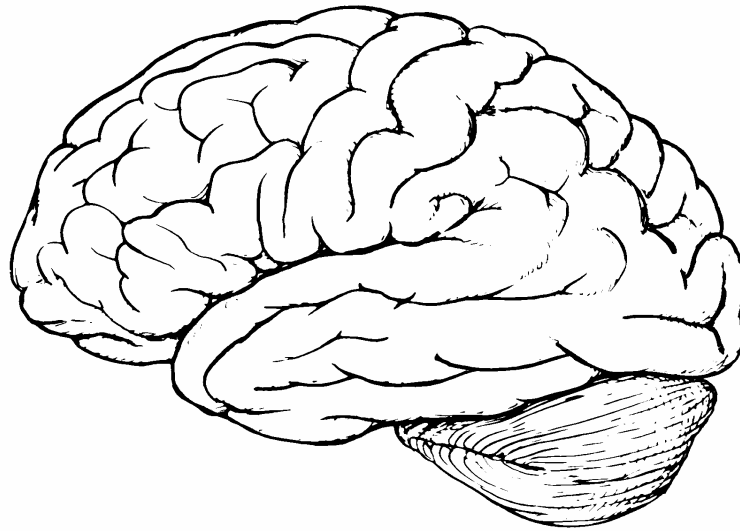# Overview of Adult Traumatic Brain Injuries

Self-Learning Packet

2004

This self-learning packet is approved for 4 contact hours for the following professionals:
1. **Registered Nurses**
2. **Licensed Practical Nurses**

Table of Contents

Purpose

This specialized self-learning packet is to educate healthcare providers who care for adult patients with head injuries as a result from a traumatic event. This program meets the continuing education requirements for the state-sponsored Level I Trauma Center.

Objectives

After completing this packet, the learner will be able to:

1.   Review the normal anatomy and physiology of the brain.

2.   Calculate and interpret cerebral perfusion pressure.

3.   Identify the mechanisms of injury associated with head injuries.

4.   Prioritize emergent treatment for the head injured patient.

5.   Differentiate between primary and secondary brain injuries and the treatment.

6.   Describe the types of facial and skull fractures associated with head injuries.

7.   Describe the factors that interfere with autoregulation that can lead to secondary brain injuries.

8.   Identify the signs and symptoms of various types of head injuries.

9.   Identify the signs and symptoms of elevated intracranial pressure.

10.   List the signs and symptoms related to Cushing's triad.

11.   Discuss the different types of herniation syndromes.

12.   Review key components of the assessment of a brain injured patient.

13.   Apply the Glasgow Coma Scale to a patient with a head injury.

14.   Describe the pathological and cellular changes which occur in the patient with a secondary head injury.

15.   Describe the nursing management of patients with brain injury.

16.   Discuss the rehabilitation care for patients with brain injury.

Instructions

In order to receive 4.0 contact hours, you must:
- complete the posttest at the end of this packet

- submit the posttest to Education & Development with your payment

- achieve an 84% on the posttest

Be sure to complete all the information at the top of the answer sheet. You will be notified if you do not pass, and you will be asked to retake the posttest.

Introduction

Trauma is a leading cause of death in the adult population. Approximately one half of all adults who have died from a trauma injury sustained a head injury. Of those 50%, approximately half are admitted to the hospital with a diagnosis of a head injury. Head injuries are associated with approximately 50% of all motor vehicle crashes. Risk-taking behaviors can also lead to accidents that cause head injuries and include: alcohol intake, mind-altering drugs, improper use or non-use of safety equipment in motor vehicles, motorcycles (helmets), bicycles (helmets), and participation in contact sports. If a detailed history is unavailable and the patient is unconscious, then the loss of consciousness may have preceded and/or caused the injury.

Anatomy/Physiology

The components of the head and brain affected by head injuries include the scalp, skull, facial bones, brain tissue, meninges, blood brain barrier, intravascular component (blood in blood vessels), and cerebral spinal fluid (CSF).

Scalp

Injuries to the scalp are usually associated with an underlying skull or brain injury, although a scalp injury can occur separately. The scalp is very vascular and prone to profuse hemorrhage due to the veins and arteries inability to vasoconstrict adequately. Bleeding can occur between layers of the scalp (subcutaneous or subgaleal layers). These hemorrhages by themselves require no intervention. However, lacerations and avulsions require a thorough clinical examination to determine the extent of the injury. The scalp wound must be palpated and explored to determine if a skull fracture is present; although the wound may not be in alignment with the fracture as the scalp is movable. Attention must be taken to clean the scalp wound prior to the repair in order to prevent an infection. If an infection of the scalp occurs, it may penetrate the periostium of the skull bone and then enter into the brain tissue.

Skull

The skull protects the brain and consists of 2 regions: the cranial bones and facial bones. The periosteum is a dense white fibrous membrane that covers the bone. It is very vascular and sends branches into the bone to provide nutrition to the bone cells, which is imperative for growth and repair. The foramen magnum is an opening of the occipital bone at the base of the skull of which the spinal cord passes.

Cranial Bones:

Base of Skull   Side View
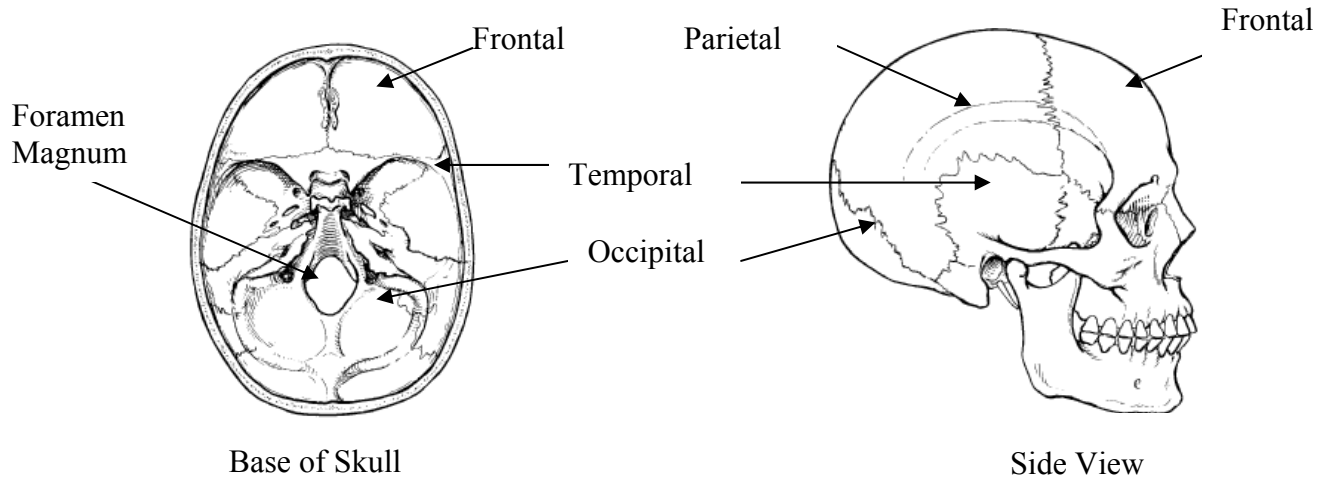
Facial Bones:

The facial bones include pairs of maxillary, zygomatic (malar), nasal, lacrimal, palatine (palate), and inferior nasal conchae (turbinates) bones; the mandible; and vomer. The mandible is considered the strongest bone in the body.
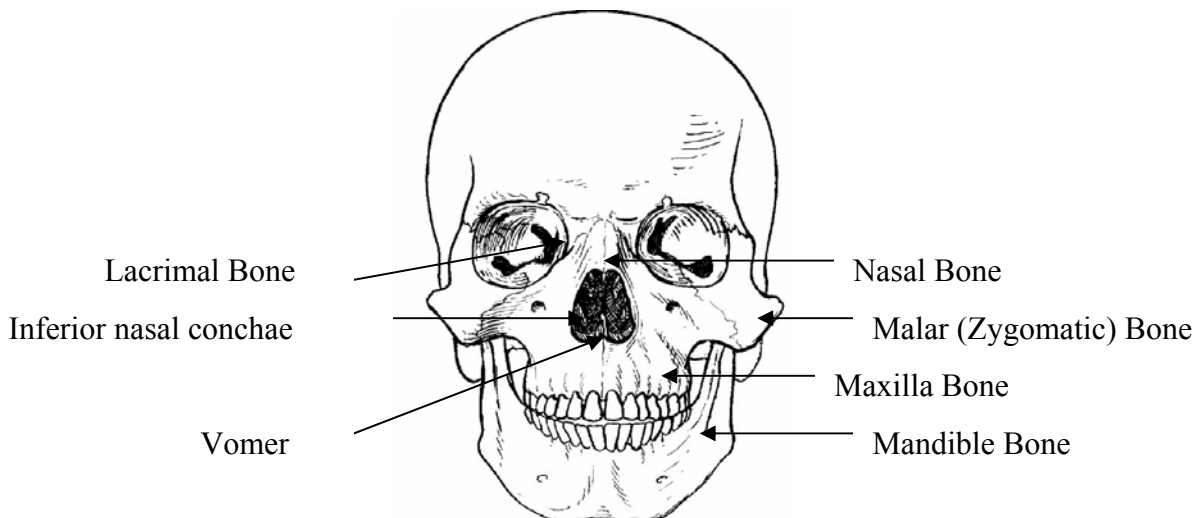
Cranial Vault

The cranial vault is used to describe where the cerebrum, cerebellum, and brainstem are housed. The three components of the cranial vault include brain tissue (80%), CSF (10%), and blood within blood vessels (10%). The Monroe-Kellie Doctrine states: *When the volume of any of the three cranial components increases, the volume of one or both of the others must decrease or the intracranial pressure will rise*. Any alteration in the volume may lead to an increase in the intracranial pressure, unless the brain can compensate. Intracranial volume can be increased by an intracranial mass, blood, CSF, or cerebral edema (cytotoxic or vasogenic).
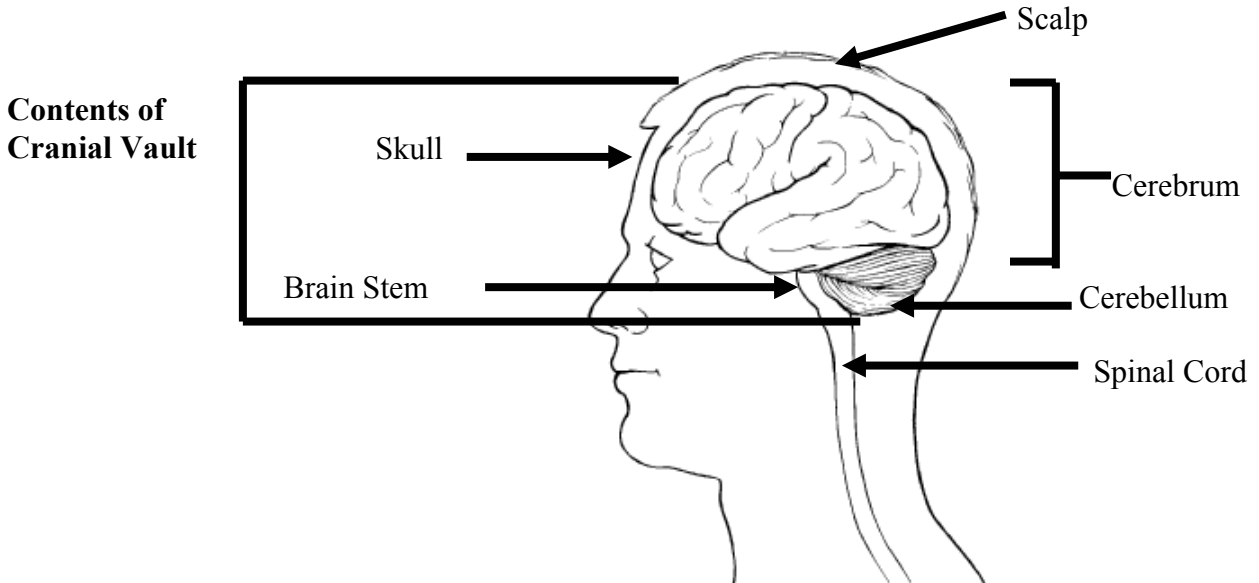
Meningeal Layers

The three meninges that cover the brain and spinal cord are the dura mater, arachnoid mater, and pia mater. The dura mater is a two-layered membrane that lines the skull and is very difficult to penetrate. The space above the dura mater is called "epidural" and below the dura mater is called "subdural." The next two layers, the arachnoid and the pia mater are called leptomeninges. They are extremely thin and difficult to visualize unless there is a space between them. This area is referred to as the subarachnoid space and it is where cerebrospinal fluid (CSF) flows around the entire brain and spinal cord. The pia mater is a mesh-like substance that covers the entire surface of the brain tissue going into the sulci and gyri (folds of the brain).
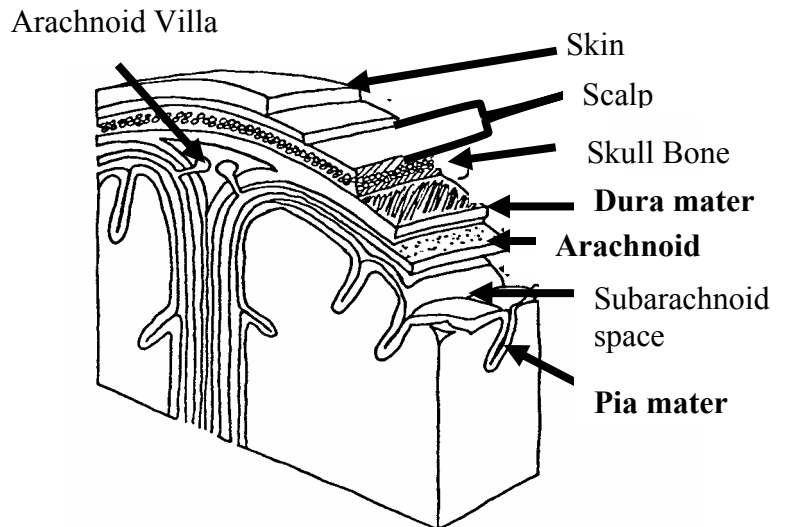
Brain Tissue

Brain tissue is composed of neurons and glial cells. Brain tissue occupies 80% of the cranial vault. Neurons are the functional units that transmit sensory and motor impulses to and from the peripheral nervous system (PNS) and the central nervous system (CNS). The glial cells, astrocytes, ependymal cells, microglia, and oligodendrocytes, under normal function, are considered neuroprotective. The glial cells are the support structure to the neurons. Astrocytes are responsible for supplying nutrients to the neurons and other glial cells and to maintain the potassium ion homeostasis for neurons. Microglia are considered the waste or debri removal system of the brain. The ependymal cells produce the CSF that carries nutrients throughout the CNS and cushion the brain and spinal cord. The oligodendrocytes are responsible for maintaining the myelin sheath after an injury.

The following figure depicts the major structures of the brain that are important.
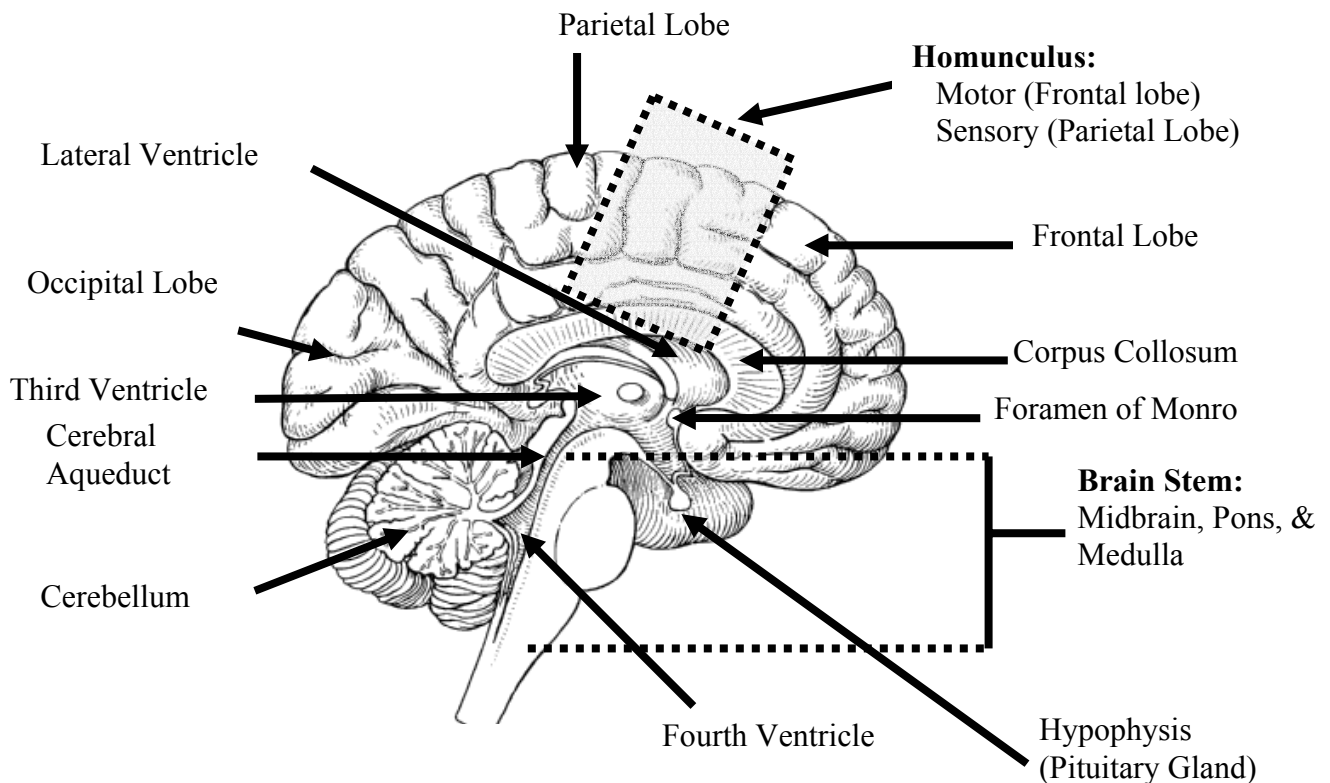
Normal Functions	
Cerebrum	Performs motor and sensory functions and a variety of mental activities
Cerebellum	Balance, muscle tone, posture and coordination
Brainstem	Motor control, reticular activating system (wakefulness), regulatory centers for heart rate, pulse, blood pressure and respiration

Each area of the CNS interacts with the others. The right hemisphere controls hand dominance on the left side, artistic functions, music, art awareness, spatial orientation, creativity and insight. The left hemisphere controls hand dominance on the right side, number skills, spoken language, written language, abstract reasoning and scientific functions. The corpus collasum connects the right and left hemispheres of the cerebrum, coordinating the function of the two halves. The cerebrum contains four lobes: frontal, parietal, temporal, and occipital.

Lobe	Function
Frontal Lobe	Judgment, reasoning, attention, short term memory, motor function (Homunculus), motor speech (Broca's area) and personality
Parietal Lobe	Sensation (Homunculus), speech organization, hand skills, grammar, perception, and proprioception
Temporal Lobe	Hearing, emotion, smell, taste, understanding speech (Wernicke's area), recall of long-term memory
Occipital Lobe	Vision, sensation

Tentorium

The tentorial notch is a triangular opening of the dura that allows the brainstem, blood vessels and nerves to pass through an oval opening. The cerebrum is located above the tentorial notch and is referred to as supratentorial. This includes the frontal, temporal, parietal and occipital lobes. Also contained in this area are the corpus collosum, 2-lateral ventricles, 3rd ventricle, cranial nerve I and cranial nerve II. The area below the tentorial notch is referred to as infratentorial, which includes the cerebellum and brainstem.

Supratentorial

Infratentorial
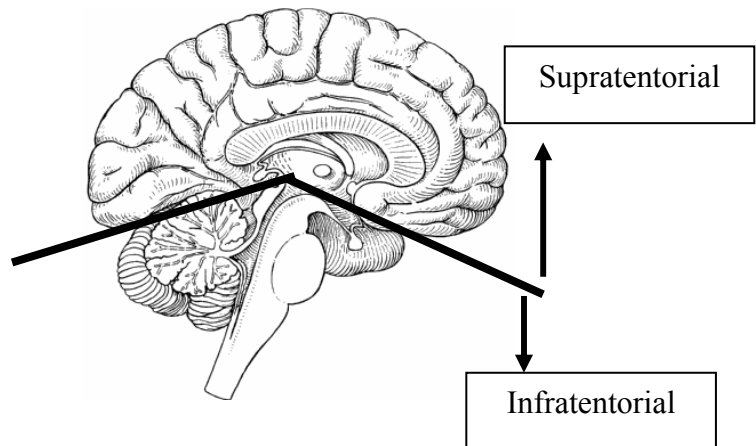
Intravascular Component

The brain must maintain a constant flow of blood in order for brain activity to occur. The arterial blood flow to the brain consists of approximately 20% of the cardiac output. Normal cerebral blood flow is 750 ml/min. The brain autoregulates blood flow over a wide range of blood pressure by vasodilation or vasoconstriction of the arteries.

Two pairs of major arteries that supply the brain are the right and left carotid and right and left vertebral arteries. The carotid arteries provide circulation to the anterior portion of the brain (frontal, temporal, parietal and occipital lobes). This accounts for approximately 80% of the blood flow to the brain. The vertebral arteries join to form the basilar artery and comprise the posterior circulation of the brain (cerebellum, brainstem, and base of occipital and temporal lobes). This accounts for approximately 20% of the blood flow to the brain. The anterior and posterior circulation function separately; however, they connect together by communicating arteries to form the Circle of Willis. In response to decreased arterial flow, the Circle of Willis can act as a protective mechanism by shunting blood from one side to the other or from the anterior to posterior portions of the brain. This compensatory mechanism is one of the reasons that there is a delay in the deteriorating neurological signs and symptoms exhibited by patients.

Arteries That Supply the Brain
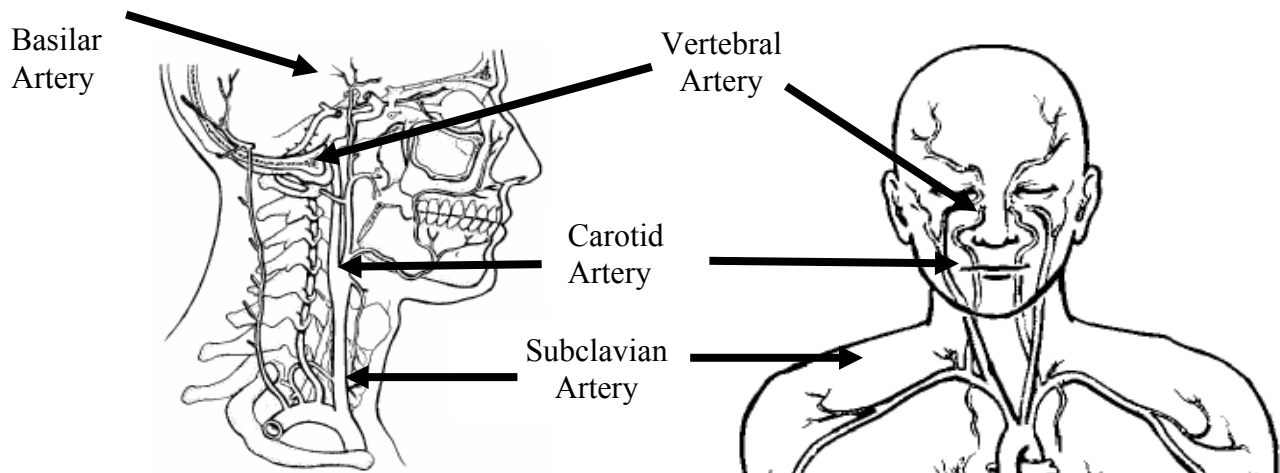
Cerebral Circulation/Artery Distribution

Anterior Circulation	
Anterior Cerebral Artery (ACA)	Supplies most medial portions of frontal lobe and superior medial parietal lobes
Anterior Communicating Artery (AcomA)	Connects the anterior cerebral arteries at their closest juncture
Internal Carotid Artery (ICA)	Ascends through the base of the skull to give rise to the anterior and middle cerebral arteries, and connects with the posterior half of the circle of Willis via the posterior communicating artery
Middle Cerebral Artery (MCA)	Trifurcates off the ICA and supplies the lateral aspects of the temporal, frontal and parietal lobes
Posterior Circulation	
Posterior Communicating Artery (PcomA)	Connects to the anterior circle of Willis with the posterior cerebral artery of vertebral-basilar circulation posteriorly
Posterior Cerebral Artery (PCA)	Supplies the occipital lobe and the inferior portion of the temporal lobe. A branch supplies the choroid plexus.
Basilar Artery (BA)	Formed by the junction of the two vertebral arteries, it terminates as a bifurcation into the posterior and cerebral arteries supplying the brainstem
Vertebral Artery (VA)	The vertebrals emerge from the posterior base of the skull (Foramen Magnum) and merge to form the basilar artery supplying the brainstem

Anterior Communicating Artery

Anterior Cerebral Artery

Middle Cerebral Artery

Posterior Communicating Artery

Basilar Artery

Vertebral Arteries
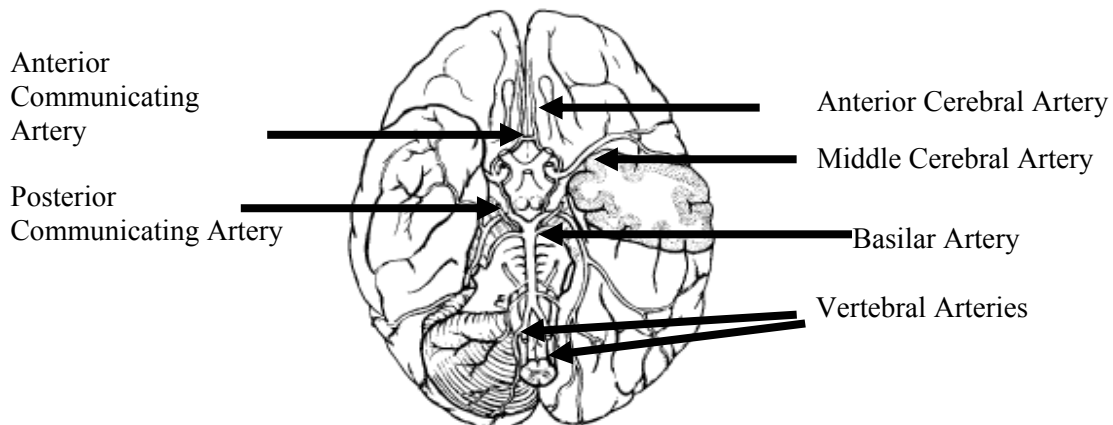
Blood-Brain Barrier

The blood-brain barrier is the area where capillaries meet and are surrounded by astrocytes. Molecules enter into these brain cells by three processes: active transport, endocytosis, and exocytosis. The barrier is very permeable to water, carbon dioxide, oxygen, glucose, and lipid soluble substances. An intact blood-brain barrier restricts the movement of larger, potentially harmful substances from the bloodstream. During ischemic or infectious states, the membrane breaks down, allowing other substances to pass into the brain.

Venous Drainage System

The cerebral veins drain into large venous sinuses and then into the right and left internal jugular veins. The venous sinuses are found within the folds of the dura mater. The veins and sinuses of the brain do not have valves so the blood flows freely by gravity. The face and scalp veins also can flow into the brain venous sinuses; therefore, infection can easily be spread into the dural venous sinuses and then enter into the brain. Patient position can prevent or promote venous drainage from the brain. Head turning and tilting may kink the jugular vein and decrease or stop venous flow from the brain, which will then increase the pressure inside the cranial vault. To promote venous drainage, the head should be maintained in a neutral position and the head of the bed elevated up to 30 degrees.

Straight Sinus · Superior Sagittal Sinus · Transverse Sinus · Jugular Vein
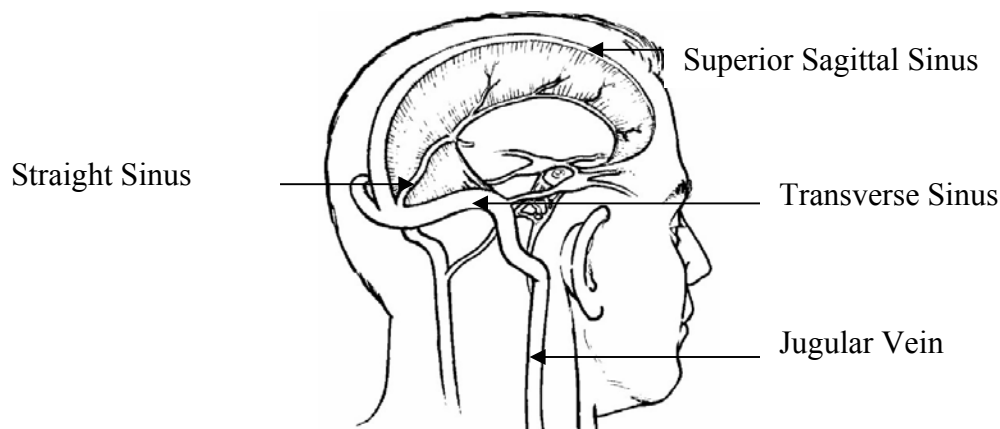
Cerebrospinal Fluid (CSF)

Cerebrospinal fluid bathes the entire brain and spinal cord. Approximately 250 –500 cc's are produced every 24 hours in the lateral ventricles by ependymal cells on the choroid plexus. The purpose of CSF is to provide nutrients, remove waste products from cellular metabolism, and act as a shock absorber. The amount of CSF in the ventricular system at one time is approximately 125 cc's. The process of CSF production and absorption must be maintained to prevent a change of the intracranial components. CSF is absorbed from the subarachnoid space by the arachnoid villi (tiny projections) into the venous system. When the CSF pressure is greater than the venous pressure, the arachnoid villi drain CSF into the venous system acting as a one-way valve. Patient position can prevent this gravitational flow of CSF. Fluctuations in pressure commonly occur due to a change in the cardiac and respiratory cycle.

Cerebral Perfusion Pressure (CPP)

Cerebral perfusion pressure is the driving force that maintains the cerebral blood flow. Currently, it is an indirect measurement and must be calculated.

In order to determine the CPP, attain the following values:

- MAP = Mean Arterial Pressure (obtained from non-invasive or invasive monitors)

- ICP = Intracranial Pressure (obtained from the closed ICP monitoring system)

$$MAP = \frac{(2 \times DBP) + SBP}{3}$$

MAP is calculated by multiplying the diastolic blood pressure (DBP) by 2, adding the systolic blood pressure (SBP), and then dividing by 3.

To calculate the CPP, subtract the ICP from the MAP (CPP = MAP – ICP). A normal CPP is between 70 mm Hg and 90 mm Hg. Hypoperfusion results when the CPP is less than 60

$$CPP = MAP - ICP$$

mm Hg. An acutely injured brain has a higher metabolic rate and therefore requires a higher cerebral perfusion pressure. The CPP should be maintained at a minimum of 70 mm Hg and up to 90 mm Hg. When the ICP is elevated, MAP should be maintained at $\geq$ 90 mmHg with the use of fluid and/or vasopressors. To effectively manage the patient with neurological compromises, a PA catheter should be inserted to monitor the MAP. A complete discussion of ICP monitoring is beyond the scope of this packet.

Cerebral Blood Flow

Cerebral blood flow (CBF) is affected by cerebral perfusion pressure and cerebrovascular resistance (CVR). CVR is the pressure across the cerebrovascular bed from the arteries to the jugular veins. CVR and CBF cannot be measured directly. The current diagnostic test available for indirect monitoring of CBF is the transcutaneous doppler. It measures the velocities of the arterial blood flow. An increase in cerebrovascular resistance (vasoconstriction due to decreased $PaCO_2$) will increase the pulsatility of the blood flow and decrease velocity. This results in a decrease in the CBF. A decrease in cerebrovascular resistance (vasodilation due to increased $PaCO_2$) will decrease the pulsatility of the blood flow and increase the velocity. This results in an increase in cerebral blood flow. These changes will be indicated on a waveform. Currently under development is an invasive parenchymal catheter that uses laser technology to measure CBF and CVR in conjunction with intracranial pressure monitoring.

CVR is influenced by the inflow pressure (systole), outflow pressure (venous pressure), cross-sectional diameter of cerebral blood vessels, and ICP. CVR is similar to systemic vascular resistance; however, due to the lack of valves in the venous system of the brain, cerebral venous pressure also influences the CVR. CVR is the amount of resistance created by the cerebral vessels and it is controlled by the autoregulatory mechanisms of the brain. Specifically, vasoconstriction will increase CVR, and vasodilation will decrease CVR.

Cerebral blood flow is calculated by subtracting the ICP from the mean arterial pressure (MAP) and dividing by the cerebrovascular resistance (CVR) or by dividing cerebral perfusion pressure (CPP) by CVR.

$$CBF = \frac{MAP - ICP}{CVR} \quad \textbf{or} \quad \frac{CPP}{CVR}$$

Cerebral Blood Flow

Average CBF	50 ml/100 Gm/min
Ischemia CBF	< 18 – 20 ml/100 Gm/min
Tissue death	< 8 – 10 ml/100 Gm/min
Hyperemia (CBF in excess of tissue demand)	> 55 – 60 ml/100 Gm/min

Cerebral blood flow can be altered by extrinsic and intrinsic factors. Extrinsic factors that affect CBF include systemic blood pressure, cardiac output, blood viscosity, and vascular tone. If the MAP falls below 70 mm Hg, cerebral blood flow will decrease. This decreased cerebral blood flow will affect cerebral autoregulation, which is the major homeostatic and protective mechanism for the brain. It operates within a MAP range of 60 – 150 mm Hg. When outside this range, there is a varying of neural activity. This results in an alteration in cerebral metabolism, which consists of synaptic activity (50%), maintenance of ionic gradient – cell membrane (25%), and biosynthesis (25%). The body responds to these demands with changes in blood flow. Aerobic metabolism is critically dependent on oxygen in order to process glucose for normal energy (ATP-adenosine triphosphate) production. The brain does not store energy. Aerobic metabolism produces 38 moles of adenosine triphosphate (ATP), and anaerobic metabolism only produces 2 moles of ATP. ATP is necessary for the cell membranes to maintain normal function (i.e. sodium-potassium pump). Therefore, without a constant source of oxygen and energy, its supply from the cerebral blood flow can be exhausted within 3 minutes.

Intrinsic factors that alter CBF include carbon dioxide content ($PaCO_2$), pH, oxygen content (PaO_2), and intracranial pressure. The vessels dilate with increases in $PaCO_2$ (hypercarbia) or low pH and with decreases in PaO_2 (hypoxia). This vasodilatation increases cerebral blood flow. Even a 1-mm Hg change in $PaCO_2$ will increase cerebral blood flow 2 – 3% (between 20 – 80 mm Hg). The vessels constrict with decreases in $PaCO_2$ or a high pH and with increases in local PaO_2. This vasoconstriction will decrease the cerebral blood flow. In addition, intrinsic factors can change the extrinsic factors by altering the metabolic mechanisms and cerebral blood flow. For example, there can be a change from aerobic to anaerobic metabolism, which increases the concentrations of other end products such as lactic acid, pyruvic acid, and carbonic acid and leads to acidosis. These end products result in a decreased pH and an increase in cerebral blood flow.

Other factors that can affect cerebral blood flow include pharmacological agents (volatile anesthetic agents and some antihypertensive agents), rapid eye movement sleep, arousal, pain, seizures, elevations in body temperature, and cerebral trauma.

Mechanism of Injury

Head injuries occur when a mechanical force strikes the head and transmits the force to the brain tissue. Forces may be blunt or penetrating. Blunt trauma is a closed head injury that results from deceleration, acceleration, combination of acceleration-deceleration, rotational or deformation forces. Deceleration forces occur when the head hits an immovable object such as the forehead hitting the windshield. This causes the skull to decelerate rapidly. The brain moves slower than the skull causing the brain tissue to collide with skull. As the brain moves over the bony prominences, it can stretch, shear or tear the tissue. Acceleration injuries can occur when an object hits the head and the skull and the brain are set in motion. Acceleration-deceleration forces occur due to the rapid changes in velocity of the brain within the cranial vault. Deformation forces occur when the velocity of the impact changes the shape of the skull and compresses the brain tissue. The brain tissue is cushioned within the cranial vault by cerebrospinal fluid, one of the protective mechanisms of the brain. Direct injury to the brain tissue can occur as contusions, lacerations, necrosis and hematomas with coup and contrecoup injuries. Coup injuries occur at the site of impact and the contrecoup injury occurs at the opposite side or at the rebound site of impact.

Coup/Contrecoup Injury
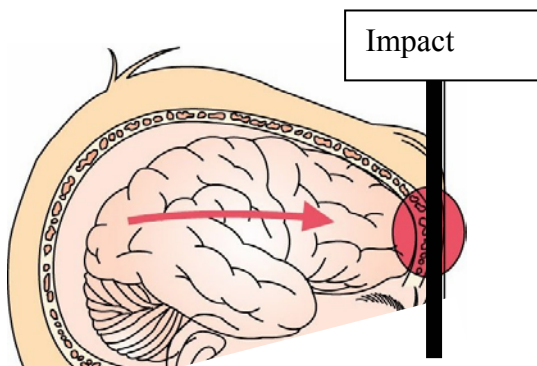
Coup Injury
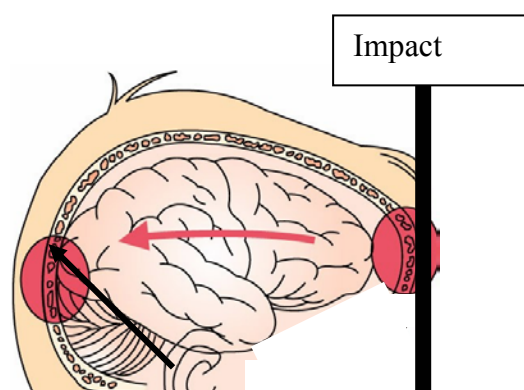
Contrecoup Injury

Bi-polar injuries may occur from front to back or side to side. Quadra-polar injuries involve all sides of the brain—front, back, and each side. The most common area of impact of a coup injury is the occipital lobe and the contrecoup injury is the frontal lobe.

Coup/Contrecoup Injury: Bipolar

Rotational forces occur from the twisting of the head usually after impact. The degree of injury depends upon the speed and direction the brain is rotated. Rotational forces affect white matter tissue of the brain. The most common areas affected include the corpus collosum and the brain stem. Diagnosis is made bas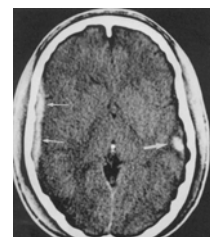ed upon clinical exam, if the patient remains in a coma greater than 24 hours, and/or the CT or MRI scan demonstrates diffuse micro-hemorrhages.

Rotational Forces

In a penetrating injury, an object breaks through the skull and enters the brain. Examples of objects that cause a penetrating injury are nail guns, guns, knifes, and other sharp objects that may be impaled into the skull. The penetrating object may cause brain tissue lacerations, contusions, and hemorrhages. The subsequent secondary injuries (cerebral edema, tissue hypoxia and necrosis) occur immediately. The severity of the injury depends on the size, shape, speed, direction, location and action as it enters the cranial cavity.

Gun shot wounds have a high mortality rate. The bullet can destroy the parenchyma along its trajectory. Shock waves occur when the bullet enters the skull and they are transmitted throughout the cranial cavity. Depending on the velocity of the bullet, it may have insufficient energy (low-velocity) to exit the cranial vault. The trajectory is unpredictable and may ricochet off the inner table opposite the entry site or off a dural structure thereby creating several tracts. High caliber bullets that enter into the cranial cavity have an increased impact of energy causing cavitation and shock wave effects to the brain tissue. These waves can create cerebral contusions on distant brain tissue, increase intracranial pressure and lead to herniation syndromes. Also, shock waves alone can be severe enough to produce cardiopulmonary arrest. A release of thromboplastin from the brain tissue can result in coagulopathy disorders. Stab wounds to the head are another type of penetrating injury. These usually occur on the left side of the brain because the majority of assailants are right-handed. Damage is caused to cerebral vasculature, parenchyma, and cranial nerves.
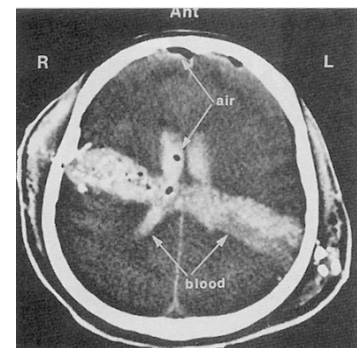
**Gun shot wound
through and through**

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Types of Injuries

The two classifications of traumatic brain injury are primary (impact damage-focal injury) and secondary injury. Primary injury occurs as an immediate result of the trauma itself. Secondary injury occurs later as a result of the primary injury. This process of secondary injury may develop over several hours and usually peaks in three to five days. Clinical management is focused on adequately resuscitating the patient and preventing or minimizing the secondary injuries that accompany the primary injury.

<u>Primary Injuries</u>

Primary injuries are a result of acceleration-deceleration and rotational forces occurring at the time of impact. These cause coup (initial impact site) and contrecoup (rebound site of impact) injuries. The forces exerted on the brain tissue may result in shearing, tensile or compressive stresses. They can lead to ruptured blood vessels causing hemorrhage, hematomas, and/or contusions. Injuries include lacerations, bone fractures, contusions, hematomas and diffuse axonal injuries.

Scalp Laceration

Scalp lacerations or abrasions are the most common minor head injury. The scalp is very vascular and has a tendency to bleed profusely; therefore, treatment includes control of bleeding, exploration of the site for bone fragments and fractures, irrigation and suturing. If no other significant findings are present, hospitalization is not required.

> **CLINICAL APPLICATION:**
>
> Scalp lacerations should be inspected cautiously, because the scalp moves on the skull and a fracture may be present in the area of laceration but not necessarily right below it. Infections of the scalp may penetrate to the periostium of the skull and then enter into the brain tissue.

Skull Fractures

The skull is very hard and requires significant force to be fractured. The outer and inner layers of bone are very hard but the middle layer consists of a spongy type of bone. The fractures need to be assessed according to the type, size, location and neurological signs and symptoms that accompany it. Treatment of a skull fracture is specific to the type of fracture and patient assessment.

<u>Linear Skull Fracture</u>
Linear fractures occur frequently and require little treatment. Forces spread over a wide area cause this type of fracture. The fractured bone can lacerate the arteries beneath causing an intracranial bleed. Most linear skull fractures heal spontaneously in two to three months. A rare complication of linear fracture is a growing fracture. A growing fracture develops over several months and causes the erosion of the bone and widening of the fracture line producing a leptomeningeal cyst. Surgical treatment is cyst removal, dural repair and cranioplasty. Linear fractures that cause the separation of the cranial suture are called diastatic fractures and require additional observation for signs of extradural bleeding.

Depressed Skull Fracture

A depressed skull fracture is a more serious fracture and signifies that a great deal of force caused the injury. The force that causes a downward displacement of the skull bones can vary from a slight depression to displacement of the outer hard bone layer below the inner hard bone that presses directly on brain tissue. Depressed skull fractures are more commonly associated with open scalp wounds, but they have an intact dural membrane. Complex depressed skull fractures involve laceration of the dura membrane with bony skull fragments. Complications associated with it are hemorrhage and laceration of the brain tissue. Treatment of depressed skull fractures include surgical repair to control bleeding, irrigation and debridement, dural repair and elevation (if > 1cm) or replacement of bone fragments.
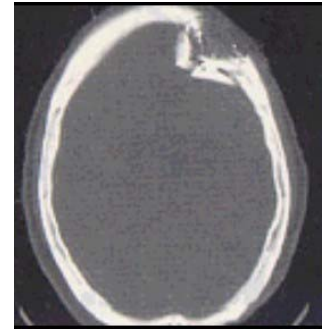
Depressed Skull Fracture

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Basilar Skull Fracture

Fractures of the cranial vault are more common than fractures of the base of the skull. A basilar skull fracture indicates a serious blow and is a break along the basilar portion of the occipital bones, the orbital plate of the frontal bones, the cribriform plate of the ethmoid, sphenoid, and petrous or squamous portions of the temporal bones. Diagnosis is difficult by x-ray and therefore is made based on clinical assessment. Clinical presentation depends on the location of the basilar fracture.

Signs and symptoms specific to each are:

Anterior Fossa: rhinorrhea (discharge from nose), raccoon eyes (periorbital ecchymosis), anosmia (loss of smell), oculomotor palsies

Middle Fossa: hemotympanum (blood in the middle ear), otorrhea, vertigo, Battle's sign (mastoid ecchymosis), unilateral hearing loss

Posterior Fossa: hypotension, tachycardia, alteration in respirations due to compression of the brainstem

Anterior Fossa

Middle Fossa
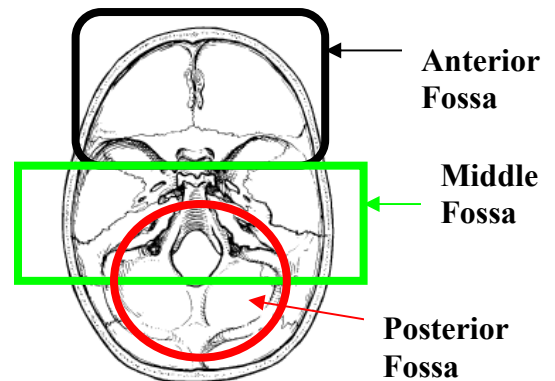
Posterior Fossa

CLINICAL APPLICATION:

An index of suspicion is high for a basilar skull fracture if your patient presents with raccoon eyes (orbital ecchymosis—single or double) indicating an anterior fossa fracture or Battle's signs (ecchymosis behind the ear) indicating a middle or posterior fossa fracture. Orbital ecchymosis usually occurs immediately. Battle's signs appear within the first 24–48 hours.

Raccoon eyes: Periorbital ecchymosis   Battle's sign: Mastoid ecchymosis

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For the most part, basilar skull fractures are uncomplicated and require observation for 24 to 48 hours. However, one potential complication that can occur is a cerebral spinal fluid leak (CSF). The patient must be assessed for CSF leaks frequently, at the time of admission, and up to several days after the injury especially when the patient begins to become more mobile (change in position, out of bed to chair, out of bed ambulating). The majority of CSF leaks resolve spontaneously without intervention.

To determine if a CSF leak is present in a conscious oriented patient, the nurse may ask the patient if he/she has a salty or sweet taste in their mouth or a post-nasal drip. Other signs include: coughing or clearing of throat, visible drainage from ear or nose. Drainage may be placed on filter paper to show evidence of a halo ring suggestive of a CSF leak. Fluid can be sent to the lab to determine the glucose content. A nasal pad placed on the upper lip or cotton ball placed on the ear lobe may be used to track the amount of leakage. The flow of CSF should never be blocked. Blockage of CSF could lead to an increase in intracranial pressure and provide a media for infection. A rare complication is meningitis (an infection of the meninges) which may occur due to CSF leakage from a tear in the meninges. The physician must be notified of the CSF leak. Patients may or may not be treated prophylaxically with antibiotics. Certain procedures can create a vacuum of pressure, which may lead to the introduction of bacteria or viruses into the brain. Types of procedures to avoid include educating the patient not to drink with a straw, drinking hot liquids, blowing of the nose, or using the incentive spirometer. Medical procedures such as insertion of a nasogastric tube via the nares should also be avoided, the mouth may be indicated as the better route. The head of the bed should be elevated as appropriate for CSF drainage.

Facial Fractures

Motor vehicle accidents are the most common cause for facial fractures. Other causes are due to assault, such as domestic violence, and sports injuries. Common locations of facial fractures are referred to as Le Forte I, II or III. Le Forte fractures usually occur to an unrestrained driver who is thrown against the dashboard or windshield. Because of the force that occurs to the head at the time of injury, a thorough assessment must also include spinal cord, skull, and neurological status. Patients with facial injuries, especially in those who are unconscious, are often at risk for an inability to maintain their airway.

The first priority of care is to clear the airway of debris (blood, teeth or bone fragments), monitor the airway for edema (soft palate tissue, or tongue), assess breathing, and initiate an alternative airway if indicated. Elevating the head of the bed, if no contraindications exist, can protect the patient's airway from occluding with secretions. Suction must be available at the bedside. Then bleeding and circulatory status must be assessed. Those with Le Forte II & III fractures are at a higher risk for bleeding because the internal maxillary artery may tear and bleed into the ethmoid or maxillary sinuses. Nasal packing with petroleum gauze or a balloon (30-mL) tamponade may be necessary for 24 – 48 hours. If the packing remains in place for more than 48 hours, necrosis of the nasal mucosal membranes or infection may occur. Fluid replacement and blood replacement is administered as indicated by the patient's response, laboratory reports, and the physician's orders. It is imperative that the nurse recognizes signs and symptoms of neurological dysfunction and immediately reports the changes to the physician. A fracture with an associated CSF leak, may develop a pathway for oral bacteria flora to enter the cranial cavity. Prophylactic antibiotics will be indicated and ordered by the physician. Assess cranial nerve function (CN V-trigeminal and CN VII-facial nerves) for motor and sensory dysfunction. Monitor for excessive salivation as it is a sign that a tear may have occurred in the parotid duct gland. The patient's level of comfort must

also be assessed. Frequent mouth care (with a toothbrush) and inspection of the oral cavity should be performed and documented every shift.

CLINICAL APPLICATION:

Warm normal saline mouth rinses should be performed every 2 hours for the initial 24 hours then every 4 hours and PRN (after liquid/solid nutrition). Irrigation helps decrease swelling, odor (old blood) and increases comfort to the patient. If dental wires are present consider using an oral irrigation device with mouthwash or a salt solution.


Treatment of facial fractures is usually surgical with plating of the bones, which most often requires the jaw to be wired shut. A liquid diet high in protein may be supplemented either through a feeding tube or orally if the patient is awake and able to protect his airway. To measure the patient's ability to swallow effectively, the physician may order a swallow study.

CLINICAL APPLICATION:

If a patient's jaw is wired shut, wire cutters must be available at the bedside at all times. If the wires need to be cut, the vertical attachment wires or rubber bands are cut, NOT the horizontal wires or rubber bands. An example of an indication requiring the wires to be cut is when the patient vomits and occludes the airway because the emesis can not pass through the wires or rubber bands.

Le Forte I Fracture
This fracture is the most common type and occurs along the maxilla bone. The patient presents with gross malocclusion, intra-oral ecchymosis and possibly epistaxis.

Fracture Line
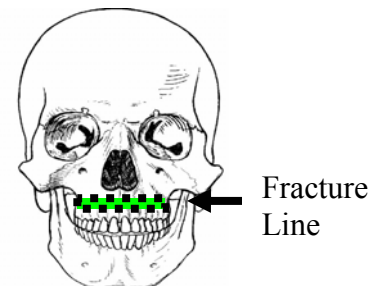
Le Forte II Fracture – Mid-face separation
This fracture occurs between the malar bone and the maxilla bone and across the nasal bone from one side to the other. It also involves the orbit and ethmoid bones. It is considered an extension of a Le Forte I fracture. The patient presents with a dishpan face, wrinkled bridge of the nose, severe epistaxis and edema along the fracture lines. There may or may not be a CSF leak.

Fracture Line
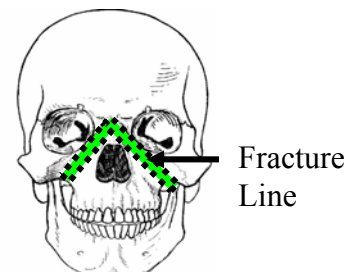
Le Forte III Fracture – Craniofacial disruption
This fracture involves the malar and the nasal bone. The patient presents with malocclusion, facial edema, free-floating maxilla, a CSF leak, and severe epistaxis. The airway can be severely compromised in these patients and an alternative airway (tracheostomy) is highly recommended.

Fracture Line
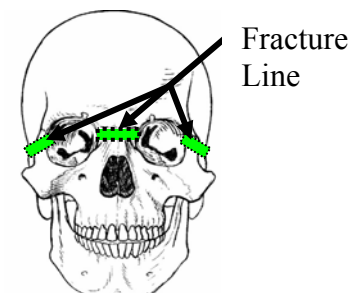
Concussion (Mild traumatic brain injury)

A concussion is the alteration of consciousness following a non-penetrating traumatic injury to the brain. There are no gross or microscopic parenchymal abnormalities. Therefore, CT scans indicate little to no abnormalities. Presentation includes confusion, disorientation, headache, dizziness, fatigue, insomnia, and a period of retrograde amnesia. Signs and symptoms usually resolve within 3 months, but may last up to a year following the injury. If there is a brief loss of consciousness, it is usually due to a transient disturbance of neuronal function. With mild traumatic brain injury, excitatory neurotransmitters are released and the brain enters a stage of hypermetabolism. The duration of this stage lasts 7–10 days from the initial injury.   If a second insult to the brain, called Second Impact Syndrome (SIS), occurs during this period (7-10 days), subsequent sequelae produces cerebral edema that is refractory to all treatment efforts and ultimately could lead to death.

> **Hallmark sign of a concussion is amnesia**

Cerebral Contusions

A cerebral contusion (bruising of the brain) is an area of bleeding and edema within the brain tissue. It begins as a primary injury then causes swelling, bleeding and increased intracranial pressure producing the secondary injury. Contusions may be caused by blunt trauma (acceleration/deceleration injuries) or penetrating trauma (knives, bullets, foreign objects or bone fragments). The contusion may occur at the site of the impact, a coup injury, or on the opposite side, contrecoup injury. The most common sites are the frontal and temporal lobes.

The clinical signs and symptoms of a cerebral contusion vary depending on the size of the contusion, degree of swelling and the location in the brain. Signs and symptoms may include a change in the level of consciousness, seizures, disorientation, headache, vomiting, and signs of increased intracranial pressure, which may lead to deterioration in neurological status. Definitive diagnosis is made by a CT/MRI scan, which shows small amounts of diffuse bleeding with edema. A follow-up CT scan (after a 24-hour period) will show an increase in bleeding and/or localized cerebral edema around the area of bleeding. Treatment may include supportive therapy, hyperventilation (if intubated, maintaining a $PaCO_2$ 30 – 35 mm Hg), osmotic diuretics (Mannitol), use of barbiturates (pentobarbital, or thiopenthol), managing intracranial pressure (ICP monitoring) or surgery (removing the contused tissue). If medical management cannot control the intracranial pressure, decompressive surgery is the last method to be considered.

Cerebral Contusions

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	Lobe Dysfunction
Frontal lobes (Blood Supply: Lateral – MCA Middle – ACA)	Left – cognition, right voluntary motor function, expressive aphasia Right – short-term memory, alteration in emotional control, motivation, inhibition; moral ethical and social value disruption; and left voluntary motor function
Parietal lobes (Blood Supply: Lateral – MCA Middle – ACA)	Left – right sensory deficits, alteration in ability to understand written word Right – visual disturbances left sensory deficits, spatial confusion, and alteration in ability to process emotions and behavior
Temporal Lobes (Blood Supply: MCA)	Left – receptive aphasia, alteration in interpretive area (causes difficulty in learning and re-learning) Right – unprovoked and abrupt aggression, and alteration in hearing, taste and smell
Occipital Lobes (Blood Supply: PCA)	visual problems including recognition of objects, alteration in reading comprehension, and conjugate deviation of eyes/head
Cerebellar Lobes (Blood Supply: PCA and BA)	problems including equilibrium, spatial, and locomotion, and altered posture
Brainstem (Blood Supply: VA and BA)	temperature regulation (hypo or hyperthermia), altered autonomic nervous system responses, thalamic syndrome (hyperthermia, tachycardia, posturing, tachypnea), involuntary motor function, may have pupillary dilation (CN III – indication of herniation) or pin point pupils (indication of hemorrhage in the pons), altered eye movements, respiratory alterations, uncontrolled vomiting, and altered swallowing abilities (CN IX)

Subarachnoid Hemorrhage

Bleeding occurs below the arachnoid meninge due to cerebral blood vessels being stretched or torn at the time of injury. A small amount of CSF occupies this space between the arachnoid and the pia meninge. The subarachnoid hemorrhage may not always be visible on a CT scan. The patient's clinical presentation and associated brain injury may be more valuable for diagnosis. Complications from blood in the subarachnoid space include focal ischemia, localized cerebral edema, vasospasm, thrombosis of blood vessels, or a traumatic aneurysm that may develop on the stretched blood vessel. The patient should be monitored for signs and symptoms of neurological deterioration, intracranial hypertension and meningeal irritation. The signs and symptoms are reviewed later in this packet.

Treatment for subarachnoid hemorrhage that results from trauma remains controversial. In some institutions, the calcium channel blocker, nimodipine, may be used. Calcium channel blockers slightly lower the MAP thereby decreasing the cerebral blood flow but potentially can cause further brain tissue ischemia. Evidence-based medicine is ever changing medical therapy and management of subarachnoid hemorrhage.

Epidural Hematomas

An epidural hematoma is a collection of blood in the extradural space (above the dura meningeal layer). The hematoma is usually located in the temporal area and is caused by the laceration of the middle meningeal artery. The laceration of the artery results in a rapidly expanding hematoma shifting brain tissue medially and immediate surgical intervention is required. If untreated, this mass effect may result in uncal herniation leading to brain death. The patient presents with the classic period of lucidity followed by rapid neurological deterioration. Symptoms may include one or all of the following: ipsilateral (same side) pupil dilation (due to direct lateral pressure on cranial nerve III from shifting brain tissue), change in the level of consciousness, posturing, contralateral limb weakness, hemiparesis, or hemiplegia.
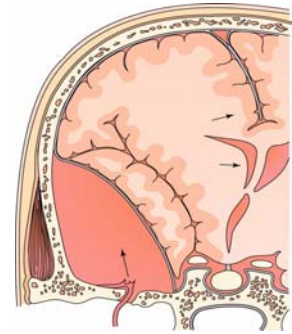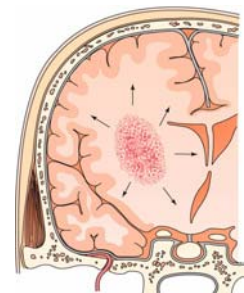

Epidural hematoma

Subdural Hematoma

A subdural hematoma is a collection of blood below the dura.
It is usually venous in origin from the bilateral bridging veins. Subdural hematomas are most frequently caused by falls, motor vehicle crashes, assaults, and violent shaking. They are classified based on the time symptoms occur: acute (24 – 48 hours), subacute (2 days to 2 weeks), or chronic (2 weeks to 3 months). The CT scan will show a crescent-shaped hematoma spreading diffusely along the inner table of the skull. Treatment includes the evacuation of the clot and control of bleeding. Medical intervention for chronic subdural hematomas usually includes keeping the patient positioned with the head of the bed flat for 24 hours to facilitate re-expansion of brain tissue with the help of gravity.
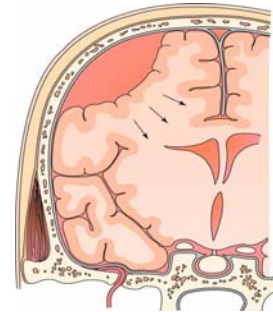
Subdural hematoma

Intracerebral Hematomas

An intracerebral hematoma (ICH) results when there is bleeding within the cerebral tissue. An amount as small as 5 cc's of blood can result in adverse neurological signs and symptoms. An ICH is most frequently caused by depressed skull fractures, penetrating injuries, or acceleration-deceleration injuries. They may also occur as a result of bleeding into necrotic brain tissue. The patient presents usually with a sudden deterioration in neurological status. Management may include both medical and surgical interventions depending upon the size and location of the bleeding.

Intracerebral hematoma

Diffuse Axonal Injury

Diffuse axonal injury (DAI) is caused by acceleration–deceleration and rotational forces during the primary head injury. This injury causes a stretching and shearing of the neurons (white matter tracts) throughout the brain, disrupting neuronal transmission. DAI is only visible on the MRI scan. However, there is a high index of suspicion when multiple small cerebral contusions appear on CT scan. Varied neurological signs and symptoms may develop. It is clinically diagnosed when the

patient presents with a prolonged coma (greater than 6 hours) and does not have signs of a mass lesion or ischemia. DAI is classified as mild, moderate or severe.

Mild DAI: Coma lasting 6-24 hours, mild to moderate memory impairment, and mild to moderate disabilities

Moderate DAI: Coma lasting > 24 hours, followed by confusion and long-lasting amnesia. Withdrawal to purposeful movements, and mild to severe memory, behavioral, cognitive, and intellectual deficits

Severe DAI: Deep prolonged coma lasting months with flexion and extension posturing. Dysautonomia can occur. Deficits are noted in cognition, memory, speech sensorimotor function and personality

Secondary Injuries

Secondary injuries occur after the initial traumatic injury and are a consequence of the primary injury. A pathological cascade occurs due to the biochemical changes in cellular structure. These changes lead to cell death and further secondary injuries such as hypoxia, hypotension, hypercarbia, hyperexcitation, cerebral edema, pathologic changes associated with increased intracranial pressure, late bleeding and expanding intracranial lesions.

Cellular Changes

The primary injury leads to an alteration in cerebral blood flow, hypoxia, and ischemia which causes a biochemical cascade and cell damage. The inflammatory process releases chemical mediators, excitatory amino acids, other neurotransmitters and cytokines that also damage the cell. The excitatory amino acids found in high numbers in the brain are glutamate and aspartate. Glutamate is the major excitatory neurotransmitter of the brain. Excessive stimulation of the glutamate receptors on the membrane leads to an alteration in the ion channels allowing sodium and calcium into the cell, further destroying the cell. Proteases and lipases are produced for membrane remodeling. This process requires a high level of energy (ATP) and since this area is already energy-deprived, it often leads to cell death.

Platelets are activated and release edema-producing factors leading to glial scarring. Additionally, neutrophils activated by the injury cause the integrity of the blood-brain barrier (between the blood vessel and the astrocyte) to collapse and allow fluid and other larger molecules into the brain. Astrocytes can become overwhelmed from the decrease in cerebral blood flow, increased acidity (lactate produced by anaerobic metabolism) and high calcium ions released by damaged neurons. Cytotoxic edema destroys the astrocyte or severely disables it leading to scarring. The injured astrocyte also secretes inhibitory chemicals that prevent regeneration of the neurons and glial cells. The microglia cells release a variety of chemicals in response to injury. The chemicals include growth factors, cytokines, complement, free fatty acids, leukotrienes, reactive oxygen species (ROS) and neurotoxins. These chemicals can be magnified when they are induced by an excess amount of calcium. These processes disrupt and destroy neuronal function.

Hypoxia/Hypercarbia

Any head-injured patient has the potential for developing hypoxia and hypercarbia. A patient with a brainstem injury will have abnormal breathing patterns because respirations are controlled by the brainstem resulting in inadequate ventilation and air exchange. A decrease in the level of consciousness will cause the muscles of the airway to relax, allowing the tongue to occlude the airway. The cough, gag and swallow reflexes are frequently diminished in head-injured patients. The loss of these protective mechanisms places the patient at an increased risk for vomiting, aspiration, and pneumonitis. Airway obstruction is managed by the chin lift and/or jaw thrust maneuver (while maintaining cervical spine immobilization), suctioning, and use of alternative airways (oral/nasal airways, intubation, and tracheostomy). The presence of hypoxia (PaO_2 <65 mm HG) significantly increases the mortality in the head-injured patient. Assisted ventilation with supplemental oxygen at 100% may be necessary to oxygenate and ventilate the patient. Blood gas results will determine if any adjustments need to be made in the therapy.

Hypotension

Hypotension (SBP< 95mm Hg) when associated with hypoxia in the head injured patient causes cerebral ischemia resulting in deterioration of the patient. The patient may present with signs and symptoms of hypotension, tachycardia (HR>100 bpm), and cool, clammy skin. Hypotension seen initially is usually not a result of the head injury, unless herniation is imminent. Other causes of hypotension may include hypovolemia (blood loss), cardiac contusion, cardiac tamponade, tension pneumothorax, and/or a possible associated spinal cord injury (quadriplegia or paraplegia). Treatment is aimed at restoring blood volume to the patient in order to prevent cerebral ischemia. The patient should have two large bore IVs infusing an isotonic solution (normal saline). It is important to monitor the patient's glucose and electrolyte levels. Hyperglycemia has been shown to be harmful to the injured brain. Hyponatremia may be associated with brain edema and seizures. Initially fluid is administered (approximately 2 liters) before vasoactive agents, such as dopamine or neosynephrine, are administered. Once the circulatory status is stable, interventions to maintain euvolemia should be implemented.

Diffuse Cerebral Swelling/Edema

Diffuse cerebral swelling is a common occurrence in the head-injured patient. It is usually caused by an increase in cerebral flood flow or hyperemia and associated with cellular changes as discussed previously. Anoxia, as seen in those with a prolonged cardiac arrest, will also cause diffuse cerebral swelling. The swelling can occur 48 to 72 hours after the initial insult and will contribute to an increase in intracranial pressure. Cerebral edema (increased water content in the brain) occurs less frequently and usually follows more severe injuries. Cerebral edema can be localized or diffuse and peaks between 24 and 48 hours after the injury occurred. Diffuse cerebral swelling contributes to a decrease in cerebral blood flow and brain tissue perfusion, increased intracranial pressure and possible herniation.

CLINICAL APPLICATION:

Vasogenic edema happens when there is a breakdown in the blood-brain barrier, allowing larger molecules of proteins and electrolytes to move into the interstitial space and results in bringing water into the extracellular space. This is seen in the white matter and peaks at 48 – 72 hours.

Cytotoxic edema happens when the cerebral tissues do not receive adequate oxygen and glucose resulting in cellular energy depletion. Failure of the sodium-potassium pump allows water and sodium to accumulate inside the cell. This is seen primarily in the grey matter.

Increased Intracranial Pressure

Since the cranial vault is a rigid container with a fixed volume, any change in the system can be detrimental. In order to maintain normal intracranial pressure, compensatory actions will include displacement of cerebrospinal fluid, compression of brain tissue and reduction of cerebral blood volume. With continued expansion of a pathological condition, the compensatory mechanisms become exhausted, which cause the intracranial pressure to rise quickly even with small increases in volume. Consequently, the patient neurologically deteriorates.

Intracranial hypertension limits cerebral flood flow and contributes to further brain injury. Cerebral blood flow is controlled by cerebral perfusion pressure that is affected by hypoxia ($PaO_2 < 60$mm Hg) and hypercarbia ($CO_2 > 45$mm Hg). This may result in vasodilitation and increased blood flow, which may increase ICP. Hypocapnia ($CO_2 < 30$mm Hg) causes vasoconstriction and decreased blood flow leading to brain ischemia. Patients with a Glasgow Coma Scale (GCS) less than 8 are most likely not able to maintain an airway; therefore, they should have an artificial airway placed and be monitored for additional ventilatory support. Clinical measuring of cerebral perfusion pressure should be obtained. Values less than 70mm Hg indicate cerebral ischemia and must be reported to the physician. Medical goals of treatment are to maintain the MAP ≥ 90mm Hg, the PaO_2 within normal range (per patient history and/or >90 mm Hg), and $PaCO_2$ 30–35 mm Hg.

Early signs of neurological deterioration include altered level of consciousness (restlessness, agitation, somnolence, obtundation, lethargy), vomiting, headache, abnormal respirations, seizures, posturing, and change in pupil size or reactivity. These signs and symptoms all represent changes in the patient's intracranial pressure. It is extremely important when any alteration in the level of consciousness or these signs are detected to immediately notify the physician because any delay may result in coma or death. Signs and symptoms of a meningeal irritation include a headache, nuchal rigidity, a positive Brudzinski's sign, and positive Kernig's sign. Brudzinki's sign is a severe neck stiffness that causes the patient's hips and knees to flex when the neck is flexed. A positive Kernig's sign is the severe stiffness of hamstrings that cause an inability to straighten the leg when the hips are flexed 90°.

Signs and Symptoms		
Early Neurological Deterioration	Meningeal Irritation	Intracranial Hypertension
• Altered level of consciousness • Vomiting • Headache • Abnormal respiratory pattern • Abnormal motor movement • Pupillary changes (shape, size, reactivity) • Seizures	• Headache • Nuchal rigidity • Brudzinski's sign • Kernig's sign	• Deterioration in level of consciousness • Ipsilateral dilated pupil (> 4mm) • Hemiparesis or hemiplegia • Abnormal posturing

Compensation for Increased ICP

The brain may try to compensate for the increase in one of the intracranial components by shunting CSF to the spinal subarachnoid space, increasing CSF absorption, decreasing CSF production, or by shunting venous blood out of the skull. Using some of these compensatory mechanisms, the brain will maintain a relatively normal ICP. When the brain has used all of these mechanisms, there will be a sharp rise in the ICP. This will lead to herniation of brain tissue downward through the foramen magnum. As this happens, blood will cease to flow to the brain and causes brain tissue hypoxia, ischemia, infarction, necrosis, and/or death.

Cushing's Triad

Cushing's Triad is the brain's last attempt to perfuse itself. These late signs of intracranial hypertension include widening pulse pressure (elevated systolic pressure with a normal diastolic pressure) and bradycardia. Treatment should be initiated **immediately** to decrease intracranial pressure and increase cerebral perfusion pressure. In an intact cranial vault, edematous brain tissue seeks an area of less pressure and a downward movement of tissue can occur towards the foramen magnum. Increased pressure on the brainstem results in dysfunction of the respiratory and cardiac centers.

CLINICAL APPLICATION:

Cushing's Triad

Signs of impending herniation include elevated systolic blood pressure, widening pulse pressure (> 40 mm Hg), and bradycardia.

Herniation results in respiratory and cardiac arrest.

Herniation

Expansion and shifting of brain tissue from one area of the brain to another decreases intracranial compliance. If untreated, it will result in herniation of the brain leading to brain death.

There are two major categories of herniation: supratentorial (above the tentorial notch) and infratentorial (below the tentorial notch). The types of supratentorial herniation are uncal, central or transtentorial, cingulate (subfalcine), and transcalvarial. The types of infratentorial herniation are upward transtentorial and downward cerebellar (tonsillar).
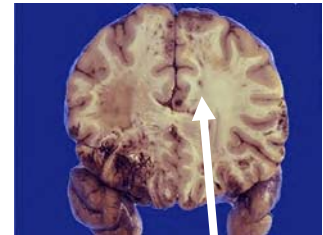
Supratentorial Herniation

Uncal herniation results when the increased ICP forces the brain (the uncus of the temporal lobe) downward into the skull and the tissue shifts medially compressing the cranial nerve III near the brainstem. The patient will usually present with ipsilateral pupil dilation (aniscoria > 1mm difference), decreased level of consciousness (restlessness, agitation), respiratory pattern changes (hyperventilation) and contralateral hemiplegia and/or posturing. The lateral displacement of the diencephalon and midbrain compresses the tentorium incisura contralateral to the expanding lesion resulting in ipsilateral hemiparesis. This phenomenon is called Kernohan's notch. The posterior cerebral artery also may be compressed on the same side as the expanding lesion. Uncal herniation is a precursor to central herniation. The goal of treatment is to prevent uncal herniation from advancing to central herniation.
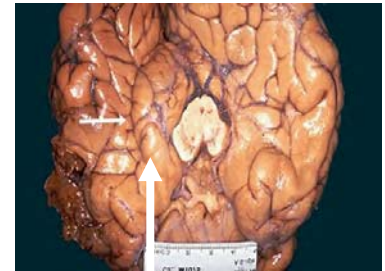
Uncal herniation

Cingulate herniation takes place when the expanding lesion of one hemisphere shifts laterally and forces the cingulated gyrus under the falx cerebri (dura mater between the two hemispheres). The patient presents with decreased level of consciousness and posturing. This usually occurs simultaneously with uncal herniation. If uncontrolled, cingulate herniation can lead to central herniation.

Cingulate herniation

Central herniation is the downward movement of the cerebrum (frontal, temporal, parietal, occipital, basal ganglia, and diencephalon) through the tentorial notch. Central herniation is usually preceded by uncal and cingulate herniation. Clinical presentation includes change in level of consciousness (lethargy, agitation, stupor leading to coma); small, reactive pupils initially (1–3 mm), then fixed and dilated; respiratory pattern changes leading to arrest (sighs, yawns, pauses, later cheyne-stokes); posturing initially then flaccidity; and diabetes insipidus.

Transcalvarial herniation is extrusion of brain tissue through the cranium due to penetrating trauma, skull fracture (most common is a basilar fracture), or craniotomy site. Clincial presentation includes severe neurological deficits and/or death.

Infratentorial Herniation

Upward transtentorial herniation is an expanding mass of the cerebellum, which protrudes upward pushing the midbrain through the tentorial notch. Cranial Nerve II compression occurs leading to

blockage of the central aqueduct which then leads to distortion of the third ventricle, obstruction of CSF flow and occlusion of arterial supply to the brainstem. Brain death will occur if untreated or uncontrolled.

Downward cerebellar herniation (tonsilar herniation) is an expanding downward lesion of the cerebellum through the foramen magnum, which compresses the brainstem. Clincial presentation includes respiratory arrest, cardiac arrest and brain death.

Tonsilar herniation

Patient Care

Assessment

The key components of the trauma assessment are as follows:

1. ABC's:
   Assess the airway with stabilization of the cervical spine, breathing, circulation, heart rate and blood pressure **before** the neurological exam.

2. Examination of the Skull:
   Assess for periorbital and postauricular ecchymosis, cerebrospinal fluid otorrhea and rhinorrhea, hemotympanum, penetrating injury or depressed fracture, and lacerations.

3. History:
   Gather information related to the mechanism of injury and care prior to hospitalization.

4. Neurological Exam:

Cerebral function	Assess the level of consciousness, mental status, awareness, arousal, cognitive function, and behavior.
Cranial Nerve and pupillary examination	This reflects brainstem function. Assess the pupils, eye movements, cough reflex, corneal reflex and gag reflex.
Motor and cerebellar function	Assess strength, movement, gait, and posture. Each extremity must be assessed separately. It is important to document the degree and type of stimulus applied to elicit the motor activity. Central stimuli include sternal rub, trapezius pinch and/or supraorbital pressure. Abnormal findings include abnormal posturing, flaccidity, and focal motor movements.
Sensory examination	Assess tactile and pain sensations.
Reflex examination	Assess superficial and deep tendon reflexes.
Glasgow Coma Scale	This is a valuable component of the neurological exam because it is nationally and internationally recognized. It is only one part of the neurological exam. See Appendix for GCS chart. Severe Head Injury: GCS # 8 or a decrease in 2 points or more after admission. Moderate Head Injury: GCS 9-12 Mild Head Injury: GCS 13-15

Diagnostic Studies

The CT scan without constrast is the gold standard for evaluation of head injuries. The CT can identify mass lesions, skull fractures, epidural hematomas, subdural hematomas, cerebral swelling and edema. Skull x-rays may be useful in identifying missile injuries and depressed skull fractures, but has essentially been replaced by the CT scan as an evaluation tool. The MRI is not effective in the initial screening of the head-injured patient, but may be useful in visualizing diffuse axonal injuries (DAI), nonhemorrhagic contusions, posterior fossa and small vascular lesions.

Management

The Brain Trauma Foundation (BTF) and the American Association of Neurological Surgeons (AANS) compiled evidence-based research and re-published in 2000 "The Guidelines for the Management of Severe Head Injury" that provides recommendations for practice. The discussion is extensive and beyond the scope of this packet.

The first priority in trauma resuscitation of the head-injured patient is the prevention of hypoxia and hypotension. All head-injured patients are treated as if they have a cervical spine injury (until clinically proven otherwise) by opening the airway with the jaw thrust maneuver. If the GCS is less than or equal to 8 or if the patient is hypoventilating, intubation may be required to secure the airway. The next step is to hyperoxygenate the patient with 100% oxygen. Hyperventilation is no longer recommended unless the patient begins to rapidly deteriorate. Rather, normocapnea or mild hypocapnea (PaCO$_2$ 30 – 35 mm Hg is used to prevent severe vasoconstriction and cerebral ischemia. Circulation should be maintained with use of normal saline, and then inotropes (Dopamine, Neosynephrine) if severe hypotension is present. In the past IV fluids were restricted to prevent brain swelling, but evidence-based medicine shows that early post injury hypotension (MAP $\leq$ 90 mm Hg) greatly increases morbidity and mortality due to secondary brain insult resulting from poor perfusion. Once the patient is hemodynamically stable, a non-crystalloid solution is infused to maintain the patient's normal volume. A solution of D$_5$W is not indicated as it can freely cross the blood brain barrier and enter into the brain tissue.

After the initial resuscitation has been successfully completed, the goal of management is to prevent and control complications of secondary head injuries especially intracranial hypertension, cerebral hypoxia and ischemia. Treatments are utilized in an attempt to control intracranial hypertension (ICP > 15 mm Hg) and to enhance cerebral perfusion (CPP > 70 mmHg).

Reduction of Cerebral Blood Flow

Reduction of cerebral blood flow includes maintaining head position, ventilation, hypothermia, and medication administration such as anticonvulsants, sedation and pain control, barbiturates, and neuromuscular drugs.

Head Position

Once the systolic blood pressure (SBP) is greater than or equal to 90 mm Hg, the head of the bed should be elevated 30 degrees and the head kept midline to enhance jugular venous return and CSF drainage. The endotracheal tube should be secured above the ears to prevent compression of the jugular veins.

Anticonvulsants (Anti-epileptic Drugs)

Anticonvulsants are used to control or prevent seizures. Seizure activity increases cerebral blood flow and intracranial pressure (ICP) which thereby increases oxygen consumption. Ativan, a drug used for sedation, also has anticonvulsant properties. Caution must be taken when giving this medication as it changes the patient's level of consciousness and may mask signs and symptoms of neurologic and respiratory deterioration. If the patient experiences seizure activity, a benzodiazepine such as Ativan, is the drug of choice to stop the seizure. It should be followed by an infusion of an anticonvulsant for prevention of further seizures. Phenytoin (Dilantin) is an

anticonvulsant frequently used, however other anticonvulsants may be indicated depending upon the location of the injury, severity of the injury and current medical therapy.

Sedation and Pain Control

Pain and agitation are still present in a patient with neurological impairment. Assessment should be performed on the patient to determine the characteristics of pain. It is important to trend the patient's neurological status to identify any changes in the level of consciousness. Agitation is frequently associated with changes in level of consciousness as evidenced by either a deterioration and/or emergence from a coma.   Pharmacologic agents can be used **only after** it has been determined that the agitation is not an indication of deterioration in the patient's condition. Sedation reduces elevated sympathetic tone and hypertension induced by movement. Oxygen saturation levels are checked prior to administering the sedation if patient is agitated. Patient safety is the first priority. Sedatives should be titrated to a Riker Sedation-Agitation Scale (SAS) score of 3 – 4 (see appendix).

> It is important to document thoroughly on the patient's flow sheet the initial level of pain the patient has prior to any medication given and the response to the medication given.

Pharmacologic therapy for sedation and pain control includes non-central nervous system depressants (non-narcotic analgesics) and central nervous system depressants (opioid-narcotic analgesics).   Aspirin, acetaminophen, and nonsteroidal and anti-inflammatory drugs are the main non-CNS depressants. In addition to their analgesic properties, these drugs have antipyretic and anti-inflammatory (except acetaminophen) effects. These non-CNS depressants are used to treat mild to moderate levels of pain.

CNS depressants are effective against moderate to severe pain; however, they have many potentially harmful side effects. Side effects may include one or all of the following: respiratory depression, orthostatic hypotension, cough suppression, nausea, vomiting, constipation and urinary retention. Codeine is a narcotic commonly administered for its short-acting analgesic property. Morphine sulfate may also be given; however, the side effects include respiratory depression, hypotension, and difficulty with neurological assessment. Morphine sulfate is easily reversible with narcan. It is important to document thoroughly on the patient's flow sheet the initial level of pain the patient has prior to any medication given and response to medication given.

Propofol is a very useful agent in controlling intracranial hypertension following brain injury. It is recommended for use after the patient has failed conventional sedation with narcotics and benzodiazepines. The patient must be intubated and on a mechanical ventilator to maintain respiratory function. A baseline 12-lead EKG and continuous cardiac monitoring are recommended during the infusion. The patient must be monitored for bradycardia and elongation of the PR or QT interval. Serum calcium levels should be maintained within normal levels and should be monitored prior to and during the infusion. The patient should also be monitored for hyperlipidemia. The infusion should be used at the lowest dose necessary to achieve the desired

Systemic Effects of Propofol

- Bradycardia
- Hypotension
- PR or QT elongation
- Increasing vasoactive medication requirements
- Arrhythmias
- Worsening metabolic acidosis
- Methemoglobinemia

clinical effect and should be discontinued as soon as possible. The infusion is titrated to the desired Riker score or ICP/CPP as indicated by physician. Typical infusion rates are 25 – 50 mcg/kg/min. Above 50 mcg/kg/min is considered anesthesia. If the infusion rate exceeds 80 mcg/kg/min, systemic effects may begin to appear.

Ventilation

The patient with severe brain injury is intubated and placed on a mechanical ventilator to control oxygenation and ventilation. PaO_2 is maintained at 90-95 mm Hg or greater. The effects of controlled hyperventilation can be noted less than 30 seconds from onset, and the peak effect is noted at approximately 8 minutes. The goal is to maintain the PCO_2 level at 30 – 35 mm Hg. Maintaining the PCO_2 at this level will vasoconstrict the cerebral blood vessels, thereby decreasing the blood flow to the brain, which decreases the ICP according to the Monroe-Kellie Doctrine. If the PCO_2 level falls below 30, it can result in the opposite effect, leading to vasodilation and increased intracranial pressure. Therefore, hyperventilation is used for acute episodes of neurological deterioration only. Continuous monitoring of the patient's end-tidal carbon dioxide ($ETCO_2$) level is necessary. Correlating the $ETCO_2$ value with the patient's arterial blood gas (ABG) value is valuable for timely adjustments in therapy.

New technology provides options for specific monitoring of brain tissue oxygenation. This is separate from ICP and temperature monitoring. The LICOX® Monitor measures the oxygen partial pressure ($PbtO_2$) of brain tissue. The LICOX® parenchymal catheter is inserted into the white matter. It is able to provide an early indication of differences between brain tissue oxygen supply and brain tissue oxygen demand, which may be an indicator for outcome prediction. If placed in the uninjured side, this will reflect global information relating to cerebral oxygenation. If placed in tissue around the injured site, this may reflect the changes in cerebral oxygenation. This placement is indicated by what information the physician seeks to monitor.

The mean normal $PbtO_2$ is >30 mm Hg, with a range of 25-50 mm Hg. If $PbtO_2$ is <15 for 30 minutes or <10 mm Hg for 10 minutes, the risk of death increases. Furthermore, ischemia is reported at ranges of <8 – 12 mm Hg, high mortality when <5 mm Hg, and neuronal death when ≤ 2 mm Hg.

This device allows for treatment to be targeted at improving oxygen delivery and to decrease its consumption. Delivery of oxygen to the brain improves oxygen content of blood by correcting anemias or increasing the FIO_2. The flow of blood can be improved through the use of fluids, vasopressors, adjusting the $PaCO_2$ and treating increased ICP. The consumption of oxygen can be decreased through sedation/paralysis, analgesia, and temperature regulation. The effectiveness of these interventions can then be quickly assessed using this type of monitoring device.

Barbiturates (Heavy Sedation)

The physician may order the patient to be placed into a medicated coma, otherwise known as a barbiturate coma. Short-acting barbiturates most commonly prescribed are pentobarbital (Nembutal) or thiopental. Barbiturates act on the central nervous system by decreasing cerebral blood flow, thereby decreasing the cerebral metabolism. Barbiturates also have a systemic effect on the cardiovascular system causing the peripheral venodilation of blood vessels and the pooling of blood in the periphery. The patient MUST be intubated, placed on mechanical ventilator, and hemodynamically monitored (including continuous arterial line pressure monitoring). It is highly recommended to have a PA catheter in place for advanced hemodynamic monitoring and continuously monitor the EEG (Electroencephalogram). Cardiovascular effects that can occur are

hypotension and myocardial depression. The nurse must anticipate a drop in blood pressure and mean arterial pressure (MAP). The use of vasoactive drugs (e.g. dopamine and/or phenylephrine hydrochloride infusion) may be indicated to keep the MAP ≥90 mm Hg.

When barbiturates are utilized, the neurological assessment is severely diminished or lost; however, pupillary assessment can still be monitored. The brain's electrical activity may also be monitored with an EEG. The nurse monitors the electrical waveforms, noting any suppression of the electrical activity. The goal is to achieve burst suppression of electrical activity as indicated by the physicians order usually between 3-6 seconds. Suppression of brain waves lasting more than 6 seconds indicates high levels of barbiturates. (See the appendix for more information on EEG monitoring.)

Therapy consists of administering a loading dose 10mg/kg infused over 30 minutes with a recommendation of 5mg/kg every hour for three doses. Then a maintenance dose titrated to 1–3 mg/kg/hr or a continuous infusion as needed to control ICP. Duration of barbiturate therapy for intracranial hypertension is up to 48 hours; however, it is the physician's decision as to the length of therapy. Barbiturate levels of 30–40 mg/dL (as measured in the serum) have been shown to control ICP's > 20 and cause burst suppression of the EEG waveform. If therapy is continued beyond 72 hours, it is recommended to monitor barbiturate levels since it is stored in the body fat. Liver and renal function should also be monitored as the drugs are metabolized and excreted through those systems. The half-life of pentobarbital is 15–48 hours and thiopental is 11-12 hours. Barbiturates must be cleared from the body before a complete neurological assessment can be performed. If cerebral death is anticipated or assumed, a cerebral blood flow study must be completed for definitive declaration of brain death.

Neuromuscular Blocking Agents (NMBA's) (Heavy Sedation/Paralytics)

In some hospital settings, neuromuscular blocking agents (NMBA's) are utilized in conjunction with barbiturate therapy; or NMBA's with analgesics (Morphine Sulfate) and amnesic (Versed) as continuous IV therapy. NMBA's act by paralyzing the muscles to decrease tissue metabolism. Continuous infusions are recommended after bolus dosing.

NMBA's utilized include:

Long-Acting Agents	
Medication	**Elimination**
Atracurium (Tracrium)	Hofmann degradation*
Cisatracurium (Nimbex)	Hofmann degradation*
Doxacurium (Nuromax)	Kidneys and Liver
Pancuronium (Pavulon)	Kidneys and Liver
Pipecuronium (Arduan)	Kidneys and Liver
Tubocurarine (Tubarine)	Kidneys and Liver
Vecuronium (Norcuron)	Kidneys and Liver

Short-Acting Agents	
Medication	**Elimination**
Mivacurium (Mivacron)	Hydrolysis by plasma esterases
Rapacuronium (Raplon)	Kidneys and Liver
Rocuronium (Zemuron)	Kidneys and Liver
Succinylcholine (Anectine)	Kidneys and Liver

* Hofmann degradation is an enzymatic process that occurs when there is a change in the pH of the medication.

If barbiturate therapy is not used with NMBA's, a continuous infusion of Morphine Sulfate and Versed is administered. The nurse must monitor the patient's Train of Four (TOF) responses using a peripheral nerve stimulator (PNS) **prior** to beginning the infusion, with changes in dosage, and as directed. (See the appendix for information on Train of Four.) In addition to monitoring the TOF, the nurse needs to maintain the patient's airway and ensure adequate ventilation. Ventilator alarms are set to detect even the slightest change in pressure. All alarms must be answered immediately and a bag-valve-mask kept at the bedside and with the patient at all times.

Nursing care for a paralyzed patient includes: turning the patient every two hours, providing passive range of motion, and using a pressure reducing device on the mattress, such as an air mattress. Since the patient is unable to blink, moisten the eyes with lubrication or artificial tears.

The appropriateness of discontinuing use of NMBA's should be assessed in conjunction with the physician. This should be done every 24 hours, comparing the current assessment to the patient's baseline condition. Evidence-based guidelines include monitoring lab values (LFT's, BUN, Cr, and Creatine clearance).

Hypothermia

The hypothalamus regulates the internal body temperature and is located in the brain above the brainstem. Head injuries, neurological disease, and/or cranial surgery can stimulate the hypothalamus causing a rise in body temperature. Mild hypothermia may have a significant beneficial effect on neurological outcome. Research has shown hypothermia (32° C) can reduce the incidence of seizure activity and cerebral metabolism, which contributes to a reduction in ICP. However, it is not routinely used due to complications that can occur such as coagulopathy and shivering, which in turn increases cerebral oxygen demand. The goal of treatment is to keep the patient normothermic.

With newer technological advances in brain monitoring, there is the capability to measure specific brain temperature utilizing the Licox® monitoring system (©Integra Neurosciences). Clincial research has shown there to be an upward of 1 - 4 degree increase in cerebral temperature over core body temperature.

Reduction in Brain Volume

Hyperosmolar Therapy

Mannitol reduces brain bulk by two mechanisms. First it reduces blood viscosity causing an osmotic gradient between healthy brain cells and the intravascular space resulting in an influx of water into the bloodstream. An immediate but transient reduction in ICP occurs after the administration of Mannitol. The second mechanism is a dehydrating effect as opposed to the osmotic effect on brain bulk, which results from prolonged use of Mannitol.

Mannitol is recommended to be given in bolus doses rather than a continuous infusion. Mannitol 0.25-0.50 gm/kg is given every four to six hours with additional doses given for intermittent increases in ICP. The serum sodium and osmolarity must be monitored when using Mannitol. Mannitol should **not** be given if the serum osmolality is >320 mOsm/L or sodium (NA+) level is ≥ 155 mEq/L. Mannitol administered for long periods of time may cause a rebound effect resulting in

movement of water back into the brain tissue, therefore it should be tapered. The overall goal is to keep the patient euvolemic with the aid of hemodynamic monitoring, CVP or PA catheter.

Surgery

A craniotomy may be necessary to evacuate a hematoma, debulk contused tissue or decompress vital structures. The risks and benefits are discussed by the neurosurgeon to the patient's family in this event.

Cerebrospinal Fluid Reduction

Cerebrospinal Fluid Drainage

A ventriculostomy may be placed to drain CSF directly and monitor ICP. The catheter is placed in one of the lateral ventricles of the brain and may be drained continuously or intermittently for increases in ICP. It is beyond the scope of this packet to discuss this in-depth.

Diuretics

The use of diuretics such as Lasix is controversial and can be used to reduce body fluids and decrease the production of CSF. Lasix may be used in conjunction with Mannitol.

Complications

Complications of patients with traumatic brain injuries depend upon the severity of injury. This includes neurological deficits, post-traumatic vasospasms, seizures, intracranial infection, pulmonary compromise (pneumonia, ARDS), coagulopathy (DIC), gastro-intestinal mucosal erosion (Cushing's ulcer), cardiac rhythm abnormalities, protein-calorie malnutrition, and complications related to immobility. The overall goal is to avoid or minimize complications from secondary injuries (hypotension, hypoxia, hypoglycemia, hypercarbia).

Rehabilitation

Recovery depends upon many factors including the area of damage to the brain, how extensive the damage was, the age of the person, and their health prior to the injury. It is shown that the faster a person progresses, the recovery period takes less time. It is important to note that recovery time does not necessarily mean full recovery; some patients will have life-long deficits. Most recovery in neurological function will take place within the first six months after the accident. However, after this time, change can continue to occur, but at a much slower pace. Rehabilitation teams (physical therapy, occupational therapy, and speech therapy) utilize the Ranchos Los Amigos Scale to determine the level of injury to direct the treatment plan (See Appendix for Scale). Levels of closed head injury are related to mild, moderate, and severe head injuries and are classified in the following manner:

    Severe   Ranchos Los Amigos Levels I - III
    Moderate   Ranchos Los Amigos Levels IV - VI
    Mild   Ranchos Los Amigos Levels VII - VIII

Severe Closed Head Injury: Ranchos Levels I-III

Time to recover: up to one year and beyond

Goals of treatment are to increase their response to external stimuli such as pain, sound, light, and verbal stimulation and to improve the appropriateness of their response to stimuli.

Treatment Strategy:
- Patients will usually respond more to family members and close friends than to hospital staff. It is important to be present during part of these interactions to document changes in the patient's level of consciousness, as well as patient tolerance to stimulation and to allow rest.
- Sensory stimulation should be done frequently (several times per day) but briefly (10 – 15 minutes).
  − Sounds such as clapping, ringing bells, radio, tapes of family members talking
  − Touching by rubbing with lotion or passive range of motion to limbs
  − Visual stimulation if the patient is able to open eyes, by showing family pictures, bright colored objects and favorite items from home

As they progress to Level III, simple questions that have yes or no answers are asked. The patient is asked to follow one-step commands such as "stick out your tongue" or "tap your foot."

Moderate Closed Head Injury: Ranchos Levels IV-VI

Time to recover: 3-6 months

Goals of treatment are related to channeling the patient's excess energy into functional activities that will tire the patient during the day so that they will sleep at night.

Treatment Strategy:
- Increase the patient's tolerance to sitting by leaving them up as long as possible. It may be necessary to redirect activities—for example, pleas to go back to bed since memory is poor they will have forgotten they just got up.
- Supervised unrestrained moments to perform functional activities such as bathing or grooming.
- Give patient ample time to complete tasks since it will take them longer.
- Do not argue with the patient, it will only increase agitation. It is important to try and redirect their attention.
- Continual orientation to their situation.
- Encourage the use of appropriate greeting and social exchanges.

As the patient progresses, independence is improved by decreasing the amount of assistance given with each task. Allow them to function with minimal supervision. Do not overwhelm them with false optimism, but be understanding and supportive without underlying and unrealistic optimism.

Mild Closed Head Injury: Ranchos Levels VII-VIII

Time to recover: 24 hours to 3 months

Goals of treatment are to improve judgment and problem-solving abilities.

Treatment Strategy:
- The current situation and associated injuries, such as extremity injuries with decreased weight bearing, are discussed.
- The patient is to discuss how they would handle a potentially dangerous situation at home such as a fire.
- Practice using a telephone book, a road map, and review the use of 911.
- Games and puzzles are great for problem solving.

As the patient progresses, increase the responsibility level, such as maintaining a medication schedule. The patient can also attempt more complex activities, such as balancing a checkbook or practicing a budget.

Discharge Planning

Discharge planning begins shortly after the patient is admitted to the hospital even if the patient is admitted to the Intensive Care Unit. The social worker or discharge planner will meet with the family to gather pertinent information about the patient and begin providing information and support to the family. A clinical social worker or mental health counselor may be of benefit to the family during this crisis period to provide emotional support and counseling. The discharge planner will collect information from family and significant others regarding the patients support system, past history of mental illness and substance abuse, employment and education history, financial resources, and the patient's prior level of functioning. The family will be informed of community resources that will be of benefit to the patient and family. It is very important that resources such as Social Security Disability, supplemental Security Income, Medicaid, and The Florida Brain and Spinal Cord Injury Program are discussed with the family early as these can be of benefit when developing the discharge plan.

The discharge planner will work with the interdisciplinary team and significant others to develop the plan. Most traumatic brain injury patients require specialized inpatient rehabilitation when the patient is stable to leave the acute medical setting. Patients who do not have significant medical needs and severe cognitive needs may be able to return home with a family supervisor and obtain outpatient rehabilitation. Patient's who have remained in a coma and possibly have respiratory needs, such as tracheostomy or ventilator, are usually discharged to skilled nursing facilities (also known as extended care facilities or nursing homes) that can accommodate these types of patients.

There are many financial and social issues facing discharge planners in working with traumatic brain injured patients that often complicate or delay the discharge. Many patients do not have adequate insurance coverage to pay for post-acute care. In the state of Florida, auto insurance is very limited in coverage. Some programs will provide "scholarships" for patients who have a good functional prognosis and a good support system to provide a stable and supportive post discharge living situation. The discharge planner will assist the family in accessing resource programs including Medicaid and the Brain and Spinal Cord Injury Programs. Unfortunately, the patients who do not have a good social support system will usually be discharged to a skilled nursing facility where they can still receive PT, OT, and ST, although not specialized for brain-injured patients. Each patient and his or her social/financial resources are different. The discharge planner must work with the resources available to make an adequate, safe discharge plan.

Prognosis

The outcome from traumatic brain injury is directly related to a decreased cerebral blood flow, duration of coma and sustained intracranial hypertension that results in ischemic brain injury. About 20% of head injuries result in severe brain injury. Due to the wide spectrum of traumatic brain injuries, it is difficult to predict the outcome from individual to individual. Implementation of the "Guidelines for the Management of Severe Traumatic Brain Injury" developed by the Brain Trauma Foundation, American Association of Neurological Surgeons, the Congress of Neurological Surgeons, and the Joint section on Neurotrauma and Critical Care assist with optimizing the treatment plan.

Prevention

Prevention is a key component in the management of traumatic head injury. Statistics show that injury can be prevented or limited if precautions are taken. The use of helmets has reduced the risk of head injury in cyclists by 80-90%. Motor vehicle manufacturers are improving car designs as a result of crash data collected. This has shown to reduce significant injury with use of seatbelts and air bags that result in the changes currently being made. Substance abuse (alcohol, illicit drugs) is frequently associated with traumatic brain injury. Programs begin in the school systems to educate adolescents and young adults about the dangers associated with the use of drugs. Preventing the devastating effects or traumatic head injury is a primary goal of the trauma management system.

Summary

Limiting or preventing hypoxia, hypercarbia, hypotension, hypovolemia, hypoglycemia, hyponatremia or hypernatremia, and hyperthermia are goals of treatment for the patient with a significant head injury. New studies are underway to evaluate medications for reducing cerebral edema and free radical scavengers to reduce hyperexcitation that results in an increased ICP.

Education & Development Answer Sheet Complete all lines and PLEASE PRINT

Orlando Regional Healthcare Employee: ( ) No ( ) Yes

_____
Employee #   Date

_____
Last Name   First Name   If employee, Department Name & Number

_____
Street Address   City   State   Zip

( ) RN ( ) LPN ( ) Rad Tech ( ) Other _____
License #

Darken the correct circle, you may use pencil or black ink

	A	B	C	D	E			A	B	C	D	E
1.	O	O	O	O	O		26.	O	O	O	O	O
2.	O	O	O	O	O		27.	O	O	O	O	O
3.	O	O	O	O	O		28.	O	O	O	O	O
4.	O	O	O	O	O		29.	O	O	O	O	O
5.	O	O	O	O	O		30.	O	O	O	O	O
6.	O	O	O	O	O		31.	O	O	O	O	O
7.	O	O	O	O	O		32.	O	O	O	O	O
8.	O	O	O	O	O		33.	O	O	O	O	O
9.	O	O	O	O	O		34.	O	O	O	O	O
10.	O	O	O	O	O		35.	O	O	O	O	O
11.	O	O	O	O	O		36.	O	O	O	O	O
12.	O	O	O	O	O		37.	O	O	O	O	O
13.	O	O	O	O	O		38.	O	O	O	O	O
14.	O	O	O	O	O		39.	O	O	O	O	O
15.	O	O	O	O	O		40.	O	O	O	O	O
16.	O	O	O	O	O		41.	O	O	O	O	O
17.	O	O	O	O	O		42.	O	O	O	O	O
18.	O	O	O	O	O		43.	O	O	O	O	O
19.	O	O	O	O	O		44.	O	O	O	O	O
20.	O	O	O	O	O		45.	O	O	O	O	O
21.	O	O	O	O	O		46.	O	O	O	O	O
22.	O	O	O	O	O		47.	O	O	O	O	O
23.	O	O	O	O	O		48.	O	O	O	O	O
24.	O	O	O	O	O		49.	O	O	O	O	O
25.	O	O	O	O	O		50.	O	O	O	O	O

Please also complete the self-learning packet evaluation at the end of the packet.

In order to receive 4.0 contact hours, you must:

- Submit the answer sheet and payment ($10.00 for Orlando Regional Healthcare employees / $20.00 for non-employees) to:

  Orlando Regional Healthcare
  Education & Development, MP 14
  1414 Kuhl Ave.
  Orlando, FL 32806

- Achieve an 84% on the posttest. (You will be notified if you do not pass and will be asked to retake the posttest.)

Post Test

Directions: Complete this test using the answer sheet provided.

Questions 1 – 4: Match the normal physiologic function to the area of the brain

1.   Frontal lobe

2.   Parietal lobe

3.   Temporal lobe

4.   Occipital lobe

a.	Hand skills
b.	Vision
c.	Understanding speech
d.	Judgment

Questions 5 – 8: Match the artery to the brain circulation

5.   Vertebral Artery

6.   Anterior Communicating Artery

7.   Basilar Artery

8.   Middle Cerebral Artery

a.	Anterior Circulation
b.	Posterior Circulation

9.   Which bone is the strongest facial bone?
   A.   Maxillary bone
   B.   Mandibular bone
   C.   Malar bone
   D.   Nasal bone

10.   The first priority in trauma resuscitation of the head-injured patient is:
   A.   Hyperventilation
   B.   Fluid restriction
   C.   Prevention of hypotension and hypoxia
   D.   Repair of skull fractures

11.   Mechanism of injury in head injured patients does not include:
   A.   Acceleration forces
   B.   Deceleration forces
   C.   Rotational forces
   D.   Compression forces

12.   An example of blunt trauma to the head resulting in deceleration forces is:
   A.   Head being hit by baseball bat
   B.   Twisting the head
   C.   Penetrating knife wound
   D.   Forehead hitting windshield

13. Which factor is NOT directly related to the effects of a gunshot wound to the head?
    A.   fragmentation
    B.   cavitation
    C.   air resistance
    D.   shock waves

14. The injury that occurs to the brain as an immediate result of the trauma itself is referred to as:
    A.   Primary injury
    B.   Contrecoup injury
    C.   Secondary injury
    D.   Kernig's sign

15. Cerebral contusions are an example of:
    A.   Primary injury
    B.   Secondary injury
    C.   Cushings triad
    D.   Rotational forces

16. Indicate the clinical examination finding that does not relate to a patient with a basilar skull fracture.
    A.   Periorbital ecchymosis / rhinorrhea
    B.   Oculomotor palsy
    C.   Battle signs / otorrhea
    D.   Hypertension

17. The index of suspicion would be high for a basilar skull fracture located in the _____ when a patient presents clinically with Battle signs.
    A.   anterior fossa
    B.   middle fossa
    C.   foramen magnum
    D.   foramen of monro

18. The following is an example of a secondary brain injury:
    A.   Scalp laceration
    B.   Diffuse brain swelling
    C.   Skull fracture
    D.   Concussion

19. Treatment for a patient with an epidural hematoma requires immediate:
    A.   Mannitol
    B.   ICP monitoring
    C.   Craniotomy - Surgical intervention
    D.   Barbiturate coma

20. Which one of the essential ions plays a key role in cerebral edema formation in penetrating injuries?
    A. Potassium
    B. Magnesium
    C. Calcium
    D. Chloride

21. Some head-injured patients with escalating intracranial pressure develop a sign called Cushings Triad. What are the clinical signs of Cushing's Triad?
    A. Elevated BP, Pulse, and Pulse Pressure
    B. Elevated BP, low Pulse Pressure, and Tachycardia
    C. Low BP, Wide Pulse Pressure and slow Pulse
    D. Increased in Systolic BP, Bradycardia, and Wide Pulse Pressure

22. To prevent secondary injuries from occurring it is important to maintain:
    A. MAP >90 mm Hg, PaO_2 > 90, CPP > 60 mm Hg
    B. MAP < 90 mm Hg, PaO_2 >90, CPP > 60 mm Hg
    C. MAP > 90 mm Hg, PaO_2 > 90, CPP < 60 mm Hg
    D. MAP < 90 mm Hg, PaO_2 >90, CPP < 60 mm Hg

23. Which of the following excitatory neurotransmitter concentration is increased following a traumatic brain injury?
    A. Glutamate
    B. Serotonin
    C. L-aspartate
    D. acetylcholine

24. Brain tissue edema occurs immediately following a gunshot wound to the head because of:
    A. disruption of endothelial tight junctions
    B. direct endothelial membrane injury
    C. disruption of the sodium-potassium pump of the injured cells
    D. increased secretion of cytokines (inflammatory mediators)

Questions 25 and 26 refer to the following scenario:

A 64 year-old male presents with right sided hemiplegia and hemiparesis, aphasia, mumbling, and moves left side to command. PERL 3 mm Sluggish bilaterally. Past medical history includes: hypertension, peripheral vascular disease, carotid endarterectomy. CT scan indicates a hemorrhage.

25. What area of the brain would you expect the hemorrhage to be:
    A. Frontal, Temporal
    B. Temporal, Parietal
    C. Parietal, Occipital
    D. Frontal, Parietal

26. Identify the cerebral artery that ruptured.
    A. Anterior cerebral artery
    B. Internal carotid artery
    C. Middle cerebral artery
    D. Posterior communicating cerebral artery

Questions 27 through 29 refer to the following scenario:

A patient is admitted s/p motorvehicle crash with a diagnosis of severe closed head injury (CHI). Vital signs are as follows: Temperature is 37.5 ° C, HR 108 Regular, BP 158/78, and ICP is 22. He is intubated and on the ventilator spontaneous rate 24, IMV 6, FiO2 30%, TV 650. The patients Glasgow coma scale is 5.

27. The Glasgow Coma Scale of 5 indicates that the patient:
    A. Does not open his eyes, flexes, and has no verbal response
    B. Opens his eyes to painful stimulus, flexes, and has no verbal response
    C. Does not open his eyes, extends, and has no verbal response
    D. Opens his eyes to painful stimulus, extends and responds with incomprehensible sounds

28. Calculate the patient's CPP.
    A. 56
    B. 60
    C. 76
    D. 82

29. You notify the physician of the patients cerebral perfusion pressure (CPP) and increased intracranial pressure (ICP). Which of the following orders would you expect to receive from the physician:
    A. Start a Dopamine infusion at 5 mcg/kg/min
    B. Increase Intravenous fluids to 150cc/hr
    C. Increase FiO2 to 50%
    D. Mannitol 0.25 gm/kg every six hours

End Scenario

30.   Select the first priority in the treatment of a patient admitted with a Glasgow coma score < 8.
        A.   Maintain the airway and place an artificial airway such as endotracheal tube (ETT)
        B.   Do not stimulate the patient
        C.   Initiate subarachnoid precautions
        D.   Sedate the patient

31.   A 21-year old male is admitted with a scalp laceration approximately 5-mm X 7-mm. CT
        scan reveals an open gunshot wound of his right parietal bone with the bullet fragment
        located in the left thalamus. The patient presents with left hemiparesis. Based on the
        knowledge of care for scalp lacerations, management includes:
        A.   debriding the scalp wound and closing it tight, ordering antibiotics to be
                administered and close neurological monitoring
        B.   surgically debriding the dura and brain, and removing the bone fragment
        C.   applying a dressing over the scalp wound, ordering antibiotics to be administered and
                monitoring the patient
        D.   applying a dressing and close neurological monitoring

32.   To maintain cerebral blood flow, autoregulation provides adequate perfusion by regulating?
        A.   cerebral vascular resistance (CVR)
        B.   diastolic blood pressure (DBP)
        C.   systolic blood pressure (SBP)
        D.   heart rate (HR)

33.   Immediate nursing care of a patient with a Le Forte III fracture that has the highest priority
        includes maintaining a patent airway, adequate ventilation, recognizing neurological
        dysfunction, and:
        A.   providing traction to the mandible for stabilization
        B.   evaluating the patient's ability to swallow secretions
        C.   providing nutrition through a nasogastric tube
        D.   controlling bleeding and intravascular volume

34.   Five days ago, a 24 year-old male presented to the emergency department status post motor
        vehicle crash (MVC). A diagnosis of TBI with anterior basilar skull fracture was made. He
        is confused, but follows commands. He has now been transferred to a non-critical care area.
        The physician orders now include increasing the patient's activity to get out of bed. Select
        the clinical assessment findings that the nurse would look for indicating a CSF leak:
        A.   clear drainage from the ear
        B.   clear drainage from the nose
        C.   bloody drainage from a scalp wound
        D.   bloody drainage from the gums

35.   Neurological deterioration in patients with a diagnosis of cerebellar hemorrhage, or posterior fossa craniotomies or sub-occipital craniotomies may increase pressure due to bleeding or edema and lead to:
A.   central herniation
B.   transcalvarial herniation
C.   tonsilar herniation
D.   subfalcine herniation

36.   Traumatic brain injury guidelines were developed to help prevent secondary neurological injuries including hypotension, hypoxia, hypoglycemia and:
A.   hyperthermia
B.   hypercarbia
C.   hyperglycemia
D.   hypertension

37.   What laboratory diagnostic test results indicate that a patient should not receive Mannitol?
A.   Serum sodium 145 mEq/L, Serum Osmo 290 mOsm/L
B.   Serum sodium 135 mEq/L, Serum Osmo 280 mOsm/L
C.   Serum sodium 150 mEq/L, Serum Osmo 300 mOsm/L
D.   Serum sodium 160 mEq/L, Serum Osmo 320 mOsm/L

38.   A severe brain injured patient who is intubated and mechanically ventilated has a GCS of 3 and fixed pupils at 5mm. There is no change from a previous neurological examination. The patient is receiving pentobarbital at 1mg/kg/hr, and Dopamine at 12mcg/kg/min. The EEG monitor indicates a flat waveform. You would:
A.   do nothing, expecting a flat waveform due to the dose of pentobarbital
B.   increase the dopamine drip for cerebral perfusion
C.   notify the physician after troubleshooting the EEG leads
D.   increase the ventilator to provide oxygen to the brain

39.   Goals of the rehabilitation team for a patient on the Rancho Los Amigos Scale Level VI include:
A.   channeling energy into functional activity
B.   improve problem solving abilities
C.   increase their response to environmental stimulation
D.   reviewing maps for directions to businesses

40.   Brain injured patients require the involvement of a rehabilitation team and social worker for discharge planning early in their hospitalization to develop a plan for discharge. Patients with no significant medical needs or no severe cognitive needs are discharged to:
A.   skilled nursing facilities with rehabilitation
B.   extended care facilities with rehabilitation
C.   home with supervision and outpatient rehabilitation
D.   nursing home with rehabilitation

Appendix 1: Glasgow Coma Scale

Eye Opening:

Assesses the functioning of the RAS located in the brainstem. It is responsible for alertness and arousal to the environment (normal wake-sleep cycles).

Verbal Response:

Reflects the functions of the cerebral cortex. It is associated with the RAS and is responsible for awareness of the environment and language content.

Motor Response:

Indicates damage to the cerebral hemispheres, the diencephalon, and/or the brain stem. Measures the function of the corticospinal tract and motor strip in the cerebral cortex. Muscle weakness is a cardinal sign of dysfunction. It is important to note the degree of stimulation required to elicit a motor response in your patient and document. Document the patient's BEST response.

Highest score 15   **Lowest score** 3

Parameter	Score	Response
Eye Opening	4	Spontaneous
	3	To voice
	2	To pain
	1	No response
Verbal Response	5	Oriented
	4	Confused
	3	Inappropriate words
	2	Incomprehensible sounds
	1	No response
Motor Response	6	Follows Commands
	5	Purposeful, localizes
	4	Withdraws to pain
	3	Abnormal Flexion (decorticate)
	2	Abnormal Extension (decerebrate)
	1	No response

Appendix 2: Glasgow Outcome Scale

The Glasgow Outcome Scale places traumatic brain injured patients into one of five categories based upon the degree of their residual function and independence. This scale does not evaluate cognitive function.

Score	Category	Definition
1	Death	
2	Coma/persistent vegetative state (PVS)	Unresponsiveness to internal or external stimuli, no visual cortical function
3	Severe disability (may be conscious)	Dependent for daily support because of mental or physical disability or both
4	Moderate disability	Independent with self-care, but requires a monitored environment
5	Good outcome (recovery)	Functional independence, may or may not have minor deficits. Returns to a full time job comparable to preinjury level. Resumes a normal life.

Appendix 3: Rancho Los Amigos Scale

Levels of Cognitive Function

This scale is used not to measure coma but function of the patient. Patients may be between levels. As the patient begins to wake up, the level should increase with more specific patient responses.

Level I:	**NO RESPONSE**
	– No response to pain, touch, sound or sight

Level II:	**GENERALIZED RESPONSE**
	– Generalized reflex response to pain
	– No voluntary responses

Level III:	**LOCALIZED RESPONSE**
	– Patient reacts specifically but inconsistently to stimuli
	– Responses are directly related to the type of stimulus presented as in turning his head toward a sound
	– Responds to physical discomfort
	– Inconsistent response to commands

Level IV:	**CONFUSED – AGITATED**
	– Alert
	– Very active
	– Aggressive or bizarre behaviors
	– Performs motor activities but behavior is non-purposeful
	– Extremely short attention span

Level V:	**CONFUSED, INAPPROPRIATE, NON-AGITATED**
	– Gross attention to environment
	– Highly distractible
	– Requires continual redirection
	– Difficulty learning new tasks
	– Agitated by too much stimulation
	– May engage in social conversation but with inappropriate verbalizations

Level VI:	**CONFUSED – APPROPRIATE**
	– Inconsistent orientation to time, place
	– Retention span/recent memory impaired
	– Begins to recall the past
	– Consistently follows simple commands
	– Goal-directed behavior with assistance

(Scale continued on next page)

Level VII:	**AUTOMATIC-APPROPRIATE**
	– Performs ADL's in highly familiar environment in a non-confused but automatic, robotic-like manner
	– Skills noticeably deteriorate in unfamiliar environment
	– Lacks realistic planning for own future

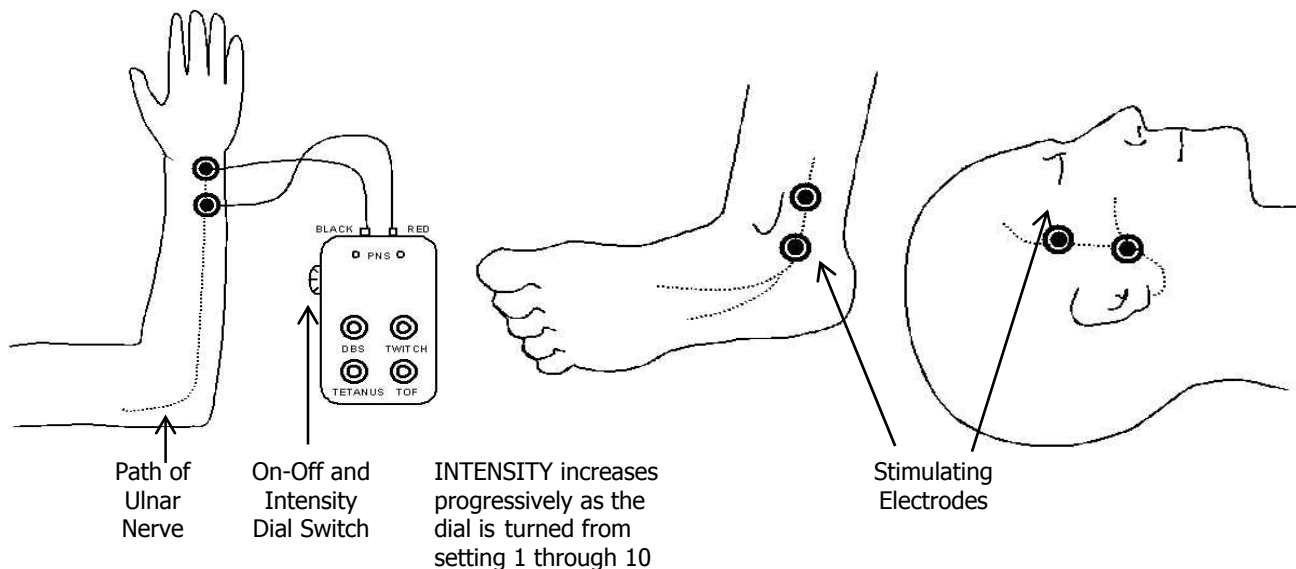| **Level VIII:**	**PURPOSEFUL AND APPROPRIATE**
	– Alert and oriented
	– Able to recall and integrate past and recent events
	– Aware of and responds to his culture
	– Shows carryover for new learning
	– Relative to premorbid skills, he may demonstrate decreased abilities
	– THIS LEVEL DOES NOT NECESSARILY MEAN THE PATIENT IS "OK"

Appendix 4: RIKER Scale (Sedation-Agitation Scale)

Goal of therapy is a level 4.

RIKER SEDATION-AGITATION SCALE (SAS)		
7	Dangerous Agitation	Pulling at ET tube, trying to remove catheters, climbing over bed rail, striking at staff, thrashing side to side
6	Very Agitated	Does not calm, despite frequent verbal reminding of limits; requires physical restraints, biting ET tube
5	Agitated	Anxious or mildly agitated, attempting to sit up, clams to verbal instructions
4	Calm & Cooperative	Calm, awakens easily, follows verbal commands
3	Sedated	Difficult to arouse awakens to verbal stimuli or gentle shaking but drifts off again, follows simple commands
2	Very Sedated	Aroused to physical stimuli but does not communicate or follow commands, may move spontaneously
1	Unarousable	Minimal or no response to noxious stimuli, does not communicate or follow commands

Appendix 5: Train of Four

Train of Four (TOF) Procedure (prior to administration of NMBA)

1. Place stimulator over target nerve (ulnar, facial, or posterior tibial).
2. Negative electrode is placed towards the target site (Red to Black 2–3 inches apart).
3. Depress "TOF" key (4 stimuli at 0.5 second intervals indicated by 4 flashing red light signals). Do NOT remove until light stops flashing.
4. Increase mA until target site responds with 4 equal twitches. Note mA at this point.
5. Increase current in 10mA increments until no further increase in intensity of response – this is known as the Supramaximal Stimulation (SMS) Point. SMS is the ONLY setting used hereafter to monitor the twitch response.
6. Administer the NMBA.
7. Monitor the number of twitches in response to a TOF stimulation indicating the % of blockage, with the preferred level of blockade for optimal patient management at 80–90%.

Path of Ulnar Nerve

On-Off and Intensity Dial Switch

INTENSITY increases progressively as the dial is turned from setting 1 through 10

Stimulating Electrodes

The electrodes can be placed at any point along the course of the ulnar nerve. It is optimal to place the electrodes approximately 2 to 3 inches apart.

(Interpretation of TOF on next page)

Interpretation of TOF

# of Twitches	Extent of Blockade	Clinical Interpretation
4 out of 4 (4/4)	Less than 75%	Spontaneous recovery
3 out of 4 (3/4)	75 – 80%	Maintenance doses may be needed to extend the duration of muscle relaxation
2 out of 4 (2/4)	80 – 90%	Adequate for short-term relaxation and long-term mechanical ventilation
1 out of 4 (1/4)	90%	Conditions suitable for endotracheal intubation and long-term mechanical ventilation
0 out of 4 (0/4)	100%	Considered TOO HIGH a level of blockade. Turn off NMBA until 1/4 or 2/4 responses are obtained.
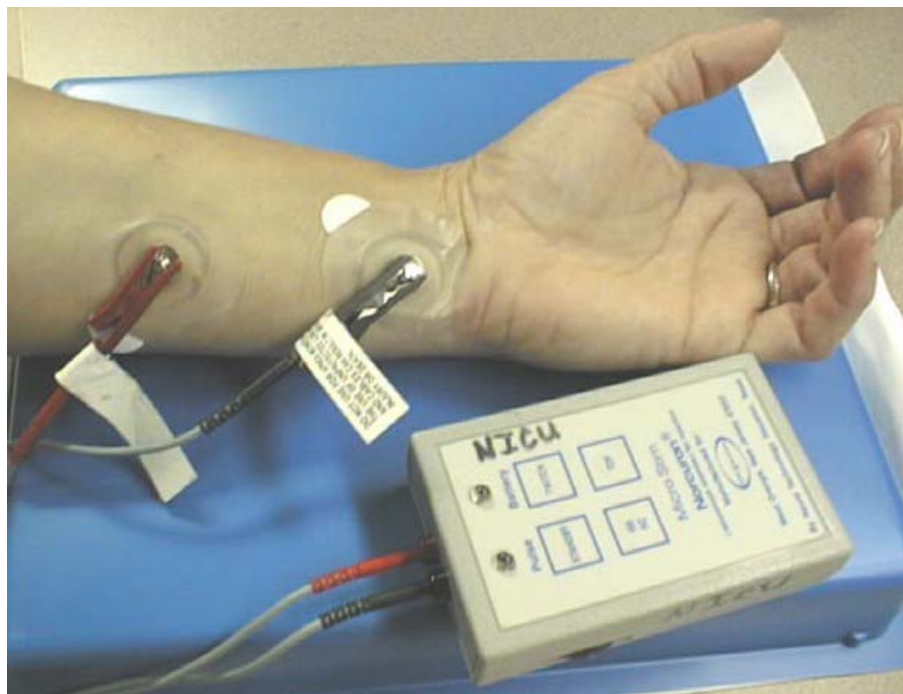
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Web Sites

American Association of Neurological Surgeons and Congress of Neurological Surgeons:
http://www.neurosurgery.org/index.asp

American Trauma Society: http://www.amtrauma.org/

The Brain Injury Association of America: http://www.biausa.org

The Brain Injury Recovery Network Organization: http://www.tbirecovery.org

The Brain Trauma Foundation: http://www2.braintrauma.org/

National Injury Prevention Foundation: http://www.thinkfirst.org/

National Safe Kids Campaign: http://www.safekids.org/

The Traumatic Brain Injury Survival Guide: http://www.tbiguide.com/

Self-Learning Packet Evaluation

Name of Packet: _____ **Date:** _____

Employee ❑   Non-Employee ❑

Your position?

RN ❑	LPN ❑	Respiratory ❑	Radiology ❑
Lab ❑	Social Work ❑	Rehab ❑	Clin Tech ❑

Other: _____

If RN/LPN, which specialty area?

Med/Surg ❑	Adult Critical Care ❑	OR/Surgery ❑	ED ❑
Peds ❑	Peds Critical Care ❑	OB/GYN ❑	L&D ❑
Neonatal ❑	Behavioral Health ❑	Cardiology ❑	Oncology ❑

Other: _____

Please take a few moments to answer the following questions by marking the appropriate boxes.

	Strongly Agree ☺	Agree	Neutral ☺	Disagree	Strongly Disagree ☹
1) The content provided was beneficial.	❑	❑	❑	❑	❑
2) The packet met its stated objectives.	❑	❑	❑	❑	❑
3) The packet was easy to read.	❑	❑	❑	❑	❑
4) The posttest reflected the content of the packet.	❑	❑	❑	❑	❑

5) The course was:

❑ Mandatory

❑ Optional

Please answer the following questions:

How long did this packet take you to complete? _____

What have you learned that you will apply in your work? _____

What was the best part of the packet? _____

What would you suggest be done differently? _____

_____

Additional Comments:

_____

_____

_____

Thank you for your input.

Please return this evaluation to **Education & Development**, either in person or by mail:
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