Rear-end impacts: vehicle and occupant response

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Charles Davis is a chiropractor from Southern California, and a friend of mine. This article is a REVIEW OF THE LITERATURE, containing 171 references. He makes the following statements, with references:

Most low-speed rear-end impacts will not show much vehicle damage or positive standard orthopedic neurological findings.

There is a lack of relationship between vehicle damage and occupant injury.

"Research with a human volunteer found that with a pre-impact automobile speed of 8.2 mph, forces applied to the head were 2.5 times greater than the forces applied to the vehicle."

A 1989 accident investigation team at the University of British Columbia in Vancouver defined low-speed impacts as 5 - 12.4 mph.

A 1989 article found that impact speeds of 14-15 km/hr (8.7-9.3 mph) were required to initiate structural deformation in the vehicle.

Bumper isolator mounting bolts move in their adjustment slots for impact speeds less than 14 km/hr [8.7 mph], but no visible metal deformation takes place.

Injury claims can easily dwarf material damage claims.

Current federal bumper requirements are designed to reduce vehicle damage, not personal injury (U.S. Federal Motor Vehicle Safety Standard 215). **[Important]**

The isolator is a common type of bumper with a piston and cylinder assembly mounted between the bumper and the car. Scrape marks on the isolator piston tubes may indicate the amount of energy delivered by the contact vehicle. This cannot be done with automobiles with foam core bumpers.

Subjects in low-speed testing are usually healthy male volunteers. Subjects have been tested in tensed and relaxed positions before the crash impacts. Although the subjects may have tried to relax, they knew what was going to happen, which significantly affects the magnitude of the startle response.

The deep multifidus muscle may not respond to surface EMG recordings, and may be the most important segmental muscle, as it withdraws the slack in the capsule facet joint.

At very low speeds of 1-2.5 mph, neck injuries can occur if either the headrest or an "elastic bumper stay" do not perform their protective functions.

"Occupant offset" from the seatback that occurs when the occupant sits forward relative to the seatback and head restraint is a problem.

Injury is probably reduced if the head is firmly fixed against the head restraint at the time of impact.

If the head is not in direct contact with the head restraint at the time of impact, injuries are possible even with low-speed impacts. **[Important]**

The ideal situation would be if the back of the head were in constant contact with a firm head restraint.

The author cites research that indicates that it is rare for a head restraint to be positioned so as to provide this optimal level of protection.

For a head restraint to be effective, it must stop the rearward head-neck motion very early on.

Even with optimal head restraints, the "ramping" effect of low-speed rear-end impacts eliminates most of the protective benefit from the head restraint and may even increase the severity of the injuries through the fulcrum effect. The ramping effect on the occupant causes the torso, shoulders, neck and head to rise along the path of the seat belt by 3.5 in. **[Important]**

During injury, there is a sudden unexpected stretching force of the skeletal muscles induced by a barrage of impulses from receptors in muscles and joint capsules that travel to the central nervous system and reflexively activate alpha motor neurons. This alters the segmental and suprasegmental controls of muscle tone, resulting in long-term painful muscle tension. This is known as the "jolt syndrome."

Extracervical symptoms related to whiplash-type trauma can be related to a stretching of the vertebral artery during injury.

Whiplash extension trauma can damage the spinal dorsal root ganglion.

In a rear-end impact, the neck forms an S-shaped curvature at 50-75 msec after accelerating motion, with the lower cervical spine in hyperextension while the upper cervical spine in flexion. At this time, the lower cervical joints are in extreme hyperextension, far beyond their normal physiological limits, causing damage.

Ventroflexion of the spine causes elongation which pulls taut and elongates the pons-cord tract and associated nerve roots.

The cervical facet capsules are innervated with both mechanoreceptors and nociceptive nerve fibers.

The human cervical intervertebral discs are innervated with both mechanoreceptors and nociceptive nerve fibers.

Cervical facet joint pain is common among patients with chronic neck pain after whiplash injury.

Slight rotation of the head at impact prestresses the capsules of the zygapophyseal joints, intervertebral discs and the alar ligament complex, rendering them more susceptible to injury.

Women have smaller spinal canals (central canal stenosis) as compared to men and consequently exhibit a greater persistence of symptoms.

The rectus capitis posterior minor muscle (RCPM) undergoes fatty degeneration in patients with whiplash-like symptoms, compromising its ability to protect the spinal dura during extension mechanisms.

Subjects with whiplash injury have a deficit in their ability to reproduce a position of the neck; they tend to overshoot the target and are often inaccurate in their assessment of neutral position. This may be caused by inadequate receptor potentials from the injured joints and muscles.

Clinical instability and abnormal motion can be demonstrated with flexion/extension radiographs or video fluoroscopy.

Extracervical symptoms may occur after whiplash trauma from injury to the cervical portion of the sympathetic chain or the 9th-12th cranial nerves as they exit from the base of the skull and pass into the deep cervical fascia.

Vertigo, disturbed eye movement and disturbed vision have been found after whiplash injuries and are often cervical in origin.

Mild traumatic brain injury occurs after whiplash injury, these patients have normal neurological examination, the injury may not involve loss of consciousness, and CT and MRI findings are usually normal.

Chronic whiplash pain is a consequence of windup, central sensitization and synaptic plasticity.

Accident reconstructionists perform energy-of-crush analysis by using computer programs. These models are accurate for the intermediate impact speeds of 20-50 mph, but at low crush speeds predictions are poor. **[Important]**

"Studies have shown that low-speed impacts result in far greater forces being applied to the neck and head than to the vehicle, and that such forces can cause significant neck injuries even when the impact is less than that which is required to damage a vehicle." **[Very Important]**

"There seems to be a consensus that 4-5 mph is probably the threshold for cervical injury in healthy people in testing conditions, especially if proper head support is available, and it may be as low as 2.5 mph." **[Very Important]**

"There does not seem to be an absolute speed or damage to a vehicle with which a specific person may experience injury." **[Important]**

Pain from cervical facet joints may take 3 months to develop in 4% of cases and up to 2 years with perineural fibrosis. **[Very Important]**

"It is very difficult for an ingenuine individual to fake a psychological profile typical of a whiplash patient, and there have been no scientific studies showing that patients are 'cured by verdict'."

25% of whiplash injuries will become chronic when there is damage to the facet joints, the disc or alar ligaments.

"There is a lack of relationship between vehicle damage and occupant injury." **[Very Important]**

"Sufficient injury forces may be produced without visible vehicle damage or radiographic findings." **[Very Important]**

"In low-speed rear-end collisions it takes more than a few photographs of the vehicles involved to conclude that the forces of the collision were not sufficient to cause injury. Each incident and occupant must be individually evaluated." **[Very Important]**