Spinal cord injury (SCI) is an insult to the spinal cord resulting in a change, either temporary or permanent, in its normal motor, sensory, or autonomic function. Patients with spinal cord injury usually have permanent and often devastating neurologic deficits and disability. In all acute cord syndromes, the full extent of injury may not be apparent initially. Incomplete cord lesions may evolve into more complete lesions. More commonly, the injury level raises 1 or 2 spinal levels during the hours to days after the initial event.

A complex cascade of pathophysiologic events related to free radicals, cacogenic enema and altered blood flow accounts for this clinical deterioration. Normal oxygenation, perfusion, and acid-base balance are required to prevent worsening of the spinal cord injury.

Spinal cord injury can be sustained through different mechanisms, with the following 3 common abnormalities leading to tissue damage:

- Destruction from direct trauma
- Compression by bone fragments, hematoma, or disk material
- Ischemia from damage or impingement on the spinal arteries

Enema could ensue subsequent to any of these types of damage.

**Neurogenic shock**

Neurogenic shock characterised by the hemodynamic triad of hypotension, bradycardia, and peripheral vasodilation resulting from severe autonomic dysfunction and the interruption of sympathetic nervous system control in acute spinal cord injury. Hypothermia is also associated with it. This condition does not usually occur with spinal cord injury below the level of T6 but is more common in injuries above T6, secondary to the disruption of the sympathetic outflow from T1-L2 and to unopposed vagal tone, leading to a decrease in vascular resistance, with the associated vascular dilatation. Neurogenic shock needs to be differentiated from spinal and hypovolumic shock. Hypovolumic shock tends to be associated with tachycardia.
Spinal shock

Shock associated with a spinal cord injury involving the lower thoracic cord must be considered hemorrhagic until proven otherwise. In this article, spinal shock is defined as the complete loss of all neurologic function, including reflexes and rectal tone, below a specific level that is associated with autonomic dysfunction. That is, spinal shock is a state of transient physiologic (rather than anatomic) reflex depression of cord function below the level of injury, with associated loss of all sensorimotor functions.

An initial increase in blood pressure due to the release of catecholamines, followed by hypotension, is noted. Flaccid paralysis, including of the bowel and bladder, is observed, and sometimes sustained priapism develops. These symptoms tend to last several hours to days until the reflex arcs below the level of the injury begin to function again (e.g., bulbocavernosus reflex, muscle stretch reflex [MSR]).

Spinal cord injuries may be primary fracture and/or dislocation of the spine. Primary spinal cord injuries arise from mechanical disruption, transaction, or distraction of neural elements. This injury usually occurs with fracture and/or dislocation of the spine. However, primary spinal cord injury may occur in the absence of spinal fracture or dislocation. Penetrating injuries due to bullets or weapons may also cause primary spinal cord injury. More commonly, displaced bony fragments cause penetrating spinal cord and/or segmental spinal nerve injuries.

Extradural pathology may also cause a primary spinal cord injury. Spinal epidural hematomas or abscesses cause acute cord compression and injury. Spinal cord compression from metastatic disease is a common oncologic emergency.

Longitudinal distraction with or without flexion and/or extension of the vertebral column may result in primary spinal cord injury without spinal fracture or dislocation. The spinal cord is tethered more securely than the vertebral column. Longitudinal distraction of the spinal cord with or without flexion and/or extension of the vertebral column may result in spinal cord injury without radiologic abnormality (SCIWORA).

SCIWORA was first coined in 1982 by Pang and Wilberger. Originally, it referred to spinal cord injury without radiographic or computed tomography (CT) scanning evidence of fracture or dislocation. However with the advent of magnetic resonance imaging (MRI), the term has become ambiguous. Findings on MRI such as intervertebral disk rupture, spinal
epidural hematoma, cord contusion, and hematomyelia have all been recognized as causing primary or secondary spinal cord injury. SCIWORA should now be more correctly renamed as "spinal cord injury without neuroimaging abnormality" and recognize that its prognosis is actually better than patients with spinal cord injury and radiologic evidence of traumatic injury.

Secondary- Vascular injury to the spinal cord caused by arterial disruption, arterial thrombosis, or hypoperfusion due to shock are the major causes of secondary spinal cord injury.

Complete vs. incomplete spinal cord syndrome

Determine first absence or presence of sacral sparing. If anyone underlying sign is present injury is called incomplete voluntary anal contraction

4-5 sensory scores = 1 or 2 at least in one side

Deep anal pressure

Voluntary anal contraction

Incomplete SCI injuries are follows-

The anterior cord syndrome

The Brown-Séquard syndrome.

The central cord syndrome

Conus medullaris syndrome, cauda equina syndrome.

Anterior cord syndrome involves a lesion causing variable loss of motor function and pain and/or temperature sensation, with preservation of proprioception.

Brown-Séquard syndrome, which is often associated with a hemisection lesion of the cord, involves a relatively greater ipsilateral loss of proprioception and motor function, with contralateral loss of pain and temperature sensation.

Central cord syndrome usually involves a cervical lesion, with greater motor weakness in the upper extremities than in the lower extremities, with sacral sensory sparing. The pattern of motor weakness shows greater distal involvement in the affected extremity than proximal
muscle weakness. Sensory loss is variable, and the patient is more likely to lose pain and/or temperature sensation than proprioception and/or vibration. Dysesthesias,

Conus medullaris syndrome is a sacral cord injury, with or without involvement of the lumbar nerve roots. This syndrome is characterized by areflexia in the bladder, bowel, and to a lesser degree, lower limbs, whereas the sacral segments occasionally may show preserved reflexes (e.g., bulbocavernosus and micturition reflexes). Motor and sensory loss in the lower limbs is variable.

Cauda equina syndrome involves injury to the lumbosacral nerve roots in the spinal canal and is characterized by an areflexic bowel and/or bladder, with variable motor and sensory loss in the lower limbs. Because this syndrome is a nerve root injury rather than a true spinal cord injury, the affected limbs are areflexic. Cauda equina syndrome is usually caused by a central lumbar disk herniation.

Spinal cord concussion is characterized by a transient neurologic deficit localized to the spinal cord that fully recovers without any apparent structural damage.

Life expectancy and mortality

Approximately 10-20% of patients who have sustained a spinal cord injury do not survive to reach acute hospitalization, whereas about 3% of patients die during acute hospitalization.

Originally the leading cause of death in patients with spinal cord injury who survived their initial injury was renal failure, but, currently, the leading causes of death are pneumonia, pulmonary embolism, or septicemia. Heart disease, subsequent trauma, suicide, and alcohol-related deaths are also major causes of death in these patients. In persons with spinal cord injury, the suicide rate is higher among individuals who are younger than 25 years.

Among patients with incomplete paraplegia, the leading causes of death are cancer and suicide (1:1 ratio), whereas among persons with complete paraplegia, the leading cause of death is suicide, followed by heart disease.

Life expectancies for patients with spinal cord injury continue to increase but are still below the general population. Patients aged 20 years at the time they sustain these injuries have a life expectancy of approximately 35.7 years (patients with high tetraplegia [C1-C4]), 40 years (patients with low tetraplegia [C5-C8]), or 45.2 years (patients with paraplegia). Individuals aged 60 years at the time of injury have a life expectancy of approximately 7.7 years (patients
with high tetraplegia), 9.9 years (patients with low tetraplegia), and 12.8 years (patients with paraplegia).

A 2006 study by Strauss and colleagues reported that among patients with spinal cord injury, during the critical first 2 years following injury, a 40% decline in mortality occurred between 1973 and 2004. During that same 31-year period, there had been only a small, statistically insignificant reduction in mortality in the post 2-year period for these patients.

Neurological Examination

Cervical spine There are 8 nerve roots. Cervical roots of C1-C7 are named according to the vertebra above which they exit (i.e. C1 exits above the C1 vertebra, just below the skull and C6 nerve roots pass between the C5 and C6 vertebrae) whereas C8 exists between the C7 and T1 vertebrae; as there is no C8 vertebra. The C1 nerve root does not have a sensory component.

The thoracic spine has 12 distinct nerve roots and the lumbar spine consists of 5 distinct nerve roots that are each named accordingly as they exit below the level of the respective vertebrae. The sacrum consists of 5 embryonic sections that have fused into one bony structure with 5 distinct nerve roots that exit via the sacral foramina. The spinal cord itself ends at approximately the L1-2 vertebral level. The distal most part of the spinal cord is called the conus medullaris. The cauda equina is a cluster of paired (right and left) lumbosacral nerve roots that originate in the region of the conus medullaris and travel down through the theca sac and exit via the intervertebral foramen below their respective vertebral levels.

Coccygeal nerves may be 0, 1, or 2 which is not used with the International Standards examination in accordance with the International Standards for Neurological Classification of Spinal Cord Injury (ISNCSCI).

Tetraparesis and paraparesis: Use of these terms is discouraged, as they describe incomplete lesions imprecisely.

Dermatome: This term defined as the area of the skin innervated by the sensory axons within each segmental nerve (root).

Myotome: This term defined as the collection of muscle fibbers innervated by the motor axons within each segmental nerve (root).
Skeletal level: This term has been used to denote the level at which, by radiographic examination, the greatest vertebral damage is found. The skeletal level is not part of the current ISNCSCI because not all cases of Achieve a bony injury, bony injuries do not consistently correlate with the neurological injury to the spinal cord.

Light touch sensation is tested with a tapered wisp of cotton stroked once across an area not to exceed 1cm of skin with the eyes closed or vision blocked.

3-pont scale is used to test touch sensation

0 = absent

1 = altered (impaired or partial appreciation, including hyperesthesia)

2 = normal or intact (similar as on the cheek)

NT = not testable

Pin prick sensation (sharp/dull discrimination) is performed with a disposable safety pin that is stretched apart to allow testing on both ends; using the pointed end to test for sharp and the rounded end of the pin for dull.

3-pont scale is

Grade 0. The inability to distinguish between dullard sharp sensation (as well as no feeling when being touched by the pin)

Grade 1 distinguishes between the sharp and dull ends of the pin but the intensity of sharpness is different in the key sensory point than the feeling of sharpness on the face. The intensity may be greater or lesser than the feeling on the face.

Grade-2 distinguishes between the sharp and dull ends of the pin but the intensity of sharpness is same on the face...

Sensory key points

C2 – At least 1 cm lateral to the occipital protuberance (alternatively 3 cm behind the ear)

C3 – Supraclavicular fossa (posterior to the clavicle) and at the midclavicular line

C4 – Over the acromioclavicular joint
C5 – Lateral (radial) side of the antecubital fossa (just proximal to elbow crease)

C6 – Thumb, dorsal surface, proximal phalanx

C7 – Middle finger, dorsal surface, proximal phalanx

C8 – Little finger, dorsal surface, proximal phalanx

T1 – Medial (ulnar) side of the antecubital fossa, just proximal to the medial epicondyle of the humerus

T2 – Apex of the axilla

T3 – Midclavicular line and the third intercostal space (IS) found by palpating the anterior chest to locate the third rib and the corresponding IS below it*

T4 – Fourth IS (nipple line) at the midclavicular line

T5 – Midclavicular line and the fifth IS (midway between T4 and T6)

T6 – Midclavicular line and the sixth IS (level of xiphisternum)

T7 – Midclavicular line and the seventh IS (midway between T6 and T8)

T8 – Midclavicular line and the eighth IS (midway between T6 and T10)

T9 – Midclavicular line and the ninth IS (midway between T8 and T10)

T10 – Midclavicular line and the tenth IS (umbilicus)

T11 – Midclavicular line and the eleventh IS (midway between T10 and T12)

T12 – Midclavicular line and the mid-point of the inguinal ligament

L1 – Midway distance between the key sensory points for T12 and L2

L2 – On the anterior-medial thigh at the midpoint drawn connecting the midpoint of inguinal ligament(T12) and the medial femoral condyle

L3 – Medial femoral condyle above the knee

L4 – Medial malleolus

L5 – Dorsum of the foot at the third metatarsal phalangeal joint
S1 – Lateral heel (calcaneus)

S2 – Midpoint of the popliteal fossa

S3 – Ischial tuberosity or infragluteal fold

S4–5 – Perianal area less than one cm. lateral to the muco cutaneous junction (taken as one level)
Deep Anal Pressure (DAP):

DAP awareness is examined through insertion of the examiner’s index finger and applying gentle pressure to the anorectal wall (innervated by the somatosensory components of the pudendal nerve (S4/5)). Alternatively, pressure can be applied by using the thumb to gently squeeze the anus against the inserted index finger. Consistently perceive pressure should be graded as being present or absent (i.e., enter Yes or No on the worksheet). Any reproducible pressure sensation felt in the anal area during this part of the exam signifies that the patient has a sensory incomplete lesion.

Optional sensation

Joint movement appreciation and position sense, A grade of 0 (absent) indicates the patient is unable to correctly report joint movement on large movements of the joint. A grade of 1 (impaired) indicates the patient is able to consistently report joint movement with 8 of 10 correct answers – but only on large movements of the joint and unable to consistently report small movements of the joint. A 2 (normal) indicates the patient is able to consistently report joint movement with 8 out of 10 correct answers on both small (approximately 10° of motion) and large movements of the joint. Joints that can be tested include the interphalangeal (IP) joint of the thumb, the proximal IP joint of the little finger, the wrist, the IP joint of the great toe, the ankle, and the knee.

Awareness of deep pressure/deep pain applying firm pressure to the skin for 3–5 seconds at different locations of the wrist, fingers, ankles and toes) can be tested for patients in whom light touch and pin prick modalities are graded as 0 (absent). Because this test is electively performed in the absence of light touch and pin prick sensation, it is graded as either a 0 for absent, or 1 for present, in reference to firm pressure, using the index finger or thumb, to the chin.
Motor examination:

10 key muscles 5 in upper limb and 5 in lower limb should be evaluated. Each key muscle function should be examined in a rostral-caudal sequence, utilizing standard supine positioning and stabilization of the individual muscles being tested.

C5 – Elbow flexors (biceps, brachialis)

C6 – Wrist extensors (extensor carpi radialis longus and brevis)

C7 – Elbow extensors (triceps)

C8 – Finger flexors (flexor digitorum profundus) to the middle finger

T1 – Small finger abductors (abductor digiti minimi)

L2 – Hip flexors (iliopsoas)

L3 – Knee extensors (quadriceps)

L4 – Ankle dorsiflexors (tibialis anterior)

L5 – Long toe extensors (extensor hallucis longus)

S1 – Ankle plantar flexors (gastrocnemius, soleus)

The strength of each muscle function is graded on a six-point scale

0 = total paralysis.

1 = palpable or visible contraction

2 = active movement, full range of motion (ROM) with gravity eliminated.

3 = active movement, full ROM against gravity.

4 = active movement, full ROM against gravity and moderate resistance in a muscle specific position.

5 = (normal) active movement, full ROM against gravity and full resistance in a muscle specific position expected from an otherwise unimpaired person.
5* = (normal) active movement, full ROM against gravity and sufficient resistance to be considered normal if identified inhibiting factors (i.e. pain, disuse) were not present.

NT= not testable (i.e. due to immobilization, severe pain such that the patient cannot be graded, amputation of limb, or contracture of >50% of the range of motion).

Voluntary anal contraction (VAC): The external anal sphincter (innervated by the somatic motor components of the pudendal nerve from S2-4) should be tested on the basis of reproducible voluntary contractions around the examiner’s finger and graded as being present or absent (i.e., enter YES or NO on the patient’s worksheet). Optional elements- non-key muscles may be evaluated; for example, the diaphragm, deltoid, finger extension, hip adductors and hamstrings the International Standards allows non-key muscles to determine motor incomplete status; AIS B versus C

Sensory Level: The sensory level is the most caudal, intact dermatome for both pin prick and light touch sensation. This is determined by a grade of 2 (normal/ intact), since the right and left sides may differ, the sensory level should be determined for each side. Testing will generate up to four sensory levels per dermatome: R-pin prick, R-light touch, L-pin prick, L-light touch. For a single sensory level, the most rostral of all is taken. If sensation is abnormal at C2, the sensory level should be designated as C1. If sensation is intact on one side for light touch and pin prick at all dermatomes C2 through S4-S5, the sensory level for that side should be recorded as “INT” that indicates “intact”, rather than as S5.

Sensory index scoring A score of 2 for each of the 28 key sensory points tested on each side of the body would result in a maximum score of 56 for pin prick, 56 for light touch, and a total of 112. The sensory score cannot be calculated if any required key sensory point is not tested. The sensory scores provide a means of numerically documenting changes in sensory function.

Motor level: The motor level is determined by examining the key muscle functions within each of 10 my tomes and is defined by the lowest key muscle function that has a grade of at least 3 (on supine MMT), providing the key muscle functions represented by segments above that level are judged to be intact (graded as a 5) when full effort from the patient is inhibited by factors such as pain, positioning and hyper tonicity or when weakness is judged to be due to disuse the muscle function should be graded as not testable (NT). C1 to C4, T2 to L1, and
S2 to S5, the motor level is presumed to be the same as the sensory level if testable motor function above (rostral) that level is normal as well.

Motor scoring- A score of 5 for each of the five key muscle functions of the upper extremity would result in a maximum score of 25 for each extremity, totalling 50 for the upper limbs. The same is true for the five key muscle functions of the lower extremity, totalling a maximum score of 50 for the lower limbs. It is no longer recommended to add the upper limb and lower limb scores together.

Neurological level of injury (NLI): The NLI refers to the most caudal segment of the cord with intact sensation and antigravity muscle function strength, provided that there is normal (intact) sensory and motor function rostrally.

Note: It is important to indicate on the worksheet, any weakness due to neurological conditions unrelated to SCI. For example, in a patient with a T8 fracture who also has a left brachial plexus injury, it should be noted that sensory and motor deficits in the left arm are due to the brachial plexus injury, not the SCI. This will be necessary to classify the patient correctly

ASIA Impairment Scale (AIS) is used in grading the degree of impairment:

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

B = Sensory incomplete. Sensory but not motor function is preserved below the neurological level and includes the sacral segments S4-S5, 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 below the neurological level**, and more than half of key muscle functions below the single neurological level of injury have a muscle grade less than 3 (Grades 0–2).

D = Motor incomplete. Motor function is preserved below the neurological level**, and at least half (half or more) of key muscle functions below the NLI have a muscle grade >3.

E = Normal. If sensation and motor function as tested with the ISNCSCI are graded as normal in all segments, and the patient had prior deficits, then the AIS grade is

E. Someone without a SCI does not receive an AIS.
Zone of partial preservation (ZPP): The ZPP is used only with complete injuries (AIS A), and refers to those dermatomes and myotomes caudal to the sensory and motor levels that remain partially innervated. For example, if the right sensory level is C5, and some sensation extends from C6 through C8, then “C8” is recorded in the right sensory ZPP block on the worksheet. If there are no partially innervated segments below a motor or sensory level, the motor and sensory level should be entered in the box for the ZPP on the worksheet.

A framework for physiotherapy Management

The ICF was introduced by the World Health Organization in 2001 and is a revised version of the International Classification of Impairment, Disability and Handicap. The ICF defines components of health from the perspective of the body; the individual and society (see Figure below). One of its primary purposes is to provide unified and standard language for those working in the area of disability

![ICF Diagram](image)

**Body functions** are the physiological functions of body systems, including psychological functions. "Body" refers to the human organism as a whole, and thus, includes the brain. Hence, mental (or psychological) functions are subsumed under body functions. The standard for these functions is considered to be the statistical norm for humans.
Body structures are the structural or anatomical parts of the body such as organs, limbs and their components classified according to body systems. The standard for these structures is considered to be the statistical norm for humans.

Impairment is a loss or abnormality in body structure or physiological function (including mental functions). Abnormality here is used strictly to refer to a significant variation from established statistical norms (i.e., as a deviation from a population mean within measured standard norms) and should be used only in this sense.

Activity is the execution of a task or action by an individual. It represents the individual's perspective of functioning.

Activity limitations are difficulties an individual may have in executing activities. An activity limitation may range from a slight to a severe deviation in terms of quality or quantity in executing the activity in a manner or to the extent that is expected of people without the health condition.

Participation is a person's involvement in a life situation. It represents the social perspective of functioning.

Participation restrictions are problems an individual may experience in involvement in life situations. The presence of a participation restriction is determined by comparing an individual's participation to that which is expected of an individual without disability in that culture or society.

Contextual factors are the factors that together constitute the complete context of an individual's life, and in particular, the background against which health states is classified in ICF. There are two components of contextual factors: Environmental Factors and Personal Factors.

Environmental factors constitute a component of ICF, and refer to all aspects of the external or extrinsic world that form the context of an individual's life and, as such, have an impact on that person's functioning. Environmental factors include the physical world and its features, the human-made physical world, other people in different relationships and roles, attitudes and values, social systems and services, and policies, rules and laws.
Personal factors are contextual factors that relate to the individual such as age, gender, social status, life experiences, and so on, which are not currently classified in ICF but which users may incorporate in their applications of the classification.

Facilitators are factors in a person's environment that, through their absence or presence, improve functioning and reduce disability. These include aspects such as a physical environment that is accessible, the availability of relevant assistive technology, and positive attitudes of people towards disability, as well as services, systems and policies that aim to increase the involvement of all people with a health condition in any area of life. Absence of a factor can also be facilitating, for example, the absence of stigma or negative attitudes. Facilitators can prevent an impairment or activity limitation from becoming a participation restriction.

Barriers are factors in a person's environment that, through their absence or presence, limit functioning and create disability. These include aspects such as a physical environment that is inaccessible, lack of relevant assistive technology, and negative attitudes of people towards disability, as well as services, systems and policies that are either non-existent or that hinder the involvement of all people with a health condition in any area of life.

Capacity is a construct that indicates, as a qualifier, the highest probable level of functioning that a person may reach in a domain in the Activities and Participation list at a given moment. Capacity is measured in a uniform or standard environment, and thus reflects the environmentally adjusted ability of the individual. The Environmental Factors component can be used to describe the features of this uniform or standard environment.

Performance is a construct that describes, as a qualifier, what individuals do in their current environment, and so brings in the aspect of a person's involvement in life situations. The current environment is also described using the Environmental Factors component.

Letter code components

<table>
<thead>
<tr>
<th>Letter</th>
<th>Description</th>
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<tr>
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<td>s</td>
<td>Body Structures</td>
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<tr>
<td>d</td>
<td>Activities and Participation</td>
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<tr>
<td>e</td>
<td>Environmental Factors</td>
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Letters b, s, d and e are followed by a numeric code that starts with the chapter number (one digit), followed by the second level (two digits), and the third and fourth level (one digit each). 34 at the chapter level (as indicated, eight Body Functions, eight Body Structures, Nine Activity/Participation), and 362 at the second level. At the third and fourth level, there are up to 1424 codes available, which together constitute the full version of the classification.

ICF Chapters

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<td>s4 Structure of the Cardiovascular, Immunological and Respiratory Systems</td>
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<td>Haematological, Immunological and Respiratory Systems</td>
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<td>b5 Functions of the Digestive,</td>
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<td>Metabolic, Endocrine Systems</td>
<td>Metabolic and Endocrine Systems</td>
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<tr>
<td>b6 Genitourinary and Reproductive Functions</td>
<td>s6 Structure Related to Genitourinary and s8 Reproductive Systems</td>
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<td>e2 Natural Environment and Human-Made Changes to Environment</td>
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The Rehab Cycle facilitates the structuring, organization and documentation of the rehabilitation process.

ICF core sets

ICF assessment sheet

ICF categorical profile

Intervention table and display

ICF core sets- *The ICF framework offers physical therapists and other rehabilitation professionals a common understanding and a standardized language to describe functioning.*
The ICF Core Sets provide a list of ICF categories applicable and relevant to specific health conditions. The ICF Core Sets serve as practical tools for the documentation and as a reference standard for the reporting of functioning. There are comprehensive core set and brief core set used for assessing the function in person with SCI. A brief ICF core set and comprehensive ICF core set has been published for post-acute SCI. The comprehensive ICF core set consists of 63 body function items, 14 body structure items, 53 activities and participation items, and 32 environmental factor items, with a total of 162 categories. Of these items, 8 body function items, 3 body structure items, 9 activities and participation items, and 5 environmental factors construct the brief ICF core set.

ICF assessment sheet- The *ICF Assessment Sheet* provides a comprehensive overview of the patient’s functioning by presenting the assessment results in all components of human functioning and environmental and personal factors. To describe the patient’s experience of functioning, his own words are used to fill in the patient’s perspective. To reflect the health professional’s perspective, all results from the clinical assessment relevant to the description of the actual functioning status are transferred to the lower part of the sheet. Thus the technical language, or standardized and common, language of the ICF categories is used.

**Assessment sheet-**

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<td>Activity</td>
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<td>B265 no touch sensation</td>
<td>D155 above average</td>
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</table>

I have back pain from time to time
My bladder and bowel are impaired
I am problem with body balance
I am not allowed bear weight in rt hand( fracture in wrist)
I cannot sit from supine
I have little balance in sitting

I need support for transport e.g. in car
Handling barrier with wheel chair is difficult
I need support for washing
I need support for skin care
I cannot dress my self
I cannot toilet my self

I spend lot with my friends
I talk much with wheel chair drivers
I will continue my study
I read more than before accident
I wants to do sports again
<table>
<thead>
<tr>
<th>ICF Categorical Profile</th>
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<tbody>
<tr>
<td><strong>below T3</strong></td>
<td></td>
</tr>
<tr>
<td>B420 Low blood pressure</td>
<td>in acquiring skill</td>
</tr>
<tr>
<td>B525 Fecal incontinence</td>
<td>D410 partial limitation</td>
</tr>
<tr>
<td>B620 Bladder incontinence</td>
<td>changing body position</td>
</tr>
<tr>
<td>B7101 Reduce mobility in knee joints Extension</td>
<td>D4153 instability in sitting position</td>
</tr>
<tr>
<td>B7013 No muscle function below T3</td>
<td>D4200 partial limitation in transferring</td>
</tr>
<tr>
<td>B735 Spasticity below T3</td>
<td>D 465 limitation in overcoming barrier with wheel chair</td>
</tr>
<tr>
<td>7800 TA tightness</td>
<td>D510 partial limitation in washing oneself</td>
</tr>
<tr>
<td>810 Pressure ulcer at risk</td>
<td>D5300 complete limitation in bowel management</td>
</tr>
<tr>
<td></td>
<td>D5300 complete limitation in bladder management</td>
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<tr>
<td>Environmental factors</td>
<td>personal factors</td>
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<tr>
<td>E 1101 Drug when in pain</td>
<td>male 19</td>
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<tr>
<td>E 1201 Need moiled wheel chair</td>
<td>living with family</td>
</tr>
<tr>
<td>E 150 No barrier in centre</td>
<td>supportive person</td>
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<tr>
<td>E 1602 Ramp and stairs disable wheel chair mobility</td>
<td>want to accept the situation</td>
</tr>
<tr>
<td>E 310 Parents support him</td>
<td>want to aim at</td>
</tr>
<tr>
<td>E 310 Big support from friends</td>
<td>independent in daily living</td>
</tr>
<tr>
<td>E 355 Heal care support him</td>
<td>want to develop idea for</td>
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<td></td>
<td>his profession</td>
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Figure 4: ICF Categorical Profile: (extraction); Illustrates the relevant aspects of the functioning status for each patient (Spinal cord Injury, ASIA A Th 3). ICF Qualifier range from 0 = no problem to 4 = complete problem in the components of body functions (b), body structures (s), activity and participation (d) and from -4 = complete barrier to +4 = complete facilitator in the environmental factors. In personal factors, the sign + and - indicates the extent to which a determined pf has a positive or negative influence on the individual’s functioning. C1, 2, 3 mark the relation to Cycle goals 1, 2, 3; SG is related to Service Program Goal, G related to the Global goal.

<table>
<thead>
<tr>
<th>Assessment (12 week post-trauma)</th>
<th>ICF Qualifier*</th>
<th>Goal relation</th>
<th>Goal Value*</th>
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<td>Global Goal: Complete independence, university entrance,</td>
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<td>Service-Program-Goal: Independence in daily activities</td>
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<td>Cycle goal 1: d4 Mobility</td>
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<tr>
<td>Cycle goal 2: d5 Self-care</td>
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<tr>
<td>Cycle goal 3: d9201 Sport</td>
<td>2</td>
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<table>
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<tr>
<th>ICF categories - Intervention targets</th>
<th>ICF Qualifier*</th>
<th>Goal relation</th>
<th>Goal Value*</th>
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<td>b260 Proprioceptive functions</td>
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<td>b265 Touch functions</td>
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<tr>
<td>b279 Sensory functions related to temperature and other stimulants</td>
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<tr>
<td>b28013 Pain in back</td>
<td>C1</td>
<td>0</td>
<td>-</td>
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<tr>
<td>b415 Blood vessel functions - at rest</td>
<td>0</td>
<td>G</td>
<td>0</td>
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<tr>
<td>b420 Blood pressure functions</td>
<td>C1</td>
<td>0</td>
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<td>b525 Defecation functions</td>
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<td>b620 Urination functions</td>
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<td>b640 Sexual functions</td>
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<td>b7101 Mobility of several joints</td>
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<tr>
<td>b7303 Muscles power functions in lower half of the body</td>
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<td>b735 Muscle tone functions</td>
<td>C1</td>
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<td>b750 Motor reflex functions</td>
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<td>b755 Involuntary movement reaction functions</td>
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<td>s810 Structure of areas of the skin - at risk</td>
<td>G</td>
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<tr>
<td>d410 Changing basic body positions</td>
<td>C1</td>
<td>0</td>
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<tr>
<td>d4153 Maintaining a sitting position</td>
<td>C1.2</td>
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<tr>
<td>d4200 Transferring oneself while sitting</td>
<td>C1</td>
<td>1</td>
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<tr>
<td>d450 Walking</td>
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<td>d465 Moving around using equipment</td>
<td>C1</td>
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<td>d4751 Driving a car</td>
<td>C1</td>
<td>0</td>
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<tr>
<td>d510 Washing oneself</td>
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<td>d520 Caring for body parts</td>
<td>C2</td>
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<tr>
<td>d5300 Regulating urination</td>
<td>C2</td>
<td>0</td>
<td>-</td>
</tr>
<tr>
<td>d5301 Regulating defecation</td>
<td>C2</td>
<td>0</td>
<td>-</td>
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<tr>
<td>d540 Dressing</td>
<td>C2</td>
<td>0</td>
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<tr>
<td>d9201 Sport</td>
<td>C3</td>
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<tr>
<th>facilitator</th>
<th>barrier</th>
<th>4+ 3+ 2+ 1+ 0 1 2 3 4</th>
</tr>
</thead>
</table>

| pf Drugs | C1.2 | 2+ |
| pf Assistive products...for personal use | C1 | 0 |
| pf Assistive products...for personal mobility | C1 | 0 |
| pf Design, construction...of buildings for private use | SP | 2 |
| pf Products...of urban land development | - | - |
| pf Immediate family | - | - |
| pf Friends | - | - |
| pf Health professionals | SP.G | 4+ |
| pf Social security services | SP.G | 3+ |
| pf General social support services | SP.G | 3+ |

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<th>negative</th>
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<tr>
<td>pf Knowledge</td>
<td>SP</td>
<td>+</td>
<td>-</td>
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<tr>
<td>pf Acceptance of disease</td>
<td>G</td>
<td>0</td>
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## Figure 4: Intervention Table


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<thead>
<tr>
<th>Intervention target</th>
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<th>Activity and Participation</th>
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<th>PF:</th>
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<td><strong>Intervention</strong></td>
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<td>Sit up-training</td>
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<td>Compression hosey, drugs</td>
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<td>Compression hosey</td>
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<td><strong>b701 Mobility of several joints</strong></td>
<td>Passive movement</td>
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<td><strong>b750 Involuntary movement functions</strong></td>
<td>Body balance training</td>
<td></td>
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<tr>
<td><strong>b7800 Sensation of muscle stiffness</strong></td>
<td>Detonisation, Stretching</td>
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<td><strong>b810 Structure of the skin at risk</strong></td>
<td>Daily inspection</td>
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<tr>
<td><strong>d410 Changing basic body positions</strong></td>
<td>Sit up-training</td>
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<tr>
<td><strong>d453 Maintaining a sitting position</strong></td>
<td>Body balance training</td>
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<td><strong>d4200 Transferring oneself while sitting</strong></td>
<td>Transfer training</td>
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<td><strong>d455 Moving around with wheelchair</strong></td>
<td>Wheelchair training outdoor</td>
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<td><strong>d510 Washing oneself</strong></td>
<td>Assistance/Instruction</td>
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<td><strong>d520 Caring for body parts</strong></td>
<td>Assistance/Instruction</td>
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<td>Assistance/Instruction</td>
<td></td>
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</tr>
<tr>
<td><strong>d5301 Regulating defecation</strong></td>
<td>Assistance/Instruction</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>d550 Dressing</strong></td>
<td>Assistance/Instruction</td>
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<td>Testing of different wheelchairs, reconstruction of car</td>
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<td>Planning and reconstruction of private building</td>
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<td><strong>e5700 Social security services</strong></td>
<td>Clarification, Organization of payments</td>
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<tr>
<td><strong>Knowledge</strong></td>
<td>Teaching, consulting and lectures</td>
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<td><strong>Acceptance/Gap of disease</strong></td>
<td>Behavioral training approaches</td>
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</table>
Figure 5: ICF Evaluation display: Illustrates the change of the functioning status over the course of one cycle (Spinal Cord Injury, ASIA A Th 3, 12 and 16 weeks after trauma. * ICF Qualifier range from 0 = no problem to 4 = complete problem in the components of body functions (b), body structures (s), activity and participation (d) and from -4 = complete barrier to +4 = complete facilitator in the environmental factors. In personal factors, the sign - and + indicates to what extent a determined pf has a positive or negative influence on the individual’s functioning. 1, 2, 3 show the relation to cycle goals 1, 2, 3; SG is related to Service Program Goal, G related to the Global goal.

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<th>Assessment (12 weeks post-trauma)</th>
<th>Evaluation (18 weeks post-trauma)</th>
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<td>Global Goal: Complete independence</td>
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<td>Service-Program-Goal: Independence in ADLs</td>
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<td>Cycle goal 1: d4 Independence in mobility</td>
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<tr>
<td>Cycle goal 2: d5 Independence in self-care</td>
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<tr>
<td>Cycle goal 3: d5001 Sport</td>
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<th>ICF categories - Intervention Targets</th>
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<th>Goal relation</th>
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<td>+</td>
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<td>4+ 3+ 2+ 1+ 0 1 2 3 4</td>
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<td>+</td>
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<td>+</td>
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<td>SP/G</td>
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</table>
ICF intervention table

Assessing impairments it includes assessments of strength, sensation, respiratory function, cardiovascular fitness and pain. Details of how to assess impairments in patients with spinal cord injury can be found in subsequent chapters. Assessing activity limitations and participation restrictions there are several well-accepted assessment tools used to measure activity limitations and participation restrictions including the Functional Independence Measure (FIM), Spinal Cord Independence Measure, and Quadriplegic Index of Function. They all measure independence across a range of domains, reflecting different aspects of activity limitations and participation restrictions. For example, they assess ability to dress, maintain continence, mobilize, transfer and feed. Some have been specifically designed for patients with spinal cord injury, and others are intended for use across all disabilities.

More physiotherapy-specific assessments of activity limitations and participation restrictions quantify different aspects of mobility and motor function. For example, some assess the ability to walk (e.g. the WISCI, 10m Walk Test, the Motor Assessment Scale, 6-minute Walk Test, Timed Up and Go), ability to use the hands (e.g. the Grasp and Release test, Silverman test, Carroll test, Jensen test) and ability to mobilize in a wheelchair. There is as yet no consensus on the most appropriate tests, and currently physiotherapists tend to use a battery of different assessments, including non-standardized, subjective assessments of the way patients move. Assessment tools for measuring activity limitations and participation restrictions

Brief description

Setting goals-Goal setting is an important aspect of a comprehensive physiotherapy and rehabilitation programme. The process needs to be patient-centred. For example, a key goal of rehabilitation might be to return to work or school. Physiotherapy-specific goals then need to be identified and linked to each participation restriction goal. The physiotherapy-specific goals should be functional and purposeful activities as defined within the activity limitation and participation restriction domains of ICF. Both short- and long-term goals need to be set. These may include goals to be achieved within a week or goals to be achieved over 6 months. Goals should be SMART. That is, they should be: Specific, Measurable, Attainable, Realistic and Time bound. Physiotherapy-related goals need to be based on predictions of
future independence, taking into account contextual factors such as patients’ and families’ perspectives, priorities and personal ambitions Goals should be SMART. That is, they should be: Specific, Measurable, Attainable, Realistic and Timebound. Physiotherapy-related goals need to be based on predictions of future independence, taking into account contextual factors such as patients’ and families’ perspectives, priorities and personal ambitions.

Setting goal for complete lesion SCI

This table shows complete spinal injury with no ZPP below level of lesion:

<table>
<thead>
<tr>
<th></th>
<th>C1-c3 tetraplegia</th>
<th>C4 tetraplegia</th>
<th>C5 tetraplegia</th>
<th>C6tetraplegia</th>
<th>C7-8 tetraplegia</th>
<th>Thoracic paraplegia</th>
<th>Lumbar and sacral paraplegia</th>
</tr>
</thead>
<tbody>
<tr>
<td>Unassisted ventilation</td>
<td>no</td>
<td>yes</td>
<td>yes</td>
<td>yes</td>
<td>yes</td>
<td>yes</td>
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<td>Push manual wheelchair</td>
<td>no</td>
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<td>limited</td>
<td>limited</td>
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<td>Hand to mouth activity</td>
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<td>yes</td>
<td>yes</td>
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</tr>
<tr>
<td>Activity</td>
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<td>No</td>
<td>Limited</td>
<td>Yes</td>
<td>Yes</td>
<td>Yes</td>
<td>Yes</td>
</tr>
<tr>
<td>-------------------------------</td>
<td>-----</td>
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<td>Self feeding</td>
<td>no</td>
<td>no</td>
<td>limited</td>
<td>yes</td>
<td>yes</td>
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</tr>
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<td>Horizontal transfer</td>
<td>no</td>
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<td>yes</td>
</tr>
<tr>
<td>Lying to sit</td>
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<td>no</td>
<td>limited</td>
<td>yes</td>
<td>yes</td>
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<td>yes</td>
</tr>
<tr>
<td>Floor to wheelchair</td>
<td>no</td>
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<td>no</td>
<td>limited</td>
<td>limited</td>
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<td>yes</td>
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<tr>
<td>Standing with orthosis in parallel bar</td>
<td>no</td>
<td>no</td>
<td>no</td>
<td>no</td>
<td>limited</td>
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</tr>
<tr>
<td>Walking with orthotics and aids</td>
<td>no</td>
<td>no</td>
<td>no</td>
<td>no</td>
<td>no</td>
<td>limited</td>
<td>yes</td>
</tr>
</tbody>
</table>

C1–C3 tetraplegia

Patients with C2 and above tetraplegia have total paralysis of the diaphragm other respiratory muscles and consequently are ventilator-dependent. Patients with C3 tetraplegia retain a small amount of diaphragm function but not usually enough to breathe spontaneously. All have paralysis of upper and lower limbs and trunk muscles but are able to move their heads. They are fully dependent. Typical level of independence attained by patients with ASIA complete spinal cord injury. C1–C3 on others for all motor tasks and personal care activities.
They mobilize in chin control power wheelchairs and can use head-, mouth- or voice-activated technology

C4 tetraplegia

Patients with C4 tetraplegia have partial paralysis of the diaphragm and total paralysis of all four limbs and trunk muscles. They retain a small amount of voluntary control around the shoulders and have good strength in the rhomboid muscles but still mobilize in a chin-control power wheelchair. They can breathe independently but in all other respects their activity limitations are similar to those of patients with C1–C3 tetraplegia.

C5 tetraplegia

Patients with C5 tetraplegia have partial paralysis of the upper limbs but full paralysis of the trunk and lower limb muscles. They have good strength of the deltoid and biceps muscles, but poor strength of other shoulder muscles. They have no functioning the triceps muscles or any muscles about the wrist or hand. Despite this, they can use a hand-control power wheelchair with the hand passively rested on or secured to the joystick (see Chapter 13). They are unable to perform gross motor tasks such as transferring, rolling or moving from lying to sitting and require assistance foremost personal care activities. They can, however, take their hands to their mouth, head and face. They can use the upper limbs to perform simple tasks provided no fine hand control is required and the appliance or utensil is attached to the hand with a splint. Upper limb function is usually possible with splints to stabilize the paralysed wrist. For example, a keyboard can be used with a typing stick attached to the hand and a steering wheel of a car can be turned with adaptations to the wheel

C6 tetraplegia

There is a large functional difference between patients with C5 and C6 tetraplegia. This is due to the preservation of the pectoralis, serratus anterior latissimus dorsi and wrist extensor muscles. The latissimus dorsi muscle, in combination with the pectoralis and serrate anterior muscles, enables weight bearing through the upper limbs. This provides the potential to lift body weight and transfer. The altissimo dorsa muscle also provides some trunk stability. Although not normally considered a trunk muscle; the altissimo dorsa become important in the absence of other trunk muscles. Preservation of the pectorals muscles makes it possible to roll over in bed and provides stability around the shoulder when weight bearing. Serratus anterior is also important for scapula stability.
Patients with C6 tetraplegia have the potential to live independently, provided they are adequately equipped and set up. Some can transfer, roll, move from lying to sitting, dress, bathe and attend to personal hygiene, although all these motor tasks are time-consuming and difficult to master. Patients with C6 tetraplegia mobilize in manual wheelchair, but most also use a power wheelchair. Voluntary control of the wrist extensor muscles provides crude grasp (tenodesis grip; see This makes it possible to hold objects between the index finger and thumb, or in the palm of the hand, despite paralysis of the finger and thumb flexor muscles.

C7 tetraplegia

Patients with C7 tetraplegia typically attain higher levels of independence than those with C6 tetraplegia because of the function provided by the triceps, wrist flexor and finger extensor muscles. The triceps muscles are particularly important because they increase the ability to bear weight through a flexed elbow. The triceps muscles also enable patients to carry and hold objects above their heads. Patients with C7 tetraplegia still have paralysis of the finger and thumb flexor muscles so, despite the ability to extend the fingers, they rely on a tenodesis grip for hand function.

C8 tetraplegia

Patients with lesions at C8 have finger and thumb flexor activity, and therefore can actively grasp and release objects. Consequently, hand function is superior to that of patients who rely on a tenodesis grip. Greater strength in the triceps and shoulder muscles enables these patients to more easily attain independence than those with lesions at C6 and C7.

T1 paraplegia

Patients with lesions at T1 have near-normal hand function, although they retain some weakness in the intrinsic and lumbrical muscles affecting fine hand control. They still have extensive paralysis of the trunk muscles and therefore, like those with higher lesions, have difficulty sitting unsupported.

Patients with thoracic paraplegia have full upper limb movement, varying degrees of paralysis of the trunk and total paralysis of the legs. They are predominantly wheelchair-dependent, although some can walk short distances with extensive bracing and walking aids. Patients with high thoracic paraplegia have more extensive paralysis of the trunk muscles
than those with lower thoracic paraplegia, primarily affecting their ability to sit unsupported and master complex transfers.

Lumbar and sacral paraplegia

Patients with lumbar and sacral paraplegia have varying extents of paralysis of the lower limbs and do not commonly have complete lesions. Most can walk with or without aids and orthoses although some remain wheelchair-dependent.

Setting goals for patients with incomplete lesions

Outcomes for patients with zones of partial preservation, or ASIA C or D incomplete lesions, are less predictable. In these patients, patterns of neurological loss are diverse, the extent of possible neurological recovery is unclear and consequently accurate and detailed predictions of motor function are difficult. Knowledge about levels of independence attainable by patients with complete spinal cord injury is used as a starting reference then modified depending on individual circumstances and neurological status. Some degree of intuition, developed with experience, is needed to generate goals that are realistic and appropriate.

Linking impairment with activity limitation and participation restrictions

Once goals of treatment are defined in terms of activity limitations and participation restrictions, it is then necessary to determine which impairments prevent the attainment of each goal. That is, key impairments need to be linked to specific activity restrictions and participation limitations. Identification and treatment of impairments without linking them to activity limitations and participation restrictions risks wasting time, money and resources on impairments which are of little consequence. For example, limited hamstring extensibility is an important impairment for some but not all patients. Unless limited hamstring extensibility is linked to activity and participation goals, physiotherapists might be tempted to direct therapeutic attention at increasing the extensibility of the hamstring muscles in some patients unnecessarily.

Transfers and bed mobility of people with lower limb paralysis

Rolling- It used for dressing, changing body position, initial step for lying to sitting.
Log rolling can be useful method in every two hours interval in acute stage of thoracic spinal cord injury.

Four people are required.

Paraplegia- They can easily move the body using momentum.(fig:1)

Person with quadriplegia with rolling steps-

1. Upper extremities and head moves opposite of rolling then swing the upper limb rolling side and momentum transfer to trunk and lower limb there by helping body rotation . crossing legs and flexion of head facilitate rolling. Person with C6 quadriplegia has lack of elbow extension of elbow there by unable to produce momentum rather hit face. Splint or gaiter is required to maintain elbow extension monkey pole can be used as an altered method for rolling.

Lying to long sitting(fig: 2)

Following steps paraplegia are follows

1. rolling to one side

2. Take weight on both hands

3. Lifts off the trunk

4. Extend elbows and straight body

Following steps quadriplegia C6 are follows

Rolling to right side take weight on left hand and lift trunk bring right hand on the bed

Positioning the top hand under the leg:

The right wrist is extended and hooked behind the right knee. A pillow is placed under the left elbow

5. Shuffling the bottom elbow around the body:
The right arm is adducted with the wrist anchored behind the knee. This momentarily removes weight from the left elbow.

At this instant the left elbow is shuffled a small distance towards the feet. This procedure is repeated several times to ‘walk’ the left elbow up towards the knees. Moving into the upright Position the right arm is adducted with the wrist anchored behind the knee. The left arm is abducted. The action at both arms pulls/pushes the trunk into the midline.

Vertical lift

The ability to vertically lift is an important task for patients to master early. Vertical lifts are used to relieve pressure, transfer, dress, and move about the bed. Patients need to be able to lift while sitting in bed with the knees extended and while sitting in their wheelchairs with their knees flexed. Lifting with the knees extended is often easier because patients can use the paralysed hamstring muscles to help maintain an upright position.

To vertically lift, the hands are placed next to the hips, usually on the seating surface but occasionally on adjacent surfaces. The patient then pushes down through the hands to lift the trunk on the stabilized arms. There are three components to the lift: elbow extension, shoulder depression and shoulder flexion. Once the elbows are extended, further lift is achieved by depressing the scapulas on the trunk and by inclining the trunk forwards on the fixed shoulders. The vertical lift is controlled by the altissimo dorsa, anterior deltid, pectoralis major and lower trapeziums muscles. The glen humeral joint is stabilized in adduction throughout the lift by the shoulder adductors and rotator cuff.

Muscles. Patients with C6 tetraplegia and paralysis of the triceps muscles have the added difficulty of preventing elbow collapse while lifting. They overcome this problem by externally rotating the shoulders, urinating the forearms and placing the elbows in a hyper-extended position. This upper limb position places the trunk’s centre of mass posterior to the elbow joint. In turn, this creates a tendency for the elbows to extend under the weight of the body even though the triceps muscles are paralysed.

Elbow collapse is also prevented by contraction of the anterior deltid muscles.

These muscles generate torques which rotates the shoulders into flexion. Shoulder flexion can extend the elbows if the forearms are stabilized. Stability in the forearms is achieved by wrist flexor torques generated as patients lean forwards. The wrist flexor torques originates from
the stretch of the paralysed wrist flexor muscles other soft tissues spanning the front of the wrists.

In this way, elbow extension is a product of the torques generated by the trunk’s centre of mass, the active contraction of the shoulder flexor muscles and the passive stretch of the structures. A patient with thoracic paraplegia moving directly into sitting from the supine position. Spanning the front of the wrist

Paraplegic transferring to bed

Moving to the front edge of the wheelchair:

The elbows are extended and the shoulders adducted and depressed to vertically lift the body. The shoulders are then extended to push the trunk and legs forwards on the seat.

2. Positioning the feet on the floor:

The left arm lifts the right leg onto the ground. The right arm is used to hold the trunk upright.

The mirror of this procedure is used to position the left leg on the floor.

3. Positioning the hands:

The right hand is placed on the bed and the left hand on the front corner of the wheelchair.

4. Lifting and shifting the body onto the bed:

The elbows are extended and the shoulders adducted and depressed to vertically lift the body.

The right shoulder is adducted and the left shoulder is abducted to laterally shift the body.

5. Lifting the legs onto the bed:

Weight is borne through the right elbow while the left arm is used to lift each leg onto the bed.
Paraplegic transferring to bed

C6 quadriplegia

1. Moving to the front edge of the wheelchair:

The head and trunk are extended over the back of the wheelchair. The hands are pushed in behind the back. The hips are levered forwards by external rotation of the shoulders and extension of the wrists. The sides and top of the backrest are used as a fulcrum.

2. Lifting the first leg onto the bed:

The left arm is hooked around the back of the wheelchair to prevent forward fall. The right armies used to lift the leg onto the bed. The leg is held with a wrist extension hook.

3. Lifting the second leg onto the bed:

4. Positioning the hands:

The left hand is placed on the bed and the right hand on the apex of the far wheel.

Passive stretch of the hamstring muscles prevents a forward fall.

5. Lifting and shifting the body onto the bed:
The shoulders are externally rotated and depressed and the elbows are ‘passively ‘extended to vertically lift the body. The left shoulder is adducted and the right shoulder is abducted to shift the body laterally.

Sitting unsupported one strategy is to use upper limb muscles to help stabilize the trunk in an upright position. The muscles capable of providing trunk stability are the latissimus dorsi, pectoralis and serratus anterior muscles. Their importance helps explain why patients with C6 tetraplegia attain a higher level of independence than those with C5 tetraplegia. Patients with thoracic paraplegia and C6 tetraplegia also use compensatory postural adjustments to sit unsupported. Small postural adjustments are normally used by able-bodied people during performance of seated tasks. For example, when reaching sideways in sitting, able-bodied people laterally flex the trunk and neck away from the direction in which they are reaching. This minimizes sideways displacement of the centre of mass. Patients with spinal cord injury exaggerate postural adjustments to compensate for the loss of leg and trunk muscles. Thus to reach sideways with one arm they abduct the contra lateral arm. Similarly, to reach forwards with one arm they reach backwards behind the body with the other arm while at the same time extending the neck. A patient with C6 tetraplegia sitting unsupported with the knees extended. In this position passive tension in the paralysed hamstring muscles helps maintain the trunk in an upright position. The patient is leaning forwards to position the centre of mass of the trunk anterior to the hip joints. This prevents a backwards fall.

Contracture management common complication in person with spinal cord injury.

Neural component e.g. spasticity

causes are

non-neural component e.g. soft

Tissue tightness prone for difficulty in performing in motor task. Pressure ulcers, pain and sleep disturbance muscle tightness is not always disadvantages light hamstring tightness can improve sitting balance in C6 SCI. Finger flexor tight improve grip capability in tetraplegia C6.
Assessment- A contracture is likely to have a neurally mediated component if there are signs of spasticity such as clonus or a velocity-dependent increase in resistance to stretch. First, sustain stretch spastic muscle for 2 to 3 minutes so spasticity will reduce then measure range of motion. However, the only definite way to determine the contribution of neurally mediated factors to a contracture is to measure passive joint range of motion when spasticity is pharmacologically blocked. Non-neural component can easily be measured with goniometer.

30 minutes of stretch were sufficient to completely prevent loss of sarcomeres. 30 minutes of stretch three to five times a week over a 4-week or 3-month period to either the plantarflexor, shoulder, thumb or hamstring muscles of patients with spinal cord injury self-positioning, splinting and standing programmes reduces tightness.

Passive movements prevents intraarticular adhesion I immobilization-induced deterioration of joint cartilage swelling. All above benefits not proved yet at least 5 movements is required to each joints passive straight leg raising up to 60 degree (FIG4) hip flexion with knee flexion to 90 degree (FIG5). Both movements can produce back strain. Contracture can be prevented rather that treated.

Assessment of strength- Manual muscle tests have traditionally been used by physiotherapists to measure strength of patients with spinal cord injury. Strength can be assessed by determining the ‘one repetition maximum’ (1 RM) this test is appropriate for muscle groups.
with grade 4/5 and 5/5 strength. One repetition maximum refers to the maximum weight a patient can lift through an entire range of motion against gravity a ‘modified’ 1 RM can be used to measure strength in muscles with grade 3/5 or less strength. Instead of lifting a weight against gravity, the patient moves a weight horizontally.

HAND held myometer-Strength can also be measured with hand-held myometers. Myometers are small portable devices used to test isometric strength. There are mechanical and electronic versions. All provide measures of force, not torque. Isokinetic dynamometers Isokinetic dynamometers (Fig6) measure torque during dynamic (concentric or eccentric) contractions at a constant angular velocity the equipment is expensive, not easy to adjust when testing many muscle groups, and not appropriate for patients with profound weakness or patients restricted to bed.

(Fig6, Is kinetic dynamometers)

Neural intact muscle-- strength can increase by 2% per week with strength training.

First 2 weeks-Improvements in strength with training may be due to better motor unit synchronization, firing and recruitment. After 4 weeks strength can be due to hypertrophy of muscle, Males and females of all ages benefit in a similar way from strength training.

Progressive resistance training-The key aspects of progressive resistance training are resistance, repetition and progression. 8-12 RM( repetition maximum)- Weight can only be lifted for 8-12 times but further lifting is not possible. This weight is normally equivalent to about 60–80% of a 1 RM, 3 settings per day, 3 days per week, rest period between sessions -1 to 3 minutes. Every week interval test 8-12 RM then used increased weight as resistance. These principles of progressive resistance training can be applied to most types of
strengthening programmes such as lifting weights, using isokinetic strengthening equipment, pulling on theraband or practising motor tasks.

Theraband—first decides which colour person with spinal cord can only stretch for 8-12 times. each set 8-12 time, 3 sets per day only, 1-3 minutes rest between set, 3 days per week change to next colour if it can stretched for 8-12 time. There is considerable debate and research around subtle questions relating to optimal protocols for progressive resistance training. For instance, while most advocate multiple sets within a training session, one review argues that there is now sufficient evidence to indicate that one set is equally effective. Some evidence suggests that training with long rests between sets (3 minutes) is superior to training with shorter rests (40 seconds) that eccentric strengthening is superior to concentric strengthening and that varying the load between sessions is better than fixing neutrally intact muscles the load. Similarly, there is some evidence that the speed at which the weight is lifted may be important. These controversies have not yet been resolved, but there remains broad agreement that regular, progressive high-resistance exercise is needed to increase strength. Such programmes continue to be recommended by the American College of Sports Medicine.

Specificity of training—progressive resistance training should also practice near real situation, e.g. for achieving function getting down from wheelchair to floor the near real function such as lifting upwards and backwards from a small stool positioned in front of the wheelchair or lifting sideways between two stools should utilised for the upper limb strength training. It is important that training done within the context of motor tasks involves contracting muscles against sufficient resistance to induce a training effect. For example, if training involves lifting from blocks, the height of the blocks should be adjusted so that the patient can perform between 8 and 12 repetitions of the lift but no more.

Sometimes it is difficult to adhere to these principles because patients are limited by factors such as their ability to maintain an upright position. If it is not possible to control training intensity within the context of motor tasks, strength training is probably best done using strengthening equipment such as dead weights.
Training muscle power and endurance - muscle power endurance can also improve with progressive strength training 30–60% of 1 RM

For example, a programme aimed at increasing upper limb power for wheelchair propulsion might consist of pushing up inclines as quickly as possible with extra weight placed on the wheelchair. Similarly, a programme to improve a patient’s ability to lift themselves upstairs with knee–ankle–foot orthoses might consist of repeated rapid shoulder depression exercises in standing within the parallel bars. Resistance can be applied by attaching weights to the ankles. Alternatively, arm ergo meters can be used to train general upper limb power. For example, patients can cycle against a fixed load as fast as possible in 30-second bouts.

Endurance - Progressive resistance training also has carry-over effects on endurance. However, to further target endurance, training needs to place a sustained demand on muscles. This requires low to moderate resistance with high repetitions (20 or more) and minimal time for recovery between sets (less than 1 minute if doing 10–15 repetitions, and 1–3 minutes if doing 15–20 repetitions). The repetitions are done at high training velocities (i.e. 180° per sec). In general, endurance exercise is likely to be most effective when the exercise closely resembles the task which is to be trained.

Strength training for partially paralysed muscles Exercises for muscles with grade 2/5 strength are done in gravity-eliminated positions. The easiest way to do this is to exercise in a horizontal plane. As soon as patients can move through range 8–12 times with gravity eliminated then patient position is changed and the limb is lifted against gravity. Strength training for patients with partial paralysis can also be done within the context of motor tasks.

Strength training for patients with flickers of movement Strength training is more difficult in patients who are extremely weak (less than grade 2/5) with little or no ability to move through range. Assistive devices need to be used to help patients move through range or strength training needs to be restricted to isometric contractions. For these patients, EMG feedback can be used to provide patients and physiotherapists with feedback and encouragement. Some commercially available EMG feedback devices can be used to structure training sessions with timed phases of effort and relaxation. These devices can also be used to ensure patients contract a pre-selected minimum effort with auditory feedback about success.
Alternatively, there may be merit in encouraging patients to use mental practice and motor imagery. The two are slightly different, involving systematic and repeated cognitive practice or imagery of an activity without movement. The benefits for people with spinal cord injury are speculative but there is strong evidence that repeated and intense mental rehearsal improves task performance in high-level athletes and possibly also in patients with various types of neurological disabilities.

The use of electrical stimulation Electrical stimulation has long been advocated as a way of inducing hypertrophy in paralysed muscles and increasing stimulated strength. Increasing ‘strength’ in fully paralysed muscles may be beneficial if electrical stimulation can subsequently bemused for purposeful tasks. For example, electrical stimulation can be used to provide crude hand function to patients with tetraplegia. Similarly, electrical stimulation continues to be used to develop sophisticated ways of enabling people with full lower limb paralysis to walk and cycle. Electrical stimulations sometimes used to induce hypertrophy and improve blood flow. Improved blood flow, particularly in the glutei area, may reduce the incidence of pressure ulcers.

Electrical stimulation is also used as a means of increasing voluntary strength of partially paralysed muscles. If electrical stimulation is to be used, then it is probably best if its applied in conjunction with voluntary strength training and appropriate resistance to ensure patients adhere to the principles of progressive resistance training

Spasticity- is “disordered sensory-motor control, resulting from an upper motor neuron lesion, presenting as intermittent or sustained involuntary activation of muscle

Clinical symptoms

difficulty in performing function,

‘Paresis

feel resistance to movements

gait abnormality

mechanism treatment of spasticity
Passive Movement-based Approaches for Reducing Spasticity

Hippotherapy may result in short-term reductions in spasticity.

A combination of neural facilitation techniques and Baclofen may reduce spasticity.

Rhythmic passive movements may produce short-term reductions in spasticity.

Prolonged standing or other methods of producing muscle stretch may result in reduced spasticity.

Electrical passive pedalling systems may result in short-term reduction in spasticity.

Active Movement-based Approaches for Reducing Spasticity

Active exercise interventions such as hydrotherapy and FES-assisted cycling and walking may produce short-term reductions in spasticity.

Direct Muscle Stimulation for Reducing Spasticity

Electrical stimulation applied to individual muscles may produce a short term decrease in spasticity. There is also some concern that long-term use of electrical stimulation may increase spasticity.

Various Forms of Afferent Stimulation for Reducing Spasticity

Ongoing (TENS) transcutaneous electrical nerve stimulation programs result in short-term reductions in spasticity which may last for up to 24 hours.

Penile vibration and rectal probe stimulation may be effective at reducing lower limb muscle spasticity for several hours.

Other forms of afferent stimulation including massage, cryotherapy, helium-neon irradiation, and whole-body vibration may result in immediate spasticity reduction but require more research to examine long-term effects.

Direct Spinal Cord and Transcranial Magnetic Stimulation

Spinal cord stimulation may provide spasticity relief over a few months but long-term effectiveness and cost-effectiveness is less certain.
Repetitive transcranial magnetic stimulation may provide spasticity relief over the short-term but long-term effectiveness is unknown.

Neuro-Surgical Interventions for Spasticity

Dorsal longitudinal T-myelotomy may result in reduced spasticity.

Oral Baclofen

Oral baclofen reduces muscle spasticity in people with SCI.

Intrathecal Baclofen

Bolus or long-term intrathecal baclofen decreases spasticity and may improve functional outcomes with low complication rates and is a cost effective intervention.

Effect of Medications Other than Baclofen

Tizanidine may be useful in treating SCI spasticity.

Clonidine may be effective in treating SCI spasticity but more evidence is required to support its routine use.

The usefulness of 4-Aminopyridine in the treatment of SCI spasticity requires confirmation through additional well-designed studies.

Cyproheptadine may be useful in treating SCI spasticity but requires additional confirmatory research.

Gabapentin may be useful in treating SCI spasticity but requires additional confirmatory research.

Orphenadrine citrate may reduce spasticity in SCI but additional confirmatory research is needed.

The use of L-threonine in the treatment of SCI spasticity requires confirmation through additional well-designed studies.

Continued use of diazepam and dantrolene would benefit from controlled comparison studies.

Cannabinoids
Oral delta-9-tetrahydrocannabinol (dronabinol) may help to reduce spasticity but requires additional evidence from controlled studies.

Nabilone has been shown to be effective in reducing spasticity but additional research is needed.

Focal Neurolysis

Botulinum neurotoxin appears to improve focal muscle spasticity in people with SCI.

Phenol block may improve pain, range of motion and function related to shoulder spasticity in individuals with tetraplegia.

Phenol block may reduce hip adductor spasticity in individuals with paraplegia and tetraplegia.

Orthostatic hypotension-defined as a reduction in systolic BP of at least 20 mmHg or diastolic BP of at least 10 mmHg within 3 min of standing or being raised greater than 60 degree on a tilt table.

symptoms -

light-headedness, dizziness, blurred vision, ringing in the ears, nausea, fatigue, cognitive impairment, palpitations, tremulousness, headache and neck ache.

Mechanisms- In normal person supine to Standing results in blood pooling of approximately 500 to 1,000 ml in the lower extremities and splanchnic circulation. there is a small reduction (<10 mm Hg) in SBP and a small increase in DBP (approximately 2.5 mm Hg).This lead to inadequate return of venous blood to heart .there is a transient reduction in cardiac output and stimulation of the baroreceptors, in the aortic arch and carotid sinus, which reflexively increases sympathetic tone and vascular resistance and inhibits parasympathetic activity resulting in an increased heart rate. But this reflex activity does not occur due to disruption This gravity-induced drop in blood pressure, is detected by arterial baroreceptors in the aortic arch and carotid sinus, of sympathetic activity.
Management-Activities that decrease venous return to the heart, such as coughing, straining, and prolonged standing, should be avoided, particularly in hot weather. Dorsiflexion of the feet before assuming an upright posture may promote venous return to the heart, accelerate the heart rate, and increase blood pressure.

Squatting and stooping forward can result in an increase in blood pressure. In patients who present with symptoms after prolonged standing, simply sitting down can often raise the blood pressure. Physical counter-manoeuvres like crossing one’s legs while standing and maintaining muscle contraction for 30 seconds can increase systemic venous return, thereby causing increased cardiac output and blood pressure. Waist high compression stockings and abdominal binders may be helpful. In patients with autonomic failure and supine hypertension, raising the head of the bed by 10 to 20 degrees at night can reduce hypertension, prevent overnight volume loss, and help restore morning blood pressure upon standing. Liberal intake of salt and water to achieve a 24-hour urine volume of 1.5 to 2 litters may attenuate fluid loss commonly seen in autonomic insufficiency.

Tilt table- first find out threshold angle at which person with spinal cord injury reveal symptom. put 10 degree below threshold angle for 20 minutes. increase 10 degree per day up to 90 degree.

If no pharmacological measures fail to improve symptoms, pharmacologic agents should be initiated. Fludrocortisone, midodrine, nonsteroidal anti-inflammatory drugs, caffeine, and erythropoietin have all been used to treat orthostatic hypotension due to autonomic failure.

Autonomic dysreflexia (AD)

AD is characterized by the acute elevation of arterial blood pressure (BP) and bradycardia (slow heart rate) in individuals with an injury at level T6 or above. The usual resting arterial BP in individuals with cervical and high thoracic SCI is approximately 15 to 20 mmHg lower than in able-bodied individuals AD occurs more often after 6 months of SCI at or above the 6th thoracic segment, there also is clinical evidence of episodes of AD in the first days and weeks after injury.

Causes- The most common noxious causes include bladder (distended or severely spastic bladder, urinary tract infection, bladder or kidney stones, urological procedure or even inserting a catheter), bowel (constipation, fecal impaction, recto sigmoid gaseous distension,
rectal irritation, enema or manual evacuation, haemorrhoids), and skin issues (ingrown toenail, burns, pressure area, tight clothing and sexual intercourse,

1) Strong sensory stimulus from below level of lesion (eg distended bladder) conveyed into spinal cord.
2) Signals transmitted up spinal cord initiate massive reflex sympathetic activation, causing widespread vasoconstriction (involving splanchnic blood vessels), and resulting in severe hypertension.

3) Brain detects a sudden rise in blood pressure (via signals from baroreceptors in aortic and carotid vessels carried in CNs 1X and X).

4) Brain responds and attempts to control BP sending descending primary inhibitory impulses from brainstem via spinal cord (which are blocked). Sympathetic activity is un-opposed.

Impulses travelling through the vagus nerve cause secondary bradycardia.

An individual with an SCI at or above T6 presents with an acute onset of signs and symptoms of autonomic dysreflexia.

1. Recognize the signs and symptoms of autonomic dysreflexia, including:

- Elevated blood pressure.
- Pounding headache.
- Bradycardia (may be a relative slowing so that the heart rate is still within the normal range).
- Profuse sweating above the level of the lesion, especially in the face, neck, and shoulders, or possibly below the level of the lesion.
- Piloerection or goose bumps above or possibly below the level of the lesion.
- Cardiac arrhythmias, atrial fibrillation, premature ventricular contractions, and atrioventricular conduction abnormalities.
- Flushing of the skin above the level of the lesion, especially in the face, neck, and shoulders, or possibly below the level of lesion.
- Blurred vision.
- Appearance of spots in the patient's visual fields.
- Nasal congestion.
- Feelings of apprehension or anxiety overran impending physical problem.
Minimal or no symptoms, despite a significantly elevated blood pressure (silent autonomic dysreflexia).

(Scientific evidence–None; Grade of recommendation–Expert consensus; Strength of panel opinion–Strong)

An individual may have one or more of these signs and symptoms when experiencing an episode of autonomic dysreflexia. Symptoms may be minimal or even absent, despite an elevated blood pressure.

Health-care providers should be aware that the varying cognitive and verbal communication abilities of adults, children, and adolescents can cause the symptoms of AD to be absent, subtle, vague, or expressed imperfectly. Because of the varying cognitive and verbal communication abilities of individuals as they progress through infancy, childhood, and adolescence, symptoms of AD may not be expressed or may be communicated in a less articulate manner compared to a cognitively intact adult with SCI. For instance, preschool-aged children, even though they are verbal, may present with vague complaints; they are not able to accurately articulate that they are experiencing a pounding headache—a cardinal feature of autonomic dysreflexia.

2. Check the individual’s blood pressure.

A sudden, significant increase in both the systolic and diastolic blood pressure above their usual levels, frequently associated with bradycardia. An individual with SCI above T6 often has a normal systolic blood pressure in the 90–110 mm Hg range. Therefore, a blood pressure of 20 mm to 40 mm Hg above baseline may be a sign of autonomic dysreflexia.

Systolic blood pressure elevations more than 15–20 mm Hg above baseline in adolescents with SCI or more than 15 mm Hg above baseline in children with SCI may be a sign of AD.

(Scientific evidence–III/V; Grade of recommendation–C; Strength of panel opinion–Strong)

Elevated blood pressures can be life-threatening and need immediate investigation and treatment(Cole et al., 1967; Guttman et al., 1965).

For children and adolescents, age and body size are determinants of normal blood pressures, with increasing blood pressures advancing with age and approximating adult norms in older teenagers(National High Blood Pressure Education Program, 1996). Similar to adults with
SCI, children and adolescents with cervical and upper thoracic SCI would be expected to have lower baseline blood pressures compared to the general population.

Therefore, it is important to determine and document baseline blood pressures on an annual basis or as needed, as the child or adolescent with SCI ages. For the purposes of these guidelines, the panel agreed that systolic blood pressures at or above 150 mm Hg in adults, 120 mm Hg in children under 5 years old, 130 mm Hg in children 6–12 years old, and 140 mm Hg in adolescents is when pharmacological agents should be considered.

Be calm and maintain a reassuring environment in the presence of the child’s parents/caregiver when obtaining blood pressures. Any anxiety associated with obtaining blood pressures in children and adolescents may make it difficult to obtain accurate measurements both for baseline determinations as well as during an episode of autonomic dysreflexia. Teaching parents how to obtain blood pressures or having school nurses obtain baseline blood pressures may be beneficial.

It is important that all health-care professionals remain calm and maintain a relaxing atmosphere.

Use appropriately sized blood pressure cuffs when measuring blood pressure in children and adolescents. The width of the blood pressure cuff should be approximately 40 percent of the arm circumference, measured midway between the olecranon and the acromiom (Perloff et al., 1993).

The cuff bladder will cover 80 to 100 percent of the circumference of the arm. A blood pressure cuff that is too small may result in an overestimation of the individual’s blood pressure. In contrast, a blood pressure cuff that is too large may result in an underestimation of the blood pressure, which is less than the error of overestimation with a cuff that is too small. If an appropriately sized blood pressure cuff is not available, interpretation of the blood pressure is complicated. However, it is important for the health-care professional or caregiver to remember that small blood pressure cuffs tend to overestimate and large cuffs tend to underestimate the true blood pressure.

3. If a pregnant woman with a spinal cord injury at T6 or above presents with signs and symptoms of autonomic dysreflexia, consider referral to an obstetric health-care provider under the following circumstances:
Determination of choice of antihypertensive medication.

Persistent hypertension after resolution of the acute autonomic dysreflexia episode.

Persistent symptoms of autonomic dysreflexia despite acute care measures.

Life-threatening autonomic dysreflexia.

Autonomic dysreflexia episode occurring in the third trimester of pregnancy.

Hypotension requiring pharmacological treatment.

First episode of autonomic dysreflexia during the pregnancy.

Presence of vaginal bleeding or suspicion of labour.

Decisions to be made about long-term medication use.

Unclear about the causes, signs, and symptoms, despite a normal blood pressure.

Care of pregnant women with AD should take into account that, due to compression of the venacava, hypotension may occur if the woman is in supine position. A lateral tilt or upright position facilitates resolution of the hypotension and improves uterine blood flow.

4. If signs or symptoms of AD are present, but the blood pressure is not elevated and the cause has not been identified, refer the individual to an appropriate consultant depending on symptoms.

5. If the blood pressure is elevated, immediately sit the person up if the individual is supine.

6. Loosen any clothing or constrictive devices.

7. Monitor the blood pressure and pulse frequently.

Blood pressures have the potential of fluctuating quickly during an AD episode. Therefore, pressures need to be monitored every few minutes (every 2 to 5 minutes is commonly cited),
until the individual is stabilized. Individuals with spinal cord injury usually have impaired autonomic regulation, and therefore blood pressures can rapidly fluctuate; Cole et al., 1967; Eric

8. Quickly survey the individual for the instigating causes, beginning with the urinary system.

9. If an indwelling urinary catheter is not in place, catheterize the individual.

10. Prior to inserting the catheter, instil 2 percent lidocaine jelly (if immediately available) into the urethra and wait 2 minutes, if possible.

11. If the individual has an indwelling urinary catheter, check the system along its entire length for kinks, folds, constrictions, or obstructions and for correct placement. If a problem is found, correct it immediately.

12. If the catheter appears to be blocked, gently irrigate the bladder with a small amount (10–15 cc) of fluid, such as normal saline at body temperature. Irrigation should be limited to 5–10 ml for children under 2 years of age and to 10–15 ml in older children and adolescents. Avoid manually compressing or tapping on the bladder.

13. If the catheter is draining and the blood pressure remains elevated, proceed to recommendation

14. If the catheter is not draining and the blood pressure remains elevated, remove and replace the catheter.

15. Prior to replacing the catheter, instil 2 percent lidocaine jelly (if immediately available) into the urethra and wait 2 minutes, if possible.

14 ACUTE MANAGEMENT OF AUTONOMIC DYSREFLEXIA Catheterization can exacerbate autonomic dysreflexia. The use of lidocaine jelly may decrease sensory input and relax the sphincter to facilitate catheterization. The peak effect of lidocaine jelly is between
2–5 minutes. Exercise clinical judgment regarding elevated blood pressure and the use of lidocaine; immediate catheterization may be necessary.

16. If difficulties arise in replacing the catheter, consider attempting to pass a coude catheter or consult a urologist.

A coude catheter may be useful if there is an associated bladder neck obstruction.

17. Monitor the individual’s blood pressure during bladder drainage.

Sudden decompression of a large volume of urine would be expected to normalize blood pressure. However, this may cause hypotension if the individual has already been given pharmacological agents to decrease blood pressure.

18. If acute symptoms of autonomic dysreflexia persist, including a sustained elevated blood pressure, suspect fecal impaction.

19. If the elevated blood pressure is at or above 150 mm Hg systolic, consider pharmacologic management to reduce the systolic blood pressure without causing hypotension prior to checking for fecal impaction.

20. If the blood pressure remains elevated but is less than 150 mm Hg systolic, proceed to recommendation.

Reviewer opinion varied on whether the next step should be investigating other causes (e.g., fecal impaction) or initiating pharmacologic management. The control of hypertension may need to be addressed prior to digital stimulation or other diagnostic manoeuvres, which may exacerbate autonomic dysreflexia. This is true for no pregnant adults, pregnant women, and children and adolescents, as well. There are no studies showing the exact point at which blood pressure becomes dangerous. For this recommendation, the panel decided to adopt 150 mm Hg systolic BP as the value at which pharmacological treatment should be considered, based on Guttman et al. (1965). An adult with an injury at or above T6 would be expected to have a baseline systolic BP between 90 and 110 mm Hg. Guttman et al. (1965) described an AD episode as occurring when the systolic BP reached 20 to 40 mm Hg above baseline. Pharmacological management of AD in children and adolescents should be considered prior
to checking for fecal impaction if the blood pressure is excessively elevated for the child’s or adolescent’s age and height. Knowing the child’s baseline blood pressure is very important when deciding whether to intervene with antihypertensive medications. Indications for pharmacological intervention may include a systolic blood pressure of 120 mm Hg in infants and younger children (under 5 years old), 130 mm Hg in older children (6–12 years old), and 140 mm Hg in adolescents.

20. Use an antihypertensive agent with rapid onset and short duration while the causes are being investigated.

21. Monitor the individual for symptomatic hypotension.

22. If fecal impaction is suspected and the elevated blood pressure is less than 150 mm Hg, check the rectum for stool, using the following procedure: With a gloved hand, instill a topical anaesthetic agent, such as 2 percent lidocaine jelly, generously into the rectum. Wait 2 minutes if possible for sensation in the area to decrease. Then, with a gloved hand, insert a lubricated finger into the rectum and check for the presence of stool. If present, gently remove, if possible.

If autonomic dysreflexia becomes worse, stop the manual evacuation. Instil additional topical anaesthetic and recheck the rectum for the presence of stool after approximately 20 minutes.

23. If the precipitating cause of the autonomic dysreflexia episode has not yet been determined, check for less frequent causes. The individual may need to be admitted to the hospital; see recommendation 25 for considerations.

24. Following an episode of autonomic dysreflexia, instruct the individual to monitor symptoms and blood pressure for at least 2 hours after resolution of the episode to make sure that it does not reoccur. Educate the individual to seek immediate medical attention if it reoccurs. Monitor inpatients closely for at least 2 hours, as deemed necessary by the healthcare provider. Seek the pregnant woman’s obstetrical care provider for evaluation. The hypertension and symptoms may have resolved because of the medication rather than the treatment of the cause. Symptoms managed by pharmacologic treatment may begin to reverse themselves within this time frame.
25. Consider admitting the individual to the hospital for monitoring to maintain pharmacologic control of the blood pressure, and to investigate other causes:

If there is poor response to the treatment specified above.

If the cause has not been identified.

If there is suspicion of an obstetrical complication.

Because of the loss of sensation, individuals with spinal cord injury can have significant pathology with minimal symptoms. These may include problems such as acute abdominal pathology, long bone fractures, and ingrown toenails (Braddom and Rocco, 1991). Individuals with spinal cord injury frequently may have a positive urine culture.

However, this may not be the precipitating cause for autonomic dysreflexia, and therefore other causes of autonomic dysreflexia also should be investigated.

26. Document the episode in the individual’s medical record, including Presenting signs and symptoms and their course.

Treatment instituted.

Recordings of blood pressure and pulse.

Response to treatment.

Evaluate the effectiveness of the treatment according to the level of outcome criteria reached:

Cause of the episode has been identified.

Blood pressure has been restored to normal limits for the individual (usually 90 to 110 systolic mm Hg for a tetraplegic individual in the sitting position).

Pulse rate has been restored to normal limits.

The individual is comfortable, with no signs or symptoms of autonomic dysreflexia, of increased intracranial pressure, or of heart failure.

An education plan has been completed and included preventive and emergency
management guidance.

27. Once the individual with spinal cord injury has been stabilized, review the precipitating cause of the AD episode with the individual, family members, significant others, and caregivers.

This preventive process entails: Adjusting the treatment plan to ensure that future episodes are recognized and treated to prevent a medical crisis or, ideally, are avoided altogether.

Discussing autonomic dysreflexia during the individual’s education program, so that he or she will be able to minimize the risks known to precipitate AD, solve problems, recognize early onset, and obtain help as quickly as possible.

Providing the individual with education about the prevention and treatment of autonomic dysreflexia at the time of discharge that can be referred to in an emergency.

Heterotopic ossification

Heterotopic ossification (HO) is the abnormal formation of true bone within extraskeletal soft tissues

The onset of HO usually is 1-4 months after injury in SCI patients, although it may occur as early as 19 days or as late as 1 year following injury. HO always occurs with complete injuries below the level of injury in SCI patients, and most authors agree that there is no relation to presence or absence of spasticity in SCI patients. In SCI patients with HO, the hips are most commonly involved.

At the hip, the flexors and abductors tend to be involved more frequently than are the extensors or adductors.

At the knee, the medial aspect is most commonly affected by HO.

Shoulders and elbows are the most commonly affected upper extremity joints

pathophysiology- The aetiology of HO remains unknown but 2 factors were found to be prerequisites for ectopic ossification: (1) traumatic ischemic degeneration of involved muscle and (2) tissue expression of bone morphogenic proteins (BMPs).

It also has been shown that expression of many genes, including BMP, is regulated by mechanical stress. The target cells in the muscle for BMP are mesenchymal stem cells, also
called satellite cells. These cells are precursors capable of differentiating into many cell types, including osteoblasts. Thus, BMP may play a role as a paracrine factor in the differentiation of satellite cells into bone-forming cells. Clinically, muscle trauma has been reported as a cause of HO after SCI by numerous investigators, including Bodley and colleagues, as well as Snoeck and co-investigators. The types of muscle trauma proposed as initiating HO are muscle tears, ruptures, oedema, and bleeding. It has also been suggested that factors such as intensive rehabilitation, transfer activities, and repeated minor trauma during activities of daily living can cause superimposed mechanical stress and initiate HO.

Clinical sign- A diagnosis of HO can be made clinically if localized inflammatory reaction, palpable mass, or limited ROM is observed. Alkaline phosphate bone scintigraphy and ultrasonography are recommended imaging studies for the early diagnosis of HO.

Prevention-

The nonselective NSAID indomethacin SR prescribed for 3 weeks in a dose of 75 mg/d, after SCI, reduced the incidence of HO by 2-3 times.

A 25 mg/d prescription of the selective COX-2 inhibitor rofecoxib decreased the risk of HO formation by 2.5 times.

Pulse low intensity electromagnetic field therapy uses magnetic fields to increase oxygen levels and decrease toxic by-products of inflammation by increasing local blood flow (Durovic et al. 2009). PLIMF is effective in preventing HO post SCI.

Surgery- Resection of immature HO leads to recurrence rates of nearly 100%. Haemorrhage may be a significant problem at the time of surgery. If bone is matured then surgery is done. PROM start after 3 days of surgery.

Radiotherapy- It is also used in HO.

Physiotherapy- During the acute inflammatory stage, the patient should rest the involved joint in a functional position, and the physical therapist should initiate gentle PROM as soon as possible. Once acute inflammatory signs have subsided, aggressive PROM and continued mobilization are recommended.

Extracorporeal Shock Wave Therapy (ESWT) is used treatment for recurrent Neurogenic Heterotrophic Ossification (NHO).
PAIN

Pain is a common complication person with spinal cord injury. Incidence of pain range from 48 - 94% following spinal cord injury. It is estimated that 30 to 40% person with SCI experience disabling pain. Severe pain was noted in 10-15% of persons with quadriplegia; 25% of those with thoracic paraplegia and 42-51% of those with lesions of the caudalequine (Ragnarsson 1997. Over 50% of SCI patients develop chronic pain. Severe pain is more common the lower down the lesion in the spinal cord. Pain post SCI most often begins within the first 6-12 months post-SCI.

Location of pain-

The most common types of pain post SCI are: 1) a burning pain (likely neuropathic) usually localized to the front of torso, buttock or legs or 2) an aching pain (likely musculoskeletal) usually localized to the neck, shoulders and back

<table>
<thead>
<tr>
<th>Table (Burchiel &amp; Hsu 2001)</th>
<th>Specific Structure/Pathology (Tier 3)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Nociceptive</td>
<td>Musculoskeletal</td>
</tr>
<tr>
<td></td>
<td>Bone, joint, muscle trauma, or inflammation</td>
</tr>
<tr>
<td></td>
<td>Mechanical instability</td>
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<tr>
<td></td>
<td>Muscle spasm</td>
</tr>
<tr>
<td></td>
<td>Secondary overuse syndromes</td>
</tr>
<tr>
<td>Visceral</td>
<td>Renal calculus, bowel, sphincter dysfunction, etc.</td>
</tr>
<tr>
<td></td>
<td>Dysreflexic headache</td>
</tr>
</tbody>
</table>
Neuropathic

Above Level

Compressive

mononeuropathies

Complex regional pain syndromes

At Level

Nerve root compression (including cauda equine)

Syringomyelia

Spinal cord trauma/ischemia (transitional zone, etc.)

Dual-level cord and root trauma (double lesion syndrome)

Below Level

Spinal cord trauma/ischemia

1 Proposed IASP Classification of Pain Related to SCI

<table>
<thead>
<tr>
<th>Broad Type (Tier 1)</th>
<th>Broad System (Tier 2)</th>
<th>Specific Structure/Pathology (Tier 3)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Nociceptive</td>
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</tr>
<tr>
<td></td>
<td></td>
<td>Mechanical instability</td>
</tr>
</tbody>
</table>
Subbarao et al (1995), in a survey of 800 SCI patients, found that 72.7% of responders reported some degree of chronic pain at the wrist and shoulder, with wheelchair propulsion and transfers being responsible for most of the pain. McCasland et al (2006) noted that in their survey, 70% of SCI had shoulder pain, one-third had a previous injury to their shoulder and 52% reported a bilateral pain. Quadriplegics were more likely to have shoulder pain (80%). Previous shoulder trauma increased the risk of having shoulder pain.

Central" dysesthesia or "deafferentation" pain is the most common type of pain experienced below the level of SCI and is generally characterized as a burning, aching and/or tingling sensation.
Central dysesthetic pain (CDP) is defined as dissociative sensory loss and absence of spinothalamic-anterolateral functions, with different degrees of dorsal column function preservation present almost exclusively in incomplete SCI patients. CDP takes weeks or months to appear and is often associated with recovery of some spinal cord function. Paradoxically CDS is often characterized by complete loss of temperature, pinprick, and pain perception below the level of the lesion. It rarely occurs in spinal cord Injuries with complete sensory loss or loss of both sensory and motor functions below the level of the lesion. Davidoff et al. (1987a) concurred and further noted dyesthetic pain was more likely to be found in incomplete paraplegia resulting from penetrating wounds of the spinal cord, and in spinal fractures treated with conservative management.

Segmental pain is further described as occurring at or just above the level of sensory loss in the cutaneous transition zone from the area of impaired/lost sensation to areas of normal sensation, involving at least one to three dermatomes.

Ragnarsson (1997) also noted that in an individual with a cervical cord injury, segmental pain may be described as tingling, burning or numbing pain in the shoulders, arms or hands, those with a thoracic cord injury frequently describe a circumferential, feeling of tightness and pain around the chest and abdomen while lumbar lesions tend to be localized to the groins and different parts of the lower extremities.

Treatment-

Massage and heat may be helpful for post-SCI pain.

Acupuncture may reduce post-SCI pain.

Regular exercise reduces post-SCI pain.

A shoulder exercise protocol reduces post-SCI shoulder pain intensity.

MAGIC wheels 2 gear wheelchair reduces shoulder pain.

Hypnosis may reduce pain intensity post SCI.

Transcranial magnetic stimulation reduces post-SCI pain.

Cognitive behavioural therapy combined with pharmacological treatment results in short term improvement in chronic pain.

Visual imagery may reduce neuropathic pain post SCI.

Transcranial electrical stimulation is effective in reducing post-SCI neuropathic pain.

Static field magnet may reduce nociceptive shoulder pain post SCI.

Transcutaneous electrical nerve stimulation may reduce pain at site of injury in patients with thoracic but not cervical injury.

Transcranial magnetic stimulation reduces post-SCI pain.

Gabapentin and pregabalin improve neuropathic pain post SCI.

Lamotrigine may improve neuropathic pain in patients with incomplete SCI.

Levetiracetam is not effective in reducing neuropathic pain post SCI.

Valproic acid does not reduce neuropathic pain post SCI.

Amitriptyline is effective in reducing pain in depressed SCI individuals.

Trazodone does not reduce post-SCI pain.

Lidocaine through a subarachnoid lumbar catheter and intravenous Ketamine improve post SCI pain short term.

Mexilitene does not improve SCI dysesthetic pain.

Intrathecal Baclofen improves musculoskeletal pain post SCI and may help dysethetic pain related to spasticity.

Motor point phenol block reduces spastic shoulder pain.

Botulinum toxin injections for treatment of focal spasticity improves pain.

Intravenous morphine reduces mechanical allodynia.

Tramadol reduces neuropathic pain.

Alfentanil reduces chronic pain post SCI.

Alfentanil is more effective in reducing wind up like pain post SCI than ketamine.
Cannabinoids are a potential new treatment for post-SCI pain in need of further study.

Intrathecal Clonidine alone does not provide pain relief although it may be helpful in combination with Intrathecal Morphine.

Topical capsaicin reduces post-SCI radicular pain.

Spinal cord stimulation may improve post-SCI pain.

Dorsal longitudinal T-myelotomy procedures reduce pain post SCI.

DREZ surgical procedure reduces pain post

Wheel chair-The customised wheelchair should provide sufficient upright stability to enable patients to sit without needing to grasp the wheelchair or rest the elbows on armrests to prop themselves upright. Those with upper limb function should also be able raise their arms without toppling forwards and propel themselves up a slope without tipping the wheelchair backward

Few key issues should be evaluated before choosing manual wheel chair for individual patients

Frame

<table>
<thead>
<tr>
<th>Type of frame</th>
<th>advantage</th>
<th>disadvantage</th>
</tr>
</thead>
<tbody>
<tr>
<td>Rigid type</td>
<td>lighter, sturdier, more adjustable and easier to push used for active patients</td>
<td>transportation</td>
</tr>
<tr>
<td>Folding type</td>
<td>Transportation easier,</td>
<td>more likely to break</td>
</tr>
</tbody>
</table>
Seat-to-floor height-the seat-to-floor height at the rear of the wheelchair < the seat-to-floor height at the front of the wheelchair. It is distance from back of knee joint to bottom of heel and add 2 cm for clearance of foot plate.

patients sits straight on cushion, thighs are parallel, wearing shoes measurement should be taken.

if the seat is too high

Difficulties in putting knees under table, less stable with risk of tipping

If the seat is too low-patients can not rest and works on top of table

Seat depth- distance between buttock to back of knee joints and add 3 to 4 cm.

If the seat is too deep, the front edge pushes up hard against the back of the knees. This can cause compression of the blood vessels and nerves in the popliteal fossa and encourage patients to slide forwards on the seat.

If the depth of the seat is too shallow, there will be a large space between the front edge of the seat and the back. reduces stability

Seat width- A) width of hip- female

B) width of shoulders- male

add 2 cm for clearance between hip side of wheel chair

If too wide-access through doorways and within tight spaces can be difficult. Excessive width also places the wheels further apart, necessitating more shoulder abduction when propelling the wheelchair if the seat is too narrow, it makes it difficult for patients to get in and out of the wheelchair. In addition, the lateral aspects of the hips can rub the inside of the back
wheels, causing damage to skin or clothing Seat rake-is determined by the difference between the distance to the ground at the front and rear of the seat- It is generally slope down word toward back rest,

If the seat is horizontal- the pelvis tends to slide forwards creating shearing forces under the ischial tuberosities as the patient slides

If the rake is increased-, weight is moved posterior off the front castors and over the back wheels. This makes it easier to get into a wheel stand position and easier to propel the wheelchair Excessive rake also makes it difficult to move forwards in the wheelchair when transferring back rest width- It should match whole trunk at different levels it . If the backrest is too wide - limits arm movement, making wheelchair propulsion difficult

Backrest height -patients with trunk paralysis require a backrest which extends just above the inferior tip of the scapula if the backrest is unnecessarily high it can interfere with propelling the wheelchair if the backrest is unnecessarily low- risk of fall back ward while climbing slope Distance between the front castors and back wheels- The distance between the front castors and back wheels is called the ‘wheelbase’. It determines a wheelchair’s rolling resistance, turning circle and ‘tipsiness’ the back wheels move forwards the distance between the back wheels and front castors is reduced,- providing a tighter turning circle .

This adjustment also moves weight from the front castors to the back wheels, decreasing overall rolling resistance and easier to push the wheelchair. but also increases the wheelchair’s ‘tipsiness’.

Front caster- sizes ranging from 5 to 19.8 cm - The effect of castor size on wheelchair propulsion is only important if large amounts of weight are borne through the front castors

Smaller caster- provide a tighter turning circle, increase rolling resistance and requires increase effort for pushing wheel chairs. dig into soft ground and get caught in cracks larger caster- Easier to propel a wheelchair but ensure that they do not rub the back of patients’ heels when the castors rotate.

Back wheels- size of back wheels is 60 cm (or 24 in)types of back wheels- solid- advantage-do not puncture and require very little maintenance
Disadvantage - they are heavy, bury into soft surfaces and transmit bumps up through the seat, providing a rougher ride pneumatic - easier to push and manoeuvre and provide a smoother ride.

Disadvantage - higher maintenance and are vulnerable to puncture

Wheels can be either fixed or removable

Wheel camber- Cambered wheels are tilted with more distance between the bottoms of the two wheels than the top advantages -they provide greater lateral stability and make it easier to turn.

Disadvantage- increase the width of wheelchairs, making them more difficult to manoeuvre in tight spaces and get through narrow doorways.

Brakes- stabilize the wheelchair when transferring push/pull brake- easy arm reach, high on the frame of the wheelchair scissor brakes- More capable patients often prefer scissor-type brakes placed out of the way and low on the frame of wheelchairs.

Anti-tip bars- They are small wheels located at the back of the wheelchair several centimetres above the ground and help prevent them toppling over backwards

Footplates - Footplates can be either rigid, fold-up or swing-away

If patients can stand- require folding or swing-away footplates

Armrests- The armrests should be positioned so the elbows are supported at 60° and shoulders are level

If the armrests are too low- the elbows fall into more extension and the shoulders drop.

if the armrests are too high -the elbows will be excessively flexed and the shoulders elevated

Power wheelchairs- a joystick which is controlled by either the hand or chin.

Some power wheelchairs are primarily designed for indoor use and are light and small, with little power and tight turning circles. They also have smaller wheels with less tread. Other power wheelchairs are designed for outdoor use and are bigger, heavier, more powerful and highly stable

Wheelchair cushions
Air-based cushions- Therapists can use their fingers to crudely check the inflation of air-based cushions by ensuring there is enough room to slide two fingers between the ischial tuberosities and seat. Insufficient space for the fingers indicates that the cushion is under-inflated. Air-based cushions relieve pressure by distributing air from pockets of high pressure to pockets of low pressure.

Gel-based cushions - They dissipate pressure by allowing gel to move from areas of high pressure to areas of low pressure. Most have a contoured foam base upon which the gel sits. The foam base has a specially-designed hollow or ‘well’ for the ischial tuberosities. This helps ensure that most pressure is borne by the soft tissues over the lateral aspect of the thighs, leaving the ischial tuberosities free to submerge within the gel-filled well.

Needless to say, if the well is too wide both the lateral thighs and ischial tuberosities fall into it with a high risk of the ischial tuberosities burying through the gel, pressing up hard against the base of the cushion or wheelchair.

Foam-based cushions- Foam-based cushions also redistribute pressure. Their effectiveness is dependent on the compressibility of the foam and the cut of the cushion. Some cushions use two or more types of foam, typically with firmer foam under the lateral aspect of the thighs and more compressible foam under the ischial tuberosities. This encourages more weight to be borne through the thighs and less weight through the vulnerable ischial tuberosities.

Standing and walking with assistive devices

People with tetraplegia. (i.e. ASIA A or B) can stand with frames, tilt tables or standing wheelchairs.

USE- physiological standing associated with being upright and weight bearing through the legs

People with thoracic paraplegia above T9- can ambulate with walking aids on level ground provided they have good upper limb strength and extensive orthotic support. Gait is slow and the energy cost of walking is high. These people usually find it difficult to perform associated tasks such as walking up and down slopes, negotiating steps and uneven terrain, putting the orthoses on and off, and turning in tight spaces.

People with thoracic paraplegia below T10 and lumbosacral lesion -people with composite ASIA lower extremity motor scores less than 20/50 generally use wheelchairs as their
primary form of mobility. They may, however, walk around the home or exercise with orthoses and aids. People with ASIA lower extremity motor scores more than 20/50 generally attain the capacity for community ambulation and are capable of walking at reasonable speeds (e.g. 1.0 m/sec\(_{-1}\); this compares to a comfortable walking speed of between 1.0 and 1.7 m/sec\(_{-1}\) for able-bodied individuals). The ability to hitch and control the pelvis increases the likelihood of attaining a functional level of ambulation. People within complete tetraplegia who are dependent on walking aids generally require more strength in their lower extremities than those with paraplegia in order to adequately compensate for their upper limb weakness.

Standing- patients with tetraplegia stand is with a tilting table, electronic standing wheelchairs and frames. The patient is strapped to the tilt table, standing chair or frame to prevent knee, hip and trunk. Patients with thoracic paraplegia has good upper limb strength can stand in relatively simple frames which block knee flexion. A strap behind the hip prevents hip flexion. At home, appropriately placed benches or sinks can be used. It is often claimed that regular standing improves psychological status, renal function and bone density. It is also said to help spasticity orthostatic hypotension and joint range of motion. While there is a good theoretical basis to believe that standing has all these beneficial effects, sound evidence is lacking.

Bilateral knee–ankle–foot orthoses- They all stabilize the knee in full extension and ankle in 5–10° dorsiflexion. Different types of knee joints can be used. Most can be unlocked so the knee can be flexed when sitting. Foot clearance during swing phase gate is done by pelvis hiking' swing-to' pattern- If the feet are moved up to the crutches the gait is called a ‘swing-to’ pattern swing through pattern- if the feet are moved past the crutches the gait is called a ‘swing through’ pattern.

reciprocal pattern-left crutch move forward followed by ,right leg forward, then right crutch move forward followed by, left leg forward Two point gait--left crutch and, right leg move forward at one time, then right crutch and, left leg move forward

Hip extension can be maintained without using the hands by leaning the trunk backwards and extending the lumbar spine. This positions the centre of mass of the trunk and head behind the hips, creating a torque which passively extends the hips. Excessive hip extension is prevented by the soft tissues spanning the front of the hips. Hip–knee–ankle–foot Orthoses the hip guidance orthosis. The hip guidance orthosis, also called the ParaWalker, was first
introduced for children with spina bifida in the 1970s. It consists of two knee–ankle–foot orthosis attached to a rigid body brace with laterally placed hip joints. The hip joints are low friction and restrict flexion and extension, although they can be released to enable sitting. During the swing phase of gait, the leg flexes like a pendulum. That is, hip flexion is achieved solely by the effects of gravity on the unweighted leg. Gravity will only act to flex the hip when the leg is extended with the mass of the leg behind the hip joint, the reciprocating gait orthosis (RGO; Early versions of reciprocating gait orthosis coupled the two hip joints together with cables The cables were attached under high tension so that forces from extension in one leg were transmitted to flexion of the other. In more recent years a pivot bar has replaced the cables. The pivot bar is positioned centrally and at the back of the corset in the lumbar region. Reciprocating gait orthosis incorporating pivot bars are called isocentric reciprocating gait orthosis. A variation is the advanced reciprocating gait orthosis the medial-linkage orthosis The medial linkage orthosis, also known as the walkabout orthosis, has a hinge-like joint positioned between the legs. The joint limits hip flexion and extension but does not mechanically assist either. Instead, gravity flexes the hip and moves the unweighted leg forward. Hip extension is achieved by leaning the trunk backwards and extending the lumbar spine Ankle–foot orthoses (AFO) isolated paralysis of the dorsiflexor muscles needs only a lightweight orthosis to resist the small torques tending to plantarflex the ankle during swing paralysis of the plantarflexor muscles needs a heavy duty orthosis to resist the large torques tending to rotate the tibia over the fixed foot during stance posterior leaf spring AFO is a type of dorsiflexion-assist AFO. It is made from thin, light, thermoplastic material and worn inside a shoe. As the name implies, it assists dorsiflexion. It is primarily used in patients with isolated paralysis of the dorsiflexor muscles. The narrow strip of plastic behind the ankle gives flexibility, allowing the tibia to move over the fixed foot during stance. However, when the foot is off the ground the plastic recoils, preventing foot-drop.

Plastic solid AFO It not only prevents plantarflexion during swing but also prevents excessive rotation of the tibia over the fixed foot during stance. For this reason it is typically used for patients with paralysis of the dorsiflexor and plantarflexor muscles. In order to stabilize the ankle joint in this way the orthosis is made from heavy duty thermoplastic material which wraps anterior to the ankle joint.

Hinged solid plastic AFO. The hinged solid plastic AFO incorporates ankle joints. There are many different types of ankle joints. One type assists dorsiflexion. It incorporates steel
springs which compress during stance but rebound during swing. In this way, it enables dorsiflexion during stance but prevents plantarflexion during swing.

Toe-off AFO-The toe-off AFO is made from resin (see Figure 6.18). It prevents foot-drop but also assists with the push-off phase of gait. It works on the principle of storing elastic energy for release at the end of stance. In this way, it is similar to the polyfibre feet of below-knee prostheses.

AFO on gait -An AFO which blocks plantar flexion necessitates additional knee flexion at heel strike to get the foot flat on the ground. This requires large knee extensor torques to prevent knee collapse. The effect of an AFO on the knee at heel strike is exacerbated when walking up or down slopes (see Figure 6.20). Placing an AFO in a less dorsiflexed position reduces knee flexion at heel strike. Occasionally the ankle joint is intentionally positioned in some plantar flexion (this type of AFO is called a floor reaction AFO).

Foot clearance can be helped by a heel raise on the opposite side.

An AFO which blocks dorsiflexion find difficulty in the task which requires full dorsiflexion.

Paralysis of the quadriceps muscles (L2, L3, L4) Knee–ankle–foot orthosis are therefore required to stabilize the knee in extension and fixate the ankle.

Knee splints to prevent hyper-extension

Patients with paralysis of the hamstring muscles may experience rapid and forceful hyper extension in mid to late stance phase. If this is repeated often over many years it produces genu recurvatum (a knee hyper-extension deformity). Genu recurvatum is undesirable because it is unsightly and may be associated with chronic kneepain. However, the cause–effect relationship between genu recurvatum and knee pain has been questioned. It is also possible to prevent knee hyper-extension with an AFO which fixes the foot in 5°
dorsiflexion.\textsuperscript{133,134} This prevents the tibia from rotating backwards on the fixed foot, thereby helping to hold the knee in a slightly flexed position.

Paralysis around Hip

The effects of hip flexor muscle paralysis on gait are particularly evident when walking up stairs or slopes, which requires lifting the leg.

There is no simple orthosis for the management of isolated paralysis of the hip flexor muscles. While the hip guidance and reciprocating gait orthosis mechanically assist hip flexion

Electrical stimulation Simpler systems which solely stimulate the peroneal nerve to initiate mass flexion of the limb during swing are also used.\textsuperscript{6,137–139} Alternatively, electrical stimulation is used to specifically target foot-drop in patients with paralysis of the dorsiflexor muscles.\textsuperscript{140} Sometimes electrical stimulation is solely used to help patients get from sit to stand. More recently entire electrical stimulation systems have been implanted, although these systems have only been used in a small number of patients.

\textsuperscript{knee–ankle–foot orthoses}
Bladder management

Anatomy and physiology

Upper Urinary tract- consists of kidney and Ureters

Lower Urinary Tract- Bladder and urethral sphincters

Urethral sphincters are traditionally divided into internal sphincters and external sphincters

Internal sphincters extends from bladder to proximal urethral where as external sphincters starts from distal to membranous urethra

Neuroanatomy. Three nerve i.e. peripheral parasympathetic, sympathetic and somatic system are controlling bladder storage and emptying.

The parasympathetic efferent originates from detrusor nucleus which is located in the intermediolateral gray matter of sacral cord (s2-s4).It supplies to detrusor through pelvic nerve and travel to -cholinergic receptors. Stimulation of Parasympathetic nervous system can cause bladder contraction.

Sympathetic efferent nerve through hypogastric nerve supply to bladder and urethra begins in intermediolateral gray matter at T11-L2 and travel to Alpha and Beta -adrenergic receptors. IT can cause inhibition of bladder and facilitates contraction of internal sphincters.
Normal voiding physiology - Two phases

Filling phase (storage phase) - urine collects in bladder but does not try to void.

Emptying (voiding) phase - when person is attempts to void or is told to void.
When bladder full

High activity of mechanoreceptors---excitation of pontine nucleus(Barrington centre)----
Descending activity from pons inhibits sympathetic preganglionic neurons(major effects)+excites sacral Para sympathetic preganglionic neurons---- bladder contraction.

High activity of mechanoreceptors---- excites sympathetic preganglionic neurons

But net effect is inhibition sympathetic preganglionic neurons---reduces bladder relaxation+ internal sphincters relaxation
Filling phase-stimulation of Beta receptors located in the body of the bladder that cause relaxation.

Stimulation of Alpha- receptors located ay base of the bladder and urethra that cause contraction.

Sympathetic stimulation also inhibits excitatory parasympathetic ganglion transmission, which help suppress bladder contraction.

Types of bladder

Detrusor -sphincters dyssynergia \ low tone bladder ,hyperreflexia sphincters

Uninhibited bladder ,hypotonicity sphincters low tone bladder ,hypotonicity sphincters

Suprasacral injuries-

spinal shock phase-does not have any bladder contraction .The electrical activity of external sphincters not affected in spinal shock phase. The tests such as the ice-filling test, Bethanechol supersensivity testing and suprapubic tapping are used to find contraction of detrusor muscle.

uninhibited bladder gradually returns after 6 to 8 weeks

Management-

indwelling catheter

IC( intermittent catheterization- If a person not receiving iv fluid, output less than 100 ml /hr then start IC.

Daily fluid output -2 litres, repeat 4 hours interval. Goal is to keep catheterization volumes more than 400 ml.

Detrusor -sphincters dyssynergia

When person wants normally to void the external and internal sphincters relax but detrusor contracts, In this condition sphincters and detrusor involuntary contract at same time

It is two types
1) Detrusor - internal sphincters dyssynergia - Inco-ordination of activity between Detrusor and internal sphincters

2) Detrusor - external sphincters dyssynergia - Inco-ordination of activity between Detrusor and internal sphincters

Symptoms - over flow urinary incontinence and various visceral sensation such as tingling, flushing, increase lower extremity spasms and autonomic dysreflexia. DSD can affect kidneys by Vesicoureteral reflux (urine travels upward toward kidney instead of coming out of urethra.).

Management - To improve bladder capacity

    To reduce out- let resistance(sphincters)

Uninhibited bladder - Pharmacological treatment - Oxybutyn, Tricyclic anti depressants alone or combination of ant cholinergic agents and C-fibre neurotoxin can reduce uninhibited bladder activity.

Prolong time periods of Indwelling catheter and reflex voiding can decrease bladder capacity. So treatment of choice is CIC (clean intermittent catheterization)

Surgical option - Bladder Augmentation - It is used to improve bladder capacity. The most common technique is clam cystoplasty (taking a piece of intestine and sewing onto partial bivalve bladder.

Depressor myomectomy - Bladder muscle is stripped away from mucosal lining so bladder gradually stretches to large size.

Urinary Diversions - standard diversion (non continent) and continent diversion. standard diversion (non continent) - 10 to 15 cm ileum isolated and ureters implanted. one end of ileum closed whereas other end is averted as stoma and pass through abdominal wall. continent diversions - person can do CIC. They are two types 1) Orthotropic diversion and continent catheterizable pouch
Orthotropic diversion- like bladder augmentation but connect to urethra continent catheterizable pouch- Small bowel used as pouch so catheterization is easier.

Reflex voiding- utilised intact sacral macturition reflex. Condom catheter can be applied to collect urine in bag. If SCI patient has balance voiding Triggering voiding can be recommended. Balance voiding is defined as the adequate bladder contraction with bladder pressure less than 70-80cm H2O in male and 40-60 cm H2O in female.

Advantage- No limit to drink fluid, and not require good hand function

Dis advantage- require assistance, potential penile skin damage and wear condom and leg bag.

If increase Sphincter tone cause of DSD-

Pharmacological treatment- Alpha blocking agents

Surgical treatment- transurethral sphincterotomy, It is not advisable to use if bladder contraction less than 30 H2O, recurrent DSD

Stainless steel woven mesh stent

Botulinum toxin

Behavioural treatment- supra pubic tapping (10 to 30 seconds)- triggers bladder contraction-- reflexibly relax sphincters

Scissoring movement in rectum reduces tone of anal and urethral sphincters

Low tone bladder, hyperreflexia sphincters

Symptoms- person will be continent, always, post void residual urine will be more Ideal for CIC

If hyperreflexia sphincters causing voiding problem treatment will be same as above for sphincters

Uninhibited bladder, hypotonicity sphincters found in mylomeningocele child,
symptoms- dribbling of urine continuously
Treatme - first reduce tone of bladder muscle as explained in above. then management will be same as low tone bladder and hypotonicity sphincters

Low tone bladder and hypotonicity sphincters

clinical features- urine leaks during exercise low tone bladder- It may be due to over distention of bladder during spinal shock phase. Early CIC can reduce the chance of flaccid bladder,

α blockers have been shown to improve compliance of bladder.

Credes/ valsava method= press down and medially over lower abdomen so that urine will be milking out

Indication- Normally post void urine amount is 50 to 100 cc but young person there should not be any post void residual urine left out. If PVR is more than 100cc credes manoeuvre is not indicated. It is used only in autonomous bladder.

Artificial sphincters can be replaced with low tone sphincters.

Pad can be used for incontinence in female SCI.

Indwelling catheter- It is used in acute phase. CIC is choice of treatment.

Suprapubic catheter can indicated in person with incomplete cervical spinal cord injury and young paraplegic for sex and where CIC cannot be used.

Electrical stimulation- Non-invasive electrical therapy modulated hyperreflexic bladder and stimulate hypocontractile bladder.

bladder inhibition can be achieved by various way such as direct stimulation of the sacral roots, pudendal nerve itself, indirectly by the dorsal penile/clitoral nerve and anal or vaginal mucosa activating its branch.

Electrical current parameters such as Time-20-30 minutes, frequency -5-10 HZ and pulse duration-0.2ms are found to be most efficient.

Clip electrode in female and ring electrode in male would be preferable to stimulating pudendal nerve.
If non-invasive treatment fails then invasive sacral modulation is considered.

Intravesical electro stimulation (IVES) discovered by kotona et al in 1951. It will stimulate mechanoreceptors afferents there by activated micturition reflex. Prerequisites for successful IVES are incomplete nerve lesion, some contraction of detrusor.

IFT and kigel exercises can be used for stimulating pelvic floor muscles in stress incontinence.

Bowel management

IF SCI above conus medularis- anal sphincters becomes spastic. voluntary control is lost but reflex activity is intact. IF SCI below conus medularis- anal sphincters becomes flaccid. voluntary and reflex activity is lost

Decrease colonic motility leads to constipation

Pharmacological agents

Stool softeners, Bulk formers, peristaltic stimulants and laxatives can used In SCI person.

Diet- high fibre diet.(vegetables, fruits, grains, cereals)

Bowel program

sitting position and if it is performed in bed the preferred position would be right side lying position. Gravity and normal curvature help in both positions

Areflexic bowel function- program twice daily

manual removal of stool.

Reflex bowel-Irritants suppository followed by digital stimulation

Digital stimulation-is insertion of gloved finger by individuals or care givers into rectum followed by rotation of finger to stimulate rectal wall for about 15-20 seconds. Repeat the same in every 10 minutes.

if this program fail then enema continence catheter is given enema for emptying.
Recreation and sport activities

It is reasonable to expect that for people with SCI, leisure activities and recreation are more important to overall quality of life (QOL) than they are for the able-bodied. Often, there also are shifts within leisure activities, with a decrease in sports participation and increases in indoors and sedentary activities, especially watching TV and listening to radio and music. There is much evidence that for people with SCI, frequent participation in leisure activities, especially sports and other active pursuits, has a positive association with life satisfaction, self esteem, and mood state. SCI persons can perform following activities such as Art, dance, fishing, videogame, weight lifting, medicine ball throwing.

Medicine ball throwing

**Functional electrical stimulation**

Clinically, a significant number of individuals with so-called complete SCI retain some connectivity across injury site; this could be represented by nonfunctioning myelin or denuded axons that could potentially provide conductivity across injury site given optimal activation. In patients with complete or incomplete SCI, there is now proof of FES-induced activation of the central pattern generator mechanism, and increased stepping responses have been observed regularly treated with FES demonstrated improvement lower limb ASIA motor and sensory scores, and decreased spasticity, indicating some degree of neuromodulation and remediation of paralysis in response to stimulation. Electrically induced exercise like leg cycling, leg cycling with upper extremity assist (hybrids), rowing, arm ergometry can be used in person with complete or incomplete SCI. Higher spinal cord injury patients can stand and walk with help of electrical stimulation, it can also be used to assist with breathing, grasping.
leg cycling s. Surface electrodes can be applied to gluteal, quadriceps, and hamstrings muscles of both legs to operate cycling. Stimulation intensity can be gradually increased to a maximum of 140 mA, pulse width of 0.3 – 0.5ms and frequency of 35Hz. 60 minutes of leg cycling four days a week for 12 week Example are the MOTOmed Letto cycle with a Hasomed RehaStim FES Unit and RT300 bike.

Increase voluntary as well as electrically stimulated isometric strength and endurance are also found in incomplete SCI. left ventricular atrophy reported in people of tetraplegia and osteopenia in lumbar spine and proximal tibia can be reversed following training. increase of circulation in lower extremities also noted with cycling.

Evidence

- Intensive FES cycling training of 48 hours a week can provide the recommended weekly exercise caloric expenditure essential to induce persistent health benefits and muscle and bone effects.
- The effects of the FES cycling are more evident with increased intensity and duration of use.
- The beneficial effects from the uptake of exercise diminish once the FES cycling ceases.

FES cycling with passive cycling showed only cardiovascular benefits. There is evidence that the use of FES bikes in conjunction with conventional therapeutic interventions (e.g. arm crank ergometry including Saratoga) can improve cardiovascular health in people with SCI.

Bipedal Ambulation-Motor complete SCI patients can walk with electrical stimulation. It is called neuroprothesis. surface electrodes can be used to stimulate quadriceps and glutus maximus for upright position and stimulation of common peroneal nerve at level of head of fibula can elicit flexion withdrawal reflex which is helpful in stepping motion. Rate of electrically stimulated walking is slow and distance of ambulation is limited but 1 mile have been achieved in some patients following training.
Voluntary Arm exercise training- It improves general fitness level in SCI by increasing peak oxygen uptake. The improvement of endurance and work capacity of subject is dependent on level of lesion. Special measures can be taken to fix hand on ergometer in person with low tetraplegia.

Neuromuscular electrical stimulation (NMES)- The large diameter neuron such as alpha motor neurones which has lowest threshold activates first then small diameter neuron, such as C fibres. Normally small fibres recruit first followed by large muscles fibres. Disused atrophy can convert type I fibres to type II. But it can be reversed with NMES. The frequency range of 12–18 Hz upper limb and 20-25 Hz in lower limb recommend for smooth contraction but less fatigue of muscle.

NESS H200 (formerly HandMaster-NMS-1)

The NESS H200 developed by Nathan et al., and produced by Neuromuscular Electrical Stimulator Systems, Ra’anana, Israel is the only commercially available upper limb surface FES system (Ragnarsson 2008). It has been FDA approved for use with stroke patients. It is predominantly used as an exercise tool for stroke subjects and is commercially available in a limited number of countries (Popovic et al. 2002). The NESS H200 has three surface stimulation channels used to generate grasping function in tetraplegic and stroke subjects. One channel is used to stimulate extensor digitorum communis muscle at the volar side of the forearm. The second channel stimulates the flexor digitorium superficialis and profundus muscles. The third stimulation channel generates thumb opposition. The system is controlled with a push button that triggers the hand opening and closing functions. The system is easy to don and doff. However, it does have some limitations in its design. The system is limited by not enough sufficient flexibility to vary the position of the electrodes for stimulation of the finger flexors for grasp; it is a stiff orthosis that fixes the wrist joint angle and prevents full supination of the forearm (Popovic et al. 2002).

Bionic Glove

Developed by Prochazka and colleagues at the University of Alberta the Bionic Glove improves hand function in people with SCI. This device uses three channels of electrical
stimulation to stimulate finger flexors, extensors and thumb flexors. The control signal comes from a wrist position transducer mounted in the garment. The actual functioning of the device can be described as greatly augmenting tenodesis (Prochazka et al. 1997; Popovic et al. 2005).

The Bionic Glove is designed to enhance the tenodesis grasp in subjects that have a voluntary control over the wrist (flexion and extension). Stimulates finger flexors and extensors during tenodesis grasp, enhances strength of grasp. The Bionic Glove is available at the University of Alberta, Alberta, Canada and used primarily for clinical evaluation. A modified version of this device will be called Tetron (Popovic et al. 2002).

Overall acceptance rate for long-term use is reported in 30% of potential users. Functions of power grasp and handling of big objects were significantly improved (Popovic et al. 2002). There have been several identified concerns with the device that include damage to the stimulator located on the forearm that is frequently damaged through accidental contact during functional activities and the transducer mechanism is delicate and has to be replaced frequently (Popovic et al. 2001).

ETHZ-ParaCare System

The ETHZ-Para Care System was developed collaboratively between ParaCare, the University Hospital Zurich, the Rehabilitation Engineering Group at Swiss Federal Institute of Technology Zurich and Compex SA, Switzerland. The system was designed to improve grasping and walking function in SCI and stroke patients. Surface stimulation FES system is programmable, with 4 stimulation channels and can be interfaced with any sensor or sensory system. The system provides both palmar and lateral grasps. The device has some reported disadvantage that includes a lengthy time to don and doff the device (7-10 minutes) and it is not commercially available. The next generation of the device will be called the Compex Motion (Popovic et al. 2001; Popovic et al. 2005). The Compex Motion device is currently available in clinical trials with approximately 80 units available. The Compex Motion stimulator was designed to serve as a hardware platform for the development of diverse FES systems that apply transcutaneous (surface) stimulation technology. One of the main designs in this system is that it is easily programmable (Popovic et al. 2005).
Home exercise programme

Check your environment - Choose a spot in your home that is spacious and clear of obstacles.

Warm-Up - Remember to do light endurance work before your strength or flexibility exercises to ensure your muscles are warm.

Cool-Down - Complete some gentle stretching at the end of your routine and ensure that your breathing has returned to normal before you stop.

KEEP HYDRATED! - Be sure to drink lots of fluids while partaking in activities and after you
Few Exercises for spinal cord injury patient

Exercise for Tetraplegic

Rolling
Lateral Weight shift

Prone push up

shoulder flexor strengthening exercise
Self stretching of hamstring

Exercise for Paraplegic
Side Bridging

Latismus Strengthening
Elbow flexor strengthening

Shoulder muscle strengthening

Sitting Balance Exercise

Standing Balance
WHAT HAPPENS WHEN A PERSON SUFFERS FROM SCI

- Spinal cord injury is a disabling condition results in severe physical dysfunction, from which the patient expect to recover soon within few days.
- But the disabilities in spinal trauma are for a much longer duration and usually full recovery is very rare.
- When the patient realizes this fact, this in turn greatly affects the psychological, social and emotional well-being of the person.
- Psychological reactions to SCI are similar to mourning & other situations of severe loss.
- As the famous physicist Stephen Hawking says “IF ONE IS PHYSICALLY DISABLE ONE CAN’T AFFORD TO BE PSYCHOLOGICALLY DISABLED ASWELL”.
- So each of this reaction play a significant role in either fostering or hindering the recovery.

ROLE OF OT
As OT’S “we choose not to put ‘DIS’ in your ability”.

- OT start their intervention at a very grassroot level aiming at maximising the residual physical ability which in turn gives a sense of satisfaction and enhance the confidence of the patient, thus ultimately regaining psychological and emotional stability.
- Whalley Hamel suggests independence is not a physical ability but more of an attitude in which an individual takes on responsibility, solves problem and establishes goals.
- And in occupational therapy that’s exactly what we do, i.e mobilise our patient towards an independent and self-satisfying life.
HOW DOES AN OTst WORK

- OT interventions are:
  - Individualised, Client centered and goal oriented
  - The goal of OTst is to give remedial therapy based on scientifically designed activity that enhance the present capacity of the patient for his/her functional performance
  - When the remedial therapy is not enough to achieve functional independence, we consider compensatory techniques by adapting alternative method and adaptive equipments to assist them with personal care, domestic task and communication
  - OTst design various enabling and purposeful activity that focus on achieving bed mobility, static and dynamic balance and lost motor function and use reinforcements to encourage active participation.

- Transfer and Mobility – OTst train the caregivers aswell as the patient for safe transfer techniques and refer various transfer modalities such as sliding boards,transfer belt etc. if needed.
- ADL - Once the patient achieve functional abilities, OTst train them to be able to perform some of her ADL independently using assistive devices or with minimum external support
- The role of OTst doesn’t end after providing adaptive devices, because its much more important to train the patient to be able to use them effectively in their daily activities
• Social Reintegration is the ultimate aim of total Rehabilitation

• OTs aim at achieving social reintegration by helping the patient to return to his/her job and leisure activities.

• OTs provide various simulated activities to prepare the patient for return to job

• Its important to assess the patient’s home and job site before planning for his return to previous life and do necessary home modification(such as nonskide floor, ramps, switchboards within arm reach, wide doorway for wheelchair accessibility, etc.) train mobility including wheelchair and driving and transfer techniques.

• If the patients present status doesn’t allow for return to the previous job and leisure activities, OTs explore new areas of interest in collaboration with the patient.

  For eg. :- a person who has interest in music and was playing piano prior to her SCI resulting in quadriplegia can be presented with an option of playing mouth organ.

Therefore OT is a KEY rehabilitation discipline, the goal of which is to assist persons in recovering function & facilitate return to productive and fulfilling life after SCI

A PATIENT’S PERSPECTIVE
Sushree Sarangi, a 22 yr old aspiring doctor, met with a road traffic accident on & suffered from C5-6 spinal trauma. She came to OT department of SVNIRTAR for treatment on . While she first came to the department she was dependant on her caregivers o a maximum extent but as very eager to be independent as soon as possible. After assessing her thoroughly, her remaining abilities were considered and a treatment regime was prepared. Initially her sitting tolerance and static as well as dynamic balance was poor which prevented her from being able to sit for a long duration to perform activities. At first we focussed on her bed mobility. As she is a
quadriplegic, it was our aim to teach her to mobilise in bed with minimum external assistance. Mat activities were designed to help her shift her weight that assisted her in rolling.

Upper body strengthening and trunk balance was done so that she was able to maintain sitting position. After she was able to maintain static sitting, weight shifting activities were planned to improve dynamic sitting balance.

After her balance improved, strengthening program for large joints of upper extremity was begin to compensate for the small muscles of hand to some extent. It was continued along with teaching easy and safe transfer techniques in which she participated actively to help in transfer. Attention was paid to regain active wrist flexion and extension which assisted in tenodesis grasp. Some finger tightness was allowed to develop and now she is able to hold objects using active tenodesis. She is able hold few objects with lateral pinch also although the force is minimal. The OTist provided her a writing device after seeing her enthusiasm to go back to her studies. At first her handwriting was poor speed was also not good. But with proper training, now she is able to write a page in 5-6 mins and her handwriting has improved a lot.
At present she is performing some basic ADL such as feeding and grooming independently and other ADLs with minimum assistance.

She was too eager to share her story with the world as soon as she discovered her ability to write with the help of the writing device and we decided to be a stepping stone for her to tell her story to the world.
Pressure ulcers in Spinal cord injury

Introduction

pressure ulcers, also called decubitus ulcers, bedsores, or pressure sores, are localized injury to the skin or underlying tissue, usually over a bony prominence, as a result of unrelieved pressure, applied with great force over a short period (or with less force over a longer period), that disrupts blood supply to the capillary network, impeding blood flow and depriving tissues of oxygen and nutrients. This external pressure must be greater than arterial capillary pressure to lead to inflow impairment and resultant local ischemia and tissue damage. Pressure ulcers, range in severity from reddening of the skin to severe, deep craters with exposed muscle or bone. Pressure ulcers significantly threaten the well-being of patients with limited mobility

Aetiological factors:

1. Pressure, or the compression of tissues: In most cases, this compression is caused by the force of bone against a surface, as when a patient remains in a single decubitus position for a lengthy period. After an extended amount of time with decreased tissue perfusion, ischemia occurs and can lead to tissue necrosis if left untreated. Pressure can also be exerted by external devices, such as medical devices, braces, wheelchairs, etc.

2. Shearing force: A force created when the skin of a patient stays in one place as the deep fascia and skeletal muscle slide down with gravity, can also cause the pinching off of blood vessels which may lead to ischemia and tissue necrosis. Friction is related to shear but is considered less important in causing pressure ulcers.

3. Temperature and moisture of the skin: Moisture on the skin causes the skin to lose the dry outer layer and reduces the tolerance of the skin for pressure and shear. The situation may be aggravated by other conditions such as excess moisture from incontinence, perspiration, or exudate. Temperature is also a very important factor. The cutaneous metabolic demand rises by 13% for every 1°C rise in cutaneous temperature. When supply can't meet demand, ischemia then occurs.

Pathophysiology
Pressure ulcers may be caused by inadequate blood supply to the tissue because of constant unrelieved pressure over a particular area with impaired sensation. Within 2 hours, this shortage of blood supply, called ischemia, may lead to tissue damage and cell death. The sore will initially start as a red, painful area. The other process of pressure ulcer development is seen when pressure is high enough to damage the cell membrane of muscle cells. The muscle cells die as a result and skin fed through blood vessels coming through the muscle die. This is the deep tissue injury form of pressure ulcers and begins as purple intact skin. Biofilm is one of the most common reasons for delayed healing in pressure ulcers. Biofilm occurs rapidly in wounds and stalls healing by keeping the wound inflamed. Frequent debridement and antimicrobial dressings are needed to control the biofilm. Common sites affected by pressure sores are: over ischial tuberosity, sacrum, over trochanter, in heel, over heads of metatarsals, buttocks, over shoulder, and over occiput.

**Stages of pressure sore**

- **Stage I**: Intact skin with non-blanchable redness of a localized area usually over a bony prominence. Darkly pigmented skin may not have visible blanching, difficult to detect in individuals with dark skin tones. Its color may differ from the surrounding area. The area may be painful, firm, soft, warmer or cooler as compared to adjacent tissue.

- **Stage II**: Partial thickness loss of dermis presenting as a shallow open ulcer with a red pink wound bed, without slough. May also present as an intact or open/ruptured serum-filled blister. Presents as a shiny or dry shallow ulcer without slough or bruising.

- **Stage III**: Full thickness tissue loss. Subcutaneous fat may be visible but bone, tendon or muscle are not exposed. Slough may be present but does not obscure the depth of tissue loss. May include undermining and tunneling. The depth of a stage III pressure ulcer varies by anatomical location.

- **Stage IV**: Full thickness tissue loss with exposed bone, tendon or muscle. Slough or eschar may be present on some parts of the wound bed. Often include undermining and tunneling. The depth of a stage IV pressure ulcer varies by anatomical location. The bridge of the nose, ear, occiput and malleolus do not have (adipose) subcutaneous tissue and these ulcers can be shallow. Stage IV ulcers can extend into muscle and/or...
supporting structures (e.g., fascia, tendon or joint capsule) making osteomyelitis likely to occur.

- **Unstageable**: Full thickness tissue loss in which actual depth of the ulcer is completely obscured by slough (yellow, tan, gray, green or brown) and/or eschar (tan, brown or black) in the wound bed. Until enough slough and/or eschar is removed to expose the base of the wound, the true depth, and therefore stage, cannot be determined. Stable (dry, adherent, intact without erythema or fluctuance) eschar on the heels serves as “the body’s natural (biological) cover” and should not be removed.

![Stage - I](image1.png) ![Stage- II](image2.png)

![Stage- III](image3.png) ![Stage - IV](image4.png)

**Prevention**

The most important care for patients at risk for pressure ulcer is the redistribution of pressure so that no pressure is applied to vulnerable areas of pressure ulcer. In the 1940s
Ludwig Guttmann introduced a program of turning paraplegics every two hours thus allowing bedsores to heal. Preventive measures should be used in at-risk patients.

**Pressure reduction to preserve microcirculation** is a mainstay of preventive therapy. There is no evidence to determine an optimal patient repositioning schedule, and schedules may need to be determined empirically. According to recommendations from the Agency for Health Care Policy and Research, patients who are bedridden should be repositioned every two hours. To minimize shear, the head of the bed should not be elevated more than 30 degrees and should be maintained at the lowest degree of elevation needed to prevent other medical complications. Pressure-reducing devices can reduce pressure or relieve pressure (i.e., lower tissue pressure to less than the capillary closing pressure of 32 mm Hg) and are classified as static (stationary) or dynamic. Static devices include foam, water, gel, and air mattresses or mattress overlays. Dynamic devices, such as alternating pressure devices and low–air-loss and air-fluidized surfaces, use a power source to redistribute localized pressure. Dynamic devices are generally noisy and more expensive than static devices. Pressure-reducing surfaces lower ulcer incidence by 60 percent compared with standard hospital mattresses.

For individuals with paralysis, pressure shifting on a regular basis and using a wheelchair cushion featuring pressure relief components can help prevent pressure wounds.

Controlling the heat and moisture levels of the skin surface, known as skin microclimate management, also plays a significant role in the prevention and control of pressure ulcers.

**Good diet and drinking enough water** is very important. It is particularly important for people at risk of developing a pressure ulcer or those with a pressure ulcer as their condition can get worse or fail to get better without it. In addition, adequate intake of protein and calories is important. vitamin C has been shown to reduce the risk of pressure ulcers. People with higher intakes of vitamin C have a lower frequency of bed sores in bed-ridden patients than those with lower intakes. If unable to maintain proper nutrition through protein and calorie intake, it is advised to use supplements to support the proper nutrition levels. Although poor nutrition is associated with pressure ulcers, a causal relationship has not been established. One large trial has shown that oral nutritional supplementation reduces risk, but several other trials have not.
Skin care is also important because damaged skin does not tolerate pressure. However, skin that is damaged by exposure to urine or stool is not considered a pressure ulcer. These skin wounds should be classified as Incontinence Associated Dermatitis.

Treatment

Debridement

Necrotic tissue should be removed in most pressure ulcers. The heel is an exception in many cases when the limb is poorly perfused. Necrotic tissue is an ideal area for bacterial growth, which has the ability to greatly compromise wound healing.

There are five ways to remove necrotic tissue.

1. Autolytic debridement is the use of moist dressings to promote autolysis with the body's own enzymes and white blood cells. It is a slow process, but mostly painless, and is most effective in patients with good immune systems.
2. Biological debridement, or maggot debridement therapy, is the use of medical maggots to feed on necrotic tissue and therefore clean the wound of excess bacteria.
3. Chemical debridement, or enzymatic debridement, is the use of prescribed enzymes that promote the removal of necrotic tissue.
4. Mechanical debridement, is the use of debriding dressings, whirlpool or ultrasound for slough in a stable wound
5. Surgical debridement, or sharp debridement, is the fastest method, as it allows a surgeon to quickly remove dead tissue.

Pressure ulcer intervention

Patients with pressure ulcers should not lie or sit on them. Continued pressure reduces the blood flow to a wound that is trying to heal. Specific wound care for pressure ulcers includes the following:

Stage I pressure ulcers: Remove all pressure from the ulcer. No topical therapies have been shown to aid healing.
Stage II pressure ulcers: Cover the wound bed with hydrocolloid or foam dressings. If the ulcer is on the buttocks, be certain the wound bed is clean before applying the dressings and replace the dressing if the wound bed becomes contaminated from urine or stool beneath the dressing. Skin care products that can be liberally applied to the ulcer are also an alternative to dressings and work well for the incontinent patient.

Stage III or IV ulcers: (Fig:1) once the necrotic tissue has been removed, fill the wound bed with a moisture retentive dressing or gel product to facilitate healing. Apply a cover dressing to hold the dressing or gel in place. Negative pressure wound therapy applied to the wound bed may also be used to improve granulation tissue formation in the pressure ulcer especially following surgical debridement. This technique uses foam or gauze placed into the wound cavity which is then covered in a film which creates an airtight seal. Once this seal is established, the negative pressure removes exudate and edema from the wound and stimulates blood supply to produce granulation tissue, capillary buds that begin the healing process in full-thickness ulcers. There are, unfortunately, contraindications to the use of negative pressure therapy. Most deal with the unprepared patient, one who has not gone through the previous steps toward recovery, but there are also wound characteristics that bar a patient from participating: a wound with inadequate circulation, a raw debrided wound, a wound with necrotized tissue and eschar, and a fibrotic wound or signs of cancer in the wound. After negative pressure wound therapy the patient should be reevaluated every two weeks to determine future therapy.

Clean full thickness pressure ulcers can be closed with surgery using tissue flaps like free flap, musculocutaneous, fasciocutaneous, or perforator-based flaps. Though musculocutaneous flaps have become the first choice in the surgical repair of pressure sores but fasciocutaneous flaps are also equally good (Fig:2). There is no statistically significant difference with regard to recurrence or complication rates among musculocutaneous, fasciocutaneous, or perforator-based flaps. Following these operations, no pressure or tension (pulling) can be applied to the flap while it is healing. Often the patient has to begin sitting up in short time increments to allow inspection of the flap for signs of pressure ulcer (unblanchable redness, pallor, incisional separation). Specialty low-air loss beds or air-fluidized beds are often required to promote healing of the flap in a low pressure environment.
Complications

Pressure ulcers can trigger other ailments, cause patients considerable suffering, and be expensive to treat. Some complications include autonomic dysreflexia, bladder distension, osteomyelitis, pyarthroses, sepsis, amyloidosis, anemia, urethral fistula, gangrene and very rarely malignant transformation (Marjolin's ulcer - secondary carcinomas in chronic wounds). Sores often recur because patients do not follow recommended treatment or develop seromas, hematomas, infections, or dehiscence. Patients with paralysis are the most likely to have pressure sores recur. In some cases, complications from pressure sores can be life-threatening. The most common causes of fatality stem from renal failure and amyloidosis.

References:

1. ThiessenFEAndrades et al. J PlastReconstrAesthetSurg.Flap surgery for pressure sores: should the underlying muscle be transferred or not?- 2011 Jan;64(1):84-90
2. Sameem M, Au M, Wood T, Farrokhyar F, Mahoney JA systematic review of complication and recurrence rates of musculocutaneous, fasciocutaneous, and


