Thoracolumbar burst fractures without neurological deficit: the role for conservative treatment

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Abstract Burst fractures are common in the thoracolumbar junction and account for 17% of all major spinal fractures. There is a considerable controversy on the efficacy of conservative treatment and the need for surgical intervention. Need for additional stability, prevention of neurological deterioration, attainment of canal clearance, prevention of kyphosis and early relief of pain are the commonly quoted reasons for surgical intervention. However, a careful review of literature does not validate any of the above arguments. The available randomised control trials prove that the results of conservative treatment for burst fractures are equal to that of surgery and also with lesser complications. Surgery for burst fractures may, however, have definite advantages in patients with poly-trauma or in the rare event of deteriorating neurology. It is also important for the treating surgeon to clearly distinguish a burst fracture from other inherently unstable injuries like fracture dislocations, chance fractures and flexion rotation injuries which require surgical stabilisation.

Keywords Burst fractures · Conservative treatment · Deformity · Neurological deficit

Introduction

“Surgical wisdom lies in avoiding doing more, When less is sufficient....”

Burst fractures result from compression failure of both the anterior and middle columns under substantial axial loads [1]. The sudden application of a supra-physiological axial load results in vertebral end plate failure as adjacent disc tissue is driven into the vertebral body. The vast majority of burst fractures are associated with some degree of canal compromise, typically as a result of retropulsion of an osseous fragment or fragments from the posterior superior end plate. As a result neurologic injury has been reported to occur in 30% of the patients with thoracolumbar fractures [2].

Burst fractures account for up to 17% of all major spinal fractures [1]. The thoracolumbar region (T11 to L2) is the commonest site of burst fractures [1] as the region forms a transition zone between the relatively fixed, kyphotic thoracic spine above, and the relatively mobile, lordotic lumbar spine below, which causes stress forces to concentrate upon the thoracolumbar vertebral column. [3].

Characteristic radiographic features of a burst fracture include loss of posterior vertebral body height, posterior vertebral body angle $>100^\circ$, a break in the posterior wall of the vertebral body with narrowing of the spinal canal on the lateral view and widening of the interpedicular distance on the anterior–posterior view with a sagittal split of the posterior arch. The radiological evaluation should also include an assessment of the kyphosis angle and interspinous widening on lateral radiographs. Plain radiological appearance may sometimes appear similar between a pure burst fracture and a burst fracture which is a part of a complex discoligamentous injury of the posterior ligament complex and facet joints. Both are two completely different injuries. 25% of burst fractures are misdiagnosed as compression fractures if radiographs alone are evaluated uncarefully or are not supported by additional investigations like computer tomography or MRI [4]. The CT imaging is

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useful for demonstrating the extent of canal compromise and occasionally occult posterior element fractures. Additional MRI imaging is required for patients with a neurological deficit, to identify a possible spinal cord, cauda equine or root injury, cord oedema, haemorrhage, epidural haematoma and posterior ligamentous disruption which cannot be identified in the other imaging modalities. AO type B injuries require a different treatment strategy than a simple burst fracture.

Treatment of burst fractures: management dilemmas

The aim of any management regime in burst fractures is primarily prevention and limitation of neurological injury with restoration of spinal stability. Correction of deformity, minimising loss in movements and rapid return to normality are the other considerations. Achieving these goals with least additional risk to the patients will be an added aim. There is considerable controversy in literature regarding how this aim can be achieved with strong proponents for both non-operative and operative treatments.

There is no controversy on the need for surgery in patients with progressive neurological loss, unstable fractures with complete neurological loss or patients with polytrauma who require fixation for early and easier rehabilitation. However they comprise only a minority in burst fractures.

There is no consensus on the management of burst fractures with intact neurology. Numerous authors have reported excellent results after non-operative management without reduction. On the other hand, proponents of surgery believe that decompression, fracture reduction and stabilisation are essential for stabilising neurology and reducing pain. Among the advocates of surgery, there is also no consensus regarding the timing of surgery, the approach and whether posterior only, anterior only or combined dorso-ventral stabilisation is required for fusion.

The argument in favour of surgery is usually based on the following factors.

1. Need for stability
2. Prevention of neurological deterioration
3. To provide canal clearance
4. To prevent kyphosis, and
5. Early relief of pain and return to work.

It is worthwhile to discuss each of the above concerns in detail and examine the evidence.

Providing stability: are burst fractures unstable?

The concept of spinal instability is poorly understood and there have been numerous classifications which have tried

to define instability on an anatomical basis. These classifications have placed emphasis on the integrity of a specific anatomical structure for evaluating stability. For example, Dennis [1] classification placed emphasis on the integrity of the middle column. McAfee et al. [5] classification was basically a modification of Dennis classification with emphasis on the CT diagnostic findings. Magerl et al. [6] proposed a classification which combines pathomorphological findings which can be evaluated with imaging modalities on one side and mechanics of the injury on the other side. The key element is whether the so-called posterior ligament complex is injured or not. The Thoraco-Lumbar Injury Classification and Severity Score (TLICS) of Vaccaro et al. [7] tries to combine the Magerl et al. classification [6] with a scoring system to have a clear base for treatment guidelines.

Overall, these classifications have the disadvantage of ignoring the stabilising effect of the paraspinal muscles which play an important protective role in *in vivo* conditions. The injured structures are also capable of healing naturally over a period of time and a spine fracture which is unstable by definition may be in fact become stable once natural healing of the injured structures occurs. Classifications for spinal fractures have also not been validated by randomised clinical trials nor have there been clinical studies comparing the clinical outcome of surgical and conservative treatment based on these classifications. It is therefore important that, we rely on the results of clinical studies rather than the proposals made by the originators of the classification.

In assessing spinal instability, the clinical concerns of the treating surgeon have been reflected by White and Panjabi's [8] definition of instability as "a loss in the ability of the spine under physiologic loads to maintain relationships between vertebrae in such a way that there is neither initial damage nor subsequent irritation to the spinal cord or nerve roots and, in addition there is no development of incapacitating deformity or pain due to structural changes." We will now see what the literature says about the three factors of concern—progressive neurological deterioration, progressive pain and increase in deformity.

Neurological deterioration

Neurological deterioration in a spinal cord injury occurs primarily due to a variety of forces at the time of injury, such as rapid cord compression, acute distraction, acceleration or deceleration with shearing, transaction or penetration. The exact force depends upon the nature of injury and occurs at the time of the accident. Secondary deterioration can occur due to "Secondary injury

pathways” where ischaemia with circulatory changes [9, 10], ionic derangements [11, 12], accumulation of neurotransmitters, arachadonic acid metabolites and free radicals [13], lipid peroxidation [14–16] and apoptosis [17] with cell death have all been implicated. Secondary injury pathways are more often responsible for the physiological insult to the spinal cord leading to neurological deterioration than secondary mechanical compression. There is no proof to suggest that there is increased displacement or progressive canal compromise by retropulsion of the fragment in a burst fracture [18, 19].

The report by Denis et al. [20] stands out amongst literature as the only study reporting a neurological deterioration in 6 out of 29 patients. Mumford et al. [21] in a follow-up of 41 cases of burst fractures could notice neurological deterioration in only one patient who developed only a radiculopathy at a single level. There is ample proof by both short and long term clinical studies that neurological deterioration is in fact very rare [7, 11, 12, 22–30].

Weinstein et al. [31], in a series with perhaps the longest follow-up had no cases of early or late neurological deterioration in 42 patients after an average period of 20 years. Chow et al. [32] treated 26 patients with unstable burst fractures with hyper extension casting or bracing and had no neurological worsening. Other authors Cantor et al. [33], Chan et al. [34], Shen et al. [19, 35], Wood et al. [36], Tropiano et al. [27] and Celibi et al. [37] had similar findings (Table 1).

The myth of ‘Canal Clearance’

Burst fractures are usually associated with some degree of canal compromise as the vertical compression forces often result in retropulsion of bone fragments into the canal. The image of retropulsed fragments in the spinal canal is often dramatic on a CT scan and a canal compromise in excess of 25% is an often quoted indication for surgery for fear of neurological deterioration either in the acute stage or during rehabilitation. Neurological injury is primarily due to the mechanical damage to the cord at the time of the accident [38]. In a patient with intact neurology the fact that he remains neurologically intact is evidence enough that the degree of canal compromise is not sufficient to cause neural damage. [35] Therefore, the need for surgical decompression of the canal is not present [38]. There are also studies to prove that the geometry of the canal is not a good guide to the extent of neurological dysfunction [39–41] and that surgical decompression of the spinal canal will not alter the outcome [24, 38, 42].

The fear that the canal compromise will progress and result in spinal canal stenosis or neurological deficit [43] in later life is also unfounded [38]. In fact the opposite is true as the retropulsed fragments gradually resorb with remodelling of the canal. Over a period of 2 years, many studies have reported an improvement in canal dimension on subsequent scanning [35, 37, 44–47]. Shen et al. [19] noted a resorption of approximately 50% of the retroplused fragment within 12 months. Interestingly, Celebi et al. [37] found that the higher the amount of initial spinal canal

Table 1 Literature on non-operative management of thoracolumbar burst fractures

Author (year)	No. of patients	Average follow-up	Neurological deterioration	Kyphosis	Pain at follow-up	Functional status
Mumford (1993)	41	2 years	1 case (radicular)	8% progression of body collapse	88% excellent to fair outcome relative to pain	90% satisfactory work status
Weinstein (1988)	42	20 years	None	Kyphosis of 26° in flex, 17° in extension	Average pain score 3.5	80% back to usual work
Cantor (1993)	18	19 months	None	Average of 1° progression (19° to 20°)	83% little or no pain	94% no restriction of activity
Chan (1993)	20	3.9 years	None	Average of 1.6° of progression (8° to 9.6°)	95% mild to no back pain	90% excellent outcome, all returned to work
Chow (1996)	26	34 months	None	Average of 2.3° of progression	79% little or no pain	75% return to usual work
Shen (1999)	38	4 years	None	Average of 4° progression (20° to 24°)	84% occasional or no pain	76% return to usual work
Tropiano (2003)	45	34 months	None	Average of 1.2° progression	64% little or no pain	81% return to usual work
Celibi (2004)	26	43 months	None	Average of 8.3° ± 4.3° progression	73% occasional or no pain	77% return to previous work

compromise, the better the remodelling. Yazici et al. [47] and Dai et al. [44] found no statistical difference in the degree of spinal canal remodelling between patients treated conservatively and operatively.

Prevention of kyphotic deformity

A much debated subject in literature is the extent and progresses of deformity following a burst fracture and its clinical relevance. In patients treated with an orthosis or hyper extension cast and early ambulation, the range of kyphosis progression is from 1° to 6° when all types of burst fractures are considered [7, 11, 12, 22, 25, 27] (Table 1). Most of the kyphotic progression occurs in the initial few months after injury after which the deformity stabilises [35]. Although posterior short segment surgery gives better correction of the deformity in the immediate post operative period [19, 36, 48], the kyphosis gradually recurs in the rehabilitative phase with the final kyphosis often being similar to the pre operated state [9, 10, 19, 25, 26, 36, 45, 49]. An increase in deformity upto 12° has been reported following surgery by posterior methods [49]. McNamara et al. [45] reviewed 13 patients treated surgically and reported a post operative kyphosis progression of 8.7° with only 69% return to pre-injury activity. In similar studies Stephens et al. [10] and Kramer et al. [49] reported a post operative progression of 11.9° and 12.9°, respectively.

A randomised control study by Wood et al. [36] comparing operative with nonoperative management showed progression of kyphosis in both groups. In the surgically managed group the average kyphosis was 10.1° at admission, corrected to 5° but progressed to 13° at follow-up. In the nonoperative group it was 11.3° on admission, corrected to 10° which progressed to 13.8° at follow-up. There was no statistically significant difference in the kyphosis between the two groups at final follow-up. Shen et al. [19] and Knight et al. [23] reported similar findings in their study which included 80 and 22 patients, respectively. There is no scientific basis to assume that kyphosis progression is more with conservative treatment.

Relief of pain and return to work (Functional status)

The relationship between post-traumatic kyphotic deformity and chronic back pain is ambiguous. The proposed mechanism of pain generation is that thoracolumbar kyphosis is balanced by lumbar hyperlordosis, which then causes muscle fatigue and pain. It is also thought that altered non physiological movements at the adjacent facet joint causes facet arthrosis and pain [50]. However, several studies indicate that there is no direct relationship between

kyphosis and back pain or functional impairment either with conservative treatment or following surgery [21, 31–33, 49] (Table 1).

Weinstein et al. [31] reported a large series of patients with burst fractures treated non-operatively who had excellent results, with more than 80% able to resume their pre-trauma occupation. Mumford et al. [21] reviewed the results of non-operative management of 41 thoracolumbar burst fractures. At average follow-up of 2 years, an overall outcome evaluation indicated that 49% of the patients had excellent outcomes relative to pain and function; 17% good; 22% fair; and 12% poor. They also noticed 8% progression due to bony collapse. Shen et al. [35] in their study used a Jewett type hyperextension brace in nine (of 38 patients) while the rest were ambulated without a brace. The patients were allowed to ambulate as soon as pain allowed with no restrictions on activity. With this line of management 76% patients returned to their original employment, they found average 4° increase in the final deformity. They also noted a poor correlation of the clinical results even when the kyphosis was greater than 30°. Tropiano et al. [27] treated 45 patients with thoracolumbar/lumbar burst fractures by closed reduction and casting. Closed reduction resulted in significant correction of vertebral wedging from a mean of 15°–5° and anterior body wedging corrected by 63%. Although deformity tended to recur by 4 months, 78% had satisfactory work and functional status at final follow-up. They had no complications due to reduction procedure.

Studies comparing operative and nonoperative management of burst fracture conclude that even though short and long term radiological results may be marginally better in the operative group, there is no significant difference in treatment outcome in terms of back pain and functionality between the groups [23, 27, 28, 36, 48, 51]. Interestingly, in the study of Wood et al. [36], conservatively managed group was found to have significantly lower pain scores than the operative group. Resch et al. [48] found all patients in the nonoperative group to be satisfied, while 15% of the surgical group were unsatisfied with the outcome.

In our experience as well as others painful progressive post-traumatic kyphosis, is a problem in less than 10% of all cases of thoracolumbar spine fractures treated nonoperatively [38]. For the few who do develop a post-traumatic painful kyphosis despite orthotic treatment, surgical stabilisation can be carried out when indicated [38].

Is surgery always successful?

There is no universal agreement amongst the proponents of surgery regarding the timing of surgery, extent of

Fig. 1 A L1 burst fracture in a 56 year old male (a and b). Patient was treated conservatively and consolidation occurred with an increase of kyphosis by 13° (c) At 4 year follow-up, patient is completely pain free and has returned to his previous work

stabilisation, the ideal approach and whether a combined anterior and posterior stabilisation is required and if it should be done in a single or as a two-staged procedure. Notwithstanding the controversies in surgical treatment, the principal disadvantage of surgery is the wide variety of complications that have been reported [9, 19, 23, 25, 28, 36, 49]. While deterioration of neurology by conservative treatment is mostly anecdotal without firm evidence, the risk of additional neurological injury during surgery is real but often under-reported by surgeons [19]. Other major disadvantages would be the risk for post operative infection, intra operative dural tears, pseudo arthrosis, failure of instrumentation and complications related to anaesthesia including atelectasis [9, 10, 25–28, 45, 49, 51]. There is also the disadvantage that many of these patients require a second stage surgery to remove the implants.

Comparative studies have found that complications were higher for patients treated surgically due to the above surgery related complications [19, 36]. This is a disadvantage as there is no proof that the final functional outcome and the rate of return to work is better in the surgical group [19, 28, 36, 37, 48].

A final advantage of conservative treatment is the low cost of treatment [36] which is an important issue for patients with spinal fractures in developing countries. Shen et al. [19] documented that the hospital expenses are four times higher in the surgically treated patients when compared to the conservatively treated group.

Non-operative management of burst fractures with normal neurology

Many different protocols have been proposed for conservative treatment, but they all involve initial rest in bed with or without postural reduction, followed by application of an extension cast or brace and early mobilisation. The prolonged bed rest for 6–8 weeks as advocated by Bedbrook [52] is no longer advocated. Numerous authors agree that early mobilisation is important for better clinical outcome and no complication has been observed related to early mobilisation [7, 11, 12, 22, 25, 27]. The need for postural reduction and bracing are debatable. Postural reduction can produce an indirect decompression of the spinal canal and a

Fig. 2 A L1 burst fracture in a 30-year old male. (a, b). He was treated conservatively in a Taylor's orthosis, consolidation occurred with progression from 12° to 18° (c). At 3 year follow-up he is completely asymptomatic and has returned to his work as a manual labourer

well-fitted cast or orthosis to maintain the reduction has been recommended by many advocating conservative treatment [7, 12, 27, 34]. Although a variety of braces such as anterior hyperextension brace (ASH), Jewett brace, Taylor–Knight brace and Total body contact TLSO are available, the correction of kyphosis obtained initially is gradually lost [7, 11, 12, 22, 25, 27]. However, the final increase in kyphosis is only around 4–6 degrees and this compares favourably with the deformity progression following surgery. There is general consensus now that bracing probably offers only pain control and facilitates early ambulation rather than preventing deformity [35, 53].

In our institutions' protocol, we do not attempt to reduce the fracture and allow the patient to rest in regular beds till there is relief of pain. Patients are allowed to roll in bed, sit with support and ambulate with a moulded brace and are discharged when they are ambulant and comfortable. They are followed periodically till radiological healing is observed and rehabilitation allows return of work.

Conclusions

It is important for the treating surgeon to distinguish a burst fracture from other inherently unstable injuries like fracture

dislocations, ligamentous Chance fractures and flexion distraction injuries as the latter require surgical stabilisation for favourable outcomes. It is surprising that there is a paucity of randomised control trials which address the issue of appropriate treatment in these common injuries. Clinical studies reported so far imply that most burst fractures can be treated highly successfully by conservative methods, and the final outcome is equal, if not superior to that reported by surgical treatment. Surgery has definitive advantages in patients with polytrauma or in the rare event of a patient having deteriorating neurology. Non-operative treatment can be safely adopted in the management of thoracolumbar burst fractures with normal neurology. (Figs. 1, 2)

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References

1. Denis F (1983) The three-column spine and its significance in the classification of acute thoracolumbar spinal injuries. *Spine* 8:817–831

2. Tator CH, Koyanagi I (1997) Vascular mechanisms in the pathophysiology of human spinal cord injury. *J Neurosurg* 86:483–492
3. Gertzbein SD (1992) Scoliosis Research Society. Multicenter spine fracture study. *Spine* 17(5):528–540
4. Ballock RT, Mackerie R, Abitbol JJ, Cervilla V, Resnick D, Garfin SR (1992) Can burst fractures be predicted from plain radiographs? *J Bone Joint Surg Br* 74:147–150
5. McAfee PC, Yuan HA, Lasda NA (1982) The unstable burst fracture. *Spine* 7:365–373
6. Magerl F, Aebi M, Gertzbein SD, Harms J, Nazarian S (1994) A comprehensive classification of thoracic and lumbar injuries. *Eur Spine J* 3(4):184–201
7. Trafton PG, Boyd CA (1984) Computed tomography of thoracic and lumbar spine injuries. *J Trauma* 24:506–515
8. Weinstein JN, Collalto P, Lehmann TR (1988) Thoracolumbar “burst” fractures treated conservatively: a long-term follow-up. *Spine* 13:33–38
9. McLain RF, Sparling E, Benson (1993) Early failure of short-segment pedicle instrumentation for thoracolumbar fractures. A preliminary report. *J Bone Joint Surg [Am]* 75:162–167
10. Stephens GC, Devito DP, McNamara MJ (1992) Segmental fixation of lumbar burst fractures with Cotrel-Dubousset instrumentation. *J Spinal Disord* 5:344–348
11. Leybaert L, de Hemptinne A (1996) Changes of intracellular free calcium following mechanical injury in a spinal cord slice preparation. *Exp Brain Res* 112:392–402
12. Wood K, Buttermann G, Mehbod A, Garvey T, Jhanjee R, Sechriest V (2003) Operative compared with nonoperative treatment of a thoracolumbar burst fracture without neurological deficit. A prospective, randomized study. *J Bone Joint Surg* 85-A(5):773–781
13. Hall ED, Braughler JM (1993) Free radicals in CNS injury. *Res Publ Assoc Res Nerv Ment Dis* 71:81–105
14. Braughler JM, Hall ED (1992) Involvement of lipid peroxidation in CNS injury. *J Neurotrauma* 9(Suppl 1):1–7
15. Hall ED (1988) Effects of the 21-aminosteroid U74006F on posttraumatic spinal cord ischemia in cats. *J Neurosurg* 68:462–465
16. Hall ED (1995) Inhibition of lipid peroxidation in central nervous system trauma and ischemia. *J Neurol Sci* 134(Suppl):79–83
17. Lu J, Ashwell KW, Waite P (2000) Advances in secondary spinal cord injury: role of apoptosis. *Spine* 25:1859–1866
18. Muller U, Berlemann U, Sledge J et al (1999) Treatment of thoracolumbar burst fractures without neurological deficits by indirect reduction and posterior instrumentation: bisegmental stabilization with monosegmental fusion. *Eur Spine J* 8:284–289
19. Shen WJ, Liu TJ, Shen YS (2001) Nonoperative treatment versus posterior fixation for thoracolumbar junction burst fractures without neurologic deficit. *Spine* 26:1338–1345
20. Denis F, Armstrong GW, Searls K, Matta L (1984) Acute thoracolumbar burst fractures in the absence of neurologic deficit A comparison between operative and nonoperative treatment. *Clin Orthop* 189:142–149
21. Mumford J, Weinstein JN, Spratt KF, Goel VK (1993) Thoracolumbar burst fractures. The clinical efficacy and outcome of nonoperative management. *Spine* 18:955–970
22. Tator CH, Fehlings MG (1991) Review of the secondary injury theory of acute spinal cord trauma with emphasis on vascular mechanisms. *J Neurosurg* 75:15–26
23. Knight RQ, Stornelli DP, Chan DP, Devanny JR, Jackson KV (1993) Comparison of operative versus nonoperative treatment of lumbar burst fractures. *Clin Orthop* 293:112–121
24. Dall DE, Stauffer ES (1988) Neurologic injury and recovery patterns in burst fractures at the T12 or L1 motion segment. *Clin Orthop* 233:171–176
25. Carl AL, Tromanhauser SG, Roger DJ (1992) Pedicle screw instrumentation for thoracolumbar burst fractures and fracture-dislocations. *Spine* 17(Suppl 8):S317–324
26. Fan KF, Tu YK, Hsu RW et al (1997) The high fixation failure rate of short segment pedicle instrumentation for unstable thoracolumbar burst fractures. *Orthop Trans* 21:267
27. Domenicucci M, Preite R, Ramieri A et al (1996) Thoracolumbar fractures without neurological involvement: surgical or conservative treatment? *J Neurosurg Sci* 40:1–10
28. Seybold EA, Sweeny CA, Fredrickson BE et al (1999) Functional outcome of low lumbar burst fractures. A multicenter review of operative and nonoperative treatment from L3–L5. *Spine* 15:2154–2161
29. Vaccaro AR, Lehman RA Jr., Hurlbert RJ et al (2005) A new classification of thoracolumbar injuries: the importance of injury morphology, the integrity of the posterior ligamentous complex, and neurologic status. *Spine* 30(20):2325–2333
30. Yazici M, Atilla B, Tepe S, Calisir A (1996) Spinal canal remodeling in burst fractures of the thoracolumbar spine: a computerized tomographic comparison between operative and nonoperative treatment. *J Spinal Disord* 9:409–413
31. Tropiano P, Huang RC, Louis CA, Poitout DG et al (2003) Functional and radiographic outcome of thoracolumbar and lumbar burst fractures managed by closed orthopaedic reduction and casting. *Spine* 28(21):2459–2465
32. Chow GH, Nelson BJ, Gebhard JS, Brugman JL, Brown CW, Donaldson DH (1996) Functional outcome of thoracolumbar burst fractures managed with hyperextension casting or bracing and early mobilization. *Spine* 21:2170–2175
33. Cantor JB, Lebowitz NH, Garvey T, Eismont FJ (1993) Nonoperative management of stable thoracolumbar burst fractures with early ambulation and bracing. *Spine* 18:971–976
34. Chan DP, Seng NK, Kaan KT (1993) Nonoperative treatment in burst fractures of the lumbar spine (L2–L5) without neurologic deficits. *Spine* 18(3):320–325
35. Shen WJ, Shen YS (1999) Nonsurgical treatment of three-column thoracolumbar junction burst fractures without neurologic deficit. *Spine* 24:412–415
36. White AA, Panjabi MM (1978) Clinical biomechanics of the spine. Lippincott, Philadelphia
37. Celibi L, Muratli HH, Dogan O et al (2004) The efficacy of nonoperative treatment of burst fractures of the thoracolumbar vertebrae. *Acta Orthop Traumatol Turc* 38(1):16–22
38. Boerger TO, Limb D, Dickson RA (2000) Does ‘canal clearance’ affect neurological outcome after thoracolumbar burst fractures? *JBJS(Br)* 82-B:629–635
39. Herndon WA, Galloway D (1988) Neurologic return versus cross-sectional canal area in incomplete thoracolumbar spinal cord injuries. *J Trauma* 28:680–683
40. Mohanty SP, Venkatram N (2002) Does neurological recovery in thoracolumbar and lumbar burst fractures depend on the extent of canal compromise? *Spinal Cord* 40:295–299
41. Wessberg P, Wang Y, Irstam L, Nordwall A (2001) The effect of surgery and remodelling on spinal canal measurements after thoracolumbar burst fractures. *Eur Spine J* 10:55–63
42. Braakman R, Fontijne WPJ, Zeegers R, Steenbeek JR, Tanghe HLJ (1991) Neurological deficit in injuries of the thoracic and lumbar spine. *Acta Neurochir (Wien)* 111(1):1–17
43. Bohlman HH, Kirkpatrick JS, Delamarter RB, Leventhal M (1994) Anterior decompression for late pain and paralysis after fractures of the thoracolumbar spine. *Clin Orthop* 300:24–29
44. Dai LY (2001) Remodeling of the spinal canal after thoracolumbar burst fractures. *Clin Orthop* 382:119–123
45. McNamara MJ, Stephens GC, Spengler DM (1992) Transpedicular short-segment fusions for treatment of lumbar burst fractures. *J Spinal Disord* 5:183–187

46. Scapinelli R, Candiottio S (1995) Spontaneous remodelling of the spinal canal after burst fractures of the low thoracic and lumbar region. *J Spinal Disord* 8:486–493
47. Wilcox RK, Boerger TO, Allen DJ, Barton DC et al (2003) A dynamic study of thoracolumbar burst fractures. *J Bone Joint Surg* 85-A(11):2184–2189
48. Resch H, Rabl M, Klampfer H, Ritter E, Povacz P (2000) Surgical versus conservative treatment of fractures of the thoracolumbar transition. *Unfallchirurg* 103(4):281–288
49. Kramer DL, Rodgers WB, Mansfield FL (1995) Transpedicular instrumentation and short-segment fusion of thoracolumbar fractures: a prospective study using a single instrumentation system. *J Orthop Trauma* 9:499–506
50. Oda I, Cunningham BW, Buckley RA, Goebel MJ et al (1999) Does spinal kyphotic deformity influence the biomechanical characteristics of the adjacent motion segments? An in vivo animal model. *Spine* 24:2139–2146
51. Jacobs RMD, Asher M, Snider R (1980) Thoracolumbar spinal injuries. A comparative study of recumbent and operative treatment 100 patients. *Spine* 5:463–477
52. Bedbrook GM (1975) Treatment of thoracolumbar dislocation and fractures with paraplegia. *Clin Orthop Relat Res* 112:27–43
53. Patwardhan AG, Li SP, Gavin T, Lorenz M, Meade KP, Zindrick M (1990) Orthotic stabilization of thoracolumbar injuries. A biomechanical analysis of the Jewett hyperextension orthosis. *Spine* 15:654–661