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Neurological Deficit in Injuries of the Thoracic and Lumbar Spine

A Consecutive Series of 70 Patients

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Summary

Seventy consecutive patients with injuries of the thoracic and lumbar spine accompanied by neurological deficit were prospectively studied and followed-up.

In 40 of these patients with a burst fracture, the degree of involvement of the cross-sectional area of the spinal canal, as revealed on first CT after admission, was not correlated with the type and degree of initial neurological deficit.

In patients with injuries of the lumbar spine, neurological deficit may be mild, although the sagittal diameter of the spinal canal may be reduced by as much as 90%.

We cannot establish a difference in neurological recovery between those cases who were managed conservatively and those in whom a surgical decompression and stabilization procedure was performed.

Surgical stabilizing procedures, however, result in immediate stabilization of the spine, they diminish pain, facilitate nursing care and allow more rapid mobilization and earlier active rehabilitation.

If major extraspinal injuries form a relative contra-indication to surgical decompression of the cord and stabilization of the spine injury, the patient can quite well be treated conservatively without endangering neurological recovery.

Keywords: Thoraco lumbar spine injury; spinal cord injury; neurological recovery.

Introduction

Injuries of the thoracic and lumbar spine can be classified according to the site of damage: i.e. whether this is present in the anterior, middle or posterior column[8]. A distinction is thus made between compression fractures, burst fractures, seat-belt injuries and fracture-dislocations (Fig. 1). Many patients with injuries to two or three parts of the spinal column show neurological deficit, due to damage to the cord, conus or cauda. This is usually the result of involvement of the bony spinal canal due to narrowing in the transverse plane and also to misalignment[4, 10, 17, 18, 19, 22, 24].

Type of Fracture	Column		
	Anterior	*Middle*	*Posterior*
Compression fracture	compression	none	none or distraction (severe)
Burst fracture	compression	compression	none
Seat-belt injury	none	distraction compression	distraction
Fracture dislocation	compression rotation shear	distraction rotation shear	distraction rotation shear

Fig. 1. Basic modes of failure of the three columns in the four major types of spinal injury (Denis, 1983)[8]

In the present study, carried out in 70 consecutive cases of injuries of the thoracic and lumbar spine with neurological deficit, we tried to assess:

a: the relationship, in burst fractures, between the type and degree of initial neurological deficit and the degree of involvement of the spinal canal revealed on the first CT after admission.

b: the post-traumatic recovery of neurological deficit in relation to type of management (surgical or conservative).

c: the relationship, at follow-up, between residual neurological deficit and remaining amount of spinal canal narrowing as shown on CT at follow-up.

Material and Methods

Patients

Between June 1981 and December 1988, 187 patients with injuries of the thoracic and lumbar spine were admitted to the departments of Neurosurgery and Orthopaedics of the University Hospital Rotterdam. The injuries of the spine were classified according to type (Fig. 1) and level of injury (thoracic: T1-T10, thoracolumbar: T11-L1 and lumbar: L2-L5). Burst fractures were encountered in 120 patients, fracture-dislocations in 28 patients. There was one case of seat-belt injury and one (rare case of) extension-injury. The remaining 37 patients had a compression fracture of a vertebral body without neurological deficit; they were nevertheless admitted because they had other major injuries requiring clinical management.

The patients who were treated as out-patients were not included in this study.

Seventy patients in this series had neurological deficit: they form the basis of this study. The distribution of injury over the spine, the classification into various types of injury and the type of neurological deficit assessed on admission using the Frankel classification (Fig. 2), are presented in Table 1. Age at time of injury varied from 17 to 65 years: average 32 years. Fifty-one patients were male, 19 female.

Complete	A	no motor or sensory function below level of lesion
Sensory only	B	no motor function, but some sensation preserved below level of lesion
Motor useless	C	some motor function without practical application
Motor useful	D	useful motor function below level of lesion
Recovery	E	normal motor and sensory function, may have reflex abnormalities

Fig. 2. Classification of neurological deficit according to Frankel *et al.*[13]

Radiology; CT

All 70 patients underwent plain X-ray examination and sometimes poly-tomography. On admission 63 patients were subjected to computer-tomography (bone-level window and width) centered over the damaged part of the spine. A slice thickness of 3 or 6 mms was used. Seven patients did not undergo computer-tomography on admission, because they were too seriously poly-traumatized and had to be placed on the respirator.

Spinal stenosis

In each patient with a burst fracture, the involvement of the spinal canal was assessed by measuring on CT the minimal sagittal diameter of the spinal canal at the level of injury. The percentage of spinal canal compromise was the difference between this sagittal diameter and the estimated normal measurement for that level of the spine, divided by the estimated normal sagittal diameter and multiplied by 100. The estimated normal diameter for that level of the spine was calculated as the average of the corresponding measurements of the adjacent uninjured vertebrae above and below the level of injury[16].

Follow-up

Two patients died from unrelated causes, but their neurological condition before death was known. Two patients could not be traced and one patient refused contact, but also in these three patients, the neurological condition was known. The remaining 65 patients were interviewed and underwent physical and neurological examination 2 to 6 years after the accident (mean follow-up 4.3 years).

The actual neurological state was classified according to the Frankel scale, making a distinction in category D between D_1 (walk-

Table 1. *Relationship Between Type and Level of Injury and the Severity of Neurological Deficit, According to the Frankel Classification*

		Frankel classification				
Type and level of injury	n	A	B	C	D	E
Burst fracture	40					
– thoracic	7	1	1	3	2	–
– thoracolumbar	23	2	3	7	10	1*
– lumbar	10	1	1	1	7	–
Fracture-dislocation	28					
– thoracic	8	7	1	–	–	–
– thoracolumbar	16	10	3	1	2	–
– lumbar	4	–	2	–	2	–
Seat-belt injury (thoracolumbar)	1	–	–	–	1	–
Extension injury (thoracolumbar)	1	1	–	–	–	–
Total	70	22	11	12	24	1

* No motor deficit; sensory deficit in area S3-S5 only with urinary retention.

ing with supports) and D_2 (walking without supports). Fifty-eight patients consented to a check-up, two-directional radiographic examination and computer-tomography. The other 12 patients had either died[2], could not be traced[2] or refused a radiological check-up[8].

Results

Forty patients had a burst fracture, 28 a fracture-dislocation, one a seat-belt injury and one an extension injury. Twenty-two patients had a complete cord lesion, 23 a severe incomplete cord lesion (Frankel grade B and C) and 25 a milder incomplete cord or cauda lesion (Frankel grade D and E) (Table 1).

Management

Of the 70 patients with neurological deficit, 36 were treated surgically and 34 conservatively.

Of the 22 patients with a *complete cord* lesion, the majority, 17 patients, were treated conservatively. The other 5 were subjected to surgical stabilization, two receiving additional posterolateral decompression of the dural sac.

Of the 48 patients with an *incomplete cord* lesion, most (31) were subjected to surgery. The other 17 were not operated upon; 10 of these patients had more than one major associated injury which formed a relative contra-indication to an early surgical procedure. At a later stage when surgery might have been performed, the lesion of the spine had either already stabilized, or the patient preferred conservative management. Seven patients were treated conservatively for other reasons.

Surgical Procedure

In the 3 cases with facet-interlocking open reduction was performed, followed by wiring and posterior fusion. In most cases of fracture-dislocations or burst fractures and incomplete neurological deficit, the posterolateral approach was used to decompress the dural sac by removal of loose fragments found in the spinal canal and tamping of the displaced vertebral body fragment(s) into the vertebral body[10, 11, 14, 24]. Subsequent instrumental reduction and stabilization was achieved either by Harrington rods[10] or – in later years – with a fixateur interne, according to Dick[9].

Conservative Management

Conservative treatment consisted of horizontal bedrest with – if possible – postural reduction for a period of 6 weeks (upper thoracic injuries) to 3 months (thoracolumbar injuries). In the case of incomplete lesions, the spine was stabilized using plaster shells. If other major injuries allowed change of position, the Stryker frame was used to alternate the patient every 3 hours between the prone and the supine positions.

Neurological Deficit and Degree of Narrowing of the Spinal Canal

Burst fracture. The relationship between the type of neurological deficit and the reduction of the sagittal diameter of the spinal canal in burst fractures is presented in Table 2. Of the 7 patients with a burst fracture of the *thoracic spine*, only one with a spinal stenosis of 70% had a complete cord lesion. The remaining 6 patients with an incomplete cord lesion had a stenosis of $<10\%$ (3 patients) and 33% (2 patients). One patient with complete motor paralysis was multitraumatized and had to be ventilated for more than one week. For this reason computer-tomography was not performed and hence the degree of stenosis could not be established.

Of the 23 patients with a burst fracture of the *thoracolumbar spine*, two with a complete cord lesion had a spinal stenosis of 44 and 80%, respectively. Five of the 10 patients with a severe incomplete cord lesion (Frankel B, C) had about 50% stenosis of the bony spinal canal, but severe incomplete cord lesions were also observed in patients with 15–30% stenosis. Patients with mild cord lesions (Frankel D and E) often had a spinal stenosis of between 40 and 70%. In one patient stenosis was less than 10%. In two patients the degree of narrowing remained unknown (no CT available).

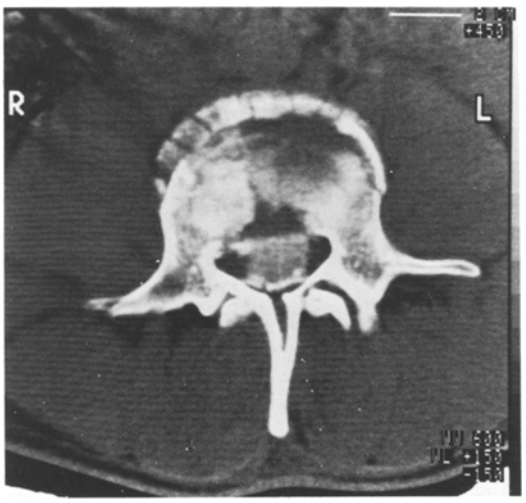

Fig. 3. CT of patient with burst fracture at L3 and 90% reduction of sagittal diameter of the spinal canal. This patient had a mild reversible lesion of the cauda equina!

Table 2. *Relationship Between Neurological Deficit and Percentage Reduction of the Sagittal Diameter of the Bony Spinal Canal in Patients with a Burst Fracture*

Frankel scale	n	Percentage reduction of sagittal diameter: level of the spine: thoracic	thoracolumbar	lumbar
		%	%	%
A	4	70	44; 80	?
B	5	?	46; 50; ?	>90
C	11	<10; <10 33	15; 30; 30; 50; 50; 57; ?	?
D	19	<10; 33	<10; 40; 40; 43; 50; 50; 50; 67; 67; 70	36; 48; 57; 68; 80; 90; 90
E	1	40		

Example: A 70 means that this patient with a complete cord lesion (A) had a 70% reduction of the sagittal diameter of the spinal canal. ?: amount of reduction unknown because CT is not available.

Only one patient with a burst fracture of the *lumbar spine* suffered a complete lesion. CT was, however, not available, because of other severe injuries. Seven of the 10 patients had a relatively mild lesion according to the Frankel classification, although the degree of narrowing was 90% in two patients (Table 1) (Fig. 3).

Fracture-dislocation. Of the 28 patients with a fracture-dislocation, 17 (61%) had a complete cord lesion (Frankel grade A) and six a complete motor paralysis with some sensory sparing (Frankel grade B). The remaining five patients had moderate or mild incomplete lesions (Table 1).

Neurological deficit and residual stenosis at follow-up. Of the 40 patients with burst fractures, 33 consented to follow-up CT. In those who were conservatively treated, the narrowing of the sagittal diameter usually showed a 10–40% improvement compared to the original narrowing (e.g. 50% initially, 35% at follow-up). In patients who underwent surgical decompression and stabilization, the remaining narrowing of the sagittal diameter was usually less than 10%; in some cases 20–40%. The improvement in narrowing in these surgically decompressed cases was on the whole much larger than in conservatively treated patients, e.g. initially 60% narrowing of the sagittal diameter of the spinal canal, at follow-up 10%. Nevertheless, a correlation between improvement in neurological deficit and improvement in narrowing, or a correlation between the remaining neurological deficit and degree of persistent narrowing,

Table 3a. *Relationship Between Degree of Neurological Deficit on Admission and at Follow-up in 69 Patients* (in one initially comatose patient, final outcome is E, but the initial category is unknown)

Initial \ Follow-up	A	B	C	D_1	D_2	E
A	22					
B	1	1	2	4	3	
C			4	2	4	1
D					7	17
E						1

Table 3b. *Relationship Between Initial Neurological Deficit and Deficit at Follow-up in the Patients Who Were Managed Conservatively.*

Initial \ Follow-up	A	B	C	D_1	D_2	E
A	17					
B		1		3	2	
C			1	1	2	1
D					2	3
E						1

Table 3c. *Relationship Between Neurological Deficit on Admission and at Follow-up in the Patients Managed Surgically.*

Initial	Follow-up A	B	C	D_1	D_2	E
A	5					
B	1		2	1	1	
C			3	1	2	
D					5	14
E						1

1 patient: final outcome E; initial category unknown (in coma)

as seen on the follow-up CT, could not be established. As an example: in the three patients who were initially grade C (Frankel) and were surgically decompressed and stabilized, the narrowing was reduced to less than 10% but they still remained in grade C. On the other hand, two patients who were initially grade B, improved to grade D_2, although the narrowing of the sagittal diameter was only slightly reduced. These findings cannot be adequately represented in a table.

Recovery of neurological deficit and type of management. Neurological recovery, categorized according to the Frankel scale, is presented in Table 3a (all patients), 3b (conservative management), 3c (surgical treatment). All 22 cases with a complete lesion remained complete. One of the patients with some sensory sparing lost this sensibility during treatment, but the others in category B improved, 3 patients even to the D_2 category. Seven of the 11 patients in grade C on admission improved to grade D or E and 17 of the 24 patients with grade D on admission became grade E.

The recovery in the 11 patients in grades B and C, who were treated operatively by decompression of the dural sac and surgical stabilization, is not strikingly different from the recovery in the 11 patients who were treated conservatively. Nor is there a significant difference between the neurological recovery in the 19 cases in grade D on admission, who were treated operatively, and the 5 patients in that category who were treated conservatively.

Discussion

As shown in Table 2, we cannot establish a correlation between degree of initial neurological deficit and treated conservatively.
amount of narrowing of the sagittal diameter of the spinal canal. There is a large overlap in ranges of narrowing, especially in thoracolumbar and lumbar injuries between the cases with severe and those with mild deficit. A remarkable finding was the 90% reduction of the sagittal diameter of the spinal canal in the lumbar area, in combination with a mild and reversible cauda lesion (Table 2), (Fig. 3).

In 80 patients with acute traumatic thoracic, thoracolumbar and lumbar injuries, Keene *et al.*[19] reported a significantly larger degree of narrowing in patients with complete and incomplete lesions compared to patients without deficit. As in our series, however, the range of narrowing was similar at all levels. Other authors[5, 19, 22] have reported the same lack of correlation.

Hashimoto *et al.*[16] evaluated the relationship between neurological deficit and the cross-sectional area of the original spinal canal and the area occupied by the retropulsed bone fragments on the CT-scan, using a microcomputer assisted digitizer. They concluded that small bony fragments brought about a greater incidence of neural damage at the epiconus-conus level than at the cauda equina level. A spinal canal stenosis ratio of 35% or more at the epiconus level (T11-T12), 45% or more at the conus medullaris level and 55% or more at the cauda equina level were reported to be significant factors for neurological impairment in thoracolumbar burst-fractures. The narrowing of the mid-sagittal diameter or the cross-sectional area of the original spinal canal, as shown on the initial CT, does not reflect the level of impact on the cord, conus or cauda at the moment of injury. It cannot, therefore, according to these authors, be used as a basis for the decision whether or not to perform surgical decompression of these structures.

Lindahl *et al.*[2] established that there was a significant correlation between the degree of involvement of the spinal cross-sectional area and the mid-sagittal diameter. In our experience it is theoretically preferable to use computerized measurement of the cross-sectional area when assessing the degree of involvement of the spinal canal. However, retrospectively, an assessment with a digitial planimeter is very difficult to obtain and there is a large degree of observer variation.

We confirm Lindahl's finding that measuring the

mid-sagittal diameter – which is easily performed – is a reliable and accurate method.

In patients with *fracture-dislocations* with, sometimes marked, misalignment and deformation of the spine, an attempt to assess the narrowing of the transverse plane of the spinal canal resulted in no more than an estimate in most cases. Complete cord lesions occurred in patients in whom the computer-tomogram (performed on admission) suggested a maximal compromise of the spinal canal at one level of 80% or more, but also in patients in whom the compromise seemed to be 35% or less. In this group, a correlation between neurological deficit and spinal canal compromise could not be established, because the often oblique plane of the narrowest section prevented accurate measurement.

Many authors have reported that recovery of neurological deficit does not correlate with the treatment method or with the amount of canal decompression[7, 17, 20, 26, 29]. Those who believe that the neurological deficit in spinal injuries is mainly the result of damage at the amount of impact, (like – before the CT era – Bedbrook[1], Frankel *et al.*[13], Guttmann[15] and – more recently – Lindahl *et al.*[22], Osebold[26] and Weinstein *et al.*[28]) favour conservative management, because in their opinion surgical decompression has nothing to offer.

On the other hand, Bohlman[2, 3], Denis[8], Dickson *et al.*[10], Flesch *et al.*[12], Jacobs *et al.*[18], McAfee[24], and others are convinced that persisting compression of neural tissue should be surgically removed. They do not deny neurological improvement with conservative management, but consider the level of improvement after surgical decompression to be superior to that reported in conservatively treated series.

According to them, when cord compression has been diagnosed on CT-myelography or MRI, this compression should be removed to enhance neurological recovery, even in patients with a persisting slight deformity of the spinal canal and an unchanging neurological deficit.

Many of these authors refer to publications such as those by Maiman *et al.*[23], McEvoy *et al.*[25], Bohlman and Freehafer[2] and Larson *et al.*[21] who report significant and prompt neurological improvement after decompressing the cord and conus weeks[21] to even 22 months after injury[2].

However, Frankel[13], Guttmann[15] and others show that in incomplete lesions, conservative management also usually results in neurological improvement, which may be remarkable. Most conservatively treated patients with incomplete lesions showed a similar degree of improvement to those who underwent surgical decompression and stabilization, despite the fact that the physical condition of the conservatively managed patients was generally far worse due to other major extraspinal injuries.

From our and other series it is clear, that it will be very difficult to prove that surgical decompression and stabilization enhances neurological recovery compared to conservative management, because differences in results between the two groups are very hard to detect (if they exist at all).

In recent years, we have also been performing surgical decompression in *incomplete lesions* via a posterolateral approach[11, 14]. In the same operative session the spine can be stabilized. The chances of neurological deterioration as a result of surgery are very small indeed.

If, however, other major injuries form a relative contra-indication to operation or the patient refuses surgery, the chances of neurological recovery are probably no less than if surgical decompression had been performed. Although with conservative management, it may take longer before the patient can be verticalized, in particular in cases of thoraco-lumbar and lumbar injuries, the end-result will usually be a stable spine.

There is no indication for surgical decompression of the dural sac or roots in *complete lesions*. The value of a surgical stabilizing procedure is mainly the immediate stabilization of the spine, which diminishes pain, facilitates nursing care and allows more rapid mobilization. This results in a shorter stay in hospital and earlier active rehabilitation.

We were unable to establish a relationship between the improvement in neurological recovery or in the degree of final neurological deficit and the amount of reduction of the narrowing of the sagittal diameter of the spinal canal. Nor could we establish a relationship between neurological recovery and the degree of persistent reduction of this sagittal diameter. This lack of correlation may be partially explained by the finding of Chakera *et al.*[6], who reported that in 15 patients with burst spinal fractures treated conservatively and available for follow-up, 13 showed spontaneous correction of the previously measured spinal canal stenosis due to resorption of intraspinal bone fragments.

We also saw a few cases showing similar resorption of retropulsed bone fragments.

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