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ORIGINAL COMMUNICATION

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Abnormality of vergence latency in children with vertigo

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Abstract It is well known that vergence movements are important for distance appreciation, depth vision and stereopsis. Moreover, vergence movements are very probably used by the CNS during head and body motion to adjust the gain of the vestibulo-ocular reflex (VOR) according to the viewing distance. A recent clinical study of Anoh-Tanon et al. suggested that vertigo in children with normal vestibular function could be associated with abnormal vergence clinically assessed. The purpose of this study was to test this hypothesis with objective vergence eye movement recordings. We examined the latency of vergence, saccades and combined movements in twelve children with the complaint of vertigo but without vestibular abnormality. Convergence and saccades combined with convergence or with divergence had abnormally long latencies (relative to normal children of matched age). In contrast, divergence and isolated saccades showed only mild latency increase relative to normals. Lengthening of latency could be due to impaired cortical control. Orthoptic vergence training reduced all latencies; however, even the reduced latency of vergence and of combined movements was still abnormal. The improvement after orthoptic vergence training could be due to increased visual attention, although such mechanism cannot eliminate completely the initiation deficit of vergence movements. Objective eye movement recordings are thus useful for a diagnosis and treatment of children with vertigo.

Key words latency · saccade · vergence · combined movements · vertigo

Introduction

Vergence eye movements are essential for clear binocular vision but also for depth vision, accurate distance evaluation and stereopsis [14]. It is also known that postural stability depends on viewing distance [4] and a recent survey shows balance problems in non-corrected hyperopic children [2]. Anon-Tanon et al. [1] reported that more than 5% of the 520 children consulting the ENT service for vertigo, headaches and equilibrium disorders showed normal vestibular function but revealed signs of vergence abnormalities assessed by orthoptic tests [29]. These authors pointed out that deficits in vergence could impair gaze stabilization during movements of the body and thus, cause double or blurry vision which can lead to vertigo and sensation of imbalance. Despite the convincing clinical evidence of the link between vertigo symptoms and vergence abnormalities there have been no studies of such abnormalities with objective eye movement recordings. Indeed, the orthoptic clinical tests provide subjective information, i. e. sense of double vision, which is attributed to incapacity to perform the appropriate vergence

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movements in static condition. However, these tests do not give any information about the vergence movements themselves and their temporal aspects such as their latency.

To prepare and perform an eye movement, e. g. a saccade, several processes at the cortical and sub-cortical level take place: the visual information from the retina is sent to visual cortex, parietal cortex, frontal lobe, superior colliculus and then, via the brain-stem the motor command is sent to the extra-ocular muscles. The latency of eye movements is the preparation time for performing the movement and includes several processes: shift of the attention to the visual target, disengagement of oculomotor fixation and computation of the movement parameters [9, 10]. All these processes involve the activation of several cortical areas, particularly of the parietal cortex and of the frontal lobe [20]. The latency is an important measure that provides information about cortical function. It is not known whether in subjects with vergence abnormalities and vertigo there are abnormal eye movement latencies. The goal of this study was to examine the latency of vergence, saccades and combined saccade – vergence movements in a young population with vertigo with normal vestibular function and a vergence abnormality in the results of orthoptic tests. In the second part of this study we address the question whether orthoptic vergence training can improve the latency of eye movements, and particularly vergence eye movements.

Methods

Selection of the patients.

The patients included in the study had the following characteristics: completely normal responses to the vestibular tests, normal neurological and otological clinical examination but one to three abnormal values at the orthoptic tests (see Table 1 A). They all were referred to have the vestibular tests for vertigo, and headache was a secondary symptom associated or not with the vertigo.

Although the symptoms were initially described as vertigo, the reported sensations were in fact sometimes rotation, but also displacement of the environment, rolling or translation. They were never intense rotatory vertigo lasting for hours but rather brief sensations related to movements of the head or the gaze. Their timing was often related to fatigue (at school or at the end of the day, or in the evening) and often after long exposure to computer or television screens.

Twelve subjects, between 6 and 15 years old participated in the study. The investigation adhered to the principles of the Declaration of Helsinki and was approved by our institutional human experimentation committee. Informed parental consent was obtained for each subject after the nature of the procedure had been explained.

Vestibular tests

All subjects underwent a complete vestibular, otological and neurological examination. Clinical vestibular examination was always completed with a quantitative vestibular function evaluation (canal and otolith vestibular function) including several tests which had been adapted to children and fully explained and detailed in previous articles [31, 32]. The vestibulo-ocular responses were recorded by the electro-oculography technique during canal vestibular stimulations (caloric test, earth vertical axis rotation, and pendular rotation) and during otolith vestibular stimulations (off vertical axis rotation) by means of a computer controlled rotating chair in a completely dark room [30–32]. The results of all these tests were normal and are listed below.

All eye movements recorded during vestibular tests were monocular at fixed viewing distance and were run on a different day from the binocular saccades and vergence eye movements recording (see oculomotor tests).

The tests performed and normal results were as follows. For the caloric test: relative directional preponderance < 15 %, relative reflectivity < 15 %. For the earth vertical axis rotation: maximum initial slow phase velocity symmetrical for clockwise and counterclockwise rotation and equal to 40 ± 10°/s- time constant symmetrical (dependent of age, varying from 8 s to 20 s for 6 to 15 years old subjects). For the pendular rotation: responses symmetrical in dark and completely inhibited by the fixation of a lead target. For the off vertical axis rotation: horizontal directional preponderance (< 0.35 ± 0.30°/s), vertical directional preponderance (< 0.90 ± 0.69°/s) and horizontal modulation amplitude (3.0 ± 1.4°/s) vertical modulation amplitude (4.8 ± 3.3°/s). Conventional hearing tests (tonal and speech audiometric techniques) were also performed to assess the function of the inner ear and all patients had normal hearing.

Orthoptic tests

Subjects underwent complete ophthalmological and orthoptic examination that is summarized next. All subjects had normal binocular vision, evaluated with the TNO random dot test for stereoscopic depth discrimination (the threshold of stereoacuity was 60 s of arc or better). No child wore spectacles and their visual acuity was 8/10 or better in both eyes. Orthoptic evaluation of vergence (made by using the cover test, bar of prisms, Maddox rod and synoptophore technique) revealed, for all subjects, one or more signs of vergence abnormalities (see Table 1 A, children examined and Table 1 B, normal children); e. g. distant near point of convergence (≥ 10 cm), exophoria (i. e. latent deviation of one eye when the other eye is covered) at far viewing that was larger than 3 Δ, and exophoria at near viewing larger than 6 Δ; the range of fusional vergence at far and at close viewing was also limited. According to Rouse et al. [23, 24] and Rainey [22] subjects S1, S3–S7, S10 and S12 were classified as having convergence abnormalities while subjects S2, S8, S9 and S11 had divergence abnormalities.

Orthoptic training

For all subjects vergence abnormalities were treated with twelve sessions (three times per week) of orthoptic training that consisted in several exercises (e. g. converging and diverging the eyes to follow a small pen-light or a letter moving in depth) in order to improve the range of vergence fusional amplitude [see for details www. childrensvision.com].

Eight of the subjects (S1, S3, S4, S7, S9, S10, S11 and S12) had a second orthoptic clinical examination 1–2 weeks after the end of the twelve sessions of orthoptic vergence training.

Oculomotor testing

Visual display. Two isovergence circles horizontally placed on a surface were used one at 20 cm and the other at 150 cm from the subject. The target used was a LED; on the circle close to the subject three LEDs were placed; one at the center and the other at ± 20°. The required mean vergence angle for fixating any of these three LEDs was 17°. On the circle far from the subject, five LEDs were placed: one at the center, two at ± 10° and two at ± 20°; fixation to any of these LEDs required a vergence angle of 2.3°. A computer directed the target-LED presentation.

Table 1A Clinical Characteristics of children tested before and after reeducation of vergence by orthoptic exercises. NPC (near point of convergence), heterophoria (in prism diopters), vergence fusion amplitude (in prism diopters) clinically measured. The vertigo symptoms (rotatory, translation rolling sensations and unsteadiness) associated or not with headache are also reported. Bold characters indicate abnormal orthoptic values (relative to references shown in Table 1B)

			Before						After orthoptic vergence training				
Subject (years)	Visual acuity	Stereo acuity (TNO)	NPC (cm)	Hetero-phoria (pD)	Diver (pD)	Conver (pD)	Vertigo: subjective sensations	Headache	NPC (cm)	Hetero-phoria (pD)	Diver (pD)	Conver (pD)	Vertigo and/or headache
S1 (6)	LE: 10/10 RE: 10/10	60″	6	far 2 E **near 6 E**	far 6 **near 6**	**far 6** near 20	rotatory, unsteadiness		6	far ortho near ortho	far 6 near 18	far 30 near 40	–
S2 (8)	LE:10:10 RE: 10:10	60″	6	**far 1 E** near 1 X	**far 3 near 10**	far 14 near 20	rotatory						
S3 (10)	LE: 10/10 RE: 9/10	60″	6	far ortho **near 8 X**	far 4 **near 10**	far 10 near 30	rotatory		6	far ortho near 2 X	far 6 near 18	far 40 near 40	–
S4 (11)	LE: 10/10 RE: 10/10	60″	**15**	far ortho **near 8 X**	far 4 **near 6**	far 14 near 20	rotatory	+	6	far ortho near 4 X	far 8 near 18	far 18 near 40	–
S5 (11)	LE: 10/10 RE: 10/10	60″	6	far ortho near 2 X	far 4 **near 14**	far 14 near 18	rotatory	+					
S6 (12)	LE: 8/10 RE: 8/10	60″	6	far ortho **near 8 X**	far 6 near 8	far 8 near 20	translation, unsteadiness	+					
S7 (12)	LE: 10/10 RE: 10/10	60″	**10**	far ortho near 2 X	far 4 near 6	far 6 **near 10**	rotatory, unsteadiness	+	6	far ortho near ortho	far 10 near 20	far 20 near 40	–
S8 (12)	LE: 10/10 RE: 10/10	60″	6	**far 4 E** near 2 X	**far 1 near 4**	far 18 near 20	rotatory	+					
S9 (12)	LE: 10/10 RE: 10/10	60″	**10**	**far 10 X** near 6 X	far 10 **near 12**	**far 6** near 20	unsteadiness		6	far 2 X near 2 X	far 10 near 18	far 20 near 40	–
S10 (14)	LE: 10/10 RE: 10/10	60″	**10**	far ortho near 4 X	far 14 **near 12**	**far 6** near 30	rolling	+	7	far ortho near 4 X	far 8 near 18	far 18 near 30	–
S11 (14)	LE: 10/10 RE: 10/10	60″	6	**far 12 X** near 6 X	far 4 **near 14**	**far 10** near 26	rotatory		4	far ortho near ortho	far 6 near 18	far 40 near 40	–
S12 (15)	LE: 10/10 RE: 10/10	60″	**10**	far ortho near 4 X	far 4 **near 10**	**far 4** near 26	rotatory	+	6	far ortho near 4 X	far 4 near 20	far 40 near 40	–

Table 1B Normal values of near point of convergence (NPC), of heterophoria and of the range of convergence and divergence amplitude at far and close distance measured in children of 6–15 years old; values based on the studies of Evans [8], von Noorden [29], and Ygge et al. [34]

NPC (cm)	Heterophoria range (pD)	Divergence range (pD)	Convergence range (pD)
< 6	far 3 X-1 E near 6 X-ortho	far 4–10 near 17–25	far 11–17 near 15–27

Eye movement recordings. Data collection was directed by REX, software developed for real-time experiments. Horizontal eye movements from both eyes were recorded simultaneously with a photoelectric device (OCULOMETER, BOUIS). This system has an optimal resolution of 2" of arc. Eye-position signals were low-pass filtered with a cut off frequency of 200 Hz and digitized with a 12-bit analogue-to-digital converter. Each channel was sampled at 500 Hz.

Procedure of oculomotor testing. Subject was seated in a chair, which could be adjusted for height, with the head stabilized by a forehead and chin support. The experiment was run in a completely dark room to ensure that stimulus detection and localization was quasi automatic, avoiding distraction from other objects [16].

The subject viewed binocularly and faced the surface containing the LEDs so that the close isovergence circle was at 20 cm from her/his eyes.

Each trial started by lighting a fixation LED at the center of one of the two circles (far or close). After a 2.5 s fixational period the central LED was turned off and a target-LED appeared for 2 s. When the target-LED was at the same circle it called for a pure saccade (rightward or leftward). When it was on the center of the other circle it called for a pure vergence eye movement along the median plane (convergence or divergence), and when it was lateral and on the other circle the required eye movement was a combined saccade and vergence eye movement. The required saccade amplitude was always 20°, and the required vergence change along the median plane (for pure vergence) or along lateral axes (for combined movements) was 15°. In each block, the three types of trials were interleaved randomly. Each block contained 24 trials, i. e. four saccades at far, four saccades at close, four pure convergence, four pure divergence, four saccades combined with divergence, and four saccades combined with convergence. For the majority of subjects four blocks were run. At the beginning and at the end of each block, a calibration task was run: subjects followed a target-LED that stepped from the center to ± 10° and ± 20° at the far isovergence surface.

Saccades, vergence and combined movements were recorded for all twelve subjects, and for eight of them, the same oculomotor recording was also done 1–2 weeks after the end of the orthoptic vergence training.

Data analysis

The analysis methods are similar to those used in prior studies [17, 33]. Briefly, a linear function was used to calibrate the individual eye position signals. From these two signals, we calculated the saccade signal [(left eye + right eye)/2] and the vergence signal (left eye – right eye). Fig. 1 shows an example of pure rightward saccade (A), pure convergence movement (B) and a saccade movement combined with convergence (C). For each type of eye movements the saccade or conjugate component and the vergence or disconjugate components are shown. For pure convergence (Fig. 1B) the position trace from the individual eye which gives information about the symmetry of the response from the two eyes have been added.

Markers indicating the start of the movement were placed on each eye position signal automatically, and were verified by an investigator. The criteria used are standard: the onset of the conjugate saccadic component was defined as the time when the eye velocity reached 5 % of the saccadic peak velocity. The onset of the vergence signals (for pure vergence movements and for the vergence component of the combined movements) were defined as the time point when the eye velocity exceeded 5 %. Eye movements that were in wrong direction or contaminated by blinks were rejected. Also, eye movements with latencies shorter than 100 ms or longer than 1000 ms were rejected. Note that the percentage of the rejected movements before training was between 10 %–40 % for pure vergence movements, 6 %–34 % for combined movements and 0 %–9 % for pure saccades; after training the percentage of rejected movements was between 0 %–34 % for pure vergence, 0 %–13 % for combined movements and 0 %–5 % for pure saccades. After training an increase of the percentage of the movements analysed was observed for subjects S1, S3 and S10, but not for subjects S4, S5, S6, S12.

For each type of eye movement (saccade, vergence and combined movements) we measured the latency in ms, that is the time between the onset of the target-LED and the beginning of the movements. For combined movements latency was measured for each component (saccade, vergence component). Analysis of variance (ANOVA) was performed with subject as a random factor and the latency of different types of eye movements as a fixed factor. The group mean latency of each type of eye movements before and after the orthoptic vergence training was compared with the mean latency values from normal subjects of comparable age extracted from the study of Yang et al. [33]. Statistical comparison was done by using the Student's t-test.

Results

Pure saccades and vergence movements

Fig. 2A shows the individual mean latency for pure saccades at far and close viewing distance. For all subjects (except S4 and S5) the latency was shorter for the saccades at near distance. The group mean latency was

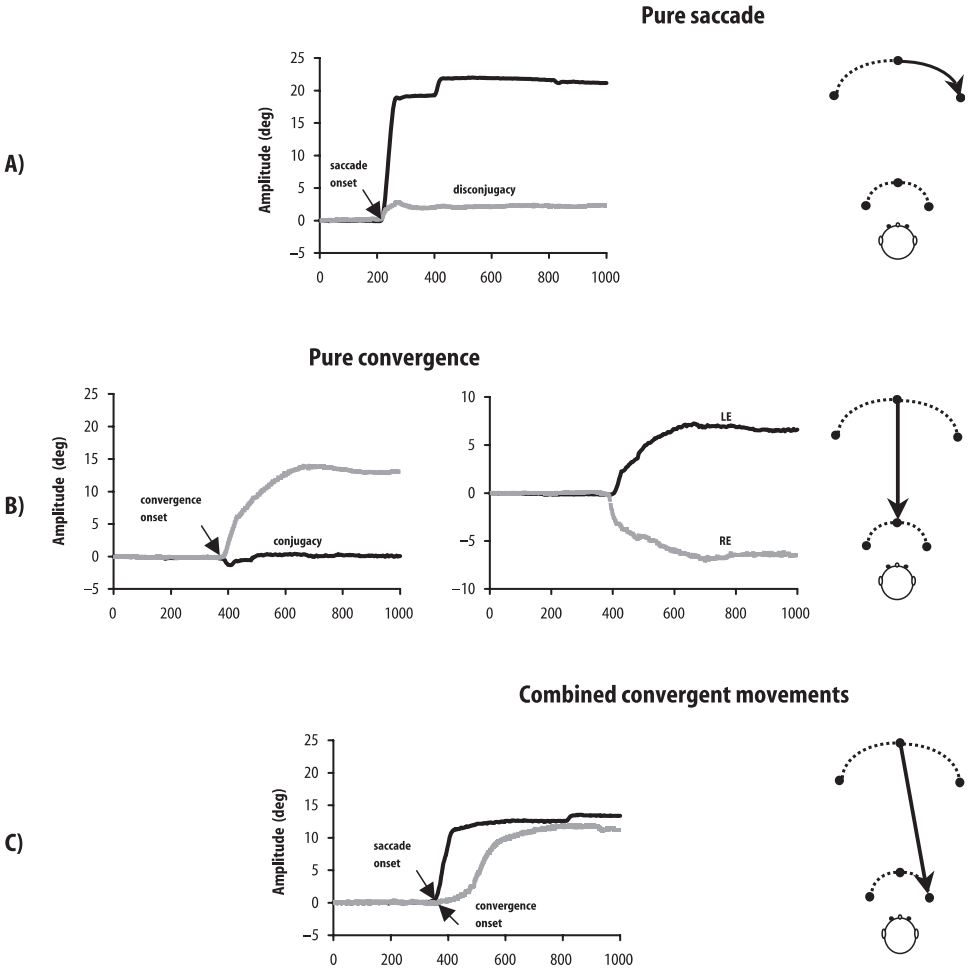

Fig. 1 Examples of eye movements. On the right side is shown the excursion of the target. The saccade or conjugate signal is the average of the position signal of the two eyes [(LE + RE/2)]; the vergence or disconjugate signal is the difference between the two signals (LE – RE). For pure convergence, the position trace of each eye is also showed. The target-LED appeared at time zero. The arrows indicate the onset of the movement. (**A**) Pure rightward saccade; (**B**) Convergence movement along the median plane; (**C**) Rightward saccade combined with convergence. Note that the saccade component starts before the vergence component

290 ± 26 ms and 248 ± 17 ms for saccades at far and at close distance respectively and this difference was significant ($F_{1,11} = 8.91$, $p < 0.012$).

Fig. 2B shows the latency of convergence and divergence eye movements. For all subjects convergence had longer latency than divergence. The group mean value was 369 ± 31 ms and 257 ± 21 ms for convergence and divergence movements respectively; the difference was significant ($F_{1,11} = 27.96$, $p < 0.0003$). The latency of convergence was also longer that the latency of saccades at far ($F_{1,11} = 17.25$, $p < 0.001$), and of saccades at close distance ($F_{1,11} = 36.28$, $p < 0.0001$).

According to the orthoptic clinical tests vergence abnormalities can be of different origin (i. e. convergence versus divergence abnormalities). Do such hypothetical abnormalities produce different deficits in convergence and divergence latency, respectively? The answer is no: indeed, we found that all our subjects had longer latency for convergence than divergence regardless of the type of pathology, i. e. convergence versus divergence abnormalities. In subjects with convergence abnormalities (S1, S3–S7, S10 and S12) convergence latency was 133 ms longer than divergence latency, while in subjects with divergence abnormalities (S2, S8, S9 and S11) this difference was smaller (about 71 ms); however, these values were not significantly different ($p = 0.19$). Thus, we suggest that the lengthening of the latency was not specific to the sub-type of vergence abnormality determined by orthoptic tests.

We also explored whether the severity of subjective complaints was correlated with abnormal longer vergence latencies, and we compared the latency of convergence and divergence in subjects complaining only of vertigo (S1, S2, S3, S9 and S11) and in subjects complaining of both vertigo and headache (S4–S8, S10, S12). On average, the latency of convergence was the same (369 ms) in subjects with vertigo and headache as well as in subjects with only vertigo; the latency of divergence was longer in subjects with only vertigo (303 ± 75 ms) than in subjects with vertigo and headache (223 ± 34 ms); however, such difference was not significant. Consequently, next we will present latencies of different types of eye movements without further insistence on the clinical orthoptic classification and on subjective symptoms.

Latency differences relative to normals

The average latency of our subjects was compared with the average latency of normal children of matched age extracted from the study of Yang et al. [33]; normal latency values are shown in Fig. 2 by horizontal lines. For saccades at far and close distance respectively, latency was longer relative to the mean normal value plus one standard deviation for ten and six subjects, respectively. The Student's t-test on the group means showed a significant difference between our subjects and normals for both saccades at far ($p < 0.01$) and saccades at close distance ($p < 0.05$). For convergence, all subjects showed abnormal longer latency and for divergence, eight sub-

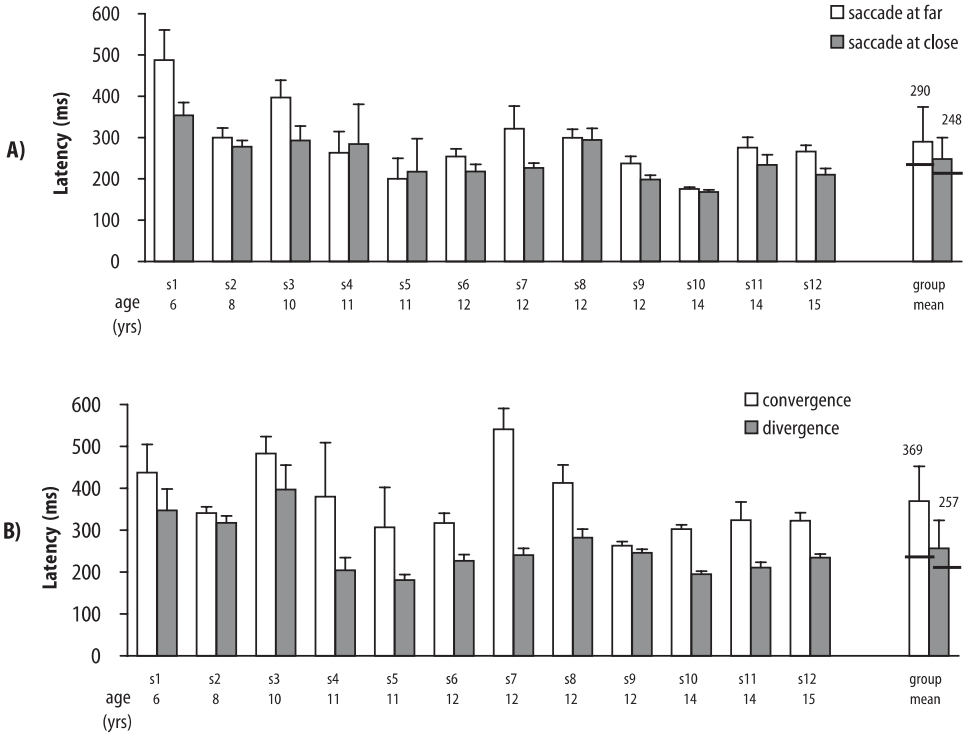

Fig. 2 (**A**) Mean latency of saccades at far (white bars) and at close (black bars) distance for each subject tested. Leftward and rightward saccades are grouped together; (**B**) Mean latency of convergence (white bars) and divergence (black bars) along the median plane. Vertical lines indicate the standard error. Group means are based on twelve subjects. Horizontal lines on the group means correspond to the latency value found in normal subjects of comparable age [33]

jects had latency longer than normal values. Similarly to saccades, the group means of convergence latency as well as divergence latency were significantly different from those of normals ($p < 0.01$).

Combined saccade – vergence eye movements

Fig. 3A shows the latency of the saccade component of combined convergent and divergent movements. For the majority of the subjects (9/12) the latency of saccades was longer when the saccade was combined with convergence than when combined with divergence: the group mean value was 384 ± 24 ms and 329 ± 33 ms respectively ($F_{1,11} = 5.79$, significant at $p < 0.03$).

The latency of the convergence and divergence component of combined movements is shown in Fig. 3B. For eight subjects (S4, S5, S6, S7, S8, S10, S11 and S12) the latency of the convergence component of combined movements was longer than that of the divergence component: the group mean value was 376 ± 25 ms and 324 ± 30 ms respectively. The difference was statistically significant ($F_{1,11} = 7.15$, $p < 0.02$).

Latency differences relative to normals

With respect to normals, latency of the saccade component of combined movements was longer for all our subjects (except S5 and S10, saccades combined with divergence); for vergence components, abnormal longer latency was observed for all subjects (except for S5 for divergence component). The Student's t-test applied for each of these two components on the group mean of our subjects and those of normals showed a difference that was always highly significant ($p > 0.01$).

In summary, in children with vertigo, similarly to normals, latencies depend on the type of eye movements: i. e., latency of saccades at far is longer than that of saccades at close, latency of convergence is longer than that of divergence, and latencies of combined movements are longer than those of pure movements. The most important observation is that all these latencies, particularly those of vergence and combined eye movements are abnormally longer relative to normal children.

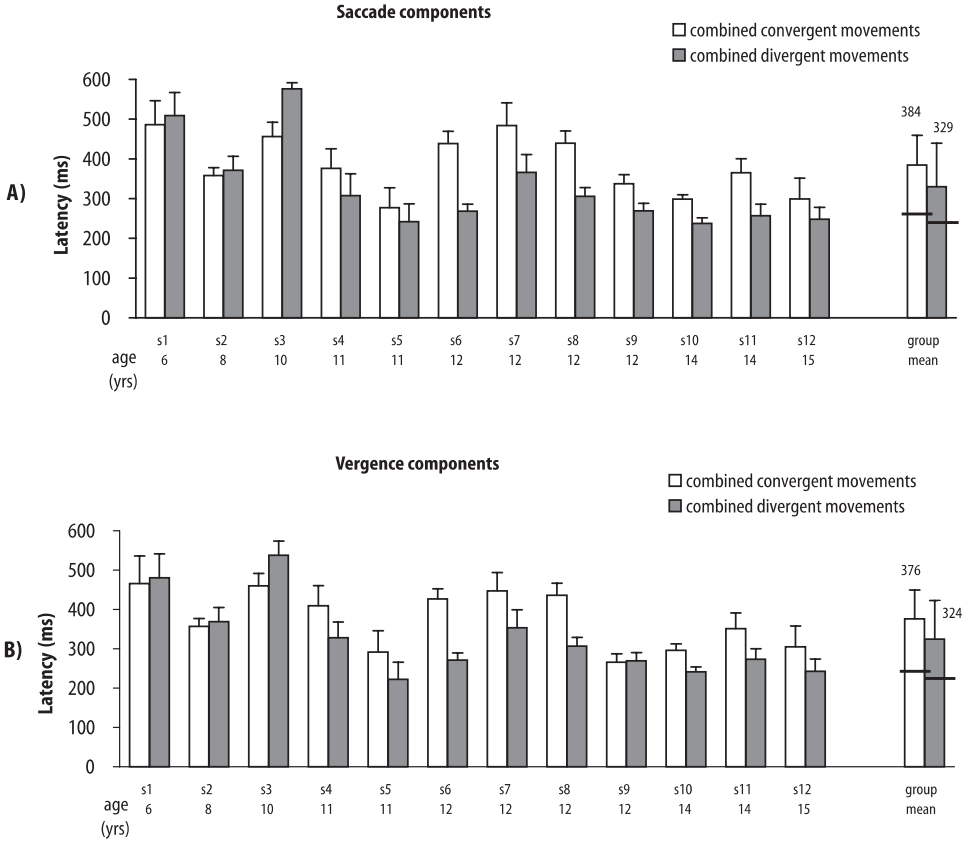

Fig. 3 (**A**) Mean latency of the saccade component of movements combined with convergence (white bars) and divergence (black bars) for each subject; (**B**): Mean latency of convergence (white bars) and divergence (black bars) component of combined movements. Other notations than in Fig. 2

Additional observation: effect of orthoptic reeducation

Effects on vestibular and orthoptic tests (see Table 1A, After training)

All eight subjects did not feel vertigo after training. Moreover, the range of vergence improved for all subjects. Indeed, the near point of convergence that before training was 9 ± 3 cm reduced to 5 ± 0.8 cm after training; the exophoria at close viewing that was before training 6 ± 2.4 pD reduced to 1.7 ± 1.6 pD after training. Finally, the convergence amplitude at close distance improved: from 21 ± 7 pD before training to 39 ± 3 pD after training.

Effect on latency

Fig. 4 shows the group means of latency before and after orthoptic vergence training for saccades at far and close distance (A), for convergence and divergence (B), and for the saccades (C) and the vergence components (D) of combined movements, respectively. Before training, the mean latency value of all these types of movements was significantly longer (t-test significant at $p < 0.01$) than the mean latency observed in normals (showed by the horizontal line on the bar graphs).

After training the mean latency was shortened: for saccades at far distance the ANOVA applied on the mean latencies before and after orthoptic vergence training tends to be significant ($F_{1,7} = 5.33, p > 0.057$); in contrast, for saccades at close distance that reduction was not significant ($F_{1,7} = 1.25$, $p = 0.25$). Note, however, that after training, the average latency of both saccades at either distance was not different from normal values (t-test, $p = 0.05$).

The training reduced substantially the latency of convergence by 140 ms ($F_{1,7} = 10.65$, $p < 0.01$), while the latency of divergence decreased by a small amount (only 9 ms); the change was not significantly different ($F_{1,7} = 0.42$, $p = 0.54$). Importantly, the mean latency of both convergence and divergence observed after the training (see Fig. 4B) was still significantly longer than normal values (t-test, significant at $p < 0.01$).

After training, the latency of the saccade as well as of the vergence components of combined movements decreased significantly (Fig. 4C and 4D). The effect was significant for both saccades combined with convergence

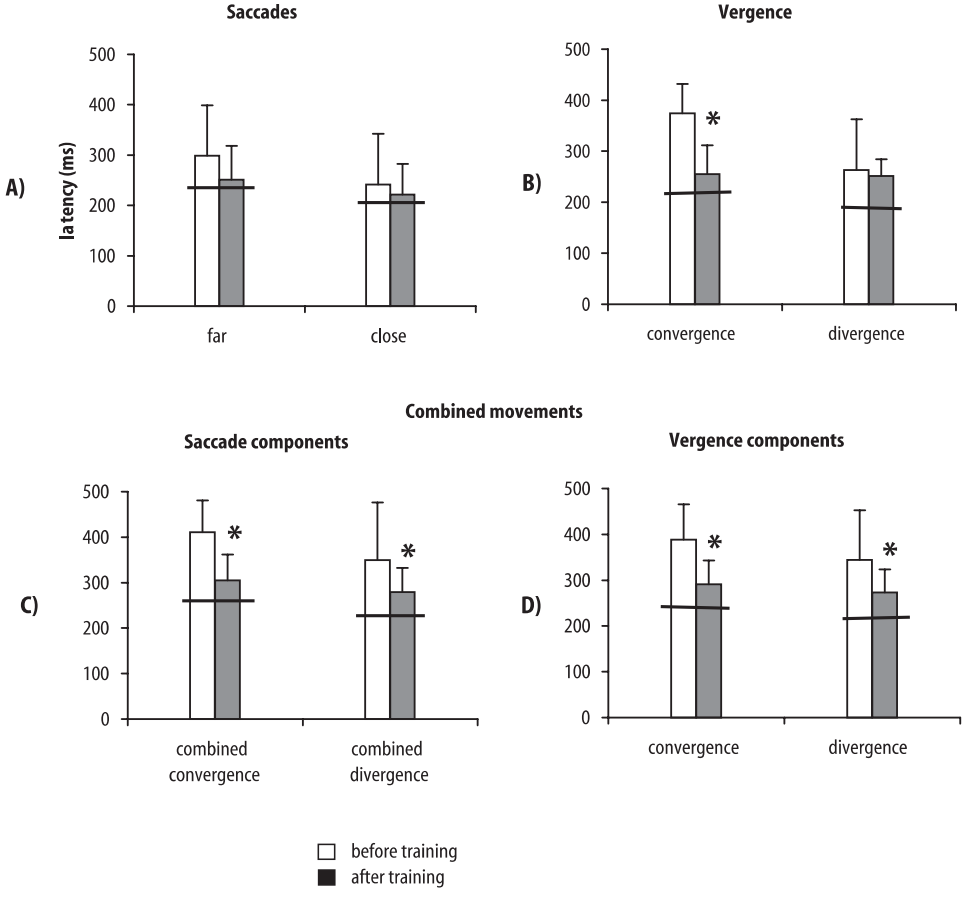

Fig. 4 Group mean (on eight subjects) of the latency before and after orthoptic vergence training for saccades at far and close distance (**A**), for convergence and divergence (**B**), and for combined movements (saccade components **C** and vergence components **D**). Vertical lines indicate the standard error. A significant before-after training change in the latency value is shown by an asterisk. The horizontal lines indicate latency values recorded in normal subjects of matched age [33]

and divergence ($F_{1,7} = 20.79$, $p < 0.003$ and $F_{1,7} = 6.21$, $p < 0.02$, respectively), and for both, convergence and divergence components ($F_{1,7} = 23.16$, $p < 0.002$ and $F_{1,7} = 8.97$, $p < 0.02$, respectively). Note, however, that all latencies of combined movements after training were still longer than normal values (t-test, significant at $p < 0.01$).

In summary, the orthoptic vergence training shortened the latency of all eye movements, but the decrease was significant only for convergence and combined movements. Importantly, after training the latency of saccades at far and close distance was similar to that of normals; in contrast, the latency of vergence and combined movements did not reach normal values.

Discussion

Latency differences between types of eye movements

As in normals, in children with vertigo, latencies of various types of eye movements were different: the latency of saccades at close was shorter than that of saccades at far distance; the latency of convergence movements was longer with respect to that of divergence but also with respect to all types of saccades (at far and close distance). Combined movements also showed long latency, particularly when combined with convergence; the latency of both saccade and vergence components of combined movements were longer than those of the corresponding pure movements. These results will be briefly discussed below.

Saccades latency: effect of distance

The shorter latency of saccades at near distance has been observed also in another study on normal children and adults using the same experimental conditions [33] and is compatible with other studies examining the far [11] or the close distance [27]. As suggested by Yang et al. [33] the shorter latency at close distance could be due to sensory, motor or attentional factors.

Interpretation of the different latencies observed between convergence and divergence

Longer latency for convergence than for divergence movements was also observed in normal adults by Krishan et al. [18] and in some subjects by Takagi et al. [27]; however, other studies [15, 25] showed opposite results. These differences could be due to different experimental procedures used. More convincing is the study of Yang et al. [33] who showed systematically longer latency for convergence for both normal children and adults using the same experimental conditions of the present study. Our findings for subjects with vertigo are compatible with this study [33].

Such latency difference between convergence and divergence suggests different neuro-physiological processes involved in the preparation of these movements. The cortical substrate of vergence in humans is not yet well explored. Hasebe et al. [13] showed with a PET study activation in the parietal and temporo-occipital cortex in relation to vergence eye movements; Kapoula et al. [17] showed that transcranial magnetic stimulation of the posterior parietal cortex lengthened the latency of both saccades as well as vergence eye movements. More importantly, Tzelepi et al. [28] showed higher amplitude of EEG responses in the parietal areas for stimuli calling for convergence than for divergence or for saccades. All these observations suggest that several cortical areas control the triggering of vergence, and that the cortical circuitry for the initiation of convergence and divergence movements seems to be not absolutely identical.

Combined movements: longer latency

The increased latency of combined movements with respect to that of pure movements could be due to the additional time needed to prepare two or more movements rather than one. This interpretation is compatible with other studies examining sequences of saccades reporting increase of latency as the number of the targets increases [3].

Differences of latencies from normal population

As mentioned in Methods, we compared the data in children with vertigo with those of children without disorders tested under identical conditions [33]. The latency of saccades at far and close distance was 50 ms and 10 ms respectively longer in our subjects than in normal children of comparable age. More interesting is that the latency of vergence was 145 ms and 50 ms (for convergence and divergence, respectively) longer than those of normals. The latency of the saccade component of combined movements was 115 ms longer than normal; finally, the latency of convergence and divergence component of combined movements was 130 ms and 110 ms longer than that of normal children.

Thus, the movements that have naturally longer latencies are the ones that are more severely affected in subjects with vertigo. In general, for normal subjects, the longer is the latency of a movement, the most volitional the movement is believed to be [10]; on the other hand, in patients with cortical lesions latencies increase because of cortical dysfunction [21]. Thus, our findings suggest the presence of a central deficit in the initiation of eye movements, particularly for convergence and combined movements, which are, perhaps, the most volitional movements.

Effect of orthoptic vergence training

Orthoptic vergence training is widely used by clinicians to treat vergence abnormalities; moreover, it is well known [e. g., 6, 7, 12, 19] that even normal subjects can benefit from orthoptic training: indeed these authors pointed out in normal subjects improvement of vergence performance after orthoptic training. Note also that for normals, even if the training procedure was similar for all subjects, the improvement due to the training was variable between the subjects [19].

Nevertheless, objective eye movement studies showing the effect of the orthoptic training are rather scarce. The study by van Leeuwen et al. [19] is, to our knowledge, the only one showing an increase, after orthoptic vergence training, of the amplitude of vergence (pure and combined with saccades) in normal subjects as well as in one subject with convergence insufficiency.

Our findings show, for the first time, that orthoptic vergence training can: (i) eliminate vertigo; (ii) improve vergence capabilities clinically assessed; (iii) shorten latency of eye movements. The decrease of the latency was significant for the movements, which before training had the longer latency, i. e., pure convergence eye movements and both components of combined movements.

As mentioned in the Introduction, latency reflects several processes, such as disengagement of attention, of oculomotor fixation, and computation of the metric of the movement [9, 10]. Thus, shortening of the latency could be mediated by shortening of the time needed by one or several of these sub-processes. Namely, attentional factors could be sensitive and modulated by training or by visual experience. Our data do not allow us to determine if the latency decrease is due to the orthoptic training itself or to spontaneous improvement related to natural visual experience. Our findings are compatible with the study of Fischer [10] who reported in a normal subject significant decrease of saccade latency after training; the training consisted of a repetition of 200 saccades per day for a period of 10 days. The author suggested that training helped the subject to disengage the attention more rapidly. Consequently, we conclude that normal subjects as well as subjects with vergence abnormalities can benefit by the training and that attention can be responsible for the decrease of eye movement's latencies.

Finally, one could argue that latencies in children with vertigo before training were simply extreme values, that is, a little standard deviation from normals, and that training brought these values back to normals. This, however, was not the case. As we pointed out, despite the reduction of latencies for all types of eye movements after training, latencies remained still longer than those of normals, particularly for vergence and for combined movements (see Fig. 4). Hence we suggest that children with vertigo have a specific problem with the initiation of vergence and combined movements. This deficit can be reduced by attentional learning mechanism but cannot be eliminated completely.

Vergence initiation abnormalities and vertigo

During head movements or body motion, the vestibulo-ocular system generates compensatory eye movements that stabilize images on the retina. Several electrophysiological and behavioral studies in the last ten years have shown that the responses generated by the vestibulo-ocular reflex (VOR) are a complex function of head movement and target distance [20]. The modulation of the gain of the vestibular system by the viewing distance is an aspect integrated in most recent models of vestibular ocular function. The CNS uses knowledge (proprioceptive input or corollary discharge related to vergence angle) to adjust the gain of the VOR as a function of the viewing distance. Importantly, Snyder et al. [26] have reported that during vergence eye movements the change of the gain of the VOR anticipates the vergence, which suggests the possibility for predictive influence of vergence on the VOR system. Coenen [5] proposed a model in which the cerebellum could construct by learning a predictive vergence input to control the VOR gain. Because of the abnormally longer vergence latency in our subjects the contribution to the VOR adjustment could be reduced or inappropriate. Consequently, we suggest that inappropriate vestibular function relative to distance could lead to vertigo and headache as reported by Brandt [4]. However, this hypothesis should be confirmed further by quantitative studies examining the link between the quality of vergence control, namely the slowness of its initiation, and the gain of the VOR at different viewing distances. This field is to our knowledge unexplored both in normals and in subjects with vertigo.

Clinical considerations

The study of Anon-Tanon et al. [1] suggested for the first time that vertigo is associated with abnormal results in subjective orthoptic evaluation of vergence. Our study substantiates this report as it shows abnormal vergence latency in such subjects. The two studies are compatible and indicate that orthoptic testing of vergence is valuable even though it cannot provide temporal information about eye movements. Objective recording of eye movements together with clinical orthoptic tests is the most useful approach for a fine diagnosis and treatment of children with vertigo.

Finally, it should be noted that for a long time, vergence abnormalities have been thought to be related to peripheral muscular deficiency only [29]. Our study

suggests that vergence abnormalities such as slowness of vergence initiation are most likely due to dysfunction of the cortical circuitry that controls the initiation of such movements.

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