Whiplash injury and oculomotor dysfunctions: clinical–posturographic correlations

Abstract Oculomotor dysfunctions are hidden causes of invalidity following whiplash injury. Many patients with whiplash injury grade II present oculomotor dysfunctions related to input disturbances of cervical or vestibular afferents. We used static posturography to investigate 40 consecutive patients with whiplash injury grade II and oculomotor dysfunctions. We demonstrated a relation between length and surface of body sway: the surface value ($A$) was higher than the length value ($L$) and this led to an open graph of body sway in the statokinesigram. Oculomotor rehabilitation can resolve the impairment of vestibular function but if therapy is delayed or the patient has been wearing an orthopaedic neck collar, more therapeutic sessions are required. In conclusion, without rehabilitation of the oculomotor muscles other therapies are not sufficient to recover the impairment caused by whiplash injury.

Keywords Whiplash injury · Oculomotor dysfunctions · Posturography · Rehabilitation · Prognosis

Introduction

The whiplash injury is defined as a distortion trauma localized at the neck mostly due to motor vehicle collisions and caused by a sudden acceleration of the trunk with hyperextension, hyperflexion or lateroflexion of the neck.

The signs and symptoms observed after whiplash injury include neck pain, headache, nausea, vertigo, emotional and cognitive disturbance [10, 14, 17]. The ophthalmic and oculomotor complications are largely unknown. Commonly impaired functions include ocular accommodation, convergence and stereocuity [4] and seem to be caused by brain stem lesions [9].

Oculomotor muscle disorders deserve greater attention because may be responsible for many symptoms and perpetuate neck pain. Moreover the proprioceptive system of the neck muscles can influence the oculomotor and vestibular systems [2, 7, 13].
Baron and Fowler [3] claimed that it was possible to cure instability and vertigo using low power prisms. Gagey et al. [5] affirmed that the oculomotor system is among the indispensable structures that supply the information useful both for the stabilization of the human body and for postural control.

Other authors investigated the oculomotor system deficit present in 62% of patients affected by whiplash injury and found a strict correlation between oculomotor system impairment and the alterations of cervical afferences [6]. Moreover they assumed that these dysfunctions could be a premature sign of illness/myelopathy of the central nervous system [20] or a lesion of the brain stem [9]. Burke et al [4] claimed that oculomotor abnormalities following whiplash injuries are generally mild and have a good prognosis.

Involvement of the oculomotor system is evident if we consider that stretching of the cervical spine and the consequent traction exercised on the brain structures (medulla, brain stem, cerebellum) can damage the oculomotor system causing visual alterations and vestibular disorders without any evident organic intrinsic vestibular problem [11].

Posturography can supply useful indications in describing postural anomalies. Various reports have addressed oculomotor abnormalities and whiplash injury [4,6,8] but very few deal with posturography and the oculomotor system.

There is general consensus on the role of the labyrinth and proprioception in the maintenance of an upright posture. The area of body sway increased more than length when a patient with labyrinthine disorder closes his eyes [21].

The aim of this study was to measure the incidence of oculomotor impairment and the deficit of ocular convergence caused by hypofunction of the eye muscles in those patients with whiplash injury grade II. We also aimed to establish a correlation between posturography and symptoms in subjects with whiplash syndrome by estimating the modifications by computerized posturography and clinical signs after oculomotor rehabilitation.

Subjects and methods

Patient selection

The study group comprised 40 consecutive patients [F/M: 30/10, mean age 33.8 years (range 12 to 56 years), standard deviation (SD) 10.8] with a whiplash injury grade II, according to the Quebec Task Force Classification on whiplash associated disorders (WAD) [19], and oculomotor dysfunctions. Grade II included neck complaints and musculoskeletal signs. Each patient in the study group was also suffering from one or more of the following clinical oculomotor dysfunctions:

- Monolateral/bilateral deficit of internal rectum muscle;
- Diplopia to near vision using red lens (8 cm from point of view);
- Pathological oculomotor divergence (8 cm from point of view);
- Nausea or vertigo after convergence test;
- Nausea or vertigo after sight of 1 m distance point.

Patients with head injury, unconsciousness, fracture or dislocation of the cervical spine were excluded.

Each patient was examined from 7 to 270 days after trauma, mean 94.2, SD 68.7, (T0), and after oculomotor rehabilitation (T1).

The normal group comprised 40 patients without any history of whiplash injury or oculomotor dysfunctions.

The control group comprised 40 consecutive patients with a history of whiplash injury grade II without oculomotor disorders.

Method of examination

Static posturography was performed on each patient using a normalized computerised static posturography platform (Basys2, Elettronica Pagani, Milan, Italy) in accordance with the French Association of Posturology [1,5]. The tests were recorded with the patient’s eyes open (EO) and closed (EC). The EO test was repeated (re-test) for a greater reliability of the examination. The second test was considered for EO parameters. The parameters considered were the surface ($A$) of the ellipse of the statokinesigram (90% of surface area covered by the trace), the trace length ($L$) of the statokinesigram and the length per unit area ($L/A$).

The values proposed by Gagey and the French Association of Posturology were considered normal. The ellipse surface in EO values was between 39 and 210 mm², the ellipse surface in EC was between 79 and 638 mm², the trace length in EO between 307 and 888 mm and the trace length in EC between 346 and 888 mm [5].

Oculomotor rehabilitation

The 40 consecutive patients with whiplash injury grade II and oculomotor dysfunctions underwent from one to four consecutive cycles of oculomotor rehabilitation (ten sessions per cycle). One session consisted in 20 min in which oculomotor convergence and motility exercises were executed [12]. Patients needing more than one rehabilitative cycle had a 2 week break. Only
symptomatic medicines were prescribed during the oculomotor rehabilitation.

Statistical analysis

The results are expressed as mean±SD and median. Statistical analyses of mean differences were performed using the Student’s t test (SPSS-Windows). The alpha value for our studies was 0.05, so we considered $P \leq 0.05$ significant.

Results

Static posturography disclosed that patients with whiplash injuries and oculomotor disorders presented a characteristic pattern of body oscillation ($A$, $L$, $L/A$). Figure 1a shows the statokinesigram values in a patient with oculomotor disorders. The morphology of the statokinesigram appears open. Figure 1b shows the same patient after oculomotor rehabilitation. The values of $A$, $L$, $L/A$ have returned within normal range and the morphology of statokinesigram is compact. Table 1 summarizes $A$, $L$, $L/A$ before and after oculomotor rehabilitation (T0, T1). Table 2 summarizes $A$, $L$, $L/A$ in the normal and control groups. There is a prevalence in the female sex in the two groups with whiplash injury.

Patients in Table 1 wore a neck collar for a longer period than the control group (27.9±17.8 vs 18.6±11.3 in Table 2). After oculomotor rehabilitation (T1) there was a significant reduction of $L$ and $A$ in EO and EC ($P < 0.01$). It is important to note the high SD at (T0), reduced at (T1).

At (T0) the $L/A$ ratio was 2.3±1.1 in EO and 1.5±0.8 in CE, in T1 3.1±1.5 in EO and 2.5±1.5 in CE. In the control group the $L/A$ ratio was 3.5±1.2 in EO and 2.8±1.1 in CE and the normal group presented an $L/A$ ratio 4.6±1.3 in EO and 3.3±1.1 in CE. In (T1) this parameter in the study group was similar to that of the control group.

Table 3 groups patients with one oculomotor rehabilitation cycle (ten sessions) and patients who underwent several cycles. The two groups are similar in age (32.3±10.5 vs 38.3±10.9) and sex distribution. The first group started the therapy after 82.9±62.2 days vs 128.0±115.0 days in the second group. The first group wore a neck collar for 22.1±6.9 days versus 45.3±27.8 days in the second group.

The first group had a significant reduction of $L$ and $A$ values, whereas the second group only had a significant reduction in the $A$ value in (T1). Static posturography

\[ \text{Vol} : 17.406 \text{ (mm/s)} \quad \text{Lungh.} : 891.185 \text{ (mm)} \]
\[ \text{A/P} : 12.266 \text{ (mm/s)} \quad \text{Vol Var.} : 199.378 \text{ (mm/s)} \]
\[ \text{Lat.} : 9.297 \text{ (mm/s)} \quad \text{Vel Sqm.} : 14.120 \text{ (mm/s)} \]
\[ \text{Area} : 689.500 \text{ (mmq)} \quad \text{Dist. x y} : -3.708 / 1.868 \text{ (mm)} \]

\[ \text{Vol} : 6.932 \text{ (mm/s)} \quad \text{Lungh.} : 354.934 \text{ (mm)} \]
\[ \text{A/P} : 4.766 \text{ (mm/s)} \quad \text{Vel Var.} : 25.689 \text{ (mm/s)} \]
\[ \text{Lat.} : 3.516 \text{ (mm/s)} \quad \text{Vel Sqm.} : 5.868 \text{ (mm/s)} \]
\[ \text{Area} : 90.000 \text{ (mmq)} \quad \text{Dist. x y} : 3.909 / -7.322 \text{ (mm)} \]
always recorded a significant increase in $L/A$ values in both groups.

**Discussion**

The analysis of posture by static posturography provides information on overall postural performance with parameters such as the surface and length of body sway. The labyrinth is the “main monitor” of surface body sway, while proprioception is the “principle monitor” of velocity (equal to the length) of body movement sway [16, 21].

Eye movements and vestibular disturbances were experimentally studied using the vestibular nerve blockade with lidocaine, confirming the relation between these two systems [15]. The inputs derived from vestibular receptors are important to maintain both posture and gait. When vestibular function is normal, these reflexes operate with accuracy and, in the case of eye movements, at very short latencies. Knowledge of vestibular anatomy and physiology is important for physical therapists to diagnose and manage patients with vestibular dysfunction [18].

Oculomotor function studies in patients with whiplash injury suggested that some patients who claimed to have no symptoms after trauma showed oculomotor dysfunctions. These dysfunctions might be related to cervical afferent input disturbances. Neck pain measured with VAS did not correlate with oculomotor performance [6].

We focused on oculomotor abnormalities in patients with whiplash injury syndrome grade II. During static computerised posturography there was a tendency for $A$ to be more increased than $L$ in patients with oculomotor dysfunction compared with the control group. This was visible in the statokinesigram as an open body sway (Fig. 1a). The results showed that the values of static posturography at (T0) presented a high SD: in fact the oculomotor dysfunction was also present in patients who claimed to have no vestibular symptoms. The importance of $A$ and $L$ values was secondary to the $L/A$ ratio. After oculomotor rehabilitation (T1) all posturography parameters returned to normal values, all the patient’s $L/A$ ratio increased and the morphology of body sway of SKG appeared compact.

We considered the $L/A$ ratio extremely important when confirming vestibular-oculomotor dysfunctions. From this study we deduced the $L/A$ ratio to be in a normal range if over 3.0 value (1/mm) in OE test and over 2.5 value (1/mm) in CE test.

The oculomotor system can be indirectly considered the “surface monitor” of body sway like the vestibular system. This is due to the interactions between these two systems.

### Table 1

Mean, median and standard deviation in the study group. The values of length, surface and length per surface at (T0 and T1) were studied using Student’s $t$-test

<table>
<thead>
<tr>
<th>Case</th>
<th>Age</th>
<th>Days after trauma</th>
<th>Neck collar (days)</th>
<th>Oculom. rehab. sessions</th>
<th>EO surface (mm²)</th>
<th>EC surface (mm²)</th>
<th>EO length (mm/51.2 in.)</th>
<th>EC (length mm/51.2 in.)</th>
<th>EO $L/A$</th>
<th>EC $L/A$</th>
</tr>
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<tbody>
<tr>
<td>T0</td>
<td>T1</td>
<td>T0</td>
<td>T1</td>
<td>T0</td>
<td>T1</td>
<td>T0</td>
<td>T1</td>
<td>T0</td>
<td>T1</td>
<td>T0</td>
</tr>
<tr>
<td>40 pts; 11M/29F</td>
<td>33.8</td>
<td>94.2</td>
<td>27.9</td>
<td>14.4</td>
<td>472.2</td>
<td>231.6</td>
<td>104.3</td>
<td>465.0</td>
<td>702.4</td>
<td>510.0</td>
</tr>
<tr>
<td>Median</td>
<td>31.5</td>
<td>60.0</td>
<td>21.0</td>
<td>10.0</td>
<td>317.5</td>
<td>148.0</td>
<td>679.0</td>
<td>381.0</td>
<td>627.0</td>
<td>471.5</td>
</tr>
<tr>
<td>SD</td>
<td>10.8</td>
<td>68.7</td>
<td>17.8</td>
<td>8.6</td>
<td>566.9</td>
<td>193.2</td>
<td>972.5</td>
<td>386.2</td>
<td>369.5</td>
<td>231.0</td>
</tr>
<tr>
<td>$t$ test</td>
<td>$P &lt; 0.01$</td>
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<td>$P &lt; 0.01$</td>
</tr>
</tbody>
</table>

### Table 2

Mean, median and standard deviation of 40 patients with whiplash injury without oculomotor dysfunctions (control group) and 40 patients without history of whiplash injury (normal group)

<table>
<thead>
<tr>
<th>Groups</th>
<th>Age</th>
<th>Days after trauma</th>
<th>Neck collar (days)</th>
<th>EO surface (mm²)</th>
<th>EC surface (mm²)</th>
<th>EO length (mm/51.2 in.)</th>
<th>EC (length mm/51.2 in.)</th>
<th>EO $L/A$</th>
<th>EC $L/A$</th>
</tr>
</thead>
<tbody>
<tr>
<td>Control Group 40 pts; 16M/24F</td>
<td>28.4</td>
<td>84.5</td>
<td>18.6</td>
<td>136.6</td>
<td>246.3</td>
<td>407.5</td>
<td>565.8</td>
<td>3.5</td>
<td>2.8</td>
</tr>
<tr>
<td>Mean</td>
<td>27.5</td>
<td>55.0</td>
<td>15.0</td>
<td>132.0</td>
<td>252.0</td>
<td>403.5</td>
<td>592.5</td>
<td>3.1</td>
<td>2.3</td>
</tr>
<tr>
<td>Median</td>
<td>8.8</td>
<td>128.2</td>
<td>11.3</td>
<td>76.3</td>
<td>127.0</td>
<td>102.9</td>
<td>150.5</td>
<td>1.2</td>
<td>1.1</td>
</tr>
<tr>
<td>Normal Group 40 pts; 17M/23F</td>
<td>33.9</td>
<td>–</td>
<td>–</td>
<td>84.1</td>
<td>180.1</td>
<td>338.0</td>
<td>494.5</td>
<td>4.6</td>
<td>3.3</td>
</tr>
<tr>
<td>Mean</td>
<td>33.0</td>
<td>–</td>
<td>–</td>
<td>70.0</td>
<td>136.5</td>
<td>322.0</td>
<td>443.0</td>
<td>4.5</td>
<td>3.1</td>
</tr>
<tr>
<td>Median</td>
<td>12.7</td>
<td>–</td>
<td>–</td>
<td>44.8</td>
<td>102.3</td>
<td>85.6</td>
<td>144.8</td>
<td>1.3</td>
<td>1.1</td>
</tr>
</tbody>
</table>
Oculomotor rehabilitation led to a gradual resolution of vestibular symptomatology. At (T1) the \(L/A\) values were similar to those of the control group and the open body sway had disappeared in SKG (Fig. 1a, b).

Table 3 highlights the importance of clinical observation of oculomotor disorders since patients with oculomotor disorders who started oculomotor rehabilitation after 3 months had greater difficulty in obtaining remission. Comparing the two groups in Table 3 we noted the importance of limiting the use of a neck collar. In fact the second group had used neck collars for longer periods of time (45.3±27.8 vs 22.1±6.9). The table shows that using a neck collar for as long as 3 weeks reduces the probability of obtaining remission from the signs and symptoms observed. The \(L/A\) ratios of these groups increased at (T1), confirming the validity of oculomotor rehabilitation. The second group presented a significantly increases ratio but the values remained below those of the first group.

**Conclusion**

Oculomotor system dysfunction induces a dysfunction of the vestibular system and consequently changes the \(L/A\) ratio [21]. Oculomotor dysfunctions are encountered in many subjects with whiplash injury grade II and should be diagnosed as soon as possible after whiplash injury as they are responsible for a wide range of symptoms such as vertigo and chronic cervical pain. A neck collar should be worn for only a short time after injury. If oculomotor dysfunctions are evident, oculomotor rehabilitation must be initiated immediately after injury. Underestimation of oculomotor dysfunction risks failure of other types of therapy.

**References**