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Cervical Spinal Cord Injury

Melissa Schimmel
Otterbein University, melissa.schimmel@otterbein.edu

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Melissa Schimmel, RN, BSN

Otterbein University, Westerville, Ohio

Introduction

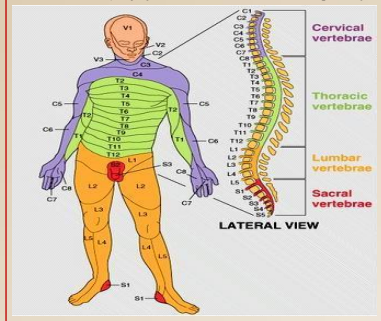
A spinal cord injury (SCI) can occur at any level of the spinal cord. A SCI involves an interruption between the brain and the body due to an injury or damage to the spinal cord (Sheppard Center, 2011, p1.). The location and extent of the injury is important because it will determine the severity and extent of dysfunction. The higher the injury is to the spinal cord, the more severe the loss of motor function (Sheppard Center, 2011, p.4). The spinal cord injuries are classified by based on the extent of injury, motor and sensory losses. The injury is considered complete when there is no motor or sensory function below the level of the injury (NINDS, 2013, p.4). An incomplete injury means there is still some sensory function and possibly some voluntary motor activity below the level of injury (NINDS, 2013, p.3). If all four extremities are affected, the patient is classified as a tetraplegic or quadriplegic (Sheppard Center, 2011, p. 4).

There are also a multitude of medical emergencies and complications that can arise with spinal cord injuries. The two most common life threatening emergencies related to spinal cord injuries are spinal shock and autonomic dysreflexia. These patients are also at high risk for several other complications such as pneumonia, circulation issues, spasticity and muscle wasting, pressure sores, chronic pain, bowel and bladder problems, sexual dysfunction and depression (NINDS, 2013, p.4-5). These complications can haunt spinal cord injury patients for the rest of their lives causing them to have several admissions to the hospital each year.

Understanding the pathophysiology of spinal cord injuries to ensure the most effective and beneficial treatments are available and provided to every SCI patient. Also having a true understanding of what is occurring at the injury site allows healthcare providers to be prepared and equipped to educate the patients on different phases of the spinal cord injury and what the patients should expect next.

Pathophysiological Process

The spinal cord is a complex structure that is composed of nerves that serve as a communication system for the body (Sarhan, Saif & Saif, 2013, p.319). The spinal cord relays sensory information along with motor and autonomic functions to and from the brain. A bony vertebral column composed of seven cervical, twelve thoracic, five lumbar and five sacral vertebral segments protect the spinal cord from injury (Sarhan, Saif & Saif, 2013, p.319).



A spinal cord injury occurs when there is any damage to the spinal cord that blocks communication between the brain and the body (Shepherd Center, 2011, p.1). When the vertebrae are dislocated or broken, pressure is placed on the spinal cord and destroys the sensitive axons that carry signals up and down the spinal cord. This is considered the primary phase of injury. Minor injuries to the spinal cord typically doesn't cause nerve cell death, but can cause demyelination without axonal damage (Colangelo, 2014, p.37). Major injuries and pressure on the vertebrae or can cause complete cell death across the spinal cord resulting in nerve cell death or paralysis (Colangelo, 2014, p.37). The cell death occurs because the spinal cord becomes edematous causing ischemia to the tissue, which quickly results in necrosis from a lack of blood and oxygen (Sarhan, Saif & Saif, 2013, p. 322).

The secondary injury phase begins after the extent of ischemia is determined. The secondary phase is the spreading of tissue damage from the injury site due to increasing edema and vascular damage (Sahran, Saif & Saif, 2013, p.322). This sets off a series of physiological and biochemical reactions in the spinal cord. Macrophages, lymphocytes and activated microlia and demyelinating axons are present within the spinal cavity. The astrocytes reproduce and surround the cord in an attempt to reduce the spread of the lesion, forming a scar (James, Rowland & Fehlings, 2013, p.41).

The fibrous scar is deposited in and around the lesion. The severed ends of the axons degenerate and are eventually phagocytosed by macrophages (James, Rowland & Fehlings, 2013, p. 41).

The acute pathophysiologic process follows the secondary injury phase. The acute pathophysiologic processes include vascular disruption, energy and electrolyte imbalances, inflammation and necrotic and apoptopic cell death. Vasospasms, hemorrhages and impaired autoregulation of intact vessels lead to vascular changes and disruption. Autoregulation of the blood pressure is lost which stimulates a reduction of blood flow through the spinal cord and is followed by hypotension (Sarhan, Saif & Saif, 2013, p.323).
Electrolyte imbalances also play a role in cell death. The injury causes an increase in intracellular sodium and calcium due to failure of ion pumps, inactivation of ion channels and membrane repolarization which in turn overexcites other neurons triggering a series of destructive events. Excessive intracellular calcium activates protein kinases and proteases resulting in cell death (James, Rowland & Fehlings, 2013, p. 43). High calcium levels also affect the mitochondria which increases reactive oxygen species (ROS) production in neurons and glia (James, Rowland & Fehlings, 2013, p. 43). The changes in the metabolism depletes ATP, drops blood glucose and increases lactate levels causing hypoxia which leads to apoptopic cell death (James, Rowland & Fehlings, 2013, p. 43).

The inflammation response after a SCI occurs within hours of the onset of injury. Neutrophils flood the injury site and secrete lytic enzymes along with cytokines such as tumor necrosis factor, interleukins and interferons (Sarhan, Saif & Saif, 2013, p.323). These enzymes may further damage local tissue and recruit other inflammatory cells like monocytes, macrophages and local microglia that will phagocytose the injured site (Sarhan, Saif & Saif, 2013, p.323).

Necrotic and apoptopic cell death is possible due to the ischemia, excitability of metabolism and oxidative stress related to the spinal cord injury. The necrosis can't be stopped, but the amount of apoptopic cell death can be slowed or stopped depending on the treatment and interventions the patient receives.

Dysfunction Severity Related to Injury Level

Cervical spine injury symptoms vary depending on the level of injury. Injuries to cervical nerves C1 to C4 are the most severe causing paralysis in all four extremities and torso. Patients with C1 to C4 injuries typically lose the ability to breathe, cough, and control their bowel and bladder functions (Shepherd Center, 2011, p.4). These patients are at risk for immediate, life-threatening cardiovascular complications due to a blocked sympathetic nervous system as well as a low body temperature, impaired peristalsis, and bradycardia (Colangelo, 2014, p37).

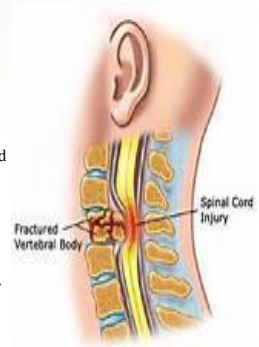
Injuries to cervical nerves C5-C8 are usually less severe but still involve some paralysis or limited movement and sensation. Patients with C5-C8 injuries have the potential to breathe and cough on their own. Injuries at C5 or C6 typically include total or partial paralysis in hands, wrists, trunk, and legs and complete incontinence of bowel and bladder (Schepherd Center, 2011, p.5).

Patients with C5 or C6 injuries should have some movement of the arms, elbows or wrists (gross motor movement) depending on the injured area and may be able to manage their bowel and bladder functions with special equipment (Shepherd Center, 2011, p.5).

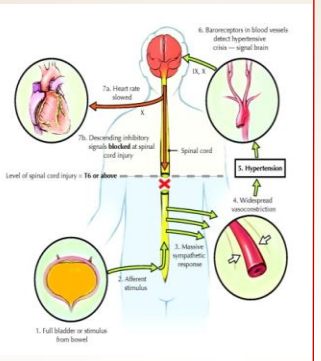
Patients with C7 or C8 nerve injuries generally have more movement than those with C1-C6 injuries. Most of these patients can straighten their arms, have full movement in their shoulders and some can grasp and release objects with their hands (Shepherd Center, 2011, p.5). Voluntary bowel and bladder control is still lost, but manageable with special equipment. Most patients with C7 or C8 nerve injuries are able to do most activities of daily living without assistance and some can even drive a vehicle that is adapted to their needs (Shepherd Center, 2011, p.5).

Spinal shock

Spinal shock occurs with physiologic or anatomic transaction of the spinal cord and can occur within an hour of initial injury and can last for several hours to several days (Atkinson & Atkinson,p 385). Typically, the more severe the transection of the spinal cord, the more extreme state of spinal shock occurs. Spinal shock results in a complete loss of reflex function below the level of injury due to increasing pressure and/or swelling of the spinal cord (NINDS, 2013, p.2). With higher level cord transections, it is more likely that the distal sacral reflex arcs may remain intact but depressed (Atkinson & Atkinson, p.386). The reflexes above the level of injury may also be depressed due to a loss of connection to the ascending distal spinal cord (Atkinson & Atkinson, p. 386). The decrease in sympathetic tone causes moderate to severe hypotension, respiratory failure, lack of body temperature control and decreased or loss of bowel and bladder control.



Autonomic Dysreflexia



Approximately 50-90% of all spinal cord injuries above T6 will encounter a life threatening emergency called autonomic dysreflexia (AD) (Colangelo, 2014, p38). Autonomic dysreflexia causes severe vasoconstriction and an acute onset of hypertension, reflex bradycardia, anxiety, and sweaty, flushed skin because the brain is unable to receive the message from the body that there is something wrong (Colangelo, 2014, p39). This occurs because there is an imbalance in the body systems that control blood pressure. The body controls blood pressure by tightening or relaxing little muscles around blood vessels (NSCIA, 2011, p.2). When the vessels are smaller (tightened), the blood pressure is higher so the blood can be circulated through the body. However, when a patient has a SCI this body system is interrupted. The signal that tells the blood vessels to relax (lower the pressure) is not able to get through the spinal cord because of the injury (NSCIA, 2011, p.2-3). This broken system causes the blood pressure to continue to rise and can lead to a stroke or death if not treated.

Autonomic dysreflexia is triggered by a noxious stimulus below the level of injury that would normally cause pain (NSCIA, 2011, p.3). The most common stimulus is the bladder either being overstretched or irritated due to urinary retention, UTI or non-compliance with intermittent catheterization. The irritated bladder tries to send a message to the brain through the spinal cord that it needs help but the reflex signal can't get through the injury site so the pressure continues to build causing an increase in blood pressure.

Nursing Implications

The major role for nurses is providing the correct treatments and medications immediately after the SCI occurs. The nurses must monitor the patients closely for spinal shock, cardiopulmonary dysfunction and autonomic dysreflexia (Phan, 2013, p.84). The nurses must also monitor electrolytes, body temperature and skin condition. The nurses are also responsible for educating the patients and their families about spinal cord injuries and what to expect each day.
There also needs to be a multidisciplinary team approach with SCI patients. The nurse should lead the team and ensure that all disciplines are involved and active in the plan of care of each patient. Rehabilitation services, social work, dieticians, behavior health and the trauma physicians all need to be included in the care of SCI patients.

Conclusion

Spinal cord injuries are very complex. A spinal cord injury is going to significantly change patients' lives forever. Healthcare providers are responsible for educating and ensuring the patients that they are getting the best treatment and resources available for them. There is currently no cure for spinal cord injuries, so it is imperative that healthcare providers understand the pathophysiology of these injuries to ensure every patient who sustains a SCI gets the highest quality of care possible.

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