Chapter 5

SPINAL CORD INJURY REHABILITATION

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INTRODUCTION

Spinal cord injury (SCI) among the civilian population of the United States occurs at the rate of 7,500 to 10,000 new injuries a year. Young men are the most common victims (80% of the total injured are males) and the peak age of injury is 19 years.[1] Impacts at high speeds are most often the cause: automobile accidents are responsible for 48%, falls for 21%, acts of violence for 15%, sports for 14%, and others for 2%.[1] The most common site of the injury is the cervical cord (54% of cases), then the thoracic (36%), and the lumbar cord (10%).[1] About 250,000 people (roughly 0.1%) of the U.S. population are spinal cord injured.

The demographic profile of the war injured is less well-documented. In Vietnam, 0.9% of those admitted to U.S. Army hospitals had incurred SCIs; 3.8% of those patients died during the initial hospitalization.[2] In various Israeli wars, the percentage of injuries with gunshot wounds 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.[3]

Up until World War II, death following SCIs commonly ensued from uncontrollable urinary tract infections (UTIs) and pressure sores. During the Balkan Wars of 1912–1913, 95% of the soldiers with SCIs died within a few weeks. During World War I, 80% of American troops with SCIs died before they could be shipped home. Prior to this time, the attitude remained essentially what it was when the first recorded description of SCI was made on Egyptian papyrus: "an ailment not to be treated,"[4] a condition for which there was no hope.

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 set up procedures for meticulous care and rehabilitation.[5] These protocols allowed the injured not only to survive, but in many cases to return to their communities.

At the conclusion of World War II, the United States government established the first comprehensive SCI unit at Hines Veterans Administration Hospital in suburban Chicago, using the British model. Compared with the dismal mortality in World War I, over 75% of the paraplegics from World War II were alive 20 years later. Of the 2,500 American paraplegics from that war, over 50% returned to the job force. In 1976, regional centers for treatment of the spinal cord injured were made available to civilians through the Spinal Cord Injury Model Systems Program.

With improved care, particularly of urologic and skin complications, mortality has fallen; late mortality (ie, after initial rehabilitation) was 1.7 times higher for those injured in the 1940s than those injured in the 1960s.[6] In the 1970s and 1980s survival has continued to improve; 7-year survival was 87% during this period.[7]

As mortality has declined, the focus for SCI care has gradually shifted. Initially, the target was defining and adopting procedures and practices to control the often fatal sequelae. Now the focus is directed more toward retraining individuals for independence and return to community.

EMERGENCY TREATMENT IN THE FIELD

Medic (Minutes Postinjury)

Field medics should suspect spine or SCI if the patient suffers loss of consciousness, major trauma, or any complaint referable to the spine (pain, tenderness) or spinal cord (sensory changes, weakness, priapism).[8] In these instances, the following measures then apply: (*a*) do not move the victim until the medic team arrives, unless he is in a life-threatening situation; (*b*) provide cardiopulmonary resuscitation (CPR) but use a jaw-thrust maneuver, rather than a head-tilt or chin-lift maneuver that could further damage the cervical spine; (*c*) immobilize the spine, using manual traction, spine board, and extrication collar or straps so that displaced bony fragments will not further compromise the cord; (*d*) insert a large gauge intravenous line; and (*e*) monitor for impaired breathing and signs of shock.[9] In conditions of war, it may not be possible to observe these protocols because of danger to the evacuation team. In Vietnam, for example, 11%[2] of war wounds were sustained by those caring for the wounded.

First and Second Echelon MTF (Min/H Postinjury)

In treating the patient, as soon as possible, the

treatment team at the first and second echelon medical treatment facility (MTF) should

- maintain systolic blood pressure ≥ 90 mm Hg with pressor agents (eg, dopamine, phenylephrine) and intravenous fluids to replace losses, but avoid over-hydration, which can lead to neurogenic pulmonary edema[10];
- maintain oxygenation with O_2 per nasal prongs or face mask and endotracheal or nasotracheal intubation (avoiding neck movements in those with possible cervical spine involvement) and Ambu bag ventilation[11];
- maintain spinal alignment;
- perform a quick motor examination of elbow flexion-extension, finger abduction, knee flexion-extension, ankle plantar and dorsiflexion, and a limited sensory examination of pin-prick and position sense in the hands and feet to monitor for neurologic deterioration or improvement;
- begin methylprednisolone intravenously as a 30 mg/kg bolus over 15 minutes followed by continuous infusion at 5.4 mg/kg/hour for 24 hours[12,13]; and,
- insert a Foley catheter to ensure bladder drainage and for close monitoring of urine output.

Third Echelon Treatment Facility (Hours Postinjury, No Neurosurgeon Available)

Once the patient has arrived at a treatment center, the staff should

- continue to monitor airway, breathing, and circulation by checking vital signs, arterial blood gas, vital capacity, and inspiratory effort;
- continue to maintain systolic blood pressure > 90 mm Hg with pressor agents (eg, dopamine, phenylephrine) and limited fluid replacement, avoiding pulmonary edema; atropine may be needed for bradycardia < 50 beats per minute;
- continue to monitor urine output via Foley catheter;
- insert nasogastric tube to decompress the gastrointestinal (GI) tract because of paralytic ileus;
- obtain a detailed history including the mechanism of injury, weakness, sensory changes or loss of consciousness at any time after the injury;
- perform a detailed neurologic examination to further define the level and severity of the SCI and to identify associated neurologic injuries (eg, peripheral nerve, plexus or head injury);
- consider a head computed tomography (CT) scan if head injury is suspected;
- rule out associated injuries of the chest with chest radiographs, of the abdomen with physical examination and diagnostic peritoneal dialysis, and of the long bones with physical examination and radiographs; and
- evaluate the spine with physical examination, looking for tenderness or step-off (ie, misalignment), and with imaging studies (Exhibit 5-1), obtaining lateral radiographs of the entire spine, and anterior-posterior and oblique views of the injured spine segments.

Adequate imaging of vertebral level C-7 to T-1 may require manual traction downward on the arms, swimmer's position of the arms, midline tomogram, or a CT scan.

ACUTE NEUROSURGICAL MANAGEMENT AT MILITARY HOSPITAL (HOURS/DAYS POSTINJURY)

Restoring and maintaining spine alignment and assuring decompression of the spinal cord are primary objectives in the military hospital. Spine instability and cord compression are identified via further imaging studies (see Exhibit 5-1). Misalignment is reduced either with skull tongs and traction or surgically, and then alignment is maintained with external or internal means. External stabilization consists of tongs and traction, halo-vest, hard cervical collar (Philadelphia or Minerva), or body jacket. Internal fixation uses wires, rods, plates and screws, and bone grafts to achieve bony fusion across unstable spinal segments.

Early indications for spinal surgery are (*a*) failure to realign the spine nonoperatively; (*b*) decompression of the spinal cord by removal of bone or soft tissue from the spinal canal; (*c*) a penetrating wound that requires debridement; and (*d*) deteriorating neurologic function, though uncommon.[9,14,15] A late indication for surgery is pseudoarthrosis with progressive deformity. A spine is considered stable when there is no progressive neurologic deficit, no progressive spinal deformity, and no spinal pain. Achieving stability requires that bony and ligamentous structures heal. Bony fusion is usually achieved within 2 to 6 months with cervical injuries, gener-

EXHIBIT 5-1

IMAGING STUDIES IN ACUTE SPINAL CORD INJURY

Cervical Spine, Lateral View

1. Include from base of skull to T-1.
2. Identify misalignment of vertebrae or spinous processes.
3. Note prevertebral soft tissue swelling; width < 7 mm at C-3.
4. Note widening of spaces between spinous processes.
5. Vertebral bodies and disk spaces should be full height.

Cervical Spine, Flexion-Extension Views

1. Perform only if NO neurologic deficit, NO spine tenderness, and NO abnormality show on static roentgenograms. These flexion-extension views are to detect subtle instability.
2. Patient must be cooperative; sitting, he slowly flexes then extends the neck, stopping if he feels pain or neurologic change. A lateral c-spine radiograph is taken both flexed and extended.
3. Greater than 3.5 mm displacement or > 11° angulation indicates instability.

Emergent Myelogram CT Scan or Magnetic Resonance Imaging (MRI)

1. Indications: (*a*) assure cord decompression if neurologically incomplete SCI, (*b*) discrepancy between spine fracture and neurologic level, (*c*) deterioration in neurologic status.
2. Nonferromagnetic cervical collar, tongs, or halo for MRI.
3. Hematoma on MRI shows increased T-2, decreased T-1 density subacutely (ie, > 24 hours, due to extracellular methemoglobin); cord hematoma suggests poor prognosis for neurologic recovery.
4. CT scan is more sensitive than MRI for spine fractures; MRI detects some ligamentous injuries but sensitivity is unknown.

ally requiring 2 to 3 months, and thoracolumbar injuries needing 4 to 6 months. Ligamentous injuries are less predictable and require immobilization of 8 weeks. They may necessitate late surgical stabilization.

Wartime SCIs are often gunshot wounds. These military missile injuries differ from civilian bullet wounds because of the missiles' high velocity, large caliber, or both. In destroying more tissue, these missiles increase the likelihood of soft tissue and bone infection. Under these circumstances, debridement, wound exploration, bullet fragment removal, and 2 weeks of broad spectrum antibiotics have been advocated, particularly if the alimentary canal has been perforated. [16–19]

Studies of civilian gunshot wounds reveal that bullets or shrapnel fragments damage the spinal cord directly by passing through or lodging in the spinal canal, or indirectly by transmitting shock waves when bullets pass through adjacent tissue. Indirect SCI by bullet shock waves was noted in 27% of civilian cases.[20] Neurologically complete SCI is associated with larger caliber bullets and thoracic gunshot wounds[20]; cervical or lumbar gunshot wounds often yield an incomplete SCI. Cybulski and colleagues found motor recovery in 56% of those with lower lumbar level gunshot wounds, in 29% of those with thoracolumbar level injuries, and in less than 3% of those with gunshot injuries at or above vertebral level T-10.[21] Reviews of military missile injuries have also demonstrated a better prognosis for motor recovery in those with cauda equina level injuries.[22–24]

Bullet fragment removal has been advocated to prevent cerebrospinal fluid (CSF) leak, infection (meningitis or osteomyelitis), pain, lead toxicity, and neurologic decline from bullet migration or chronic inflammatory response. A few have advocated it to promote neurologic recovery. For civilian missile injuries, bullet removal does appear to promote motor recovery for injuries at the T-12 to L-4 vertebral level but not at higher thoracic or cervical levels.[19] Bullet removal does not prevent complications of CSF leak, pain, or meningitis.[19,25] Fourteen days of

antibiotic administration appears to reduce the incidence of spinal infection when a missile passes through the colon prior to striking the spine.[19]

In addition to spine stability and neurologic recovery, another major focus of early treatment is the prevention of SCI complications. A partial list of the early complications includes acute respiratory failure, atelectasis, pneumonia, bradyarrythmia, hypotension, autonomic hyperreflexia, deep venous thrombosis (DVT) and pulmonary emboli, ileus, gastritis and ulcers, fecal impaction, UTI, detrusor-sphincter dyssynergia, kidney and bladder stones, hypercalcemia, heterotopic ossification (HO), myotendinous contracture and capsule tightness, musculoskeletal and neuropathic pain, spasticity and other hypertonus, posttraumatic syringomyelia, and depression. The prevention and treatment of these complications is addressed in the later section entitled Preventing and Managing SCI Complications.

PATHOPHYSIOLOGY OF SPINAL CORD INJURY

The two types of SCIs are penetrating and nonpenetrating. Penetrating injuries are less common and typically result from a knife blade or bullet lacerating the cord directly.

Nonpenetrating injuries commonly result from bone or herniated disk material compressing the spinal cord or nerve roots. Those with narrow spinal canals, either congenital or because of degenerative changes, are at greater risk for this type of cord injury (Figure 5-1). Nonpenetrating SCI may also result from traction, hemorrhage, or ischemia. The severity of cord damage is proportional to the force and duration of the mechanical trauma; it is also dependent upon the ability of the vertebral column to dissipate those forces.[26]

Either penetrating or nonpenetrating injuries may compromise gray matter, white matter, or nerve roots or any combination. Gray matter contains the interneurons, which receive descending motor and segmental reflex input, and the motoneurons, which provide output to muscles. Being more vascular, gray matter is thought to be more vulnerable to mechanical trauma. 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 are known as the zone of injury. White matter is composed of ascending and descending fibers at the periphery of the cord. Pathologic studies of cord trauma show greater gray

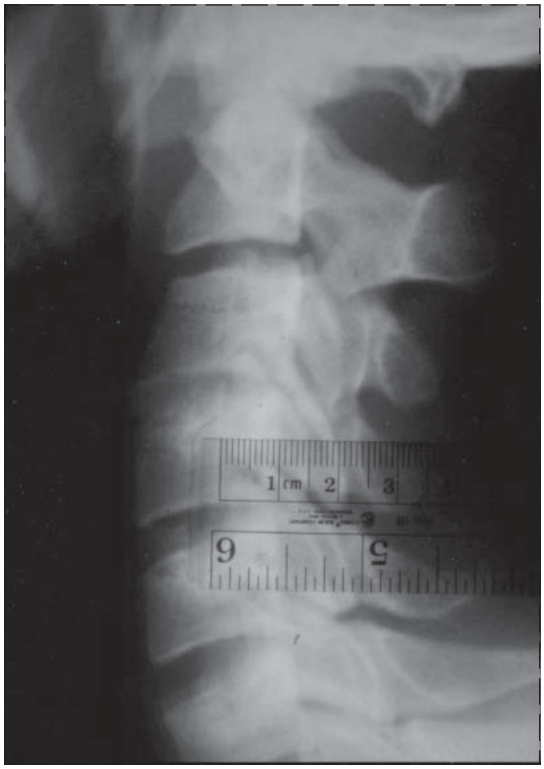

Fig. 5-1. Congenital cervical stenosis. This 33-year-old male football player and All-Service safety became immediately quadriplegic after tackling a ball-carrier. Acute respiratory failure required emergent intubation and artificial ventilation. Note the congenitally narrow cervical canal with an anterior–posterior (AP) diameter of 12 mm. The Pavlov ratio, the ratio of the canal AP diameter to the vertebral body AP diameter, is 0.5; the lower limit of normal is 0.8. Roentgenograms also demonstrated anterior subluxation of C-3 on C-4, for which he underwent anterior diskectomy and fusion. Over several weeks, the patient recovered sufficient diaphragm movement to wean from the ventilator. Over six months, he regained sufficient lower extremity movement to be independent in ambulation with a single point cane. He also experienced good recovery in his left upper extremity such that he became independent in self-care skills; his right upper extremity remained nonfunctional. Voluntary bowel and bladder control returned.

than white matter involvement.[27–29] Damage to gray matter causes segmental changes with denervation muscle atrophy and impaired reflexes. Damage to white matter is more disabling because it results in loss of motor control and sensory input not only at but also below the site of injury; hypertonia and hyperreflexia accompany weakness and sensory loss in such white matter involvement. Nerve root injury often results in an asymmetric level of injury.

Spinal cord damage can arise indirectly from vascular disruption (Figure 5-2). Thus, laceration of the aorta, artery of Adamkiewicz, or a vertebral artery can result in cord impairment.[30–32] Another type of ischemic cord injury is decompression sickness, where hyperbaric exposure (as in underwater diving) followed by sudden decompression, results in gas bubble formation and bubble emboli, which occlude the arterial supply to the spinal cord.[33,34]

While the force of the injury is the most important initial factor, there are several secondary processes that may contribute to cord damage. Studies show that even when a cord injury is clinically complete, the spinal cord is not usually transected. Some argue that much damage results from secondary neurologic injury, a consequence of ischemia, edema, hematomyelia, demyelination, persisting mechanical pressure, lactic acidosis, intracellular influx of calcium, increase of lipid peroxidation, and

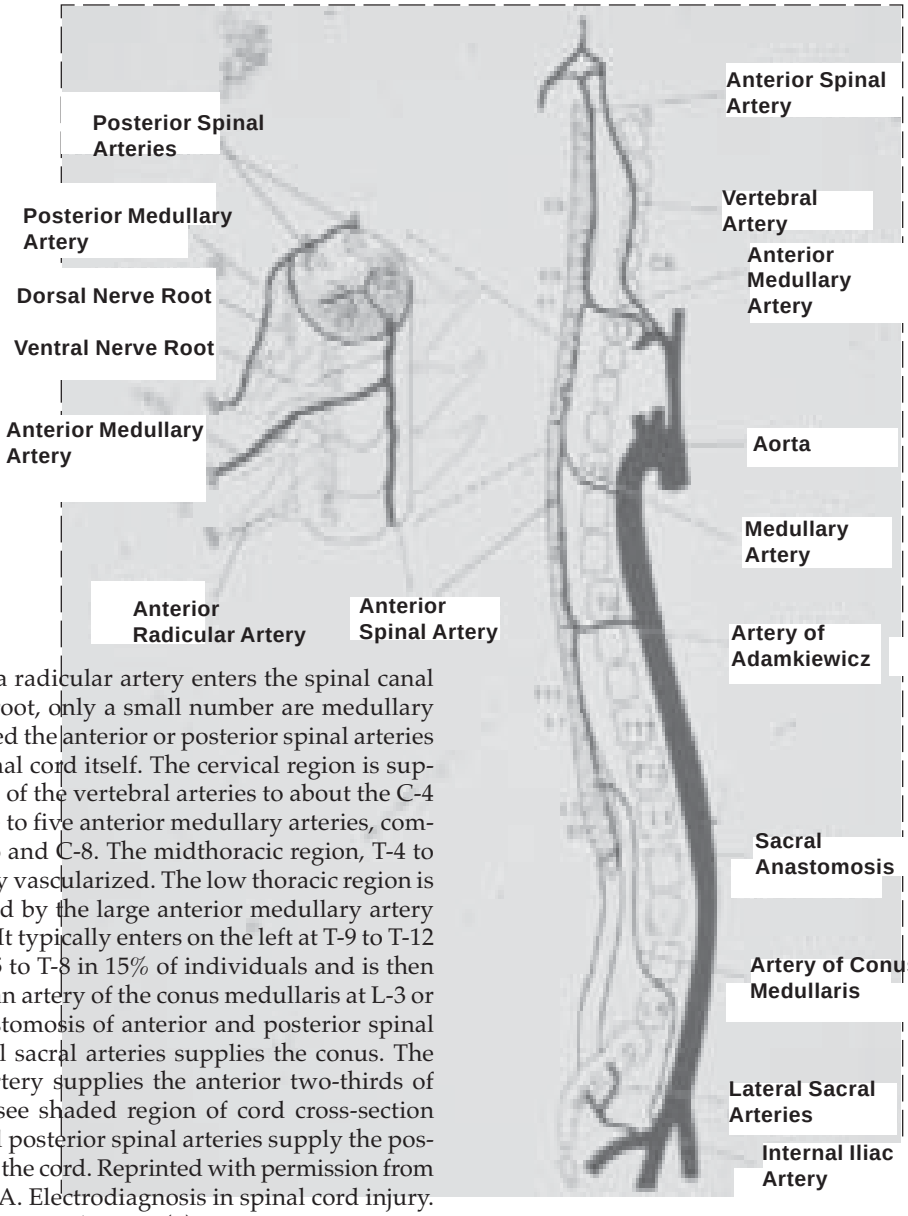

Fig. 5-2. Though a radicular artery enters the spinal canal with each nerve root, only a small number are medullary arteries, which feed the anterior or posterior spinal arteries and thus, the spinal cord itself. The cervical region is supplied by branches of the vertebral arteries to about the C-4 level and by three to five anterior medullary arteries, commonly at C-3, C-6 and C-8. The midthoracic region, T-4 to T-8, is often poorly vascularized. The low thoracic region is primarily supplied by the large anterior medullary artery of Adamkiewicz. It typically enters on the left at T-9 to T-12 but it arises at T-5 to T-8 in 15% of individuals and is then accompanied by an artery of the conus medullaris at L-3 or L-4. A sacral anastomosis of anterior and posterior spinal arteries and small sacral arteries supplies the conus. The anterior spinal artery supplies the anterior two-thirds of the spinal cord (see shaded region of cord cross-section above); the paired posterior spinal arteries supply the posterior one-third of the cord. Reprinted with permission from Little JW, Stiens SA. Electrodiagnosis in spinal cord injury. *New Dev Electrodiagn Med*. 1995;5(3):581.

free radical formation.[35–38] Various early treatments such as hyperbaric oxygen, cord cooling, naloxone, thyrotropin releasing hormone, osmotic diuretics, and others have been proposed to minimize this secondary neurologic injury.[38] However as yet, only high-dose methylprednisolone[12,13] and late anterior decompression[14,39] have been shown to promote neurologic recovery after SCI in humans. Methylprednisolone is effective if given within the first 8 hours postinjury, but even then only modest gains in sensory and motor recovery have been demonstrated.[12,13,40] Because all motor and sensory improvements were grouped together and no measure of functional outcome was used in these studies, the functional significance of methylprednisolone intervention is not known. Another intervention to promote recovery is late anterior decompression (ie, 1–12 mo post-SCI). This procedure reportedly allowed functional recovery in those with incomplete SCI, and residual cord or root compression for those whose recovery had plateaued for 4 weeks or more.[14] Another small study suggests that intravenous gangliosides given in the first month may also improve motor recovery; this recovery is thought to be mediated by white matter long tracts.[41]

In contrast, animal models of incomplete SCI demonstrate that much of the recovery is mediated by spared white matter axons that substitute for those pathways that have degenerated, rather than by resolution of conduction block in white matter axons due to resolution of ischemia, edema, and demyelination. 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 this trauma-induced reactive synaptogenesis, the result is both motor recovery and spinal hyperreflexia. The slow pace of this motor recovery and the gradual onset of this spasticity may be explained by the slowness of synaptogenesis.

One way to enhance this process may be to increase the activity in the spared neural pathways since neural activity seems to be a necessary condition for such recovery mediated by spared pathways.[42] In animal models of stroke, administration of central nervous system stimulants (eg, amphetamine) 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.

Two other methods of regulating activity in the spared neural pathways of the cord, medications and growth factors, are as yet unexplored, and a third issue, the optimal treatment for minimizing developing spasticity during this period of recovery, is unresolved. With stroke patients, some advocate use of reflex facilitation during strengthening exercises to enhance motor recovery; however, this may promote the development of more spasticity. Alternatively, aggressive early treatment to suppress spasticity 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 cord neurons, (gray matter recovery) and root or roots at the site of injury, as is well-documented by Ditunno and colleagues.[43,44] Mechanisms to explain zone of injury recovery include resolution of conduction block or reactive synaptogenesis by descending pathways in the spinal cord, resolution of conduction block or motor axon sprouting by lower motoneurons, or muscle fiber hypertrophy. These recovery mechanisms mitigate two types of weakness—upper motoneuron weakness and lower motoneuron weakness, both of which can be identified electrophysiologically.[45] The optimal rehabilitation interventions for these two types of weakness are not yet known.

NEUROLOGICAL EVALUATION AND PROGNOSIS

The degree of impairment depends on the level and extent of the injury. During development, the spinal cord grows less than does the spine; thus, both for the cervical (Figure 5-3) and the lumbosacral cord (Figure 5-4), the vertebral bodies and dorsal root ganglia are displaced caudal to their corresponding cord segments.

The following are standards in completing the initial evaluation and determining prognosis. The level of a complete SCI is defined as the last level with normal sensation (Figure 5-5, Table 5-1) and antigravity motor strength (ie, grade 3 or better; Table 5-2). If there is a difference from side-to-side, then the level of injury on each side should be described (eg, right C-5, left C-6). If there is a difference between the sensory and motor levels, then each should be described separately (eg, sensory C-6, motor C-8).[46–50] The American Spinal Injury As-

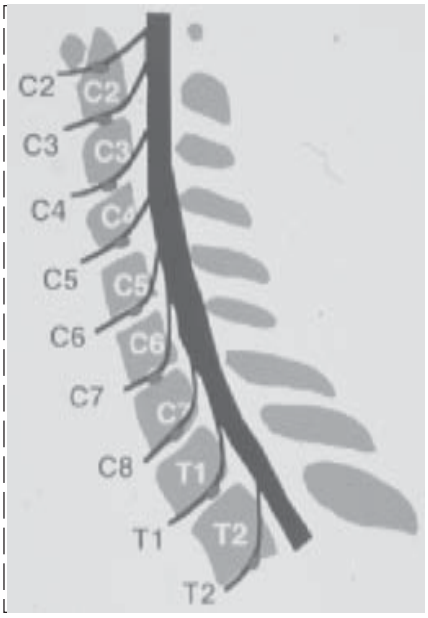

Fig. 5-3. Cervical spine, spinal cord, nerve roots, dorsal root ganglia (shaded ovals), and spinal nerves are shown. Note that there are seven cervical vertebrae but eight cervical cord segments and nerve roots. The C-1 to C-7 spinal nerves exit above their respective bony vertebrae but T-1 exits below. The dorsal root ganglia lie in the intervertebral foramina.

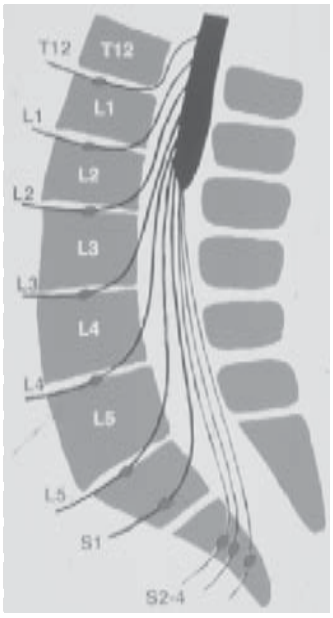

Fig. 5-4. Lumbosacral spine, spinal cord, conus medullaris, and cauda equina relationships are shown. Note that during development, the spine grows much longer than does the spinal cord; thus, lumbosacral cord segments are rostral to their respective spine segments. Observe the considerable length of the nerve roots of the cauda equina, that individual roots exit the canal below their respective spine segment, and that the dorsal root ganglia (shown as shaded ovals) lie in the intervertebral foramina. The spinal cord typically ends at the Ll–2 spinal level in a tapered conus medullaris that contains the S2–4 cord segments, but may end as high as T-12 or as low as L-2, as in this drawing.

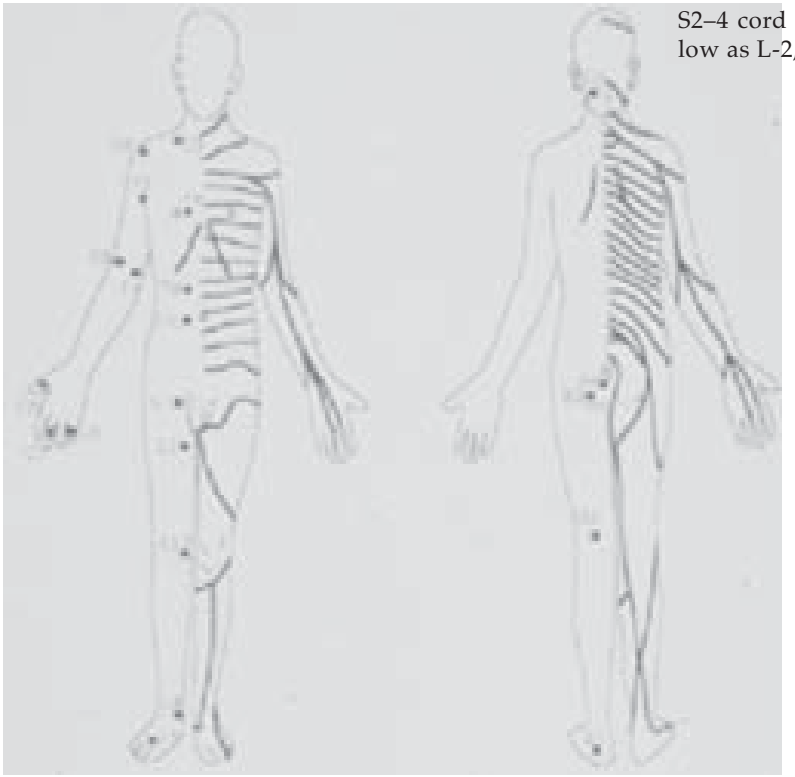

Fig. 5-5. Dematomes and midpoints (squares) commonly used for clinical testing are shown.

TABLE 5-1

NEUROLOGIC EVALUATION: SENSORY LEVELS

Level	Area of Cutaneous Sensation
Cranial Nerve V	Face (pain, temperature)*
C-2	Parietoccipital scalp
C-3	Lateral neck
C-4	Acromioclavicular
C-5	Lateral antecubital fossa
C-6	Digit 1
C-7	Digit 3
C-8	Digit 5
T-1	Medial antecubital fossa
T-2	Apex of axilla
T-4	Nipple line
T-10	Umbilicus
T-12	Inguinal ligament
L-2	Midanterior thigh
L-3	Medial femoral condyle
L-4	Medial malleolus
L-5	Dorsum of foot, 2nd metacarpophalangeal joint
S-1	Lateral heel
S-2	Midline popliteal fossa
S-3	Ischial tuberosity
S-4,-5	Perianal area

*The nucleus of the spinal trigeminal tract relaying pain and temperature sensation extends caudally into the upper cervical spinal cord and is somatotopically organized. Perioral afferents descend the least and synapse in the medulla; afferents from the lateral face descend most caudally into the upper cervical cord. This concentric representation around the mouth is known as the "onion skin pattern of Dejerine." Lesions of the rostral cervical spinal cord result in sparing of pain and temperature periorally but absence, over the lateral, upper, and lower face.

TABLE 5-2

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	*Fexor digitorum profundus* (3rd digit)
T-1	*Abductor digiti minimi*, 1st dorsal interosseus
T-6,-7,-8,-9,-10	Bevor's sign*
L-2	*Iliopsoas*, hip adductors
L-3	*Quadriceps femoris*
L-4	*Tbialis anterior*
L-5	*Extensor hallucis longus*, hip abduction
S-1	*Gastrocnemius, soleus*
S-2,-3,-4	Anal sphincter

SCM: sternocleidomastoid
*Bevor's sign represents upward movement of the umbilicus when the patient attempts a sit-up from supine-lying.
The italicized muscles are the standard muscles used for classification of the level of injury by the American Spinal Injury Association.[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. *Standards for Neurological and Functional Classification of Spinal Cord Injury.* 1992.

sociation (ASIA) has stated that partial sparing of sensation and voluntary movement (ie, less than grade 3 strength) may be present up to three segmental levels below the level of a complete cord injury; this is referred to as the zone of partial preservation. Any sparing four or more segments below this level has been classified as an incomplete SCI. Recently, ASIA has adopted a new classification of complete vs incomplete SCI based on the preservation of perianal sensation and voluntary anal sphincter or toe flexor contractions.[24]

Incomplete SCI is more variable. The level of injury is established similar to complete SCI; the sensory level is the most caudal segment with normal sensation and the motor level is the most caudal segment with antigravity strength. Incomplete SCI has been classified in three ways: (1) by the preservation of sensation and motor strength for the sacral segments,[24] (2) by the Frankel scale (Tables 5-3 and 5-4),[47] and (3) by the pattern of clinical deficits (eg, central, anterior or Brown-Séquard's syndrome) suggesting the transverse extent of the cord injury (Table 5-5).[51–53]

The likelihood of a complete vs incomplete SCI relates to the level of injury. If the injury is at the cervical level (C-5 being most common), 40% to 60% will be motor complete. If it is at the thoracic level, (T-12 being most common) 70% to 80% will be complete except at T-12 where the rate is 44%. At the lumbosacral level, only 10% to 25% are complete.[1]

TABLE 5-3

FRANKEL GRADING OF INCOMPLETE SPINAL CORD INJURY

Grade	Neurological Deficits*
A	Sensory and motor loss complete
B	Sensory incomplete, motor loss complete
C	Motor loss incomplete, nonfunctional
D	Motor loss incomplete, functional
E	Normal function

*Lower extremity sensation, motor strength, and function are assessed.

TABLE 5-4

FUNCTIONAL RECOVERY VERSUS INITIAL FRANKEL GRADE

Grade on Admission*	D or E† (%)	Regressing (%)
A	2	NA
B	20	4
C	50	1
D	98	2

*Frankel grade on admission for initial rehabilitation
†D and E: functional or normal recovery
NA: not applicable
Data source: Stover SL, et al. *Spinal Cord Injury: The Facts and Figures.* Birmingham: University of Alabama; 1986.

The neurologic findings at 72 hours postinjury are better predictors of functional lower extremity return than is an examination performed within hours of the cord injury; a wait of this length allows mild conduction block in the spinal cord to resolve and associated injuries (eg, mild head injury) and alcohol or drug effects to dissipate.[49,54] The most favorable prognostic sign for recovery is the return of voluntary movement below the injury; even a trace of voluntary toe movement at 72 hours is highly favorable for some functional motor recovery (see Table 5-4).[55] Another positive indicator is preserved pain and temperature sensations below the level of injury, often perianally (ie, sacral sparing); 66% to 88% of those with spared pin-prick sensation ambulated, whereas only 11% to 14% of those with just touch sensation spared went on to ambulate.[54,56] In the absence of any voluntary movement,

TABLE 5-5

SYNDROMES OF INCOMPLETE SPINAL CORD INJURY

	Clinical Features	Prognosis
Central cord	Common; lower > upper limb recovery; intact pain and temperature sensation	Often recover bladder and bowel function/control and ambulation
Brown-Séquard	Common; unilateral impaired pain and temperature sensation contralateral to paretic lower limb; unilateral impaired vibration and position sense ipsilateral to paretic limb	Usually recover bladder and bowel function/control and ambulation
Anterior cord	Common; bilateral absent pain and temperature sensation; marked paresis	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	Often with cauda equina injury; symmetric paresis and sensory loss	May recover ambulation; rarely recover bladder and bowel function/control

TABLE 5-6

NEUROLOGIC EVALUATION: SEGMENTAL REFLEXES

	Level	Stimulus	Response	Reflex Type
Biceps jerk	C-5,-6	Tap biceps tendon	Elbow flexion	Stretch
Brachioradial jerk	C-5,-6	Tap radius	Elbow flexion	Stretch
Triceps jerk	C-7	Tap triceps tendon	Elbow extension	Stretch
Finger jerk	C-8,T-1	Tap FDP, FDS tendon	Finger flexion	Stretch
Hoffmann's	C-8,T-1	Flick distal finger	Thumb flexion	Stretch
Deep abdominal	T-5 to T-12	Tap abdominal wall	Abdomen contraction	Stretch
Superficial abdominal	T-5 to T-12	Firmly stroke abdomen	Abdomen contraction	Cutaneous
Cremasteric	L-1,-2	Stroke inner thigh	Testicle elevation	Cutaneous
Adductor jerk	L-2,-3	Tap medial thigh	Knee adduction	Stretch
Knee jerk	L-3,-4	Tap patellar tendon	Knee extension	Stretch
Medial hamstring jerk	L-5	Tap medial hamstring tendon	Knee flexion	Stretch
Ankle jerk	S-1	Tap Achilles tendon	Ankle plantarflexion	Stretch
Extensor plantar	L-5,S-1	Scrape plantar foot	Great toe down (up=Babinski), pathological	Cutaneous
Bulbocavernosus	S-2,-3,-4	Pinch/tug glans	Anal sphincter contraction	Cutaneous
Anal reflex	S-2,-3,-4	Prick perianally	BC sphincter contraction	Cutaneous

BC: bulbocavernosus FDP: flexor digitorum profundus FDS: flexor digitorum superficialis

presence of a bulbocavernosus reflex or other reflex below the level of SCI (Table 5-6), is an unfavorable prognostic sign. It indicates the presence of segmental spinal cord excitability without any descending voluntary activation. From the civilian SCI model system, 31% of all injuries are motor incomplete and recover some lower extremity motor function.[1]

Various incomplete SCI syndromes have been described (see Table 5-5). Several relate to the presumed transverse extent of the cord injury; they are Brown-Séquard and central 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 is good.[52,57] Central cord syndrome is typically due to a cervical cord injury and is associated with greater lower than upper limb recovery. This pattern of greater upper than lower limb deficit may reflect greater gray than white matter damage or greater damage to medial descending motor pathways in lateral funiculi of the white matter.[58] Central cord syndrome often occurs in individuals with a narrow cervical canal, either congenital or from osteophytes or other degenerative changes when the neck is hyperextended as in "whiplash," or when they suffer a fall. 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 factor. The outlook for functional recovery of the lower limbs is poor in anterior cord syndrome.

Cauda equina syndrome results from lumbar fractures at L-2 and below, with damage to the anterior and posterior roots. Central lumbar disk herniation or major trauma, as with gunshot wounds or seat belt injuries, are common causes of cauda

TABLE 5-7

ASHWORTH SCALE FOR GRADING MUSCLE TONE (RESISTANCE TO PASSIVE JOINT MOVEMENT)

Grade	Muscle Tone
0	Normal tone
1	Slight hypertonus
2	Mild hypertonus, joint moves easily
3	Moderate hypertonus, joint moves with difficulty
4	Rigid

Source: Ashworth B. Preliminary trial of carisoprodol in multiple sclerosis. *Practitioner*. 1964;192:540–542.

equina injury. Prognosis for functional recovery is good because the roots are less vulnerable to mechanical trauma than is the cord itself, and the motor axons have some capacity for regeneration to proximal muscles and to the bladder.[59] Often in conjunction with cauda equina syndrome, conus medullaris syndrome involves damage to the sacral segments of the cord, resulting in bowel, bladder, and sexual dysfunction. Fractures at T-12 to L-2 level or damage to the arterial supply of the lumbosacral cord are the most common causes of conus medullaris syndrome.

"Spinal shock" represents depressed spinal reflexes and weakness caudal to an SCI. This is likely caused by loss of normal tonic descending facilitation, since a block of conduction in suprasegmental pathways results in hyperpolarization of cord neurons.[60] Spinal reflexes then gradually return over days, to weeks, to months and become hyperactive as spasticity, flexor and extensor spasms, and hypertonia.[61,62] 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 those with incomplete SCI, tendon reflexes and spasticity may appear within days of the injury (see Tables 5-6 and 5-7).[63] No spinal shock is noted if myelopathy develops gradually as with a cord tumor, cervical stenosis, or syringomyelia; presumably hyperactive reflexes, such as a Babinski sign and ankle clonus, develop before overt weakness because neuroplasticity mechanisms, such as sprouting by reflex afferents and by spared descending pathways, mediate hyperreflexia and spared voluntary movement.

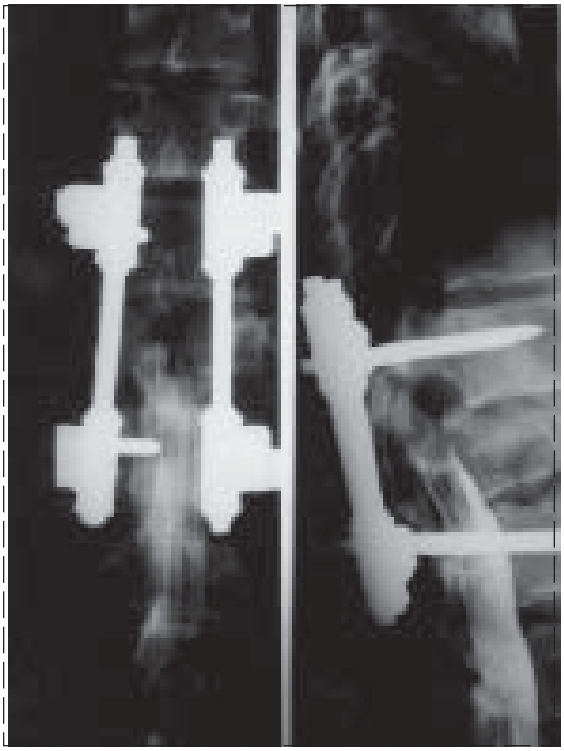

Fig. 5-6. Persisting bony impingement of the cauda equina. This 36-year-old male fell about 100 feet, landing on his buttocks, when his parachute partially collapsed. He was immediately paraplegic and radiographs revealed an L-1 burst fracture. An L-1 laminotomy, decompression of a retropulsed bony fragment and posterior Synthes instrumentation were performed. By two months some strength had returned in the lower extremities and ambulation training began in physical therapy; however, serial Cybex testing revealed worsening of his hamstring strength and repeat peroneal F-wave latencies, and motor evoked potential (MEP) latencies became more prolonged. The above lumbar myelogram was obtained and revealed a persisting complete block to dye flow at L-1 and inadequate bony decompression of the cauda equina. The patient was referred to orthopedic surgery for an anterior decompression; he subsequently regained functional strength in his lower extremities, becoming ambulatory without aids except for bilateral ankle-foot orthoses.

Motor and sensory recovery at the zone of injury is common, even in those with complete SCI. For example, 100% of C-5 and 25% of C-4 quadriplegic subjects regained functional wrist extension.[64] Fifty percent of Frankel A & B subjects and 100% of Frankel C & D subjects regained one segment of cord function by 2 months.[65] Seventy-five percent of those with grade 1 strength and 90% with grade 2 strength at 1 week regained functional use in up-

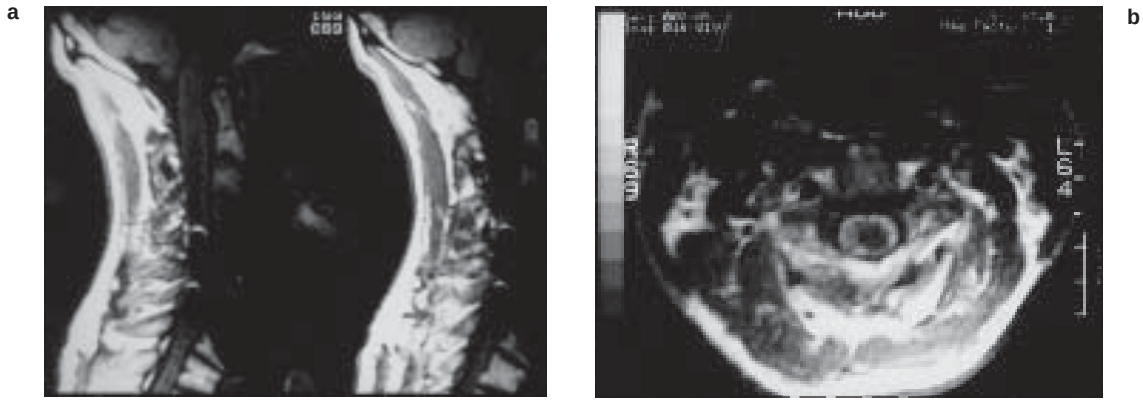

Fig. 5-7. Acute posttraumatic syrinx. This 31-year-old male developed complete C-6 quadriplegia after diving into shallow water; a C-6 burst fracture was evident on radiographs. Initially, wrist extensor strength improved but then at 5 months began to deteriorate, worse on the left than the right. Motor evoked potential latencies to the left biceps and extensor carpi radialis longus became more prolonged. Magnetic resonance imaging revealed a large posttraumatic syrinx extending rostrally to the C-1 level (**a**) and more evident on the left (**b**). Shunting of the syrinx resulted in marked improvement of wrist extension strength and lessening of the patient's neck and arm pain.

per limb muscles by 6 months.[43] Different types of upper limb weakness can be identified electrophysiologically[45] but the optimal rehabilitation interventions for these different types of weaknesses have not been clearly established.

Electrophysiologic studies can be helpful in evaluating SCI. [59,66–69,] They aid in understanding the pathophysiology of the neurologic deficits. For example, they can answer questions such as: Is this primarily upper motoneuron or lower motoneuron-type weakness? Is axonal conduction block or axonal degeneration present? Such knowledge about the pathophysiology can improve prognoses and thus rehabilitation planning. Serial long tract or segmental conduction studies (eg, somatosensory evoked potentials, motor evoked potentials, F-waves or H-reflexes) can aid in determining if residual cord or root compression is present or if neurologic deterioration has occurred (Figures 5-6 and 5-7).

SPINE EVALUATION AND MANAGEMENT

To minimize pain, optimize recovery of the spinal cord, and prevent progressive deformity, it is essential to reduce malalignment and immobilize the spine so bony and ligamentous injuries can heal. The following issues apply to specific vertebral levels of spinal injury.

Atlanto-Occipital Dislocations

These dislocations are commonly missed because they are typically ligamentous injuries; plain radiographs may reveal distraction between the occiput and C-1, often with anterior, vertical, and posterior displacement or any combination of these.[70] Occiput-C-1 injury should also be suspected if (*a*) prevertebral soft tissue swelling is present; (*b*) basion (anterior margin of the foramen magnum)-to-dens is more than 5 mm; (*c*) the clivus is not pointing to the tip of the dens; (*d*) the mastoid does not overlie the dens; or (*e*) the Powers ratio is > 1.0 (Powers ratio is the distance from the basion-to-posterior arch of C-1 divided by the distance from the opisthion [posterior margin of foramen magnum-to-the anterior arch of C-1]). Computed axial tomography with sagittal and coronal reconstructions is usually needed to fully define the instability. Definitive treatment is occipit-to-C-2 fusion with postoperative halo-vest immobilization (Exhibit 5-2). Because the stabilizing ligaments are potentially damaged, axial traction can cause brainstem injury and must be used with extreme caution.

C-1 Ring Fracture

The C-1 Jefferson fracture is a burst fracture of the ring of the atlas, the result of axial loading. If open mouth radiographs reveal lateral displacement of the lateral masses of C-1 on C-2 (sum total overhang on both sides ≥ 7 mm), then the transverse ligament is likely ruptured and the fracture is un-

EXHIBIT 5-2

TYPES OF CERVICAL SPINE ORTHOSES

Soft Cervical Collar: Comfort only, no limitation of spine movement

Philadelphia, Miami J Collar: Limits flexion, not rotation; not effective for upper cervical segments

SOMI* Cervicothoracic Orthosis: Limits flexion, not as good for extension; has forehead strap to allow removal of mandibular pad for eating

Minerva Cervicothoracic Orthosis: Limits flexion, extension, and rotation; not as restrictive as halo-vest

Halo-Vest Cervicothoracic Orthosis: Most restrictive cervical immobilization, but some "snaking" between vertebrae; minimal distraction, may lose alignment mobilizing in halo-vest from traction

*Sternal-Occipital-Mandibular

Source: Benzel EC, Hadden TA, Saulsbery CM. A comparison of the Minerva and halo jackets for stabilization of the cervical spine. *J Neurosurg*. 1989;70:411–414.

stable. The fracture usually decompresses the canal because bony fragments move laterally, often sparing the cord. Treatment is halo-vest immobilization for 3 months.[71]

C-2 Odontoid Fracture

C-2 odontoid or dens fractures are common; they are classified according to the level of dens injury. Type I is a fracture through the upper dens. Type II is at the junction of the dens and the body of the axis. Type III extends into the cancellous bone of the C-2 vertebral body. Type I is rare but usually heals in a Minerva cervicothoracic brace. Type II is associated with poor healing and is managed with a halo-vest or C-1 to C-2 fusion; odontoid displacement ≥ 6 mm is associated with nonunion, so operative fusion is recommended.[72,73] Type III usually heals with halo-vest immobilization.

C-1 to C-2 Atlanto-Axial Instability

C-1 to C-2 instability is due to transverse ligament disruption and is diagnosed on lateral radiograph as increased atlanto-dens separation (ie, > 3 mm for adults, > 5 mm for children). It is usually managed with posterior C-1 to C-2 fusion and postoperative halo-vest immobilization.[74]

C-2 Ring or Hangman's Fracture

The Hangman's fracture is a misnomer; the hanged man, not the hangman, suffers this fracture. Neurologic function may be spared. Type I is minimally displaced (ie, < 3 mm) and is managed with a Minerva cervicothoracic brace for 8 to 12 weeks. Type II is displaced ≥ 3 mm and is managed with a halo-vest for 12 weeks. Type III is associated with C2-3 facet dislocation and usually requires C2-3 fusion with postoperative halo-vest immobilization.[75]

Lower Cervical Spine Injuries

Unilateral facet fracture or dislocation or both is associated with about 25% displacement of adjacent vertebral bodies; bilateral facet involvement is associated with 50% or more displacement. Most can be reduced with cervical traction, although posterior fusion is often used to control persisting instability. Anterior injuries such as a vertebral body burst fracture or acute cervical disk herniation are treated via anterior vertebrectomy or diskectomy followed by a bone graft and often a metal plate to hold the graft in place; postoperatively, a Minerva cervicothoracic brace or a Philadelphia cervical collar may be used to help assure spinal alignment until bony fusion is achieved. Posterior arch injuries, as evidenced by widened spinous processes or facet/laminae fractures, are managed through posterior bony fusion supplemented by wiring between spinous processes or posterior metal plates with pedicle screws. Again, an external cervical brace is usually recommended to assure bony fusion.[76]

Thoracolumbar Fractures

Fractures are most common at the thoracolumbar junction (T-11 to L-2), the junction between the stiff thoracic spine and the mobile lumbar spine. Compression fractures represent wedging of the vertebral body anteriorly with preserved height posteriorly (the anterior column of Denis)[77]; compression fracture of a single thoracic level can be followed with serial radiographs and managed with bed rest, analgesics, and a lumbosacral corset or thoracolumbar Taylor brace for comfort. Lumbar or multilevel thoracic compression fractures are often

managed with TLSO (thoracolumbosacral orthosis, a custom-molded, body jacket); posterior fusion may be required if angulation is greater than 60°. Burst fractures are common axial loading injuries with failure of both the anterior and posterior vertebral body (ie, the anterior and middle columns of Denis) with variable compression of the cord by retropulsed bony fragments.[78] Characteristic imaging studies include loss of height with cortical fracture of the posterior vertebral body on lateral radiograph, increased interpedicular distance on AP radiograph, and retropulsed vertebral bone fragment into the canal on CT scan. Burst fractures may be managed with bed rest in extension for 7 weeks then mobilized in a TLSO for 7 to 11 weeks if there is less than 15° kyphosis, less than 50% canal narrowing, and less than 40% vertebral body wedging. More severe burst fractures require operative treatment to decompress the cord and to prevent progressive deformity. For those neurologically intact, posterior Edwards rods and bony fusion at T-10 to L-2 and posterior plate/pedicle screw constructs at L-3 to L-5 have been recommended.[79] For those neurologically impaired, anterior decompression and Kaneda instrumentation (anterior rods with screws into the vertebral body above and below the fracture) at T-11 to L-3 and posterior decompression with posterior plates/pedicle screws at L4-5 have been recommended.[79] Common flexion-distraction thoracolumbar fractures include seat-belt type injuries and fracture dislocations. Seat-belt type injuries cause damage to the middle and posterior columns of Denis[77]; they do not typically cause neurologic injury. Fracture-dislocation injuries damage all three columns of Denis and result in subluxation or dislocation of adjacent vertebrae. In general, if the middle column of Denis is disrupted then the spine is unstable; exceptions include fractures above T-8 if the ribs and sternum are intact, fractures below L-4 if the posterior elements are intact, and Chance fractures (single-level seat-belt type injuries of the middle and posterior columns with the latter involvement through bone).[77,80] Unstable flexion-distraction thoracolumbar injuries, such as seat-belt type injuries and fracture dislocations, are managed using posterior stabilization with posterior rods (Harrington, Luque, Edwards, or Cotrel-Dubousset rods) or a plate/pedicle screw system. All of the above anterior or posterior operative stabilization procedures are followed by immobilization in a TLSO for 4 to 6 months to assure solid bony fusion.[80,81]

INITIAL REHABILITATION (WEEKS/MONTHS POSTINJURY)

Functional Outcome

Rehabilitation goals vary according to the level of injury and the extent of damage to the cord.[82] When the injury is complete, the functional outcome depends to a large degree on the level of injury. The lower the cord level, the more voluntary movement is preserved and the greater the expectations for independence.

Mobility

Essential to a resumption of life outside the hospital is mobility. 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 down tasks into components and begin with discrete units that can be learned separately and then combined into completed units; and (*c*) teach the patient to substitute with head motions, momentum, and preserved muscles for weakened or absent 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 5-8).[82]

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

Wheelchair Use

One of the first objectives is to get the patient sitting upright, a task usually attended to by physical therapists (PTs) 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 (see section titled Orthostatic Hypotension later is this chapter). Two aids in achieving upright sitting are the tilt table and the reclining wheelchair. Progressive increase of the verticality of the tilt table challenges the cardiovascular system; over time, this improves systolic blood pressure and cerebral blood flow in the

TABLE 5-8

EXPECTED FUNCTIONAL OUTCOME FOR COMPLETE SPINAL C0RD INJURY

Cord Level	Preserved Muscle	Eating	Dressing	Transfers	Mobility		Writing
					Indoor	Outdoor	
C-1,-2	Trapezius	D	D	D	I	I	I
	Sternocleidomastoid			lift or pivot	power wc sip and puff		computer sip and puff
C-3,-4	Diaphragm	D	D	D	I	I	I
	Neck flexor/extensor			lift or pivot	chin control		mouthstick
C-5	Deltoid	I	D	D	I	I	I
	Biceps	cuff		lift or pivot	power wc		cuff
C-6	Latissimus	I	I?	I?	I	I?	I
	Serratus anterior	cuff		loops	manual wc		wrist-driven
	Extensor carpi radialis			sliding board			splint
C-7	Triceps	I cuff	I reachers adapted clothes	I	I manual wc	I	I wrist-driven
C-8 to T-1	Hand	I	I	I	I	I	I
	Intrinsics				manual wc		
T-2 to T-6	Intercostals	I	I	I	I	I	I
	Paraspinals				manual wc		
T-7 to T-12	Abdominals	I	I	I	I manual wc	I	I
L-2	Iliopsoas	I	I	I	I crutches	I? KAFOs	I
L-3	Quadriceps	I	I	I	I crutches	I AFOs	I
L-4,-5	Tibialis anterior	I	I	I	I	I	I
	Extensor hallucis longus				crutches	AFOs	
L-5 to S-1	Hamstrings	I	I	I	I	I	I
	Gluteus medius				AFOs	AFOs	
S-1,-2	Gluteus maximus	I	I	I	I	I	I
	Gastrocnemius-soleus						
S-3,-4	Anal/urethral sphincters	I	I	I	I	I	I

AFO: ankle-foot orthosis
D: dependent
I: independent
I?: possibly independent
KAFO: knee-ankle-foot orthosis
lift: hydraulic lift transfer
pivot: quad pivot transfer
wc: wheelchair

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; the staff can immediately recline the chair back if the 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. Once stable sitting is achieved, the patient is fitted for a wheelchair.

The wheelchair is the patient's key to mobility. Helping the patient select the correct chair is a complex task. The chair should be custom fit according to leg length, thigh length, pelvis width, trunk height, and forearm length. It should be efficient, appropriately stable, maneuverable, and matched to the strength and coordination of the user; a wheelchair that enables one patient to negotiate a curb with a "wheelie" may put another at risk for a dangerous fall. Extras to consider include antitip devices, lap trays, arm rests, and foot and leg supports—items that further tailor the chair to the physical condition and needs of its user. Aesthetics and cost must be considered as well.

Individuals with quadriplegia as high as the C-5 level may be able to propel a manual wheelchair. The benefits are exercise, backup in the event that the power wheelchair malfunctions, and the option of traveling in a passenger car. Propulsion for these higher level cervical injuries is possible by the use of 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 the wheelchair handrim by friction. Many of these same individuals will also require a power chair for outdoor mobility involving distances, rough terrain, or grades.

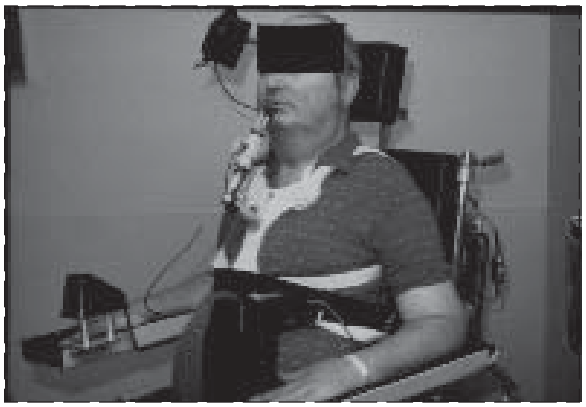

Fig. 5-8. Chin control. This individual with C-4 quadriplegia uses chin control to steer and to recline his power wheelchair and to operate an environmental control unit (ECU) with an infrared interface to a speaker phone, television and light controls.

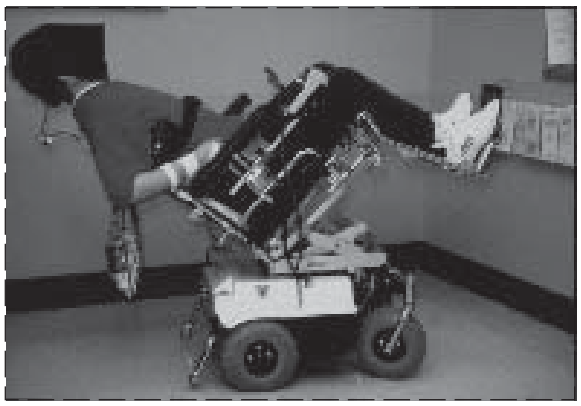

Fig. 5-9. Joystick control and tilt-in-space recline. This individual with C-5 quadriplegia has had sufficient recovery of C-5 innervated shoulder external rotators (supraspinatus, infraspinatus), shoulder abductors (supraspinatus, deltoid), and shoulder flexors and extensors (anterior and posterior deltoid) to operate a joystick hand control for his power wheelchair and a forearm lever for his tilt-in-space recline. This latter type of recline eliminates the hip extension stimulus, which can elicit extensor spasms.

Power wheelchairs are the only feasible means of mobility for some. With the power chair, in addition to the above considerations, the method of control must be determined: manual joystick, chin, sip and puff straw, mouth stick, or tongue. (Figure 5-8). For these patients, reclining mechanisms to minimize the risk of skin breakdown are essential. Two such mechanisms are the zero-shear recline or the tilt-in-space recline; the latter is less likely to elicit extensor spasms (Figure 5-9).

Walking

From sitting, some patients will move to walking. Ambulation goals differ depending on the level of the SCI and whether the cord injury is complete or incomplete. For complete SCI, the goals are exercise, short distance, and level mobility. Those with complete injuries at the T-6 level or below with no other major medical complications are the usual candidates for this training. Contraindicators are limited lumbar spine extension or limited hip, knee, and ankle joint motion. Initially, patients are taught to stand in parallel bars and maintain balance with-

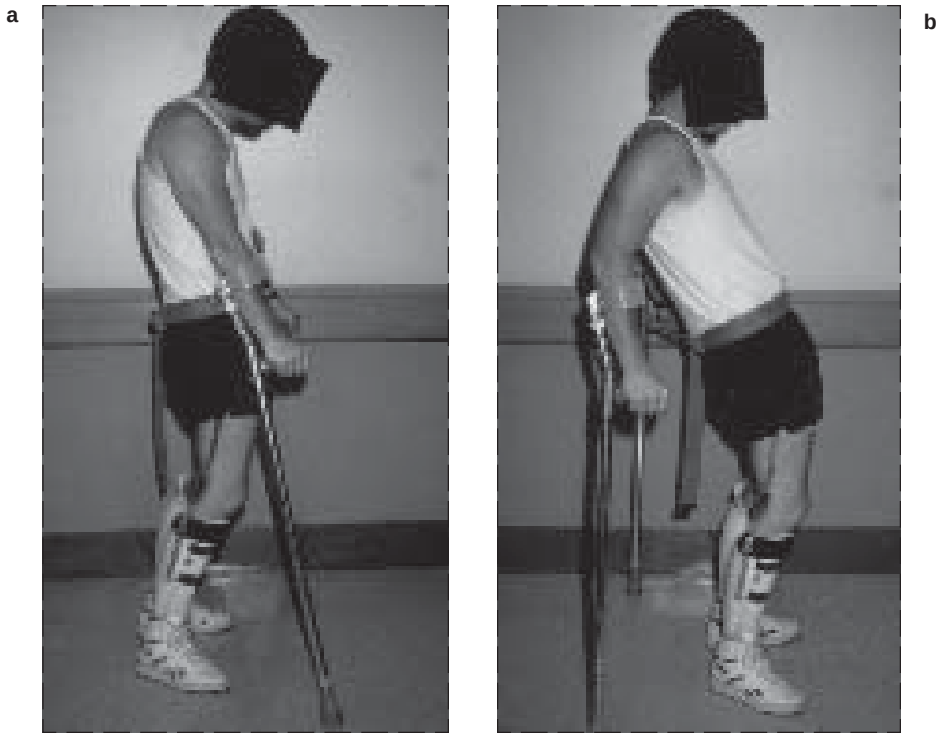

Fig. 5-10. Swing-through gait. This T-6 paraplegic individual ambulates up to one block at high energy cost, using bilateral knee-ankle-foot orthoses, locked at the knee and ankle. He maintains hip extension by positioning the center of gravity of his upper body behind the axis of rotation of his hip joints, both (**a**) at the beginning and (**b**) at the end of each swing-through stride.

out arm support, using temporary knee-ankle-foot orthoses. They then learn to walk with a swing-to gait, using 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 5-10). The energy expended for this type of walking can be as much as 800% of able-bodied walking and manual wheelchair propulsion.[83] This type of walking also requires considerable motor coordination. Because of the high energy and coordination demands, many patients trained in this skill do not continue after discharge. Accordingly, only those who demonstrate good progress and sustained motivation with temporary bracing should be fitted with custom knee-ankle-foot orthoses and proceed to learning a swing-through gait. This requires use of forearm crutches while the patients plant their feet in front of their shoulders for faster walking.

For incomplete SCI, the goals vary widely from assisted standing-pivot transfers to long distance ambulation, depending on the extent of sparing 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.

Driver's Training and Community Mobility

The ultimate community mobility is the automobile. Some patients with injuries as high as C-6 can learn to drive with hand controls, although at the C-6 level, a specialized van with sensitized steering and braking is often required. Those at C-7 or lower can use a passenger car with hand controls. 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 the patient is a candidate for driver's training. If the patient passes this screening, then a vehicle with ap-

propriate adaptations should be acquired. Adaptations include hand controls for brakes and gas, steering wheel attachments, power seats, and vans with hydraulic lifts.

Self-Care

Along with increased mobility, independence in self-care is a major step toward independence for those with SCI. Self-care includes feeding, bathing, dressing, grooming, and toileting. Those with injuries at the C-7 level or below can usually achieve independence in all of these activities. Occupational therapists (OTs) and rehabilitation nurses work with patients to master new techniques for accomplishing these tasks. Alternate strategies such as using a tenodesis grip for holding eating utensils or writing implements must be learned and mastered. A tenodesis grip involves using wrist extension to passively achieve some finger flexion for grip. A wrist-driven flexor hinge splint improves the strength of this tenodesis grip. Other special equipment also aids independence; for example, reachers, universal cuffs, built-up handles on utensils, and a tub bench. Tendon transfers[84] or an implantable functional electrical stimulator[85] may achieve a more functional pinch, although costs and training time are considerable.

With C-6 level or higher injury, partial independence may be achieved. For those tasks that require assistance, instruction takes place on two levels: the patient is taught how to teach others; and a caregiver, often a member of the family, is instructed in how to render assistance or complete the activity. Independence in self feeding with set-up is an example. The assistance needed may include fitting the universal cuff and utensil on the 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.

Living Skills

Living skills (meal preparation, shopping, check writing, housekeeping, and so forth) are necessary tasks of every day life and must be relearned and adaptation made. These skills are often reacquired with the help of OTs. Again, reachers and other specialized equipment are used as aids. Spared muscles substitute for paretic muscles, a motor learning task that requires practice. These tasks can usually be managed by patients with injuries sparing C-7 or more. Those with higher injuries will need assistance that may include 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 powered by electricity through remote control. The complexity of the system will vary according to the extent of the incapacitation and financial resources. Computers allow word processing and written communication, spreadsheet use and money management, graphics and computer-aided design, access to books through CD-ROM discs, on-line bibliographic services and databases, and recreation. The use of a computer, accessed through the keyboard using hand or mouth sticks or through scanning and Morse code, allows a degree of control and a range of vocational and recreational options heretofore impossible.

Vocational and Avocational Pursuits

Computers may be one significant component of an individual's return to an abundant life that includes work and recreation. The patient's significant stake in the outcome makes it essential that he or she take the initiative in exploring what opportunities are available. Tools to aid the person in this search include psychological testing, vocational counseling, assessment of physical capacities, vocational remediation and training (where appropriate), identification of sources of financial assistance to support the training and education, peer counseling, and instruction in job seeking tactics. The rehabilitation staff can also communicate with potential employers. Hiring an individual with a disability can raise a host of unsettling issues that may center on physical accommodations for a disabled person and the psychology of responding to someone who is permanently disabled. Providing technical assistance for work space modifications and the dispelling of fears and stereotypes about the disabled can also aid patients in their employment pursuits.

How the patient will want to utilize time that is not controlled by the demands of living is his or her domain. SCI, at least initially, opens enormous amounts of time for personal pursuits. The patient should be helped to use this opportunity. Eventually, many will want to return to ordered forms of recreation and public activities. Community outings such as attending sports and cultural events, eating at restaurants, shopping, and so forth may serve this need; some will want to return to a form of

physical exercise or sport. Archery, boating, football, camping, hunting, flying, golf, scuba diving, swimming, sailing, basketball, table tennis, weight lifting, tennis, horseback riding, wheelchair racing, wheelchair dancing, and sit skiing have all been successfully pursued by people with SCI. Learning that even with a disability, satisfaction and camaraderie can be found in play with others is essential for SCI patients to realize the positive potential of their future life.

Patient and Family Education

A critical element in a patient's adjustment to disability is the patient's family. Initially, the concerns of the patient and family center on prognosis. Response to these concerns should respectfully acknowledge the uncertainty and difficulty the patient may have in accepting new limitations. Hope, even if based on improbable outcome, helps patients cope with the initial grief. Acceptance of the new reality generally comes with time. Patients and their families can be coached into accepting the necessity of learning alternate methods of mobility and self care even though they long for recovery to the preinjury state.

During rehabilitation the focus is to educate the patient, family, and caregivers on the nature of SCI, how to maximize health, and how to access 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 hyperreflexia;
- psychological adjustment to disability;
- attendant care management;
- sexual adjustment and fertility;
- vocational and educational options;
- avocational outlets such as wheelchair sports;
- finances; and
- housing.

Various educational formats can be used: a patient manual for self instruction and later reference, weekly class discussions, peer interaction, and one-on-one instruction provided by each member of the rehabilitation team. Intake and discharge panels with the rehabilitation team and the patient and his or her family further complement this instruction. The intake panel defines specific goals for the hospitalization and addresses specific questions related to prognosis and course of treatment. The discharge panel discusses plans for follow-up, equipment needs, and accessing community resources. For those who will be discharged partially dependent, family members and other caregivers need to be instructed in their roles. An on-site apartment for independent living allows patients and their caregivers to rehearse typical days that include shopping, meal preparation, transfers, toileting, and bathing with staff backup available as needed. Day and weekend passes are also scheduled once the patient is medically stable and sufficiently trained in self care and mobility. Community outings with the recreation therapist that include experience with public transportation, shopping malls, movies, grocery shopping, banking, and eating in restaurants allow patients to practice their recently acquired mobility and self care skills and to begin experiencing community reintegration.

Housing, Finances, and Community Reintegration

Social work can assist patients in understanding social security disability, veterans affairs (VA) benefits, separation from the military, Medicare/Medicaid benefits, accessible public housing, nursing homes, housing modifications, advocacy groups for the disabled, legal resources, and protections. In addition, occupational therapy can assist patients in understanding accessibility, including ramp inclines, door widths, and so forth.

HEALTH MAINTENANCE (YEARS POSTINJURY)

Lifetime annual evaluations are standard for discharged SCI patients, although initially the patient may need more frequent visits. Because of the risk of medical complications and functional decline, the annual assessment should be comprehensive. In addition to a general history and physical examination, assessments of urologic and neurologic, skin functional status, and equipment needs should be included. The physical health of the patient is only one aspect under review during this examination; the patient's psychosocial and vocational adjustments should also be assessed with additional ex-

pertise sought from other members of the rehabilitation team when appropriate.

Late mortality and morbidity due to SCI are higher in those patients with quadriplegia than paraplegia (1.4-fold higher) and higher in those with complete rather than incomplete SCI (1.5-fold higher).[6] At age 20, the late mortality rate of those with SCI is 8-fold that of the age-matched general population (7.2 vs 0.9 per 1,000); at age 70, the mortality rate on those with SCI is 1.5-fold that of others (75.1 vs 50.2 per 1,000).[6] Mortality is due to urologic causes in 24%, cardiovascular in 23% and pulmonary in 14%; however, mortality rate due to urologic causes has fallen dramatically from 43% of the deaths occurring in the 1940s and 1950s to 10% of the deaths occurring in the 1980s and 1990s.[6]

Preventing and Managing SCI Complications

During acute rehabilitation and thereafter, the SCI team must watch for medical complications associated with SCI. Many need early diagnosis and treatment to minimize long-term sequelae. In one clinical study, pressure sores and UTIs had annual incidences of 23% and 20%, respectively, and were the most common complications.[6] Some complications develop most often in the first several years postinjury (eg, hydronephrosis, spasticity, contractures, and suicides). Other complications demonstrate greater prevalence with increasing time since the SCI (eg, musculoskeletal problems). Still other complications are most closely associated with the patient's age (eg, cardiovascular complications, pneumonia, and renal stones). Common SCI complications are discussed below.

Pulmonary Complications

The inspiratory phase of breathing requires active muscle contraction and depends primarily on the diaphragm innervated at 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; the clavicular portion of the pectoralis major contributes to active expiration in quadriplegic individuals.[86] Impairment of these two pulmonary actions (inspiratory phase of breathing and forced expiration) cause most SCI pulmonary complications.[87]

Acute Respiratory Failure. The earliest pulmonary complication is acute respiratory failure. This condition develops most commonly in those with cervical cord injury at the C-5 level or higher and may appear immediately after the injury or develop over hours to several days as respiratory muscles fatigue. Close monitoring is needed to detect the muscle fatigue *before* a respiratory arrest, and emergent intubation is required; the fatigue manifests itself as tachypnea, decreased tidal volume, decreased inspiratory pressure, hypoxemia, and hypercapnia. A partial pressure of CO_2 above 40 mm Hg or vital capacity below 1,000 mL suggests the need for intubation and mechanical ventilation. Careful attention must be directed to aggressive pulmonary toilet and early detection and treatment of pneumonia.[88] The majority of these patients eventually wean from the ventilator over a period of days, to weeks, to months. Forced vital capacities (FVCs) typically improve over a period of months, presumably a combination of spontaneous neurologic recovery and strengthening of the respiratory muscles. After 1 to 2 weeks, if weaning is not imminent, a tracheostomy is performed; prolonged endotracheal intubation has been associated with airway complications such as subglottic stenosis. A tracheostomy tube also allows direct tracheal suctioning and bronchoscopy and communication through leak speech.

For intubated individuals, communication is a major issue. Initially, tongue clicking or exaggerated eye blinks can be used for yes or no signaling, but head nodding may not be possible because of spine instability or neck muscle paralysis with C-1 or brainstem level injuries. Communication can also be pursued through lip reading or a communication board. Eventually, as pulmonary status stabilizes, patients can use leak speech, where the tracheostomy cuff is deflated and large inspiratory volumes are delivered, enabling air to leak around the tracheostomy tube and upward through the larynx, allowing speech. Fenestrated tracheostomy tubes, with built-in windows to provide continuous air leak for speech, and talking tracheostomy tubes, which supply alternative sources of air, are available at more expense. Ventilator-dependent patients must always have with them a suction device for clearing secretions and an Ambu bag in the event of ventilator failure.

Other factors affecting inspiration are abdominal distention and posture.[89] The quadriplegic patient begins at a considerable disadvantage because the chest wall retracts during inspiration when the descent of the diaphragm creates a negative intrathoracic pressure. This intercostal retraction, which is normally prevented by intercostal muscles,

cancels a portion of the inspiratory effort. Further exacerbating the problem is the tendency of the atrophied abdominal wall muscles and abdominal organs to displace anteriorly, pulling the diaphragm downward when the patient is sitting. Diaphragm muscle contractions then begin in a shortened position and generate less inspiration. Ileus, acute gastritis, and fecal impaction can all cause abdominal distention to further compromise diaphragm descent. An elastic abdominal binder can help counteract this displacement when patients are sitting upright; it may also increase the loudness of speech.[90] Obstructive sleep apnea, more prevalent in SCI individuals than in the general population, may lead to disrupted sleep, daytime drowsiness, personality changes, and even cor pulmonale; quadriplegia, obesity, sleeping in the supine position, and sedative ingestion may be predisposing factors.[91,92]

Improved pulmonary function has been reported following breathing exercise programs, arm ergometry, and functional electrical stimulation.[86,93,94] Prevention programs (eg, smoking cessation, flu and pneumococcal vaccinations) should be established for all SCI patients, with a particular emphasis on quadriplegic individuals. Periodic pulmonary function tests are useful as part of a health maintenance program.

Atelectasis, Pneumonia, Aspiration, Impaired Cough. Pneumonia is the leading cause of death during the first several months following SCI.[57,95] 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 cough-elicited abdominal muscle contraction. Those with cervical cord injury have impaired inspiratory effort as well. Impaired cough and impaired inspiration predispose to atelectasis. Mucous hypersecretion and hyperviscosity, along with impaired cough, contribute to mucous plugging. All of these factors predispose to pneumonia. Those most vulnerable are patients with high cervical cord injuries. The site of pneumonia is most often the left lower lobe, where the sharper angulation of the left mainstem bronchus makes suctioning more difficult.

The following precautions promote clearing 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*) clap the chest wall and assist cough by manually compressing the abdomen in synchronization with the patient's cough (ie, quad cough), (*d*) institute intermittent positive pressure breathing with bronchodilator followed by assisted coughing, and (*e*) use a rotating bed (eg, Roto-Rest Traumabed, Kinetic Concepts, San Antonio, Texas).[70,96] Training in glossopharyngeal breathing (GPB) can improve assisted coughs and may allow time off a ventilator for some patients with rostral cervical cord injuries.[97]

Fever, increased purulent sputum, altered auscultation of the lungs, and chest radiographic changes suggest pneumonia. Treatment for pneumonia involves more frequent and more aggressive pulmonary hygiene and parenteral antibiotics. Thick secretions and consequent mucous plugs may require bronchoscopy.

An additional pulmonary problem is aspiration. Those with SCI at T-10 or above are less able to cough effectively and clear the airway if they aspirate. Precautions to minimize aspiration risk include (*a*) no oral intake and gastric decompression by nasogastric tube for those with gastroparesis or ileus, (*b*) upright or side-lying for eating, and (*c*) no 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 is a predisposing factor to aspiration, which should be considered. As a long-term precaution, family members and caregivers should be trained to perform the Heimlich maneuver for someone who is lying down or sitting.

Cardiovascular Complications

A variety of cardiac complications can compromise health for the spinal cord injured. Several etiologic factors contribute. With cervical or upper thoracic cord lesions, sympathetic outflow to the heart and blood vessels is disrupted; the heart is then influenced by unbalanced parasympathetic activity. Manifestations of deficient sympathetic outflow include bradyarrhythmia, asystole, other rhythm disturbances, postural hypotension, and autonomic hyperreflexia. Loss of tonic arteriolar vasoconstriction results in vascular pooling and a lower baseline blood pressure; expected blood pressure for a person with quadriplegia is 90/60 mm Hg. In addition, decreased lower extremity muscle activity leads to venous stasis and the compromise of venous return. A low level of high density lipoprotein (HDL) as a result of reduced physical exertion is a long-term cardiovascular risk factor.

Bradyarrhythmia, Sinus Arrest. Bradyarrhythmia leading to cardiac arrest is a serious, early complication, its incidence peaking at day 4 postinjury

in those with cervical cord injuries.[98] These injuries impair cardiac sympathetic outflow arising at T-1 to T-4, which accelerate the heart. The remaining innervation is parasympathetic; vagal input slows the heart rate. If deceleration is severe, cardiac arrest may result. Tracheal suctioning or hypoxemia can trigger such a bradyarrhythmia, presumably via a vagovagal reflex. Preventive measures include preoxygenation and atropine prior to suctioning to inhibit the cholinergic receptors of the vagal efferents to the heart. An atropine dose of 0.6 mg to 1.0 mg given intravenously 15 minutes prior to suctioning may prevent these vagal-mediated bradycardic episodes. Oral ephedrine sulfate or continuous intravenous infusion of very low-dose isoproterenol have also been suggested; quadriplegic subjects are hypersensitive to these adrenergic agents.[98] Occasionally patients may require transvenous cardiac pacing.

Succinylcholine, a depolarizing paralytic medication, is often given as an adjunct to general anesthesia, but in SCI patients this can result in profound hyperkalemia with cardiac arrest because the neuromuscular junction is hypersensitive to cholinergic agents.[47] Succinylcholine is absolutely contraindicated in SCI patients.

Deep Venous Thrombosis, Pulmonary Embolus. Another common and potentially fatal complication of acute SCI is DVT with consequent pulmonary emboli.[88,99] Deep venous thrombosis usually develops within the first 3 months postinjury, with a peak at 10 to 14 days. It is uncommon after 3 months. 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 (5,000 units used twice daily), and applying venous sequential compression pumps to the lower extremities.[100,101] Twice daily thigh and calf circumference measurements aid early detection of DVT. A difference of as little as 1 cm is suggestive; care for accuracy and consistency of measuring points is essential. If a sudden increase or asymmetry in lower extremity circumference develops or if the patient experiences unexplained low-grade fever or sudden onset dyspnea or chest pain, then DVT must be suspected and prompt action taken.[102] Clinicians must always have a high index of suspicion. Terpie found that fewer than 50% of patients with documented venous thrombosis had the characteristic signs of fever, redness, and swelling.[103] Diagnosis can be made with duplex scanning alone or with a simple Doppler examination and confirmatory contrast venogram. Surveillance via Doppler, duplex, or impedance plethysmography scanning may need to be done at least twice weekly for the first 2 weeks. The differential diagnosis includes lower extremity fracture, lower extremity hemorrhage, HO, dependent edema, cellulitis, and postphlebitic edema. If confirmed, DVT is treated with continuous intravenous heparin, venous compression stockings and Ace wraps, and bed rest without lower extremity range-of-motion exercises for 1 week. Heparin is discontinued after oral Coumadin becomes therapeutic; Coumadin is then continued for 3 months. If anticoagulation therapy is not possible in a given patient or if pulmonary emboli occur despite anticoagulation, then an inferior vena cava filter should be considered. Quad coughing should not be performed in patients with vena caval filters.

Autonomic Hyperreflexia. Autonomic hyperreflexia, also often called autonomic dysreflexia, is a delayed sequela of high thoracic or cervical cord injury.[104] This unique manifestation of SCI above the T-7 level presents after skeletal muscle hyperreflexia returns. 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). The trigger is bladder or bowel overdistension or some other noxious stimulation. Symptoms are pounding headache, flushing, and diaphoresis. These symptoms result from high and potentially life-threatening systolic hypertension, sometimes reaching 250 mm Hg and requiring the following emergency measures: (*a*) elevate the head of the bed to promote dependent pooling of blood and to lower the risk of intracerebral bleed; (*b*) eliminate viscus distension or noxious stimulus quickly by catheterizing the bladder or untwisting a condom catheter; an indwelling catheter may need to be replaced; (*c*) if hypertension persists, give sublingual nifidepine 10 mg and repeat every ten minutes as needed to a total of 30 mg.[104,105] Other antihypertensive medications are also used including phenoxybenzamine, prazosin, mecamylamine, nitrates, and clonidine.[105] Nitropaste may be useful for hyperreflexia that lasts hours or days. Once the blood pressure has been lowered, then an attempt must be made to identify and eliminate or minimize other noxious stimuli contributing to autonomic hyperreflexia. Bowel impaction should be removed with digital disimpaction, using premedication with

nifidepine and xylocaine jelly as a rectal anesthetic to avoid further aggravating the condition. Peptic ulcer, cholecystitis, appendicitis, bowel obstruction, rectal fissure, ureteral stone, UTI, infected ingrown toe nail, fracture, or labor and delivery are other common stimuli that should be considered.[104] In SCI patients with renal disease, autonomic hyperreflexia must be distinguished from renal vascular hypertension.

Severity varies among patients; in some, blood pressure elevations cause excessive diaphoresis while in others, hypertension can lead to retinal or intracerebral hemorrhage, seizures, or death.[106,107] Patients are encouraged to carry an Autonomic Hyperreflexia 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 orthostatic hypotension (OH). With OH, patients complain of dizziness, lightheadedness, and fainting. In general, OH is most severe in patients with higher lesions (typically cervical and high thoracic SCI) and in patients with longer bed rest. Several factors may contribute: impaired vasoconstriction and consequent pooling of blood in the lower limbs and splanchnic bed, decreased sympathetic drive to the heart, and relative volume depletion. Most at risk are quadriplegic patients; they typically run blood pressures of 90/60 mm Hg. These patients often become orthostatic when first sitting in the morning. To minimize these episodes, such patients can be fitted with an abdominal binder, venous compression stockings, and Ace wraps; liberal salt and fluid intake and elevating leg rests may also help avert orthostasis. The patients may require ephedrine sulfate (25–50 mg one-half hour before sitting) or mineralocorticoid as well. In acute SCI, OH improves with repeated sitting trials, which promote fluid retention; quadriplegics are known to run high renin and aldosterone levels, a presumed mechanism for compensating for orthostasis. Late worsening of OH could suggest development of posttraumatic syringomyelia or a silent myocardial infarction.

Silent Myocardial Infarction and Coronary Artery Disease. 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 HDL levels after SCI.[108,109] Another cardiac complication for the cervical cord injured is a silent heart attack. 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 T-1 to T-4. In such cases, the clinical manifestations may be subtle and nonspecific, perhaps increased orthostasis, dyspnea on exertion, orthopnea, or increased pedal edema.

Gastrointestinal Complications

Altered sympathetic outflow due to thoracic or cervical SCI may also alter GI function and cause delayed gastric emptying, increased gastric acid secretion, and disturbed colonic myoelectric activity.[110] SCI can alter motility of the descending and sigmoid colon and anal sphincter by impairing parasympathetic outflow from sacral segments. Acute abdomen is often diagnosed late with a resulting high morbidity and mortality in patients with SCI above T-10 having impaired somatic sensation; such patients may report poorly localized pain at a later stage of acute abdomen.[111,112] 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, which may be limited to tachycardia, increased spasticity, and autonomic hyperreflexia.

Ileus. Ileus or loss of GI motility is noted in 63% of SCI patients for several days and occasionally for weeks after acute SCI.[113] To minimize the possibility of vomiting, aspiration, and compromised respiration, acutely injured SCI patients often should have a nasogastric tube placed for decompression. The patient may report nausea and loss of appetite. Abdominal or gastric distention and absent bowel sounds may require managing the patient NPO (nothing by mouth) with intravenous fluids and a nasogastric tube for suctioning. Metoclopramide is reportedly of benefit for prolonged gastroparesis or ileus. [114]

Stress Gastritis or Ulcer. Stress gastritis or ulcer is another early complication, reported in 5% to 20% of acute SCI patients.[115,116] The most vulnerable are the aged and those with injuries above T-5, where spinal shock leaves the parasympathetic stimulation to the stomach unopposed, resulting in a consequent increased in acid secretions. Additional sources of irritation to the stomach lining are corticosteroids, which decrease mucosal resistance; gastric distention; hypotension; and sepsis. Additional risk factors are mechanical ventilation and heparinization. Prevention involves use of an H_2 blocker and antacids to maintain a pH greater than 4, continuous suction via nasogastric tube to prevent gastric overdistension, and monitoring gastric secre-

tions for occult blood. Should prophylaxis prove inadequate, upper GI endoscopy or angiography (if the cervical spine is unstable) will confirm GI bleeding. If active bleeding is detected, then blood pressure must be maintained with fluids and transfusions and coagulation defects corrected. Laser coagulation and intraarterial vasopressin may be of benefit.

Obstipation, Fecal Impaction, Pseudo-Obstruction, Megacolon. Decreased colonic motility, increased colonic compliance, and perhaps anal sphincter spasticity in those with chronic SCI may result in obstipation and fecal impaction.[117] Key elements of a bowel program include (*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 muciloid, (*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 as necessary. 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.[118]

Patients reporting poor results with a bowel 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, the patient may require periodic hyperosmotic laxatives (milk of magnesia, lactulose, sorbitol). If this is 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 autonomic hyperreflexia, 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 an optimal solution.[119] The mean time spent on bowel care in 20 SCI patients with chronic bowel problems, dropped from 99 minutes to 18 minutes per day after a colostomy.

Cholelithiasis and Colon Carcinoma. The prevalence of gallstones and colon carcinoma is elevated in spinal cord injured patients.[118] The increase is 3-fold in cholelithiasis and 4-fold with colon carcinoma. In quadriplegic or high thoracic SCI, cholelithiasis can be associated with few symptoms, delayed diagnosis, and autonomic hyperreflexia. Treatment with chenodeoxycholic acid or cholecystectomy should be considered. Slow colon motility could contribute to the increased incidence of colon carcinoma. Delay in diagnosis of colorectal disease is common, sometimes leading to life-threatening abdominal emergencies.[120] Regular screening with fecal occult blood tests, flexible proctosigmoidoscopy, barium enema (beware of barium retention), and colonoscopy should be considered.

Endrocrinologic Complications

Two endocrinologic complications that are brought on or exacerbated by SCI are hypercalcemia and insulin-dependent diabetes.

Hypercalcemia. Hypercalcemia is uncommon but occurs most often in male adolescent quadriplegics in the first months after injury. 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, it can progress to lethargy and coma. Laboratory studies reveal hypercalcemia, hypercalciuria, and low creatinine clearance. Treatment includes intravenous saline with furosemide and remobilization. Additional measures may be needed to inhibit osteo-clast-mediated bone resorption: bisphosphonates (etidronate, pamidronate), calcitonin, mithramycin, glucocorticoids, and gallium nitrate.[121,122]

Insulin-Dependent Diabetes Mellitus. Spinal cord injury has profound metabolic consequences resulting in disorders of carbohydrate and lipid metabolism.[108] Abnormal glucose tolerance and hyperinsulinemia are common following SCI. SCI complicates the management of diabetes mellitus in two ways: (1) increased insulin resistance caused by the decreased muscle mass and obesity of the SCI patient, and (2) absence of adrenergic hypoglycemic symptoms (diaphoresis and tachycardia) in quadriplegics. For the second complication, cultivating an increased awareness of the neuroglycopenic symptoms (drowsiness, impaired mental status, seizure) is vital. Treatment for hypoglycemia with subcutaneous or intramuscular glucagon or intravenous dextrose should be anticipated. Treatment for hyperglycemia is standard: caloric restriction, exercise, oral hypoglycemic agents, and insulin therapy.

Skin Issues

Pressure Sores. Pressure sores are a major bane of SCI care. Incidence has been estimated as high as 42% to 85% in some centers.[123] Prevention is vital to controlling SCI health care costs and optimizing patient functioning. The cost of treating pressure sores in the United States is estimated to be $3 to $5 billion per year.[124,125] Causes are unrelieved direct pressure, skin shearing, and prolonged exposure to moisture. Activities and conditions that predispose to the development of pressure sores are prolonged sitting, severe spasticity, contractures, edema, anemia, poor nutrition, bruises and skin damage resulting from falls or scrapes, worn or inadequate cushions, urinary or fecal incontinence, and smoking.[126,127] Avoiding these conditions is a matter of education, awareness, behavioral change, and commitment—factors heavily dependent on the psychological well-being of the individual and the supportiveness and competency of the medical staff and caregivers. Biochemical changes have also been noted in skin following SCI and can contribute to increased susceptibility to pressure ulcers.[128–130]

Standard preventive measures for the acutely injured SCI patient include avoiding pressure by using a foam mattress and turning every 2 hours. Some conditions that put acute SCI patients at risk are anesthetic skin; immobility from paralysis; hypotension; spine immobilization measures; poor nutrition and subsequent weight loss; fever; urinary and fecal incontinence; flexor spasm; and sometimes altered mental status such as coma, depression, or chronic alcohol use. From 20% to 40% of patients develop pressure sores during the first month following SCI.[131]

Alternatives to standard measures are specialized beds such as the Roto-Rest bed and Stryker or Foster turning frames. These are expensive but of particular use when patients are in cervical traction. Low-air-loss beds (eg, Clinitronbed, Support Systems International, Inc., Charleston, South Carolina, or Kinnairbed, Kinetic Concepts, San Antonio, Texas) are also expensive but are useful for patients with multiple active pressure sores that result in insufficient lying surfaces and for those who are recovering from myocutaneous flap surgery. Less frequent turning is usually possible with use of these special beds.

No matter the predisposing factors, the cause of pressure sores is prolonged pressure or shear or both, sufficient to cause underlying skin and muscle necrosis from ischemia. Pressures as small as 20 cm H_2O occlude capillary blood flow. This low level of tolerance means it is impossible to distribute the weight of the torso over buttocks and posterior thighs without blocking capillary flow. Thus, for all but children (with less body weight), it is theoretically impossible to design a cushion that will eliminate ischemia. However, optimal distribution of weight over sitting or lying surfaces can prevent the development of necrosis as the inevitable capillary ischemia is regularly relieved by turning or by pressure releases. When lying down, patients require turning every 2 to 4 hours. When sitting, a pressure release for 15 seconds every 15 minutes is usually recommended. The greater frequency of pressure releases in sitting derives from the distribution of body weight over a smaller surface.

Pressures are highest over bony prominences; consequently, pressure sores arise most commonly over the ischial tuberosities and calcanei while sitting, and the greater trochanters, occiput, sacrum, scapulae, malleoli, and calcanei while lying. Other secondary factors that contribute to pressure sores are impaired circulation resulting from peripheral vascular disease as in diabetes mellitus, poor nutrition with hypoproteinemia and anemia, and obesity.

Relief of pressure while sitting in a wheelchair is achieved with cushions that distribute weight optimally. A well-fitted wheelchair, with a solid rather than sling seat, a high-density foam cushion, and pressure releases for 15 seconds every 15 minutes are the standard precautions. Specialized cushions are available for those at high risk: the Jay cushion (Jay Medical Ltd., Boulder, Colorado), made of contoured foam with an overlying gel pad that redistributes weight from the ischial tuberosities to the posterior thighs; and the Roho cushion, constructed of multiple inflated bulbs that distribute weight over a wide area by varying the position of the upper body.[132]

In addition, patients or caregivers must be taught to check the skin twice daily for any nonfading redness or skin breakdown. If noted, patients must alter their positions and routines to keep the affected areas free of pressure until the redness disappears or the skin heals. Patients are taught to avoid burns by avoiding cigarette ash falling on anesthetic skin, to avoid hot water bottle use or placing hot plates or mugs on their thighs, to sit away from fires or radiators, and, to check bath water with a thermometer (less than 98°F or 36.5°C). They also learn to avoid applying condom catheters or leg straps too tightly (Figure 5-11), to keep nails trimmed, to check shoes for nails or other sharp objects before donning, and to avoid tight shoes. Males must routinely

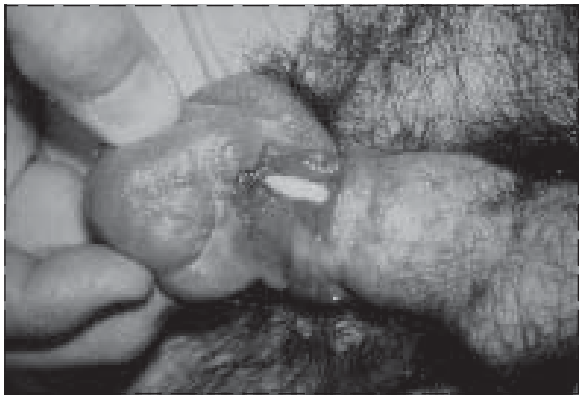

Fig. 5-11. Penile and urethral sore from tight condom catheter. This individual applied his condom catheter too tightly using elastic tape, resulting in a deep penile sore, which extended into the urethra. Such complications can be avoided only through patient or attendant education.

pull the scrotum forward after transferring to sitting positions, to avoid sitting on insensate testicles.

Pressure sores usually develop in 30% of acute SCI patients because adequate preventive measures have not been instituted and followed. In defining and classifying these sores, the following scale has evolved:

- Grade 1: nonfading redness, an incipient sore.
- Grade 2: partial thickness skin loss, involving epidermis or dermis, but not exposing subcutaneous tissue.
- Grade 3: full thickness skin loss exposing or involving subcutaneous tissue but not involving muscle.
- Grade 4: exposed muscle or fascia, no bone observed.
- Grade 5: bone exposed.
- Grade 6: joint space involved.

Superficial pressure sores can heal with conservative treatment; deeper wounds may require surgical intervention. The choice to use surgery (generally with ulcers of grades 4-6) is made when the sore is too large to heal with conservative measures over a period of 3 to 4 months, when a deep infection must be drained, or when a bony deformity must be corrected. For superficial wounds without bacterial infection, special hydrocolloid dressings are available.[133] Bedside debridement with scalpel, enzymatic ointments, or wet-to-dry dressings are used to clean the wound of necrotic tissue. Deep or undermined sores are packed with wet-to-dry sterile gauze to clean the wound thoroughly. When clean, granulated tissue is noted, wet-to-wet gauze or hydrocolloid dressings can be used to maintain a moist wound environment, which encourages epithelization.[120] Minimizing anemia and promoting nutrition are other measures that enhance healing. Recent research suggests that growth factors (eg, platelet-derived growth factors) increase the rate of healing. High voltage pulsed current has also been evaluated.[5] These interventions, along with a detailed analysis and correction or mitigation of the factors contributing to the development of the sores, are essential.

If the choice is surgery, myocutaneous flaps are the standard treatment. A myocutaneous flap frees up skin and underlying muscle while preserving the blood supply, and repositions it over the sore. The most common examples are gluteus maximus rotation flaps to cover sacral or ischial sores; hamstring V-Y advancement flaps to cover ischial sores; and tensor fascia lata, vastus lateralis, or rectus abdominus flaps to cover trochanteric sores. Skin grafts can be used over granulated tissue. The possible need for future grafts should be considered when tissue sites are chosen. Postoperative care requires rigorous attention to ensure success. Following surgery, patients are best served by management on a Clinitron bed (Support Systems International, Inc., Charleston, South Carolina) for a period of 3 weeks to minimize pressure on the flap. During this time, avoiding any tension on flap incisions is essential. For patients with spasms, this may mean increased spasmolytic medication and positioning to minimize hip flexor spasms. After 3 weeks, if the flap is healing well, patients progress to a regular hospital bed with a foam mattress, and lower extremity range-of-motion with direct observation of the flap suture lines. At a later date, patients are gradually remobilized in a fitted wheelchair with appropriate cushion and reinforcement of the need to perform regular pressure releases and twice daily skin checks.

Cellulitis, Osteomyelitis. Most cellulitis and osteomyelitis connected with SCI originate in pressure sores through the direct spread of infection.[134] With cellulitis (a spreading local infection in the skin), the common presenting symptoms are swelling, redness, local warmth, and fever. In osteomyelitis, the symptoms are 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 (Figure 5-12). Bone scans are of limited use because

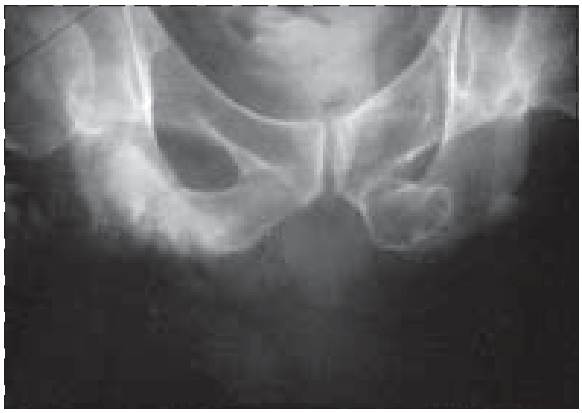

Fig. 5-12. Osteomyelitis underlying an ischial decubitus. This paraplegic individual with a history of paranoid schizophrenia developed recurrent ischial decubiti despite patient education to perform twice daily skin checks and pressure releases every 15 minutes and despite being fitted with a contoured foam/gel cushion. Note the irregular, sclerotic ischial bone indicating osteomyelitis below the decubitus.

they are usually positive below a pressure sore, whether the underlying bone is infected or not. Definitive diagnosis of osteomyelitis is possible through bone biopsy and histologic confirmation, with more reliable results obtained through a needle biopsy, which avoids the bacteria in the decubitus tract.

Cellulitis is treated with oral or parenteral antibiotics usually for 10 to 14 days. Osteomyelitis requires 6 weeks or more of antibiotic administration and may also require local bone debridement and a myocutaneous flap to cover an associated pressure sore.

Musculoskeletal Issues

The musculoskeletal effects of SCI are many and include HO, muscle contracture, spinal deformity, osteoporosis predisposing to fractures, and tendon or muscle rupture related to use of upper limbs for mobility.

Heterotopic Ossification. HO, also known as myositis ossificans and paraosteoarthropathy, is abnormal bone formation in the soft tissue around large joints below the level of the cord injury, most commonly the hips.[135] The reported incidence is 16% to 53% after SCI.[136,137] The cellular basis for HO is unknown, but presumably it represents a metaplastic transformation of mesenchymal or endothelial cells to osteoblasts. The onset of HO is usually 1 to 4 months following SCI, but it may develop late after surgery or local trauma. Its presence alone is often not significant; it becomes significant in 10% to 20% of the SCI population if it compromises joint range-of-motion, thus impairing function and predisposing to pressure sores; slows venous return leading to increased peripheral edema and DVT; or leads to local peripheral nerve damage.[103,138] Heterotopic ossification is reportedly more common in males, younger patients (< 30 years old), more complete injuries, and those with spasticity.[139,140]

Symptoms are swelling, warmth, redness, and fever. Without this inflammatory phase, a noted reduction in joint motion may be the first warning. Early confirmation is made in the presence of high level serum alkaline phosphatase and with a positive technetium 99 bone scan. Ultimately, a radiograph will reveal fluffy densities in the soft tissues around the joint (Figure 5-13). Cellulitis, hematoma, tumor, and DVT should be excluded.

Initial treatment, which ideally begins early when the ossification process is most amenable to reversal, usually involves maintenance of joint range-of-motion and oral etidronate disodium. Indomethacin prevents HO formation after total hip replacement and may be useful after SCI as well,

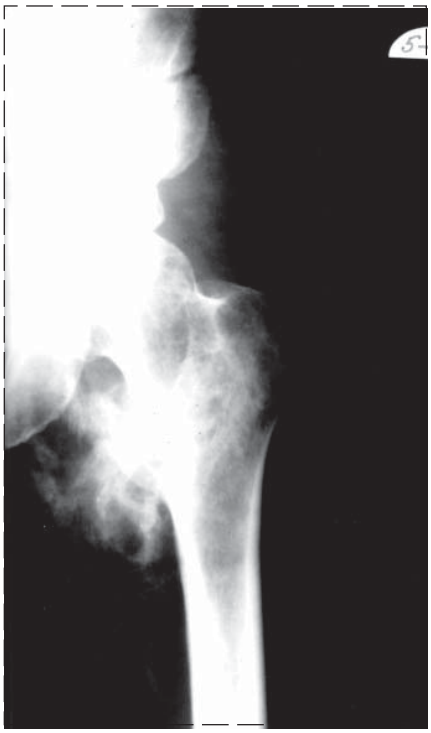

Fig. 5-13. Heterotopic ossification. Fluffy, amorphous radiograph densities around the hip indicate heterotopic ossification.

although this needs to be evaluated. Coumadin may also reduce the incidence of HO.[141]

For established HO, forceful manipulation or surgical resection can be considered once a bone scan confirms the HO is mature. These procedures often reinitiate the HO formation.[119,135] Associated local radiation therapy and continued treatment with disodium etidronate or indomethacin may minimize this recurrence.[137]

Myotendinous Contracture, Joint Capsule Tightness. In addition to HO, myotendinous contracture and joint capsule tightness can limit joint motion. Finger flexion deformities compromise skin care; ankle plantar flexion contractures compromise transfers, standing, and walking; and hip flexion contractures can prevent prone lying. Hip and knee extension contractures interfere with wheelchair sitting. Even when mild, hip and knee flexion contractures increase the energy cost of standing and walking, and contractures of finger flexors commonly impair grasp and pinch. Prevention requires daily joint range-of-motion exercises, provided by a caregiver if the patient is unable to do them without help. Early contracture or capsule tightness may be reversed with ultrasound diathermy and static stretch; ultrasound must be used cautiously in those with impaired or absent sensation. Established severe contracture or capsule tightness requires surgical release.

Osteoporosis, Fractures. The paralysis of SCI results in loss of bone density, for which there is no known treatment. This osteoporosis predisposes to fracture, particularly of the femur (Figure 5-14). In non-weight-bearing limbs, such fractures are usually managed with soft, well-padded splints that can be removed twice daily to ensure intactness of the skin.

Spine Instability, Charcot's Spine, Spine Deformity. A late consequence of spinal fracture may be progressive spinal deformity, arising from unrecognized ligamentous injury or other spinal instability.[142] This instability and associated excessive spinal movements can result in severe degenerative changes of the vertebral body, which must be differentiated from a malignant or infectious process. These degenerative changes are referred to as neurogenic spinal arthropathy or Charcot's spine. A needle or open biopsy of the degenerated vertebral body may be required to rule out malignancy or osteomyelitis. This condition can lead to pain and a progressive gibbus of the thoracic spine with a pressure sore affecting overlying skin, which can in turn become infected and lead to vertebral osteomyelitis and psoas abscess. Progressive spinal deformity may also lead to ascending neurologic loss, particularly when it affects the cervical spine. Typically, surgical stabilization is required, although external bracing may be most appropriate if age or medical complications preclude surgery.

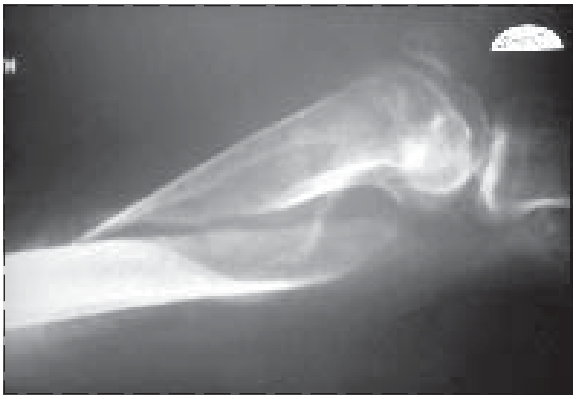

Fig. 5-14. Femur fracture in osteoporotic bone. This male with long-standing paraplegia flexed his trunk forward with his knees extended in long-sitting while tying his shoes; this resulted in a distal femur fracture. The osteoporosis that develops in long bones of paralyzed individuals due to the absence of normal compressive and tensile forces of weight bearing and muscle contraction increases the risk of fracture with minor falls or moderate stresses.

Weight-Bearing Shoulder. The assault to the musculoskeletal system is not limited to muscle, bone, and joint below the level of the injury, it also impacts the musculoskeletal components above the SCI level, which must compensate for the paralysis. Prolonged use of the upper extremities for weight-bearing, such as in push-up transfers, predisposes to degenerative changes in the shoulder.[83,143–145] Impingement syndrome, rotator cuff tears, and muscle rupture may all be evident.[146] Prevention and treatment involve controlling obesity, minimizing weight-bearing as in sliding board or hydraulic lift transfers, avoiding use of an overhead trapeze for transfers, substitution of forward or side-lean pressure releases for push-up pressure releases, nonsteroidal antiinflammatory medications, strengthening exercises, power wheelchairs, and surgery such as acromioplasty with rotator cuff tendon repair.[144]

Neurologic Issues

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

Spasticity and Other Hypertonus. Spasticity develops in those with upper motoneuron damage and spared reflex pathways; it is one type of hypertonus, an increased resistance to passive joint movement. Spasticity appears when segmental and descending inhibition is lost and new synaptic connections by the spared reflex pathways are acquired. Other forms of hypertonus associated with SCI include hyperactive cutaneomuscular reflexes, tonic stretch reflexes, segmental myoclonus, and dystonia.

Spasticity results from 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. True spasticity then is triggered by fast movements or muscle vibration, stimuli that elicit discharge from 1A afferents. 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 (Figure 5-15) or as the patient performs a push-up pressure release.[147] These extensor spasms often interfere during transfers, although some patients use them to aid standing.

Hyperactive cutaneomuscular reflexes are another form of hypertonus, which manifests as flexor spasms and a Babinski response. These are common indicators of ongoing noxious stimulation as with a chronic decubitus ulcer (Figure 5-16), or recurrent hip subluxation (Figure 5-17).[148] Such flexor spasms can interfere with prone or supine positioning in bed, with bed mobility, transfers, and walking; they can also interfere with sleep and contribute to decubiti.

Fig. 5-16. Flexor spasm associated with a trochanteric decubitus. This male with spastic paraparesis demonstrates hip and knee flexor spasms in association with a large, grade 4 trochanteric decubitus. The decubitus is a noxious stimulus that increases flexor tone, and in turn, the flexor hypertonus limits bed rest to left or right side lying, aggravating the trochanteric pressure sore; supine and prone lying are difficult to achieve with such severe hip and knee flexor spasms.

Less common than either of the above, but more disabling are hyperactive tonic stretch reflexes. These appear to result from length-dependent discharge of Group II stretch reflex afferents of the muscle spindle. They can be distinguished from

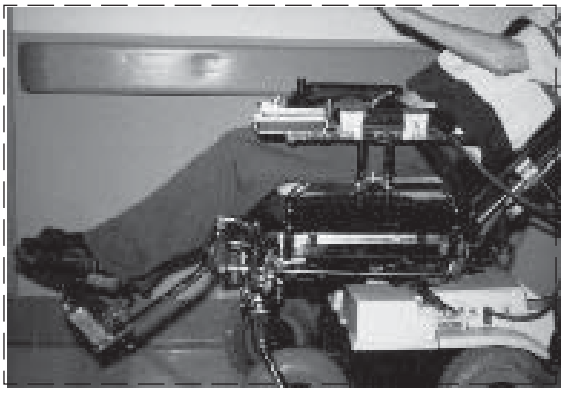

Fig. 5-15. Extensor spasms. These are a common manifestation of spasticity after SCI. This quadriplegic male demonstrates an extensor spasm, including hip and knee extension; hip adduction; ankle plantarflexion; abdominal muscle contraction; and elbow, wrist, and finger extension, elicited by the hip flexor stretch during rapid recline in his power wheelchair.

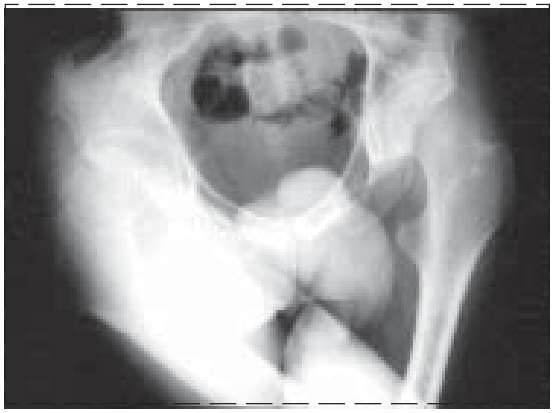

Fig. 5-17. Hip flexor/adductor spasms and hip subluxation. Severe hip flexor and adductor spasms in this paraparetic male have resulted in hip subluxation. In turn, the hip subluxation acts as a noxious stimulus, which further aggravates hip flexor and adductor spasms.

myotendinous contracture or joint capsule tightness by simultaneously recording electromyographic activity from the muscle during passive stretch or by performing an anesthetic nerve block.

Spasticity and other hypertonus require treatment depending on the degree to which they interfere with function, hinder the caregivers' tasks, or contribute to medical complications as with skin break down, falls, interrupted sleep, or pain.[149–151] Treatment benefits must always be weighed against the possible beneficial effects of spasticity; for example, positioning extremities or aiding standing transfers and walking (Figure 5-18).

Once spasticity is determined to be more detrimental than beneficial and any nociceptive sources such as UTI, renal stone, or fecal impaction have been ruled out, then 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. Often this is not enough and oral medications—baclofen, diazepam, dantrolene, or clonidine—must be tried alone or in combination. If these prove insufficient, as they often do with moderate or severe spasms, and the patient has some preserved voluntary movement, the physician should consider a reversible treatment such as intrathecal baclofen. Severe spasms can sometimes be suppressed only by irreversible treatments; among them are phenol motor-point or nerve blocks, tenotomy or tendon transfers, lumbar cord myelotomy, or complete or selective posterior rhizotomy.

Fig. 5-18. Functional extensor spasms. This male with T-4 paraparesis can elicit lower extremity extensor spasms by positioning his upper body behind the axis of rotation of his hips. This stretch of his hip flexors elicits and maintains extensor spasms, which he uses functionally for standing transfers. His only voluntary movement in his lower extremities is trace movement in his toes.

Pain. Severe, disabling pain is present in 5% to 49% of chronic SCI patients.[25,152] Surveys of SCI outpatients have revealed that chronic pain, rather than paralysis, often prevents employment. Commonly this pain originates centrally, within the spinal cord. This central pain presents with any combination of a burning, tingling, or lancinating quality that is unaffected by neck movements or posture and is thought to represent spontaneous discharge of neurons in the ascending pain pathways. Generally it is not present acutely, but develops weeks to months following SCI. The pain associated with incomplete cervical cord injury tends to be continuous and burning.

A patient with cauda equina injury experiences a series of stabbing pains that radiate down one or both lower limbs every few seconds to minutes; these shocklike pains may be continuously present or reappear 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. Occasionally it responds to desensitization or transcutaneous electrical nerve stimulation. Other treatments for these three types of central pain include nonnarcotic analgesics (acetaminophen, acetylsalicylic acid, ibuprofen); anticonvulsants (phenytoin, carbamazepine, clonazepam); and tricyclic antidepressants (amitriptyline, doxepin). Anticonvulsants may inhibit spontaneous discharge of central neurons that are mediating the pain sensations. Tricyclic antidepressants may reduce monoamine reuptake and thereby reduce pain transmission. Side effects of these medications can be significant. Carbamazepine has resulted in aplastic anemia, agranulocytosis, and thrombocytopenia. Other adverse effects of anticonvulsants include confusion, ataxia, nystagmus, and hepatic abnormalities. Tricyclic antidepressant side effects include sedation, urine retention, other anticholinergic effects, and OH.

Chronic narcotic use (eg, methadone, oxycodone, etc.) should be administered only in a

highly structured setting in which the patient's compliance and psychological status can be carefully monitored.[95] Nerve blocks, surgically sectioning the ascending pain pathways, and even cordotomy or cordectomy do not permanently relieve these types of pain.[153] Recently, epidural electrical stimulation and focal lesions of the dorsal root entry zone (DREZ) have been advocated.[154,155] The indications and long term success of these procedures is not known.

It is essential to distinguish these kinds of central dysesthetic SCI pains from pain arising due to mechanical causes such as persisting disk herniation impinging a root or the cord, an enlarging posttraumatic syrinx, or degenerative arthritis. These latter types of pain are generally aggravated by spinal movements or postural changes and thus may be clinically distinguishable from central pain. Treatment involves addressing the underlying cause of mechanical pain, if possible.

Posttraumatic Syringomyelia. Posttraumatic syringomyelia is an uncommon but clinically subtle and potentially severely disabling complication.[64,156] The prevalence of clinically significant posttraumatic syringomyelia has been estimated at 4.5% for quadriplegic subjects and 1.7% for paraplegics. Posttraumatic syringomyelia results from an enlarging fluid-filled cyst (syrinx) at the level of the cord injury, which leads to progressive spinal cord damage and is also referred to as ascending cystic myelopathy. One proposed pathophysiological explanation of posttraumatic syringomyelia is that the initial SCI leads to hemorrhagic cysts at the site of injury, which liquefy into fluid-filled cysts. The cysts enlarge with Valsalva's maneuver or repeated spine movements, which traction the cord and force CSF from the subarachnoid space by way of perivascular spaces (Virchow-Robin spaces) into the cysts. Such enlarging cysts may develop within months after an SCI but more typically develop over many years. They may extend rostrally or caudally from the cord injury and can extend the length of the spinal cord up to the brainstem.

The earliest symptoms and signs may be subtle (Figure 5-19). 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. Its appearance often indicates that the condition is advanced and may not be reversible, although the pain may be remediated. Magnetic resonance imaging (MRI) is definitive for diagnosing posttraumatic syringomyelia and has revealed that 60% of all SCI patients have small cysts. Serial clinical examinations, repeat MRI, and serial electrodiagnostic studies (specifically, F-wave latencies and central motor conduction times by motor evoked potentials) are the available methods for determining if the cysts are progressive.[69,157]

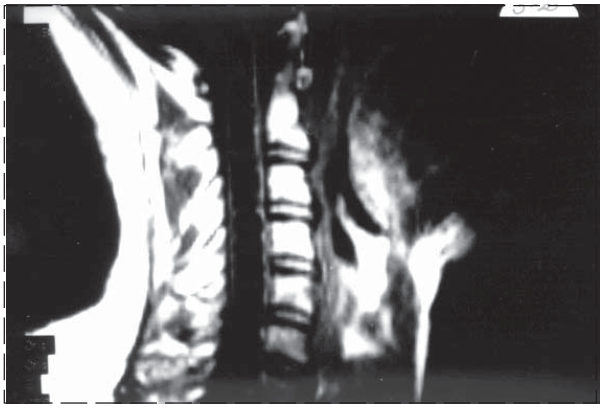

Fig. 5-19. Posttraumatic syringomyelia. This male with T-6 paraplegia at eight years postSCI noted loss of reflex bladder emptying and intermittent numbness in his fingers. Physical exam revealed pin prick sensory loss at T-4 on the left and at T-6 on the right and asymmetric tendon reflexes in the upper extremity. An MRI revealed a large syrinx extending rostrally and caudally from the level of the midthoracic cord injury; rostrally the syrinx extended to the foramen magnum. Despite this large syrinx, he denied any weakness or functional loss. This case illustrates the often subtle manifestations of posttraumatic syringomyelia as it initially presents.

Treatment may include minimizing Valsalva maneuvers and spine movements and surgical decompression with syringosubarachnoid, syringopleural, or syringoperitoneal shunts to remove the CSF and prevent its reaccumulation. For those with complete cord injury, cordectomy is an alternative.

Peripheral Nerve Entrapment. Peripheral nerve entrapment results from the high demands on the upper extremities of quadriplegics and paraplegics and is common after SCI.[158] Predisposing factors include (*a*) increased use of the upper extremities for transfers and wheelchair propulsion, resulting in an increased incidence of carpal tunnel syndrome; (*b*) absent pain sensation, particularly in posttraumatic syringomyelia, resulting in ulnar nerve entrapment; and (*c*) proximal compression of motor neurons and motor axons, resulting in double crush syndrome. Radial nerve entrapment, as in Saturday night palsy and thoracic outlet syndrome, also occurs with increased frequency. Preventive

measures include use of wheelchair gloves 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

Urosepsis and renal failure cause significant morbidity and mortality for those with SCI, although this has markedly improved since the 1940s. Standard care must provide annual urologic evaluations to eliminate conditions predisposing to urosepsis and renal failure. Understanding how the level of the cord lesion relates to bladder function is useful.[159,160] Upper motor neuron dysfunction is generally associated with spinal fractures at T-12 and above (ie, cervical and thoracic fractures). For patients with these conditions, voluntary control from the brain is disrupted, yet reflex activity recovers after spinal shock dissipates. Lower motoneuron dysfunction generally occurs when the bony injury is at L-1 and below. These individuals do not regain reflex contraction; the bladder remains flaccid and overfills. Mixed dysfunctions occur with partial injuries to the lumbar spine such as combined conus-cauda equina injuries, with combinations of upper and lower motor neuron damage. Whatever the type of dysfunction, the goals of treatment are to imitate as closely as possible normal lower urinary tract functions with low-pressure storage, continence with adequate emptying when voiding, infection control, and protection of the ureters and kidneys.[138,161]

SCI patients are at increased risk for renal compromise.[162] Acutely, rhabdomyolysis and shock can lead to acute renal failure. Sepsis can lead to ischemic renal injury and aminoglycoside antibiotics can cause toxic acute tubular necrosis. Radiocontrast dye for intravenous urography can cause acute renal insufficiency. Various antibiotics, including penicillin derivatives, methicillin, cephalosporins and sulfas can cause acute interstitial nephritis, while nonsteroidal antiinflammatory medications can impair renal hemodynamics and yield azotemia. Factors predisposing to chronic renal failure in SCI patients include high pressure voiding with reflux nephropathy, nephrolithiasis from urea-splitting bacteria and hypercalciuria, chronic pyelonephritis, and amyloidosis from chronic decubiti and osteomyelitis. Renal function should be regularly followed and impairment detected early and treated.

Urinary Retention, Incontinence. 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. Patients are then managed with sterile intermittent catheterization (IC) every 4 to 6 hours to keep maximal bladder distension to less than 500 mL.

After several months, reflex detrusor contractions develop; this can be anticipated in those with preserved bulbocavernosus and anal reflexes. When spontaneous reflex bladder emptying develops, anticholinergics may be needed and IC may need to be continued; or in males, the frequency of IC is decreased, postvoid residuals monitored, and urinary drainage collected in a condom catheter and leg bag.[163] Recently, some[164] have advocated intentional bladder overdistension for women in conjunction with IC to avoid the need for anticholinergics. Postvoid residuals should be less than 100 mL to minimize concentrations of bacteria in the urine. The less common lower motoneuron bladder (where the S-2, S-3, S-4 reflex arc is interrupted) can be managed with IC and fluid restriction or condom catheter drainage and Valsalva's maneuver. High bladder pressures can be associated with Credé over the bladder and can lead to vesicoureteral reflux and hydronephrosis.[165]

Detrusor-Sphincter Dyssynergia, Vesicoureteral Reflux, Hydronephrosis. Spinal cord injury patients with upper motoneuron lesions rostral to and sparing the conus (S-2, -3, -4) usually develop reflex bladder emptying several months following SCI. Many of these patients will also develop detrusor-sphincter dyssynergia, in which the reflex detrusor contraction is opposed by a simultaneous external urethral sphincter contraction.[166–168] The result is high bladder pressures that lead to detrusor muscle hypertrophy and a further increase in bladder pressure. This increased pressure may, in turn, result in vesicoureteral reflux, hydroureter, hydronephrosis, and renal failure. This process is often clinically silent.[169] Urodynamics (ie, cystometrogram with simultaneous external urethral sphincter electromyography and fluoroscopy) is used to detect the presence of high pressure bladder emptying or vesicoureteral reflux. Yearly upper tract imaging (intravenous pyelogram, or renal ultrasound, or nucleide renal scan) and creatinine clearance should be obtained to further monitor renal status.

Treatment of high pressure voiding may include one of the following approaches: anticholinergic medication (oxybutynin, propantheline) and every-

6-hour catheterization, indwelling Foley catheter, or transurethral sphincterotomy.[170,171] Detrusor-sphincter dyssynergia is not the only cause of high pressure voiding. Outflow obstruction and increased bladder pressure can also be caused by internal sphincter (bladder neck) spasms, prostate enlargement, urethral stricture, or a too tightly applied condom catheter. Internal sphincter spasm may improve with α-adrenergic antagonist medication (phenoxybenzamine, prazosin) or bladder neck incision. A promising new treatment for external sphincter spasm or prostate enlargement is transurethral placement of a permanent stent, a small mesh coil that holds the urethra open.

Urinary Tract Infection

Bacteriuria ($> 10^5$ organisms per mL) is a nearly inevitable consequence of neurogenic bladder.[161,172] If asymptomatic, this bacteriuria is not treated. Symptoms of UTI prompting antibiotic treatment may include fever, shaking chills, leukocytosis, hematuria, pyuria (white blood cell count > 10 per high power field), and unexplained increase in spasticity. Preventing UTI may involve ensuring adequate bladder drainage with a low postvoid residual, perineal hygiene, cleaning drainage tube and bag, clean catheterization (washing hands and catheter with soap and water for those on IC), and eliminating urinary tract stones.[173] Those with recurrent UTI may benefit from bacterial suppression with methenamine mandelate or hippurate, trimethoprim-sulfamethoxazole, or nitrofurantoin, though resistant bacteriuria invariably develops.[161] High fevers and shaking chills suggest urosepsis, which requires broad antibiotic coverage (including pseudomonas coverage), placement of an indwelling catheter to assure bladder drainage, and a renal ultrasound to rule out upper tract obstruction. Failure of response to antibiotic within 48 to 72 hours or septic shock may prompt an abdominal CT scan to rule out perinephric abscess.

Bladder and Renal Calculi. Bladder stones may cause bladder spasms and are often removed by transurethral cystolithectomy. Renal stones are most commonly infectious struvite stones caused by vesicoureteral reflux and urea-splitting organisms. Another risk factor for stone formation is hypercalciuria in response to paralysis and urinary stasis. If stones are large enough to obstruct the ureter, then SCI patients must be monitored closely since upper tract obstruction may be relatively silent until urosepsis develops. Such upper tract stones may be removed by extracorporeal shock wave lithotripsy (ESWL) or percutaneous procedures.

Bladder Carcinoma. Spinal cord injury patients with long term indwelling catheters have an increased risk of bladder carcinoma.[174] One approach to early diagnosis and treatment of such bladder cancers is to perform yearly cystoscopy and biopsy of the bladder for those who have had indwelling catheters for more than 10 years.

Sexual Function and Fertility. Sexuality is the disclosure of maleness or femaleness in an individual. SCI may alter but never negates that expression.[175] The potential exists to make the expression richer and more thoughtful and ingenious. The recently injured may need to be reassured of this. In particular, young males may find their definition too narrowly focused on genitals. To broaden this, they need to be helped in making a compassionate and honest reevaluation of their physical selves and provided with, if they are willing to explore alternatives, detailed information about aids and options. They should also be helped to realize that in sexual as well as in other human relationships, qualities such as kindness, humor, and intelligence are powerful and even erotic. A thorny issue for many is their increased physical dependency and how in this altered situation they retain a sense of power, control, and responsibility to those around them.

If such patients are willing to explore the topic, this is some of the specific information they should have. They should know that sexual desires for both males and females are psychological and hormonal and not affected by nerve damage. In addition, although the level of sensation below the site of injury may be diminished or absent, males, particularly those with neurologic lesions above S-2, -3, -4 or incomplete lower lesions, are sometimes able to achieve and maintain erections (the overall rate is 50% for all categories of injury). External appliances, intracavernosal injection (prostaglandin E, papaverine), or an implanted penile prosthesis provide additional options.[176]

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. Autonomic hyperreflexia is a small risk with these procedures in men with injuries at or above T-6.

Fertility in men diminishes to less than 5% with clinically complete injuries. Semen quality declines rapidly after SCI, possibly from recurrent UTIs and

increased temperature of the testicles. Nonetheless, artificial insemination with semen obtained by electroejaculation has resulted in some pregnancies and some healthy livebirths.[177]

For females, loss of sensation and lack of vaginal lubrication are the primary physical changes. Pleasurable sexual experience for both sexes may be realized through focusing on skin, lips, and other areas where sensation is intact. Use of a vibrator and lubricants can also be helpful. The fear of bladder incontinence is often a concern, but provision of towels, some humor and acceptance, bladder emptying by catheterization, and decreased fluid intake prior to intercourse should resolve this. Information about manual stimulation, oral–genital sex, positioning, and reducing or using spasticity can be discussed if the patient is willing to explore these topics.

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 these patients, making foam and condoms better birth control choices. Undetected labor pains leading to an unsupervised birth, a prolonged labor because of compromised ability to push, and autonomic hyperreflexia in those with SCI above T-7 are additional risks of pregnancy and childbirth in this population.

Because of the complex issues and the extraordinary demands on the patient for reworking many aspects of his or her life after SCI, sexual and marital counseling are standard components of rehabilitation. Respect for individual values and sensitivity are essential in handling this emotionally loaded, highly personal topic.

Psychological Issues

Assessment and support are the two major psychological services in SCI. Assessment is an evaluation of the individual's potential to learn, to think, and to interact with his environment and other human beings. It includes a description of impairments that may be remediated and those that are unlikely to be altered. Some of the tools of assessment are standardized tests that measure psychosocial function and intelligence. Those most commonly used include the Minnesota Multiphasic Personality Inventory,[178] the Strong-Campbell Interest Inventory[178] for vocational interest testing, and intelligence tests like the Wechsler Adult Intelligence Scale[178] and the Wide Range Achievement Test.[178] Other tests can be used to assess neuropsychological functions in patients where head injury is a complicating factor, such as the Halstead-Reitan Neuropsychological Battery[178] or the Luria-Nebraska Neuropsychological Battery.[178] What should emerge from these assessments is a clear picture of the individual's potential for rehabilitation and appropriate guidelines for educational and vocational pursuits.

Support, the other major psychological service to SCI patients, should extend to both patients and family members. The support should be grounded in knowledge of the patient's history, disposition, values, limitations, and potential. SCI psychologists assist individuals in making new lives when part of the old has been shattered. Other members of the rehabilitation team also contribute to the patient's psychosocial adjustment to SCI, particularly the social worker, vocational counselor, and recreation therapist. Most important, no matter what the level or completeness of injury, is the patient's willingness to engage with life. In a population that is mostly male, young, and action oriented, this is a Herculean remaking that requires the patient to find necessary energy reserves to make a new contract with life that will include a radical shift in self-image and paradigm. What makes this possible is hope—hope for satisfaction in meaningful relationships and genuine contributions. Helping the patient to find this hope is the rehabilitation team's most crucial assignment. Quality of life gradually improves in most SCI patients so that by 4 years postinjury, it is 87% of normal on a visual analogue scale.[179] Quality of life is slightly worse for those with quadriplegia as compared with paraplegia and slightly worse for those with complete as compared to incomplete cord injuries.[180]

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

Associated Head Injury. A significant percentage of SCI patients incur closed head injury.[181–183] Resulting agitation, impulsiveness, impaired judgment, and impaired new learning may all impact rehabilitation.

Premorbid Personality. Premorbid factors affect rehabilitation outcomes. 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; thus, among those with less than 12 years of education only 38% have worked at some time after SCI, compared to 93% of those with 16 or more years of education.[62] These premorbid factors should be considered during acute rehabilitation.

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 was the known cause of death in 6.3% of 9,135 SCI patients, 4.9 times higher than a matched general population.[184] 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, physical dependence on spouse as a caregiver with subsequent burnout, and impaired sexual function.

ORGANIZATION OF A SPINAL CORD INJURY UNIT

Acute trauma facilities, such as those in the U.S. military, do not usually have comprehensive SCI units, but rather, they have orthopedists, neurosurgeons, physiatrists, PTs and OTs, psychologists, social workers, and skilled nurses who can decompress the spinal cord, establish spine stability, prevent complications, and begin the initial process of rehabilitation. As soon as they are medically stable, patients are transferred to a specialized SCI rehabilitation facility; the U.S. military has a longstanding agreement with the U.S. Department of Veterans Affairs to transfer patients to one of the 23 VA SCI centers.

Physician support for a comprehensive SCI facility includes physiatrists, urologists, orthopedic surgeons, neurosurgeons, internists, and plastic surgeons. Other essential health professionals are rehabilitation nurses, PTs and OTs, psychologists, social workers, vocational counselors, orthotists, recreation therapists, respiratory therapists, and dietitians.

Specialized facilities for an SCI unit are uro-dynamics with videofluoroscopy, myelography with CT, MRI, 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 on their own with spouse or attendant support, is particularly useful when the patient's ability to return to the community is uncertain.

A specialized SCI unit can anticipate and minimize complications and enhance functional outcomes compared to a medical, surgical, or general rehabilitation ward, which results in shorter hospitalizations and less economic cost. The features of an SCI unit that allow these gains are specialized knowledge, unique team relationships, and specialized equipment and facilities. Such specialized SCI centers allow clinical and applied basic research to further improve care for SCI individuals.

CONCLUSION

Spinal cord injury was fatal prior to World War II. Most SCI patients now survive, return home completely or partially independent, and live near-normal life spans with satisfactory quality of life. For most SCI patients, these favorable outcomes can be expected if the emergency, acute, and rehabilitation interventions during the first 6 months postinjury are appropriate. Spinal cord injury leads to multiorgan dysfunction. Strength, sensation, blood pressure control, bladder and bowel emptying, and sexual function are often impaired. Life threatening or disabling conditions affecting different organ systems must be prevented or diagnosed and treated early, such as pressure sores, deep venous thrombosis with pulmonary embolism, autonomic hyperreflexia, orthostatic hypotension, heterotopic ossification, posttraumatic syringomyelia, depression, and suicide. In addition, SCI patients must resume function to the degree possible, given the level and completeness of SCI. Training and adaptive equipment is required for independent mobility and independent self-care. Guidance and instruction is required for psychological, social, financial, vocational, and avocational adjustment to the disability.

An SCI rehabilitation unit includes an interdisciplinary clinical staff and specialized equipment and protocols to prevent and treat SCI complications and to train and instruct SCI patients functionally. These specialized facilities achieve greater functional outcome with fewer complications. U.S. military facilities refer spinal cord injured soldiers to the 23 SCI Services of the Department of Veterans Affairs for comprehensive rehabilitation.

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