SPINE Volume 23, Number 9, pp 1057–1060
©1998, Lippincott–Raven Publishers

# Spontaneous Remodeling of the Spinal Canal After Conservative Management of Thoracolumbar Burst Fractures

Luuk W. L. de Klerk, MD, PhD,* W. Peter J. Fontijne, MD, PhD,*
Theo Stijnen, MSc, PhD,§ Reiner Braakman, MD, PhD,‡
Herve L. J. Tanghe, MD,‡ and Bert van Linge, MD, PhD*

**Study Design.** Forty-two conservatively treated patients with a burst fracture of the thoracic, thoracolumbar, or lumbar spine with more than 25% stenosis of the spinal canal were reviewed more than 1 year after injury to investigate spontaneous remodeling of the spinal canal.

**Objectives.** To investigate the natural development of the changes in the spinal canal after thoracolumbar burst fractures.

**Summary of the Background Data.** Surgical removal of bony fragments from the spinal canal may restore the shape of the spinal canal after burst fractures. However, it was reported that restoration of the spinal canal does not affect the extent of neurologic recovery.

**Methods.** Using computerized tomography, the authors compared the least sagittal diameter of the spinal canal at the time of injury with the least sagittal diameter at the follow-up examination.

**Results.** Remodeling and reconstitution of the spinal canal takes place within the first 12 months after injury. The mean percentage of the sagittal diameter of the spinal canal was 50% of the normal diameter (50% stenosis) at the time of the fracture and 75% of the normal diameter (25% stenosis) at the follow-up examination. The correlation was positive between the increase in the sagittal diameter of the spinal canal and the initial percentage stenosis. There was a negative correlation between the increase in the sagittal diameter of the spinal canal and age at time of injury. Remodeling of the spinal canal was not influenced by the presence of a neurologic deficit.

**Conclusion.** Conservative management of thoracolumbar burst fractures is followed by a marked degree of spontaneous redevelopment of the deformed spinal canal. Therefore, this study provides a new argument in favor of the conservative management of thoracolumbar burst fractures. [Key words: spinal canal remodeling, thoracolumbar burst fractures] **Spine 1998;23:1057–1060**

From the Departments of *Orthopaedics, †Neurosurgery, ‡Radiology, §Epidemiology and Biostatistics, University Hospital and Erasmus University Rotterdam, The Netherlands.
Acknowledgment date: March 6, 1997.
First revision date: May 12, 1997.
Acceptance date: October 22, 1997.
Device status category: 1.

Surgical removal of bony fragments from the spinal canal may restore the shape of the canal after burst fractures. However, several reports have indicated that restoration of the spinal canal does not affect the extent of neurologic recovery.[3,9,17,25,27,29–31] The natural development of the changes in the spinal canal after burst fractures needs further definition.[6,7,13,19,24,26] 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 September 1990, 125 patients with a burst fracture of the thoracic, thoracolumbar, or lumbar spine were treated in the Departments of Orthopedics or Neurosurgery of the authors' hospital.

After the patients were admitted to the hospital, anteroposterior and lateral radiographs were obtained of all patients suspected of having a spinal injury. The spine of the patient suspected of having a burst fracture was stabilized with plaster shells, in the emergency room, before the patient was submitted to computed tomography. All patients with a burst fracture of the spinal column underwent a neurologic examination by a neurosurgeon in the emergency room. Within 24 hours and during the follow-up visit, a sagittal computed tomogram was made of all patients with a fracture of the spine. On admission and on follow-up examination, the window width was 3200 Hounsfield units, and the window level was 300 Hounsfield units. A slice thickness of 3 mm or 6 mm (four scans) was used.

The least midsagittal diameter of the spinal canal at the level of injury (x) was measured on the computed tomography scan. The normal midsagittal diameter of the spinal canal was estimated by calculating the average of the corresponding measurements at the adjacent uninjured level above and below the injury (y). The percentage spinal canal stenosis was calculated as: $(1 - x/y) \times 100\%$.[16]

Forty-two patients with an initial spinal canal stenosis of more than 25% underwent conservative treatment. The locations of the 42 burst fractures are shown in Table 1.

Sixteen patients (38%) had a neurologic deficit as defined by Frankel et al[15] (Table 2). There were 35 men and 7 women, with a mean age at the time of injury of 35.6 years (range, 17–82 years).

**Table 1. Numbers of Patients With a Burst Fracture With and Without a Neurologic Deficit at the Three Levels**

| | Thoracic (T1–T10) | Thoracolumbar (T11–L1) | Lumbar (L2–L5) | Mean (SD) Reduction | *P* Value |
|---|---|---|---|---|---|
| With deficit | 1 | 13 | 2 | 22.0 (14.8) | |
| Without deficit | 2 | 14 | 10 | 26.7 (12.7) | |
| *P* value | | | | NS | |
| Mean (SD) reduction | 18.3 (19.9) | 24.9 (12.9) | 28.6 (14.1) | | NS |

NS = not significant (one-way ANOVA + trend test).

Treatment of all patients was supervised by a neurosurgeon and an orthopedic surgeon. Both departments used the same treatment protocol. The conservative treatment included stabilization on a spinal surgy bed with removable plaster shells. After clinical stabilization, a circular plaster body cast from symphysis pubica to the sternoclavicular joints was made. Two months after injury, the patient, if neurologically well enough, was mobilized in an erect position in the plaster body cast with the help of a physical therapist. Four months after injury the plaster shell was removed.

The follow-up period varied from 12 months to 108 months (mean, 43.3 months).

## ■ Results

In all patients, except one with a thoracic burst fracture without the presence of a neurologic deficit, there was a reduction of the percentage spinal canal stenosis. Within the first 12 months after injury, the mean percentage spinal canal stenosis significantly decreased from 50 ± 15.4% to 25 ± 12.5% (paired *t* test, $P < 0.0001$). After this period there was no longer a significant correlation between the reduction in the percentage spinal canal stenosis and the length of the follow-up period (Figure 1).

The level of the fracture (thoracic, thoracolumbar, and lumbar) and the Frankel grading did not influence the redevelopment of the spinal canal significantly (Table 1). Eleven patients improved one or more Frankel grades (Table 2). None of the 42 patients was neurologically worse at the follow-up examination. Figure 2 shows the

**Table 2. The Number of Patients With a Neurological Deficit (n = 16) Classified According to Frankel on Admission and at Follow-up**

| | Frankel Classification at Follow-up | | | | |
|---|---|---|---|---|---|
| Initial Frankel Classification | A (n = 3) | B (n = 0) | C (n = 1) | D (n = 5) | E (n = 7) |
| A (n = 3) | 3 | 0 | | | |
| B (n = 2) | | | | 2 | |
| C (n = 4) | | | 1 | 2 | 1 |
| D (n = 7) | | | | 1 | 6 |

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.

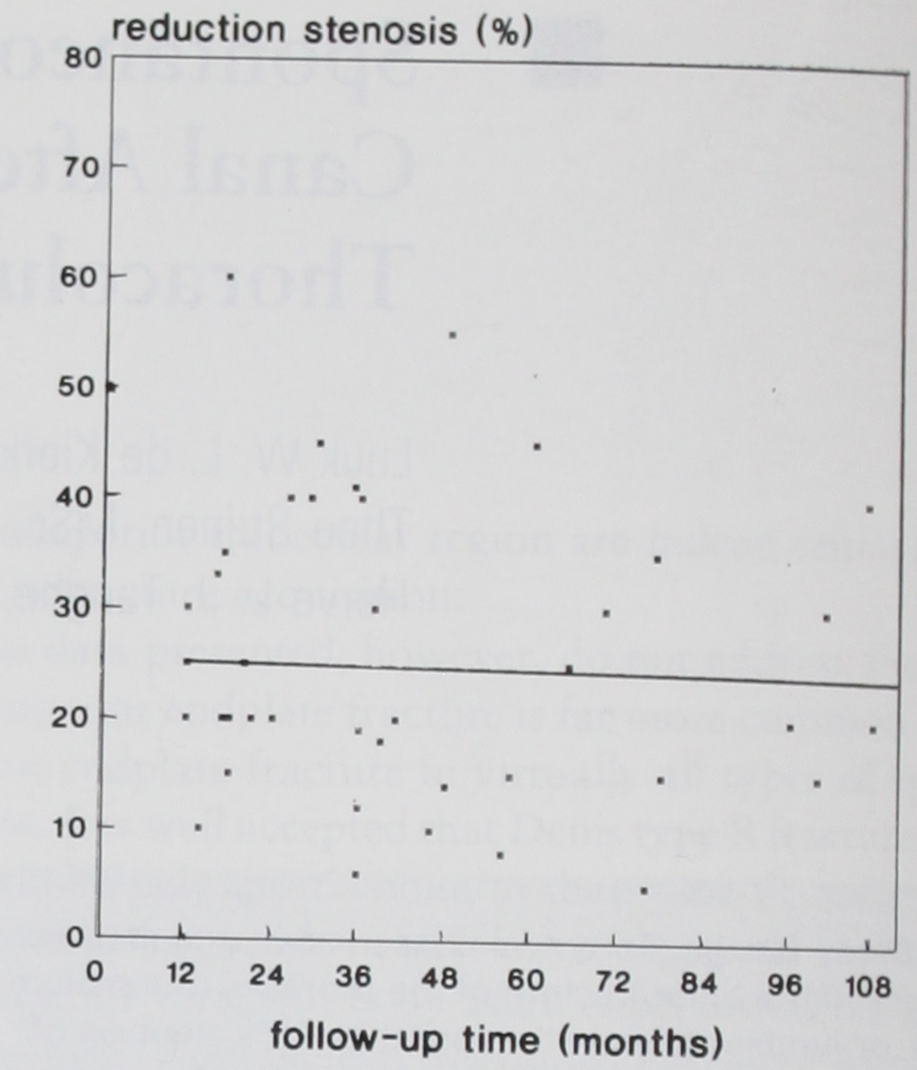


Figure 1. The reduction in the percentage stenosis *versus* the follow-up time in months. The reduction in percentage stenosis is the initial percentage stenosis minus the percentage stenosis at the the follow-up examination. *The mean initial percentage stenosis.

correlation between the reduction in the percentage spinal canal stenosis and age at the time of injury ($P < 0.001$). This correlation also was seen for each separate level of injury (thoracic, thoracolumbar, and lumbar).

The reduction in the percentage spinal canal stenosis was very significantly associated with a higher initial percentage spinal canal stenosis ($P < 0.0001$; Figure 3).

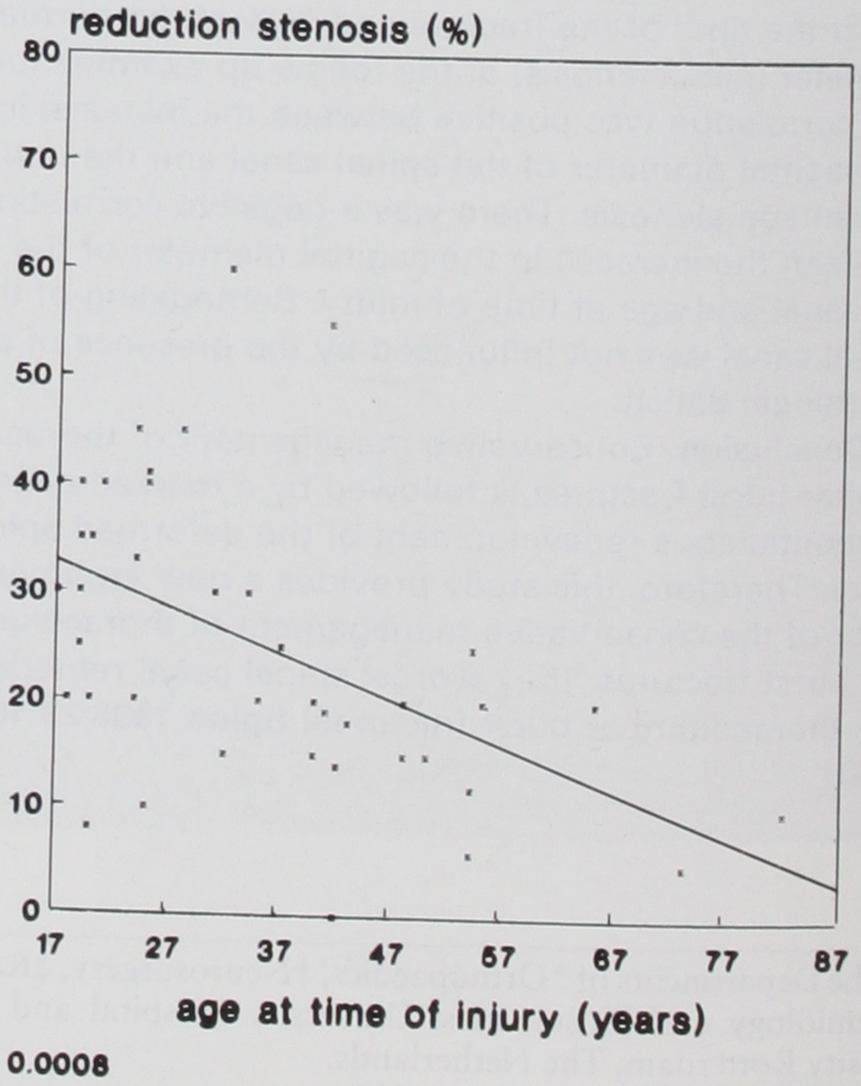


Figure 2. The reduction in the percentage stenosis *versus* the age at time of injury. The reduction is more evident in younger patients.

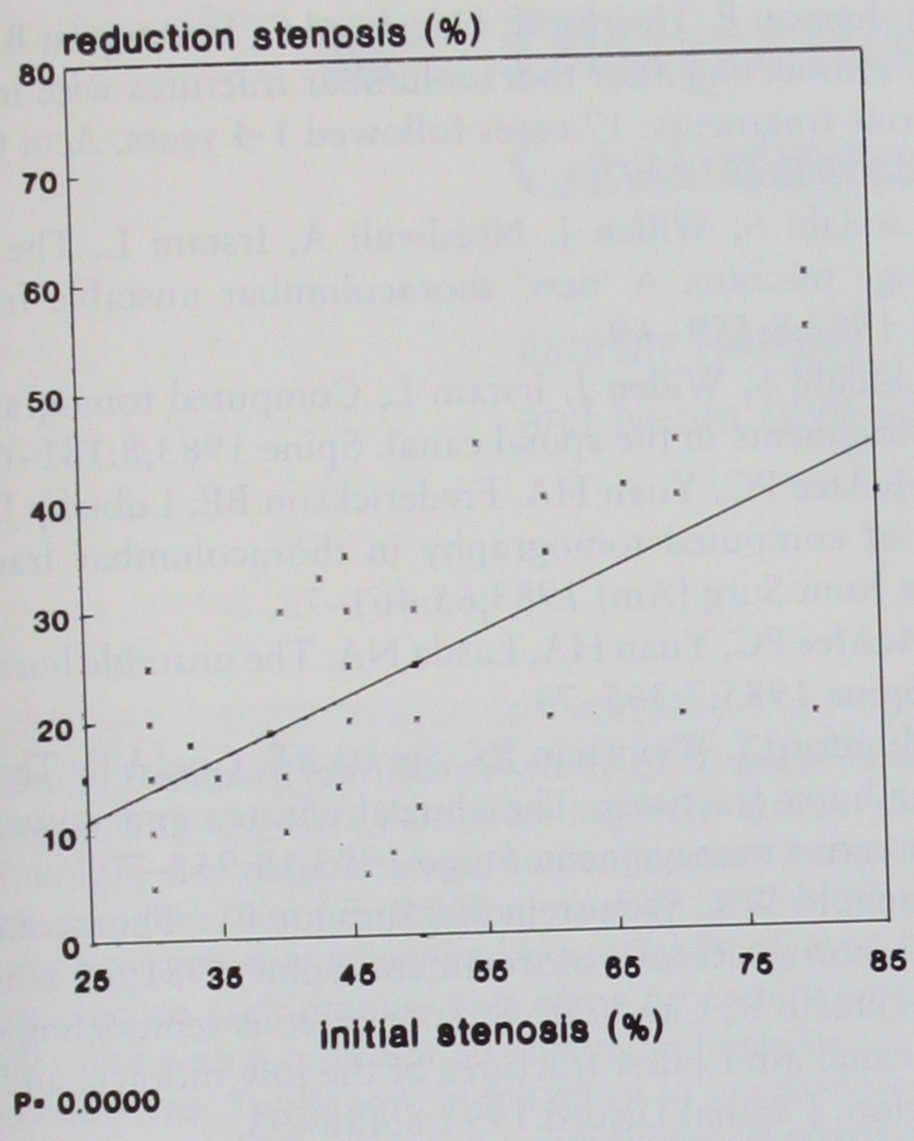


Figure 3. The reduction in the percentage stenosis *versus* the initial percentage stenosis. The higher the initial percentage stenosis, the greater the reduction.

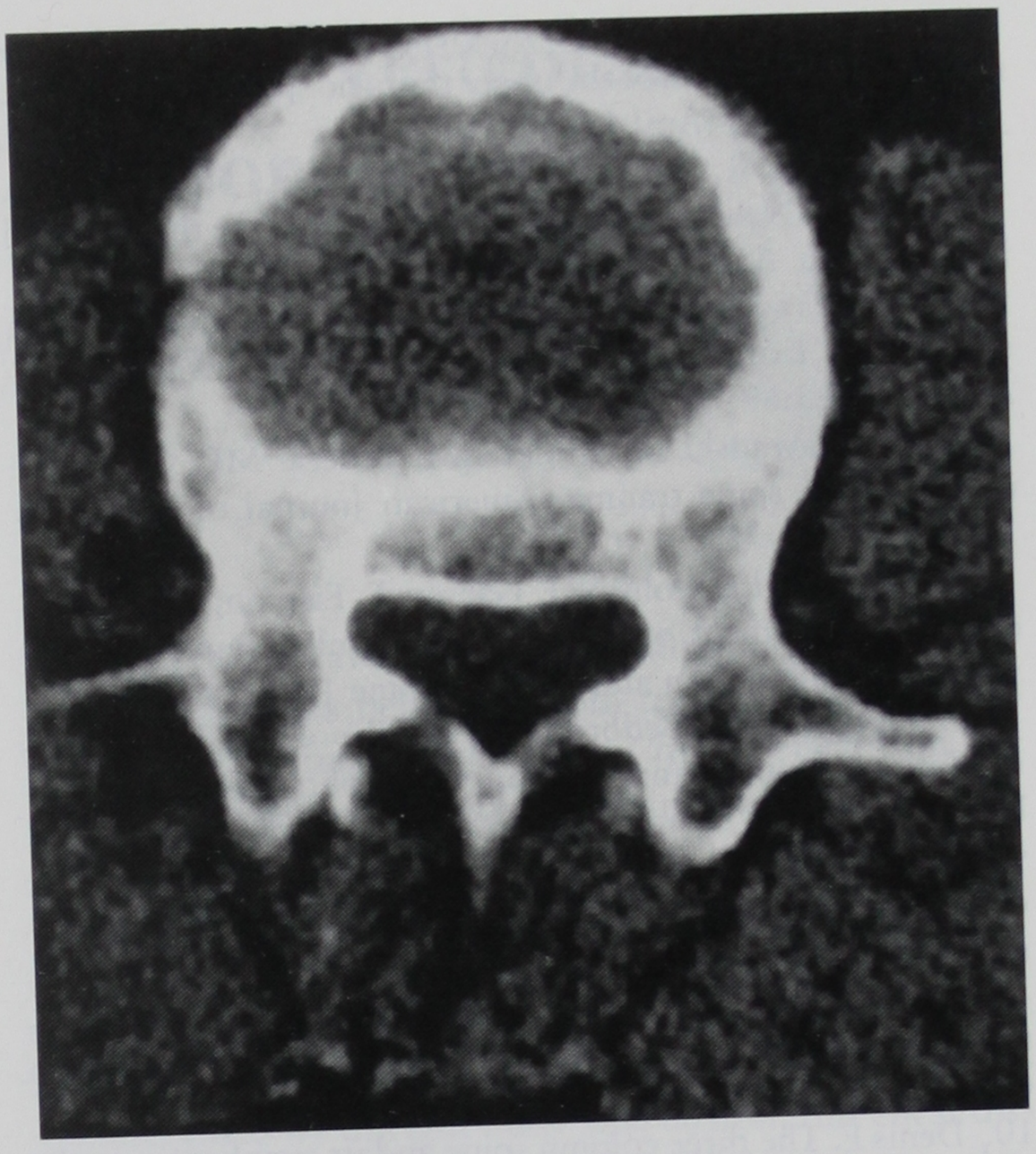

Figure 5. Computed tomography scan of the same burst fracture in Figure 4, 28 months after injury, at almost the same level (the least sagittal diameter).

## ■ Discussion

Excessive axial loading and flexion causes retropulsion of bone into the spinal canal.[10] Computed tomography scanning has increased our knowledge of spinal trauma. However, the computed tomograms also cause apprehension by clearly showing bony fragments in the spinal canal.[4,5,8,11,20–22,28] 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.[1,2,10,12,18,23] There have been reports about the redevelopment of the spinal canal in burst fractures.[6,7,13,19,24,26] Most of these reports concern only a small group of patients who have a burst fracture, but have no neurologic deficit. In the reports of studies involving a small number of patients, no adequate statistical analysis could be applied. In the current study, it is shown that there is a significant reduction in the stenosis and redevelopment of the spinal canal.

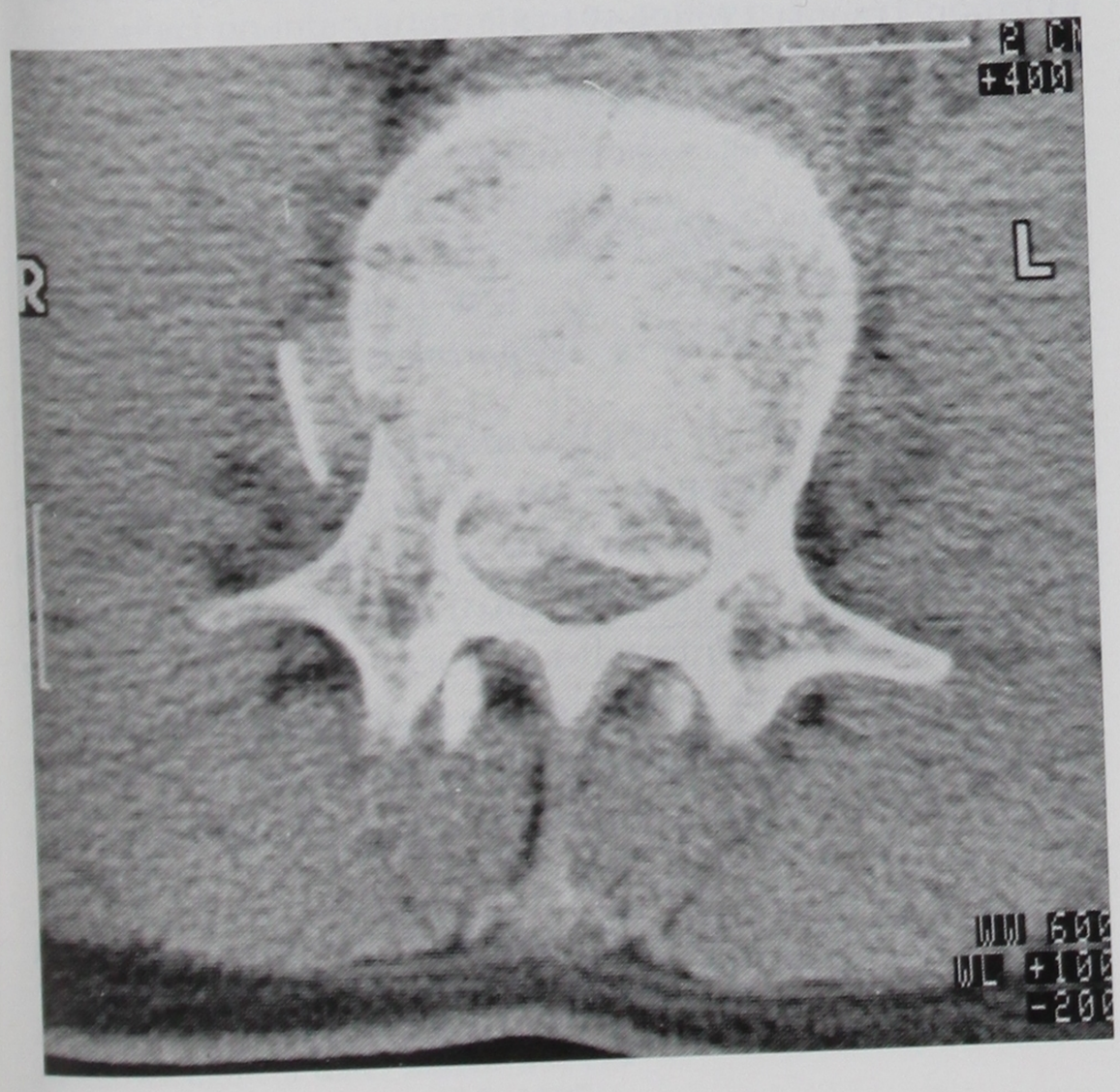

Figure 4. Computed tomography scan of a 33-year-old man with a burstfracture of L2, 6 hours after injury.

The process of remodeling takes place during the first year after injury; after this period there is no further significant reduction in the percentage spinal canal stenosis. The mechanism of the reduction in the spinal canal stenosis was not influenced by the presence or by the degree of neurologic deficit. The authors did not observe neurologic deterioration during the follow-up period.

Previous studies have found a correlation between the percentage spinal canal stenosis and the presence of a neurologic deficit, but no difference was found in neurologic recovery between conservatively and operatively treated patients with a burst fracture.[3,14,16,28]

In this study, a significant spontaneous reduction in spinal canal stenosis was found in patients with a burst fracture. (Figures 4 and 5) Conservative management of thoracolumbar burst fractures is followed by a marked degree of spontaneous redevelopment of the deformed spinal canal.

## References

1. Bohlman HH, Freehafer A. Late anterior decompression of spinal cord injuries. J Bone Joint Surg [Am] 1975;57:1025–8.

2. Bohlman HH, Freehafer A, Dejak K. The results of treatment of acute injuries of the upper thoracic spine wih paralysis. J Bone Joint Surg [Am] 1985;67:360–9.
3. Braakman R, Fontijne WPJ, Zeegers R, Steenbeek JR, Tanghe HLJ. Neurological deficit in injuries of the thoracic and lumbar spine. Acta Neurochir (Wien) 1991;111:11–7.
4. Brant-Zawadski M, Jeffrey B Jr, Minagi H, Pitts LH. High resolution CT of thoracolumbar fractures. AJNR Am J Neuroradiol 1982;3:69–74.
5. Brant-Zawadski M, Miller EM, Federle MP. CT in the evaluation of spine trauma. American Journal of Radiology 1981;136:369–75.
6. Cantor JB, Lebwohl NH, Garvey T, Eismont FJ. Nonoperative management of stable thoracolumbar burst fractures with early ambulation and bracing. Spine 1993;18:971–6.
7. Chakera TMH, Bedbrook G, Bradley CM. Spontaneous resolution of spinal canal deformity after burst dispersion fracture. AJNR Am J Neuroradiol 1988;9:779–85.
8. Colley DP, Dunsker SB. Traumatic narrowing of the dorso-lumbar spinal canal demonstrated by computed tomography. Radiology 1978;129:95–8.
9. Dall DE, Stauffer ES. Neurologic injury and recovery patterns in burst fractures at the T12 or L1 motion segment. Clin Orthop 1988;233:171–6.
10. Denis F. The three column spine and its significance in the classification of acute thoracolumbar spinal injuries. Spine 1983;8:817–31.
11. Dewald RL. Burstfractures of the thoracic and lumbar spine. Clin Orthop 1984;189:160–1.
12. Dickson JH, Harrington PR, Erwin WD. Results of reduction and stabilization of the severely fractured thoracic and lumbar spine. J Bone Joint Surg [Am] 1978;60:799–805.
13. Fidler MW. Remodeling of the spinal canal after burstfracture. A prospective study of two cases. J Bone Joint Surg [Br] 1988;70:730–2.
14. Fontijne WPJ, Klerk de LWL, Braakman R, et al. CT scan prediction of neurological deficit in thoracolumbar burstfractures. J Bone Joint Surg [Br] 1992;74:683–5.
15. Frankel HL, Hancock DO, Hyslop G, et al. The value of postural reduction in the initial management of enclosed injuries of the spine with paraplegia and tetraplegia. Paraplegia 1969;7:179–92.
16. Hashimoto T, Kaneda K, Abumi K. Relationship between traumatic spinal canal stenosis and neurologic deficits in thoracolumbar burstfractures. Spine 1988;13:1268–72.
17. Herndon WA, Galloway D. Neurologic return versus cross-sectional canal area in incomplete thoracolumbar spinal cord injuries. J Trauma 1988;28:680–3.
18. Jacobs RR, Asher MA, Snider RK. Thoracolumbar spinal injuries: A comparative study of recumbent and operative treatment in 100 patients. Spine 1980;5:463–77.
19. Johnsson R, Herrlin K, Hägglund G, Strömqvist B. Spinal canal remodeling after thoracolumbar fractures with intraspinal bone fragments: 17 cases followed 1-4 years. Acta Orthop Scand 1991;62:125–7.
20. Lindahl S, Willen J, Nordwall A, Irstam L. The crush-cleavage fracture: A 'new' thoracolumbar unstable fracture. Spine 1983;8:559–69.
21. Lindahl S, Willen J, Irstam L. Computed tomography of bone fragments in the spinal canal. Spine 1983;8:181–6.
22. McAfee PC, Yuan HA, Frederickson BE, Lubicky JP. The value of computed tomography in thoracolumbar fractures. J Bone Joint Surg [Am] 1983;65:461–73.
23. McAfee PC, Yuan HA, Lasda NA. The unstable burst fracture. Spine 1985;7:365–73.
24. Mumford J, Weinstein JN, Spratt KF, Goel VK. Thoracolumbar burst fractures: The clinical efficacy and outcome of nonoperative management. Spine 1993;18:955–70.
25. Osebold WR, Weinstein SL, Sprague BL. Thoracolumbar spine fractures. Results of treatment. Spine 1981;6:13–34.
26. Scapinelli R, Candiotto S. Spontaneous remodeling of the spinal canal after burst fractures of the low thoracic and lumbar region. J Spinal Disord 1995;8:486–93.
27. Sjostrom L, Jacobson O, Karlstrom G, Pech P, Rauschning W. Spinal canal remodeling after stabilization of thoracolumbar burst fractures. Eur Spine J 1994;3:312–7.
28. Trafton PG, Boyd CA. Computed tomography of thoracic and lumbar spine injuries. J Trauma 1984;24:506–15.
29. Vornanen MJ, Bostman OM, Myllynen PJ. Reduction of bone retropulsed into the spinal canal in thoracolumbar vertebral body compression burst fractures: A prospective randomized comparative study between Harrington rods and two transpedicular devices. Spine 1995;20:1699–703.
30. Willen J, Lindahl S, Nordwall A. Unstable thoracolumbar fractures: A comparative clinical study of conservative treatment and Harrington instrumentation. Spine 1985;10:111–22.
31. Yazici M, Gulman B, Sen S, Tilki K. Saggital contour restoration and canal clearance in burst fractures of the thoracolumbar junction (T12–L1): The efficacy of timing of surgery. J Orthop Trauma 1995;9:491–8.

*Address reprint requests to*

L.W.L de Klerk, MD, Phd
*Department of Orthopaedics*
*University Hospital*
*Dr. Molewaterplein 40*
*3015 GD Rotterdam*
*The Netherlands*