Ron Hazani · Mohamed Amir Mrad
David Tauber · Jason Ulm · Alan Yan
Michael J. Yaremchuk *Editors*

Clinical Diagnosis in Plastic Surgery

Springer

Clinical Diagnosis in Plastic Surgery

Ron Hazani
Mohamed Amir Mrad
David Tauber • Jason Ulm
Alan Yan • Michael J. Yaremchuk
Editors

Clinical Diagnosis in Plastic Surgery

Springer

Editors
Ron Hazani
Division of Plastic
and Reconstructive Surgery
Massachusetts General Hospital
Harvard Medical School
Boston, Massachusetts
USA

Mohamed Amir Mrad
Division of Plastic Surgery
Department of Surgery
King Faisal Specialist Hospital
and Research Center
Alfaisal University
Riyadh
Saudi Arabia

David Tauber
Division of Plastic
and Reconstructive Surgery
Craniofacial Plastic Surgery
Massachusetts General Hospital
Harvard Medical School
Boston, Massachusetts
USA

Jason Ulm
Division of Plastic
and Reconstructive Surgery
Craniofacial Plastic Surgery
Massachusetts General Hospital
Harvard Medical School
Boston, Massachusetts
USA

Alan Yan
Division of Plastic
and Reconstructive Surgery
Craniofacial Plastic Surgery
Massachusetts General Hospital
Harvard Medical School
Boston, Massachusetts
USA

Michael J. Yaremchuk
Division of Plastic
and Reconstructive Surgery
Craniofacial Plastic Surgery
Massachusetts General Hospital
Harvard Medical School
Boston, Massachusetts
USA

ISBN 978-3-319-17093-0 ISBN 978-3-319-17094-7 (eBook)
DOI 10.1007/978-3-319-17094-7

Library of Congress Control Number: 2015953157

Springer Cham Heidelberg New York Dordrecht London
© Springer International Publishing Switzerland 2016
This work is subject to copyright. All rights are reserved by the Publisher, whether the whole or part of the material is concerned, specifically the rights of translation, reprinting, reuse of illustrations, recitation, broadcasting, reproduction on microfilms or in any other physical way, and transmission or information storage and retrieval, electronic adaptation, computer software, or by similar or dissimilar methodology now known or hereafter developed.
The use of general descriptive names, registered names, trademarks, service marks, etc. in this publication does not imply, even in the absence of a specific statement, that such names are exempt from the relevant protective laws and regulations and therefore free for general use.
The publisher, the authors and the editors are safe to assume that the advice and information in this book are believed to be true and accurate at the date of publication. Neither the publisher nor the authors or the editors give a warranty, express or implied, with respect to the material contained herein or for any errors or omissions that may have been made.

Printed on acid-free paper

Springer International Publishing AG Switzerland is part of Springer Science+Business Media (www.springer.com)

Preface

Plastic surgery is a visually oriented surgical specialty. Laboratory investigations may be important in preparation for, or recovery from, surgical treatment of a clinical problem – but, most often, are not crucial in making the clinical diagnosis. The majority of diagnoses are made through visual inspection. This atlas is a collection of clinical photographs depicting clinical findings which warrant a plastic surgeon's evaluation and possible surgical intervention. Accompanying each photograph (which we believe is worth a thousand words) are a few words describing the clinical problem including its typical presentation, symptoms, further diagnostic measures, treatment options, and recent references from the literature. Photographs of clinical problems are categorized by anatomic area including the face and facial skeleton, the hand, the integument, the breast, and the trunk.

The Atlas of Plastic Surgical Diagnoses is not only intended to benefit the patients of plastic surgeons in training, but also those who present to other medical and surgical practitioners for diagnosis and appropriate referral.

The authors are grateful for the many contributions to this Atlas from both the staff and residents of the Harvard Plastic Surgery Training Program. We are especially indebted to faculty members Amir Taghinia, MD; Arin Greene, MD; Bonnie Padwa, DMD, MD; Brian Labow, MD; Sam Lin, MD; Eric Liao, MD; and Simon Talbot MD. We are especially grateful to Professor Nivaldo Alonso, MD (Faculdade de Medicina de Universidade de Sao Paulo), who graciously shared his extraordinary collection of craniofacial deformities gleaned from his extraordinary clinical experience.

Boston, MA, USA — Ron Hazani, MD
Riyadh, Saudi Arabia — Mohamed Amir Mrad, MD
Boston, Massachusetts, USA — David Tauber, MD
Boston, Massachusetts, USA — Jason Ulm, MD
Boston, Massachusetts, USA — Alan Yan, MD
Boston, Massachusetts, USA — Michael J. Yaremchuk, MD

To my father...
Whose strength, courage, wisdom, love and support guided me to where I am today.

Mohamed Amir Mrad, MD

Contents

1 Craniofacial 1
Mohamed Amir Mrad, Jason Ulm, Michael J. Yaremchuk, Alan Yan, W. McIver Leppard, Angel E. Rivera-Barrios, Jennifer Dixon Swartz, Saad Al-Kharsa, Oubai Alhafez, Arin Greene, Samuel Lin, Nivaldo Alonso, and Ron Hazani

2 Hand 69
Ron Hazani, Alan Yan, Michael J. Yaremchuk, Amir Taghinia, Jesse Jupiter, Simon Talbot, Fuad Hashem, Camela A. Pokhrel, Arun J. Rao, and Angel E. Rivera-Barrios

3 Breast 107
Jason Ulm, Mohamed Amir Mrad, Alan Yan, Michael J. Yaremchuk, Eric Liao, Fuad Hashem, and Brian Labow

4 Integument 127
David Tauber, Alan Yan, Michael J. Yaremchuk, Fuad Hashem, Ellen Roh, Mohamed Amir Mrad, Arin Greene, John Mullen, Moath Alhakami, Khalid Murrad, Tahira I. Prendergast, and W. McIver Leppard

Contributors

Oubai Alhafez, BDS, MS Al-Kharsa Orthodontics Private Practice, Riyadh, Saudi Arabia

Moath Alhakami, MD Division of Plastic and Reconstructive Surgery, King Faisal Specialist Hospital, Riyadh, Saudi Arabia

Saad Al-Kharsa, DDS, MS Al-Kharsa Orthodontics Private Practice, Riyadh, Saudi Arabia

Nivaldo Alonso, MD Division of Plastic Surgery, Livre Docente FMUSP, San Paulo, Brazil

Arin Greene, MD Division of Plastic Surgery, Children's Hospital of Boston, Harvard Medical School, Boston, MA, USA

Fuad Hashem, MD Department of Plastic Surgery, Department of Plastic Surgery, King Faisal Specialist Hospital, Riyadh, Saudi Arabia

Ron Hazani, MD Division of Plastic and Reconstructive Surgery, Massachusetts General Hospital, Harvard Medical School, Boston, Massachusetts, USA

Jesse Jupiter, MD Massachusetts General Hospital, Harvard Medical School, Boston, MA, USA

Brian Labow, MD Department of Plastic and Oral Surgery, Children's Hospital of Boston, Harvard Medical School, Boston, MA, USA

W. McIver Leppard, MD Division of Plastic and Reconstructive Surgery, Department of Plastic Surgery, Medical University of South Carolina, Charleston, SC, USA

Eric Liao, MD Division of Plastic Surgery, Department of Surgery, Massachusetts General Hospital, Harvard Medical School, Boston, MA, USA

Samuel Lin, MD Division of Plastic Surgery, Beth-Israel Deaconess Medical Center, Harvard Medical School, Boston, MA, USA

John Mullen, MD Massachusetts General Hospital, Harvard Medical School, Boston, MA, USA

Mohamed Amir Mrad, MD Division of Plastic Surgery, Department of Surgery, King Faisal Specialist Hospital and Research Center, Alfaisal University, Riyadh, Saudi Arabia

Khalid Murrad King Saud University, Riyadh, Saudi Arabia

Camela A. Pokhrel, MD Division of Plastic Surgery, Department of Surgery, Kleinert-Kutz Hand Surgery, Louisville, KY, USA

Tahira I. Prendergast, MD Division of Plastic and Reconstructive Surgery, Medical University of South Carolina, Charleston, SC, USA

Arun J. Rao, MD Division of Plastic Surgery, Department of Surgery, Private Practice, Tucson, AZ, USA

Angel E. Rivera-Barrios, MD Division of Plastic Surgery, Department of Surgery, Medical University of South Carolina, Charleston, SC, USA

Ellen Roh, MD Massachusetts General Hospital, Harvard Medical School, Boston, MA, USA

Jennifer Dixon Swartz, MD Division of Plastic and Reconstructive Surgery, Medical University of South Carolina, Charleston, SC, USA

Amir Taghinia, MD Division of Plastic Surgery, Department of Surgery, Children's Hospital of Boston, Harvard Medical School, Boston, MA, USA

Simon Talbot, MD Division of Plastic Surgery, Department of Surgery, Children's Brigham and Women's Hospital, Harvard Medical School, Boston, MA, USA

David Tauber, MD Division of Plastic and Reconstructive Surgery, Craniofacial Plastic Surgery, Massachusetts General Hospital, Harvard Medical School, Boston, Massachusetts, USA

Jason Ulm, MD Division of Plastic and Reconstructive Surgery, Craniofacial Plastic Surgery, Massachusetts General Hospital, Harvard Medical School, Boston, Massachusetts, USA

Alan Yan, MD Division of Plastic and Reconstructive Surgery, Craniofacial Plastic Surgery, Massachusetts General Hospital, Harvard Medical School, Boston, Massachusetts, USA

Michael J. Yaremchuk, MD Division of Plastic and Reconstructive Surgery, Craniofacial Plastic Surgery, Massachusetts General Hospital, Harvard Medical School, Boston, Massachusetts, USA

John Meara, MD Division of Plastic Surgery, Department of Surgery, Massachusetts General Hospital, Harvard Medical School, Boston, MA, USA

Craniofacial

1

Mohamed Amir Mrad, Jason Ulm, Michael J. Yaremchuk, Alan Yan, W. McIver Leppard, Angel E. Rivera-Barrios, Jennifer Dixon Swartz, Saad Al-Kharsa, Oubai Alhafez, Arin Greene, Samuel Lin, Nivaldo Alonso, and Ron Hazani

1.1 Craniofacial, Congenital

1.1.1 Deformational Plagiocephaly

(Plagiocephaly: Derived from the Greek words plagios (oblique or slant) and kephale (head)).

M.A. Mrad, MD (✉)
Division of Plastic Surgery, Department of Surgery, King Faisal Specialist Hospital and Research Center, Alfaisal University, Riyadh, Saudi Arabia
e-mail: mmrad@kfshrc.edu.sa
http://www.drmrad.com

J. Ulm, MD • M.J. Yaremchuk, MD • A. Yan, MD
Division of Plastic and Reconstructive Surgery, Craniofacial Plastic Surgery, Massachusetts General Hospital, Harvard Medical School, Boston, Massachusetts, USA
e-mail: jasonulm@gmail.com; http://www.dryaremchuk.com; alanyanmd@gmail.com

W.M. Leppard, MD • A.E. Rivera-Barrios, MD
J.D. Swartz, MD
Division of Plastic and Reconstructive Surgery, Department of Plastic Surgery, Medical University of South Carolina, Charleston, SC, USA
e-mail: leppardwm@gmail.com; Barian17@hotmail.com; Jendixon.swartz@gmail.com

S. Al-Kharsa, DDS, MS • O. Alhafez, BDS, MS
Al-Kharsa Orthodontics Private Practice, Riyadh, Saudi Arabia
e-mail: info@kharsaortho.com; oubai_alhafez@hotmail.com
http://www.kharsaortho.com

A. Greene, MD
Department of Plastic Surgery, Children's Hospital of Boston, Harvard Medical School, Boston, MA, USA
e-mail: arin.greene@childrens.harvard.edu

S. Lin, MD
Department of Plastic Surgery, Beth-Israel Deaconess Medical Center, Harvard Medical School, Boston, MA, USA
e-mail: sjlin@bidmc.harvard.edu

N. Alonso, MD
Department of Plastic Surgery, Livre Docente FMUSP, San Paulo, Brazil
e-mail: nivalonso@gmail.com

R. Hazani, MD
Division of Plastic and Reconstructive Surgery, Massachusetts General Hospital, Harvard Medical School, Boston, Massachusetts, USA
e-mail: info@drhazani.com
http://www.drhazani.com

© Springer International Publishing Switzerland 2016

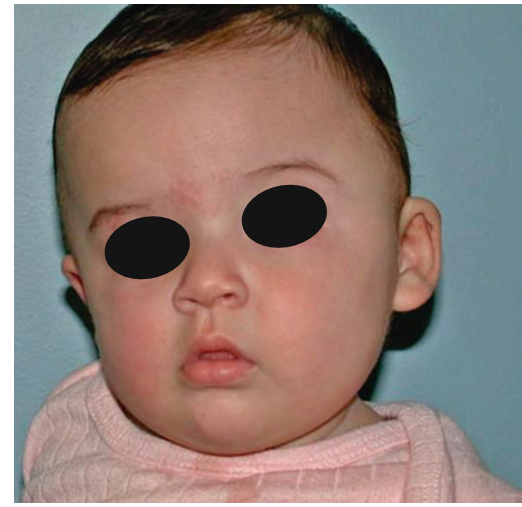
R. Hazani et al. (eds.), *Clinical Diagnosis in Plastic Surgery*, DOI 10.1007/978-3-319-17094-7_1

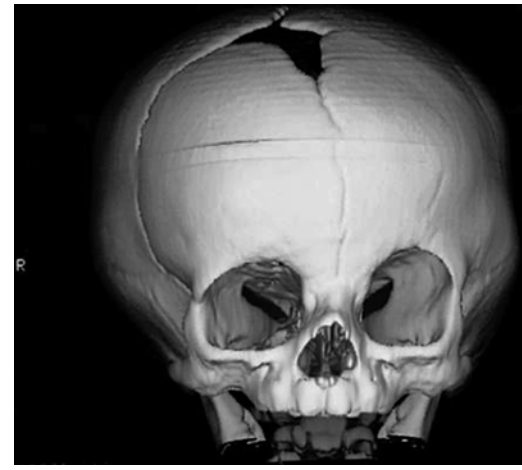
History Patient presents with an acquired skull deformity, as a result of being placed in a fixed supine position for sleep.

Exam Unilateral features noted on physical examination include occipital flattening; anterior displacement of the ipsilateral ear, forehead, and zygoma; and widening of the ipsilateral palpebral fissure, causing a parallelogram-shaped cranium.

Treatment If diagnosed early, treatment that involves repeatedly repositioning the child out of the flat spot will be sufficient. Cranial-molding helmets are used for more severe cases or for those who are diagnosed late.

1.1.2 Diagnosis: Anterior Plagiocephaly (Unilateral Coronal Synostosis (UCS))



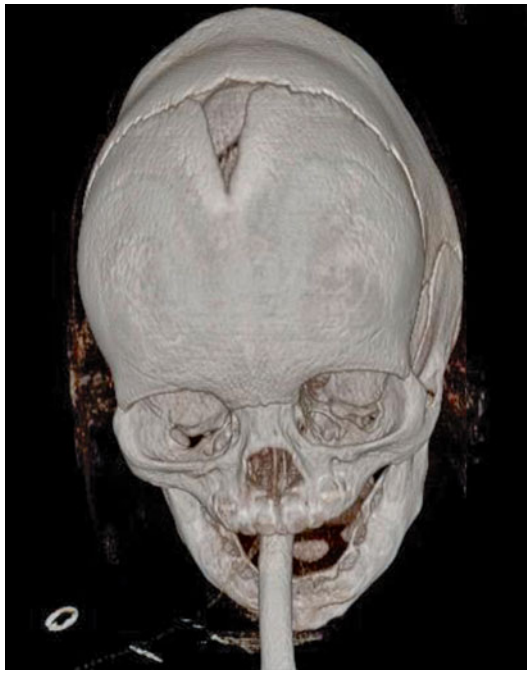


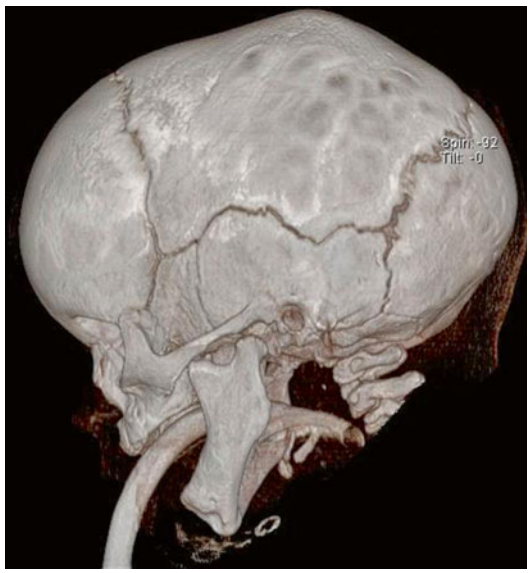
History Patients present with progressive facial deformities that are not seen with other nonsyndromic craniosynostosis.

Exam Anterior plagiocephaly is the result of unilateral coronal synostosis, characterized by superior and posterior displacement of the supraorbital rim and eyebrow on the ipsilateral side, widening of the ipsilateral palpebral fissure, frontal bossing of the contralateral side, deviation of the nasal root toward the affected side, occipital protrusion of the ipsilateral side, and flattening of the contralateral occiput.

Treatment Surgical intervention entails release of the synostosed suture with fronto-orbital advancement, usually between 3 and 6 months of age.

1.1.3 Diagnosis: Scaphocephaly (Sagittal Synostosis)



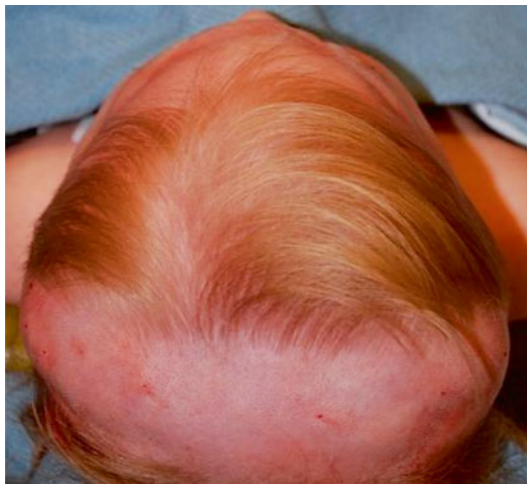


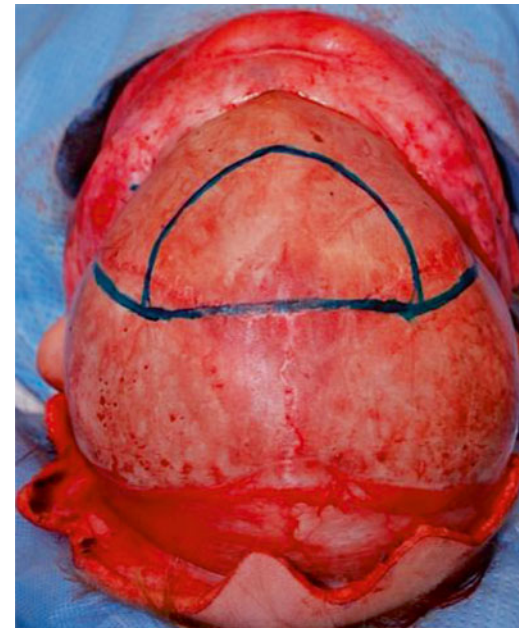
History Patients present with boat-shaped skull deformity, hence the term scaphocephaly. It is the most frequent nonsyndromic synostoses with a male predominance of 4:1.

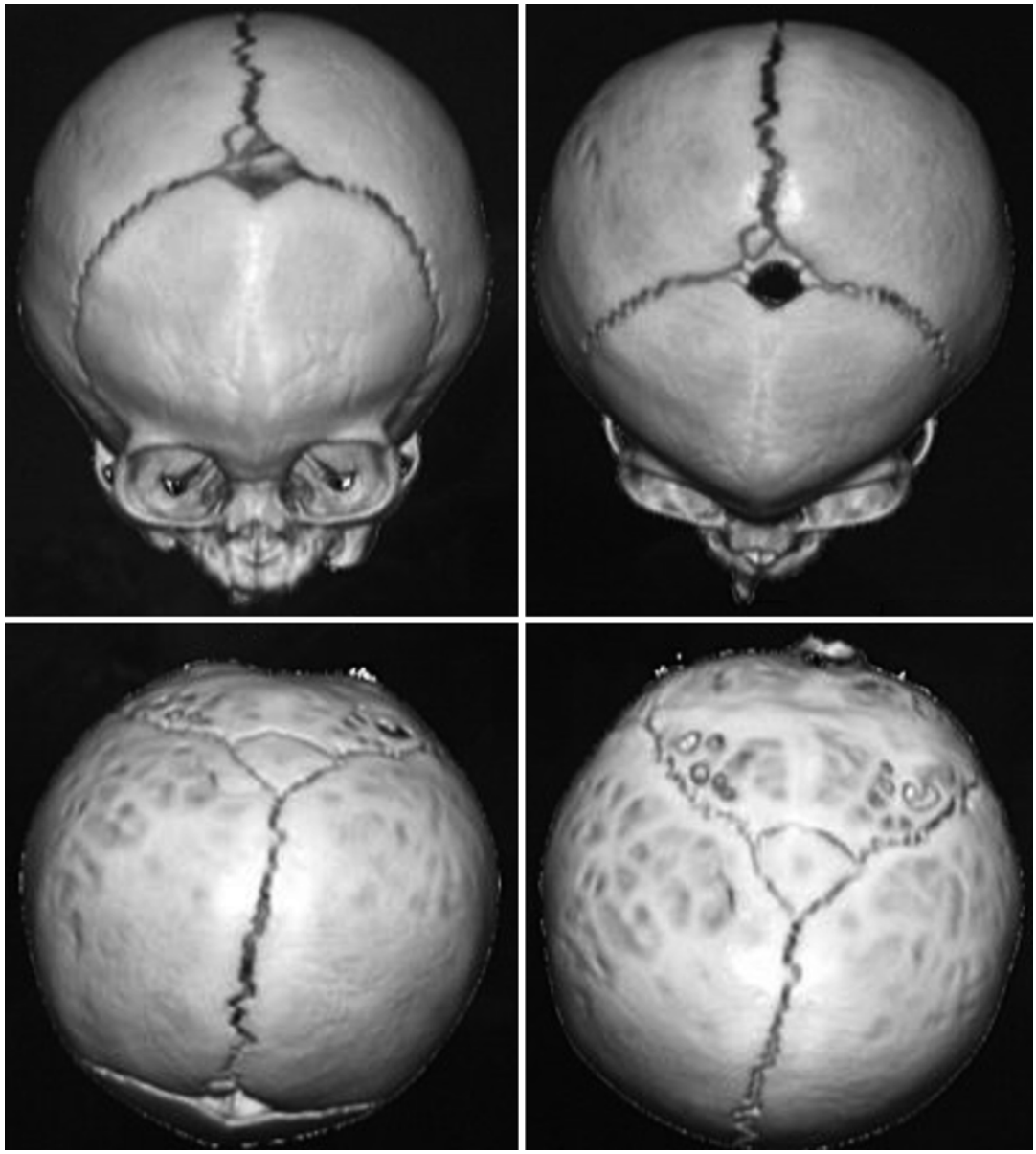
Exam The fusion of the sagittal suture impairs expansion of the skull width. The cranium is therefore long, narrow, and keel-shaped. This is accompanied by frontal and occipital bossing.

Treatment Cranial vault remodeling with barrel-staving technique and subtotal cranial vault remodeling are the main surgical options. Endoscopic correction has been described in patients between 2 and 4 months of age.

1.1.4 Diagnosis: Trigonocephaly (Metopic Synostosis)





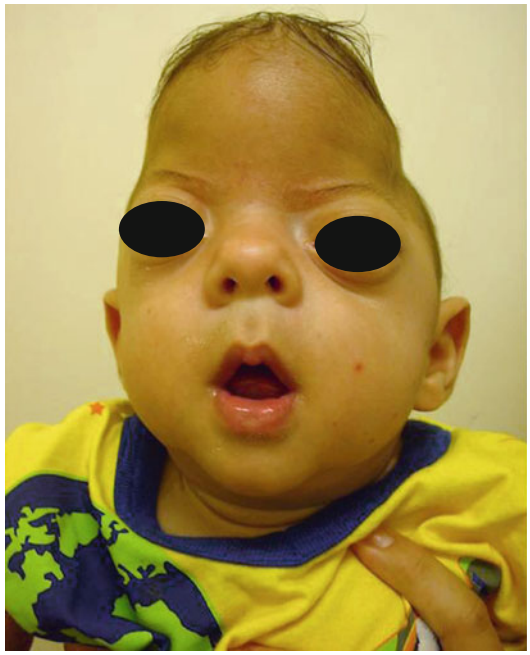


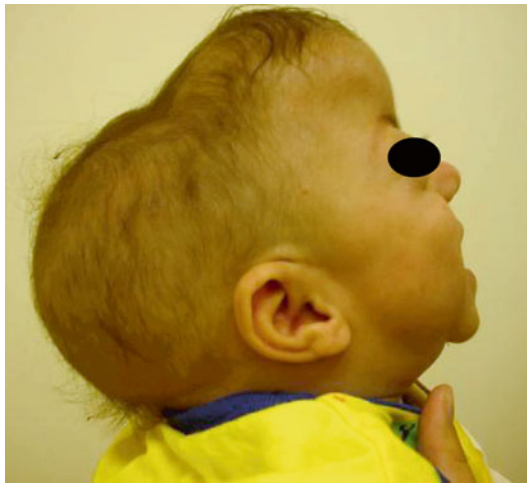
History Patients present with triangular-shaped skull deformity when viewed from above, hence the term trigonocephaly. Metopic synostosis represents the third most common nonsyndromic synostosis with male predominance.

Exam Physical examination shows a midfrontal keel with bitemporal narrowing and orbital hypotelorism, epicanthal folds, and low nasal dorsum.

Treatment The risk for congenital and behavioral impairment is greater in metopic synostosis than in other nonsyndromic synostosis. Surgical treatment includes bifrontal craniotomy, frontal reshaping and radial osteotomies, and frontoorbital advancement.

1.1.5 Diagnosis: Kleeblattschadel Skull Deformity



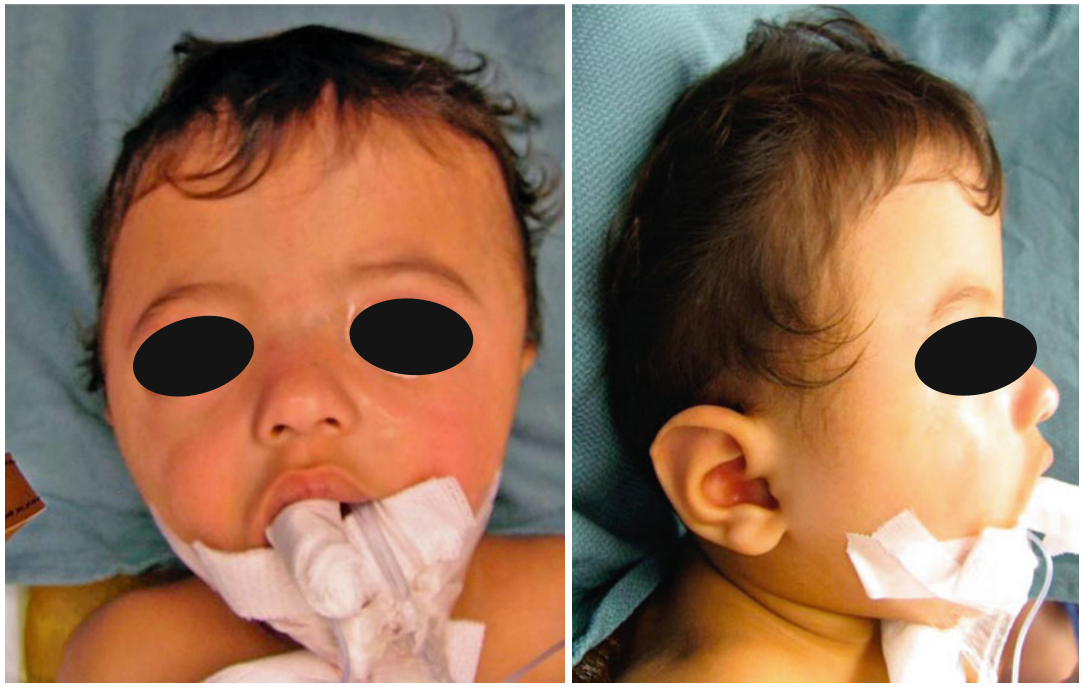


History Patient presents with a cloverleaf-shaped skull deformity (Kleeblattschadel).

Exam Kleeblattschadel skull deformity is a result of multiple-suture craniosynostoses characterized by frontal bossing, bitemporal bulging, orbital exposure, and downward displacement of the ears. Increase in intracranial pressure results in visual compromise, venous hypertension, and hindbrain herniation.

Treatment Staged calvarial decompression and remodeling are the treatment of choice.

1.1.6 Diagnosis: Turricephaly (or Oxycephaly)



History Patients present with a tall, tower-shaped skull.

Exam The vertically tall head shape, accompanied by short anterior-posterior dimension, is a result of bicoronal syndromic craniosynostoses such as Apert, Crouzon, or Pfeiffer syndromes. Other clinical findings include elevated intracranial pressure, hydrocephalus, Chiari malformations, and ocular exposure.

Treatment Treatment is typically addressed before 12 months of age with resection of the suture, fronto-orbital advancement, and cranial vault remodeling, though other techniques have been described. Staged procedures may be required.

1.1.7 Diagnosis: Apert Syndrome

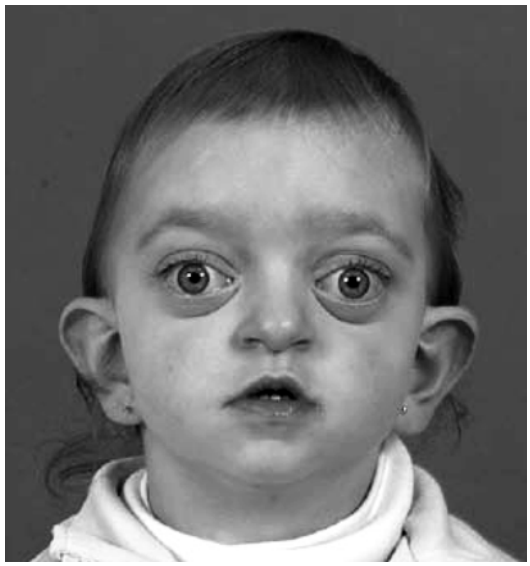
History It is an autosomal dominant syndrome with incidence of 1:100,000 caused by mutations in fibroblast growth factor gene 2 (FGFR2).

Exam Physical findings show significant turribrachycephaly, proptosis, hypertelorism, and down slanting of palpebral fissures. The midface hypoplasia is accompanied by depressed nasal dorsum and septal deviation and acne. Patients also present with complex syndactyly of the hands and feet. Mental impairment is common with a high likelihood of increased ICP and cerebral palsy.

Treatment Treatment is typically addressed before 12 months of age with resection of the suture, fronto-orbital advancement, and cranial vault remodeling. Airway and visual compromise may prompt for emergent treatment. Le Fort III advancement of the midface is often performed at 6–8 years of age.

The craniofacial dysostosis syndromes have several common features: brachycephaly (premature fusion of the coronal sutures), variable severity midface hypoplasia, variable involvement of other cranial sutures, and identification of the responsible genetic mutations. Some have characteristic extremity deformities.

1.1.8 Diagnosis: Crouzon Syndrome

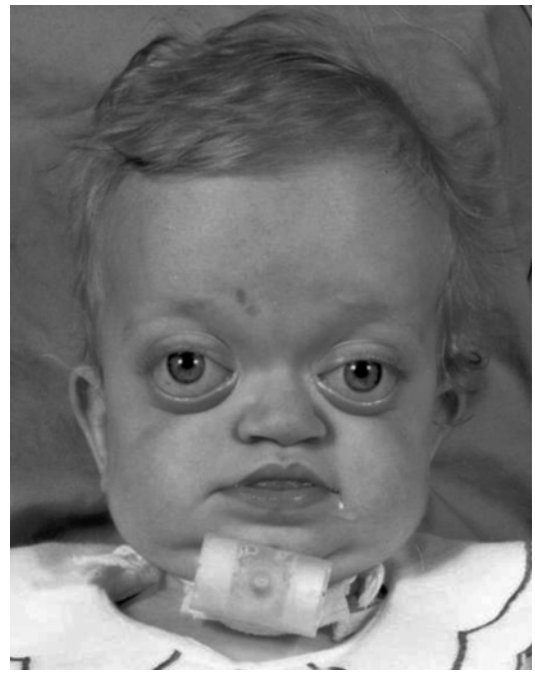


History It is an autosomal dominant syndrome with an incidence of 1:25,000, as a result of multiple mutations in the fibroblast growth factor receptor 2 (FGFR-2) gene.

Exam Characterized by a brachycephaly due to premature fusion of both coronal sutures, Crouzon syndrome is a frequent form of craniofacial dysostosis. Other cranial sutures may also be involved. There is also midface hypoplasia, exorbitism, and proptosis. *The extremities are normal*. These patients may also exhibit mild mental status impairment, hydrocephalus, and elevated intracranial pressure in almost half of the cases.

Treatment Surgical treatment is typically addressed before 12 months of age with resection of the suture, fronto-orbital advancement, and cranial vault remodeling. Airway and visual compromise may dictate emergent treatment. Le Fort III advancement of the midface is often performed at 6–8 years of age.

1.1.9 Diagnosis: Pfeiffer Syndrome

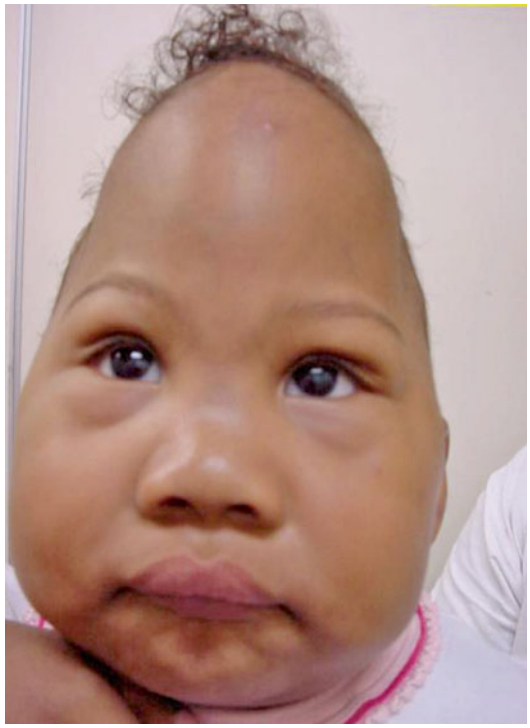


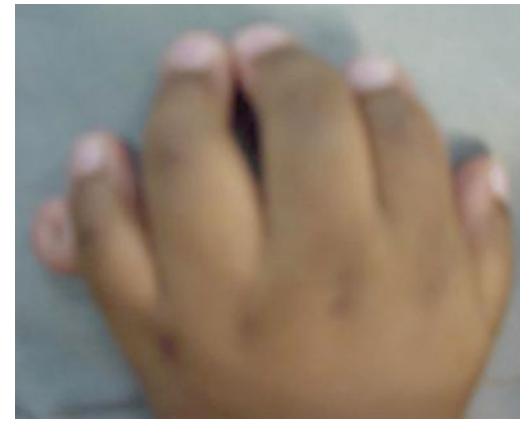
History An autosomal dominant craniosynostosis syndrome. Patients present with normal mental status in the majority of the cases.

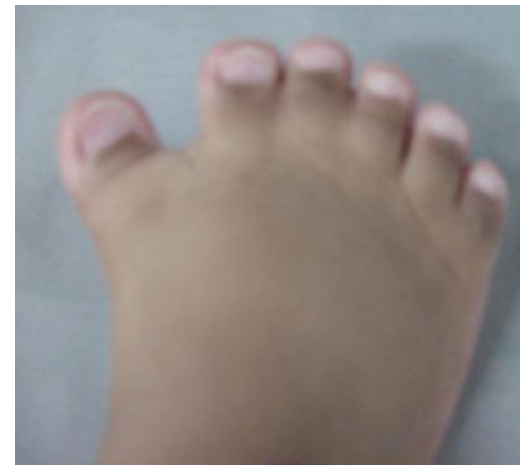
Exam Clinical findings include turribrachycephaly with coronal or sagittal synostosis. Orbital and midface features are similar to Apert syndrome with an additional distinct findings of broad thumbs and halluces, mild simple syndactyly, and tracheal cartilage anomalies.

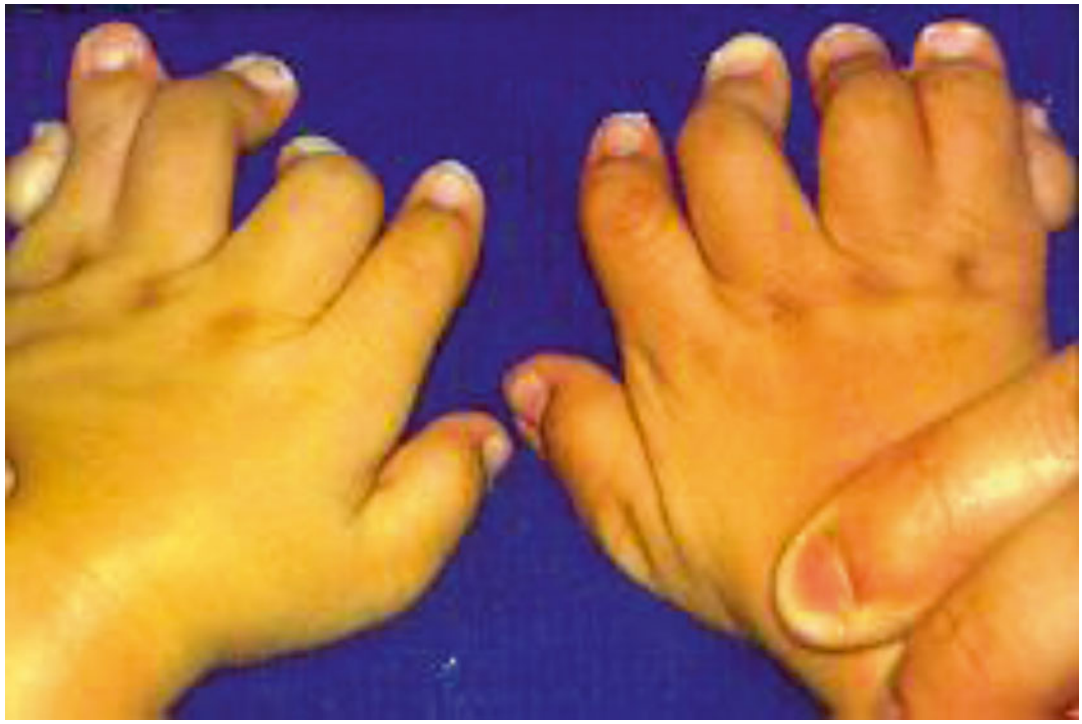
Treatment Cranial vault remodeling is usually performed before 18 months of age. Le Fort III advancement is indicated for those with worsening nasopharyngeal airway obstruction.

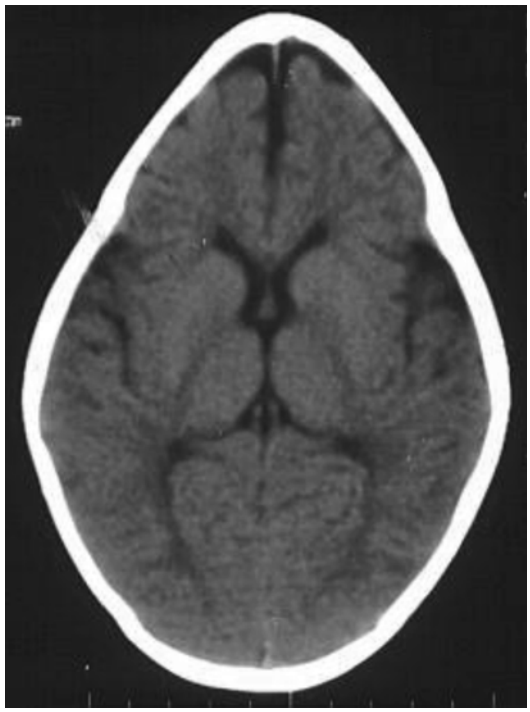
1.1.10 Diagnosis: Carpenter Syndrome











History Carpenter syndrome, also known as acrocephalopolysyndactyly type II, is a rare congenital disorder characterized by craniosynostosis and musculoskeletal abnormalities. Unlike other acrocephalosyndactylies, it is autosomal recessive and has recently been linked to mutations in RAB23 and the hedgehog signaling pathway.

Exam Craniosynostosis patterns in Carpenter syndrome are variable but often involve lambdoid, sagittal, and metopic sutures, the latter resulting in hypotelorism. There is underdevelopment of the anterior cranial fossa and expansion of the middle cranial fossa giving the classic "diamond-shaped" face originally described by Dr. Carpenter in 1909. Common limb anomalies include syndactyly and polydactyly of the hands and feet but can also include clinodactyly and brachydactyly.

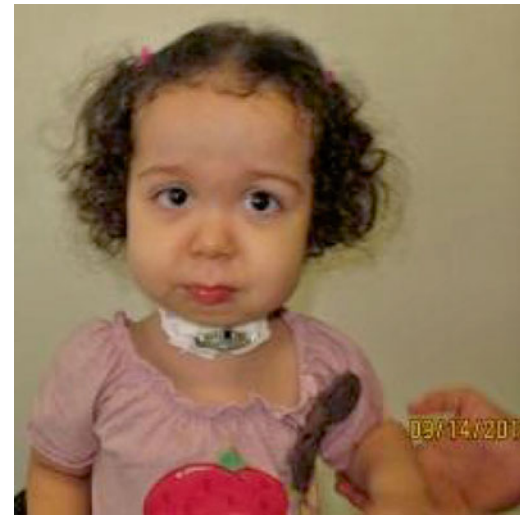
Treatment Treatment of Carpenter syndrome is similar to other craniosynostotic syndromes and includes cranial vault remodeling usually between 3 and 9 months of age. Limb anomalies should also be treated as necessary as the child develops.

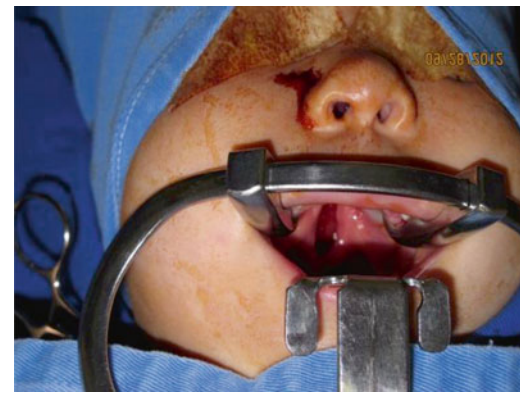
Perlyn, C.A., Marsh, J.L.. Craniofacial Dysmorphology of Carpenter Syndrome: Lessons from Three Affected Siblings. Plast Reconsr Surg. March 2008; 121: 971.

Cottrill C., et al. Carpenter Syndrome. The Journal of Craniofacial Surgery. January 2009 20(1): 254–256

Carpenter, G. Case of acrocephaly with other congenital malformations. Proc R Soc Med Sect Study Dis Child. 1909; 2:45–53

1.1.11 Diagnosis: Stickler Syndrome



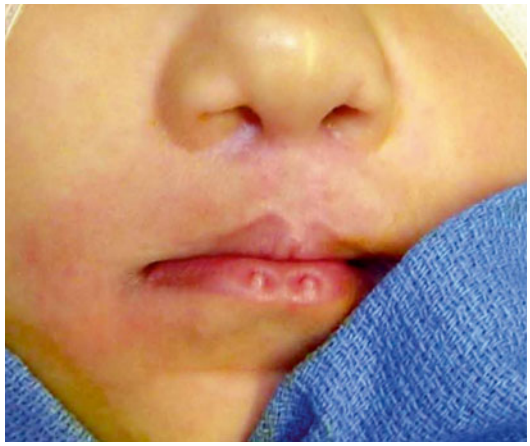


History Progressive autosomal dominant connective tissue disorder (collagen) with variable penetrance. It is the most common genetic syndrome associated with Pierre Robin sequence ~1–3/10,000 births.

Exam Can exhibit hypotonia, micrognathia, flat facies with mild to moderate midface hypoplasia, shallow orbits with proptosis, cleft of secondary palate, flat nasal bridge with epicanthal folds, hypermobility of joints with enlarged wrist/knee/ankle joints, scoliosis, mitral valve prolapse, hearing loss (conductive/sensorineural/mixed), retinal detachment, glaucoma, cataracts, and strabismus.

Treatment Airway maintenance for micrognathia, vigilant ophthalmic and otologic examinations for treatment of above, cleft palate repair, maxillofacial surgery, and orthodontics as needed.

1.1.12 Diagnosis: Van der Woude Syndrome

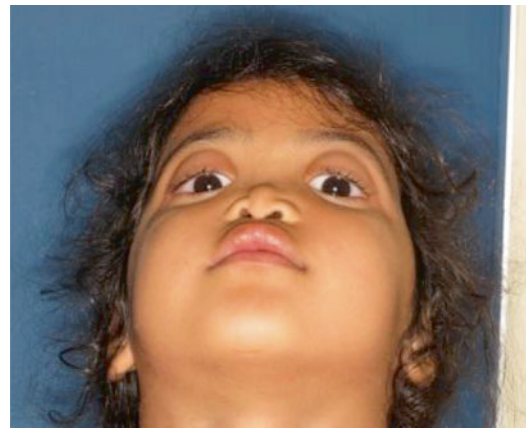


History Autosomal dominant with variable penetrance that has been linked to a mutation of interferon regulatory factor 6 (IRF6) gene on chromosome 1.

Exam Usually symmetrical bilateral paramedian sinuses or depressions of the vermilion lower lip (lip pits), possible tongue tie (short lingual frenulum), cleft lip, palate, or submucous cleft palate.

Treatment Direct excision of sinus pits and treatment of associated cleft.

1.1.13 Diagnosis: Binder Syndrome (Maxillonasal Dysplasia)

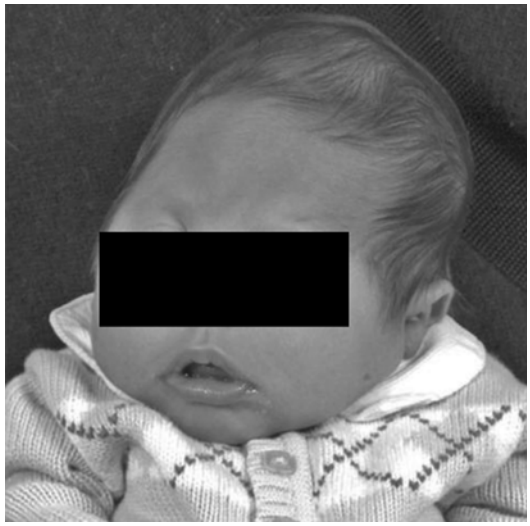


History It is generally a sporadically occurring syndrome characterized by varying degrees of nasomaxillary hypoplasia, absent anterior nasal spine, and small frontal sinuses.

Exam Midface hypoplasia; obtuse frontonasal angle; a short, vertical nose with acute nasolabial angle; and perialar and nasal tip flattening. Patient may also have class III occlusion with an anterior open bite.

Treatment Staged reconstruction during adolescence, including orthodontic treatment, possibly orthognathic surgery (Le Fort I), and eventual nasal reconstruction.

1.1.14 Diagnosis: Saethre-Chotzen (Acrocephalosyndactyly Type III)

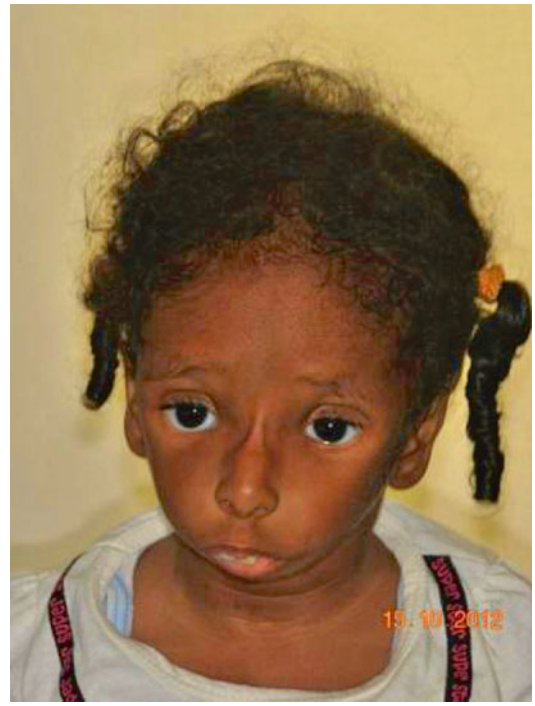


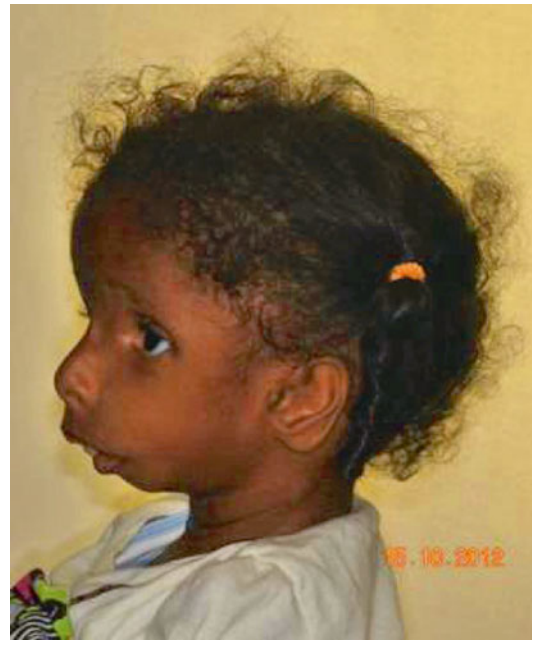
History An autosomal dominant syndrome with an incidence of 1:25,000–50,000. Patients usually present with normal mental status.

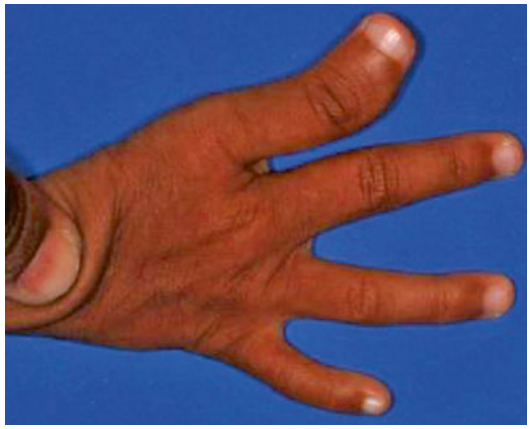
Exam Asymmetric brachycephaly, ptosis of the eyelids, antimongoloid slanting of palpebral fissures, midface hypoplasia, narrow palate, low-set hairline, and partial syndactyly.

Treatment Cranial vault remodeling with fronto-orbital advancement is the mainstay of treatment. Patients with syndactyly will also require syndactyly release.

1.1.15 Diagnosis: Nager Syndrome





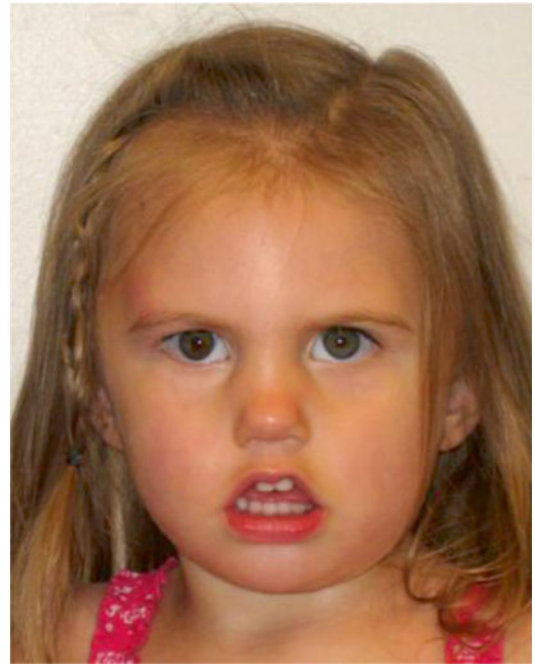


History A rare and sporadic congenital condition that involves preaxial upper limb abnormalities and mandibulofacial dysostosis.

Exam Similar craniofacial findings to Treacher Collins syndrome: mandibular, malar, and maxillary hypoplasia, colobomas, downslanting palpebral fissures, absent eyelashes medially, low-set malformed ears, and palatal defects. Airway compromise due to micrognathia and glossoptosis may occur, as well as velopharyngeal incompetence and conductive hearing loss. Although preaxial abnormalities (hypoplastic thumb/radius) are described classically, a variety to hand anomalies may occur, as well as urogenital anomalies. Patients usually have normal intelligence.

Treatment Initial management includes assessment and management of the airway (positioning, lip-tongue adhesion, tracheostomy). Management of the craniofacial anomalies may include repair of the palate, mandibular distraction osteogenesis, midface reconstruction with bone grafts or custom implants, and ear reconstruction. Directed surgical correction of the upper limb and urogenital anomalies is also done.

1.1.16 Diagnosis: Möbius Syndrome

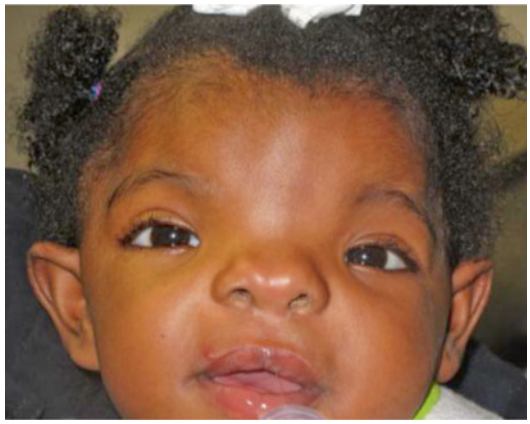


History Möbius syndrome is a rare congenital disorder characterized by a variety of cranial nerve (CN) defects, usually the facial and abducens cranial nerve paralysis. The syndrome is usually bilateral. However, unilateral cases have been described.

Exam Diagnosis is based on physical examination alone and depends on the cranial nerve involved. As CN VI and VII are the most involved, the majority of these patients display the absence of facial expression with an inability to smile and lack of eye movement. A masked appearance, especially while crying, is typical of this syndrome.

Treatment Traditionally, local muscle transfers involving the masseter, temporalis, and the platysma have been used to restore functions lost due to facial paralysis. More recently, transfer of free neurovascularized muscle such as the free gracilis muscle transfer has been used, and it has become first line of treatment to restore partial function of the facial nerve allowing for the ability to smile, adequate excursion of the commissure, oral competence, and improved speech.

1.1.17 Diagnosis: Frontonasal Dysplasia/Median Cleft Face Syndrome

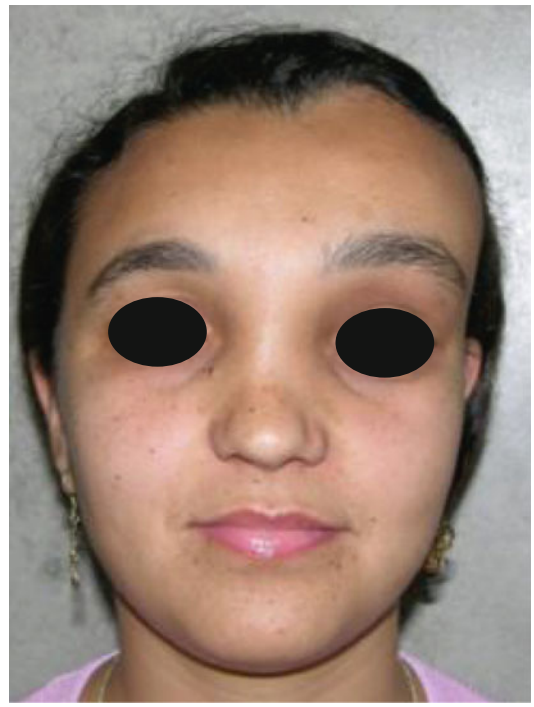


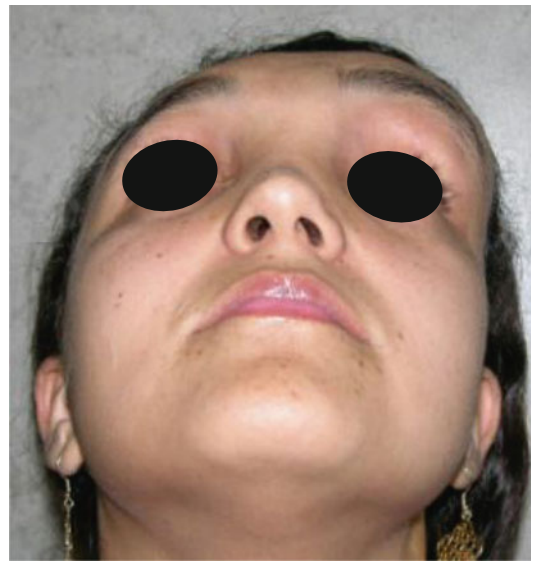
History Congenital malformation with variable degrees of severity of Tessier 0/14 median facial cleft; may be associated with other syndromes.

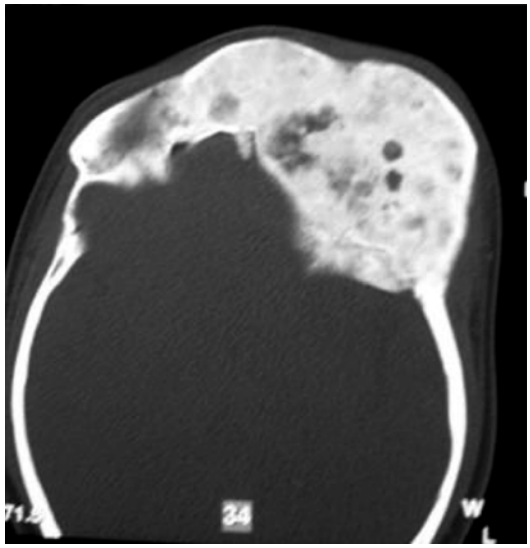
Exam Orbital hypertelorism, broad nasal bridge, cleft lip/palate/maxilla, bifid nose, +/– other associated anomalies (encephalocele, absent corpus callosum, ocular abnormalities, hearing loss).

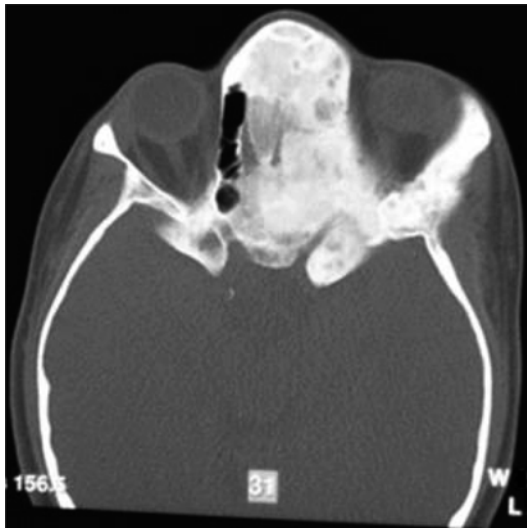
Treatment Surgical treatment of hypertelorism, nasal abnormalities, and cleft lip/palate as appropriate.

1.1.18 Fibrous Dysplasia







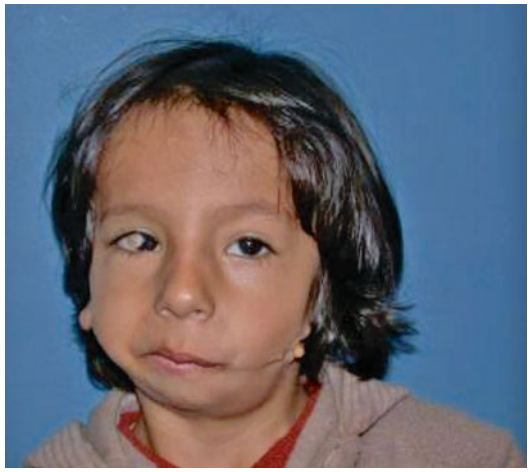


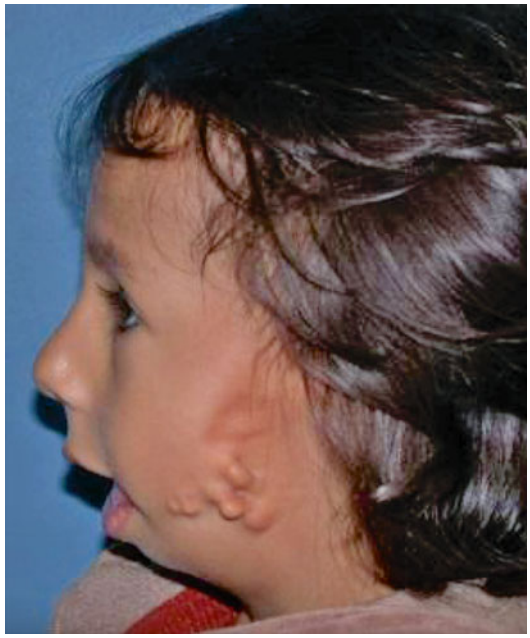
History Patient presents with craniomaxillofacial or axial bony abnormalities.

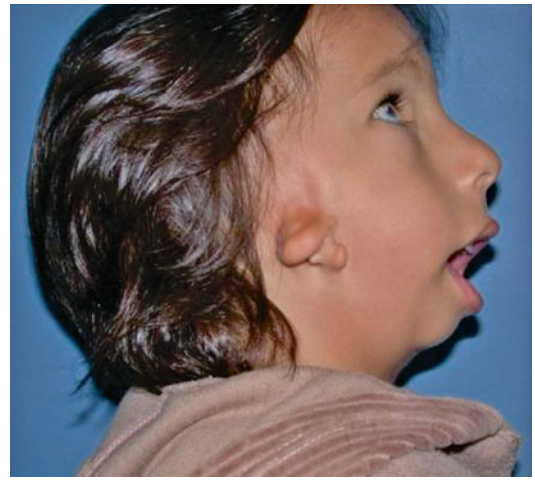
Exam It is a progressive benign bone disorder that occurs sporadically, often presenting in late childhood or adolescence. It may be classified as monostotic, polyostotic, or a part of McCune-Albright syndrome (polyostotic fibrous dysplasia, precocious puberty, skin pigmentation). Histologically, normal bone is replaced with an unorganized mixture of fibrous connective tissue and structurally weak trabecular bone. For craniomaxillofacial involvement, thorough head and neck, ocular, and otologic examinations are performed. CT face will demonstrate a ground glass appearance of woven bone.

Treatment As this is a benign and unpredictable process, surgical management must consider the functional and aesthetic concerns of the area involved, skeletal maturity of the patient, and type of disease process (mono- vs. polyostotic). Complete resection is not always mandated, particularly if vital structures are not compromised. If subtotal resection is done, long-term monitoring is warranted, especially if the orbit is involved. Threatened optic nerves require orbital decompression. Reconstruction is tailored toward the nature of the defect and includes free tissue transfer, autologous bone grafts, as well as alloplastic materials in select cases.

1.1.19 Diagnosis: Goldenhar Syndrome







History Patients present with asymmetry of the bony and soft tissues of the face.

Exam Also known as oculo-auriculo-vertebral (OAV) syndrome, this rare congenital abnormality is a subset of hemifacial microsomia and is a result of anomalous development of the first and second branchial arch. It is characterized by epibulbar dermoid/lipodermoid/colobomas, preauricular skin tags, unilateral facial hypoplasia, unilateral macrostomia, vertebral anomalies, mandibular/maxillary hypoplasia, and/or abnormalities of internal organs.

Treatment Patients with Goldenhar syndrome should be treated in a multistage, multidisciplinary fashion with a long-term plan. Although there is no consensus on the methods of surgical treatment, priority should be given to deficits causing functional problems such as airway compromise in maxillary/mandibular hypoplasia. Commonly employed surgical options include costochondral bone grafts or classic osteotomy.

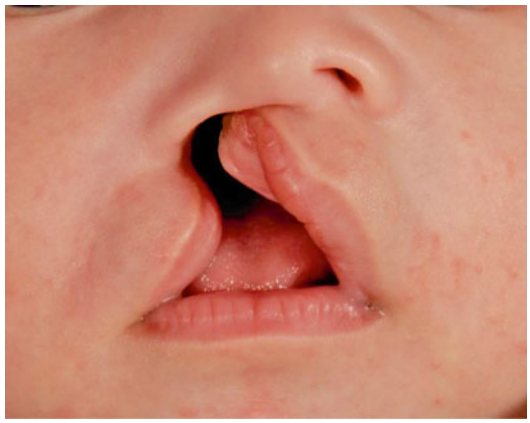
1.1.20 Diagnosis: Progressive Hemifacial Atrophy/Romberg Disease/Parry-Romberg Syndrome

History Patient presents with progressive hemifacial atrophy in the first or early second decade of life.

Exam It is a rare neurocutaneous syndrome characterized by progressive shrinkage and degeneration of progressive atrophy of skin and subcutaneous tissue within the dermatome of one of the branches of the trigeminal nerve on unilateral face (95 %) but can occasionally extend to other parts of the body. Findings include a circumscribed patch of frontal scleroderma with hair loss and a depressed linear scar extending down through the midface on the affected side, hence the term "coup de sabre."

Treatment There is no adequate medical treatment for Parry-Romberg syndrome. After it is allowed to run its course, reconstruction options include fat injection, soft tissue augmentation with free tissue transfer, bone grafting, and orthognathic surgery as indicated.

1.1.21 Diagnosis: Unilateral Cleft Lip

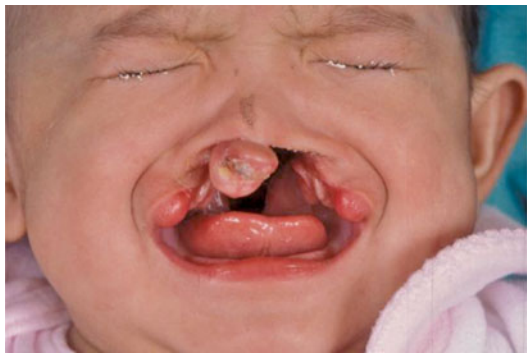


History A cleft lip represents a deformity affecting the projection and outward rotation of the premaxilla with abnormal insertion of the orbicularis muscle. The incidence of cleft lip, with or without a cleft palate, is higher among Asians, males, and is most frequently found on the left side. Cleft lips are associated with a syndrome in only 15 % of the cases.

Exam A unilateral complete cleft lip is characterized by a disruption of the lip, nostril sill, and alveolus. A unilateral incomplete cleft lip does not extend through the entire lip to the nasal floor (Simonart band). An abnormal attachment of the orbicularis oris muscles is present on both sides of the cleft. The vertical height of the lip is decreased. On the non-cleft side, the philtral column, philtral dimple, and two-thirds of Cupid's bow are preserved.

Treatment A multidisciplinary approach is needed for the care of the patient with cleft lip. Children with this condition should be assessed shortly after birth, and parents should be counseled before birth if cleft lip is suspected. Special feeding bottles and nipples are needed by the patient to ensure adequate growth and weight gain. Presurgical orthopedics such as a Latham device or nasoalvelolar molding (NAM) can be used to narrow and align cleft segments. Primary cleft repair is often performed at the age of 3 months. For unilateral cleft lips, the four main techniques for repair include straight line repair (Rose-Thompson), triangular flap repair (Randall-Tennison), the rotation-advancement repair (Millard) and the anatomical subunit approximation technique (Fisher). It is common for these patients to require secondary procedures. The nasal deformity is addressed at the same time. Some recommend primary alveolar cleft repair before 2 years of age. Secondary bone grafting is recommended during age of transitional dentition (between ages 8–12). Definitive rhinoplasty is performed at adolescence.

1.1.22 Diagnosis: Bilateral Cleft Lip

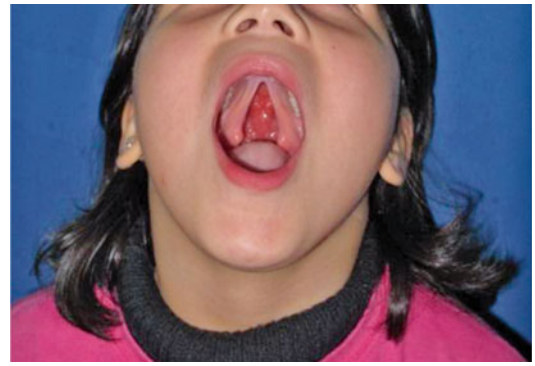


History Bilateral lip deformity

Exam Bilateral cleft lip results from failure of fusion of the maxillary prominences with the medial nasal prominence. The deformity is characterized by a disruption of the lip, nostril sill, and alveolus. The unattached premaxillary segment results in varying degrees of protrusion and angulation.

Treatment As in unilateral cleft lip, treatment requires a multidisciplinary approach addressing the associated anomalies, genetic assessment, feeding techniques, and presurgical orthopedics. The timing of the repair is ideally around 3 months of age. Different techniques of repair have been described, but common principles of repair include bilateral cleft repair, reduction of the prolabium, formation of Cupid's bow and tubercle from lateral lip elements, repair of orbicularis muscle, deepening of the central gingivolabial sulcus using prolabial mucosa, and primary rhinoplasty.

1.1.23 Diagnosis: Primary Cleft Palate

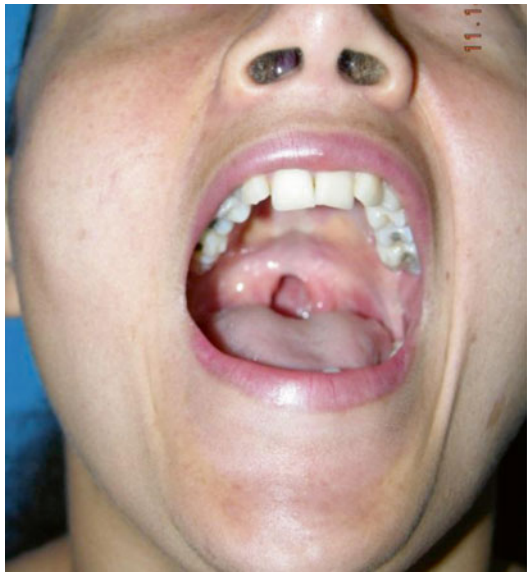


History A cleft palate deformity is caused by a disruption in the migration and fusion of the medial and lateral nasal prominences of the frontonasal process with the maxillary prominence. The incidence of isolated cleft palate with associated anomalies is approximately 70 %. Patients with an untreated cleft palate will present with feeding difficulties, impaired facial growth, and speech abnormalities.

Exam The lip, alveolus, and hard palate anterior to the incisive foramen are affected.

Treatment Surgical management of a primary cleft palate requires a repair of the anterior hard palate. A two-flap palatoplasty is preferred with bilateral mucoperiosteal flaps based on the greater palatine arteries. The flaps will extend to the cleft margin and provide adequate tissue coverage with tension-free closure.

1.1.24 Diagnosis: Submucous Cleft

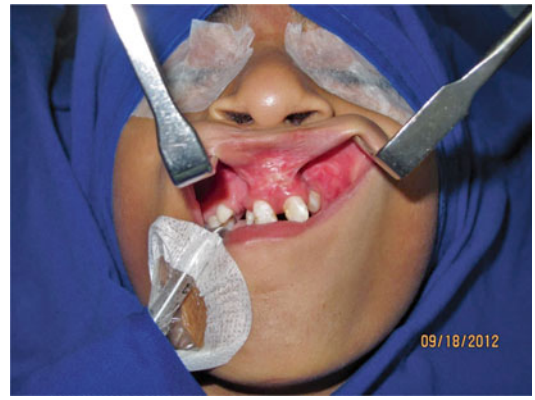


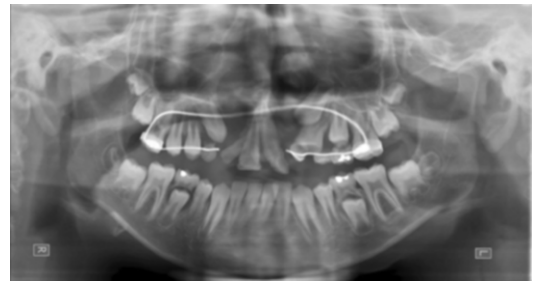
History Submucous cleft palate occurs in 1:10,000–1:20,000 births. Patients often present because of velopharyngeal insufficiency and other speech/resonance disorders. Initial evaluation focuses on extent of nasal air flow escape and speech difficulties. Results of these studies are used to guide treatment.

Exam The classical form of submucous cleft is defined by the presence of the following anatomical features: (1) a bifid or distorted uvula, (2) a bony notch at the back of the hard palate, and (3) a translucent zone in the midline of the soft palate due to separation of normally fused muscles. Occult forms may exist that lack these clinical findings, but the patient presents with velopharyngeal insufficiency.

Treatment The goal of treatment centers on establishment of velopharyngeal competence. Surgical treatment includes palatal-lengthening procedures, repositioning of the levator muscles of the velum and sphincter, or pharyngoplasty. Techniques that have been described include Furlow Z-plasty, Wardill technique, minimal incision palatopharyngoplasty, and variations of the above.

1.1.25 Diagnosis: Alveolar Cleft



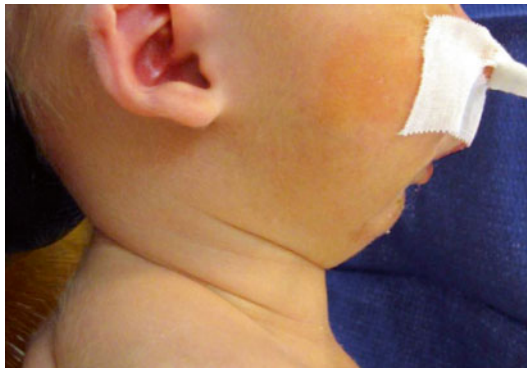


History Patient presents with a gap in the alveolus.

Exam An alveolar cleft or gap is the result of abnormal primary palate formation during weeks 4–12 of gestation. Alveolar gap can be unilateral/bilateral, and it may or may not be associated with a palatal defect. This defect is also associated with mostly anterior nasal septal deviation, which is more extreme and posterior as well when associated with a palate defect. Exam should also include a hearing exam.

Treatment The rationale for closure of alveolar clefts includes stabilizing the maxillary arch, permitting support for tooth eruption, eliminating oronasal fistulae, and providing improved aesthetic results. Surgical treatment includes (1) appropriate flap design, (2) wide exposure, (3) nasal floor reconstruction, (4) closure of oronasal fistula, (5) packing bony defect with cancellous bone, and (6) coverage of bone graft with gingival mucoperiosteal flaps. Orthodontic preparation may be required for certain alveolar clefts. Surgery should be performed prior to permanent teeth eruption.

1.1.26 Diagnosis: Pierre Robin Sequence

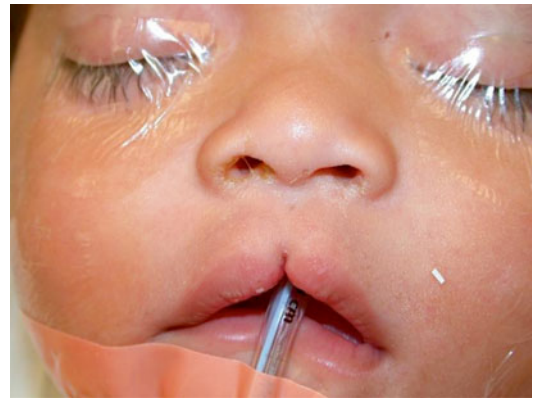


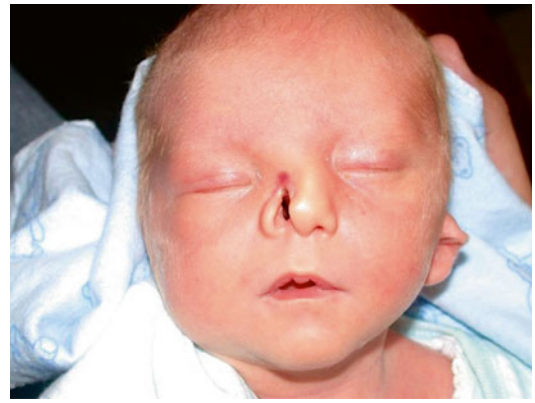
History A sequence of events that occur secondary to a small mandible and includes: glossoptosis, cleft palate, and possible airway obstruction.

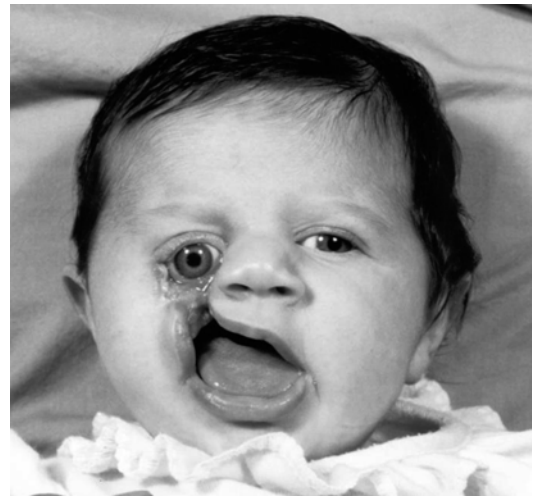
Exam Micrognathia, retrodisplaced tongue, possible cleft palate (often U shaped), and possible difficulty breathing. Must rule out other associated syndromes: Stickler, velocardiofacial, Treacher Collins, hemifacial microsomia, trisomy 18, and Nager.

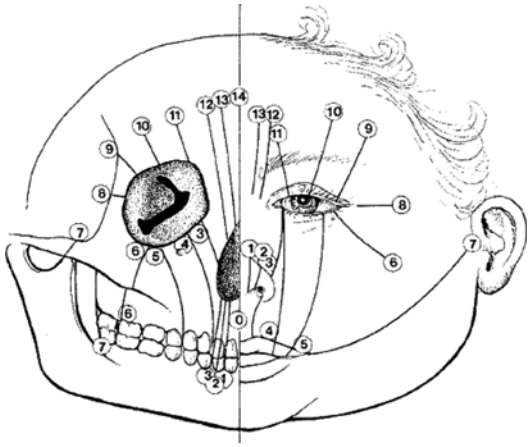
Treatment Successful management of airway includes prone or semi-prone positioning (70 %), nasopharyngeal airway, palatal plates, tongue-lip adhesion, or a short course of intubation. Tracheostomy may be required in severe cases (~10 %), and mandibular distraction is reserved for refractory ventilator-dependent patients. Cleft palate repair as per protocol.

1.1.27 Diagnosis: Craniofacial Clefts









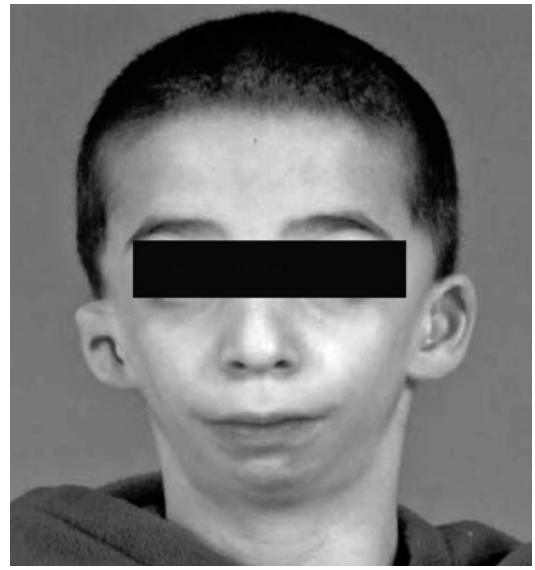
History Patient presents with anatomic distortions of the face and cranium with deficiencies in a linear pattern.

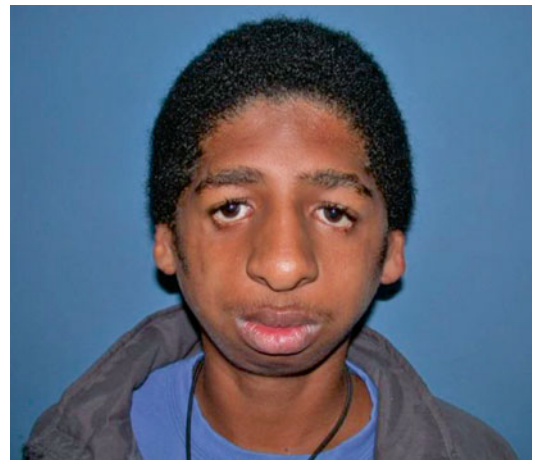
Exam Craniofacial clefts exist in a multitude of locations with varying degrees of severity. In the Tessier classification, clefts are numbered from 0 to 14. The orbits separate the cranial clefts (8–14) from the facial clefts (0–7). The clefts are numbered so that the facial and the cranial component of the cleft always add up to 14. Tessier cleft number 7 is the lateral-most craniofacial cleft. CT scan is obtained to evaluate underlying skeletal deformities.

Treatment Treatment of craniofacial clefts requires a multidisciplinary approach. Timing of surgery depends on severity of the problem. Functional problems like ocular exposure are treated early. Cranial defects and soft tissue clefts are usually corrected during infancy. Midface reconstruction and bone grafting are performed around 6–9 years of age. Orthognathic procedures are deferred until skeletal maturity.

Photos showing examples of facial clefts.

1.1.28 Diagnosis: Treacher Collins Syndrome



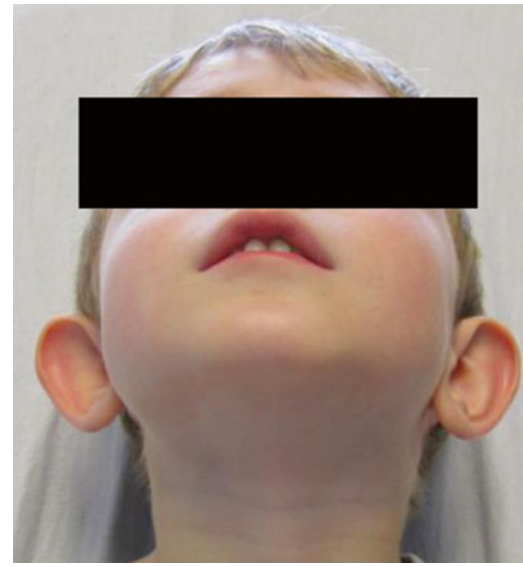


History A common autosomal dominant bilateral craniofacial cleft syndrome with an incidence of 1:10,000. Initially described by Treacher Collins in 1900, it has later been recognized as a combination of Tessier cleft numbers 6, 7, and 8.

Exam Multiple facial findings of malar and mandibular bone hypoplasia, colobomas, and lower lid retraction with an antimongoloid slant as the lateral canthus is displaced inferiorly. Loss of facial width with the absence of the zygomatic arch and hypoplasia of the temporalis muscle and ear abnormalities.

Treatment Given the narrow pharyngeal size and mandibular hypoplasia, airway management is imperative. Facial reconstruction may occur which includes eyelid reconstruction for colobomas, distraction osteogenesis for mandibular hypoplasia, skeletal augmentation, and orthognathic surgery.

1.1.29 Diagnosis: Prominent Ear

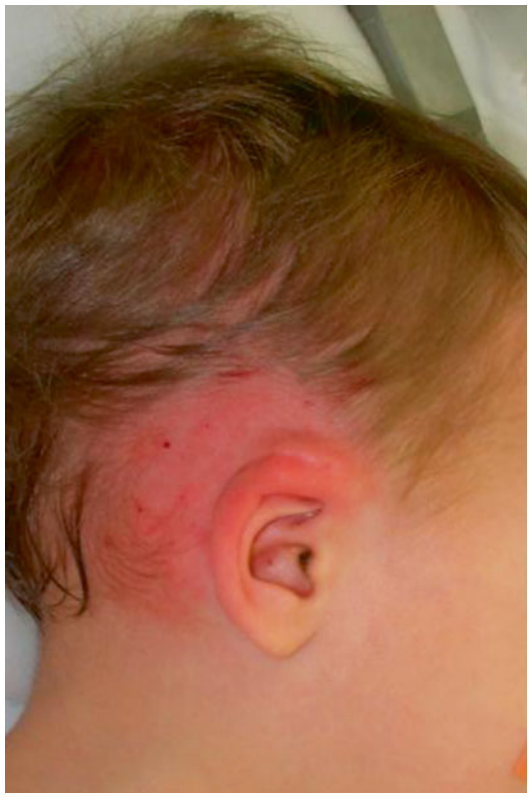


History A congenital ear deformity that results from embryonic arrest during the final convolutions of the ear, with failure of folding of the antihelix.

Exam The most common feature of a prominent ear is the effacement of the antihelical fold with a conchoscaphal angle >90°. Other features include conchal hypertrophy defined as a deep conchal bowl with excessive height of the conchal wall >1.5 cm.

Treatment Many authors advocate combining techniques to minimize relapse. Commonly used techniques for the antihelical fold deformity are the conchoscaphal suture technique (Mustarde), antihelical scoring (Stenstrom), or antihelical excision (Luckett). The conchal bowl hypertrophy is managed with conchomastoid suture technique (Furnas) or conchal bawl excision (Davis).

1.1.30 Cryptotia

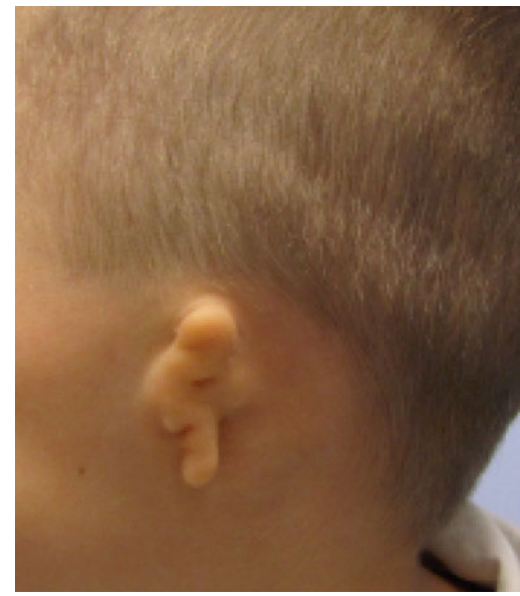


History An abnormal adherence of the superior ear to the temporal skin with varying degrees of severity. Cryptotia is commonly seen in Asians with an incidence of 1:400. The deformity is bilateral in 40 % of the cases with the right ear being more affected than the left.

Exam Cartilaginous malformation in the scaphal-antihelix complex with loss of the post-auricular sulcus definition.

Treatment Splinting in the neonatal period can be effective. Surgical intervention requires division of the abnormally positioned transverse and oblique ear muscles. The surgically created retro-auricular sulcus is then covered with a skin graft.

1.1.31 Microtia



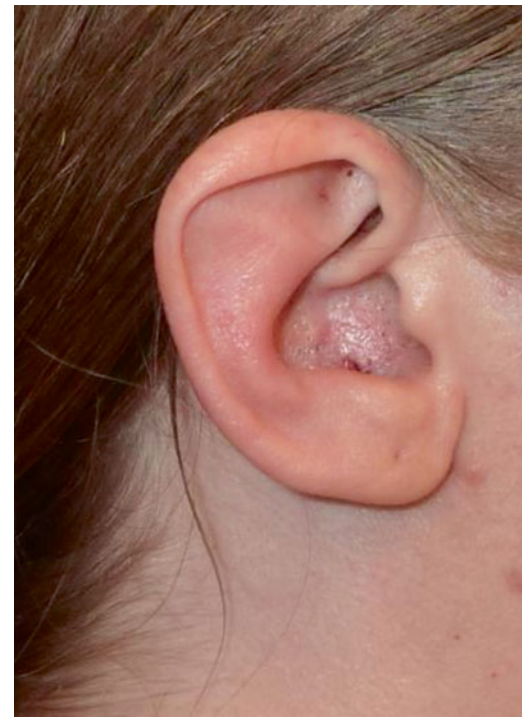
History It is the most common major congenital anomaly of the external ear. The etiology is likely a result of in utero tissue ischemia secondary to obliteration of the stapedial artery or actual hemorrhage into the local tissues. Microtia is also associated with several craniofacial syndromes. The classification system is based on a gradient from mild ear deformity to total absence of the external ear (anotia).

Exam Grade I: The pinna is mildly deformed and smaller than normal.

- Grade II: The pinna is smaller than in grade II. The helix may not be fully developed. The triangular fossa, scaphae, and antihelix have much less definition.
- Grade III: The pinna is essentially absent, except for a vertical sausage-shaped skin remnant. The superior aspect of this sausage-shaped skin remnant consists of underlying unorganized cartilage, and the inferior aspect of this remnant consists of a relatively well-formed lobule.
- Grade IV: Total absence of the pinna.

Treatment Staged reconstruction of the external ear begins with excision of the remnant cartilaginous component and placement of a cartilagenous framwork made of rib grafts. In subsequent procedures, the lobule and tragus are reconstructed. Creation of a post-auricular sulcus then follows with placement of a skin graft. Surgical management of the middle ear should be delayed until after the auricle is reconstructed.

1.1.32 Stahl's Ear

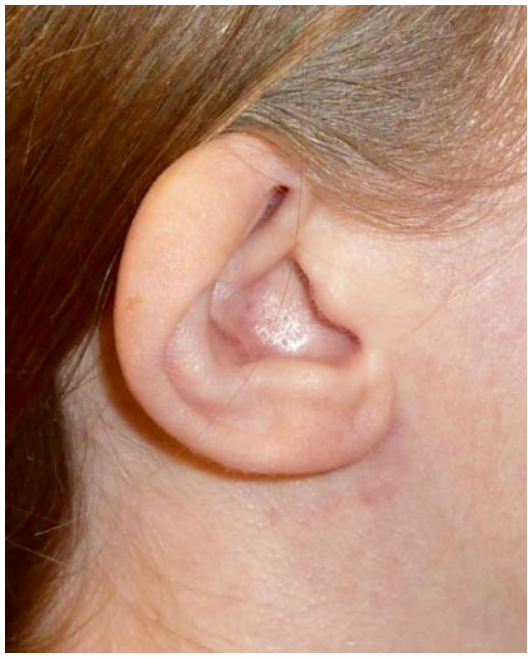


History A congenital hereditary auricular deformity of the helical rim commonly found in patients of Asian descent. Stahl's ear deformity, first described in the nineteenth century, is also known as "Spock's ear."

Exam The deformed ear is recognized by the presence of an abnormal cartilaginous pleat, known as the third crus, which extends from the antihelix to the edge of the helix. In addition, a flat helix and an anomalous scaphoid are present.

Treatment In the neonatal age, it can be treated with splinting given the pliability of the cartilage found in the early months of life. Surgical management is indicated for children and young adults. Operative techniques include wedge excision of the third crus, third crus helical advancement, Z-plasty, cartilage reversal, or periosteal tethering.

1.1.33 Cup/Constricted Ear



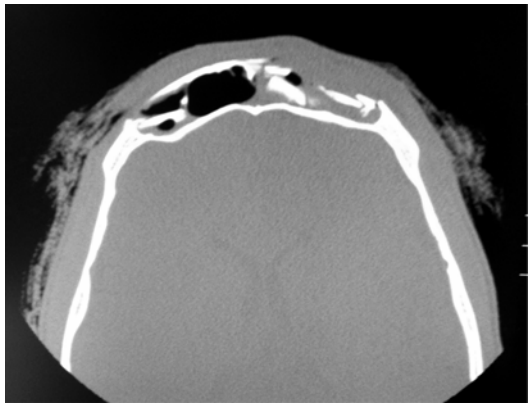
History Constricted ear anomalies can be categorized as either lop or cup ear deformities. The lop ear is a malformed auricle with an acute downward folding of the superior ear. A cup ear is a malformed ear that combines the features of a lop ear and a protruding ear.

Anatomy/Exam In a lop ear, the folding or deficiency is in the helix and scapha at the level of Darwin's tubercle. Malformed superior crus of the antihelix may also be present. The features of a cup ear are overdeveloped, cup-shaped concha, a deficient superior aspect of the helical margin and antihelical crus, and a small vertical height.

Treatment In cases of helical involvement only, the folded helix is detached and reattached in an upright position. Scaphal involvement is treated with the use of local tissues such as banner flaps of cartilage and skin flaps from the medial surface of the ear. In severe cases involving the antihelix and conchal wall, Kislov's technique is utilized with unfurling of the superior pole, the use of remnant tissue for the middle portion of the ear, and contralateral conchal cartilage for missing tissue.

1.2 Craniofacial, Trauma

1.2.1 Frontal Sinus Fracture

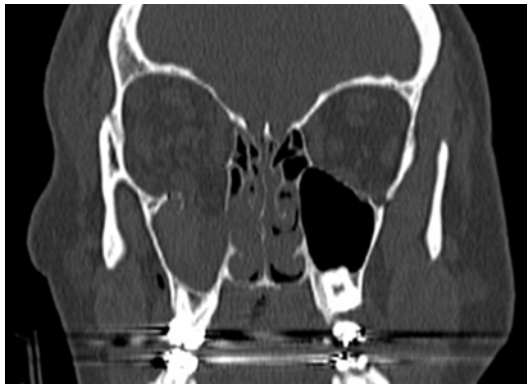


History Severe trauma to the head and/or face.

Exam Lacerations, palpable step-offs, or contour irregularities of the forehead should prompt for a craniomaxillofacial CT scan (1.5 mm cuts) with axial and sagittal views. Key features of the fracture are the degree of comminution, displacement of the anterior and/or posterior tables, and involvement of the frontal sinus outflow tract (FSOT or nasofrontal duct). Nasal discharge may be present, and CSF leak should be ruled out with beta-transferrin.

Treatment Depends on the characteristics of the fracture with the goal of minimizing contour deformities and late complications (mucocele, osteomyelitis, epidural abscess, CSF leak, etc.). *Observation* is acceptable for minimally displaced anterior or posterior wall fractures without FSOT involvement or CSF leak. *Open reduction and fixation* of anterior table fractures with microplates are done for anterior wall irregularities. For fractures that involve the anterior wall and FSOT, *obliteration* of the sinus and outflow tract (with bone, fat, pericranial flap), exenteration of all sinus mucosa, and plating of anterior wall are done. CSF leak and severe bone loss require *cranialization* of the frontal sinus where the posterior table is removed, in addition to obliteration of the sinus.

1.2.2 Orbital Floor: Blow-Out Fracture

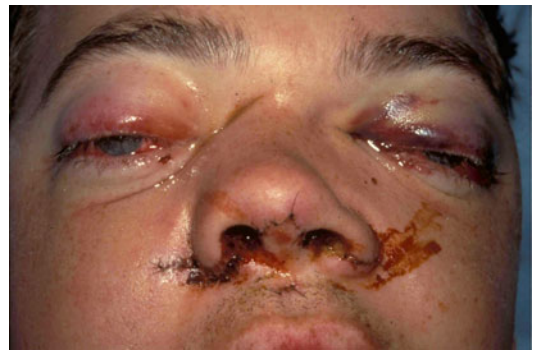


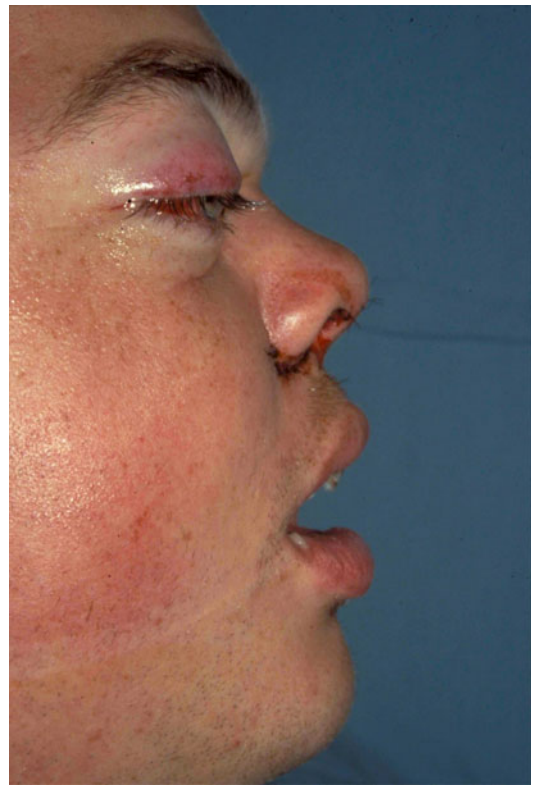
History Periorbital trauma and possible diplopia.

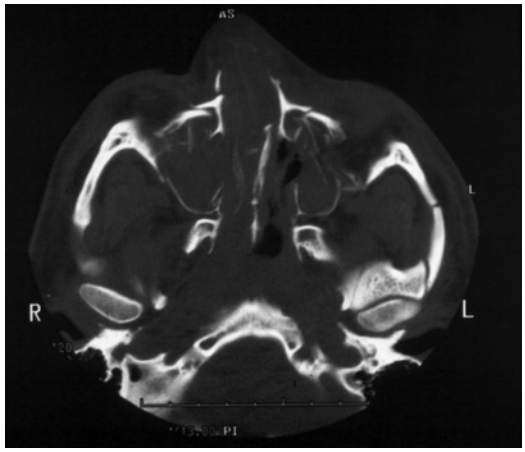
Physical Exam Periorbital edema/ecchymosis. Swelling may mask enophthalmos, which may be severe enough to result in proptosis and possible diplopia, Forced duction test will differentiate swelling from entrapment of extraocular muscles. Blow-out fractures involve isolated fractures of the orbital walls—typically the floor and medial wall. *CT face will show* displacement of the orbital floor into maxillary sinus with herniation of orbital contents.

Treatment Preoperative ophthalmic exam is warranted to rule out globe injury. Thin cut CT with axial, coronal, and sagittal views define extent of injury and need for repair. Operative repair is performed to restore orbital anatomy and avoid enophthalmos.

1.2.3 NOE Fracture







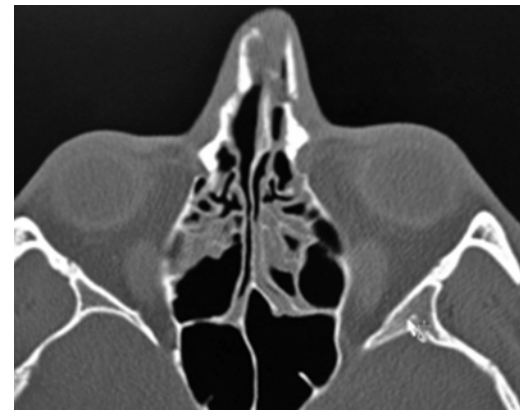
History Central midface impact.

Physical Exam Periorbital edema/ecchymosis, subconjunctival hematoma, telecanthus, shortened palpebral fissures, epiphora, and displaced nasal bridge (saddle nose deformity).

X-Ray Exam Displacement of canthal bearing bone segments.

Treatment Surgical goals include restoration of the intercanthal distance, palpebral shape, nasal projection/width/height/length, and ORIF of fractured segment(s) to adjacent stable bone. Intercanthal wires must be placed superior and posterior to the lacrimal fossa.

1.2.4 Nasal Fractures



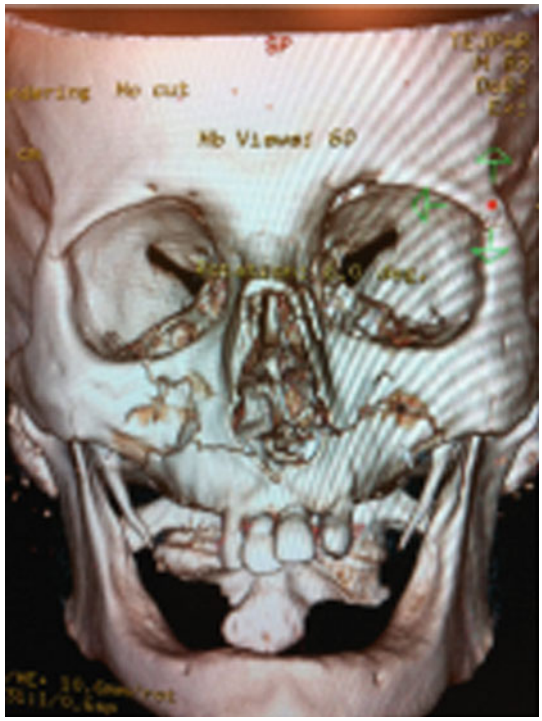
History Trauma to the nose; determine age of injury and prior nasal surgery.

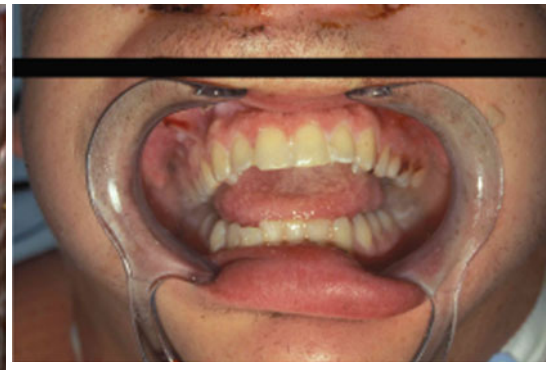
Exam Epistaxis, bony crepitus, periorbital ecchymosis, asymmetries or step-offs, and septal deviation. Must rule out a septal hematoma, and evaluate bony and cartilaginous nasal septum and vault.

Treatment Septal hematomas are drained immediately to avoid long-term sequelae, especially necrosis. Closed nasal reduction is successful in most isolated nasal bone fractures and is best done within 7–10 days post-injury when most edema has resolved. Obtain pre-injury photos if possible.

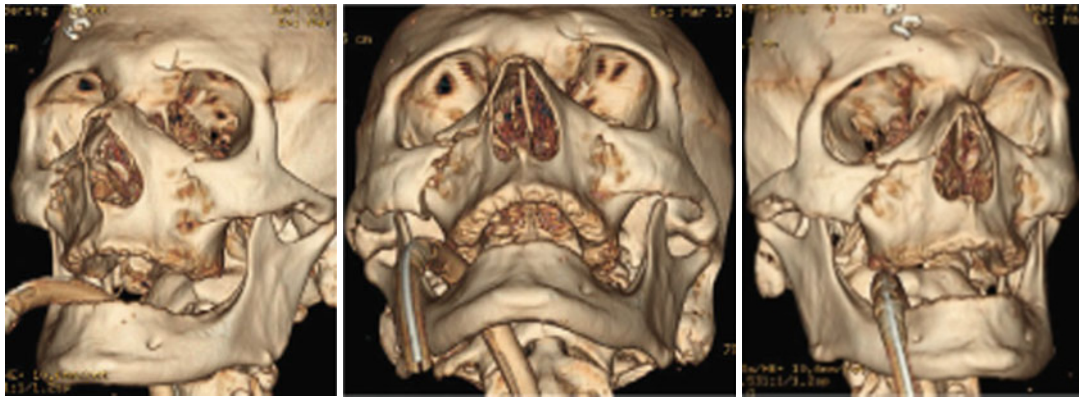
1.2.5 Maxillary Fractures

1.2.5.1 Le Fort 1





1.2.5.2 Le Fort 2

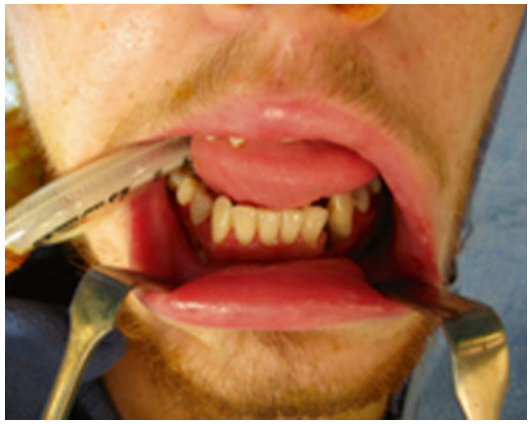


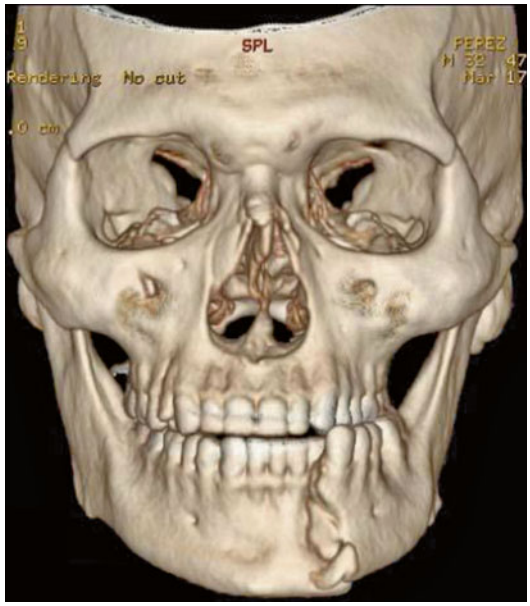
History Facial trauma.

Exam Midface instability, malocclusion, trismus, enophthalmos, restricted ocular movement, V2 paresthesia, loss of facial projection/height, increased facial width, and CSF leak. Maxillofacial CT with three views (axial, sagittal, coronal) and 3D reconstruction will show type of Le Fort fracture on *each side*, as bilateral Le Fort fractures at the same level are less common.

Treatment Initial control of airway and hemorrhage is paramount, followed by a coordinated operative plan with concomitant injures. Operative goals are restoration of dental occlusion, facial height/width/projection, and orbital anatomy. Nasotracheal intubation, dental splints, and multiple approaches to the facial skeleton may be necessary.

1.2.6 Mandibular Fractures: Parasymphyseal





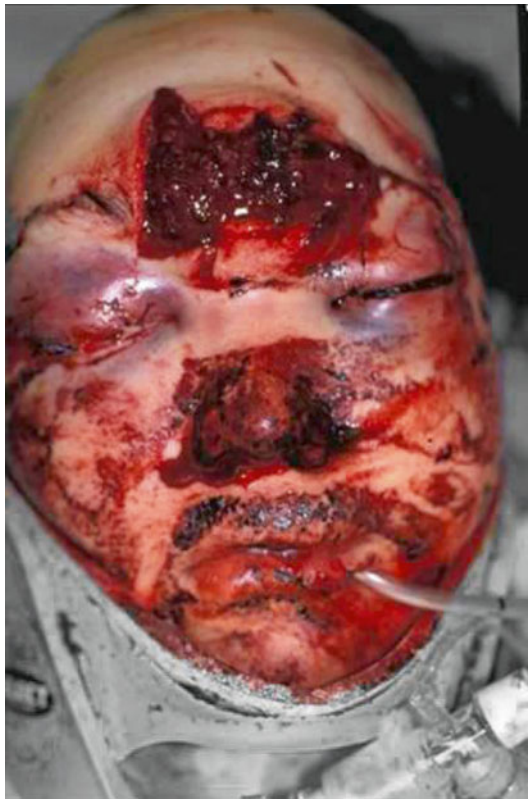
History Trauma to the lower face and malocclusion.

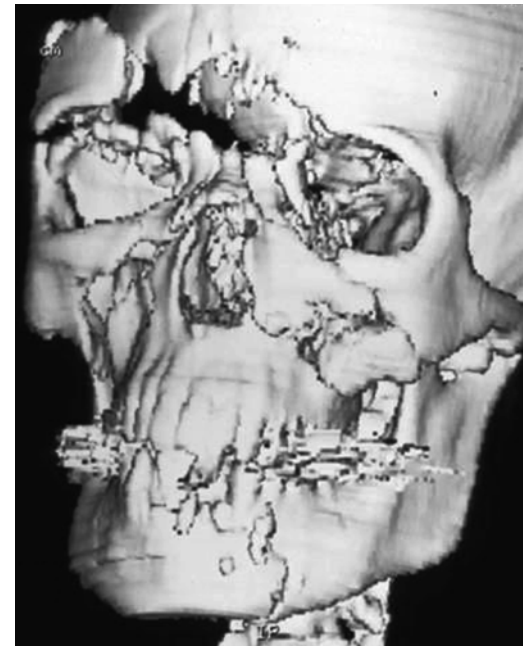
Physical Exam Intraoral lacerations, palpable bony step-offs or mobility, malocclusion, diminished range of motion, crossbite, anterior or posterior open bite, and V3 paresthesia.

X-Ray Exam Imaging should include maxillofacial CT (with axial, coronal, and 3D reconstruction views) and/or plain X-rays (panoramic and posteroanterior views).

Treatment Depends on fracture location and pattern (unstable/stable, comminuted, etc.) and occlusion. Simple, non-displaced, stable fractures with pre-injury occlusion are managed with soft diet. Minimally displaced condylar or subcondylar fractures may be treated with intermaxillary fixation (arch bars/IMF screws). ORIF is needed for more significant fractures of the body, ramus, angle, parasymphyseal/symphyseal, bilateral subcondylar, or dislocation of the condyle into the middle cranial fossa. Severely contaminated and/or comminuted fractures may require external fixation.

1.2.7 PAN Face Fracture



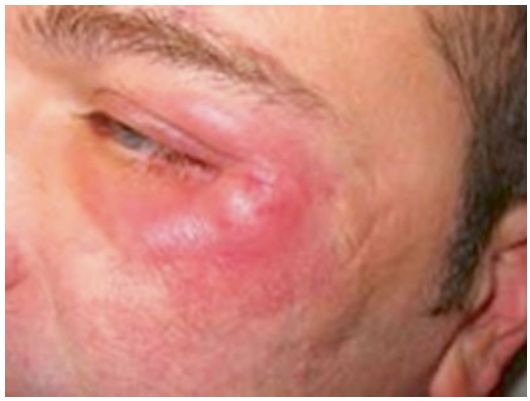


History Severe trauma to the craniofacial skeleton involving the upper, middle, and lower face.

Exam Any combination of the previously mentioned findings of facial fractures. Obtain maxillofacial CT with axial, coronal, sagittal, and 3D reconstruction views.

Treatment Stability of airway, hemorrhage, and concomitant injuries is a priority. Preoperative planning, based on CT imaging, ensures a methodical sequence of fracture ORIF.

1.2.8 Orbital Cellulitis



History

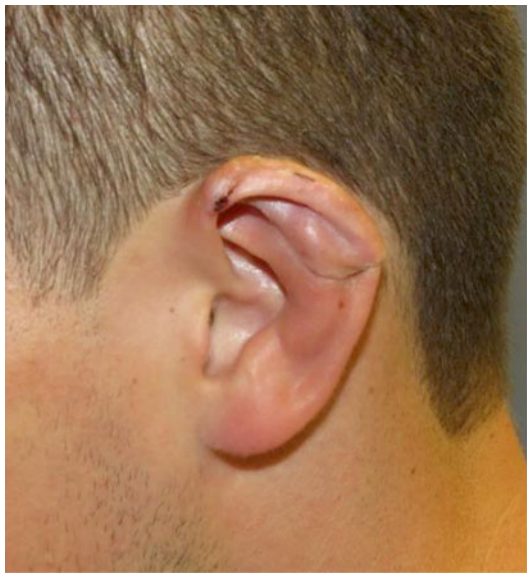
History of trauma, orbital surgery, or sinusitis.

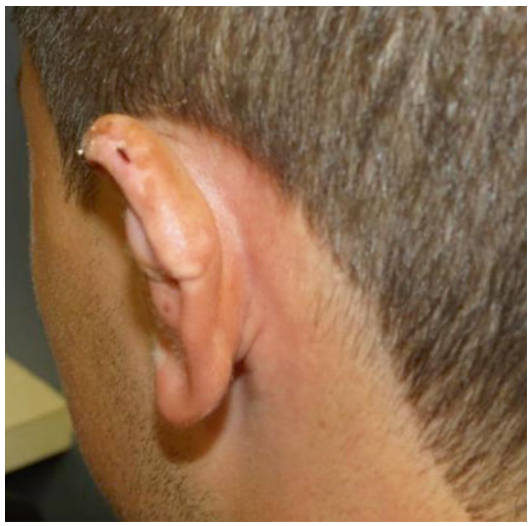
Physical Exam: Periorbital Swelling and Erythema Sinus, eye, and orbital abscess aspirates yielded the greatest number of positive cultures, though these invasive surgical procedures should be performed only when clinically indicated. Blood cultures have a low yield.

Treatment

Staphylococcus and *Streptococcus* species are the most common pathogens. The occurrence of MRSA in pediatric orbital cellulitis is increasing; therefore, empiric intravenous antimicrobial therapy should be directed against these organisms. Prompt recognition and early aggressive treatment to control the spread of infection is crucial in preventing complications such as permanent visual loss, diplopia, ophthalmoplegia, optic neuropathy, central retinal artery occlusion, cavernous sinus thrombosis, meningitis, intracranial abscess formation, and septic embolus.

1.2.9 Cauliflower Ear



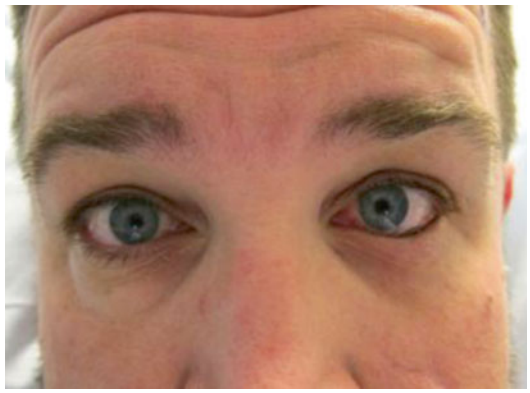


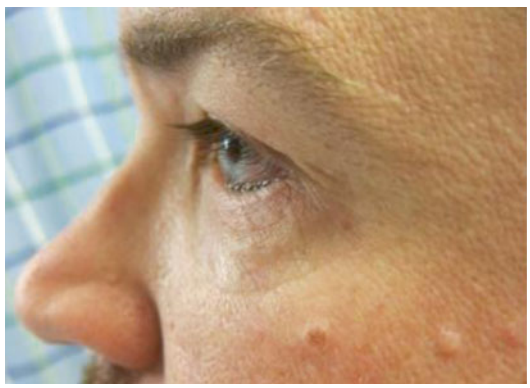
History A deformity commonly seen as a result of repeated trauma to the ear with multiple episodes of hematoma formation. Subperichondrial bleeding on the anterior surface of the ear leads to new cartilage formation within the confines of the perichondrium.

Exam The auricle appears curled and thickened.

Treatment Open drainage of the hematoma and total excision of the fibrocartilaginous layer and perichondrium with skin closure under a bolster dressing.

1.2.10 Entropion



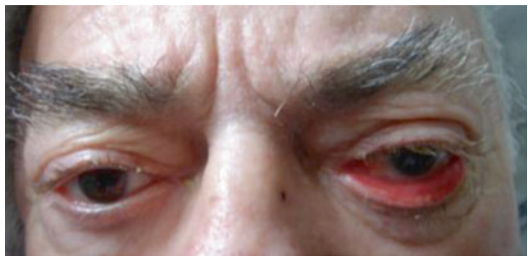


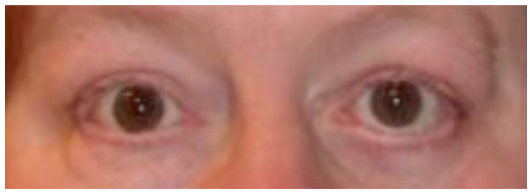
History Entropion is defined as the inward rolling of the eyelid margins (usually the lower lid). Entropion can be acquired (involutional, cicatricial) or congenital. Patients usually present with symptoms of excessive tearing and irritation.

Exam The findings of lower eyelid margin inversion, scleral show, and horizontal lid laxity are indicative of involutional entropion. Lid tightness with shortening of the posterior lamella is consistent with cicatricial entropion. Trichiasis will develop when the lid margin is overly rotated causing the lower lid lashes to rub against the sclera. Involutional ectropion can be confused with involutional entropion because of the lower lid laxity that is present in both states. The two can be distinguished with animation of the orbicularis oculi muscle. Inversion of the lower lid occurs on attempted eyelid closure in involutional entropion.

Treatment Involutional entropion repair can be achieved with shortening or repositioning of the lower lid. Cicatricial entropion of the posterior lamella can be improved with placement of a spacer graft.

1.2.11 Ectropion



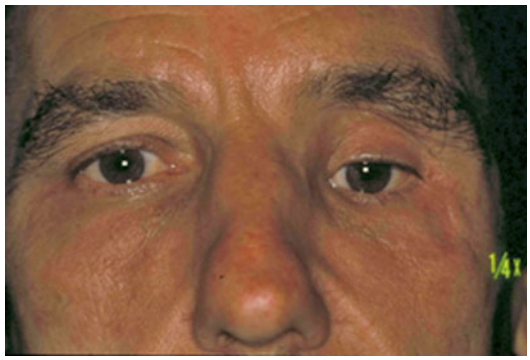


History Ectropion is the outward turning of the eyelid (usually the lower eyelid) such that the inner surface is exposed. Patients usually present with symptoms of dryness, tearing, and irritation of the eye. Causal factors leading to ectropion are involutional, congenital, or cicatricial. Paralysis and loss of orbicularis muscle tone or neoplasm within the lower eyelid can also lead to the pulling of the eyelid away from the globe.

Exam Eversion of the eyelid along with findings of abnormal snap-back test and distraction test suggests horizontal lid laxity (involutional ectropion). Vertical shortening of the eyelid is likely due to a congenital deformity or scarring from a burn or iatrogenic causes.

Treatment Early treatment of ectropion with scleral show consists of massaging of the eyelids and artificial tears. Surgical management of involutional ectropion includes a horizontal lid shortening procedure, a canthopexy or a canthoplasty, and possibly a midface cheek suspension. Anterior lamellar deficiencies are corrected with a full-thickness skin graft.

1.2.12 Enophthalmos

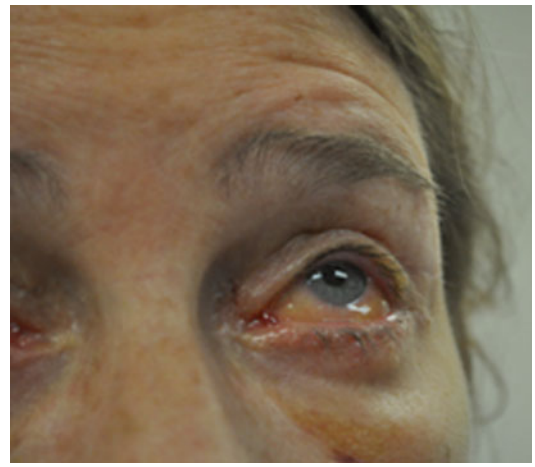


History Enophthalmos is the recession of the ocular globe within the bony orbit. The most common cause is inadequate treatment of orbital fractures where the orbital floor is disrupted and the globe is displaced inferiorly.

Exam Malpositioning of the globe results in dystopia (also seen in the photo). Recession of the globe causes the lower lid to invert and appear entropic. Pseudoptosis is present when the upper lid is poorly suspended over the globe. Hertel exophthalmometry is used to determine the degree of globe recession. Two mm of enophthalmos is clinically detectable, and more than 5 mm is disfiguring.

Treatment The goal in posttraumatic enophthalmos correction is restoration of the internal orbital volume to its pre-injury state. Internal orbital reconstruction is achieved with various biological or alloplastic implants, including anatomic pre-bent implants that are designed to conform to the contour of the orbit.

1.2.13 Chemosis

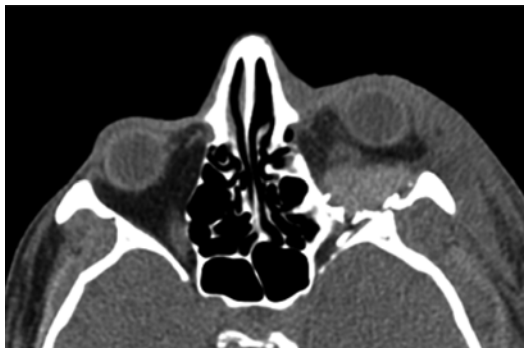


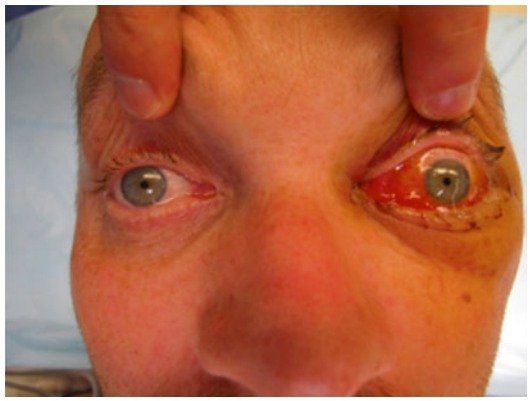
History Chemosis represents edema of the conjunctiva from irritation of the eye. It results from iatrogenic causes, trauma, viral infection, or various other medical conditions. In the surgical patient, the cause is multifactorial including exposure, periorbital edema, and postoperative lymphatic dysfunction with obstruction of the surrounding lymphatic channels. Chemosis can cause persistent discomfort and aggravation in the postoperative period following surgery of the eyelids.

Exam Chemosis can present intraoperatively or postoperatively with swelling of the palpebral and bulbar conjunctiva. In severe cases it can prohibit complete lid closure, leading to chemosis-induced lagophthalmos or even chemosis-associated lower lid malposition.

Treatment Successful treatment exists along a continuum from liberal lubrication, ophthalmic steroid preparations, ocular decongestants, eye patching, to minor surgical procedures such as drainage conjunctivotomy and temporary tarsorrhaphy. In all cases, chemosis ultimately resolves.

1.2.14 Retrobulbar Hematoma



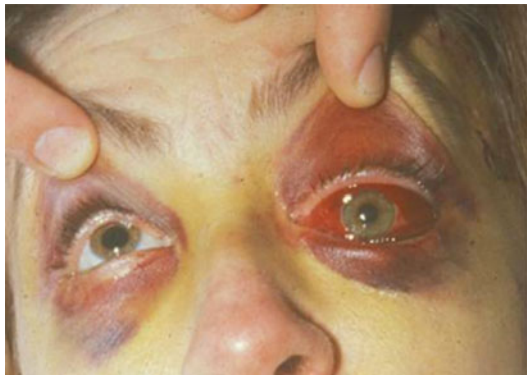


History Retrobulbar hematoma is an accumulation of extravagated blood behind the globe. The clinical symptom of acute orbital hemorrhage is rapid onset of pain.

Exam Clinical findings include proptosis that is usually accompanied by eyelid ecchymosis, globe induration, dilated pupils, tenderness, and possible vision loss.

Treatment Retrobulbar hemorrhage is a medical and surgical emergency that must be treated immediately to prevent blindness. A hematoma without visual impairment is managed by decompression via existing incisions, exploration, and evacuation of the hematoma. If visual impairment is present, it is recommended to perform surgical decompression including lateral canthotomy, reduction of intraocular pressure with mannitol and acetazolamide, and administration of 95 % O_2/5 % CO_2.

1.2.15 Superior Orbital Fissure Syndrome

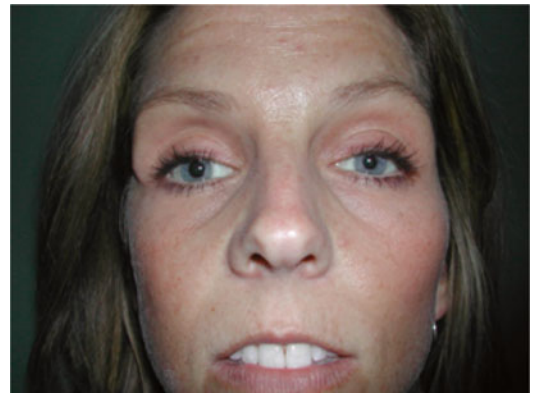


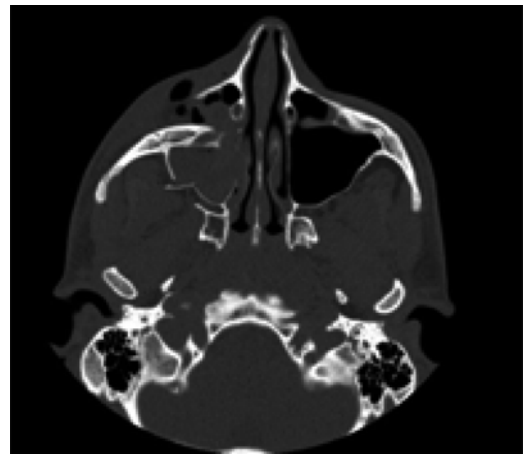
History A superior orbital fissure syndrome is caused by a high-velocity injury to the orbit with an extension of an orbital fracture into the superior orbital fissure. The characteristics of superior orbital fissure syndrome are attributable to a constellation of cranial nerve III, IV, and VI palsies.

Exam Affected patients have paralysis of the extraocular muscles and the levator palpebrae superioris muscle (lid ptosis). In addition, loss of reflex and accommodation to direct light, loss of corneal reflex, and a fixed dilated pupil are part of the syndrome. Anesthesia of the forehead is present when the trigeminal nerve (V_1) is involved.

Treatment Supportive care and observation are the mainstay of treatment. Decompression of the optic nerve is controversial.

1.2.16 Zygomaticomaxillary Complex (ZMC) Fracture



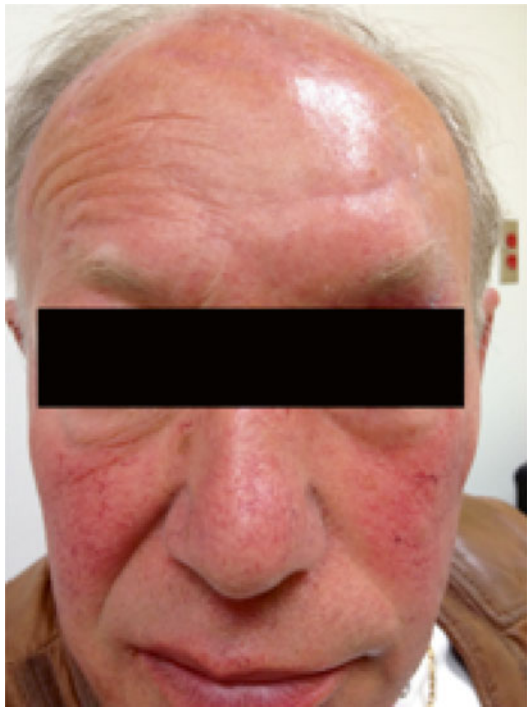


History Midfacial trauma.

Exam Depressed malar eminence (flattening), altered facial width, periorbital ecchymosis/edema, enophthalmos, proptosis, palpable orbital rim step-offs, paresthesia in the infraorbital nerve (V_2) distribution, trismus, diplopia, and restricted ocular movement.

Treatment Non-displaced or minimally displaced ZMC fractures can be managed nonoperatively. Significantly displaced ZMC fractures require open reduction internal fixation via multiple approaches to restore facial width, projection, and height as well as orbital volume. Oftentimes after reduction of the ZMC fracture, an orbital floor defect will be revealed that requires repair. Zygomaticosphenoid suture (lateral orbital wall) provides the best guide to anatomic reduction.

1.2.17 Facial Nerve Injury

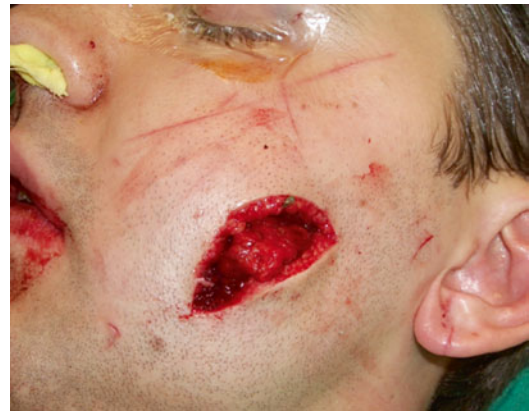


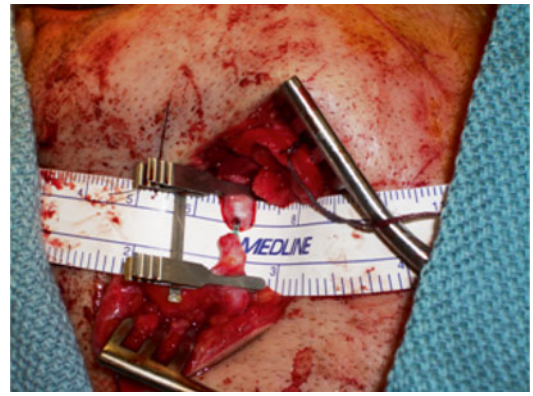
History Facial nerve injury is a devastating result of a multitude of processes which can be categorized as congenital, idiopathic, traumatic, neoplastic, or inflammatory. The resulting sequela can include exposure keratitis, drooling, and speech and swallowing difficulties. The psychosocial effects of the resulting facial paralysis can be incapacitating to patients.

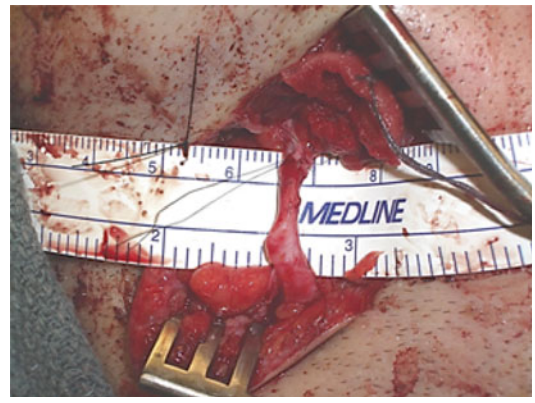
Exam Patients display a lack of tone in addition to no movement on the affected side of the face. The House-Brackmann grading scale can provide objective description of the degree of facial paralysis. Careful inspection for signs of exposure keratitis, ability to close the eye, and the presence or absence of a Bell phenomenon is important.

Treatment A multitude of procedures are available for repair of the facial nerve, including direct repair, nerve grafting, and nerve crossover techniques. Facial nerve decompression for cases of intact but damaged nerves offers another option. Muscle transfers offer another treatment modality when reinnervation is not possible.

1.2.18 Parotid Injury







History Traumatic facial wound.

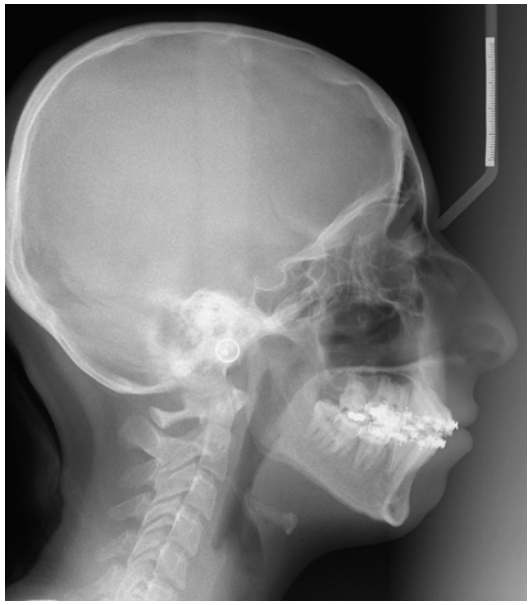
Exam Injuries to the parotid gland and its duct are usually associated with traumatic facial wounds and may be accompanied by damage to the facial nerve. Failure to recognize these injuries may lead to sequela such as sialocele or parotid fistula development. Classification of

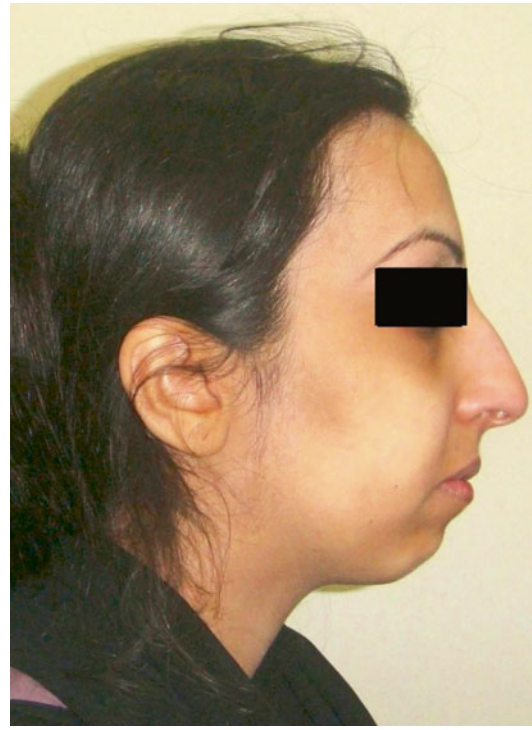
parotid injuries has been described: within the gland (region A), over the masseter muscle (region B), and from the masseter muscle to entry of the oral cavity (region C). In penetrating injuries occurring along a line drawn from the tragus to the midportion of the upper lip, parotid duct injury should be suspected. The duct can be cannulated or injected with saline or methylene blue to rule out injury.

Treatment Early treatment of parotid gland and duct injuries is ideal and should be directed by the injury region. Glandular injuries can be closed primarily with or without duct stenting. Parotid duct injury treatment should be repaired over a stent. Injury to the duct orifice can be treated by drain placement.

1.3 Craniofacial, Orthognathic

1.3.1 Microgenia



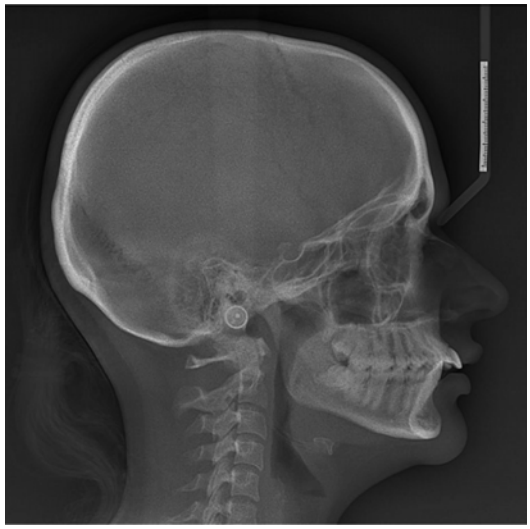


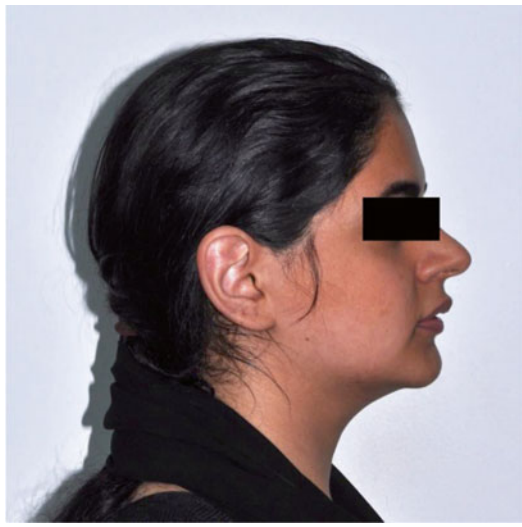
History Congenital hereditary deficient chin. It can be combined with mandibular retrognathia/prognathia.

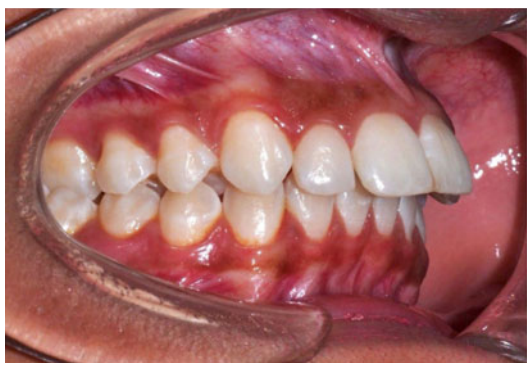
Exam Deficient bony structure of the chin. However, the patient is usually in class I occlusion.

Surgical Treatment Genioplasty or chin implant.

1.3.2 Prenormal Skeletal and Dental Relationship (Angle Class II)







History Class II can happen as a result of one or more from the following:

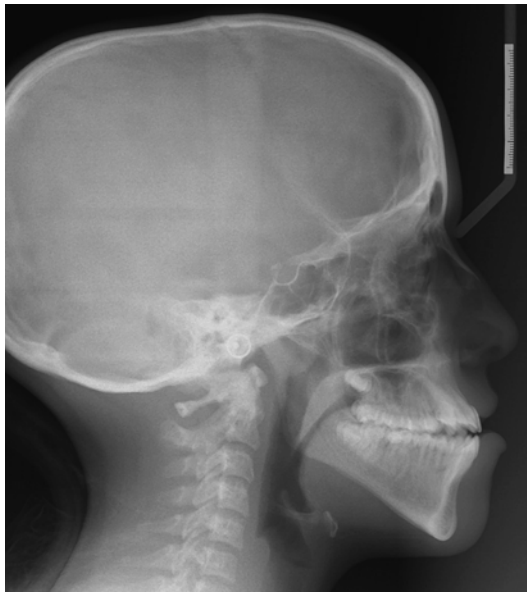
1. Congenital hereditary retrognathic mandible
2. Congenital hereditary prognathic maxilla
3. History of mandibular trauma affected on condylar growth
4. Any syndromic or pathological conditions which cause deficient mandibular growth

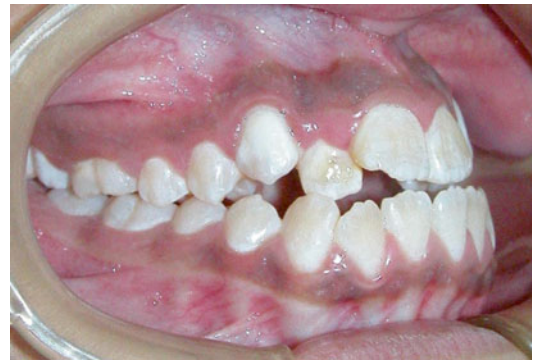
Exam Dentally: Molars and canines are in class II occlusion: The buccal groove of the mandibular first molar is distal to the mesiobuccal cusp of the maxillary first molar. There is increased facial convexity (convex profile) with retrognathic mandible and/or prognathic maxilla.

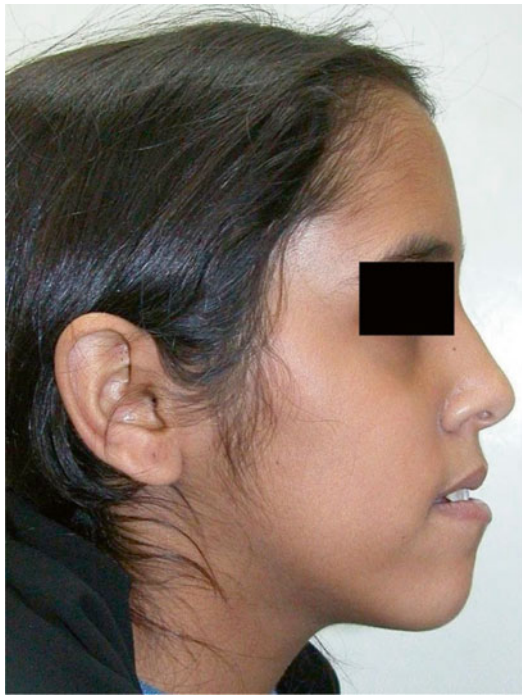
Surgical Treatment Based on patient exam, surgery can be one of the following:

- Single jaw orthognathic surgery: Advancing the mandible forward
- Double jaw orthognathic surgery: Advancing the mandible forward through bilateral sagittal split osteotomy in addition to a Le Fort I osteotomy setting back the maxilla

1.3.3 Postnormal Skeletal and Dental Relationship (Angle Class III)







History A class III can happen as a result of one or more from the following:

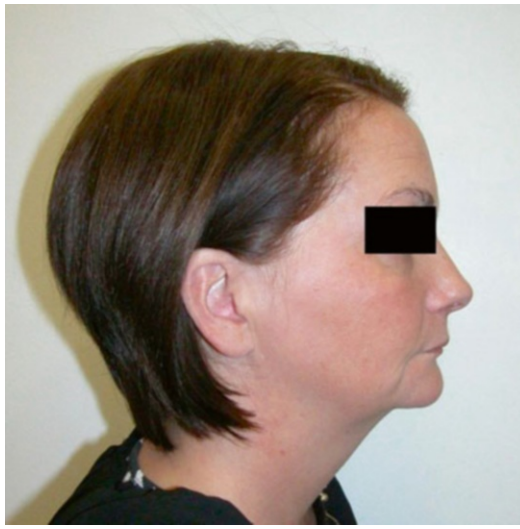
1. Congenital hereditary prognathic mandible
2. Congenital hereditary deficient maxilla/midface
3. History of facial trauma-affected maxillary growth
4. Any syndromic or pathological conditions which cause excess mandibular growth or deficient maxillary growth

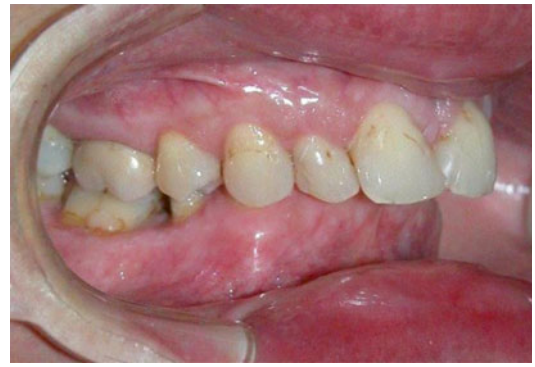
Exam Dentally: Molars and canines are in class III occlusion: The buccal groove of the mandibular first molar is mesial to the mesiobuccal cusp of the maxillary first molar. The facial profile is termed prognathic. There is decreased facial convexity (straight or concave profile) due to prognathic mandible and/or deficient maxilla.

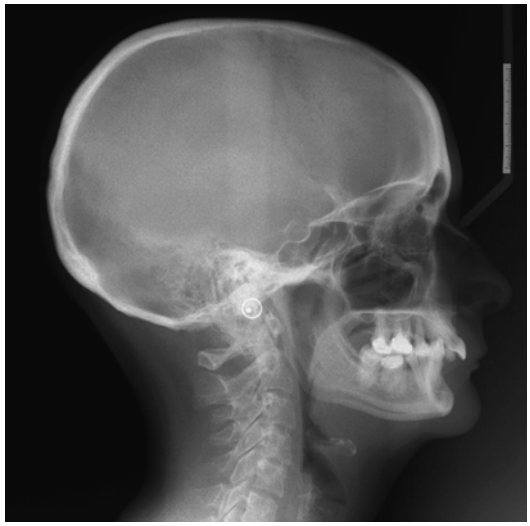
Surgical Treatment Based on patient exam, surgery can be one of the following:

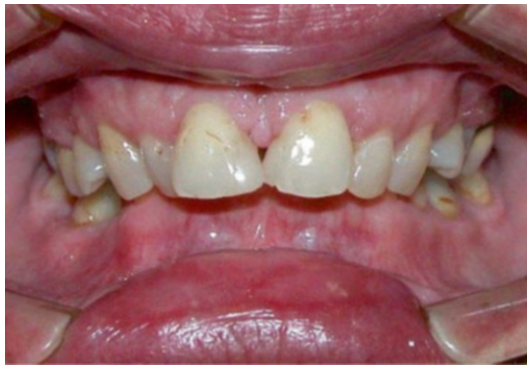
- Single jaw: Setting back the mandible
- Double jaw: Setting back the mandible and Le Fort I, II, or III advancing the maxilla forward

1.3.4 Skeletal Deep Bite









History This can happen as a result of one or more from the following:

1. Severe forward mandibular rotation (counterclockwise)
2. Forward rotation of the maxilla (clockwise)

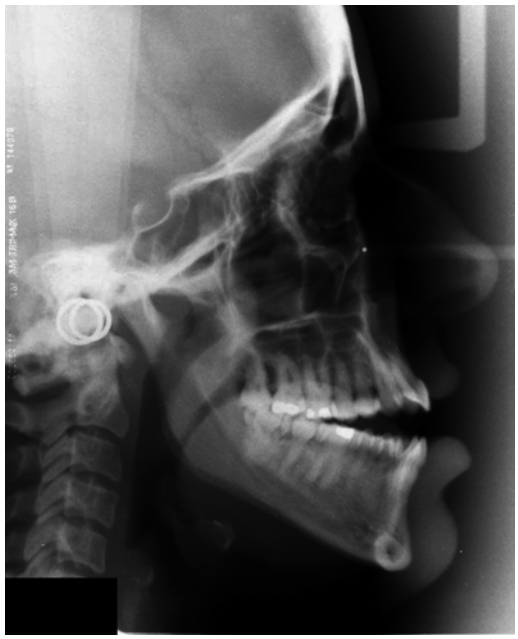
Exam Dentally: Severe deep bite (increased overbite).

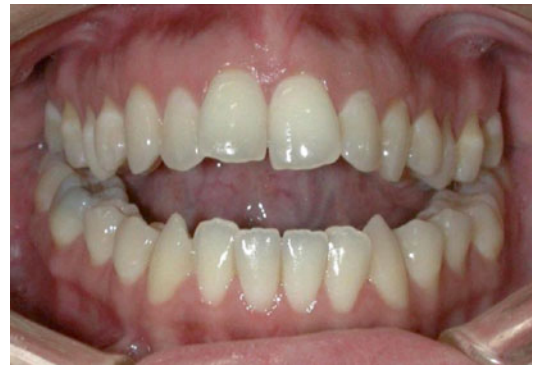
Skeletally: Short lower face. Decreased MM angle (maxillary and mandibular planes are almost parallel).

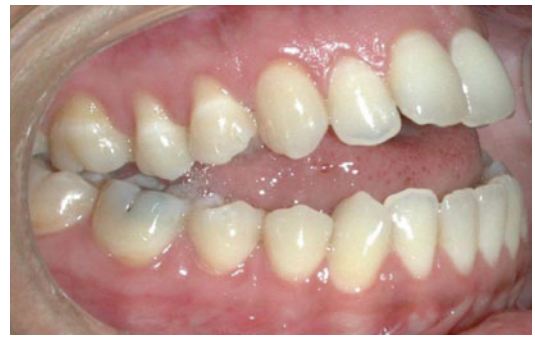
Surgical Treatment Based on patient exam, surgery can be one of the following:

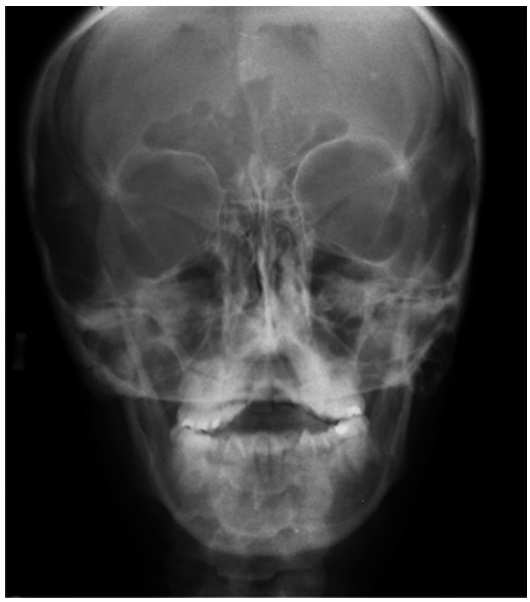
- Single jaw orthognathic surgery
- Double jaw orthognathic surgery

1.3.5 Open Bite









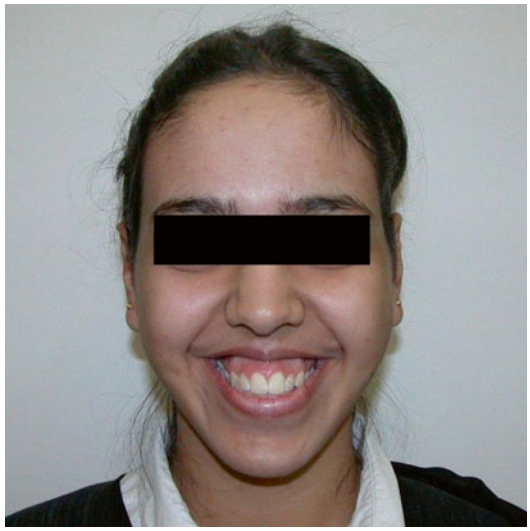
History Any congenital or developmental condition that cause one or more of the following:

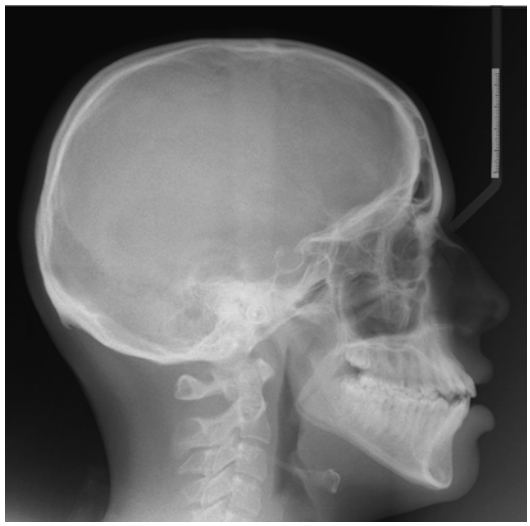
1. Severe backward mandibular rotation (Clockwise).
2. Backward rotation of the maxilla (Counter-clockwise).

Exam Dentally: Open bite (lack of overbite).
Skeletally: Long lower face. Increased MM angle.

Surgical Treatment Posterior maxillary impaction.

1.3.6 Vertical Maxillary Excess (Gummy Smile)





History Forward rotation of the maxilla (clockwise).

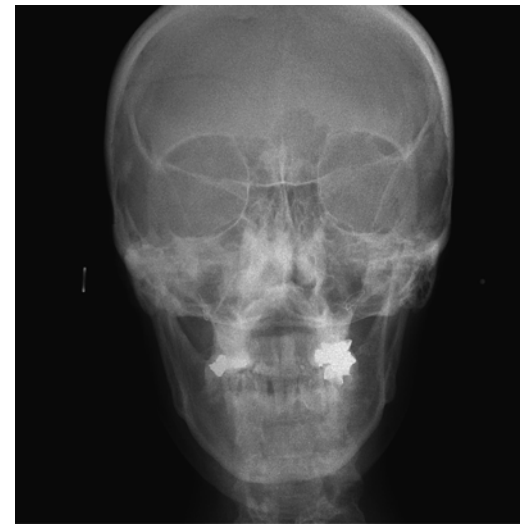
Exam Dentally: Increased gingival show in smiling.

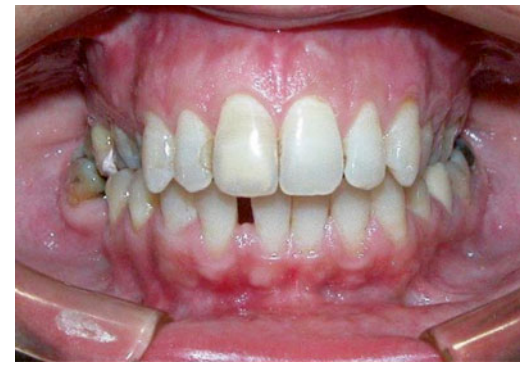
Skeletally: Long lower face.

Treatment In minor cases, using botulinum toxin to stop the action of the lip levators might be sufficient. In more advances cases, surgical intervention might be needed through orthognathic surgery, mainly with anterior impaction of the maxilla.

Note: Gummy smile can be a result of short upper lip (soft tissue problem).

1.3.7 Skeletal Posterior Crossbite





History Developmental or hereditary skeletally narrow maxilla.

Exam Dentally: Posterior teeth in crossbite.

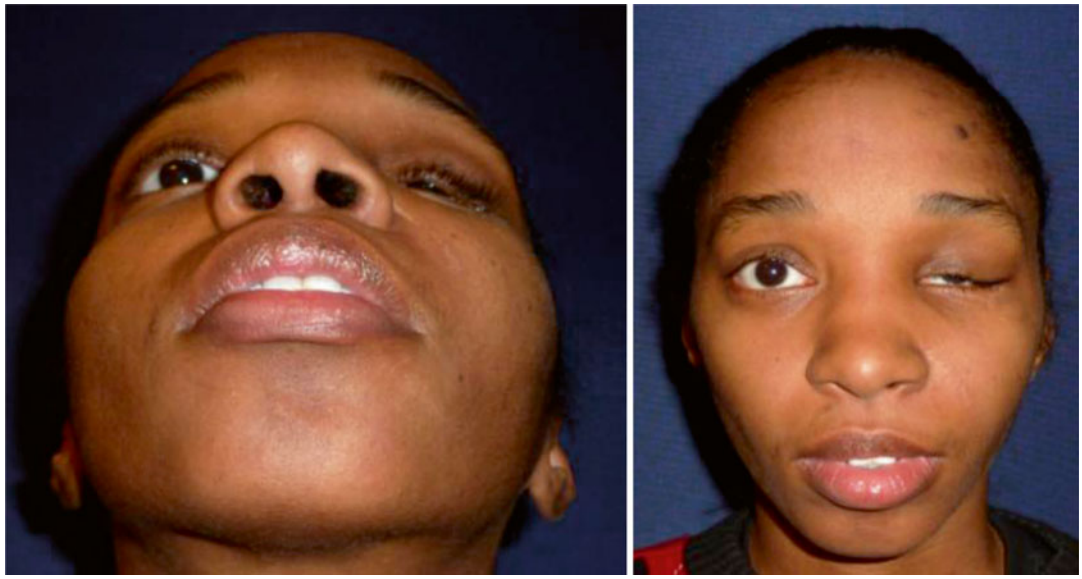
Skeletally: Skeletally narrow maxilla.

Surgical Treatment Expanding the maxilla surgically can be achieved by one of the following procedures:

- Three-piece maxilla Le Fort I (if the maxilla has anterior-posterior or vertical skeletal problems with the transverse problem)
- Surgically assisted rapid palatal expansion (SARPE), if skeletal width deficiency is the only skeletal problem in the maxilla

1.4 Craniofacial, Aesthetic

1.4.1 Eyelid Ptosis

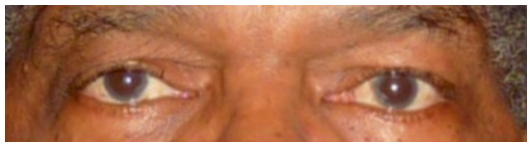


History The most common cause of ptosis in the elderly is senile ptosis characterized by dehiscence of the levator aponeurosis. Other causes are congenital, as seen in young patients, or neurogenic. Neurogenic causes include myasthenia gravis (MG) presenting as unilateral or bilateral ptosis exaggerated with fatigue or as part of Horner's syndrome in which loss of sympathetic innervations of the cervical ganglion.

Exam Evaluation of the margin crease distance (MCD), the levator function, and the degree of ptosis (margin reflex distance) is essential. In senile ptosis, patients have a positive Nesi sign (the iris shadow is visualized with eyelid closure) and elevation of the supratarsal crease. In congenital ptosis, patients have a moderate-to-severe sagging of the eyelids with absence of the eyelid crease. Pharmacologic testing with neostigmine establishes the diagnosis of MG. In Horner's syndrome, patients present with ptosis, miosis, and anhidrosis.

Treatment Severe ptosis with poor levator function is managed surgically with a frontalis sling. Moderate ptosis with levator dehiscence is treated with a levator resection and suspension procedure. Mild cases of ptosis with a good levator excursion can benefit from a mullerectomy or a tarsoconjunctival resection procedure. Myasthenia gravis is treated medically with cholinesterase inhibitors.

1.4.2 Dermatochalasis

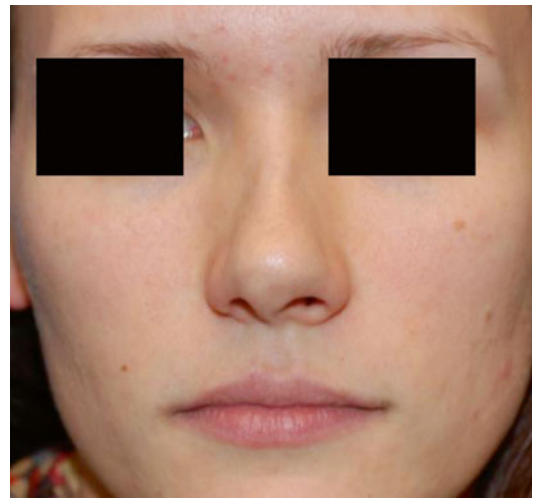


History Beer first described the condition of excess upper eyelid skin that is now known as dermatochalasis. This cosmetic deformity is common in middle age with loss of elasticity secondary to the aging process. It should not be confused with blepharochalasis, a condition caused by repeated episodes of extreme eyelid edema resulting in chronic puffy eyelids.

Exam Baggy eyelids with skin redundancy and hooding of the upper eyelids bilaterally.

Treatment Rejuvenation of the upper eyelids is achieved with a blepharoplasty procedure, combining a muscle with skin or skin only excision. Management of the distended orbital fat compartments involves a conservative excision of the adipose tissue in order to avoid the appearance of a hallowed orbit.

1.4.3 Inverted V Deformity

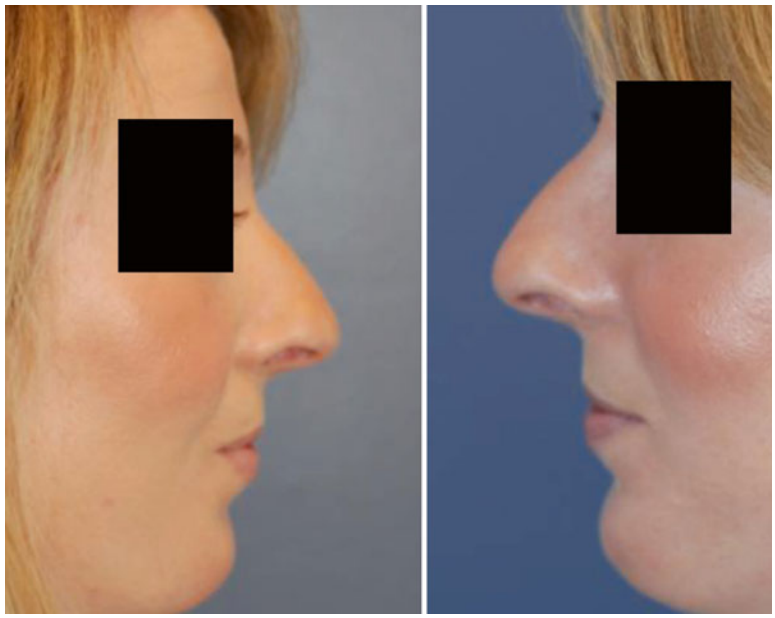


History A patient with a history of rhinoplasty presents with narrowing or collapse of the lateral walls, resulting in a visible inverted V deformity together with nasal airway obstruction.

Exam It is the result of avulsion of the upper lateral cartilages or excessive removal of the transverse portion of the upper lateral cartilage during dorsal septal resection. The internal nasal valve function is compromised as well. A Cottle maneuver significantly improves the patient's breathing.

Treatment Surgical management by placement of spreader grafts will correct the deformity as well as maintain patency of the internal valve.

1.4.4 Supratip/Pollybeak Deformity

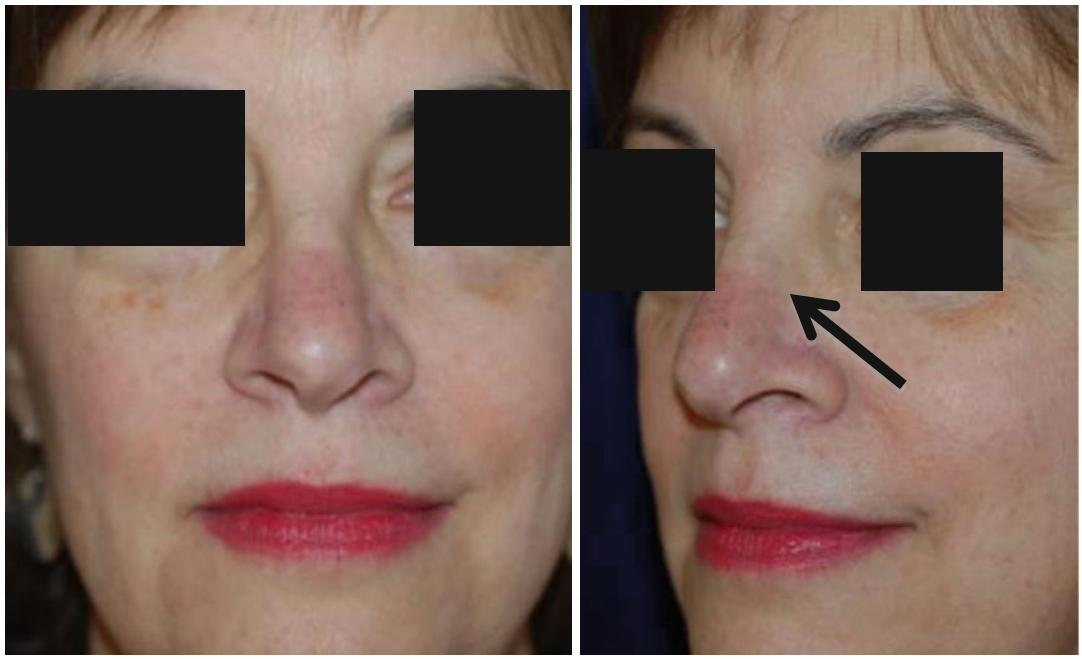


History A patient presents with a nasal deformity where the nasal tip is pushed down.

Exam A supratip/pollybeak deformity (or a parrot's beak) is characterized by supratip fullness from inadequate resection of the lower dorsal septum or over-resection of the supratip structures with subsequent scar tissue formation. The nasal supratip assumes a convex shape in relation to the nasal dorsum. This fullness, just cephalad to the tip, distorts the dorsal aesthetic lines and obscures tip definition.

Treatment Immediate postoperative fullness is managed with compressive taping. Steroid injections can be helpful in cases of persistent fullness following a period of taping. Fullness resistant to conservative therapy can be offered surgery but not until at least 1 year after the initial surgery. When over-resection is the cause, increasing tip projection with cartilage grafts can correct the deformity.

1.4.5 Rocker Deformity

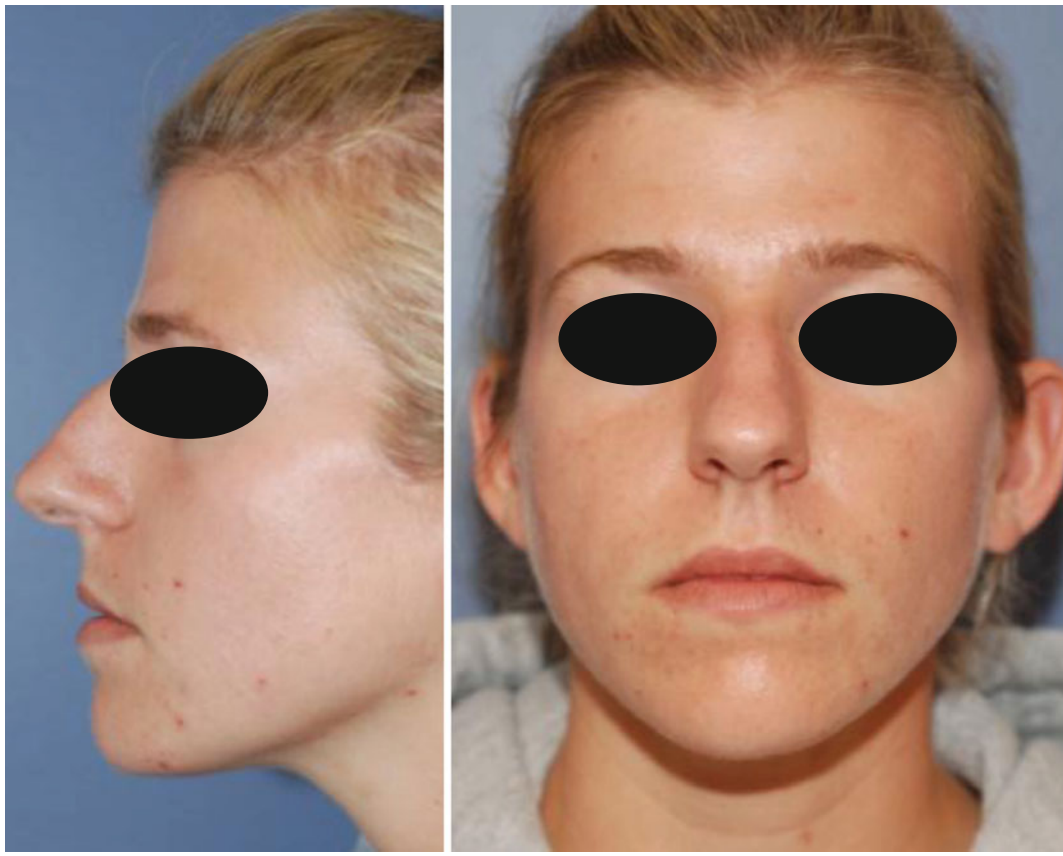


History A deformity of the nasal radix resulting from the medial osteotomy following an inward curve and ending in the thick bone of the nasal root. As the osteotome travels more cephalad into the root, the thicker the nasal bone, the more difficult it is to outfracture. The highly placed medial osteotomy that connects with the lateral osteotomy allows a heavy upper nasal bone to be included in the lateral segment, causing the caudal ends of the nasal bones to migrate medially and the upper portion to "rock out."

Exam The upper dorsum appears wider with the fulcrum of the deformity in the thick, immobile radix.

Treatment In order to avoid a "rocker deformity," the medial osteotomy is canted in a superior oblique manner, taking care not to extend above the intercanthal line. By reaching close enough to the lateral osteotomy, the intervening bone can be greenstick fractured at the intercanthal line.

1.4.6 Dorsal Hump

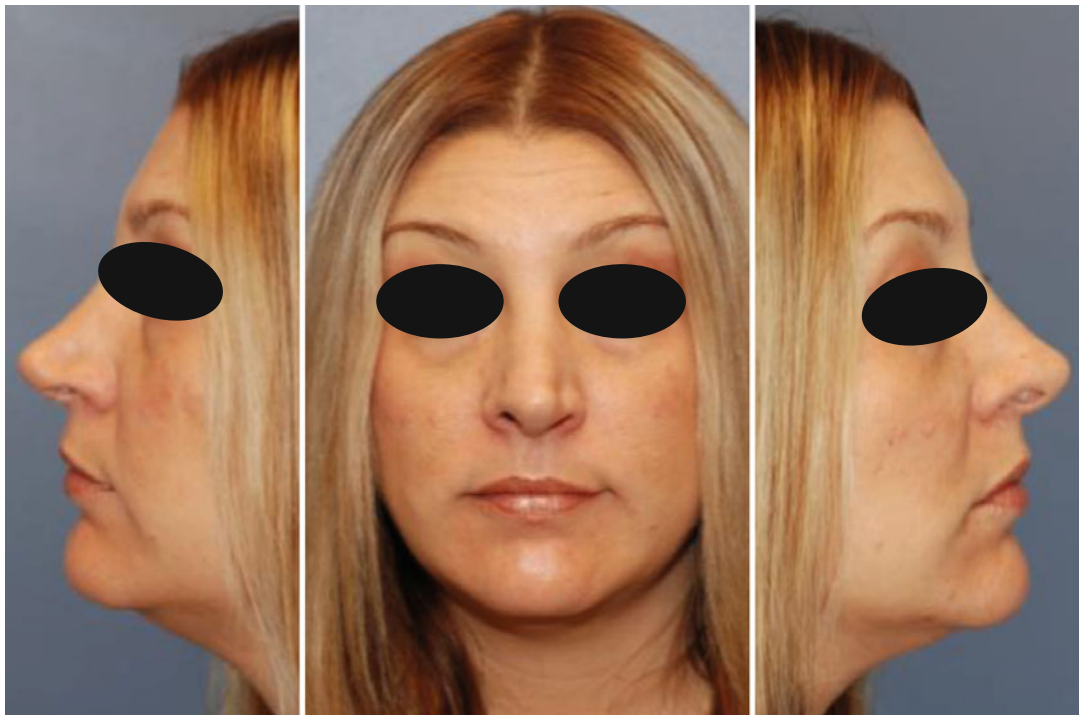


History A patient presents with convex nasal deformity at the nasal dorsum.

Exam A dorsal hump refers to the presence of convexity at the nasal dorsum, producing an aesthetic deformity. It may be due to cartilage or nasal bone or a combination of both. In women, an aesthetic nasal dorsum lies 2 mm behind a line drawn from the radix to the tip defining point, and therefore, a convexity in this location is not ideal.

Treatment The skin and soft tissue envelope is dissected in the suprapericondrial plane so as to expose the upper lateral cartilage (ULCs). Once the bony pyramid is encountered, dissection is continued in the subperiosteal plane to expose the area of the dorsal hump. Next, the ULC is separated from the septum. The dorsal cartilaginous septum is reduced by incrementally shaving off the cartilage contributing to the dorsal hump. The bony dorsum is reduced with a rasp. The skin envelope is then redraped, and the dorsum is inspected and palpated. Finally, any other modifications can be made. Notably, ULC reduction is often not required, but if necessary, should be done last.

1.4.7 Retracted Ala

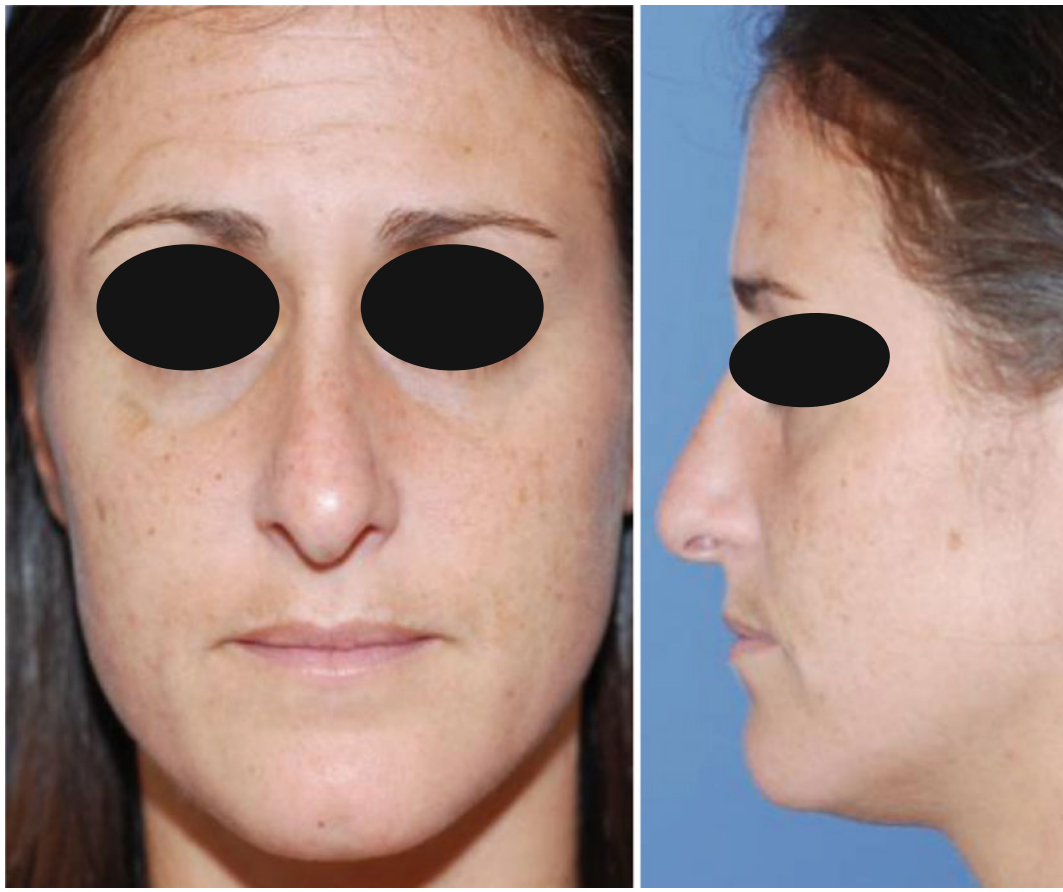


History A retracted, high-arched, or notched ala may be congenital, the result of alar interruption, or iatrogenic from rhinoplasty suture techniques that attempt to reshape the lateral crus.

Exam On lateral view, the alar rim should be within 1.5–2 mm to a line connecting the apex to the nadir of the nostril. If this distance is greater, the ala is retracted. If it is less, the ala is overhanging.

Treatment For mild to moderate alar retraction of up to 1.5 mm, nonanatomic alar rim grafts, using septal, conchal, or costal cartilage, are useful. Severe retraction requires either the addition of a lateral crural strut graft, or a composite graft, or an internal V-Y advancement.

1.4.8 Overhanging Columella

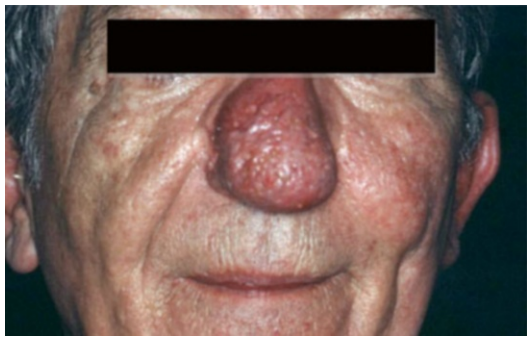


History An overhanging columella is a deformity where the caudal border of the columella appears to be downwardly bowing secondary to a disproportion of the nasal ala and the columella. Most commonly this is a congenital condition. A true overhanging columella is differentiated from a pseudo hanging columella, which is a secondary defect that occurs after overaggressive trimming of the lateral alar cartilages or scar contracture that develops in the lateral alar cartilages after rhinoplasty.

Exam The normal relationship between the ala and the columella is such that the greatest distance from the long axis of the nostril to either the columella or alar rim should be between 1 and 2 mm in a horizontal line drawn between the inferior and superior points of an oval nostril. A true hanging columella is identifiable by a curvature of the columella that is greater than the curvature of the alar rim and characteristically has an acute subnasal angle. A pseudo hanging columella is defined as having a more severe curvature of the caudal alar rim than the columella and accompanied by an obtuse subnasal angle.

Treatment Treatment of a true hanging columella is most commonly performed using a complete transfixion incision. The excess medial crura is exposed by dissecting the nasal lining. The cephalad margins of the medial crura are exposed, and a C-shaped crescent of cartilage is trimmed bilaterally as needed from the cranial aspect of the exposed medial crura. Routine use of sutures to secure the medial crura after excision is not necessary. If part of the nasal lining needs to be excised due to the redundancy that remains after cartilage excision, it is done so at this point. The lining is then reapproximated to repair the transfixion incision.

1.4.9 Rhinophyma

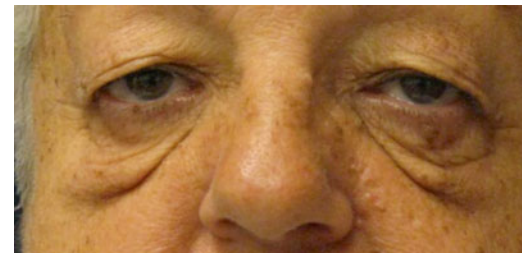


History A (Caucasian male) patient in the 50s–70s presents with tuberous enlargement of the lower half of the nose.

Exam The nasal tip is enlarged with areas of erythema and telangiectasia. The skin is irregularly thickened with prominent follicles and inspissated sebum. The excess growth is due to enlargement of the sebaceous glands and surrounding connective tissue, and the lymphedema is associated with late rosacea. Malignant degeneration is rare. In severe cases, the skin is scarred with fissures and pits. The bony and cartilaginous infrastructure is rarely distorted.

Treatment Tangential excision by shaving, dermabrasion, or laser therapy is the treatment of choice; the wound will heal by second intention.

1.4.10 Festoons



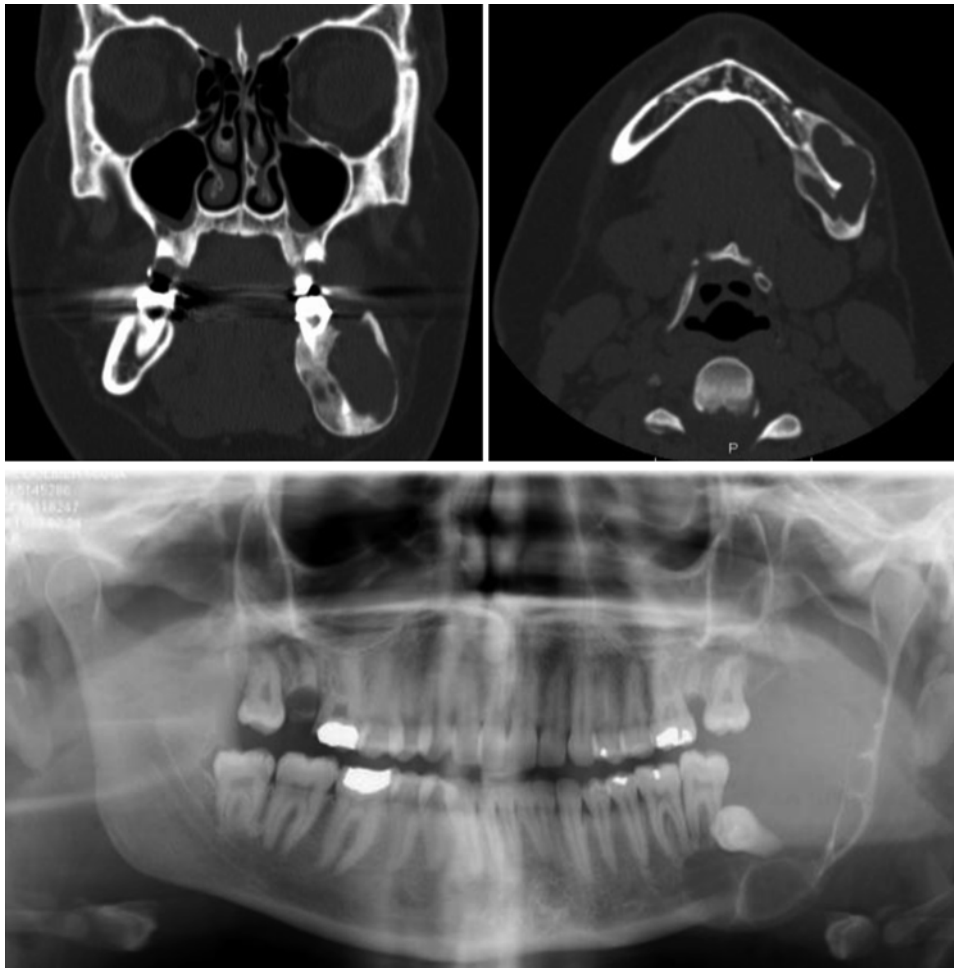
History A patient presents with swollen mounds on the cheeks.

Exam Characterized by Furnas in 1978, festoons are cascading hammocks of loose skin, orbicularis muscle, and possibly herniated fat, which hang from the medial to lateral canthi over the malar eminence. Festoons are hypothesized to be the result of multifactorial processes, such as aging-related structural changes and lymphatic imbalance. They can pose a challenge in creating a smooth lid-cheek junction during periorbital rejuvenation. The "squinch test" is performed when the patient is instructed to squeeze the eyelids together tightly to determine the degree of muscle involvement responsible for the festoon and observe the extent of fat suspended in the festoon. Pinching the skin of the festoon at different sites as the patient makes facial expressions allows the surgeon to determine the degree of orbicularis oculi involvement (the pinch test).

Treatment Depending on the degree of severity, festoons can be addressed with several different approaches, including lower blepharoplasty via subciliary incision with skin-muscle flap dissection and orbitomalar ligament release, midface lift techniques, and even direct excision. A lid-anchoring procedure is often also utilized concomitantly.

1.5 Tumors

1.5.1 Ameloblastoma



History Painless swelling leading to progressive facial deformity.

Exam Ameloblastoma is a rare, benign yet locally aggressive odontogenic neoplasm. It preferentially occurs in the vicinity of the mandibular molars or ramus, equally affecting males and females most often in the third to fourth decades of life. X-ray shows uni- or multi-locular cystic masses associated with thinning of the surrounding bone. Biopsy of the lesion shows palisading odontogenic cells.

Treatment Segmental mandibular resection, including a margin of normal bone and any adjacent teeth, followed by immediate reconstruction is the mainstay of treatment.

References

Deformational Plagiocephaly

1. Seruya M, Oh AK, Taylor JH, Sauerhammer TM, Rogers GF. Helmet treatment of deformational plagiocephaly: the relationship between age at initiation and rate of correction. Plast Reconstr Surg. 2013;131(1):55e–61.

Diagnosis: Anterior Plagiocephaly (Unilateral Coronal Synostosis (UCS)

2. Bartlett SP, Whitaker LA, Marchac D. The operative treatment of isolated craniofacial dysostosis (plagiocephaly): a comparison of the unilateral and bilateral techniques. Plast Reconstr Surg. 1990;85:677.
3. Brunetau RJ, Mulliken JB. Frontal plagiocephaly: synostotic, compensational, or deformational. Plast Reconstr Surg. 1992;89:21.
4. Claren SK, Smith DW, Hanson JW. Helmet treatment for plagiocephaly and congenital muscular torticollis. J Pediatr. 1979;94:43.

Diagnosis: Scaphocephaly (Sagittal Synostosis)

5. Marsh JL, Jenny A, Galic M, et al. Surgical management of sagittal synostosis: a quantitative evaluation of two techniques. Neurosurg Clin N Am. 1991;3:629.
6. Posnick JC, Lin KY, Chen P, et al. Sagittal synostosis: quantitative assessment of presenting deformity and surgical results based on CT scans. Plast Reconstr Surg. 1993;92:1015.

Diagnosis: Trigonocephaly (Metopic Synostosis)

7. Posnick JC, Lin KY, Chen P, et al. Metopic synostosis: quantitative assessment of presenting deformity and surgical results based on CT scans. Plast Reconstr Surg. 1994;93:16.
8. Sidotti EJ, Marsh JL, Marty-Grames L, et al. Long-term studies of metopic synostosis: frequency of cognitive impairment and behavioral disturbances. Plast Reconstr Surg. 1996;97:276.

Diagnosis: Kleeblattschadel Skull Deformity

9. Holtermueller K, Wiedemann H. The clover-leaf skull syndrome. Med Monastsschr. 1960;14:439–46 (Ger).
10. Manjila S, et al. History of the Kleeblattschadel deformity: origin of concepts and evolution of management in the past 50 years. Neurosurg Focus. 2010;29(6):1–8.

Diagnosis: Turricephaly (or Oxycephaly)

11. Mulliken JB, Seinberger D, Kunze S, Muller U. Molecular diagnosis of bilateral coronal synostosis. Plast Reconstr Surg. 1999;104:1603–15.
12. Wagner JD, Cohen SR, Maher H, Dauser RC, Newman MH. Critical analysis of results of craniofacial surgery for nonsyndromic bicoronal synostosis. J Craniofac Surg. 1995;6:32–7.

Diagnosis: Apert Syndrome

13. Fearon JA. The treatment of the hands and feet in Apert syndrome: an evolution in management. Plast Reconstr Surg. 2003;112:1.
14. Fearon JA. Halo-distraction of the Le Fort III in syndromic craniosynostosis: a long-term assessment. Plast Reconstr Surg. 2005;115:1524.
15. Posnick JC. Monobloc and facial bipartition osteotomies: a step-by-step description of the surgical techniques. J Craniofac Surg. 1996;3:229.
16. Raulo Y, Tessier P. Frontofacial advancement for Crouzon and Apert syndromes. Scand J Plast Reconstr Surg. 1995;53:358.

Diagnosis: Crouzon Syndrome

17. Fearon JA. Halo-distraction of the Le Fort III in syndromic craniosynostosis: a long-term assessment. Plast Reconstr Surg. 2005;115:1524.
18. Posnick JC. Monobloc and facial bipartition osteotomies: a step-by-step description of the surgical techniques. J Craniofac Surg. 1996;3:229.
19. Posnick JC, Lin KY, Jhawar BJ, et al. Crouzon syndrome: quantitative assessment of presenting deformity and surgical results based on CT scans. Plast Reconstr Surg. 1993;92:1027.
20. Raulo Y, Tessier P. Frontofacial advancement for Crouzon and Apert syndromes. Scand J Plast Reconstr Surg. 1995;53:358.

Diagnosis: Pfeiffer Syndrome

21. Forrest CR, Hopper RA. Craniofacial syndromes and surgery. Plast Reconstr Surg. 2013;131(1):86e–109.

Diagnosis: Carpenter Syndrome

22. Carpenter G. Case of acrocephaly with other congenital malformations. Proc R Soc Med Sect Study Dis Child. 1909;2:45–53.
23. Cottrill C, et al. Carpenter Syndrome. J Craniofac Surg. 2009;20(1):254–6.
24. Perlyn CA, Marsh JL. Craniofacial Dysmorphology of Carpenter Syndrome: Lessons from Three Affected Siblings. Plast Reconsr Surg. 2008;121:971.

Diagnosis: Stickler Syndrome

25. Antunes RB, et al. Importance of early diagnosis of Stickler syndrome in newborns. J Plast Reconstr Aesthet Surg. 2012;65(8):1029–34.
26. Rose PS, et al. Stickler syndrome: clinical characteristics and diagnostic criteria. Am J Med Genet. 2005;9999:1–9.
27. Stickler GB, et al. Hereditary progressive arthroophthalmopathy. Mayo Clin Proc. 1968;40:433.

Diagnosis: Van der Woude Syndrome

28. Rizos M, Spyropoulos M. Van der Woude syndrome: a review. Cardinal signs, epidemiology, associated features, differential diagnosis, expressivity, genetic counseling and treatment. Eur J Orthod. 2004;26:17–24.
29. Van der Woude A. Fistula labii inferioris congenital and its association with cleft lip and palate. Am J Hum Genet. 1954;6:244.

Diagnosis: Binder Syndrome (Maxillonasal Dysplasia)

30. Von Binder K. Dysostosismaxillo-nasalis, einarhinencephalermissbildungscomplex. Dtsh Zahnaerztl. 1962;17:438.

Diagnosis: Saethre-Chotzen (Acrocephalosyndactyly Type III)

31. Chozten F. Eine eigenarttige familiaere Entwicklungsstoerung (Akrocephalosyndaktylie, Dysostosis craniofacialis und Hypertelorismus). Mschr Kinderhielk. 1932;55:97–122.
32. Cohen MM. Saethre-Chotzen syndrome. In: Cohen MM, Maclean RE, editors. Craniosynostosis: diagnosis, evaluation, and management. 2nd ed. New York: Oxford Univ Press; 2000. p. 374–6.
33. Saethre M. Ein Beitrag zum Turmschaedelproblem (Pathogenese, Erblichkeit und Symptomatologie). Dtsch Z Nerveheilk. 1931;119:533–333.

Diagnosis: Nager Syndrome

34. Meyerson MD, et al. Nager acrofacial dysostosis: early intervention and long-term planning. Cleft Palate J. 1977;14:35–40.
35. Nager FR, de Reynier JP. Das Gehoerorgan bei den angeborenen Kopfmissbildungen. Pract Otorhinolaryng. 1948;10(Supp 2):1–128.

Diagnosis: Möbius Syndrome

36. Amer TA. A single hypoglossal nerve for bilateral smile reconstruction in Möbius syndrome. J Craniofac Surg. 2010;21(6):1926–7.
37. Marre D, Hontanilla B. Brain plasticity in Möbius syndrome after unilateral muscle transfer. Ann Plast Surg. 2012;68(1):97–100.
38. Terzis JK, Noah EM. Dynamic restoration in Möbius and Möbius-like patients. Plast Reconstr Surg. 2003;111(1):40–55.

Diagnosis: Frontonasal Dysplasia/ Median Cleft Face Syndrome

39. Sedano H, et al. Frontonasal dysplasia. J Pediatr. 1970;76(6):906–13.
40. Sisco M, Bauer B. Craniofacial syndromes and sequences. In: Bentz M, Bauer B, Zuker R, editors. Principles and practice of pediatric plastic surgery. St. Louis: Quality Medial Publishing, Inc; 2008. p. 401–3.
41. Tessier P. Anatomical classification of facial, craniofacial, and latero-facial clefts. J Maxillofac Surg. 1976;4(2):69–92.

Fibrous Dysplasia

42. Fattah A, et al. Pediatric craniofacial fibrous dysplasia: the Hospital for Sick Children experience and treatment philosophy. J Plast Reconstr Aesthet Surg. 2013;66(10):1346–55.
43. Gabbay JS, et al. Fibrous dysplasia of the zygomaticomaxillary region: outcomes of surgical intervention. Plast Reconstr Surg. 2013;131(6):1329–38.

Diagnosis: Goldenhar Syndrome

44. Bogusiak K, Arkuszewski P, Skorek-Stachnik K, Kozakiewicz M. J Craniofac Surg. Treatment strategy in Goldenhar syndrome. 2014;25(1):177–83.
45. Gosain AK, McCarthy JG, Pinto RS. Plast Reconstr Surg. Cervicovertebral anomalies and basilar impression in Goldenhar syndrome. 1994;93(3):498–506.
46. Kokavec R. Cleft Palate Craniofac J. Goldenhar syndrome with various clinical manifestations. 2006;43(5):628–34.

Diagnosis: Progressive Hemifacial Atrophy/Romberg Disease/Parry-Romberg Syndrome

47. Hu J, et al. Combined skeletal and soft tissue reconstruction for severe Parry-Romberg syndrome. J Craniofac Surg. 2011;22(3):937–41.
48. Slack GC, et al. Parry-Romberg reconstruction: optimal timing for hard and soft tissue procedures. J Craniofac Surg. 2012;23(7 Suppl 1):1969–73.
49. Upton J, et al. The use of scapular and parascapular flaps for cheek reconstruction. Plast Reconstr Surg. 1992;90:959–71.

Diagnosis: Unilateral Cleft Lip

50. Burt JD, Byrd HS. Cleft lip: unilateral primary deformities. Plast Reconstr Surg. 2000;105(3):1043–55.
51. Hopper RA, Cutting C, Grayson B. Cleft lip and palate. In: Thorne CH, editor. Grabb & Smith's plastic surgery. 6th ed. Philadelphia: Lippincott Williams & Wilkins; 2007. p. 203.
52. Sullivan W. Cleft lip with or without cleft palate in blacks: an analysis of 81 patients. Plast Reconstr Surg. 1989;84(3):406–8.
53. Witkop C. Cleft lip with or without cleft palate. In: Bergsma D, editor. Birth defects compendium. New York: Wiley; 1979. p. 223.
54. Yuzuriha S, Mulliken JB. Minor-form, microform, and min-microform cleft lip: anatomical features, operative techniques and revisions. Plast Reconstr Surg. 2008;122(5):1485–93.

Diagnosis: Bilateral Cleft Lip

55. Fisher DM, Sommerlad BC. Cleft lip, cleft palate, and velopharyngeal insufficiency. Plast Reconstr Surg. 2011;128(4):342e–60.

Diagnosis: Primary Cleft Palate

56. Afifi GY, Kaidi AA, Hardesty RA. Cleft palate repair. In: Evans GRD, editor. Operative plastic surgery. New York: McGraw Hill; 2000. p. 487–98.
57. Gosian AK, Moore FO. Embryology of the head and neck. In: Aston SJ, Beasley RW, Thorne CH, editors. Grabb & Smith's plastic surgery. 5th ed. Philadelphia: Lippincott-Raven; 1997. p. 223–36.
58. Morris HL, Bardach J, Ardinger H, et al. Multidisciplinary treatment results for patients with isolated cleft palate. Plast Reconstr Surg. 1993;92(5):842–51.

Diagnosis: Submucous Cleft

59. Gosain AK, et al. Submucous cleft palate: diagnostic methods and outcomes of surgical treatment. Plast Reconstr Surg. 1996;97(7):1497–509.
60. Kelly AB. Submucous cleft palate. Br J Plast Surg. 1954;6:264.
61. Nasser M, Fedorowicz Z, Newton T, Nouri M. Interventions for the management of submucous cleft palate. Cochrane Database Syst Rev. 2008;(1).

Diagnosis: Alveolar Cleft

62. Bajaj AK. Management of alveolar clefts. J Craniofac Surg. 2003;14(6):840–6.
63. Calvo AM. Increase in age is associated with worse outcomes in alveolar bone grafting in patients with bilateral complete cleft palate. J Craniofac Surg. 2014;25(2):380–2.
64. Nagasao T. An anatomical study of the three-dimensional structure of the nasal septum in patients with alveolar clefts and alveolar-palatal clefts. Plast Reconstr Surg. 2008;121(6):2074–83.

Diagnosis: Pierre Robin Sequence

65. Mackay D. Controversies in the diagnosis and management of the Robin sequence. J Craniofac Surg. 2011;22:415–20.
66. Robin P. A fall of the base of the tongue considered as a new cause of nasopharyngeal respiratory impairment: Pierre Robin sequence, a translation. 1923. Plast Reconstr Surg. 1994;93:1301–3.

Diagnosis: Craniofacial Clefts

67. Alonso N, Freitas Rda S, de Oliveira e Cruz GA, Goldenberg D, Dall'oglio Tolazzi AR. Plast Reconstr Surg. Tessier no. 4 facial cleft: evolution of surgical

treatment in a large series of patients. 2008;122(5): 1505–13.

Diagnosis: Treacher Collins Syndrome

68. Forrest CR, Hopper RA. Craniofacial syndromes and surgery. Plast Reconstr Surg. 2013;131(1):86e–109.

Diagnosis: Prominent Ear

69. Bauer BS, Song DH, Aitken ME. Combined otoplasty technique: chondrocutaneous conchal resection as the cornerstone to correction of the prominent ear. Plast Reconstr Surg. 2002;110(4):1033–40. discussion 1041.
70. Furnas DW. Correction of prominent ears by conchamastoid sutures. Plast Reconstr Surg. 1968;42(3):189–93.
71. Janis JE, Rohrich RJ, Gutowski KA. Otoplasty. Plast Reconstr Surg. 2005;115(4):60e–72.
72. Mustarde JC. The treatment of prominent ears by buried mattress sutures: a ten-year survey. Plast Reconstr Surg. 1967;39(4):382–6.

Cryptotia

73. Byrd HS, Langevin CJ, Ghidoni LA. Ear molding in newborn infants with auricular deformities. Plast Reconstr Surg. 2010;126(4):1191–200.
74. Mutimer KL, Mulliken JB. Correction of cryptotia using tissue expansion. Plast Reconstr Surg. 1988;81(4):601–4.
75. Paredes Jr AA, Williams JK, Elsahy NI. Cryptotia: principles and management. Clin Plast Surg. 2002;29(2):317–26.
76. Thorne CH, Wilkes G. Ear deformities, otoplasty, and ear reconstruction. Plast Reconstr Surg. 2012;129(4):701e–16.

Microtia

77. Bauer BS. Reconstruction of microtia. Plast Reconstr Surg. 2009;124(1 Suppl)):14e–26.
78. Brent B. The correction of mi-rotia with autogenous cartilage grafts: I. The classic deformity.? Plast Reconstr Surg. 1980;66(1):1–12.
79. Zhang Q, Zhang R, Xu F, Jin P, Cao Y. Auricular reconstruction for microtia: personal 6-year experience based on 350 microtia ear reconstructions in China. Plast Reconstr Surg. 2009;123(3):849–58.

Stahl's Ear

80. Kaplan HM, Hudson DA. A novel surgical method of repair for Stahl's ear: a case report and review of current treatment modalities. Plast Reconstr Surg. 1999;103(2):566–9.
81. Nakajima T, Yoshimura Y, Kami T. Surgical and conservative repair of Stahl's ear. Aesthetic Plast Surg. 1984;8(2):101–7.
82. Ono I, Gunji H, Tateshita T. An operation for Stahl's ear. Br J Plast Surg. 1996;49(8):564–7.
83. Sugino H, Tsuzuki K, Bandoh Y, Tange I. Surgical correction of Stahl's ear using the cartilage turnover and rotation method. Plast Reconstr Surg. 1989;83(1):160–4.

Cup/Constricted Ear

84. Horlock N, Grobbelaar AO, Gault DT. 5-year series of constricted (lop and cup) ear corrections: development of the mastoid hitch as an adjunctive technique. Plast Reconstr Surg. 1998;102(7):2325–32. discussion 2333–25.
85. Kislov R. Surgical correction of the cupped ear. Plast Reconstr Surg. 1971;48(2):121–5.
86. Tanzer RC. The constricted (cup and lop) ear. Plast Reconstr Surg. 1975;55(4):406–15.

Frontal Sinus Fracture

87. Manolidis S, Hollier L. Management of frontal sinus fractures. Plast Reconstr Surg. 2007;120(7 supp 2):S32–48.
88. Rodriguez ED, et al. Twenty-six-year experience treating frontal sinus fractures: a novel algorithm based on anatomical fracture pattern and failure of conventional techniques. Plast Reconstr Surg. 2008;122(5):1850–66.

Orbital Floor: Blow-Out Fracture

89. Cole P, et al. Comprehensive management of orbital fractures. Plast Reconstr Surg. 2007;120 Suppl 2:57S–63.
90. Grant MP, Iliff NT, Manson PN. Strategies for the treatment of enophthalmos. Clin Plast Surg. 1997;24:539.

NOE Fracture

91. Markowitz B, et al. Management of the medial canthal tendon in nasoethmoid orbital fractures: the importance of the central fragment in classification and treatment. Plast Reconstr Surg. 1991;87:843–53.

Nasal Fractures

92. Colton JJ, Beekhuis GJ. Management of nasal fractures. Otolaryngol Clin North Am. 1986;19(1):73–85.
93. Procacci P, Ferrari F, Bettini G, et al. Soccer-related facial fractures: postoperative management with facial protective shields. J Craniofac Surg. 2009;20(1):15–20.
94. Rohrich RJ, Adams Jr WP. Nasal fracture management: minimizing secondary nasal deformities. Plast Reconstr Surg. 2000;106(2):266–73.

Le Fort 2

95. Le Fort R. Etude experimentale sur les fractures de la machoire superieur. Rev Chir Paris. 1901;23:208. 360, 479.
96. Manson P. Some thoughts on the classification and treatment of Le Fort fractures. Ann Plast Surg. 1986;17(5):365–63.
97. Manson P, et al. Toward CT-based facial fracture treatment. Plast Reconstr Surg. 1990;85(2):202–12.
98. Rhea J, Novelline R. How to simplify the CT diagnosis of Le Fort fractures. AJR Am J Roentgenol. 2005;184:1700–5.

Mandibular Fractures: Parasymphyseal

99. Champy M, et al. Mandibular osteosynthesis by miniature screwed plates via a buccal approach. J Maxillofac Surg. 1978;6:14.
100. Ellies 3rd E, Miles BA. Fractures of the mandible: a technical perspective. Plast Reconstr Surg. 2007;120(7 Suppl 2):76S–89.
101. Stacey D, et al. Management of mandible fractures. Plast Reconstr Surg. 2006;117:48e–60.

PAN Face Fracture

102. Kelly K. Sequencing LeFort fracture treatment (Organization of treatment for a panfacial fracture). J Craniofac Surg. 1990;1:168.
103. Manson P, et al. Subunit principles in midface fractures: the importance of sagittal buttresses, soft-tissue reductions, and sequencing treatment of segmental fractures. Plast Reconstr Surg. 1999;103:1287–306.
104. Markowitz BL, Manson PN. Panfacial fractures: organization of treatment. Clin Plast Surg. 1989;16:105.

Orbital Cellulitis

105. Chaudhry IA, Shamsi FA, Elzaridi E, Al-Rashed W, Al-Amri A, Al-Anezi F, Arat YO, Holck DE. Outcome of treated orbital cellulitis in a tertiary eye care center in the middle East. Ophthalmology. 2007;114(2):345–54.
106. Ferguson MP, McNab AA. Current treatment and outcome in orbital cellulitis. Aust N Z J Ophthalmol. 1999;27(6):375–9.
107. Liu IT, Kao SC, Wang AG, Tsai CC, Liang CK, Hsu WM. Preseptal and orbital cellulitis: a 10-year review of hospitalized patients. J Chin Med Assoc. 2006;69(9):415–22.
108. McKinley SH, Yen MT, Miller AM, Yen KG. Microbiology of pediatric orbital cellulitis. Am J Ophthalmol. 2007;144(4):497–501. Epub 2007 Aug 15.

Cauliflower Ear

109. Greywoode JD, Pribitkin EA, Krein H. Management of auricular hematoma and the cauliflower ear. Facial Plast Surg. 2010;26(6):451–5.
110. Stuteville OH, Janda C, Pandya NJ. Treating the injured ear to prevent a “cauliflower ear”. Plast Reconstr Surg. 1969;44(3):310–2.
111. Yotsuyanagi T, Yamashita K, Urushidate S, Yokoi K, Sawada Y, Miyazaki S. Surgical correction of cauliflower ear. Br J Plast Surg. 2002;55(5):380–6.

Entropion

112. Glat PM, Jelks GW, Jelks EB, Wood M, Gadangi P, Longaker MT. Evolution of the lateral canthoplasty: techniques and indications. Plast Reconstr Surg. 1997;100(6):1396–405. discussion 1406–398.
113. Spinelli HM, Tabatabai N, Nunn DR. Correction of involutional entropion with suborbicularis septal and lateral canthal tightening. Plast Reconstr Surg. 2006;117(5):1560–7. discussion 1568–70.
114. Li TG, Shorr N, Goldberg RA. Comparison of the efficacy of hard palate grafts with acellular human dermis grafts in lower eyelid surgery. Plast Reconstr Surg. 2005;116(3):873–8. discussion 879–80.

Ectropion

115. Eliasoph I. Current techniques of entropion and ectropion correction. Otolaryngol Clin North Am. 2005;38(5):903–19.
116. McCord Jr CD, Ellis DS. The correction of lower lid malposition following lower lid blepharoplasty. Plast Reconstr Surg. 1993;92(6):1068–72.
117. Patipa M. The evaluation and management of lower eyelid retraction following cosmetic surgery. Plast Reconstr Surg. 2000;106(2):438–53. discussion 454–39.

Enophthalmos

118. Bite U, Jackson IT, Forbes GS, Gehring DG. Orbital volume measurements in enophthalmos using three-dimensional CT imaging. Plast Reconstr Surg. 1985;75(4):502–8.
119. Hazani R, Yaremchuk MJ. Correction of posttraumatic enophthalmos. Arch Plast Surg. 2012;39:11–7.
120. Manson PN, Ruas EJ, Iliff NT. Deep orbital reconstruction for correction of post-traumatic enophthalmos. Clin Plast Surg. 1987;14(1):113–21.

Chemosis

121. Weinfeld AB, Burke R, Codner MA. The comprehensive management of chemosis following cosmetic lower blepharoplasty. Plast Reconstr Surg. 2008;122(8):579–86.

Retrobulbar Hematoma

122. Callahan MA. Prevention of blindness after blepharoplasty. Ophthalmology. 1983;90(9):1047–51.
123. Tenzel RR. Complications of blepharoplasty. Orbital hematoma, ectropion, and scleral show. Clin Plast Surg. 1981;8(4):797–802.

Superior Orbital Fissure Syndrome

124. Chen CT, Wang TY, Tsay PK, Huang F, Lai JP, Chen YR. Traumatic superior orbital fissure syndrome: assessment of cranial nerve recovery in 33 cases. Plast Reconstr Surg. 2010;126(1):205–12.
125. Salin MB, Smith BM. Diagnosis and treatment of midface fractures. In: Fonseca RJ, editor. Oral and maxillofacial trauma. 3rd ed. St. Louis: Elsevier; 2005. p. 682.

Zygomaticomaxillary Complex (ZMC) Fracture

126. Kelley P, Hopper R, Gruss J. Evaluation and treatment of zygomatic fractures. Plast Reconstr Surg. 2007;120:5S–15.
127. Manson P, Markowitz B, Mirvis S, Dunham M, Yaremchuk M. Toward CT-based facial fracture treatment. Plast Reconstr Surg. 1990;85(2):202–12.

Facial Nerve Injury

128. Ghali S, MacQuillan A, Grobbelaar AO. Reanimation of the middle and lower face in facial paralysis: review of the literature and personal approach. J Plast Reconstr Aesthet Surg. 2011;64(4):423–31.
129. Sardesai MG, Moe KS. Recent progress in facial paralysis: advances and obstacles. Curr Opin Otolaryngol Head Neck Surg. 2010;18(4):266–71.

Parotid Injury

130. Lewkowicz AA, Hasson O, Nabelieli O. Traumatic injuries to the parotid gland and duct. J Oral Maxillofac Surg. 2002;60:676–80.
131. Van Sickels JE, Alexander JM. Parotid duct injuries. Oral Surg Oral Med Oral Pathol. 1981;52:364–7.
132. Van Sickels JE. Management of parotid gland and duct injuries. Oral Maxillofac Surg Clin North Am. 2009;21(2):243–6.

Microgenia

133. Polido WD, de Clairefont RL, Bell WH. Bone resorption, stability, and soft tissue changes following large chin advancements. J Oral Maxillofac Surg. 1991;49:251–6.
134. Proffit WR, White RP, Sarver DMD. Contemporary treatment of dentofacial deformity. St. Louis: Mosby; 2003. p. 312–44.
135. Yaremchuk MJ. Improving aesthetic outcomes after alloplastic chin augmentation. Plast Reconstr Surg. 2003;112(5):1422–32.

Prenormal Skeletal and Dental Relationship (Angle Class II)

136. Proffit WR, Vig KWL, Turvey TA. Early fracture of the mandibular condyle: frequently an unsuspected cause of growth disturbances. Am J Orthod. 1980;78:1–21.

137. Proffit WR, White RP, Sarver DMD. Contemporary treatment of dentofacial deformity. St. Louis: Mosby; 2003. p. 312–44.

Postnormal Skeletal and Dental Relationship (Angle Class III)

138. Proffit WR, White RP, Sarver DMD. Contemporary treatment of dentofacial deformity. St. Louis: Mosby; 2003. p. 312–44.
139. Sinclair PM. Orthodontic considerations in adult surgical orthodontic cases. Dent Clin North Am. 1988;32:509–28.
140. Vig KD, Ellis 3rd E. Diagnosis and treatment planning for the surgical orthodontic patient. Dent Clin North Am. 1990;34:361–84.

Skeletal Deep Bite

141. Turley PK. Orthodontic management of the short face patient. Semin Orthod. 1996;2(2):138–53.
142. Watted N, Wieber M, Teuscherand T, Bill J, Reuther J. Surgical lengthening of the lower face in patients with class II deformities and skeletal deep bite–"short face syndrome" controlled clinical study on the "Würzburger Konzept". Mund Kiefer Gesichtschir. 2002;6(6):415–20. Epub 2002 Jul 2.

Open Bite

143. Dawson PE. Evaluation, diagnosis, and treatment of occlusal problems. 2nd ed. St Louis: CV Mosby Co; 1989. p. 535–42.
144. Ngan P, Fields HW. Open bite: a review of etiology and management. Pediatr Dent. 1997;19(2):91–8.
145. Proffit WR, White RP, Sarver DMD. Contemporary treatment of dentofacial deformity. St. Louis: Mosby; 2003. p. 312–44.

Vertical Maxillary Excess (Gummy Smile)

146. Sarver D, Weissman S. Long-term soft tissue response to LeFort I maxillary superior repositioning. Angle Orthod. 1991;61(4):267–76.
147. Sarver D. Esthetic orthodontics and orthognathic surgery. St. Louis: Mosby; 1998. p. 168–201.

Skeletal Posterior Crossbite

148. Pogrel MA, Kaban LB, Vangervik K. Surgically assisted rapid maxillary expansion in adults. Int J Adult Orthodon Orthognath Surg. 1992;7:37.
149. Vanarsdale R, White R. Three dimensional analysis for skeletal problems. Int J Adult Orthodon Orthognath Surg. 1994;9:159.

Eyelid Ptosis

150. Cetinkaya A, Brannan PA. Ptosis repair options and algorithm. Curr Opin Ophthalmol. 2008;19(5):428–34.
151. de la Torre JI, Martin SA, De Cordier BC, Al-Hakeem MS, Collawn SS, Vasconez LO. Aesthetic eyelid ptosis correction: a review of technique and cases. Plast Reconstr Surg. 2003;112(2):655–60. discussion 661–52.

Dermatochalasis

152. Conder MA, Mejia JD. Upper eyelid blepharoplasty. In: Nahai F, editor. The art of aesthetic surgery: principles and practice, vol I. 2nd ed. St. Louis: Quality Medical Publishing; 2011. p. 864–5.
153. Trussler AP, Rohrich RJ. MOC-PSSM CME article: blepharoplasty. Plast Reconstr Surg. 2008;121(1 Suppl):1–10.

Inverted V Deformity

154. Rohrich RJ, Hollier LH. Use of spreader grafts in the external approach to rhinoplasty. Clin Plast Surg. 1996;23:255–62.
155. Rohrich RJ, Muzaffar AR, Janis JE. Component dorsal hump reduction: the importance of maintaining dorsal aesthetic lines in rhinoplasty. Plast Reconstr Surg. 2004;114:1298–308.
156. Sheen JH. Spreader grafts: a method of reconstructing the roof of the middle nasal vault following rhinoplasty. Plast Reconstr Surg. 1984;73:230–9.

Supratip/Pollybeak Deformity

157. Guyuron B, DeLuca L, Lash R. Supratip deformity: a closer look. Plast Reconstr Surg. 2000;105:1140–51.
158. Sheen JH, Sheen A. Problems in secondary rhinoplasty, supratip deformity. In: Aesthetic rhinoplasty. 2nd ed. St. Louis: Quality Medical Publishing; 1998. p. 1200–41.

Rocker Deformity

159. Harshbarger RJ, Sullivan PK. The optimal medial osteotomy: a study of nasal bone thickness and fracture patterns. Plast Reconstr Surg. 2001;108(7):2114–9. discussion 2120–11.

160. Sullivan PK, Freeman MB, Harshbarger RJ, Oneal RM, Landecker A. Nasal osteotomies. In: Gunter JP, Rohrich RJ, Adams WPJ, editors. Dallas rhinoplasty, vol. I. 2nd ed. St. Louis: Quality Medical Publishing, Inc; 2007. p. 254.

Dorsal Hump

161. Rohrich RJ, Muzaffar AR, Janis JE. Component dorsal hump reduction: the importance of maintaining dorsal aesthetic lines in rhinoplasty. Plast Reconstr Surg. 2004;114(5):1298–308.

Retracted Ala

162. Ellenbogen R. Alar rim lowering. Plast Reconstr Surg. 1987;79:50.
163. Guyuron B. Alar rim deformities. Plast Reconstr Surg. 2001;107:856.
164. Rohrich RJ, Raniere Jr J, Ha RY. The alar contour graft: correction and prevention of alar rim deformities in rhinoplasty. Plast Reconstr Surg. 2002;109:2495.

Overhanging Columella

165. Bilen BT, Ayetkin AH, Erbatur S, Geyik Y. Correction of true hanging columella using medial crural tuck-up technique. Aesthetic Plast Surg. 2013;37(2):210–5.
166. Gunter JP, Rohrich RJ, Friedman RM. Classification and correction of alar-columellar discrepancies in rhinoplasty. Plast Reconstr Surg. 1996;97(3):643–8.
167. Matarasso A, Greer SE, Longaker MT. The true hanging columella: simplified diagnosis and treatment using a modified direct approach. Plast Reconstr Surg. 2000;106(2):469–74.

Rhinophyma

168. Bogetti P, Boltri M, Spagnoli G, Dolcet M. Surgical treatment of rhinophyma: a comparison of techniques. Aesthetic Plast Surg. 2002;26:57–60.
169. Little SC, Stucker FJ, Compton A, Park SS. Nuances in the management of rhinophyma. Facial Plast Surg. 2012;28(2):231–7.
170. Rohrich RJ, Griffin JR, Adams Jr WP. Rhinophyma: review and update. Plast Reconstr Surg. 2002;110(3):860–9. quiz 870.

Festoons

171. Furnas DW. Festoons of orbicularis muscle as a cause of baggy eyelids. Plast Reconstr Surg. 1978;61:540–6.
172. Kpodzo DS, Nahai F, McCord CD. Malar mounds and festoons: review of current management. Aesthet Surg J. 2014;34(2):235–48.

Ameloblastoma

173. Mendenhall WM, Werning JW, Fernandes R, Malyapa RS, Mendenhall NP. Ameloblastoma. Am J Clin Oncol. 2007;30(6):645–8.
174. Reichart PA, Phillipsen HP, Sonner S. Ameloblastoma: biologic profile of 3677 cases. Eur J Cancer B Oral Oncol. 1995;31B(2):86–99.

Hand

2

Ron Hazani, Alan Yan, Michael J. Yaremchuk, Amir Taghinia, Jesse Jupiter, Simon Talbot, Fuad Hashem, Camela A. Pokhrel, Arun J. Rao, and Angel E. Rivera-Barrios

R. Hazani, MD (✉)
Division of Plastic and Reconstructive Surgery, Massachusetts General Hospital, Harvard Medical School, Boston, Massachusetts, USA
e-mail: info@drhazani.com
http://www.drhazani.com

A. Yan, MD • M.J. Yaremchuk, MD
Division of Plastic and Reconstructive Surgery, Craniofacial Plastic Surgery, Massachusetts General Hospital, Harvard Medical School, Boston, Massachusetts, USA
e-mail: alanyanmd@gmail.com
http://www.dryaremchuk.com

J. Jupiter, MD
Division of Plastic Surgery, Department of Surgery, Massachusetts General Hospital, Harvard Medical School, Boston, MA, USA
e-mail: jjupiter1@partners.org

A. Taghinia, MD
Division of Plastic Surgery, Department of Surgery, Children's Hospital of Boston, Harvard Medical School, Boston, MA, USA
e-mail: amir.taghinia@childrens.harvard.edu

S. Talbot, MD
Division of Plastic Surgery, Department of Surgery, Children's Brigham and Women's Hospital, Harvard Medical School, Boston, MA, USA
e-mail: sgtalbot@partners.org

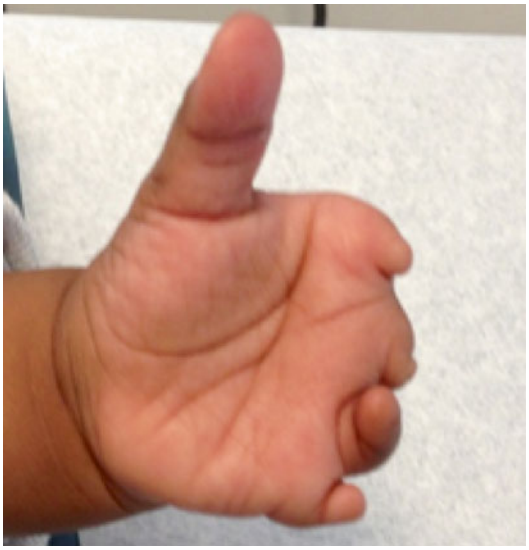
F. Hashem, MD
Department of Plastic Surgery, Department of Surgery, King Faisal Specialist Hospital, Riyadh, Saudi Arabia
e-mail: fuadkhashem@gmail.com

C.A. Pokhrel, MD
Division of Plastic Surgery, Department of Surgery, Kleinert-Kutz Hand Surgery, Louisville, KY, USA
e-mail: crao539@yahoo.com

A.J. Rao, MD
Division of Plastic Surgery, Department of Surgery, Private Practice, Tucson, AZ, USA
e-mail: ajr938@yahoo.com

2.1 Hand, Congenital

2.1.1 Transverse Deficiency



History Patient presents with the absence of a distal portion of the limb with an otherwise normal proximal portion.

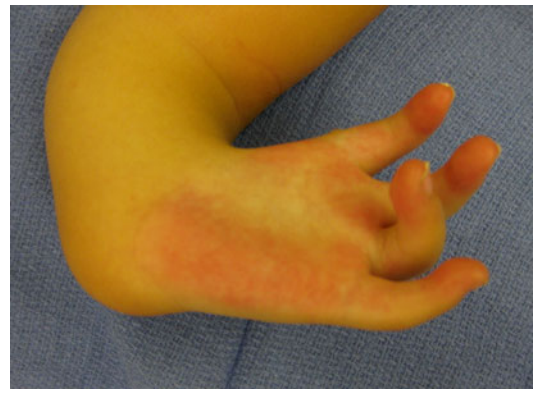
A.E. Rivera-Barrios, MD
Division of Plastic Surgery, Department of Surgery, Medical University of South Carolina, Charleston, SC, USA
e-mail: Barian17@hotmail.com

© Springer International Publishing Switzerland 2016
R. Hazani et al. (eds.), *Clinical Diagnosis in Plastic Surgery*, DOI 10.1007/978-3-319-17094-7_2

Exam This disorder is usually 98 % unilateral, mostly at upper forearm level. If it is bilateral, it may be an AR trait. It is nonsyndromic and is reported to appear with hydrocephalus, spina bifida, meningomyelocele, and clubfoot. It is associated with hypoplasia of proximal muscles which differentiates it from constriction band syndrome.

Treatment Treatment is mostly nonsurgical. Prosthesis adapted to normal hand function according to the age of the patient.

2.1.2 Radial Clubhand

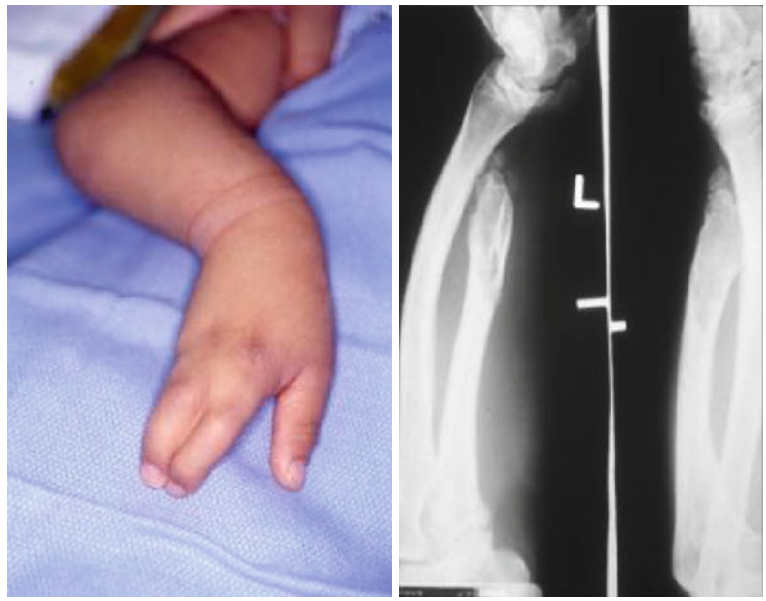


History A form of preaxial deficiency that is typically sporadic and unilateral. Radial dysplasias are more frequent in males, on the right side, and are frequently associated with syndromes including Fanconi anemia, TAR syndrome, Holt-Oram syndrome, and VATER syndrome.

Exam A foreshortened forearm with radial deviation at the wrist. The deformity affects all structures including the bone, tendons, joints, and soft tissue with the absence of the radius as the most severe and most common type.

Treatment Mild forms of dysplasia are treated with splinting. Evaluation and treatment of associated syndromes must be considered prior to surgical reconstruction. Distraction lengthening is recommended for a short or hypoplastic radius. Centralization is performed in cases of severe hypoplasia or the absence of a radius.

2.1.3 Ulnar Deficiency



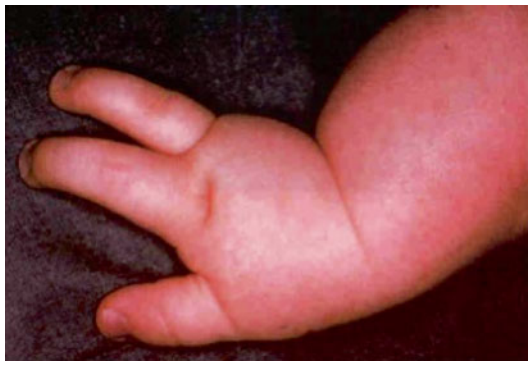
History Patient presents with shortened, bowed forearm.

Ulnar longitudinal deficiency (ULD) (also known as ulnar clubhand) is a lack of formation of the pinky side of the upper extremity. It usually affects the forearm but can affect the hand, forearm, and upper arm. It can affect the bone, muscle, tendon, nerves, and blood vessels. The severity is different in each affected child.

Exam It usually affects the forearm, but the hand and upper arm can be involved as well. The first webspace is often narrowed. There can be syndactyly between other digits, and in more severe cases, a variable number of digits may be absent. Abnormal bony connections between the carpal bones and metacarpals may exist. The ulna is shortened and bowed or absent. The radius usually is usually normal or fused to the humerus (radioulnar synostosis).

Treatment Early stages of treatment include splinting. Surgical treatment entails syndactyly release, digital rotation osteotomies, resection of radial head, and osteotomy of the synostosis.

2.1.4 Cleft Hand

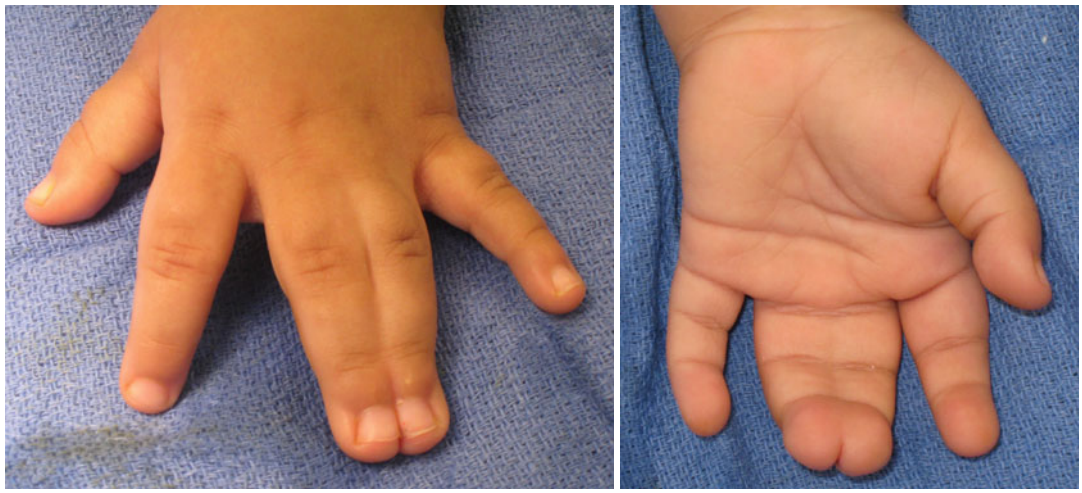


History It is associated with other anomalies including cleft lip and palate congenital heart disease and significant deformities of the upper and lower extremities. It has been named various medical and layperson names such as ectrodactyly, split hand, or lobster claw hand. It is a type I failure of formation—longitudinal arrest. Classified as typical (true cleft hand) or atypical based on physical findings and genetic predilection.

Exam Patients with typical central deficiencies have an absence of the third ray, often with bilateral findings.

Treatment Improving the function and cosmetic appearance of the hand is the goal when the thumb or first webspace is absent. Different procedures have been described: Snow-Littler procedure is designed to create a wide first webspace and to minimize the limitations of a cleft in the hand. Ueba, Miura, and Komada described additional procedures with modifications or simplifications of the Snow-Littler procedure.

2.1.5 Diagnosis: Syndactyly

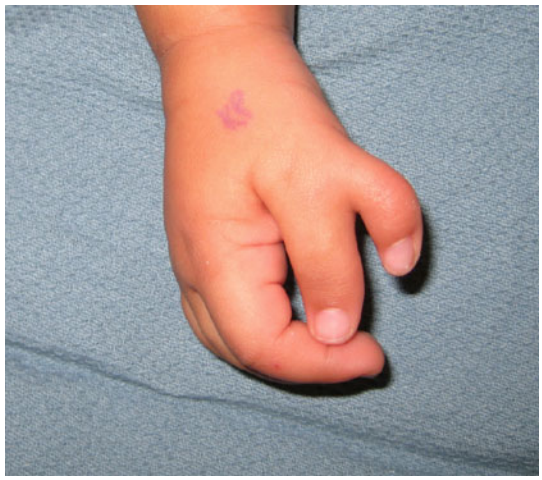


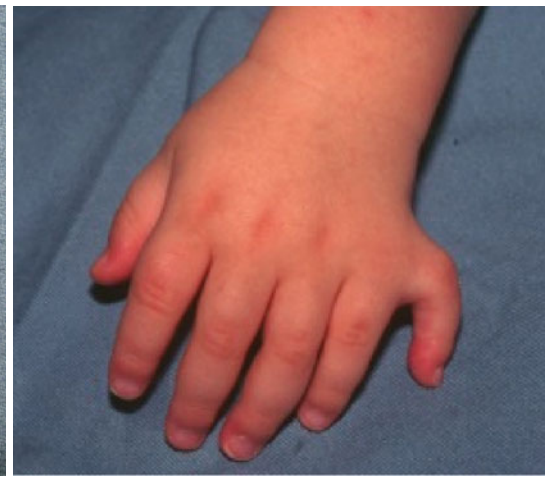
History Syndactyly is the second most common congenital hand anomaly in which 1:2,000 of live births are affected. Syndactyly represents a form of failure of differentiation in the recession process at the webspaces. It commonly affects Caucasian males with the third webspace being the most common site of fusion.

Exam In complete syndactyly, the fusion encompasses the entire webspace. Complex syndactyly implies bony fusion in addition to skin involvement. In complicated syndactyly, other findings are found on the exam, such as polydactyly, symphalangism, or brachydactyly.

Treatment Border digits and complex syndactyly fusions should be addressed early. Separation should not be performed simultaneously on adjacent webs. Separation is carried out with the creation of interdigitating flaps, while the residual raw areas are reconstructed with a skin graft.

2.1.6 Duplicated Thumb (Polydactyly)



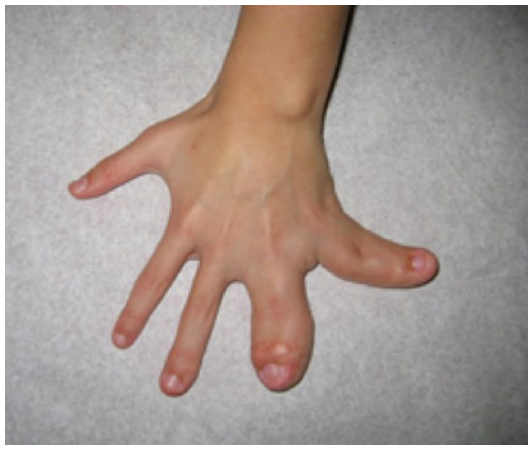


History A radial-sided (preaxial) polydactyly is commonly found in Caucasians, as opposed to the postaxial polydactyly which is more common in African Americans.

Exam The supernumerary digit can be well developed or pedunculated and rudimentary. The Wassel classification (seven types) is used to determine the level of differentiation of the duplicated thumb, and it ranges from a bifid distal phalanx to complete duplication at the CMC joint (types I–VII). Type VII is a triphalangeal thumb.

Treatment Types I–II benefit from a Bilhaut procedure or resection of the radial thumb. In types III–IV, the best phalangeal portions of both thumbs are incorporated to create a thumb. In types V–VI, the radial thumb is amputated with RCL reconstruction.

2.1.7 Macrodactyly



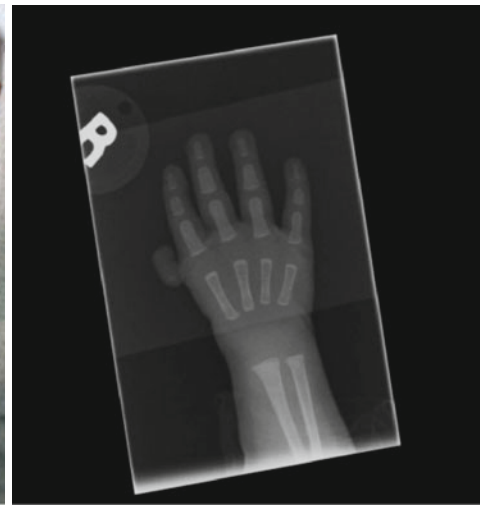
History It is an overgrowth congenital anomaly. Etiology is mostly unknown, but some believe it is related to nerve-stimulated pathology with abnormal neural control in sensory distribution of the median nerve.

Exam Four subtypes have been described. Patients can be born with an enlarged digit. The enlargement of the digit may be associated with signs of neurofibromatosis.

Treatment Skin and subcutaneous resection, extensive neurolysis and resection, epiphysiodesis, arthrodesis, and amputation. Amputation is reserved for the digit that is of no use, significantly enlarged, and a source of embarrassment.

2.1.8 Hypoplastic Thumb

Diagnosis Thumb hypoplasia.

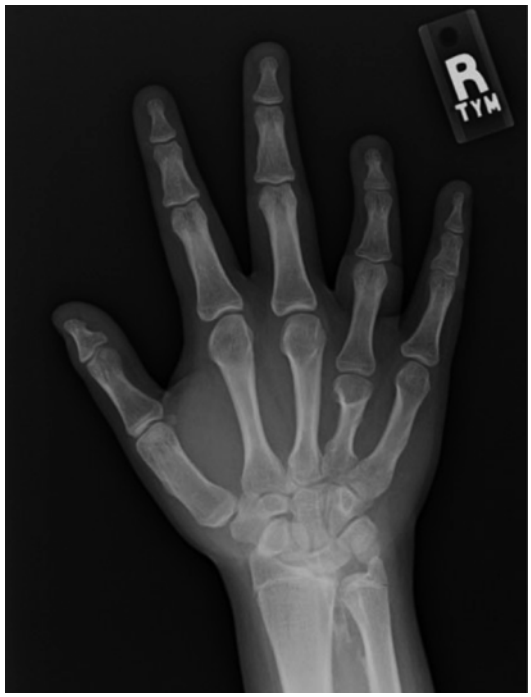


History It is a variant form of undergrowth (class V) involving the radial half of the upper limb and often associated with radial clubhand. Other anomalies are to be ruled out, such as the Holt-Oram syndrome, VACTERL, TAR, and Fanconi anemia.

Exam The thumb presents with small to absent tendons, muscles, bones, and joints. Based on the modified Blauth classification, five types have been described. Type 1 implied a grossly diminished size. In type 2, the first webspace is affected. In type 3, it is necessary to determine if an unstable thumb CMC joint is present as in type 3B in order to guide treatment. Type 4 is a floating thumb (pouce flottant) indicating rudimentary phalanges. In type 5, the thumb is completely absent.

Treatment Types 1–3A require an opponensplasty procedure combined with a first webspace deepening procedure such as a four-limb Z-plasty. In types 3B–5, complete reconstruction with pollicization of the index finger is required.

2.1.9 Brachydactyly

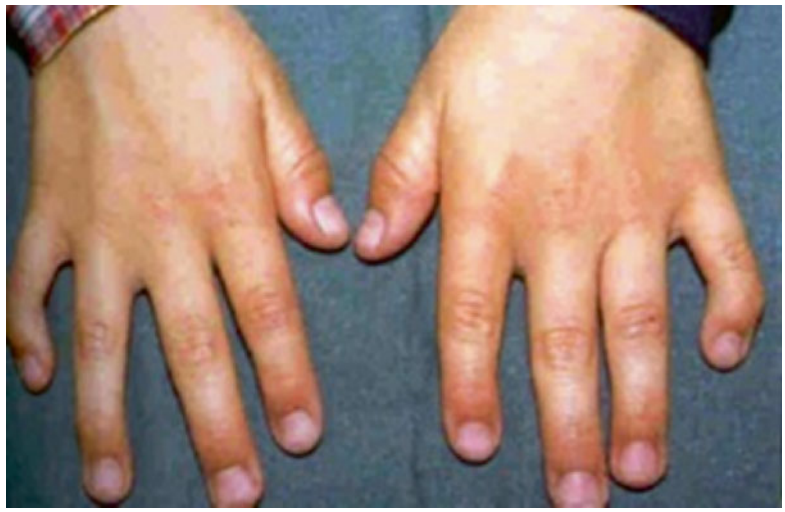


History Brachydactyly can occur as an inherited condition with an autosomal dominant pattern, as a sporadic anomaly, or as part of a syndrome. Brachydactyly can also result as a condition following trauma, infection, or frostbite.

Exam Short digits in which parts of the skeleton are present but reduced in size. The middle phalanx is most affected, while the index and small finger are the most commonly affected digits. Associated findings on exam are syndactyly, clinodactyly, and symphalangism. In Poland syndrome, additional upper extremity abnormalities and chest wall findings are expected.

Treatment Management is based on the degree of shortening and the existing functional deficit. Hand function is usually intact in brachydactyly patients, and the surgical outcome is often disappointing. It is therefore preferred to avoid surgery in patients with short phalanges. Lengthening techniques have been used in selected cases with distraction osteogenesis or immediate lengthening with bone grafts.

2.1.10 Clinodactyly



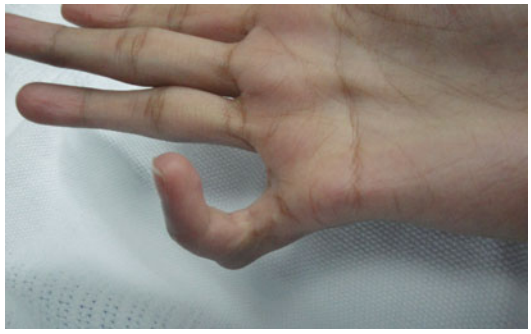
Clinodactyly is a congenital anomaly relating to the failure of differentiation. It is defined as curvature of a digit in a radioulnar plane. The etiology is unknown. It can be inherited in an autosomal dominant fashion with variable expression.

History Deformity may be associated with syndromes such as Apert.

Exam An ulnar angulation of the small finger is seen. A delta phalanx is identified on an X-ray, which is the triangular bracketed-shaped bony wedge in angulated digit. Limited motion is noted when a bracketed phalanx is present.

Treatment Bracket resection with preservation of the growth plate and fat graft or wedge osteotomy. Skin deficit after correction may require Z-plasties. A K-wire is used to hold correction in place.

2.1.11 Diagnosis: Camptodactyly

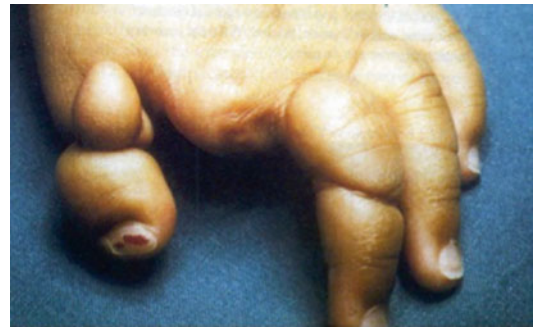


History Camptodactyly is a congenital constriction deformity of the PIP joint and represents a form of soft tissue failure of differentiation. It is present in approximately 1 % of the population with unclear etiologic factors. The function of the hand is rarely impaired. Most patients seek medical attention due to appearance.

Exam Abnormal flexion posture of the PIP joint, most commonly of the little finger.

Treatment Complete correction is difficult to achieve, and surgical management should be discouraged. Nonoperative treatment consists of splinting and stretch exercises. If the contracture is more than 30°, consider release of the involved lumbricals, FDS, and accessory collateral ligaments.

2.1.12 Constriction Band Syndrome (CBS)



History A congenital upper extremity deformity manifested as an intrauterine amputation of the digits, hand, or limb. Amniotic constriction band syndromes have no known genetic transmission. It is frequently associated with anomalies such as clubfoot, cleft lip and palate, craniofacial defects, hemangioma, and meningocele.

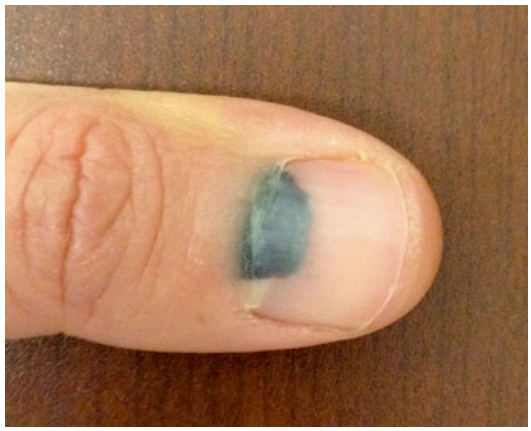
Exam The presentation of CBS varies greatly. Patients may have a spectrum of findings that vary from superficial grooving to complete amputation of a limb.

Treatment Simple constrictions may not require any surgical treatment. More severe cases can be managed with Z-plasties in a staged fashion for release of the soft tissue bands and separation of fused digits to allow for unimpeded grow. Distractions lengthening with bone grafting and toe-to-hand transfers have also been described.

2.2 Hand, Trauma

2.2.1 Soft Tissue

2.2.1.1 Subungual Hematoma

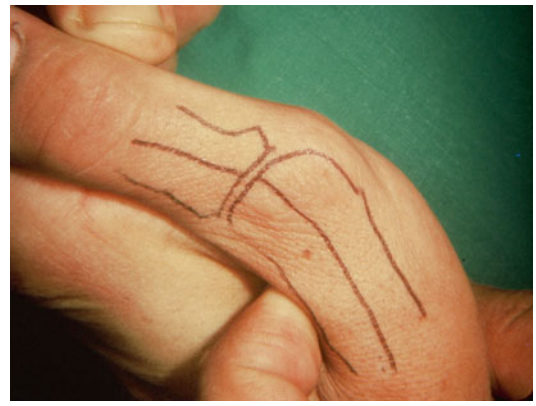


History Trauma to the nail bed.

Exam Throbbing pain is present on the exam because of pressure in the closed space between the nail plate and nail bed.

Treatment Hematoma drainage (trephination) can be done with microcautery device or heated sterile paper clip. The hole should be large enough to allow for prolonged drainage. Further injury to the nail bed should be avoided. Repair of the nail bed may be required, and this can be done by removing the torn nail plate to provide exposure for the repair.

2.2.1.2 Radial Collateral Ligament (RCL) Injury



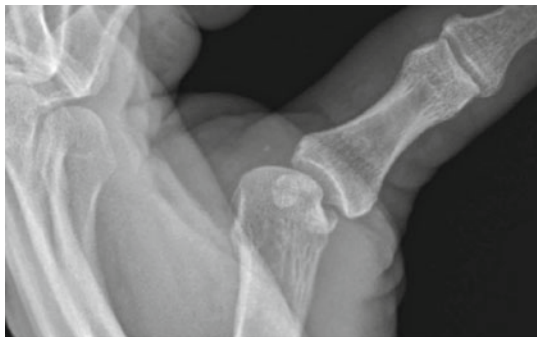
A partial or complete tear of the RCL of the thumb at the MP joint.

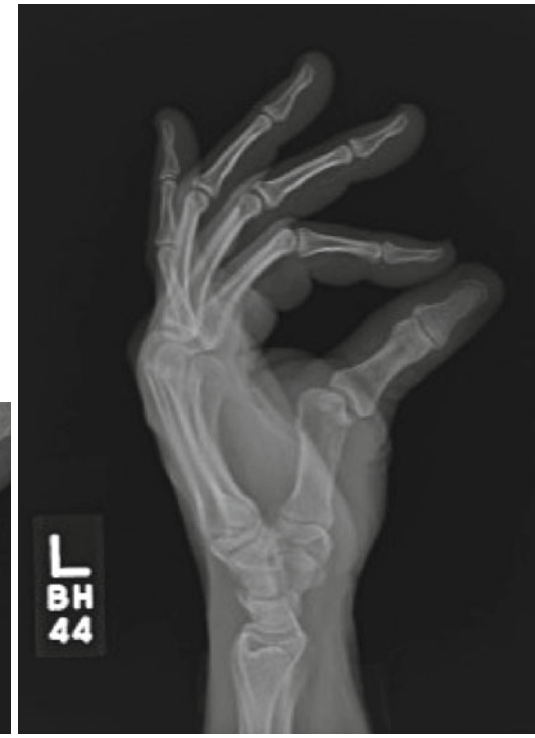
History Occurs with forced adduction on the thumb when the MP joint is flexed. The RCL can be torn at its proximal or distal end, but no aponeurosis displaces the lesion and prevents interposition.

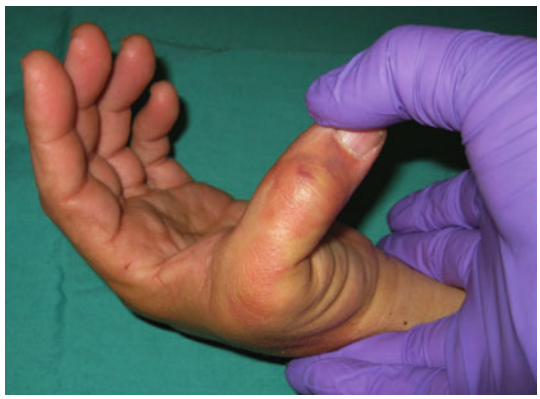
Exam Partial tears are characterized by swelling, tenderness, and possibly ecchymosis on the radial side of the metacarpal head and MP joint. When compared with the contralateral side, a laxity of 30° in flexion or extension or 15° of increased laxity is suggestive of a complete tear.

Treatment Acute RCL ruptures are managed conservatively if a partial tear is suspected. Surgical intervention is reserved for complete tears that are associated with volar subluxation of the proximal phalanx. If an RCL reconstruction is indicated, a free tendon graft is used. The thumb is immobilized in a thumb spica for 6 weeks postoperatively.

2.2.1.3 Ulnar Collateral Ligament (UCL) Injury







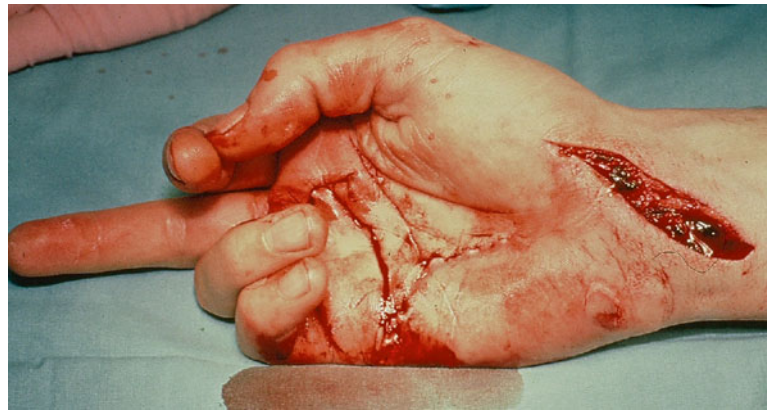
UCL injuries of the thumb MP joint occur ten times more frequently than RCL injuries.

History The injury is from a forced radial deviation of the joint. This occurs during a fall on an outstretched hand while the thumb is abducted. Patients complain of pain and swelling on the ulnar aspect of the MP joint.

Exam Tenderness, swelling, and ecchymosis over the ulnar aspect of the thumb at MP joint with unopposed deviation to the radial side with manipulation. The classic findings of a Stener's lesion, the UCL avulses and retracts proximally, and the interposed adductor aponeurosis interferes with primary healing. When avulsion occurs with a fracture, a mass can be palpated. The thumb demonstrates laxity of the ulnar capsule.

Treatment Incomplete ligament tears not associated with instability are treated conservatively. Partial tears are managed surgically if the UCL instability is greater than 30°. Stener's lesions are treated with surgical debridement of the fracture fragment, division of the adductor aponeurosis, and stabilization of the residual UCL to the proximal phalanx.

2.2.1.4 Flexor Tendon Laceration

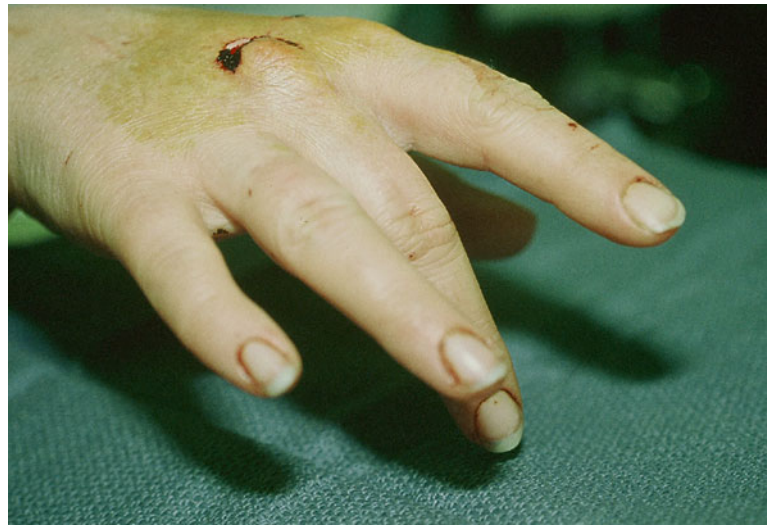


History Flexor tendon lacerations following sharp injuries require evaluation of the patient's age, occupation, mechanism of injury, and hand position during the time of injury. The five clinical tendon zones in the hand are based on anatomic landmarks and influence the prognosis of repair.

Exam The normal hand cascade is inspected. Active flexion of the DIP joint while stabilizing the PIP joint tests the function of the profundus tendon. Blocking the movement of the other fingers and instructing the patient to flex at the PIP joint will test the function of the sublimis tendon.

Treatment Different tendon retrieval techniques exist for each injury zone. Delaying the definitive repair up to 14 days has not been shown to have an adverse outcome. Tendon lacerations of 50 % or less of the cross-sectional area need not be repaired unless they cause triggering in a pulley. Early mobilization is encouraged.

2.2.1.5 Extensor Tendon Injury

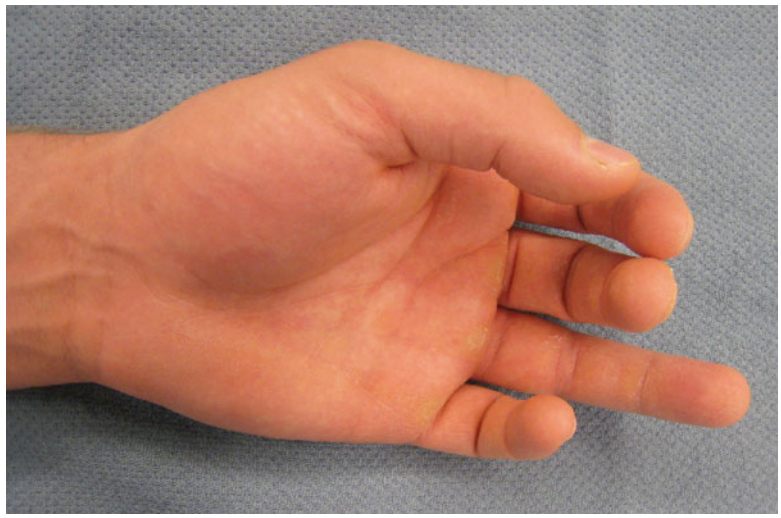


History A patient presents with deformity of the finger after laceration/trauma.

Exam Patient is unable to extend the affected finger.

Treatment Nonoperative treatment consists of immobilization of the affected joint with extension splinting. Operative treatment includes tendon repair, fixation of bony avulsion, tendon reconstruction, central slip reconstruction, or tendon transfer.

2.2.1.6 Jersey Finger

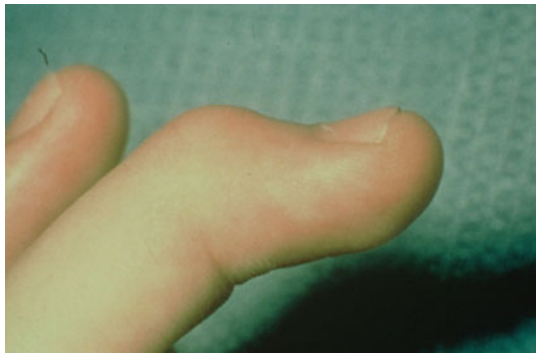


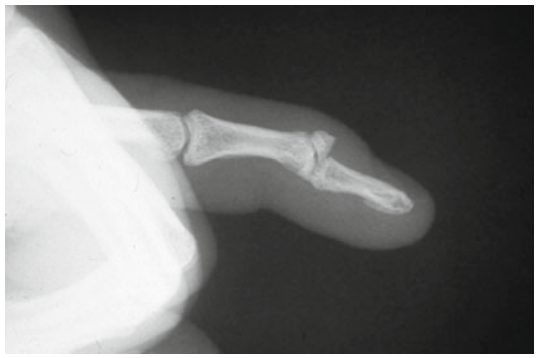
History The injury pattern of a sudden failure of the fingertip to grasp. Common mechanism of injury is of a football player attempting to tackle an opponent by the jersey. Subsequently, the patient is unable to flex the tip of his finger. Injuries are classified based on whether this is a pure soft tissue allusion of the FDP tendon or a certain portion of the volar distal phalanx bone is attached to the ruptured tendon.

Exam No active flexing of the DIP joint, however, the DIP joint can be passively flexed from zero degrees to full flexion position.

Treatment For soft tissue injuries alone, immediate retrieval and repair is recommended. When the bony fragment is held at the A3 pulley, it is possible to delay treatment for up to 3 weeks and achieve osteosynthesis with the use of stable bony fixation. Bony fragments held at the A4 pulley prevent tendon retraction and maintain the fragment close the fracture site. Hence, repair can be delayed almost indefinitely. Repair is performed with a Bunnell suture and tied dorsally over a button.

2.2.1.7 Mallet Finger





Definition Disruption of the terminal extensor tendon.

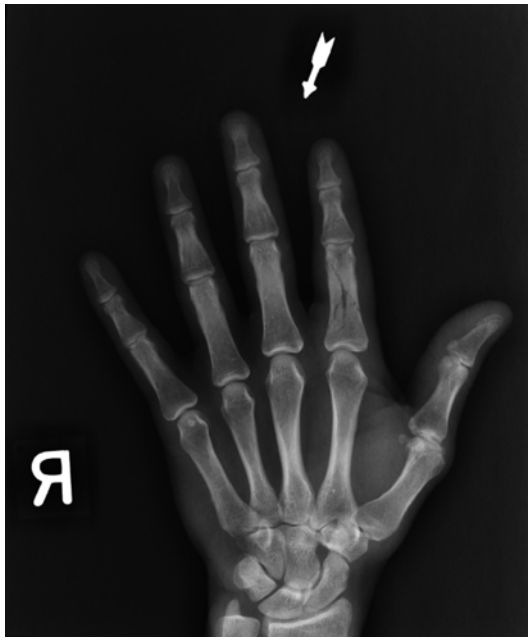
History There are a variety of mechanisms of injury that can lead to a mallet finger. Typically a mallet finger is the result of a closed injury that occurs when sudden flexion is applied to an extending DIP joint. Forceful hyperextension of the DIP joint can lead to a mallet finger and typically a fracture associated with this type of injury. Open injuries to the distal phalanx can also lead to a mallet deformity. Mallet fingers most commonly involve the ulnar digits. If untreated the patient will go on to develop an extensor lag and potentially compensatory hyperextension of the PIP resulting in a swan neck deformity. Injuries presenting within 4 weeks are considered acute, while a chronic mallet finger is greater than 4 weeks out from initial injury.

Exam Extensor lag of the involved DIP joint. There may be associated tenderness over the distal phalanx depending on timing of presentation. Plain films may demonstrate associated fracture.

Treatment When a mallet finger is diagnosed acutely, then the DIP joint of the affected digit is splinted in extension. The patient is to wear the splint continuously for 8 weeks. They are then transitioned to night splinting for an additional 4 weeks. Surgery is indicated in the acute setting if there is associated fracture with a fragment greater than 1/3 of the articular surface. Open injuries usually require operative intervention. Suture repair that incorporates both the skin and tendon eliminates need for extensive dissection that could compromise blood supply and healing. Chronic mallet injuries may require a Fowler tenotomy or spiral oblique retinacular ligament reconstruction depending on the amount of extensor lag that is present.

2.2.2 Skeletal

2.2.2.1 Phalanx Fractures

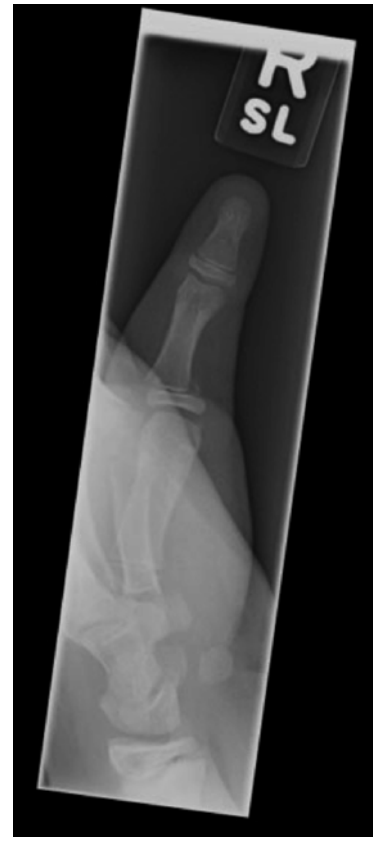


History Trauma to the hand; fracture of the phalanx is the most common skeletal injuries, accounting for 10 % of all fractures. Distal phalanx is the most common fractured bone in the hand.

Exam Physical findings include local tenderness and deformity, with or without an open wound.

Treatment Proximal and middle phalanx fracture can be treated with buddy taping, or close reduction with percutaneous pinning, or open reduction internal fixation. Distal phalanx fracture can be treated with close reduction and splinting as well. However, if there is concurrent nail bed injury, nail bed repair is warranted as well.

2.2.2.2 Pediatric Hand Fracture

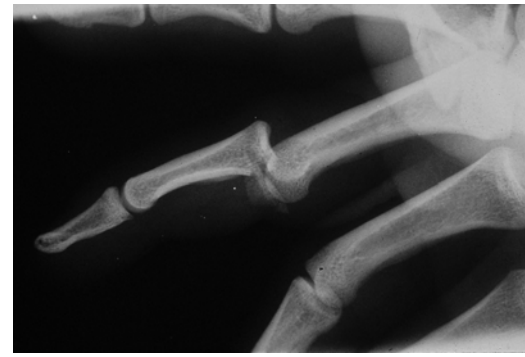


History The mechanism by which pediatric fractures are sustained is not different from adult fractures. These include a fall, crush injuries, or other accidental means. The difference is in the prognosis after the injury, given the susceptibility of the injured epiphyseal plate to poor healing. The Salter-Harris (SH) classification system describes fractures involving the epiphyseal plate in children. The degree of growth disturbance correlates directly with the level of fracture classification.

Exam A pediatric hand fracture can present as an obvious deformity with irregularity in the bony surface, tenderness, swelling, ecchymosis, and reduced range of motion secondary to pain. According to the SH classification, the epiphysis is separated from the metaphysis in type I fractures. Type II fractures involve the metaphysis and growth plate as opposed to the type III fractures which involve the epiphysis and growth plate. In SH IV, the fracture is through the epiphysis, growth plate, and metaphysis. In type V fractures, the epiphyseal plate is crushed with no involvement of the metaphysis.

Treatment In children with types I and II fractures, conservative treatment with closed reduction, casting, or pin fixation is recommended. Types III, IV, and V fractures are managed with similarly but also include reconstruction of the articular surface.

2.2.2.3 PIP Dislocation

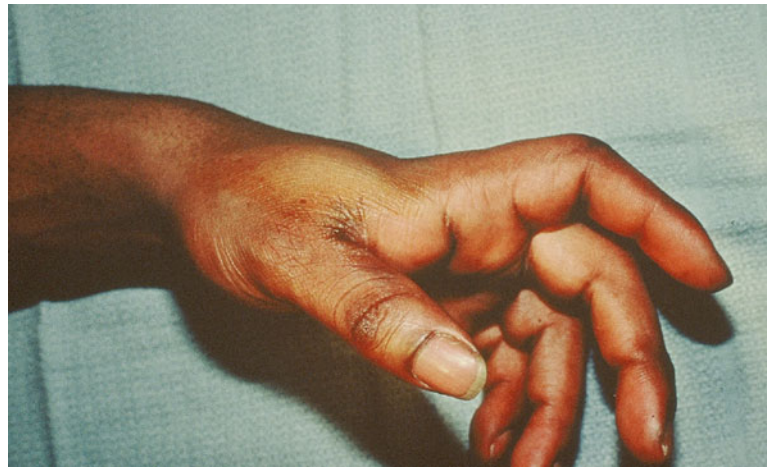


History A patient presents with deformity at the PIP joint after trauma.

Exam PIP joint injuries can be categorized as dorsal dislocations, dorsal fracture dislocations, volar dislocation, volar fracture dislocation, and rotatory dislocations. Finger and hand X-rays must be obtained to evaluate the extent of injury.

Treatment Nonoperative treatments include close reduction with buddy taping and dorsal extension block splinting. Surgical options include open reduction internal fixation, dynamic distraction external fixation, and volar plate arthroplasty or arthrodesis.

2.2.2.4 MCP Dislocation

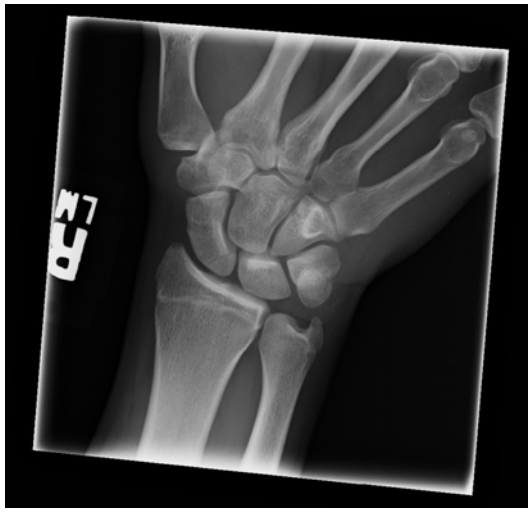


History A patient presents with deformity at the MCP joint after trauma.

Exam Skin dimpling may be seen in complex dislocations. X-rays show that joint space widening may indicate interposition of volar plate.

Treatment Simple dislocations can be treated with close reduction. Complex dislocations often require open reduction internal fixation via dorsal or volar approach.

2.2.2.5 Scaphoid Fracture

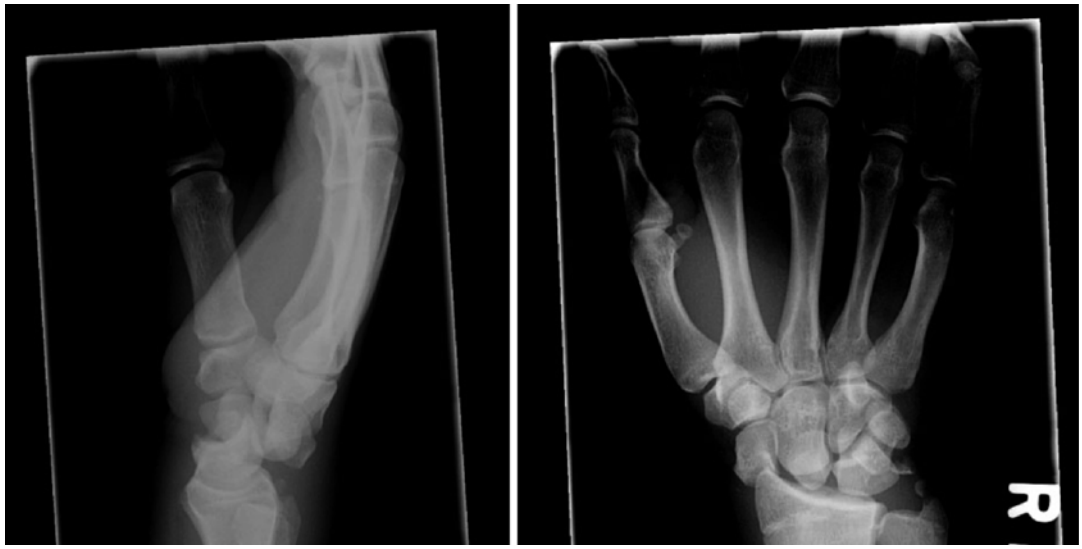


History Scaphoid injuries occur mostly from a hyperextension stress such as in the case of a *fall* on an outstretched hand or from an axial load as in the case of a *punch*. Hyperextension injuries produce a bending force at the distal pole of the scaphoid. Shear stress at the waist causes external axial forces with flexion moment on the distal pole of the scaphoid. Patients will complain of pain at the base of the thumb.

Exam Signs of a fracture can be subtle. The surgeon is required to have a high index of suspicion. Ecchymosis, edema, and radial-sided wrist pain. The tenderness is localized to the anatomic snuffbox and worse with axial compression or over the scaphoid tubercle. Radiographic imaging may require a bone scan, CT scan, or MRI if suspicion is high and the plain films are negative.

Treatment Non-displaced fractures are treated mostly with immobilization, 6 weeks with a long-arm thumb spica cast, and 6 weeks with a short-arm thumb spica cast. Displaced fractures are managed with percutaneous screw fixation or open reduction screw fixation.

2.2.2.6 Perilunate Dislocation



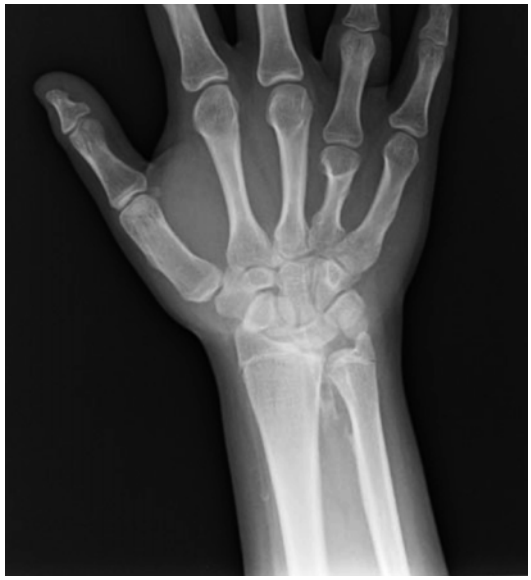
A pattern of injury traversing both the greater and lesser carpal arcs of the wrist. It is characterized by the disruption of multiple lunar attachments, such as the scapholunate and lunotriquetral ligaments.

History An uncommon wrist injury following a high-energy impact of the wrist such as a fall from a great height or a vehicle collision. Patients complain of pain at the wrist with numbness in the thumb, index, and long finger.

Exam Physical findings include wrist tenderness and acute carpal tunnel signs of numbness in digits along the median nerve distribution. Plain films demonstrate disruption of the Gilula's arcs in multiple levels. A lateral view of the wrist shows a spilled teapot sign of a lunate bone that is tipped volarly.

Treatment Immediate intervention of a closed perilunate dislocation is closed reduction followed by splinting, if an operating room is not available. Open reduction and internal fixation is indicated for evacuation of the hematoma and correct alignment of lunate dislocation.

2.2.2.7 SLAC Wrist



Definition Scapholunate advanced collapse of the wrist.

History Most patients present with a missed diagnosis of a scapholunate dissociation after a fall on an outstretched hand. Patients complain of progressive wrist pain and stiffness. Untreated dissociation progresses into degenerative arthritis of the wrist. The most common cause of SLAC wrist is the rotatory subluxation of the scaphoid followed by scaphoid nonunion, but other causes include intra-articular carpal fractures, midcarpal instability, and Kienbock's disease.

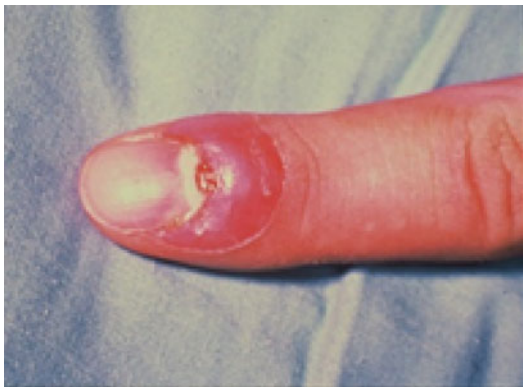
Exam Wrist tenderness and decreased grip strength. Plain films of an advanced SLAC wrist may show widening of the scapholunate joint, narrowing of the radioscaphoid joint, sclerosis of the proximal scaphoid, and ulnar translocation of the lunate with proximal migration of the capitate.

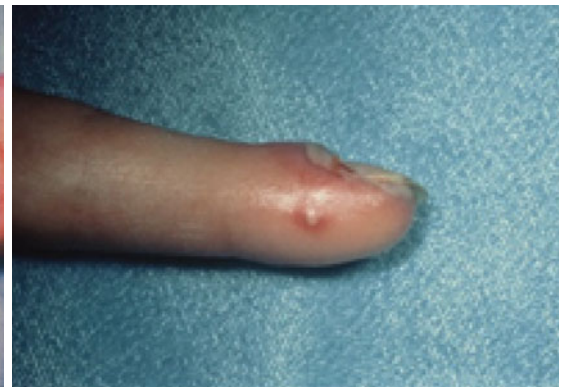
Treatment SLAC wrist reconstruction involves excision of the scaphoid and a four-corner fusion with arthrodesis of the capitate, lunate, triquetrum, and hamate. The key component to the success of the procedure is adequate reduction of the dorsal intercalated segmental instability (DISI) deformity of the lunate. Proximal row carpectomy is appropriate when the lunate fossa of the radius or capitate head is not involved with arthritis.

2.3 Hand, Acquired

2.3.1 Infection

2.3.1.1 Paronychia





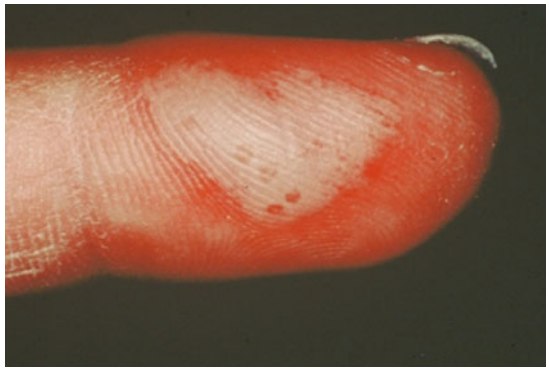
Paronychia is an infection of the tissue of the nail fold. It is the most common hand infection.

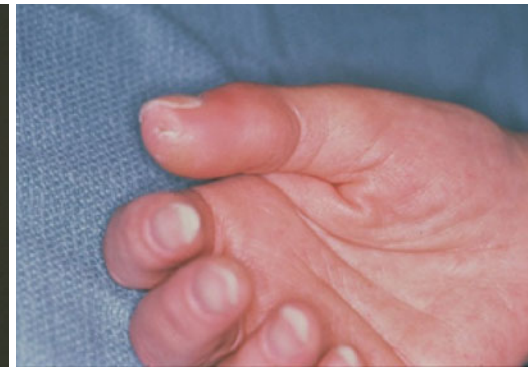
History Trauma to the paronychia occurs through such instances as nail biting, a hangnail, or a foreign body. Bacteria are introduced into the paronychial space. *S. aureus* is the most common causative bacteria, although mixed bacteria can be responsible.

Exam Swelling, tenderness, and erythema of the nail fold are hallmarks of paronychia. Infection can involve the entire eponychium, below the nail plate, and may even extend into the pulp. In an uncomplicated paronychia, X-rays are unnecessary. For complicated paronychia, where there is suspicion for foreign body or osteomyelitis, X-ray of the digit should be obtained. Cultures should be sent if atypical bacteria are suspected.

Treatment Treatment depends on the extent of infection. For early paronychia, soaking the digit and oral antibiotics are sufficient. Incision and drainage is required for abscess. The incision should be made in such a way as to direct the scalpel away from the nail bed and germinal matrix. The affected perionychial fold and eponychium must be released based on involvement. If the infection extends below the nail plate, the affected part of the nail plate must be excised. Patients should be treated with Dakin's soaks and oral antibiotics covering *S. aureus* for 7–10 days. Early range of motion should be initiated. The patient should be warned about causative factors such as biting hangnails or receiving manicures. Most cases should resolve in 3–4 days. If the infection fails to resolve, X-ray of the digit should be performed looking for osteomyelitis, cultures should be sent, and underlying systemic disease should be considered.

2.3.1.2 Felon



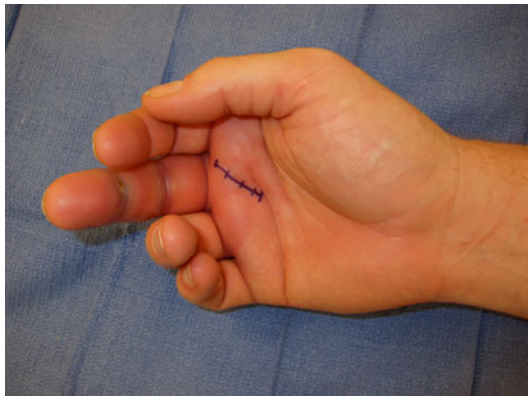


History The patient presents with pain and swelling at the affect fingertip.

Exam Felons are infections of the pulp space (which is compartmentalized by septa) typically caused by *Staphylococcus aureus*. Tenderness at the pulp is present, especially when the finger is placed in a dependent position.

Treatment Treatment of felon requires incision and drainage followed by antibiotic regimen.

2.3.1.3 Flexor Tenosynovitis



History The patient presents with pain and swelling, usually localized to the palmar aspect of one digit.

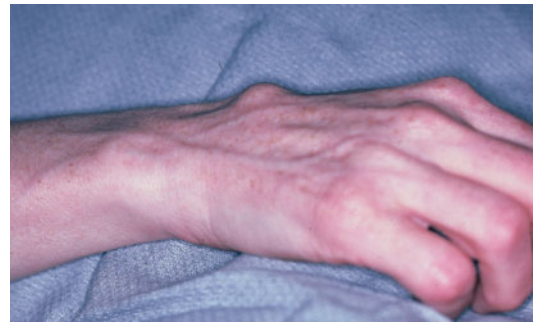
Exam An infection of the synovial sheath of the flexor tendon, there is increased warmth and erythema of the affected digit. In addition, Kanaval signs are present:

1. Flexed posturing of the involved digit
2. Tenderness to palpation over the tendon sheath
3. Marked pain with passive extension of the digit
4. Fusiform swelling of the digit

Treatment The most appropriate management is surgical irrigation and/or drainage of the tendon sheath via a proximal incision at the level of the A1 pulley and a distal incision at the distal flexor crease. The fibro-osseous canal is then irrigated copiously. More extensive infection requires open drainage and debridement.

2.3.2 Tumor

2.3.2.1 Ganglion Cyst

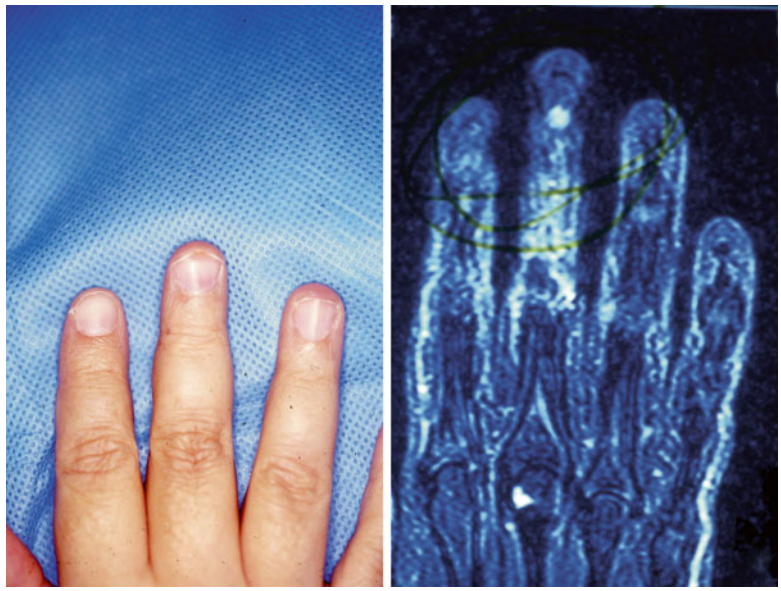


History The most frequent benign hand lesion present mostly in women in their second to fourth decade of life. They represent mucoid degeneration of fibrous connective tissue in joint capsules or tendon sheaths.

Exam Ganglion cysts are mobile masses that transluminate and may present with pain. In adults they are found mostly in the dorsal wrist (70 %) but among children are more common in the volar wrist. Also common are flexor tendon sheath ganglions, which attach to the tendon at the A1 pulley, or mucous cysts which are associated with the dorsal aspect of the DIP joint.

Treatment Medical management consists of aspiration or injection of steroids and sclerosing agents. In children, most ganglia resolve with conservative management (rosson walker). In adults regression is approximately in 50 % of the times.

2.3.2.2 Glomus Tumor

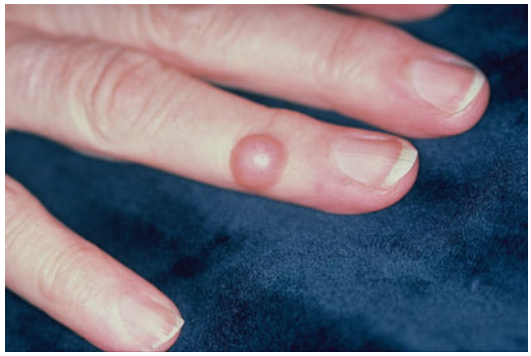


History Glomus tumors are benign hamartomas of the neuromyoarterial receptor, a regulator of skin temperature and blood flow. These rare vascular tumors represent 1 % of all hand tumors.

Exam Glomus tumors are marked by the exquisite pain, pinpoint tenderness, and cold intolerance. The tumors are commonly subungual and present as a nail deformity with a reddish-blue lesion underneath the nail plate. Hildreth's test and Love's pin test are reliable methods of diagnosing glomus hand tumors with sensitivity and specificity exceeding 90 %.

Treatment Simple excision is the treatment of choice. Meticulous nail bed repair is essential for preventing postoperative nail deformities. Pulp lesions can be approached through a lateral incision.

2.3.2.3 Mucous Cyst

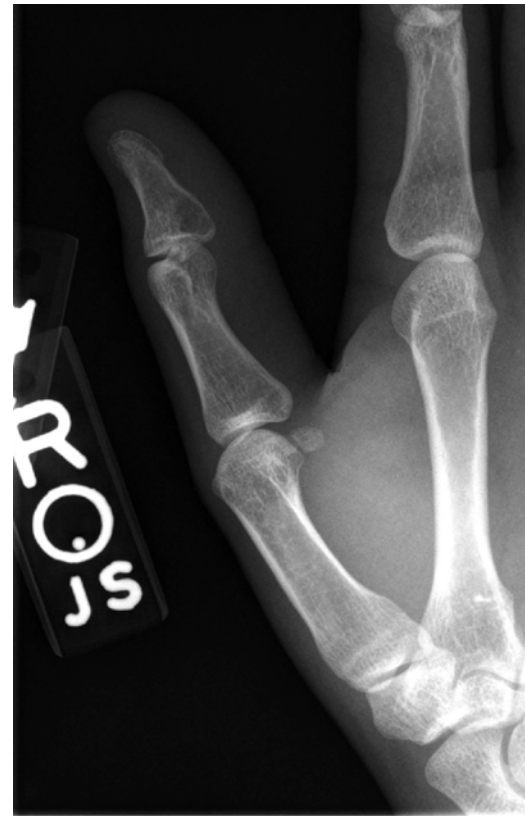


History The patient presents with a lesion in the DIP joint or proximal nail fold.

Exam It is a benign ganglion cyst typically at the dorsolateral aspect of the DIP joint or in the proximal nail fold. If located under the nail fold, the lunula is discolored with depression or grooving of the nail.

Treatment Excision of the lesion together with debridement of the underlying osteophyte is the treatment of choice to reduce the risk of recurrence.

2.3.2.4 Enchondroma



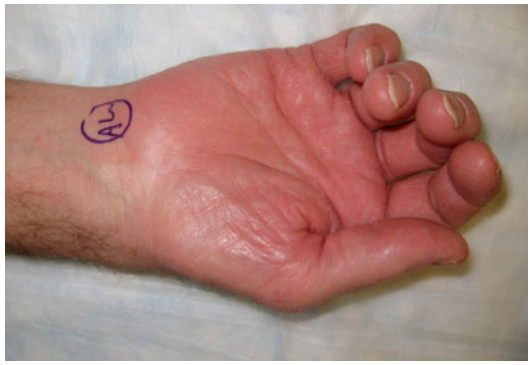
History An enchondroma is a benign cartilaginous tumor that usually develops during the second or third decade of life. It is the most common primary tumor of bones in the hand. Conditions associated with multiple enchondromas are Ollier disease or Maffucci syndrome in which subcutaneous hemangiomas are associated with the enchondromas.

Exam Enchondromas are often asymptomatic and discovered as an incidental finding on radiographic studies. Commonly, a pathological fracture occurs in the location of the tumor and leads to the diagnosis of the tumor.

Treatment Curettage of the bony lesion is recommended followed by bone grafting or other bony substitutes.

2.3.3 Systemic

2.3.3.1 Carpal Tunnel Syndrome (CTS)

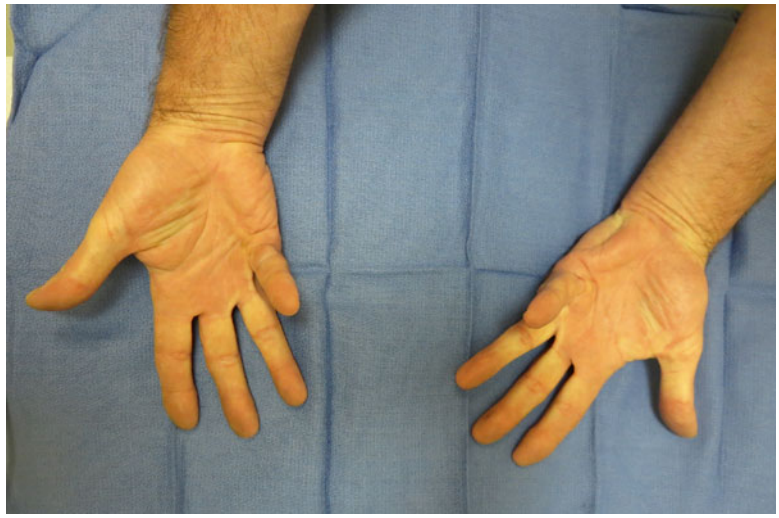


History The most common nerve entrapment in the upper extremity. Carpal tunnel syndrome is a compression neuropathy of the median nerve with nocturnal symptoms of pain and numbness. The location of symptoms corresponds to the anatomic distribution of the median nerve—the radial digits.

Exam Provocative testing includes a positive Phalen's sign and a Tinel's sign. Thenar atrophy (figure) is indicative of late phase of the syndrome, and electrodiagnostic testing demonstrates objective findings of decreased nerve conduction test and altered latency time.

Treatment Nonsurgical management with steroid injections is effective in patients with early symptoms. Early surgery is an option with clinical evidence of median nerve denervation or when the patient so elects. Surgical decompression with an open technique facilitates a direct visualization of the critical structures, complete transverse carpal ligament release, and concomitant Guyon's canal decompression. Endoscopic release can offer patients a smaller scar with earlier time to return to work but with higher rates of recurrent CTS.

2.3.3.2 Dupuytren's Contracture

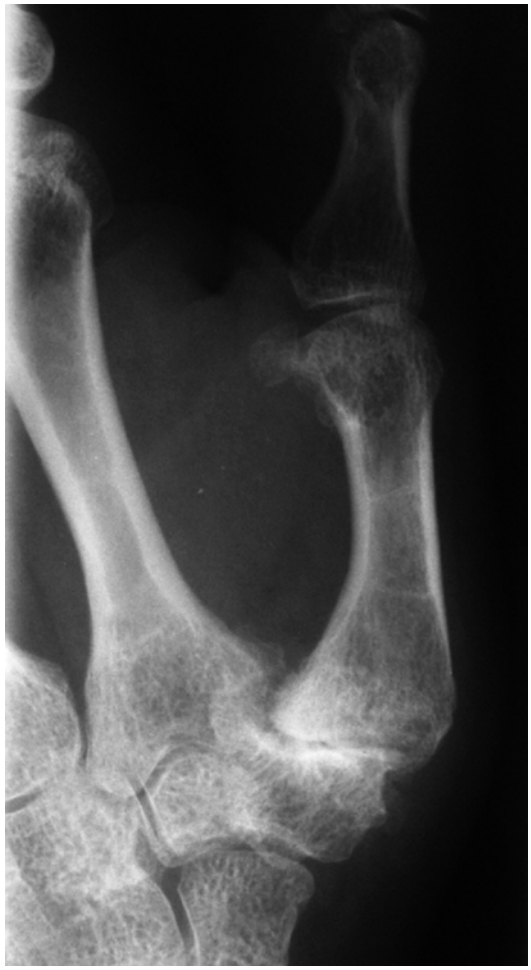


History Dupuytren's disease is a benign fibromatosis of the palmar and digital fascia with unclear etiology. *Dupuytren's* disease is especially common in people of northern Europe. Diathesis is an aggressive form of Dupuytren's contracture associated with knuckle pads, Peyronie's disease, and involvement of the plantar fascia.

Exam The palpable palmar nodules and cords are associated with debilitating flexion finger contracture. The fourth and fifth digits are the most commonly involved. A tabletop test is positive when the palm and fingers cannot be simultaneously placed on the flat surface of the tabletop.

Treatment The indications for surgical treatment are MCP joint contracture of 30° or greater or PIP joint contracture of 20° or greater with documented progression. Operative choices include open fasciectomies, closed fasciotomy, or needle fasciotomy. Collagenase injection has been shown to be effective in correcting flexion deformities of the MCP joint but not the PIP joint. Administration of steroid injections has been shown to improve palmar nodules.

2.3.3.3 Basal Joint Arthritis

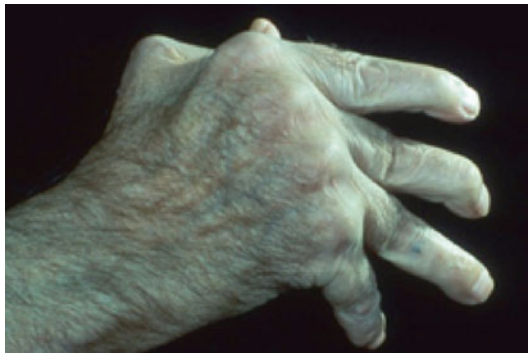


History Osteoarthritis of the basal joint most frequently affects middle-aged women. It presents as pain in the radial wrist. Although it is not the most commonly affected joint in the hand, symptomatic basal joint arthritis is the most debilitating.

Exam Poor abduction of the thumb, weakened grip, tenderness at the anatomic snuffbox, and a positive grind sign. X-ray findings demonstrate subluxation of the first metacarpal on the trapezium, osteophytic spurring, sclerosis, and articular irregularities of the joint surfaces. Radiographic staging may not relate to symptoms.

Treatment Initial conservative management includes nonsteroidal anti-inflammatory drugs, splinting, and steroid injections. Surgical treatment is recommended for pain relief with trapezius excision alone or with ligament reconstruction and tendon interposition.

2.3.3.4 Rheumatoid Arthritis (RA)



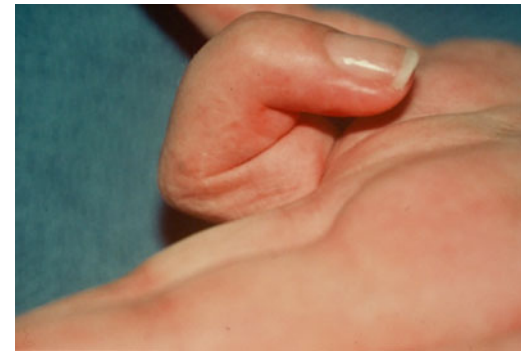
Definition A chronically progressive disease that affects the synovial tissues and associated with autoimmune inflammatory findings.

History Patients complain of progressively worsening pain in the joints of the hand and wrist. The pain is often worse in the morning. The disease predominantly affects women at least three times more than men.

Exam The disease affects the skin with rheumatoid nodules as subcutaneous inflammatory masses. The joint findings are of "z deformities" with ulnar deviation at MP joints. Other clinical findings include tenosynovitis and rupture of the extensor tendons, swan neck and Boutonniere deformities, muscle tightness of the intrinsic muscles from disuse, vasculitis, nerve compression, and extra-articular involvements including the lungs, heart, orbits, polyneuropathy, and splenomegaly. Labs are positive for the rheumatoid factors, and imaging confirms osteoporosis, joint narrowing, and ulnar deviation of the carpus and MP joints.

Treatment Nonsurgical therapy is the mainstay treatment of RA with NSAIDs, steroids, and immunomodulators. Recently antimonoclonal antibody compounds have significantly improved the management of RA patients while avoiding the negative sequela of steroid treatment. Surgical management is aimed at elevating joint pain, functional restoration of the joints, and cosmesis. These include MP arthroplasties, tendon rupture repair, and correction of swan neck and Boutonniere deformities to name a few.

2.3.3.5 Trigger Finger

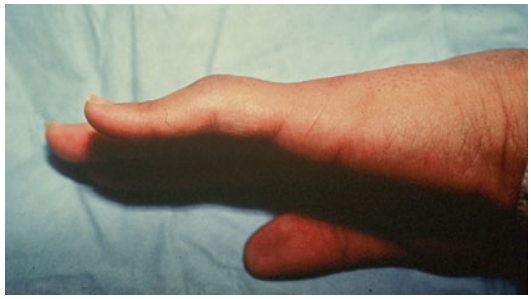


History The patient presents with "clicking" of the affected digit locked in a flexed position.

Exam Trigger finger is caused by entrapment of the flexor tendons at the level of the A1 pulley. It is a stenosing tenosynovitis caused by inflammation of the flexor tendon sheath. The ring finger is commonly involved. On exam, there is tenderness at the A1 pulley, as well as a palpable nodule.

Treatment Nonoperative treatment includes activity modification and NSAIDS. Surgery entails release of the A1 pulley.

2.3.3.6 Diagnosis: Boutonniere Deformity

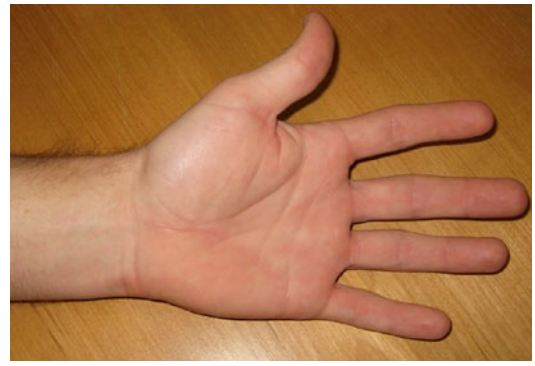


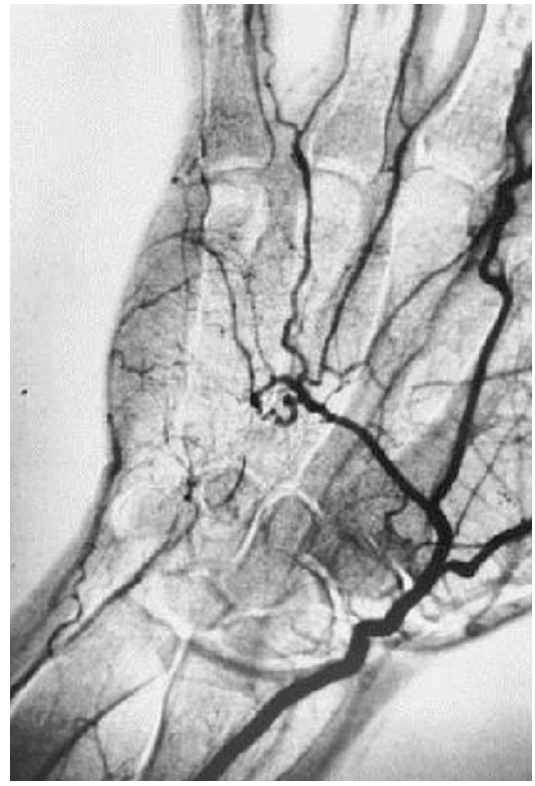
History Boutonniere deformity can occur in patients with rheumatoid arthritis or after a traumatic central slip disruption and PIP joint volar dislocation. In rheumatoid patients, the PIP joint synovitis causes extensor stretch and central slip attenuation. Weakening of the central slip can lead to the volar movement of the lateral bands and tightness of the ligament of Landsmeer.

Exam PIP joint flexion contracture with hyperextension of the DIP joint. Patients are unable to actively extend the PIP joint but full passive range of motion of the finger.

Treatment Early synovectomy and dynamic splinting. In mild cases of PIP flexion, the central slip can be shortened and the lateral bands repositioned. In severe cases of fixed PIP joint flexion, arthrodesis or arthroplasty is an option.

2.3.3.7 Hypothenar Hammer Syndrome



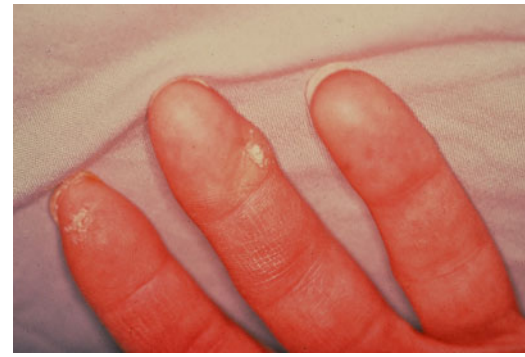


History The patient (usually a construction worker) presents with pain in the ring and small fingertips. Ulcerations may be present. There is no history of trauma, systemic disease, or hand claudication.

Exam There are tenderness and swelling over the hypothenar eminence, with small ulceration on the ring and small fingertips. Sensation is intact. MRA shows an aneurysm or thrombus in the ulnar artery.

Treatment Optimal treatment involves resection of the diseased segment and reconstruction.

2.3.3.8 Scleroderma



History The patient presents with cold intolerance and painless ulcer at the affected fingertips.

Exam Scleroderma is an autoimmune disease that affects small blood vessels and connective tissue. Affected organs include the skin, hands, heart, lungs, gastrointestinal tract, and kidneys. Shiny edema of the skin and stiffness of the joints may be present. Ulceration at the fingertips is due to poor circulation. Skin breakdown can also occur at the proximal interphalangeal (PIP) and metacarpophalangeal (MP) joints.

Treatment Tip ulcerations often heal over time without surgery. Conservative treatment includes conservative debridement, topical antibiotics, and limited resection of exposed bone. More aggressive treatment includes amputation and digital sympathectomy.

References

Transverse Deficiency

1. Oda T, Pushman AG, Chung KC. Treatment of common congenital hand conditions. PRS. 2010;126(3):121e–33e.
2. Ogino T, Saitou Y. Congenital constriction band syndrome and transverse deficiency. J Hand Surg (Br). 1987;12:343–8.

Radial Clubhand

3. Choi M, Sharma S, Louie O. Congenital hand abnormalities. In: Thorne CH, editor. Grabb & Smith's plastic surgery. 6th ed. Philadelphia: Lippincott-Raven; 2007. p. 856–7.

Ulnar Deficiency

4. Johnson J, Omer Jr GE. Congenital ulnar deficiency. Natural history and therapeutic implications. Hand Clin. 1985;1(3):499–510.
5. Netscher DT, Baumholtz MA. Treatment of congenital upper extremity problems. Plast Reconstr Surg. 2007;119(5):101e–29e.

Cleft Hand

6. Kay SP, McCombe D, Kozin SH. Deformities of the hand and fingers. In: Hotchkiss RN, Green DP, editors. Green's operative hand surgery. Philadelphia: Elsevier Churchill Livingstone; 2011. p. 1303–69.
7. Netscher DT. Congenital hand problems. Terminology, etiology, and management. Clin Plast Surg. 1998;25(4):537–52.

Diagnosis: Syndactyly

8. Dao KD, Shin AY, Billings A, Oberg KC, Wood VE. Surgical treatment of congenital syndactyly of the hand. J Am Acad Orthop Surg. 2004;12(1):39–48.
9. Mishra A, McArthur P. Different approaches to syndactyly repair without skin graft. Plast Reconstr Surg. 2010;126(4):1411.
10. Netscher DT, Baumholtz MA. Treatment of congenital upper extremity problems. Plast Reconstr Surg. 2007;119(5):101e–29e.

Duplicated Thumb

11. Ganley TJ, Lubahn JD. Radial polydactyly: an outcome study. Ann Plast Surg. 1995;35(1):86–9.
12. Netscher DT, Baumholtz MA. Treatment of congenital upper extremity problems. Plast Reconstr Surg. 2007;119(5):101e–29e.
13. Zuidam JM, Selles RW, Ananta M, Runia J, Hovius SE. A classification system of radial polydactyly: inclusion of triphalangeal thumb and triplication. J Hand Surg Am. 2008;33(3):373–7.

Macrodactyly

14. Akinci M, Ay S, Ercetin O. Surgical treatment of macrodactyly in older children and adults. J Hand Surg Am. 2004;29(6):1010–9.
15. Ishida O, Ikuta Y. Long-term results of surgical treatment for macrodactyly of the hand. Plast Reconstr Surg. 1998;102(5):1586–90.

Hypoplastic Thumb

16. Bates SJ, Hansen SL, Jones NF. Reconstruction of congenital differences of the hand. Plast Reconstr Surg. 2009;124(1 Suppl):128e–43e.
17. Manske PR, McCaroll Jr HR. Index finger pollicization for a congenitally absent or nonfunctioning thumb. J Hand Surg Am. 1985;10(5):606–13.

Brachydactyly

18. Garcia Julve G, Martinez Villen G. The multiple monoblock toe-to-hand transfer in digital reconstruction. A report of ten cases. J Hand Surg Br. 2004;29(3):222–9.
19. Nguyen ML, Jones NF. Undergrowth: brachydactyly. Hand Clin. 2009;25(2):247–55.

Clinodactyly

20. Aucourt J, Budzik JF, Manouvrier-Hanu S, Mezel A, Cotten A, Boutry N. Congenital malformations of the hand and forearm in children: what radiologists should know. Semin Musculoskelet Radiol. 2012;16(2):146–58.
21. Yesilada AK, Sevim KZ, Sucu DO, Kilinc L. Congenital hand deformities – a clinical report of 191 patients. Acta Chir Plast. 2013;55(1):10–5.

Diagnosis: Camptodactyly

22. Choi M, Sharma S, Louie O. Congenital hand abnormalities. In: Thorne CH, editor. Grabb & Smith's plastic surgery. 6th ed. Philadelphia: Lippincott-Raven; 2007. p. 860.
23. McFarlane RM, Classen DA, Porte AM, Botz JS. The anatomy and treatment of camptodactyly of the small finger. J Hand Surg Am. 1992;17(1):35–44.

Constriction Band Syndrome (CBS)

24. Tiwari VK, Sarabahi S, Mulla Rani UK. Heterotopic implantation of amputated digits following constriction ring syndrome: a case series. J Plast Reconstr Aesthet Surg. 2013;66(12):1815–7.
25. Wiedrich TA. Congenital constriction band syndrome. Hand Clin. 1998;14(1):29–38.

Subungual Hematoma

26. Seaberg DC, Angelos WJ, Paris PM. Treatment of subungual hematomas with nail trephination: a prospective study. Am J Emerg Med. 1991;9(3):209–10.
27. Wilhelmi BJ, Zook EG. Fingernail and fingertip injuries. In: Guyuron B, Eriksson E, Persing JA, editors. Plastic surgery: indications and practice. Philadelphia: WB Saunders; 2009. p. 992–1000.

Radial Collateral Ligament (RCL) Injury

28. Iba K, Wada T, Hiraiwa T, Kanaya K, Oki G, Yamashita T. Reconstruction of chronic thumb metacarpophalangeal joint radial collateral ligament injuries with a half-slip of the abductor pollicis brevis tendon. J Hand Surg Am. 2013;38(10):1945–50.
29. Taylor KF, Lanzi JT, Cage JM, Drake ML. Radial collateral ligament injuries of the thumb metacarpophalangeal joint: epidemiology in a military population. J Hand Surg Am. 2013;38(3):532–6.

Ulnar Collateral Ligament (UCL) Injury

30. Stener B. Skeletal injuries associated with rupture of the ulnar collateral ligament of the metacarpophalangeal joint of the thumb. A clinical and anatomical study. Acta Chir Scand. 1963;125:583–6.

Flexor Tendon Laceration

31. Lalonde DH, Kozin S. Tendon disorders of the hand. Plast Reconstr Surg. 2011;128(1):1e–4.
32. Tang JB. Clinical outcomes associated with flexor tendon repair. Hand Clin. 2005;21(2):199–210.

Extensor Tendon Injury

33. Doyle JR. Extensor tendons – acute injuries. In: Green DP, Hotchkiss RN, Pederson WC, editors. Operative hand surgery. 4th ed. New York: Churchill Livingstone; 1999. p. 2. 1963.
34. Rockwell WB, Butler PN, Byrne BA. Extensor tendon: anatomy, injury, and reconstruction. Plast Reconstr Surg. 2000;106:1592.

Jersey Finger

35. Peterson JJ, Bancroft LW. Injuries of the fingers and thumb in the athlete. Clin Sports Med. 2006;25(3):527–42. 3.
36. Tuttle HG, Olvey SP, Stern PJ. Tendon avulsion injuries of the distal phalanx. Clin Orthop Relat Res. 2006;445:157–68.

Mallet Finger

37. Eichstadt S, Chang J. Extensor injuries-acute and chronic. In: The American Society for Surgery of the Hand, editor. Textbook of hand and upper extremity surgery. Chicago: ASSH; 2013. p. 406–18.
38. Strauch RJ. Extensor tendon injury. In: Hotchkiss RN, Green DP, editors. Green's operative hand surgery. Philadelphia: Elsevier Churchill Livingstone; 2011. p. 159–86.

Phalanx Fractures

39. Trumble TE. Hand fractures. In: Trumble TE, editor. Principles of hand surgery and therapy. Philadelphia: WB Saunders; 2000. p. 49–55.

Pediatric Hand Fracture

40. Campbell Jr RM. Operative treatment of fractures and dislocations of the hand and wrist region in children. Orthop Clin North Am. 1990;21(2):217–43.
41. Le TB, Hentz VR. Hand and wrist injuries in young athletes. Hand Clin. 2000;16(4):597–607.

PIP Dislocation

42. Merrell G, Slade JF. Dislocation and ligament injuries in the digits. In: Wolfe SW, Hotchkiss RN, Pederson WC, et al., editors. Green's operative hand surgery. 6th ed. Philadelphia: Churchill Livingstone; 2011. p. 291–332.

MCP Dislocation

43. Merrell G, Slade JF. Dislocation and ligament injuries in the digits. In: Wolfe SW, Hotchkiss RN, Pederson WC, et al., editors. Green's operative hand surgery. 6th ed. Philadelphia: Churchill Livingstone; 2011. p. 291–332.

Scaphoid Fracture

44. Gellman H, Caputo RJ, Carter V, Aboulafia A, McKay M. Comparison of short and long thumb-spica casts for non-displaced fractures of the carpal scaphoid. J Bone Joint Surg Am. 1989;71(3):354–7.
45. Weber ER, Chao EY. An experimental approach to the mechanism of scaphoid waist fractures. J Hand Surg Am. 1978;3(2):142–8.

Perilunate Dislocation

46. Mayfield JK, Johnson RP, Kilcoyne RK. Carpal dislocations: pathomechanics and progressive perilunar instability. J Hand Surg Am. 1980;5(3):226–41.
47. Yaghoubian R, Goebel F, Musgrave DS, Sotereanos DG. Diagnosis and management of acute fracture-dislocation of the carpus. Orthop Clin North Am. 2001;32(2):295–305. 2.

SLAC Wrist

48. Krakauer JD, Bishop AT, Cooney WP. Surgical treatment of scapholunate advanced collapse. J Hand Surg Am. 1994;19(5):751–9.
49. Watson HK, Ballet FL. The SLAC wrist: scapholunate advanced collapse pattern of degenerative arthritis. J Hand Surg Am. 1984;9(3):358–65.

Paronychia

50. Jebson PJ. Infections of the fingertip. Paronychias and felons. Hand Clin. 1998;14(4):547–55. 4.
51.Zook EG. Understanding the perionychium. J Hand Ther. 2000;13(4):269–75.

Felon

52. Jebson PJ. Infections of the fingertip. Paronychias and felons. Hand Clin. 1998;14(4):547–55. 4.
53. Neviaser RJ. Acute infections. In: Green DP, Hotchkiss RN, Pederson WC, editors. Operative hand surgery, vol. 2. 4th ed. New York: Churchill Livingstone, Inc; 1999. p. 1033–47.

Flexor Tenosynovitis

54. Juliano PT, Eglseder WA. Limited open sheath irrigation in the treatment of pyogenic flexor tenosynovitis. Orthop Res. 1991;20:1065–9.
55. Schall SB, Va-Rose T, Holtom P, et al. Tissue pressures in pyogenic flexor tenosynovitis of the finger. J Bone Joint Surg. 1996;78B:793–5.

Ganglion Cyst

56. Nahra ME, Bucchieri JS. Ganglion cysts and other tumor related conditions of the hand and wrist. Hand Clin. 2004;20:249–60.

Glomus Tumor

57. Hazani R, Houle JM, Kasdan ML, Wilhelmi BJ. Glomus tumors of the hand. Eplasty. 2008;8, e48.
58. Tomak Y, Akcay I, Dabak N, Eroglu L. Subungual glomus tumours of the hand: diagnosis and treatment of 14 cases. Scand J Plast Reconstr Surg Hand Surg. 2003;37(2):121–4.

Mucous Cyst

59. Rizzo M, Beckenbaugh RD. Treatment of mucous cysts of the fingers: review of 134 cases with minimum 2-year follow-up evaluation. J Hand Surg Am. 2003;28(3):519–24.

Enchondroma

60. Bauer RD, Lewis MM, Posner MA. Treatment of enchondromas of the hand with allograft bone. J Hand Surg Am. 1988;13(6):908–16.
61. Bickels J, Wittig JC, Kollender Y, et al. Enchondromas of the hand: treatment with curettage and cemented internal fixation. J Hand Surg Am. 2002;27(5): 870–5.

62. Floyd 3rd WE, Troum S. Benign cartilaginous lesions of the upper extremity. Hand Clin. 1995;11(2):119–32.

Carpal Tunnel Syndrome (CTS)

63. Keith MW, Masear V, Amadio PC, et al. Treatment of carpal tunnel syndrome. J Am Acad Orthop Surg. 2009;17(6):397–405.
64. Shores JT, Lee WP. An evidence-based approach to carpal tunnel syndrome. Plast Reconstr Surg. 2010;126(6):2196–204.

Dupuytren's Contracture

65. Hurst LC, Badalamente M. Associated diseases. In: McFarlane RM, McGrouther DA, Flint MH, editor. Dupuytren's disease: biology and treatment, vol. 5, The hand and upper limb series. Edinburgh: Churchill Livingstone; 1990. p. 253–60.
66. McFarlane RM. Various views and techniques: extensive fasciectomy. In: McFarlane RM, McGrouther DA, Flint MH, editor. Dupuytren's disease: biology and treatment, vol. 5, The hand and upper limb series. Edinburgh: Churchill Livingstone; 1990. p. 360–4.

Basal Joint Arthritis

67. Glickel SZ. Clinical assessment of the thumb trapeziometacarpal joint. Hand Clin. 2001;17(2):185–95.
68. Manske PR. Commentary: excision of the trapezium. J Hand Surg Am. 2004;29(6):1078–9.
69. Tomaino MM, Pellegrini Jr VD, Burton RI. Arthroplasty of the basal joint of the thumb. Long-term follow-up after ligament reconstruction with tendon interposition. J Bone Joint Surg Am. 1995;77(3):346–55.

Rheumatoid Arthritis (RA)

70. Arnett FC, Edworthy SM, Bloch DA, et al. The American Rheumatism Association 1987 revised criteria for the classification of rheumatoid arthritis. Arthritis Rheum. 1988;31(3):315–24.
71. O'Brien ET. Surgical principles and planning for the rheumatoid hand and wrist. Clin Plast Surg. 1996;23(3):407–20.

Trigger Finger

72. Trumble TE, editor. Hand surgery update 3: hand, elbow, and shoulder. Rosemont: American Society for Surgery of the Hand; 2003. p. 3–28.

Diagnosis: Boutonniere Deformity

73.Lalonde DH, Kozin S. Tendon disorders of the hand. Plast Reconstr Surg. 2011;128(1):1e–14e.
74. O'Brien ET. Surgical principles and planning for the rheumatoid hand and wrist. Clin Plast Surg. 1996;23(3):407–20.

Hypothenar Hammer Syndrome

75. Dethmers RSM, Houpt P. Surgical management of hypothenar and thenar hammer syndromes: a retrospective study of 31 instances in 28 patients. J Hand Surg (Br). 2005;30:419–23.

Scleroderma

76. Bogoch ER, Gross DK. Surgery of the hand in patients with systemic sclerosis: outcomes and considerations. J Rheumatol. 2005;32:642–8.
77. Gahhos F, Ariyan S, Frazier WH, et al. Management of sclerodermal finger ulcers. J Hand Surg (Am). 1984;9:320–7.

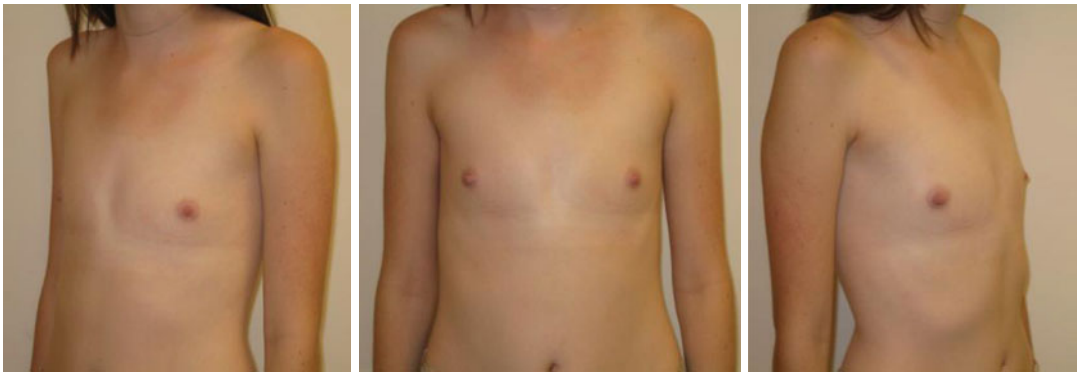
Breast

3

Jason Ulm, Mohamed Amir Mrad, Alan Yan, Michael J. Yaremchuk, Eric Liao, Fuad Hashem, and Brian Labow

3.1 Breast, Congenital

Amasia (see 3.1.1)
Nineteen-year-old female with amasia



J. Ulm, MD (✉) • A. Yan, MD
M.J. Yaremchuk, MD
Division of Plastic and Reconstructive Surgery, Craniofacial Plastic Surgery, Massachusetts General Hospital, Harvard Medical School, Boston, Massachusetts, USA
e-mail: jasonulm@gmail.com; alanyanmd@gmail.com; http://www.dryaremchuk.com

M.A. Mrad, MD
Division of Plastic Surgery, Department of Surgery, King Faisal Specialist Hospital and Research Center, Alfaisal University, Riyadh, Saudi Arabia
e-mail: mmrad@kfshrc.edu.sa
http://www.drmrad.com;

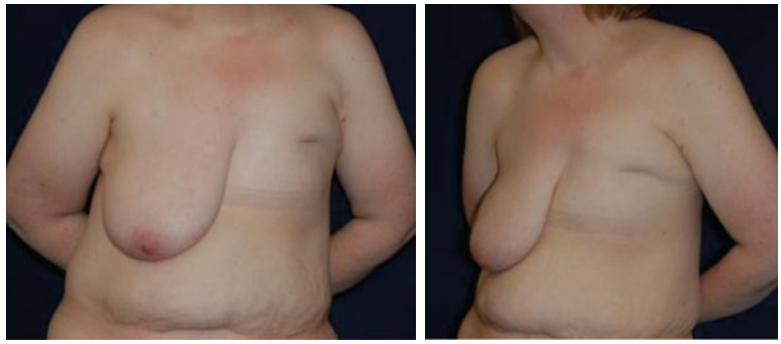
E. Liao, MD
Division of Plastic Surgery, Department of Surgery, Massachusetts General Hospital, Harvard Medical School, Boston, MA, USA
e-mail: eliao1@partners.org

F. Hashem, MD
Department of Plastic Surgery, King Faisal Specialist Hospital, Riyadh, Saudi Arabia
e-mail: fuadkhashem@gmail.com

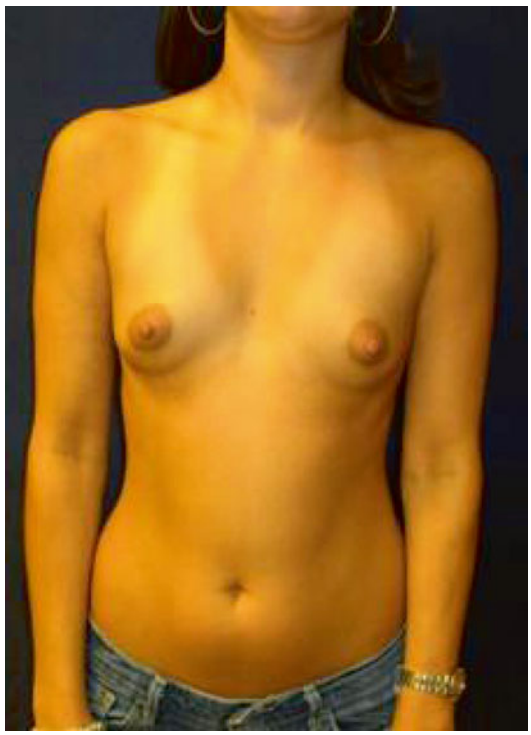
B. Labow, MD
Department of Plastic and Oral Surgery, Children's Hospital of Boston, Harvard Medical School, Boston, MA, USA
e-mail: Brian.labow@childrens.harvard.edu

© Springer International Publishing Switzerland 2016
R. Hazani et al. (eds.), *Clinical Diagnosis in Plastic Surgery*, DOI 10.1007/978-3-319-17094-7_3

Amastia (see 3.1.1)



Micromastia (see 3.1.1)



3.1.1 Hypoplastic Breast

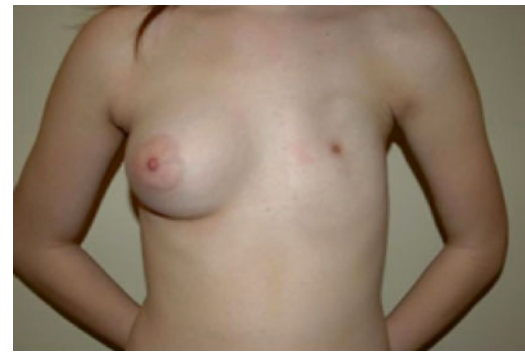
Athelia: isolated absence of the NAC
Amasia: absence of breast parenchyma
Amastia: absence of both the breast tissue and the NAC
Micromastia: postpubertal underdevelopment of breast tissue (hypoplasia)

History History of injury/surgery to breast and ovaries, delayed puberty, chronic disease/endocrinology. Ask about primary amenorrhea and secondary sexual characteristics. Family history of delayed development (constitutional) or family history of similar diagnosis. Psychological distress for school age, and the family should be addressed. These diagnoses are usually diagnosed by examination. Athelia is discovered during childhood. Amasia, amastia, and micromastia are usually discovered later during pubertal growth. If 6 months passes after the age of 17 with no changes to the size, the diagnosis is confirmed.

Examination Whole body exam for puberty characteristics, specifically tanner stage. Inspect and palpate, measure the breast size and width. Compare both sides (if less than 2/5 of the contralateral breast, then the breast is called hypoplastic). Body length and weight and growth percentile should be assessed. Photograph should be taken, and a bone age is ordered. Rule out Poland's syndrome and endocrine causes—e.g. adrenal hyperplasia, turner syndrome, and testicular feminization disorders (which are usually bilateral).

Treatment Breast augmentation after clinically confirming end of pubertal growth. NAC reconstruction is done to treat athelia.

3.1.2 Poland's Syndrome



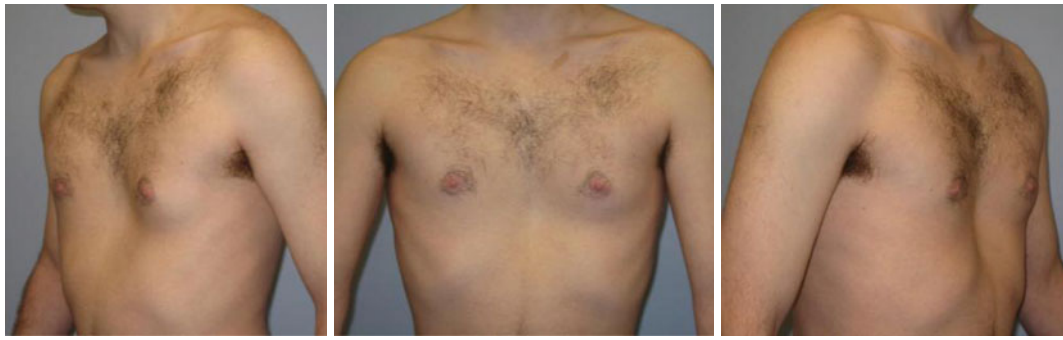
History A rare congenital disorder that is characterized by hypoplasia of the pectoralis muscles and ipsilateral webbing of the fingers. It can include other deformities such as scoliosis, hypoplasia of the rib cage and lungs, upper extremity hypoplasia, breast and nipple hypoplasia, and deficiency in the skin layers and content. Other muscles in the shoulder can also be affected.

Exam Upper limbs and chest should be examined thoroughly to identify the structures involved in the disorder. X-rays should be taken for the whole upper limb including both hands.

Treatment Is focused on replacing or reconstructing the deficiency involved in the disorder. In regard to the breast, autogenous or alloplastic material can be used to replace breast tissues. Nipple-areolar complex (NAC) reconstruction is usually needed in cases where the NAC is involved.

The hand treatment is tailored to the extent of the deformity. Syndactyly release is the commonest operation needed for these patients.

3.1.3 Pectus Excavatum



History Congenital chest wall deformity in which several ribs and the sternum grow abnormally, producing a concave, or caved in, appearance in the anterior chest wall.

Examination Examine the posture of the patient, any shoulder slope. Assess the severity of the defect and the asymmetry of the chest. You should also assess the degree of sternal involvement (related to severity), the diameter and movement of the chest wall, anterior flaring of the lower ribs, and the presence of scoliosis. Complete skeletal examination should be performed to rule out Marfan and Poland associations. Complete examination of the heart and lung should be performed.

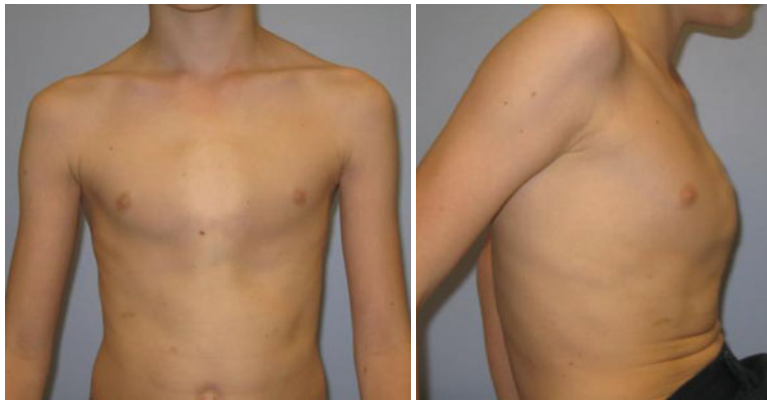
Treatment Nonsurgical treatment includes physiotherapy to correct the posture. Usually have minimal effect on the shape of the chest.

Surgical treatment includes:

1. Alloplastic implant: as a camouflaging technique only with silicone implant
2. Ravitch procedure: subperichondrial cartilage and rib resection, sternal osteotomy, and fixation of the sternum in the appropriate position
3. WADA technique: sternal molding and turnover

Minimally Invasive Repair of Pectus Excavatum (MIRPE): Nuss procedure

3.1.4 Pectus Carinatum

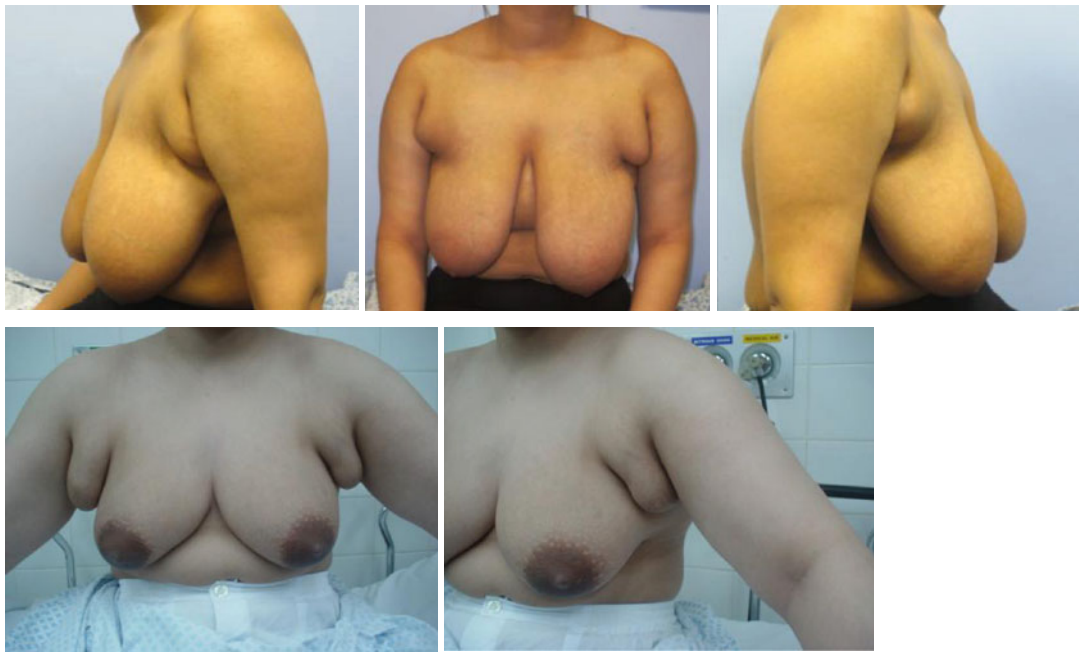


History Pectus carinatum is the second most common chest wall deformity observed in children (4:1 M:F). It is the protrusion of the sternum and ribs and is the opposite of pectus excavatum. It causes less functional limitation; however, pectus carinatum can lead to significant psychological distress and thus warrants an equally aggressive management approach. Physical symptoms can include musculoskeletal chest wall discomfort particularly while lying prone, respiratory symptoms, or palpitations.

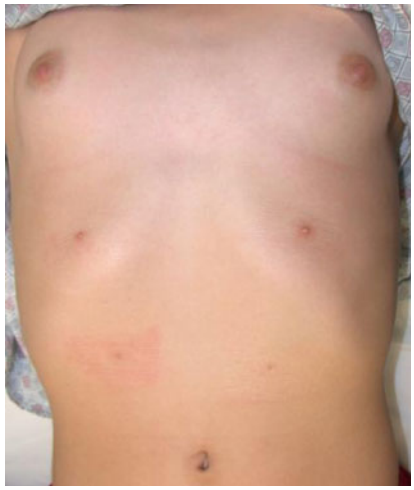
Exam Children with pectus carinatum may present at any age but typically are brought to medical attention at the time of puberty, as the deformity becomes more prominent during the adolescent growth spurt. Two distinct entities exist. The chondromanubrial variant describes protrusion of the manubrium, or superior component of the sternum, and has been termed the "pouter pigeon breast." The chondrogladiolar variant describes protrusion of the gladiolus or body of the sternum. This deformity has also been referred to as "keel chest."

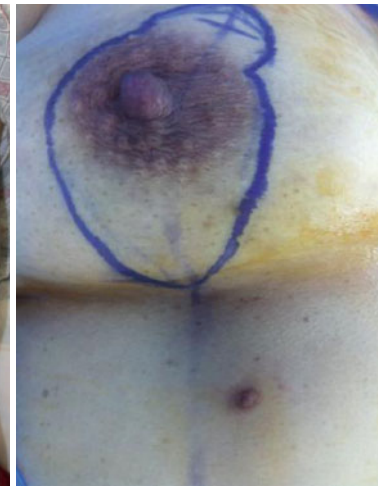
Treatment Compressive orthotic bracing, anterior sternal compression applied via an implanted metal bar (Abramson/Nuss repair), or removal of the abnormal rib cartilage while preserving the perichondrium and allowing regrowth of rib cartilage to the sternum in a more anatomic fashion combined with sternal osteotomy (Ravitch procedure).

3.1.5 Accessory Breast and Polythelia



Polythelia



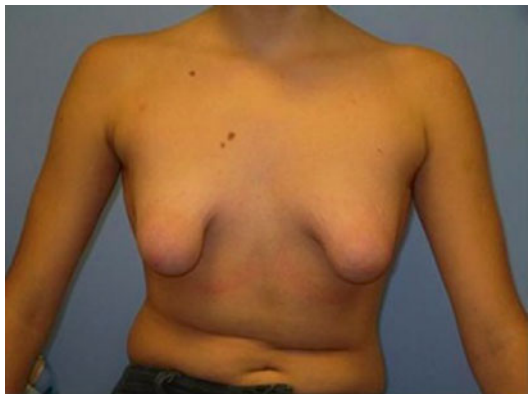


History Abnormal accessory breast tissue can be found as a mass that is located along the milk line and occur in 0.4–6 % of women. The tissue is usually asymptomatic, but patients may have complaints including swelling and tenderness of the affected region, irritation from clothing, and secreting milk or discharges. If constitutional symptoms are present, cancer should be ruled out. A complete family history should be taken of similar conditions and also of any history of breast cancer.

Examination Ectopic breast tissue is most common in the anterior axillary line but may also be present in locations such as the face, back, labia, and thighs and can involve multiple nipples (polythelia).

Treatment Surgical excision is recommended for adequate pathological evaluation as accessory breast tissue is subject to the same diseases as normal breast tissue. Liposuction is another option.

3.1.6 Tuberous Breast



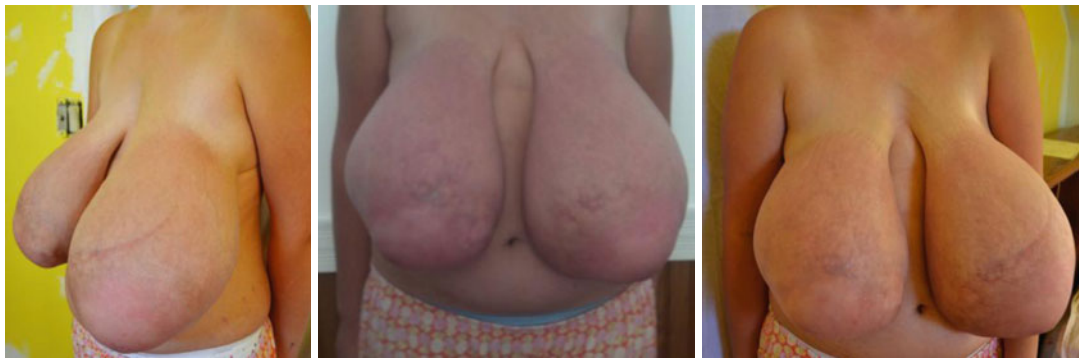
History A hypoplastic breast deformity that may affect bilateral or unilateral breasts in varying severity. Von Heimburg, Grolleau, and Meara have all proposed similar classification schemes that are the most widely used.

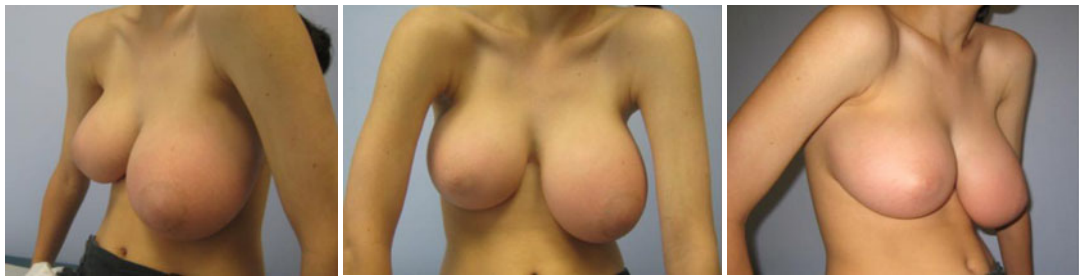
Exam The range of physical findings includes elevation of the IMF, a constricted breast base, glandular hypoplasia, deficient skin envelope, and herniation of the breast parenchyma into the NAC.

Treatment Numerous methods have been described with all attempting to release or enlarge a constricted base, augment volume as needed (implant and/or autologous), reposition the IMF, and reduce the size and herniation of the NAC. For mild-moderate deformities, this can be done via a periareolar incision in a single-stage manner, while severe cases may require multiple stages with tissue expansion.

3.1.7 Juvenile Hypertrophy

Virginal breast hypertrophy, juvenile mammary hypertrophy, juvenile gigantomastia





History Rare, extreme breast hypertrophy in peripubertal girls causing physical/psychological debilitation. Patients have a history of alarming rapid growth with breast pain, neck or back pain, and discomfort. This enlargement can occur at any time during the peripubertal period, sometimes occurring with the onset of thelarche and usually has a rapid phase lasting several months followed by a slower more indolent growth lasting years.

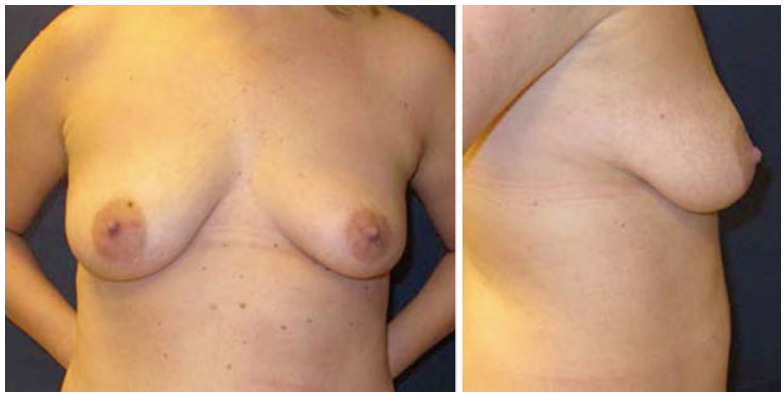
Exam Exam demonstrates a firm glandular breast that may be tender and asymmetric. Patients may have a slouching posture and shoulder grooving (due to bra straps) similar to benign macromastia. Enlargement of the NAC is present as are skin changes such as thinning, ulcer or necrosis, and prominent veins. Nodules or masses may be palpated. Diagnosis is done after ruling out endocrine factors or masses in the breast.

Treatment At the time of diagnosis, tamoxifen may be considered. A subcutaneous mastectomy with complete removal of breast tissue is the best option for recurrence but more deforming. Reduction mammoplasty has a better aesthetic but has a higher chance of recurrence; however, if followed by tamoxifen, the rate of recurrence is decreased. Patient should be counseled on the risk of recurrence with close follow-up.

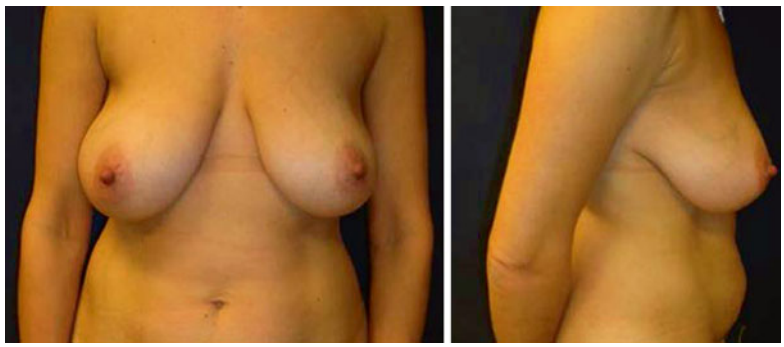
3.2 Breast, Acquired

3.2.1 Breast Ptosis

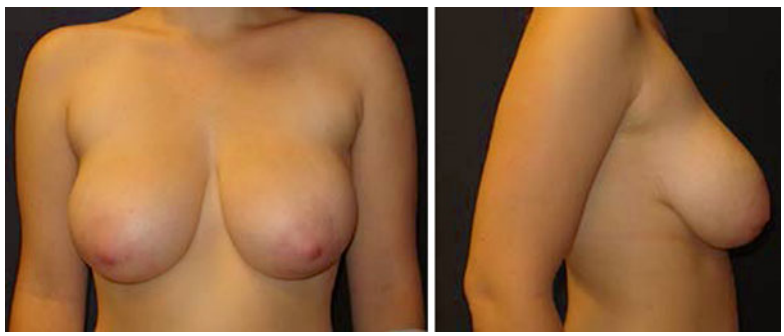
Ptosis grade 1



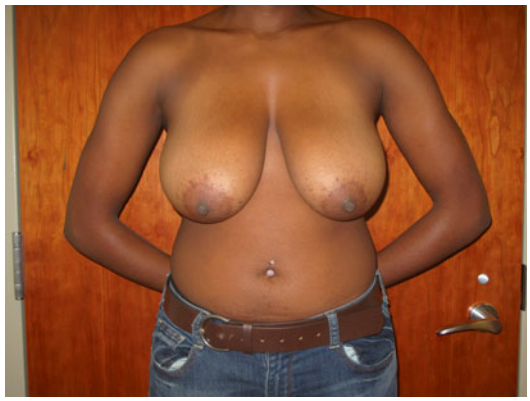
Ptosis grade 2



Ptosis grade 3



Ptosis grade 3 (severe)



Diagnosis Breast ptosis

History Breast ptosis describes a descent of the breast parenchyma (nipple-areolar complex (NAC) and breast tissue). Etiologies include congenital (lower pole hypoplasia), acquired (idiopathic, involutional—postpartum/menopause), and iatrogenic (post-reduction or augmentation). Presurgical history should include personal or family history of breast cancer, stage of breast development, pregnancy/menopause changes, size changes with weight gain or loss, previous procedures, lactation, and future pregnancy plans.

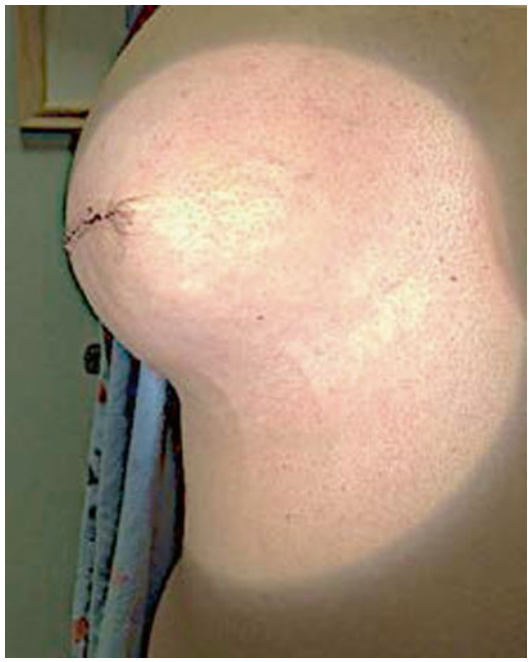
Exam Initial exam should include a breast exam and assessment of the degree of breast ptosis (Regnault's). Other important points to look for are symmetry, quality of breast tissue, skin quality and elasticity, scars and striae, and NAC shape and size.

Regnault's Classification

- *Grade I: Mild ptosis*—The nipple is at the level of the inframammary fold and above most of the lower breast tissue.
- *Grade II: Moderate ptosis*—The nipple is located below the inframammary fold but still lies anterior and superior to the lowest contour of the breast.
- *Grade III: Severe ptosis*—The nipple is below the inframammary fold and at the lowest portion of the breast, pointing downward.
- *Pseudoptosis (Glandular ptosis)*—The nipple is located either at or above the inframammary fold, while the lower portion of the breast falls below the fold. This is most often seen when a woman stops nursing, as her milk glands atrophy, and her breast tissue descends.

Treatment Directed by the classification, ranging from augmentation plus limited periareolar pexy to major skin resection. Mastopexy typically involves redistribution of the breast tissues with removal of excess skin. The procedure can be combined with breast augmentation to increase upper pole fullness.

3.2.2 Mondor's Disease



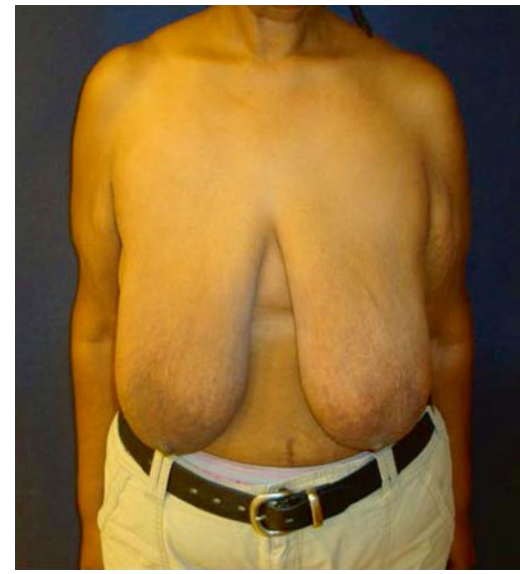
History Mondor's disease is a self-limiting benign disease caused by thrombosis of the superficial veins of the anterolateral chest wall possibly secondary to retractile scarring of the fascia after breast surgery or biopsies with concomitant vein thrombosis.

Examination Skin of the chest demonstrates tender cord-like lump. The diagnosis is confirmed by ultrasound—direct or indirect signs of thrombosis in the superficial veins.

Treatment Chest wall: Spontaneous recovery in 2–8 weeks, anticoagulation is not needed.

Post breast surgery: This is not a thrombotic process. Reports suggest that manual rupture of the fibrous bands ensures immediate functional recovery and pain relief.

3.2.3 Macromastia



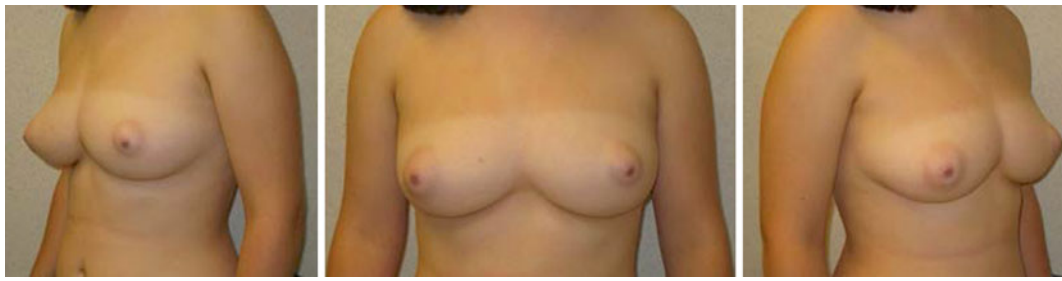
History Patients with macromastia present to the clinic with enlarged breasts that tend to be ptotic and that cause chest, neck, back, and shoulder pain; difficulty performing deep inspirations; and the inability to fit into proper clothing.

Exam Patients may show shoulder indentations from the brassiere and inframammary intertrigo. In patients over 30, a mammogram should be obtained for breast cancer screening pre-op. Measurements are taken to evaluate extent of breast ptosis and weight of the breasts.

Treatment Breast reduction using liposuction or surgical resection using one of many described techniques. Pedicle choice of central, inferior, superior medial, superior, lateral, or bucket handle. Skin resection including periareolar, lollipop, J scar, T scar, and a full Wise pattern chosen based on surgeon preference and operative need.

3.2.4 Symmastia "Uniboob"

Sixteen-year-old female

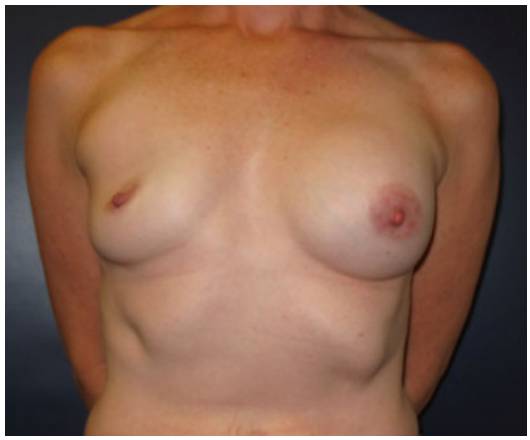


History Can be congenital, where there is a confluence of breast tissue across midline (rare), or acquired, after a breast augmentation in which one or both of the implants are displaced to far medially or across midline. This is more common when implants are placed subpectorally and when oversized implants are used.

Exam There will be an absence of the intermammary sulcus with either conjoined breast tissue or medialization of an implant, possibly across midline.

Treatment Treatment plan is determined by prior implant positioning, prior revisional surgery, existing parenchyma/skin thickness, and desired implant size. Different techniques utilizing capsulorrhaphy, capsular flaps, acellular dermal matrix, and/or creation of "new" implant pockets aim to define proper medial implant positioning.

3.2.5 Deflated/Ruptured Implant

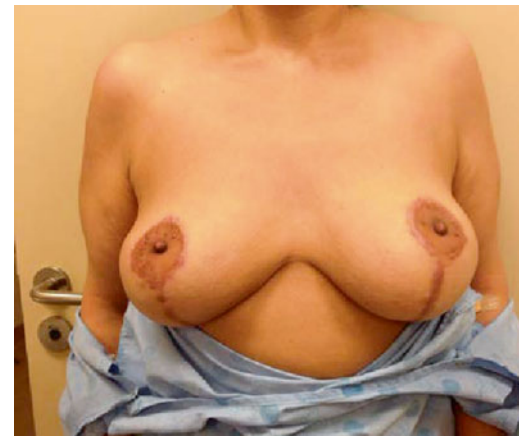


History For saline implants, patients will likely report a sudden or gradual (days) change in the size and shape of the affected breast. Alternatively, patients with gel implants may not experience a volumetric change after rupture but may have pain or an altered feel of the breast. Sometimes findings of a ruptured gel implant are discovered "incidentally" on routine radiographic screening (i.e., MRIs, mammograms).

Exam Can vary from decreased breast volume on affected side with possible implant palpability to normal physical exam. ± tenderness. As shown in the example above, the right breast implant is ruptured, and the breast appears deflated with less projection compared to the contralateral side.

Treatment Directed toward restoring symmetry with contralateral breast with implant replacement if the patient desires. This includes removal of ruptured implant, capsulectomy or capsulotomy as needed, and exchange of the contralateral implant, and/or revision of the contralateral side.

3.2.6 Bottoming-Out

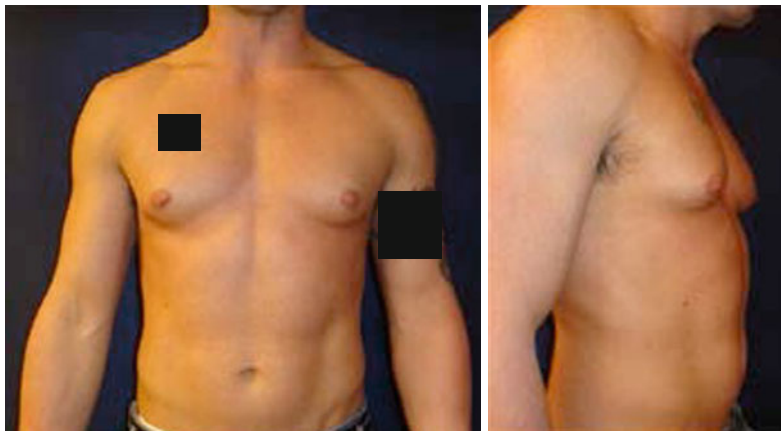


History This can occur following reduction mammoplasty, implant augmentation, or augmentation/mastopexy procedures in which of too much breast parenchyma (or implant) descends below the nipple-areolar complex. This can be caused by inadequate resection of lower pole tissue, alteration of original IMF, postoperative gravitational forces and skin stretching, improper nipple placement (high), and/or a long nipple-IMF distance.

Exam Inferior pole fullness with lack of upper pole volume. The nipple may be too high and the N-IMF distance too long.

Treatment Revisionary surgery is directed toward the underlying etiology. This may require resection of inferior pole tissue (parenchyma and/or skin), repositioning of the implant and/or NAC, and alteration (or repair) of the IMF.

3.2.7 Gynecomastia

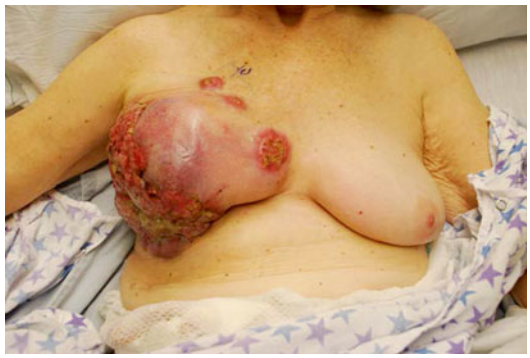


History Excessive growth of glandular breast tissue, may be unilateral or bilateral. Etiologies include physiologic (puberty, elderly), pathologic (adrenal/testicular tumors, cirrhosis), pharmacologic (marijuana, steroids, cimetidine, etc), or idiopathic (most common). There is no increased risk of breast cancer, *except* in cases of Klinefelter's syndrome.

Exam A full H&P should be completed to identify sources of androgen deficiency or estrogen excess, including testicular and breast exam. The amount of glandular tissue, fatty tissue, and excess skin or ptosis and the size of the NAC are key aspects that will direct surgical treatment.

Treatment Should be directed by the severity of the disease and the goals of the patient, with particular attention to scarring. Direct excision via a peri- or circum-areolar incision is best for limited fibrous subareolar involvement or when decreasing the size of the NAC. For larger cases, power-assisted or ultrasound-assisted liposuction with "pull-through" technique is required. Persistent skin excess and/or ptosis can be corrected at the expense of visible scarring.

3.2.8 Breast Cancer



The full scope of breast cancer is beyond the scope of this atlas. The plastic surgeon should be familiar with all breast cancer reconstruction procedures and timing of the reconstruction. Some features are discussed below.

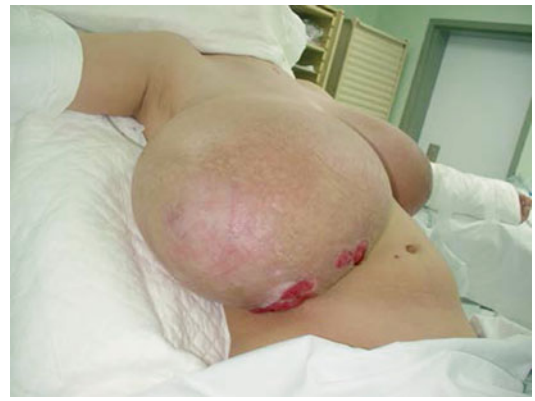
History Cancer of the breast is the most frequently diagnosed life-threatening cancer in women and accounts for 29 % of all female cancers. It is the leading cause of cancer death in women. Breast cancer is usually asymptomatic and first detected as a mammogram abnormality.

Exam Exam can demonstrate a change in breast shape, skin and or nipple changes, bloody discharge, or a lump. Further evaluation is through a mammogram, breast MRI, and possible biopsy.

Treatment Breast cancer is treated surgically with radiation and/or chemotherapy with hormonal or chemotherapeutic agents as adjuncts.

If breast reconstruction is desired, the current choices are implant or autologous reconstruction (or a combination of both).

3.2.9 Peau d'Orange

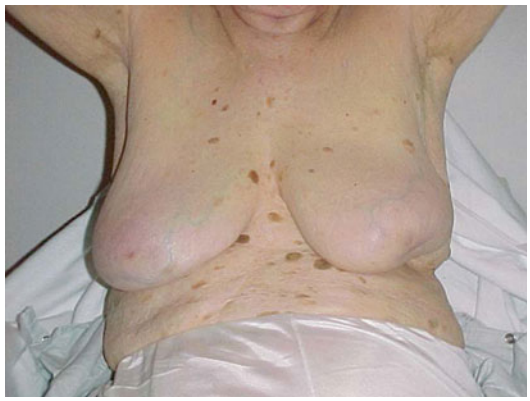


History Refers to a dimpled appearance or texture of skin (French for "skin of an orange") which is caused by lymphatic edema. While it can occur anywhere and is used for various disease states, the term is often used to describe skin changes in advanced or inflammatory breast cancer.

Exam Orange peel appearance of edematous skin with numerous pits.

Treatment Directed at underlying condition, but often skin change is irreversible. For malignant etiology, excisional lines should not cross affected dermis.

3.2.10 Nipple Retraction

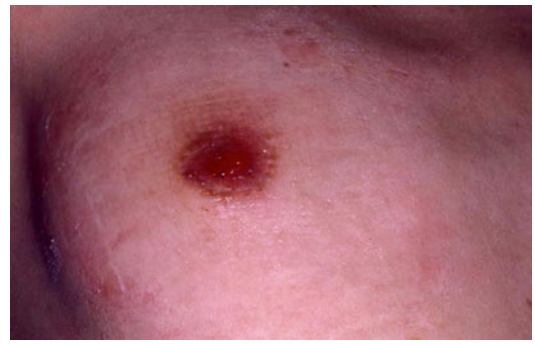


History May be congenital or acquired and unilateral or bilateral and often not evident until puberty or postpartum. Although often unpleasing to the patient aesthetically, it may also be a source of irritation or difficulty with lactation. Neoplastic etiologies (breast cancer, Paget's disease) should be considered and ruled out, as well as a history and/or future desire for breast feeding.

Exam A thorough breast exam with concomitant age-appropriate imaging (ultrasound, mammography, etc.). The ability to pull the nipple out to length and the ability to maintain its projection are key factors in treatment planning.

Treatment Multiple techniques have been described including purse-string sutures, ductal division with tissue rearrangement, or various duct-preserving techniques.

3.2.11 Mammary Paget's Disease

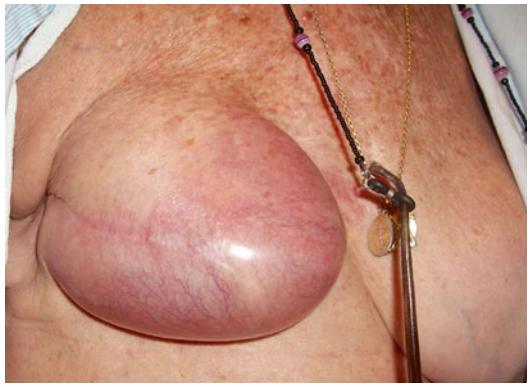


History A rare type of breast cancer (1–3 %), often associated with an underlying in situ or invasive carcinoma. It is characterized by eczematoid changes in the nipple-areolar complex that is refractory to topical treatments and can cause burning, itching, or soreness. More extensive destruction of the NAC with ulceration may ensue. A thorough breast workup is indicated including imaging (mammography, MRI, etc.) and biopsy.

Exam Inflamed, red, and flaky eczema like rash of the NAC. Diagnosis is confirmed by presence of Paget's cells on biopsy.

Treatment Mastectomy or breast conservation with XRT in certain cases.

3.2.12 Implant Hematoma (Breast hematoma)



History A patient presents with pain and swelling of the breast after undergoing breast augmentation or reconstruction with implant.

Exam Breast hematoma has been shown to develop in 1–3 % of patients who have undergone breast augmentation. Hematomas can be seen as late as 14 days postoperatively. The affected breast appears significantly larger and is more firm to palpation than the contralateral breast. There may not be erythema or ecchymosis.

Treatment Prompt surgical exploration to evacuate the hematoma and careful hemostasis are the mainstay of treatment. The implant can be replaced if there is no evidence of infection.

References

Hypoplastic Breast

1. Gautam AK, Allen Jr RJ, LoTempio MM, Mountcastle TS, Levine JL, Allen RJ, Chiu ES. Congenital breast deformity reconstruction using perforator flaps. Ann Plast Surg. 2007;58(4):353.
2. Sadove AM, van Aalst JA. Congenital and acquired pediatric breast anomalies: a review of 20 years' experience. Plast Reconstr Surg. 2005;115:1039–50.
3. Georges LK. Tumors, tumor-Like masses, nipple discharge, and anomalies of the breast. Symptoms and Signs in Pediatric Surgery. Springer 2012, pp. 229–238.

Poland's Syndrome

4. Cingel V, Bohac M, Mestanova V, Zabojnikova L, Varga I. Poland syndrome: from embryological basis to plastic surgery. Surg Radiol Anat. 2013;35(8):639–46.
5. Ram AN, Chung KC. Hand/peripheral nerve: special topic Poland's syndrome: current thoughts in the setting of a controversy. Plast Reconstr Surg. 2009;123(3):949–53. doi:10.1097/PRS.0b013e318199f508.

Pectus Excavatum

6. Robicsek F. Surgical treatment of pectus excavatum. Chest Surg Clin N Am. 2000;10(2):277–96.
7. Snel BJ, Spronk CA, Werker PM, van der Lei B. Pectus excavatum reconstruction with silicone implants: long-term results and a review of the english-language literature. Ann Plast Surg. 2009;62(2):205–9.

Pectus Carinatum

8. Cohee AS, Lin JR, Frantz FW, Kelly Jr RE. Staged management of pectus carinatum. J Pediatr Surg. 2013;48(2):315–20. 2013-01-01.
9. Desmarais TJ, Keller MS. Pectus carinatum [review]. Curr Opin Pediatr. 2013;25(3):375–81.

Accessory Breast and Polythelia

10. Aydogan F, Baghaki S, Celik V, et al. Surgical treatment of axillary accessory breasts. Am Surg. 2010;76:270–2.
11. Down S, Barr L, Baildam AD, Bundred N. Management of accessory breast tissue in the axilla. Br J Surg. 2003;90(10):1213–4.
12. Madej B, Balak B, Winkler I, et al. Cancer of the accessory breast—a case report. Adv Med Sci. 2009;54:308–10.
13. Sadove AM, van Aalst JA. Congenital and acquired pediatric breast anomalies: a review of 20 years' experience. Plast Reconstr Surg. 2005;115:1039–50.

Tuberous Breast

14. Grolleau J, et al. Breast base anomalies. Treatment strategy for tuberous breasts, minor deformities and asymmetry. Plast Reconstr Surg. 2000;104(7): 2040–8.
15. Meara J, et al. Tuberous breast deformity: principles and practice. Ann Plast Surg. 2000;45:607–11.
16. Rees T, Aston S. The tuberous breast. Clin Plast Surg. 1976;3:339–47.
17. Sadove A, van Aalst J. Congenital and acquired pediatric breast anomalies: a review of 20 years' experience. Plast Reconstr Surg. 2005;115(4):1039–50.
18. van Aalst J, et al. Pediatric chest wall and breast deformities. Plast Reconstr Surg. 2009;124(Supp): 38–49e.
19. von Heimburg D. Refined version of the tuberous breast classification. Plast Reconstr Surg. 2000;105: 2269e70.

Juvenile Hypertrophy

20. Baker SB, Burkey BA, Thornton P, LaRossa D. Juvenile gigantomastia: presentation of four cases and review of the literature. Ann Plast Surg. 2001;46:517–25; discussion 525–26.
21. Hoppe IC, Patel PP, Singer-Granick CJ, Granick MS. Virginal mammary hypertrophy: a meta-analysis and treatment algorithm. Plast Reconstr Surg. 2011; 127:2224–31.

Breast Ptosis

22. Jones, G. Mastopexy. In: Jones, G. editor. Bostwick's Plastic and Reconstructive Breast Surgery. 3rd Ed. St. Louis: Quality Medical Publishing; 2010. pp. 642–729.
23. Regnault P. Breast ptosis. Definition and treatment. Clin Plast Surg. 1976;3(2):193–203.
24. Rinker B, Veneracion M, Walsh CP. Breast ptosis: causes and cure. Ann Plast Surg. 2010;64(5):579–84.

Mondor's Disease

25. Bejanga BI. Mondor's disease, analysis of 30 cases. J R Coll Surg Edinb. 1992;37:322–4.
26. Pugh CM, Dewitty RL. Mondor's disease. J Natl Med Assoc. 1996;88:359–63.
27. Salemis NS, Merkouris S, Kimpouri K. Mondor's disease of the breast. A retrospective review. Breast Dis. 2011;33(3):103–7.

Macromastia

28. Cruz-Korchin N, Korchin L. Vertical versus wise pattern breast reduction: patient satisfaction, revision rates, and complications. Plast Reconstr Surg. 2003;112(6):1573–8. discussion 1579–81.
29.Degeorge BR Jr, Colen DL, Mericli AF, Drake DB. Reduction mammoplasty operative techniques for improved outcomes in the treatment of gigantomastia. Eplasty. 2013;13:e54. eCollection 2013.
30.Hammond DC, Loffredo M. Breast reduction. Plast Reconstr Surg. 2012;129(5):829e–39e.

Symmastia "Uniboob"

31. Hammond DC. Discussion. The "neosubpectoral" pocket for the correction of symmastia. Plast Reconstr Surg. 2009;124(3):704–5.
32. Spear S, et al. The "neosubpectoral" pocket for the correction of symmastia. Plast Reconstr Surg. 2009; 124:695–703.
33. Spence RJ, Feldman JJ, Ryan JJ. Symmastia: the problem of medial confluence of the breasts. Plast Reconstr Surg. 1984;73(2):261–9.

Deflated/Ruptured Implant

34. Chung KC, Malay S, Shauver MJ, Kim HM. Economic analysis of screening strategies for rupture of silicone gel breast implants. Plast Reconstr Surg. 2012;130(1): 225–37.

35. Song JW, Kim HM, Bellfi LT, Chung KC. The effect of study design biases on the diagnostic accuracy of magnetic resonance imaging for detecting silicone breast implant ruptures: a meta-analysis. Plast Reconstr Surg. 2011;127(3):1029–44.

Bottoming-Out

36. Brown RH, Izaddoost S, Bullocks JM. Preventing the "bottoming out" and "star-gazing" phenomena in inferior pedicle breast reduction with an acellular dermal matrix internal brassiere. Aesthetic Plast Surg. 2010;34(6):760–7.
37. Hall-Findlay EJ. A simplified vertical reduction mammaplasty: shortening the learning curve. Plast Reconstr Surg. 1999;104(3):748–59. discussion 760–3.
38. Tebbetts JB. A process for quantifying aesthetic and functional breast surgery: I. Quantifying optimal nipple position and vertical and horizontal skin excess for mastopexy and breast reduction. Plast Reconstr Surg. 2013;132(1):65–73.

Gynecomastia

39. Braunstein G. Clinical practice: gynecomastia. N Engl J Med. 2007;357:1229–37.
40. Rohrich R, et al. Classification and management of gynecomastia: defining the role of ultrasound-assisted liposuction. Plast Reconstr Surg. 2003;111:909–23.
41. Simon B, et al. Classification and surgical correction of gynecomastia. Plast Reconstr Surg. 1973;51:48.

Breast Cancer

42. Jones G. Bostwick's plastic and reconstructive breast surgery. 3rd Ed. St. Louis: Quality Medical Publishing; 2010.
43. Disa JJ. Breast reconstruction: prosthetic techniques. In: Thorne CH, editor. Grabb and Smith's plastic surgery. Philadelphia: Lippincott; 2007. p. 625–34.
44. Gill PS, et al. A 10 year retrospective review of 758 flaps for breast reconstruction. Plast Reconstr Surg. 2004;113(4):1153–60. Source: Lippincott Williams & Wilkins (LWW).
45. Kaplan JL, Allen RJ. Cost-based comparison between perforator flaps and TRAM flaps for breast reconstruction. Plast Reconstr Surg. 2000;105(3):943–8.
46. Namnoun JD. Breast reconstruction: TRAM flap techniques. In: Thorne CH, editor. Grabb and Smith's plastic surgery. Philadelphia: Lippincott; 2007. p. 641–8.

Peau d'Orange

47.Kell MR, Morrow M. Surgical aspects of inflammatory breast cancer. Breast Dis. 2005–2006;22:67–73.
48. Singletary SE, Cristofanilli M. Defining the clinical diagnosis of inflammatory breast cancer. Semin Oncol. 2008;35(1):7–10.

Nipple Retraction

49. Han S, Hong YG. The inverted nipple: its grading and surgical correction. Plast Reconstr Surg. 1999;104(2):389–95.
50. Kim DY, et al. Correction of inverted nipple: an alternative method using two triangular areolar dermal flaps. Ann Plast Surg. 2003;51(6):636–40.

Mammary Paget's Disease

51. Sabir S, James W, Schuchter L. Cutaneous manifestations of cancer. Curr Opin Oncol. 1999;11(2):139–44.
52. Sakorafas GH, et al. Paget's disease of the breast. Cancer Treat Rev. 2001;27(1):9–18.

Implant Hematoma

53. Maxwell GP, Clugston PA. Management of complications following augmentation mammoplasty. In: Georgiade GS, Riefkohl R, Levin LS, editors. Textbook of plastic, maxillofacial and reconstructive surgery. Baltimore: Williams & Wilkins; 1997. p. 736.

Integument

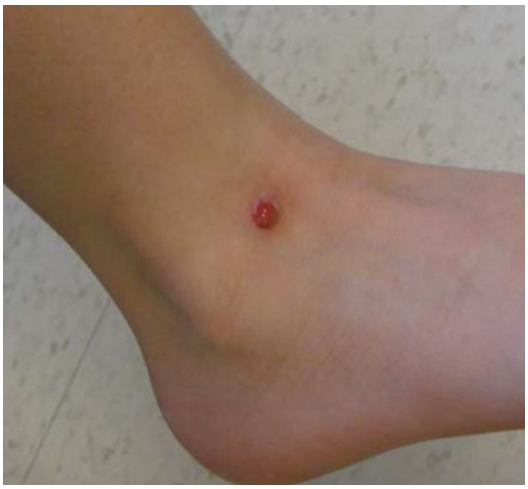
4

David Tauber, Alan Yan, Michael J. Yaremchuk, Fuad Hashem, Ellen Roh, Mohamed Amir Mrad, Arin Greene, John Mullen, Moath Alhakami, Khalid Murrad, Tahira I. Prendergast, and W. McIver Leppard

4.1 Tumors, Benign

4.1.1 Pyogenic Granuloma

History Also referred to as lobular capillary hemangioma (LCH), pyogenic granuloma is a common, benign vascular proliferation that can be found on the skin or subcutaneous tissue. Histology includes hyperplastic clusters of capillaries arranged in a lobular architecture. Unclear etiology, however, female sex hormones, minor trauma, chronic wounds, and viral infections have been implicated.



D. Tauber, MD (✉) • A. Yan, MD • M.J. Yaremchuk, MD
Division of Plastic and Reconstructive Surgery, Craniofacial Plastic Surgery, Massachusetts General Hospital, Harvard Medical School, Boston, Massachusetts, USA
e-mail: dmtauber@yahoo.com; alanyanmd@gmail.com
http://www.dryaremchuk.com

E. Roh, MD • J. Mullen, MD
Division of Plastic Surgery, Department of Surgery, Massachusetts General Hospital, Harvard Medical School, Boston, MA, USA
e-mail: ekroh@partners.org

F. Hashem, MD
Department of Plastic Surgery, King Faisal Specialist Hospital, Riyadh, Saudi Arabia
e-mail: fuadkhashem@gmail.com

M.A. Mrad, MD
Division of Plastic Surgery, Department of Surgery, King Faisal Specialist Hospital and Research Center, Alfaisal University, Riyadh, Saudi Arabia
e-mail: mmrad@kfshrc.edu.sa
http://www.drmrad.com

A. Greene, MD
Children's Hospital of Boston, Harvard Medical School, Boston, MA, USA
e-mail: Arin.Greene@childrens.harvard.edu

M. Alhakami, MD
Division of Plastic and Reconstructive Surgery, Riyadh, Saudi Arabia
e-mail: muath.hakami@hotmail.com

K. Murrad
King Saud University, Riyadh, Saudi Arabia
e-mail: khalidmurad1992@gmail.com

T.I. Prendergast, MD • W.M. Leppard, MD
Division of Plastic and Reconstructive Surgery, Medical University of South Carolina, Charleston, SC, USA
e-mail: Tahira.prendergast@gmail.com; leppardwm@gmail.com

© Springer International Publishing Switzerland 2016
R. Hazani et al. (eds.), *Clinical Diagnosis in Plastic Surgery*, DOI 10.1007/978-3-319-17094-7_4

Exam Usually smooth, red to purple, sessile or pedunculated lesions that can bleed easily.

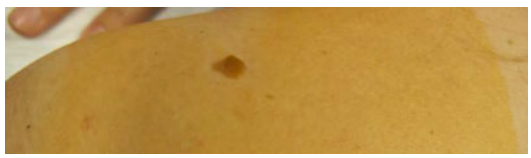
Treatment While some pyogenic granulomas may resolve spontaneously, most have a tendency to bleed easily and require treatment. Current therapeutic modalities include topical imiquimod, cryotherapy, electrodessication, curettage, excision, laser therapy, sclerotherapy, and microembolization. Recurrence rates range from 3.7 to 43.5 %.

4.1.2 Seborrheic Keratosis

History A benign lesion seen mostly in middle-aged and older patients.

Exam A well-circumscribed dark brown lesion with waxy appearance.

Treatment Options include shave excision, electrodesiccation, freezing with liquid nitrogen, or simple excision. Excision will also prevent the potential confusion with melanoma.

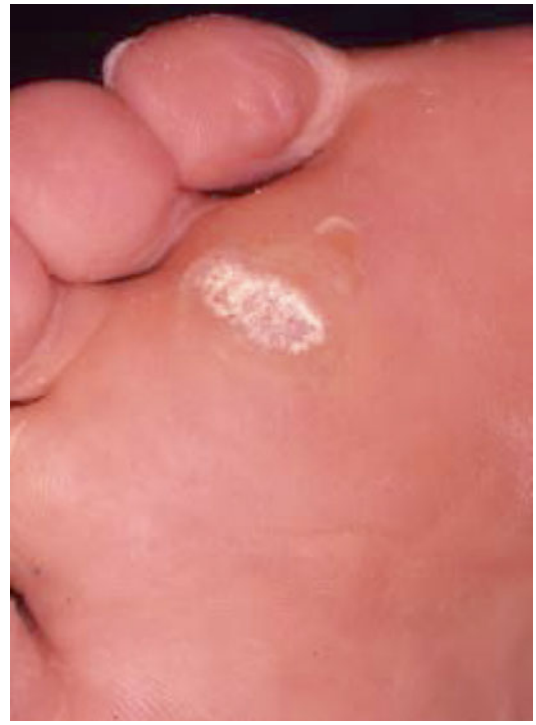


4.1.3 Verrucae (Warts)

History Cutaneous warts are common skin lesions caused by human papillomavirus infection.

Exam Histopathologically, warts contain dilated, congested blood vessels in the dermal papillae extending along the rete ridges, covered by a thick layer of ortho- and parakeratosis. The clinical picture reveals black dots, which are thrombosed capillaries.

Treatment There is some evidence that topical treatments containing salicylic acid have a therapeutic effect and some evidence for the efficacy of dinitrochlorobenzene and pulsed dye laser. Cryotherapy demonstrates no efficacy over placebo.

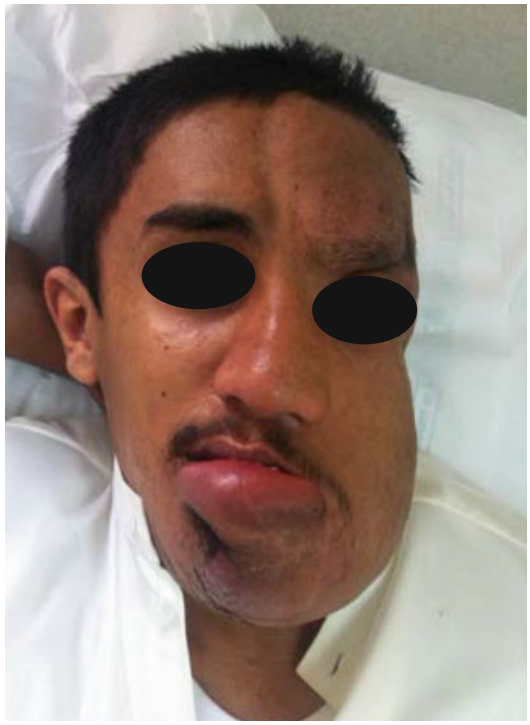


4.1.4 Neurofibromatosis

History A neurofibroma is a benign nerve sheath tumor in the peripheral nervous system. It is usually found in individuals with neurofibromatosis type I (NF1), an autosomal dominant genetically inherited disease. It affects an estimated one in 3,000–4,000 individuals. The gene for NF1 was cloned on chromosome 17q11.2. Four types of neurofibromatous lesions exist: cutaneous, subcutaneous, nodular plexiform, and diffuse plexiform. Cutaneous neurofibromas are the most common type; they can vary in number from just a few to several thousands, with the highest density occurring over the trunk. The cutaneous and subcutaneous types are both benign, but a slightly greater risk of malignant transformation exists.

Exam The hallmark clinical features present in over 90 % of all patients are café-au-lait spots and neurofibromas; however, only 20 % of children and 80 % of adults demonstrate cutaneous lesions. Neurofibromatosis can result in a range of symptoms from physical disfiguration and pain to cognitive disability. MRI may be a useful adjunct.

Treatment Removal of cutaneous lesions can be performed through excision with or without tissue expansion, cauterization, or CO_2 laser. Removal of deeper lesions can be technically demanding and must be weighed against the potential harm to involved structures (especially in the face).

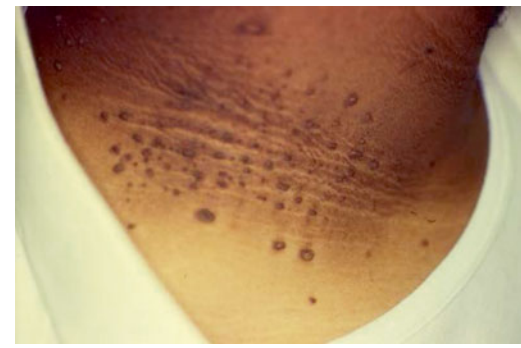


4.1.5 Acrochordon (Skin Tag)

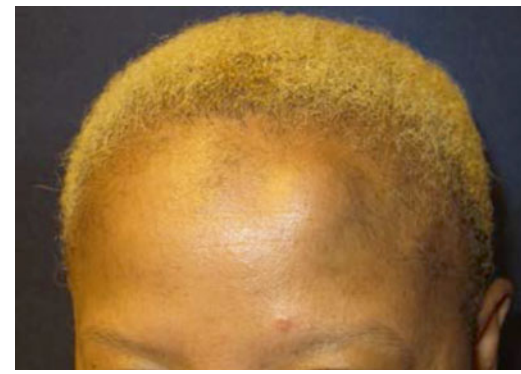
History An acrochordon (plural acrochorda, and also known as a (cutaneous) skin tag, or fibroepithelial polyp) is a small benign tumor that forms primarily in areas where the skin forms creases, such as the neck, armpit, and groin. They may also occur on the face, usually on the eyelids. They are derived from ectoderm and mesoderm and represent a hyperplastic epidermis. They are found in 25 % of persons and increase in number with age. Obesity is a predisposing factor.

Exam Skin tags usually are attached to the skin by a thin stalk (pedunculated) but also can be sessile. They range in size from less than 1 mm to 1 cm in diameter and are skin colored or brown. Acrochorda are harmless and typically painless and do not grow or change over time.

Treatment Simple electrocautery or scissor excision at the base of the stalk is sufficient. Local anesthesia usually is not necessary. Cryotherapy is effective for small lesions. Recurrences of skin tags are common. Pathological evaluation is unnecessary unless skin tags are present in childhood, because they may be the initial presentation of nevoid basal cell carcinoma syndrome.



4.1.6 Lipoma

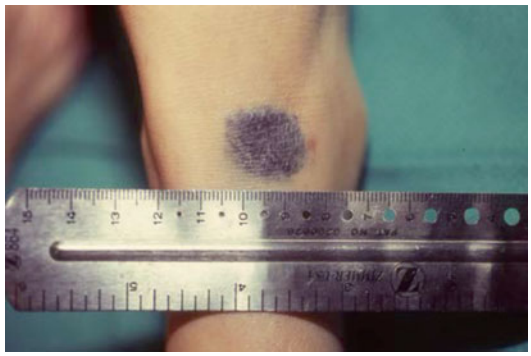


History Lipomas are commonly found in adults from 40 to 60 years of age but can also be found in children. A lipoma is a benign tumor composed of adipose tissue. It is the most common form of soft tissue tumor. Some sources claim that malignant transformation can occur, while others say that this has yet to be convincingly documented.

Exam Lipomas are soft to the touch, usually movable, and are generally painless. Many lipomas are small (under one centimeter diameter) but can enlarge to sizes greater than 6 cm.

Treatment Excision for cosmetic reasons or if transformation is expected.

4.1.7 Blue Nevus



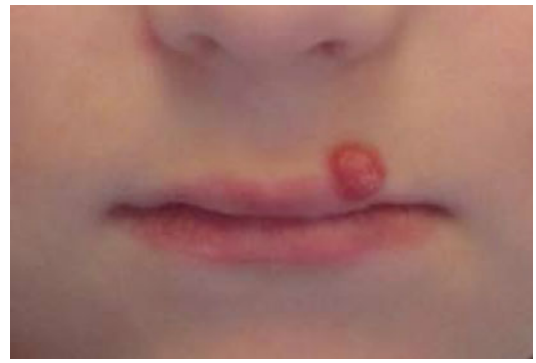
History Described over a century ago, blue nevus is a type of melanocytic nevus. True pathogenesis is unknown but is believed to be associated to dermal arrest of neural crest melanocytes that fail to reach the epidermis. It usually presents as a solitary lesion on head/neck area, dorsal aspect of the hand and feet, or sacral region.

Exam Blue nevus usually presents as a small (<1 cm) smooth-surfaced papule, macule, or plaque with a gray/blue to blue/black color.

Treatment

No treatment is needed for blue nevus; however, if required for cosmetic reasons or in order to rule out malignancy, surgical excision is recommended.

4.1.8 Spitz Nevus



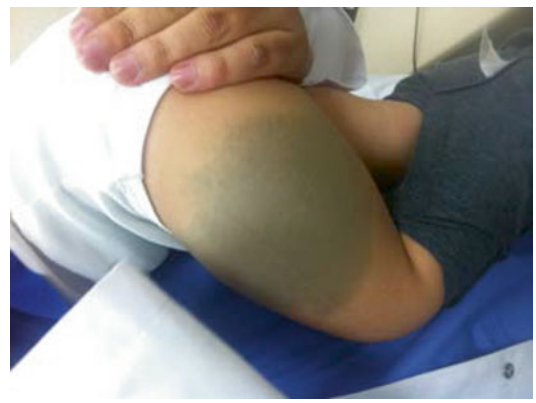
History Spitz nevus is a type of melanocytic nevus which presents a rapid growth phase, up to 1 cm, with a subsequent static phase, with some of them showing spontaneous resolution. It may rarely present with color changes, bleeding, and/or pruritus. Over 70 % of cases present during the first two decades of life.

Physical Spitz nevus typically presents as red or pigmented, dome-shaped papules or nodules, most commonly localized at the face or legs.

Treatment

Because of its unpredictable prognosis, surgical excision with histopathologic evaluation of margins is recommended.

4.1.9 Mongolian Spot

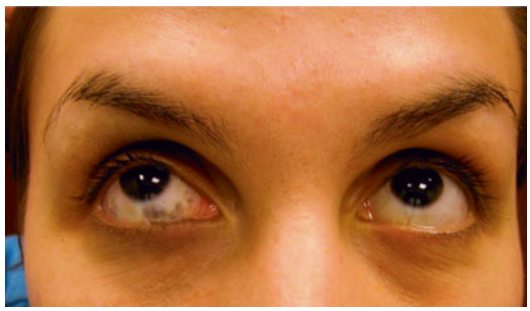


History
Also known as congenital dermal melanocytosis, Mongolian spot is an asymptomatic birthmark, usually overlying sacrococcygeal area. It represents dermal arrest of neural crest melanocytes during migration into epidermis. It usually shows spontaneous resolution within the first 5 years of age.

Physical Exam
Mongolian spot classically presents as a blue-gray macular pigmentation involving the lumbosacral region; however, more extensive involvement up to shoulders and flanks can be seen.

Treatment
No treatment is required due to its benign prognosis. Most patients show resolution during the first years of life. However, if persistent, camouflage makeup and Q-switched laser therapy is usually recommended.

4.1.10 Nevus of Ota

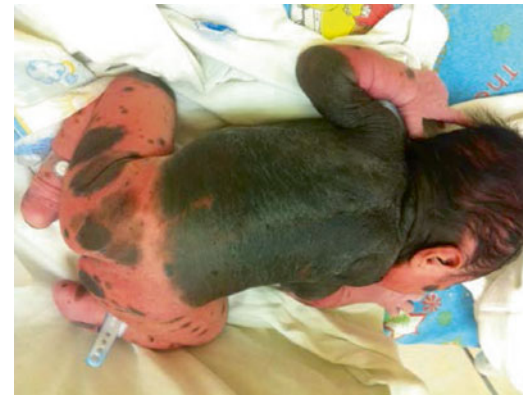


History Patient presents with a bluish gray facial lesion.

Exam Nevus of Ota, or nevus fuscoceruleus ophthalmomaxillaris, is a dermal melanocytic hamartoma that presents as bluish hyperpigmentation along the first or second branches of the trigeminal nerve. Extracutaneous involvement has been reported, especially ocular involvement. Approximately 60 % of these lesions are present at birth or develop by age 10, 80 % are seen in female children, and 5 % occur bilaterally.

Treatment Malignant transformation is rare for nevus of Ota. Treatment (Q-switched ruby laser) is only indicated for aesthetic reasons.

4.1.11 Giant Nevus

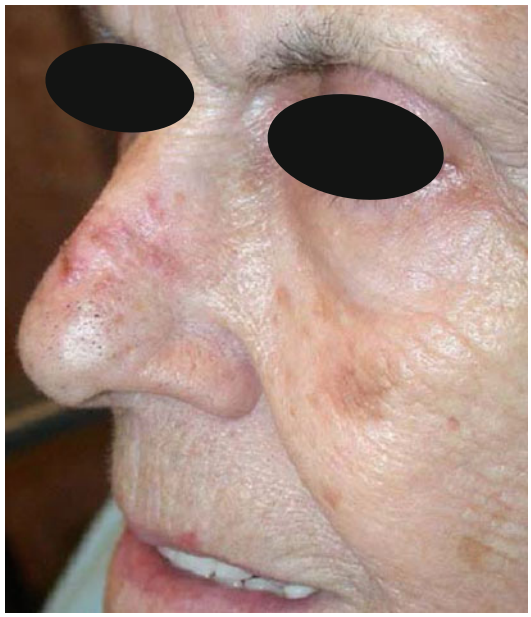


History Congenital pigmented nevus with a diameter greater than 20 cm in adults. In infants, involvement of total body surface more than 1 % in the head and neck and 2 % in other body parts. Lesion is present at birth and grows in proportion to the growing child.

Examination Lesion is an elevated, pale brown. Older lesions are characterized by increasing pigmentation and excessive hair growth. If lesion is axial, rule out spinal defect. Biopsy any suspicious lesion with ulceration/uneven pigmentation, bleeding, or change in shape. The goal is to rule out melanoma. If lesion is over the scalp, MRI will be useful to rule out neurocutaneous melanosis.

Treatment The risk of malignant transformation to melanoma is 3–12 %. Treatment is total excision (staged or with expander according to size). Shaving in neonatal period may improve appearance. If treatment is not feasible, periodic cancer surveillance should be done.

4.1.12 Actinic Keratosis

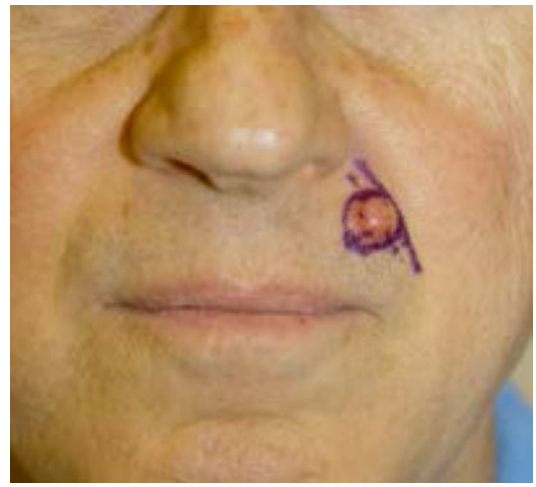


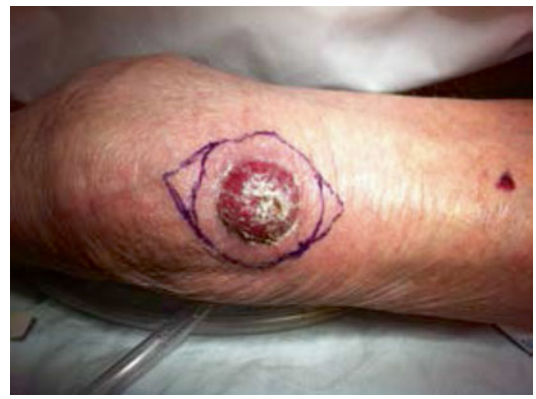
History Found in middle- to old-aged persons with a history of chronic sun exposure. Chronically immunocompromised transplant surgeries. It is the most frequently occurring premalignant lesion.

Examination Rough-surfaced epidermal lesions that present as an erythematous macule or thin papule with adherent scale or crust on sun-exposed areas.

Treatment Has the potential to transform into squamous cell carcinoma. Photodynamic therapy, cryotherapy, curettage and cautery, topical diclofenac, topical 5-fluorouracil, and imiquimod can be used. The use of sunscreens reduces the rate of formation of new solar keratoses.

4.1.13 Keratoacanthoma





History A benign solitary lesion found on sun-exposed parts of the body. Typically found on the face and dorsum of the hand. Despite its benign nature, it can differentiate into a subtype of squamous cell carcinoma and can exhibit a rapid growth phase with extensive tissue destruction.

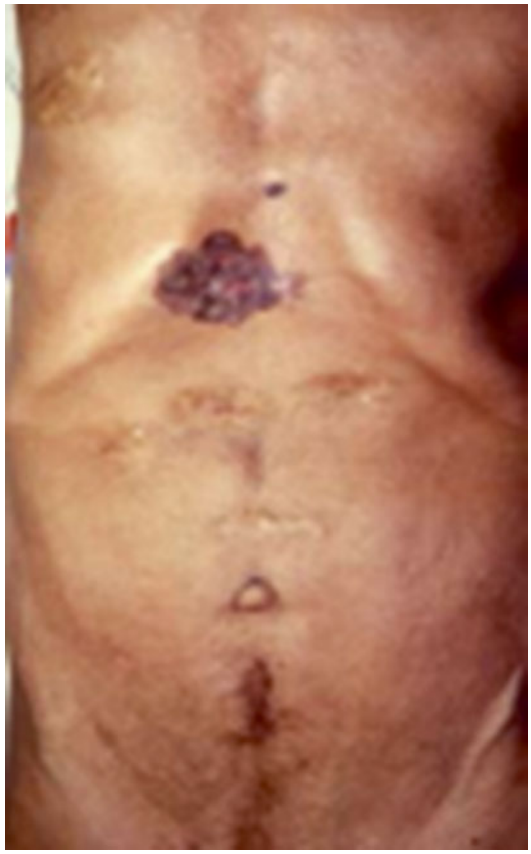
Exam A firm, raised, half-moon lesion covered by a normal skin except for a central destructive crater filled with a keratotic plug.

Treatment Complete excision to avoid confusion with a highly differentiated squamous cell carcinoma. The scar resulting from the excision

is cosmetically preferred over the scar from spontaneous resolution of the lesion. Other modalities include chemotherapy, radiotherapy, phototherapy, and watchful waiting. Recurrence of lesions after local therapy is approximately 3–5 % depending on the treatment modality.

4.2 Tumors, Malignant

4.2.1 Bowen's Disease

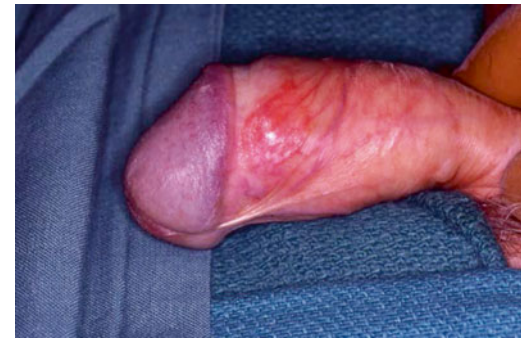


History An intraepidermal squamous cell cancer (SCC in situ). Lesion is slowly expanding and presents in patients with a history of chronic UV exposure, chronic arsenic ingestion from contaminated water, immunosuppression, or HPV. Five percent of lesions progress to SCC.

Examination It is a well-demarcated persistent red scaly plaque or patch usually found on the legs of women and ears and scalp of men. Multiple lesions may indicate previous exposure to carcinogen.

Treatment Destructive therapies like cryotherapy, curettage, and cautery for small areas and those with good healing potential (such as the face). Surgical excision is an alternative and to some is the choice. Topical 5-fluorouracil and photodynamic therapies are also options.

4.2.2 Erythroplasia of Queyrat

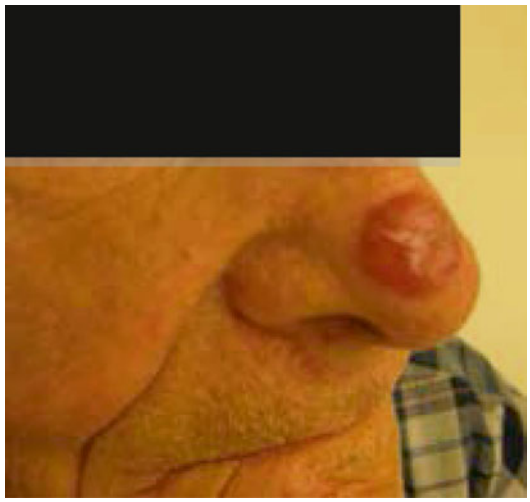


History Predominantly a disease of uncircumcised men aged 20–80 years. Distinct clinical variant of intraepidermal squamous carcinoma. The disease progresses slowly, and the interval between onset and diagnosis may be years. The cause is unknown.

Exam Manifests itself by single or multiple asymptomatic papules or plaques on the glans penis or periurethrally. The bright red lesions may be ulcerated.

Treatment A therapeutic regimen of 5 % 5-fluorouracil cream applied to lesion(s) twice daily for 4–5 weeks has produced a high cure rate and maintained penile integrity and function.

4.2.3 Basal Cell Carcinoma (BCC)

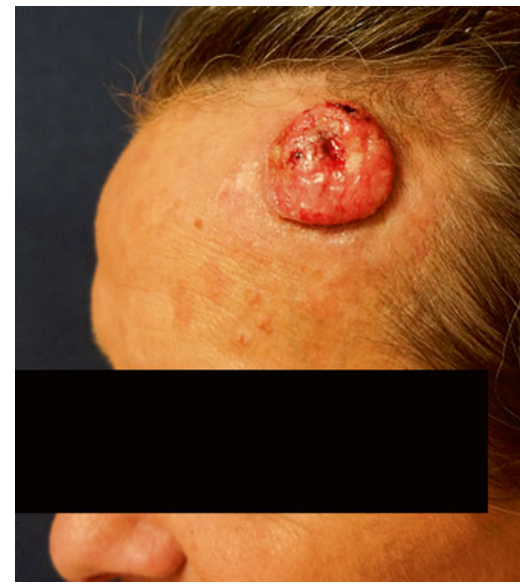


History BCC is a slow-growing, locally invasive malignant epidermal skin tumor predominantly affecting Caucasians. Metastasis is extremely rare, and morbidity results from local tissue invasion and destruction particularly on the face, head, and neck. The most significant etiological factors appear to be genetic predisposition and exposure to ultraviolet radiation. The sun-exposed areas of the head and neck are the most commonly involved sites. Sun exposure in childhood may be especially important.

Exam Clinical appearances and morphology are diverse and include nodular, cystic, superficial, morpheaform (sclerosing), keratotic, and pigmented variants. Lesions often begin as a small, dome-shaped bump and are often covered by small, superficial blood vessels called telangiectasias. The texture of such a spot is often shiny and translucent, sometimes referred to as "pearly." Histology shows three-dimensional infiltration through the irregular growth of subclinical finger-like outgrowths, which remain connected to the main tumor, mass.

Treatment Surgical resection remains the gold standard in BCC treatment, with Mohs micrographic surgery typically utilized for high-risk lesions. Standard excision is with 3–5 mm margins (lager in more diffuse subtypes). Suitable alternate treatment options for appropriately selected primary low-risk lesions may include pulsed dye therapy, cryotherapy, topical imiquimod, and 5-FU. Radiotherapy is a suitable alternate for surgical methods for treatment in older patient populations. Electrodesiccation and curettage (ED&C) is a commonly used primary treatment option for low-risk lesions.

4.2.4 Squamous Cell Carcinoma (SCC)

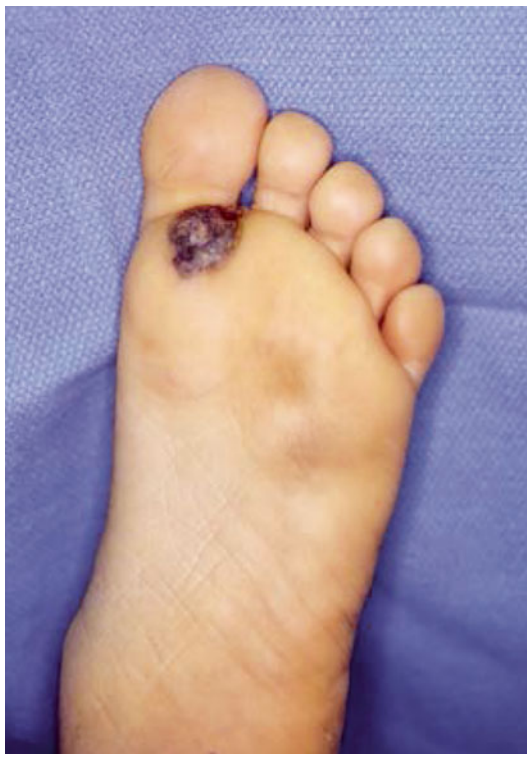


History A squamoproliferative lesion due to malignant, invasive, proliferation of epidermal keratinocytes. Found in the elderly with a history of chronic sun exposure. Additional risk factors are exposure to arsenic, tar, HPV, photochemotherapy, and chronic immunosuppression. Chronic sites of radiation and ulceration can see malignant transformation. Smoking history (lip SCC) and other premalignant lesions are risk factors. Annual incidence rate ranges between 15 and 35 per 100,000 inhabitants.

Exam Expanding erythematous plaque or nodule with an ill-defined, indurated base, usually ulcerated, with surface scale and crust in sun-exposed sites. Lymph node enlargement if metastasized. Diagnosis is through biopsy.

Treatment Surgical excision with 8–10 mm margins confirmed with frozen sections and Mohs micrographic surgery are options. Radiotherapy if rapidly enlarging or not fit for surgery. Alternative second-choice treatment options are curettage, electrodesiccation, or cryosurgery. Elective nodal dissection based on suspicion or exam.

4.2.5 Melanoma



History Melanoma is a malignant proliferation of melanocytes. Patients often report a change in a preexisting pigmented lesion. Patients with a prior history of melanoma, intermittent high UVR with sunburns in childhood/adolescence, or immunosuppression are at higher risk. Patients with paler Fitzpatrick skin types (red or blonde hair, blue eyes, and the inability to tan) are also at higher risk. A history of freckles, lentigines, large numbers of benign melanocytic nevi, and the presence of atypical nevi should raise suspicions.

Examination Diagnostic criteria include a diameter greater than 1 cm, increasing size, variation in pigment, irregular edge, the presence of inflammation, crusting or bleeding, altered sensation, or itch. Histological diagnosis with Breslow thickness, nodal status, and the presence of metastasis used for staging.

Types

- *Lentigo maligna melanoma*: hyperpigmented patch with varying shades of brown, irregular border, evolves from lentigo maligna (elderly people, slowly enlarging irregularly pigmented macule, on the cheek or temple).
- *Superficial spreading melanoma*: greater than 0.5 cm in diameter at presentation, with variable pigmentation from pale brown to blue black, an irregular edge, and surface oozing or crusting. The most common site is the back (men) and lower legs (women); horizontal growth.
- *Nodular melanoma*: a rapidly enlarging, frequently ulcerated, blue-black nodule; vertical growth.
- *Acral lentiginous melanoma*: irregularly pigmented macule/patches with varying shades of brown or black and irregular borders on the sole of the foot or palm of the hand.

Treatment Surgical excision. Recommendations are a 5mm margin for in situ melanoma, 1 cm margin for invasive melanoma up to 1 mm depth, and 2cm margin for melanoma thicker than 1mm. Consider Mohs resection for smaller margins. SNL biopsy is recommended for lesions thicker than 0.75mm.

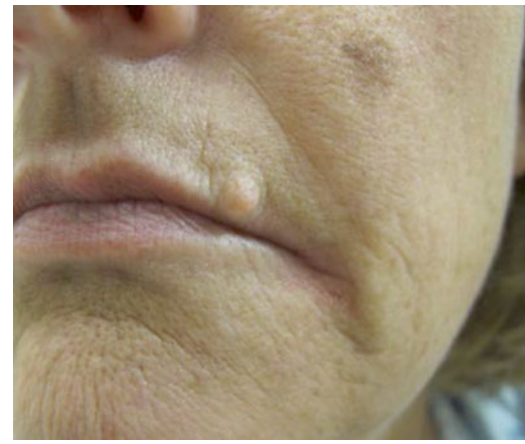
4.2.6 Subungual Melanoma

History A rare manifestation of cutaneous melanoma representing 0.7–3 % of all cases. Carries a poor prognosis with reported 5-year survival rates ranging from 18 to 58 %. Factors that contribute to this include delayed diagnosis and consequent advanced Breslow depth as well as the high incidence of ulceration in the primary lesion. In many patients, a presumed diagnosis of subungual hematoma, trauma, and/or onychomycosis leads to the long delay.

Examination Linear pigmented streak in the nail or an isolated nail dystrophy, accompanied by pigmentation of the proximal nail fold (Hutchinson's sign).

Treatment Amputation at mid-level of proximal bone (mid-phalanx). More distal amputations are associated with greater recurrence. Sentinel lymph node biopsy is performed with elective dissection if positive.

4.2.7 Lentigo Maligna

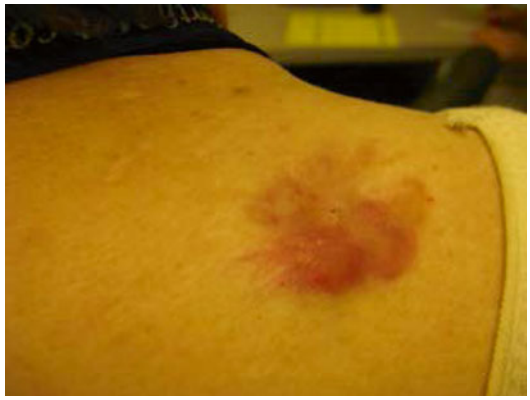


History Lentigo maligna (LM) is a subtype of melanoma in situ that typically develops on sun-damaged skin. Presentation may be quite subtle and delayed diagnosis is common. Recurrence following standard therapies is common. The estimated lifetime risk of LM progressing to LM melanoma is 5 %.

Exam LM appears as a large patch that expands centrifugally with variable shades of tan, brown, dark brown, or black. Clinical margins are often ill defined. Histologic evaluation can be difficult due to the widespread atypical melanocytes that are present in the background of long-standing sun damage.

Treatment Surgical excision remains the treatment of choice. Five to ten millimeter margins are recommended. Recurrence, however, is 8–20 % of LM with 5 mm margins. Frozen section, staged repair, and Mohs micrographic excision can help achieve more complete excision. Conservative treatment for poor operative candidates can include radiation therapy, immunotherapy, and cryotherapy.

4.2.8 DFSP (Dermatofibrosarcoma Protuberans)

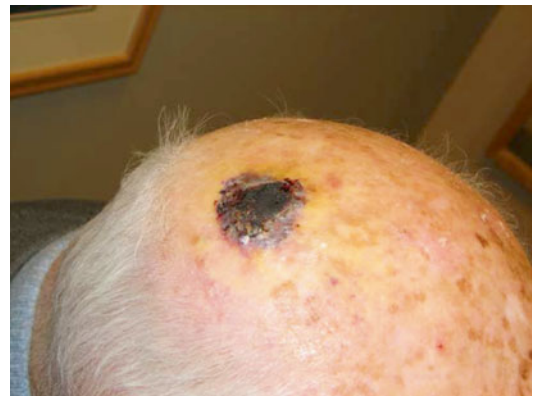


History Dermatofibrosarcoma protuberans (DFSP) is an uncommon, intermediate-grade fibrohistiocytic sarcoma of the dermis that accounts for 2 % of all tissue sarcomas. The estimated incidence of DFSP is 0.8 per 1 million persons per year. It is locally aggressive tumor with tendency for recurrence. Patients have a history of trauma or scar at the site with size changes and progression (sudden progression after slow-growing period) being typical. Lesion often recurs after excision.

Examination Slow-growing large nodule or plaque with multiple protuberances, commonly on trunk > proximal extremities. Fixed to the skin but moving freely to deeper tissue. DFSP rarely metastasizes. If so, the metastasis-free interval is generally long, and metastases are frequently preceded by multiple local recurrences.

Treatment Mohs surgery or wide local excision with 2.5–3 cm margins (frequently recurs with latter) ± adjuvant radiotherapy as an adjuvant for positive surgical margins, after planned marginal excision in critical anatomic sites or as an exclusive treatment in advanced cases with no feasible surgical approach.

4.2.9 Angiosarcoma



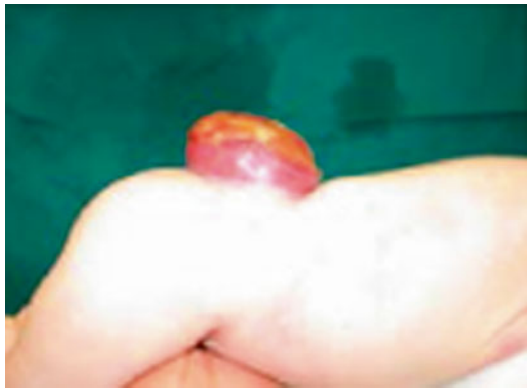
History Angiosarcoma is a rare, aggressive, malignant vascular sarcoma with a poor prognosis, historically associated with 5-year overall survival (OS) rates between 10 and 30 %. Patients have a previous history of radiotherapy or chronic lymphedema: Stewart-Treves syndrome.

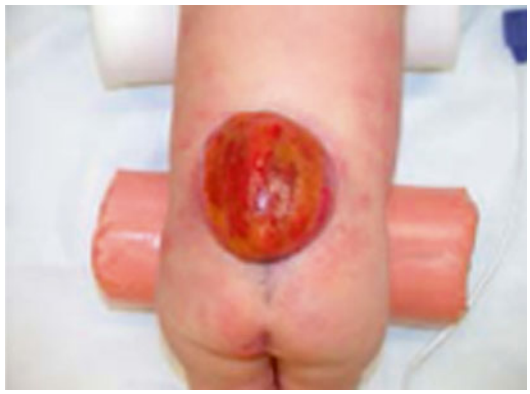
Examination Presents as a spreading bruise-like discoloration with poorly defined margins and satellite lesions. Lesion is initially a violaceous plaque on the face or scalp but is later followed by the appearance of dusky red nodules, ulceration, and edema.

Treatment Wide excision and wide field radiotherapy but the prognosis is poor. Chemotherapy is routinely used in patients with advanced regional or distant metastatic disease with progression-free survival rates ranging from 1 to 5 months. The probability of hematogenous dissemination is relatively high.

4.3 Integument, Congenital

4.3.1 Myelomeningocele



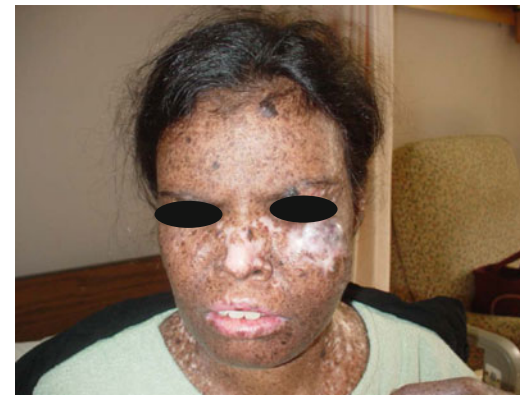


History Myelomeningocele occurs at a rate of approximately 0.5–1/1,000 live births. Also known as spina bifida, myelomeningocele occurs because of failure of neural tube closure between 20 and 28 days of gestation.

Exam A sac consisting of the meningeal membranes enclosing the spinal elements can be seen protruding from the midline back through the unfused portion of the spine. Myelomeningocele is associated with severe morbidity including but not limited to musculoskeletal deformities, neurogenic bladder and bowel dysfunction, learning disabilities, and varying degrees of sensorimotor compromise of the lower extremities.

Treatment Surgical repair includes the dissection and closure of five separate layers: the arachnoid, dura, fascia, a subcutaneous layer, and skin. Closure of the soft tissue defect is individualized based on the size of the defect and can involve primary closure and myocutaneous and fasciocutaneous flaps. The rate of complications including CSF leak and wound complications can be as high as 3 % and 10 %, respectively.

4.3.2 Xeroderma Pigmentosa

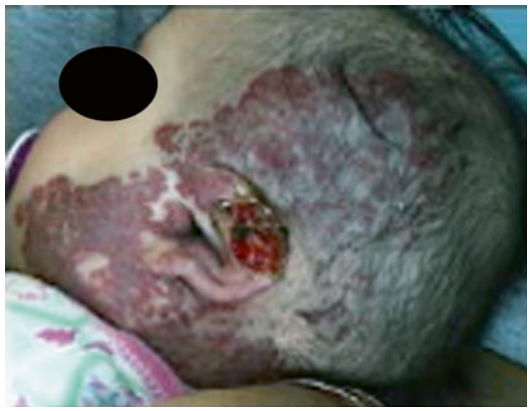


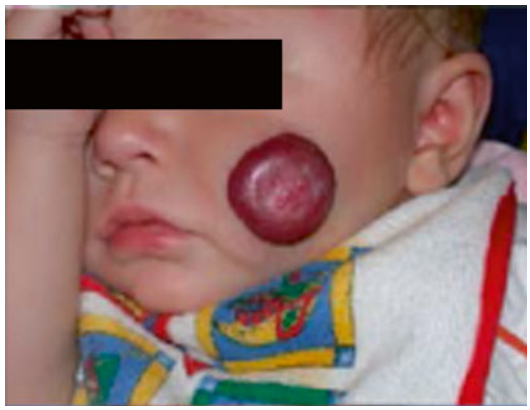
History Family history and/or personal history of extreme photosensitivity, exaggerated blistering sunburn, accelerated photoaging, and early onset of skin malignancy. Autosomal recessive, due to defect in DNA repair. Prone to a variety of benign and malignant skin tumors with at least a thousandfold increased risk of melanoma and nonmelanoma skin cancer.

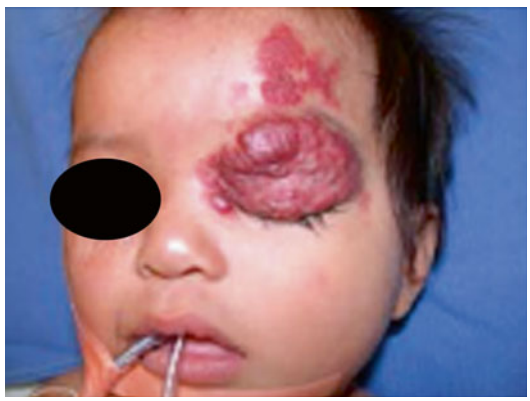
Examination Exposed skin ages prematurely with dryness, freckles, and roughness. Solar lentigines by age of 2. Atrophic skin shows telangiectasia. Eye abnormalities (photophobia, conjunctivitis, ectropion, corneal opacification) and neurologic abnormalities can occur. Workup includes irradiation skin tests, which show enhanced, delayed erythemal responses to UVB, and by demonstration of reduced DNA repair in cultured fibroblasts.

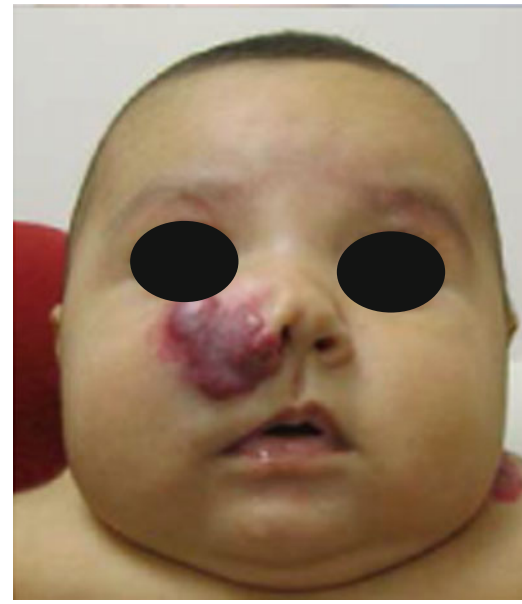
Treatment Minimize UV exposure with highly protective clothing, sunglasses, and sunscreens. Frequent examination and aggressive surveillance.

4.3.3 Hemangioma







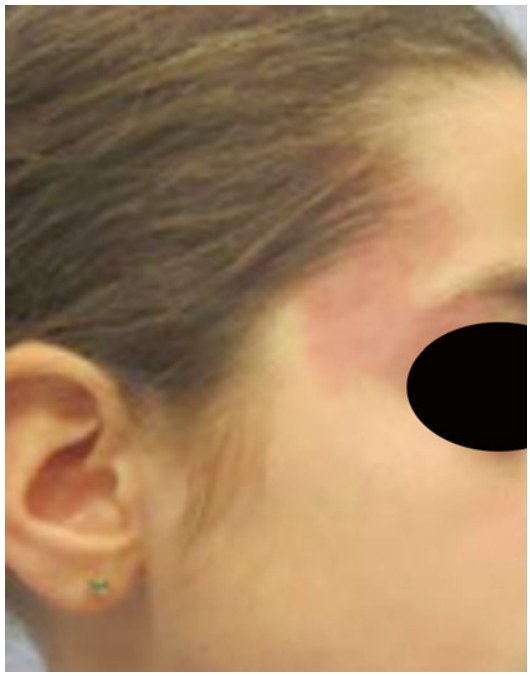


History Hemangiomas are the most common true benign vascular neoplasm of infancy. The incidence is 1.0–5 %. Risk factors include low birth weight, female sex, and Caucasian. There is some family tendency toward formation. The lesion is typically noted 1–2 weeks following birth, rapidly enlarges over the first several months of life, and then regresses during early childhood. Early intervention is indicated when the lesion causes visual field disruption, respiratory obstruction, congestive heart failure, severe hemorrhage, or serious disfigurement.

Exam Hemangiomas are heterogeneous in appearance and their appearance changes throughout their life cycle. They can be superficial or deep and be found anywhere in the body.

Treatment Most infantile hemangiomas are observed, but problematic lesions are treated. First-line treatment is now propranolol; however, corticosteroid injection, oral prednisolone, lasers, and excision are also used with success.

4.3.4 Capillary Malformation

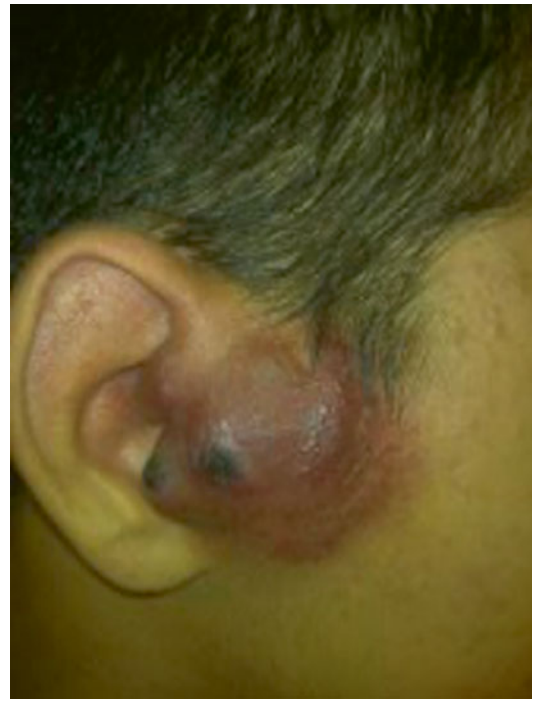


History Capillary malformation is present at birth and slowly darkens over time. Affects about 0.5 % of the population and is often referred to as "port wine stain."

Exam Lesions are frequently on the face in the V1 and V2 distribution. Lesions usually start as a blemish with a pink or purplish hue but can be highly variable in appearance. MRI is recommended if it involves V1 to rule out Sturge-Weber.

Treatment Treatment is pulsed-dye laser (580–595 nm) to lighten the appearance of the lesion. Surgery is used in refractory cases.

4.3.5 Arteriovenous Malformation (Parkes Weber Syndrome)

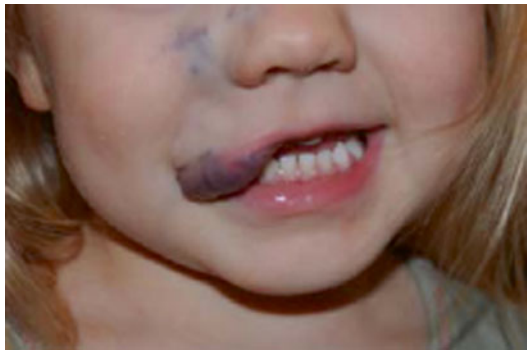


History AVM is the most frequent high-flow vascular malformation. They are present at birth but usually become apparent in the first or second decade of the patient's life (earlier in male than in female individuals) and slowly enlarge over time. AVM is characterized by the presence of a "nidus," a central mass of a low-resistance arteriolovenular shunt that is connected to afferent arteries and efferent veins.

Exam Demonstrates a mass that is slightly compressible and may demonstrate a reddish or bluish discoloration if present near the surface of the skin. In addition to causing a deformity, lesions can become ulcerated and bleed. Bruits are frequently present. U/S, MRI, and CT are useful adjuncts.

Treatment Observation is advocated in asymptomatic cases given the difficulty of achieving cure. Embolization and/or resection may be attempted to decrease symptoms or attempt cure; however, these can carry significant risk.

4.3.6 Venous Malformation (Klippel-Trenaunay Syndrome)

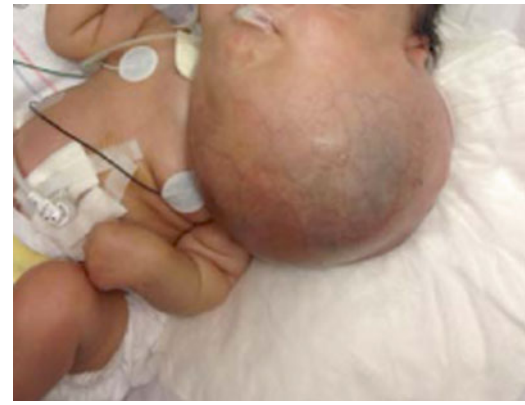


History Venous malformation is the most common cervicofacial vascular malformation, affecting 1–4 % of individuals. Venous malformations are present at birth and can slowly enlarge. They consist of masses of veins and venules of different dimensions with single layers of epithelial cells. Hyperplasia is absent.

Exam The lesion presents as a soft, bluish, compressible mass that enlarges when the patient reclines. The formation of phleboliths from localized intravascular coagulation (LIC) can be palpated as hard mobile masses and are pathognomonic of venous malformation. US, Doppler, and MRI are used for diagnosis.

Treatment First-line treatment is sclerotherapy with ethanol or sodium tetradecyl sulfate (STS). Resection for cure is very challenging and is reserved for removal after sclerotherapy or for the removal of small lesions or symptomatic lesions that have failed sclerotherapy.

4.3.7 Lymphatic Malformation



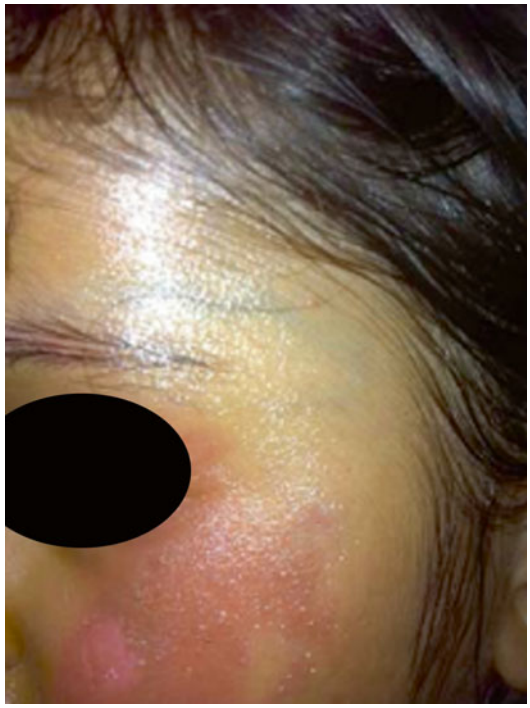
History Also know as "cystic hygroma," these lesions are composed of vessels or large chambers lined by single layer of epithelial cells. They are present at birth and can slowly enlarge, become infected, and can bleed. Classified by size as macro- or microcystic (<2 cm^3).

Exam Extremely variable appearance from small and focal to diffuse causing elephantiasis. Can be distinguished from other malformations because it lacks bruising and pulsations, is noncompressible, and does not swell when reclined. Diagnosis with US, Doppler, or MRI.

Treatment First-line therapy for macrocystic lymphatic malformations is sclerotherapy; usually with ethanol, bleomycin, doxycycline, and picibanil. Microcystic lesions with spaces too small to treat with sclerotherapy are only amenable to resection.

4.4 Integument, Acquired

4.4.1 First Degree: Superficial



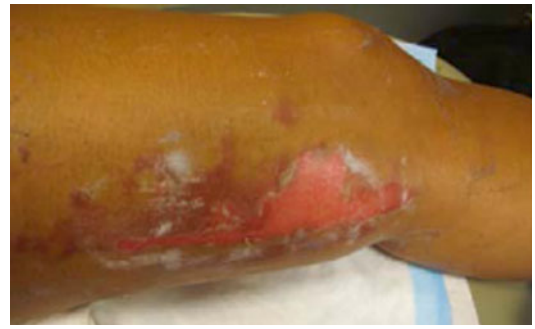
History Burns occur in varying severity from first degree to fourth degree. First-degree burns are the least severe and occur from flash burns and sunburns. These burns only involve the superficial epidermis, and the epidermal barrier remains intact. The risk of infection and the metabolic response are minimal.

Exam The skin is red, dry, and painful. The epithelium is intact and does not look wet.

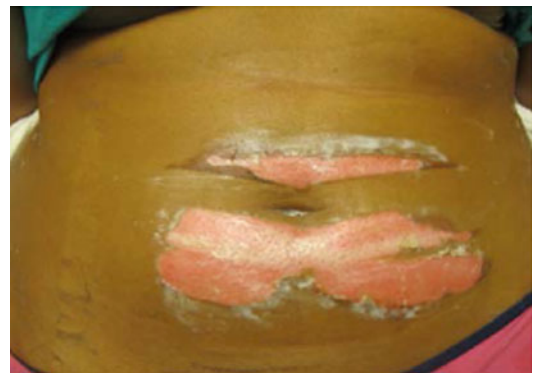
Treatment The key component of treatment is immediate cooling under cool water and analgesics. Addition of antimicrobial ointment is helpful. There is very little risk of poor healing and scarring.

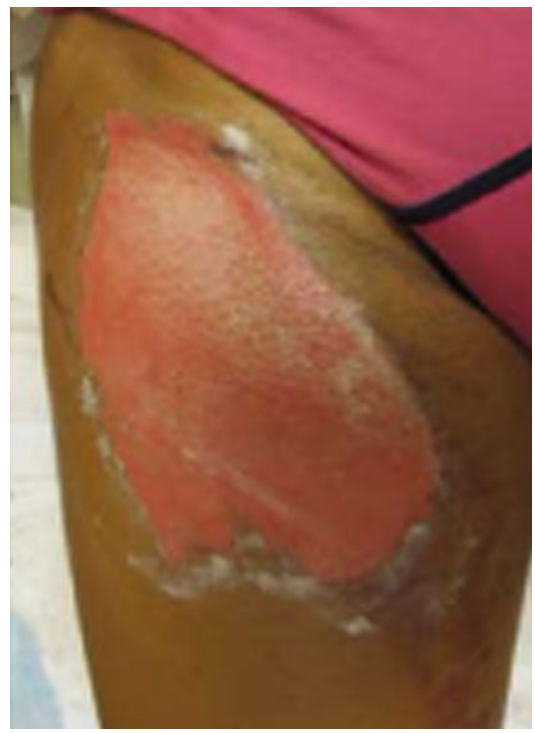
4.4.2 Second Degree: Partial Thickness

4.4.2.1 Superficial Partial Thickness



4.4.2.2 Deep Partial Thickness



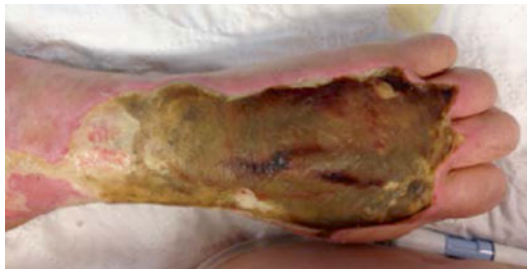


History The most common causes of second-degree burns are hot water and soup. In second-degree burns, the epidermis is completely destroyed and there is partial dermal loss. The depth of the injury is important and is described as superficial partial thickness or deep partial thickness. When the epidermal appendages are spared, the wound can heal by proliferation and migration of these cells.

Exam On exam, the skin is edematous, red, painful, wet, and possibly blistering. Deep partial-thickness burns may have a yellow or white appearance and be more dry.

Treatment Immediate treatment involves cooling the area with cool (not ice) water. A superficial dermal injury can heal in 7–10 days, while a deep dermal injury can take 3 weeks to heal. The depth of the wound is an important factor. There is a likelihood of having scar contracture and hypertrophy. The wounds are treated with antibiotic dressing to prevent infection during the healing process. There is a possibility of surgery to cover the wound if deep.

4.4.3 Third Degree: Full Thickness

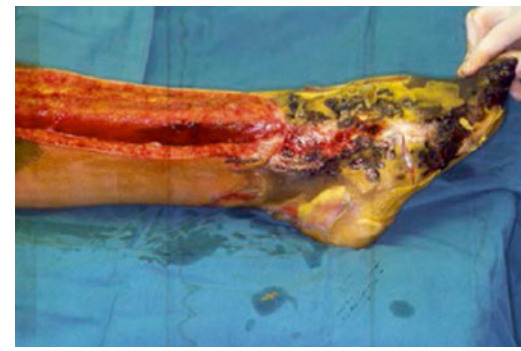


History The most common causes of third-degree burns are flame and grease. The burn is full thickness through the epidermis and dermis.

Exam The burn wound is pale and non-blanching. Also, the wounds are contracted, insensate, and leathery. There are typically surrounding areas of first- and second-degree burns.

Treatment Depending on the size of the wound, the patient may need resuscitation using the Parkland formula. The Parkland formula is based on the percent of total body burn, and this is based on the "rule of nines." The burns need to be covered with antibiotic dressing and require debridement. After debridement, the wounds are typically covered with autografts. If there is limited donor site for autografts, homograft, xenografts, or cultured epidermal autografts are used for coverage. There is often scarring and contractures requiring secondary revisional surgery.

4.4.4 Fourth Degree



History Fourth-degree burns are the most severe. They are completely through the dermis and into the underlying subcutaneous tissues including the fat, bone, and muscle.

Exam The burns are black and charred with eschar. They are painless and dry. There are also surrounding areas of first-, second-, and third-degree burns.

Treatment Patients with fourth-degree burns are often critical, and the initial approach to them is similar to the ABCs of trauma. The Parkland formula is used to guide resuscitation. This is based on the percent of total body burn utilizing the "rule of nines." Early debridement is typically required, and escharotomies are used as well. After debridement, the wounds are covered with autografts. If there is limited donor site for autografts, homograft, xenografts, or cultured epidermal autografts are used for coverage. These patients are at a high risk of infection and possible death. As with third-degree burns, there are often scarring and contractures requiring secondary revisional surgery.

4.4.5 Burn Contracture

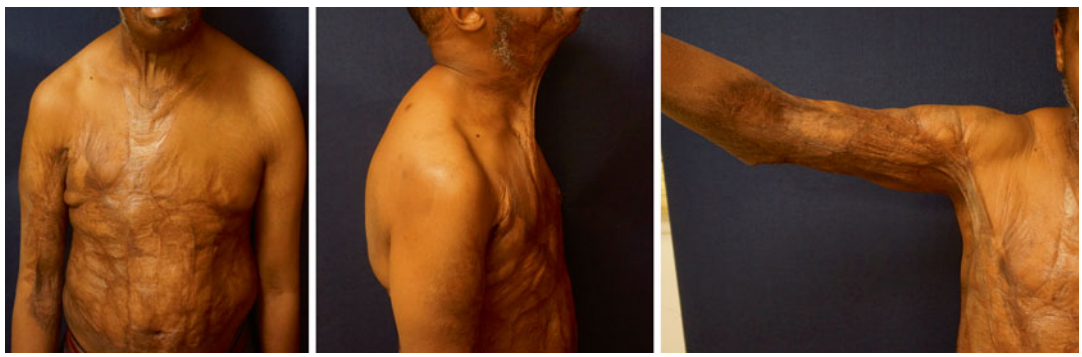
History Full-thickness burns can lead to the formation of scar contractures despite optimal treatment in the immediate and early postburn period with up to 70 % of burn patients requiring surgical intervention for contractures.

Exam Bands or areas of scar formation with resulting limitations on range of motion.

Treatment Treatment initially is aimed at prevention. Full-thickness burns are excised with approximation of healthy tissue or excised and sheet grafted. Areas of flexion are splinted to counter the contractile forces of the healing tissue. Wounds are managed meticulously with topical antimicrobials. Multiple modalities are used to treat immature scars including steroid injections, silicone gels, and pressure therapy.

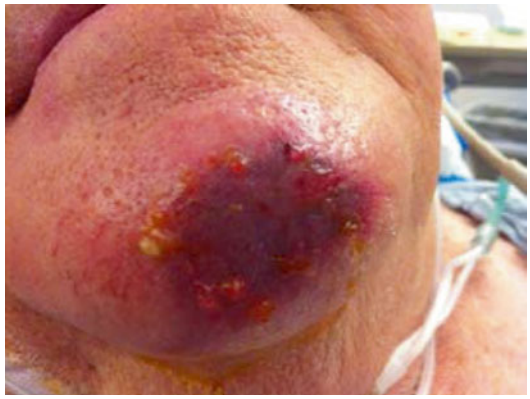
Established contractures can be treated with incision and local tissue rearrangement, excision with reconstruction with grafts or flaps with or without tissue expansion, or laser therapy. Often a combination of these modalities is used.

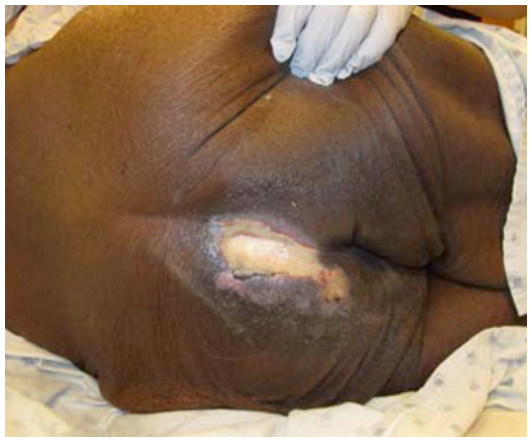
Once the scar has been reset to an acute wound, preventative treatment is again used to help prevent recurrence.

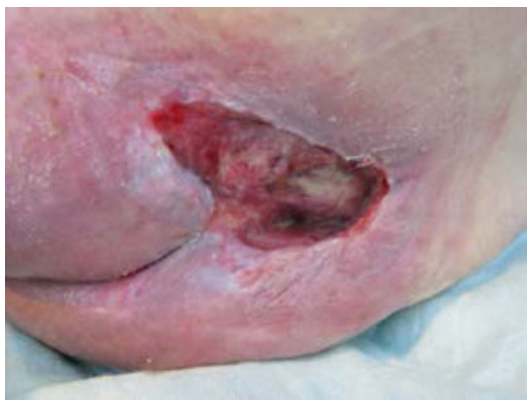


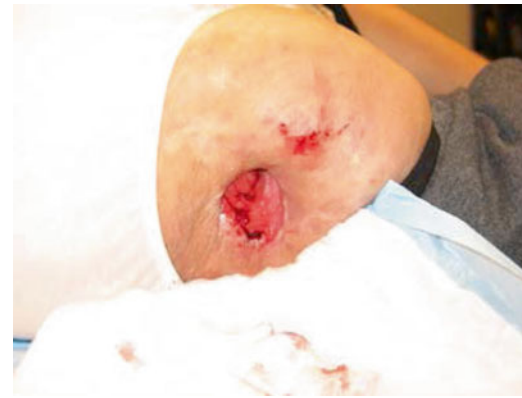
4.4.6 Pressure Sores

Stages 1, 2, 3, and 4









History Pressure sores typically develop in dependent areas of immobilized patients and can occur anywhere. There is tissue ischemia from prolonged pressure (above 32 mmHg). The National Pressure Ulcer Advisory Panel has classified these wounds into four progressive stages. The patient's social situation, comorbidities, and insight into the inciting etiologies are vital components to preoperative planning.

Exam Stage I: Non-blanchable erythema, skin which remains intact, ± tenderness

Stage II: Partial-thickness loss of the dermis, may be either intact blister or shallow ulcer

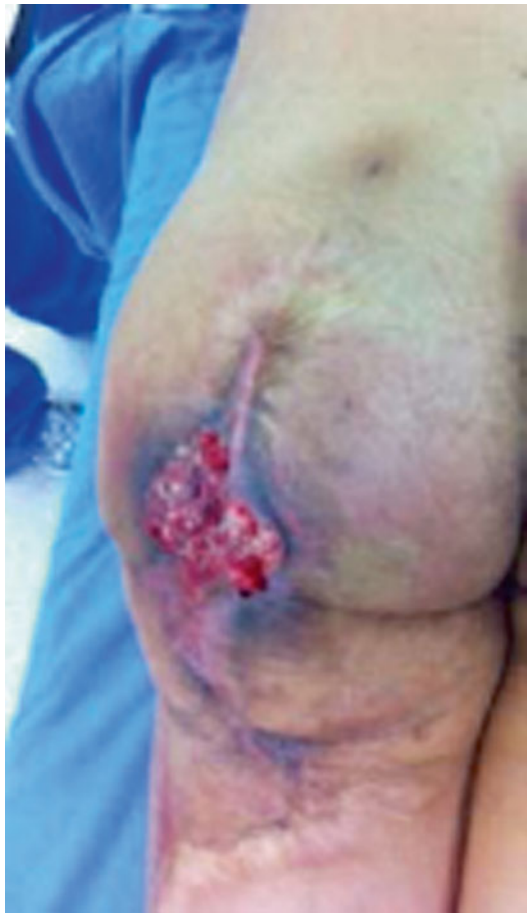
Stage III: Full-thickness dermal loss with ulcer extending into the subcutaneous fat

Stage IV: Full-thickness tissue loss through the subcutaneous fat with exposed muscle, tendon, or bone

Unstageable: Full-thickness skin or tissue loss with depth of wound unknown due to the presence of eschar or slough

Treatment Optimization of the patient's nutritional status and comorbidities (diabetes, osteomyelitis, spasticity, etc.) as well as adequate debridement and proper urinary and fecal diversion are performed prior to reconstructive surgery. Patient education, pressure source alleviation, and the use of air-fluidized mattresses are also ensured. Complete excision of the pressure ulcer and bone debridement should precede myocutaneous or fasciocutaneous flaps for coverage. Flap selection is dependent on the ambulation status of the patient, location of the pressure ulcer, and prior surgical history. Recurrence is common.

4.4.7 Diagnosis: Marjolin's Ulcer

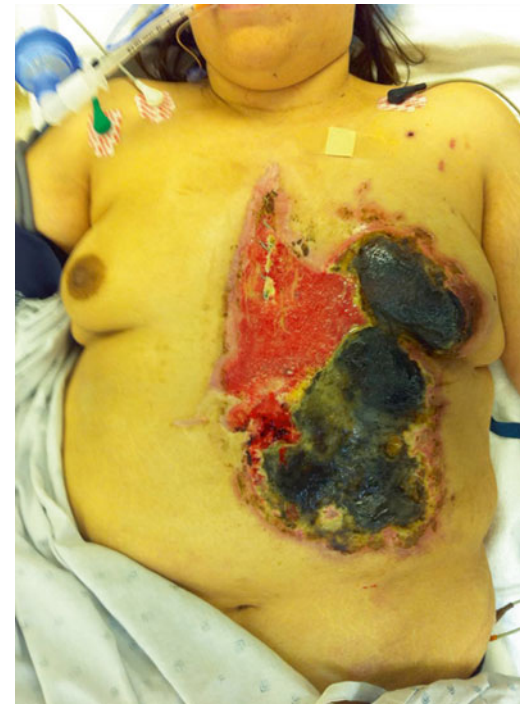


History Marjolin's ulcer is the malignant degeneration of a chronic wound, ulcer, or scar that is often the result of trauma, burn, or radiation injury. It is typically a slow-growing wound that fails conventional therapy; however, fungating, rapidly growing lesions are possible. The majority of these lesions are squamous cell carcinoma, although basal cell carcinoma and melanoma have been described.

Exam Chronically draining ulcer with recent changes in appearance. Lesion can be foul smelling with irregular raised borders and intermittent bleeding. Need a high degree of suspicion.

Treatment Multiple biopsies should be obtained in all suspicious wounds, including central and peripheral zones. Wide local excision (>1 cm margins) is the treatment of choice ± regional lymph node dissection, with radiation therapy alone or adjuvant therapy in select cases.

4.4.8 Calciphylaxis

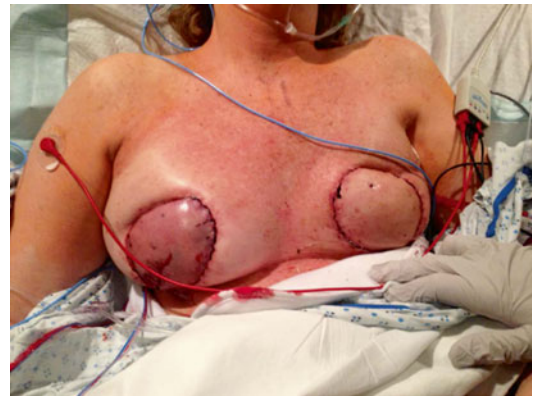


History Calciphylaxis is a rare syndrome of vascular calcification, thrombosis, and skin necrosis seen in 1–4 % of patients with chronic kidney disease (CKD) on hemodialysis. It results in chronic nonhealing wounds and has a 60–80 % mortality rate. It is characterized by small vessel mural calcification with or without endovascular fibrosis, extravascular calcification, and vascular thrombosis, leading to tissue ischemia. Risk factors include hypercalcemia, hyperphosphatemia, female gender, Caucasian race, obesity, diabetes mellitus, warfarin use, hypercoagulability, hypoalbuminemia, albumin infusion, trauma, primary

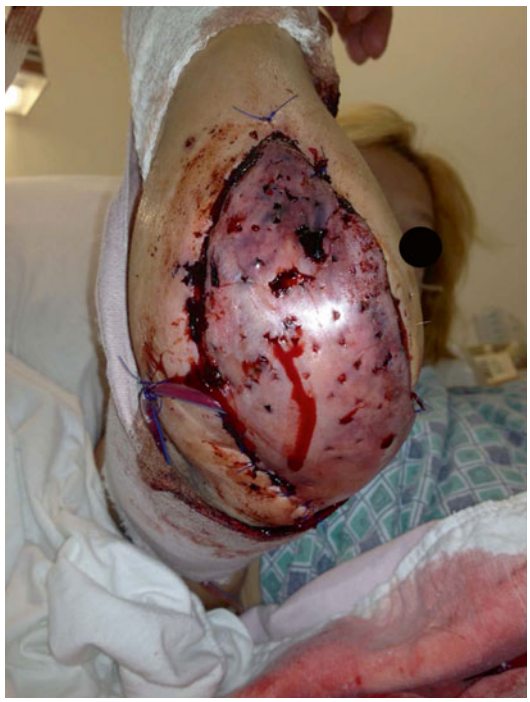
hyperparathyroidism, alcoholic liver disease, malignancy, connective tissue disease, prior corticosteroid use, and protein C or S deficiencies.

Exam The characteristic lesions are the ischemic skin lesions (usually with areas of skin necrosis). The necrotic skin lesions are typically dark bluish purple to black. They can be extensive. The suspected diagnosis can be supported by a skin biopsy.

Treatment The optimal treatment is prevention. Rigorous and continuous control of phosphate and calcium balance most probably will avoid the metabolic changes which may lead to calciphylaxis. After it begins, primary treatment is supportive. One should avoid local tissue trauma (including avoiding all subcutaneous injections and all not-absolutely-necessary infusions and transfusions). Newer treatments include sodium thiosulfate, bisphosphonates, and cinacalcet (used to treat hyperparathyroidism).



4.4.9 Free Flap Compromise



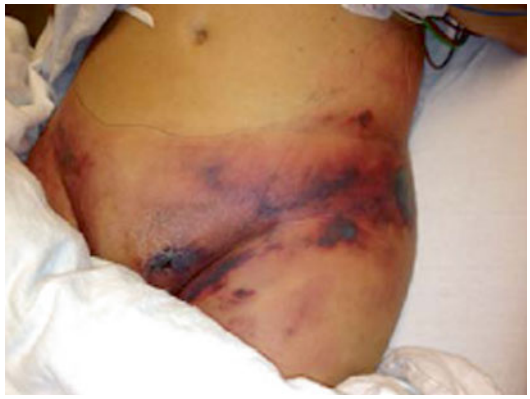
History Free flaps are an integral part of the reconstruction of bony and soft tissue defect when local or regional options do not exist or are inferior. Venous congestion and arterial compromise can, however, threaten flap viability after reconstruction.

Exam Venous congestion is characterised by a flap that is purple, edematous, and hot. Rapid capillary refill, dark venous blood on pinprick, and a boggy feeling from excess edema are typical.

In contrast, arterial compromise is characterised by a cool white flap that lacks turgor. Pinprick demonstrates no bleeding or slow bleeding, and capillary refill will be prolonged. Adjunct methods of flap evaluation include trans-cutaneous and implantable tissue monitoring.

Treatment Bedside treatment for a compromised flap includes releasing the insetting sutures to relieve pressure on the pedicle. Urgent return to the operating room for exploration is indicated.

4.4.10 Necrotizing Fasciitis



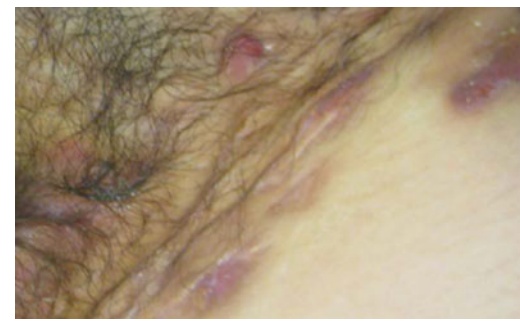
History Necrotizing fasciitis is commonly known as the flesh-eating disease. It is a rare condition that results from a bacterial infection of the subcutaneous tissues of the skin. The infection is common in immunocompromised patients such as diabetic and cancer patients. It has a sudden onset and progresses rapidly. It is classified as:

- Type I (polymicrobial) which represents the majority of cases
- Type II (monomicrobial, group A *Streptococcus* or MRSA) which only represents 15 % of cases

Exam The infection begins at the site of trauma or surgery. Patients complain of severe pain that may seem excessive given the appearance of the skin. Look for signs of inflammation, fever, and tachycardia. Patients are usually very sick and ill looking. Rapid progression occurs with swelling of the tissues and discoloration of the skin and the development of blisters. Look for crepitus and "dish-water" fluid discharge. General symptoms such as diarrhea and vomiting are also common. Mortality rates can be as high as 73 % if left untreated.

Treatment Antibiotics should be started as soon as the condition is suspected. Imperative treatment is started, and cultures are taken to determine the appropriate antibiotic coverage. Aggressive surgical debridement is the only available treatment. Diagnosis is confirmed clinically. Tissue samples can be taken but should not delay surgical debridement. Interdisciplinary care team should be involved depending on the area infected. Fournier gangrene is a subtype of the perineum that usually necessitates involvement of urology and/or gynecology team in the care of the patient.

4.4.11 Hidradenitis Suppurativa



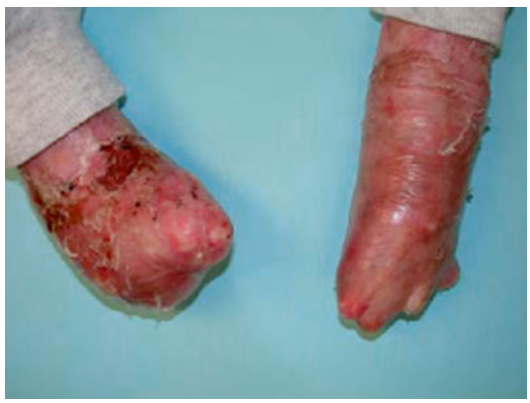
History Hidradenitis is a skin disease that most commonly affects areas bearing apocrine sweat glands or sebaceous glands, such as armpits, under the breasts, inner thighs, groin, and buttocks.

Exam The disease manifests as clusters of chronic abscesses, epidermoid cysts, sebaceous

cysts, or pilonidal cyst. The cysts can be extremely painful to touch and may persist for years with occasional to frequent periods of inflammation, culminating in incision and drainage of pus, often leaving open wounds that will not heal.

Treatment Treatments vary depending on the presentation and severity. Possible treatments include lifestyle modification (diet, weight loss, smoking cessation) and medications including antibiotics (tetracycline, minocycline, rifampin, and clindamycin), intralesional steroids, vitamin A supplements, antiandrogen therapy, infliximab, zinc gluconate, chlorhexidine, and topical resorcinol. Electron beam radiotherapy has been a successful treatment of hidradenitis. When the process becomes chronic, wide surgical excision with skin grafting is the treatment of choice.

4.4.12 Epidermolysis Bullosa

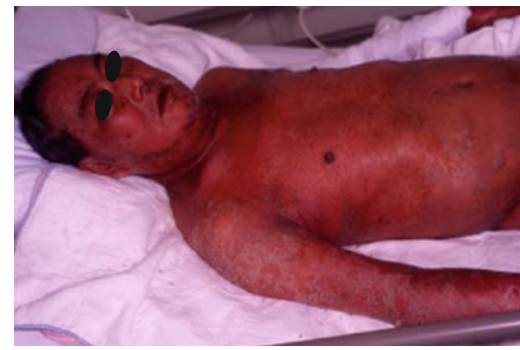


History Epidermolysis bullosa is a mucocutaneous disease resulting from genetic mutations in any of the 14 genes encoding proteins that maintain the integrity of the skin. Most forms of EBS are inherited in an autosomal dominant manner, with mild to no mucosal involvement, and permit a normal lifespan.

Exam Characterized by the formation of blisters in the skin or mucous membranes triggered by minimum trauma or traction (Nikolsky sign). Classified into four major types based on the level of skin cleavage at the ultrastructural level. Skin biopsies from the edge of a fresh blister subjected to immunofluorescent mapping (IFM) are the primary method of diagnosis. Electron microscopy can also be used if IFM is inconclusive.

Treatment There is no cure. Supportive treatments including blister prevention, wound care, nutritional support, monitoring for complications, and psychosocial support are mainstays.

4.4.13 Stevens-Johnson Syndrome (SJS)



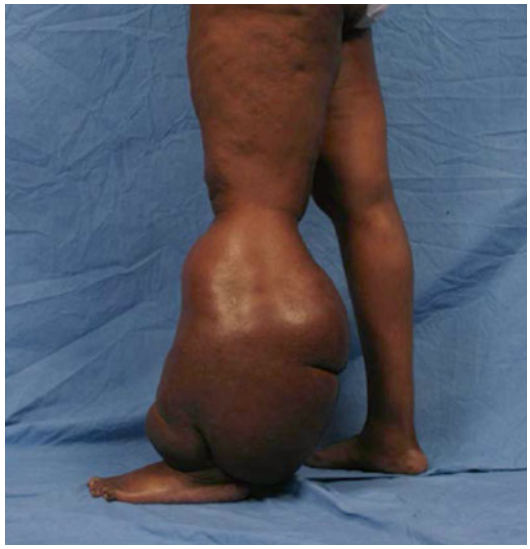
History Incidence is between 1 and 7 cases per million person-years. Drugs are most commonly cited as the cause, but other causes include infection, vaccination, systemic diseases, physical agents, and foods.

Exam Presents as an acute febrile illness with malaise, headaches, cough, and rhinorrhea, which is followed by cutaneous manifestations in a few days. Early sites are the presternal region of the trunk, face, palms, and soles. Skin lesions are erythematous and livid macules, which rapidly coalesce. Mucus membrane involvement is common. There can also be gastrointestinal, respiratory tract, and ocular involvement. In the second stage, there are large areas of epidermal detachment, spontaneously or with tangential mechani-

cal pressure (Nikolsky sign). SJS is differentiated from toxic epidermal necrosis based on the surface area of detachment. Histology shows widespread necrotic epidermis involving all layers.

Treatment Rapid identification and withdrawal of the culprit drugs with rapid initiation of supportive care being the mainstay of treatment. High-dose steroids show good results. IVIG, immunosuppresive agents, and plasmapheresis have also been used with some benefit. SJS has 1 % to 5 % mortality. The risk is determined by the age and extent of skin detachment.

4.4.14 Lymphedema

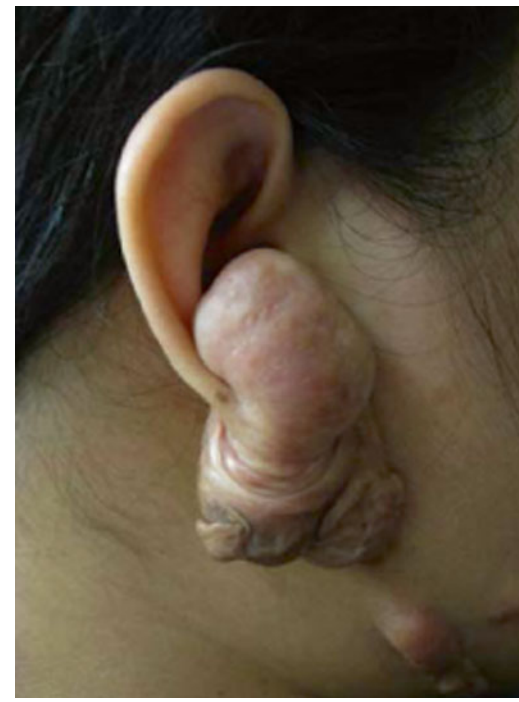


History In the United States, lymphedema can be primary (a rare, X-linked, autosomal) or secondary to surgery, radiation therapy, or neoplasm. In the developing world, lymphedema is often secondary to filariasis. Lymphedema is a progressive pathological condition of the lymphatic system in which there is interstitial accumulation of protein-rich fluid and subsequent inflammation, adipose tissue hypertrophy, and fibrosis.

Exam Unilateral enlargement of the extremity with soft pitting progressing to a more indurated appearance and the development of trophic skin changes in later stages. Patients are prone to repeated bouts of cellulitis and lymphangitis. Peau d'orange changes of the skin and a positive Stemmer sign (the inability to grasp the skin of the dorsum of the second digit of the feet) are diagnostic. Evaluation with CT, MRI, lymphoscintigraphy, and indocyanine green lymphography can be helpful.

Treatment Conservative management includes compression devices, lymph massage, and compression garments that can alleviate the symptoms and control the progression of the disease. Surgical management of lymphedema includes liposuction, excision and grafting (Charles procedure), and free lymph node transfer.

4.4.15 Keloid

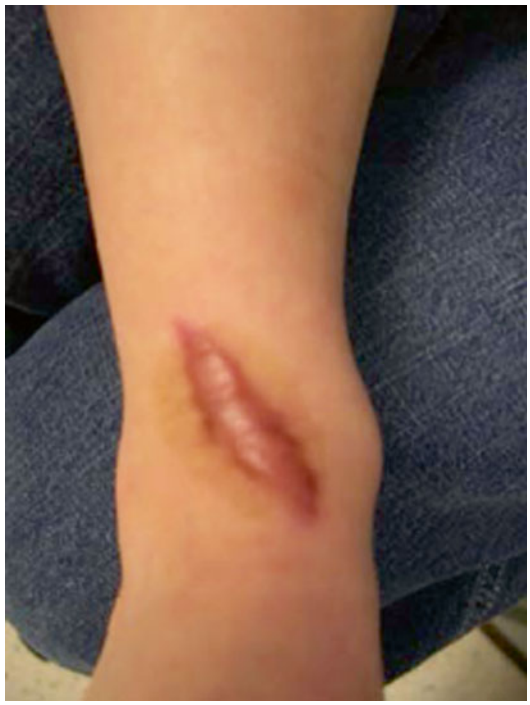


History Keloids represent an exuberant healing response. Patients at high risk of keloids are usually younger than 30 years and have darker skin. Sternal skin, shoulders and upper arms, earlobes, and cheeks are most susceptible to developing keloids. Risk factors include high-risk trauma including burns and ear piercing and any factor that prolongs wound healing.

Exam A scar that grows beyond the boundaries of the original wound. Keloids do not regress over time and usually recur with excision. They are often pruritic and painful.

Treatment Keloid formation often can be prevented if anticipated with immediate silicone elastomer sheeting, taping to reduce skin tension, or corticosteroid injections. Treatment includes surgery, steroids, radiation, lasers, and/or immunotherapy alone, or as part of a multimodal therapy.

4.4.16 Hypertrophic Scar



History An overexuberant healing process that occurs shortly after injury (6–8 weeks after). Often associated with major wounds that are slow to heal because of tension, infection, or foreign body. The scar worsens up to 6 months and then subsides with time. Can be found on flexor surfaces of joints where they can be associated with contractures.

Exam Elevated scars that do not extend outside the original borders of the wound.

They have increased blood vessels and are typically pruritic. Histology demonstrates an increased amount of myofibroblasts with an absence of thick eosinophilic (hyalinizing) collagen bundles.

Treatment Observation, silicone sheeting, intralesional steroids, pressure therapy, and paper tape used alone or in combination.

References

Pyogenic Granuloma

1. Lee J, Sinno H, Tahiri Y, Gilardino MS. Treatment options for cutaneous pyogenic granulomas: a review. J Plast Reconstr Aesthet Surg. 2011;64(9):1216–20.
2. Millsop JW, Trinh N, Winterfield L, Berrios R, Hutchens KA, Tung R. Resolution of recalcitrant pyogenic granuloma with laser, corticosteroid, and timolol therapy. Dermatol Online J. 2014;20(3). http://escholarship.org/uc/item/43q5g62w#page-1

Seborrheic Keratosis

3. Brodsky J. Management of benign skin lesions commonly affecting the face: actinic keratosis, seborrheic keratosis, and rosacea. Curr Opin Otolaryngol Head Neck Surg. 2009;17(4):315–20.
4. Sutton Jr RL, Weakley DR. Seborrheic keratoses. Removal by curettage. Skin (Los Angeles). 1964;3:166–8.

Verrucae (Warts)

5. Dall'oglio F, D'Amico V, Nasca MR, Micali G. Treatment of cutaneous warts: an evidence-based review. Am J Clin Dermatol. 2012;13(2):73–96.
6. Kwok CS, Gibbs S, Bennett C, Holland R, Abbott R. Topical treatments for cutaneous warts. Cochrane Database Syst Rev. 2012;(9):CD001781.
7. Sparreboom EE, Luijks HG, Luiting-Welkenhuyzen HA, Willems PW, Groeneveld CP, Bovenschen HJ. Pulsed dye laser treatment for recalcitrant viral warts: a retrospective case series of 227 patients. Br J Dermatol. 2014;171(5):1270–3.

Neurofibromatosis

8. Farris SR, Grove Jr AS. Orbital and eyelid manifestations of neurofibromatosis: a clinical study and literature review. Ophthal Plast Reconstr Surg. 1996;12(4):245–59.
9. Grabb WC, Dingman RO, Oneal RM, Dempsey PD. Facial hamartomas in children: neurofibroma, lymphangioma, and hemangioma. Plast Reconstr Surg. 1980;66(4):509–27.
10. Korf BR. Diagnosis and management of neurofibromatosis type 1. Curr Neurol Neurosci Rep. 2001;1(2):162–7.
11. Onesti MG, Carella S, Spinelli G, Scuderi N. The megasession technique for excision of multiple neurofibromas. Dermatol Surg. 2010;36(9):1488–90.
12. Singhal D, Chen YC, Fanzio PM, Lin CH, Chuang DC, Chen YR, Chen PK. Role of free flaps in the management of craniofacial neurofibromatosis: soft tissue coverage and attempted facial reanimation. J Oral Maxillofac Surg. 2012;70(12):2916–22.

Acrochordon (Skin Tag)

13. Habif TP. Chapter 20. Benign skin tumors. In: Habif TP, editor. Clinical dermatology. 5th ed. Philadelphia: Mosby Elsevier; 2009.
14. Luba MC, Bangs SA, Mohler AM, Stulberg DL. Common benign skin tumors. Am Fam Physician. 2003;67(4):729–38.
15. Tan O, Atik B, Bayram I. Skin tag. Dermatology. 2005;210(1):82–3. No abstract available.

Lipoma

16. Rao SS, Davison SP. Gone in 30 seconds: a quick and simple technique for subcutaneous lipoma removal. Plast Reconstr Surg. 2012;130(1):236e–8e.

Blue Nevus

17. Phadke PA, Zembowicz A. Blue nevi and related tumors. Clin Lab Med. 2011;31(2):345–58.
18. Rhodes HA. Benign neoplasias and hyperplasias of melanocytes. In: Freedberg IM, Eisen AZ, Wolff K, et al., editors. Fitzpatrick's dermatology in general medicine. 5th ed. New York: McGraw-Hill; 1999. p. 1037–43.

Spitz Nevus

19. Gelbart SN, Tripp JM, Marghoob AA, et al. Management of Spitz nevi: a survey of dermatologists in the United States. J Am Acad Dermatol. 2002;47(2):224–30.
20. Lyon VB. The spitz nevus: review and update. Clin Plast Surg. 2010;37(1):21–33.
21. Murphy ME, Boyer JD, Stashhower ME, Zitelli JA. The surgical management of Spitz nevi. Dermatol Surg. 2002;28(11):1065–9.
22. Sepehr A, Chao E, Trefrey B, et al. Long-term outcome of Spitz-type melanocytic tumors. Arch Dermatol. 2011;147(10):1173–9.

Mongolian Spot

23. Cordova A. The Mongolian spot: a study of ethnic differences and a literature review. Clin Pediatr (Phila). 1981;20(11):714–9.
24. Mosher DB, Fitzpatrick TB, Yoshiaki H, et al. Disorders of pigmentation. In: Fitzpatrick TB, editor. Dermatology in general medicine, vol. 1. New York: McGraw-Hill; 1993. p. 903–95.

Nevus of Ota

25. Chan HH, Kono T. Nevus of Ota: clinical aspects and management. Skinmed. 2003;2(2):89–96. quiz 97–8. Review.
26. Sinha S, Cohen PJ, Schwartz RA. Nevus of Ota in children. Cutis. 2008;82(1):25–9. Review.

Giant Nevus

27. Egan CL, Oliveria SA, Elenitsas R, Hanson J, Halpern AC. Cutaneous melanoma risk and phenotypic changes in large congenital nevi: a follow-up study of 46 patients. J Am Acad Dermatol. 1998;39(6):923–32.

28. Shah KN. The risk of melanoma and neurocutaneous melanosis associated with congenital melanocytic nevi. Semin Cutan Med Surg. 2010;29(3):159–64. doi:10.1016/j.sder.2010.06.007.
29. Tannous ZS, Mihm Jr MC, Sober AJ, Duncan LM. Congenital melanocytic nevi: clinical and histopathologic features, risk of melanoma, and clinical management. J Am Acad Dermatol. 2005;52(2):197–203.
30. Watt AJ, Kotsis SV, Chung KC. Risk of melanoma arising in large congenital melanocytic nevi: a systematic review. Plast Reconstr Surg. 2004;113(7):1968–74.

Actinic Keratosis

31. Gupta AK, Paquet M, Villanueva E, Brintnell W. Interventions for actinic keratoses. Cochrane Database Syst Rev. 2012;(12):CD004415.
32. Ulrich C, Arnold R, Frei U, Hetzer R, Neuhaus P, Stockfleth E. Skin changes following organ transplantation: an interdisciplinary challenge. Dtsch Arztebl Int. 2014;111(11):188–94.

Keratoacanthom

33. Mandrell JC, Santa Cruz D. Keratoacanthoma: hyperplasia, benign neoplasm, or a type of squamous cell carcinoma? Semin Diagn Pathol. 2009;26(3):150–63.
34. Savage JA, Maize Sr JC. Keratoacanthoma clinical behavior: a systematic review. Am J Dermatopathol. 2014;36(5):422–9.

Bowen's Disease

35. Neubert T, Lehmann P. Bowen's disease – a review of newer treatment options. Ther Clin Risk Manag. 2008;4(5):1085–95.
36. Weyers W. The centennial of Bowen's disease-a critical review on the occasion of the 100th anniversary of its original description. Dermatol Pract Concept. 2012;2(4):204a02.

Erythroplasia of Queyrat

37. Bleeker MC, Heideman DA, Snijders PJ, Horenblas S, Dillner J, Meijer CJ. Penile cancer: epidemiology, pathogenesis and prevention. World J Urol. 2009;27(2):141–50.
38. Goette DK. Review of erythroplasia of Queyrat and its treatment. Urology. 1976;8(4):311–5.

Basal Cell Carcinoma (BCC)

39. Bath-Hextall FJ, Perkins W, Bong J, Williams HC. Interventions for basal cell carcinoma of the skin. Cochrane Database Syst Rev. 2007;(1):CD003412.
40. Clark CM, Furniss M, Mackay-Wiggan JM. Basal Cell Carcinoma: an evidence-based treatment update. Am J Clin Dermatol. 2014;15(3):197–216.
41. Telfer NR, Colver GB, Morton CA; British Association of Dermatologists. Guidelines for the management of basal cell carcinoma. Br J Dermatol. 2008;159(1):35–48.

Squamous Cell Carcinoma (SCC)

42. Kolk A, Wolff KD, Smeets R, Kesting M, Hein R, Eckert AW. Melanotic and non-melanotic malignancies of the face and external ear – a review of current treatment concepts and future options. Cancer Treat Rev. 2014;40(7):819–37.

Melanoma

43. Gorantla VC, Kirkwood JM. State of Melanoma: an historic overview of a field in transition. Hematol Oncol Clin North Am. 2014;28(3):415–35.
44. Sondak VK, Gibney GT. Surgical management of melanoma. Hematol Oncol Clin North Am. 2014;28(3):455–70.

Subungual Melanoma

45. Cohen T, Busam KJ, Patel A, Brady MS. Subungual melanoma: management considerations. Am J Surg. 2008;195(2):244–8.
46. Nguyen JT, Bakri K, Nguyen EC, Johnson CH, Moran SL. Surgical management of subungual melanoma: mayo clinic experience of 124 cases. Ann Plast Surg. 2013;71(4):346–54.

Lentigo Maligna

47. McKenna JK, Florell SR, Goldman GD, Bowen GM. Lentigo maligna/lentigo maligna melanoma: current state of diagnosis and treatment. Dermatol Surg. 2006;32(4):493–504.

DFSP (Dermatofibrosarcoma Protuberans)

48. Fiore M, Miceli R, Mussi C, Lo Vullo S, Mariani L, Lozza L, Collini P, Olmi P, Casali PG, Gronchi A. Dermatofibrosarcoma protuberans treated at a single institution: a surgical disease with a high cure rate. J Clin Oncol. 2005;23(30):7669–75.
49. Hancox JG, Kelley B, Greenway Jr HT. Treatment of dermatofibroma sarcoma protuberans using modified Mohs micrographic surgery: no recurrences and smaller defects. Dermatol Surg. 2008;34(6):780–4.

Angiosarcoma

50. Mendenhall WM, Mendenhall CM, Werning JW, Reith JD, Mendenhall NP. Cutaneous angiosarcoma. Am J Clin Oncol. 2006;29(5):524–8.
51. Perez MC, Padhya TA, Messina JL, Jackson RS, Gonzalez RJ, Bui MM, Letson GD, Cruse CW, Lavey RS, Cheong D, Forster MR, Fulp WJ, Sondak VK, Zager JS. Cutaneous angiosarcoma: a single-institution experience. Ann Surg Oncol. 2013;20(11):3391–7.

Myelomeningocele

52. Kobraei EM, Ricci JA, Vasconez HC, Rinker BD. A comparison of techniques for myelomeningocele defect closure in the neonatal period. Childs Nerv Syst. 2014;30(9):1535–41.
53. de Chalain TM, Cohen SR, Burstein FD, Hudgins RJ, Boydston WR, O'Brien MS. Decision making in primary surgical repair of myelomeningoceles. Ann Plast Surg. 1995;35(3):272–8.
54. Ozçelik D, Yildiz KH, Iş M, Döşoğlu M. Soft tissue closure and plastic surgical aspects of large dorsal myelomeningocele defects (review of techniques). Neurosurg Rev. 2005;28(3):218–25. Epub 2004 Nov 16.

Xeroderma Pigmentosa

55. Butt FM, Moshi JR, Owibingire S, Chindia ML. Xeroderma pigmentosum: a review and case series. J Craniomaxillofac Surg. 2010;38(7):534–7.
56. Feller L, Wood NH, Motswaledi MH, Khammissa RA, Meyer M, Lemmer J. Xeroderma pigmentosum: a case report and review of the literature. J Prev Med Hyg. 2010;51(2):87–91.

Hemangioma

57. Greene AK. Management of hemangiomas and other vascular tumors. Clin Plast Surg. 2011;38(1):45–63.
58. Izadpanah A, Izadpanah A, Kanevsky J, Belzile E, Schwarz K. Propranolol versus corticosteroids in the treatment of infantile hemangioma: a systematic review and meta-analysis. Plast Reconstr Surg. 2013;131(3):601–13.
59. Holland KE, Drolet BA. Infantile hemangioma. Pediatr Clin North Am. 2010;57(5):1069–83.

Capillary Malformation

60. Maguiness SM, Liang MG. Management of capillary malformations. Clin Plast Surg. 2011;38(1):65–73. doi:10.1016/j.cps.2010.08.010.
61. Colletti G, Valassina D, Bertossi D, Melchiorre F, Vercellio G, Brusati R. Contemporary management of vascular malformations. J Oral Maxillofac Surg. 2014;72(3):510–28.

Arteriovenous Malformation (Parkes Weber Syndrome)

62. Greene AK, Orbach DB. Management of arteriovenous malformations. Clin Plast Surg. 2011;38(1):95–106.
63. Colletti G, Valassina D, Bertossi D, Melchiorre F, Vercellio G, Brusati R. Contemporary management of vascular malformations. J Oral Maxillofac Surg. 2014;72(3):510–28.

Venous (Klippel-Trenaunay Syndrome)

64. Greene AK, Alomari AI. Management of venous malformations. Clin Plast Surg. 2011;38(1):83–93.
65. Colletti G, Valassina D, Bertossi D, Melchiorre F, Vercellio G, Brusati R. Contemporary management of vascular malformations. J Oral Maxillofac Surg. 2014;72(3):510–28.

Lymphatic Malformation

66. Greene AK, Perlyn CA, Alomari AI. Management of lymphatic malformations. Clin Plast Surg. 2011;38(1):75–82.
67. Colletti G, Valassina D, Bertossi D, Melchiorre F, Vercellio G, Brusati R. Contemporary management of

vascular malformations. J Oral Maxillofac Surg. 2014;72(3):510–28.

First Degree: Superficial

68. Garner WL, Magee W. Acute burn injury. Clin Plast Surg. 2005;32:187–93.
69. Kao CC, Garner WL. Acute burns. Plast Reconstr Surg. 2000;101(7):2482–92.

Second Degree: Partial Thickness

70. Garner WL, Magee W. Acute burn injury. Clin Plast Surg. 2005;32:187–93.
71. Kao CC, Garner WL. Acute burns. Plast Reconstr Surg. 2000;101(7):2482–92.

Third Degree: Full Thickness

72. Garner WL, Magee W. Acute burn injury. Clin Plast Surg. 2005;32:187–93.
73. Kao CC, Garner WL. Acute burns. Plast Reconstr Surg. 2000;101(7):2482–92.

Fourth Degree

74. Garner WL, Magee W. Acute burn injury. Clin Plast Surg. 2005;32:187–93.
75. Kao CC, Garner WL. Acute burns. Plast Reconstr Surg. 2000;101(7):2482–92.

Burn Contracture

76. Barbour JR, Schweppe M, O SJ. Lower-extremity burn reconstruction in the child. J Craniofac Surg. 2008;19(4):976–88.
77. Grevious MA, Paulius K, Gottlieb LJ. Burn scar contractures of the pediatric neck. J Craniofac Surg. 2008;19(4):1010–5.

Pressure Sore

78. National Pressure Ulcer Advisory Panel. www.npuap.org
79. Tchanque-Fossuo CN, Kuzon Jr WM. An evidence-based approach to pressure sores. Plast Reconstr Surg. 2011;127(2):932–9.

Diagnosis: Marjolin's Ulcer

80. Copcu E. Marjolin's ulcer: a preventable complication of burns? Plast Reconstr Surg. 2009l;124(1):156e–64e.
81. Mustoe T, Upton J, Marcellino V, et al. Carcinoma in chronic pressure sores: a fulminant disease process. Plast Reconstr Surg. 1986;77(1):116–21.

Calciphylaxis

82. Hayden MR, Goldsmith D, Sowers JR, Khanna R. Calciphylaxis: calcific uremic arteriolopathy and the emerging role of sodium thiosulfate. Int Urol Nephrol. 2008;40(2):443–51.
83. Sood AR, Wazny LD, Raymond CB, Leung K, Komenda P, Reslerova M, Verrelli M, Rigatto C, Sood MM. Sodium thiosulfate-based treatment in calcific uremic arteriolopathy: a consecutive case series. Clin Nephrol. 2011;75(1):8–15.
84. Vedvyas C, Winterfield LS, Vleugels RA. Calciphylaxis: a systematic review of existing and emerging therapies. J Am Acad Dermatol. 2012;67(6):e253–60.

Free Flap Compromise

85. Chang EI, Chang EI, Soto-Miranda MA, Zhang H, Nosrati N, Crosby MA, Reece GP, Robb GL, Chang DW. Comprehensive evaluation of risk factors and management of impending flap loss in 2138 breast free flaps. Ann Plast Surg. 2014. [Epub ahead of print].
86. Roehl KR, Mahabir RC. A practical guide to free tissue transfer. Plast Reconstr Surg. 2013;132(1):147e–58e.
87. Smit JM, Zeebregts CJ, Acosta R, Werker PM. Advancements in free flap monitoring in the last decade: a critical review. Plast Reconstr Surg. 2010;125(1):177–85. doi:10.1097/PRS.0b013e3181c49580. Review. PubMed PMID: 20048610.
88. Thoma A, Jansen L, Sprague S. Outcomes in microsurgery. Plast Reconstr Surg. 2009;124(6 Suppl):e303–12.

Necrotizing Fasciitis

89. Chernyshev O, Shatil M, Akinchits L, Vedernykova N, Kotlov V, Semak M, Bubnova N. Necrotizing fasciitis: modern clinical view. Crit Care. 2012;16(Suppl 3):P60. doi: 10.1186/cc11747. Published online 2012 November 14.
90. Kao LS, Lew DF, Arab SN, Todd SR, Awad SS, Carrick MM, Corneille MG, Lally KP. Local varia-

tions in the epidemiology, microbiology, and outcome of necrotizing soft tissue infections: a multi-center study. Am J Surg. 2011;202(2):139–45. Author manuscript; available in PMC 2012 August 1.

Hidradenitis Suppurativa

91. Alikhan A, Lynch E Hidradenitis suppurativa: a comprehensive review. J Am Acad Dermatol. 2009;60(4):539–61.doi:10.1016/j.jaad.2008.11.911. DOI:10.1016%2Fj.jaad.2008.11.911. PMID 19293006.
92. Slade DEM, Powell BW, Mortimer PS. Hidradenitis suppurativa: pathogenesis and management. Br J Plast Surg. 2003;56(5):451–61. doi:10.1016/S0007-1226(03)00177-2. DOI:10.1016%2FS0007-1226%2803%2900177-2. PMID 12890458.

Epidermolysis Bullosa

93. Elluru RG, Contreras JM, Albert DM. Management of manifestations of epidermolysis bullosa. Curr Opin Otolaryngol Head Neck Surg. 2013;21(6):588–93.
94. Fine JD, Bruckner -Tuderman L, Eady RA, et al. Inherited epidermolysis bullosa. Updated recommendations on diagnosis and classification. J Am Acad Dermatol 2014;70(6):1103–26.

Stevens-Johnson Syndrome (SJS)

95. Harr T, French LE. Toxic epidermal necrolysis and Stevens-Johnson syndrome. Orphanet J Rare Dis. 2010;5:39.
96. Letko E, Papaliodis DN, Papaliodis GN, et al. Stevens-Johnson syndrome and toxic epidermal necrolysis: a review of the literature. Ann Allergy Asthma Immunol. 2005;95:419–36.
97. Stevens AM, Johnson FC. A new eruptive fever associated with stomatitis and ophthalmia: report of 2 cases in children. Am J Dis Child. 1922;24:526–33.

Lymphedema

98. Penha TR, Ijsbrandy C, Hendrix NA, Heuts EM, Voogd AC, von Meyenfeldt MF, van der Hulst RR. Microsurgical techniques for the treatment of breast cancer-related lymphedema: a systematic review. J Reconstr Microsurg. 2013;29(2):99–106.
99. Schook CC, Mulliken JB, Fishman SJ, Grant FD, Zurakowski D, Greene AK. Primary lymphedema: clinical features and management in 138 pediatric patients. Plast Reconstr Surg. 2011;127(6):2419–31.
100. Warren AG, Brorson H, Borud LJ, Slavin SA. Lymphedema: a comprehensive review. Ann Plast Surg. 2007;59(4):464–72.

Keloid

101. Ogawa R. The most current algorithms for the treatment and prevention of hypertrophic scars and keloids Plast Reconstr Surg. 2010;125(2):557–68. Juckett G, Hartman-Adams H. Management of keloids and hypertrophic scars. Am Fam Physician. 2009;80(3):253–60.

Hypertrophic Scar

102. Friedstat JS, Hultman CS. Hypertrophic burn scar management: what does the evidence? A systematic review of randomized controlled trials. Ann Plast Surg. 2014;72 Suppl 2:S198–201.
103. Ogawa R. The most current algorithms for the treatment and prevention of hypertrophic scars and keloids. Plast Reconstr Surg. 2010;125(2):557–68.