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Eye Movement Disorders after Whiplash Injury

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Visual disturbances are quite frequent after whiplash injury (23), and most of them result from eye movement disorders. These disorders are usually considered to be caused by brainstem or cerebellar lesions (21), but voluntary eye movements are perceptivo-motor processes and some of the dysfunctions may have their origin in the sensorial and cognitive steps. This pre-motor stage can be easily assessed by evaluating the efficiency of different stimuli that would generate the same eye movement. In this study, we have modulated the presentation time-of-target for visually guided saccades in patients suffering from the consequences of a whiplash injury.

METHODS

Eye movement disorders were assessed by clinical examination and oculography in 50 patients (mean age, 39.3 years) referred to our neuro-ophthalmological unit after a whiplash injury. The selection was made according to a history of sudden head acceleration relative to the trunk, without head trauma or cervical fracture. Most patients were victims of rear-end car collisions. The median delay between the injury and the examination was 12.4 months (range, 3 weeks to 113 months). Adequate questions were asked to specify the visual complaints; for instance, does the disorder affect near or distance vision, or fixed or moving visual targets?

The clinical examination searched for ocular lack of alignment (phoria or tropia), spontaneous eye instability (during fixation or with Frenzel lenses), fusional amplitude, and convergence power. When the neck movements were well tolerated, the eye stability was also assessed after the head-shaking test and in different head positions known to induce positional vertigo.

Ocular pursuit and visually guided saccades were recorded by DC-electro-oculography. The targets (Nicolet light bar controlled by a computer) were randomly presented at

10°, 20°, and 30° to the right or to the left of a central fixation point. A succession of 40 stimulations was carried out in accordance with two paradigms. First, the lateral target was switched on for 1 sec after the extinction of the central fixation point. Second, the target was switched on for 10 msec after the disappearance of the central fixation point and then switched on again 1 sec later for 1 sec in the same position. The subject was warned of the lighting of the target by a nonlateralized sound. Horizontal eye movements were recorded for 2 sec after the central point was switched off. The data were then digitalized at a sampling time of 2 msec. The amplitude and velocity measurements were computed on the basis of the initial calibration and the stable position of the eyes at the end of the 1-sec target presentation. The automatic analysis was visually controlled for each saccade off-line. The computer then calculated the theoretical velocity–amplitude relationship (VAR) that best fit the velocity data, with the function

$$Velocity = a[1 - e^{(b.amplitude)}]$$

using an algorithm that minimizes the residual sum of squares. The amplitudes and velocities considered were those of the first saccade phase that followed the first modality target presentation. This VAR was then used to compute a theoretical velocity for the first phase amplitude of the saccade generated after the 10-msec stimulus. Differences between these theoretical velocities and the measured velocities for the short stimuli were assessed with a nonparametric permutation test for paired observations (19). These data were calculated independently for each eye in each direction.

The accuracy, latencies, and velocities of these visually guided saccades for sustained (1 sec) and short (10 msec) stimuli were quantified and compared with a control group of 36 subjects (mean age, 32.6 years) without neurologic or neuro-ophthalmological history. Latencies and amplitudes must be characterized by an asymmetric distribution, whereas velocity distributions are Gaussian. Using these various distributions, we computed the limits including 95% (l_{95}) and 99% (l_{99}) of the control group data. The normative data were also analyzed according to age. No significant change was observed within the range of patient age. Our normative data were computed separately for each eye in each direction and published previously (24). We have considered that a saccade parameter was impaired for a given subject when it was outside the l_{99} for at least 5 of the 40 saccades. This takes the distribution flattening of these parameters into account, which is not always reflected by means comparisons.

RESULTS

Because of the neuro-ophthalmological recruitment, most of these patients (73%) complained of visual disorders (Table 1). Impaired near-sight, diplopia, oscillopsia, wrong distance evaluation, and intolerance to moving objects in the visual field were the more frequent visual symptoms. Most diplopias were intermittent and, at the time of examination, an ocular misalignment was observed in only 20 patients: 3 with convergence spasms, 4 with hypertropia of one eye, and 13 with horizontal diplopia for near or distance vision. One case of hypertropia resulted from trauma to the fourth cranial nerve, but increased vertical fusional power beyond 5 diopters in the two other cases did not allow us to establish the difference between a decompensated congenital fourth nerve palsy and a posttraumatic but long-lasting fourth nerve palsy (17). The fourth patient presented with a left hypertropia with right extorsion and shift of the subjective visual vertical of both eyes suggesting an ocular tilt reaction.

TABLE 1. *Complaint frequencies*

Complaint	Patients with complaint (out of a total of 50) (N)	Complaints seen after a delay < 12.4 mo (total, 33 examinations) (%)	Complaints seen after a delay > 12.4 mo (total, 34 examinations) (%)
Headaches	36	88	72
Impaired near-sight	21	40	34
Diplopia	20	40	32
Unsteadiness	17	21	35
Positioning vertigo	10	21	20
Nucalgia	10	24	11
Oscillopsia	10	21	29
Wrong distance evaluation	10	21	18
Intolerance to moving objects in the visual field	10	21	29
Rotational vertigo (nonpositioning)	4	12	5
Linear vertigo	3	3	5
Topographical disorientation	2	9	5
Scotoma	2	3	8
Acrophobia	2	6	3
Intolerance to acceleration	2	3	3

Only seven patients were examined on several occasions. Two were free of symptoms between 15 and 20 months but one of them kept saccadic impairments. The five remaining patients continued to have visual complaints and eye movement disorders. Therefore, we tried to document the possible evolution of the complaints and related eye movement disorders by splitting the patient group according to the median of examination delay from the whiplash injury. Except for postural and visual instability, the complaint frequencies remained quite similar. Findings of clinical examination and eye movement recordings (Table 2) showed the same invariability except for paroxystic positional vertigo and convergence spasms, which were more frequent, and fusional vergence disorders, which were less common in the second subgroup. Saccade impairments were the most frequently recorded abnormalities, especially for short targets, and they persisted for a long time after the injury for some patients.

Table 3 shows the incidence of impaired parameters in relation to the side and presentation time of the saccade stimuli, or the direction of the pursuit stimulus. There was a significant asymmetry with a left-side predominance of the latency increase and saccade hypometry but not for saccade hypermetry and pursuit. These latency and amplitude asymmetries were accentuated when the shorter stimuli were used. Although these stimuli also induced a further saccade slowing, this was not significantly asymmetric.

DISCUSSION

The specific recruitment of a neuro-ophthalmological unit clearly explains the higher incidence of visual disturbances seen there compared with a neurology department (23).

TABLE 2. *Eye movement disorders*

Disorder	Patients seen with the disorder after a delay < 12.4 mo (total, 33 examinations) (%)	Patients seen with the disorder after a delay > 12.4 mo (total, 34 examinations) (%)
Decreased fusional vergence	13 (39%)	6 (18%)
Convergence insufficiency	8 (24%)	5 (15%)
Horizontal misalignment	7 (21%)	6 (18%)
Positioning nystagmus	3 (9%)	7 (21%)
Spontaneous nystagmus	3	3 (9%)
Abnormal head shaking test	1	2
Convergence spasm		3 (9%)
Vertical misalignment	2	2
Abnormal accommodation	1	
Voluntary nystagmus		1
Disorders of eye movement recordings:		
Pursuit	20 (61%)	15 (44%)
Saccades (target of 1 sec)		
Latencies	19 (58%)	15 (44%)
Accuracy	14 (42%)	17 (50%)
Velocities	7 (21%)	6 (18%)
Saccades (target of 10 msec)		
Latencies	32 (97%)	26 (76%)
Accuracy	31 (93%)	29 (85%)
Velocities	26 (79%)	24 (76%)

The most striking result of this study is the predominance of saccade impairment for visual targets appearing in the left hemifield. It is unlikely that this results from an asymmetry of lesions along the anatomic pathways that generate visually guided saccades. We suggest that it is related to the different skills governed by the right and left brain hemispheres. Symmetrical dysfunctions of both hemispheres have different consequences on perceptive performances within the left or right hemispace. In fact, the eye movement is the last step of the perceptivo-motor program generating visually guided saccade. The further impairment of saccade parameters when using shorter targets at the same location suggests that this lack of efficiency lies at a pre-motor level. The occurrence of a new target in the visual field periphery during a central point fixation triggers at first an attentional process. The attention must be disengaged from the central fixation and then reengaged to the new peripheral stimulus. This is performed without any eye movement (15). This attentional process would depend of a cortico-limbo-reticular loop (25). A second process must then decide whether this new stimulus is sufficiently interesting to be accurately analyzed by the central portion of the retina. Simultaneously, the target position in space is computed. If the decision process gives an affirmative answer, a saccade is performed. This decision process would be performed in a part of the loop (i.e., the posterior parietal cortex) (12).

Inattention is dependent on the disruption at any level of these cortico-limbo-reticular loops. Lesions of mesencephalic reticular formation, thalamus, or frontal lobes also induce attentional deficits. An attentional deficit would explain why saccade latencies and accuracies are the most asymmetrically impaired. Pursuit of a moving target does not require this attentional engagement and disengagement process, because it remains projected in the center of the visual field.

Although less sensitive to hemispheric dysfunctions, saccade velocities may also be slowed by supracollicular lesions (24). The hypothesis that an attentional deficit is at the

TABLE 3. *Frequency of impaired saccades or pursuits*

Saccades	Left side only (%)	Both sides (left > right) (%)	Right side only (%)	Both sides (right > left) (%)	Both sides (symmetric) (%)	Probability
1-sec stimulus						
Latencies						
Increased	22	6	4	2	22	< .01
Normal					44	
Amplitudes						
Decreased	20	4	4		10	< .01
Increased	6		4		6	n.s.
Normal					50	
Velocities						
Decreased	10		2		8	< .05
Normal					80	
10-msec stimulus						
Latencies						
Increased	16	20		2	46	< .01
Normal					16	
Amplitudes						
Decreased	32	12	4	2	32	< .01
Increased		2	4		6	n.s.
Normal					14	
Velocities						
Decreased	18	10	18	2	26	n.s.
Normal					26	
Pursuit						
Saccadic aspect	22	2	6	4	16	n.s.
Normal					50	

Frequencies of impaired saccade or pursuit parameters in relation to the presentation side (saccades) or direction (pursuit) of the stimulus. Pure and predominantly unilateral impairments were pooled to test the significance of the asymmetry.

$N = 50$ patients.

root of eye movement disorders after whiplash injuries is also consistent with other neuropsychological studies (5,16). In a prospective study by Di Stefano and Radanov (4), pain and medication were not found to be major factors in explaining the cognitive impairment.

The question is now, why does this attention disorder prevail for visual targets in the left hemispace? Some hypothetical mechanism may be formulated.

Loops of both hemispheres are not equally efficient for the attention in the ipsi- and contralateral visual hemispace. The right hemisphere seems to modulate the attention in both ipsi- and contralateral hemispaces, whereas the left hemisphere is less efficient for attentional processing in the left hemispace (25). This could explain why left attentional deficit is more pronounced in patients with mild bilateral hemispheric dysfunction.

Another proposal is that left hemispheric dysfunction induces a right inattention for verbal task, whereas a left inattention for space-perceptual task would be more the consequence of the right hemispheric dysfunction (11). Kinsbourne (10) conjectured that the verbal communication occurring between the doctor and the patient would result in the activation of the patient's left hemisphere and would foster an increase of his tendency of gaze to the right.

Pre-motor skills for eye movements are also not symmetric between the two hemispheres. The incidence of conjugate head and gaze deviation, with preserved reflex eye movements, after a stroke is significantly higher in the right-brain-damaged group than

in the left-brain-damaged group, and the sign recovers slowly in the former (3). Hemiinattention was still present in many of these patients who had recovered from gaze palsy. This supports a relationship between attentional mechanisms and oculomotor balance. These authors also showed that gaze paresis appears predominantly associated with parietal injury in the right brain or with a widespread involvement of the left hemisphere. In that way, bilateral parietal dysfunctions after whiplash injuries may impair more markedly leftward saccades. Such bilateral parietal dysfunction is supported by brain single-photon emission computed tomography (SPECT) in late whiplash syndrome (13,14).

Finally, depressive disorders may modify the balance between the cerebral hemispheres, but the attentional direction shift remains controversial and very dependent on the stimulus and gender (2,18,20). Moreover, we would expect that this effect would increase with delayed recovery and this was not obvious in our group.

Posttraumatic impaired near-sight has been known for a long time (26). It is generally attributed to convergence insufficiency, poor accommodation, and fusional vergence. These functions depend on volitional effort and subject attention capacity. Lesions at midbrain level but also at hemispherical levels (9,22) may induce convergence and fusional disorders. It is interesting to note that acquired parietal lobe lesions may lead to impaired stereopsis and fusional vergence (6). In that way, hemispheric arousal deficits may also partly explain these disorders.

The spasm of the near reflex characterized by pseudomyopia, convergent strabismus, and miosis is more often related to functional than organic disorders (7). This etiologic aspect and its poor spontaneous remission probably explain their later occurrence in the group we studied.

Vestibular problems are quite frequent after whiplash injuries (8), especially benign positional vertigo (1). Its tendency to recur accounts for the persistence of the 20% positive tests we recorded 1 year after the injury.

From this study, it can be concluded that near-sight, pursuit, and saccade impairments are long-drawn-out deficits after a whiplash injury. Attentional dysfunctions probably explain the asymmetric (prevailingly left-sided) impairment of saccade latencies and accuracies. Similar hemispheric arousal deficits are likely to interfere with nonlateralized tasks such as convergence, accommodation, and visual fusion, which are related to the impaired near-sight, the most frequent visual complaint of our patients after a whiplash injury. Many brain structures, from midbrain to parietal cortex, are involved in these attentional processes and a restricted location could not be stated precisely, whereas diplopia and nystagmus are more related to brainstem, cranial nerve, or vestibular lesions.

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