Rehabilitation of Persons With Spinal Cord Injuries

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Overview

Traumatic spinal cord injury (SCI) is perhaps the most devastating orthopedic injury, and with prolonged survival being the rule, rehabilitation of these injuries has an increasingly important role. The primary goals of rehabilitation are prevention of secondary complications, maximization of physical functioning, and reintegration into the community.

Rehabilitation following SCI is most effectively undertaken with a multidisciplinary, team-based approach, as follows[1, 2, 3, 4, 5] :

- Physical therapists typically focus on lower extremity function and on difficulties with mobility
- Occupational therapists address upper extremity dysfunction and difficulties in activities of daily living
- Rehabilitation nurses are concerned with the issues of bowel and bladder dysfunction and the management of pressure ulcers
- Psychologists deal with the emotional and behavioral concerns of the newly injured patient and with any potential cognitive dysfunction
- Speech-language pathologists address with issues of communication and swallowing
- Case managers and social workers are the primary interface between the rehabilitation team, the patient and his or her family, and the payer source

The rehabilitation team functions under the direction of a physiatrist (ie, a physician who specializes in physical medicine and rehabilitation) or a physician with a subspecialty certification in spinal cord medicine. While each team member has primary responsibilities, any member of a properly functioning interdisciplinary team can contribute to the resolution of any problem.

SCIRehab project

The Spinal Cord Injury Rehabilitation (SCIRehab) project, a 5-year project led by the Rocky Mountain Regional Spinal Injury System (RMRSIS) at Craig Hospital, used practice-based evidence (PBE) research to identify the rehabilitation interventions most strongly associated with positive outcomes.

The aim of the SCIRehab project was to provide detailed information on treatments delivered by rehabilitation disciplines and to contribute to outcomes-based guidelines for clinical decision-making. The SCIRehab project included 1376 patients with acute SCIs, with outcome data being abstracted from medical records (clinical outcomes data) at six SCI rehabilitation facilities and obtained from patient interviews at 6 and 12 months after injury.[6, 7, 8, 9, 10, 11, 12, 13, 14]

The final phase (phase 3) of the SCIRehab Project was published in November 2012.[15, 16, 17, 18, 19, 20, 21, 22, 23, 24]

For further information on this topic, see Spinal Cord Injury.

Common Medical Problems

Persons with spinal cord injury (SCI) are at particular risk for certain types of morbidity, with some differences between problems in the acute and chronic phases. Prevention and treatment of these problems is considered in later sections.

Morbidity during the acute rehabilitation phase (which follows the initial acute hospitalization) includes pressure ulceration, which occurs in about 25% of patients treated in Model Systems centers in the United States. The most common location of pressure ulceration is over the sacrum.

Atelectasis and/or pneumonia occurs in 13% of cases.

Deep vein thrombosis (DVT) is found in 10% of patients acutely, and pulmonary embolus (PE) occurs in 3% of cases. This incidence of DVT and PE is lower than that noted historically, probably owing to improved vigilance and prophylaxis.

Autonomic dysreflexia occurs in about 8% of cases initially and in 29% of those with complete tetraplegia.

Urinary tract infection commonly is acute.

A study of persons with chronic SCI monitored in England for many years revealed an annual incidence of 23% for pressure ulceration and 20% for urinary tract infection. Pressure ulcers also were noted to be the most common morbidity in a study of patients monitored in US Model Systems, occurring in 15% of patients during the first year post injury and increasing in subsequent years. Autonomic dysreflexia, urinary tract problems, and pneumonia or atelectasis also were noted to be common.

Thromboembolic Disease

Thromboembolic disease is common following SCI. Approximately 40% of patients without prophylaxis develop deep venous thrombosis (DVT) during the acute phase. The risk of death from pulmonary embolism (PE) during the first year following SCI is more than 200 times that for the general population. DVT most commonly occurs in the weeks following SCI, with a much lower risk in persons with chronic injury.

The increased risk of thromboembolism is likely due to venous stasis and hypercoagulability. Classic symptoms of DVT, such as calf tenderness, may be lacking, owing to sensory loss. Symptoms of PE, such as shortness of breath, may be wrongly attributed to concurrent problems such as atelectasis. DVT can present as fever of unknown origin, and PE can result in sudden death.

The high incidence and unreliable presentation of DVT suggest that screening studies should be considered. For example, daily physical examination by a physician and a nurse can be supplemented by weekly venous imaging by ultrasound for the first several weeks, when the incidence is highest.

A prophylactic strategy can address venous stasis and hypercoagulability.[25] Pneumatic compression devices can be used for the first 2 weeks, followed by use of a compression hose. Unfractionated heparin (5000 U SC every 12 hours) or a low-molecular-weight heparin (30 mg SC every 12 hours), such as enoxaparin, can be administered for 2-3 months following injury.

Placement of a caval filter can be considered in patients with multiple risk factors (eg, lower limb fracture, history of DVT, cancer, heart failure, obesity, age >70 years) or in those with a high complete cord lesion.

In a review of DVT occurring after SCI, Teasell et al found that there was strong evidence supporting low-molecular-weight heparin to reduce venous thrombosis, and a higher adjusted dose of unfractionated heparin was found to be more effective than 5000 units administered every 12 hours, though bleeding complications were more common. Nonpharmacologic treatments were also reviewed, but limited evidence was found to support such treatments.[25]

Autonomic Dysfunction

High thoracic and cervical spinal cord injury (SCI) can cause loss of supraspinal control of sympathetic activity with dysregulation of functions normally impacted by sympathetic mechanisms. Baseline sympathetic activity following SCI is low, though there may be hyperresponsiveness of peripheral sympathetic receptors, perhaps as an adaptive response.

Clinical problems result from inappropriately low or high sympathetic responses, the former during the acute phase and the latter in the subacute and chronic phases. Problems are most common in those with injuries to level T6 and above, as such levels isolate the sympathetic outflow to the splanchnic vascular bed.

Resting blood pressure is low with higher cord lesions, which is asymptomatic. Orthostatic blood pressure changes can cause weakness, light-headedness, and fainting. Management includes gradual mobilization, liberal sodium intake, use of compression hose, and an abdominal binder.[26, 27]

Fludrocortisone acetate (0.1 mg PO qd) can expand intravascular volume and therefore is helpful. Midodrine (titrated upward from a dose of 5 mg PO bid/tid qd) may be helpful. Midodrine can cause supine hypertension and presumably may exacerbate any tendency toward autonomic hyperreflexia (AH).

Bradycardia is common soon after injury and usually resolves after several weeks. Tracheal suctioning can exacerbate bradycardia and can cause asystole, perhaps through a reflex increase in vagal output. Symptomatic bradycardia can be treated with intravenous atropine or by a transvenous, external, or implanted pacemaker.

Autonomic hyperreflexia

Autonomic hyperreflexia (AH), or autonomic dysreflexia, is an acute, potentially lethal complication particular to patients with spinal injuries above T7. The classic presentation is a profound headache in the presence of elevated blood pressure. The pathophysiology appears to involve an unmodulated sympathetic response to a noxious stimulus below the level of the lesion. The 2 most common inciting stimuli are bladder and bowel distention, respectively; the former commonly results from an obstructed urinary catheter.

Adrenoceptor hypersensitivity below the level of the lesion contributes and may be an essential factor, accounting for the observation that AH does not occur acutely following injury. AH occurs only later, when such hypersensitivity has had a chance to develop. The incidence of AH is increased within the first 6 months following SCI. Patients with complete motor injuries (ie, American Spinal Injury Association [ASIA] A or ASIA B) are at higher risk of AH than patients with incomplete injuries.

Headache may be due to intracranial arterial dilatation, which occurs as part of the effort by the parasympathetic system to adjust for the hypertension. Management includes placing the patient in a sitting position (which decreases intracranial pressure), checking for an inciting stimulus (eg, distended bladder), and, if necessary, administering antihypertensive medications such as topical nitroglycerin.

Following an initial episode of AH, the patient may be sensitized and prone to further episodes of AH until autonomic equilibrium is established. For recurrent bouts of AH, prophylaxis with alpha-blocking agents may be warranted.

Neuropathic Pain

Neuropathic (spinal) pain following spinal cord injury (SCI) is perceived at or below the level of injury. Descriptors often involve temperature (eg, hot, burning, sunburned, frostbitten) and electricity (eg, an electric shock).

Pain can exist apart from any external stimulus (rest pain) or can result from a stimulus that would, under normal conditions, not cause any pain (allodynia), or pain can be excessive in response to a painful stimulus (hyperalgesia). These symptoms may result from changes in central neuronal function, including increased spontaneous activity and reduced thresholds of response.

Evaluation of neuropathic pain following SCI must include consideration of the possibility of a treatable underlying condition, such as an unstable spine or posttraumatic cystic myelopathy. A change in an established pattern of neuropathic pain may be induced by an unrelated disease process (eg, renal stone).

Patients are relieved to be informed that their pain need not reflect any active problem and need not cause them to curtail their activities. Indeed, an increased level of activity may decrease suffering. Medicinal treatment includes the use of anticonvulsants and antidepressants.

Anticonvulsants may be particularly useful in cases of lancinating electrical pain. Gabapentin (initial dose of 100 mg PO tid, gradually titrated upward) typically is used, with precautions for sedation.

Tricyclic antidepressants may be useful for more constant diffuse pain. Amitriptyline (initial dose of 10 mg PO qhs, gradually titrated upward) is one of several agents. Precautions must be taken for its anticholinergic effects. Patients should be informed that relief with these agents may not be immediate, as the initial dose may require modification, and in any case the effect of the medication may not be apparent for days or weeks.

Data from the CONECSI (COping with NEuropathiC Spinal cord Injury pain) trial noted decreases in pain intensity, decreases in pain-related disability, reduced anxiety, and increases in participation in activities for the patients who

participated in a multidisciplinary cognitive-behavioral therapy program. These data suggest that such programs may be beneficial for those with chronic neuropathic SCI pain.[28]

Neurogenic Bladder Dysfunction

Spinal cord injury (SCI) typically is followed by a period of bladder flaccidity. With suprasacral injury, reflexes eventually return. However, these reflexes may be unable to cause efficient voiding because of the tendency of reflex sphincter activity to directly oppose reflex detrusor contraction. This opposition occurs because of the isolation of the urinary tract apparatus from higher centers, which normally coordinate reflex activity. This problem is called detrusor-sphincter dyssynergy.

Acute bladder management is by use of an indwelling catheter, as the bladder is likely to be flaccid. Intermittent catheterization is not practical during the initial phase, when urine output cannot be controlled and is likely to be high because of the administration of intravenous fluids.

Long-term management has several objectives. Patients require a drainage method that is socially acceptable and that avoids wetting the skin. Bladder emptying should be complete, avoiding high residual volumes. Storage and drainage of urine should occur under low intravesical pressure, as pressures over 40 cm water have been found to correlate with renal deterioration. Chronic use of an indwelling catheter is avoided when possible, as it can cause various soft-tissue problems, renal problems, and, possibly, bladder cancer.

Selection of a bladder drainage method ideally is made following urodynamic evaluation. Clean intermittent catheterization is a method available to those with good hand function or to skilled attendants. The patient is instructed to limit fluid intake, and catheterization is performed every 4-6 hours. Reflex bladder contractions, which could cause high storage pressure and incontinence between catheterizations, can be inhibited by agents such as oxybutynin (5 mg PO tid) or tolterodine (2 mg PO bid).

Reflex voiding into a condom catheter is an option available to men with reflex bladder contractions. Problems can include urinary retention or high intravesical voiding pressure due to detrusor-sphincter dyssynergy. Voiding pressure sometimes can be decreased by alpha-blocking agents such as terazosin (initial dose 1 mg PO hs, titrated upward) or tamsulosin (0.4 mg PO qd). More definitive control of voiding pressure can be obtained by sphincter-defeating surgery, such as sphincterotomy or placement of a urethral stent.

A woman with high tetraplegia (and lack of skilled attendants) will often elect to use a long-term indwelling catheter. This frequently leads to soft-tissue problems or to renal problems and necessitates eventual intervention (eg, ureteral diversion). Bladder management by electrical stimulation has become available.

In the past, renal disease was a frequent cause of death for those with chronic SCI; however, it now is unusual for this to happen. However, significant problems such as urinary tract calculi and hydronephrosis still occur. Thoughtful planning of bladder management can avoid this.

Annual surveillance of the urinary tract may detect subclinical problems and allow modification of the bladder regimen before significant complications occur. A testing regimen that evaluates the anatomy and function of the upper and lower tracts includes a renal ultrasound, renal nuclear scan, cystogram, and urodynamic examination.

Neurogenic Bowel Management

Neurogenic colonic dysfunction is a particularly distressing and limiting impairment for a substantial proportion of those with spinal cord injury (SCI). Lower motor neuron dysfunction, as with cauda equina and conus medullaris injury, causes constipation with slow colonic transport and incontinence due to a flaccid sphincter mechanism.

Upper motor neuron dysfunction also causes constipation with slow colonic transit and stool retention because of spasticity of the sphincter apparatus. However, with upper motor neuron injury, reflexes allowing defecation may remain functional and can be exploited in establishing a bowel program. The bowel program is a regimen, repeated on a daily or every-other-day basis, that can include diet, specified fluid intake, oral medication, medication per rectum, timing, and positioning. The goals of the program are continence and convenience.

The following steps are involved in establishment of a bowel program:

- Evaluation
- Preparation of the patient
- Trials of a specific bowel program
- Adjustment of the program

Evaluation includes obtaining patient history to determine any preinjury problems or patterns. Neurologic assessment, with examination of the bulbocavernosus and anocutaneous reflexes, can suggest the presence of upper or lower motor neuron bowel dysfunction.

Patients presenting with problems, including diarrhea, often are impacted. This can be suggested by physical examination and can be confirmed by radiographs. Patients may be taking medications (eg, antibiotics) that can have unintended effects upon the bowel, such as diarrhea. Evaluation may include testing of a stool specimen for *Clostridium difficile* toxin.

Preparation of the patient includes education about the anticipated program. Complicating problems, such as impaction and *C difficile* infection, must be treated before the program can succeed.

The specific program may include several measures. A typical problem is stool that is too hard because of the prolonged colonic transport time, which leads to drying of the stool.

Intervention includes maintenance of adequate intake of fluid and fiber, with fiber acting as a sponge to hold moisture within the stool. Docusate sodium (100 mg PO bid) can increase the ease with which water enters the stool. Patients with lower motor neuron dysfunction may experience greater continence with stool that is firmer than would be optimal for patients with upper motor neuron dysfunction.

A second problem is prolonged colonic transit time. Intervention includes maintenance of adequate stool bulk, which stimulates contractions of the colon. Fiber is helpful with this. A bowel stimulant (eg, 2 senna tablets PO qd) can be effective. These tablets typically are taken 8 hours before planned bowel evacuation. As these measures decrease bowel transit time, stool consistency may become softer.

A third problem is incontinence. The goal is to establish a set time for daily bowel evacuation, ideally after a meal to take advantage of any gastrocolic reflex that may be present.

Specific evacuation strategies may differ for upper and lower motor neuron problems. With upper motor neuron injury, defecation can be triggered with application of an irritant to the anorectal area, such as stimulation with a gloved finger or application of a bisacodyl enema or suppository. With lower motor neuron bowel dysfunction, evacuation may be by use of the Valsalva maneuver and digital removal. In either case, emptying is facilitated by a seated position on a commode, as opposed to side-lying in bed.

Adjustment of the bowel routine over time is commonly needed, usually with a good eventual result. For those with persistent difficulty, use of a pulsed-irrigation enhanced evacuation device can be used, and colostomy can be considered. In the future, bowel control may be achieved (as with bladder control) by electrical stimulation.

Heterotopic Bone Formation

Heterotopic ossification (HO) is the formation of new bone in soft-tissue planes surrounding a joint. HO can occur subsequent to various types of injuries, including spinal cord injury (SCI), in which case it most commonly involves the hips. Presentation can include some combination of generalized or localized lower extremity swelling, loss of hip range of motion (ROM), fever, and elevated alkaline phosphatase level.

HO may occur subclinically and may be noted incidentally on radiographs. Laboratory examination includes serum alkaline phosphatase, radiography, and bone scan. Bone scan may be positive before radiographic changes are noted.

The main clinical problem (apart from distinguishing it from other problems, such as deep venous thrombosis, which can present similarly) is the loss of ROM that can occur. Loss of hip ROM may complicate bed and chair positioning and can make dressing and bathing difficult. ROM exercises can be used in an attempt to limit eventual loss of joint range, despite concern over possibly exacerbating the underlying process of ossification.

Measures to possibly limit the eventual amount of bone mass formed include use of etidronate, nonsteroidal anti-inflammatory drugs (NSAIDs), and irradiation. Etidronate (20 mg/kg/day PO for 2 wk, followed by 10 mg/kg/day for at least 10 wk) can be given. An intravenous preparation of etidronate also is available for acute use. Indomethacin (25 mg PO tid) also can be given. Low-dose irradiation also can be applied.

Severe loss of ROM can be treated surgically. The possibility of recurrence may be limited by use of medications and irradiation, as described above.

Pressure Ulceration

Pressure ulceration is among the most common complications of spinal cord injury (SCI), along with urinary tract infections. Both of these conditions have an annual incidence of nearly 25% in people with chronic SCI. Pressure ulceration can be the most limiting sequelae of SCI and can confine an otherwise independent individual to bedrest.

Pressure to soft tissue that exceeds capillary pressure is the principal cause of skin breakdown. Shear, which refers here to prolonged displacement of soft tissue relative to the underlying bone, can distort interposed blood vessels and also can lead to tissue breakdown. Shear can occur over the sacral area when a person sits at an angle in bed, with the bed surface fixing the skin at one point and gravity causing descent of the underlying sacrum. Whether SCI makes tissue more sensitive to pressure is uncertain.

The location provides information about the cause. Ischial ulcers typically are due to sitting for prolonged periods without adequate pressure relief. Trochanteric ulcers are commonly due to excessive side-lying. Sacral ulcers, if high, may be due to supine lying. Lower ulcers in the intergluteal area may be related to sitting up at an angle.

Evaluation includes an assessment of the ulcer’s depth. Destruction of tissue often is more extensive beneath the surface, and breakdown indeed may begin here rather than at the skin.

Prophylaxis involves limiting the amount of pressure and the time over which pressure is applied. Wheelchair and bed cushions that limit pressure in any one area by distributing it evenly over the available body surface are available. Weight-shifting while in a wheelchair and turning in bed reduce the time of exposure to pressure. Sophisticated bed surfaces use baffles, which are alternately inflated and deflated, avoiding exposure that is prolonged enough to damage tissue. These surfaces are expensive, though not as expensive as treating a pressure ulcer.

Treatment of an established ulcer involves limiting or eliminating pressure to the area. This effectively confines a person with an ischial ulcer to bedrest. Local care includes removal of necrotic tissue by sharp debridement and by topical enzymatic debriding agents. Cleansing is accomplished with normal saline solution. Topical antibiotics are used only for foul wounds.

Dressing selection depends in part upon the amount of drainage present. Gauze can wick away excessive wetness. However, a relatively dry wound may benefit from dampened gauze or from the use of an occlusive or semiocclusive dressing, because a moist wound bed fosters healing more so than a dry environment.

Little healing can be expected in the absence of proper nutrition, including adequate provision of calories, protein, vitamin C, and zinc. Smoking slows healing.[29]

Deep ulcers can be treated surgically with debridement and repair by myocutaneous flap. Surgery is best deferred until nutritional status is adequate. Postsurgical care is prolonged and crucial.

Spasticity

Spasticity is a velocity-dependent increase in muscle tone and occurs commonly following spinal cord injury (SCI) and other types of upper motor neuron injury.[30] Spasticity causes resistance to passive motion of the limbs, exaggerated deep tendon reflexes, clonus, and involuntary cocontraction of muscle groups. Spasticity occurs following complete and incomplete cord injuries. SCI usually is immediately followed by a period of flaccidity, with spasticity developing over subsequent weeks.

Spasticity has desirable and undesirable effects. It can be used to assist with mobility, especially by those with incomplete injuries. It can improve circulation and may be useful for decreasing the risk of deep venous thrombosis and osteoporosis. On the other hand, spasticity can interfere with positioning, mobility, and hygiene, and spasms can be painful. A decision to intervene must take into consideration both the positive and the negative aspects of a patient's spasticity.

Intervention can occur in stages. The bedrock of treatment is the elimination of exacerbating factors and regular

muscle stretching. Less invasive methods typically are employed before the more invasive methods. Interventions to reduce spasticity, with the less invasive interventions listed first, are as follows:

- Prevention and treatment of noxious stimuli (eg, pressure ulcer, urinary tract infection, urinary tract stone, ingrown toenail)
- Regular muscle stretching and joint range of motion
- Oral medication (see Table 1, below)
- Botulinum toxin injection (useful for treatment of problems caused by specific muscle groups)
- Intrathecal baclofen delivered by an implanted pump (an involved but effective, nondestructive treatment)
- Peripheral procedures, including neurolysis and contracture release
- Central ablative procedures, such as rhizotomy and myelotomy

Several medications are available, which, if necessary, can be used in concert with the interventions listed above. Table 1 provides information regarding pharmacologic agents for the treatment of spasticity.

Table 1. Oral Pharmacologic Agents for the Treatment of Spasticity (Open Table in a new window)

Drug	Daily Dosage Range	Common Adverse Effects
Baclofen	5-200 mg (in divided doses)	Hypotonia Sedation/confusion Withdrawal syndrome
Tizanidine	2-36 mg (in divided doses)	Fatigue Dry mouth Sedation Elevated LFTs*
Clonidine†	0.2-0.6 mg (in divided doses)	Orthostatic hypotension
Diazepam	5-40 mg (in divided doses)	Sedation Cognitive effects Tolerance Dependence
Gabapentin	200-3200 mg (in divided doses)	Sedation/fatigue Ataxia Dizziness
Dantrolene	25-400 mg (in divided doses)	Weakness

		Elevated LFTs

*LFTs: Liver function tests.

† Transdermal delivery available.

Functional Rehabilitation

The neurologic examination within the first few days following SCI can predict neurologic recovery with great accuracy. Patients can be classified based on the American Spinal Injury Association [ASIA] impairment scale, as follows:

- A – No motor or sensory function below the level of the lesion
- B – No motor function below the level of the lesion but sensory function that continues into the sacral segments
- C – Most motor function below the level of the lesion preserved, and more than half of key muscles have a motor grade of 3 or less.
- D – Most motor function below the level of the lesion preserved, and more than half of key muscles have a motor grade of greater than 3
- E – Normal motor and sensory function

Neurologic recovery can be divided into 2 categories: (1) recovery within the zone of injury and (2) recovery below the zone of injury. The zone of injury is typically considered the first 3 abnormal dermatomes or myotomes. Muscles that are completely plegic at the time of injury within the zone of injury have a fair possibility of regaining some motor power, although this power typically is nonfunctional. Muscles that had even a small amount of contraction at the time of injury have a very good possibility of attaining functional motor power.

With regard to recovery below the level of the lesion, ASIA A patients typically do not show significant recovery in this area. Individuals who are in ASIA B have approximately a 31% chance of improving to grade D at 1-year follow-up, while those with initial grades of C have a 67% likelihood.

In addition to physical examination, MRI examination of the spinal cord can provide information regarding future recovery. The appearance of hemorrhage within the cord suggests an unfavorable prognosis, with a gradually improved prognosis suggested by (in order) contusion, edema, and a normal appearance of the cord.

Improvement following SCI may be mediated in part by recovery of partially damaged neurons. In addition, recovery at the level of injury may occur because of peripheral sprouting of spared neurons. Distal recovery following incomplete lesions may be mediated by receptor upregulation and by an expansion of synaptic fields, allowing an increase in the influence of spared pathways. A similar mechanism may underlie the development of spasticity.

Correlation of impairment on admission and 1 year afterward are described in Table 2, below.

Table 2. Correlation of Admission and 1-year Follow-up Frankel Grades (Open Table in a new window)

	One-Year Follow-up Frankel Grade			
Admission* Frankel Grade	A	B	C	D
A	84%	8%	5%	3%
B	10%	30%	29%	31%
C	2%	2%	25%	67%
D	2%	1%	2%	85%

*Admission refers to admission to a Model SCI center, which may have occurred up to 7 days following injury.

Note: Frankel grades closely correspond to ASIA impairment scale grades. Rows need not sum to 100 because Frankel E grades are not included.

Expected Levels of Function

While goals must be individualized, there are certain expectations according to injury level for healthy individuals with spinal cord injury (SCI) who have received rehabilitation training. A person with tetraplegia with injury above the level of C5 is dependent upon others for activities such as feeding, dressing, and bathing and requires the availability of an attendant at all times. However, a powered wheelchair offers such a person independence in mobility in an accessible environment and independence in weight-shifting.

Persons with injury levels of C5 and C6 have increased functional capacity but still require physical assistance for

activities such as dressing, bathing, and transfers. A person with an injury level of C7 may be independent with the proper equipment in all of these areas, requiring only some assistance with bowel management. Paraplegia is compatible with total independence at a wheelchair level. However, even these individuals may require the assistance of a homemaker.

Results of one longitudinal cohort study showed that a prediction rule that included age and 4 neurologic tests can provide an early prognosis of a patient's ability to walk after traumatic SCI. The predicted outcome can then be used to set rehabilitation goals and to improve the stratification of patients into interventional trials.[31]

Gait

Some degree of ambulation may be possible for persons with thoracic level complete paraplegia and no lower extremity function. However, bilateral knee-ankle-foot orthoses (KAFOs) and a walker or crutches are required. For patients with paraplegia, performing a sit-to-stand transfer is laborious, the gait is in a swing-to rather than a reciprocal pattern, and the gait velocity is slow. Such a pattern cannot match wheelchair mobility and is useful for exercise only. Most patients with no lower extremity function are not trained in gait.

With an injury level of L2, active hip flexion and reciprocal gait become possible. With an injury level of L3, ambulation with ankle-foot orthoses (AFOs), rather than KAFOs, is possible; however, the hips remain unstable due to the lack of active hip abduction and extension. Therefore, bilateral canes or crutches or a walker must be used. This gait may be sufficient for community ambulation but still is laborious, and some patients may prefer a wheelchair.

Functional ambulation may become possible for patients admitted with ASIA B tetraplegia. Sparing of sacral pin sensation may indicate a favorable prognosis in these patients. Most patients admitted with ASIA C tetraplegia achieve some level of functional gait. Knee extensor muscle strength of at least 3/5 on one side by 2 months following injury suggests a favorable prognosis for those with ASIA C tetraplegia. A central cord or Brown-Sequard pattern also is associated with eventual ability to ambulate.

Persons with spastic incomplete SCI tend to walk at a low velocity and have characteristic changes in gait pattern due in part to spasticity. Knowledge of the effects of antispasticity treatment upon gait in these patients is incomplete, but evidence suggests that improvement is possible.

Animal and human studies have shown that externally induced stepping movements applied to a subject with complete paraplegia suspended over a treadmill can bring about rhythmic locomotor-like responses. This suggests not only the presence of a central pattern generator located within the lumbar spinal cord but also the potential for a training effect. A multicenter study is in progress to determine whether treadmill training initiated following incomplete SCI can lead to subsequent improved ambulatory capacity compared to conventional treatment.

Upper Extremity Reconstructive Surgery

Tendon transfer surgery offers the opportunity to utilize an innervated but nonessential muscle to provide a lost function. Such surgery usually is not considered until a year following injury. Candidates must be willing to tolerate several weeks of postoperative immobilization, during which previously gained abilities may be lost. Techniques are well developed for the upper limb but not for the lower limb.

A person with a spinal injury level of C5 may have good shoulder control and strong elbow flexion. Active elbow extension is lacking, making overhead activity impossible. Such a person may benefit from a transfer procedure to the triceps tendon. One of the muscles available for transfer is the posterior deltoid. A person with an injury level of C6 may lack effective lateral pinch and may benefit from transfer of a muscle (eg, brachioradialis) to the tendon of the flexor pollicis longus. Other procedures are available to provide active finger flexion and extension.

Functional Neuromuscular Stimulation

Electrical stimulation of intact peripheral nerves can bring about contraction in muscles paralyzed by upper motor neuron injury. Stimulation can be achieved by transcutaneous, percutaneous, or implanted electrodes. Such stimulation can be useful for exercise and for function.[32, 33]

Functional neuromuscular stimulation (FNS) can be used in the upper extremity to provide lateral pinch and palmar grasp to persons with, for example, C5 and C6 tetraplegia. A totally implantable system is available with control by the position of the contralateral shoulder. Upper extremity FNS often is combined with tendon transfer surgery.

Patterned lower extremity FNS by external electrodes can allow a stationary bicycle exercise program, with beneficial cardiopulmonary, soft-tissue, and psychological effects. As with the general population, benefits are obtained only with the patient's commitment to the exercise regimen. FNS combined with lower extremity orthoses and a walker can allow gait. Control is by switches placed on the walker. The performance of such a system is not sufficient to substitute for a wheelchair, but it can be useful for specific limited mobility.

One case study involved a 23-year-old man with paraplegia from a C7-T1 subluxation, no voluntary motor function, and partial preservation of sensation below the T1 cord segment. Approximately 42 months after injury, the patient received a surgically placed 16-electrode array on the dura (L1-S1 cord segments) to allow for long-term electrical stimulation.

After experimentation with several stimulation combinations and parameters, the patient achieved full weight-bearing standing (with assistance only for balance) for 4.25 minutes; locomotor-like patterns for stepping were also noted. The patient also recovered supraspinal control of some leg movements, but only during epidural stimulation. The findings suggest that epidural stimulation may reactivate previously silent spared neural circuits or promote plasticity and may be a viable treatment for functional recovery after severe paralysis.[34]

Life In The Community

Residence

The great majority of patients with traumatic spinal cord injury (SCI) are discharged to private residences in the community. Community discharge is a goal of the rehabilitation process and occurs in well over 90% of patients treated with the Model Systems. Community discharge can place a significant burden upon the patient's support system but usually is preferable to a custodial care facility where the population typically is older and where procedures may not be geared to fostering the resident's function.

Marriage

While those married at the time of SCI have an increased rate of subsequent separation and divorce, over 75% of such marriages remain intact 5 years following injury. Persons with SCI subsequently marry at a rate below that of the general population, perhaps owing to the poverty in which many of them live, as well as a decrease in the amount of social contact. Nearly 90% of those single at the time of SCI are still single 5 years following injury, compared with an expected rate of 65% in the absence of SCI.

Employment and education

Persons with SCI may in many instances have a need to improve their educational level in order to find employment. While the average educational level at 5 years following injury is below that of the general population, at 10 and 15 years it exceeds that average. As a group, those with tetraplegia achieve a higher educational level than those with paraplegia, and those with complete injuries reach a higher level than those with incomplete injuries.

About 59% of persons sustaining traumatic SCI are employed at the time of injury. The rate of employment declines immediately following SCI but increases over subsequent years. The employment rate does not reach the level of the general population. Factors associated with a greater chance of employment include white race, young age at time of injury, and the number of years of education. Severity of injury correlates inversely.

Sexual Physiology

Sexual drive persists following spinal cord injury (SCI), though sexual physiology may be altered.[35] In men with upper motor neuron syndromes, erections in response to local stimulation (reflex erections) are common, while erections in response to cortical stimuli, such as thoughts and sights (psychogenic erections), are lost. Reflex erections, while common, may not persist long enough for sexual activity.

Those with lower motor neuron syndromes do not demonstrate reflex erections, but when sympathetic outflow from the lower thoracic and upper lumbar segment is spared, they may have psychogenic erections.

Management of erectile dysfunction can include exploration of sexual expression not involving erection. Most men also are interested in options for improving erectile function. Sildenafil (Viagra) has proven effective in cases of upper or lower motor neuron injury. Sildenafil is taken orally 20-60 minutes prior to the time of desired erection.

The use of nitrate-containing medications is an absolute contraindication to sildenafil use because of the possibility of a fatal response. Patients must be educated regarding this, as nitrates are used by some to control episodes of autonomic hyperreflexia. Nitrates also are used in cardiac disease. Prior to prescribing sildenafil treatment, cardiologists should evaluate patients with known cardiac illness.

Other options for improving erectile function following SCI include intracavernosal injection of papaverine or prostaglandin E1. Priapism and penile fibrosis are possible. Prostaglandin E1 also can be applied transurethrally using a catheter device. Hypotension is a possible side effect, though the risk can be decreased by the use of a venous constrictive band at the base of the penis.

Vacuum tumescence devices are effective. Precautions must be taken for local tissue injury. Implantation of a penile prosthesis carries several risks, including extrusion, probably due to the lack of local protective sensation.

A substantial proportion of women retain the capacity for orgasm following SCI, regardless of severity of injury. Following SCI, vaginal vasocongestion can occur in response to local stimulation. However, women with complete injuries above T6 do not demonstrate vaginal vasocongestion in response to psychogenic stimulation alone; this is because of the isolation of the brain from the sympathetic outflow to the genitals.

Fertility

Men can be infertile following SCI due to ejaculatory dysfunction and problems with the quantity and quality of sperm. The coordination of events leading to ejaculation, including seminal emission, bladder neck closure, and perineal muscle contraction, is disrupted. The reasons for poor sperm quality are unclear. Systemic endocrine changes, histologic abnormalities in the testis, seminal abnormalities, urinary tract infection, and local factors (eg, increased scrotal temperature) may play a role.

Techniques are available to induce ejaculation in men with SCI who are otherwise anejaculatory.[36] The semen can then be used for in vitro fertilization. External vibratory stimulation involves the use of a vibrator over the glans and frenulum to induce an ejaculatory reflex. Electroejaculation is the rhythmic delivery of current using a rectal probe to sympathetic efferent fibers. These techniques carry the risk of autonomic hyperreflexia.

Much less is known about female sexuality issues following SCI than is known about male sexuality issues following SCI. After an SCI, most women typically experience amenorrhea that can last for up to a year. With intervention, menstruation then begins again. SCI does not contraindicate pregnancy, but no prospective studies have examined fertility rates or pregnancy complications in these patients.

Pregnancy in a female with an SCI should be considered high risk, and such patients should be referred to a specialized center. A woman with SCI may not sense the usual indicators of labor, which raises the possibility of an unattended preterm delivery.

Psychological Adjustment and Life Satisfaction

No characteristic pattern of adjustment exists for those with spinal cord injury (SCI). Theories of stages through which patients pass have not been supported empirically. Many of those with SCI are able to respond constructively to the enormous stressor with which they are faced. Group and individual psychological treatment, including a cognitive behavioral approach, may be conducive to positive adjustment.

Significant depression occasionally occurs and may require pharmacologic intervention. Persons with SCI have an increased risk of death from suicide, particularly in the years immediately following injury. After 10 years, the rate of suicide approaches that of the general population.

Most long-term survivors of SCI feel fortunate to be alive. Even among patients with a high level of injury, including many cases of ventilator dependence, quality of life has been found to be acceptable, and most have a clear desire to live.

Quality of life following SCI is influenced less by neurologic status than by the degree to which the person is able to

resume roles in society. While research pursues the goals of preventing and reversing neurologic deficit, work must continue to remove barriers impeding the return of persons with SCI to active roles in the community.

To measure the association between mode of locomotion (ie, ambulation vs wheelchair use), as well as locomotion independence (independent vs assisted), and health, participation, and well-being in patients with SCIs, Krause et al analyzed survey data of 1493 rehabilitation patients 18 years of age or older who had had an SCI for at least 1 year.[37] They found that there were small but significant associations between independence in locomotion and each measure.

In this study, ambulation was associated with greater participation but mixed patterns of favorable and unfavorable health and well-being.[37] Those who were independent on wheelchair use reported substantially better outcomes than non-wheelchair users and those dependent on others for wheelchair use. The authors noted that although ambulation is often a recovery goal in persons with SCIs, those who ambulate do not uniformly report better outcomes than wheelchair users do.

Mortality

The life expectancy of those who survive spinal cord injury (SCI) long enough to reach a hospital has improved over the past decades, although it is below normal. The 18-year cumulative survival for those treated at a Model System is 75%, compared with 92% for the general population. Persons sustaining paraplegia at age 20 years have an average subsequent life expectancy of 44 years, compared with 57 years for the general population.

Mortality following SCI is highest in the first year after injury, after which rates decline. Despite encouraging results for many subsets of SCI, mortality remains high for the elderly patient. The hope is that those injured today will have a better prognosis than that noted above, which is from retrospective data.

The causes of death following SCI have changed. In the past, urinary tract disease and renal failure were leading causes of mortality. At present, renal failure in those with SCI is unusual. The leading cause of death at present is pneumonia, and the risk for a person with SCI far exceeds that for the general population. Nonischemic heart disease ranks second, and sepsis is third.

Pulmonary embolus is one of the leading causes of death for younger patients, a leading cause for those with paraplegia, and a leading cause in the first year following injury, when its frequency far exceeds that of the general population.

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