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Computed Tomography of Thoracic and Lumbar Spine Injuries

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CT scans of 73 patients with acute thoracic/lumbar spine injuries from T3 to L5 were reviewed. Injuries were classified as burst fractures (48), fracture-dislocations (ten), wedge compression fractures (11), and seatbelt-type injuries (four). Thirty-one (42%) had motor deficits due to spinal cord or nerve root damage. Such neurologic deficits were present in all patients with fracture-dislocations, and 60% of those with burst fractures. Seven patients, four initially normal, developed progressive neurologic impairment early after injury. Burst fractures, one with dislocation, were the spinal injury associated with each progressive deficit. Burst fractures at T12 or L1 with 50% or more decrease of the mid-sagittal neural canal diameter had a significant risk of neurologic involvement, and of progressive deficit. CT scans demonstrate vertebral column damage well, and help identify those patients at risk of acute neurologic compromise.

The excellent image quality, safety, and reliability of computed tomography (CT) have been well described, and used to justify its nearly routine use for evaluating severely injured spines. The need for high resolution sagittal and coronal plane reconstruction is recognized, since fracture lines and displacements in the transverse plane can be missed if only transverse CT images are examined (1–3, 10, 16a, 18, 22, 23). We are impressed by the crisp images of our CT scanner, and consider it the definitive second-level study for thoracolumbar spine fractures, after adequate AP and lateral radiographs have identified the involved areas, and demonstrated the gross alignment of the spinal column, which can sometimes be lost from view when one focuses on a small segment of the spine. In spite of increasing experience, we remained uncertain about the clinical significance of pathologic changes demonstrated by CT of vertebral column injuries. We therefore reviewed our acute injuries of the thoracic and lumbar spine, to correlate the CT observations with neurologic examinations, early course, and choice of treatment.

MATERIALS AND METHODS

Patients. Orthopaedic Surgery and Radiology Department logs identified those San Francisco General Hospital inpatients evaluated for acute injuries of the thoracic or lumbar spine with computed tomography during the study period, from May 1979, when our CT scanner was first used for this purpose, until July 1983. Patients with gunshot wounds, pathologic fractures, and injuries more than 10 days old were excluded. CT scans were available for essentially all patients with neurologic deficits related to fractures and dislocations of the thoracic and lumbar spine. Additionally, a number of neurologically normal individuals were scanned, based on our evolving procedures for assessment of spine injuries identified with routine radiographs. As well as spinal injury with neurologic deficit, these criteria include suspected instability, significant radiographic vertebral deformity or displacement, and the concurrent use of body scans to evaluate other organs in the region of a vertebral column injury. Some of these patients have been previously reported in the radiology literature by other authors (1).

CT Scans. All scans were performed with a General Electric 8800 scanner, with several significant improvements incorporated as they became available (1, 2). Digital radiographs (Scout View®) were used for localization, and occasionally provided better quality AP and lateral radiographs than those obtainable under Emergency Room conditions. The Scout View® images not only demonstrate clearly the location and orientation of the scanned plane, but guide adjustment of the gantry angle so that transverse scans can be perpendicular to the neural canal in the area of concern.

Other software advances permitted enhancement of bone detail (Bone Target technique) and reconstructions in multiple planes. Another benefit accompanying these additional functions was improved postoperative demonstration of the neural canal, in spite of artifacts produced by metal fixation devices. At the discretion of the attending physicians, metrizamide CT myelography was performed in place of, or in addition to, the routine CT scan studies (23% of patients). Special precautions were taken to move patients on and off the CT scanner table without flexing or rotating the spine. If traction was being used to help stabilize the spine, it was maintained during CT scanning. A physician from the Trauma, Orthopaedic, or Neurosurgery Service stayed with the patient during the CT scan if there was any suspicion of respiratory or hemodynamic instability, or undiagnosed multiple injuries.

Study Techniques. Clinical details including neurologic examinations, treatments, and hospital course were obtained from the medical records. Neurologic deficits were classified by the system of Frankel (8). CT scan photographs and routine radiographs of the study patients were reviewed to assess the

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vertebral level and type of injury, the involvement of various portions of the vertebral body, and, as much as possible, ligamentous disruption as indicated by displacement of normally adjacent structures. Injuries were classified according to the system of Denis as follows, making use of a three-column model of the spine (5, 18).

Wedge Compression Fractures: Anteriorly compressed vertebral body fractures without involvement of the posterior surface of the vertebral body (the bony part of the middle osteoligamentous column), and without radiographic evidence of tensile failure of the posterior column.

Burst Fractures: Comminuted vertebral body fractures with decreased height, and anteroposterior, lateral, or both forms of fragment displacement (11). They invariably showed retropulsion of vertebral body fragments into the spinal canal, confirming involvement of both anterior and middle columns. Posterior column damage could be present or absent. No sharp distinction was made between 'stable' and 'unstable' burst fractures.

Fracture-dislocations: These injuries are indicated by displacement in the transverse plane at the level of injury, no matter what the mechanism. The amount of required displacement was chosen as 8% or more of the AP diameter of the endplate (3 mm on plane film), rotation of more than 9°, or complete dislocation of one or both facet joints (16, 20, 25, 30).

Seatbelt-type Injuries: Purely tensile failure is present in both posterior and middle columns, either through bone (as in Chance's fracture), or through ligaments. If linear or rotational displacement was present in the transverse plane, injuries produced by this mechanism were classified as fracture-dislocations (9, 30).

Spinal Canal Compromise. In burst fractures, spinal canal narrowing is produced by retropulsion of vertebral body fragments into the spinal canal. We approximated the amount of narrowing with the percentage of the mid-sagittal diameter occupied by the retropulsed fragments. The mid-sagittal diameter was determined by measurement with a transparent ruler of immediately adjacent, uninvolved 'cuts' of the same magnification. Measuring this distance anteriorly from the inner margin of the posterior elements at the maximally involved level permitted determination of the depth of the bone fragments displaced into the canal, and thus calculation of the 'percentage of narrowing.'

RESULTS

Seventy-three patients with acute fractures and dislocations of the thoracic and lumbar spine were evaluated with CT scans during our first 4 years using this technique (57 were male, 16 female). Their ages ranged from 15 to 73, with a mean of 34 years. The great majority (54—74%) had fallen or jumped, often from a significant distance; eight were passengers in automobile accidents; one on a motorcycle; and two were struck by vehicles. Eight had other mechanisms of injury, including crushing by a wind-toppled wall, and involvement in the collapse of a human pyramid. Less than a third (23) of the patients had isolated spinal trauma, the others sustaining additional injuries, usually of the musculoskeletal system. One or both calcanei were fractured in 32%, and 12% had pelvis or hip fractures severe enough to risk hypovolemia. Serious intra-abdominal and/or intrathoracic injuries were found in 10%.

Spinal Injuries. Forty-eight had burst fractures (two with more than one level involved), 11 had wedge compression fractures, ten had fracture dislocations (one with a significant burst component as well), and four had seatbelt-type injuries. Most involved the thoracolumbar junction (Table I).

Neurologic Injuries. At initial assessment, 46 patients had normal neurologic exams. Four (8.7%) of them developed motor deficits during the first 48 hours of hospitalization. Of the 27 patients with abnormal neurologic exams, 15 were Frankel Grade D (useful motor function), one was Frankel Grade C (useless motor function), nine had no motor function (Frankel B or A), and two were not assessable due to impaired mental status.

Table II correlates neurologic grade with type of injury. Fracture-dislocations were associated with significant neural damage in all our cases. Of the patients with burst fractures 60% were and remained neurologically normal during their SFGH hospitalization. However, four patients, representing 12.5% of those with burst fractures and initially normal neurologic exams, developed motor deficits, as noted above. Wedge compression fractures and seatbelt-type injuries were not associated with neurologic deficits, with the exception of one patient with a T8 wedge compression fracture, and no CT evidence of canal compromise. Descriptions of his initial examination were conflicting, but it was soon agreed that he had no voluntary motor function distal to his injury. At anterior transthoracic exploration, a large fragment of

TABLE I
All patients, by vertebral level and fracture type

Spinal Level	Wedge	Burst	Fracture-dislocation	Seatbelt type	Totals
T3	—	1	—	—	1
T5	2	—	—	—	2
T5/6	—	—	1	—	1
T6/7	—	—	1	—	1
T8	1	—	—	—	1
T8/9	—	—	1	—	1
T11/12	—	—	3	1	4
T12	—	7	—	1	8
T12/L1	—	—	4	—	4
L1	3	16	—	2	21
L2	1	12	—	—	13
L3	1	7	—	—	8
L4	1	3	—	—	4
Multiple	2	2	—	—	4
Subtotals	11	48	10	4	73

TABLE II
All patients, by neurologic grade (Frankel), and fracture type

Fracture Type	A or B (No Motor)	C (Useless)	D (Useful)	E—>D	E (Normal)	(?)
Fx-dis (10)	7	1	2	—	—	—
Burst (48)	2	—	13	4	28	(1)
Wedge (11)	—	—	—	—	10	(1)
St-blt (4)	—	—	—	—	4	—
Totals (73)	9	1	15	4	42	(2)

herniated disc was found between the body of T9 and the posterior longitudinal ligament.

There were sufficient burst fractures to evaluate the relationship between level of injury and neurologic involvement. At the thoracolumbar level, 12 of 23 spinal injuries were neurologically abnormal. In the lower lumbar spine, from L2–L4, only four of 21 burst fractures injured nerve roots ($p < 0.05$ by Chi square with Yates's correction) (two patients with multilevel involvement, one with T3 fracture, and one with an equivocal neurologic exam are excluded).

Neurologic Deficit with Spinal Canal Compromise in Burst Fractures. Narrowing of the spinal canal by burst fractures was due primarily to posterior displacement of one or more fragments of the fractured vertebral body, generally its upper half, and usually at the level of the pedicles, which tend to hide such displacement on routine lateral radiographs. Careful inspection of such films, however, shows either loss or posterior displacement of the vertical posterior border line in this region in almost all of the vertebrae with retropulsed body fragments (Fig. 1). In addition to that caused by body fragment retropulsion, effective narrowing of the spinal canal could be due to angulation at the level of injury, but this was slight in most burst fractures (mean = 5°, range, 0–25° for those with normal neurologic exams; mean = 6.5°, range 0–25° for those with a neurologic deficit). Posterior vertebral elements were rarely displaced into the spinal canal by burst fractures, although occasionally the inferior articular processes of the intact cephalad vertebra were subluxed slightly anteriorly between the laterally displaced superior articular processes of the involved vertebra.

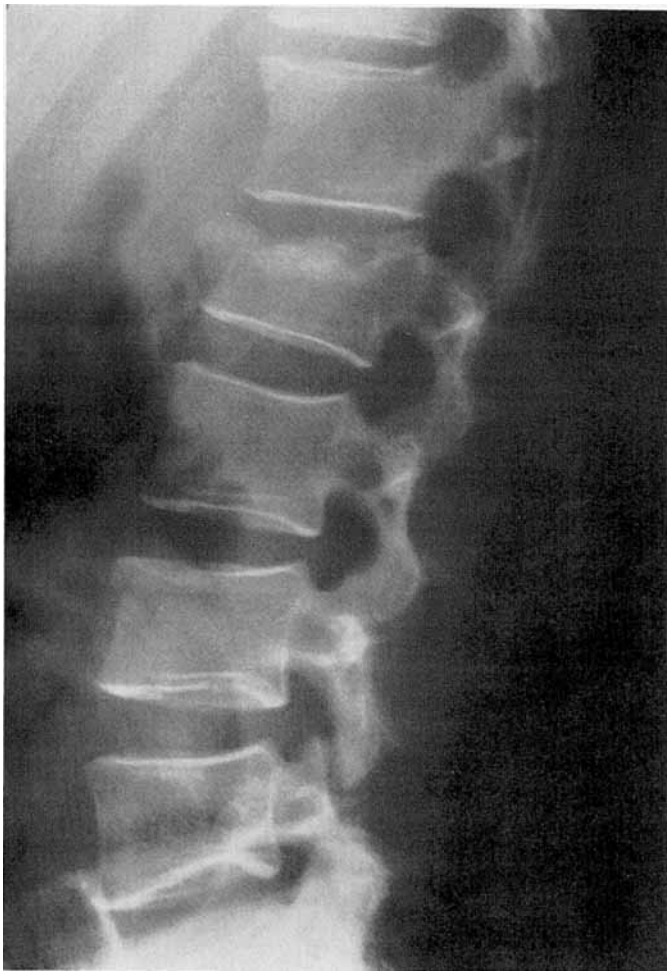

FIG. 1. Burst fracture of L2 showing posterior displacement of cephalad half of posterior body into neural canal. Note the intact posterior body line of the other lumbar vertebrae, including L4 with a minor superior end plate compression fracture.

Twenty-eight burst fractures from T12 to L4 were associated with normal neurologic examinations. Their mean canal narrowing (of the sagittal diameter) was 45%. Sixteen burst fractures with neurologic deficits present initially or developing during the early postinjury period occurred in the same region. (A single burst fracture at T3 with 42% narrowing and a complete neurologic deficit is excluded, because of the different anatomic characteristics of the upper thoracic spine.) For these, the mean occlusion was 57%. The difference between these two groups is insignificant ($0.10 > p > 0.05$, by Student's t-test).

A different situation pertains at the thoracolumbar junction, however. Burst fractures of T12 and L1 may damage the conus medullaris as well as the adjacent nerve roots of the cauda equina. Therefore these levels were considered separately (Table III). Eleven burst fractures of T12 or L1 were associated with normal neurologic exams. Their mean canal narrowing was 34% (only two values >50%). There were 12 burst fractures at the same two levels with neurologic abnormality, including those that became abnormal in their early stages. The

TABLE III
Thoracolumbar burst fractures

Level	% Narrowing	Frankel Grade
T12	6%	E (Normal)
T12	10%	E
T12	20%	E
T12	42%	E
T12	36%	E
L1	17%	E
L1	31%	E
L1	36%	E
L1	40%	E
L1	58%	E
L1	80%	E
T12	40%	D (Useful motor)
T12	62%	D
L1	30%	D
L1	50%	E–>D
L1	50%	D
L1	50%	D–>D–
L1	56%	D
L1	57%	D
L1	60%	E–>D
L1	62%	E–>D
L1	70%	A
L1	86%	D–>D–

mean canal narrowing for this group was 56% (only two values <50%). The difference in canal narrowing between these two groups was highly significant ($p < 0.01$, by Student's t-test).

Increasing Neurologic Deficits. Seven patients had well documented progression of their neurologic deficit during the first few days after injury. That is, they demonstrated new or significantly more profound motor deficits in specific muscles or muscle groups, compared with consistently described findings for the same muscle group on the initial examinations. As mentioned above, four with progressive neurologic involvement had normal examinations on initial evaluation. The other three were initially Frankel Grade D (useful motor function), and subsequently deteriorated.

When their deficits progressed, five of the seven patients were at bed rest with spinal injury precautions. Another developed delirium tremens 36 hours after admission, and his deficit, which had initially resolved shortly after application of halo-tibial traction, became significantly worse in spite of this provisional stabilization. These six patients with progressive deficits had burst fractures (five at L1, one at L3) with marked compromise of the neural canal (50–86%), averaging 66% decrease of mid-sagittal diameter. This was significantly greater than the canal compromise of neurologically normal patients with burst fractures ($p < 0.05$ by Student's t-test). The seventh patient with neurologic progression had a fracture-dislocation of T12 on L1, with significant associated burst fracture of L1 (Fig. 2.) Initially Grade D with only weakness of voluntary anal sphincter contraction, this patient lost function of the left L5 nerve root when taken out of halo-tibial traction and positioned for AP and lateral routine tomograms of her injury. In contrast, we are unaware of any patient's developing neurologic progression related to CT scanning.

Twelve thoracolumbar burst fractures had more than 50% canal compromise. Of the five neurologically normal patients, three developed motor deficits. Of the seven with motor deficits at admission, two deteriorated further. Thus the risk of neurologic deterioration was 42% for patients with 50% or more canal compromise due to burst fractures at the thoracolumbar level. At L3, three patients had greater than 50% canal compromise with normal motor function. One of the three (33%) deteriorated.

Posterior Element Fractures. Sagittal fissures, often with little if any displacement, and not infrequently incomplete, were present in 25 of the 46 single level burst fractures (Fig. 3). Such cracks in the laminae, occasionally visible on plain films, and well displayed on CT transverse sections, were present in the more severe fractures. Mean narrowing was 59% for those with fissures, and 37% for those without ($p < 0.001$). Neurologic involvement was also more common, seen in 56% of burst fractures with split laminae, but only 15% of those without ($p < 0.02$ by Chi-square with Yates's correction).

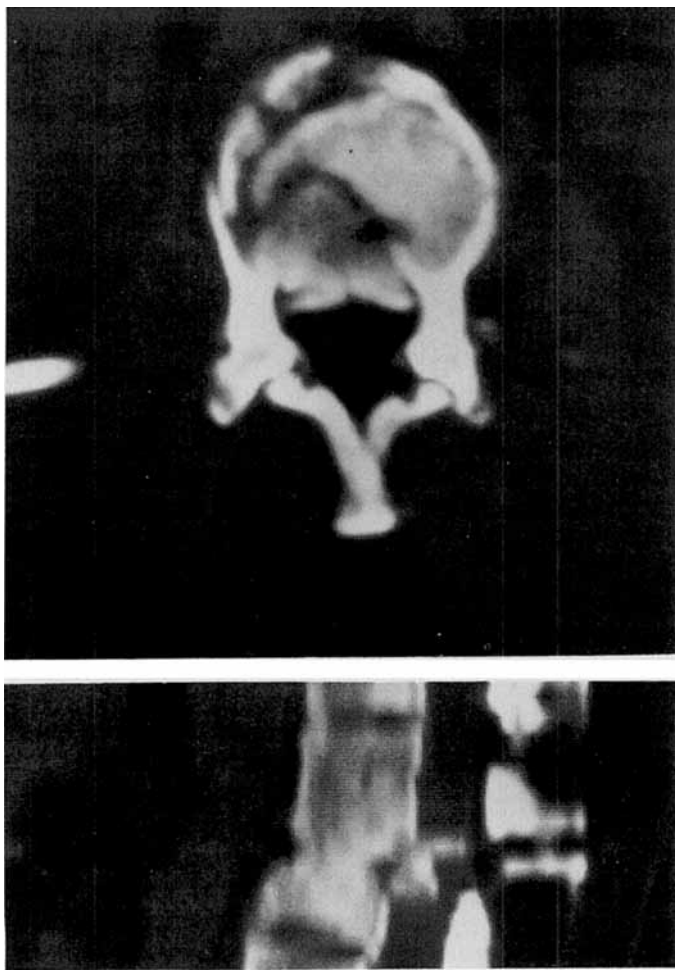

FIG. 2. Burst fracture of L1 with anterior translational displacement of L1 under T12 shown on midsagittal reconstruction. Although canal compromise by the retropulsed fragment is moderate, effective canal narrowing is severe due to translational displacement.

Other Posterior Element Injuries. Effective detachment of one or both pedicles from the remainder of the vertebral body was typical of burst fractures. Pedicle involvement was unilateral or bilateral. Often one or both displaced pedicles were connected to more or less substantial portions of the comminuted vertebral body. On routine AP radiographs, pedicle displacement was indicated by increased distance between the pedicles. Such widening of the inter-pedicular space did not correlate with neurologic deficit, and was not frequently associated with the finding of a torn dura by myelography or surgical exploration. (Two tears were found in 21 thoracolumbar burst fractures with operative descriptions, or adequate myelograms.) Convincing evidence of the status of the interspinous and yellow ligaments was rarely present on CT scans of thoracolumbar burst fractures, although local increase of the interspinous distance, when seen, suggests the inability of the posterior osteo-ligamentous column to sustain a tensile load. However, relatively increased interspinous distance, with engaged facet joints, may also represent physiologic flexion

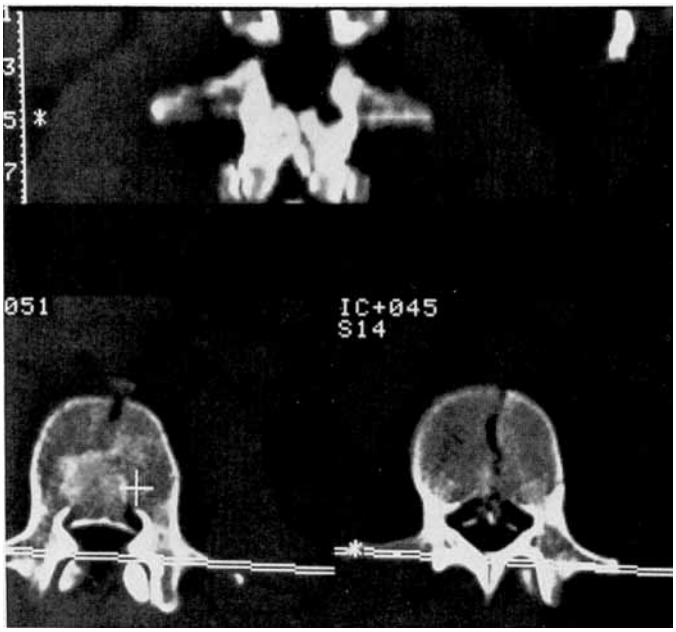

FIG. 3. L2 burst fracture with typical retropulsed fragment, pedicles attached to substantial fragments of superior body, and a minimally displaced sagittal fracture of the posterior elements. Note slight widening of the superior articular processes of L2 with gap in the L1–L2 right apophyseal joint (lower left corner).

at the involved level, due to loss of vertebral body height (Fig. 4).

Signs of Fracture-dislocation. Facet joint dislocation, indicated by the so-called naked facet sign on transverse sections (21), was confirmed by parasagittal and oblique CT reconstructions. Gross disruption of posterior elements, often with evident shearing displacement suggested by a 'double margin sign' (18), and confirmed by appropriately oriented reconstructions, typified fracture dislocations, as did rotational malalignment between adjacent vertebrae. Vertebral body comminution was variably evident in fracture-dislocation.

Flexion Injuries. Wedge fractures and seatbelt-type injuries appeared unimpressive on transverse CT images, but were well demonstrated with high-resolution reconstructed views in the mid-sagittal plane to show vertebral body deformity and angulation, and in correctly positioned parasagittal (for thoracic) or paracoronal (for lumbar) facet visualization. In spite of multiple attempts to reconstruct clearly the transitional thoracolumbar facet joints, their obliquity frequently led to suboptimal imaging at this level, so that distraction might have been obscured.

Transitional Categories. Several cases with apparently compressive vertebral body comminution and transverse plane displacement demonstrated the lack of a clear dividing line between burst fractures and fracture-dislocations (Figs. 2 & 5). Other burst fractures might have permitted similar displacement, for while we classified such injuries according to their appearance on standard radiographs and CT scans, reduction of deformity could have occurred from careful positioning for

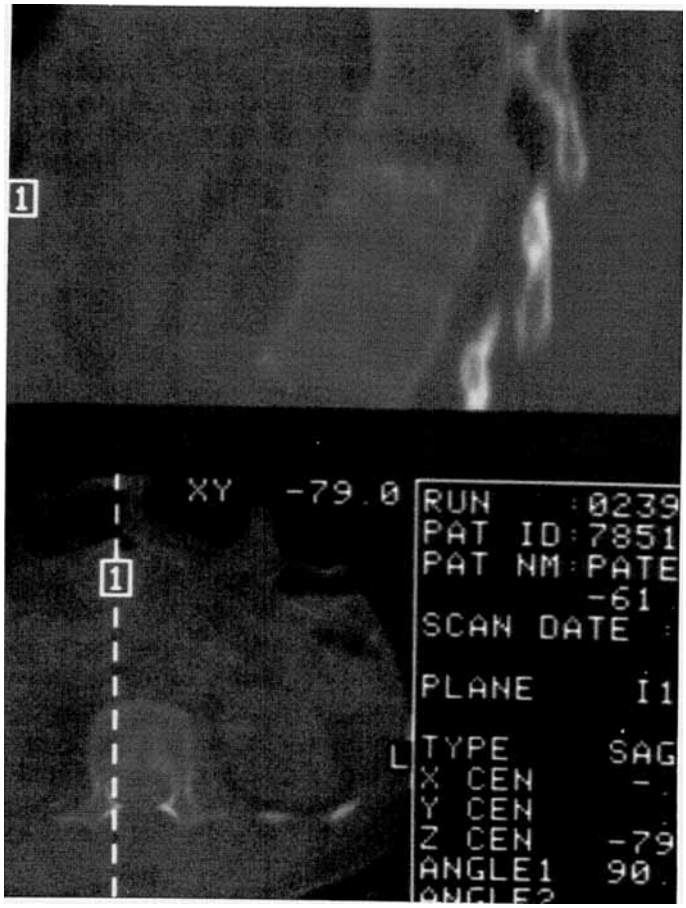

FIG. 4. Burst fracture of L1 with T12–L1 flexion, and near disengagement of right T12–L1 facet joint suggested by this parasagittal reconstruction. At surgery, however, the posterior ligaments appeared intact.

transportation, or by halo-tibial traction, which we frequently use for severe thoracolumbar injuries. It is thus possible that our radiographic studies underestimate the extent of ligamentous disruption, and that transverse or rotational displacement, or flexion with distraction of the posterior elements, might have been possible in other presumed burst fractures. Seatbelt-type injuries or wedge compression fractures severe enough to produce facet dislocations are associated with transverse plane displacement sufficient to narrow the neural canal, and are thus classified as fracture-dislocations in this series.

Treatment. Forty-four of the burst fractures were treated definitively in our hospital. Fourteen were operated upon: ten posteriorly, usually with lateral rachotomy for anterior decompression, with Harrington distraction rod stabilization and posterolateral fusion of the unstable levels; three anteriorly with debridement of retropulsed body fragments and anterior fusion; and one with halo-pelvic fixation. The other 30 burst fractures were treated nonoperatively, generally with at least 6 weeks bedrest, and an extension bodycast. Nine fracture-dislocations were treated definitively. Eight received posterior ORIF with Harrington rods, the ninth was treated nonoperatively because of medical problems, but recovered sig-

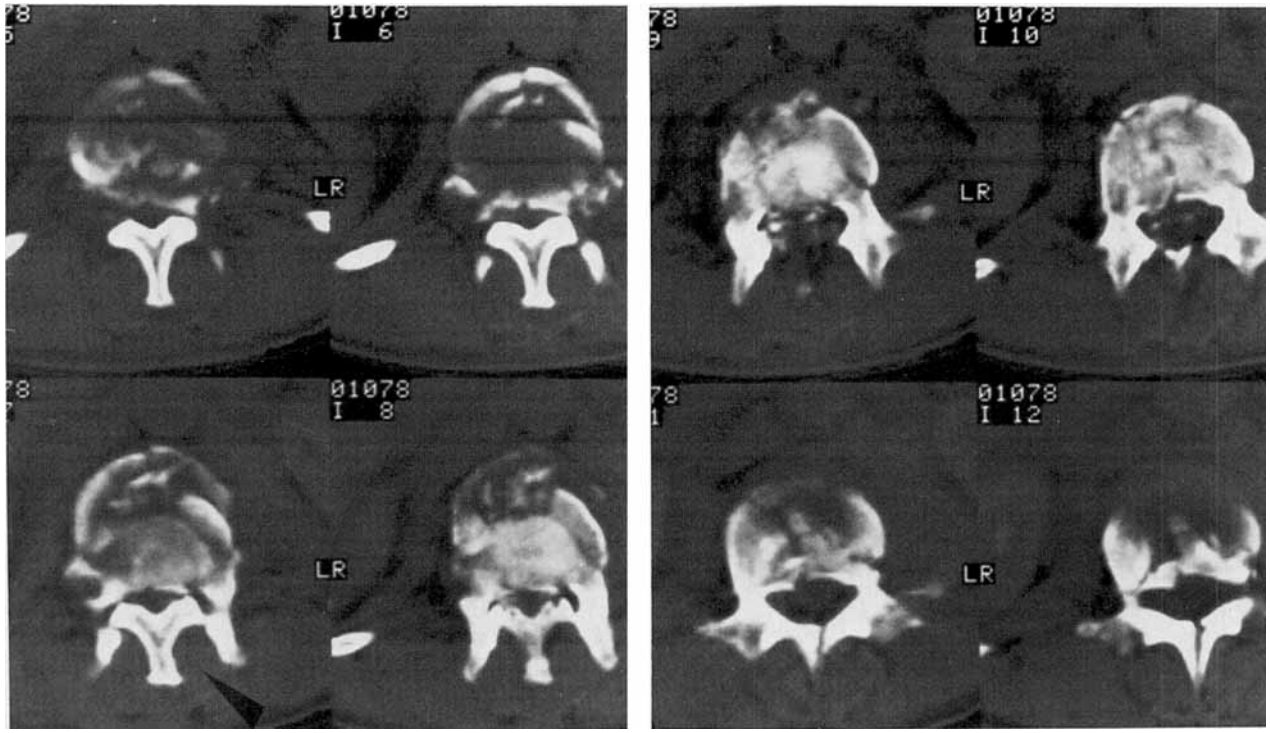

FIG. 5. T11–12 dislocation. Black arrow in lower left indicates anterior dislocation of posterior elements of T11 into spinal canal through widened superior articular processes of T12. Subsequent images of T12 demonstrate typical burst fracture appearance at this level.

nificant neurologic function. Ten wedge fractures were treated definitively, nine nonoperatively and one, mentioned above because of his neurologic deficit, underwent transthoracic debridement and anterior arthrodesis. Of the two seatbelt-type injuries treated definitively, one received posterior ORIF, and the other was managed nonoperatively. Results of treatment, incomplete at this time, will be the subject of another report.

DISCUSSION

This series is atypical, since falls (often suicide attempts) caused almost three quarters of the injuries. In most recent series, motor vehicle accidents account for the majority of cases (12, 13, 16–18, 24, 26). Injuries due to falls might be expected to produce more burst fractures, and possibly to cause less attendant ligamentous disruption, than those associated with vehicular accidents (28). Because of selection bias, more severe injuries are over-represented. Forty-two per cent had neurologic involvement, compared with 6% of the unselected hospitalized thoracic and lumbar spine fractures in the study of Riggins and Kraus (24).

The neurologic function of seven patients (10%) deteriorated acutely, in spite of careful positioning and nursing, predicated on the assumption that they had unstable spine injuries. Documentation of neurologic 'progression' is always difficult and potentially suspect in a busy Trauma Center, where examinations are performed by several team members, often junior house officers, and where recording of data may be less than systematic. Therefore we required specific and consistent descriptions of new or clearly worsened motor deficits before accepting that a deficit was progressive. This incidence is not unusual: others have reported that 4 to 14% of thoracic and lumbar spine fractures show early neurologic deterioration (4, 5, 16, 26).

There is as yet no universally accepted classification for injuries of the thoracic and lumbar spine. Furthermore, although the same terms are used by different authors, their criteria vary. One's burst fracture might be another's fracture-dislocation. We believe the system of Denis (5) is the most useful, because it distinguishes among grossly unstable fracture dislocations, burst fractures with a spectrum of clinical behavior, seatbelt-type injuries which are unstable in flexion only, and stable wedge compression fractures. Morphologic differentiation among the types is generally easy. However, several of our patients had transitional forms of injury: facet dislocation with minor anterior column disruption or burst fracture with transverse plane displacement. We therefore believe that fracture-dislocations can be produced by axial loading with more or less flexion, as well as by forces and moments restricted to the transverse plane. Because of their serious neurologic and mechanical consequences, we classify as fracture-dislocations all those injuries with transverse plane displacement or rotation significant by Posner's criteria (20).

Injury to the spine may damage the spinal cord and nerve roots by crushing, stretching, or both. Fracture-dislocations, which distort the spinal canal severely, pose a serious threat to the neural elements. All of our patients

with such injuries had neurologic damage. In most spinal injuries, maximal deformation of the spine and its neural elements occurs upon impact, at the moment of maximal loading. Radiographs are not taken then, but after the patient has been carefully positioned on a spine board and transported supine and in neutral rotation to the hospital. Thus plain films and CT scans show *residual*, not maximal, deformity. We might thus expect a somewhat vague relationship between the extent of radiographically evident damage and the functional status of the neural elements. Any correlation is likely to be further skewed by the anatomy of the thoracic and lumbar spines. From T1–T11, the spinal canal is small, and relatively filled by the spinal cord. Little tolerance exists for canal deformity, as indicated by the severity of neurologic involvement in our thoracic injuries. At the thoracolumbar junction, the canal diameter increases, and the cord gives off the lumbar and sacral nerve roots of the cauda equina before terminating at the lower border of L1. The increased space, and the fact that most of the neural tissue is more durable root rather than cord, enable greater deformation to be withstood. Below L1, the canal is large, usually with abundant space for the cauda equina. Sudden, nearly total canal occlusion can occur without clinical evidence of neurologic abnormality. Debate continues about the relationship of residual canal deformity to neurologic impairment, and whether or not it limits recovery of incompletely damaged neural tissue (4, 7, 12, 15, 18, 26). Mechanical instability of the injured area also interferes with recovery of an incompletely injured spinal cord (6). Therefore continued or increasing neural damage could be due to excessive motion at the site of injury as well as to the amount of observed deformity.

Precise measurement of spinal canal dimensions, including cross-sectional area, is possible with CT techniques, using programs supplied with the scanner, or modifications such as those described by Ullrich (3, 7, 29). It is thus tempting to relate computer-measured spinal canal dimensions to post-traumatic neurologic deficit, in the same manner that spinal stenosis from other causes is evaluated (27). However, the correlation between absolute (18, 19) or relative (7) narrowing of the spinal canal is claimed to be poor, based on small series of cases, involving different levels of the thoracic and lumbar spine, and including various types of injuries.

Burst fractures, unlike fracture-dislocations and flexion-distraction injuries, produce a localized constriction of the spinal canal by retropulsion of vertebral body fragments, the size of which varies from case to case (1, 7, 14, 23). Thus they may be compared with one another to evaluate the neurologic consequences of acute canal narrowing. Because thoracic spine injuries are so likely to produce neurologic deficit, and the lower lumbar spine frequently tolerates severe acute canal narrowing without clinically evident neurologic damage, we considered the thoracolumbar region as the best place to assess the effect of spinal canal compromise by burst fractures, most of which fortuitously occur at this level.

To quantitate neural canal narrowing produced by burst fractures, we measured the mid-sagittal diameter on CT photographs of the most involved level, and expressed narrowing as a percentage of the adjacent normal diameter. This less precise technique for evaluation of canal encroachment does not measure the area of the irregularly shaped neural canal. It does correct for obliquity of the scanned plane, can be applied to CT photographs at any magnification, requires no valuable computer time, and can be done at night when an attending radiologist might be unavailable.

Our initial experience with this estimate of canal narrowing included patients with astonishing canal compromise, yet normal neurologic examinations, as well as others with major neural deficits in spite of relatively modest canal encroachment. This was consistent with previously published results, and led us to question the significance of spinal canal narrowing when the patient's neurologic exam was normal. We were therefore surprised when our enlarging series of burst fractures at the thoracolumbar junction demonstrated that canal narrowing actually correlated strongly with neurologic deficit, and that it was also a valuable predictor of those patients at risk of neurologic progression. A 50% decrease in mid-sagittal diameter proved to be a significant boundary between worrisome narrowing, and that which was usually well tolerated, at least acutely, given otherwise satisfactory management. This amount of canal encroachment is less than the 67% figure chosen by Jelsma et al. as CT evidence for neural compression at the thoracolumbar level (15).

The association of neural injury with this degree of spinal canal compromise does not prove that continuing canal encroachment is the cause of either the initial neurologic damage, or its subsequent progression. Some other effect of the injury might be to blame, for it may be assumed that the greater the displacement of bone into the canal, the more severe was the injury in the first place. It cannot be assumed that surgical removal of the retropulsed bone fragments necessarily promotes neurologic recovery. It is well known that the natural history of incomplete thoracolumbar neural injuries is to recover cauda, if not conus, function (4, 8, 11). Additionally, any beneficial effect following surgery could be due to stabilization rather than decompression (6). Finally, our data do not yet allow determination of the long-term significance of such a degree of canal compromise. How frequently will it be associated with the late development of neurologic deficits? Will its early surgical correction prevent or cure those deficits? Only properly controlled, long-range studies will tell.

It is impossible to discuss spine injuries without considering the several concepts of 'instability' (5, 11, 12, 19, 20, 25, 26, 30, 31). Used in the acute sense, an unstable injury is one that risks neurologic injury. From a longer

term perspective, it implies an injury that fails to heal securely, and demonstrates increasing deformity, with or without late neurologic deficit. Another aspect of instability is that associated with excessive late mobility and pain at the injured level. An 'unstable' spine fracture may show one or more of these problems. Criteria for instability remain topics of debate and experimentation (20). There is probably a spectrum of instability, ranging from completely stable to grossly unstable, without a sharp dividing line between the two. This must be more true when the effects of healing are considered, for many 'unstable' spine injuries will heal to stability if protection and alignment are maintained long enough (4, 8, 11). The present study evaluates only the first type of instability—that causing immediate risk to the spinal cord and its nerve roots. Our experience suggests that fracture-dislocations and burst fractures with at least 50% canal compromise are at least acutely unstable, i.e., have a high immediate risk of neurologic problems. Most of the posterior element fractures noted on transverse plane CT images are probably due to compressive loading with bursting of the ring. As such, they more properly suggest a loss of capacity to withstand compressive load, rather than the tensile incompetence stressed by Whitesides (31). While we noted a significant association between laminar fractures and early neurologic involvement, we believe that this is because they are indicative of more forceful injuries, rather than because of any mechanical instability due to the cracks themselves.

CT contributed to the management of many of our patients. It helped distinguish burst fractures from wedge-compression fractures, thus separating patients with more severe injuries from those who could be mobilized safely without precautions, as their symptoms permitted. By providing detailed demonstrations of the pathologic anatomy, CT imaging was invaluable in planning the details of a surgical procedure, such as whether or not to perform lateral rachotomy for anterior decompression, and if so, on which side (7, 12, 16). When multiple levels were involved, the suitability of Harrington rod hook placement sites could be assessed. As McAfee points out, the type of failure of the spine's middle column is a valuable guide for choosing the type of internal fixation (18).

Since our indications for surgery were primarily neurologic involvement, or fracture-dislocation (which in our cases was always associated with neurologic involvement), the CT findings themselves rarely led directly to the decision to operate. Based on the present observation that, for burst fractures, 50% encroachment of the thoracolumbar canal is associated with a significant neurologic risk, we now consider it relatively indicated to advise decompression and stabilization for neurologically normal individuals with thoracolumbar burst fractures that have this degree of retropulsion, as well as for those with gross mechanical instability, or significant deformity not adequately corrected by nonoperative means. Spinal instrumentation and fusion also decrease the length of hospitalization and disability, and possibly decrease the risk of persistent pain (4, 12). Should routine CT studies fail to explain the level or severity of neurologic deficit, we advise metrizamide CT myelography, to search for extradural soft-tissue displacement (disc herniation), a rare finding among our patients. In spite of others' experience (17), we found that dural tears were unusual in burst fractures, and do not believe that their incidence is sufficient to warrant routine exploration of thoracolumbar fractures with widened pedicles.

CONCLUSIONS

1) Detailed CT evaluation contributes significantly to management of thoracic or lumbar spine fractures. With careful transfer, observation, and support of the seriously ill patient, it safely demonstrates the spinal injury, and aids accurate classification and planning of therapy.

2) Fracture-dislocations carry a high risk of neurologic involvement. Because of this, and their intrinsic lack of mechanical stability, they should be identified, reduced as quickly as possible, and stabilized surgically as soon as this is safe for the patient.

3) Thoracolumbar burst fractures with 50% or more compromise of their mid-sagittal diameter by retropulsed fragments, or with fractures of the lamina, are at significant risk of neurologic involvement. They should be observed carefully for this, and treated with either prolonged recumbency, or surgery that adequately provides both anterior debridement and secure stabilization.

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DISCUSSION

DR. THOMAS B. DUCKER (University of Maryland Hospital, Baltimore 21201): This is an important contribution. There were 54 patients with a fall or a jump, which is three quarters of their series. This makes it a unique series, and it should be reported without complicating the number with the few motor vehicular accidents that they have.

[Slide] While in Baltimore we have a lot of crazy people, I don't think they jump out of windows. This gives you some idea of what we like to treat. This was described by Galen eons ago, with a complete cord injury. If you get only half the cord you know it is called a Brown-Sequard syndrome. That was a neurologist by the name of Charles Brown. He wrote only one paper. He married Mrs. Sequard, a member of a prominent family, so now we have the Brown-Sequard syndrome.

[Slide] Let me re-give you terms as to what the paper said. Doctor Trafton described ten patients with flexion injuries—in other words, shearing off the axis. This is called a fracture-dislocation. This mechanism is a flexion injury where there is a shearing off. In all of his patients with this there was 100% neurologic damage. In our center it runs in the 80's or 90's. So, that is a very devastating injury.

They had 11 patients with a wedge fracture. A wedge fracture is a flexion compression. Those do much better because there is only about 10 to 20% neurologic damage, and it basically gives you a wedge fracture of the anterior part of the vertebral body. If there is no great shearing it simply bends the spinal cord or nerve roots, and the patients do fine.

Of more importance, they had 48 patients in their series with what they called burst fractures. To me, that is a compression injury where the axial load comes right down and you have a burst vertebral body. Sixty % of these patients ended up with a deficit. Twelve % were normal initially, only to end up with a deficit that progressed. In the thoracic area where you have this kind of fracture, nearly all of them have a deficit. At T12–L1 in this paper, 50 to 60% of them end up with a deficit. At L2–3–4 where we have only cauda equina or nerve roots, the deficit now drops to 20%. So, the cauda equina can obviously move aside.

The next important figures are: At T12–L1 with this compression fracture which is a burst fracture, which is the most important part of their paper, if there is a narrowing there greater than 50%, nearly 80 to 90% of their patients had or developed a deficit. If narrowing was less than 50%, only about 20% of them had a deficit. Even if you ended up without a deficit coming down here and had a narrowing, then your chances are that three out of five of the five patients who came in that way will progress.

If you superimpose splitting the lamina, which adds more to the picture as to the force of the injury, then that increases your chance of deficit further. The overall deterioration rate of 10% in these patients is compatible with that in other series of 4 to 14%, and 9% in our own institution.

[Slide] This shows you the problem of how this burst fracture can be very pure and come back in as to the compression.

[Slide] Sometimes when you get the rotary component it is very difficult to know exactly how much narrowing there is.

First of all, disregard the abstract. When I first read the abstract I was about ready to go to San Francisco and beat him over the head. The abstract and the conclusions are different. He agrees. We both agree. We calculate the recovery rate by the standard indices that we have developed ourselves, which are accepted by the American Spinal Injury Association, which are more important because we put numbers on the muscle function below, and when you get to the cauda equina it is extremely important as far as useful motor function is concerned.

We stress first that the mechanism of injury is more important than the pre-existing anatomy. For example, if you have a distraction flexion like a seatbelt (a Chance fracture) you are going to have the nerve roots torn in half; therefore they are going to do badly. Or if you have flexion down in that area they are going to be torn in half, as opposed to here: you are just going to sit on them. Once you sit on them, we agree that compression is a very important element. You don't wrap tumor, you don't wrap disk, you don't wrap fractured bone around trauma, so therefore you have to achieve a decompression.

The authors don't add a lot to their treatment and describe what they feel; they say it is another paper. We feel those patients should be aggressively treated.

I enjoyed the abstract and the paper. Thank you very much.

DR. RONALD E. ROSENTHAL (Department of Orthopedics and Rehabilitation, Metropolitan-Nashville General Hospital, Nashville, TN 37210): Our series is comparable to the series

Doctor Ducker described. Likewise, we don't have a lot of jumpers in Nashville. Perhaps we don't have enough tall buildings and our bridges aren't that high over the Cumberland River.

What we have been doing are CT scans preoperatively and postoperatively on patients, particularly those with burst fractures, which are our most common ones as well as in the series described in the paper.

What we are finding is that the posterior protrusion, the retropulsion of the bony fragments, does not change following instrumentation. We don't have enough cases; we have three now, and that is not a series, but in our preoperative and postoperative CT scans the size of the canal does not change even after the fracture has been instrumented, and a standard lateral X-ray makes us jump for joy: oh, what a wonderful reduction we've gotten!

We are not replacing these retropulsed fragments. They are not being sucked back in, and we are getting some evidence to demonstrate this. Likewise, that has no bearing on neurologic recovery, at least not in our series.

I have one question for the author. You described several patients who had neurologic progression, that is, progressive neurologic deterioration as they went along. In our institution, when someone begins to deteriorate we operate on him and stabilize him. What is your protocol for a patient who is deteriorating neurologically before your eyes?

DR. ROBERT J. WHITE (3395 Scranton Road, Cleveland, OH 44109): I would like to ask the authors if in their series of investigations of these patients they have used a metrizamide marker to investigate the spine. Many of us really have moved beyond just the CT scan in attempting to correlate the damage within the canal particularly in reference to the spinal cord. As the authors know, with metrizamide and the CT scan one can outline the spinal cord and/or the roots, which is the basic problem here.

DR. TOM PHILLIPS (Department of Surgery, Downstate Medical Center, Brooklyn, NY 11203): I have one question for the authors. They indicated that in some patients they were more or less able to predict progression of neurologic deterioration on the basis of their CT scan findings. However, they don't mention anything about the results of the surgical treatment of those patients.

What was the prognosis in that group of patients who you felt were at high risk for neurological progression, and therefore were treated aggressively? Did you prevent subsequent neurologic loss, or were they able to attain a meaningful recovery based on your aggressive treatment by identifying them as being at high risk by CT scan?

DR. PETER G. TRAFTON (Closing): Thank you for your questions. I appreciate Doctor Ducker's kind remarks. I certainly concur with his comments about the need for more precise measurement of neural function than Frankel's scale. In the future we must all use standards for neurological classification such as those advocated by the American Spinal Injury Association. I agree strongly with Doctor Rosenthal's comments about the results of neural canal decompression with Harrington distraction rods in burst fractures. One can regain canal alignment in fracture-dislocations quite successfully with early and well-conceived operation. However, getting rid of the retropulsed fragments of burst fractures is not reliably accomplished by merely distracting the spine. We have done intraoperative myelograms to evaluate canal decompression, but find that routine posterolateral decompression is the most reliable technique in our hands, and that is our standard approach now for dealing with the problem of significantly retropulsed fragments.

Doctor White's question about the benefit of metrizamide scans opens an area of interest. We have done metrizamide scans in approximately 23% of the patients involved. I am not sure they have added significantly to either our understanding of the pathology or to our specific plans for management of the patients. Rarely this identifies a situation where the routine CT scan does not tell us exactly where the problem is. We have had only one patient in whom the CT scan showed no good explanation for the problem, whereas a metrizamide scan might have identified the lesion—a herniated disk associated with a high thoracic wedge compression fracture.

I must emphasize that it was a retrospective conclusion that 50% canal compromise is significant. The literature suggests, as was our initial impression, that there was no correlation between canal compromise and neural deficit. I hope we will be able to present in the future the prospective results of using this indicator for patients at risk of neurologic progression, for whom we feel spine decompression and stabilization is indicated as promptly as is prudent.

Thank you very much.