Chapter 16

SPINAL CORD INJURY REHABILITATION

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INTRODUCTION

Spinal cord injury (SCI) among the US civilian population occurs at the rate of 11,000 new injuries per year. The most recent data from US National SCI Statistical Center indicate that young men are the most common demographic to sustain SCI (78% of the total injured are males). Although the most frequent age at injury is 19, the mean age at injury is 38. Impacts at high velocity are the most common cause of SCI. Motor vehicle collisions are responsible for 50%, falls for 24%, acts of violence for 11%, and sports for 9% of SCIs.[1,2] Low-velocity gunshot injuries are responsible for most acts of violence that result in SCI; other penetrating injuries account for 0.9%, and blasts account for 0.1%.[3] The most common SCI site is the cervical cord (54% of cases), followed by the thoracic cord (36%), and the lumbar cord (10%). The most common neurological category is incomplete tetraplegia (34.1% of all injuries; the term "tetraplegia" replaced "quadriplegia" to indicate a cervical neurological level, and "paraplegia" refers to a thoracic, lumbar, or sacral neurological level).[1] About 253,000 Americans (roughly 0.1% of the US population) are traumatic SCI survivors.

The demographic profile of war-injured SCI survivors is less well documented. In Vietnam, 0.9% of those admitted to US Army hospitals had sustained SCI, and 3.8% of those patients died during initial hospitalization.[4] In various Israeli wars, the percentage of those injured by gunshots to the spinal cord varied between 0.2% during the War of Independence in 1948–1949 and 1.1% in the Sinai Campaign of 1956.[5] As of December 31, 2008, 432 active duty US service members serving during Operations Enduring Freedom and Iraqi Freedom sustained SCI and received treatment in Department of Veterans Affairs SCI units. Of these individuals, 141 were injured in theater.

From the time the first recorded description of SCI was written on Egyptian papyrus until World War II, SCI was viewed as "an ailment not to be treated." Death following SCI commonly ensued from uncontrollable urinary tract infections (UTIs) and pressure ulcers. During the Balkan Wars of 1912–1913, 95% of soldiers with SCI died within a few weeks. During World War I, 80% of overseas American troops with SCI died before they could return to the United States.[6]

During World War II, the attitude of fatalism toward the spinal cord injured began to change. Advances in anesthesia, surgical techniques, blood transfusions, and antibiotics all contributed to increased survival. Teams of physicians, nurses, and therapists in Great Britain established SCI units and developed procedures for meticulous care and rehabilitation of the injured.[7] These protocols not only allowed the injured to survive, but in many cases made it possible for them to return to their communities.

At the conclusion of World War II, the US government, following the British model, established the first comprehensive SCI unit at Hines Veterans Administration Hospital in suburban Chicago. Over 75% of the paraplegics from World War II were alive 20 years later, and of the 2,500 American paraplegics from that war, over half returned to the job force. The first federally designated SCI care system center (part of the national Model SCI System) for civilians in the United States opened in 1970, and additional regional centers were developed through the 1970s. Fourteen centers are currently funded through the National Institute on Disability and Rehabilitation Research to provide comprehensive care for individuals with new, traumatic SCI; contribute data to the National SCI Statistical Center; and conduct SCI research.

With improved care, particularly of urologic and skin complications, late mortality (ie, after initial rehabilitation) from SCI also declined. Late mortality was 1.7 times higher for those injured in the 1940s than for those injured in the 1960s.[8] For those injured at age 20 and with minimal paralysis, life expectancy is only reduced by about 6 years. With more than minimal paralysis, life expectancy is progressively reduced across higher injury levels, with life expectancy of about 43 years for a person who becomes ventilator dependent at age 20 and survives for the first year following injury.[9] As mortality has declined, the focus of SCI care has gradually shifted. Initially, the target was defining and adopting procedures and practices to control the often fatal sequelae. Now the focus also includes retraining individuals for independence, return to community, a lifetime of healthy behaviors, and full quality of life.

PATHOPHYSIOLOGY OF SPINAL CORD INJURY

The two primary types of traumatic SCI mechanisms are penetrating and nonpenetrating injuries. Penetrating injuries typically result from a bullet, fragment, or knife blade directly lacerating the spinal cord. Penetrating injury accounts for only 17% of traumatic SCI in the civilian population, and nearly all of these are due to gunshots. Nonpenetrating injuries commonly result from bone or herniated disk material compressing the spinal cord or cauda equina, or from traction from spinal dislocation. In the civilian population, automobile

and motorcycle crashes, falls, and sports injuries are the primary causes of nonpenetrating SCI. Blast injuries cause SCI through secondary effects (eg, fragments that penetrate the spinal canal) or tertiary effects (eg, nonpenetrating injuries from acute angulation or loading of the spine due to blast wind, structural collapse, or deceleration after being thrown by a blast).[10] Penetrating and nonpenetrating SCI may involve vertebral fracture with displacement of bony fragments into the spinal canal, or ischemia due to disruption in the blood supply to the cord. Nonpenetrating injuries often result in ligament disruption, with instability and resulting vertebral malalignment. Individuals with narrow spinal canals, either congenital or acquired due to degenerative changes, are at greater risk for cord injury, even from relatively minor trauma.

Penetrating and nonpenetrating injuries may compromise gray matter, white matter, and nerve roots. Gray matter contains motor neurons that provide output to muscles and interneurons that receive descending motor and segmental reflex input. Gray matter is thought to be more vulnerable to mechanical trauma than white matter because it is relatively more vascular. Gray matter damage typically extends one or two segments rostral and caudal to the cord injury, but may be more extensive if the cord blood supply has been disrupted. All segments involved compose the "zone of injury." Damage to gray matter causes segmental changes with denervation, muscle atrophy, and impaired reflexes. White matter is comprised of ascending and descending fibers at the periphery of the cord. Pathologic studies of cord trauma show greater gray than white matter involvement.[11–13] Damage to white matter is more disabling because it results in loss of motor control and sensory input at and below the injury site, and hypertonia and hyperreflexia accompany weakness and sensory loss with such white matter involvement. Nerve root injury often results in an asymmetric level of injury.

Spinal cord damage can also arise indirectly from vascular disruption. Thus, cross clamping of the thoracic aorta or disruption of the artery of Adamkiewicz or a vertebral artery can result in cord ischemia or infarction.[14–16] Another type of ischemic cord injury is decompression sickness, in which hyperbaric exposure (as in underwater diving) followed by sudden decompression results in gas bubble formation and bubble emboli that occlude blood vessels.[17,18]

In the majority of cases, the spinal cord is not completely transected anatomically, and secondary processes contribute to the degree of neurological deficit. These processes include ischemia, edema, hematomyelia, demyelination, persisting mechanical pressure, lactic acidosis, intracellular influx of calcium, increase of lipid peroxidation, and free radical formation.[19–22] Various early treatments, such as hyperbaric oxygen, cord cooling, naloxone, thyrotropin-releasing hormone, and osmotic diuretics, have been proposed to minimize this secondary neurologic injury.[22] Methylprednisolone has been investigated in three multicenter trials as a neuroprotective agent following acute SCI,[23–25] and it may modestly improve neurological outcomes when given within 8 hours of nonpenetrating SCI. However, trial findings have been questioned,[26] and there are concerns about increased infection rates with use of methylprednisolone. GM-1 gaglioside showed promising results as a neuroprotective agent in an early trial, but a multicenter trial with 760 subjects failed to show any benefit in the primary neurologic outcome.[27] Another intervention to promote recovery is late (ie, 1–12 months after SCI) anterior decompression. This procedure reportedly allowed functional recovery in subjects with incomplete SCI, and residual cord or root compression for those whose recovery had plateaued for 4 weeks or more.[28]

Animal models of incomplete SCI demonstrate that much of the spontaneous neurologic recovery is mediated by spared white matter axons that substitute for those pathways that have degenerated, rather than by resolution of conduction block, remyelination or resolution of ischemia, or edema. The mechanisms that allow for this substitution of function by spared pathways likely include rapid-acting denervation supersensitivity and slower-acting synaptogenesis. When spared, descending white matter pathways and spinal reflex pathways undergo trauma-induced reactive synaptogenesis, which may result in both motor recovery and spinal hyperreflexia. The slow pace of this motor recovery and the gradual onset of spasticity may be explained by the slowness of synaptogenesis. Because neural activity seems to be a necessary condition for such recovery, one way to enhance this process may be to increase the activity in the spared neural pathways.[29] In animal models of stroke, administration of central nervous system stimulants (eg, amphetamine), combined with exercise, has enhanced recovery. These observations suggest that remobilization of the patient and active exercise are essential factors in optimizing recovery of function after SCI.

Medications and growth factors are unexplored methods of regulating activity in the cord's spared neural pathways. The optimal treatment for minimizing developing spasticity during this period of recovery is also unresolved. With stroke patients, some advocate reflex facilitation during strengthening exercises to enhance motor recovery; however, this may result in greater spasticity. Alternatively, aggressive early treatment with medication and physical modalities may

suppress the development of spasticity, but it may not optimize recovery of motor function. Resolving these issues will allow more effective rehabilitation of acute, incomplete SCI in the future.

In contrast to white matter or long-tract recovery, zone-of-injury recovery involves recovery of lower motor neurons (gray matter recovery) and roots at the site of injury.[30,31] Mechanisms to explain zone-of-injury recovery include resolution of conduction block, reactive synaptogenesis by descending pathways in the spinal cord, motor axon sprouting by lower motor neurons,[32] and muscle fiber hypertrophy. These recovery mechanisms mitigate upper and lower motor neuron weakness, both of which can be identified electrophysiologically.[33] The optimal rehabilitation interventions for these two types of weakness are not yet known.

NEUROLOGICAL ASSESSMENT AND CLASSIFICATION

The methods described in the *International Standards for Neurological Classification of Spinal Cord Injury* are the most widely used ways of classifying SCI.[34,35] Commonly referred to as "the American Spinal Injury Association (ASIA) classification," this assessment requires manual muscle testing of 10 key muscles bilaterally (Table 16-1), sensory testing for light touch and sharp/dull discrimination in all dermatomes, and a rectal exam for sensation and presence of voluntary anal contraction. These tests are used to classify injury levels and ASIA Impairment Scale (AIS) grade (Figure 16-1). The sensory level is the most caudal level with normal light touch and sharp/dull discrimination (provided that all rostral levels are normal), and is defined separately for the right and left sides. Motor level is defined as the most caudal level that is grade 3 or greater with all rostral levels normal (grade 5), and it is also defined separately for the right and left sides. If the sensory level falls in a segment where there are no corresponding limb muscles to test (levels C2–C4, T2–L1, or S2–S4/5), the motor level is assumed to be the same as the sensory level. A single neurological level can be derived from the four levels (right and left motor, right and left sensory) by taking the most rostral of the four; however, motor level is more closely associated with functional capacity.

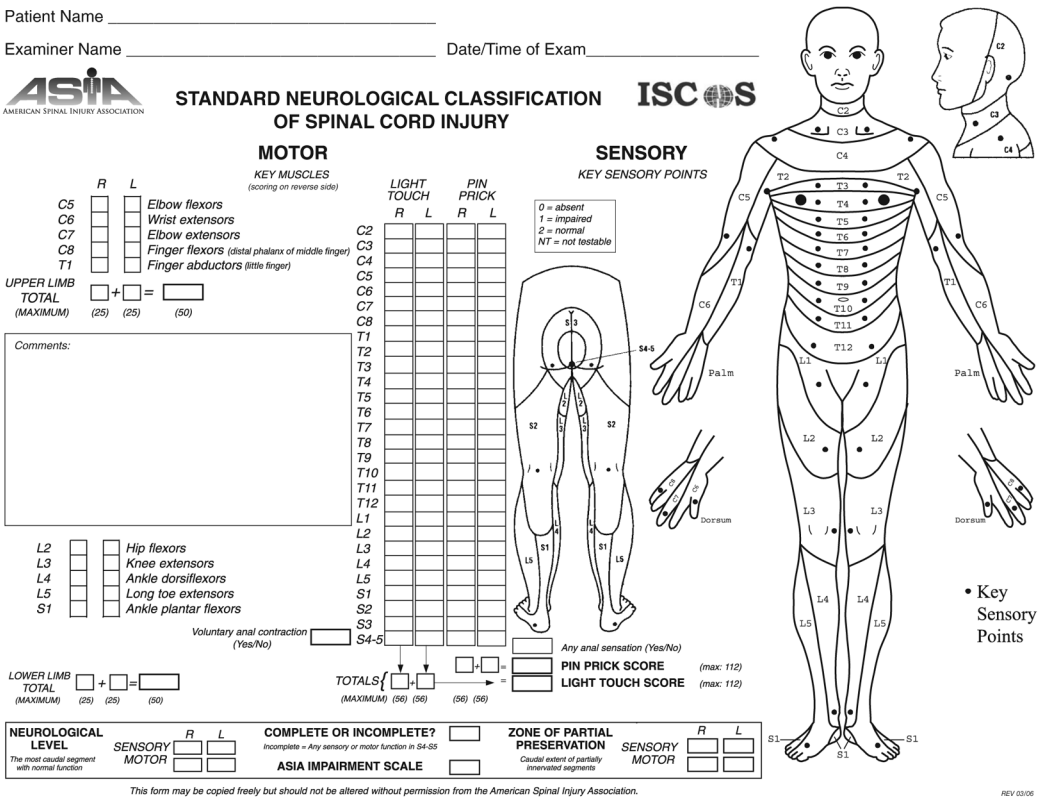
The AIS grade is a measure of the completeness of SCI. A complete injury is one in which no sensory or motor signals traverse the entire zone of cord injury. Because sparing of sensation or motor is most likely to occur in the sacral segments, the classification has been standardized to examine neurologic function in this region for the purpose of classifying injury completeness. An AIS score of *A* indicates a complete SCI and is defined as the absence of sensory and motor function in segments S4–5. AIS *B* is an incomplete SCI, with preservation of sensory function but not motor function below the injury level (patients may have some motor function in the segments adjacent to the neurological level, but it does not extend more than three segments below the neurological level). AIS *C* indicates an incomplete SCI, with motor function preserved below the neurological level and the majority of muscles below the neurological level having less than grade 3 strength. AIS *D* indicates an incomplete SCI, with motor function preserved below the neurological level and at least half the muscles below the neurological level with muscle grade 3 or greater. AIS *E* indicates the patient had an SCI but made a complete neurologic recovery for all sensory and motor functions used in this classification. The exam required for this classification system does not involve deep tendon reflexes or muscle spasticity, and the muscles selected for the examination are not necessarily those

TABLE 16-1

NEUROLOGIC EVALUATION: MYOTOMAL LEVELS

Level	Area of Sensation
C-1,-2,-3	Trapezius, SCM, upper cervical paraspinals, prevertebral neck muscles
C-4	Diaphragm
C-5	*Biceps brachii*, brachialis
C-6	*Extensor carpi radialis longus*
C-7	*Triceps brachii*
C-8	*Flexor digitorum profundus* (3rd digit)
T-1	*Abductor digiti minimi*, 1st dorsal interosseus
T-6,-7,-8,-9,-10	Beevor's sign*
L-2	*Iliopsoas*, hip adductors
L-3	*Quadriceps femoris*
L-4	*Tibialis anterior*
L-5	*Extensor hallucis longus*, hip abduction
S-1	*Gastrocnemius, soleus*
S-2,-3,-4	Anal sphincter

*Beevor's sign represents upward movement of the umbilicus when the patient attempts a sit-up from supine lying.
SCM: sternocleidomastoid
The italicized muscles are the standard muscles used by the American Spinal Cord Injury Association for classifying injury level.[1] These muscles are innervated by more than one root level, but reduction to a representative level is useful for injury classification.
(1) American Spinal Injury Association. *International Standards for Neurological Classification of Spinal Cord Injury*. Chicago, Ill: ASIA; 2000.



MUSCLE GRADING

0 total paralysis

1 palpable or visible contraction

2 active movement, full range of motion, gravity eliminated

3 active movement, full range of motion, against gravity

4 active movement, full range of motion, against gravity and provides some resistance

5 active movement, full range of motion, against gravity and provides normal resistance

5* muscle able to exert, in examiner's judgement, sufficient resistance to be considered normal if identifiable inhibiting factors were not present

NT not testable. Patient unable to reliably exert effort or muscle unavailable for testing due to factors such as immobilization, pain on effort or contracture.

ASIA IMPAIRMENT SCALE

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

☐ **B = Incomplete:** Sensory but not motor function is preserved below the neurological level and includes the sacral segments S4-S5.

☐ **C = Incomplete:** Motor function is preserved below the neurological level, and more than half of key muscles below the neurological level have a muscle grade less than 3.

☐ **D = Incomplete:** Motor function is preserved below the neurological level, and at least half of key muscles below the neurological level have a muscle grade of 3 or more.

☐ **E = Normal:** Motor and sensory function are normal.

CLINICAL SYNDROMES (OPTIONAL)

☐ Central Cord
☐ Brown-Sequard
☐ Anterior Cord
☐ Conus Medullaris
☐ Cauda Equina

STEPS IN CLASSIFICATION

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

1. Determine sensory levels for right and left sides.
2. Determine motor levels for right and left sides.
   Note: in regions where there is no myotome to test, the motor level is presumed to be the same as the sensory level.
3. Determine the single neurological level.
   This is the lowest segment where motor and sensory function is normal on both sides, and 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 (sacral sparing).
   If voluntary anal contraction = **No** *AND all S4-5 sensory scores* = **0** *AND any anal sensation* = **No**, *then injury is COMPLETE. Otherwise injury is incomplete.*
5. Determine ASIA Impairment Scale (AIS) Grade:

Is injury Complete?
NO ↓ — If **YES**, AIS=A Record ZPP (For ZPP record lowest dermatome or myotome on each side with some (non-zero score) preservation)

Is injury motor incomplete?
YES ↓ — If **NO**, AIS=B (Yes=voluntary anal contraction OR motor function more than three levels below the motor level on a given side.)

Are at least half of the key muscles below the (single) neurological level 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.

Figure 16-1. Standard neurological classification of spinal cord injury worksheet produced by the American Spinal Injury Association.
Reproduced with permission from: American Spinal Injury Association. *International Standards for Neurological Classification of SCI*. Chicago, Ill: ASIA; 2002.

of the most functional importance.

It has been reported that early neurological classification reliably predicts the classification of civilian SCI at follow up. Earlier studies focused on the examination 3 to 7 days after injury, but it appears the initial emergency department examination has high predictive value when distracting facts, like intoxication or brain injury, have been removed.[36] For example, of patients with initial AIS *A* injuries and no distracting factors, 6.7% converted to AIS *B* and none converted to AIS *C* or *D* at 1 year following initial examination. In the presence of factors affecting the exam reliability, 17.4% of those with initial AIS *A* scores converted to AIS *B*, and 13% converted to AIS *C*. Sparing of sharp/dull discrimination in sacral dermatomes is favorable for motor recovery to a degree that allows ambulation, possibly due to a similar location of corticospinal and spinothalamic tracts within the spinal cord.[37] The prognosis for improvement in AIS grade is much better for those initially classified AIS *B*, with 54% converting to AIS *C* or *D*. Patients initially classified AIS *C* or *D* often show large improvements in neurologic function; a trace of toe movement in the first few days after injury is highly favorable for functional recovery. Motor function typically descends at least one level between the acute exam and 1-year follow-up. Nearly all muscles near the injury level that initially show grade 1 on manual muscle test will regain at least grade 3 strength by follow-up.[31]

Various SCI clinical syndromes have been described (Table 16-2).[38] Several relate to the presumed extent of the cord injury in the transverse plane, such as Brown-Séquard, central cord, and anterior cord syndromes. Brown-Séquard syndrome is attributed to a cord hemisection and is often the result of a penetrating injury, such as a knife or gunshot wound. The prognosis for return of functional ambulation and voluntary bladder control following Brown-Séquard syndrome is good.[39,40] Central cord syndrome is a cervical-level, motor-incomplete injury with greater weakness in upper versus lower limbs. Central cord syndrome often occurs from a cervical hyperextension injury in the presence of an underlying narrow cervical canal, either from congenital or acquired stenosis. Bony fractures or instability are often absent. Anterior cord syndrome typically results from a cervical burst fracture or disk herniation, impinging on the anterior spinal artery, anterior cord, or both. It is not known whether direct mechanical pressure or disruption of arterial blood flow is the major cause. The likelihood of a return of lower limb function in anterior cord syndrome is reduced.

Cauda equina syndrome results from injury below the termination of the spinal cord (levels L-1–L-2), damaging the anterior and posterior roots, but sparing the cord itself. Midline lumbar disk herniations and major trauma (eg, gunshot wounds or seatbelt injuries) are common causes of cauda equina injury. Prognosis for functional recovery is good because the roots are less vulnerable to mechanical trauma than the spinal

TABLE 16-2

SPINAL CORD INJURY CLINICAL SYNDROMES

	Clinical Features	Prognosis
Central Cord	Common; greater weakness of upper limbs than lower limbs	Often recover bladder and bowel function/control and ambulation
Brown-Séquard	Common; unilateral impaired pain and temperature sensation contralateral to the more paretic side; unilateral impaired vibration and position sense ipsilateral to the paretic side	Usually recover bladder and bowel function/control and ambulation
Anterior cord	Uncommon as pure syndrome; loss of motor, pain, and temperature, with preserved vibratory and proprioception	Functional recovery uncommon
Posterior cord	Rare; absent position and vibratory sensation; intact pain and temperature sensation	Usually recover bladder and bowel function/control and ambulation
Cauda equina	About 10% of all cord injuries; often motor incomplete and with asymmetric deficits	Commonly recover bladder and bowel function/control and ambulation
Conus medullaris	Mixed upper motor neuron and lower motor neuron findings through lumbar and sacral myotomes	May recover ambulation; less likely to recover bladder and bowel control

cord. In addition, motor axons have some capacity for regeneration to proximal muscles and to the bladder.[41] Conus medullaris syndrome involves damage to the spinal cord at the lumbar or sacral segments, resulting in bowel, bladder, and sexual dysfunction, with some upper motor neuron findings present due to sparing of the terminal portion of the conus, and lower motor neuron findings due to root injury or direct compression of lower motor neuron cell bodies. In cases where the entire conus has been injured at its termination at level L-1 or level L-2, the clinical findings are identical to cauda equina syndrome.

"Spinal shock" represents depressed spinal reflexes and weakness caudal to an SCI. This is likely caused by loss of normal tonic descending facilitation because a block of conduction in suprasegmental pathways results in hyperpolarization of cord neurons.[42] Spinal reflexes gradually return over days to months, typically becoming hyperactive leading to spasticity, flexor and extensor spasms, and hypertonia.[43,44] Some reflex activity may return as early as 24 hours after complete SCI, such as the bulbocavernosus reflex or the tibial H-reflex, even though tendon reflexes usually return weeks or months later in patients with complete injuries. In those with incomplete SCI, tendon reflexes and spasticity may appear within days of the injury.[45] Spinal shock is not observed if the myelopathy develops gradually, as is often seen with a cord tumor, cervical stenosis, or syringomyelia. Presumably, hyperactive reflexes, such as a Babinski sign and ankle clonus, develop before overt weakness because mechanisms of neuroplasticity (such as sprouting by reflex afferents and spared descending pathways) mediate hyperreflexia and spared voluntary movement.

INITIAL REHABILITATION

Functional Outcome

Rehabilitation goals vary according to the level of injury and the extent of damage to the spinal cord. When the injury is motor-complete (AIS *A* or *B*), the functional outcome depends to a large degree on the level of injury. The lower the injury is on the cord, the more voluntary movement is preserved and the greater the expectations for independence. Expected functional outcomes for the average person with SCI may be found in recent clinical practice guidelines.[46]

Mobility

Mobility is essential to resuming life outside the hospital. In teaching mobility, self-care, and other functional tasks, the following general principles apply: (*a*) start with the simple and move toward the more complex; (*b*) break tasks down into components, beginning with discrete units that can be learned separately and then combined into completed units; and (*c*) teach patients to substitute for weakened or absent muscles with head motions, momentum, and preserved muscles. The expected optimal outcome for a given patient, based on the level and the completeness of the injury, dictates how and which muscles should be trained (Table 16-3).[46]

Mobility encompasses a spectrum of movement, including bed mobility (ie, turning from side to side, moving from supine to sitting), sitting balance, wheelchair transfers (ie, from wheelchair to bed, wheelchair to car, and wheelchair to floor), standing balance, and ambulation (wheelchair or walking). Each task is mastered in physical therapy.

Wheelchair Use

One of the first objectives of therapy for the spinal cord injured is to get the patient sitting upright, a task usually attended to by physical therapists and nurses. During this procedure, the patient is monitored closely for orthostatic hypotension (OH), a common condition brought on by prolonged bed rest and the deficient vasoconstriction that accompanies disruption of the sympathetic nervous system. Tilt tables and reclining wheelchairs can aid in achieving upright posture. Progressively increasing the verticality of a tilt table challenges the cardiovascular system, which eventually improves systolic blood pressure and cerebral blood flow in the upright sitting and standing positions. For expected wheelchair users, the same can be achieved at the bedside in a reclining wheelchair with elevating leg rests; therapy staff can immediately recline the chair if a patient develops symptomatic hypotension.

As the patient regains mobility, spinal alignment and neurologic status may be affected. If this is suspected, frequent radiographs and neurologic examinations are required to be certain that gravity, postural changes, and muscular forces do not compromise alignment.

The wheelchair is a patient's key to mobility, and helping the patient select the correct chair is a complex task. The chair should be custom fit according to the user's pelvis width, trunk height, and leg, thigh, and forearm length. A clinician must review the wheelchair setup and make appropriate adjustments to ensure proper posture and seating. The chair should be efficient, appropriately stable, maneuverable, and matched to the strength and coordination of the user; a manual wheelchair that is configured to allow one

TABLE 16-3

EXPECTED FUNCTIONAL OUTCOME FOR COMPLETE SPINAL CORD INJURY

Cord Level	Preserved Muscle	Eating	Dressing	Transfers	Mobility		Writing
					Indoor	Outdoor	
C-1, -2	Trapezius, sternocleidomastoid	D	D	D lift or pivot	I Power WC Sip and puff	I	I Computer Sip and puff
C-3, -4	Diaphragm, neck flexor/extensor	D	D	D lift or pivot	I Chin control	I	I Mouth stick
C-5	Deltoid, biceps	I cuff	D	D lift or pivot	I Power WC	I	I cuff
C-6	Serratus anterior, extensor carpi radialis	I	I?	I? loops sliding board	I (+/– power WC)	I?	I
C-7	Triceps	I	I reachers adapted clothes	I	I Manual WC	I	I
C-8 to T-1	Hand intrinsics	I	I	I	I Manual WC	I	I
T-2 to T-6	Intercostals, paraspinals	I	I	I	I Manual WC	I	I
T-7 to T-12	Abdominals	I	I	I	I Manual WC	I	I
L2	Iliopsoas	I	I	I	I (+ manual WC)	I	I
L-3	Quadriceps	I	I	I	I (+/– manual WC)	I	I
L-4, -5	Tibialis anterior, extensor hallucis longus	I	I	I	I Crutches	I AFOs	I
L-5 to S-1	Hamstrings, gluteus medius	I	I	I	I AFOs	I AFOs	I
S-1, -2	Gluteus maximus, gastrocnemius-soleus	I	I	I	I	I	I
S-3, -4	Anal/urethral sphincters	I	I	I	I	I	I

AFO: ankle-foot orthosis
cuff: universal cuff
D: dependent
I: independent
I?: possibly independent
lift: hydraulic lift transfer
pivot: quad pivot transfer
WC: wheelchair

patient to negotiate a curb with a "wheelie" may put another at risk for a dangerous fall. Extra features to consider include antitip devices, lap trays, arm rests, and foot and leg supports. Aesthetics and cost must be considered as well. Proper training is essential for long-term protection of the upper extremities.[47]

An individual with tetraplegia as high as the C-5 level may be able to propel a manual wheelchair. Propulsion for individuals with C-5 level cervical injuries is possible by using the biceps brachii, brachialis, and anterior deltoid muscles in a closed kinetic chain to convert the regular elbow–shoulder flexion to elbow extension when the hand is fixed on a wheelchair handrim by friction. The benefits to operating a manual wheelchair include greater upper limb and cardiovascular exercise, having a backup means of mobility in the event that a power wheelchair malfunctions, and the ability to travel in a passenger car without a wheelchair lift or ramp. Despite this, most C-5 level manual wheelchair users will require a power chair for outdoor mobility involving distances, rough terrain, or grades. For many of these individuals, power wheelchairs are the only feasible means of mobility. Fit and function should be considered when selecting power chairs as

Figure 16-2. A chin joystick controller can be mounted on a boom attached to the chair frame (as shown) or on a plastic yoke placed over the sternum. In addition to driving the power wheelchair, it can be used to operate a tilt-in-space mechanism or environmental control unit.

well, as should the method of control (eg, manual joystick, chin, sip-and-puff straw, mouth stick, or tongue; Figure 16-2). For these patients, reclining mechanisms are essential to minimize the risk of skin breakdown. Two such mechanisms are the zero-shear mechanism and the tilt-in-space recline. The latter is less likely to elicit extensor spasms (Figure 16-3).

Walking

Some patients will progress from sitting to walking. Ambulation goals differ depending on the level of the SCI and whether the cord injury is complete or incomplete. For motor-complete SCI in the absence of lower limb movement, the goals are exercise and short-distance mobility on level surfaces. Those with complete injuries at the T-6 level or below with no other major medical complications are the usual candidates for this training. Contraindications include limited lumbar spine extension or limited hip, knee, and ankle joint motion. Initially, patients are taught to stand between parallel bars and maintain balance without arm support, using temporary knee-ankle-foot orthoses. They then learn to walk with a swing-through gait, using the parallel bars for support. With this gait, upper body center of gravity must be behind the axis of rotation of the hip joint to maintain hip extension (Figure 16-4). The energy expended for this type of walking can be as much as 800% of that of able-bodied walking and manual wheelchair propulsion.[48] Swing-through walking also requires considerable motor coordination and can place excessive loads across the upper limb joints. Previously, this form of ambulation was considered a goal for all young patients with new paraplegia, but that has changed in recent years because most patients did not continue to ambulate after discharge from the hospital. Only those who demonstrate progress and sustained motivation with temporary bracing should be fitted with custom knee-ankle-foot orthoses and proceed to learning a swing-through gait, which requires use of forearm crutches.

For individuals with some preserved lower limb movement, the goals vary from assisted standing-pivot transfers to long-distance ambulation, depending on the extent of motor and proprioceptive function and the degree of spasticity. For those with considerable sparing and rapid recovery, therapy is primarily the reconditioning of muscles that have atrophied from disuse. Those with little sparing and slow recovery require months of spontaneous healing, strengthening, and functional training. For the latter, recovery is less complete and spasticity often interferes with movement, although extensor spasms can aid standing.

Activity-Based Therapy

In past years, there has been a relatively static view of neuroplasticity and neurologic recovery following SCI. Most interventions were pharmacologic and surgical, with the intention of minimizing the primary injury and secondary effects, such as edema. As general knowledge of plasticity and remodeling in the central nervous system has grown, there is renewed interest in developing therapies to stimulate these processes and enhance functional recovery. Physical rehabilitation strategies have recently been employed for these purposes.

Activity-based therapy is now advocated by many rehabilitation centers throughout the world. The general approach is to use neuromuscular activity below the level of an incomplete SCI to stimulate neuroplasticity and promote recovery of function. Locomotor training is the most well-developed activity-based therapy.[49,50,51] Bodyweight support

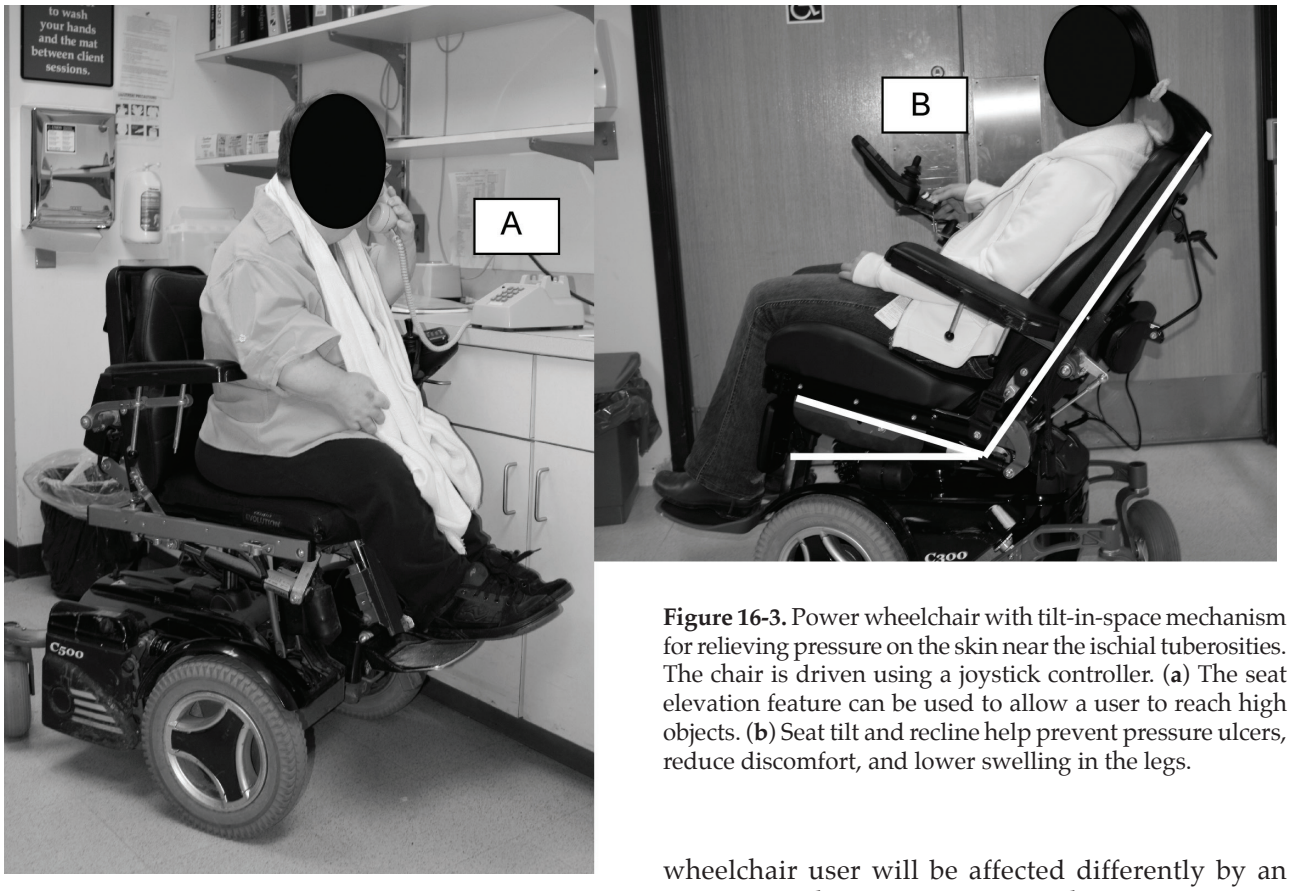


Figure 16-3. Power wheelchair with tilt-in-space mechanism for relieving pressure on the skin near the ischial tuberosities. The chair is driven using a joystick controller. (**a**) The seat elevation feature can be used to allow a user to reach high objects. (**b**) Seat tilt and recline help prevent pressure ulcers, reduce discomfort, and lower swelling in the legs.

and a treadmill, overground ambulation with upper limb aids, electrical stimulation during walking to activate the neuromuscular system, and functional electrical stimulation bikes are used for locomotor training.

Spinal Cord Injury Complicated by Concomitant Traumatic Amputation

Review of data from Operations Iraqi Freedom and Enduring Freedom reveals that the incidence of SCI and amputation is low, albeit with a high functional impact. In addition to managing the multitude of medical issues associated with both SCI and amputation, specific attention must be directed toward identifying and managing heterotopic ossification (HO), which is a known complication after SCI[52] and combat amputations.[53]

The functional impact of SCI complicated by amputation depends on the level of SCI, AIS grade, and the anatomic location of the amputation. When amputation level can be elected, consideration should be given to preserving length in individuals without SCI. An individual with SCI who will be a full-time wheelchair user will be affected differently by an amputation than an individual with SCI presentation consistent with partial or full-time ambulation. For the wheelchair user, a lower limb amputation must be considered relative to the prescribed seating system because a unilateral amputation is likely to cause postural compromise. Specific modifications may be needed to provide postural support and skin protection. The functional impact of lower extremity amputation can be minimized for wheelchair users who acquire compensatory techniques to optimize balance and stability while performing self-care tasks and mobility skills. For individuals with SCI who can stand and walk, the impact of lower limb amputation is a greater challenge because the individual may desire to use a prosthetic leg despite impaired neuromuscular function in the residual limb and trunk. Given the known significant physical requirements for prosthetic limb use and the additional demands resulting from SCI-related impairments, part-time or intermittent wheelchair use should be considered. Upper limb amputation in individuals with SCI creates significant self-care and mobility challenges and indicates a high risk for upper limb pain and injury due to repetitive strain of the intact arm. Clinical interventions and patient education must be targeted toward optimizing functional skills while preserving remaining upper limb function, as

Figure 16-4. Swing-through gait.

supported by published clinical practice guidelines.[47] This may be partially addressed by providing a power wheelchair with power seat functions. Regardless of level of SCI, AIS, or amputation location, maintaining normal range of motion across all joints, maximizing strength of innervated muscles, developing efficient mobility skills, and learning joint preservation techniques are critical to optimize functional outcomes and long-term quality of life.

Driver's Training and Community Mobility

Automobiles usually provide the greatest convenience and flexibility for accessing the community and participating in vocational and avocational activities. Some patients with motor-complete (AIS *A* or *B*) injuries as high as level C-5 can learn to drive with hand controls, although even at the C-6 level, a specialized van with sensitized steering and braking is often required. Those with SCI at level C-7 or lower can often use a passenger car with hand controls, provided they have the ability to transfer themselves and stow their wheelchairs independently. Vision, including visual scanning (the absence of which can sometimes be compensated for with panoramic and side mirrors), reaction time, absence of seizures, adequate spasm control, cognitive awareness, hand function, transfer skills, and a financial source for the necessary equipment and insurance are additional factors determining whether or not a patient is a candidate for driver's training. Adaptations may include hand controls for brakes and gas, steering wheel attachments, power seats, and vans with wheelchair lifts or ramps.

Self-Care

Along with increasing mobility, minimizing the need for assistance in self-care is a major step toward independence for those with SCI. Self-care includes feeding, bathing, dressing, grooming, and toileting. Those with motor-complete injuries at the C-7 level or below can usually achieve independence in all of these activities. Occupational therapists and rehabilitation nurses work with patients to master new techniques for accomplishing these tasks. Alternate strategies, such as using a tenodesis grip (which uses wrist extension to passively flex fingers) for holding eating utensils or writing implements, must be learned and mastered. A wrist-driven, flexor-hinge splint improves

the strength of a tenodesis grip. Other special equipment such as reachers, universal cuffs, built-up handles on utensils, and tub benches also aid independence. Tendon transfers[54] or implantable functional electrical stimulators[55] may achieve a more functional pinch, but costs and training time are considerable. Full independence and self-management of the neurogenic bladder and bowel is a goal for patients with a motor level of C-7 or caudal.

Partial independence may be achieved with level C-6 or level C-5 motor-complete injury. For those tasks that require assistance, the patient is shown how to teach others to complete the task and a caregiver is instructed in how to help with the activity. For example, the assistance needed for self-feeding may include fitting a universal cuff and utensil on a patient, positioning food, opening containers, and cutting meat. With similar assistance, some grooming and hygiene tasks, such as combing hair, brushing teeth, shaving, and washing face and upper body, may be mastered. With motor-complete injury at a C-4 level or rostral, a person will typically be dependent for all self-care activities.

EXHIBIT 16-1

EXAMPLES OF ACTIVITIES AVAILABLE TO INDIVIDUALS WITH SPINAL CORD INJURIES

- archery
- boating
- football
- camping
- hunting
- flying
- golf
- scuba diving
- swimming
- sailing
- basketball
- table tennis
- weight lifting
- tennis
- horseback riding
- wheelchair racing
- wheelchair dancing
- sit skiing

Living Skills

Living skills (eg, meal preparation, shopping, check writing, housekeeping, etc) are necessary tasks of everyday life and must be relearned and adapted to a patient's needs. These skills are often reacquired with the help of occupational therapists. Again, reachers and other specialized equipment are used as aids and spared muscles substitute for paretic muscles, a motor learning task that requires practice. These tasks can usually be managed by patients with injuries sparing level C-7 or more. Those with higher injuries need assistance, including environmental control systems and computers. These can be accessed via mouth stick, sip-and-puff, tongue touch pads, eye movements, or voice. Environmental control systems allow the use of telephones, appliances, sound equipment, intercoms, televisions, lights, and door openers—all items that can be accessed by remote control. The complexity of the system will vary according to the extent of the impairment and the patient's financial resources. The use of a computer, accessed through the keyboard using hand or mouth sticks, head controls, or voice activation, allows a degree of control and a range of communication, vocation, and recreation options.

Vocational and Avocational Pursuits

Tools to help patients explore available vocational and avocational opportunities include psychological testing, vocational counseling, assessing physical capacities, vocational remediation and training, identifying sources of financial assistance to support training and education, peer counseling, and job-seeking guidance. Rehabilitation staff can also communicate with potential employers. Hiring an individual with a disability can raise issues about physical accommodations for a disabled person and the psychology of responding to someone who is permanently disabled. Providing technical assistance for workspace modifications and information about disability can also aid patients in their employment pursuits.

Healthcare providers should organize and encourage outings for SCI patients, such as attending sporting and cultural events, eating at restaurants, and shopping. Many individuals with SCI will want to return to a form of physical exercise or sport, although they may initially be apprehensive. They should be made aware that numerous activities have been successfully pursued by people with SCI (Exhibit 16-1).

Patient and Family Education

Families are critical to helping patients adjust to disability. Initially, the concerns of the patient and family often center on prognosis for neurological and functional recovery. Response to these concerns should respectfully acknowledge the uncertainty and difficulty a patient may have in accepting new limitations.

Hope, even if based on an improbable outcome, helps patients cope with the initial grief that accompanies disability. Acceptance of the new reality generally comes with time. Patients and their families can be convinced of the need to learn alternate methods of mobility and self-care.

The focus during rehabilitation is to educate the patient, family, and caregivers on the nature of SCI, maximizing health, and accessing community resources. Educational topics include the following:

- level and completeness of the SCI, which determine the anatomic and physiological correlates;
- prognosis for motor recovery and spasticity;
- current research on spinal cord regeneration and new technology;
- care of neurogenic bladder and bowel;
- prevention of skin breakdown;
- management of autonomic dysreflexia (AD), if applicable;
- psychological adjustment to disability;
- attendant care management;
- sexuality, impaired sexual function, and fertility;
- vocational and educational options;
- avocational outlets, such as wheelchair sports;
- finances; and
- housing.

Various educational tools can be used, such as patient manuals for instruction and reference, weekly class discussions, peer interaction, and one-on-one instruction provided by each member of the rehabilitation team. Intake and discharge meetings with the rehabilitation team further complement instruction. The intake meeting defines specific goals for the hospitalization and addresses specific questions related to prognosis and course of treatment. The discharge meeting discusses plans for follow up, equipment needs, and accessing community resources. Family members and other caregivers of partially dependent patients need to be instructed in their roles. Onsite independent-living apartments allow patients and their caregivers to rehearse tasks, such as shopping, meal preparation, transfers, toileting, and bathing, with staff backup. Day and weekend passes can also be granted once a patient is medically stable and sufficiently trained in self care and mobility. Patients can practice their recently acquired mobility and self-care skills by participating in community outings organized by a recreation therapist. These outings typically include activities such as taking public transportation, visiting shopping malls, going to movies, grocery shopping, banking, and eating in restaurants.

Housing, Finances, and Community Reintegration

Social workers can help patients understand social security disability, Veterans Affairs and Medicare/Medicaid benefits, separation from the military, accessible housing, housing modifications, advocacy groups for the disabled, and legal resources and protections. Occupational therapists can help patients understand accessibility features, such as ramp inclines and door widths.

PREVENTING AND MANAGING SPINAL CORD INJURY COMPLICATIONS

Mortality and morbidity due to SCI are higher in patients with tetraplegia than paraplegia and complete injuries compared to incomplete injuries, both acutely and chronically. Medical complications associated with SCI commonly occur during the first few months following the injury, and patients are frequently rehospitalized to treat acute medical conditions, particularly during the first year postinjury. Many need early diagnosis and treatment to minimize long-term sequelae.

Some conditions occur more frequently in the first several years following injury, such as hydronephrosis, spasticity, and contractures. Suicides are also more common during this time. Other complications become more prevalent with increasing time (eg, musculoskeletal problems), and some complications are most closely associated with the patient's age (eg, cardiovascular complications) or neurological classification (eg, pneumonia, AD). The most common causes of death after the first year following injury are respiratory disorders (22% of deaths), cardiovascular disorders (21%), cancer (11.9%), and sepsis (9.8%).[9] More than 75% of respiratory-related deaths following SCI are due to pneumonia. Urologic causes, which were previously the leading causes of death following SCI, now account for only 2.3% of deaths.

Pulmonary Complications

The inspiratory phase of breathing requires active muscle contraction and depends primarily on the diaphragm innervated at the C-3, C-4, and C-5 levels. Resting expiration is passive and depends on the viscoelastic properties of the lung and chest wall; forced expiration is active, as in a cough for clearing

secretions from the lungs—an action requiring rapid contraction by the abdominal muscles innervated at the T-6 to L-1 levels and the thoracically innervated intercostals. If they are not paralyzed, the clavicular portion of the pectoralis major and the latissimus dorsi can contribute to active expiration.[56] Impairment of the inspiratory phase of breathing and forced expiration cause most SCI pulmonary complications.[57]

Acute Respiratory Failure

The earliest pulmonary complication to manifest in SCI patients is acute respiratory failure. This condition develops most commonly in those with cervical cord injury at the C-5 level or rostral. It may appear immediately after the injury or develop over hours to days as respiratory muscles fatigue. Close monitoring is needed to detect muscle fatigue and avoid emergent intubation. Oxygen saturation may not be a sensitive marker for respiratory failure, especially in patients who are receiving supplemental oxygen, so the vital capacity and partial pressure of carbon dioxide need to be closely monitored. Attention must be directed to aggressive pulmonary toilet and early detection and treatment of atelectasis and pneumonia, which occur in 50% of all patients with motor-complete SCI during the first month following injury.[58,59] The majority of patients who have initial ventilatory failure will eventually wean from the ventilator over a period of days to months. Forced vital capacities in patients with mid-cervical level tetraplegia typically improve over a period of months, presumably due to a combination of inspiratory muscle strengthening and changes in the stiffness of the ribcage. Tracheostomy is usually performed early if rapid weaning is not anticipated (prolonged endotracheal intubation has been associated with airway complications, such as subglottic stenosis). A tracheostomy tube can also allow for direct tracheal suctioning, oral intake of food, and leak speech, in which the tracheostomy cuff is deflated and larger ventilator volumes are delivered, enabling air to leak around the tracheostomy tube and upward through the larynx.[60]

Communication can be a major problem for intubated individuals. Initially, tongue clicking or exaggerated eye blinks can be used for "yes" and "no" signaling, but head nodding may not be possible because of spine instability or neck muscle paralysis with injuries at the C-1 or brainstem levels. Communication can also be pursued through lip reading, communication board, or computer. Eventually, as pulmonary status stabilizes, patients can use leak speech. Ventilator-dependent patients must always carry a suction device for clearing secretions and a self-inflating bag valve mask in the event of ventilator failure.

Atelectasis, Pneumonia, Aspiration, Impaired Cough

Pneumonia is the leading cause of death for both acute and chronic SCI.[61] Almost all SCI patients, with the exception of those with lesions at a low thoracic level or below, have impaired cough because of the loss of abdominal muscle strength. Those with tetraplegia and higher-level paraplegia have impaired inspiratory effort as well. Impaired cough and impaired inspiration predispose a patient to atelectasis. Mucous hypersecretion and hyperviscosity, along with impaired cough, contribute to mucous plugging. All of these factors predispose a patient to pneumonia; those most vulnerable are patients with high cervical cord injuries. The site of pneumonia during the acute phase is most often the left lower lobe, where the sharper angle of the left mainstem bronchus makes suctioning more difficult.[59]

The following strategies promote clearing secretions and reduce the risk of pneumonia: (*a*) turn the patient at least every 2 hours to promote gravity-assisted postural drainage; (*b*) offer incentive spirometry; (*c*) assist cough by manually compressing the abdomen in synchronization with the patient's cough (ie, quad cough; this is contraindicated in the presence of an inferior vena caval filter); (*d*) consider use of a mechanical insufflator-exsufflator, which delivers + 40 cm H_2O pressure, then rapidly switches to – 40cm H_2O pressure, producing an airflow that approximates a normal cough and mobilizes bronchial secretions. Fever, increased purulent sputum, altered auscultation of the lungs, and change in chest radiographs suggest pneumonia. Treatment for pneumonia involves more frequent and more aggressive pulmonary hygiene and antibiotics.

An additional pulmonary problem is aspiration. Those with SCI at level T-10 or above are less able to cough effectively and clear their airways if they aspirate. Major risk factors for dysphagia in patients with recent SCI include tracheostomy and recent anterior cervical surgery.[62,63] Precautions to minimize aspiration risk include restricting oral intake, gastric decompression by nasogastric tube for those with gastroparesis or ileus, upright or side-lying for eating, and avoiding assisted coughing by manual abdominal compression immediately after meals. Patients with suspected aspiration should have a swallowing evaluation that compares the effects of various consistencies, amounts, and techniques and develops optimal feeding strategies. Hyperextension of the neck in a cervical brace can make it easier for a patient to aspirate. Family members and caregivers should be trained to perform the Heimlich

maneuver on someone who is lying down or sitting.

Certain interventions should be taken to reduce the long-term risk of respiratory complications in individuals with SCI, including smoking cessation and pneumococcal and annual influenza vaccination. When a patient has achieved a stable neurological exam and is free of acute respiratory disease, pulmonary function tests should be performed to establish a baseline. For patients with reduced vital capacity, an arterial blood gas test should also be performed as part of the baseline assessment.

Cardiovascular Complications

A variety of cardiac complications can compromise acute and chronic health for the spinal cord injured. Cervical and upper thoracic cord lesions disrupt sympathetic outflow to the heart and blood vessels, and the heart is influenced by unopposed parasympathetic activity. Abnormalities in sympathetic outflow manifest as arrhythmias (including bradycardia and asystole), postural hypotension, and AD. Loss of tonic arteriolar vasoconstriction results in vascular pooling and a lower baseline blood pressure; the expected blood pressure for a person with tetraplegia is around 90/60 mmHg. In addition, decreased muscle activity in the lower extremities leads to venous stasis and the compromise of venous return. Reduced physical function as a result of motor paralysis and lipid dysfunction contribute to long-term cardiovascular risk.

Bradycardia and Cardiac Arrest

Bradycardia that leads to cardiac arrest is a serious early complication of SCI; its incidence in those with tetraplegia is greatest in the first 5 weeks after injury.[64,65] Tetraplegic injuries impair cardiac sympathetic outflow arising at the T-1 to T-4 levels, which normally accelerates the heart. The remaining cardiac innervation is parasympathetic; vagal input slows heart rate. If deceleration is severe, cardiac arrest may result. Tracheal suctioning or hypoxemia can trigger such a bradyarrhythmia, presumably via a vagovagal reflex. Oxygen and atropine can be administered prior to suctioning to inhibit the cholinergic receptors of the vagal efferents to the heart. Patients may occasionally require transvenous cardiac pacing.

Succinylcholine is a depolarizing paralytic medication rarely given as an adjunct to general anesthesia, but in patients with recent SCI, it can result in profound hyperkalemia with cardiac arrest because the neuromuscular junction is hypersensitive to cholinergic agents.[66] Succinylcholine is contraindicated in SCI patients.

Deep Venous Thrombosis, Pulmonary Embolus

Another common and potentially fatal complication of acute SCI is deep venous thrombosis (DVT) with consequent pulmonary emboli. More than 50% of individuals with acute SCI develop DVT if they do not receive prophylaxis. DVT usually develops within the first 3 months following injury, peaking at 10 to 14 days; it occurs less commonly thereafter. Known predisposing factors are venous stasis, a hypercoagulable state following trauma, and venous intimal damage. All of these factors are commonly present in SCI patients. Preventive measures include performing a baseline venous Doppler examination to exclude early DVT, prescribing subcutaneous heparin (typically using low-molecular–weight heparin) and applying venous sequential compression pumps to the lower extremities.[67,68,69] In patients whose thromboprophylaxis has been delayed for more than 72 hours after injury, tests should be performed to exclude the presence of DVT prior to applying venous sequential compression pumps. D-dimer has limited utility as a screening test in this population because of a high false-positive rate. Regular thigh and calf circumference measurements aid early detection of DVT. If a sudden increase or asymmetry in lower extremity circumference develops, or if a patient experiences unexplained low-grade fever or sudden onset dyspnea or chest pain, DVT or pulmonary embolism must be suspected and prompt action taken.[70] The differential diagnosis for lower limb findings can include lower extremity fracture or hemorrhage, HO, dependent edema, or cellulitis. DVT can be diagnosed with ultrasound and, if inconclusive, contrast venogram. DVT is treated with low-molecular–weight heparin, transitioning to oral warfarin for at least 3 months. Bed rest, without lower extremity range-of-motion exercises, is recommended for 48 to 72 hours, until medical therapy is implemented.[67] If anticoagulation therapy is contraindicated or if pulmonary emboli occur despite anticoagulation, an inferior vena cava filter should be considered. The routine placement of inferior vena cava filters in all patients with SCI is discouraged because their placement precludes the practice of quad coughing and their presence may lead to long-term complications. For patients with short-term contraindications to anticoagulation, a temporary filter can be placed and then removed once anticoagulation has been started.

Autonomic Dysreflexia

AD is a delayed sequela of high thoracic or cervical cord injury.[71] This unique manifestation of SCI at

or above the T-6 level presents after the resolution of spinal (neurogenic) shock, typically no sooner than 8 weeks after injury. AD occurs in response to a noxious stimulus below the level of injury, most often due to overdistension of a hollow viscus, such as a bladder or bowel. Loss of supraspinal control results in unmodulated norepinephrine release and exaggerated vasoconstriction of arterioles receiving sympathetic innervation from below the level of the cord injury. Resultant blood pressure elevation may be moderate (140–160/90–100 mm Hg) or severe (> 180/110 mm Hg). In order to compensate for the elevated blood pressure, parasympathetic stimulation to the heart is increased via the vagus nerve and often causes bradycardia.[72] Symptoms of AD include pounding headache, flushing, and diaphoresis. Severity varies among patients; in some, there are no manifestations besides these symptoms, while in others, hypertension can lead to retinal or intracerebral hemorrhage, seizures, and death.[73,74] These symptoms result from high and potentially life-threatening systolic hypertension. To treat the condition, the head of the patient's bed should be promptly elevated to promote dependent blood pooling and to lower the risk of intracerebral bleed, then the noxious triggering agent should be identified and eliminated (adequate bladder drainage can be ensured by catheterizing the bladder, flushing or replacing a possibly clogged indwelling catheter, or untwisting a condom catheter, and bowel impaction can be removed by digital evacuation; lidocaine jelly may be used as a rectal anesthetic to avoid further aggravating the condition). Other triggers of AD may include peptic ulcer, cholecystitis, appendicitis, bowel obstruction, rectal fissure, ureteral stone, UTI, ingrown toenail, fracture, or labor and delivery. If hypertension persists, nitroglycerine paste or oral hydralazine (10 mg) can be administered. Other antihypertensive medications are also used, including phenoxybenzamine, prazosin, mecamylamine, and clonidine.[75] Patients are encouraged to carry an AD treatment card to facilitate prompt and appropriate treatment by health professionals less familiar with complications of SCI.

Orthostatic Hypotension

An early and occasionally chronic problem that is less threatening but nonetheless disabling is symptomatic OH. With OH, patients complain of dizziness, lightheadedness, and fainting when in an upright position. OH is most severe in patients with higher lesions (typically cervical and high thoracic SCI), complete injuries, and after prolonged periods of bed rest. Several factors contribute to OH, including impaired sympathetically-mediated vasoconstriction and consequent blood pooling in the lower limbs and splanchnic bed, decreased sympathetic drive to the heart, and relative volume depletion. Tetraplegic patients who typically run blood pressures of 90/60 mm Hg are at highest risk for developing OH. These patients often become orthostatic when first sitting in the morning. To minimize these episodes, patients can be fitted with an abdominal binder, lower limb compression stockings, and elastic wraps. Liberal salt and fluid intake and elevating leg rests may also help avert orthostasis. Patients may also require midodrine or ephedrine sulfate administered 30 minutes before sitting. In patients with acute SCI, OH improves with repeated sitting trials; individuals with tetraplegia are known to have high renin and aldosterone levels, which presumably compensate for orthostasis. Late worsening of OH could suggest development of posttraumatic syringomyelia or a silent myocardial infarction.

Cardiovascular disease

Cardiovascular disease has emerged as a leading cause of mortality in individuals with chronic SCI.[76] Risk factors include hyperlipidemia, obesity, diabetes, sedentary lifestyle, and reduced physical function. As individuals with SCI live longer, cardiovascular disease has become a major source of morbidity and mortality, and is likely related to increased incidence of obesity, diabetes, and lowered high-density lipoprotein levels after SCI.[77,78] Another cardiac complication for the cervical-cord injured is silent myocardial infarction. Cardiac ischemia and infarction in this population may be painless because the cardiac afferents course along cardiac-sympathetic nerves and enter the spinal cord at the T-1 to T-4 levels. In such cases, the clinical manifestations of myocardial infarct may be subtle and nonspecific, such as hypotension, dyspnea on exertion, orthopnea, or increased pedal edema.

Gastrointestinal Complications

SCI disrupts central nervous system coordination and direct nerve supply to the gastrointestinal tract. Consequences are addressed by region below.

Oral Cavity

The prevalence of periodontal disease and caries increases rapidly with age after SCI. A few of the major causes include failure of patients to control plaque, diet, deficits in saliva production, smoking, and problems with tooth occlusion.[79] Plaque removal is affected by upper extremity function, habit, and caregiver commitment for those who require assistance. Saliva production is often reduced by anticholinergic medications, such as

those used to treat depression or detrusor overactivity. In addition, smoking deactivates salivary leukocytes, further promoting plaque formation and caries. Removing plaque once or twice per day with a fluoride toothpaste and integrating dental care into personal or attendant responsibilities reduces risk for tooth loss.

Esophagus

Dysphagia and esophagitis are the most common problems with the esophagus after SCI. The esophagus can be damaged by traumatic injury or surgery; perforation can cause mediastinitis, and local manipulation to place spine stabilization hardware can damage nerve supply, reducing sensation and peristalsis. Further iatrogenic contributions to swallowing problems include forced supine position, tong traction, and halo-vest immobilization. Dysphagia after acute cervical SCI occurs in up to 20% of cases, and half of the patients are asymptomatic. Predictors include age, tracheostomy, and anterior approach for cervical spinal surgery.[80] Early specific diagnosis can be accomplished with bedside examination by a speech pathologist. Fiberoptic endoscopic examination and videofloroscopic study of swallowing provide further objective evidence of swallowing capabilities. Elevating the trunk to 30° or more for drinking and eating reduces aspiration risk. Interdisciplinary treatment of severe dysphagia can reduce up to 90% of symptoms and can prevent pnemonia.[63] Reflux esophagitis is common after SCI because of abdominal distention, recumbent position, and altered peristaltic patterns. Low-amplitude esophageal contraction, a motility disorder commonly found in neurologically intact patients with gastroesophageal reflux disease, is highly prevalent among people with SCI.[81,82] Patients should be queried for symptoms of heartburn or sour eructation. Mild symptoms should be managed with antacids, and persistent symptoms should be treated with acid-suppressing medication.

Stomach

For the first few weeks following SCI, there is a risk for incomplete gastric emptying and vomiting, particularly in patients with lesions above the T-1 level. Slow gastric emptying can persist into chronic SCI.[83] Acute treatment with intravenous metoclopramide (5–10 mg per dose) or bethanechol (25 mg 30 min before meals) is effective.[84] Without prophylaxis, stress gastritis or peptic ulcers occur in 5% to 20% of acute SCI patients[85,86]; risk increases with age and in lesions above the T-5 level. Parasympathetic stimulation of acid secretion is unopposed because of reduced sympathetic tone, increasing the acidity of gastric juice. Additional sources of gastric lining irritation are corticosteroids (which decrease mucosal resistance), gastric distention, hypotension, and sepsis. Mechanical ventilation and anticoagulation with heparin are also independent risk factors for gastrointestinal bleeding. All these risks can be reduced with preventive use of H_2 blockers, proton pump inhibitors, and stool fecal occult blood surveillance for gastrointestinal bleeding. Continuous suction via a nasogastric tube prevents gastric overdistension, and periodically monitoring gastric secretions for occult blood reduces risk and can detect gastric bleeding early. If prophylaxis is inadequate, upper gastrointestinal endoscopy or angiography (if the cervical spine is unstable) will locate gastrointestinal bleeding and facilitate treatment.

Duodenum

Superior mesenteric artery syndrome is attributed to intermittent functional obstruction of the third segment of the duodenum between the superior mesenteric artery and the aorta. The syndrome is more common in patients with tetraplegia.[87] The diagnosis can be confirmed with a barium upper gastrointestinal series. Findings include dilatation of the proximal duodenum and a "cut off" of the transverse duodenum, blocking barium flow. Sitting the patient up reduces the superior mesenteric forces on the duodenum and facilitates gravity flow in the gut.

Pancreas

Sympathetic–parasympathetic imbalance may result in hyperstimulation of the sphincter of Oddi, leading to stasis of secretions and pancreatic damage.[88] Pancreatitis may appear as early as 3 days after injury, but its presence may be masked because of a patient's loss of sensory, motor, and reflex functions.[89] A pancreatitis diagnosis is supported by increases in amylase and lipase over three times the upper limit of normal.

Gallbladder

Imbalances in parasympathetic and sympathetic nervous system innervation and modulation of gallbladder activity after SCI may reduce contractility and lead to cholestasis. The gallbladder receives sympathetic innervation from the T-7 through T-10 levels. The development of cholelithiasis during acute SCI has been related to acute trauma, decreased gut motility, reduction in food intake, intravenous hyperalimentation, parenteral nutrition, rapid weight loss, and mobilization of peripheral fat stores.[90,91] Gallstones are most prevalent in individuals with SCI and can affect 20% to 30% of chronic populations.[92] In spite of sensory defi-

cits, patients with SCI usually present with symptoms that allow for acute diagnosis and cholecystectomy.[93] The rate of complication following cholecystectomy is not significantly greater in individuals with SCI than in the neurologically intact population[94]; therefore, gallstones detected by ultrasound or other imaging should be documented and considered when patients present with nausea and right upper quadrant pain.

Ileum

Abdominal distention is common during the first few days to weeks after SCI. This is often attributed to nonmechanical intestinal obstruction or paralytic adynamic ileus. Loss of gastrointestinal motility is noted in 63% of SCI patients for several days, and occasionally for weeks, after acute SCI.[89] Several risk factors for loss of gastrointestinal mobility are common in people with SCI, including major trauma, surgery, anesthesia, and severe medical illness.[95] Evaluation requires a careful check for the presence of bowel sounds and focal tenderness (if sensation is present). An abdominal radiograph can gauge severity by revealing nonspecific gas patterns or dilated loops of intestine and air fluid levels, as seen in obstruction or severe ileus. Upright sitting and lateral decubitus position films screen for air, which indicates perforation, under the diaphragm or the abdominal wall. Management includes reviewing amylase levels, electrolytes, and complete blood counts. Hyponatremia and hypochloremia should be treated with intravenous electrolyte therapy. The intestines can be decompressed by placing a nasogastric tube and running it to suction. Symptoms typically improve in days. If not, intravenous metoclopramide is helpful in resolving prolonged gastroparesis or ileus.[96]

Colon

Neurogenic bowel is a term that relates colon dysfunction (constipation, incontinence, and discoordination of defecation) to a lack of nervous system control. There are two basic patterns of dysfunction. Upper motor neuron bowel results from a spinal cord lesion above the conus medullaris and typically manifests as fecal distention of the colon, overactive segmental peristalsis, hypoactive propulsive peristalsis, and a hyperactive holding reflex with spastic external anal sphincter constriction (requiring mechanical or chemical stimulus to trigger reflex defecation). Lower motor neuron bowel results from a lesion that affects the parasympathetic and somatic pudendal cell bodies or axons at the conus, cauda equina, or inferior splanchnic nerve and the pudendal nerve. Lower motor neuron bowel results in low descending colon wall tone and flaccid pelvic floor and anal sphincter. No spinal cord mediated reflex peristalsis occurs. Slow stool propulsion is coordinated by the myenteric plexus alone, and incontinence is common with movement. The denervated colon produces a drier, rounder stool because the prolonged transit time results in increased absorption of moisture from the stool.[97]

Obstipation, Fecal Impaction, Pseudoobstruction, Megacolon

Decreased colonic motility, increased colonic compliance, and anal sphincter spasticity in those with chronic SCI may result in obstipation and fecal impaction.[98] Key elements of a bowel management program include the following: (*a*) adequate fluid intake (1,500–2,000 mL/day); (*b*) high fiber diet (40–60 gm/day); (*c*) bulk cathartics, such as psyllium hydrophilic mucilloid; (*d*) regular mealtimes; and (*e*) timed bowel programs, with evacuations scheduled for every day or every other day, using rectal suppositories (glycerin or bisacodyl) and digital stimulation. Evacuation intervals of more than 3 days increase the risk of impaction and incontinence. The goal of a good program is continence, with focus on techniques that minimize mechanical damage and irritation to the colon and promote long-term health.[99]

Patients reporting poor results with a bowel care program may manifest nausea, vomiting, abdominal distension, early satiety, and shortness of breath from compromised diaphragm descent. Reviewing the elements of the bowel program and ensuring that patients are not on medications that slow gut motility (eg, anticholinergics, narcotics, tricyclics, clonidine, etc) are the first steps to remediation. In addition, patients may require periodic hyperosmotic laxatives (milk of magnesia, lactulose, sorbitol). If these are unsuccessful, saline or phosphosoda enema or whole gut irrigation can be tried. Because enemas tend to stretch the bowel, causing a loss of muscle tone and perhaps AD, they should be used judiciously. In severe cases, nasogastric decompression, with or without a rectal tube, may be required. Rarely, colonoscopy or surgical decompression must be considered if cecal diameter is greater than 12 cm. For those with recurrent bowel obstruction or markedly prolonged bowel programs, refractory to conservative measures, colostomy may be a desirable solution.[100]

Acute abdomen

Acute abdomen is often diagnosed late, with a resulting high morbidity and mortality in patients with SCI. Patients with injury at level T-10 or rostral

and resultant impaired sensation do not perceive abdominal pain or report poorly localized pain at a later stage of acute abdomen.[101,102] The physician caring for the SCI patient must maintain a high index of suspicion for acute abdomen and be alert to its minimal manifestations in SCI patients, which may be limited to tachycardia, increased spasticity, and AD.

Endocrinologic Complications

SCI can directly and indirectly contribute to endocrine and metabolic disorders. One neurologic mechanism for determining endocrine function is via sympathetics that innervate the pancreas, adrenal medulla, and juxtaglomerular apparatus of the kidney and originate from the spinal cord at the T-5 through T-12 levels.[103] Sympathetic activity inhibits insulin secretion, while parasympathetic activity stimulates insulin secretion. Reduced sympathetic activity, as occurs with SCI, elevates plasma renin, which can lead to production of angiotensin II, a potent vasoconstrictor. Aldosterone promotes sodium retention and potassium release.

Acute complications

Within days after SCI, antidiuretic hormone secretion is reduced, resulting in diuresis that can continue for a few days. Adrenocorticotrophic hormone is normally secreted in a diurnal pattern that disappears soon after SCI, causing a drop in blood pressure. Clinicians should be prepared for hypothalamic and pituitary abnormalities following SCI, as well. Stress can contribute to impaired release of growth hormone, which can lead to weakness. At the time of injury, the stress response triggers release of corticotrophin, and resultant release of hormone from the hypothalamus. Adrenocorticotrophic hormone is released and stimulates cortisol production. The syndrome of inappropriate antidiuretic hormone can contribute to free-water retention and electrolyte dilution.

Hypercalcemia, though uncommon, occurs most often in the first months after injury in adolescent males with complete tetraplegia and spinal shock. In addition to gender and youth, risk factors include complete neurologic injury, high cervical injury, and prolonged immobilization. Symptoms and signs are subtle and include malaise, anorexia, nausea, vomiting, constipation, polydipsia, and polyuria. Left untreated, hypercalcemia can progress to lethargy and coma. Treatment includes intravenous saline with furosemide and remobilization. Additional measures may be needed to inhibit osteoclast-mediated bone resorption, such as administering bisphosphonates (eg, etidronate or pamidronate), calcitonin, mithramycin, glucocorticoids, and gallium nitrate.[104]

Osteoporosis

Fracture risk increases with injury duration in individuals with chronic SCI. In general, there is no demineralization in bones above the SCI level. The major contributors to mineral loss after SCI include the amount of paralyzed muscle, lower limb weight-bearing status, presence of spasticity, age, sex, and the time elapsed since injury. Long bones lose more calcium than the axial skeleton, and trabecular bone is more affected than cortical bone. The efficacy of preventive treatments used in the general population has not been conclusively demonstrated in nonambulatory individuals with SCI.

Insulin-Dependent Diabetes Mellitus

SCI can produce profound metabolic consequences that result in disorders of carbohydrate and lipid metabolism.[77] Abnormal glucose tolerance and hyperinsulinemia are common following SCI. SCI complicates the management of diabetes mellitus in two ways. First, individuals with SCI are prone to increased insulin resistance because of decreased muscle mass and obesity that can result from limited mobility. Second, those with tetraplegia fail to exhibit adrenergic hypoglycemic symptoms (diaphoresis and tachycardia). For the latter, clinicians must develop an increased awareness of the neuroglycopenic symptoms (drowsiness or impaired mental status).

Dermatologic Complications

Pressure Ulcers

Pressure ulcers remain a major cause of morbidity and mortality after SCI. Pressure ulcers in individuals with acute SCI commonly delay remobilization, full participation in rehabilitation, and discharge to the community. A clinical practice guideline on the prevention and treatment of pressure ulcers in people with SCI was published in 2000 to improve the quality and consistency of care.[105]

Pressure ulcers are common in the acute care phase, during rehabilitation, and when living in the community.[106–109] Despite efforts to prevent pressure ulcers, there is little evidence that the overall incidence or prevalence of pressure ulcers in people with SCI is decreasing. In addition to medical complications, prolonged bed rest, and loss of function, the cost of treating pressure ulcers is high, estimated to be $3

to $5 billion per year in the United States.[110,111] Braun and colleagues reported that the cost to heal a severe, full-thickness pressure ulcer is $70,000.[112]

A common misconception is that pressure ulcers must first involve the skin; however, most severe pressure ulcers develop in deep body wall tissues over bony prominences and on areas of the body that have little body fat. This includes areas over the ischial tuberosity, sacrum, greater trochanter, heel, scapula, vertebra, malleolus, and occiput. The pathophysiology of pressure ulcers has been related to sustained high loads without pressure relief. Deep, severe pressure ulcers typically start at the bone-muscle interface. Frictional and shear injuries can result in more superficial ulcers.

External forces that result in pressure ulcers include unrelieved direct perpendicular pressure, skin shearing, and friction. Risk factors that predispose an individual to developing pressure ulcers are prolonged sitting, severe spasticity, contractures, edema, anemia, poor nutrition, bruises and skin damage from falls or scrapes, worn or inadequate cushions, urinary or fecal incontinence, excessive sweating, and smoking.[113,114] Some conditions that put acute SCI patients at risk of developing pressure ulcers are anesthetic skin, immobility from paralysis, hypotension, spine immobilization, poor nutrition and subsequent weight loss, febrile illness, urinary and fecal incontinence, flexor spasm, and altered mental status, such as coma, depression, or chronic alcohol use. From 20% to 40% of patients develop pressure ulcers during the first month after SCI.[105,115] Education, awareness, and behavioral change can minimize the risk of developing pressure ulcers. Optimizing equipment, posture, and seating are also critical to prevention, as is avoiding trauma and injury during activities of daily living.

Risk should be assessed comprehensively and systematically, and is high with acute SCI and a significant degree of paralysis. Risk can be decreased by avoiding prolonged immobilization, periodically relieving pressure, using pressure-reducing support surfaces, preventing moisture accumulation, and applying pillows and cushions to pad-contacting surfaces. Use of bed-positioning devices and periodic turns while in bed are also important preventive measures. Pressure-reducing bed support surfaces can be used for at-risk individuals during acute hospitalization, rehabilitation, and in the instance of acute illness.

Specialized wheelchair cushions and mattresses should be used for all people with SCI who are at high risk for skin breakdown. Interface pressures should be evaluated when using foam, gel, and air cushions in a wheelchair. Foam, static air, alternating air, gel, and water mattresses are common pressure-reducing tools. In people with one or more pressure ulcers, pressure-reducing bed support surfaces are an important consideration for healing and preventing ulcers on other surfaces.

Regardless of predisposing factors, pressure ulcers are caused by prolonged pressure or shear sufficient to cause underlying skin and muscle necrosis from ischemia. Yet the exact timing of pressure reliefs (ie, relieving pressure on a periodic basis while sitting in a wheelchair or lying in bed) is still the subject of debate. It is reasonable to turn patients in bed every 2 hours during their acute stay and during rehabilitation. When sitting, body weight is distributed over a smaller surface, so it is recommended that pressure be relieved for at least 15 seconds every 15 minutes.

Pressure can be relieved while sitting in a wheelchair by cushions that distribute weight optimally. Well-fitted wheelchairs with solid (rather than sling) seats; high-quality foam, gel, or air cushions; and pressure releases are standard preventative equipment.[116] The region over the ischial tuberosities is generally at greatest risk when an individual is seated, although postural abnormalities and incorrect seating can put excessive pressure on the sacrum or greater trochanters.

Patients and caregivers must be taught to check the skin daily. If redness or skin breakdown is noted, patients must alter their positions and routines to keep the affected areas free of pressure until redness disappears or the skin heals. For pressure ulcers in regions that receive high pressure while seated, this usually requires strict bed rest. Patients should be made aware that burns can occur by dropping cigarette ash on anesthetic skin, using hot water bottles, placing hot plates or mugs on the thighs, sitting too close to fires or radiators, and bathing in water that is too hot (greater than 98°F or 36.5°C). To avoid skin breakdown, condom catheters or leg straps should not be applied too tightly, feet and nails should be cared for, and shoes should fit well or loosely and be checked for sharp objects before donning. To avoid scrotal breakdown, men must routinely reposition the scrotum forward after transferring to a wheelchair or sitting surface.

Following onset of a pressure ulcer, it is important to assess and describe the ulcer so appropriate treatment can be promptly initiated. Several parameters should be described, including anatomical location, general appearance, size (length, width, and depth of the wound area), stage, exudate, odor, necrosis, undermining, sinus tracts, healing, wound margins, surrounding tissue, and signs of infection. Staging ulcers is an important part of pressure ulcer assessment (Table 16-4).[117]

Superficial pressure ulcers may heal with conservative treatment; deeper wounds may require surgical intervention. Pressure ulcer treatment involves debrid-

TABLE 16-4

NATIONAL PRESSURE ULCER ADVISORY PANEL PRESSURE ULCER STAGES

Suspected Deep Tissue Injury	Purple or maroon localized area of discolored intact skin or blood-filled blister due to damage of underlying soft tissue from pressure or shear. The area may be preceded by tissue that is painful, firm, mushy, boggy, warmer, or cooler as compared to adjacent tissue.
Deep Tissue Injury	Purple or maroon localized area of discolored intact skin or blood-filled blister due to damage of underlying soft tissue from pressure or shear.
Stage I	Intact skin with nonblanchable redness of a localized area usually over a bony prominence. Darkly pigmented skin may not have visible blanching; its color may differ from the surrounding area.
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.
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.
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.
Unstageable	Full thickness tissue loss in which actual depth of the ulcer is completely obscured by slough (yellow, tan, gray, green or brown) or eschar (tan, brown, or black) in the wound bed.

Adapted from: National Pressure Ulcer Advisory Panel. Updated Staging System Web site. Available at: http://www.npuap.org/pr2.htm. Accessed September 15, 2008.

ing necrotic tissue, cleaning the wound bed at each dressing change, and applying appropriate dressings. Dressings that maintain a continuously moist ulcer bed and keep the surrounding intact skin dry are generally preferred. Other important considerations include controlling exudate and filling wounds that have tracks and undermining. Many comorbid conditions may interfere with wound healing. Cardiac, pulmonary, and metabolic (eg, diabetes mellitus) diseases should be stabilized. Nutritional status requires optimization, and tobacco cessation should be strongly encouraged. Wound infection and osteomyelitis will interfere with healing and should be treated. Other issues related to SCI that may interfere with pressure ulcer healing include incontinence, spasticity, HO, and UTI.

Surgical intervention is made when an ulcer is too large to heal with conservative measures, a deep soft tissue or bone infection must be surgically debrided, there are nonhealing tracts, or a bony deformity must be corrected. Myocutaneous or fasciocutaneous flaps are the standard surgical treatment, preceded by inspection of bony prominences and debridement of osteomyelitis (Figure 16-5). A pedicle flap with the suture line away from the area of direct pressure releases skin and underlying muscle while preserving blood supply, repositioning it over the ulcer. The most common examples of pedicle flaps are gluteus maximus rotation flaps to cover sacral or ischial ulcers, hamstring V-Y advancement flaps to cover ischial ulcers, and tensor fascia lata, vastus lateralis, or rectus abdominus flaps to cover trochanteric ulcers. Skin grafts can be used over granulated tissue, but they are not very durable over weight-bearing areas. The possibility for future surgeries should be considered when tissue sites are chosen.

Postoperative care requires rigorous attention to keep

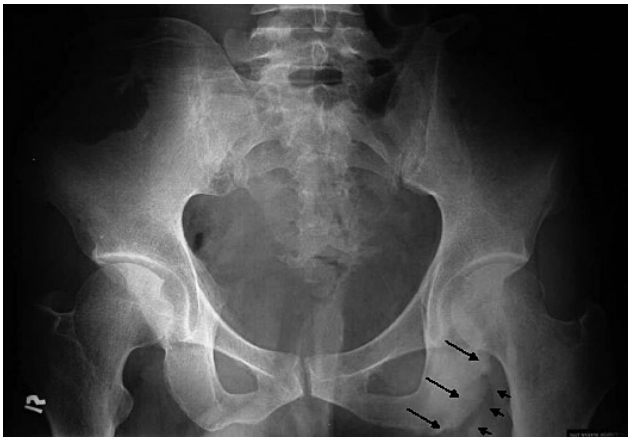


Figure 16-5. With deeper ischial pressure ulcers, the ischial tuberosity commonly develops osteomyelitis. This pelvic radiograph shows an area of marked sclerosis, with a wide zone of transition and cortical irregularity involving the left ischial tuberosity (thin arrow). Areas of lucencies are seen in the adjacent soft tissue (arrowheads).

pressure off the surgical site and allow healing. Following surgery, patients should remain on an air-fluidized bed for a period of 3 to 6 weeks. During this time, tension on flap incisions should be avoided. For patients with spasms, this may mean increased spasmolytic medication and positioning to minimize hip flexor spasms. After 3 to 6 weeks, if the flap is healing well, the patient progresses to a regular hospital bed and lower extremity range-of-motion exercises are allowed, with direct observation of the flap suture lines. Later, the patient is gradually remobilized in a fitted wheelchair. Using progressively longer flap-sitting times over several weeks has been successful following surgery. It is important to reinforce the need to perform regular pressure releases and twice-daily skin checks.

Pressure ulcers may result in complications such as cellulitis and osteomyelitis, which frequently originate through the direct spread of infection. The common presenting symptoms of cellulitis are swelling, redness, local warmth, and fever. The symptoms of osteomyelitis include fever, shaking chills, and purulent drainage. Laboratory findings in both include leukocytosis, elevated erythrocyte sedimentation rate, and positive blood cultures. A radiograph may show periosteal reaction or lytic lesions. Bone scans are of limited use because they are usually positive below a pressure ulcer, whether the underlying bone is infected or not. Magnetic resonance imaging (MRI) findings are somewhat more specific, but false positive tests are still possible. Definitive diagnosis of osteomyelitis is possible through bone biopsy and histologic confirmation, with more reliable results obtained through a needle biopsy or at the time of operative treatment of an ulcer. Cellulitis is treated with oral or parenteral antibiotics (usually taken for 10 to 14 days). Osteomyelitis is usually treated with 6 weeks or more of antibiotic administration and requires bone debridement. Other complications of pressure ulcers that require treatment include wound infection, AD, and malnutrition.

Musculoskeletal Disorders

Musculoskeletal disorders are common among individuals with SCI. Many types of musculoskeletal problems have been reported, including soft tissue and osseous injuries at the time of SCI; traumatic injuries after SCI; unique musculoskeletal disorders precipitated by SCI; and chronic upper limb disorders related to repetitive use, poor biomechanics, and aging. The impact of musculoskeletal problems on the overall function and well-being of a person with SCI cannot be overstated. Even relatively minor musculoskeletal pathologies can result in significant secondary disabilities and new limitations.

Fractures

Fractures at the time of SCI are common and directly related to extrinsic factors (eg, force of injury) and intrinsic factors (eg, bone density). Fractures of vertebrae, limb girdles, and long bones are most common. A high index of suspicion and use of imaging studies are necessary to identify fractures below the neurologic level of injury.

Fractures also commonly occur after SCI (Figure 16-6). The incidence of long bone fractures in the lower limb has been estimated to be 4% to 7%.[118] Fractures are related to osteoporosis that occurs within months after injury below the level of injury. The injury that results in fracture may be minor as bone mineral density decreases and less force is required to produce failure in the bone. The Model SCI System has reported fracture rates based on time following SCI, with cumulative

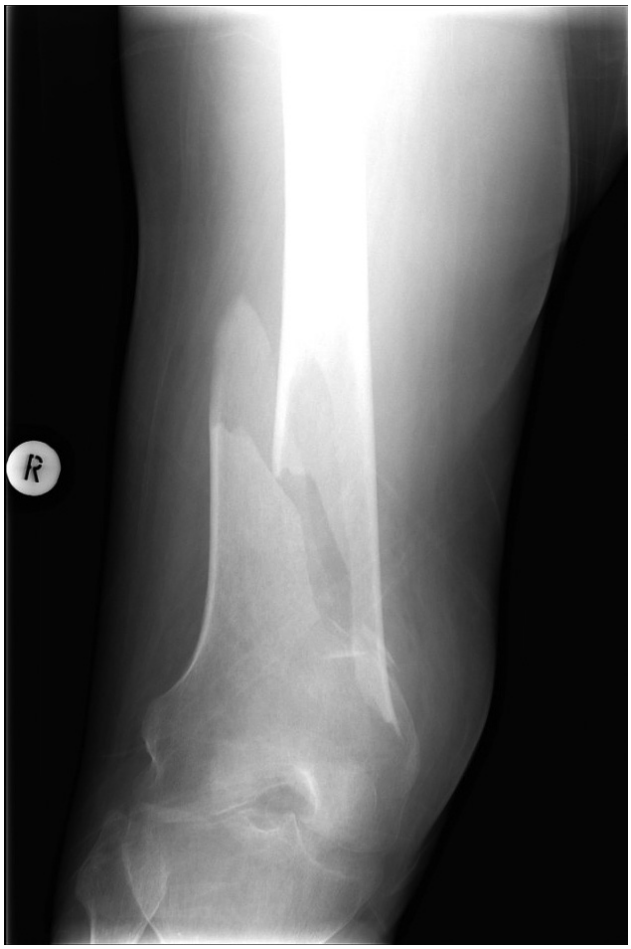


Figure 16-6. Supracondylar fracture in osteoporotic bone in a male with long-standing paraplegia. The fracture occurred secondary to a fall from a gurney.

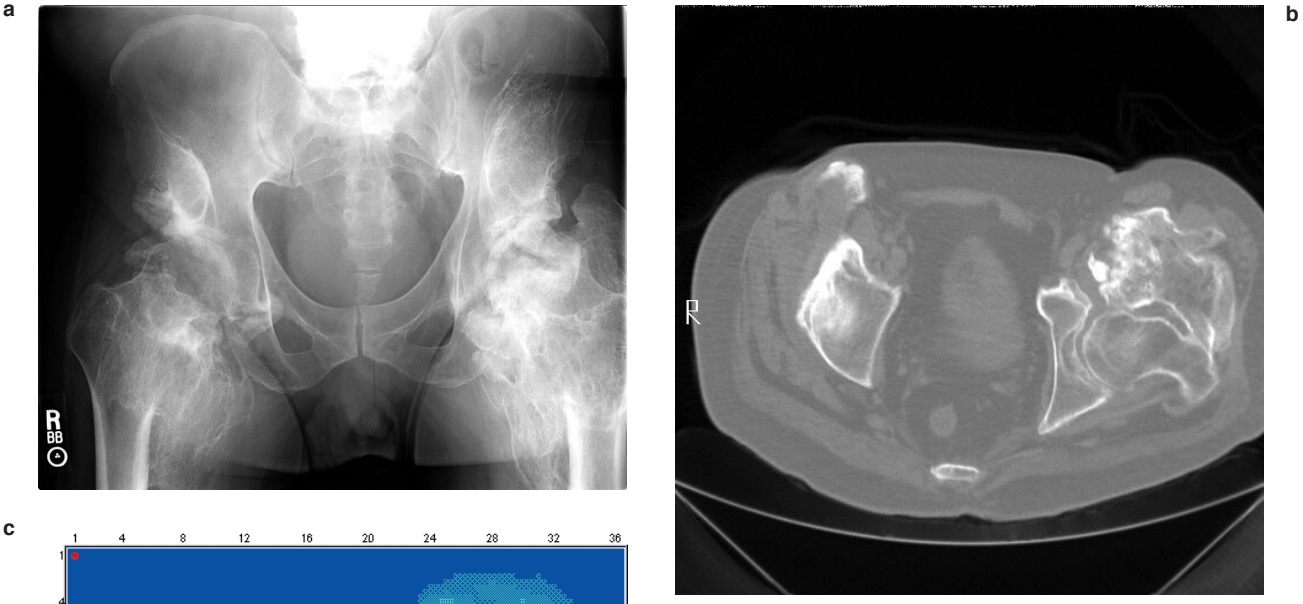


Figure 16-7. (**a**) Radiograph showing severe heterotopic ossification involving bilateral hips. (**b**) Computed tomography scan shows large mass of bone anterior to the left acetabulum. (**c**) Heterotopic ossification can indirectly cause pressure ulcers through restriction of joint range of motion, which results in pelvic obliquity when seated. In this patient with severely limited left hip flexion, a seating interface pressure transducer map placed between the patient and wheelchair cushion demonstrates increased pressure (yellow and red colors) posterior to the right greater trochanter.

incidences of 14% at 5 years, 28% at 10 years, and 39% 15 years after injury.[119]

Managing fractures includes immobilizing the limb and preventing complications. AD may occur in individuals with SCI at level T-6 and above. Other potential complications include DVT, increased spasticity, and the development of pressure ulcers. Well-padded, bivalved casts allow frequent inspection of skin. Delayed union, malunion, and nonunion are common problems after fracture in SCI. Functional consequences, such as difficulty transferring, must be anticipated and addressed.

Heterotopic Ossification

HO is the development of ectopic bone within periarticular soft tissues and is a well-described complication of SCI and traumatic brain injury. Although HO may develop around any joint or within muscle below the level of SCI, it develops most commonly about the hips (Figure 16-7). Onset is typically within the first 5 weeks after SCI, although it may not become clinically apparent for an additional few weeks. The prevalence is thought to be approximately 20% to 30%, although only about half the cases are clinically significant. Between 3% and 8% develop complete ankylosis of the involved joint.[120,121]

Diagnosing hip and shoulder HO may be challenging because the periarticular structures are not as easy to examine as more superficial joints (eg, knee and elbow). HO may present with erythema, swelling, warmth, decreased range of motion, increased spasticity, pain, and fever. Bone scintigraphy and MRI are more sensitive diagnostic tests than plain film radiographs, which may not be helpful if the HO matrix is not yet calcified. Complications include decreased range of motion affecting transfers and seating, skin breakdown, increased spasticity, and DVT.

The mainstay of treatment for HO has been pharmacologic. Bisphosphonates, such as etidronate

disodium, and nonsteroidal antiinflammatories (eg, indomethacin) have been used to treat HO. Low-dose radiation, manipulation, and surgical excision are also reportedly useful for treating HO that does not respond to medications.[122,123]

Contractures

Progressive shortening (contracture) of muscles, tendons, ligaments, and joint capsules results in stiffness, movement limitation, and deformity. Contractures limit self-care, transfers, bed positioning, standing, sitting, and walking. Finger flexion deformities compromise hand function and skin care. Ankle plantar flexion contractures may lead to pressure ulcers and limit footwear. Knee and hip flexion contractures increase the energy cost of standing and walking. Hip adductor contractures limit perineal care. Preventing contractures involves daily range-of-motion exercise, which may be complicated by spasticity. Early contracture may be reversed with stretching with or without heat treatments, such as ultrasound diathermy. Severe contractures may require surgical release.

Chronic Upper Limb Disorders

Upper limb pain and dysfunction are common following SCI. Using the upper limbs for weight-bearing purposes over decades creates biomechanical challenges for limbs that are designed primarily for prehension and mobility. Rotator cuff disease, tendonitis, epicondylitis, arthritis, and carpal tunnel syndrome are all common upper limb problems. Recommendations in *Preservation of Upper Limb Function Following Spinal Cord Injury* address biomechanical stressors, techniques, equipment, musculoskeletal health, and the environment.[47] Most address well-established ergonomic associations between strenuous tasks and musculoskeletal disorders. Minimizing the force and frequency of tasks, such as wheelchair pushing, is achieved by optimizing equipment (eg, wheelchair weight and setup) and technique. Optimizing technique includes education and training in performing tasks (eg, transfer, wheelchair propulsion) and avoiding extreme limb postures. Equipment, technological, and environmental interventions are effective. For example, an elevated wheelchair seat, standing position, or lowered environment prevents repetitive overhead reaching.

Neurological Disorders

A variety of secondary neurologic complications can develop following SCI, including spasticity, neuropathic pain, posttraumatic syringomyelia, and peripheral nerve entrapment.

Spasticity

Spasticity develops in individuals with upper motor neuron damage and spared reflex pathways, with hyperactive phasic stretch reflexes mediated by 1A afferents from the muscle spindle. The 1A afferents respond to rapid stretch, as in a tendon tap, or rapid passive movement of a joint. Clinically, spasticity manifests as hyperactive tendon reflexes, clonus, velocity-dependent hypertonus, and extensor spasms. The latter are commonly elicited by the passive stretch of hip flexors, for example, as the patient moves from sitting to supine-lying position or performs a push-up pressure release. Extensor spasms may interfere during transfers, although some patients use them to aid standing. Flexor spasms can interfere with prone or supine positioning in bed, bed mobility, transfers, and walking; they can also interfere with sleep and contribute to pressure ulcers.

Spasticity and other hypertonus require treatment only if they interfere with function, cause discomfort or poor sleep, hinder caregivers' tasks, or contribute to medical complications, as with pressure ulcer formation.[124] Treatment benefits must always be weighed against the possible beneficial effects of spasticity, such as positioning extremities or aiding standing transfers and walking.

Once spasticity is determined to be more detrimental than beneficial and nociceptive sources such as UTI, renal stone, or fecal impaction have been ruled out, various treatments may be undertaken. The least invasive is daily passive stretching of hypertonic muscles, which reduces tone and spasms for several hours and maintains joint range of motion. Oral medications, such as baclofen, tizanidine, or diazepam most commonly, gabapentin or dantroline rarely, must often be administered. If these prove insufficient, invasive techniques should be considered. Focal spasticity can often be treated with percutaneous injection of botulinum toxin, phenol, or alcohol. Generalized and severe spasticity usually responds to continuous intrathecal baclofen delivered via a subcutaneous pump. Surgical options, such as tenotomy or rhizotomy, are rarely required for severe spasticity that fails to respond to other treatments.

Pain

About one third of individuals with chronic SCI experience severe pain, and up to 80% experience at least some pain on a regular basis. Pain is far more

common in the spinal cord injured than in the general population,[125] and it frequently contributes to lower psychological functioning, interferes with daily activities, and hinders social integration.

Pain usually originates centrally within the spinal cord. It presents with any combination of burning, tingling, or lancinating unaffected by neck movements or posture, and is thought to represent spontaneous discharge of neurons in the ascending pain pathways. Individuals with cauda equina injury experience a series of stabbing pains that radiate down one or both lower limbs every few seconds to minutes. These shock-like pains may be continuously present or appear intermittently. Another type of pain presents as a segmental hyperesthesia over one or two dermatomes at the level of injury. This hypersensitivity to light touch may only be a minor inconvenience and often does not require treatment. Central pain has been treated with a variety of agents, including nonnarcotic analgesics (acetaminophen, acetylsalicylic acid, ibuprofen); anticonvulsants (gabapentin, phenytoin, clonazepam); tricyclic antidepressants (amitriptyline, doxepin); and opioids. Chronic opioid use (eg, methadone and sustained-release morphine) should be administered only in a highly structured setting in which the patient's compliance and psychological status can be carefully monitored.

It is essential to distinguish central dysesthetic SCI pains from pain arising due to mechanical causes, such as persisting cord or nerve root impingement, an enlarging posttraumatic syrinx, or a number of common musculoskeletal conditions. These latter types of pain are generally aggravated by spinal movements or postural changes and may be clinically distinguishable from central pain. Treatment involves addressing the underlying cause of mechanical pain, if possible.

Posttraumatic Syringomyelia

Symptomatic posttraumatic syringomyelia is an uncommon but potentially severely disabling complication of SCI.[126,127] The prevalence of clinically significant posttraumatic syringomyelia has been estimated at 4.5% for tetraplegia and 1.7% for paraplegia. Posttraumatic syringomyelia results from a syrinx at the level of the cord injury (Figure 16-8). It can cause progressive spinal cord damage by extending rostrally or caudally from the injury site, and can extend the length of the spinal cord up to the brainstem. Syringomyelia may develop within months after SCI, but more typically develops over many years.

The earliest symptoms of syringomyelia may be subtle. They include pain, often aggravated by postural change; altered spasticity; sweating; worsening OH; ascending loss of pain and temperature sensation; and ascending loss of segmental reflexes. Ascending weakness is a late manifestation, the appearance of which often indicates that the condition is advanced and may not be reversible. MRI is definitive for diagnosing posttraumatic syringomyelia, but up to 60% of all SCI patients have at least a small intramedullary cystic structure at their level of injury. Careful serial clinical examinations and MRI are useful for determining whether a syrinx is causing neurologic deterioration. Treatment may include minimizing Valsalva maneuvers and spine movements, surgical decompression with syringosubarachnoid, syringopleural or syringoperitoneal shunting of syrinx fluid, or duraplasty. For those with complete cord injury, cordectomy is an alternative.

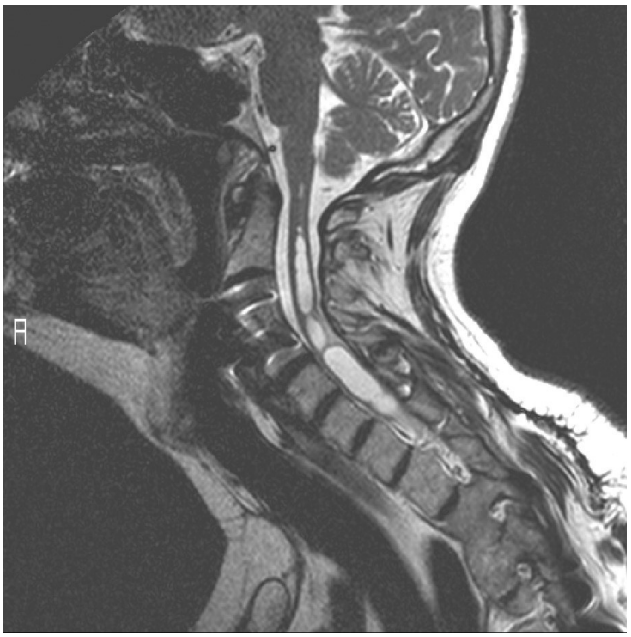


Figure 16-8. Nine months following a motor vehicle accident with upper thoracic fractures and level T-3 complete paraplegia, this patient presented with an ascending sensory level and increasing left hand weakness. Magnetic resonance imaging demonstrated a posttraumatic syrinx extending from the C-1 level through the conus.

Peripheral Nerve Entrapment

Upper limb peripheral nerve entrapment is highly prevalent in individuals with SCI because of the physical demands placed on the upper limbs during functional tasks.[128] Predisposing factors include increased use of the upper extremities for transfers and wheelchair propulsion, resulting in an increased incidence of carpal tunnel syndrome; absent pain sen-

sation, particularly in posttraumatic syringomyelia, resulting in ulnar nerve entrapment; and proximal compression of motor neurons and motor axons in the presence of cervical spondylosis. Radial nerve entrapment, as in Saturday Night Palsy and thoracic outlet syndrome, also occurs with increased frequency. Preventive measures include use of wheelchair gloves, optimized wheelchair setup and propulsion technique, and avoidance of direct pressure to the ulnar groove. New sensory loss, weakness, or loss of function should prompt electrodiagnostic studies to rule out nerve entrapment. Wrist splints, elbow pads, and surgical decompression are common treatments.

Genitourinary Complications

Morbidity and mortality due to urosepsis and renal failure in individuals with SCI have decreased significantly since the 1940s because of improved management principles and techniques. Understanding how the level of the cord lesion relates to bladder function aids in anticipating possible urinary dysfunctions.[129,130] Upper motor neuron/spastic bladder dysfunction is generally associated with fractures at the T-12 vertebral level and above (ie, cervical and thoracic fractures). For patients with these conditions, voluntary control from the brain is disrupted, but reflex activity recovers after spinal shock dissipates. SCI patients with upper motor neuron lesions rostral to and sparing the conus (S-2 through S-4 neurological levels, located at the L-1 vertebral body) usually develop reflex bladder emptying after the resolution of spinal shock. Many of these patients also develop detrusor-sphincter dyssynergia, in which the reflex detrusor contraction occurs simultaneously with external urethral sphincter contraction.[131–133] The result is high bladder pressures that may lead to detrusor muscle hypertrophy, vesicoureteral reflux, hydroureter, hydronephrosis, and renal failure. This process is often clinically silent and needs to be regularly monitored. Lower motor neuron/flaccid/areflexic bladder dysfunction generally occurs when the bony injury is at level L-1 and below and affects the conus medullaris or cauda equina. Individuals do not regain reflex contraction; the bladder remains flaccid and overfills. Mixed dysfunctions may occur with injuries at the T-12 or L-1 vertebral levels, with a variable degree of upper and lower motor neuron dysfunction and less predictable effects on voiding.

Goals for bladder management in individuals with SCI include urinary continence, complete bladder emptying, maintaining safe vesicular pressures during storage and voiding, and minimizing the need for physical assistance. Factors predisposing individuals with SCI to chronic renal failure include high-pressure voiding with reflux nephropathy, nephrolithiasis from urea-splitting bacteria, chronic pyelonephritis, and amyloidosis from chronic pressure ulcers and osteomyelitis.

Bladder Management Techniques

During the initial period of spinal shock, before reflex emptying develops, bladder drainage is managed with an indwelling Foley catheter until fluid intake can be regulated to less than 2 L per day. If they have adequate hand function to perform self-catheterization, patients may be transitioned to intermittent catheterization every 4 to 6 hours to keep maximal bladder distension to less than 500 mL. Fluid intake must be limited to prevent the need for more frequent catheterizations. After several months, reflex detrusor contractions develop in patients with upper motor neuron/spastic bladder; this can be anticipated in those with preserved bulbocavernosus and anal reflexes. When spontaneous reflex bladder emptying develops, anticholinergics may be needed to prevent incontinence between catheterizations. In individuals whose hand function is inadequate to perform self-catheterization, long-term use of an indwelling Foley catheter or suprapubic catheter may be considered. In males with SCI, the use of a condom catheter in conjunction with sphincterotomy or urethral stents may be a reasonable bladder management option. Post-void residuals should be less than 100 mL to prevent bacterial colonization in the bladder from achieving a concentration that will cause symptoms. The less common lower motor neuron bladder (in which the S-2, S-3, S-4 reflex arc is interrupted) can be managed with intermittent catheterization. Although voiding into a condom catheter can sometimes be achieved with prolonged direct compression over the bladder (Crede's maneuver), this can lead to vesicoureteral reflux and hydronephrosis and is therefore discouraged.[134]

Pharmacological treatment can be used to improve urinary continence. Anticholinergic drugs bind to muscarinic receptors in the bladder. Medications, including oxybutinin and tolterodine, are used to decrease detrusor contractility and increase bladder compliance. Side effects are dry mouth, decreased sweating, and constipation. Alpha antagonists (eg, prazosin, terazosin, doxazosin) can occasionally allow patients with incomplete injuries to initiate voiding. Side effects may include hypotension and dizziness. Injections with botulinum toxin have been used to control detrusor hyperactivity.[135]

Surgery is sometimes used to manage neurogenic bladder.[136] Surgeries for hyperreflexic bladder include bladder augmentation with or without urinary diversion (ie, continent ileal conduit diversion and cath-

eterization via an abdominal stoma) or cystoplasty and detrusor myectomy. In instances of areflexic bladder, outlet resistance may be enhanced by artificial sphincters or bladder neck procedures. Additionally, periurethral injections and fascial slings have been used to achieve continence. Newer strategies are arising to manage hyperreflexic bladder.[136] Electrode stimulation involves surgical posterior rhizotomy of the sacral nerve roots and electrode placement at the anterior nerve roots. Stimulation of the anterior roots causes contraction of the detrusor and voiding.

Renal function should be regularly monitored and compromise detected and treated early. A yearly urologic examination is recommended for individuals with chronic SCI, although there is no consensus on the examination components.[136] The exam often includes an evaluation of the upper (via ultrasound, renal scan, computed tomography [CT] scan, or intravenous pyelogram) and lower (via urodynamics, cystogram, or cystoscopy) tracts.

Urinary Tract Infection

Bacteriuria (> 100,000 organisms per mL) is a nearly inevitable consequence of neurogenic bladder.[137] Bacteriuria is not treated if it is asymptomatic. Symptoms of UTI prompting antibiotic treatment may include fever, shaking chills, leukocytosis, hematuria, pyuria, and unexplained increase in spasticity. Preventing UTI may involve ensuring adequate bladder drainage with a low postvoid residual, perineal hygiene, cleaning the drainage tube and bag, clean catheterization (washing hands and catheter with soap and water for those on intermittent catheterization), and eliminating urinary tract stones.[138] The role of prophylactic antibiotics in preventing recurrent UTIs has not been clarified because of concerns about promoting multidrug-resistant organisms.[137] High fevers and shaking chills suggest urosepsis, which requires broad antibiotic coverage (including antipseudomonal coverage), placement of an indwelling catheter to assure bladder drainage, and a renal ultrasound to rule out upper tract obstruction. If the patient fails to respond to antibiotics within 48 to 72 hours or sustains septic shock, an abdominal CT scan should be performed to rule out perinephric abscess.

Bladder and Renal Calculi

Bladder stones may cause bladder spasms or urinary sediment and are often removed by transurethral cystolithectomy. Renal stones are most commonly infection-associated struvite stones caused by vesicoureteral reflux and urea-splitting organisms, such as *Proteus mirabilis*. Another risk factor for stone formation in recently injured patients is hypercalciuria in response to paralysis. If stones are large enough to obstruct the ureter, SCI patients must be closely monitored until treated because upper tract obstruction may be relatively silent in patients with impaired sensation until urosepsis develops. Upper tract stones may be removed by extracorporeal shock wave lithotripsy or percutaneous procedures.

Bladder Carcinoma

SCI patients with long-term indwelling catheters have an increased risk of bladder carcinoma.[139] There is no consensus on appropriate screening, although some advocate for yearly cystoscopy and biopsy of the bladder for those who have had indwelling catheters for more than 10 years.

Sexual Function and Fertility

Individuals with SCI need to be reassured of their ability to express their sexuality, and they may need to be provided with information about techniques and devices they can use to assist them with that expression. Individuals should be encouraged to consider how increased physical dependency will play a role in their feelings of control or powerlessness and desirability. Sexual activities and barriers to sexual expression should be identified and addressed.

Individuals with SCI should be provided with information on sexual function and made aware that sexual desires are psychological and hormonal, not affected by nerve damage. Pleasurable sexual experience may be realized by focusing on skin, lips, and other areas where sensation is intact. Information about manual stimulation, oral–genital sex, positioning, and effects of spasticity can be discussed.

Despite absent sensation below the level of injury, many males, particularly those with neurologic lesions above the S-2 to S-4 levels or incomplete lower lesions, are sometimes able to achieve and maintain erections (the overall rate is 50% for all categories of injury). Oral medications (ie, phosphodiesterase type 5 inhibitors), external appliances, intracavernosal injections (eg, prostaglandin E, papaverine), and implanted penile prostheses are additional options to maintain sexual function. Although many men with SCI are able to achieve erections, most are unable to ejaculate. Vibration-induced ejaculation or electroejaculation can yield sperm for artificial insemination in some individuals. AD is a small risk with these procedures in men with injuries at or above level T-6. Fertility in men diminishes to less than 5% with clinically complete injuries. Semen quality declines rapidly after SCI,

possibly because of recurrent UTIs and increased testicle temperature. Nonetheless, artificial insemination with semen obtained by electroejaculation or more invasive techniques has resulted in pregnancies and healthy live births.

For females, loss of sensation and lack of vaginal lubrication are the primary sexual changes after SCI. Many women with SCI are able to achieve orgasm, although the perception of orgasm may be altered from preinjury experience.[140] Use of a vibrator and lubricants can be helpful when engaging in sexual activity. Emptying the bladder by catheterization and decreasing fluid intake prior to intercourse can help reduce the chance of bladder incontinence.

Female fertility returns to preinjury level once the body has recovered from the initial trauma and menses return. Oral contraceptives and intrauterine devices carry extra risks for women with SCI, making foam and condoms better birth control choices. The physiology of labor is unaltered by SCI; however, impaired sensation may lead to undetected labor pains and presentation in later stages of labor, or, rarely, unsupervised birth. Prolonged or mechanically assisted labor (due to compromised ability to push), and AD in those with SCI above level T-7 are additional risks of pregnancy and childbirth in this population.

Because of the extraordinary physical and emotional demands on patients following SCI, sexual and marital counseling should be offered during rehabilitation. It is essential that the rehabilitation team respect individuals' values and approach this topic with sensitivity.

Psychological Issues

Assessment and support are the two major psychological services offered to individuals with SCI. Assessment is the evaluation of the individual's potential to learn, think, and interact with the environment and others. Standardized tests that measure psychosocial function and intelligence are used for assessment. These tests provide a clear picture of an individual's potential for rehabilitation and suggest appropriate guidelines for educational and vocational pursuits.

The other major psychological service to SCI patients, support, should extend to patients and family members. It should be grounded in knowledge of the patient's history, disposition, values, limitations, and potential. In addition to psychologists, members of the rehabilitation team, particularly social workers, vocational counselors, and recreation therapists, also contribute to a patient's psychosocial adjustment to SCI.

Denial or inability to accept SCI is common and can interfere with rehabilitation, although expectations for recovery may be a source of motivation. Patients are counseled to focus on current rehabilitation issues without destroying hopes of recovery. Confronting a patient's denial can destroy the physician–patient relationship. Eventually, many patients accept their conditions and approach their physicians for information on prognosis.

Associated Traumatic Brain Injury

A significant percentage of SCI patients incur traumatic brain injuries at the time of SCI or have a prior history of traumatic brain injury.[141–143] Resulting agitation, impulsiveness, impaired judgment, and impaired new learning may all impact rehabilitation.

Premorbid Personality

Premorbid factors affect rehabilitation outcomes and should be considered during acute rehabilitation. Risk-taking behavior and drug and alcohol abuse are associated with some cases of SCI and affect adjustment to disability after SCI. More preinjury education is associated with greater likelihood of employment after SCI; among those with less than 12 years of education, only 38% returned to work after SCI, compared to 93% of those with 16 or more years of education.[43]

Reactive Depression and Suicide

A depressed mood is common following SCI. When depression is extreme or prolonged, it can interfere with rehabilitation and precipitate suicide. Suicide rates are higher in individuals with SCI than in a matched general population.[144] Risk factors for suicide include chronic pain and alcohol or drug abuse.

Marital Adjustment and Discord

Divorce is common after SCI. Factors that may contribute to divorce are altered family roles, dependence on a caregiver–spouse (with subsequent burnout), and impaired sexual function.

Health Maintenance

Periodic comprehensive evaluations of medical and functional status are recommended for individuals with SCI after initial rehabilitation. Although the optimal frequency for each type of assessment has not been determined, it is standard practice to perform comprehensive evaluations an-

nually. In addition to a general history and physical examination, the assessment should evaluate the neurologic system, skin, urogenital system and neurogenic bladder management, functional status, and equipment needs. The patients' psychosocial and vocational adjustments should also be assessed.

ORGANIZATION OF A SPINAL CORD INJURY UNIT

Acute trauma facilities, including those used by the US military, usually lack comprehensive SCI units. Rather, they have orthopedists, neurosurgeons, physiatrists, physical and occupational therapists, psychologists, social workers, and skilled nurses who can decompress the spinal cord, establish spine stability, prevent acute medical complications, and begin the initial process of rehabilitation. As soon as patients are medically stable, they are transferred to specialized SCI rehabilitation facilities. The US military has a long-standing agreement with the US Department of Veterans Affairs to transfer patients to one of 23 Veterans Affairs SCI centers. A comprehensive SCI facility employs physiatrists, urologists, orthopaedic surgeons, neurosurgeons, internists, plastic surgeons, rehabilitation nurses, physical and occupational therapists, psychologists, social workers, vocational counselors, orthotists, recreation therapists, respiratory therapists, and dietitians. Specialized facilities in an SCI unit include urodynamics with videofluoroscopy, advanced neuroimaging, a physical and occupational therapy gym, and a wheelchair-accessible pool. All patient care space must be wheelchair and gurney accessible. An independent living apartment, where SCI patients can live with spouse or attendant support, is particularly useful when the patient's ability to return to the community is uncertain.

Compared to a medical, surgical, or general rehabilitation ward, a specialized SCI unit can anticipate and minimize complications and enhance functional outcomes, which results in shorter hospitalizations, fewer rehospitalizations, and less economic cost. Expertise, unique team relationships, and specialized equipment and facilities contribute to the efficiency of an SCI unit. Specialized SCI centers allow clinical and applied basic research to further improve SCI care.

SUMMARY

Prior to World War II, SCI was usually fatal. Now, most SCI patients survive, return home completely or partially independent, have only modestly reduced life expectancy, and achieve satisfactory quality of life. For most SCI patients, these favorable outcomes can be expected if the emergency, acute, and rehabilitative interventions during the first 6 months following injury are appropriate. SCI leads to multiorgan dysfunction, partially mediated by autonomic dysfunction, and loss of somatic sensation, making diagnosis challenging. Strength, sensation, blood pressure control, bladder and bowel emptying, and sexual function are often impaired in individuals with SCI. Life-threatening or disabling conditions that affect organ systems, such as pressure ulcers, DVT with pulmonary embolism, AD, OH, HO, posttraumatic syringomyelia, depression, and suicide, must be prevented or diagnosed and treated early. In addition, SCI patients must undergo extensive training in order to resume maximum function. Specialized training and adaptive equipment is required for mobility and independent self care. Guidance and instruction is required for psychological, social, financial, vocational, and avocational adjustment to disability.

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