Spinal Cord Injury: An Overview

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The World Health Organization defines spinal cord injury (SCI) as damage to the spinal cord, conus medullaris or cauda equina.[1] Damage to the spinal cord can result in loss of sensation and motor control to the limbs and trunk as well as loss of autonomic control. This in turn can result in abnormal bowel and bladder control, sexual function, breathing, blood pressure, heart rate and temperature control. The extent of these symptoms depends on the level at which the spinal cord injury occurred as well as the extent to which the spinal cord is damaged. *Tetraplegia* (previously known as quadriplegia) is the term used to describe a loss of sensation and motor control in all four limbs and the trunk. Tetraplegia usually results from a cervical SCI. *Paraplegia* is used to describe a loss of sensation and/or motor control in only the lower limbs and sometimes also the trunk. Paraplegia is commonly caused by an SCI in the thoracic or lumbar regions.

The estimated global incidence of SCI is 40 to 80 cases per million annually.[1] In Canada, the prevalence is approximately 1,298 cases per million.[1] Trauma causes include motor vehicle accidents, falls and violence. Non-traumatic SCIs may be caused by degenerative changes of the spine, neoplastic tumours, vascular insults, autoimmune disorders and infections. The incidence of traumatic SCI is higher in adult men, with at least a 2:1 ratio of male to female injuries.[1] The incidence of non-traumatic SCI is also higher in men. Traumatic SCI is most likely to occur in young adults (15 to 29 years) and the elderly (over 60 years), whereas non-traumatic SCI is more common in the elderly. Tetraplegia is slightly more common than paraplegia and accounts for 52% to 57% of SCI injuries.[2]

How is spinal cord injury classified?

The extent of SCI is described using the International Standards for Neurological Classification of Spinal Cord Injury published by the American Spinal Injury Association (ASIA). The classification is based on a systematic sensory and motor examination of neurological function. The neurological level of injury (NLI) is the lowest segment of the spinal cord with intact sensation and antigravity muscle strength, provided there is normal motor and sensory function above. A level of injury from C1 to T1 results in tetraplegia. A level of injury below T2 results in paraplegia.

The severity of the injury is described by the American Spinal Injury Association Impairment Scale (AIS), which includes five scales from A to E:

A = Complete: No sensory or motor function is preserved in the sacral segments S4–5.

B = Sensory Incomplete: Sensory, but no motor function is preserved below the neurological level and includes the sacral segments S4–5 (light touch or pin prick at S4–5 or deep anal pressure) AND no motor function is preserved more than three levels below the motor level on either side of the body.

C = Motor Incomplete: Motor function is preserved at the most caudal sacral segments for voluntary anal contraction (VAC) OR the patient meets the criteria for sensory incomplete status (sensory function preserved at the most caudal sacral segments [S4–S5] by light touch, pin prick or deep anal pressure), and has some sparing of motor function more than three levels below the ipsilateral motor level on either side of the body. For AIS C, less than half of key muscle functions below the single NLI have a muscle grade ≥ 3.

D = Motor Incomplete: Motor incomplete status as defined above, with at least half (or more) of key muscle functions below the single NLI having a muscle grade ≥ 3.

E = Normal: If sensation and motor function as tested with the International Standards for Neurological Classification of Spinal Cord Injury (ISNCSCI) are graded as normal in all segments, and the patient had prior deficits, then the AIS grade is E.

Complications Following a Spinal Cord Injury

In additional to motor and sensory impairments, SCI also caus-

es many other impairments and chronic complications, including psychosocial consequences such as adjustment, depression, vocation, and caregiver burden issues, can be significant and complex. For the purpose of this review, we focus on physiological consequences, which include but are not limited to the following.

Neurogenic Bladder

The Impact

SCI above the sacral segments results in an upper motor neuron (UMN) bladder. In a UMN bladder, there is detrusor muscle hyperactivity, with patients presenting with urgency, frequency and incontinence. Intravesical pressures may be elevated. Injuries involving the sacral segments from S2 to S4 cause a lower motor neuron (LMN) bladder. In an LMN bladder, the bladder is areflexic and atonic—so the person cannot voluntarily empty the bladder—and it is prone to leaking, but intravescial pressure remains low.

The Implications

Urinary tract infections are the most common complication in people with SCIs and are often heralded by increased spasticity, fever, incontinence, autonomic dysreflexia and vague abdominal discomfort. Other urinary complications include vesicoureteral reflux, renal and bladder calculi, hydronephrosis and chronic renal failure.

The Interventions

Management of neurogenic bladder aims to drain the bladder sufficiently, to ensure continence and to maintain normal intravesical pressure to protect the upper urinary tract. Intermittent catheterization is commonly used for bladder management, but where intermittent catheterization is not feasible, some may choose to have an indwelling urethral or suprapubic catheter.

Neurogenic Bowel

The Impact

SCI impairs bowel function and can result in poor colonic motility, delayed transit time, chronic constipation and fecal incontinence. Similar to neurogenic bladder, SCI above the sacral segments results in a UMN bowel pattern, while lesions at the sacral segments lead to an LMN bowel pattern. In a person with a UMN bowel, voluntary defecation cannot be initiated, and the anal sphincter may be spastic, leading to stool retention. Intrinsic and reflex-mediated colonic peristalsis are intact, however, which allows stool to move through the colon and rectum reflexively in response to distension. In a person with an LMN bowel, voluntary defecation and the reflexes are impaired. This leads to an even slower colonic transit time. The anal sphincter is often atonic and prone to leakage of

stool, so incontinence may be a problem.

The Implications

Bowel continence plays a large role in a person's ability to return to former social roles and activities. Neurogenic bowel may also cause chronic constipation, hemorrhoids, rectal prolapse, acute ileus or bowel obstruction.

The Interventions

Management of a UMN bowel involves using stool-softening laxatives, colonic stimulants to assist in stool propulsion, and a suppository with digital stimulation to trigger the colonic reflexes to stimulate evacuation of stool. Management of an LMN bowel involves using a bulking agent to achieve a stool consistency that allows for manual disimpaction from the rectum. Adequate fibre and fluid intake also assist with developing bulky, formed stool. A bowel program is individualized to each patient but generally occurs at the same time every day and should take less than one hour to complete. The goals of a bowel program are to efficiently evacuate the colon to prevent incontinence, constipation and complications such as hemorrhoids.

Spasticity

The Impact

Spasticity is a common complication of SCI characterized by hyperreflexia, velocity-dependent increased resistance to passive stretch and involuntary muscle contractions or spasms. Spasticity occurs in the muscles below the level of injury in SCIs above the cauda equina.

The Implications

Spasticity may be painful and interfere with activities of daily living, ambulating, positioning and transfers. However, spasticity may also be helpful for ambulation and transfers by providing more rigid support from the lower limbs. Worsening spasticity is often related to other complications, such as a urinary tract infection—hence the importance to rule this out.

The Interventions

Non-pharmacological treatments for spasticity include proper positioning, stretching and serial casting. Pharmacological treatments include oral baclofen and other anti-spasticity medications. Focal chemo-denervation with botulinum toxin type A is helpful when specific muscles can be targeted. Intrathecal baclofen is another option for those with diffuse, severe spasticity that is not well managed with other conservative treatment options.

Bone Health

The Impact

An imbalance between bone resorption and formation occurs following SCI, leading to increased bone resorption in the bones below the level of injury. This may result in symptomatic hypercalcemia typically in the first three to fourth months after SCI, causing symptoms such as nausea, lethargy, abdominal pain, polyuria and anorexia. Bone mineral density declines by six weeks post-injury, and bone loss continues for years.

Loss of bone density is greater typically in the distal femur and proximal tibia. Tetraplegics may also lose bone density in the distal radius and ulna.

The Implications

Osteoporosis in bones below the level of injury may cause fragility fractures during movements such as repositioning in bed or transfers. People with SCIs have twice the risk of a fragility fracture in the lower extremity compared to the general population.[3] Fragility fractures are more common in paraplegics or tetraplegics who are more active and likely to fall.

The Interventions

Vitamin D and calcium supplementation is effective in minimizing bone loss. Medications for osteoporosis have been used (e.g., bisphosphonates), but there is no standard guideline for their use after SCI. There is some evidence that standing, and use of functional electrical stimulation, may be helpful.

Musculoskeletal Complications

The Impact

Musculoskeletal complications associated with SCI may cause pain and limit function. Contractures are common in paralyzed limbs due to prolonged joint immobilization. People with SCIs often have very high demands placed on their upper limbs from their work, activities of daily living, and mobilization in a wheelchair. Repetitive motions and recurrent microtrauma can result in overuse injuries, most commonly in the shoulder, causing rotator cuff impingement, subacromial bursitis, osteoarthritis, bicipital tendonitis or capsulitis.

The Implications

These are common causes for chronic pain after SCI. Contractures may lead to difficulties in positioning, pressure injuries, or limit the use of a joint if delayed motor recovery occurs. Functionally, these may limit activities such as dressing, hygiene and transfers. Overuse injuries may also limit mobilization and activities of daily living.

The Interventions

Prevention of overuse injuries and contractures is key. Prevention of overuse injuries can be achieved by appropriate preservation, optimizing biomechanics and use of upper extremities, as well as strengthening exercises. Contractures can be prevented by daily passive range of motion of all joints, proper positioning, and splinting if necessary. Treatment of established contractures may involve serial casting, surgical tenotomies and/or tendon lengthening.

Pain

The Impact

Pain is a very common complication following SCI, with approximately 50% of people with SCIs experiencing chronic

pain that interferes with activities.[4] The International Spinal Cord Injury Pain Classification organizes pain following SCI into nociceptive, neuropathic and other.[5] Neuropathic pain can occur at or below the level of injury. It is often described as a burning, shock-like, shooting sensation and may be accompanied by allodynia and hyperalgesia.

The Implications

Pain following SCI can present acutely and often persists long-term. Chronic pain frequently interferes with activities and work, reducing quality of life.[6]

The Interventions

There are a number of non-pharmacologic and pharmacologic treatments for nociceptive and neuropathic pain. Pregabalin and gabapentin are two of the most commonly used medications for treating neuropathic pain. An interdisciplinary approach to chronic pain management is recommended.

Respiratory Insufficiency

The Impact

Respiratory complications are the leading causes of death for people with SCI for all years after injury.[8] Respiratory insufficiency can occur post-SCI due to respiratory muscle weakness, changes in ventilator control and changes in lung and chest wall compliance. The extent of respiratory muscle weakness depends on the severity and level of SCI. Persons with injuries above C3 result in near total respiratory muscle paralysis, requiring mechanical ventilation. Persons with injuries between C3 and C5 result in partial respiratory muscle paralysis and may require mechanical ventilation during acute hospitalization. Persons with injuries between C6 and C8 result in weak forced exhalation, but the inspiratory muscles are functional.

The Implications

The risk of pulmonary complications, including atelectasis, pneumonia and pulmonary edema, is high for those with cervical- and (to a lesser extent) thoracic-level injuries. Injuries below the thoracic levels typically have little to no respiratory compromise. Reduced lung and chest wall compliance results in a restrictive ventilatory defect. A lack of supraspinal sympathetically mediated bronchodilation may exacerbate respiratory difficulties.

The Interventions

Management of respiratory insufficiency varies depending on the extent of injury but may involve secretion management with assisted cough, lung volume recruitment and ventilator support with non-invasive and/or invasive ventilation.

Venous Thromboembolism

The Impact

Patients with acute SCI have a predisposition to venous thromboembolism (VTE) due to Virchow's triad: immobility causing venous stasis in paralyzed limbs, endothelial injury and alterations in the clotting cascade leading to hypercoagulability.[7] As a result, people with acute SCI have a greater risk of developing VTE than persons with other general trauma.[8]

The Implications

Lack of appropriate VTE prophylaxis may result in pulmonary embolism, which may be fatal.

The Interventions

VTE prophylaxis with mechanical methods of thromboprophylaxis and anticoagulation is recommended. Mechanical thromboprophylaxis with intermittent pneumatic compression devices with or without graduated compression stockings is recommended as soon as feasible after acute SCI when not contraindicated by lower extremity injury. Anticoagulation with a low molecular weight heparin (e.g., enoxaparin) is recommended in the acute care phase after SCI once there is no evidence of active bleeding and if there is no medical contraindication. In the post-acute and rehabilitation phase, low molecular weight heparin, oral vitamin K antagonists (e.g., warfarin) or a direct oral anticoagulant (DOAC) (e.g., dabigatran) may be used for anticoagulation. Anticoagulant thromboprophylaxis should be continued for at least eight weeks after SCI for those with limited mobility. People with chronic SCI who are re-hospitalized for medical illnesses or surgical procedures should also receive thromboprophylaxis during the period of increased risk.[7]

Orthostatic Hypotension

The Impact

Low resting blood pressure and orthostatic hypotension can occur due to interruption of excitatory descending sympathetic input.[9] Pooling of venous blood in the lower extremities also contributes to lower ventricular end-diastolic pressure and stroke volume. Orthostatic hypotension is more common acutely following injury but may persist chronically.

The Implications

Orthostatic hypotension can lead to lightheadedness and syncope. These symptoms may impair mobilization and increase the risk of falls.

The Interventions

Symptomatic orthostatic hypotension is treated non-pharmacologically with compression stockings and abdominal binders to prevent venous pooling, or pharmacologically with medications that raise blood pressure, such as midodrine.

Autonomic Dysreflexia

The Impact

Autonomic dysreflexia (AD) is a syndrome caused by imbalanced reflex sympathetic discharge in response to a noxious stimulus, resulting in a sudden onset of excessively high blood pressure. Patients with a T6 level injury or above are at risk. Symptoms of AD include a pounding headache, blurred vision, sweating above the level of injury, goosebumps and cool skin below the level of injury, a flushed face, and usually bradycardia.

The Implications

AD is a medical emergency that, if untreated, can result in cerebral hemorrhage, seizures, arrhythmias, myocardial damage and even death. Triggers of AD are most commonly genitourinary or gastrointestinal issues such as bladder distension or fecal impaction but may range from ingrown toenails to childbirth.

The Interventions

Treatment is aimed at first correcting the trigger. General measures include emptying the bladder or rectum and loosening tight clothing. If hypertension persists after these measures, antihypertensive medication may be necessary. It is important for patients and caregivers to recognize the symptoms of AD and know how to manage them.

Sexual Dysfunction

The Impact

SCI can affect the psychological, physiological and practical aspects of sexual function and fertility. Men and women with SCI may experience reduced sensation, impaired ability to achieve orgasm and difficulties with self-positioning.

The Implications

Men may experience complete or impaired ability to achieve an erection and ejaculation, which has implications for fertility. Infertility in men with SCI is common due to low sperm viability and motility. Women may have disrupted menstruation acutely following injury, but it usually returns after a few months, and fertility is unchanged. Pregnancy in women with SCI is associated with greater risks of complications such as venous thromboembolism and premature labour, which requires special considerations.

The Interventions

Oral and injectable medications as well as surgical implantations are available for erectile dysfunction. Fertility treatments

may require semen retrieval and insemination. Management of sexual dysfunction involves careful discussion with the individual and their partner. Patients should be educated on preparation for sexual activity, management of autonomic dysreflexia, fertility and family planning.

Pressure Injuries

The Impact

People with SCI have a high risk of pressure injuries due to poor sensation, immobility, compromised nutrition, muscle atrophy over bony prominences, incontinence, spasticity and contractures. Fifty to 80% of people with SCI will develop a pressure injury.[10]

The Implications

Pressure injuries are the second leading cause of hospitalization acutely and long-term[11] and can lead to serious complications including osteomyelitis, septic arthritis, endocarditis and amputation.

The Interventions

Prevention of pressure injuries involves the use of appropriate surfaces for wheelchairs and mattresses, frequent weight shifting, proper transfer techniques, as well as adequate skin care, moisture control and nutrition. The Braden Scale is often used to predict the risk of pressure injury and address underlying causes. Treatment of pressure injuries involves local management of the wound, as well as systemic management such as nutritional treatment, with a patient-centred approach. Treatment of associated soft tissue or bony infections may also be required.

Functional Limitations Following SCI

The functional limitations that result from SCI vary depending on the level of injury. While every patient is unique, there are general functional outcomes that can be expected based on the level of SCI.[12] Since incomplete injuries have variable neurological involvement, these generalizations are made based on a motor complete injury.

- People with C1 to 4 injuries are expected to be independent in a power wheelchair but dependent on others for transfers and most activities of daily living.
- People with C5-level injuries require assistance for most activities of daily living and transfers but are independent in a power wheelchair. Those with a C5 or lower injury are able to drive independently in a specially adapted vehicle.
- Those with C6-level injuries are independent for eating, hygiene and dressing the upper limbs using adaptive technologies after receiving assistance with set-up. They require assistance for other activities of daily living. They are able to mobilize independently in a manual wheelchair indoors and use a power wheelchair outdoors.
- People with injuries at the C7 to C8 level are independent for most activities of daily living but may require assistance for bowel and bladder care, dressing and cleaning their lower limbs. They are independent in transfers and manual wheelchair mobility.
- People with T1 to T9 injuries are independent in all basic activities of daily living, transfers and manual wheelchair propulsion.
- People with T10 to L5 injuries are able to do the same activities as the T1 to T9 level. In addition, they may have some functional ambulation with assistance or even independ-

ently using knee-ankle-foot orthoses or ankle-foot orthoses and forearm crutches or a walker.

People with SCI use assistive technologies to achieve optimal independence to get around and perform their daily activities. Wheelchairs are the most commonly used assistive technology and can be manual or powered. Powered wheelchairs may be propelled by a joystick or head or chin, or be breath-controlled for those who have inadequate hand function. The wheelchair is often customized based on the person's functional goals, environment, age, cognitive abilities, spasticity, skin and cardiopulmonary endurance. Wheelchairs may recline or tilt-in-space to accommodate pressure relief for the prevention of pressure injuries. Transfer aids such as sliding boards and mechanical lifts allow people with SCI to safely move from one place to another with or without the help of others.

A variety of orthoses can be used to assist with activities such as eating, grooming and ambulating. Upper limb orthoses include static and dynamic splints. Static splints provide hand and wrist positioning to prevent contractures. Dynamic splints support weak muscles to facilitate hand function. Lower limb orthoses include knee-ankle-foot orthoses and ankle-foot orthoses that can help support weak leg muscles to facilitate walking with or without other gait aids.

Numerous adaptive self-care devices can be used to compensate for weak grip, poor co-ordination or limited range of motion, and allow the completion of activities of daily living with little to no assistance. Some examples include a universal cuff, built-up handle on a utensil, skin inspection mirror and digital stimulator. Home modifications may also be required to allow people with SCI to safely and efficiently function and move around.

Conclusion

Spinal cord injury is a medically complicated and life-altering condition. Although there is no cure for SCI, advances in clinical practice have reduced morbidity and increased the life expectancy of those with SCI. Through effective rehabilitation and health interventions, assistive technologies and more accessible environments, people with SCI can live full and productive lives.

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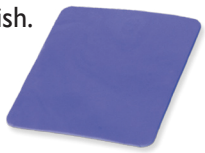

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