Obectives

Cognitive

4-5.1 Describe the incidence, morbidity, and mortality of facial injuries. (p 21.29)
4-5.2 Explain facial anatomy and relate physiology to facial injuries. (p 21.4)
4-5.3 Predict facial injuries based on mechanism of injury. (p 21.16)
4-5.4 Predict other injuries commonly associated with facial injuries based on mechanism of injury. (p 21.16)
4-5.5 Differentiate between the following types of facial injuries, highlighting the defining characteristics of each:
   a. Eye
   b. Ear
   c. Nose
   d. Throat
   e. Mouth (p 21.16–21.29)
4-5.6 Integrate pathophysiological principles to the assessment of a patient with a facial injury. (p 21.16)
4-5.7 Differentiate between facial injuries based on the assessment and history. (p. 21.17)
4-5.8 Formulate a field impression for a patient with a facial injury based on the assessment findings. (p 21.16)
4-5.9 Develop a patient management plan for a patient with a facial injury based on the field impression. (p 21.21)
4-5.10 Explain the pathophysiology of eye injuries. (p 21.17)
4-5.11 Relate assessment findings associated with eye injuries to pathophysiology. (p 21.18)
4-5.12 Integrate pathophysiological principles to the assessment of a patient with an eye injury. (p 21.21)
4-5.13 Formulate a field impression for a patient with an eye injury based on the assessment findings. (p 21.21)
4-5.14 Develop a patient management plan for a patient with an eye injury based on the field impression. (p 21.21)
4-5.15 Explain the pathophysiology of ear injuries. (p 21.23)
4-5.16 Relate assessment findings associated with ear injuries to pathophysiology. (p 21.24)
4-5.17 Integrate pathophysiological principles to the assessment of a patient with an ear injury. (p. 21.25)
4-5.18 Formulate a field impression for a patient with an ear injury based on the assessment findings. (p 21.25)
4-5.19 Develop a patient management plan for a patient with an ear injury based on the field impression. (p 21.25)
4-5.20 Explain the pathophysiology of nose injuries. (p 21.7)
4-5.21 Relate assessment findings associated with nose injuries to pathophysiology. (p 21.15)
4-5.22 Integrate pathophysiological principles to the assessment of a patient with a nose injury. (p 21.17)
4-5.23 Formulate a field impression for a patient with a nose injury based on the assessment findings. (p 21.17)
4-5.24 Develop a patient management plan for a patient with a nose injury based on the field impression. (p 21.17)
4-5.25 Explain the pathophysiology of throat injuries. (p 21.26)
| 4-5.53 | Develop a management plan for a patient with a skull fracture. (p 21.38) |
| 4-5.54 | Explain the pathophysiology of cerebral contusion. (p 21.33) |
| 4-5.55 | Relate assessment findings associated with cerebral contusion to pathophysiology. (p 21.33) |
| 4-5.56 | Develop a management plan for a patient with a cerebral contusion. (p 21.36) |
| 4-5.57 | Explain the pathophysiology of intracranial hemorrhage, including: |
| | a. Epidural |
| | b. Subdural |
| | c. Intracerebral |
| | d. Subarachnoid (p 21.34, 21.35) |
| 4-5.58 | Relate assessment findings associated with intracranial hemorrhage to pathophysiology, including: |
| | a. Epidural |
| | b. Subdural |
| | c. Intracerebral |
| | d. Subarachnoid (p 21.34, 21.35) |
| 4-5.59 | Develop a management plan for a patient with an intracranial hemorrhage, including: |
| | a. Epidural |
| | b. Subdural |
| | c. Intracerebral |
| | d. Subarachnoid (p 21.36) |
| 4-5.60 | Describe the various types of helmets and their purposes. (p 22.24, 22.25) |
| 4-5.61 | Relate priorities of care to factors determining the need for helmet removal in various field situations including sports related incidents. (p 22.24, 22.25) |
| 4-5.62 | Develop a management plan for the removal of a helmet for a head injured patient. (p 22.24, 22.25) |
| 4-5.63 | Integrate the pathophysiological principles to the assessment of a patient with head/brain injury. (p 21.36) |
| 4-5.64 | Differentiate between the types of head/brain injuries based on the assessment and history. (p 21.36) |
| 4-5.65 | Formulate a field impression for a patient with a head/brain injury based on the assessment findings. (p 21.38) |
| 4-5.66 | Develop a patient management plan for a patient with a head/brain injury based on the field impression. (p 21.38) |

**Affective**
None

**Psychomotor**
None
Head and Face Injuries

As a paramedic, you will commonly encounter patients with injuries to the head, neck, and face, ranging in severity from a broken nose to traumatic brain injury. The first part of this chapter provides a detailed review of the anatomy and physiology of the head and face. The second part discusses head and face injuries, including their respective signs and symptoms and appropriate prehospital care: maxillofacial injuries, eye and ear injuries, oral and dental injuries, injuries to the anterior part of the neck, and head and traumatic brain injuries.

The Skull and Facial Bones

The Scalp

The brain—the most important organ in the body—requires maximum protection from injury. The human body ensures that it receives this protection by housing the brain within several layers of soft and hard wrappings.

Starting from the outside and proceeding inward toward the brain, the first protective layer is the scalp, which consists of the following layers, given in descending order:

- Skin, with hair
- Subcutaneous tissue, which contains major scalp veins that bleed profusely when lacerated.
- Galea aponeurotica, a tendon expansion that connects the frontal and occipital muscles of the cranium
- Loose connective tissue (alveolar tissue), which is easily stripped from the layer beneath in “scalping” injuries. The looseness of the alveolar layer also provides room for blood to accumulate after blunt trauma between the scalp and skull bone (subgaleal hematoma).
- Periosteum, the dense fibrous membrane covering the surface of bones

The Skull

At the top of the axial skeleton is the skull, which consists of 28 bones in three anatomic groups: the auditory ossicles, the cranium, and the face. The six auditory ossicles function in hearing and are located, three on each side of the head, deep

You are the Provider Part 1

You respond to the scene of a motorcycle crash. The patient, a young male, was ejected from his motorcycle when it struck a tree; he was not wearing a helmet. The scene is safe, and two police officers are at the scene directing the flow of traffic. As you approach the patient, you note that he is lying in a supine position. His eyes are closed, and he is not moving.

1. What should be your initial concern about this patient?
2. How should you direct your initial care of this patient?
within the cavities of the temporal bone. The remaining 22 bones constitute the cranium and the face. The cranial vault consists of eight bones that encase and protect the brain: the parietal, temporal, frontal, occipital, sphenoid, and ethmoid bones. The brain connects to the spinal cord through a large opening at the base of the skull called the foramen magnum.

The bones of the skull are connected at special joints known as sutures. The paired parietal bones join together at the sagittal suture. The parietal bones abut the frontal bone at the coronal suture. The occipital bone attaches to the parietal bones at the lambdoid suture. Fibrous tissues called fontanelles, which are soft in infants, link the sutures. The tissues felt through the fontanelles are layers of the scalp and thick membranes overlying the brain. Under normal conditions, the brain may not be felt through the fontanelles. By the time a child is 18 months old, the sutures should have solidified and the fontanelles closed.

At the base of each temporal bone is a cone-shaped section of bone known as the mastoid process. This area is an important site for attachment of various muscles. In addition, a portion of the mastoid process contains hollow mastoid air cells.

The Floor of the Cranial Vault
Viewed from above, the floor of the cranial vault is divided into three compartments: the anterior fossa, middle fossa, and posterior fossa. The crista galli forms a prominent bony ridge in the center of the anterior fossa and is the point of attachment of the meninges, the three layers of membranes that surround the brain and spinal cord. On the other side of the crista galli is the cribriform plate of the ethmoid bone, a horizontal bone that is perforated with numerous openings (foramina) allowing the passage of the olfactory nerve filaments from the nasal cavity. The olfactory nerves, the cranial nerves for smell, send projections through the foramina in the cribriform plate and into the nasal cavity, the chamber inside the nose that lies between the floor of the cranium and the roof of the mouth.

The Base of the Skull
When the mandible is removed, the base of the skull appears amazingly complex, with numerous foramina visible. The occipital condyles on the occipital bone, which are the points of articulation between the skull and the vertebral column, lie on either side of the foramen magnum. Portions of the maxilla and the palatine bone, the irregularly shaped bone in the posterior nasal cavity, form the hard palate, which is the bony anterior part of the palate, or roof, of the mouth. The zygomatic arch is the bone that extends along the front of the skull below the orbit.

The Facial Bones
The frontal and ethmoid bones are part of the cranial vault and the face. The
14 facial bones form the structure of the face, without contributing to the cranial vault. They include the maxillae, vomer, inferior nasal concha, and the zygomatic, palatine, nasal, and lacrimal bones (see Figure 21-1A).

The facial bones protect the eyes, nose, and tongue; they also provide attachment points for the muscles that allow chewing. The zygomatic process of the temporal bone and the temporal process of the zygomatic bone form the zygomatic arch, which lends shape to the cheeks.

Two major nerves provide sensory and motor control to the face: the trigeminal nerve (fifth cranial nerve) and the facial nerve (seventh cranial nerve). The trigeminal nerve branches into the ophthalmic nerve, maxillary nerve, and mandibular nerve. The ophthalmic nerve (a sensory nerve) supplies the skin of the forehead, upper eyelid, and conjunctiva. The maxillary nerve (another sensory nerve) supplies the skin on the posterior part of the side of the nose, lower eyelid, cheek, and upper lip. The mandibular nerve (a sensory and motor nerve) supplies the muscles of chewing (mastication) and skin of the lower lip, chin, temporal region, and part of the external ear. The facial nerve supplies the muscles of facial expression.

Blood supply to the face is provided primarily through the external carotid artery, which branches into the temporal, mandibular, and maxillary arteries. Because the face is highly vascular, it tends to bleed heavily when injured.

The Orbits

The orbits are cone-shaped fossae that enclose and protect the eyes. In addition to the eyeball and muscles that move it, the orbit contains blood vessels, nerves, and fat.

A blow to the eye may result in fracture of the orbital floor because the bone is extremely thin and breaks easily. A

You are the Provider Part 2

As your partner maintains manual stabilization of the patient’s head and simultaneously opens his airway with the jaw-thrust maneuver, you perform an initial assessment.

<table>
<thead>
<tr>
<th>Initial Assessment</th>
<th>Recording Time: 0 Minutes</th>
</tr>
</thead>
<tbody>
<tr>
<td>Appearance</td>
<td>Supine, not moving, massive facial trauma</td>
</tr>
<tr>
<td>Level of consciousness</td>
<td>P (Responsive to painful stimuli)</td>
</tr>
<tr>
<td>Airway</td>
<td>Blood is draining from the patient’s mouth</td>
</tr>
<tr>
<td>Breathing</td>
<td>Respiration is gurgling, slow, and irregular</td>
</tr>
<tr>
<td>Circulation</td>
<td>Radial pulses are rapid and bounding; bleeding from the mouth; no other gross bleeding</td>
</tr>
</tbody>
</table>

3. How will you manage this patient's airway?
4. Would it be appropriate to intubate this patient? If so, when?
blowout fracture  results in transmission of forces away from the eyeball itself to the bone. Blood and fat then leak into the maxillary sinus.

The Nose
The nose is one of the two primary entry points for oxygen-rich air to enter the body. The nasal septum—the separation between the nostrils—is located in the midline. Often, it bulges slightly to one side or the other. The external portion of the nose is formed mostly of cartilage.

Several bones associated with the nose contain cavities known as the paranasal sinuses . These hollowed sections of bone, which are lined with mucous membranes, decrease the weight of the skull and provide resonance for the voice. The contents of the sinuses drain into the nasal cavity.

The Mandible and Temporomandibular Joint
The mandible is the large movable bone forming the lower jaw and containing the lower teeth. Numerous muscles of chewing attach to the mandible and its rami. The posterior condyle of the mandible articulates with the temporal bone at the temporomandibular joint (TMJ), allowing movement of the mandible (see Figure 21-3).

The Hyoid Bone
The hyoid bone “floats” in the superior aspect of the neck just below the mandible. While it is not actually part of the skull, it supports the tongue and serves as a point of attachment for many important neck and tongue muscles.

The Eyes, Ears, Teeth, and Mouth

The Eye
The globe, or eyeball, is a spherical structure measuring about 1 inch in diameter that is housed within the eye socket, or orbit. The eyes are held in place by loose connective tissue and several muscles. These muscles also control eye movements. The oculomotor nerve (third cranial nerve) innervates the muscles that cause motion of the eyeballs and upper eyelids. It also carries parasympathetic nerve fibers that cause constriction of the pupil and accommodation of the lens. The optic nerve (second cranial nerve) provides the sense of vision.

The structures of the eye include the following:
- The sclera ("white of the eye") is a tough, fibrous coat that helps maintain the shape of the eye and protect the contents of the eye. In some illnesses, such as hepatitis, the sclera become yellow (icteric) from staining by bile pigments.
- The cornea is the transparent anterior portion of the eye that overlies the iris and pupil. Clouding of the cornea during aging results in a condition known as cataract.
- The conjunctiva is a delicate mucous membrane that covers the sclera and internal surfaces of the eyelids but not the iris. Cyanosis can be detected in the conjunctiva when it is not easily assessed on the skin of dark-skinned patients.
The iris is the pigmented part of the eye that surrounds the pupil. It consists of muscles and blood vessels that contract and expand to regulate the size of the pupil.

The pupil is the circular adjustable opening within the iris through which light passes to the lens. A normal pupil dilates in dim light to permit more light to enter the eye and constricts in bright light to decrease the light entering the eye.

Behind the pupil and iris is the lens, a transparent structure that can alter its thickness to focus light on the retina at the back of the eye.

The retina, which lies in the posterior aspect of the interior globe, is a delicate, 10-layered structure of nervous tissue that extends from the optic nerve. It receives light impulses and converts them to nerve signals that are conducted to the brain by the optic nerve and interpreted as vision.

The anterior chamber is the portion of the globe between the lens and the cornea. It is filled with aqueous humor, a clear watery fluid. If aqueous humor is lost through a penetrating injury to the eye, it will gradually be replenished.

The posterior chamber is the portion of the globe between the iris and the lens which is filled with vitreous humor, a jelly-like substance that maintains the shape of the globe. If vitreous humor is lost, it cannot be replenished, and blindness may result.

Light rays enter the eyes through the pupil and are focused by the lens. The image formed by the lens is cast on the retina, where sensitive nerve fibers form the optic nerve. The optic nerve transmits the image to the brain, where it is converted into conscious images in the visual cortex.

There are two types of vision: central and peripheral. Central vision, facilitates visualization of objects directly in front
of you, and is processed by the macula, the central portion of the retina. The remainder of the retina processes \textit{peripheral vision}, which gives us visualization of lateral objects while looking forward.

The \textit{lacrimal apparatus} secretes and drains tears from the eye. Tears produced in the lacrimal gland drain into lacrimal ducts, then into lacrimal sacs that pass into the nasal cavity via the \textit{nasolacrimal duct}. Tears moisten the \textit{conjunctiva}.

\textbf{The Ear}

The ear is divided into three anatomic parts: external, middle, and inner. The \textit{external ear} consists of the pinna, external auditory canal, and the exterior portion of the tympanic membrane or what is commonly known as the eardrum. The \textit{middle ear} consists of the inner portion of the tympanic membrane and the ossicles while the \textit{inner ear} consists of the cochlea and semicircular canals.

Sound waves enter the ear through the \textit{auricle}, or \textit{pinna}, the large cartilaginous external portion of the ear. They then travel through the \textit{external auditory canal} to the \textit{tympanic membrane}. Vibration of sound waves against the tympanic membrane sets up vibration in the \textit{ossicles}, the three small bones on the inner side of the tympanic membrane. These vibrations are transmitted to the \textit{cochlear duct} at the \textit{oval window}, the opening between the middle ear and the vestibule. Movement of the oval window causes fluid within the \textit{cochlea}, a shell-shaped structure in the inner ear, to vibrate. Within the cochlea at the \textit{organ of Corti}, vibration stimulates hair movements that form nerve impulses that travel to the brain via the auditory nerve. The brain then converts these impulses into sound.

\textbf{The Teeth}

The normal adult mouth contains 32 permanent teeth. The primary or deciduous teeth are lost during childhood. Adult teeth are distributed about the maxillary and mandibular arches. The teeth on each side of the arch are mirror images of each other and form four quadrants: right upper, left upper, right lower, and left lower. Each quadrant contains one central incisor, one lateral incisor, one canine, two premolars, and three molars. The third molars or what are called wisdom teeth (which have nothing to do with wisdom) do not appear until late adolescence.

The top portion of the tooth, external to the gum, is the \textit{crown}, containing one or more \textit{cusps}. Below the crown lie the neck and the root. The pulp cavity fills the center of the tooth and contains blood vessels, nerves, and specialized connective tissue, called \textit{pulp}. Dentin and enamel surround the pulp cavity and protect the tooth from damage.
The Anterior Part of the Neck

The principal structures of the anterior part of the neck include the thyroid and cricoid cartilage, trachea, and numerous muscles and nerves. The major blood vessels in this area are the internal and external carotid arteries and the internal and external jugular veins. The vertebral arteries run laterally to the cervical vertebrae in the posterior part of the neck.

The major arteries of the neck—the carotid and vertebral arteries—supply oxygenated blood directly to the brain. Therefore, in addition to causing massive bleeding and hemorrhagic shock, injury to any of these major vessels can produce...
cerebral hypoxia, infarct, air embolism and/or permanent neurologic impairment.

Other key structures of the anterior part of the neck that may sustain injury from blunt or penetrating mechanisms include the vagus nerves, thoracic duct, esophagus, thyroid and parathyroid glands, lower cranial nerves, brachial plexus (which is responsible for function of the lower arm and hand), soft tissue and fascia, and various muscles.

### The Brain

The brain, which occupies 80% of the cranial vault, contains billions of neurons (nerve cells) that serve a variety of vital functions. The major regions of the brain are the cerebrum, diencephalon (thalamus and hypothalamus), brain stem (medulla, pons, midbrain [mesencephalon]), and the cerebellum. The remaining intracranial contents include cerebral blood (12%) and cerebrospinal fluid (8%).

The brain accounts for only 2% of the total body weight, yet it is the most metabolically active and perfusion-sensitive organ in the body. The brain metabolizes 25% of the body's glucose, burning approximately 60 mg/min, and consumes 20% of the total body oxygen (45 to 50 L/min). Because the brain has no storage mechanism for oxygen or glucose, it is totally dependent on a constant source of both fuels via cerebral blood flow provided by the carotid and vertebral arteries. As such, the brain will continually manipulate the physiology as needed to guarantee that a ready supply of oxygen and glucose are available.

### The Cerebrum

The largest portion of the brain is the cerebrum, which is responsible for higher functions, such as reasoning. The cerebrum is divided into right and left hemispheres by a longitudinal fissure. The hemispheres of the cerebrum are not entirely equivalent functionally. In a right-handed person, for example,
21.12 Section 4 Trauma

The speech center is located in the **temporal lobe**. In approximately 85% of the population, the speech center is located on the left side of the temporal lobe. The temporal lobe also controls long-term memory, hearing, taste, and smell. It is separated from the rest of the cerebrum by a lateral fissure.

**The Diencephalon**

The **diencephalon**, which is located between the brain stem and the cerebrum, includes the thalamus, subthalamus, hypothalamus, and epithalamus. The **thalamus** processes most sensory input and influences mood and general body movements, especially those associated with fear and rage. The **subthalamus** controls motor functions. The functions of the epithalamus are unclear. The most inferior portion of the diencephalon, the **hypothalamus**, is vital in the control of many body functions, including heart rate, digestion, sexual development, temperature regulation, emotion, hunger, thirst, vomiting, and regulation of the sleep cycle.

**The Cerebellum**

The **cerebellum** is located beneath the cerebral hemispheres in the inferoposterior part of the brain. It is sometimes called the “athlete’s brain” because it is responsible for the maintenance of posture and skilled movements.

The largest portion of the cerebrum is the **cerebral cortex**, which regulates voluntary skeletal movement and the level of awareness. Injury to the cerebral cortex may result in paresthesia, weakness, and paralysis of the extremities.

Each cerebral hemisphere is divided functionally into specialized areas called lobes. **The frontal lobe** is important for voluntary motor action and personality traits. Injury to the frontal lobe may result in seizures or placid reactions (flat affect). The **parietal lobe** controls the somatic or voluntary sensory and motor functions for the opposite (contralateral) side of the body; as well as memory and emotions; it is separated from the frontal lobe by the central sulcus. Posteriorly, the **occipital lobe**, from which the optic nerve originates, is responsible for processing visual information. After a blow to the back of the head, a person may “see stars” which results when the occipital poles of the brain (the vision centers) bang against the back of the skull.

![Figure 21-18 The major regions of the brain.](image1)

![Figure 21-19 Lobes of the cerebrum.](image2)
of posture and equilibrium and the coordination of skilled movements.

The Brain Stem

The brain stem consists of the midbrain, pons, and the medulla. It is located at the base of the brain and connects the spinal cord to the remainder of the brain. The brain stem houses many structures that are critical to the maintenance of vital functions. High in the brain stem, for example, is the reticular activating system (RAS), which is responsible for maintenance of consciousness, specifically one’s level of arousal. The centers that control basic but critical functions—heart rate, blood pressure, and respiration—are located in the lower part of the brain stem. Damage to this area can easily result in cardiovascular derangement, respiratory arrest, or death.

The midbrain lies immediately below the diencephalon and is the smallest region of the brain stem. Deep within the cerebrum, diencephalon, and midbrain are the basal ganglia, which have an important role in coordination of motor movements and posture. Portions of the cerebrum and diencephalon constitute the limbic system, which influences emotions, motivation, mood, and sensations of pain and pleasure.

The oculomotor nerve (third cranial nerve) originates from the midbrain; it controls pupillary size and reactivity.

The pons, which lies below the midbrain and above the medulla, contains numerous important nerve fibers, including those for sleep, respiration, and the medullary respiratory center.

The inferior portion of the midbrain, the medulla, is continuous inferiorly with the spinal cord (see Figure 21-18). It serves as a conduction pathway for ascending and descending nerve tracts. It also coordinates heart rate, blood vessel diameter, breathing, swallowing, vomiting, coughing, and sneezing. The vagus nerve (tenth cranial nerve), a bundle of nerves that primarily innervates the parasympathetic nervous system, originates from the medulla.

The Meninges

The meninges are protective layers that surround and enfold the entire central nervous system—specifically the brain and spinal cord. The outermost layer is a strong, fibrous
The second meningeal layer is a delicate, transparent membrane called the arachnoid. It is so named because the blood vessels it contains resemble a spider web. The third meningeal layer, the pia mater (“soft mother”), is a thin, translucent, highly vascular membrane that firmly adheres directly to the surface of the brain.

The meningeal arteries are located between the dura mater and the skull. When one of these arteries (usually the middle meningeal artery) is disrupted, bleeding occurs above the dura mater, resulting in an epidural hematoma.

The meninges float in cerebrospinal fluid (CSF), which is manufactured in the ventricles of the brain. CSF flows in the subarachnoid space, located between the pia mater and the arachnoid.

CSF is manufactured by cells within the choroid plexus in the ventricles, hollow storage areas in the brain. These areas normally are interconnected, and CSF flows freely between them. CSF is similar in composition to plasma. The meninges and CSF form a fluid-filled sac that cushions and protects the brain and spinal cord.

**Face Injuries**

**Soft-Tissue Injuries**

Although open soft-tissue injuries to the face—lacerations, abrasions, and avulsions—by themselves are rarely life threatening, their presence, especially following a significant mechanism of injury, suggests the potential for more severe injuries (eg, closed head injury, cervical spine injury). Furthermore, massive soft-tissue injuries to the face, especially if associated with oropharyngeal trauma and bleeding, can compromise the patient's airway and lead to ventilatory inadequacy.

Maintain a high index of suspicion when a patient presents with closed soft-tissue injuries to the face, such as contusions and hematomas. These indicators of blunt force trauma suggest the potential for more severe underlying injuries.

Impaled objects in the soft tissues or bones of the face may occur in association with facial trauma. Although these objects can damage facial nerves, the risk of airway compromise is of far greater consequence. This is especially true when an object passes through the soft tissues and into the oropharynx.

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**You are the Provider Part 3**

Your partner is appropriately managing the patient’s airway. You perform a rapid trauma assessment, which reveals a hematoma to the patient’s forehead, massive soft-tissue trauma to the face, unstable facial bones, and bilaterally angulated femurs.

<table>
<thead>
<tr>
<th>Vital Signs</th>
<th>Recording Time: 5 Minutes</th>
</tr>
</thead>
<tbody>
<tr>
<td>Level of consciousness</td>
<td>Glasgow Coma Scale score of 6</td>
</tr>
<tr>
<td>Respiration</td>
<td>6 breaths/min and irregular (baseline); your partner is providing bag-mask ventilation at a rate of 10 breaths/min and 100% oxygen</td>
</tr>
<tr>
<td>Pulse</td>
<td>110 beats/min; regular and bounding</td>
</tr>
<tr>
<td>Skin</td>
<td>Warm and dry</td>
</tr>
<tr>
<td>Blood pressure</td>
<td>140/90 mm Hg</td>
</tr>
<tr>
<td>Sao2</td>
<td>96% (with assisted ventilation and 100% oxygen)</td>
</tr>
</tbody>
</table>

5. How can facial trauma complicate airway management?
6. Is this patient in hypovolemic shock? Why or why not?
impaled object penetrates the cheek, because massive oropharyngeal bleeding can result in airway obstruction, aspiration, and ventilatory inadequacy. In addition, blood is a gastric irritant. For many people, just swallowing a couple of tablespoons of blood can make them vomit, further increasing the likelihood of aspiration.

Maxillofacial Fractures
Maxillofacial fractures commonly occur when the facial bones absorb the energy of a strong impact. The forces involved may be massive. For example, a force up to 150 g (g = acceleration of the body due to gravity) is required to fracture the maxilla; a force of that magnitude will likely produce closed head injuries and cervical spine injuries as well. Therefore, when assessing a patient with a suspected maxillofacial fracture, you should protect the cervical spine and monitor the patient's neurologic signs, specifically their level of consciousness.

The first clue to the presence of a maxillofacial fracture is usually ecchymosis, so a black-and-blue mark on the face should alert you to this possibility. A deep facial laceration should likewise increase your index of suspicion that the underlying bone may have been fractured, and pain over a bone tends to support the suspicion of fracture. General signs and symptoms of maxillofacial fractures include ecchymosis, swelling, pain to palpation, crepitus, dental malocclusion, facial deformities or asymmetry, instability of the facial bones, impaired ocular movement, and visual disturbances.

Nasal Fractures
Because the nasal bones are not as structurally sound as the other bones of the face, nasal fractures are the most common facial fracture. These fractures are characterized by swelling, tenderness, and crepitus when the nasal bone is palpated. Deformity of the nose, if present, usually appears as lateral displacement of the nasal bone from its normal midline position.

Nasal fractures, like any maxillofacial fracture, are often complicated by the presence of an anterior or a posterior nosebleed (epistaxis), which can compromise the patient’s airway.
Le Fort fractures can occur as isolated fractures (Le Fort I) or in combination (Le Fort I and II), depending on the location of impact and the amount of trauma.

**Orbital Fractures**

The patient with an orbital fracture (such as a blowout fracture [see Figure 21-7]) may complain of double vision (diplopia) and lose sensation above the eyebrow or over the cheek secondary to associated nerve damage. Massive nasal discharge may occur, and vision is often impaired. Fractures of the inferior orbit are the most common type and can cause paralysis of upward gaze (the patient’s injured eye will not be able to follow your finger above the midline).

**Zygomatic Fractures**

Fractures of the zygomatic bone (cheek bone) commonly result from blunt trauma secondary to motor vehicle crashes and assaults. When the zygomatic bone is fractured, that side of the patient’s face appears flattened, and there is loss of sensation over the cheek, nose, and upper lip; paralysis of upward gaze may also be present. Other injuries commonly associated with zygomatic fractures include orbital fractures, ocular injury, and epistaxis.

**Assessment and Management of Face Injuries**

Table 21-1 summarizes the characteristics of various maxillofacial fractures. It is not important to distinguish among the various maxillofacial fractures in the prehospital setting; this determination requires radiographic evaluation in the emergency department. Rapid patient assessment, management of life-threatening conditions, full spinal precautions, and prompt transport are far more important considerations.

Management of the patient with facial trauma begins by protecting the cervical spine. Because many severe facial injuries are complicated by a spinal injury, you must assume that one exists.

If the patient is semiconscious or unconscious, open the airway with the jaw-thrust maneuver while simultaneously maintaining manual stabilization of the head in the neutral position unless the patient complains of severe pain or discomfort upon movement. Should that occur, the head/neck should be immobilized in the position found. Inspect the mouth for fragments of teeth, dentures, or any other foreign bodies that could obstruct the airway, and remove them immediately. Suction the oropharynx as needed to keep the airway clear of blood and other liquids.

Insert an airway adjunct as needed to maintain airway patency. However, do not insert a nasopharyngeal airway or attempt nasotracheal intubation in any patient with suspected nasal fractures or in patients with CSF or blood leakage from the nose. After establishing and maintaining a patent airway, assess the patient’s breathing and intervene appropriately. Apply 100% oxygen via nonrebreathing mask if the patient is breathing adequately. Patients who are breathing inadequately (ie, fast or slow rate, reduced tidal volume [shallow breathing], irregular pattern of inhalation and exhalation) should receive bag-mask ventilation with 100% oxygen. Maintain the patient’s oxygen saturation at greater than 95%.

Airway management can be especially challenging in patients with massive facial injuries.

![Figure 21-25](image-url) Airway management can be especially challenging in patients with massive facial injuries.
Positioning of the patient with epistaxis is important to prevent their side, unless contraindicated by a spinal injury. Proper nares together. Unconscious patients should be positioned on their side while maintaining a mask-to-face seal for bag-mask ventilation. Performing tracheal intubation of patients with facial trauma, especially those who are unconscious, to protect their airway from aspiration and to ensure adequate oxygenation and ventilation. Cricothyrotomy (surgical or needle) may be required for patients with extensive maxillofacial injuries when endotracheal intubation is extremely difficult or impossible to perform (ie, in cases of unstable facial bones, massive swelling, severe oral bleeding).

Treat facial lacerations and avulsions as you would any other soft-tissue injury. Control all bleeding with direct pressure, and apply sterile dressings. If you suspect an underlying facial fracture, apply just enough pressure to control the bleeding. Leave impaled objects in the face in place and appropriately stabilize them, unless they pose a threat to the airway (such as an object impaled in the cheek). When removing an object from the cheek, carefully remove it from the same side that it entered. Next, pack the inside of the cheek with sterile gauze and apply counterpressure with a dressing and bandage firmly secured over the outside of the wound. If profuse bleeding continues, position the patient on his or her side—while maintaining a mask-to-face seal for bag-mask ventilation. Monitoring the pulse oximeter during this process can further serve to keep the patient from becoming hypoxic.

For severe oropharyngeal bleeding in patients with inadequate ventilation, suction the airway for 15 seconds and provide ventilatory assistance for 2 minutes; continue this alternating pattern of suctioning and ventilating until the airway is cleared of blood or secured with an endotracheal (ET) tube. Monitoring the pulse oximeter during this process can further serve to keep the patient from becoming hypoxic.

Epistaxis following facial trauma can be severe and is most effectively controlled by applying direct pressure to the nares. If the patient is conscious and spinal injury is not suspected, instruct the patient to sit up and lean forward as you pinch the nares together. Unconscious patients should be positioned on their side, unless contraindicated by a spinal injury. Proper positioning of the patient with epistaxis is important to prevent blood from draining down the throat and compromising the airway either by occlusion or by vomiting and then aspirating gastric contents. If the conscious patient with severe epistaxis is immobilized on a backboard, you should consider pharmacologically assisted intubation (eg, rapid-sequence intubation [RSI]) to gain definitive control of the airway.

Although facial lacerations and avulsions can contribute to hemorrhagic shock, they are rarely the sole cause of this condition in adults. Severe epistaxis, however, can result in significant blood loss. To counter this problem, you should carefully assess the patient for signs of hemorrhagic shock and administer intravenous (IV) crystalloid fluid boluses as needed to maintain adequate perfusion.

If the facial fracture is associated with swelling and ecchymosis, cold compresses may help minimize further swelling and alleviate pain. Do not apply a compress to the eyeball (globe) if you suspect that it has been injured following an orbital fracture; doing so may increase the intraocular pressure (IOP) and further damage the eye. Other than protecting the airway, little can be done to treat facial instabilities; however, firmly applying a self-adhering roller bandage (such as Kerlix or Kling) can stabilize the mandible. Make sure that you do not compromise the airway when stabilizing the mandible.

After addressing all life-threatening injuries and conditions, you should attempt to ascertain the events that preceded the injury and determine whether the patient has any significant medical problems. The incident that caused the injury may have been preceded by exacerbation of an underlying medical condition (such as acute hypoglycemia, cardiac dysrhythmia, seizure). For unconscious patients, medications that the patient is taking may provide information about his or her medical history. Determine the approximate time that the injury occurred, and ask about any drug allergies and the last oral intake during your SAMPLE history.

**In the Field**

Blood or CSF drainage from the nose (cerebrospinal rhinorrhea) suggests a skull fracture. Do not make any attempt to control this bleeding; doing so may increase intracranial pressure (ICP) if the patient has a concomitant brain injury. Furthermore, the insertion of nasal airway adjuncts and nasotracheal intubation should be avoided in patients with suspected nasal fractures, especially if rhinorrhea is present. A nasally inserted airway device could enter the cranial vault through an occult fracture (such as a cribiform plate fracture) and penetrate the brain further worsening the situation.

Oropharyngeal bleeding poses an immediate threat to the airway, and unstable facial bones can hinder your ability to maintain an effective mask-to-face seal for bag-mask ventilation. Therefore, perform tracheal intubation of patients with facial trauma, especially those who are unconscious, to protect their airway from aspiration and to ensure adequate oxygenation and ventilation. Cricothyrotomy (surgical or needle) may be required for patients with extensive maxillofacial injuries when endotracheal intubation is extremely difficult or impossible to perform (ie, in cases of unstable facial bones, massive swelling, severe oral bleeding).

**Special Considerations**

Relative to younger, healthy adults, elderly patients are at high risk for severe epistaxis following even minor facial injuries, especially in those with a history of hypertension or anticoagulant medication use (such as warfarin [Coumadin]). This bleeding often originates in the posterior nasopharynx and may not be grossly evident during your assessment.

**Eye Injuries**

Approximately 1.5 million eye injuries occur in the United States each year, of which 50,000 result in some degree of visual loss. Because trauma to the eyes is so common and the potential consequences are so serious, you must know how to assess and manage ocular injuries.

Eye injuries are frequently caused by blunt trauma, penetrating trauma, or burns. Blunt mechanisms of injury may
include motor vehicle crashes, motorcycles crashes, falls, and assaults. Penetrating injuries are often secondary to foreign bodies on the surface of the eye (such as sand) or an object impaled in the globe. Burns to the eye can result from a variety of corrosive chemicals or during industrial accidents (such as welding burns).

**Lacerations, Foreign Bodies, and Impaled Objects**

Lacerations of the eyelids require meticulous repair to restore appearance and function. Bleeding may be heavy, but it usually can be controlled by gentle, manual pressure. If there is a laceration to the globe itself, apply no pressure to the eye; compression can interfere with the blood supply to the back of the eye and result in loss of vision from damage to the retina. Furthermore, pressure may squeeze the vitreous humor, iris, lens, or even the retina out of the eye and cause irreparable damage or blindness.  

The protective orbit prevents large objects from penetrating the eye. However, moderately sized and smaller foreign objects can still enter the eye and, when lying on the surface of the eye, produce severe irritation. The conjunctiva becomes inflamed and red—a condition known as conjunctivitis—almost immediately, and the eye begins to produce tears in an attempt to flush out the object. Irritation of the cornea or conjunctiva causes intense pain. The patient may have difficulty keeping the eyelids open, because the irritation is further aggravated by bright light.

Foreign bodies ranging in size from a pencil to a sliver of metal may be impaled in the eye. Clearly, these objects must be removed by a physician. Prehospital care involves stabilizing the object and preparing the patient for transport. The greater the length of the foreign object sticking out of the eye, the more important stabilization becomes in avoiding further damage. Whenever possible, cover both eyes to limit unnecessary movement as the patient tries to use the uninjured eye to compensate for the loss or limited vision of the injured eye.
Blunt Eye Injuries

Blunt trauma can cause serious eye injuries, ranging from swelling and ecchymosis to rupture of the globe. Hyphema is bleeding into the anterior chamber of the eye that obscures vision, partially or completely. It often follows blunt trauma and may seriously impair vision. Approximately 25% of hyphemas are associated with globe injuries.

In orbital blowout fractures, the fragments of fractured bone can entrap some of the muscles that control eye movement, causing double vision (diplopia). Any patient who reports pain, double vision, or decreased vision following a blunt injury about the eye should be assumed to have a blowout fracture and should be promptly transported to an appropriate medical facility.

Another potential result of blunt eye trauma is retinal detachment, or separation of the inner layers of the retina from the underlying choroid (the vascular membrane that nourishes the retina). Retinal detachment is often seen in sports injuries, especially boxing. This painless condition produces flashing lights, specks, or “floaters” in the field of vision and a cloud or shade over the patient’s vision. Because it can cause devastating damage to vision, retinal detachment is an ocular emergency and requires immediate medical attention.

Burns of the Eye

Chemicals, heat, and light rays can all burn the delicate tissues of the eye, often causing permanent damage. Your role is to stop the burning process and prevent further damage.

Chemical burns, which are usually caused by acid or alkali solutions, require immediate emergency care. Flush the eye with water or a sterile saline solution for a minimum of 15 minutes and then seek medical attention.
Figure 21-31  Swelling and ecchymosis are hallmark findings associated with blunt trauma to the eye.

Figure 21-32  A hyphema, characterized by bleeding into the anterior chamber of the eye, can occur following blunt trauma to the eye. This condition should be considered a sight-threatening emergency. A. Actual hyphema. B. Illustration.

Figure 21-33  In a patient with a blowout fracture, the eyes may not move together because of muscle entrapment, so the patient sees double images of any object.

Figure 21-34  A. Chemical burns typically occur when an acid or alkali is splashed into the eye. B. A chemical burn from lye, an alkaline solution.
Assessment and Management of Eye Injuries

The first step in assessing a patient with an eye injury is to note the mechanism of injury (i.e., blunt or penetrating trauma, burn). If it suggests the potential for a spinal injury, use spinal motion restriction precautions. Ensure a patent airway and adequate breathing, and control any external bleeding. If the mechanism of injury is significant, or if the patient's clinical status dictates it, perform a rapid trauma assessment.

When obtaining the history, determine how and when the injury happened, when the symptoms began, and what symptoms the patient is experiencing. Were both eyes affected? Does the patient have any underlying diseases or conditions of the eye (such as glaucoma)? Does the patient take medications for his or her eyes?

A variety of symptoms may indicate serious ocular injury:
- **Visual loss** that does not improve when the patient blinks is the most important symptom of an eye injury. It may indicate damage to the globe or to the optic nerve.
- **Double vision** usually points to trauma involving the extraocular muscles, such as a fracture of the orbit.
- **Severe eye pain** is a symptom of a significant eye injury.
- A **foreign body sensation** usually indicates superficial injury to the cornea or the presence of a foreign object trapped behind the eyelids.

During the physical examination of the eyes, evaluate each of the visible ocular structures and ocular function:
- **Orbital rim:** for ecchymosis, swelling, lacerations, and tenderness
- **Eyelids:** for ecchymosis, swelling, and lacerations
- **Corneas:** for foreign bodies
- **Conjunctivae:** for redness, pus, inflammation, and foreign bodies
- **Globes:** for redness, abnormal pigmentation, and lacerations
- **Pupils:** for size, shape, equality, and reaction to light

In the Field

Anisocoria, a condition in which the pupils are not of equal size, is a significant finding in patients with ocular injuries or closed head trauma. However, simple or physiologic anisocoria occurs in approximately 20% of the population. Usually, the patient’s pupils differ in size by less than 1 mm; however, approximately 4% of people have pupils that vary in size by more than 1 mm. This is not a clinically significant finding.

Unilateral cataract surgery may also cause inequality of pupil size. The pupil of the eye affected by cataract will be nonreactive to light.
Eye movements in all directions: for paralysis of gaze or discoordination between the movements of the two eyes (dysconjugate gaze).

Visual acuity: Make a rough assessment by asking the patient to read a newspaper or a hand-held visual acuity chart. Test each eye separately and document the results.

Treatment for specific eye injuries begins with a thorough examination to determine the extent and nature of any damage. Always perform your examination using body substance isolation precautions, taking great care to avoid aggravating the injury.

Although isolated eye injuries are usually not life-threatening, they should be evaluated by a physician. More severe eye injuries often require evaluation and treatment by an ophthalmologist.

Injuries to the eyelids—lacerations, abrasions, and contusions—require little in the way of prehospital care other than bleeding control and gentle patching of the affected eye. No eyelid injury is trivial, however, so every patient with eyelid trauma should be transported to the hospital.

Most injuries to the globe—including contusions, lacerations, foreign bodies, and abrasions—are best treated in the emergency department, where specialized equipment is available. Aluminum eye shields (not gauze patches) applied over both eyes are generally all that are necessary in the field. Follow these three important guidelines in treating penetrating injuries of the eye:

1. Never exert pressure on or manipulate the injured globe in any way.
2. If part of the globe is exposed, gently apply a moist, sterile dressing to prevent drying.
3. Cover the injured eye with a protective metal eye shield, cup, or sterile dressing. Apply soft dressings to both eyes, and provide prompt transport to the hospital.

If hyphema or rupture of the globe is suspected, take spinal motion restriction precautions. Such injuries indicate that a significant amount of force was applied to the face and, thus, may include a spinal injury. Elevate the head of the backboard approximately 40° to decrease IOP and discourage the patient from performing activities that may increase IOP (e.g., coughing).

On rare occasions following a serious injury, the globe may be displaced (avulsed) out of its socket. Do not attempt to manipulate or reposition it in any way! Cover the protruding eye with a moist, sterile dressing and stabilize it along with the uninjured eye to prevent further injury due to sympathetic eye movement, the movement of both eyes in unison. Place the patient in a supine position to prevent further loss of fluid from the eye, and provide prompt transport to the hospital.

In the Field

As soon as you cover both of the patient’s eyes, he or she can no longer see. Therefore, you will have to serve as the patient’s eyes, keeping him or her constantly reassured and oriented to your location and what you are doing.

Burns to the eye that are caused by ultraviolet light are most effectively treated by covering the eye with a sterile, moist pad and an eye shield. The application of cool compresses lightly over the eye may afford the patient pain relief if he or she is in extreme distress. Place the patient in a supine position during transport, and protect the patient from further exposure to bright light.

Chemical burns to the eye—acid or alkali—can rapidly lead to total blindness if not immediately treated. The most important prehospital treatment in such cases is to begin immediate irrigation with sterile water or saline solution. Never use any chemical antidotes (such as vinegar, baking soda) when irrigating the patient’s eye; use sterile water or saline only.

The goal when irrigating the eye is to direct the greatest amount of solution or water into the eye as gently as possible. Because opening the eye spontaneously may cause the patient pain, you may have to force the lids open to irrigate the eye adequately. Ideally, you should use a bulb or irrigation syringe, a nasal cannula, or some other device that will allow you to control the flow. In some circumstances, you may have to pour water into the eye by holding the patient’s head under a gently running faucet. You can have the patient immerse his or her face in a large pan or basin of water and rapidly blink the affected eyelid. If only one eye is affected, take care to avoid contaminated water getting into the unaffected eye.

Irrigate the eye for at least 5 minutes. If the burn was caused by an alkali or a strong acid, irrigate the eye continuously for 20 minutes because these substances can penetrate deeply. One common possibility occurs where anhydrous ammonia is used during the process of cooking methamphetamine. If the eyes are not irrigated promptly and efficiently, permanent damage is likely. Whenever you have to irrigate the eye(s), continue to irrigate the eye en route to the hospital if possible.

Irrigation with a sterile saline solution will frequently flush away loose, small foreign objects lying on the surface of the eye. Always flush from the nose side of the eye toward the outside to avoid flushing material into the other eye. After its removal, a foreign body will often leave a small abrasion on the
Chapter 21 Head and Face Injuries

21.23

Injuries to the ear may be isolated, or they may occur in conjunction with other injuries to the head or face. Although isolated ear injuries are typically not life-threatening, they can result in sensory impairment and permanent disfigurement.

**Soft-Tissue Injuries**

Lacerations, avulsions, and contusions to the external ear can occur following blunt or penetrating trauma. The pinna can be
Trauma

The pinna has an inherently poor blood supply, so it tends to heal poorly. Healing of the cartilaginous pinna is often complicated by infection.

Ruptured Eardrum

Perforation of the tympanic membrane (ruptured eardrum) can result from foreign bodies in the ear or from pressure-related injuries, such as blast injuries resulting from an explosion, or diving-related injuries that result in barotrauma to the ear. Signs and symptoms of a perforated tympanic membrane include loss of hearing and blood drainage from the ear. Although the injury is extremely painful for the patient, the tympanic membrane typically heals spontaneously and without complication. Nevertheless, a careful assessment should be performed to detect and treat other injuries, some of which may be life threatening.

Assessment and Management of Ear Injuries

Assessment and management of the patient with an ear injury begins by ensuring airway patency and breathing adequacy. If the mechanism of injury suggests a potential for spinal injury, apply full spinal motion restriction precautions.

To remove a hard contact lens, use a small suction cup, moistening the end with saline Figure 21-39A. To remove soft lenses, instill 1 or 2 drops of saline or irrigating solution. Figure 21-39B. Gently pinch the lens between your gloved thumb and index fingers, and lift it off the surface of the eye Figure 21-39C. Place the contact lens in a container with sterile saline solution. Always advise emergency department staff if a patient is wearing contact lenses.

Figure 21-39 Removing contact lenses should be limited to patients with chemical burns to the eye. A. To remove hard contact lenses, use a specialized suction cup moistened with sterile saline solution. B. To remove soft contact lenses, instill 1 or 2 drops of saline or irrigating solution. C. Pinch off the lens with your gloved thumb and index fingers.

Special Considerations

Contact Lenses and Artificial Eyes

There are three types of contact lenses: hard, rigid gas-permeable, and soft (hydrophilic). Small, hard contact lenses usually are tinted, making them relatively easy to see. Large, soft contact lenses are clear and can be very difficult to see even more so if they “float” up or down under an eyelid.

In general, you should not attempt to remove contact lenses from a patient with an eye injury, lest you aggravate the injury. The only indication for removing contact lenses in the prehospital setting is a chemical burn of the eye. In this situation, the lens can trap the offending chemical and make irrigation difficult thus worsening the injury.

To remove a hard contact lens, use a small suction cup, moistening the end with saline Figure 21-39A. To remove soft lenses, place one to two drops of saline in the eye Figure 21-39B, gently pinch the lens between your gloved thumb and index finger, and lift it off the surface of the eye Figure 21-39C. Place the contact lens in a container with sterile saline solution. Always advise emergency department staff if a patient is wearing contact lenses.

Occasionally, you may care for a patient who is wearing an eye prosthesis (artificial eye). You should suspect an eye of being artificial when it does not respond to light, move in concert with the opposite eye, or appear quite the same as the opposite eye. If you are unsure as to whether the patient has an eye prosthesis, ask him or her. Although no harm will be done if you care for an artificial eye as you would a normal one, you need to be totally clear about the patient’s eye function. In addition, it can be quite embarrassing to pass on information during your radio report that the patient has a nonreactive pupil only to find out at the ED that the patient has a prosthetic eye.
dressing in place. An icepack can also help reduce swelling and pain.

If the pinna is partially avulsed, carefully realign the ear into position and gently bandage it with sufficient padding that has been slightly moistened with normal saline. If the pinna is completely avulsed, attempt to retrieve the avulsed part, if possible, for reimplantation at the hospital. If the detached part of the ear is recovered, treat it as any other amputation; wrap it in saline-moistened gauze, place it in a plastic bag, and place the bag on ice. If a chemical icepack is used, it is recommended to shield the avulsed part with several $4 \times 4$s to diffuse the cold, as chemical icepacks are actually colder than ice and inadvertent freezing of the part can occur.

If blood or CSF drainage is noted, apply a loose dressing over the ear—taking care not to stop the flow—and assess the patient for other signs of a basilar skull fracture.

Do not remove an impaled object from the ear. Instead, stabilize the object and cover the ear to prevent gross movement and minimize the risk of contamination of the inner ear.

Because isolated ear injuries are typically not life threatening, you must perform a careful assessment to detect or rule out potentially more serious injuries. You may then proceed with specific care of the ear, provide emotional support, and transport the patient to an appropriate medical facility.

Oral and Dental Injuries

Oral and dental injuries are commonly associated with trauma to the face. Blunt mechanisms are commonly the result of motor vehicle crashes or direct blows to the mouth or chin. Penetrating mechanisms are commonly the result of gunshot wounds, lacerations, and punctures.

The primary risk associated with oral and dental injuries is airway compromise from oropharyngeal bleeding, occlusion by a displaced dental appliance such as a bridge or partial plate, or possibly by the aspiration of avulsed or fractured teeth. Any patient with significant facial trauma should be carefully assessed for injuries to the mouth and teeth.

Soft-Tissue Injuries

Lacerations and avulsions in and around the mouth are associated with a risk of intraoral hemorrhage and subsequent airway compromise. Therefore, your assessment of any patient with facial trauma should include a careful examination of the mouth, including the teeth. Fractured or avulsed teeth and lacerations of the tongue may cause profuse bleeding into the upper airway. A conscious patient with severe oral bleeding is often unable to speak unless he or she is leaning forward—this position facilitates drainage of blood from the mouth.

Patients may swallow blood from lacerations inside the mouth, so the bleeding may not be grossly evident. Because blood irritates the gastric lining, the risks of vomiting and aspiration are significant. Objects that are impaled in or through
the soft tissues of the mouth (such as the cheek) can also result in profuse bleeding and once again, the threat of vomiting with aspiration.

**Dental Injuries**
Fractured and avulsed teeth—especially the anterior teeth—are common following facial trauma. Dental injuries may be associated with mechanisms that cause severe maxillofacial trauma (such as motor vehicle crashes), or they may occur in isolation (such as a direct blow to the mouth from an assault).

You should always assess the patient’s mouth following a facial injury, especially in cases of fractured or avulsed teeth. Teeth fragments (or even whole teeth) can become an airway obstruction and should be removed from the patient’s mouth immediately.

**In the Field**
When assessing a patient with fractured or avulsed teeth following an assault, you should also assess the individual who struck the patient, if it is safe to do so. The human mouth is filled with bacteria and other microorganisms, and lacerations to the person’s hands or knuckles can easily become infected without proper care. Occasionally, you may encounter fragments of broken teeth impaled in a person’s knuckles!

**Assessment and Management of Oral and Dental Injuries**
Ensuring airway patency and adequate breathing are the priorities of care when managing patients with oral or dental trauma. Suction the oropharynx as needed, and remove fractured tooth fragments to prevent airway compromise. Apply spinal motion restriction precautions as dictated by the mechanism of injury. If profuse oral bleeding is present and the patient cannot spontaneously control his or her own airway (such as with a decreased level of consciousness), pharmacologically assisted intubation (such as RSI) may be necessary.

Impaled objects in the soft tissues of the mouth should be stabilized in place unless they interfere with the patient’s breathing or your ability to manage the patient’s airway. In those cases, remove the impaled object from the direction that it entered, and control bleeding with direct pressure.

An avulsed tooth may be successfully reimplanted even if it has been out of the mouth for up to 1 hour. Medical control may sometimes ask you to reimplant the tooth in its original socket. Carefully place the tooth in its socket, and hold it in place with your fingers or have the patient gently bite down. If prehospital reimplantation of a tooth is not possible, follow the guidelines established by the American Association of Endodontists and the American Dental Association.

<table>
<thead>
<tr>
<th>Table 21-2</th>
<th>Care for an Avulsed Tooth</th>
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<tr>
<td>■ Handle the tooth by the crown only. Avoid touching the root surface of the tooth.</td>
<td></td>
</tr>
<tr>
<td>■ Gently rinse the tooth with sterile saline or water. Avoid the use of soap or chemicals, and do not scrub the tooth!</td>
<td></td>
</tr>
<tr>
<td>■ Do not allow the tooth to dry. Place it in one of the following:</td>
<td></td>
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<tr>
<td>— Emergency tooth preservation system (such as EMT Tooth Saver, 3M Save-a-Tooth): a break-resistant storage container with soft inner walls and a pH-balanced solution (such as Hanks Balanced Salt Solution) that nourishes and preserves the tooth</td>
<td></td>
</tr>
<tr>
<td>— Cold whole milk</td>
<td></td>
</tr>
<tr>
<td>— Sterile saline solution (for storage periods of less than 1 hour)</td>
<td></td>
</tr>
<tr>
<td>■ Transport the tooth with the patient, and notify the hospital of the situation.</td>
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Retrieval and reimplantation or storage of an avulsed tooth is a low priority if the patient is in a clinically unstable condition (such as compromised airway or shock). In such cases, aggressive airway management, spinal precautions, and rapid transport of the patient are obviously more important with the dental problem being addressed at a later time.

**Injuries to the Anterior Part of the Neck**
The neck is a very vulnerable stretch of anatomy because it houses a critical portion of the airway (ie, larynx, trachea), the major blood vessels to and from the head, and the spinal cord. Other structures contained within the neck that are also vulnerable to injury include muscles, nerves, and glands. Any injury to the anterior part of the neck—blunt or penetrating—must be considered critical until proved otherwise.

**Soft-Tissue Injuries**
Blunt and penetrating mechanisms can damage the soft tissues of the anterior part of the neck and its associated structures. In both cases, you must be alert for the possibility of cervical spine injury and airway compromise.

Common mechanisms of blunt trauma include motor vehicle crashes, direct trauma to the neck, and hangings. Such injury often results in swelling and edema; injury to the various structures such as the trachea, larynx, or esophagus; or injury to the cervical spine. Less commonly, blunt injuries may damage the vasculature of the anterior part of the neck. Because blunt trauma to the neck is associated with a high incidence of airway compromise and ventilatory inadequacy, you must carefully assess the patient and be prepared to initiate aggressive management.

Common mechanisms of penetrating trauma include gunshot wounds, stabbings, and impaled objects. The lacerations or puncture wounds produced may be superficial and involve
only the fascia or fatty tissues of the neck, or they may be deep and involve injury to the larynx, trachea, esophagus, nerves, or major blood vessels. The primary threats from penetrating neck trauma are massive hemorrhage from major blood vessel disruption and airway compromise secondary to soft-tissue swelling or direct damage to the larynx or trachea.

A special danger associated with open neck injuries is the possibility of a fatal air embolism. If the jugular veins of the neck are exposed to the environment, they can suck in air, resulting in a potentially fatal air embolism.

Impaled objects should not be removed but rather stabilized in place and protected from movement. The only exception is if the object is obstructing the airway or impeding your ability to effectively manage the airway. In some cases, an emergency cricothyrotomy may be necessary to establish and maintain airway patency.

**Injuries to the Larynx, Trachea, and Esophagus**

A variety of life-threatening injuries can result if the structures of the anterior part of the neck are crushed against the cervical spine following blunt trauma or if they are penetrated by a knife or similar object. The larynx and its supporting structures (ie, hyoid bone, thyroid cartilage) may be fractured, the trachea may be separated from the larynx (tracheal transection), or the esophagus may be perforated. Many injuries to the larynx, trachea, and esophagus are occult; because they are not as obvious and dramatic as penetrating neck injuries, they can be easily overlooked. Therefore, you must maintain a high index of suspicion and perform a careful assessment of any patient with blunt trauma to the anterior part of the neck.

Significant injuries to the larynx or trachea pose an immediate risk of airway compromise due to disruption of the normal passage of air, soft-tissue swelling, or aspiration of blood into the lungs. In addition, esophageal perforation can result in mediastinitis, an inflammation of the mediastinum often due to leakage of gastric contents into the thoracic cavity. Mediastinitis is associated with a high mortality rate if not surgically repaired in a timely manner.

Patients with injuries to the anterior part of the neck may experience concomitant maxillofacial fractures, which can make bag-mask ventilation difficult (usually because of an inadequate mask-to-face seal). Likewise, endotracheal intubation may be extremely challenging, if not impossible, owing to distortion of the normal anatomic structures of the upper airway. If basic and advanced techniques to secure the patient’s airway are unsuccessful or impossible, a surgical or needle cricothyrotomy may be your only means of establishing a patent airway and ensuring adequate oxygenation and ventilation. Prior to deciding to perform a surgical airway, use of a lighted stylette or a gum bougie may get the airway secured in a timely fashion while avoiding more risky procedures.
Begin your assessment by noting the mechanism of injury and maintaining a high index of suspicion, especially if the patient has experienced blunt or penetrating trauma between the upper part of the chest and head. Fractures of the first rib are associated with close to 50% mortality, not because of the rib fracture, but because the force it takes to fracture such a short, stout bone takes so much force that significant face, head, and neck trauma are almost always present as well. Remember that obvious and dramatic-appearing soft-tissue injuries may mask occult injuries to the larynx, trachea, or esophagus. Also, the patient may have experienced trauma to multiple body systems, especially following a significant mechanism of injury.

To control bleeding from an open neck wound and prevent air embolism, immediately cover the wound with an occlusive dressing. In the case of a small wound, or wounds, ECG electrodes can be fast and effective ways to seal a small hole or holes. Apply manual direct pressure over the occlusive dressing with a bulky dressing. As a last resort, you can secure a pressure dressing over the wound by wrapping roller gauze loosely around the neck and then firmly through the opposite axilla.

As you begin your initial assessment, manually stabilize the patient’s head in a neutral in-line position and simultaneously open the airway with the jaw-thrust maneuver if the patient is semiconscious or unconscious. Use suction as needed to clear the airway of blood or other liquids. Assess the patient’s breathing—rate, regularity, and depth—and intervene immediately. If the patient is breathing adequately, apply a nonrebreathing mask at 15 L/min. If breathing is inadequate (ie, reduced tidal volume, fast or slow respirations), assist with bag-mask ventilation and 100% oxygen.

Brusing, redness to the overlying skin, and palpable tenderness are common signs associated with all injuries to the anterior part of the neck. Table 21-3 summarizes the signs and symptoms of specific injuries.

If signs of shock are present, keep the patient warm, establish vascular access with at least one large-bore IV en route to the hospital if possible, or on-scene if indicated and infuse an isotonic crystalloid solution (such as lactated Ringer’s or normal saline) as needed to maintain adequate perfusion.

Many patients with serious laryngeal trauma require a surgical airway. Endotracheal intubation may be hazardous in such cases because you cannot see the tip of the ET tube once it passes between the vocal cords; it may pass straight through a defect in the laryngeal or tracheal wall or could result in the
complete transection of the trachea. Signs of this complication include increased swelling of the neck and worsening subcutaneous emphysema during assisted ventilation.

If the patient has experienced an open tracheal wound, you may be able to pass a cuffed ET tube directly through the wound to establish a patent airway. Use caution, however: The trachea may be perforated anteriorly and posteriorly, which would increase the risk of false passage of the ET tube outside the trachea. It is critical to use multiple techniques for confirming correct tube placement: frequently monitor breath sounds, use capnometry, assess for adequate chest rise, and assess for vapor mist in the ET tube during exhalation.

**Table 21-3** Signs and Symptoms of Injuries to the Anterior Part of the Neck

<table>
<thead>
<tr>
<th>Injury</th>
<th>Signs and Symptoms</th>
</tr>
</thead>
</table>
| Laryngeal fracture, tracheal transection | - Labored breathing or reduced air movement  
- Stridor  
- Hoarseness, voice changes  
- **Hemoptysis** (coughing up blood)  
- Subcutaneous emphysema  
- Swelling, edema |
| Vascular injury            | - Gross external bleeding  
- Signs of shock  
- Hematoma, swelling, edema  
- Pulse deficits |
| Esophageal perforation     | - **Dysphagia** (difficulty swallowing)  
- Hematemesis  
- Hemoptysis (suggests aspiration of blood) |
| Neurologic impairment      | - Signs of a stroke (suggests air embolism or cerebral infarct)  
- Paralysis or paresthesia  
- Cranial nerve deficit  
- Signs of neurogenic shock |

Controversies

Some clinicians advocate aiming for air bubbles, which indicate the general location of the glottis, when attempting to intubate a patient with a head, face, or neck injury and severe oropharyngeal bleeding. While this practice is dangerous, it can also be both practical and potentially the best approach! A common mechanism that might require this tactic is a failed suicide with a shotgun. As the patient attempts to pull the trigger with the toe, the gun shifts slightly and subsequent physical devastation to the front of the face, though not fatal, results in significant bleeding along with difficult airway management challenges. Whenever possible, suction the airway as needed to facilitate an adequate view of the vocal cords. If this is unsuccessful, you should consider performing a cricothyrotomy without delay if the “tube the bubbles” approach doesn’t work or doesn’t seem practical.

**Head Injuries**

A head injury is a traumatic insult to the head that may result in injury to soft tissue, bony structures, or the brain. Approximately 4 million people experience head injuries of varying severity in the United States each year. According to the Brain Trauma Foundation (BTF), 52,000 deaths occur annually as the result of severe head injury. More than 50% of all traumatic deaths result from a head injury. When head injuries are fatal, the cause is invariably associated injury to the brain.

Motor vehicle crashes are the most common mechanism of injury, with more than two thirds of people involved in motor vehicle crashes experiencing a head injury. Head injuries also occur commonly in victims of assault, when elderly people fall, during sports-related incidents, and in a variety of incidents involving children.

---

You are the Provider Part 4

You apply full spinal motion restriction precautions and quickly load the patient into the ambulance. You and your partner agree that the patient should be intubated. After administering the appropriate drugs, the ET tube is placed and placement is confirmed by auscultation and capnography.

<table>
<thead>
<tr>
<th>Reassessment</th>
<th>Recording Time: 10 Minutes</th>
</tr>
</thead>
<tbody>
<tr>
<td>Level of consciousness</td>
<td>Sedated and pharmacologically paralyzed, with a Glasgow Coma Scale score of 3</td>
</tr>
<tr>
<td>Respirations</td>
<td>Intubated and ventilated at a rate of 10 breaths/min</td>
</tr>
<tr>
<td>Pulse</td>
<td>70 beats/min; regular and bounding</td>
</tr>
<tr>
<td>Skin</td>
<td>Warm and dry</td>
</tr>
<tr>
<td>Blood pressure</td>
<td>160/100 mm Hg</td>
</tr>
<tr>
<td>( {\text{SaO}_2} )</td>
<td>98% (intubated and ventilated with 100% oxygen)</td>
</tr>
</tbody>
</table>

7. What do the patient’s vital signs indicate?
8. What else should you assess for in this patient?
Section 4 Trauma

There are two general types of head injuries: open and closed. A closed head injury (the most common type) is usually associated with blunt trauma. Although the dura mater remains intact and brain tissue is not exposed to the environment, closed head injuries may result in skull fractures, focal brain injuries, or diffuse brain injuries. Furthermore, these injuries are often complicated by increased ICP.

With an open head injury, the dura mater and cranial contents are penetrated, and brain tissue is open to the environment. Gunshot wounds—the most common penetrating mechanism of injury—have a high mortality rate, and for those who survive there is almost always significant neurologic deficit and a decreased quality of life.

Scalp Lacerations
Scalp lacerations can be minor or very serious. Because of the scalp’s rich blood supply, even small lacerations can lead to significant blood loss. Hypovolemic shock in adults is rarely caused by scalp lacerations alone; this is more common in children. However, bleeding from the scalp can contribute to hypovolemia in any patient, especially one with multiple injuries. In addition, because scalp lacerations usually result from direct blows to the head, they often indicate deeper, more severe injuries.

Skull Fractures
Four types of skull fractures are distinguished: linear, depressed, basilar, and open. The significance of a skull fracture is directly related to the type of fracture, the amount of force applied, and the area of the head that suffered the blow. Skull fractures are most commonly seen following motor vehicle crashes and significant falls. They may or may not be associated with soft-tissue scalp injuries. Potential complications of any skull fracture include intracranial hemorrhage, cerebral damage, and cranial nerve damage, among others.

**Linear Skull Fractures**
Linear skull fractures (nondisplaced skull fractures) account for approximately 80% of all fractures to the skull; approximately 50% of linear fractures occur in the temporal-parietal region of the skull (see Figure 21-46A). Radiographic evaluation is required to diagnose a linear skull fracture because there are often no gross physical signs (such as deformity, depression). If the brain is uninjured and the scalp is intact, linear fractures are relatively benign. However, if a scalp laceration occurs in conjunction with a linear fracture—making it an open fracture—there is a risk of infection. In addition, if the fracture occurs over the temporal region of the skull, injury to the middle meningeal artery may result in epidural bleeding.

**Depressed Skull Fractures**
Depressed skull fractures result from high-energy direct trauma to a small surface area of the head with a blunt object (such as...
a baseball bat to the head) (see Figure 21-46B). The frontal and parietal regions of the skull are most susceptible to these types of fractures because the bones in these areas, compared with other bones of the skull, are relatively thin. As a consequence, bony fragments may be driven into the brain, resulting in underlying injury. The overlying scalp may or may not be intact. Patients with depressed skull fractures often present with neurologic signs (such as loss of consciousness).

**Basilar Skull Fractures**

Basilar skull fractures also are associated with high-energy trauma, but they usually occur following diffuse impact to the head (e.g., falls, motor vehicle crashes). These injuries generally result from extension of a linear fracture to the base of the skull and can be difficult to diagnose with radiography (x-ray) (see Figure 21-46C).

Signs of a basilar skull fracture include CSF drainage from the ears[Figure 21-47](http://example.com), which indicates rupture of the tympanic membrane and freely flowing CSF through the ear. Patients with leaking CSF are at risk for bacterial meningitis.

Other signs of a basilar skull fracture include periorbital ecchymosis that develops under or around the eyes, which is also known as raccoon eyes[Figure 21-48A](http://example.com), or ecchymosis behind the ear over the mastoid process known as Battle’s sign[Figure 21-48B](http://example.com). Depending upon the extent of the damage, raccoon eyes and Battle’s sign may appear relatively quickly, but in many cases, they may not appear until up to 24 hours following the injury, so their absence in the prehospital setting does not rule out a basilar skull fracture.

**Open Skull Fractures**

Open fractures of the cranial vault result when severe forces are applied to the head and are often associated with trauma to multiple body systems (see Figure 21-46D). Brain tissue may be exposed to the environment, which significantly increases the risk of a bacterial infection (such as bacterial meningitis). Open cranial vault fractures have a high mortality rate.

**Traumatic Brain Injuries**

The National Head Injury Foundation defines a traumatic brain injury (TBI) as “a traumatic insult to the brain capable of producing physical, intellectual, emotional, social, and vocational changes.” Traumatic brain injuries are classified into two broad categories: primary (direct) injury and secondary (indirect) injury. **Primary brain injury** is injury to the brain and its associated structures that results instantaneously from impact to the head. **Secondary brain injury** refers to the “after effects” of the primary injury; it includes abnormal processes such as cerebral edema, intracranial hemorrhage, increased ICP, cerebral ischemia and hypoxia, and infection. Secondary brain injury can occur anywhere from a few minutes to several days following the initial injury.

The brain can be injured directly by a penetrating object, such as a bullet, knife, or other sharp object. More commonly,
Intracranial Pressure

For adults the skull is a rigid, unyielding globe that allows little, if any, expansion of the intracranial contents. It also provides a hard and somewhat irregular surface against which brain tissue and its blood vessels can be injured when the head suffers trauma.

Accumulations of blood within the skull or swelling of the brain can rapidly lead to an increase in intracranial pressure (ICP), the pressure within the cranial vault. Increased ICP squeezes the brain against bony prominences within the cranium.

Normal ICP in adults ranges from 0 to 15 mm Hg. An increase in ICP (such as from cerebral edema or intracranial hemorrhage) decreases cerebral perfusion pressure and cerebral blood flow. Cerebral perfusion pressure (CPP), the pressure of blood flow through the brain, is the difference between the mean arterial pressure (MAP), the average (or mean) pressure against the arterial wall during a cardiac cycle, and ICP (CPP = MAP – ICP). Obviously, decreasing cerebral blood flow is a potential catastrophe because the brain depends on a constant supply of blood to furnish the oxygen and glucose it needs to survive.

The critical minimum threshold, or minimum CPP required to adequately perfuse the brain, is 60 mm Hg in the adult. A CPP of less than 60 mm Hg will lead to cerebral ischemia, potentially resulting in permanent neurologic impairment or even death. In fact, according to the BTJ, a single drop in CPP below 60 mm Hg doubles the brain-injured patient’s chance of death!

The body responds to a decrease in CPP by increasing MAP, resulting in cerebral vasodilation and increased cerebral blood flow; this process is called autoregulation. However, an increase in cerebral blood flow causes a further increase in ICP. As ICP continues to increase, CSF is forced from the cranium into the spinal cord.

Clearly, the patient with increased ICP is caught in the midst of a vicious cycle. As ICP increases, cerebral blood flow increases secondary to autoregulation, which in turn leads to a potentially fatal increase in ICP. Conversely, if cerebral blood flow decreases, CPP decreases as well, and the brain becomes ischemic.

CPP cannot be calculated in the prehospital setting. Therefore, prehospital treatment must focus on maintaining CPP (and cerebral blood flow), while mitigating ICP as much as possible—a very fine balance to maintain.

If increased ICP is not promptly treated in a definitive care setting, cerebral herniation may occur. In herniation, the brain is forced from the cranial vault, either through the foramen magnum or over the tentorium.

You must closely monitor the head-injured patient for signs and symptoms of increased ICP. The exact clinical signs encountered depend on the amount of pressure inside the skull and the extent of brain stem involvement. Early signs and symptoms include vomiting (often without nausea), headache, an altered level of consciousness, and seizures. Later, more ominous signs include hypertension (with a widening pulse pressure), bradycardia, and irregular respirations (Cushing’s triad), plus a unilaterally unequal and nonreactive pupil (caused by oculomotor nerve compression), coma, and posturing. Decorticate (flexor) posturing is characterized by flexion of
Diffuse Axonal Injury

Diffuse axonal injury (DAI) is associated with or similar to a concussion. Unlike a concussion, however, this more severe diffuse brain injury is often associated with a poor prognosis. DAI involves stretching, shearing, or tearing of nerve fibers with subsequent axonal damage. An axon is a long, slender extension of a neuron (nerve cell) that conducts electrical impulses away from the neuronal soma (cell body) in the brain.

DAI most often results from high-speed, rapid acceleration-deceleration forces (such as motor vehicle crashes, significant falls). The severity and, thus, the prognosis of DAI depends on the degree of axonal damage (ie, stretching versus shearing or tearing); DAI is classified as being mild, moderate, or severe.

Focal Brain Injuries

A focal brain injury is a specific, grossly observable brain injury (ie, it can be seen on a CT scan). Such injuries include cerebral contusions and intracranial hemorrhage.

Cerebral Contusion

In a cerebral contusion, brain tissue is bruised and damaged in a local area. Because a cerebral contusion is associated with physical damage to the brain, greater neurologic deficits (such as prolonged confusion, loss of consciousness) are more commonly observed than with a concussion. The same mechanisms of injury that cause concussions—acceleration-deceleration forces and direct blunt head trauma—also cause cerebral contusions.

The area of the brain most commonly affected by a cerebral contusion is the frontal lobe, although multiple areas of contusion can occur, especially following coup-contrecoup injuries. As with any bruise, the reaction of the injured tissue will be to swell. This swelling inevitably leads to increased ICP and the negative consequences that accompany it.

Intracranial Hemorrhage

The closed box of the skull has no extra room for accumulation of blood, so bleeding inside the skull also increases ICP. Bleeding can occur between the skull and dura mater, beneath the
dura mater but outside the brain, within the parenchyma (tissue) of the brain itself (intracerebral space), or into the CSF (subarachnoid space).

**Epidural Hematoma**

An epidural hematoma is an accumulation of blood between the skull and dura mater; it occurs in approximately 0.5% to 1% of all head injuries.

An epidural hematoma is nearly always the result of a blow to the head that produces a linear fracture of the thin temporal bone. The middle meningeal artery courses along a groove in that bone, so it is prone to disruption when the temporal bone is fractured. In such a case, brisk arterial bleeding into the epidural space will result in rapidly progressing symptoms.

Often, the patient loses consciousness immediately following the injury; this is often followed by a brief period of consciousness ("lucid interval"), after which the patient lapses back into unconsciousness. Meanwhile, as ICP increases, the oculomotor nerve (third cranial nerve) is compressed against the tentorium, and the pupil on the side of the hematoma becomes fixed and dilated. Death will follow very rapidly without surgery to evacuate the hematoma.

**Subdural Hematoma**

A subdural hematoma is an accumulation of blood beneath the dura mater but outside the brain. It usually occurs after falls or injuries involving strong deceleration forces and occurs in approximately 5% of all head injuries. Subdural hematomas are more common than epidural hematomas and may or may not be associated with a skull fracture. Bleeding within the subdural space typically results from rupture of the veins that bridge the cerebral cortex and dura.

A subdural hematoma is associated with venous bleeding, so this type of hematoma—and the signs of increased ICP—typically develops more gradually than with an epidural hematoma. The patient with a subdural hematoma often

---

**Table 21-4**  **Diffuse Axonal Injury**

<table>
<thead>
<tr>
<th>Pathophysiology</th>
<th>Incidence</th>
<th>Signs and Symptoms</th>
<th>Prognosis</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Mild DAI</strong></td>
<td>Most common result of blunt head trauma; concussion is an example</td>
<td>Loss of consciousness (brief, if present); confusion, disorientation, amnesia (retrograde and/or anterograde)</td>
<td>Minimal or no permanent neurologic impairment</td>
</tr>
<tr>
<td><strong>Moderate DAI</strong></td>
<td>20% of all severe head injuries; 45% of diffuse axonal injures</td>
<td>Immediate loss of consciousness: secondary to involvement of the cerebral cortex or the reticular activating system of the brain stem; Residual effects: persistent confusion and disorientation; cognitive impairment (e.g., inability to concentrate); frequent periods of anxiety; uncharacteristic mood swings; sensory/motor deficits (such as altered sense of taste or smell)</td>
<td>Survival likely, but permanent neurologic impairment common</td>
</tr>
<tr>
<td><strong>Severe DAI</strong></td>
<td>16% of all severe head injuries; 36% of diffuse axonal injures</td>
<td>Immediate and prolonged loss of consciousness; posturing and other signs of increased ICP</td>
<td>Survival unlikely; most patients who survive never regain consciousness but remain in a persistent vegetative state</td>
</tr>
</tbody>
</table>

---

**In the Field**

It is generally not possible, or necessary, to distinguish between a cerebral contusion and intracranial hemorrhage in the prehospital setting. Instead, you should recognize the signs of increasing ICP and appreciate that those signs represent a critically injured patient who needs immediate treatment and prompt transport to an appropriate facility.
Chapter 21  Head and Face Injuries  21.35

You are the Provider Part 5

Your patient’s condition is clearly critical, so you ask one of the police officers to drive the ambulance to a trauma center located approximately 15 miles away to free up your partner to work in the back with you. En route to the hospital, you establish two large-bore IV lines with normal saline and apply the cardiac monitor. The patient’s pupils are bilaterally dilated and sluggish to react.

<table>
<thead>
<tr>
<th>Reassessment</th>
<th>Recording Time: 15 Minutes</th>
</tr>
</thead>
<tbody>
<tr>
<td>Level of consciousness</td>
<td>Sedated and pharmacologically paralyzed, with a Glasgow Coma Scale score of 3</td>
</tr>
<tr>
<td>Respirations</td>
<td>Intubated and ventilated at a rate of 10 breaths/min</td>
</tr>
<tr>
<td>Pulse</td>
<td>50 beats/min; regular and bounding</td>
</tr>
<tr>
<td>Skin</td>
<td>Warm and dry</td>
</tr>
<tr>
<td>Blood pressure</td>
<td>180/90 mm Hg</td>
</tr>
<tr>
<td>$\text{SaO}_2$</td>
<td>98% (intubated and ventilated with 100% oxygen)</td>
</tr>
</tbody>
</table>

9. Should this patient receive IV fluid boluses? When are IV fluid boluses indicated in a head-injured patient?

10. When is hyperventilation indicated for a head-injured patient? What ventilation rate defines hyperventilation in an adult?
A sudden, severe subarachnoid hematoma usually results in death. People who survive often have permanent neurologic impairment.

### Assessment and Management of Head and Brain Injuries

Prehospital assessment and management of the head-injured patient should be guided by factors such as the severity of the injury and the patient's level of consciousness. As with any patient, your treatment priorities must be based on what will kill the patient first.

#### Assessment of Head and Brain Injuries

Motor vehicle crashes, direct blows, falls from heights, assault, and sports-related injuries are common causes of head and traumatic brain injuries. A patient who has experienced any of these events should immediately elevate your index of suspicion and prompt a search for signs and symptoms of these types of injuries.

- A deformed windshield or dented or cracked helmet indicates a major blow to the head.

### Level of Consciousness

A change in the level of consciousness is the single most important observation that you can make when assessing the severity of brain injury. The level of consciousness usually indicates the extent of brain dysfunction. Whenever you suspect a head injury, you should perform a baseline neurologic assessment using the AVPU scale (Alert; responsive to Verbal stimuli; responsive to Pain; Unresponsive) and record the time.

### Documentation and Communication

A single assessment of the patient’s GCS score cannot reliably capture his or her clinical progression. Obtain a baseline GCS score and frequently (at least every 5 minutes if possible) reassess it in a head-injured patient. Document all GCS scores and the times they were obtained on the patient care report. The physician will compare his or her neurologic assessment with those you performed in the field.
Chapter 21  Head and Face Injuries

21.37  Pupillary Assessment

Frequently monitor the size, equality, and reactivity of the patient's pupils. The nerves that control dilation and constriction of the pupils are very sensitive to ICP. When you shine a light into the eye, the pupil should briskly constrict. A pupil that is slow (sluggish) to constrict is a relatively early sign of increased ICP; a sluggish pupil could also indicate cerebral hypoxia.

Unequal or bilaterally fixed and dilated (“blown”) pupils are later, more ominous signs of increased ICP and indicate pressure on one or both oculomotor nerves.

Figure 21-55  Glasgow Coma Scale scores should be assessed frequently in head-injured patients. The lower the score, the more severe the extent of brain injury.

Table 21-6  Brain Injury Classification Based on the GCS

- 13 to 15. Mild traumatic brain injury
- 8 to 12. Moderate traumatic brain injury
- 3 to 8. Severe traumatic brain injury

Notes from Nancy

The most important aspect of neurologic assessment is whether the patient's findings are changing and in what direction.

Assessing ICP

Although ICP cannot be quantified (assigned a numeric value) in the prehospital setting, the severity of increase can be estimated based on the patient's clinical presentation.

Critical treatment decisions for brain-injured patients are based...
on the presence or absence of certain key findings—specifically, posturing, hypotension or hypertension, and abnormal pupil signs. Use serial assessments of the patient’s GCS scores and pupillary assessment as indicators of the progression of ICP.

**Management of Head and Brain Injuries**

Patients with head injuries often have cervical spine injuries as well. Therefore, as you begin your initial assessment of a head-injured patient, manually stabilize the cervical spine in a neutral, in-line position. Avoid moving the neck unnecessarily, and continue manual stabilization until full spinal motion restriction precautions have been applied.

**Managing Airway and Breathing**

The most important step in the treatment of any type of head injury is to establish and maintain a patent airway. Open the airway with the jaw-thrust maneuver if the patient is semiconscious or unconscious or is otherwise unable to maintain his or her own airway spontaneously.

Patients with a head injury often vomit (especially children). Therefore, after opening the airway, you must be prepared to roll the patient to the side—while maintaining spinal stabilization—to prevent aspiration. If it is safe to do so, manually remove any large debris from the patient’s mouth by sweeping the oropharynx with your gloved finger. Use suction to clear secretions, such as blood or thin secretions from the oropharynx. Mortality increases significantly if aspiration occurs.

If the patient has a decreased level of consciousness, insert a basic airway adjunct (ie, oral or nasal airway). Do not insert a nasal airway if CSF or bloody rhinorrhea is present or if you suspect a nasal fracture.

After you have cleared the airway, assess the patient’s ventilatory status. Cerebral edema and ICP are aggravated by hypoxia and hypercarbia; therefore, you must constantly ensure adequate oxygenation and ventilation in any patient with a head injury.

Administer 100% oxygen via nonrebreathing mask if the patient is breathing adequately (ie, adequate rate and depth [tidal volume], regular respiratory pattern). An injured brain is even less tolerant of hypoxia than a healthy one, and research has demonstrated that prompt administration of supplemental oxygen can reduce the amount of brain damage and improve neurologic outcome.

If the respiratory center of the brain (pons, medulla) has been injured, the rate, depth, or regularity of breathing may be ineffective. Ventilation may also be impaired by concomitant chest injuries or, if the spinal cord is injured, by paralysis of some or all of the respiratory muscles. Patients with inadequate ventilation, especially if associated with a decreased level of consciousness, should receive bag-mask ventilation and 100% oxygen. Ventilate a brain-injured adult at a rate of 10 breaths/min or as dictated by local protocols. Avoid routine hyperventilation of brain-injured patients. Although hyperventilation causes cerebral vasoconstriction, which will shunt blood from the cranium and lower ICP, this outcome will merely provide additional room for the injured brain to swell or for more blood to accumulate in the skull. Most important, cerebral vasoconstriction shunts oxygen away from the brain, resulting in a drop in CPP and bringing on cerebral ischemia. The BTG recommends hyperventilation (20 breaths/min for adults) only if signs of cerebral herniation are present. In such brain-injured patients, brief periods of hyperventilation may be beneficial. If available, end-tidal carbon dioxide (ETCO2) should be monitored with digital capnometry. Optimally, you should ventilate

**You are the Provider Part 6**

Your estimated time of arrival at the trauma center is 5 minutes. Without time to perform a detailed physical examination, you reassess the patient’s vital functions and call in your radio report to the receiving facility.

<table>
<thead>
<tr>
<th>Reassessment</th>
<th>Recording Time: 20 Minutes</th>
</tr>
</thead>
<tbody>
<tr>
<td>Level of consciousness</td>
<td>Sedated and pharmacologically paralyzed, with a Glasgow Coma Scale score of 3</td>
</tr>
<tr>
<td>Respiration</td>
<td>Intubated and ventilated at a rate of 10 breaths/min</td>
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<td>Skin</td>
<td>Warm and dry</td>
</tr>
<tr>
<td>Blood pressure</td>
<td>170/80 mm Hg</td>
</tr>
<tr>
<td>S&lt;sub&gt;O2&lt;/sub&gt;</td>
<td>98% (intubated and ventilated with 100% oxygen)</td>
</tr>
</tbody>
</table>

11. Should you be concerned with the exact etiology of this patient’s head injury? Why or why not?
12. What are the most important interventions you can perform to maintain cerebral perfusion in this patient?
the patient to maintain $ETCO_2$—an approximation of arterial $PACO_2$—between 30 and 40 mm Hg. Under no circumstances should the $PACO_2$ be allowed to ever drop below 25 mm Hg as the subsequent vasoconstriction will almost assuredly result in brain death due to anoxia.

Endotracheal intubation of a head-injured patient requires special precautions or it may precipitate dangerous increases in ICP. If intubation of a head-injured patient is required (eg, unresponsive, unable to effectively perform bag-mask ventilation), observe the following guidelines:

1. Preoxygenate with 100% oxygen for at least 2 to 3 minutes.
2. Administer 1 to 1.5 mg/kg of lidocaine IV push. Lidocaine has been shown to blunt an acute increase in ICP, which may occur during intubation.
3. Perform intubation with the patient's head in a neutral in-line position. Intubation of a head-injured patient or any patient with significant trauma should be performed by two people: one to maintain manual stabilization of the patient's head and the other to intubate.

If a head-injured patient requires intubation but will otherwise not tolerate laryngoscopy and ET tube placement (for example, because of combativeness or clenched teeth [trismus]), perform pharmacologically assisted intubation (ie, RSI). This procedure involves using a sedative-hypnotic drug (such as midazolam [Versed]) and a neuromuscular blocking drug (such as vecuronium bromide [Norcuron] or rocuronium bromide [Zemuron]) to facilitate placement of the ET tube. (Refer to Chapter 11 for more on pharmacologically assisted intubation.)

Closely monitor the patient's oxygen saturation ($SaO_2$), and maintain it at 95% or higher.

Severe head injuries, especially if the lower brain stem is involved, can produce a variety of cardiac rhythm disturbances, so use a cardiac monitor for any critically injured patient. Use of a cardiac monitor also allows you to monitor the patient for acute heart rate changes. If cardiac arrest occurs, follow the ACLS pulseless arrest protocol.

**Other Management**

**Thermal Management**

Do not allow the patient to become overheated. Patients with head injury, unlike those with shock, can develop a very high body temperature (hyperpyrexia), which in turn may worsen the condition of the brain. Do not cover the patient with blankets if the ambient temperature is 70°F (21°C) or higher.

**Treatment of Associated Injuries**

If the patient has an open fracture of the skull with brain tissue oozing out, cover it lightly with a sterile dressing that has been moistened with sterile saline. Likewise, for leakage of CSF from the ears or nose, apply loose sterile dressings, just to keep the area clean. Objects impaled in the skull should be stabilized in place and protected from being jarred.
Pharmacologic Therapy
Pharmacologic therapy, other than that used to facilitate intubation or treat seizures, is usually not indicated for brain-injured patients in the prehospital setting. However, if transport will be prolonged, medical control may order the administration of certain medications, such as mannitol (Osmotrol) and/or furosemide (Lasix) to reduce cerebral edema and decrease ICP.

Seizures in a brain-injured patient must be terminated as soon as possible, lest they provoke further increases in ICP or body temperature. Benzodiazepines, such as diazepam (Valium) or lorazepam (Ativan), should be used to control seizure activity in brain-injured patients. Follow local protocol or contact medical control regarding the doses of these drugs.

Transport Considerations
Prompt transport to a definitive care facility (ie, a trauma center) is crucial to survival of a brain-injured patient. If available, consider air transport if your transport time will be prolonged. If transporting the patient by ground, do so expeditiously, yet cautiously; the use of lights and a siren could precipitate seizures and exacerbate ICP.

Many patients with severe brain injuries and increased ICP require neurosurgical intervention. The extra time it takes to move the patient from one hospital to another could mean the difference between life and death. Therefore, transport the patient directly to a trauma center that has neurosurgical capabilities, even if it means bypassing the nearest hospital.
Head and Face Injuries

Chapter 21

1. What should be your initial concern about this patient?
   Any motorcycle collision is a significant mechanism of injury, but the fact that the patient was not wearing a helmet dramatically increases his risk of mortality due to a severe head injury. This should be your primary concern, especially considering your general impression of the patient (lying on the ground, not moving).

2. How should you direct your initial care of this patient?
   Your first action is to manually stabilize the patient’s head while simultaneously opening his airway with the jaw-thrust maneuver. Then, you should perform an initial assessment to identify and treat life-threatening injuries and/or conditions.

3. How will you manage this patient’s airway?
   This patient’s airway is in serious jeopardy! He has two major airway problems that must be addressed promptly: blood in the oropharynx, which places him at immediate risk for aspiration, and slow, irregular respirations that are clearly not adequate. You should suction his oropharynx for 15 seconds and then proceed with bag-mask ventilation with 100% oxygen for 2 minutes. Continue this alternating pattern of suctioning and ventilating until his airway is clear or has been secured with an ET tube.

4. When would it be appropriate to intubate this patient?
   Do not immediately intubate! Endotracheal intubation should not be performed before adequate oxygenation and ventilation have been achieved with basic means (ie, bag-mask ventilation and airway adjunct). Furthermore, you must clear this patient’s airway with suction first because (1) blood in the airway will make for a difficult intubation and (2) attempting to intubate when you know it will be difficult—especially if the attempt would clearly be facilitated by clearing the airway with suction—will merely increase the risk of hypoxia. Few situations necessitate immediate intubation in any patient; this patient is no exception.

5. How can facial trauma complicate airway management?
   Many factors can complicate airway management in a patient with facial trauma, including blood in the airway and unstable facial bones (makes for a difficult mask-to-face seal). Sometimes facial trauma is a mirror image of the mouth and upper airway structures—devastation! You should, therefore, anticipate a difficult intubation and be prepared to secure the airway with other means (such as cricothyrotomy) if intubation is unsuccessful or impossible.

6. Is this patient in hypovolemic shock? Why or why not?
   Although the mechanism of injury and bilaterally deformed femurs are clearly a recipe for hypovolemic shock, the patient’s present vital signs are not suggestive of this diagnosis. Typical signs of shock include rapid, shallow respirations while this patient’s are slow and irregular; a rapid, weak pulse (his pulse quality is bounding); and cool, clammy skin (his skin is warm and dry). Although hypotension is a late sign of shock—and a normal BP does not rule out shock—young patient’s BP (140/90 mm Hg) is higher than one would expect, even in a state of compensated shock. Perform a careful assessment to detect any early signs of shock, and implement immediate treatment should they appear, before the patient’s condition deteriorates.

7. What do the patient’s vital signs indicate?
   Hypertension, a slowing pulse rate, and irregular respirations, especially with a mechanism of injury that suggests head injury (motorcycle crash without a helmet), constitute the Cushing’s triad, a classic sign of intracranial pressure. When the brain is injured, the body’s autoregulatory mechanism shunts more blood to the injured brain, which causes a rise in systemic arterial BP and reflex bradycardia. Brain stem involvement results in a variety of abnormal respiratory patterns (such as Cheyne-Stokes respirations, central neurogenic hyperventilation, Biot’s respirations).

8. What else should you assess for in this patient?
   In addition to closely monitoring the patient’s BP and pulse rate, you should perform and document serial assessments of his GCS scores. Other signs to assess for in the head-injured patient include cranial deformities or depressions; pupil size, equality, and reactivity; abnormal posturing (decorticate and/or decerebrate); CSF rhinorrhea or otorrhea; Battle’s sign; and raccoon eyes. Battle’s sign and raccoon eyes are often not evident in the prehospital setting.

9. Should this patient receive IV fluid boluses? When are IV fluid boluses indicated in a head-injured patient?
   In the absence of hypotension, IV fluids should be restricted in a head-injured patient to avoid increasing ICP; set your IV line(s) to keep the vein open at between 25 to 50 mL/hour. Persistent hypotension in a head-injured adult indicates lower brain stem herniation or occult hemorrhage elsewhere in the body. Regardless of the etiology, a single episode of hypotension in a head-injured adult (systolic BP < 90 mm Hg) can be lethal; it results in decreased CPP and cerebral ischemia and doubles mortality. Closely monitor the patient’s BP, and be prepared to infuse isotonic crystalloids to maintain a systolic BP of at least 90 mm Hg.

10. When is hyperventilation indicated for a head-injured patient?
    What ventilation rate defines hyperventilation in an adult?
    Brief periods of hyperventilation in a brain-injured adult (20 breaths/min) may be beneficial and are indicated only if signs of cerebral herniation are present. Signs of cerebral herniation include an unresponsive patient with both of the following: (1) unequal pupils or bilaterally fixed and dilated pupils and (2) decerebrate (extensor) posturing or no motor response to painful stimuli. Do not routinely hyperventilate brain-injured patients because doing so will cause cerebral vasoconstriction. Although hyperventilation may transiently decrease ICP by shunting blood from the brain, it may also decrease CPP and may cause cerebral ischemia.

11. Should you be concerned with the exact etiology of this patient’s head injury? Why or why not?
    It is not possible to determine the exact etiology of this patient’s head injury in the prehospital setting. The underlying injury that is causing his increased ICP can be determined only in a trauma center with a computed tomographic (CT) scan of the head. Your role is to recognize the signs of increased ICP, provide aggressive prehospital treatment to maintain CPP, and rapidly transport the patient to an appropriate facility.

12. What are the most important interventions you can perform to maintain cerebral perfusion in this patient?
    Three factors will cause CPP (and cerebral blood flow) to fall: hypoxia, hypercarbia, and hypotension. Therefore, the most important prehospital interventions for a brain-injured adult with increased ICP include constantly maintaining adequate oxygenation and ventilation and maintaining a systolic BP of at least 90 mm Hg. To do so, you must ensure that the patient’s airway remains patent and monitor his or her oxygen saturation and blood pressure closely. Ventilation rates should be dictated by the presence or absence of signs of brain herniation. ETCO2 monitoring can assist you in approximating the patient’s Paco2 level, which should be maintained in a range of 30 to 40 mm Hg (slightly decreased from normal).
A strong working knowledge of anatomy and physiology of the face, head, and brain is essential to accurately assess and manage patients with injuries to these locations.

Personal safety is your primary concern when treating any patient with head or face trauma; never enter an unsafe scene.

Head and face trauma most often result from direct trauma or rapid acceleration-deceleration forces.

Trauma to the face can range from a broken nose to more severe injuries, including massive soft-tissue trauma, maxillofacial fractures, oral or dental trauma, and eye injuries.

Your primary concerns with assessing and managing a patient with facial trauma are to ensure a patent airway and maintain adequate oxygenation and ventilation.

Remove impaled objects in the face or throat only if they impair airway patency or breathing or if they interfere with your ability to effectively manage the airway. Otherwise, stabilize them in place and protect them from being jarred.

Never remove impaled objects from the eye; stabilize them in place and put a protective cone (such as a cup) over the object to prevent accidental movement along with bandaging the unaffected eye to prevent sympathetic movement.

Flush burns to the eye with copious amounts of sterile saline or sterile water. Never use chemical antidotes when treating burn injuries to the eye.

The primary threat from oral or dental trauma is oropharyngeal bleeding and aspiration of blood or broken teeth. Keep the airway clear, and ensure adequate oxygenation and ventilation. Endotracheal intubation may be required.

A patient with head or face trauma should be suspected of having a spinal injury. Apply spinal motion restriction precautions as indicated.

The skull is a rigid, unyielding box that does not accommodate a swelling brain or accumulations of blood.

Normal ICP is 0 to 15 mm Hg in adults. Increased ICP can squeeze the brain against the interior of the skull and/or press it into sharp edges within the cranium. If severely increased ICP is not promptly treated, cerebral herniation will occur.

CPP is the pressure of blood flowing through the brain; it is the difference between the MAP and ICP.

If CPP drops below 60 mm Hg in the adult, cerebral ischemia will likely occur, resulting in permanent brain damage or death.

Begin treatment of a head-injured patient by stabilizing the cervical spine, opening the airway with the jaw-thrust maneuver, and assessing the ABCs.

All head-injured patients should receive 100% oxygen as soon as possible. If the patient is breathing adequately, apply a nonrebreathing mask set at 15 L/min. If the patient is breathing inadequately, assist ventilation and consider intubation.

Ventilate a brain-injured adult at a rate of 10 breaths/min. Avoid routine hyperventilation unless signs of cerebral herniation are present. Hyperventilation in a brain-injured adult is defined as a ventilation rate of 20 breaths/min.

Restrict IV fluids in a head-injured patient unless hypotension (systolic BP < 90 mm Hg) is present. Hypotension in a brain-injured patient should be treated with crystalloid fluid boluses in a quantity sufficient to maintain a systolic BP of at least 90 mm Hg.

Frequently monitor a head-injured patient’s level of consciousness, and document your findings. The GCS is an effective, reliable tool. The GCS must be repeated frequently if it is to be a reliable indicator of the patient’s clinical progression.

Intubation of a brain-injured patient may require pharmacologic adjuncts (such as sedation, neuromuscular-blocking drugs).

Seizures may occur in a brain-injured patient and can aggravate ICP and cause or worsen cerebral ischemia. Treat seizures with a benzodiazepine (such as diazepam, lorazepam).

A brain-injured patient’s survival depends on recognition of the injury, prompt and aggressive prehospital care, and rapid transport to a trauma center that has neurosurgical capabilities. Consider air transport if ground transport time will be prolonged.

**Vital Vocabulary**

alveolar ridges The ridges between the teeth, which are covered with thickened connective tissue and epithelium.

alveoli Small pits or cavities, such as the sockets for the teeth.

anisocoria A condition in which the pupils are not of equal size.

anterior chamber The anterior area of the globe between the lens and the cornea that is filled with aqueous humor.

anterograde (posttraumatic) amnesia Loss of memory relating to events that occurred after the injury.

aqueous humor The clear, watery fluid in the anterior chamber of the globe.

arachnoid The middle membrane of the three meninges that enclose the brain and spinal cord.

auricle The large outside portion of the ear through which sound waves enter the ear; also called the pinna.

autoregulation An increase in mean arterial pressure to compensate for decreased cerebral perfusion pressure; compensatory response of the body to shunt blood to the brain; manifests clinically as hypertension.

axon Long, slender extension of a neuron (nerve cell) that conducts electrical impulses away from the neuronal soma.

basal ganglia Structures located deep within the cerebrum, diencephalon, and midbrain that have an important role in coordination of motor movements and posture.

basilar skull fractures Usually occur following diffuse impact to the head (such as falls, motor vehicle crashes); generally result from extension of a linear fracture to the base of the skull and can be difficult to diagnose with a radiograph (x-ray).

Battle’s sign Bruising over the mastoid bone behind the ear commonly seen following a basilar skull fracture; also called retroauricular ecchymosis.

Biot’s respirations Characterized by an irregular rate, pattern, and volume of breathing with intermittent periods of apnea; also called ataxic respirations.

blowout fracture A fracture to the floor of the orbit usually caused by a blow to the eye.

brain Part of the central nervous system located within the cranium; contains billions of neurons that serve a variety of vital functions.

brain stem The midbrain, pons, and medulla, collectively.

central neurogenic hyperventilation Deep, rapid respirations; similar to Kussmaul, but without an acetone breath odor; commonly seen following brain stem injury.

central vision The visualization of objects directly in front of you.

cerebellum The region of the brain essential in coordinating muscle movements in the body; also called the athlete’s brain.

cerebral concussion Occurs when the brain is jarred around in the skull; a mild diffuse brain injury that does not result in structural damage or permanent neurologic impairment.
cerebral contusion A focal brain injury in which brain tissue is bruised and damaged in a defined area.
cerebral cortex The largest portion of the cerebrum; regulates voluntary skeletal movement and one’s level of awareness—a part of consciousness.
cerebral edema Cerebral water; causes or contributes to swelling of the brain.
cerebral perfusion pressure (CPP) The pressure of blood flow through the brain; the difference between the mean arterial pressure (MAP) and intracranial pressure (ICP).
cerebrospinal fluid (CSF) Fluid produced in the ventricles of the brain that flows in the subarachnoid space and bathes the meninges.
cerebrospinal rhinorrhea Cerebrospinal fluid drainage from the nose.
cerebrum The largest portion of the brain; responsible for higher functions, such as reasoning; divided into right and left hemispheres, or halves.
Cheyne-Stokes respirations The respirations that are fast and then become slow, with intervening periods of apnea; commonly seen following brain stem injury.
choroid plexus Specialized cells within the hollow areas in the ventricles of the brain that produce CSF.
cochlea The shell-shaped structure within the inner ear that contains the organ of Corti.
cochlear duct A canal within the cochlea that receives vibrations from the ossicles.
conjunctiva A thin, transparent membrane that covers the sclera and internal surfaces of the eyelids.
conjunctivitis An inflammation of the conjunctivae that usually is caused by bacteria, viruses, allergies, or foreign bodies; should be considered highly contagious; also called pink eye.
cornea The transparent anterior portion of the eye that overlies the iris and pupil.
coronal suture The point where the parietal bones join with the frontal bone.
coup-contrecoup injury Dual impacting of the brain into the skull; coup injury occurs at the point of impact; contrecoup injury occurs on the opposite side of impact, as the brain rebounds.
cranial vault The bones that encase and protect the brain, including the parietal, temporal, frontal, occipital, sphenoid, and ethmoid bones; also called the cranium or skull.
craniofacial disjunction A Le Fort III fracture; involves a fracture of all of the midfacial bones, thus separating the entire midface from the cranium.
cribiform plate A horizontal bone perforated with numerous foramina for the passage of the olfactory nerve filaments from the nasal cavity.
crista galli A prominent bony ridge in the center of the anterior fossa and the point of attachment of the meninges.
critical minimum threshold Minimum cerebral perfusion pressure required to adequately perfuse the brain; 60 mm Hg in the adult.
crown The part of the tooth that is external to the gum.
Cushing’s triad Hypertension (with a widening pulse pressure), bradycardia, and irregular respirations; classic trio of findings associated with increased ICP.
cusps Points at the top of a tooth.
decerebrate (extensor) posturing Abnormal posture characterized by extension of the arms and legs; indicates pressure on the brain stem.
dentin The principal mass of the tooth, which is made up of a material that is much more dense and stronger than bone.
depressed skull fractures Result from high-energy direct trauma to a small surface area of the head with a blunt object (such as a baseball bat to the head); commonly result in bony fragments being driven into the brain, causing injury.
diencephalon The part of the brain between the brain stem and the cerebrum that includes the thalamus, subthalamus, and hypothalamus.
diffuse axonal injury (DAI) Diffuse brain injury that is caused by stretching, shearing, or tearing of nerve fibers with subsequent axonal damage.
diffuse brain injury Any injury that affects the entire brain.
diplopia Double vision.
dura mater The outermost layer of the three meninges that enclose the brain and spinal cord; it is the toughest meningeal layer.
dysconjugate gaze Paralysis of gaze or lack of coordination between the movements of the two eyes.
dysphagia Difficulty swallowing.
edpidural hematoma An accumulation of blood between the skull and dura.
epiglottis Nosebleed.
external auditory canal The area in which sound waves are received from the auricle (pinna) before they travel to the eardrum; also called the ear canal.
external ear One of three anatomic parts of the ear; it contains the pinna, the ear canal, and the external portion of the tympanic membrane.
facial nerve The seventh cranial nerve; supplies motor activity to all muscles of facial expression, the sense of taste, and anterior two thirds of the tongue and cutaneous sensation to the external ear, tongue, and palate.
facial brain injury A specific, grossly observable brain injury.
fornixes The soft spots in the skull of a newborn and infant where the sutures of the skull have not yet grown together.
foramen magnum The large opening at the base of the skull through which the spinal cord exits the brain.
foramina Small natural openings, perforations, or orifices, such as in the bones of the cranial vault; plural of foramen.
frontal lobe The portion of the brain that is important in voluntary motor actions and personality traits.
galea aponeurotica Tough, tendinous layer of the scalp.
Glasgow Coma Scale (GCS) A widely accepted method of assessing level of consciousness that is based on three independent measurements: eye opening, verbal response, and motor response.
globe The eyeball.
glossopharyngeal nerve Ninth cranial nerve; supplies motor fibers to the pharyngeal muscle, providing taste sensation to the posterior portion of the tongue, and carrying parasympathetic fibers to the parotid gland.
hard palate The bony anterior part of the palate, which forms the roof of the mouth.
head injury A traumatic insult to the head that may result in injury to soft tissue, bony structures, or the brain.
hemoptysis Coughing up blood.
herniation Process in which tissue is forced out of its normal position, such as when the brain is forced from the cranial vault, either through the foramen magnum or over the tentorium.

hyoid bone A bone at the base of the tongue that supports the tongue and its muscles.

hyperpyrexia A very high body temperature.

hyphema Bleeding into the anterior chamber of the eye; results from direct ocular trauma.

hypoglossal nerve Twelfth cranial nerve; provides motor function to the muscles of the tongue and throat.

hypothalamus The most inferior portion of the diencephalon; responsible for control of many body functions, including heart rate, digestion, sexual development, temperature regulation, emotion, hunger, thirst, and regulation of the sleep cycle.

inner ear One of three anatomic parts of the ear; it consists of the cochlea and semicircular canals.

intracerebral hematoma Bleeding within the brain tissue (parenchyma) itself; also referred to as an intraparenchymal hematoma.

intracranial pressure (ICP) The pressure within the cranial vault; normally 0 to 15 mm Hg in adults.

iris The colored portion of the eye.

lacrimal apparatus The structures in which tears are secreted and drained from the eye.

lambdoid suture The point where the occipital bones attach to the parietal bones.

Le Fort fractures Maxillary fractures that are classified into three categories based on their anatomic location.

lens A transparent body within the globe that focuses light rays.

limbic system Structures within the cerebrum and diencephalon that influence emotions, motivation, mood, and sensations of pain and pleasure.

linear skull fractures Account for 80% of skull fractures; also referred to as nondisplaced skull fractures; commonly occur in the temporal-parietal region of the skull; not associated with deformities to the skull.

malocclusion Misalignment of the teeth.

mandible The movable lower jaw bone.

mandibular nerve A sensory and motor nerve that supplies the muscles of chewing and skin of the lower lip, chin, temporal region, and part of the external ear.

mastication The process of chewing with the teeth.

mastoid process A cone-shaped section of bone at the base of the temporal bone.

maxillary nerve A sensory nerve; supplies the skin on the posterior part of the side of the nose, lower eyelid, cheek, and upper lip.

mean arterial pressure (MAP) The average (or mean) pressure against the arterial wall during a cardiac cycle.

mediastinitis Inflammation of the mediastinum, often a result of the gastric contents leaking into the thoracic cavity after esophageal perforation.

medulla Continuous inferiorly with the spinal cord; serves as a conduction pathway for ascending and descending nerve tracts; coordinates heart rate, blood vessel diameter, breathing, swallowing, vomiting, coughing, and sneezing.

meninges A set of three tough membranes, the dura mater, arachnoid, and pia mater, that encloses the entire brain and spinal cord.

middle ear One of three anatomic parts of the ear; it consists of the inner portion of the tympanic membrane and the ossicles.

nasal cavity The chamber inside the nose that lies between the floor of the cranium and the roof of the mouth.

nasal septum The separation between the right and left nostrils.

nasolacrimal duct The passage through which tears drain from the lacrimal sacs into the nasal cavity.

neuronal soma The body of a neuron (nerve cell).

occipital condyles Articular surfaces on the occipital bone where the skull articulates with the atlas on the vertebral column.

occipital lobe The portion of the brain that is responsible for the processing of visual information.

oculomotor nerve Third cranial nerve; innervates the muscles that cause motion of the eyeballs and upper eyelid.

olfactory nerves Participates in the transmission of scent impulses.

optic nerve Either of the second cranial nerves that enter the eyeball posteriorly, through the optic foramen.

orbits Bony cavities in the frontal part of the skull that enclose and protect the eyes.

organ of Corti A structure located in the cochlea that contains hairs that are stimulated by vibrations to form nerve impulses that travel to the brain and are perceived as sound.

ossicles The three small bones in the inner ear that transmit vibrations to the cochlear duct at the oval window.

oval window An oval opening between the middle ear and the vestibule.

palatine bone An irregularly shaped bone found in the posterior part of the nasal cavity.

paranasal sinuses The sinuses, or hollowed sections of bone in the front of the head, that are lined with mucous membrane and drain into the nasal cavity.

pial lobe The portion of the brain that is the site for reception and evaluation of most sensory information, except smell, hearing, and vision.

periorbital ecchymosis Bruising under or around the orbits that is commonly seen following a basilar skull fracture; also called raccoon eyes.

peripheral vision Visualization of lateral objects while looking forward.

pia mater The innermost and thinnest of the three meninges that enclose the brain and spinal cord.

pinna The large outside portion of the ear through which sound waves enter the ear; also called the auricle.

pons Lies below the midbrain and above the medulla and contains numerous important nerve fibers, including those for sleep, respiration, and the medullary respiratory center.

posterior chamber The posterior area of the globe between the lens and the iris.

primary brain injury An injury to the brain and its associated structures that is a direct result of impact to the head.

pulp Specialized connective tissue within the pulp cavity of a tooth.

pupil The circular opening in the center of the eye through which light passes to the lens.

raccoon eyes Bruising under or around the orbits that is commonly seen following a basilar skull fracture; also called periorbital ecchymosis.
reticular activating system (RAS) Located in the upper brain stem; responsible for maintenance of consciousness, specifically one’s level of arousal.

retina A delicate 10-layered structure of nervous tissue located in the rear of the interior of the globe that receives light and generates nerve signals that are transmitted to the brain through the optic nerve.

retinal detachment Separation of the inner layers of the retina from the underlying choroid, the vascular membrane that nourishes the retina.

retrograde amnesia Loss of memory relating to events that occurred before the injury.

sagittal suture The point of the skull where the parietal bones join.

sclera The white part of the eye.

secondary brain injury The “after effects” of the primary injury; includes abnormal processes such as cerebral edema, increased intracranial pressure, cerebral ischemia and hypoxia, and infection; onset is often delayed following the primary brain injury.

skull The structure at the top of the axial skeleton that houses the brain and consists of 28 bones that comprise the auditory ossicles, the cranium, and the face.

subarachnoid hemorrhage Bleeding into the subarachnoid space, where the cerebrospinal fluid (CSF) circulates.

subarachnoid space The space located between the pia mater and the arachnoid mater.

subdural hematoma An accumulation of blood beneath the dura but outside the brain.

subthalamus The part of the diencephalon that is involved in controlling motor functions.

sympathetic eye movement The movement of both eyes in unison.

temporal lobe The portion of the brain that has an important role in hearing and memory.

temporomandibular joint (TMJ) The joint between the temporal bone and the posterior condyle that allows for movements of the mandible.

tentorium A structure that separates the cerebral hemispheres from the cerebellum and brain stem.

thalamus The part of the diencephalon that processes most sensory input and influences mood and general body movements, especially those associated with fear or rage.

tracheal transection Traumatic separation of the trachea from the larynx.

traumatic brain injury (TBI) A traumatic insult to the brain capable of producing physical, intellectual, emotional, social, and vocational changes.

trigeminal nerve Fifth cranial nerve; supplies sensation to the scalp, forehead, face, and lower jaw and innervates the muscles of mastication, the throat, and the inner ear.

trismus Clenching of the teeth owing to spasm of the jaw muscles.

tympanic membrane A thin membrane that separates the middle ear from the inner ear and sets up vibrations in the ossicles; also called the eardrum.

ventricles Specialized hollow areas in the brain.

visual cortex The area in the brain where signals from the optic nerve are converted into visual images.

vitreous humor A jellylike substance found in the posterior compartment of the eye between the lens and the retina.

zygomatic arch The bone that extends along the front of the skull below the orbit.
Section 4 Trauma

Assessment in Action

Your unit is dispatched to a residence for an assault. An on-scene police officer advises you that the scene is safe to enter. Your response time to the scene is approximately 7 minutes. When you arrive, a police officer escorts you to the patient, a man in his late 30s. According to witnesses, the patient was struck in the side of the head with a steel pipe during an altercation with his neighbor. As you approach the patient, you note that he is lying in a supine position and is not moving; there is no gross bleeding. The neighbor is in police custody.

1. After your partner manually stabilizes the patient’s cervical spine, you should:
   A. vigorously shake the patient to determine his level of consciousness.
   B. open his airway with the head tilt–chin lift maneuver or tongue jaw lift.
   C. suction his oropharynx for 30 seconds to ensure that it is clear of blood.
   D. determine his level of consciousness, and ensure that his airway is clear.

2. Your initial assessment reveals that the patient is unconscious and unresponsive. You insert an oropharyngeal airway and assess his respirations, which are slow and shallow. His radial pulses are slow and bounding. What must you do next?
   A. Perform immediate endotracheal intubation.
   B. Provide bag-mask ventilation and 100% oxygen.
   C. Apply a nonrebreathing mask, and reassess him.
   D. Start an IV line and administer atropine sulfate.

3. The patient’s BP is 170/100 mm Hg, his pulse rate is 50 beats/min and bounding, and his baseline respirations are 6 breaths/min and have now become irregular. What is the pathophysiology of this patient’s vital signs?
   A. An increase in mean arterial pressure, cerebral vasodilation, and pressure on the brain stem
   B. Cerebral vasoconstriction, shunting of blood from the brain, and complete brain stem herniation
   C. A decrease in mean arterial pressure, cerebral vasodilation, and a decrease in cerebral perfusion pressure
   D. Cerebral vasodilatation, a decrease in cerebral blood flow, and increased parasympathetic tone

4. All of the following are clinical signs of pressure on the upper brain stem, EXCEPT:
   A. Cheyne-Stokes respirations.
   B. an increase in the patient’s BP.
   C. a marked increase in heart rate.
   D. bilaterally fixed and dilated pupils.

5. Which of the following are indications for hyperventilation of a brain-injured patient?
   A. A systolic BP that exceeds 200 mm Hg
   B. Bilaterally dilated and slowly reactive pupils
   C. An absent motor response to painful stimuli
   D. Withdrawal from pain with flexor posturing

6. Your patient has been intubated and ventilations are continuing. Further assessment reveals that the patient is unresponsive to all stimuli, has unequal pupils, and shows extensor posturing. How many ventilations per minute should this patient receive?
   A. 10
   B. 20
   C. 25
   D. 30

7. Which of the following is the most appropriate IV fluid regimen for a head-injured patient with a BP of 70/50 mm Hg?
   A. An amount sufficient to maintain a systolic BP of at least 90 mm Hg
   B. 1,000 mL to 2,000 mL followed by a reassessment of the patient’s BP
   C. A crystalloid solution infusion set to run at approximately 120 mL/h
   D. Set the IV line(s) to keep the vein open because fluids will worsen cerebral edema

8. Which of the following drugs would you be least likely to use when treating a patient with a severe head injury?
   A. Lorazepam (Ativan)
   B. Lidocaine
   C. 50% dextrose
   D. Normal saline

9. Which of the following parameters does the Glasgow Coma Scale (GCS) measure?
   A. Pupil size, eye opening, verbal response
   B. Eye opening, motor response, heart rate
   C. Verbal response, pupil size, motor response
   D. Eye opening, verbal response, motor response

10. You have arrived at the hospital and have transferred patient care to the attending physician. You later learn that the patient had bleeding between the outer meningeal layer and the skull. This is called a/an:
    A. subdural hematoma.
    B. epidural hematoma.
    C. subarachnoid hemorrhage.
    D. intraparenchymal hematoma.
Points to Ponder

You are transporting a 30-year-old woman with blunt head trauma. She is conscious but persistently confused. You have applied 100% oxygen via nonrebreathing mask, started an IV line of normal saline and set the flow rate to keep vein open, and applied the cardiac monitor. Because of the mechanism of injury, full spinal motion restriction precautions have been applied. The patient’s BP is 138/88 mm Hg, pulse rate is 100 beats/min, and respirations are 20 breaths/min and regular. As you are conversing with the patient, you note that her level of consciousness is progressively decreasing. You reassess her airway, which is still patent, but her respirations are now slow. The patient’s pupils have increased in size but are still equal and reactive to light. She responds to pain by pushing your hand away. Noting these changes, you insert an airway adjunct and begin hyperventilating by bag-mask ventilation at a rate of 24 breaths/min and continue to do so until you arrive at the hospital 20 minutes later. After delivering the patient to the hospital and returning to service, you learn that the patient experienced an anoxic brain injury.

Why did this occur? Could you have done something to prevent it?

Issues: Recognizing Clinical Signs of the Different Levels of Intracranial Pressure, Knowing the Appropriate Ventilation Rates for Head-Injured Patients, Understanding the Importance of Maintaining Cerebral Perfusion Pressure.