Review

Vestibular compensation following vestibular neurotomy

A. Devève a,b,*1, M. Montava a,b,1, C. Lopez*, M. Lacourc, J. Magnanc,d, L. Borelc

a Service d'oto-rhino-laryngologie et d'otoneurologie, hôpital Nord, CHU, Assistance publique–Hôpitaux de Marseille, chemin des Bourrelly, 13915 Marseille cedex 20, France
b Aix Marseille University. Laboratoire de Biomécanique Appliquée, IFSTTAR UMR T24, Boulevard Pierre Dramard, 13015 Marseille, France
c Aix Marseille University, Laboratoire de neurobiologie intégrative et adaptative, CNRS UMR 6149, 3, place Victor-Hugo, 13331 Marseille cedex 03, France

A R T I C L E   I N F O

Keywords:
Vestibular compensation
Unilateral vestibular neurotomy
Menière's disease
Postural recovery
Visual dependency

A B S T R A C T

Objectives: Four studies assessing vestibular compensation in Menière’s disease patients undergoing unilateral vestibular neurotomy, using different analysis methods, are reviewed, with a focus on the different strategies used by patients according to their preoperative sensory preference.

Material and methods: Four prospective studies performed in a university tertiary referral center were reviewed, measuring the pattern of vestibular compensation in Menière’s disease patients before and after unilateral vestibular neurotomy on various assessment protocols: postural syndrome assessed on static posturography and gait analysis; perceptual syndrome assessed on subjective visual vertical perception; and oculomotor syndrome assessed on oculocyclotorsion.

Results: Vestibular compensation occurred at variable intervals depending on the parameter investigated. Open-eye postural control and gait/walking returned to normal one month after neurotomy. Fine balance analysis found that visual perception of the vertical and oculocyclotorsion impairment persisted at long-term follow-up. Clinical postural disturbance persisted only when visual afferents were cut off (eyes closed). These impairments were the expression of a postoperative change in postural strategy related to the new use of visual and non-visual references.

Conclusions: Understanding pre-operative interindividual variation in balance strategy is critical to screening for postural instability and tailoring vestibular rehabilitation.

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1. Introduction

The neurologic phenomenon of vestibular compensation underlies the surgical treatment of Menière’s disease (MD) by unilateral vestibular neurotomy (UVN) [1]. Selective vestibular neurotomy causes sudden, total and enduring deafferentation of the vestibular apparatus while sparing the cochlear pathways [2]. Vestibular compensation is based on central nervous system reorganization, leading to functional rehabilitation. This functional recovery after destruction of the labyrinth has long been known as an empirical fact [1], since Flourens introduced the topic in 1824 in animal experiments demonstrating functional recovery one week after labyrinthectomy in birds and frogs. Recent scientific work on neural recovery after labyrinthectomy or unilateral vestibular neurotomy has made a decisive contribution to the foundations of surgical management of peripheral vertigo in the light of vestibular compensation [1]. Vestibular neurotomy thus provides an ideal clinical model for the study of individual vestibular function compensation strategies. This surgery removes the dysfunctional afferents of the pathological ear so as to prevent rotational vertigo due to acute conflict between visual, proprioceptive and vestibular balance-related afferents. Optimal compensation is promoted by sensorimotor rehabilitation specific to the individual patient [3].

The vestibule or posterior labyrinth is a mechanoreceptor essential to balance, with multimodal (visual and somesthetic) sensory afferents converging as of the vestibular nuclei and throughout the central vestibular pathways, with information from all of the various peripheral receptors being coordinated at brain level. Deafferentation of one vestibular nucleus induces asymmetry in the vestibular system, with unilateral static and dynamic vestibular deficit causing postural, perceptual and oculomotor syndromes. The postural syndrome involves inclination of the head and body toward the affected side, with locomotor disorder. The perceptual syndrome comprises vertigo with deviation of the perceived visual vertical and spatial disorientation. The oculomotor syndrome...
comprises spontaneous nystagmus, vertical strabismus and ocular cyclotorsion. After a few weeks to months, the deafferented vestibular nuclei show spontaneous activity comparable to the contralateral side, thanks to cerebral plasticity, contributing to recovery of balance. Vestibular neurotomy totally and definitively abolishes the vertigo attacks suffered by patients with disabling MD. To avoid a poor result, vestibular nerve sectioning must be complete, on whatever approach: supratemporal, [4], retro-labyrinthine [5] or retrosigmoid [6]. Although neurotomy abolishes vertigo in 90% of cases [6,7], 10% of patients show residual instability despite unilateral vestibular areflexia [8,9], due to poor vestibular compensation.

The present article reviewed studies of the mechanisms of vestibular compensation using various methods of analysis in MD patients who had undergone UVN.

2. Methods

A review of our team’s recent studies in MD patients who had undergone UVN sought to determine whether unstable patients displayed specific characteristics during post-deafferentation vestibular compensation (Table 1). Vestibular compensation was analyzed in distinct studies: postural syndrome on static posturography (n = 50 patients) [10], walking performance on kinematic analysis (n = 9) [11], perceptual syndrome via visual perception of the vertical (n = 40) [12], and oculomotor syndrome by analysis of ocular cyclotorsion and the torsional optokinetic reflex (n = 17) [13].

2.1. Static posturography

This study [10] analyzed postural control in a homogeneous population of 50 MD patients who had undergone UVN, determining the contribution of the visual system before and after induced vestibular deficit. Pre-UVN static posturography was performed on a force platform, with the patient’s eyes open and then closed. Postural stabilization was assessed by calculating body oscillation area with and without visual input and the differential postural performance index between the two conditions [area EC – area EO]/area EC+ area EO) (Figs. 1 and 2), and the study was repeated at one week, two weeks, one month, three months and one year post-UVN.

2.2. Walking performance study

The walking performance study [11] quantified locomotor syndrome in 9 MD patients before and after (one week, one and three months) UVN and walking performance in 10 control subjects. Kinematic gait analysis used an ELITE video movement analyzer with reflective receptors, with subjects’ eyes open versus closed. Trajectories were analyzed during normal walking (Figs. 3–5).

Locomotor data (mean speed, step rate, step length) were assessed for normal and fast walking (Figs. 3–5). For each subject and study phase, trajectory deviation and study parameters were recorded for three consecutive 3-meter walks at normal then fast speed under the two conditions (eyes open and closed).

2.3. Subjective visual vertical

Visual perception of the vertical, or subjective visual vertical [12], was studied to shed light on perceptual impairment. Perception of the vertical is fundamental to integrating the direction of the gravitational vector and thereby organizing the maintenance of posture. The study was conducted on 40 MD patients, before and one week, one month and one year after UVN. Perception of the static visual vertical (SVV) was investigated using an immobile visual field (Fig. 6: field of scattered dots), and that of the dynamic visual vertical (DVV) using circular optokinetic stimuli by rotating the field of dots around the visual axis at a rate of 5°–120°/s (Fig. 6), ipsi- and contra-laterally to the lesion.

2.4. Ocular cyclotorsion

The ocular cyclotorsion study [13] analyzed oculomotor impairment on videonystagmography. The objective was to analyze static ocular cyclotorsion amplitude and evoked torsional optokinetic nystagmus (tOKN) speed in 17 MD patients before and one week, one month, three months and one year after UVN. Ocular cyclotorsion amplitude following unilateral vestibular defect was measured facing an immobile visual environment (Fig. 7). The dynamic study was performed during optokinetic stimulation by a disk with black marks on a white ground turning around the visual axis on the affected or on the contralateral side at angular speeds ranging from 5°/s to 120°/s. Fig. 8 shows the tOKN obtained at 80°/s.

3. Results

3.1. Static posturography

Pre-operatively, patients could be divided into two groups according to their reaction to eye closure: 54% displayed increased and 46% unchanged body oscillation (Fig. 1): i.e., “visual-dependent” subjects having a visual postural strategy, and “visual-independent” subjects with a non-visual (proprioception-based) strategy. The former were characterized by a differential postural performance index greater than 20%. There was interindividual variation in the degree of reliance on visual input for fine postural regulation. Strategy was then reassessed for all patients following UVN. All showed change in strategy (Fig. 1): pre-operatively visual-dependent patients adopted a non-visual
strategy following unilateral vestibular loss; conversely, visual-independent patients adopted a visually based postoperative strategy (Fig. 2). These changes were observed as early as one week post-UVN. Strategy (visual versus non-visual) did not correlate with age, gender or clinical signs of MD [10].

3.2. Walking performance

Pre-operatively, MD patients showed no deviation of trajectory, but reduced mean speed, step length and step frequency, eyes open and closed, compared to controls (Fig. 3). Postoperative changes on walking performance parameters were seen on both normal and fast walking (Fig. 3). The pre-operative locomotor pattern disturbance was significantly reduced on both normal and fast walking, eyes open and closed (Fig. 3). Likewise, in the eyes-closed condition, the locomotor pattern normalized by one month for normal walking but only at three months for rapid walking (Fig. 3) [11]. The acute post-UVN phase showed trajectory deviation, which was ipsilateral to the lesion with eyes closed and contralateral eyes open (Figs. 4 and 5); deviation showed rapid compensation in the eyes-open condition, but persisted at three months postoperatively with the eyes closed (Fig. 5).

Fig. 1. Illustration of visual and nonvisual strategies. A. Before vestibular nerve section. Histogram distributions plotting the number of subjects (ordinates) in the 10% class intervals (abscissae) representing the percentage difference of sway (différence posturale) with eyes closed compared with eyes open. Note that the populations are split (see arrows) into significantly distinct visual (filled histograms) and nonvisual (open histograms) subgroups. Mean percentage differences of sway (±SD) calculated in each subgroup are reported. B. One week after vestibular nerve section. The change of strategy from nonvisual to visual and vice-versa is seen by the displacement and overlapping of open and filled histograms. Modified after [10].

Fig. 2. Raw stabilograms recorded for two controls displaying either a nonvisual (left) or a visual (right) postural strategy. The center-of-force positions in the anteroposterior (Y position) and left-right (X position) directions are plotted with their 90% confidence ellipse. Body sway was recorded with eyes open (yeux ouverts) and eyes closed (Yeux fermés). The percentage difference of sway (PDS score) calculated on sway area (surface) is reported in the graphs for each subject. Modified after [10].
3.3. Subjective visual vertical

In the acute phase of unilateral vestibular loss, SVV showed inclination toward the affected side, becoming normal at around one year post-UVN. DVV showed inclination toward the affected side in the acute phase, whatever the optokinetic stimulation; with stimulation ipsilateral to the lesion, compensation was achieved by one month, but remained incomplete at one year for contralateral stimulation at whatever speed [12] (Fig. 6).

3.4. Ocular cyclotorsion

Post-UVN, static ocular cyclotorsion was oriented toward the lesional side as compared to pre-operative findings (Fig. 7). It peaked in the acute phase, at 9° at one week, then gradually diminished although still persisting at three months post-UVN. In the acute phase, tOKN was highly asymmetric and dependent on stimulation direction (Fig. 8): slow-phase tOKN speed increased under stimulation ipsilateral to the lesion and decreased under
contralateral stimulation; this asymmetry was still not compensated for at three months post-UVN [13].

4. Discussion

4.1. Study limitations

All patients underwent unilateral vestibular neuroneurothomy through a retrosigmoid approach under videoneystagmographic control comprising pendular and caloric tests demonstrating absence of vestibular reflex. Although these tests analyze only low frequencies, the populations as a whole were considered homogeneous, all being free of residual symptomatic instability or oscillopsia.

4.2. Acute vestibular deafferentation induces change in balance strategy via cerebral plasticity

The various studies described here showed varying degrees of compensation, with distinct strategies depending on whether the patient had been visual-dependent or proprioception-dependent before UVN. The postural study distinguished two populations pre-operatively (Fig. 1): 54% showed increased body oscillation on eye closure and could be considered visual-dependent, with a visual balance strategy; 46% showed no change and could be considered visual-independent, with a non-visual, probably proprioceptive, strategy. There is thus an interindividual difference in the importance of visual input for fine postural regulation.

Following unilateral vestibular loss, postural strategy is seen to change (Fig. 1): visual-dependent subjects adopt a non-visual and visual-independent subjects a visual strategy. This represents a central compensation mechanism, suggesting rapid adaptation and a change in the reference framework used in postural control. The mechanism can be seen as early as one week post-UVN.

The sensory postural strategy selected after UVN may correspond to a mainly visual or mainly proprioceptive sensory substitution [14]. The present hypothesis is that the original centrally governed selection becomes maladapted after UVN, when vestibular input becomes suddenly, totally and definitively asymmetric. The vestibular-spinal deficit caused by surgery is interpreted centrally as an error message, cancelling the pre-operative strategy in favor of a new stabilization strategy. This change in postural strategy and of reference frameworks by a change in sensory selection may be a means of reducing anticipated postural instability.

4.3. Can the change in balance strategy guide vestibular rehabilitation?

This interindividual difference in the role of visual versus proprioceptive input in fine postural regulation and the associated change in postural strategy suggest that customized vestibular rehabilitation would be more appropriate, taking account of the loss of sensitivity to visual or to proprioceptive stimuli according to the individual’s preferred pre-operative strategy. Pre-operative static posturography can identify the patient’s strategy and thus predict post-deafferentation evolution, thereby potentially being a key examination for the design of postoperative rehabilitation so as to optimize vestibular compensation [15]. A further question is whether patients should be rehauled according to their previous or to their new strategy. The change in postural strategy and of sensory selection is a means of compensating postural instability, so that vestibular rehabilitation founded on the new strategy will probably be more effective; only a prospective comparative study, however, could answer these questions.

4.4. How should persistent walking performance disturbance be interpreted?

Immediate postoperative trajectory deviation toward the side ipsilateral to the lesion with eyes closed is due to unilateral vestibular information loss and toward the contralateral side with eyes open probably due to overcompensation by visual information.
(Figs. 3 and 4). It is rapidly compensated for with the eyes open, but persists for 3 months postoperatively with the eyes closed (Fig. 3). Likewise, while normal-speed walking is no longer disturbed by one month post-UVN, disturbance persists for rapid walking with the eyes closed (Fig. 5). None of the patients, however, reported any sensation of clinical instability. The vestibular system plays a major role in orienting and stabilizing the body, and the visual system is essential for the achievement of good compensation and to offset the loss of vestibular information [16]. The visual system plays an enhanced role in locomotor function when the vestibular system has undergone unilateral loss, which explains why disturbance should be revealed in the eyes-closed condition: the lack of visual input causes the fine disturbances of walking that can be observed, even late after unilateral vestibular loss, and these persist over time although without any clinical impact.

4.5. How should persistent disturbance of visual vertical perception and ocular cyclorsion be interpreted?

Fine analysis reveals major disturbance of the subjective visual vertical (Fig. 6) and of cyclorsion (Figs. 7 and 8) as of the immediate postoperative phase. Although diminished, these disturbances persist late after neurotomy, despite the lack of any reported sensation of instability. These functions remain impaired long after UVN; compensation is long and gradual: DVV, for example, remains disturbed at one year (Fig. 6). The vestibular system is involved in stabilizing gaze under optokinetic stimulation. Analysis of perceptual impairment demonstrates the role of the vestibular system, in interaction with other sensory modalities, in constructing internal spatial representation, which is transiently disorganized, at a high level of processing, by unilateral abolition of vestibular information [17]; compensation again is long, but this does not mean that vestibular rehabilitation should be intensified if there is no clinical disturbance.

4.6. Do these disturbances explain persistent instability?

In reality, none of the patients reported instability, despite the fine disturbances revealed on the study parameters. Psychological factors such as stress have been shown to underlie persistent instability [18]. Subjectively, 90% of patients report no instability; the other 10% may be subject to an inappropriate rehabilitation strategy. Incomplete compensation in this population may also be caused by poor integration or central adaptation due to coexisting lesions or a fragile psychological context.

A dedicated comparative study of unstable patients will be needed to specify the causes of persistent instability.

5. Conclusion

Vestibular compensation after neurotomy usually proceeds under good conditions and with good results. Residual instability is rare, but requires attending to. Understanding persistent instability and individual variation is essential for patients and to shed light on the phenomenon of vestibular compensation. Fine analysis of compensation strategies discloses the individual degree of visual dependence, which is detectable pre-operatively and can guide rehabilitation. It could find a strategy of customized vestibular rehabilitation as of the pre-operative phase.

Disclosure of interest

The authors declare that they have no conflicts of interest concerning this article.

References


