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Acute Traumatic Spinal Cord Injury

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ABSTRACT
Approximately 17,000 new spinal cord injuries occur each year in the United States, with motor vehicle collisions and falls being the most common causes. Even though 94% of patients survive the initial injury and corresponding hospitalization, their life expectancy is reduced secondary to the long-term complications that stem from their injury. Every patient should be approached in the same manner (i.e., as if they have a spinal cord injury) until proven otherwise to prevent additional injury. Important considerations that should take place in the emergency department include the patient's airway, the presence of shock, and the prevention of complications secondary to the primary injury. This article briefly summarizes the epidemiology and pathophysiology of spinal cord injuries and the therapies that may be recommended and initiated following a thorough assessment of the patient.
Key words: atropine, hemodynamics, monitoring, spinal cord injury, steroids, vasopressors

MEDICAL ATTENTION is a priority when it comes to trauma patients, but this is especially true in the setting of head and/or neck trauma. There is no way to reverse damage to the spinal cord when injury occurs; however, there are important concepts that should start in the emergency department and continue with the inpatient admission and discharge processes. Pertinent patient care issues that need to be addressed upon arrival to the emergency department include, but are not limited to, the patient's airway, circulation and the prevention and/or treatment of shock, immobilization and identification, and prevention of complications due to the primary injury. In addition to these interventions, intravenous methylprednisolone may be considered despite the controversy that surrounds this pharmacologic intervention (Sandean, 2020).

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SPINAL CORD INJURY DISEASE REVIEW

Epidemiology

According to the most recent data from the National Spinal Cord Injury Statistical

Center, there are approximately 17,900 new spinal cord injury (SCI) cases each year in the United States. This equates to approximately 54 cases per 1 million people. The number of individuals with SCI living in the United States is approximately 296,000. The economic impact of SCI is enormous, with estimated lifetime costs (health care costs and living expenses) ranging from $1,217,266 to $5,162,152 depending on age and severity of injury.

Approximately 78% of all new SCI cases since 2015 are male and the average age at injury is 43 years. Vehicle crashes (38.2%) are the most common cause of SCI followed closely by falls (32.3%). Other less common causes include violence, sports, and medical/surgical complications. In terms of neurological category, incomplete tetraplegia accounts for a majority of SCIs at 47.4%. This is followed by complete paraplegia (19.9%), incomplete paraplegia (19.7%), and complete tetraplegia (12.4%). Less than 1% of individuals experience complete recovery. Among persons enrolled in the National SCI Database since 1973, disease of the respiratory system was the leading cause of death. Death related to infectious and parasitic diseases was the second leading cause and cancer ranked third (National Spinal Cord Injury Statistical Center, 2020 and 2021).

Pathophysiology

Primary Injury

SCI is a devastating neurological condition that alters the physical, social, and emotional well-being of patients. SCI occurs as a result of damage to the spinal cord due to some form of trauma. Consequently, a degenerative loss of motor, sensory, and autonomic function occurs (Hamid et al., 2018). Primary injury can be described as the initial mechanical injury on the spine that produces immediate mechanical disruption and dislocation of the spinal column. The insult causes fracture and/or dislocation of the vertebrae as well as compression and/or laceration/transection of the spinal cord. Subsequently, a disruption in axons, blood vessels, and cell membranes occurs.

Spinal cord compression is the most frequent mechanism of SCI and correcting this issue is often a cornerstone of the acute treatment for patients (Zhang et al., 2021). Regardless of the form of primary injury, direct damage to the ascending and descending pathways in the spinal cord causes a sustained injury cascade, which leads to further spinal cord damage and neurological complications. The first detectable pathological change following primary injury is swelling of the spinal cord (Rowland, Hawryluk, Kwon, & Fehlings, 2008). This process disrupts the normal vasculature and compromises the blood–spinal cord barrier. Also, hemorrhaging and/or hypoperfusion of both the gray and white matter is commonly seen. Several other pathophysiological processes have begun but may not yet be observable. This includes a pro-inflammatory cytokine response (elevations in tumor necrosis factor [TNF]-α, interleukin [IL]-β, and IL-6), an increase in free radical production, and excitotoxic levels of extracellular glutamate (Rowland et al., 2008).

Secondary Injury

Following primary injury is several pathophysiological processes leading to prolonged secondary injury. Secondary injury develops over a period of hours after the initial trauma due to a host of intracellular, molecular, and biochemical changes. Secondary injury can be divided into multiple contiguous phases, which includes the acute (2–48 hr), subacute (2 days to 2 weeks), intermediate (2 weeks to 6 months), and chronic stages (>6 months) after SCI (Rowland et al., 2008).

During the acute phase, there is a continuation of the primary injury marked by a continuation in hemorrhage, ischemia, inflammation, cytotoxic edema, free radical, and pro-inflammatory cytokine production, and glutamate-mediated excitotoxicity. Progressively, these processes add to spinal cord swelling and trigger cell dysfunction and death by necrosis or apoptosis (Ahuja et al., 2017).

The subacute phase is categorized by further ischemia that occurs secondary to cytotoxic edema and vessel hemorrhage. Additionally, excitotoxicity and cellular death continue. One such population of cells that are majorly affected is the astrocytes in the lesion core. In the hours to days following injury, these cells undergo necrotic cell death. Soon to follow is an astrocytic response in which the astrocytes proliferate and tightly interweave into cytoplasmic processes to form glial scar (Ahuja et al., 2017; Rowland et al., 2008). This astroglial scarring, in turn, potentially restricts axon regeneration. One beneficial process that does occur during the subacute phase is referred to as phagocytic response (Rowland et al., 2008). This cellular response plays an important role during tissue remodeling and wound healing (Garcia-Garcia & Rosales, 2006). Phagocytic inflammatory cells clear myelin debris at the injury site and promote axon growth (Ahuja et al., 2017; Rowland et al., 2008). However, in addition to having beneficial effects, this process prevents restoration.

In the intermediate phase, there is continued astroglial scarring and axonal remodeling. However, to complicate matters, after the acute inflammatory response subsides, cystic cavitations begin to form in response to vast cell death and degeneration. Like astroglial scarring, these cavities coalesce and become a barrier to axon regeneration (Ahuja et al., 2017). Nonetheless, regenerative axonal sprouting does occur in the intermediate phase.

Approximately 6 months following injury, the chronic phase begins. This phase continues throughout the lifetime of the patient as long-term complications exist, and is mainly characterized by maturation/stabilization of glial scars and cystic cavities. Together, these processes inhibit axonal regeneration in the area bordering the lesion. Additionally, disturbances such as demyelination, wallerian degeneration, oligodendrocyte apoptosis, and collagen fiber scarring occur (Garcia, Rodriguez-Barrera, Mondragon-Caso, Carvajal, & Ibarra, 2018).

Systemic Compromise

Patients experiencing SCI develop complications characterized by organ dysfunction or failure and can range from mild to severe. The pathophysiology regarding systemic compromise is very complex, which can include neurogenic causes or a complication of treatment itself. Organ systems commonly affected include the cardiovascular, muscular, nervous, respiratory, skeletal, and urinary systems. Furthermore, immune suppression can be induced by SCI, which puts patients at increased risk of various infections such as pneumonia, urinary tract infections, and wound infections (Ahuja et al., 2017).

The degree of organ dysfunction after SCI mainly depends on the site of injury within the spine and the completeness of said injury (Sun et al., 2016). The site of the injury largely determines the effect on motor and nonmotor function. In terms of completeness of the injury, the amount of function preserved in patients will vary depending on injury extent. For complete injuries in which the spinal cord is fully severed, an absence of motor and sensory function in S4–S5 segments will occur (Sun et al., 2016). On the contrary, individuals who experience an incomplete SCI retain some motor and/or sensory function below the level of the injury.

TREATMENT

Steroids

In the early 1990s, it was thought that steroids played a role in acute SCI through their neuroprotective mechanism as an anti-inflammatory treatment modality. This early literature was based on animal models that led to the development of the National Acute Spinal Cord Injury Studies (NASCIS). The NASCIS I trial was a randomized controlled trial that compared two different doses of methylprednisolone (i.e., high-dose vs. low-dose). Upon conclusion of the trial, there was no significant difference in the neurological recovery at 6 weeks, 6 months, or 1 year following injury.

About 8 years later, the NASCIS II trial was conducted. This was a multicenter randomized, double-blind, placebo-controlled trial with patients who were treated approximately 14 hr following SCI. Patients were randomized to receive methylprednisolone, naloxone, or placebo to determine whether or not neurological improvement would occur. Methylprednisolone was dosed as a 30-mg/kg bolus infused over 1 hr followed by a maintenance dose of 5.4 mg/kg/hr for 23 hr. The conclusion derived from NASCIS II was that patients treated with methylprednisolone within 8 hr of injury had greater improvements in motor and touch function (Bracken et al., 1990; Braughler & Hall, 1983; Hall, 1992). It was this trial that led to methylprednisolone becoming the standard of care in SCIs despite controversy over the trial's validity and lack of reproducibility. Discussions regarding the trial's design, timing of drug administration, and statistical analyses continue to spark debate.

The NASCIS III study published 7 years later revealed no significant increase in motor or functional recovery after treatment with methylprednisolone. Like NASCIS II, NASCIS III was a prospective randomized study that consisted of three treatment arms. The design was created to test the effects of methylprednisolone (30-mg/kg bolus followed by 5.4 mg/kg/hr) when administered over 48 hr in patients who were to be treated within 8 hr of their injury. The authors of the NASCIS III trial concluded that patients with acute SCI may receive methylprednisolone within 3 hr of the injury for a total of 24 hr of treatment (Bracken et al., 1997).

Steroids following acute SCI are not without their complications, and have been associated with the development of pneumonia and gastrointestinal bleeding. To date, methylprednisolone is not recommended for the treatment of acute SCI by the Congress of Neurological Surgeons and American Association of Neurological Surgeons guidelines (Hurlbert et al., 2013). Should the decision be made to administer methylprednisolone, it should be administered within the first 8 hr of injury at the dosing and timing reflected above in the NASCIS II trial (Fehlings, Wilson, et al., 2017).

Hemodynamics

Neurogenic shock is a common complication following spinal cord injuries. It occurs when there is a disruption of sympathetic tone, resulting in unopposed vagal tone. This disruption in sympathetic tone leads to reduced vascular resistance, reduced heart contractility, and bradycardia. There is an increased risk of neurogenic shock with injuries above T6. The higher the injury, the more severe and refractory the shock may be (Velmahos et al., 2003). Neurogenic shock occurs more frequently in those with complete spinal cord injuries compared with those with incomplete spinal cord injuries, but it is still a complication that should be expected and vigilantly monitored.

Patients who present with neurogenic shock have severe hypotension defined as a systolic blood pressure of 90 mmHg or less and bradycardia defined as a heart rate of less than 60 beats/min due to the inability to redirect blood flow from the periphery to the core circulation. These patients are warm and dry to the touch and flushed in the face due to reduced peripheral vascular resistance and blood pooling in the peripheries. The peak of hemodynamic instability in these patients typically occurs 3–5 days following the injury and will take 2–6 weeks to resolve (Lehmann, Lane, Piepmeier, & Batsford, 1987). The goal in these patients is to restore hemodynamic parameters and avoid severe hypotension and symptomatic bradycardia.

Fluids

As with other types of shock, fluid resuscitation is the first-line treatment to ensure euvolemia in SCI patients. Fluids increase circulating blood volume to enhance organ tissue perfusion, thereby preventing organ damage and further dysfunction. Patients in the emergency department should be given up to 2 L of an isotonic crystalloid such as lactated ringers or normal saline to enhance perfusion to the spinal cord to promote healing

at the site of injury. SCI patients are at increased risk of developing acute respiratory distress syndrome and pulmonary edema with fluid resuscitation, and therefore should not be administered more than 2 L of crystalloid fluids (Consortium for Spinal Cord Medicine, 2008).

Vasopressors

Vasopressors are often necessary for these patients to enhance systemic vascular resistance and prevent hypotension and/or bradycardia (if using norepinephrine) after fluid resuscitation. Norepinephrine works on the α-1 receptors in vascular smooth muscle to activate vasoconstriction and on the β-1 adrenergic receptors in the heart to activate inotropy and chronotropy, increasing contractility and heart rate. Currently, there is no recommended agent for neurogenic shock; however, norepinephrine is most commonly used due to its activity on both blood pressure and heart rate. Phenylephrine is a pure α-1 agonist that activates vasoconstriction on vascular smooth muscle, like norepinephrine, but lacks β-1 activity on the heart's contractility and heart rate. This makes phenylephrine more favorable for patients with an injury below T6 where there is less concern for bradycardia (Consortium for Spinal Cord Medicine, 2008).

In the emergency setting, vasopressors may be administered to patients in neurogenic shock through a peripheral line until central access is established. The American Association of Neurological Surgeons and the Congress of Neurological Surgeons Guidelines for the Management of Acute Cervical Spine and Spinal Cord Injuries recommend that vasopressors should be titrated to a maintenance of mean arterial blood pressure at 85–90 mmHg for the first 7 days following injury to improve spinal cord perfusion (Catapano et al., 2016).

Bradycardia

Bradycardia is a common complication in patients with high spinal cord injuries and can last longer than 7 days (Lehmann et al., 1987). It is important that a patient's heart rate is frequently monitored to prevent progression to cardiac asystole. Cardiac events are the primary cause of death in patients with high spinal cord injuries during the first year (Devivo, Kraus, & Lammertse, 1999). Bradycardia occurs due to disruption in the autonomic nervous system, but it may also be provoked when manipulating a patient's airway and positioning (Manogue, Hirsh, & Lloyd, 2017).

Atropine

Atropine is given to block vagal tone, which improves conduction and increases heart rate. This anticholinergic agent blocks acetylcholine at the parasympathetic nervous system, which reduces vagal tone that is enhanced in SCI patients. It is a first-line agent for treating bradycardia in this patient population, and should be readily available at the bedside to prevent acute episodes that may occur while manipulating the airway and during the first few days following injury. Atropine is administered rapidly as 0.5 to 1-mg intravenous push every 3–5 min as needed, with a maximum total dose of 3 mg (Kusumoto et al., 2019). Nurses should frequently monitor heart rate prior to and following administration.

Pacing

In patients who have symptomatic bradycardia refractory to atropine, cardiac pacemaker implantation may be necessary to control heart rate. The American Heart Association and the American College of Cardiology in conjunction with the Heart Rhythm Society recommend temporary cardiac pacing as a possible intervention for patients with symptomatic bradycardia refractory to medical management until a permanent pacemaker can be placed or until symptoms resolve, whichever comes first (Kusumoto et al., 2019). However, cardiac pacemakers have risks such as infection, malfunction, and risk of death during placement. Criteria for optimal timing and use of pacemakers are poorly defined.

MONITORING

Airway Protection

As with most trauma-related injuries, airway, breathing, and circulation are top priorities when it comes to the initial management of SCI patients. This particular type of injury does make airway management more complex and challenging, as the cervical spine should be kept in neutral alignment at all times to avoid further harm to the patient. Approximately 2%–12% of major trauma victims have a cervical spine injury and 7%–14% of these are not stable (Cranshaw & Nolan, 2006). In a substudy of the National Emergency X-Radiography Utilization Study (NEXUS), 2.4% of trauma patients had cervical spinal injuries, with the second cervical vertebra, C2, being the most common level of injury (Crosby, 2006).

Secondary to the complexity and potential risks when it comes to airway management in patients with cervical injuries, the presence of an anesthesia provider may be warranted in the hospital setting. A thorough airway assessment is critical, though challenging due to immobilization, so that an airway plan can be developed. Techniques for managing the airways in these patients vary based on the results of the airway assessment, but the list includes mask ventilation, direct laryngoscopy, indirect laryngoscopy, extraglottic airway, and surgical airway (Diedrich, Rose, & Brown, 2013). The airway of trauma patients should also be cleared of secretions and/or debris to optimize patency and reduce the risk of aspiration (Cranshaw & Nolan, 2006).

Skin Breakdown/Pressure Ulcers

Skin breakdown and pressure ulcer management is critical in patients with chronic SCI, as these patients are more susceptible to this complication and recurrence as well. The incidence of pressure ulcers in these patients is 25%–66%, with the risk being higher in those patients with higher-level injuries. Secondary to this, it is critical that those caring for these patients have an understanding as to the management. Both extrinsic and intrinsic factors that can be corrected should be identified as prevention is the best approach as most are avoidable (Kruger, Pires, Ngann, Sterling, & Rubayi, 2013).

Strategies in the prevention and treatment of pressure ulcers include pressure relief, debridement, wound management, nutrition, and identifying/addressing factors that could contribute to recurrence. For patients who present to the emergency department, it is important that a thorough skin assessment be completed and pressure relief measures implemented when indicated. Examples of these measures include, but are not limited to, the use of specialty beds, turning the patient, heel protectors, and the removal of any other external factors that could contribute to making a pressure ulcer worse. Home care needs should be assessed as well, so that risk factors can be identified and addressed to help with avoidance of recurrence. In order for pressure ulcers to be appropriately assessed, staged, and heal, debridement is a key measure. Following staging, treatment will be determined. Stage I and II pressure ulcers can be managed nonoperatively, whereas Stage III and IV ulcers are likely to require surgical intervention (Kruger et al., 2013).

VTE Prophylaxis

SCI patients have one of the highest rates of venous thromboembolism (VTE) when it comes to patients presenting with traumatic injuries. Patients with advanced age, paraplegia, thrombophilia, and concomitant lower-extremity fractures are at an even higher risk for developing VTE post-injury. Chemical VTE prophylaxis should be initiated within the first 72 hr once there is no clinical sign of bleeding or plans for operative intervention within the next 12 hr. VTE prophylaxis is typically safe to resume within 24 hr after surgical intervention (Christie, Thibault-Halman, & Casha, 2011; Fehlings, Tetreault, et al., 2017). Enoxaparin is the preferred agent in this patient population, with the exception of subcutaneous heparin being preferred in

patients with spinal drains in place (Rappold et al., 2021). Enoxaparin and heparin are both administered subcutaneously into either the left or right posterolateral abdominal wall, and should be rotated between each injection. Common prophylactic doses of enoxaparin consist of 30 mg every 12 hr or 0.5 mg/kg every 12 hr and 5,000 mg every 8 hr for heparin.

CONCLUSION

In summary, SCI is often a life-changing event for most if not all patients. The injury has the potential to be devastating for the patient, their family, and society as a whole given immediate and long-term sequelae. Being familiar with the initial management and the most common complications is important when it comes to the care of these patients. Of the utmost importance is preservation of the patient's cord function, which is why immobilization is a priority. The use of Advanced Trauma and Life Support protocols helps ensure other injuries are not missed while necessary interventions and treatments for SCI are discussed and initiated. Airway management, shock identification, and the controversial use of intravenous methylprednisolone are treatments that will occur early on in the patient's care, but preparing, assessing, and being proactive when it comes to long-term complications, such as pressure ulcers and VTE, are also key.

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