

Chapter 35

CRANIAL NERVE INJURIES

JOSE E. BARRERA, MD, FACS*

INTRODUCTION

TRIGEMINAL NERVE INJURY

FACIAL NERVE INJURY

Management of Facial Nerve Injuries

Other Cranial Nerve Injuries

Associated Nerve Injuries From Neck Trauma

CASE PRESENTATIONS

Case Study 35-1

Case Study 35-2

**Lieutenant Colonel, Medical Corps, US Air Force; Chairman, Department of Otolaryngology, San Antonio Military Medical Center, 3551 Roger Brooke Drive, Joint Base San Antonio, Fort Sam Houston, Texas 78234-5004; Associate Professor of Surgery, Uniformed Services University of the Health Sciences*

INTRODUCTION

The literature is devoid of reports of cranial nerve injuries due to wartime trauma from Operation Iraqi Freedom and Operation Enduring Freedom. Cranial nerve injuries have been reported to result from temporal bone trauma, blunt trauma, and penetrating trauma. The mechanism can be either low-velocity or high-velocity in nature.

The traumatic cranial nerve injuries that are potentially repairable involve the

- trigeminal nerve (cranial nerve V),
- facial nerve (cranial nerve VII), and
- recurrent laryngeal nerve (segment of cranial nerve X).

In addition, blunt and penetrating injuries to the neck can be associated with injury at the jugular foramen involving cranial nerves IX to XI. Injury to cranial nerve XII can also occur in these injuries. Methods of repair for cranial nerve injuries include direct suture repair, cable or autogenous nerve grafting, entubulization of the nerve, nerve decompression, and microneuronal anastomosis. Neuronal repair of cranial nerve injuries depends on the nature of the injury, timing of the injury, and available expertise. Low-velocity injuries may cause crushing or laceration of these nerves, whereas high-velocity injuries often result in disruptive nerve transections from direct trauma and cavitation.

TRIGEMINAL NERVE INJURY

Evidence-based medicine should be applied when considering repair of cranial nerves. The best available evidence shows that the optimal results are obtained from direct, tension-free anastomosis of nerve endings. In the delayed setting in a patient with a nerve deficit, this is not always possible. More often, the patient presents with a sensory or motor deficit that is weeks to months old from direct trauma. The first step is to define the patient's clinical problem, thus assessing if autogenous nerve grafting or using a conduit to reconstruct the gap is appropriate. Weigh-

ing the evidence, MacKinnon and Dellon¹ developed a grading scale for sensory nerve recovery. The Medical Research Council (MRC) Scale demonstrates that patients with peripheral nerve deficits who have an MRC score of S3 or higher were found to have useful sensory recovery. Management of trigeminal nerve injuries is challenging because the appropriate method and timing of repair are controversial. Data on return of sensory function after inferior alveolar or lingual nerve injury are lacking. Consequently, using data derived from upper extremity injuries, patients

TABLE 35-1

HOUSE BRACKMANN GRADING SCALE FOR FACIAL NERVE INJURIES

Grade	Appearance	Forehead	Eye	Mouth	Synkinesis
I	Normal	Normal	Normal	Normal	Normal
II	Slight weakness Normal resting tone	Moderate-to-good movement	Complete closure Minimal effort	Slight asymmetry	Synkinesis barely noticeable Contracture or spasm absent
III	Nondisfiguring weakness Normal resting tone	Slight-to-moderate movement	Complete closure Maximal effort	Slight weakness Maximal effort	Obvious, but not disfiguring synkinesis Mass movement or spasm present
IV	Disfiguring weakness Normal resting tone	None	Incomplete closure	Asymmetric with maximal effort	Severe synkinesis, mass movement, or spasm
V	Minimal movement Asymmetric resting tone	None	Incomplete closure	Slight movement	Synkinesis, contracture, and spasm usually absent
VI	Asymmetric	None	None	None	No synkinesis, contracture, or spasm

treated within 1 year of injury with autogenous nerve grafting experience an overall 85% to 94% return of functional sensation and an MRC greater than or equal to S3. Inferior alveolar nerve repair has been reported to show a 33% to 75% functional return of sensation with immediate repair and a 20% functional return with delayed repair.² Methods of repair for cranial nerve include tubulization of cranial nerve deficits using autogenous grafts and alloplastic con-

duits. Autogenous vein grafts to reconstruct nerve defects ranging from 1.8 to 3.4 cm resulted in 66% to 90% functional return of sensation when grafting occurred immediately or within 1 year. One report shows the use of a polyglycolic acid conduit for an inferior alveolar nerve injury at 16 months postinjury without functional return of sensation. No other reports associated with the trigeminal nerve show return of function using alloplastic conduits.²

FACIAL NERVE INJURY

Facial nerve injuries are described based on the segmental pattern of where the site of injury occurred. Figure 35-1 describes the interaction of preganglionic parasympathetic afferent and efferent nerve fibers and how they relate to the location of the segment of the facial and trigeminal nerves. Therefore, injuries proximal to this segment would affect function at that innervation level.

Facial nerve deficits are described according to the House-Brackmann (HB) Scale (Table 35-1):

- HB I = normal examination in facial nerve function,
- HB II = minimal effort with eye closure,
- HB III = maximal effort with eye closure,
- HB IV = unable to close eyes,
- HB V = facial asymmetry at rest, and
- HB VI = complete paralysis.

Complex facial nerve injuries described by the HB grade may be difficult to assess in patients with HB III and HB IV due to synkinesis of regenerating nerve fibers. Therefore, a segmental approach may be warranted to describe the functional result at each level of peripheral nerve arborization.

Stage 1 or neurapraxia is consistent with a conduction block with complete recovery of nerve function expected. Wallerian degeneration occurs with axonotmesis or neurotmesis, but not with neurapraxia. Axonotmesis has a better prognosis than neurotmesis because the nerve can regenerate through the intact neural tubule at 1 mm/day. Nerve function tests can only differentiate between neurapraxia and Wallerian degeneration, but cannot identify the type of Wallerian degeneration. With complete transection, 100% Wallerian degeneration occurs in 3 to 5 days. In chronic conditions, such as facial nerve neuromas, individual nerve fibers undergo simultaneous degeneration and regeneration. Regenerating nerve fibers conduct at differing rates, producing dyssynchrony or synkinesis.

The most common diagnostic test to perform to evaluate facial nerve function is electroneurography

(ENoG). ENoG measures the maximal electrically evoked stimulus and measures the amplitude of facial muscle compound action potentials. It is useful between days 3 and 21 after complete loss of facial nerve function. The response is evaluated by comparing the peak-to-peak amplitude of the maximal response for the two sides of the face. An excellent prognosis is seen when the decline in compound action potential does not reach 90%. The Nerve Excitability Test stimulates the extratemporal portion of the facial nerve with a small-amplitude, pulsed direct current. The face is observed for the lowest current to produce a twitch. Threshold difference between the two sides is obtained. It is not recommended to test until Wallerian degeneration has occurred (3 to 4 days after injury) and is not useful after 3 weeks postinjury. The Maximal Stimulation Test shows the difference between the strength and amount of contraction of the facial musculature caused by supramaximal electrical stimulus.

The electromyogram (EMG) measures muscle action potentials caused by spontaneous and voluntary activity. Loss of voluntary motor unit potentials within 3 days postinjury suggests a poor prognosis. Denervation potentials are seen 10 days or longer after onset of palsy. However, retention of voluntary motor action potentials past the seventh day suggests that complete degeneration will not occur. Polyphasic action potentials are seen 4 to 6 weeks after injury and precede clinically detectable recovery by weeks. Absence of polyphasic potential 15 to 18 months after repair indicates failure of repair. Other diagnostic tests include Schirmer's test wherein excessive lacrimation isolates lesions to the greater superficial petrosal nerve that suggests the site of injury is proximal to the geniculate ganglion. The stapedial reflex may show an absent ipsilateral response implying lesions proximal to the second genu. The presence of a stapedial reflex suggests non-Bell's etiology for the facial nerve paralysis.

In the delayed posttraumatic setting, the radiological workup includes a gadolinium-enhanced magnetic resonance imaging (MRI) from the internal auditory canal that examines the facial nerve coursing periph-

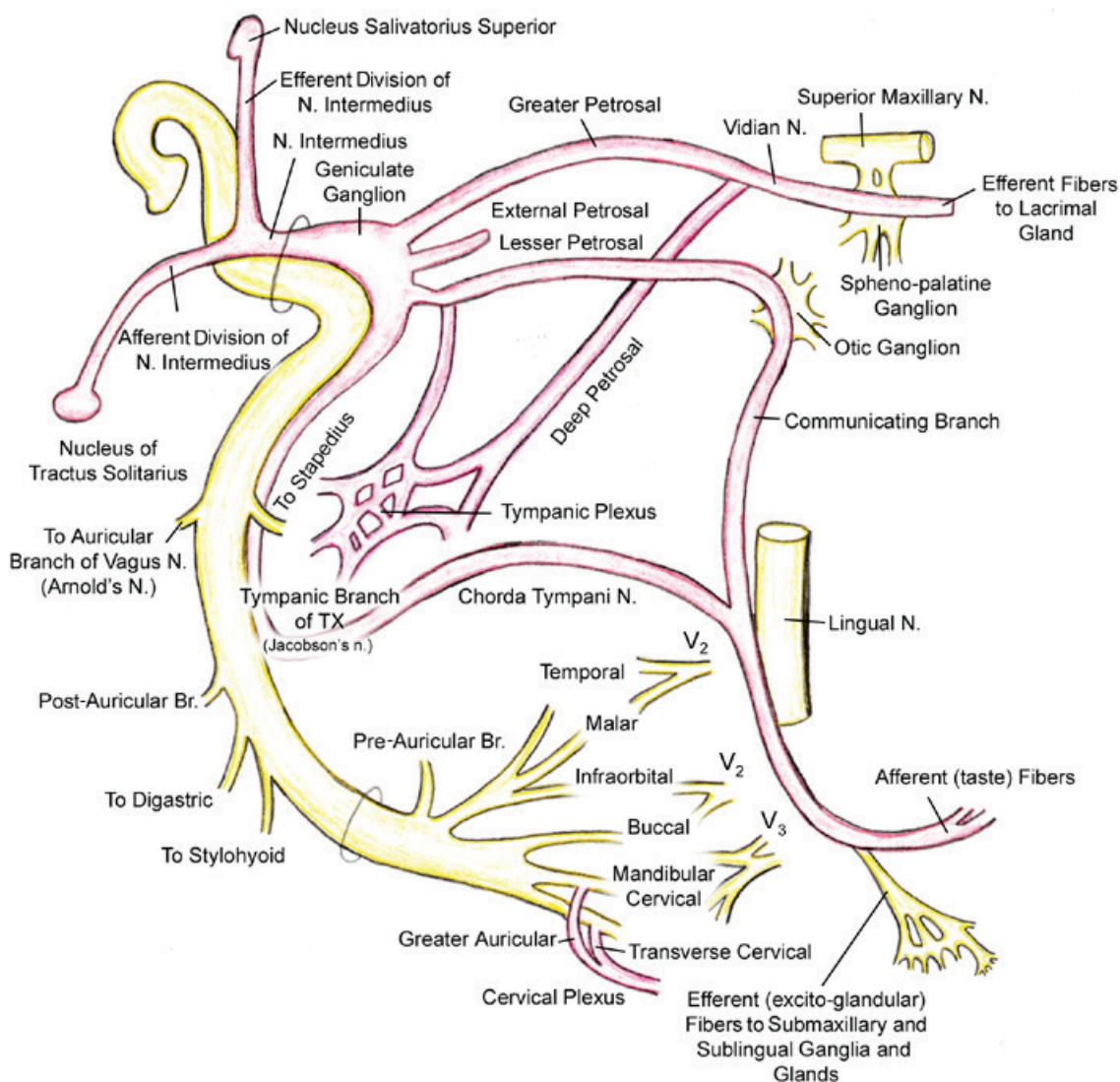


Figure 35-1. Anatomy of the preganglionic parasympathetic, afferent, and efferent nerve fibers as they relate to cranial nerves V and VII.

Br.: branch; N./n.: nerve; TX: trigeminal nerve; V2: second division trigeminal nerve; V3: third division trigeminal nerve

erally out of the stylomastoid foramen. Enhancement of the facial nerve is common in healthy persons that may cloud evaluation of the MRI. It is normal to see facial nerve enhancement in the greater superficial petrosal nerve, labyrinthine, tympanic, and mastoid segments, but it is abnormal in the cisternal, meatal, or extracranial portions of the facial nerve. Moreso, enhancement distal to the meatal and labyrinthine segments is indicative of Bell's palsy. Computed tomography (CT) is also used to evaluate trauma; determine the site of injury, the extent of intracranial, temporal bone, and extracranial involvement; and establish the severity of injury.

Injury to the temporal bone commonly occurs in head injuries. Approximately 4% to 30% of head injuries involve a fracture of the cranial base, including 18% to 40% with temporal bone involvement. The more common sequelae of a temporal bone fracture include

- injury to the facial nerve with facial paresis or paralysis;
- disturbance of the cochleovestibular apparatus with associated sensorineural hearing loss, conductive hearing loss, balance disturbance, tinnitus, and vertigo; and

- cerebrospinal fluid leak through the fracture lines.

Based on historical findings and more recent analysis of temporal bone fracture patterns, temporal bone fractures are 82% longitudinal, 11% transverse, and 7% mixed. Injury to the bony facial canal was shown in 44% of longitudinal fractures and in 64% of transverse fractures. In cases of longitudinal fracture, most facial nerve injuries occurred in the region of the genu, and transection of the nerve was rarely seen. In contrast, with transverse fractures, the nerve injury predominantly occurred in the labyrinthine portion and was usually associated with complete transection of the facial nerve. Ossicular chain injuries consisted of dislocation at the incudomalleolar joint in 51% of patients and at the incudostapedial joint in 57% of patients, the malleus fracture in 8% of patients, and the stapes fracture in 17% of patients.³ Temporal bone fractures require tremendous forces to produce a fracture, and patients frequently have

- multiple injuries, with associated nontemporal skull fractures (47%);
- maxillofacial fractures (21%); and
- orthopaedic injuries outside the head and neck (16%).⁴

Strict adherence to Advanced Trauma Life Support protocols is critical in treating these patients, given the multiple injuries that are often encountered when evaluating patients at the Role 4 setting. Many patients present with intracranial injury. In a series of 115 patients with facial paralysis secondary to temporal bone fracture, 33% presented with a Glasgow Coma Scale (GCS) score of <7.⁵ In another series, the mean GCS was 12 (range: 3–15), with 49% of patients showing mental status changes and 7% having limb paralysis.⁴ Despite improved care at the immediate field hospital and Role 3 staging facility, mortality related to neurological sequelae in this population still runs as high as 10%. For those who do survive, these severe injuries frequently have significant long-term consequences, with up to 16% of patients requiring institutional care beyond the initial acute management period.³

Management of Facial Nerve Injuries

The reported incidence of facial nerve paralysis after temporal bone fracture varies according to fracture pattern. Using the traditional classification system, facial nerve injury occurs in 10% to 25% of longitudinal fractures and in 38% to 50% of transverse fractures. The management of facial nerve paralysis depends

on the timing of paralysis relative to the injury. Cases involving immediate-onset paralysis are traditionally managed with surgical exploration after imaging, and electrical studies indicate a need for nerve decompression or repair. Cases in which the timing of onset cannot be determined are best considered part of the immediate-onset group.^{6–8} Patients who have delayed-onset or incomplete paralysis are typically treated with high-dose corticosteroids, with further intervention based on results of electrodiagnostic testing or imaging. Steroid treatment typically begins with 1 mg/kg/day of prednisone or equivalent corticosteroid for 1 to 3 weeks followed by a taper.⁸ The delay between onset and recovery in spontaneously recovering patients ranges from 1 day to 1 year, with 59% recovery by 1 month and 88% recovery by 3 months.⁶ Patients who have delayed-onset paralysis have an excellent prognosis. In two series, 84% to 93% of patients who had delayed-onset paralysis met the criteria for conservative treatment. In both series, 100% of these patients recovered to HB I or HB II. Further intervention in cases of immediate or uncertain timing of paralysis onset is based on the results of electrodiagnostic testing, including ENoG and electromyography. Because Wallerian degeneration is incomplete in the first 2 weeks after injury, it is preferable to wait until postinjury day 10 to perform an ENoG or EMG to improve test reliability.⁸ In cases with complete degeneration, testing will often show fibrillation potentials and no response after stimulation. If ENoG shows absent responses, EMG is indicated because ongoing degeneration and regeneration can cause dyssynchrony of neural input and phase cancellation. In cases of neurapraxia, testing will usually show an absence of voluntary action potentials, but a synchronized evoked response can be observed on nerve stimulation testing.⁸ Many authors agree that the threshold for surgical intervention is reached when 90% or greater degeneration is seen on ENoG, with some investigators reporting this as the key indicator for surgery regardless of timing of paralysis onset. Others base surgical intervention on the correlation between electromyography findings and high-resolution CT⁸ or on the documented complete immediate paralysis alone.^{6,8}

Despite earlier reports, nerve exploration has been encouraging in appropriate patients who have facial paralysis related to temporal bone fracture. At 2 years follow-up, 94% to 100% of patients experienced at least an HB III recovery, 45% had an HB I recovery, and no patients had worse than an HB IV recovery.⁸ In another series of 11 operated nerves, 5 recovered to HB I, 4 to HB II, and 2 to HB III.⁹ In a subset analysis, 78% of patients who underwent nerve suturing recovered to at least HB III. For patients pre-

senting with delayed and persistent facial paralyses weeks to months after the initial injury period, late decompression surgery can be beneficial, as shown in seven of nine patients undergoing decompression as late as 3 months who recovered to HB I or HB II when followed up for at least 1 year.¹⁰ Most patients who have facial paralysis or paresis that does not completely recover in the initial 3 to 4 months are observed for at least 1 year. Preserving eye function is paramount during this time, and attention must be given to proper eye protection with gold weight lid implants or tarsorrhaphy for the nonclosing eye. After 1 year of observation to allow maximal spontaneous recovery, reanimation and reinnervation techniques are usually used.¹¹ Using the HB grading system, patients who have spontaneous recovery of function at the HB V to HB VI levels are candidates for reinnervation using techniques such as hypoglossal facial nerve grafts or cross-facial grafting. Because these interventions require another year for development of function, some authors advocate concomitant reanimation with dynamic temporalis slings.¹² Patients who experience recovery to HB III to HB IV are often offered augmentation procedures, such as brow lift, alar suspension, and botulinum toxin injection for synkinesis. However, no attempt is made to surgically address the facial nerve itself. Patients who experience HB I to HB II recovery are typically satisfied with their functional and cosmetic outcomes and desire no intervention.

Other Cranial Nerve Injuries

Temporal bone fractures may result in palsies to cranial nerves other than the facial nerve, with a reported incidence of 7.8%.⁸ Petrous apex fractures involving the jugular foramen syndrome can cause palsies of cranial nerves IX to XI. As with the facial nerve, the palsy may be immediate or delayed, with delayed onset portending a better prognosis. Darrouzet and colleagues⁸ reported a 2.6% incidence of lower cranial nerve palsy in three cases of jugular foramen hematoma. Injury to cranial nerve VI occurs with an incidence of 5% to 7%, all of which occurred in fractures involving the petrous apex and included three patients with bilateral cranial nerve VI palsies associated with bilateral temporal bone fractures.⁸ Coup-contrecoup injuries can cause stretching and impingement of cranial nerves V to VII at the entrance of Meckel's cave.³

Associated Nerve Injuries From Neck Trauma

Neck trauma can present with injury to the cervical trachea resulting in blunt and penetrating injuries to

cranial nerves. Although uncommon, blunt injury to the cervical trachea can result in

- long-term morbidity,
- necessity of tracheotomy,
- tracheal stenosis, and
- injury to the recurrent laryngeal nerve.

In a combined case series reporting blunt injuries to the cervical trachea, 51 patients presented with injuries occurring at the cricotracheal junction (40%), and between the second and fourth tracheal rings (38%). The majority of injuries occurred above the fourth tracheal ring (88%). Respiratory distress was the most common symptom due to complete tracheal transections (69%), partial transections (26%), and two tears (5%). The most common nerve injured was the recurrent laryngeal nerve in 49% of patients.¹³

Although penetrating neck injuries are a common cause of cranial nerve deficit (including injury to cranial nerves IX to XII), the literature is devoid of reports associated with this injury pattern. High-velocity mechanisms can cause significant disruption to the carotid triangle with injury to the vagus nerve, as well as injury with the nearby hypoglossal and recurrent laryngeal nerves. In addition, cervical and posterior injuries can lead to cranial nerve XI palsy. Base-of-skull injury can also cause trauma to the jugular foramen resulting in cranial nerve IX to XI disruption. These patients often require life-saving treatments, including tracheotomy, vessel ligation, and neck exploration. An effort should always be made to identify cranial nerve injuries in the acute setting. Nevertheless, the diagnosis of cranial nerve deficits in the delayed setting requires an astute clinician and functional diagnostic tests, including a complete head-and-neck examination, modified barium swallow testing, high-resolution CT from the skull base to the base of the neck, and MRI imaging following the pathway of the cranial nerve in question from origin to destination. Laryngeal evaluation using stroboscopy is necessary to further evaluate recurrent laryngeal nerve paresis.

The hypoglossal nerve can be used in laryngeal reinnervation for traumatic recurrent laryngeal nerve injuries. Although not reported in posttraumatic combat injuries, laryngeal reinnervation may restore recurrent laryngeal nerve and/or superior laryngeal function. Several different donor nerves are available and have been described. The technique used may be direct end-to-end anastomosis (neurorrhaphy), direct implantation of a nerve into a muscle, nerve-muscle pedicle technique, or muscle nerve-muscle methods.¹⁴

CASE PRESENTATIONS

Case Study 35-1

Presentation

A 24-year-old white male presented to the emergency room in Bagram Air Force Theater Hospital, Afghanistan, with penetrating facial trauma caused by an improvised explosive device. The young Army sergeant was transported via helicopter with a GCS of 15. He was alert and oriented times three, and his initial vital signs were stable. However, he had no facial movement of the left side of his face in all cranial nerve divisions (HB VI). Physical examination showed several deep facial lacerations extending distal to the lateral canthus and zones II/III of the neck with active bleeding from the upper neck lacerations. Trauma resuscitation began in the emergency room.

Preoperative Workup/Radiology

None.

Because this patient with zone II/III penetrating facial trauma was symptomatic with active bleeding from his face and upper neck, he was taken immediately to the operating room to secure his airway and control bleeding. In addition, after the airway and bleeding were controlled, facial exploration and repair of the facial nerve were planned.

Operative Planning/Timing of Surgery

Trauma patients with symptomatic penetrating facial injury lateral to the lateral canthus of the eye need exploration. If these patients are otherwise stable, then consideration should be given to obtaining appropriate imaging studies (like CT angiography) en route to the operating room. Because this high-velocity penetrating injury was distal to the lateral canthus, a high likelihood of facial nerve injury is common. The decision to repair the facial nerve within 3 days of the injury affords the best prognosis for return of facial nerve function after a transection injury. Moreso, a facial nerve stimulator can be used to identify distal branches at this point.

Operation

After intubation, hemostasis was obtained, and the left neck was explored. The patient had active bleeding from the external jugular vein that was controlled, and the carotid sheath was intact. In addition, the greater auricular nerve was identified and protected. Then, the

facial nerve transection was identified at the intersection of the main trunk with the zygomaticofrontal and buccal-mandibular branches. Using a 5-0 silk, the main trunk and distal branches were tagged (Figure 35-2). Since there was a 2- to 3-cm defect and a tension-free closure could not be obtained, primary anastomosis was not performed. Consequently, a greater auricular graft was harvested and placed between the proximal and distal segments, thus ensuring that a tension-free closure could be obtained. Then, using proper microsurgical technique, a 9-0 nylon was used to secure the proximal and distal facial nerve segments to the graft. A 1/2-inch Penrose drain was placed, the wound was closed, and the patient was prepared for aerovacuation to Landstuhl Regional Medical Center (Landstuhl, Germany).

Complications

None.

The patient was followed in the Role 4 hospital setting and had a functional return to HB II/HB III according to postoperative ear-nose-throat notes 1 year later and based on a personal interview with the patient 2 years postoperatively.

Lessons Learned

The Army sergeant healed well without sequelae, except for the obvious scar on his left face. The lessons learned are that the head and neck surgeon must be prepared to treat acute facial nerve transection injuries

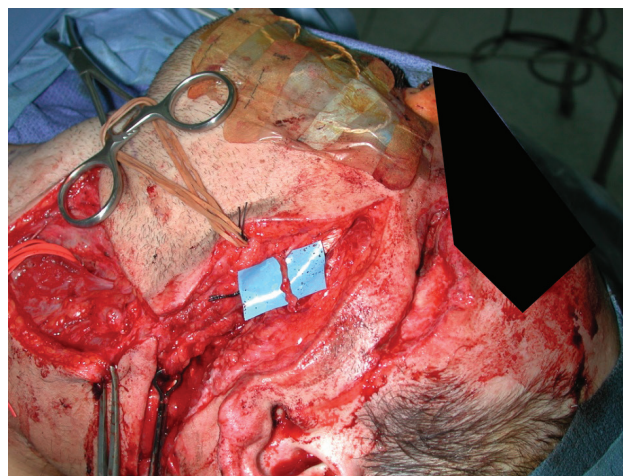


Figure 35-2. Facial nerve transection at the intersection of the main trunk with the zygomaticofrontal and buccal-mandibular branches.

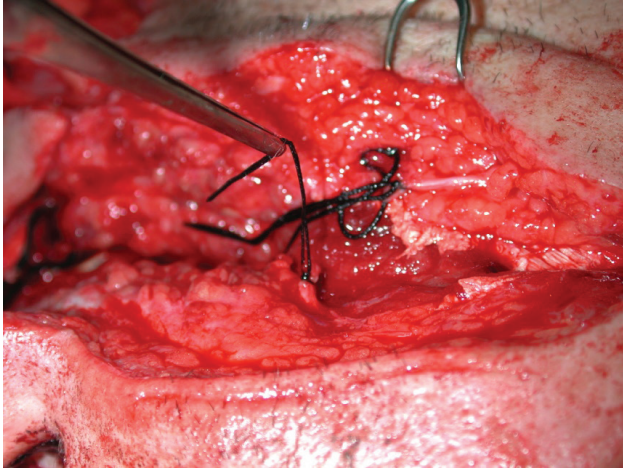


Figure 35-3. Facial nerve transection tagged in the operative field.

in the combat theater to ensure the best possible outcome for facial nerve function. If primary repair is accomplished within 3 days using proper microsurgical technique, then proper reinnervation of facial musculature can occur leading to successful facial nerve function. In this case, the old surgical axiom that the “sun should never rise and set on a facial nerve injury” should be followed, and exploration/repair of facial nerve injuries should be performed as soon as possible after injury. This is critically important because distal nerve stimulation, which is critically important for nerve identification, *may* occur for up to 3 days after nerve injury. It makes little sense to wait until 72 hours after injury because the ability to perform nerve stimulation may be lost by this time.



Figure 35-5. Open laceration.

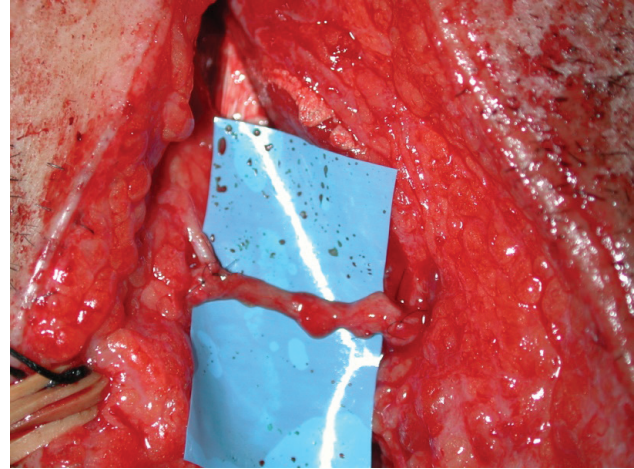


Figure 35-4. Primary anastomosis of facial nerve.

Case Study 35-2

Presentation

A 22-year-old white male presented to the emergency room with a facial injury medial to the lateral canthus of the eye at the region of the left cheek. The Army soldier was intubated with stable vital signs without active bleeding (ie, bleeding controlled by referring American trauma surgeons) and arrived at Bagram Air Base, Afghanistan, the day after his injury.

Preoperative Workup/Radiology

CT showed a left midface defect with multiple stellate lacerations adjacent to his nose (three layers) in the region of his left cheek. CT also demonstrated no evidence of naso-orbital-ethmoid fracture or Le Fort fractures, and no loss of upper dentition.

Operative Planning/Timing of Surgery

The patient was stable, and it was decided to take the patient to the operating room on day 1 after his injury for minimal wound debridement, irrigation, and exploration. Because the injury was medial to the lateral canthus, there was less concern that a facial nerve injury needed to be repaired.

Operation

The patient underwent three-layer reconstruction of the left cheek defect. There was no maxillary defect, parotid duct injury, or facial nerve injury (Figures 35-3 to 35-6).



Figure 35-6. Closure of cheek wound.

Complications

None.

Lessons Learned

The stellate facial laceration obtained by the soldier was explored, and there was no evidence of facial nerve injury. However, if the nerve was transected medial to the lateral canthus, then repair was not necessary. The facial nerve at this level will spontaneously regenerate into the distal musculature in the medial portion of the face. Regeneration may take up to 1 year to occur.

REFERENCES

1. MacKinnon SE, Dellon AL. *Surgery of the Peripheral Nerve*. New York, NY: Thieme Medical Publishers; 1988.
2. Dodson TB, Kaban LB. Recommendations for management of trigeminal nerve defects based on a critical appraisal of literature. *J Oral Maxillofac Surg*. 1997;55:1380–1386.
3. Johnson F, Semaan MT, Megerian CA. Temporal bone fracture: evaluation and management in the modern era. *Otolaryngol Clin North Am*. 2008;41:597–618.
4. Alvi A, Bereliani A. Acute intracranial complications of temporal bone trauma. *Otolaryngol Head Neck Surg*. 1998;119:609–613.
5. Darrouzet V, Duclos JY, Liguoro D, et al. Management of facial paralysis resulting from temporal bone fractures: our experience in 115 cases. *Otolaryngol Head Neck Surg*. 2001;125:77–84.
6. Brodie HA, Thompson TC. Management of complications from 820 temporal bone fractures. *Am J Otol*. 1997;18:188–197.
7. Ishman SL, Friedland DR. Temporal bone fractures: traditional classification and clinical relevance. *Laryngoscope*. 2004;114:1734–1741.
8. Darrouzet V, Duclos JY, Liguoro D, et al. Management of facial paralysis resulting from temporal bone fractures: our experience in 115 cases. *Otolaryngol Head Neck Surg*. 2001;125:77–84.
9. Ulug T, Arif Ulubil S. Management of facial paralysis in temporal bone fractures: a prospective study analyzing 11 operated fractures. *Am J Otolaryngol*. 2005;26:230–238.
10. Quaranta A, Campobasso G, Piazza F, et al. Facial nerve paralysis in temporal bone fractures: outcomes after late decompression surgery. *Acta Otolaryngol*. 2001;121:652–655.
11. Cheney ML, Megerian MA, McKenna MJ. Rehabilitation of the paralyzed face. In: Cheney ML, ed. *Facial Surgery: Plastic and Reconstructive*. Baltimore, MD: Williams & Wilkins; 1997: 655–685.
12. Cheney ML, McKenna MJ, Megerian CA, et al. Early temporalis muscle transposition for the management of facial paralysis. *Laryngoscope*. 1995;105(9 pt 1):993–1000.
13. Reece GP, Shatney CH. Blunt injuries of the cervical trachea: review of 51 patients. *South Med J*. 1988;81:1542–1547.
14. Paniello RC. Laryngeal reinnervation. *Otolaryngol Clin North Am*. 2004;37:161–181, vii–viii.

