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Craniofacial and Skull Base Trauma

J. Timothy Katzen, MD, Reza Jarrahy, MD, Joseph B. Eby, MD, Ronald A. Mathiasen, MD, Daniel R. Margulies, MD, FACS, and Hrayr K. Shahinian, MD

Background: **Traumatic craniofacial and skull base injuries require a multidisciplinary team approach. Trauma physicians must evaluate carefully, triage properly, and maintain a high index of suspicion to improve survival and enhance functional recovery. Frequently, craniofacial and skull base injuries are overlooked while treating more life-threatening injuries. Unnoticed complex craniofacial and skull base fractures, cerebrospinal fluid fistulae, and cranial nerve injuries can result in blindness, diplopia, deafness, facial paralysis, or meningitis. Early recognition of specific craniofacial and skull base injury patterns can lead to identification of associated injuries and allow for more rapid and appropriate management.**

Conclusion: **Early detection and treatment of craniofacial and skull base traumatic injuries should lead to decreased morbidity and mortality. This review discusses the most common of these injuries, their possible complications, and treatment.**

Key Words: **Craniofacial trauma, Skull base trauma, Facial fractures, Temporal bone fractures, Anosmia, Diplopia Otorrhea, Rhinorrhea Cerebrospinal fluid leaks, Cranial nerve trauma, Mandible fractures Maxillary fractures, LeFort fractures, Zygomatic fractures, Orbital fractures.**

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In the United States in 1999, there were over 6 million automobile crashes. Over 2 million crashes resulted in injuries and over 37,000 deaths.[1] Over 75% of these injuries have craniofacial or cervical spine injuries.[2] With the advent of ever-increasing sophistication in computed tomographic (CT) imaging, trauma surgeons can rapidly diagnose small facial fractures and intracranial hemorrhages. However, despite imaging improvements and more thorough physical examination, subtle complex facial fractures with cerebrospinal fluid (CSF) leaks, temporal bone fractures, and cranial nerve injuries can remain undiagnosed. Unfortunately, delayed or missed diagnoses can lead to significant morbidity (blindness, diplopia, deafness, facial paralysis, and meningitis) or death. Greater awareness of potential cranial injuries is needed to facilitate more rapid diagnosis and appropriate treatment.

A careful history and physical examination are paramount for accurate diagnosis of craniofacial injury. After performing the primary survey outlined by Advanced Trauma Life Support, a more thorough secondary survey should proceed systematically. The clinical examination of the craniofacial skeleton begins with inspection for localized tenderness, numbness, bleeding, asymmetry, deformity, ecchymosis, periorbital edema, otorrhea, and rhinorrhea. All bony surfaces should be palpated, including the superior and inferior orbital rims, zygomatic arches, nose, maxilla, mandible, and both alveolar ridges. Even if the eye is swollen shut, both eyes should be examined closely; examination should include visual acuity and extraocular muscles. Midface stability should be appraised. Alveolar ridges and teeth should be examined for dental trauma and occlusion should be assessed.[3,4]

In the conscious and cooperative patient, a detailed cranial nerve (CN) examination should be performed. The optic nerve, CN II, is assessed by visual field acuity. Extraocular movements test the integrity of CN III, IV, and VI.[5] Hypoesthesia of the face suggests CN V injury. Facial nerve injury, CN VII, produces paresis or paralysis of the muscles of facial expression.

The cranial nerve examination of the comatose patient is slightly more difficult and relies on testing of brain stem reflexes.[6] In the comatose patient, assessing vision can be difficult; even with complete unilateral visual loss, pupils can remain equally reactive as long as the efferent pathway of CN III is intact.[7] The optic and oculomotor systems should be evaluated by the "swinging flashlight test." The test requires an intact afferent CN II pathway and an intact efferent CN III parasympathetic pathway.[7] Testing patients with unilateral afferent CN II damage reveals bilateral, equal pupillary constriction when light is directed toward the eye with vision. However, when light is directed toward the eye with diminished vision, bilateral pupils will dilate. The phenomenon is referred to as the Marcus Gunn pupil.[7] In the comatose patient, extraocular movements can be tested with the oculocephalic (or "doll's-eye") reflex. The corneal reflex consists of touching the cornea with a piece of cotton; afferent fibers of CN V send the message to the brain and CN VII responds by eyelid closure. CN VIII is assessed with the cold-water

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From the Department of Surgery, Division of Trauma Surgery (J.T.K., R.J., D.R.M.), and Skull Base Institute (R.B.E., R.A.M., H.K.S.), Cedars-Sinai Medical Center, Los Angeles, California.
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Address for reprints: Hrayr K. Shahinian, MD, Skull Base Institute, 8635 West Third Street, Suite 490W, Los Angeles, CA 90048; email: hrayr.shahinian@cshs.org.

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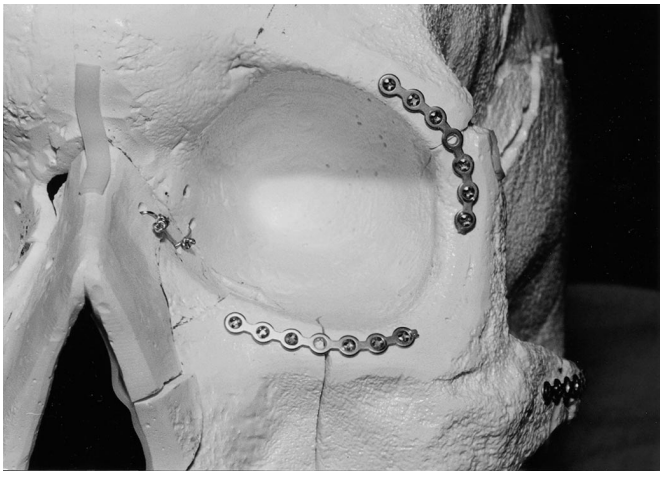

Fig. 1. *Internal fixation of the orbital rim.*

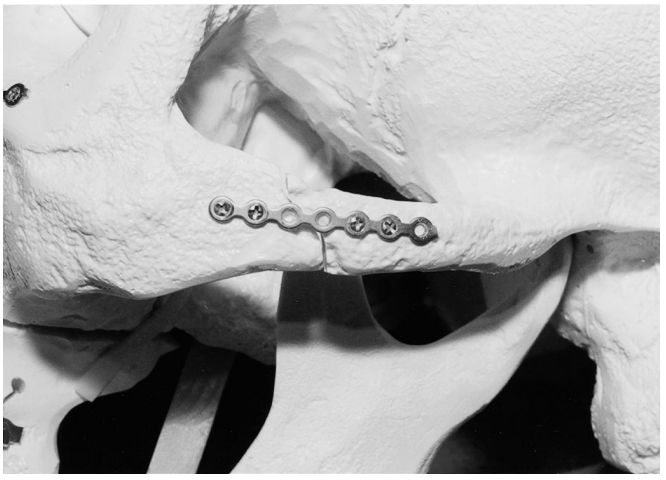

Fig. 2. *Internal fixation of the zygoma.*

caloric test, in which ice water is injected into the ear and elicits nystagmus. Testing of the gag reflex evaluates CN IX and CN X. After careful physical examination, the trauma surgeon should focus on specific areas of common craniofacial injuries.

CRANIOFACIAL FRACTURES

Orbital Fractures

Forceful impact to the skull can cause a fracture along the weak points of the orbit. The thinnest and weakest area of the orbit is the floor. Typically, the posteromedial region of the orbital floor is fractured. Often, orbital soft tissues herniate inferiorly into the maxillary sinus and become entrapped.[8] Entrapment of the inferior oblique or inferior rectus muscle can lead to diplopia and restriction of globe movement. In addition, the globe is displaced posteriorly and inferiorly, which causes enophthalmos and further diplopia.

The degree of orbital floor displacement is diagnosed accurately with axial and, especially, coronal CT scans of the orbit and facial bones. Surgical intervention is indicated when there is significant orbital floor disruption, persistent diplopia, entrapment, or enophthalmos.[9] Surgical access to orbital floor fractures involves a subciliary or transconjunctival incision in the lower eyelid.[9] The incarcerated orbital tissue is reduced and bony defects are reconstructed with a variety of allografts or autografts harvested from assorted sites.

Fractures of the superior, lateral, and inferior orbital rims may occur in isolation or in conjunction with other craniofacial fractures. Physical examination may reveal stepoffs in the line of the fracture. Cheek paresthesias are common because of inferior orbital rim fractures traumatizing the infraorbital nerve. Orbital fractures are repaired by realignment and fixation with miniplates (Fig. 1).[10]

Zygomatic Fractures

The zygoma forms the malar eminence, determines anterior and lateral cheek projection, and supports the lateral orbital wall and floor. The zygoma has four processes. Superiorly, the frontal process articulates with the frontal bone at the zygomaticofrontal suture. Inferiorly, the maxillary process articulates with the maxilla at the zygomaticomaxillary suture. Laterally, the temporal process joins the temporal bone, anterior to the external auditory canal. Medially, the orbital process articulates with the greater wing of the sphenoid.

Because of the projection of the zygoma, traumatic injury is common. Most zygomatic fractures occur in the arch and include a portion of the lateral orbital wall.[11,12] Zygomatic arch fractures cause depression of the cheek because of the pull of the masseteric muscle in an inferior, medial, and posterior vector.[12] Subconjunctival hematomas and infraorbital nerve paresthesias are so common that their absence makes the diagnosis of zygomatic fracture questionable.[13]

Many zygomatic fractures are minimally displaced and do not require surgical correction. Noncomminuted, posterior zygomatic arch fractures can be treated through a 1-cm temporal incision by simple reduction, without the need for internal fixation.[12] However, any other displaced zygomatic fracture requires open reduction and internal fixation.[13] Successful reduction relies on an accurate three-dimensional reduction with an emphasis on careful realignment of the lateral orbital wall.[14] Fractures are reduced and secured with miniplates (Fig. 2).

Maxillary Fractures

Maxillary fractures result from direct blows. Transmitted forces follow a predictable path along the thinner portions of the maxilla. The predictable patterns form the basis of the LeFort classification of maxillary fractures.[15] LeFort I fractures are the most caudal of maxillary fractures. LeFort I fractures begin in the lower margin of the piriform aperture and extend laterally above the roots of the teeth, through the anterior maxillary wall, and posterolaterally to involve the pterygoid processes (Fig. 3). LeFort II fractures are centrally

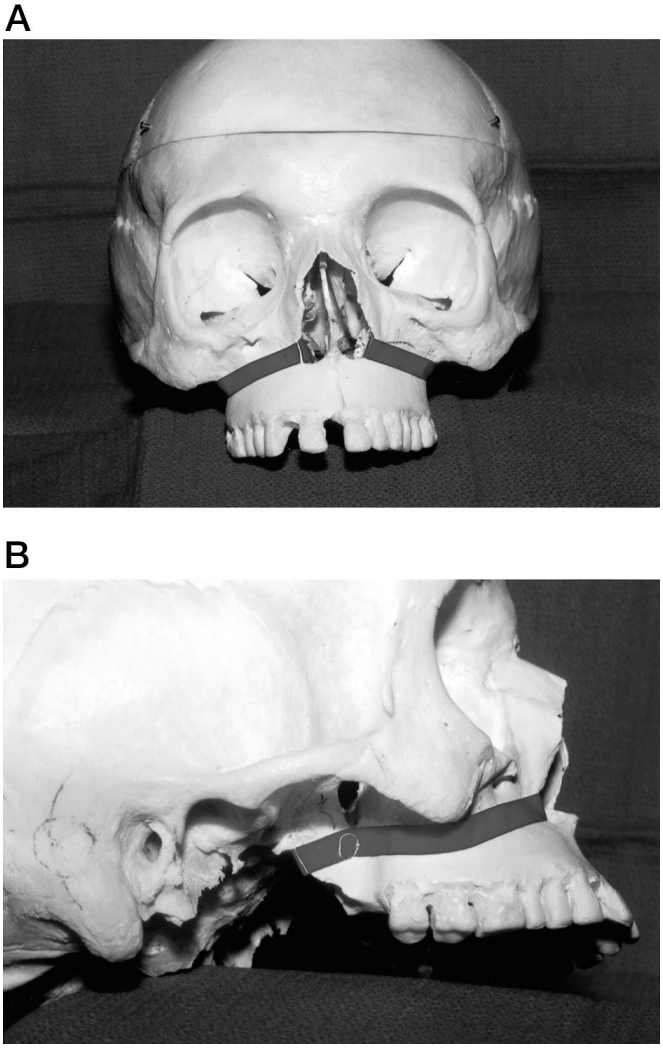

Fig. 3. *(A) LeFort I fractures begin in the lower margin of the piriform aperture and extend laterally above the roots of the teeth, and through the anterior maxillary wall (frontal view). (B) LeFort II fractures extend posterolaterally to involve the pterygoid processes (lateral view).*

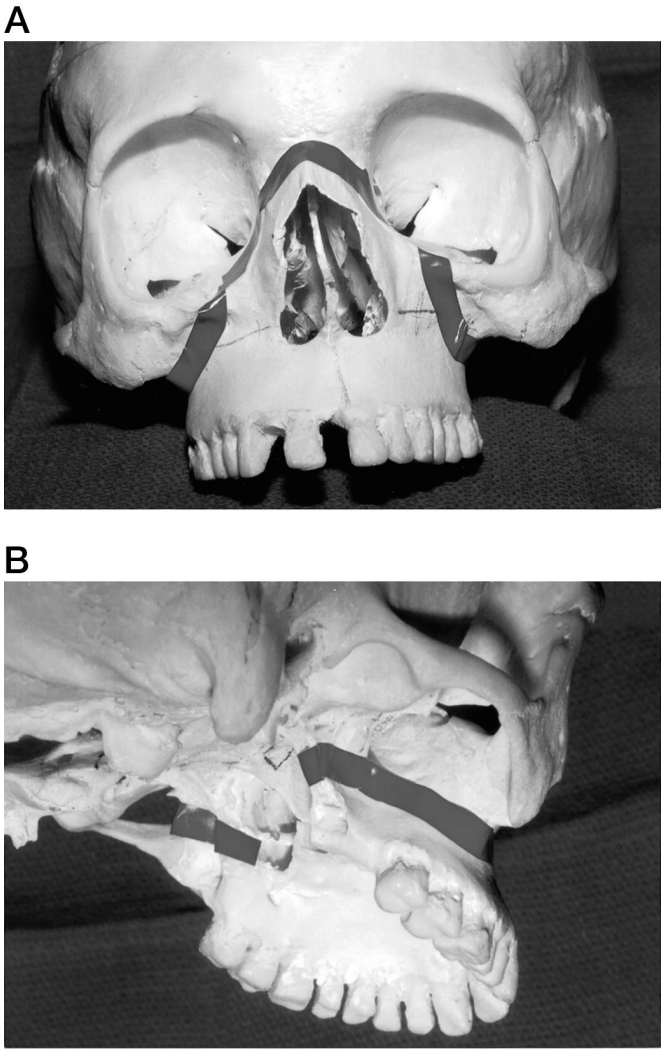

Fig. 4. *(A) LeFort II fractures begin at the nasal bridge, extend inferolaterally inside the medial orbit, exit through the infraorbital foramen, and travel through the zygomaticomaxillary suture (frontal view). (B) As LeFort I fractures, LeFort II fractures extend posterolaterally to involve the pterygoid processes (lateral view).*

more cephalad and, because of their shape, are called "pyramidal fractures." LeFort II fractures begin at the nasal bridge, extend inferolaterally inside the medial orbit, exit through the infraorbital foramen, travel through the zygomaticomaxillary suture, and extend posteriorly to involve the pterygoid processes (Fig. 4). LeFort III fractures begin medially as LeFort II fractures; however, instead of exiting the orbit over the infraorbital rim, they progress laterally along the entire orbital floor and extend to disrupt the zygomaticofrontal suture. LeFort III fractures result in complete craniofacial dysjunction because the facial bones and structures of the middle third of the face become totally separated from the cranium (Fig. 5).

The original facial fracture studies of Dr. Rene LeFort were performed on cadavers sustaining direct blows to the center of the face.[16] Because most facial trauma consists of blows from the side or slightly off center, ideal, symmetric LeFort I, II, and III patterns are rarely followed.[14] Most maxillary fractures are more comminuted on one side than the other. Thus, LeFort fractures may be seen in any combination: a right "hemi"-LeFort II fracture can coexist with a left "hemi"-LeFort III fracture (Fig. 6).[14]

Prolonged delay in the operative repair of maxillary fractures results in poor healing and should be discouraged.[12] One of the major goals in the treatment of LeFort fractures should be reestablishment of preinjury dental occlusion. Therefore, LeFort fracture patients should always be placed in intermaxillary fixation, before open reduction and internal fixation. A second but equally important goal in the treatment of LeFort fractures should be reconstruction of the orbital floor (see Orbital Fractures, above). A third goal should be

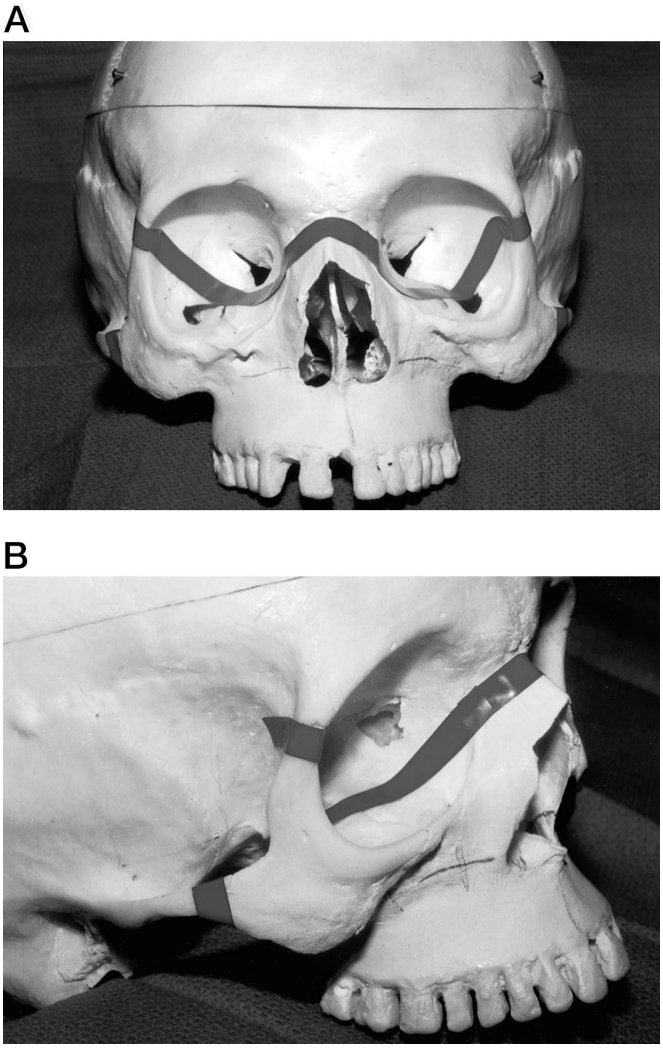

Fig. 5. *(A) LeFort III fractures begin medially as LeFort II fractures; however, instead of exiting the orbit over the infraorbital rim, they progress laterally along the entire orbital floor and extend to disrupt the zygomaticofrontal suture (frontal view). (B) LeFort III fractures result in craniofacial dysjunction (lateral view).*

reestablishment of the patient's facial height and projection; pretraumatic facial form can be achieved by accurate open reduction and internal fixation.

Mandibular Fractures

Along with the zygoma, the mandible is one of the most frequently fractured facial bones and constitutes approximately 20% of all facial fractures.[17–19] Areas of mandibular weakness are the most likely to fracture and include the mandibular neck, subcondylar region, and angle.[18] Because greater than 50% of mandibular fractures occur in two or more locations, a second fracture site should almost always be suspected when examining a patient.[18]

Presence of teeth, position of mandibular fracture, and pull of mandibular musculature all determine presenting

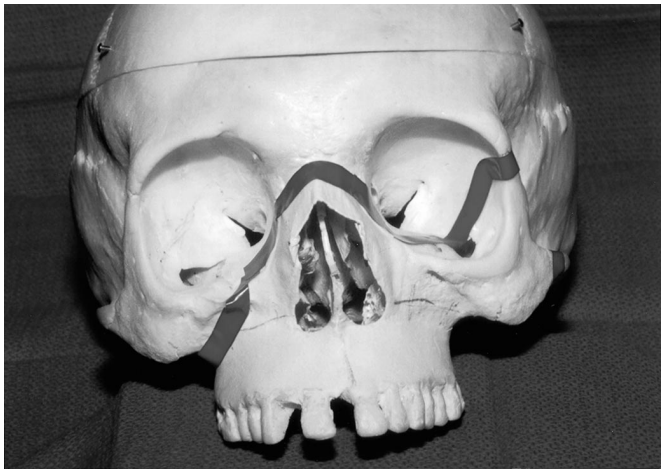

Fig. 6. *A right "hemi"-LeFort II fracture with a left "hemi"-LeFort III fracture.*

symptomatology. Mandibular fractures frequently present with malocclusion and asymmetry.[18] The most important goal in the treatment of mandibular fractures is to reestablish the patient's preinjury dental occlusion.[18] Most mandibular fractures require open reduction and internal fixation. Because of the bacterial load of the mouth, open mandibular fractures should be irrigated immediately, reduced, and fixated. Closed mandibular fractures should be openly reduced and internally fixated within 3 to 5 days after the injury, to allow for deceased edema and intraoperative bleeding. If there is any question as to the stability of a mandibular fracture, the patient should be left in intermaxillary fixation for 4 to 6 weeks to ensure proper bone healing.[18]

SKULL BASE FRACTURES

Five bones form the base of the skull. The bones include the orbital plate of the frontal bone, cribriform plate of the ethmoid bone, sphenoid bone, occipital bone, and the squamous and petrous portions of the temporal bone. Up to 24% of patients sustaining blunt head trauma have a skull base fracture.[20] Despite the clinical importance of skull base fractures, many are undiagnosed. Because of the complex anatomic relationships of the skull base, the fractures may damage critical neighboring structures, including cranial nerves, the internal carotid artery, and the cavernous sinus. The fractures may lacerate the dura and create a potential CSF fistula.

When a fracture of the skull base is suspected, insertion of a nasogastric tube should be avoided. The orogastric route is preferred, as there have been cases of intracranial nasogastric tube placement in the presence of cribriform plate fractures.[21]

Temporal Bone Fractures

Clinical signs of temporal bone fractures include blood in the external auditory canal, hemotympanum, ecchymosis

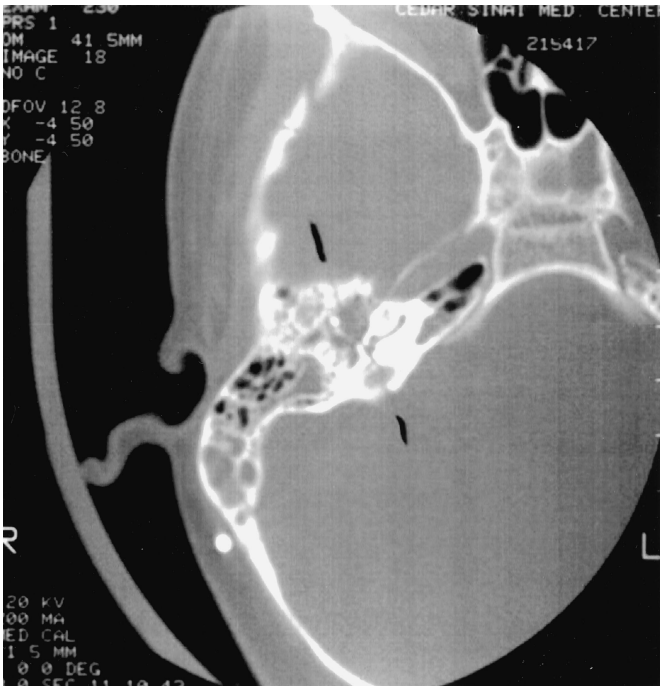

Fig. 7. *High-resolution temporal bone CT scan showing a transverse temporal bone fracture in a patient who was struck on the occiput with a blunt object.*

overlying the mastoid bone, otorrhea, hearing loss, vestibular dysfunction, and facial nerve paresis or paralysis. High-resolution non–contrast-enhanced CT scan should be performed in all suspected temporal bone injuries.[22] Coronal sections and three-dimensional reconstructions provide information about the facial nerve canal, carotid canal, and otic capsule.

Temporal bone fractures are classified according to their relationship to the long axis of the petrous pyramid. Although most temporal bone fractures are mixed, temporal bone fractures are classified as longitudinal or transverse.

Seventy percent to 90% of temporal bone fractures are longitudinal and occur after direct lateral blows to the temporoparietal skull.[22,23] These fractures usually begin in the weaker squamous portion of the temporal bone and course toward the carotid and jugular foramina. Usually, the tympanic membrane is torn and the middle ear ossicles are disrupted, resulting in a conductive hearing loss. Bleeding from the external auditory canal is common. Approximately 25% of patients have facial nerve injury, which usually occurs in the geniculate ganglion or facial canal.

Transverse temporal bone fractures are much more rare and occur after severe trauma to the occiput.[24] These fractures begin in the jugular foramen and course across the petrous pyramid, through the foramina spinosum and lacerum to the foramen magnum (Fig. 7). Approximately 50% of patients notice immediate facial paralysis from CN VII injury.[25] Unless corrected surgically, facial paralysis may be permanent. Typically, CN VII is injured in the internal auditory meatus or on the medial wall of the tympanic membrane. In addition, damage to the labyrinth, cochlea, or CN VIII can result in sensorineural hearing loss and vestibular dysfunction. Transverse temporal bone fractures often course through the otic capsule. Because the otic capsule heals by fibrous union rather than bony callus formation, patients have a lifelong risk for developing meningitis.[26]

Penetrating Temporal Bone Trauma

Penetrating trauma to the temporal bone usually results from self-inflicted gunshot wounds.[27] After initial stabilization, a complete vascular and neurologic evaluation should be performed. Vascular examination should include digital subtraction angiography with venous phasing or magnetic resonance angiography.[28] Complete neurologic examination should place, with special emphasis on cranial nerves examination. Because of the close proximity of vital structures, one neurologic deficit may point toward another injury. For example, vocal cord paralysis from an injured vagus nerve may be associated with a carotid artery or jugular vein injury.

SKULL BASE FRACTURE TREATMENT

In the absence of a CSF fistula, temporal bone fracture, facial paralysis, hearing loss, or blindness, the management of skull base fractures is nonoperative and expectant. Conservative treatment includes a 5-day course of intravenous antibiotics to allow potential dural tears to heal.[20]

Operative treatment is indicated for posttraumatic CSF fistulae with meningitis, transverse petrous fractures with otic capsule involvement, temporal bone fractures with complete facial paralysis, and ballistic injury to the temporal bone.[29] Treatment includes a subtotal petrosectomy. The operation consists of complete exenteration of temporal bone air cell tracts and obliteration of the eustachian tube.[26] After the injured structures are repaired (e.g., the facial nerve or carotid artery) or exenterated (e.g., the otic capsule), the resulting cavity is obliterated with an endogenous fat graft and temporalis muscle flap.

CSF FISTULAE

Approximately 20% of skull base fractures will develop a CSF fistula, with 80% occurring within 48 hours of injury.[30,31] Manifestations include rhinorrhea and otorrhea. The drainage is usually clear and nonmucoid and may be difficult to detect when mixed with blood. To facilitate the diagnosis of CSF leak, a few drops of the fluid are placed on a tissue paper. CSF has a more rapid diffusion pattern than blood, and when the discharge is mixed with blood, a larger, clearer CSF ring will surround the sanguineous central ring. The clinical finding is termed the "double-ring" sign. Alternatively, the fluid glucose concentration can be measured. Values should be compared with serum glucose levels, and quantities greater than 30 mg/dL are usually consistent with a CSF leak.[32] In addition, the fluid should be sent for beta-2-transferrin testing. Presence of beta-2-transferrin confirms a CSF leak.

Rhinorrhea

CSF draining from the nose results from fractures through the cribriform plate, ethmoid, sphenoid, petrous portion of the temporal bone, or orbital plate of the frontal bone.[33] Initially, patients are managed conservatively. Patients are maintained at total bed rest with the head of bed elevated, to reduce the flow of CSF drainage. If drainage has not ceased after 72 hours of conservative therapy, a lumbar drain should be inserted to drain 150 mL of CSF per day for 3 to 4 days. Diversion of CSF from the site of the dural tear facilitates spontaneous closure. Current data support placing patients on penicillin 1 to 2 million U/day in the presence of a CSF fistula.[34,35] Nasal and throat cultures should be taken, and antibiotics should be selected on culture results.

The CSF fistula is localized with CT scans using 3.0-mm coronal sections. Two other studies may help localize the fistula: an indium-111 DTPA or metrizamide CT cisternogram. An indium-111 cisternogram begins with the placement of cotton pledgets in the anterior and posterior roof of the nose, sphenoethmoidal recess, and middle meatus.[36] Indium-111 DTPA is introduced into the spinal subarachnoid space via lumbar puncture. The patient's head is flexed, causing an increase in intracranial pressure and thereby increasing the flow of CSF through the dural tear. The radioactivity of the cotton pledgets is measured and used as a guide to the site of the leak. A metrizamide CT cisternogram begins by introducing metrizamide into the lumbar subarachnoid space. Then, the patient undergoes a coronal CT scan. Contrast material will be seen in the paranasal sinuses near the fistulous tract (Fig. 8). An actively draining fistula is required for the technique. Because most fistulae drain only intermittently, false-negative studies are common.

After localizing the site of the CSF fistula, operative repair may be undertaken. However, there is no consensus regarding the timing of operative repair. Current recommendations for patients with an isolated CSF fistula include deferring surgery for at least 5 days.[20,31,37] Surgical intervention should be reserved for patients with meningitis, large defects with brain herniation into paranasal sinuses, pneumocephalus, or persistent CSF leak over 5 days.[31,37]

Recent advances in endoscopy allow for a minimally invasive, fully endoscopic transethmoidal or transsphenoidal approach to repair CSF fistulae.[38] The technique is best used to access leaks through the sphenoid and ethmoid sinuses and the sella turcica.[35]

Otorrhea

Drainage of CSF from the ear results when a fracture of the petrous portion of the temporal bone both tears the dura mater and perforates the tympanic membrane. CSF drainage can also occur from fractured mastoid air cells causing a laceration of the external auditory canal.

As with rhinorrhea, the initial management of otorrhea is conservative. The patient should be positioned to minimize fistula drainage. Irrigation and probing of the ear increase the risk of meningitis and should be discouraged. Most patients will stop draining spontaneously within several days. Rarely, otorrhea persists beyond 5 to 7 days. When otorrhea lasts beyond 7 days, high-resolution CT scanning with coronal sectioning should be performed to localize the site of the fracture. Detailed auditory and vestibular testing should be performed at 6 to 8 weeks to diagnose abnormalities.

Operative intervention consists of a middle or posterior fossa craniotomy, fashioning a bone flap to expose the dura overlying the petrous bone.[35] Primary repair is attempted, but if not possible, a graft of pericardium or fascia lata is used. Occasionally, endogenous fat or muscle is used to pack the defect.

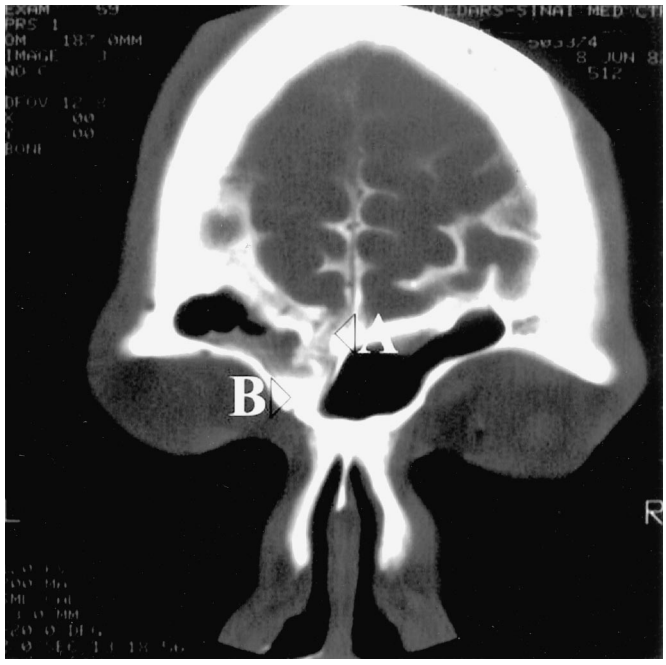

Fig. 8. *Metrizamide CT cisternogram localizing CSF leak (*A*) to left frontal sinus in a patient who was stabbed in the left orbit. Contrast material in seen in the frontal sinus (*B*).*

CRANIAL NERVE INJURIES

On exiting the skull, cranial nerves are especially prone to damage. Skull base fractures particularly predispose patients to cranial nerve damage. Table 1 lists the 12 cranial nerves and the common neurologic deficits after injury.

Olfactory Nerve (CN I) Injury

Injury to the olfactory nerve results in anosmia. Typically, anosmia occurs from anterior fossa floor fractures. In almost half of the cases, a patient's sense of smell returns in several months.[39] In addition to a CT scan, workup may require an olfactory electroencephalogram. Most of CN I injuries can be managed conservatively.

Table 1 **The 12 Cranial Nerves and the Respective Deficits after Injury**

Nerve	Deficit
(I) Olfactory	Anosmia
(II) Optic	Blindness; visual field deficits
(III) Oculomotor	Pupillary enlargement; diplopia (paralysis of extraocular muscles)
(IV) Trochlear	Paralysis of superior oblique muscle causing diplopia
(V) Trigeminal	Loss of corneal reflex; facial numbness; weak muscles of mastication
(VI) Abducens	Inability to move the eye laterally causing diplopia on lateral gaze
(VII) Facial	Paralysis of muscles of facial expression
(VIII) Vestibulocochlear	Vestibular dysfunction; nystagmus; hearing loss
(IX) Glossopharyngeal	Loss of gag reflex; dysphagia
(X) Vagus	Vocal cord paralysis; tachycardia
(XI) Spinal accessory	Paralysis of sternocleidomastoid
(XII) Hypoglossal	Ipsilateral tongue deviation and atrophy

Optic Nerve (CN II) Injury

Optic nerve injury can result in blindness. Optic nerve injuries are usually caused by isolated fractures of the optic canal or orbit or extensions of skull base fractures.[40] Skull base fractures involving the sphenoid body and extending through the sella turcica and pars petrosa can damage the optic chiasm, producing blindness or bitemporal hemianopsia.[41]

The optic nerve is unique and not a true cranial nerve. The optic nerve is a direct extension of the brain and, thus, the axons of the optic nerve do not regenerate. Therefore, prognosis is poor after optic nerve injury. With complete optic nerve transection distal to the optic chiasm, there is monocular blindness, a dilated pupil, and an absent pupillary reflex.[42]

Results of surgical decompression of the optic nerve in the optic canal are similar to rates of spontaneous recovery.[43] Surgical decompression is reserved for cases of a narrowed optic canal, bony fragment in the optic canal, or deterioration of previously good vision after head trauma.[42]

When indicated, acute decompression is conducted through a bifrontal craniotomy. In addition, optic chiasm decompression may be accomplished using an endoscopic transsphenoidal approach.

Oculomotor Nerve (CN III) Injury

Injury to CN III is typically from a direct, frontal blow. Trauma stretches and contuses CN III on entry into the brain, at the posterior aspect of the cavernous sinus. Clinically, patients complain of diplopia resulting from impaired extraocular movements. Examination reveals an ipsilateral dilated pupil and an inability to move the eye medially, superiorly, or inferiorly.

Fractures through the superior orbital fissure cause damage to CN III, IV, VI, and the ophthalmic division of CN V.[44] The clinical result is the superior orbital fissure syndrome. Patients may present with paralysis of the levator, superior rectus, inferior rectus, inferior oblique, superior oblique, and lateral oblique muscles and anesthesia of the brow, upper lid, and forehead. When superior orbital fissure syndrome symptoms are accompanied by blindness, the complex is called the orbital apex syndrome and indicates involvement of the optic foramen.

Treatment of ocular nerve palsies consists of wearing a patch over the affected eye. Spontaneous recovery of ocular movement usually occurs in 4 to 6 weeks.

Trochlear Nerve (CN IV) Injury

The trochlear nerve is the least frequently injured cranial nerve. Damage to CN IV results from stretching near the exit from the dorsal midbrain. Lateral rectus weakness results. Treatment is conservative and involves an eye patch to prevent diplopia. Function usually returns by 4 to 6 weeks.

Trigeminal Nerve (CN V) Injury

Injury to the trigeminal nerve causes sensory deficits to the face. The three branches of the trigeminal nerve are the supraorbital nerve (V1), the maxillary branch (V2), and the mandibular branch (V3).

V1 is damaged most commonly. The branch is particularly susceptible to injury at the supraorbital notch. Complete transection may result in anesthesia of the nose, eyebrow, and forehead.[45] Typically, V2 is injured by maxillofacial fractures with resultant sensory defects of the ipsilateral cheek, upper lip, gums, and hard palate. Typically, V3 is injured by mandibular fractures and results in anesthesia of the chin.

Centrally, the trigeminal ganglion can be damaged by a penetrating head injury. This is associated with CN III, CN IV, or carotid-cavernous fistula.[46] The nerve is especially vulnerable coursing through the dura, proximal to Meckel's cave.

Incomplete transection or scarring of the branches of CN V may result in intractable facial pain and neuroma formation. Corticosteroid injections, endoscopic decompression, or endoscopic division may be required for relief of symptoms.[47]

Abducens Nerve (CN VI) Injury

Injury to CN VI results from fractures in the clivus. Vertical movement of the brain stem during trauma may stretch or avulse the nerve on leaving the pons (Fig. 9). As mentioned above, CN VI may be damaged in the superior orbital fissure and is classically accompanied by CN III and CN IV palsies.

The diagnosis of abducens palsy in the unconscious patient can be made when the affected eye fails to abduct as the head is passively turned away from the side of injury.

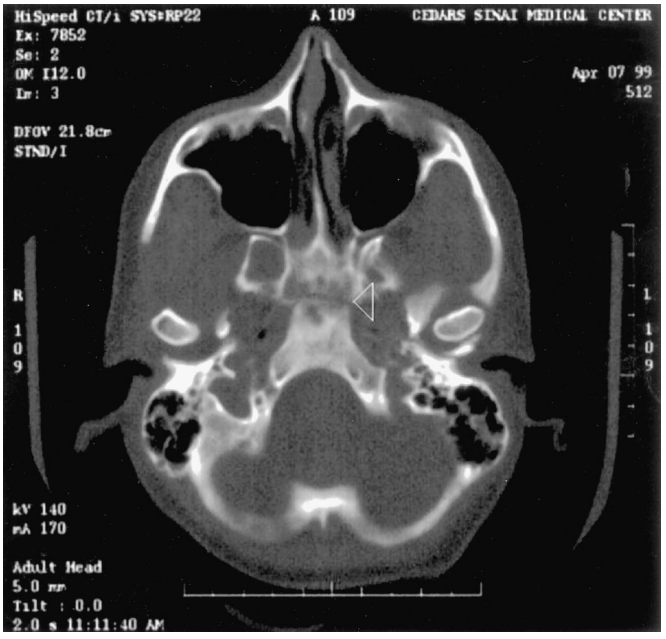

Fig. 9. *CT scan showing a fracture of the clivus after blunt head trauma. The patient presented with CN III, VI, and VII palsies.*

Treatment is conservative and most cases of abducens nerve injury recover spontaneously after 4 weeks.

Facial Nerve (CN VII) Injury

Temporal bone fractures are the most common cause of facial nerve injuries.[22,25] Fifty percent of patients with transverse fractures of the temporal bone and 25% of patients with longitudinal fractures will have associated facial nerve injury causing ipsilateral facial paralysis.[48]

Although facial nerve injury within the temporal bone is the most common site, CN VII can be damaged anywhere along its course.[49] In transverse temporal fractures, the nerve may be injured at the internal auditory meatus or in the horizontal portion of the fallopian canal. In longitudinal temporal fractures, the nerve may be damaged at the geniculate ganglion.

After a detailed clinical examination, all patients suspected to have a facial nerve injury should undergo CT scanning and be evaluated with transcutaneous nerve excitability tests and electroneurography. Transcutaneous nerve excitability tests predict irreversible nerve injury by comparing the normal and injured side. When the difference is greater than 3.5 mA, surgical intervention is usually required.[50] Operative intervention is also indicated when there is complete, immediate, facial paralysis with greater than 90% denervation documented by electroneurography.[51]

Microsurgical techniques are used to explore, decompress, or directly repair the nerve. A subtotal petrosectomy approach is used. The severed nerve fascicles are sutured together under a microscope. Most patients with traumatic facial paralysis recover well without surgical intervention; however, the eye must be guarded against exposure keratitis during the recovery period.[25]

Vestibulocochlear Nerve (CN VIII) Injury

Damage to CN VIII is common after transverse fractures of the temporal bone from frontal or occipital impact. Cochlear and vestibular damage can result with deafness and labyrinthine dysfunction. In addition, fractures involving the otic capsule can lead to total degeneration of the cochlear and vestibular organs.

A baseline neurotologic evaluation should be performed in all patients with head injury to detect hearing loss and vestibular dysfunction. Electronystagmography can be used to assess labyrinthine function. Audiometry and brain stem evoked potentials are used to evaluate hearing loss.

Previously, the prognosis of sensorineural hearing loss was poor. However, recent advances in cochlear implantation have allowed a return to speech understanding in 84% of patients after an intensive rehabilitation program.[52]

Glossopharyngeal (CN IX), Vagus (CN X), Spinal Accessory (CN XI), and Hypoglossal (CN XII) Nerve Injury

The glossopharyngeal, vagus, and spinal accessory nerves exit the skull base in the jugular foramen. The hypoglossal nerve passes though the hypoglossal foramen just medial to the jugular foramen.

Injury to glossopharyngeal nerve produces dysphagia and loss of gag reflex. Vagus nerve injury results in paralysis of the ipsilateral vocal cord and resultant voice hoarseness. Spinal accessory nerve injury results in paralysis of the sternocleidomastoid muscle and weakness of the trapezius muscle; the result is weakness in contralateral head rotation and shoulder elevation. Hypoglossal nerve injury causes hemiatrophy of the tongue and ipsilateral tongue deviation. Treatment is usually supportive, using physical, occupational, and speech therapy.

CONCLUSION

Because of the proximity of vital structures in the craniofacial and skull base region, localized trauma can result in unrecognized injuries. CSF fistulae and cranial nerve injuries in complex fractures can carry devastating consequences. Accordingly, appropriate surgical referral should be made whenever the injuries are suspected. Recent advances in skull base approaches have allowed for highly successful surgical correction of these potentially devastating injuries.

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