

Spinal Cord Injury and Life Care Planning

Written By: Dr. Terry Winkler, M.D.

Spinal Cord Injury (SCI) presents a challenge for the rehabilitation professional not only because of the potentially severe consequences but also because such injuries occur primarily among the young and have long lasting physical, psychological, social, and vocational implications in rehabilitation. For years, physicians were frustrated by their inability to affect meaningful change for SCI patients. Though this is evidenced in many ways in health care, perhaps the most vivid was Dr. William Asher's description of a paralyzed patient:

“Picture the pathetic patient lying long a bed, the urine leaking from his distended bladder, the lime draining from his bones, the blood clotting in his veins, the flesh rotting from his seat, the scybala stacking up in his colon, and the spirit evaporating from his soul.”

This is a description that the elite, intelligent academia of medicine used to describe SCI patients just three generations ago (Asher, 1947). Fortunately, there have been many positive changes in health care and in society for SCI patients. Technology is rapidly changing and continues to provide positive changes in life expectancy and quality of life for SCI people. There are a large number of physicians and health care providers present today who still hold a somewhat view of SCI similar to Dr. William Asher's.

One goal of this reading assignment is to provide the background information that the vocational rehabilitation counselor and life care planner need to prevent the above portrait from developing in a SCI person's life. The rehabilitation professional should become involved with the SCI patient after the acute hospital care. The life care planner must have a thorough working knowledge of the physiological effects of SCI, the most common side effects of SCI, and proper medical interventions of SCI.

He or she must work with other health professionals to provide counseling for the patient and the family, offer suggestions for environmental modifications, equipment and services for the patient that offer greater mobility and independence. The following reading assignment is intended to provide a foundation of basic medical knowledge of SCI, vocational information, functional abilities of SCI people, discuss future medical and non-medical needs, and as such, serves as an introduction to life care planning and SCI.

Introduction

Spinal cord injury (SCI) is a traumatic insult to the spinal cord that can result in alterations of normal motor, sensory, and autonomic function (Stass, et al., 1993). The discussion will be confined to traumatic SCI; however, the principles will apply to SCI of all etiologies.

The spinal cord has three basic functions:

(1) It serves as a conduit to bring sensory messages from the body and internal organs to the brain where the brain can monitor activities of all structures and act as a central processing unit to interpret messages from the body;

(2) The spinal cord, in a similar fashion, carries messages from the brain to the effector organs or structures in the body. In this regard, the spinal cord can be viewed as a series of cables or connections between the brain and the body;

(3) Protective and coordination function where the spinal cord contains reflex mechanisms to protect the body (i.e., withdrawal reflexes) and centers that facilitate or coordinate some bodily functions, such as urination controlled by the micturition center in the sacral cord.

This is, of course, an over simplification of the spinal cord. There is a tremendously complex structure and organization to the spinal cord with literally hundreds, if not thousands, of functions being performed. Many of the body's autonomic functions are coordinated and regulated at least in part in the spinal cord, and we have learned that the modulation and control of pain is in part based in the spinal cord. There is a very complicated group of interneurons and proprioneurons in the spinal cord whose roles are to facilitate or inhibit the activity of other neurons in the spinal cord. There is virtually no bodily function occurring below the level of the foramen magnum that is not influenced in some way by the integrity of the spinal cord. Therefore, the number of complications and problems that occur as a direct result of SCI are enormous with implications for almost every body system. (Schoenen, 1991.)

Epidemiology

The most recent and current discussion of epidemiological factors of SCI can be found by Bette Go (Go et al., 1995). The model systems data provides good information on general trends in SCI and represents a good starting point for discussion of epidemiology in SCI. It should, however, be pointed out that the model systems data perhaps is skewed towards individuals who have a higher level of lesion and who have an adequate funding source. Individuals who have lower level SCIs or incomplete SCIs or who do not have adequate funding for extended hospital stays tend to be treated locally rather than referred to the model systems. Krause and colleagues found an incidence of SCI of 32 cases per million per year who survived or reached a hospital with an additional 21 cases per million per year dying prior reaching the hospital (Krause, 1975). Griffin and colleagues describe an incidence that approached 55 per million with 35 cases per million surviving to reach the hospital (Griffin et al 1981).

The incidence of SCI has been reported as low as 29 cases per million to a high of 60 cases per million in various studies. There appears to be some variability from state to state regarding the exact rate of SCI. However, overall the annual

range of hospitalized individuals with SCI is between 30 and 40 cases per million. This would correspond with between 7,000 and 10,000 new cases of SCI per year in the United States. The prevalence of individuals with SCI at any one given time in the U.S. is between 180,000 and 230,00 persons (Go et al., 1995).

Etiology of SCI

Gibson (Gibson, 1992) has described the four leading causes of SCI as motor vehicle accidents, falls, violence, and sports injuries.

Etiology of SCI

Injury Source	Percent of Total Injuries
Motor Vehicle Accidents	45%
Falls	20%
Violence	15%
Sports	15%
Other	5%

Automobile accidents remain the number one cause of traumatic SCI, but there has been a recent decline reported (Go et al., 1995). The peak incidence occurred between 1978 and 1980 at 47% and has recently dropped to as low as 38% reported in 1990. This reduction in SCI and motor vehicle accidents may in part be attributed to the improved safety features of some automobiles. Another interesting trend is the reduction of sports related SCIs that has occurred over the last 15 years. SCIs as a result of falls has increased by 5% in the same period of time, and SCI as a result of violence has almost doubled from 1978 to 1990. In some areas, violence is the number two cause of SCI. Louisiana ranks it as the number two cause resulting in 32% of its reported cases of SCI (Lawrence et al. 1992).

Go (Go et al., 1995) pointed out that the etiology of SCIs differs substantially by age, gender, and race. The most common age of injury is age 19 with a range of 16 to 30. 80% of all SCIs are males. The mean age at the time of SCI from 1973 through 1992 has increased by 4.9 years with the mean age of 28.5 years increasing to a mean age of 33.4 years. This trend has important implications. Since older persons with SCIs tend to have more preexisting major medical conditions and are more likely to have tetraplegia, they develop a higher rate of secondary complications and more frequent hospitalizations than their younger counterparts (Go et al., 1995 and Roth et al., 1992).

There are seasonal variations of SCI with the lowest number of SCIs occurring during the winter months, particularly February. The highest number of SCIs occurs during the summer months with July having the highest incidence. 50% of all SCIs occur on the weekend days with 20% occurring on Saturday, which is

nearly double the rate of SCIs occurring during weekdays.

Fifty five percent of all spinal cord injured people have tetraplegia with the remainder being paraplegia. Tetraplegia is defined as paralysis or partial paralysis in four extremities, with paraplegia being paralysis or partial paralysis in two extremities. Between 50 and 55% of all SCIs have some sensory sparing and can be classified as incomplete.

Basic Anatomy of the Spinal Column

The spinal column is made of thirty-three vertebrae, intervertebral disks, and ligaments. The purpose of the vertebrae are to provide a weight bearing structure or spinal column and to house the spinal cord and protect it. In addition, the vertebral column allows a great deal of flexibility in the cervical and lumbar spines. There is a relatively high degree of rigidity in the thoracic spines, which is easily identified by the rib cage, which houses the internal organs for support and protection.

Vertebrae

- . *Seven Cervical Vertebrae (Neck)*: These support the head and provide a great deal of mobility. There are eight spinal nerves, C1 through C8. This is accomplished by the first cervical vertebra having a spinal nerve exiting above and below it, with each vertebral body from that level down having a spinal nerve exiting below the vertebra.
- . *Twelve Thoracic Vertebrae, which* support the ribs.
- . *Five Lumbar Vertebrae* (lower back), which allow flexion and extension, some rotation and side bending.
- . *Five Sacral Vertebrae, which* provide a base of support and attachment for the pelvis.
- . *Four Coccygeal Vertebrae* fused together to form the tailbone.

Each vertebral body is separated by an intervertebral disk, which is made of cartilage and acts as a shock absorber and a cushion for the spinal cord. The intervertebral disks make up one-fourth of the total height of the spinal column and allow a great deal of flexibility between vertebral bodies. The vertebral disks have the ability to herniate and can cause injury to the spinal cord or nerve root in cases of severe herniation.

There are numerous ligaments that are responsible for maintaining the integrity of the spinal column and its alignment. Two of the most important are the anterior longitudinal ligaments on the front of the vertebral bodies and the posterior longitudinal ligaments on the back of the vertebral bodies. If either of these ligaments is torn, the column is said to be unstable and this greatly increases the likelihood of SCI or damage.

The central nervous system is made up of the brain and the spinal cord. It is completely encased in a very protective membrane, the dura mater, and is bathed in cerebrospinal fluid. The spinal cord begins at the base of the skull, the foramen magnum, and extends to the L1 or L2 vertebral level, ending in the shape of a cone called the conus medullaris. From the conus medullaris down, nerve roots continue down through the spinal canal to exit at their proper levels. These nerve roots are referred to as the cauda equina.

It is important to note that there is a disparity between the bony level and the neurological level. For example, the nervous segments that are adjacent to the L1 vertebral body in the spinal canal are S2, S3, and S4. Therefore, an injury to the L1 vertebral body would result in damage to S2, S3, and S4 nerves. This is a very important concept in life care planning. When developing a life care plan, it is extremely important that the life care plan be developed for the neurological level of injury, not the bony level of injury.

Mechanisms of Spinal Cord Damage

- (1) Overstretching or tearing of the nervous tissue of the spinal cord.
- (2) Direct pressure on the spinal cord from the bony fragments, bulging disks, or hematoma.
- (3) Second intention factors that occur at the level of the SCI with swelling and edema produce increased pressure and decreased blood flow in the area of the SCI, leading to further damage.

The initial SCI usually does not result in a complete disruption of the cord. The factors that occur in the first few hours after the SCI may contribute to the spinal cord deficit. For this reason there has been a great deal of attention focused on treatment of SCI early on. Numerous interventions have been tried with variable results. It is generally felt that a high dose steroids given within four hours after the SCI may have some beneficial effect, although there are conflicting reports in this regard. This area remains an intense area of research now in SCI and may hold the most promise at providing some relief from the effects of SCI.

It is possible to determine the mechanism of injury in SCI by reviewing x-rays and CT scans. Axial compression alone such as a diving accident will result in a burst type fracture. Rotation combined with flexion is the most damaging type of force on a spinal column and it will result in disruption of the posterior ligamentous structure (Staas et al., 1993). Central cord syndromes are the result of a hyperextension injury. Thoracic spine injury require much greater forces due to the protective effect of the rib cage and the stability of the spine. These are usually only involved in very high -speed vehicular type traumas or accidents that involve very high forces. They also can occur when the occupant is ejected from the vehicle (Zigler- Field, 1992).

Distraction forces placed at a vertebral body can result in a chance fracture. This type of injury is seen in motor vehicle accidents where a lap belt only is worn. This can be prevented with the use of a shoulder harness belt in addition to the lap belt. A similar injury occurs in automobiles that are provided with passive restraints in which the shoulders are restrained but the hips are not restrained. A collision can result in the person's hips moving forward causing a hyperflexion of the neck and resultant spinal cord injury at a higher level.

The stability of the spinal column is determined by the intactness of the anterior and posterior longitudinal ligaments as well as the vertebral body and will dictate whether or not surgery is indicated. In the past, it was felt that in the case of a complete SCI that surgical decompression was not beneficial. This has been shown by studies at the Miami Project not to be accurate, and that even late surgical decompression a number of years after the SCI can result in improvement. This issue is of the utmost importance in cervical SCI where, for example, a late decompression can result in a person with C5 motor function having an improvement to C6 motor function, which would make a tremendous difference in their functional outcome. In general, for the lower level SCIs, decompression is not as crucial. Spinal cord management may include traction, halos, bracing, Harrington or similar rods, and/or fusion.

Nomenclature

It is extremely important to establish a worldwide standard for the nomenclature and classification of SCI. Without this, it is impossible to perform meaningful research from center to center or country to country in SCI. Likewise, it is impossible to talk about SCI in terms of life care planning and to critique and review and make recommendations in life care planning without a standardized nomenclature system.

The American Spinal Injury Association (ASIA) and the International Medical Society of Paraplegia have developed a worldwide nomenclature system. This system of classification gives key sensory levels to identify dermatomes of injury and key muscle levels to identify the levels of muscle functions. SCIs may be complete or incomplete with partial sparing. The ASIA classification system includes a level for the sensory impairment and a level for the motor impairment, as well as a letter designation for the degree of completeness.

The Frankel classification system is used to describe completeness, with five classes being recognized

Class A: Complete SCI - All motor and sensory function is absent below the zone of partial preservation.

Class B: Incomplete SCI - Sensation preserved below the level of injury, but no voluntary motor being preserved.

Class C: Incomplete preserved motor below the level of lesion, but nonfunctional. There may be some minimal motor activity below the level of the lesions, such as moving a foot or extremity, but it is nonfunctional.

Class D: Incomplete SCI: Some motor preserved below the level of lesion that is useful. This is a level of motor strength that is graded at 3 or more. The person may be able to use the motor. For example, for a brief transfer.

Class E: Complete return of all motor and sensory function below the level of the lesion, but may have abnormal reflexes.

Specific Types of Incomplete Syndromes

1. Central Cord Syndrome. Central cord syndrome is said to be present when the individual has paralysis greater in the upper extremity than the lower extremities.
2. Brown- Sequard Syndrome. Brown-Sequard syndrome is a hemisection of the spinal cord and is characterized by ipsilateral paralysis with contralateral sensory loss from the level of the lesion down.
3. Cauda Equina Syndrome. Cauda equina syndrome is an injury to the lumbosacral nerve roots within the neural canal below the conus medullaris resulting in a loss of bowel and bladder control and weakness of the lower extremities or paralysis.
4. Conus Medullaris Syndrome. An injury to the sacrospinal cord at the level of the conus and the lumbosacral nerve roots which results in areflexic bladder and bowels, and lower limb paralysis (American Spinal Injury Association, 1992).
5. Anterior Cord Syndrome. A lesion that produces variable loss of motor function and of sensitivity to pinprick and temperature while preserving proprioception (American Spinal Injury Association, 1992).

Functional Outcomes

SCI is considered to be a permanent condition with very few people experiencing significant recovery from SCI. Individuals with complete SCI have very little improvement with only 2% improving to Frankel's Class D. Of those who present with a Frankel's Class B SCI, 20% will improve to a Frankel's Class D or E. For Frankel's Class C, 50% will improve to a Frankel's Class D or E. The length of time since the SCI is also a factor in prognosis. Individuals who have had no improvement within the first six months to one year are considered to have permanent SCI with no likelihood for improvement.

Upper motor neuron (UMN) lesions refer to a lesion in the spinal cord that occurs at the T11 or T12 level or higher. Most tetraplegics have a UMN lesion. UMN lesions are characterized by increased spasticity and intact reflex bladder, bowel and sexual functioning.

Lower motor neuron (LMN) injuries occur at T12 or below, usually seen in paraplegics, especially cauda equina syndromes. In general, LMN lesions have impairment of the reflex arcs that control bowel and bladder functioning and

sexual functioning. These individuals will have flaccid bowel and bladder functioning, which results in much greater difficulty controlling bowel and bladder incontinence. In general, erectile function in the male is impaired.

Individuals with intact reflex voiding mechanisms of the bladder and the bowel may experience less complications, infection, and incontinent episodes.

To view table 1 “Muscles Supplied and Functions Served by Spinal Nerve Motor Roots”, click [here](#) (please see attached pdf file- SCI- Lesson 1- “Muscles Supplied and Functions Served by Spinal Nerve Motor Roots”)

Works Cited:

American Spinal Injury Association, 1992.

Asher, Sir R.A.: On the Dangers of Going to Bed. *British Medical Journal*, 1947; December 13; 967-68.

Gibson, C.J. Overview of Spinal Cord Injury. *Physical Medicine and Rehabilitation Clinics of North America*. 1992; 3: 699-709. W.B. Saunders.

Go, Bette K. et al.: The Epidemiology of Spinal Cord Injury. *Spinal Cord Injury Clinical Outcomes from the Model Systems*. Editor Samuel L. Stover, Joel A. DeLisa, Gale G. Whiteneck. Aspen Gaithersburg 1995.

Griffin, M.R., et al.: Traumatic Spinal Cord Injury in Omstead County, Minnesota, 1935 through 1981. *American Journal of Epidemiology*, 1985; 121: 884.

Krause, J.F., et al.: Incidence of Spinal Cord Lesion. *Journal of Chronic Disease* 28: 471, 1975.

Lawrence D.W., et al.: Traumatic Spinal Cord Injury in Louisiana: 1990 Annual report. New Orleans, Louisiana: Louisiana Office of Public Health; 1992.

Roth, E.J. et al. The Older Adult with a Spinal Cord Injury. *Paraplegia*, 1992; 30:520-26.

Schoenen, Jean: Clinical Anatomy of the Spinal Cord. *Disorders of the Spinal Cord, Neurological Clinics*. Editor Robert M. Wozzly and Robert R. Young, Volume 9, #3, August 1991, W.B. Saunders, Philadelphia.

Stass, William E. Jr., et al.: *Rehabilitation Medicines, Principles and Practice*. 2nd Edition, edited by Joel A. DeLisa. J.B. Lippincott Company, Philadelphia, 1993.

Stolov, W.C.: Clower, M.R. *Handbook of Severe Disabilities*, US Department of Education Rehabilitation Services Administration, 1981, p.67.

Zigler, Jack E. and B. Field. *Surgical Procedures for Spinal Cord Injury. Traumatic Spinal Cord Injury*. *Physical Medicine and Rehabilitation Clinics of North America*. Volume 3.4, November 1992.