

CHAPTER 9

PEDIATRIC PLASTIC SURGERY

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Children are not just small adults. Conditions, treatment principles, and concerns for certain complications are different and must be considered. The most common pediatric plastic surgery problems comprise congenital craniofacial anomalies, acquired craniofacial problems (such as facial fractures), brachial plexus injuries, hand anomalies, congenital nevi, and vascular malformations. Cleft lip and palate are discussed in a separate chapter.

I. CONGENITAL CRANIOFACIAL ANOMALIES

A. Craniofacial Embryology and Development:

1. Facial embryology
 - a. Facial development occurs between 3-8 weeks of development
 - b. Face originates from 5 prominences that appear during 4th week (Figure 1)
 - i. Central frontonasal prominence, arising from mesenchyme ventral to forebrain
 - ii. Paired maxillary prominences
 - iii. Paired mandibular prominences
 - c. Paired maxillary and mandibular prominences both arise from neural crest cells migrating from 1st branchial arch
 - d. These prominences surround the primitive mouth (stomodeum)
 - e. Prominences give rise to
 - i. Frontonasal prominence, which divides into:
 - (a) Medial nasal process: nasal tip, columella, philtrum and premaxilla
 - (b) Lateral nasal process: nasal alae
 - ii. Maxillary prominences: upper jaw, upper lip (lateral to philtrum), orbital floor, inferior portion of lateral nasal wall
 - iii. Mandibular prominence: lower jaw
 - f. Between 5-6 weeks nasal processes enlarge, migrate and coalesce in midline to unite with maxillary process and form upper lip.

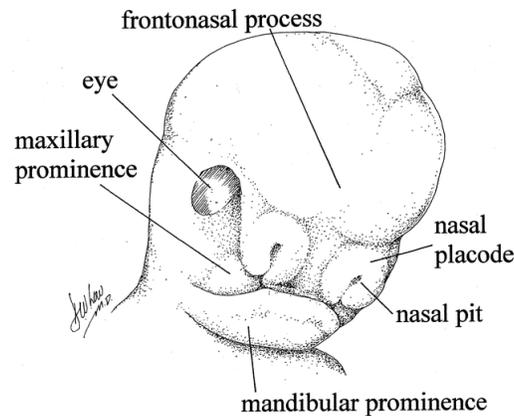


Figure 1. Craniofacial embryology. The five facial prominences.
 From Losee, JE, Kirschner, RE, Whitaker, LA, Bartlett, SP. *Congenital nasal anomalies: a classification scheme. Plast Reconstr Surg* 2004;113:676-689.

2. Cranial development
 - a. Cranium divided into
 - i. Neurocranium: structures that surround the brain
 - ii. Viscerocranium: structures that surround oral cavity, pharynx, upper respiratory system and face
 - b. Ossification
 - i. Cranial base (occipital, sphenoid and temporal bones): endochondral ossification
 - ii. Cranial vault: intramembranous ossification
 - c. Cranial growth responds to increased brain volume
 - i. Brain size triples by 1 year
 - ii. Brain size quadruples by 2 years
 - iii. Cranial vault is 90% of adult size by age 5
 - iv. Brain has reached 95% of adult size by age 8-10
 - d. Normal cranial growth occurs through
 - i. Suture growth: perpendicular to suture
 - ii. Appositional growth: bone resorption of the inner surface and bone deposition on the outer surface
- B. Craniosynostosis (CS)
 1. Definition: Premature fusion of one or more cranial vault sutures.
 2. Incidence: 1:2,500 live births
 3. Normal suture and fontanelle closure
 - a. Suture fusion:
 - iii. Metopic: 6-8 months
 - i. Sagittal: 22 years
 - ii. Coronal: 24 years
 - iii. Lambdoid: 26 years
 - b. Fontanelle closure:
 - i. Posterior: 3-6 months
 - ii. Anterior: 9-12 months

4. Characteristic head shape according to suture affected (Figure 2)
 - a. Sagittal: scaphocephaly (Gr., *scapho*, meaning boat-shaped)
 - b. Metopic: trigonocephaly (Gr., *trigono*, meaning triangular or keel-shaped)
 - c. Coronal, bilateral: brachycephaly (Gr., *brachy*, meaning short in AP direction)
 - d. Coronal, unilateral: plagiocephaly (Gr., *plagios*, meaning oblique or slanted.
 - e. Important: distinguish from *positional* plagiocephaly, where suture is normal. Head deformity is similar, but there is a parallelogram configuration of the head (if seen from above) with anterior displacement of the ipsilateral ear and occipital flattening.

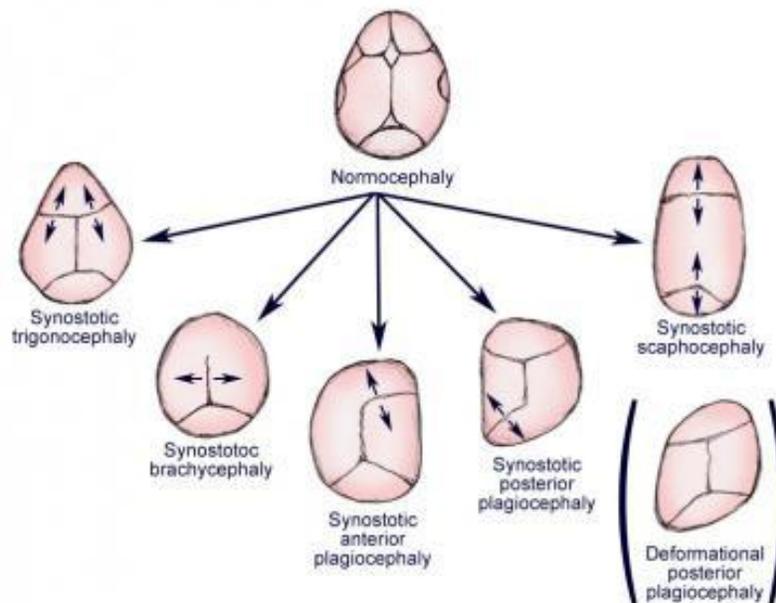


Figure 2. Craniosynostosis and cranial deformities depending on suture fused

5. Categorized into non-syndromic and syndromic types
6. Non-syndromic CS (most common, 67-80%)
 - a. Order of frequency according to suture type
 - i. Sagittal (40-50%)
 - ii. Metopic (25%)
 - iii. Coronal (5-10% bilateral; 15-20% unilateral)
 - iv. Lambdoid (<3%)
 - b. Longstanding debate as to whether non-syndromic patients have increased incidence of developmental delay. New evidence shows some degree of executive dysfunction in up to 50% of these children.
 - c. Treatment indications:
 - i. Prevent potential increased intracranial pressure (ICP)
 - ii. Correct the cranial deformity and normalize appearance
 - e. Treatment:
 - i. Vary depending on suture affected and severity

- ii. Usually performed within first year of life to take advantage of molding capacity of skull
 - iii. Minimally invasive procedures: extended strip suturectomy (<6 months) +/- springs, +/- postoperative helmet therapy.
 - iv. Anterior vault reshaping (fronto-orbital advancement (FOA)/reshaping)
 - v. Total vault reshaping
 - vi. Posterior vault reshaping
7. Syndromic CS
- a. Higher incidence of ICP than non-syndromic
 - b. Major associated syndromes:
 - i. Apert: CS, exorbitism, severe midfacial retrusion, complex syndactyly of the 2-4 digits of the hands/feet.
 - ii. Crouzon: CS, exorbitism, midfacial retrusion, no limb anomalies.
 - iii. Pfeiffer: CS, exorbitism, midfacial retrusion, broad thumbs and toes)
 - c. Characteristic head shape involves turribrachycephaly (Gr., *turri*, tower)
 - d. 50% of Apert syndrome patients have substantial mental delay; Crouzon and Pfeiffer syndrome patients usually have normal mental development.
 - e. Genetic defect identified in fibroblast growth factor receptor (FGFR) genes (Apert, Crouzon---FGFR2, Pfeiffer—FGFR1)
 - f. Goals of surgery
 - i. Expand intracranial volume (83% ICP in Apert, less in others)
 - ii. Normalize head shape and appearance
 - iii. Correct profound exorbitism to prevent corneal exposure/blindness
 - iv. Correct malocclusion
 - g. General timeline of surgical interventions: may vary
 - i. Suturectomy for decompression of elevated ICP, if present (<3 months)
 - ii. Posterior cranial vault distraction/remodeling (6-12 months)
 - iii. Anterior/total vault reshaping/fronto-orbital advancement (FOA) (4-12 months)
 - iv. Midface procedures: Le Fort III or monobloc advancement (4-12 years).
 - v. Orthognathic surgery/canthopexies/other revisions. (12 years - adulthood)
 - vi. In general, craniofacial distraction leads to greater advancement, less relapse than conventional procedures.
- C. Craniofacial Clefts
1. Lack of fusion of facial processes that results in abnormal separation of skeletal and soft tissue structures of the face and cranium (alternative theory of lack of mesodermal penetration)
 2. Rare. Estimated 1.4 – 5.1 per 100,000 births
 3. Defined by Paul Tessier, who classified them
 4. Tessier Classification system relates soft tissue to skeletal landmarks (Figure 3)
 5. Any combination of clefts is possible
 6. Facial dysostoses are associated with certain clefts:
 - a. Craniofacial/hemifacial microsomia: #7 Cleft (most common facial cleft)
 - b. Treacher Collins: Clefts # 6, 7, 8

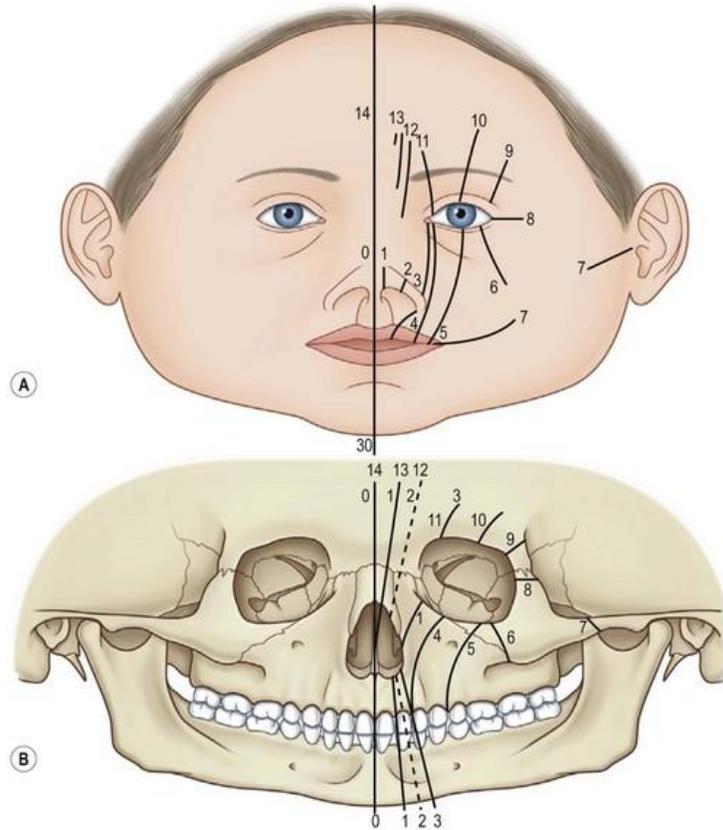


Figure 3. Tessier Classification of Orofacial Clefts. Soft tissue clefts (above) and bony clefts (below).

D. Facial Dysostoses

1. Treacher Collins Syndrome (Mandibulofacial Dysostosis)

- a. Rare, autosomal dominant, variable penetrance disorder
- b. Affected gene on chromosome 5q
- c. 1st branchial arch, groove and pouch affected
- d. Defined by the bilateral presence of three Tessier Clefts #6, 7, 8, which result in all the phenotypic manifestations
- e. Clinical manifestations:
 - i. Hypoplasia/aplasia of the zygomatic arch
 - (a) Lateral orbit deficiency
 - (b) Midface retrusion
 - (c) Lateral canthus hypoplasia/downward slanting palpebral fissures
 - ii. Hypoplasia of temporalis muscle
 - iii. Coloboma and retraction of lower lid
 - iv. Variable ear malformations (microtia/antia) and deafness
 - v. Mandibular hypoplasia with microretrognathia
 - (a) Airway compromise due to narrow pharyngeal diameter
 - (b) Require tracheostomy and distraction of mandible

- vi. Parrot-beak nose +/- choanal atresia
- vii. Normal intelligence
- f. Treatment
 - i. Skeletal and soft tissue augmentation of deficient areas with autologous bone grafts (calvaria, rib, iliac crest) and autologous fat/tissue transfer, respectively.
 - ii. Mandibular distraction may be necessary for achieving a stable airway
- 2. Craniofacial Microsomia (a.k.a. Hemifacial Microsomia)
 - a. Spectrum of morphogenetic abnormalities affecting the cranial skeleton, soft tissues and neuromuscular structures derived from the 1st and 2nd branchial arches
 - b. Theory is that problems derive from hematoma or thrombosis of stapedial artery
 - c. Third-most common congenital malformation (following club foot and cleft lip and palate)
 - d. 3:2 male>female
 - e. 1:3500-5600 live births affected
 - f. Usually unilateral (therefore the more common name hemifacial microsomia)
 - g. Manifestations
 - i. Hypoplasia of mandibular ramus (uni or bilateral)
 - (a) +/- hypoplasia of the maxilla, zygoma and temporal bone
 - (b) Deviated chin
 - (c) Tilted occlusal plane – malocclusion
 - ii. Microtia +/- hearing loss
 - iii. Paresis of CN V or CN VII common
 - h. Associated with Tessier #7 facial cleft
 - i. Pruzansky classification:
 - i. I: Small ramus with identifiable anatomy
 - ii. II: A functioning TMJ but with abnormal shape and glenoid fossa (IIA glenoid fossa is in acceptable functional position; IIB TMJ is abnormally placed)
 - iii. III: Absent ramus and non-existent glenoid fossa
 - j. Treatment
 - i. Augment deficient areas
 - (a) Skeletal: autologous bone (calvarium, rib, iliac crest)
 - (b) Soft tissue: free flap and/or fat grafting
 - ii. Mandibular correction depends upon severity of hypoplasia. Distraction may be necessary to achieve correction of malocclusion versus conventional orthognathic procedures to correct jaw discrepancies in adolescence.
 - iii. Bone-anchored hearing aids
- 3. Goldenhar Syndrome
 - a. Variant of craniofacial/hemifacial microsomia with additional manifestations
 - b. Only 5% of hemifacial microsomia cases have these features
 - c. Sporadic occurrence

- d. Also known as oculo-auriculo-vertebral (OAV) spectrum
- e. Manifestations
 - i. Prominent frontal bossing, low hairline
 - ii. Low set ears
 - iii. Anterior accessory auricular appendages
 - iv. Colobomas of upper eyelid
 - v. Hemifacial microsomia
 - vi. Vertebral spine abnormalities
 - vii. Abnormalities of heart, kidneys, lungs
- E. Pierre Robin Sequence
 1. 1:8,500-20,000 live births
 2. Triad of
 - a. Micrognathia
 - b. Glossoptosis (tongue retracted back into the pharynx)
 - c. Upper airway obstruction
 3. Not a syndrome per se, but a sequence:
 - a. Intrinsic/extrinsic disturbance of mandible development (leads to micrognathia)
 - b. Arrest of rotation/descent of tongue from between palatal shelves
 - c. Lack of palatal fusion or high, U-shaped palate
 - d. Tongue remains pressed against posterior pharynx = airway obstruction and feeding issues
 4. May be isolated or associated with certain craniofacial syndromes
 - a. Stickler
 - b. Nager
 - c. Treacher-Collins
 5. Immediate treatment goals in the neonate are establishing a patent airway and improving feeding. Four treatment modalities available
 - a. Conservative treatment: if newborn demonstrates adequate feeding and weight gain. Mild airway obstruction relieved by change in positioning.
 - b. Tongue-lip adhesion: old technique; temporary suturing of tongue to lower lip to address glossoptosis.
 - c. Tracheostomy: immediate airway control in emergent scenario, but significant morbidity. Currently used as last resort.
 - d. Mandibular distraction: newest procedure preferred by most centers where available. Addresses mandibular hypoplasia; tongue moves anteriorly and relieves airway obstruction.
 6. Requirements
 - a. Continuous pulse oximetry and airway monitoring
 - b. Multidisciplinary evaluation by genetics, ophthalmology, PRS, feeding specialist
 - c. Laryngoscopy/bronchoscopy
 - d. Polysomnogram (sleep apnea)
- F. Other Embryologic Defects
 4. Branchial cyst, sinus, or fistula

- a. Epithelial-lined tract frequently in the lateral neck presenting along the anterior border of the sternocleidomastoid muscle.
- b. May present as a cyst or as a sinus connected with either the skin or oropharynx, or as a fistula between both skin and oropharynx openings
- c. Treatment: excision
- 5. Thyroglossal duct cyst or sinus
 - a. Cyst in the mid-anterior neck over or just below the hyoid bone, with or without a sinus tract to the base of the tongue (foramen cecum).
 - b. Treatment: excision
- 6. Ear Deformities
 - a. Congenital
 - i. Anotia: Complete absence; very rare
 - ii. Microtia: Vestigial remnants or absence of part of ear
 - iii. May present with other mandibular deformities, such as hemifacial microsomia or Treacher Collins
 - iv. Abnormalities of position (prominent ears)
 - b. Treatment
 - i. Anotia or microtia
 - (a) Brent technique (several modifications exist): Construction of sculpted ear framework from autologous cartilage graft that is buried under mastoid skin. Usually requires 2-4 operations starting at age 6-8, when rib cartilage is big enough.
 - (b) Synthetic implant covered by vascularized fascial flap (Medpor). Risk of extrusion from minor trauma.
 - ii. Prominent ears: creation of an antihelical fold and/or re-positioning/reduction of concha
 - iii. Traumatic loss of part or all of ear: treated similarly to microtia/anotia
 - iv. Use of a prosthetic ear may be indicated in some patients

II. PEDIATRIC FACIAL FRACTURES

- A. Epidemiology
 - 1. Uncommon. Comprise less than 15% of all facial fractures.
 - 2. Frequency increases with age
- B. Unique characteristics in children:
 - 1. At birth, ratio of cranial:facial volume is 8:1; by completion of growth it becomes 2.5:1. This changing ratio produces variation in fracture site frequency with age.
 - 2. Protective anatomical factors:
 - a. Larger fat pads
 - b. Decreased pneumatization of sinuses
 - c. Skeletal flexibility
 - d. Compliant sutures
 - 3. Fractures that entrap orbital contents (trap-door fractures) are more common secondary to greater bony elasticity

4. Up to 75% can have associated serious trauma (especially cervical spine, neurological, ophthalmologic and abdominal)
- C. Causes:
1. Most common overall (per National Trauma Databank): Motor vehicle crashes (MVC), violence and falls
 2. Some variation by age:
 - a. 0-5 years: falls
 - b. 6-11: MVC and play or sports
 - c. 12-18 years: sports and violence
- D. Fracture sites:
1. Vary with age
 2. Most common overall are orbital or mandible
 3. Cranial vault fractures more common in younger children
 4. Facial fractures more common as they grow older, following midface growth (maxilla, zygomaticomaxillary complex, nasal, mandible)
- E. General treatment principles (*see Chapter 8*):
1. Growing skeleton possesses inherent plasticity that may render operative intervention unnecessary for a given injury
 2. Usually more conservative with operative repair in this patient population due to fear of altering growing facial skeleton and developing dentition

III. BRACHIAL PLEXUS BIRTH PALSY

- A. Anatomy
1. Spinal roots: C5-T1
 2. Trunks: Upper, middle and lower
 3. Divisions: Anterior and posterior from each trunk
 4. Cords: Lateral, posterior and medial
 5. Terminal branches: Major peripheral nerves of upper extremity: myocutaneous, axillary, radial, ulnar, median
- B. Incidence: 1.5 per 100 full-term births
- C. Risk factors
1. Shoulder dystocia
 2. Forceps delivery
 3. Gestational diabetes (macrosomia)
 4. Breech delivery
- D. Presentation
1. Upper root cervical injury in 73%, or Erb-Duchenne palsy
 - a. Caused by injury in Erb's point (convergence of C5-C6 roots as they form upper trunk)
 - b. "Waiter's tip" appearance: lack of deltoid, supraspinatus and biceps functions, causing the arm to hang straight down at side with forearm pronated
- E. Evaluation
1. Passive and active ROM

- a. Evaluate with reflexes, such as Moro
- 2. Imaging usually not helpful
- 3. Physical therapy to preserve ROM
- F. Indications for surgical exploration
 - 1. Absent biceps or deltoid function by 3-6 months
 - 2. Absent elbow flexion, wrist, thumb or finger extension by 9 months
 - 3. Flail limb with Horner's syndrome
- G. Follow patients longitudinally. Partial recoveries might need tendon transfers or nerve grafting (controversial)
- H. 90% will have spontaneous resolution within 2 months. If biceps function recovered by 6 months, near-normal function can be expected.

IV. CONGENITAL HAND ANOMALIES

- A. Total body examination essential to look for associated deformities and syndromes
- B. Upper limbs develop from weeks 5-8
- C. Several pathologies exist; many are uncommon. Most common are syndactyly and polydactyly.
- D. Syndactyly: congenital fusion of digits
 - 1. Incidence
 - a. 1:200 live births
 - b. 10:1 whites > blacks
 - c. 2:1 males > females
 - d. Bilateral = unilateral
 - e. Long + ring finger most common (57%); thumb + index finger most rare (3%)
 - 2. May be familial (15-40% cases), sporadic simple, or associated with a syndrome (Apert, Poland)
 - 3. Types
 - a. Simple: no bony fusion
 - b. Complex: bony fusion
 - c. Complicated: associated with syndrome
 - d. Incomplete: web recessed
 - e. Complete: syndactyly to fingertip
 - 4. In simple form: ligaments usually normal. Duplicated tendons, nerves, sheaths
 - 5. In complex or complicated form: various fusion levels, fingernail synechia, abnormal tendons
 - 6. Evaluation: careful physical examination and X-rays (critical)
 - 7. Treatment: surgical. Dozens of techniques exist
 - a. Usually reassurance and waiting until hand is larger (6-12 months)
 - b. Earlier if growth will worsen deformity
- E. Polydactyly: more than 5 digits in one hand
 - 1. Very common
 - 2. 10:1 African descent > whites
 - 3. Usually sporadic

4. Ulnar polydactyly (postaxial)
 - a. African descent > whites
 - b. Classification
 - i. Type A: supernumerary digit well developed. Syndromic association in 29%.
 - ii. Type B: digit rudimentary and pedunculated. Rarely associated conditions.
 - c. Treatment:
 - i. Narrow stalk: ligation and autoamputation
 - ii. Type A requires operative separation with transfer of structures to adjacent finger
5. Radial polydactyly (preaxial)
 - a. May be associated with systemic conditions
 - b. Whites > African descent
 - c. Radiographic evaluation necessary to determine point of thumb duplication
 - d. Surgical reconstruction is more complex as its rarely a "floating finger"
 - e. Wassel Classification:
 - i. Type I: bifid distal phalanx (3%)
 - ii. Type II: suppicated distal phalanx (15%)
 - iii. Type III: Bifid proximal phalanx
 - iv. Type IV: Duplicated proximal phalanx (43%)
 - v. Type V: Bifid metacarpal
 - vi. Type VI: Duplicated metacarpal
 - vii. Type VII: Triphalangism
 - f. Treatment
 - i. Floating finger and narrow stalk: ligation
 - ii. All others: surgical but wait until 6-18 months. Ulnar side is preserved to keep ulnar collateral ligament

V. VASCULAR ANOMALIES

- A. Divided in tumors vs. malformations. Differentiate through physiology:
 1. Tumors: endothelial cell proliferation, malignant or benign (hemangiomas)
 2. Malformations: congenital malformation of vessels (venous, capillary, lymphatic, arteriovenous)
- B. Infantile hemangioma
 1. Most common benign tumor of infancy
 2. Incidence 4-10% by 1 year
 3. Female:male 3:1
 4. 60% in head and neck, 25% in trunk
 5. Appears in first weeks of life as a telangiectasia or clustered pinhead red lesions
 6. Phases:
 - a. Proliferative: rapid evolution until 0-12 months. 80% of tumor size by 5 months.

- b. Involuting phase: lasts 1 to 10 years. Tumor shrinks, color fades, lesion flattens
 - c. Involved phase: involution complete at age >10 years. Half of cases will have residual atrophy and contour deformity.
7. Treatment
- a. Conservative: total involution occurs in 50% by 5 years, 70% by 7 and 90% by 9.
 - b. Propranolol: induction of apoptosis and fat development
 - c. Intralesional steroids: in growth phase they can arrest growth but will not regress. Oral when lesion too large.
 - d. Laser: pulsed light and Nd:YAG lasers, mainly for ulcerated hemangiomas and for residual color
 - e. Surgery:
 - i. Urgent if hemangioma threatens important structures or function (i.e., visual, nasolaryngeal or auditory obstruction)
 - ii. Typically delayed until school age otherwise to give time for regression
8. Complications
- a. Bleeding
 - b. Ulceration
 - c. Infection
 - d. Kasabach-Merrit: Profound thrombocytopenia with kaposiform hemangioendothelioma
 - e. High output heart failure if large visceral hemangiomas
 - f. Emotional distress
9. Associated disorder: Congenital hemangioma
- a. Fully grown at birth
 - b. Two forms:
 - i. Rapidly involuting congenital hemangioma (RICH): disappears by first year
 - ii. Non-involuting congenital hemangioma (NICH): does not respond to pharmacotherapy
- C. Vascular malformations
1. Structural and morphologic anomalies resulting from faulty embryologic development
 2. Present at birth, grow proportionately with the child, and do NOT regress, unlike hemangiomas
 3. Capillary malformations or Port-Wine stains:
 - a. 0.3% newborns; females > males 3:1
 - b. Face in 80%
 - c. Usually affect distribution of V1-V2
 - d. Treatment: photocoagulation with laser +/- pharmacologic with imiquimod (antiangiogenesis)
 - e. Associated with
 - i. Sturge-Weber syndrome: may be associated with ocular and CNS anomalies

- ii. Klippel-Trenauney: in extremity, overlying deeper venous malformation and skeletal hypertrophy
 - iii. Parkes Weber syndrome: similar to previous, but with associated AV fistula
- 4. Venous malformation
 - a. Incidence 1-4%
 - b. Appear as bluish/purple lesions with spongy texture that swell with dependency and deflate with elevation
 - c. Treatment: sclerotherapy, compression garments for symptoms, Nd:YAG or argon lasers, surgical resection
- 5. Lymphatic malformations
 - a. Previously called hygroma.
 - b. Can be Macrocystic or microcystic.
 - c. Soft and compressible
 - d. Can cause bony overgrowth
 - e. Combined venous-lymphatic are common
 - f. Get frequently infected. Aggressive antibiotics crucial
 - g. Treatment: sclerosing agents, laser ablation, surgery, compression. Antibiotics for inflammatory episodes.
- 6. Arteriovenous malformations
 - a. Pulsatile high-flow lesion
 - b. Anatomy and hemodynamics defined by angiography
 - c. Varying clinical states:
 - i. Quiescent, appearing as only a pink stain
 - ii. Expansive, with thrill and dilated venous network
 - iii. Destructive, with cutaneous ulcers, necrosis, bleeding
 - iv. Decompensated, causing cardiac compromise
 - d. Treatment: embolization prior to surgical resection; wide local excision (high recurrence), ischemic suture techniques.
 - e. Complications
 - i. Consumptive coagulopathy
 - ii. Heart failure
 - iii. Local destruction of anatomy
 - iv. Bleeding

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