NEURO-OTOLOGICAL DYSFUNCTIONS IN REAR-END CAR COLLISIONS

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**ACADEMISCH PROEFSCHRIFT**

ter verkrijging van de graad van doctor
aan de Universiteit van Amsterdam,
op gezag van de Rector Magnificus
Prof.dr. P.W.M. de Meijer
ten overstaan van een door het College van
Decanen ingestelde commissie in het openbaar
te verdedigen in de Aula der Universiteit
op donderdag 16 november 1995 te 12.00 uur
door Hendrika Willemina Kortschot
geboren te Winterswijk

CopyPrint 2000
Enschede
1995

Promotor: Prof.dr. W.J.Oosterveld

Publication of this thesis was supported by "Duphar Nederland, producent van Betaserc®", UCB Farma Nederland BV and Entermed BV.

Ter nagedachtenis aan
mijn ouders
en
Bram Meijer

This research was performed in the Vestibular Department, ENT Clinic, University of Amsterdam, The Netherlands.

ISBN 90-9008813-X
NUGI 742



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## Introduction

The cervical acceleration syndrome is the entity of signs and symptoms originating in an accident in which the body is subjected to a sudden acceleration, putting a severe strain on the cervical structures. This kind of trauma has synonyms such as whiplash injury, cervical sprain, cervical strain, hyperextension/hyperflexion injury, coup de lapin, HWS-Schleudertrauma, Peitschenslagsyndrom, Zervico-zephales Beschleunigungstrauma and nakkesleng.

The assembly of the European section of the Cervical Spine Research Society (CSRS) at Erlangen in 1995 advised strongly to use the term "cervical distorsion syndrome" (95).

The Quebec Task Force on Whiplash-Associated Disorders (80) adopted in 1995 the following definition of whiplash: " 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)".

Patients with symptoms or with residual disability 6 months or more after the injury are designated as chronic (80).

Not any theory is yet able to explain the variety of complaints.

Interdisciplinical deliberation is a necessity to gain more insight into the complexity of symptoms following cervical acceleration injuries.

The aim of this study in patients suffering from a chronic cervical acceleration injury was twofold:

- to investigate the origin of neuro-otological signs and symptoms.
- to examine the effect of fatigue resulting from the strain of a neuro-otological investigation.

## I Mechanism and effect of a rear-end car collision.

### Mechanism

H.Crowe (18) was the first person to use the term "whiplash". He intended to describe the motion, but not the name of a disease, as was done by others later on (19). The term "whip lash" was first used by Davis (20,30).

Gay and Abbott (34) reported that most whiplash injuries of the neck were caused by rear-end car collisions, which resulted in a sudden, forceful flexion of the neck, sometimes followed by several other less violent oscillations of the neck in alternating flexion and extension.

As a result of a rear-end collision the car is accelerated forward, whereby the victim's body is forced into the seatback, resulting into a forward acceleration of the body and shoulders, while the head and neck initially remain fixed. The forward acceleration of the trunk causes a pull of the trunk to the lower part of the head resulting in retroflexion of the cervical spine, which occurs within 250 milliseconds. This period is too short for a neck muscles reaction (16).

Walz (96) mentioned that retroflexion is only followed by flexion of the cervical spine, when the car after the rear- end impact collides with a car in front. Another possibility, resulting in hyperflexion of the head, is the construction of a head restraint, causing forward movement of this head restraint at the moment when the cervical spine retroflects. Matsushita et al (62) performed sled tests in 26 subjects to simulate rear-end collisions with a vehicle velocity change of less than five km/h, while their subjects were observed with cineradiographic, accelerometrical and electromyographic technics. Six of their subjects experienced mild discomfort symptoms, probably due to micro-injuries of the soft tissues of the neck because of the passive loads beyond the physiological tolerance occurring at the onset rate or because of the overactive muscles defense. When the subject was leaning forward in the stooped shoulder posture, the cervical spine was subjected to a compression load, while the length of the cervical spine seemed to shorten and the cervical lordosis and the thoracic kyphosis were straightened. Cervical flexion occurred before extension.

Walz (95) observed that after a rear-end collision the cervical vertebrae rotated backwards one by one. While the lowest cervical vertebra is rotated backward, the next

upper vertebra flexes. So the torque forces starting at the lower level climb up. According to Walz the degree of backward rotation depends on the initial distance between the head and the head restraint.

Most people are not looking straight ahead at the moment of the impact. When the head is rotated at the moment of the impact, the rotation will increase before the hyperextension resulting in a greater chance of injury (95).

The magnitude of loading forces of an occupant during a rear-end car collision is influenced by:

1. Collision velocity
2. Size and weight of colliding cars
3. Peak acceleration of the struck car
4. Strength, position and height of seatback
5. Design and position of head restraint
6. Posture of the victim at the moment of impact
7. Head position of the victim at the moment of impact
8. Wearing a seatbelt
9. Body size of the victim

When the head restraint extends not above the center of gravity of the victim's head, it acts as a fulcrum over which the cervical spine pivots (16).

According to Croft (16) Unterharnscheidt observed in rhesus monkeys that the position of the head at the beginning of the impact was very important. After the acceleration the next sequence of events occurred, when the initial position was slight flexion of the neck: compression of the entire cervical spine, followed by marked flexion of the cervical spine in the second phase and also by rotation of the head in forward direction, a phenomenon termed the "concertina effect". This initial sequence absorbs a substantial amount of kinetic energy and is followed by hyperextension.

Some cars are equipped with shoulder harnesses that can be locked in a position at a distance from the occupant's chest.

Because of this slack the victim's body can travel to make contact with parts of the car's interior, for example the steering wheel. Also this slack can potentiate forward rotation of the cervical, thoracic and lumbar spine resulting in a more severe injury to these areas.



Depending on the initial position of the head and the neck several loading forces such as axial tension and compression forces, shearing forces, flexion and extension forces and torsion forces can be generated on these structures (98). The loading forces acting on the head can cause an impact of the skull on the brain, because the brain, relatively lying free in the skull, tends to maintain its position in space due to its relative inertia (10). Already Gay and Abbott (34) mentioned not only a sudden mechanical deformity and pressure on the frontal and temporal lobes of the brain, but they also suggested an effect of acceleration or deceleration on the brain mass, which can be understood as effect of the forces on the brain structures in itself. Furthermore, animal experiments revealed that rotational acceleration of the head could induce lesions in the brain as well as through the entire lenght of spinal cord (94).

McConnell et al (64) analysed in four healthy male test subjects the responses to low velocity rear-end impacts using four different cars. Data were obtained from high speed and video films as well as electronic accelerometer measurements. The stationary striking car was subjected to velocity changes of four to eight km/h. The resultant acceleration force can only be calculated, when weight and velocity of the colliding vehicles are known. The authors (64) did not mention these data, but they described one test subject's response to one test condition. The photographic analysis revealed five phases: 1) the initial response (0 to 100 msec), 2) the principal forward acceleration (100 to 200 msec), 3) the head overspeed/trunk recovery (200 to 300 msec), 4) the head deceleration/trunk rest (300 to 400 msec) and 5) the restitution (400 to 600 msec).

During the first phase about 60 milliseconds after the collision the lower part of the seatback pushed the subjects hip and low back forward and upward, while the upper part of the seatback retroflected and the seatback cushion was being compressed on the subject's almost stationary upper trunk. After 100 milliseconds during the second phase the subject's upper torso began to move forward and upward, while his head and neck were still nearly stationary. In 120 milliseconds, when the subjects trunk and hips still moved upward, an axial compression of the neck was observed. The neck straigtened as soon as the top of the cervical spine started to move upward and rearward. Next the head began an upward and rearward rotation with respect to the shoulders. After 160 milliseconds the ascended upper trunk pulled the base of the neck forward into apparent tension and begun to move the subject's head forward, even as the occiput continued to

move downward to the headrest. At the beginning of the third phase after about nine centimeters rise of trunk and shoulders and 45 degrees rotational rearward angulation the upward movement of the trunk had stopped and the head moved forward. After 250 milliseconds the trunk, neck and head moved downward parallel to the seatback,which had returned to its pre-collision position. The forward moving head had not yet reached the vertical. In the fourth phase the descent of the trunk was completed. The head still moved forward, but was decelerated by the tension in the neck. After 400 milliseconds the head reached its most forward position and begun to move rearward and upward to a normal upright position. In the last phase, about 450 milliseconds after the collision the total body had gained the car's velocity. The head returned to the pre-collision position, but the shoulders and hips came in the 3,8 centimeters higher rest position.

During these experiments the cervical extension and flexion of the subjects were always within their voluntary physiological limits. Three out of four test subjects reported only mild neck pain after multiple test exposures.

The response analysis of high velocity rear-end impacts and of low velocity impacts on humans with rotated neck is never done before.

**Effects**

Structures that can be damaged by a rear-end car collision are:

the neck

- cervical vertebrae
- zygapophysial joints
- intervertebral discs
- ligaments
- muscles
- oesophagus
- autonomic plexus
- nerve roots
- cervical spinal cord
- vascular structures

the temporo-mandibular joints

the brain



- indirectly
  - vascular damage
  - central disregulation due to damage of the nerve endings in the cervical region
- directly
  - shearing/tearing of cranial nerves
  - mechanical impact of the skull on the brain
  - internal brain deformation by rotation of brain within the skull
  - internal shearing of axons and/or other brain tissue

the shoulders and the low back

**Neck**

During extension the spinal processes and the zygapophysial joints are subjected to compression sometimes resulting in fractures or dislocation. Furthermore the intervertebral discs, the longitudinal anterior ligament, the sternocleidomastoid muscles, the prevertebral muscles and the oesophagus can be damaged (5). An initial rotation of the head at the moment of the impact increases during the acceleration after the rear-end collision. This can lead to more bone and tissue damage. The superficial branches of the cervical plexus can be stretched, when the neck is extended, especially when the extension is combined with rotation (59).

Flexion can cause compression and damage to the vertebrae, intervertebral discs, the zygaphophysial joints as well as cause injuries of the posterior ligaments e.g. the posterior longitudinal ligament, the interspinous ligament and the nuchal ligament. During lateral flexion the intervertebral discs and the zygapophysial joints are most endangered (5).

C4-C5 is the region of greatest strain and stress in extension, whereas in flexion this region is C5-C6 (5). The cadaver studies of Clemens and Burow showed the most injuries in the regions C5-C6 and C6-C7 (5).

With respect to the cervical spinal cord Raynor and Koplik (82) described four clinical syndromes resulting from cervical cord trauma which cover findings from minor root involvement to complete cord dysfunction. These authors (82) mention that the initially applied force leads to pathologic changes, whether the mechanisms be mechanical,

vascular or chemical. An applied force can lead to a mechanical lesion of the spinal cord when the cord strikes a surface, such as a dislocated vertebra, a fractured bone or an infolded ligament or is impressed by these structures. Also the force displaces the segment above with respect to the segment below the point of force application. This displacement induces a shearing stress in the plane of the force. According to these authors (82) the injury patterns with respect to the nature of injury, hyperflexion or hyperextension, are similar. So, there is little relevance to an antero-posterior or a postero-anterior force application. The four clinical syndromes with increasing severity of injury, because of a progression in the magnitude of the applied forces are: a root syndrome, a central cord syndrome, an anterior cord syndrome and a complete cord dysfunction. Variations in the clinical syndromes are not only due to the magnitude, but also to the direction of applied forces. Furthermore, Raynor and Koplik (82) describe that gray and white matter react differently to a trauma. The white matter seems to sustain a blunt contusion with delayed and variable effects on blood flow, while the gray matter seems to undergo a sharp contusion with rapid and long-lasting effects.

Martin et al (61) reported about a 64-year-old man complaining of weakness of his limbs immediately after a rear-end car collision in which his car was hit at about 10 km/h. The patient had neither a head trauma nor neckache. A neurological examination revealed a tetraparesis. Radiographs of the cervical spine demonstrated a narrowing of the spinal canal at the C3-4 en C4-5 levels without vertebral fractures or displacement. The day after admission the MRI disclosed disc protrusions at the C3-4, C4-5 and the C6-7 levels. An area of hyperintense signal occupied the whole width of the cord and was centered at the C3-4 intervertebral space. Two days after the accident an anterior resection of the C4 and C5 vertebral bodies was done to relieve any compression. The patient died from heart failure a few hours after the operation. During autopsy macroscopic examination demonstrated a slight softening and a brownish coloration of the right anterior horn in the C4 and C5 segments and no abnormalities of the meninges. Microscopic examination disclosed oedema in some areas and axonal swelling and disruption, especially in the white matter tracts of the lateral and dorsal funiculi. These axonal injuries were restricted to the C3 and C4 segment. A recent coagulative necrosis was demonstrated in the right anterior horn of C4 and partially of C5, while most motor neurons were destroyed or shrunken. Some of these neurons showed central



chromatolysis. Considering the autopsy was done in the acute stage, the authors (61) supposed that the axonal injuries are due to mechanical tearing forces, because the lesions were restricted to one or two segments and did not correspond to a well-defined vascular territory.

In this case report the cervical cord damage was likely caused by the mechanical disc protusion and axonal injury.

**Temporo-mandibular joint**

The sudden hyperextension can cause anterior mandibular displacement as the mouth opens, sometimes leading to temporomandibular joint dysfunction. In case of sudden deceleration the mandible moves fast posteriorly, closing the mouth, which can damage the teeth without impact to the jaw itself (97).

**Brain**

Indirect mechanism

Brain damage could be caused indirectly by vertebral artery injury or by damage of the internal carotid artery, because extracranial injury may cause intracranial neurologic complications, e.g. an occlusion of the posterior inferior cerebellar artery by a thrombus from the vertebrobasilar arteries resulting in the Wallenberg syndrome. Shearing forces due to rotational forces or direct injuries of the vessels can induce a tearing of the intima, endothelial damage, activation of thrombine and subsequent thromboembolism. Symptoms can be delayed for weeks or months (91). In case of zygapophysial joint dislocation damage of the vertebral artery should be considered (57).

Kessler et al (50) examined a 64-year-old man after a rear-end collision. He was conscious, but complained about neckache immediately after the accident. Twelve days after the accident the patient got an amaurosis of his right eye, a hemiparesis of his left arm and the left part of his face and a right-sided elevation of the reflexes. He also had a leftsided hemineglect and an anosognosis for the hemiparesis. The CT-scan disclosed an infarct in the right middle cerebral artery area. Angiography revealed a stenosis of the right internal carotid artery and also an irregular intima at the origin of the middle

cerebral artery. Scintigraphy showed a cluster of platelets in the right internal carotid artery. According to Kessler et al (50) the posttraumatic onset of neurologic symptoms in these cases can vary from hours to several days.

Barontini and Mauri (6) reported about a 45-year-old man with a whiplash injury after a traffic accident. Neurologic examination immediately after the accident did not reveal any pathology, but the patient got headache and sporadic attacks of somnolence and sweatening. After three weeks the patient became confused and forgetful and a hetero-anamnestic upward gaze palsy was mentioned. Neurologic examination disclosed no gaze palsy, but a slight disorientation in time and space, a global amnesia, confabulation and an aprosopognosia. The CT-scan was negative, but the MRI scan showed a small infarct in the left temporal lobe and two small bilateral paramedian infarcts. A Doppler scan of the carotid and vertebral arteries disclosed no abnormalities. These could not be confirmed by cerebral angiography, because it was refused by the patient.

Neuropsychologic examinations showed not only a deficit in verbal and short-term memory, but also in non verbal and long term memory. However, acquisition and storage of new information was most deficient. The memory impairment was improved one year after the accident.

The authors (6) supposed a relation between the cervical injury and the thalamic infarcts was plausible, because the patient had no risk factors for stroke, for example advanced age, diabetes, hypertension, hypercholesterolaemia or smoking.

Central disregulation because of change or jam of proprioceptive and/or nociceptive impulses from the cervical region is another possibility to explain brain dysfunction. This assumption will be discussed in chapter 3 as a possible explanation of neuro-otological dysfunction.

A brain nerve dysfunction because of shearing or tearing forces is plausible.

Helliwell (39) investigated a 65-year-old woman after a rear-end car collision in which she lost consciousness for several minutes without striking her head. Afterwards she felt immediately neck and chest pain. She had a tachycardia, a stridor, a weak phonation and she complained of diplopia. The examination showed bilateral lateral rectus palsies. A laryngoscopy demonstrated immobile vocal cords in the paramedian plane. The nerve functions did not recover. Helliwell (39) ascribed the nerve lesions to the strecthing forces and the tachycardia to unopposed sympathetic action on the patients



heart. He assumes that an acceleration trauma of the neck, a non contact necktrauma, can cause unconsciousness and brain stem dysfunction because of the shearing and stretching forces within the skull, particularly in downward direction.

Dukes and Bannerjee (24) reported about a 57-year-old woman who developed within a week after a rear-end car collision a right hypoglossal nerve dysfunction. Without any treatment she was asymptomatic 5 months after the accident with only minor asymmetry remaining. They assumed that the mechanism of the injury could be due to both a traction of the hypoglossal nerve at the atlanto-occipital level and oedema or haematoma around the hypoglossal canal.

Direct mechanism

It is likely that the loading forces due to a rear-end car collision can also lead directly to brain dysfunction/damage and not only by mechanical forces resulting in an impact of the skull against the brain (10). The degree of damage will depend on both the structure of the brain tissue and the covering surface. So parts of the brain covered by smooth surfaces, e.g. the occipital lobes, should suffer less damage than the parts covered by rough surfaces, as there are the frontal, temporal and orbital cortex (71). According to Ommaya and Gennarelli (71) the distribution of brain damage induced by inertial loading decreases in magnitude from the surface to the center. In case of this mechanism the injuries at low inertial loading should be localized at cortical and subcortical levels.

A second mechanism of brain damage could be due to rotation of the brain and dura within the skull, while internal shearing of the brain because of the different morphology of the brain tissue could be assumed the third mechanism.

The existence of this last mechanism became plausible considering the findings of Maxwell et al (63). They demonstrated axonal injuries in non-human primates after subjecting these animals to a single episode of angular acceleration of the head in lateral direction. Furthermore, these investigators (63) discovered that regions with axonal injuries were related to regions with a normal axonal structure, that shearing occured more in small than in large axons, while the node/paranode junctions most frequently were injured.

Hildingson et al (40) studied the oculomotor function in 20 symptomatic and 19

asymptomatic patients with a previous soft- tissue injury of the cervical spine following a car collision. These two groups were matched with 25 healthy subjects without a history of a head injury or a soft-tissue injury of the neck. The study was performed six months or more after the accident. The asymptomatic patients had no complaints anymore. The symptomatic patients not only complained amongst others about neck pain, headache and shoulder and arm pain, but 18 of them also had vertigo or dizziness. Neurological examination revealed no abnormalities. With respect to the oculomotor tests the symptomatic patients had significant reduced velocity gain of smooth pursuit, reduced peak velocity of the saccadic eye movements and significant prolonged latencies of the saccades as compared to both the group of asymptomatic patients and the control subjects. The investigators (40) observed a moderate oculomotor dysfunction in 14 symptomatic patients and a pronounced dysfunction in four of them. They considered that the moderate oculomotor dysfunctions could be caused by affection of the proprioceptive system in the cervicocranial area. The pronounced dysfunctions could probably be ascribed to brainstem lesions.

So both an indirect and a direct mechanism plays a role in the the oculomotor dysfunction. But these authors (40) give no explanation about the cause of directly induced brainstem lesions.

Burke et al (13) performed an ophthalmic and oculomotor examination in 39 whiplash patients within one week after the trauma. The examination was repeated after six weeks. Eighteen of them had no ophthalmic symptoms or signs. Four patients already having an opthalmic abnormality before the accident, had no new symptoms or signs.

In seventeen other patients abnormalities were observed. Two of them complained of symptoms related to the accident, but they had no abnormalities on physical examination. Ophthalmic examination disclosed a reduced stereoacuity in one patient and abnormal pursuits and fusion abnormality in another one. Fourteen of these patients complained of symptoms related to the accident. They had a reduced mobility of their neck, but no neurologic abnormalities. Ophthalmic examination revealed decreased convergence, accommodation, fusion range and reduced stereoacuity. Two patients had superior oblique muscle weakness. In another patient bilateral vitreous detachments were seen. Four patients had abnormal pursuits. One patient with symptoms, reduced mobility of the neck and "objective" neurological loss also had a decreased accommo-



dation, but the pursuit was normal. In most patients the ophthalmic signs resolved within nine months. Only in two of them the signs did not disappear.

According to Burke et al (13) all but one's patient's symptoms were caused by ocular motor abnormalities. They assume that the etiology of the convergence and accommodation disturbances in the context of whiplash should be at brainstem level. With respect to the muscle weakness they consider axonal shearing at brainstem level, damage to minute vessels supplying nuclei or small hemorrhages at this level. The abnormal pursuit is prescribed to a secondary brainstem dysfunction with/without proprioceptive dysfunction in the cervical region. Normal fusion and stereoacuity needs a normal semidecussation of optic nerve fibers at the optic chiasm and an intact corpus callosum. So these authors (13) could not explain the combination of normal fusion and decreased stereoacuity, because lesions in the corpus callosum were never described in whiplash patients. However, Maxwell et al (63) demonstrated axonal injuries in the corpus callosum of non-human primates after they were subjected to an angular acceleration of the head in lateral direction.

Knibestöl et al (52) described a trigeminal sensory impairment in patients suffering for six months or more from the after-effects of a soft-tissue injury of the cervical spine following front-end, rear-end, side and single and roll-over car collisions. They examined 16 symptomatic, 14 asymptomatic patients and 17 healthy control subjects. These investigators (52) observed a significant impairment of vibration and temperature sensitivity in the trigeminal skin area in 14 of the symptomatic patients, while two of them had normal cutaneous sensations. The findings obtained in the asymptomatic patients revealed no significant difference compared to the control subjects. Only one asymptomatic patient had slight trigeminal impairment.

An injury to the peripheral parts of the trigeminal nerve seems unlikely because of the bilateral distribution of the sensory changes. Knibebestöl et al (52) point to the brainstem and upper spinal cord as the probable site of injury. Two mechanisms: a direct injury of the brainstem caused by the movements of the head and an indirect vascular insufficiency could explain their findings. The authors (52) also state that subtle damage to brain and brainstem structures may not be detected by morphologic techniques as CT or MRI, but only by sensitive functional tests based on neurophysiologic methods.

Blakely and Harrington (8) saw a 34-year-old woman four months after a rear-end car collision. She suffered from encephalitis and probably Guillain-Barré syndrome at age 15 and 20 years, respectively. A shelf struck her on the occiput, when she was 25 years old. Alteration of sensorium was not mentioned, but the accident caused headache, photophobia and diplopia. These symptoms disappeared completely within three months. After the rear-end collision she complained again of diplopia. She was not able to make even simple drawings. Furthermore, she had problems with a serial verbal learning task and word finding difficulties. Brain mapping disclosed an abnormality in the right temporal area. This woman already suffered from three other neurological incidents, but she was asymptomatic before the rear-end car collision. The investigators (8) mentioned that although the patient had clinically recovered from the first three incidents, it was not sure the damage to the brain was completely reversed. They assumed that the "reserve capacity" was sufficient to "normal" level of functioning. The cumulative damaging effect of the fourth incident, the rear-end car collision, presumably exceeded the limits causing the symptoms in these patient.

Keidel et al (49) registered a jaw opening reflex in 69 control subjects and 61 whiplash patients within two weeks after the accident. In the whiplash patients the jaw opening reflex was impaired. These authors assumed that the findings were based on a transient dysfunction of this brainstem- mediated reflex circuit. They measured a shortened duration of the late exteroceptive EMG suppression (ES2) of the masseter inhibitory reflex in patients with neckpain and headache following a whiplash injury. However, the latency of the early exteroceptive EMG suppression (ES1) did not change in these patients. The ES1 is mediated by a oligosynaptic circuit, while the ES2 are relayed through interneurons, a multisynaptic circuit. Keidel et al (49) presumed the findings could be explained by posttraumatic changes at the neurotransmitter levels. Affection of the multisynaptic circuit by proprioceptive and/or nociceptive stimuli of the cervical region could be another explanation.

The phenomenon of a posttraumatic change at neurotransmitter level already was described in rats by Boismare et al (12).

They measured in rats two days after an experimental whiplash injury without any direct blow to the head a change in dopamine and noradrenaline levels in various areas of the brain.



The injured rats had to be housed in individual cages because of aggressiveness. There was also a decrease of learning ability in the injured rats as compared to the control rats.

Furthermore, the injured rats had a significantly increased norepinephrine level in the hippocampus, increased dopamine and noradrenaline levels in the thalamus-hypothalamus and corpus striatum and a decreased noradrenaline level in the medulla oblongata. The authors (12) did not have an explanation for the mechanism by which a whiplash injury modified the metabolism of central catecholamines.

An overview of the presumed mechanisms leading to spinal cord and brain dysfunction is given in table 1.

| Authors | Signs and findings | Explanation |
|---|---|---|
| Raynor and Koplik (82) | root syndrome<br>central cord syndrome<br>anterior cord syndrome<br>complete cord dysfunction | shearing forces, increase of applied forces resulting in the first to the fourth syndrome, respectively, by mechanical, vascular or chemical mechanims |
| Martin et al (61) | tetraparese<br>disc protrusion at C3 to C7 levels<br>axonal injuries at autopsy | mechanical pression<br>mechanical tearing forces |
| Kessler et al (50) | hemiparesis left arm and face, rightsided reflexelevation, leftsided hemineglect, anognosis of hemiparesis<br>right middle cerebral artery infarct<br>stenosis of right internal carotis artery<br>irregular intima at origin right middle artery | vascular mechanism |
| Barontini and Mauri (6) | headache, somnolence and sweating attacks<br>after three weeks confused, forgetful, disoriented in time and space<br>left temporal lobe and bilateral paramedian infarcts | vascular mechanism |

| | | |
|---|---|---|
| | cerebral angiography refused | |
| Helliwell (39) | unconsciousness for few minutes, tachycardia, stridor, immobile vocal cords, diplopia, bilateral rectus palsies | shearing and stretching forces brain<br>sympathetic action on heart<br>nerve lesions by stretching forces |
| Dukes and Bannerjee (24) | after one week a swollen tongue and speech impairment<br>after a month waste of right side of tongue, deviation to the right when protruding | traction of nerve at atlanto-occipital region<br>oedema or heamatoma around hypoglossal canal |
| Hildingson et al (40) | a.o. neck pain, headache, shoulder and arm pain, vertigo and dizziness<br>no neurologic abnormalities-reduced gain of smooth pursuit<br>reduced eye movement velocities<br>prolonged latencies of saccade test | indirect dysfunctions by affection of cervical proprioceptors<br>direct brainstem dysfunction-vascular disturbances at brainstem level |
| Burke et al (13) | neck pain and reduced mobility of neck<br>no neurological abnormalities<br>decreased convergence and accommodation, reduced fusion range and decreased stereoacuity<br>abnormal pursuit<br>superior oblique muscle weakness<br>bilateral vitreous detachement | axonal shearing at brainstem level |
| Knibestöl et al (52) | impairment of vibration and temperature sensitivity | injury of trigeminal nerve at upper spinal cord or brainstem level due to direct brainstem injury or indirect injury because of vascular insufficiency |
| Blakely and Harrington (8) | diplopia, word finding difficulties, serial verbal learning task problems<br>abnormality in right temporal | cumulative brain damaging effect of several incidents |



| | | |
|---|---|---|
| | area during brain mapping | |
| Keidel et al (49) | impaired jaw opening reflex | posttraumatic changes of neurotransmitter levels directly or indirectly by proprioceptive or nociceptive stimuli from the cervical region |
| Boismare et al (12) | decrease of learning abilities and increased aggressiveness in rats | change of central catecholamine metabolism |

Table 1. Mechanism probably causing spinal cord and brain dysfunction.

**Shoulder, chest and low back**

Shoulder pain is frequently mentioned by patients. Mostly it is a pain originating from the trapezius muscles, which are often in spasm. However, the pain can also represent a herniated cervical disc or can be referred pain from deeper muscular, ligamentous or osseous structures of the cervical spine (17).

Pain over the female breast and chest pain can result from the seatbelt. Furthermore, many patients complain of low back pain. So during physical examination inspection of the complete spine has to be included (17).

## I Review of literature concerning electronystagmographic, neuropsychological and radiological findings.

### Electronystagmographic examinations

Gay and Abbott (34) already described that all 50 patients suffering from the after-effects of a whiplash injury, mostly following a rear-end car collision, complained of vertigo. However, they were not able to perform an electronystagmographic examination in their patients. These authors explained the vertigo by the assumption of a vasomotor instability or a disturbance of the vestibulospinal system through a direct nerve root irritation or a hypertonicity of the cervical spinal muscles.

Pang (74) reported about vestibular aspects in 20 patients referred because of vertigo, dizziness, lightheadedness, loss of balance or a tendency to fall. Electronystagmography (ENG) was performed within one year after the accident in 10 patients. In another 10 the accident occurred more than one year ago.

The test battery consisting of the registration of spontaneous nystagmus, postural tests including rotation of the neck and the caloric test. Oculomotor tests were not performed. Only in one patient, examined one week after the accident, a spontaneous nystagmus was registered. In seven patients a postural nystagmus was present, which tended to disappear in time. In eight patients the caloric test revealed a unilateral canal paresis. Pang (74) explained the vertigo with the theories of Compere (15). According to the latter author the dysequilibrium was due to an imbalance in the central processing system because of an alteration of proprioceptive impulses from the deep muscles and tissues of the neck. He also considered that changes of blood circulation in the inner ear based on a disorder of the sympathetic vertebral plexus or a physically obstruction of the vertebral artery could be the cause.

Rubin (85) conducted an ENG examination in 54 whiplash patients. The oculomotor tests were not included in his test battery. His examination revealed a positional nystagmus, usually when the patients were turning their head. The percentage of cases is not given. In 50 % of the cases a reduced unilateral labyrinth response was present. Rubin (85) assumed that the dizziness was due to an inadequate integration of sensory

inputs because of an impaired brainstem circulation.

Toglia (92) performed ENG without registration of oculomotor tests in 309 patients complaining of disequilibrium, lightheadedness and positional vertigo, mostly after rapid head movements. In 29 % of the patients a latent(= spontaneous) nystagmus was registrated. A positional nystagmus, induced by adopting several head positions, was only seen in a small group and usually disappeared a few weeks or months after the accident. A unilateral canal paresis was seen in 40%, which decreased to less than 22% when the test was repeated more than one year after the accident. Toglia (92) mentioned that abnormal vestibular test results are more frequent in patients with whiplash injuries than in those with closed head injuries. He ascribed the vertigo to disorders of sensory prioprioceptive cervical spinal afferents, to cervical sympathetic irritation or to vertebrobasilar artery insufficiency.

Hinoki (41) conducted ENG in several groups of whiplash patients. He applied not only a caloric test, but he performed also the optokinetic nystagmus test (OKN). In this test a recording is made of the patient's eye movements as he watched an optokinetic stimulus moving horizontally, first to the left and then to the right. Normally the speed of the eyes during the nystagmus slow phase matches the speed of the stimulus, up to a stimulus speed of approximately 30°/sec. Hinoki (41) observed several groups of whiplash patients. He described a unilateral labyrinth dysfunction in only eight out of 136 whiplash patients. In 26 out of 46 whiplash patients pulse stimulation to tender spots of the nuchal region revealed an increase of vertigo complaints and a reduction of the slow phase velocity (SPV) of the OKN. Furthermore, Hinoki (41) discovered a parallel of the severity of lumbar pain with the degree of vertigo during treatment in 33 out of 200 whiplash patients. Nine patients suffering from vertigo and severe lumbar pain, but no or little neck pain, showed also a decreased SPV of OKN. The SPV normalised by wearing a corset resulting also in a diminishing of the vertigo complaints.

This author (41) assumed that after a whiplash injury overexcitation of the cervical and lumbar proprioceptors act as a trigger where the central nervous system acts as a target. He postulated that the cervical proprioceptors, about 60% of all proprioceptive



sensors in the human body (3), also act as a trigger with the labyrinths, including the vestibular nuclei as a target.

Chester (14) examined 48 patients with chronic pain after a rear-end car collision seven months to seven years before. From this group of patients 27 had fibromyalgia, but the author did not mention when this diagnosis was made: before or after the car accident. In another 27 of these patients caloric irrigation and positional tests were done. In nine of them a unilateral labyrinthine weakness was registered. The positional test revealed a nystagmus on head changing position in 18 patients, while in 16 patients were showing benign positional nystagmus, BPPN. Seven patients had undergone surgery to correct fistulas.

Oculomotor screening test were conducted in 29 patients without a description which test battery was conducted. In two patients abnormalities were seen. In one patient abnormal visual pursuit. The second patient was unable to maintain gaze fixation. It is remarkable that 17 patients with fibromyalgia had normal oculomotor tests, because in patients suffering from fibromyalgia abnormal ocular motor function is frequently observed. Especially with regard to saccadic eye movements and smooth pursuit (84).

Moving platform posturography was performed in all 48 patients. In 20 of them an inappropriate motor response strategies to sensory interaction abnormalities were diagnosed. Because of the incomplete mentioning of the patients' medical history, contradictions in the data described in the article and the tables and the change of test conduction in the patients, it is impossible to draw conclusions from the report of Chester (14).

Roos (83) observed during electronystagmography vestibular dysfunctions in 5% of 85 patients with a neck trauma. His test battery consisted of the registration of spontaneous and positional nystagmus, the caloric test and oculomotor tests, including gaze test, OKN, smooth pursuit and the fixation suppression test. Furthermore, he examined his patients in a tilting room. The results of measurement of the subjective horizontal with the head in lateroflexion were out of normal range in 18% of the cases. Stabilometry revealed that extension of the neck proved to have a negative effect on posture. There was an increased antero-posterior and left-right body sway. With regard to left-right

bodysway (mm) dynamic tilting room examination showed that the 90th percentile of the control subjects was exceeded by about 47/46 percent of the patients for the stimulus frequencies of 0.025, 0.05, 0.1 and 0.2 Hz respectively. The author (83) concluded that the relative weight of sensory information had changed in favour of vision. The same finding was done in patients with an unilateral labyrinthine loss of function after surgery because of an acoustic neurinoma. Roos (83) assumed that lesions of the afferent neck proprioceptor explained the findings in his patients with a neck trauma, because he excluded the patients with disorders in their visual-ocular control, in spite of the assumption that these disorders could be a result of the neck trauma.

Oosterveld et al (72) and Kortschot and Oosterveld (53) performed electronystagmography in patients involved in rear-end car collisions. A complete review of the results in 552 patients wil be given in chapter three. These authors (72,53) registered unilateral labyrinthine weakness in 3 % of their cases, a cervical nystagmus/ influence in 33% and a central-vestibular dysfunction in 53% of their cases. They supposed that the central-vestibular dysfunctions originated either from a direct effect of the trauma on the central nervous system or from an indirect effect because of a changed proprioceptive input of the cervical spine.

Ettlin et al (29) performed ENG in 18 patients within two weeks after the accident. They presumed a peripheral vestibular deficit in case of registration of horizontal spontaneous nystagmus, canal paresis of more than 30% or asymmetry in the rotation chair test and a central vestibular deficit in case of registration of vertical, rotatory or dissociated spontaneous nystagmus, low gain in OKN, low gain or saccades in the smooth pursuit, deficient fixation suppression or pathological, but symmetrical brainstem auditory responses. Six patients had cervicogenic nystagmus. One of them with peripheral vestibular dysfunction and another one with central vestibular dysfunction. Three other patients had central vestibular dysfunction and one patient had only a peripheral vestibular dysfunction. The authors (29) considered that the accident caused amongst others functional abnormalities in the vestibular nuclei, but they did not mention the aetiology.

Serra et al (89) performed ENG in 120 patients suffering from the after-effects of a



rear-end, frontal or lateral car collision within 14 days after the accident. The examination was repeated 6, 12 and 24 months after the accident. ENG revealed a cervicogenic nystagmus in 10 patients, central vestibular dysfunction in 38 patients and peripheral vestibular dysfunction in 36 other patients. The investigators (89) did also BAEP registration. They demonstrated a significant positive correlation between central vestibular dysfunction and bilateral BAEP changes. Furthermore, pathological EMG changes were recorded. The authors (89) ascribed these changes to the consequence of over-excitation of the cervical proprioceptors and dysfunction of the central nervous system. They (89) as other investigators (45,76) reported about EEG abnormalities. Serra et al (89) observed focal EEG slowing in 36 patients, occipital focus in 16, parieto-occipital focus in 12 and temporal-parietal focus in 8 patients. After six months these foci were still present in 22 patients.

The authors (89) concluded that all functional abnormalities of their patients could be mainly considered as an organic brain dysfunction resulting from a disturbance in vestibular nuclei, reticular formation and midbrain.

An overview of the mechanism considered to play a role in the origin of neuro-otological dysfunctions is given in table 2.

| Authors | ENG Findings | Explanation |
|---|---|---|
| Pang (74) | spontaneous nystagmus<br>positional nystagmus<br>unilateral labyrinthine weakness<br>no oculomotor test | vascular mechanism in inner ear because of sympathetic disorders or vertebral artery obstruction |
| Rubin (85) | positional nystagmus after head movements<br>unilateral labyrinthine weakness<br>no oculomotor tests | vascular mechanism resulting in adequate integration of sensory input on brainstem level |
| Toglia (92) | spontaneous nystagmus<br>positional nystagmus after head movements<br>unilateral labyrinthine weakness | cervical proprioceptive disorders<br>cervical sympathetic irritation<br>vertebrobasilar artery insuf |

| | | |
|---|---|---|
| | no oculomotor tests | ficiency |
| Hinoki (41) | unilateral labyrinthine weakness<br>reduction of slow phase velocity OKN | overexcitation cervical and lumbal proprioceptive triggers CNS and labyrinths |
| Hildingson et al (40) | only oculomotor tests<br>reduced gain of smooth pursuit<br>reduced eye movement velocities<br>prolonged latencies saccade test | direct brainstem lesions<br>indirect dysfunctions because of affection of cervical proprioceptors |
| Serra et al (89) | cervicogenic nystagmus<br>unilateral labyrinthine weakness<br>central vestibular dysfunction | organic brain dysfunction because of disturbance in vestibular nuclei, reticular formation and midbrain |
| Oosterveld and Kortschot (72, 53) | spontaneous nystagmus<br>cervicogenic nystagmus<br>gaze nystagmus<br>impaired smooth pursuit<br>impaired fixation suppression during rotation<br>prolonged latencies saccade test<br>reduced eye movement velocities | direct CNS dysfunction because of neurotransmitter changes or axonal injuries<br><br>indirect CNS dysfunction because of change of cervical proprioceptive input |

Table 2. Probable origin of neuro-otological dysfunctions.

**Neuropsychological reports**

Gay and Abbott (34) reported about poor concentration in patients with whiplash injury of the neck.

Fischer (31) described amnesia in a 67-year-old woman involved a rear-end car collision, while sitting on the right front seat of a stopped car and wearing her seat belt. The driver of the car saw her passenger moving forward and backward in her seat without striking her head. She observed that her passenger, though keeping alert, immediately became confused and amnestic. Furthermore, this woman complained of



pain in the chest and upper back on breathing. General medical and neurological examination revealed no abnormalities, except a disorientation in time and place and a horizontal nystagmus on conjugate lateral gaze. The next day the patient was dizzy while turning in bed, but the nystagmus had disappeared. The patient was oriented in time , but she could not recall the previous three days. After three days she still could not remember the details of these days. Besides, she complained of palpitations and an ECG revealed an atrial flutter. X-ray examination demonstrated a compression fracture of Th5, while no abnormalities of the cervical spine were seen. The next day her memory became normal, but afterwards she never could remember the details of the first two days after the accident. One month after the accident her dizziness disappeared spontaneously. Fischer (31) supposed that the brain dysfunction in this patient was due to shear strains because of rotation of the brain within the skull resulting in disturbance of the hippocampal-fornical system. A vertebral artery insufficiency seemed less likely because the patient had no retention of alertness, no vision problems and no brainstem deficits.

Olsnes (70) examined 34 patients six to 18 months after they had been involved in a car collision, most of them in rear-end car collisions. These patients were matched with 21 nonhospitalized patients with disabling cervico-brachialgia without a history of trauma. The whiplash patients scored worse on four out of 48 neuropsychological test variables. So Olsnes concluded that the whiplash patients were not impaired much in their test performance as compared to patients with chronic neck pain not caused by an injury. Besides, he mentioned that neurobehavioral deficit seemed of secondary importance to most patients. According to Olsnes (70) his test result neither gives evidence for nor exclude the possibility of brain damage.

Ettlin et al (28) illustrated the importance of a neuropsychological examination with their case history of a couple who were involved in the same rear end car collision two years ago. The husband was the driver and his wife sat in the front seat. Both patients had complaints of head- and neck pain, sleep problems, vertigo, and problems with memory, concentration and attention. A neurological and neuropsychological examination was done. The examinations revealed signs of a neurotic-conversive behaviour in the

wife and deficits of memory, concentration and attention in her husband. The case reports illustrate the controversy on the organic versus psychogenic cause of cerebral dysfunction following whiplash injury of the neck. Nevertheless the authors (28) supposed from test results in other patients that the neuropsychological deficits due to whiplash injuries probably are located on a subcortical level.

Kischka et al (51) studied retrospectively the reports of 52 patients with pure whiplash injury by excluding patients with an additional head trauma or evidence of a cervical herniated disc. Three to 11 years after the accident fifty percent of these patients suffered from cognitive dysfunctions. Eighteen of them were examined with a neuropsychological test battery with regard to attention and concentration, memory and higher cognitive functions. They were matched with healthy control subjects on gender, age, education and professional level. The patients had significant worse test results in concentration and attention, but hardly in memory and in the higher cognitive functions. In case of a psychogenic cause the higher cognitive functions should have been impaired. Kischka et al (51) assumed that their findings support the hypothesis that an organic damage to frontobasal cerebral structures and the upper brainstem, sites of reticular formation and its projections caused the neuropsychological symptoms. They explained the damage to the central nervous system structures by intracranial acceleration forces, because the brain is thrown against the inner surface of the skull and the brainstem, which lies in the axis of the rotatory movement of the head, is exposed to high compression, tearing and shearing forces.

Keidel et al (48) reported about neuropsychological deficits due to whiplash injury in 30 patients with an acute cervico-cephalic syndrome. They investigated their patients for the first time within 14 days after the accident. The patients were included into the study only if they did not suffer from a direct neck trauma or from a head trauma. In case of pretraumatic neurologic -, psychiatric disorders or headache they were also excluded. None of the patients had neurological deficits. In the acute phase attention, concentration, cognition, verbal and visual memory functions were below the patients' normal level. Attention and concentration recovered within six weeks. Deficits in visual memory, imagination and analytic capacity recovered within the next six weeks. But



cognitive selectivity, information processing speed and the capability of verbal memory and abstraction were impaired for a longer time. No differences in recovery were observed between women and men. The authors (48) suggest that the neuropsychological deficits can be caused by direct damage of structures or functions of the central nervous system, by secundary reaction and psychovegetative dysfunctions due to a primary cervico-cephalic pain syndrome, by both functional-somatic and organic deficits or by a secondary reaction to subjective experience and objective deficit. They also state that in case of an acute whiplash injury the diagnosis of a neurotic or a pseudoneurasthenic syndrome should be made with caution.

Ettlin et al (29) reported about 21 patients, examined within one day after the accident. None of the patients had a direct head trauma. Most subjects, 15, were involved in a rear-end collision, the other ones in lateral and head-on collisions. Immediately after the accident five patients had a reduced concentration, while 13 other patients had concentration and memory problems within three days after the accident. Sleep disturbances were mentioned by 18 patients within three days after the accident. Twenty out of 21 patients showed a higher motor reaction time and 15 subjects performed poorer in digit span, concentration test, sustained attention test Stroop II and Wechsler's mental control subtest "ABC" as compared to control subjects, matched with respect to sex, age and education. Three months after the accident two out of 14 retested subjects showed results equal to their controls with respect to their initial attentional deficits. In ten patients the results were improved, but in two the results were worsed. With regard to 13 patients with initial concentration deficits eight were improved, four unchanged and one deteriorated. Three patients were taking medication, diclofenac sodium or ibuprofen. One of them showed improvement, one no changes and one poorer results as compared to the first examination. The neuropsychological tests were repeated after one year in two and after two years in another two patients. The results were unchanged. They still had a cognitive dysfunction. These four patients are part of the five which were still partially disabled, because the cognitive dysfunctions interfered with their performance at work. The authors (29) postulated that pain could be a probable factor in causing cognitive impairmant in some of their patients and

the use of medication played a minor role in their patients. They excluded secondary gain because of the absence of inhomogenic test results and the fact that the neuropsychological impairment was limited to psychomotor functions and the higher cognitive functions were not affected. Ettlin et al(29) ascribed the cerebral symptoms to organic brain dysfunction. The findings suggested possible damage to basal frontal and upper brainstem structures, reticular formation and its connections, sites of limbic and hypothalamic connections.

Bohnen et al (11) reported about two women with cognitive and neuro-otological dysfunctions after rear-end car collisions.

The first patient was examined 23 months after a rear-end car collision. She complained amongst others about cervical stiffness, dizzines, decreased libido, increased irritability, difficulties in reading and organizing. Electronystagmographic examination revealed a bilateral gaze nystagmus and a cervicogenic nystagmus. The neuropsychological tests showed a deficient acquisition and retrieval and an increased sensitivity to interference. The performance of complex information processing tasks was extremely slow. The patient compensated her reduced speed of information processing by working more carefully by an increased use of mental effort leading to a rapid development of fatigue. The second patient was also involved in a rear-end car collision. Immediately after the accident she felt nauseous and dizzy and she had paresthesias in the right arm and leg. Resuming her duty after six months she complained of blurred vision and an increase of sleep disturbances and cognitive problems. Physical and radiological examinations showed no abnormalities. Electronystagmography revealed disturbances of saccadic eye movements and a positional nystagmus increasing by cervical provocation. The neuropsychological examination demonstrated deficits on memory tasks, slowing on speed tests. She had a decreased rate of information processing.

These investigators (11) assumed a relation between the cognitive problems and a dysfunction of both the subcortical and orbital prefrontal cortex because of a neuronal damage due to a different angle of rotation, duration and direction of force.

A prospective clinical study, including neuropsychological testing, goes on in Switzerland at the University of Bern (81,88). These patients are referred and examined as



soon as possible after the trauma. After the baseline examination follow-up examinations are performed at three, six and twelve months. The investigators included only patients aged less than 55 years, following a trauma causing cervical musculo-ligamental sprain or strain due to hyperflexion/hyperextension without cervical fractures or dislocations, head trauma or alteration of consciousness. The patients had to speak German as native language and did not have a history of persistent neurological dysfunction. The 117 patients, 58% females, were involved in car accidents, from which 60% rear-end collisions. One year after the accident 76% of the patients were recovered. The others still suffered from symptoms, but only 5% were disabled. Persisting symptoms a year after the accident were related with higher age, previous history of head trauma, pre-traumatic headache, rotated or inclined head position at the moment of the impact, intensity of initial neck pain and headache, sleep disturbances immediately after the accident, symptoms of radicular irritation, score on scale nervousness from the personality inventory, score on speed of information processing and poor concentration (81). Delayed recovery was related with the severity of the initial injury. Especially a higher intensity of initial neck pain and symptoms of radicular irritation are prognostic signs. The higher score on the nervousness scale, which can be observed during a somatic illness, was ascribed to pain.

With regard to the neuropsychological test results Di Stefano and Radanov (88) mentioned that due to the speed of information processing (Trailmaking test part B and Pasat) the results were poorer and recovered less in the symptomatic group. They assumed this phenomenon could be explained in part by utilized medication and the higher age.

An overview of the mechanisms and the localisation of the neuropsychological deficits suggested by the several investigators is given in table 3.

| Authors | Neuropsychologic signs | Explanation |
|---|---|---|
| Fischer (31) | alert, confused, amnestic, disorientation in time and place | dysfunction of hippocampal-fornical system by shear strains because of brain rotation within skull |

| | | |
|---|---|---|
| Kischka et al (51) | impaired concentration<br>attention deficits<br>normal higher cognitive functions | organic damage frontobasal structures, reticular formation and upper brainstem by compression, shearing, tearing forces due to rotation of brain within skull |
| Keidel et al (48) | impaired attention, concentration and visual memory in acute phase<br>impaired information processing speed and verbal memory after six weeks | direct brain dysfunction<br>indirect brain dysfunction through cervical pain |
| Ettlin et al (29) | impaired concentration, memory problems and sleep disturbances<br>higher motor reaction time<br>impaired sustained attention | organic brain dysfunction with probable damage to basal frontal and upper brainstem structures, reticular formation and sites of limbic and hypothalamic connections |
| Bohnen et al (11) | deficient acquisition and retrieval<br>increased sensitivity to interference<br>slowing on speed tests<br>decreased rate of information processing | neuronal damage in subcortex and orbital prefrontal cortex by subjection to different angle of rotation during impact |
| Radanov et al (81,88) | impaired speed of information processing | previous history of head trauma<br>pretraumatic headache<br>severity of trauma<br>partially: utilized medication<br>higher age |
| | higher score on nervousness scale | pain |

Table 3. Localisation and presumable mechanism in neuropsychological deficits.



## Imaging of the cervical spine

Although the symptomatology can be profuse, physical and X-ray examination reveal often no pathology. Foreman and Dabco (32) stated that after a trauma a plain radiographic examination is mandatory besides the physical examination and the medical history. To their opinion a minimum of seven radiographs is essential: an AP open mouth radiograph, an AP lower cervical radiograph, a lateral radiograph, lateral flexion and extension and two oblique radiographs. Sometimes further examination with MRI or CT is necessary.

With regard to plain radiography Driscoll et al (22,23) give a systematic description how non-radiologists can analyse cervical spine views and detect signs of ligamental injuries. They emphasize that the C1-C7 vertebrae and the junction of C7 and T1 should be visible. Furthermore alignment, bones, cartilage and joints and soft tissue should be checked.

Golding (35) postulated that in case of persistence of symptoms also the involvement of the thoracic spine should be excluded.

Dvorák et al (25, 26) discovered differences between healthy subjects and patients with respect to functional flexion/extension radiographs. In 1988 these authors (25) reported about the different results of active and passive functional radiograms. During radiography the subjects have to stand in upright position with their head in neutral position and their shoulders hanging down as low as possible to show the cervical-thoracic junction. The mid-thoracic spine and the sternum have to be fixed between two pellots to prevent flexion or extension in the thoracic spine. After the subjects were placed in position first active functional radiographs were taken and next the passive cervical flexion and extension was examined. Afterwards the flexion and extension images of the lower vertebra are superimposed. The remaining displacement between the two images of the upper vertebra represents the segmental motion of the level or the motion relative to the fixed position of the lower vertebra. Using the passive examination as compared to the active examination the authors (25) discovered more hypermobile segments in patients, because the passive examinations resulted in a greater range of motions. Their study of 1993 revealed a trend to hypermobility in the upper and middle

cervical levels as compared to healthy control subjects (26).

However, small nondisplaced fractures can easily be overlooked.

Jónsson et al (46) studied the cervical spines from 22 traffic accident victims with lethal skull or skull base fractures. After the autopsy the cervical spines with soft tissue were frozen in situ. The specimens were screened with fluoroscopy and radiography. In six specimens additional CT-scans were made. After serial section of the specimens with a cryomicrotome 245 bone and discoligamentous lesions were demonstrated, while these were not detected on the radiograms. Only four of them were seen at second-look evaluation. The authors (46) concluded that plain radiograms fail to show fractures and soft-tissue lesions and they assumed that simular injuries also could be overlooked in clinical situations.

Functional CT-scanning of the upper cervical spine, seemed to be a method to detect a hypermobility at the upper cervical level due to a disruption of the alar ligaments (27,2).

Hall et al (38) reported that MRI in acute cervical trauma cannot only demonstrate cord oedema, contusions and acute disc hernations, but also reveals locking of facets and disruption of the posterior longitudinal ligament.

Petterson et al (79) examined 39 whiplash patients. Most of them were involved in front- or rear-end collisions. After the physical examination radiographs of the cervical spine were obtained. Within 15 days after the accident a neurological examination and a MRI was performed. MRI revealed changes in 26 patients, whereas 22 of them had also neurological signs. Out of 26 subjects four had foramina stenosis, while 25 of them had disc lesions. Ten patients had disc herniation, resulting in dura impingement in 7 patients and in medullary impingement in three of them.

Out of 39 patients 29 had neurological signs, including seven out of the ten subjects with disc herniation, but only in three patients the clinical signs and the MRI findings were in agreement. So only in three patients the segmental radicular pain and the sensitivity disturbances corresponded with the affected level on the MRI scan. After six months eight patients still had neurological deficits. The authors (79) state that their patients following whiplash injuries have a higher frequency of disc herniations on MRI



(25%) than asymptomatic subjects (10%), investigated with MRI by Boden et al (9). Petterson et al (79) presume that it is not necessary to investigate patients with MRI early after their injury, because the MRI findings did not reveal significant soft-tissue lesions explaining the symptoms and physical findings. This assumption is not in accordance with the findings of Jónsson et al (47). These investigators reported about clinical and imaging findings in 50 patients suffering from whiplash-type neck distorsions. All had a clinical examination at their emergency department. Radiographic examination was done in all subjects with plain lateral, antero-posterior and 45° oblique views. After six weeks 26 out of 50 patients had completely recovered and had resumed all their activities, while after five years 19 of them were still completely asymptomatic. Six others had minor neckstiffness, tenderness and tiredness, so they needed occasionally physiotherapy. Only one of this group was on sick leave because of "bizarre vertigo", dizziness and headache.

Nineteen out of the 24 patients, who were symptomatic after six weeks, had radiating pain. Six of them mentioned radiating pain at the first visit. Although flexion and extension views showed no abnormalities, magnetic resonance images with contrast were made. In 13 patients the MRI revealed disc protrusions on the T1-weighted sagittal images. The protrusions were graded as grade 0, when a straight contour of the posterior annulus fibrosus was seen, which was in line with the posterior walls of the adjacent vertebrae. Small and medium protrusions of the posterior annulus fibrosus that obliterate one-third or two-third of the combined anterior epidural and subarachnoid space were defined as grade 1 and 2. Grade 3 protrusions push the spinal cord posteriorly and obliterate all epidural and subarachnoid spaces, while grade 4 protrusions compress and indent the spinal cord anteriorly. On the T2-weighted images disc signal intensities were classified as normal or reduced. Most patients with persisting severe radiating pain had large disc protrusions on MRI. These findings were confirmed as herniations on surgery. After five years all 24 patients, symptomatic after six weeks, complained of neck pain and they had been treated with physiotherapy and chiropraxy again and again. They were on sick leave repeatedly.

Jónsson et al (47) postulated that plain radiography is still the prime imaging modality

in distortion accidents involving the cervical spine. Functional flexion/extension views demonstrated posterior soft-tissue ruptures and segmental instabilities in patients with persistent neck pain (25), while MRI six weeks after the accident showed not any of the posterior injuries in these patients although flexion/extension radiograms demonstrated abnormal posterior gaping. These findings were confirmed at surgery as fresh soft-tissue ruptures or scar tissue.

The radiating pain of most patients, 13 out of 19, developed at well-defined segmental levels. The authors (47) ascribed the poor correlation between radiating pain and MRI abnormalities in several adjacent levels to multiple level injuries. Innervation of discs by sinuvertebral nerves one or two levels above their entrance (10) could be the explanation for radiating pain and disc protrusions above the symptomatic level. Furthermore, not only local but also referred pain can originate from the zygapophysial joints (5).

Beneliyahu (7) described three patients complaining of neck pain with radiation and tingling into the upper extremities, two due to rear-end and one to front-end collision. Infrared thermography revealed thermal asymmetry. The MRI showed cervical disc herniations. These findings were in accordance with the neurophysiologic, electro-diagnostic and thermographic test results. According to Beneliyahu (7) thermographic imaging can also be helpful to observe the pathoneurophysiology of cervical disc herniation.

Conclusion

Plain radiography is still the best and cheapest screening examination in patients with neck distortions due to a car collision. An AP open mouth, an AP lower cervical and a lateral cervical radiograph in neutral position are sufficient, when the first physical examination demonstrates no neurological deficits. In case of persisting neck pain within six weeks functional flexion/extension radiograms are indicated.

It has to be emphasized that only technical adequate, complete radiographs and a conscientious diagnosis of these radiograms are necessary to reach a maximum reliability of this screening method, resulting in the prevention of misdiagnosis with sometimes catastrophical consequences for the patients.



CT scanning is valuable in case of bony injuries, while functional CT scanning can reveal upper cervical spine instability.

MRI scanning is necessary in case of spinal cord involvement.

**Therapy and settlement**

Other important aspects of neck distortions as therapy and settlement will not be reviewed in this thesis.

However, it is clear that not any specific therapy is succesful in chronic neck pain (5), while in acute neck pain ice applications in the first twenty hours and after a few days the instruction of exercises of the cervical spine within the limits of pain, while the neck is supported for the other time, are assumed to give the best chance of complete recovery (55,56,65,66,93). But the existence of patients with chronic complaints demonstrates that not any therapy has prevented the development of chronic symptoms in some patients.

Although some authors (67) assume that complaints of pain are made in order to secure financial gain, several reports describing the persistence of complaints and symptoms two to 15 years after settlement contradict the assumption of litigation neurosis in these patients (33,42,59,60,75).

## III Localization of neuro-otological dysfunctions.

The review of literature with respect to electronystagmographic examinations suggests the existence of three origins of neuro-otological dysfunctions in patients involved in rear-end car collisions: the labyrinth (unilateral weakness), the neck (cervical nystagmus/influence) and the central nervous system (central-vestibular dysfunction).

According to Hinoki (41) cervical proprioceptors can act as a trigger with the labyrinths and the vestibular nuclei as targets. Also Neuhuber and Bankoul (68) consider that a cervical-vestibulo-cervical loop has a significant role in the pathogenis of cervical disorders of equilibrium. Proprioceptive neck afferents project directly to the medial vestibular nucleus, where they converge with oculomotor and labyrinthine input. These proprioceptive afferents project also to the external and main cuneate nuclei, communicating on their turn with cerebellum, thalamus and cortex, and to the perihypoglossal nuclei, which are related to the vestibulo-oculomotor-cerebellar system. Neuhuber and Zenker (69) demonstrated in rats that mainly the upper cervical afferents, C2 to C4, project to the caudal half of the medial vestibular nuclei. Furthermore, they report that neurons in this region project to the C1 to C4 segments, while neurons in the upper half of the medial vestibular nucleus project to lower cervical and thoracic segments. These authors (69) suggest that direct projections from the upper cervical ganglia are linked closely to the vestibulospinal neurons projecting to the corresponding levels of spinal cord. The vestibulospinal neurons project to the ventral horn and only a small part of them to the dorsal horn of the spinal cord, where also muscle and skin afferents are connecting. Jam of information from these muscle and skin afferents because of inflammation can influence the vestibular nuclei, which regulate this information by means of the vestibulospinal neurons to the dorsal horn. In case of extreme jam of information this regulation is disturbed. Neuhuber and Bankoul (68) assume that this disregulation can cause dizziness in patients.

### Symptoms and signs

It is well-known that occupants of a car involved in a rear-end collision can develop a

variety of complaints. Symptoms can be related to:

1. Locomotor/musculoskeletal system

   Neck pain, headache, shoulder/arm pain, chest pain, backache, pain in the legs, cranio-mandibular dysfunction

   Wasting

   Decreased motor control e.g. dropping objects

2. Sensory perception

   Paraesthesia and hypoesthesia

   Dizziness and/or balance disorders

   Changes of taste and smell

   Lump in throat, swallowing discomfort

   Blurring of vision, problems estimating distances

   Light- and noise hypersensitivity

   Tinnitus, hearing loss, earache

3. Autonomic nervous system

   Nausea, vomiting

   Perspiration

4. Neuro-endocrine system

   Irregular periods, sometimes in spite of the use of oral contraceptives

   Loss of sexual desire and impairment of sexual function

5. Problems with automatic actions

   Pooring tea next to the cup

   Missing one's hold

6. Cognition

   Mental sluggishness



   Forgetfulness

   Poor concentration

   Difficulties to follow a conversation

   Problems putting something into words

   Wrong word formation

7. Changes of personality

   General irritability

   Mood changes

8. Vitality

   Fatigue, varying from mild tiredness to exhaustion

   Non- refreshing sleep

   Sleep disturbances: needing more hours of sleep, restless sleep or insomnia

**Onset**

The occupant can have complaints immediately after the collision. But the discomfort can be delayed for hours. According to Balla (4) symptoms can develop even after a week. Hohl (43) and Kischka (51) reported symptom-free periods of 72 hours. Most authors (21,34,59,60) describe that the onset of complaints varies from immediately till 24 hours after the accident.

Because of the possibility of a delay in onset of the complaints Dodson (21) advised to repeat the examination of the patient 12 to 24 hours after the accident, when the first examination was done immediately after the accident.

Gay and Abbott (34) already described the intermittence of the symptoms. Most patients recover. But about 30% of them get chronic symptoms. These patients have also days with more or with less complaints.

Furthermore, our patients reported about development of new symptoms or the appearence of former symptoms after a longer time at the moment their use of mental or physical efforts increased.

**Patients visiting the vestibular department of the AMC**

Until 1994 552 patients, 205 males (37%) and 347 females (63%) involved in rear-end car collisions, visited the vestibular department of the AMC because of complaints of a.o. vertigo, dizziness, headache, neck pain and tinnitus.

Their most common complaints were neck pain (85%), headache (76%), dizziness (70%): spinning sensations were mentioned by 47% and light-headedness and floating sensations in 23% of the patients. Furthermore concentration- and memory problems were mentioned by respectively 55% and 53% of the subjects. Just 21% complained about tinnitus, while 5% mentioned hearing problems.

The neuro-otological investigation was done from six months till five years after the accident. In 361 of the patients the accident was two or more years before the examination in our department.

During the examination by means of ENG DC binocular eye movements were recorded with a Siemens Elema inkjet mingograph till 1994. From 1994 on stimulus control, data acquisition and analysis were done with a Toennius Nystagliner.

**ENG test battery**

**Calibration**

Each test is started with a calibration, so that in each test the slow phase velocity of the recorded nystagmus can be calculated. The patient has to fix alternately on two light dots placed at a visual angle of 20°. It is important to keep the patients alert in all tests.

**Spontaneous nystagmus**

A spontaneous nystagmus is registered in sitting subjects with upright head position and closed eyes looking straight forward. This nystagmus is defined as a "cervical nystagmus", when in three or four head positions a nystagmus is provoked, while no spontaneous nystagmus is present. When merely in one or two head positions a nystagmus can be provoked, while no spontaneous nystagmus is present, the phenomenon is defined as "cervical influence". A spontaneous nystagmus can be present in 2%-3% of



healthy control subjects (58).

**Gaze test**

During the gaze test the patient has to look with open eyes to a light straight ahead, then to a light 30° to the left, back to a light straight ahead, then to a light 30° to the right and again straight ahead. Eye movements are recorded in each position for 30 seconds. The appearance of a nystagmus while the eyes fix the light 30° to the right and the left respectively, means the presence of a bilateral gaze nystagmus. When the nystagmus is only present in the left or the right gaze, it is defined as an unilateral gaze nystagmus, when there is no spontaneous nystagmus with a fast phase to the side of eye deviation.

**Smooth pursuit**

Smooth pursuit is performed by asking the patient to follow in the horizontal plane a sinusoidally moving visual target with a maximum speed of 20°/sec and with a visual angle of 20°. Smooth symmetrical conjugate pursuit is normal. From 1994 on the gain (the quotient of the eye velocity and the target velocity) was calculated. Examination of healthy control subjects showed that the gain has values of $0.98 \pm 0.14$.

**Saccade test**

Till 1994 the saccade test was registered during calibration, while 20° eye movements were performed. The subjects had to make saccades by moving their eyes rapidly from a light 10° left from centered gaze to a light 10° right from centered gaze and back to the left target again. From 1994 on the subjects were instructed to perform 10° eye movements. They had to look to a center light, then to a light 10° to the left, back to the center light, next to a light 10° to the right, back to the center light, again to the light 10° to the left and so on. The maximum latency or reaction time, the interval between the appearance of a light and the onset of the eye movement in normal subjects is 230 msec (54,90). The peak eye movement velocity are calculated. In healthy control subjects the velocity of the lateral eye muscles has to reach a value of 190°/sec and the

velocity of the medial eye muscles has to have a value of 200°/sec.

### Optokinetic test

During this test the patient has to watch an optokinetic stimulus, which moves horizontally, first to the left and then to the right, with different stimulus speeds and a maximum speed of 36°/sec. In normal subjects the responses are symmetrical and there is no declining response to increasing stimulus velocity. Until 1994 the stimulus varied from 20°/sec to 100°/sec. Up to a stimulus speed of approximately 30°/sec the speed of the eyes matches the speed of the stimulus.

### Visual Suppression Test (VST)

During rotation and also during the caloric test the subjects have to fix at a dot straight ahead. In case of rotation it is a small light, which the subject holds in his hand. Performing the VST during rotation the subject has to suppress the rotation nystagmus completely. In case of the caloric test the fixation index, the quotient of slow phase nystagmus velocity with eyes open and eyes closed, has to be less than 50%.

### Caloric test

This test is performed with the patient in supine position, while his head is flexed 30°. The external auditory canal is irrigated with 250 ml water for 30 sec. Each canal is irrigated twice with water of 30° and of 44°. After the irrigation the maximum slow phase velocity (SPV) of the caloric nystagmus is calculated for each response. Unilateral weakness is defined by the amount by which the SPV of the two ears differ, expressed as a percentage of the sum of all four SPV's. An unilateral weakness or vestibular paresis means an asymmetry greater than 25%.

### Test results

Spontaneous nystagmus was observed in 270 (49%) of the patients. Only in four of them the slow phase velocity had values of more than 7°/sec. In 52 cases a cervical nystagmus with a slow phase velocity from 3-7°/sec was present. Cervical influence



was registered in 120 (22%) patients.

Only in 17 (3%) of the patients a unilateral labyrinth weakness was found.

An abnormal smooth pursuit was recorded in 143 (26%), a bilateral gaze nystagmus in 89 (16%), while in 54 (10%) of the cases a unilateral gaze nystagmus was present. The visual suppression test, exercised during the rotation test, revealed disturbances in 209 (38%) of the patients. No central-vestibular dysfunction was found in 252 (47%) of the cases, while 119 (21%) patients had no vestibular disturbances at all.

### Discussion

More females than males suffered from chronic complaints after rear-end car collisions (table 4).

| Authors | Year | N | ♂ | ♀ |
|---|---|---|---|---|
| Chester (14) | 1991 | 48 | 18 | 30 |
| Ettlin (29) | 1992 | 21 | 3 | 18 |
| Gay & Abbott (34) | 1953 | 50 | 15 | 35 |
| Hildingson et al (40) | 1989 | 39 | 17 | 22 |
| Jónsson et al (47) | 1994 | 50 | 17 | 33 |
| Keidel et al (49) | 1994 | 61 | 25 | 36 |
| Kischka et al (51) | 1991 | 52 | 17 | 35 |
| Knibestöl et al (52) | 1990 | 30 | 16 | 14 |
| Kortschot & Oosterveld (53) | 1994 | 462 | 183 | 279 |
| Olsnes (70) | 1989 | 34 | 16 | 18 |
| Pang (74) | 1971 | 20 | 7 | 13 |
| Petterson (79) | 1994 | 39 | 19 | 20 |
| Serra et al (89) | 1994 | 120 | 53 | 67 |

Table 4. Gender incidence.

The phenomenon that the incidence of suffering from the after effects of car collisions is greater in females, has already been reported by Gay and Abbott (34). Later on many other authors confirmed these findings (table 4).

Schutt and Doham (87) assumed that this phenomenon was due to lower cervical muscle strength in women. They also suggested that women were more at the right front seat (1968!) and therefor they were less alerted to an impending collision. The last assumption is in contradiction with the findings of the Quebec Task Force on

Whiplash-Associated Disorders (80). They reported that in Quebec in 1987 the incidence rates among female and male drivers were respectively 126 and 73 per 100.000 licensed drivers per year.
Measurements of the Dutch Confection Center demonstrated that men have a greater circumference of the neck as compared to women suggesting that a greater neckmass plays a preventive role in neck distortion due to car collision, likely because men have more neck musculature than women (80).

Our test results confirm the findings of other authors (29, 89) that three localizations of neuro-otological dysfunctions exist in patients involved in rear-end car collisions: the labyrinth, the neck and the central nervous system. However, our observation that only 57 (10%) patients have cervicalnystagmus/influence and central-vestibular dysfunction opposes the assumption of the cervical proprioceptors as the one and only cause of the central-vestibular dysfunction. Also the reports of some of our patients that fatigue can increase dizziness or unsteadiness and deterioration of pain does not, pleads in favour of a direct central-vestibular dysfunction. Fatigue prolongs the initiation times (= reaction times) of the Motor Choice Reaction Test (MCRT) (86). The neuropsychologists Matlung and Van der Scheer (86) frequently observed in patients involved in car collisions a prolonged initiation time of the MCRT, presumably due to the efforts of undergoing their neuropsychologic test battery. They conducted the MCRT twice, both at the start and the end of their test battery. During the second measurement an increase of the initiation time was observed (86). The time between both tests was at least three hours. Considering the clinical fact that patients report an extraordinary influence of fatigue on their performance combined with observations of Matlung and Van der Scheer (86), it was decided to conduct a study aimed at the effects of the efforts of performing the neuro-otological examination on eye movement velocities and reaction time.



**IV A neuro-otological study aimed at the influence of performing the examination on eye movement velocities and reaction times.**

In this study not only the standard neuro-otological test battery is conducted, but also the effects of the strain of performing the tests on eye movement velocities and reaction times are evaluated. With respect to the reaction time the effect on the latency of the saccades and on the MCRT initiation time are studied. This became possible, because in 1994 the Maatschap Neuropsychologie Matlung & van der Scheer placed for three months at our disposal their equipment to conduct the Motor Choice Reaction Test (MCRT).
All patients suffering from the after effects of a rear-end car collision, forwarded to the Vestibular Department in these three months, were subjected to both an neuro-otological examination and a Motor Choice Reaction Test.
The MCRT test was conducted twice, at the start and at the end of the neuro-otological examination. In this study the time between the two MCRT tests was about one hour, which means two hours less than was maintained during the neuropsychological test battery.

**Design**

Before the examination all patients were subjected to an extensive questionnaire, in which they were asked about the accident history, the complaints immediately after the accident and the delayed ones. The past (medical) history was also documented.
The examination started with the MCRT in three different versions. The subjects had to press a red button on a small square box with their favorite forefinger; so they used their right forefinger, if they were right-handed and the left forefinger in case of left-handedness. Above the red button five white buttons, from which each one had the same distance to the red button, were placed in a semicircle.
In the first version the subject had to press as fast as possible the white button, directly above the red one, as soon as the white one was illuminated. Then they had to push the red button again as soon as possible till the procedure repeated itself. In each version

25 trials were offered. Six of these trials were practised and 19 of them were registered. The pre-illumination time had an interval of 500 or 1500 milliseconds selected at random. The initiation time, the time between the illumination of the white button and the release of the red one, was measured in milliseconds.

During the second version one of the three white buttons above the red one was illuminated. Again the patients were instructed to push as fast as possible on the illuminated white button.

In the third version one of four buttons was enlighted. If the patient was right-handed, he had to push the button at the right side of the illuminated one and in case of left-handedness the button to the left of the illuminated button. In all versions the patient had to push the red button as soon as possible after they had pushed the white one.

Because the third version was the most complicated, it was expected that this one would take the most time both in patients and controls. In each version the mean and median of 19 measurements were calculated.

The neuro-otological examination included registration of vertical smooth pursuit, first measurement of horizontal saccadic eye movements, first registration of horizontal smooth pursuit, fixation, horizontal gazetest, registration of spontaneous nystagmus, horizontal optokinetic test, nystagmus registration in four different head positions, second registration of saccadic eye movements, second measurement of horizontal smooth pursuit test, rotation test and visual suppression test. The caloric test was performed after the second MCRT test. In this group of patients a caloric test was only conducted on the suspicion of peripheral vestibular dysfunction, e.g in case of vertigo complaints, unsteadiness, drop attacks and asymmetric reaction during the rotation test. The horizontal and vertical eye movements were recorded by DC-electronystagmography. Stimulus control, data-acquisition and analysis were done with a Toennies Nystagliner.

Analogous to the MCRT the saccades were examined twice, both at the beginning and at the end of the registration of the horizontal eye movements test battery. Special attention was given to the latency, the reaction time. During the registration of the saccadic eye movements the first 32 seconds of the registration, in which three



movements to the left and three movements to the right are conducted, were considered as practices analogous to the MCRT test. Afterwards the eye movements were registered for 80 seconds so at least 15 measurements were available. From these measurements also the mean and median of the latency were calculated. Also the horizontal smooth pursuit was observed two times (table 5).

| Test battery |
|---|
| MCRT I |
| Vertical smooth pursuit |
| Horizontal saccade test I |
| Horizontal smooth pursuit I |
| Fixation test |
| Horizontal gaze test |
| Spontaneous nystagmus |
| Horizontal optokonetic test |
| Cervical test |
| Horizontal saccade test II |
| Horizontal smooth pursuit II |
| Rotation test |
| Visual suppression test |
| MCRT II |
| Caloric test |

Table 5. Test Battery

## Aim

The aim of the study was to investigate:

1. the localization of the neuro-otological dysfunctions
2. the effect of the efforts of the neuro-otological examination on the duration of the reaction times (= initiation times) of the MCRT.
3. the effect of the conduction of the horizontal eye movements tests on the reaction time, the eye movement velocity of the saccade test and on the gain of smooth pursuit.

## Inclusion criteria

Patients, aged from 20 to 58 years, were included in the study. They had all been

involved in a rear-end car collision. They were matched with control subjects with regard to age, gender and education. The patients were only included, when they had not been unconscious because of the accident. In case of direct head impact on a window or the steering wheel they were also excluded from the study.

The control subjects were only included if they had not had any biological life events at all, because three or more biological life events can influence the initiation times of the MCRT (43). So the control subjects were only included when they had no accidents, concussions, contusions or operations with anaesthesia in their medical history.

After three months 40 patients, 17 males and 23 females, and 40 control subjects could be included in the study.

None of the patients used antidepressants, muscle relaxants or vestibular sedatives. Some of them used pain releaving medication or oral contraceptives.

**Complaints**

All patients were forwarded to the vestibular department because of persistent complaints after a rear-end car collision. One patient was investigated two months after the accident. Four patients had their examination six months after the accident, another 18 one to two years after the accident; in 17 patients the accident had happened more than two years, but less than five years ago.

In 31 patients the complaints started immediately after the accident, in eight of them there was a symptom-free period of 30 minutes to a few hours and only one subject got complaints the next day.

Most patients, 39, complained about neck pain (table 6). Irradiating pain to one or both shoulders and arms existed in 32 of them. Complaints of headache were present in 35 patients. Back pain was mentioned by 25 subjects.

Thirty three patients had feelings of dizziness: vertigo in 13 of them, in another 15 floating sensations and drop attacks in three patients. Sometimes these sensations were accompanied by unsteadiness (eight subjects).

Twenty four patients complained of nausea, but only six of them also vomited. In three patients the vomiting started immediately after the accident and in two the first week



after the accident. This symptom lasted for weeks or several months.

Poor concentration and forgetfulness was mentioned by 35 respectively 34 of the patients. Also 34 of them had vision problems. Nineteen of them were very sensitive to light and noise. Tinnitus was present in 15 patients. Only three of them had hearing impairment. Eight subjects had swallowing difficulties. One patient had changes of his smell as well as his taste, while in another one only a change of taste was mentioned. Change of voice was described only once.

| Complaints | N |
|---|---|
| Neck pain | 39 |
| - irradiating to shoulders and/or arms | 32 |
| Impaired cervical movement | 18 |
| Headache | 35 |
| Back pain | 25 |
| Wasting | 21 |
| Numbness | 28 |
| Dizziness | 33 |
| - Vertigo | 13 |
| - Floating | 15 |
| - Unsteadiness | 8 |
| - Drop attacks | 3 |
| Nausea | 24 |
| Vomiting | 6 |
| Fatigue | 32 |
| - Exhaustion | 4 |
| Poor concentration | 35 |
| Forgetfulness | 34 |
| Vision problems | 34 |
| Sensitivity to light and noise | 19 |
| Hearing impairment | 3 |
| Tinnitus | 15 |
| Difficulty swallowing | 8 |
| Change of smell | 1 |
| Change of taste | 2 |
| Change of personality | 36 |
| Loss of sexual desire | 11 |
| Irregular periods | 3 |

Table 6. Complaints of 40 patients involved in a rear-end car collision

An increased fatigability was mentioned by 32 patients. Most of them have also a non-

refreshing sleep when compared to the period before the accident. The increased fatigability and the non-refreshing sleep causes in some patients exhaustion and an accentuation of other symptoms. Sleep disturbance can be a big problem after an accident. Seventeen patients needed one to five hours more sleep after the accident. Restless sleep is mentioned by many patients.

Changes of personality were mentioned in 36 patients.

Three women had irregular periods after the accident, one of them in spite of the use of oral contraceptives. Loss of sexual desire was mentioned by four males and seven females. An overview of the complaints is given in table 6.

### Statistical analysis

The data were analysed with SPSS PC+. The data were compared by non-parametric tests, Mann-Whitney U and Wilcoxon Matched-pairs Signed-ranks. A two-tailed p level less than or equal to 0.05 was considered to indicate statistical significance. With regard to the repeated measurement of latency of the saccade test and the initiation time of the MCRT analysis of variance was done with the Manova test/ WSFactor for repeated measurements.

### Test results

1. Neuro-otological examination.
2. Repeated measurements of saccade test and horizontal smooth pursuit.
3. Repeated measurements of MCRT.

### Neuro-otological test results

In one patient as well as in one control subject a spontaneous nystagmus was observed and only one patient had a cervical nystagmus. The slow phase velocity of both the spontaneous and the cervical nystagmus did not reach values of five degrees per second.

An unilateral gaze nystagmus was seen in two patients and two control subjects. Eight of the patients and none of the control subjects at all had a bilateral gaze nystagmus



(Mann-Whitney: $p < 0.002$).

The gain of visual pursuit during the first measurement was decreased to pathological values in three patients and in one of the control subjects (table 7).

| Test | Patients N=40 | Control subjects N=40 |
|---|---|---|
| Spontaneous nystagmus | 1 | 1 |
| Cervical nystagmus | 1 | 0 |
| Gaze test | | |
| - unilateral | 2 | 2 |
| - bilateral | 8 | 0 |
| Saccade test I | | |
| - decreased eye movement velocity | 8 | 1 |
| - prolonged latency (mean + median) | 14 | 1 |
| Decreased gain smooth pursuit I | 3 | 1 |
| Impaired OKN | 5 | 0 |
| Impaired VST | 5 | 0 |
| Saccade test II | | |
| - decreased eye movement velocity | 10 | 1 |
| - prolonged latency (mean + median) | 20 | 4 |
| Decreased gain smooth pursuit II | 8 | 0 |
| Caloric test | | |
| - asymmetry | 3 | |
| - symmetry | 8 | |
| - not performed | 29 | 40 |

Table 7. Neuro-otological test results

The horizontal optokinetic test showed a declining response intensity to increasing stimulus velocity in five patients and in none of the control subject (Mann-Whitney: $p < 0.02$). Also five patients and none of the control subjects had problems with the Visual Suppression Test during rotation (Mann-Whitney: $p < 0.02$).

The first saccade test showed a decreased velocity of the lateral recti muscles in eight

patients and in one control subject (table 7). In seven patients the velocity of the left lateral rectus muscle was decreased, while three of them had also a diminished velocity of the right lateral rectus muscle. Another patient and one control subject had only a decrease of the eye movement velocity of the right lateral rectus muscle.

The mean and median of the latency of the first saccade test was prolonged to pathologic values in 14 patients and in one control subject (Mann-Whitney: $p < 0.003$).

The caloric test was only conducted in 11 patients. These patients complained about unsteadiness and drop attacks. In only three of them an asymmetry was found. One of these patients, in whom the examination took place two months after the accident, the test was repeated after another six months. During the second examination the caloric test showed no asymmetry any more. An overview of the results is shown in table 7.

**Repeated measurements of saccade test and gain of horizontal smooth pursuit.**

Saccade test

Only the eye movement velocity of the left lateral rectus muscle during the second saccadic eye movement measurements was different with regard to patients and control subjects (Mann-Whitney: $p < 0.004$). Within the two groups separately there was no significant difference between the eye movement velocities of each muscle with respect to the first and the second test.

The latency was significantly prolonged in patients both during the first (Mann-Whitney: $p < 0.003$) and the second (Mann-Whitney: $p < 0.001$) registration of the saccade test. (fig. 1, fig. 2, fig. 3, fig. 4, appendix A).

In each group separately, patients and control subjects, there is also a difference between the first and the second measurement (fig. 5).

The latency in both groups increased during the second measurement (Wilcoxon Matched-pairs Signed-ranks Test: patients $p < 0.0005$, control subjects $p < 0.004$).

The Manova WSFactors repeated measurements revealed that the latency during the second measurement increased more in patients than in control subjects ($p < 0.009$).

So, the conduction of the horizontal eye movement test battery does increase the reaction times.



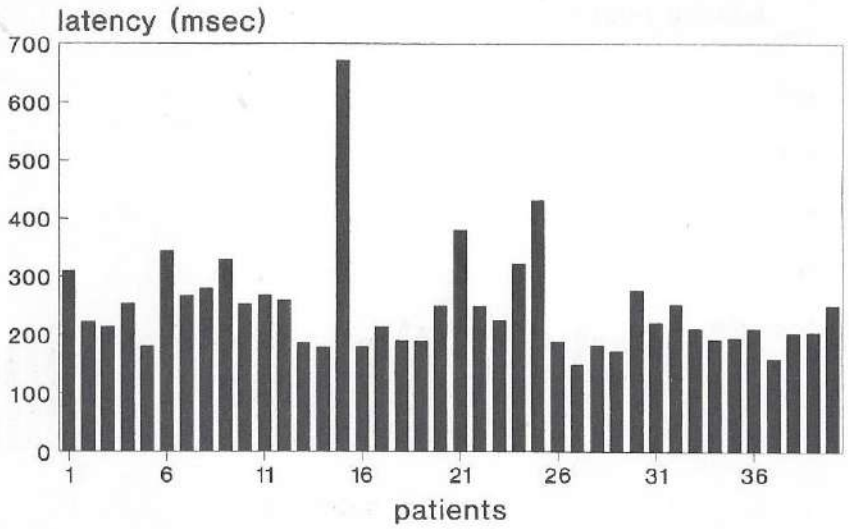


Fig. 1A. Latency of saccade test I in 40 patients. The mean of 15 measurements per patient.

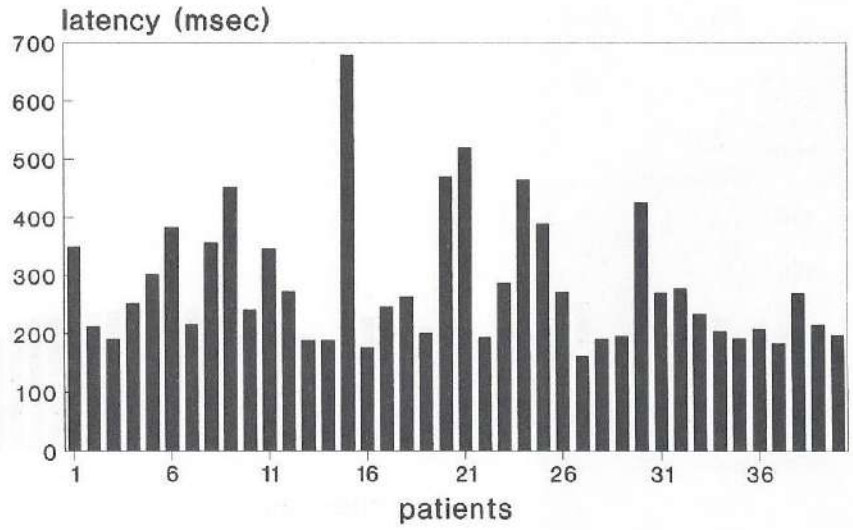


Fig. 1B. Latency of saccade test II in 40 patients. The mean of 15 measurements per patient.

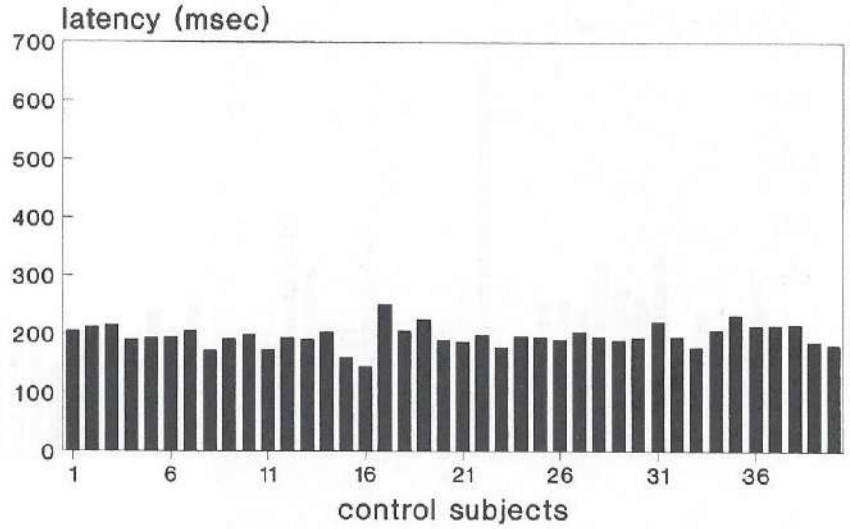


Fig. 2A. Latency of saccade test I in 40 control subjects. The mean of 15 measurements per subject.

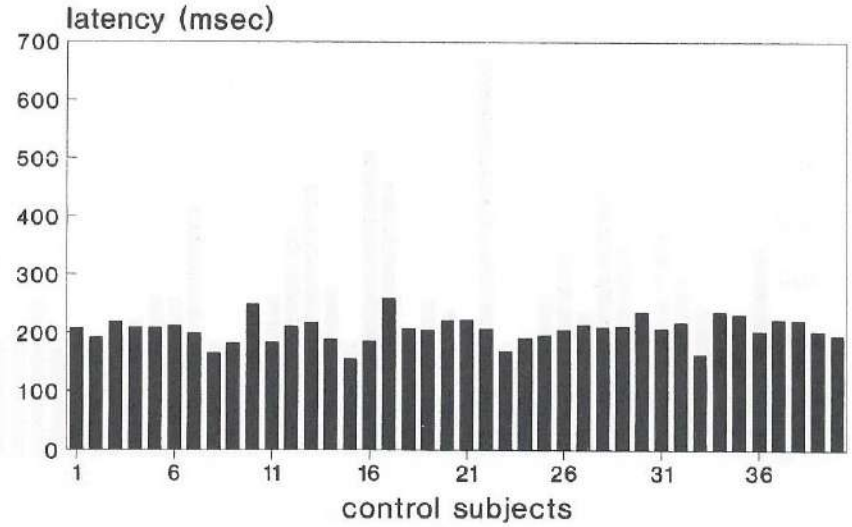


Fig. 2B. Latency of saccade test II in 40 control subjects. The mean of 15 measurements per subject.



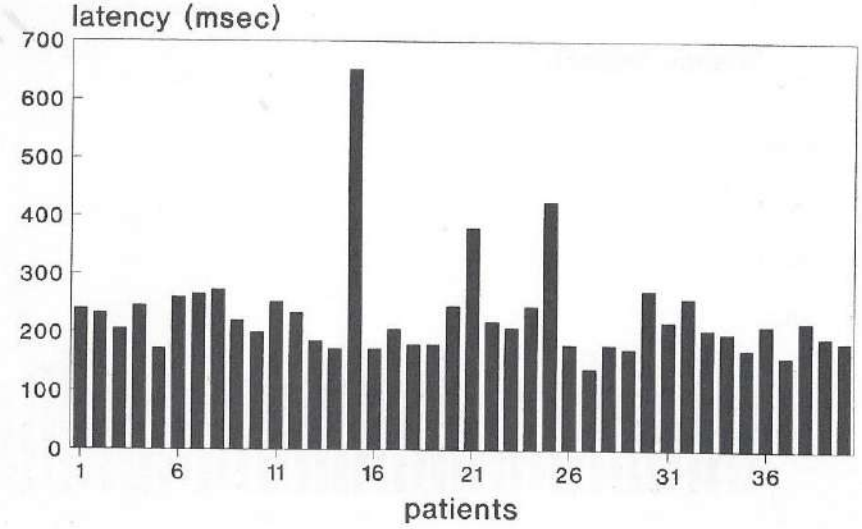


Fig. 3A. Latency of saccade test I in 40 patients. The median of 15 measurements per patient.

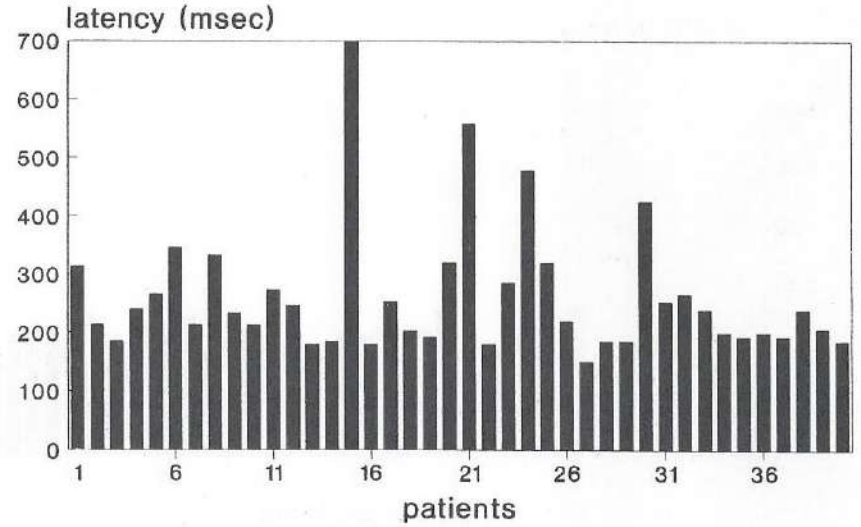


Fig. 3B. Latency of saccade test II in 40 patients. The median of 15 measurements per subject.

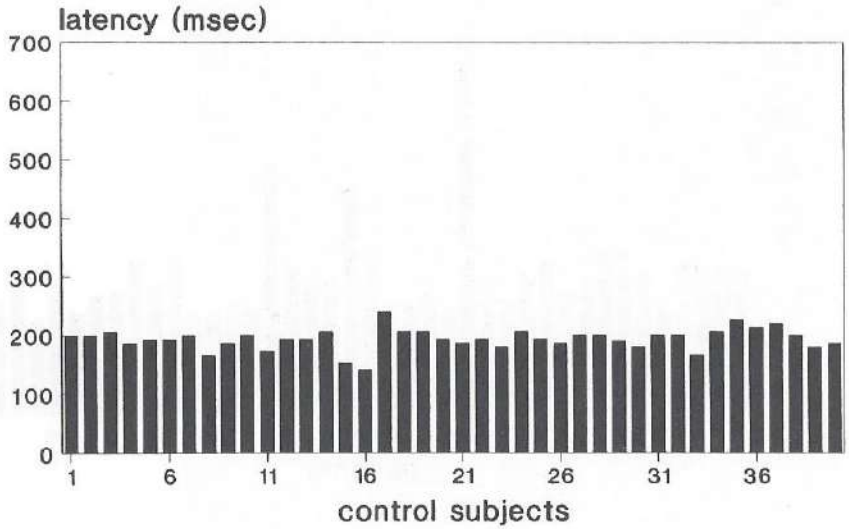


Fig. 4A. Latency of saccade test I in 40 control subjects. The median of 15 measurements per subject.

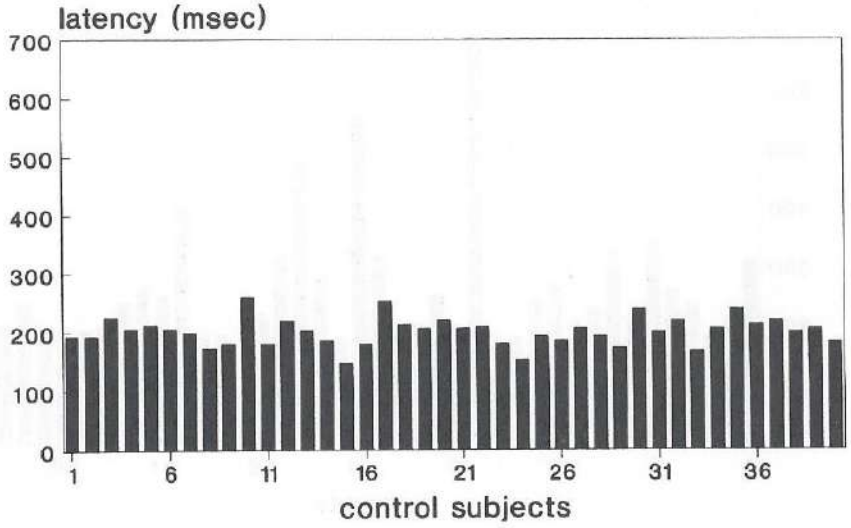


Fig. 4B. Latency of saccade test II in 40 control subjects. The median of 15 measurements per subject.



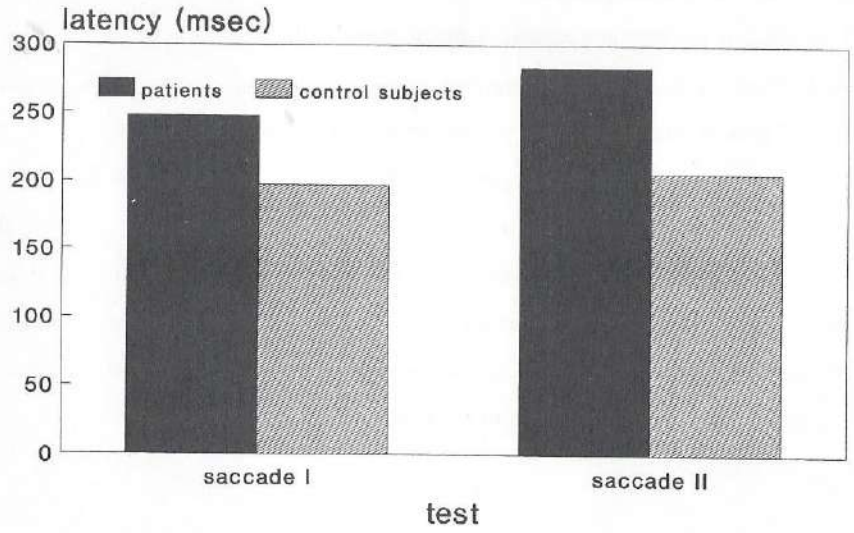


Fig. 5A. Latency of saccade test I and II. Mean in patients and control subjects.

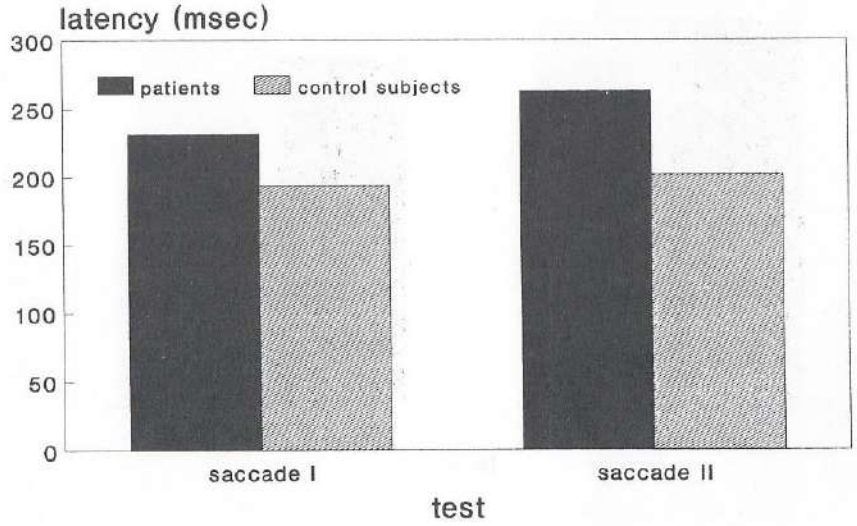


Fig. 5B. Latency of saccade test I and II. Median in patients and control subjects.

Gain of horizontal smooth pursuit

When comparing patients and control subjects there is no difference in gain during the first registration. The second measurement reveals a difference between the groups. A decrease of gain is measured in the patients (Mann-Whitney: $p < 0{,}0008$), although no pathologic value is reached in the group of 40 patients. Only eight patients and one control subject had a pathologically decreased gain. The analysis of the gain was done three times by two investigators. One of them analysed the gain two times, with an interval of six months, in order to obtain only the best gain for comparing the results between both groups (fig 6). The conduction of the horizontal eye movement test battery caused a decrease of the gain of smooth pursuit.

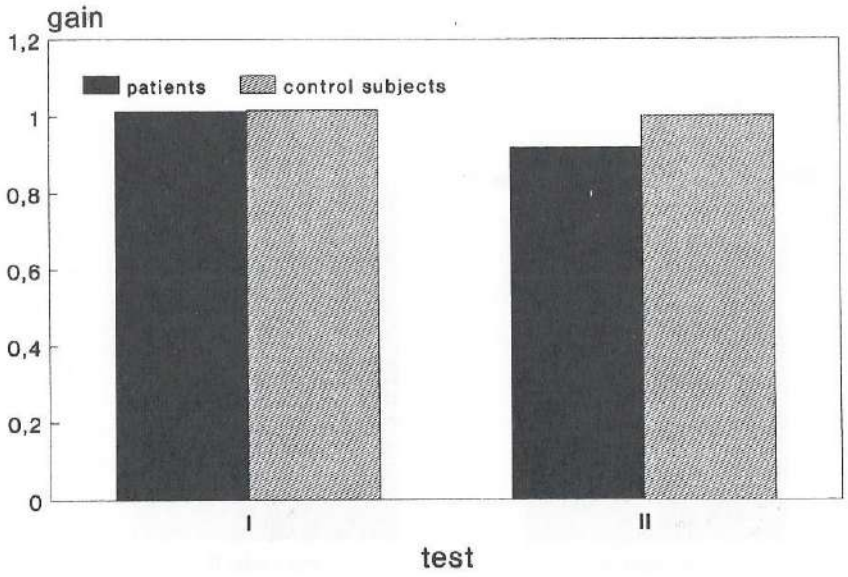


Fig. 6. Smooth pursuit I and II. Gain in patients and control subjects.



**Repeated measurements of MCRT**

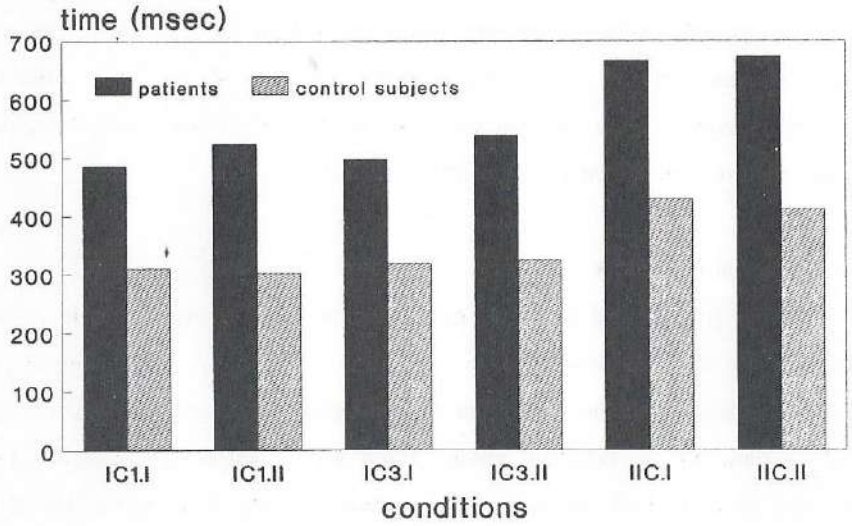


Fig. 7A. Motor Control Reaction Test I and II in three conditions. Mean in patients and control subjects.

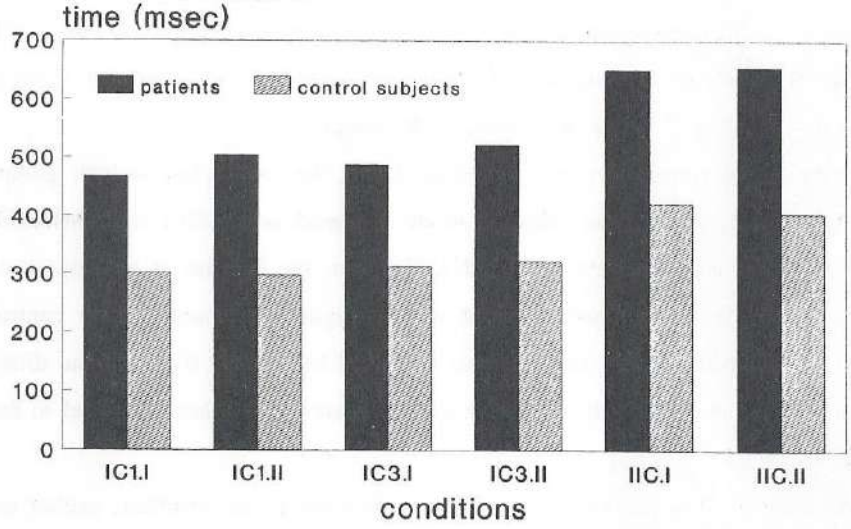


Fig. 7B. Motor Control Reaction Test I and II in three conditions. Median in patients and control subjects.

The initiation times of the MCRT in all three conditions were prolonged in patients (Mann-Whitney: $p < 0.0005$, fig 7).

Within the two groups, neither in patients, nor in the control subjects, an increase of initiation time was measured during the second conduction of the three different conditions when compared to the first one. The conduction of the neuro-otological examination had no effect on the reaction time of the MCRT.

**Effect of biological life events**

Fifteen out of 40 patients had had three or more biological life events, while 25 of them had only one or two of these events.

Analysis of the 25 patients who had one or two biological life events, revealed results anologous to these of the complete group. There was a difference in latency (= reaction time) of both measurements of the saccade test and in initiation time (= reaction time) of the MCRT in all three versions. The patients reacted slower than the control subjects (Mann-Whitney: $p < 0.03$, $p < 0.003$ for the saccade test and $p < 0.0005$ for all conditions of the MCRT). Also a difference was observed in the gaze test, optokinetic test, visual suppression test, second eye movement velocity of the left lateral rectus muscle and second gain of smooth pursuit (Mann-Whitney: $p < 0.03$, $p < 0.01$, $p < 0.03$, $p < 0.002$, $p < 0.03$, respectively).

The latency of the saccade test was increased during the second test in both groups separately, patients and control subjects, when compared to the first test (Wilcoxon matched-pairs Signed-ranks Test: $p < 0.02$). However, the increase of the latency of the second saccade test did not differ in these patients when compared to control subjects. Also in patients with less than two biological life events, there was no difference in gain of smooth pursuit during the second measurement when compared to the first one.

The conduction of the horizontal eye movement test battery had no effect, neither on the eye movement velocities nor on the reaction times in the patients with one or two biological life events in their medical history.



**Discussion**

The results of the neuro-otological test battery reveal again three localisations of neuro-otological dysfunctions: a labyrinthine, a cervical and a central-vestibular localisation.

In our former group of 552 patients only a small percentage (3%) of the patients revealed a labyrinthine dysfunction, while in this study in 7% a labyrinthine dysfunction was observed. One patient, examined for the first time two months after the accident, showed a recovery of the dysfunction during the second examination eight months after the accident.

Meanwhile we have observed more often a recovery of unilateral labyrinthine weakness in acute patients. The same observation was made by Toglia (92). This observation pleads in favour of labyrinthine dysfunction and not labyrinthine damage. According to Hinoki (41) the cervical proprioceptors can act as a trigger not only on the central nervous system, but also on the labyrinth. However, acting as a trigger on the labyrinths is only possible by means of the vestibular nuclei, because to my knowledge no direct pathways are described between the labyrinth and the cervical proprioceptors.

First in all our patients caloric tests were performed. Later on, only in case of suspicion of unilateral labyrinthine weakness. However, in some of our patients we got the clinical observation that the caloric test can act as a trigger to reveal other, non neuro-otological signs and symptoms. For example a patient complaining of short periods of a paretic right arm and aphasia, had such an attack during the caloric irrigation of his right ear. In another patient the irrigation elicited signs she had the first months after the accident: a change of taste, paretic legs, not to be able to sit without help and low back pain. These observations suggest that the conduction of the caloric test can act as a trigger mechanism on the central nervous system and can elicit other non neuro-otological signs. So, we consider to reintroduce the performing of the caloric test in all our patients, because of the importance of the clinical observations that can be made.

Furthermore, this observation pleads in favour of the existence of a central nervous system dysfunction in these patients.

In this study only one out of 40 patients had a cervical nystagmus, while 39 of them

complainted about neck pain. Our group of 552 patients revealed a cervical nystagmus/cervical influence in 31% of the patients, but only in 10% both a cervical nystagmus/influence and a central vestibular dysfunction was observed, while 53 % of them had a central-vestibular dysfunction. This opposes the opinion that all central-vestibular dysfunctions can be explained by the effect of the cervical proprioceptors on the central-vestibular system (41).

The existence of a direct brain dysfunction is confirmed by other authors (13,40,89).

A change in neurotransmitter level can be the cause of the direct brain dysfunction. Boismare et al (12) described a posttraumatic change in neurotransmittor level in rats two days after these animals were subjected to an experimental whiplash injury without any direct blow to the head. Furthermore, Keidel et al (49) presumed that a change of neurotransmitter level caused an impaired jaw opening reflex in whiplash patients. These authors (49) measured mainly reflex changes in the multisynaptic and not in the oligosynaptic circuit of this reflex. Neuro-otological information processing also occurs by means of a multisynaptic circuit. So a change of neurotransmitter level in the central- vestibular information processing circuit can explain the central-vestibular dysfunctions in whiplash patients. It is not yet clear wether the change of neurotransmitter level is due to the a direct change in level or to indirect changes because of axonal injuries due to the impact, as described by Maxwell et al (63) in primates, or to change of proprioceptive and nociceptive input from the cervical region.

Perhaps in the future brain SPECT studies can give more insight into these mechanisms. Otte et al (73) reported about changes in parieto-occipital perfusions in 24 out of 25 chronic whiplash patients when compared to normal control subjects. They demonstrated the same effects in six out of seven patients with non-traumatic chronical pain and only in one out of eight patients with low back pain. Their (73) conclusion is that the perfusion irregularities are not a non-specific consequenuence of pain, but they presume that the hypoperfusion in chronic whiplash patients is due to stimulation of pain-sensitive afferents in the upper cervical region and not to a direct brain injury.

A PET study demonstrated that in healthy test subjects during reflexive saccades amongst others occipital, temporal, parietal and frontal areas are involved (1).



A SPECT study in acute, chronic whiplash patients and their healthy control subject, also examined with our special test procedure can perhaps elucidate the cause of the neuro-otological dysfunctions.

The observation that the strain of a neuro-otological examination can decrease the eye movement velocities of respectively, the lateral rectus muscles and the gain of smooth pursuit tells strongly in favour of the existence of a brain dysfunction. The importance of this finding should be emphasized. The effort of the conduction of the horizontal eye movement test battery with an interval of 20 minutes between the repeating of the saccade test and the smooth pursuit, has frequently an effect on patients sitting in a dark, silent room with only the instructions to conduct eye movements and no other interferences.

The initation times of the MCRT did not increase because of the efforts of the neuro-otological examination, while Matlung and Van der Scheer (86) observed frequently such an effect due to the efforts of the neuropsychological examination. However, the performance of the neuro-otological test battery took one hour, while the neuropsychological test battery takes minimally three hours.

The group of patients with less than three biological life events showed no effect due to the strain of the neuro-otological examination, while in the group of patients with more than two biological life events, indeed, an effect was observed. This observation suggests that biological life events can have a cumulative effect with regard to brain dysfunction/damage. This cumulative effect was already described by Houx (44) with respect to the initiation times of the MCRT. The case report of Blakely and Harrington (8) confirmed these findings. Furthermore, Gronwall and Wrightson (37) reported a cumulative effect in patients with a second concussion, when compared to patients with a first concussion. In patients with a second concussion the rapid information processing decreased more and these patients recovered slower than patients with a first concussion.

To get a better insight into the cumulative effect of biological life events on the outcome of the neuro-otological dysfunctions in information processing more research has to be done. Several healthy control subjects without complaints of dizziness and a

history of car accidents have to be examined: the first group without biological life events, the second group with one or more biological life events only with anaesthesia and no concussions and a third group only with concussions and no anaesthesia.

However, our study revealed that in some patients one biological life event influences the reaction times more than two or more biological life events in other patients. So an individual sensitivity to biological life events is likely. To get more insight into the course and the persistence of the neuro-otological dysfunction a prospective investigation has to be done, so that patients can be examined at several times.

Four patients complaining of increased fatigability and bordering at exhaustion, revealed a prolonged reaction time of the saccade test and the MCRT test and also an increase of the reaction time during the second saccade test. This observation suggests the existence of a correlation between the complaints of the patients and their neuro-otological and neuropsychological test results. At the moment a reliable statistical analysis can not be done (only four patients).

Meanwhile we enlarge our group of patients, in which the strain of the horizontal eye movements are observed, so in the future a better insight can be gained into the existence of a correlation between complaints as increased fatigability, exhaustion and other symptoms and the neuro-otoneurological findings.

**Conclusions**

The study revealed that:

1. three localizations of neuro-otological dysfunctions are involved in patients suffering from chronic aftereffects of a rear-end car collision.
2. the repeated conduction of the saccade test and the smooth pursuit test, besides the standard neuro-otological test battery, gives valuable information about the measure of fatigability of information processing in these patients when compared to healthy control subjects.
3. enlarging of the group of patients, examined with this special test procedure, opens the possibility to find correlations between complaints and neuro-otological test results.



4. the influence of biological life events in healthy control subjects on neuro-otological test results have to be studied.
5. the conduction of a prospective study will give a better insight into the course and the persistence of the neuro-otological dysfunctions.

## V Summary

### Introduction

The cervical acceleration syndrome has been the subject of many discussions and controversies. In 1995 The Quebec Task Force on Whiplash-Associated Disorders suggested a new definition. Furthermore, the European section of the Cervical Spine Research Society strongly advised to use the term "cervical distorsion syndrome" for the entity of signs and symptoms originating in a car accident in which the body is subjected to a sudden acceleration.

### I Mechanism and effect of a rear-end car collision

During a rear-end car collision the magnitude of loading forces is influenced by many factors. New technologies as high speed films, videoregistration and electronic equipment can give an better insight in the sequelae of rear-end car accident on the occupants. At the moment only low velocity impacts in humans with their head straight ahead are studied. In these studies only mild neck pain was mentioned. The response analysis of high velocity or low velocity rear-end impacts on humans with rotated neck is never done before, because of the risks for the occupant to get injuries.

The loading forces can damage the neck, the temporomandibular joints, the brain, the shoulders and the low back. Damage of the cervical cord, the nerve roots, the brain nerves and the brain is explained by shearing, tearing, rotating and compression forces resulting in mechanical, vascular and chemical damages or dysfunctions.

### II Review of literature concerning electronystagmographic, neuropsychological and radiological findings.

Eleven articles, reviewed on electronystagmographic findings revealed labyrinthine, cervical and central-vestibular dysfunctions. These dysfunctions were considered to be caused by vascular mechanisms, irritation of the sympathetic autonomic nervous system, excitation of the cervical proprioceptors or by direct brain lesions.

The review of nine neuropsychological articles revealed the existence of impaired

(sustained) attention, decreased concentration, memory problems, an increased sensitivity to interferences and an impaired speed of information processing in whiplash patients. These signs and symptoms were mainly ascribed to neuronal damage and dysfunctions in the brainstem, reticular formation, hypothalamus, hippocampus and frontal cortex and subcortex due to shearing strains.

Radiographs, CT- and MRI scans can demonstrate mechanical damage to the cervical spinal cord. Plain radiographs are the best screening methods: when the first physical examination is normal a lateral cervical radiograph, an AP open mouth and lower cervical radiographs are sufficient. In case of persisting neckpain functional flexion/extension radiographs are indicated.

**III Localization of neuro-otological dysfunctions.**

A summary is given the variety of complaints, which patients after a rear-end impact can develop.

Furthermore, our neuro-otological test battery is described.

Three origins of neuro-otologic dysfunctions are considered:

The labyrinth, the neck and the central nervous system.

These considerations are confirmed by our neuro-otological test results in 552 patients involved in a rear-end car collisions. Because of our test results suggested that central-vestibular dysfunctions played an important role and clinical observations revealed that fatigue can increase dizziness and other symptoms, it was decided to conduct a study aimed at the influence of the strain of the examination on the reaction time and velocity of eye movements and and on the reaction time (= initiation time) of the neuropsychological Motor Control Reaction Test (MCRT).

**IV A neuro-otologic study aimed at the influence of performing the examination on eye movement velocities and reaction times.**

In this study 40 patients with chronic complaints and signs after a rear-end car collision without being unconscious and without direct head impact on steering wheel or window were included.



The patients were matched on age, gender and education with healthy control subjects without biological life events as concussions, anaesthesia or accidents.

Besides the standard neuro-otological test battery, the effect of performing this examination on eye movement velocities and reaction times and on the reaction times of MCRT was registered.

The results showed significantly more central-vestibular dysfunctions in the patients than in control subjects. Furthermore, the patients frequently had prolonged reaction times while conducting the saccadic eye movements and performing the MCRT test.

Repeating of these tests revealed an further increase of the reaction times of the saccadic eye movements in patients with more than two biological life events. They had also an decrease of the gain of smooth pursuit. These effects were not measured in patients with one or two biological life events. The consequence of this findings is that the effect of biological life events on the performance of the neuro-otological tests have to be investigated. The reaction time of the MCRT was frequently prolonged in all patients, but an effect of the strain of the neuro-otological test battery could not be measured neither in the patient group with more than two biological life events, nor in the group with one or two biological life events.

The central-vestibular dysfunctions can be explained by a posttraumatic change in neurotransmitter level, directly or indirectly by axonal injuries or change of proprioceptive or nociceptive input from the cervical region.

**Conclusions**

1. Three localizations of neuro-otological dysfunction are involved in patients suffering from the aftereffects of a rear-end car collision.
2. The repeated conduction of the saccade test and the smooth pursuit test, besides the standard neuro-otological test battery, gives valuable information about the measure of fatigability of information processing in these patients when compared to healthy control subjects.
3. Enlarging of the group of patients, examined with this special test procedure, opens the possibility to reveal correlations between complaints and neuro-otological test results.

4. The influence of biological life events in healthy control subjects without dizziness on neuro-otological test results have to be studied.
5. The conduction of a prospective study will give a better insight into the course and the persistence of the neuro-otological dysfunctions.



## Samenvatting

### Inleiding

Het cervicale acceleratie syndroom is het onderwerp van vele discussies en controversen. In 1995 heeft de Quebec Task Force on Whiplash Associated Disorders opnieuw gedefiniëerd. Bovendien heeft de Europese afdeling van de Cervical Spine Research Society met nadruk geadviseerd voortaan de term " cervicaal distorsie syndroom" te gebruiken voor de entiteit van klachten en symptomen, welke ontstaan ten gevolge van een auto ongeval, waarbij het lichaam aan een plotselinge versnelling wordt onderworpen.

### I Mechanisme en effect van een aanrijding van achteren

Tijdens een aanrijding van achteren wordt de mate van de inwerkende krachten bepaald door diverse factoren. Nieuwe technologieën, zoals het gebruik van snelle films, videoopnames en electronische apparatuur geeft een beter inzicht in wat de gevolgen zijn voor de inzittenden van een auto die van achteren wordt aangereden. Momenteel zijn er met gezonde proefpersonen alleen experimenten gedaan waarbij zij met het hoofd recht vooruit worden blootgesteld aan een aanrijding van achteren met een geringe verandering van snelheid. Deze personen meldden na de experimenten soms een lichte pijn in de nek te hebben. Er zijn geen experimenten gedaan waarin de personen met gedraaid hoofd werden onderworpen aan een aanrijding van achteren met een lage of een hoge impact, omdat het risico te groot is.

De inwerkende krachten kunnen beschadigingen van de nek, de kaakgewrichten, de hersenen, de schouders en de onderrug veroorzaken. De beschadiging van het cervicale merg, de zenuwwortels, de hersenzenuwen en de hersenen worden verklaard door de inwerking van trek, druk, comprimerende en roterende krachten in deze structuren, hetgeen een beschading of een dysfunktie van mechanische, vasculaire aard of een verandering in de werking van neurotransmitters kan veroorzaken.

**II Electronystagmografische, neuropsychologische en radiodiagnostische bevindingen**

Uit elf artikelen met electronystagmografisch bevindingen blijkt dat er labyrinthaire, cervicale en centraal-vestibulaire dysfunkties aantoonbaar zijn. Men denkt dat deze dysfunkties worden veroorzaakt door vasculaire reacties, door prikkeling van het sympathische zenuwstelsel of van de cervicale proprioceptoren of door een directe beschadiging van het centrale zenuwstelsel.

Uit negen neuropsychologische artikelen blijkt dat whiplash patiënten moeite hebben met aandacht en concentratie. Verder hebben zij problemen met het geheugen, hebben zij interferentie stoornissen en is de informatie verwerking vertraagd. Deze klachten en symptomen worden voornamelijk toegeschreven aan neuronale dysfunkties of beschadigingen in de hersenstam, de reticulaire formatie, de hypothalamus, de hippocampus en de frontale cortex en subcortex ten gevolge van de inwerking van scherende krachten op de hersenen.

Door middel van röntgenfoto's van de nek, een CT- en een MRI scan kunnen beschadigingen van de cervicale wervelkolom worden aangetoond. Als er bij lichamelijk onderzoek geen afwijkingen worden gevonden, kan men volstaan met het maken van gewone röntgenfoto's, waarbij een laterale opname wordt gemaakt met het hoofd in neutrale stand en twee foto's in voor-achterwaartse richting, waarbij de dens, de cervicale

wervelkolom en de eerste thoracale wervel goed in beeld moeten worden gebracht. Bij persisteren nekpijn zijn functie foto's geïndiceerd.

**III Lokalisatie van de neuro-otologische dysfunkties**

In dit hoofdstuk wordt een overzicht gegeven van de vele klachten die patiënten na een aanrijding van achteren kunnen krijgen.

Verder wordt een beschrijving van onze neuro-otologische test batterij gegeven.

Er zijn drie lokalisaties waar neuro-otologische dysfunkties kunnen ontstaan: het labyrint, de nek en het centrale zenuwstelsel.

Dit wordt bevestigd door neuro-otologische bevindingen bij 552 patiënten die betrokken



zijn geweest bij een aanrijding van achteren. Aangezien de bevindingen er op wezen dat centraal-vestibulaire dysfunkties op zich een belangrijke rol speelden bij deze patiënten en uit anamnestische gegevens naar voren kwam dat vermoeidheid de duizeligheidsklachten en de andere symptomen, waaronder cognitieve,kan verergeren, werd het besluit genomen een studie uit te voeren met het doel na te gaan of de inspanning die een neuro-otologisch onderzoek vergt een effect heeft op de snelheid en reactietijden van oogbewegingen en eveneens op de reactietijden (= initiatietijden) van de neuropsychologische Motor Control Reaction Test (MCRT).

**IV Neuro-otologisch onderzoek met het doel het effect van de inspanning van het onderzoek op de snelheid en de reactie tijden van de oogbewegingen te bestuderen.**

In dit onderzoek werden 40 patiënten opgenomen met chronische klachten en symptomen ten gevolge van een aanrijding van achteren. De patiënten werden van het onderzoek uitgesloten, indien zij ten gevolge van het ongeval het bewustzijn hadden verloren of met hun hoofd hadden gestoten tegen het raam of het stuurwiel.

De patiënten werden gematched op leeftijd, geslacht en opleiding met gezonde proefpersonen zonder "biological life events", zoals hersenschuddingen, operaties onder narcose of ongevallen.

Naast het standaard neuro-otologisch onderzoek werd het effect bestudeerd van de inspanning van het onderzoek op de snelheid en de reactietijden van de oogbewegingen en eveneens op de reactietijden van de Motor Control Reaction Test (MCRT).

Bij het standaard onderzoek werden bij de patiënten beduidend meer centraal-vestibulaire dysfunkties geregistreerd dan bij de proefpersonen. Bovendien waren de reactietijden van de saccade test en de MCRT duidelijk vertaagd in vergelijking met die van de controle groep. Herhaling van deze testen deed de reactietijden van de oogbewegingen nog meer toenemen bij die patiënten die meer dan twee "biological life events" hadden doorgemaakt, maar niet bij die patiënten die met minder "biological life events" in de anamnese. Ook de gain van de gladde oogvolgbeweging bleek tijdens het tweede onderzoek te zijn afgenomen bij de patiënten met meer dan twee "biological life events". De consequentie van deze bevindingen is dat nader onderzoek gedaan moet worden naar

het effect van de inspanning van het uitvoeren van het neuro-otologisch onderzoek bij gezonde personen zonder duizeligheidsklachten en auto-ongevallen, maar met andere "biological life events" in de anamnese.

Ook bleken ook de reactie tijden van MCRT bij patiënten verlengd te zijn, maar een toename van de reactietijden van de MCRT door de inspanning van het neuro-otoneurologisch onderzoek werd bij de patiënten niet waargenomen.

De centraal-vestibulaire dysfunkties kunnen worden verklaard door een posttraumatische verandering in de neurotransmitter werking, op directe wijze of op indirecte wijze ontstaan doordat het ongeval axonale beschadigingen in de hersenen of veranderingen in de proprioceptie en nociceptie in de nek heeft veroorzaakt.

**Conclusies**

1. Bij patiënten die lijden onder de gevolgen van een aanrijding van achteren zijn drie localisaties aan te wijzen, waar neuro-otologische dysfunktie kan onstaan.
2. Naast het onderzoek met de standaard neuro-otologische test batterij, geeft de herhaling van de saccade test en de slinger test waardevolle informatie over de mate van vermoeibaarheid van de informatie verwerking bij deze patiënten.
3. Uitbreiding van deze groep patiënten, onderzocht met deze speciale test procedure, biedt de mogelijkheid correlaties aan het licht te brengen tussen klachten en bevindingen bij neuro-otologisch onderzoek.
4. Het effect van "biological life events" op neuro-otologische test resultaten bij gezonde personen zonder duizeligheidsklachten en auto-ongevallen in de anamnese moet onderzocht worden.
5. Een prospectief onderzoek zal een beter inzicht geven in het beloop en het persisteren van de neuro-otologische dysfunkties.



| P | saccade median I | saccade median II | saccade mean I | saccade std I | saccade mean II | saccade std II | pursuit gain I | pursuit gain II |
|---|---|---|---|---|---|---|---|---|
| 1 | 240.0 | 313.0 | 309.1 | 216.3 | 349.3 | 168.0 | .65 | .53 |
| 2 | 233.0 | 213.0 | 222.2 | 36.2 | 212.4 | 28.7 | .98 | .76 |
| 3 | 206.0 | 186.0 | 213.7 | 44.7 | 191.6 | 29.8 | .99 | .93 |
| 4 | 246.0 | 240.0 | 253.6 | 40.2 | 251.4 | 45.5 | .89 | .86 |
| 5 | 173.0 | 266.0 | 181.4 | 49.4 | 302.8 | 211.3 | 1.18 | 1.02 |
| 6 | 260.0 | 346.0 | 344.2 | 203.8 | 383.0 | 215.9 | 1.01 | .84 |
| 7 | 266.5 | 213.0 | 267.3 | 64.1 | 216.4 | 47.0 | 1.38 | .88 |
| 8 | 273.0 | 333.0 | 279.4 | 77.3 | 356.6 | 98.7 | 1.04 | 1.00 |
| 9 | 220.0 | 233.0 | 329.8 | 182.7 | 452.9 | 326.5 | 1.10 | .88 |
| 10 | 200.0 | 213.0 | 253.0 | 155.9 | 241.8 | 112.4 | .90 | .86 |
| 11 | 253.0 | 273.0 | 268.7 | 61.2 | 346.8 | 208.2 | 1.08 | .99 |
| 12 | 233.0 | 246.0 | 259.6 | 111.9 | 272.4 | 103.1 | .94 | 1.61 |
| 13 | 186.0 | 180.0 | 186.8 | 30.0 | 188.6 | 45.5 | .97 | 1.06 |
| 14 | 173.0 | 186.0 | 178.6 | 31.8 | 189.6 | 31.9 | .96 | .96 |
| 15 | 653.0 | 753.0 | 671.7 | 123.3 | 678.8 | 212.4 | .71 | .75 |
| 16 | 173.0 | 180.0 | 180.4 | 28.2 | 175.6 | 25.1 | 1.04 | 1.00 |
| 17 | 206.0 | 253.0 | 214.0 | 46.2 | 246.2 | 72.9 | 1.10 | .84 |
| 18 | 180.0 | 203.0 | 189.9 | 39.0 | 263.3 | 150.4 | 1.11 | 1.00 |
| 19 | 180.0 | 193.0 | 189.2 | 80.4 | 200.6 | 120.1 | .84 | .86 |
| 20 | 246.0 | 320.0 | 250.2 | 50.3 | 470.1 | 271.8 | .98 | .78 |
| 21 | 380.0 | 560.0 | 379.8 | 144.7 | 521.4 | 118.1 | .87 | .85 |
| 22 | 220.0 | 180.0 | 249.8 | 94.6 | 194.0 | 43.7 | 1.03 | 1.00 |
| 23 | 209.5 | 286.0 | 225.3 | 38.3 | 287.2 | 39.1 | 1.03 | .87 |
| 24 | 246.0 | 480.0 | 321.6 | 175.7 | 465.4 | 162.3 | .90 | .96 |
| 25 | 426.0 | 320.0 | 431.8 | 154.1 | 389.5 | 207.8 | .97 | .95 |
| 26 | 180.0 | 220.0 | 189.0 | 25.6 | 271.8 | 144.9 | .74 | .91 |
| 27 | 140.0 | 150.0 | 149.1 | 51.9 | 161.5 | 56.7 | 1.10 | 1.10 |
| 28 | 180.0 | 186.0 | 182.4 | 30.3 | 192.0 | 64.1 | .94 | .81 |
| 29 | 173.0 | 186.0 | 172.2 | 47.6 | 197.0 | 42.8 | 1.56 | .97 |
| 30 | 273.0 | 426.0 | 276.5 | 92.2 | 426.3 | 146.9 | 1.03 | 1.11 |
| 31 | 220.0 | 253.0 | 221.4 | 33.8 | 271.0 | 90.6 | 1.03 | .91 |
| 32 | 260.0 | 266.0 | 252.0 | 49.1 | 278.7 | 95.5 | .90 | 1.03 |
| 33 | 206.0 | 240.0 | 210.6 | 30.3 | 235.1 | 29.5 | 1.02 | .91 |
| 34 | 200.0 | 200.0 | 191.7 | 50.9 | 205.5 | 34.9 | 1.05 | .81 |
| 35 | 173.0 | 193.0 | 194.4 | 61.5 | 193.3 | 38.3 | .97 | .91 |
| 36 | 213.0 | 200.0 | 210.4 | 52.6 | 208.0 | 69.6 | .94 | .87 |
| 37 | 160.0 | 193.0 | 159.3 | 31.3 | 184.0 | 37.2 | 1.54 | .74 |
| 38 | 220.0 | 240.0 | 203.3 | 58.9 | 270.3 | 110.2 | 1.11 | .83 |
| 39 | 193.0 | 206.0 | 204.4 | 74.8 | 215.7 | 39.9 | 1.01 | .85 |
| 40 | 186.0 | 186.0 | 249.8 | 136.7 | 198.7 | 38.4 | .98 | .95 |

**Appendix A1. Latency of saccade test I and II in patients,**

**15 measurements per test.**

**Gain of smooth pursuit I and II.**

| | saccade median I | saccade median II | saccade mean I | saccade std I | saccade mean II | saccade std II | pursuit gain I | pursuit gain II |
|---|---|---|---|---|---|---|---|---|
| 41 | 200.0 | 193.0 | 204.8 | 32.7 | 206.8 | 29.2 | .85 | .98 |
| 42 | 200.0 | 193.0 | 212.6 | 26.7 | 192.5 | 17.9 | .94 | .85 |
| 43 | 206.0 | 226.0 | 214.8 | 28.6 | 218.6 | 30.9 | .92 | .93 |
| 44 | 186.0 | 206.0 | 190.0 | 62.8 | 209.2 | 31.7 | 1.00 | .90 |
| 45 | 193.0 | 213.0 | 193.8 | 24.9 | 209.0 | 30.7 | 1.38 | 1.10 |
| 46 | 193.0 | 206.0 | 194.8 | 44.6 | 212.6 | 24.7 | .87 | .93 |
| 47 | 200.0 | 200.0 | 204.9 | 30.6 | 199.8 | 36.3 | 1.07 | .93 |
| 48 | 166.0 | 173.0 | 172.2 | 39.2 | 165.8 | 33.1 | .91 | 1.00 |
| 49 | 186.0 | 180.0 | 191.6 | 25.8 | 182.8 | 22.0 | 1.04 | 1.03 |
| 50 | 200.0 | 260.0 | 198.0 | 35.0 | 249.8 | 42.6 | .94 | 1.02 |
| 51 | 173.0 | 180.0 | 173.0 | 18.9 | 184.9 | 40.8 | .90 | .84 |
| 52 | 193.0 | 220.0 | 193.0 | 37.6 | 212.1 | 25.6 | 1.10 | .93 |
| 53 | 193.0 | 203.0 | 90.6 | 29.0 | 218.3 | 38.5 | .96 | 1.02 |
| 54 | 206.0 | 186.0 | 203.2 | 32.8 | 189.4 | 40.5 | 1.07 | 1.51 |
| 55 | 153.0 | 146.0 | 159.6 | 35.0 | 156.1 | 34.2 | 1.05 | 1.05 |
| 56 | 140.0 | 180.0 | 145.0 | 35.5 | 186.8 | 45.7 | 1.10 | 1.03 |
| 57 | 240.0 | 253.0 | 250.8 | 35.9 | 260.3 | 42.8 | 1.36 | 1.01 |
| 58 | 206.0 | 213.0 | 204.7 | 22.7 | 208.0 | 26.7 | 1.49 | 1.01 |
| 59 | 206.0 | 206.0 | 224.4 | 43.4 | 205.4 | 26.3 | .90 | 1.06 |
| 60 | 193.0 | 220.0 | 190.2 | 43.9 | 222.0 | 24.5 | 1.04 | .91 |
| 61 | 186.0 | 206.0 | 186.6 | 28.4 | 222.8 | 51.7 | .94 | .87 |
| 62 | 193.0 | 209.5 | 198.3 | 23.9 | 207.3 | 30.2 | .86 | 1.39 |
| 63 | 180.0 | 180.0 | 177.5 | 35.6 | 169.7 | 53.1 | 1.39 | 1.09 |
| 64 | 206.5 | 153.0 | 196.4 | 50.9 | 191.6 | 86.5 | .78 | 1.03 |
| 65 | 193.0 | 193.0 | 194.3 | 14.5 | 196.1 | 21.4 | .99 | .94 |
| 66 | 186.0 | 186.0 | 190.7 | 31.1 | 205.8 | 76.9 | .92 | .90 |
| 67 | 200.0 | 206.0 | 203.7 | 24.5 | 214.3 | 36.9 | 1.02 | .94 |
| 68 | 200.0 | 193.0 | 195.2 | 23.8 | 209.9 | 41.4 | 1.00 | .97 |
| 69 | 190.0 | 173.0 | 189.3 | 24.9 | 211.2 | 94.2 | .96 | .98 |
| 70 | 180.0 | 240.0 | 193.9 | 37.2 | 236.2 | 44.1 | .98 | 1.05 |
| 71 | 200.0 | 200.0 | 222.0 | 104.8 | 207.8 | 45.3 | 1.14 | .92 |
| 72 | 200.0 | 219.5 | 195.2 | 38.7 | 217.5 | 31.5 | .96 | .97 |
| 73 | 166.0 | 166.5 | 177.8 | 42.4 | 163.0 | 26.8 | 1.12 | 1.03 |
| 74 | 206.0 | 206.0 | 206.8 | 54.6 | 236.0 | 141.2 | .88 | .89 |
| 75 | 226.0 | 240.0 | 232.5 | 65.9 | 232.1 | 49.9 | .89 | 1.17 |
| 76 | 213.0 | 213.0 | 214.4 | 49.1 | 203.2 | 49.4 | .96 | .97 |
| 77 | 220.0 | 220.0 | 214.8 | 59.8 | 223.2 | 43.0 | .97 | .93 |
| 78 | 200.0 | 200.0 | 217.5 | 49.4 | 221.9 | 69.1 | 1.00 | 1.03 |
| 79 | 179.5 | 206.0 | 187.7 | 37.6 | 203.2 | 18.4 | 1.05 | .99 |
| 80 | 186.0 | 183.0 | 181.8 | 23.1 | 196.4 | 31.4 | .93 | .95 |

**Appendix A2. Latency of saccade test I and II in control subjects, 15 measurements per test. Gain of smooth pursuit I and II.**



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## Dankwoord

Veel waardering heb ik voor mijn promotor, Prof.dr. W.J.Oosterveld. Zijn nauwgezette en toegewijde begeleiding waren onmisbaar bij de het tot standkomen van dit proefschrift.

Karin Matlung en Elly van der Scheer hebben mij vertrouwd gemaakt met de Motor Control Reaction Test. Dat zij hun apparatuur belangeloos aan mij hebben willen uitlenen voor het onderzoek heb ik zeer gewaardeerd.

Eric Sondag dank ik voor het wegwijs maken in het gebruik van statistische programma's en voor het vervaardigen van de figuren in dit proefschrift.

Leo van Elferen ben ik erkentelijk voor de adviezen betreffende de statistische analyze.

Met Henk Herngreen heb ik veel vruchtbare discussies mogen voeren. Hij heeft mij veel waardevolle suggesties gegeven bij de opzet en de uitwerking van het onderzoek.

De kritische opmerkingen over het manuscript van Jan van Gemert heb ik gewaardeerd.

Andrea van Dongen hielp mij bij het opstellen van de literatuurlijst: een grote inspanning, vooral omdat de lijst een veelvoud van artikelen bevatte vergeleken met de nu gepubliceerde lijst.

Ide Oosterveld-Paumen ben ik erkentelijk voor de adviezen betreffende de engelse tekst.

Pauline Schrama was onvermoeibaar bij de planning van de onderzoeksafspraken. Zij heeft mij weten te motiveren op momenten dat ik het werkelijk nodig had.

De medewerkers van de vestibulaire afdeling ben ik dankbaar voor de steun die zij mij gegeven hebben.

Henk Kortschot stond altijd klaar als het werken met de computer problemen opleverde.

De vormgeving van het omslag heb ik te danken aan Ineke Baas.

De proefpersonen ben ik erkentelijk voor de bereidheid deel te nemen aan het onderzoek.

Tenslotte ben ik al diegenen die op enige wijze hebben bijgedragen aan het tot standkomen van dit proefschrift dankbaar.



## Curriculum vitae

De schrijfster van dit proefschrift werd geboren op 8 juli 1953 te Winterswijk. Aan de Rijksscholengemeenschap in deze plaats behaalde zij in 1972 het diploma Gymnasium ß.

Na de middelbare school nam zij deel aan het Brugjaar Welzijnszorg te Rotterdam.

Het artsenexamen werd in 1981 aan de Vrije Universiteit te Amsterdam afgelegd.

In 1982 vervulde zij een vakantieassistentschap in het Burgerziekenhuis te Amsterdam.

Sinds 1983 is zij werkzaam op de Vestibulaire Afdeling van het AMC.