

[National Institute of Neurological Disorders and Stroke](#)

Spinal Cord Injury: Hope Through Research

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A Short History of the Treatment of Spinal Cord Injury

Accounts of spinal cord injuries and their treatment date back to ancient times, even though there was little chance of recovery from such a devastating injury. The earliest is found in an Egyptian papyrus roll manuscript written in approximately 1700 B.C. that describes two spinal cord injuries involving fracture or dislocation of the neck vertebrae accompanied by paralysis.* The description of each was "an ailment not to be treated."

Centuries later in Greece, treatment for spinal cord injuries had changed little. According to the Greek physician Hippocrates (460-377 B.C.) there were no treatment options for spinal cord injuries that resulted in paralysis; unfortunately, those patients were destined to die. But Hippocrates did use rudimentary forms of traction to treat spinal fractures without paralysis. The Hippocratic Ladder was a device that required the patient to be bound, tied to the rungs upside-down, and shaken vigorously to reduce spinal curvature. Another invention, the Hippocratic Board, allowed the doctor to apply traction to the immobilized patient's back using either his hands and feet or a wheel and axle arrangement.

Hindu, Arab, and Chinese physicians also developed basic forms of traction to correct spinal deformities. These same principles of traction are still applied today.

In about 200 A.D., the Roman physician Galen introduced the concept of the central nervous system when he proposed that the spinal cord was an extension of the brain that carried sensation to the limbs and back. By the seventh century A.D., Paulus of Aegina was recommending surgery for spinal column fracture to remove the bone fragments that he was convinced caused paralysis.

In his influential anatomy textbook published in 1543, the Renaissance physician and teacher Vesalius described and illustrated the spinal cord in all its parts. The illustrations in his books, based on direct observation and dissection of the spine, gave physicians a way to understand the basic structure of the spine and spinal cord and what could happen when it was injured. The words we use today to identify segments of the spine - *cervical*, *thoracic*, *lumbar*, *sacral*, and *coccygeal* - come directly from Vesalius.

With the widespread use of antiseptics and sterilization in surgical procedures in the late nineteenth century, spinal surgery could finally be done with a much lower risk of infection. The use of X-rays, beginning in the 1920s, gave surgeons a way to precisely locate the injury and also made diagnosis and prediction of outcome more accurate. By the middle of the twentieth century, a standard method of treating spinal cord injuries was established - reposition the spine, fix it in place, and rehabilitate disabilities with exercise. In the 1990s, the discovery that the steroid drug *methylprednisolone* could reduce damage to nerve cells if given early enough after injury gave doctors an additional treatment option.

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What Is a Spinal Cord Injury?

Although the hard bones of the spinal column protect the soft tissues of the spinal cord, vertebrae can still be broken or dislocated in a variety of ways and cause traumatic injury to the spinal cord. Injuries can occur at any level of the spinal cord. The segment of the cord that is injured, and the severity of the injury, will determine which body functions are compromised or lost. Because the spinal cord acts as the main information pathway between the brain and the rest of the body, a spinal cord injury can have significant physiological consequences.

Catastrophic falls, being thrown from a horse or through a windshield, or any kind of physical trauma that crushes and compresses the vertebrae in the neck can cause irreversible damage at the cervical level of the spinal cord and below. Paralysis of most of the body including the arms and legs, called *quadriplegia*, is the likely result. Automobile accidents are often responsible for spinal cord damage in the middle back (the thoracic or lumbar area), which can cause paralysis of the lower trunk and lower extremities, called *paraplegia*.

Other kinds of injuries that directly penetrate the spinal cord, such as gunshot or knife wounds, can either completely or partially sever the spinal cord and create life-long disabilities.

Most injuries to the spinal cord don't completely sever it. Instead, an injury is more likely to cause fractures and compression of the vertebrae, which then crush and destroy the *axons*, extensions of nerve cells that carry signals up and down the spinal cord between the brain and the rest of the body. An injury to the spinal cord can damage a few, many, or almost all of these axons. Some injuries will allow almost complete recovery. Others will result in complete paralysis.

Until World War II, a serious spinal cord injury usually meant certain death, or at best a lifetime confined to a wheelchair and an ongoing struggle to survive secondary complications such as breathing problems or blood clots. But today, improved emergency care for people with spinal cord injuries and aggressive treatment and rehabilitation can minimize damage to the nervous system and even restore limited abilities.

Advances in research are giving doctors and patients hope that all spinal cord injuries will eventually be repairable. With new surgical techniques and exciting developments in spinal nerve *regeneration*, the future for spinal cord injury survivors looks brighter every day.

This brochure has been written to explain what happens to the spinal cord when it is injured, the current treatments for spinal cord injury patients, and the most promising avenues of research currently under investigation.

Facts and Figures About Spinal Cord Injury

- There are an estimated 10,000 to 12,000 spinal cord injuries every year in the United States.
- A quarter of a million Americans are currently living with spinal cord injuries.
- The cost of managing the care of spinal cord injury patients approaches \$4 billion each year.
- 38.5 percent of all spinal cord injuries happen during car accidents. Almost a quarter, 24.5 percent, are the result of injuries relating to violent encounters, often involving guns and knives. The rest are due to sporting accidents, falls, and work-related accidents.
- 55 percent of spinal cord injury victims are between 16 and 30 years old.
- More than 80 percent of spinal cord injury patients are men

Source: Facts and Figures at a Glance, May 2001. [National Spinal Cord Injury Statistical Center](#).

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How Does the Spinal Cord Work?

To understand what can happen as the result of a spinal cord injury, it helps to know the anatomy of the spinal cord and its normal functions.

Spine Anatomy

The soft, jelly-like spinal cord is protected by the spinal column. The spinal column is made up of 33 bones called vertebrae, each with a circular opening similar to the hole in a donut. The bones are stacked one on top of the other and the spinal cord runs through the hollow channel created by the holes in the stacked bones.

The vertebrae can be organized into sections, and are named and numbered from top to bottom according to their location along the backbone:

- Cervical vertebrae (1-7) located in the neck
- Thoracic vertebrae (1-12) in the upper back (attached to the ribcage)
- Lumbar vertebrae (1-5) in the lower back
- Sacral vertebrae (1-5) in the hip area
- Coccygeal vertebrae (1-4 fused) in the tailbone

Although the hard vertebrae protect the soft spinal cord from injury most of the time, the spinal column is not all hard bone. Between the vertebrae are *discs* of semi-rigid cartilage, and in the narrow spaces between them are passages through which the spinal nerves exit to the rest of the body. These are places where the spinal cord is vulnerable to direct injury.

The spinal cord is also organized into segments and named and numbered from top to bottom. Each segment marks where spinal nerves emerge from the cord to connect to specific regions of the body. Locations of spinal cord segments do not correspond exactly to vertebral locations, but they are roughly equivalent.

- Cervical spinal nerves (C1 to C8) control signals to the back of the head, the neck and shoulders, the arms and hands, and the diaphragm.
- Thoracic spinal nerves (T1 to T12) control signals to the chest muscles, some muscles of the back, and parts of the abdomen.
- Lumbar spinal nerves (L1 to L5) control signals to the lower parts of the abdomen and the back, the buttocks, some parts of the external genital organs, and parts of the leg.
- Sacral spinal nerves (S1 to S5) control signals to the thighs and lower parts of the legs, the feet, most of the external genital organs, and the area around the anus.

The single *coccygeal nerve* carries sensory information from the skin of the lower back.

Spinal Cord Anatomy

The spinal cord has a core of tissue containing nerve cells, surrounded by long tracts of nerve fibers consisting of axons. The tracts extend up and down the spinal cord, carrying signals to and from the brain. The average size of the spinal cord varies in circumference along its length from the width of a thumb to the width of one of the smaller fingers. The spinal cord extends down through the upper two thirds of the vertebral canal, from the base of the brain to the lower back, and is generally 15 to 17 inches long depending on an individual's height.

The interior of the spinal cord is made up of neurons, their support cells called *glia*, and blood vessels. The neurons and their *dendrites* (branching projections that help neurons communicate with each other) reside in an H-shaped region called "grey matter."

The H-shaped grey matter of the spinal cord contains motor neurons that control movement, smaller *interneurons* that handle communication within and between the segments of the spinal cord, and cells that receive sensory signals and then send information up to centers in the brain.

Surrounding the grey matter of neurons is white matter. Most axons are covered with an insulating substance called *myelin*, which allows electrical signals to flow freely and quickly. Myelin has a whitish appearance, which is why this outer section of the spinal cord is called "white matter."

Axons carry signals downward from the brain (along descending pathways) and upward toward the brain (along ascending pathways) within specific tracts. Axons branch at their ends and can make connections with many other nerve cells simultaneously. Some axons extend along the entire length of the spinal cord.

The descending motor tracts control the smooth muscles of internal organs and the striated (capable of voluntary contractions) muscles of the arms and legs. They also help adjust the autonomic nervous system's regulation of blood pressure, body temperature, and the response to stress. These pathways begin with neurons in the brain that send electrical signals downward to specific levels of the spinal cord. Neurons in these segments then send the impulses out to the rest of the body or coordinate neural activity within the cord itself.

The ascending sensory tracts transmit sensory signals from the skin, extremities, and internal organs that enter at specific segments of the spinal cord. Most of these signals are then relayed to the brain. The spinal cord also contains neuronal circuits that control reflexes and repetitive movements, such as walking, which can be

activated by incoming sensory signals without input from the brain.

The circumference of the spinal cord varies depending on its location. It is larger in the cervical and lumbar areas because these areas supply the nerves to the arms and upper body and the legs and lower body, which require the most intense muscular control and receive the most sensory signals.

The ratio of white matter to grey matter also varies at each level of the spinal cord. In the cervical segment, which is located in the neck, there is a large amount of white matter because at this level there are many axons going to and from the brain and the rest of the spinal cord below. In lower segments, such as the sacral, there is less white matter because most ascending axons have not yet entered the cord, and most descending axons have contacted their targets along the way.

To pass between the vertebrae, the axons that link the spinal cord to the muscles and the rest of the body are bundled into 31 pairs of spinal nerves, each pair with a sensory root and a motor root that make connections within the grey matter. Two pairs of nerves - a sensory and motor pair on either side of the cord - emerge from each segment of the spinal cord.

The functions of these nerves are determined by their location in the spinal cord. They control everything from body functions such as breathing, sweating, digestion, and elimination, to gross and fine motor skills, as well as sensations in the arms and legs.

The Nervous Systems

Together, the spinal cord and the brain make up the central nervous system (CNS).

The CNS controls most functions of the body, but it is not the only nervous system in the body. The peripheral nervous system (PNS) includes the nerves that project to the limbs, heart, skin, and other organs outside the brain. The PNS controls the somatic nervous system, which regulates muscle movements and the response to sensations of touch and pain, and the autonomic nervous system, which provides nerve input to the internal organs and generates automatic reflex responses. The autonomic nervous system is divided into the sympathetic nervous system, which mobilizes organs and their functions during times of stress and arousal, and the parasympathetic nervous system, which conserves energy and resources during times of rest and relaxation.

The spinal cord acts as the primary information pathway between the brain and all the other nervous systems of the body. It receives sensory information from the skin, joints, and muscles of the trunk, arms, and legs, which it then relays upward to the brain. It carries messages downward from the brain to the PNS, and contains motor neurons, which direct voluntary movements and adjust reflex movements. Because of the central role it plays in coordinating muscle movements and interpreting sensory input, any kind of injury to the spinal cord can cause significant problems throughout the body.

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What Happens When the Spinal Cord Is Injured?

A spinal cord injury usually begins with a sudden, traumatic blow to the spine that fractures or dislocates vertebrae. The damage begins at the moment of injury when displaced bone fragments, disc material, or *ligaments* bruise or tear into spinal cord tissue. Axons are cut off or damaged beyond repair, and neural cell membranes are broken. Blood vessels may rupture and cause heavy bleeding in the central grey matter, which can spread to other areas of the spinal cord over the next few hours.

Within minutes, the spinal cord swells to fill the entire cavity of the spinal canal at the injury level. This swelling cuts off blood flow, which also cuts off oxygen to spinal cord tissue. Blood pressure drops, sometimes dramatically, as the body loses its ability to self-regulate. As blood pressure lowers even further, it interferes with the electrical activity of neurons and axons. All these changes can cause a condition known as *spinal shock* that can last from several hours to several days.

Although there is some controversy among neurologists about the extent and impact of spinal shock, and even its definition in terms of physiological characteristics, it appears to occur in approximately half the cases of spinal cord injury, and it is usually directly related to the size and severity of the injury. During spinal shock, even undamaged portions of the spinal cord become temporarily disabled and can't communicate normally with the brain. Complete paralysis may develop, with loss of reflexes and sensation in the limbs.

The crushing and tearing of axons is just the beginning of the devastation that occurs in the injured spinal cord and continues for days. The initial physical trauma sets off a cascade of biochemical and cellular events that

kills neurons, strips axons of their myelin insulation, and triggers an inflammatory immune system response. Days or sometimes even weeks later, after this second wave of damage has passed, the area of destruction has increased - sometimes to several segments above and below the original injury - and so has the extent of disability.

- **Changes in blood flow cause ongoing damage**

Changes in blood flow in and around the spinal cord begin at the injured area, spread out to adjacent, uninjured areas, and then set off problems throughout the body.

Immediately after the injury, there is a major reduction in blood flow to the site, which can last for as long as 24 hours and becomes progressively worse if untreated. Because of differences in tissue composition, the impact is greater on the interior grey matter of the spinal cord than on the outlying white matter.

Blood vessels in the grey matter also begin to leak, sometimes as early as 5 minutes after injury. Cells that line the still-intact blood vessels in the spinal cord begin to swell, for reasons that aren't yet clearly understood, and this continues to reduce blood flow to the injured area. The combination of leaking, swelling, and sluggish blood flow prevents the normal delivery of oxygen and nutrients to neurons, causing many of them to die.

The body continues to regulate blood pressure and heart rate during the first hour to hour-and-a-half after the injury, but as the reduction in the rate of blood flow becomes more widespread, self-regulation begins to turn off. Blood pressure and heart rate drop.

- **Excessive release of neurotransmitters kills nerve cells**

After the injury, an excessive release of *neurotransmitters* (chemicals that allow neurons to signal each other) can cause additional damage by overexciting nerve cells.

Glutamate is an excitatory neurotransmitter, commonly used by nerve cells in the spinal cord to stimulate activity in neurons. But when spinal cells are injured, neurons flood the area with glutamate for reasons that are not yet well understood. Excessive glutamate triggers a destructive process called *excitotoxicity*, which disrupts normal processes and kills neurons and other cells called *oligodendrocytes* that surround and protect axons.

- **An invasion of immune system cells creates inflammation**

Under normal conditions, the blood-brain barrier (which tightly controls the passage of cells and large molecules between the circulatory and central nervous systems) keeps immune system cells from entering the brain or spinal cord. But when the blood-brain barrier is broken by blood vessels bursting and leaking into spinal cord tissue, immune system cells that normally circulate in the blood - primarily white blood cells - can invade the surrounding tissue and trigger an inflammatory response. This inflammation is characterized by fluid accumulation and the influx of immune cells - *neutrophils*, *T-cells*, *macrophages*, and *monocytes*.

Neutrophils are the first to enter, within about 12 hours of injury, and they remain for about a day. Three days after the injury, T-cells arrive. Their function in the injured spinal cord is not clearly understood, but in the healthy spinal cord they kill infected cells and regulate the immune response. Macrophages and monocytes enter after the T-cells and scavenge cellular debris.

The up side of this immune system response is that it helps fight infection and cleans up debris. But the down side is that it sets off the release of *cytokines* - a group of immune system messenger molecules that exert a malign influence on the activities of nerve cells.

For example, *microglial* cells, which normally function as a kind of on-site immune cell in the spinal cord, begin to respond to signals from these cytokines. They transform into macrophage-like cells, engulf cell debris, and start to produce their own pro-inflammatory cytokines, which then stimulate and recruit other microglia to respond.

Injury also stimulates resting *astrocytes* to express cytokines. These "reactive" astrocytes may ultimately participate in the formation of scar tissue within the spinal cord.

Whether or not the immune response is protective or destructive is controversial among researchers. Some speculate that certain types of injury might evoke a protective immune response that actually reduces the loss of neurons.

- **Free radicals attack nerve cells**

Another consequence of the immune system's entry into the CNS is that inflammation accelerates the production of highly reactive forms of oxygen molecules called *free radicals*.

Free radicals are produced as a by-product of normal cell metabolism. In the healthy spinal cord their numbers are small enough that they cause no harm. But injury to the spinal cord, and the subsequent wave of inflammation that sweeps through spinal cord tissue, signals particular cells to overproduce free radicals.

Free radicals then attack and disable molecules that are crucial for cell function - for example, those found in cell membranes - by modifying their chemical structure. Free radicals can also change how cells respond to natural growth and survival factors, and turn these protective factors into agents of destruction.

- **Nerve cells self-destruct**

Researchers used to think that the only way in which cells died during spinal cord injury was as a direct result of trauma. But recent findings have revealed that cells in the injured spinal cord also die from a kind of programmed cell death called *apoptosis*, often described as cellular suicide, that happens days or weeks after the injury.

Apoptosis is a normal cellular event that occurs in a variety of tissues and cellular systems. It helps the body get rid of old and unhealthy cells by causing them to shrink and implode. Nearby scavenger cells then gobble up the debris. Apoptosis seems to be regulated by specific molecules that have the ability to either start or stop the process.

For reasons that are still unclear, spinal cord injury sets off apoptosis, which kills oligodendrocytes in damaged areas of the spinal cord days to weeks after the injury. The death of oligodendrocytes is another blow to the damaged spinal cord, since these are the cells that form the myelin that wraps around axons and speeds the conduction of nerve impulses. Apoptosis strips myelin from intact axons in adjacent ascending and descending pathways, which further impairs the spinal cord's ability to communicate with the brain.

- **Secondary damage takes a cumulative toll**

All of these mechanisms of secondary damage - restricted blood flow, excitotoxicity, inflammation, free radical release, and apoptosis - increase the area of damage in the injured spinal cord. Damaged axons become dysfunctional, either because they are stripped of their myelin or because they are disconnected from the brain. Glial cells cluster to form a scar, which creates a barrier to any axons that could potentially regenerate and reconnect. A few whole axons may remain, but not enough to convey any meaningful information to the brain.

Researchers are especially interested in studying the mechanisms of this wave of secondary damage because finding ways to stop it could save axons and reduce disabilities. This could make a big difference in the potential for recovery.

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What Are the Immediate Treatments for Spinal Cord Injury?

The outcome of any injury to the spinal cord depends upon the number of axons that survive: the higher the number of normally functioning axons, the less the amount of disability. Consequently, the most important consideration when moving people to a hospital or trauma center is preventing further injury to the spine and spinal cord.

Spinal cord injury isn't always obvious. Any injury that involves the head (especially with trauma to the front of the face), pelvic fractures, penetrating injuries in the area of the spine, or injuries that result from falling from heights should be suspect for spinal cord damage.

Until imaging of the spine is done at an emergency or trauma center, people who might have spinal cord injury should be cared for as if any significant movement of the spine could cause further damage. They are usually transported in a recumbent (lying down) position, with a rigid collar and backboard immobilizing the spine.

Respiratory complications are often an indication of the severity of spinal cord injury. About one third of those with injury to the neck area will need help with breathing and require respiratory support via *intubation*, which involves inserting a tube connected to an oxygen tank through the nose or throat and into the airway.

Methylprednisolone, a steroid drug, became standard treatment for acute spinal cord injury in 1990 when a large-scale clinical trial supported by the National Institute of Neurological Disorders and Stroke showed significantly better recovery in patients who were given the drug within the first 8 hours after their injury. Methylprednisolone appears to reduce the damage to nerve cells and decreases inflammation near the injury site by suppressing activities of immune cells.

Realignment of the spine using a rigid brace or *axial traction* is usually done as soon as possible to stabilize the spine and prevent additional damage.

On about the third day after the injury, doctors give patients a complete neurological examination to diagnose the severity of the injury and predict the likely extent of recovery. The ASIA Impairment Scale is the standard diagnostic tool used by doctors. X-rays, MRIs, or more advanced imaging techniques are also used to visualize the entire length of the spine.

ASIA (American Spinal Injury Association) Impairment Scale*

Classification	Description
A	Complete: no motor or sensory function is preserved below the level of injury, including the sacral segments S4-S5
B	Incomplete: sensory, but not motor, function is preserved below the neurologic level and some sensation in the sacral segments S4-S5
C	Incomplete: motor function is preserved below the neurologic level, however, more than half of key muscles below the neurologic level have a muscle grade less than 3 (i.e., not strong enough to move against gravity)
D	Incomplete: motor function is preserved below the neurologic level, and at least half of key muscles below the neurologic level have a muscle grade of 3 or more (i.e., joints can be moved against gravity)
E	Normal: motor and sensory functions are normal

* Used with permission of the American Spinal Injury Association.

Spinal cord injuries are classified as either complete or incomplete, depending on how much cord width is injured. An incomplete injury means that the ability of the spinal cord to convey messages to or from the brain is not completely lost. People with incomplete injuries retain some motor or sensory function below the injury.

A complete injury is indicated by a total lack of sensory and motor function below the level of injury.

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How Does a Spinal Cord Injury Affect the Rest of the Body?

People who survive a spinal cord injury will most likely have medical complications such as chronic pain and bladder and bowel dysfunction, along with an increased susceptibility to respiratory and heart problems. Successful recovery depends upon how well these chronic conditions are handled day to day.

■ Breathing

Any injury to the spinal cord at or above the C3, C4, and C5 segments, which supply the phrenic nerves leading to the diaphragm, can stop breathing. People with these injuries need immediate ventilatory support. When injuries are at the C5 level and below, diaphragm function is preserved, but breathing tends to be rapid and shallow and people have trouble coughing and clearing secretions from their lungs because of weak thoracic muscles. Once pulmonary function improves, a large percentage of those with C4 injuries can be weaned from mechanical ventilation in the weeks following the injury.

■ Pneumonia

Respiratory complications, primarily as a result of pneumonia, are a leading cause of death in people with spinal cord injury. In fact, intubation increases the risk of developing ventilator-associated pneumonia

(VAP) by 1 to 3 percent per day of intubation. More than a quarter of the deaths caused by spinal cord injury are the result of VAP. Spinal cord injury patients who are intubated have to be carefully monitored for VAP and treated with antibiotics if symptoms appear.

- **Irregular heart beat and low blood pressure**

Spinal cord injuries in the cervical region are often accompanied by blood pressure instability and heart *arrhythmias*. Because of interruptions to the cardiac accelerator nerves, the heart can beat at a dangerously slow pace, or it can pound rapidly and irregularly. Arrhythmias usually appear in the first 2 weeks after injury and are more common and severe in the most serious injuries.

Low blood pressure also often occurs due to loss of tone in blood vessels, which widen and cause blood to pool in the small arteries far away from the heart. This is usually treated with an intravenous infusion to build up blood volume.

- **Blood clots**

People with spinal cord injuries are at triple the usual risk for blood clots. The risk for clots is low in the first 72 hours, but afterwards anticoagulation drug therapy can be used as a preventive measure.

- **Spasm**

Many of our reflex movements are controlled by the spinal cord but regulated by the brain. When the spinal cord is damaged, information from the brain can no longer regulate reflex activity. Reflexes may become exaggerated over time, causing *spasticity*. If spasms become severe enough, they may require medical treatment. For some, spasms can be as much of a help as they are a hindrance, since spasms can tone muscles that would otherwise waste away. Some people can even learn to use the increased tone in their legs to help them turn over in bed, propel them into and out of a wheelchair, or stand.

- **Autonomic dysreflexia**

Autonomic dysreflexia is a life-threatening reflex action that primarily affects those with injuries to the neck or upper back. It happens when there is an irritation, pain, or stimulus to the nervous system below the level of injury. The irritated area tries to send a signal to the brain, but since the signal isn't able to get through, a reflex action occurs without the brain's regulation. Unlike spasms that affect muscles, autonomic dysreflexia affects vascular and organ systems controlled by the sympathetic nervous system.

Anything that causes pain or irritation can set off autonomic dysreflexia: the urge to urinate or defecate, *pressure sores*, cuts, burns, bruises, sunburn, pressure of any kind on the body, ingrown toenails, or tight clothing. For example, the impulse to urinate can set off high blood pressure or rapid heart beat that, if uncontrolled, can cause stroke, seizures, or death. Symptoms such as flushing or sweating, a pounding headache, anxiety, sudden high blood pressure, vision changes, or goosebumps on the arms and legs can signal the onset of autonomic dysreflexia. Treatment should be swift. Changing position, emptying the bladder or bowels, and removing or loosening tight clothing are just a few of the possibilities that should be tried to relieve whatever is causing the irritation.

- **Pressure sores (or pressure ulcers)**

Pressure sores are areas of skin tissue that have broken down because of continuous pressure on the skin. People with paraplegia and quadriplegia are susceptible to pressure sores because they can't move easily on their own.

Places that support weight when someone is seated or recumbent are vulnerable areas. When these areas press against a surface for a long period of time, the skin compresses and reduces the flow of blood to the area. When the blood supply is blocked for too long, the skin will begin to break down.

Since spinal cord injury reduces or eliminates sensation below the level of injury, people may not be aware of the normal signals to change position, and must be shifted periodically by a caregiver. Good nutrition and hygiene can also help prevent pressure sores by encouraging healthy skin.

▪ Pain

People who are paralyzed often have what is called *neurogenic* pain resulting from damage to nerves in the spinal cord. For some survivors of spinal cord injury, pain or an intense burning or stinging sensation is unremitting due to hypersensitivity in some parts of the body. Others are prone to normal musculoskeletal pain as well, such as shoulder pain due to overuse of the shoulder joint from pushing a wheelchair and using the arms for transfers. Treatments for chronic pain include medications, acupuncture, spinal or brain electrical stimulation, and surgery.

▪ Bladder and bowel problems

Most spinal cord injuries affect bladder and bowel functions because the nerves that control the involved organs originate in the segments near the lower termination of the spinal cord and are cut off from brain input. Without coordination from the brain, the muscles of the bladder and urethra can't work together effectively, and urination becomes abnormal. The bladder can empty suddenly without warning, or become over-full without releasing. In some cases the bladder releases, but urine backs up into the kidneys because it isn't able to get past the urethral sphincter. Most people with spinal cord injuries use either intermittent catheterization or an indwelling catheter to empty their bladders.

Bowel function is similarly affected. The anal sphincter muscle can remain tight, so that bowel movements happen on a reflex basis whenever the bowel is full. Or the muscle can be permanently relaxed, which is called a "flaccid bowel," and result in an inability to have a bowel movement. This requires more frequent attempts to empty the bowel and manual removal of stool to prevent fecal impaction. People with spinal cord injuries are usually put on a regularly scheduled bowel program to prevent accidents.

▪ Reproductive and sexual function

Spinal cord injury has a greater impact on sexual and reproductive function in men than it does in women. Most spinal cord injured women remain fertile and can conceive and bear children. Even those with severe injury may well retain orgasmic function, although many lose some if not all of their ability to reach satisfaction.

Depending on the level of injury, men may have problems with erections and ejaculation, and most will have compromised fertility due to decreased motility of their sperm. Treatments for men include vibratory or electrical stimulation and drugs such as sildenafil (Viagra). Many couples may also need assisted fertility treatments to allow a spinal cord injured man to father children.

Once someone has survived the injury and begun to psychologically and emotionally cope with the nature of his or her situation, the next concern will be how to live with disabilities. Doctors are now able to predict with reasonable accuracy the likely long-term outcome of spinal cord injuries. This helps patients set achievable goals for themselves, and gives families and loved ones a realistic set of expectations for the future.

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How Does Rehabilitation Help People Recover From Spinal Cord Injuries?

No two people will experience the same emotions after surviving a spinal cord injury, but almost everyone will feel frightened, anxious, or confused about what has happened. It's common for people to have very mixed feelings: relief that they are still alive, but disbelief at the nature of their disabilities.

Rehabilitation programs combine physical therapies with skill-building activities and counseling to provide social and emotional support. The education and active involvement of the newly injured person and his or her family and friends is crucial.

A rehabilitation team is usually led by a doctor specializing in physical medicine and rehabilitation (called a physiatrist), and often includes social workers, physical and occupational therapists, recreational therapists, rehabilitation nurses, rehabilitation psychologists, vocational counselors, nutritionists, and other specialists. A case-worker or program manager coordinates care.

In the initial phase of rehabilitation, therapists emphasize regaining leg and arm strength since mobility and communication are the two most important areas of function. For some, mobility will only be possible with the

assistance of devices such as a walker, leg braces, or a wheelchair. Communication skills, such as writing, typing, and using the telephone, may also require adaptive devices.

Physical therapy includes exercise programs geared toward muscle strengthening. Occupational therapy helps redevelop fine motor skills. Bladder and bowel management programs teach basic toileting routines, and patients also learn techniques for self-grooming. People acquire coping strategies for recurring episodes of spasticity, autonomic dysreflexia, and neurogenic pain.

Vocational rehabilitation begins with an assessment of basic work skills, current dexterity, and physical and cognitive capabilities to determine the likelihood for employment. A vocational rehabilitation specialist then identifies potential work places, determines the type of assistive equipment that will be needed, and helps arrange for a user-friendly workplace. For those whose disabilities prevent them from returning to the workplace, therapists focus on encouraging productivity through participation in activities that provide a sense of satisfaction and self-esteem. This could include educational classes, hobbies, memberships in special interest groups, and participation in family and community events.

Recreation therapy encourages patients to build on their abilities so that they can participate in recreational or athletic activities at their level of mobility. Engaging in recreational outlets and athletics helps those with spinal cord injuries achieve a more balanced and normal lifestyle and also provides opportunities for socialization and self-expression.

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How Is Research Helping Spinal Cord Injury Patients?

Can an injured spinal cord be rebuilt? This is the question that drives basic research in the field of spinal cord injury. As investigators try to understand the underlying biological mechanisms that either inhibit or promote new growth in the spinal cord, they are making surprising discoveries, not just about how neurons and their axons grow in the CNS, but also about why they fail to regenerate after injury in the adult CNS. Understanding the cellular and molecular mechanisms involved in both the working and the damaged spinal cord could point the way to therapies that might prevent secondary damage, encourage axons to grow past injured areas, and reconnect vital neural circuits within the spinal cord and CNS.

There has been successful research in a number of fields that may someday help people with spinal cord injuries. Genetic studies have revealed a number of molecules that encourage axon growth in the developing CNS but prevent it in the adult. Research into embryonic and adult *stem cell* biology has furthered knowledge about how cells communicate with each other.

Basic research has helped describe the mechanisms involved in the mysterious process of apoptosis, in which large groups of seemingly healthy cells self-destruct. New rehabilitation therapies that retrain neural circuits through forced motion and electrical stimulation of muscle groups are helping injured patients regain lost function.

Researchers, many of whom are supported by the National Institute of Neurological Disorders and Stroke (NINDS), are focused on advancing our understanding of the four key principles of spinal cord repair:

- Protecting surviving nerve cells from further damage
- Replacing damaged nerve cells
- Stimulating the regrowth of axons and targeting their connections appropriately
- Retraining neural circuits to restore body functions

A spinal cord injury is complex. Repairing it has to take into account all of the different kinds of damage that occur during and after the injury. Because the molecular and cellular environment of the spinal cord is constantly changing from the moment of injury until several weeks or even months later, combination therapies will have to be designed to address specific types of damage at different points in time.

Discoveries in Basic Research

A decade ago, researchers demonstrated a small but significant neuroprotective and anti-inflammatory effect from an adrenal corticosteroid drug called methylprednisolone if it was given within 8 hours of injury. It is the only treatment currently available to limit the extent of spinal cord injury and its risks are relatively low. Researchers continue to search for additional anti-inflammatory treatments that might prove even more effective.

Preliminary clinical trials of another compound, *GM-1 ganglioside*, indicate that it could be useful in preventing secondary damage in acute spinal cord injury. A large, randomized clinical trial suggested that it might also improve neurological recovery from spinal cord injury during rehabilitation.

These observations and others have led to optimism that recovery can be improved by altering cellular responses immediately after injury. Using what they know about the mechanisms that cause secondary damage - excitotoxicity, inflammation, and cell suicide (apoptosis) - researchers are creating and testing additional neuroprotective therapies to prevent the spread of post-injury damage and preserve surrounding tissue.

Some of the findings in these three different areas follow:

▪ **Stopping excitotoxicity**

When nerve cells die, they release excessive amounts of a neurotransmitter called glutamate. Since surviving nerve cells also release glutamate as part of their normal communication process, excess glutamate floods the cellular environment, which pushes cells into overdrive and self-destruction. Researchers are investigating compounds that could keep nerve cells from responding to glutamate, potentially minimizing the extent of secondary damage.

Recently, investigators tested agents called *receptor antagonists* that selectively block a specific type of glutamate receptor that is abundant on oligodendrocytes and neurons. These agents appear to be effective at limiting damage. Some of these receptor antagonists have already been tested in human trials as a therapy for stroke. Similar agents could enter clinical trials within several years for patients with spinal cord injury.

▪ **Controlling inflammation**

Some time within the first 12 hours after injury, the first wave of immune cells enters the damaged spinal cord to protect it from infection and clean up dead nerve cells. Other types of immune cells enter afterwards. The actions of these immune cells and the messenger molecules they release, called cytokines, are the hallmarks of inflammation in the spinal cord.

Researchers have discovered that these inflammatory processes aren't entirely bad for the injured spinal cord. Although cytokines can be toxic to nerve cells because they stimulate the production of free radicals, nitric oxide, and other inflammatory substances that cause cell death, they also stimulate the production of *neurotrophic factors*, which are beneficial to cell repair.

Currently researchers are looking for ways to control these immune system cells and the molecules they produce by encouraging their potential for neuroprotection and reining in their neurotoxic effects. One approach being tested clinically is to exploit the ability of the PNS to mount a healing response in macrophages by injecting macrophages already stimulated by injured peripheral nerves into injured spinal cords. Recent experiments have indicated that selectively boosting the T-cell response to spinal cord injury could reduce secondary damage. Because of the possibility that these cells can also damage tissue, they must be very carefully controlled if they are to be used therapeutically.

Clinical investigators are also looking at how cooling the body protects surviving spinal cord tissue and nerve cells. Experiments have shown that cooling the body to a state of mild *hypothermia* (about 92° F) for several hours immediately following the injury limits damage and promotes functional recovery. Researchers aren't yet sure why mild hypothermia is neuroprotective, but the ability of body temperature to affect many different kinds of physiological mechanisms may be one of the reasons.

▪ **Preventing apoptosis**

Days to weeks after the initial injury, apoptosis sweeps through oligodendrocytes in damaged and nearby tissue, causing the cells to self-destruct. Although genes have been identified that appear to regulate apoptosis, researchers still don't know enough to be able to specify the exact biochemical events that cause a cell to switch it on - or turn it off. Further studies are aimed at understanding these cellular mechanisms more fully. These studies will provide an opportunity to develop neural protective strategies to combat apoptotic cell death.

By understanding the process of apoptosis, researchers have been able to develop and test apoptosis-inhibiting drugs. In rodent models, animals given a drug that blocks a known apoptotic mechanism retained more ambulatory ability after traumatic spinal cord injury than did untreated animals.

Once the secondary wave of damage ends, the spinal cord is left with areas of scar tissue and fluid-filled gaps, or cysts, that axons can't penetrate or bridge. Unless these areas are reconnected by functioning nerve cells, the spinal cord remains disabled. Discovering how to bridge the gap between functioning axons and figuring out how to encourage axons to grow and make new connections could be the key to spinal cord repair.

■ Promoting regeneration

Researchers are experimenting with cell grafts transplanted into the injured spinal cord that act as bridges across injured areas to reconnect cut axons, or that supply nerve cells to act as relays. Several types of cells have been studied for their potential to promote regeneration and repair, including *Schwann cells*, *olfactory ensheathing glia*, *fetal spinal cord cells*, and *embryonic stem cells*. In one group of experiments, investigators have implanted tubes packed with Schwann cells into the damaged spinal cords of rodents and observed axons growing into the tubes.

One of the limitations of cell transplants, however, is that the growth environment within the transplant is so favorable that most axons don't leave and extend into the spinal cord. By using olfactory ensheathing glia cells, which are natural migrators in the PNS, researchers have gotten axons to extend out of the initial transplant region and into the spinal cord. But it remains to be seen whether or not regenerated axons are fully functional.

Fetal spinal cord tissue implants have also yielded success in animal trials, giving rise to new neurons, which, when stimulated by growth-promoting factors (neurotrophins), extend axons that stretch up and down several segments in the spinal cord. Animals treated in these trials have regained some function in their limbs. Some patients with long-term spinal cord injuries have received fetal tissue transplants but the results have been inconclusive. In animal models, these transplants appear to be more effective in the immature spinal cord than in the adult spinal cord.

Stem cells are capable of dividing and yielding almost all the cell types of the body, including those of the spinal cord. Their potential to treat spinal cord injury is being investigated eagerly, but there are many things about stem cells that researchers still need to understand. For example, researchers know there are many different kinds of chemical signals that tell a stem cell what to do. Some of these are internal to the stem cell, but many others are external - within the cellular environment - and will have to be recreated in the transplant region to encourage proper growth and differentiation. Because of the complexities involved in stem cell treatment, researchers expect these kinds of therapies to be possible only after much more research is done.

Researchers are also looking at ways to compensate for axons that, having lost their myelin sheaths, have a decreased ability to conduct the electrical impulses essential for axonal communication. Preliminary studies with compounds known as potassium channel blockers, which block the flow of ions through the demyelinated membrane and increase the potential for messages to get through, have shown some success, but mostly in terms of reducing spasticity in muscles. Further studies might show how remyelinating axons could also improve function.

■ Stimulating regrowth of axons

Stimulating the regeneration of axons is a key component of spinal cord repair because every axon in the injured spinal cord that can be reconnected increases the chances for recovery of function.

Research on many fronts reveals that getting axons to grow after injury is a complicated task. CNS neurons have the capacity to regenerate, but the environment in the adult spinal cord does not encourage growth. Not only does it lack the growth-promoting molecules that are present in the developing CNS, it also contains substances that actively inhibit axon extension. For axon regeneration to be successful, the environment has to be changed to turn off the inhibitors and turn on the promoters.

Investigators are looking for ways to take advantage of the chemicals that drive or halt axon growth: growth-promoting and growth-inhibiting substances, neurotrophic factors, and *guidance molecules*.

In the developing CNS, thread-like axons grow and lengthen behind the *axonal growth cone*, an active tip only a few thousandths of a millimeter in diameter, which interacts with chemical signals that encourage growth and direct movement. But the environment of the adult CNS is hostile to axon growth, primarily because *growth-inhibiting proteins* are embedded in myelin, the insulating material around

axons. These proteins appear to preserve neural circuits in the healthy spinal cord and keep intact axons from growing inappropriately. But when the spinal cord is injured, these proteins prevent regeneration.

At least three growth-inhibitory proteins operating within the axonal tract have been identified. The task of researchers is to understand how these inhibitory proteins do their job, and then discover ways to remove or block them, or change how the growth cone responds to them.

Growth-inhibiting proteins also block the glial scar near the injury site. To get past, an axon has to advance between the tangles of long, branching molecules that form the *extracellular matrix*. A recent experiment successfully used a bacterial enzyme to clear away this underbrush so that axons could grow.

A treatment that combines both these approaches - turning off growth-inhibiting proteins and using enzymes to clear the way - could create an encouraging environment for axon regeneration. But before trials of such a treatment can be attempted in patients, researchers must be sure that it could be controlled well enough to prevent dangerous miswiring of regenerating axons.

Neurotrophic factors (or neurotrophins) are key nervous system regulatory proteins that prime cells to produce the molecular machinery necessary for growth. Some prevent oligodendrocyte death, others promote axon regrowth and survival, and still others serve multiple functions. Unfortunately, the natural production of neurotrophins in the spinal cord falls instead of rises during the weeks after injury. Researchers have tested whether artificially raising the levels post-injury can enhance regeneration. Some of these investigations have been successful. Infusion pumps and gene therapy techniques have been used to deliver growth factors to injured neurons, but they appear to encourage sprouting more than they stimulate regeneration for long distances.

Axonal growth isn't enough for functional recovery. Axons have to make the proper connections and re-establish functioning *synapses*. Guidance molecules, proteins that rest on or are released from the surfaces of neurons or glia, act as chemical road signs, beckoning axons to grow in some directions and repelling growth in others.

Supplying a particular combination of guidance molecules or administering compounds that induce surviving cells to produce or use guidance molecules might encourage regeneration. But at the moment, researchers don't understand enough about guidance molecules to know which to supply and when.

Researchers hope that combining these strategies to encourage growth, clear away debris, and target axon connections could reconnect the spinal cord. Of course, all these therapies would have to be provided in the right amounts, in the right places, and at the right times. As researchers learn more and understand more about the intricacies of axon growth and regeneration, combining therapies could become a powerful treatment for spinal cord injury.

Discoveries in Clinical Research

Advances in basic research are also being matched by progress in clinical research, especially in understanding the kinds of physical rehabilitation that work best to restore function. Some of the more promising rehabilitation techniques are helping spinal cord injury patients become more mobile.

▪ Restoring function through neural prostheses and computer interfaces

While basic scientists strive to develop strategies to restore neurological connections between the brain and body of spinal cord injured persons, bioengineers are working to restore functional connections via advanced computer modeling systems and *neural prostheses*. Discovering ways to integrate devices that could mobilize paralyzed limbs requires a unique interface between electronics technology and neurobiology. A *functional electrical stimulation* (FES) system is one example of this kind of innovative research.

FES systems use electrical stimulators to control muscles of the legs and arms to encourage functional walking and to stimulate reaching and gripping. Electrodes are taped to the skin over nerves or surgically implanted and then controlled by a computer system under the command of the user. For example, to assist reaching, electrodes can be placed in the shoulder and upper arm and controlled by movements of the opposite shoulder. Through a computer interface, the spinal cord injured person can then trigger hand and arm movements in one arm by shrugging the opposite shoulder.

These systems are useful not just for restoring functional movements. They also help people exercise

paralyzed muscle systems, which can provide significant cardiovascular benefits. So far, relatively few people utilize them because the movements are so robotic, they require extensive surgery and electrode placement, and the computer interface systems are still limited. Bioengineers are working to develop more natural interfaces.

Because the brain plans voluntary movements several seconds before the command is sent out to the muscles, people whose spinal cords no longer carry signals to their limbs might still be able to complete the planning phase in their brains but use a robotic device to carry out the command. A recent experiment used microwires implanted in the motor cortex area of the brain (in this case a monkey's brain) to record brain-wave activity, which was then relayed to a computer that analyzed the data, predicted the movement, and sent the command to a robotic arm. A device such as this could be used to control a wheelchair, a prosthetic limb, or even a patient's own arms and legs.

In the future, researchers expect that these kinds of brain-machine interfaces could be planted directly into the brain using microchips that would do the processing and transmit the results without wires. Work is already being done with hybrid neural interfaces, implantable electronic devices with a biological component that encourages cells to integrate into the host nervous system.

■ **Retraining central pattern generators**

Scientists have known for years that animals' spinal cords contain networks of neurons called *central pattern generators* (CPG) that produce rhythmic flexing and extension of the muscles used in walking. They assumed, however, that the bipedal walking of humans was more dependent on voluntary control than on CPG activation. Therefore, scientists thought that without control from the brain, movements produced by a spinal CPG weren't likely to be useful in restoring successful walking without regulation from the brain. Current research is showing, however, that these networks can be retrained after spinal cord injury to restore limited mobility to the legs.

Using a technique called sensory patterned feedback, researchers are attempting to retrain CPG networks in spinal cord injured patients with special programs that break down walking movements into their component patterns and force paralyzed limbs to repeat them over and over again. In one of these programs, the patient is partially supported by a harness above a moving treadmill while a therapist moves the patient's legs in a stepping motion. Other researchers are experimenting with combining body weight support and electrical stimulation with actual walking rather than treadmill training.

Another technique uses an FES bicycle in which electrodes are attached to hamstrings, quadriceps, and gluteal muscles to stimulate the pedaling motion. Several studies have shown that these exercises can improve gait and balance, and increase walking speed. NINDS is currently funding a clinical trial with paraplegic and quadriplegic subjects to test the benefits of partial weight-supported walking.

■ **Relieving pressure through surgery**

The timing of surgical decompression (alleviating pressure on the spinal cord from fractured or dislocated vertebrae or disks) is a controversial topic. Animal studies have shown that early decompression can reduce secondary damage, but similar results haven't been reliably reproduced in human trials. Other studies have shown neurological improvement without decompression surgery, which has led some to believe that either avoiding or delaying surgery, and using pharmacologic interventions instead, is a reasonable (and non-invasive) treatment for spinal cord injuries. Additional research is needed to determine if early surgical intervention is sufficiently beneficial to offset the risk of major surgery in acute trauma.

■ **Treating pain**

Two thirds of people with spinal cord injury report pain and a third of those rate their pain as severe. Nonetheless, both diagnosis and treatment of post-injury pain still remain a clinical challenge. There is no universally recognized scheme for classifying pain from spinal cord injury, nor is there a uniformly successful medical or surgical treatment to prevent or reduce it. The mainstays of neuropathic pain treatment are antidepressants and anticonvulsants, even though they are not uniformly effective.

Research suggests that spinal cord pain syndromes stem from the spread of secondary damage to spinal cord segments above and below the injury site. Pain can be at the level of the injury or below the level of the injury, even in areas where sensation is limited or absent. Findings indicate that at-level (junctional)

pain probably results from damage to grey and white matter one or more segments above the injury site, whereas pain below the injury results from the interruption of axon pathways and the formation of abnormal connections within the spinal cord near the site of injury.

Studies suggest that functional changes in neurons, which make them hyperexcitable, could be a cause of chronic pain syndromes. Consequently, giving more aggressive treatment for spinal cord injury in the first few hours after injury could limit secondary damage and prevent or reduce the development of chronic pain afterwards.

Investigators are currently testing neuroprotective and anti-inflammatory strategies to calm overexcited neurons. Other studies are also looking at pharmacological options, including sodium channel blockers (such as lidocaine and mexiletine), opioids (such as alfentanil and ketamine), and a combination of morphine and clonidine. Drugs that interfere with neurotransmitters involved in pain syndromes, such as glutamate, are also being investigated. Other researchers are exploring the use of genetically engineered cells to deliver pain-relieving neurotransmitters. These treatments appear to alleviate pain in animal models and in preliminary clinical studies with terminally ill cancer patients.

■ **Controlling spasticity**

The mechanisms of muscle spasticity after spinal cord injury are not well understood. Recent studies indicate that the loss of particular descending axonal pathways most likely results in the decreased activity of inhibitory interneurons, which causes the overreaction of motor neurons to excitatory stimuli.

Unlike treatments for post-injury pain, medical and surgical treatments for spasticity are established and highly successful. These include oral medications that act within the central nervous system (baclofen and diazepam) and one that acts directly on skeletal muscle (dantrolene). For spasticity that is resistant to drug interventions, surgical *rhizotomy* or *myelotomy* is sometimes performed to sever reflex pathways.

Investigators are currently exploring *neuromodulation* procedures based on preliminary results showing that electrical spinal cord stimulation below the injury can modulate spasms. Other techniques used clinically and experimentally involve implanting pump systems that continuously supply antispasmodic drugs such as baclofen.

■ **Improving bladder control**

A promising area of research on treatments for bladder dysfunction involves using electrical stimulation and neuromodulation to achieve bladder control. The current treatment for reflex incontinence includes a surgical procedure that cuts the sacral sensory nerve roots from S2 to S4. With the hope that a cure for spinal cord injury could be imminent, and the reluctance among men to lose any of their already compromised sexual function, few patients are willing to have these nerves cut.

Development of a sacral posterior and anterior root stimulator implant is being explored to better coordinate bladder and sphincter contractions. In preliminary studies people were able to achieve suppression of reflex incontinence and clinically useful increases in bladder volume with the use of the implanted stimulator.

Researchers hope that by combining neuromodulation for reflex incontinence with *neurostimulation* for bladder emptying, the bladder could be completely controlled without having to cut any sacral sensory nerves.

■ **Understanding changes in sexual and reproductive function**

Sperm count in men may or may not change due to spinal cord injury, but sperm motility often does. Researchers are investigating whether or not spinal cord injury causes changes in the chemical composition of semen that make it hostile to sperm viability. Preliminary studies show that the semen of men with spinal cord injury contains abnormally high levels of immunologically active leukocytes, which appear to have a negative impact on sperm motility.

Recent animal studies have revealed what appears to be a neural circuit within the spinal cord that is critical for triggering ejaculation in animal models and may play the same role in humans. Triggering

ejaculation by stimulating these cells might be a better option than some of the current, more invasive methods, such as *electroejaculation*.

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The Future of Spinal Cord Research

Fueled by significant federal and private funding, the past decade of spinal cord injury research has produced a wealth of discoveries that are making the repair of injured spinal cords a reachable goal. This is good news for the 10,000 to 12,000 Americans every year who sustain these traumatic injuries.

Because spinal cord injuries happen predominantly to people under the age of 30, the human cost is high. Major improvements in emergency and acute care have improved survival rates but have also increased the numbers of individuals who have to cope with severe disabilities for the rest of their lives. The cost to society, in terms of health care costs, disability payments, and lost income, is disproportionately high compared to other medical conditions.

Considering the biological complexity of spinal cord injury, discovering successful ways to repair injuries and create rehabilitative strategies that significantly reduce disabilities is not an easy task. Researchers, many of them supported by the NINDS, are actively developing innovative research strategies aimed at making the kinds of exciting new discoveries that will translate into better clinical care and better lives for all.

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Where can I get more information?

For more information on neurological disorders or research programs funded by the National Institute of Neurological Disorders and Stroke, contact the Institute's Brain Resources and Information Network (BRAIN) at:

BRAIN
P.O. Box 5801
Bethesda, MD 20824
(800) 352-9424
<http://www.ninds.nih.gov>

Information also is available from the following organizations:

Christopher and Dana Reeve Foundation

636 Morris Turnpike
Suite 3A
Short Hills, NJ 07078
info@christopherreeve.org
<http://www.christopherreeve.org>
Tel: 973-379-2690 800-225-0292
Fax: 973-912-9433

The Christopher Reeve Foundation raises money to help fund spinal cord injury research and operates the Christopher and Dana Reeve Paralysis Resource Center, located in Short Hills, New Jersey and online at www.paralysis.org.

Miami Project to Cure Paralysis/ Buoniconti Fund

P.O. Box 016960
R-48
Miami, FL 33101-6960
mpinfo@miamiproject.med.miami.edu
<http://www.themiamiproject.org>
Tel: 305-243-6001 800-STANDUP (782-6387)
Fax: 305-243-6017
Supports and conducts research and related programs in the area of spinal cord injury.

National Rehabilitation Information Center (NARIC)

4200 Forbes Boulevard
Suite 202
Lanham, MD 20706-4829
naricinfo@heitechservices.com
<http://www.naric.com>
Tel: 301-459-5900/301-459-5984 (TTY) 800-346-2742
Fax: 301-562-2401

National Spinal Cord Injury Association

1 Church Street
#600
Rockville, MD 20850
info@spinalcord.org
<http://www.spinalcord.org>
Tel: 800-962-9629
Fax: 301-963-1265
Works to help those suffering from the catastrophic results of spinal cord injury and disease. Offers educational materials and also sponsors programs to

empower survivors of spinal cord injury and disease through a toll-free help-line and a network of chapters and support groups.

Paralyzed Veterans of America (PVA)

801 18th Street, NW
Washington, DC 20006-3517
info@pva.org
<http://www.pva.org>
Tel: 202-USA-1300 (872-1300) 800-424-8200
Fax: 202-785-4452

Non-profit organization dedicated to serving the needs of its members—more than 19,000 veterans paralyzed by spinal cord injury or disease, as well as caregivers and others affected by these disabilities—through advocacy, education, and research programs.

Spinal Cord Society

19051 County Highway 1
Fergus Falls, MN 56537
<http://members.aol.com/scsweb>
Tel: 218-739-5252 or 218-739-5261
Fax: 218-739-5262

International advocacy organization that supports research, publishes a newsletter, and sponsors an international network of chapters.

Clearinghouse on Disability Information

Special Education & Rehabilitative Services
Communications & Customer Service Team
550 12th Street, SW, Rm. 5133
Washington, DC 20202-2550
<http://www.ed.gov/about/offices/list/osers>
Tel: 202-245-7307 202-205-5637 (TTD)
Fax: 292024507636

National Institute on Disability and Rehabilitation Research (NIDRR)

U.S. Department of Education Office of Special
Education and Rehabilitative Services
400 Maryland Ave., S.W.
Washington, DC 20202-7100
<http://www.ed.gov/about/offices/list/osers/nidrr>
Tel: 202-245-7460 202-245-7316 (TTY)

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Glossary

agonist - a drug capable of combining with a receptor and initiating action.

antagonist - a drug that opposes the effects of another by physiological or chemical action or by a competitive mechanism.

apoptosis - also called programmed cell death. A form of cell death in which a programmed sequence of events leads to the elimination of old, unnecessary, and unhealthy cells.

arrhythmia - an abnormal heart rhythm. The heartbeats may be too slow, too rapid, too irregular, or too early.

astrocyte - a type of glial cell responsible for neurotransmission and neuronal metabolism.

autonomic dysreflexia - a potentially dangerous complication of spinal cord injury in which blood pressure rises to dangerous levels. If not treated, autonomic dysreflexia can lead to stroke and possibly death.

axial traction - the application of a mechanical force to stretch the spine; used to relieve pressure by separating vertebral surfaces and stretching soft tissues.

axon - the long, thin extension of a nerve cell that conducts impulses away from the cell body.

axonal growth cone - dynamic structures present at the tip of developing and regenerating axons that respond to chemical cues for growth and direction.

central pattern generators (CPG) - neural circuits that produce self-sustaining patterns of behavior independent of their sensory input. Researchers have found evidence of a locomotor CPG in the spinal cord that synchronizes muscle activity during alternating stepping of the legs and feet.

cervical - the part of the spine in the neck region.

coccygeal - the part of the spine at the bottom of the spinal column, above the buttocks.

cytokine - a small protein released by immune cells that has a specific effect on the interactions between cells,

or communications between cells, or on the behavior of cells. dendrite - a short arm-like protuberance from a neuron. Dendrite is from the Greek for "branched like a tree." disc - shortened terminology for an intervertebral disc, a disc-shaped piece of specialized tissue that separates the bones of the spinal column.

electroejaculation - a technique that uses an electric probe to stimulate ejaculation.

embryonic stem cells - undifferentiated cells from the embryo that have the potential to become a wide variety of specialized cell types.

excitotoxicity - a neurological process that is the result of the release of excessive amounts of the neurotransmitter glutamate.

extracellular matrix - the material found around cells composed of structural proteins, specialized proteins, and proteoglycans.

fetal spinal cord cells - cells used by scientists to derive undifferentiated embryonic stem cells for transplant into the damaged spinal cord.

free radicals - highly reactive chemicals that attack molecules and modify their chemical structure.

functional electrical stimulation (FES) - the therapeutic use of low-level electrical current to stimulate muscle movement and restore useful movements such as standing or stepping; also called functional neuromuscular stimulation.

glia - supportive cells in the brain and spinal cord. Glial cells are the most abundant cell types in the central nervous system. There are three types: astrocytes, oligodendrocytes, and microglia. glutamate - an excitatory neurotransmitter.

growth-inhibiting proteins: protein molecules that inhibit axon regeneration.

guidance molecules - molecules that guide axons to their target. Some guidance molecules attract certain axons while repelling others.

hypothermia - abnormally low body temperature.

interneurons - neurons with axons that remain within the spinal cord.

intubation - the process of putting a tube into a hollow organ or passageway, often into the airway.

ligament - a tough band of connective tissue that connects various structures such as two bones.

lumbar - the part of the spine in the middle back, below the thoracic vertebrae and above the sacral vertebrae.

macrophage - a type of white blood cell that engulfs foreign material. Macrophages are key players in the immune response to foreign invaders such as infectious microorganisms. Macrophages also release substances that stimulate other cells of the immune system.

methylprednisolone - a steroid drug used to improve recovery from spinal cord injury.

microglia - glial cells that function as part of the immune system in the brain and spinal cord.

monocyte - a white blood cell that has a single nucleus and can engulf foreign material. Monocytes emigrate from blood into the tissues of the body and evolve into macrophages.

myelin - a structure of cell membranes that forms a sheath around axons, insulating them and speeding conduction of nerve impulses.

myelotomy - a surgical procedure that cuts into the spinal cord.

neural prostheses - prosthetic devices that can respond to signals from the brain.

neurogenic pain - generalized pain that results from nervous system malfunction.

neuromodulation - a series of techniques employing electrical stimulation or the administration of medication by means of devices implanted in the body. These techniques allow the treatment of a range of disorders including certain forms of pain, spasticity, tremor, and urinary problems.

neuron - also known as a nerve cell; the structural and functional unit of the nervous system. A neuron consists

of a cell body and its processes: an axon and one or more dendrites.

neurostimulation - the act of stimulating neurons with electrical impulses delivered via electrodes attached to the brain.

neurotransmitter - a chemical released from neurons that transmits an impulse to another neuron, muscle, organ, or other tissue.

neurotrophic factors - proteins responsible for the growth and survival of neurons.

neutrophil - a type of white blood cell that engulfs, kills, and digests microorganisms.

oligodendrocyte - a type of nerve cell in the brain and spinal cord that surrounds and insulates axons.

olfactory ensheathing glia - non-myelinating glial cells that ensheath olfactory axons within both the PNS and CNS portions of the primary olfactory pathway. They are being used in experiments to build bridges between damaged areas of the spinal cord.

paralysis - the inability to control movement of a part of the body.

paraplegia - a condition involving complete paralysis of the legs.

pressure sore (also known as a pressure ulcer or bed sore) - a reddened area or open sore caused by unrelieved pressure on the skin over bony areas such as the hip-bone or tailbone.

quadriplegia - a condition involving complete paralysis of the legs and partial or complete paralysis of the arms.

receptor - a structure on the surface or interior of a cell that selectively receives and binds to a specific substance.

regeneration - repair, regrowth, or restoration of tissues; opposite of degeneration.

rhizotomy - an operation to disconnect specific nerve roots in order to stop severe spasticity.

sacral - refers to the part of the spine in the hip area.

Schwann cell - the cell of the peripheral nervous system that forms the myelin sheath.

spasticity - increased tone in muscles of the arms and legs (due to lesions of the upper motor neurons).

spinal shock - a temporary physiological state that can occur after a spinal cord injury in which all sensory, motor, and sympathetic functions of the nervous system are lost below the level of injury. Spinal shock can lower blood pressure to dangerous levels and cause temporary paralysis.

stem cell - special cells that have the ability to grow into any one of the body's more than 200 cell types. Unlike mature cells, which are permanently committed to their fate, stem cells can both renew themselves and create cells of other tissues.

synapse - a specialized junction between two nerve cells. At the synapse, a neuron releases neurotransmitters that diffuse across the gap and activate receptors situated on the target cell.

T-cell - an immune system cell that produces substances called cytokines, which stimulate the immune response. thoracic - the part of the spine at the upper-back to mid-back level.

vertebrae - the 33 hollow bones that make up the spine.

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