Cervical Spinal Cord Injury

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The spinal cord is a complex structure that is composed of nerves that serve as a communication system for the body (Sarhan, Safi, & Alfalah, 2013, p.319). The spinal cord relays sensory information along with motor and autonomic functions to and from the brain. The spinal cord consists of seven cervical, twelve thoracic, five lumbar and five coccygeal vertebrae that provide a barrier to the spinal cord from injury (Sarhan, Safi, & Alfalah, 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, pinching or 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 may limit motor and sensory function of the nerve cell body, but can cause demyelination without neural damage (Fehlings, Sarhan, & Saif, 2013, p.47). Major injuries and pressure on the vertebrae or can cause complete cell death across the spinal cord due to the severing in the spinal cord (Colangelo, 2014, p.37).

The spinal shock occurs because the spinal cord becomes edematous and causes ischemia to the tissue, which quickly results in increased flow from a lack of blood and oxygen (Sarhan, Safi, & Alfalah, 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 original trauma (Brower, John, & Pfeiffer, 2011, p.323). These injuries may further damage local tissue and recruit other inflammatory cells like monocytes, macrophages and local microglia that will phagocytose the injured site (Sarhan, Safi, & Alfalah, 2013, p.323).

Necrotic and apoptotic cells death is possible due to the ischemia, excitotoxicity and metabolic stress related to the spinal cord injury. The necrosis can be stopped, but the amount of apoptotic cell death can be slowed or reversed depending on the treatment and interventions the patients receive. 

The fibrous scar is deposited in and around the lesion. The fibrous tissue is a combination of connective tissue and is eventually replaced by macrophages (James, Rowland, & Fehlings, 2013, p.41).

The acute pathophysiological process follows the secondary injury phase. The acute pathophysiology includes cellular disruptcy, energy and electrolyte imbalances, inflammation and necrotic and apoptotic cell death. The episodes of injured neuronal cells at the site of the impalpable antegrade of intact vessels leads to autoregulation of blood flow. The autoregulation of blood flow is lost when the secondary phase obscures blood flow through the spinal cord and is expressed in patients (Sarhan, et al.,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 metabolism peripheral which in turn overexes other neurons triggering a series of destructive events. Excessive intracellular calcium activates protein kinase and proteases resulting in cell death (James, Rowland, & Fehlings, 2013, p.45). High calcium levels also affect the mitochondria which increases reactive oxygen species (ROS) production in neurons and glia (James, Rowland, & Fehlings, 2013, p.45). Changes to the metabolism depletes ATP, drop glucose and increase lactate levels. Ischemic injuries to the spinal cord (James, Rowland, & Fehlings, 2013, p.43).

The inflammation response after a SCI occurs within hours of the onset of injury. Neurovascular fluid increases in the leaky tissue and secrete cytokines enzymes along with cytokines such as tumor necrosis factor, IL-1 and interferons (Sarhan, & Alfalah, 2010, p.23). These enzymes may further develop local tissue and recruit other inflammatory cells like monocytes, macrophages and local microglia that will phagocytose the injured site (Sarhan, Safi, & Alfalah, 2013, p.323).

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Cervical spine injury depends on the level of injury. Patients with C1 to C4 or the more severe injuries have paraplegia and respiratory function (Atkinson, Shepherd, & Colangelo, 2013, p.4). These patients are at risk for dysreflexia. A C3-C4 spinal cord injury typically includes to total or partial paralysis, as well as torso, arms, hands, face and limbs. (Colangelo, 2014, p.37). Patients with C5 or C6 injuries are usually less severe but still require constant monitoring and movement and sensation. Patients with C7 injuries have the potential to breathe and cough on their own. Injuries at C7 or C6 typically include total or partial paralysis, as well as torso, arms, hands, face and limbs. (Colangelo, 2014, p.37).

Dysreflexia occurs in patients with C6-C8 injuries at the site where there is still some sensory function and is a reaction in the spinal cord. Macrophages, astrocytes and lymphocytes are recruited to the injury site due to increasing edema and vascular changes and disruption. The secondary phase is the spreading of tissue damage from the injury site due to increasing edema and vascular changes and disruption (Shepherd Center, 2011, p.323). These enzymes may further develop local tissue and recruit other inflammatory cells like monocytes, macrophages and local microglia that will phagocytose the injured site (Sarhan, Safi, & Alfalah, 2013, p.323).

Almost 50-90% of all spinal cord injuries above T6 will experience a life-threatening emergency called autonomic dysreflexia (AD) (Colangelo, 2014, p.38). Autonomic dysreflexia causes severe vasoreactivity and an acute onset of hypertension, reflex bradycardia, anxiety and nausea. Flushed skin because the brain is unable to receive the message from the body that there is something wrong (Colangelo, 2014, p.37).

This happens because there is an imbalance in the homeostatic balance of blood pressure. The blood pressure controls blood pressure by tightening or relaxing little muscles around blood vessels (NSGA, 2013, p.2). When the vessels are smaller (tightened), the blood pressure is higher so the blood can be circulated through the body. However, a patient with SCI this body system is interrupted. The signal that tells the blood vessels how to relax (lower pressure) is not present to get through the spinal cord because of the injury (NSGA, 2013, p.2-3). This broken system causes the blood pressure to continue to lead to a stroke or death if not treated. Autonomic dysreflexia is triggered by a neurostimulus below the level of injury that would normally cause pain (NSGA, 2013, p.313). The most common stimulus is the bladder either being overactive due to spinal cord injury or a neurostimulus (NSGA, 2013, p.316). The reflux above the level of injury may also be depressed, causing a dysreflexic event. The connection to the autonomic spinal cord injury (Atkinson & Alidomin, 2013, p.366). The delayed reflexes below the level of injury may be due to a motor or sensory deficit and moderate to severe hypotension, excitement or hypoxia as an event (NSGA, 2013, p.316). Irritated bladder signals 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 as the pressure can't cause enough of a signal in blood pressure.