NEUROLOGICAL RECOVERY AFTER CONSERVATIVE TREATMENT OF CERVICAL CORD INJURIES

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We reviewed a series of 53 patients with closed traumatic complete injuries of the cervical spinal cord. They were admitted within two days to a spinal injuries centre, treated conservatively by six weeks of bedrest and skull traction, then mobilised in a neck support for six weeks.

Eight patients had temporary neurological deterioration, four spontaneously and four after cervical manipulation; seven of these recovered to the initial neurological level without surgery. Of 40 patients followed for more than 12 months, 19 recovered useful motor power in local muscles which were initially paralysed (zonal recovery); one patient showed distal motor recovery. Zonal recovery did not correlate with the mechanism of skeletal injury or with the degree of residual canal stenosis. Sensory sparing and an initial neurological level higher than the level of skeletal injury were both good prognostic signs for zonal recovery.

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It has been claimed that patients treated for complete injuries of the cervical spinal cord by early internal fixation have a lower incidence of ascending myelopathy and better nerve-root recovery than after non-surgical treatment (Yablon et al 1989, 1991). Anderson and Bohlman (1992) reported zonal recovery after anterior decompression and stabilisation for fracture-dislocations, but to date no comparison has been made with the natural history of recovery in patients treated conservatively in a spinal injuries centre.

Our aim was to document neurological deterioration and recovery after complete traumatic injury of the cervical cord treated conservatively and to compare the results with those available in the literature for patients treated surgically.

PATIENTS AND METHODS

We describe 66 adult patients who were admitted to the Midlands Centre for Spinal Injuries, within two days of such an injury, between January 1983 and December 1992. None had sacral sparing or any sensory or motor function distal to the third neurological segments below the last normal level (Waters, Adkins and Yakura 1991), and all were classified as Frankel A on admission (Frankel et al 1969). None had multiple spinal injuries. There were 58 men and 8 women and their ages at the time of injury ranged from 15 to 77 years (mean 33.7). They were followed up for 2 to 104 months (mean 33.3). The causes of injury are given in Table I. Twenty-six patients were admitted on the day of the injury, 32 the day after, and eight between 24 and 48 hours after injury.

Thirteen patients were excluded from the neurological evaluation. Seven of these had died, six of whom had post-mortem examinations. Two deaths were due to gastrointestinal lesions, one to a pulmonary embolism and one to a brainstem lesion. In three cases the cause of death was not determined. The other six patients underwent surgical treatment. Two had open reduction with posterior fusion. Late posterior fusion in situ was carried out on four patients who had instability which could not be controlled by conservative treatment. No patient underwent surgery because of neurological deterioration.

Table I. Causes of injuries to the spinal cord in 66 patients

Cause	Number	*Percentage*
Road-traffic accident	34	*51.5*
Fall from a height	11	*16.7*
Rugby football	7	*10.6*
Diving	6	*9.1*
Fall on level ground	5	*7.6*
Horse-riding	2	*3.0*
Other	1	*1.5*
Total	66	

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The neurological outcome of the remaining 53 patients was evaluated. Our protocol of conservative treatment consisted of bedrest with skull traction for six weeks, followed by mobilisation with a neck support for six weeks. Closed reduction was attempted on 17 patients within 48 hours of the injury, in five instances under general anaesthesia. Almost all patients had anticoagulant therapy and H2-receptor antagonist medication. Three patients developed thromboembolic disease and one had a paralytic ileus. Before mobilisation the stability of the cervical spine was confirmed radiographically by dynamic views.

The neurological status was evaluated using Frankel's classification (Frankel et al 1969) and the motor score system of the American Spinal Injury Association (Donovan et al 1990) in which the power of five key muscles in each limb is recorded by manual testing on a scale of 0 to 5. Worsening of the neurological level or decrease in motor strength by more than one grade was regarded as neurological deterioration. Zonal recovery was recorded when motor power increased from grade zero on admission to grade two or more at follow-up. The mere increase in strength of a distal muscle which had been functioning on admission was not considered as zonal recovery. The skeletal level of injury was determined from the initial cervical radiographs, and the mechanism of injury was classified according to Allen et al (1982) (Table II). The sagittal canal diameter at the level of residual stenosis and the percentage of the stenosis (Donovan, Cifu and Schotte 1992) were measured on the lateral radiographs.

Table II. The types of cervical injury, according to the mechanistic classification of Allen et al (1982), of the 53 patients reviewed

Type of injury	Number	*Percentage*
Compressive flexion	24	*45.3*
Distractive flexion	18	*34.0*
Vertical compression	4	*7.6*
Compressive extension	1	*1.9*
No apparent injury or type unknown	6	*11.3*
Total	53	

Table III. The neurological improvement and the types of skeletal injury in the 40 patients who were observed for more than 12 months

	Number of levels recovered				
Type of injury	**0**	**1**	**2**	**3**	**To legs**
Compressive flexion	12	7	1	1	0
Distractive flexion	5	5	2	0	1
Vertical compression	3	0	0	0	0
Compressive extension	1	0	0	0	0
No apparent injury or unknown	0	1	1	0	0
Total	21	13	4	1	1

RESULTS

Eight of the 53 patients had a deterioration of their neurological level; by two segments in three patients and by one segment in five. Four occurred spontaneously and four followed attempts at closed reduction. The mean interval between deterioration and injury was 2.4 days (1 to 6). Seven of these patients later improved to the initial neurological level. The eighth patient, who had ankylosing spondylitis, regained one of the two lost segments but was lost to follow-up ten months after injury. All four patients who deteriorated spontaneously started to recover within seven weeks and reached their initial level by three months from injury. The two patients with ankylosing spondylitis who deteriorated after closed reduction started to recover within three weeks and one of them reached his initial neurological level about one year later. The other two patients who deteriorated after attempted closed reduction started to recover within a week, and reached the initial level two months later. Partial loss of motor power of more than one grade occurred in three patients within seven days of injury. All three recovered within four weeks.

Forty of the 53 patients were observed for more than 12 months (mean 43.2). Of these, 35 (87.5%) remained at Frankel A level, four (10.0%) improved to Frankel B and one (2.5%) improved to Frankel C. The mean motor score in the ten key muscles of the upper extremities improved by 9.2 ± 5.5, from 11.6 on admission to 20.8 at follow-up.

The neurological level improved in 19 patients (47.5%) (Table III). One patient recovered to Frankel C scoring 16 motor points in the lower limbs. Most of the muscles which recovered had power grade 3 or more. Using the two-tailed Student's *t*-test we compared the 19 patients who showed zonal recovery with the 21 who did not. There was no significant difference in either the mean age (29.5 years and 32.5 years), mean residual canal diameter (11.8 mm and 12.4 mm) or mean percentage of canal stenosis (25.1% and 24.0%). Nor was there a significant difference between the mechanism of injury and the incidence of zonal recovery (Yates-corrected chi-squared analysis). Zonal recovery occurred in 12 of the 19 patients (63.2%) who had sensory sparing in a dermatome in which motor power was initially zero, and four of these recovered beyond the segment in which sensation was initially preserved. Only seven of the 21 patients (33.3%) who had no sensory sparing in a dermatome in which motor power was zero showed zonal recovery, and only one recovered more than one segment.

A second factor which influenced zonal recovery was the discrepancy between the neurological and the skeletal level of injury. The patients who had an initial

neurological deficit one segment below the skeletal injury (Stauffer 1975) did not recover further; those in whom the neurological level coincided with or was higher than the level of bone injury showed zonal recovery (Table IV).

Table IV. Zonal recovery related to discrepancy between vertebral and initial neurological level of injury

Discrepancy	Recovery	No recovery
Neurological higher than skeletal level	13	4
Neurological same as skeletal level	6	11
Neurological lower than skeletal level	0	6

DISCUSSION

Although the treatment for injury to the spinal cord is controversial (Stauffer 1984; Tator et al 1987; Donovan et al 1992; El Masri and Jaffray 1992), surgery has been increasingly advocated to facilitate zonal root recovery (Stauffer 1984; Benzel and Larson 1986; Yablon et al 1991; Anderson and Bohlman 1992). Advocates of conservative treatment, however, have claimed results at least as good with a low incidence of neurological deterioration and late instability (Bedbrook 1979). Decompressive laminectomy has given uniformly poor results (Holdsworth 1970; Morgan, Wharton and Austin 1971) but the complications that used to occur with anterior interbody fusion for fracture-dislocation (Stauffer and Kelly 1977) have been reduced due to better anaesthetic and surgical techniques.

Holdsworth (1970) stated that if the paraplegia below a cord lesion remained complete for 24 hours, the cord was irreparably damaged and recovery could never occur. This was not borne out by the result in our one patient who had complete somatosensory loss for three days before recovery to Frankel C.

Deterioration of the neurological level can occur from any method of treatment. Frankel (1969) stated that "The commonest type of neurological deterioration is a rise in the level of the lesion usually by one segment, sometimes by two segments. This usually occurs in the first four days and the deterioration is almost always temporary, the final neurological lesion being the same or lower than the lesion found immediately after the injury.". Yablon et al (1989) reported ascending myelopathy in 14 of 134 patients with cervical cord injuries. They found zonal neurological deterioration in 10 of the 54 patients (18.5%) treated nonoperatively and in 4 of the 80 patients (5.0%) who were treated surgically. In our study, four conservatively-treated patients had spontaneous temporary increase of the neurological level and another four deteriorated after manipulation of the cervical spine, two of them having ankylosing spondylitis. Comparison between our series and that of Yablon et al (1989) shows that the incidence of deterioration in these two series is similar. It is not clear, however, how many of their patients deteriorated spontaneously and how many after closed reduction, nor was recovery documented in their series.

Harris et al (1980) defined zonal recovery as the return of motor power from an initial grade of zero to a grade of two or more. Patients who had even a flicker of movement on admission in muscles which improved even to grade five were not deemed to have shown zonal recovery. Using these strict criteria, 47.5% of our patients treated conservatively had zonal recovery. Unilateral recovery from power zero was not included if the same muscles on the opposite side had some power initially. If these cases are included, the percentage of recovery in our series is 57.5%. Using the criteria of Yablon et al (1991) the recovery rate of our patients was 57.5%, not significantly different from the 50% recovery rate that they reported for patients treated by surgery.

Anderson and Bohlman (1992) reported good results with late anterior decompression and stabilisation for patients with cervical fracture-dislocations and complete motor loss. Their indications for operation were "myelographic demonstration of compression of the anterior aspect of the cervical cord and motor-roots by fragments of bone or discs, and the presence of a neurological plateau". They stated that the results were poor if decompression was performed more than 18 months after injury or in patients of 53 years of age or more. Of their 32 patients with complete motor and sensory loss, all four who had excellent results and six of the 12 who had good results were operated on within four months of injury. Among the 19 patients in our series who showed zonal recovery, five spontaneously recovered one or more segments later than four months after injury. Ditunno et al (1992) found that simple increase in muscle strength usually ceased between three and six months but that recovery of initially completely paralysed muscles could occur as long as one year or more after injury.

Zonal sensory sparing and the relationship between the neurological and bone levels of injury proved to be good predictors of zonal recovery or the lack of it. Uniform methods of documentation including these two criteria are required to determine if zonal recovery is improved by surgical stabilisation and/or decompression. Increase of the neurological level occurs spontaneously and, with adequate conservative treatment, we observed zonal recovery in 47.5% of our patients. No patient suffered permanent deterioration from the neurological level present on admission.

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