

Whiplash injuries from MedLink Neurology available at www.medlink.com

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Synonyms

Acceleration-deceleration injury; [Cervical sprain](#); Cervical myofascial pain syndrome; [Chronic whiplash syndrome](#); Hyperextension injury; [Late whiplash syndrome](#); Whiplash

Historical note and nomenclature

Whiplash is an acceleration-deceleration mechanism of energy transfer to the neck that may result from rear-end or side-impact motor vehicle collisions. [Common whiplash](#) is a trauma causing cervical musculoligamental sprain or strain due to hyperextension-flexion and excludes fractures or dislocations of the cervical spine, head injury, or alteration of consciousness.

Chronic or [late whiplash syndrome](#) refers to persistent symptoms present more than 6 months after the accident. Other terms that some clinicians prefer include "[cervical sprain](#)," "[cervical myofascial pain syndrome](#)," "acceleration-deceleration injury," and "hyperextension injury." Terms for this type of injury in other languages include *le coup du lapin* in French and *schleudertrauma* in German ([Evans 1995](#)).

In 1995, the Quebec Task Force proposed a classification system for whiplash injuries ([Spitzer et al 1995](#)). Grade 1 signifies neck complaints of pain, stiffness, or tenderness without physical signs. Grade 2 represents neck complaints and musculoskeletal signs including decreased range of motion and point tenderness. Grade 3 indicates neck complaints and neurologic signs including decreased or absent deep tendon reflexes, muscle weakness, and sensory deficits. Grade 4 includes neck complaints and fracture or dislocation.

Controversy about late whiplash syndrome is paralleled by similar controversy about the sequelae of usually mild head and neck injuries that often occurred in railway accidents in the second half of the 19th century: "railway spine" ([Evans 1994](#)). In publications from 1866 to 1882, Erichsen proposed that these injuries were due to "molecular disarrangement" or anemia of the spinal cord. In 1879, Rigler proposed compensation neurosis as the cause of an epidemic of compensation claims for railway accidents in Prussia. In 1888, Oppenheim disagreed with this explanation and popularized the term "traumatic neurosis." Charcot felt that posttraumatic symptoms were actually due to hysteria and [neurasthenia](#). Throughout the 19th century in the United States, as today, there were misgivings about physicians becoming involved in medico-legal cases. A common concern was that physicians were available as hired guns with any opinion for a price.

The whiplash mechanism of injury may have first been recognized in United States Navy pilots who developed neck injuries from acceleration-deceleration forces when their planes were launched by catapults following World War I. Although the pilots were shortly thereafter provided with headrests and shoulder harnesses, automakers in the civilian sector took some 50 years to provide the same safety equipment. Crowe, an orthopedist, is often cited as coining the term "whiplash" during a lecture in 1928 ([Crowe](#)

1964); however, the first use I have found in the medical literature appeared in an article by another orthopedist, Davis, in 1945 (Davis 1945).

The term "whiplash" became widely used in the 1950s. In an influential paper in 1953, Gay and Abbott provided a good clinical review, but incorrectly attributed the injury to flexion followed by hyperextension of the neck in rear-end collisions (Gay and Abbott 1953). In 1955, Severy and colleagues reported a pioneering series of staged rear-end collisions using humans and anthropomorphic dummies and correctly identified the sequence of hyperextension followed by flexion of the neck (Severy et al 1955). Human volunteers were used in the front car at collision speeds up to 10 mph; dummies were used for higher-velocity collisions. However, volunteers were used in the rear car even in 20-mph collisions without injury, as the flexion-extension injury is not nearly as harmful. This observation should be remembered when critics of whiplash injuries wonder why they rarely, if ever, see the drivers of the rear car as patients.

Many clinicians believe that whiplash primarily results in myofascial injuries. In 1938, Kellgren described distinctive patterns of referred pain from injection of different muscles with a 6% solution of sodium chloride (Kellgren 1938). An American orthopedist, Steindler, used the terms "trigger point" and "myofascial pain" for the first time in 1939 (Steindler 1939).

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Clinical manifestations

Table 1 lists the sequelae of whiplash injuries, which include neck and back injuries, headaches, dizziness, paresthesias, weakness, cognitive, somatic, and psychological sequelae, as well as visual symptoms and rare sequelae (Evans 1996; 2000).

Table 1. Sequelae of Whiplash Injuries

Neck and back injuries

- Myofascial
- Fractures and dislocations
- Disc herniation
- Spinal cord compression
- Spondylosis
- Radiculopathy
- Facet joint syndrome
- Increased development of spondylosis

Headaches

- Muscle contraction headache
- Greater occipital neuralgia
- Temporomandibular joint injury
- Migraine
- Third occipital headache

Dizziness

- Vestibular dysfunction
- Brainstem dysfunction
- Cervical origin
- Barre syndrome
- Hyperventilation syndrome

Paresthesias

- Trigger points
- Thoracic outlet syndrome

- Brachial plexus injury
- Cervical radiculopathy
- Facet joint syndrome
- Carpal tunnel syndrome
- Ulnar neuropathy at the elbow

Weakness

- Radiculopathy
- Brachial plexopathy
- Entrapment neuropathy
- Reflex inhibition of muscle contraction by painful cutaneous stimulation

Cognitive, somatic, and psychological sequelae

- Memory, attention, and concentration impairment
- Nervousness and irritability
- Sleep disturbances
- Fatigability
- Depression
- Personality change
- Compensation neurosis

Visual symptoms

- Convergence insufficiency
- Oculomotor palsies
- Abnormalities of smooth pursuit and [saccades](#)
- Horner syndrome
- Vitreous detachment

Rare sequelae

- Torticollis
- Tremor
- Transient global [amnesia](#)
- Esophageal perforation and descending mediastinitis
- Hypoglossal nerve palsy
- Superior laryngeal nerve paralysis
- Long thoracic neuropathy
- Spinal accessory neuropathy
- Cervical epidural hematoma
- Internal carotid and vertebral artery dissection

Sixty-two percent of patients presenting to the emergency room after a motor vehicle accident complain of neck pain ([Deans et al 1986](#)). The onset of neck pain occurs within 6 hours in 65% of patients, within 24 hours in 28% of patients, and within 72 hours in the remaining 7% of patients ([Deans et al 1987](#)). Most neck pain is due to [cervical sprain](#), a myofascial injury ([Bohr 1996](#)). Cervical disc herniations, cervical spine fractures, and dislocations are uncommon. Facet (zygapophyseal) joint injury at different levels can produce characteristic patterns of [referred pain](#) over various parts of the occipital, posterior cervical, shoulder girdle, and scapular regions ([Dwyer et al 1990](#)). Neck pain may arise from at least 1 facet joint in 54% of patients with [chronic pain](#) from whiplash injuries ([Barnsley et al 1993](#)). Injury of the soft tissues of the upper cervical spine may also be responsible for pain complaints. Kaale and colleagues performed cervical spine [MRI](#) studies on 92 subjects with chronic neck pain following whiplash injuries (whiplash associated disorder, grade 2 using the Quebec classification) and 30

random controls (Kaale et al 2005). Lesions of the alar ligaments showed the most pronounced association with the severity of neck pain and functional disability. There was a weaker association with lesions of the posterior atlanto-occipital membrane and transverse ligament.

In a prospective study of 180 patients seen within 4 weeks of the whiplash injury, 82% complained of headaches, which were occipitally located in 46%, generalized in 34%, and in other locations in 20% (Balla and Karnaghan 1987). The pain was present more than half the time in 50% of the patients. Headaches following whiplash injuries are usually of the muscle contraction type and are often associated with greater occipital neuralgia (Magnusson 1994). Greater occipital neuralgia or referred pain from trigger points from suboccipital muscles can produce a pattern of radiating pain variably over the occipital, temporal, frontal, and retro-orbital distribution. Whiplash trauma can also injure the temporomandibular joint and cause jaw pain often associated with headache (Brooke and LaPointe 1993). Headache may be referred from the C2-3 facet joint that is innervated by the third occipital nerve and is appropriately called a "third occipital headache" (Bogduk and Marsland 1986). C2-3 facet joint injury can result in pain complaints in the upper cervical region, extending at least onto the occiput and, at times, toward the ear, vertex, forehead, or eye. Using third occipital nerve blocks to diagnose the condition, the prevalence of this type of headache among patients with persistent headaches after whiplash injury has been reported as ranging from 38% to 50% (Lord et al 1994; 1996a). Occasionally, whiplash injuries can precipitate recurring common, classic, and basilar migraines de novo (Weiss et al 1991). Trigeminal sensory impairment of uncertain etiology has also been reported (Sterner et al 2001). In a study of patients with chronic posttraumatic headaches, 37% had tension-type headaches, 27% had migraine, 18% had cervicogenic headache, and 18% did not fulfill criteria of a particular category (Radanov et al 2001).

In 1 study of 262 patients with persistent neck pain and headaches for 4 months or longer after the injury, symptoms were reported as follows: vertigo, 50%; floating sensations, 35%; tinnitus, 14%; and hearing impairment, 5% (Oosterveld et al 1991). Posttraumatic vertebral insufficiency and dysfunction of the vestibular apparatus, brainstem, cervical sympathetics (Barre syndrome), and cervical proprioceptive system have all been postulated as causing dizziness. Hyperventilation syndrome can also occur in patients who are anxious and in pain, producing dizziness and paresthesias periorally or of the extremities, either bilaterally or unilaterally.

In 1 study, 33% of patients with symptoms but no objective findings complained of paresthesias acutely, and 37% reported paresthesias after a mean follow-up of 19.7 months (Norris and Watt 1983). Paresthesias can be referred from trigger points, brachial plexopathy, facet joint syndrome, entrapment neuropathies, cervical radiculopathy, and spinal cord compression. Thoracic outlet syndrome is commonly caused by whiplash injuries, occurring 4 times more often in women than in men (Capistrant 1986). Thoracic outlet syndrome has been controversial, as at least 85% of cases are of the nonspecific neurogenic or so-called "disputed type" that is a diagnosis of exclusion. This nonspecific type may actually be a myofascial pain syndrome with referred pain from the anterior neck muscles, such as the anterior scalene, or from the shoulder area from the pectoralis minor and not due to neural or vascular compression. Entrapment neuropathies can occur from several mechanisms. Carpal tunnel syndrome can be caused by acute hyperextension of the wrist on the steering wheel (Label 1991). If the patient has a cervical radiculopathy or neurogenic thoracic outlet syndrome from the injury, then a double crush syndrome resulting in carpal tunnel syndrome or cubital tunnel syndrome may ensue.

Complaints of upper extremity weakness, heaviness, or fatigue are common after whiplash injuries even when no evidence is present of cervical radiculopathy, myelopathy, brachial plexopathy, or entrapment neuropathy. The nonspecific type of

thoracic outlet syndrome can produce these complaints. Alternatively, patients may have a sensation of weakness or heaviness because of reflex inhibition of muscle due to pain that can be overcome by more central effort (Aniss et al 1988).

In a study of patients with chronic symptoms after a whiplash injury, cognitive, psychological, and somatic symptoms occurred in the following percentages: nervousness and irritability, 67%; cognitive disturbances, 50%; sleep disturbances, 44%; fatigability, 40%; disturbances of vision, 38%; symptoms of depression, 37%; headache, 85%; neck pain, 100%; vertigo, 72%; and brachialgia, 60% (Kischka et al 1991). These symptoms are nonspecific and are also common in patients with **postconcussion syndrome**, chronic pain syndrome, depression, and anxiety neurosis. Psychological factors (ie, premorbid neurosis) are commonly cited as the cause of persistent complaints; however, psychosocial factors, negative affectivity, and personality traits are not significant in predicting the duration of symptoms (Radanov et al 1991). Instead, cognitive and psychological symptoms may be due to somatic symptoms (Radanov et al 1994a; Wallis et al 1998). Although a 2-year prospective study did find that symptomatic subjects were impaired on tasks of divided attention but not on memory tests (Di Stefano and Radanov 1995), another prospective study of 39 patients found no evidence of cognitive deficits (Karlsborg et al 1997). It is controversial whether persistent neuropsychological deficits following whiplash injury are evidence for mild **traumatic brain injury** (Taylor et al 1996).

A variety of other problems may follow whiplash injuries. About one third of patients complain of interscapular and low **back pain** after whiplash injuries. Patients often report visual symptoms, especially blurred vision, usually due to convergence insufficiency (Brown 2003), although oculomotor palsies can occasionally occur (Burke et al 1992). Rare sequelae are listed in Table 1.

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Clinical vignette

A 45-year-old man employed as a handyman and carpenter was a passenger in a pickup truck. He was hit from behind by a sport utility vehicle while stopped on the freeway and was jarred. In the emergency room, a cervical spine series was negative and he was diagnosed with a **cervical sprain**. He saw an orthopedist with complaints of neck pain with an intensity of 8 out of 10 without upper extremity sensorimotor symptoms or signs. There was no prior history of neck problems.

He was placed on nonsteroidal antiinflammatory medications and sent to physical therapy 3 times weekly for 20 weeks (billed at \$13,626). A cervical spine **MRI** revealed a posterocentral C5-6 disc protrusion without neural compression. He underwent 2 cervical **epidural** steroid injections (billed at \$2700 each) without benefit. Two left superior trapezius **trigger point** injections resulted in 80% improvement. He was discharged from treatment 7 months after the accident with a diagnosis of a cervical sprain and given a full release to work. As part of his lawsuit against the driver of the vehicle that hit him, he gave a deposition 21 months after the accident. He reported intermittent neck pain and no plans for additional medical treatment.

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Etiology

Physicians generally attribute symptoms of **common whiplash** within the first 3 months to soft tissue injuries; however, when symptoms persist, the etiology of the **chronic or late whiplash syndrome** is controversial (Evans et al 1994). Nonorganic explanations advanced for persistent complaints include emotional problems, a culturally conditioned and legally sanctioned illness or cultural expectations (Mills and Horne 1986; Russell and Ferrari 2008), social and peer copying (Livingston 1993), secondary gain and malingering (Schmand et al 1998; Cassidy et al 2000), expectations for recovery (Carroll et al 2009), and demands for an explanation outside the realm of organic psychiatry and neurology (Pearce 1994). Harth critiques the unrealistic cultural expectations argument (Harth

2008). In addition, persistent complaints of those involved in low-speed, rear-end collisions are not seen in volunteer subjects exposed to speed changes from 4 to 14 km/h (Castro et al 1997). However, in another study, approximately 29% and 38% of the subjects exposed to 4 and 8 km/h speed changes, respectively, experienced other symptoms with cervical symptoms, with headaches predominating (Brault et al 1998).

A study that retrospectively examined the incidence of chronic symptoms from persons reporting rear-end motor vehicle accidents to police in Lithuania, where few people are covered by insurance, also challenged the organicity of chronic complaints (Schrader et al 1996; Obelieniene et al 1999). Chronic pain and headaches were no more common in 202 accident victims than in controls. The authors concluded that expectation of disability, a family history, and attribution of preexisting symptoms to the trauma may be important determinants for those who develop chronic symptoms. Although the results are intriguing, the study is probably not valid because of significant sampling bias (Bjorgen 1996; Freeman et al 1999). Despite the faults of this study, the medico-legal setting is important. When the tort compensation system in Saskatchewan, Canada was changed to a no-fault system without payments for pain and suffering, the number of claims decreased by about 25% (Cassidy et al 2000).

Pobereskin performed a similar study in the United Kingdom of 503 adults (66% female) who reported a rear end collision to the Devon and Cornwall police (response rate of 44%). Of the respondents, 78% had neck pain lasting for more than a week and 52% still had pain at 1 year (the 1 year response rate was 80.5%). The most important predictors of pain at 1 year were the initial neck visual analogue scale score (1.03, 1.01 to 1.05) and the presence of a compensation claim (4.09, 1.62 to 10.32). There was no improvement in symptoms once the claim was settled. In fact, people who had settled their claim by 2 years seemed more likely to have neck pain of similar severity compared to those who had ongoing claims. Pobereskin states, "This suggests that something about the stress and anxiety of the claim itself that tends to prolong symptoms in people seeking compensation" (Pobereskin 2005). However, it is also possible that the subjects had persistent pain complaints because they were really injured and were not cured by a verdict. The low response rates and disproportionate percentage of female respondents may have biased the outcome of the study.

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Pathogenesis and pathophysiology

Both animal and human studies have demonstrated structural damage from whiplash type injuries. In different species of monkeys, experimentally caused acceleration and extension injuries have revealed a variety of lesions: muscle tears, avulsions, and hemorrhages; rupture of the anterior longitudinal and other ligaments, especially between C4 and C7; avulsions of disc from vertebral bodies and disc herniations; retropharyngeal hematoma; intralaryngeal and esophageal hemorrhage; cervical sympathetic nerve damage associated with damage to the longus colli; nerve root injury; cervical spinal cord contusions and hemorrhages; cerebral concussion; and gross hemorrhages and contusions over the surface of the cerebral hemispheres, brainstem, and cerebellum (MacNab 1964; Ommaya et al 1968).

Human studies have similarly revealed damage of multiple structures. An MRI study done within 4 months of the whiplash-type injury revealed ruptures of the anterior longitudinal ligament, horizontal avulsion of the vertebral end plates, separation of the disc from the vertebral end plate, occult fractures of the anterior vertebral end plate, acute posterolateral cervical disc herniations, focal muscular injury of the longus colli muscle, posterior interspinous ligament injury, and prevertebral fluid collections. An MRI study of 92 patients with chronic whiplash injuries found abnormal tectorial and posterior atlanto-occipital membranes (Krakenes et al 2003a) and alar (Krakenes et al 2002) and transverse ligaments (Krakenes et al 2003b) in the injured as compared to controls. Autopsy series have shown injuries similar to those in the animal studies, including

injuries to intervertebral discs and soft tissue injuries of facet joints (Taylor and Kakulas 1991; Taylor and Twomey 1993).

Ishikawa and associates provide evidence that some patients with persistent symptoms may have a dural cerebrospinal fluid leak (Ishikawa et al 2007). They performed a prospective study of 124 patients with neck injuries and at least 2 of the following symptoms occurred for at least 3 months after the injury: headache, cervical pain, dizziness, nausea, visual impairment, auditory symptoms, and memory loss. Fifty-eight patients were excluded for a variety of reasons. The remaining 66 patients (whiplash injuries in 51) underwent radioisotope cisternograms, and 37 (28 with whiplash injuries) were found to have cerebrospinal fluid leaks, most in the thoracic and/or lumbar spines with a mean duration of symptoms of 33 months. Thirty six of the patients underwent 2.2 ± 0.7 lumbar epidural blood patches with significant improvement at 6 month follow-up. MRI scans of the brain to demonstrate diffuse dural enhancement or confirmation of cerebrospinal fluid leaks by MRI of CT-myelography are not reported. Reproduction of this study would be of interest.

Central hyperexcitability or sensitization (sensitization of spinal cord neurons which results in increased responsiveness to peripheral stimuli) is a possible mechanism for increased levels of subacute pain and chronic pain. In a study of 80 subjects evaluated within 1 month of the accident, acute whiplash subjects with higher levels of pain and disability were distinguished by sensory hypersensitivity to a variety of stimuli suggestive of central nervous system sensitization (Sterling et al 2004). These responses occurred independently of psychological distress. Spinal cord hypersensitivity was also suggested in a study of 29 patients with whiplash pain as compared to controls who had significantly lower reflex thresholds of the nociceptive withdrawal reflex on stimulation of the sural nerve (Banic et al 2004).

In a prospective study of 76 whiplash subjects evaluated within 1 month of injury and then 2, 3, and 6 months postinjury, whiplash groups demonstrated local mechanical hyperalgesia in the cervical spine at 1 month postinjury (Sterling et al 2003). This hyperalgesia persisted in those with moderate-to-severe symptoms at 6 months but resolved by 2 months in those who had recovered or reported persistent mild symptoms. Only those with persistent moderate-to-severe symptoms at 6 months demonstrated generalized hypersensitivity to all sensory tests. These changes occurred within 1 month of injury and remained unchanged throughout the study period. These findings suggest that those with persistent moderate-to-severe symptoms at 6 months display, soon after injury, generalized hypersensitivity suggestive of changes in central pain processing mechanisms. This phenomenon did not occur in those who recover or those with persistent mild symptoms.

Compared with controls, chronic whiplash patients had muscle hyperalgesia and large areas of referred pain after intramuscular injections of hypertonic saline both in the infraspinatus and anterior tibialis muscles, which suggest a generalized central hypersensitivity (Koelbaek Johansen et al 1999). Similar findings in whiplash patients have also been reported with electrical cutaneous and intramuscular stimulation of both the neck and lower limbs (Curatolo et al 2001).

Finally, in a study of brainstem-mediated antinociceptive inhibitory reflexes of the temporalis muscle of 82 patients with acute posttraumatic headache following whiplash injury, abnormal durations and latencies were present as compared to controls (Keidel et al 2001). This suggests a transient dysfunction of the brainstem-mediated reflex circuit, mainly of the late polysynaptic pathways. The reflex abnormalities are considered as a neurophysiological correlate of the posttraumatic (cervico)-cephalic pain syndrome and point to an altered central pain control in acute posttraumatic headache due to whiplash injury.

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Epidemiology

In 2007, there were 10,600,000 motor vehicle accidents, including 3,350,000 rear-end collisions, in the United States ([National Safety Council 2009](#)). No reporting system exists and, thus, the actual number of whiplash injuries per year is unknown; however, if Dolinis' finding that 35% of Australian drivers in rear-end collision sustained whiplash injuries ([Dolinis 1997](#)), then more than 1 million persons in the United States may have whiplash injuries yearly. Although neck injuries can commonly occur after side- or front-impact collisions, rear-end collisions are responsible for about 85% of all whiplash injuries ([Deans et al 1986](#)). In a low-velocity, rear-end collision, occupants of the vehicle struck are more likely to develop neck pain than are the occupants of the striking or rear vehicle, who sustain a flexion type injury ([Severy et al 1955](#); [MacNab 1964](#)). In rear-end collisions, the incidence of whiplash injuries decreases as crash severity increases ([Kahane 1982](#)). Neurologists frequently evaluate and treat patients with whiplash injuries. According to 1 survey, neurologists see an average of 10.3 patients per month with whiplash injuries ([Evans et al 1994](#)). Women, especially in the 20- to 40-year age group, have persistent neck pain more often than men do by a ratio of 7:3 ([Balla 1980](#)). The greater susceptibility of women to whiplash injuries might be due to a narrower neck with less muscle mass supporting a head of roughly the same volume or a narrower spinal canal compared with men ([Pettersson et al 1995](#)). Another possible explanation is the greater early neck displacements in the average woman with less weight in the stiffer car seats used in recent years as compared to the yielding seats ([Viano 2003](#)).

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Prevention

General measures directed at driving safety, such as reducing the number of drunk drivers or improving the driving habits of young men, would reduce the number of whiplash injuries. Vehicle safety measures have been implemented. Proper use of head restraints can reduce the incidence of neck pain in rear-end collisions by 24% to 28.3% ([Morris 1989](#); [Viano and Gargan 1996](#)); however, in 1 study, only 10% of drivers had headrests adjusted to the most favorable position to prevent neck extension ([Viano and Gargan 1996](#)). In a study of a self-aligning head restraint available designed to move upward and forward by occupant motion in a rear crash, providing earlier neck support even when the head restraint is positioned low, the incidence of whiplash injuries was reduced from 18% in a Saab with a standard restraint to 4% in a Saab with the self-aligning restraint ([Viano and Olsen 2001](#)). Center high-mounted stop lamps have reduced the number of rear-end collisions. Even though seat belt use should be encouraged, 73% of occupants wearing a seat belt develop neck pain as compared to 53% not wearing seat belts ([Deans et al 1987](#)).

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Differential diagnosis

Although most whiplash injuries result in myofascial or facet joint injuries, less common consequences include fractures, cervical disc herniations, spondylitic [radiculopathy](#), and myelopathy. Table 1 lists the differential diagnosis for other symptoms after whiplash injuries.

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Diagnostic workup

Cervical spine series are often obtained to exclude the occasional fracture ([Lovell and Galasko 2002](#)). In patients with abnormal neurologic examinations or persistent complaints suggesting the possibility of [radiculopathy](#) or myelopathy, a cervical spine MRI study may be indicated. In patients without radicular complaints, cervical MRI studies have a low yield ([Voyvodic et al 1997](#)). A cervical myelogram followed by CT scan may be helpful if the MRI study cannot be done or if the study demonstrates equivocal findings. In some cases, especially for spondylitic disease, CT scan or myelography may be more sensitive than MRI for nerve root compression. Asymptomatic radiographic

findings are common and, thus, it is often difficult to determine what findings are new and what findings are preexisting. Cervical spondylosis and degenerative disc disease occur with increasing frequency with older age and are often asymptomatic (Friedenberg and Miller 1963). Cervical disc protrusions are also common in the general population and are often asymptomatic. Protrusions occur in 20% of patients who are 45 to 54 years of age and in 57% of patients who are older than 64 years (Teresi et al 1987).

EMG and nerve conduction studies may be helpful to demonstrate evidence of radiculopathy, brachial plexopathy, or entrapment neuropathies. Somatosensory and dermatomal evoked potential studies are not adequately sensitive or specific to justify use for the evaluation of possible radiculopathy.

Cerebral hypoperfusion has been reported after whiplash injuries. In a small study of 6 patients compared to 12 controls, positron emission tomography and SPECT evidence of parieto-occipital hypometabolism was reported (Otte et al 1997). One possible explanation is stimulation of pain-sensitive afferents in the cervicotrigenal system, which could have widespread effects on local vasoactive peptides and the cranial vascular system (Otte et al 1995; 1997). In another study of 20 chronic whiplash patients, most with cognitive complaints (Lorberboym et al 2002), 65% had brain perfusion abnormalities in 1 or more regions. Eight out of 15 patients tested had abnormal P300 event-related potential studies. There was no significant correlation between the SPECT findings or the P300 results and the scores of attention and working memory on neuropsychological testing. There was, however, close agreement between the SPECT and P300.

However, in another functional imaging study of 21 patients with late whiplash syndrome, Radanov and colleagues found no significant correlations between regional perfusion or metabolism in any brain area on SPECT or positron emission tomography studies and the scores of divided attention or working memory. There were significant relations between state anxiety and divided attention (Radanov et al 1999). In an MRI-based brain volumetry study of 21 patients with persistent concentration and memory deficits that are subjectively reported but not objectively verifiable as neuropsychological deficits, there were no abnormalities suggesting diffuse axonal injury as compared to matched controls (Sturzenegger et al 2008).

Although HmPAO and ECD brain SPECT studies in patients with late whiplash syndrome and cognitive complaints have demonstrated parieto-occipital hypoperfusion (Otte et al 1995; 1996), similar findings have also been seen in patients with nontraumatic chronic cervical pain (Otte et al 1995). Depression can also cause perfusion abnormalities (Alexander 1998) and FDG-PET does not allow reliable diagnosis of metabolic disturbances for individual patients (Bicik et al 1998); therefore, FDG-PET and HmPAO SPECT should not be used as diagnostic tools in the routine evaluation of patients with late whiplash syndrome (Bicik et al 1998).

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Prognosis and complications

Studies on the prognosis of whiplash injuries are difficult to compare because of multiple methodological differences, including selection criteria of patients, prospective and retrospective designs, patient attrition rates, duration of follow-up, and treatments used (Barnsley et al 1994a; Evans 1997; 2000; Cote et al 2001; Kamper et al 2008). Although most patients may have only soft tissue injuries, imaging studies other than plain spine films have not been routinely performed.

Multiple studies have documented that neck pain and headaches can persist in significant numbers of patients. A well-designed prospective study reported the following percentages of patients with complaints of neck pain and headaches, respectively, at various times after the injury: 92% and 57%, 1 week; 38% and 35%, 3 months; 25% and 26%, 6 months; 19% and 21%, 1 year; and 16% and 15%, 2 years (Radanov et al 1994b; 1995). Symptoms present 2 years after injury are still present 10 years after the

injury ([Gargan and Bannister 1990](#)). In a 17 year follow-up study of patients first seen in the emergency room, 55% reported persistent neck pain with no gender difference as compared to 29% of control subjects ([Bunketorp et al 2005](#)).

The following risk factors have been reported for persistent symptoms ([Pettersson et al 1995](#); [Radanov et al 1995](#); [Dolinis 1997](#); [Karlsborg et al 1997](#); [Kasch et al 2001](#); [Kasch et al 2008](#); [Carroll et al 2009](#)).

Table 2. Risk Factors for Chronic Symptoms

Accident mechanisms

- Inclined or rotated head position
- Unpreparedness for impact
- Car stationary when hit

Occupant's characteristics

- Older age
- Female gender
- Pretraumatic headache for injury-related headache
- Significant life events unrelated to the accident
- Depression
- Early expectations of prolonged recovery

Symptoms

- Intensity of initial neck pain or headache
- Occipital headache
- Interscapular or upper [back pain](#)
- Multiple symptoms or [paresthesias](#) at presentation

Signs

- Reduced range of movement of the cervical spine
- Objective neurologic deficit

Radiographic findings

- Preeexisting degenerative osteoarthritic changes
- Abnormal cervical spine curves
- Narrow diameter of cervical spinal canal

Although psychological factors such as neurosis are commonly cited as the cause of persistent symptoms, a prospective study of 78 consecutive patients with whiplash injuries demonstrated that psychosocial factors, negative affectivity, and personality traits were not significant in predicting the duration of symptoms ([Radanov et al 1991](#)). An additional study found that psychosocial factors and vocation were not predictive of persistent symptoms during a 1-year follow-up ([Radanov et al 1994b](#)). In another prospective study, successful radiofrequency neurotomy resolved psychological distress in those with chronic neck pain due to a single painful facet joint following whiplash injuries ([Wallis et al 1997](#)).

Some evidence exists for a role for psychological factors. In a prospective study comparing the results of the Structured Clinical Interview for [DSM-IV](#) for patients with [chronic pain](#) from whiplash injuries after 1 year to those who had recovered, a history of psychiatric disease (most commonly depression) was more common in patients with chronic symptoms both before and after the accident ([Kivioja et al 2004](#)). Richter and colleagues performed a 6 month prospective study of 43 consecutive patients with grade 1 or 2 whiplash associated disorders with a 74% follow-up rate ([Richter et al 2004](#)).

Psychological factors were more relevant than collision severity in predicting the duration and severity of symptoms. Atherton and colleagues also found that a high level of general psychological distress and a precollision history of widespread body pain predicted the persistence of symptoms (Atherton et al 2006).

Radanov and colleagues performed a prospective study to assess psychological risk factors for disability (Radanov et al 1993). At 6 months, 7% of the patients had partial or complete disability. The disabled and nondisabled patients still symptomatic at 6 months did not differ with respect to psychosocial stress, negative affectivity, and personality traits as initially assessed at baseline. Nygren reported that permanent medical disability occurred in 9.6% of patients involved in rear-end collisions and in 3.8% of patients involved in front- or side-impact accidents (Nygren 1984). Mayou and Bryant found no special psychiatry of whiplash neck injury (Mayou and Bryant 2002).

Many clinicians and certainly the insurance industry and defense attorneys believe that pending litigation is a major cause of persistent symptoms that promptly resolve once the litigation is completed (Livingston 1993). In 1 study of litigants, 88.7% were found to have inconsistent, nongenuine abnormalities on examination (Peterson 1998). Schmand and colleagues performed neuropsychological studies of patients with late whiplash syndrome reporting memory or concentration problems (Schmand et al 1998). In the context of litigation, the prevalence of underperforming was 61% as defined by a positive score on the malingering test.

However, most of the literature does not support this position. Litigants and nonlitigants have similar recovery rates (Pennie and Agambar 1990) and similar response rates to treatment for facet joint pain (Lord et al 1996a; Sapir and Gorup 2002). Filing a lawsuit within 1 month of the injury does not influence recovery at 1 year (Kasch et al 2001). The majority of plaintiffs who have persistent symptoms at the time of settlement of their litigation are not cured by a verdict (Shapiro and Roth 1993; Evans 1996). Certainly, some patients exaggerate or lie about persisting complaints to help or make their legal case. Neurotic, histrionic, or sociopathic patients may thrive on the attention and endless treatments recommended by some physicians and encouraged by some plaintiff attorneys. The clinician should evaluate the merits of each case individually (Conomy 1998). The available evidence does not support bias against patients just because they have pending litigation (Benoist 1998).

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Management

Few prospective controlled studies of treatment exist (Spitzer et al 1995; Rodriguez et al 2004; Kroeling et al 2005; Verhagen et al 2007; Hurwitz et al 2008). Early mobilization of the neck using the Maitland technique followed by local heat and neck exercises produces more rapid improvement after acute injuries than does the use of a cervical collar and rest (Mealy et al 1986) and is as effective as physical therapy performed during the first 8 weeks after the injury (McKinney et al 1989). In a large, randomized prospective study, Vassiliou and colleagues found that physical therapy and active exercise for 10 sessions produced significant pain reduction compared to a soft collar only over 7 days at 6-week and 6-month assessments (Vassiliou et al 2006). Another study found significant benefit of active over passive physical therapy (Dehner et al 2009). In another study, the outcome was better for patients who were encouraged to continue engaging in their normal preinjury activities than it was for patients who took sick leave from work and were immobilized during the first 14 days after the injury (Borchgrevink et al 1998). Cervical traction may be no more effective than exercises alone (Pennie and Agambar 1990). Overall, the evidence for benefit of physical therapy compared to no treatment is limited (Verhagen et al 2007).

A review of educational interventional studies did not show benefit for pain, function, global perceived effect, quality of life, or patient satisfaction (Haines et al 2009). In one study in an insurance setting, early multidisciplinary evaluation and advice actually

increased the risk for chronic neck pain when participants were followed-up 3 years postinjury (Pape et al 2009).

In a small, prospective, randomized study of patients with neck pain and musculoskeletal signs (75% of study group) and others with additional neurologic signs (25% of study group), the administration of high-dose methylprednisolone within 8 hours of the injury prevented extensive sick leave as compared to the placebo controls at 6 months (Pettersson and Toolanen 1998). The evidence does not support the use of cervical epidural steroid injections for neck pain without radicular symptoms and signs, and there is limited evidence of efficacy for neck pain with radicular symptoms (Peloso et al 2006).

According to uncontrolled studies, trigger point injections can be beneficial for acute and chronic myofascial injuries (Garvey et al 1989). One group reports benefit from injection of sterile water in, or subcutaneous to, trigger points caused by whiplash injuries (Byrn et al 1991; 1993). A single case report describes cervicogenic headache relief for 3 months at a time with injections of botulinum toxin in a trapezius muscle tender area (Hobson and Gladish 1997). A small series found a reduction in neck pain and headache following trigger point injections with botulinum toxin as compared to the control groups who received trigger point injections with saline (Freund and Schwartz 2002). However, another study found no benefit of injection of cervical and thoracic trigger points with botulinum toxin type A as compared to injections with saline (Ferrante et al 2005). Transcutaneous electrical nerve stimulator units may also be beneficial.

Treatment of pain arising from facet joint injury may be effective. A controlled prospective study showed a lack of effect of intraarticular corticosteroid injections in the cervical facet joints for chronic pain after whiplash injuries (Barnsley et al 1994b). Percutaneous radiofrequency neurotomy and lower cervical medial branch neurotomy should be used cautiously for the treatment of chronic facet joint pain documented by anesthetic blocks (Lord et al 1995). In a small study of patients with chronic facet joint pain confirmed with double-blind, placebo-controlled local anesthesia, percutaneous radiofrequency neurotomy with multiple lesions of target nerves provided at least 50% relief for a median duration of 263 days compared to similar relief for 8 days in the control group (Lord et al 1996b).

In a study of 15 patients with cervical spondylotic radicular pain after whiplash injuries, a mean of 3.7 therapeutic selective nerve roots blocks was not effective (Slipman et al 2004). Alpar and colleagues have proposed that chronic neck and shoulder pain in chronic whiplash syndrome is due to carpal tunnel syndrome with normal electrodiagnostic studies. In a series of 38 such patients, 90% were reported as improved (Alpar et al 2002). This study should certainly be replicated.

Routine treatment for acute injuries often consists of pain medications, nonsteroidal antiinflammatory medications, muscle relaxants, and the use of a cervical collar for 2 to 3 weeks. Neurologists frequently prescribe range-of-motion exercises, physical therapy with a variety of modalities, and transcutaneous electrical nerve stimulator units. Standard treatments are provided for posttraumatic headache. For example, some patients with greater occipital neuralgia benefit from nerve blocks (Sjaastad 1990; Anthony 1992).

For persistent complaints, tricyclic antidepressants are often prescribed. The chronic frequent use of narcotics, benzodiazepines, barbiturates, and carisoprodol should be sparingly recommended because of the potential of habituation. Medication abuse headaches can also develop. Patients with chronic complaints seek out a multitude of unproven treatments such as chiropractic adjustments (occasionally under general anesthesia), acupuncture, prolotherapy, and pain clinics. Well-meaning practitioners often uncritically provide treatments without allowing for the importance of placebo effects, whereas others are more economically motivated. Some plaintiffs and their attorneys encourage excessive treatment in an attempt to magnify an alleged injury.

Clearly, adequately controlled prospective studies of current treatments and more effective treatments for chronic pain are greatly needed ([Newman 1990](#); [Spitzer et al 1995](#)). Until then, a compassionate, sympathetic approach by the neurologist might result in greater patient satisfaction and reduce unnecessary expenditures from patients' therapeutic quests.

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Pregnancy

Medications should be avoided or, if necessary, prescribed in cooperation with the obstetrician.

[Top ↑](#)

Anesthesia

Not applicable.

[Top ↑](#)

ICD codes

ICD-9:

Whiplash injury: 847.0

ICD-10:

Whiplash injury: S13.4

[Top ↑](#)

Associated disorders

Compensation neurosis

Facet joint injury

Posttraumatic [vertigo](#)

[Thoracic outlet syndrome](#)

[Top ↑](#)

Related summaries

[Headache associated with head trauma](#)

[Headache associated with cervical spine dysfunction](#)

[Head trauma: neurobehavioral aspects](#)

[Myofascial pain syndrome](#)

[Posttraumatic movement disorders](#)

[Posttraumatic sleep disturbance](#)

[Psychogenic neurologic disorders](#)

[Tension-type headache](#)

[Top ↑](#)

Differential diagnosis

myofascial injury

facet joint injuries

fracture

cervical disc herniations

spondylitic [radiculopathy](#)

[myelopathy](#)

dislocation

disc herniation

spinal cord compression

spondylosis

radiculopathy

facet joint syndrome

increased development of spondylosis

muscle-contraction headache

greater occipital neuralgia
temporomandibular joint injury
[migraine](#)
third occipital headache
vestibular dysfunction
brainstem dysfunction
cervical origin
Barré syndrome
[hyperventilation syndrome](#)
trigger points
[thoracic outlet syndrome](#)
brachial plexus injury
cervical radiculopathy
[carpal tunnel syndrome](#)
[ulnar neuropathy at the elbow](#)
brachial plexopathy
entrapment neuropathy
reflex inhibition of muscle contraction by painful cutaneous stimulation
memory, attention, and concentration impairment
nervousness and irritability
sleep disturbances
fatigability
depression
personality change
compensation neurosis
convergence insufficiency
oculomotor palsies
abnormalities of smooth pursuit and [saccades](#)
[Horner syndrome](#)
vitreous detachment
[torticollis](#)
tremor
[transient global amnesia](#)
esophageal perforation and descending mediastinitis
hypoglossal nerve palsy
superior laryngeal nerve paralysis
cervical [epidural hematoma](#)
internal carotid and vertebral artery dissection

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Demographics

For more specific demographic information, see the Epidemiology, Etiology, and Pathogenesis and pathophysiology sections of this clinical summary.

Age

02-05 years
06-12 years
13-18 years
19-44 years
45-64 years
65+ years

Population

None selectively affected.

Occupation

None selectively affected.

Sex

female>male, >2:1

female>male, >1:1

Family history

None

Heredity

None

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