Spinal Cord Injuries

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A recent article by Dr Robert Heimburger in the Journal was titled, “Is there hope for return of function in lower extremities paralyzed by spinal cord injury?” This question can be answered by showing that innovative attempts are currently being pursued in the hope, as requested by Dr Heimburger, to improve the neurologic results of patients who tragically sustain a spinal cord injury (SCI).

Dr Heimburger paid tribute in his article to Dr LW Freeman, who spent the major portion of his laboratory life producing evidence that axons could progress in an experimental animal from the cut end of a divided intercostal nerve when the distal end of the nerve is implanted into the lower segment of an anatomically transected spinal cord. Freeman reported that large numbers of animals who underwent this procedure demonstrated the return of muscular function and walking ability in their paralyzed limbs.

Fortunately, only a very few patients sustain an anatomically transected spinal cord as a result of their injury. What does occur in an overwhelming number of SCI patients is not an anatomic transection of the cord, but a physiologic transection of the injured cord, which results in complete lack of neurologic activity below the site of injury.

Laboratory efforts today are directed toward development of axonal activation at the site of the SCI. This effort should be strenuously continued. But improvement in the future treatment of an SCI is also of extreme importance and, if successful, elevating the longterm results of spinal cord injuries might well occur.

When a spinal cord injury is surgically treated today, efforts are directed to firmly stabilizing the vertebral column, but little, if any, attention is focused precisely on the injured spinal cord, which, after all, is the critical area of any such injury. If treatment could be directed specifically to the injured spinal cord, perhaps longterm improvement in postoperative motor function might follow.

Studies have shown little difference if an operation is carried out early or later in the presence of an SCI. Regardless of the timing of the operation, all that apparently may be done to treat an edematous spinal cord is to perform a decompressive laminectomy. This maneuver does little to decrease the high interstitial pressure present in an edematous spinal cord restricted by the firm, enveloping dura mater that surrounds the swollen spinal cord.

The reason there is a strong reluctance to open the dura over a swollen edematous spinal cord is that such an opening can lead to instant extrusion of edematous spinal cord material. Even if a traumatized spinal cord is exposed after an opening in the dura mater, nothing is done specifically to the injured spinal cord that might lead to improved clinical results.

Dr Heimburger’s article asked whether there might be hope that something could be done in the future for patients with an SCI. This hope is currently addressed in a recent article entitled “Can the standard treatment of acute spinal cord injury be improved: perhaps the time has come.” In this recent article, two considerations are presented that propose a direct surgical approach to a traumatized spinal cord, which should be done as early as possible after SCI. The problem of extrusion of edematous spinal cord tissue after the opening of the dura is first addressed. This problem can be controlled by making two small midline longitudinal incisions in the dura mater 1 to 1½ cm directly above and below the area of spinal cord impaction and edema formation. These two longitudinal incisions are slowly drawn toward each other over a 10-minute period until the longitudinal incisions connect directly over the major spinal cord injury site. This technique prevents edematous material from being extruded at the site of the SCI because the material is slowly squeezed up and down the spinal cord (comparable to squeezing the middle of a toothpaste tube with one’s thumb—a high interstitial tissue pressure forces itself into areas of lower interstitial pressure).

After an injured spinal cord is exposed after the widely opened dura mater, an intact omental pedicle can be placed directly on the site of the SCI. The reason for this maneuver is based on the omentum’s enormous ability to absorb edema fluid. The edema fluid that is present at
the site of an SCI is a plasma transudate, in which fibrinogen is activated to form fibrin (scar). Laboratory evidence has shown that placing the omentum on an injured spinal cord results in a dynamic equilibrium that develops between the production of edema fluid produced by the SCI and the absorption of this edema fluid by the omentum. This absorption of edema fluid allows for the associated absorption of fibrinogen, decreasing the potential for its activation to fibrin, which routinely occurs and is readily observed during operation in patients with a chronic spinal cord injury.

Dr Heimburger, in his article, and Dr Nashold, in his commentary, both suggest that new avenues should be explored to see if improvement can be generated in lessening the severe consequences of spinal cord injuries. Present day treatment of spinal cord injuries has not substantially improved over the last half century. Perhaps the two surgical suggestions made in this letter to the editor—opening the dura mater widely and placing the omentum on the spinal cord for traumatic edema absorption—might prove to be a hopeful step toward improving the neurologic results after the surgical treatment of acute spinal cord injuries.

REFERENCES


Reply

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Harry S Goldsmith’s letter about treatment of spinal cord injury (SCI) is a significant step toward the hope for return of function in muscles paralyzed by spinal cord injury. His insightful method of early gradual decompression of the swollen spinal cord resembles the method described in LW Freeman’s 1953 article on pial incision to release pressure on axons not torn in the original trauma, but permanently damaged by intense pressure from edema. In his studies, including those on rats and dogs, he found that “wide pial incision after trauma is not harmful,” and “rapid debridement offers the best possibility for preservation of function.” Freeman found “quite clearly that better functional results can be expected when primary debridement of necrotic cord tissue and blood is conducted.” He didn’t add that this surgery must be conducted by surgeons trained and skilled in dealing with the spinal cord, but did require residents to have performed five rat or dog laminectomies, with dural incision, without any loss of function, before they could participate in ongoing experimental operations. Although the results of this spinal cord concussion study were not 100% good or bad, they did give definite indication that pial incision of the swollen spinal cord provided a better chance for recovery of function in paralyzed muscles.

One young man with a cervical cord injury had such severe spasticity of both legs and both arms, which is unusual, that we feared for his life. He was unable to eat enough or sleep for several months after his injury. With consent from his parents and his own insistence, cervical laminectomy and midline dural incision were made over a frightfully swollen cervical spinal cord. With great trepidation, a midline pial incision was made to allow black hematoma and white necrotic cord to extrude. After gentle irrigation, the dura could be closed easily, and muscle and skin reapproximated. Spasticity was no longer present when he awoke from anesthesia. He was extremely active in physical therapy, and became able to take a few steps holding parallel bars, and in a few weeks to reasonable walking, with a stiff-legged gait.

Dr Goldsmith’s letter provides me the chance to mention additional procedures that give hope for those paralyzed by SCI. Dr Goldsmith suggests placing “an intact omentum pedicle flap directly on the site of the SCI” to help absorb edema fluid. This reminds me of Freeman’s use of urea to decrease swelling in the laboratory and also in humans right after injury. Steroids soon replaced urea and provided better control of spinal cord edema.

Orthopaedic surgeon, Dr Shaocheng Zhang, connected peripheral nerves above a spinal cord injury to those below the injury in the Changhai Hospital, Shanghai, China, with encouraging return of function in paralyzed muscle, bowel, and bladder function in hundreds of spinal cord–injured patients. Zhang’s experience tends to confirm Freeman’s contention that axons have a