CHAPTER 1
INTRODUCTION

1.0 INTRODUCTION

1.1 DEFINITION OF SPINAL CORD INJURY (SCI):

WHO defines Spinal Cord Injury (SCI) as: "Any injury to the spinal cord from traumatic and non-traumatic causes. Injury to the spinal cord that is caused by trauma or damage resulting from the application of an external force of any magnitude, e.g. in the event of road traffic crashes, falls or acts of violence, is known as Traumatic Spinal Cord Injury (TSCI)".

Damage to the spinal cord from a non-traumatic cause, e.g. congenital/genetic malformation such as spina bifida or acquired damage caused by infection, loss of blood supply (infarction), compression by a cancer or tumor, or by slow degeneration of the vertebrae because of osteoarthritis, is known as Non-traumatic Spinal Cord Injury (NTSCI). Damage or trauma to the spinal cord results in an impairment or loss of function."

SCI is an insult to the spinal cord resulting in a change, either temporary or permanent, in its motor, sensory, or autonomic function.SCI affects conduction of sensory and motor signals across the site(s) of lesion(s), as well as the autonomic nervous system. By systematically examining the dermatomes and myotomes, one can determine the cord segments affected by the SCI (Kirshblum et al., 2011).

The following terminology has developed around the classification of SCI:

- ***Tetraplegia*** (replaces the term Quadriplegia) - Injury to the spinal cord at the cervical region with associated loss of muscle strength in all 4 extremities.
- ***Paraplegia*** – Injury in the spinal cord in the thoracic, lumbar, or sacral segments, including the cauda equine and conus medullaris.

Definitions of complete and incomplete SCI are based on the above ASIA definition with sacral-sparing (Ditunno, et al., 1994).

- ***Complete***–Absence of sensory and motor functions in the lowest sacral segments
- ***Incomplete***–Preservation of sensory or motor function below the level of injury including the lowest sacral segments.

Sacral-sparing is evidence of the physiologic continuity of spinal cord long tract fibers (with the sacral fibers located more at the periphery of the cord). Indication of the presence of sacral fibers is of significance in defining the completeness of the injury and the potential for some motor recovery. These findings tend to be repeated and better defined after the period of spinal shock.

1.2 MECHANISM OF INJURY

Various mechanisms, often in combination, produce injuries to the spinal cord. SCI most frequently occurs from indirect forces produced by movement of the head and trunk and less often from direct injury to a vertebra. Common mechanisms operating in SCI include flexion, compression, hyper-extension, and flexion- rotation. These forces result in either a fracture and/or dislocation. The intensity and combination of forces imposed have direct influence on the type and location of fractures, the amount of dislocation, and the extent of soft tissue damage.

Table 1: Mechanism of spinal cord injury (SCI).

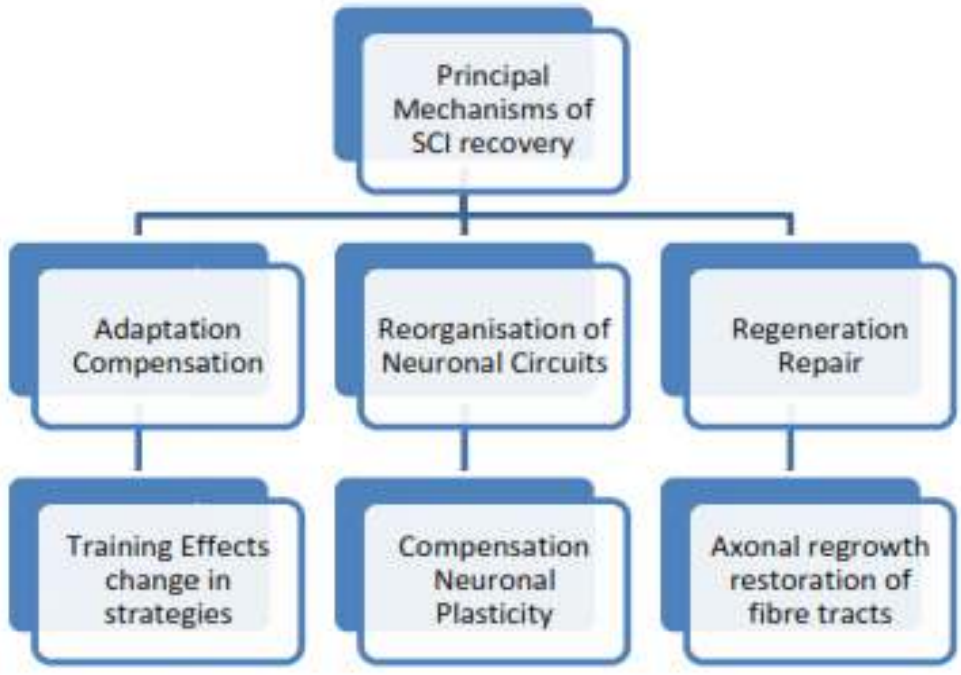
FORCE	ETIOLOGY	ASSOCIATED FRACTURES	POTENTIAL ASSOCIATED INJURIES
Flexion	1. Head-on collision in which head strikes steering wheel or wind- shield. 2. Blow to back of head or trunk. 3. Most common mechanism of SCI.	1. Wedge fracture of anteriorvertebral body (vertebral body compressed). 2. High percentage of injuries occurs from C4 to C7 and from T12 to L2.	1. Tearing of posterior ligaments. 2. Fractures of posterior elements: spinous processes, laminae, or pedicles. 3. Disruption of Disc. 4. Anterior dislocation of vertebral
Compression	1. Vertibral or axial blow head (diving, surfing or falling objects). 2. Closely associated with flexion injuries.	1. Concave fracture of endplate. 2. Explosion or burst fracture (comminuted)	1. Bone fragments may lodge incord. 2. Rupture of disc.
Hyper-extension	1. Strong posterior forcesuch as rear-end collision. 2. Falls with stationary object (more commonly seen in elderly populations).	1. Fractures of posteriorelements: spinous processes, laminae, and facets. 2. Avulsion fracture of anterior aspect of vertebrae.	1. Rupture of anterior longitudinal ligament. 2. Rupture ofdisc. 3. Associated with cervical lesions: only of minor influence in thoraco-lumbar injuries.
Flexion-rotation	1. Posterior to anterior force directed at rotated vertebral column (e.g. rear-end collision with passenger rotated toward driver).	1. Fracture of posterior pedicles, particular facets, and laminae (fracture is very unstable if posterior ligaments rupture).	1. Rupture of posterior and inter-spinous ligament. 2. Subluxation or dislocation of facet joints. 3. In thoracic and lumbarregions, facets may "lock".

The spine demonstrates various degrees of susceptibility to injury. Some areas are inherently more vulnerable because of their high mobility and relative lack of stability as compared with other segments of the spine (e.g. the rigid thoracic region). The areas of the spine that demonstrate the highest frequency of injury are between C5 and C7 in the cervical region and between T12 and L2 in the thoraco-lumbar region.

Mechanisms of recovery of SCI

The mechanisms involved in the recovery of motor and sensory function after SCI may include different approaches. Experimental approaches to enhance motor function after SCI are typically based on protecting neural tissue, promoting regeneration, and/or facilitating plasticity. Many of these approaches elicit anatomical and physiological changes in the spinal cord or brain and some of them result in enhanced motor and sensory performance (Dietz & Colombo, 2004) illustrated in the Figure 1.

Figure 1: Mechanisms of recovery of SCI[Source by: (Dietz & Colombo, 2004)].



1.3 BURDEN OF SCI

1.3.1 Global prevalence

SCI leads to immense economic burden on the country's health care system (B. B. Lee, Cripps, Fitzharris, & Wing, 2013; Singh, Tetreault, Kalsi-Ryan, Nouri, & Fehlings, 2014). The true impact of SCI can be reflected through the average prevalence rate of 1:1000, and the mean incidence estimated to be between 4 and 9 cases per 100,000 populations per year, worldwide (Thietje, 2017). It was also reported that the gender ratio in traumatic SCI is 3:1 (men: women), whereas gender is equally distributed in non-traumatic SCI (Lee et al., 2013).A survey by Cripps et al., (2011) shows that the incidence, prevalence, and causation of SCI varies among developing and developed nations and proposes that administration and preventative methodologies should be customized to provincial patterns as demonstrated in Table 2.

Table 2: Prevalence of traumatic spinal cord injury by Region and Author(s) of published data [Source by: (Cripps et al., 2011)].

Region	***Country***	***Author(s) of published data***	***Observation period***	***Prevalence per million population***
Asia, South	India (Kashmir)	Razdan	1986	236
Asia, South east-asia	Viet Nam	Weerts	2006–2007	464
Australia				
	Australia	Walsh	1987	370
	Australia	O'Connor	1997	681
	Australia	Yeo	1998	540 (Estimate)
Europe, Western				
	Finland (Helsinki)	Dahlberg	1999	280
	Iceland	Knutsdottir	1973-1989	316
	Norway (Western)	Hagen	2002	365
North America, High Income				
	Canada	Rick Hansen SCI Register	2001–2002	Approximately 1173/million (assuming a population of30.7 million)
	USA	Kurtzke	1971	800
	USA	Jackson	1973–2003	–
	USA	DeVivo	1980	906 (Approx.)
	USA	Ergas	1984	1009
	USA	Stover	1984	906
	USA	Harvey	1988	721
	USA	Ditunno	1993	Est. 776 (Estimate)
	USA	Lasfargues	1994	790
	USA	Rick Hansen SCI Register	2006	*253 000* (Individuals, estimate)
	USA (Olmsted County, Minnesota)	Griffin	1935–1981	473

1.3.2 Indian prevalence

In the Indian setup, as in most developing countries, very little is known about the exact incidence of SCI as there is no national database. In India, approximately 1.5 million people live with SCI. Approximate 20,000 new cases of SCI are added every year and 60-70% of them are illiterate, poor villagers. Majority of them are males in the age group of 16-30 years, signifying higher incidence in young, active and productive population of the society. There has been substantial decrease in male female ratio from the past which reflects changing face of social norms where females are becoming more active and outgoing in the modern era. In India fall from height rates highest among the etiological factors. There is a gradual trend towards increasing incidence of road traffic accidents indicating gradual urbanization of society and increase in number of vehicles on roads in India. Seasonal distribution of SCI shows a marked increase during summer, signifying increased movement of people in this season. To conclude, in India, management and rehabilitation of patients with SCI lags far behind. Rescue and retrieval systems for these patients are inadequate. There are few specialized centers for the management of such patents. The frequency of decubitus ulcers and UTI is unacceptably high. There is a strong need to identify the risk factors and to take steps to control them by disseminating information to masses, to train paramedical staff in rural areas about initial handling and transportation of patient shaving spinal cord injuries. A comprehensive multidisciplinary management and rehabilitation approach is the need of the hour to reintegrate patients with SCI to the community (Singh, 2012).

1.3.3 Economic burden of SCI

Historically, SCI has been associated with very high mortality rates. Yet today, in high-income countries, SCI can be viewed less as the end of a worthwhile or productive life

and more as a personal and social challenge that can be successfully overcome. This change reflects better medical provision, which means that people are able to survive, live and flourish after injury. For instance, people who develop SCI can now usually benefit from improved emergency response, effective health and rehabilitation interventions and technologies such as respirators and appropriate wheelchairs, together with more extensive social services and more accessible environments. As a result, lives can be saved and functioning can be maximized. Many people with SCI can now anticipate not just a longer life, but also a fuller and more productive lifethan they would have had in previous generations. In low-income countries the situation is very different. Traumatic SCI often remains a terminal condition. Poverty makes life even harder for people with SCI. Yet the fact that such dramatic progress in survival and participation has been seen in high-income countries over a relatively short period of time should be a reason to be optimistic for other parts of the world. With the right policy responses, it should be possible to live, thrive and contribute with SCI anywhere in the world. The quality of life with SCI depends greatly on whether the environment is facilitating – appropriate resources and services are available, there are supportive relationships and community inclusion – or whether it acts as a barrier when people have to confront discriminatory attitudes and other obstacles, including the failure to provide supportive and facilitating services and resources. In principle, an individual with SCI will experience nearly every clinical setting that his or her country provides: emergency services, intensive care, surgery, stabilizing medical careand particularly rehabilitation, including return to the community, vocational rehabilitation and ongoing primary care. SCI care thus provides evidence about the adequacy of a country's services, systems and policies (Jerome, Bickenbach, 2013).

1.3.4 SCI as social burden

Traumatic spinal cord injury (TSCI) is a catastrophic event that is sudden and unexpected and can be devastating and costly in human and social terms (Lee, Cripps, Fitzharris, Wing, 2014). Spinal cord injury (SCI) results in disturbances to normal sensory, motor or autonomic function and ultimately impacts a patient's physical, psychological, and social well-being. The management of SCIs requires significant health care resources and can place a substantial financial burden on patients, their families, and the community (Sekhon, Fehlings, 2001). Varying injury level and severity generate a spectrum of neurological dysfunction and reduction in long-term QOL (Iono-Morin, Noonan, White, et al, 2017).Another survey demonstrates that epidemiology of SCI seems to have changed during the last decades with a higher percentage of tetraplegia and of complete lesions (Wyndaele& Wyndaele, 2006), which is represented in Table 3.

Table 3: Epidemiology of spinal cord injury, literature data [Source by: (Wyndaele & Wyndaele, 2006)].

References	*Paraplegia* (%)	*Tetraplegia* (%)	*Complete* (%)	*Incomplete* (%)	*Age (years)*	*Men/women*
Kurtzke,1975	86.40	13.60	40.00	60.00	15–34	5.0/1
Tricot,1981	42.68–91.3	8.7–57.32			38.2	4.6/1
van Asbeck *et al*,1994	43.00	57.00	48.70	51.30		3.0/1
Maharaj, 1996	69.00	31.00	52.10	47.90	16–30: 35%	4.0/1
Dahlberg *et al*, 2005	54.00	46.00	43.00	57.00	31.00	3.0/1
Karacan *et al*,2000	67.80	32.18			35.5±15.1	2.5/1
Karamehmetoglu *et al*,1995	67.00	33.00			33.00	3.0/1
Karamehmetoglu *et al*, 1997	58.70	41.30			31.3	5.8/1
Chen *et al*,1997					46.1	3.0/1
Martins *et al,* 1998					50.00	3.0/1
Surkin *et al,* 2000						4.4/1

1.4 DIAGNOSIS OF SCI

At the beginning of the injury the crisis doctor looks at the person to check whether there is any movement or sensation at or below the level of injury. Strategies to survey autonomic function likewise have been set up (American Spinal Injury Association, or ASIA, Autonomic Standards Classification). Diagnostic medicinal tests for SCI include:

- **Magnetic resonance imaging (MRI):** uses computer-generated radio waves and a powerful magnetic field to produce detailed three-dimensional images of body structures, including tissues, organs, bones, and nerves. It can record brain and spinal injury from trauma, just as help in diagnosing brain and spinal cord tumors, herniated disks, vascular (blood vessel) irregularities, bleeding and inflammation that might compress the spine and spinal cord, and injury to the ligaments that support the cervical spine.

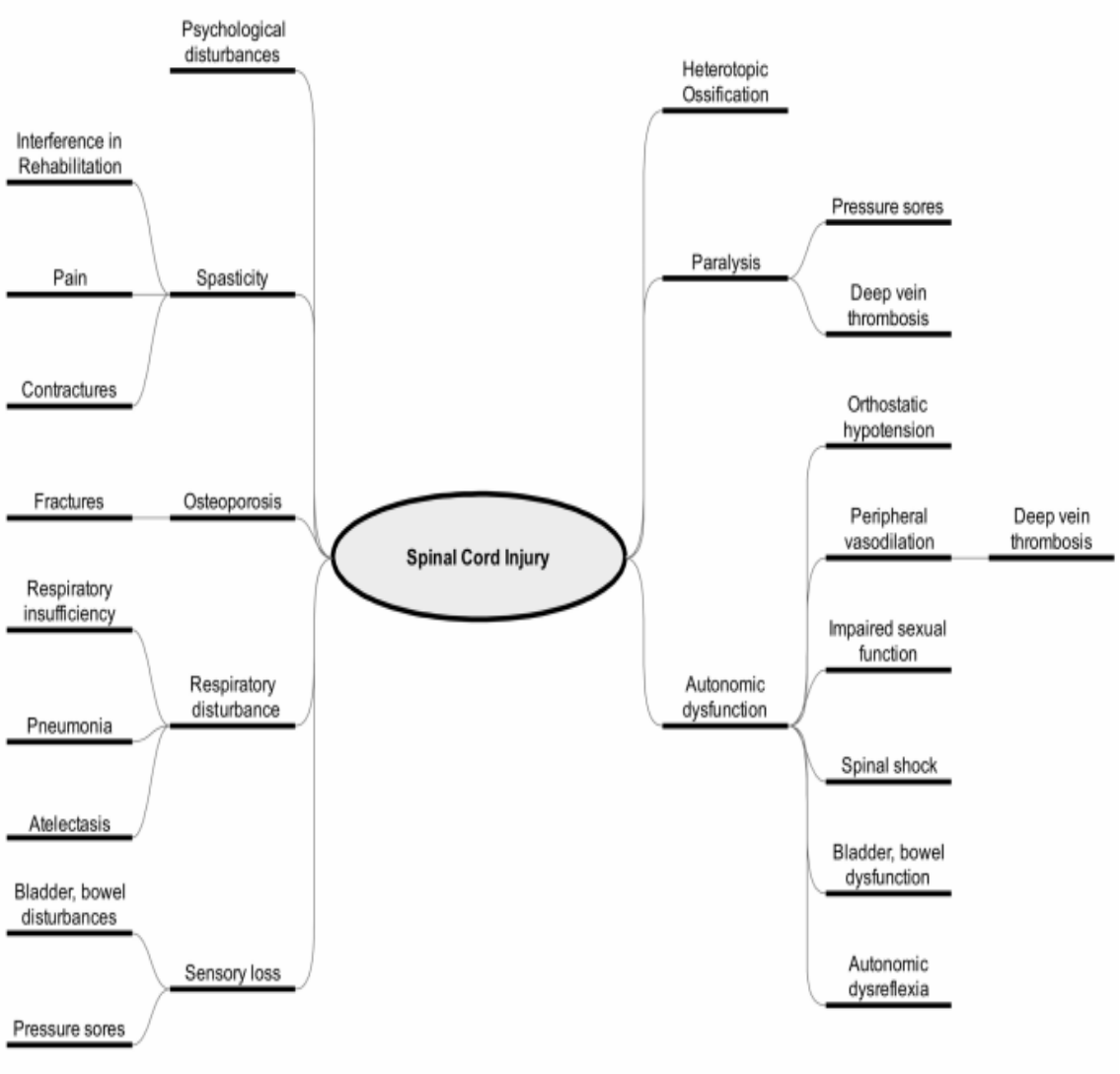
- **Computerized tomography (CT)**: gives fast, clear two-dimensional x-ray images of organs, bones, and tissues. Neurological CT scans are utilized to see the brain and spine. CT is amazing in bone breaks, dying, and spinal stenosis (narrowing of the spinal trench), yet CT has less capacity to picture the spinal cord or recognize ligament damage related with a unstable spine than MRI.

- **Plane X-rays**: chest and skull X-rays are often taken as part of a neurological assessment. X-rays can be utilized to see most parts of the body, such as a joint or major organ system. Vertebral misalignment or fracture can be seen within minutes. X-rays taken in different neck positions (i.e., flexion and extension views) detect instability of the cervical spine. Tissue masses, for example,injured ligaments or a bulging disc are not visible on conventional x-rays.

- **Clinical Features**

  SCI is characterized by the following complications: Respiratory complications, Cardiovascular complications, Bowel and Bladder problems, Joints and limbs complications, The skin and pressure areas, etc.Figure 2 illustrates an overview of risk factors, signs, symptoms, prevention and treatment approaches for secondary long-term complications in patients with SCI (Dietz & Colombo, 2004).

Figure 2: Illustrating the Clinical Features of SCI[Source by: (Dietz & Colombo, 2004)].



- **Differential diagnosis**

  The principal differential diagnosis of SCI is head injury. Other major diagnosis areusually easier to differentiate from SCI, such as Pneumothorax/hemothorax and fractures(Do-Dai, Brooks, Goldkamp, Erbay, & Bhadelia, 2010).

1.5 CLASSIFICATION OF SCI

The International Standards for Neurological and Functional Classification of Spinal Cord Injury is a widely accepted system describing the level and extent of injury based on a systematic motor and sensory examination of neurologic function (Ditunno, Young, Donovan, & Creasey, 1994; Kirshblum et al., 2011).

1.5.1 Neurological Classification

American Spinal Injury Assessment (ASIA) impairment scale (modified from Frankel)

The following scale is used in grading the degree of impairment:

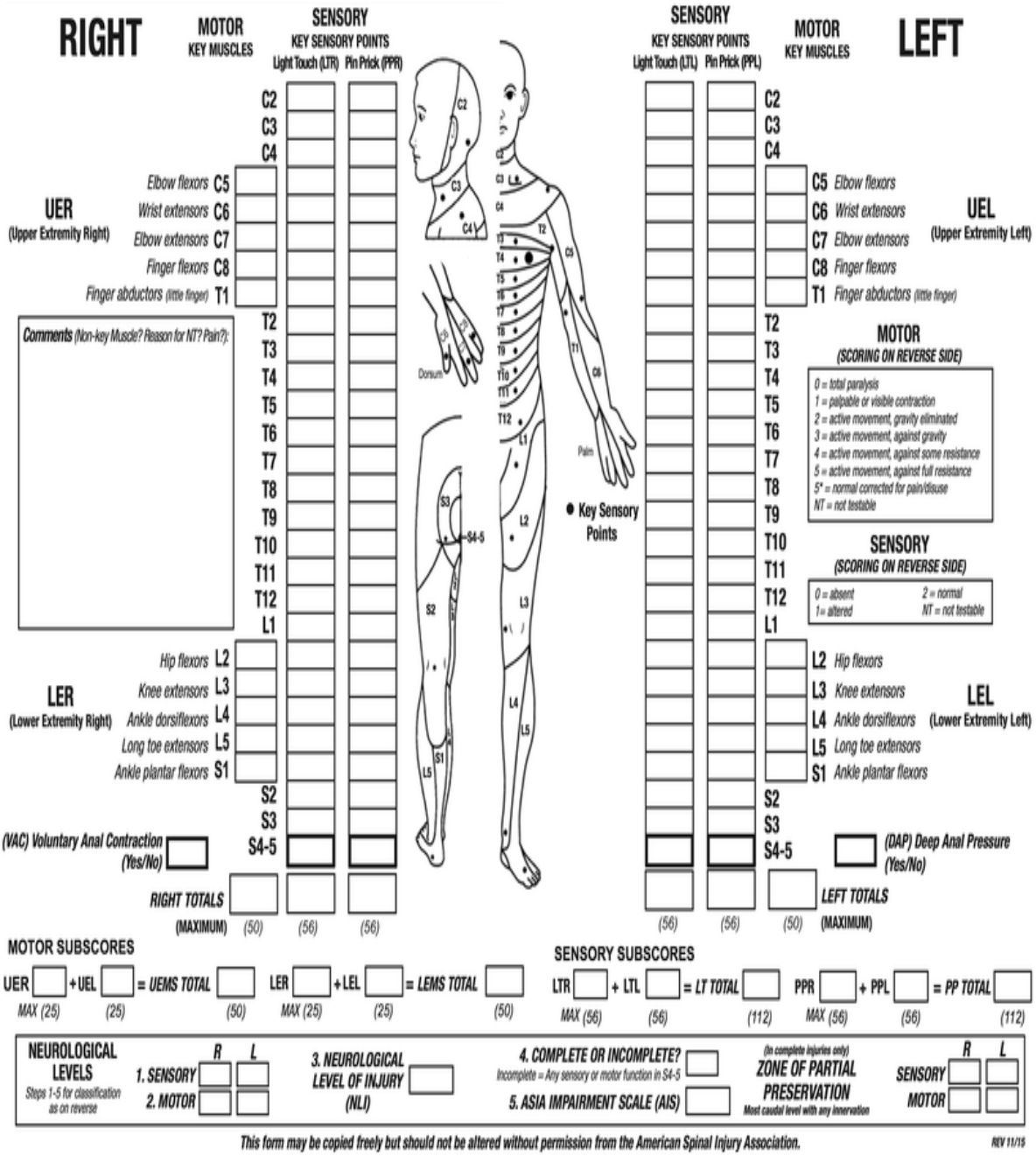
- **ASIA A** – COMPLETE: No motor or sensory function is preserved below the level of injury (and in the sacral segments S4 – S5).
- **ASIA B** – INCOMPLETE: Sensory but not motor function is preserved below the neurological level (includes the sacral segments S4 – S5).
- **ASIA C** – INCOMPLETE: Motor function is preserved below the neurological level, but too little to represent a practically usable function (more than half of key muscles below the neurological level have a muscle grade less than 3).
- **ASIA D** – INCOMPLETE: Motor function is preserved below the neurological level, to an extent that provides practically usable function (at least half of

key muscles below the neurological level have a muscle grade of 3 or more on a scale from 0 to 5).

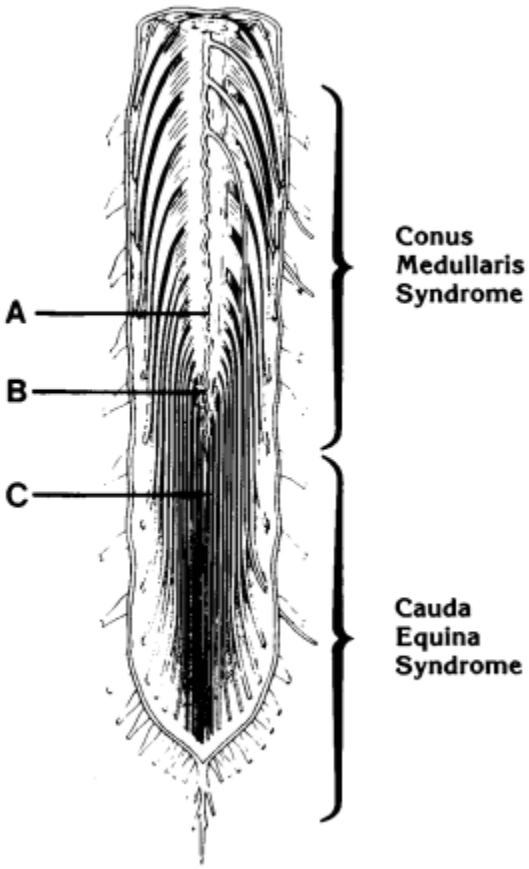
- **ASIA E** – Motor and sensory functions are normal.

ASIA A implies a complete injury, **ASIA B – D** describe incomplete injuries.

Figure3: ASIA scoring sheet for the neurological classification of the SCI {Source by: (Kirshblum et al., 2011)}.



1.5.2 Functional Classification



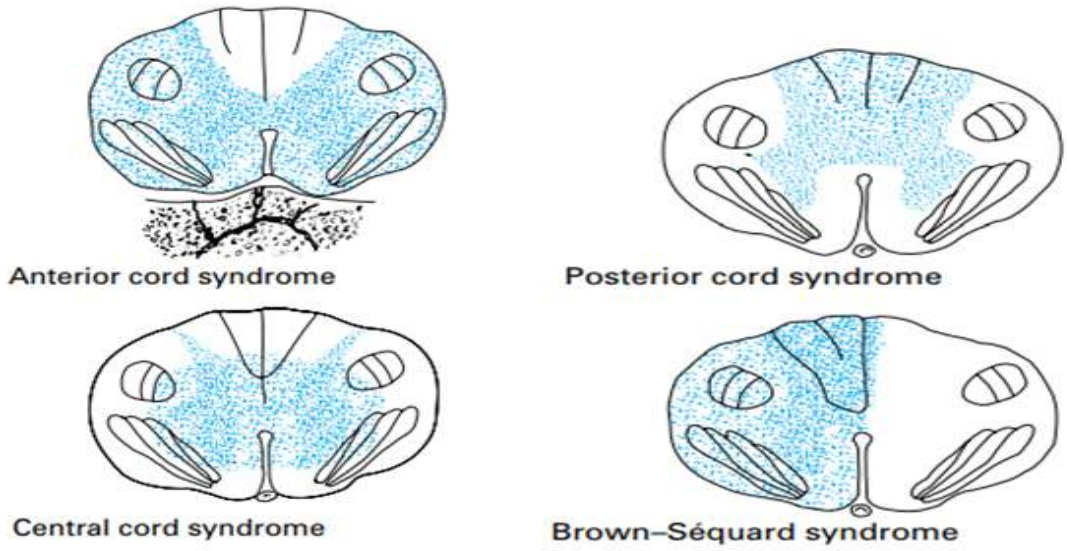
Clinical syndromes Central cord syndrome: A lesion, occurring almost exclusively in the cervical region, produces sacral sensory sparing and greater weakness in the upper limbs than in the lower limbs.

Brown-Sequard syndrome: A lesion that produces relatively greater ipsilateral proprioceptive and motor loss and contralateral loss of sensitivity to pain and temperature.

Anterior cord syndrome: A lesion that produces variable loss of motor function and of sensitivity to pain and temperature, while preserving proprioception.

Conus medullaris syndrome: Injury of the sacral cord (conus) and lumbar nerve roots within the spinal canal, which usually results in an areflexic bladder, bowel,and lower limbs, with lesions as at B in Figure. Sacral segments may occasionally show preserved reflexes, e.g., Bulbo-Cavernosus and micturition reflexes, with lesions as at A in Figure.

Cauda equina syndrome: Injury to the lumbosacral nerve roots within the neural canal resulting in the areflexic bladder, bowel, and lower limbs, with lesions as at C in Figure.



1.6 PATHOPHYSIOLOGY OF SCI

A portion of the pathophysiological events may not transiently overlap and can happen at different periods of SCI, which are portrayed here.Following primary injury, activation of occupant astrocytes and microglia and consequent penetration of blood-borne immunecells results in a strong neuroinflammatory reaction.This intense neuroinflammatory response assumes a key role in organizing the secondary injury mechanisms in the sub-acute and chronic stages that lead to cell death and tissue degeneration, just as the development of the glial scar, axonal degeneration, and demyelination.

Amid the acute stage, monocyte-derived macrophages possess the focal point of the damage to scavenge tissue debris. T and B lymphocytes additionally penetrate the spinal cord amid sub-acute stage and produce pro-inflammatory cytokines, chemokines, auto-antibodies reactive oxygen and nitrogen species that add to tissue degeneration. Then again, M2-like macrophages and regulatory T and B cells produce growth factors and pro-regenerative cytokines, for example, IL-10 that encourage tissue repair and wound mending. Loss of oligodendrocytes in acute and sub-acute phases of SCI prompts axonal

demyelination pursued by unconstrained remyelination in sub-acute and chronic stages. Amid the sub-acute and chronic periods of SCI; astrocytes, OPCs, and pericytes, which typically live in the spinal line parenchyma, multiply and relocate to the site of damage and add to the formation of the glial scar.

The glial scar and its related matrix encompass the injury epicenter and make a cellular and biochemical zone with both useful and impeding roles in the repair procedure. Acutely, the astrocytic glial scar restrains the spread of neuroinflammation from the injury site to the sound tissue. Nonetheless, the foundation of a mature longstanding glial scar and upregulation of matrix chondroitin sulfate proteoglycans (CSPGs) are supposed to repress axonal regeneration/sprouting and cell differentiation in sub-acute and perpetual stages(Norenberg, Smith, & Marcillo, 2004).

A schematic diagram illustrating the pathophysiology of traumatic spinal cord injury is shown in Figure 4 below.

Figure 4: Pathophysiology of traumatic spinal cord injury: This schematic diagram illustrates thecomposition of the normal and injured spinal cord [Source by: (Norenberg et al., 2004)**].**

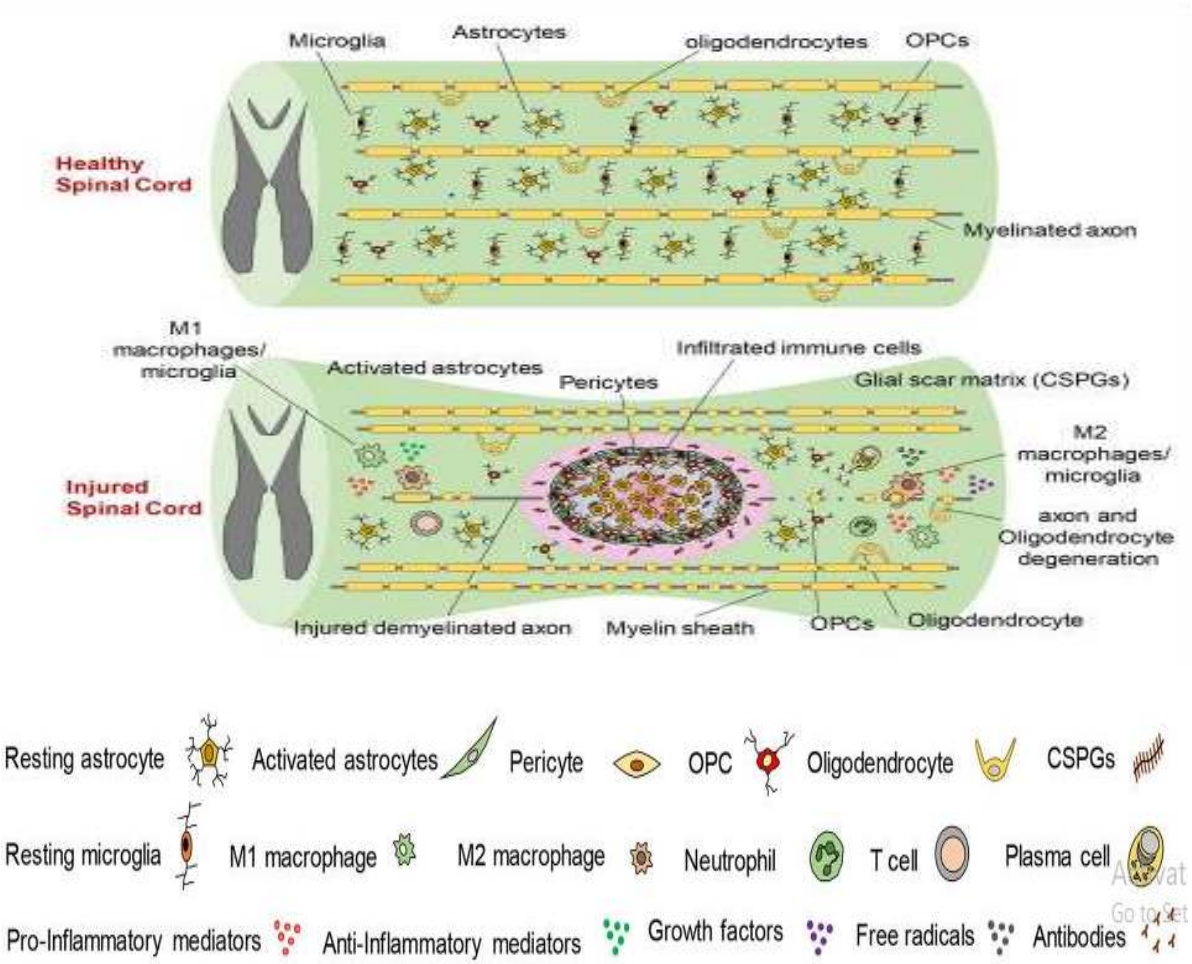


Figure 5: Summary of secondary injury processes following traumatic spinal cord injury [Source by: (Alizadeh, Dyck, & Karimi-Abdolrezaee, 2019)**].**

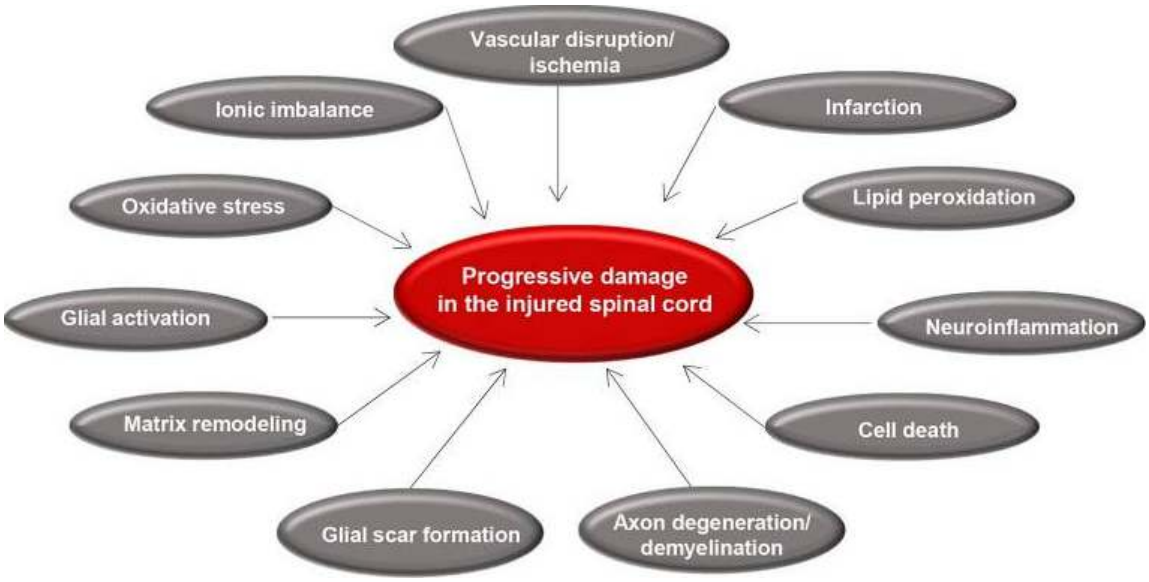


Figure 5 demonstrates the secondary damage events including pathophysiological phenomena that happen after primary injury and lead to dynamic tissue degeneration. Vascular disturbance and ischemia happen following primary injury that starts glial activation, neuroinflammation, and oxidative stress. These acute changes result in cell demise, axonal damage, matrix rebuilding, and production of a glial scar(Alizadeh et al., 2019).

Immune response in spinal cord injury: Under normal circumstances, there is a balance between pro-inflammatory effects of CD4+ effector T cells (T_{eff}) and anti-inflammatory effects of regulatory T and B cells (T_{reg} and B_{reg}). T_{reg} and B_{reg} suppress the activation of antigen-specific CD4+ T_{eff} cells through the production of IL-10 and TGF-β. Injury disrupts this balance and promotes a pro-inflammatory environment. Activated microglia/macrophages release pro-inflammatory cytokines and chemokines and present antigens to CD4+ T cells causing activation of antigen-specific effector T cells. T_{eff} cells stimulate antigen-specific B cells to undergo clonal expansion and produce autoantibodies

against spinal cord tissue antigens. These autoantibodies cause neurodegeneration through FcR mediated phagocytosis of complement-mediated cytotoxicity. M1 macrophages/microglia release pro-inflammatory cytokines and reactive oxygen species (ROS) that are detrimental to neurons and oligodendrocytes. B_{reg} cells possess the ability to promote T_{reg} development and restrict T_{eff} cell differentiation. B_{reg} cells could also induce apoptosis in T_{eff} cells through F_{as} mediate mechanisms (Alizadeh et al., 2019).

1.7 CONVENTIONAL MANAGEMENT OF SCI

For the most part, non-operative treatment was the standard. Regardless, continuous advances in radiological systems have incited a prevalent perception of damage designs. The significant issues to consider are the strength of the injured spinal column and neurological status. Instability may result in neural tissue injury, progressive deformity, and ceaseless chronic pain. Advances in anesthesiology, critical care, surgical procedures, and instrumentations have supported forceful yet more secure strategies for spinal fixation. Surgical fixation, where indicated, gives unrivaled control of deformation, permits early mobilization, recovery and diminishes hospital stay. Bethat as it may, an accord on the role of surgery and its planning stays subtle. Its job in improving the neurological results is questionable. The main indication for surgery is a progressive neurological deficiency. Figure 7 illustrates guideline which provides evidence-based recommendations for the optimal type and timing of rehabilitation in patients with acute SCI(Dietz & Colombo, 2004).

Figure 6: Immune response in spinal cord injury [Source by:(Alizadeh et al., 2019)].

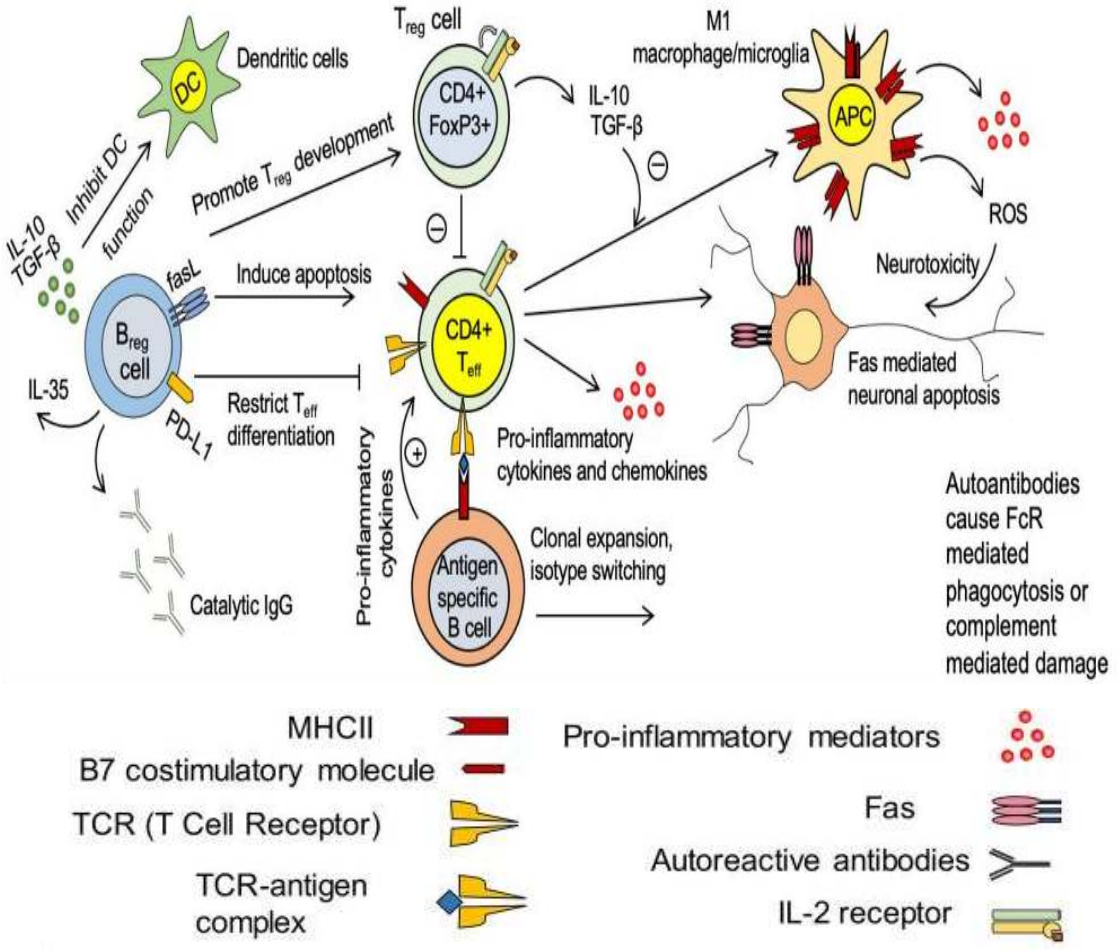
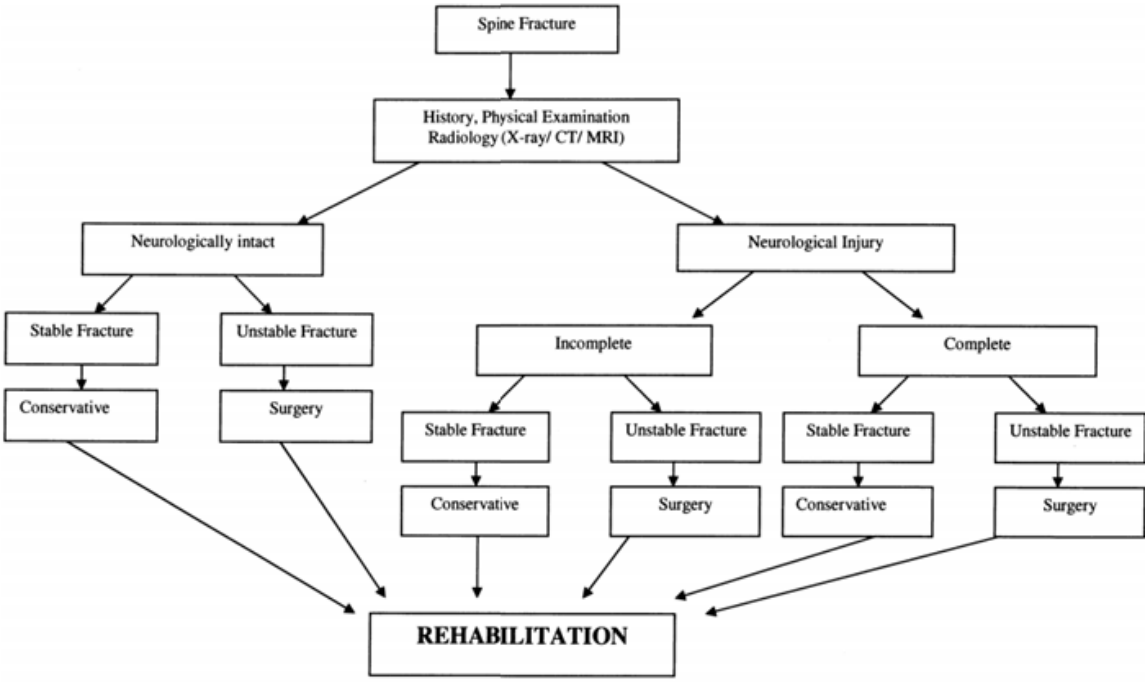


Figure 7: Conventional Management of SCI [Source by:(Dietz & Colombo, 2004)].



1.8 YOGA FOR SCI

A review reported that psychopharmacology is a foundation treatment method for the management of neurological and psychiatric disorders. But they are associated with metabolic side effects such as weight gain, diabetes, and dyslipidemias, and cardiovascular and sexual dysfunction. Also, the therapeutic benefit of these medications is often inadequate (Meyer et al., 2012). Various other studies have proved that yoga and physical therapy (PT) interventions have enhanced recovery in various neuropsychiatric illnesses (Bhargav, Nagendra, Gangadhar, & Nagarathna, 2014).Yoga is a form of mind-body intervention comprised of adaptation of specific body poses or *asanas,* breath control, and meditation and it is also a way of life which helps in bringing the harmony at physical, physiological, mental, social and spiritual aspects of individual (Williams et al., 2005).Other scientific studies reported that yoga improves spasticity, gait, and cognition, functional independence, mental health and QoL among patients with neurological disorders (Meyer et al., 2012). Yoga enhances motor and sensory function, ADL, gait, mental flexibility, psychological well-being and relaxation in individuals with SCI (Curtis et al., 2017; Telles et al., 2017; Zwick, 2006).

Furthermore, yoga has shown that incorporation of different yogic techniques into rehabilitation protocol of individuals with SCI, with proper guided assistance, is believed to stimulate neural pathways and neurotransmitters (Raju, 2017). This, in turn, can be valuable instrument in the regeneration of nerve fibers in SCI patients (Smith & Boser, 2013).

In a single case study, it was found that Iyengar yoga can easily be incorporated into an exercise program of an individual with SCI and practiced over the years has been shown to improve muscle strength, flexibility, coordination and proprioception through

stretching of the muscles (Zwick, 2006). However, a large number of studies recommended RCTs to assess the impact of yoga in SCI (Curtis et al., 2017).

1.9 NEED OF THE STUDY

Strategies that seek out the complementary effects of combination treatments and that efficiently integrate relevant technical advances in biomechanics represent an untapped potential and are likely to have an immediate impact. There are no published randomized control trials to date available to explore the efficacy of combination of complementary alternative therapies with conventional mainstream rehabilitation treatments in the management of SCI. Thus, there is a clear need for complementary, non-psycho-pharmacological therapy such as yoga which can be an effective adjunctive treatment option for neurological and psychiatric disorders. Herein, we want to compare the effect of add-on of Yoga to Physiotherapy in the management of spinal cord injury patients.