A New Pattern of Spine Injury Associated with Lap-Type Seat Belts: A Preliminary Report

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THE automobile lap-type seat belt has been shown to be an effective means of reducing the severity of injury and the incidence of fatality.[1-3] An unrestrained passenger sustains injury when forces generated by a collision are dissipated on the skull, the thorax, or other exposed and fragile structures. A seat belt focuses collision forces on the pelvis and lower torso. Due to their rugged construction, the pelvis and lumbar spine are usually able to absorb the applied force without structural failure. Inevitably in some cases, applied forces will exceed the stress resistance of these musculoskeletal parts and will produce fracture and or fracture dislocation. A number of recent publications have reported pelvic fractures, lumbar fractures, and lumbar fracture dislocations in seat belt users.[4-6] Somewhat more common are reports of abdominal visceral injuries.[4,7-9] The visceral injuries are frequently associated with "improper" seat belt application (either too high, or too loose). Some authors have suggested that visceral injuries constitute a seat belt syndrome.[4] However, it must be pointed out that most of the reported injuries are not unique to the seat belt user and in no way differ from injuries produced by other forms of blunt trauma.[10]

This study was stimulated by experience with 17 lumbar spine injuries in seat belt wearers. In 10 of these (58 percent) we were able to identify a specific pattern of spine disruption which is extremely rare in unbelted individuals.[11] Material in this paper is limited to those 10 selected injuries. The seat belt injury pattern is characterized by marked spreading of posterior elements without the usually expected decrease in anterior vertebral height. This pattern suggests that the lumbar spine failed under tension with production of a distraction lesion. We believe that the lap-type seat belt is capable of modifying collision forces so that segments of the lumbar spine opposite the seat belt are subjected to tension force of sufficient magnitude to produce a distraction injury. This mechanism has heretofore not been considered in the production of lumbar injuries. Lumbar spine injuries are generally considered to be the result of compression or shear or torsion stress acting singly or in combination.[12,13] However, in the cervical spine the lesion produced by judicial hanging[14] is one in which tension stress is of obvious importance.

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Material

In the past year, the combined clinical services of the University of Michigan Section of Orthopaedics have treated 14 lumbar fractures and/or dislocations in seat belt users. Two additional cases were observed during the military experience of one of us (H.K.) and another case was under the care of Francis M. Howard, M.D., of Oak Lawn, Illinois. Lesions that were not demonstrable by x-ray films (sprains and strains) were not considered. Ten of these 17 cases exhibited the distraction pattern.

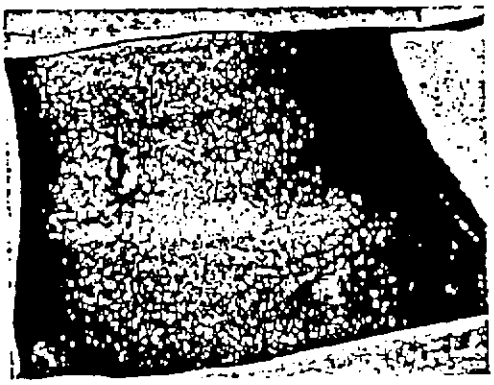

Fig. 1.—Seat belt contusion. Characteristically extends transversely across the lower abdomen at a level just cephalad to the anterior superior iliac spines.

Of these 10, there were 6 females and 4 males. Age ranged from 9 to 23 years. One patient had a total permanent T-12 paraplegia (the only peripheral neural deficit in all 10 cases). Three individuals had sevgre associated injuries, ranging from cerebral concussion to multiple bowel perforations. Nine of the 10 patients had a well defined lower abdominal seat belt contusion (Figure 1). In 8 patients spine disruption occurred through the L-2, L-3 segment or the L-2, L-3 interspace. This is in sharp contrast

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TABLE 1.—*Clinical Data on 10 Patients with Seat Belt Injuries*

Case No.	Age	Sex	Position in Vehicle	Impact Conditions	Spine Injury	Associated Injury	Treatment	Results
1	23	F	Right front seat	Estimated pre-impact speed of 50 MPH. Subject's vehicle struck the rear of a stationary automobile.	"Chance" type of fracture of L-2 (Fig. 11). No neural deficit.	Lower abdominal seat belt contusion (Fig. 4).	Three point hyperextension brace.	Returned to previous occupation. Symptom-free.
2	15	M	Rear seat	Pre-impact speed of 45 MPH. Vehicle left the road, ran down a four foot embankment, and struck a tree where it came to rest.	Dislocation of L-1 on L-2 with gross displacement and marked upper lumbar gibbus. No neural deficit	Seat belt contusion; fracture of left radius and ulna; complete transverse rupture of all left lateral abdominal muscles, including rectus abdominus and anterior 1/3 of latissimus dorsi; complete transection of jejunum and small bowel mesentery; full thickness tear of ascending and descending colon; contusion of omentum.	Abdominal exploration; subtotal small bowel resection & repair of colon lacerations. Two months post-injury underwent lumbar exploration and posterior fusion of L-1 to L-2.	Residual upper lumbar gibbus. Enjoys unrestricted activities. Symptom-free.
3	17	F	Driver	Pre-impact speed of 50 MPH. Vehicle struck a bridge abutment.	Dislocation of L-1 on L-2. No neural deficit.	Seat belt contusion; multiple minor contusions of extremities and head; protruded intervertebral disc, L-1, 2.	Lumbar exploration; posterior fusion of L-1 to L-2.	Unrestricted activities. Symptom-free.
4	19	M	Rear seat	Pre-impact speed of 60 MPH. Vehicle left the road and struck a tree.	"Chance" type of fracture of L-3. No neural deficit	None	Extension body cast.	Unrestricted activities. Symptom-free.
5	20	M	Right front seat	Pre-impact speed of 50 MPH. Head-on collision with another automobile.	"Chance" type of fracture of L-3. No peripheral neural deficit.	Seat belt contusion; cerebral concussion.	Extension body cast.	Lost to follow-up.
6	9	F	Right front seat	Combined collision speed of 60 MPH. Head-on collision with another automobile.	Dislocation of L-2 on L-3. No neural deficit.	Seat belt contusion; undisplaced basilar skull fracture; fracture of superior alveolar ridge with loss of central upper incisors; rupture of ileum discovered at laparotomy 14 days post-injury.	Subtotal small bowel resection; lumbar exploration; posterior fusion L-2 to L-3.	Persistent gastrointestinal symptoms. No skeletal symptoms.
7	19	F	Right front seat	Estimated combined pre-impact speed of 65 MPH. Head-on collision.	Complete dislocation of T-12 on L-1. Total paraplegia at T-12.	Seat belt contusion; multiple extremity contusions.	Early laminectomy. Reduction not attempted. No neural recovery.	Severe gibbus decubiti. Ambulates with bilateral braces and crutches
8	19	F	Right front seat	Estimated pre-impact speed of 45 MPH. Collided with a stationary vehicle.	Dislocation of L-2 on L-3.	Seat belt contusion.	Early open reduction; internal fixation & fusion.	No residual deformity. Unrestricted activity. Symptom-free.
9	23	F	Right front seat	Pre-impact speed of 55 MPH. Struck a stationary vehicle.	Dislocation of L-2 on L-3. No neural deficit.	Seat belt contusion; right olecranon fracture.	Early open reduction; internal fixation & fusion.	Failure of fusion. Persistent back pain. Refusion was successful with relief of symptoms.
10	22	M	Right front seat	Pre-impact speed of 45 MPH. Struck a truck which was making a left hand turn across the subject's lane.	Dislocation L-3 to L-4. No neural deficit.	Scalp and face laceration; cerebral concussion; fracture left 5th metacarpal; seat belt contusion.	Open reduction; internal fixation & fusion.	Still in cast. Doing well.

to the usual lumbar injury in which the site of disruption is T-12, L-1 in 60 to 80 percent of cases.[11,12] Only 1 of our 10 patients was in the driver's seat, 2 were rear seat occupants, and the remaining 7 occupied the front right seat. All these injuries were the result of severe accidents with an average estimated pre-impact speed in excess of 50 miles per hour. For additional clinical data refer to Table 1.

Pattern

A unifying feature in our 10 cases was a remarkably consistent pattern of spread between posterior elements while anterior compression was either absent or only minimally present. In some cases gross separation of posterior elements was permitted by rupture of the posterior ligaments without any evidence of neural arch fracture. Roentgenograms characteristically showed marked widening of the

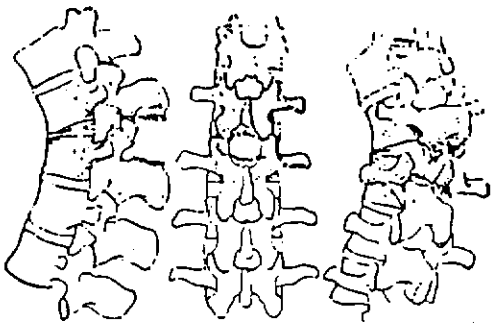

Fig. 3.—Extensive ligament tearing is an outstanding feature of this variety of the seat belt injury pattern. Exploration reveals gross inability due to rupture of lumbodorsal fascia, interspinous ligaments, ligamentum flavum, facet joint capsules, and posterior longitudinal ligament. In one patient (Case 3) there was a protruded disc.

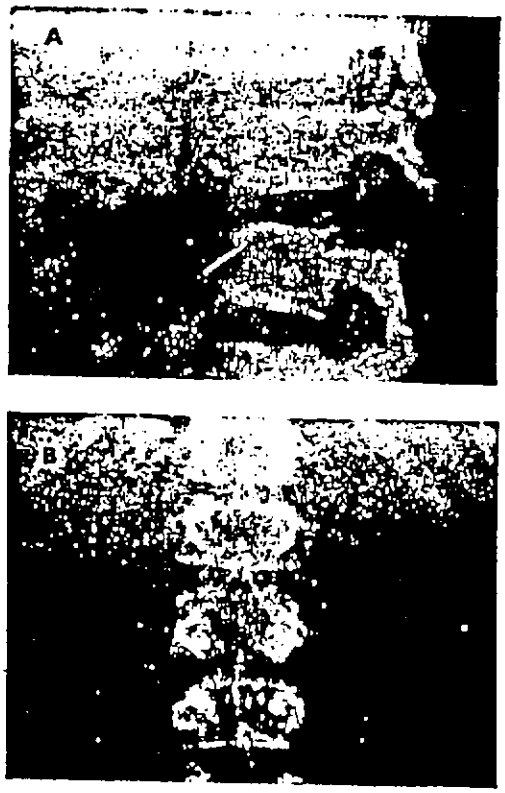

Fig. 2.—This variety of seat belt injury is essentially a pure dislocation. (A) There is obvious spread between neural arches and marked widening of the intervertebral nerve foramen. Posteriorly, the intervertebral disc space is increased. The tiny anterior compression defect is probably insignificant. (B) The increased interspinous distance is obvious as are the bilateral facet dislocations. There is no suggestion of rotational displacement.

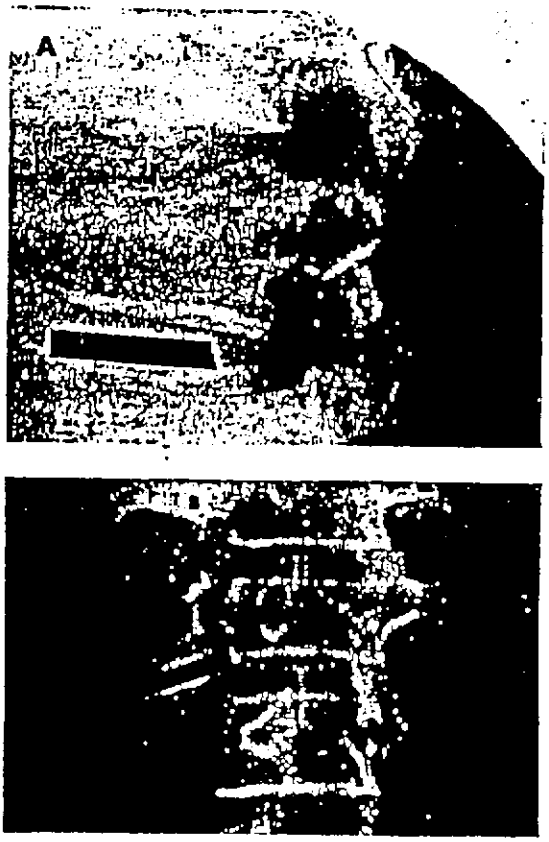

Fig. 4.—"Chance" fracture. (A) Lateral projection shows no interspinous spread and no enlargement of the nerve root foramen. There is a horizontal fracture which involves the entire segment from anterior to posterior and passes through bone all the way. There are no major ligament rupture and no intervertebral disc lesion. (B) Oblique projection demonstrates the pedicle fractures to best advantage, as well as the fracture line passing through transverse processes.

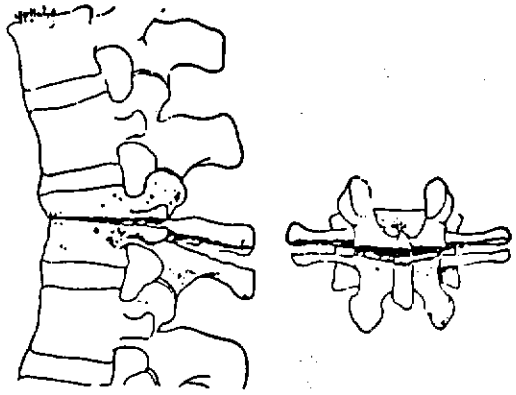

Fig. 5.—A composite of our 3 cases of "Chance" fracture. This highlights the unique and highly characteristic features of this injury.

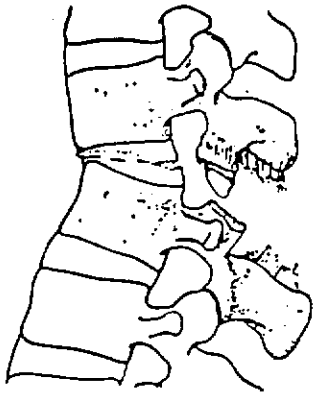

Fig. 7.—This is essentially a dislocation without any degree of vertebral body compression. There is, however, an avulsion of the facets with intact facet joint capsules.

interspinous distance and increased height of the intervertebral foramen, with only minimal anterior compression (Figure 2). Surgical exploration in these cases demonstrated complete rupture of interspinous ligaments, interlaminar ligaments, ligamentum flavum, facet joint capsules, and posterior longitudinal ligaments (Figure 3). In one individual (Case 3) there was a protrusion of the nucleus pulposis. This purely ligamentous variety of the "dislocation" pattern was noted in 3 cases.

In three individuals, the line of disruption passed entirely through bone with a horizontal fracture line (not a compression fracture) through the vertebral body continuing posteriorly through the pedicles, transverse processes, lamina, and spinous process. X-ray films showed no evidence of increased interspinous distance (Figure 4). Posterior spread occurred between the fragments of the fractured segment. The posterior spine ligaments remained intact (Figure 5). This is an extremely unusual lesion first described by C. Q. Chance[18] in 1948 in 2 cases. A relation between this injury and use of a lap-type seat belt was noted in a single case report by Howland, Curry, and Buffington[9] in 1965. Three of our patients exhibited this exact pattern and equal in number all previously reported "Chance" fractures.

Four of our patients showed posterior ligament rupture as well as avulsion fracture. This variety

Fig. 6.—There is gross separation of neural arches due to ligament rupture. An intact posterior longitudinal ligament has produced an avulsion fracture of the posterior portion of the vertebral body.

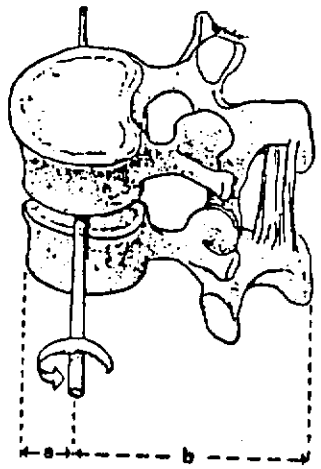

Fig. 8.—Normally, flexion and extension of the intact lumbar spine occur around an axis which passes through the center of the nucleus pulposis. The distance from this axis to the anterior margin of the body (a) is one-fourth the distance from the axis to the tip of the spinous process. Applying the law of the lever, it is apparent that the anterior vertebral body will be subjected to compression force four times as great as the tension force which is generated in the interspinous ligaments.

usually exhibits rupture of interspinous ligaments and ligamentum flavum combined with avulsion fracture of the posterior vertebral body (Figure 6) or an avulsion fracture of articular facets (Figure 7).

Discussion

The most common lumbar fracture is the simple compression fracture at the thoracolumbar junction produced by a combination of vertical load and hyperflexion.[15] If the lumbar area is subjected to a vertical load, the spine will be forced into flexion. The flexion axis of an intact lumbar spine passes through the nucleus pulposis[13] (Figure 8). As flexion occurs around the axis, that portion of the vertebral body anterior to the axis is subjected to compression while that portion of the vertebra posterior to the axis is subjected to tension. In most individuals the distance b is four times the distance a. The anterior vertebral body is, therefore, subjected to compression forces four times as great as the tension forces on the posterior ligaments.[13] For this reason, when subjected to pure flexion forces, the vertebral body will be crushed long before posterior ligament rupture can occur (Figure 9).

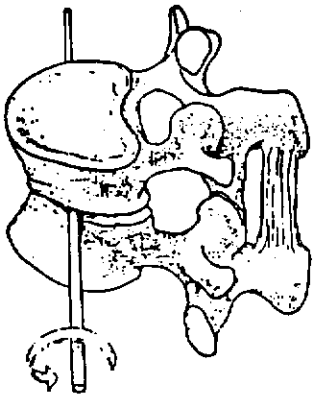

Fig. 9.—If hyperflexion occurs around the normal flexion-extension axis, then an anterior vertebral compression fracture occurs. More flexion produces more compression, not dislocation.

Failure during distraction is the simplest mechanism that could account for the pattern of disruption and displacement that was observed. Primary tension stress has to our knowledge not been considered instrumental in the production of any lesion in the lumbar spine. This is not surprising since it is difficult to imagine a situation that would subject the lumbar area to tension stress of significant magnitude to produce structural failure. It is our opinion that the spine injuries described in this paper are true distraction injuries of the lumbar spine.

If the wearer of a lap-type seat belt is subjected to sudden deceleration, his body will bend over the restraining belt (Figure 10). In this pathologic situation, the axis around which flexion occurs has moved forward and passes through the point of contact between the belt and the abdominal wall (Figure 11). The entire spine is now posterior to

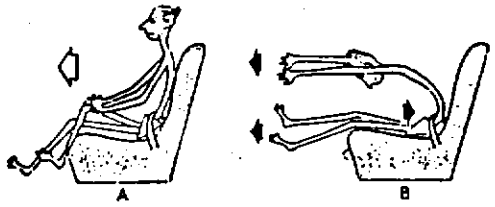

Fig. 10.—The seat-belted passenger, when subject to sudden deceleration is literally wrapped around the seat belt. Hyperflexion in this situation occurs around the belt which is necessarily anterior to the normal flexion extension axis.

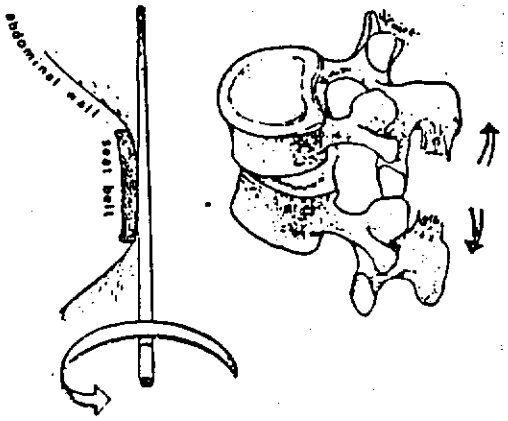

Fig. 11.—When hyperflexion occurs around the seat belt, all parts of those segments opposite the belt are subjected to tension stress with resultant distraction. There is no compression. More flexion produces more distraction, not compression.

the flexion axis so all of its components are subjected to tension stress. If a bone-ligament unit in a healthy young person is subjected to tension stress, the lesion most often encountered is an avulsion fracture rather than ligament rupture. With this in mind, we believe that the "Chance" fracture is most likely an example of a pure tension injury. In our cases that exhibit the other varieties of disruption, the primary tension stress may have been significantly modified by shear or torsion. Obviously this hypothesis requires laboratory confirmation. Studies currently in progress are expected to shed additional light on the pathomechanics of various patterns of spine injury.

Summary

Of 17 x-ray-demonstrated spine injuries in seat belt wearers, 10 were found to exhibit an unusual and consistent pattern of separation between posterior elements without the unusually expected decrease in anterior vertebral height.

It is suggested that primary tension stress is responsible for the unusual pattern seen in these injuries.

The mechanism by which large tension forces may be brought to bear on the lumbar spine has been described.

The authors believe that this report is in no way an indictment of the lap-type seat belt. All cases studied were the product of very severe collisions. We are convinced that if these individuals had not been wearing seat belts, they would not have survived. It is likely that these injuries would not have occurred if the patients had been wearing shoulder type seat belts. The possibility that the shoulder harness might focus similar stress at the cervical dorsal junction with production of a far more serious spine injury in the highly mobile and relatively unstable cervical spine is a matter for conjecture.

References

1. Gikas, P. W., and Huelke, D. F.: Causes of Death in Automobile Accidents, Can Seat Belts Really Save Lives. Michigan Med. *63*:351, 1964.
2. Huelke, D. F., and Gikas, P. W.: Determination of Seat Belt Effectiveness for Survival in Fatal Highway Accidents. *Proceedings of Seventh Stapp Car Crash Conference*. Springfield, Ill.: Charles C Thomas, 1965, pp. 1–19.
3. Tourin, B., and Garrett, J. W.: Safety Belt Effectiveness in Rural California Automobile Accidents. Automotive Crash Injury Research of Cornell University, New York, February 1960.
4. Fish, J., and Wright, R. H.: The Seat Belt Syndrome—Does It Exist? J. Trauma *5*:746, 1966.
5. Garrett, J. W., and Braunstein, P. W.: Seat Belt Syndrome. J. Trauma *2*:220, 1962.
6. Howland, W. J.; Curry, J. L.; and Buffington, C. B.: Fulcrum Fracture of the Lumbar Spine. J.A.M.A. *193*:240, 1965
7. Cocke, W. M., and Meyer, K. K.: Splenic Rupture Due to Improper Placement of Automobile Safety Belt. J.A.M.A. *183*:693, 1963.
8. Kulowski, J., and Rost, W. B.: Intra-Abdominal Injury from Safety Belt in Auto Accident. A.M.A. Arch. Surg. *73*:970, 1956.
9. Williams, J. S.; Lies, B. A. Jr.; and Hale, H. W. Jr.: The Automobile Safety Belt, In Saving a Life May Produce Intra-Abdominal Injuries. J. Trauma *6*:303, 1966.
10. Allen, J. G.; Harkins, H. N.; Moyer, C. A.; and Rhoads, J. E.: *Surgery Principles and Practice*, 3rd ed. Philadelphia: J. B. Lippincott, 1965, pp. 901–4.
11. Kaufer, H., and Hayes, J. T.: Lumbar Fracture-Dislocation: A Study of Twenty-One Cases. J. Bone Surg. *48A*:712, 1966.
12. Holdsworth, F. W.: Fractures, Dislocations, and Fracture Dislocations of the Spine. J. Bone Surg. *45B*:6, 1963.
13. Roaf, R.: A Study of the Mechanics of Spinal Injuries. J. Bone Surg. *42B*:810, 1960.
14. Schneider, R. C.; Livingston, L. E.; Cave, A. J. E.; and Hamilton, G.: "Hangmans Fracture" of the Cervical Spine. J. Neurosurg. *2*:141, 1965.
15. Nicoll, E. A.: Fractures of the Dorsolumbar Spine. J. Bone Surg. *31B*:376, 1949.
16. Chance, G. Q.: Note on a Type of Flexion Fracture of the Spine. Brit. J. Radiol. *21*:452, 1948.