

TREATMENT OF SPINAL CORD INJURIES IN THE THORACOLUMBAR REGION

Carl-Axel Carlsson and Luigi Pellettieri

From the Department of Neurosurgery, University of Göteborg, Sahlgren's Hospital, Göteborg, Sweden

ABSTRACT. Forty patients with spinal cord injuries in the thoracolumbar region were studied. Clinical and experimental data support the conservative approach. In some patients acute stabilisation is needed and in a few patients operative decompression may be considered. Promising experimental non-invasive techniques to improve recovery are presented, but there are no convincing clinical results so far. The authors believe that regeneration will be the key area for future research.

Key words: thoracolumbar spinal injuries, outcome of treatment, treatment alternatives.

If you line up ten neurosurgeons and orthopedic surgeons you will probably get five to ten different ways to treat a patient with a spinal cord injury in the thoracolumbar region. Probably no one is right because there have not been prospective randomized studies good enough to say what is right. Moreover, the details of the disturbances of nervous function following spinal cord injury are only roughly known to us. The discussion concerning the best treatment therefore has to be kept on a pragmatic level. The main questions are whether to operate or not and, if operation, how and when to operate.

CLINICAL OBSERVATIONS

Between 1961 and 1981, 46 patients were treated at our hospital for spinal cord injuries in the thoracolumbar region involving the Th 12 and/or the L 1 vertebra. Pathological fractures due to vertebral metastases and fractures without neurological deficit were excluded. The patients were treated conservatively or by laminectomy with or without fusion or with Harrington rods with fusion. The neurological status was assessed according to a modified Fraenkel scale (Fig. 1). The duration of follow-up ranged from two to 22 years. Six patients had died. The remaining 40 patients are presented in Table I classified according to treatment and neurological status at admis-

sion. There was no significant difference between the groups.

The results are summarized in Table II. The average improvement signifies how the average patient had moved on the modified Fraenkel scale. There is no difference between the treatment groups. This is in agreement with other studies, e.g. Osebold et al. (18) who found that no treatment method so far available was superior to the others with respect to neurological function.

Table III shows the duration of hospitalisation. There is no difference between conservatively treated and Harrington rod patients, while those subjected to laminectomy stay significantly longer in hospital. Patients with Harrington rods can be mobilised very early but that seems to be the only advantage of Harrington rod treatment over conservative treatment.

So, we have the choice between conservative treatment which means that the patient has to stay in bed for 6 to 12 weeks depending on what policy is chosen and Harrington rod treatment which means that he or she can be mobilised during the first week but has to have two operations. For some the choice may be easy, for others not. Early mobilisation has many advantages but two operations involve a certain risk. We agree with Bedbrook (2) when he states that the treatment procedures must be carried out in a comprehensive unit and then only after adequate assessment and consideration, not only by the surgeon, but principally by the overall physician whether he be surgeon, physician or paraplegist.

TRAUMATIC SPINAL CORD DISEASE

Now, let us analyse the problem from another point of view. A spinal cord injury is in many respects similar to a brain injury. The late Sir Geoffrey Jefferson spoke of brain concussion as a sometimes progres-

1. Complete paraparesis (complete absence of motor function)
2. Severe paraparesis (insignificant motor function in some muscle groups)
3. Moderate paraparesis (motor function in several muscle groups)
4. Slight paraparesis (motor function in most of the muscle groups)

Fig. 1. Classification of patients.

sive disease and coined the expression brain injury disease (17). In the same way, the autodestructive process of the spinal cord starts at the moment of injury—if the impact is of sufficient magnitude—and proceeds up to a certain point. As for head injuries this process is governed by the force and mechanism of the injury. So far, there is no miracle drug or operation that can stop this process. Simultaneous with and intermingled with this destructive process reparative process starts. Macrophages migrate into the traumatised area and begin to remove dead tissue; intact but paralysed fibers and tracks start to conduct impulses; in disarranged fibers rebuilding starts and in interrupted pathways regeneration of axons occurs. Research today is concerned with the search for therapies which arrest the autodestructive process and enhance the reparative processes. Although some beneficial effects have been reported in animal experiments, we think it is right to say that there are no convincing clinical studies so far. Thus, in the acute phase of a spinal cord injury current knowledge supports the conservative approach. There does not seem to be any indication for early operation in the average patient.

INDICATIONS FOR SURGERY

But there are exceptions. Firstly, when there is an obvious instability, i.e. when there is marked anterior-posterior or lateral displacement between the involved vertebra, we think it is necessary to stabilize immediately to prevent secondary damage. This

Table I. Neurological status at admission

	Conservative n=16	Laminectomy n=14	Harrington n=10
Complete	4	3	2
Severe	5	5	3
Moderate	2	2	3
Slight	5	4	2

Table II. Neurological improvement

	Number of patients	Number of patients improved	Average improvement (number of step)
Conservative	16	13	1.3
Laminectomy	14	11	1.2
Harrington	10	9	1.3

applies only to a limited number of patients. The majority are stable enough to be treated conservatively. Bedbrook (1) reports in a review of thoracolumbar injuries that some 5% might be considered for technical fixation. The second exception concerns those patients in whom there is compression of the spinal cord and nerve roots by bony elements or disc material. In these cases it seems logical to remove compression to create better conditions for the reparative processes. Fig. 2a and b show cross-sections at the level of the Th12-L1 disc and the L1-L2 disc. The cross-section area has to be reduced by at least 50% before the cord and roots are significantly compressed at L1-L2 but only with about 20% at the Th12-L1 disc level. Since the position of the conus differs between individuals one has to make the decision from patient to patient. To visualize the conus a small amount of contrast must be introduced into the subarachnoid space.

The dislocation of fragments into the spinal cord is sufficiently reduced after application of Harrington rods in 90% of the patients (20). In the remaining 10%, decompression can be performed. A lateral approach to the spinal canal is then recommended. It is our experience that the degree of recovery does not correlate to the time of compression. Therefore, the removal of the compression can be postponed until the cord is in a more stable cytochemical condition and less vulnerable not only for additional mechanical impact but also for a drop in blood pressure (7). How long one has to wait is difficult to predict, but a week seems a reasonable time for the cytochemical

Table III. Duration of hospitalisation

	Weeks	SD
Conservative	29	14
Laminectomy	39	10
Harrington	23	18

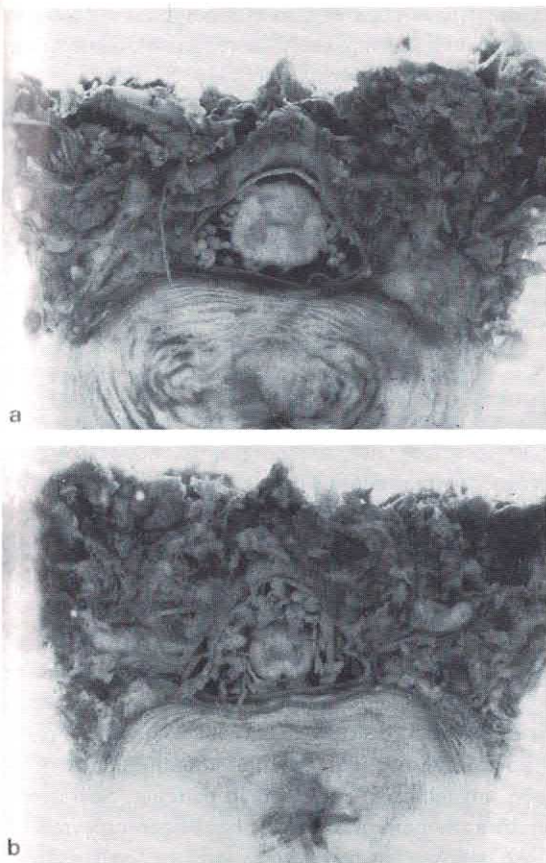


Fig. 2. For explanation see text.

processes to stabilize, the edema to vanish and the autoregulation and changes in the arteriovenous blood flow pattern to normalise.

CURRENT TREATMENT METHODS

We differ between acute methods, which aim to prevent further damage as stabilization or to arrest or reduce the effects of the destructive processes which start at the moment of injury and elective methods, which aim to enhance reparative processes or to maintain what the patients have by using various techniques of rehab engineering.

Surgical treatment (Fig. 3). We have already discus-

ACUTE	ELECTIVE
Stabilisation	Decompression
Hypothermia	Neural bypasses
Omental transposition	Functional electrical stimulation

Fig. 3. Surgical.

ACUTE	ELECTIVE
Hyperbaric oxygen	Rehab. engineering
Pulsed electromagnetic fields	

Fig. 4. Non-invasive approaches.

sed stabilization as an acute treatment and decompression as an elective treatment. The basis for using cooling of the injured area is its ability to lower the metabolic and oxygen demand of nerve cells and possibly also that it could reduce the swelling of the injured cord (24). Omental transposition is interesting not only for spinal cord injuries but also for cerebral injuries and cerebral ischemic lesions (22). The omentum has powerful capacity to reduce edema and to revascularize the injured area.

On the elective side we have neuronal bypasses which means that we try to shunt activity from roots or nerves which have a central connection to roots belonging to the isolated spinal segment (6). Functional electrical stimulation refers to implantable systems, e.g. conus stimulation (5) or stimulation of sacral roots to achieve bladder emptying (3) and epidural spinal stimulation to normalize a detrusor-sphincter dysnergia to reduce spasticity and to improve coordination of movements in partial lesions (8). It also includes high technology computerized systems implanted on peripheral nerves to make paralysed individuals walk as reported by a group in Vienna (23).

Non-invasive approaches (Fig. 4). Experimental work on sheep has shown that hyperbaric oxygen can modify the degree and extent of the pathology in the spinal cord following an injury (25). It has been shown that contusion of the spinal cord causes massive Ca entry into cells, which is known to have deleterious effects on cellular function and survival. Electromagnetic fields applied to spinal cord shortly after contusion reduces Ca accumulation and is associated with a better recovery of motor and sensory functions (16, 27).

On the elective side rehab engineering has exploded as a field (12). Paralysed patients have nervous tissue that is viable but not functioning. By

ACUTE	ELECTIVE
Antiadrenergic compounds	Neurotrophic (growth) factors
Naloxone	
Dimethyl sulfoxide	
Anticoagulants	
Corticosteroids	
Nimodipine	

Fig. 5. Pharmacological

Spinal cord: feeble sprouting; functionally not useful
 Dorsal roots: fibers regenerate in central direction but do not enter spinal cord; functionally not useful
 Ventral roots: regenerate similar to peripheral nerves; functionally useful

Fig. 6. Regenerative capacity.

application of high technology you can convert this tissue to functional tissue. It is a matter of maximizing of what the patients have. Through various techniques with surface electrodes it is possible to improve a patient's functional capacity by changing the muscle bulk and strength, to help the patients to find their muscles by biofeedback to strengthen osteoporotic bones with vibration and to reduce spasticity with low intensity laser.

Pharmacological treatment (Fig. 5). Osterholm et al. (19) proposed that toxic amounts of noradrenaline (NA) is released at the site of injury. This NA accumulated is considered to be responsible for the necrosis and haemorrhage that has been observed within 1–2 hours after injury. Subsequently a number of compounds that could decrease the level of central NA have been used in animal experiments. Alpha-methyl tyrosine was reported to be very effective in controlling the post-traumatic progression pathological picture with necrosis and haemorrhage. Other investigators however found no increase of NA in the injured area (21). Sympathectomized cats did not exhibit the posttraumatic ischemia typically produced by contusion injury (28). The critical factor appears to be the presence of the paravertebral sympathetic ganglia and not the adrenal glands.

Naloxone (11, 13), dimethyl sulfoxide (9), anti-coagulants (15) and cortico-steroids (26) have been found in animal experiments to improve spinal cord blood flow and neurological recovery at the experi-

mental spinal cord injury. Nimodipine, a calcium channel blocker markedly increased blood flow in the spinal cord in normal rats (14).

On the elective side neurotrophic factors are mentioned as a common title for all drugs that could possibly enhance regeneration. The reader is referred to the excellent review by de la Torre (10).

At present there is no breakthrough in regeneration and the situation is described in Fig. 6.

CONCLUSION

The lack of knowledge in this area is tremendous and it is therefore not meaningful to advocate any method or treatment as better than the others. With this reservation in mind we would summarize our view of the treatment as follows (Fig. 7): The first decision has to be made at admission because markedly unstable fractures must be stabilized. The majority of patients can be treated conservatively. After a week it is time to make the second decision. If there is significant compression of the spinal cord and roots, it should be removed.

The promising experimental therapies have not been satisfactorily evaluated in patients. So far no significantly beneficial effects have been reported.

We believe that regeneration will be the key area for future research since in the long run we probably have better possibility here than we have to arrest the autodestructive processes which start at the moment of impact.

REFERENCES

1. Bedbrook, G. M.: Treatment of thoracolumbar dislocation and fractures with paraplegia. *Clin Orthop* 112: 27–43, 1975.
2. Bedbrook, G. M.: Spinal injuries with tetraplegia and paraplegia. *J Bone J Surg 61-B*: 267–284, 1979.
3. Brindley, G. S., Polkey, C.-E. & Rushton, D. N.: Sacral anterior root stimulators for bladder control in paraplegia. *Paraplegia* 20: 365–381, 1982.
4. Björklund, A. & Stenevi, U.: Nerve growth factor: Stimulation of regenerative growth of central noradrenergic neurons. *Science* 175: 1251–1253, 1972.
5. Carlsson, C.-A. & Fall, M.: Electrical stimulation of the conus medullaris for bladder emptying in a paraplegic. *Paraplegia* 22: 87–91, 1984.
6. Carlsson, C. A. & Sundin, T.: Reconstruction of afferent and efferent nervous pathways to the urinary bladder in two paraplegic patients. *Spine* 5: 37–41, 1980.
7. Collins W. F.: A review and update of experiments and clinical studies of spinal cord injury. *Paraplegia* 21: 204–219, 1983.

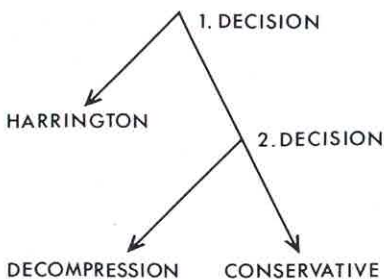


Fig. 7. For explanation see text.

8. Cook, A. W.: Electrical stimulation in multiple sclerosis. *Hospital Practice II*: 51-58, 1976.
9. de la Torre, J. C., Johnson, C. M., Goode, D. J. & Mullan, S.: Pharmacological treatment and evaluation of permanent experimental spinal cord trauma. *Neurology* 25: 508-514, 1975.
10. de la Torre, J. C.: Spinal cord injury. Review of basic and applied research. *Spine* 6: 315-335, 1981.
11. Faden, A. I., Jacobs, T. P. & Holaday, J. W.: Opiate antagonist improves neurologic recovery after spinal cord injury. *Science* 211: 493-494, 1981.
12. First Vienna International Workshop on Functional Electrostimulation Basics, Technology and Application. Vienna (Austria), October 19th-22nd, 1983.
13. Flamm, E. S., Young, W., Collins, W. F., Piepmeier, J., Clifton, G. L. & Fischer, B.: A phase I trial of naloxone treatment in acute spinal cord injury. *J Neurosurg* 63: 390-397, 1985.
14. Guha, A. B., Tator, C. H. & Piper, I.: Increase in rat spinal cord blood flow with the calcium channel blocker, nimodipine. *J Neurosurg* 63: 250-259, 1985.
15. Hallenbeck, J. M., Jacobs, T. P. & Faden, A. I.: Combined PGI, indomethacin, and heparin improves neurological recovery after spinal trauma in cats. *J Neurosurg* 58: 749-754, 1983.
16. Ito, H. & Bassett, C. A.: Effect of weak, pulsing electromagnetic fields on neural regeneration in the rat. *Clin Orthop* 181: 283-290, 1983.
17. Jeffersson, G.: Selected Papers, p. 473. Pitman Medical Publishing Co., London, 1960.
18. Osebold, W. R., Weinstein, S. L. & Sprague, B. L.: Thoracolumbar spine fractures. Results of treatment. *Spine* 6: 13-34, 1981.
19. Osterholm, J. L. & Mathews, G. J.: Altered norepinephrine metabolism following experimental spinal cord injury. Part 1. Relationships to hemorrhagic necrosis and post-wound neurological deficits. Part 2. Protection against traumatic spinal cord hemorrhagic necrosis by norepinephrine by synthesis blockage with alpha-methyl tyrosine. *J Neurosurg* 36: 386-401, 1972.
20. Nordwall, A.: personal communication.
21. Rawe, S. E., Roth R. H. & Collins, W. F.: Norepinephrine levels in experimental spinal cord trauma. Part 1. Biochemical study of hemorrhagic necrosis. *J Neurosurg* 46: 342-349, 1977.
22. Steward, E. & Duckett, S.: Early application of pedicled omentum to the acutely traumatised spinal cord. *Paraplegia* 23: 100-112, 1985.
23. Stoehr, H., Bochdanský, T., Frey, M., Holle, J., Kern, H., Schwanda, G. & Thoma, H.: In 1st Vienna international workshop on functional electrostimulation. Vienna (Austria), 1983.
24. White, R. J.: Current status of spinal cord cooling. *Clin Neurosurg* 20: 400-408, 1973.
25. Yea, J. D., McKencie, B., Hardwood, B. & Kidman, A.: Treatment of paraplegic sheep with hyperbaric oxygenation. *Med J Aust* 1: 538, 1976.
26. Young, W. & Flamm, E. S.: Effect of high-dose corticosteroid therapy on blood flow, evoked potentials, and extracellular calcium in experimental spinal injury. *J Neurosurg* 57: 667-673, 1982.
27. Young, W.: A critical overview of spinal injury research presented at the First International Symposium on CNS Trauma. *Cent Nerv Syst Trauma Fall*; 1: 75-79, 1984.
28. Young W., DeCresito V. & Tomasula J. J.: Effect of sympathectomy on spinal blood flow autoregulation and posttraumatic ischemia. *J Neurosurg* 56: 706-710, 1982.

Address for offprints:

Docent Carl-Axel Carlsson
 Department of Neurosurgery
 Sahlgrenska Hospital
 41345 Göteborg
 Sweden