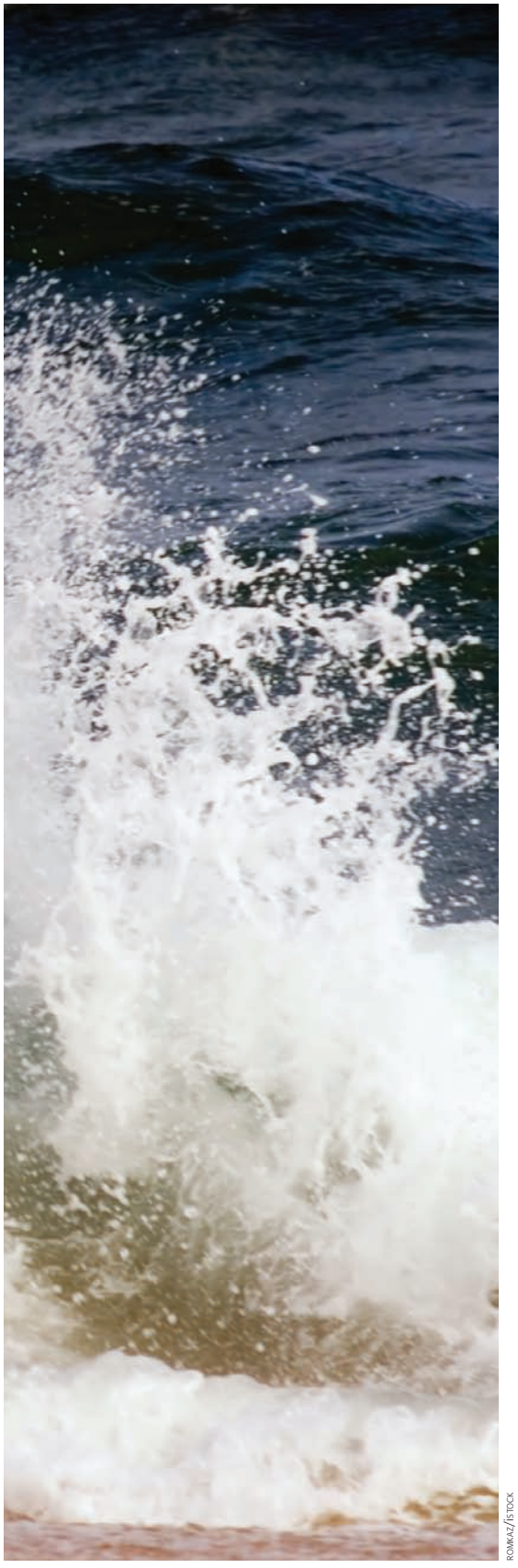

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Caring for patients with a traumatic spinal cord injury

BY TERESA E. HILLS, MSN, RN, ACNP-BC, CNRN

Abstract: The physical, emotional, and financial impact of a traumatic spinal cord injury (TSCI) can be devastating. This article discusses the pathophysiology of TSCI, medical and surgical management during the acute and subacute phases of injury, and nursing care for patients with TSCI.

Keywords: AIS, ASIA Impairment Scale, dermatomes, neurogenic shock, SCI, spinal cord injury, spinal shock, traumatic spinal cord injury, TSCI

NC is a healthy 17-year-old White male with no significant health history who presents to the ED following a surfing crash in which he was thrown head-first into the sand. The patient was pulled from the water unconscious and 911 was called. He quickly regained consciousness with shallow respirations and a palpable but slow pulse. He was unable to move his arms and legs on command despite trying. Suspecting a spinal cord injury, a bystander with a healthcare background initiated spinal stabilization until emergency medical services (EMS) arrived.

TRAUMATIC SPINAL cord injury (TSCI) often results in severe and permanent disability. Because victims experience life-altering changes to their health and quality of life, the physical, emotional, and financial impact can be devastating. According to the National Spinal Cord Injury Statistical Center, an estimated 249,000 to 363,000 people in the US are living with a spinal cord injury (SCI) and approximately 17,730 new cases of SCI are reported in the US annually.[1,2] The World Health Organization estimates that 90% of SCI cases have a traumatic etiology with the remainder caused by disease or degeneration.[3]

Advances in medical management and therapies have significantly improved the survival of patients with TSCI.[4] For patients experiencing TSCI in the 1970s, the average length of stay in an acute care hospital was 24 days. Today, this has decreased to 11 days. Days in an acute rehabilitation facility have also decreased, from 98 days to 31 days.[1]

Despite these advances, however, the achievement of significant improvements in neurologic recovery after TSCI has been limited; most patients continue to face permanent disability and lifelong challenges, including a

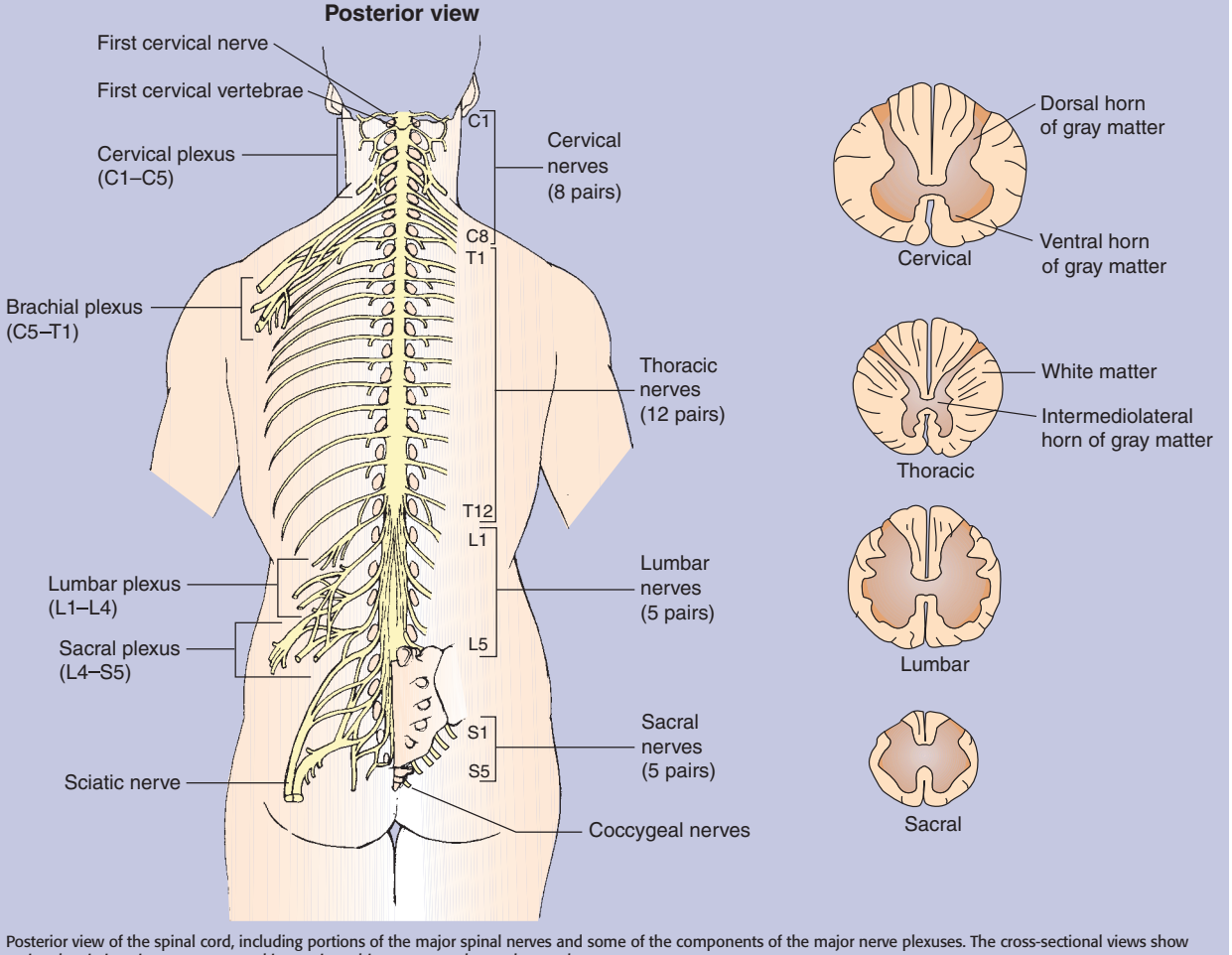
Anatomy of the spinal cord[33]

As shown in the posterior view below, the spinal cord is found in the upper two-thirds of the spinal canal of the vertebral column. It extends from the foramen magnum at the base of the skull to a cone-shaped termination, the conus medullaris, usually located at the level of the first or second lumbar vertebra (L1 or L2) in the adult. The dorsal and ventral roots of the more caudal portions of the cord elongate during development and angle downward from the cord, forming what is called the cauda equina (from the Latin for "horse's tail"). The filum terminale, which is composed of nonneural tissues and the pia mater, continues caudally and attaches to the second sacral vertebra (S2).

As shown in the cross sections below at right, the spinal cord is somewhat oval on transverse section, with the gray matter that forms the dorsal and ventral horns having the appearance of a butterfly or the letter "H." The central portion of the cord connects the dorsal and ventral horns. Called the intermediate gray matter, it surrounds the central canal.

In the thoracic area, the small, slender projections that emerge from the intermediate gray matter are called the intermediolateral columns of the horns. These columns contain the visceral output association neurons and the efferent neurons of the sympathetic nervous system.

The gray matter is proportional to the amount of tissue innervated by a given segment of the cord. Larger amounts of gray matter are present in the lower lumbar and upper sacral segments, which supply the lower extremities, and in the fifth cervical segment to the first thoracic segment, which supply the upper limbs. The white matter in the spinal cord also increases progressively toward the brain because more ascending fibers are added and the number of descending axons is greater.



Posterior view of the spinal cord, including portions of the major spinal nerves and some of the components of the major nerve plexuses. The cross-sectional views show regional variations in gray matter and increasing white matter as the cord ascends.

high risk of complications such as urinary tract infection (UTI) and deep vein thrombosis. This article discusses the pathophysiology of TSCI, medical and surgical management during the acute and subacute phases of injury, and nursing care for patients with TSCI. For a review of spinal anatomy, see *Anatomy of the spinal cord* and *Spinal cord and nerve roots*.

Time is spine

Advances in triage guidelines have reduced time wasted on implementing the workup and treatment of patients with TSCI and decreased

the time it takes to get these patients to an appropriate trauma center.[5] As with brain injury where "time is brain," with TSCI "time is spine." Preventing or minimizing secondary SCI after the primary insult is crucial for preserving as much function as possible.[5]

Primary SCI refers to the initial trauma to the cord. Examples include an axial load on the cervical spine from a dive into shallow water striking the head, or a hyperflexion injury from a front-end motor vehicle crash. The primary injury results from compression, contusion, and/or shearing injury to the spinal cord, causing damage at the cellular level and disrupting perfusion to the spinal cord that can lead to cell death.[5,6]

However, it is the *secondary SCI* that worsens functional outcomes in those with TSCI and should be the focus of the patient's care. The cascade of events that can occur in secondary SCI leads to permanent damage and disability.

Secondary injuries can be divided into four categories.

- acute: within the first 48 hours of injury. Hemorrhage, edema, ischemia, inflammatory changes, and release of cytotoxins at the cellular level increase the risk of necrosis and/or cell death, which can lead to further demyelination and loss of neural circuits.
- subacute: within 2 to 14 days after injury. In this phase, continued edema, inflammatory changes, and hypoperfusion to the spinal cord cause further ischemic changes to the spinal cord.
- intermediate: from 14 days to 6 months after injury.
- chronic: more than 6 months after injury. In the intermediate and chronic phases, axons continue to degenerate and scar tissue formation inhibits cellular regeneration.[5,6]

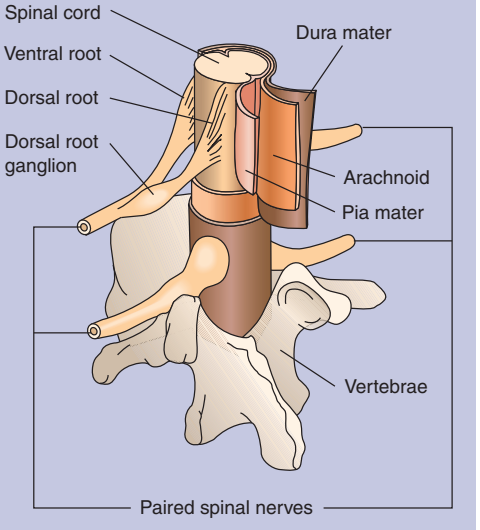
SCIs can also be categorized as complete or incomplete. *Complete SCI* signifies no sensation or movement below the level of injury, including the sacral segments.[6] An *incomplete SCI* is defined by the presence of "sacral sparing," which is sensation in the perineal area. Signs and symptoms of an incomplete SCI vary depending on the severity of injury to the spinal cord and the degree of neurologic dysfunction caused by the injury.[6]

Most cases of TSCI result from injury to the vertebral column. One or more vertebral fractures, dislocation or subluxation of the vertebral column at any level, disk herniations, or ligamentous injury/disruption are common causes.[6]

Fractures that involve both the anterior and posterior elements are considered unstable in most cases and increase the risk of TSCI. People with underlying degenerative spine disease such as spinal stenosis, spondylosis, or spinal arthropathies are also at increased risk of TSCI.[6]

Spinal cord and nerve roots[33]

The spinal cord and the dorsal and ventral roots are covered by a connective tissue sheath, the pia mater, which also contains the blood vessels that supply the white and gray matter of the cord. On the lateral sides of the spinal cord, extensions of the pia mater, the denticulate ligaments, attach the sides of the spinal cord to the bony walls of the spinal canal. Thus, the cord is suspended by both the denticulate ligaments and the segmental nerves. A fat- and vessel-filled epidural space intervenes between the spinal dura mater and the inner wall of the spinal canal.



Injuries to the cervical region of the spinal cord account for about 50% of all TSCI.[6] In cervical TSCI, bilateral upper and lower extremities can be affected, causing quadriplegia (also called tetraplegia) or quadriparesis (also called tetraparesis). Injuries to the thoracic and lumbar spine affect the lower extremities only, causing paraplegia or paraparesis.[6]

Various types of SCI are associated with the location of the SCI.

- *Posterior (dorsal) cord syndrome* occurs from injury to bilateral dorsal columns, the corticospinal tracts, and descending autonomic tracts all the way down to the bladder control centers in the sacral area. Posterior cord syndrome may be characterized by the loss of proprioception (position sense) with the preservation of some degree of movement and sensation. It is not commonly seen in trauma. Nontraumatic causes such as spinal cord tumors, degenerative spine disease, and demyelinating diseases such as multiple sclerosis are the usual cause of this type of SCI.[7,8]
- *Anterior (ventral) cord syndrome* occurs when the anterior two-thirds of the spinal cord, not including the dorsal columns, are injured. This results in motor weakness, reflex changes, and loss of bilateral pain and temperature sensation below the level of injury.[7,8] Loss of bladder function can also be present. Anterior cord syndrome is characterized by preservation of proprioception

but may entail loss of other functions. One way to assess for it is to determine if the patient can identify whether a finger or toe (depending on the level of spine injury) is up or down with eyes closed. This type of injury often results from trauma but can also be associated with injuries to the anterior spinal arteries that cause ischemia to the anterior portion of the spinal cord.[7,8]

• *Brown-Sequard syndrome* is caused by a hemisection of the spinal cord and is most commonly associated with penetrating trauma affecting the dorsal column, corticospinal tracts, and spinothalamic tracts on the side of injury to the cord. Patients will present with motor weakness or paralysis, loss of vibration sense, loss of proprioception ipsilateral to the level of injury, and loss of pain and temperature sensation on the contralateral side.[8] In other words, the side that can move cannot feel, and the side that can feel cannot move. The loss of motor function occurs on the side where the injury occurred. Traumatic causes include knife and bullet wounds. Nontraumatic causes include spinal cord tumors, cord infarctions, infection, and disk herniations.

• *Central cord syndrome* is the most common type of TSCI and very commonly seen in older adults. It often occurs from a fall in which the person strikes the chin or a low-speed trauma that causes hyperextension of the neck in someone who has underlying cervical spondylosis and stenosis. This results in injury to the central region of the spinal cord, causing damage to the medial aspect of the corticospinal tracts or the anterior horn of the gray matter. In many cases, the vertebral column remains intact. Patients present with bilateral upper extremity motor weakness. Their lower extremities may be weak or have normal motor function, and bladder dysfunction may be present. Associated sensory abnormalities can occur to varying degrees.[5,7,8]

Examples of primary SCI include an axial load on the cervical spine from a dive into shallow water striking the head.

• *Conus medullaris syndrome* occurs in patients with injury at the L2 level caused by spinal fractures, disk herniations, and tumors. The conus medullaris is compromised, causing sphincter dysfunction with flaccid paralysis of the bladder and rectum, saddle anesthesia, and impotence.[7]

• *Cauda equina syndrome* is caused by the loss of function of two or more of the 18 nerve roots that make up the cauda equina. Deficits can effect one or both legs.[7] Signs and symptoms include radiating pain into one or both legs, weakness of dorsiflexion or plantar flexion, bladder and rectal sphincter paralysis, sexual dysfunction, and loss of sensation along the dermatomal pattern of the affected nerve root.[7] Cauda equina syndrome has many causes, the most common being disk herniations (traumatic and nontraumatic), epidural abscess, and spinal tumors.[7]

Other injuries to the spinal cord include ***spinal cord concussions,*** also known as cervical cord neurapraxia, which is often seen in injuries related to sports, especially football. Due to hyperextension, hyperflexion, or an axial load on the head, the spinal cord is injured resulting in temporary paresis or paralysis of one or more extremities with or without associated sensory abnormalities. Signs and symptoms are transient and usually resolve within minutes to 24 hours.[7,8]

Spinal cord injury without radiologic abnormality is more common in the pediatric population and most often involves the cervical spine. Because of advances in imaging, this diagnosis has become less common. MRI can now be used to identify injuries to neural elements that are not apparent on X-rays or computed tomography (CT).[7,9]

Neurogenic vs. spinal shock

Another cascade of events that can occur with SCI is neurogenic shock, which is often confused with spinal shock.

• *Neurogenic shock* is most commonly seen in SCIs at the level of T6 (the level of the sympathetic outflow tract) or higher. Resulting from a loss of sympathetic tone, neurogenic shock is a distributive type of shock characterized by hypotension, bradycardia, and warm skin caused by widespread vasodilation.[10,11] Uninhibited parasympathetic nervous system stimulation along the intact vagus nerve can also occur. Hypotension is caused by decreased sympathetic mediated arterial and venous vascular resistance, along with loss of preload and venous pooling. Because of this, bradycardia is often observed when the patient is hypoxic, being suctioned, having a bowel movement, or being turned.[11]Neurogenic shock generally lasts from a few days to a few weeks.

• *Spinal shock,* which describes the loss of sensation and motor function below the level of injury with flaccid paralysis, is not associated with circulatory alterations from loss of sympathetic tone.[11] This can occur at any level of injury, is temporary (days to months), and can occur in conjunction with neurogenic shock at injuries to T6 or higher.[11] Spinal shock may not fully resolve for 6 to 12 months due to cord edema. The sign that spinal shock is beginning to resolve is the change from flaccid muscle tone below the injury to the development of muscle tone, reflex activity (typically abnormal), and muscle spasms/spasticity. The return of muscle tone alone does not necessarily predict functional recovery.

Initial assessment of injury

In patients with TSCI, worsening edema from secondary SCI can cause neurologic deterioration to ascend to a level above the level of injury. Because ascension of injury may be first detected on clinical exam, accurate spinal cord assessments are essential in the acute and subacute phases of TSCI.

The American Spinal Injury Association (ASIA) offers a standardized neurologic scale, the ASIA Impairment Scale (AIS), to classify the severity of SCI. Scoring ranges from level A (a complete SCI with no motor or sensory function below the injury) to level E, which is normal (see *Using the AIS to grade motor and sensory impairment*).[12]

The examiner performs a thorough motor and sensory assessment including sacral nerve root function and rectal tone.[12] This exam gives a quick view of the level and severity of injury. Understanding dermatomes, or areas of skin controlled by a specific spinal nerve, will make spinal cord assessments more accurate (see *Tracking the body's dermatomes*).

A complete spinal cord assessment is obtained using the ASIA International Standards for Neurological Classification of Spinal Cord Injury (ISNCSCI). A worksheet for the 2019 ISNCSCI Revision is available at www.asia-spinalinjury.org.

The spinal cord assessment is scored by grading key muscles and sensory points. Muscle groups are graded on a scale of 0 to 5, with 5/5 being normal and 0/5 being total paralysis. Sensation is graded as present/absent and normal/abnormal.[12]

Following the ASIA scoring system, the examiner will be able to describe the patient's level and severity of injury. For example, a patient with a complete SCI at C5 will be described as having a "C5 ASIA A SCI."

Diagnostic studies

Diagnosing TSCI involves both clinical assessment and imaging. *Plain X-rays* can be a useful initial too for identifying fractures of the spine and for assessing spinal alignment.[8] Anterior/posterior and lateral views may identify fractures and level of compression associated with the fractures.[10] Lateral upright and supine X-rays are often obtained to assess for stability of the fractures when the patient is placed in a cervical collar or back brace.[8]

With the advances in technology and the safety in use of CT, however, CT has become the gold standard in the preliminary workup for TSCI.[8] Depending on the mechanism of injury, CT of the cervical spine is often part of a routine trauma workup. CTs of the thoracic spine and lumbar spine are based on clinical assessment and the patient's signs and symptoms. CT of the chest, abdomen, and pelvis also capture the thoracic and lumbar spine and are sufficient for evaluating fractures at these levels.

Although CT aids the accurate radiologic assessment of bone fractures, it is not sufficient for visualizing the spinal cord, vertebral disks, nerve roots, and ligaments. Consequently, MRI may be warranted to investigate possible injury to any of these neural elements as it provides a more sensitive and detailed view of these structures.[8,13] Possible contraindications to MRI include the presence of certain incompatible implants such as pacemakers or spinal cord stimulators and the patient's inability to remain still during the scan because of pain or claustrophobia. In addition, some patients are too large to fit in the scanner. When MRI is contraindicated, myelography is an alternative study that assesses, to a lesser degree, injury to neural elements; this may be helpful in deciding on the need for surgical intervention.[13]

Using the AIS to grade motor and sensory impairment[12]

In the following grading system, the AIS is used to document the degree of sensation during light touch and a pin prick at multiple points and tests key motions on both sides of the body.

Grade A: Complete. No sensory or motor function is preserved in the sacral segments S4-S5.

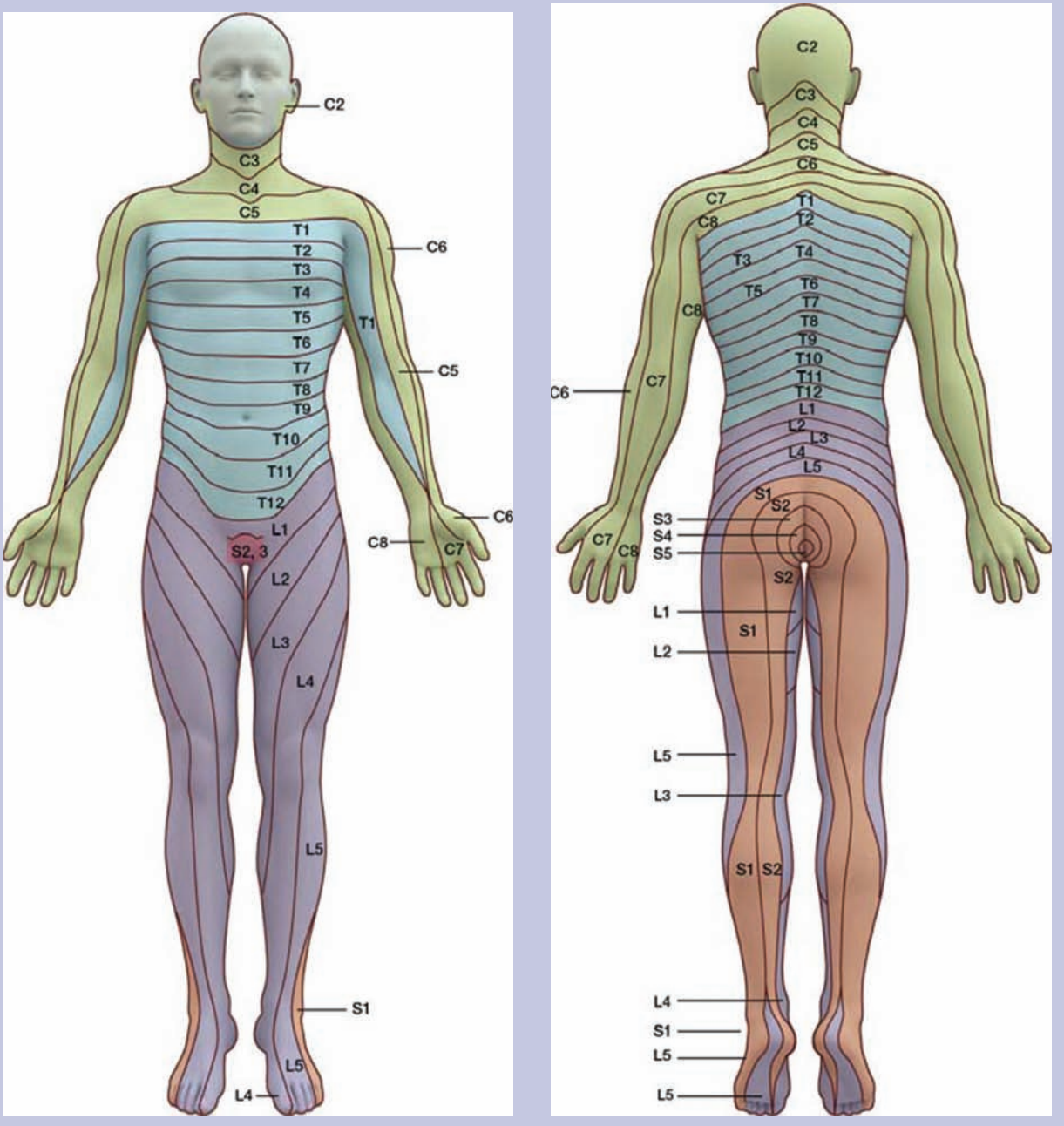
Grade B: Sensory incomplete. Sensory but no motor function below the neurologic level and includes the sacral segments S4-S5.

Grade C: Motor incomplete. Motor function is preserved below the neurologic level and more than half of key muscles below the neurologic level have a muscle grade less than 3.

Grade D: Motor incomplete. Motor incomplete status as defined above, with at least half of key muscle below the neurologic level having a muscle grade of 3 or more.

Grade E: Normal. No motor or sensory impairment.

Tracking the body's dermatomes



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NC's case progression

When EMS arrive at the scene, NC is in respiratory distress with minimal chest rise/fall. His SpO_2 is 88% on room air, his BP is 84/45 mm Hg, and his heart rate is 52. He appears to have some weak proximal upper extremity motor function bilaterally but no lower extremity motor function bilaterally on gross exam.

Spinal stabilization is initiated at the scene. NC is endotracheally intubated and given 100% oxygen, placed in a rigid cervical collar with lateral head supports on a backboard, and transferred to the ambulance, where he receives 1 L of 0.9% sodium chloride solution I.V.

After further workup in the ED, the patient is found to have a fracture-dislocation at C5-6 with possible associated SCI.

Medical and nursing management

Immediately after a patient like NC experiences TSCI, interventions to reduce the risk of worsening neurologic function are vital and should

start in the prehospital setting. At the scene, the first responders are responsible for identifying patients who may have an SCI and following prehospital guidelines to safely stabilize the patient. This includes taking actions to assess, maintain, and protect the airway; preventing hypoxemia, bradycardia, and hypotension; and quickly transferring the patient to the ED at a trauma center.[2,14]

Upon arrival in the ED, early medical management should be initiated incorporating a complete Advanced Trauma Life Support assessment with the focus on protecting the airway and preventing hypoxemia, bradycardia, and hypotension.[15] If the patient was not endotracheally intubated at the scene, clinicians should consider emergent airway protection with rapid sequence intubation while maintaining spinal stabilization, especially in patients with suspected high cervical SCI.

Intubation can be challenging in a patient with a suspected cervical spine injury. Fiberoptic intubation can decrease the risk of hyperextension of the neck, making the procedure safer.[2]

Oxygen saturation should be maintained above 93%, Pa_{O_2} above 80 mm Hg, and mean arterial pressure (MAP) above 85 mm Hg.[15-19] To maintain MAP, I.V. fluid resuscitation is initiated first. If this fails to achieve the MAP goal, a vasopressor such as norepinephrine or phenylephrine may be prescsibed.[2,15-19]

A complete assessment of motor and sensory function can be obtained according to the ASIA ISNCSCI guidelines.[12] Sending specimens for basic lab tests, monitoring arterial blood gas results, obtaining a chest X-ray, and performing an ECG should all be part of the initial workup.

The patient will undergo CT when stabilized. CT of the head, cervical spine, and chest/abdomen/pelvis are commonly part of the initial trauma workup, depending on the mechanism of injury and the patient's symptoms. If vertebral fractures, dislocations, or subluxation are identified, urgent neurosurgical consultation is vital.

Interventions to reduce the risk of worsening neurologic function should start in the prehospital setting.

Once hemodynamically stable, the patient should be transferred to the ICU as soon as possible.

The use of steroids in the medical management of acute TSCI has been well studied and is not recommended. The National Acute Spinal Cord Injury Studies (NASCIS I, II, and III) were randomized, prospective studies that looked at how effective methylprednisolone is in protecting the spinal cord after injury. Timing, dosing, and complications were all studied closely. The NASCIS II trial showed a very slight improvement in motor score in a subset of test subjects when steroids were given within 8 hours of injury. Because of many irregularities in the study, however, these results were subsequently discredited.[20] The results of NASCIS I and III showed that use of methylprednisolone increases the risk of sepsis, respiratory failure, and death.[21,22]

Nursing considerations in the ICU

Critical care management of the patient with TSCI requires a multisystems approach with the goal of preventing or minimizing secondary SCI. Following hospital-specific guidelines in the management of TSCI will help the care team achieve this goal.[15] Evidence-based institutional guidelines for management of TSCI enable the team to act fast to manage complications and achieve the best possible outcome.

The clinical nurse is likely to be the first clinician to detect respiratory and/or hemodynamic changes requiring interventions. Primary nursing responsibilities are to protect the airway; maintain spinal stabilization; prevent hypoxemia, bradycardia, and hypotension; and prepare the patient for surgical decompression and stabilization if indicated. Frequent spinal cord assessments (for example, at least every 2 hours or per hospital protocols) should be conducted for at least the first 72 hours postinjury. The nurse's spinal cord assessment is crucial for detecting subtle neurologic changes that may warrant changes in treatment or surgery.[15]

The nurse should anticipate that patients with TSCI may be angry and depressed, and many will also be in denial about their diagnosis. Providing care with patience and compassion is imperative. The nurse should be prepared to review the daily plan of care with the patient; explain procedures, tests, and interventions as they are occurring; and answer any questions the patient or family may have.

Nursing care and assessment during the acute and subacute phases of TSCI focus on the following.

• **Providing respiratory care.** Once the patient is endotracheally intubated, maintain Pao_2 above 80 mm Hg or oxygen saturations above 93%.[15] Closely monitor patients with high cervical SCI (C1–C5) for impaired diaphragmatic breathing, respiratory depression, and hypercapnia. Forced vital capacity (the volume delivered during an expiration made as forcefully and completely as possible starting from a full inspiration) and negative inspiratory force (also known as the maximal inspiratory pressure) provide information on inspiratory muscle strength. The patient is instructed to maximally inhale against a closed valve (usually starting from resting tidal volume), and the force/pressure generated at the mouth is recorded.[23]

The peak expiratory flow (also known as a peak flow or peak flow rate) is the maximal rate that a person can exhale during a short maximal expiratory effort after a full inspiration. This should be measured at least each shift.[2,15] If the negative inspiratory force is less than 30 cm H_2O or the forced vital capacity is less than 15 to 20 mL/kg, the patient should be admitted to an ICU in anticipation of the need for mechanical ventilation.[24]

Maintain appropriate ventilator settings to optimize ventilation and oxygenation. Some studies report earlier extubation in those patients with higher tidal volume settings. There is evidence to suggest that higher tidal volumes may improve the production of surfactant, prevent airway collapse, promote recruitment of alveoli, and be better tolerated by the patient.[19]

Performing frequent and effective pulmonary hygiene, especially in patients who initially do not require intubation, is an important intervention of the respiratory therapist and clinical nurse as these patients are at very high risk of respiratory failure or pneumonia.

Initial treatment with aggressive fluid resuscitation followed by vasopressors may be needed to achieve BP goals.

• **Maintaining hemodynamic stability.** Hypotension with or without associated neurogenic shock must be prevented at all costs. Initial treatment with aggressive fluid resuscitation followed by vasopressors may be needed to achieve BP goals. Many studies have found improvement in outcome when maintaining MAP above 85 mm Hg for the first 5 to 7 days. For example, a retrospective study by Dakson and colleagues showed patients who maintained MAP below 85 mm Hg for at least 2 consecutive hours during the first 5 days after injury were 11 times less likely to have an improvement in the AIS grade when compared with those patients whose MAPs remained above 85 mm Hg.[17]

The Consortium for Spinal Cord Medicine recommends the type of vasopressor based on the level of injury. In TSCI at T6 or higher where a loss of sympathetic tone increases the risk of bradycardia, vasopressors with both alpha and beta-adrenergic properties (such as norepinephrine or dopamine) are preferred.[25] For lower thoracic TSCI where hypotension may be the result of vasodilation, a pure alpha-adrenergic agent such as phenylephrine is appropriate. Of note, in ASIA grade A penetrating TSCI, maintaining MAP above 65 mm Hg may be acceptable because recovery is less likely.[19]

• **Preparing the patient for surgical intervention.** Research into the optimal timing of surgical intervention in acute TSCI (within 24 hours versus after 24 hours postinjury) has yielded conflicting results. However, newer studies are showing that early surgical decompression of the spinal cord reduces the risk of secondary SCI, decreases the risk of injury to the neural elements, and potentially improves outcomes.[2,16,26]

The Surgical Trial in Acute Spinal Cord Injury Study looked at outcomes when comparing extradural surgical decompression within 24 hours of injury versus 48 hours or more. It found that 20% of test subjects with cervical SCI had a 2 grade or greater improvement in their AIS score compared with 9% in those whose surgery was delayed.[27]

AIS score and severity of injury (complete versus incomplete) also play an important role in the timing of surgery. For injuries to the thoracolumbar spine, a decision to operate can be based on the Thoracolumbar Injury Classification and Severity Score.[27]

• **Managing pain.** Opioids, nonsteroidal anti-inflammatory drugs, acetaminophen, and muscle relaxants are often used to treat pain from vertebral column fractures and/or injury to the spinal cord and associated nerve roots. However, many

patients with acute TSCI experience various extremes of neuropathic pain that do not respond to opioids or muscle relaxants. Medications such as gabapentin or pregabalin are preferable in these situations.[19,25,28] Use of muscle relaxants or placement of a baclofen pump has improved pain control in some.[28]

• **Preventing venous thromboembolism (VTE).** Approximately 40% of multisystem trauma patients with TSCI may develop a VTE within the first 12 weeks following injury.[19] All patients should have intermittent pneumatic compression during the acute phase of TSCI unless contraindicated.[15,19,29] The 2013 guidelines from the Congress of Neurological Surgeons and the American Association of Neurological Surgeons Joint Section on Disorders of the Spine and Peripheral Nerve support starting VTE prophylaxis with low-molecular-weight heparin within 72 hours of injury and continuing the regimen for 12 weeks.[29] In patients for whom pharmacologic VTE prophylaxis is contraindicated, placement of an inferior vena cava filter can be considered.[19,25,29]

• **Managing bowel and bladder function.** Depending on the location of TSCI and its involvement of the sacral nerve roots, patients with TSCI may experience neurogenic bowel and/or bladder. Many patients start a bowel and bladder training program while in the hospital or upon their arrival at a rehabilitation facility. This is integral to their overall recovery because approximately 20% of patients with TSCI who are readmitted to the hospital report UTI as the reason.[19]

Patients with T6 or higher TSCI are at risk for autonomic dysreflexia, a potentially life-threating event that is triggered by some irritant below the level of injury. The most common triggers are bladder distension, bowel impaction, UTIs, skin infections, pressure injuries, and restrictive clothing.[6,8,19] Initial signs and symptoms of autonomic dysreflexia are sudden onset of hypertension (typically 15 to 20 mm Hg higher than baseline BP), severe headache, diaphoresis, and flushed face.[6,8,19] Because BP can rise to very dangerous levels if the noxious cause is not rapidly identified and effectively addressed, treatment requires identifying the irritant and immediately eliminating it. For example, if an indwelling urinary catheter is kinked, fix the kink.

Nutritional interventions should start as soon as possible with a goal for the patient to be at full caloric intake by day 7.

• **Providing skin care.** Patients with TSCI face a high risk for pressure injuries due to their lack of protective sensation and limited mobility. Any comorbidities such as diabetes, malnutrition, or anemia increase the risk further.[30] Frequent repositioning, use of pressure-reducing support surfaces and products, daily skin care, and improvement in nutrition can help prevent pressure injuries.[30]

• **Ensuring adequate nutrition.** TSCI results in a hypermetabolic state, accelerated catabolism, and significant nitrogen losses. These deplete energy stores and lean muscle mass and decrease protein synthesis. Nutritional interventions should start as soon as possible with a goal for the patient to be at full caloric intake by day 7 of injury.[31]

• **Initiating rehabilitation.** Physical and occupational therapy should start as early as possible in the ICU to promote overall recovery and prepare patients for what is ahead when they transition to an acute SCI rehabilitation center.

NC's treatment regimen

NC undergoes a C4–C7 posterior decompressive laminectomy and fusion. Postoperatively he receives norepinephrine via I.V. infusion titrated to keep MAP above 85, and an infusion of 0.9% sodium chloride at 100 mL/h. He is orally intubated and receiving mechanical ventilation. He is also receiving I.V. fentanyl and propofol infusions. Tube feedings are started via gastric tube. An indwelling urinary catheter has been inserted. Physical and occupational therapy are on hold until NC's BP is more stable.

Future directions

Many research studies are underway to investigate pharmacologic, cell-based, and physiologic approaches to improving functional outcomes in patients with TSCI.[32] Although some of these trials show promise, no study has yet shown an overwhelming improvement in neurologic recovery after SCI. Given the number of clinical trials taking place worldwide, patients and caregivers have reason to hope that a breakthrough in treatment will occur soon. ■

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