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Dural Laceration Occurring with Burst Fractures and Associated Laminar Fractures*

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ABSTRACT: The cases of sixty patients in whom a burst fracture of a thoracic or lumbar vertebral body had been treated with posterior instrumentation and arthrodesis less than two weeks after the injury were retrospectively reviewed. Thirty of the patients had an associated laminar fracture. Eleven of the thirty, all of whom had a lumbar fracture and a preoperative neurological deficit, were noted at operation to have dural laceration. In four of the patients who had dural laceration, neural elements were entrapped between the fragments from the laminar fracture. None of the remaining thirty patients who did not have a laminar fracture had dural laceration ($p = 0.0002$). Univariate and multivariate statistical analysis revealed no significant association of the dural laceration with the patients' age or sex, or with the radiographic characteristics of the spine. There was a significant association between dural laceration and neurological deficit ($p = 0.0001$).

In our series, the presence of a preoperative neurological deficit in a patient who had a burst fracture and an associated laminar fracture was a sensitive (100 per cent) and specific (74 per cent) predictor of dural laceration. The presence of this fracture pattern and an associated neurological deficit also predicted a risk of dural laceration with entrapped neural elements. This information may influence decisions as to whether an anterior or a posterior surgical approach should be used in such patients.

The incidence of dural laceration in patients who have a burst fracture and associated laminar fracture has not been well documented in the orthopaedic literature. Denis did note a "vertical laminar fracture" that was secondary to splaying of the posterior arch of the vertebra under axial loading, which he described as a "greenstick fracture of the

anterior cortex of the lamina". He did not, however, discuss the incidence of this injury. In his review, thirty of fifty-nine patients who had a burst fracture were seen to have no dural tear at operation, although twenty-eight of the fifty-nine did have a neurological deficit. McAfee et al.¹⁵ noted that eleven of sixteen patients who had an unstable burst fracture had "nondisplaced fractures of the posterior elements". None of their patients was noted to have a dural tear secondary to the injury, although twelve patients had a preoperative neurological deficit.

In the neurosurgical literature, Miller et al.^{16,17} accurately described lumbar vertebral burst injuries associated with laminar fracture and dural laceration. They noted that neural elements were often entrapped between the fracture fragments of the lamina. Eighteen of their nineteen patients who had this fracture pattern had dural laceration and an associated neurological deficit.

This retrospective review was undertaken to determine the incidence of dural laceration in patients who had a thoracic or lumbar burst fracture with associated laminar fracture. In addition, we wanted to determine whether specific clinical and radiographic factors, such as a neurological deficit, interpedicular spreading, the degree of compromise of the spinal canal, and the percentage of vertebral compression, were predictive of dural laceration.

Materials and Methods

The hospital records and radiographs of sixty patients who had been treated for a thoracic or lumbar burst fracture at the University of Miami/Jackson Memorial Hospital Medical Center between July 1, 1984, and February 29, 1988, were reviewed. All of the patients had been operated on through a posterior approach within two weeks after injury, to realign the spine or decompress nerves, or both. All patients were treated with posterior spinal instrumentation. During this period, the treatment protocol included intraoperative spinal sonography to assess the patency of the spinal canal, necessitating laminotomy at the level of injury. The laminotomy permitted visualization of the posterior part of the dura, making it possible to determine the presence or absence of posterior dural laceration.

There were forty-seven male and thirteen female pa-

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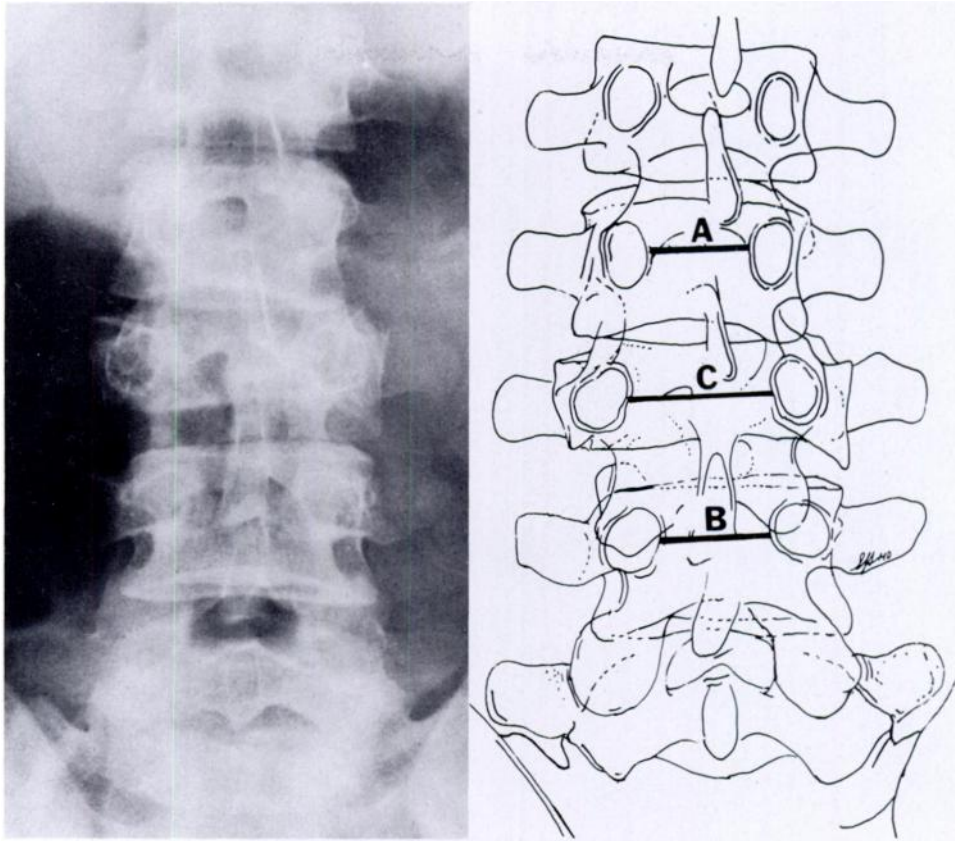


FIG. 1-A

Figs. 1-A and 1-B: Burst fracture of the third lumbar vertebra.

Fig. 1-A: The interpedicular distance was measured between the pedicles on an anteroposterior radiograph and expressed as a percentage increase compared with the mean interpedicular distance of the two adjacent vertebrae ($C [A + B/2] \times 100 =$ percentage increase). Note the increased interpedicular distance of the third lumbar vertebra compared with that of the second and fourth lumbar vertebrae.

tients, and the mean age was thirty-four years and seven months (range, fourteen to seventy years). The preoperative neurological status, including motor and sensory function and control of the bowel and bladder, was determined from the records of each patient. Each patient had had anteroposterior and lateral radiographs; computed tomography; and, when available, magnetic resonance imaging. The extent of osseous vertebral damage was ascertained by determining the interpedicular distance (Fig. 1-A), the height of the vertebral body (Fig. 1-B), the grade of compromise of the canal (Figs. 2-A, 2-B, and 2-C), and the type of laminar fracture (Figs. 3-A, 3-B, and 3-C).

Statistical analysis was done with both univariate and multivariate methods. For univariate analysis, the Fisher exact test was used for categorical data and the Student *t* test, for continuous data. An unconditional logistic regression, using the method of maximum likelihood ratio, modeled the predicted probability of dural laceration as a function of several covariates simultaneously.

Results

Thirty of the sixty patients who sustained a vertebral burst fracture did not have an associated laminar fracture, and no dural lacerations were identified in them at operation. This was significant ($p = 0.0002$) and indicated that pos-

terior dural lacerations occurred only if a laminar fracture was present.

The remaining thirty patients who had a burst fracture had an associated laminar fracture (Table I). Twenty-six of them were male and four were female, and the mean age was thirty-one years and four months (range, fourteen to sixty-one years). Only five of the burst fractures were in the thoracic spine; the remainder were in the lumbar spine. Three patients had a fracture at two levels. One had involvement of the first and second lumbar levels; another, of the first and fifth lumbar levels; and the third, of the fourth and fifth lumbar levels. Three patients had multiple laminar fractures. One had burst fractures of the first and fifth lumbar vertebrae and associated laminar fractures of the first, fourth, and fifth lumbar vertebrae; another had both burst fractures and associated laminar fractures of the first and second lumbar vertebrae; and the third had a burst fracture of the fourth lumbar vertebra and laminar fractures of the third and fourth lumbar vertebrae. Twenty-nine laminar fractures occurred at the same level as the burst injury, while five occurred one level cephalad to it.

The mean interpedicular distance in the vertebra that had the burst injury was increased by 25 per cent (range, 3 to 54 per cent; standard deviation, 11 per cent). The mean height of the vertebral body was decreased by 47 per cent

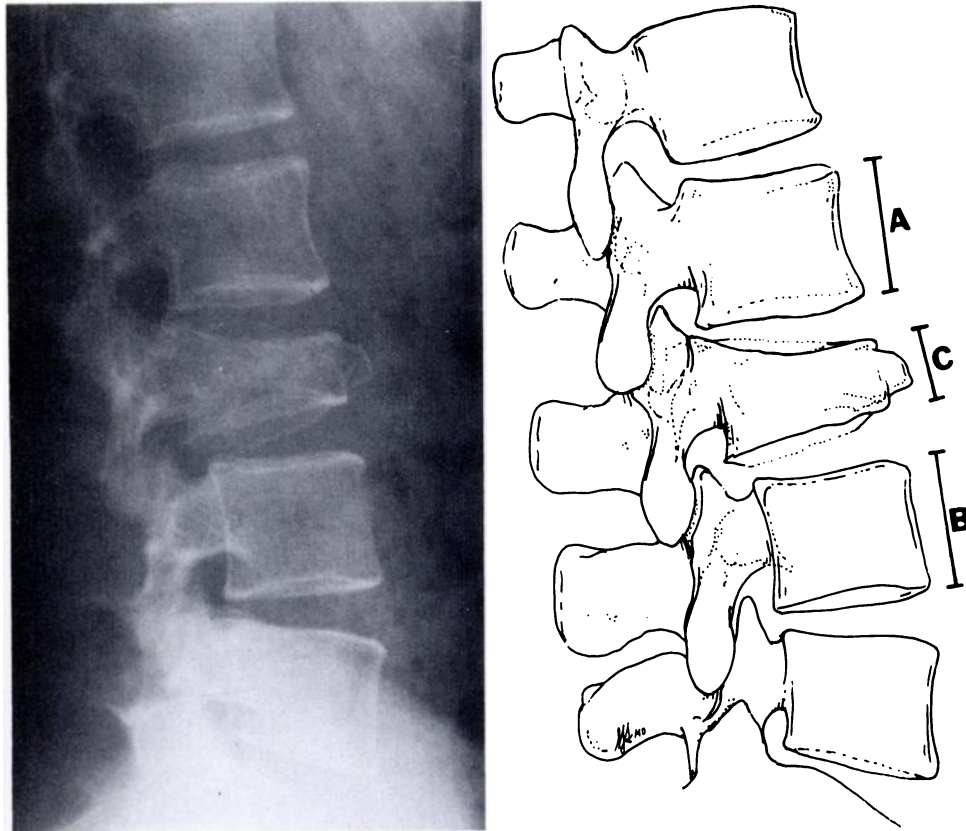


FIG. 1-B

The height of the vertebral body was measured as the distance between the superior and inferior end-plates at the anterior border of the vertebral body on a lateral radiograph and was expressed as a percentage decrease compared with the mean height of the two adjacent vertebrae ($C [A + B/2] \times 100 =$ percentage decrease). Note the decreased height of the third lumbar vertebra compared with that of the second and fourth lumbar vertebrae.

(range, 0 to 73 per cent; standard deviation, 13 per cent). Grade-I compromise of the canal was found in association with six fractures; Grade II, with fourteen; and Grade III, with thirteen. There were fifteen Type-I, fourteen Type-II, and five Type-III laminar fractures.

Sixteen (53 per cent) of the thirty patients had a neurological deficit, and fourteen (46 per cent) were neurologically intact. One patient had complete paraplegia secondary to a fracture of the eleventh thoracic vertebra; eleven patients

had incomplete injury of either the conus medullaris or of the cauda equina, or both; and four patients had isolated dysfunction of the bowel and bladder.

Eleven (69 per cent) of the sixteen patients who had a neurological deficit had dural laceration, all in association with lumbar fractures. In four (36 per cent) of these eleven patients, neural elements protruded and were entrapped between the fracture fragments of the lamina. Although no anterior dural lacerations were observed in our patients, their

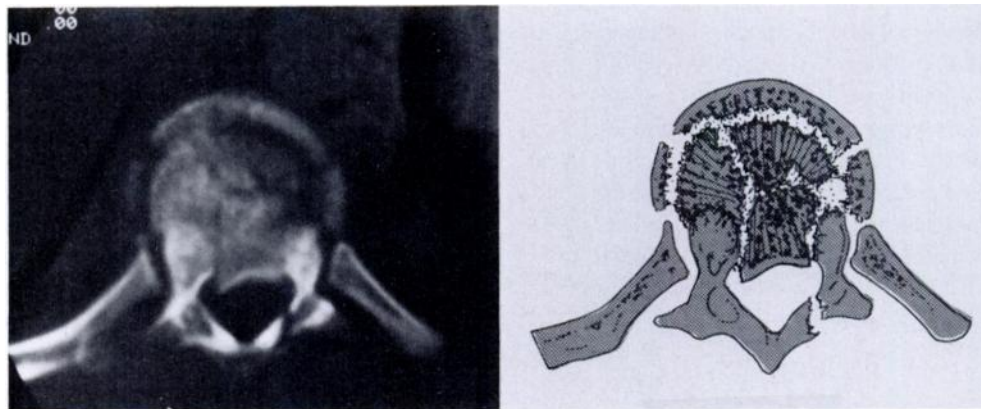


FIG. 2-A

Figs. 2-A, 2-B, and 2-C: The percentage of compromise of the spinal canal was noted on transverse computerized-tomography images and was classified into three grades.

Fig. 2-A: Grade I referred to a 0 to 35 per cent compromise of the canal.

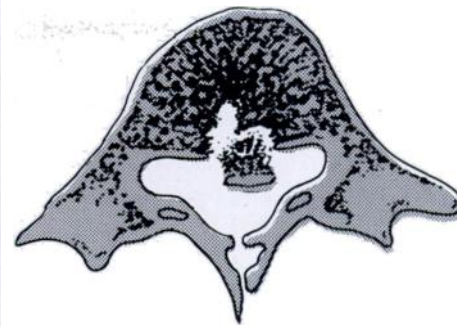
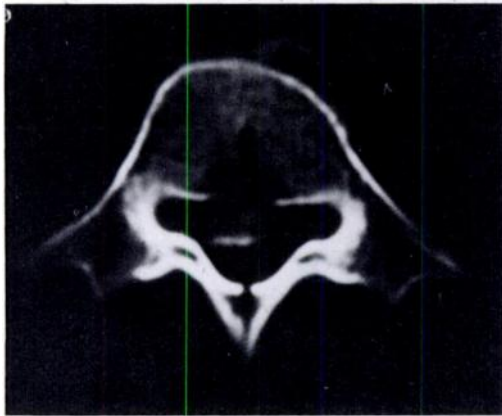


FIG. 2-B

Grade II referred to a 36 to 70 per cent compromise of the canal.

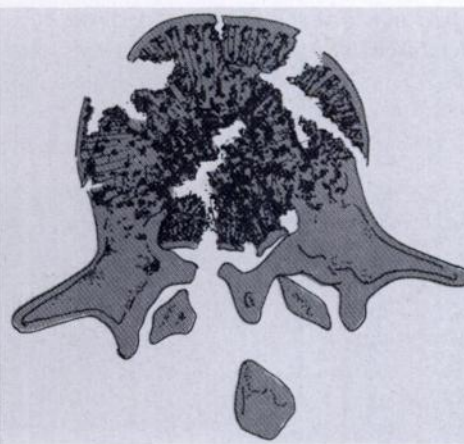


FIG. 2-C

Grade III referred to a 71 to 100 per cent compromise of the canal.



FIG. 3-A



FIG. 3-B

Figs. 3-A, 3-B, and 3-C: Laminar fractures were noted on transverse computerized-tomography images and were classified into three types.
Fig. 3-A: Type-I laminar fractures occurred through the midline of the spinous process.
Fig. 3-B: Type-II laminar fractures occurred off the midline, without involvement of the spinous process.

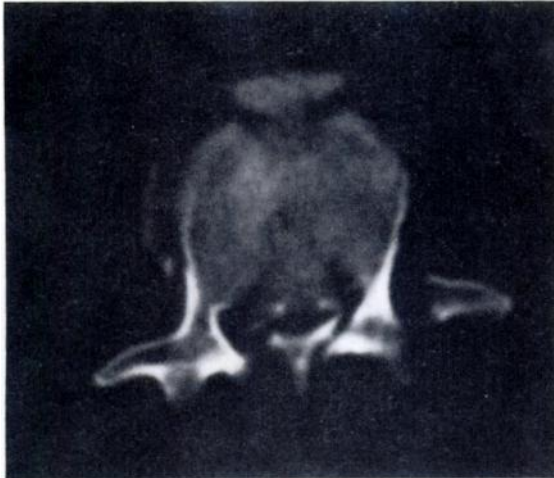


FIG. 3-C

Type-III laminar fractures were comminuted.

absence cannot be absolutely verified, as only posterior surgical exposures were used. The remaining five patients who had a neurological deficit did not have an identifiable dural tear.

Thus, sixteen (53 per cent) of the thirty patients who

had both a burst fracture of the vertebral body and laminar fracture had some degree of neurological deficit. If a dural laceration was present, the incidence of neurological deficit was 100 per cent.

Dural laceration was statistically associated with neurological compromise ($p = 0.0001$). All of the eleven dural lacerations occurred in the lumbar spine. None were seen in patients who had a thoracic burst fracture and associated laminar fracture ($p = 0.0001$). Dural laceration was not significantly associated with age, sex, interpedicular distance, percentage of compression of the vertebral body, grade of compromise of the spinal canal, or type of laminar fracture. There was no statistical significance regarding the level of the fracture in the lumbar spine.

A preoperative neurological deficit in the presence of a burst fracture and an associated laminar fracture was a sensitive (100 per cent) and specific (74 per cent) indicator of dural laceration. The specificity increased to 86 per cent if only the lumbar fractures were considered. Therefore, the false-positive rate for the presence of a dural laceration was 14 per cent in patients who had a lumbar burst fracture, an associated laminar fracture, and neurological compromise.

A multiple logistic regression model was developed to

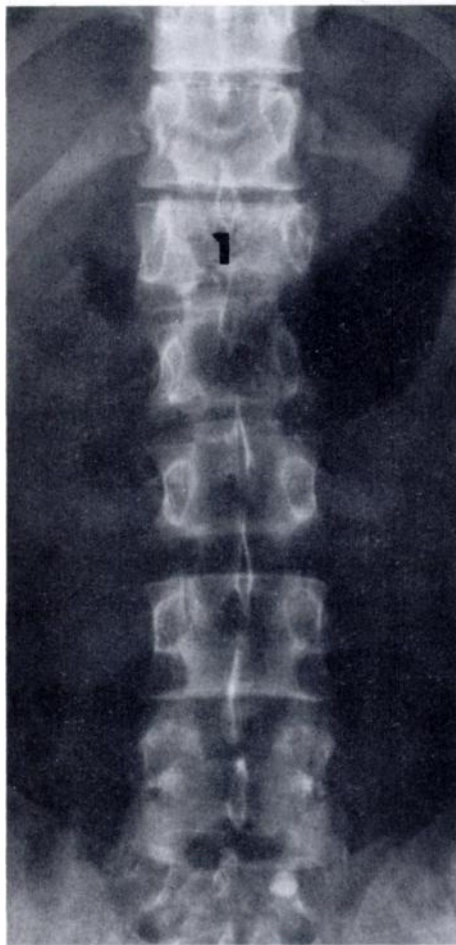


FIG. 4-A

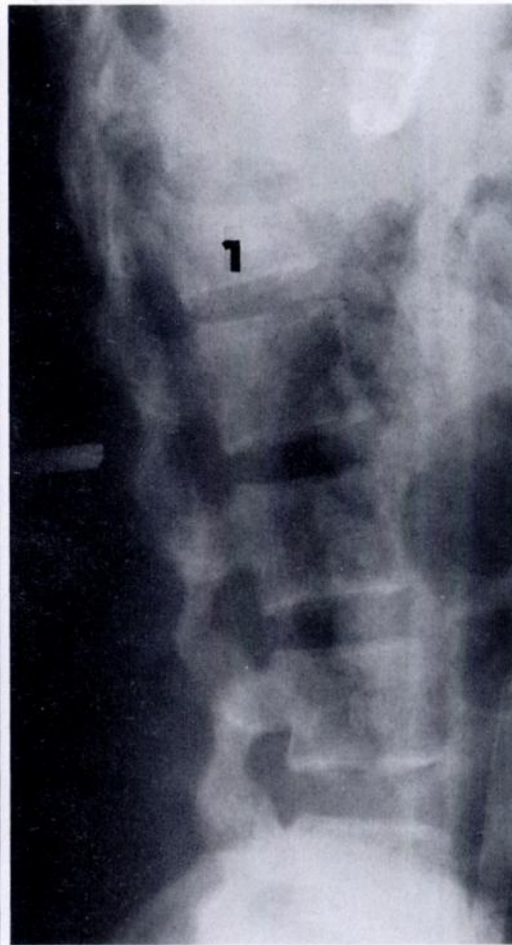


FIG. 4-B

Figs. 4-A and 4-B: Case 26. This patient had a burst fracture of the first lumbar vertebra and incomplete paraplegia.

Fig. 4-A: Anteroposterior radiograph.

Fig. 4-B: Lateral radiograph.

TABLE I
DATA ON THE PATIENTS WHO HAD ASSOCIATED LAMINAR FRACTURE

Case	Sex	Age (Yrs.)	Date of Operation	Level of Injury	Neurological Status*	Grade of Compromise of Canal†	Laminar Fracture (Level-Type‡)	Dural Laceration
1	M	17	11/84	L1	Intact	I	L1-I	-
2	M	20	3/85	L1	IP	III	T12-III	-
3	M	22	3/85	L3	IP	III	L3-II	+
4	M	37	3/85	T12	B/B	II	T12-III	-
5	M	21	3/85	L2	IP	III	L2-I	-, L3 nerve-root laceration
6	M	22	3/85	T11	CP	II	T11-II	-
7	F	21	9/85	L1	IP	II	L1-III	+, 2 distinct dural lacerations, with conus laceration
8	F	27	1/86	L1	Intact	II	L1-II	-
9	M	51	2/86	L2	Intact	II	L2-I	-
10	M	26	5/86	L2	IP	III	L2-II	+
11	M	61	7/86	L1	IP	II	L1-I	+
12	M	24	10/86	L3	Intact	III	L3-I	-
13	M	32	10/86	T12	Intact	I	T11-II	-
14	M	49	2/87	L1	Intact	II	L1-I	-
15	M	31	4/87	L1	Intact	I	L1-III	-
16	M	36	7/87	L1, L5	B/B	III	L1-II, L4-III, L5-III	+
17	M	23	7/87	L1, L2	IP	II	L1-I, L2-I	+, 2 distinct lacerations with entrapped rootlets
18	F	14	2/87	L4	Intact	III	L3-II L4-I	-
19	M	19	8/87	L1	B/B	II	L1-III	+, entrapped rootlets
20	M	28	8/87	L4, L5	IP	III	L4-I	+, entrapped rootlets
21	M	48	9/87	L1	Intact	II	L1-I	-
22	M	26	9/87	L2	B/B	III	L2-I	+, poliomyelitis
23	M	52	10/87	L3	Intact	II	L3-I	-
24	M	57	10/87	L1	IP	II	T12-II	+, laceration extended over 2 levels
25	M	35	10/87	T12	IP	I	T12-II	-
26	M	26	11/87	L1	IP	III	L1-II	+, entrapped nerve rootlets
27	M	24	11/87	L3	Intact	II	L3-III	-
28	F	16	12/87	L5	Intact	III	L5-I	-
29	M	18	2/88	T10	Intact	I	T10-II	-
30	M	58	2/88	L1	Intact	I	L1-I	-

* CP = complete paraplegia, IP = incomplete paraplegia, and B/B = bowel-bladder dysfunction only.

† I = 0 to 35 per cent, II = 36 to 70 per cent, and III = 71 to 100 per cent.

‡ I = midline fracture, involving the spinous process, II = adjacent to the spinous process, and III = comminuted.

examine the simultaneous effect of all of the previously mentioned potential predictors on the probability of dural laceration. Again, a preoperative neurological deficit in association with a lumbar burst fracture and a laminar fracture was predictive of dural laceration, with the probability being as high as 80 per cent. Conversely, the probability of a patient who has a lumbar fracture and a normal neurological examination having a laceration of the dura could be as low as 10 per cent.

Illustrative Case Report

CASE 26. A twenty-six-year-old man fell from a height while performing construction work in November 1987. He was transferred to our institution four days after injury. Preoperatively, he had motor and sensory changes in the right lower extremity, consisting of decreased sensation to light touch and pinprick in the fifth lumbar and first sacral dermatomes and weakness (grade 2 of 5) of the quadriceps, tibialis anterior, hamstrings,

gastrocnemius-soleus, and abductors of the hip. He had no function of the bowel or bladder.

Anteroposterior and lateral radiographs, computerized tomographic scans, and magnetic resonance images (Figs. 4-A through 4-D) revealed Grade-III (Fig. 2-C) compromise of the spinal canal and a Type-II laminar fracture. At operation, a dural tear was found, and there were entrapped nerve rootlets between the laminar fracture fragments (Fig. 4-E). The rootlets were freed from the fracture site, and the dura was repaired using previously described methods⁶. Posterolateral decompression of the canal was done from an approach through the right pedicle, and the decompression was documented by intraoperative spinal sonography. An arthrodesis with Harrington instrumentation was done from the tenth thoracic to the third lumbar vertebra (Fig. 4-F).

Five months postoperatively, the patient had occasional pain in the posterior aspect of the right calf, intact sensory function, no function of the bowel or bladder, and normal sexual function. Motor examination showed improvement in the strength of the quadriceps (grade 4 of 5), tibialis anterior (grade 5), hamstrings (grade 3+), gastrocnemius-soleus (grade 3), and abductors of the hip (grade 3).



FIG. 4-C

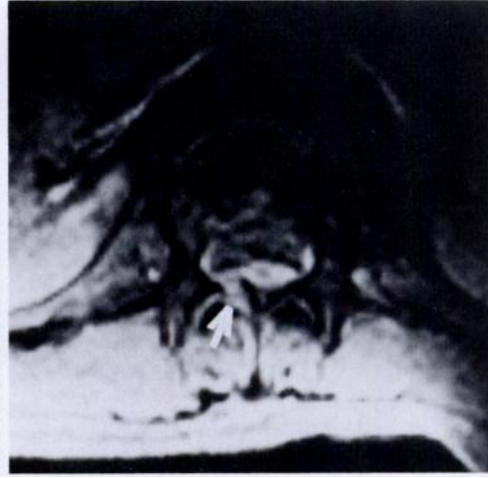


FIG. 4-D

Fig. 4-C: A computerized tomography scan shows Grade-III compromise of the canal with Type-II laminar fracture.

Fig. 4-D: A magnetic resonance image shows the Grade-III compromise of the canal and laminar fracture. It is also suggestive of a dural tear and a leak of cerebrospinal fluid, since the signal intensity behind the lamina (arrow) is the same as the signal intensity in the spinal canal.

The case of this patient suggests that, in the future, it may be possible to establish the presence of dural laceration preoperatively, using magnetic resonance imaging. The T2-weighted magnetic-resonance image (Fig. 4-D) was interpreted as showing a leak of cerebrospinal fluid, secondary to dural laceration, as the signal posterior to the lamina was of the same intensity as that in the dural sac.

Discussion

Dural laceration in patients who had a fracture of the lumbar spine secondary to an axial loading injury has been described in the neurosurgical literature^{16,17}, but it has not

been emphasized in the orthopaedic literature. In the neurosurgical literature, all of the dural lacerations were seen in conjunction with laminar fractures. Miller et al.¹⁷ reported on nineteen patients who had what they called an impaction fracture of the lumbar spine as a result of primary axial loading and secondary hyperextension from lumbar lordosis. Each patient had a disruption of the middle column of the spine with lateral displacement of the pedicles. All but one had an associated dural laceration. In eight patients, neural elements were trapped between the fracture fragments and there was an associated neurological deficit.

Considering the high incidence of dural laceration that



FIG. 4-E

Intraoperative photograph (right side, cephalad; left side, caudad). The inferior half of the lamina of the first lumbar vertebra remains (between curved black arrows). The inferior half of the lamina of the twelfth thoracic vertebra and the superior half of the first lumbar lamina have been removed, as have the interspinous ligament and ligamentum flavum at the junction of the first and second lumbar laminae. The forceps are holding frayed nerve rootlets that have extruded through the dura and are still entrapped within the laminar fracture. The edges of the torn dura (open arrows) are being held in place with retention sutures to facilitate dissection of the nerve rootlets and later repair.



FIG. 4-F

Lateral radiograph showing posterior instrumentation and fusion from the tenth thoracic to the third lumbar vertebra, using Harrington distraction instrumentation, Edwards hooks and sleeves, and sublaminar wiring.

was described by Miller et al., we wondered why this injury had apparently not been reported in the orthopaedic literature — particularly when many series included a large number of patients^{3,4,8-10,12,15,19}. One explanation might be that, in orthopaedic treatment, the emphasis has been placed on reduction and stabilization of the fracture. Exploration of the neural elements has not been encouraged because laminectomy further destabilizes the fractured spine and, before the introduction of adequate spinal instrumentation, had been associated with poor results¹⁸.

The clinical importance of the dural lacerations in our patients must be questioned. By itself, a laceration of the dura does not present appreciable problems, since the complications of leakage of cerebrospinal fluid (such as the development of either a dural-cutaneous fistula or a pseudomeningocele, or both) have not been documented as being more frequent in such patients. However, a dural laceration does indicate possible entrapment of neural elements between laminar fracture fragments. Such entrapment may delay, if not totally preclude, neurological recovery.

Of the thirty patients in our series who had a burst fracture of the vertebral body and a laminar fracture, sixteen (53 per cent) had a neurological deficit. Eleven of the sixteen had posterior dural laceration, and four had documented entrapment of neural elements between the laminar fracture fragments. In the remaining five patients, the neurological deficit was thought to be due either to neural contusion or to compression by retropulsed bone from the fractured vertebral body, or both.

A laminar fracture in association with a burst fracture predicts the possibility of a dural laceration, as no dural lacerations occurred in our patients who did not have a laminar fracture. Similarly, a preoperative neurological deficit in association with a lumbar burst fracture and laminar fracture was also predictive of dural laceration. It cannot be

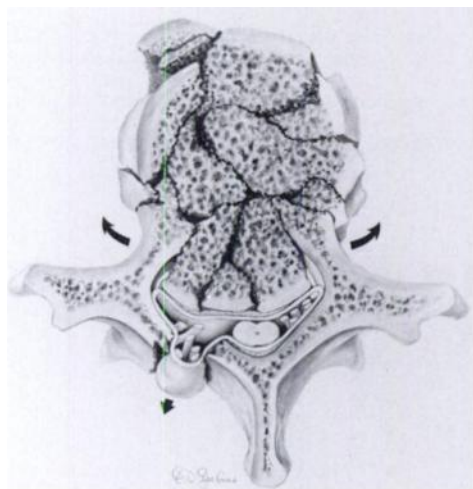


FIG. 5-A

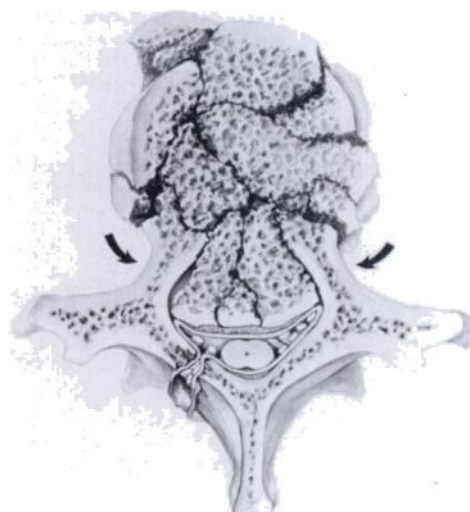


FIG. 5-B

Figs. 5-A and 5-B: Illustrations depicting the proposed mechanism of injury.

Fig. 5-A: With axial loading, the pedicles are spread laterally, the lamina fractures, and bone is retropulsed from the vertebral body. This may result in protrusion of the dura between the laminar fracture fragments.

Fig. 5-B: As the axial load is dissipated, the laminar fracture fragments recoil, possibly entrapping the dura and nerve rootlets.

predicted which patients who have dural laceration will have entrapment of nerve roots, although four (36 per cent) of the eleven patients in our study and eight (44 per cent) of the patients in the series of Miller et al.¹⁷ who had a dural laceration did have entrapment of nerve roots.

The mechanism by which posterior dural lacerations occur in association with burst and laminar fractures is not known with certainty. It is reasonable to assume that additional energy may be necessary to cause such a fracture pattern, and that this energy causes more severe soft-tissue injury and results in laceration of the dural sac. However, to our knowledge, this energy has not yet been quantified in any way.

Our theory (Figs. 5-A and 5-B) is that, with axial loading, the pedicles and posterior elements splay laterally, and bone is retropulsed from the vertebral body. This causes the dura to protrude between the laminar fracture fragments.

When the axial load dissipates, the dura and nerve rootlets are entrapped posteriorly as the laminar fragments recoil.

This review adds to the present controversy concerning whether an anterior or a posterior approach should be used to treat thoracic and lumbar burst fractures. Each exposure offers both advantages and disadvantages^{1,2,4,5,7,8,10-14,19}. However, if a neurological deficit and a laminar fracture are associated with a lumbar vertebral burst fracture, a posterior exposure allows repair of the dura and, more importantly, extrication of any entrapped neural elements.

A patient who has a neurological deficit and a laminar fracture associated with a lumbar burst fracture has an increased risk of dural laceration and, possibly, entrapment of the neural elements. This possibility should be recognized and perhaps should influence decisions regarding operative treatment.

NOTE: The authors thank Dale Glasser, M.S., for her assistance in the statistical analysis.

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