# INJURIES OF THORACOLUMBAR SPINE : TREATMENT PROTOCOL AND EFFECTS OF LAMINECTOMY

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***ABTRACT :***

*Laminectomy is a surgical technique which has been used as a decompressive procedure in the management of vertebral fractures with neurological injury since late 19th century. Despite its widespread use, the role of this technique in the treatment is still debatable. This paper reviewes the literature about the classification of vertebral injuries, treatment protocols and effects of laminectomy on neurological recovery and vertebral stability.*

## CLASSIFICATION OF INJURIES TO THE THORACOLUMBAR SPINE

The classification of injuries to the thoracolumbar spine should adress both the skeletal and neurologic injuries including presumptive mechanism of injury, the fracture anatomy and the degree of fracture fragment displacement which all may represent the spinal stability or instability, so that it could offer a composite picture of the nature of any specific injury (5) and is of real value in treatment (31). With the advent of computed tomography (CT) Denis complemented work from Holdsworth (30) and developed a three-column classification (12) which is probably the best classification scheme currently available. Ferguson and Allen (23) agree that the three-column view of spinal architecture is necessary to evaluate injury and stability properly. According to Denis the minor injuries represented by fractures of transverse processes, articular processes, pars interarticularis and spinous processes involve only a part of the posterior column and do not lead instability since stability of the middle column is the key to spinal stability in the three-column theory. Four different categories which include the more significant spinal injuries are given below.

**Compression Fractures :** This is the failure under compression of the anterior column. As the middle column is intact and acts as a hinge, minimal or moderate compression fractures are stable injuries. Severe compression fractures are considered to have instability of first degree and they have a risk of chronic kyphosis.

**Burst Fractures :** This results from failure under axial load of both the anterior and the middle columns originating at the level of one or both end-plates of the same vertebra. Severe burst fractures presenting neurologic deficits are unstable fractures of the third degree which has both a mechanical and a neurologic instability. Late neurologic instability, usually secondary to an unreduced burst fracture, distinguishes instability of second degree.

**Seat - Belt - Type Injuries :** These injuries represent a failure of both the posterior and middle columns under tension forces generated by flexion with its axis placed in the anterior column and are considered to have instability of the first degree.

**Fracture - Dislocations :** This is the most unstable (third degree) of injuries and presents with failure of all three columns under compression, tension rotation or shear (5, 12).

This classification scheme could offer a planning of management in thoracolumbar injuries. But the lack of accurate assesment of circumferantial soft tissue disruption of the vertebral body somewhat compromise the oparetor to plan a single operative approach which may minimize patient morbidity. It is hoped that magnetic resonance imaging technology (MRİ) and some others will probably offer detection of disk and Ligamentous injuries that should stimulate new classification of trauma to the spinal cord which include two precise classifications-one neurologic and one structural for every injury (5, 10).

A consideration of fracture stability, degree of canal compromise and neurologic evaluation of the patient is now significant factors in determining operative or nonoperative treatment.

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## NEUROLOGIC EVALUATION OF THE INJURED PATIENT

Absence of perianal sensation and loss of voluntary control of the sacral innervated muscles, to toe flexors, or the rectal sphincter can either be spinal shock or a complete lesion. If total loss of neurologic function persists for 24 to 48 hours in the presence of intact bulbocavernosus reflex, it is a complete lesion and these patients have no chance for neural recovery (26, 27, 28, 35, 38). If this lumbar and sacral areflexia is transient and persist less than 24-48 hours than it is a spinal shock. The signs of the termination of spinal shock is the return of bulbocavernosus reflex or anal wink, normal cord-mediated reflexes. The probable mechanism of this transient travmatic loss of cord function are reversible anatomical lesions such as adema or mild hemorrhage or a physiologic disturbance (e.g. contussion) without an anatomical lesion (26, 35, 38). The evidence of sparing of the sacral segments, such as retainced perianal skin sensation, toe flexion or sphincter control indicates an incomplete lesion (26).

## TREATMENT PROTOCOL IN THE INJURIES TO THE THORACOLUMBAR SPINE

**Stable Injuries:** A Stable spine is one that can withstand stress without progressive deformity or further neurologic damage (12, 51, 53).

In neurologically intact patients with stable fractures which were stated above (minor injuries to the posterion column and minimal of moderate compression fractures) non-operative treatment is shown to be succesful in the functional rehabilitation of the patient (26, 28, 34, 38, 40).

Incomplete or complete lesion is a rare occurance in stable injuries. A complete lesion could probably a vascular syndrome and an infarct as a result of this, if no lesion is visualized on radiographs and CT (6, 11, 30) it can also be a result of a transection of the cord at the time of injury which is especially seen in children (30). Treatment efforts should be directed to the patient's prompt rehabilitation as a paraplegic in this situation. Incomplete lesion of stable nature can occur when the site of compression is posterior, for example a displaced fracture to the neural arch. It is determined by CT and may have some benefit from decompression (27, 28, 35, 39, 47), then a partial laminectomy and a subsequent stabilization is performed to relieve the compressive lesion. Extensive laminectomy is not indicated since decompression can be achieved by removing fragments and debris through the fracture site (38) and such a laminectomy is considered to be deleterious as it converts the stable injury into an unstable one as a result of additional surgical disruption of the posterior column (31, 32, 39, 52, 56).

**Unstable Injuries:** An unstable spine is one that may lead to an increased deformity or an increased neurologic damage (12, 18, 19, 23, 53). The aim of treatment of unstable fractures is to achieve stability of the injured segment and healing of the fracture in a good position. In cases with neurological impairment, the treatment aims at achieving as complete and rapid a neurological restitution as possible. The unstable fractures of the thoracolumbar spine and their degrees of instability are stated above.

In neurologically intact patients with unstable fractures, early operative reduction and stabilization of the fracture is shown to have advantages in the preventation of neurological impairment and provides early mobilization, because reduction improves spinal stability (1, 6, 13, 14, 15, 17, 20, 21, 37).

Neurologic deficit secondary to unstable thoracolumbar injuries may be produced by displacement of fracture fragments or intervertebral disc, distortion of the spinal canal due to segmental displacement, penetration of foreign bodies, interruption of the vascular supply, extradural hemamtoma and stretching of the spinal cord (6, 11, 26, 36, 38).

If the cord syndrome is complete, early surgical reduction and stabilization is proposed for the ease of nursing care and prompt rehabilitation. Any neurological recovery is not anticipated, but early decompression may yield neurologic recorvery if the cord is not transsected but only compressed (1, 6, 16, 21, 33, 34).

In unstable fractures with incomplete neurologic deficit, the patient's prognosis would be better and some neurologic recovery is expected, if early proper decompressive procedure and stabilization is done (1, 6, 15, 17, 20, 21, 52, 56). In the severe compression fractures in which there is more than 50 % loss of vertebral height and kyphosis angle more than 30 degrees (12, 25, 40) angular displacement and the stretching of dural contents over the convexity of the displaced vertebral bodies may result in neurologic deficit (11, 25,

36). As shown in Figure 1, the site of neural compression is anterior over the convexity of the deformed spine.

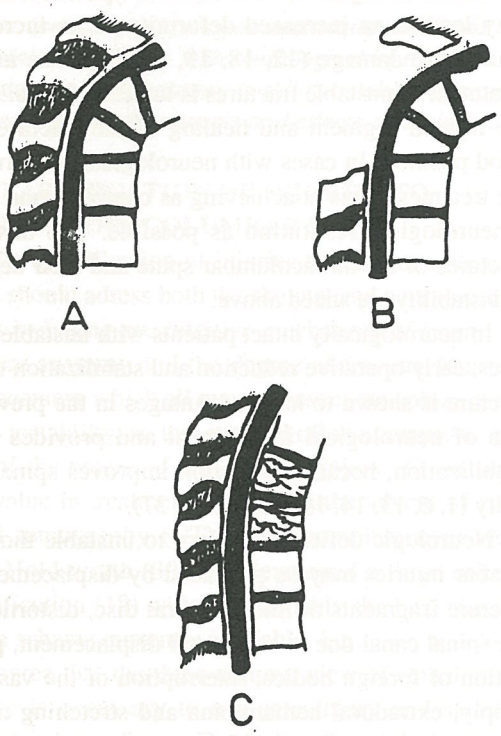


**Figure 1.** Schematic representation of dural compression due to angular spinal diplacement and the effect of laminectomy or reduction of decompression. A. Severe Angular deformity due to an anterior wedge fracture of more than one vertebral body may cause dural compression as a result of neural streching over the convexity of the deformed vertebral bodies. B. Laminectomy even at multiple levels. does nothing to relieve anterior dural compression produced by a severe angular deformity, C. Decompression can be achieved by a reduction which reduces the bony prominence and relieves anterior dural compression. This procedure does not impair spinal stability.

It is obvious that, lanimectomy alone is futile for correcting the stretching of dural contents and can not relieve anterior compression due to angular displacement (3, 11, 20, 25, 38). It is more appropriate to obtain neurological decompression and restitution by reduction of the deformity (1, 2, 3, 11, 1, 17, 20, 21, 24, 25, 41, 48). Displaced fragments compress the dural contents in most of the burst fractures, seat-belt-type injuries and fracture-dislocations which effect the middle and anterior, middle and posterior and all three columns respectively (5, 12). As the spinal cord can not and move up and down in the neural canal and accomadates very poorly the lengthening of the neural canal, it may also have damaged due to stretching during seat-belt-type injuries and fracture-dislocations (11, 51). When neural compression is due to displaced fragments, surgical decompression, stabilization and early rehabilitation should be done (1, 3, 6, 20, 21, 25, 48, 52, 56). Decompression can be achieved by direct operative removel or replacement of the displaced fragments by anterior and/or posterior surgical approach to the injured spine. Many methods including Harrington instrumentation (16, 25), rod-sleve technique (17, 21) pedicle screw plates (6) segmental spinal instrumentation (42), weiss springs (2, 24), "fixateur interne" (15). Alıcı posterior spinal instrumentation (1) are all based on anatomical reduction and internal stabilization by posterior approach and maintain it. If posterior approach fails, athough in rare cases, then anterior decompression is an effective technique for relief of dural compression (1, 3, 4, 16, 20, 34, 52).

In the early 1960s several authors used laminectomy as a routine decompressive procedure in the early management of spinal cord injuries in patients with incomplete lesions (8, 37, 50). Soon after, Carey (7) and Harris (29) Limited indications for decompression laminectomy and Carey emphasised that neurological deterioration after laminectomy was a significant risk. In the literature Guttmann (27, 28) was among the first who clearly recommended avoidance of laminectomy in patients with acute spinal cord injuries even with incomplete neural deficit, because it delays rehabilitation and is seldom benefical. In their large series of patients, Comarr and Morgan (9, 43) made a comparision between patients who were conservatively treated and those who had undergone laminectomy as a decompressive procedure. They bot showed that return of neural function is better, and the number of complications are less in the conservatively treated patients.

Morgan also noted that laminetectomy alone may cause further severel and irreversible damage to the spinal cord and should not be performed routinly as an exploratory or diagnostic procedure (43). The neurologic recovery rate was found to be the same whether or not the patients with incomplete lesions had a laminectomy (11, 16, 54). Stauffer showed that laminectomy had no benefical effect in patients with incomplete neurologic injuries from gunshot wounds of the spine (49). He also confirmed the decisions of Morgan about exploratory laminectomy and reported that the degree of neural impairment could not be correlated with the gross lesion described during surgical visualization since some of his patients had complete and incomplete lesions whose cords appeared to be normal and conversly some patients with incomplete paralysis whose records showed transected cords. Flesch (25) performed laminectomy as a decompressive procedure in patients with incomplete lesions, however he was convinced about the fruitless of laminectomy after seecing the postoperative myelograms of some of the patients as laminectomy did not always decompress the cord. One can see easily that laminectomy can not relieve anterior compression and even harmful as an isolated procedure since it exacerbates the spinal instability in fracture-dislocations as shown in Figure 2 (25, 38, 52, 56).

In this situation, reduction of the fracture and anatomical aligment of the spinal canal can most effectively relieve the anterior as well as the posterior site of compression (Figure 3) (1, 15, 16, 20, 21, 24, 25).

It has been showed that normal spinal cord or cauda equina occupies no more than about 40 % of area of a normal spinal canal (45). The large diameter of the spinal cord in relation to the relatively small volume of its neural contents assures that decompression by reduction is truly effective, especially in the lumbar spine (22, 45). Furthermore, reduction definitely enhances stability by allowing the dislocated neural arch processes to reengage (5, 25, 31, 37) thereby affording protection to uninjured neural structures.

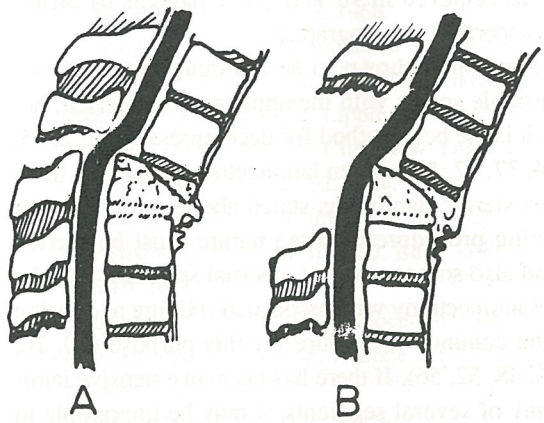


**Figure 2.** Diagram showing the mechanism of dural compression in fracture-dislocation and effect of Laminectomy. A. Anterior compression of the dura caused by the posterosuperior portion of the fractured vertebra, B. After removal of the posterior elements by Laminectomy, the anterior dural compression is unchanged and instability and increasing deformity may develop.

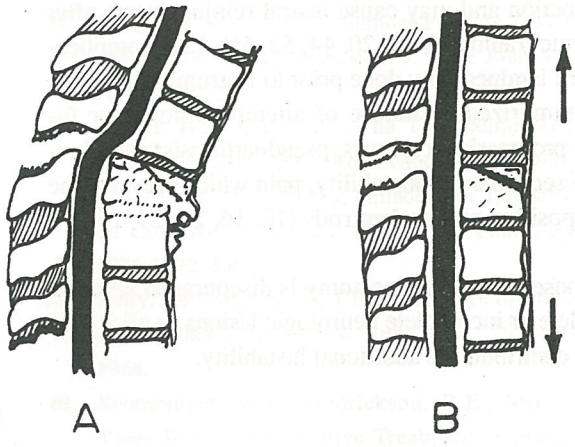


**Figure 3.** Sheumatic representation of dural compression due to fracture-dislocation and effect of reduction of decompression, A. Anterior compression of the dura caused by posterosuperior portion of fractured vertebra, B. Reduction and restoration of the spinal canal relieves both sides of compression and enhances spinal stability.

Edwards (21) reported that canal area was sufficiently restored in most of the injured segments of incomplete paraplegics by reduction. He showed that decompression was achieved in 96 % of these patients by means of intraoperative myelography.

Reduction is shown to be the treatment of choice for unstable spines with incomplete neural deficit, because it is the best method for decompression (1, 2, 15, 21, 24, 37, 52, 56). When laminectomy has done, there is no posterior stability as stated above, so a posterior stablizing procedure of some nature must be carried out and also some means of external splinting must be used. Laminectomy with associated rodding and fusion was the common procedure for this purpose (10, 16, 24, 25, 48, 52, 56). If there has been an extensive laminectomy of several segments, it may be impossible to achieve stability immediately even with Harrington rods, which are preferred for spine stabilization (16, 20, 34, 38, 52, 56). A transverse process fusion does nothing to enhance stability until bone union occurs. This means prolonged immobilization and nursing care. When fusion fails to develop (16, 20, 24, 25, 39, 46) progressive deformity (chronic instability) becomes as a frequent complication of unstable injuries. It has cosmetic significance, but also the patient may objet from pain at the deformity (44, 45) or in the compensatory curves. As time passes and deformity increases, angulation becomes an increasing treat to neural function and may cause neural reinjury years after the acute trauma (4, 13, 20, 44, 52, 55). Late complications of laminectomy done prior to instrumentation can be summarized as failure of attempted posterior fusions, progressive kyphosis, pseudoarthrosis, neural reinjury secondary to instability, pain which prevents the erect position and broken rods (10, 16, 20, 21, 25, 44, 55).

Consequently laminectomy is discouraged either in complete or incomplete neurologic lesions as all it generally contributes is additional instability.

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