvic – check for bleeding or ROM
Labs	CBC, Kleihauer–Betke (if Rh –), amylase, biochemistries, type and cross match, fibrinogen, platelets, FDP, PT and PTT
Fetal evaluation	Monitor if ≥ 20–24 weeks Continue to monitor if: tachycardia, late decels, non-reactive NST, > 4 contractions in 1 h, ROM, bleeding or if there is any serious maternal injury. If these are not present then discharge with follow-up plans

Anatomic and physiologic changes relevant to trauma management during pregnancy

Anatomic/physiologic change	*Relevance to trauma management*	*Implication/action*
Increased maternal blood volume	Increases by up to 50% in the third trimester	Blood loss may be underestimated
Increased RBC mass	RBC mass increases to a lesser degree than total plasma volume, resulting in decreased hematocrit	Hematocrit as low as 30–32% may be physiologic
Decreased blood pressure	Blood pressure decreases by 10–15% mmHg, particularly in the midtrimester	Must be taken into consideration when evaluating for hypovolemia/hemorrhagic shock
Increased pulse rate	Pulse rate increases by 5–10 beats/min during pregnancy	Same as above
Decreased gastrointestinal motility	Gastric emptying time is prolonged, increasing risk for aspiration	Consider use of nasogastric tubes when aspiration is a risk
Cephalad displacement of intra-abdominal contents	Small bowel is compressed within the upper abdomen in latter pregnancy	Penetrating trauma to the upper abdomen is likely to cause complex intestinal injuries
Respiratory rate increases	pCO_2 is normally 32 mmHg; pCO_2 in the "normal" range (40–42 mmHg) may indicate impending respiratory failure	
Bladder is displaced superiorly into the abdomen after 12 weeks' gestation	Bladder is subject to blunt or penetrating injury with lower abdominal trauma	Suspect bladder injury in traumatic events to the lower abdomen

Estimation of blood loss based on clinical variables

	Class I	*Class II*	*Class III*	*Class IV*
Blood loss (ml)	Up to 750	750–1500	1500–2000	≥ 2000
Blood loss (% BV)	Up to 15%	15–30%	30–40%	≥ 40%
Pulse rate	< 100	> 100	> 120	≥ 140
Blood pressure	Normal	Normal	Decreased	Decreased
Pulse pressure (mmHg)	Normal or increased	Decreased	Decreased	Decreased
Capillary blanch test	Normal	Positive	Positive	Positive
Respiratory rate (min)	14–20	20–30	30–40	> 35
Urine output (ml/h)	≤30	20–29	5–15	Negligible
CNS/mental status	Slightly anxious	Mildly anxious	Anxious and confused	Confused/lethargic
Fluid replacement (3 : 1 rule)	Crystalloid	Crystalloid	Crystalloid + blood	Crystalloid + blood

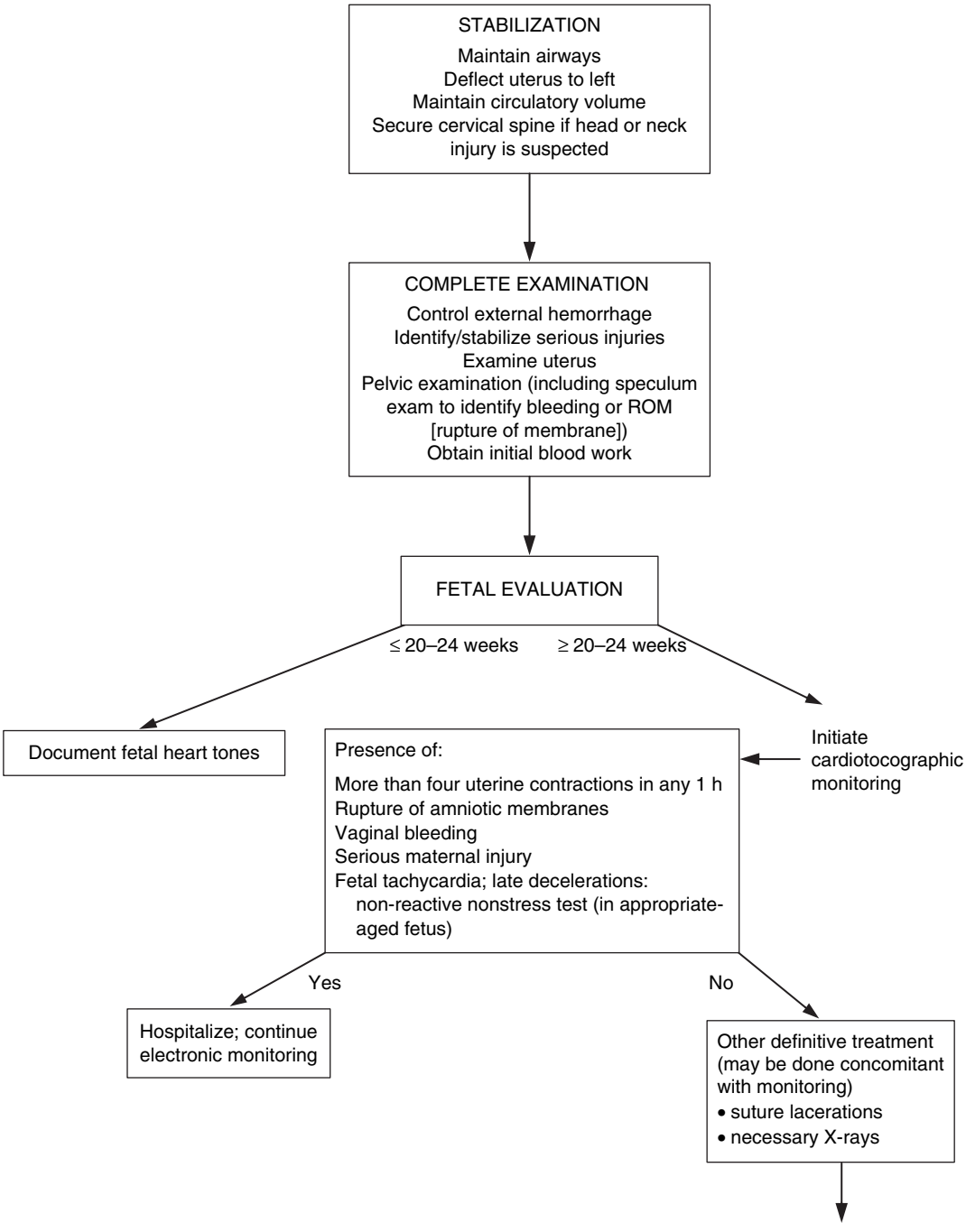
Interpretation of diagnostic peritoneal lavage (positive)

Free aspiration of blood (> 10 ml)	
Grossly bloody lavage fluid	
RBC count	> 100 000/mm^3
WBC count	> 500/mm^3
Amylase	> 175

Indications for diagnostic peritoneal lavage during pregnancy

Abdominal signs or symptoms suggesting intraperitoneal hemorrhage
Unexplained shock
Altered mental status
Major thoracic injuries
Multiple major orthopedic injuries (including pelvic fracture)

Trauma in pregnancy



TRIPLET PREGNANCY

Incidence	Rate recorded in 1998	1/570
Increased rate of	Intensive prenatal care, antenatal hospitalization, longer perinatal hospitalization	
Greater risk for	Anemia, PIH and gestational diabetes, PPROM and especially PTL	
	Anemia in triplet pregnancy occurs approximately what %?	35%
	What proportion of newborn triplets experience RDS?	40%
	Stillbirth rate is how many times higher than in singletons?	3 ×

TROPHOBLASTIC DISEASE

	Incidence	1/1000
	Incidence in Orient	2/1000
Diagnosis	hCG is usually over	100 000
	Ultrasound – echoes in placental mass – "snowstorm" appearance	
Signs and symptoms	Vaginal bleeding usually 6–16 weeks' gestation	80–90%
	Large for dates	50%
	Small for dates	25–28%
	Theca lutein cysts	15%
	Hyperemesis	8%
	PIH in first or second trimester	1%

Hydatidiform mole

Complete	46XX (haploid fertilization by sperm completely replaces maternal contribution) in	90%
	46XY (dispermy of empty egg) in	10%
	No gestational sac or fetus. No fetal vessels. Hydropic swelling.Prominent hyperplasia	
	Invasive mole or choriocarcinoma follows in	15–20%
Partial	69XXX (karyotype is triploid due to extra haploid from father) XXY, XYY	
	Gestational sac or fetus is usually present at some point. Vessels are present. Focal swelling only. Focal hyperplasia	
	Risk of subsequent molar	5–10%
	Invasive mole follows in	4–11%

Gestational trophoblastic tumor

Invasive mole	Mole that penetrates and may perforate the uterine wall	
	Locally destructive and may invade parametrial tissue or blood vessel	
	Hydropic villi may embolize to distant sites as lungs and brain but do not grow in these organs as true metastasis	
	It is associated with persistent elevated hCG	
	Responds well to chemotherapy	
Choriocarcinoma	No ultrasound features	
	Absent vessels	
	Absent swelling	
	Poorly differentiated	
	Risk of subsequent molar	10–30%
	Arise in hydatidiform moles	50%
	Arise in previous abortion	25%
	Arise from normal pregnancies	25%
	Arise out of what number of pregnancies in the USA	1/25 000
Placenta site tumors	Trophoblastic tissue deeply invading the myometrium	
	Low level of hCG. Locally invasive, many are self-limited and cured by curettage	
	What % result in disseminated metastasis and death?	10%
	Are not sensitive to chemotherapy. Hysterectomy is treatment of choice	

Treatment

Suction D&C with Pitocin intraop and postop
Follow-up how often until two normal hCGs? q. 10 days
After three normal hCGs, follow how often for a year? every 3 months

Average time for β-hCG to reach nl levels after evacuation 73 days
Evacuation is curative in > what % of patients? 80%

Chemotherapy

Methotrexate and actinomycin D; for resistance add cyclophosphamide MAC

Poor prognosis

Brain or liver mets, β-hCG > 40 000, symptoms > 4 months, failed chemo. Give multiple chemotherapy

LIVER METS carries WORSE PROGNOSIS than BRAIN mets

Patients at highest risk

(1) Pre-evac uterine size > expected dates or > than what gestation? 20
(2) Bilateral theca lutein cysts > 6 cm
(3) Age > how many years? 40
(4) Elevated hCG levels over what level? 100 000
(5) Medical complications of molar
(6) Repeat hydatidiform mole

TUBAL LIGATION SYNDROME

Does not exist! It is a nonentity! Tubal ligation has no effect on hormonal parameters or menstrual characteristics when compared with women not having undergone tubal sterilization. (Harlow BL, Missmer SA, Cramer DW, *et al*. Does tubal sterilization influence the subsequent risk of menorrhagia or dysmenorrhea? *Fertil Steril* 2002;77:754–60)

TUBERCULOSIS SALPINGITIS

Granuloma, giant cells, CALCIFIED lymph nodes
PIPESTEM proximal to obstruction, multiple STRICTURES along tube, irregularities of ampulla
DEFORMITY of endometrial cavity. + Acid fast bacillus with endo bx and culture

Treatment

PZA and rifampin. Sterile – refer for IVF

Tuberculosis in pregnancy

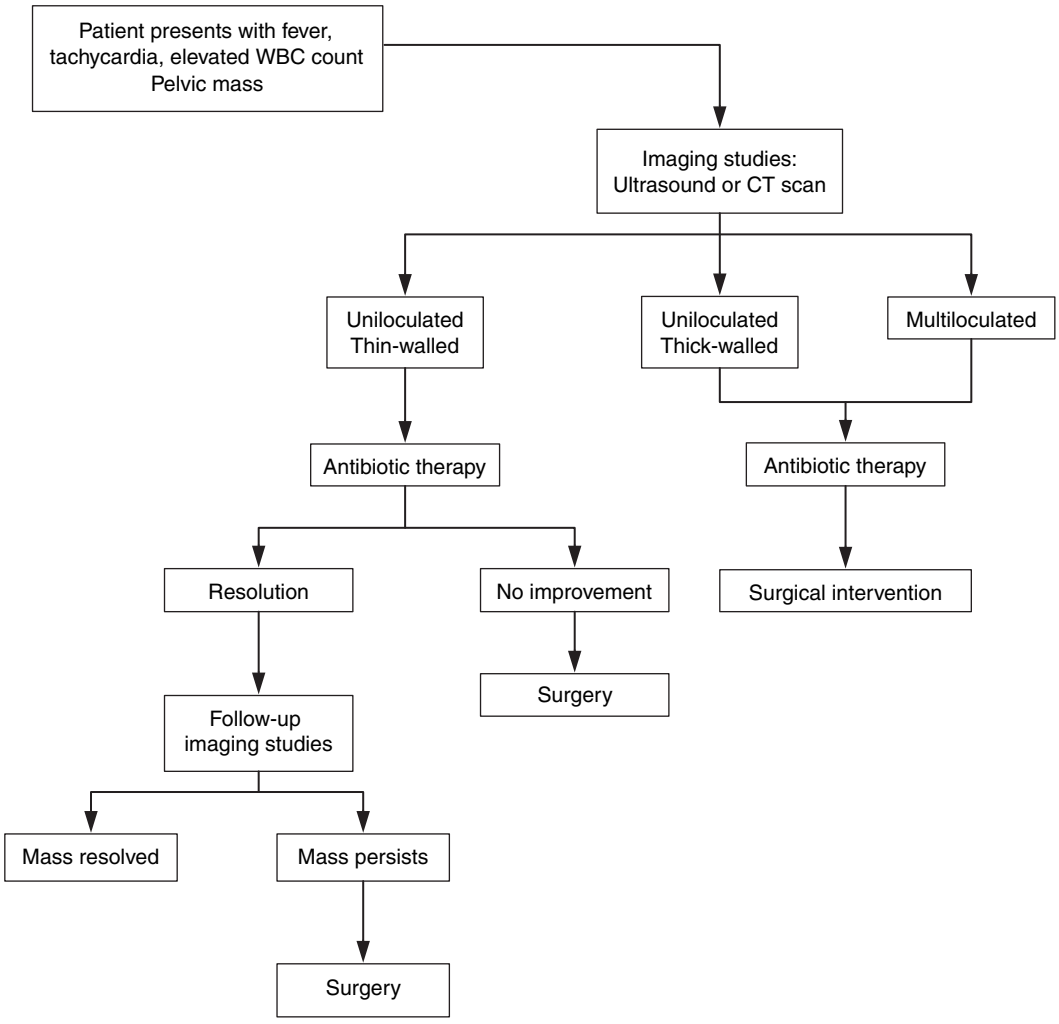
Treat essentially the same but shield during CXR
Treat active TB with isoniazid with pyridoxine and rifampin
Non-pregnant: < 35 years with + PPD, give isoniazid
Pregnant: < 35 years with + PPD, start treatment after delivery
Exception: < 35 years who likely recently was infected with TB – start prophylaxis (isoniazid) after the first trimester

TUBO-OVARIAN ABSCESS

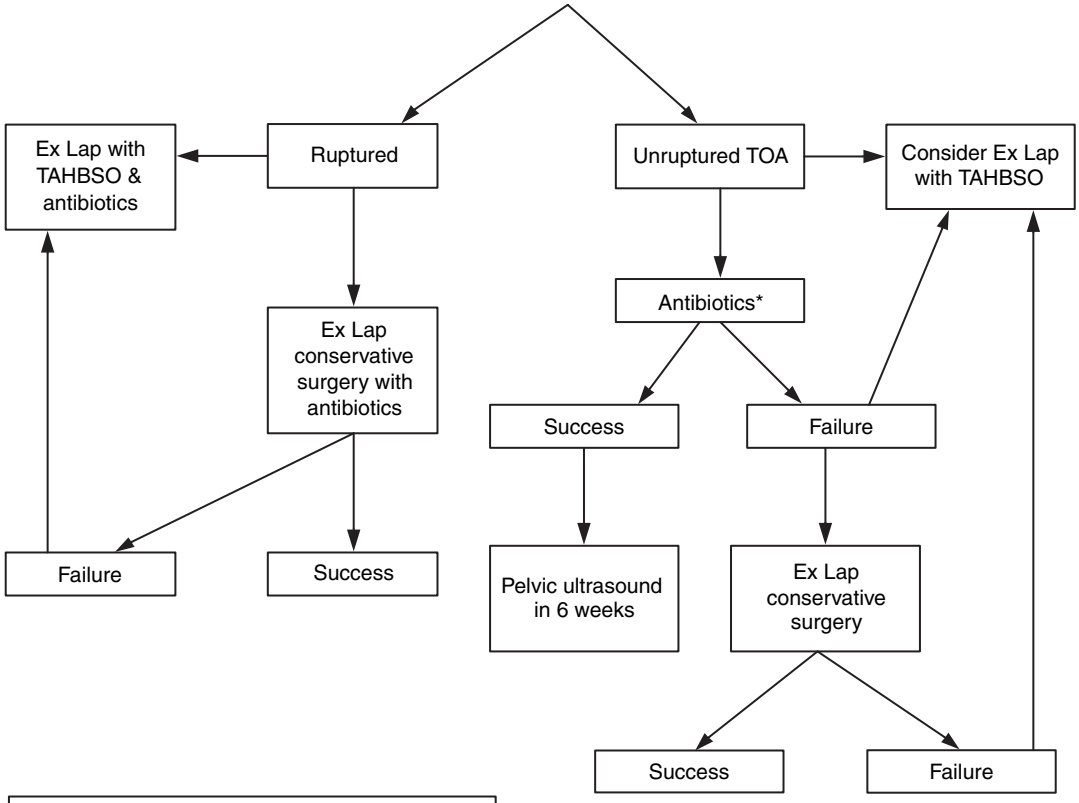
What % of patients hospitalized for PID have TOA? 34%
Initial offending pathogen in TOA is usually either *Neisseria gonorrhoeae* or *Chlamydia trachomatis*
Typical pathogens isolated from TOAs include *E. coli*, *B. fragilis*, peptostreptococci
Risk factors for TOA include use of an IUD, multiple sexual partners, low socioeconomic status, adolescents
Classic features of TOA include fever, pelvic or abdominal pain and a pelvic mass
Other common signs are nausea and vaginal discharge or bleeding
Diagnosis of TOA include ultrasound, CT or MRI but the "Gold Standard" is laparoscopy
Treatment with antibiotic regimens include:
(1) Cefoxitin 2 g IV q. 6 h or cefotetan 2 g IV q. 12 h with doxycycline 100 mg IV or p.o. q. 12 h
(2) Clindamycin 900 mg IV q. 8 h plus gentamicin: loading dose 2 mg/kg; maintenance dose 1.5 mg/kg

IV antibiotics not considered a “treatment failure” until after 72 h
Surgical treatment of choice for TOA is TAHBSO
Conservative surgical treatments for preservation of fertility include:
Unilateral salpingo-oophorectomy
Laparoscopy with endoscopic drainage of the abscess
Posterior colpotomy with transvaginal drainage of the abscess
Proportion of TOAs occurring in postmenopausal women is 2%

Management of the patient with a pelvic abscess



Tubo-ovarian abscess



CDC Inpatient Treatment Regimens

Regimen A: Cefoxitin, 2 g IV q. 6 h or cefotetan, 2 g IV q. 12 h, plus doxycycline, 100 mg IV p.o. q. 12 h

Regimen B: Clindamycin, 900 mg IV q. 8 h plus gentamicin, loading dose 2 mg/kg followed by maintenance dose of 1.5 mg/kg q. 8 h

Ex Lap, exploratory laparotomy

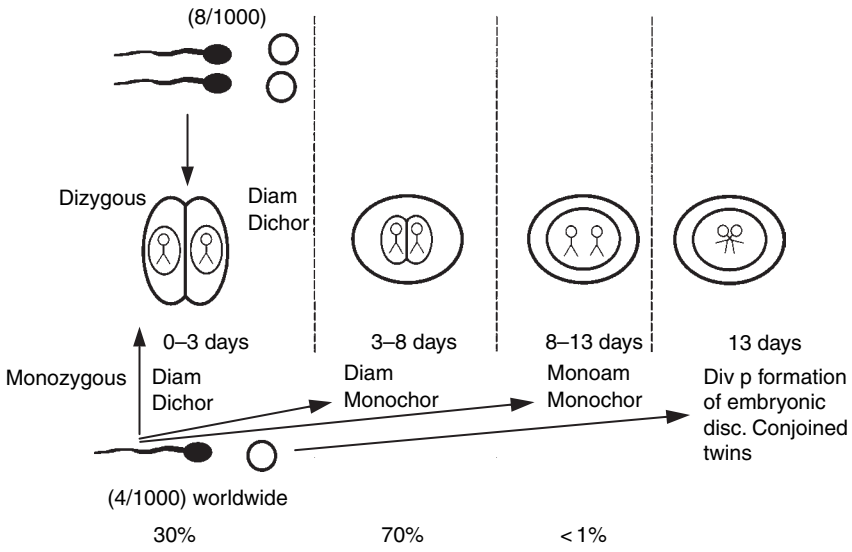
TWINS

Incidence	Dizygous	8/1000
	Monozygous	4/1000
	Increase in incidence during last decade include accepted reasons of:	
	(1) Advanced maternal age at conception	
	(2) Reduced fecundity at advanced maternal age	
	Fertilization occurs with dizygous (diamniotic/dichorionic) and monozygous (diamniotic/dichorionic)	0–3 days
		30%
	Monozygous (diamniotic/monochorionic)	3–8 days
		70%
	Monozygous (monoamniotic/monochorionic)	8–13 days
		< 1–3%
	Mortality in monoamniotic twins is	40–75%
	Deliver by C-section by	34 weeks
	Monochorionic twins with increased incidence of PTL due to hydramnios. Arteriovenous malformations – watch for TTTS	
	Division after formation of embryonic disc > day 13 results in conjoined twins	1/50 000
	Interlocking twins occur	1/1000
	Total incidence of any twins in a pregnancy is	1–1.5%
	Velamentous insertions are how much more likely in twins?	10 ×
	Fetal anomalies in singletons are 2–3% compared to actual incidence in twins being	8%
	PIH increased (probably due to enlarged placental mass)	
	In twins	40%
	In triplets	> 50%
Weight gain for twin gestation	(1) 24 lb by 24 weeks' gestation	
	(2) 40–70 lb total okay	
Visits for twin gestation	(1) Biweekly until 20 weeks and then weekly after 32 weeks	
	(2) House calls p.r.n.	
	(3) Cervical checks (?) p.r.n. US or digital	
	(4) US at 18 weeks	
	(5) US + NST from 28–30 weeks' gestation	
	(6) BPP if NSTs are non-reactive and equivocal or US demonstrates discordant growth	
Decrease of physical activity after 28 weeks in twin gestation	(1) If IUGR of one or both twins is suspected, patient to be placed on strict bed-rest at home or in hospital to monitor each twin's growth closely by serial ultrasound measurements of BPB, head circumference and femur length	
	(2) Examine for twin–twin syndrome, etc.	
Mean age at delivery	Singletons	40 weeks
	Twins	37 weeks
	Triplets	33 weeks
	Quads	30 weeks
Management	Twins – deliver vaginally if possible	
	Triplets – now may deliver vaginally if ALL can be monitored	70%
	Quads or > – C - section	
	Ultrasounds for growth every	4 weeks
	Weekly NSTs after	32 weeks

Intrapartum management for twins to include:

(1) Fetal monitoring throughout labor
(2) US needs to be in labor and delivery room in the event that version is required for a second twin
(3) Epidural anesthesia is preferred to facilitate manipulation of the second twin in addition to relieving pain
(4) OR staff would be required to be on stat back-up secondary to the anticipated vaginal delivery of second twin which may need to be reversed at a moment's notice
(5) C-section might be required if advanced labor with premature twin gestations is noted between 26–34 weeks' gestation
(6) C-section would be strongly considered if the first twin's presentation is shoulder, transverse or breech
(7) If the second twin is breech and the first twin is vertex, the first twin could be delivered if there are no other complications followed by external version, internal version, partial breech extraction with piper forceps of the second twin under general anesthesia using halothane for uterine relaxation p.r.n. (C-section may be required for the delivery of the second twin in these cases)

Twin types



TWIN–TWIN TRANSFUSION SYNDROME

Most often occurs with diamniotic/monochorionic twins	1%
Incidence in monochorionic twins	5–10%
Mortality with TTTS increased with earlier diagnosis. Diagnosis with serial ultrasounds. Abdominal circumference most reliable	
Increased neonatal deaths, congenital anomalies, IUGR if	> 30%
Look for same sex, single placental mass, dividing membrane, lack of twin peak sign. There is a "T" sign	
Abnormal AF volume	
Oligo	≤ 2 cm
Poly	≥ 8 cm
EFW discordance	≥ 20%
Hydrops with skin edema, effusion, ascites	≥ 5 mm
Urinary bladder – small with donor, large with recipient	

Treatment

Amnioreduction, laser, septostomy, percutaneous cord ligation

(1) Membrane septostomy results in a prolongation of pregnancy from time of initial diagnosis to delivery compared to amnioreduction according to Johnson and collleagues (Johnson JR, Rossi KQ, O'Shaughnessy RW. Amnioreduction versus septostomy in twin–twin transfusion syndrome. *Am J Obstet Gynecol* 2001;185:1044–7)

(2) Bipolar diathermy may be used in severe twin–twin transfusion in order for one twin to survive according to Taylor and colleagues (Taylor MJ, Shalev E, Tanawattanacharoen S, *et al*. Ultrasound-guided umbilical cord occlusion using bipolar diathermy for stage III/IV twin–twin transfusion syndrome. *Prenat Diagn* 2002; 22:70–6)

TWO-VESSEL CORD

Etiology

(1) Fails to form (aplasia or agenesis)
(2) Involutes after forming (atrophy)

20% SUA have associated abnormalities

Associated with variety of fetal abnormalities

Trisomy 18 and trisomy 13 — increased rate 10% (0.5–1% normally)

When one artery is missing the *most common* anomaly is one kidney missing

(1) Increased rate IUGR — x 2
(2) Increased rate PTL and preterm birth — x 2
(3) Increased rate of *spontaneous abortion*
(4) *Frequently* observed in *multiple pregnancies*
(5) *Seen more frequently* in conjunction with fetal malformations

CNS anomalies
Cardiac defects
GI defects
- Esophageal atresia — x 5
- Tracheoesophageal fistula — x 5
- Anorectal atresia — x 5

Multicystic dysplastic kidneys
Limb reduction defects

Velamentous cord insertions — increased 2.7 (1% gen OB pts)

Sonographic diagnosis

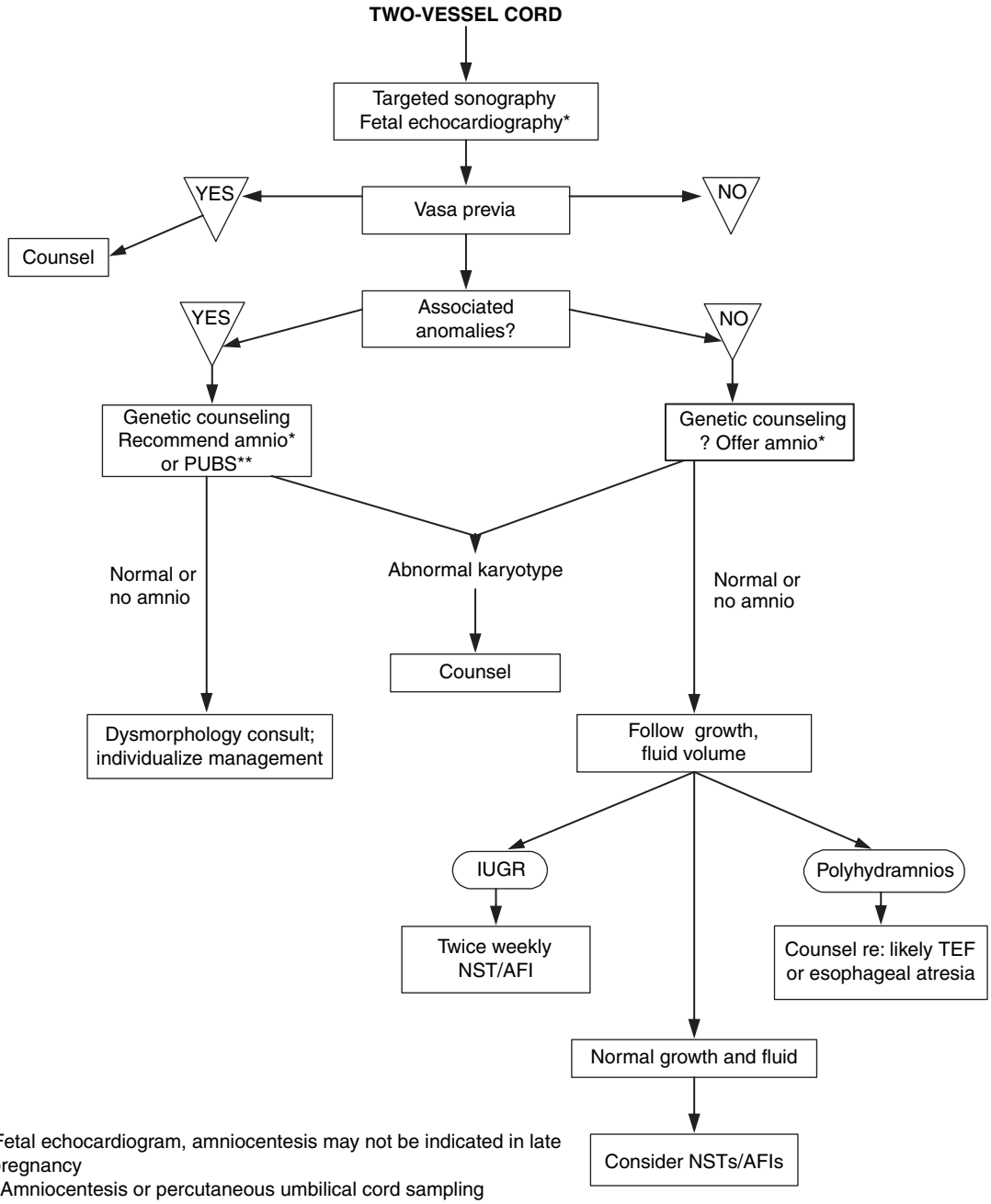
Many obstetricians have never noticed an umbilical cord with a single umbilical artery *in utero*

(1) Almost impossible to detect a single umbilical artery without a 5-MHz transducer and challenging even with a 5-MHz scan head
(2) Not all imaging studies include a careful look at the umbilical cord vasculature
(3) May become atretic after the original study is done

Once the fetus is determined to have just one umbilical artery:

(1) Assess growth and fluid volume
(2) See if determination can be made of where the cord enters the placenta
(3) Offer amniocentesis
(4) Provide PTL information and cautions
(5) NSTs > 32 weeks (2 degrees to increased rate stillbirth)

Evaluation and management of pregnancy with a two-vessel cord



* Fetal echocardiogram, amniocentesis may not be indicated in late pregnancy

** Amniocentesis or percutaneous umbilical cord sampling recommendation. Depends on specific defects and gestational age

TZANCK TEST

	What is the % of correlation to positive viral cultures? This is the most COST-EFFECTIVE way to diagnose HSV 2 but not the Gold Standard (cultures)	94.1%
How to do the Tzanck test	(1) Lesion is scraped at base with scalpel blade (2) Scalpel is touched to slide and allowed to dry per air (3) Apply 0.1% aqueous solution of toluidine blue for 15 s (4) Wash with tap water and dry (5) Apply permanent cover slip (6) Look for multinucleated giant cells	

ULTRASOUND

	Specificity on identifying fetus without anomalies? Sensitivity on identifying fetus with anomalies cannot be estimated TI (Thermal index – is estimation of temperature rise due to US) should be MI (Mechanical index – is measurement of compressive and decompressive effects of US pulses)	> 99% < 1 < 1
Acoustic output regulations	FDA limits for fetal application Manufacturers require the limit to machines to be	94 mW/cm^2 720 mW/cm^2
ALARA	As low as reasonably achievable	
Anomalies and abnormal karyotype	Any cardiac abnormality has what rate of aneuploidy? Endocardial cushion defect associated with Coarctation of aorta associated with *Conotruncal lesions:* Interrupted aortic arch Double-outlet right ventricle Tetralogy of Fallot – deletions of chromosome 22 Thickened nuchal fold – modest increased risk of aneuploidy Choroid plexus cysts – normal karyotype most of the time	2–12% trisomy 21 45X 4–14%
Nuchal translucency	Approximately this % have anomalies of heart and great vessels Prevalence of major cardiac abnormalities increase with increase in NT size First-trimester MS screening combined with NT on US increased detection rate for Down's Detailed echocardiography should be done in all fetuses with increased nuchal translucency because of the increased incidence of major cardiac abnormalities	50–90%

Ultrasound and its association with β-hCG

Ultrasound	*Days from LMP*	*IRP*	*Second International Standard*
Sac	34 (4½ weeks)	1 398	914
Fetal pole	40 (6 weeks)	5 113	3 783
Fetal cardiac motion	46 (7 weeks)	17 208	13 178

UMBILICAL ARTERY DOPPLER VELOCIMETRY

Umbilical artery S/D ratio is abnl if diastolic flow is either absent or reversed after what week gestation? 18–20 weeks

The absent or reversed flow may suggest serious fetal compromise. In some cases there is a deterioration in the Doppler flow studies prior to deterioration in the biophysical profile in the IUGR fetus

- An appropriate transverse sonographic imaging of the umbilical cord is accurate in detecting 2-vessel umbilical cords. Ability to visualize the number of vessels in the cord varies with gestation:

15 weeks	74%
17 weeks to @36 weeks	98%
36 weeks to 40 weeks	83%

Ultrasound screening criteria

- Controversial
  May or may not be standard of care in some communities. No proven cost-effectiveness

UMBILICAL CORD BLOOD ACID–BASE ASSESSMENT

Cord blood sample in a syringe flushed with heparin is stable x 60 min

Fetal/newborn acidemia (3 types)

(1) Respiratory pCO_2 high, HCO_3 normal
(2) Metabolic pCO_2 normal, HCO_3 low
(3) Mixed pCO_2 high, HCO_3 low

Umbilical artery pH and blood gas – adjunct to Apgar scores

Technique

(1) 10–20-cm cord segment clamped on either end
(2) Perform immediately after delivery
(3) Aspirate umbilical *artery*
(4) Sample may be obtained from chorionic surface of placenta (arteries cross *over* veins)
(5) 1–2-cc sample aspirated into heparinized syringe
(6) Residual air bubble expelled
(7) Cord segment sample stable for 1 h at room temp

"Normal" umbilical artery pH = 7.27 (mean)
± 2 standard deviations = 7.15–7.39

Pathologic fetal acidemia
Traditional threshold < 7.20
Realistic threshold (i.e. pH associated with adverse neonatal sequelae, neurologic dysfunction/death) < 7.0
Birth asphyxia/hypoxia = low Apgars
(0–3 at 5 min) + pH < 7

Protocol

(1) Doubly clamp cord segment (10–20 cm) immediately after birth in all deliveries and place on table
(2) pH and acid–base determinations indicated for:
  – prematurity
  – meconium (requiring tracheal visualization, suctioning and/or intubation)
  – nuchal cord
  – low Apgar scores (< 7 at 5 min)
  – abnormal antepartum fetal heart tracing
  – any serious problem with delivery or neonate's condition
(3) If unable to obtain cord specimen, aspirate artery on chorionic surface of placenta
(4) Discard cord segment if 5-min Apgar score satisfactory and newbom stable/vigorous

UMBILICAL CORD CLAMPING

Delayed cord clamping at 30–45 s versus 5–10 s decreased intraventricular hemorrhage and sepsis in premature singleton infants (especially males) less than 32 weeks' gestation according to a study by Mercer JS, Vohr BR, McGrath MM, *et al* (*Pediatrics* 2006; 117:235–42)

Delayed cord clamping and waiting 2 min rather than 10 s in normal weight, full-term infants helps prevent iron deficiency from developing before 6 months of age, according to the results of a randomized, controlled trial involving almost400 mother–infant pairs in Mexico City (Chaparro CM, Neufeld LM, Tena Alavez G, *et al*. Effect of timing of umbilical cord clamping on iron status in Mexican infants: a randomized controlled trial. *Lancet* 2006; 367:1997–2004)

URETERAL INJURY

	What % of ureteral injuries are recognized intraoperatively?	25%
	What % occur at uterine artery/cardinal ligament (the distal 3–4 cm)?	75%
	What % of bladder and ureteral injuries occur at hysterectomy?	75%
	Gyn surgery causes this % of urinary tract fistula?	75%
	Abdominal surgery causes	75%
	Vaginal surgery causes	25%
	Most injuries to bladder and urethra in developing countries are from OBSTRUCTED LABOR	
	Most common injury is when ligation of ovarian blood supply at the pelvic brim is performed on the infundibulopelvic ligament. Other areas include the level of the uterine artery and lateral to the vaginal cuff	
Prevent more problems	(1) Anticipate especially in patients with previous surgeries	
	(2) Prevent if possible with identification of ureters	
	(3) Recognize as soon as injury occurs – cystoscopy if needed	
	(4) Evaluate fully and plan repair	
	(5) Repair immediately: % that can be corrected by removing suture	69%
	(6) Test the integrity of the repair	
	(7) Follow up post op to verify the repair remains intact	
Finding the ureter	Locate the umbilical and round ligament. Open the retroperitoneal medial to the external iliac vessels using the umbilical ligament as a landmark. Move the umbilical ligament lateral to ureter as peristalsis is noted as the ureter is palpated or stroked	
Symptoms	The most common symptom is flank pain, which occurs	33–75%
	These occur in what % of gyn surgery and C-sections?	1%
	Ratio of bladder to ureter injury is	5 : 1
Diagnosis	Subtle rise of serum creatinine > preop levels as early as 24 h	0.8 mg/dl
	IVP or US	
	If BILATERAL injury = anuria, increased BUN + creatinine, unresponsive to fluid challenge	
	Patient without renal disease with an increase in this should really be ALERT	1.5 mg/dl
Fistula	Tampon – give Pyridium® p.o. or methylene blue in bladder. If still not orange or blue, give IV indigo carmine. If blue now, suspect uterovaginal fistula	
Treatment	Double J ureteral stent	
	If at level of uterine vessels (4–5 cm from ureterovesicle junction) URETERONEOCYSTOSTOMY (Boari operation or Psoas hitch)	
	If midureter – ureteroureterostomy	
	Extraperitoneal drain, intubate ureter for 10 days and ureteroureteral anastomosis or if 4–5 cm from bladder – plant into bladder	
	(1) Extraperitoneal drain – use a round Jackson–Pratt	
	(2) Intubation – double J ureteral stent	
	(3) Anastomosis suture – use 4–0 Vicryl on SH needle	

URETHRAL INTRINSIC SPHINCTER DEFICIENCY

Diagnosis with low urethral pressures < 20–25 cmH_2O
Leak point > 60 cm with Valsalva
See Urinary Incontinence

URETHRAL SYNDROME

Dysuria, frequency, non-tender. Negative leucocytes on dipstick with low bacterial count. Most common cause is *E. coli* or staphylococci, but rule out herpes, vaginitis, etc.
Consider gen probe
Chlamydia – lacks urgency, hematuria or suprapubic pain. Symptoms gradually > 7–21 days (TCN)
GC – pain and hematuria with rapid onset of symptoms

Treatment
TMP/SMX, nitrofurantoin, Augmentin
Patients sleep through the night, sometimes complain of lower abdominal pressure, dyspareunia

Diagnosis of exclusion
URETHRAL DILATION helpful

Interstitial cystitis
Symptoms – pts void to avoid pain. Get up all night to void
Dxn – glomerulations × 3 or Hunner ulcers
Rx – ELMIRON (pentosan polysulfate sodium) corrects defect in the mucosal GAG layer
Hyperdistention

URINARY INCONTINENCE

Nearly 50% of women > 45 years of age will, at some time, complain of urinary incontinence (Sherburn M, Guthrie JR, Dudley EC, *et al*. Is incontinence associated with menopause? *Obstet Gynecol* 2001;98:628–33)

Risk factors
SUI is influenced by caucasian race, high waist-to-hip ratio, hx of diabetes, age, parity, mode of delivery and possibly genetics

SUI is influenced most strongly by mode of delivery in middle-aged women. Later in life, genetic factors play a more important role in risk of SUI. Elective cesarean protects only against stress incontinence – not other urinary or fecal symptoms
Urge incontinence (DI) is strongly influenced by heritability in both middle-aged and older women

Causal agents
Diuretics, caffeine, anticholinergics, alcohol, narcotics, psychotropics, adrenergics, calcium channel blockers

Common causes
Loss of pelvic support structures, ISD or increased BMI

Diagnosis
Evaluation
(1) History – medical/surgical, obstetric, medications. SUI or DI?
(2) Physical
  (a) Neurologic (sphincter tone, motor/sensory exam)
  (b) Pelvic – assess for estrogen deficiency and loss of pelvic support
  (c) Become accustomed to massaging the anterior vaginal wall underneath the urethra. Any discharge or excretion of fluid from the urethral meatus as massage takes place is pathognomonic for urethral diverticulum. Voiding cystourethrography or MRI will confirm the finding
(3) Labs – urine analysis/culture if U/A is abnormal. Urine cytology if patient is over 50 with irritative symptoms, smoking history, or hematuria
(4) Testing in office establishes diagnosis 75–90%
  (a) Voiding studies – postvoid residual volume normal is < 50 to 100 ml
  (b) Urethrovesical junction mobility – pelvic, US and Q-tip test < 30

(c) Stress test (standing cough test and/or Bonney test)
Patient coughs with full bladder of @ 250 ml
Leakage of urine suggests presence of GSUI
Bonney test (also known as Marshall test) – repeat stress test with anatomic correction (fingers lightly correcting anatomy or other device such as tampon). Should correct leak if GSUI
(d) Single channel standing cystometry (passes first urge test)

Criteria
(1) History of pure SUI without urgency, frequency, nocturia or urgency
(2) Normal neuro exam
(3) Normal postvoid residual volume
(4) Urethrovesical junction hypermobility (+Q-tip test)
(5) Urine leakage during stress test.
(6) Stable bladder during cystometry

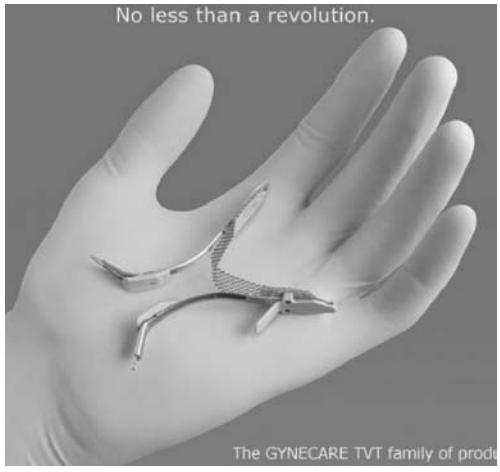


Figure 22 GYNECARE TVT SECUR™ system. This is the third generation of TVTs. ©ETHICON, INC. Reproduced with permission.

What % require more urodynamic testing? 10–25%
Urodynamics in office – UA/UC
SUI is more reliably diagnosed via urodynamic testing than is detrusor overactivity

Office cystometry

(1) Cath for residual < 30–50 cc
Residual should be what after corrective surgery? < 100 cc
What amount of residual urine is consistent with overflow incontinence? > 50 ml
Postvoid residual volume greater than what is considered abnormal? 200 ml
(2) First urge to void 150–200 cc
Most can maintain continence to 400–500 cc
This finding makes DI very unlikely. Also unlikely if plungerless syringe demonstrates no excursion up to 400 cc
Bladder capacity is @ 300 cc
Q-tip test should be < 30–40 degrees
Urethra length 3–5 cm

Treatments for SUI

(1) Incontinence dish – lubricate with estrogen or other cream prior to use. Clean every 4–6 weeks
Autoclave with 5# pressure at 250°F (121°C) × 10 min
or cold cidex or boil × 15 min
(2) Pelvic floor exercises or biofeedback?

(3) HRT in postmenopausal women can increase the blood flow around the bladder neck thus improving SUI after 3 months of treatment

(4) Biofeedback has been shown to be more effective than pelvic floor exercises in treating genuine SUI

(5) FES (functional electrical stimulation), vaginal weights, mechanical devices and extracorporeal magnetic innervation are additional methods that can be used in bladder training

(6) Retropubic urethropexy (Burch) – attach endopelvic fascia to iliopectineal ligament (Cooper's)
Poorly suited for patient with small vaginal size or poor vaginal mobility. Success rate is 80%
Complication rate is 20%
Enterocele develops postop with change in pelvic axis in @ 8%
Significant higher failure rate with laparoscopic Burch

(7) Paravaginal repair – attach arcus tendineus (white line) to endopelvic fascia and bilateral superior anterior vaginal sulcus. This is for lateral defects of the vagina causing SUI or prolapse. The Capio Suture Capturing Device by Boston Scientific (Urology/Gynecology, Natick, MA) can be used laparoscopically to suture during the Burch or the paravaginal repair

(8) Suburethral sling – SUI with ISD (intrinsic sphincter deficiency)
See also Sling Procedure

Diagnosis

ISD – low urethral pressures ≤ 20 cm H_2O and/or leak point with Valsalva that is > 60 cm
Consider low urethral pressure ≤ 35 cm H_2O if patient supine.
Excellent procedure with urethral hypermobility and/or ISD. *See* Sling Procedure; to see how these procedures are informed, see Turrentine JE. *Surgical Transcriptions and Pearls in Obstetrics and Gynecology*, 2nd edn. London: Informa Healthcare, 2006. Graft used is either cadaveric fascia lata, Repliform (Lifecell Corporation, Woodlands, TX; distributed by Boston Scientific, Urology/Gynecology, Natick, MA), DermMatrix (Carbon Medical Technologies, St Paul, MN), Pelvisoft, and others. EndoSurgical Inc., Boston Scientific, American Medical, and many other companies manufacture many TVT and/or TOT kits. In regards to drilling procedures, Precision Twist (Boston Scientific, Urology/Gynecology, Natick, MA) is preferred drill. However, In-Fast drill (American Medical Systems, Minnetonka, MN) is also OK. TVTs, TOT (Also TOP) are even better alternatives that can be performed – these are quicker, easier, and do not require drilling into posterior pubic bone. There are many options available for these procedures. PelviSoft, Gynecare TVT, Synthetic Mesh as IntePro or Biologic Grafts as InteXen LP, and other new innovations are excellent grafts

(9) Durasphere is a good alternative if urethra is not hypermobile and/or if the patient is high risk for major surgery (Carbon Medical Technologies, St Paul, MN; distributed by Boston Scientific, Urology/Gynecology, Natick, MA)

(10) Needle suspension procedures – success rate over 5 years is < 50%
Pereyra – no vaginal dissection, small abdominal incision and #30 silver wire suture
Stamey – endoscopic at bladder neck. #1 cm Dacron to buttress fascia to avoid or decrease risk of suture pulling through fascia

(11) Kelly's operation (bladder neck plication usually with ant. rep)
Long-term success rate is 35–65%
Reserved for those who do not have significant SUI but have cystocele

(12) Vaginal tape procedure may be useful in the treatment of not only incontinence but also a variety of subgroups of incontinence (Mutone N, Mastropietro M, Brizendine E, *et al*. Effect of tension-free vaginal tape procedure on urodynamic continence indices. *Obstet Gynecol* 2001; 98:638–45). However, trials evaluating efficacy of the tension-free vaginal tape operation for urinary incontinence as compared with other established incontinence operations are lacking (Tamussino KF, Hanzal E, Kolle D, *et al*. Tension-free vaginal tape operation: results of the Austrian registry. *Obstet Gynecol* 2001;98:732–6)

Testing after surgery for voiding dysfunction

(1) Voiding efficiency can be predicted in 92% of patients who voided > 50% of 300 ml of instilled sterile water. 100% of patients who voided > 68% of 300 ml of instilled water. If the patients void < 50% of their postresidual – leave catheter!!! Whenever possible, however, remove an indwelling catheter and teach the patient intermittent self-catheterization. If patients are unable to void for up to 2 weeks, offer intermittent self-catheterization. (Kleeman S, Goldwasser S, Vassallo B, *et al*. Predicting postoperative voiding efficiency after operation for incontinence and prolapse. *Am J Obstet Gynecol* 2000; 187:49–52)

(2) Check the Operative Report. If a large cystocele was also repaired during a sling procedure, it is common for there to be some form of retention or voiding dysfunction for 2 weeks or longer. However,if a midurethral sling was done but with no other procedure and there was still retention at 2 weeks post-op, consider the sling may have been placed too tightly

(3) Is there actual (or impending) lower-tract injury or foreign body penetration? Endoscopy of the urethra, vesical neck, and bladder walls will rule this out

(4) Can the patient relax the pelvic floor when she voids? Valium may help the patient relax to void. Avoid urethral dilatation, as it might cause urethral erosion of the sling. Also avoid meds such as bethanechol, as it is ineffective and can cause discomfort

(5) Consider cutting the sling but inform the patient of possible recurrent incontinence. Usually cutting the sling will result in normal voiding. With synthetic, allograft, and xenograft slings, SUI recurs in at least 50% of patients over time compared with an autologous sling, whereas recurrence rates are less than 10%. With synthetic slings, consider reoperating in a few weeks. For a patient with retention who has an autologous, allograft, or xenograft sling, it is best to wait approximately 3 months before operating

Detrusor instability (DI)

OAB is caused by involuntary bladder contractions, which create bladder pressures high enough to overcome the continence mechanism

Symptoms and diagnosis

Urgency, frequency, nocturia. Urgency prior to urinary leak

Decreased postvoid residual volumes. CMG to definitely diagnose

Treatment options

(1) Bladder retraining – "Bladder drills" micturate at regular intervals and suppress urge to void between these times — 66% success
Start with behavorial therapy for DI first
Avoid bladder irritants (caffeine, nicotine, spicy foods)

(2) Pharmacotherapy

(A) *Anticholinergic/antispasmodic agents*:
Oxybutynin (Ditropan®) XL (5 mg t.i.d.) or — 10–15 mg daily
Oxybutynin Transdermal Patch (Oxytrol): Patch applied twice weekly

(B) *Tricyclic antidepressant; locally antispasmodic and also acts centrally:*
Imipramine (Tofranil) — 10 mg b.i.d.

(C) *Muscarinic receptor antagonists:*
Tolterodine tartrate (Detrol) — 2 mg b.i.d.
or Detrol — 4 mg LA daily
Solifenacin succinate (Vesicare) — 5 mg and 10 mg daily
Trospium chloride (Sanctura) — 20 mg b.i.d.
Darifenacin (Enablex) — 7.5 to 15 mg daily

(D) *Pain drug for OAB*:
Tramadol (Ultram) — 100 mg b.i.d. for 12 weeks
Tramadol was effective for reducing the number of urge incontinence episodes

Both oxybutynin and tolterodine reduced urge incontinence, but oxybutynin also reduced urinary frequency

Overflow incontinence	*Symptoms* – constant wetness, intermittent dribbling, SUI (not GSUI), voiding difficulty, recurrent infections, suprapubic discomfort *Diagnosis* – postvoid cath > 50 cc (usually exceeds 350 cc) *Treatment* – clean intermittent self-catheterization Caused by abdominal or pelvic surgery, fecal impaction, infection, L&D, neuro conditions, obstructions, pharmacologic, diabetes, MS, spinal cord tumors and psychiatric
Potential incontinence	Occurs temporarily when severe prolapse is mechanically reduced such as use of a pessary
Mixed incontinence	What % of patients have both GSUI and DI? 30% What % are corrected with surgery? 50% Mixed incontinence can be better determined by urodynamic studies, aided by a provocative measure, such as a cough or standing heel bounce. It results in a leakage of urine that appears to be stress induced, but actually is caused by a detrussor contraction
Increase fluid volume intake	Weight watchers, increased fluid. Voiding diary to diagnose How much fluid intake per day is correct? @ 1600 cc One should drink when thirsty or until urine is clear. Drinking eight 8-ounce glasses of H_2O per day is calculated for 70 kg man Could be too much
Extraurethral incontinence	Involuntary loss of urine due to anatomic bypass of normal continence mechanisms (i.e. vesicovaginal fistula, ectopic ureter, urethral diverticulum)
Multichannel urodynamics (cystometrics)	Indicated for: (1) Failed non-surgical intervention (2) Failed incontinence surgery (3) High postvoid residual volumes or "continuous leakage" (4) Older female with medical problems (5) Neurological disease What % of neuro disease is present with incontinence? 16–25%
Other tests	*Levator ani electromyography* Useful when diagnosis of neurogenic bladder is suspected to more fully assess degree of neurologic deficit *Cystourethroscopy* Indicated: (1) If lesions of bladder or urethra are suspected (2) Hematuria (3) Persistent discomfort *Maximal urethral closure* Indicated to evaluate if ISD is present (1) Maximal urethral closure pressure in the supine position is 38.45 cm ± 2.58 cm of water (2) Maximal urethral closure pressure in the sitting position is 22.80 cm ± 3.2 cm of water According to one study, the cutoff point for diagnosis of intrinsic sphincteric deficiency should be raised to 35 cm of water as compared to 20 cm of water when the supine position is used for measurement (Krissi H, Pansky M, Halperia R, Langer R. Maximal urethral closure pressure <20 cm H_2O: Does it predict ISD? *J Reprod Med* 2005; 50:824–6)
Other treatments	*Biofeedback treatment* (1) Magnetic neuromodulation – extracorporeal magnetic innervation effective for SUI, urge or mixed UI. Patient sits in chair – magnetic pulses. 10 min at 5 Hz then 10 min at 50 Hz twice weekly for 8 weeks (2) Pelvic power program – disk on wrist vibrates when to perform Kegel's every 2 h – rings when patient is to urinate. Can be programmed to change length (3) FemiScan – home muscle monitor with headset. Instructions. Visits – computer based in office

(4) Estring – soft, flexible, silicone ring that is inserted like a diaphragm into upper part of vagina. Releases estrogen × 3 months at rate of 7.5 μg /24 h × 90 days (normally takes 2–3 weeks for symptoms to manifest). Improves vaginal and urinary symptoms and mucosal appearance without provoking bleeding

(5) Anti-incontinence devices
- (a) Incontinence dish or ring (Milex 1-800-621-1278)
  If estrogenized, remove only for coitus and check every 3–4 months. If unestrogenized, need to remove nightly or every other night
  Risk of vaginal erosion – check every 2–3 months
- (b) Conveen continence guard
  One time polyurethane foam that expands to vagina. It absorbs vaginal secretions and worn morning to night. European manufacturer recommends removal every 4 h during menses due to theoretical risk of toxic shock
- (c) FemAssist (Insight Medical, Marlboro, MA 1-800-232-4344)
  External urethral cap that may be used for 1 week (2 sizes)
- (d) FemSoft Insert (Rochester Medical Corp., Stewartville, MN 1-507-533-9600)
  Transurethral device (silicone) inserted into urethra. Must be changed at every void or after 6 h. Cost is < $2 per insert
  Rate of UTI is 22%
- (e) Complex valve catheters that have one-way valves to allow urination while insert is in place are under investigation/not on the market as of this publication

(6) Transvaginal electrical stimulation – twice daily for 8 weeks
  Cure rate for DI is 50%
  Cost is $500

(7) Sacral Neuromodulation Stimulation (SNS, "*Interstim*")
  Approved for pharmacological and behavioral failures.
  Pain, wound problems, or lead fracture led to surgical revision 15.5%
  See how procedure is performed in Turrentine JE. *Surgical Transcriptions and Pearls of Obstetrics and Gynecology*, 2nd edn. London: Informa Healthcare, 2006

(8) Botulinum A toxin is a promising alternative to first-line drug therapy for refractory detrusor overactivity. (Kuo H-C. Urodynamic evidence of effectiveness of botulinum A toxin injection in treatment of etrusor overactivity refractory to anticholinergic agents. *Urology* 2004; 63:868–72)

Urinary innervation of bladder

CNS – continence, norepinephrine, sympathetics

MAP – parasympathetic, acetylcholine, contraction

Urological endoscopes

Urethroscope – lens is what degree? 0
  sheaths are 15F, 18F, 24F
Cystoscope – lens is what degree? 30 and 70
  sheaths are 17F, 19F and 21F (for bx)

Urine residual increased

MS, recent surgery, post herpes simplex genital infection, recent delivery, hypotonic bladder dysfunction (DM or hypothyroidism)

Nerve supply for bladder and urethra

Sympathetics T11–L2
Parasympathetics and pudendal nerve S2–S4

Nerve testing

Bulbocavernosus reflex (lateral labia minora) and clitoral reflex test the nerve supply + when anal sphincter reacts. (Evaluate estrogen effect at this time too)

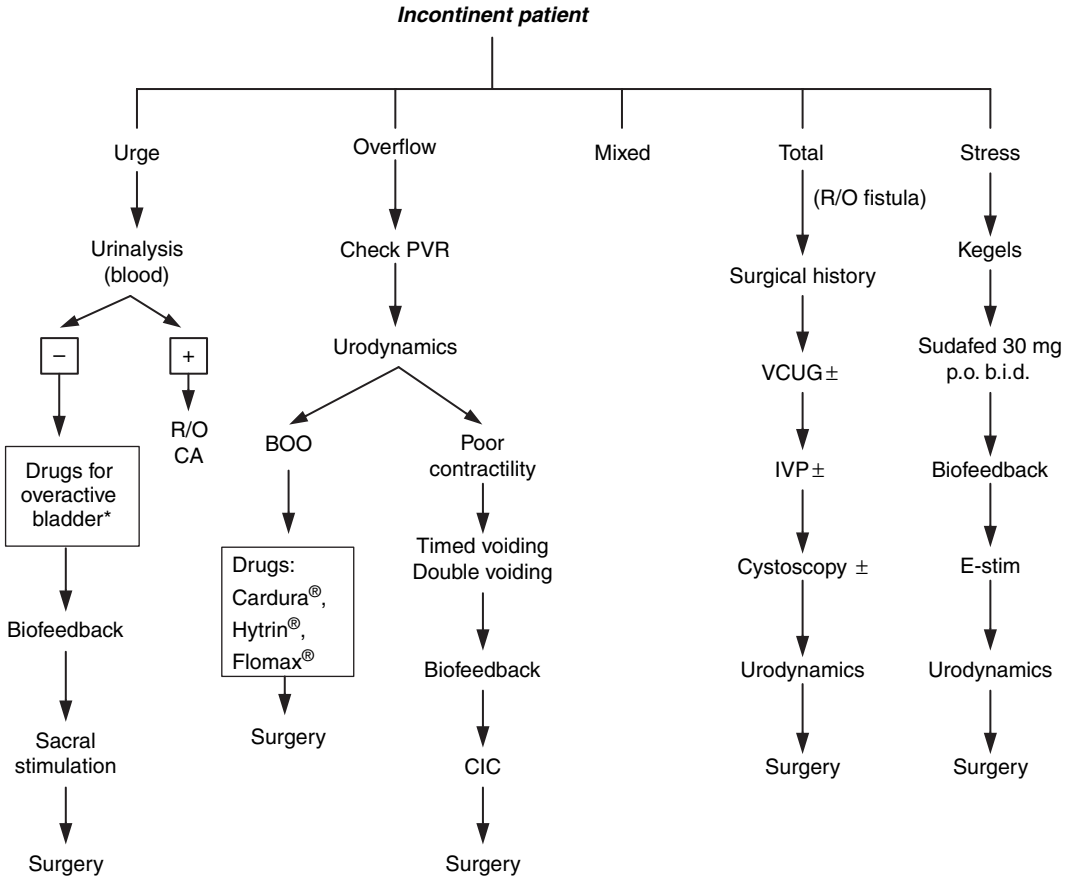
Fistula evaluation

Tampon – methylene blue 200 ml in bladder – ambulate 15 min – STAIN – vesicovaginal fistula
Tampon – indigo carmine 1 ml
IV – repeat ambulation 15 min – STAIN – ureterovaginal fistula

Evaluation for sling?

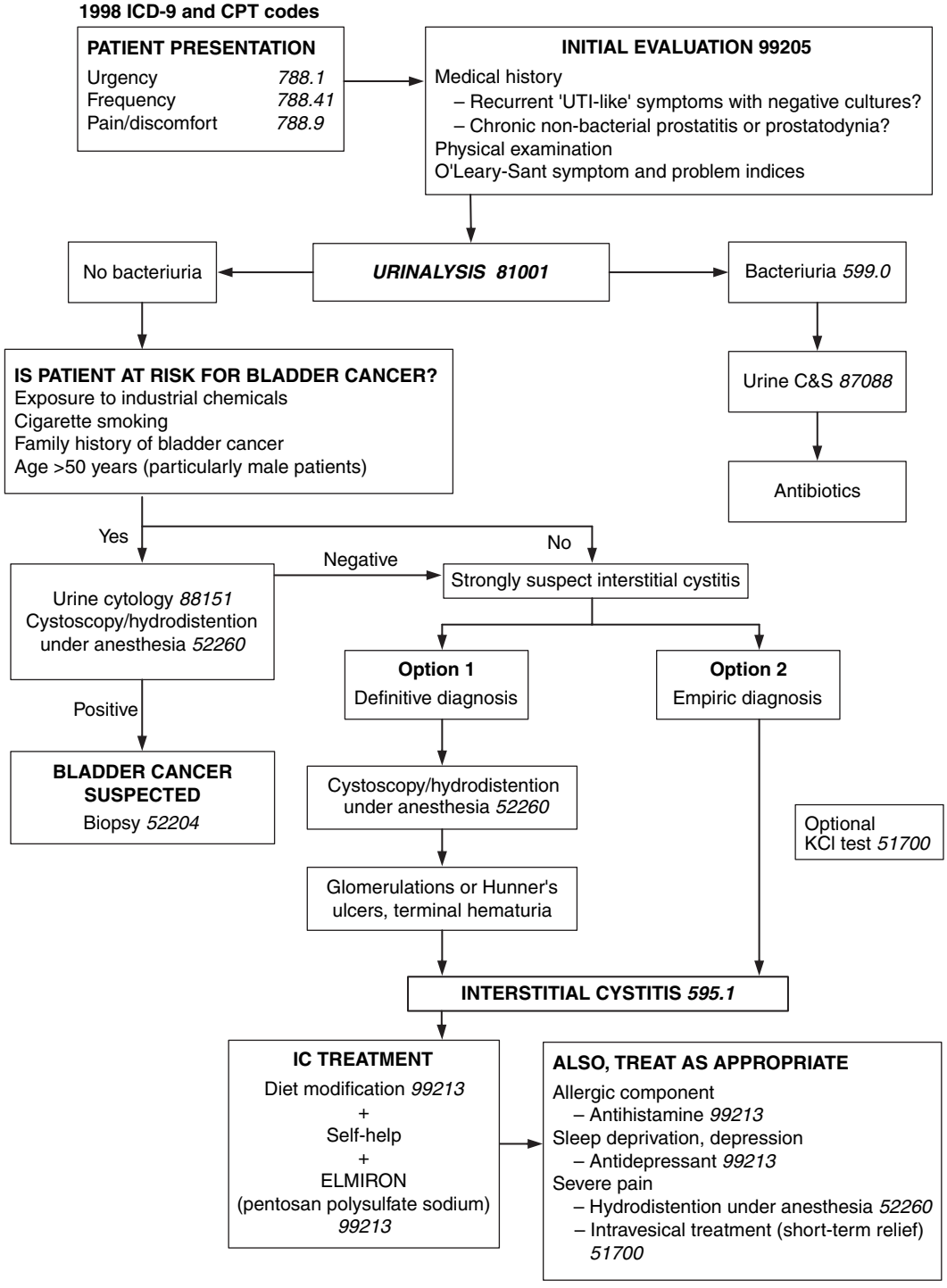
Urethroscopy
Withdraw till UVJ closes 1/3 way then
"Hold urine" or "squeeze rectum" – closes
"Strain" or "cough" – opens
Mobility during above procedures or flaccid, short, open entire distance = Type III incontinence of Blavius associated with decreased urethral closure or ISD

	Treatment would require sling, periurethral collagen or artificial sphincter. The Q-tip test may be unnecessary in patients who demonstrate any advanced pelvic prolapse since virtually all of these patients will have urethral hypermobility. (Cogan SL, Weber AM, Hammel JP. Is urethral mobility really being assessed by the pelvic organ prolapse quantification (POP-Q) system? *Obstet Gynecol* 2002; 99:473–6)
CMG (cystometrogram)	Indications (1) Urgency, urge incontinence, frequency (2) Sudden urinary loss or GSI to rule out DI (3) GSI for possible surgery or > age 50 (4) Recent incontinence after surgery Pressure should not rise > 15 cmH_2O This is a study of pressure/volume relationship in bladder during fill
Multichannel urodynamics	Multiple transducer catheters Vagina – intra-abdominal pressure Urethral closure pressure – urethra/bladder pressure 40–60 cmH_2O True detrusor pressure – bladder/intra-abdominal pressure 2.5–3.5 cmH_2O *Diagnosis* – GSI when urethral closure pressure at cough without DI = 0 Damaged urethral sphincter with urethral closure pressure < 20 cmH_2O sitting
Pearls	Suspect detrusor instability if urethral opening is uncontrollable with or without leaking @ scope. Urethral syndrome – suspect if exudate is seen when withdrawing urethroscope with finger against urethra through bladder. Diverticula are usually seen posterior and lateral and are multiple what % of the time? 50%
Inhibit voiding	*Antispasmodics* Oxybutynin (Ditropan XL) 5–10 mg t.i.d. Tolterodine tartrate (Detrol) 4 mg daily (Detrol is a potent antimuscarinic to decrease detrussor symptoms) Bentyl® 10 and 20 mg capsules Urispas® (flavoxate) 100 mg 2 q.i.d. *Tricyclics* Tofranil (imipramine) 50–150 mg daily. (Good for mixed and detrusor instability especially for nocturnal frequency and urge incontinence due to sedative effect.) Start with 25 mg b.i.d. Sinequan® (doxepin) 75–150 mg/day *Anticholinergics* Pro-Banthine® (propantheline bromide) 15 mg t.i.d. – q.i.d. Cystospaz® (hycosymine) 0.15 mg t.i.d. – q.i.d. or Cystozpaz® M 0.375 mg t.i.d. – q.i.d. *Urethral contraction* Tofranil (imipramine) 50–150 mg daily (Tofranil is supplied in 25, 50 and 100 mg tablets) Ephedrine Phenylpropanolamine (Propagest®) 75–150 mg q. daily. (Good for mild to moderate SUI)
Promote voiding	*Urethral relaxants* Aldomet (methyldopa) 250–500 mg b.i.d. Phenothiazines Phenoxybenzamine (Dibenyline®) Bladder contractants Prostigmine® (neostigmine) Urecholine® (bethanechol) 25 mg b.i.d.



*Dicyclomine HCl 10–20 mg t.i.d.
Flavoxate HCl (Urispas) 100–200 mg t.i.d.
Imipramine HCl (Tofranil) 10–50 mg b.i.d.
Oxybutynin HCl (Ditropan) 2.5–5.0 mg t.i.d.– q.i.d.
Controlled-release oxybutynin (Ditropan XL) 5–20 mg q.d.
Propantheline bromide (Pro-Banthine) 15 mg t.i.d.
Tolterodine (Detrol) 2 mg b.i.d. + Pyridium Plus
(Pyridium 150 mg + hyocyamine HBr 0.3 mg + butabarbital 15 mg) one q.i.d.

Interstitial cystitis (IC): diagnosis and treatment algorithm



URINARY TRACT INFECTION (UTI)

Cystitis

Increased risks are sexual activity or decreased estrogen
Diagnosis – RBCs indicative of cystitis
10 to 5th CFU (colony forming units) correlates with unspun magnification (400 ×) with + bacteria moving 2–6 WBCs per HPF or clumps of leukocytes with minimal epithelial cells
Dipstick + nitrites
Produced by bacterial enzyme, nitrate reductase on dietary nitrates

Lower UTI

(1) Cystitis
(2) Acute urethritis
(3) Chronic urethritis

Treatment – 3-day course of TMP/SMX 160/800 mg #6 every	12 h
Nitrofurantoin 100 mg #6 every	12 h

Fluoroquinolones → very effective × 3 days but very expensive
Ciproxin®, Noroxin®, Floxin® or Penetrex®
Reserve these for recurrent infections or allergies
Cephalosporins and ampicillin → increased expense and increased complications and resistance
Recurrent cystitis – treat with quinolones (Tequin 400 mg daily or Levaquin 500 mg daily)
Consider continuous or postcoital therapy using nitrofurantoin or TMP/SMX

Upper UTI

Pyelonephritis
Treatment
Outpatient → TMP/SMX 160/800 mg p.o. q. 12 h or a quinolone (Tequin or Levaquin) daily
In hospital (if evidence of sepsis)
(1) Ceftriaxone 1–2 g IV q. 24 h
(2) Gentamicin 1 mg/kg of body weight IV q. 8 h with or without ampicillin 1 g IV every 6 h
(3) Ofloxin or ciprofloxacin 200–400 mg IV q. 12 h

If evidence of sepsis – ceftriaxone IV every 24 h	1–2 g

Most common organism	*E. coli*	35%
Increased risk	Decreased estrogen and increased sexual activity	

Urethral syndrome

Dysuria, frequency, non-tender. Negative WBCs on dipstick, decreased bacterial count
Rule out HSV, do GENPROBE to rule GC and *Chlamydia*. Could be *E. coli* and/or staphylococci

UTERUS (ABNORMAL)

Septate

Partial resorption of midline septum
Total failure – long vaginal septum (double vagina)

Increased PTL and spontaneous abortion rate	88%

Dxn – vag US and MRI. Evaluate urinary tract
Treatment – Resect with hysteroscopy

Bicornuate

Partial lack of fusion of Müllerian ducts
Relatively common. Pregnancy outcome near normal

Increase PTL and spontaneous abortion rate	70%

Treatment – Strassmann metroplasty. Use tourniquets at cervix and infundibular ligaments. CD recommended unless performed hysteroscopically

Uterus didelphys

Abortion rate	30%

Treatment – Jones–Tompkins

Uterus unicollis

Abortion rate	15%

Treatment – McDonald's cerclage

Chance of live-born infant with septate and bicornuate uterus is	60%
Chance of live-born infant with unicornate and didelphic uterus is	40%

Most common associated anomaly with unicornate uterus is RENAL AGENESIS. IVP diagnosis 30–50%
(Usually opposite side)

Septate uterus

Bicornuate uterus

UTERINE ABLATION

Methods

Laser, roller ball, roller barrel
Uterine balloon ablation (Thermachoice, Gynecare, Somerville, NJ).
Cryoblation therapy ("heroption" – CryoGen, San Diego, CA)
Bipolar mesh (Novacept, Palo Alto, CA)
Hydro ThermAblator (Boston Scientific, Urology/Gynecology, Natick, MA)

UTERINE ARTERY EMBOLIZATION

How the procedure is done

(1) Patient is hydrated intravenously
(2) Given conscious sedation after informed consent is given
(3) 1% lidocaine given for local anesthesia
(4) 5-French vascular cath is placed in right common femoral artery
(5) 5-French "hook-shaped" cath advanced into abdominal aorta (Omni Flush Angiodynamics Inc., Queensbury, NY)
(6) AP and oblique abdominal digital subtraction arteriograms done
(7) Withdraw hook cath so tip "straddles" iliac bifurcation
(8) Floppy-tipped guidewire is advanced "up and over" bifurcation
(9) "Hook-shaped" cath withdrawn and exchanged for hydrophilic-coated 'hockey stick-shaped' cath (JB!, Bentson-Hanafee-Wilson 1, Glide Cath, Meditech, Boston Scientific, Urology/Gynecology, Natick, MA)
(10) This cath is advanced into main trunk of contralateral (left) internal iliac artery and mapping via fluoroscopy is done to map the tortuous path of the uterine artery
(11) PVA (polyvinyl alcohol particles 500–710 u) are suspended in contrast material and injected until stasis of left uterine artery is achieved (PVA by Contour, Boston Scientific, Urology/Gynecology, Natick, MA; Meditech, Target Therapeutics, Fremont, CA). Stasis is when forward flow stops
(12) The cath is withdrawn, tip into ipsilateral (right) internal iliac artery and same done on other side

Future pregnancy?

Gelfoam can be used rather than the permanent PVA. However, long-term studies will have to be conducted before questions of fertility and ovarian function can be answered (small % have ovarian failure)

Post UAE care

Patient needs about 1–2 weeks before resuming her routine. Most common complaint after UAE is pelvic pain – PCA is given IV antiemetics are given every 8 h on fixed dose schedule. IV antibiotics are continued for 24 h
Follow-up care is at 3, 6 and 12 months. (MRI at 6- and 12-month visit)

Outcomes

Technical success rate	98%
Bleeding and other fibroid-related symptoms resolved	80–90%

Complications

Substantial pain for	8–12 h
Less severe pain for the following	3–5 days
Fever of 38°C (100.4°F) is experienced by	33%
Ischemia-related postembolization syndrome	10%
Permanent amenorrhea	2%

	Deaths from septicemia 2/6000 (known worldwide cases) Pyometria and expelling necrotic fibroids vaginally rare but has occasionally happened
Radiation exposure	22.34–162.32 cGy for UAE. This compares to: Hysterosalpingography 0.04–0.55 cGy CT of trunk 0.1–1.9 cGy Pelvic irradiation for Hodgkin's disease 263–3500 cGy UAE is "unlikely to result in acute or long-term radiation injury to the patient or to a measurable increase in the genetic risk to the patient's future children." (Nikolic B, Spies JB, Lundsten MJ, *et al*. Patient radiation dose associated with uterine artery embolization. *Radiology* 2000; 214:121–5)
Durability of the procedure	Unknown at the time of this publication

UTERINE BLEEDING

Decreased	Oligomenorrhea – infrequent, irregular episodes of bleeding How many days between cycles? > 37 Hypomenorrhea – regular but decreased bleeding Amenorrhea – no period for how many months? 6
Increased	Menorrhagia – excessive bleeding in amount and duration What is the amount and duration? 85 cc or > 7 days Metrorrhagia – usually not excessive, occurs irreg intervals Menometrorrhagia – usu. excessive occurring at irreg intervals Polymenorrhea – frequent but regular episodes of uterine bleeding, usually occurring at intervals of how many days or less 21
Management in adolescents	Within the first year of menarche approximately 55% of cycles are anovulatory. The hypothalamic–pituitary–ovarian axis takes time to mature and to develop its finely tuned feedback system. Up to a third of adolescents still have anovulatory cycles in the fifth year of menarche
Possible causes of menorrhagia	*Anovulation* Hypothalamic dysfunction Polycystic ovary disease *Pregnancy-related conditions* Threatened or spontaneous abortion Retained products of conception after elective abortion *Primary coagulation disorders* *Systemic diseases* Diabetes mellitus Hepatic dysfunction Renal dysfunction Thyroid dysfunction *Trauma* Accidental injury Coital trauma Sexual abuse *Lower reproductive tract infections* Chlamydia Pelvic inflammatory disease *Neoplasms* Endometrial hyperplasia Hormonally active ovarian tumors Leiomyoma Vaginal tumors *Iatrogenic causes* Exogenous hormone use Ingestion of medications containing estrogenic activity

Office evaluation of bleeding

(1) A complete menstrual history, including the following:
  (a) Date of menarche
  (b) Frequency and regularity of menstrual cycles
  (c) Date of onset of most recent period or bleeding episode
  (d) An estimate of the number of pads used per day
  (e) Whether the patient has cramps or pain, clotting or symptoms of syncope or nausea with menses
(2) Ask about history of excessive bleeding after surgical or dental procedures and any family history of endocrine or coagulation disorders
(3) Ask the patient whether she has been sexually active; whether she has used any method of contraception; and whether she feels there is any possibility of pregnancy. This interview must be done in privacy, after an explanation to mother and daughter of the importance of confidentiality in the relationship of a physician to an adolescent

Laboratory tests

Complete blood counts
Platelet counts
Pregnancy test
Thyroid function test

For severe bleeding
  bleeding time
  partial thromboplastin time
  prothrombin time
  serial hemoglobin and hematocrit
  type and screen

Therapy

A patient who is mildly anemic will benefit from hormonal management
(1) Combination low-dose oral contraceptive; then re-evaluate after 3–6 cycles to decide whether to continue this regimen
(2) An alternative is: medroxyprogesterone 5–10 mg/day for 10–14 days

Patients with heavy bleeding, but who are stable, will require higher-dose hormonal therapy
(1) Monophasic OC (Ovral) two pills until stop bleeding – then one daily

Acute bleeding: emergency management

(1) Either conjugated estrogens 25–40 mg IV every 4–6 h or oral estrogen 2.5 mg every 6 h, will be effective × 24 h
(2) If not, a D&C is indicated
(3) The failure of hormonal management suggests that a local cause of bleeding is more likely
(4) If IV or oral estrogen controls the bleeding successfully oral progestin therapy must be added and continued for several days to stabilize the endometrium. This therapy can be accomplished by switching to a combination oral contraceptive
(5) Remember that up to 19% of patients hospitalized with heavy uterine bleeding had an underlying coagulation disorder

UTERINE CANCER

For stages of uterine cancer, see Oncology, under Uterus

Increased risks

Nullip	2–3 ×
Menopause and > 52 years of age	2–3 ×
Overweight by 21–50 pounds	3 ×
over 50 pounds	10 ×
Unopposed estrogen therapy	8 ×
Diabetes	2 ×

Other risk factors → early menarche, late menopause, increased B/P, estrogen-secreting tumors, history of pelvic radiation therapy

Work-up for endometrial cancer

Endometrial biopsy and ECC
Fractional D&C

Examine:
(1) Cervix, vagina, parametria and adnexa
(2) Supraclavicular and inguinal nodes
(3) Abdomen

Obtain:
(1) CXR
(2) Labs (electrolytes, CBC, liver and renal status, U/A)

Consider:
(1) Sigmoidoscopy/colonoscopy
(2) IVP
(3) CA-125
(4) CT and/or MRI

Endometrial cancer and radiation

Survival rate similar with and without radiation plus surgery especially with Grade 1 + 2 lesions. However, if poorly differentiated – radiation and surgery

Stage I

10% medically inoperable. D&C after how many months to reassess?	3 months
TAHBSO and cytology	Grade 1
Controversial	Grade 2
TAHBSO and cytology with pelvic and periaortic node dissection	Grade 3
Radiation therapy – poor prognostic factors or inoperable. Positive lymph node involvement?	
Positive cytology is controversial. Second opinion is good policy	
Survival rate for Stage I is	85%

Stage II

TAHBSO and cytology with pelvic and periaortic lymph node dissection (patients with lymphadenectomy did better without radiation)	
Endometrial cancer with endocervical involvement – radical hysterectomy with pelvic lymphadenectomy and periaortic lymphadenectomy	
Survival rate for Stage II is	60%

Stage III and IV

Individualize	
Usually hormone rx or chemo rx or both in addition to surgery and radiation therapy	
Survival rate for Stage III is	30%
Survival rate for Stage IV is	10%

Low risk

Grade 1 or 2 with superficial or no myometrial invasion 1/3

Intermediate risk

Grade 1 or 2 with mid 1/3 invasion (no external uterine spread)

High risk

Grade 3 or outer 1/3 invasion into myometrium. Give whole pelvis radiation

UTERINE INVERSION

Incidence	1/2000–1/2500
Corpus to cervix	Grade 1
Corpus through cervix	Grade 2
Uterus to perineum	Grade 3
Vagina with uterus	Grade 4

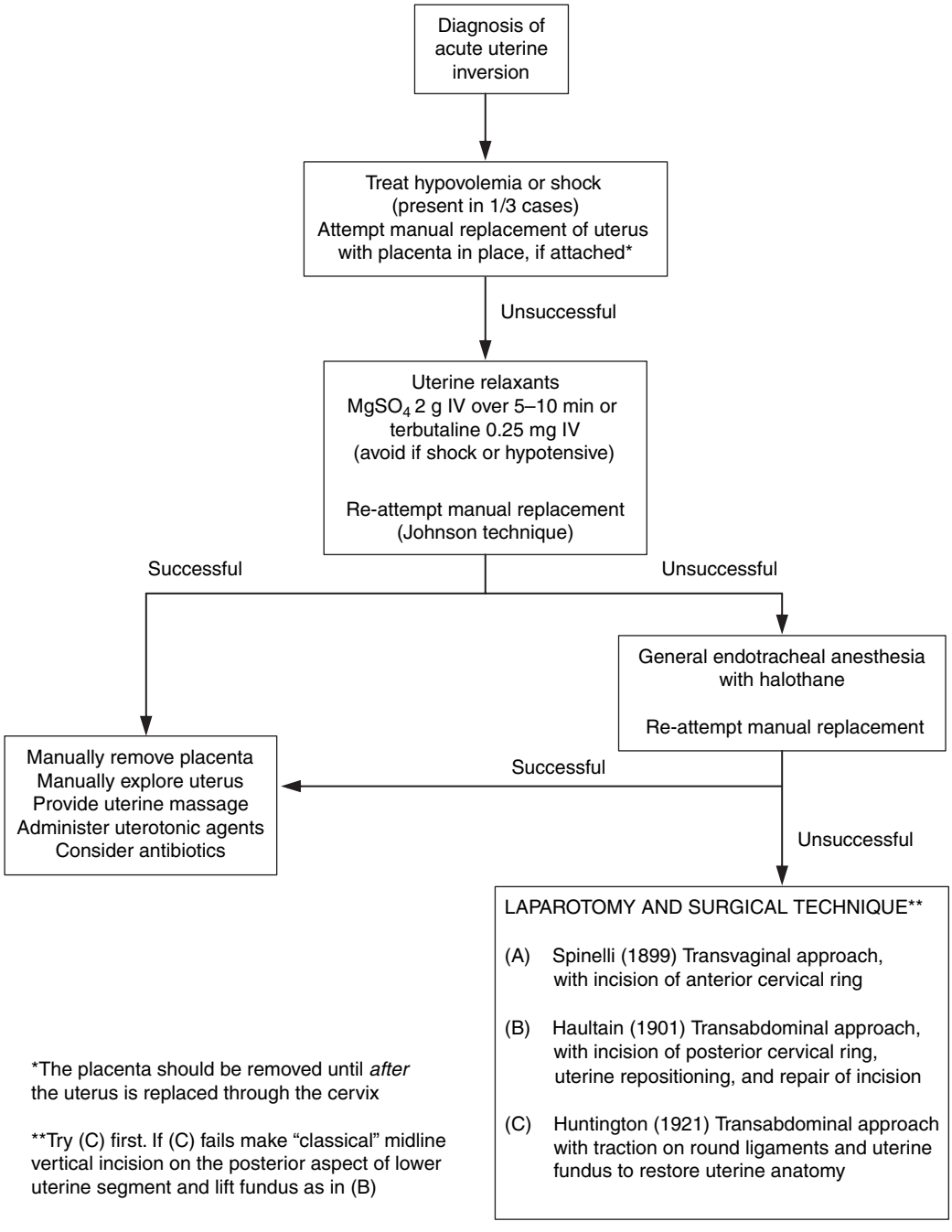
Treatment

(1) Johnson technique – do not remove placenta until replaced

Except with decreased B/P, RELAX uterus with IV Brethine®	250 µg
or with nitroglycerine	125 µg
Slow $MgSO_4$ if hypotensive	2–4 g

(2) Round ligament technique
(3) Midline vertical posterior incision
Give two large IV lines
Give Pitocin or Hemabate after replacement of uterus

Management of acute puerperal uterine inversion



*The placenta should be removed until *after* the uterus is replaced through the cervix

**Try (C) first. If (C) fails make "classical" midline vertical incision on the posterior aspect of lower uterine segment and lift fundus as in (B)

UTERINE RUPTURE

While there are specific risk factors associated with uterine rupture, the prediction of who might rupture their uterus and how to prevent it is extremely difficult. (Diaz DS, Jones JE, Seryakov M, *et al*. Uterine rupture and dehiscence: ten-year review and case–control study. *South Med J* 2002; 95:431–5)

Incidence 1 in 1148–2250

Types of uterine rupture

(1) Complete
(2) Incomplete

Classic signs

(1) Vaginal hemorrhage
(2) Shock
(3) Cessation of labor
(4) Recession of the presenting part

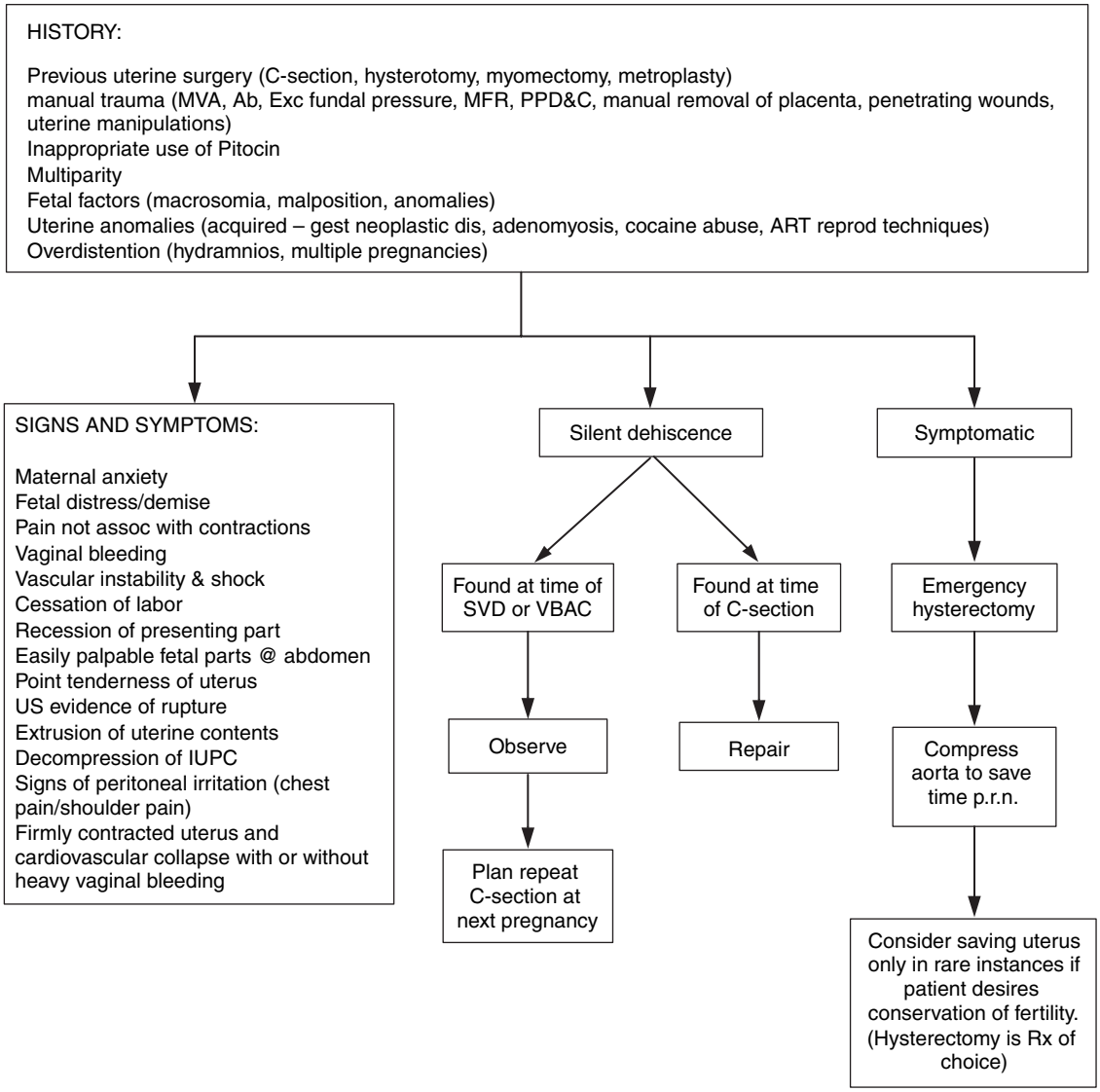
78% of patients with uterine rupture have evidence of fetal distress prior to onset of bleeding or pain. Fetal distress and loss of uterine contractions in patients with a history of previous uterine scar puts diagnosis of uterine rupture high on differential diagnosis

Examine uterus directly after delivery of placenta and before uterus contracts

Management

(1) Silent dehiscence
    (a) SVD – observation with expectation of spontaneous healing – plan repeat C-section
    (b) Repeat C-section – repair at time of repeat C-section
(2) Symptomatic rupture – emergency hysterectomy
    Causes of emergency hysterectomy:
    (a) Atony 43%
    (b) Placenta accreta 30%
    (c) Uterine rupture 13%
    (d) Extension of low transverse scar 10%
(3) Complete rupture
    (a) Intact uterus 13.5% maternal mortality
    (b) Scarred uterus 0% maternal mortality
    (c) Intact uterus 76% fetal mortality
    (d) Scarred uterus 32% fetal mortality

Suspect uterine rupture



UTERINE TACHYSYSTOLE

How many contractions in 10 min? 6 or >

VACCINES

May give these as if non-pregnant:
Tetanus (post exposure prophylaxis)
Rabies (post exposure prophylaxis)
Influenza (underlying diseases, patient request, health-care worker)
Pneumococcus (same as non-pregnant)
Hepatitis B (with indications)

In pregnancy

NEVER GIVE THESE: MMR, Pertussis

May give others if high risk or traveling to endemic areas – hepatitis B, yellow fever, cholera, polio, etc.

Live attenuated viruses – MMR and varicella
Killed viruses – hepatitis B, influenza, rabies, polio (Salk)
Killed bacteria – cholera, meningococcus, pneumococcus, typhoid, plague and pertussis
Toxoids – anthrax, tetanus–diphtheria
Indications for hepatitis B vaccination – drug abuse, health-care worker, newborn, sexual promiscuity

Evidence for immunity against measles and rubella:
Birth before 1957
Serologic evidence of immunity. Documentation of physician-diagnosed infection (for measles and mumps but not rubella).
Documentation of adequate vaccination

Passive immunization of the fetus achieved through maternal vaccination is likely with:
Protection against neonatal tetanus
Reduced neonatal morbidity of influenza in newborns
Potential to decrease neonatal morbidity associated with respiratory syncytial virus and *Haemophilus influenzae* b

Immunizations – general

Vaccinate according to age group and risk factors

Age 13–18

Tetanus–diphtheria booster (age 14–16 × 1)

At-risk groups:

(1) Child-bearing age and no evidence of immunity – MMR
(2) Blood products, household/sexual contacts of Hep B carriers, multiple sexual partners in past 6 months – Hep B vaccine

Age 19–65

Tetanus–diphtheria booster (every 10 years)
Influenza vaccine (every year starting at age 55)
At-risk groups:

(1) Child-bearing age and no evidence of immunity – MMR
(2) IV drug users; blood products recipients; health-care workers; household/sexual contacts of Hep B carriers; multiple sexual partners in past 6 months – Hep B vaccine
(3) Chronic cardiopulmonary disease; metabolic diseases; diabetes, hemoglobinopathies, immunosuppression, renal dysfunction – influenza vaccine annually
(4) Conditions prone to pneumococcal infection (i.e. immunosuppression), chronic cardiopulmonary disease, sickle cell disease, renal disease, s/p splenectomy, diabetes, alcoholism, cirrhosis – Pneumovax

Age 65+

Tetanus–diphtheria booster (every 10 years)
Influenza vaccine (annually)
Pneumovax (once)

At-risk groups:

(1) Exposure to blood products; household/sexual contacts with chronic Hep B carriers – Hep B vaccine

Immunizations in pregnancy

Theoretical concern of congenital infection by live vaccines during pregnancy (no reported cases)
Must weigh several factors: risk of exposure, maternal risk, fetal risk, risk from vaccine/toxoid

Rule of thumb: No live vaccines unless:

(1) Susceptibility/exposure probable and
(2) Disease threat to woman/fetus – vaccine risk

Only routinely administered immunizations during pregnancy:

(1) Tetanus–diphtheria toxoids
(2) At-risk group for Hep B virus (see above)

MMR: 3 months before pregnancy or immediate postpartum

Polio/yellow fever vaccine – when traveling to endemic area

Immune globulins:

(1) After exposure to: measles, Hep A, B, tetanus, chickenpox or rabies
(2) VZIG for newborns of mothers who develop chickenpox 5 days before, until 2 days after delivery
(3) All women without a history of chickenpox should be passively immunized with VZIG within 96 h of an exposure to chickenpox

Indications for vaccines and immune serum globulins during pregnancy

Immunizing agent	*Indications*
Vaccines	
Live virus	
Poliomyelitis (Sabin)	Immediate protection against poliomyelitis for previously unimmunized individuals
Yellow fever	Travel to endemic areas
Measles	Contraindicated
Mumps	Contraindicated
Rubella	Contraindicated
Live bacteria	
Tularemia	Rabbit handlers, laboratory workers
Bacille Calmette-Guérin	Not recommended
Killed virus	
Hepatitis B	Pre- and postexposure prophylaxis for individuals at high risk
Influenza	Chronic cardiopulmonary or renal disease; diabetes mellitus
Poliomyelitis (Salk)	Travel to epidemic areas; laboratory workers
Rabies	Exposure to potentially rabid animals
Killed bacteria	
Cholera	Entry requirement for some countries
Meningococcus	Epidemic meningococcal–non-B disease
Plague	Laboratory workers; travel to areas with human disease
Pneumococcus	Cardiopulmonary disease, splenectomy, alcoholism, Hodgkin's
Typhoid	Household contact with chronic carrier; travel to endemic areas
Pertussis	Not recommended
Toxoids	
Anthrax	Laboratory workers; handlers of furs and animal hides
Tetanus–diphtheria	Primary immunization; booster
Immune globulins	
Pooled human	
Hepatitis A	Pre- and postexposure prophylaxis
Measles	Postexposure prophylaxis
Hyperimmune	
Hepatitis B	Postexposure prophylaxis
Rabies	Postexposure prophylaxis
Tetanus	Postexposure prophylaxis
Varicella zoster	Postexposure prophylaxis
Horse serum	
Botulism	Treatment of infection
Diphtheria	Treatment of infection

Immunizations for children

Although we do not give immunizations to pediatric patients, we are often asked by mothers about the times when children are due for their immunizations. This list should help answer those questions. Adults might also require some of these vaccines

Hepatitis A

Two doses needed 6 months apart. (Brands can be used interchangeably)

Hepatitis B
1 month (Hep B-1)
2 months (Hep B-2)
12–15 months (Hep B-3)
11–12 years (Hep B*) (For those who have not completed the full series of three doses)

Tdap (tetanus and diphtheria toxoids with acellular pertussis) or Tp (DTP)
2 months
4 months
6 months
15–18 months
4–6 years
11–16 years *Td (Tetanus booster)*
A one-time dose of Tdap should replace a dose of Td for any adult younger than 65 years, either as part of a primary series of tetanus and dipththeria toxoid or as a 10-year booster. Certain adults should get Tdap with an interval of 2 years or less following their previous Td dose if they are (1) a parent or caregiver of a child younger than age 12 months, (2) a healthcare worker having direct patient contact, or (3) at risk for pertussis due to increased pertussis activity or during outbreaks

H. influenzae type b
2 months
4 months
6 months
12–15 months

Polio
2 months
4 months
15 months
4–6 years

Measles, mumps, rubella
12–15 months
4–6 years or 11–12 years
Two doses are needed for an adult – no sooner than 4 weeks apart

Varicella
15 months
11–12 years
Two doses are needed if an adult – 4 to 8 weeks apart

Human papillomavirus
9–26 years
Gardasil is a 3-dose series with #2 dose given 2 months after first dose and #3 dose given 4 months after the #2 dose

Meningococcal
Give MCV4 to those at risk (college freshmen living in dorms, etc.)
One dose and repeat every 5 years if risk of disease continues

VACUUM EXTRACTION

Need for CAUTION	
FDA reported how many deaths in @ 4 years?	12
How many serious injuries did the FDA report?	9
This calculates to 1 event per	45 455
The incidence of severe fetal injury or death from vacuum extraction ranges from 0.1–3 cases per 1000 cases	
What is the diameter of the soft cups?	65 mm
How many centimeters should the cup be placed in front of the posterior fontanelle?	3 cm
The VE pressure should not exceed what?	580 mmHg or 10 lb/in^2
The Green Zone pressures are	35–45 cmH_2O 350–450 mmHg

Vacuum extraction requires less general and regional anesthesia than do forceps deliveries because it is not applied against the vaginal walls

The center of the cup should be placed over the sagittal suture, 3 cm in front of the posterior fontanelle, no maternal tissue should be trapped along the edge and underneath the cup

Coordinate pulls with maternal expulsive efforts. Do not exceed limits.

No consensus on pulls … some say limit traction pulls to → 3–5?

A vacuum procedure should not exceed 30 min, with a total suction time of less than 10 minutes

A vacuum should not be used to deliver fetuses under 36 weeks' gestation

Vacuum + forceps criteria

- Presented with an OP presentation, would you rotate?
- There are definitely some risks. It would depend on the experience of the practitioner. One would also need to evaluate if the fetal head was large, not floating and individualize each particular case

VACUUM EXTRACTOR

Types

Malmstrom metal cup with diameter 40–60 mm

Soft cups (polymeric silicone). Introduced in 1973 65 mm

VE cups indicated for outlet and low OA < 45° extractions:

Soft cups (silicone or plastic)

Kiwi ProCup and Tender Touch cups

Standard Mityvac and Soft Touch cups

Silc, Gentle Vac, and Secure cups

Silastic, Reusable, and Vac-U-Nate cups

Rigid "anterior" cups (plastic or metal)

Kiwi OmniCup

M-Style Mityvac cup

Flex cup

Malmstrom, Bird, and O'Neil anterior cups

VE cups indicated for low OA > 45°, OP, OT extractions

Rigid "posterior" cups (plastic or metal)

Kiwi OmniCup

M-Select Mityvac cup (i.e. One-piece Mystic MitySoft Bell Cup)

Bird and O'Neil posterior cups

Advantage of vacuum over the use of forceps

Vacuum extraction requires less general and regional anesthesia than do forceps deliveries because it is not applied against the vaginal walls

Technique

Place center of cup over sagittal suture 3 cm in front of posterior fontanelle. Check to make sure NO maternal tissue trapped under along edge. Coordinate pulls with maternal expulsive efforts

A 5-cm cup with 600 mmHg of vacuum provides 16 kg (35 lb) of attachment force

Green Zone 35–45 cmH_2O or 350–450 mmHg

VE pressure – NEVER EXCEED 580 mmHg or 10 lb/in_2

Pearls

How many pulls can one perform? NO CONSENSUS, but some have recommended only 3–6

A VE procedure should not exceed 30 min, with a total suction time of less than 10 min

Incidence of severe fetal injury or death per 1000 VE procedures is in the range of 0.1–3 cases

A vacuum should not be used to deliver fetuses under 36 weeks' gestation

It is suggested that all infants undergoing VE have an umbilical cord hematocrit to monitor for changes that could signify a subgaleal bleed

Shoulder dystocia is the most prominent risk factor for brachial plexus palsy in the setting of vacuum extraction 1.1%

In the U.S., VE is used 2–3 times more often than forceps for operative delivery

VAGINAL ANATOMY

(1) Longitudinal vaginal septum – "double-barrel vagina"
Failure of fusion of lower Müllerian ducts
Difficulty using tampons, dyspareunia, possible infertility of repeated ab if outside didelphic uterus
EXCISE SEPTUM. IVP to rule out other anomalies

(2) Transverse vaginal septum – incidence 1/2100–72 000
Etiology unknown. Incomplete fusion *between* Müllerian duct and urogenital sinus
Most (what %) occur at junction of upper 1/3 and lower 2/3 of vagina? 46%
Hydrocolpos or HEMOCOLPOS (> puberty). Complete – cyclic pain with no menses
Partial – dyspareunia or routine exam
I&D then delay surgery 6–8 weeks. Usually not associated with urological or other anomalies

VAGINAL AGENESIS

Primary amenorrhea and absence of Müllerian structures

Complete Müllerian agenesis	*Complete androgen insensitivity*
(M-R-K-H syndrome)	
46XX	46XY
Normal ovaries	Often have undescended testes
Defect is Müllerian	Defect is in androgen receptor
50% renal and vertebral defects	Scant pubic and axillary hair is noted
IVP to check for R&V defects	Check karyotype prior to gonadectomy
FSH, LH, testosterone – normal	Testosterone (same or elevated more than in normal males)
	LH is elevated secondary to resistance of hypothalamic–pituitary to androgen

Remember

Complete Müllerian agenesis
Mayer–Rokitansky–Kustner–Hauser syndrome 46XX
Normal ovaries
Defect also associated with renal or vertebral defects in 50%
Check vertebra and renal system. Do an IVP
These labs are all normal – FSH, LH, testosterone
Complete androgen insensitivity 46XY
Often have undescended testes
Defect in androgen receptor. The axillary and pubic hair is scant
Prior to gonadectomy, check this karotype
These labs are elevated – testosterone
Increased due to resistance of hyp-pit to androgen LH

VAGINAL BIRTH AFTER C-SECTION

Incidence of uterine rupture

After one C-section < 1%
After one low transverse C-section 0.2–1.5%
After two C-sections 2–5%
Has been reported as low as 1–1.3%
Incidence of rupture with low transverse PRIOR to labor rare
After a classical or T-shaped incision CD (Cesarean delivery) 4–9%
Has been reported as high as 12%
Incidence of rupture with classical or vertical PRIOR to labor 33%
After an unknown scar ?

	After rupture of the lower uterine segment	6%
	After rupture of upper uterine segment	32%
	Spontaneous rupture of an UNSCARRED uterus	1/15 000
	TOL (trial of labor) success rate should be	60–80%
	#VBACs/# pts with prior CDs × 100	VBAC rate
	#VBACs/# pts who had TOL after CD × 100	VBAC success rate
	Overall success rate is	75%
	Success rate with history of CD for breech	90%
	Success rate with history of CD for fetal distress	80%
	Success rate with history of CD for dystocia	70%
	Previous vaginal delivery lowered the uterine rupture rate	60%
Definition	Rupture – separation of entire incision, ROM, fetus out, increased bleeding Dehiscence – separation of part of the incision, intact membranes, fetus in, no or minimal bleeding	
Symptoms of rupture include	Decreased FHR (severe variable decelerations) are the most common early symptom seen in what % of patients?	80%
	Loss of station, decreased uterine activity and shock are symptoms	
	Acute abdominal pain is seen in	10%
Prognosis	Fetal mortality rate is	50–75%
	Maternal mortality rate is	44%
	The less time between deliveries, the more likely is uterine rupture Personally review the prior operative note before attempting a trial of labor Srinivas demonstrated that significant clinical variables (prelabor and labor) cannot reliably predict VBAC failure (Srinivas SK, Stamilio DM, Stevens EJ, *et al*. Predicting failure of a vaginal birth attempt after Cesarean delivery. *Obstet Gynecol* 2007; 109:800–5)	
Treatment	Prompt diagnosis, STAT SURGERY, blood and antibiotic therapy	
VBAC criteria	Females deliver vaginally after previous LTCS in the USA @	27%
	Literature does not set policy one way or another. Recently VBAC was discouraged after it had been encouraged – it waffles back and forth depending on rise in C-section rate Some key points; (1) Selection criteria useful for identifying candidates for VBAC include: a limit of 1 prior low-transverse Cesarean, clinically adequate pelvis, no other uterine scars or previous rupture, and no contraindications (2) Offer VBAC only if obstetric care and anesthesiology are available throughout active labor, in case emergency Cesarean is necessary (3) Single-layer uterine closure may increase the risk of rupture during subsequent labors (4) Epidural anesthesia is safe for women undergoing a trial of labor	
Candidates	1 prior C-section, adequate pelvis, no other uterine scars, and STAT available staff	
Contraindications	Vertical or T-shaped classical or fundal incisions, contracted pelvis, medical complications, previous uterine rupture, contraindications to vaginal birth, and/or inability to do STAT C-section	

VAGINAL CREATION

Neovagina	*Split-thickness skin graft* Easiest, mold Cong abs of vagina, status post-vaginectomy or stenosis after radiation

Myocutaneous graft
Use after exenteration

Gracilis flaps
Pressure sensitivity excellent. Increase skin loss
What % of these flaps are lost due to vascular compromise? 10–20%

Vulvobulbocavernosus cutaneous graft
Tactile sensation increased due to neovagina tissue enervated by pudendal nerve

VAGINITIS (*See also* Vulvovaginitis)

	Normal vaginal pH	3.8–4.2
Yeast	Negative whiff test and pH	< 4.5
BV	Positive whiff test and pH	> 4.7

VAGINAL INTRAEPITHELIAL NEOPLASIA (VAIN)

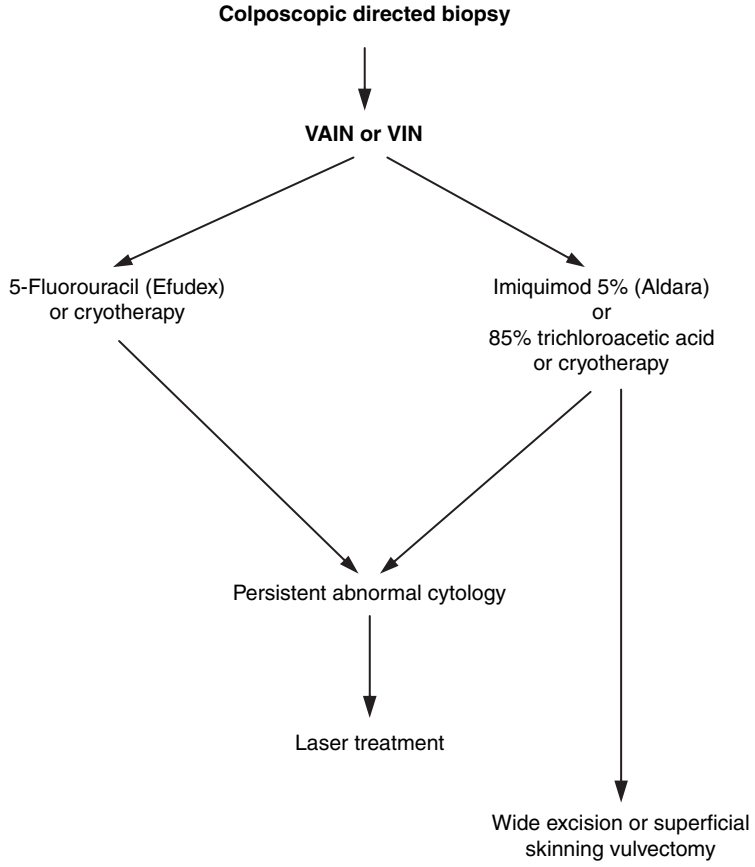
Two factors that predict the recurrence of VAIN are:
(1) Multifocality
(2) Method of treatment

Risk of recurrence according to treatment:
- Risk of recurrence when treatment is with 5-FU is 59%
- Risk of recurrence when treatment is with CO_2 laser is 38%
- Risk of recurrence when treatment is with partial vaginectomy 0%
- Interestingly, age, smoking, HRT use, grade of VAIN, location of VAIN and association with either CIN or VIN were not predictive of recurrence

VAIN is associated with CIN and VIN
5-FU is no longer considered a good treatment for VAIN and may not even have any indication for the use of 5-FU in lower genital tract
Imiquimod 5% (Aldara®) might be an option prior to excision
Most VAIN occurs in the upper vagina

Vaginal or vulvar intraepithelial neoplasia (VAIN/VIN)

Vaginal or vulvar intraepithelial neoplasia, dysplasia of the vulvovagina, and papillomatosis are often noted on vaginal/vulvar cytology prior to and after hysterectomies. Although many sources are now recommending discontinuance of Pap smears after hysterectomy, this is empirically continued at least once every 3 years secondary to the continued findings of this vaginal pathology in our area



Informed consent and instructions for 5-fluorouracil cream

You have been given a prescription for 5-fluorouracil (5-FU, Efudex®) cream for the treatment of lesions on your vagina and/or cervix. 5-FU has been used for more than 25 years in treatment of various lesions or growths of the skin. However, this medication has not been approved by the Food and Drug Administration (FDA) for use in treating warts or other precancerous growths on the genitals. A number of studies have proven the effectiveness of this drug in treating warts and "dysplasias" or abnormal growths from the wart virus. One of the major concerns using the drug is its effect on pregnancy. It is therefore vital that you are not pregnant while you are using 5-FU cream because its safety for the developing fetus is unknown. You should use close to perfect birth control (birth control pills, sterilization, abstinence, IUD or condoms and diaphragm together)

Side-effects of this medication are mainly vaginal or vulvar irritation or burning which may be significant enough to stop treatment temporarily. If you notice this happening, please call the office for further instructions.

Instructions for vaginal use

(1) Use only the specially marked applicator that has been given to you or the prefilled applicators
(2) If you do not have the prefilled applicators, please fill your applicator to the 2.0-g mark. Double check this for the correct level
(3) Put the applicator with the cream high into your vagina and push the plunger in
(4) Take the applicator apart and wash with warm soapy water or throw away the prefilled applicator container
(5) Go to bed
(6) In the morning, get into a tub of warm water and wash out the vagina as well as you can with your fingers
(7) You should not have intercourse for 24 h after each cream dose
(8) You should repeat this procedure using one dose every week for a total of 10 doses or 10 weeks

Instruction for vulvar or external use

(1) Dab a small amount (size of pea or bean) of cream onto the entire vulva while looking into a mirror. This would be best done at bedtime. Rub the white cream entirely into the vulvar skin until the cream disappears. Leave no patches of cream on the skin. Check again with a mirror
(2) Repeat this procedure two times a week for 10 weeks
(3) The morning after the treatment, sit in a tub of warm water and wash off any remaining cream

After either the vaginal or vulvar use, you should make an appointment for a repeat colposcopy 6–8 weeks after completing your last dose. If you have any questions, please call.

INFORMED CONSENT

I understand that the medication 5-FU has been prescribed for me to treat condyloma (warts) or skin changes believed to be from the wart virus. I understand that the FDA has not approved this medication for use. I also understand that it is unsafe to become pregnant while using this medication as its effects on pregnancy are unknown and that it is my responsibility to avoid pregnancy. I have had the opportunity to ask any questions I might have regarding this medication.

_____

PATIENT'S SIGNATURE

_____

DATE

_____

PROVIDER/PRACTITIONER

VAGINAL CIS

VAIN – most commonly in upper 1/3 of vagina. Pap – colpo with biopsy to diagnose

Symptoms

Asymptomatic, occasionally postcoital bleeding
Risks: increased with HPV, radiation, immunosuppressive therapy and previous history of CIN/cervical cancer. Most often multifocal so get Paps from multiple sites

Treatment

(1) Local excision of small lesions
(2) 5-FU and/or laser therapy for larger size or multiple lesions
(3) Upper colpectomy or total vaginectomy

VARICELLA-ZOSTER VIRUS

What % of patients are immune to varicella virus? 90%
Primary infection is chickenpox with maculopapular/vesicular rash with symptoms + fever × 3–5 days
No evidence that zoster increases frequency of congenital abnormalities of varicella

Complications of chickenpox

Maternal
Most common is secondary skin infections (streptococcal and staphylococcal)
Most serious is pneumonia that develops in 20%
Varicella pneumonia has what % mortality? 35%

Fetal
The risk of congenital varicella is increased during 13–20 weeks
There is NO risk after what week gestation? 20 weeks
There is an increased risk if fetus exposed to virus just prior to or during delivery. VZIG to be given @5 days prior to delivery or 2 days postpartum

Treatment

Acyclovir 10 mg/kg IV q. 8 h, O_2, ventilation p.r.n.
VZIG @ 96 h after exposure in dose of 125 u per 10 kg IM

Prevent with

Varivax 0.5 ml – recommended for ages 12 months thru 12 years
Suspected adults and adolescents 2 doses 6 weeks apart
CONTRAINDICATED IN PREGNANCY

VARICOCELES

Present in what % of postpubertal males (either unilateral or bilateral)? 15%

VASA PREVIA

Incidence 1/2000–1/3000

Diagnosis

ELUSIVE DIAGNOSIS
Palpable abnormalities and color flow Doppler?
Must have a high degree of clinical suspicion. Sometimes one might palpate abnormalities in the fetal membranes at the level of the cervix

Definition

Velementous insertion when some vessels cross os
Presents usually with SUDDEN VAGINAL BLEEDING associated with unresponsive fetal bradycardia

Treatment

STAT C-section
Prior to amniotomy – stain blood with Wright's stain for nucleated fetal RBCs
Not always time for APT test
Fetal mortality rate 70–75%

VASECTOMY

What % of men develop sperm antibodies in serum after a vasectomy?	50%

VENOUS THROMBOEMBOLISM

Incidence	General population	0.1–0.3%
	Affects this % of pregnancies	0.05–0.3%
	What % of untreated DVT will develop pulmonary embolism?	24%
	The mortality for pulmonary embolism is	15%
	Treating DVT will reduce the incidence of the occurrence of pulmonary embolism to	4.5%
	The reduced mortality of pulmonary embolism will be	1%
Symptoms of VTE	Tachypnea	90%
	Dyspnea	> 80%
	Pleuritic chest pain	< 70%
	Apprehension	60%
	Cough	50%
Diagnosis of VTE	Ascending venography – most accurate test for DVT	5 rads
	Doppler US and impedance plethysmography	
	PaO_2 usually associated with O_2	< 85 mmHg
	EKG – tachycardia most commonly seen	
	What wave inversion is seen only in massive PE?	T
	Perfusion and ventilation lung scanning – most useful for suspected PE	
	Pulmonary angiography – gold standard	
Treatment	Heparin IV 5–10 days then SC q. 12 h during pregnancy	
	PTT to be kept	1.5–2.5 × out
	or INR	2–3 x
	How much PROTAMINE will neutralize 100 u of heparin?	1 mg

VERSION

See External cephalic version

VIOLENCE

What % of all lone-offender violence against women was perpetrated by those who knew victim?	75%
What % of men who abuse partners also abuse children?	50%
Violence usually begins or escalates during pregnancy	
Race or ethnicity are not associated with an increased risk	
Domestic violence Hot Line is	1 - 800 - 799 - SAFE

VITAMIN THERAPY

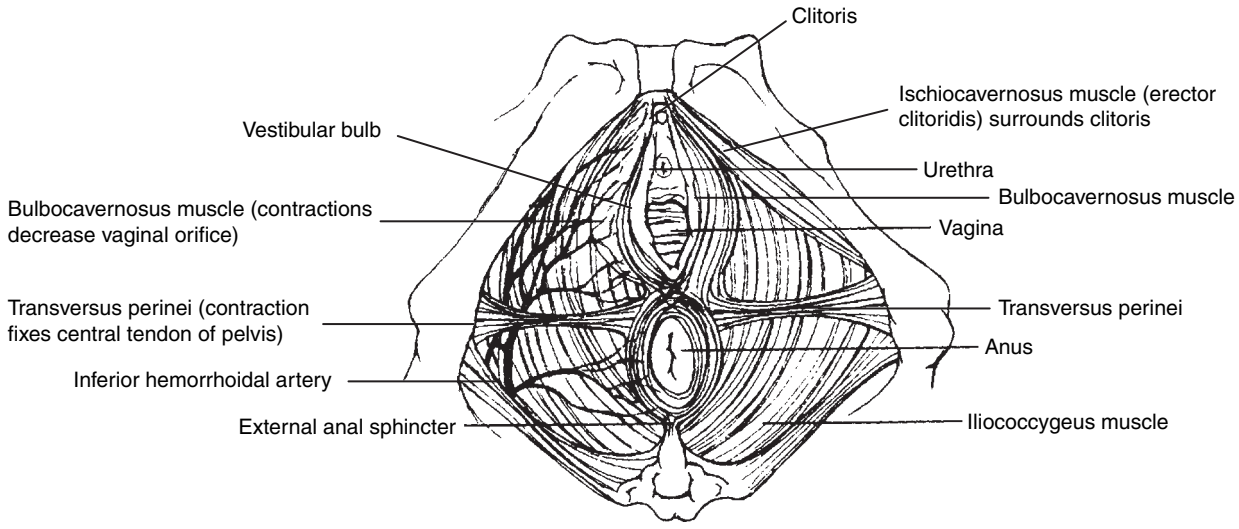
Vitamin A

Minimum human teratogenic dose of Vit A is probably at least this IU daily	25 000–50 000
Risk begins with as little as how much per day?	10 000 IU
Risks include neural tube defects, cleft lip and palate defects	
RDA of Vit A for non-pregnant females and not increased during pregnancy is	800 retinol eq/day
RDA for pregnancy and lactation is	2700 IU/day
A balanced diet usually has how much Vit A per day?	7000–8000 IU/day
Most women in the U.S.A. have adequate stores of Vit A in their livers	
Supplementation of how much should be considered maximum intake (p.r.n.)	5000 IU
Balanced diet provides how much Vit A?	7000–8000 IU

Vitamin B_6	Decreases homocysteine which is a significant risk factor in CHD, MI, stroke and VTE	
Vitamin B_{12}	Vegetarians need more of this. Also decreases homocysteine	
Vitamin C		
Vitamin D	May need extra for women with limited sun exposure	
	RDA for pregnancy and lactation is	400 IU
	Risks include hypercalcemia (fatigue, depression, confusion, anorexia, nausea and vomiting)	
	Risk of toxicity usually after chronic ingestion of over	50 000 IU
Vitamin E	Decreased risk of CHD and Alzheimer's	
Folate	Need this much periconceptional	0.4 mg/day
	Need to increase with multiple gestations to	1 mg/day
	Need to increase with epileptics, hemoglobinopathies and previous history of NTD to	4 mg/day
Ferrous sulfate	Give how much t.i.d.?	325 mg
	How much iron is in a 325 mg tablet?	60 mg
	Only this % is absorbed from the tablet	10–20%

VULVAR ANATOMY

Ischiocavernosus m., bulbocavernosus m. and transversus perinei m.



VULVAR ATYPIA

	Squamous cell hyperplasia	
	Lichen sclerosus	
Intraepithelial neoplasia	Mild dysplasia	VIN I
	Moderate dysplasia	VIN II
	Severe dysplasia	VIN III
Others	Paget's disease	
	Melanoma *in situ*	Level I

VULVAR HEMATOMA

Signs are subacute volume loss or vulvar pain – can be severe
Blood loss is limited by:
(1) Colle's fascia
(2) Anal fascia
(3) Urogenital diaphragm
If small, treat expectantly
If severe – I&D and obliterate cavity

If no bleeding sites, pack cavity for	12–24 h
Blood replacement p.r.n., catheterize for how many hours?	24–36 h
Pressure dressing for how many hours?	12 h

VULVAR INTRAEPITHELIAL NEOPLASIA (VIN)

	Average age for VIN	40
	Average age for vulvar cancer	70
Signs and symptoms	Asymptomatic in what % of cases?	50%
	Pruritis is predominant symptom.	
	VIN lesions are hyper- or pseudopigmented in about	30%of cases
Etiology	HPV is associated with VIN in what % of cases?	80–90%
Diagnosis	(1) History and physical (inspection)	
	(2) Colposcopy	
	Multifocal more common in premenopausal women	
	Unifocal more common in postmenopausal women	
	(3) Biopsy with 4–6 mm Keyes Punch Derm	
	Use Gelfoam × 24 h for bleeding	
	• Biopsy is essential for correct diagnosis of VIN	
	What % of females evaluated for VIN had vulvar cancer?	20%
	VIN I	Mild dysplasia
	VIN II	Moderate dysplasia
	VIN III	Severe dysplasia
	VIN IV	Carcinoma *in situ*
Treatment	(1) Wide local excision with disease-free border of	≥ 5 mm
	5-FU has a failure rate in VIN treatment of	50%
	Multifocal lesions – vulvectomy + skin graft. Laser, cryo, cautery increase ulcer formation	
	(2) Laser is treatment of choice for multifocal disease	
	Main complaint after laser therapy is PAIN	
	Post-laser therapy care includes – topical steroids, Sitz baths, local anesthesia and pain meds especially for 3–4 days postop	
	Vaporization of the vulva should be limited to the following depths:	
	(a) Labia minora (hair-free)	0.5 mm
	(b) Labia majora (hair-containing)	1.5–2 mm
	If CIS is present, SIMPLE VULVECTOMY (20% have invasion)	
	If cancer present, RADICAL VULVECTOMY AND BILAT INGUINAL LYMPHADENECTOMY	

Lymph node dissection is the single most important factor in decreasing mortality from early recurrence of vulvar cancer

VULVAR MASS (OR LABIAL MASS)

Differential

Bartholin's cyst
Papilloma
Vulvar varicosity
Testes

Leiomyoma
Gartner's duct cyst
Lymphogranuloma venereum
Sebaceous cysts

VULVAR ULCER

Differential

(1) Herpes
3–7 days' incubation, painful, vesicle formation
(2) LGV
1–4 days' incubation, painless, superficial tender lymph nodes
(3) Granuloma inguinale
8–10 weeks' incubation, painless, red base with rolled elevated edge
(4) Trauma
(5) Syphilis
10–60 days' incubation, painless, indurated with raised edges, solitary or "kissing" lesions
(6) Chancroid
2–6 days' incubation, painful, tender, irregular, undermined lesions
Red "halo" with bubo – inguinal adenopathy – chronic drainage
(7) Crohn's disease
(8) Scabies

VULVAR VESTIBULITIS

Severe pain and dyspareunia. Little known. Culture any raw areas
Hallmark of vestibulitis: Severe pain on touch, with tenderness localized with the vestibule in a horseshoe pattern

Etiology

Questionable. Possibly associated with:

(1) *Candida albicans*
(2) Human papillomavirus
(3) Neurologic
(4) Psychologic (marital conflict, history of sexual abuse, somatic)

Rule out

(5) Herpes vulvitis
(6) Contact dermatitis
(7) Focal infection
(8) Vulvar dystrophy

Many patients have depression. Rx with antidepressants
Tricyclic antidepressants (amitriptyline HCl)
Do NOT use benzodiazapines!

Symptoms

Exquisite sensitivity to touch (especially laterally from hymenal ring to HART line of labia min)
Burning pain/pressure for how many months? 3 months or >
Application of this causes exquisite pain 3–5% acetic acid
Bartholin's glands often are dilated. Digital exam may be associated with levator ani spasm

Treatment

Triamcinolone 0.1% then reduce to hydrocortisone 1%. Topical lidocaine 2% gel may be used prior to intercourse. Kegel pelvic floor exercises, biofeedback or behavioral therapy. Injectable interferon
Many treatments but BEST cure in this % is surgical excision in 60–80%
If excision is to depth of 2 mm
Vulvar vestibulitis is associated with a decreased incidence of sexual activity in what % of cases? > 80%
Patients most likely to benefit from vestibulectomy are those patients who are totally unable to have intercourse (Schneider D, Yaron M, Bukovsky I, *et al*. Outcome of surgical treatment for superficial dyspareunia from vulvar vestibulitis. *J Reprod Med* 2001; 46:227–31)
Electromyographic biofeedback of pelvic floor musculature may be an effective treatment (McKay E, Kaufman RH, Doctor U, *et al.* Treating vulvar vestibulitis with electromyographic biofeedback of pelvic floor musculature. *J Reprod Med* 2001; 46:337–42)

VULVODYNIA

Chronic vulvar discomfort, especially that characterized by the patient's complaint of burning, stinging, irritation, or rawness
Vulvodynia pain may never subside completely

Classification of vulvodynia (vulvar pain)

Dermatologic:
(1) Contact dermatitis
(2) Erosive lichen planus
(3) Rare dermatoses (Behçet's, pemphigus, cicatricial pemphigoid)
Atrophic vulvovaginitis

Chronic infections:
(1) Yeast (*Candida glabrata*)
(2) HPV
(3) Herpes genitalis

Neoplasia:
(1) VIN
(2) Cancer of the vulva

Vestibulitis
Others
Rule out contact irritants or sensitizing agents of the vulvar skin

Examples of irritants

Laundry detergents, fabric softeners and dryer sheets
Body soap
Pads and panty liners (especially if scented)
Perfumes
Synthetic underwear and pantyhose
Povidone-iodine and other surgical skin cleansers
Agents used for treatment of warts (5-FU, podophyllin and Aldara)
Deodorants, douches, moistened wipes, powders
Washcloths
Urinary or fecal incontinence
Vaginal discharge and menstrual flow
Semen
Topical medications in the form of creams or gels (ETOH/glycol, etc.)
Lubricants and lubricated condoms
Spermicides

Examples of sensitizers

Topical antibiotics (neomycin)
Spermicides
Dyes (found in clothing)
Rubber (exam gloves/condoms)
Nickel (pierced jewelry)
Corticosteroids
Topical anesthetics (benzocaine)
Fragrances
Preservatives in topical meds (parabens, formaldehyde)
Emollients in topical meds (lanolin)

General skin care of vulvodynia

(1) Avoid contact irritants and sensitizers as much as possible (see above)
(2) Use laundry detergent free of perfumes and enzymes
(3) Whenever possible use medications in the form of ointments rather than creams or gels
(4) Use only water and the hand to wash the vulva
(5) Wear cotton underwear during the day and do not wear any underwear in bed at night
(6) Use vegetable oil as lubricant for intercourse
(7) Use non-lubricated condoms with vegetable oil
(8) Apply a bland ointment free of fragrances regularly as an occlusive skin protectant (zinc oxide or A+D)
(9) Soak with baking soda for 15 min daily (4–5 tablespoons of baking soda in bathtub of lukewarm water)

Diagnosis and management of various forms of vulvodynia

(1) Contact dermatitis – erythema and edema – triamcinolone 0.1% b.i.d. × 1 week, daily x second week, then 3 × weekly for 2–4 weeks then use hydrocortisone 1% cream for residual or recurrences

If no improvement – consider allergic contact dermatitis → refer for patch testing

(2) Erosive lichen planus
Dyspareunia worsens as the disease progresses. Erosions in vagina. Adhesions of labia minora. Micro demonstrates immature cells (basal and parabasal epithelial cells) and many white blood cells. Tacrolimus 0.1% ointment for severe cases. Mild steroid treatment for less severe cases

(3) Atrophic vulvovaginitis
Discharge is brownish with spots of blood. Erythema and erosion. Skin may appear thin. Estrogen therapy and sometimes low potent steroids

(4) HPV (human papillomavirus)
Acetowhite changes along post fourchette and Hart's line of inner labia minora. Imiquimod (Aldara) 3 × per week for at least 6 weeks

(5) Herpes genitalis
Isolate and identify by culture. Acyclovir, valacyclovir or famciclovir

(6) VIN I–II – biopsy. Treat same as HPV
VIN III → CIS – biopsy. Wide local excision. Vulvectomy as last resort

(7) Cancer
Usually does not produce pain unless fissuring of lesions occur. Radical vulvectomy with bilateral inguinal lymphadenectomy

(8) Vestibulitis – etiology unclear. Many. *See Vulvar* vestibulitis

(9) Others
(a) Sjögren's syndrome – autoimmune disease causing dryness and burning of the vagina, mouth and eyes. Amitriptyline 10–25 mg at night. Topical 0.25% menthol in aquaphilic ointment
(b) Gabapentin 100–3000 mg daily 64% patients had 80% relief

VULVOVAGINITIS

RECURRENT

Normal vaginal pH is	3.2–4.2
Physiologic pH is	< 4.5
Suspect BV or Trich if pH	> 4.7
C. albicans is culprit in RVVC @ what %?	90%

Management

(1) Clotrimazole, butoconazole, miconazole, nystatin, terconazole, tioconazole (Monistat®, Femstat®, Terazol®) for 14 days, then weekly × 6 weeks

(2) Ketoconazole (Nizoral®) 400 mg daily for 14 days, then 100 mg daily × 6 months
Watch hepatic enzymes, GI distress, rash, headache. No Seldane®

(3) Diflucan 150 mg, then 100 mg weekly × 25 weeks

Symptomatology

Vulvar/vaginal burning, discharge

Evaluate vagina

Inspect external genitalia (r/o excoriations, blisters, ulcerations erythema, edema, atrophy)

Examine vaginal discharge – gross and microscopic
pH level: > 4.5 (bacterial vaginosis OR trichomoniasis)
< 4.5 (physiologic OR uncomplicated candidal vaginitis)
Whiff test: + fishy odor = amines = anaerobic bacteria
(10% KOH) – fishy odor = normal flora
Rule out allergic/chemical irritation – careful history

Candidal vaginitis

Part of normal vaginal flora
Self-diagnosis, telephone nurse diagnosis, and even clinician diagnostic workups are often inaccurate or incomplete

Risk factors:
Recent Abx, diabetes (2 h GTT – 75 g), immunosuppression (HIV)
Diagnosis:
History – pruritus, burning (worsened with urination/sexual activity)
Physical exam – non-malodorous, thick, white "cottage cheese" discharge; vagina hyperemic/edematous

Diagnostic tests – pH < 4.5 (normal)
- microscopic – hyphal forms/budding yeast
- a woman with complicated candidiasis should have a yeast culture to find out what species of yeast is causing her infection

Treatment:
Topical (first line) – terconazole, butoconazole, clotrimazole, miconazole, tioconazole
Oral (second line) – fluconazole 150 mg (not in pregnancy)

Resistant vulvovaginal candidiasis (RVVC)
(1) Fluconazole 100 mg orally every week × 6 months
(2) Boric acid capsules 600 mg per vagina q.d. × 14 days

Treatment for uncomplicated candidiasis

Agent	*Brand name*	*Dosage*
Butoconazole 2% cream	Femstat*	5 g intravaginally × 3 days
Clotrimazole 1% cream	Gyne-Lotrimin*	5 g intravaginally × 7–14 days
	Mycelex-7	5 g intravaginally × 7–14 days
Clotrimazole vaginal tabs	Gyne-Lotrimin vaginal inserts*	One 100 mg insert × 7 days
	Mycelex-7 vaginal inserts*	One 100 mg insert × 7 days
	Mycelex-G vaginal tablets	One 500 mg tablet
Fluconazole oral tablets	Diflucan tablets	One 150 mg tablet
Miconazole 2% cream	Monistat 7*	5 g intravaginally for 7 days
Miconazole suppositories	Monistat 7*	One 100 mg suppository × 7 days
	Monistat 3	One 200 mg suppository × 3 days
Terconazole 0.4% cream	Terazol 7	5 g intravaginally × 7 days
Terconazole 0.8% cream	Terazol 3	5 g intravaginally × 3 days
Terconazole suppositories	Terazol 3	One 80 mg suppository × 3 days
Tioconazole 6.5% vaginal	Vagistat-1	5 g intravaginally once

*Available without prescription

Bacterial vaginosis

History: pruritus burning, malodorous discharge (worsened during menses/after intercourse)
Physical exam: discharge, malodorous, thin, grey, homogenous
Diagnostic tests (traditionally diagnosed when 3 of 4 Amsel's criteria are met. These criteria include:
(1) pH > 4.5
(2) +Whiff test (3 out of 4)
(3) Clue cells at least equal to 20% of epithelial cells
(4) White or gray homogenous discharge
Treatment:
Topical – 0.75% metronidazole (Vandazole or MetroGel) gel, intravaginal × 5 days (not for ophthalmic, dermal, or oral use!)
– 2% clindamycin cream, intravaginally q.d. × 7 days

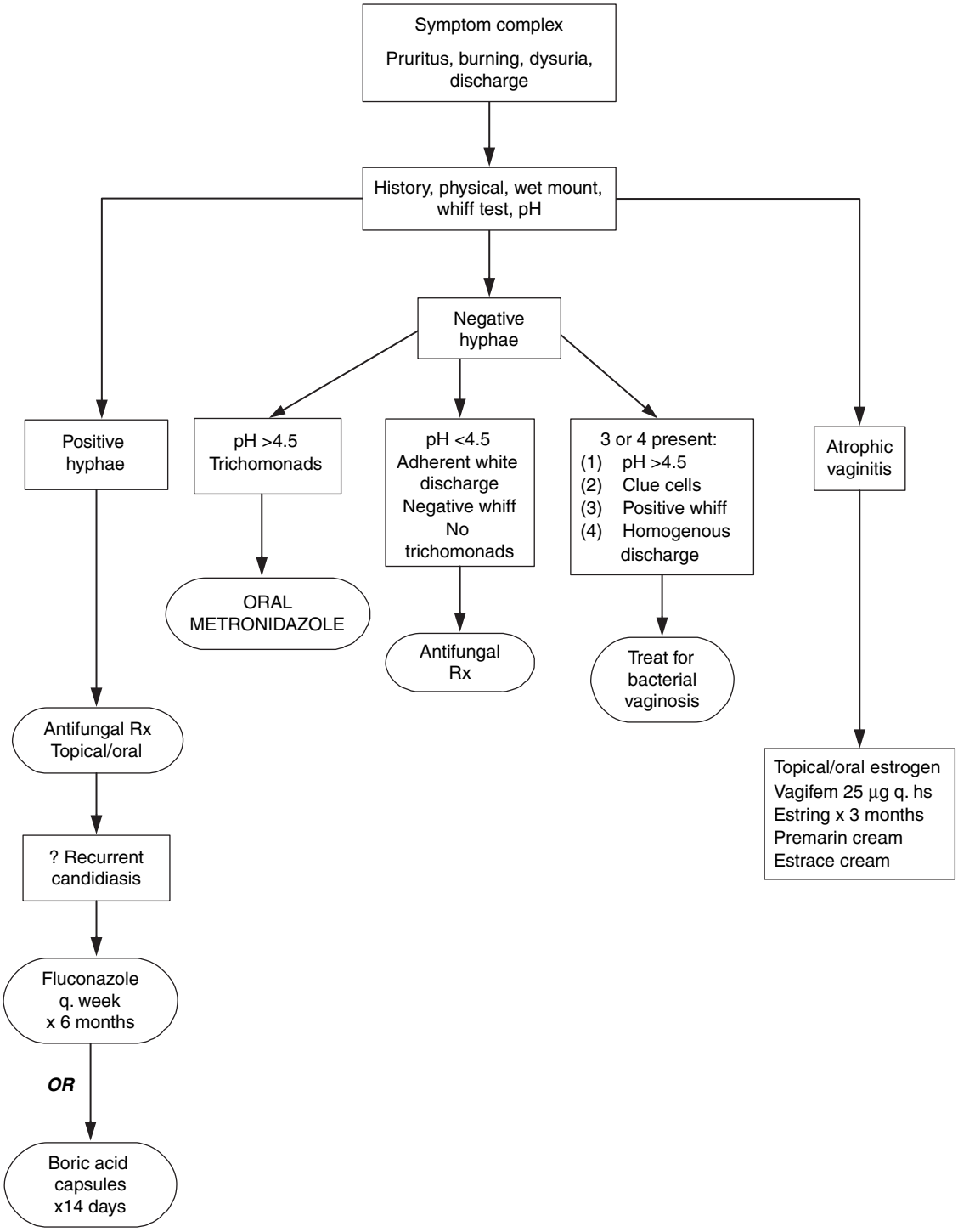
Oral – metronidazole 500 mg b.i.d. × 7 days (or 250 mg t.i.d. × 7 days)
- clindamycin 300 mg b.i.d. × 7 days
- twice weekly intravaginal metronidazole greatly reduces relapse

Treatment for bacterial vaginosis

Agent	*Brand name*	*Dosage*
Metronidazole oral tablets	Flagyl	One 500 mg tab twice daily for 7 days ***or***
Metronidazole 0.75% gel	Metrogel-Vaginal	5 g intravaginally twice daily × 5 days* or
Clindamycin phosphate 2% cream	Clindesse	1 prefilled applicator vaginally one time or
Clindamycin 2% cream	Cleocin	5 g intravaginally × 7 days or
Clindamycin oral tablets	Cleocin HCl capsules	Two 150 mg capsules twice daily × 7 days

*Some recommend that Metrogel can be used once daily at night for 5 days, especially for milder infections

Trichomonas vaginalis	*History*: discharge (copious, yellow-green, homogenous, malodorous), vulvovaginal irritation, dysuria *Physical exam*: frothy, malodorous discharge, "strawberry cervix" *Diagnostic tests* (If purulent, requires exclusion of cervicitis, PID, estrogen deficiency plus finding of elevated pH and inflammatory cells): (1) pH > 4.5 (2) wet mount (mobile, flagellated organisms) (3) trichomonads on Pap *Treatment*: Oral metronidazole – 2 g p.o. × 1 dose or 500 mg b.i.d. × 7 days
Resistant trichomoniasis	Combination oral/vaginal metronidazole Culture for resistant strains Confirm treatment of partner IV metronidazole (requires hospitalization)
Atrophic vaginitis	Thinning of vaginal epithelium, loss of rugae, friable *Treatment:* Oral – 0.625 mg conjugated estrogens q. day. Topical – estrogen cream 2–4 g q.d. × 2 weeks and then q.o.d. × 2 weeks. Maintenance: estrogen 1–3 × week
GAS *(Group A streptococc*al *purulent vaginitis)*	Young mothers Immediate family history of GAS pharyngitis/proctitis Children (prepubertal): – Vulvitis – Proctis Usually misdiagnosed as *Candida* Clue – Lack of response to antimycotics – Saline microscopy – increased PMNs, cocci, and increased pH Diagnosis – culture Treatment – penicillin Noninfectious forms of purulent vaginitis include DIV and erosive lichen planus
DIV *(desquamative inflammatory vaginitis)*	– Chronic inflammatory process that involves the vagina but not the vulva • Unresponsive to estrogen therapy alone • Typically seen in perimenopausal Caucasian women but very rare in African-American women and other minorities • May be an autoimmune disease Symptoms – purulent discharge, irritation, soreness, burning, and pain *Diagnosis* – high pH, increase in PMNs and parabasal cells, absence of lactobacilli, and an overgrowth of other organisms Rule out *T. vaginalis*, cervicitis, endometritis, atrophic vaginits, ELP and pemphigus syndromes *Treatment* – 10% hydrocortisone cream or 2% clindamycin cream or in treatment-resistant cases, with both agents
ELP *(erosive lichen planus*)	• May affect the mouth and throat, vagina, vulva, and vestibule • *Diagnosis* – may cause gingival erythema and erosion or white reticulate lesions • If ELP of skin involved, diagnosis is easy • Complications – can frequently involve fibrosis and synechia that may become lifelong causing shortening or obliteration of the vagina or lead to neoplasia • *Treatment* – high-dose intravaginal steroids sometimes with clindamycin cream 2% and/or topical tacrolimus gel



WEIGHT

BMI =	Wt (kg) / ht squared (m^2)
Normal weight is BMI between	19–25 kg/m^2
Overweight is BMI between	25.1–29.9 kg/m^2
Obesity is BMI over	30 kg/m^2
Rapid weight loss of how much during the first week can cause gallbladder dysfunction?	2–5 kg or 4–11 lb
Then continued weight loss of how much per week thereafter can cause GB dysfunction?	2.2–4.5 lb
What % of patients develop gallstones while losing weight rapidly?	50%

Weight gain during pregnancy

Approximate wt gain recommended since 1960s	25 lb
If patient obese	15 lb
If patient underweight	30–37 lb
Recommended extra calories per day while pregnant	300

WOUND CLOSURE

Continuous suture closure is
(1) Faster
(2) Cost-efficient
(3) Decreased risk of infection

Suture is to be left how far apart and how far back from fascial edge?	1 cm
Dexon and polyglactin 910 lose half tensile strength in	2 weeks
Maxon loses half tensile strength in	3 weeks
PDS loses half tensile strength in	6 weeks

Vaginal repair of bladder injuries

If you are going to be doing ANY vaginal surgery including minimally invasive surgeries – you will eventually injure the bladder and need to know how to repair it

(1) Lacerations 2 cm or less in size are usually amenable to vaginal repair
(2) Make sure the the perforation is well away (> 1 cm) from the ureteral orifices and there is free efflux from both orifices
(3) Close the defect from the vaginal side in 3 imbricating layers, being careful to keep the suture knots out of the bladder lumen
(4) Dissect the overlying vaginal mucosa off the endopelvic fascia for 1 cm around the defect to expose the bladder adventitia
(5) Reapproximate the bladder adventitia by placing the first suture in a running layer horizontally using a 3-0 synthetic rapid absorbable monofilament suture (Monocryl)
(6) Place the 2nd layer in a running fashion to imbricate the first suture line extending just beyond the angles of the first layer using a 3-0 delayed absorbable synthetic monofilament suture (PDS) for this layer
(7) Place the third-level suture in the adventitia to imbricate the second suture line, also extending this layer just beyond the ends of the second-layer suture. (3-0 PDS or some type of delayed absorbable synthetic monofilament suture)

ZIDOVUDINE (AZT)

Administration in pregnancy

Two-thirds relative reduction in vertical transmission (control 26%, treatment group 8% transmission)
Transient neonatal anemia noted in some study subjects
Consider treatment for all HIV-positive pregnant women after 14 weeks' EGA
All patients to receive zidovudine should be counseled regarding benefits/risks above

Antepartum therapy

Zidovudine 100 mg p.o. 5 ×/day

Intrapartum therapy

Recommended for any woman in pre-term labor requiring IV tocolytics and those scheduled for elective C-section

Loading dose (2 mg/kg)
Zidovudine _____ mg in 50 ml 5% dextrose in water. Administer over 60 min or
Zidovudine _____ mg in 50 ml 1.0% NaCl. Administer over 60 min

Maintenance infusion
Zidovudine 500 mg or 250 ml D5W. Rate: _____ mg/h
or
Zidovudine 500 mg or 250 ml 0.9% NaCl. Rate: _____ mg/h
Zidovudine _____ is stable in both NS and D5W
Choice of diluent dependent on patient needs (e.g. diabetic)

No data on IV compatibility of zidovudine, therefore, requires separate IV line for infusion

The concentration of the maintenance solution is 2 mg/ml. To calculate the rate for the infusion, divide the patient's weight (in kg) by two and round to the nearest whole number. Infuse at this rate until the patient delivers. Alternatively, the following chart can be used:

Patient's weight (kg)	*Rate (ml/h)*
50	25
52	26
54	27
56	28
58	29
60	30
62	31
64	32
66	33
68	34
70	35
72	36
74	37
76	38
78	39
80	40
82	41
84	42
86	43
88	44
90	45
92	46
94	47
96	48
98	49
100	50

KNOW THESE FOR THE BOARDS OR STAY AT HOME

Development of secondary sex characteristics

B	Breast bud → thelarche
P	Pubic hair → pubarche
A	Axillary hair → adrenarche
M	Menstruation → menarche
	Average age is 12.8 years
	The maximum growth spurt is just prior to menarche

Catheter	French = 3× diameter in millimeters	
	For example:	24 French = 8 mm diameter
Uterine weight	Normal is	60–90 g
	Myometrial hypertrophy begins at	120 g
Blood loss normally from menstruation is approximately		30–35 cc
	Menorrhagia is	> 80–85 cc or
		> 7 days of bleeding
Definition of amenorrhea is	No period for at least 6 months (some define it for at least 12 months)	
Definition of oligomenorrhea	>37 days between cycles	

Do you know the significance of the color of the tanks in the operating room?

If you do not know this one, you are in trouble. This has actually been asked during oral boards

Oxygen	Green
Nitrous oxide	Blue
Carbon dioxide	Gray

CIS is found on cervical biopsy.	What should be done prior to hysterectomy?	Conization

Why are normal ovaries sometimes removed?

(1) Patient's desire
(2) Family history of epithelial ovarian cancer
(3) Family member or friend had to have a reoperation
(4) Cancer risk is 1/70 for ovarian cancer
(5) 5—20% later have reoperation for pathology involving ovaries

List the steps to manage a shoulder dystocia

Adenomyosis is defined by what?

Endometrial glands and stroma invading myometrium by one of the following:
(1) 1 low-power field
(2) 2 high-power field
(3) 3 mm

Müllerian structures	All reproductive structures except the ovaries (arises from genital ridge) and lower 1/3 of the vagina (arises from urogenital sinus)

Name some ingredients that are in Premarin:

(1) Estrone
(2) Equilin
(3) Equilenin
(4) 17α-estradiol
(5) 17α-dihydroequilin
(6) 17α-dihydroequilenin

What dose of Premarin is the	Yellow pill?	1.25 mg
	White pill?	0.9 mg
	Dark red pill?	0.625 mg

Name the five characteristics of serous tumors of the ovary:

Serous
Single loculation
Ciliated

Psammoma bodies
Pseudostratified epithelium

Preterm labor (term is 37—42 weeks' gestation)

Why do we hydrate patients with preterm labor?
Because oxytocin and ADH is produced in the posterior pituitary and there are two theories why hydration may work:
(1) Flood gate theory — oxytocin spills with ADH when one becomes dehydrated
(2) Similar structure theory (oxytocin and ADH are similar structures)

Target heart rate with exercise

Formula is (220–age) × 0.8 for non-pregnant female
(220–age) × 0.7 for pregnant female

The maximum BPM desired is 140 BPM for pregnant female

Know the unit of measurements of the relevant hormones

Estradiol	pg/ml
Estrone	pg/ml
Estriol	pg/ml
Progesterone	ng/ml
17-OH progesterone	ng/ml
Androstenedione	ng/ml
DHEA	ng/ml
Testosterone	ng/ml
Prolactin	ng/ml
FSH	mIU/ml
LH	mIU/ml
TSH	micU/ml

Know the treatment of PID

Know Apgar scoring

	0	1	2
(1) Tone			
(2) Respirations			
(3) Heart rate	Absent >6 s	<100	>100
(4) Color			
(5) Grimace			

Know what is in the various blood products and when to use the each for specific indications

Maternal mortality

Ratio = # maternal deaths per 100 000 live births

Rate = # maternal deaths per 100 000 women of reproductive age

Perinatal death

22 week' gestation to 28 days postpartum

Neonatal death

Early First 7 days after birth
Late 7–29 days after birth

Infant death

Death that occurs anytime from birth through 12 months

Birthrate

# of live births per 1000

Fertility rate

# of live births per 1000 females aged 15–44

Cardinal movements of labor

Remember mnemonic–'Every Darn Fool In Egypt Eats Elephants'

Engagement
Descent
Flexion
Internal rotation
Extension
External rotation
Expulsion

Describe the biophysical profile

What is the definition of engagement?

The BPD passed the plane of the inlet with the presenting part is at the ischial spines

Estimated fetal weights for gestational age

	Weeks	*Weight (g)*
	20	500
	28	1000
	32	1600
(+250 g/week > 34 weeks)	34	2000
	36	2500
	40	3500
	Low birth weight	<2500g
	Very low birth weight	<1500g
	Extreme low birth weight	<1000g

Ultrasound findings associated with hCG level

	Sac seen	hCG level
	With vaginal probe	1500
	With abdominal probe	6000
	Cardiac motion with either probe	10 000

Explain the Bishop scoring system

	Factor	*Possible points*
	Dilatation	3
	Effacement	3
	Station	3
	Consistency	2
	Position of cervix	2
	Total possible points	13

Know how to diagnose and treat hyperemesis:

	Hyperemesis gravidarum	
	Normal specific gravity	1.020—1.030
	Ketones	acetone, aceto-acetate, β-OH butyrate
	Labs to obtain	CBC, lytes, U/A, TSH, LFTs, amylase
	Treatments	Phenergan = Category C and causes sedation
		Zofran ODT or oral = Category B with *no* sedative effects. This is an excellent choice for working pregnant women as it does not sedate
		Disadvantage compared to Zofran is that it is more expensive

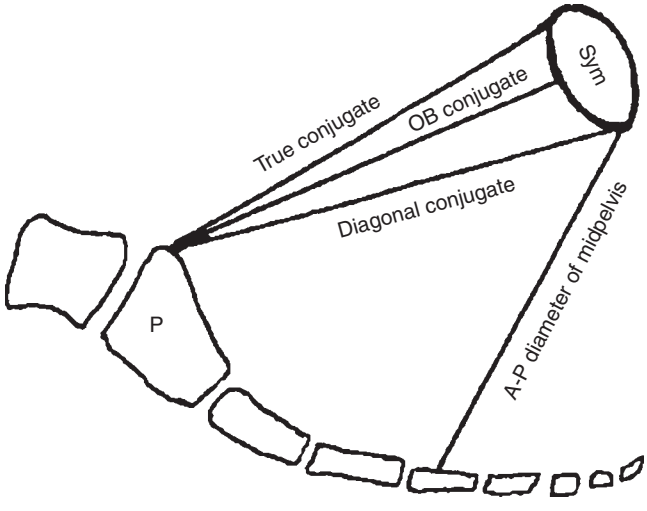
Fetal cord gases (normal)

	Arterial	*Venous*
*p*H	7.26	7.36
pCO_2	46	36
pO_2	19	29

Pelvic configurations

Gynecoid	Oval and round	50%
	Arch wide, sidewalls straight	
Android	Spines prominent, sidewalls converge	1/3 of women
	Worse prognosis	
Anthropoid	AP diameter > long transverse	1/4 of women
	OP frequent	1/2 black women and 1/4 white women
Platypelloid	Short AP and wide transverse	3%
	OT frequent	
Clinical pelvimetry	Inlet (average transverse diameter of pelvic inlet is 13.5 cm)	
	Diagonal conjugate should be	≥11.5 cm
	OB conjugate should be	≥10 cm (10.5 cm is normal)
Midpelvis	Interspinous diameter should be	≥10 cm
	A–P (anterior–posterior) diameter should be	≥11.5 cm
Pelvic outlet	Transverse diameter should be	≥11 cm
	AP diameter should be between	9.5–11.5 cm
	Posterior sagittal diameter should be	≥7.5 cm
	Biischial diameter (fist measurement) should be	>.8 cm

Pelvic conjugates



P, sacral promontory
Sym, symphysis

IMPORTANT TOPICS OFTEN DISCUSSED DURING ORALSV

Examples are given of how the author discussed or would have discussed some of these issues. Keep in mind that many examiners may be very opinionated one way or another regarding a particular subject

How would you predict shoulder dystocia?

The US predictability for > 4000 g has an average predictability error of >300—400 g. All methods of predictability have comparable sensitivities of no more than 60%. Therefore, it is very difficult to predict shoulder dystocia. One could only assess the patients' risks and alert her of that possibility if one was suspicious

How do you manage a post-term patient?

It would be reasonable to follow a patient after 42 weeks with a BPP or other protocol. However, one could justify induction as a reasonable alternative if the cervix was favorable or there were other mitigating circumstances keeping in mind that perinatal mortality rates double by 43 weeks and increase 4—6 times by 44 weeks' gestation

How do you calculate a Pitocin infusion?

For the Dublin Protocol (active management II)
10 units in 1000 cc
10 000 milliunits
10 milliunits in 1 cc
1 milliunit in 0.1 cc
× 60 = 6 cc per min

What are the Rh antigens? E, D, C, e, c (small d has not been identified)

FHR monitoring criteria No differences have been seen in patients who were monitored electronically versus intermittent Doppler auscultation Depends on the standard of care for the community

Ultrasound screening criteria Controversial

No proven cost-effectiveness
May or may not be standard of care in some communities

VBAC criteria 27% of females deliver vaginally after a previous LTCS in the USA

Literature does not set policy one way or other
Evaluate on individual case by case basis:
(1) Candidate?
(2) Type of uterine incision
(3) Unknown scar?

Anesthesia	Some studies seemingly show or support postponing epidurals until the cervix is 4–5 cm. One could use Sublimaze® (fentanyl) or other narcotic until the initiation of active labor. However, if a narcotic does not relieve the pain, it is feasible to administer the epidural earlier. In the author's own experience of over several thousand epidurals, it seems reasonable in that it seems to help the patient relax and enjoy her labor more
Breech criteria	Consider breech delivery if the obstetrician is experienced in this type of delivery and if: (1) Well-flexed head (2) Frank breech (3) Zatuchni—Andros Score is >5 (Parity, age of gestation, EFW, dilatation, station and previous breech) having been scored
ECV criteria	Completion of 36 weeks Reactive NST or BPP US prior to and after INFORMED CONSENT Scoring system (parity, dilatation, station, EFW, placent) ≥8 Rh p.r.n. Terbutaline p.r.n. especially for nullipara
VE criteria	There is need for caution FDA reported 12 deaths in a 4-year period and nine serious injuries in that time. This calculates to one event per 45 455 One needs to check placement, not exceed recommended pressures and limits
Forceps AND vacuum criteria	Presented with an OP presentation, would you rotate? There are definitely some risks. It would depend on the experience of the practitioner. One would need to evaluate the size and station of the head and individualize each particular case

Name the indications for forceps or VE use

(1) Maternal exhaustion
(2) Prolonged second stage
(3) Fetal bradycardia
(4) Maternal cardiac condition

Febrile morbidity: Defined as two temperature elevations to ≥38°C (110.4°F) outside the first 24 h > delivery or surgery and a temperature ≥ 38.7°C (101.5°F) at any time

Why was or might a hysterectomy be done in the secretory phase of endometrium?

The husband may have had a vasectomy or the patient may have had a tubal ligation. Both these situations essentially rule out possibility of an early pregnancy

When should a LEEP or conization be done?

(1) When biopsy does not explain abnormal cells
(2) When ECC has CIN
(3) When there is microinvasion on biopsy
(4) When atypical epithelium extension to endocervical canal
(5) When abnormal cytology with no visible colposcopic lesion

Incidence of accreta and previa

Previa	1/200
Previa with increased AMA >35	1/100
Previa with increased AMA >40	1/50
Previa with history of one C-section	1%
Previa with history of two C-sections	2%
Previa with history of three C-sections	4%
Creta	
Creta with placenta previa	5%
Creta with placenta previa and history of C-section	25%
Creta with placenta previa and history of two C-sections	50%
Creta with placenta previa and history of > three C-sections	>60%
Percent of placenta previa with creta that will have C-hysterectomy is	66%

Four conditions associated with a 25—50% mortality risk during pregnancy:

(1) Pulmonary hypertension
(2) Eisenmenger's syndrome
(3) Marfan's syndrome with aortic involvement/aortic root >4 cm
(4) Coarctation of the aorta

GBBS prophylaxis (ACOG)

Selective prophylaxis to all at risk, regardless of culture Risks:
(1) ROM equal or > 18 h
(2) LBW
(3) Preterm < 37 weeks
(4) Chorioamnionitis
(5) Intra-amniotic infection
(6) Previously affected infant
(7) GBBS bacteriuria in pregnancy
(8) Temperature > 100.4°F

Treatment:
Penicillin G 5 million units IV load, then 2.5 million units IV q. 4 h or ampicillin 2 g IV load, then 1 g IV q. 4 h
Allergy: clindamycin 900 mg IV q. 8 h till delivery

Etiology of cervical cancer — E6 and E7 viral proteins produced by high-risk type HPV → binds and disables p53 and Rb host proteins

Bowel prep — Give Golytely (polyethylene glycol electrolyte) 1 liter/hour on day prior to surgery. None > 4 liter or 4 h or phospho soda (4 oz at 1–3 p.m. then 4 oz at 5–7 p.m. on the day prior to surgery

Give either one of these till rectal effluent is clear
Cefotan 1 g or Unasyn 3 g IV @ 30 min to 1 h prior to surgery

Low versus outlet forceps–definition

Outlet:
Visible scalp
Fetal skull on pelvic floor
Sagittal suture in essentially the OA position
Fetal head on the perineum
Rotation can occur, but only up to 45 degrees
Low-forcep delivery:
Station of at least 2 +
Rotation can be more than 45 degrees
Mid-forcep delivery:
Station above 2 +
Engaged head

LFD vs outlet — LFD–2 + station or rotation > 45 degrees

Outlet–3 + station and rotation < 45 degrees

Ectopic dosage of methotrexate — 50 mg /m^2 or

1 mg/kg — 70 mg for 70 kg woman

Mean arterial pressure — Systolic – diastolic / 3 + diastolic = MAP

Example: 100/70 = (100 – 70) / 3 + 70 = 80 mmHg

Placenta previa — painless third-trimester bleeding

Management
(1) Marginal → to the os–expectant (depends on quantity of bleed)
(2) Partial → partially covers os–expectant (depends on quantity of bleed)
(3) Total → covers os completely–C-section

Placental abruption — painful third-trimester bleeding

Can only be a normally implanted placenta
Hemorrhage may be concealed
US only 5–10% accurate

Causes of high FSH
(1) 99% ovarian failure
(2) 0.9% 17β-hydroxylase deficiency
(3) 0.1% oat cell cancer

Induction with VBAC

- PGE_2 → dinoprostone preparations → two are approved (Cervidil and Prepidil) by FDA
- PGE_1 → misoprostol (Cytotec → given for PUD for patients on NSAID) → NOT approved by FDA or for prior C-section per ACOG. Dose is 25 μg q. 3 h or 50 μg q. 6 h. The 50 μg dose increases risk of tachysystole, meconium and uterine hyperstimulation

Vaginal vault prolapse

Non-surgical management:

Pessary (#3 donut is usual)

Surgical management:

(1) Sacral spinous ligament fixation
(describe pulley stitch, Miya hook, 2 cm medial to right of ischial spine)

(2) Abdominal sacral colpopexy with retropubic urethropexy and modified Halban's culdoplasty
(describe vaginal vault, Marlex mesh or synthetic graft, sacrum, middle sacral artery – bone wax and sterile thumb tacks on table)

Hirsutism

Most patients with androgen excess can be screened efficiently by measuring = total serum testosterone and serum DHEA-S

Free testosterone is hormonally active, but measurement of total testosterone is suffieient for clinical test
Principal clinical entity that is associated with an increase in testosterone is PCO
Testosterone-secreting tumors are usually associated with testosterone levels >200ng/dl
Ovary and adrenal glands make roughly equal amounts of testosterone
DHEA-S is produced almost entirely by adrenal gland
Measurement of DHEA-S indicates if there is significant adrenal component
Very increased levels of DHEA-S (>700 μg/dl) consistent with rare adrenal tumors

Treatment:
Low-dose OCPs are as effective as higher-dose preparations
Spironolactone is helpful in the treatment of idiopathic hirsutism because this drug competes for the androgen receptor at the site of the hair follicle and decreases 5α-reductase activity (associated with peripheral conversion of testosterone to DHT)

Neural tube defects

Why does folic acid reduce the incidence of neural tube defect?

Although NTD is multifactorial, the cause is an abnormal gene that is a variation of the gene that normally produces an enzyme (5,10-methylenetetrahydrofolate reductase) which is critical for folate use

This is why folic acid reduces the incidence of NTD by 50%

Thrombosis factors

Factor VIII	25%
Leiden V	20%
Homocysteine	10%
Protein 20280	6%
Protein C deficiency	3%
Protein S deficiency	1–3%

Tay–Sachs

Highlights to remember:

(1) Autosomal recessive disease
(2) Lysosomal storage disease in which GM2 gangliosides accumulate throughout body
(3) Increased incidence in Jews of East European descent (Ashkenazi) 1/30
(4) French Canadian and Cajuns have an increased incidence too

Chemotherapy reactions to remember using mnemonics

(1) Pulmonary fibrosis–Taxol, bleomycin 'TB'
(2) Alopecia–ifosfamide, 5-FU, doxyrubicin, methotrexate
'I've 5 hairs on my Dang Mutt'
(3) Severe inflammatory/ulcerative reactions–doxyrubicin, mitomycin-C, actinomycin D
'Dang My Arm'
(4) Bone marrow toxicity–doxirubicin, vinblastin, methotrexate, carboplatinum
'Death Via Marine Corps'
(5) Others to remember:
Hemorrhagic cystitis Cytoxin

Leukemia	Alkeran
Coma	Ifosfamide
Cerebellar ataxia	5-FU
Bone toxicity	Vinblastine
Neurotoxicity	Vincristine
Renal toxicity	Cisplatin

Chemotherapy basics

S-DNA synthesis phase:
Alkylating agents
Antitumor antibiotics
Antimetabolites
Synthetic compounds
Mitosis phase:
Vinca alkaloids
(Most sensitive to radiation)

Shoulder dystocia basics

Incidence	1%
Head-to-body delivery time–normal	Approximately 24 s
Shoulder dystocia	> 60 s
No compromise if up to but not over	2½ min or 150 s

Morbidity and mortality–brachial plexus injury, fractured clavicle or humerus, Erb's palsy, severe asphyxia or death. Erb's palsy involves C5–6, sometimes C7

Risk factors – diabetes, obesity, post-term — Unpredictable

Management:
(1) HELP
(2) Episiotomy (extend if already present)
(3) Suprapubic pressure (NOT fundal)
(4) McRobert's
(5) Wood's
(6) Posterior arm
(7) Fracture
(8) Zavenelli

Drugs to consider for leaking bladder, remember:

Continence	Micturition
Norephinephrine	Acetylcholine
Sympathetic	Parasympathetic
Adrenergic	Cholinergic

Infant with ambiguous genitalia

Incidence — 1/5000–1/15 000

Female with classic CAH (congenital adrenal hyperplasia) to be ruled out since this is most common
Classic CAH is characterized by either:
(1) Salt-wasting
21-Hydroxylase deficiency (90%)
(2) Non-salt-wasting
No salt-losing crises have been reported < 7 days of age
Say to parents at birth, 'Your baby appears healthy, but the sexual organs have not completely developed'

MISCELLANEOUS TOPICS WITH PERCENTAGES AND NUMBERS

Death of one remaining twin following MFPR	15%
Success rate of abdomino-pelvic I&D of abscess	80%
Risk of recurrence for many major anomalies	2—4%
Risk of woman getting breast cancer (iifetime) if mother had bilateral breast cancer	40—50%
% of women who are infected with *Trichomonas* that are asymptomatic	50%
% of twin pregnancies that require at least one admission prior to labor	50%
% of Rh sensitization in current pregnancy without RhoGAM	2%
Risk of Rh sensitization after first Rh + child	8%
Risk of Rh sensitization after fifth Rh + child	50%

Incidence of asymptomatic bacteriuria	5%
Asymptomatic bacteriuria that develop into pyelo if untreated	25%
Asymptomatic bacteriuria that develops into pyelo if treated	2%
Sickle cell trait increases risk of pyelo	2×
If a pt is pregnant with dizygotic twins and couple are both carriers for Tay—Sachs, one twin affected?	44%
% of females that cease menstruation after age 50	25%
Approx. distance from plane of pelvic inlet to level of ischial spine	5 cm
Risk of primary peritoneal cancer after prophylactic oophorectomy in increased risk pts	2–15%
VBAC success rate after prior C-section for delay of descent in second stage of labor (Netherlands)	80%
If a sickle cell pt has children by a man known to have SC trait, chance each child will have disease	50%
What % of eclamptic patients will be eclamptic in a subsequent pregnancy?	<5%
Atypical endometrial hyperplasia (if unrxed) progresses to endometrial cancer in @ what % pts?	25%
Risk of a woman becoming infected with HIV after transfusion with a unit of allogenic blood	1/680 000
Approximately how many pts in the USA receive the wrong unit of blood each year?	1000
% women who will get ab rx if current strategies are used to prevent GBS disease in newborn	25%
Each unit increase (1 kg/m^2) in body mass index in pregnancy is associated with what % increase in odds of C-section?	7%
Women most often begin to smoke during what years of age?	11–14
Increased incidence of venous thrombophlebitis in pregnant women compared to women using second-generation oral contraceptives is	2×
Risk of HIV infection after percutaneous exposure to HIV infected blood	0.3%
The reported incidence of shoulder dystocia varies with definition, but the range is	1–5%
Probability of delivering viable infant after recurrent pregnancy loss in pt with septate uterus who does not have surgical therapy	80%
Probability of delivering viable infant after three consecutive spontaneous abortions	60% (50—70%)
Balanced translocation	
Of surviving offspring, the theoretically affected	33%
OBSERVED RISK (maternal)	10%
OBSERVED RISK (paternal)	3%
Addition (affected)	25%
Deletion (usually fatal)	25%
Carrier of translocation	25%
Normal	25%
Germ cell tumors (malignant)	50%
Epitheliai cell tumors (malignant)	70%
Approx. risk of congenital rubella at 7–8 weeks' gestation exposure with maternal symptoms and incidence (1:160 titer)	25–50%
Perinatal mortality for pt with chronic htn with superimposed PIH	20%
Massive hydramnios (over 3000 ml) is associated with congenital malformation	20–30%
Consanguinity:	
Share autosomal recessive traits	1:4
First cousins	1/8
Second cousins	1/16
Perinaiai mortality rate in eclampsia	20%

GFR (during pregnancy) increases as much as	50%
Achondroplasia caused by mutation accounts for this % of cases	90%
Risk of uterine rupture in presence of previous LTCS is	1%
Chance that a couple unable to conceive after 1 year (all test WNL) will conceive after 3 years trying	50%
In using GnRH agonist, headaches occur in what % of patients?	25%
Rhabdomyosarcoma accounts for this % of malignant disease of children under 15 years old?	4–8%
Hyperreflexia is present with PIH in what % of cases?	80%
Theca lutein cysts occur in association with hydatidiform mole in what % of cases?	50–60%
Theca lutein cysts occur in association with choriocarcinoma in what % of cases?	5–10%
The false-negative Pap smear rate is	10–19% or 15–40%
Quantitative hCG doubles normally every 48 h in what % ectopics?	20%
Another ectopic will occur following salpingostomy in what % cases?	15%
Fetal occult blood screening for colorectal cancer can detect	70%
Accuracy of crown–rump length measurement for estimated gestational age	95% ± 4.7 days
Untreated atypical hyperplasia progresses to carcinoma of endometrium in what % patients?	25%
% of USA population that does not use any means of birth control	33%
% of female homicide victims due to domestic violence	40–50%
Rate of shoulder dystocia in macrosomic (over 4000 g) of diabetic mothers	30%
Females with gestational diabetes who will develop overt DM in 5–15 years after pregnancy	60%
Combined incidence of carcinoma (both invasive and *in situ*) of cervix in pregnancy	<0.5%
G6PD homozygous deficiency is present in African-American females in what %?	2%
Ovarian dysgenesis are chromatin-positive	38%
Chromosomal abnormalities in live-birth infants	<1%
Drop in urethral closing pressure at rest and during stress in a postmenopausal patient is	30%
Earliest time for a reliable Hgb determination after transfusion of PRBCs	15 min
Incidence of vulvar carcinoma (% of gyn malignancies)	8%
Incidence of vaginal carcinoma (% of gyn malignancies)	2%
Fetomaternal transfusion of over 30 ml has been found in less than what % of pts at delivery?	1%
Interspinous diameter of the pelvis should be at least equal to	10 cm
Sickle cell disease – if one parent has the disease and one the trait, what % children will have disease?	50%
Couples who suffer habitual abortion – rate of chromosomal abnl disease	25%
MHC (major histocompatibie complex) in humans is located on what chromosome?	6
% of reproductive-age couples unable to conceive after 1 year of coitus without contraception is	15%
Ureter at its closest position from the cervix is separated by	12 mm or 1.2 cm
Overall subsequent conception rate in women with an ectopic pregnancy is	60%
In a high-risk obstetric population undergoing antepartum fetal testing, perinatal mortality rate is	12/1000
Of women with an ectopic pregnancy, the number who have subsequent live birth is about	1/3
Genuine SUI evaluated by the Q-tip test, if + will dem urethral—vesical hypermobility	67%
Perinatal loss in twins weighing more than 1000 g is greater than singletons by	3×
What % of DUB is associated with the ovulatory menstrual cycle?	20%
In the second half of pregnancy, changes in fetal skeletal muscle are responsible for this % of fetal wt increase	25%
Women at risk for osteoporosis + not treated – the % of bone mass loss per year after menopause	1–1.5%
Childhood sexual abuse is reported when questioned in what % of chronic pelvic pain pts?	60–70%

Unilateral ureteral compromise is most often associated with what degree of increase in ser creatinine?	0.8 mg/dl
The ratio of infused crystalloid solution to estimated blood loss should be approximately	3:1
Fetal loss resulting from minor trauma in pregnancy is approximately	1.7%
Approximately what % of pregnancies in the USA are unplanned?	50%
What % of women at risk for osteoporosis who were prescribed estrogen rx, continue for 1 year?	50%
After 10 years of annual mammographs, estimated cumulative risk of at least one false + mammography screening is	50%
After 10 years of annual exams, estimated cumulative risk of at least one false + screen test by PE of breast is	25%
National rate of false-positive results for a single screening mammography test is	8–9%
Combining results of mammography and exam of breast, age group with lowest cumulative false + rate	70–79
Increased mortality is observed in women consuming more than how many alcoholic drinks per day?	2½
Diagnostic accuracy of clinical assessment in IUGR is	35%
Use of DeLee catheter by an Ob clears upper airway of meconium in what % of cases?	90%
Correct dose of naloxone to neonate showing signs of respiratory depression due to meperidine	0.01 mg/kg
What % of SGA infants will still be less than two standard deviations below normal wt at 3 years old	50%
In term infant, hypoglycemia is defined as blood sugar below what level on two occasions during first 72 h of life?	30 mg/dl
Klumpke's palsy usually involves which branches of the brachial plexus?	C8 & T1
Anemia of the newborn is defined as Hgb of less than what level?	12 g
Indomethacin therapy in the newborn is successful in @ what % of infants with PDA?	75%
What % of patients with spina bifida will have the communicating type of hydrocephalus?	75%
What % of infants with neural tube defects will be identified by a MSAFP screening program?	80%
What % of neural tube defects will have a skin covering the defect (i.e. closed defect)?	5%
Using X-ray techniques, how early can the distal femoral epiphysis be visualized?	32 weeks
The crown–rump length as determined by ultrasound is accurate with what range of error?	5 days
Using the 10th percentile for birth weight to ID the growth-restricted fetus, what % of nl fetuses?	7%
What % of the placental tissue would have to be altered by infarction for fetal compromise to occur?	50%
What % of post-term pregnancies will be associated with a macrosomic infant?	25%
What % of macrosomic infants (>4000g) of diabetic mothers have shoulder dystocia?	30%
Necrotizing fasciitis secondary to an episiotomy site infection has been reported to be associated with what degree of mortality with aggressive surgical treatment?	50%
What % of pts after C-section will experience bacteremia secondary to uterine infection?	20%
What % of pts presenting in preterm labor are candidates for long-term therapy to prevent PTD?	15%
Percentage of women who have eclampsia without evidence of severe hypertension	20—25%
In what % of couples who have had two or more spontaneous abortions, will one member of couple carry a balanced recessive translocation?	3%
What % of pts with acute pyelo experience transient decrease in GFR in conjunction with a rise in blood creatinine?	10%
What % likelihood of a successful pregnancy for pt on chronic hemodialysis?	20%
What level of creatinine would denote severe renal insufficiency in prepregnancy renal dysfunction evaluation?	10 mg/100 ml
What % of Caucasians are Rh negative?	15%
What period of time does one have to give RHIG prophylaxis if not given within 72 h of delivery?	28 days
To undertake an elective abortion at 10 menstrual weeks' gestation, the correct size of suction cannula is	8 mm
What % of elective abortions are second-trimester abortions?	10%

What % of pts thought to have clinically certain DVT are found to have normal venography?		45%
What % of pulmonary arterial circulation must be included for diagnosis of massive pulmonary embolism?		50%
What period of time would umbilical cord blood gases be considered valid if put on ice?		3 h
Risk of perinatal transmission of the HIV virus is		35%
A dietary increase of how many calories is necessary to maintain body weight during lactation?		500
Best estimate of the risk for developmental defects in man secondary to drug exposure during pregnancy is		3%
A pt with neg EEG plans pregnancy, what seizure-free period should pass before withdrawal of anticonvulsants?		4 years
Hereditary ovarian cancer syndromes (Lynch syndrome II, Ca Fam syndrome, etc.) account for ovarian cancers		< 15%
Likelihood of another required surg after resection of endometriosis after hysterectomy but leaving ovaries		< 10%
Chance that asymptomatic pt whose mother just had hysterectomy for endometriosis will develop endometriosis?		6—8%
Emergency contraception reduces pregnancy by		55—94% or 75%
Moniliasis is caused by treatment of UTIs by ampicillin and tetracycline by		25%
What % of UTIs resolve without therapy? 50%		
Reversal of sterilization performed by clips/bands has success rate of		70%
What % of sexual assaults occur in the victim's home?		50%
PID	Lower abdominal pain	90%
	Mucopurulent cervical discharge	75%
	Sed rate > 15	75%
	WBC > 10 000	50%
Third-trimester bleeding		
	Placenta previa	20%
	Abruption	30%
HIV – risk of perinatal transmission is about		25%
Risk of malformations in an insulin-dependent diabetic pregnancy with $HgbA_{ic}$ > 8.5 is		22%
Varicella zoster		
	% of maternal infections resulting in evidence of fetal infection	25%
	% of first-trimester maternal varicella resulting congenital varicella syndrome	<3%
	% of pregnant women that develop varicella pneumonia	10—30%
	Mortality rate of varicella pneumonia is	40%
Rubella fetal infection depends on stage of gestation		
	< 11 weeks – risk on cong infection	90%
	11–12 weeks	33%
	13–14 weeks	11%
	15–16 weeks	24%
	> 16 weeks	0%
Rubella anomalies		
	First month	50%
	Second month	25%
	Third month	10%
	Second trimester	<1%
	16–20 weeks	sensory only
	20 weeks	no reported cases
Cytomegalovirus (CMV) complicates what % of pregnancies?		0.2–2%

What % of mothers have already been infected with CMV?	80%
Toxoplasmosis rate of infection	
First trimester	15%
Second trimester	30%
Third trimester	60%
Herpes shedding occurs at time of delivery in what % of all patients?	0.1–0.4%
Recurrence risk of abruptio placentae is	5–16%
Recurrence risk of abruptio placentae rises to what % after two previous abruptions?	25%
Oxygen consumption increases how much % in pregnancy?	25%
Postpartum blues (mild transient depression) occurs within 1–2 weeks of delivery – its incidence	10%
What % of untreated climacteric women have hot flashes for more than 5 years?	25%
The diagnostic accuracy of clinical assessment in IUGR is @	35%
Sarcoidosis most likely relapses in puerperiurn:	
Disease onset is abrupt	25%
Asymptomatic at discovery	10%
Interstitial pneumonitis is hallmark of pulmonary involvement with permanent X-ray changes in	50%
Lymphadenopathy especially in mediastinum is present in	75—90%
Uveitis present in	25%
Skin involvement (usually erythema nodosum)	25%
Overall prognosis good but % patients that die is	10%
The maternal X is missing in Turner's syndrome in what % of cases of Turner's?	70%
Two unaffected parents who just delivered a child with cleft lip have what % chance of delivering another with cleft lip?	4%
Adolescents aged 15—19 years old that use birth control, use oral contraceptives in what %?	44%
Among typical large group of insurers (indemnity plans) @ what % cover no contraception whatsoever?	49%
What % of women with breast cancer in pregnancy have positive lymph nodes?	50—80%
What is the daily folic acid requirement or recommendation for twin pregnancy?	1 mg
For daily recommendation prior to and during normal pregnancy?	0.4 mg
For pregnancy complicated with history of NTD or epilepsy, etc?	4 mg
GnRH analog flare usually lasts	5 days
Longest time period during which fetal body movements are absent	13 min
Mean length of the quiet or inactive state for term fetuses (i.e. 'sleep cyclicity')	23 min
Maternal mortality in the USA is	8/100000 live births
Postpartum development of pulmonary embolism is relatively uncommon with an incidence of about	1:5000
Varicoceles are found in approximately what % of the general population?	15%
Normal Sims—Huhner test should reveal at least how many motile sperm per high-power field?	1–20
Time required for the full effect of an increase in oxytocin dosage to be evident is	30–40 min
Pheochromocytoma is known as the ? % tumor because it is bilateral, outside the adrenal and malignant?	10%
The Copper T380A (ParaGard) IUD is approved for what maximum duration of use?	10 years
% of American women who will develop breast cancer sometime in their lives	12%
70-year-old debilitated patient receiving D5LR at rate of 125 cc/h – what is 24-h total calorie input pt is receiving?	600 calories
% of estriol from FETAL source of placental estriol precursors	90%
How many new cases of ovarian cancer are diagnosed in the USA each year?	20 000

By what % is a patient's risk of ovarian cancer felt to decrease with each child that she *delivers*?	20%
Incidence of ovarian cancer among the overall population of American women today is approximately what?	1:70
Incidence of ovarian cancer among American women under age of 40?	1:424
PID indicates that incidence of tubal infertility is @ 12%, 23% and 54% after one, two and three episodes – what is the risk of ectopic pregnancy after PID?	6–7 times
Number of annual deaths related to ectopic pregnancy is	25–50
The 5-year survival rate of Stage II uterine cancer is	60%
Transfusion rate with placenta previa	30%
Normal infant, after normal delivery, will have a normal adult pH in about	1 h
CT can detect pelvic masses as small as	2 cm
After an ectopic pregnancy, the risk of subsequent ectopic pregnancy is increased by how many fold?	10
Normal daily fluid requirement in the average adult is	2000–3000 ml
An MI is treated with tPA — how many days after the discontinuance of tPA will pt be able to undergo major surgery?	10 days
False-positive rate for a contraction stress test	25–75%
False-negative rate of a contraction stress test	15%
What % of women who have abnormal bleeding will have endometrial polyps in the uterine cavity?	25%
% of endometrial polyps that undergo malignant transformation?	0.5%
% of endometrial polyps that are solitary	80%
% of endometrial polyps that are multiple	20%
Clonal rearrangement of what chromosome is common in the mesenchymal (stromal) ceils of the polyp?	6p21
Hysteroscopy is best method of management of endometrial polyps because only ? % are removed with curettage?	25%
Placenta accreta, increta and percreta (incidence)	1:7000
What % of pts with placenta previa/placenta accreta will have to have Cesarean hysterectomy?	66%
What is the chance that there will be subsequent developmental delay at 32 weeks' gestation if a fetus is noted to have ventriculomegaly?	>25%
Long-term condom use reduces infertility use by what %?	40%
Long-term condom use reduces invasive cancer of the cervix by what %?	60%
Invasive prenatal diagnosis for the detection of fetal aneuploidy should be offered to women with twin gestation at what age?	31 years
In relationship to all gyn cancers, the frequency of cancer of the fallopian tube is	>1%
Hydatidiform mole	
Present with vaginal bleeding	75%
Hyperemesis	8%
Therapy is curative	80%
Twin-twin transfusion	
Monochorionic pregnancies (does not occur in dichorionic)	1%
Birth weights discordant by	20%
Hemoglobin differences	5g/dl
Poor prognosis	< 10% survival
Dermoid chance of malignant transformation (usually squamous)	2%
Dermoids are bilateral	25%
Struma ovarii (what % ovarian teratoma?)	2–3%
Hyperprolactinemia with only galactorrhea is	62%

With galactorrhea and amenorrhea	88%
Have hypothyroidism with it (measure TSH)	3–5%
Microadenoma	<1 cm
Macroadenoma	>1 cm
Duodenal atresia	
Is often identified in	third trimester
% have associated anomalies	50%
% have trisomy 21	30%
Ureteral injury	
Incidence	0.1—2.4%
Due to gyn procedures	52%
What % recognized intraoperatively?	20—30%
Most occur beneath the uterine artery	75%
Most asymptomatic postop but flank pain is present	33—75%
Serum creatinine increases ×24 h up above preop levels	0.8 mg/dl
Postop creatinine of ? in pt without renal disease?	1.5 mg/dl
At what stage of fetal development is feminization of the external genitalia complete?	250—300 mm crown—rump length
The pO_2 of the umbilical venous blood is approximately	30—35 mmHg
The perinatal mortality in IUGR is higher than in normally grown fetuses by	5—10 times
Haif-iife of progesterone is	30 min
Cocaine can generally be detected in the urine for no longer than	3 days
Antibodies to the HIV virus take how long to develop?	6—12 months
% of chronic pelvic pain patients who report childhood sexual abuse when questioned is	60—70%
Recommended IV dose of epinephrine in patients with cardiovascular collapse is	5 ml of 1:10000 solution
The human conceptus is most susceptible to teratogens at which embryonic week (week since conception)?	6
In performing a menstrual extraction 6—7 weeks after the LMP, what size suction cannula is recommended?	6 mm
Remission after the use of single agent chemo for non-metastatic trophoblastic disease is how many normal weekly hCG titers?	3
Rx of good prognosis gestational trophoblastic disease, how many courses of chemotherapy should be given after a negative titer?	1
Average interval between IM injection of DMPA and resumption of ovulation is approximately how many months?	7–9
With high-grade dysplasia, laser vaporization and destruction of tissue should be carried to a depth of	5–7 mm
Hemoglobin F is what % of the total hemoglobin at birth?	75%
Average transverse diameter of the pelvic inlet in the female measures	13–14 cm
Max contractile response occurs when intracellular Ca* increases to	500 nm
USP categorizes suture material as non-absorbable if tensile strength is maintained for more than	60 days
Fertilized ovum reaches uterus in	5–6 days
Extra calories per day required for pregnancy are	300
The empiric risk for a fetus with a balanced translocation, to have anomalies or to develop mentail delay	10%
Basal O_2 consumption increases by the second trimester by	20 ml/min
Exposure to rubella at 7—8 weeks' gestation — days later rash and titer to 1:160 when seen at 11 weeks — what is @ risk of fetus having serious congenital abnormalities?	25–50%

Chorioamnionitis	
Occurs in term pregnancies	1–5%
Occurs in preterm pregnancies	25%
Increased infant mortality in term infants	1–4%
Increased infant mortality in preterm infants	15%
Increased FHR abnormalities (increased tach and dec variability)	75%
Chorioamnionitis	
Increased dysfxn labor	
Require oxytocin	75%
Require C-section	34–40%
During the last month of pregnancy, the fetus grows at a rate of @	250 g/week
Oogenesis begins between what weeks of development?	11–12 weeks
Rate of shoulder dystocia in macrosomic infants >4000 g of diabetic mothers is	30%
Increased incidence of venous thrombophlebitis in pregnant women compared with women using second-generation OCPs	2×
Fetal *breathing* movements may be totally absent for	120 min (2 h)
Normal vaginal pH	3.8–4.4
Immunity from hepatitis B vaccine appears to last at least	8 years
Detection of anencephaly is effective as early as	1o weeks
LGSIL regresses spontaneously in what % of cases?	60%
Hereditary ovarian cancer syndromes account for what % of ovarian cancers?	<15
What is the shelf-life of whole blood?	40 days
Contractions decrease uterine blood flow by what %?	60%
Maximal contractile response occurs when intracellular calcium increases to	500 nm
If pt has esophageal candidiasis — HIV is + then do CD4 count or CD4%, if CD4 count is <? or CD4% is <?, pt has AIDS	<200, <14%
What % of newborns weigh >4000 g? Over 4500 g?	5.3% and 0.4%
Overall incidence of shoulder dystocia is	0.6—1.4%
If a woman weighs >300 lb, what is the risk that her fetus will be macrosomic?	30%
Define shoulder dystocia in time from head-to-body delivery	>60 s
How much time does one have to deliver the baby without compromise to neonatal outcome?	150 s (2½ min)

ABBREVIATIONS

17-OHP	17α-hydroxyprogesterone
ab	antibiotics
ABG	arterial blood gas
abnl	abnormal
AC	abdominal circumference
ACIS	adenocarcinoma *in situ* of the cervix
ACOG	American College of Obstetrics and Gynecology
ACTH	adrenocorticotropic hormone
AD	Alzheimer's disease
AED	anti-epileptic drug
AF	amniotic fluid
AFE	amniotic fluid embolism
AFI	amniotic fluid index
AFP	alpha-fetoprotein
AFV	amniotic fluid volume
AGCUS	atypical glandular cells of undetermined significance
AIDS	acquired immune deficiency syndrome
ALL	acute lymphoblastic leukemia
ALT	alanine aminotransferase
AMA	advanced maternal age
AML	acute myelogenous leukemia
AMP	adenosine monophosphate
ANA	antinuclear antibody
ANP	arterial natriuretic peptide
AP	anterior–posterior
APLA	antiphospholipid antibodies
APP	apolipoprotein
APT	aspartate transaminase
APTT	activated partial thromboplastin time
ARDS	acute respiratory distress syndrome
AROM	artificial rupture of membranes
ASA	acetylsalicylic acid (aspirin)
ASCUS	abnormal squamous cells of undetermined significance
ASD	atrial septal defect
ASHD	atrial septal heart defect
AST	aspartate aminotransferase
AZT	zidovudine
b.i.d.	bis in die (L. twice a day)
B/P	blood pressure
BBT	basal body temperature
BE	barium enema
BMD	bone marrow depression; bone mineral density
BMI	body mass index
BNP	brain natriuretic peptide
BOO	bladder outlet obstruction
BPD	biparietal diameter
BPM	beats per minute

BPP	biophysical profile
BS	bowel sounds
BSO	bilateral salpingo-oophorectomy
BTB	breakthrough bleeding
BUN	blood urea nitrogen
BV	bacterial vaginitis
Bx	biopsy
Ca	carcinoma
CAD	coronary artery disease
CAH	congenital adrenal hyperplasia
CBAVD	congenital bilateral absence of the vas deferens
CBC	complete blood count
cc	clomiphene citrate
CD	Cesarean delivery
CEA	carcinoembryonic antigen
CEE	conjugated equine estrogen
CF	cystic fibrosis
CFTR	cystic fibrosis transmembrane regulator
CHD	congestive heart disease
CHF	congestive heart failure
CHO	carbohydrate
CIN	cervical intraepithelial neoplasia
CIS	carcinoma *in situ*
CKC	cold knife conization
CMV	cytomegalovirus
CNS	central nervous system
COH	clomid ovarian hyperstimulation
COPD	chronic obstructive pulmonary disease
CP	cerebral palsy
CPD	cephalopelvic disproportion
CPM	confined placental mosaicism
CPR	cardiopulmonary resuscitation
C–R	crown—rump length
CRF	corticotropin releasing factor
CSE	combined spinal /epidural
C-section	Cesarean section
CST	contraction stress test
CT	computed tomography
CVP	central venous pressure
CVS	chorionic villous sampling
CXR	chest X-ray
cysto	cystoscopy
D&C	dilatation and curettage
D&E	dilatation and evacuation
D/c	discharge
DCIS	ductal carcinoma *in situ*
DES	diethylstilbestrol
DHEA	dehydroepiandrosterone
DHEA-S	dehydroepiandrosterone sulfate
DI	detrusor instability, diabetes insipidus

DIC	disseminated intravascular coagulation
DKA	diabetic ketoacidosis
DM	diabetes mellitus
DMPA	depo medroxyprogesterone acetate
DOC	deoxycorticosterone
DTaP	diphtheria—tetanus—acellular pertussis
DUB	dysfunctional uterine bleeding
DVT	deep vein thrombosis
DXA	dual energy X-ray absorptiometry
Dxn	diagnosis
E_2	estradiol
EBL	estimated blood loss
EBT	electron beam tomography
EC	emergency contraception
ECC	endocervical curettage
ECV	external cephalic version
EDC	estimated date of conception
EDD	estimated due date
EDRF	endothelium-derived relaxing factor
EFM	external fetal monitoring
EFW	estimated fetal weight
EGA	estimated gestational age
EKG	electrocardiogram
ER	Emergency Room
ER	estrogen receptor
ERCP	endoscopic retrograde cholangiopancreatography
ERT	estrogen replacement therapy
ET	embryo transfer
ETON	ethyl alcohol
F/u	follow-up
FBS	fasting blood sugar
FDA	Food and Drug Administration
FDP	fibrinogen degradation product
FEV	forced expiratory flow rate
FFN	fetal fibronectin
FFP	fresh frozen plasma
FH	fetal heart
FHR	fetal heart rate
FIGO	International Federation of Obstetrics and Gynecology
FL	femur length
FLM	fluorescent polarization test
FMH	family medical history
FSH	follicle stimulating hormone
FSI	Foam Stability Index
FSP	fibrinogen split product
FT_4	free thyroxine
GABA	gamrna-aminobutyric acid
GAG	glycoaminoglycan
GBBS	group B beta streptococcus

GC	gonococcal
GDM	gestational diabetes mellitus
GERD	gastroesophageal reflux disease
GFR	glomerular filtration rate
GH	growth hormone
GI	gastrointestinal
GnRH	gonadotropin releasing hormone
GSUI	genuine stress urinary incontinence
gtt	guttae (L. drops)
GTT	glucose tolerance test
GV	great vessels
Gyn	gynecology
H&H	hematocrit and hemoglobin
H&P	history and physical
HAMA	human antimouse antibody
HC	head circumference
hCG	human chorionic gonadotropin
Hct	hematocrit
HDL	high-density lipoprotein
HF	heart failure
Hgb	hemoglobin
HGSIL	high-grade squamous intraepithelial lesion
HIV	human immunodeficiency virus
HLA	human leukocyte antigens
hMG	human menopausal gonadotropin
HNPCC	hereditary non-polyposis colorectal cancer
HPF	high-power field
HPL	human placental lactogen
HPV	human papilloma virus
hs	hora somni (L. at bedtime)
HRT	hormone replacement therapy
HSDD	hypoactive sexual desire disorder
HSG	hysterosalpingography
HSV	herpes simplex virus
ht	height
HT	hormone therapy
htn	hypertension
hx	history
I&D	incision and drainage
I&O	intake and output
IBS	irritable bowel syndrome
IBW	ideal body weight
ICU	intensive care unit
IGD	isolated gonadotropin deficiency
IGF-1	insulin-like growth factor-1
IgG	immunoglobulin G
IgM	immunoglobulin M
IM	intramuscular
IMP	intermediate progestational
INR	international normalized ratio

IPPB	intermittent positive pressure breathing
IRP	International Reference Percentiles
ISD	intrinsic sphincter deficiency
ITP	idiopathic thrombocytopenic purpura
IUD	intrauterine device
IUFD	intrauterine fetal demise
IUGR	intrauterine growth restriction
IUI	intrauterine insemination
IUP	intrauterine pregnancy
IUPC	intrauterine pressure catheter
IV	intravenous
IVF	*in vitro* fertilization
IVFs	intravenous fluids
IVH	intraventricular hemorrhage
IVIG	intravenous immunoglobulins
IVP	intravenous pyelogram
JVD	jugular venous distention
L	left
LA	long acting
LBW	low birth weight
LCIS	lobular carcinoma *in situ*
LDH	lactate dehydrogenase
LDL	low-density lipoprotein
LEEP	loop electrocoagulation excision procedure
LES	lower esophageal sphincter
LFT	liver function tests
LGSS	low-grade endometrial stromal sarcoma
LGSIL	low-grade squamous intraepithelial lesion
LH	luteinizing hormone
LHF	left heart failure
LMP	last menstrual period
LOP	left occipitoparietal
Lp(a)	lipoproteln(a)
LPF	low-power field
LR	Ringer's lactate
LTCS	low transverse C-section
MAM	menstrually associated migraine
MCV	mean cell volume
MG	myasthenia gravis
MHC	major histocompatibility complex
MI	myocardial infarction
MIF	Müllerian inhibiting factor
MIVH	minimally invasive vaginal hysterectomy
MPA	medroxyprogesterone acetate
MRI	magnetic resonance imaging
M-R-K-H	Mayer-Rokitansky-Kuster-Hauser syndrome
MS	multiple sclerosis
MSAFP	maternal serum alpha-fetoprotein
MSH	melanocyte stimulating hormone

Mtx	methotrexate
MVP	mitral valve prolapse
NCEP	National Cholesterol Education Program
NE	nectrotic enteritis
NG	nasogastric
NGU	non-gonococcal urethritis
NIDDM	non-insulin-dependent diabetes mellitus
nl	normal
NMG	neonatal myasthenia gravis
NPH	isophane insulin
NPO	non per os (L. nothing through the mouth)
NS	normal saline
NSAID	non-steroidal anti-inflammatory drug
NST	non-stress test
NTD	neural tube defect
N&V	nausea and vomiting
Ob	obstetrics
OCP	oral contraceptive pill
ODT	oral disintegrating tablet
OHSS	ovarian hyperstimulation syndrome
OP	occiput posterior
OTC	over-the-counter
p.o.	per os (L. by mouth)
p.r.n.	pro re nata (L. as required)
PAC	premature atrial contraction
Paps	Papanicolaou
PATs	premature atrial tachycardia
PCN	penicillin
PCO	polycystic ovary
PDA	patent ductus arteriosus
PE	pulmonary embolism
PEFR	peak expiratory flow rate
PG	phosphatidylglycerol
PGD	prenatal genetic diagnosis
PGE	prostaglandin E
PH	pregnancy hypertension
PID	pelvic inflammatory disease
PIF	prolactin inhibiting factor
PIH	pregnancy-induced hypertension
plts	platelets
PMP	postmenopausal patient
POC	products of conception
POP	pelvic organ prolapse
postop	postoperative
PP	postprandial; placenta previa
ppd	packs per day
PPD	purified protein derivative (TB skin test)
PPH	postpartum hemorrhage
PRBC	packed red blood cells
PROM	premature rupture of membranes

PS	pulmonary stenosis
PST	placental site tumors
pt	patient
PT	plasma thromboplastin
PTB	preterm birth
PTD	preterm delivery
PTL	perinatal telencephalic leukoencephalopathy
PTT	partial thromboplastin time
PTU	propylthiouracil
PUBS	percutaneous umbilical cord sampling
PUD	peptic ulcer disease
PUVA	psoralen plus ultraviolet A
PVC	premature ventricular contraction
PVR	post-voiding residual
PZA	pyrazinamide
q.	quaque (L. every)
q.d.	quaque die (L. every day)
q.i.d.	quater in die (L. four times a day)
R	right
r/o	rule out
RBC	red blood cell
RDA	recommended daily allowance
RDS	respiratory distress syndrome
RF	rheumatic fever
Rh	rhesus
RHIG	RhoGAM immune globulin
RLQ	right lower quadrant
ROM	rupture of membranes
ROP	right occipitoparietal
RPF	renal plasma flow
RPR	rapid plasma reagin
RPW	recurrent pregnancy wastage
RR	respiratory rate
RTO	return to office
RUQ	right upper quadrant
R&V	rectovaginal
Rx	treatment
SAB	spontaneous abortion
SAD	sexual aversion disorder
SBE	self breast examination
SBO	small bowel obstruction
SC	subcutaneous
SCCA	squamous ceil carcinoma
SDLDL	small-density low-density lipoproteins
sed	sedimentation
SERM	selective estrogen receptor modulator
SGA	small for gestational age
SGOT	serum glutamic oxaloacetic transaminase
SHBG	sex hormone binding globulin
SICU	surgical intensive care

sig	sigmoidoscopy
SIRS	systemic inflammatory response syndrome
SLE	systemic lupus erythematosus
S&O	salpingo-oophorectomy
SOB	shortness of breath
SPT	septic thrombophlebitis
SS	somatostatin
SSLF	sacrospinous ligament fixation
SSPE	subacute sclerosing panencephalitis
staph	staphylococcus
STD	sexually transmitted disease
STS	serological test for syphilis
SUI	stress urinary incontinence
SVD	spontaneous vaginal delivery
T	testosterone
T_3	triiodothyronine
T_4	thyroxine
TAB	therapeutic abortion
TAH	total abdominal hysterectomy
TBG	thyroid binding globulin
TC	total cholesterol
TDF	testis-determining factor
TE	thromboembolism
TEF	tracheoesophageal fistula
TENS	transcutaneous electrical nerve stimulation
TG	triglyceride
TIA	transient ischemic attack
TIBC	total iron-binding capacity
t.i.d.	ter in die (L. three times a day)
TIUV	total intrauterine volume
TNF	tumor necrosis factor
TOA	tubo-ovarian abscess
TOL	trial of labor
TPN	total parenteral nutrition
TRH	thyrotropin releasing hormone
Trich	*Trichomonas*
TSH	thyroid stimulating hormone
TSS	toxic shock syndrome
TTTS	twin-twin transfusion syndrome
TV	tidal volume
TVH	total vaginal hysterectomy
TVUS	transvaginal ultrasound
U/A	urinalysis
UADV	umbilical artery Doppler velocimetry
UOP	urine output
URI	upper respiratory infection
US	ultrasound
UVB	ultraviolet B
VAC	vincristine, actinomycin D, cyclophosphamide
VBAC	vaginal birth after Cesarean

VCUG	voiding cystourethrogram
VDRL	venereal disease research laboratory
VLDL	very low-density lipoprotein
VSD	ventricular septal defect
VTE	venous thromboembolism
VZIG	varicella zoster immune globulin
WBC	white blood cell
WNL	within normal limits
wt	weight
yo	year old
ZDV	(or AZT) zidovudine

OBSTETRICS AND GYNAECOLOGY/ REPRODUCTIVE MEDICINE

Informa Healthcare provides a wide range of Obstetrics and Gynaecology journals.

Journal of Obstetrics and Gynaecology

Journal of Obstetrics and Gynaecology represents an established forum for the entire field of obstetrics and gynaecology with an affiliation with the British Society for the Study of Vulval Disease (BSSVD). It publishes a broad range of high quality original, peer-reviewed papers, from scientific and clinical research to reviews relevant to practice and case reports.

www.informaworld.com/cjog

Editors in Chief: Harry Gordon, Alan MacLean

Climacteric

Climacteric is the official Journal of the International Menopause Society with a current Impact Factor of 2.299, ranking it in the top tier of all journals in obstetrics and gynaecology. It has an increased frequency of 6 issues per year.

www.informaworld.com/dcli

Editor in Chiefs: David Sturdee, Alastair MacLennan

The Journal of Maternal-Fetal and Neonatal Medicine

This monthly publication is the official journal of the European Association of Perinatal Medicine, the Federation of Asia and Oceania Perinatal Societies, and the International Society of Perinatal Obstetricians. It publishes a wide range of peer-reviewed original research on the obstetric, medical, genetic, mental health and surgical complications of pregnancy and their effects on the mother, fetus and neonate.

www.informaworld.com/djmf

Editors in Chief: Gian Carlo Di Renzo, Dev Maulik

The European Journal of Contraception and Reproductive Health Care

For over 11 years, ***The European Journal of Contraception and Reproductive Health Care*** is the official journal of the European Society of Contraception. It has published published original peer-reviewed research papers, review papers and other appropriate educational material on all aspects of contraception and reproductive health care.

www.informaworld.com/dejc

Editor in Chief: Jean-Jacques Amy,
Editors: Medard Lech, Dan Apter, Dimitris Lazaris

Journal of Psychosomatic Obstetrics and Gynecology

Journal of Psychosomatic Obstetrics and Gynecology is the official journal of the International Society of Psychosomatic Obstetrics and Gynecology. It provides a scientific forum for gynaecologists, psychiatrists and psychologists as well as for all those who are interested in the psychosocial and psychosomatic aspects of women's health.

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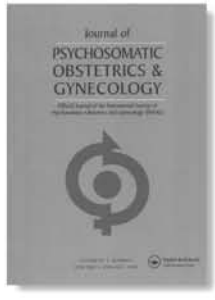

Gynecological Endocrinology

Gynecological Endocrinology, published monthly is the official journal of the International Society of Gynecological Endocrinology. It includes topics relating to the control and function of the different endocrine glands in females, the effects of reproductive events on the endocrine System and the consequences of endocrine disorders on reproduction.

www.informaworld.com/dgye

Editor in Chief: Andrea R. Genazzani

Human Fertility

Human Fertility is the official Journal of the British Fertility Society, the Association of Clinical Embryologists, the British Infertility Counselling Association, the Royal College of Nursing Fertility Nurses Group, and the British Andrology Society. Topics include molecular medicine and healthcare delivery.

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Editor in Chief: Henry Leese

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CLINICAL PROTOCOLS in OBSTETRICS and GYNECOLOGY

THIRD EDITION

The first edition of *Clinical Protocols in Obstetrics and Gynecology* (published in 2000) quickly became known as 'the tan book' and was used as a definitive reference by physicians and other health-care practitioners, residents, and students alike. With the topics in simple alphabetical order, the layout made it easy to locate solutions to everyday clinical problems and to ensure that everyone in an office or hospital team worked consistently. Now enlarged, revised, and updated in a third edition, the book retains and enhances the straightforward layout, flow charts, and simple presentation of relevant statistics that made its previous editions an international bestseller and includes some further illustrations also.

This up-to-date and authoritative Ob/Gyn compilation should help anyone pass the ACOG written or Oral Board examinations and be invaluable for use as a quick-reference while practicing Obstetrics and Gynecology.

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A strong point of this edition is the wide range of topics that are covered. Not only are all the subspecialties represented, but medically related topics, including primary care medicine, are also included.

Fertility and Sterility

There is a lot of groundwork behind these seemingly simple rules… it certainly is a valuable contribution to this particular category of medical literature… It will be of use to specialists in hospitals and private practice, residents, students and the nursing staff.

Acta Obstetricia et Gynecologica Scandinavica

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