Kimberly Dale March 25, 2013
Advanced Neuromuscular

Incomplete Cervical Spinal Cord Injuries

Introduction/Epidemiology

Multidisciplinary healthcare knowledge has certainly come a long way since the first reference to spinal cord injury (SCI) made over 5,000 years ago by an Egyptian physician as "an ailment not to be treated"[1]. Today, traumatic SCI in the United States has an incidence of approximately 12,000 patients per year with an estimated prevalence of 270,000[2]. Motor vehicle crashes continue to be the primary cause of SCI (39.2%), followed by falls (28.3%) and violence-related causes (14.6%)[2, 3]. The proportion of sport-related injuries (8.2%) has decreased over time while the proportion of injuries related to falls has increased. Making up 80.6% of spinal cord injuries, males are predominantly affected with the majority of injuries occurring between the ages of 16 and 30 years old[3].

Injuries to one of the eight cervical segments of the spinal cord result in tetraplegia and these cervical lesions make up approximately one half of all traumatic SCI cases[2]. Of all traumatic SCIs, the most common neurological level of injury is C5, followed by C4 and then C6[3]. Specifically, incomplete tetraplegia is the most frequently diagnosed neurologic category at discharge (40.8%) and continuous to demonstrate an increasing incidence trend. By definition, incomplete tetraplegia involves loss of motor or sensory function of the cervical segments of the spinal cord with some preservation of these functions below the neurological level of injury that includes the lowest sacral segments[3]. Incomplete spinal cord injuries are often categorized based on different clinical syndromes such as central cord, Brown-Sequard, anterior cord, conus medullaris, and cauda equina syndromes.

Central Cord Syndrome (CCS) is the most common of the incomplete syndromes compromising 15-25% of cases and is most frequently seen affecting the cervical spinal cord[3, 4]. This type of injury, characterized by motor weakness in the upper extremities greater than the lower limbs with sacral sparing, most frequently occurs with hyperextension injuries in older adults with cervical spondylosis[3]. However, this syndrome may occur in individuals at any age and from various etiologies such as severe spinal column traumatic injuries and subsequent spinal instability (as seen in motor vehicle crashes) or low velocity traumatic injuries resulting from acute central cervical disk herniations[5]. Trauma is the most common cause of CCS occurring most in motor vehicle accidents, falls, and diving injuries though it can also have nontraumatic etiologies such as spinal epidural absess[4].

Pathology and Pathophysiology

Though exact etiologies of the trauma vary, acute spinal cord injuries are a two-step process. The initial mechanical trauma leads to possible deformation of the spine, direct compression of the spinal cord, and damage of neural elements and blood vessels local through fracture/displaced bone fragments or disc material. Energy delivered to the spinal cord at the moment of impact dictates the extent of

initial tissue disruption and secondary injury. This primary injury of the spinal cord then causes secondary mechanisms including:

- Vascular changes (including ischemia, autoregulation abnormalities, neurogenic shock, vasospasm, hemorrhage, etc)
- Dysregulation of ionic homeostasis (including increase intracellular calcium, extracellular potassium, and sodium permeability)
- Neurotransmitter accumulation (serotonin, catecholamines, and extracellular glutemate)
- Formation of free radicals and lipid peroxidation
- Endogenous opiods
- Edema
- Post-traumatic inflammatory reaction
- Excitotoxic cell injury and apoptosis[6-8].

In terms of primary of injury of CCS specifically, the initial pathology is specific to injury that primarily affects the center of the spinal cord. It is thought that the simultaneous compression of the spinal cord by anterior osteophytes and posterior impingement cause by bulking of the ligamentum flavum during hyperextension injuries is the primary mechanism of injury[3]. This type of etiological mechanism results in damaging of the spinothalamic tract, corticospinal tract, and dorsal columns. Original pathogenesis theories believed that injury to the central spinal cord disrupts the medial lamination of corticospinal tracts (which serve the hands and upper extremity function) while sparing the lateral tracts, which control the sacral and lower extremities[3]. This would help to explain the typical clinical presentation of upper extremity involvement greater than lower extremity as well as motor deficits more prominent than sensory impairments. However, this theory is controversial due to speculation of exact anatomical distribution and contribution to motor function of the corticospinal tracts[5]. Patients will also suffer from secondary injuries described above and exhibit Wallerian degeneration of lateral corticospinal axonal tracts distal to the site of injury[4].

PT Guide To Practice Guidelines

Individuals with incomplete cervical spinal cord injuries can be classified under multiple APTA Practice Patterns defined by the *APTA's Guide to Physical Therapy Practice*[9]. Practice patterns that the APTA particularly classifies tetraplegia/quadriplegia include:

- **Pattern 5H**: Impaired Motor Function, Peripheral Nerve Integrity, and Sensory Integrity Associated with Nonprogressive Disorders of the Spinal Cord
- Pattern 4A: Primary Prevention/Risk Reduction for Skeletal Demineralization
- Pattern 6B: Impaired Aerobic Capacity/Endurance Associated with Deconditioning

- Pattern 6E: Impaired Ventilation and Respiration/Gas Exchange Associated with Ventilatory Pump Dysfunction or Failure
- Pattern 7A: Primary Prevention/Risk Reduction for Integumentary Disorders
- Pattern 7B: Impaired Integumentary Integrity Associated with Superficial Skin Involvement
- Pattern 7C: Impaired Integumentary Integrity Associated with partial-Thickness Skin Involvement and Formation

Effects on Systems:

Incomplete cervical spinal cord injuries result in loss of function of the cervical segments of the spinal cord with some preservation of motor of sensory function below the level of injury. The exact impact of such an injury on all other systems is vast, variable, and dependent on the location and extent of injury. For this reason, it is these individuals with cervical lesions are at the highest risk for significant involvement or effects. However, the intent of an incomplete lesion fairs better than complete. Some of the effects of incomplete cervical spinal cord injuries may include:

- **Central/Peripheral Nervous System:**

  Following SCI there is an interruption between receptor organs and brainstem centers communication as well as the autonomic nervous system leading to dysfunction in the CNS. One autonomic (sympathetic) dysfunction that arises is impaired temperature control because the hypothalamus can no longer control cutaneous blood flow or degree of sweating. In this loss of internal thermoregulatory response, individuals may lose the ability to shiver, vasodilate/vasoconstrict in response to heat/cold, and cool through normal evaporative cooling due to loss of thermoregulation sweating. Lack of sweating may sometimes be compensated for through diaphoresis above the level of injury. It is not uncommon for individuals with incomplete lesion to report "spotty" area sweating below the lesion. In addition, individuals with cervical SCI's thermal regulation is more influenced by the external environment than all other levels of injury and are more likely to experience long-term impairment of body temperature regulation[10].

  In addition, autonomic dysreflexia, a dangerous pathological autonomic reflex, is estimated to occur in as many as 85% of those with tetraplegia during their course of rehabilitation. Episodes of autonomic dysreflexia gradually subside and are more uncommon 3 years following injury[10]. Damage to nerve roots at or near the site of cord damage may result in sharp, stabbing, burning, or shooting nerve root pain or dysesthesias.

  Characterized by hypertonicity, hyperactive stretch reflexes, and clonus; approximately 70% of patients with SCI develop spasticity within 1 year of injury. It typically occurs below the level of lesion following spinal shock and continues to gradually increase during the first year. It

is more common among those with minimal sparing of voluntary movement (incomplete injuries). Decreased sensation, abnormal reflexes, increased deep tendon reflexes, and decreased body awareness may also be apparent following an incomplete cervical SCI[3, 10, 11].

Though uncommon (evident in only 2% of SCI), post-traumatic syringomyelia may occur as early at 2 months after injury. At this time, no relationship has been found between the level of SCI and the likelihood of developing this intramedullary cyst filled with cerebrospinal fluid. Fortunately, those with incomplete injuries are at a lower risk for development than those with complete tetraplegia[12, 36].

- **Musculoskeletal:**

Partial or complete paralysis of voluntary musculature and the associated muscle atrophy are not the only musculoskeletal effects of SCI. Drastic bone mass loss occurs during the first 6 months following SCI and continues over time leading to osteoporosis and increased risk for fracture in the involved limbs. The majority of individuals with SCI experience the most dramatic bone loss in their pelvis and lower extremities, with significant loss occurring in a distal to proximal pattern. However, location of bone loss varies dependent on the location and extent of SCI as well as involved structures. In contrast to the extremities, the spinal column does not appear to be affected by demineralization however[3, 13-15].

Heterotrophic ossification (HO) occurs in up to 53% of individuals with SCI and will limit a patients ROM and ability to perform ADLs in 20% of cases. HO is most commonly develops in the anterior hip followed by the medial aspect of the knee though individuals with tetraplegia are also affected by HO in their shoulders and elbow. Clinical features of HO include swelling, warmth, and fever with decreased ROM and is most often occurs 1 to 4 months following injury[3].

Because of the lack of active muscle functioning, reciprocal muscle groups and surrounding structures do not stretch as they normally would. This unopposed shortening, flaccidity, faulty positioning, HO, edema, and muscle imbalances all contribute to the development of contractures. Hip flexion with components of internal rotation and adduction, as well as elbows, wrists, fingers, knees, ankles, and toes most common in this population[10].

Additionally, various musculoskeletal injuries may result from direct impact or trauma in association with the spinal cord injury. For example, CCS is most often caused by cervical hyperextension injuries. This etiology is also with fractures of cervical spinous processes, laminae, and facets; avulsion fractures of the anterior aspect of cervical vertebrae, ruptured anterior longitudinal ligament, or ruptured disk[10].

As one type of incomplete cervical injury, CCS is also characterized by a unique disproportionately altered motor

impairment of the upper extremities greater than the lower extremities[3]. The typical pattern of recovery also occurs earliest and greatest in the lower limbs, followed by return of bladder function, then proximal upper limbs, and intrinsic hand function[10, 16].

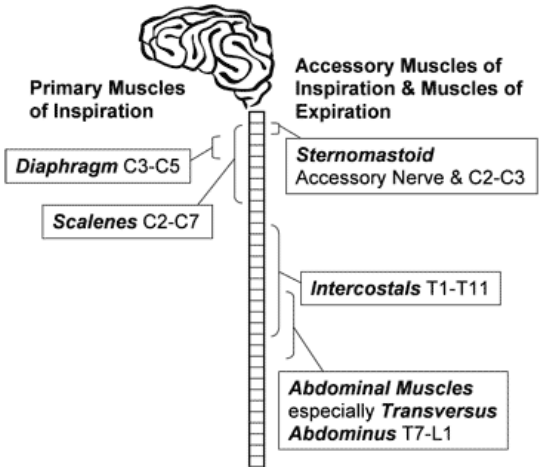
- **Cardiovascular:**

  During acute SCI, hypotension and cardiac arrhythmias such as bradycardia are typically seen due to the loss of sympathetic tone, systemic vascular resistance, and dilation of the venous vessels. Individuals with high-level tetraplegia are most at risk for bradycardia from stimulation of the vagus nerve such as tracheal suction, turning to prone, defecation, and burping. Though orthostatic hyotension is more commonly seen in patients with complete cervical SCI than incomplete, this is still a prominent condition following incomplete cervical SCI. Deep vein thrombosis (DVT) is the most common of the cardiovascular complications and more common in patients with tetraplegia. 1-7% of patients with SCI will develop a pulmonary embolism from deep veins of the lower limbs but this is not influenced by the degree or level of SCI[3, 10].

- **Pulmonary:**

  Injuries between C1 and C3 affect phrenic nerve innervation, resulting in impaired or lost spontaneous respiration. In these cases, an artificial ventilator or phrenic stimulator is needed to live. At least some level of respiratory function compromise exists in all patients with tetraplegia. The level of impairments is directly related to the lesion level, residual respiratory muscle function, presence of additional trauma at the time of injury, and premorbid respiratory status. The higher the lesion level, the greater the loss of respiratory function can be expected. Possible involvement or paralysis of the following muscles may result in the associated impairments:

  - Intercostals- decreased chest expansion, lower inspiratory volume, decreased expiratory efficiency (may not increased involvement of accessory muscles during inspiration)
  - Abdominals/External Obliques- decreased expiratory efficiency, expiratory reserve volume, cough effectiveness, and ability to expel secretions



Individuals with respiratory involvement may also present with an altered breathing pattern characterized by upper chest wall flattening, decreased chest wall expansion, and dominant epigastiric rise during inspiration. This alter way of breathing could lead to permanent postural change. Reduced ventilation and inability to effectively cough and clear secretions can result in atelectasis of pneumonia, the most common cause of death in individuals with SCI (71.2%)[3, 6, 8, 10, 36].

- **Integumentary:**

  Skin breakdown and pressure ulcers are a common and costly sequelae following SCI. Paralysis, decreased sensation, incontinence, infections, obesity, spasticity, joint contracture, edema, anemia, and poor nutrition are all risk factors that predispose this population to pressure ulcers. Inadequate pressure relief, shear forces, friction, and improper transfer techniques further increase the coincidence of skin breakdown. The most common sites of skin breakdown include the sacrum, heels, ischial tuberosities, trochanters, scapula, and occiput[3].

- **Gastrointestinal/Genitourinal:**

  Acutely, almost all patients have paralytic ileus, gastric atony, and distention during the early phases of spinal shock (about 5 days). The most frequent gastrointestinal problem following SCI is altered bowel elimination. Decreased mobility, lack of sensation, and disrupted autonomic control of GI all cause conditions such as delayed gastric emptying, poor colonic motility, prolonged bowel transit time, constipation, postprandial, and abdominal distension[3, 17].

  Bladder dysfunction is over a key acute characteristic of CCS. Bladder outcome in patients with CCS is shown to be better than other patients with incomplete tetraplegia and considerable function recovery can occur, typically within the first 6 months post-injury. Approximately 52-84% of patients with CCS will achieve spontaneous voiding[18]. Patients with cervical injuries will most likely suffer from neurogenic spastic bowel dysfunction.[10]

  Many patients with incomplete cervical SCI will also suffer from sexual dysfunction. Males with UMN lesions and incomplete injuries have a greater erectile capacity than complete and LMN lesions however most males are infertile following injury. In women, the potential for conception remain unimpaired.[10]

From the perspective of the ICF Model:

- **Impairments:**

  Individuals with incomplete cervical spinal cord injuries may suffer from multiple various impairments. The extent and location of the injury will then determine the severity and presence of the impairment. A few impairments that may be present in this population include[3, 9, 10, 19]:

  - Impaired touch sensation
  - Impaired sensation of pain

- Impaired motor function (paralysis, decreased strength, atrophy)
- Decreased muscle endurance and performance
- Impaired respiratory function
- Impaired exercise tolerance/decreased aerobic capacity
- Impaired peripheral nerve integrity
- Decreased ROM
- Joint Contractures
- Abnormal Tone
- Pain (traumatic pain, musculoskeletal, neuropathic, dysesthesias)
- Impaired reflexes and responses to stimuli
- Impaired coordination
- Impaired Gait
- Impaired skin integrity
- Bowel/bladder dysfunction
- Edema
- Inflammation
- Ischemia

- **Activity/Participation/QOL**

  As is the theme, the extent and location of the injury is a key determinant in establishing a patient's required dependence with activities or capabilities in participation. For example, individuals with CCS tend to have a favorable prognosis to other incomplete cervical lesions due to the significant functional recovery achieved by this patient population. The majority of these individuals will gain independence with ambulation and dressing as well as bowel and bladder function[3, 10, and 18].

  Patients with the most proximal nerve root innervation at C1, C2, or C3 are often fully dependent with activities of daily living (ADLs) and transfers, requiring full time assistance. Despite the inability to perform these skills independently, it is crucial that they can verbalize self-care needs. They can be independent with a power wheelchair however increasing their access to the community and ability to participate. Individuals with a C4 injury are primarily still dependent for ADLs and transfers but gain limited ability to self feed, turn pages, and participate in recreational table games or painting with adaptive sticks and using their mouth. An individual with a C5 injury may need less assistance with ADLS at a tabletop and with some setup assistance. These individuals gain function in limited upper extremity dressing, washing, brushing teeth, grooming and other self care activities with adaptive equipment. A patient may also be able to participate in driving using van hand controls. . Injuries at C6 or below gain a significant advantage in independence with the individual's use of wrist extension and shoulder flexion. Patients may gain full independence in ADLs, self-feeding, dressing, self care, wheelchair propulsion, transfers, skin pressure relief, bowel/bladder care, cough, driving, and meal preparation. Limited participation in sports such as bowling or fishing are also available. Patients continue to gain independence and efficiency in these activities with progressively less need for adaptive equipment with C7 and C8 injuries[3, 10]. Common

activity limitations (of course dependent on location and extent of injury):

- Lying down
- Standing
- Bending
- Shifting center of gravity
- Maintaining a sitting position
- Maintaining a standing position
- Transferring oneself
- Lifting/carrying heavy objects
- Hand and arm use
- Walking short/long distances
- Walking on different surfaces
- Walking around obstacles
- Using adaptive equipment

Limitations in many of these activities then greatly impact the individual's ability and desire to participate in the community. Common limitations for full community participation in this patient population include the inability to keep up with peers, inability to perform work (job/schools/play), and difficulty accessing the community[9]. Environmental factors such as familial support, living in an accessible physical and social community, as well as self-esteem all increase the chance that an individual with participate in their community and increase their quality of life (QOL). Impairments of pain, bowel/bladder/sexual dysfunction, and spasticity have been associated with a negative QOL in individuals with SCI. Mobility and increased participation in physical activity or sports have all been associated with improved quality of life[20-22, 36].

Interventions

Cervical level spinal cord injuries result in the loss upper limb and hand motor and/or sensory function. Even in incomplete spinal cord injuries where some neuronal information can pass through the spinal cord, there are still often distorted transmissions leading to chronic pain or spasticity. The loss of upper limb function, especially the hands, is reported to be one of the most significant and devastating losses an individual may experience, as it is needed for completion of basic ADLs, transfers, and optimal mobility[23]. In fact, over 75% of patients with tetraplegia consider upper extremity function their highest priority and that regaining this function would most improve their quality of life[24, 25]. According to patients, this priority remains superior to all others regardless of how long someone has been injured[24]. Even in central cord injuries which have the best prognosis of incomplete spinal cord injuries, upper limb and hand functions are the last to recover, if ever[3, 36]. Therefore, management of the upper limb following tetraplegia is critically important and tends to be very eclectic with the use of traditional rehabilitation interventions, use of orthosis and surgery. During the acute phase of SCI rehabilitation, realistic goals of maintaining ROM, improved strength, tone management, spasticity, and the prevention of secondary complications in order to achieve the person's maximal functional ability are priority[23, 35].

Muscle weakness, loss of autonomic control below the injury level, reduced activity, and changes in metabolic/vascular function significantly decrease an individual's physical capacity in the acute stage. In individuals with tetraplegia, the level of injury is significantly correlated with functional ability, peak oxygen intake, muscle strength of sitting balance, spasticity of the lower limb, handgrip strength, wrist extensor strength, and global upper extremity strength[23]. It is believed that natural muscle strength recovery can occur up to two years post -injury, with most of the recovery occurring in the first 6 months. It is also thought that changes in strength noted in the first 6 months of injury are due to collateral sprouting in the spinal cord while changes at 2-8 months are caused by peripheral nerve sprouting and fiber hypertrophy[23, 35].

Interventions aiming to improve strength such as neuromuscular stimulation (NMS)-assisted exercise have been shown to be effective at improving not only strength, but preventing injury, and increasing independence in patients with tetraplegia. NMS-assisted exercise ergomety has been found effective at strengthening the upper limb in this population during the acute stage and even well after injury[26]. Also, with upper extremity exercise training, patients often report decreases in stress, pain, and depression along with enhanced self-concept and overall QOL[27]. Other studies suggest the NMS-assisted exercises improve upper limb strength over conventional rehabilitation strategies[28]. Utilizing augmented feedback strategies such as biofeedback to reacquire motor skills of the upper extremity has been tested as well in this population. Unfortunately, at this time all evidence suggests that the use of augmented feedback to improve upper extremity function is not effective in rehabiliation[23].

Because cortical, subcortical, and much of the local spinal cord circuitry remains intact and partially connected during incomplete SCI; the CNS is capable of substantial reorganization. Images even show changes in the cortex following SCI with expansion of areas controlling muscles spared after SCI into the cortical areas previously related to muscles reinnervated below the lesion[29, 30]. Massed practice such as constraint-induced therapy and somatosensory peripheral nerve stimulation (SS) are two therapy interventions believed to contribute to changes in the cortex. In two randomized control trials, Beekhuizen and Field-Fote[31, 32] combined mass practice with afferent median nerve stimulation (500 ms train, 10Hz, 1 ms pulse duration) for 2 hours, 5 days/week for 3 weeks in patients with incomplete cervical spinal cord injuries. This combination resulted in significant improvements in upper extremity function, pinch grip strength, sensory scores, and promotes cortical reorganization. It is believed that the afferent inputs from sensory stimulation in association with repetitive movements and peripheral nerve stimulation are a key component in the cortical neuroplasticity needed to improve upper extremity function[23].

One other innovative method to achieve this cortical change is repetitive transcranial magnetic stimulation (rTMS). This noninvasive technique allows for electromagnetic currents in a coil to produce magnetic pulses to cross the cranium and induce neuron depolarization and changes in cortical excitability. The effects can last minutes to years and have been shown to reduce pain and otherwise intractable spasticity as well. In one study, Belci and colleagues applied rTMS to 5

patients with chronic incomplete C5 SCIs for 5 days of sham rTMS followed by 5 days of therapeutic rTMS and found efficacy in reduced corticalspinal inhibition and improved functional recovery. Patients increased in their AIS sensory and motor scores as wel as their response to cutaneous estim over the thenar muscles and improved hand/finger function[33].

Though advanced innovative technology and knowledge of neuroplasticity is exciting, there are traditional interventions, such as splinting, that cannot be forgotten. Early protective splinting in the acute stage of tetraplegia is just as important in order to prevent deformity and contractures, protect insensate areas from injury, prevent or reduce edema, counteract hypertrophic scars, support weak muscles, decrease pain, and promote a functional hand[3, 23]. Resting pan or paddle, wrist extension, short hand, and enuresis splints are the most common splints given to patients with tetraplegia. Though this is very common practice, very little research exists regarding the effectivness of splinting and most appropriate selection for various levels of injury and stage of SCI. Recommendations do exist however. For individuals with acute C1-C4 injuries, the wrist should be positioned in 30 degrees of extension and thumb placed in full abduction and extension in a long opponenes wrist-hand-orthosis during the day. Nighttime splinting then places the hand in an intrinsic plus posture. For patients with midcervical injuries, there are two distinct groups: those with gravity-eliminated wrist extension strength and those with against-gravity strength. The flexor hinge orthosis is the functional orthosis of choice for individuals with C6 injuries. This training should include self-catheterization activities, feeding, tooth brushing, writing, and other ADLS as well as donning/doffing the orthosis[3, 34].

When applying this information clinically, it is important to remember that ROM exercises and proper bed positioning, are all also key in preventing upper extremity injuries further along the rehabilitation and recovery path. Strengthening should be encouraged early in the acute phase with the assistance of NMS as appropriate. Feedback should be used throughout the rehabilitation process toaid in motor learning though biofeedback may not be the best method of choice for this population. Though innovative technologies such as rTMS may be beneficial in the acute stage, it is important to set patients up with a program that they can sustain after leaving acute or subacute care. Therefore, strengthening, ROM, and protective methods should be available at home or beyond the care of a hospital for increased compliance. Patients should be educated that the shoulder joint is the most common joint for patients to complain of pain following tetraplegia, as it is not well designed to handle the pressures required of it during transfers and mobility. Patients with tetraplegia are at even higher risk for injury due to partial innervation and impaired balance of the shoulder, scapular, and thoracolumbar muscles. Therefore upper extremity range of motion, strength, injury prevention, and restorative interventions continue to play a large role not only during the acute rehabilitation phase, but later phases and for the rest of the individual's life[34].

References:

1. Guttman L. *Spinal cord injuries: comprehensive management and research*. Oxford: Blackwell Scientific Publications. 1973.
2. National Spinal Cord Injury Statistical Center (NSCISC). *Spinal Cord Injury Facts and Figures at a Glance*. Birmingham Ala: The University of Alabama at Birmingham; 2012. Available: https://www.nscisc.uab.edu/PublicDocuments/fact_figures_docs/Facts%202012%20Feb%20Final.pdf
3. Sisto S, Druin E, Sliwinski M. Spinal Cord Injuries: Management and Rehabilitation. Mosby Elsevier. St. Louis Missouri. 2009.yrngomyelia. *Spinal Cord Injury Rehabilitation Evidence.* 2012.
4. NowakD, Lee J, Gelb D, Poelstra K, Ludwig S. Central Cord Syndrome. *Journal of Acad of Ortho Surg*. 2009;17(12):756-765.
5. Harrop J, Sharan A, Ratliff J. Central Cord Injury: pathophysiology, management, and outcomes. *The Spine Journal*. 2006; 6: 198S-206S.
6. Ambrozaitis KV, Kontautas E, Spakauskas B, Vaitkaitis D. Pathophysiology of acute spinal cord injury. *Medicina (Kaunas)*. 2006; 42(3):255-261. Abstract only.
7. Kwon B, Tetzlaff W, Grauer J, Beiner J, Vaccaro A. Pathophysiology and pharmacologic treatment of acute spinal cord injury. *Spine*. 2004; 4(4):451-464.
8. Sekhon L, Fehlings M. Epidemiology, Demographics, and Pathophysiology of Acute Spinal Cord Injury. *Spine*. 2001; 26:S2-S12.
9. American Physical Therapy Association. *Interactive online guide to physical therapist practice.* Vol 2012. APTA; 2010. http://guidetoptpractice.apta.org.libproxy.lib.unc.edu/. Accessed March 20, 2013. 10.2522/ptguide.978-1-931369-64-0.
10. O'Sullivan S. & Schmitz T. Traumatic spinal cord injury. *Physical Rehabilitation: Assessment and Treatment.* 5th ed. 2001. F.A. Davis Company. Philadelphia, PA. p.873-923.
11. Hsieh JTC, Wolfe DL, Townson AF, Short C, Connoly SJ, Mehta S, Curt A, Foulon BL. Spasticity following Spinal Cord Injury. *Spinal Cord Injury Rehabilitation Evidence.* 2012.
12. Teasell RW, Metha S, Janzen S, Boyd M, Sequira K. Post-traumatic syringomyelia. *Spinal Cord Injury Rehabilitation Evidence.* 2012.
13. Charmetant C, Phaner V, Condemine A, Clamels P. Diagnosis and treatment of osteoporosis in spinal cord injury patients: a literature review. *Annals of Physical and Rehabilitation Medicine.* 2010; 53: 655-668.
14. Craven C, Krassioukov A, Ashe MC, Eng JJ (2012). Bone Health Following Spinal Cord Injury. In: Eng JJ, Teasell RW, Miller WC, Wolfe DL, Townson AF, Hsieh JTC, Connolly SJ, Mehta S, Sakakibara BM, editors. Spinal Cord Injury Rehabilitation Evidence. Version 4.0 Vancouver: p 1-32.
15. Giangregorio L, McCartney N. Bone loss and muscle atrophy in spinal cord injury: epidemiology, fracture, prediction, and rehabilitation strategies. *J Spinal Cord Med.* 2006; 29(5):489-500.

16. Bosch A, Stauffer ES, Nickel VL. Incomplete traumatic quadriplegia: a ten year review. *JAMA*. 1971; 216: 473-478.
17. Krassioukov A, Eng JJ, Venables B. Neurogenic Bowel Following Spinal Cord Injury. Spinal Cord Injury Rehabilitation Evidence. 2012. Version 4.0. Vancouver, p 1-39.
18. Tow AM, Kong KH. Central Cord Syndrome: functional outcome after rehabilitation. *Spinal Cord.* 1998; 36: 156-160.
19. Rauch A, Escorpizo R, Riddle D, Eriks-Hoogland I, Stucki G, Cieza A. Using a case report of a patient with spinal cord injury to illustrate the application of the International Classification of Functioning, Disability, and Health during Multidisciplinary patient management. *Phys Ther*. 2010;90(7):1039-1052
20. Anneken V, Hanssen-Doose A, Hirschfeld S, Scheuer T, Thietje R. Influence of physical exercise on quality of life in individuals with spinal cord injury. *Spinal Cord*. 2010; 48:393-399.
21. Noonan V, Kopec J, Zhang H, Dvorak M. Impact of associated conditions resulting from spinal cord injury on health status and quality of life in people with traumatic central cord syndrome. *Arch Phys Med Rehabil*. 2008; 89: 1074-1082.
22. Slater D, Meade M. Participation in recreation and sports for persons with spinal cord injury: Review and recommendations. *NeuroRehabilitation*. 2004; 19: 121-129.
23. Connolly SJ, Mehta, S, Foulon BL, Teasell RW, Aubut JL. Upper Limb Rehabilitation Following Spinal Cord Injury. *Spinal Cord Injury Rehabilitation Evidence.* 2012.
24. Anderson KD. Targeting recovery: priorities of the spinal cord-injured population.*J Neurotrauma*. 2004;21:1371-1383.
25. Hanson RW, Franklin MR. Sexual loss in relation to other functional losses for spinal cord injured males. *Arch Phys Med Rehabil*. 1976;57:291-293.
26. Haisma JA, Bussmann JB, Stam HJ, Sluis TA, Bergen MP, Dallmeijer AJ, de Groot S, van der Woude LH. Changes in physical capacity during and after inpatient rehabilitation in subjects with spinal cord injury. Arch Phys Med Rehabil 2006;87:741-748.
27. Hicks Al, Martin KA, Ditor DS, Latimer AE, Craven C, Bugaresti J, McCartney N. Long-term exercise training in persons with spinal cord injury: effects on strength, arm ergometry performance and psychological well-being. Spinal Cord 2003;41:34-43.
28. Cameron T, Loeb G, Peck R, Schulman J, Strojnik P, Troyk P. Micromodular implants to provide electrical stimulation of paralyzed muscles and limbs. IEEE Trans Biomed Engng 1997;44:781-790.
29. Bruehlmeier M, Ditz V, Leebdersk L, Roelcke U, Missimer J, Curt A. How does the human brain deal with spinal cord injury? *Eur J Neurosci*. 1998; 10:3918-3922.
30. Raineteau O, Schwab ME. Plasticity of motor systems after incomplete spinal cord injury. Nature Reviews/Neuroscience 2001;2:263-273.
31. Beekhuizen KS, Field-Fote EC. Massed practice versus massed practice with stimulation: Effects on upper extremity function and cortical plasticity in

individuals with incomplete cervical spinal cord injury. *Neurorehabiliation and Neural Repair*. 2005; 19: 33-45.

32. Beekhuizen KS, Field-Fote EC. Sensory stimulation augments the effects of massed practice training in persons with tetraplegia. *Arch Phys Med Rehabil*. 2008;89:602-608.

33. Belci M, Catley m, Husain M, Frankel HL, Davey NJ. Magnetic brain stimulation can improve clinical outcome in incomplete spinal cord injured patients. *Spinal Cord*. 2004; 42:417-419.

34. Consortium for Spinal Cord Medicine: Preservation of upper limb function following spinal cord injury: a clinical practice guideline for health care professionals. *J Spinal Cord Med.* 2005. 5:433-470.

35. Drolet M, Noreau L, Vachon J, Moffet H. Muscle strength changes as measured by dynamometry following functional rehabilitation in individuals with spinal cord injury. Arch Phys Med Rehabil 1999;80:791-800

36. Pollard ME, Apple DF. Factors associated with improved neurologic outcomes in patients with incomplete tetraplegia. *Spine*. 28 (2003);33-39.