



Acute Spinal Cord Injury

27 February 2003

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I receive many phone calls and emails from people and families with spinal cord injury. It is better today compared to 1977 when I took care of my first patient with spinal cord injury and had to tell the family that there was nothing that we could do. However, every call is still difficult. What do I say to the families?

- **Focus on solvable problems.** Make sure that methylprednisolone is given within 8 hours after injury (this drug may improve recovery by 20%). Find the best and most experienced surgeon. If the spinal cord is compressed, make sure that it is decompressed as soon as possible. Prevent complications by insisting on aggressive care of lung, bladder, and skin. Start rehabilitation as soon as possible.
- **Recovery is the rule and not the exception in spinal cord injury.** Most people recover some function after spinal cord injury. People with “complete” injuries will recover an average of 8% of function they had lost within 6–12 months, compared to 21% if they received methylprednisolone. People with “incomplete” injuries recover an average of 59% of lost function compared to 75% if they received methylprednisolone. Recovery takes a long time and work. Many people recover function for 2 or more years after spinal cord injury.
- **Do not give up hope.** Most scientists believe that it is not a matter of if but a matter of when therapies will restore function in spinal cord injury. Several clinical trials are testing therapies for the first few weeks after injury. Carefully weigh the potential risks and benefits because participating in such trials. Many clinical trials are and will be assessing therapies aimed at restoring chronic spinal cord injury. The therapies will get better over time.

What to ask your doctor?

Families and friends often don't even know what questions to ask their doctors. Here are some questions to ask during the first hours after injury:

- ***Was methylprednisolone given?*** This is the high-dose steroid (30 mg/kg intravenous bolus followed by 5.4 mg/kg/hour for 23 hours if it is started within 3 hours and for 47 hours if between 3 to 8 hours after injury). It should not be started more than 8 hours after injury. Several clinical trials have shown that this treatment improves recovery by about 20% when given within 8 hours after injury. While methylprednisolone is not a cure, every little bit of recovery counts. As long as the treatment time is limited, complications are minimal.
- ***What is the level and severity of spinal cord injury?*** The consequences of spinal cord injury depend on the level and severity of injury. Levels of injury are usually determined from where the spinal column shows a fracture. The injury causes loss of sensation and voluntary movement below the injury site. If the person has any motor or sensory function below the injury level, the likelihood of substantial recovery is high. But even people with so-called “complete” spinal cord injuries will recover some function.
- ***Has the spinal cord been decompressed?*** The spinal cord injury usually results from fracture of vertebral bones that then compress the spinal cord. Many studies have shown the compression of the cord increases spinal cord damage and reduces functional recovery. If the fracture is in the neck or cervical segments, the vertebral column often can be straightened out by traction. Fractures of vertebral segments in the chest are more difficult to straighten out by traction. Surgery may be necessary to decompress the spinal cord. Decompression should be done as soon as possible.
- ***Has anticoagulation been started?*** A serious complication of spinal cord injury is formation of clots in the legs that can migrate to the lungs. Clot formation can be prevented by anticoagulants such as heparin to prevent blood clots. It is also possible to place a filter in the large vein going to the heart to catch any clots. Both may be necessary in cases of severe spinal cord injuries.
- ***Pulmonary, bladder, and skin care?*** Aggressive care is necessary to prevent other complications of spinal cord injury. If the injury involves the neck or chest, breathing and coughing may be compromised. Artificial respiration may be necessary and pneumonia may occur. Spinal cord injury paralyzes the bladder and a catheter must be placed in the bladder to ensure that urine is drained. Because spinal cord injury causes loss of sensation, lying in a particular position may cause skin sores called decubiti. Cushioning vulnerable areas and regular turning can prevent this.

Some frequently asked questions and answers

Families and friends often search the Internet and encounter a bewildering array of information that is often out of date and contradictory. Here are some commonly asked questions and quick answers:

- ***Will he/she recover?*** Recovery is the rule and not the exception after spinal cord injury. The probability of recovery is high, especially after “incomplete” spinal cord injury. Clinical trial data indicate that if a person had even slight sensation or movement below the injury site shortly after injury, they will recover an average of 59% of the function they lost and, if they receive high-dose methylprednisolone, they will recover an average of 75% of what they had lost. People admitted to hospital with no motor or sensory function below the injury site recover an average of 8% of the function they had lost but will recover an average of 21% if they received methylprednisolone.
- ***How long will recovery take?*** Recovery takes a long time. While most of the recovery should occur within 6 months, many people continue to recover function for a year or more. A recent [poll](#) of the CareCure Community suggests that 61% of the people with spinal cord injury on the site recovered function more than one year after injury. According to another [poll](#), 16–18% of people who are “complete” spinal cord injury will recover additional function 3 or more years after injury. A recent study detailed how Christopher Reeve continued to recover function for over 7 years after his injury. So, recovery frequently continues for years after injury.
- ***What experimental clinical therapies are available?*** Several clinical trials are assessing therapies applied within 2 weeks after injury. These include implantation of activated macrophages (which may help repair the injured cord), alternating currents (to stimulate regeneration), and AIT-082 (a drug that may stimulate growth factors and stem cell proliferation). Two of the trials are limited to people who have thoracic spinal cord injury (activated macrophages and alternating current). Potential risk and benefits of the therapies must be carefully considered, including the risk of moving somebody to another center for clinical trial.
- ***Do therapies have to be applied shortly after injury?*** Several therapies are aimed at restoring function to people more than a year after injury. These include 4-aminopyridine (a drug that increases the excitability of demyelinated axons), porcine fetal stem cells transplants (stem cells from pigs), olfactory ensheathing glial transplants (cells from the nasal mucosa or from olfactory bulbs of aborted fetuses). Other therapies for chronic spinal cord injury are likely to go into clinical trial in the coming year, including drugs and chemicals that block growth inhibitors. Thus, there will be plenty of opportunity to be involved in clinical trials at later periods.

What is the spinal cord?

While this may seem to be a silly question, it is really not so trivial. Until people get spinal cord injury or know somebody who is spinal-injured, most pay little attention to their spinal cords. Most people don't know the different parts of the spinal cord, what each part does, and how the spinal cord transmits sensory and motor information. Many think that the spinal cord conducts information like a telephone wire and that all that is necessary to fix the spinal cord is to reconnect it. Some people don't know what the spinal cord is and mistakenly believe that the spinal cord is the vertebral column. While almost everybody knows that spinal cord injury causes paralysis, many are not aware that the spinal cord also controls the bladder and bowel, sexual function, blood pressure, skin blood flow, sweating, and temperature regulation.

The spinal cord connects the brain to the body. The spinal cord resides in the a bony spinal or vertebral column that has 24 segments. Seven vertebra in the neck are called cervical (C1–C7), twelve chest or thoracic (T1–T12) segments form the rib cage, five segments for the lower back or lumbar (L1–L5), and five segments form the tail or sacral (S1–S5) vertebra. The vertebral bodies are in the front of the spinal column. Spinal discs are located between the vertebral bodies. The front of the spinal cord is referred to as anterior while the back is referred to as posterior. The sides of the spinal cord are called lateral. Note that in animals that walk on four legs, posterior is dorsal and anterior is ventral.

Each segment has four spinal roots (left and right, posterior and anterior) that send and receive information from each side of the body. Posterior roots receive sensation while anterior roots send motor signals to muscles. For example, the C1–C3 segments send and receive information from the back of the head and neck, C4 covers the shoulder and deltoid muscles, C5 the biceps, C6 the wrist extensors, C7 the triceps, C8 the wrist flexors, and T1 the intrinsic muscles of the hand. The spinal roots leave the vertebral column between the bony segments through openings in the vertebral column called foramina. Note that there are only 7 cervical vertebra but 8 sets of cervical roots. This is because the C1 roots are between the skull and C1.

The spinal cord is shorter than the vertebral column and occupies the spinal canal from the C1 to L1 vertebral levels. In general, the bony vertebral segments are lower than the spinal cord levels. The spinal roots exit through the spinal column through openings between vertebral segments called foramina. The spinal cord stops just below the L1 vertebral level and only spinal roots are present from L1 to S5 vertebral spinal column. The end of the cord is called the conus. Spinal roots that go between the conus and lower lumbosacral vertebra are collectively called the cauda equina because they resemble a horse's tail.

How does the spinal cord work?

Neurons in the brain, spinal cord, and peripheral nerve send axons or nerve fibers up and down the spinal cord in spinal tracts. These spinal tracts are called white matter because the axons are coated with a membrane called myelin and myelin appears white and hence is called white matter. White matter is usually situated close to the surface of the spinal cord, arranged into several columns called the anterior, posterior, and lateral columns. The spinal cord contains neurons located in the middle part of the spinal cord. This part of the spinal cord is butterfly shaped, is grayish in color, and hence is called gray matter. The gray matter is most prominent in the vertebral segments that connect to the arms and legs, respectively called the cervical and lumbosacral enlargements.

The spinal cord transmits sensory information and movement control to and from the brain, as well as breathing, bladder, bowel, sweating, blood pressure, sexual, and other essential functions of the body. The spinal cord contains much of the neuronal circuitry for reflexes that control all these functions. Over 20 million axons or nerve fibers ascend and descend in the human spinal cord to and from the brain. These axons are organized into spinal tracts, usually named according to their source and destination. For example, the spinal tract that sends axons from the cerebral cortex to the spinal cord is called the corticospinal tract. Likewise, the tract that sends axons from the red nucleus in the midbrain to the spinal cord is called the rubrospinal tract. The sensory tract that transmits pain and temperature sensation from the spinal cord to the thalamus is called the spinothalamic tract. Some tracts, however, are called by their position. For example, the posterior column transmits sensory information from the spinal roots to the brainstem.

Neurons in the spinal cord send axons to muscles and are called motoneurons while neurons that send axons to other neurons are called interneurons. Motoneurons and interneurons receive information from descending axons and sensory axons. When you activate sensory input to the spinal cord by tapping a tendon, the activity turns on motoneurons that cause the muscle of that tendon to contract. This is called a monosynaptic reflex. To signal the muscles to move, the brain sends information directly to motoneurons or indirectly through interneurons that can either excite or inhibit other neurons.

Sensory neurons send axons from the spinal cord to the brain. Some sensory axons go from peripheral nerve neurons in posterior sensory ganglia located just outside of the spinal column. Posterior sensory ganglion neurons send an T-shaped axon to the body where it collects information like touch and movement while the other end goes into the spinal cord and branches. One branch goes into the gray matter where it activates motoneurons and the other end goes up the posterior column all the way to the brainstem.

What is spinal cord injury?

Many misconceptions abound concerning spinal cord injury. For example, many people believe that the whole spinal cord below the injury site dies after injury. Others think that the injured spinal cord is like a cut telephone wire and it is just a matter of reconnecting the cut ends. Some people think that the vertebral column is the spinal cord. Even doctors have misleading and inaccurate ideas about spinal cord injury. For example, many doctors casually use the word “transection” to refer to the severely injured spinal cord. The word should be applied to the extremely rare situation that the spinal cord has been cut and the cut ends are separated.

Spinal cord injury usually results from trauma to the vertebral column and the bone then compresses the spinal cord. Spinal cord injury can occur without obvious vertebral fractures and vice versa. It can also result from loss of blood flow to the spinal cord that sometimes can occur from damage to the aorta or the large blood vessel from the heart. Many people may have had mild spinal cord injury without thinking that it is spinal cord injury. For example, over a million people per year get “whiplash” in car accidents, have neck pain, weakness, and sensory loss that many last days or even months. Athletes who play football or other contact sports often suffer a transient loss of function that they call a “stinger”, i.e. paralysis and sensory loss for minutes or even hours. There is a condition called transverse myelitis where a person gets spinal cord injury without any obvious trauma.

Spinal cord injuries are usually defined by vertebral level and neurological level, as well as severity. Vertebral levels are indicated by which bony vertebra have been fractured or show damage. Multiple bony vertebra may be injured. For example, an injury that causes the C5 vertebra to slip forward relative to C4 may be called a C4/C5 injury because it causes compression of the C4 and C5 spinal cord. Note that the spinal cord at each vertebral level may not be the same as the vertebral level. For example, as pointed out above, the spinal cord is shorter than the vertebral column and ends at the L1 vertebral level even though the spinal roots continue downward and exit between the appropriate vertebral segments.

For many years, there was no standardized way of referring to spinal cord injury levels. Surgeons generally referred to the level of injury by the vertebra that are damaged. Neurologists and physiatrists, however, tend to refer to the level of spinal cord injury based on the neurological loss. Neurologists tend to identify the level of injury as the first segmental level that shows sensory or motor loss. In contrast, physiatrists identify injury level from lowest level that has normal motor and sensory function. In 1990, the American Spinal Cord Injury Association proposed a uniform classification system for spinal cord injury.

How is spinal cord injury classified?

Spinal cord injuries are classified by the American Spinal Injury Association (ASIA) into five categories, defined in Table 1. Motor level is defined as the level at which the key muscle innervated by the segment has at least 3/5 of its normal strength. Sensory level is defined as the lowest spinal cord level that still has normal pinprick and touch sensation. If there is a spinal cord level below which there is no voluntary motor or conscious sensory function, the person is called a “complete” spinal cord injury. Since the S5 is the lowest spinal cord level that innervates the anal sphincter, a person that has no voluntary anal sphincter control or sensation is defined as a “complete” spinal cord injury. A person who has any anal control or sensation is an “incomplete” spinal cord injury. Some people may have a “complete” spinal cord injury but still has preserved motor or sensory function between the injury level and S5. This is called the “zone of partial preservation”. Usually, the spinal cord injury level and severity is classified between 72 hours and 7 days after injury. Note that some people have neurological loss at a given spinal cord level but partially preserved function for several or even many segments; this is called the zone of partial preservation (ZPP).

Table 1: Neurological Classification of Spinal Cord Injury

A	No motor or sensory function preserved in the lowest sacral segments
B	Sensory but not motor function preserved in the lowest sacral segments
C	Motor function present below the injury but most key muscles are $<3/5$
D	Motor function present below the injury but most key muscles are $\geq 3/5$
E	Motor and sensory function normal in key muscles and dermatomes

Some patterns of spinal cord injury have special names.

- “Central Cord Syndrome” is when arm function is affected more than the legs. This paradoxical condition is attributed to greater damage of inner spinal cord tracts and sparing of spinal tracts close to the cord surface where motor and sensory fibers to and from the legs are located. However, recent studies of people with central cord syndrome suggest that the syndrome may be associated with destruction of the lateral spinal tracts.
- “Brown-Sequard Syndrome” refers to Injuries limited to one side of the cord. People may have weakness and loss of touch sense in the leg on the side of the injury but loss of pain and temperature sensation in the other side.
- “Anterior Cord Syndrome” refers to the condition when sensation is preserved but motor function is absent while “Posterior Cord Syndrome” refers to the condition when motor function is preserved in the absence of sensation.
- “Conus Medullaire” refers to injury of the conus or lower tip of the spinal cord. This damages the lower lumbar and sacral spinal cord segments.
- “Cauda Equina Injury” refers to the condition when the damage is limited to the spinal roots below L1.

How is acute spinal cord injury treated?

Acute spinal cord injury refers to hours or days after spinal cord injury during which continued deterioration or tissue damage occurs. Shortly after an injury, the spinal cord often does not appear to be severely damaged even though there may be immediate functional loss. The injury initiates a cascade of chemical and cellular responses that contribute to further tissue damage, including inflammation, free radicals, and swelling (edema). The spinal cord may be compressed during this period and animal studies have shown that compression or decreased perfusion (blood flow) of the spinal cord aggravate the injury. These causes of progressive tissue damage can and should be relieved as rapidly as possible. The goal of acute spinal cord injury care is to stabilize the spinal cord to prevent further damage, save as much tissue as possible, and prevent complications of spinal cord injury.

- **Emergency management.** The first objective of emergency management of spinal cord injury is to establish ABC (airway, breathing, and circulation). The spine must be immobilized to prevent further injury. The patient must be transported rapidly to the nearest medical center, preferably a Level 1 Trauma Center. If blood pressure is low, fluid and drug therapies must be given to maintain blood flow in the spinal cord. In cervical spinal cord injuries that affect breathing, ventilatory support may be necessary. A foley catheter is usually placed in the bladder to drain urine.
- **Methylprednisolone therapy.** At or *en route* to the hospital, the patient should receive intravenous high-dose methylprednisolone (30 mg/kg bolus followed by 5.4 mg/kg/hour for 23 hours). This steroid therapy improves neurological recovery by about 20% compared to untreated controls. If the methylprednisolone is started between 3–8 hours after injury, the infusion should be extended to 48 hours. If the methylprednisolone cannot be started within 8 hours, it should not be given since clinical trials suggest that delayed therapy beyond 8 hours does not result in improved functional recovery.
- **Decompression of the spinal cord.** If the spinal cord is compressed by bone, every effort must be made to decompress the cord as soon as possible. Cervical spinal injuries can often be decompressed by traction of the spinal column to realign the vertebral bodies. However, thoracic and lumbosacral spinal fractures often cannot be decompressed by traction. In such cases, surgery may be necessary to decompress the cord or spinal roots. Since decompression of thoracic or lumbosacral spinal cord may require opening the chest cavity or retroperitoneal space, the surgery may require a team of surgeons and may be delayed for several days. Surgeons sometimes take their time with spinal cord injuries that result in complete loss of neurological function below the injury site. However, I believe that complete spinal cord injury should be treated as aggressively as incomplete spinal cord injuries.

What is spasticity and neuropathic pain?

Spinal cord injury disconnects the brain from the spinal cord below the injury site. The spinal cord below the injury site does not die unless it has been damaged by loss of blood flow (ischemia). The lower spinal cord often becomes hyperactive because spinal cord injury interrupts not only excitatory but also inhibitory connections to the cord. The spinal cord above the injury site may also become hyperactive, generating abnormal sensations.

- ***Spasticity and spasms.*** Spasticity is increased reflex activity in the lower spinal cord isolated from the brain by injury. The spinal cord contains the neurons that mediate simple muscle reflexes for feedback control, more complex reflexes such as the withdrawal reflex, anti-gravity reflexes for standing and postural control, and locomotor programs that mediate walking and running. Hyperactive reflexes may be present even when there is voluntary control of the muscle. In contrast, spasms are spontaneous or evoked movements of muscles, that involve multiple muscles. Spasms can occur in limbs that a person has little or no control of, and can be violent enough to throw a person out of a wheelchair. Pain, bladder infection, and irritation of the spinal cord can aggravate both spasticity and spasms. A drug called baclofen is often used to control spasticity. Baclofen usually does not prevent spasms unless such high doses are used that it causes weakness or flaccidity. Baclofen can be given directly to the spinal cord (intrathecally) to treat severe spasticity when maximum oral doses of 100–120 mg per day are insufficient. Several other drugs also suppress spasticity, including clonidine and tizanidine.

- ***Dysesthesia and pain.*** Abnormal sensations (dysesthesia) and neuropathic pain are the flip side of the coin to spasticity and spasms. When the spinal cord loses sensory input, spinal sensory neurons above the injury site may become hyperexcitable and transmit abnormal sensations and pain. The pain is akin to “phantom pain” after limb amputations and peripheral nerve injuries. Neuropathic pain typically is described as “burning” or “pressure”, involving areas of the body that have little or no sensation. It can also occur in deeper organs and the guts. Neuropathic pain may be associated with spasticity and spasms. For many years, doctors did not recognize neuropathic pain and treated it as psychogenic pain. However, it is real and several therapies are available for reducing neuropathic pain. For example, the tricyclic antidepressant amitriptyline (Elavil) may reduce dysesthesia. Some of the most promising therapies, interestingly, are drugs that are anti-epileptic. For example, gabapentin (Neurontin) is an anti-epileptic drug that has been reported to reduce neuropathic pain when given in very high doses. Some recent studies suggest that glutamate receptor blockers such as dextromethorphan and oral ketamine may be useful for refractory neuropathic pain.

Atrophy and Learned Non-Use

Due to loss of activity, muscle, bone, and skin atrophy occur after spinal cord injury. In addition, parts of the neural circuitry in the brain and spinal cord may turn off.

- **Atrophy.** When parts of the body are not used, they undergo atrophy. For example, muscles shrink, bones lose calcium and strength, and skin gets thinner. Activity of muscles, stress on bones, and contact with skin will prevent atrophy. Even passive movement will help prevent atrophy and fibrosis of muscle. Both spasticity and spasms prevent atrophy and indicate that the lower spinal cord is alive and active. They maintain muscle bulk and it is not a good idea to take so much anti-spasticity medication that the legs become flaccid (i.e. show no movement). Electrical stimulation (functional electrical stimulation) can be used to activate muscles to drive the legs pedal bicycles and prevent muscle atrophy. Weight bearing may prevent bone loss or osteoporosis while ambulation training on treadmills may reverse the osteoporosis. While a variety of drugs are now available for increasing calcium in bones, without exercise or stress on the bones, such drugs may increase the brittleness of bone without increasing the ability of the bones to withstand weight.

- **Learned non-use.** Muscles, bone, and skin are not the only parts of the body to undergo atrophy when they are not used. Neural circuits in the spinal cord may also turn off when they are not used. Spinal cord injury causes a prolonged period of inactivity in people. For example, a person may not walk for many months after a spinal cord injury and this may turn off neuronal circuits needed for walking. In the early 1990's, several groups reported that intensive ambulation training can restore independent locomotion to 50% or more of people who have some residual sensory or motor function but have never walked after spinal cord injury. Suspending a person over a treadmill and manually moving the legs until they start stepping on their own is one approach to ambulation training. Many rehabilitation centers around the world are studying these effects of weight-supported treadmill walking.

Preventing atrophy and reversing “learned non-use” are important goals of rehabilitation. Learned non-use may prevent recovery of function despite regenerative and remyelinating therapies. Some rehabilitation programs offer intensive motor training programs that can prevent or reverse learned non-use. Unfortunately, intensive and prolonged ambulation programs are very labor-intensive and consequently costly. Various clinical trials are being conducted to determine the optimal parameters for weight-supported ambulation, biofeedback, and other forms of motor training. Many rehabilitation centers in the United States have biofeedback, weight-supported ambulation, and functional electrical stimulation (FES) programs.

What happens to the bladder, bowel, and sexual function?

The spinal cord also carries “autonomic” signals that control blood pressure, blood flow, breathing, sweating, bowel, bladder, sexual, and other autonomic functions.

- ***Bladder Paralysis and Spasticity.*** Spinal cord injury paralyzes the bladder. The bladder must be catheterized to release urine. Indwelling catheters such as a foley catheter inserted through the urethra have a high risk of infections. Sterile intermittent catheterization is recommended but may be complicated by bladder spasticity or spontaneous contractions of the bladder. Bladder spasticity can push urine backward into the kidney and this may lead to kidney damage. A drug called Ditropan suppresses bladder spasticity but often have side effects such as dry mouth and eyes. Several alternative approaches are available, including cutting the bladder sphincter so that urine drains freely into a condom catheter but this approach is not suitable for women. Another approach that does not involve compromise of the urinary sphincter is placement of a suprapubic catheter or creation of a intestinal conduit from the abdominal wall to the bladder, i.e. a Mitrafanoff procedure or an ileal conduit.
- ***Bowel constipation and incontinence.*** The bowels usually operate without much voluntary control. However, spinal cord injury slows bowel activity and the transit time of food in the gut. People use a variety of gut stimulants and suppositories to stimulate bowel activity. Bowel incontinence is a problem, often restricting social activity and employment options. A common technique is to establish a bowel routine to empty the gut on a set schedule. While there are some surgical procedures that implant artificial sphincters, the success rates of such procedures are still limited. Finally, alterations in secretion patterns can lead to indigestion, appetite changes, nausea, gallbladder stones, and other problems, especially in people with cervical spinal cord injury.
- ***Erection and ejaculation.*** Most people assume that spinal cord injury eliminates the possibility of sexual function. However, this is not true for a majority of people with spinal cord injury. Penile erection is a reflex and many men are able to have erections after spinal cord injury unless the injury involves the lower spinal cord or roots that control erection. Recent studies suggest that Viagra works for people with spinal cord injury. Vibrators or electrical stimulation can be used to facilitate ejaculation. Due to sphincter spasticity or poor coordination of the bladder sphincters, the ejaculate often goes into the bladder rather than outward. However, with a combination of electroejaculation and semen collection, it is possible to collect ejaculates from nearly all males. While spinal cord injury may interfere with menstrual cycles, a vast majority of young women with spinal cord injury remain fertile and can conceive.

How does spinal cord injury affect the skin?

Spinal cord injury reduces or eliminates skin sensation in dermatomes below the injury site. Because people cannot feel or move, they may sit or lie for long periods of time on particular parts of their body. Pressure impedes blood flow in the skin. In addition, due to muscle atrophy, the normal padding of tissue that cushions the butt may be reduced or absent. Absence of sensation, loss of muscle padding, and long periods of pressure can lead to skin breakdown and development of pressure sores or decubiti. Decubiti are potentially life threatening but preventable. They must be treated aggressively if they develop.

Spinal cord injury impairs skin blood flow responses. Normally, skin responds to pressure, mechanical stimulation, or inflammation with increased blood flow. Loss of this response not only adds to the vulnerability of the skin to pressure sores but reduces the ability of the skin to repair decubiti. Thus, great care must be taken to prevent decubiti by shifting sitting positions and frequent turning. Special seats that distribute the pressure are used in wheelchairs to prevent sacral decubiti. Vulnerable areas such as the heels must be padded. If a decubitus develops, all pressure must be removed or the decubitus can progress to loss of skin and tissues to the point of exposing bone. The sores must be kept clean or they can become infected. Plastic surgery may be necessary to repair the decubitus.

Spinal cord injury also paralyzes sweating in dermatomes below the injury level. People with spinal cord injury must be very careful to maintain their body temperatures. In contrast to loss of sweating below the injury site, many people with spinal cord injury may have abnormal increases of sweating above the injury site, often in their upper torso and face. This is a form of autonomic hyperexcitability or spasticity. It is not unusual for people to sweat profusely on one side of the face and not the other. Such abnormal sweating responses may develop early or late after injury.

Spinal cord injury disables vascular responses that maintain blood pressure when a person sits or stands up. Blood vessels in the guts and legs normally constrict when a person stands up, in order to keep blood from pooling. Thus, when people with spinal cord injury sit up for the first time shortly after injury, their blood pressure may drop sharply. Postural hypotension may prevent a person with spinal cord injury from sitting or standing up during the first weeks after spinal cord injury. These vascular responses do recover over time but people must be initially be tilted gradually into the vertical position over the several weeks after spinal cord injury. Loss of vascular responses in the legs leads to a tendency for fluid to accumulate in the legs when people sit for long times. Such dependent edema can be prevented to some extent with stockings.

What is autonomic dysreflexia?

The autonomic nervous system often becomes hyperactive in people with spinal cord injury. Autonomic dysreflexia manifests in large increases in blood pressure (hypertension) with systolic pressures exceeding 200 mm Hg, slow (bradycardia) or fast heart rate (tachycardia), headaches, facial flushing, exuberant sweating, hyperthermia, stuffy nose, goose pimples, nausea, and other signs of autonomic hyperactivity. Called autonomic dysreflexia, these episodes may come on spontaneously or may be instigated by infection, pain, or other conditions that stimulate the autonomic nervous system. Severe episodes of autonomic dysreflexia may be life-threatening.

Emergency treatments of autonomic dysreflexia should initially focus on identifying potential causes that can be relieved. If the episode occurred during manipulation of the body, such as rectal stimulation, that activity of course should be stopped. The person should remain sitting and check for any blockage of bladder outflow. If necessary, place a foley catheter to drain the bladder. If the cause cannot be identified and eliminated, drugs can be used to relieve the symptoms. These include Procardia (a calcium channel blocker), nitroglycerin (a vasodilator), clonidine (alpha adrenergic agonist anti-hypertensive drug), or hydralazine (a vasodilator) to reduce blood pressure. People with spinal cord injury should carry a card with instructions to inexperienced emergency personnel.

Causes of autonomic dysreflexia may sometimes be masked by the spinal cord injury. For example, a bladder infection, kidney or bladder stones, bowel cramps, gallbladder stones, gastric ulcers, hemorrhoids, pressure sores, back pain, bone fractures, and many other potential causes may not be felt by an individual due to the spinal cord injury but may manifest in autonomic dysreflexia. Autonomic dysreflexia may result from heterotopic ossification (a condition where abnormal and painful bone growth occurs on the hip and other bones). Sometimes, back pain resulting from Harrington rods and other instrumentation may lead to autonomic dysreflexia that occur only when sitting up or lying down. These causes must be considered and either eliminated or ruled out.

Autonomic dysreflexia often occur during sexual activity, labor, and delivery. Fortunately, the autonomic dysreflexia associated with orgasm and other sexual activity is usually mild and controllable with drugs but obstetricians should be aware and prepared to treat autonomic dysreflexia in women undergoing labor. Some individuals who have uncomfortable autonomic dysreflexia during sexual activity should consult their doctors for the possibility of having medication on hand (such as nitroglycerin) to counter some of the symptoms before or after the activity. Sometimes, a glass of wine can help reduce autonomic dysreflexia.

Does recovery occur after spinal cord injury?

Until recently, many doctors told patients and families that recovery does not occur after spinal cord injury. Some recovery is the rule even after severe spinal cord injury.

- **Segmental recovery.** Almost all patients recover 1–2 segments below the injury site, even after so-called “complete” spinal cord injuries. For example, a person with a C4/5 injury usually has only deltoid function on admission to hospital but will frequently recover biceps (C5), wrist extensors (C6), and perhaps even triceps (C7) over several months. This is often associated with return of 2–3 segments of sensory function.
- **Recovery due to methylprednisolone.** The second National Acute Spinal Cord Injury Study (NASCIS 2) showed that patients with “complete” spinal cord injuries and who did not receive the high-dose steroid methylprednisolone recovered on average 8% of motor function that were lost on admission by 6–12 months after injury. If they received methylprednisolone within 8 hours after injury, they recovered on average 21% of what they had lost. In contrast, people with “incomplete” spinal cord injury recovered on average 59% of motor function and 75% if treated with high dose methylprednisolone.
- **Recovery of postural reflexes.** Most people with cervical or upper thoracic spinal cord injury are initially unable to control their trunk muscles. However, a majority will recover better trunk control over several months or even years after injury. This may be in the form of better vertebral or even abdominal reflexes.
- **Walking quads and paras.** A majority of people with “incomplete” spinal cord injuries, i.e. ASIA B or C, will recover ability to walk to some extent. Such recoveries after “complete” spinal cord injuries, i.e. ASIA A, are more rare but can occur in as much as 5% of the cases. In the 1980’s, less than 40% of spinal cord injuries admitted to hospital were “incomplete”. However, in the 1990’s, over 60% of spinal cord injuries are “incomplete” and thus the incidence of “walking quads” or “walking paras” may be higher than most people think.

Both animal and human studies indicate that as little as 10% of spinal cord tracts can support substantial function, including locomotion. People often can walk even though a tumor has damaged 90% of their spinal cord. This is because of the redundancy and plasticity of the spinal cord. Multiple spinal pathways serve similar or overlapping functions. Plasticity refers to the ability of axons to sprout and make new connections. Because transected spinal cords are rare, most people have some spinal axons passing through the injury site. This is the basis of the hope that even minimal regeneration of the spinal cord will restore substantial function in many people with spinal cord injury.

Experimental Therapies for Subacute Spinal Cord Injury

Several therapies have been reported to improve recovery after spinal cord injury and are in clinical trial. More information is available [Clinical Trial Forum](#).

- ***Monosialic ganglioside (GM1, Sygen)***. In 1991, Fred Geisler and colleagues reported that GM1 injected daily for 6 weeks after injury improve locomotor recovery 37 patients. Fidia Pharmaceutical subsequently tested this therapy in a large multicenter clinical trial in 800 patients, showing that the GM1 accelerated recovery during the first six weeks but did not significantly improve the extent of recovery at 6–12 months after injury. Note that this trial is no longer active. Although the drug is still available in Europe, the company Fidia has been bought by another company. [CareCure Forum \(GM1\) Link](#)
- ***Activated macrophage transplants***. In 1998, Michal Schwartz at the Weizmann Institute reported that activated macrophages obtained from blood and transplanted to the spinal cord improve functional recovery in rats. The company Proneuron initiated a phase 1 clinical trial to examine the feasibility and safety of macrophage transplants in human spinal cord injury. The clinical trial has been completed (although not yet published) and preliminary reports suggest that the treatment is feasible and safe in about 10 patients after spinal cord injury. All the patients had “complete” thoracic spinal cord injury and received macrophage transplants within 2 weeks after injury. Three of the 10 patients recovered from ASIA A to ASIA C, more than the expected 5%. A phase 1 clinical trial is continuing at Erasmus Hospital in Brussels, Belgium. A phase 2 trial is being planned in two U.S. centers including Craig Hospital in Denver (CO) and Mt. Sinai in New York City (NY). [CareCure Forum \(Macrophage\) Link](#)
- ***Alternating Current Electrical Stimulation***. In 1999, Dick Borgens and colleagues at Purdue University reported that alternating currents applied to dog spinal cords stimulated regeneration and recovery of function in dogs with spinal cord injury. A clinical trial has commenced at Purdue University for people who are within 2 weeks after acute spinal cord injury. [CareCure Forum \(AC Stim\) Link](#)
- ***AIT-082 (Neotrofin)***. This drug is a guanosine analog that can be taken orally and reportedly increases neurotrophins or neural growth factors in the brain and spinal cord. Neotherapeutics tested this drug in patients with Alzheimer’s disease. A year ago, they started a multicenter clinical trial at Ranchos Los Amigos in Downey (CA), Gaylord Hospital in Wallingford (CT), and Thomas Jefferson Hospital in Philadelphia. The treatment is started within 2 weeks after spinal cord injury. [CareCure Forum \(AIT-082\) Link](#)

Experimental Therapies for Chronic Spinal Cord Injury

Several therapies are being tested in clinical trials for chronic spinal cord injury. These trials all are aimed at people who are more than one year after injury. Many other treatments are being considered for clinical trial (see [article](#) on Advances in Spinal Cord Injury Therapy 25 November 2002).

- **4-aminopyridine (4-AP).** This drug is a small molecule that blocks fast voltage sensitive potassium channel blockers. The drug can be obtained by physician prescription from compounding pharmacies in the United States. In addition, Acorda Therapeutics is carrying out a multicenter phase 3 clinical trial of a sustained release formulation of the drug in people who are more than one and a half years after “incomplete” spinal cord injury. The drug may improve conduction of demyelinated axons in the spinal cord and preliminary clinical trial results suggest that the drug may reduce spasticity and improve motor or sensory function in as many as a third of people with chronic spinal cord injury. See [CareCure Forum \(4-AP\) Link](#)
- **Fetal porcine stem cell transplants.** Embryonic stem cells have attracted much attention. Several studies of human fetal cell transplants have been carried out in Sweden, Russia, and the United States, showing that transplanted fetal cells will engraft in human spinal cords. However, due in part of the lack of availability of adult human stem cells for transplantation and politics associated with the use of embryonic human stem cells, the first and only stem cell therapy trial for spinal cord injury in the United States used fetal stem cells from pigs. A phase 1 clinical trial at Washington University in St. Louis (MO) and Albany Medical Center in Albany (NY) has transplanted fetal stem obtained from pig fetuses and treated with antibodies to reduce the immune rejection. Sponsored by Diacrin, this trial is aiming to test 10 patients. See [CareCure Forum \(Diacrin\) Link](#)
- **Olfactory ensheathing glial transplants.** Olfactory ensheathing glia (OEG) normally reside in the olfactory nerve and the olfactory bulb. They are believed to be the reasons why the olfactory nerve continuously regenerates in adults. OEG’s are made in the nasal mucosa and migrate up the nerve to the olfactory bulb. Several laboratories have shown that OEG transplants facilitate regeneration of the spinal cord. Three clinical trials have started in Lisbon (Portugal), Brisbane (Australia), and Beijing (China). In Lisbon, they are transplanting the nasal mucosa obtained from the patient into the spinal cord. In Brisbane, they are culturing OEG cells from the nasal mucosa and transplanting the cells to the spinal cord. In Beijing, they are culturing OEG from human fetal olfactory bulbs and transplanting into the spinal cord. See [CareCure Forum Link \(Brisbane\)](#) and [CareCure Forum Link \(Beijing\)](#)

Summary

Spinal cord injury is devastating, not only for the injured person but for families and friends. While much information is available on Internet, most of the material is scattered and out of date. This article summarizes answers to some of the most frequently asked questions by people who are encountering spinal cord injury for the first time. Spinal cord injury disconnects the brain from the body. This leads not only to loss of sensation and motor control below the injury site but may be associated with abnormal activities of the spinal cord both above and below the injury site, resulting in spasticity, neuropathic pain, and autonomic dysreflexia. Many functions of our body that we take for granted, such as going to the bathroom, sexual function, blood pressure and heart rate, digestion, temperature control and sweating, and other autonomic functions may not only be lost but may be abnormally active. Finally, contrary to popular notions about spinal cord injury, recovery is the rule and not the exception in spinal cord injury. The recovery takes a long time and may be slowed down or blocked by the muscle atrophy and learned non-use. Finally, there is hope. Many therapies have been shown to regenerate and remyelinate the spinal cord. Some of these are now in clinical trials and many more should be in clinical trial soon.