

# Neurologic Injuries

## LEARNING OBJECTIVES

Upon completion of this chapter, the reader will be able to:

1. Differentiate between various types of neurologic injuries.
2. Discuss the immediate care of the patient with a neurologic injury.
3. Plan the care of the neurologic patient to maximize safety and minimize complications.
4. List optimal outcomes that may be achieved through evidence-based management of neurologic injuries.

Kelly Nadeau

“I have a neuro patient?!” Sometimes those words strike fear in the most experienced nurse, so it is understandable that a nursing student or a new nurse might have a moment of pause when receiving that assignment. It is unclear why neuro patients are so unsettling to so many nurses, but here’s one theory. For each system, there is a “gold standard” for diagnosis. For example, all patients with suspected myocardial infarctions have trending of their cardiac enzymes, especially troponin. This allows for a reasonably certain diagnosis that is based on laboratory or radiology findings. Unfortunately, there really isn’t a defining test like that for neurologically compromised patients. Although radiology and technological advances have progressed, the most reliable indication for neurologically compromised patients remains the trending of neurologic assessment findings and the critical thinking skills of the nurse.

## PATHOPHYSIOLOGY

When a cell is injured, the cell membrane permeability changes. This allows the normally extracellular sodium to rush into the cell. As the sodium enters, water follows it, causing the cell to swell. The sodium–potassium pump kicks into action, trying to rid the cell of the excess sodium. The water that followed the sodium into the cell puts pressure on the mitochondria. (Recall that the mitochondria are the source of energy for the cell.) If the mitochondria are under pressure, they cannot produce the normal amount of energy. The energy from the mitochondria is what fuels the sodium–potassium pump; without energy, the sodium–potassium pump is not efficient and edema continues to accumulate within the cell. The other result of not having the mitochondria as the energy source is that the cell changes from aerobic to anaerobic metabolism, producing lactic acid and free oxygen radicals as by-products.

The next step is critical: Either the source of injury to the cell is removed at this point, allowing the cell to regain aerobic metabolism, rid itself of excess sodium and water, and return to normal functioning, or the cell may rupture. If the cell bursts, the intracellular contents are released, which include lysosomes. These enzymes clean up the debris from the ruptured cell, but also irritate the surrounding cells, causing injury to them, and the process begins again. Although cells can be injured by thermal, mechanical, or toxic sources, the most common cause of cellular injury is hypoxia (Jacobs & Hoyt, 2000).

What does cellular pathophysiology have to do with neurologic injuries? Everything! Once a cell ruptures and dies, new cells will be formed—except in the central nervous system and heart. We do not regenerate cells in the brain or spinal cord at this time, so as cells are lost, so is function. It is imperative to retain as many functioning neurologic cells as possible.

The actual mechanism that causes the neurologic injury is called the primary injury. Except for injury prevention tactics, such as wearing helmets and seatbelts, it isn't possible to eliminate the primary injury as we care for the patient. For example, a primary injury would occur if the patient has a mechanical injury to a cranial vessel from a fragment of fractured skull, causing a hemorrhage. Secondary injury occurs when the cells distal to the injured vessel become hypoxic. The goal in caring for patients with neurologic injuries is to minimize secondary injury.

## INTRACRANIAL PRESSURE AND CEREBRAL PERFUSION PRESSURE

The other critical concepts to understanding head injuries are intracranial pressure (ICP) and cerebral perfusion pressure (CPP). The skull is a closed box with one opening, the foramen magnum. The contents of the skull are brain tissue, blood, and cerebrospinal fluid (CSF). As the amount of one component increases, something else has to decrease. For example, if a patient has cerebral edema causing an increase in the space occupied by brain tissue, blood and/or CSF will be shunted away from the cranial vault to accommodate the swollen tissue. The pressure exerted by the three components within the skull is ICP. If there is an increase in one of the components, the ICP will rise.

Because a continual supply of oxygenated blood is critical for brain cells to function, it is important to maintain a CPP between 60 and 70 mm Hg. Calculation of CPP is described in Chapter 27. Calculation of mean arterial pressure (MAP) is explained in **Box 29-1**.

### Box 29-1

## Calculation of Mean Arterial Pressure

$$\frac{\text{Systolic Pressure} + \text{Diastolic Pressure} (\times 2)}{3}$$

Example: A patient has a blood pressure of 110/65. To calculate the MAP:

$$\text{Diastolic pressure} \times 2 = 65 \times 2 = 130$$

$$\text{Systolic pressure} = 110$$

$$130 + 110 = 240$$

$$240/3 = \mathbf{80 \text{ mm Hg}}$$

## HEAD INJURIES

The most common mechanism of injury for head trauma is motor vehicle crashes. Falls, assaults, and penetration-like gunshot wounds are also frequently seen causes. Head injuries also occur with sports or recreational activities, where the risk of injury increases when the appropriate protective equipment is not used—for example, helmets in football or skateboarding.

### Concussion

The most common closed head injury is a concussion. This condition occurs when the brain is jostled inside the cranial vault. Symptoms will vary depending on which part of the brain is affected. The frontal lobe is often the victim; the symptoms of an injury to this area of the brain are classically repetitive questioning and amnesia of the event since the frontal lobe controls short-term memory. The victim may or may not have a loss of consciousness, nausea, and slight visual disturbances. Headache is a frequent complaint. The computerized tomography (CT) scan will be normal, and symptoms usually clear within 24 hours (Brain Trauma Foundation, 2000; McQuillan & Mitchell, 2002).

### Contusions

Contusions range in severity, depending on which lobe or lobes of the brain are involved. Contusions are bruised areas of brain tissue that become damaged as the brain bounces off ridges of bone inside the skull during a motor vehicle crash or fall, for example. Just as with any bruise, they look worse before they begin to improve, so they will require serial CT scans over several days to watch the contusion “blossom” and then resolve. Reviewing the functions of the lobes of the brain will help guide the assessment of the patient as described in Chapter 26. For example, a patient with an occipital lobe contusion from a backward fall down the stairs will need special monitoring for visual acuity and visual interpretation (Brain Trauma Foundation, 2000; McQuillan & Mitchell, 2002).

### Diffuse Axonal Injury

Diffuse axonal injury is the most severe type of closed head injury. It occurs when the neurons are stretched or sheared so that they can no longer synapse with the next neuron. This type of damage is seen after a deceleration injury, such as a motor vehicle crash or a fall. The patient is usually immediately unresponsive and requires assistance with ventilation. The CT scan may show some slight diffuse edema on presentation, but then appears normal. Complete supportive care is required. Although it is not clear how the mechanism of the recovery works, if the patient is going to arouse and make some type of recovery, that response usually occurs in the first three months after the injury.

There are exceptions to this observation, of course, but the three-month window is a very common guideline (Brain Trauma Foundation, 2000; McQuillan & Mitchell, 2002).

## Fractures

Skull fractures may be linear, depressed, or compound. Linear skull fractures look like a cracked egg shell. They are visible on plain skull films or on CT scan. The danger with linear fractures is that they may have nicked a blood vessel that lies just under the skull itself.

Depressed skull fractures occur when a section or more of bone fragment becomes embedded in the brain tissue. This patient usually requires surgery to debride that section of underlying brain and elevate the skull fragments (Samii & Tataba, 2002).

Compound skull fractures are open fractures with communication between the brain and the environment. These are of concern because if the dura is interrupted, the patient has a greater risk for infection because the blood–brain barrier is violated. Gunshot wounds cause compound skull fractures and then enter into the brain tissue itself.

A basilar skull fracture is a linear fracture at the base of the skull that is associated with a dural tear. Signs and symptoms include spinal fluid leakage from the nose or ears, raccoon's eyes (periorbital ecchymosis), and Battle's sign (bruising on the mastoid bone, behind the ear). Drainage from the nose or ear should be tested for glucose levels using a bedside glucometer. If the glucose of the fluid is greater than 200 mg/dL, it is indicative of CSF. Another technique to test for CSF is to test the leakage on filter paper. The blood will separate from the CSF and leave a halo or ring sign on the paper. However, the presence of a ring sign is not exclusive to CSF. A highly sensitive test for CSF measures beta-2 transferrin, which requires as little as 1/50th of a drop of fluid for analysis (Meco, Oberascher, Arrer, Moser, & Albegger, 2003). Management of basilar skull fracture includes observation for meningitis or encephalitis and for signs of an epidural hematoma. While administration of prophylactic antibiotics is commonplace, data suggest that they are not effective in preventing the development of meningitis in patients with a basilar skull fracture (Ratilal, Costa, & Sampaio, 2006).

## Intracranial Hemorrhage

### Epidural Hematoma

An epidural hemorrhage or bleed is a collection of blood between the skull and the dura. The most common cause is a linear fracture of the temporal bone that nicks the middle meningeal artery, causing a hemorrhage. Patients with epidural hematomas often exhibit a classic symptom pattern. Immediately post-injury, they will experience a brief loss of

consciousness. This often occurs before prehospital personnel arrive, so patients may be unaware they have experienced a loss of consciousness. A period of lucidity follows, which usually coincides with emergency prehospital care. Patients are brought to the emergency department with a Glasgow Coma Scale score of 14 or 15 and equal, reactive pupils. Later during their evaluation, they experience a decline in level of consciousness from increasing ICP. Additionally, their pupils become unequal. The pupil on the side of the hemorrhage will become dilated and nonreactive to light. The other side will still have a round, reactive pupil. This effect is caused by the hemorrhage putting pressure on cranial nerve III (CN III), which controls whether pupils can react to light. With CN III pinched from pressure, it can no longer signal the pupil to constrict to light (Cushman et al., 2001).

Epidural hematomas require immediate surgical intervention to remove the clot. While the risk of infection is always a concern postoperatively, these patients do not have an elevated risk because the meninges and the blood–brain barrier remain intact. With prompt diagnosis and treatment, these patients usually have a good prognosis (Cushman et al., 2001).

### Subdural Hematoma

Subdural hematomas usually have a venous origin and are caused by tears in the bridging veins that connect various hemispheres and lobes of the brain. They vary in terms of their severity and symptoms depending on the size of the vein that is bleeding. Large venous collections may produce lateralizing symptoms of a unilateral fixed and dilated pupil and require immediate surgical intervention. Smaller collections may have more subtle findings. If the patient has a small subdural hematoma that is not producing lateralizing symptoms, the treatment may be close observation with serial CT scans over the next hours to days. These patients have a higher infection risk postoperatively because the meninges must be violated to evacuate the clot (Cushman et al., 2001).

### Subarachnoid Hematoma

A subarachnoid hemorrhage is caused by the rupture of capillaries, usually from shearing injuries that bleed into the subarachnoid space. The blood mixes with the CSF that normally circulates in that space and disperses over the brain. These fluids cause a more global ICP increase. This kind of hemorrhage is considered inoperable because it is spread throughout the entire subarachnoid space in the central nervous system. Blood mixed with the CSF also impairs the reabsorption of CSF through the arachnoid villa, because they may be coated in blood. This can further increase ICP if the CSF begins to accumulate (Cushman et al., 2001).

### Intracranial Hemorrhage

Intracranial hemorrhage within the brain tissue varies in severity depending on its location and the number of areas of hemorrhage. **Box 29-2** and **Box 29-3** detail drug therapy and nursing management for head injuries, respectively.

### SPINAL CORD INJURIES

The same principles of cellular response to injury, primary injury, and secondary injury noted with head injury also apply to spinal cord injury. The goal of treatment is to minimize secondary injury and prevent complications. Secondary damage to the spinal cord can also occur from hypovolemic shock, resulting in hypoperfusion to the spinal cord (Jacobs & Hoyt, 2000).

Common mechanisms of spinal cord injury include motor vehicle crashes, falls, and diving accidents. Take the time to review the motor and sensory functions associated with the various levels of the spinal cord. For example, the cervical area is the most frequently damaged because it is the most mobile. This area controls the phrenic nerve, which exits the spinal cord around C4–C5. The phrenic nerve controls the function of the diaphragm. If this nerve is involved, the patient will experience breathing difficulties, which could be life-threatening and require immediate intervention (Jacobs & Hoyt, 2000).

### VERTEBRAL FRACTURES

Vertebral fractures may occur in any portion of the spinal column and may involve different portions of the vertebrae. Fractures are classified as stable or unstable. Stable fractures have no potential to impinge upon the cord. Unstable fractures may have already caused cord damage or have the potential to cause damage. Patients with such injuries must remain immobilized until the vertebral fracture can be repaired.

### Concussion

The spinal cord may sustain a concussion, similar to the brain in a closed head injury. In such a case, there is a temporary al-

teration in motor and/or sensory function with return to full function.

### Contusion

Cord contusion may occur from a vertebral fracture that bruises the cord or from the cord impacting intact vertebrae in a deceleration injury (from abrupt stopping after high speed). Severity and symptoms depend on the location of the contusion, other physiologic conditions such as hemorrhage, and the amount of cell death.

### Transection

Cord transections may be complete or incomplete. Complete cord transections at the time of the injury cause a total loss of motor and sensory function below the level of the injury. Incomplete transections are much more common and vary in terms of their severity and symptoms. Some common types of incomplete transections are described here:

- Anterior cord syndrome from acute anterior cord compression—exhibits loss of motor function, loss of pain, temperature, crude touch, and pressure. Proprioception, fine touch, fine pressure, and vibration are intact.
- Posterior cord syndrome from acute posterior cord compression—proprioception, vibration, fine touch, and fine pressure are lost. Motor function, pain, temperature, crude touch, and pressure are intact.
- Central cord syndrome from swelling in the center of the cord—loss of motor and sensory function below the level of the lesion with greater loss in the arms than in the legs.
- Brown-Sequard Syndrome from transverse hemisection of the cord—usually caused by a penetrating injury. It results in loss of motor function on the same side as the injury and loss of sensory function on the opposite side.

Remember the principles of cellular injury: What may have started as an incomplete transection may become complete with cellular swelling and rupture. Additionally, a cord

#### Box 29-2

### Drug Therapy

Class	Drug	Indications
Diuretics	mannitol, furosemide	Decreases intracranial pressure
Steroids	dexamethasone, methylprednisolone	Decreases inflammation
Anticonvulsants	phenytoin, fosphenytoin, diazepam, lorazepam, phenobarbital sodium	Inhibits seizures



**Box 29-3****Nursing Management of Head Injury**

Interventions	Rationale
Assess neurological status	Changes may indicate worsening condition
Monitor ICP	Changes may indicate worsening condition
Maintain airway	Hypoxia can cause cerebral vasodilation
Hyperventilation	Maintains PCO <sub>2</sub> at 35 mm Hg to cause vasoconstriction
Minimize stimuli	Decreases ICP
Head of bed elevated	Decreases ICP
Implement early nutrition	Positive nitrogen balance
Emotional support	Provides communication
Education	Provides knowledge of patient condition and treatment

injury at C6, for example, may seem to progress upward to C5 because of the progressive cellular edema post injury. These patients must be reevaluated frequently and consistently to observe for changes in symptoms.

**Neurogenic Shock and Spinal Shock**

Neurogenic shock and spinal shock are frequently seen immediately post-incident in the patient with a spinal cord injury. Neurogenic shock is actually shock that causes cellular ischemia. The mechanism of injury in this case is that sympathetic tracts in the spinal column become damaged and may block sympathetic transmission, leaving parasympathetic symptoms. Patients will exhibit heart rates that are normal to bradycardic, blood pressures that are normal to hypotensive, slowed respiratory rates, and skin that is warm with pink mucous membranes and good capillary refill because veins are dilated and filled with blood. These symptoms are counter to the typical “shock” picture of hypotensive, tachycardic, cool, clammy, and pale and may fool the inexperienced nurse into thinking this patient is compensating well, when actually the blood volume is in the periphery and not perfusing the vital organs of heart, lungs, brain and injured area. This situation can actually lead to increased organ damage due to the enhanced ischemia. The treatment for neurogenic shock is not more fluids; the patient already has blood volume, but it needs to be diverted back to the areas of concern. This type of patient may actually receive a vasoconstrictor to force blood out of the venous storage.

Spinal shock is really a misnomer: It isn’t “shock” at all, in the classic definition of inadequate cellular perfusion. When the spinal cord is injured, regardless of the degree of injury, it seems to shut down to assess the damage. During this time,

all motor, sensory, and deep tendon reflexes are lost below the level of the injury. Because the patient then presents to the emergency department immediately post-injury, there may be flaccidity and total loss of sensation noted. It is impossible to actually know the full extent of the injuries until the spinal shock resolves. This may take hours to days to weeks and will be determined when deep tendon reflexes return. In planning the care of this patient and the family, remember that waiting is difficult. Actual rehabilitation and future planning depend on knowing the extent of injury.

**Care of the Patient with a Spinal Cord Injury**

The priorities of care for injured patients remain the same regardless of the mechanism of injury. The primary survey for injured patients consists of the ABCDs:

- A—airway with cervical spine control
- B—breathing, with particular attention to the cervical area and intercostal muscles
- C—circulation, which includes checking pulses, stopping external bleeding, and obtaining intravenous (IV) access
- D—disability; a quick neurologic exam for responsiveness and pupil checks

After the primary survey is completed and the necessary interventions have been made (e.g., intubation, ventilation, and starting IV fluids), the patient must be assessed thoroughly to identify all other injuries (Moore, Feliciano, & Mattox, 2004).

Immediately post-injury, the patient will require immobilization with a long spine board during assessment of the injury. Spine boards are very hard and cause pressure areas on

**Box 29-4****Nursing Management of Spinal Cord Injury****Interventions**

Assess neurologic status (loss of consciousness, motor, and sensory)  
 Maintain alignment  
 Assess respiratory status  
 Assess for autonomic dysreflexia  
 Provide nutrition  
 Skin care protocol  
 Monitor bowel movement  
 Emotional support  
 Education

**Rationale**

Changes may indicate worsening condition  
 Decreases risk of cord compression  
 Early identification of respiratory dysfunction  
 Removes stimuli causing dysreflexia  
 Balances nitrogen state  
 Prevents skin breakdown  
 Prevents impaction  
 Provides communication and support  
 Provides knowledge of patient condition and treatment

the posterior surface from the occiput to the heels. Minimize the amount of time the patient is on the board as much as possible, and begin skin care early (Russo-McCourt, 2002). **Box 29-4** describes additional nursing management that is required for this patient.

**Steroid Use after Spinal Cord Injury**

Steroid administration is a controversial issue in the treatment of spinal cord injuries. Steroids stabilize cell membranes, which limits cord edema and ischemia in some patients. They are potentially useful in blunt trauma. The initial dose of steroids must be given within the first eight hours of injury.

**Areas of Research**

Scientists and other members of the healthcare community are aggressively researching methods to minimize the effects of spinal cord injury. Much attention was gained for this cause due to the efforts of the Christopher Reeve Foundation after his injury. Currently, an ongoing study called Procord is seeking to determine the effects of taking the patient's own macrophages (a type of white blood cell), treating them in a proprietary process, and then injecting them into the injured area of the patient's spinal cord. This research has produced positive early results in clinical trials. For more information, see [www.proneuron.com](http://www.proneuron.com).

**PATIENT OUTCOMES**

Nurses can help ensure attainment of optimal patient outcomes such as those listed in **Box 29-5** through the use of evidence-based interventions for neurologic injuries.

**SUMMARY**

Nurses working in the intensive care unit (ICU) may encounter a number of patients with a neurologic injury. An understanding of the risk factors, pathophysiologic changes, and management is essential for optimal patient outcomes to occur. One of the pivotal competencies that an ICU nurse must demonstrate when caring for this patient population is advocacy/moral agency. This is in light of the high morbidity and mortality rates reported.

**Box 29-5****Optimal Patient Outcomes**

- Uses effective coping strategies
- Cerebral perfusion pressure in expected range
- Mean arterial pressure in expected range
- Intracranial pressure in expected range
- Airway remains patent
- Physical comfort in expected range
- Modifies lifestyle as needed

## CASE STUDY

A 28-year-old male patient arrives at a trauma center following a fall from his roof. He has an open tibia fracture, a bruise to his left lower chest, and a tender left upper quadrant. He presents with a Glasgow Coma Scale score of 8 and is moving all extremities. His right pupil is fixed and dilated, and his left pupil is normal size and reactive to light. Even without an ICP monitor, it can be safely assumed that his ICP is elevated. His blood pressure on admission is 90/60, so his MAP is 70 mm Hg. A MAP of 70 mm Hg, after subtracting an elevated ICP, drops the CPP to below 60 mm Hg and is not allowing for adequate perfusion, which will increase the amount of secondary brain injury.

### CRITICAL THINKING QUESTIONS

1. Which disciplines should be consulted to work with this client?
2. What types of issues may require you to act as an advocate or moral agent for this patient?
3. How will you implement your role as a facilitator of learning for this patient?
4. Write a case example from the clinical setting, highlighting one patient characteristic. Explain how the characteristic was observed through subjective and objective data.
5. Utilize the form to write up a plan of care for one client in the clinical setting.
6. Write a case example from the clinical setting. Rate the patient as a level 1, 3, or 5 on each characteristic. Identify the level of nurse characteristics needed in the care of this patient.
7. Take one patient outcome for a patient and list evidence-based interventions found in a literature review for this patient.

### Using the Synergy Model to Develop a Plan of Care

	Patient Characteristics	Subjective and Objective Data	Evidence-based Interventions	Outcomes
SYNERGY MODEL	Resiliency			
	Vulnerability			
	Stability			
	Complexity			
	Resource availability			
	Participation in care			
	Participation in decision making			
	Predictability			

## Online Resources

**Proneuron Technologies:** [www.proneuron.com](http://www.proneuron.com)

**Steroids for Spinal Cord Injury:** [www.trauma.org/spine/steroid.html](http://www.trauma.org/spine/steroid.html)

**National Spinal Cord Injury Association:** [www.spinalcord.org](http://www.spinalcord.org)

**Spinal Cord Injury Resource Center:** [www.spinalinjury.net](http://www.spinalinjury.net)

**Brain Injury Resource Center:** [www.headinjury.com](http://www.headinjury.com)

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