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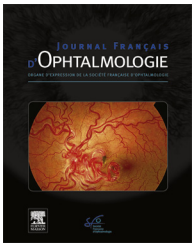

ORIGINAL ARTICLE

Vestibular asthenopia

L'asthénopie vestibulaire

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KEYWORDS
Vestibular asthenopia;
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Dizziness

Summary Vestibular asthenopia, analogous to visual asthenopia, is a sensory (or sensory-motor) discomfort consisting of a set of subjective symptoms, the expression of which is essentially visual and whose origin is a transient vestibular incident. It can be considered the result of a sudden global central disorder, such as a ''computer glitch,'' following a chain of events in response to an initial vestibular disease, even minor and devoid of clinical signs. This disorder results in inadequate processing and imperfect integration of afferent visual and vestibular input, leading to ocular fatigue, pain associated with eye movement, and sensitivity to retinal slip.

MOTS CLÉS
Asthénopie vestibulaire ;
Deficit vestibulaire ;
Rétine périphérique ;
Rehabilitation vestibulo-visuelle ;
Dizziness

Résumé L'asthénopie vestibulaire est, par analogie à l'asthénopie visuelle, un inconfort sensoriel (ou sensori-moteur) comprenant un ensemble de symptômes subjectifs, dont l'expression est essentiellement visuelle et dont l'origine est un incident vestibulaire transitoire. Elle peut être considérée comme étant le résultat d'un désordre central global survenu soudainement, tel un « *bug* informatique », suite à une succession de mécanismes en chaîne, en réponse à une pathologie vestibulaire initiale, même mineure et dénuée de signes cliniques. De ce désordre découle un traitement inadéquat et une intégration imparfaite des afférences visuelles et

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vestibulaires, conduisant à une fatigue des yeux, à une pénibilité associée à leurs mouvements, ainsi qu'à une sensibilité au glissement rétinien.

Introduction

The chain of gaze stabilization is ensured, in dynamic vision, by different reflexes, the most important of which is the vestibulo-ocular reflex (VOR). This one aims at stabilizing an image of interest on the retina in order to guarantee foveal vision while the individual is moving. It is also the vestibular sensor that allows, during a ballistic movement of the head and eyes, to encode and integrate the speed and amplitude of the head so that the ensuing saccade is executed with precision. Beside the VOR the cervico-ocular (COR) reflex also intervenes, but its role remains minor, as evidenced by its extremely low gain.

The vestibule is therefore inseparable from the oculomotor system and it forms, with it, a couple in perpetual activity. Even in the state of relative rest and immobility, it ensures, as a tonigenic element of the optostatic function, the maintenance of the primary position of the eyes relative to the gravitational vector. In addition important information is provided by a complex of proprioceptive sensors.

This small silent sensor at the origin of our sixth sense (the non-conscious sense of balance), is therefore of paramount importance: its close relationship which intimately links it with the visual system gives it a status as a functional organ of first importance.

Integration of the visual, vestibular and postural input is realized in the cortex.

A subject devoid of vestibular sensors has great difficulties of equilibration but also a significant decrease of the eye-head coordination. Such a condition represents a huge disability whose impact on everyday life is not sufficiently recognized (oscillopsia and falls, with serious consequences on both personal and societal level).

Most people have experienced vertigo at least once in their lives. This symptom is, all in all, rather banal; it is a frequent reason for consultation and is regularly mentioned by the patient when the doctor questions him. Very often, when this symptom [1] does not exceed a certain threshold of tolerance, it disappears spontaneously, usually after a little rest or restorative night.

However, because of inter-individual variability of the tolerance threshold, an ordinary dizziness can have different consequences for different individuals. Very anxiety-provoking, the dizziness can quickly trigger stress, even panic, and sometimes significant neurovegetative reactions, even if the vestibular disturbance itself is minor. This negative experience will then become part of the patient's memory and become the starting point for a vicious circle of anxiety in which the apprehension of movement will be one of the main characteristics. Aside from this psycho-emotional consequence, there is the risk of developing other sensory complaint which patients find very hard to describe, often diagnosed as ''visual hyperdependency''.

Patient's complaints are very similar to complaints of visual asthenopia such as visual fatigue, blurred vision, frontal headache, discomfort to eye movements, discomfort in front of TV and computer screens, discomfort at reading, lower visual concentration and so forth.

Other symptoms are more specific to vestibular impairment: discomfort with respect to peripheral visual scrolling or in relation to an unstable visual scene, discomfort when stabilizing spacio-environmental references are absent (in dim light, in large spaces or crowds) or discomfort in the absence of a fixed visual anchor (during exploratory jerks or pursuit).

''Vestibular asthenopia''

It is customary for oto-neurologists to consider the symptoms mentioned above as evidence of ''visual hyper dependence''. This notion has been advanced by Reason and Brand [2] in their ''sensory rearrangement theory'' and resulted from better understanding of balance strategies. Thanks to the contribution of dynamic posturography (Equitest®) [3], it has been possible to isolate the three main sensory detectors intervening in the function of equilibration: vision, vestibule and proprioception. In their view, for maintaining their balance, these patients are overdependent on visual input.

In recent years several authors have challenged this vision and have introduced a more nuanced concept: ''Visual Vertigo'' (Bronstein) [4] or ''Visual Vestibular Mismatch'' (Mallinson) [5]. Bisdorff et al. hold the terms of ''Visually induced vertigo'' or ''Visually induced dizziness'' [1].

Partnership ''peripheral retina — vestibule''

This notion of ''visual hyperdependence'' may unfortunately lead to confusion as to the origin of the symptoms it is supposed to encompass. Its explanation seems to be based solely on a purely integrative dysfunction, playing out at the level of a rivalry between regions of the cortex. It is that known that the peripheral retina is the complementary ally of the vestibule.

Unfortunately, the concept of ''vision'' on which the whole theory of equilibration of the symptomatic subject is based is presented as that of a cyclops, without nuance and without reference to binocularity. We have emphasized

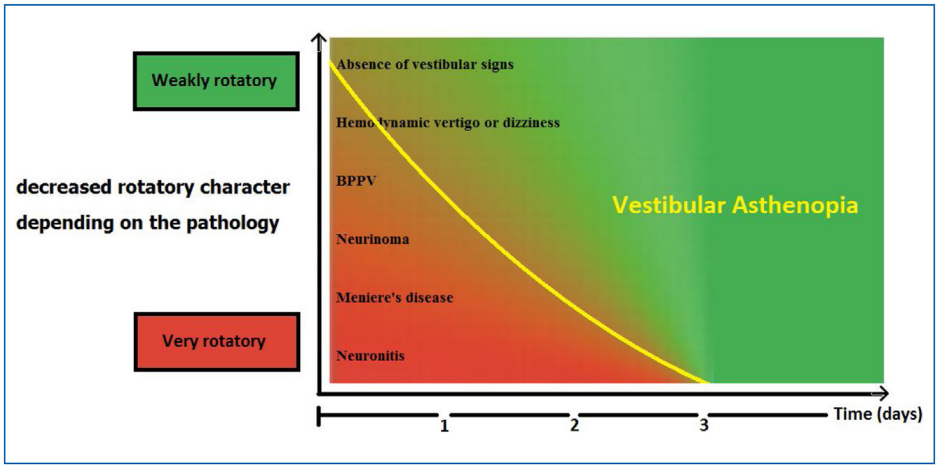

Figure 1. Evolution of the patient's subjective complaints over time.

in the introduction the crucial role of the very intimate link between vestibule and oculomotricity. The two vestibules and two eyes are linked almost mechanically as are the steering wheel of a car and its front wheels. Recall that these two vestibules are intended to transmit, via the vestibular and oculomotor nuclei, a speed signal and a position signal. If there is indeed an integrative dysfunction consecutive to initial failure of the vestibular sensor, it must be borne in mind that the binocularity resulting from an extremely complex and delicate balance, may also have been upset by the oculomotor disorder. It is therefore also at this level that part of the complaints can originate. Integrative disorder is inseparable from the binocular sensorimotor state [6]. Kapoula [7] has shown alterations in binocular dynamics in all subjects who had suffered unilateral vestibular disease. This study demonstrated the existence, in these subjects, of a drop in the quality of saccades and vergences, in terms of temporality and precision, even in the absence of head movement.

These considerations led us to coin this complex of symptoms ''vestibular asthenopia'' (Bauwens) [8].

The scale of sensory symptoms of vertiginous patient

It is important to dissect, by taking a thorough history, the chain of causal influences that can explain complex subjective symptoms.

This history must be so accurate that it highlights all possible indicators that may suggest any underlying cause of vestibular asthenopia.

This elaborate history must be followed by a range of direct and appropriate explorations to determine the clinical features of a vestibular lesion, whether peripheral or central.

The most important clinical test is the measurement of the slow phase velocity of the pathological nystagmus. It has long been accepted that vestibular involvement becomes responsible for rotational vertigo from the moment that the slow phase velocity of the pathological nystagmus is higher than or equal to 4 degrees per second. At this velocity, a retinal slip occurs that no longer allows fixation, thus, the altered vestibulo-ocular reflex (VOR) cannot be inhibited by fixation any longer.

However, below this value, the patient will no longer describe rotation of the visual scene, but the complaints will always have a visual connotation (as decribed above, Introduction) and may possibly even include a misperception of movement (oscillopsia).

This explains how patients who initially present with important rotatory complaints, may evolve towards vestibular asthenopia without rotatory components if partial spontaneous recovery occurs (Fig. 1).

Rotational vertigo is an extremely distressing complaint when it lasts longer than a minute because it will then be accompanied almost systematically by neurovegetative reactions (nausea, vomiting).

The vestibular asthenopia that may follow at a later stage is much less distressing but remains very uncomfortable because of its permanent nature and its impact on virtually all activities of daily living. A scale of evaluation of these subjective symptoms has been established recently [9].

Physiopathogenesis

When a breakdown occurs of the vestibular sensory system, certain neurophysiological mechanisms will react immediately to limit as much as possible the negative consequences linked to the loss of vestibular function, both visually and on the level of postural balance.

A complete unilateral vestibular lesion has static and dynamic oculomotor consequences: it gives rise to a static oculomotor syndrome including spontaneous nystagmus, vertical deviation (skew deviation) and conjugate cyclotorsion of both eyes. The slow phase of the nystagmus is directed towards the injured side and its direction is horizontal. The vertical deviation is expressed by a low position of the eye at the injured side, the conjugate cyclotorsion is also directed towards the side of the lesion (eg right sided lesion: RE excyclo. LE incyclo) The subjective visual vertical

will also be affected, which can be considered to be the perceptive reflection of the conjugate cyclotorsion. On a dynamic level, unilateral vestibular involvement affects the horizontal vestibulo-ocular reflex, whose gain drops sharply when the head is rotated on the injured side, resulting in erroneous eye-head coordination.

During the days following the onset of the vestibular attack, gaze stabilisation being disrupted, the processing of the visual information will be perturbed and this generates as a consequence a hypersensitivity to the optical flow that hampers central fixation. Other consequences are: rupture of fine binocular vision, fusional disorders and, sometimes, accommodational disorders.

The first mechanism to intervene at the onset of such a unilateral deficit is a process of inhibition in which the vestibulocerebellar loop plays an important role. It aims to extinguish as quickly as possible the very uncomfortable spontaneous nystagmus caused by the vestibular lesion. From this moment, a second mechanism, slower, is also set up: this is a mechanism of central compensation, whose purpose is to rebalance the relative importance of the two vestibular nuclei via the commissural connections.

However, overdependence on visual information in the absence of efficient vestibular signals results in poor or incorrect processing of the various relevant signals in the cortical region.

The peripheral retina plays an important role in the processes that compensate. However, the normal function of the peripheral retina is to detect movement in the peripheral visual field. The ''incorrect'' use of the peripheral retina as a balancing crutch leads to integrative disorders.

Once the spontaneous nystagmus has disappeared and the directional preponderance is compensated for centrally, the vestibular balance tends to return to its original state, acting as an accelerometer, except at high velocities where some indelible, often symptomless traces of the affection will persevere. Specific tests (e.g., the head impulse test) [10] will show a persistent deficit of gain of the VOR.

However, beyond the signs of good vestibular recovery, other symptoms often persist. The causes of these sensory sequels must be sought mainly at the level of the binocular equilibrium and at the level of processing the various relevant signals in the cortical region. Very often, a pre-existing unstable or fragile binocular condition decompensates because of a vestibular disorder which gives rise to visual disturbances. It follows that binocular vision (phorias, suppression, fusional amplitudes, convergence, refractive errors) must be examined thoroughly, and remedies where possible.

Overdependence on visual information in the absence of efficient vestibular signals results in this poor or incorrect processing of the various relevant signals in the cortical region.

The reverse switch - once the vestibular function has been restored—occurs less naturally. Rehabilitation consists of re-harmonizing the use of vestibular and visual sensors and ensuring that the brain modulates the relevant sensory input in the most optimal way possible, depending on the physical context (physical environment, posture, visual surroundings,...).

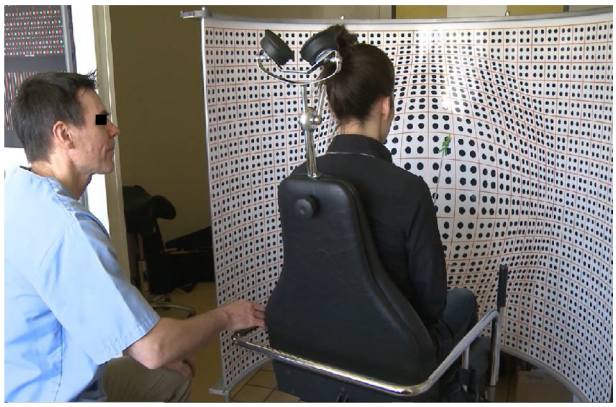

Figure 2. Exercise of the ''baguette chair'' in the presence of the PSP; attaching a staff during a chair movement.

Treatment vestibulo-visual rehabilitation

The aim of the treatment of a vestibular condition is to relieve patient symptoms.

Depending on the start of treatment in comparison to the onset of the affection, the intensity of the symptoms can be very variable, depending on the stage of the disease. The initial treatment will therefore vary in function of the moment that patients are referred.

The neurovegetative reactions, generally very distressing to live with, appear only in acute phase. They respond well to appropriate sedative rehabilitation, which will be explained below.

Rotational vertigo, characterized by the presence of a spontaneous nystagmus, can be treated by the reeducative techniques using the rapid swivel chair, developed by A. Sémont and J-M. Sterkers [11]; they will accelerate the neurophysiological processes of central compensation.

In the case of vestibular asthenopia, after performing an assessment of the binocular sensorimotor functions, the role of the orthoptist will consist in stabilizing central fixation and maintaining this fixation during head movements (vestibulo-visual re-education [12].

The task of fixation, under dynamic conditions, on a low-speed swivel chair restores and sharpens mechanisms involved in the highest degrees of visual perception [8]. It allows to refine vestibular central compensation thus diminishing the overdependence on peripheral retinal functions.

We have recently proposed a new device to improve this treatment, called the Stereoscopic Panoramic Panel (Fig. 2).

Under controlled conditions, sensory conflicts between the various relevant sensors are intensified and stimulate the restoration of the order of priorities of the integrative tasks: stable foveal fixation (object recognition), perception of depth (not to be confused with stereoscopic vision in the stricter sense) and a correct interconnection of visual, postural and visual perception [12].

The equipment consists of a swivel chair, a visual panel and a fixation target.

The patient is sitting in a swivel chair while fixating a target placed in front of him (attached to the armchair) (Fig. 2). The exercise consists of rotating the chair, at

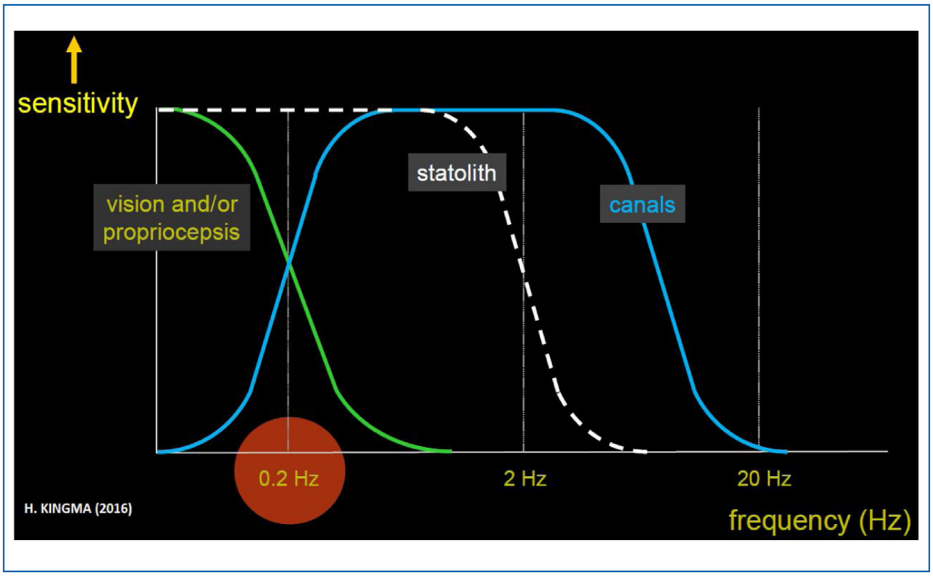

Figure 3. The angular velocity in this exercice is very slow and occurs in a transition zone of two captors competences (with kind permission of H. Kingma http://hermankingma.com/onewebmedia/2VestibularPhysiologyPhysicsLabyrinth.pdf).

low speed (less than 20°/s) and in a pendulum mode at a frequency of 0.1 to 0.2 Hz, in front of the stereoscopic panoramic panel (PSP). The exercise aims to increase the sensory conflicts resulting from the incongruence of different afferents involved in dynamic vision.

It is very efficient because the angular velocity used in this exercice is very slow and takes place in a transition zone of two captors competences (Fig. 3).

This exercise is usually very fast and very effective, and can, if necessary, be supplemented by conventional static orthoptic exercises.

Discussion, theoretical considerations concerning the Stereoscopic Panoramic Panel©

During the exercise, the peripheral retina provides motion information, while the fovea perceives a fixed and stabilized image. The work of N. Kanwisher and E. Wojciulik [13] demonstrates that the voluntary act of fixation of the foveal target is essential because it favours the parvocellular pathway, increases cortical activation in the temporal zone dedicated to discrimination and recognition of the object, while reducing the weight of the magnocellular pathway.

The result of this task is rapid desensitization with respect to optical flow, improved visual fixation and reduced intolerance to movement.

Moreover, the passive mobilization of the patient on a swivel chair, at low speed, in front of a visual decoration producing a powerful optokinetic stimulation but incoherent compared to the VOR (Fig. 4), makes it possible to act positively on the mechanism of storage of velocity by reducing the vestibular time constant.

This observation is consistent with the results of the Mingjia Dai, Ted Raphan and Bernard Cohen [14] study in which the authors showed that simultaneous and low frequency stimulation of angular and optokinetic vestibular reflexes by incongruous chair rotations. With the visual scene scrolling, brought, thanks to a decrease of the vestibular time constant, a lasting benefit for the subjects sensitive to the motion sickness.

Intolerance to visual flow, often coupled with vestibular hypersensitivity, are the main causes of vestibular asthenopia. Acting in a powerful and direct way on these two levels, the exercise has an immediate action on the neurovegetative reactions related to these symptoms.

Moreover, recent studies (A. Séverac Cauquil et al.) [15—18] have highlighted the existence of a relationship between optical flow and facilitation of 3D perception in a close space. It is likely, according to this same study, that the optical flux also has a tonigene effect facilitating convergence. This would explain why many patients experience the sensation of ''squinting'' during the fixation of the sight in the foreground of the perceived PPS in motion.

In addition, the exercise of concentration and solicitation multi-sensory, true brain gymnastics lasting about 15 minutes, results in a form of sedation thanks to the hypnotic effect it produces. This soothing sedation is an interesting therapeutic asset as many patients live with anxiety of their symptoms of vertigo, whatever their intensity [19]. This aspect needs further study.

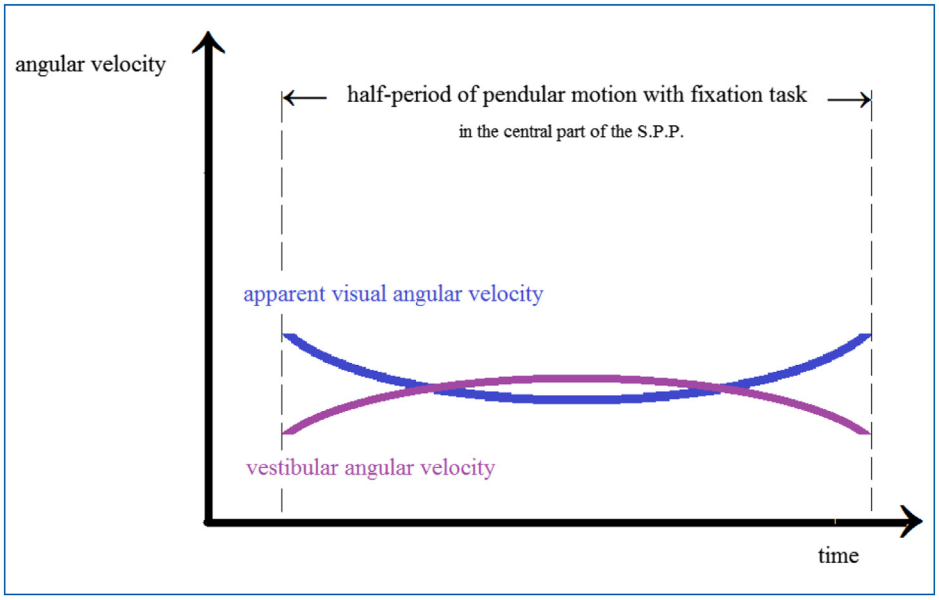

Figure 4. Incongruence between actual angular velocity (vestibular input) and visually perceived velocity (visual input).

Conclusion

Many patients who have experienced vertigo often have persistent residual symptoms, responsible for dizziness. These sub-clinical symptoms are usually unexplained and are not treated. Vestibular asthenopia is one of the possible explanations. It comprises a set of sensory symptoms, mainly visual, which are the result of a cascade process at the start of the vestibular incident.

Vestibular asthenopia could undoubtedly explain some of the disorders included under the name persistent postural perceptual dizziness (PPPD).

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Disclosure of interest

The authors declare that they have no competing interest.

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