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Repeated optokinetic stimulation in conditions of active standing facilitates recovery from vestibular deficits

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Abstract Successful results obtained by training sessions using optokinetic (OK) stimulations in order to rehabilitate patients with balance disorders motivated this study. The purpose of the study was to measure eye movement parameters and body stabilization during OK stimulation before and after the rehabilitation program. Two populations of patients were studied: bilateral and unilateral labyrinthine-defective patients. Before training, the OK nystagmus (OKN) showed irregularities of the slow-phase velocity (SPV) as well as a reduced number of beats (frequency) when compared with a control group of age-matched healthy volunteers. After training, the SPV became more regular (decrease in SD) and the frequency was similar to the control group's (3 Hz). Body stabilization was measured by dynamic posturography (Equitest) at the beginning and at the end of the training program. At the end of the training program, the patients were asymptomatic and there was a significant correlation between the Equitest results and the modification of the OKN parameters.

Key words Optokinetic nystagmus
Optokinetic stimulation · Postural sway
Body stabilization · Labyrinthine-defective patients
Human

Introduction

Complex, repeated optokinetic (OK) stimulation in standing humans has been used successfully for the treatment of unsteadiness in elderly patients with presbyastasia (Sémont et al. 1992). The same training protocol has been used with encouraging results to suppress motion sickness encountered by aircraft pilots and commercial airline flight attendants (Vitte et al. 1992).

The purpose of this study was to understand the mechanisms underlying this surprising beneficial effect. We hypothesized that the improvements of postural control could be mediated by a decrease in retinal slip due to a recovery of a good OK nystagmus (OKN) control.

It is well known that a moving visual environment induces in humans an OKN response (Ter Braak 1936; Dubois and Collewijn 1979; Collewijn 1985; Van Den Berg and Collewijn 1988) made of a slow component in the direction of the movement of the visual field (due to a combination of pursuit and of true OK reflex action, Robinson 1978; Leigh and Zee 1991) and a fast phase in the opposite direction. The quantitative parameters of OKN and the effect of visual scene characteristics are well documented (Zee et al. 1976; Tjissen et al. 1989).

In addition to these two dynamic eye movements, two slow processes have been described: a shift of the beating field toward the direction of the fast phase, which is probably an anticipatory mechanism, and a slow build-up of slow-phase velocity (SPV), which is acompanied by a storage of this velocity leading to an OK afternystagmus (OKAN; Cohen et al. 1977; Jell et al. 1984). This OKAN can be measured in darkness after the exposure to the OK stimulus for a certain time. The influence of vestibular stimulation on OKN demonstrates the close link between these two oculomotor subsystems (Waespe and Henn 1977; Cohen et al. 1981; Koenig et al. 1991).

The effect of unilateral vestibular lesions on optokinetic nystagmus have been previously studied in humans (Zee et al. 1976; Baloh et al. 1982; Blakley et al. 1993). The disappearance of OKAN after labyrinthine lesions is a general and well documented effect (Zee et al 1976). OKAN has not been studied in the present work.

In addition to eye movements, it has been shown that the movement of large visual scenes induced postural

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reactions during both circular (Lee and Aronson 1974; Dichgans and Brandt 1978; Van Asten et al. 1988) and linear (Lestienne et al. 1977; Nashner and Berthoz 1978) motion. Generally the postural sway is observed in the direction of the slow phase of nystagmus. This postural sway has been attributed to at least three mechanisms:

1. A direct action of the visual input on the structures controlling posture, for instance visual input to the vestibular nuclei
2. An indirect action through the central representation of body orientation to the vertical
3. A consequence of the coupling between eye movement and neck muscles

In the present work we explored two series of parameters in a population of patients with unilateral or bilateral loss of the vestibular function. These results were compared with those of a population of age-matched healthy volunteers considered as the control group.

Firstly we measured the modifications of OKN SPV per stimulation, the number of nystagmus beats during a 10-s period, in order to obtain a frequency value; we observed the modification of the beating field shifts but we did not quantify it during the recording sessions. Recordings were carried out before and after a rehabilitation program consisting of OK exposure sessions.

Secondly we measured the overall stability of the same population of patients at the beginning and at the end of the training program. We shall describe the parallel evolution of the improvement of balance and of OKN.

Materials and methods

OK

The OK device was a planetarium (Simpson et al. 1981) mounted on a three-axis system in order to project different stimulation patterns. Walls, ceiling, and floor of a completely dark room were used as screens. On the extreme left and right of the frontward screen were two black horizontal bars (with a dihedral angle of 160°), which gave a stable reference to enhance "vection." The three axes of rotation (x, y, z) could be used separately or together in order to induce complex patterns and go from pattern to pattern in a smooth continuous change in direction without stopping the stimulation. The rotation of the sphere with respect to the z-axis induced a horizontal drum-like pattern of white dots; the y-axis, a vertical upward or downward moving pattern; the x-axis, a torsional pattern.

In this study, the eye movement recordings were done with the z-axis of the sphere tilted 45° backward in order to have a complex pattern (Fig. 1). The provoked OKN was horizontal with probably a slight torsional component, too small to be measured but enough to enhance vection. This complex pattern was chosen because patients reported having more difficulty enduring the slightly curved stimulus than a pure horizontal stimulus. The upright standing subject was at a distance of 2 m from the frontal "nearest wall," with the OK device on his right side at head level. The angular velocity of the sphere was 40°/s and the temporal frequency of dot presentation was 5.55 Hz. The angle between each projected white dot was 7°55'.

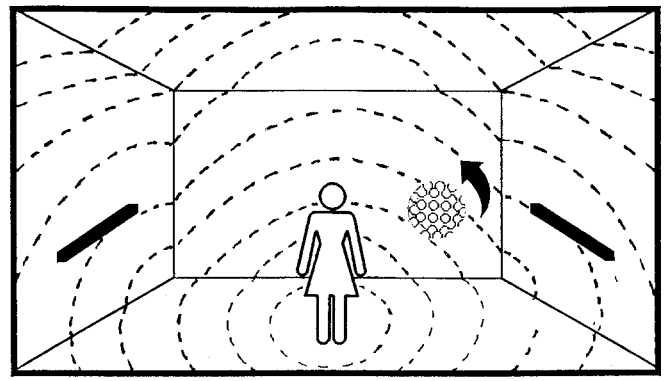

Fig. 1 The standing subject is shown in the room with walls as screens. The *two black bars* to enhance "vection" are seen on each side. The *curved dotted lines* represent the opokinetic stimulus on the front, nearest wall, side walls, ceiling, and floor

OK stimulation sessions (rehabilitation)

Sessions of rehabilitation using OK stimulations were performed as follows:

A. The subject was standing in an upright orthostatic position. He or she was instructed to look at the passing dots and let the eyes move freely (stare OKN) without moving the head, and try to keep his balance. The stimulation sessions started with the stimulus which was used for the recordings of the present study; that is, horizontal with slight torsional component stimulus at an angular velocity of 40°/s. The stimulus was kept until the subject began to sway and stopped before the off-limit of stability was reached. The direction was then changed to the opposite direction without modification of the velocity for a few seconds and set back to the disabling direction until the subject once more overbalanced, and so forth.

B. Pure vertical stimulus (with the z-axis of the sphere horizontal) was then used with the same protocol. Upward or downward direction was chosen in accordance to its effect on vection and body sway; the most destabilizing direction being the one that had to be done again and again.

Duration of the sessions never went beyond 15 min. The first sessions were usually shorter, stopped because of patient fatigue due to the numerous postural readjustments. The necessary number of sessions to obtain an asymptomatic patient averaged 8 ± 2. The number of sessions increased with the patient's age.

During OK sessions, body sway was visually observed and the overall results on balance were quantified using the Equitest system at the beginning and at the end of the training program, but not after each rehabilitation session.

Equitest

Dynamic posturography (Equitest) equipment was composed of a moving platform with a visual surround; both could move exactly as the patient did, inducing visual and/or somato-sensory conflicts (Nashner 1976; Nashner et al. 1982). The sensory organization test assessed the patient's ability to make use of visual, vestibular, and somatosensory inputs separately and to suppress senses at times when they provided inaccurate information. The patient was exposed, in order of increasing difficulty, to six combinations of normal vision, eyes closed, and sway-referenced support and visual surround conditions. During testing, the patient wore a modified parachute harness attached to the ceiling to prevent injury from a fall. Test conditions 1 and 2 provided the patient normal (fixed) support surface inputs with eyes open and closed.

These two conditions provided performance baselines against which changes in performance during the other four altered sensory conditions can be compared. Test condition 3 fixed the support surface, while the patient stood eyes open within a sway-referenced visual environment. Differences in stability during conditions 1, 2, and 3 showed whether the patient could suppress the influence of an incorrect visual input. In test condition 4, vision was normal and the support surface sway-referenced. Test condition 5 had the patient standing on the sway-referenced surface, eyes closed, thereby eliminating all useful inputs but the vestibular. Test 6 exposed the patient to sway-referenced visual and support surface at the same time. During conditions 5 and 6 the subject was maintaining his balance using vestibular input only. The equilibrium score was calculated by comparing the angular difference between the patient's maximum anterior and posterior center of gravity displacements and a theoretical maximum displacement of 12.5°. This is based on the assumption that a normal individual can exhibit anterior and posterior sway over a total range of 12.5° without losing balance (Nashner et al. 1990). The result is expressed as a percentage between 0 and 100, with 0 indicating a fall and 100 perfect stability. The global equilibrium score (composite score) was calculated by independently averaging the scores for condition 1 and 2, adding these two scores to the equilibrium scores from each trial of sensory condition 3, 4, 5, and 6, and dividing the sum by 14, which is the total number of trials. Missing trials in conditions 3, 4, 5, and 6 were replaced by the averaged equilibrium score for that condition. The highest possible score was 100 (Nashner et al. 1982). The patient's performance on the six conditions were compared with responses of the control group.

Recordings: electrooculographic assessment

Horizontal eye movements were recorded using bitemporal d.c. electrooculography (EOG; Meditrace) and/or the I.R.I.S (Infra-Red light Eye-Movement Measurement) system. The d.c. signal of the horizontal component of eye movement was amplified, processed on-line through an analog-digital (AD) converter Data Translation card in a 286 microcomputer. The sampling rate was 50 Hz and the signal was not filtered. SPV, frequency, and amplitude of OKN were calculated using an interactive software. Vertical OKN was not recorded in the present work. Representation of data on the monitor were as follows: raw data of the eye movement, velocities of slow phases, and fast phases of nystagmus. For calculation of mean SPV a keyboard-driven cursor allowed more accuracy in the choice of nystagmus by rejecting inappropriate eye movements and blinks.

Patients

Two groups of patients were submittted to OK exposure sessions. These patients were tested at the Ear, Nose, and Throat (ENT) Department of Hôpital Lariboisière in Paris with the following methodology: audiometry, impedance-audiometry, caloric test, electrooculographic recordings of smooth pursuit and saccades, and eye-head movement recordings in order to test gaze stabilization in the horizontal plane (Freyss et al. 1988). These patients were also submitted to high velocity step stimulations (>400°/s) on a rotatory chair (Sémont and Sterkers 1980) to measure the postrotatory nystagmus. Cerebral imaging (computed tomography scan and magnetic resonance imaging) were carried out in order to rule out brain and/or hindbrain lesions.

The first group of five bilateral labyrinthine-defective patients (three women, two men) ranged in age from 38 to 58 years (mean 42 years). These patients had no responses on either side during the caloric test and did not present any postrotatory nystagmus. All these patients had been treated with aminoglycoside (gentamycine) for severe infections.

The second group of five unilateral labyrinthine-defective patients (two women, three men) ranged in age from 33 to 57 years (mean 44 years). These patients were seen 1–3 months after surgery for acoustic neuroma (three subjects) or after vestibular neurectomy (two subjects). They did not demonstrate any spontaneous nystagmus.

All the subjects were fully compensated. The patients were seen firstly in the ENT department of Hôpital Lariboisière for testing. Only patients fully compensated to classical ENT testing but still complaining of balance disorders went through this study. Control responses were obtained by recording from five age-matched normal healthy volunteers (two women, three men) ranging in age from 35 to 58 years (mean 45 years).

Results

Control subjects

For an OK stimulation session at 40°/s with the z-axis of the sphere tilted 45° backward, the per stimulation SPV of the OKN was regular (mean $\pm$ SD 28.7 ± 4.3°/s) and the frequency (number of nystagmus beats per second or number of fast phases per second $\pm$SD) of the OKN was 2.9 ± 0.5 Hz. The number of fast phases was used to quantify the frequency, assuming that each slow phase was followed by a fast phase. The beating field was normally shifted in order to anticipate the stimulus.

Minimal body sway was observed. Reported vection was appropriate for the direction of the stimulus. For normal subjects, the vection was appropriate for the given stimulus direction. Equitest results before and after OK exposure were normal and unchanged. The mean equilibrium scores $\pm$SD were: for composite, 78 ± 4; for condition 4, 82 ± 5; for condition 5, 69 ± 6 and for condition 6, 67 ± 10. Table 1 gives the normal Equitest data base for reference.

Patients

Bilateral areflexic patients

Before the rehabilitation program had started, the patients showed an important irregularity of the SPV of the OKN (Fig. 2A) with a mean of 32.1°/s ± 12.1 (the regularity of the OKN referred to the respective SDs of the OKN SPVs). The frequency of the OKN $\pm$SD was 1.9 ± 0.4 Hz (Fig. 3A).

Table 1 Normal Equitest data base equilibrium scores

Condition	Subject 20–59 years ($n=112$)	
	Means	$\pm$SD
1	94	2.3
2	92	4.2
3	91.5	3.3
4	82.5	7.6
5	69.2	10.5
6	67.2	11.6
Composite	79.8	5.6

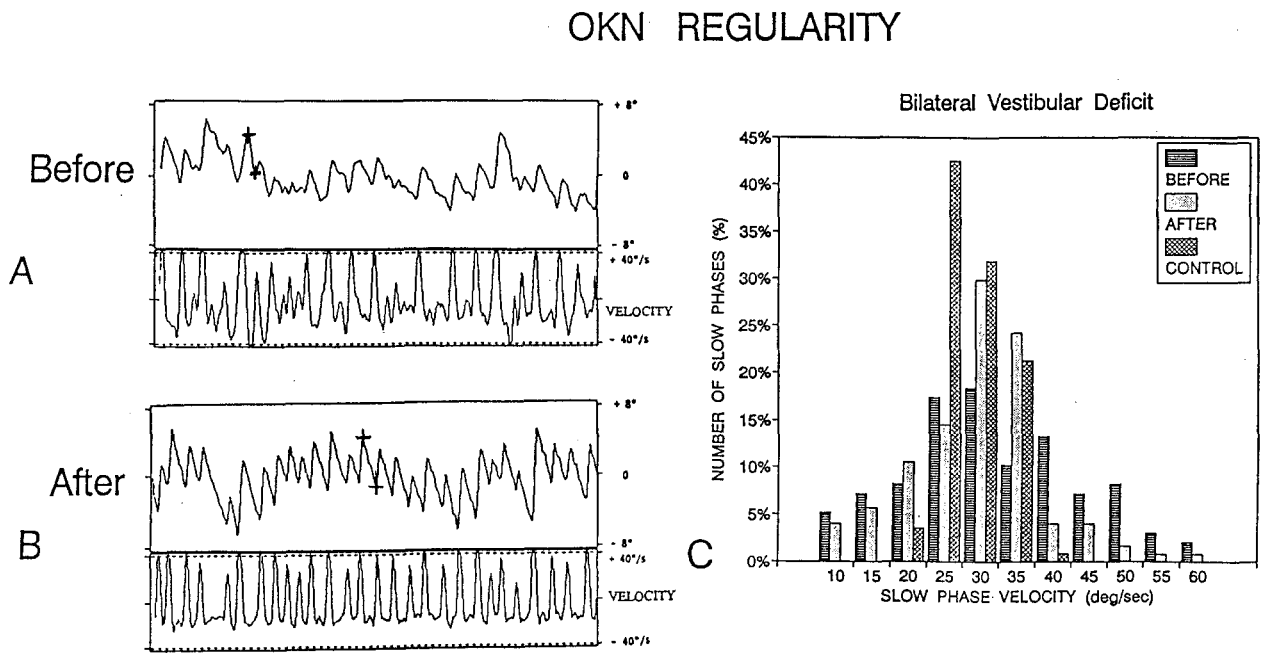

Fig. 2A–C Bilateral vestibular deficit: Optokinetic nystagmus (OKN) regularity. **A** Before training: recordings of OKN (*z*-axis of the sphere tilted 45° backward). *Top trace*, amplitudes; *bottom trace*, velocities. Total duration of the screen, 10 s. (The + sign indicates slow-phase limits for velocity computation.) **B** After training: recordings of OKN (*z*-axis of the sphere tilted 45° backward). *Top trace*, amplitudes; *bottom trace*, velocities. Total duration of the screen, 10 s. An improvement in the OKN regularity can be seen. (+ slow phase limits for velocity computation). **C** Histogram of normalized number of slow phases (with respect to total number of slow phases for each condition) for the 5 patients. (*Before* before training, *After* after training, *Control* control group)

Table 2 Equitest equilibrium scores for bilateral labyrinthine-defective patients. (*OK* optokinetic)

		Condition 4	Composite
Patient 1	Before OK exposure	60	46
	After OK exposure	76	50
Patient 2	Before OK exposure	65	45
	After OK exposure	85	51
Patient 3	Before OK exposure	41	40
	After OK exposure	63	53
Patient 4	Before OK exposure	52	43
	After OK exposure	74	55
Patient 5	Before OK exposure	59	47
	After OK exposure	79	58

Body sway occurred in accordance with the direction of the stimulus and would have led to falls if not prevented by the observer. Using pure vertical, upward OK stimulus, the whole group was unable to remain stable, the stimulus throwing the patient off balance backward with the OK beating field shifted in the direction of the stimulus. This instability occurred with all kinds of stimulation, but was more important when the stimulus induced backward body sway. Mean Equitest equilibrium scores $\pm$ SD were: for composite, 44 ± 3; for condition 4, 55 ± 10; and for conditions 5 and 6, 0 (Table 2). Patients fell during conditions 5 and 6 (those requiring vestibular information).

After repeated OK exposures, at the end of the training program, mean OKN SPV decreased (from 32.1 ± 12.1°/s to 29.6 ± 6.9°/s). The SD also decreased. An *F*-test on the variances, $F_{97,\ 123} = 1.82$ with $P = 0.0009$, demonstrated an improvement of the regularity of the OKN.

The frequency of OKN $\pm$ SD was 2.8 ± 0.5 Hz (Fig. 3B). The mean value of the OKN frequency was significantly improved ($P < 0.05$ with Student's *t*-test). The fast-phase frequency, after repeated OK exposure, became closer to that of the normal control subjects (2.9 ± 0.5 Hz) and more periodic, with the standard deviation of the fast phases becoming smaller, which can be considered an improvement.

Patients still fell during conditions 5 and 6, and mean equilibrium scores $\pm$ SD were: for composite, 53 ± 10; for condition 4, 75 ± 4, and for conditions 5 and 6, 0. Scores of test 4 and composite were significantly increased ($P = 0.01$) using the distribution-free *U*-test of Mann and Whitney (Table 2).

Unilateral vestibular patients

At the beginning of the training program, the patients did not exhibit spontaneous nystagmus but demonstrated an important asymmetry of the OKN (*z*-axis of the sphere tilted 45° backward) with an irregularity of SPV. The mean SPV $\pm$ SD was 22.7°/s $\pm$ 8.5 when OKN was beating ipsilaterally to the lesion (quick phase directed toward the lesion) and 32.3°/s $\pm$ 6.9 when beating contralaterally (Fig. 4). The OKN frequency was decreased especially when the OKN was beating ipsilaterally to

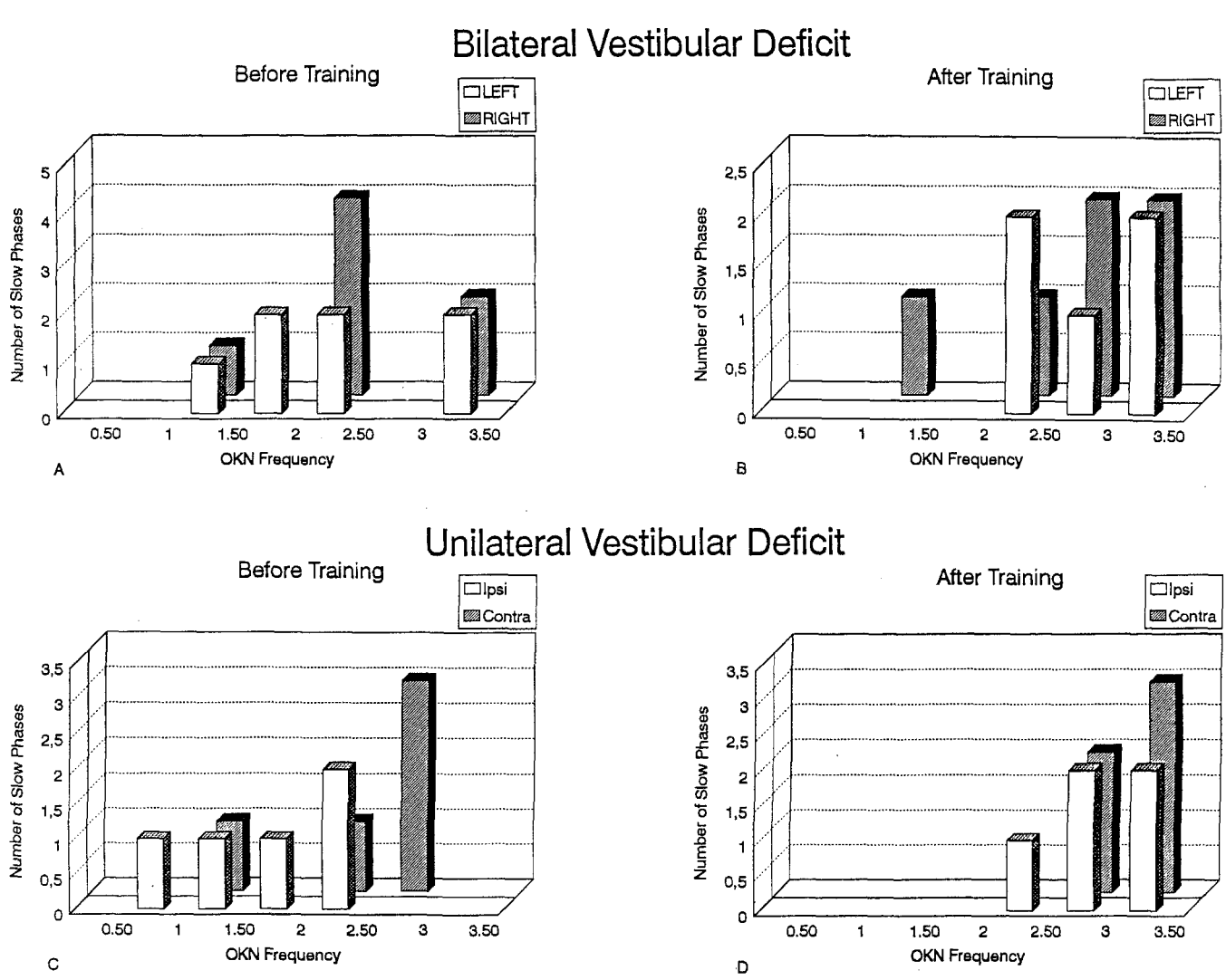

Fig. 3A–D Optokinetic nystagmus (OKN) frequency. **A** Bilateral vestibular deficit (5 patients). Before training: the OKN frequencies ranged from 1.3 to 3.2 Hz without differing between both sides. **B** Bilateral vestibular deficit (5 patients). After training: the OKN frequencies were shifted from 2.3 to 3.2 Hz with a mean of 2.8 ± 0.5 Hz (normal 2.9 ± 0.5 Hz). **C** Unilateral vestibular deficit (5 patients). Before training: there was an asymmetry between both sides and OKN frequencies were decreased when directed ipsilaterally to the lesion. **D** Unilateral vestibular deficit (5 patients). After training: the asymmetry of the OKN frequencies was significantly reduced

the lesion (Fig. 3C). The values of the OKN frequency were, respectively, 1.6 ± 0.8 Hz (ipsi) and 2.3 ± 0.7 Hz (contra).

The body sway was maximum when the direction of the OK stimulus was opposite to the side of the lesion. Mean Equitest scores $\pm$ SD were decreased compared with normal values, with 56 ± 10 for the composite, 79 ± 15 for condition 4, 11 ± 20 for condition 5, and 8 ± 10 for condition 6 (Table 3).

After training, the asymmetry of the OKN frequency (z-axis of the sphere tilted 45° backward) between the two directions was strongly reduced (Fig. 3D). The means of the SPV were 28.7 ± 7.4°/s (ipsi) and 26.7 ± 6.4°/s (contra). The averaged OKN SPV $\pm$ SD decreased (from 32.3 ± 6.9 to 26.7 ± 6.4°/s) when beating contralaterally and increased (from 22.7 ± 8.5 to 28.7 ± 7.4°/s) when beating ipsilaterally. There was a statistically significant improvement of the regularity asymmetry of the OKN, by referring to the respective SDs of the OKN SPVs between right and left, before and after training. Before training, F-test on the variances of OKN SPV was $F_{60,76} = 1.49$ with $P = 0.046$. After training, F-test on the variances of OKN SPV was $F_{95,97} = 1.36$ with $P = 0.067$. Since unilateral labyrinthine-defective patients even fully compensated to classical ENT tests, while complaining of balance disorders, still demonstrated this kind of asymmetry; the reduction in the regularity asymmetry seemed to be related to OK exposure.

The frequencies of the OKN were 2.9 ± 0.4 Hz when beating ipsilaterally to the lesion and 3.1 ± 0.5 Hz when beating contralaterally. A stastistically significant improvement of the frequency of the OKN was noticed for

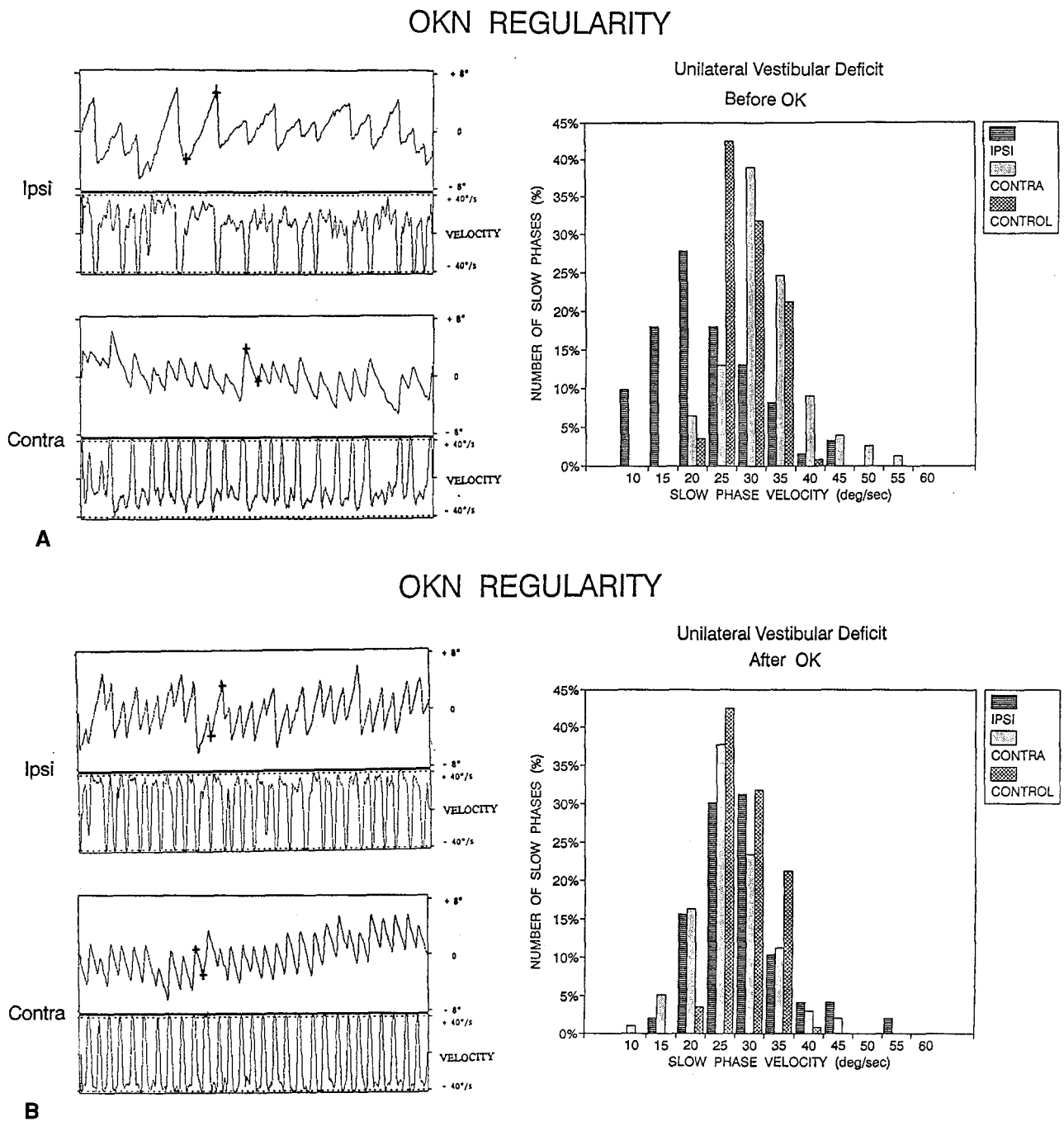

Fig. 4 **A** Left vestibular deficit: optokinetic nystagmus (OKN) regularity. Before training: recordings of OKN (*z*-axis of the sphere tilted 45° backward). *Top trace*, amplitudes, *bottom trace*, velocities. Total duration of the screen, 10 s. *Right*: histogram of normalized number of slow phases (with respect to total number of slow phases for each condition) for the 5 patients. There is a strong difference between both sides: OKN is very irregular when directed toward the lesion (*ipsi*). (The + sign indicates slow phase limits for velocity computation.) **B** Left vestibular deficit: OKN regularity. After training: recordings of OKN (*z*-axis of the sphere tilted 45° backward). *Top trace*, amplitudes, *bottom trace*, velocities. Total duration of the screen, 10 s. *Right*: histogram of normalized number of slow phases (with respect to total number of slow phases for each condition) for the 5 patients. The difference between the two sides is reduced and there is a significant improvement of the OKN regularity especially when directed toward the lesion; *F*-test on the variances, $P=0.046$ before and $P=0.067$ after training. (The + sign indicates slow phase limits for velocity computation)

the whole group and especially for the OKN beating ipsilaterally to the lesion ($P<0.05$ with Student's *t*-test). Also, for bilateral labyrinthine-defective patients, these scores became closer to that of the normal control subjects (2.9 ± 0.5 Hz) which can be considered as an improvement.

The mean scores of equilibrium of the Equitest $\pm$ SD were: for the composite, 78 ± 5; for condition 4, 88 ± 5; for condition 5, 60 ± 10 and for condition 6, 65 ± 5. These scores were significantly increased ($P=0.01$) using the distribution-free *U*-test of Mann and Whitney (Table 3).

Table 3 Equitest equilibrium scores for unilateral labyrinthine-defective patients. (*OK* optokinetic)

		Condition 4	Condition 5	Condition 6	Composite
Patient 1	Before OK exposure	72	26	0	55
	After OK exposure	86	68	76	82
Patient 2	Before OK exposure	90	0	39	55
	After OK exposure	95	41	64	74
Patient 3	Before OK exposure	75	0	0	52
	After OK exposure	84	80	51	76
Patient 4	Before OK exposure	85	0	0	58
	After OK exposure	90	42	65	72
Patient 5	Before OK exposure	75	29	0	59
	After OK exposure	85	70	76	85

For the two groups of patients

After training, patients did not sway anymore whatever might be the direction and velocity of the stimulus, but a large number of the patients still had the feeling that they did not know what was moving: the room or themselves. Equitest showed improvement in the scores of test 4 for the two groups of patients.

Discussion

The first striking result of this study is the remarkable improvement in OKN parameters after only a small number of sessions of exposure to OK stimulations. This finding seems to be consistent and indicates that complex optokinetic stimulation in a standing human subject induces some adaptive process which restores OKN symmetry, SPV and beating field normal value.

It has been suggested by Igarashi et al. (1975; in primates) and by Lacour (1981; in the cat) that activity is fundamental in the recovery of vestibular deficits and, more generally, it is known that adaptive mechanisms are more efficient when the subject is dealing actively with sensory conflicts and when sensory inputs are as natural and complex as possible. Although we have not explored the effects of OKN training on seated subjects, we would like to propose the hypothesis that the rapid improvement of both OKN parameters and postural control were due to the fact that our subjects were standing and therefore actively counteracting the effects of OKN on posture. In addition the complex visual pattern motion induced by the planetarium projected on the walls of the room may have required an additional impetus for active adaptation.

Unilateral vestibular patients

It is noted that OK stimulations induced body sway toward the direction of the stimulus. The population of unilateral vestibular-defective patients demonstrated body sway only when the stimulus was opposite to the lesioned side. The patient has a self-motion perception with body sway when OKN is opposite to vestibular spontaneous nystagmus. A possible interpretation of the decrease in postural sway which is observed in unilateral vestibular deficits after OKN training could be the following: before training the amplitude of OKN is greater when the visual scene moves toward the side of the lesion because the slow phase of latent spontaneous vestibular nystagmus is driving the eye in this direction and facilitates the OKN slow phase. Therefore retinal slip is small and consequently the visually induced postural effects are smaller in this direction. This observation is similar to what can be seen in central-diseased patients with spontaneous downbeat nystagmus. When the OK stimulus provokes an upbeat nystagmus the patient strongly sways forward. This has to be related to the visual stimulation of the pavement shifting under the feet of somebody walking straight ahead and may partly explain the insecurity encountered by the elderly when they need to walk. By contrast, when the visual scene moves toward the intact side three factors contribute to a large postural sway in this direction: (a) The slow phase of OKN is small and very irregular and therefore retinal slip is increased. (b) This increase of retinal slip induces a larger body sway by the visual projections to the neck and postural muscles. (c) The beating field of OKN seems shifted toward the side of the visual field motion. The well known oculo-nucal coupling may also induce an eye movement-related postural effect toward the side of the moving visual scene.

The effect of training can then be seen as inducing the following changes: (a) Probably a general increase in OKN amplitude and a recovery of symmetry between the two sides whose effect is to reduce retinal slip. Also a decrease in the OKN irregularities has been observed. (b) The consequence of this decreased retinal slip is that there is less visually induced postural sway. (c) In addition it could be suggested that the recovery of a well-balanced OKN allows also a recovery of a normal beating field which tends to drive the eye in the direction opposite to the movement of the visual scene, therefore inducing a counter-effect to the postural sway.

The improvement in Equitest condition 5, when eyes were closed, suggests perhaps a central restoration of symmetry that is not visually dependent. The patients were fully compensated at the classic ENT investigations, which means that the sane ear had already com-

pensated for the diseased one. The restoration in symmetry may be initiated by a central adaptive process.

Bilateral vestibular patients

We have previously demonstrated the role of OK stimulation in rehabilitation of elderly patients with presbyastasia (Sémont et al. 1992). These patients with normal vestibular function behaved like bilateral vestibular deficient patients (free falls in conditions 5 and 6 of the Equitest). After training, the scores of these patients on tests 5 and 6 were normalized, demonstrating the back-to-normal equilibrium sensory organization with full use of the vestibular input.

On the other hand, bilateral labyrinthine-defective patients demonstrated, after training, an increase in the score of condition 4 of the Equitest. This could be due to a more appropriate use of somatosensory cues helped by the visual input. This is correlated with the improvement of the regularity of the OKN associated with a normalization of OKN frequency.

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