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Review

Measurement of endolymphatic pressure



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ABSTRACT

Endolymphatic pressure measurement is of interest both to researchers in the physiology and pathophysiology of hearing and ENT physicians dealing with Menière's disease or similar conditions. It is generally agreed that endolymphatic hydrops is associated with Menière's disease and is accompanied by increased hydrostatic pressure. Endolymphatic pressure, however, cannot be measured precisely without endangering hearing, making the association between hydrops and increased endolymphatic pressure difficult to demonstrate. Several integrated in vivo models have been developed since the 1960s, but only a few allow measurement of endolymphatic hydrostatic pressure. Models associating measurement of hydrostatic pressure and endolymphatic potential and assessment of cochlear function are of value to elucidate the pathophysiology of endolymphatic hydrops. The present article presents the main types of models and discusses their respective interest.

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1. Introduction

The clinical signs of hydrops classically associate recurrent vertigo, hearing loss that is often fluctuating and initially low frequency, and tinnitus that is often of a buzzing type with a sensation of ear fullness. Since these clinical signs of endolymphatic hydrops were first described by Prosper Menière in the 19th century, the exact underlying pathophysiology has remained unclear. In particular, the question of the change in pressure that may be induced by distension of the endolymphatic compartment (or vice versa) characterizing hydrops is a pertinent one. On the one hand, the scala media endolymph communicates with other fluid sectors, notably with the saccule via the ductus reuniens; on the other, the membranes around the cochlear endolymphatic sector (Reissner's membrane and basilar membrane) may be distended, thereby limiting the pressure increase induced by volume inflation. In clinical practice, Menière's disease is often grouped together with similar but clinically incomplete presentations, accepting the idea of physiopathological similarity between such so-called "pressure" syndromes. According to the American Association of ORL and Head and Neck Surgery (AAO-HNS) 1995 classification, certain diagnosis of Menière's disease can only be founded on histologic cross-sections demonstrating hydrops (i.e., post-mortem) [1], and this

definition has yet to be challenged. The scientific community thus recognizes endolymphatic hydrops as the anatomic substrate of Menière's disease, but without requiring proof of endolymphatic hyperpressure.

Intralabyrinthine pressure measurement is crucial to better understanding Menière's disease. The major obstacle is the risk to the inner ear. Integrated experimental animal models are of great value in this regard.

The present update is based on a PubMed review of the literature, presenting known fundamental and clinical experimental results and an outline of ongoing research.

2. Discussion

Many animal models of hydrops are found in the literature. Recently, Salt and Plontke published a review of findings based on such models [2]. A prime distinction is between acute and chronic models, the latter probably corresponding more closely to established non-reversible hydrops and the former to early stages of the disease when hydrops is probably fluctuating. The two are complementary and together have shed light on the pathophysiology of Menière's disease. Functionally, most models test cochlear function and few focus on vestibular function. It is noteworthy that not all allow concomitant measurement of endolymphatic pressure and gross cochlear potentials: indeed, it is not easy to obtain reliable measurements of pressure in a very small volume (about 4.7 μ L in guinea pig) [3].

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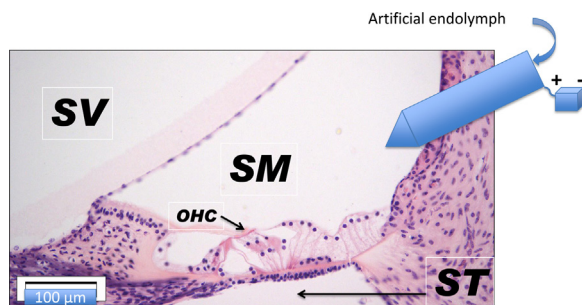


Fig. 1. Glass micropipette in the endolymphatic compartment, across the bony labyrinth and stria vascularis, at the basal turn of the cochlea, allowing injection of artificial endolymph and measurement of the endolymphatic potential (symbolized by the cube with “+” and “-” signs above it). SV: stria vascularis; SM: scala media; ST: scala tympani; OHC: outer hair cell.

2.1. Chronic hydrops models

In 1965, Kimura and Schuknecht published a guinea pig model of chronic endolymphatic hydrops with ablation of the endolymphatic sac [4]. Although extreme, the model proved effective in inducing chronic endolymphatic hydrops, as demonstrated by histologic findings of endolymphatic fluid inflation after sacrifice. The experimental chronic hydrops was associated with impairment of vestibular function (notably nystagmus) and hearing. Gross cochlear potentials were altered, notably with a reduction in endolymphatic potential (EP) [5] exacerbated by chronicity [6]. Disorders of endolymph ionic composition were observed, and notably a reduced K⁺ concentration [7]. Histology found severe atrophy of the stria vascularis, again exacerbated by chronicity, establishing a link between reduction in EP and stria involvement in these models [5]. The chronic models also showed reduced composite action potential (AP) and an increase in summation potential (SP) and hence in SP/AP ratio [8]. This altered SP/AP ratio in chronic hydrops models is reminiscent of certain reports in Menière's disease [9].

More recently, a more subtle chronic model was published, intended to reflect the reality of Menière's disease more closely [10], with partial lesion of the endolymphatic sac. Endolymphatic hydrops was confirmed on histology in 68% of cases, but without pressure difference between peri- and endolymph. The report thus demonstrated that hydrops is not necessarily associated with increased hydrostatic pressure. Like in Kimura's model [5], EP was reduced.

2.2. Acute hydrops models

The first problem is how to create a model of acute hydrops without destroying the cochlea. Researchers have thus frequently opted for gradual inflation of endolymphatic volume. It turns out to be very hard to induce significant change in endolymphatic pressure without destroying cochlear function [11].

The second problem is how to couple pressure and functional measurements while preserving access to the endolymph so as to change its volume.

Endolymph access may be lateral via the bony wall of the basal turn of the cochlea, through the spiral ligament bearing the stria vascularis [12] (Fig. 1), so that only the scala media is likely to be catheterized. A sufficiently fine pipette can freely perfuse artificial endolymph so that the resultant functional and pressure changes can be analyzed (Fig. 2).

One of the first such models was that of Kakigi and Takeda in 1998 [12]. EP can be measured simultaneously and auditory thresholds are determined on electrocochleography. However, the authors did not integrate hydrostatic pressure among the

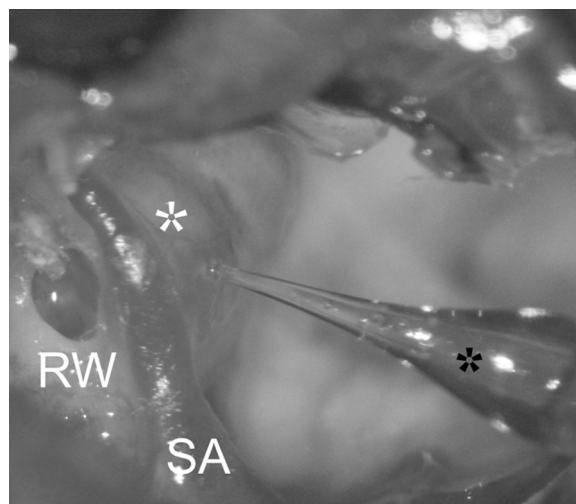


Fig. 2. Posterolateral view of a right cochlea (gerbil) with micropipette (black star) in position in the cochlear duct. RW: round window; white star: basal turn of the cochlea; SA: stapedial artery persisting in gerbil (personal experience).

functional assessments. EP measurements turned out to contradict those of Kimura-type chronic models [5]: overall, slow injection of artificial endolymph (300–500 nL/min) induced a transient rise in EP. The explanation is by no means obvious, contrasting with the EP reduction found in chronic models. It does, however, go to show that EP reduction in chronic models is not due to endolymph volume inflation as such but more to its likely impact on the stria vascularis, which is moreover often atrophied in these models [5].

There have been other published models of endolymphatic pressure measurement, some via the round window rather than the lateral wall of the cochlea. Two ducts, the scala media and the scala tympani, can be catheterized for differential measurement [13]: continuous measurement of probe-tip electrolytic potential confirms the passage of the probe into the scala media, EP, at about 150 mV, being considerably greater than the perilymphatic potential. Dedicated systems allow probe-tip potential and concomitant hydrostatic pressure to be measured: e.g., the WPI 900A micropressure system (World Precision Instruments Inc., Sarasota, FL, USA). Table 1 presents the characteristics of the main hydrops models known to measure endolymphatic pressure.

The question arises as to which is the key element actually underlying the cochlear functional disturbances observed in endolymphatic hydrops: change in endolymphatic pressure or in endolymph volume?

One of the simplest explanations that could account for the known clinical presentation of hydrops concerns displacement of the operating point of the hair cells of the organ of Corti in equilibrium. Volume inflation inevitably induces a transient increase in hydrostatic pressure, tending to displace the basilar membrane toward the scala tympani and Reissner's membrane toward the scala vestibuli [18]. This change in position affects the position of the cilia of the hair cells and may disturb mechanical-electrical transduction and the sensitivity of the organ of Corti, which are determined by the position of the cilia [19,20]. The presently available patch-clamp data demonstrate that outer hair cell (OHC) cilia positioning determines the probability of mechanical transduction ion-channel opening, thereby regulating cell excitation, which is maximal for a probability of 50% and decreases as the probability falls below or rises above this value. The resulting cochlear micromechanics are subject to more or less intense electromechanical feedback from the OHCs [21]. The same kind of explanation can be given for the reversibility of hydrops-induced disorder. The

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