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COMMON SYMPTOMS OF CERVICAL / ACCELERATION / DECELERATION (CAD) INJURY

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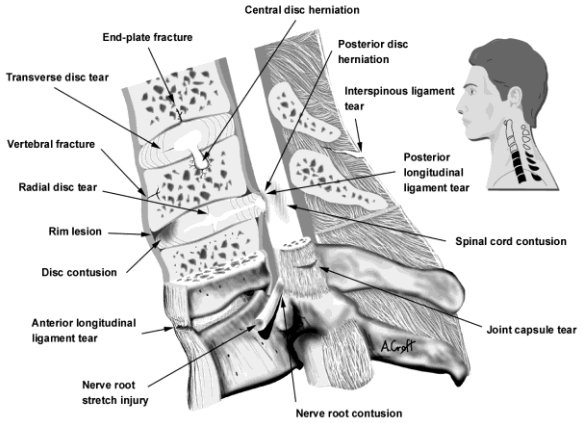


Common Symptoms Following Whiplash, In Order Of Prevalence

The symptoms most commonly described following CAD injury are described in their general order of prevalence. This is not however, an exhaustive list.

Neck pain

Headache

Interscapular pain

Back Pain

Paresthesiae

Extremity pain/weakness

Cognitive difficulties a

Dizziness/light-headedness

Facial pain and TMJ related symptoms (clicking; closed lock etc.)

Auditory symptoms (phonophobia; tinnitus; loss of hearing)

Vertigo

Ocular dysfunction (blurred vision; photophobia)

Dysphagia/hoarseness

a. May be part of postconcussion syndrome (PCS)

b. May be part of Barré-Liéou syndrome

c. May be part of TMJ dysfunction

Neck Pain

Tearing or damage to any soft tissue (including nervous tissue), fracture of bone, or disc herniation/prolapse/protrusion/disruption can cause neck pain. Immediate pain often indicates more severe injury, but many disabling injuries have delayed onset of symptoms. Very early onset of severe pain is sometimes an indication of disc or ligament injury. The balance of current evidence implicates paraspinal soft and hard tissue as the chief locus of pain generation. This includes discs, ligaments, joint capsules, end plates, neural tissue, and the vertebrae themselves. Moreover, although the muscles are often found to be in spasm, sore, and tender, and treatment rendered to the muscles does provide some symptomatic relief, they are not usually the source of pain. However, Mooney (1217) provides an argument in favor of muscles as an injured or directly involved tissue. He notes that cervical muscles contain the highest concentration of muscle spindles--up to 500 per gram of muscle--and these contain intrafusal fibers which enhance sensitivity. These have afferent and efferent connectivity to the sympathetic nervous system.

Another interesting study (1218) found that patients with late whiplash have a sense of pain, stiffness, and tension in their muscles as a result of an inability to relax between repetitive contractions. A similar finding has been reported in fibromyalgia patients and in those with work-related trapezius myalgia. In addition to somatic complaints of neck, back, and shoulder pain, a high proportion of the patient group had cognitive dysfunction. A variety of explanations for the muscular abnormalities was proffered: (1) the vicious cycle of pain, (2) sensory activity from ligaments and joint capsules increasing muscle spindle activity, and (3) the increased muscle metabolites stimulating

group 3 and 4 muscle afferents, which activate both static and dynamic gamma motoneurons projecting to homonymous as well as heteronymous muscles. And, not to leave out the psychological-stress-muscle tension theory. It was recently also shown, using sEMG, that whiplash subjects had more activity in superficial muscles and were less able to control pressure changes than controls (1334). They reported that sEMG appears to detect poorer motor control in the neck flexors in whiplash subjects, the increased co-activation of the superficial neck flexors being a likely compensation for reduced deep neck flexor function.

Another paper (1219) reported a higher tenderness and sensitivity to palpation and algometer in whiplash patients compared to controls. The authors conjectured a disturbance of pain modulation. They stated that a, "central dysmodulation of nociceptive impulses does not necessarily imply a central lesion caused by the crash. It might be induced by a long-lasting peripheral noxious input with sensitization of central synapses."

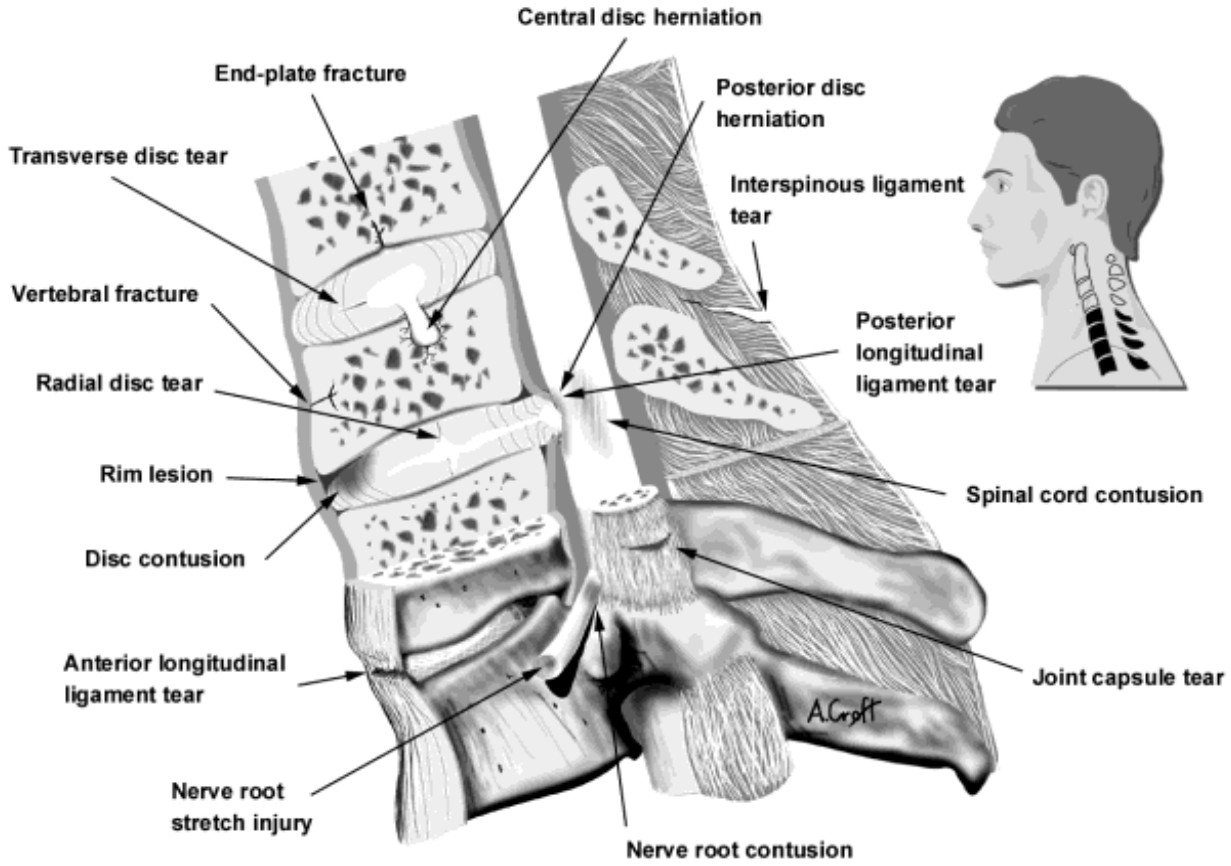
Barnsley et al. (457) have made a convincing case for the prominence of facet joint pain as the culprit in a large percentage of chronic whiplash pain patients by injecting local anesthetics into the joints. Painful joints were identified in 54% of their patients. Having identified the origin of the pain in those patients, it would have been interesting to compare a trial of manipulation on them to determine how much of that pain was biomechanical. More recently, using the same technique, these authors reported that between 46% and 73% of their CAD patients had pain localized to the cervical zygapophyseal joint or supporting ligaments.

In perhaps one of the most interesting recent discoveries, Yoganandan et al. (1564), using a cryomicrotome, sectioned six cadaver neck specimens into 20-40 micrometer slices. This allowed them to measure, quantitatively and in situ, the articular cartilage in terms not only of coverage, but also of depth in a way not possible using conventional radiography, MRI, or CT. This is first study of its kind to demonstrate these sex-based, facet joint anatomy variations. The authors also did not use any degenerative sections. They found that in the upper cervical spine, articular cartilage covered approximately 90% of the joint surface compared to only 68% in the lower cervical spine. Thickness of the cartilage was greatest in the center of the joint surface and also varied by segment, being thickest (and widest) at C1 and C2, perhaps compensating for the lack of an intervertebral disc there. Gaps were also documented between the end of cartilage coverage and the periphery of the articular surface. These were 5% and 16% on each side in the upper and lower cervical spines, respectively. However, in the upper cervical spine, the anterior gaps were generally smaller than the posterior gaps. This relationship was reversed in the lower cervical spine. And these were wider in females in the posterior portions of the joint than in males, suggesting a lower level of protection by articular cartilage. However, as the authors noted, the greater muscle mass of the male neck may be a factor in the development of cartilage. Still, the questions remains: do these greater gaps predispose females to greater facet joint injury risk during CAD trauma? Might this help explain the variance between male and female risk?

In 26 of 31 patients, a single segment was the causative factor, with C2-3 the most common cause of headache and upper cervical spine pain, and C5-6 the most common level associated with lower cervical spine pain and referred arm pain--a particularly interesting finding in light of the recent work by Ono et al. (1150), Grauer et al. (1174), and Panjabi et al. (1182), who measured peak hyperextensions in lower cervical levels, while upper levels rotated into flexion. Jónsson et al. (453) reported the highest proportion of ligamentous instability at this level in their series of CAD patients. This association has been consistently demonstrated (1532). I've made a composite illustration of the types of lesions reported to occur in CAD trauma. These are illustrated in Figure below.

This is an illustration of part of the cervical spine (black portion of inset). Part is cut in half for better viewing. This illustrates all of the lesions that have been reported to occur in whiplash trauma.

A Patient's Guide to Recovery. San Diego, (c) Spine Research Institute of San Diego, 1999.



One of the interesting recent findings was that the two most common segments to be affected in an Australian CAD fluoroscopic anesthetic block study were C2-3 and C5-6,

with a much greater occurrence on the right than on the left (1333). Note that this is an Australian study and they, like their British cousins, drive on the wrong side of the car. As seen during the s-shaped curve, the upper and lower segments are most affected. This is also consistent with the PMHS tests.

The most recent literature points also strongly toward the facet joint as a major contributor to neck pain. Kaneoka et al. (1273) referred to the pinching that can be seen to occur in this joint under dynamic crash testing as facet synovial fold impingement syndrome. Siegmund et al. (1495) have offered evidence in quasi-static PMHH tests for the possibility of facet joint capsular ligament injury in whiplash.

Experimental neurophysiological support is offered by Thunberg et al. (1442). They demonstrated that, in cats, the fusimotor-muscle spindle system is subject to a positive feedback error initiated by either (in the context of this study) chemosensory excitation of receptors in the facet joints or mechanoreceptor activity. We are drawn back to the facet joint itself as a major source of neck pain, with muscle stiffness as a likely concomitant factor.

Neck stiffness

Stiffness may be the result of protective muscle spasm secondary to most injuries of the neck. Chronic stiffness indicates myofascial fibrosis or contraction, ligamentous contraction (particularly of joint capsules), disc degeneration, or secondary osteoarthritic change.

Shoulder pain

This can be either from direct injury to the shoulder, such as from a shoulder harness, or impact with the car's interior, or from referred pain from soft tissues of the neck or a disc lesion. Other causes of shoulder pain, such as impingement syndrome, thoracic outlet syndrome (TOS) (1522), and rotator cuff inflammation, may develop secondary to CAD trauma, either as a result of muscular disuse or paresis of muscles or muscle groups. Imbalance between agonist and antagonist muscles can precipitate shoulder disorders or convert subclinical conditions to clinical conditions. In one study, 53% of the patients were found to have a periarticular shoulder disorder (131).

In a recent study of 476 CAD injury subjects the incidence of impingement-type pain was found to be 9% (1567). Notably, all of the shoulder complaints were delayed in onset. About 12.3% of cases with shoulder pain not diagnosed as impingement syndrome were thought to be the result of referred pain. Most had abnormal scapulohumeral rhythm motion. Scapulohumeral motion is known to be a composite of glenohumeral and scapulothoracic motion. The trapezius is believed to play a role in abnormalities in this motion in CAD patients.

In an uncontrolled, poorly described report, a group of surgeons reported their success using a combination of arthroscopic shoulder stabilization surgery and brachial plexus neuroloysis (1569). These surgical procedures were not described in detail and the

authors methods of patient selection, inclusion/exclusion criteria, statistical analysis, and outcome assessment were not provided, other than the assessment of two point discrimination.

Observing the replay of high speed video coverage of human subject crash tests, Dr. Croft witnessed an interesting phenomenon. A male subject had been warned not to grip the steering wheel. For some reason, even after such exhortations, subjects very often do grip the wheel. At impact, the car is essentially thrust forward beneath the subject. This grip results in a violent stretching of the upper extremity and a pulling of the glenohumeral joint. It also acts to accentuate the extension of the neck. It seems likely that under such circumstances, direct shoulder injuries might occur.

Headaches

These are the result of either injury to the upper cervical spine, or reflex or protective muscle spasm in the neck. Trauma can also play an important role in converting postural faults into clinical headaches (105). Headaches are also attributed to TMJ dysfunction and, rarely, the Barré-Liéou syndrome. Magnússon et al. (1200) reported that headaches with both a frontal and occipital component responded best to surgical release in cases of occipital neuralgia. Rather than suggesting surgery as a treatment for these patients, present this for its differential diagnostic value.

Interscapular pain

This pain is caused either by direct injury to paraspinal muscles or, more commonly, as a result of referred (scleratogenous) pain from the neck. It also may be seen with disc lesions. In the chronic stage, one of the most common causes of this type of pain is myofascial pain disorder (MPD), which is chiefly a secondary effect.

Back pain

Croft and Foreman (315) found low back pain (LBP) in 57% of their CAD cases (71% in broadside collisions). Most recently we've confirmed this relationship (1240). Braaf and Rosner (316) found LBP in 42% of their cases. Hohl (240) found LBP in 35% of his cases. Twenty-five percent of the patients in Hildingsson and Toolanen's study (121) had low back pain. In a recent study of chiropractic therapy in CAD patients, it was found that patients with low back pain generally required more treatment than those with neck pain alone (349). In a prospective study, Gargan and Bannister (456) reported 32% of their group as having LBP and an additional 10% developed LBP as a late manifestation. This same percentage (32%) was found by Bring and Westman (450). Magnússon (131) reported an incidence of 48% with LBP. In their recent follow-up of 35 patients followed for an average of 10.8 years, Watkinson et al. (249) found that initially, 24% of the group complained of LBP. Of these, 5% resolved later. However, after a 10.8 year mean follow-up, 34% complained of low back pain. It probably represents chronic postural adaptations to pain that, in most cases, developed later. Squires et al. (1201) followed previously studied patients (242,249) and reported a prevalence of back pain in half of this group after 15 years. More recently 40.5% of Brison et al. (1329) group of rear

impacted subjects had low back pain at 24 month follow-up. And Jakobsson et al. (1443) found thoracic and lumbar spine injuries second only to cervical injuries in rear impact crashes. In a recent study it was found that of those with neck pain following CAD, 43% of females and 31% of males also had back pain--a statistic that is found frequently in this literature (1368).

The exact mechanism of low back injury in rear impact collisions, although not entirely clear, is probably multifactorial. Factors affecting the incidence, nature, and severity of low back injury in automobile crashes include the following: (1) position of the occupant in vehicle, (2) the use or non-use of seat belts and shoulder harness, (3) deployment of airbag system, (which are designed to deploy only with frontal impacts, but may deploy in more severe secondary collisions), (4) type of restraint system (i.e., conventional restraints vs. restraints with pretensioners), (5) stiffness of the seat back, (6) inclination of the seat back, (7) properties of the seat back padding, (8) degree of ramping, (9) vector and severity of the collision, (10) second collisions inside or outside the occupant's vehicle, (11) snugness of the restraint system, (12) positioning of the restraint system on the occupant, (13) positioning of the restraint system anchors within the vehicle, (14) physical makeup of the occupant, including stature, build, age, and level of fitness, and (15) preparedness for the collision.

Frontal impacts are the most frequent type of crash and are also responsible for the most severe injuries. However, controlling for the severity of the crash (i.e., the actual amount of force involved), injuries are much more frequent in rear impact collisions. One of the prime reasons for this is that drivers who hit other cars are typically aware of the impending collision in time to brace for the impact. Steering wheels, brake pedals, and floorboards, in combination with the ride down of vehicle crush, provide adequate additional support to prevent many injuries.

In side impacts, the occupants of most production car seats are offered little protection to lateral acceleration forces by either seat back or restraint system. This probably explains why Dr. Foreman and Dr. Croft found such a high incidence of low back pain following this type of collision. [Incidentally, it was later Dr. Freeman and Dr. Croft who confirmed this relationship (1240).] The initial response to broadside impact is lateral flexion toward the striking vehicle, with compression of spinal structures on the concave side and stretching of myofascial and other structures on the convex side. The lap belt will serve as an anchor for the pelvis thus preventing serious injury. It may, however, intensify bending moments in the lumbosacral or thoracolumbar spine, thus increasing the likelihood of soft tissue injury. Intervertebral disc injuries, as well as ligamentous and muscular injuries, are common.

Broadside collisions should be analyzed carefully. Forces incurred by striking and struck vehicles are not commonly perfectly perpendicular. For example, if a broadside collision occurs between two cars in an intersection, and both are traveling at 20 mph, the actual resulting vectors of deceleration for the occupants will be oblique to the direction they are traveling in: in the case of the car that is struck on the driver's side, the occupant will decelerate in a vector that is forward and toward the striking car; in the case of the striking car, the occupant will decelerate forward and opposite to the direction the other

car. Such oblique collisions are particularly difficult to describe both kinetically and kinematically. However, it is clear from studies conducted by Viano (206) that oblique rear impact collisions hold much greater potential for injury than the pure rear impact variety. This is also true for other vectors of collision.

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