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## REVIEW

# Whiplash injury. A clinical review with emphasis on neurological aspects

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Although whiplash injury was mentioned as early as the 1920s<sup>1</sup> and the first report appeared in the 1950s<sup>2</sup> it did not start to receive special interest until 20 to 30 years ago. A number of possible explanations can be given for the apparent increase in prevalence of whiplash injuries, such as the increase in the absolute number of cars and car densities in most countries with the associated increase in the prevalence of car accidents. In addition, injuries from car accidents have increasingly led (and are leading) to litigation in Western countries.

The most common cause of whiplash injury is a rear-end car collision, but other types of collision, such as frontal or lateral, can also cause it.

Although there is still some controversy about the definition, whiplash injury can be defined as a non-contact acceleration–deceleration injury to the head and neck complex. The essential biomechanical feature of the mechanism is that no direct force is imparted to the head by the car and that the head undergoes acceleration forces that bend the neck. This definition is fairly similar to that of the Quebec Task Force on Whiplash Associated Disorder:<sup>3</sup> ‘Whiplash is an acceleration–deceleration mechanism of energy transfer to the neck. It may result from rear end or side-impact motor vehicle collisions, but can also occur during diving or other mishaps. The impact may result in bony or soft-tissue injuries (whiplash injury), which in turn may lead to a variety of clinical manifestations (Whiplash-Associated Disorders)’.

In a rear-end collision, the head tends to lag behind because of its inertia, while the struck car accelerates forwards; the head thus moves towards the collision partner. The trunk is generally held in place by the back of the seat but the head is accelerated backwards with reference to the car, particularly if the collision comes unexpectedly, when the subject is (still) relaxed. The head is ‘hypertranslated’ in a horizontal (back-

ward) direction<sup>4,5</sup> and then the neck (the craniovertebral part) is hyperextended if the headrest, as unfortunately is often the case, has not been carefully adjusted. In most cars the seat is inclined slightly, or substantially, backwards and the head of the occupant is leaning forwards at some distance in front of the headrest. In such a condition, the headrest does not prevent whiplash motion from occurring. The headrest is only effective if it makes contact with the head before the impact, or if it makes contact with the head at the same moment as the back of the seat makes contact with the trunk. Proper adjustment of headrests can reduce the prevalence of neck pain in rear-end collisions by some 25%.<sup>6,7</sup> Nygren *et al.*<sup>8</sup> demonstrated that the relative frequency of neck injury in rear-end collisions increased with the vertical distance between the head and the headrest. A rotated or inclined head position at the moment of impact has been found to be a primary feature related to the persistence and severity of symptoms,<sup>9,10</sup> as well as to signs of musculoligamental cervical strain and neural, particularly radicular, damage.<sup>11</sup> Unprepared car occupants had a higher frequency of multiple symptoms.

In some instances, whiplash injury seems to be enhanced by using seat belts.<sup>12–15</sup> In Germany, a marked increase in cervical spine injuries has been noted since a law concerning seat belts was implemented in 1984.<sup>13,15</sup> Cushioning of the impact by an airbag may reduce the risk in such cases.

## The whiplash syndrome

Sudden quick movements that occur between the head and neck or between the head and neck complex and the trunk may cause damage to the ligaments, joints, bones and muscles in the cervical region. In addition, the damage may involve the blood vessels, nerve roots or peripheral nerves, the cervical part of the sympathetic trunk, the oesophagus, trachea, mandibular joints, the medulla oblongata and brain.<sup>16</sup> Whiplash is a biomechanical event that can occur during, for example, a rear-end car collision; if, after the accident, the victim develops signs or symptoms, it can be presumed that he or she has the

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whiplash syndrome. A multitude of signs and symptoms are associated with this syndrome, which often constitute a typical pattern. It should be emphasized that a clear correlation between the (apparent) severity of the injury and that of the symptoms may be lacking: even a minor injury can cause severe and persistent symptoms. Classically, a patient with the whiplash syndrome initially has no or only minor symptoms and is doing well. About one-third of the patients who develop symptoms do so within 1 h of the event and the others within a few days. The typical pattern of symptoms comprises: (1) neck pain, headaches (mostly occipital headaches), radiating pain in one or both arms and pain between the shoulder blades; (2) paraesthesia, sensory disturbances (localized to the upper cervical dermatomes), or transient muscular weakness; (3) dizziness or giddiness, imbalance, tinnitus and vision impairment. The majority of patients show spontaneous recovery or their condition improves after a few months of symptomatic treatment. In some patients, the symptoms may persist and then comprise: (4) concentration, attention or memory impairment; (5) hypersensitivity for sound and light; (6) behavioural disturbances, including irritability; (7) sleep disorders, anxiety and depression. The last symptoms (numbers 4–7) pertain to the so-called 'late' whiplash syndrome,<sup>17–20</sup> which is a matter of controversy in medicine and the law, especially because of the multiplicity of symptoms and the poverty of signs:<sup>21</sup> see, for example, Malleson<sup>22</sup> and the Editorial by Pearce,<sup>23</sup> as well as the subsequent correspondence.<sup>24–27</sup> Recently, Schrader *et al.*<sup>28</sup> did not find any disabilities or persistent symptoms as a result of rear-end car collisions in a retrospective controlled cohort study in Lithuania, a country where financial gain was an unlikely influence; they suggest that the expectation of (permanent) disability and aggravation of pre-existing symptoms might be more important determinants.

## Dizziness

From a neuro-otological perspective, the symptoms of dizziness and imbalance are of particular importance; these are among the most consistent, frequent and incapacitating features in whiplash injury.<sup>1,29–34</sup> Some patients have vertigo, which is sometimes accompanied by nausea, but seldom by vomiting. It has been known for several decades that disorders of the neck can cause dizziness; according to Pfaltz and Richter,<sup>35</sup> so-called 'cervicogenic dizziness' may occur after traumatic damage to the cervical vertebrae, which therefore might also include whiplash injury. 'Cervical vertigo', however, is a very controversial subject.<sup>36–38</sup> Various hypotheses have been proposed to explain dizziness following neck injury. Neurovascular hypotheses featuring compression or stenosis of one or both of the vertebral arteries and stimulation of the sympathetic nervous system<sup>39</sup> have been abandoned, whereas neuromuscular hypotheses on disturbed (lumbar and)

cervical proprioception<sup>39</sup> are still under consideration. Dysfunction of the central nervous system,<sup>30,32,34,40</sup> peripheral labyrinthine damage associated with benign paroxysmal positional vertigo<sup>41,42</sup> or vestibular hyperreactivity and hyperventilation<sup>43,44</sup> are presently considered to be possible causes of the dizziness.

## Neuro-otological examination

Neuro-otological examination can be performed in an attempt to substantiate a possible cause underlying the symptoms of dizziness and imbalance in patients after whiplash injury or to find out whether treatment is possible. The examination comprises assessment of the peripheral labyrinth and the vestibulo-ocular reflex (VOR) system, which includes the (vestibulo)cerebellum and (ocular motor) functions localized in the pons and the brain stem. The tests include the assessment of visually guided eye movements, such as smooth pursuit (SP), optokinetic nystagmus (OKN) and saccades, as well as vestibular tests, such as the caloric test and rotatory chair stimulation using, for example, the velocity step test. Eye movement responses are (usually) evaluated by means of electro-oculography or electro-nystagmography.<sup>45</sup>

Neuro-otological examination should take place as soon as possible after the event in order to establish a causal relationship and, possibly, in anticipation of any medical reviews by outside parties or insurance claims. The presence of vestibular symptoms is another obvious indication for such an examination. The absence of such symptoms does not mean that this examination can be omitted, because vestibular signs may be present without causing any symptoms; the latter may develop later. Ideally, if any meaningful neuro-otological abnormalities are found, it should be established whether they occurred before the event or whether they can be attributed to it.

Previous reports about whiplash have hardly indicated any substantial findings that can be related to peripheral dysfunction. Ettlin *et al.*<sup>46</sup> described 'peripheral' vestibular deficits (either canal paresis or VOR asymmetry) in two out of 18 patients evaluated within 2 weeks of the injury. Benign paroxysmal positional vertigo (BPPV) and nystagmus were reported by Grimm *et al.*<sup>41</sup> and by Chester,<sup>42</sup> who related these features to perilymphatic leakage secondary to the injury. BPPV was a major finding in the series of patients described by Davies & Luxon,<sup>47</sup> but this series included patients with head injury with amnesia. Other reports on patients with whiplash injuries who underwent vestibulo-ocular examination with electronystagmography mentioned finding positional nystagmus,<sup>29,32,34,42,48,49</sup> unilateral caloric hypoflexia<sup>29,32,43,44,48,50</sup> and directional preponderance of nystagmus elicited by caloric stimulation<sup>48,50</sup> or rotatory stimulation.<sup>42,50,51</sup> Unfortunately, most of these reports did not refer to any suitable control groups or to the use of

normal values. Several reports have appeared about 'cervical nystagmus' and abnormal ocular motor and VOR findings that could be attributed to whiplash injuries (see below).

### **'Cervical nystagmus' and the cervico-ocular reflex (COR)**

One of the most popular suggestions in relation to whiplash is that it may lead to disturbances of cervical proprioception, caused by the tension-extension forces that occur during rear-end collisions. One of the signs of such a disturbance is assumed to be 'cervical nystagmus', i.e. nystagmus that arises from neck rotation without labyrinthine stimulation.<sup>29,32</sup> It should be emphasized that in our opinion the label 'cervical nystagmus' does not apply to the spontaneous/positional nystagmus that occurs in certain static head positions,<sup>34,52</sup> because static labyrinthine stimulation is or can be involved. In most instances, static neck stimulation consists of some 60° rotation of the trunk to the left or right under the hand-fixed head for about 2 min. One of the present authors applied this type of stimulation to 160 consecutive patients with a whiplash injury and did not observe any tangible eye movement associated with it (A.J.E.M.F., 1994, unpublished observations). In a previous study, we could not find any nystagmus especially pertaining to static neck rotation (ramp/plateau COR test) at 30° amplitude,<sup>43,44</sup> while Kingma *et al.*<sup>53</sup> using comparable tests and equipment, reported similar findings at 60° amplitude in women. Our conclusion was that the ramp/plateau COR test was not productive, because the static neck torsion did not produce any relevant response, whereas the nystagmic responses to the 5°/s ramps were unsuitable for distinguishing between normal and 'abnormally high' COR gains; this could be done in a reliable way with the sinusoidal COR test (see below).

Holtmann *et al.*<sup>54</sup> failed to find a causal relationship between nystagmus and hypertonicity of the neck muscles. These authors stated that 'cervical nystagmus' is not typical of vertigo associated with disturbances of cervical proprioception.

The COR can be elicited by dynamic sinusoidal stimulation. Normal subjects have a low COR gain,<sup>55</sup> while their phase behaviour indicates a low-pass system.<sup>56</sup> However, when a normal subject is instructed to 'follow' visually (i.e. in the dark with the eyes open) some part of his moving body, the (position) gain of the COR increases drastically by invoking (anticompensatory, 'orientating') saccades.<sup>56</sup> This type of gain enhancement may also occur in patients after whiplash and be misinterpreted as 'cervical nystagmus'. Oosterveld *et al.*<sup>32</sup> reported 'cervical nystagmus' in 79% of the patients (with eyes closed). Unfortunately, the precise stimulus conditions were not specified and no normal values or statistical analysis were reported. Normal subjects can show a wide range of COR response levels. Our own measurements<sup>56</sup> indicated that normal gains were in the range of between 0 and 0.6. It is

difficult, therefore, if not impossible, to distinguish 'positive' and 'negative' responses in a valid way without statistical testing. A stimulus amplitude of 60° was used by Oosterveld *et al.*<sup>32</sup> although they reported that they encountered 'restriction of the rotation ability of the head in one or two directions' in 28% of their patients (which motivated our choice of an amplitude of only 30°). It was stressed by both Hülse<sup>57</sup> and Reker<sup>58</sup> that in the case of (bi)laterality of the 'cervical nystagmus', the possibility of arousal of any 'latent' spontaneous nystagmus should be considered. Interestingly, spontaneous (or positional) nystagmus was shown by the majority of patients described by Oosterveld *et al.*<sup>32</sup> and Kortschot *et al.*<sup>34</sup> (see above and below) and many of their patients (also) showed gaze-evoked nystagmus (see below). However, these authors did not describe how they distinguished nystagmus observed during neck stimulation from pre-existing nystagmus. Compere<sup>29</sup> also reported a high prevalence of spontaneous nystagmus (see below) in his patients and in most of them 'the nystagmus was precipitated or definitely aggravated by rotation and extension of the head and neck'.

### **Ocular motor system**

Hildingsson *et al.*<sup>40</sup> and Burke *et al.*<sup>59</sup> reported that the most common ocular motor abnormality following whiplash was disturbance of convergence and/or accommodation. Ocular motor paresis following whiplash injury was reported by Duke-Elder and McFaul<sup>60</sup> and Burke *et al.*<sup>59</sup> It concerned temporary abducens and superior oblique paresis in only a few patients. These findings of disturbances of convergence and ocular motor paresis are fairly similar to those reported after (severe) head trauma.<sup>61</sup> Chester<sup>42</sup> found ocular motor abnormalities in only two out of the 29 patients tested; one patient showed fixation instability, while the other showed saccadic SP responses. Ettlin *et al.*<sup>46</sup> described the presence of 'central' vestibular deficits within the first 2 weeks in four out of their 18 patients; these abnormalities included defective SP/OKN in at least some of them. Burke *et al.*<sup>59</sup> reported that five of their 39 patients developed an SP deficit and two showed hypometric horizontal saccades which resolved within 1-3 months in those followed up. In contrast with these findings, Hildingsson *et al.*<sup>40</sup> found a significant reduction in (mean) saccade peak velocity and SP gain in a subgroup of 20 patients (who all had severe symptoms of neck pain, neck stiffness and headaches); such abnormalities were lacking in a group of asymptomatic patients ( $n = 19$ , interval  $> 6$  months.) Only eight and 10 patients respectively in these two groups had been involved in rear-end car collisions. Oosterveld *et al.*<sup>32</sup> and Kortschot *et al.*<sup>34</sup> reported unilateral gaze-evoked nystagmus in 6-10%, bilateral gaze-evoked nystagmus in 18-26% and saccadic SP responses in 26-43% of their symptomatic patients (after unspecified trauma). Neither of the latter reports mentioned the use of any drugs by the

symptomatic patients, although it is not unusual for such patients to receive medication that may influence oculomotor functions or cause (gaze-evoked) nystagmus.<sup>62</sup> Interestingly, one of the co-authors of these reports<sup>32,34</sup> recently examined a group of symptomatic patients, and after excluding the patients who used antidepressants, muscle relaxants or vestibular depressants,<sup>52</sup> she found fewer abnormalities than in the previous study. Spontaneous nystagmus and 'cervical' nystagmus were both found in only 2.5% and a decreased SP gain was detected in only 7.5% of patients. Hinoki<sup>30</sup> mentioned that 'abnormal optokinetic nystagmus is often observed' in symptomatic patients. The abnormality was described as an 'irregularity', so we assume that the responses showed episodes with a very low gain. Ushio *et al.*<sup>63</sup> as cited by Hinoki,<sup>30</sup> reported inverted OKN responses after whiplash injury, which is a peculiar finding unless it was due to pre-existing congenital nystagmus or the superposition of other spontaneous or gaze-evoked nystagmus. In patients with head injuries, gaze-evoked nystagmus was reported in less than 1%<sup>64</sup> and in about 2% after contusion;<sup>65</sup> impaired SP/OKN responses were found in less than 1%.<sup>65</sup> Meran *et al.*<sup>64</sup> mentioned impaired SP/OPN responses in 10%, especially within the first 6 months. Davies and Luxon<sup>47</sup> found an SP deficit in 6% of their patients. Similar abnormalities were reported in 35–43% of patients soon after the trauma, which decreased to 8–12% later on.<sup>66,67</sup> Fischer *et al.*<sup>43,44</sup> noted the absence of gaze-evoked nystagmus following whiplash injury. Saccades and SP/OKN responses were normal in all of their patients. The only possibly central oculomotor disturbance encountered in their study was the incidental occurrence of double saccadic pulses (back-to-back saccades) in one patient. Hotson<sup>68</sup> reported that this phenomenon also occurs in normal subjects.

Repeat ocular motor examinations (SP, saccades) were performed in a few studies,<sup>40,52</sup> which revealed a slight tendency towards decreased ocular motor system performance, which proved to be correlated with similar changes in neuropsychological test scores.<sup>52</sup>

### Vestibulo-ocular reflex (VOR)

Spontaneous nystagmus was reported in symptomatic patients by Oosterveld *et al.*<sup>53</sup> and Kortschot *et al.*<sup>54</sup> in 53–63% of the patients and by Compere<sup>29</sup> and Toglia<sup>69</sup> in 29–30%, whereas Pang<sup>48</sup> reported only one patient (5%). Zenner<sup>51</sup> mentioned the clinical observation of provoked(?) or gaze-evoked nystagmus in only 5% of his 100 patients. Fischer *et al.*<sup>43,44</sup> found no spontaneous nystagmus.

Abnormal caloric responses were generally lacking in our previous study, i.e. substantial unilateral caloric hyporeflexia (canal paresis) was found in 4% and bilateral caloric weakness also in 4% of the patients.<sup>43,44</sup> Similar low percentages have been reported by others.<sup>29,32,49</sup> Other studies, however, mentioned canal dysfunction in 33 to 40%.<sup>42,48,69</sup> In patients with

closed head injuries, canal paresis has been reported in a broad range of percentages (10–60%) unilaterally<sup>50,65–67,70,71</sup> and in 17% bilaterally.<sup>65</sup> A reduction to lower percentages (3–10%) of unilateral canal paresis has been noted in patients much later after the head injury.<sup>47,66,67,70</sup> Directional preponderance of rotatory responses was generally lacking in our previous study.<sup>43,44</sup> Toglia<sup>69</sup> reported a significant directional preponderance of rotatory responses (i.e. 'abnormal responses', 'the abnormality was basically preponderance...') in 51%, whereas Chester<sup>42</sup> mentioned 'abnormalities of the vestibulo-ocular reflex' in one out of nine patients tested with sinusoidal rotation.

Fischer *et al.*<sup>43,44</sup> found vestibular hyper-reactivity, which has not been reported previously in any study on whiplash, in a significant proportion of their patients. Compere<sup>29</sup> specifically mentioned that 'hyperactive caloric responses' were lacking. In patients with head trauma, Montandon and Dittrich<sup>72</sup> found hyper-reactivity (their type VI) of rotatory responses in almost 12%. In a group of patients with mild head injuries, 48% of whom had suffered a traffic accident, Tuohimaa<sup>66</sup> found a significant increase in the mean nystagmus slow phase velocity, but not in the nystagmus frequency, during culmination of the post-caloric response, measured within the first 4 days after the accident. This had normalized after 6 months. Vestibular step testing showed a significant increase in initial velocity and cumulative eye displacement only at the first examination. Kirtane *et al.*<sup>73</sup> found a significant increase in the mean (post-caloric) nystagmus frequency in patients with head injuries (caused in a proportion of them by a car accident). A very special type of vestibular disinhibition, or rather VOR instability, may be represented by periodic alternating nystagmus.<sup>45</sup> We know of only one reported case following whiplash.<sup>74</sup>

Vestibular hyper-reactivity has been reported in multiple sclerosis,<sup>75</sup> vestibulocerebellar dysfunction<sup>76–82</sup> which may be associated with kernicterus,<sup>83</sup> in (idiopathic) spasmodic torticollis<sup>84</sup> and in the hyperventilation syndrome.<sup>85</sup> The finding of a high VOR gain in spasmodic torticollis may be relevant, because we have previously proposed that this may represent (plastic) VOR enhancement to compensate for apparent limitation of active head movements.<sup>84</sup> We made a similar observation and suggestion in relation to patients with facioscapulohumeral dystrophy, some of whom showed dystrophic changes in the muscles of the neck and shoulder on CT scans.<sup>86</sup> Similar VOR enhancement in patients after whiplash has been previously suggested by us<sup>43,44</sup> because the patients generally had neck pain and neck stiffness and often showed limitation of neck rotation. A significant number of our whiplash patients also demonstrated the hyperventilation syndrome.<sup>43,44</sup> In our previous work on the hyperventilation syndrome<sup>85,87,88</sup> we not only found close linkage between the features of this condition and vestibular hyper-reactivity but also became aware of the 'mental state' in patients with the hyperventilation syndrome. Vestibular hyper-reactivity may have

been caused by a lesion in, or rather dysfunction of, the vestibular commissural system.<sup>75</sup> A lesion in the brain stem may involve both this system and the respiratory centre with its central connections. Both this centre and the VOR are under the influence of the reticular formation (reticular activating system), which may be involved in emotional (stress) disorders and be under the influence of the 'mental state', which has an important bearing on both breathing behaviour and the VOR. Noradrenalin excretion is significantly increased in patients with hyperventilation;<sup>89</sup> this would fit into a stress model in which both vestibular hyper-reactivity and hyperventilation can occur concomitantly. The latter, apparently, is also the case in patients after whiplash.<sup>43,44</sup> It is generally recognized that patients after whiplash may exhibit abnormal psychological distress. Their excess psychological symptomatology, which generally includes anxiety and depression, may be secondary to the injury<sup>90</sup> with its 'profound physical, social, legal and vocational effects'<sup>33</sup> (see also below). Jenzer<sup>20</sup> demonstrated a remarkable similarity in neuropsychological symptomatology between the late whiplash syndrome and other non-traumatic conditions, not only chronic disturbances of cervical origin, but also the chronic fatigue syndrome, the fibromyalgia syndrome and chronic daily headaches. Anxiety is clearly linked with VOR enhancement. This was confirmed by Yardley *et al.*<sup>91</sup> who examined subjects suffering from panic attacks or agoraphobia; fear ratings were significantly correlated with vestibular response levels. Anxiety and depression, as well as sleep disorders, form part of the 'late' (chronic) whiplash syndrome. There are indications that this syndrome includes the symptom of 'dizziness' and the features of vestibular hyper-reactivity. In our study group,<sup>43,44</sup> the presence of dizziness correlated significantly with a long interval (>6 months) between the injury and presentation. As discussed in previous reports,<sup>43,44</sup> we reviewed the clinical records of a group of 35 consecutive patients who were referred for routine examination after suffering a whiplash injury. The women in this group demonstrated a significant link between the features of vestibular hyper-reactivity and a long time interval (>1 year) between the injury and examination. These observations suggest that we experienced the selective referral of patients (women) who had persistent complaints (about dizziness) or had complaints only in the long-term, in both instances presumably on the basis of their vestibular hyper-reactivity.

### Additional examinations

Roos<sup>92</sup> found disturbances in the subjective horizontal during lateroflexion of the head following whiplash injury ('neck trauma') in 18% of patients. 'Functional' stabilometry, i.e. during neck extension, with visual stimulation alone revealed increased anteroposterior and lateral body sway, whereas testing in a dynamically tilted room disclosed significantly

increased lateral sway in 67–75%, depending on the stimulus frequency. These results indicate increased reliance upon visual information. The latter notion also applies to the findings reported by Rubin *et al.*<sup>93</sup> in relation to anteroposterior sway measured with ('static') stabilometry. Roos<sup>92</sup> re-examined his patients 'after treatment' and found significantly increased lateral sway in the tilting room in only 17–25%, which indicated that, with improvement, the patients showed decreased reliance upon visual information and thus improved inherent postural stability.

Brain stem auditory evoked potential (BAEP) findings were normal in patients in some studies.<sup>46,94</sup> In contrast, Serra *et al.*<sup>95</sup> reported increased I–III and I–V interpeak intervals on BAEPs unilaterally in 45 out of their 120 patients, bilaterally in 32 soon after the injury, and in 31 and 16 after 6 months, respectively.

Several EEG studies reported abnormalities in 30–50% of patients (review<sup>46</sup>), although others found minor abnormalities in less than 10%.<sup>94,96</sup>

It can be questioned whether conventional radiography of the neck can demonstrate anything other than reduced intervertebral spaces, vertebral problems or spondylosis. Using functional (flexion–extension) radiography of the cervical spine, a trend towards hypermobility was found<sup>97</sup> at the upper and middle cervical level, as well as soft-tissue ruptures and segmental instability<sup>98</sup> and abnormal (localized) kinking and fanning.<sup>99</sup> Functional CT scans demonstrated hypermobile (left/right) rotation at the upper cervical spine level.<sup>100</sup> MRI findings were normal in some studies.<sup>46,94,101</sup> Other studies<sup>98,102,103</sup> have reported that discoligamentous injuries, especially disc protrusions amenable to surgery, could be identified in patients with immediate and persistent severe radiating pain.<sup>98,102</sup> Although Pettersson *et al.*<sup>104</sup> found MRI abnormalities (generally disc lesions) in two-thirds of patients and fairly discrete neurological deficits (mostly sensibility disturbances) in the majority of patients in the acute stage, the correlation between the MRI findings and the clinical signs and symptoms was poor. Schnarkowski *et al.*<sup>105</sup> compared static and functional MRI and found ligamentous instability and disc protrusions only using functional MRI. These findings correlated with the neurological symptoms. Nägele *et al.*<sup>106</sup> also mentioned ligamentous lesions that were not seen with other methods. Peterson *et al.*<sup>107</sup> found that on MRI the spinal canal was significantly more narrow at the level of C2–C6 in symptomatic than in asymptomatic patients; a narrow sagittal diameter of the cervical spinal canal has been related with neurological deficits in degenerative conditions and following spinal fractures.<sup>108–110</sup>

Neuropsychological assessment of patients after a whiplash injury revealed significant impairment of attention, concentration, memory and other functions.<sup>46,111–114</sup> Patients who were anxious or had a long history of pain tended to show higher ratings of depression and anxiety and (cold-induced) pain.<sup>94,115</sup> Soon after the injury, about 20% suffered from an

acute stress syndrome characterized by anxiety or depression combined with horrifically intrusive memories of the accident.<sup>116</sup> The presence of this syndrome was significantly associated with neuroticism and being conscious after the accident. The patients with persistent symptoms showed great similarities to patients with chronic pain.<sup>115,117</sup> According to Radanov *et al.*,<sup>112</sup> the psychological problems of symptomatic patients can be attributed to difficulty in adjusting to somatic symptoms such as pain, and at least part of their cognitive impairment may be secondary to chronic pain. Similarly, Hollnagel & Nrrrelund<sup>118</sup> found that patients with chronic refractory headaches not associated with closed head injury or whiplash had many symptoms in common with post-whiplash syndrome patients (e.g. fatigue, poor concentration, memory and sleep disturbances, depression and dizziness).

## Prognosis

Evans<sup>7</sup> reported that most patients recovered fully within 3 months of the accident; however, after 2 years, persistent neck pain and headaches were respectively reported by more than 30% and 10% of the patients. There was clear correlation between the outcome and the severity of the initial symptoms, as was later confirmed by the Swiss studies.<sup>9,10</sup> Gargan and Bannister<sup>119</sup> reported that 93% (100% = 15) of the patients who were asymptomatic after 3 months were still symptom free after 2 years. Of the patients who were symptomatic after 3 months ( $n = 35$ ), 86% were still symptomatic after 2 years. The overall recovery rate after 1 year was 52% and after 2 years 38% had recovered. Also Mayou *et al.*<sup>116</sup> reported that the mental state at 3 months was highly predictive of the mental state at 1 year. Ettlin *et al.*<sup>46</sup> reported general improvement after 1 year, but the neuropsychological deficits, as well as anxiety and depression, persisted in some.<sup>116</sup> Persistent psychiatric sequelae of 'minor' head injuries (whiplash injury in particular) have previously been mentioned by Merskey and Woodforde.<sup>120</sup> Rimel *et al.*<sup>121</sup> stated that 'emotional stress caused by persistent symptoms' following minor head injury was a crucial factor in the long-term disability of patients. According to Radanov *et al.*<sup>111</sup> all neuropsychological dysfunction had (generally) improved to normal after 6 months in both the symptomatic and asymptomatic patients. However, the symptomatic group showed delayed recovery regarding complex attentional functioning. Patients with a history of a head trauma or pretraumatic headaches tended to be at risk for delayed recovery after whiplash.<sup>9,122</sup> Factors that have an adverse effect on the prognosis include objective neurological signs, older age, pre-existing degenerative spondylosis and pretraumatic headaches.<sup>111,123,124</sup> The timing of compensation in litigation procedures was not associated with the timing of improvement of the symptoms.<sup>7,124</sup> Contradicting Watkinson,<sup>125</sup> Parmar and Raymakers<sup>124</sup> found that whiplash did not accelerate the development of degenerative changes during long-term follow-up.

The type of therapy that should be given to patients after a whiplash injury in the acute and chronic phases is as unclear as all the other aspects of the injury. No studies have properly addressed the efficacy of cervical collars or rest. Electrotherapy, acupuncture, ultrasound and a number of other modalities have not proved to be effective.<sup>3</sup> According to Barnsley *et al.*<sup>33</sup> there are no efficacious treatments for a patient with chronic pain after whiplash. However, they have considered the possibility of investigating symptomatic patients with techniques such as cervical zygapophyseal joint blocks and, possibly, discography. If localized anaesthesia produces complete resolution of the pain,<sup>126</sup> then orthopaedic surgery might be indicated, because there is little chance of healing or spontaneous recovery. Jónsson *et al.*<sup>98</sup> reported successful discectomy and fusion in patients with severe radiating pain whose MRI showed large disc protrusions. Intra-articular betamethasone in the cervical zygapophyseal joints was not effective for chronic pain.<sup>127</sup> Subcutaneous sterile water injections to alleviate chronic neck and shoulder pain have been both recommended<sup>128</sup> and rejected.<sup>129,130</sup> McKinney<sup>131</sup> found that early mobilization and not wearing a neck collar reduced the number of patients with symptoms at 2 years. He also mentioned that only 10% of the patients who were still symptomatic at 6 months after injury recovered in the long term.

## Conclusions

A critical review of the literature on neuro-otological findings after whiplash injury showed that the methodology of most of the reported studies was far from perfect. The following comments apply fairly generally: (1) the type of trauma/injury was defined poorly (or not at all); (2) the type of accident (rear end = caudal, lateral or frontal collision) was not specified and relevant details about the use of headrests and seat belts, or about whether the head came into contact with any object or not, etc. were lacking; (3) the type and severity of the injury were not specified; (4) most of the study groups comprised patients with long-standing symptoms, but the causal relationship between the findings and the event is more difficult to establish at a later date; (5) symptomatic and asymptomatic patients were not distinguished; (6) no distinction was made between men and women, but the latter seem to be more vulnerable, more frequently affected and take longer to recover;<sup>6,7,18,19,131</sup> (7) age was not taken into account, but Gargan and Bannister<sup>119</sup> found that a more advanced age was significantly correlated with a poor outcome; (8) medication was not mentioned or specified, although patients with long-standing problems are especially likely to be using drugs such as antidepressants, tranquillizers or antivertiginous medication; (9) comparison with an adequate control group, or normal values, as well as statistical analyses were lacking.

As regards the neuro-otological findings in themselves, per-

ipheral labyrinthine function was generally intact. 'Cervical nystagmus' or abnormal cervico-ocular reflex findings did not apply. The same was generally valid for abnormal ocular motor findings, in as far as the methodology was satisfactory. VOR findings relating to rotatory responses have substantiated the existence of vestibular hyper-reactivity, i.e. VOR enhancement. VOR enhancement may represent plastic adaptation to compensate for any apparent limitation of active head movements in patients with chronic neck pain. Chronic pain may influence the 'mental state' of a patient in such a way that he or she develops vestibular hyper-reactivity with the associated symptom of dizziness. Additional support for the latter possibility is provided by the finding that these patients also develop the tendency to hyperventilate and show decreased performance in neuropsychological tests with increased cognitive impairment and high stress and anxiety scores. The tendency towards developing VOR enhancement and hyperventilation might demonstrate that there is a behavioural (patho)physiological component to the psychological problems of symptomatic patients, which can be attributed to the difficulty of adjusting to somatic symptoms, such as chronic pain.

## References

- 1 CROWE H.E. (1928) Injuries to the cervical spine. Paper presented at the meeting of the Western Orthopaedic Association, San Francisco
- 2 GAY J.R. & ABBOTT K.H. (1953) Common whiplash injuries of the neck. *J. Am. Med. Assoc.* **152**, 1698-1704
- 3 SPITZER W.O., SKOVRON M.L., SALMI L.R., CASSIDY J.D., DURANCEAU J., SUISSA S. & ZEISS E. (1995) Scientific monograph of the Quebec Task Force on Whiplash-Associated Disorders: redefining 'whiplash' and its management. *Spine* **20**, (Suppl. 8), 1S-73S
- 4 PENNING L. (1992) Acceleration injury of the cervical spine by hypertranslation of the head. Part II. Effect of hypertranslation of the head on cervical spine motion: discussion of literature data. *Eur. Spine J.* **1**, 13-19
- 5 WALZ F. (1994) Biomechanische Aspekte der HWS-Verletzungen. *Orthopäde* **23**, 262-267
- 6 KALLIANE C.J. (1982) *An Evaluation of Head Restraints. Federal Motor Vehicle Safety Standard 202*. US Department of Transportation, National Highway Traffic Safety Administration, Technical Report DOT HS-806-108, Washington, DC
- 7 EVANS R.W. (1992) Some observations on whiplash injuries. *Neurol. Clin.* **10**, 975-997
- 8 NYGREN A., GUSTAFSSON H. & TINGVALL C. (1985) Effects of different types of headrests in rear-end collisions. *Tenth International Technical Conference on Experimental Safety Vehicles, Oxford, England, 1-4 July 1985*. US Department of Transportation; National Highway Traffic Safety Administration, 85-90
- 9 RADANOV B.P., STURZENEGGER M. & DI STEFANO G. (1995) Long-term outcome after whiplash injury. A 2-year follow-up considering features of injury mechanism and somatic, radiologic, and psychosocial findings. *Medicine (Baltimore)* **74**, 281-297
- 10 STURZENEGGER M., RADANOV B.P. & DI STEFANO G. (1995) The effect of accident mechanisms and initial findings on the long-term course of whiplash injury. *J. Neurol.* **242**, 443-449
- 11 STURZENEGGER M., DI STEFANO G., RADANOV B.P. & SCHINDRIG A. (1994) Presenting symptoms and signs after whiplash injury: the influence of accident mechanisms. *Neurology* **44**, 688-693
- 12 DEANS G.T., MAGALLIARD J.N., KERR M. & RUTHERFORD W.H. (1987) Neck-sprain a major cause of disability following car accidents. *Injury* **18**, 10-12
- 13 THEEKE A., DEGREIF J., GEIST M., SCHILD H., STRUNK H. & SCHUNK K. (1993) Der Sicherheitsgurt: Auswirkungen auf das Verletzungsmuster von Autoinsassen. *Fortschr. Röntgenstr.* **159**, 278-283
- 14 IMMIGIA G. (1995) Whiplash injuries increase with seatbelt use. *Can. Fam. Physician* **41**, 203-204
- 15 MÜNKER H., LANGWIEDER K., CHEN E. & HELL W. (1995) Verletzungen der Halswirbelsäule bei Pkw-Unfällen. *Ver-sicherungsmedizin* **47**, 26-32
- 16 OMMAYA A.K., FAAS F. & YARNELL P.H. (1968) Whiplash injury and brain damage: an experimental study. *J. Am. Med. Assoc.* **204**, 285-289
- 17 BRAAF M.M. & ROSNER S. (1958) Whiplash injury of the neck: symptoms, diagnosis, treatment, and prognosis. *N.Y. State J. Med.* **58**, 1501-1507
- 18 BALLA J.I. (1980) The late whiplash syndrome. *Aust. N.Z. J. Surg.* **50**, 610-614
- 19 KRÄMER G. (1983) HWS-Schleudertraumen. *Med. Welt.* **34**, 1134-1140
- 20 JENZER G. (1995) Klinische Aspekte und neurologische Begutachtung beim Zustand nach Beschleunigungsmechanismus und der Halswirbelsäule. *Nervenarzt* **66**, 730-735
- 21 BRING G. & WESTMAN G. (1991) Chronic posttraumatic syndrome after whiplash injury. A pilot study of 22 patients. *Scand. J. Prim. Health Care* **9**, 135-141
- 22 MALLESON A. (1994) Chronic whiplash syndrome. Psychosocial epidemic. *Can. Fam. Physician* **40**, 1906-1909
- 23 PEARCE J.M.S. (1994) Polemics of chronic whiplash injury. *Neurology* **44**, 1993-1997
- 24 BOUNDS J.A. (1995) Chronic whiplash syndrome. *Neurology* **45**, 2117
- 25 EVANS R.W. (1995) Chronic whiplash syndrome. *Neurology* **45**, 2117
- 26 MERSKEY H. (1995) Chronic whiplash syndrome. *Neurology* **45**, 2116
- 27 PEARCE J.M.S. (1995) Chronic whiplash syndrome. *Neurology* **45**, 2117-2118
- 28 SCHIRADER H., OBELIENE D., BOVIM G., SURKIENE D., MICK-EVICIENE D. & SAND T. (1996) Natural evolution of late whiplash syndrome outside the mediolateral context. *Lancet* **347**, 1207-1211
- 29 COMPERE W.E. (1968) Electronystagmographic findings in patients with 'whiplash' injuries. *Laryngoscope* **78**, 1226-1233
- 30 HINOKI M. (1985) Vertigo due to whiplash injury: a neurological approach. *Acta Otol. (Stockh.) Suppl.* **419**, 9-29
- 31 OMMAYA A.K. (1988) Mechanisms and preventive management of head injuries: a paradigm for injury control. *Proceedings of the 32nd AAAM, Seattle, Washington*, pp. 360-391
- 32 OOSTERVELD W.J., KORTSCHOT H.W., KINGMA G.G., DE JONG H.A.A. & SAATCHI M.R. (1991) Electronystagmographic findings following cervical whiplash injuries. *Acta Otolaryngol. (Stockh.)* **111**, 201-205
- 33 BARNSELY L., LORD S. & BOGDUK N. (1994) Whiplash injury (Clinical review). *Pain* **58**, 283-307
- 34 KORTSCHOT H.W. & OOSTERVELD W.J. (1994) Otoneurologische Störungen nach HWS-Schleudertrauma. *Orthopäde* **23**, 275-277



- 35 PFALTZ C.R. & RICHTER H.R. (1958) Die cochleo-vestibuläre Symptomatologie des Cervicalsyndroms. *Arch. Ohren-Nasen-Heilk.* **172**, 519–534
- 36 DE JONG J.M.B.V. & BLES W. (1986) Cervical dizziness and ataxia. In *Disorders of Posture and Gait*, (Bles W. & Brandt T. eds) pp 185–206, Elsevier, Amsterdam
- 37 BRANDT T.H. (1991) *Vertigo. Its Multisensory Syndromes*. Springer Verlag, London
- 38 BROWN J.J. (1992) Cervical contribution to balance: cervical vertigo. In *The Head Neck Sensory Motor System* (Berthoz A., Graf W. & Vidal P.P. eds) pp. 644–647, Oxford University Press, New York
- 39 BOGDUK N. (1986) The anatomy and pathophysiology of whiplash (Review). *Clin. Biomech.* **1**, 92–101
- 40 HJEDINGSSON C., WENNGREN B.-L., BRING G. & TOOLANEN G. (1989) Oculomotor problems after cervical spine injury. *Acta Orthop. Scand.* **60**, 513–516
- 41 GRIMM R.J., HEMENWAY W.G., LEBRAY P.R. & BLACK F.O. (1989) The perilymph fistula syndrome defined in mild head trauma. *Acta Otolaryngol. (Stockh.) Suppl.* **464**, 1–40
- 42 CHESTER JR J.B. (1991) Whiplash, postural control, and the inner ear. *Spine* **16**, 716–720
- 43 FISCHER A.J.E.M., HUYGEN P.L.M., FOLGERING H.T., VERHAGEN W.I.M. & THEUNISSEN E.J.J.M. (1995) Hyperactive VOR and hyperventilation after whiplash injury. *Acta Otolaryngol. (Stockh.) Suppl.* **520**, 49–52
- 44 FISCHER A.J.E.M., HUYGEN P.L.M., FOLGERING H.T., VERHAGEN W.I.M. & THEUNISSEN E.J.J.M. (1995) Vestibular hyperreactivity and hyperventilation after whiplash injury. *J. Neurol. Sci.* **132**, 35–43
- 45 LEIGH J.R. & ZEE D.S. (1991) *The Neurology of Eye Movements*, 2nd edn. FA Davis, Philadelphia
- 46 ETTIN T.M., KISCHKA U., REICHMANN S., RADII E.W., HEIM S.Á., WENGEN D. & BENSON D.F. (1992) Cerebral symptoms after whiplash injury of the neck: a prospective clinical and neuropsychological study of whiplash injury. *J. Neurol. Neurosurg. Psychiatry* **55**, 943–948
- 47 DAVIS R.A. & LUXON L.M. (1995) Dizziness following head injury: a neuro-otological study. *J. Neurol.* **242**, 222–230
- 48 PANG L.Q. (1971) The otological aspects of whiplash injuries. *Laryngoscope* **81**, 1381–1387
- 49 ROBIN W. (1973) Whiplash with vestibular involvement. *Arch. Otolaryngol.* **97**, 85–87
- 50 TOGLIA J.U., ROSENBERG P.E. & RONIS M.L. (1970) Post-traumatic dizziness. Vestibular, audiological, and medicolegal aspects. *Arch. Otolaryngol.* **92**, 485–492
- 51 ZENNER P. (1991) *Die Schulterverletzung der Halswirbelsäule und ihre Beurteilung*. Springer Verlag, Berlin
- 52 KORTSCHOT H.W. (1995) Neuro-otological dysfunctions in rear-end car collisions. Thesis, University of Amsterdam
- 53 KINGMA H., PATIN J., PINENBERG H., PAQUA Y. & DOLMANS M. (1992) A study of eye movements elicited by trunk rotation during head fixation in normals and patients with cervical spine injuries (Abstract). *Proceedings of the XVIIIth Bárány Society Meeting* 1–5 June, Prague, pp. 61–62
- 54 HOLTMANN S., REIMAN V. & SCHÖPS P. (1993) Zur klinischen Bedeutung zerviko-okulärer Reaktionen. *Laryngol. Rhinol. Otol.* **72**, 306–310
- 55 SAWYER JR R.N., THURSTON S.E., BECKER K.R., ACKLEY C.V., SHIDMAN S.H. & LEIGH R.J. (1994) The cervico-ocular reflex of normal human subjects in response to transient and sinusoidal trunk rotations. *J. Vestib. Res.* **4**, 245–249
- 56 HUYGEN P.L.M., VERHAGEN W.I.M. & NICOLASEN M.G.M. (1991) Cervico-ocular reflex enhancement in labyrinthine-defective and normal subjects. *Exp. Brain Res.* **87**, 457–464
- 57 HÜLSE M. (1982) Die differentialdiagnostische Auswertung des Zervikalnystagmus. *HNO* **30**, 192–197
- 58 REKER U. (1983) Zervikalnystagmus durch Propriozeptoren der Halswirbelsäule. *Laryngol. Rhinol. Otol.* **62**, 312–314
- 59 BURKE J.P., ORTON H.P., WEST J., STRACHAN I.M., HOCKEY M.S. & FERGUSON D.G. (1992) Whiplash and its effect on the visual system. *Graefes Arch. Clin. Exp. Ophthalmol.* **230**, 335–339
- 60 DUKE-ELDER S. & McFAUL P.A. (1972) Injuries in travel and sport. In *System of Ophthalmology*, (Duke-Elder S. ed.) Vol. 14, pp. 28 and 716, Kempton, London
- 61 LEPORE F.E. (1995) Disorders of ocular motility following head trauma. *Arch. Neurol.* **52**, 924–926
- 62 PEARSON B.W. & BARBER H.O. (1973) Head injury. Some otoneurological sequelae. *Arch. Otolaryngol.* **97**, 81–84
- 63 USHIO N., ISHIDA I., KOIKE S., SHIZUKU S., TADA Y. & INOUE T. (1971) Studies on inverted optokinetic nystagmus in cases with whiplash injury (in Japanese). *Pract. Otol. (Kyoto)* **64**, 493–509
- 64 MERAN A., ROHNER Y. & PFALTZ C.R. (1978) Zur Symptomatologie und Diagnostik der vestibulären Funktionsstörungen nach Schädel-Hirntrauma. *HNO* **26**, 41–48
- 65 LANGE G. & KORNUBER H.H. (1962) Zur Bedeutung peripher- und zentral-vestibulärer Störungen nach Kopftraumen. Elektronystagmographisch-statistische Untersuchungen. *Arch. Ohren-Nasen-Kehlk. Heilk.* **179**, 366–385
- 66 TUOHIMAA P. (1978) Vestibular disturbances after acute mild head injury. *Acta Otolaryngol. (Stockh.) Suppl.* **359**, 1–67
- 67 VARTAINEN E., KARJALAINEN S. & KÄRJÄ J. (1985) Vestibular disorders following head injury in children. *Int. J. Ped. Otorhinolaryngol.* **9**, 135–141
- 68 HOTSON J.R. (1982) Cerebellar control of fixation eye movements. *Neurology* **32**, 31–36
- 69 TOGLIA J.U. (1976) Acute flexion-extension injury of the neck. Electronystagmographic study of 309 patients. *Neurology* **26**, 808–814
- 70 PODOSHIN L. & FRADIS M. (1975) Hearing loss after head injury. *Arch. Otolaryngol.* **101**, 15–18
- 71 GANNON R.P., WILSON G.N., ROBERTS M.E. & PEARSE H.J. (1978) Auditory and vestibular damage in head injuries at work. *Arch. Otolaryngol.* **104**, 404–408
- 72 MONTANDON A. & DITTRICH F. (1962) Ergebnisse der neuen Vestibularisuntersuchungsmethoden bei Schädeltrauma. *Monatsschr. Ohrenheilk. Laryngo-Rhinol.* **96**, 331–343
- 73 KIRTANE M.V., MEDIKERI S.B. & KARNIK P.P. (1982) E.N.G. after head injury. *J. Laryngol. Otol.* **96**, 521–528
- 74 NUTI D., CIACCI G., GIANNINI F., ROSSI A. & FEDERICO A. (1986) Aperiodic alternating nystagmus: report of two cases and treatment by baclofen. *Ital. J. Neurol. Sci.* **7**, 453–459
- 75 HUYGEN P.L.M. (1983) Vestibular hyperreactivity in patients with multiple sclerosis. *Adv. Otorhinolaryngol.* **30**, 141–149
- 76 BALOH R.W., KONRAD H.R. & HONRUBIA V. (1975) Vestibulo-ocular function in patients with cerebellar atrophy. *Neurology* **25**, 160–168
- 77 THURSTON S.E., LEIGH R.J., ABEL L.A. & DELL'ORSO L.F. (1987) Hyperactive vestibulo-ocular reflex in cerebellar degeneration: pathogenesis and treatment. *Neurology* **37**, 53–57
- 78 THEUNISSEN E.J.J.M., HUYGEN P.L.M. & VERHAGEN W.I.M. (1989) Familial vestibulocerebellar dysfunction: a new syndrome? *J. Neurol. Sci.* **89**, 149–155
- 79 HARRIS C.M., WALKER J., SHAWKAT F., WILSON J. & RUSSELL-EGGITT I. (1993) Eye movements in a familial vestibulocerebellar disorder. *Neuropediatrics* **13**, 107–118
- 80 HUYGEN P.L.M., VERHAGEN W.I.M. & RENIER W.O. (1992) Oculomotor and vestibular anomalies in Pelizaeus-Merzbacher disease: a study of a kindred with 2 affected and 3 normal males, 3 obligate and 8 possible carriers. *J. Neurol. Sci.* **113**, 17–25

- 81 HUYGEN P.L.M., VERHAGEN W.I.M., LENSSEN P.P.A. & THEUNISSEN E.J.J.M. (1994) Familial vestibulocerebellar disorder. *Neuropediatrics* **25**, 277
- 82 VERHAGEN W.I.M., HUYGEN P.L.M. & ARTS W.F.M. (1996) Multi-system signs and symptoms in X-linked ataxia carriers. *J. Neurol. Sci.* (in press)
- 83 HUYGEN P.L.M., VAN RIJN P.M., CREMERS C.W.R.J. & THEUNISSEN E.J.J.M. (1993) The vestibulo-ocular reflex in pupils at a Dutch school for the hearing impaired; findings relating to acquired causes. *Int. J. Ped. Otorhinolaryngol.* **25**, 39-47
- 84 HUYGEN P.L.M., VERHAGEN W.I.M., VAN HOOFF J.J.M. & HORSTINK M.W.I.M. (1989) Vestibular hyperreactivity in patients with idiopathic spasmodic torticollis. *J. Neurol. Neurosurg. Psychiatry* **52**, 782-785
- 85 THEUNISSEN, E.J.J.M., HUYGEN P.L.M. & FOLGERING H.T. (1986) Vestibular hyperreactivity and hyperventilation. *Clin. Otolaryngol.* **11**, 161-169
- 86 VERHAGEN W.I.M., HUYGEN P.L.M. & PADBERG G.W. (1995) The auditory, vestibular, and oculomotor system is facioscapulothoracic dystrophy. *Acta Otolaryngol. (Stockh.) Suppl.* **520**, 140-142
- 87 THEUNISSEN, E.J.J.M. (1987) Vestibulaire hyperreactiviteit in relatie tot het hyperventilatiesyndroom (in Dutch, English summary), Thesis, University of Nijmegen
- 88 THEUNISSEN, E.J.J.M., HUYGEN P.L.M., FOLGERING H.T. & NICOLAISEN M.G.M. (1988) The velocity step test: its use in the evaluation of the effects of drugs in dizzy patients. *Acta Otolaryngol. (Stockh.) Suppl.* **520**, 104-113
- 89 FOLGERING H. & COX A. (1981) Betablocker therapy with metoprolol in the hyperventilation syndrome. *Respiration* **41**, 33-38
- 90 MERSKEY H. (1993) Psychological consequences of whiplash (State of the Art Reviews). *Spine* **7**, 471-480
- 91 YARDLEY L., WATSON S., BRITTON J., LEAR S. & BIRD J. (1995) Effects of anxiety arousal and mental stress on the vestibulo-ocular reflex. *Acta Otolaryngol. (Stockh.)* **115**, 597-602
- 92 ROOS J.W.P. (1991) Posturography in the tilting room. Thesis, Free University of Amsterdam
- 93 RUBIN A.M., WOOLLEY S.M., DAILY V.M. & GOEBEL J.A. (1995) Postural stability following mild head or whiplash injuries. *Am. J. Otol.* **16**, 216-221
- 94 YARNELL P.R. & ROSSIE G.V. (1988) Minor whiplash head injury with major debilitation. *Brain Injury* **2**, 255-258
- 95 SERRA L.L., GALLICCHIO B., SERRA F.P., GRILLO G. & FERRARI M. (1994) BAEP and EMG changes from whiplash injuries. *Acta Neurol. Ital.* **16**, 262-270
- 96 JACOME D.E. (1987) EEG in whiplash: a reappraisal. *Clin. Electroencephalogr.* **1**, 41-45
- 97 DVORÁK J., PANJABI M.M., GROB D., NOVOTNY J.E. & ANTINNES J.A. (1993) Clinical validation of functional flexion/extension radiographs of the cervical spine. *Spine* **18**, 120-127
- 98 JÓNSSON JR. H., CESARINI K., SÄLLSTEDT B. & RAUSCHNING W. (1994) Findings and outcome in whiplash-type neck distortions. *Spine* **19**, 2733-2743
- 99 GRIFFITHS H.J., OLSON P.N., EVERSON L.I. & WINEMILLER M. (1995) Hyperextension strain or 'whiplash' injuries to the cervical spine. *Skeletal Radiol.* **24**, 263-266
- 100 ANTINNES J.A., DVORÁK J., HAYEK J., PANJABI M.M. & GROB D. (1994) The value of functional computed tomography in the evaluation of soft-tissue injury in the upper cervical spine. *Eur. Spine J.* **3**, 98-101
- 101 BORCHGREVINK C.E., SMEVIK O., NORDBY A., RINCK P.A., STILES T.C. & LERHEIM I. (1995) MR imaging and radiography of patients with cervical hyperextension flexion injuries after car accidents. *Acta Radiol.* **36**, 425-428
- 102 DAVIS S.J., TERESI L.M., BRADLEY JR. W.G., ZIEMBA M.A. & BLOZE A.E. (1991) Cervical spine hyperextension injuries: MR findings. *Radiology* **180**, 245-251
- 103 HALL A.J., WAGLE V.G., RAYCROFT J., GOLDMAN R.L. & BUTLER A.R. (1993) Magnetic resonance imaging in cervical spine trauma. *J. Trauma* **34**, 21-26
- 104 PIETTERSSON K., HILDINGSSON C., TOOLANEN G., FAGERLUND M. & BJÖRNEBRINK J. (1994) MRI and neurology in acute whiplash trauma. No correlation in prospective examination of 39 cases. *Acta Orthop. Scand.* **65**, 525-528
- 105 SCHNARKOWSKI P., WEIDENMAIER W., HEUCK A. & REISER M.F. (1995) MR-Funktionsdiagnostik der Halswirbelsäule nach Schläudertrauma. *Fortschr. Röntgenstr.* **162**, 319-324
- 106 NÄGELE M., KOCH W., KADEN B., WÖLL B. & REISER M. (1992) Dynamische Funktions-MRT der Halswirbelsäule. *Fortschr. Röntgenstr.* **157**, 222-228
- 107 PIETTERSSON K., KARRHOLM J., TOOLANEN G. & HILDINGSSON C. (1995) Decreased width of the spinal canal in patients with chronic symptoms after whiplash injury. *Spine* **20**, 1664-1667
- 108 LESTINI W.F. & WIESEL S.W. (1989) The pathogenesis of cervical spondylosis. *Clin. Orthop.* **239**, 69-93
- 109 FUKUI K., KATAOKA O., SHIO T. & SUMI M. (1990) Pathomechanism, pathogenesis, and results of treatment in cervical spondylotic myelopathy caused by dynamic canal stenosis. *Spine* **15**, 1148-1152
- 110 TORG J.S. (1990) Cervical spinal stenosis with cord neurapraxia and transient quadriplegia. *Clin. Sports Med.* **9**, 279-296
- 111 RADANOV B.P., DI STEFANO G., SCHNIDRIG A., STURZENEGGER M. & AUGUSTINY K.F. (1993) Cognitive functioning after common whiplash. A controlled follow-up study. *Arch. Neurol.* **50**, 87-91
- 112 RADANOV B.P., DI STEFANO G., SCHNIDRIG A. & STURZENEGGER M. (1994) Common whiplash: psychosomatic or somatopsychic? *J. Neurol. Neurosurg. Psychiatry* **57**, 486-490
- 113 DI STEFANO G. & RADANOV B.P. (1995) Course of attention and memory after common whiplash: a two-years prospective study with age, education and gender pair-matched patients. *Acta Neurol. Scand.* **91**, 346-352
- 114 PARKER R.S. & ROSENBLUM A. (1996) IQ loss and emotional dysfunctions after mild head injury incurred in a motor vehicle accident. *J. Clin. Psychol.* **52**, 32-43
- 115 LEE J., GILES K. & DRUMMOND P.D. (1993) Psychological disturbances and an exaggerated response to pain in patients with whiplash injury. *J. Psychosom. Res.* **37**, 105-110
- 116 MAYOU R., BRYANT B. & DUTHIE R. (1993) Psychiatric consequences of road traffic accidents. *Br. Med. J.* **307**, 647-651
- 117 OLSNES B.T. (1989) Neurobehavioral findings in whiplash patients with long-lasting symptoms. *Acta Neurol. Scand.* **80**, 584-588
- 118 HOLLNAGEL H. & NORRELUND M. (1980) Headache among 40-year-olds in Glostrup. An epidemiologic study. *Ugeskr. Laeger* **142**, 3071-3077
- 119 GARGAN M.F. & BANNISTER C.G. (1994) The rate of recovery following whiplash injury. *Eur. Spine J.* **3**, 162-164
- 120 MERSKEY H. & WOODFORD J.M. (1972) Psychiatric sequelae of minor head injury. *Brain* **95**, 521-528
- 121 RIMEI R.W., GIORDANI B., BARTH J.T., BOLL T.J. & JANE J.A. (1981) Disability caused by minor head injury. *Neurosurgery* **9**, 221-228
- 122 RADANOV B.P. & STURZENEGGER M. (1996) Predicting recovery from common whiplash. *Eur. Neurol.* **36**, 48-51
- 123 NORRIS S.H. & WATT I. (1983) The prognosis of neck injuries resulting from rear-end vehicle collisions. *J. Bone Joint Surg. [Br]* **65B**, 608-611

- 124 PARMAR H.V. & RAYMAKERS R. (1993) Neck injuries from rear impact road traffic accidents: prognosis in persons seeking compensation. *Injury* **24**, 75-78
- 125 WATKINSON A.F. (1990) Whiplash injury. *Br. Med. J.* **301**, 983
- 126 BARNESLEY L., LORD S.M., WALLIS B.J. & BOGDUK N. (1995) The prevalence of chronic cervical zygapophysial joint pain after whiplash. *Spine* **20**, 20-26
- 127 BARNESLEY L., LORD S.M., WALLIS B.J. & BOGDUK N. (1994) Lack of effect of intraarticular corticosteroids for chronic pain in the cervical zygapophysial joints. *N. Engl. J. Med.* **330**, 1047-1050
- 128 BYRN C., OLSSON L., FALKHEDEN L., LINDH M., HÖSTEREY U., FOGELBERG M., LINDER L.-E. & BUNKETORP O. (1993) Subcutaneous sterile water injections for chronic neck and shoulder pain following whiplash injuries. *Lancet* **341**, 449-452
- 129 PEARCE J.M.S. (1993) Pain relief by water injections. *Lancet* **341**, 905
- 130 SKRABANEK P. (1993) Pain relief by water injections. *Lancet* **341**, 905
- 131 MCKINNEY L.A. (1989) Early mobilisation and outcome in acute sprains of the neck. *Br. Med. J.* **299**, 1006-1008
- 132 SCHUTT C.H. & DOHAN F.C. (1968) Neck injury to women in auto accidents. A metropolitan plague. *J. Am. Med. Assoc.* **206**, 2689-2692
- 133 HOHL M. (1974) Soft tissue injuries of the neck in automobile accidents: factors influencing prognosis. *J. Bone Joint Surg. [Am]* **56A**, 1675-1682
- 134 GARGAN M.F. & BANNISTER G.C. (1990) Long-term prognosis of soft-tissue injury of the neck. *J. Bone Joint Surg. [Br]* **72B** 901-903