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Burst fractures of the thoracolumbar spine: changes of the spinal canal during operative treatment and follow-up

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Abstract Although multiple studies have concluded operative decompression of a traumatically narrowed spinal canal is not indicated because of spontaneous remodeling, instrumental decompression is frequently used as part of the operative treatment of spinal fractures. To investigate the process of remodeling, we studied the diameter of the spinal canal in 95 patients with burst fractures at the thoracolumbar junction (T9-L2). To measure and compare the spinal canal's diameter we used either computed tomography (CT) scans or radiographs, made preoperatively, postoperatively, after 9 months and after 24 months. In lateral plain radiographs we found that the initial percentage of cases with bony canal narrowing preoperatively of 76.5 was reduced to 18.4% postoperatively, to 8.2% at 9 months, and to 2.4% at 24 months. In CT scans in a selection of patients, the mean residual

diameter of the spinal canal was 53% preoperatively and 78% at 24 months. The posterior segmental height increases during operation and decreases in the respective periods after operation. So ligamentotaxis can only play a role in the perioperative period. We conclude that a significant spontaneous remodeling of the spinal canal follows the initial surgical reduction. Two years after operation, bony narrowing of the spinal canal is only recognizable in 2.4% of the patients on plain lateral radiographs. The remodeling of the spinal canal can be seen on plain radiographs, although not as accurately as on CT scans.

Keywords Spine · Spinal fractures surgery · Spinal canal · Thoracic vertebrae radiography · Lumbar vertebrae radiography · Ligamentotaxis

Introduction

One of the goals of operative treatment of spinal fractures is restoration of the anatomy of the spinal chain, including the spinal canal. Since 1988 we have been using instrumental decompression of the spinal canal at the thoracolumbar junction in an indirect way, a mechanism known as ligamentotaxis, through distraction applied via pedicle screws of an internal fixator [1, 10, 11, 12, 17, 23, 24, 25].

In a dorsal approach, all fractures were treated by instrumental angular reduction, distraction, and stabilization with Dick's internal fixator [9, 10, 12]. Since 1995 we have used the Universal Spine System, Synthes. The procedure was combined with unilateral (1988–1989) or bilateral (1989–1996) transpedicular cancellous bone grafts, as described by Daniaux [5]. Posterior spondylodesis was performed only at the level of the disturbed cranial or caudal end plate [26, 27].

Several authors have described spontaneous remodeling of the spinal canal during the course of treatment, with or without instrumentation. This finding has been used as an argument against all operative treatment or against direct (open) manipulation of the bony fragment [3, 4, 18,

22, 23, 34]. One of the parameters that correlate with spinal canal encroachment is the posterior vertebral height (PVH) [21]. Another important factor concerning the reduction is the antikyphosed position of the disc and its rigid bony fixation [1, 8]. To quantify the process of reduction during operation and spontaneous remodeling during the period of convalescence, we studied this process in patients with burst fractures of the thoracolumbar junction who were treated by indirect fracture reduction and internal fixation. There were three questions to answer:

- Are there any changes of the posterior segmental height PSH (the sum of posterior vertebral height and posterior intervertebral height) on plain lateral radiographs during operation and in the course of further treatment?
- Does bony narrowing of the spinal canal, as identified on plain lateral radiographs, disappear in the course of treatment, and if so, how many of these patients have developed a normal width of the spinal canal 2 years after the initial treatment?
- Are there changes of the midsagittal diameter of the spinal canal in selected cases with considerable preoperative spinal canal narrowing, as measured on CT slices?

Presumably, the effect of the so-called ligamentotaxis can be measured as changes of the above-mentioned parameters in the perioperative period. The PSH reflects the length of the segmental part of the posterior longitudinal ligament that is involved in the ligamentotaxis. The effect of spontaneous remodeling is reflected in changes of the studied parameters after the operation.

Materials and methods

Between March 1988 and August 1996, 183 consecutive patients with thoracolumbar fractures (T9–L5) were treated operatively at the Traumatology Department of the University Hospital Groningen. Of these patients, according to Magerl's classification [29], 95 had A3 fractures of the thoracolumbar junction (T9–L2) that were treated with indirect operative reduction and fixation with Dick's internal fixator, combined with transpedicular cancellous bone grafting and posterior spondylodesis (Table 1). In these patients, the preoperative plain lateral radiographs (*t*=0) were studied, as well as the postoperative radiographs (*t*=1), the radiographs after 9 months (before implant removal, $t=9$), and at 24 months $(t=24)$. The subclassification, according to the comprehensive classification, of the fractures and the percentage of patients with neurological deficit are shown in Table 2 [29].

Table 2 Comprehensive classification, according to Magerl [29], in 95 patients with A3 fractures, and the number and percentage of patients with neurological deficit

Classification	N	Neurological deficit $N(\%)$	
A3.1	55	9(16.4)	
A3.2	25	3(12.0)	
A3.3	15	3(20.0)	
A3 total	95	15(15.8)	

Posterior segmental height

The changes in the posterior segmental height PSH over the course of treatment and follow-up were analyzed (Fig. 1). Technical differences in radiographic technique were corrected using the proportion of the measured and unchanged height of the adjacent vertebral bodies. Correction of the imaging amplification factor in lateral radiographs (15–20%) was not done, because this would not influence the statistical tests. Most measurements were performed at the center of the photograph. In case of distortion of the radiograph by a non-centered beam, the projected area of the disc was determined from the oval outlines of the rims of the vertebral endplates (Fig. 1). Guidelines as described by Frobin were followed, resulting in a relative measurement error of approximately 3%, consisting of a relative error of the posterior vertebral height of 2.2% (0.7 mm) and of the disc height of 4.2% (0.5 mm) [16].

Bony narrowing

In all 95 patients, the plain lateral radiographs were studied and recognizable bony narrowing of the spinal canal (with or without recognition of a fracture part) was registered (Fig. 2). After an initial period in which conventional tomographies were used in the preoperative workup, all patients had preoperative CT scans. In all preoperative CT scans, the midsagittal diameter was measured and patients were classified into four groups: no narrowing, less than one-third canal narrowing, more than one-third but less than twothirds narrowing, and more than two-thirds canal narrowing [28].

Midsagittal diameter

In 66 patients, preoperative CT scans were available to measure the midsagittal diameter. In 13 patients with a preoperative narrowing of the spinal canal of more than one-third, we compared the

.
Рін **PVH**

Fig. 1 Measurement of posterior segmental height (posterior intervertebral height + posterior vertebral height). See Materials and methods for technique

Fig. 2 Recognizable bony narrowing on plain lateral radiographs in a patient with an A3.1 fracture: (*left to right*) preoperative, postoperative (*horizontal arrows* point to the fragment), at 9 months, and at 24 months (*vertical arrow* points to completely reduced posterior wall)

preoperative midsagittal diameter of the spinal canal with the diameter at 2 years after operation by CT scanning. In CT of the spinal canal, preoperatively and after 2 years, the minimum midsagittal diameter of the spinal canal was compared to the mean of the minimum diameter of the spinal canal one level cranial and one level caudal. These percentages represent the remaining space in the spinal canal, and changes in diameter represent the summed effect of reduction and remodeling. The CT data of these patients were compared to the data of the plain lateral radiographs.

Statistical analysis was performed with the paired samples *t*-test, a parametric test.

Results

Posterior segmental height

The mean PSH on preoperative radiographs measured 40.5 mm, after operation 43.2 mm, before implant removal 41.0 mm and at 24 months it measured 38.7 mm (Table 3). Analysis of the (calculated) change in the PSH in paired

Table 3 Mean (SD) posterior segmental height [PSH; summed posterior vertebral height (PVH) and posterior intervertebral height (PIH)] and percentages of patients with recognizable bony canal narrowing on plain lateral radiographs during the course of treatment (*n*=95)

	PSH (mm) Mean (SD)	Bony narrowing $(\%)$	
Preoperative	40.5(5.7)	76.5	
Postoperative	43.2(4.1)	18.4	
At 9 months	41.0(3.9)	8.2	
At 2 years	38. (3.8)	2.4	

Table 4 Paired samples *t*-test comparing the calculated differences in the length of the ligament along PVH and PIH to the test value zero, in the perioperative period (period 1), the period between initial operation and implant removal (period 2), and the period after implant removal until end of follow-up at 24 months (period 3) (*CID* confidence interval of the difference, *SEM* standard error of the mean, *t* test value, *df* degrees of freedom)

measurements in the perioperative period, the period until implant removal and the period until 24 months after the initial operation shows an increase in the PSH during operation of 2.7 mm (*P*<0.001). After the operation, decreases of 2.2 mm (until implant removal) and 2.3 mm (until 24 months postoperatively) occur (*P*<0.005) (Table 4).

Bony narrowing

On plain lateral radiographs, recognizable bony encroachment of the vertebral canal was seen preoperatively in 76.5% of the patients, postoperatively in 18.4%, at 9 months in 8.2% and at 2 years in 2.4% (Table 3, Fig. 3).

Midsagittal diameter

Preoperative CT scans in 66 patients with A-fractures showed a measurable spinal canal narrowing in 89.4%. Less than one-third canal narrowing was found in 48.5%, while in 34.8% spinal canal narrowing was greater than one-third, but less than two-thirds. Only 6.1% of the patients had greater than two-thirds canal narrowing. Patients with spinal canal narrowing of between one-third and two-thirds showed the highest percentage of neurological deficit (Table 5).

Fig. 3 Percentage of patients with bony spinal canal narrowing on plain lateral radiographs taken preoperatively, postoperatively and at 9 and 24 months after operation, in 95 patients with A-type fractures

Table 5 Narrowing of the spinal canal measured on computed tomographic (CT) scan in 66 patients with burst fractures, and the number (percentage) of them with neurological deficit

Narrowing	$N(\%)$	Neurological deficit $(\%)$	
$\bf{0}$	7(10.6)	1(14.3)	
$0 - 1/3$	32(48.5)	4(12.5)	
$1/3 - 2/3$	23(34.8)	5(21.7)	
>2/3	4(6.1)	0(0)	
Total	66 (100)	10(15.2)	

Table 6 Residual percentage of midsagittal spinal canal diameter on CT scan and number (percentage) with recognizable bony narrowing on lateral radiograph in 13 patients

	Canal narrowing on CT (%) Mean (SD)	Bony narrowing $N(\%)$
Preoperative	47.7(21.6)	12(92)
Two-year	21.7(20.5)	2(15)

Table 7 Paired *t*-test, comparing differences between preoperative residual ratio of the midsagittal diameter of the spinal canal and the residual spinal canal at 2 years

Traumatic narrowing of the spinal canal in the sagittal plane on the preoperative CT scans left a mean residual midsagittal diameter of 52.3% (*n*=66). After 2 years, the midsagittal diameter was 78.3% (*n*=13). Bony narrowing on lateral radiographs in these patients was recognized in 92% preoperatively and 15% after 2 years (Table 6). The paired samples *t*-test showed that the changes between preoperatively and 2 years postoperatively accounted for 25.0% (two-tailed significance *P*<0.001) (*n*=14) (Table 7).

Discussion

It is generally accepted that bony encroachment of the spinal canal in the thoracolumbar region by one-third or more may jeopardize the spinal cord [19, 28]. There is a correlation between the level of the spinal fracture and the probability of neurological deficit. More cranial levels of fracture have a higher probability of neurological deficit [13]. The percentage of spinal canal narrowing as measured on CT scan has a positive correlation with the probability of neurological deficit as well [13, 19]. In this regard, one should recognise that the measured residual diameter at the time of the preoperative radiographs is greater than the diameter at the moment of trauma [30]. Another aspect of interest is that the midsagittal diameter overestimates the canal narrowing, compared to measurements of the cross sectional area [14]. Posterior segmental height

Through intact spinal ligaments and discs, a partial fracture reduction will be induced when the patient is put in a supine position during initial care and transport. Extension does not widen the spinal canal in an unfractured spinal column [21], but the combination of angular fracture reduction and distraction will widen the spinal canal by means of ligamentotaxis [15]. For anatomical reasons, ligamentotaxis below the L2 level is weak or even absent [23]. If the longitudinal ligaments – especially the posterior longitudinal ligament – are not (completely) disrupted, distraction and antikyphosis can achieve a reduction of bony fragments, narrowing the spinal canal of the injured spine.

The effect of the forces conducted via the attachment of the annulus to the end plates by instrumental and postural antikyphosing reduction will add to the restoration of the spinal canal wall.

Bony canal narrowing

It is the trabecular structure of the spinal body that causes the typical trapezoid fracture part in the posterior wall in burst fractures, which results in narrowing of the spinal canal. The trabeculae are found at the medial corner of the base of the pedicles and extend in a radial array throughout the vertebral body. A stress concentration near the base of the pedicles results in the typical fracture at the posterior wall of the vertebral body in severe compression [20].

In 1991, Johnsson et al. reported on 17 thoracolumbar fractures and concluded that manipulative open reduction of spinal canal wall and bony fragments in the spinal canal is not necessary because spontaneous spinal canal remodeling occurs in non-operated as well as operated patients. In their study, 14 operated and 3 non-operated patients with a follow-up of 1–4 years were evaluated. One of the conclusions was that there were no differences between non-operated and operated cases concerning the restitution of the spinal canal. In that study, the measurements were difficult to interpret because laminectomy in some cases or interference from Harrington rods in other cases influenced the quality of the radiographs and the measurements [22].

Scapinelli reported in 1995 on five adult patients with thoracolumbar spinal fractures with associated intracanal displacement of a large bone fragment. Three of these patients had neurological deficit and two did not. Four patients underwent operative posterior stabilization by Harrington rods and bone autografts without surgical decompression. These treatments led to neurological recovery in all but one case. Comparison of CT scans in all patients after 1.5–5 years showed remodeling of the spinal canal. Rhythmic respiratory oscillations in the cerebrospinal fluid pressure were suggested as a factor in the mechanism of bone resorption. It was concluded that removal of intraspinal fragments was no longer necessary [32].

was found between the spontaneous reduction of spinal

The discussion about laminectomy as the procedure of choice for decompression in patients with neurological deficit will probably never end completely. We have previously reported on 183 patients (17% of them with neurological deficit) treated by dorsal instrumentation, in which we used operative decompression by laminectomy in one case only, because of progressive neurological deterioration after the initial dorsal operative procedure [27]. Boerger et al. [2] showed that no reason for surgical decompression can be found in published research. In the present study, we confirm a link between initial canal narrowing and the risk for traumatic neurological deficit. We could not confirm a higher incidence of neurological deficit in the subclassification of more severe A3 fractures, but low numbers of A3.3 type fractures and low numbers of severe (greater than 2/3) traumatic canal narrowing make it impossible to draw any conclusions from this observation. One should recognize that the midsagittal diameter reduction overestimates the traumatic reduction of the spinal canal compared to CT cross-sectional area measurements [14].

The present study shows that fracture reduction by angular correction and distraction is associated with a marked increase in the percentage of patients with a cleared spinal canal – from 23.5% to 81.6%. This very large change is followed by a further increase in the number of patients with complete canal clearance, to 91.8% at 9 months after surgery. At the end of follow-up $-$ at 24 months postoperatively – only 2.4% of the patients have residual canal narrowing (Fig. 3).

Midsagittal diameter

The caliber of the vertebral canal in the lumbar region shows a large variation. The anteroposterior diameter in normal individuals decreases from 17.3 mm (range 13– 22 mm at level L1) to 15.9 mm (range 9–21 mm at level L4) [7, 28]. At the thoracolumbar zone, the cross-section of spinal canal shows the transition from an oval format (thoracic spine) to a triangular form with rounded angles (lumbar spine) [28]. The diameter of the spinal cord has a mean caliber of 10 mm, although it may be broader at the thoracolumbar region. Normally this results in considerable spare room around the spinal cord.

In 1998, de Klerk et al. reported a retrospective study of 42 trauma patients with initial spinal canal stenosis of more than 25%, who were treated conservatively [6]. CT in their study was performed at between 12 and 108 months after the injury. One of their conclusions was that conservative treatment is followed by a marked degree of spontaneous restitution of the deformed spinal canal. They showed that the higher the initial percentage of canal stenosis, the greater the spontaneous reduction. Age at the time of injury was inversely correlated with the reduction in the percentage of spinal canal stenosis. No correlation canal stenosis and the time gone by since the injury. This suggests that the changes occurred within the first 12 months. In 1992, Gertzbein et al. showed a reduction of the preoperative canal encroachment by distraction forces, delivered by an internal fixator, from 54% to only 40%, but a selection of patients operated within the first 4 days after injury showed a reduction from 56% to 38% [17]. The importance of the sagittal alignment with respect to the forces that act on the fracture parts has to be stressed. The antikyphosing reduction is reflected in an increase in the postoperative intervertebral angle [27]. The rigid fixation of the annulus to the upper and lower fragment parts will certainly add to the reduction forces. Segmental stability by rigid fixation will add to the persistence of the reduction force. In our series, the lateral radiographs show that in only 18.4% of the cases canal encroachment could be visualized postoperatively.

It seems logical to measure the midsagittal diameter on lateral plain radiographs, but in contrast to the dorsal part of the vertebral body, the arch can not be sufficiently distinguished in the lateral radiograph to gain reliable measurements.

The incidence of spinal cord injury in spine fractures and dislocations is approximately 14% of the total, as ascertained from a survey of these injuries in Northern California [31]. With respect to location, injuries to the cervical spine produced the highest rate of neurological damage, the incidence of neurological deficit being 39% [31]. With respect to type of injury, patients sustaining fractures of the vertebral bodies and posterior elements with some degree of malalignment of the spine had a 61% incidence of neurological deficit. In our series we found more than 20% neurological deficit in patients with between one-third and two-thirds spinal canal narrowing, but we saw four patients with a high degree of traumatic canal narrowing $(>=2/3)$ without any neurological deficit (Table 3). This is surprising, because canal narrowing influences the risk of neurological involvement. L1 fractures with more than 65% of canal narrowing have a high probability of being accompanied by neurological deficit [19, 33].

Conclusions

In this study we showed that the initial restoration of the spinal canal by indirect manipulation and disco-ligamentotaxis is incomplete. The posterior segmental height increases in the perioperative period, and clearance of the spinal canal is observed in about 75% of the patients with traumatic canal narrowing on plain lateral radiographs. Later, ligamentotaxis does not play a role anymore, because the PSH even diminishes. The clearance of the spinal canal as interpreted from CT scans and lateral radiographs continues in the course of follow-up. At 2 years after operation, about 97% of all patients with burst fractures have a completely free spinal canal on lateral radiograph, but not all fractures and fracture parts can be identified on plain lateral radiographs. So plain lateral radiographs seem to overestimate the process of remodeling.

This study provides clinical data for the description of (partial) spontaneous remodeling. This phenomenon can be observed on plain lateral radiographs, but not as accurately as on CT scans.

References

- 1. Aebi M, Etter C, Kehl T, Thalgott J (1987) Stabilization of the lower thoracic and lumbar spine with the internal spinal skeletal fixation system. Indications, techniques, and first results of treatment. Spine 12:544–551
- 2. Boerger TO, Limb D, Dickson RA (2000) Does 'canal clearance' affect neurological outcome after thoracolumbar burst fractures? J Bone Joint Surg Br 82:629–635
- 3. Chakera TM, Bedbrook G, Bradley CM (1988) Spontaneous resolution of spinal canal deformity after burstdispersion fracture. Am J Neuroradiol 9:779–785
- 4. Dai LY (2001) Remodeling of the spinal canal after thoracolumbar burst fractures. Clin Orthop 382:119–123
- 5. Daniaux H (1982) Technik und erste Ergebnisse der transpedikulären Spongiosaplastik bei Kompressionsbrüchen im Lendenwirbelsäulenbereich. Acta Chir Austriaca [Suppl] 43:79
- 6. de Klerk LW, Fontijne WP, Stijnen T, Braakman R, Tanghe HL, van Linge B (1998) Spontaneous remodeling of the spinal canal after conservative management of thoracolumbar burst fractures. Spine 23:1057–1060
- 7. Delmas A, Pineau H (1970) Sur le canal vertébral de la colonne lombaire. CR Assoc Anat 145:135–138
- 8.Dick W (1984) Osteosynthese schwerer Verletzungen der Brust- und Lendenwirbelsäule mit dem Fixateur interne. Langenbecks Arch Chir 364:343–346
- 9. Dick W (1987) Innere Fixation von Brust- und Lendenwirbelfrakturen. In: Burri C, Harder F, Bauer R (eds) Aktuelle Probleme in Chirurgie und Orthopädie, 2nd edn, vol 2. Hans Huber, Bern, pp 1–137
- 10. Dick W (1987) The "fixateur interne" as a versatile implant for spine surgery. Spine 12:882–900
- 11. Dick W (1992) Dorsale Stabilisierung bei Brust- und Lendenwirbelverletzungen. Langenbecks Arch Chir Suppl Kongressbd 290–292
- 12. Dick W, Kluger P, Magerl F, Woersdorfer O, Zach G (1985) A new device for internal fixation of thoracolumbar and lumbar spine fractures: the 'fixateur interne'. Paraplegia 23:225–232
- 13. Fontijne WP, de Klerk LW, Braakman R, Stijnen T, Tanghe HL, Steenbeek R, van Linge B (1992) CT scan prediction of neurological deficit in thoracolumbar burst fractures. J Bone Joint Surg Br 74:683–685
- 14. Frank E, Bonsell S (1994) The accuracy of anterior-posterior measurements in the assessment of spinal canal compromise in burst fractures. Neurol Res 16:410–412
- 15. Fredrickson BE, Mann KA, Yuan HA, Lubicky JP (1988) Reduction of the intracanal fragment in experimental burst fractures. Spine 13:267–271
- 16. Frobin W, Brinckmann P, Biggemann M, Tillotson M, Burton K (1997) Precision measurement of disc height, vertebral height and sagittal plane displacement from lateral radiographic views of the lumbar spine. Clin Biomech 12:S1-S63
- 17. Gertzbein SD, Crowe PJ, Fazl M, Schwartz M, Rowed D (1992) Canal clearance in burst fractures using the AO internal fixator. Spine 17:558–560
- 18. Ha KI, Han SH, Chung M, Yang BK, Youn GH (1996) A clinical study of the natural remodeling of burst fractures of the lumbar spine. Clin Orthop 323:210–214
- 19. Hashimoto T, Kaneda K, Abumi K (1988) Relationship between traumatic spinal canal stenosis and neurological deficits in thoracolumbar burst fractures. Spine 13:1268–1272
- 20. Heggeness MH, Doherty BJ (1997) The trabecular anatomy of thoracolumbar vertebrae: implications for burst fractures. J Anat 191:309–312
- 21. Isomi T, Panjabi MM, Kato Y, Wang JL (2000) Radiographic parameters for evaluating the neurological spaces in experimental thoracolumbar burst fractures. J Spinal Disord 13:404–411
- 22. Johnsson R, Herrlin K, Hagglund G, Stromqvist B (1991) Spinal canal remodeling after thoracolumbar fractures with intraspinal bone fragments. 17 cases followed 1–4 years. Acta Orthop Scand 62:125–127
- 23. Kuner EH, Kuner A, Schlickewei W, Wimmer B (1992) Die Bedeutung der Ligamentotaxis für die Fixateur-interne-Osteosynthese bei Frakturen der Brustund Lendenwirbelsäule. Chirurg 63: 50–55
- 24. Kuner EH, Kuner A, Schlickewei W, Mullaji AB (1994) Ligamentotaxis with an internal spinal fixator for thoracolumbar fractures. J Bone Joint Surg Br 76:107–112
- 25. Kuner EH, Schlickewei W, Kuner A, Hauser U (1997) Restoration of the spinal canal by the internal fixator and remodeling. Eur Spine J 6:417–422
- 26. Leferink VJM, Nijboer JMM, Zimmerman KW, Veldhuis EFM, ten Vergert EM, ten Duis HJ (2001) Thoracolumbar spinal fractures: segmental range of motion after dorsal spondylodesis in 82 patients: a prospective study. Eur Spine J 11:2–7
- 27. Leferink VJM, Zimmerman KW, Veldhuis EFM, ten Vergert EM, ten Duis HJ (2001) Thoracolumbar spinal fractures: radiological results of transpedicular fixation combined with transpedicular cancellous bone graft and posterior fusion in 183 patients. Eur Spine J 10: 517–523
- 28. Louis R (1983) Surgery of the spine: surgical anatomy and operative approaches. Springer, Berlin Heidelberg New York, pp 77–83
- 29. Magerl F, Aebi M, Gertzbein SD, Harms J, Nazarian S (1994) A comprehensive classification of thoracic and lumbar injuries. Eur Spine J 3:184–201
- 30. Panjabi MM, Kifune M, Wen L, Arand M, Oxland TR, Lin RM, Yoon WS, Vasavada A (1995) Dynamic canal encroachment during thoracolumbar burst fractures. J Spinal Disord 8:39–48
- 31. Riggins RS, Kraus JF (1977) The risk of neurological damage with fractures of the vertebrae. J Trauma 17:126–133
- 32. Scapinelli R, Candiotto S (1995) Spontaneous remodeling of the spinal canal after burst fractures of the low thoracic and lumbar region. J Spinal Disord 8:486–493
- 33. Sjostrom L, Karlstrom G, Pech P, Rauschning W (1996) Indirect spinal canal decompression in burst fractures treated with pedicle screw instrumentation. Spine 21:113–123
- 34. Wessberg P, Wang Y, Irstam L, Nordwall A (2001) The effect of surgery and remodeling on spinal canal measurements after thoracolumbar burst fractures. Eur Spine J 10:55–63