Persistent neuropsychological deficits in cervical whiplash patients without direct headstrike

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Abstract

Persistent cognitive and neurasthenic complaints can be the consequence of a cervical whiplash injury. In contrast to otonurological and orthopedic reports, neuropsychological studies on whiplash patients are scarce. We report disturbances of information processing and memory functions in two patients with a pure cervical whiplash injury with demonstrated central otoneurological abnormalities, whereas there was no evidence of intellectual deterioration or focal cortical deficits. The results suggest that the cognitive disturbances and neurasthenic symptoms may be part of one syndrome which appears to manifest itself under conditions of time pressure and distraction.

Key words: Cognitive functioning; Whiplash injury.

Introduction

There is controversy on the question whether the nonspecific cranial symptoms following cervical whiplash injury have an organic or psychogenic etiology (Miller and Cartridge, 1972; Jacone, 1987; Etting et al., 1989; Radanov et al., 1989; Yarnell and Rossie, 1988). The subjective complaints can be very invalidating, and include head and neck pain, adynamia, easy fatigability, decreased tolerance to light and sound and difficulties with memory or concentration. Neuropsychological and otonurological evidence is accumulating that a cervical injury may indirectly damage subcortical brain structures (Hinoko, 1985; Jacone, 1987). In addition, there is increasing evidence that also mild head injury (MHI) without loss
of consciousness is characterized by persistent neurocognitive disturbances in a significant number of cases (Bohnen et al., 1992a). However, neuropsychological evidence for organic brain damage in isolated cervical whiplash injuries without direct headstrike is scarce (Kischka et al., 1991). Information regarding the indure of possible neurocognitive disturbances is especially missing. We report two patients with a cervical whiplash injury without remembered headstrike who were referred for neuropsychological and neuropsychiatric evaluation because of litigation affairs. All patients experienced an extensive disruption of everyday activity and were unable to continue their jobs.

Case reports

Patients were neurologically and psychiatrically examined. EEG or CT-scans were obtained as needed. Neuro-otological examination consisted of appraisal of ocular movements. Vestibular function was evaluated by electronystagmography, positional and rotational tests.

The neuropsychological investigation was performed essentially as described earlier (Jolles, 1985, see Lezak, 1983 for more specific information). Briefly, standardized psychometric tests and information processing tasks were used as well as a behavioral neurological investigation (Luria-Christensen). Thus, tests of perception, language, visuconstruction, motor and intellectual functions were used. The Rey Auditory Verbal Learning Test (RAVLT; Brand and Jolles, 1985; Bohnen et al., 1992a) was administered under conditions with and without verbal distraction. In addition, simple and more complex speed tasks, such as the Trail Making Test (TMT), Stroop Color Word Interference Test and a paper and pencil version of the Sternberg type scanning task (MCT; Brand and Jolles, 1987; Houx, Vreeling and Jolles, 1991) as well as a sustained attention test (Bourdon-Wiersma) were used. The tests used, enable an interpretation as to the nature of performance deficits on neurocognitive tests. More specifically besides information as to performance on an intelligence test as a measure of knowledge and intelligence, performance on timed tests and information processing tests is of importance in the assessment of patients with possible global brain dysfunction. For instance, Shum et al. (1990) show clear effects on attentional processes by use of a Sternberg type Memory Scanning Task, such as used in the present study. Likewise, earlier studies by our research group show clearcut effects of (very) mild brain trauma, depression and age-associated cognitive complaints on Stroop test and Modified Trail Making Test (e.g. Bohnen et al., 1992b; Vink and Jolles, 1985; Houx et al., 1991).
Patient 1 is a 37-year-old scientist. Twenty-three months earlier she had been a passenger in the right front seat of an automobile that was struck from the left rear end. Initially, the head was swung to the left, followed with a turn to the right. The patient did not remember a headstrike, and an amnesic period — if present — must have been less than one minute. Within three hours after the accident, she started to have persistent dizziness, and cervical stiffness together with paresthesias in the arms. Before the accident, the patient was a healthy and intellectually creative woman. Despite an early bedrest of three weeks, complaints of adynamia, dizziness, decreased libido and irritability increased over the following years. The neck pain, muscular stiffness and dizziness persisted, but decreased in intensity. She was unable to continue her responsible job as a manager due to difficulties with reading and organizing. She slept well and was not depressive. She did not take any medication, did not smoke and drank alcohol only occasionally.

On medical examination the spinous processes of the lower neck were tender to percussion with limited motion of the cervical spine in flexion and extension. She was neurologically intact except for a sensory alteration in the left ulnar region. The Romberg test was slightly positive. Radiographs of the cervical spine showed loss of the normal curvature together with a calcification in the interspinosal ligament at C7-Th1. The spinous processes at C1 and C2 were slightly rotated. EEG recording was unremarkable. Otoneurological investigations yielded a bilateral gaze nystagmus, indicative of diffuse damage in the brain stem, together with a cervical nystagmus of the proprioceptive type (Biemond and De Jong, 1969).

The psychiatric investigation was aimed at obtaining information regarding her present status and the possible presence of psychiatric symptoms and psychological or relational problems in the years preceding the accident. There was not any indication for psychopathology, either before or after the trauma. More specifically, evidence of major depression, anxiety disorder, post-traumatic stress disorder, psychosis, neurotic defense mechanism and personality disorder were absent as well as any indications of purely psychogenic problems. At the time of the accident, she was moving upward in her career and her prospects were good.

At neuropsychological investigation the patient appeared well oriented in time and place. The intellectual functions were above average (IQ = 125). Perception and motor functions were intact as well as spontaneous speech, speech comprehension, fluency and expressive speech. Visuoconstructive abilities were somewhat deficient. In contrast to these findings with respect to intelligence, perceptual, motor and language functions which were normal or close to normal, profound dysfunctions were found on memory tests
and timed tests. For instance a relatively low performance on memory span tasks (see table 1, second decile) and profound performance decrement were found on the Rey Auditory Verbal Learning task in the immediate recall, delayed recall and delayed recognition (performance in the first decile and nearly two standard deviations inferior to healthy control groups). These findings are taken to be a manifestation of deficient acquisition and retrieval. In addition, the patient showed increased sensitivity to interference indicating that new information is more easily lost when she is distracted by irrelevant information. Findings on a Sternberg type information processing task (see table 1) show that she is both slow (first decile and far beyond two standard deviations inferior to age matched controls) on the simple subtasks as well as extremely slow on complex subtasks which have to be performed under increased memory load (first decile and > 2 SD different from controls) similar findings were found for Stroop test and Modified Trail Making Test (see also Vink & Jolles, 1985) in which general slowness was present in the Stroop and especially a bad performance under conditions of increased attentional requirements (Stroop III, TMT digits/letters). A qualitative impression was that she compensates for the reduced speed of information processing by working more carefully: this is only possible by increased use of mental effort which appeared to lead to rapid development of fatigue. The subject stated that tasks which were performed automatically in the past, now required an active mental control. Generally, interpretation as to the nature of the underlying deficit can be described in terms of the neurocognitive model adhered to; thus interpretations as to an effect on vigilance, attentional processes, planning deficits or increased sensibility to interference are equally well possible. The present study was not devised to find contrasts between these interpretations. Suffice it to state that the deficits which are found in the neuropsychological testing make the complaints more objective. The patient is able to cope with the demands of everyday life only when no time pressure is laid upon her and when she is allowed to do one thing at a time in her own tempo (Bohnen et al., 1992a).

**Patient 2.** Two and a half year earlier this patient, a previous mentally and physically healthy 26 year old nurse, was sitting in a parked car which was hit from the left-rear end. She remembered that her head initially swung to the right, then forward. Immediately after the accident she felt nauseated and dizzy, and experienced paresthesias in the right arm and leg. She was able to drive home without assistance. The week after the trauma she was given a soft collar. After more than six months of rest she continued to go to work each day, but was unable to function on the job. She in-
creasingly complained of cognitive problems, disturbed sleep and blurred vision. She did not take sedative medication and drank alcohol only incidentally.

At physical examination she was neurologically intact, except for paresthesias in the right shoulder and fingers which were provoked by hyperabduction of the right arm. There was a limited motion of the cervical spine, especially to the left and right plane. The spinothalamic processes of the cervical spine were painful to percussion. Radiographs of the cervical spine were unremarkable. A CT-scan of the brain gave no evidence of brain damage. Otoneurological investigations indicated a positional nystagmus with disturbed saccadic movements of the eyes. Cervical provocation aggravated the positional nystagmus. Rotational tests demonstrated a dysfunction of the brain stem vestibular nucleus. Psychiatric investigation into her present status and the possible presence of psychiatric symptoms, psychological and relational problems in the years preceding the accident was unremarkable. There were no symptoms of post-traumatic stress disorder, personality disorder, psychosis, major depression or anxiety disorder, on any indications for purely psychogenic problems.

At neuropsychological investigation the patient was motivated and well oriented in time and place. Her intelligence was in the higher normal range (IQ = 115). Speech, perceptual and language functions were normal; whereas motor functions were slow but normal. In contrast to the findings with respect to intelligence, perceptual, speech and language and under functions which were normal or close to normal, profound deficits were found on memory tasks and in simple and more complex speed tasks that had to be performed under time pressure (see table 1). Specifically, a very profound dysfunction was found for nearly all aspects of memory tests, including acquisition and retrieval in a word learning test and even recognition memory (table 1).

With respect to timed tasks, which had to be performed under conditions of increasing memory load (Sternberg task), both a general slowness and a profound dysfunction with respect to memory scanning speed was found (first decile and > 2 SD beyond healthy controls). Similar findings were done for Stroop, Trail Making test and other tests (see table 1). There was a very inflexible learning strategy but she was not especially sensitive to distraction and interference on memory tasks. Likewise, her memory span was normal (fifth decile). She was very slow on simple speed tests and especially on the more complex tests. It appeared that she compensated for the decreased rate of information processing by working more carefully. The neuropsychological findings correspond to the complaints in normal life, especially with respect to memory deficits and
Table 1
Summary of the neuropsychological findings on selected memory and timed tests
(s = second; decile = decile of normative data)

<table>
<thead>
<tr>
<th>Neuropsychological test</th>
<th>Patient 1</th>
<th>Control group to patient 1</th>
<th>Patient 2</th>
<th>Control group to patient 2</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>score</td>
<td>mean SD</td>
<td>decile</td>
<td>score</td>
</tr>
<tr>
<td>RAVLT immediate recall (# correct)</td>
<td>40</td>
<td>52.0 ± 7.5</td>
<td>1</td>
<td>26</td>
</tr>
<tr>
<td>RAVLT delayed recall (# words)</td>
<td>9</td>
<td>14.2 ± 2.0</td>
<td>3</td>
<td>1</td>
</tr>
<tr>
<td>RAVLT delayed recognition (# words)</td>
<td>12</td>
<td>14.1 ± 1.1</td>
<td>7</td>
<td>1</td>
</tr>
<tr>
<td>Utrecht Trail Making Test Digits (s)</td>
<td>31</td>
<td>33.0 ± 9.2</td>
<td>57</td>
<td>1</td>
</tr>
<tr>
<td>Utrecht Trail Making Test Letters (s)</td>
<td>27</td>
<td>35.0 ± 9.4</td>
<td>51</td>
<td>1</td>
</tr>
<tr>
<td>Utrecht TMT Digits/Letters (s)</td>
<td>77</td>
<td>55.8 ± 20.0</td>
<td>88</td>
<td>1</td>
</tr>
<tr>
<td>Stroop test Naming (s)</td>
<td>48</td>
<td>40.0 ± 4.6</td>
<td>119</td>
<td>1</td>
</tr>
<tr>
<td>Stroop Test Colour Naming (s)</td>
<td>92</td>
<td>52.3 ± 8.6</td>
<td>94</td>
<td>1</td>
</tr>
<tr>
<td>Stroop Test Colour Word Naming (s)</td>
<td>155</td>
<td>88.7 ± 19.9</td>
<td>390</td>
<td>1</td>
</tr>
<tr>
<td>Memory Scanning Task (1 letter)</td>
<td>47</td>
<td>28.5 ± 5.4</td>
<td>59</td>
<td>1</td>
</tr>
<tr>
<td>Memory Scanning Task (2 letters)</td>
<td>55</td>
<td>39.1 ± 7.4</td>
<td>87</td>
<td>1</td>
</tr>
<tr>
<td>Memory Scanning Task (3 letters)</td>
<td>95</td>
<td>47.2 ± 9.8</td>
<td>120</td>
<td>1</td>
</tr>
<tr>
<td>Memory Scanning Task (4 letters)</td>
<td>103</td>
<td>55.2 ± 12.8</td>
<td>219</td>
<td>1</td>
</tr>
<tr>
<td>Digits Forward (# digits)</td>
<td>4</td>
<td>5.5 ± 0.9</td>
<td>5</td>
<td>5</td>
</tr>
</tbody>
</table>

Patients were compared in their performance on selected neurocognitive tests with a control group consisting of respectively 34 and 29 normal healthy subjects without cognitive complaints. The age range of group 1 and group 2 were respectively 30 ± 2 and 40 ± 2. The mean and standard deviations are given per (sub)task. The decile scores are also derived from comparison with these control groups (see Houx 1991, for further elaboration).

lack of cognitive speed and efficiency. In addition, the fatigue which is found on testing is a manifestation of the increased mental effort to perform tasks which went automatically in the premorbid period (see also Ettlin et al., 1989).

Discussion
Reports on neuropsychological deficits as organic evidence for brain damage after a cervical whiplash injury are scarce. Kischka et al. (1991) presented evidence for neuropsychological dysfunction in whiplash injured patients, but do not substantiate these findings with further otoneurological abnormalities. Fisher (1982) reported a case with a pure whiplash trauma of the neck that caused anterograde amnesia for 72 hours. In addition, Ettlin et al. (1989) demonstrated that a cognitive profile of deficits in attention, concentration and memory distinguished a physiogenic origin of the post-whiplash syndrome from psychogenic causes. Further organic evidence for cerebral symptoms after a cervical whiplash injury
include electrophysiological and otoneurological abnormalities (Torres and Shapiro, 1961; Hinoko, 1985; Jacome, 1987), and disturbances in brain amine metabolism in subcortical brain structures (Boismare et al., 1985). These central changes may be caused by an indirect shearing effect of the neck injury (Seletz, 1958; Ommaya et al., 1968). In the present study it was found that two patients with a direct cervical whiplash injury persistingly complained of a variety of nonspecific neurasthenic symptoms together with an extensive disruption of everyday activity. The patients displayed no signs of a neurotic-conversive behavior or of other psychopathology either before or after the trauma. Neuropsychological evaluations of the patients demonstrated a comparable picture of cognitive disturbance: there were no signs of intellectual or higher cortical disturbances, whereas there were clear memory problems. Although it could be possible that a chronic stress condition may adversely affect cognitive efficiency, such a condition cannot cause these clearcut cognitive deficits. In addition, the general slowness which is found in Sternberg-type information processing tasks and other tests, and the rapidly increasing time which is needed to perform subtasks for which an increased memory load is needed may well correspond to the complaints in everyday life (see also Brand & Jolles, 1987; Houx, Vreeling & Jolles, 1991; Shum et al., 1990). The rapid increase of fatigue in cognitively complex situations may be related to the increased mental effort needed to perform tasks which went automatically in the premorbid period (Bohnen et al., 1992a). The patients appear to be able to cope with the demands of everyday life only when no time pressure is laid upon them and when they are allowed to do one-thing-at-the-time in their own tempo. Future research with larger groups of whiplash patients and matched controls should be performed in order to be able to state clearly what type of cognitive processes are likely to be involved (e.g. van Zomeren et al., 1984). Considering the otoneurological disturbances in both patients and given the nature of the cognitive disturbances, it is tempting to relate the cognitive deficits to damage in subcortical brain structures as well as to orbital prefrontal dysfunction (e.g. Fuster, 1989). More research should be directed at investigating the role of the prefrontal cortex in the neuropsychological sequelae of whiplash trauma.

With respect to a pathophysiological mechanism it is possible that a pure whiplash trauma causes a more prolonged period of rotational deceleration force in comparison with a direct head strike. Given the different angle of rotation, duration and direction of the forces it is possible that a pure whiplash trauma may indirectly cause neuronal damage (see also Ommaya and Gennarelli, 1974).

It can be concluded that pure cervical whiplash trauma could indirectly damage cerebral tissues and may cause cognitive impair-
Cognitive-energetic deficits may contribute to the neurasthenic symptoms of the post-whiplash syndrome. Although there was no evidence of major depressive disorder in these patients, we cannot completely rule out effects of mild dysthymia on cognition in the neurasthenic syndrome. Further neuropsychobiological research is needed to elucidate the interaction between neural substrate, cognition and secondary effects on behavior.

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