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Cervical Spinal Cord Injury

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Recommended Citation
The spinal cord is a complex structure that is composed of nerves that serve as a communication system for the body (Saran, Safi, & Asl, 2013, p.319). The spinal cord relays sensory information along with motor and autonomic functions to and from the brain. It controls the body's voluntary and involuntary activities. Dysfunction of the spinal cord can occur at any level of the spinal cord (Saran, Safi, & Asl, 2013, p.319). Any spinal cord injury (SCI) can affect any aspect of a patient's body, depending on the level of injury and the extent of the injury. The extremities are affected more than the trunk because the spinal cord functions are regulated by the brain. The higher the injury is in the spinal cord, the greater the extent of the injury. There is a direct correlation between the extent of the injury and the severity of the SCI. If the injury is complete, there is no motor or sensory function below the level of the injury (NINDS, 2013, p.3). The higher the lesion, the more severe the injury, because the higher level of the spinal cord controls the lower levels of the body. If the injury is partial, there is partial function below the level of the injury (NINDS, 2013, p.3). Full autonomic functions are affected. The patient is classified at a tetraplegic or quadriplegic level (Shepherd Center, 2011, p.4).

There are also a multitude of medical emergencies and complications that can arise with spinal cord injuries. The most common life-threatening emergencies related to a spinal cord injury are related to ischemia (Saran, Safi, & Asl, 2013, p.319). Ischemia means there is still some sensory function and possibly some voluntary motor activities below the level of injury (NINDS, 2013, p.3). This broken system causes the blood vessels to become constricted. The blood vessels in the brain are supplied by the blood flow through the spinal cord and the blood flow to the brain is decreased. This can cause cell death (Saran, Safi, & Asl, 2013, p.323).

A spinal injury occurs when there is any damage to the spinal cord that blocks communication between the brain and the body (Shepherd Center, 2011, p.4). When the vertebrae are dislocated or broken, they can be displaced on the spinal cord and destroy the sensitive axons that carry signals up and down the spinal cord. This is considered the primary phase of injury. Minor injuries such as compression fractures, contusions, bruised, and bleeder problems, and soft tissue injuries can cause complete cell death across the spinal cord (Saran, Safi, & Asl, 2013, p.4). These complications can cause cell death if the injury is not treated because the level of the injury continues to increase. After 72 hours of injury, the level of injury remains the same. The blood vessels in the brain are supplied by the blood flow through the spinal cord and the blood flow to the brain is decreased. This can cause cell death (Saran, Safi, & Asl, 2013, p.323).

The secondary injury phase begins after the extent of ischemia is determined. This secondary phase is the spreading of tissue damage from the injury site allowing for cell death (Saran, Safi, & Asl, 2013, p.323). These injuries may further damage local tissue and recruit other inflammatory cells like macrophages, lymphocytes, and neutrophils. The secondary injuries cause inflammation and vasodilatation in the spinal cord. Macrophages, lymphocytes, and neutrophils within the spinal cord are part of the innate immune response and are capable of recruiting inflammatory cells to reduce the spread of the lesion, forming a scar (James, Rowland & Foldings, 2013, p.41).

**Pathophysiologic Process**

Spinal shock occurs when there is a loss of communication between the brain and the body (Saran, Safi, & Asl, 2013, p.45). High calcium levels also affect the mitochondria which increases reactive oxygen species (ROS) production in neurons and glia (James, Rowland & Foldings, 2013, p.45). Increasing levels of calcium in the metabolism-depletes ATP, drops blood flow and increases local lactic acid levels causing injury to the spinal cord neurons (James, Rowland & Foldings, 2013, p.45). The cells die off because the blood supply to the spinal cord is insufficient. The tissue that is damaged cannot receive nutrients or oxygen. The spinal cord continues to die because the unresolved issues cause ischemia to the tissue, which quickly results in increased loss of blood and oxygen (Saran, Safi, & Asl, 2013, p.322).

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The inflammatory response after a SCI occurs within hours of the event (Saran, Safi, & Asl, 2013, p.323). Neuronal cells release cytokines and secrete inflammatory enzymes along with cytokines that cause increased gene expression (James, Rowland & Foldings, 2013, p.323). These enzymes may further damage local tissue and recruit other inflammatory cells like macrophages, lymphocytes, and neutrophils that will phagocytose the injured site (Saran, Safi, & Asl, 2013, p.323).

Neurogenic and neuropathic pain is possible due to the ischemia, edema, and metabolic changes in the spinal cord (James, Rowland & Foldings, 2013, p.41). The flow of blood to the spinal cord decreases, leading to an increase in the temperature control and cardiovascular disorders of low blood pressure (Shepherd Center, 2011, p.5).

**Pathophysiology**

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