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Benign Paroxysmal Positioning Vertigo: A Disease Explainable by Inner Ear Mechanics

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Key Words
Benign paroxysmal positioning vertigo · Inner ear mechanics · Free otoliths · Semicircular canals · Vestibular pathophysiology

Abstract
Many arguments give evidence that benign paroxysmal positioning vertigo, one of the most frequent kinds of vertigo, can be reduced to mechanical processes in the vestibular part of the labyrinth. Based on the assumption that otoliths have departed from their normally fixed position in the otolithic membrane and travel in the semicircular canals, clinical observations find their explanation. Latency between the onset of a head movement and the beginning of the vertiginous sensation, its duration and the nystagmic pattern provoked by specific head movements are in good correlation with the canalith hypothesis. Further arguments are the successful treatment by liberatory maneuvers, the relatively high number of recurrences and the change of the affected canal.

Introduction

Benign paroxysmal positioning vertigo (BPPV) is characterized by short attacks of vertigo, necessarily linked with head movements. These complaints were first described by Bárány [1] in 1921 as a clinical entity. He pointed to the dependence of this kind of vertigo on head positioning. BPPV became understood in 1968, when Schuknecht [2] presented two cases, in which otolith material was detected after death on the canal's cupula of patients who had suffered from positioning vertigo (fig. 1).

This cupulolithiasis hypothesis was criticized by Schuknecht himself, because not all characteristics of BPPV could be explained. Not till Parnes and McClure [3] developed the idea of canalolithiasis (fig. 1), the occurrence of free otoliths in the canal endolymph, did the characteristics of BPPV find a convincing explanation.

In what follows, we will point out some features of BPPV and its mechanical correlates.

Anatomy

The anatomical picture (fig. 2) of the labyrinth shows the structural and geometrical relations between the semicircular canals and the otolithic apparatus [4]. Two points are important for further considerations. First, it can be noted that the vestibular apparatus of each side is a system of communicating tubes, which allows the transport of the endolymph, but also of other material within the endolymphatic space. Second, the semicircular canals are built in an angle of approximately 90° among themselves.

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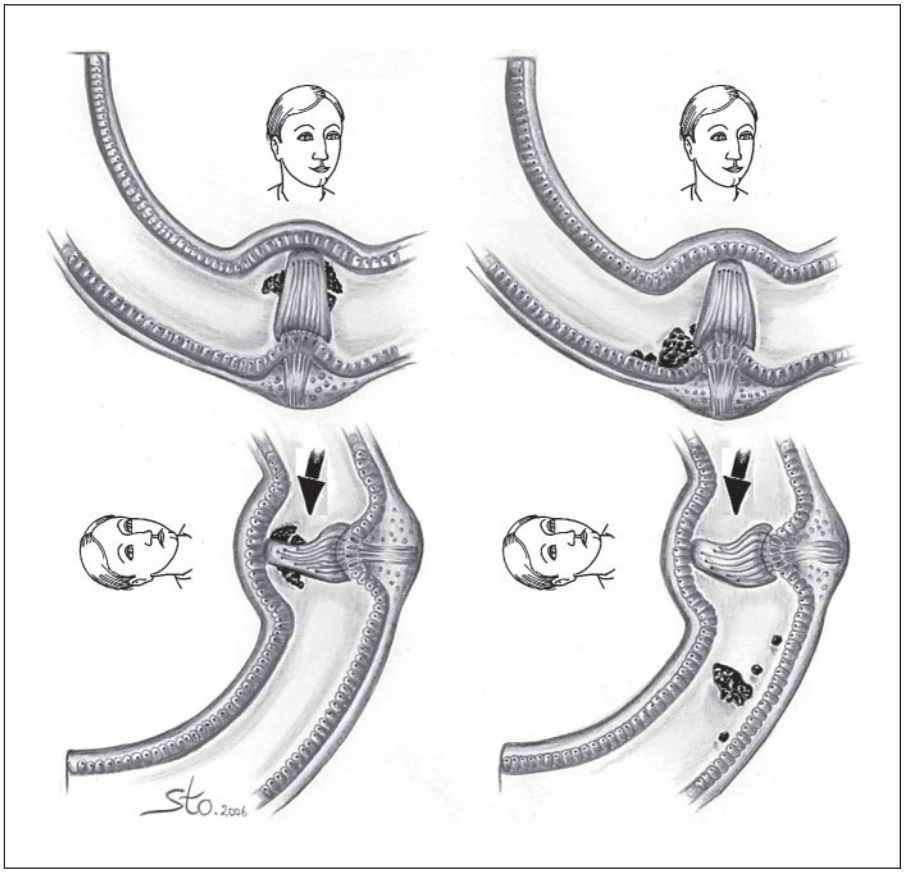

Fig. 1. The cupulith hypothesis (left) versus canalith hypothesis (right): free otoliths in the semicircular canal provoke an increase of endolymph flow and trigger nystagmus of limited duration (right), otoliths fixed on the cupula (left) induce also a deflection of the cupula, which triggers nystagmus of longer duration.

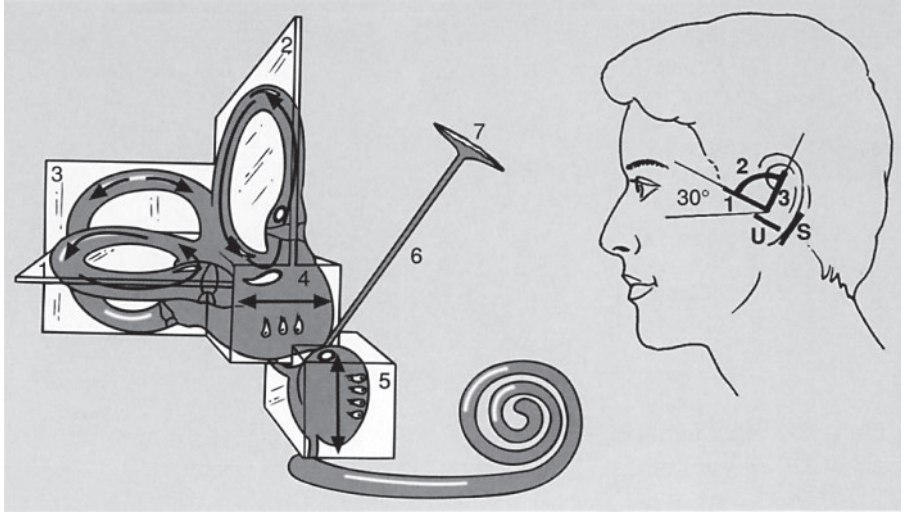

Fig. 2. Relationship and localization of the vestibular receptors. 1 = Working plane of the horizontal canal; 2 = working plane of the anterior canal; 3 = working plane of the posterior canal; 4 = main working plane of the macula utriculi; 5 = main working plane of the macula sacculi; 6 = endolymphatic duct; 7 = endolymphatic sac (after 4); S = sacculus; U = utriculus.

If one assumes that free otoliths are the cause of BPPV, an anatomical proof is required. The first proof came from the petrous bone observations of Schuknecht [2], who found inorganic material on the cupula in cases of BPPV. Further confirmations stem from microsurgery observations. In some cases of BPPV after drilling the bony wall of a semicircular canal, free-floating particles were seen [5]. The definite proof came from Welling et al. [6], who identified the free-floating particles as otoliths by electromicroscopic methods.

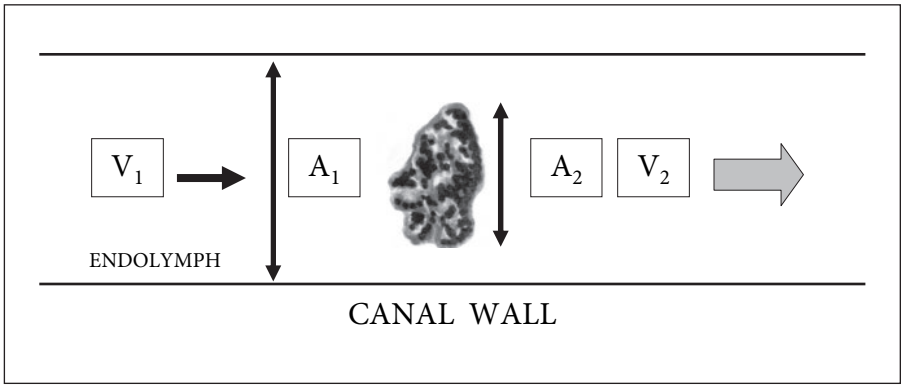

Fig. 3. Increase in endolymph velocity induced by occurrence of free otoliths in the endolymphatic space.

Pathophysiology

It is generally accepted that head movements induce the flow of the endolymph which leads to a deflection of the cilia in the cupula. The cilia deflection is an important step for the mechanoelectrical transformation and is finally the reason for the excitation of the sensory cells, which triggers the vestibular ocular reflex, i.e. gaze fixation during head movements [7]. If the stimulation is strong enough (a rapid head rotation), nystagmus appears. So, under normal conditions, head movements are followed by ocular movements to fixate, and under strong stimulus conditions, nystagmus is induced.

Based on these considerations, one can predict that nystagmus will appear even during normal head rotations if the force of the endolymphatic flow is strengthened. This is the case when otolithic material enters the endolymphatic space of the semicircular canals. The velocity of the fluid in a tube increases, proportionally to the cross section of the added material (fig. 3). The reason is the relationship given by the 'equation of continuity':

$A \cdot V = \text{constant} = Q$
A = area of the cross section
V = velocity
Q = volume flow
$A_1 \cdot V_1 = (A_1 - A_2) \cdot V_2 = \text{constant}$

If the cross section of the semicircular canal is reduced by the additional occurrence of a clot of free otoliths, the velocity increases (fig. 3).

Observations on the frog's vestibular apparatus gave experimental support for the canalith hypothesis [8].

Given that this hypothesis is true, the properties of BPPV can be predicted as will be explained in what follows.

Latency of the Positioning Nystagmus

Due to the inertia of the endolymphatic fluid, there must be a delay between the onset of the head movement and the beginning of the nystagmic reaction. Whereas the normal rapid gaze fixation by the vestibular ocular reflex runs instantly, the trigger mechanism of the positioning nystagmus needs a longer delay. Due to the occurrence of an inorganic material clot in the endolymphatic space, additional gravitational forces become effective which develop in a short time delay. This fits well with the clinically known latency of 3–5 s [9].

Duration of the Positioning Nystagmus

At the end of a normal head movement, endolymphatic flow stops within a short delay, which is defined by a characteristic time constant. In the case of canalolithiasis, the increased endolymph velocity is so strong that the maximally deflected cilia need a certain time to go back to the position at rest. The time course of the behavioral effects follows a time constant of approximately 30 s [7, 10].

Direction of the Positioning Nystagmus

It is generally accepted that for each semicircular canal a specific pathway to certain eye muscles exists in order to induce a typical eye movement. For example, the horizontal canal innervates the musculus rectus lateralis (contraction of the agonist) and musculus rectus medialis (relaxation of the antagonist); these muscles are responsible for the movements of the eye in the horizontal plane. Studies in cats by Suzuki et al. [11] give the experimental background and allow functional predictions.

Thus, it can be concluded that in case of canalolithiasis in the posterior canal geotropic beating horizontal-rotating nystagmus will be evoked, when the head is turned

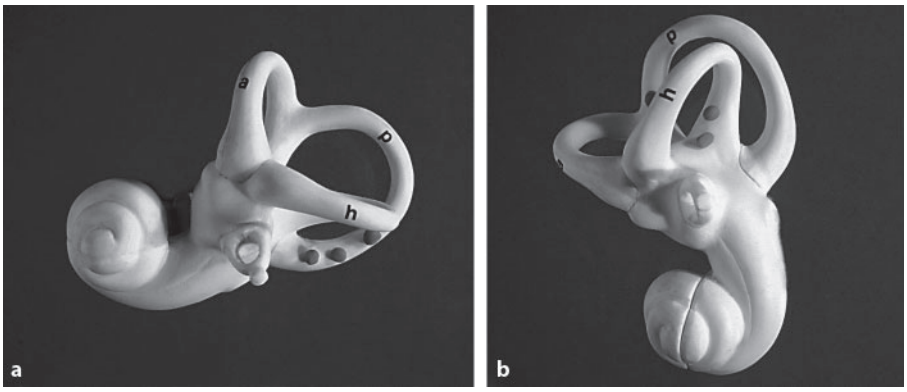

Fig. 4. Change of free otoliths, symbolized by red globules, from the posterior canal (p) (**a**) into the horizontal canal (h) (**b**) via the common crus during a liberatory maneuver.

in the plane of the affected canal and to the affected side.

Of course, in clinical practice the analysis starts with the observation of eye movements followed by a deduction of which canal is affected. For this, a specific positioning of the head in different planes depending on the semicircular canal to be examined must be executed.

Treatment

The knowledge about canal mechanics during BPPV leads to an effective treatment based on liberatory maneuvers. Following the anatomic assumptions and mechanical events, specific maneuvers [12, 13] were elaborated. The aim of the maneuvers is a clearing of the affected canal with a likely repositioning of the free otoliths to a site near the utricle, where they no longer cause pathological endolymph flow. The striking success of this type of treatment also supports the canalith hypothesis.

Recurrences and Change of the Canal

Two clinical facts, seen after the physical treatment, also reinforce the canalith hypothesis: the relatively high number of recurrences on the one hand and the change of the affected semicircular canal on the other hand.

Although the high efficiency of liberatory or repositioning maneuvers is generally accepted, the incidence of recurrences is remarkable. In our own series of 124 successfully treated cases of posterior canal BPPV, 10% of the patients reported reoccurrence of the positioning vertigo [14] within two years, which could be successfully treated by further liberatory maneuvers.

However, the high incidence of recurrences [15] is an additional argument for the canalith hypothesis, because it shows that free otoliths can be displaced by certain movements, but they cannot be dissolved.

The observation that free otoliths can change from one semicircular canal into another canal is highly interesting [16]. One patient out of 124 posterior canal BPPV cases showed this phenomenon [14]. Originally, canalolithiasis of the posterior canal had been diagnosed and treated specifically. A few days after the liberatory maneuver, the patient reported that vertigo continued, but now provoked by other head movements. The positioning test showed that there was no canalolithiasis in the originally affected posterior canal. But when testing the horizontal canal of the same side, a typical positioning nystagmus in the horizontal plane could be elicited (fig. 4). This observation, too, can only be explained if one accepts free-floating particles in the semicircular canal system, which can change location depending on certain head movements during the liberatory maneuver.

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