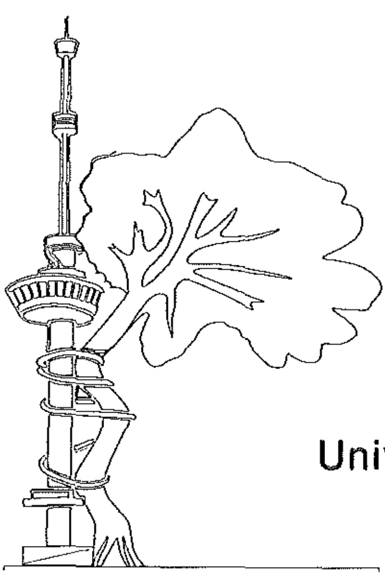
Burst fractures of the thoracic and lumbar spine

Operative versus Conservative treatment



University Hospital Rotterdam

Department of Orthopaedics

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Cover: From *'Dix livres de chirurgie'*, 1564 by Ambroise Paré

Reduction of a spine by traction and direct manual and mechanical pressure. Paré was the one of the first to emphasize the importance of handling dislocations and fractures of the spine with great care.

Cover lay-out: Hans Dolieslager, Goedereede

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Burst fractures of the thoracic and lumbar spine

Operative versus Conservative treatment

Verbrijzelings fracturen van de thoracale en lumbale wervelkolom

Operatieve versus Conservatieve behandeling

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	Prof.Dr.Ir. C.J. Snijders

Voor Ans

Aan mijn ouders

Contents

Chapter I

Introduction

History

Aesculapius was believed by most poets to have been the son of Apollo, the god of medicine, and the nymph, Coronis. He has been referred to as a perfect physician living in the thirteenth century B.C. Aesculapius became an object of worship to his followers, known as Aesclepiads, after Zeus slew him with a thunderbolt. According to biographers, Hippocrates was born on the island of Cos in 460 B.C. His father, who was an Aesclepaid, was his first teacher.[2] Hippocrates is believed to be the first to disassociate medicine from religion and to systematize the various teachings into medical science.[37] Hippocrates described his personal observations in the section of his work on the treatment of fractures and dislocations. In the treatment of spinal injuries Hippocrates did not differentiate between fractures and dislocations, nor was his differentiation between traumatic lesions and spinal disease very clear. Hippocrates began his comments on the treatment of spinal injuries with a procedure known as 'succussion on a ladder', which should be done in the following manner:

'The ladder is to be padded with leather or linen cushions, laid across, and well secured to one another, to a somewhat greater extent, both in length and breadth, than the space which the man's body will occupy; he is then to be laid on the ladder on his back, and the feet, at his ankles, are to be fastened, at no great distance from one another, to the ladder, with some firm but soft band; and he is further to be secured, in like manner, both above and below the knee; and at the groins and chest loose shawls are to be put round in such a fashion as not to interfere with the effect of the succussion; and his arms are to be

fastened along his sides to his own body, and not to the ladder. When you have arranged these matters thus, you hoist up the ladder, either to a high tower or to a gable-end of a house; but the place where you make the succussion should be firm, and those who perform the extension should be well instructed, so that they may let go their hold equally to the same extent, and suddenly, and that the ladder may neither tumble to the ground on either side, nor they themselves fall forward. But, if the ladder be let go from a tower, or mast of a ship, fastened into the ground with its cordage, it will be better, so that the ropes run upon a pulley or axel-tree. But it is disagreeable even to enlarge these matters; and yet, by the contrivances now described, the proper succussion may be made.'

In today's perspective, treatment of fractures of the thoracolumbar spinal column by 'succussion', especially with more or less loss of stability, is regarded as very dangerous; it can even be fatal in several types of fractures. More important than the succussion are the various methods of traction which Hippocrates used.[61]

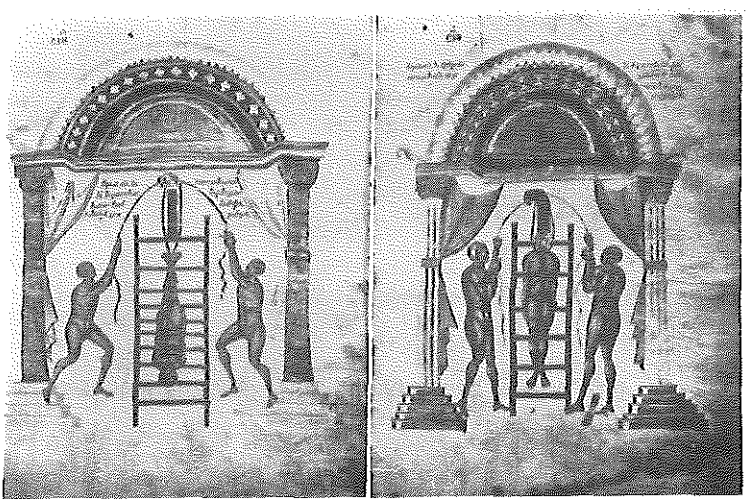

Figure 1.1 *Reduction of spinal vertebrae on a ladder; Apollonius of Citium (c. 81-58 B.C.).*[61]
These illustrations are believed to be direct copies from original Greek drawings.

Oribasius (325-400 A.D.) was a Greek physician who gained fame by improving the earlier methods described by Hippocrates (Fig.1.2).[59] Another Greek physician, living between 625 and 690, was Paul of Aegina. Although his remarks on dislocations of the spine were not original, his comments on treatment of fractures of the spine were; he originated the laminectomy and suggested that the bone, compromizing the spinal cord, should be removed if possible.[3]

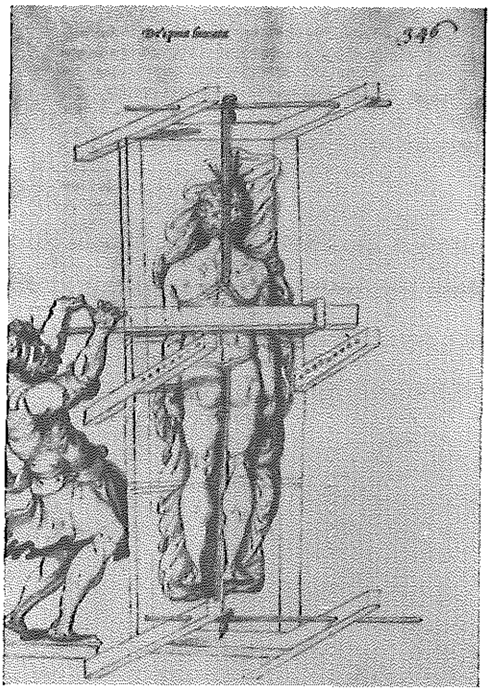

Figure 1.2 A modification of Hippocrates' bench made by Oribasius.

During the Renaissance, the Italian, Hieronymus Fabricus ab Aquapendente (1537-1619), who was a surgeon as well as an anatomist, seems to have been interested in various kinds of braces. In his Opera Chirurgica, there is a description, although not very understandable, of the instruments to be used for the reduction of spinal gibbosities (Fig.1.3).[33]

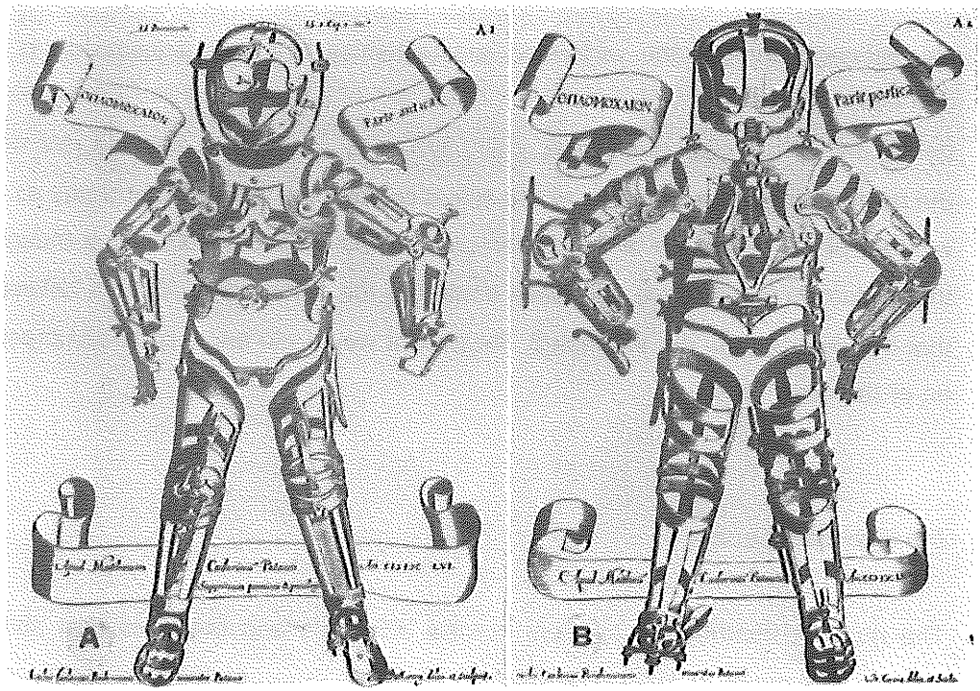

Figure 1.3 *Surgical armor, Hieronymus Fabricius ab Aquapendente.*

The Nineteenth Century

In the nineteenth century, when Louis Pasteur discovered that micro-organisms cause disease and ether anesthesia was introduced, there was a particular interest in the treatment of spinal injuries. Some of the most time-honored errors were corrected. For example, since Hippocrates, crushed fractures of the vertebrae had been confused with dislocations. In the nineteenth century, surgeons realized this mistake and corrected it in their treatises by stating that in trauma of the spinal column, fracture of some part of the vertebrae is almost always present.

In 1807, Sir Charles Bell wrote a treatise advocating non-operative treatment of spinal injuries, mainly as a attack on Sir Ashley Cooper, who favored a laminectomy, although none of the performed laminectomies had been successful up to that time.[10,20]

By the end of the nineteenth century all physicians agreed that absolute rest was the best treatment for fractures of the spinal column, but they did not agree as to whether or not reduction of spinal fractures should be performed. Some thought that reduction would drive any loose bone into the spinal canal. Others believed that if reduction was not attempted, the patient would die because of compression of the cord.

The Twentieth Century

The greatest contribution to more precise diagnosis and better care of spinal injuries was made in 1895. The German physicist, Konrad Röntgen, discovered the X-ray, which led to the development of radiography. Radiography enabled the diagnosis and understanding of injuries of the vertebrae to reach a high level of accuracy.

Treatment of spinal injuries has improved greatly during the twentieth century. Braces and plaster casts were used for spinal support. In 1929, Davis introduced hyperextension as a method of correction and immobilization for compression fractures in the lower thoracic and lumbar vertebral bodies.[23] Similar methods were advocated by Watson-Jones, and were widely used and considered the methods of choice for two decades (Fig. 1.4).[63]

Böhler, however, was not convinced that correction alone was practical or advisable, and used support for a shorter period and emphasized exercise and early return to activity.[12] He stated that fractures of the thoracic and lumbar spine should be treated as any other fractures. He strongly emphasized the importance of a good reduction of the fracture.

This philosophy has been advocated by Watson-Jones who stated in 1944 that a perfect recovery is only possible if there is a perfect reduction.[64]

Nicoll reported in 1949 that 152 coal miners with 166 fractures of the thoracic and/or lumbar spine returned to work sooner without fracture reduction, thus excepting the deformity of the vertbrae.[52] He stated that a good functional result does not depend on a good anatomical result. Nicoll was the first to classify fractures of the thoracic and lumbar spine in stable and unstable fractures. According to Nicoll stable fractures should be treated functionally, without an orthosis, in order to get early mobilization. Unstable fractures should be treated, without reduction, in an orthosis in order to get anterior fusion of the fractured vertebrae. He accepted the deformity of the vertebrae as long as the functional results were good.

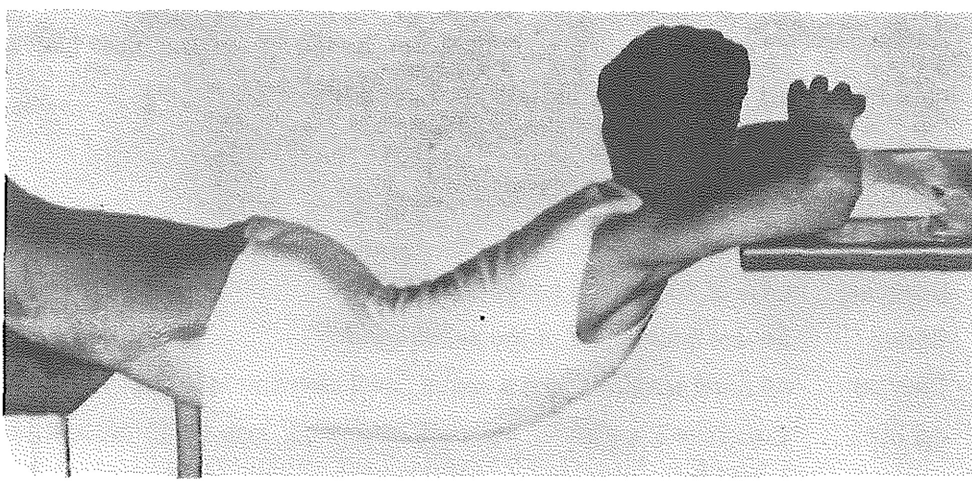

Figure 1.4 *The hyperextension method of Watson-Jones for obtaining correction of vertebral compression.*

Since the availability of effective instrumentation and, later, with the introduction of computed tomography, the optimal treatment of thoracolumbar burst fractures has become more controversial.[4,13,16,27,29,32,40,41,42,45,46,48,50,53,62]

Harrington instrumentation has, since 1967, been increasingly used to stabilize fractures of the thoracic and lumbar spine in order to get early mobilization and rehabilitation and to decrease the risks of complications due to a long bedrest.[1-9,13,19,29,35,40,44,45,53,59]

A number of researchers have reported that conservative treatment results in neurological injury, progressive spinal canal stenosis, progressive body collapse, increased kyphosis and late severe radiculitis.[25,27,28,30,47,49,67,68] Many favoring operative treatment feel that failure of the middle osteoligamentous complex, particularly with retropulsion of bone fragments into the spinal canal, indicates spinal canal instability with impending neurological deficit.[11,14,26,27,28,29,30,38,44,47,54,55,66,70] Several empiric criteria, largely based on radiographic findings, have been used as indications for operative treatment.

Some of these indications are:

Bohlman:	Loss of 40 percent body height.[11]
Denis:	Severe obstruction of the spinal canal.[27]
DeWald:	All burst fractures with a spinal canal diameter less than 10 mm.[28]
Jacobs:	Evidence of posterior ligament disruption; loss of 40 percent body height.[44]
Kostuik:	Significant retropulsion.[47]
Willen:	Fifty percent spinal canal stenosis; 50 percent compression of the anterior column.[69,70]

Failure of the instrumentation used in the operative treatment of thoracolumbar burst fractures is mentioned by a few authors.[9,31,56] Arthrodesis of several vertebrae alters the biomechanics of the spine. Loss of motion, especially between lumbar vertebrae, has been implicated as a cause of degenerative changes in the joints immediately cranial and caudal to the fusion mass in patients with a spinal arthrodesis.[1,15,24,51]

Until the 1970s, conservative treatment was the worldwide standard. Many centers found that postural reduction and prolonged bedrest can yield acceptable results in the treatment of thoracolumbar burst fractures. The recommended duration of recumbency and bracing are largely empiric.

Guttmann, and his successor Frankel, started the more active treatment of unstable fractures of the spine by introducing postural reduction and bedrest.[39,36] Bedbrook felt clinical stability could be achieved after 6-8 weeks postural reduction and bedrest, after which the paravertebral callous is radiographically evident.[7,8] No bracing was necessary after this period, the strong paravertebral muscles providing satisfactory support.

Holdsworth et al recommended a longer period of immobilization for unstable fractures; 8-12 weeks in plaster followed by a few weeks in a light jacket.[43]

Krompinger et al set more specific criteria regarding management of burst fractures.[49] If the spinal canal compromise is less than 50 percent, patients are managed with 3-4 days of bedrest followed by bracing; if the spinal canal compromise is greater than 50 percent, bedrest was extended to 4-6 weeks also followed by bracing.

Weitzman noted that poor outcome of some conservatively treated patients was related to the duration of the treatment. His study supports the shortening of bedrest in stable fractures to 8 days.[66]

A series of reports described the outcome of conservative treatment.[6,7,8,16,17,21,22,34,44,57,60,65] Mumford et al reported that the outcome of conservative treatment in neurologically intact patients with a thoracic or lumbar burst fracture was acceptable, regarding bony deformity.[57] Cantor et al concluded the same in a group of neurologically intact patients without disruption of the posterior column.[16]

The choice to operate on a burst fracture results from a large number of empiric clinical factors. Because questions regarding classification, spinal canal stenosis, remodeling and neurological recovery have not been answered satisfactorily, it is not possible to decide with certainty whether or not to operate on any given burst fracture. In our opinion, an indication for the operative treatment is a burst fracture of the thoracolumbar spine with obvious progressive neurological deterioration. Recent literature reflects a strong trend towards operative management of burst fractures of the thoracic and lumbar spine, in order to get early mobilization, to prevent late deformity stenosis and disabling back pain, and to reduce hospital costs.[11,38,47,48,50] The evidence supporting these indications for operative treatment, however, is insufficient.

Incidence of fractures of the thoracic and lumbar spine

SIG (Informatiecentrum voor de Gezondheidszorg, Utrecht) registers 99.4 percent of all hospital admissions in the Netherlands. Between 1987 and 1991 a yearly average of 2,254 admissions were registered with the main diagnosis: a fracture of the thoracic or lumbar spine. Patients with a fracture of the thoracic or lumbar spine, but with another main diagnosis, were not included in this registration; neither was the type of the fracture registered. A yearly average of 130 patients have a neurological deficit due to the trauma. An average of 170 patients are treated operatively every year (Table 1.1).

Table 1.1 *The yearly average of thoracic and lumbar spine fractures with and without a neurological deficit recorded by SIG between 1987 and 1991.*

THORACIC	Male	Female	Total	Operative (%)
With neurological deficit	43	28	71	24 (34)
Without neurological deficit	335	414	749	40 (5)
Total	378	442	820	64 (8)

LUMBAR	Male	Female	Total	Operative (%)
With neurological deficit	32	27	59	20 (35)
Without neurological deficit	644	731	1375	86 (6)
Total	676	758	1434	106 (7.4)

Aim of this retrospective study

Over the last 150 years a controversy has persisted regarding the management of spinal fractures, especially the early management of unstable fractures. Recent literature continues to address the controversy of early operative management versus conservative management of thoracolumbar burst fractures. However, there are only a few small studies comparing the outcome of the operative and conservative treatment of these fractures.
The purpose of this study was to review outcome of the operative and the conservative treatment of burst fractures of the thoracic and lumbar spine.

The principal objectives of this thesis are:

1. To determine the correlation between the presence of a neurological deficit and the extent of spinal canal stenosis in thoracolumbar burst fractures.

2. To investigate the phenomenon of spontaneous redevelopment of the spinal canal after conservative treatment of thoracolumbar burst fractures.

3. To review the neurological recovery in both operative and conservative treated patients with a burst fracture of the thoracolumbar spine.

4. To compare the outcome in burst fractures treated operatively and conservatively, in terms of social functioning and work status.

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Chapter II

Anatomy and Classification

Anatomy of the thoracic and lumbar spine

Usually there are 12 thoracic and 5 lumbar vertebrae, but there may be one more or less in either region. Certain features of the last two thoracic vertebrae resemble the lumbar vertebrae. Especially related to trauma of the spinal column it is practical to divide the thoracic and lumbar spine into three sections; these are:

Thoracic *(T1 to T10)*
Thoracolumbar *(T11 to L1)*
Lumbar *(L2 to L5)*

The thoracolumbar junction (T11 to L1) is particularly vulnerable. This region represents a transition from the relatively rigid thoracic spine (fixed by the spine-ribcage-sternum complex) to the more highly mobile lumbar spine, uninhibited by anatomical structures. In fact, the facet joints in the upper lumbar region promote flexion and extension because of their coronal facing.

Each vertebra is composed of several major parts:

The vertebral body with at T2 to T9 an inferior and superior costal pit at each site
The pedicles
The posterior (laminar) arch
The inferior, superior, transverse and spinous processes
The facet joints
The spinal or vertebral canal

The vertebral body has seven processes: two superior and two inferior articular processes, two transverse processes with a costal facet at the thoracic region, and one spinous process. Viewed from above the superior articular processes face dorsally, cranially and laterally while the inferior articular processes face in a ventral, caudal and medial direction. Between the superior and inferior articular processes laterally lies the foramen. Through it pass the nerve (root) and blood vessels.

The facet joints in the thoracic region restrict flexion, extension and axial rotation. In the lower lumbar region, the position of the facet joints allows only flexion and extension.

The intervertebral disc is a synarthrosis between two vertebrae. This disc is composed of two major parts:

The annulus fibrosus
The nucleus pulposus, which lies in the posterior part of the annulus

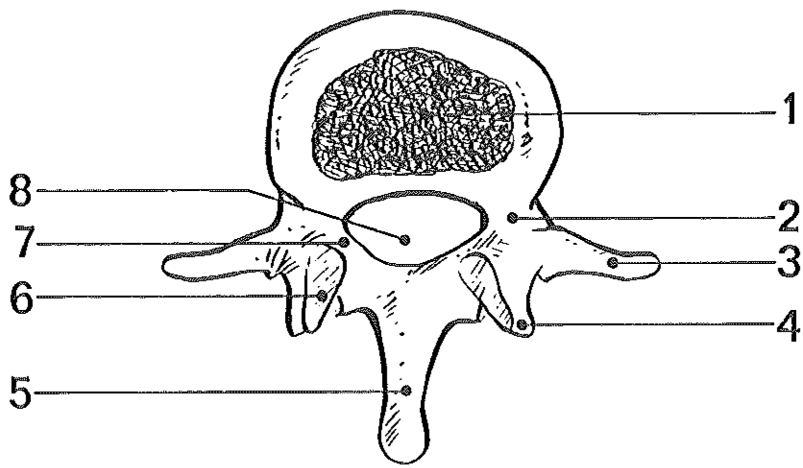

Figure 2.1 *A vertebra viewed from above.*

1. *Vertebral body*
2. *Pedicle*
3. *Transverse process*
4. *Inferior articular process*
5. *Spinous process*
6. *Superior articular process*
7. *Posterior (laminar) arch*
8. *Vertebral canal*

The thoracic spinal canal is smaller and more rounded than the lumbar spinal canal, which is more triangular shaped. The spinal cord lies in the vertebral canal and extends from the atlas to the interval between the first and second lumbar vertebrae where it terminates in the conus. Thirty-one pairs of spinal nerves arise from the spinal cord: 8 cervical, 12 thoracic, 5 lumbar, 5 sacral and 1 coccygeal.

In the thoracic region the anterior spinal artery is the most important provider of blood supply to the spinal cord. It receives tributaries from three sources:

1. *The first intercostal artery*
2. *The vessel of Adamkiewicz (arteria radicularis magna) via its upward watershed blood supply, ascending from below usually at the level T10-11*
3. *Multiple, less important segmental intercostal arteries, which contribute to the overall flow within the anterior spinal artery*

The two posterior spinal arteries, which receive their blood supply from adjacent intercostal arteries, contribute little to the overall vascular supply of the thoracic spinal cord. With regard to fractures of the spinal column, the 'sparsity' of the bloodsupply of the spinal cord and the narrowness of the thoracic spinal canal makes the thoracic region more vulnerable than the lumbar region.
The venous system plays no major or specific role in the metabolisme of the spinal cord because it provides a high vascular turnover and it communicates directly with the venous system draining the head, chest and abdomen.

Several major ligaments can be identified:

The anterior longitudinal ligament is connected with the anterior part of the vertebral bodies and the anterior part of the annulus fibrosus.
The posterior longitudinal ligament is connected with the posterior part of the annulus and the posterior part of the vertebral bodies.
The ligamentum flavum connects two successive laminar arches.
The interspinous ligament lies at the anterior part of two adjacent spinous processes.
The supraspinous ligament lies at the posterior part of two adjacent spinous processes.

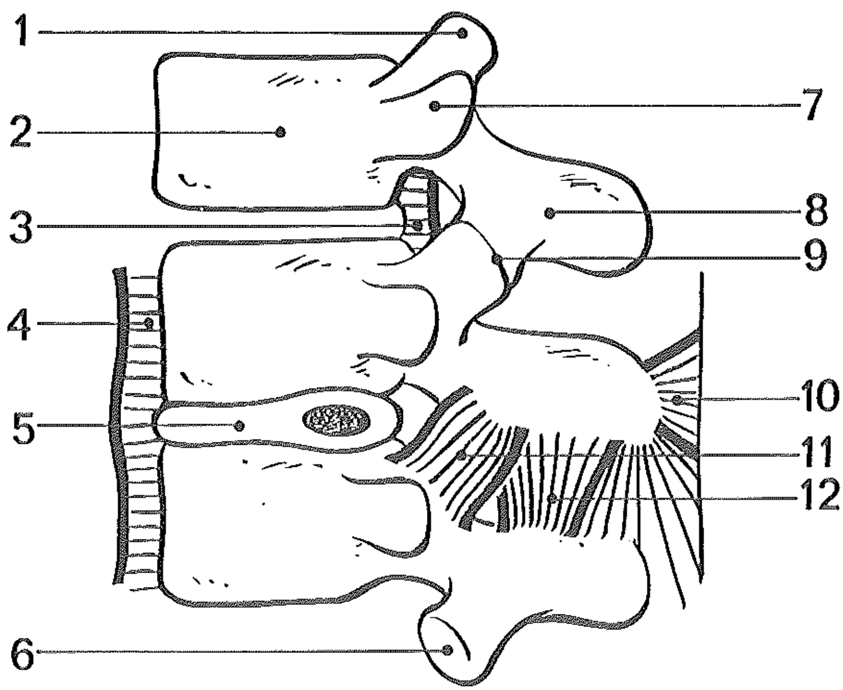

Figure 2.2 *Three vertebrae with surrounding structures.*

1.	*Superior articular process*	7.	*Transverse process*
2.	*Vertebral body*	8.	*Spinous process*
3.	*Posterior longitudinal ligament*	9.	*Facet joint*
4.	*Anterior longitudinal ligament*	10.	*Supraspinous ligament*
5.	*Intervertebral disc*	11.	*Ligamentum flavum*
6.	*Inferior articular process*	12.	*Interspinous ligament*

Classification of fractures of the thoracolumbar spinal column

Since Holdsworth subdivided the spine into two columns, our understanding of thoracolumbar injuries has improved dramatically.[4] However, he insisted that rupture of the posterior column was sufficient to create instability of the spine. But, several reports have demonstrated that only after rupture of the posterior longitudinal ligament and of the annulus fibrosus (middle column) loss of stability occurs.[1,5,6] Therefore, a new biomechanical concept and classification of thoraco-lumbar fractures was introduced by Denis.[3] He replaced the two-column theory by the three-column theory. The third, or middle column, is represented by structures that have to be torn in addition to the posterior ligamentous complex in order to create more or less loss of stability.

The criteria on which most classifications are based are fracture stability and neurological injury.

In our view, instability represents a spectrum rather than an 'all or none' phenomenon. In many instances, it is not possible to state that a spinal fracture is unstable or stable. To futher complicate these classifications, the presence or absence of neurologic injury cannot be used in the stability classification, because there are many cases in which a stable fracture is associated with the presence of a neurological deficit, and an unstable fracture without the presence of a neurological deficit.

In this study we used the three-column system according to Denis.

The three-column system

The anterior column

The anterior longitudinal ligament

The anterior part of the annulus fibrosus

The anterior part of the vertebral body

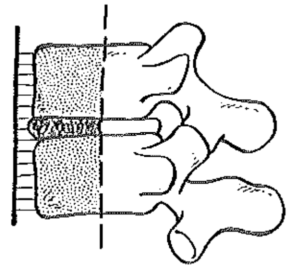

Figure 2.3 *Anterior column*

The middle column

The posterior longitudinal ligament

The posterior part of the annulus fibrosus

The posterior wall of the vertebral body

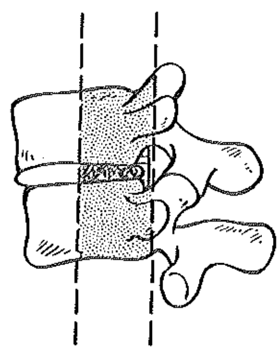

Figure 2.4 *Middle column*

The posterior column

The posterior (laminar) arch

The pedicles

The supraspinous ligament

The interspinous ligament

The facet joint capsule

The ligamentum flavum

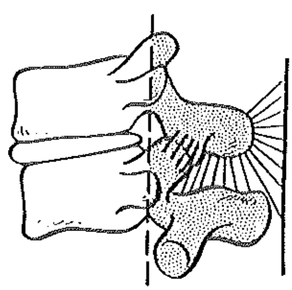

Figure 2.5 *Posterior column*

Failure of the three columns divides spinal fractures into four different types:

Compression fracture | **Fracture dislocation**
Seat-belt type fracture | **Burst fracture**

Table 2.1 *Basic modes of failure of the three columns in the four major types of spinal injury; Denis*[3]

Type of fracture	COLUMN		
	ANTERIOR	MIDDLE	POSTERIOR
COMPRESSION	compression	none	none or distraction
SEAT-BELT TYPE	none or compression	distraction	distraction
FRACTURE DISLOCATION	compression axial rotation shear	distraction axial rotation shear	distraction axial rotation shear
BURST FRACTURE	compression	compression	none/slight

Compression fracture

The compression fracture is a failure under compression of the anterior column caused by either anterior or lateral flexion. The middle column is intact.

In severe cases, there may be a partial failure of the posterior column. This type of fracture is regarded as stable. Usually the compression involves the superior end plate and the ventral part of the vertebral body, occasionally the fracture is localized at the inferior end plate and the inferior surface of the vertebral body.

Radiographic characteristics

These fractures are best identified in the lateral projection. The vertebral body is wedge-shaped with a decrease in height of the anterior vertebral body. The posterior height remains unchanged. The CT-scan demonstrates a rupture of the anterior end plate. The vertebral ring (posterior wall, pedicles and lamina) is intact (Fig.2.6).

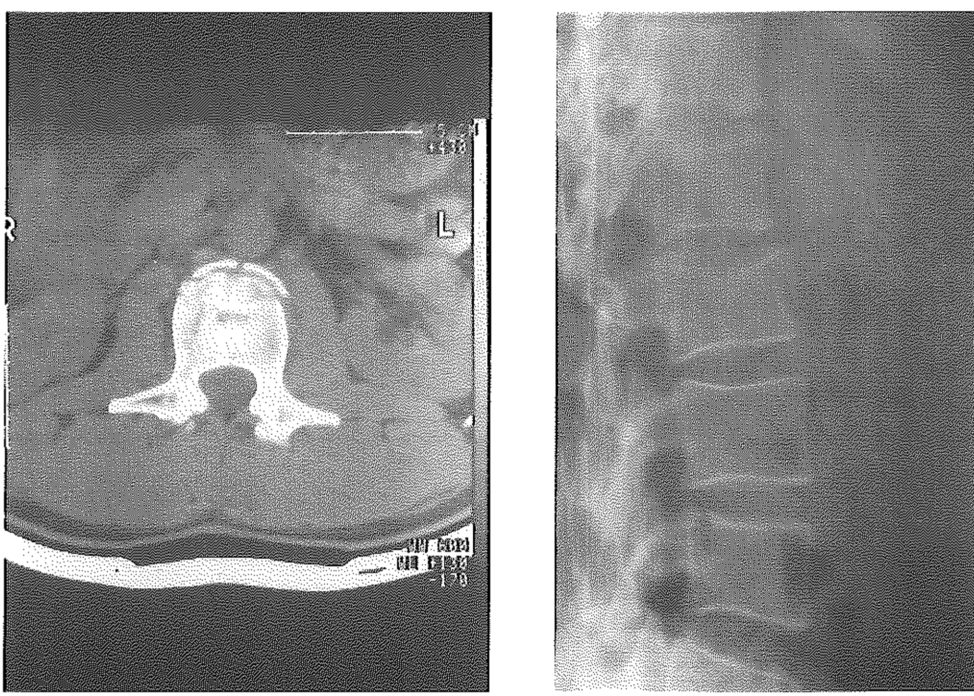

Figure 2.6 *CT scan and a lateral radiograph of a compression fracture of lumbar vertebra 2.*

Seat-belt type fracture

This type of transverse or horizontal fracture represents failure of both the posterior and middle column under tension forces caused by flexion and sometimes by superimposed distraction. The anterior part of the anterior column may also partially fail, but without losing its role as a hinge (this will differentiate it from the fracture dislocation, where the hinge is also disrupted). Pure transverse fractures of the spine were first described by Chance and are known as 'Chance fractures' (one level bone injury).[2] Seat-belt type fractures may involve one (most common) or two levels:

One level lesion:

Chance Fracture (Fig. 2.7)

Ligamentous injury, involving all ligaments from the supraspinous to the middle of the intervertebral disc

Two level lesion:

Middle column rupture through bone

Middle column rupture through posterior annulus fibrosis

Radiographic characteristics

There is an increase of the interspinous distance, with a horizontal split of the transverse processes, a horizontal split of the pedicles and pars interarticularis fractures. Typical is the increased height of the posterior vertebral body and the fracture of the posterior wall of the vertebral body and posterior disc space (rupture of the annulus fibrosis). The CT scan does not provide much information because the CT-slices are often parallel to the plane of the fracture itself.

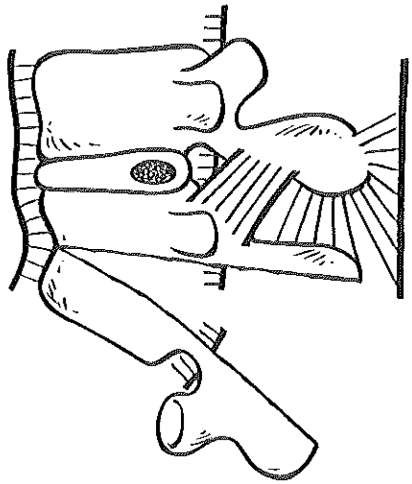

Figure 2.7 *Lateral view of a seat-belt type fracture through bone ('Chance' fracture).*

Fracture dislocation

The main characteristic of the fracture is that there is a failure of all three columns caused by flexion, compression and rotation/shear. This leads to subluxation or dislocation often in combination with facets interlocking. The typical fracture dislocation shows an anterior displaced vertebra, fracture of the posterior elements, fracture of the superior or inferior facets and, sometimes, a fracture of the anterior vertebral body.

Radiographic characteristics

The most important characteristic of the fracture dislocation is the (sub)luxation or dislocation (lateral view), and the CT-scan may show a reduction of the spinal canal.

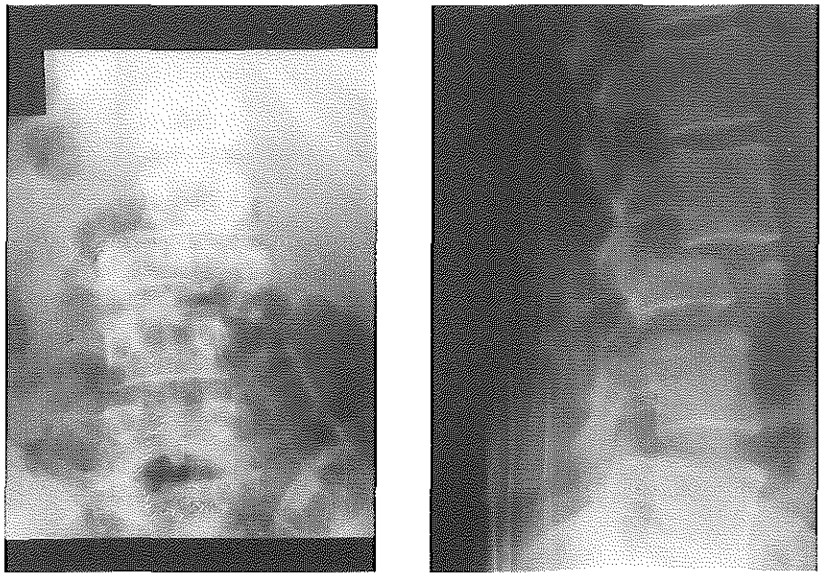

Figure 2.8 *Anteroposterior and lateral radiograph of a fracture dislocation of lumbar 3.*

Burst fracture

Severe compressive forces can result in an explosion of the vertebral body displacing the fragments centripetally. Frequently, the posterior superior fragment is driven into the spinal canal. The burst fracture is a result of failure of both the anterior and middle column caused by axial loading and flexion.

Radiographic characteristics

There is a fracture of the posterior wall cortex of the vertebral body, loss of posterior height, an increase of interpediculate distance, fracture of the lamina, and the CT-scan frequently shows retropulsion of bone into the spinal canal.

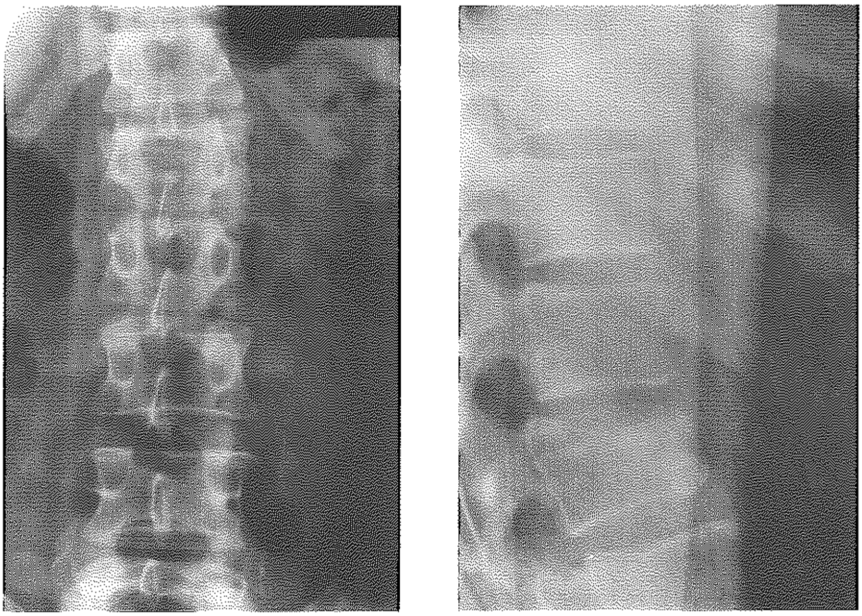

Figure 2.9 Anteroposterior and lateral radiograph of a burst fracture of lumbar 1.

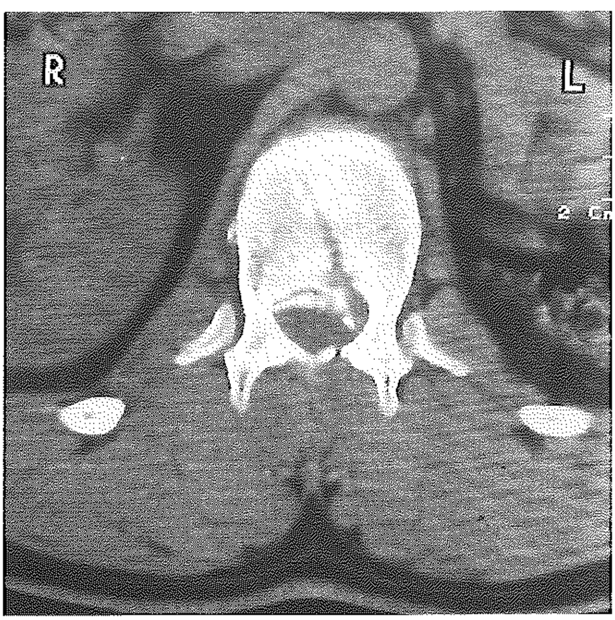

Figure 2.10 CT scan of the same burst fracture (lumbar 1).

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Chapter III

Introduction to the clinical investigation

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Patients and Methods

Introduction to the clinical investigation

The purpose of this study was to review the course of the conservative treatment of thoracolumbar burst fractures and to compare the outcome of the operative and conservative treatment (Chapter I, page 12).

In **Chapter III** the patients and methods are described of this study. Futhermore, this chapter also includes results relating to the cause of injury.

Chapter IV describes the results of the relationship between the percentage spinal canal stenosis, the presence of a neurological deficit and the level of injury. This study included 139 patients with a thoracolumbar burst fracture. Forty-nine patients had a neurological deficit and the severity of the neurological deficit was correlated with the percentage spinal canal stenosis.

In **Chapter V** we investigated the remodeling of the spinal canal after conservative treatment of thoracic, thoracolumbar and lumbar burst fractures. The population of the study of this chapter is formed by 42 patients with an initial spinal canal stenosis of more than 25 percent.

In Chapter VI we investigated the amount of neurological recovery in relation to the type of treatment (operative vs conservative), the initial percentage spinal canal stenosis, the initial kyphosis and increase in kyphosis. Forty-one patients were included in this study (19 operatively and 22 conservatively treated).

After a follow-up period of 12 to 108 months the outcome of the operative and conservative treatment of thoracolumbar burst fractures was reviewed in terms of radiographic findings, social functioning and work status. The population of the study of Chapter VII was formed by 28 operatively and 73 conservatively treated patients

Patients and Methods

Between January 1981 and January 1991, 226 patients with a fracture of the thoracic or lumbar spine were admitted to the Departments of Orthopaedics or Neurosurgery of the University Hospital Rotterdam.
The fractures were classified according to the three-column system of Denis (Chapter II) (Compression fracture, Seat-belt type fracture, Fracture dislocation and Burst fracture). A seat-belt type fracture was encountered in 1 patient, fracture dislocations in 31 patients and burst fractures in 145 patients. The remaining 49 patients had a compression fracture of the vertebral body (Fig.3.1).

Burst fractures

Computerised tomographs were available of 139 patients with a burst fracture of the thoracic, thoracolumbar or lumbar spine. These patients were included in this study and all were admitted to the Departments of Orthopaedics or Neurosurgery. Treatment of all patients was supervised by a neurosurgeon and an orthopaedic surgeon; both departments used the same treatment protocol (appendix A). Ninety-seven patients were male and 42 were female. Age at time of injury varied from 12 to 83 years (mean: 36 years).

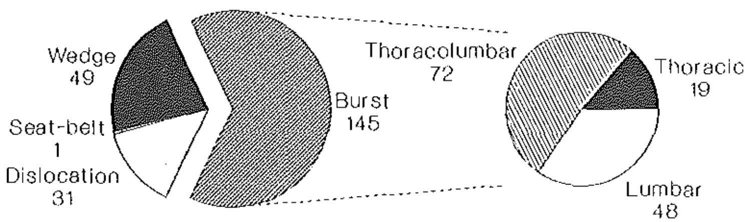

Figure 3.1 *Number of the four types of fractures of the thoracolumbar spine and the location of the burst fractures included in the study population.*

Location of the burst fractures

All burst fractures of this study were classified according to the level of injury in three categories:

1. **Thoracic:** *T1 to T10*
2. **Thoracolumbar:** *T11 to L1*
3. **Lumbar:** *L2 to L5*

Thoracic burst fractures were encountered in 19 (13.7%) patients (14 male, 5 female); thoracolumbar burst fractures in 72 (51.8%) patients (52 male, 20 female); and lumbar burst fractures in 48 (34.5%) patients (31 male, 17 female) (Fig.3.1). No correlation was found between gender and the three levels of injury.

Presence of a neurological deficit

Neurological impairment should always be suspected after severe injury of the spinal column. The effects of the injury on the spinal cord, nerve roots or cauda equina depend on the location, severity and extent of the damage. The mechanisms are primilary local pressure or traction by displaced bone fragments, intervertebral disk and ligaments and, secundarily, ischemia, hemorrhage and edema.

Cord damage at the thoracic level will affect the function of the lower extremities, bladder and rectum, and the intercostal and abdominal muscles. The abdominal muscles are innervated by the roots T5 to T12. The iliopsoas, the anterior and median thigh muscles, and the knee jerk are supplied by the roots L2 to L4. The remaining muscles of the lower extremities, the ankle jerk and the plantar reflex are innervated by the roots L5 to S2. The roots of L1 to L3 innervate the muscles of urine retention. The roots S3 to S5 are responsible for the evacuation of the bladder, rectal control and the anal reflex.

At the thoracolumbar level, the spinal cord and the lumbar and sacral spinal nerves lie together, innervating the lower extremities, bladder and rectum.

On admission 49 (35.3%) of the 139 patients with a burst fracture of the thoracolumbar spine had a neurological deficit, classified according to Frankel.[1]

Neurological deficit was observed in 6 patients (5 male, 1 female) at the thoracic level, in 28 patients (22 male, 6 female) at the thoracolumbar level, and in 15 patients (9 male, 6 female) at the lumbar level.

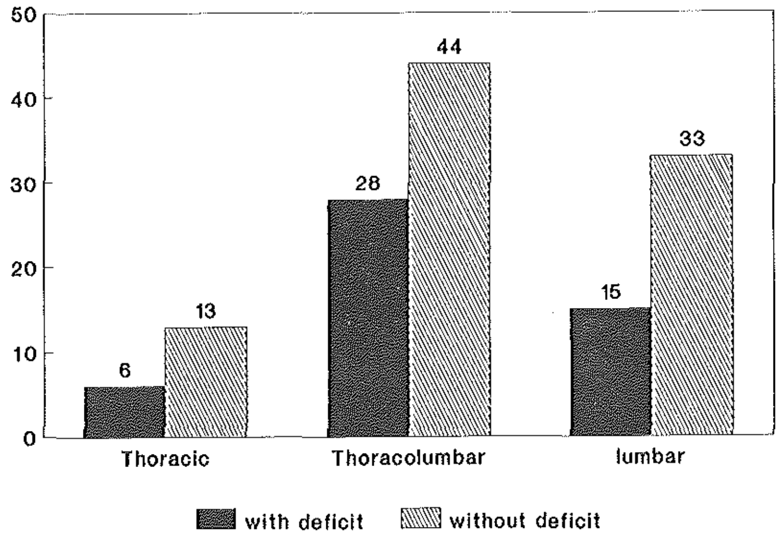

Figure 3.2 *Number of patients with and without a neurological deficit at the three spinal levels.*

Type of neurological deficit

The type or degree of the neurological deficit in the 49 patients with a neurological deficit was assessed on admission by a neurosurgeon using the Frankel[1] scale (Fig.3.2; Table 3.1). The classification of the neurological deficit used in this study ranges from a complete cord lesion (Frankel A) to some reflex abnormalities (Frankel E).

The classification according to Frankel includes five grades:

Frankel A	**Complete**	*No motor or sensory function below the level of injury.*
Frankel B	**Sensory only**	*No motor function, but some sensation preserved below the level of the lesion.*
Frankel C	**Motor useless**	*Some motor function without practical application.*
Frankel D	**Motor useful**	*Useful motor function below the level of the lesion.*
Frankel E	**Recovery**	*Normal motor and sensory function, may have some reflex abnormalities.*

Table 3.1 *Number of patients and percentage of the total group with a neurological deficit classified according to Frankel.*[1]

Frankel classification	Number of patients	*Percentage* of total with a neurological deficit
A (Complete)	5	*10.2*
B (Sensory only)	3	*6.1*
C (Motor useless)	11	*22.5*
D (Motor useful)	29	*59.2*
E (Recovery)	1	*2.0*

Radiology

Following admission, anteroposterior and lateral radiographs were taken of all patients suspected to have a spinal injury. Within 24 hours a computed tomogram was made of those patients with a fracture of the spine. Starting in the emergency room, before the patient was transported to the Department of Radiology, the spine was stabilized with a plaster shell to avoid (futher) damage to the myelum and cauda. On the conventional radiographs, the local kyphosis was measured using the method of Cobb (measuring the angle between the superior end plate above the fracture and the inferior end plate of the vertebral body below the fracture) (Fig. 3.3).
The 139 patients with a burst fracture of the thoracic or lumbar spine were subjected to computed tomography (CT) centered over the damaged part of the spine. On admission as well as at follow-up, the window width was 3200 Hounsfield Units (HU) and the window level was 300 HU. Slice thickness of 3 or 6 mm was used.

The least mid-sagittal diameter of the spinal canal at the level of injury (X) was measured on the CT scan.[2] The normal mid-sagittal diameter of the spinal canal was estimated by calculating the average of the corresponding measurements at the adjacent uninjured vertebrae above (Y_1) and below (Y_2) the fractured vertebrae. The percentage spinal canal stenosis was calculated as:

$$(1 - X / 0.5 (Y_1 + Y_2)) \cdot 100 \%$$

The selected CT-slices for the measurements always demonstrated the least mid-sagittal diameter (Fig.3.4).

It is theoretically preferable to use computerized measurements of the cross-sectional area to assess the degree of involvement of the spinal canal. However, retrospectively, an assessment with a digital planimeter is very difficult to obtain and, as stated by Mumford et al, the visual estimation can compare well with the more sophisticated computer digitization.[3]

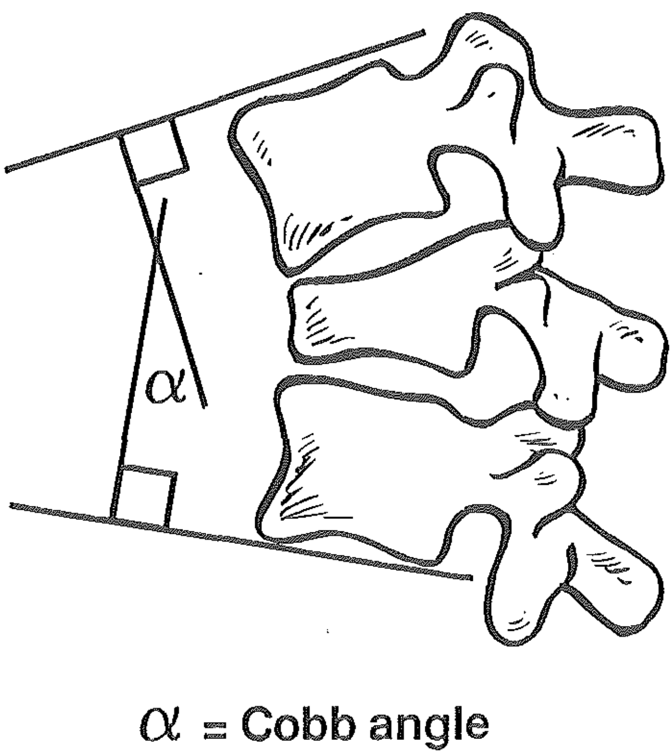

Figure 3.3 *Measurement of the Cobb angle to measure the anterior body collapse (local kyphosis).*

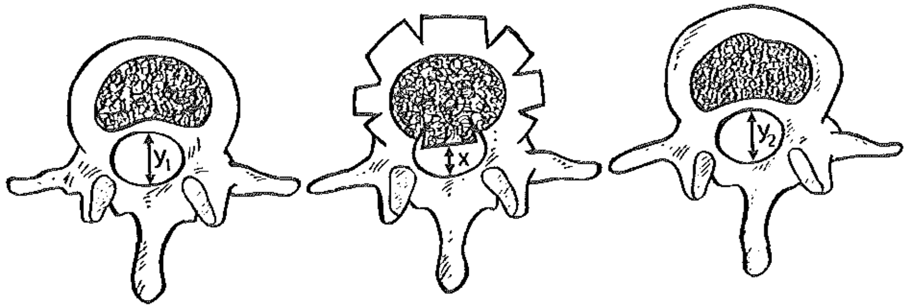

Figure 3.4 *Measurement of the percentage spinal canal stenosis.* $Y = 0.5\ (Y_1 + Y_2)$
Percentage stenosis = $(1\text{-}X/Y).100\%$

Follow-up

Between January 1981 and January 1990, 125 patients, with available CT scans, with a burst fracture of the thoracic, thoracolumbar or lumbar spine were treated in our hospital. At follow-up (Chapter VII), 101 patients were available (10 patients were non-traceble, 5 patients died of unrelated causes, 7 patients refused to cooperate and, the initial radiographs of 2 patients were lost). There were 75 men and 26 women with a mean age at time of injury of 35 years (12-82 years). Thirty-eight patients (37.6 percent) had a neurological deficit on admission (Chapter VII). The study described in Chapter VI included 41 patients with a neurological deficit because sufficient data of three of the non-traceble patients were available. The location of the 101 burst fractures is shown in Fig. 3.5. The follow-up period varied from 12 to 108 months (mean: 40 months). The severity of the neurological deficit was classified according to Frankel (Table 3.2).[1]

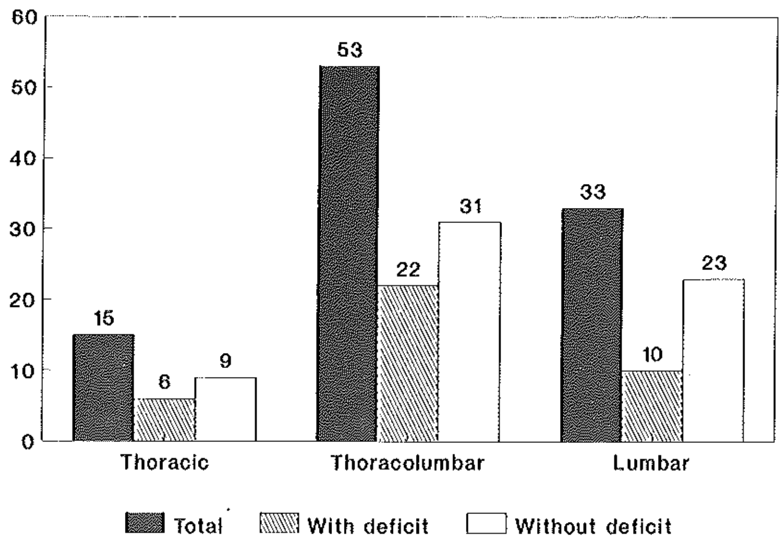

Figure 3.5 *Number of patients at follow-up, with and without a neurological deficit, at the three levels.*

Table 3.2 *Number and percentage of patients included in the follow-up study classified according to Frankel.*[1]

Frankel classification	Number of patients	*Percentage* of total with a neurological deficit
A (Complete)	3	*7.9*
B (Sensory only)	4	*10.5*
C (Motor useless)	9	*23.7*
D (Motor useful)	22	*57.9*
E (Recovery)	0	*0*

Cause of injury

In order to get more than an indication of the cause of burst fractures of the thoracic and lumbar spine, we interviewed the 101 patients included in the follow-up study.
We registered the cause of injury in five categories:

1. Accident at work
2. Traffic accident
3. Sport accident
4. Accident at home
5. Suicide attempt (SA)

Figure 3.6 shows the distribution of the five categories and the gender differences. Gender significantly correlated with the cause of the trauma ($p < 0.001$). Most accidents of the male group of patients occurred at work, while most accidents of the female group occurred at home.

Sixty-eight burst fractures resulted from a fall of more than one meter; mean 6 meters (1-15 meters). As can be expected, these burst fractures occurred mostly at work or were the result of a suicide attempt. While most of the traffic, sport accidents and accidents at home form the group of thirty-three burst fractures which did not result from a fall. We could not establish a correlation between the height of the fall and the presence of a neurological deficit or the level of injury (thoracic, thoracolumbar or lumbar).

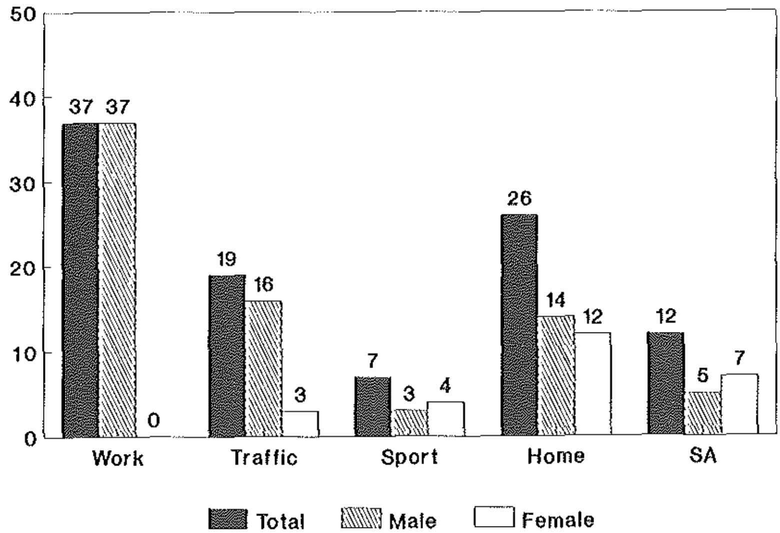

Figure 3.6 *Distribution of patients (total, male and female) according to the cause of injury.*

Operative vs Conservative treatment

The 101 patients with a burst fracture of the thoracolumbar spine included in the follow-up study were either treated operatively or conservatively. With the advent of modern surgical techniques and the introduction of the computed tomography, which clearly showed the retropulsed bony fragments in the spinal canal, since the 1980s burst fractures have been treated more aggressively. In our hospital, the operative treatment was chosen more often in the period from 1981 to 1986 (21 patients).

After this period a few burst fractures, with the presence of a neurological deficit, had to be treated conservatively because other major injuries contra-indicated operative treatment. The recovery of these patients proved to be as good as the operatively treated patients. Between 1986 and 1991 only seven patients with a burst fracture were operated.

This reflects the trend towards conservative treatment in our hospital. In the last years (1990 -to date) only patients with a burst fracture of the thoracolumbar spine, with neurological deterioration, have been treated operatively.

Operative treatment

The posterolateral approach was used in the majority of patients to decompress the dural sac by removal of the loose fragments found in the spinal canal, and by tamping of the displaced vertebral body fragments forward into the vertebral body. Subsequent instrumental reduction and stabilization was achieved either by Harrington rods or - in later years - with a fixateur interne according to Dick (Fig.3.7; Fig.3.8; Fig.3.9).

Conservative treatment

The conservative treatment of the thoracic or lumbar burst fracture included protection of the spine in plaster shells, starting in the emergency room before the patient was subjected to the CT scan. The patients were treated, on a 'Stryker frame' or in bed, in removable plaster shells for two months (Fig.3.10), followed by mobilization in the orthosis during two months.

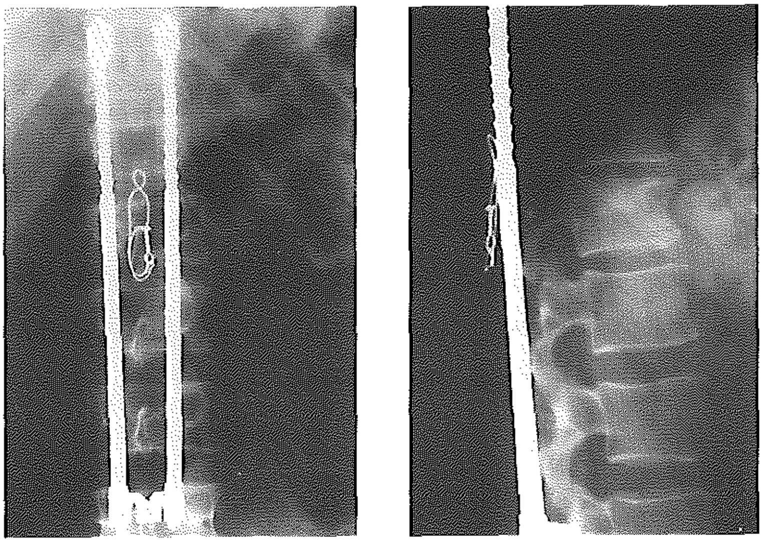

Figure 3.7 *Anteroposterior and lateral radiograph of an osteosynthesis with Harrington rods of lumbar 1 (long rod, short fusion).*

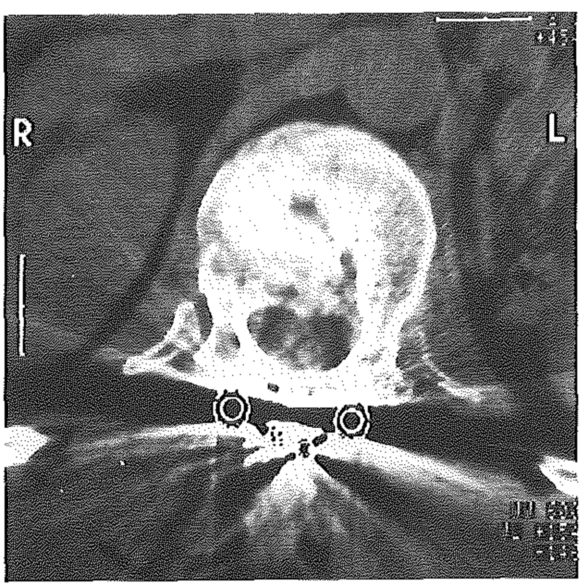

Figure 3.8 *CT scan of the same burst fracture (lumbar 1) treated with a Harrington rod osteosynthesis .*

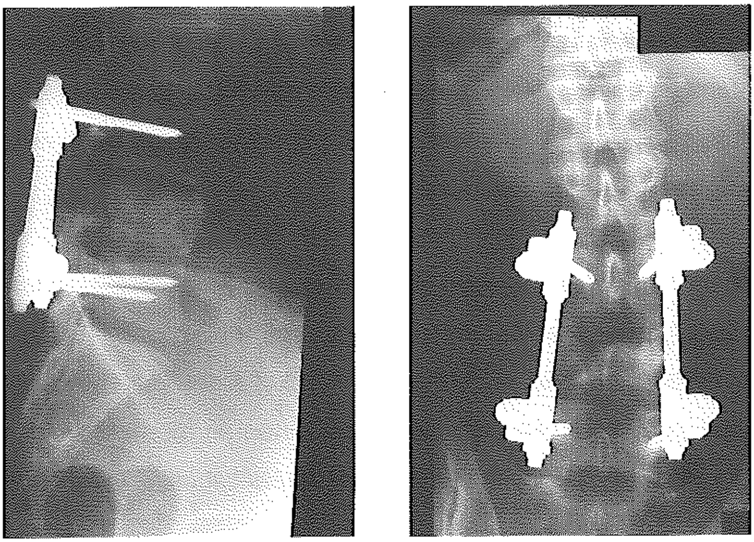

Figure 3.9 *Lateral and anteroposterior radiograph of a fixateur interne according to Dick of a burst fracture of lumbar 4.*

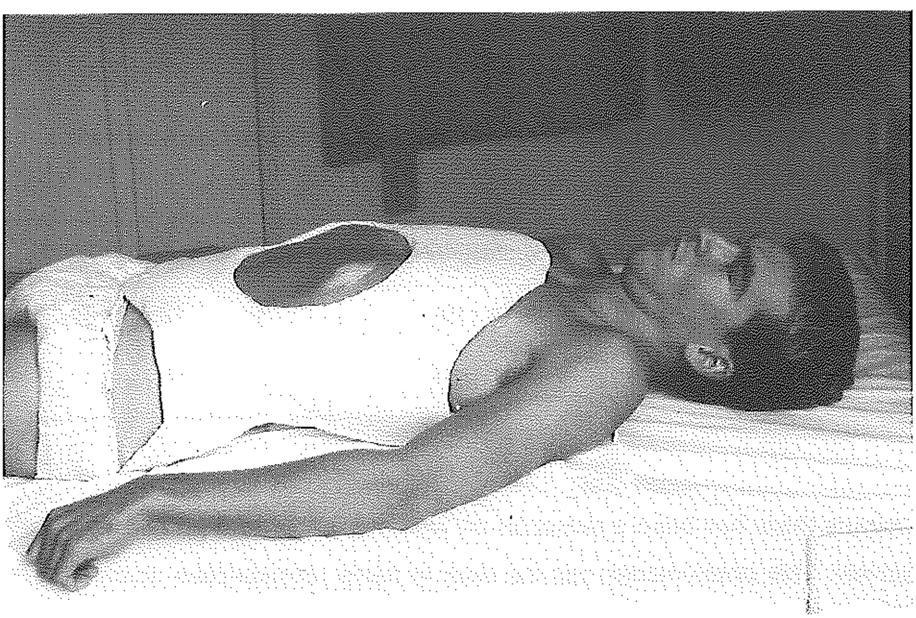

Figure 3.10 *Protection of the spine with plaster shells.*

After reconstruction of the shape of the spinal canal 28 patients were treated, either with Harrington rods (19 patients), a Dick osteosynthesis (6 patients) or with only a decompression and a laminectomy (3 patients); 73 patients were treated conservatively as previously described.

There was a significant correlation between the treatment (operative/conservative) and the presence of a neurological deficit (chi-square test; $p < 0.0001$) and the severity of the deficit classified according to Frankel (chi-square test; $p < 0.0001$). The surgical procedure was chosen more often in patients with a neurological deficit and in patients with the Frankel classification D (Table 3.4; Fig. 3.9).
There was a preponderance of women among the patients treated operatively (chi-square test; $p < 0.005$). We could not establish an explanation for this phenomenon (Table 3.3).

Table 3.3 *Number and percentage of patients treated operatively and conservatively.*

Treatment	Male (*percentage*)	Female (*percentage*)
Operative	14 (*19*)	12 (*46*)
Conservative	59 (*81*)	14 (*54*)

This means there has been a selection on whether or not to treat burst fractures operatively. This phenomenon can be seen in many retrospective studies. In the analysis of the results of this study we tried to correct for these and other differences between the operatively and conservatively treated patients.

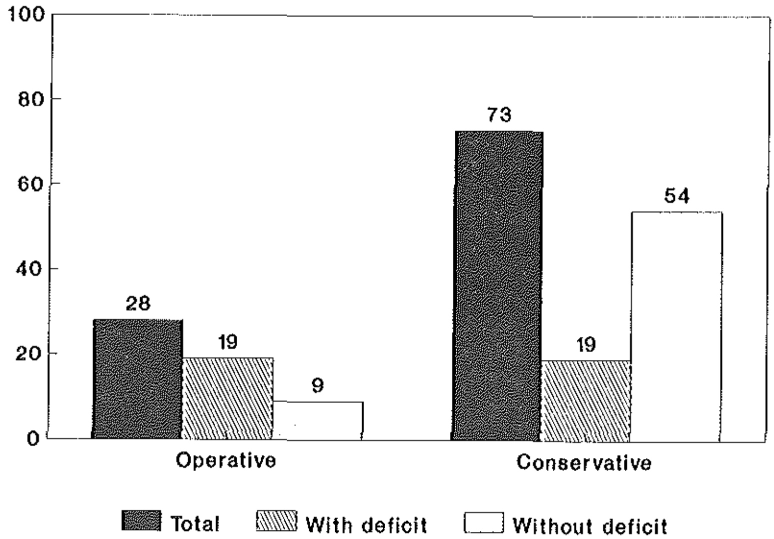

Figure 3.11 *Number of patients treated operatively and conservatively.*

Table 3.4 *Number of patients with a neurological deficit classified according to Frankel for the operative and conservative treatment.*

TREATMENT	FRANKEL CLASSIFICATION				
	A	B	C	D	E
Operative	0	2	3	14	0
Conservative	3	2	6	8	0

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Chapter IV

Predicted probability of a neurological deficit in thoracolumbar burst fractures

The principal objective of this study is:

To determine the relationship between the presence of a neurological deficit and the extent of spinal canal stenosis in thoracolumbar burst fractures.

Introduction

Our understanding of fractures of the thoracolumbar spine has improved since the introduction of computerised tomography (CT).[2,3,4,16] Denis made a practical classification of thoracolumbar spine fractures using the three column system.[6] In particular, in thoracic, thoracolumbar and lumbar burst fractures CT clearly shows a narrowing of the spinal canal by the retropulsed bony fragments of the vertebral body (Fig.4.1).[7,14]

The aim of this study was to investigate the joint correlation of the level of the burst fracture and the percentage spinal canal stenosis with the probability of an associated neurological deficit.

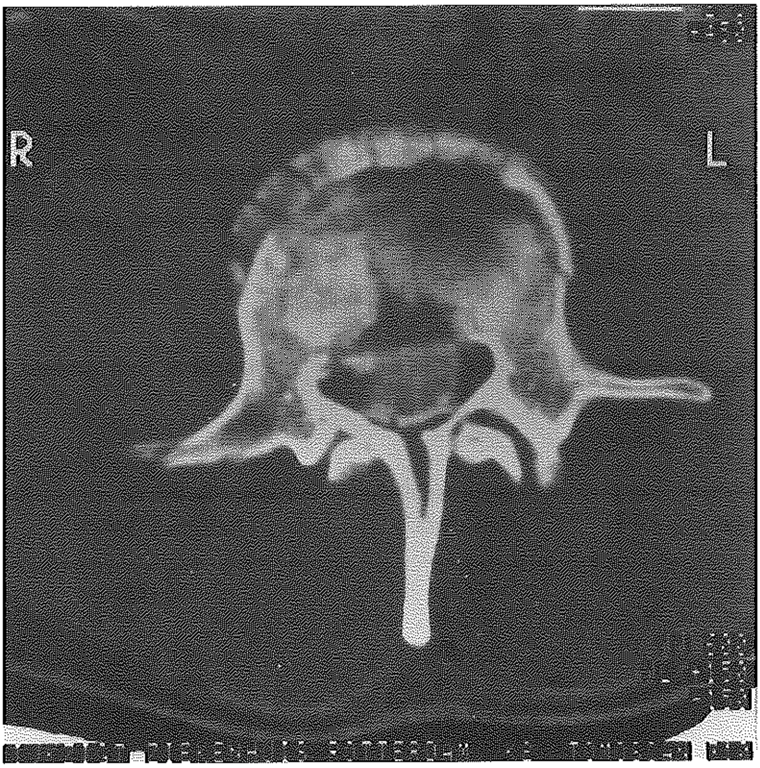

Figure 4.1 *CT scan of a burst fracture of lumbar 4 with a spinal canal stenosis of about 80 percent and with a Frankel classification D.*

Patients and Methods

Between January 1981 and January 1991 we treated 139 patients with thoracic, thoracolumbar or lumbar burst fractures (with available computerised tomographs) in the Departments of Orthopaedics or Neurosurgery of our hospital.

As stated on page 40, there were 97 men and 42 women with a mean age at time of injury of 36 years (12 to 83 years). The location of the 139 burst fractures, included in this study, with and without a neurological deficit is shown in Table 4.1. Forty-nine patients (35.3 percent) had a neurological deficit as defined by Frankel et al (page 42).[8] Anteroposterior and lateral radiographs were taken of all patients, followed within 24 hours by CT. The percentage spinal canal stenosis at the level of injury was calculated (Chapter III).

Table 4.1 *Number (percentage) of patients with a burst fracture with and without a neurological deficit at the three spinal levels.*

Spinal level	With deficit	Without deficit
Thoracic	6 (*32*)	13 (*68*)
Thoracolumbar	28 (*39*)	44 (*61*)
Lumbar	15 (*31*)	33 (*69*)
P-value	NS	NS

Statistical analysis

The chi-square test was used to determine the correlation between the location of the burst fracture (thoracic, thoracolumbar or lumbar) and the presence of a neurological deficit; and the Wilcoxon's two-sample test to determine the relation ship between the percentage spinal canal stenosis and neurological deficit.

Predicted probability of a neurological deficit

Multiple logistic regression[15] was used to determine the joint correlation between the location of the burst fracture and the percentage spinal canal stenosis with the probability of the presence of a neurological deficit.

Results

The percentage of patients with a neurological deficit did not differ between the three levels ($p > 0.65$) (Table 4.1). The presence of a neurological deficit was very significantly associated with a high percentage spinal canal stenosis, independent of the level of the burst fracture (Table 4.2). For the thoracolumbar and lumbar levels the percentage of spinal canal stenosis was significantly higher in those with a neurological deficit than those without a neurological deficit. This trend was also seen at the thoracic level, but it did not reach statistical significance, probably due to the small number of patients with a neurological deficit.

Table 4.2 *Mean percentage (SD) of spinal canal stenosis at the three spinal levels.*

Spinal level	With deficit	Without deficit	P-value
Thoracic	20 *(27)*	14 *(10)*	NS
Thoracolumbar	47 *(20)*	27 *(22)*	0.0003
Lumbar	64 *(27)*	42 *(27)*	0.01
All patients	49 *(26)*	30 *(25)*	0.0002

A multiple logistic regression model without the interaction between the level of injury and the percentage of spinal canal stenosis could describe the data adequately. The results of the model are given in Table 4.3.
The presence of a neurological deficit remains very significantly correlated with the percentage of spinal canal stenosis. Moreover, there is now a marginally significant correlation with the level of injury (the higher the level of the burst fracture, the greater the probability of a neurological deficit) (Table 4.3).

Table 4.3 *Results of the multiple logistic regression model.*

	Odds Ratio	95% confidence interval	P-value of difference
Thoracic vs lumbar level	1.73	0.78 to 3.87	0.03 *
Thoracolumbar vs lumbar level	1.22	0.72 to 2.07	NS
Percentage stenosis * *	1.036	1.02 to 1.05	0.0001

** by trend test.*

*** (constant 0.145) an estimate of the odds of neurological deficit in a patient with a lumbar burst fracture and 0 percent stenosis.*

The probability of a neurological deficit as predicted by this model is illustrated in Figure 4.2. For example, the predicted probability of a neurological deficit in burst fractures at the thoracic level with a spinal canal stenosis of 25 percent, 50 percent and 75 percent is respectively 0.38, 0.60 and 0.78. At the thoracolumbar level the predicted probability with a spinal canal stenosis of 25, 50 and 75 percent is respectively 0.29, 0.51 and 0.71. At the lumbar level it is 0.14, 0.28 and 0.48. As shown in Figure 4.2 the predicted probability of a neurological deficit in in burst fractures of the thoracic, thoracolumbar or lumbar spine is never 1.0. even with a spinal canal stenosis of 90 percent.

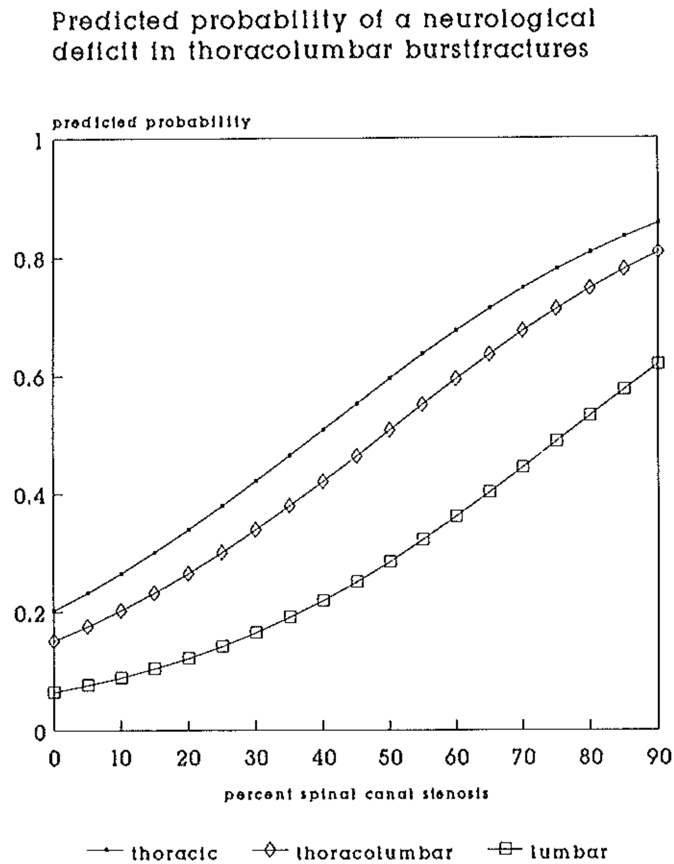

Figure 4.2 *Predicted probability of a neurological deficit in thoracic, thoracolumbar and lumbar burst fractures.*

The severity of the neurological deficit could not be predicted from the spinal canal stenosis. The differences between the mean percentages spinal canal stenosis of the five groups classified according to Frankel (A to E) did not reach statistical significance (Table 4.4).[8]

Table 4.4 *Lack of correlation between the severity of the neurological deficit, using the Frankel classification, and the mean percentage spinal canal stenosis.*

Frankel classification	Number	Mean percentage (*SD*) stenosis
A (Complete)	5	55 (*28*)
B (Sensory only)	3	55 (*12*)
C (Motor useless)	11	37 (*31*)
D (Motor useful)	29	51 (*28*)
E (Recovery)	1	39 (-)

Discussion

Some previous studies have reported no relationship between the patient's initial neurological impairment and the percentage stenosis of the spinal canal.[11,12] Lindahl et al could not demonstrate any significant correlation between the degree of narrowing of the spinal canal and the presence of symptoms of neurological deficit.[12] This lack of correlation is probably due to the small number of cases. However, they did establish a significant correlation between the degree of involvement of the spinal cross-sectional area and the mid-sagittal diameter.[14] We confirm Lindahl's finding that measuring the mid-sagittal diameter is a reliable method.

Other reports mentioned a correlation between the occurrence of a neurological deficit and the percentage spinal canal stenosis in burst fractures of the thoraco-lumbar spine, but in most of these studies no adequate statistical analysis was applied.[6,10,12] Hashimoto et al 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.[10] They concluded that a spinal canal stenosis ratio of 35 percent or more at the epiconus level (T11 to T12), 45 percent or more at the conus medullaris level (L1), and 55 percent or more at the cauda equina level (L2 to L5) to be significant factors for neurological impairment in thoracolumbar burst fractures.

In our study, we were able to predict the presence of a neurological deficit from measurements of spinal canal stenosis at the level of injury. The higher the level of the burst fracture (thoracic, thoracolumbar or lumbar) and the higher the percentage spinal canal stenosis, the greater the probability of a neurological deficit. A neurological deficit is not inevitable, however, even if there is a spinal canal stenosis of 90 percent (Fig 4.2).

Prediction of the type or severity of the neurological deficit was not possible, perhaps because of the small number of patients in the various categories of the Frankel classification; several other authors have also reported the same lack of correlation[1,5,9,12].

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Chapter V

Spontaneous remodeling of the spinal canal after conservative treatment of thoracolumbar burst fractures

The principal objective of this study is:

To investigate the phenomenon of spontaneous redevelopment of the spinal canal after conservative treatment of thoracic, thoracolumbar and lumbar burst fractures.

Introduction

Surgical removal of bony fragments from the spinal canal may more or less restore the shape of the canal after burst fractures. However, this operation does not affect the extent of recovery.[3,9,16,24,26] The natural development of the changes in the shape of the spinal canal after burst fractures needs further definition.[6,7,13,16] Therefore, this study was initiated to investigate the phenomenon of spontaneous redevelopment of the spinal canal after thoracic, thoracolumbar or lumbar burst fractures.

Patients and Methods

Between January 1981 and January 1990, 125 patients with a burst fracture of the thoracic, thoracolumbar or lumbar spine were treated in the Departments of Orthopaedics or Neurosurgery of our hospital (Chapter III, page 47).

Following admission, anteroposterior and lateral radiographs were taken of all patients suspected of having a spinal injury. Within 24 hours, a computerised tomograph (CT) was made of all patients with a fracture of the spine. The least mid-sagittal diameter of the spinal canal at the level of injury was calculated on the CT scan (Chapter III, page 45).[15] The CT scan was repeated at follow-up. In order to investigate the extent of the spinal canal remodeling, only patients with an initial percentage spinal canal stenosis of at least 25 percent were included in this study. Forty-two of the conservatively treated patients had an initial spinal canal stenosis of more than 25 percent. These patients form the population of the study of this chapter. Treatment of all patients was supervised by a neurosurgeon and an orthopaedic surgeon. Both departments used the same treatment protocol (Appendix A).

There were 35 men and 7 women with a mean age at the time of injury of 35.6 years (17 to 82 years); 16 patients (38 percent) had a neurological deficit classified according to Frankel.[14] The location of the 42 burst fractures is shown in Figure 5.1. The follow-up period ranged from 12 to 108 months (mean: 43.3).

Results

Within the first 12 months after injury the mean percentage spinal canal stenosis significantly decreased from 50 percent (SD: 15.4 percent) to 25 percent (SD:12.5 percent) (paired t-test: $p < 0.0001$). After this period there was no longer a significant correlation between the reduction in percentage spinal canal stenosis and the length of the follow-up (Fig. 5.2). The level of the fracture (thoracic, thoracolumbar and lumbar) did not significantly influence the redevelopment of the spinal canal (Table 5.1).

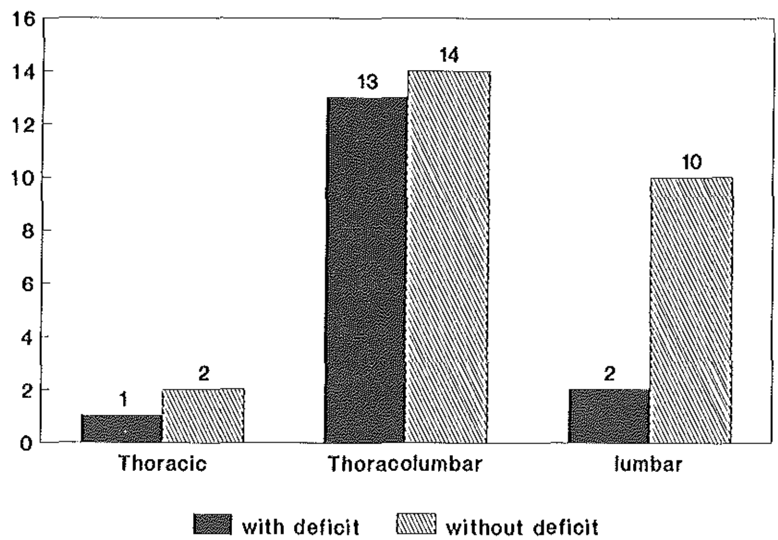

Figure 5.1 *Number of patients with and without a neurological deficit at the three spinal levels.*

Table 5.1 *Mean (SD) percentage reduction of the spinal canal stenosis at the three levels. (The reduction in percentage stenosis is the initial percentage stenosis minus the percentage stenosis at follow-up)*

Mean (*SD*) percentage reduction stenosis				P-value
Thoracic	Thoracolumbar	Lumbar	Total	
18.3 (*19.9*)	24.9 (*12.9*)	28.6 (*14.1*)	24.9 (*13.6*)	NS *

* *chi-square test*

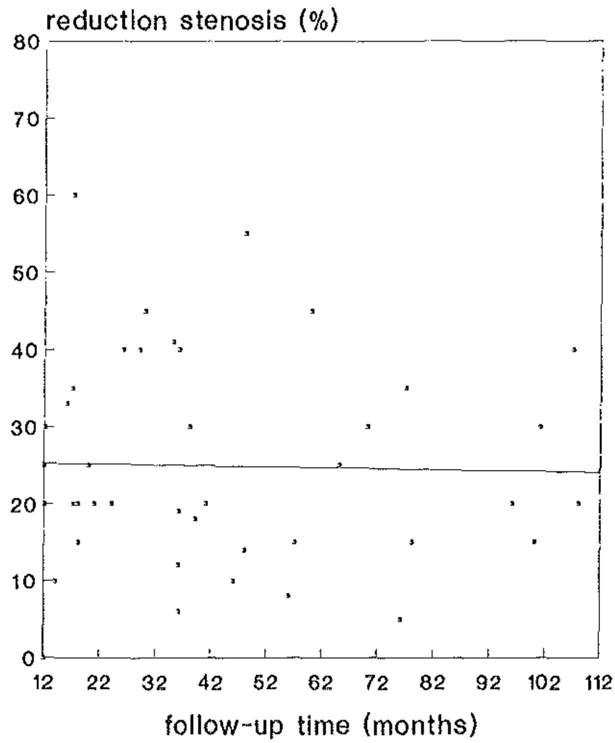

Figure 5.2 *The reduction in percentage spinal canal stenosis versus the follow-up time in months. The reduction in percentage stenosis is the initial percentage stenosis minus the percentage stenosis at follow-up.*

After a follow-up period of one year, there is no futher decrease of the spinal canal mid-sagittal diameter (spinal canal stenosis).

There was no significant difference in the reduction of canal stenosis between patients with a neurological deficit (mean (SD) reduction: 22.0 (14.8)) and patients without a neurological deficit (mean (SD) reduction: 26.7 (12.7)). Figure 5.3 shows the correlation between the reduction in percentage spinal canal stenosis and age at the time of injury ($p < 0.001$). This correlation was also seen for each separate level of injury (thoracic, thoracolumbar and lumbar).

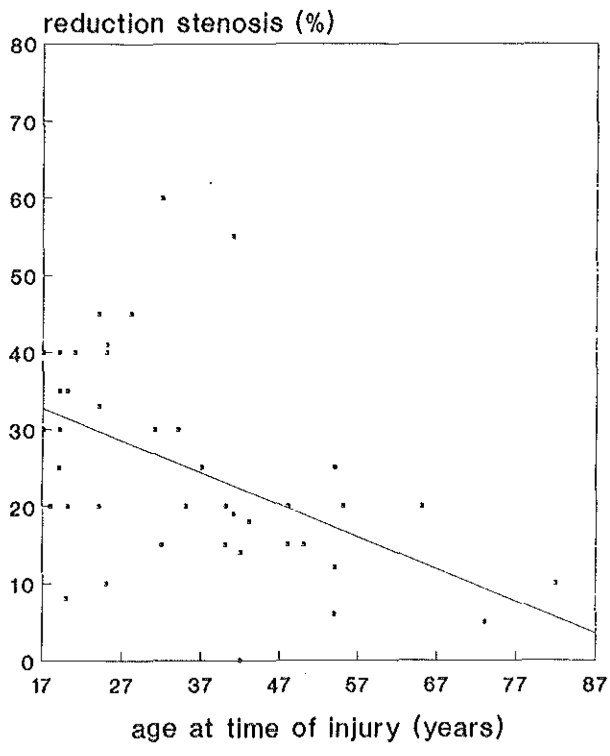

Figure 5.3 *The reduction in percentage spinal canal stenosis versus age at time of injury; the reduction in percentage spinal canal stenosis is more evident in the younger patients (at time of injury).*

None of the 42 patients was neurologically worse at follow-up. No patient had to be operated for symptoms relating to spinal canal stenosis.

The reduction in percentage stenosis significantly correlated with a higher initial percentage spinal canal stenosis ($p < 0.0001$) (Fig. 5.4).

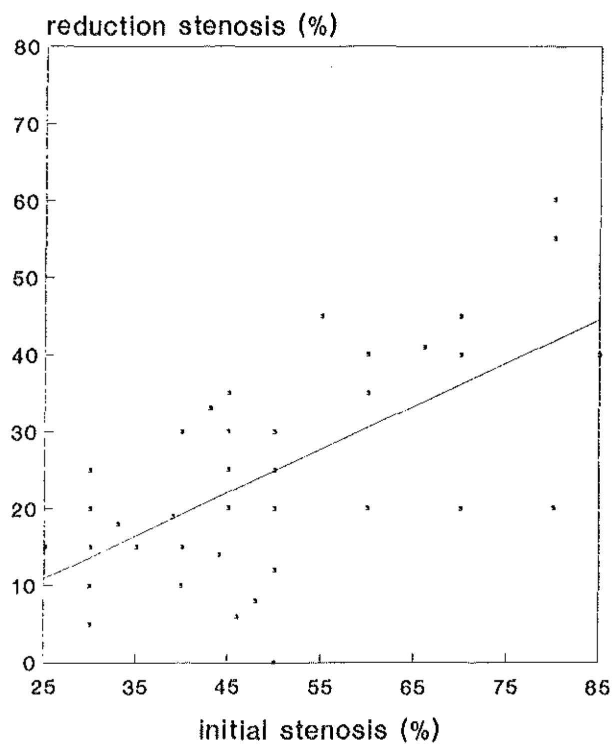

Figure 5.4 *The reduction in percentage spinal canal stenosis versus the initial percentage spinal canal stenosis; the higher the initial percentage stenosis the greater the reduction in percentage spinal canal stenosis.*

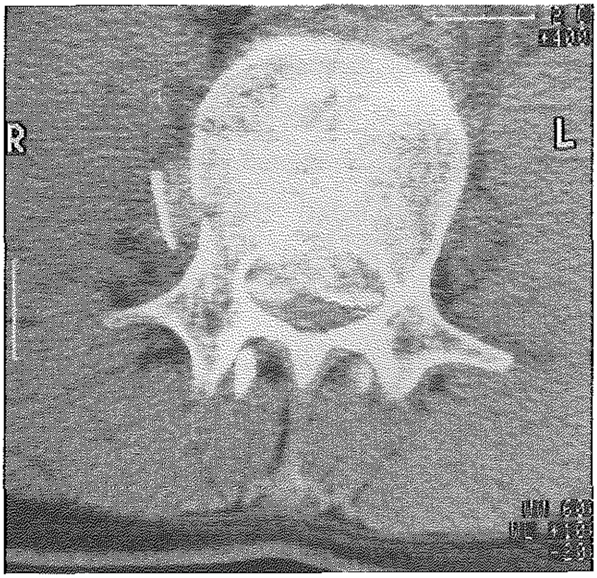

Figure 5.5 CT scan of a 33-year-old male patient with a burst fracture of lumbar 2 (6 hours after injury).

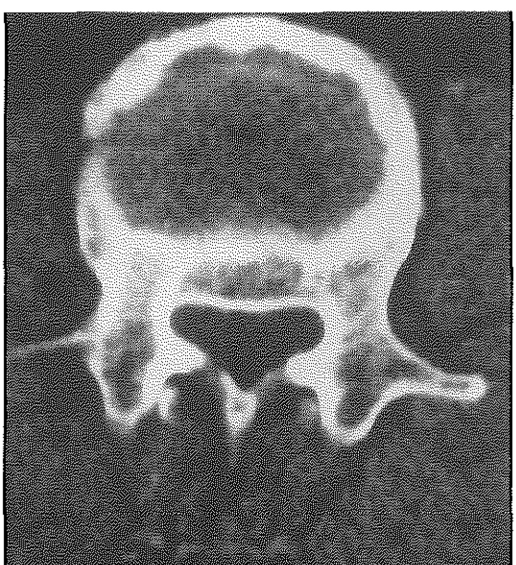

Figure 5.6 CT scan (of the same patient in Fig 5.5) 28 months after injury.

Discussion

Excessive axial 'loading' and flexion causes retropulsion of bone into the spinal canal.[10] CT-scanning has increased our knowledge of spinal trauma. However, the computed tomograms also cause apprehension by clearly showing bony fragments in the spinal canal.[4,5,8,11,19,20,21,25] It has been assumed that canal stenosis causes symptoms at a later date and, therefore, removal of all bone from the spinal canal is necessary.[1,2,10,12,17,22]

There have been reports on the redevelopment of the spinal canal in burst fractures.[6,7,13,18,23] Most of these reports concern only a small group of patients with a burst fracture without a neurological deficit and with an average follow-up of 2 years. In the reports with a small number of patients, no adequate statistical analysis could be applied. In our study it is shown that there is a significant reduction of stenosis and redevelopment of the spinal canal. The process of remodeling takes place during the first year after injury. The mechanism of the reduction in spinal canal stenosis was not influenced by the presence or degree of neurological deficit. We did not observe neurological deterioration during follow-up.

In this study, a significant spontaneous reduction in spinal canal stenosis was found in patients with a burst fracture. No late operations for spinal canal stenosis were needed. Conservative treatment of thoracolumbar burst fractures is followed by a marked degree of spontaneous redevelopment of the deformed spinal canal.

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Chapter VI

Neurological recovery in burst fractures of the thoracolumbar spine treated operatively and conservatively

The principal objective of this study is:

To review the neurological recovery in both operatively and conservatively treated patients with a burst fracture of the thoracolumbar spine.

Introduction

Many patients with burst fractures of the thoracic, thoracolumbar or lumbar spinal column show neurological deficit, due to cord, conus or cauda damage. This can be the result of the narrowing in the transverse plane of the bony spinal canal.[4,7,11,12,13,16,18] As reported in chapter IV, there is a relationship between the degree of traumatic narrowing of the spinal canal in thoracolumbar burst fractures and the presence of a neurological deficit. However, a correlation between the severity of the neurological deficit and the percentage initial spinal canal stenosis could not be established.

A few authors reported that recovery of neurological deficit does not correlate with the treatment method.[5,11,14,20,22] On the other hand, others are convinced that the bony fragments in the spinal canal should be removed in order to get a better recovery.[2,3,6,7,8,12,18]

In this study we assessed:

The amount of post-traumatic neurological improvement in relation to the type of treatment (operative or conservative), the initial percentage spinal canal stenosis, the initial kyphosis and increase in kyphosis.

Patients and Methods

Between January 1981 and January 1990, 125 patients (with available computed tomograms) with a burst fracture of the thoracic (Th1-Th10), thoracolumbar (Th11-L1) or lumbar (L2-L5) spine were treated in the Departments of Orthopaedics or Neurosurgery of our hospital (Chapter III, page 47). Forty-three patients had a neurological deficit on admission. One patient died of unrelated causes, one patient refused to cooperate and three patients were non-traceble for the follow-up study, but sufficient data were available for these three patients. Thus, 41 patients form the basis of the study of this chapter; all 41 patients were seen again in 1991 and 1992, with a follow-up period of at least 12 months.
The distribution of the level of injury and the classification of the severity of the initial neurological deficit assessed on admission using the Frankel classification are presented in Table 6.1.[9] There were 30 men and 11 women with a mean age at time of injury of 29.9 years (12 to 65 years).
Following admission, conventional radiographs were taken and within 24 hours a computed tomogram was made of all 41 patients. On the conventional radiographs the Cobb angle was measured. The involvement of the spinal canal was assessed by measuring the minimal mid-sagittal diameter of the spinal canal at the level of injury (Chapter III).

Table 6.1 *Number of patients with injury at the three spinal levels classified according to Frankel.*

Frankel classification	Level of injury			Total
	Thoracic	Thoracolumbar	Lumbar	
A (Complete)	1	4	0	5
B (Sensory only)	0	3	1	4
C (Motor useless)	3	5	1	9
D (Motor useful)	2	13	8	23
E (Recovery)	0	0	0	0
Total	6	25	10	41

Of the 41 patients, 19 were treated operatively and 22 conservatively. Between 1986 and 1990, in our hospital, most patients with a burst fracture of the thoracolumbar spine with the presence of a neurological deficit were treated conservatively. Patients who showed neurological deterioration after admission were treated operatively in this period (Chapter III, page 47). Seven patients with a neurological deficit were treated operatively in the period 1986 to 1990, because their neurological status deteriorated after admission.

Five patients with a complete cord lesion were treated conservatively, because it was thought that decompression and stabilization of the spinal canal would not improve the rehabilitation.

In the operative treatment the posterolateral approach was used to decompress the dural sac by removal of the loose fragments and tamping these into the vertebral body. Subsequently, in 17 patients instrumental reduction and stabilization was achieved either by Harrington rods (14 cases) or with a fixateur interne according to Dick (3 cases).

The mean follow-up time was 27.7 months (12 to 101 months). The neurological state, at the follow-up, was again classified according to the Frankel scale.
In this study, only improvement of one or more Frankel grades was considered as improvement of the neurological deficit. Improvement within one Frankel grade was disregarded in this study.
The Cobb angle was measured on conventional radiographs taken of all patients at follow-up.

Results

Twenty-six (63 percent) of the 41 patients improved one or more Frankel grade. Patients with an initial classification D more often showed improvement than those with classifications A to C (Table 6.2).

Table 6.2 *All patients (operatively and conservatively treated) (n = 41) classified according to Frankel on admission and at follow-up.*

Initial Frankel classification	Frankel classification at follow-up				
	A n=4	B n=2	C n=4	D n=12	E n=19
A (n=5)	4	1			
B (n=4)		1		3	
C (n=9)			4	3	2
D (n=23)				6	17
E (n=0)					

No correlation was established between the improvement of one or more Frankel grade(s) and the initial percentage spinal canal stenosis or the initial kyphosis. No difference was found in occurrence of improvement was found between the different locations of the fracture (thoracic, thoracolumbar or lumbar).

There was no significant correlation between improvement of the neurological deficit and the presence of an initial kyphosis of more than 15 degrees Cobb (Table 6.3).[5]

No relationship could be established between the improvement of the neurological deficit and the age at time of injury.

Table 6.3 *Relationship between the improvement of the neurological deficit and:*

1. *Level of injury.*
2. *Mean initial percentage spinal canal stenosis.*
3. *Mean initial kyphosis (degrees Cobb).*
4. *Number of patients with an initial kyphosis of more than 15 degrees.*

Neurologicaly improved	Level of injury			Mean (SD) initial kyphosis	Mean (SD) initial stenosis	Initial kyphosis $> 15^0$
	Thoracic	Thoraco lumbar	Lumbar			
YES	5	15	6	17.4 (10)	44.6 (25)	12
NO	1	10	4	17.0 (7)	58.0 (20)	10
P-value	NS*			NS**	NS**	NS*

* *Chi-Square test*

** *t-test*

Operative vs Conservative treatment

There was a significant difference in distribution of patients over the four initial Frankel grades (A to D) between operatively and conservatively treated patients ($p < 0.03$, chi-square test). Patients with an initial Frankel classification D were more frequently treated operatively.

For the initial percentage spinal canal stenosis and the initial kyphosis (Cobb angle) there was no significant difference between the operatively and conservatively treated patients (t-test) (Table 6.4).

Table 6.4 *Mean initial stenosis and kyphosis for both the operatively and conservatively treated patients with a neurological deficit.*

	Treatment		Total group
	Operative	Conservative	
Mean (SD) percentage initial stenosis	53.7 (25.1)	45.9 (22.2)	49.5 (23.7)
Mean (SD) initial kyphosis	18.7 (10.6)	16.1 (7.8)	17.3 (9.3)

Of the 19 operatively treated patients, 10 patients (53 percent) neurologically improved on the Frankel scale. Of the 22 conservative treated patients 16 (73 percent) neurologically improved ($p < 0.03$; chi-square test).

None of the patients was neurologically worse at follow-up.

We used multiple logistic regression to determine the correlation between the improvement of the neurological deficit classified according to Frankel and treatment method (operative vs conservative). In the regression model we corrected for the differences in the initial distribution on the Frankel scale between operatively and conservatively treated patients. It appeared that conservatively treated patients more often showed neurological improvement than operatively treated patients ($p < 0.02$). This difference was also seen for each separate Frankel grade.

Tables 6.5 and 6.6 show the improvement, according to the Frankel scale, of the operatively and conservatively treated patients, respectively.

Table 6.5 *Operatively treated patients (n = 19) classified according to the Frankel scale on admission and at follow-up.*

Initial Frankel classification	Frankel classification at follow-up				
	A n=0	B n=1	C n=3	D n=6	E n=9
A (n=0)					
B (n=2)		1		1	
C (n=3)			3		
D (n=14)				5	9
E (n=0)					

Table 6.6 *Conservatively treated patients (n=22) classified according to the Frankel scale on admission and at follow-up.*

Initial Frankel classification	Frankel classification at follow-up				
	A n=4	B n=1	C n=1	D n=6	E n=10
A (n=4)	4	1			
B (n=2)				2	
C (n=6)			1	3	2
D (n=9)				1	8
E (n=0)					

When we additionally included in the multiple logistic regression model a correction for the differences between the operatively and conservatively treated patients on the initial percentage spinal canal stenosis and the initial kyphosis, the conservatively treated patients still showed significantly more neurological improvement ($p < 0.04$).

The mean increase in kyphosis (kyphosis at follow-up minus the initial kyphosis) in degrees Cobb of the conservatively treated patients was 4.3 degrees Cobb (SD: 6 degrees Cobb). The mean increase in kyphosis of the conservatively patients who neurologically improved was 3.56 degrees Cobb (SD: 6 degrees) did not significantly differ from the conservatively treated patients who did not neurologically improve (mean: 7.5 degrees; SD: 6 degrees Cobb) (t-test).

Discussion

Many authors have reported that neurological recovery does not correlate with the treatment method or with the percentage spinal canal stenosis.[5,11,14,20,22]
Frankel, Guttmann and others report that conservative treatment usually results in neurological improvement.[1,9,10,16,20,21] Their conservatively treated patients showed the same degree of improvement as those who underwent surgical decompression and stabilization.
On the other hand Bohlman, Denis and others are convinced that persisting compression of neural tissue should be operatively removed.[2,3,6,7,8,12,18] They consider the level of improvement after operative decompression to be superior to the recovery reported after conservative treatment. According to them, the cord compression should be removed to enhance neurological recovery even in patients with a very low percentage spinal canal stenosis. Many of those favoring operative treatment refer to reports in which a significant and prompt neurological improvement is described after decompression.[15,17,19]
Dall and Stauffer reported in 1988 that burst fractures with an initial kyphosis of more than 15 degrees (Cobb) have a better prognosis in terms of neurological improvement than burst fractures with a mild initial kyphosis, probably due the transmission of less force compared to the burst fractures with a mild kyphosis.[5]

In our series we could not find a correlation between the improvement of the neurological deficit and the initial kyphosis, the increase in kyphosis or the initial percentage spinal canal stenosis.
We do not agree with authors who state that operative treatment results in a better neurological improvement. In contrast, in our series the conservatively treated patients showed significantly more often neurological improvement than

those treated operatively. Even after correction for differences in the initial percentage spinal canal stenosis, the initial kyphosis and the initial distribution on the Frankel scale, this finding still existed. In this retrospective study there has obviously been a selection whether or not to operate a patient with a burst fracture with a neurological deficit. Therefore, a small possibility exists that the operatively treated patients would show a poorer outcome if treated conservatively.

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Chapter VII

Outcome of the operative and conservative management of thoracolumbar burst fractures

The principal objective of this study is:

To compare the outcome in burst fractures treated operatively and conservatively in terms of radiographic findings, social functioning and work status.

Introduction

Over 150 years controversy has persisited regarding the management of spinal fractures.[1,6,10,14,17,19,22,25,26,28,30,31] Watson-Jones stated in 1944 that a perfect recovery is only possible if there is a perfect reduction.[36] On the other hand Nicoll reported in 1949 that a good functional result does not depend on a good anatomical result.[34] In general, the conservative management of thoracolumbar burst fractures is based on the hypothesis of Nicoll.

A series of reports described a good overall outcome in conservatively managed patients with a burst fracture of the thoracolumbar spine, but most reports concern only patients without a neurological deficit.[3,4,5,8,12,24,33,35,37] Other authors reported a better outcome in operatively managed patients.[13,14,15,16,18,27,28,29,39,40]

The purpose of the study of this chapter was to review the outcome of the operative and conservative management in patients with a burst fracture of the thoracic, thoracolumbar or lumbar spine.

Patients and Methods

The 101 patients included in this study are described in Chapter III (page 47). Thirty-eight patients had a neurological deficit as defined by Frankel. Anteroposterior radiographs were taken of all patients followed within 24 hours by computed tomography. The Cobb angle was measured on the conventional radiographs and the percentage spinal canal stenosis was calculated at the level of injury.[23]

The work status at follow-up was recorded in three different categories:

1. *Not able to work, due to back complaints*
2. *Able to work, but at a lower activity level*
3. *Able to work at previous or higher activity level*

Pain intensity due to back complaints was record at the follow-up on a four-point scale:

1 = *Unbearable*
2 = *Severe*
3 = *Moderate*
4 = *No pain*

Results

The cause of the trauma significantly correlated with: gender, and the mean height of the fall (Chapter III).

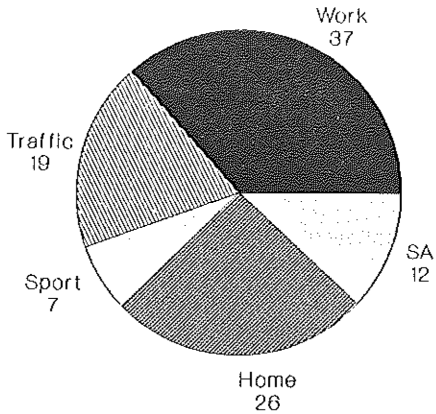

Figure 7.1 *Number of patients according to the five different causes of injury.*

Thirty-three burst fractures resulted from a direct trauma. Sixty-eight burst fractures resulted from a fall of more than 1 meter; mean 6 meters (range 1-15). The trauma caused by a fall of more than 1 meter did not correlate with the initial stenosis, the location of the fracture or with the increase in the Cobb angle in the conservatively treated patients.

Twenty-eight patients were treated operatively and 73 patients were treated conservatively. There was a significant correlation between the treatment method (operative or conservative) and the presence of neurological deficit (chi-square test; $P < 0.0001$) and with the degree of the neurological deficit classified according to Frankel (chi-square test; $P < 0.0001$). The operative treatment was chosen more often in patients with a neurological deficit and with an initial Frankel grade D.

The mean initial percentage spinal canal stenosis was significantly higher in the operatively treated patients (t-test; $P < 0.0001$), but no significant difference was found between the operatively and conservatively treated patients for the initial kyphosis.

Conservative treatment

There was a significant increase in the Cobb angle (kyphosis) mean: 5.5 degrees (SD: 6.1 degrees) (paired t-test; $p < 0.0001$). The increase in kyphosis did not correlate with the level of injury (Table 7.1). We found no correlation between the increase in the Cobb angle and the follow-up time (Fig.7.2). There was no futher increase in the Cobb angle after a period of 12 months after trauma.

Table 7.1 *The mean initial and increase in kyphosis (SD) for the three levels of injury in conservatively treated patients.*

	Level of injury			P-value
	Thoracic n = 13	Thoraco-Lumbar n = 40	Lumbar n = 20	
Mean initial kyphosis	22.1 (18.5)	12.3 (7.0)	11.1 (9.0)	0.000*
Mean increase kyphosis	5.8 (5.3)	5.7 (5.8)	4.9 (7.3)	NS*

* *chi-square test*

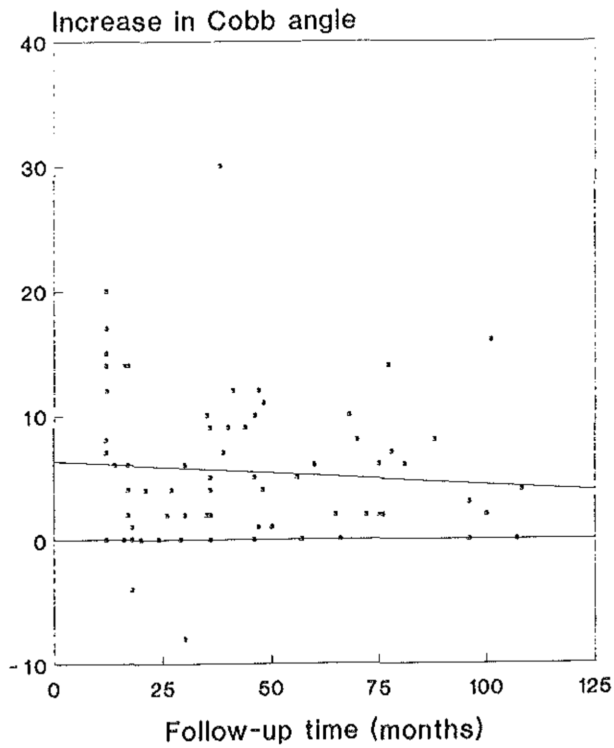

Figure 7.2 *Increase in kyphosis (Cobb angle) in relation to follow-up time in months. The increase in kyphosis is the Cobb angle at follow-up minus the initial Cobb angle. The increase in kyphosis must have been completed within the first twelve months after injury.*

The increase in the Cobb angle did not correlate with the age at time of injury, the presence of a neurological deficit or gender. We found a positive correlation between the increase in kyphosis and the initial percentage spinal canal stenosis (multiple logistic regression; $p < 0.005$) (Fig.7.3).

There was a significant reduction in percentage spinal canal stenosis mean: 21 percent (SD: 15.5 percent) (paired t-test; $p < 0.000$). As in the series described in chapter V; no correlation was found between the reduction in percentage spinal canal stenosis and follow-up time, which is in agreement with our earlier finding that the resorption of the intraspinal bone (as the increase in kyphosis) must have been completed within the first 12 months after injury.

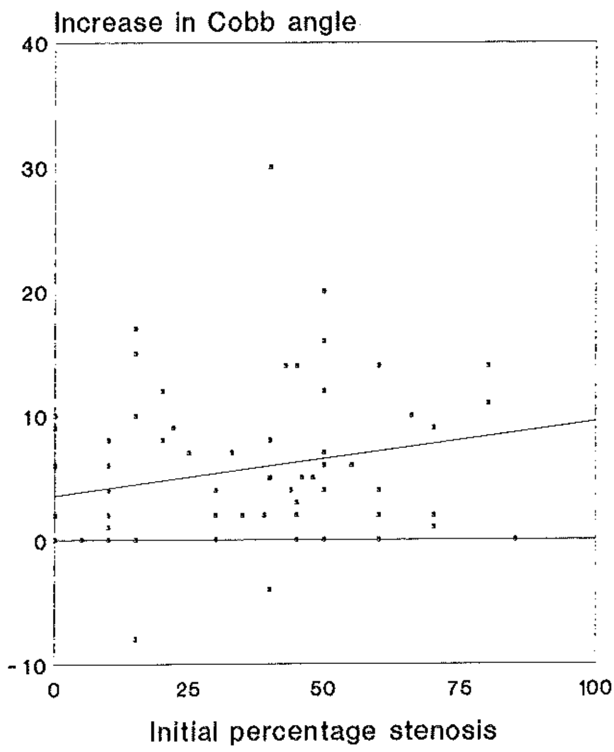

Figure 7.3 *The relationship between the increase in kyphosis (degrees Cobb) and the initial percentage spinal canal stenosis.*

In this series, a relationship was found between the reduction in percentage stenosis and the level of injury, probably due to the larger group of patients compared to the group of patients decribed in chapter V (chi-square test; $p < 0.01$) (Table 7.2).

Table 7.2 *The mean initial and the mean reduction percentage stenosis (SD) at the three levels of injury in conservatively treated patients.*

	Level of injury			P-value
	Thoracic	Thoraco-Lumbar	Lumbar	
Mean stenosis	15.9 (18.5)	36.8 (22.7)	34.7 (25.2)	NS
Mean reduction stenosis	6.4 (14.9)	22.0 (14.1)	26.8 (15.0)	0.01

As with the results described in chapter V, the reduction in percentage spinal canal stenosis correlated with the age at time of injury.

Operative treatment

Nineteen patients were treated with Harrington rods, 6 patients with a fixateur interne according to Dick and 3 patients had only decompression with a laminectomy.

The mean initial kyphosis was 16.6 degrees (SD: 10.7) and correlated with the level of injury (thoracic, thoracolumbar and lumbar) (chi-square test; $p < 0.01$) (Table 7.3). In the operatively treated patients the Cobb angle decreased significantly; the mean decrease was 2.9 degrees (SD:10.6) (paired t-test; $p < 0.001$). The decrease in kyphosis did not correlate with the level of injury. The mean initial percentage spinal canal stenosis was 55.7 percent (SD: 27.7), but no relationship could be found with the level of injury, probably due to the small number of thoracic burst fractures (n= 2). The reduction in percentage spinal canal stenosis is significantly greater in the operatively treated patients than in the conservatively treated patients (t-test; $p < 0.001$).

Table 7.3 *Mean (SD) initial and reduction in percentage spinal canal stenosis and mean (SD) initial and decrease in kyphosis (degrees Cobb) for the three levels of injury in the operatively treated patients.*

	Level of injury			P value
	Thoracic n= 2	Thoraco-Lumbar n= 13	Lumbar n= 13	
Mean initial stenosis	21.5 (16.3)	51.2 (27.1)	65.5 (25.5)	NS*
Mean reduction stenosis	11.5 (16.2)	43.1 (24.0)	49.5 (23.9)	NS*
Mean initial kyphosis	36.5 (2.1)	15.8 (7.6)	14.4 (11.4)	0.01*
Mean decrease kyphosis	13.5 (17.7)	2.1 (10.0)	1.9 (10.5)	NS*

* *chi-square test*

Work status at follow-up

In order to get an indication about the work status we interviewed all patients at follow-up. Ninety-four patients worked before the trauma; the work status at follow-up included these 94 patients. Twenty-two patients with a neurologic deficit and 12 patients without a neurologic deficit were unable to work at the time of follow-up due to back complaints. Forty-six patients returned to the previous or higher level of actvity and 14 patients to a lower activity level. No differences were found between the operatively and conservatively treated patients (chi-square test; $p < 0.5$) (Table 7.4).

Table 7.4 *Work status at follow-up for patients with and without a neurological deficit.*

	Treatment	Work status at follow-up			P-value
		Not able to work	Lower activity level	Previous activity level	
With deficit	operative	7	5	3	NS*
	conservative	15	2	1	
Without deficit	operative	3	1	6	NS*
	conservative	9	6	36	
All patients	operative	10	6	9	NS*
	conservative	24	8	37	

* *chi-square test*

No relationship was established between the work status at follow-up and gender. The initial percentage spinal canal stenosis correlated well with the work status at follow-up; even when corrected for the presence of a neurological deficit the correlation was still significant. Burst fractures as a result of a fall (more than 1 meter) correlated with the work status at follow-up (Table 7.5).

Table 7.5 *Relationship between the work status at follow-up and height of the fall (meters) and the mean initial percentage spinal canal stenosis.*

	Work status at follow-up			P-value
	Not able to work	Lower activity level	Previous acitivity level	
Mean (SD) height of the fall	7.5 (3.8)	4.7 (3.4)	5.1 (3.4)	0.03*
Mean (SD) initial percentage stenosis	44.7 (27.4)	45.2 (21.3)	31.6 (26.4)	0.05*

* *chi-square test*

Of the 11 patients with a neurological deficit the mean duration from injury to re-employment was 10.2 months (SD: 2.6 months). Of the 49 patients without a neurological deficit the mean duration from injury to re-employment was 7.8 months (SD: 2.9 months) which significantly differed from the group of patients with a neurologic deficit (t-test; $p< 0.01$). No significant correlation was found between the operatively and conservatively treated patients. Neither was there a correlation between the time from injury to re-employment and gender, the location of the burst fracture, the initial percentage spinal canal stenosis, or the burst fracture resulting from a fall from a height of more than 1 meter.

The pain intensity caused by the back complaints was recorded on a four-point scale (1 = unbearable pain to 4 = no pain). The mean pain score for all patients was 3.3 (SD: 0.78). We could not establish a significant difference in the pain intensity between the operative and conservative treated patients (Table 7.6). Neither could we find a relationship between the pain score (intensity) and gender or location of the fracture.

Table 7.6 *Mean pain score for the operatively and conservatively treated patients with and without a neurological deficit.*

	Treatment	Mean Pain Score (SD)	P-value
With a neurological deficit	Operative	2.78 (0.87)	NS*
	Conservative	2.82 (0.72)	
Without a neurological deficit	Operative	3.50 (0.62)	NS*
	Conservative	3.63 (0.56)	
All patients	Operative	3.00 (0.85)	NS*
	Conservative	3.40 (0.73)	

* *t-test*

As shown in Table 7.6, all groups of patients with a burst fracture have a low degree of pain at follow-up.

Discussion

Several authors described an increase in the Cobb angle of 6 to 10 degrees and an anterior body collapse of about 6 percent, both within the first 12 months after injury, which suggest stabilization of the deformity after one year.[1,29,33,38,40] In our series, the increase in the Cobb angle was 5.5 degrees and also did not correlate with the follow-up time. The process of remodeling is described in several reports.[7,9,20,33] The phenomenon of spontaneous resolution of bony fragments is completed within the first year after the trauma. Due to a larger series compared to chapter V, the remodeling of the spinal canal correlated with the level of injury. None of the patients was neurologically worse at follow-up. Radiographic reviewing of the outcome of the treatment of burst fractures of the thoracic, thoracolumbar and lumbar spine can be done after a follow-up time of at least one year.

The pattern of pain intensity after conservative treatment is relatively consistent in the literature; all reported some low degree of pain in conservatively treated patients.[2,33,34,37] We agree with these reports, but in our study a low degree of pain was also seen in operatively treated patients. Moreover, in our series there was no difference regarding pain intensity at follow-up between operatively and conservatively treated patients; even after correction for the presence of a neurological deficit no relationship could be found.

In our series, no difference was found in the work status at follow-up between operatively and conservatively treated burst fractures. As expected, patients with a neurological deficit differed from those without a neurological deficit regarding work status at follow-up. More patients without a neurological deficit were able to return to work.

We could find no difference in the time between injury and return to work between the operatively and conservatively treated patients. Although operatively treated patients can be mobilized earlier, they need the same amount of time to recover from the injury.

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Chapter VIII

General discussion and summary

General discussion and summary

The studies in this thesis are focused on two main themes:

> **Investigation of the course of the conservative treatment of burst fractures of the thoracic and lumbar spine; and comparison of the outcome of the operative and conservative management.**

As indicated in the introduction (Chapter I), for many decades controversy persists regarding the management of thoracolumbar burst fractures. Several empiric criteria, such as the percentage spinal canal stenosis or the Cobb angle (kyphosis) have been used as indicators for operative treatment, regardless of the presence of a neurological deficit.

In Chapter IV the ability to predict the probability of a neurological deficit is discussed. There is a positive correlation between the presence of a neurological deficit and percentage spinal canal stenosis, but even at the thoracic level the predicted probability of a neurologic deficit will never be 1. No correlation could be found between initial percentage spinal canal stenosis and the severity of the neurological deficit, which suggests that percentage spinal canal stenosis does not reflect the degree of impact on the spinal cord at the moment of injury.

Remodeling and reconstitution of the spinal canal in burst fractures takes place within the first 12 months after injury (Chapter V). The mean percentage spinal canal stenosis decreased from 50 to 25 percent at follow-up. These natural changes of the spinal canal were not influenced by the presence of a neurological deficit. None of the conservatively treated patients showed neurological deterioration. Conservative treatment of thoracolumbar burst fractures is followed by a marked degree of spontaneous redevelopment of the deformed spinal canal, regardless of the presence or severity of a neurological deficit.

Surgical removal of bony fragments in thoracolumbar burst fractures may more or less restore the shape of the spinal canal, but the operative treatment does not affect the extent of the neurological recovery (Chapter VI). Moreover, in our study, the conservatively treated patients showed a higher level of neurological improvement compared to the operatively treated patients.

The pattern of pain intensity after conservative treatment is relatively consistent in the literature (Chapter VII). All reported some low degree of pain in conservatively treated patients. We agree with these authors, but in our study a low degree of pain was also seen in operatively treated patients. Moreover, in our series, there was no difference regarding pain intensity at follow-up between operatively and conservatively treated patients. As expected, patients with a neurological deficit differed from those without a neurological deficit regarding work status at follow-up; more patients without a neurological deficit were able to return to work. No difference was found between the operatively and conservatively treated patients for the work status at follow-up. So, the treatment method did not influence the work status at follow-up. We found no difference in the time from injury to return to work between the operatively and conservatively treated patients. It appears that although operatively treated patients can be mobilized earlier, they need the same amount of time to recover from the injury.

Recent reports mention good results following conservative management of patients with a burst fracture of the thoracolumbar spine without a neurological deficit. We agree with these results; moreover, in many instances conservative treatment may be a good option in patients with a burst fracture and neurological deficit.

In patients with neurological deterioration, in the first days after injury, we consider operative treatment to be the method of choice. This emphasizes the importance of a thorough neurological examination at the first admission.
Although with conservative management it may take longer before the patient can be verticalized, particularly in case of thoracolumbar and lumbar burst fractures, the end result will usually be a stable spine and, as shown in Chapter VII, the time from injury to re-employment (recovery time) is the same as in operatively treated patients.

We are aware of the limitations of this retrospective un-controlled study; therefore, this study should be considered as a preliminary study. Until the results of a prospective randomized study are known, the conclusions of this study are of importance to those who have to decide whether to operate patients with a burst fracture of the thoracolumbar spine or to treat them conservatively .

Discussie en samenvatting

Discussie en samenvatting

Bij de onderzoeken die in dit proefschrift zijn beschreven, hebben twee problemen centraal gestaan:

> In de eerste plaats werden onderzoeken verricht die nader inzicht trachten te verwerven in het beloop van de conservatieve behandeling van thoracolumbale 'burst' fracturen. Ten tweede werd dit beloop vergeleken met de operatieve behandeling van de thoracolumbale 'burst' fractuur.

Zoals aangegeven in de introductie (Hoofdstuk I) bestaat er nog steeds geen consensus betreffende de behandeling van thoracolumbale 'burst' fracturen. Verscheidende, voornamelijk empirische, criteria zoals het percentage kanaal stenose of de locale kyphose zijn beschreven in de literatuur als indicaties voor de operatieve behandeling ongeacht de neurologische status.

In Hoofdstuk IV konden we aan de hand van het percentage spinaal kanaal stenose de kans op neurologische uitval berekenen. Het bleek dat naarmate de fractuur hoger was gelokaliseerd de kans op neurologische uitval groter werd.

Zelfs bij een spinaal kanaal stenose van 90 procent zal de kans op uitval nooit 1 zijn. De ernst van het neurologische letsel was echter niet te voorspellen, hetgeen suggereert dat het percentage stenose niet correleerd met de 'impact' op het myelum.

'Remodeling' van het spinale kanaal na een 'burst' fractuur vindt plaats gedurende de eerste twaalf maanden na het ongeval (**Hoofdstuk V**). De gemiddelde stenose neemt dan af van 50 naar 25 procent en wordt niet beïnvloed door de aanwezigheid van neurologisch letsel. Er werd bij geen enkele patiënt neurologische achteruitgang gezien. De conservatieve behandeling gaat dus gepaard met een spontane resorptie van bot uit en herstel van de mid-sagittale diameter van het wervelkanaal.

Chirugische verwijdering van fragmenten uit het wervelkanaal bij thoracolumbale 'burst' fracturen zal de mid-sagittale diameter van het kanaal min of meer herstellen, echter deze behandeling zorgt niet voor een sterkere neurologische verbetering (**Hoofdstuk VI**). In onze studie toonden de conservatief behandelde patiënten vaker een neurologische verbetering vergeleken met de operatief behandelde patiënten.

De literatuur beschrijft het voorkomen van een lichte graad van pijn volgend op de conservatieve therapie van 'burst' fracturen. In onze studie bleek echter dezelfde graad van pijn aanwezig te zijn bij de operatief behandelde patiënten (**Hoofdstuk VII**). Het al dan niet kunnen verrichten van arbeid na een 'burst' fractuur werd niet beïnvloed door de behandelings methode (operatief vs conservatief), echter wel door de aanwezigheid van neurologisch letsel. Hetzelde gold voor de duur van de arbeidsongeschiktheid. Hetgeen impliceert dat de operatief behandelde patiënten ondanks een vluggere mobilisatie een evenlange herstel periode nodig hadden als de conservatief behandelde patiënten.

In de recente literatuur worden goede resultaten beschreven van de conservatieve behandeling van patiënten met een thoracolumbale 'burst'fractuur zonder neurologisch letsel. Uit onze studie blijkt echter ook dat in vele gevallen de conservatieve therapie een goede optie is bij patiënten met neurologisch letsel.

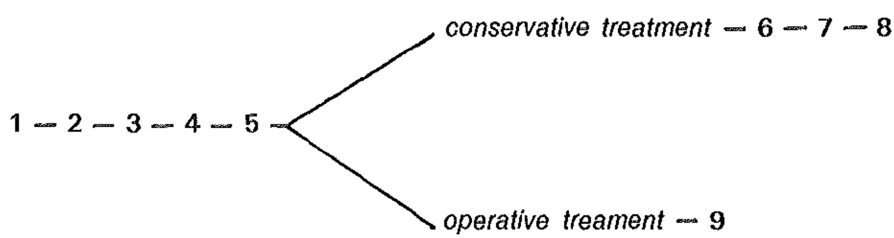
Indien er, aansluitend aan het ongeval, neurologische achteruitgang wordt geconstateerd zal ons inziens de operatieve behandeling de voorkeur genieten. Hiermee wordt het belang van een goed neurologisch onderzoek bij opname onderstreept.
Ondanks een vertraagde mobilisatie van de patiënt zal de conservatieve therapie resulteren in een stabiele wervelkolom, en vergeleken met de operatieve behandeling resulteert de conservatieve behandeling niet in een vertraging van de hersteltijd (o.a. met betrekking tot werkhervatting).

Waar de resultaten van de operatieve en conservatieve behandeling worden vergeleken zijn wij ons terdege bewust van de beperkingen van deze retrospectieve studie. Onze conclusies dienen daarom gezien te worden als voorlopig. Echter totdat de resultaten bekend zijn van een prospectieve gerandomizeerde studie, waarin bij de behandelingsmethoden met elkaar vergeleken worden, zijn de resultaten van deze studie van belang voor hen die moeten beslissen over de behandeling van patiënten met een 'burst'fractuur.

Appendix A

Treatment protocol of patients with a thoracolumbar burst fracture

Algorithum of the treatment protocol



1. Following admittance, anteroposterior and lateral radiographs of the thoracic and lumbar spinal column are taken of all patients suspected of having a spinal injury.

2. All patients with a fracture of the spinal column will have a neurological examination by a neurosurgeon.

3. The spine of a patient with a burst fractures of the thoracic and lumbar spine is stabalized with plaster shells, in the emergency room, before the patient is subjected to computed tomography.

4. The neurosurgeon and the orthopaedic surgeon decide together whether or not to perform a surgical decompression and stabilization.

5. At admission the patient will receive 500 ml dextran. Sintrom is given during a 3-month period in order to prevent thrombosis.

6. The conservative treatment of burst fractures includes stabilization on a 'Stryker frame' in removable plaster shells.

7. After clinical stabilization a circular plaster shell, from the symphisis to the sterno-clavicular joints, will be made.

8. Two months after the injury the patient will, if neurologically possible, be mobilized in the plaster shells. Four months after trauma, the shells are removed and radiographs taken to see the position of the fracture and to see whether the fracture has united.

9. In the operative treatment the dural sac will be decompressed and reduction and stabilization will be achieved by osteosynthesis. The patient will be mobilized as soon as possible, depending on the stability of the spine (sometimes in a plaster shell).

In the early 1980s the Department of Orthopaedics of our hospital favored a more agressive treatment of burst fractures than the Department of Neurosurgery. Also within the departments, there was no consensus on whether or not to operate a thoracolumbar burst fracture. Therefore, the method of treatment (operative or conservative) depended largely on the personal preferences of the neurosurgeon and orthopaedic surgeon on duty. Obviously this means an uncontrolled selection of treatment methods in this retrospective study.

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Een proefschrift schrijf je alleen, maar niet zonder steun !.

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Lieve Ans, je bent voor mij van onschatbare waarde. Zonder jouw steun en vriendschap had dit niet zo voorspoedig kunnen verlopen.

Curriculum Vitae

De auteur van dit proefschrift werd geboren op 5 november 1964 te Best. Na het volgen van het VWO aan het Thomas More College te Oudenbosch werd in september 1984 aangevangen met de studie geneeskunde aan de Erasmus Universiteit te Rotterdam. Het doctoraal examen werd in juli 1989 behaald en het arts examen in september 1991. Aansluitend diende de auteur bij de Koninklijke Landmacht (Koning Willem III kazerne te Apeldoorn) tot maart 1993. In de periode van maart 1993 tot heden werd gewerkt aan dit proefschrift als wetenschappelijk assistent bij het instituut Orthopaedie (Faculteit der Geneeskunde en Gezondheidswetenschappen, Erasmus Universiteit Rotterdam).

Uittie II