

CHAPTER 4

HEAD AND NECK

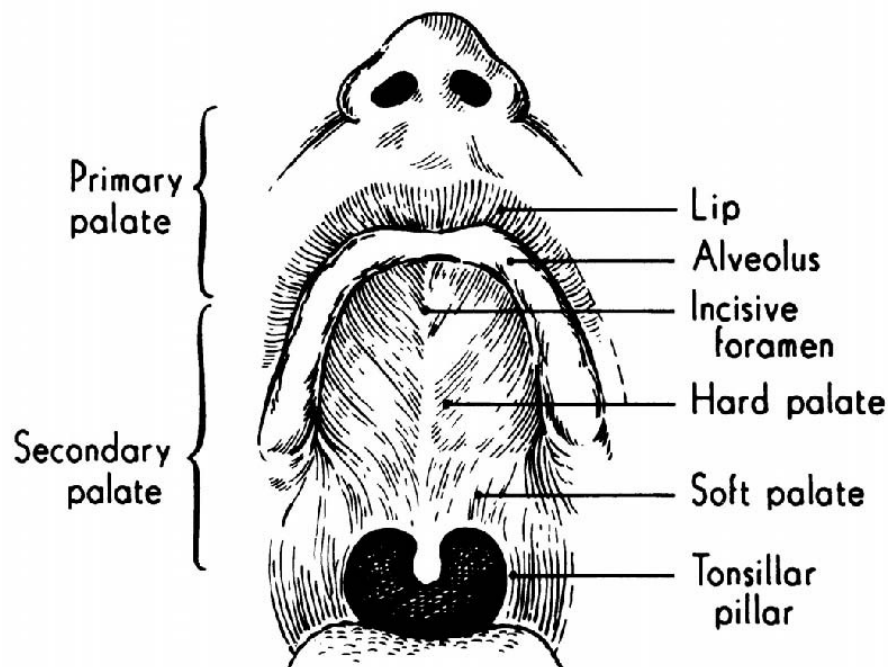
Problems of the head and neck in the practice of plastic surgery include congenital, traumatic, infectious, neoplastic, and other conditions. A working knowledge of embryology and anatomy of the head and neck is crucial in the diagnosis and surgical treatment of these diseases.

I. CONGENITAL

A. Cleft Lip and Cleft Palate

1. Anatomy (Fig. 4-1)

- a. Cleft Lip: occurs anterior to the incisive foramen and may also involve the alveolar process
- b. Cleft Palate:
 - i. Primary cleft palate: failure of fusion of median and lateral palatine processes
 - ii. Secondary cleft palate: failure of fusion of lateral palatine processes
- c. Submucous cleft palate (SMCP):
 - i. occult cleft of the soft palate
 - ii. classic clinical triad:
 - (a) bifid uvula
 - (b) notching of the hard palate
 - (c) zona pellucida – thinned area of soft palate containing only mucosa due to levator veli palatini muscles inserting on hard palate



(FIGURE 4-1)

- 2. Classification
 - a. Cleft Lip (Fig. 4-2)
 - i. Unilateral
 - (a) Complete
 - (b) Incomplete
 - ii. Bilateral
 - (a) Complete
 - (b) Incomplete

CLASSIFICATION OF LIP CLEFTS



**Unilateral
incomplete**



**Unilateral
complete**

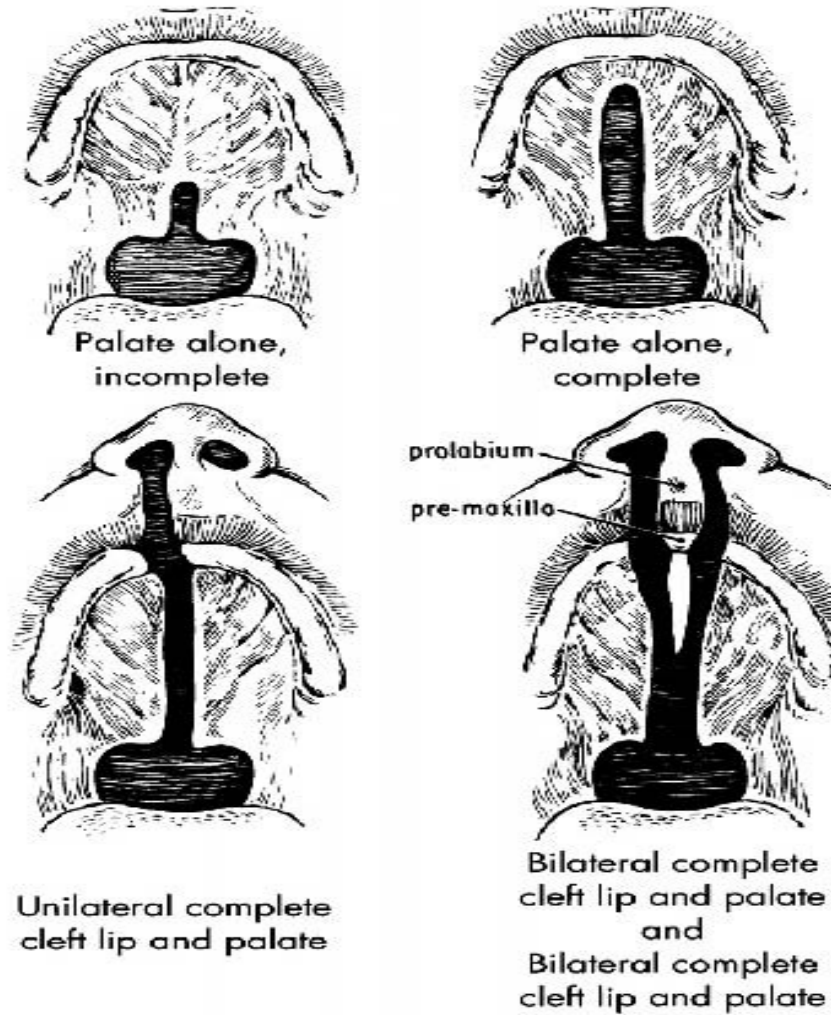


**Bilateral
complete**

(FIGURE 4-2)

- b. Palate (Fig. 4-3)
 - i. Palate alone
 - (a) Incomplete
 - (b) Complete
 - ii. Complete cleft palate
 - (a) Unilateral
 - (b) Bilateral

CLASSIFICATION OF PALATE CLEFTS



(FIGURE 4-3)

3. Prevalence of Unilateral Clefts
 - a. Cleft lip with or without cleft palate (CL±CP)
 - b. Ethnicity/Sex
 - i. 1:1000 Caucasians
 - ii. 1:2000 African-Americans
 - iii. 1:500 Asians
 - iv. 2:1 males:females
 - c. Cleft of palate alone (CP)
 - i. 1:2000 (all ethnicity)
 - ii. 1:2 males:females
4. Occurrence risk in offspring (Table 4-1)

Affected Relatives	Predicted Outcomes*
<i>CL±CP</i>	
One sibling	= 4%
One Parent	= 4%
Sibling and a Parent	= 16%
<i>CP</i>	
One Sibling	= 2.4%
One Parent	= 2.4 %
Sibling and a Parent	= 15%

Note — If congenital lip pits, inherited as autosomal dominant gene with variable penetrance (Van der Woude's Syndrome) — 50% incidence

**General predictions; individual cases may vary*

Table 4-1

5. Etiology
 - a. Multifactorial combination of heredity with or without environmental factors
 - b. Teratogenic agents — e.g. phenytoin, alcohol
 - c. Nutritional factors may contribute — folate deficiency
 - d. 3% of CL±CP are syndromic
6. Embryology
 - a. Cleft lip with palate forms at 4-6 weeks due to lack of mesenchymal penetration (merging) and fusion
 - b. Isolated cleft palate forms later, at 7-12 weeks, from lack of fusion
7. Pathophysiology and Functional Deficits
 - a. Cleft lip
 - i. Inability to form fluid and air seal in eating or speech
 - ii. Malocclusion as a result of intrinsic deformities of alveolar process and teeth
 - iii. Lack of continuity of skin, muscle and mucous membrane of lip with associated nasal deformity and nasal obstruction
 - iv. Deformity

- b. Cleft palate
 - i. Inability to separate nasal from oral cavity so that air and sound escape through nose in attempted speech
 - ii. Feeding impaired by loss of sucking due to inability to create intra-oral negative pressure
 - iii. Loss of liquids and soft foods through nose due to common nasal-oral chamber
 - iv. Middle ear disease and chronic otitis media due to Eustachian tube dysfunction
 - v. May be associated with Pierre-Robin sequence (cleft palate, micrognathia, glossoptosis). In these cases, airway obstruction and failure to thrive may be present. These cases may require ICU monitoring, prone positioning, nasopharyngeal airway, tongue-lip adhesion, tracheostomy, and now mandibular distraction (moving the base of the tongue forward by mandibular advancement). Distraction has been used with some good effect in severe cases, avoiding tracheostomy.
- 8. Team concept

Because of multiple problems with speech, dentition, hearing, etc., management of the patient with a cleft should be by an interdisciplinary team, preferably in a cleft palate or craniofacial center. Team members include: plastic surgeon, orthodontist, dentist, geneticist, pediatrician, speech therapist, audiologist, social worker, and psychologist.
- 9. Timing of Surgical Intervention
 - a. Cleft lip
 - i. Most common 10 weeks of age.
 - ii. Once followed “rule of 10’s” (10 weeks of age, Hgb 10, 10 lbs.), but now this rule is more historical.
 - iii. Range of cleft lip repair varies from 0-3 months of age in full-term, otherwise healthy, infant.
 - b. Cleft palate
 - i. Before purposeful sounds made (9-12 mos)
 - ii. depending upon health of infant, extent of cleft, but certainly before 18 months of age, if possible
 - c. Cleft nasal deformity
 - i. Most centers perform primary correction at the time of lip repair
 - ii. Secondary rhinoplasty at preschool age (4-5 years)
 - d. Alveolar cleft
 - i. Most centers perform secondary bone grafting at the stage of mixed dentition (9-12 years of age), just before eruption of the permanent canine, which is often affected by the cleft.
 - e. Dentofacial skeletal abnormality
 - i. In most cleft patients, this manifests as maxillary retrusion/hypoplasia
 - ii. In 25% of cleft patients, orthognathic surgery (jaw-straightening procedure) has to be performed to correct a malocclusion (abnormal bite).
 - iii. Orthognathic surgery can only be performed in skeletally mature individuals (14-16 years of age, women; 17-19 years of age, men).

- iv. With the advent of craniofacial distraction, surgical intervention can be performed earlier, but both patient and parents must be advised that the growing child may “outgrow” the correction, necessitating a repeat procedure.

10. Principles of Repair

- a. Cleft lip (3 months)
 - i. Repair of skin, muscle and mucous membrane to restore complete continuity of lip, symmetrical length and function
 - ii. Simultaneous repair of both sides of a bilateral cleft lip
 - iii. Preference for primary nasal reconstruction at time of lip repair
 - iv. In wide clefts (>10mm), presurgical orthodontics (palatal appliance, nasoalveolar molding) may be indicated, or a cleft lip adhesion (surgery to initially bring lip segments together, followed by definitive repair of lip 3 months later).
- b. Cleft palate (9-12 months)
 - i. One stage repair of both hard and soft palate
- c. Alveolar cleft (6-12 years)
 - i. At the time of eruption of permanent canine teeth

11. Secondary Repair

- a. Cleft lip
 - i. Orthognathic Lefort I osteotomy for maxillary hypoplasia (16 years)
 - ii. Secondary rhinoplasty (16-18 years)
- b. Cleft palate (4-6 years)
 - i. Correction of velopharyngeal inadequacy (nasal escape air due to remaining palatal defect): 4-6 years of age
 - ii. Repair of any oronasal palatal fistula

B. Other Congenital Anomalies

1. Craniosynostosis

- a. Definition: Premature fusion of one or more cranial vault sutures.
- b. 343 out of 1,000,000 live births
- c. Categorized into syndromic and nonsyndromic types.
- d. Nonsyndromic craniosynostosis:
 - i. Order of frequency according to suture type:
 - (a) Sagittal
 - (b) Metopic
 - (c) Coronal
 - (d) Lambdoid
 - ii. Characteristic head shape according to suture affected:
 - (a) Sagittal - scaphocephaly (scapho, Gr., meaning boat-shaped)
 - (b) Metopic - trigonocephaly (trigono, Gr., meaning triangular- or keel-shaped forehead)
 - (c) Coronal - brachycephaly (brachy, Gr., meaning short in AP direction).
 - iii. Ongoing debate as to whether or not these patients have an increased incidence of developmental delay
 - iv. Treatment:
 - (a) anterior vault reshaping (fronto-orbital advancement/reshaping)
 - (b) total vault reshaping

- (c) posterior vault reshaping
 - (d) depending on location and severity of craniosynostosis.
 - (e) Usually performed within first year of life to take advantage of molding capacity of skull
- e. Syndromic:
- i. Major associated syndromes:
 - (a) Apert (craniosynostosis, exorbitism, midfacial retrusion with complex syndactyly of the 2-4 digits of the hands/feet)
 - (b) Crouzon (craniosynostosis, exorbitism, midfacial retrusion)
 - (c) Pfeiffer (craniosynostosis, exorbitism, midfacial retrusion, broad thumbs and toes)
 - ii. Characteristic head shape involves turribrachycephaly (turri-, Gr., tower)
 - iii. 50% of Apert syndrome patients have substantial mental delay; Crouzon and Pfeiffer syndrome patients usually develop normally
 - iv. Genetic defect identified in fibroblast growth factor receptor (FGFR) genes (Apert, Crouzon---FGFR2, Pfeiffer—FGFR1)
 - v. Goals of surgery:
 - (a) Release fused cranial sutures
 - (b) correct profound exorbitism to prevent corneal exposure/blindness
 - (c) correct malocclusions
 - vi. Surgical interventions:
 - (a) Anterior/posterior/total vault reshaping (0-1 years)
 - (b) Monobloc (osteotomy and advance forehead and face simultaneously with bone grafts/fixation)
 - (c) Le Fort III (osteotomy and advance face) (4-6 years)
 - (d) Craniofacial distraction leads to greater advancement, less relapse than conventional procedures.
2. Facial Dysostoses
- a. Treacher-Collins Syndrome (Mandibulofacial Dysostosis)
 - i. Rare, autosomal dominant, variable penetrance disorder
 - ii. Affected gene on chromosome 5q
 - iii. Clinical manifestations:
 - (a) hypoplasia/aplasia of the zygomatic bone
 - (i) lateral orbit deficiency
 - (ii) midface retrusion
 - (iii) lateral canthus hypoplasia/downward slanting palpebral fissures
 - (b) colobomas
 - (c) variable external ear malformations and deafness
 - (d) mandibular hypoplasia with microretrognathia
 - (i) airway compromise
 - (ii) necessitates tracheostomy and distraction of mandible
 - (e) choanal atresia
 - (f) bilateral cleft palate
 - (g) normal intelligence
 - iv. Treatment:

- (a) Skeletal and soft tissue augmentation of deficient areas with autogenous bone (calvarium, rib, iliac crest) and autologous fat/tissue transfer, respectively.
 - (b) Mandibular distraction may be necessary for achieving a stable airway
 - b. Hemifacial Microsomia
 - i. Third-most common congenital malformation (following club foot and cleft lip and palate).
 - ii. 1:7000 live births affected
 - iii. No genetic defect ascribed; leading theory of cause is related to stapedia artery thrombosis during embryogenesis
 - (a) 1st & 2nd branchial arches affected
 - iv. Usually associated with microtia
 - v. Manifestations:
 - (a) hemifacial deficiency (skeletal and soft tissue)
 - (i) C-shape deformity
 - (ii) off center position of chin
 - (b) microtia
 - (c) mandibular hypoplasia
 - (i) malocclusion from an abnormal cant (secondary to reduced vertical height of the ramus)
 - (d) macrostomia
 - (e) hearing loss
 - vi. Associated with Tessier #7 facial cleft and variable facial nerve palsy
 - vii. Pruzansky classification for mandibular discrepancy classification (OMENS):
 - (a) Orbit
 - (b) Mandible
 - (c) Ear
 - (d) Nerve
 - (e) Soft tissue
 - viii. Treatment:
 - (a) Augment deficient areas
 - (i) Skeletal: autogenous bone (calvarium, rib, iliac crest)
 - (ii) Soft tissue: free flap and/or fat grafting
 - (b) Mandibular depends upon severity of hypoplasia. Distraction may be necessary for achieving correction of malocclusion versus conventional orthognathic procedures to correct jaw discrepancies in adolescence.
 - (c) bone-anchored hearing aids
 - c. Goldenhar Syndrome
 - i. Also known as oculoauriculo-vertebral (OAV) spectrum
 - ii. Manifestations:
 - (a) hemifacial microsomia,
 - (b) vertebral spine abnormalities
 - (c) abnormalities of heart, kidneys, lungs
- 3. Embryologic Defects
 - a. Branchial cyst, sinus, or fistula

- i. Epithelial-lined tract frequently in the lateral neck presenting along the anterior border of the sternocleidomastoid muscle.
 - ii. 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
 - iii. Treatment — excision
- b. Thyroglossal duct cyst or sinus
 - i. 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)
 - ii. Treatment — excision
- c. Ear deformities
 - i. Complete absence (anotia) — very rare
 - ii. Vestigial remnants or absence of part of ear (microtia)
 - iii. Absence of part or all of external ear with mandibular deformity (hemifacial microsomia)
 - iv. Abnormalities of position (prominent ears)
 - v. Treatment:
 - (a) Anotia or microtia—construction from autogenous cartilage graft or synthetic implant, vascularized fascial flap, skin graft — usually requires more than one operation. (Traumatic loss of part or all of ear is treated similarly).
 - (b) Use of a prosthetic ear may be indicated in some patients
 - (c) Prominent ears — creation of an antihelical fold and/or re-positioning/reduction of concha

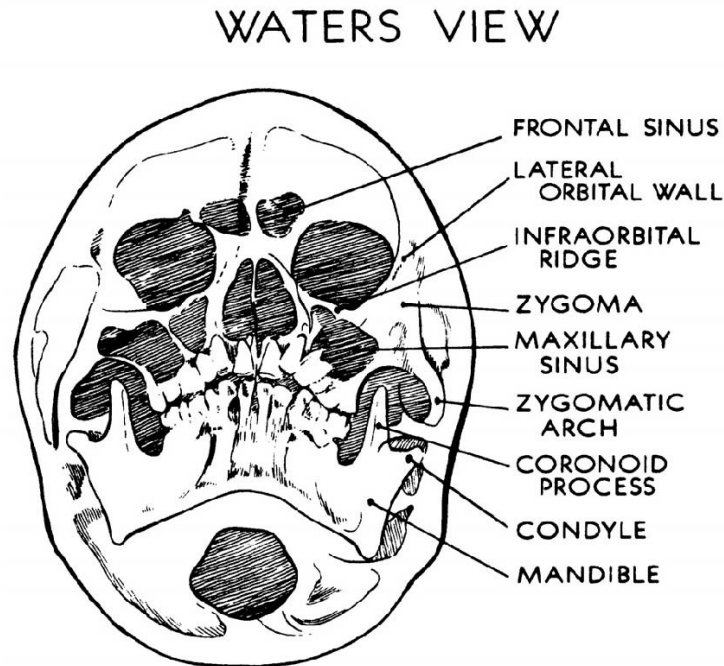
II. TRAUMATIC

- A. Facial soft tissue injuries
 - 1. Stabilize patient and manage concomitant traumatic injuries (ABCDE, primary survey)
 - 2. Establish airway (may be obstructed by blood clots or damaged parts)
 - a. Finger (jaw thrust, e.g.)
 - b. Suction
 - c. Endotracheal intubation
 - d. Cricothyroidotomy or tracheotomy
 - 3. Control of active bleeding by pressure until control by directly ligating in operating room or embolization in interventional radiology suite
 - 4. Palpate facial skeleton for underlying bone injury; rule out injury to facial nerve, parotid duct, etc.
 - 5. Radiologic evaluation (C-spine X-rays, CT scan, panorex)
 - 6. Tetanus and antibiotic prophylaxis
 - 7. Repair as soon as patient's general condition allows with
 - a. Preferably less than 8 hours post-injury
 - b. Primary closure may be delayed up to 24 hours
 - c. Conservative debridement of nonviable tissue and foreign bodies
 - d. Careful wound irrigation with physiologic solution
 - e. Meticulous re-approximation of anatomy

B. Facial bone fractures

1. Diagnoses

- a. Consider patient history
- b. Physical examination for asymmetry, bone mobility, diplopia, extraocular muscle entrapment, sensory loss, malocclusion, local pain
- c. Old (pre-injury) photographs often useful to assess baseline
- d. Imaging
 - i. Skull x-ray (rarely performed today) and cervical spine
 - (a) Waters view for facial bones (Fig. 4-6); good for orbital floor

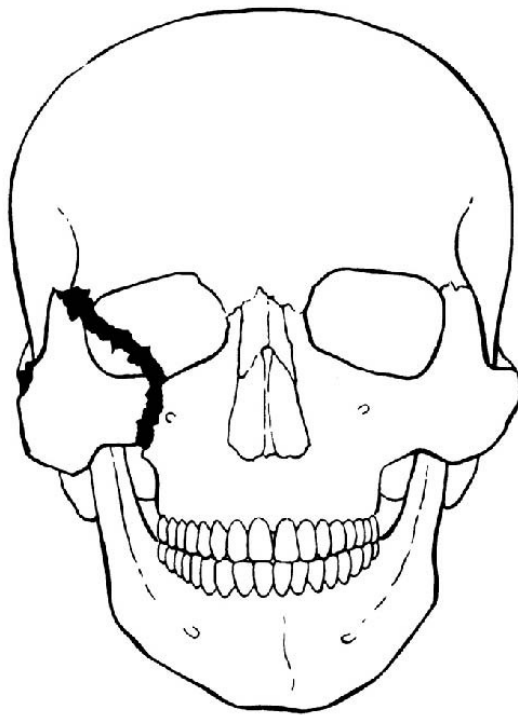


(FIGURE 4-6)

- ii. CT scan (now imaging modality of choice)
 - iii. C-spine x-ray
 - iv. Panorex x-ray if mandible fracture present and C-spine cleared
- ### 2. Treatment
- a. Re-establishment of normal occlusion is of primary importance
 - b. Use of interdental wiring (mandibulomaxillary fixation/MMF), plating, or other devices in patient with teeth
 - c. Use of patient's dentures or fabricated temporary dentures in edentulous patient
 - d. Reduction and immobilization of other fractures
 - e. When dealing with panfacial fracture, stabilize articulating element (mandible), first by mandibulomaxillary fixation (MMF)
 - f. Once occlusion is aligned, work systematically, either "outside-in" (Gruss) or "inside-out" (Manson), establishing facial height, width, and projection by aligning key facial buttresses (open reduction) and plating of fractures (internal fixation)

3. Specific Fractures

- a. Mandible — often bilateral (ring concept)
 - i. Depending on anatomical region (parasymphysis, body, angle, subcondyle) and overall function (malocclusion), open reduction and internal fixation (ORIF) may be indicated.
 - ii. Clinical signs:
 - (a) Malocclusion
 - (b) Sensation of chin decreased due to mental nerve injury
 - iii. Imaging
 - (a) Panorex x-ray
 - (b) CT scan
 - (c) C-spine x-ray: 10-13% of mandible fractures coincide with c-spine fracture; maintain C-spine stabilization until absence of injury can be confirmed
- b. Zygomatic complex (Fig. 4-4)

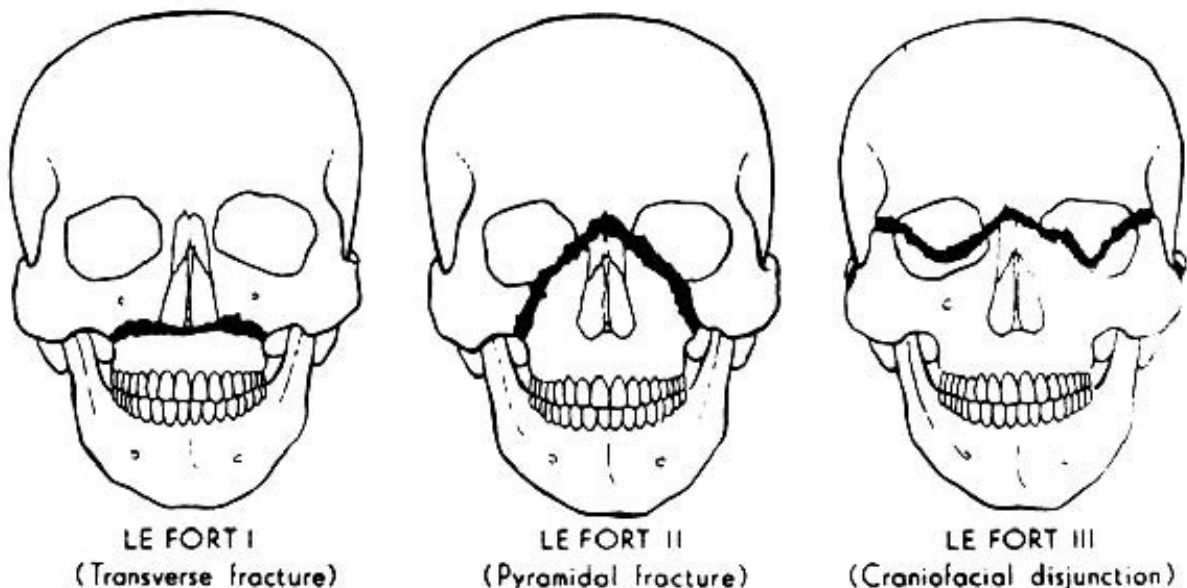


FRACTURE OF ZYGOMATIC COMPLEX

(FIGURE 4-4)

- i. Commonly associated with orbital floor fractures
 - (a) Eye exam
 - (i) Extraocular movements / entrapment
 - (ii) Visual acuity

- (iii)Globe injury
 - (b) Ophthalmology consultation if suspicious of globe injury
 - ii. Superior orbital fissure syndrome
 - (a) Due to injury to contents of superior orbital fissure (CN III, IV, VI)
 - (b) Ophthalmoplegia (CN III, IV, VI)
 - (c) Proptosis
 - (d) Ptosis (CN III)
 - (e) Dilated pupil (CN III)
 - (f) If also blindness (CN II), called ORBITAL APEX SYNDROME (surgery urgent)
 - iii. Indications for surgery
 - (a) Entrapment
 - (b) Enophthalmos
 - (i) Severe displacement creating facial asymmetry
- c. Maxilla
 - i. Le Fort fractures (Fig. 4-5)
 - (a) Disrupts vertical maxillary buttresses
 - (i) Zygomaticomaxillary
 - (ii) Nasofrontal / piriform
 - (iii)Zygomaticofrontal
 - (iv)Pterygomaxillary
 - (b) Treatment involves open reduction and internal fixation with miniplates to re-establish facial proportions and occlusion



(FIGURE 4-5)

- d. Naso-orbital-ethmoidal (NOE)
- e. Isolated orbital floor fractures: blow-out versus blow-in

- i. Check for entrapment (failure to move eye in all directions)—if present, must decompress orbit within 48 hours
 - ii. Check for enophthalmos (position of globe in relation to unaffected globe in worm's eye view).
 - iii. Must operate for enophthalmos 2mm or greater.
 - f. Frontal sinus
 - g. Pediatric craniofacial fractures: Usually more conservative with operative repair in this patient population, due to growing facial skeleton and developing dentition.

III. INFECTIONS

- A. The head and neck are relatively resistant to infection due to their robust vascularity
- B. Routes of spread
 - 1. Upper aerodigestive infections may track into the mediastinum
 - 2. Scalp and orbital infections may spread intracranially via the dural sinuses and ophthalmic veins
- C. Facial cellulitis — mostly due to staph or strep — may use a cephalosporin
- D. Oral cavity infections — mostly due to anaerobic strep and bacteroides. Use extended spectrum penicillin or other anaerobic coverage
- E. Acute Sialadenitis — fever, pain, swelling over the involved parotid gland. Seen with dehydration, debilitation, diabetics, poor oral hygiene. Treat with antibiotics, fluids
- F. Atypical mycobacteria — seen in enlarged lymph nodes; drainage rarely required. Special cultures may be necessary

IV. NEOPLASTIC (*exclusive of skin — see Chapter 3*)

- A. Salivary gland tumors or disorders
 - 1. Classification of tumors by location
 - a. Parotid — most common (80%), most are benign (80%)
 - b. Submandibular — 55% incidence of malignancy
 - c. Minor salivary glands — least common, with highest incidence of malignancy (about 75%)
 - 2. Diagnosis
 - a. Primarily by physical examination
 - i. Any mass in the pre-auricular region or at the angle of the jaw is a parotid tumor until proven otherwise
 - b. Bimanual palpation — simultaneous intraoral and external palpation
 - c. X-rays occasionally helpful for diagnosis of stone; sialography (injection of contrast material into duct) is rarely if ever indicated
 - d. Signs more commonly seen with malignancy
 - i. Fixed or hard mass
 - ii. Pain
 - iii. Loss or disturbance of facial nerve function
 - iv. Cervical lymph node metastases
 - 3. Treatment

- a. For stone near duct orifice
 - i. Simple removal
 - b. For benign tumors (or stones in duct adjacent to gland)
 - i. Surgical removal of gland with sparing of adjacent nerves, e.g. facial nerve with parotid; lingual and hypoglossal nerves with submandibular
 - c. For malignant tumors
 - i. Surgical removal of entire gland with sparing of nerve branches that are clearly not involved
 - (a) Radiation therapy if tumor not completely removed
 - (b) Cervical lymph node dissection with tumors prone to metastasize to nodes
4. Pathology
- a. Benign
 - i. Pleomorphic adenoma — (benign mixed) high recurrence rate with local excision
 - ii. Papillary cystadenoma lymphomatosum (Warthin's tumor) — may be bilateral — (10% male, age 40-70)
 - b. Malignant
 - i. Mucoepidermoid
 - ii. Malignant mixed
 - iii. Adenocarcinoma
- B. Tumors of oral cavity
- 1. Classification
 - a. Anatomical — malignancies behave differently according to anatomic site and prognosis worsens from anterior to posterior
 - i. Lip
 - ii. Anterior two-thirds tongue
 - iii. Floor of mouth
 - iv. Buccal
 - v. Alveolar ridge
 - vi. Posterior tongue
 - vii. Tonsillar fossa and posterior pharynx
 - viii. Hypopharynx
 - b. Histopathologic
 - i. Benign — according to site — fibroma, osteoma, lipoma, cyst, etc.
 - ii. Malignant
 - (a) Most are squamous cell carcinoma or variants
 - (b) Palate carcinomas are often of minor salivary gland origin
 - (c) Sarcomas in mandible, tongue, other sites are rare
 - (d) TNM staging is helpful for treatment planning and prognosis (i.e. tumor size, lymph node metastases, systemic metastases)
 - 2. Diagnosis
 - a. Examination — including indirect laryngoscopy and nasopharyngeal endoscopy when indicated
 - b. Biopsy of any lesion unhealed in 2-4 weeks
 - c. X-rays and scans as indicated
 - i. Conventional views, panorex, etc.

- ii. Tomography
 - iii. Computerized axial tomography
 - iv. Bone scan
 - v. Magnetic resonance imaging
3. Treatment
- a. Surgical
 - i. Benign
 - (a) Simple excision
 - ii. Malignant
 - (a) Wide local excision with tumor-free margins
 - (b) Regional lymph node dissection when indicated
 - (c) Palliative resection may be indicated for comfort and hygiene
 - (d) Immediate reconstruction with vascularized flaps when indicated by size and location of defect
 - b. Radiation therapy
 - i. Preoperative
 - (a) To increase chance for cure, especially with large lesions
 - (b) May make an inoperable lesion operable
 - ii. Postoperative
 - (a) If tumor-free margin is questionable
 - (b) For recurrence
 - (c) Prophylactic — controversial
 - c. Chemotherapy — usually for advanced disease

V. MISCELLANEOUS

A. Disorders of the jaw

- 1. Deformities of the mandible
 - a. Classification
 - i. Retrognathia — mandibular retrusion with respect to maxilla
 - ii. Prognathia — mandibular protrusion with respect to maxilla
 - iii. Micrognathia — underdeveloped, retruded mandible
 - iv. Open bite — teeth cannot be brought into opposition
 - v. Crossbite — lower teeth lateral to upper teeth
 - vi. Micro — and macrogenia — under- or overdevelopment of chin
 - b. Diagnosis
 - i. Physical examination
 - ii. X-rays, including a cephalogram (lateral x-ray at a fixed distance) to measure relationships of skull, maxilla and mandible
 - iii. Dental casts are made (usually by an orthodontist) and “model” or mock surgery is performed on the casts to determine degree of advancement/setback of bone.
 - c. Treatment
 - i. Establishment of normal or near normal occlusion of primary importance
 - ii. Use of osteostomies with repositioning of bone segments, bone grafts as needed, with or without orthodontic corrective measures as needed
 - iii. Mandibular distraction for severe discrepancies

2. Deformities of the maxilla
 - a. Most commonly, retrusions or underdevelopment, “dish-face”
 - b. Must also examine the vertical height of the midface (vertical maxillary excess, VME versus vertical maxillary deficiency, VMD)
 - c. Diagnosis — as for lower jaw
 - d. Treatment — as for lower jaw
3. Temporomandibular joint disorder
 - a. Etiology
 - i. Previous trauma
 - ii. Arthritis
 - iii. Bone overgrowth
 - iv. Bruxism
 - v. Tumors
 - b. Symptoms:
 - i. Pain
 - ii. Crepitus
 - iii. Joint Noises
 - iv. Limited opening
 - v. Occlusion change
 - c. Diagnosis
 - i. Consider patient history
 - ii. Examination
 - (a) Auscultation
 - (b) Opening
 - (c) Occlusion
 - iii. X-rays
 - (a) Tomograms
 - (b) Arthrogram/arthroscopy
 - (c) MRI
 - d. Treatment
 - i. Conservative: joint rest, analgesics, bite plate, etc.
 - ii. Surgery — seldom indicated

B. Facial paralysis

Loss of facial nerve results in very significant asymmetry and deformity of the face, drooling, exposure of the cornea on the affected side. Deformity is accentuated by muscle activity of normal side (if unilateral)

1. Etiology
 - a. Idiopathic (Bell’s palsy)
 - b. Congenital
 - c. Traumatic
 - d. Infectious
 - e. Tumor
 - f. Vascular (intracranial)
2. Diagnosis
 - a. Demonstrated by asking patient to raise eyebrow, smile, etc.

3. Treatment includes:
 - a. Supportive — for most Bell's palsies
 - b. Protect cornea by taping lids, lid adhesions—ophthalmology consultation is critical
 - c. Re-establishment of nerve function by repair or nerve graft (sural nerve common donor nerve)
 - d. Other measures, such as muscle transfers, static suspension, skin resections, free tissue transfers of muscle, etc.

CHAPTER 4 — BIBLIOGRAPHY

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