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Traumatic Spinal Cord Injury: Recovery, Rehabilitation, and Prognosis

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Abstract The impact of traumatic spinal cord injury (TSCI) on function and quality of life (QOL) is substantial, and the incidence of people living with complete TSCI is increasing over time as our emergent and critical care capabilities improve. Much of TSCI treatment and prognosis is based on initial assessment of the neurological level of injury (NLI), which is followed throughout the continuum of rehabilitation. Medical treatment and surveillance of the patient's vulnerable organs begins in the intensive care unit and continues into acute rehabilitation, with pulmonary, cardiovascular, integumentary, genitourinary, and gastrointestinal as priority systems. The multidisciplinary rehabilitation team collaborates to help patients achieve specific functional goals. Determining outcome and prognosis for ambulation in TSCI is based upon completeness of injury and NLI, as well as other variables. Traditional TSCI rehabilitation techniques when done in concert with new methods, i.e., augmentative motor devices and novel neuromedical treatments such as stem cell work, continues to expand the possibilities for QOL for this challenging patient population.

Keywords Rehabilitation · Spinal cord injury · Recovery · Prognosis

This article is part of the Topical Collection on *Blunt Spinal Trauma*

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Introduction

The functional, psychological, and financial impacts of traumatic spinal cord injury (TSCI) are broad [1]. Trauma centers utilize multidisciplinary expertise in both acute surgical and medical management and bridge these complicated patients to specialized rehabilitation centers following early stages of recovery and mobilization. The principles of projection of specific functional goals or realistic forecasting of patient's abilities upon home transition following weeks to months of inpatient rehabilitation remain largely unchanged, in that much of what we know about prognosis is dictated both neurological level of injury (NLI) and the completeness of injury [2•].

There has indeed been progress in neurological rehabilitation of TSCI with the advances in assisted ambulation, functional electrical stimulation, assistive technology, and medical management of associated systems and complications of immobility. The goal of this report is to provide an overview of the acute to subacute journey for those with TSCI as they transition toward a functional "new normal" with respect to mobility, activities of daily living (ADLs), and community and social reintegration. A recovery continuum emphasizing diagnosis, rehabilitation treatment, prognostic education, and realistic goals for patients with TSCI is discussed.

Defining the Population

Affected patients with TSCI are typically in school and early career-aged, and as of 2010, the average age has increased to 42, with about 80 % male predominance. Patients are surviving polytrauma of higher severity in larger numbers due in large part to advances in neurotrauma critical care is improving, and overall this patient population is older that it was even 10 years ago. Roughly 10,000 people in the USA sustain TSCI each year and currently it is estimated that about 300, 000 people live in our communities following TSCI. Motor vehicle-related TSCI makes up 37 %, which has slowly decreased over the past three decades due to prevention efforts and technology, and falls have increased over time to 29 % with perhaps a more functional, yet aging population. Violence (majority from gunshot wounds) amounts to 13 %, and sports-related injury 9 %, with 11 % "other or unknown" mechanism. Sixty percent of all TSCI is classified as incomplete injury and 30 % complete injury, with 53 % of all injuries affecting all four limbs, classified as tetraplegia [3].

Recovery and the Initial Patient Evaluation

The American Spinal Injury Association (ASIA) exam should be completed within 72 h post injury, and has been developed and validated with high inter-rater reliability (Figs. 1 and 2). Subsequent exams are often repeated throughout the recovery process [4••]. With an appropriate mental status, the initial exam is performed with a safety pin and a cotton-tipped swab. The exam defines the NLI and the completeness of the injury, offering prognostic information regarding current and future functional goals (typically at 1 year). The patient is always examined supine and with the same systematic approach to all dermatomes and myotomes in both upper and lower extremities, with the NLI determined by the most caudal level possessing both intact *sensory* and 3/5 (antigravity) or greater *motor* strength bilaterally. With any preservation of sensation in the S4-5 segments (ano-rectal) and/or motor function manifested by *voluntary* external anal sphincter contraction, the TSCI is deemed "incomplete" [4••].

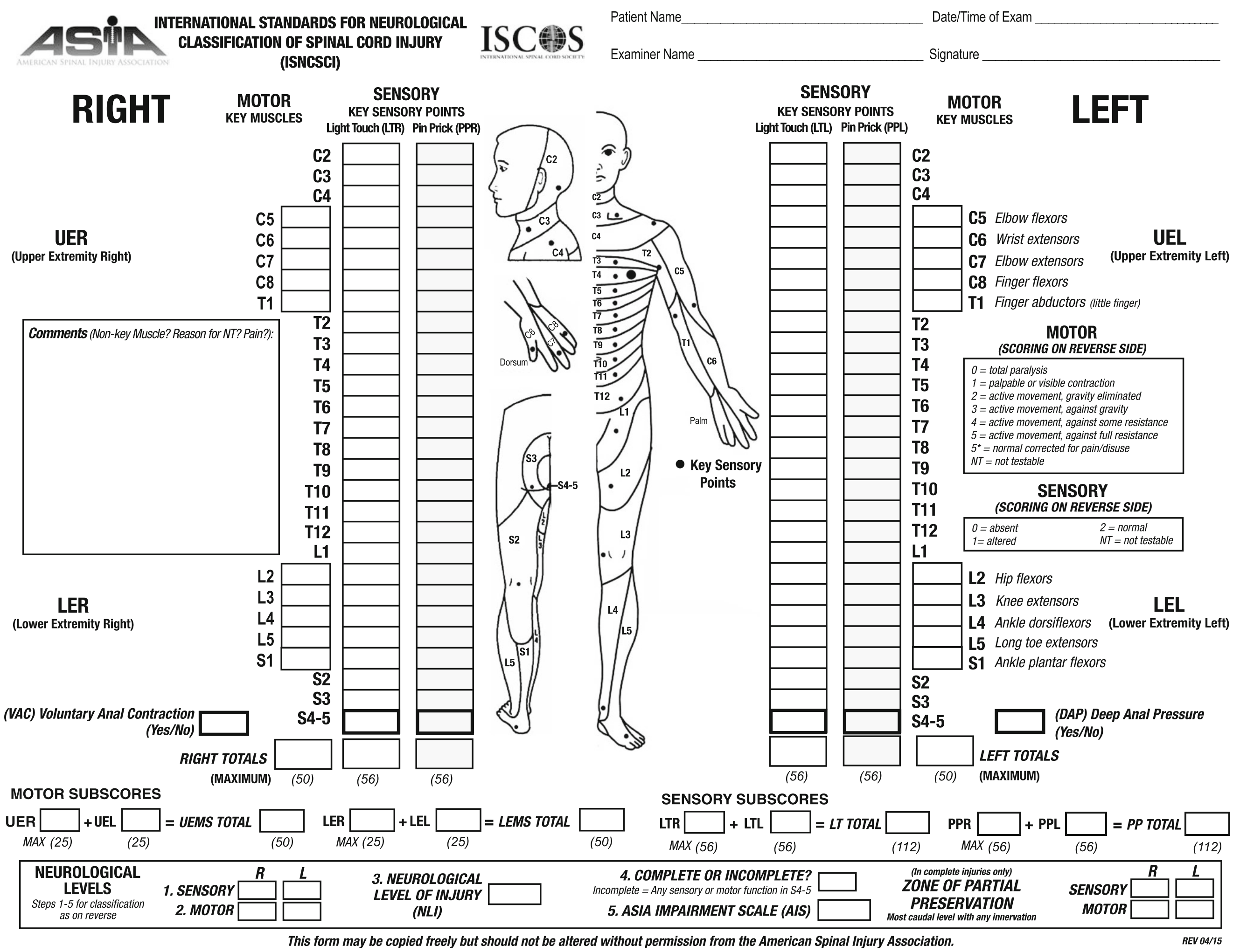
Recognizing Early TSCI Review of Systems

Whether in the intensive care unit (ICU) or the inpatient rehabilitation facility (IRF), recovery in TSCI is a continuum, and goals are based on patient stability and how far along the patient is from injury. Three major categories of focused rehabilitative efforts during early recovery are *pulmonary management*, *mobilization* (as relates to skin protection, spasticity, and thromboembolic phenomena), and *neurogenic bowel/bladder care*.

In terms of weaning the patient from the ventilator, tetraplegia is not synonymous with dependency on mechanical ventilation. The patient with a complete C4 tetraplegia has roughly an 80 % chance of weaning from the ventilator. Even a complete C3 patient potentially has a 60 % chance of weaning after the first 2–4 weeks of acute care [5]. During the initial days to weeks, secretions develop from the parasympathetic surge, and cough is absent or significantly weak. Therefore, cough-assist or mechanical insufflator-exsufflator (M-IE) devices, "quad-cough" with clinician assistance, and abdominal binders all support pulmonary toilet and clearance. Vital capacity is markedly reduced due to respiratory fatigue in all complete cervical patients, and due to the combination of secretions and inefficient ventilation, 85 % of complete C1–C4 and 50 % C5–C8 patients develop pneumonia and/or atelectasis within the first month from injury [6, 7].

Complete C1 and C2 patients will be apneic. For these patients, the option of a diaphragmatic pacing system (DPS) has become possible, provided that the phrenic nerve is determined to be intact. The DPS has been tested in both chronic high cervical tetraplegia as well as with acute patients prior to rehabilitation. Although variable reports in efficacy, the majority of paced patients have the ability to be partially (several hours per day) or completely ventilator-free. Given that electrodes are placed directly at the motor unit junction of the bilateral hemidiaphragm as opposed to the traditional direct-to-phrenic nerve placement, this is a less invasive (laparoscopic), well-tolerated approach [8].

Mobilization in the ICU for all patients is essential for both deconditioning and building cardiopulmonary reserve. The TSCI population specifically benefits from early mobility exercises to prevent contractures from spasm [9, 10]. Especially for patients with flaccid tetraplegia, the risk of ischial and sacral decubitus ulcers is significant from day 1 of injury and therefore frequent turning and positioning is vital in the ICU [11]. Basic goals of communication for high and mid-cervical patients (i.e., "sip and puff" call light proficiency and head control communication systems) are difficult initial hurdles but essential for care and recovery. High level motor goals of sitting up and chair transfers enable patients to achieve confidence in their unique needs, articulating to providers and family the steps required to move and be moved. Neurogenic bladder is treated immediately with indwelling catheter regardless of completeness of injury. Once fluid management is stable, intermittent straight catheterization is instituted. "Bowel programs" or regular bowel medications along with non-pharmacological regimens are typically formalized in SCI rehabilitation. Once a patient is eating and/or tolerating tube feeding and forming stools, standing AM or PM suppositories and stool softeners are introduced.



Figs. 1 and 2 ASIA exam sheet and scoring sheet for the International Standards for Neurological Classification of Spinal Cord Injury. (Used with permission American Spinal Injury Association: International Standards for Neurological Classification of Spinal Cord Injury, revised 2013; Atlanta, GA. Reprinted 2015)

Preparation and Beginning the Conversation

The acute change in functioning after TSCI, even in those with excellent functional outcomes, is something that the patient may not adjust to for an extended period of time. Education regarding impairments that indicate eventual disability is a process that begins early in recovery. Initial discussions with patients and caregivers are best introduced with the surgical and rehabilitation medicine specialist. Asking what the patient and/or family may already know about the TSCI is essential for formulating a presentation. There are a myriad of differences in personality, cultural beliefs, and intellectual abilities that may affect how information is received. Retaining vital information at this stage is very often not optimal for both patient and family, and given that roughly 28 % of TSCI patients have sustained some degree of traumatic brain injury, it is important to take into account possible concomitant cognitive impairment [12]. In this light, the initial conversation should be seen as a gateway and not a closed chapter.

Prognosis, reviewed in detail below, will be discussed at several points in time, including into the rehabilitation phase. Communicating prognosis in TCSI should be done with direct eye contact while seated close to the patient and in the presence of supportive family or friends. Being forthcoming without negating hope is essential. The statement “We cannot do anything more for you” is not necessarily accurate, in that goals for less dependence and a good quality of life exist more than the individual likely will realize [13].

Principles of Rehabilitation

Spinal cord injury rehabilitation provides disease-specific care to optimize quality of life, foster independence, and

Muscle Function Grading

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 functional muscle 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 normal ROM)

Sensory Grading

0 = Absent
1 = Altered, either decreased/impaired sensation or hypersensitivity
2 = Normal
NT = Not testable

When to Test Non-Key Muscles:

In a patient with an apparent AIS B classification, non-key muscle functions more than 3 levels below the motor level on each side should be tested to most accurately classify the injury (differentiate between AIS B and C).

Movement	Root level
Shoulder: Flexion, extension, abduction, adduction, internal and external rotation **Elbow:** Supination	C5
Elbow: Pronation **Wrist:** Flexion	C6
Finger: Flexion at proximal joint, extension. **Thumb:** Flexion, extension and abduction in plane of thumb	C7
Finger: Flexion at MCP joint **Thumb:** Opposition, adduction and abduction perpendicular to palm	C8
Finger: Abduction of the index finger	T1
Hip: Adduction	L2
Hip: External rotation	L3
Hip: Extension, abduction, internal rotation **Knee:** Flexion **Ankle:** Inversion and eversion **Toe:** MP and IP extension	L4
Hallux and Toe: DIP and PIP flexion and abduction	L5
Hallux: Adduction	S1

ASIA Impairment Scale (AIS)

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

B = Sensory Incomplete. Sensory but not motor function is preserved below the neurological level and includes the sacral segments S4-5 (light touch or pin prick at S4-5 or deep anal pressure) AND no motor function is preserved more than three levels below the motor level on either side of the body.

C = Motor Incomplete. Motor function is preserved at the most caudal sacral segments for voluntary anal contraction (VAC) OR the patient meets the criteria for sensory incomplete status (sensory function preserved at the most caudal sacral segments (S4-S5) by LT, PP or DAP), and has some sparing of motor function more than three levels below the ipsilateral motor level on either side of the body.
(This includes key or non-key muscle functions to determine motor incomplete status.) For AIS C – less than half of key muscle functions below the single NLI have a muscle grade ≥ 3.

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

E = Normal. If sensation and motor function as tested with the ISNCSCI are graded as normal in all segments, and the patient had prior deficits, then the AIS grade is E. Someone without an initial SCI does not receive an AIS grade.

Using ND: To document the sensory, motor and NLI levels, the ASIA Impairment Scale grade, and/or the zone of partial preservation (ZPP) when they are unable to be determined based on the examination results.

AMERICAN SPINAL INJURY ASSOCIATION

INTERNATIONAL STANDARDS FOR NEUROLOGICAL CLASSIFICATION OF SPINAL CORD INJURY

ISCOS
INTERNATIONAL SPINAL CORD SOCIETY

Steps in Classification

The following order is recommended for determining the classification of individuals with SCI.

1. Determine sensory levels for right and left sides.
The sensory level is the most caudal, intact dermatome for both pin prick and light touch sensation.

2. Determine motor levels for right and left sides.
Defined by the lowest key muscle function that has a grade of at least 3 (on supine testing), providing the key muscle functions represented by segments above that level are judged to be intact (graded as a 5).
Note: in regions where there is no myotome to test, the motor level is presumed to be the same as the sensory level, if testable motor function above that level is also normal.

3. Determine the neurological level of injury (NLI)
This refers to the most caudal segment of the cord with intact sensation and antigravity (3 or more) muscle function strength, provided that there is normal (intact) sensory and motor function rostrally respectively.
The NLI is the most cephalad of the sensory and motor levels determined in steps 1 and 2.

4. Determine whether the injury is Complete or Incomplete.
(i.e. absence or presence of sacral sparing)
*If voluntary anal contraction = **No** AND all S4-5 sensory scores = **0** AND deep anal pressure = **No,** then injury is **Complete.***
*Otherwise, injury is **Incomplete.***

5. Determine ASIA Impairment Scale (AIS) Grade:

Is injury Complete? **If YES, AIS=A** and can record ZPP (lowest dermatome or myotome on each side with some preservation)

NO ↓

Is injury Motor Complete? **If YES, AIS=B**

NO ↓ (No=voluntary anal contraction OR motor function more than three levels below the motor level on a given side, if the patient has sensory incomplete classification)

Are at least half (half or more) of the key muscles below the neurological level of injury graded 3 or better?

NO ↓ **AIS=C**

YES ↓ **AIS=D**

If sensation and motor function is normal in all segments, AIS=E
Note: AIS E is used in follow-up testing when an individual with a documented SCI has recovered normal function. If at initial testing no deficits are found, the individual is neurologically intact; the ASIA Impairment Scale does not apply.

Figs. 1 and 2 (continued)

reintegrate patients into the community. Care is delivered through a multidisciplinary team including Physical Medicine and Rehabilitation physicians and allied health professionals including occupational, speech, physical, respiratory, and recreational therapists; rehabilitation nurses; psychologists; and social workers. Well-equipped centers can also provide custom wheelchair fitting, assistive technology, specialized wound care, sexuality/reproductive education, and opportunities for adaptive sports. Peer volunteers, community support groups, and vocational counseling are vital components of comprehensive rehabilitation in this population.

Rehabilitation for patients with TSCI has evolved over the years and is undergoing a paradigm shift. Historically, therapy was focused on learning compensatory strategies; however, with our increasing understanding of neuroplasticity, goals have shifted toward neuromuscular reeducation and recovery of lost function.

In the acute care hospital, patients with acute SCI are generally seen two to five times per week by therapists in one or more disciplines for 15–60 min sessions. In the acute phase, early goals include upright position tolerance, which can be introduced with sitting exercises, raising the head of the bed, and utilizing a tilt table or standing frame (Fig. 3). These interventions promote early weight-bearing, improved trunk and head control, and vascular compliance in light of the high risk of orthostatic hypotension (OH) and deconditioning/muscle atrophy in this population. Staff also initiates a passive range of motion stretching program in which family can actively participate. Bed mobility, endurance training, and transfer training (bed to chair and chair to commode) are also central components of *early* SCI rehabilitation.

Given the high risk of pressure sores and likelihood of bowel and bladder incontinence, SCI teaching includes helping patients understand the need for weight-shifting maneuvers as well as learning to self-catheterize and insert suppositories to promote bowel and bladder care independence [14]. Therapists and nursing staff play a key role in this process.

During acute rehabilitation, the attention soon shifts to utilization of advanced equipment and emphasis on neuromuscular reeducation. Patients typically receive therapy 3 h per day, 6 days per week by specialized SCI therapists. Functional goals can be set based on NLI (Table 1) [15], though clinicians

Fig. 3 Patient in standing frame with abdominal belt (loose) and tibial support. (Courtesy of Nancy Kelly)

Table 1 Goals for the first 5 months of rehab based on NLI in patients with complete TSCI

Level	Goals
C4	Independent with power wheelchair mobility (sip and puff vs. head array) Partial or full assist ventilation Dependent ADLs
C5	Independent with power wheelchair mobility (joystick/arm control) Can assist with transfers May need extra respiratory care Can assist with some ADLs Adapted driving possible
C6	Independent with manual wheelchair, but may need power for efficiency Assist or independent with transfers using slide board Independent weight shifting Perform some ADLs with equipment Adapted driving possible
C7	Independent community mobility in a manual wheelchair Independent transfers without board Drives car with adaptations
C8–L2	Advanced wheelchair skills—wheelies, curbs, escalator negotiation Transfers without board including floor and Independent ADLs Drives car with adaptations
L3 and below	Possible household and community ambulation with braces and equipment. Independent ADLs Drives car with/without adaptations

Adapted from Field-Fote [15]

must be careful not to limit expectations given patient variability. To achieve these goals, there are a number of treatment tools available to the therapist.

Occupational therapists (OT) focus on ADLs with a range of goals from full independence to establishing compensatory strategies tasks. They utilize equipment to assist with transferring from one surface to another, dressing, bathing, grooming, eating, and preparing food. They often coordinate with nursing to train patients to self-catheterize and perform bowel programs. OTs assist in wheelchair training and often lead the charge for home accessibility modifications including ramps, shower chairs, and other accommodating equipment. Upper extremity strengthening and endurance training and driving adaptations fall under OT treatments as well and are key for wheelchair and vehicle-based mobility.

Speech and language pathologists and respiratory therapists have an important role especially in the treatment of patients with cervical SCI. The goals of these therapies are voice production, secretion clearance, ventilator weaning, resistive expiratory muscle training, and utilization of assisted communication devices. There are a number of communication devices beyond the scope of this review including eye-gaze technology-based systems, type and speak devices, and point and press communication.

Physical therapists are directly engaged in gait training. The rationale for specific gait training following TSCI is to provide the injured nervous system with task-specific sensory input to stimulate the remaining spinal cord networks even when supraspinal input (above the lesion) is compromised [16, 17]. Gait training typically includes some combination of *Body Weight Supported Treadmill Training* (BWSTT) or *robotic-assisted gait training* (Fig. 4). In BWSTT, the patient is connected to a harness and placed on a treadmill on which limb stepping movements are assisted by a therapist. Robotic-assisted gait training uses the same principle, except the power for locomotion is driven mechanically by a device attached externally to the patient's legs. To compliment gait training, electrical stimulation-equipped devices such as bicycles or rowing machines (Fig. 5) are utilized as they promote retention of muscle bulk and improved lower extremity strength.

No clinically significant difference in walking outcome (speed, capacity, independence, safety) has been found upon comparing BWSTT vs. robotic training [18]. Clinically, the decision to utilize one method over another is made based on the safety and physical support the therapist can provide. Increase in corticospinal tract connectivity from training following 3–5 months of daily BWSTT has been demonstrated. The percentage increase of motor-evoked potential was significantly correlated with the degree of locomotor recovery noted by Walking Index for Spinal Cord Injury II (WISC II) score, 6-min walk test distance, and electromyography (EMG), thus demonstrating promising results for intensive walking programs for SCI [19].

Fig. 4 Patient using Lokomat © over a treadmill. Patient wears a body weight support harness around the trunk and straps around the groin that attach to ceiling suspension. This suspension rises allowing unweighting of lower extremities. The Lokomat can control for hip, knee, and ankle range of motion and movement. The patient is able to actively or passively take steps using the system. (Used with permission of Spaulding Rehabilitation Network)

Exoskeletal systems are wearable battery-powered devices that attach to the limbs to provide the power needed for movement. They have recently become available to facilitate gait training for patients with TSCI as well as for home and community use. Aach et al. performed a pilot study of eight patients with traumatic complete and incomplete TSCIs to assess exoskeletal locomotor training and identify beneficial effects on functional mobility. Preliminary results demonstrate statistically significant improvements in timed walk tests, number of steps, Timed Up and Go (TUG) test, and distance covered from pre- to post 90-day training. [20].

Fig. 5 Patient using rowing system attached to a functional electrical stimulator (FES). The FES uses leads placed at key muscles to facilitate muscle contraction for lower extremity movement during rowing. (Used with permission of Spaulding Rehabilitation Network)

Wheelchairs

For patients with minimal or no ambulatory capacity, a custom wheelchair is essential. Power wheelchairs can be highly specific to patients' needs and include those with head control, "sip and puff" breath control (Fig. 6), tongue control (*for C1–C4 injuries*), and joystick control (*for ASIA C5 and below*). Power-assist manual wheelchairs are manual chairs with available powered assistance for propulsion (*for ASIA C5 and below*). Manual wheelchairs (*for ASIA C6 and below*) are lighter-weight but also customizable. Patients with high cervical injuries who are unable to shift their own bodyweight for pressure relief will use manual "tilt-in-space" wheelchairs that rotate on a sagittal axis. For the athletic patient, sport-specific wheelchairs are available as well.

Prognosis

Recovery of ambulation is often the most important self-reported goal of rehabilitation after SCI [21] and has therefore become an increasingly attractive focus of pharmacological and rehabilitative approaches and outcome studies. For reference, functional ambulation in this paper is defined as the ability to walk independently in the community, with or without the use of devices and braces.

Fig. 6 Power wheelchair with head support, custom cushion, and sip and puff attachment at level of mouth. (Used with permission of Spaulding Rehabilitation Network)

Neurologic Level of Injury and AIS Grade

The NLI and ASIA Impairment Scale (AIS) impairment level in the acute stage are generally agreed upon as the most important prognostic factors for functional recovery. Patients with AIS grade A (motor and sensory complete) on initial evaluation are unlikely to recover function below the NLI. In fact, only 20 % of these patients "convert" to AIS B or C though conversion from any grade has not been proven to predict outcomes [22, 23]. If the ASIA examination is performed after 72 h, the percentage of patients improving drops to 2.5 % [24]. AIS A patients with cervical lesions have essentially no chance of functional walking. However, in thoracic and lumbar injury, up to 8 % of patients recover the ability to ambulate, though assistive devices are required and distances are limited [25].

Typically, one third of AIS B (motor complete, sensory incomplete) patients recover ambulatory function but outcomes can vary dramatically [23]. Recent studies have validated older research that showed a relationship between preserved pinprick sensation (in addition to light touch) and recovery of ambulation in this group. This is anatomically reasonable given the proximity of the spinothalamic tracts to the corticospinal tracts in the spinal cord [26].

For AIS C (motor incomplete), the prognosis for functional ambulation is better than sensory incomplete with an overall rate of 80–90 % [22, 23], though age is an extremely important prognostic factor. Patients over age 50 only become successful ambulators 30–40 % of the time [22, 27].

Virtually all patients with AIS D recover ambulatory function, but as those with grade C injuries, age is a poor prognostic factor, decreasing the odds by 20 % [22, 23, 27].

Other Prognostic Factors

Several studies have found that motor scores [especially the lower extremity motor score (LEMS)], younger age, and the presence of pinprick sensation were correlated with better outcomes [28, 29, 30••, 31]. One group found that all incomplete paraplegics with initial LEMS of at least 10 ambulated by 1 year; for paraplegics, this number approached 65 %. More specifically, all patients with initial iliopsoas or quadriceps strength of at least 2 were ambulatory at 1 year follow-up [32, 33]. Timing of strength return is important as well; if quadriceps strength did not show improvement by month 2, chance of ambulation decreased to 25 % [34].

Magnetic resonance imaging (MRI) is now routinely acquired with prognostic relevance in TSCI. In general, hemorrhagic lesions are almost always associated with complete injuries (Ramón et al. 1997). On the other hand, cord edema is usually a positive prognostic sign, typically associated with milder, incomplete injuries. Further, if edema is limited to only one level, most patients convert to less severe AIS grades [35–37].

Electrophysiologic tests can also be helpful prognostically. As might be expected, motor-evoked potentials (MEPs) are more predictive of ambulatory potential than sensory-evoked potentials (SSEPs) given that they can better detect motor tract. One group found that all patients with initial MEPs present recovered at least antigravity strength in the muscles tested [38].

Medical Issues in Acute Rehabilitation

In the SCI rehabilitation setting, chronic medical complications are common and can negatively affect health, functional independence, and quality of life (QOL). Prevention, early diagnosis, and prompt treatment of these issues are key in maximizing patient outcomes [39•].

Pulmonary

Respiratory complications associated with SCI are the most important cause of morbidity and mortality in this population [40, 41]. After TSCI, a variety of respiratory function deficits develop including reduced vital capacity and chest wall compliance, insufficient respiratory muscles, ineffective cough, and excess oxygen cost of breathing [42]. Obesity, smoking, and duration of injury may further contribute to compromised respiratory function [43, 44]. Higher level SCI and lower motor scores place patient at risk for increasingly serious respiratory complications including atelectasis, pneumonia, respiratory failure, and sleep-related breathing disorders, most commonly obstructive sleep apnea. Of note, pneumonia is cited as the primary cause of death during chronic SCI [45]. Every effort should be directed at prevention of respiratory complications including proper positioning and postural changes, breathing techniques, spontaneous cough and cough assistance, secretion management, respiratory muscle training, ventilation techniques and education, vaccinations, and pharmacological interventions.

Cardiovascular

Individuals with SCI also are at high risk of multiple cardiovascular complications including thromboembolism, autonomic dysreflexia, OH, impaired cardiovascular reflexes and sensation of cardiac pain, and loss of and cardiac atrophy [46–48].

Autonomic dysreflexia (AD) typically occurs in complete SCI with lesions at T6 and above. Symptoms are due to a spinal reflex mechanism typically initiated by noxious stimulus (e.g., bladder distention) below the level of injury. The stimulus leads to sympathetic overactivity with vasoconstriction below the neurological lesion, particularly in the splanchnic circulation. Subsequent vasoconstriction yields hypertension which triggers reflexive vasodilation above the lesion with resultant headache, flushing, sweating, and nasal congestion. Eliminating noxious stimuli and ensuring prompt blood pressure control are key in management [49, 50].

Up to 80 % of patients with tetraplegia and 50 % of patients with paraplegia develop OH [51]. The condition may be related to excessive pooling of blood in the abdominal viscera and lower extremities due to decreased sympathetic nervous system activity and loss of reflexive vasoconstriction [52]. Arm exercises during tilt table use, body weight support treadmill training, abdominal binders or compression stockings, and use of salt tablets have not been proven effective in the treatment of OH [51].

Bowel and Bladder

Bowel and bladder dysfunction are expected after most TSCIs and can affect the psychological and social well-being of patients in ways other medical issues do not. Detrusor or sphincter hypereflexia and/or areflexia are the etiologic basis for most forms of neurogenic bladder. Urodynamic studies are the gold standard to diagnosing the precise etiology of neurogenic bladder but are not readily available in the acute and subacute stages of injury. Independent of cause, the ultimate goal of bladder management is to adequately drain the bladder to preserve upper tract function and maintain continence. Clean intermittent catheterization (CIC) is the safest bladder emptying method for SCI patients who cannot void independently but indwelling catheters, medications, and additional surgical options are also utilized [53].

Neurogenic bowel (NB) affects up to half of patients with SCI [54]. Bowel dysfunction is either due to upper (hyperreflexic; above conus medullaris) motor neuron syndrome in which there is increased colonic wall and anal tone leading to constipation or lower (areflexic; at or below conus medullaris) motor neuron syndrome in which the anal sphincter is denervated and risk of incontinence is high. Treatment is tailored to symptoms and includes a high fiber diet, digital rectal stimulation and manual evacuation, rectal suppositories, timed toileting program, laxatives, stool softeners, and electrical stimulation.

Spasticity

Spasticity usually affects patients in the chronic phase of injury and can cause considerable pain and disability as well as abnormal postures, contracture, and pressure ulcers. It has been reported to affect up to 78 % of patients, and of those, 41 % report it as a major functional impairment [55]. The pathogenesis is unclear. Physical therapy is often the first intervention to address spasticity. Interventions such as prolonged standing on a tilt table or standing frame may help temporarily reduce spasticity. Passive range of motion, serial casting, and electrical stimulation can also be beneficial. Pharmacologic management includes oral, intramuscular, and intrathecal agents. Surgical intervention, typically to release contracture, can be utilized when other methods fail.

Pain

Up to 80 % of patients experience chronic pain. The International Association for the Study of Pain published a tiered classification of pain related to SCI in which pain types are divided into nociceptive (musculoskeletal or visceral) and neuropathic types [56]. Musculoskeletal and neuropathic pain are the commonest among these and are often treated with a combination of analgesics, NSAIDS, anticonvulsants, opioids, spinal cord stimulation, and physical modalities. A detailed review is beyond the scope of this article.

Skin

Pressure ulcers and skin diseases were reported as the second commonest reason for hospital readmission after SCI [57]. In chronic patients, the ischium, trochanters, sacrum, and heel are the principle areas where pressure ulcers develop. Risk factors include immobility, skin moisture, impaired sensation, poor nutrition, and muscle atrophy. Diligent skin care is essential to preventing skin breakdown. In advanced ulcers, surgical debridement may be necessary.

Osteoporosis

Bone loss is very common after TSCI and occurs most aggressively in the first 1–2 years [58]. Disuse as well as non-mechanical factors including nutrition deficiency, abnormal vasoregulation, hypercortisolism, and endocrine disorders [59] seem to be important. Bisphosponates are central to pharmacologic therapy as the focus in these patients is on reversing bone resorption. Weight-bearing exercises, functional electrical stimulation, and pulsed electromagnetic fields have also been studied in the literature.

Horizons

In recent years, regenerative therapy has become the Holy Grail in spinal cord injury research and has been largely centered on stem cell transplantation and spinal cord stimulation. Embryonic stem cells have been central in research trials due to their pluripotent nature and ability to differentiate into cells of all three germ layers [60]. Stem cell transplantation aims to replace lost cells (oligodendrocytes, motor neurons, etc.) and provide therapeutic effects by secreting neuroprotective factors or promoting neuroregeneration (growth factors) [61]. Thus far, animal studies have shown improved functional recovery through remyelination of demylenated cells and de novo growth of neurons and glia, and human trials are gaining momentum [62••, 63].

Spinal cord stimulators are promising devices that have been shown to restore motor circuitry and subsequently improve limb movement. Stimulators work through epidural, intraspinal, and subdural excitation and have been found to be generally safe and effective in humans [64–66]. Enzymes, neurotrophic factors, and growth factors have also been investigated though with mixed results.

Conclusion

Rehabilitation of patients after TSCI is multipronged and begins with education, followed by implementation of early rehabilitation intervention. Aggressive prevention and treatment of common secondary complications is key to reducing morbidity and mortality. Functional goals can be clearly set soon after a TSCI, and through multidisciplinary care, patients work to maximize outcomes. Several neurobiological therapeutic options continue to be explored.

Compliance with Ethics Guidelines

Conflict of Interest Nicole Mazwi, Kate Adeletti, and Ronald Hirschberg declare that they have no conflicts of interest.

Human and Animal Rights and Informed Consent This article does not contain any studies with human or animal subjects performed by any of the authors.

References

Papers of particular interest, published recently, have been highlighted as:
• Of importance
•• Of major importance

1. S, Ho C. Rehabilitation of spinal cord injury. In: Kirshblum S, Campagnolo D, Delisa JA, editors. Spinal cord medicine. Philadelphia: Lippincott; 2002. pp. 275-283.
2.• Sabharwal S. Essentials of spinal cord medicine. New York: Demos Medical; 2014. pp. 4–10. **A current and comprehensive review of salient issues for the patient with spinal cord injury.**
3. Savic G, Bergström E, Frankel HL, Jamous MA, Jones PW. Inter-rater reliability of motor and sensory examinations performed according to American Spinal Injury Association standards. Spinal Cord. 2007;45(6):444–51.
4.•• Kirshblum S, Burns S. International standards for neurological classification of spinal cord injury (Revised 2011). J Spinal Cord Med. 2011;34(6):535–46. **This document serves as the definitive resource for the national standardized examination and classification system for for spinal cord injuries.**
5. Wicks AB, Menter RR. Long-term outlook in quadriplegic patients with initial ventilator dependency. Chest. 1986;90:406–10.
6. Kiwerski J. Respiratory problems in patients with high lesion quadriplegia. Int J Rehabil Res. 1992;15(1):49–52.
7. Jackson AB, Groomers TE. Incidence of respiratory complications following SCI. Arch Phys Med Rehabil. 1994;75:270–5.
8. Ducko CT. Clinical advances in diaphragm pacing. Innovations (Phila). 2011;6(5):289–97.
9. Morris PE, Goad A. Early intensive care unit mobility therapy in the treatment of acute respiratory failure. Crit Care Med. 2008;36(8): 2238–43.
10. Needham DM, Korupolu R, et al. Early physical medicine and rehabilitation for patients with acute respiratory failure: a quality improvement project. Arch Phys Med Rehabil. 2010;91:536–42.
11. Giuglea C, Marinescu S. Pressure sores—a constant problem for plegic patients and a permanent challenge for plastic surgery. J Med Life. 2010;3(2):149–53.
12. Go BK, DeVivo MJ. The epidemiology of spinal cord injury. In: Stover SL, Delisa JA, Whiteneck GG, editors. Spinal cord injury: clinical outcomes for the model systems. Gaithersburg: Aspen; 1995. p. 21–51.
13. Kirshblum S, Fichtenbaum J. Breaking the news in spinal cord injury. J Spinal Cord Med. 2008;31(1):7–12.
14. Paralyzed Veterans of America. Clinical practice guidelines: pressure ulcer prevention and treatment follow spinal cord injury: a clinical practice guideline for health-care professionals (2nd ed.). Consortium for Spinal Cord Medicine, 2014.
15. Field-Foote E. Spinal cord injury rehabilitation. Philadelphia: F.A. Davis; 2009.
16. Harkema S et al. Locomotor training: as a treatment of spinal cord injury and in the progression of neurologic rehabilitation. Arch Phys Med Rehabil. 2012;93:1588–97.

17. Edgerton VR et al. Topical review: retraining the injured spinal cord. J Physiol. 2001;533:15–22.
18. Mehroholz J, Kugler J, Pohl M. Locomotor training for walking after spinal cord injury [Review]. Cochrane Collab. 2012:1-51.
19. Thomas S, Gorassini M. Increases in corticospinal tract function by treadmill training after incomplete spinal cord injury. J Neurophysiol. 2005;94:2844–55.
20. Aach M et al. Voluntary driven exoskeleton as a new tool for rehabilitation in chronic spinal cord injury: a pilot study. Spine. 2014;14:2847–53.
21. Ditunno PL, Patrick M, Stineman M, Ditunno JF. Who wants to walk? Preferences for recovery after SCI: a longitudinal and cross-sectional study. Spinal Cord. 2008;46:500–6.
22. Burns SP, Golding DG, Rolle Jr WA, Graziani V, Ditunno JF. Recovery of ambulation in motor incomplete tetraplegia. Arch Phys Med Rehabil. 1997;78:1169–72.
23. van Middendorp JJ, Hosman AJ, Pouw MH, EM-SCI Study Group, Van de Meent H. ASIA impairment scale conversion in traumatic SCI: is it related with the ability to walk? A descriptive comparison with functional ambulation outcome measures in 273 patients. Spinal Cord. 2009;47(7):555–60.
24. Scivoletto G, Morganti B, Molinari M. Neurologic recovery of spinal cord injury patients in Italy. Arch Phys Med Rehabil. 2004;85:485–9.
25. Ditunno JF, Scivoletto G, Patrick M, Biering-Sorensen F, Abel R, Marino R. Validation of the walking index for spinal cord injury in a US and European clinical population. Spinal Cord. 2008;46:181–8.
26. Oleson CV, Burns AS, Ditunno JF, Geisler FH, Coleman WP. Prognostic value of pinprick preservation in motor complete, sensory incomplete spinal cord injury. Arch Phys Med Rehabil. 2005;86:988–92.
27. Scivoletto G, Morganti B, Ditunno P, Ditunno JF, Molinari M. Effects on age on spinal cord lesion patients' rehabilitation. Spinal Cord. 2003;41:457–64.
28. Lee BA, Leiby BE, Marino RJ. Neurological and functional recovery after thoracic spinal cord injury. J Spinal Cord Med. 2014:1-9.
29. Zörner B, Blanckenhorn WU, Dietz V, EM-SCI Study Group, Curt A. Clinical algorithm for improved prediction of ambulation and patient stratification after incomplete spinal cord injury. J Neurotrauma. 2010;27:241–52.
30.•• Wilson JR, Grossman RG, Frankowski RF, Kiss A, Davis AM, Kulkarni AV, et al. A clinical prediction model for long-term functional outcome after traumatic spinal cord injury based on acute clinical and imaging factors. J Neurotrauma. 2012;29:2263–71. **Novel clinical prediction tool for spinal cord injury prognosis**.
31. van Middendorp JJ, Hosman AJ, Donders AR, Pouw MH, Ditunno Jr JF, Curt A, et al. A clinical prediction rule for ambulation outcomes after traumatic spinal cord injury: a longitudinal cohort study. Lancet. 2011;377(9770):1004–10.
32. Waters RL, Adkins RH, Yakura JS, Sie I. Motor and sensory recovery following incomplete tetraplegia. Arch Phys Med Rehabil. 1994;75:306–11.
33. Waters RL, Adkins RH, Yakura JS, Sie I. Motor and sensory recovery following incomplete paraplegia. Arch Phys Med Rehabil. 1994;75:67–72.
34. Crozier KS, Cheng LL, Graziani V, Zorn G, Herbison G, Ditunno Jr JF. Spinal cord injury: prognosis for ambulation based on quadriceps recovery. Paraplegia. 1992;30:762–7.
35. Ramón S, Domínguez R, Ramírez L, Paraira M, Olona M, Castelló T, et al. Clinical and magnetic resonance imaging correlation in acute spinal cord injury. Spinal Cord. 1997;35:664–73.
36. Andreoli C, Colaiacomo MC, Rojas Beccaglia M, Di Biasi C, Casciani E, Gualdi G. MRI in the acute phase of spinal cord traumatic lesions: relationship between MRI findings and neurological outcome. Radiol Med. 2005;110:636–45.
37. Shimada K, Tokioka T. Sequential MR studies of cervical cord injury: correlation with neurological damage and clinical outcome. Spinal Cord. 1999;37:410–5.
38. Curt A, Dietz V. Ambulatory capacity in spinal cord injury: significance of somatosensory evoked potentials and ASIA protocols in predicting outcome. Arch Phys Med Rehabil. 1997;78:39–43.
39.• Nebahat S et al. Chronic complications of spinal cord injury. World J Orthop. 2015;6(1):24–33. **Updated summary of common complications of spinal cord injury and accepted treatment**.
40. Tollefsen E, Fondenes O. Respiratory complications associated with spinal cord injury. Tidsskr Nor Laegeforen. 2012;132:1111–4.
41. Garshick E, Kelley A, Cohen SA, Garrison A, Tun CG, Gagnon D, et al. A prospective assessment of mortality in chronic spinal cord injury. Spinal Cord. 2005;43:408–16.
42. Brown R, DiMarco AF, Hoit JD, Garshick E. Respiratory dysfunction and management in spinal cord injury. Respir Care. 2006;51:853–68. discussion 869-870.
43. Linn WS, Adkins RH, Gong H, Waters RL. Pulmonary function in chronic spinal cord injury: a cross-sectional survey of 222 southern California adult outpatients. Arch Phys Med Rehabil. 2000;81:757.
44. Stepp EL, Brown R, Tun CG, Gagnon DR, Jain NB, Garshick E. Determinants of lung volumes in chronic spinal cord injury. Arch Phys Med Rehabil. 2008;89:1499–506.
45. McKinley WO, Gittler MS, Kirshblum SC, Stiens SA, Groah SL. Spinal cord injury medicine. 2. Medical complications after spinal cord injury: identification and management. Arch Phys Med Rehabil. 2002;83:S58–64.S90-S98.
46. Hagen EM, Rekand T, Grønning M, Færestrand S. Cardiovascular complications of spinal cord injury. Tidsskr Nor Laegeforen. 2012;132:1115–20.
47. Hagen EM, Faerestrand S, Hoff JM, Rekand T, Gronning M. Cardiovascular and urological dysfunction in spinal cord injury. Acta Neurol Scand Suppl. 2011;191:71–8.
48. Phillips WT, Kiratli BJ, Sarkarati M, Weraarchakul G, Myers J, Franklin BA, et al. Effect of spinal cord injury on the heart and cardiovascular fitness. Curr Probl Cardiol. 1998;23:641–716.
49. Blackmer J. Rehabilitation medicine: 1. Autonomic dysreflexia. CMAJ. 2003;169:931–5.
50. Somani BK. Autonomic dysreflexia: a medical emergency with spinal cord injury. Int J Clin Pract. 2009;63:350–2.
51. Krassioukov A et al. A systematic review of the management of orthostatic hypotension after spinal cord injury. Arch Phys Med Rehabil. 2009;90:876–85.
52. Claydon VE et al. Orthostatic hypotension following spinal cord injury: understanding clinical pathophysiology. Spinal Cord. 2006;44:341–51.
53. Weld KJ, Dmochowski RR. Effect of bladder management on urological complications in spinal cord injured patients. J Urol. 2000;163:768–72.
54. Liu CW, Huang CC, Yang YH, Chen SC, Weng MC, Huang MH. Relationship between neurogenic bowel dysfunction and health-related quality of life in persons with spinal cord injury. J Rehabil Med. 2009;41:35–40.
55. Retal L. The Stockholm Spinal Cord Injury Study: medical problems in a regional SCI population. Paraplegia. 1995;33(6):308–15.
56. Siddall PJ, Middleton JW. A proposed algorithm for the management of pain following spinal cord injury. Spinal Cord. 2006;44:67–77.
57. Cardenas DD, Hoffman JM, Kirshblum S, McKinley W. Etiology and incidence of rehospitalization after traumatic spinal cord injury: a multicenter analysis. Arch Phys Med Rehabil. 2004;85:1757–63.
58. Middleton JW, Leong G, Mann L. Management of spinal cord injury in general practice—part 2. Aust Fam Physician. 2008;37:331–2. 335-338.
59. Jiang SD, Dai LY, Jiang LS. Osteoporosis after spinal cord injury. Osteoporos Int. 2006;17:180–92.

60. Mury CE, Keller G. Differentiation of embryonic stem cells to clinically relevant populations: lessons from embryonic development. Cell. 2008;132:661–80.
61. Himes BT et al. Recovery of function following grafting of human bone marow-derived stromal cells into the injured spinal cord. Neurorehabil Neural Repair. 2003;20:278–96.
62.•• Iwai H, Shimada H, Nishimura S, Kobayashi Y, Itakura G, Hori K, et al. Allogeneic neural stem/progenitor cells derived from embryonic stem cells promote functional recovery after transplantation into injured spinal cord of nonhuman primates. Stem Cells Transl Med. 2015; 4:708–19. **One of the first trials to show safety and efficacy of allografted embryonic stem cell-derived progenitor cells in non-human primates with spinal cord injury.**
63. Lu P, Wang Y, Graham L, McHale K, Gao M, Wu D, et al. Long-distance growth and connectivity of neural stem cells after severe spinal cord injury. Cell. 2012;150:1264–73.
64. Jackson A, Zimmermann JB. Neural interfaces for the brain and spinal cord—restoring motor function. Nat Rev Neurol. 2012;8: 690–9.
65. Harkema S, Gerasimenko Y, Hodes J, Burdick J, Angeli C, Chen Y, et al. Effect of epidural stimulation of the lumbosacral spinal cord on voluntary movement, standing, and assisted stepping after motor complete paraplegia: a case study. Lancet. 2011;377:1938–47.
66. Sharpe AN, Jackson A. Upper-limb muscle responses to epidural, subdural and intraspinal stimulation of the cervical spinal cord. J Neural Eng. 2014;11:016005.