

## Review Article: Managing Spinal Cord Injury on Anesthesiologists' Perspective

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### Abstract

**Background:** Spinal cord injury (SCI) is trauma to the area of the vertebrae resulting in spinal cord lesions resulting in neurological disorders, depending on the location of the spinal nerve damage and the injured nerve tissue. The symptoms of SCI can range from pain and paralysis to incontinence. SCI due to trauma is estimated to occur in 30-40 per million population per year, and about 8,000-10,000 sufferers each year; generally, occurs in adolescents and young adults. Although the annual incidence of events is relatively low, the cost of care and rehabilitation for spinal cord injuries is very high, at around US \$ 53,000 / patient.

**Methods:** This study aims to provide an overview of how to manage spinal cord injury. This study reviewed various sources then reviewed as a literature review.

**Conclusion:** Treatment in the hospital includes all systems that may experience complications from SCI, starting from the respiratory, cardiovascular, urological, gastrointestinal, skin, to non-operative and operative reduction measures.

**Keywords:** Spinal Cord Injury; Emergency; Management

### Introduction

Spinal cord injury (SCI) is a life-changing neurological condition with substantial socioeconomic implications for patients and their caregivers. Recent advances in the medical management of SCI have significantly improved the diagnosis, stabilization, survival rate and well-being of SCI patients. However, there have been small advances in treatment options to improve the neurological outcomes of SCI patients. This additional success primarily reflects the pathophysiological complexity of SCI and the variety of biochemical and physiological changes that occur in injured spinal cord [1].

SCI was first recorded in the history of medical science around 1700 BC on the Edwin Smith papyrus. The most common causes of SCI were traffic accidents (50%), falls (25%), and sports-related injuries (10%); and the rest is due to work accidents. SCI due to trauma is estimated to occur in 30-40 per million population per year, and about 8,000-10,000 sufferers each year; generally, occurs in adolescents and young adults. Although the annual incidence of events is relatively low, the cost of care and rehabilitation for spinal cord injuries is very high, at around US \$ 53,000 / patient [1].

The mortality rate is estimated to be 48% in the first 24 hours. About 80% died on the spot from cervical spine injury with the greatest risk of trauma, with the most frequent levels at C5, followed by C4, C6, then T12, L1, and T10. Based on the disability that occurred, 52% of cases had paraplegia and 47% had tetraplegia [1].

SCI usually occurs as a result of a sudden traumatic impact on the spine resulting in a fracture or spinal dislocation. The initial mechanical force that is sent to the spinal cord at the time of injury is known as a primary injury in which "displaced bone fragments, disc material, and / or ligaments are bruised or torn into the spinal cord tissue". In particular, most injuries do not completely sever the spinal cord. Four main characteristic mechanisms of primary injury have been identified which include: (1) impact plus continuous compression; (2) Self impact with transient compression; (3) Distraction; (4) laceration / transection [2-6]. The most common form of primary injury is impact plus persistent compression, which usually occurs through a fractured fracture with a bone fragment pressing on the spinal cord or through a fracture dislocation injury. Impact alone with transient compression is observed less frequently but occurs most frequently in hyperextension injuries. A nuisance injury occurs when two adjacent vertebrae are pulled causing the spine to stretch and tear in the axial plane. Lastly, laceration and transection injuries can occur via missile injury, severe dislocation, or dislocation of sharp bone fragments and can vary widely from minor injuries to complete transection. There are also striking differences between the results of the SCI in the military and civilian cases. Compared to civilian SCI, blast injury is a common cause of SCI on the battlefield which usually involves multiple segments of the spinal cord [7-11]. The SCI

blast also resulted in a higher severity score and was associated with a longer hospital stay. A study of American military personnel, who maintained SCI in combat zones from 2001 to 2009, showed increased severity and worse neurological recovery compared to civilian SCIs. In addition, lower lumbar fractures and lumbosacral dissociation were more common in combat injuries. Regardless of the form of the primary injury, these forces directly impair the up and down pathways in the spinal cord and disrupt blood vessels and cell membranes leading to spinal shock, systemic hypotension, vasospasm, ischemia, ionic imbalance, and accumulation of neurotransmitters. To date, the most effective clinical treatment to limit tissue damage after primary injury is early surgical decompression (<24 h post injury) of the injured spinal cord. Overall, the extent of the primary injury determines the severity and outcome of the SCI [12-16].

## Etiology

As many as 10% of patients with decreased consciousness who are sent to the Emergency Room (ER) due to traffic accidents always get cervical injuries, both injuries to the cervical spine, supporting tissues, and injuries to the cervical spine. Traffic accidents and falls are the cause of the majority of cervical fractures. Sub axis cervical trauma (C3-7) is more common than C1 and C2, and is a potential trauma that deserves a lot of attention. It is almost always thought that cervical trauma will occur in patients with a history of high-speed motor vehicle accidents, significant face and head trauma, neurological deficits, neck pain, and multiple trauma. According to the mechanism of injury, cervical injuries are divided into flexion, rotational flexion, extension, rotational extension, vertical compression, lateral flexion and mechanisms that are not yet clear [1].

## Anatomy of the Spinal Medulla

The spinal cord is a cylindrical organ that starts from the foramen magnum in the skull up to two thirds of the entire length of the vertebral canal, is continuous with the medulla oblongata in the brain, and the tip of the spinal cord is located at the lower boundary of the first lumbar vertebra in adults and the lower border of the vertebrae. third lumbar in children. The spinal medulla is surrounded by 3 layers of the meninges, including the dura mater, arachnoid mater, and pia mater. In addition, the cerebrospinal liquor (CSF) in the subarachnoid cavity also provides additional protection for the spinal cord [2].

The spinal cord consists of 31 segments, including 8 cervical segments, 12 thoracic segments, 5 lumbar segments, 5 sacral segments, and 1 coccygeal segment. The spinal nerves come out of each segment of the spinal cord (totaling 31 pairs of spinal nerves) and consist of motors or anterior roots (roots) and sensory or posterior root. The name of the spinal nerve is carried out based on the area where the nerve arises through the vertebral canal. Spinal nerves C1 to C7 arise from above the vertebral column C1-C7, while C8 between vertebral columns C7-T1. The other spinal nerves emerge from below the vertebral column in question [2].

The motor functions of the spinal nerves include, C1-C2 innervates the neck muscles, C3-C5 forms the phrenic nerve which supplies the diaphragm, C5-T1 supplies the muscles of the upper limb, thoracic segment supplies the thoracoabdominal muscles, and L2- S2 supplies the muscles of the lower extremities. Some important dermatomes that provide an overview for the sensory function of the spinal nerve, include C2-C3 for the posterior part of the head-neck, T4-5 for the areola mammae area, T10 for the umbilicus, parts of the upper limb: C5 (anterior shoulder), C6 (mother fingers), C7 (index and middle fingers), C8 (little finger), T1 (medial antebrachia), T2 (medial part of the brachial), T2 / T3 (axillary), lower limb parts: L1 (anterior and medial part of femoral), L2 (anterior part of the femoral), L3 (knee), L4 (medial malleolus), L5 (dorsum pedis and fingers 1-3), S1 (finger 4-5 and lateral malleolus), S3 / Cox (anus) [2].

Nerves from the lower spinal segment exit terminal to the conus medullaris and form the cauda equina. Ventral spinal nerve roots - make up the motor component of the spinal innervation with rootlets arising from the anterior horn of the spinal segment, sending motor impulses to muscles and joints, etc. Dorsal spinal nerve roots - make up the sensory component, with the dorsal ganglia sending sensory impulses via nerve rootlets to the posterior horn of spinal segments [14].

The spinal cord consists of two substances, namely the gray matter which is located internally and the white matter which is located externally. In general, the substantia alba consists of ascending (sensory) and descending (motor) tracts, while the gray matter can be divided into 10 laminae or 3 parts (anterior, posterior, and lateral horns) which are composed of nuclei that play a role in the action potential of neurons-neuron [2].

The sensory tracts (ascending tracts) of the spinal cord include, among other things, the lateral spinothalamic tract which carries the senses for pain and temperature, the anterior spinothalamic for touch (coarse / crude touch) and pressure, the dorsal column tract (posterior white column) for smooth touch (two -point discrimination), proprioceptive function and vibration, and other tracts such as, spinocerebellar (posterior and anterior), Cuneo cerebellar, spinotectal, spinoreticular, spinotectal, and spino-olivary [2].

The review of the tracts (especially regarding the level at which decussation occurs) in the white matter of the spinal cord will provide a comprehensive understanding of the clinical manifestations of patients with spinal cord trauma. Perceptions like soft touch, proprioceptive, and vibration (from the dorsal column tract) are not crossing (decussation) before they reach the medulla oblongata, whereas the lateral and anterior spinothalamic tracts intersect in 3 levels of the segment where it enters. On the other hand, the main motor tract (corticospinal) undergoes a decussation at the level of the medulla oblongata. This results in a lesion of

the corticospinal tract or dorsal column causing ipsilateral (for corticospinal) motor paralysis and loss of ipsilateral (for corticospinal) sense of touch, proprioceptive, and vibration of the lesion. Conversely, lesions on the tract that carry the perception of pain, temperature, pressure, and rough touch cause a loss of that perception in the contralateral region of the lesion [2].

Apart from the tract for sensory and motor functions, the spinal cord also plays a role in autonomic function. Sympathetic nerve function is influenced by the cranial nerve T1-L3 (thoracolumbar), while the parasympathetic nerve function is in S2-S4. Spinal cord lesions in the area concerned can cause autonomic nerve disorders according to the level of the lesion. One of the clinical correlations of impaired sympathetic nerve function due to lesions above T6 is neurogenic shock due to loss of sympathetic tone in arterial vessels, whereas micturition and erectile dysfunction are due to parasympathetic tone disorders [2].

The perfusion of the spinal cord consists of 1 anterior spinal artery and 2 posterior spinal arteries. The anterior spinal arteries provide blood supply to 2/3 of the anterior part of the spinal cord. The presence of lesions in these vessels causes dysfunction of the corticospinal tract, lateral spinothalamic, and autonomic pathways (paraplegia, loss of pain and temperature perception, and autonomic dysfunction). The posterior spinal arteries primarily provide blood supply to the dorsal column and posterior gray matter. Both arteries arise from the vertebral artery. Several radicular branches of the thoracic and abdominal aorta provide collateral hemorrhage to the spinal cord [6].

## Secondary damage Mechanism

Primary damage is a trigger or starting point for secondary damage. Secondary damage is caused, among other things, by neurogenic shock, vascular processes, such as bleeding and ischemia, excitotoxicity, calcium-mediated secondary lesions, electrolyte disturbances, damage due to immunological processes, apoptosis, disorders of the mitochondria, and other processes [4].

## Classification of Spinal Cord Injury (SCI)

Spinal cord injuries can be divided into complete and incomplete based on the presence / absence of sustained function under the Spinal Medullary Injury (SCI) lesion classified as complete and incomplete. Complete SCI is a total loss of sensation and voluntary motor function whereas incomplete is a mixture of loss of sensation and voluntary motor function. Another definition is that complete SCI is characterized by the absence of sensory and motor functions that come from below the level of the injury while incomplete SCI still has sensory and motor functions below the level of the injury [5].

In incomplete cord syndrome (about 50% of cases), the loss of sensory and / or motor function below the degree of injury is partial, and the type of neurological deficit varies. In complete spinal cord syndrome (the remaining 50%), motor and sensory functions below the level of the lesion are completely lost. The so-called spinal cord syndrome is not clinically complete, but neurophysiologically incomplete. Thus, even in a clinically complete SCI, there is an effect of residual brain on spinal cord function under the lesion. In one study, more than four out of five clinical complete syndromes could be classified neurophysiologically as incomplete, and, thus, in many patients, there may be a central nervous circuit over a spinal cord lesion that could potentially evolve into a clinically complete spinal cord syndrome [15].

## Clinical Manifestations

There are 5 main syndromes of incomplete spinal cord injury according to the American Spinal Cord Injury Association, namely: (1) Central cord syndrome; (2) Anterior cord syndromes; (3) Brown-Sequard syndromes; (4) Cauda equina syndrome; and (5) Conus medullary syndromes. A very rare incomplete syndrome is Posterior cord syndrome. Central cord syndrome results from incomplete injury to the central cervical segment of the spinal cord, most commonly the middle to lower cervical segment. Trauma that causes the ligamentum flavum (a strong ligament that connects the laminae of the vertebrae to protect the nerves and spinal cord and stabilizes the spina so that there is no excessive movement of the vertebrae) which eventually pinches the spinal cord from the posterior and / or as a result of compression by osteophytes or disc material from the anterior. Compression also causes impaired perfusion of the anterior spinal artery. On physical examination Central cord syndrome is usually limited to disorders of the neurologic system, consisting of combined upper motor neuron (UMN) and lower motor neuron (LMN) lesions supplying the upper limb and resulting in partial flaccid paralysis; and lesions predominantly on the UMN supplying the lower extremities resulting in spastic paralysis. Upper limb disorders are usually more severe than lower limb disorders, and mainly occur in the distal hand muscles. Sensory loss occurs to some degree, although sacral sensations are usually intact. The ability of anal contraction and sphincter tone and Babinski reflex should be checked [1].

Central cord syndrome (CCS) may result from bleeding into the central portion of the spinal cord, or as a result of disruption of axons in the lateral cornu at the level of injury but does not result in significant damage to the gray matter. CCS can also occur as a result of dislocation and compression fractures, particularly in individuals with congenital narrowing of the spinal canal. Compression pressure which is antero-posterior in direction results in more severe damage to the central area. The above injury mechanisms result in the most severe damage to the central spinal cord and less damage to the periphery of the spinal cord. Injury to this area results in damage to the lateral spinothalamic tract and corticospinal tract with characteristic symptoms. Both motor and sensory disturbances in CCS result from the distinctive lamination pattern of the corticospinal and spino-thalamic tracts in

the spinal cord. The lateral spinothalamic tract has a laminated arrangement in a somato-topic pattern, where the fibers originating from the sacral segment are most dorsolateral, followed by the fibers of the lumbar and thoracic segments, while the fibers of the cervical segments are the most ventromedial. Because CCS is caused by an injury to the central part, the cervical fibers are seriously injured while the sacral fibers are not injured [6].

Anterior spinal cord syndrome is an incomplete spinal cord syndrome that predominantly affects the anterior 2/3 of the spinal cord, typically resulting in motor paralysis below the level of the lesion and loss of pain and temperature at and below the level of the lesion. The patient presentation usually includes these two findings; However, there is variability depending on the part of the spinal cord that is affected. Other findings include back pain, or autonomic dysfunction such as hypotension, neurogenic bowel or bladder, and sexual dysfunction [9].

Brown Sequard syndrome (BSS) is a pattern of incomplete injury showing hemisection of the spinal cord resulting in weakness and paralysis on one side of the damage and loss of pain and temperature sensation on the opposite side. This depends on the location of the injury which can also involve the cervical or sympathetic thorax resulting in Horner's syndrome. Overall, the symptoms depend on which part of the spinal cord is affected. It is also known as hemisection of the spinal cord. As an incomplete spinal cord syndrome, the clinical features of BSS can range from mild to severe neurological deficits [10].

The cauda equina and conus medullaris syndromes have similar anatomy and clinical presentation. Therefore, for the purposes of this discussion, they will be grouped, and important differences will be highlighted. The conus medullaris is the terminal end of the spinal cord, which usually occurs at the vertebral level L1 in an average adult. Medullary cone syndrome (CMS) occurs when there is compressive damage to the spinal cord from T12-L2. The cauda equina is a group of nerves and nerve roots that originate from the distal end of the spinal cord, usually at the L1-L5 level and contains nerve axons that provide motor and sensory innervation to the feet, bladder, anus, and perineum. Cauda equina syndrome (CES) results from compression and impaired function of these nerves and can include the medullary or distal cones, and most commonly occurs when damage occurs to the L3-L5 nerve roots. Both syndromes are neurosurgical emergencies because they can present with back pain radiating to the legs, motor and sensory dysfunction in the lower extremities, bladder and / or bowel dysfunction, sexual dysfunction, and saddle anesthesia. CMS and CES also carry a high risk of litigation because late diagnosis and management can cause devastating lifelong damage [11].

SCI is characterized by the presence of tetraplegia or paraplegia, partial or complete and the grade or level depends on the area of the lesion or SCI. Tetraplegia or quadriplegia is a loss of sensory and motor function in the cervical segment of the spinal cord. Meanwhile, paraplegia is a disturbance of sensory and motor function in the lumbar thoracic segment and the sacrum. Respiratory failure and pulmonary disorders mainly occur in spinal cord injury to the cervical and thoracic segments. Lesions above C3 will result in complete paralysis of the respiratory muscles and diaphragm. In any lesion above C4, function of the diaphragm muscles, intercostal muscles and additional respiratory muscles may be lost. Lesions above C5 can affect diaphragm function and clearly this necessitates mechanical / artificial ventilation. C4-C5 lesions will present a variety of diaphragm disorders. Lesions of C6-T12 are usually characterized by an intact diaphragm that can provide 90% of the volume of the diaphragm expansion but the intercostal muscles cannot function properly to stabilize the ribs [6].

## Management

Treatment in the hospital includes all systems that may experience complications from SCI, starting from the respiratory, cardiovascular, urological, gastrointestinal, skin, to non-operative and operative reduction measures [8].

After traumatic SCI, the number of complications during the acute phase of hospitalization, depending on the time of surgery, with fewer complications if surgery is performed immediately after injury. It is proposed that patients with traumatic SCI should be operated on within 24 hours of injury to reduce complications. If surgery is not possible within 24 hours, efforts should be made to perform the operation earlier than 72 hours after the injury [16].

## Early Handling

The initial principle when accepting patients in the hospital emergency room is generally the same, namely being followed up according to trauma management; Advanced Trauma Life Support (ATLS), namely primary and secondary surveys. If at the time of admission to the hospital, immobilization of the spine has not been carried out, then the initial action that must be taken is immobilization. The difference with pre-hospital treatment is that a complete neurological examination must be performed (if the ABC vital signs have stabilized). A complete neurological examination was performed according to the 2011 revised International Standards for Neurological Classification of Spinal Cord Injury published by ASIA. At the time of initial neurologic examination, the level of the lesion can be determined, complete or incomplete lesions, and the presence or absence of a spinal shock phase. Radiological examination is then performed to see or rule out the possibility of SCI [8].

## Specific Treatment for Complications Of SCI

### Respiratory System

Respiratory tract complications are a major cause of morbidity and mortality in patients with SCI. Lesions directly related to respiratory function were lesions at the level of C5 and above, whereas lesions at the thoracic level only interfered with coughing and lesions in the lumbar did not affect at all. Patients with lesions above C5 should be intubated and mechanically ventilated because a gradual decrease in respiratory function may occur. Respiratory function must be closely monitored by checking oxygen saturation, vital capacity (VC) of the lungs, and periodic blood gas analysis. Sputum retention generally occurs within a few days of injury due to impaired effective cough function, leading to atelectasis and pneumonia. Chest physiotherapy, assisted cough and regular breathing exercises can prevent atelectasis and lung infections [8].

### Cardiovascular System

The major and crucial complication of the cardiovascular system due to SCI is neurogenic shock resulting from spinal shock. In general, neurogenic shock occurs in lesions above T6 due to loss of sympathetic tone. The loss of tone causes vasodilation and bradycardia leading to hypotension and shock. Shock in SCI must be distinguished between hypovolemic and neurogenic because if too much fluid is given in neurogenic shock, pulmonary edema will occur. Management of neurogenic shock includes IV fluids, vasopressors with alpha- and beta-adrenergic characteristics (such as norepinephrine, epinephrine, and dopamine), atropine to increase pulse, and avoidance of hypothermia due to vasodilation. Mean Arterial Pressure (MAP) should be targeted at above 70 mmHg, although several studies have shown MAP > 85 mmHg provides a better prognosis [8].

Thromboembolism is a complication that may also occur in paraplegia / tetraplegia patients due to SCI. The highest incidence of pulmonary embolism occurs at week 3 after injury and is the most common cause of death in SCI patients who survive trauma. If there are no contraindications such as capitis or thoracic trauma, antiembolism stockings are used during the first 2 weeks after trauma and anticoagulant use is started within 72 hours after trauma for 8-12 weeks (Low molecular weight heparin is better than warfarin) [8].

### Urological System

After the onset of severe SCI, the bladder is unable to pass urine spontaneously, and untreated patients may develop urinary retention which leads to urinary reflux and renal failure. As soon as the patient arrives at the hospital, a Foley catheter must be placed. The recovery time for the micturition reflex varies, generally 6-8 weeks, but can be up to 1 year (there is literature that says no return).

The intermittent catheterization program begins during the subacute phase, when fluid intake and output stabilize. This is done to prevent urinary tract infections. However, if the Foley catheter is removed too early, detrusor muscle damage and reflux may occur due to the high-pressure filling of the bladder [8].

Complications of SCI in the urinary tract are urinary tract infections (UTIs). Symptomatic UTIs accompanied by fever, leukocytosis, and pyuria should be treated with adequate antibiotics for 7-14 days, whereas asymptomatic infections do not need to be treated routinely. The application of sterile methods is important for prevention of UTIs [8].

### Gastrointestinal System

Patients with SCI should at least receive fluids intravenously for 48 hours because paralytic ileus is common with severe SCI. In this condition, a nasogastric tube is inserted (NGT) and nil per oral (NPO) is done until the bowel sounds return to normal. Total parenteral nutrition should be given. If the paralytic ileus lasts a long time, abdominal distension occurs and can cause disruption of diaphragmatic movement. Acute peptic ulcers can occur with bleeding or perforation, although they are not common, they are dangerous complications. Therefore, the administration of H2 receptor antagonists or proton pump inhibitors (PPIs) should be started immediately and given at least 3 weeks after trauma [8].

Evaluation of defecation function should be performed early and management started aggressively as soon as bowel sounds develop and bowel motility is normal. The height of the lesion determines the defecation function, among others, the lesion above T12 causes hyperreflexia and spasticity of the anal sphincter, while the lesion below it causes areflexia and flaccid of the sphincter. Bowel emptying method, with a combination of suppositories and anorectal stimulation, stimulates an evacuation pattern in the distal colon [8].

### Skin

Decubitus ulcers will always be a complication of SCI; therefore, prevention needs to be done early. In the acute phase, the patient is positioned on a left-right oblique every 2 hours to prevent ulcers. The use of a foam or water mattress can help reduce the pressure on the bony ridges, however, the patient's position must be changed every 2 hours [8].

### Use of Corticosteroids

The use of corticosteroids (especially high doses of methylprednisolone) is currently subject to controversy. A study conducted by NASCIS 2 (National Acute Spinal Cord Injury Study) shows that high doses of methylprednisolone (bolus 30 mg / kg in 15 minutes

then followed by 5.4 mg / kg in 23 hours) starting within 8 hours after closed SCI improve prognosis. neurological patient. The NAS-CIS 3 study later added that methylprednisolone therapy initiated within 3 hours after trauma should be continued for 24 hours, whereas those initiated between 3-8 hours post-trauma should be continued for 48 hours. The Consortium for Spinal Cord Medicine does not recommend the use of any neuroprotectants (steroids, ganglioside GM-1, gacyclidine, tirilazad and naloxone) because clinical evidence of improvement in the final prognosis has not been definitively obtained [8].

### Non-Operative and Operative Reduction Therapy

After the systemic parameters have stabilized, attention will be paid to stabilization and alignment of the spine and spinal cord. Any unstable SCI must be stabilized to prevent further damage from movement and also to release compression of the spinal cord. Patients with cervical SCI can be treated using skeletal traction to reduce dislocations, release compression of the spinal cord in burst fractures, and spinal splints. Skeletal traction to restore or maintain normal alignment is a fast and effective method. Some of the tools that can be used include spring-loaded tongs (Gardner-Wells), cones, and the University of Virginia. The load used depends on the dislocation or not. In a non-dislocated fracture, the weight is generally 3-5 kg, whereas in a dislocation a 4 kg increase every 30 minutes (up to a total of 25 kg) is used in a flexed neck position. The patient should be checked for neurologic status at any increase in load, and the traction load should be reduced as soon as possible in the event of deterioration in neurological status. In addition, halo traction can be used as an alternative means of skeletal traction [8].

### Conflicts of Interest

There are no conflicts of interest and ethical proved needed in this article since it is a literature review.

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