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Review

Vestibular compensation following vestibular neurotomy

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ABSTRACT

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 ocular cyclotorsion.
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 ocular cyclotorsion 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.

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

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Table 1
Summary of the 4 studies reported.

Study	Patients (*n*)	Type of analysis	FU	Reference
Postural	50	Static posturography	D – 1, D + 7, D + 15, D + 30, D + 90, D > 365	Lacour, et al. [10]
Walk	9	Kinematic gait analysis	D – 1, D + 7, D + 30, D + 90	Borel, et al. [11]
Subjective visual vertical	40	Immobile (static condition) and rotational visual field (dynamic condition: ipsi- and contra-lateral optokinetic stimulation)	D – 1, D + 7, D + 15, D + 30, D + 75, D + 135, D > 365	Lopez, et al. [12]
Ocular cyclotorsion	17	Videonystagmography, in static and dynamic condition (ipsi- and contra-lateral optokinetic stimulation)	D – 1, D + 7, D + 30, D + 90	Lopez, et al. [13]

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], retrolabyrinthine [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