

REVIEW ARTICLE

ACUTE AND CHRONIC WHIPLASH DISORDERS – A REVIEW

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Objective: This review examines acute and chronic whiplash-associated disorders to facilitate assessment, treatment and rehabilitation for further research and evidence-based practises.

Design: A review of the literature.

Results and conclusion: Whiplash-associated disorders account for a large proportion of the overall impairment and disability caused by traffic injuries. Rarely can a definite injury be determined in the acute (or chronic) phase. Crash-related factors have been identified, and several trauma mechanisms possibly causing different injuries have been described. Most whiplash trauma will not cause injury, and the majority of patients (92–95%) will return to work. Litigation is not a major factor. Cognitive impairments are not the same as brain injury. Variables such as pain intensity, restricted motion, neurological symptoms and signs, together with central nervous system symptoms can be used to predict a situation with risk of remaining complaints. Influences of other factors – the same as for other chronic pain conditions – also exist. Persistent/chronic pain is not merely acute pain that persists over time; changes occur at different levels of the pain transmission system. Chronic whiplash-associated disorders are associated with problems concerning social functioning, daily anxieties and satisfaction with different aspects of life. Adequate information, advice and pain medication together with active interventions might be more effective in the acute stage. Early multidisciplinary rehabilitation focusing on cognitive-behavioural changes might be of value. To develop specific treatment and rehabilitation, it is important to identify homogenous subgroups.

Key words: neck, whiplash, risk factors, prevalence.

J Rehabil Med 2004; 36: 193–210

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Submitted May 5, 2003; accepted March 2, 2004

INTRODUCTION

This review examines acute and chronic whiplash disorders (WAD) to facilitate assessment, treatment and rehabilitation for further research and evidence-based practises. Evidence-based

guidelines concerning WAD are sparse due to lack of randomized, controlled and prospective studies. Assessment, investigation and treatment strategies for WAD should be based on science and experience-based practise, remembering that statistical results generally are on a group level and cannot be correlated directly to individuals.

During the First World War, it became clear that the violence inflicted on the cervical spine of pilots during emergency ejection was great enough to cause a blackout for several seconds and accidents occurred that were due to a whiplash effect. This understanding resulted in the development of a headrest and a shoulder harness to protect pilots. Although a great deal of biomechanical research has been done to diminish forces acting on the head and neck (1), even today a substantial number of the pilots report neck pain (50%) (2). Higher G-forces (>5 Gs) and age have been identified as significant risk factors (3, 4).

In 1928, Crowe (5) introduced the term *whiplash injury* to describe 8 injuries caused from rear-end motor vehicle accidents. This “injury” has usually been associated with motor vehicle accidents (MVA), in particular rear-end accidents, although several authors include other injury mechanisms (6). In 1955, it was reported that even in low impact (20 km/h) rear-end collisions the head and neck were exposed to acceleration forces that could cause injuries that present symptoms 12–24 hours later (7). When a car is hit and accelerated by 17 km/h within 100 ms, the car and the driver experience 5 Gs (8). Although major (fatal) injuries have decreased due to the use of seatbelts and safer car design, minor traffic accidents and whiplash injuries have increased over the past decades (9–11). Not all patients who suffer a whiplash trauma develop acute or chronic disabling symptoms, but there is a substantial risk of developing chronic symptoms after whiplash trauma (12). Chronic conditions related to whiplash account for a large number of the overall impairment and disability from traffic injuries (13, 14). Whiplash injuries result in an increase of cost for medical care, increase in disability pension, decrease in income, decrease in income tax and an overall decrease in well being (6, 14–16).

TERMINOLOGY

Due to the heterogeneity of definitions and classification in the literature, the Scientific Monograph of the Quebec Task Force on Whiplash Associated Disorders (QTF) in 1995 adopted some definitions that will be used in this review (6). *Whiplash* is an

acceleration-deceleration mechanism of energy transfer to the neck. It may result from rear or side impact from a motor vehicle, but can occur during diving or other mishaps. The impact can result in bony or soft tissue injuries (*whiplash injury*), which in turn can lead to a variety of clinical manifestations (*whiplash associated disorders, WAD*).

WHAT IS THE INCIDENCE OF WHIPLASH TRAUMA?

The incidence of whiplash trauma corresponds to minor traffic accidents. Therefore, it is not possible to give a reliable figure because one can assume that many people involved in a minor traffic accident do not report the accident to the police. Two studies comparing police reports and actual injuries found that 15% and 35% of the subjects sustained an injury (pain) (17, 18). Thus the incidence of a whiplash trauma is at least 3 times the incidence of an acute symptomatic whiplash injury.

ACUTE PHASE

What is the incidence of acute WAD?

The annual incidences vary from 0.8–4.2 per 1000 inhabitants (10, 15, 19–22). The variation depends on the population studied, type of accidents, inclusions, and exclusions.

Can traffic accidents result in acute conditions?

In 1955, Severy et al. (7) noted that even low impact (20 km/h) rear-end collisions could cause injuries that could present symptoms 12–24 hours later. Most symptoms develop in the first 24 hours (6, 23–25). According to our experience, stiffness of the neck is often the first symptom and pain of the neck and headache will develop somewhat later (within the first 24 hours). However, there is a considerable variation and some subjects report pain immediately after the trauma. Some Swedish insurance companies have stated that accident-related symptoms (i.e. pain) must develop within 72 hours after the accident. According to our experiences accident-related pain will usually develop within 3 days, but there is no scientific evidence for a definite time limit. Some subjects are very shocked after the MVA, which could mask pain and other symptoms for some hours.

Are there any signs of tissue injury in acute WAD?

The prevalence of positive signs from plain X-ray and flexion-extension projections (used to detect segmental instability) are rare. Signs of straight or kyphotic curve and degenerative spondylosis are seen in about 25% of the cases (23), but similar prevalences are seen in control groups. Neuroimaging seldom shows any sign of an injury (26–28).

Rises in serum creatine kinase (CK) generally occurs within 24 hours after muscle injury. However, in a prospective study of whiplash injury after low-velocity rear-end collisions, only 2 out of 25 subjects had CK increases above the normal limit in the

acute phase (within 24 hours after trauma) (29). Patients with WAD examined within 3 days after the trauma had a significant increase in pro-inflammatory tumour necrosis factor (TNF)- α and interleukin (IL)-6 and of anti-inflammatory IL-10 (30). These changes were normalized when the patients were re-examined after 24 days.

In a recent systematic review (26) 3 studies including a study that used surface cryo-planing microtomy autopsy technique of fatal road accidents and a control group (non-accidental death) (31–33), indicated that subtle lesions in the cervical intervertebral disc and zygapophysial joints could be clinically relevant in WAD. A very high proportion of these lesions are not seen on X-ray post mortem (31).

Controlled diagnostic blocks of cervical zygapophysial joints of patients with chronic WAD have shown that the zygapophysial joints can be the source of pain (34, 35). The results of these studies might indicate that these joints were injured in the trauma situation. The authors reported prevalences between 54% and 60% from their consecutive patients with chronic WAD.

In contrast to injuries to ligaments and discs, muscle injuries would normally heal in a few weeks and reasonably not cause chronic pain according to some authors (20).

In conclusion, a definite injury can very seldom be determined in the acute (or chronic) phase. Techniques that can show an injury (other than fractures) of a tissue in the neck are needed. Present incomplete data indicate that different types of injuries can be found. Hence acute WAD is a syndrome with different subgroups that have to be identified in order to optimize management and treatment.

Injury mechanisms

The knowledge of injury mechanisms is incomplete, although it is often loosely defined as an acceleration injury caused by hyperextension, the primary mechanism of injury. In recent years, the theory of a plain hyperextension flexion injury has more or less been abandoned (36). Theories based on both human and “dummy” experiments reveal a more complex mechanism of injury with the “S” shaped curve of the vertebra and forces that act (retraction) on the neck rather than a pure hyperextension/flexion injury mechanism (36–39). A neck injury criterion has been suggested that considers the relative velocity between the top and bottom of the cervical spine, but the validity has not been systematically investigated (40). In animal experiments, high-pressure gradients in the cerebrospinal fluid can cause injury to the spinal ganglia (40). Pressure injuries on the spinal cord that have been registered in experiments have not been linked to real life injuries (41). Head contact during trauma causes a different loading pattern of the cervical spine and induces higher loads (42).

The muscles are recruited relatively late during the trauma event (recruitment start after 100–125 ms) (43). Sternocleidomastoid muscles and the cervical paraspinal muscles contract in response to impact and acceleration; furthermore, potential muscle injury exists if muscles contract to resist head motion (i.e. eccentric contraction) (44, 45).

Based upon above referred autopsy studies and the studies of controlled blocks of the zygapophyseal joints, one might expect that there are several mechanisms causing different injuries.

What is known about risk factors for acute WAD?

Crash-related factors. A tow bar at the rear of the car has been identified as a risk factor for WAD (46, 47). The risk of neck injury is related to seat-belt use (47) and how the head restraint is positioned (48). In a crash, a car seat's ability to transfer the energy to the occupant's neck and head can be an important factor in determining the risk for WAD (49). A correlation between change of velocity and acute symptoms has been reported (47). More recent studies have shown that the magnitude of the peak acceleration (G) and the shape of the pulse according to crash pulse registration are more important (47). In addition, car design and construction affect the risk of acute and chronic WAD (50). A correlation between the mass of the car and the risk for WAD has been found in some studies (51). Bus or truck accidents, being a passenger colliding with a moving object seems to increase the risk of a slow recovery (6, 52). Castro et al. (53) exposed healthy subjects to a placebo (a low-velocity rear-end collision) and at follow-up they reported that 18%, 20%, and 10% of the subjects indicated symptoms from 3 days to 4 weeks after the trauma. They also reported that certain psychological profiles were linked to the report of symptoms. The validity of this study has been questioned (54).

Individual factors. Several studies found a female dominance of whiplash injuries (6, 19, 55). In a cohort of 246 subjects involved in rear-end MVA, 2 factors were identified as independent risk factors for "whiplash injury": a history of neck injury and female sex (56). In a study of MVA in a northern Swedish town were found no gender differences in acute WAD (21). Head position (rotated or inclined) and low awareness of the accident have been proposed to be negative factors (57, 58).

Which symptoms and signs are most common in acute WAD?

Symptoms. According to several studies, the most common symptoms reported within the first week of an accident are pain in the neck and head and stiffness (Table I) followed by interscapular pain, paresthesias in arms and hands, dizziness, temporomandibular symptoms, visual and auditory symptoms, cognitive problems and emotional/psychological disturbances. The psychological consequences have attracted relatively little attention, but acute stress disorders, phobic anxiety associated with travel and post-traumatic stress disorder can develop (59, 60). Higher emotional responses in the acute phase were associated with more neck pain 4 weeks later (61). Neuropsychological symptoms such as decreased concentration, memory problems and irritability in the acute phase can be confirmed at neuropsychological testing (i.e. decreased attention and concentration) (62). The authors discussed different aetiologies as responsible for these results. Many patients that seek healthcare in the acute phase will present several symptoms (63). This is reasonable because acute nociception reflecting

Table I. Prevalence of symptoms in acute (within 1 week) whiplash-associated disorders (WAD) based on the following studies: (i) Hildingsson & Toolanen 1990 (n = 93) (23); (ii) Norris & Watt 1983 (n = 61) (78); (iii) Radanov et al. 1991 (n = 78) (184); (iv) Maimaris et al. 1988 (n = 102) (229); (v) Drottning et al. 1995 (n = 107) (61)

Symptoms	n in total sample	Prevalence (%)	Based on studies (1-5)
Neck pain	334	94	1-4
Neck stiffness	195	96	1, 3
Interscapular pain	107	35	5
Headache	334	44	1-4
Numbness/paresthesias	232	22	1, 3, 4
Dizziness	232	15	1, 3, 4
Visual symptoms	232	12	1, 3, 4
Auditory symptoms	232	13	1, 3, 4
Sleeping problems	78	35	3
Memory problems	78	15	3
Intrusion/avoidance	107	30	5

tissue injury can result in pain and also in reflex withdrawal, and in increases in arousal, emotional, autonomic, and neuro-hormonal responses (64).

To facilitate a better understanding of the different symptoms in the acute phase, we will briefly summarize some aspects of pain. According to the *model of Descartes*, it was held that injury activated specific pain receptors and fibres that project impulses through a spinal pathway to a pain centre in the brain (65). The pain experience was held to be proportional to the injury (66), and psychological contributions to pain were not recognized in this model, and consequently pain without signs of organic disease were thought to reflect psychological or psychiatric conditions. Even today, this model influences clinical practise and the interpretation of results from different research studies concerning WAD. The *gate control theory of pain* (66) proposed that the brain had a dynamic role in modulating the inputs in the spinal dorsal horns, which were sites associated with considerable dynamic activities. The brain is an active system that filters, selects, and modulates input (65), and psychological factors were seen as an integrated part of pain processing. The gate control theory of pain has had a great influence on pain research and a model of pain with respect to the function of the brain has been formulated, the *neuromatrix model* (67, 68). A genetically built-in matrix of neurones for the whole body produces characteristic nerve-impulse patterns for the body and the different somatosensory qualities that we feel. The output ("the neuro-signature") from this neuromatrix (i.e. patterns of nerve impulses) will secondarily determine the pain experience and behaviour. The matrix is activated and modulated by inputs from the body (for instance nociception), but it can act without any inputs (for example, the phantom limb or as a consequence of "the thermal grill illusion"¹ (69)). Figure 1 summarizes possible

¹ When people place their hands on an interlaced pattern of warm and cool metal they usually feel a burning pain sensation, although no potential tissue-damaging stimulus is presented (69).

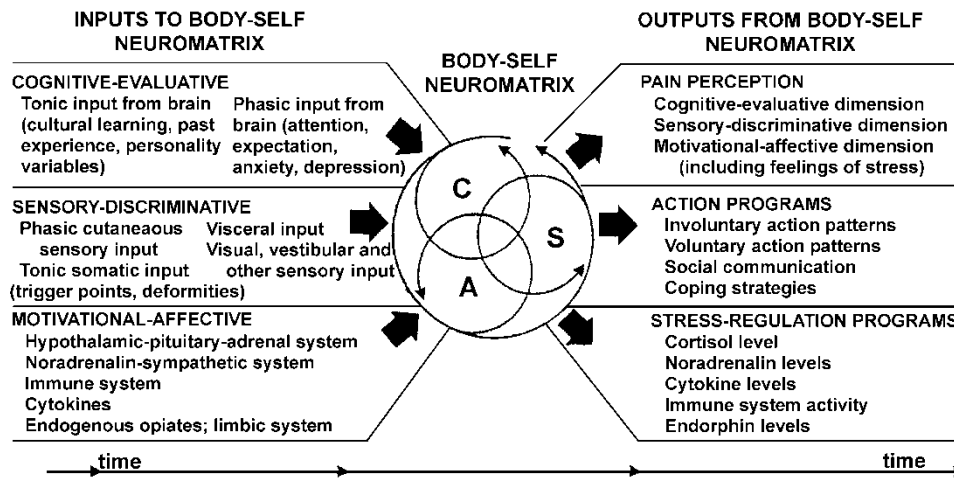


Fig. 1. Pattern-generating mechanism or neuromatrix modulated by multiple inputs and the internal milieu (217). (Reprinted with permission from Melzack R. Pain and the neuromatrix in the brain. *J Dental Educ* 1999; 65: 1378–1382.)

factors that contribute to the output pattern from the neuromatrix that in turn produces the sensory, affective, and cognitive dimensions of pain experience and behaviour. The theory notes that injury can produce pain and disrupt the homeostatic regulation systems of the brain, producing “stress”. Stress will lead to neural, hormonal and behavioural activities with the goal of re-establishing homeostasis. Pain behaviours can be generated or perpetuated by previously conditioned cues in the environment or by the expectation of pain and suffering. In accordance with this model, pain is processed by a highly distributed brain system, although individual brain regions and networks of brain regions exhibit some degree of specialization (69, 70). Activated brain areas are lateral and/or medial thalamus, S-I and S-II somatosensory cortex, anterior/posterior insular cortex, anterior cingulate area 24b, perigenual cingulate, posterior cingulate, supplementary motor area, periaqueductal grey, prefrontal areas, and cerebellum (71). The distributed system provides many routes for transmission of nociceptive information, ensuring the ability to detect injury in extreme situations. The number of brain regions activated appears to be a function of the *intensity* of pain (71, 72). The intensity appears to be processed by the least specialized brain mechanisms and serve as a foundation upon which other components of pain processing are constructed (70). Serial interactions exist between pain intensity, pain unpleasantness, and secondary affect (for instance suffering); pain intensity is a causal factor (72). The fact that several parts of the brain are activated also reflects the complex nature of the pain experience including sensory-discriminative, affective-motivational and autonomic components (69). Thus the generally found relationships between pain and certain cognitions and fear-avoidance beliefs are not surprising from this point of view (73–75). Factors such as attention, hypnotic suggestion, mood, emotional state, attitudes and expectations could alter pain perception according to a recent review (69).

In conclusion, acute pain will result in both psychological

and physiological responses. Psychological concerns and other experiences due to events in the impact situation also influence acute pain.

Signs. X-ray is used in the acute phase in order to exclude fractures and major injuries to ligaments. The prevalence of positive signs from plain X-ray and flexion-extension projections (used to detect segmental instability) are low in acute WAD. At clinical examination stiffness and decreased range of motion (ROM) due to pain and pain at palpation of soft tissues are most frequently noted. According to several authors, decreased ROM and pain in muscles and bony structures when palpated can be useful indicators of dysfunction (23, 63, 76, 77). A negative relationship exists between ROM of the neck and neck pain in the acute stage (77). Definite neurological deficits seem to be rare (23, 78, 79).

How can acute WAD be classified?

There are different systems of classification, but all systems are based on signs and symptoms. It would have been a great advantage to have a classification based on the type of injury or the pain generating mechanisms. One main difference between the systems of classification presented below is that 2 of them incorporate neuropsychological symptoms. This is not done in the QTF classification, which is most used in research and clinical practise.

The classification of Radanov and co-workers. Radanov and co-workers (80) suggested a classification based on subjective complaints and formal testing of self-estimated cognitive impairment, divided attention, and speed of information processing:

- Lower cervical spine syndrome (LCS) accompanied by cervical and cervicobrachial pain.
- Cervicoencephalic syndrome (CES) characterized by headache, fatigue, dizziness, poor concentration, disturbed accommodation, and impaired adaptation to light intensity.

Table II. Classification of whiplash associated disorders according to Quebec Task Force (6)

Anatomical-clinical axis* Grade	Description
0	No complaint about the neck, no physical sign(s)
I.	Neck complaint of pain, stiffness, or tenderness only. No physical sign(s)
II.	Neck complaint and musculoskeletal sign(s) (i.e. decreased range of motion and point tenderness)
III.	Neck complaint and neurological sign(s) (i.e. decreased or absent deep tendon reflexes, weakness, and sensory deficits)
IV.	Neck complaint and fracture or dislocation
Time axis	Duration (days)
	<4
	4–21
	22–45
	46–180
	>180

* Symptoms and disorders that can be presented in all grades include deafness, dizziness, tinnitus, headache, memory loss, dysphagia and temporomandibular joint pain.

The classification of Quebec Task Force on Whiplash Associated Disorders. The QTF proposed a clinical classification of WAD based on an anatomical-clinical axis and a time axis (Table II). Unfortunately, there are not many studies that consequently have used this classification. In a review, it was concluded that none of the articles published completely satisfied the QTF definitions either before or after the introduction in 1995 (81). Nevertheless, the classification of QTF seems to have had some impact on either the published inclusion or exclusion criteria. The classification had prognostic value in that the risk for WAD at follow-up increased with increasing grade in the acute stage (82). Similar results have also been reported in other studies (21, 83). These results are in line with earlier results that poor recovery is related to severity of symptoms initially (84). Hartling et al. (82) proposed a subdivision of Grade II with or without limited ROM.

The classification of Gerdle and co-workers. The classification of the injuries is based on an anatomical axis and a time axis (Table III). This classification seems to have advantages especially when combined with the QTF classification (85), and some indirect proof of this is found in 2 other recent studies (63, 86).

What is the effect of different treatments in the acute stage?

Many therapies such as heat, ice, ultrasound, acupuncture, massage, subcutaneous sterile water and soft collar have not been evaluated or show little or no evidence of efficacy (6). A more recent review of conservative treatment (only randomized studies included) indicates that active treatments show a beneficial effect on at least 1 of the primary outcome measures – preferably pain (87). Adequate information and successive mobilization without a collar as early as possible supported by a physiotherapist are the best treatments for reducing pain and increasing ROM at 6 months follow-up according to 1 study (88). Similar results have been reported by other authors but without any difference in sick leave at 6-month follow-up (89). Due to the fact that high levels of pain and psychological symptoms might have a negative impact on recovery (21, 61, 82–84) it also seems important to apply adequate information

Table III. Classification of Gerdle and co-workers of whiplash associated disorders (230)

Anatomical axis	Description
Category A	Head, neck and shoulder
Category B	Head, neck and shoulder and arm†
Category C	Head, neck and shoulder and central nervous system‡
Category D	Head, neck and shoulder, arm† and central nervous system‡
Time axis	
Number of weeks with complaints	Acute ≤12 weeks Chronic ≥12 weeks

† Numbness, pain, and motor weakness.

‡ Dizziness, visual problems, sensitivity to light and sound, stress intolerance, cognitive problems.

(for instance concerning natural development, prognosis and common symptoms) and advice concerning mobilization together with pain medication in the acute phase.

Recovery rates

Depending on what cohort is studied, recovery rates of acute WAD vary a great deal between different studies. The literature concentrates mainly on the occurrence of symptoms and pays relatively little attention to severity and consequences on daily life (90). Few epidemiological or clinical studies of WAD have adequately documented its prognosis (91). However, studies with fairly large groups of non-selected patients overall show that at follow-up (generally >6 months) approximately 5–8% of the patients have major problems that negatively affect their work capacity (6, 21, 22, 63, 92). Using a population-based cross-sectional survey, Côté et al. (93) reported that neck pain and severe headaches are more prevalent in subjects with a history of neck injury due to a car collision. One-third of those with residual symptoms suffered from work disability compared with 6% in the subgroup without residual symptoms.

The figures for reporting remaining complaints (generally pain) are reported in a much larger variation (18–60%) (21, 22, 63, 94, 95). In an analysis of 121 patients registered 17 years earlier for neck complaints at emergency departments,

55% had residual disorders (neck pain, radiating pain and headache were most common) associated with an original accident (96). Berglund et al. (97) studied drivers exposed to a rear-end collision without and with acute WAD 7 years earlier and used 2 comparison groups unexposed to MVA and found no increased risk of neck/shoulder pain in those exposed to MVA but without acute WAD (relative risk 1.3, 95% CI 0.8–2.0). Those with acute WAD showed a significantly increased risk (relative risk 2.7, 95% CI 2.1–3.5). They found that 39.6% of the subjects with acute WAD in connection with the collision reported neck pain 7 years later compared with 14.5% of the unexposed comparison group. Exposed subjects with acute WAD compared with unexposed subjects had increased relative risks (1.6–3.7) for headache, and thoracic and low back pain, fatigue, sleep disturbances and ill health 7 years later (98).

Many consider 6 months as an endpoint in the estimation of outcome in WAD, but some recent studies have reported that recovery can occur for long periods (several years) after injury (10, 99).

In conclusion, especially for work capacity the majority of patients with acute WAD has a good prognosis, which is important to inform the patients about in the acute phase.

CHRONIC PHASE

As with other pain conditions, some patients will develop chronic conditions. However, there is debate about whether chronic WAD exists or not and how pain and other symptoms in chronic WAD are maintained. To achieve a more balanced view upon chronic pain, we will briefly summarize some relatively new aspects concerning pain.

Persistent/chronic pain is not a simple extension in time of acute pain: “A cascade of changes initiated by tissue or neural damage elicits a collection of synaptic, neurotransmitter, and modulatory events that mimics synaptic plasticity and remodeling similar to that seen in learning and memory” (100). Thus chronic pain is not different from acute pain in degree; in part it is linked to unique mechanisms in the peripheral and central nervous system (101). Plastic changes occur at different levels of the pain transmission system. For instance, pain is associated with major changes at multiple levels of the somatosensory system in patients with chronic cervical radicular pain (102). The plastic changes induced in the peripheral and central nervous system will probably be less reversible when the stimuli remain and may be related to the pathogenesis of chronic pain (103).

Physiological (acute) pain is initiated in the peripheral terminals of nociceptors with the activation of nociceptive transducer receptor/ion channel complexes, which then generates a depolarizing current (64). When the current is sufficient, action potentials are generated and conducted to the spinal cord (dorsal horns) (64). The thinly myelinated A δ and unmyelinated C fibres are involved in transmission of nociception. The A δ fibres mediate “first” pain (i.e. fast, acute, sharp and pricking pain)

while C-fibres mediate “second” pain (i.e. delayed, burning, dull and aching pain). Different subtypes of A δ -fibres and C-fibres have been identified (104). These might have different relevance with respect to chronic pain, and there are silent nociceptors that only become active in pathological states (105). In the dorsal horn transmitters are released; glutamate acting on alpha amino-3-hydroxy-5-methyl-4-isoxazole-propionate (AMPA) and kainate subtypes of glutamate receptors (64, 106). The spinal neurons are controlled by peripheral inputs, interneurons and descending controls (103). There is no single pathway of pain, but the pathways from the dorsal horn to the brain are multiple (for instance, the spinothalamic tract, spinoreticular tract, spino-cervicothalamic tract, the post synaptic dorsal column fibres) (105, 107, 108).

Both pain syndromes with (e.g. in repetitive strain injuries, focal hand dystonias in writers or keyboard operators) and without (i.e. phantom limb sensation) afferent inflow, can result in plastic changes of sensorimotor cortex (102, 109). A disorganized or inappropriate cortical representation of proprioception may result in pathological pain (109). The degree of expansion of the cortical representation zone is related to the nociceptive input (110). Chronic pain may lead to the development of somatosensory pain memories that manifest themselves in alterations in the somatotopic map in the somatosensory cortex as well as altered processing in associative areas (110). Psychological processes can influence the somatosensory pain memories. Plasticity changes probably also occur at subcortical levels (111). Abnormal brain chemistry and chemical network in chronic pain have been found and interpreted as results of long-term cortical reorganization (112). Hunt & Mantyh (105) concludes that noxious stimulation result in changes in gene expression within the CNS, and different chronic pain states generate unique neurochemical signatures in the nerve system.

Increasing evidence indicates that glia in the spinal cord dorsal horns can create and maintain chronic pain (113). The descending modulation involves both inhibitory and facilitating mechanisms in persistent pain (114). In part, severe pain may be the result of a net increase in endogenous descending facilitation (114, 115). This was found in patients with back pain; patients with acute pain had an inhibitory response while patients with chronic pain had an excitatory response (116).

There are different types of neuronal plasticity with relevance for pain. Woolf & Salter (64) suggested a conceptual framework for the contribution of plasticity in primary sensory and dorsal horn neurones to the pathogenesis of pain. They defined 3 forms of plasticity (activation, modulation and modification) that can elicit pain hypersensitivity. The plasticity responsible for clinical pain hypersensitivity (allodynia and hyperalgesia) has 2 general forms: modulation and modification (64).

Activation

An activity dependent and reversible plasticity will occur after repeated stimuli to nociceptive pathways; i.e. a progressive increase. In the nociceptor terminals changes in the transducers occur as a result of prior activation (*autosensitization*). In the

dorsal horn, neurones will *windup*; i.e. high frequency inputs result in release of neuromodulators and glutamate producing slow excitatory postsynaptic potentials (EPSPs) lasting several seconds. In addition, the activation of the N-methyl D-aspartate (NMDA) receptor can contribute. The net effect of these changes is a progressive increase of action potential discharge (64, 106).

Modulation

Modulation represents reversible changes in the excitability of primary sensory and central neurones, and the major mechanism is phosphorylation of receptor/ion channels or associated regulatory proteins (64). The modulation of the peripheral terminals of nociceptors (*heterosensitization*) is due to the exposure of the terminal to sensitizing agents (inflammatory mediators and neurotrophic factors). Different sensitizing molecules act on different receptors. Tissue injury leads to an inflammatory response, which includes the liberation of substances such as potassium ions, substance P, bradykinin and prostaglandins. Modulation of central pain pathways (*central sensitization*) is triggered by input from peripheral nociceptors and results in enhanced responsiveness of pain transmission neurones, which outlasts the initiating input (up to many hours) or require low-level peripheral drive to maintain it. Most of the activity in pathway neurones is sub-threshold and without pain. In central sensitization, low-threshold afferent inputs lead to pain and spread of pain. In addition, innocuous inputs will lead to amplified responses in pain pathways (106). These changes are linked to NMDA receptor activity. Studies of healthy subjects indicate that induction of central sensitization involves NMDA receptor mechanisms. In patients with chronic pain, NMDA receptor blockade inhibits abnormal temporal summation and sometimes other characteristics related to central sensitization (117). Referred pain (i.e. pain felt at a site remote from the site of origin/stimulation) has been discussed in relation to plasticity of dorsal horn and brain stem neurones (118, 119). The size of referred pain is related to the intensity and duration of pain. Proximal spread of referred pain is seldom seen in healthy subjects but often present in patients with chronic pain (118, 119).

Another mechanism for facilitation in the dorsal horn neurones can occur via a subtype of AMPA receptors. Central sensitization is also associated with depression of spinal inhibitory mechanisms (e.g. it requires NMDA activation).

Modification

Modification represents long-lasting alterations in the expression of transmitters/receptors/ion channels or in the structure, connectivity, and survival of neurones (64). Thus modification is a condition when the pain system is highly distorted. For instance, modification can involve an increase in gene expression (including novel genes) (120) that in turn results in increased synthesis of peripheral receptors (121). Hence it has been reported that the A δ fibres may function as nociceptors (122). A delayed denervation of C-fibres can occur after nerve injury. A

changed connectivity between neurones in the dorsal horn and transcriptional changes can occur.

Many patients with chronic pain also report symptoms other than pain. Recently, it has been suggested that plastic changes such as central sensitization might explain an increased prevalence of other complaints (123). Vangåite Vingen & Stovner (124) found that subjects with cervicogenic headache were significantly more sensitive to light and sound during headache-free periods than headache-free controls.

For some subjects with WAD, Munglami (125) proposes a model of ongoing peripheral input, individual genetic difference response to an injury and psychological disorders that cause sensitization. Some of the authors that discuss chronic WAD appear to perceive it as a single condition. But the injuries described above indicate that chronic (and acute) WAD is a syndrome. Data from pharmacological challenges indicate the existence of subgroups (126), and studies that investigate the degree of plasticity and reversibility are needed in the future to develop specific treatment and rehabilitation procedures.

What is known about risk factors for chronic WAD?

From 2 relatively large insurance cohorts it was reported that several socio-demographic factors (female gender, old age, having dependents, and not having full-time employment independently) and several symptoms and signs were associated with a slow and costly recovery (52, 86). In 2001, Côté et al. (91) in a systematic review included 13 studies satisfying their conceptual framework and concluded that besides baseline neck pain intensity, baseline headache intensity, and baseline radicular signs and symptoms, there is little consistency in the literature about the prognostic factors for the recovery of whiplash. Moreover, insurance and systems of compensation have an impact on recovery. Two of the included Lithuanian studies (127, 128) have been criticized; the former for major statistical problems (129). These studies claim that chronic WAD is very rare. The authors postulated that a large number of cases of chronic WAD are caused by expectation of disability and attribution of pre-existing symptoms to the neck trauma (127) and that symptoms after an acute whiplash injury are self limiting, brief and do not seem to evolve chronic WAD (128). The Lithuania studies were both based on police reports, there were mainly men involved in these accidents and low prevalences of fixed and operative seat belts. Moreover, the results of these 2 studies clearly contrast the 2 studies of Berglund et al. (97, 98) referred above.

In a later study from 2002, 353 persons involved in rear-end MVA were followed up to 2 years after visiting the emergency room (63). Risk factors of chronic WAD (excluding occasionally minor pain) were increased age, number of initial physical symptoms, and early development of upper back pain, upper extremity numbness, or weakness or disturbances in vision (63). For 296 patients visiting the emergency room due to MVA, female gender, low education and prior neck pain were independently associated with a poor outcome at follow-up on average 16 months later (21). Self-efficacy at an early stage after

whiplash injury predicted pain intensity and disability at 3-month and 1-year follow-up (83). In 123 patients, 1 month after visiting the emergency room due to a whiplash injury the variable interference in Multidimensional Pain Inventory had the strongest correlation with the outcome (130). The results of these and the other studies referred in this section indicate a complexity behind chronic WAD, which are reasonable to expect from other pain conditions.

Models of prediction

It is difficult for clinicians and insurers to predict accurately the outcome of patients with whiplash injuries, due to the lack of epidemiological studies on the prognosis of WAD (91). However, a few attempts have been presented in the literature. Radanov & Sturzenegger (131) employed their significant variables for poor outcome (impaired neck movement, history of pre-traumatic headache, history of head trauma, higher age, initial neck pain intensity, initial headache intensity, nervousness score, neuroticism score and test score on focused attention). Correct outcome prediction at 1 year was found in 88% of patients recruited from the insurance company. The authors conclude "that a comprehensive assessment of whiplash patients early after trauma enables physicians to identify patients at delayed recover". In a Canadian study on rear-end MVA, significant independent factors (increased age, number of initial physical symptoms, and early development of the following symptoms – upper back pain, upper extremity numbness, or weakness or disturbances in vision) – made the base for a simple clinical decision rule that requires asking up to 3 basic questions of each patient: (i) Did the MVC occur other than at an intersection in the city? (ii) Have you experienced upper back pain since the MVC?; and (iii) Do you still experience neck/shoulder pain? (63). In 118 cases, patients were identified with a sensitivity of 91.5%, a specificity of 51.4%, a positive predicted value of 50.7% and a negative predicted value of 91.7%.

Research on prognostic factors and prediction of chronic WAD indicate that some observations and signs in the acute phase (i.e. pain intensity, WAD grade, neurological symptoms and signs especially in the arm together with CNS symptoms) can be used to identify subjects with increased risk for chronic symptoms. However, more studies are needed.

What is the role of insurance factors?

Mayou & Bryant (132) propose influence of compensation on course, and outcome is complex, partly because proceedings are more likely, and also more likely to be prolonged, in those with the most distressing physical symptoms. Ninety-two percent of the patients participating in a rehabilitation program reported that they had had contact with a lawyer, regional social insurance office, or insurance company, and 6.4% had appealed or were going to appeal a decision from the insurance company or the social insurance authorities (133). Compared with other chronic pain conditions, compensation rates are much higher in WAD depending of the traffic insurance systems. Furthermore, in

Sweden travels during work and to/from work are included in the system for economic compensation according to work injury insurance act. Another factor might be that many of the victims are innocent (133). Litigation status did not predict employment status or psychological distress but influenced the report of pain (134). It is possible that frustration, anger, and stress could increase the perception of pain in patients with WAD during the litigation process (134). Cassidy et al. (135) studied the shift from a tort system allowing compensation for pain and suffering to a no-fault system and found that the no-fault system was associated with a decreased incidence and improved prognosis of whiplash injuries. Independent of system (tort or no-fault) neck pain intensity, physical functioning, and depressive symptomatology are associated with time-to-claim-closure (136). Employment status, heavy manual occupation, and a previous history of psychological disease were significantly associated with disruption of work (137). In their prospective study of road traffic accidents, Mayou & Bryant (132) found no differences in the proportion of compensation claim between those with WAD and those with bone injuries. The insurance companies' use of time-loss payments as an indicator of recovery excluding recurrences is considered inappropriate (138, 139).

In conclusion, different insurance systems, social contexts, and expectations will have different and substantial effects on the incidence and prevalence rate and on what the healthcare system is willing to diagnose/treat.

Symptoms and signs in chronic WAD

Pain. The most prevalent symptom in subjects who suffer chronic WAD is still pain, but several not directly pain-related symptoms are prevalent (Table IV). Also temporomandibular symptoms are prevalent in the chronic phase. The prevalences in the chronic phase vary between studies due to study population and study design. Typically, patients with sub-acute or chronic WAD that are referred to specialist clinics report pain from areas that theoretically (i.e. head, neck and shoulders) could have been injured during the trauma and from areas more distant. Buskila and co-workers reported that fibromyalgia according to the 1990 criteria of the American College of Rheumatology (ACR) was significantly more frequent following neck injury (22%) than following lower extremity fracture (1%) (140). The group with neck injury also had significantly higher prevalence of different symptoms when compared with leg injuries (140). In a review, it was concluded that there was some evidence for a relationship between trauma and fibromyalgia, but the evidence was not definite (141). Several neurophysiological studies have reported signs of central sensitization in patients with chronic WAD (76, 142–145). Due to the fact that general allodynia and/or fibromyalgia are associated with high levels of disability and low quality of life, it is important to apply assessments that can identify this subgroup of patients.

Range of motion. Recently, Kasch et al. (77) reported that neck mobility is not significantly reduced 3 months after trauma in contrast to the situation in the acute phase. In contrast, most other authors have reported reduced active ROM (146–148).

Table IV. Prevalence (%) of pain and other symptoms noted in some studies (23, 78, 94, 99, 210, 220) on chronic whiplash associated disorders (≥ 3 months)

Symptoms	Authors and year					
	Norris & Watt 1983 (n = 42)	Hildingsson & Toolanen 1990 (n = 41)	Radanov et al. 1995 (n = 21)	Provinciali et al. 1996 (n = 60)	Olivegren et al. 1999 (n = 22)	Sterner et al. 2002 (n = 32)
Neck pain	98	66	90	93	100	100
Neck stiffness		56				83
Headache	62	34	87	70	32	68
Shoulder pain		39	71	53	41	88
Back pain			33	30	9	64
Numbness/arm	55	19	24	27	41	40
Dizziness		10	23	73		43
Visual	19	15	43	18		14
Auditory	18	12	52			22
Sleeping problems			62	53		34
Concentration problems				67		34
Memory problems			57	40		25
Fatigue			71	60		

Disc degeneration. There is some evidence that whiplash injury can predispose one to premature degenerative disc disease (149, 150) and especially those with injuries to the ligaments might have an increased risk for localized/segmental changes. However the literature is not homogenous in this respect (151) and controlled studies are needed.

Increased muscle tension. In clinical practise, it is often assumed that patients with chronic pain have increased muscle tension. Several studies have reported that patients with WAD have increased levels of muscle tension (i.e. decreased ability to relax) according to surface EMG, but these findings are not specific for WAD (152–154). A recent study shows that the WAD group is heterogeneous with respect to muscle tension (155).

Neurological symptoms and signs. Neurological signs are reported to occur in both low and high frequencies that probably can be explained by the effects of patient selection and the criteria the authors use to define a sign (23, 78, 154, 156). Sensory disturbances in the face have been detected in a group of chronic WAD subjects compared with subjects who have recovered (157). In a prospective study, sensory impairment detected in sub-acute WAD remained for years in most cases (158), in some cases with deterioration over time.

Sensory disturbance symptoms (tingling and/or numbing) are relatively prevalent in the arm (Table IV). Irritation of the brachial plexus and symptoms of radicular irritation have been reported to be prevalent in the acute phase and associated with worse outcome (84, 159). Arm pain, sensory symptoms, and signs especially in the arm are often misinterpreted as signs of a cervical dischernia with radiculopathy (neuropathy) and seem to result in non-motivated MRT. The frequency of sensory disturbance noted in our clinical practise is relatively frequent in the pain area (neck and head) as well as outside the pain area (arm). Sensory alterations were originally described in the context of neuropathic pain conditions, but also nociceptive pain

can be associated with sensory disturbances. Hansson (160) characterized the sensory disturbances associated with nociceptive pain as intermittent and variable, non-neuroanatomical distributed, both hypo- and hyperesthesia occur, and one or several modalities can be involved. Sensory disturbances have been reported to occur both in the local pain area and in the area of pain referral in clinical nociceptive pain conditions as well as in experimental pain (161, 162). A significant positive correlation has been found between the pain intensity in the local pain area and the size of both the local pain area and the referred pain area (162). A thorough examination and explanation to the patient (who might perceive these symptoms as frightening) will probably be a better alternative in most cases than MRT and not delay other kinds of intervention.

In patients with chronic WAD and “atypical” carpal tunnel syndrome, significantly increased plasma levels of substance P and calcitonin gene-related peptide (CGRP) have been found (163). After operation, these levels were normalized. The authors suggest that “atypical” carpal tunnel syndrome can explain pain in the neck and shoulders in some cases. They proposed that stretching of the neck in the trauma situation injured the median nerve.

Otoneurological symptoms and signs. Otoneurological symptoms are common in the acute stage (62). Eye motility dysfunction can occur in patients with WAD (164) and appear to persist over time. In some patients, dysfunction appears to develop over time (164). Intentional saccadic eye movements showed impaired function in subjects with persistent complaints, which the authors suggested could indicate dysfunction of prefrontal and frontal cortical structures (165). In a prospective study of WAD (grade II and III), eye motility dysfunction and pathological neurootological (ABR) findings in a few WAD patients with severe symptoms could be explained by lesions on the brain/brainstem (166). Another alternative could be brain stem sensitization/alteration affected inhibition (167). Eye

movement disturbances are not pathognomic for WAD and are seen in other pain conditions such as fibromyalgia syndrome (168), tension headaches (169), and menstrual syndrome (170).

Persisting symptoms (164), restricted neck movement (171), dizziness/vertigo (172), self-reported reading problems (173) and driving problems (174) correlate with disturbed eye movement. Kinaesthetic tests have shown that patients with WAD have significantly greater joint position errors of the neck than control subjects (175–177). Within the WAD group, those with dizziness had greater errors than those without (177). The dizziness/vertigo findings suggest that injuries to the neck can cause dysfunction of the posture control system (174).

Psychological problems and post-traumatic stress. Stress disorders, phobic anxiety about travel and post-traumatic stress disorder can develop in the acute phase and remain for months (59, 60). In a 2-year follow-up, half of the respondents still reported travel anxiety, pain, fear and fatigue (178). Stressful life events unrelated to the accident and a high level of stress 1 month after acute WAD have been linked to a poor prognosis (179, 180). Obviously, having had a hard time before a trauma, high emotional response, and stress related to the trauma including loss of control and difficulties in making decisions lead to psychological problems and general stress (133).

In most cases with long-standing non-malignant pain, psychological stress is considered secondary to pain (181). One can expect that chronic WAD is under the same influence of probable potential risk factors as other “non-acceptable disabling conditions” (182), such as fear, depression, distress, pain severity and stress. Although psychological factors play a significant role in chronic pain, and in the aetiology of acute pain, they account for only a portion of the variance (183). Several authors have interpreted that psychological problems are a result rather than a cause of somatic symptoms in WAD (184, 185). Wallis et al. (186) used the Symptom Checklist 90-revised (SCL-90R) for assessing psychological status in WAD patients and found that WAD patients did not markedly differ from patients with chronic low back pain or patients with rheumatoid arthritis, but suggested that patients with WAD had another profile (187). However, the specificity of the proposed profile has not been confirmed and instead the psychological consequences of experiencing chronic pain from WAD are similar to the consequences found in patients with other musculoskeletal injuries (188). The personality profile (on average 2 days after trauma) did not differ between previously symptomatic, recovered and symptomatic WAD patients and thus did not predict outcome (189).

About 30–50% of WAD subjects, 3 months to 2 years post-injury, reported cognitive disturbances (memory and/or concentration difficulties) (Table IV). Neuroimaging studies so far have not shown any signs of functional brain damage (190). Neuropsychological tests of cognitive performance show lowered results compared with controls according to a recent review (191). There are several possible explanations for lowered cognitive performance such as pain intensity (190), post-traumatic stress disorder (PTSD) and failure to use effective coping strategies leading to post-traumatic stress-like symptoms

or depression (192). In a recent review, it was concluded that minor trauma exposure without a significant unconscious period, including amnesia, is unlikely to have caused diffuse axonal injury or brain trauma (193). An unconsciousness period of less than 10 minutes or an amnesia period that spans less than 4 hours was not likely to cause any lasting brain damage or dysfunctional mental sequel according to that review. With the prerequisite that patients with signs of head injury are anamnesticly excluded, it is generally not reasonable to expect any brain damages in patients with WAD with cognitive impairments using imaging techniques.

A minority (11–25%) also have signs of PTSD (60, 132, 133, 194). Symptoms such as memory loss, concentration problems, and fear and avoidance of driving can reflect an emotional response to intrusive memories of an accident (195). PTSD and other psychiatric complications can be maintained by psychological variables (reminders of the accident), ongoing physical problems, and disability (132) and these symptoms might remain for years for some subjects.

Coping

The concept of *coping*, having roots in the studies of stress (196) aimed at describing ways of coming to terms with a stressor. In the literature, studies that investigate the relationship between coping and WAD are rare. There is a debate whether coping should be considered a trait or whether situational aspects should be taken into consideration. In a prospective study of 59 patients with WAD, it was found that the importance of coping strategies as an explanatory factor for disability increased over time during the 1-year follow-up and the catastrophizing strategy (of significant importance from 6 weeks and onwards) was the strongest coping variable explaining disability (197). Furthermore, coping might have a mediating role between self-efficacy and disability (95) and the direct effect of self-efficacy upon disability decreased over time. In patients with acute WAD referred to an orthopaedic clinic, self-efficacy at an early stage strongly predicted (inverse correlation) pain intensity and disability at follow-up (at 3 and at 12 months) (83). In non-specific chronic pain patients with no prior depression, regression analysis supported self-efficacy as a mediator of the relationship between pain intensity and disability (198).

Catastrophizing has been found to be strongly associated with anxiety and depression scores in chronic pain patients (199–201). Vlaeyen et al. (202) showed a strong correlation between kinesiophobia and catastrophic cognitions in chronic low back pain. Some authors have also argued a strong association between estimations of one’s health and the way to cope (200, 203).

Coping strategies have been differentiated into active and passive ones (203), where *active* refers to instrumental actions as engaging in activities and using one’s body actively while *passive* refers to activities as withdrawal, resting, etc. It has been argued that active coping is related to a relatively high estimation of well-being and functional improvement in patients with low back pain and WAD (204, 205). Disabling pain was associated

with the use of passive strategies regardless of levels of active coping in neck and low back pain patients (206). Thus, active coping strategies were relatively less important when it came to mastering pain. Neither a strong relation between the degree of pain intensity and active coping nor relations between depression and coping combinations were found (206). In a study of 275 patients with chronic WAD, 3 subgroups of patients were identified with respect to coping strategies; whether or not active coping strategies were used seemed to have little influence on health-related quality of life and instead a mixture of used strategies appeared relevant (207). Health-related quality of life was significantly related to depression, number of not directly pain-related symptoms, catastrophizing cognitions and pain intensity.

Consequences in daily life

Turk (208) has pointed out that chronic pain is associated with a difficult situation: "It is a demoralizing situation that confronts the individual not only with the stress created by organic factors and pain, but with a cascade of ongoing stressors that compromise all aspects of the life of the sufferer. Living with chronic pain requires considerable emotional resilience and tends to deplete one's emotional reserve, and taxes not only the individual but also the capacity of family, friends, co-workers, and employers to provide support." In line with this statement, WAD in the chronic stage is associated with problems with social functioning, daily problems, and satisfaction with different aspects of life (209, 210).

What is known about treatment in the chronic stage?

Although many different treatment modalities are used for patients with chronic WAD, so far no convincing evidence exists for any treatment for chronic WAD. The efficacy of conservative treatments in patients with WAD has recently been systematically reviewed, but most studies had low methodological quality (87). A cautious conclusion was that *active* interventions might have a tendency to be more effective. Despite the lack of scientific evidence, clinical practise guidelines for the physiotherapy have been published, but these have yet to be validated (211).

Some authors consider the findings of the Australian group promising (6, 150, 212). This group reports in their controlled trials of nerve blocks that 60% of the studied subjects with WAD had cervical zygapophyseal-joint pain from C2 to C3 or below (35). They showed that in subjects with WAD with chronic cervical zygapophyseal-joint pain percutaneous radio-frequency neurotomy with multiple lesions of target nerves could provide lasting relief (213). These findings have not yet been confirmed in other studies and a recent systematic review of radio frequency denervation for neck and back pain concluded that there is limited evidence for short-term relief for chronic neck pain of zygapophyseal-joint origin and for chronic cervicobrachial pain and points out the need for further high-quality randomized control trials (214).

Decompression of the carpal tunnel in 38 selected patients

with 'atypical' neuropathic pain arising from the median nerve with normal electromyographic and nerve-conduction reduced pain and symptoms significantly compared with conservative treatment (163).

What is known about rehabilitation in chronic WAD?

The QTF group concludes that most treatments in the chronic state of WAD need a multidisciplinary approach and states that this approach should start during the sub-acute phase within 3 months of injury. Similar approaches have been suggested by other authors (61, 195, 215, 216). The neuromatrix model also opens up so that multiple form treatments have the ability to change the inputs and influences on the neuromatrix (217). Typically, in rehabilitation practise it is common that the subject with pain is assessed multivariately including somatic, psychological, societal and other aspects when planning for treatment or rehabilitation. Hence, a broad screening technique with respect to different categories (impairment, disability and global level) as well as coping strategies are considered as important. These clinical implicit multivariate "models" have been expressed scientifically as biopsychosocial models of pain and/or disability. The biopsychosocial model appears to be superior over the biomedical model when predicting disability (218). Rehabilitation can be viewed as a process of enhancing an individual's ability to reach or regain an optimal desired quality of life and perception of health consistent with his or her impairments and disabilities. Patient autonomy should be supported and encouraged and the patient should be encouraged to be an active team member in his recovery (219). Thus rehabilitation in chronic pain patients can be viewed as a co-ordinated multi-modal process.

Only a few studies (inclusion and exclusion criteria differed between the studies) have investigated the effects of multi-modal rehabilitation programs for patients with chronic WAD (133, 220–222). One as early as 3 months, 1 < 6 months, 1 over 2 years, and 19 months after the injury (133, 220, 221, 223). Results varied, but a trend towards better results (back to work) for earlier interventions can be noted (220, 223), which can indicate that an early intervention also with respect to multi-modal rehabilitation is needed. Individuals with low life satisfaction and few coping resources predicted a poor prognosis (221). A multi-modal program with postural training, manual technique, and psychological support ($n = 30$), had significantly better results than a control program with only physical agents ($n = 30$) in patients recruited 2 months after neck trauma (a randomized controlled single-blind prospective study) (220). Heikkilä and colleagues (221) reported that patients with chronic WAD ($n = 40$) showed a major increase in coping resources following a multi-modal rehabilitation program and "normalized" their coping resources profile compared with subjects without neck trauma. A multi-modal treatment program with cognitive behavioural approach for 26 chronic WAD patients (grade I–II) (without control group) was associated with a complete return-to-work rate of 65% combined with partial return to work rate of 92% (223). Sterner et al. (133) followed 90 chronic

WAD patients (without control group) that participated in a multidisciplinary rehabilitation program. Markers for psychosocial stress were high in the group. This study had no exclusion criteria concerning job situation or the severity of injury as the other studies and few patients declined participation after an interdisciplinary assessment and few patients were not available at follow-up (133). The pain intensities in the neck and upper back were significantly decreased at 6 months follow-up, but for most of the functional and psychological markers, no significant changes were found (133). However, retrospective evaluation at follow-up indicated increased ability to cope with and control pain and, to some extent, psychological aspects. For many of the patients, acceptance/adjustment started at the end of the program, and one can assume that this process needs time.

As there are very few studies on WAD, we need to look into rehabilitation of the ordinary chronic pain patients (a very non-specific definition). Multidisciplinary rehabilitation programs are superior to single discipline approaches or no rehabilitation (224). Cognitive-behavioural treatments are associated with significant effect sizes in all domains of measurement when compared with other active treatments (225), but it is still unknown what types of patients benefit most from what type of behavioural treatment (226). Although most of the referred studies do not focus on chronic neck pain (especially WAD), one can assume that some of the recommendations also relate to WAD. Pain and early cognitive-behaviour group intervention can lower the risk of a long-term disability developing and can be used in primary care (227).

Is there a need for chronic WAD rehabilitation?

Is there a need for special rehabilitation program for patients with chronic WAD or can these patients participate in general multi-modal rehabilitation programs? Two recent studies (209, 210) on chronic WAD, although with different instruments, conclude the following: (i) the situation with both pain and neuropsychological/cognitive symptoms will indicate a worse situation both at disability and global levels (life satisfaction) than if “only” pain is present; (ii) the amount and seriousness of perceived problems contribute to psychological distress; (iii) stress might be even higher in those with low education. In an analysis of 104 patients with chronic WAD, results showed that patients with chronic WAD differ from other comparable groups in “quality-of-life related indicators” such as a worse psychosocial situation (205). However, this has only been sparsely investigated in other studies. On the other hand, the study found no major difference between WAD and other chronic pain condition concerning disability, anxiety, depression, or self-efficacy. Söderlund (228) suggests that “early identification and modification of self-efficacy and coping are crucial for successful rehabilitation”.

CONCLUSION

Because of a lack of randomized, controlled and prospective studies, there is a lack of evidence-based guidelines for WAD.

Therefore, we must adopt strategies that rely on the evidence guidelines on chronic spinal pain and to some extent on results from single studies and our own experience.

The most common acute symptoms are pain in the neck and head and stiffness followed by interscapular pain, paresthesias in arms and hands, dizziness, temporomandibular symptoms, visual and auditory symptoms, cognitive problems and emotional/psychological disturbances. Acute pain will result in both psychological and physiological responses. Psychological concerns and other experiences due to events in the impact situation influence acute pain. Most subjects with acute WAD recover within weeks or a few months. As with other pain conditions, some patients will develop chronic conditions. The risk factors for chronic development and maintenance are insufficiently known, but a complex pattern of factors is expected. Different studies indicate that both acute and chronic WAD are syndromes with different subgroups. Studies that investigate the degree of plasticity and reversibility in the pain transmission system are needed in the future to develop specific treatment and rehabilitation.

We propose that rehabilitation and treatment effort for prolonged disability, whatever the cause, after whiplash injury should encourage patients to adopt an active, positive, and realistic attitude and strategies at all stages of recovery after an injury. Most injuries with increasing pain during or after physical activity should not be equated with a worsening of the injury. In most cases, we should avoid extensive investigations because these interfere with the rehabilitation process and promote ideas that something serious has occurred. Adequate examination and assessment of all circumstances such as fear and avoidance, loss of control, anxiety signs of post-traumatic stress, high intensity pain, bio-mechanical and psychosocial factors at work (studies), and social support (positive or negative) are important. Advice, proper medication and information to the patients about the mechanisms of physical and psychological symptoms are all important aspects of recovery for patients. For all individuals with symptoms at 3 months and major problems in participation in different activities to the extent they desire, there is a need for multidisciplinary evaluation. In addition, rehabilitation focusing on cognitive-behavioural changes might be of value.

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CONTINUING MEDICAL EDUCATION IN
REHABILITATION MEDICINE AND EXERCISE
FOR DOCTOR'S SPECIALIST TRAINING

CME – questions

1. Incidence – recovery: select the correct statement.
 - a) The incidence of whiplash trauma can not be reliably estimated
 - b) The annual incidence of whiplash injury varies in different studies between 0.1/1000 and 64/1000
 - c) 50–60% of whiplash traumas lead to a whiplash injury
 - d) Reported recovery in prospective studies (no pain) varies between 40–90%
2. Mechanism of injury: select the correct statement.
 - a) Hyperextension and hyperflexion of the neck are the pure mechanisms for injury
 - b) Hyperextension and late concentric muscle contraction can cause muscle injury
 - c) Retraction of the spine and/or eccentric muscle contractions are possible sources of an injury
 - d) Increased pressure of cervical spinal fluid causes injury in humans
3. Risk factor for whiplash injury (acute WAD): identify the incorrect statement.
 - a) Crash-related factors are significantly more important than individual risk factors
 - b) Crash-related factors such as a smaller car, having a tow-bar increase the risk for an injury
 - c) Change of velocity, magnitude of the peak acceleration (G), and $G > 5$ probably increase the risk
 - d) Car design and construction affect the risk of acute and chronic WAD
4. Signs of an injury: select the correct statement.
 - a) A negative X-ray excludes any injury
 - b) Tingling and numbing in the arm always indicate an evaluation with MRT
 - c) X-ray with flexion and extension projections can in a few cases show instability
 - d) Levels of CK and pro-inflammatory factors (TNF-alpha) can be used to exclude or verify an injury in acute WAD
 - e) Cognitive problems often indicate examination by use of neuro-imaging techniques
5. Risk factors for chronic WAD: identify the incorrect statement.
 - a) Gender and age are factors associated with outcome
 - b) Decreased range of movement (ROM) near to the accident increase the risk for chronic WAD
 - c) Neurological symptoms and signs in the acute stage can indicate a worse situation
 - d) Models of prediction should not include high emotional response to the accident or other psychological reactions
6. Clinical classification of WAD – identify the correct statement.
 - a) Spitzer et al. (Quebec Task Force; QTF) proposed a classification consisting of 4 grades. Many studies have used the classification
 - b) The classification of Spitzer et al. (QTF) is better than other classifications
 - c) WAD Grades 3 and 4 (according to QTF) are most frequent in the acute and chronic phases
 - d) Most people will develop signs of an injury after 48 hours
 - e) WAD grade 1 is in several studies significantly associated with a poor outcome
7. Chronic WAD – select the correct statement.
 - a) Cognitive problems are in most cases caused by subtle minor brain injuries
 - b) Up to 50% report concentration and memory problems
 - c) Radiating pain and sensory disturbance in neck and arm always motivates a MRT of the cervical spine
 - d) Visual problems, dizziness and tinnitus are most probably the result of a brainstem lesion caused by the accident
8. Treatment of acute WAD – select the correct statement.
 - a) In the acute phase a few days rest is the best recommendation
 - b) Cooling of neck muscles and acupuncture are evidence-based ways of treating acute WAD
 - c) Information, medication, active treatment and a soft collar might be the best
 - d) Active treatment seems to have some evidence to effect pain
9. Pain mechanisms – identify the false statement.
 - a) Research so far indicates heterogeneity of WAD but treatment strategies according to subgroups have not been investigated
 - b) Chronic pain processing in WAD and other pain condition are represented in a few distinct areas of the brain
 - c) Heterosensitization, central sensitization, individual genetic disposition and psychological response to pain/coping are all factors perpetuating neuropathic pain, but not nociceptive pain
 - d) Modulation of central pathways has been found in WAD

Correct answers: 1a, 2c, 3a, 4c, 5d, 6a, 7b, 8d, 9c