Lumbar Spinal Strains Associated with Whiplash Injury: A Cadaveric Study

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FROM ABSTRACT

Objectives:
To study and quantify the effects of rear-end collision on the lumbar spine.

Design:
The lumbar spine of a cadaver was instrumented with rosette strain gauges applied on the lateral and anterior surfaces of T12, L2, and L4. Biaxial accelerometers were mounted on L1, L3, and L5. The cadaver was seated, restrained, and subjected to rear impacts of 5g and 8g.

Results:
The anterior shear strains had a biphasic shape. Spinal strains peaked at the T12 at approximately 120 and 370 msec, whereas in the L4 vertebra, it peaked at 200 and 380 msec. The anterior strain pattern of the L4 and T12 vertebrae were in diametrically opposite directions.

In the second set of tests (8g experiment), the acceleration forces and strains pattern were similar to the 5g test but of higher magnitude. The principal anterior strain was 480 mm/m for 5g and 530 mm/m for 8g; the lateral shear strain was 680 mm/m and 1500 mm/m in the 5g and 8g experiments, respectively.

Conclusions:
Forces generated during simulated whiplash collision induce biphasic lumbar spinal motions (increased-decreased lordosis) of insufficient magnitude to cause bony injuries, but they may be sufficient to cause soft-tissue injuries.

THESE AUTHORS ALSO NOTE:

Up to half of those involved in a whiplash accidents may develop low back pain.

“Frequently, seemingly implausible claims of lumbar injuries are mentioned after low-speed rear-end collisions, even when little vehicular damage is reported.”

“Apparently, rear-end collisions lead to soft-tissue injuries with paucity of findings on physical examination, negative radiograph studies, profuse symptomatology, and prolonged disability.”
Because there is scant biomechanical data dealing with the low back after whiplash injuries, these authors conducted this cadaver experiment to quantify lumbar spinal strains during rear impact collisions.

METHODS

The authors attached strain gauges to an unembalmed seated restrained cadaver, and exposed it to an acceleration of 5g (delta V of 13 km/hr \([13 \times 0.62 = 8 \text{ mph}]\)) and 8g (delta V of 19 km/hr \([19 \times 0.62 = 12 \text{ mph}]\)).

RESULTS

Spinal x-rays after exposure revealed no bony injuries after the experiments.

Lumbar spinal acceleration started at about 17 msec after impact and reached peak horizontal acceleration at about 80 msec.

The T1 vertebra reached peak acceleration at about 125 msec.

Spinal peak strains occurred at 200-400 msec.

T1 vertical acceleration reached 3g, and was probably the result of the head's movements on the spine.

The anterior shear strains at the vertebral bodies started at about 30 msec after impact and peaked at 120 - 380 msec.

The 8g rear impact tests showed similar acceleration forces and strain patterns to the 5g tests but were of higher magnitude (some magnitudes were more than doubled).

At 8g, as the body first translated rearward into the seat back, the seat back rotated backwards 5-10 degrees.

At 8g, as the spine was loaded into the seat it straightened because of extension of the thoracic spine, lifting the shoulder, neck, and head, causing axial spinal loading.

DISCUSSION

These authors proved that there are both horizontal and vertical spinal forces that result from a strictly horizontal force applied experimentally.
This vertical axial loading is explained by two mechanisms:

(1) ramping (sliding-up) of the torso against the seat.

(2) straightening of the spine from decreased thoracic and lumbar curves.

“These findings are in accordance with recently published literature suggesting that a most important mechanism leading to cervical symptoms after whiplash is spinal ramping.”

CONCLUSIONS:

(1) The vertical loads measured were too small to cause lumbar spine injury.

(2) Excessive lumbar spinal motions that could injure the lumbar spine were not noted.

The authors “propose that irritation of or injury sustained by the richly innervated spinal soft tissues (i.e., muscles, ligaments, capsules) plays an important role in the pathogenesis of lumbar pain after whiplash injury.”

“This proposition is supported by work of others who performed lumbar spinal segment shear tests and showed that soft-tissue injuries occurred with a shear load as low as 1200 N.”

KEY POINTS FROM DAN MURPHY

(1) Maybe half of those involved in whiplash accidents develop low back pain.

(2) Claims of lumbar injuries are mentioned after low-speed rear-end collisions, even when there is little vehicular damage.

(3) The forces generated during simulated whiplash collision are too low to cause lumbar spine bony injuries.

(4) The forces generated during simulated whiplash collision may be sufficient to cause lumbar spine soft-tissue injuries.

(5) Rear-end collision soft-tissue injuries often have a paucity of findings on physical examination and x-rays, but can have profuse symptomatology, and prolonged disability.

(6) Spinal x-rays often revealed no bony injuries.
(7) Lumbar and thoracic initial acceleration and peak accelerations occurs prior to protective stretch reflex activation [which other references note to be about 200 ms].

(8) The axial (vertical) spinal loading was greater in the upper thoracic and cervical spines than in the lumbar spine.

(9) The most important mechanism of cervical injury from whiplash may be spinal ramping that causes the axial (vertical) spinal loading.

(10) Seat back spinal contact during rear-end collisions will straighten the thoracic and lumbar spinal curves.

(11) Irritation or injury to the richly innervated spinal soft tissues (i.e., muscles, ligaments, capsules) plays an important role in the pathogenesis of lumbar pain after whiplash injury.