CHAPTER 87  ■  FACIAL TRAUMA

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Facial trauma is any injury of the midface, including the maxillary complex. Panfacial injuries involve trauma to the upper, middle, and lower facial bones. Although injuries to the mandible often accompany other facial injuries, mandibular trauma is usually discussed as a separate entity. Injuries to the frontal bone and frontal sinus are also discussed separately since they are often complicated by injuries to the cranial vault and violate the intracranial space. Often, such distinctions based on anatomic boundaries are used for the sake of organization, as injuries to the nasal cavity or sinuses, orbit, and ethmoids may also involve violations of the cranium and cranial space.

Facial trauma may present in clinically different ways: with lacerations of skin and soft tissues, obstruction of the nasal cavity or sinuses, problems with vision, and problems with occlusion, which often are more outward manifestations of underlying facial fractures. Maxillofacial injury may result from both penetrating and blunt trauma. Comprehensive management and treatment of facial trauma involves airway control, control of bleeding, reduction of swelling, prevention of infection, repair of soft tissue lacerations, and repair of bone fractures to restore function and esthetic form to the face. The aim of this chapter is to discuss the potential pitfalls and problems that facial injuries may create. The operative management of facial fractures has undergone many changes in the last decade. With the advent of rigid fixation and continued new technologies, detailed discussions of methods of operative reconstruction of different types of facial fractures are best discussed in specialty textbooks.

PATHOPHYSIOLOGY

In the treatment of a patient with multiple maxillofacial injuries, it is critical to differentiate injuries that require immediate operative intervention from those for which operation can be deferred. However, immediate intervention may be indicated for stabilization. Procedures that require an extensive workup are delayed until the patient is clinically stable.

The most essential component of initial care begins with the ABC’s (airway, breathing, circulation), as well as cervical spine assessment. As with any other trauma patient, the facial trauma patient should be evaluated in a systematic and comprehensive fashion in cooperation with the trauma team. Although rare, isolated facial trauma may be severe and life threatening. Critical facial injuries are usually obvious upon presentation (e.g., gunshot wound to the face, profuse bleeding from orifices). Abdominal, thoracic, cervical spine, and neurosurgical emergencies take priority over maxillofacial injury. If possible, a detailed history of the event and past medical history should be obtained from the patient, paramedics, and/or family members. Dental records are typically difficult to obtain but very helpful in diagnosis and treatment planning.

Particularly in the facial trauma patient, early airway control and neurologic assessment of a potential head injury are critical because the eyes and oropharynx are the “shock organs” of the face and development of facial edema will obscure the pupils and obstruct the upper airway. Immediate treatment of maxillofacial trauma patients is indicated in the following situations: (a) airway compromise, (b) severe hemorrhage, (c) large open wounds, (d) superior orbital fissure and orbital apex syndrome, (e) mandibular condylar impaction into the cranial fossa, and (f) if urgent surgical procedures need to be performed by other services.

CONTRAINDICATIONS

Definitive treatment of maxillofacial injuries is delayed if the patient has severe, concomitant, and/or undetermined systemic trauma. Definitive treatment of facial fractures can be delayed as much as 2 weeks after injury as long as the fractures do not violate the cranial space. Patients with neurologic or cranial injury are operated on when stable. Blood volume, electrolytes, and acid-base problems should all be addressed prior to the surgical procedure. In addition, the resolution of facial edema and perineural conusion during this period of time allows for a much more accurate evaluation and simplifies surgical planning and operative treatment.

EPIDEMIOLOGY: CAUSES, INCIDENCE, AND RISK FACTORS

Facial trauma is multifactorial with a variety of etiologies ranging from sports, falls, penetrating injuries, assault, and violence, to motor vehicle accidents. The incidence and frequency of any specific etiology varies with culture and within geographic regions. Prominent factors include lifestyle, population density, and socioeconomic status. Urban trauma centers evaluate and treat many facial trauma patients on a daily basis. Many university hospitals are well known for their high volume of facial fracture management. Oral and maxillofacial surgery, plastic surgery, and otorhino-laryngology services are heavily consulted by the emergency department and trauma team to assist with management of facial injuries. A large body of research has focused on data collection regarding types of facial trauma and studies on the outcome and morbidity associated with the treatment of facial fractures. Data regarding age, race,
gender, social habits, mechanism of injury, and incidence of pre-
vious facial trauma are available from many centers in many
countries (4). In most urban areas, maxillofacial fractures account
for the majority of injuries, followed by lacerations and mis-
cellaneous facial injuries. Interpersonal violence is the primary
cause of injury and motor vehicle accidents are the next most
frequent culprit. Half of the patients who experience facial in-
juries as victims of assault are likely to have interpersonal vio-
lence and have a high likelihood for a future injury (5).

AIRWAY MANAGEMENT

The possibility of cervical spine injury makes airway manage-
ment more complex in the facial trauma patient. Spinal injuries
are increased fourfold if there is a clinically significant head
injury (Glasgow coma scale [GCS] score <9). A cervical spine
injury should be suspected in all patients involving forced blunt
trauma. Cervical spine injury may be occult, in which case sec-
ondary injury to the spinal cord must be avoided. Immobiliza-
tion of the cervical spine must be instituted immediately until a
complete clinical and radiologic evaluation has excluded injury
(6).

A fully conscious, coherent patient is maintaining his or
her own airway. Because overall status may deteriorate at any
time, the ABCs must constantly be reassessed. The following
subsets of patients require immediate securment of airway to
prevent respiratory failure: (a) patients with GCS score <9; (b)
patients with sustained seizure activity; (c) patients with un-
stable midface trauma; (d) patients with direct injuries to the
airway (teeth), blood, and secretions. The classic “chin lift” or “jaw
thrust” maneuvers are commonly employed for assessment of
airway patency and to remove obstruction of the tongue base.
However, jaw thrust and chin lift may cause distraction of at
least 5 mm in a cadaver with C5–6 instability that is unaffected
by the use of a rigid collar (6). Manual in-line axial stabiliza-
tion must therefore be maintained throughout. Bag and mask
ventilation also produce significant degrees of cervical spine
movement at zones of instability. The “sniffing the morning
air” position for standard endotracheal intubation should sim-
ilarly be avoided as it flexes the lower cervical spine and extends
the occiput on the atlas. Atlanto-occipital extension is neces-
sary to visualize the vocal cords. Patients with unstable C1 or
C2 injuries might therefore be at more risk from this technique.
The hard C-collar may interfere with intubation efforts. If nec-
essary, the front part of the collar can be removed to facilitate
intubation as long as manual stabilization remains in effect.

The safest method of securing an endotracheal tube remains
debatable. The Advanced Trauma Life Support (ATLS) recom-
mends a nasotracheal tube in the spontaneously breathing pa-
tient, and orotracheal intubation in the apneic patient. Orotra-
cheal intubation is the fastest and surest method of intubating
the trachea and therefore the more commonly used method.
At Shock Trauma in Baltimore, Maryland, more than 3,000
patients were intubated orally with a modified rapid sequence
induction technique with preoxygenation and cricoideal pressure.
Ten percent of these patients were found to have cervical spine
injury and none deteriorated neurologically following intuba-
tion (7). Blind nasal intubation is ultimately successful in 90% of
patients but requires multiple attempts in up to 90% of these. 
Nasotracheal intubation is (relatively) contraindicated in pa-
tients with potential skull base fractures or unstable midface
injuries that typically involve the naso-orbito-ethmoid (NOE)
complex. The same holds true for the use of nasogastric tube
placement. Any paramanipulation may notoriously pro-
duce or recreate local hemorrhage, making airway manipula-
tions difficult or impossible. Inadvertent placement and con-
tamination/violation of the cranial space is a theoretical possi-
bility. 

LIFE-THREATENING HEMORRHAGE AND BLEEDING FROM FACIAL FRACTURES

In the multisystem-injured patient, hemorrhage is the most
common cause of hypovolemia. Hemorrhage can be external
or internal into body cavities. Because the face and neck have a
rich vascular supply, injuries in these areas can lead to substan-
tial blood loss. Major hemorrhage can result from large scalp
wounds, nasal or midface fractures, and penetrating wounds.
As opposed to bleeding into body cavities, hemorrhage in the
head and neck area is almost always immediately detectable in
the trauma bay on clinical examination and often external in
nature. Hence, external hemorrhage can usually be controlled
by direct pressure to the wound and/or bleeding areas. Pressure
can also be applied proximal to major arteries if direct pressure
to the wound is not effective.

Scalp wounds are notorious for large amounts of blood loss
in a short time if the galea is involved. Scalp wounds can be
rapidly approximated with 2.0 nonabsorbable sutures (nylon,
Prolene) or staples if available. Sutures should be placed away
from the wound edge to ensure hemostasis as the gauze tends to retract. Direct pressure over the wound can be applied as well. The patient can be stabilized first before continuing with further diagnostic studies.

Nasal fractures and midface fractures can result in tearing of the ethmoidal arteries. Most of these can be controlled with direct pressure or packing. Nasal packing can be made of gauze, foam, or cotton. The term “packing” refers to commercially available gauze strips or cotton pledges that are packed as they are inserted into the nasal cavity via the nares to form a compression plug. Packings may be made by cutting the fingers of a sterile examination glove and stuffing with gauze. Nasal packing may be coated with petrolatum, antibiotic ointment, or agents such as lidocaine and thrombin that aid in hemostasis and clot formation. Perforated foam nasal packs may have small tubes in the center of the pack to allow nasal breathing while the packing is in place as nasal packing prevents air exchange through the nose. Nonintubated patients with nasal packing in place should have the head of the bed elevated 30 degrees and be observed and monitored for respiratory distress. Continued bleeding may not be apparent on the nasal side of the packing. Nasal packing easily slips posteriorly with swallowing or out with movement or sneezing. The posterior oropharynx should be checked regularly.

Fractures of the posterior maxillary wall, as in LeFort I and II fractures, may be associated with profuse bleeding from the internal maxillary artery. Bleeding from this artery can be very difficult to control by gauze packing. Epinephrine and liquid thrombin can be added to the packing and the head elevated to help achieve hemostasis. However, a postnasal pack has to be used in patients who cannot control the bleeding in the postnasal area. This is a difficult area to pack. A balloon catheter can be passed through the nose and pulled out through the mouth. The safety and length of nasal packing is not evidence based. In rhinoplasty surgery, nasal stents and packs are routinely left in place for 7 to 10 days. Complications can be packed related. The most common complication of nasal packing is that removal of the packing dislodges healing tissue and causes recurrent hemorrhage. Hypoxemia and hypercarbia can cause respiratory and cardiac complications. Airway obstruction and asphyxiation can occur if the nasal packing slips back into the airways, particularly during sleep. Complications may occur if a pack compresses the eustachian tube. Rarely, infections can develop in the nose, sinus, or middle ear after nasal packing and lead to toxic shock syndrome (TSS). Risk factors for TSS include any wound and respiratory infections, such as sinusitis, sore throat (pharyngitis), laryngitis, tonsillitis, or pneumonia. Foul odor is alarming as the nasal pack ages over the next 48 hours. Bruising or swelling of the eyelids secondary to nasal packing may develop. Therefore, packing is best removed within 24 to 48 hours following placement provided the patient’s clinical condition has stabilized.

When tight naso/oral packing fails in unstable patients, supralselective arteriography and embolization is the treatment of choice in institutions where this modality is available (8). Ligation of the external carotid artery is a last resort in the unstable multitrauma patient who cannot be transported. However, due to collateral circulation of the face, ligation is seldom truly effective. Best control of hemorrhage is obtained by exploratory surgery and fixation of fractures. In patients with isolated LeFort fractures, open reduction/internal fixation (ORIF) is the first line of treatment (9).

**WOUND MANAGEMENT**

The management of facial soft tissue injuries depends on the area of injury. However, there are some basic rules that apply in treating soft tissue injuries. Soft tissue injuries are only properly evaluated after the wound is cleaned of dirt, foreign bodies, debris, and dry blood. A local anesthetic is usually necessary to properly clean the wound and perform a thorough examination. In the awake patient, most local infiltrative anesthetics cause great discomfort, which may compromise spinal precautions. Very slow injection using a fine needle (30 gauge) as well as adding bicarbonate in a 1:10 ratio may help. Facial nerve function should be assessed in all patients with facial lacerations and nerve function should be documented prior to anesthetic use. Anatomic landmarks are of great importance: if facial nerve paralysis results from a laceration anterior to a line perpendicular to the lateral canthus of the eye, the terminal nerve branches are involved. If facial nerve paralysis results from a laceration posterior to this imaginary line, the facial nerve should be explored. Ideally, repair of the facial nerve should occur as soon as possible, but no later than 72 hours unless the wound is heavily contaminated. In this case, the nerve endings are tagged with a permanent suture and repair is performed when the wound is clean. In patients with deep lacerations of the cheek, the wound should also be explored for injury to the parotid duct (Stensen duct). One may see saliva in the wound if the duct is lacerated. The parotid duct is repaired over a stent to prevent stenosis. Lacerations and contusions of specialized three-dimensional structures such as the eyelid, nasal alae, and ear are often best referred to a specialist, especially if flaps show signs of devascularization.

Optimum timing of facial laceration repair is a topic of debate. After tetanus prophylaxis, soft tissue repair can be performed within 12 to 24 hours provided the wounds are irrigated, cleaned, and kept moist. Because of the abundant blood supply, definite wound closure can be delayed and, in general, requires minimal debridement. “Traumatic tattooing” is a greater problem in the face than skin loss. A perfect repair is difficult to obtain in the acute setting as areas of contusion have to declare themselves and often leave irregularities later on. As long as important anatomic landmarks are aligned (e.g., vermilion border of the lip, gray line of the eyelid) and like tissue are approximated (mucosa to mucosa, muscle to muscle, cartilage to cartilage, and skin to skin), revisions can be done later. Deep sutures are used to close dead space to avoid hematomas and to remove tension from the skin closure, preventing an unsightly scar. Good aesthetic results depend less on suture technique than on proper redraping of tissues. Scars are noticeable as a result of reflection of light and creation of shadow. For cosmetics, it is of importance to create an “even” closure and, if possible, to place scars in areas of shadow and along lines perpendicular to facial muscle pull. Photographic documentation is important so that the patient may later realize the extent of the original injury, to follow healing, to document subsequent revisions, and for medical-legal reasons.

**CRANIAL NERVE EXAM**

Olfaction (cranial nerve [CN] I): Olfaction is typically not examined in the acute trauma bay setting but reserved...
for later trauma surveys (e.g., tertiary survey). Damage to CN I should be considered with NOE fractures and frontal sinus fractures if disruption of the cribiform plate is present.

Pupillary responses (CN II, III): Examine the pupil size and shape at rest. This can be difficult in patients with extensive orbital trauma as the eyelid swells rapidly and is difficult to open. Next, examine with a flashlight. Note the direct constriction of the illuminated pupil, as well as the consensual constriction of the opposite pupil. In an afferent pupillary defect there is decreased direct response in the affected eye. This can be demonstrated by moving the flashlight back and forth between the two eyes, with a lag of 2 to 3 seconds. The afferent defect becomes evident when the flashlight is moved from the normal to the affected eye because the affected eye will dilate in response to light. Brief pupillary oscillations of the stimulated pupil (hippus) are normal and should be distinguished from pathologic response. Finally, test the pupillary response to accommodation, by moving an object (e.g., finger) from far to near. The pupils should constrict. The direct response of the ipsilateral pupil is absent in lesions to the ipsilateral optic nerve, the pretectal area, the ipsilateral sympathetic nerves traveling with CN III, or the pupillary constrictor muscle of the iris. The consensual response is impaired (contralateral pupil illuminated) in lesions of the contralateral optic nerve, the pretectal area, the sympathetic nerves, or the pupillary constrictor of the iris. Accommodation is affected for the same reasons and in pathways from optic nerve to the visual cortex. Accommodation is spared in injury to the pretectal area.

Extraocular movements (CN III, IV, VI): Extraocular movement is readily checked by asking patients to look in all directions without moving their head and asking them if they experience any diplopia in any direction. Test “smooth pursuit” by slowly moving an object or finger up and down and side to side. Test convergence by asking the patient to fixate on an object that is moved toward a point between the eyes. During these tests, look closely for nystagmus and dysconjugate gaze.

Facial sensation and muscles of mastication (CN V): Test facial sensation using a soft object or finger in the forehead, cheek, and lower jaw line to capture all three branches of the nerve. Test the masseter muscles during jaw clench. In facial fractures, the most commonly affected nerve is the Vb branch, which may indicate maxillary, orbital, or zygomati-comaxillary complex (ZMC) fractures.

Muscles of facial expression and taste (CV II): Look for asymmetries in spontaneous facial expressions and blinking, smiling, and squinting. Taste testing is usually not performed. Facial weakness can be caused by lesions of upper motor neuron in the contralateral cortex or in descending nerve pathways (ipsilateral). Upper motor neurons to the upper face cross over to both facial nuclei so in intracranial injury or stroke, motor functions of the upper face remain intact. Lower motor neuron lesions typically cause weakness to the entire ipsilateral face.

Hearing and vestibular sense (CN VIII): Hearing and vestibular sense are seldom checked in the acute setting. Vestibular sense is typically not tested except in patients with vertigo.

Pallate elevation and gag reflex (CN IX, X): Perform an intraoral exam and observe palatal motion when the patient says “aaah.” Observe the gagging motion when the posterior pharynx is touched. The gag reflex is usually checked in patients with suspected brainstem pathology.

Sternocleidomastoid and trapezius muscles (CN XI): These muscles are examined by asking the patient to shrug the shoulders and turn the head from side to side. Of note is that bilateral upper motor neuron projections control the sternocleidomastoid, analogous to the bilateral CN VII projections controlling the upper face.

Tongue (CN XII): The tongue will deviate toward the weak side. Lesions of the motor cortex cause contralateral tongue weakness as opposed to lower motor lesions or lesions of the tongue muscles.

### SPECIFIC SIGNS AND SYMPTOMS OF FACIAL FRACTURES

#### Nasal Bones

The clinical features of an isolated nasal fracture are as follows:

- Tenderness over nasal bones
- Mobility
- Swelling
- Flattened or deviated nose
- Epistaxis
- Septal deviation
- Septal hematoma
- Mouth breathing

Due to the prominence of the nose, nasal injuries are fairly common and the nose is the most commonly fractured bone in the facial skeleton. Nasal fracture diagnosis is often a clinical, and not a radiologic, diagnosis. External nasal deformities are usually obvious during examination. Crepitus will distinguish recent trauma from a nasal deformity due to a previous injury. Septal hematoma must be ruled out in every patient. A septal hematoma forms between the septal cartilage and periosteum from which it gets its blood supply. It appears as edema and ecchymosis of the septum with narrowing of the nasal airway on speculum exam. Septal hematoma is treated with incision and drainage. Failure to treat can lead to a septal abscess, intracranial complications, or delayed saddle nose deformity due to cartilage loss.

Leakage of cerebrospinal fluid (CSF) indicates a fracture through the cribiform plate of the ethmoid bone. This potentially carries a risk of meningitis. There is controversy on the use of prophylactic antibiotics. Epistaxis is treated by packing the nose as discussed above. If this is not successful, an epistaxis catheter can be inserted to control bleeding from branches of the anterior ethmoidal artery. Treatment of most noncomminuted nasal fractures is closed reduction. Manipulation is required to restore an obstructed nasal airway and for restoration of facial cosmesis. The ideal timing for manipulative treatment varies. If reduction is not performed within the first few hours following injury, treatment is delayed 3 to 5 days for swelling to resolve. After a prolonged period (7-14 days), manipulation becomes increasingly difficult as the nasal bones will be

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Naso-orbito-ethmoid Fractures

The clinical features of a nasoethmoidal fracture are as follows:

- Flat nasal bridge with sparing of nasal complex and crepitation
- Saddle-shaped deformity of nose ("punched-in" look)
- Telecanthus (increased distance between the medial canthi)
- Circumorbital edema and ecchymosis ("raccoon eyes")
- Subconjunctival hemorrhage
- Epistaxis
- CSF rhinorrhea
- Supraorbital/supratrochlear nerve paresthesia

With true nasoethmoidal fractures, a CSF leak should be assumed even if not clinically evident. Classically, an increased intercanthal distance (greater than 35 mm) and depression of the nasal root are unequivocal clinical signs of traumatic telecanthus. Closed manipulation of naso-orbito-ethmoid injuries notoriously gives a poor result, with a high incidence of persistent or recurrent deformity postoperatively. The results of secondary surgery of this deformity are seldom satisfactory. ORIF, often with bone grafting to the nose, is usually necessary as the NOE complex is a very difficult area of the facial skeleton to reconstruct. Access to the nasoethmoidal region can be obtained through an existing laceration, if present, or through a coronal incision for adequate access to the frontal bone, nasal root, and orbits (11).

The evaluation of the stability of the medial canthal ligament forms an integral part of the clinical assessment. The clinical classification of status of the medial canthal ligament and its attachment to underlying bone can be classified according to Gruss et al. (12) or Markowitz et al. (13). The medial canthus must be stabilized usually by wiring to the opposite anterior lacrimal crest (transnasal canthopexy). If both canthal ligaments are detached, then the telecanthus can be addressed by means of wiring the two medial canthal ligaments to each other (transnally).

Orbital Blowout Fracture

The term orbital blowout fracture is reserved for a fracture of the bones of the orbit. This may involve the orbital floor, walls, or roof. The majority of cases involve the orbital floor and medial wall, as these areas comprise the thinnest bone of the orbit. An isolated orbital blowout fracture is usually secondary to a blunt blow. Smith and Regan demonstrated that when an object of slightly greater diameter than the orbital rim strikes the orbit and incompressible eyeball, a fracture results in the middle orbit likely due to increased intraorbital pressure (14). Often, the thin bone of the floor displaces downward into the maxillary antrum, remaining attached to the orbital periosteum as one fragment ("trap door"). The periorbital fat herniates through the defect, thereby interfering with the inferior rectus and inferior oblique muscles, which are contained within the same fascia sheath. This prevents upward movement and outward rotation of the eye and the patient experiences diplopia on upward gaze. This clinical finding should be distinguished from true "entrapment," which indicates impingement of the ocular muscles. Those patients who will present with pain, tenderness around the eye, swelling, and subjective diplopia in all outer fields of gaze. Painful eye movement is common with significant swelling and hemorrhage in the orbit, and restriction of eye motility and double vision are not necessarily an indication for surgical repair of the fracture. Ophthalmologic evaluation is advised if significant eye trauma is detected. If the patient is unresponsive, an afferent pupillary defect may uncover occult visual loss. If indicated clinically, tonometry may be used to assess intraocular pressure. This may serve as a baseline for serial examinations. Also, forced duction testing can be done to check extraocular movements. This is done by grasping the sclera in the fornix and mechanically moving the globe. To test inferior rectus entrapment, the globe is moved superiorly. Inhibition of this motion would indicate need for exploration. A computed tomography (CT) scan will determine the presence or absence and size of the fracture. Surgical repair of orbital fractures depends on symptoms and largely on the size of the fracture itself. Small fractures (less than 50% of the floor or roof), even if associated with double vision, can be observed for 1 to 2 weeks to assess if repair is indicated if symptoms do not resolve. Patients are instructed to avoid blowing their nose and to use nasal decongestants. Should symptoms persist and/or if the fracture is large, surgical intervention is required to return the orbital contents to their correct position and to restore orbital volume. This is done by placement of a graft in the orbital floor. Many different graft materials can be used but autologous bone remains the gold standard and may be required if the defect is large enough, especially in a young patient (15).

Complications of unrecognized orbital floor fracture are as follows:

- Posttraumatic persistent enophthalmos
- Hypoglobus (inferior displacement of the orbit)
- Persistent diplopia
- Lower eyelid retraction (ectropion) and scleral show
- Persistent edema of the lower eyelid

Zygoma Fractures

Clinical features of ZMC fractures are as follows:

- Swelling and bruising over the cheek/flattening
- Step-off deformity at the orbital rim
- Periorbital ecchymosis
- Subconjunctival hemorrhage
- Para-anesthesia of the infraorbital nerve
- Trismus and restricted lateral excursion
- Para-anesthesia of Z-facial/temporal nerves

The zygomaticomaxillary complex is both a functional and aesthetic unit of the facial skeleton and the prominent zygoma is the second most commonly fractured facial bone. Zygomatic fractures usually result from high-impact trauma. Leading causes of fractures include assault, motor vehicle or motorcycle accidents, sports injuries, and falls. The majority of zygomatic fractures occur in men in the third decade of life. The zygoma separates the orbit from the maxillary sinus and temporal fossa. Because the zygoma articulates superficially with
the maxilla, frontal, and temporal bones, zygomatic fractures in the past have been referred to as tripod or trimilar fractures. However, the fourth articulation with the sphenoid really makes it a quadripod fracture. The ZMC can be defined by two arcs: a vertical arc from the zygomaxillofrontal suture downward to the lateral antrum, and a horizontal arc from the zygomatic arch to the inferior orbital rim. The intersection of these two arcs defines the malar prominence (1). The zygoma itself is a relatively strong bone, and isolated fractures of the body of the zygoma are rare unless there is a direct blow to the zygomatic arch. Due to traction on the infraorbital nerve, patients often complain of upper lip/teeth numbness. Trismus may be present as the masseter muscle pulls the malar fragment down, which impinges on the mandible. Radiologic imaging remains an important step in the evaluation of orbital/zygomatic-maxillary fractures. CT scanning offers advantages over plain films that justify the increased cost. Important areas to evaluate on CT scanning include the butresses, the orbital walls, the zygomatic arch, the palate, and the mandibular condyles (16).

For the zygoma, timing of repair is 5 to 7 days postinjury to allow tissue edema and swelling to subside. After 10 days, masseter contracture may complicate closed reduction of the zygoma. If the zygomatic arch is minimally displaced and there is no comminution, the patient is a candidate for “simple” reduction. If there is moderate displacement or comminution of the maxillary wall, the maxilla will have to be platted for stability. For true ZMC fractures, ORIF is typically necessary at the lateral maxilla, the inferior orbital rim, the zygomaticofrontal (ZF) suture, the zygomatic arch, and commonly the orbital floor as well. Full access to the arch unfortunately requires a coronal approach (17).

The Midfacial Skeleton: LeFort Fractures

LeFort fractures tend to result from anterior forces. The fracture possibilities and combinations thereof are numerous; hence, classification schemes fail to describe them all. The original fracture patterns described by LeFort in 1901 are based on experimentally induced midface trauma. LeFort established that midface fractures tend to occur in reproducible patterns along weaker areas of the craniofacial skeleton. The LeFort I fracture essentially separates the lower maxilla, including the alveolar ridge and teeth, from the rest of the midface. The fracture classically travels through the inferior portion of the piriform aperture across the maxilla to the pterygoid fissure. This fracture pattern may occur as a single entity or in association with LeFort II and III fractures. The LeFort II fracture is a pyramidal fracture that includes the entire piriform aperture in the distracted midface. The fracture line includes the frontonasal suture, passes through the intersinusoidal orbit, and runs between the zygoma and maxilla for a larger area of dissociation. The LeFort III fracture is a suprazygomatic fracture through the lateral orbit. The fracture line extends from the dorsum of the nose and the cribiform plate along the medial and lateral wall of the orbit to the ZF suture line. This is also known as craniofacial dissociation as the bones of the midface are essentially completely disarticulated from the cranium (18).

Signs and Symptoms of LeFort Fractures

All complete LeFort fractures will create mobility of the maxilla, especially the upper alveolus (tooth-bearing portion of the maxilla). Hence, all will lead to subjective (and objective) malocclusion in varying degrees of severity. Intraorbital nerve paresthesia may be present. There can be palpable crepitations in the upper buccal sulcus from the fracture line. An intraoral hematoma or ecchymosis is likely. In LeFort II and III fractures, the nose is often involved and epistaxis common. In LeFort III fractures, this should be distinguished from CSF rhinorrhea. Periorbital ecchymosis and edema, subconjunctival hemorrhage, and visual disturbance occur only in LeFort III fractures.

MANAGEMENT OF MAXILLARY FRACTURES

Minimally displaced fractures can be clinically observed provided no malocclusion is present. The patient is allowed oral intake but only full liquid/soft foods as load bearing (chewing) may displace fracture fragments. Commminated fractures and fractures with malocclusion are treated with maxillomandibular fixation (MMF) and/or ORIF. Truly rigid fixation of the midface, unlike the mandible, is unattainable due to the thin bones and correspondingly thin plates.

For comminuted or displaced fractures, the status of the mandible is critical for management. If the mandible is intact, it serves as a guide for placing the upper dentition into occlusion. MMF is placed, and then the midface is treated with appropriate ORIF. Intraoperatively used MMF can be released after fracture fixation and the patient allowed range of motion (soft diet only). If the mandible is also fractured, the patient is placed in MMF for 2 to 3 weeks (19).

Evaluating the facial trauma patient can be challenging. The basics of ATLS courses apply to the facial trauma patient as well. Airway stabilization and securement may be difficult in extensive facial trauma with the possibility of basilar skull base injury where endotracheal intubation is relatively contraindicated and one must consider a surgical airway. Any patient who has sustained forces adequate to cause facial fractures must be assumed to have a cervical spine injury until proven otherwise. Epistaxis can be troublesome and hemodynamically significant. In the clinical evaluation of facial fractures, subjective data that the patient is able to provide offer clues to facial fracture diagnosis and include pain, malocclusion, numbness in portions of the face, trismus, and diplopia. Malocclusion is a very sensitive indicator of injury due to the high sensitivity of the periodontal ligaments. Numbness often indicates disruption or compression of a peripheral nerve. Trismus may result from mandibular trauma or from an impacted zygoma impinging on the temporals muscle. Diplopia may result from entrapment of the extraocular muscles or gross globe malposition. Of special note, monocular diplopia indicates an intrinsic globe problem and mandates prompt ophthalmologic evaluation.

Unfortunately, many trauma patients are obtunded or intoxicated and unable to provide any subjective information. Physical examination alone is inconclusive in the majority of cases. On physical examination the examiner should note presence