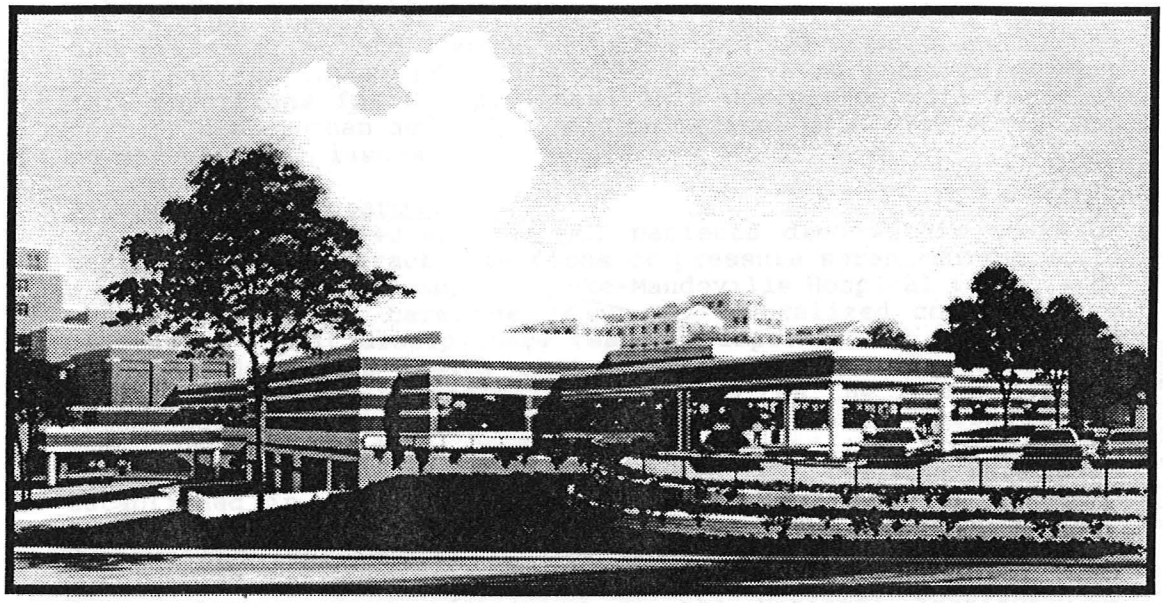
Medical Consequences of Chronic Spinal Cord Injury



Internal Medicine Grand Rounds

University of Texas Southwestern Medical School

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INTRODUCTION

Many internists are unfamiliar with chronic spinal cord injury (SCI) because it is relatively uncommon and SCI patients are cared for by other specialists. SCI presents unique problems and modifies the presentation and course of other medical conditions. It is important for internists to be able to serve as informed primary care physicians for SCI patients. This discussion will focus on chronic rather than acute SCI, and on medical problems rather than on psychosocial issues.

History of SCI treatment

Before the 1940's, most SCI patients died within weeks or months of urinary tract infections or pressure sores. During World War II Sir Ludwig Guttman, at Stoke-Mandeville Hospital in England, revolutionized SCI care. He organized centralized comprehensive care using a multidisciplinary team. He improved and interrelated acute and long-term health care facilities, providing social and vocational counseling and after care services. He introduced the technique of intermittent bladder catheterization, while at the same time, sulfa drugs became available for the treatment of urinary tract infections. Specialized SCI centers were also established in the United States, particularly by the Veterans Administration. In the 1970's the development of Emergency Medical Systems and improvements in respiratory support increased acute survival of patients with high cord injuries. Model Spinal Cord Injury Systems were instituted by the National Institute on Disability and Rehabilitation Research of the US Department of Education. The Independent Living Movement, a program initiated by the disabled, set goals of removing barriers to mobility and independent living, and increasing the ability of SCI patients to manage and direct their own care. In 1990 the Americans with Disabilities Act was passed, facilitating the rehabilitation and reintegration of SCI patients into the workplace. Despite these improvements, 50-70% of SCI patients are currently not cared for in specialty centers, but in community facilities. Many SCI patients still have trouble locating centers with expertise in long-term care of SCI or in obtaining help after discharge from rehabilitation. SCI patients may become medical orphans because their ongoing care is not the province of any medical discipline. There is limited access to patient directed, community based, long term care services or attendant care which can make the difference between independence and dependence on family members or institutionalization.

Causes of spinal cord injury

There are a number of causes of SCI or dysfunction, but the greatest number of SCI patients are victims of trauma, and this review will concentrate on traumatic SCI. The incidence of traumatic SCI in the US is about 8000-10,000 cases per year, while the prevalence is 700-800/million. Thus, at any one time, there are about 175-200,000 individuals with SCI in the U.S.

Causes of spinal cord injury or dysfunction

•Trauma	•Paget's Disease
•Cervical Spondylosis	•Arachnoiditis
•Tumor	•Multiple Sclerosis
•Infection	•AVM
•Radiation	•Meningomyelocele
•Decompression (bends)	•Syringomyelia

Causes of traumatic SCI

Causes	% SCI
• MVAs	48
• Falls	21
• Violence	15
• Sports	14
• Other	12

Risk taking behavior, including alcohol and other substance abuse, violence, and reckless driving accounts for many cases of SCI. The largest proportion is due to motor vehicle accidents (MVAs). The elderly are susceptible to SCI from falls. Gunshot and stab wounds cause a number of SCI. Sports take their toll. In particular, diving accidents are likely to result in C4-6 injuries and tetraplegia.

Demographics

Age at time of SCI

Age group	% patients
0-15 yrs	5
16-30	61
31-45	19
46-60	9
61-75	4
> 75	1

About 80% of SCI patients are men, and the great majority are in their 20's at the time of injury.

Prognosis of SCI

Long term survival of SCI patients has improved markedly since the 1940's, and now approaches that of the non-SCI population. About 50% of patients will live 30 years or more after SCI. Survival is longer for paraplegics than tetraplegics and for those injured at an earlier age. Improved long-term survival has led to aging of the SCI population. About 40% of SCI patients are older than 45. Over 90% of SCI patients live at home, and 30% return to full-time work, while many others volunteer or work part-time. Despite their disabilities, most SCI patients regard their quality of life as good or excellent. Life satisfaction after SCI is more closely related to psychosocial adjustment and support than to level of injury.

Long-Term Survival after SCI

Yrs post SCI	% Surviving
10	85
20	71
30	53
40	35

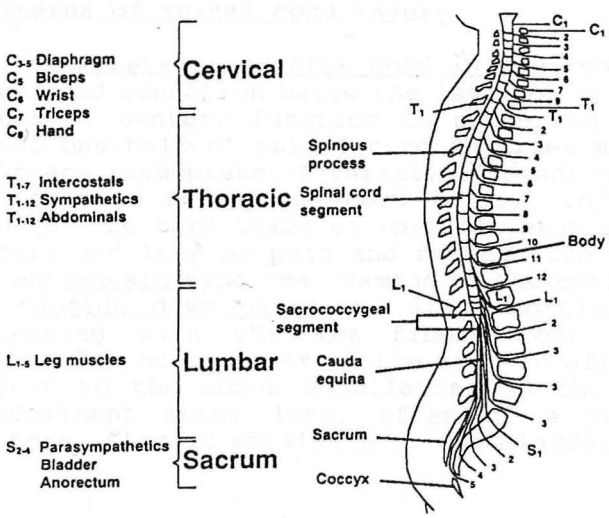
Causes of death in chronic SCI

Condition	% Deaths
•Respiratory disease	22
•Heart disease	19
•Trauma/suicide	14
•Cerebrovascular dis.	14
•Sepsis	9
•Urological disease	3
•Other	19

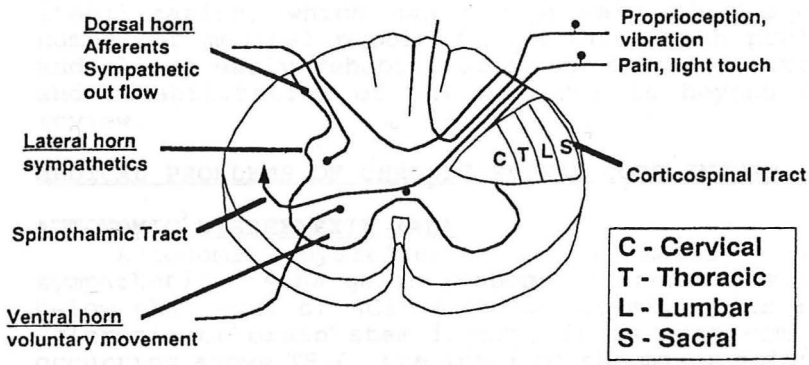
Pneumonia and heart disease have replaced urological problems as the most common causes of death in chronic SCI.

ANATOMY OF THE SPINAL CORD AND PATTERNS OF INJURY

The spinal cord gives rise to 8 paired cervical, 12 thoracic, 5 lumbar, and 5 sacral nerves. Innervation of the diaphragm derives from C3-5. The sympathetic autonomic system arises in T1-L2, and the caudal division of the parasympathetic system in S2-4. The cord extends to the L1-2 vertebral body, where it ends in the conus medullaris. The lower lumbar and sacral nerves continue as the cauda equina.



In the gray matter of the cord the ventral horn contains cell bodies primarily mediating voluntary movement. The lateral horn contains cell bodies of the sympathetic autonomic system, while the dorsal horn contains interneurons of the sensory or afferent system. The white matter consists of ascending and descending myelinated nerve tracts. The spinothalamic tract transmits ascending sensations of pain and temperature from the dorsal horn to the opposite thalamus. Spinothalamic fibers cross to the opposite side of the cord within one or two segments of entry. The dorsal column transmits afferent information of fine touch, vibration, and position sense, but unlike spinothalamic fibers, dorsal column fibers ascend to the brainstem on the same side of the cord that they enter. The corticospinal tract contains descending fibers for voluntary motor control from the cortex to the anterior horn cells.



Patterns of spinal cord injury

Complete transverse cord injury results in loss of all motor power and sensation below the level of injury. A variable degree of motor or sensory function is preserved after incomplete injury. About one-half of spinal cord injuries are complete and the other half are incomplete. A variety of syndromes results from different patterns of incomplete cord injury. Injury to the right or left side of the cord leads to loss of motor control on the side of the injury and loss of pain and temperature on the opposite side, the Brown-Sequard syndrome. Damage to the central cervical cord results in "upside down paresis". Since corticospinal tract fibers are organized with the arm fibers most centrally and the legs laterally, motor power of the arms is affected more than the legs. Injury to the conus medullaris and the cauda equina results in predominant motor loss, often of a variable and asymmetrical pattern. Flaccid paralysis of the bladder and anorectum may occur.

ACUTE MANAGEMENT OF SPINAL CORD INJURY

High-dose methylprednisolone in acute SCI

The publication of the second National Acute Spinal Cord Injury Study (NASCIS II) in 1990 established the effectiveness of early high-dose intravenous methylprednisolone in improving the outcome of traumatic spinal cord injury. The regimen consists of a bolus of 30 mg/Kg of intravenous methylprednisolone, given within 8 hours of acute SCI, followed by an infusion of 5.4 mg/Kg/hr for the next 23 hours. Physicians who work in emergency rooms should be familiar with this treatment, since it is now considered standard care.

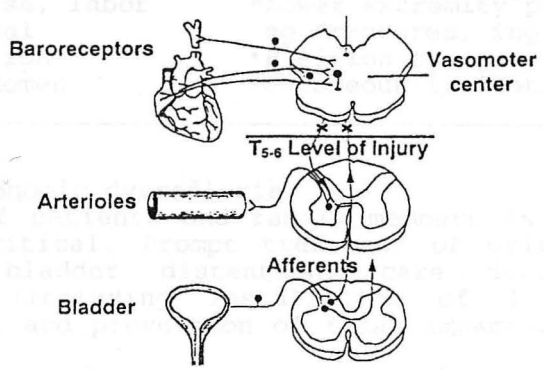
Acute management of spinal cord injury is best provided in a SCI center where a team of experienced individuals can provide comprehensive management and start the rehabilitation process as quickly as possible. The first priority of care is stabilization of the spinal column and attention to other injuries and acute medical problems. There has been a trend toward early operative spinal stabilization, which has the primary advantage of reducing the number of medical problems associated with prolonged immobility, and allows early rehabilitation. A detailed account of early care and rehabilitation of SCI patients is beyond the scope of this review.

MEDICAL PROBLEMS OF CHRONIC SPINAL CORD INJURY

AUTONOMIC DYSREFLEXIA (AD)

Autonomic dysreflexia is a massive paroxysmal reflex sympathetic discharge in response to noxious stimuli arising from below the level of SCI. Although it may occur in severe multiple sclerosis or brain stem injury, it is most common in chronic SCI occurring above T5-6, the level of the major splanchnic sympathetic outflow. Of SCI patients at risk, 30-85% will suffer autonomic dysreflexia. If destined to develop, it occurs after recovery from spinal shock, usually within the first 6 months after injury, but may be delayed for years.

Physiology of Autonomic Dysreflexia



Noxious or other sensory afferent impulses enter the cord and ascend until blocked at the level of injury. Interneurons excited by this input synapse with preganglionic sympathetics in the intermediolateral gray and activate splanchnic sympathetic outflow. The result is a rapid rise in systemic blood pressure, sometimes to levels of 2-3 times normal. Normal blood pressure in a person with tetraplegia may be 90/60, so that a pressure of 120/80 may represent a manifestation of AD. Carotid and aortic baroreceptors are intact, and relay information to supraspinal regulatory centers, but except for vagal outflow, feedback regulation is blocked because of the SCI. Bradycardia and vasodilation above the level of injury are unable to compensate for the unrestrained sympathetic activity below the level of injury.

Clinical presentation

Episodes of autonomic dysreflexia are paroxysmal. The most common symptoms are pounding headache, which may be severe, and flushing, skin blotching, and sweating above the level of SCI. Other symptoms are anxiety, nasal congestion, goosebumps above the injury level, cutaneous vasoconstriction below the level of injury, nausea, abdominal discomfort, and penile erections. The cardiovascular consequences are the most serious, and may be catastrophic. The hypertensive crises may cause retinal or cerebral hemorrhage or seizures, myocardial ischemia or acute congestive heart failure, atrial fibrillation or heart block. Although bradycardia is the classic finding, many patients have tachycardia during episodes. The noxious stimuli arise below the level of injury. About 75% of episodes of autonomic dysreflexia occur in response to urological stimuli such as bladder distention, spasm, or infection. Bowel distention accounts for another 15% of episodes. In susceptible patients even innocuous stimuli, such as tight clothes, may provoke an episode.

STIMULI PROVOKING AUTONOMIC DYSREFLEXIA

- Genitourinary
  - Bladder distention, infection, spasm
  - Intercourse, labor
- Gastrointestinal
  - Constipation
  - Acute abdomen
- Surgical procedures, post-op pain
- Radiological procedures
- Lower extremity problems, eg fractures, ingrown nails
- Position changes, ROM
- Cutaneous irritation, sores

Treatment of autonomic dysreflexia

Education of patients and family members is important, and prevention is critical. Prompt treatment of urinary infection, prevention of bladder distention, care during urological instrumentation (including installation of lidocaine before catheterization), and prevention of fecal impaction will prevent many episodes.

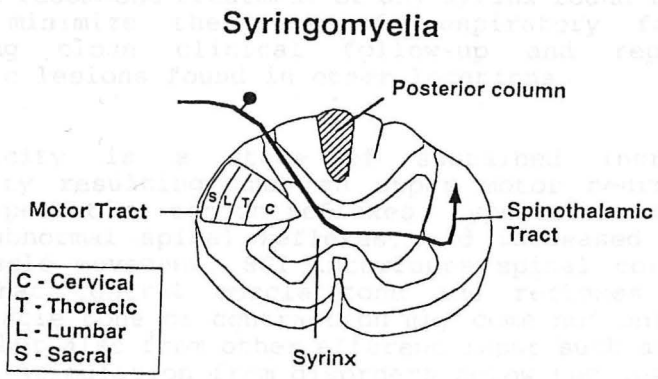
Immediate management should include sitting the patient, as that may lower blood pressure. The cause should be sought immediately. The urinary catheter should be checked for blockage. A distended bladder must be drained, but gently, and using lidocaine gel. If fecal impaction is suspected, local anaesthetic ointment should be used for the rectal examination. The skin and legs should be examined. Tight clothes and shoes should be loosened. Drug treatment should be given if the cause cannot be quickly corrected and if the systolic pressure is over 160 mm Hg. Nitroglycerine paste, 1" applied above the level of injury, is effective. Nifedipine 10 mg as a punctured capsule is also effective, and may be repeated in 10 minutes if the diastolic pressure is still higher than 100 mm Hg. If the episode is severe and does not respond quickly, the patient should be placed in the intensive care unit and given a short-acting titratable antihypertensive agent such as nitroprusside, while continuing the investigation for a cause. An acute intra-abdominal condition may provoke autonomic dysreflexia, and should be investigated in persistent cases. However, overtreatment must be avoided as severe hypotension may result when the cause is corrected. For patients with frequent episodes prophylactic prazosin has been used.

SYRINGOMYELIA

Syringomyelia is a potentially devastating complication occurring in about 1-3% of SCI patients, anytime from several months to as long as 30 years after injury.

Pathophysiology

The initial event is the formation of a cavity in the cord by liquefaction of injured tissue or hematoma. The cavity usually starts at the level of trauma in the central cord and may eventually communicate with the central canal. In some patients the cavity begins to expand in diameter and extent, both in the rostral and caudal directions, compromising the cord in the process.



Clinical presentation

Rostral extension first leads to loss of pain and temperature sensation above the level of trauma. Vibratory sensation and proprioception may be spared. Sensory loss is almost universally present even if it is not the presenting complaint. If the syrinx is untreated, progressive motor deficits may occur. Rostral extension may lead to loss of residual arm function or respiratory function in a tetraplegic. Since these patients are already devastated, the further loss of function may be life threatening. Caudal extension may convert spastic paralysis of the legs or bladder to flaccid paralysis. Initial symptoms of numbness or weakness may be unilateral. Pain is another common symptom, and can appear at any time. It may be intermittent or constant, aching or stabbing. It is located at or above the level of the injury, often in the upper back, shoulder blades, shoulders, and arms. It is often increased by straining, coughing, or sneezing.

Diagnosis

Symptoms of syringomyelia may be subtle, and mistaken for other conditions. A high level of suspicion is important in order to make the diagnosis before serious neurological deterioration has occurred. The diagnosis is best confirmed by magnetic resonance imaging (MRI). The low signal of the cystic fluid contrasts with the intermediate signal of the spinal cord. With increasing use of MRI, it has been found that spinal cord cysts are present in about 50% of SCI patients. The great majority of these are asymptomatic, and do not progress. The mean diameter of symptomatic cysts is about 7 mm, and the length can vary from 3 to 10 segments. There is surprisingly little correlation between the size of the syrinx and the severity of the deficits.

Treatment

The standard treatment of symptomatic syringomyelia is creation of a shunt from the cyst to the subarachnoid or another low pressure space such as the pleural or peritoneal cavity. Results are good, with at least partial relief of pain or improvement in motor weakness in the great majority of patients. The natural history of asymptomatic syringomyelia is variable. Some authorities recommend treatment of any syrinx found in the cervical cord, to minimize the risk of respiratory failure, while recommending close clinical follow-up and repeat MRI for asymptomatic lesions found in other locations.

SPASTICITY

Spasticity is a state of sustained increased muscle contractility resulting from an upper motor neuron lesion. It includes hyperactive tendon reflexes, hyperactive muscle stretch reflexes, abnormal spinal reflexes, and increased resistance to passive muscle movement. SCI interrupts spinal cord integrative circuits that control muscle tone and reflexes. Stimuli for abnormal muscle tone or contraction may come not only from muscle stretching but also from other afferent input such as cutaneous or nociceptive stimulation from disorders below the level of injury.

Muscle groups served by lower motor neurons at the level of injury may be flaccid rather than spastic. Patients with damage to the conus medullaris or cauda equina will have flaccid paralysis.

Clinical presentation

Spasticity evolves over 1-2 years post injury and then tends to stabilize. Most SCI patients have some degree of spasticity in muscles below the level of injury, but there is considerable variation among patients in the degree of spasticity. The reason for this variability is not understood.

Not all spasticity is dysfunctional or requires treatment. A certain degree of spasticity helps maintain posture at the hips and trunk. On the other hand, excessive spasticity may cause a number of problems, such as decreased range of motion and function. For example, strong muscle spasms may cause dangerous movements during transfers or during driving. They may awaken patients at night, or cause pain. Chronic spasticity is often asymmetric. Flexors are generally more spastic than extensors. This imbalance may lead to contractures or postural abnormalities such as scoliosis. Excessive spasticity of the abdominal muscles may lead to respiratory difficulty. Positioning problems caused by spasticity may predispose to pressure ulcer formation and great difficulty in bowel, bladder, and perineal care. An important point to remember in caring for patients with SCI is that changes in spasticity may be a sign of a problem in another organ system. For example, increasing spasticity may be the only symptom of a urinary tract infection, bladder distention, or intra-abdominal infection. Certain patients may be able to use changes in spasticity as a sign of the need for emptying the bowel or bladder.

Treatment of spasticity

Treatment of spasticity includes regular stretching and range of motion exercises. Positioning may be important. For example, the prone position inhibits flexor spasticity and thus may help patients control nighttime spasms.

As mentioned, not all spasticity is harmful, and many patients do not require therapy. When drug treatment is needed, baclofen is usually the first choice, because of safety. Baclofen binds to gamma-aminobutyric acid (GABA) receptors in the spinal cord, and reduces release of excitatory neurotransmitters from large afferent fibers. About 75% of SCI patients have a satisfactory response to baclofen. The usual starting dose is 5 mg three times daily, increased by 5 mg every 3 days up to a total daily dose of 80 mg. Sedation is the main side effect. Sudden discontinuation of baclofen may lead to rebound spasms, hallucinations, and seizures. Physicians hospitalizing SCI patients need to be aware of this, and avoid inadvertent omission of baclofen. Diazepam is an alternative to baclofen. It is equally effective, but can result in depression or dependence. It is started in a dose of 2 mg once or twice a day and gradually increased. Doses above 40-60 mg per day do not provide additional benefit. Dantrolene acts on the muscle itself by

interfering with calcium transport. Generalized muscle weakness may occur, as well as drowsiness, fatigue, dizziness and diarrhea, but these are usually transient. Therapy is started at 25 mg per day and increased up to 400 mg per day in divided doses. The main drawback to dantrolene is the occurrence of serious hepatotoxicity at high doses. Occasionally severe spasticity which does not respond to conventional drug therapy requires more dramatic measures. Recently it has been found that continuous intrathecal infusion of small doses of baclofen is effective. Various invasive therapies such as dorsal root block or ablation have been used. Local tendon lengthening procedures may be helpful for specific problems, such as Achilles lengthening for equinus deformity (which makes proper shoe fitting or wheelchair footrest position difficult).

VENTILATORY FUNCTION

Physiology of normal breathing

Normal inspiration involves use primarily of the diaphragm, with contributions by the external intercostals and the accessory muscles. Normal expiration is passive, caused by the elastic recoil of the lungs and chest wall. Effective cough requires the ability to generate adequate inspiratory volume and then expiratory force with the internal intercostal and abdominal muscles. The accessory muscles are innervated by cranial nerve XI and cord segments C1-8, the intercostals by T1-11, the diaphragm by C3-5, and the abdominal muscles by T6-12. Chronic SCI results in loss of inspiratory capacity because of muscle weakness and decreased chest wall compliance due to muscle spasticity. Loss of effective cough occurs in patients who have significant weakness of the abdominal muscles.

Inspiration	Innervation
Diaphragm	C3-5 (mostly C4)
Ext. intercostals	T1-11
Accessories	C1-8, CN XI
Passive expiration	
Elastic recoil (Chest wall and lungs)	
Forced expiration (cough)	
Abdominals	T6-12
Int. intercostals	T1-11

Ventilatory function and level of SCI

Level	Function
C1-2	Ventilator dependent
C3-4	Variable: C3 at least partially ventilator dependent; no cough
C5-T1	Ventilator independent; no cough
T2-4	Weak cough
T5-10	Fair cough

Patients with SCI at C1-2 are universally ventilator dependent. Patients with injury at C3 and C4 vary in their ventilatory function. Some diaphragmatic function is normally required for ventilatory independence. Thus patients with C3 are usually at least partially ventilator dependent, while many patients with C4 injuries may be ventilator-independent. They have an ineffective cough. Patients with SCI at C5 or below have normal diaphragmatic function and should be ventilator independent, but may have weak cough, depending on the level of injury. The presence of other neurological deficits from head injury or of intrinsic lung disease may alter these generalizations.

Tracheostomy after SCI

During the acute phase of SCI, a number of patients require tracheostomy. Long-term tracheostomy may be necessary in some SCI patients with other neurological injury, intrinsic lung disease, or no caretaker, but should be avoided if possible. The disadvantages of tracheostomy include impairment of speech, stimulation of increased bronchial secretions, and local complications such as tracheal stenosis, bleeding, and infection.

Respiratory therapy

Respiratory therapy after SCI should include measures to preserve normal inspiratory capacity and chest wall compliance, strengthen the residual ventilatory muscles, and remove secretions. Intercostal muscle spasticity may lead to decreased chest wall compliance and inspiratory capacity. This can be ameliorated by ensuring periodic lung inflation to maximum capacity, using positive pressure ventilation with a mask and bag, positive pressure ventilator, or glossopharyngeal breathing, as described below. Removal of secretions is most effectively done by the use of assisted cough. After maximal inspiration, using the techniques just described, the patient closes his glottis and then attempts to cough as the respiratory therapist or caregiver applies a firm

upward thrust just below the diaphragm. Assisted cough can produce expiratory flow rates of 5 to 7 l/sec, which are effective in clearing secretions. It can be done in patients with a tracheostomy if the balloon is inflated to reduce air leak. It should not be done in patients with a full stomach, as it may lead to regurgitation and aspiration.

Ventilatory support

Glossopharyngeal breathing (GPB) is a technique which should be taught to all SCI patients dependent on ventilatory support. It was first developed for polio patients. In GPB the tongue and pharyngeal muscles are used to add to a maximal inspiratory effort by projecting boluses of air past the vocal cords. The vocal cords close with each gulp. GPB can provide normal tidal volume for some time. Some patients use GPB to supplement inspiratory capacity in assisted coughing or speech. It is also important for use during transfers and as protection against ventilator failure. GPB is difficult for patients with tracheostomy because the gulped air leaks around the tube even if it is plugged. A training manual and videotape are available for teaching GPB.

Abdominal binders are useful for patients with marginal diaphragmatic function. In the sitting position an abdominal binder pushes the abdominal contents in and the diaphragm up, restoring a favorable length-tension relationship.

Ventilatory support can also be provided by devices to produce intermittent abdominal pressure, such as the Pneumobelt. A binder fitted around the abdomen contains an air bladder, which is cyclically inflated to push the diaphragm up and produce active expiration. These devices are useful for daytime ventilatory support when the patient is sitting up. They are not effective in the supine position.

There are a number of negative pressure body ventilators. The iron lung is the classic example, but has the disadvantages of reducing access to the patient for nursing care and producing claustrophobia for some patients. A Cuirass ventilator consists of a hard shell fitted snugly over the thorax within which cyclic negative pressure can be generated. The Pulmowrap is another variation of negative pressure body ventilator.

Positive pressure ventilators are required by less than 5% of SCI patients. Some require continuous ventilatory support while others require intermittent support, particularly when fatigued or sleeping. A standard volume ventilator is used at night, and a small portable ventilator attached to a wheelchair allows the patient mobility. Intermittent positive pressure ventilation may be provided through a tracheostomy, but, as discussed, should be avoided if possible. Ordinary mouthpieces can be used during the day, leaving the patient free to speak or operate a sip-and-puff wheelchair or computer. Fitted oral, nasal, or combined oral-nasal masks can be used at night for continuous ventilatory support.

Phrenic nerve stimulation has never become a widespread technique. Patients must have intact phrenic nerves and high cervical cord injuries. Electrodes are implanted around the phrenic nerves in the

neck or chest and connect to a subcutaneous implanted stimulator which receives signals from an external transmitter. Phrenic nerve stimulation has been used most often to provide respirator-free time at night. Long-term use has been limited by progressive loss of phrenic nerve responsiveness.

Living on the edge

SCI injury patients with high thoracic or cervical injuries live on the edge of ventilatory failure because of their reduced vital capacity and ability to cough. Thus, physicians must recognize that respiratory problems in SCI patient must be taken seriously. Even minor respiratory infection may lead to acute respiratory failure in SCI patients. Influenza and pneumococcal vaccines should be current, and bronchitis should be treated aggressively. Gastric distention or any condition which worsens abdominal spasticity and limits diaphragmatic excursion may compromise breathing. For the same reasons pregnant SCI patient must be closely followed.

Chronic changes may also result in the gradual development of respiratory insufficiency in a previously compensated patient. Syringomyelia may compromise diaphragmatic function, as previously described. SCI patients are susceptible to scoliosis because of loss of postural muscle tone or unbalanced spasticity. Kyphosis may occur after high thoracic SCI, and is worsened by osteoporosis, obesity, long wheelchair use or spasticity of the abdominal wall. Both kyphosis and scoliosis may worsen respiratory function.

CARDIOVASCULAR FUNCTION

SCI injury results in a number of cardiovascular disturbances. With the acute injury "spinal shock" results in loss of all spinal reflexes below the level of injury. Since sympathetic outflow originates from segments T1-L2 (of which T1-4 are the most important for the cardiovascular system), lesions above this level result in bradycardia and hypotension due to unopposed parasympathetic stimulation in the face of loss of sympathetic tone. Vagal stimulation, from tracheal suctioning, for example, may cause severe bradycardia, and even cardiac arrest during the acute phase of SCI. The acute phase of spinal shock usually lasts only 24-48 hours. After recovery from spinal shock, patients with SCI above T5-6 may develop chronic autonomic dysreflexia. Severe hypertension during episodes of autonomic dysreflexia may result in myocardial infarction, acute congestive heart failure, and cardiac arrhythmias.

Postural hypotension is a problem for some patients with high SCI injury due to loss of sympathetic vasoregulation. Average systolic blood pressure in tetraplegics is generally low, about 100 mm Hg. The development of spasticity in the legs over several months after acute SCI may improve orthostatic hypotension. In those who have persistent problems, elastic hose and abdominal binders may help.

Cardiovascular deconditioning

Cardiovascular diseases have become an important cause of death in SCI, and deconditioning is a major contributing factor. The exercise capacity of chronic SCI patients is related to the level of injury. The muscle below the level of injury cannot be exercised, and undergoes disuse atrophy. High SCI injury also results in decreased sympathetic response to exercise. Inactive lower level paraplegics have a maximum oxygen uptake of 20-30 ml/min/kg. This can be improved by training. Paraplegic wheelchair athletes can attain maximum oxygen uptake levels of 35-50 ml/min/kg. For reference, elite non-handicapped athletes can attain maximum oxygen uptake levels of 75 -80 ml/min/kg. Tetraplegics are more limited, although they may also have enough preservation of upper extremity function to use wheelchair training, swimming, calisthenics, and weight training. Chronic deconditioning leads to decreased left ventricular mass and chamber size. Functional electrical stimulation (FES), as discussed below, is an alternative for providing conditioning and preserving muscle mass.

HDL levels

Mean HDL levels are lower than normal in SCI patients. In one study, about 25% of SCI patients had HDL-C levels of less than 35 mg/dl. In combination with deconditioning, this may lead to premature atherosclerosis. Exercise may raise HDL levels, but even wheelchair athletes have relatively low levels. Mean total and LDL cholesterol levels are normal.

Functional electrical stimulation

After SCI, muscles undergo disuse atrophy below the level of injury. This is particularly pronounced in muscles which bear weight and which cross single joints. Denervation atrophy also occurs in those muscles served by the cord at the level of the SCI. Sprouting in residual neurons may compensate for some denervation atrophy. Functional electrical stimulation (FES) is technique in which low frequency (eg. 20 Hz) electrical stimulation of muscles is provided by skin or implanted electrodes. FES has a variety of uses. Sequentially programmed FES has been used to enable walking in paraplegics, or facilitate upper extremity use, but the systems required are complicated and cumbersome. FES may be used to strengthen marginal residual muscle function of the upper extremities in tetraplegics. More commonly, FES has been used to provide exercise of paralyzed muscles for the purpose of improving conditioning. Preservation of muscle mass may result in a decreased incidence of pressure sores. For some patients, the improvement of self esteem that results from prevention of atrophy is worthwhile.

TEMPERATURE REGULATION

SCI above T-6 results in impaired temperature regulation, with high cervical lesions resulting in the most profound disturbance. Thus, SCI patients are susceptible to both hypothermia and hyperthermia from environmental exposure.

Normal body temperature regulation depends on the hypothalamic integration of information received from temperature sensors. Most of this sensory input proceeds from receptors in brain, but some temperature receptors are located in the skin, and input from these is lost below the level of SCI. The loss of peripheral afferent temperature information seems to alter the threshold for shivering and sweating in SCI slightly. Normal acute defenses against hypothermia include shivering and vasoconstriction, of which shivering is the most powerful. During shivering, heat production may rise to 4 to 5 times the normal resting rate. SCI leads to inability to shiver below the level of injury. In response to cold, tetraplegics can only double heat production. The ability to respond to cold by cutaneous vasoconstriction is also impaired by SCI above the sympathetic outflow. Hyperthermia normally leads to sweating and vasodilatation. The intensity of sweating in response to hyperthermia is diminished below the level of SCI. Although some sweating occurs as a spinal reflex, it seems that central nervous system facilitation is necessary for normal sweating intensity. Non-thermoregulatory sweating persists in high SCI, as, for example during episodes of autonomic dysreflexia. Despite impairments of thermoregulation, SCI patients have a normal ability to mount febrile responses to infections. Thermogenesis in response to pyrogens may occur through different mechanisms than those which occur in response to cold.

GASTROINTESTINAL FUNCTION

Most gastrointestinal function is mediated by the enteric nervous system, the vagus, and gastrointestinal hormones, and thus independent of the spinal cord. Sympathetic innervation of the gastrointestinal tract originates in segments T6-L3, but chronic interruption of splanchnic sympathetics does not have major effects on digestive function, with the important exception that visceral pain afferents are carried in the sympathetic nerves. The vagus provides parasympathetic innervation to the proximal gut as far as the transverse colon. However, parasympathetic innervation of the left colon and rectum and voluntary motor control of the external sphincter originate in sacral segments S2-4. The most important chronic effects of SCI on the digestive tract are loss of visceral pain sensation in high thoracic injury and abnormalities of left colon transport and defecation.

Upper gastrointestinal problems

A moderately increased frequency of upper gastrointestinal problems has been reported after SCI. The incidence of gastroesophageal reflux is increased. Cases of chronic gastric dilatation have been noted, primarily in elderly patients with tetraplegia, and acute gastric dilatation may occur after abdominal surgery. Chronic abdominal distention is common after SCI, but is likely due to secondary effects of colonic dysmotility. There is an increased incidence of gallstones with injuries above T10 during the first year after injury, although gallbladder emptying time and residual volume are normal.

Colonic and anorectal problems

Studies of colonic function after SCI show prolonged transit time in the left colon. Changes in motility and reduced compliance of the left colon have been found, and some studies have shown absence of the normal postprandial gastrocolic reflex. However, it is not clear whether prolonged colon transit after SCI is due primarily to colonic dysmotility or to problems with defecation. About 25% of SCI patients develop significant chronic problems related to colonic function, including difficulty with defecation, bloating, distention, and abdominal discomfort.

The reflex defecation center and lower motor neurons controlling the external sphincter are located in segments S2-4. In normals, filling of the rectum (or the gastrocolic reflex) stimulates strong reflex contractions of the rectosigmoid, and stool descends into the anal canal. In response, the internal sphincter relaxes, but there is reflex contraction of external sphincter, protecting against incontinence. The sensation of the urge to defecate results in a decision to delay (in which case the rectum accommodates) or a decision to defecate, in which case descending cortical controls allow voluntary relaxation of the external sphincter and contraction of the abdominal muscles, increasing intra-abdominal pressure and expelling the stool. SCI results in the loss of the sensation of rectal fullness. However, other signs, such as autonomic dysreflexia or increased spasticity, may occur, and serve as signals that the rectum should be emptied.

Spastic bowel

In SCI above S2-4 reflex defecation is intact. However, voluntary control of the external sphincter is lost, and the sphincter may be spastic, causing anorectal dyssynergia. This protects against incontinence but may impede defecation. In addition, weakness of abdominal muscles after injury above T6-12 results loss of effective Valsalva maneuver.

Flaccid bowel

SCI at S2-4 or below creates a flaccid anorectal area. There is loss of the normal awareness of a need to defecate, and autonomic signs are absent. Reflex defecation does not occur. Sphincters are also flaccid. The result is retention of stool with overflow incontinence. Since the innervation of abdominal muscles is spared, the ability to Valsalva is preserved.

Bowel management

A reasonable goal of bowel management in SCI is to evacuate the rectum about every 2-3 days with a regimen that is acceptable to the patient, does not involve an excessive commitment of time, and which avoids precipitation of autonomic dysreflexia. Dietary fiber or supplements are important to maintain proper stool consistency.

In patients with mid and high cord lesions and intact reflex defecation, planned bowel movements may be stimulated by leaning forward on the toilet, which increases abdominal pressure, by digital stimulation of the internal sphincter, or by glycerine or bisacodyl suppositories. The suppository must contact the rectal

mucosa. Patients with lower cord lesions and a flaccid rectum will not have reflex defecation, and may have to empty the bowel manually. On the other hand they may have better balance and manual dexterity, and a more effective Valsalva.

Laxatives should be avoided if possible, especially harsh laxatives. If laxatives are found necessary, it should be remembered that the paralyzed bowel is relatively insensitive and often takes 24 hours longer than normal to respond.

Fecal impaction is a common precipitating stimulus for episodes of autonomic dysreflexia. If impaction is found, disimpaction should be done gently, after application of a topical anaesthetic ointment, as the process may worsen the autonomic dysreflexia.

Hemorrhoids and intermittent rectal bleeding are reported by most SCI patients. Solitary colonic ulcer and early diverticulosis may occur. Surveillance for colon cancer should not be neglected in SCI patients over the age of 50. Periodic fiberoptic sigmoidoscopy is probably the best screening test, as stool studies for occult blood are likely to have a higher than normal false positive rate.

Evaluation of the acute abdomen in SCI

SCI makes the evaluation of the acute abdomen difficult, but it is usually possible to make the diagnosis quickly and accurately if certain principles are understood.

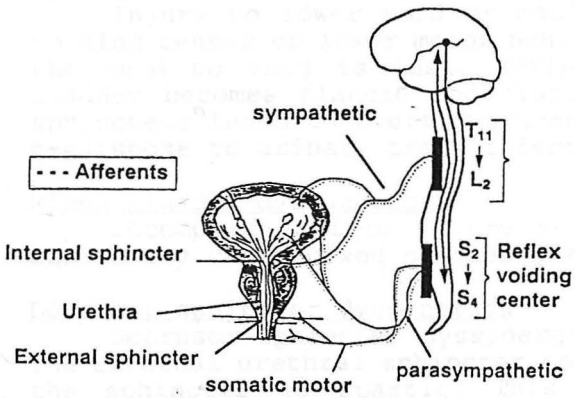
Cord lesions below T11-12 spare the visceral afferent pain pathways, which ascend via the sympathetic chain for several segments before entering the cord. In these patients, the presentation of abdominal disease is similar to that in patients with an intact spinal cord. High cord lesions, above T5, interfere with the ability to perceive abdominal pain. Even when most visceral sensation is lost, patients may report a vague sense that something is wrong in their abdomen, and this report should be taken seriously. Referred pain may still occur. For example, pain referred to the shoulder is valuable as a clue of an upper abdominal process. Anorexia, nausea, vomiting and an increase in distention commonly occur. Autonomic dysreflexia or an increase in spasticity may be signs of an acute abdomen in patients with lesions above T5-6, and this should be considered when another cause is not apparent. SCI patients preserve the ability to respond to inflammation or infection with fever. Abdominal tenderness is a reliable physical finding, but abdominal rigidity cannot be assessed because of abdominal muscle spasticity, and many SCI patients have chronic abdominal distention. Radiographic studies, such as plain films, sonography, and CT are often helpful. It should be remembered that SCI patients may normally have increased small bowel gas. It is a common mistake to attribute the symptoms of an acute abdomen to a urinary tract infection. The finding of pyuria should not lead to complacency on the part of the physician. Rupture of the bladder and fecal impaction with complicated stercoral ulcer are two rare conditions which should be considered in SCI patients with an acute abdomen.

BLADDER FUNCTION

The number of deaths due to urinary disease in SCI has been greatly reduced by antibiotics and improved bladder management techniques. SCI usually causes some degree of bladder storage and emptying dysfunction- loss of voluntary control, lack of coordination of the bladder and the sphincters, or changes in bladder capacity, power of bladder contraction, and resistance of urinary sphincters. If not properly managed, these problems may lead to urinary tract infection, stone formation, or chronic renal failure.

Bladder innervation and function

The body of the bladder is composed of the detrusor muscle. The internal sphincter, at the neck of the bladder, is composed of smooth muscle, while the external sphincter is composed of striated muscle under voluntary control. Voiding occurs when the detrusor muscle contracts while the sphincters relax. The anatomy of the vesico-ureteral junction prevents reflux of urine into the ureters at the pressures usually generated by voiding.



Spinal segments T11-L2 provide sympathetic innervation to the bladder. Sympathetic stimulation relaxes the detrusor muscle through beta-adrenergic receptors and stimulates tonic contraction of the internal sphincter through alpha-adrenergic receptors, facilitating bladder filling and storage. Spinal segments S2-4 increase detrusor contraction through parasympathetic cholinergic stimulation as well as supply voluntary motor control of the external sphincter. These segments coordinate contraction of the detrusor muscle and relaxation of the internal and external sphincters.

Sensory input from the sphincters and bladder travel back to the cord through the same pathways as the sympathetic system. Complete SCI injury results in loss of the sensation of the urge to void. However, bladder filling may cause symptoms of increased spasticity or, in patients with SCI above T6, symptoms of autonomic dysreflexia which can be used by the SCI patient as a signal to empty the bladder.

Although debated, a reflex voiding center is thought to be located in the S2-4 section of the sacral cord. When the bladder fills to a threshold volume, sensory input is relayed to the sacral reflex center, thence to the micturition centers in the pons and

hypothalamus and to the cortex. In adults without SCI, bladder filling is first sensed at volumes of 175 to 250 ml, and the urge to void is experienced at 350 to 500 ml. Upon release by descending tracts, the reflex voiding center coordinates detrusor contraction and sphincter relaxation. After SCI the reflex voiding center functions autonomously, if intact.

Upper motor neuron dysfunction (spastic bladder)

With SCI above the reflex voiding center, awareness of the need to void is lost. Reflex voiding occurs the bladder fills to a threshold volume. However, the bladder and the external sphincter may become spastic. Bladder spasticity (hyperreflexia) causes detrusor contractions at lower than normal filling volumes, leading to frequent urination of small volumes, sometimes with incontinence. Spasticity of the sphincter may lead to failure of the sphincter to relax during detrusor contraction (detrusor-sphincter dyssynergia). Urinary retention, vesico-ureteral reflux, and hydronephrosis occur if voiding pressures are over 40 cm of water.

Lower motor neuron dysfunction (flaccid bladder)

Injury to lower cord or cauda equina may damage the reflex voiding center or lower motor neurons to the bladder. Awareness of the need to void is lost. Reflex voiding does not occur. The bladder becomes flaccid and distended. Flaccid paralysis of the sphincters leads to overflow incontinence. Large residual volumes predispose to urinary tract infections.

Mixed bladder dysfunction

Incomplete SCI or injury to the conus medullaris or cauda equina may cause mixed bladder dysfunction of various types.

Detrusor-sphincter dyssynergia

Detrusor-sphincter dyssynergia occurs when the detrusor and the external urethral sphincter contract at the same time, or when the sphincter is spastic. This condition can be detected by combined cystometrogram and electromyogram, or by video examination of voiding under fluoroscopy (video-urodynamics).

Urological complications of SCI

Autonomic Dysreflexia

As previously mentioned, autonomic dysreflexia may occur in SCI above T6. Bladder problems are the cause of 75% of episodes of autonomic dysreflexia, which may be precipitated by bladder overdistention due to catheter blockage, by bladder spasticity, urinary tract infection, and by urological instrumentation. It is important to perform catheterization with gentle technique. The installation of lidocaine into the urethra before catheterization may help. Recurrent episodes of autonomic dysreflexia precipitated by detrusor-sphincter dyssynergia is one indication for sphincterotomy.

Urinary tract infections

Symptomatic urinary tract infection is common in SCI patients, with an estimated annual incidence of 20%. Urinary tract infection may be difficult to diagnose in SCI patients. As mentioned, UTIs may present as autonomic dysreflexia or increased spasticity. Some patients use tests for urine leukocyte esterase at home to screen for infection. Urine culture colony counts of 10^4/ml should be considered significant in patients using intermittent catheterization, condom catheters, or indwelling bladder catheters. A change in urinary flora may have more clinical significance than the colony count. However, treatment of asymptomatic bacteriuria without pyuria (>10-20 WBC/hpf) is not recommended unless culture shows a urease-splitting organism such as Proteus, which predisposes to stones. Chronic prophylactic urinary antiseptics are not generally recommended. Fever usually indicates an upper urinary tract infection. In men with indwelling catheters, periurethral gland abscesses, epididymitis, prostatitis, seminal vesiculitis, and orchitis also occur. Recurrent infections should prompt an evaluation for abnormal bladder physiology or an anatomic problem.

Other complications

Intermittent catheterization may cause urethral trauma such as false passage. Chronic urethral catheters are complicated by catheter blockage, erosion, stricture, and diverticula arising from blockage of the periurethral glands. In women, chronic urethral catheterization may lead to incontinence due to stretching of the sphincter. Relatively small urethral catheters (14 F) should be used.

SCI patients are at risk of kidney and bladder stones. There is hypermobilization of calcium for several weeks after injury. However, stasis and chronic infection, especially with urease-producing organisms, are responsible for most stones.

The risk of bladder cancer is increased in SCI patients who have had chronic indwelling catheters for 10 years or more. The incidence is estimated at 2%. Yearly surveillance with bladder cytology and/or cystoscopy is recommended.

With aging, various changes may affect urinary function. Deterioration of spinal cord function (syringomyelia, for example) may affect bladder function. Development of prostatic hypertrophy may lead to bladder obstruction. Chronic recurrent infection and/or vesico-ureteral reflux may lead to renal failure, although this is uncommon now. Medications added because of other, age-related diseases (such as beta blockers, alpha blockers, and calcium channel blockers) may affect bladder function.

Bladder care in SCI

The goals of bladder care in SCI are to ensure adequate bladder reservoir capacity, ensure adequate emptying with minimal residual, prevent bladder overdistention, and avoid high bladder pressures. Low capacity leads to frequent voiding and/or high pressures. High pressures cause vesico-ureteral reflux and damage to the upper tracts. Chronic overstretching of bladder wall leads

to failure of bladder muscle or fibrosis. A bladder capacity of 400-500 ml and a residual of <20% of bladder capacity are desirable. The regimen should be socially acceptable and be one that the patient can manage independently or teach others to perform.

Evaluation of bladder function after SCI

After acute SCI, each patient must be evaluated individually when bladder function has stabilized, which may take up to 6 months. Cystometrograms measure bladder pressure during filling and during voiding. The ability of the bladder to store appropriate amounts of urine at acceptable pressures is determined, as is the ability of the bladder to empty without generating pressures which may lead to vesico-ureteral reflux (>40 cm H_2O). Simultaneous electromyography of the external sphincter can determine whether detrusor-sphincter dyssynergia is present. Contrast cystography can show signs of bladder spasticity, such as trabeculation or vesico-ureteral reflux.

Methods of bladder care

There are a number of different methods to manage bladder dysfunction due to SCI. The methods chosen depend on individual bladder physiology, the physical abilities of the patient or caregiver, and the preferences of the patient.

Intermittent catheterization has markedly reduced infection rates and other complications associated with indwelling bladder catheters. Patients do clean (not sterile) catheterization about every 4 to 6 hours. Sterile technique should be used in the hospital to prevent contamination by resistant organisms. Fluid intake must be regulated to avoid extremes in bladder filling. Self catheterization requires a moderate level of upper extremity function. It may be impossible for those with high cervical lesions, and for anatomic reasons is more difficult for women than men. Infection rates are higher if intermittent catheterization is performed by caregivers rather than the patient. Urethral obstruction from stricture, benign prostatic hypertrophy, or sphincter spasm may cause difficulty. If the detrusor is very spastic, reflex voiding may occur between catheterizations. If the sphincter is flaccid, incontinence will be a problem.

Trigger voiding can be used when the patient has residual function of the reflex voiding center and a relatively normal sphincter. Stimulation (tapping, rubbing, or pinching) of trigger points on the lower abdomen or inner thigh elicits a strong reflex contraction of the bladder and initiates voiding. Gentle digital dilatation of the rectal sphincter may relax the external urethral sphincter to allow more complete bladder emptying.

Reflex voiding into an external collection device such as a condom catheter can be used by patients fortunate to have balanced bladder physiology and an intact sacral reflex voiding center. This

is also necessary when the sphincter does not maintain continence, as when a sphincterotomy has been done to overcome detrusor-sphincter dyssynergia. These patients must be re-evaluated periodically to make sure that residual volumes or voiding pressures are acceptable.

Straining to void can be used by patients with lower SCI who have strong intact abdominal muscles and flaccid bladders and sphincters. This should not be attempted by patients with spastic bladders or outlet obstruction, as it will lead to vesico-ureteral reflux. Manual expression (Crede maneuver) may provoke vesico-ureteral reflux, and should only be done if a voiding cystourethrogram has proved that there is no reflux during the maneuver. Straining to void may leave unacceptably high residual volumes.

Chronic indwelling catheterization may be necessary for a variety of reasons. It should be avoided when possible because of complications such as urinary tract infections, bladder stones, mechanical problems and bladder cancer. Suprapubic catheterization may be preferable to chronic urethral catheterization, particularly in women. It may be used temporarily for treatment of urethral fistula or infection such as prostatitis or epididymitis.

Pharmacological therapy is most often used to improve bladder spasticity resulting in low bladder capacity. Ditropan is an anticholinergic which inhibits bladder contractility. In doses of 10-40 mg/day it facilitates bladder filling. Detrusor-sphincter dyssynergia may be relieved by alpha adrenergic antagonists (terazosin in doses of 2-10 mg/day, for example), which relax the internal sphincter and decrease detrusor pressures, or by baclofen, which reduces external sphincter spasticity.

Surgical intervention is most often considered when detrusor-sphincter dyssynergia has not responded to medications and if intermittent catheterization has failed. Transurethral sphincterotomy is the most common procedure. Recently, stenting has also been found to be effective. Sphincterotomy may preserve renal function in the long run, but incontinence or loss of erectile function may result. For severely spastic or fibrotic bladders, bladder augmentation may be necessary to provide adequate storage function and protect the kidneys from vesico-ureteral reflux. Bladder augmentation is performed with a pouch of ileum or colon separated from the gastrointestinal tract. Ureteral diversion with an ileal pouch (Bricker procedure), is less commonly done now than in the past because of long-term complications.

Urological followup of SCI patients

Bladder function may change in SCI patients. Urological evaluation is recommended every 12-24 months. This should include creatinine clearance, renal sonography, cystometrogram, and voiding contrast cystography to evaluate bladder configuration and

the integrity of the vesico-ureteral junction. An intravenous pyelogram is not necessary unless stone disease or some other anatomic abnormality is suspected. As mentioned, periodic bladder cytology and cystoscopy should be done in patients with chronic bladder catheters.

Sexual function

Complete SCI results in the loss of conscious genital sensation, the orgasmic response, and, in men, loss of normal ejaculation of sperm, although penile erection and vaginal lubrication are often preserved. Sexual function is more likely to be intact after incomplete SCI, but is variable. Despite these and other disabilities, the majority of SCI patients continue to be sexually active, although with less frequency than before injury. Sexuality is important to many SCI patients, and health care workers should provide opportunities for patients to discuss concerns and work out problems in this area, despite the discomfort that the topic may present.

Normal sexual function requires spinal cord sympathetic input from segments T10-L1, an intact sacral reflex center at S2-4, as well as intact lower motor neurons. The functions of erection (and vaginal lubrication) and ejaculation (and comparable female response) are controlled separately.

With injury above the sacral cord, the genital sacral reflex center is intact, and reflex erections occur in response to touch, but not to mental stimuli. Ejaculation is not possible with ordinary stimulation. Complete SCI involving the sacral reflex center at S2-4 prevents reflex erection with touch stimulation, but erection with mental stimulation is possible through sympathetic output originating in T10-L1. Reflex mental erections in SCI men are not as complete as reflex touch erections, and are often difficult to sustain. Complete lesions at L2-S1 may allow reflex erections with both touch and mental stimuli.

Erectile dysfunction may be treated with various techniques, including intracavernous papaverine injection, although men with SCI are more susceptible to abnormally prolonged erections with this technique. Penile prostheses have been used successfully in SCI men, but complications such as erosion are more common. Physicians should be aware that touch reflex erection may be impaired with antispasmodics.

Fertility in SCI men is decreased by inability to ejaculate normally and by a low percentage of fertile sperm. If the L2-S1 segments are intact, vibratory stimulation of the glans produces ejaculation in the majority of patients, as does transrectal electrical stimulation of nerves on the posterior surface of the prostate (electroejaculation). Side effects include autonomic dysreflexia in SCI above T5-6 and increased spasticity.

Women with SCI recover normal menstrual function, and have fertility and miscarriage rates not much different from normal. The risk of thromboembolism with birth control pills may be higher, and the use of IUDs may be hazardous. Pregnancy poses special problems, including urinary tract infections, difficulties with mobility, and

pressure sores. Labor may be painless, and close monitoring for dilatation of the cervix is necessary. Autonomic dysreflexia may occur during labor with SCI above T4-6, and require spinal or epidural block for treatment.

PRESSURE SORES

SCI patients are susceptible to pressure sores for several reasons. Irreversible tissue changes may occur with two hours or less of unrelieved pressure on the skin. Loss of sensation due to SCI leads to decreased stimulation for normal weight shifting, as well as unrecognized trauma such as burns. Shearing forces and friction result from transfers or abnormal postures.

Prevention of skin breakdown in SCI patients consists primarily of periodic pressure relief. Patients in wheelchairs should do posture shifts every 15 minutes. Patients in bed must shift position every 2-4 hours, even during the night. The use of special mattresses and cushions is no substitute for position changes. Transfers should be done gently to avoid shearing trauma. Friction can be created by poor position in the wheelchair or when the head of the bed is elevated. A careful skin examination should be done twice daily. Skin should be kept dry to avoid maceration. Problems interfering with proper posture or posture shifts, such as excessive spasticity or contractures, should be attended to promptly. Transparent film dressing may be used in areas of skin at risk from shear and friction.

Risk factors for pressure ulcers

•Depression	•Immobilization
•Malnutrition	•Incontinence
•Smoking	•Loss of caretaker
•Severe spasticity	•Contractures
•Heterotopic ossif.	•Fractures

Characteristics of early pressure areas should be familiar to health care providers. These include persistent redness, localized warmth, hardening, edema or blistering.

Treatment of pressure sores

The first principle of pressure sore treatment is removal of pressure. Without this all other treatments are futile.

Necrotic tissue requires debridement. If extensive tissue necrosis is present, debridement must initially be surgical, after which gentle treatment with wet-to-dry dressings using gauze and normal saline is best. Hydrogen peroxide, povidone iodine, acetic acid, and antiseptic detergents cause damage to granulation tissue, and delay healing. The best treatment of superficial infection is proper debridement and cleansing, rather than topical antiseptics. Well debrided, clean pressure sores heal better if covered with semiocclusive or occlusive dressings which prevent drying. Even

with optimal care, no more than 1 mm of healing per day can be expected. Malnutrition is associated with poor healing and should be corrected. Management of bowel and bladder incontinence may be important. Nonhealing ulcers may be associated with osteomyelitis. A few require surgical treatment, such as myocutaneous flaps.

Pressure ulcer classification system
The following staging system for pressure ulcers has been recommended by the National Pressure Ulcer Advisory Panel.

Stage	Characteristics
I	Nonblanching erythema of intact skin
II	Partial skin thickness loss involving epidermis and/or dermis (abrasion, blister, shallow crater)
III	Full thickness skin loss involving damage or necrosis of subcutaneous tissue that may extend down to, but not through, underlying fascia (deep crater without undermining)
IV	Full thickness skin loss with extensive destruction, tissue necrosis, or damage to muscle, bone, or supporting structures (eg tendon or joint)

CHRONIC PAIN

Estimates of the prevalence of severe and/or disabling pain in SCI patients have ranged from 18 to 63%, with a mean of about 40%. Chronic pain commonly interferes with activities of daily living or with work in SCI. It is often associated with depression and other psychological distress. For some SCI patients chronic pain is their dominant concern, despite other disabilities. Several types of chronic pain occur, some of which are unique to SCI.

- Musculoskeletal
  - -Spinal arthritis
  - -Overuse syndromes
  - -Spasticity
- Radicular
  - -Central
  - -Peripheral
- Syringomyelia
- Deafferentation
  - -Segmental
  - -Phantom

Musculoskeletal pain often occurs at the level of vertebral injury, and may be from nonunion of spinal fracture, or from damaged facet joints. It is usually a dull aching discomfort aggravated by activities. Another source of mechanical pain is that of overuse syndromes of the upper extremities, such as tendinitis, in patients who are active in wheelchairs. Muscle pain due to spasticity is a frequent problem, and is often only partially relieved by baclofen or diazepam. Radicular nerve pain arises from damage to

the peripheral nerve or nerve root at the site of spinal fractures, particularly with cauda equina injury. It is usually stabbing or shooting, although it may become continuous and be aggravated by touch or pressure. Nerve root decompression may help, but radicular pain may be difficult to treat. Peripheral nerve root compression pain syndromes involving the ulnar and median nerve are common in patients who use wheelchairs or crutches.

Pain may be a symptom of syringomyelia. Usually located in the shoulders or upper extremities, it often has a radicular distribution. As mentioned before, it is very important to consider this diagnosis early, since progression is dangerous, and treatment is effective.

Deafferentation or central pain arises from the spinal cord itself. The physiology of the cord is disturbed so that pain is generated long after the peripheral sources of acute pain have resolved. Healing in the damaged cord results in disorganized axonal regeneration. Dorsal horn interneurons mediating pain (T cells) may generate pain sensations in response to a variety of afferent input which would not ordinarily be painful. There is loss of normal descending pathways which inhibit pain. Spinal cord interneurons mediating pain sensation may also begin to fire autonomously and repetitively. This has been described as a convulsive syndrome of the spinal cord. Segmental deafferentation pain occurs at the level of injury, whereas phantom pain is perceived below the level of injury, in the area of sensory loss. The pain may be experienced in a number of forms. An uncommon variant may occur in the perineum in which there is a sensation of a large mass in the rectum or of sitting on a hot fire. A sensation of distorted position of the legs may occur. Most often the pain is continuous, without aggravating or relieving factors, but may be aggravated by distended bladder or bowel, urinary tract infection, increases in spasticity, pressure sores, or cutaneous stimulation at the level of the injury.

Management of chronic pain in SCI

Management of chronic pain in SCI must address both psychosocial and physiological components. Severity of chronic pain and disability caused by it are more closely associated with psychosocial problems than with the cause of the pain. Therefore, it is important to promote good social and psychological adjustment as part of any program to treat chronic pain.

Pharmacological treatment

Antidepressants are commonly used for neuropathic pain. The analgesic benefit does not seem to be related to the antidepressant effect, as maximum pain relief usually occurs within 2 weeks and at lower doses than customarily required for depression. The tertiary amines, such as amitriptyline, are used, starting with the lowest dose and increasing about every third day.

Anticonvulsants, in particular carbamazepine and phenytoin, may have a beneficial effect on chronic SCI pain by stabilizing hyperactive pain-mediating neurons. Both of these medications require careful monitoring for adverse effects. Neuroleptics, most commonly fluphenazine, are also occasionally used for neuropathic

pain, particularly in combination with an antidepressant. Narcotics are used only very reluctantly by most physicians treating chronic pain not associated with cancer, but there is some experience with the use of methadone in the setting of a multidisciplinary pain treatment program.

Surgical and ablative treatments

Nerve blocks are not effective for chronic deafferentation pain, but may be useful diagnostically to distinguish it from peripheral pain. Neuroablative procedures such as sympathectomy, dorsal nerve root section, and cordotomy have been largely abandoned as ineffective. Recently selective destruction of a small zone of afferent interneurons in the dorsal root entry zone (DREZ) has produced benefit in 60% to 80% of SCI patients with segmental or phantom leg pain originating at the thoracolumbar level.

Other methods of pain control

Physical therapy, such as range of motion exercises, stretching, and education regarding proper techniques for transfer and wheelchair use are important in treating pain resulting from spasticity, contractures, and overuse syndromes.

SKELETAL FUNCTION

Bone loss and fractures

Immobility after SCI leads to acute bone loss. Hypercalciuria occurs in the first year, and 5-10% of patients will develop renal stones. Bone density has been found to decrease by about 30% in the legs, but vertebral density remains normal. Tetraplegics have more bone loss than paraplegics. Fracture rates after SCI are increased because of decreased bone density but also by increased risk of trauma, such as falls during transfers or from the wheelchair. Leg fractures may occur with minimal trauma, and produce minimal symptoms. Treatment of fractures below the level of SCI is complicated by the risk of casting a limb without sensation, and by a high frequency of malunion.

Degenerative disease of the spine

SCI patients may have an increased frequency of degenerative disease of the spine close to the level of injury. This is a common cause of chronic pain. Repeated spinal torquing, especially during transfers, may cause progressive degeneration in unstable segments. If these segments are painless, a Charcot spine may result. Complications include instability during sitting, gibbus deformity, and conversion of spastic paralysis to flaccid paralysis.

Overuse and abnormal stress syndromes

SCI patients suffer increased stress on the upper extremities during use of the wheelchair, during raises, and during transfers. About 50% of SCI patients who use wheelchairs have shoulder pain due to conditions such as rotator cuff tears and subacromial bursitis. Elbow and wrist problems such as pain or carpal tunnel

syndrome occur in 30-50%. For those with tetraplegia or high paraplegia, even minor upper extremity problems result in major impairments of mobility and independence. Some of these problems may be helped by the use of electric wheelchairs and transfer boards. In paraplegics who walk with orthoses and crutches, knee problems may occur. The knee joint tends to be in hyperextension because of spasticity, which places abnormal forces on it. Artificial joints have been placed in SCI patients.

Heterotopic ossification

Heterotopic ossification (HO), or heterotopic bone formation, is the appearance of new bone within the soft tissue surrounding peripheral joints. The cause is unknown, but complete SCI, pressure sores, increased spasticity, or joint trauma all increase the risk. About 15% of SCI patients will develop HO, usually within the first months to one year after injury. It is most common in the legs around the hips or knees. Of those who develop HO, about 5-10% will have severe limitation of joint motion. Symptoms of HO include swelling, redness, heat, decreased range of motion, and sometimes pain. The differential includes deep venous thrombosis, local trauma or fracture, cellulitis, joint infection, and hematoma. Severe HO may require surgical removal of bone. If the heterotopic bone is not seen on plain x-rays, a triple phase bone scan will help with the diagnosis. Treatment with disodium etidronate has been used to reduce the risk of recurrence after surgery.

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SPINAL CORD INJURY ORGANIZATIONS

National Spinal Cord Injury Hotline: 1-800-5263456

American Paraplegia Society
American Association of Spinal Cord Injury Nurses
American Association of Spinal Cord Injury Psychologists and Social Workers
75-20 Astoria Blvd.
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Paralyzed Veterans of American
801 18th Street NW
Washington, DC 20006

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