A bolt out of the blue

Dealing with the aftermath of spinal cord injury
IT HAPPENS IN AN INSTANT: On a hot summer day, a handsome, laughing 19-year-old delivers a Tarzan yell, dives into a swimming pool…and nothing’s ever the same again. The water was too shallow. The boy’s forehead hit the bottom of the concrete pool with sickening force, snapping his skull backward and crushing the C1 and C2 vertebrae.

The Miami Project to Cure Paralysis/Buoniconti Fund estimates that 11,000 spinal cord injuries (SCIs) occur each year. Eighty percent of the injuries happen in men, and more than half occur between the ages of 16 and 30.

The population most prone to SCI is described as “risk takers”—young adult men who play hard, drive fast, and hate to walk away from a fight. The most common causes of SCI are motor vehicle accidents, acts of violence, sports injuries, and falls. Not surprisingly, a high correlation exists between alcohol and drug abuse and SCI.

Before World War II, the life expectancy of someone with an SCI ranged from a few months to 10 years from onset of injury; most died within a year. Back then, the leading causes of death were kidney failure and sepsis.

Life expectancies remain substantially below normal today, particularly for people with tetraplegia (quadriplegia) who are ventilator-dependent. Premature death in people with an SCI is usually a result of compromised respiratory function related to pneumonia. Sadly, between 10% and 20% of people who sustain an SCI don’t survive to make it to the hospital. (Acute respiratory failure is the leading cause of death in high cervical injury.) Another 3% die while in acute care.

Your first assessment of a patient with SCI can be scary—and heartbreaking. The patient’s likely someone who, in the blink of an eye, went from being a rambunctious guy ready to take on the world to the victim of a life-threatening injury who feels helpless, hopeless, and terrified.

In this article, I’ll help you understand
what happens to the body in an SCI, how to minimize the damage and loss of function, and what treatments are available to help patients achieve the best possible outcome. I’ll also give you a peek at some promising research. Let’s start with an examination of the anatomy of the spinal cord.

**Wired**

The brain and spinal cord make up the central nervous system (CNS). The spinal cord transmits messages that coordinate movement and sensation. Surrounded and protected by cerebrospinal fluid, the cord extends from an opening in the base of the skull (foramen magnum) through a protective passageway formed by the cavities of the vertebrae.

The cord is composed of 31 pairs of spinal nerves: 8 cervical, 12 thoracic, 5 lumbar, 5 sacral, and 1 coccygeal. Each nerve has a ventral root and a dorsal root. The dorsal roots are sensory and transmit sensory impulses—pain, temperature, touch, and position sense (proprioception)—from tendons, joints, and body surfaces.

Each muscle in the body is supplied by a particular level or segment of the spinal cord and by its corresponding spinal nerve. A group of muscles primarily supplied with nerves (innervated) by the motor fibers from a single nerve root is known as a myotome.

A dermatome is an area of the skin supplied by nerve fibers originating from a single dorsal nerve root. Each is named according to the spinal nerve that supplies it. Those on the trunk resemble horizontal bands; those on the extremities are vertical and elongated. There’s considerable overlap of innervation between adjacent dermatomes. In other words, if function is lost in one spinal nerve, sensation usually isn’t completely gone because of overlap from the adjacent spinal nerves. Sensitivity, however, will be reduced. See **Dermatomes** for a detailed map.

The levels of the spinal cord segments don’t relate exactly to the levels of the vertebral bodies because there are seven cervical vertebrae and eight cervical nerve roots branching off from the cord. Therefore, damage to the bone at a particular level doesn’t necessarily mean damage to the spinal cord at the same level.
The thoracic spinal cord segments are compressed into the level between the T1 and T10 vertebrae. The lumbar and sacral segments are at the level of the T11 and T12 vertebrae. In adults, the spinal cord ends around the level of the disc between the T12 and L1 vertebrae.

Below T12 is an extension of the spinal cord called the cauda equina (literally translated from Latin as “horse’s tail”). The cauda equina contains spinal nerves that continue downward before passing out between the vertebrae.

Now let’s take a look at what happens when trauma disrupts the workings of this exquisitely complex structure.

**Shattered**

Injuries to the spinal cord most often stem from a sudden, traumatic blow to the spine that fractures, dislocates, crushes, or compresses one or more vertebrae. The injuries may also result from a penetrating wound or deep laceration that breaches the vertebrae and damages or severs the cord. Injury can disrupt communication between the brain and muscles when neurons lose their connection to axons located below the site of injury. These neurons may still be alive, but the path of communication is cut.

Clinical manifestations of SCI depend on the type and level of damage. The type of injury refers to the cord itself. And here’s an important point to remember: Injury to the spinal cord is a dynamic process, meaning the full extent of the injury may not be evident right away. For example, an incomplete cord lesion may evolve into a more complete lesion, or the injury level may rise one or two spinal levels in the first hours to days following the trauma.

The degree of spinal cord involvement in an SCI is classified as incomplete (partial) or complete.

In an *incomplete injury*, the spinal cord is partially able to transmit messages to and from the brain. The patient retains some motor and/or sensory function below the level of the injury. Incomplete spinal cord lesions are classified according to the area of damage. The cluster of physical manifestations of the injury is given a specific label. **Central cord syndrome** is characterized by motor deficit and sensory loss in the upper extremities. Injury is in the cervical area. In **anterior cord syndrome**, the patient experiences loss of pain and temperature sensation and motor function below the level of injury. Light touch, position, and vibration sensation are preserved. **Lateral cord syndrome** (also called Brown-Séquard’s syndrome) appears as ipsilateral paralysis or paresis; ipsilateral loss of touch, pressure, and vibration sensation; and contralateral loss of pain and tem-

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**Dermatomes**

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**did you know?**

Because spinal cord injury is a dynamic process, the full extent of the patient’s injury may not be known right away.
Effects of spinal cord injuries

Central cord syndrome
- Characteristics: Motor deficits (in the upper extremities compared to the lower extremities; sensory loss varies but is more pronounced in the upper extremities); bowel/bladder dysfunction is variable, or function may be completely preserved.
- Cause: Injury or edema of the central cord, usually of the cervical area.

Anterior cord syndrome
- Characteristics: Loss of pain, temperature, and motor function is noted below the level of the lesion; light touch, position, and vibration sensation remain intact.
- Cause: The syndrome may be caused by acute disk herniation or hyperflexion injuries associated with fracture-dislocation of vertebra. It also may occur as a result of injury to the anterior spinal artery, which supplies the anterior two-thirds of the spinal cord.

Brown-Séquard syndrome (lateral cord syndrome)
- Characteristics: Ipsilateral paralysis or paresis, together with ipsilateral loss of touch, pressure, and vibration and contralateral loss of pain and temperature.
- Cause: The lesion is caused by a transverse hemisection of the cord (half of the cord is transected from north to south), usually as a result of a knife or missile injury, fracture/dislocation of a unilateral articular process, or possibly an acute ruptured disk.

temperature sensation (see Effects of spinal cord injuries).

Complete injury is characterized by the absence of message transmission and motor and sensory function below the level of the injury. Depending on where it is, a complete spinal cord lesion can result in permanent paraplegia (paralysis of the lower body) or quadriplegia (paralysis of all four extremities). See the American Spinal Injury Association impairment scale for an alternate standard of classification. And, see Check out the circuitry for tests that may be ordered to evaluate the injury.

The ripple effect
An SCI affects more than just sensation and movement. Let’s look at what’s happening to the body’s systems just after the initial insult.

Soon after the injury, petechial hemorrhages are noted in the central gray matter of the cord. Hemorrhagic areas in the gray matter are grossly visible within 1 hour; within 4 hours there may be infarction in the gray matter. Additional injury may accrue over days or weeks because of bleeding, swelling, inflammation, and fluid accumulation in and around the spinal cord.

Ischemia, the most prominent event postinjury, can occur up to 2 hours after the initial injury; it’s made worse by loss of autoregulation of the spinal cord microcirculation. Blood flow is decreased and depends on the systemic arterial pressure if the patient is hypotensive or has vasogenic spinal shock. Hypoxia may occur as a result of inadequate airway maintenance and ventilation.

If treatment isn’t provided, or if the patient doesn’t respond to treatment within 24 hours of the initial insult, permanent damage is done due to edema. Edema secondary to the inflammatory response is particularly harmful because of the lack of space within the spinal column for tissue expansion. The resulting compression above and below the injury increases ischemic damage.

The spinal cord loses function below the level of the lesion immediately following the injury. Spinal shock may also immediately follow the injury; it’s characterized by decreased reflexes and flaccid paralysis. Spinal shock usually appears at the time of injury and spontaneously resolves days to weeks later. It affects musculoskeletal, bowel, and bladder function.

Neurogenic shock is caused by the sudden disruption of the sympathetic nervous system that occurs with an SCI. It’s characterized by hypotension, bradycardia, hypothermia, and warm, dry extremities. Peripheral vasodilatation, venous pooling, and decreased cardiac output occur.

Now that you understand what your

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**American Spinal Injury Association impairment scale**

<table>
<thead>
<tr>
<th>Grade</th>
<th>Description</th>
</tr>
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<tbody>
<tr>
<td>A</td>
<td>Complete: No motor or sensory function is preserved in the sacral segments S4-S5.</td>
</tr>
<tr>
<td>B</td>
<td>Incomplete: Sensory but no motor function is preserved below the neurologic level and includes the sacral segment S4-S5.</td>
</tr>
<tr>
<td>C</td>
<td>Incomplete: Motor function is preserved below the neurologic level, and more than half of key muscles below the neurologic level have a muscle grade less than 3.</td>
</tr>
<tr>
<td>D</td>
<td>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.</td>
</tr>
<tr>
<td>E</td>
<td>Normal: Motor and sensory functions are normal.</td>
</tr>
</tbody>
</table>

**Muscle grades**

<table>
<thead>
<tr>
<th>Grade</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>0</td>
<td>Total paralysis</td>
</tr>
<tr>
<td>1</td>
<td>Palpable or visible contraction</td>
</tr>
<tr>
<td>2</td>
<td>Active movement, gravity eliminated</td>
</tr>
<tr>
<td>3</td>
<td>Active movement against gravity</td>
</tr>
<tr>
<td>4</td>
<td>Active movement against some resistance</td>
</tr>
<tr>
<td>5</td>
<td>Active movement against full resistance</td>
</tr>
<tr>
<td>NT</td>
<td>Not testable</td>
</tr>
</tbody>
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patient’s body is going through, let’s look more closely at what you need to do to provide the best possible care in one of the worst possible situations.

**Breathing is fundamental**
When a patient presents with a possible SCI, airway and ventilation management are the top priorities. Individuals with injuries to the lumbar or sacral vertebrae usually retain use of the muscle groups needed for breathing. Injuries to C1 through C3, however, present a particular challenge because of the total loss of respiratory muscle function. In certain cases where the injury occurs below C4, spinal cord edema and hemorrhage can affect the function of the phrenic nerve and cause respiratory insufficiency, necessitating ventilatory support.

Once the patient gets past the early crisis, he’ll still need help with breathing and airway clearance. To achieve these goals, provide aggressive pulmonary toilet, which can include chest physiotherapy or assisted coughing every 4 hours to help loosen and clear secretions. In assisted coughing, chest or abdominal thrusts are administered synchronously with the patient’s own efforts at coughing.

Implement measures to promote lung and chest wall expansion to improve breathing, such as routine turning from prone to supine on a turning frame. Help the patient into a sitting or upright position as tolerated. Encourage him to perform exercises that strengthen the accessory muscles used in breathing. A movement as simple as shrugging the shoulders can strengthen the trapezius muscles.

Patients with an SCI are at risk for respiratory complications, including:

- **atelectasis.** In this complication of respiratory insufficiency, the lungs partially collapse because the alveoli and terminal airways don’t receive adequate airflow to keep them inflated.

- **pulmonary embolism.** This is the second-leading cause of death in patients with an SCI within the first year after injury.

- **bacterial infections.** Patients with an SCI have an impaired ability to clear secretions. Retained secretions can plug up bronchi and lead to atelectasis and, eventually, pneumonia.

Because certain patients with an SCI may have decreased sensation of respiratory distress associated with head injury or effects of alcohol and/or drug ingestion,

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**Check out the circuitry**
A patient suspected of having a spinal cord injury will be assessed for sensory function and movement. If pain, signs of weakness, or neurologic injury are present, the following tests may be ordered:

- **X-ray**, which can reveal vertebral abnormalities, tumors, fractures, or degenerative changes

- **computed tomography scan**, which may provide a clearer picture of abnormalities seen on X-ray

- **magnetic resonance imaging** (MRI), which is helpful in visualizing the spinal cord and identifying herniated disks, blood clots, or other masses that may be compressing the cord. MRI may be contraindicated in trauma patients supported by certain life-support machines or cervical traction devices.

- **myelography**, which helps to visualize spinal canal structures (spinal nerves and roots). It’s used when an MRI isn’t possible or when it may yield information not provided by other tests. The technique combines X-ray and fluoroscopy, and can detect abnormalities like blockage and infection.
your first hint of impending respiratory failure from mucous plug may be anxiety and a higher respiratory rate. Fever and purulent sputum may be signs of pneumonia. Chest X-rays can show atelectasis or pneumonia. Swallowing studies may identify an increased risk of aspiration, and fluoroscopy or ultrasound may reveal paralysis of the diaphragm.

Keeping the pump primed
More than 60% of patients with an SCI experience organ injuries that ultimately may affect their cardiovascular status. That’s why it’s so important to provide volume resuscitation during the first 24 to 48 hours after the injury. Continuous hemodynamic monitoring is crucial to the patient’s survival during this vulnerable phase.

Even when organ injury is absent, cardiovascular and hemodynamic status must be assessed and stabilized during emergency and acute care. The patient should be placed on continuous electrocardiographic monitoring because bradycardia (slow heart rate) and asystole (lack of electrical activity) are common cardiac signs in acute injury. Symptomatic bradycardia may result from loss of sympathetic outflow to the heart with subsequent vagal hyperactivity. Bradycardia and hypotension may be treated with atropine. Use of a pacemaker may be indicated to ensure adequate cardiac output and spinal cord perfusion.

To treat decreased cardiac output, the health care provider may order dopamine or other vasopressor agents to maintain the mean blood pressure above 80 mm Hg. Measure pulmonary artery wedge pressure and cardiac output to assess circulatory status.

Altered tissue perfusion related to decreased cardiac output and peripheral pooling of blood below the level of injury will typically be your next consideration. First, assess for signs of diminished tissue perfusion, like decreased blood pressure, restlessness, confusion, and diminished or absent peripheral pulses. Then, implement appropriate measures to maintain adequate tissue perfusion. Avoid any procedure that causes vagal stimulation, like suctioning, unless it’s necessary. The health care provider may order anticholinergics like atropine or sympathomimetics like dopamine to increase cardiac output and maintain arterial pressure.

Deep vein thrombosis (DVT) is a common problem in SCI. The estimated incidence in the initial period after injury ranges from 47% to 72%, depending on the diagnostic tool used.

Unless contraindicated, prophylactic compression hose or sequential compression devices should be applied to the lower extremities for the first 2 weeks following the injury. Low-molecular-weight heparin or unfractionated heparin should also be administered for 2 to 3 months after the original injury. An inferior vena cava filter may be used when anticoagulation is contraindicated or ineffective. In this procedure, a device is placed in the large vein in the abdomen that connects the leg veins to the heart to trap emboli traveling up from
the legs. The filter is commonly placed below the renal vein.

Other strategies to prevent peripheral pooling of blood and increase venous return include passive range-of-motion exercises, which should be done at least three times a day, and elevation of the lower extremities for 20 minutes during each shift, unless contraindicated.

**Ups and downs**
A potentially life-threatening complication, autonomic dysreflexia is well recognized in 48% to 90% of all patients injured at T6 or above. Ironically, incidence increases just as patients are recovering from spinal shock—a real one-two punch. As reflexes return, various neurotransmitters, like nor-epinephrine and dopamine, are released, causing piloerection (goose bumps), skin pallor, and severe arterial vasoconstriction. The result is sudden elevation in blood pressure above the level of injury.

Autonomic dysreflexia presents as a dramatic rise in blood pressure with associated excessive sweating, facial flushing, and tachycardia. The patient may complain of blurry vision, headache, and a feeling of anxiety. Changes in the level of consciousness, seizures, and apnea may result in the most severe cases. See Going hyper! for more on this syndrome.

Complications from the hypertension caused by autonomic dysreflexia may include myocardial infarction and retinal or cerebral hemorrhage. Medical treatment includes use of an antihypertensive like nifedipine (Procardia), phenoxybenzamine (Dibenzyline), or mecamylamine (Inversine). Preventive measures include good bladder and bowel care; fecal impaction and bladder distension are common triggers of autonomic dysreflexia.

Orthostatic (postural) hypotension can be a significant issue when the patient has an incomplete injury that’s progressed past the acute phase so that some degree of mobility has returned. The patient lacks the normal sympathetic tone in the lower extremities, so blood tends to pool in the lower body when he’s upright.

To lower the risk of orthostatic hypotension, apply an abdominal binder, thigh-high antiembolism stockings, or elastic bandages before lifting the patient from the supine to the upright position. Perform the position change slowly to allow the body to adjust. Adequate hydration can also help control the problem.

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**Going hyper!**
Autonomic dysreflexia is related to the loss of autonomic nervous system control below the level of injury; those who are most at risk have an injury at or above T6. You may hear this condition referred to as “going hyper.”

To lower the probability of autonomic dysreflexia occurring, avoid stimulating the sympathetic nervous system and eliciting a reflex response below the level of cord injury by:

- preventing distension of the bladder and bowel by ensuring the patient follows his elimination programs on schedule
- ensuring that all clothing and the urinary catheter leg-bag strap aren’t binding; excess pressure on any part of the body can trigger autonomic dysreflexia
- providing good nail care; ingrown toenails can produce a reflex response
- applying topical anesthetic as ordered to existing pressure ulcers or other skin injury; even a minor skin trauma can generate a reflex response.

Signs and symptoms of autonomic dysreflexia to look out for include:

- a sudden rise in blood pressure
- seeing spots or having blurred vision
- pounding headache
- nasal stuffiness
- facial flushing
- red blotches on the chest
- excessive sweating above the level of injury
- goose bumps
- cool, clammy skin
- nausea
- anxiety
- bradycardia
- flushing.

If autonomic dysreflexia occurs, implement measures to promote lower extremity venous pooling and decrease the blood pressure, such as raising the head of the bed and removing any abdominal binder, antiembolism stockings, or elastic bandages. The health care provider will likely order a rapid-onset, short-acting antihypertensive, like nifedipine (Procardia), nitroglycerin, phenoxybenzamine (Dibenzyline), or mecamylamine (Inversine). Frequently monitor the patient’s blood pressure and pulse.
A stabilizing influence
A patient with a suspected SCI will be brought to the hospital with his head, neck, and spine immobilized. Once diagnosed, he may need skeletal traction with calipers or Crutchfield, Vinke, or Gardner-Wells tongs to stabilize or bring his spine back into proper alignment, after which reduction (the positioning of bone fragments or dislocated bones into correct alignment) can take place. Most experts recommend gradual application of weight in 5- to 10-pound increments to achieve reduction. A careful neurologic exam should be performed and documented and a lateral X-ray should be checked with each addition of weight. A general rule is that 5 to 10 pounds of traction should be applied to counteract the weight of the head, and then 5 pounds added for each cervical level above the level of the injury. Once reduction is achieved, the weight is reduced to just the amount needed to maintain correct alignment.

A halo may be used initially with traction or later after the tongs are removed. The stainless steel circle is fixed onto the skull with four pins; a frame attaches it to a removable halo vest. This setup helps to immobilize the cervical spine while allowing for early ambulation.

Closely monitor the patient with a halo for pin movement. Assess and report any signs of pin-site infection, such as fever, redness at the site, pain, and swelling. Keep hair away from pin sites, and clean the sites according to your facility’s policy. Teach visitors and other caregivers to avoid touching the pins or tongs. Proper hand hygiene is the key to preventing infection.

Surgery may be performed to aid in reducing spinal fracture or dislocation or to decompress the spinal cord. It’s also indicated to remove fragments of bone, foreign objects, or bone fragments that may be compressing the spine. The goal of surgery is to gain greater neurologic recovery and to protect the patient from further neurologic dete-
rioration or intraspinal compression. Early surgical stabilization can sometimes eliminate the need for cervical traction in some patients.

See Don’t move! for details.

Problems of the bowel
Bowel and bladder dysfunction are common in patients with an SCI. For example, constipation can occur as a result of decreased gastrointestinal (GI) motility due to loss of autonomic nervous system function, decreased activity, lost CNS control of the defecation reflex, and diminished gravity filling of the lower rectum because of horizontal positioning.

Regularly assess the patient’s bowel sounds and watch for signs of constipation. Encourage the patient to eat high-fiber foods, stay well hydrated, and consume hot drinks to stimulate peristalsis. Suppositories and enemas may be ordered as appropriate. An individualized bowel management program should be initiated based on the patient’s normal daily routine.

Decreased GI motor activity can also contribute to a condition called paralytic ileus, which is the loss of movement in the small intestine that causes gas and fluid buildup. It usually lasts a few days after injury. Symptoms can be treated with an orogastric or nasogastric tube to decompress the stomach.

Problems of the bladder
In the immediate aftermath of an SCI, the bladder is atonic due to disruption of the spinal cord and the onset of spinal shock. So placing an indwelling urinary catheter and carefully monitoring intake/output are standard procedures in the early stages of an SCI. In addition, a catheter is often needed after fluid resuscitation. The catheter stays in place until volume is normalized and the patient’s stable, usually 3 to 4 days postinjury.

A patient with SCI may also have bladder dysfunction resulting in urine retention or urinary incontinence. Carefully monitor input/output and hemodynamic status, especially if the patient’s receiving intravenous therapy and has a catheter in place.

As I discussed earlier, spinal shock occurs at the time of injury and spontaneously resolves. It affects musculoskeletal, bowel, and bladder function. Following spinal shock, start a program of having the patient void at regular intervals. To do this, stimulate the trigger zones of the sacral reflex by tapping the suprapubic area or stroking the abdomen. Teach the patient to keep his amount of fluid intake fairly constant throughout the day to avoid excessive bladder distension.

The patient’s health care provider may order a centrally acting muscle relaxant, like baclofen (Lioresal), or a smooth muscle relaxant, like oxybutynin (Ditropan), to decrease spastic contraction of the bladder and the tone of the external urinary sphincter.

Assess and report any signs of urinary tract infection, such as cloudy urine, fever, increase in spasticity, and positive urinalysis.

The skin at risk
A pressure ulcer is defined as any wound caused by unrelieved pressure resulting in damage to the underlying tissue. In patients with an SCI, immobility and lack of sensation can quickly raise the risk for developing pressure ulcers. Urinary and/or fecal incontinence can contribute to skin breakdown. Hypotension can decrease the amount of oxygenated blood to the skin, which can compromise skin integrity and

did you know?
Patients with tetraplegia arising from injury to C1 through C4 are more likely to die from diseases of pulmonary circulation compared with the general population because of lack of respiratory effort caused by paralysis.
its ability to fight infection. Splints and braces can also contribute to the formation of pressure ulcers where the devices make contact with the skin.

Preventive nursing care must include proper positioning, turning, skin care, and good nutrition. Areas of skin over bony prominences are especially vulnerable. The sacrum/coccyx area is the most common site for pressure ulcers, with the heels a close second. Turning protocols vary from patient to patient, but a good rule of thumb for a chair-bound patient is repositioning every 15 minutes.

**It’s a real pain**
A patient with an SCI may experience headache, neck pain, or upper shoulder and arm pain. Administer ordered analgesia and assess for effective symptom relief. Also, ensure that the cervical spine remains immobilized by using neck supports as needed. Shoulder massage is comforting, relaxing, and a powerful remedy for all manner of aches and pains.

**Hot and cold**
A patient with a lesion above T1 will have trouble maintaining normal body temperature. This is due to the interruption in the autonomic feedback system between the area below the level of the injury and the hypothalamus; loss of vasomotor tone below the level of injury is also a contributing factor. The risk of hypothermia may be related to reduced heat generation associated with limited body movement.

Carefully monitor the patient for signs of ineffective thermoregulation and implement measures to prevent hypothermia or hyperthermia. You can apply a warming or cooling blanket to correct body temperature as needed.

**Fragile, handle with care**
Patients with an SCI are especially vulnerable to trauma from falls and burns. Follow your facility’s policy for safe transfers to reduce the risk of falls when, for example, you move the patient from a wheelchair to a bed or exam table.

Patients may have diminished sense of hot and cold. Let food and beverages cool slightly before serving to avoid burning the patient’s lips, mouth, and throat, and assess bath water temperature per your facility’s policy.

**Everything changes**
An SCI doesn’t affect just the physical part of the patient. Addressing his fear and anxiety related to the loss of sensory and motor function should be one of your primary considerations.

Generally help the patient to orient himself to his new temporary environment, with all of its strange equipment and unfamiliar routines. Strategically placed mirrors can help broaden his view of the surroundings. Explain all diagnostic tests in layperson’s terms and reinforce the health care provider’s explanations; clarify any misconceptions and answer all questions about his injury and prognosis.

Impaired physical mobility associated with tetraplegia, spasticity, decreased motivation, pain, and weakness can lead to self-care deficits. A referral to an occupational therapist can help your patient develop a realistic plan for meeting his daily physical needs. The health care provider may order baclofen to reduce spasticity. It may also help to schedule care when the patient most feels up to it, like when analgesia is at its peak. Allow ample time for tasks so he doesn’t feel rushed and frustrated. Make sure the objects he can use independently are within his reach.

Your patient needs time to adjust to his diagnosis; all of the implications won’t sink in right away. Provide information on technology available to improve his independence, and encourage his active participation in all decision-making processes. Suggest resources that can assist with managing the immediate trauma and provide
guidance for long-term needs. Arrange for consults with specialists who can counsel the patient on how best to make the adjustment to living with an SCI (see Help’s out there).

His immediate family will need education, support, and guidance too. Tongs or halos can be alarming for people who’ve never seen them before. Explain what the traction device is and what it does. The same goes for the turning frame and ventilator.

Clearing hurdles
Once the immediate crisis is over and the patient is stabilized, he’ll need to begin his all-important rehabilitation program. Rehab can improve his overall health and teach him how to realize the full potential of his abilities. Rehab can teach him new ways to do everyday tasks and what things he needs to do to stay strong and healthy.

As part of the rehab program, functional goals will be set; these are the realistic expectations of activities the patient should be able to do at his level of injury. To be meaningful, developing functional goals should be a collaborative effort between the patient and his medical team. Rehab continues well beyond hospital discharge.

Rehab techniques are constantly undergoing improvement. Let’s take a look at some high-tech advances that patients with an SCI may be able to take advantage of in the near future.

Hope on the horizon
Research by biomedical engineers using robotics may revolutionize treatment for patients with an SCI. Some works in progress include a “bladder pacemaker” that may effectively treat urinary incontinence and a neuroprosthetic microchip implant that would allow certain patients to walk again.

Functional electrical stimulation (FES) artificially generates neural activity to overcome lost function. A series of electrodes stimulates the muscles to recreate walking motions. In certain cases, muscle mass may be preserved. It’s theorized that in certain

Help’s out there
Organizations specializing in spinal cord injury include the following:

Christopher Reeve Paralysis Foundation/Paralysis Resource Center
500 Morris Ave.
Springfield, NJ 07081
http://www.christopherreeve.org

Miami Project to Cure Paralysis/Buoniconti Fund
P.O. Box 016960, R-48
Miami, FL 33101
http://www.themiamiproject.org

National Rehabilitation Information Center
4200 Forbes Blvd., Suite 202
Lanham, MD 20706
http://www.naric.com

National Spinal Cord Injury Association
6701 Democracy Blvd., #300-9
Bethesda, MD 20817
http://www.spinalcord.org

Paralyzed Veterans of America
801 18th St. NW
Washington, DC 20006-3517
http://www.pva.org
In this issue...

A bolt out of the blue: Dealing with the aftermath of spinal cord injury

Case Study

Patients with spinal cord injuries (SCI) experience a variety of physical and emotional challenges. Some may be able to lead fulfilling lives, while others may struggle with severe disabilities.

1. Describe the physical and emotional challenges faced by patients with SCI.
2. Identify the leading causes of death among people with SCI.
3. Discuss the need for integrated care in the treatment of SCI.

Learn more about it

For more information on SCI, visit the following resources:


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A bolt out of the blue: Dealing with the aftermath of spinal cord injury

GENERAL PURPOSE: To provide the registered professional nurse with an overview of what happens to the body in a spinal cord injury (SCI), how to minimize the damage and loss of function, and what treatments are available to help patients achieve the best possible outcomes.

LEARNING OBJECTIVES: After reading the preceding article and taking this test, you should be able to: 1. Explain the pathophysiology and discuss the types of SCI. 2. Identify the complications of and treatment strategies for SCI.

1. Premature death in people with an SCI is usually a result of
   a. kidney failure.
   b. compromised respiratory function related to pneumonia.
   c. sepsis.

2. How many pairs of spinal nerves are in the spinal cord?
   a. 31
   b. 12
   c. 8

3. An area of skin supplied by the nerve fibers originating from a single nerve root is called
   a. an innervated nerve.
   b. a myotome.
   c. a dermatome.

4. In adults, the spinal cord ends around the level of
   a. T1 and T10.
   b. T11 and T12.
   c. T12 and L1.

5. The degree of spinal cord involvement from an injury is classified as
   a. partial or central.
   b. incomplete or complete.
   c. central or lateral.

6. A patient who presents with a motor deficit and sensory loss in the upper extremities is likely to have
   a. central cord syndrome.
   b. anterior cord syndrome.
   c. Brown-Séquard syndrome.

7. According to the American Spinal Injury Association impairment scale, when no motor or sensory function is preserved in S4-S5, the injury is graded as
   a. A = complete.
   b. B = incomplete.
   c. C = normal.

8. Which of the following is the most prominent event post-SCI?
   a. petechial hemorrhage
   b. inflammation
   c. ischemia

9. Which type of shock is caused by the sudden disruption of the sympathetic nervous system that occurs with an SCI?
   a. vasogenic spinal shock
   b. neurogenic shock
   c. spinal shock

10. Injuries to which of the following areas would result in total loss of respiratory function?
    a. C1 through C3
    b. the lumbar vertebrae
    c. the sacral vertebrae

11. Which of the following respiratory complications is the second-leading cause of death in patients with an SCI within the first year of injury?
    a. atelectasis
    b. pulmonary embolism
    c. bacterial infection

12. Autonomic dysreflexia presents as
    a. a dramatic rise in blood pressure.
    b. a dramatic drop in blood pressure.
    c. severe bradycardia.

13. Oxybutynin (Ditropan) is used to
    a. decrease spastic contraction of the bladder.
    b. increase spastic contraction of the bladder.
    c. stimulate peristalsis in the bowel.

14. Baclofen (Lioresal) may be ordered to
    a. increase contraction of the muscles.
    b. reduce muscle spasticity.
    c. control pain.

15. If autonomic dysreflexia occurs, which of the following interventions should be used?
    a. Lower the head of the bed.
    b. Raise the head of the bed.
    c. Place an abdominal binder on the patient.

16. Which of the following diagnostic tests can detect abnormalities like blockage and infection in the spinal canal structures?
    a. computed tomography (CT) scan
    b. magnetic resonance imaging (MRI)
    c. myelography

17. After applying additional weight to the patient in skeletal traction, which diagnostic test should be ordered?
    a. lateral X-ray
    b. CT scan
    c. MRI

18. Skeletal traction is used for a patient with an SCI primarily to
    a. immobilize him.
    b. properly align his spine.
    c. decrease pressure to his spine.

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