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Hyperactive VOR and Hyperventilation After Whiplash Injury

A. J. E. M. FISCHER,1 P. L. M. HUYGEN,2 H. T. FOLGERING,3 W. I. M. VERHAGEN4 and E. J. M. THEUNISSEN5

From the Departments of Otolaryngology,1 St. Jans Hospital, Weert, 2University Hospital Nijmegen and 3St. Maarten’s Hospital, Venlo, 4Department of Pulmonology, University Hospital Nijmegen, and 5Department of Neurology, Canisius-Wilhelmina Hospital, Nijmegen, the Netherlands

INTRODUCTION

Whiplash injury is defined as a noncontact acceleration-deceleration injury to the head-neck system. The most common cause is a rear-end car collision. Presenting symptoms usually include neck pain, headaches, disequilibrium, blurred vision, paraesthesiae, changes in cognition, fatigue, insomnia, and hypersensitivity to light and sound. Other symptoms, often associated with long-term disability, include imbalance, light-headedness and vertigo. Vestibulo-ocular reflex (VOR) was hyperactive (n = 17; 53%) and the respiratory test results were typical of the hyperventilation syndrome (HVS) (n = 12; 38%). Hyperactive VOR and the HVS occurred significantly more often in combination (n = 7; 22%) than could be accounted for by combined false positivity. Most of the significant findings were due to high relative frequencies in the women. The hyperactive VOR might have been the result of plastic adaptation to limited head mobility secondary to neck pain. Behavioural and emotional distress might offer alternative explanations for both the hyperactive VOR and HVS. Key words: vestibulo-ocular reflex (VOR), cervico-ocular reflex (COR), emotional disorder.

MATERIAL AND METHODS

Vestibular, oculomotor and respiratory function tests were performed on 32 patients after whiplash injury (15 men, 17 women, age range 21–66 years) caused by a rear-end car collision 1–26 months previously. They were referred to us (TF, ET) by general practitioners from a local region who had been informed about this study. Before enrolment, the potential participants completed a questionnaire (4) to check whether the collision and injury fulfilled our study criteria. These criteria concerned only the type of accident and injury and disregarded the symptoms and/or signs. Candidates who reported any other relevant disorder or who were using any relevant drugs were excluded from the study. Each participant underwent routine diagnostic, radiodiagnostic (standard and functional X-rays of the cervical spine), otoneurological and respiratory function tests. The latter comprised the usual oculomotor tests to evaluate saccade velocity, smooth pursuit (SP) and optokinetic nystagmus (OKN) responses (6), vestibular tests, i.e. caloric tests and the velocity step (VS) test (5), a COR test (7) and a hyperventilation test (5, 8) previously described. P5–95 confidence limits were used for the HVS (5), the caloric test (mean slow phase velocity at culmination 7–45°/s (9)) and the single VS response variables (i.e. initial velocity V = 30–65°/s, time constant T = 11–26 s and “Gesamtamplitude”—or cumulative eye displacement—G = 485–1135° (5, 10)). The values P14–26 applied to the combined criteria for hyperactive VOR (5) and the values P0.7–1.3 applied to the combined occurrence of hyperactive VOR and a positive hyperventilation test (P5). These P values were used as the mean probability in a calculated binomial distribution for the relevant sample size, in order to determine the P5 limit beyond which a given observed frequency would be significantly high. For significantly high frequencies, the (P5) frequency limit is indicated.

RESULTS

General, oculomotor and COR findings
No gaze-evoked nystagmus was observed, and saccades, SP and OKN responses were generally normal. The COR responses were within the normal range in all of the patients, except for one who had a high gain.

Vestibular findings
None of the patients showed any spontaneous nystagmus. Caloric responses revealed only one case of bilateral weakness and one case of unilateral weakness (40% relative difference in caloric sensitivity). The VS responses were generally symmetrical, but a hyperactive VOR was found in 17 patients (53%), which was statistically significant (P < frequency limit: 9–14). Most of the relatively high frequencies were due to the women’s results (65% of them showed a hyperactive VOR).

DISCUSSION

None of our whiplash patients showed any gaze-evoked nystagmus and saccades and the SP/OKN responses were generally normal. Chester (1) only found minor oculomotor abnormalities in a few instances (2 out of 39 chronic patients tested). In contrast to these findings, Hildingsson et al. (11) found a significant reduction in saccade peak velocity and SP gain in a subgroup of 20 chronic patients with severe symptoms of neck pain, neck stiffness and headaches, but no such abnormalities in a group of 19 nonsymptomatic patients (less than half of the patients in both categories had experienced rear-end car collisions). Oosterveld et al. (12) reported unilateral gaze-evoked nystagmus in only 6%, but bilateral gaze-evoked nystagmus in 26% and saccadic SP responses in 43% of the (chronic symptomatic) cases (after unspecified accidents). The use of drugs was not mentioned or discussed in either of the two latter reports; drugs may have been used especially by the chronic symptomatic patients. Hinoki (13) mentioned “irregular” OKN responses in chronic symptomatic patients; he also mentioned that Ushio et al. (14) reported inverted OKN responses after whiplash injury.

Only one patient showed an abnormally high COR gain; this was not beyond the 5% probability level (i.e. false positivity). This agrees with the findings previously reported for women (only) by others (15), who used a similar stimulation method. Although Oosterveld et al. (12) reported “cervical nystagmus” in 79% of the patients (with the eyes closed) they did not indicate the precise stimulus conditions, or any control values or statistical analysis. Normal subjects may show a wide range of COR gains (7) and it is therefore almost impossible to distinguish between “positive” and “negative” responses in a valid way without statistical testing. This has recently also been stressed by Holtmann et al. (16), who could not find any significant difference in COR responses between a group of normal subjects and a group of patients with an upper cervical spine syndrome, which was the result of whiplash injury in 6 cases. Interestingly, spontaneous nystagmus was shown by the majority of patients described by Oosterveld et al. (12) (see below). Many of their patients also showed gaze-evoked nystagmus (see above), but the authors did not describe how they distinguished between nystagmus observed during neck stimulation and pre-existing nystagmus. For a further (critical) discussion and a review of “cervical nystagmus”, we refer to the recent report by Holtmann et al. (16).

None of our whiplash patients showed any spontaneous nystagmus. Spontaneous nystagmus was reported in 63% (chronic symptomatic patients) by Oosterveld et al. (12) and in 29 to 30% by Compere (17) and Toglia (2), whereas Pang (18) and Zenner (4) mentioned the clinical observation of provoked (?) or gaze-evoked nystagmus in only 5% of their cases. Abnormal caloric responses were generally lacking in the present study, i.e. substantial canal paresis was found in 4% and bilateral weakness also in 4% of our patients. Similar low percentages have been reported in 3 studies (12, 17, 19), while other authors mentioned canal dysfunction in 33 to 40% (1, 2, 18).

Asymmetry of rotatory responses was generally lacking in the present study. Toglia (2) reported significant asymmetry of rotatory responses in 51%, but Chester (1) mentioned similar abnormalities in only 11%.

As far as we know, a hyperactive VOR has not been reported in any previous study on whiplash injury. This feature is known to occur in multiple sclerosis (20), in vestibulocerebellar dysfunction (21), which may be associated with kernicterus (22) and, furthermore, in (idiopathic) spasmodic torticollis
(ST) (23) and the HVS (5). The finding of a high VOR gain in ST may be relevant because we have previously suggested that this may represent (plastic) VOR enhancement to compensate for apparent limitation of active head movements (23). Similar enhancement can be suggested for the present whiplash patients, because they generally had neck pain and neck stiffness and often showed limitation of neck rotation. This has also been mentioned in other reports (2, 4, 12), but we have to admit that about half of our patients showed an increased time constant of the VOR instead of an increased gain.

We know of no previous reports about respiratory tests performed on whiplash patients. Almost 40% of our patients (almost 50% of the women) showed evidence of the (paroxysmal) HVS. The HVS may be placed in the context of a posttraumatic syndrome with either organic or non-organic (“emotional”) components (or both, see below).

The high frequency of the combination of a hyperactive VOR and the HVS could not be explained by combined false positivity. Apart from possibly representing behavioural adaptation, i.e. to limited neck mobility, the hyperactive VOR may have been caused by a lesion (dysfunction) of the vestibular commissural system (20). A lesion in the brain stem may also involve the sensory centre with its central connections. Both this centre and the VOR are under the influence of the reticular formation (the reticular activating system). The latter structure may be involved in emotional disorders and be under the influence of the “mental state” which has an important bearing on both breathing behaviour and the VOR. It is interesting to note that noradrenalin excretion is significantly increased in patients with the HVS (24), because this would fit into a stress model in which both a hyperactive VOR and the HVS can occur concomitantly.

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Address for correspondence:
P. L. M. Huygen
Department of Otolaryngology
University Hospital Nijmegen
P.O. Box 9101
NL-6500 HB Nijmegen
The Netherlands
Fax: +31-80-540251