BURST FRACTURES OF THORACOLUMBAR SPINE
(CURRENT CONCEPTS REVIEW)

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ABSTRACT:

Burst fractures are one of the most common traumatic vertebral injuries. And one of the most controversial in means of treatment, and classification is burst fracture of the spine. In this article, we reviewed the literature on the definition, mechanism, classification, and management of burst fracture. We want to bring a different perspective to the burst fractures in every respect by synthesizing the knowledge and our experimentations.

Key Words : *Burst fracture, Mechanism, Management.*

When you explode a vertebral body by putting a bomb inside, a very complex fracture hard to understand, to classify, and controversial in treatment, appears.

Today, one of the most controversial fracture in means of treatment, and classification is burst fracture (BF) of the spine.

Every day, thousands of people admit to emergency departments because of falling down or traffic accident, sustaining burst fracture. In the future more and more burst fracture will be seen because of increasing world population. This is the main cause of why we must learn completely the pathomechanics and treatment modalities of burst fractures and popularize it.

In this article, we reviewed the literature on the definition, mechanism, classification, and management of burst fracture. There is no agreement in the literature on above mentioned situations. Treatments of similar fractures were greatly different at different centers. Thus, we want to bring a different perspective to the burst fractures in every respect by synthesizing the knowledge in the literature and our experimentations.

Holdsworth (35) initially defined the BF as a violent axial compression force in which the superior end plate is fractured and the disc extruded into the vertebral body.

We think that the most important difference of the burst fractures from compression or other vertebral fracture is the fracture of the middle column. For this reason, we prefer to define the burst fracture as the disruption of middle column together with the other columns and/or ligaments of vertebra or alone.

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30-60% of burst fractures at the thoracolombar junction have an associated neurologic injury (11). Whitesides determined that the burst fracture is the most common cause of neural injury in the thoracolumbar spine (54).

When Holdsworth (35) introduced the concept of burst fracture, he believed that since the ligaments remained intact, this fracture was stable despite the comminution.

Kelly and Whitesides (41) modified the Holdsworth's concept, and they considered burst fractures with retropulsed fragment to be unstable and believed that they should be treated by anterior decompression.

DeWald (14) stated that anterior column resists compression and posterior columns resist tension.

Denis (13) classified the burst fractures according the three column model and focused attention on the middle column.

McAffe (45) proposed that the key anatomic structure is the middle column osteoligamentous complex. He thought that when posterior column failure accompanied to this structure's disintegrity the fracture is considered unstable.

Argenson (1) demonstrated that the annulus fibrosus and longitudinal ligaments are extremely important in the stability of the spine.

Farcy and Weidenbaum (21) modified the Denis classification to include expression of bone and soft tissue in each of the three columns, thereby arriving at a six grade classification. They noted that injuries greater or equal to grade 3 were unstable (fig. 1).

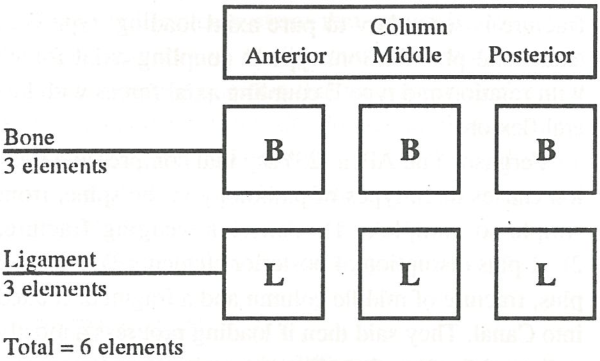

Figure 1

MECHANISM OF BURST FACTURES

At the moment of occurring of a burst fracture, loads that are going to form the fracture, effect the vertebra with various combinations in three different anatomic axis and different periods. This means that fracture pattern is changeable when one of the loads that are in the x, y, and z axis is less or more (Fig. 2).

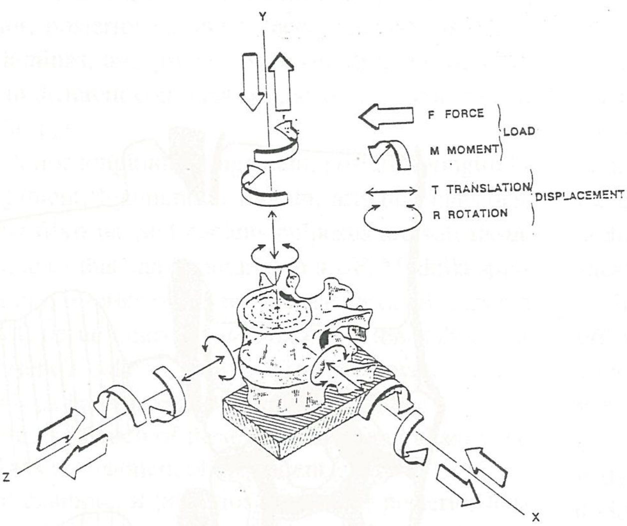

Figure 2

Another factor that changes the fracture type is the place where the resultant load effects on the vertebral topography (Fig 3).

We suppose that fracture type would be effected by the time period which resultant force acts actively. This means that, a load, which acsts for a short time, can destroy some components o the vertebral body or ligaments. However, if the force acts with same intensity for a prolonged period, it could destroy more parts of osseous or ligamentous structures, and the fracture would have be much comminuted when you compare with the previous one.

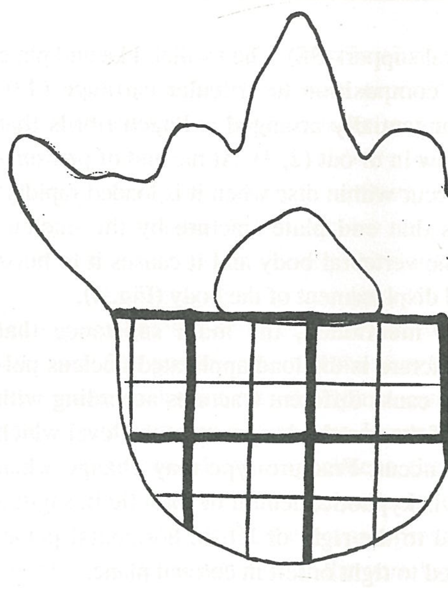

Figure 3

The above mentioned items are valid for most of fractures, for the BFs, too. But the factor which causes the fracture is different biomechanicaly in burst fractures. This factor is presence of nucleus pulposus (35).

Under normal loading conditions, the stiffness of vertebral body cancellous bone exceeds that of the disc, which is compressible and viscoelastic (20, 34, 50). The characteristics of the disc allow distrubution of large stress if load application is slow enough to allow creep response (20, 37). When the disc is loaded normally, fluid movement through the porous end plate dissipates energy and redistributes stresses. However, when rapid loading exists, as at the instant of injury, the relationship in terms of stiffness between disc and bone change (53). Impact loading in hyaline cartilage and hydrated biologic tissue has been shown to lead to fluid pressurization, since there is not enough time to allow for normal fluid redistribution (3). The disc is a hydrated tissue with hyaline like cartilaginous end plate on either end as well as a calcified layer that gives structural support (38). The hyaline like end plate is similar in composition to articular cartilage (49), containing tangentially arranged collagen fibrils that rapid fluid flow in or out (2, 3). At the end of pressurization may occur within disc when it is loaded rapidly. The result is that end plate fracture by the nucleus forced into the vertebral body and it causes it to burst with outward displacement of the body (Fig. 4).

As above mentioned, the main substance that creates the fracture is the load applicated nucleus pulposus. It may cause different fractures according with the position of the vertebral segment at the level which fracture will occur. Fracture type may change when this segment is kyphotic, neutral or lordotic in sagittal plane, rotated to the right or left in horizontal plane, laterally flexed to right or left in coronal plane.

In Denis's classification (12), he claims that type A fracture is secondary to pure axial loading, type B is axial load plus flexion, type D coupling axial forces with rotation and type E coupling axial forces with lateral flexion.

Ferguson and Allen (23) say that compressive flexion causes three types of pathology in the spine, from simple to complex: 1) Anterior wedging fracture, 2) 1 plus disruption of posterior elements 3) 1 plus 2 plus, fracture of middle column and a fragment rotated into Canal. They said then if loading progress a mostly unilateral fracture that also compromises posterior and middle columns will occur.

In our experience, we think that every BF is quite different from each other due to action of different mechanisms. Also, we believe that different position of any body segment from the toe to the occipitoatlantal articulation changes the type of the fracture.

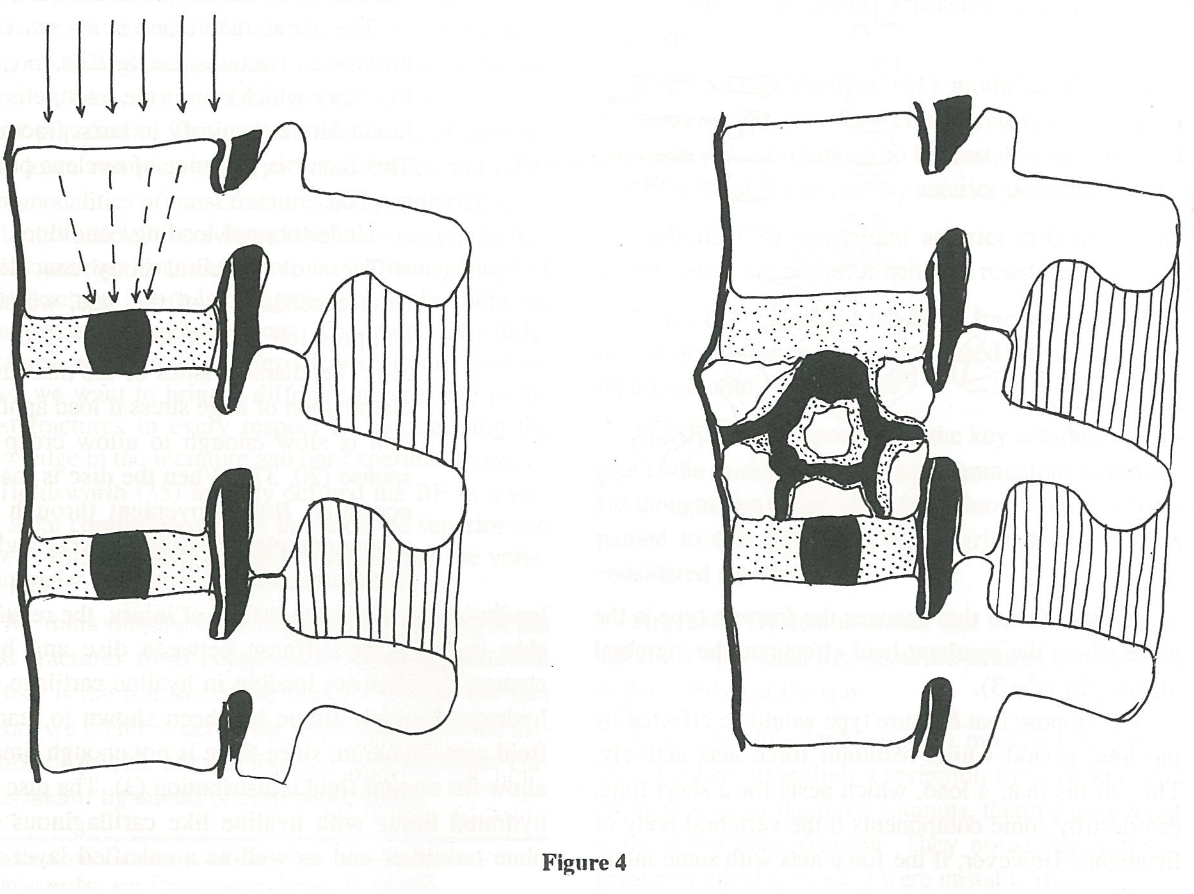

Figure 4

The classification systems that categorize the actual different fractures as similar must be questioned. In fact, the important question that we have to think about is that if burst fractures should be classified or not? Perhaps, only the understandings of pathomechanism of fracture occurring and fracture reduction is the most important cornestone in achieving treatment and management of the burst fractured patient.

PATHOLOGY

Fractures can occur in different parts of vertebral corpus, and fracture of a lamina together with burst fracture can worsen the clinic of the patient (56). For this reason, a surgeon treating a BF, should have a knowledge of the pathology of this fracture. We use conventional x-rays, tomography, computerized tomography, magnetic resonance imaging, and operative findings to understand the pathology of the fracture.

The pathology can be investigated as soft tissue and osseous tissue pathology. Superior end plate, inferior end plate, both superior and inferior end plates, middle, anterior, posterior columns, facet articulations, pedicles, laminas, and processes of vertebra can be fractured in different combinations, as osseous component of pathology.

Anterior longitudinal ligament, posterior longitudinal ligament, ligamentum flavum, articular capsules, annulus fibrosus, and nucleus pulposus are soft tissue components that can be injured in a BF. Medulla spinalis injury, injuries of the nerve roots or dural tears are also soft tissue injuries but don't effect the stability of the vertebral column, however the presence of neural injury is an indirect sign of clinical instability.

Injuries of each of these structures, can change the prognosis or treatment of the patient.

For example, if posterior annulus or posterior longitudinal ligaments are disrupted, an indirect reduction maneuver, so called ligamentotaxis could not be succesful. Besides, posterior distraction techniques can have deleterious affect on neurologic status of the patient (26).

Injuries of each structure, in different combinations make the exact pathology of the fracture. In otherwords, each fracture may contaion the different combinations of these structural injuries. For this reason, we think that it is very important to understand the exact pathology and treatment principles in each pathologic conditions, than to classify the burst fracture.

Although it was not a classification, Willen et al. (55) grouped the burst fractures according to the pathology observed in their cadaver study. In group 1 (Denis A and B), the vertebral body was compressed with a tendency for anterior wedging. The vertebral body was comminutely fractured mainly in the upper parts. The lower vertebral end plate was fractured with associated rupture of the lower disc, but the injury of the upper vertebral end plate and disc was more pronounced. The posterior part of the vertebral body was protruding backward into the spinal canal. PLL showed of laxity and partial rupture. However, it was not completely ruptured. In all specimens, there were signs of partial rupture/fracture of the posterior column.

In group 2 (Denis D), there was a pronounced comminution of the upper half of the vertebral body. The upper half of the vertebral body. The upper disc was severely lacerated in all cases, with bone fragments protruding in to the disc material. ALL, sometimes, was totally ruptured, sometimes was avulsed from the injured vertebra and from the disc below. Bone fragments narrowing the spinal canal were larger than those group 1. PLL was totally ruptured at the level of fractures. The injuries of the left and right sides of vertebral body differed by the size and localization of the bone fragments in to the spinal canal. In all specimens there was total rupture of the bony part of the vertebral arch. Extradural hemorrhage and compression of dural sheath and neural tissues observed in all cases.

In group 3 (Denis D2), a large fragment was split off anteriorly and posteriorly from the upper part of vertebral body. In the center of motion, corresponding to the transition between the middl and posterior third of the intervertebral disc, the relatively intact vertebral body intruded tentlike into the disc, which was entirely divided up to the lower vertebral end plate of the vertebral body alone.

Frederickson et al. (26) showed that there were important injury at the superior and inferior end plate of fractured vertebra, annulus fibrosus were disrupted from the both end plate, and there were bleeding into nucleus in their study which they studied upon the BF had been created specimens they have had from the cadaver. Sometimes PLL was ruptured, but sometimes it was intact. Annulus fibrosus was ruptured or injured in various degrees. They also showed that when PLL was ruptured alone, reduction could be achieved by distraction posteriorly, but when it was ruptured together with posterior annulus fibrosus it could not be achieved.

Controversies exist in literature concerning medullar canal compression and neurologic injury. Some authors say that there is a correlation between medullar canal compression and neurologic status (16, 17, 19, 44, 52), some authors don't (4, 11, 28, 33).

We think that usually intracanal fragment is responsible from the neurologic status. But it is impossible to detect the relation between the fragment and neural elements at the moment of fracture occurring. Postural reduction occurs in supine position (9). The fragment seen at CT minimally compressed to the spinal cord cold be totally dissected the medulla spinalis at the instant of fracture occurring, and then retracted forward again. Sometimes, especially in lumbar region, when canal is compressed as high as 75% there might not be any neurologic findings. We agree with Hashimato et al (32). They say that the intracanal fragment is not always correlated with the neurologic status. But if the compression is about 35% at T11-12 level, 45% at L1-2, and 55% at cauda equina level, the possibility of neurologic injury is very high.

Nerve injuries are variable in BFs. It can be a simple contusion or a serious transection. Subarachnoid bleeding, dural tears may be seen. Microscopically, acute necrosis of central gray matter or bleeding may be seen also. Edema and/or hematoma may enhance the neurologic findings (55).

If neurologic deficits with clinical examination and level of fracture are inharmonious, it must be checked for a herniated nucleus pulposus at the above level or for laminar fractures (10). Trafton et al (52) and Cammisa et al (10) claimed that if there is a laminar fracture together with fragment in the canal, neurologic deficits will enhance.

CLASSIFICATION

Classification of burst fracture is difficult. First of all, classifications up today depend on either radiographic methods (x-rays, tomography, computerized tomography, magnetic resonance imaging) or cadaver studies.

Radiographs are taken at supine position and they don't reflect the position of the vertebra under a physiologic load, nor does it reveal a deformity that may exist in the future (30).

In cadaver studies, fracture is occurred when a part of the vertebral segment above and below is stable, due to an axial load applicated, but we think, preparation like that does not reflect a true burst fracture. Because, the minimal changes in spatial position of the vertebral body and related segments may change the fracture type.

An ideal classification should be easily understandable, cover all types of fractures with different pathologies, and give treatment options to the surgeon.

There is no ideal classification for burst fractures. Now we want to give a perspective for classifications up to date with their advantages and disadvantages.

DENİS'S CLASSIFICATION

Denis (12) divided these injuries, based on their CT characteristics into five subjects (fig. 5).

Type A : Fracture of the both vertebral endplates
Type B : Fracture of the superior end plate
Type C : Fracture of the inferior end plate
Type D : Burst rotation type fracture
Type E : Burst fracture plus lateral flexion

We think that Denis classification is very important because of it had been a reference for other studies about classification of burst fractures after published.

However, it does not reflect the clinic, include ligamentous structures, and posterior column. But it is still most popularly used classification for burst fractures.

Applying the mobile segment concept, Farcy and Weidenbaum (21) modified the Denis's classification to include expression of bone and soft tissue in each of the three columns, thereby arriving at a six grade classification. They noted that injuries greater than or equal to grade 3 were unstable (fig 5).

The bony subtance of each column is labeled with a "B", the ligamentous (soft tissue) subtance of each column with an "L". The mobile segment (one disc and one vertebra) is thus composed of three B elements and three L elements, a total of six elements. Disruption of three or more bony or ligamentous elements of a column results in instability.

DALL AND STAUFFER'S CLASSIFICATION

Dall and Stauffer (11) classified the burst fractures to determine the correlation between fracture type and neurologic outcome-prognosis (Fig 6).

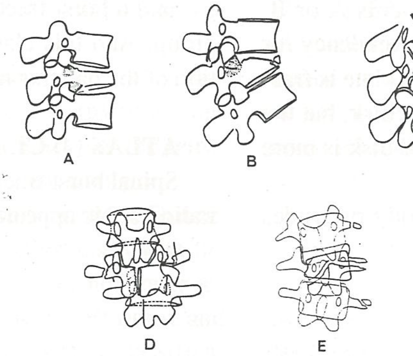

Figure 5. Denis's Classification of Burst fractures

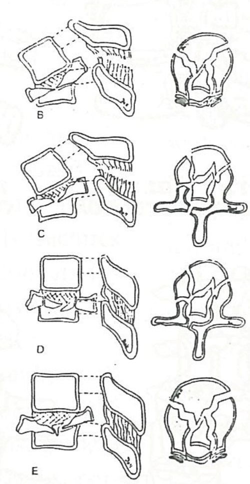

Figure 6. Dall and Stauffer's Classification of Burst fractures

Type 1 fracture has>15° kyphosis and maximal canal compromise at the level of the ligamentum flavum. Fracture type 2 has>15° kyphosis and maximal canal compromise where bone encircles the canal. Fracture type 3 has<or equal to 15° kyphosis and maximal canal compromise at the level of the ligamentum flavum.

They emphasized that type 1 and 2 fractures have good, type 3 and 4 fractures have poor prognosis.

In this study, fourteen consecutive patients with burst fractures at T12 or L1, partial paralysis, and more than 30% canal compromise were prospectively evaluated; pretreatment and posttreatment with roentgenograms to determine the initial fracture pattern, CT scans to determine the percent canal compromise and subsequent improvement, and a quantitative motor trauma index scale and bladder sphincter evaluation to determine neurologic recovery. As a result, they said that the initial severity of paralysis did not correlate with the initial fracture roentgenographic pattern or the amount of initial CT canal compromise, and neurologic recovery did not correlate with conservative or operative treatment.

The major disadvantage of this classification system is that, it does not give treatment options for surgeon, and does not include all types of burst fractures.

WILLEN'S CLASSIFICATION

Willen (55) classified burst fractures into three types depending on pathologic findings of cadaver with burst fracture.

Group 1: Group 1 corresponds to Denis A or B. The vertebral body is compressed, with a tendency for anterior wedging. The lower vertebral end plate is fractured with associated rupture of the lower disk, but the injury to the upper vertebral end plate and disk is more pronounced.

The posterior pat of the vertebral body protrudes backward into the spinal canal. The bone fragments are kept in place by the posterior longitudinal ligament, which shows laxity and partial rupture. However, PLL is still in continuity. There can be signs of partial rupture or fracture of the posterior column.

Group 2: Group 2 corresponds to Denis Type D. There is pronounced comminution of the upper half of the vertebral body. The upper disk lacerated, with bone fragments protruding into the disk material. Sometimes ALL can be totally ruptured. PLL is totally ruptured. The injuries to the left and right sides of the vertebral body differs by the sizes and location of the bone fragments protruding into the spinal canal. When the spinal column is manually provoked in flexion or traction, the fragments did not alter the position. However, in extension and in compression the fragments are put further back.

Group 3: It corresponds to Denis type D2. A large fragment splits off anteriorly and posteriorly from the upper part of the vertebral body. In the center of motion, corresponding to the transition between the middle and posterior third of the intervertebral disc, relatively intact vertebral body, which is entirely divided up to the lower vertebral end plate of the vertebral body above. PLL is ruptured.

This classification depends on pathologic findings of burst fractured cadavers. So it is possible that it could not cover all different combinations of pathology, and a burst fracture may not be one of these three group. And this classification does not give information of the patients neurologic status.

ATLAS (4) CLASSIFICATION

Spinal burst fractures can be classified according to radiographic appearances (Fig 7).

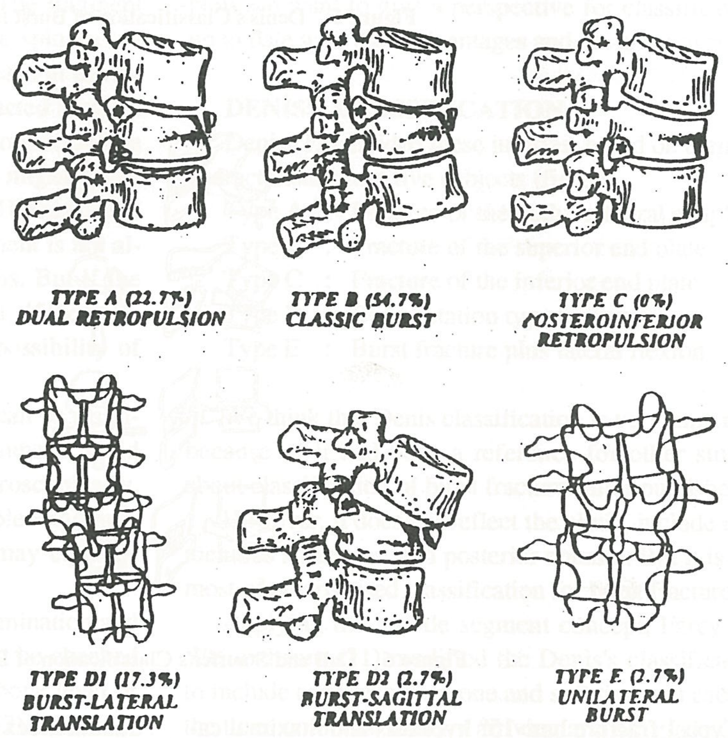

Figure 7. Atlas's Classification of Burst fractures

Type A: Dual retropulsion burst. In these fractures, fragment originating from both the posterosuperior and postero-inferior margins of the involved vertebral body are retropulsed into the spinal canal.

Type B: In this type of burst fracture there is retropulsion from the posterosuperior corner, the inferior endplate remain intact.

Type C: There is isolated posteroinferior retropulsion and intact superior endplate.

Type D1: These complex fracture-dislocations result from axial compression with accompanying hyperflexion, so that facet pearching or frank locking occur.

Type E: The unilateral burst fracture. In these unusual fractures, marked comminution involves predominantly one side of the vertebra, so that the retropulsed fragment originated from that side only.

Atlas's classification is a modification of Denis's classification. So, it has similar disadvantages with the Denis's classification. In addition, it does not cover rotational burst. Also it lacks of clinical, soft tissue and posterior column information like Denis's classification.

WEIDENBAUM AND FARCY

Farcy and Weidenbaum (22) developed the sagittal index (SI) concept and evaluated burst fractures into three types.

Type A: SI<15° and instability grade < 3

Type B: SI 15°- 25°, instability grade > or equal 3

Type C: SI > 25°, instability grade > or equal 3

Farcy and Weidenbaum advocated that fracture type A can be treated conservatively, type B fractures can be treated by posterior reduction, fixation and fusion, type C fractures can be treated by posterior reduction and fixation followed by anterior fusion.

In this prognostic classification system, authors advocate that, surgery is not needed for group 1. But sometimes, patient has less than 15° kyphosis, grade 3 instability may have neurologic deficits and need operative treatment. In group 2, authors recommended posterior surgery. But PLL and posterior annulus may be ruptured and ligamentotaxis could not be decompress the canal. So, an anterior decompressive approach could be needed such a situatition.

TREATMENT

Controversy still exist in the treatment of burst fractures. Some authors advocate conservative treatment, while others, operative treatment for the same fracture types. The purpose of the treatment has to be restore patient's life quality. Operative and nonoperative treatment modalities must be considered in the treatment of burst fractures.

Three criteria are used to determine if nonoperative treatment is an acceptable therapeutic option. They are: 1. Less than 15° of kyphosis, 2. Less than 30% of loss of vertebral body height and, 3. No neurological deficit (11, 25, 31, 43). If these criteria are present, we believe that a long-term successful result may be obtained by nonoperative measures. But, there is again some controversy in conservative treatment of BFs, since few parameters exist for predicting which fractures will progress. Krompinger (43) indicated that local kyphosis greater than 15° warranted stabilization, whereas Dall and Stauffer (11) noted that neurological recovery is best when canal compromise is accompanied by kyphosis more than 15°. Reid (47) felt that 50% canal compromise in conjunction with loss of height and local kyphosis indicate surgery. Bradford (7) cited loss of vertebral body height 60% to 70% canal stenosis, and angular deformity of 30°- 40° as indication for stabilization in the absence of neurologic deficit.

Goutallier (29) identified a need for surgical intervention in two types of burst fractures: 1) fractures with comminution of vertebral body with local kyphosis from endplate to endplate greater than 15° and with posteriar wall retropulsion greater than 4 mm. into canal and 2) fractures with local kyphosis greater than 25° in which postural reduction fails to correct kyphosis to less than 20°. Sagittal index (Fig. 8) recently described in an effort to further quantify deformity and to help assessing the likelihood of progression.

In our concept, nonoperative treatment consist of 3 to 4 weeks of bed rest to allow for resolution of pain and some consolidation of the injured tissues. The patient than is placed in hyperextension thoracolumbar corset and is permitted to be ambulatory but braced and closely monitored clinically and radiographically for three to six months.

Operative Treatment

The criteria we use for determining whether surgical interventions that will be recommended are: kyphosis greater than 15° and/or spinal canal compromise more than 25% with or without a correlative neurological deficit (7, 11, 17). Despite of good functional results have been documented with nonoperative approaches, particularly in the absence of neurologic deficit, surgical intervention facilitates faster mobilization, better correction of deformity, and greater neurologic improvement. However, apporaches to decompression and stabilization remain hotly debated.

Surgical philosophies have included the following: posterolateral decompression with or without instrumentation, posterior reduction with intrumentation without decompression, anterior decompression followed by posterior instrumentation, posterior instrumentation followed by anterior decompression and fusion without instrumentation, and anterior decompression and fusion with instrumentation.

Routes of decompression include posterior, lateral, and direct anterior. Posterior decompression by laminectomy is mentioned only to be condemned as a proven poor approach with dire consequences. We think laminectomy is never indicated alone for thoracolumbar burst fractures. This has been demonstrated by Holdsworth and Hardy (36), who documented that laminectomy added to the instability and could increase the incidence of early and late neurologic deficit.

Mc Affe et al (44) studied the degree of neurological recovery in incomplete lesions. Their study suggested a more favorable outcome with anterior surgery when results were compared to postural reduction and posterior instrumentation without decompression. The findings of Bradford and Mc Bride (7) were similar in that they noted a correlation between neurologic recovery and the adequacy of surgical spinal canal decompression. However Gertzbein et al İ(8) found no statistical difference in the neurologic outcome comparing anterior surgery and posterior surgery. Farcy and Weidenbaum (53) said that direct anterior decompression via thoracotomy or thoracoabdominal approach has the distinct advantage of providing total visualization of the involved fracture for decompression, arthrodesis and stabilization. The quality and extend of anterior decompression are greater, with less neurologic risk than with a posterior procedure. Unlike posterior decompression, little manipulation of neural elements is necessary during anterior decompression. Surgical complications of anterior approach are minimal when done by an experienced team. Riska (48) reported no major complications in 79 anterior procedures. Therefore, anterior decompression is safer and more complete than a posterior procedure, while still facilitating anterior bone grafting and stabilization.

Decompression is an important component of treatment of a burst fractured patient. It has been documented experimentally and clinically (5, 6, 13, 16, 27, 40, 42, 44) decompression leads to neurologic improvement of compressed neural elements, including cauda equina, spinal cord, and peripheral nerves. Mechanical distortion of neural tissues as well as transient impairment of spinal cord circulation may be involved in reversible neurologic deficit due to compression (51). Although some authors have correlated neurologic injury with the degree of canal compromise (32, 52), other studies have not confirmed these findings. Studies by Edwards et al (18), Fidler (24), and Jhonsons et al (39) documented the occurrence of gradual resorption of such displaced fragments. Reports vary greatly on the extend of canal encroachment necessary for neurologic injury, varying from 35% to 55% canal compromise, to 50%, to 67%. These variations may exist as a result of corresponding canal size and content. As described previously, Hashimato et al (32) noted that minor canal compromise at the level of spinal cord which correlates the narrowest portion of spinal canal can cause more neurologic deficit than thase of level of cauda equina or conus medullaris. Farcy and Weidenbaum (37) states indications for surgical decompression in neurologic compromised patient as: 1. acute burst fracture with incomplete neurologic injury and 2. acute or late burst fracture with worsening of neurologic setting. We are also in accord with these authors in their indications however, a neurological intact patient having a canal compromise should be decompressed because of the risk of future stenosis in this area. On the other hand, there are reports in favor of remodeling in minor compromises (18, 24, 39).

We think that, the important question is what would be the correct method for surgical decompression. The answer of this question depends of pathomechanics of different decompression procedures. Decompression can be accomplished indirectly, by realignment of the spine or directly, removing the bone fragments from the canal under direct vision. Indirect decompression is accomplished by fracture reduction and restoration of sagittal contour with posterior instrumentation. This uses the ligamentotaxis effect by applying tension to the posterior longitudinal ligament and posterior annulus (Fig 8) to restore vertebral body height and reduce displaced anterior fracture fragments while they are still loose. Frederickson et al (26) documented that posterior longitudinal ligament complex provides only a minor contribution in the reduction of

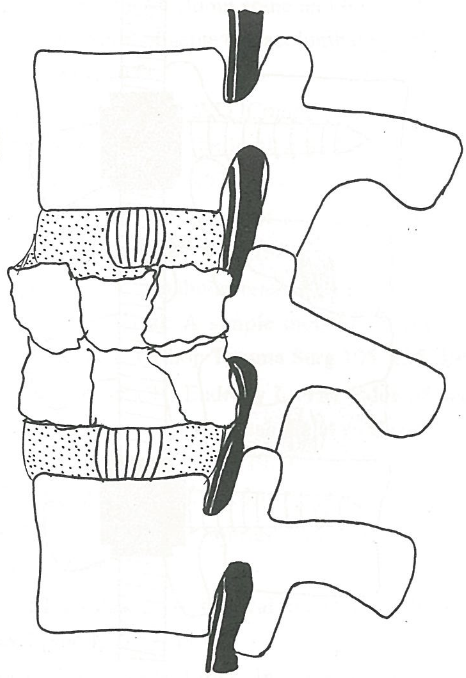

Figure 8 - a

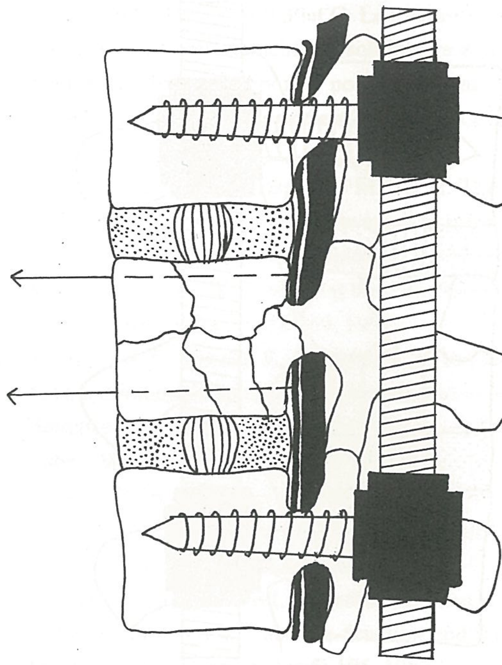

Figure 8 - b

the intracanal fragment in comparison to the posterior half of the annulus fibrosus. The annular complex, specifically the attachments of the intracanal fragment, appears to be the main contributor to the reduction of this fracture. If these structures are intact after injury, adequate reduction of the fracture should be possible using posterior instrumentation. This means that if these structures, especially posterior half of the annulus are ruptured at the instant of trauma, reduction of the intracanal fragment would not be possible by the posterior distraction. Actually, we observed worsening of neurologic status after distracting the fractured vertebra by posterior instrumentation. Retrospectively, we think that PLL and posterior annulus were ruptured in this case. For this reasons, we strongly advocate that MRG must be routine in all burst fractures with intracanal fragment. If it is not possible, decompression of the canal anteriorly would be advisable. If reduction is lack from bony support at the anterior corpus, possibility of implant failure is high with posterior instrumentation. In such a situation, an anterior approach must be chosen (Fig 9). In indirect reduction maneuver by posterior distraction, there is a neurologic risk, not only further compression of the intracanal fragment, but by the decreasing the blood flow to the neural elements due to stretched blood vessels. Another disadvantage of this technique is that it can be used within a 48 to 96 hours following trauma, since after this period, consolidation of the bone fragments will not permit indirect reduction.

Posterolateral decompression is usually done in conjunction with posterior instrumentation. This technique involves removing the transverse process and varying amounts of facet, pars interarticularis, and pedicle. We think that this procedure may cause further destabilization at the level of fracture by removal of compressing but stabilizing bony elements.

Anterior grafting by posterior approach is an other alternative described in conjunction with posterior fixation. Transpedicular approach is used to introduce bone graft ateriorly as a bone paste injected directly into the pedicle (15, 46). However, this technique clearly requires to use of a posterior device that provides reduction and fixation, also it has potentially risk of protruding graft into the canal.

As a result, we advocate that surgical planning must be guided with magnetic resonance imaging. This may offer a means to asses the integrity of the posteri-

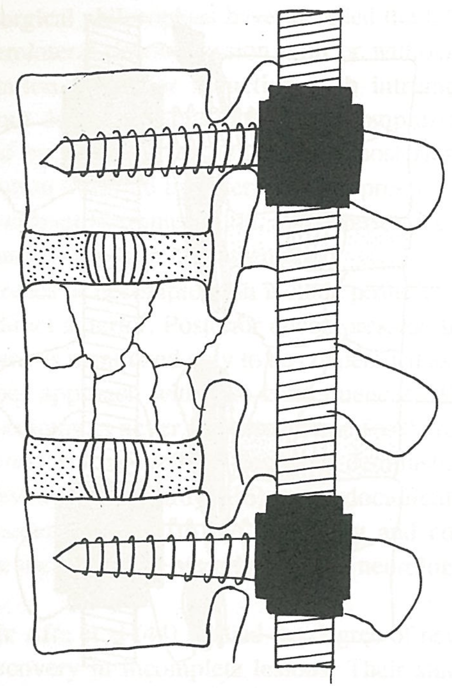

Figure 9 - a

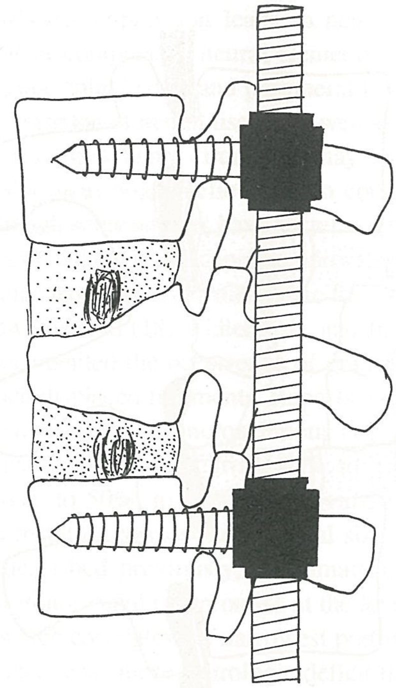

Figure 9 - b

or annular attachments to the intracanal fragments. If a magnetic resonance imaging of the patient is not possible, and it has been shown by CT that patient has intracanal fragment with canal compromise canal more than 25%, an anterior decompression technique must be chosen.

REFERENCES :

1. Argenson C, Lovet J, Cambas PM, et al: Osteosynthesis of the thoracolumbar spine fractures with Cotrel-Dobousset instrumentation. Fifth Proceedings of the 1988 International Cangress on Cortel-Dubousset Instrumentation. Sauramps Medical, 1989.
2. Armstrong CG, Mow VC: Variations in the intrinsic mechanical properties of human articular cartilage with age, degeneration, and water content. J Bone Joint Surg 64 A: 88, 1982.
3. Armstrong CG, Mow VC, Wirth CRİ: Biomechanics of impact-induced microdamage to articular cartilage. In Finerman G (ed): AAOS Symposium on Sports Medicine, p 70-84. St. Louis CV Mosby, 1985.
4. Atlas SW, Regenbogen V, Rogers LF, Kim KS: The radiographic characterization of burst fractures. AJR 147: 575, 1986.
5. Bohlman HH: Traumatic fractures of the upper thoracic spine with paralysis: A study of 180 cases. J Bone and Joint Surg 56 A: 1299, 1974.
6. Bohlman HH: Treatment of fractures and dislocation of the thoracic and lumbar spine - Current concepts review. J Bone Joint Surg 56 A: 165, 1985.
7. Bradford DS, Akbarnia BA, Winter RB, Seljeskog EL: Surgical stabilization of fracture and fracture-dislocations of the thoracic spine. Spine 2: 185, 1977.
8. Bradford DS, McBridge GG: Surgical management of thoracolumbar spine fractures with incomplete neurologic deficits. Clin Orthop 218: 201, 1987.
9. Cain EJ, De Jong JT, Dinenberg AS, Stefko RM, Platenburg RC, Lauerman WC: Pathomechanical analysis of thoracolumbar burst fracture reduction. A calf spine model. Spine 18 (12): 1647, 1993.
10. Cammisa FP, Eismont FJ, Green BA, YUAN HA: Dural laceration occuring with burst fractures and associated laminar fractures. J Bone Joint Surg 71 A: 1044, 1989.
11. Dall BE, Stauffer ES: Neurologic injury and recovery patterns in burst fractures at the T 12 or L1 motion segment. Clin Orthop 233: 171, 1988.

12. Denis F: The three column spine and its significance in the classification of acute thoracolumbar spinal injuries. Spine 8: 817, 1983.
13. Denis F, Armstrong GWD: Compression fractures versus burst fractures in the lumbar and thoracic spine. J Bone Joint Surg 63 B: 462, 1981.
14. DeWald RL: Burst fractures of the thoracic and lumbar spine. Clin Orthop 189: 150, 1984.
15. Dick W: Use of acetabular reamer to harvest autogenic bone graft material: A simple method for producting bone paste. Acta Orthop Trauma Surg 105: 235, 1986.
16. Dolan EJ, Tater CH, Endreny L: The value of decompression for acute experimental spinal cord compression injury. J Neurosurg 53: 749, 1980.
17. Dunn HK: Anterior spine stabilization and decompression for thoracolumbar injuries. Orthop Clin North Am 17: 113, 1986.
18. Esses SI: Evaluation of surgical treatment for burst fractures. Spine 15: 667, 1990.
20. Eyre D: Intervertebral disk. In Frymoyer JW, Gordon SL (eds): New Perspectives on Low Back Pain, pp 131-214. Park Ridge, AAOS, 1989.
21. Farcy JPC, Weidenbaum M: A preliminary rewiev of the use of Cotrel Dubousset Instrumentation for spinal injuries. Bull Hosp JT Dis Orthop Inst 48, 1: 44, 1988.
22. Farcy JPC, Weidenbaum M, Glassman SD: Sagittal index in management of thoracolumbar burst fractures. Spine 15: 958, 1990.
23. Ferguson RL, Allen BL: A mechanistic classification of thoracolumbar spine fractures. Clin Orthop 189: 77, 1984.
24. Fidler M: Remodeling of the spinal canal after burst fracture. J Bone and Joint Surg: 70 B: 730, 1988.
25. Frankel HL, Hancock DO, Hyslop G, etal: The value of postural reduction i the initial management of closed injuries of the spine with paraplegia and tetraplegia. Paraplegia 7: 179, 1969.
26. Frederickson BE, Rausching WE, Bayley JC, Yuan HA: Vertebral burst fractures: An Experimental, Morphologic, and Radiographic Study. Spine 17 (9): 1012, 1992.
27. Gelfan S, Tarlow LM: Physiology of spinal cord nerve root and peripheral nerve compression. Am J Physiol 185: 217, 1956.
28. Gertzbein S: The neurologic outcome following surgery for spinal fractures. Spine 13: 641, 1988.
29. Goutallier D, Hernigou P, Piat C: Le treatment orthopedique des fractures du rachis dorsolumbaire et lumbaire avec recul du mur corporeal posterieur sans ou avec troubles neurologiques mineurs. Rev Chir Orthop 74: 77, 1988.
30. Haher TR, Tozzi JM, Lospinuso MF, et al: The contribution of the three columns of the spine to spinal stability: Biomechanical model. Paraplegia 27: 432, 1989.
31. Hanley EN, Starr JK: Junctional burst fractures. Orthopaedic Transactions 15 (2): 269, 1991.
32. Hashimato T, Kaneda K, Kuniyoshi A: Relationship between traumatic spinal canal stenosis and neurologic deficitis in the thoracolumbar burst fractures. Spine 13: 1268, 1988.
33. Herndon WA, Gallaway D: Neurologic return versus cross-sectional canal area in incomplete thoracolumbar spinal cord injuries J Trauma 28: 680, 1988.
34. Hickey DS, Hukins DWL: Relation between structure of the annulus fibrosus and the function and failure of the intervertebral disc. Spine 5: 106, 1980.
35. Holdsworth F: Rewiev article: Fractures, dislocation, and fracture-dislocations of spine. J Bone and Joint Surg 52 A: 1534, 1970.
36. Holdsworth FW, Hardy A: Early treatment of paraplegia from fractures of the thoracolumbar spine. J Bone Joint Surg 35 B: 540, 1953.
37. Humzah MD, Soames RW: Human intervertebral disc: Structure and function. Anat Rec 220: 337, 1988.
38. Inoue H: Three-dimensional architecture of lumbar intervertebral discs. Spine 6: 139, 1981.
39. Johnson R, Herrlin K, Hagglund G, Stromqvist B: Spinal canal modelling after thoracolumbar fractures with interspinal bone fragments. Acta Orthop Scand 62: 125, 1991.
40. Kaneda K, Abumi K, Fijiya M: Burst Fractures with neurologic deficits of the thoracolumbar-lumbar spine: Results of anterior decompression and stabilization with anterior instrumentation. Spine 9: 788, 1984.
41. Kelly RP, Whitesides TE: Treatment of lumbodorsal fractures-dislocations. Ann Surg 167: 705, 1968.
42. Kostuik JPİ: Anterior fixation for burst fractures of the thoracic and lumbar spine with or without neurological involvement. Spine 13: 286, 1988.
43. Krompinger WJ, Frederickson BE, Mino DE, et al.: Conservative treatment of fractures of the thoracic and lumbar spine. Orthop Clin North Am 17: 161, 1986.

44. McAffe PC, Bohlman HH, Yuan HA: Anterior decompression of traumatic L1 fractures with incomplete neurological deficit using a retroperitoneal approach. J Bone Joint Surg 67 A: 89, 1985.

45. McAffe PC, Yuan HA, Frederickson BE, Bubicky JP: The value of computed tomography in thoracolumbar fractures. J Bone Joint Surg 65 A: 461, 1983.

46. Olerud iS, Karlstrom G, Sjostrom L: Transpedicular fixation of thoracolumbar vertebral fractures. Clin Orthop 227: 44, 1988.

47. Reid DC, Hu R, Davis LA, Saboe LA: The nonoperative treatment of burst fractures of the thoracolumbar junction. J Trauma 28: 1188, 1988.

48. Riska EB, Myllynen P, Bostman. O: Anterolateral decompression for neural involvement in thoracolumbar fractures. A rewiev of 78 cases. J Bone Joint Surg 69 B: 704, 1987.

49. Roberts S, Menage J, Urban JPG: Biochemical and structural properties of the cartilage end plate and its relation te the intervertebral disc. Spine 14: 166, 1989.

50. Stokes IAF: Surface strain of human intervertebral discs. J Orthop Res 5: 348, 1987.

51. Tarlov IM: Acute spinal cord compression paralysis. J Neurosurg 36: 10, 1972.

52. Trafton PG, Boyd CA: Computed tomography of thoracic and lumbar spine injuries. J Trauma 24: 506, 1984.

53. Weidenbaum M, Farcy JPC: Surgical management of thoracic and lumbar burst fractures. Philadelphia, J.B. Lippincott Company, 914, 1991.

54. Whitesides TE: Traumatic kyphosis of the thoracolumbar spine. Clin Orthop 128: 78, 1977.

55. Willen JAG, Gaekwad UH, Kakulas BA: Acute burst fractures: A comprerative analysis of a modern fracture classification and pathologic findings. Clin Orthop 276: 169, 1992.

56. Wood GE, Hanley NE: Spinal trauma management update. Thoracolumbar fractures: An overview with emphasis on the burst injury. Orthopedics 15 (3): 319, 1992.