

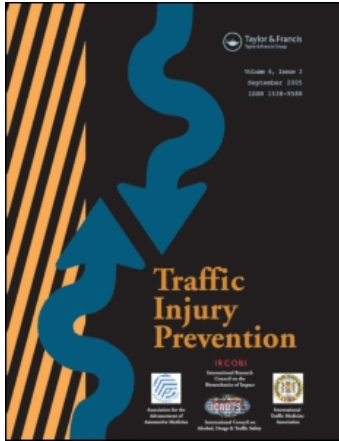
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The Anatomy and Biomechanics of Acute and Chronic Whiplash Injury

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The Anatomy and Biomechanics of Acute and Chronic Whiplash Injury

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Whiplash injury is the most common motor vehicle injury, yet it is also one of the most poorly understood. Here we examine the evidence supporting an organic basis for acute and chronic whiplash injuries and review the anatomical sites within the neck that are potentially injured during these collisions. For each proposed anatomical site—facet joints, spinal ligaments, intervertebral discs, vertebral arteries, dorsal root ganglia, and neck muscles—we present the clinical evidence supporting that injury site, its relevant anatomy, the mechanism of and tolerance to injury, and the future research needed to determine whether that site is responsible for some whiplash injuries. This article serves as a snapshot of the current state of whiplash biomechanics research and provides a roadmap for future research to better understand and ultimately prevent whiplash injuries.

Keywords Whiplash injury; Biomechanics; Neck; Injury mechanisms; Tolerance; Acute and chronic injury

INTRODUCTION

Neck sprains and strains—commonly known as whiplash injuries—are the most common motor vehicle injuries treated in U.S. hospital emergency departments (Quinlan et al. 2004). Incidence rates for whiplash injury range from 28 to 834 per 100,000 each year (Cassidy et al. 2000; Ostremski et al. 1989), and data stratified on gender and age show that females aged 20 to 24 have the highest incidence (~965 per 100,000 annually; Quinlan et al. 2004). Chronicity rates for whiplash patients also vary widely. In one study, 66 percent of subjects had residual neck pain after two years (Norris and Watt 1983), whereas in another study only 6 percent of subjects had residual neck pain after one month (Schrader et al. 1996). Such wide-ranging incidence and chronicity rates may stem from differing sample sizes, sampling methods, and injury definitions, but despite these differences, acute and chronic whiplash injuries are by a wide margin the most frequent automobile-related injury (Viano 2003). It also remains one of the most poorly understood automotive injuries.

Clinically, whiplash patients present with neck, shoulder, or back pain; headaches; dizziness; paresthesias; vertigo; or cognitive/psychological symptoms (Evans 1992; Radanov et al. 1995; Sterner and Gerdle 2004). The source of the initial symptoms is often uncertain (Binder 2007), but it is generally presumed these initial symptoms have an organic basis. Multiple anatomical sites in the neck have been postulated for this initial injury, including the facet joints, spinal ligaments, intervertebral discs, vertebral arteries, dorsal root ganglia, and neck muscles (Figure 1, Table I). Some chronic pain also appears to be organic in nature (Lord et al. 1996a; Sterling 2006), although late whiplash syndrome is viewed by some not as a chronic injury but rather as a self-perpetuating cycle of maladaptive behaviors possibly initiated by an acute organic lesion (Ferrari and Schrader 2001).

Despite these disparate views regarding their origin, some symptoms of whiplash injury likely have organic bases that are related in some way to the forces transmitted through the neck and the strains experienced by tissues in the neck during a collision exposure. Indirect evidence supporting this premise is the 31 to 75 percent reduction in whiplash injuries reported for collisions in vehicles with new anti-whiplash seats designed to reduce these forces (Farmer et al. 2003; Jakobsson and Norin 2004; Viano and Olsen 2001). If there was no underlying injury caused by the collision exposure, then these new seats

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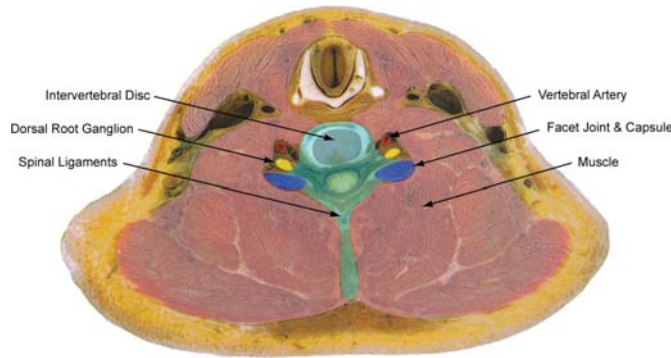


Figure 1 Cross section of the neck showing the anatomical arrangement of the proposed sites of whiplash injury. The shaded areas show muscle (pink), spinal ligament (aqua), facet joints (blue), dorsal root ganglia (yellow), vertebral arteries (red), and intervertebral disc (grey). (Adapted from Rohen and Yokochi 1993.)

would presumably have little or no effect on the rate of injury. Moreover, some whiplash injuries likely do not resolve for organic reasons rather than psychosocial ones. This latter proposition is supported by the delayed recovery and higher chronicity rates for patients with more severe initial symptoms (Scholten-Peeters et al. 2003; Suissa et al. 2001; Williams et al. 2007). What remains unclear, however, is whether chronic pain originates from the acutely injured tissue or whether other physiologic processes account for the persistence of pain.

Here we review the evidence supporting an organic basis for acute and chronic whiplash injuries. For each proposed anatomical site of whiplash injury—facet joints, spinal ligaments, intervertebral discs, vertebral arteries, dorsal root ganglia, and neck muscles—we present the clinical evidence supporting that injury, the relevant anatomy, the mechanism of and tolerance to injury, and the future research needed to definitively determine whether that site is responsible for some whiplash injuries.

FACET JOINT AND CAPSULAR LIGAMENT

Clinical Evidence of Injury

The cervical facet joints are the most common source of neck pain (Aprill and Bogduk 1992; Barnsley et al. 1994). Medial branch blocks and provocative testing have also implicated the facet joint in neck pain, particularly in chronic whiplash patients (Barnsley et al. 1993; Bogduk and Marsland 1988). This strong clinical evidence of facet-mediated neck pain has led to the

development of diagnostic tests (e.g., facet blocks) and treatment procedures (e.g., radiofrequency neurotomies) that can reduce or eliminate pain for a period of time (Lord et al. 1996b).

Relevant Anatomy

There are two facet joints between each pair of cervical vertebra from C2 to C7. The facet joint is a synovial joint enclosed by a thin, loose ligament known as the facet capsule. A synovial fold on the inner capsule extends between the margins of the articulating bony surfaces. The facet capsule itself lacks the stiffness to alter the intervertebral kinematics and instead follows the motions of its surrounding bony vertebrae (Winkelstein et al. 2000).

Cervical facet joints are innervated by the medial branches of the dorsal primary ramus from the two levels surrounding each joint (Lang 1993). Several histologic and anatomic studies have identified mechanoreceptors and unmyelinated nociceptors in the cervical facet joint (Giles and Harvey 1987; Inami et al. 2001; Kallakuri et al. 2004; McLain 1994; Ohtori et al. 2003). Though the size of the receptive fields of these pain fibers remains unknown, it has been proposed that each fiber innervates an area large enough to collectively cover the entire joint (Cavanaugh 2000). The facet capsule also contains Aδ- and C-fibers, both of which transmit nociceptive signals; i.e., pain (Cavanaugh 2000; Cavanaugh et al. 1989; Giles and Harvey 1987; Inami et al. 2001; Kallakuri et al. 2004; McLain 1994; Ohtori et al. 2003). Nociceptors reactive for substance P and calcitonin gene-related peptide have also been identified in the cervical facet capsules (Inami et al. 2001; Kallakuri et al. 2004; McLain 1994; Ohtori et al. 2003). Both of these neuropeptides are neurotransmitters and nociceptive neuromodulators (Ma and Eisenach 2003; Munglani et al. 1996). Thus, the cervical facet joints have the necessary anatomical features to initiate and potentially modulate more widespread neck pain.

Injury Mechanism and Tolerance

The motion of the facet joint articular processes and the capsule during whiplash-like impacts have been characterized in both human volunteers and cadaveric specimens (Cusick et al. 2001; Kaneoka et al. 1999; Pearson et al. 2004). Based on documented joint motion, two mechanisms of facet joint injury have been proposed: pinching of the synovial fold and excessive strain of the capsule. Ono et al. (1997) and Kaneoka et al. (1999) observed that the cervical vertebrae rotate about a higher instantaneous

Table I Summary of the potential location, type, and duration of the injuries sustained at each proposed anatomical site of whiplash injury

Anatomical site	Specific site or level	Type of injury	Possible duration of injury or related pain		
			<1 Month	1–6 Months	>6 Months
Facet joint	C2/3 to C7/T1	Synovial fold pinching; excess capsule strain	Yes	Yes	Yes
Ligaments	Occiput to T1	Excess strain	Yes	Yes	Yes
Vertebral artery	Occiput to C6	Excess strain/pinching	Yes	Yes	Yes
Nerve root	C3 to T1	Cell membrane dysfunction	Yes	Yes	Yes
Muscles	Multiple muscles, each with associated tendons and fascia	Excess strain while active	Yes	No, but may mediate other pain sources	No, but may mediate other pain sources

center during a whiplash exposure than during normal voluntary motion and proposed that this abnormal motion compresses the posterior facet surfaces together, pinching the synovial fold. Although the synovial folds are innervated by nociceptors (Inami et al. 2001), no further work attempting to isolate this potential mechanism of whiplash pain has been performed.

Excessive facet capsule strain during whiplash has been demonstrated by numerous groups (Luan et al. 2000; Pearson et al. 2004; Yang and King 2003; Yoganandan et al. 2002). Peak strains of 29 to 40 percent have been measured in the C6/C7 capsule of cadaveric specimens exposed to whiplash dynamics, whereas peak strains experienced during normal bending are only 6 ± 5 percent (Panjabi et al. 1998a; Pearson et al. 2004). Head-turned postures can double peak capsule strain during simulated whiplash loading (Siegmund et al. 2008b). Prior to the occurrence of tissue failure, partial ruptures of the facet capsule have been observed in both tension and shear loading of this joint (Siegmund et al. 2001; Winkelstein et al. 2000). Further, the maximum capsule strains at partial rupture (35–65 percent) do not exceed those strains observed in some capsules during the simulated whiplash loading (Siegmund et al. 2001; Winkelstein et al. 2000). These data suggest that capsule elongation during whiplash is a potential mechanism of injury in some individuals.

More recently, *in vivo* animal models have related facet joint biomechanics to afferent activity and pain symptoms. In a goat model, afferents in the facet capsule are activated by tensile loading of the C5/C6 facet joint (Lu et al. 2005a, 2005b). Capsule strains of 10 ± 3 percent activated nociceptive afferents, whereas strains of 44 to 47 percent were sufficient to saturate the mechanoreceptors and nociceptors. Similar strains in the C6/C7 capsule of the rat during joint distraction also produce persistent pain symptoms (Dong et al. 2008; Lee et al. 2004; Lee et al. 2004; Quinn et al. 2007). More importantly, however, the intensity and duration of persistent pain in the rat depend upon the magnitude of strain in the capsule. A maximum principal strain of about 21 percent is associated with persistent sensitivity (Dong et al. 2008; Lee et al. 2004; Lee et al. 2004b). These strains are consistent with those detected in the human capsule during whiplash simulations (Pearson et al. 2004; Siegmund et al. 2001; Sundararajan et al. 2004; Winkelstein et al. 2000). For the same levels of joint distraction that produce pain, the fiber organization in the capsular ligament is also altered (Quinn et al. 2007), indicating that collagen in the capsule is being disorganized by the joint distraction, despite the absence of complete ligament failure (Figure 2).

Physiologic responses can contribute to pain in the absence of major mechanical failure. For instance, Lu et al. (2005a, 2005b) reported persistent after-discharges from afferents after joint loads were removed. At the cellular level, both neurons and other cells in the dorsal root ganglia demonstrate sensitive responses to painful and non-painful joint loading (Lee et al. 2008). Persistent increased expression of binding protein (BiP), a marker of cellular stress response (Dong et al. 2008), occurs predominantly in neurons of the dorsal root ganglia following painful facet joint loading similar to that which develops in

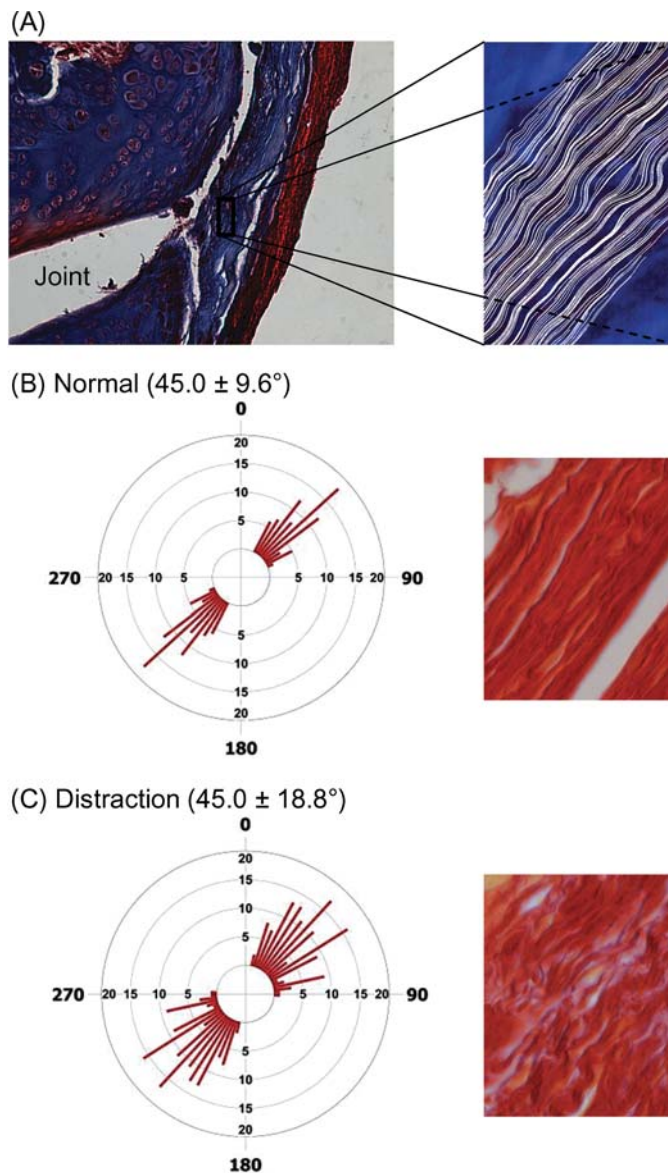


Figure 2 Facet capsule histology demonstrating collagen fiber organization in the rat facet capsular ligament. (A) Masson trichrome staining demonstrating a facet joint with its enclosing capsule (blue stain) and surrounding muscle (red). The inset shows a closeup of the facet capsule ligament with fibers—outlined in white—showing their typical undulation. (B), (C) Following a joint distraction sufficient to produce persistent pain symptoms, the collagen fiber organization (measured by angular deviation) is significantly larger. Shown here are representative histograms of angular deviation and sample tissue from (B) normal and (C) distracted ligaments.

whiplash. Inflammatory responses in the spinal cord are induced and sustained following painful joint loading and depend on the strain imposed on the capsule (Lee et al. 2004, 2008). These local and more widespread neuro-inflammatory cascades contribute to a variety of other chronic pain syndromes (DeLeo and Yezierski 2001). Their induction, persistence, and relationship to joint/capsule mechanics in painful whiplash loading supports the facet joint's involvement in whiplash pain.

Future Directions

Continued biomechanical research is needed to define how collagen injury during subfailure ligament loading initiates pain responses, their temporal response, and how such scenarios may be produced during whiplash. Moreover, continued research is needed to identify and define the specific physiologic pathways (electrophysiologic, immunologic, and otherwise) that are responsible for chronic pain following this joint's injury. Using this information, better diagnosis and treatment for facet-mediated, or at least facet-initiated, whiplash pain can be developed.

LIGAMENT AND DISC

Clinical Evidence of Injury

Magnetic resonance and autopsy studies of whiplash patients have documented injuries to the neck ligaments and intervertebral discs in addition to the facet joints (Jonsson et al. 1991; Kaale et al. 2005a, 2005b; Krakenes and Kaale 2006; Pettersson et al. 1997). Whiplash-related symptoms may be due, in part, to injuries of cervical ligaments and discs and their embedded mechanoreceptive and nociceptive nerve endings. Ligament injuries may cause acute neck pain and lead to chronic spinal instability, and injured mechanoreceptors may corrupt normal sensory signals and could lead to abnormal muscle response patterns and decreased neck mobility and proprioception (Panjabi 2006).

Relevant Anatomy

The cervical vertebrae are joined by multiple ligaments. The main ligaments below the axis include the anterior and posterior longitudinal, capsular, interspinous, and supraspinous ligaments and the ligamentum flavum (Figure 3). The anterior and posterior longitudinal ligaments are thin sheets of tissue that span the anterior and posterior surfaces of the vertebral bodies, respectively, and blend with the underlying annular fibers. The capsular ligaments, as described earlier, encase the facet joints. The interspinous ligaments join adjacent spinous processes and are not present in all adults. When present, these ligaments are thin, weak tissues of high collagen content that blend posteriorly with the supraspinous ligament. The ligamentum flavum is the most elastic tissue in the human body—comprised of up to 80 percent elastin—and joins adjacent laminae bilaterally (Yahia et al. 1990). The intervertebral disc, located between adjacent vertebral bodies, consists of a central nucleus pulposus encased by annulus fibrosis fibers.

Ligaments of the upper cervical spine—occiput through the axis—have unique functional and structural anatomy. Alar and transverse ligaments play key roles in providing stability in this region due to the absence of intervertebral discs and the horizontal alignment of the facet joints (Dvorak et al. 1988). These ligaments have a high collagen and low elastin content, predisposing them to partial or complete rupture at low strains during high-speed elongation (Panjabi et al. 1998b).

Ligaments provide joint position sense during normal motion and combined with discs provide passive stability and ab-

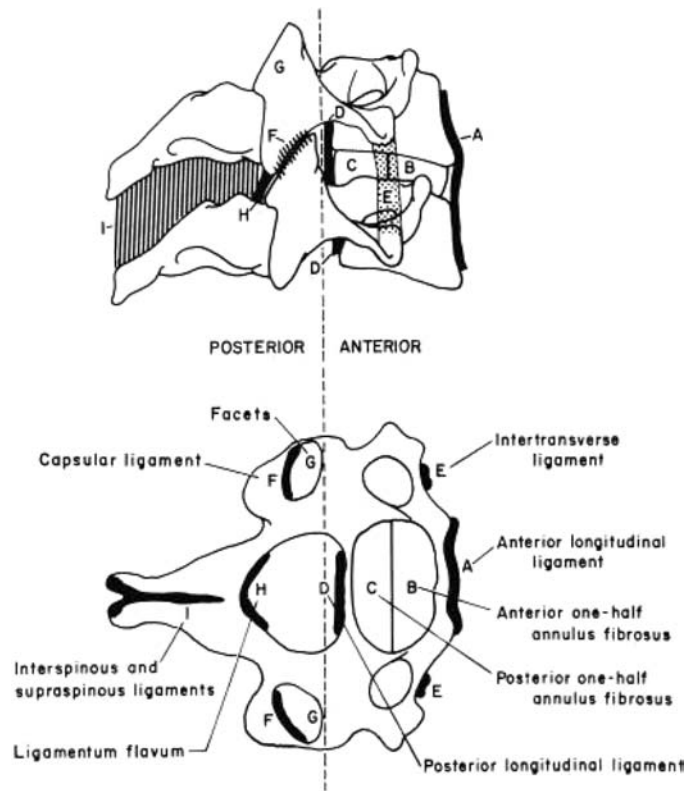


Figure 3 Ligaments of the middle and lower cervical spine (from White and Panjabi 1990).

sorb energy during high-speed trauma. The specific function of each cervical ligament and disc in resisting whiplash loading is dependent upon its specific anatomical location, orientation, geometry, and unique mechanical properties.

Injury Mechanism and Tolerance

Spinal ligaments and annular fibers encapsulating the discs can partially or completely rupture when stretched beyond their physiological limit. The whiplash-related response of the cervical ligaments and discs have been quantified for frontal, side, and rear impacts using a whole cadaveric cervical spine model with muscle force replication and a surrogate head (Figure 4A; Ivancic et al. 2005). During rear impacts with the head facing forward, dynamic strains in the anterior longitudinal ligament and annular fibers above physiological levels (Ivancic et al. 2004; Panjabi et al. 2004a) and increased joint laxity (Ito et al. 2004) were observed. The C5/C6 disc was found to be at highest risk of injury during both frontal and rear impacts (Ito et al. 2005). In addition to the C5/C6 disc, excessive strains were observed in superior discs, including C2/C3, during frontal impacts. The disc injuries occurred at lower impact accelerations during rear impacts compared to frontal impacts. During frontal impacts, the supraspinous ligament, interspinous ligament, and ligamentum flavum at C2/C3 through C7/T1 are at risk for injury due to excessive strain (Panjabi et al. 2004b). The T1 horizontal acceleration at which ligament and/or disc injuries were detected in those studies using pre- and post-impact flexibility tests was 5 g

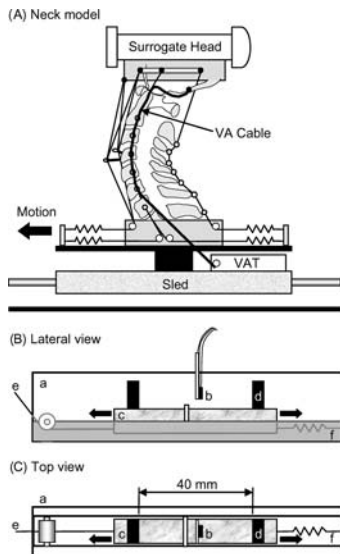


Figure 4 (A) Schematic of the biofidelic whole cervical spine model with surrogate head and muscle force replication used to simulate whiplash, showing the position of vertebral artery transducer (VAT) and vertebral artery (VA) cable passing through the foramen and attaching to the occiput. (B) Lateral and (C) top schematic views of the vertebral artery transducer: frame (a), Hall effect sensor (b), movable carriage (c) carrying two rare earth magnets (d), vertebral artery cable (e), and tension spring (f). (Adapted from Ivancic et al., 2006.)

for rear impacts, 6.5 g for side impacts, and 8 g for frontal impacts. For all impact configurations, the spinal levels at greatest risk of ligament and/or disc injury were C3/C4 through C7/T1.

Injuries to the alar and transverse ligaments of the upper cervical spine are reportedly more severe in individuals who have their head rotated at impact (Kaale et al. 2005a). These injuries have not been reproduced in cadaveric studies at impact accelerations (applied at T1) up to 8 g (Hartwig et al. 2004; Maak et al. 2006). This suggests that the upper cervical spine symptomatology reported by some whiplash patients may be due to impacts causing T1 accelerations in excess of 8 g or from some other anatomical structure.

Future Directions

Additional research is needed to further our understanding of ligament and disc injury mechanisms during whiplash and to investigate preventative mechanisms. Biomechanical studies are needed to correlate increased ligament and disc laxity with specific ligament injuries for each impact configuration. Future work is also needed to correlate the severity of ligament and disc injuries, in the form of biomechanical instability, with the onset of neck pain and, ultimately, to link specific ligament injuries to neck pain, or pain patterns, in whiplash patients. Biomechanical studies of simulated whiplash are needed to determine whether dynamic neck loads and high-speed ligament and disc strains are reduced by implementing specific injury prevention systems; e.g., active head restraint or energy-absorbing seat. These results may be correlated with those of epidemiological studies that investigate the effectiveness of injury prevention systems in reducing neck injury in real-life automobile collisions.

VERTEBRAL ARTERY

Clinical Evidence of Injury

Altered blood flow rates due to spasm and/or narrowing of vertebral arteries in whiplash patients have been associated with chronic symptoms of headache, blurred vision, tinnitus, dizziness, and vertigo (Reddy et al. 2002; Seric et al. 2000). Intimal tears of the vertebral artery are most common at the primary site of cervical axial rotation, the atlanto-axial joint (Barton and Margolis 1975; Chung and Han 2002; Davis and Zimmerman 1983; Pollanen et al. 1996; Sherman et al. 1981; Stahmer et al. 1997; Taneichi et al. 2005). Vertebral artery injury causing inadequate perfusion of the brainstem and surrounding tissues could explain some of the whiplash-related symptoms (e.g., headache, dizziness, and vertigo).

Relevant Anatomy

The vertebral arteries supply blood to the head, brain, and neck tissues. The vertebral arteries enter the spine at the C6 transverse processes bilaterally and run superiorly in the transverse foramen of each cervical vertebra. After exiting C1, the vertebral arteries travel along the C1 posterior arch and enter the foramen magnum of the skull. The vertebral artery is a viscoelastic structure: the adventitia is composed primarily of collagen fibers and the media consists of collagen as well as more substantial portions of smooth muscle and elastic fibers. It is encased in a fibrous tunnel and affixed to adjacent structures via a transected collagen network (Chopard et al. 1992).

Injury Mechanism and Tolerance

Coupled extension and axial rotation of the upper cervical spine has been hypothesized to cause vertebral artery injury (Barton and Margolis 1975; Chung and Han 2002; Davis and Zimmerman 1983; Sherman et al. 1981). Vertebral artery elongation causes a decrease in the vessel diameter due to Poisson's effect and could cause transient vascular compromise (Dobrin 1978). Alternatively, stretching or pinching of the vessel along a turn in its circuitous course is also possible (Barton and Margolis 1975). These mechanisms can also precipitate tearing of the intimal layer of the vertebral artery (Chung and Han 2002).

Cadaveric neck models have demonstrated coupled extension and axial rotation during side and rear impacts with the head turned but not during frontal or rear impacts with the head facing forward (Carlson et al. 2007; Ivancic et al. 2006). In those studies, average vertebral artery elongation was measured between the occiput and C6 vertebra using a custom transducer mounted in a cadaveric neck (Figure 4B). Peak vertebral artery elongation of 30.5 mm during head-turned rear impacts and 17.4 mm during side impacts significantly exceeded physiological elongation limits. Moreover, peak elongation occurred early—about 85 ms following the onset of T1 acceleration—with elongation rates reaching 1340 mm/s during head-turned rear impacts and 610 mm/s during side impacts. The magnitude, rate, and timing of vertebral artery elongation are thus sufficient to potentially cause vertebral artery injury.

Future Directions

Further biomechanical research is needed to determine the strain distribution throughout the vertebral artery during physiological movements and whiplash-related loading rates from different initial neck postures and in various impact directions.

DORSAL ROOT GANGLION AND DORSAL ROOT

Clinical Evidence of Injury

The dorsal root ganglion contains the cell bodies of most peripheral sensory nerves at each spinal level. Direct injury to cell bodies within the dorsal root ganglion could thus explain many of the typical whiplash symptoms (e.g., neck pain, cervicogenic headache, vertigo, vision disturbance, and neurological symptoms in the upper extremities). Generalized hypersensitivity to pressure acutely and chronically and decreased thermal pain thresholds in the skin over the cervical spine can be explained by impaired local sensory processing (Greening et al. 2005; Kasch et al. 2001b; Scott et al. 2005; Sterling et al. 2003, 2006; Sterner et al. 2001). In addition, increased electrical activity in the spinal cord and widespread reductions in electrical and pressure thresholds after whiplash suggest altered central pain processing (Banic et al. 2004; Curatolo et al. 2001; Kasch et al. 2001a; Scott et al. 2005). Increased sensitivity to pain (hyperalgesia) and larger areas of referred pain are also reported for whiplash patients (Koelbaek Johansen et al. 1999). These studies documenting both local and referred pain after whiplash injury provide clinical evidence for altered sensory transmission and pain pathways in the central nervous system.

Relevant Anatomy

The anterior and posterior rootlets coming off the spinal cord combine to form dorsal and ventral nerve roots, which make up the spinal nerves at each spinal level. The location, direction, and number of nerve rootlets vary at each cervical level. The dorsal and ventral roots come together in the region of the neural foramen and continue more distally into the periphery as the spinal nerve to innervate structures outside the spinal column. Posterior rootlets making up the dorsal root are the sensory (afferent) fibers, whereas the anterior rootlets making up the ventral root are the effector (efferent) fibers. Cell bodies of peripheral afferents are housed in the dorsal root ganglion, which has been shown to be particularly sensitive to loading—even slight compression of normal dorsal root ganglia can produce sustained electrical activity and pain (Howe et al. 1977). Unlike peripheral nerves, the nerve roots themselves are not enclosed by a thick epineurial sheath, and thus they lack the mechanical strength of their peripheral counterparts, potentially exposing nerve roots to increased risk of injury when loaded.

Injury Mechanism and Tolerance

Movements of the cervical spine in flexion, extension, and lateral bending cause the volume of the spinal canal to change. During normal voluntary neck motions, blood volumes in the internal and external vertebral venous plexa can easily move to compen-

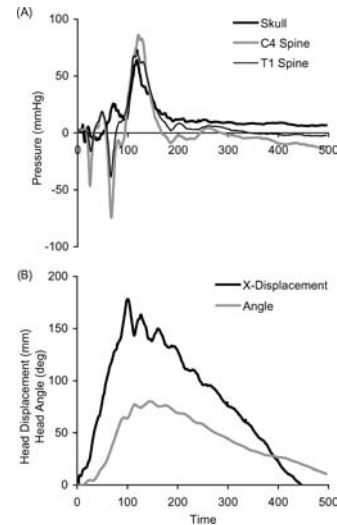


Figure 5 Pressure and displacement during a whiplash extension experiment using a pull-force on the porcine head-plate of 600 N. Graphs show (A) the pressure in the CNS at the skull, C4 and T1 vertebral levels, and (B) the angular and linear X-displacement of the head center of mass. (Adapted from Svensson et al., 2000.)

sate for these volume changes. During rapid whiplash-induced motions, however, resistance to blood flow and the inertia of the fluid mass itself can generate transient pressure gradients between the inside and outside of the spinal canal (Aldman 1986). These pressure gradients can directly load the spinal ganglia and nerve roots, potentially leading to whiplash-related symptoms.

Whiplash experiments carried out on anesthetized pigs in extension, flexion, and lateral bending revealed a transient pressure drop inside the spinal canal during rapid motion in all directions (Figure 5; Svensson et al. 2000). Follow-up histology showed leakage of the plasma membrane of spinal ganglia nerve cells consistent with cellular injury (Örtengren et al. 1996). Eichberger et al. (2000) reported similar pressure recordings in cadavers exposed to whiplash and Schmitt et al. (2003) have since recreated the pressure pattern in a computational fluid dynamics model of the human cervical spine. These experimental findings are supported by an autopsy study of individuals who had sustained severe inertial neck loading (Taylor et al. 1998). Interstitial hemorrhage in the cervical dorsal root ganglia was observed in those autopsies despite an absence of injury to other structures surrounding the ganglia.

The relationship between the head-neck motion and the pressure magnitude in the spinal canal is quantified by the neck injury criterion (NIC; Boström et al. 2000). NIC is related to the relative horizontal acceleration and velocity of the head with respect to the torso, and a low NIC equates to a low risk of long-term neck injury (Krafft et al. 2003). Because many other loads and strains within the neck tissues also vary with NIC, this relationship between NIC and long-term neck injuries is not proof that dorsal root ganglion injuries explain all long-term whiplash injuries.

Deformation of the nerve roots themselves is another potential mechanism for producing persistent neck pain. The neural

foramina change shape and decrease their diameter during extreme neck motions (Carter et al. 2000; Krivickas and Wilbourn 2000; Yoo et al. 1992). This can compress the nerve root within the intervertebral foramen during whiplash motions. Nuckley et al. (2004) reported a 20 percent decrease in area for the C4-C7 intervertebral foramina of cadaveric cervical spines in extension. The intervertebral foramen at C5/C6 narrowed by as much as 1.8 mm during simulated rear impacts of a cadaveric head-neck model using horizontal T1 accelerations up to 8 g (Panjabi et al. 2006; Tominaga et al. 2006). This dynamic narrowing of the foramen during whiplash may compress the nerve roots and ganglia in the lower cervical spine, particularly in individuals with congenitally narrow foramen or those with osteophytes.

Transient loads on the cervical dorsal nerve roots have produced significantly elevated pain symptoms in a rat model (Hubbard and Winkelstein 2005; Hubbard et al. 2008; Rothman et al. 2005). Wallerian degeneration, disrupted axonal transport, and altered neuronal responses in the dorsal root ganglion are also produced (Hubbard and Winkelstein 2008). These data further support direct and indirect relationships between tissue loading, neuronal function, and altered physiology locally, in the dorsal root ganglia and throughout the nervous system for painful loading conditions.

Future Directions

Refined finite element and fluid dynamics models of the human head and neck may lead to better understanding of the flow and pressure phenomena that appear to result in ganglion dysfunction. This improved understanding would enable the development of more accurate injury criteria and tolerance limits for ganglion injury and would guide the development of improved crash dummies and performance requirements for injury protection systems in vehicles. Additional work is also needed to establish the link between the observed pressure transients and the generation and time course of ganglion dysfunction. The influence of nerve cell membrane dysfunction on nerve function and pain sensitization also needs to be investigated following experimentally induced ganglion injury.

MUSCLE

Clinical Evidence of Injury

Muscle or myofascial pain is a common symptom reported by whiplash patients (Evans 1992), although evidence of direct injury to muscle remains inconclusive. Injury-related muscle soreness is associated with a rise in serum creatine kinase detected at 3 to 24 h after high-intensity exercise and may persist for up to 9 days (Evans et al. 1986). In some whiplash patients, elevated serum creatine kinase has been observed 24 h after injury but not 48 h after injury, despite neck pain extending beyond 3 months (Scott and Sanderson 2002). Although this work suggests that direct muscle injury may not be responsible for chronic whiplash pain, muscles may nevertheless play an indirect role in modulating pain caused by injuries to other structures.

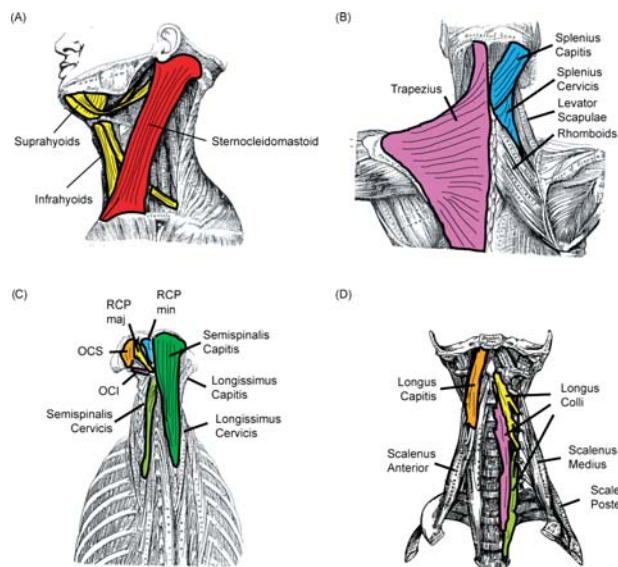


Figure 6 Neck muscle anatomy. (A) Lateral view of superficial muscles. (B) Posterior view of superficial muscles. (C) Posterior view of deeper muscles. (D) Anterior view of deep muscles. (Adapted from Gray, 1977.) (Figure appears in color online).

Relevant Anatomy

Muscles comprise the majority of the neck's volume (Figure 1). The superficial muscles, such as sternocleidomastoid or trapezius (Figures 6A and 6B), are often implicated in the pain and tenderness associated with whiplash injury. These superficial muscles attach to the skull, shoulder girdle, and ligamentum nuchae but do not generally attach directly to the cervical vertebrae. Deeper muscles, such as splenius, semispinalis, longissimus, scalenes, and longus, attach on multiple cervical vertebrae (Figures 6B, 6C, 6D). The deepest neck muscles, the multifidus muscles, insert directly on the facet capsule of cervical vertebrae (Figure 7) and may be relevant to injury of the capsular ligaments (Anderson et al. 2005; Winkelstein et al. 2001). Most neck muscles have complex architecture, with extensive internal tendon (Kamibayashi and Richmond 1998) and a high density of muscle spindles (Boyd-Clark et al. 2002). Although this has not been explored, the presence and arrangement of the internal tendon may be related to musculotendinous pain.

Injury Mechanism and Tolerance

The direct mechanism of neck muscle injury occurs from eccentric contractions; i.e., imposed lengthening during active contraction. Computer simulations using experimental kinematics of human subjects exposed to rear-end collisions have shown that both anterior and posterior neck muscles experience active lengthening during rear impacts (Brault et al. 2000; Vasavada et al. 2007). The anteriorly located sternocleidomastoid is active and lengthened during the retraction phase of whiplash, whereas posterior muscles are active and lengthened during the rebound phase. For simulated impacts with a speed change of 8 km/h, peak muscle fascicle strains averaged about 7 percent (max. 15%) in the sternocleidomastoid and 21 percent (max. 50%) in the posterior muscles such as semispinalis capitis. These

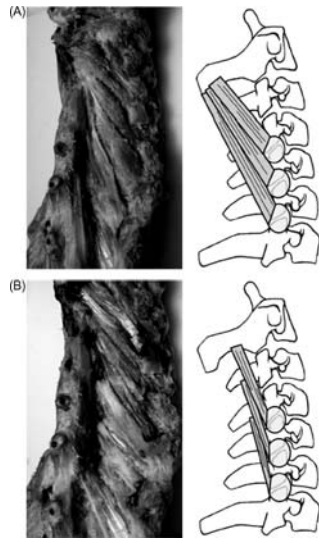


Figure 7 Anatomy of (A) superficial and (B) deep layers of the cervical multifidus muscles, depicting attachments on the facet capsules. (Adapted from Anderson et al., 2005.)

strains exceeded those shown to cause muscle injury (5–20%) in laboratory studies (Macpherson et al. 1996; McCully and Faulkner 1985). Thus, acute neck muscle injury may occur during rear-end impacts.

Interactions with Other Anatomical Sites

Neck muscles potentially interact with other anatomical sites of whiplash injury in at least three ways: (1) neck muscles attach directly to the facet capsule, which has been implicated in chronic pain following whiplash; (2) neck muscle activation indirectly affects the loads and strains in other anatomical structures; and (3) altered neuromuscular control may contribute to chronic pain via elevated and inappropriate muscle activation.

The pathomechanical evidence for facet capsular ligament involvement in whiplash injury and chronic neck pain has been outlined earlier in this article. Direct attachment of the multifidus muscles to the capsular ligament (Anderson et al. 2005; Winkelstein et al. 2001), combined with early activation of these muscles in some subjects during a rear-end collision may exacerbate peak strain in the capsular ligaments (Siegmund et al. 2008a).

Neck muscle activation also affects spinal tissue loads by increasing intervertebral compression and altering intervertebral kinematics. Because neck muscles are oriented primarily vertically, their activation produces axial compression of the cervical spine, increasing loads on the intervertebral disc and facet joints. Reflex muscle activation also affects the kinematic response of the head and neck. In subjects exposed to a series of identical perturbations, habituation of the muscle response amplitude by about 50 percent was accompanied by 10 to 30 percent changes in peak head kinematics (Siegmund et al. 2003). By altering head and neck kinematics, load and strain thresholds for injury may be exceeded in other structures such as ligaments, discs, and facet joints.

Finally, the interaction between muscles and the nervous system—i.e., via neuromuscular control—may be related to

chronic pain. Patients with chronic pain demonstrate altered neuromuscular patterns (Falla et al. 2004; Nederhand et al. 2002), but it is not known whether the observed muscle activities are a physiological deficit in motor control or a protective strategy to avoid pain. A further complication is that different types of adaptive responses have been observed in different populations of whiplash patients (Nederhand et al. 2000, 2003). An inability to relax after exercise and excessive coactivation are associated with cervical pain (Elert et al. 1992; Nederhand et al. 2000; Westgaard et al. 1993), and relaxing selected neck muscles with botulinum toxin improves range of motion and reduces pain in these patients (Freund and Schwartz 2002). This suggests that pain and increased muscle activity may cyclically reinforce one another (Johansson and Sojka 1991). Contrasting evidence supports a pain adaptation model in which nociceptive interneurons inhibit the activity of painful muscles or those in the vicinity of pain sources (Lund et al. 1991). Nederhand et al. (2003) found that whiplash patients had a normal ability to relax the trapezius following exercise, but during exercise those with the highest disability levels had the lowest muscle activity. It remains unclear, however, whether muscle dysfunction is a cause (leading to damage of other anatomical structures) or effect (due to disuse or pain avoidance) of pain or merely an associated correlation.

Future Directions

Future research is needed to explore the role of neck muscles in the mechanism of acute whiplash injury, especially the interactions with other neck structures. Specifically, the effect of multifidus activity on capsular ligament mechanics and nociceptive physiologic responses needs to be studied to determine the relevant magnitude of loads from muscle forces on the ligament. Ideally, this type of research should be conducted *in vivo*, where muscles can be stimulated and ligament mechanical parameters measured. Research is also needed to explore how altered neuromuscular control relates to chronic pain. Specifically, studies are needed to analyze deep muscle activity in patients with chronic neck pain due to whiplash injury. In addition, validated mathematical models may be used to assess the effect of abnormal muscle activation on the loads in other anatomical structures.

SUMMARY

This review provides a brief summary of the anatomical structures being investigated by many groups to potentially explain whiplash injury. Each of the tissues described is strained during a whiplash exposure and thus could be injured if the crash-induced strain exceeds that tissue's tolerance. For each of the tissues summarized here, continued research is needed to better understand the biomechanical and physiological link between crash-induced loading and acute and chronic whiplash-related pain. A better understanding of each potentially injured tissue will help improve the diagnosis and treatment of whiplash injuries. Elimination or reduction of tissue strains through improved vehicle, seat, and head restraint design will help reduce the frequency of whiplash injury.

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