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Simulation of the Basilar Membrane Vibration of Endolymphatic hydrops

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Abstract

Endolymphatic hydrops (EH) causes fluctuating hearing loss especially at low frequencies. In this paper, we analyze EH with the human cochlear finite element model which includes activity of the outer hair cells (OHC). EH was simulated by loading static pressure on the surface of the basilar membrane (BM) of the cochlear model. The vibrations of the BM in the EH model were calculated by applying sound pressure to the stapes, and the results were compared with those in the normal cochlea model. As a result, the distribution of characteristic frequency (CF) was changed and the displacement of the BM was decreased by loading static pressure to the BM. This suggests that the mechanism of hearing loss was caused by EH.

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Keywords: Endolymphatic hydrops; Meniere's disease; Fluctuating hearing loss; Outer hair cells; Basilar membrane; Finite element method; Human cochlear model

1. Introduction

The bony labyrinth of the cochlea is divided into three compartments by the BM and the Reissner's membrane (RM), i.e., the scala vestibuli (SV), the scala media (SM), and the scala tympani (ST) (Fig. 1 (a)). The SV and the ST are filled with perilymph fluid and the SM is filled with endolymph fluid. EH is a disorder of the inner ear and is associated with the anomalous flow of the endolymph fluid. This anomalous flow causes an increase of pressure in the SM of the cochlea. Hallpike et al., [1] proposed that EH is a pathogenesis of Meniere's disease based on autopsy

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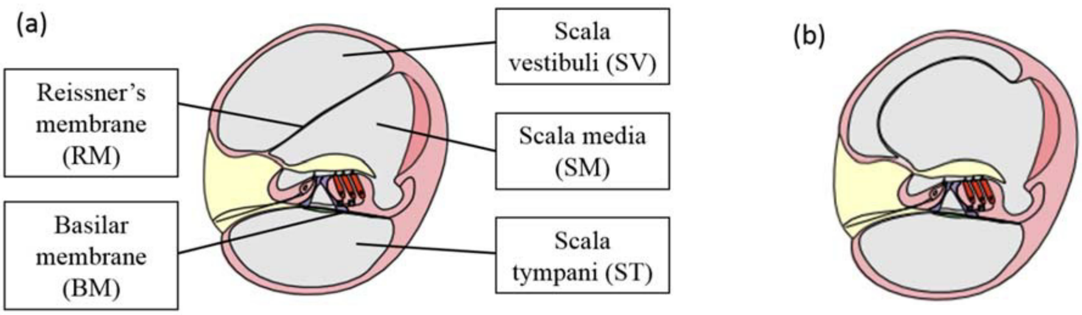

Fig. 1. Cross section of the cochlea (a) normal cochlea; (b) cochlea of EH. The volume of the SM in (b) is larger than that in (a)

cases in which there was a larger volume of endolymph than in the normal cochlea, and an abnormality of the RM was observed (Fig. 1 (b)). The initial symptoms of EH include fluctuating hearing loss (a hearing loss that seems to frequently change) especially at low frequencies, and balance problems. Pathogenesis of these symptoms have not yet been elucidated. There are two types of hypotheses on the pathogenesis of fluctuating hearing loss and vertigo caused by EH. Schuknecht [2] suggested that RM rupture causes an increment in the concentration of potassium ions of perilymph. This irreversible damage causes such symptoms. On the other hand, Tonndorf [3] suggested that EH causes an increase of pressure in the SM. This increase of pressure causes poor blood circulation which then brings about the same symptoms. Many clinical reports suggest that an effective cure for Meniere's disease is blood circulation promotion with a diuretic, which reduces the pressure in the SM. However, the mechanism of hearing loss at low frequencies is unclear.

Therefore, the aim of this study is to clarify the mechanism of fluctuating hearing loss caused by an increase of pressure in the SM using the human cochlear finite element model which includes the activity of the OHC. The effect of this increase-pressure in the SM was analyzed using the model.

2. Methods

2.1. Human cochlear finite element model

The human cochlear finite element model (Fig. 2) of this study was determined following the model of Koike et al., [4]. The vestibular part was modeled with the stapes, the stapedial annular ligament (SAL), and the oval window. The cochlear labyrinth part was modeled with the BM, the osseous spiral lamina (OSL), the round window, and the cochlear aqueduct. This model is simplified to avoid complications of calculation. The model has a linear shape, and the width and thickness of the BM change linearly from the base to apex (Table 1). The SV and SM were unified and the RM was ignored. The volume of the lymph and the shape of the BM were determined on the basis of reported values in human cadaver. Each parameter of the model is determined based on the reported values. The unknown parameters were determined by conforming calculation results to the values of the measurements. The lymph was considered incompressible viscose fluid. The viscosity of the lymph was determined based on that of blood with a low hematocrit value. The BM was assumed to be an orthotropic material because the collagen fibers run in the direction of the width of the BM (in the direction of the X-axis).

In this study, the human cochlear model included the amplification mechanism of the OHC because the human cochlea under physiological conditions have a motility which amplifies the vibration of the BM. This mechanism was represented by the excitation force $\mathbf{P}_{\mathrm{OHC}}$ as a function of the BM velocity, $\mathbf{V}_{\mathrm{BM}}$ in Eq. (1) below:

$$\mathbf{P}_{\mathrm{OHC}} = \mathbf{a}(z)\mathbf{b}(z)\tan^{-1}\{\mathbf{c}(z)\mathbf{V}_{\mathrm{BM}}\} \tag{1}$$