SECOND EDITION

BIOMECHANICS *of* MUSCULOSKELETAL INJURY

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can impinge on and injure the spinal cord. The level of cervical spinal cord injury (C1-C7) is critical in determining the degree of neuromuscular dysfunction and motor deficit (Gardner 2002; Watkins and Watkins 2001). The following summarizes function and deficits according to cervical level.

- **C1-C3**—Limited movement of head and neck. Complete paralysis of trunk and extremities (quadriplegia). Patient requires ventilatory support.
- **C3-C4**—Usual head and neck control. C4 may shrug shoulders. Patient requires initial ventilatory assistance.
- **C5**—Head, neck, and shoulder control. Patient can bend elbows and supinate arm to turn palms up.
- **C6**—Head, neck, shoulder, and elbow flexion control. Patient can turn palms up and down and can extend wrist.
- **C7**—Added ability to extend elbow.
- **C8**—Added strength and some finger control, but lack of fine precision hand movements.

Whiplash-Related Injuries

Of all cervical disorders, whiplash-related injuries are among the most common and most misunderstood. The very definition of whiplash is controversial. In one context whiplash describes an injury mechanism, whereas in another it refers to a clinical syndrome. This latter usage is inappropriate because it lacks specificity.

In 1995, the Quebec Task Force on Whiplash-Associated Disorders defined whiplash as "an acceleration–deceleration mechanism of energy transfer to the neck which may result from rear-end or side impact, predominately in motor vehicle accidents, and from other mishaps. The energy transfer may result in bony or soft tissue injuries (whiplash injury), which may in turn lead to a wide variety of clinical manifestations (whiplash-associated disorders)" (Cassidy et al. 1995, p. 22).

In recent decades many studies have been conducted to determine the mechanisms of whiplash. Although these studies have been of variable quality (Kwan and Friel 2003; McClune et al. 2002) and have raised concerns about sampling, experimental design, and data interpretation, we have a much better understanding of whiplash mechanisms than we did not long ago.

Whiplash typically has been characterized as involving a hyperextension mechanism. In this view, at the instant of impact, the head remains stationary (according to Newton's first law) while the vehicle is violently pushed forward. When the occupant's trunk and shoulders are accelerated anteriorly, the head is forced into hyperextension. Once its inertia is overcome, the head is thrown (whiplashed) forward into flexion.

Recent work has shown that the hyperextension model is too simplistic and does not adequately describe the complex motion of the cervical spine during whiplash. "The critical revision brought about by modern research into whiplash is that it is not a cantilever movement that is injurious; i.e., it is not an extension–flexion movement of the head, as was commonly believed previously. Rather, within less than 150 ms after impact, the cervical spine is compressed. During this period the cervical spine buckles; upper cervical segments are flexed while lower segments extend around abnormally located axes of rotation" (Bogduk and Yoganandan 2001, p. 272). The simultaneous upper cervical flexion and lower cervical extension results in an S-shaped neck curvature as shown in figure 8.19 (50 ms and 75 ms). The S-shaped configuration happens within 75 ms after impact and then gives way to a C-shaped hyperextension curvature (Grauer et al. 1997). Both the lower cervical spine and upper cervical spine are at risk of injury from the rear-impact mechanism (Panjabi, Pearson et al. 2004).

Although usually considered a sagittal plane injury caused by a rear-end impact, whiplash can also result from lateral or frontal forces that exact their own unique pattern of injury. In addition, motion of the neck sometimes is not confined to a single plane. If a driver is looking to the side at the instant of impact, for example, the injury mechanism involves a combination of hyperextension and rotation. In this case the rotation is enhanced by the impact forces prior to cervical hyperextension, and cervical structures are prestretched and more predisposed to severe injury.

At first glance, whiplash might appear a simple injury mechanism. However, "in an individual accident there is likely to be a complex interaction between different forces depending upon the speed and direction of impact and the attitude of the head and neck" (Barnsley et al. 1994, p. 288). Many structures can be injured in whiplash accidents: structures in the brain, temporomandibular joint, muscles, spinous ligaments, intervertebral discs, vertebral bodies, and facet (zygapophyseal) joints (e.g., Davis 2000; Ito et al. 2004; Panjabi, Ito et al. 2004; Pearson et al. 2004). Various mechanisms and potential injury sites are shown in figure 8.20. Whiplash-associated disorders can manifest as both clinical and psychosocial symptoms (Eck et al. 2001). Possible symptoms are listed in table 8.5.

In summary, not all victims of whiplash sustain injuries. "Whether or not a victim sustains an injury is a function of multiple factors: the magnitude of the impact, their posture at the time, their anatomy, and the material strength of the components of their cervical spine" (Bogduk and Yoganandan 2001, p. 272).

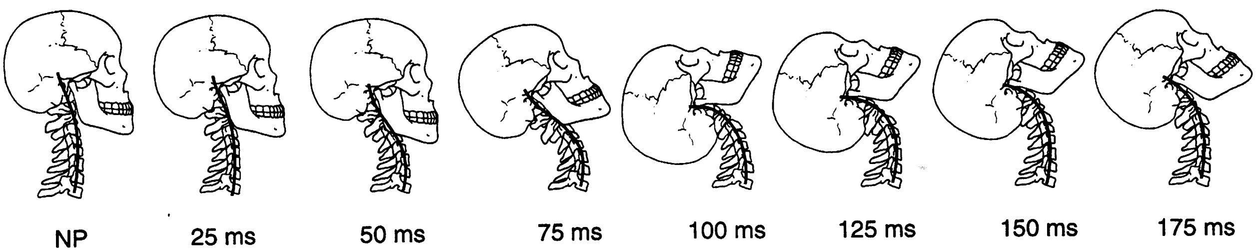


Figure 8.19 S-shaped neck curvature resulting from simultaneous upper cervical flexion and lower cervical extension. NP = neutral position.

Reprinted, by permission, from J.N. Grauer et al., 1997, "Whiplash produces an S-shaped curvature with hyperextension at lower levels," *Spine* 22(21): 2492.

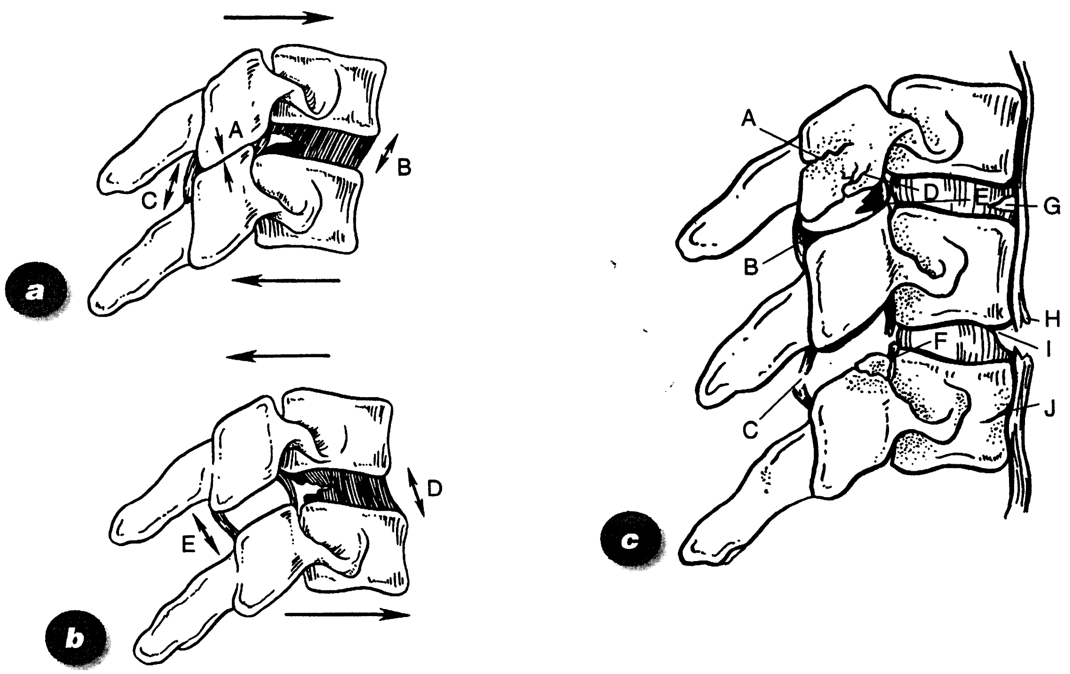


Figure 8.20 Potential mechanisms and injury sites for whiplash-related injuries. Shear forces affecting a spinal motion segment. *(a)* Translation of the superior vertebral body anteriorly relative to the inferior. This movement stresses the articular surfaces of the zygapophyseal joint (A), the anterior annulus fibrosus (B), and the zygapophyseal joint capsule (C). *(b)* Translation of the superior vertebral body posteriorly relative to the inferior body, which stresses the intervertebral disc (D) and the zygapophyseal joint capsules (E). *(c)* Common lesions affecting the cervical spine following whiplash injury. A, articular pillar fracture; B, hemarthrosis (hemorrhage into a joint) of the zygapophyseal joint; C, rupture or tear of the zygapophyseal joint capsule; D, fracture of the subchondral plate; E, contusion of the intra-articular meniscus of the zygapophyseal joint; F, fracture involving the articular surface; G, tear of the annulus fibrosus; H, tear of the anterior longitudinal ligament; I, end-plate avulsion fracture; J, vertebral body fracture.

TABLE 8.5

POSSIBLE CLINICAL AND PSYCHOSOCIAL SYMPTOMS RELATED TO WHIPLASH INJURY

Clinical symptoms	Psychosocial symptoms
Neck pain and stiffness	Depression
Headache	Anger
Shoulder pain and stiffness	Frustration
Vertigo	Anxiety
Dizziness	Family stress
Fatigue	Occupational stress
Temporomandibular joint symptoms	Hypochondriasis
	Compensation neurosis
Arm pain	Drug dependency
Paresthesias	Posttraumatic stress syndrome
Weakness	Sleep disturbances
Visual disturbances	Litigation
Tinnitus	Social isolation
Dysphasia	
Back pain	

Cervical Spondylosis

The etiology of chronic cervical conditions, such as cervical spondylosis, stands in marked contrast with the potentially catastrophic traumatic injuries just described. Although their onset is less dramatic, chronic injuries nonetheless can cause considerable dysfunction. Cervical spondylosis is a term used to describe degenerative changes in the cervical intervertebral discs and surrounding structures, including osteophytosis of the vertebral bodies, ligamentous instability, and osseous hypertrophy of the laminal arches and facets (Lestini and Wiesel 1989). Cervical spondylosis most often affects the C3-C7 vertebrae (McCormack and Weinstein 1996).

As part of the normal aging process, discs lose vertical height and become less extensible, attributable in large part to reduced water content and degradation of the disc substance. Disc degeneration is accompanied by osteophyte (bony outgrowth) formation and increased stresses on articular cartilage. These structural alterations increase the risk of spinal stenosis (narrowed canal), impingement on neural tissue, and impaired blood perfusion of the spinal cord. Symptoms associated with cervical spondylosis include *paresthesia*, neck and arm pain, weakness, and sensory loss. Recent advances in imaging technologies (e.g., magnetic resonance imaging) have improved diagnostic accuracy. Considerable debate continues, however, on the advisability of surgical intervention in treating the lesions associated with cervical spondylosis.

Disturbance or disease of the spinal cord (myelopathy) associated with cervical spondylosis is a well-recognized clinical entity. Mechanical factors that may cause cervical spondylotic myelopathy include a narrowing of the spinal canal, kyphotic conditions causing cervical flexion, spinal cord compression and related ischemia, and ligamentous ossification. Despite

REAR-END COLLISIONS

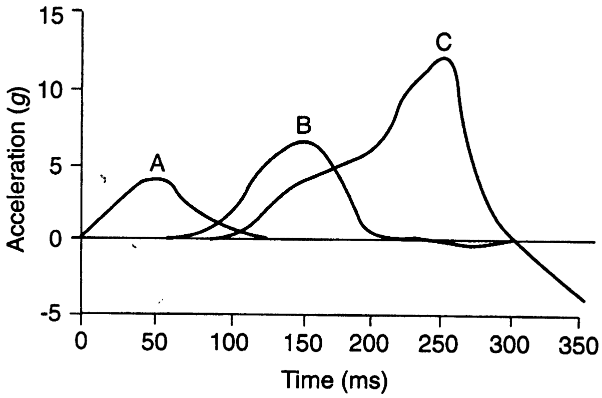
Over the past half-century, hundreds of research studies have sought to detail the mechanisms involved in rear-end collisions. These studies have involved use of live subjects (in low-speed rear-end impacts), cadaveric simulations, accelerometry, electromyography, and mathematical modeling. As a result of these studies, we have a better understanding of rear-impact dynamics, but controversy remains. The experts do agree on one point—cervical dynamics during rear-impact scenarios are complex and not entirely understood (e.g., Luan et al. 2000).

Pioneering work by Severy (1955) showed that rear-end collisions cause a sequential acceleration of the vehicle, the occupant's trunk and shoulders, and the occupant's head. As the vehicle is impacted (e.g., in an automobile rear-end collision), it accelerates first, reaching a peak acceleration of almost 5 g, that is, five times the acceleration of gravity (A in figure). The vehicle occupant's shoulders reach their peak acceleration of about 7 g 100 ms later (B in figure). Finally, the occupant's head reaches its peak acceleration of greater than 12 g at 250 ms after initial impact (C in figure). This sequential progression of peak acceleration is evidence of both momentum and energy transfers.

Response of the cervical spine depends on impact awareness, muscle involvement, and direction of impact (Kumar et al. 2005). In an unaware vehicle occupant, muscles are recruited late during the whiplash episode. Muscle recruitment and tension development may not happen until 200 to 250 ms after impact. Given that much of the critical cervical motion occurs during the first 200 ms, muscle involvement may only play a role in the late stages of whiplash. Injury may have already happened before the muscles become involved (Bogduk and Yoganandan 2001).

On a positive note, epidemiological evidence suggests that many victims of rear-end collision do not sustain injuries, and most of those who are injured show no long-lasting effects. In one study, 18% of patients had injury-related symptoms 2 years postinjury—82% were asymptomatic (Radanov et al. 1995).

In addition to impact awareness, muscle involvement, and direction of impact, many other factors determine injury risk in rear-end impacts: vehicle mass, velocity, and ability to withstand crashes; road conditions; use of restraint systems; and the passenger's or driver's body and head position at impact, neck rotation, gender, history of neck injury, and age.



Idealized acceleration curves of (A) an impacted vehicle, (B) an occupant's shoulders, and (C) an occupant's head.

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