蝸牛内コルチ器における変形挙動解析用プログラムの開発

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研究発表

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Abstract

The organ of Corti (OC) in the cochlea transforms sounds into action potentials in auditory nerve fibers. As the extreme vulnerability to trauma exhibited by the cochlea prevents the experimental observations that could confirm the inheritance in this process, the two-dimensional finite-element model of the OC was constructed. Using this model, the dynamic behavior of the OC was analyzed. As the OC is immersed in lymph fluid, the interaction between the OC and the lymph fluid must be taken into account when the dynamic behavior of the OC and fluid pressure distribution are numerically analyzed. However, the complex structure of the OC and the large difference in material properties between the fluid and the structure of the OC complicate modeling of the lymph-OC interaction using commercially available FEM applications. Therefore, the original program was developed to consider the fluid-structure interaction.

When a fluid pressure fluctuation is induced by vibration of the stapes, two types of pressure waves, so-called fast and slow waves, occur in the cochlea. As the OC is driven by these pressure waves, it is important to understand their frequency characteristics. However, there have been no reports on empirical observations of these waves, because of the difficulty of measuring them independently. Using the model, the magnitude and phase of the fast and slow waves to the model were predicted so as to fit the numerically obtained pressure distribution in the scala tympani with that of the intracochlea pressure measurement. Next, the dynamic behavior of the OC was analyzed when these predicted pressures were applied to the OC. Consequently, it was found that the magnitude of the fast wave increases with increasing frequency for the entire frequency range, and the magnitude of the slow wave increases gradually with increasing frequency until it reaches a maximum at the characteristic frequency (CF), and it then falls sharply. It was also found that the OC shows a rotational movement around a point near the foot of the inner pillar cell. Finally, by comparing our numerical results with experimental data, it was confirmed that the availability of the model to simulate dynamic behavior of the OC.
1. Introduction

It is known that the remarkable sensitivity and frequency selectivity exhibited by the cochlea are properties that are established by the mechanical motion of the organ of Corti (OC). Eighty percent of the significant hearing loss patients are attributed to pathological changes in this motion, so an accurate understanding of the cochlear amplifier would have profound clinical significance. For example, it may help to analyze firing behavior in the auditory nerve and it will be of special use to predict the responses when new hearing aid devices are developed. This requires characterization of the role played by each of the structures of the OC in vivo, which is loaded by the fluids surrounding them.

The auditory transduction process relies upon specific details of this motion to generate an accurate neural representation of the sound stimulus. However, how the OC moves under the physiological condition is controversial because it is difficult to observe the dynamic behavior of the OC directly. The extreme vulnerability to trauma exhibited by the cochlea prevents the experimental observations that could confirm the inherence in this process. In order to dissolve the mechanics of cochlea, numerical analyses of the cochlea have been developed. Most cochlear models were reconstructed using the electrical circuit that finds applications in many areas of electrical engineering. Many of them are able to realistically simulate the gross mechanical response of the OC, but their formulation simplifies the complex structure of the cochlea to reduce independent variables.

Our study intends to analyze the dynamic behavior of the OC, which is the key of the mechano-electrical transduction exhibited in the cochlea. As the OC is immersed in lymph fluid, the interaction between the OC and the lymph fluid was taken into account when the dynamic behavior of the OC and fluid pressure distribution are numerically analyzed. The two-dimensional finite-element models of the OC and lymph fluid were constructed. The fluid-structure interaction between the model of the OC and those of the lymph fluid was considered by means of a staggered approach. Using these models, first,
unknown Young's moduli of individual portions within the OC model were determined based on the static stiffness measurement of the OC (Naidu and Mountain, 1998). The frequency characteristics of the fast and slow waves, which are pressure waves in the cochlea, were then predicted so as to fit the numerically obtained distribution of the intracochlea pressure with the experimentally obtained one (Olson, 2001). Finally, the dynamic behavior of the OC was analyzed when these predicted pressures were applied to the OC, and it was compared with experimental data to confirm the availability of the model.
2. Role of the cochlea in hearing

2.1. Anatomy

Figure 2.1 shows a simplified sketch of the human auditory system. The sound entering the external auditory meatus vibrates the tympanic membrane. These vibrations are mechanically transmitted by the ossicles (malleus, incus and stapes) to the cochlea. The cochlea contains the sensory cells which convey the acoustic signals to the auditory nerves.

The mammalian cochlea consists of a fluid-filled duct that is coiled like a snail shell and is embedded in bone. Figure 2.2 shows a simplified sketch of the cross section of the cochlea. The cochlea is divided into three compartments by two longitudinal membranes. The upper membrane, i.e., Reissner’s membrane, divides the scala vestibuli (SV) from the scala media (SM). Reissner’s membrane is very compliant, and has little effect on cochlea hydromechanics. Its main function is an ionic barrier between the SV and the SM. The fluid in the SM has an electrical potential (endocochlear potential) that is 90 mV more positive than that of the SV and the scala tympani (ST). These ionic and potential gradients are maintained by specialized cells located in the stria vascularis, which makes up much of the outer wall of the SM.

Figure 2.3 shows some of the key features of the OC. The OC sits upon the basilar membrane (BM) which separates the SM from the ST. Today, it is known that the OC is the key element which provides the mechano-electrical transduction. The BM is attached to a bony shelf called the spiral lamina at its inner side and to a specialized tissue called the spiral ligament at its outer edge. The spiral ligament is attached to the bone by anchoring cells and may serve to create or maintain tension in the spiral ligament-BM complex (Henson et al., 1985). The width of the cochlea duct and the spiral lamina decreases from base to apex while the width of the BM increases from base to apex.

The cells of the OC are usually divided into two groups: the sensory cells (hair cells
and nerve endings), and the supporting cells. The pillar cells form an arch that divides the OC along its radial direction into two regions, one containing a single row of inner hair cells (IHCs) and the other containing three rows of outer hair cells (OHCs). The pillar cells are quite rigid and appear to play an important structural role within the OC. The IHCs get their name from their location on the inner side of the cochlea spiral. The OHCs are located toward the outer side of the cochlear spiral and are believed to play a motor function in the mechanical performance of the cochlea. They receive significant efferent innervation from fibers that originate in the medial superior olivary nucleus of the brainstem providing neural control of cochlear mechanics. The OHCs are always located at a significant angle to the BM and are approximately parallel to the outer pillar cells.

The BM is divided along its radial direction into two regions, i.e., the arcuate and pectinate zones. The arcuate zone extends from the spiral lamina to the foot of the outer pillar cell, and the pectinate zone extends from the outer pillar cell to the spiral ligament. The force needed to displace the BM has been measured as a function of radial position (Olson and Mountain, 1994; Naidu and Mountain, 1998). The region of the BM directly under the foot of the outer pillar cells is stiffer than the regions of either side. The pectinate zone was found to be stiffer than the arcuate zone.

The apical membranes of the pillar cells, hair cells, and some other supporting cells are tightly joined together to form a platelike structure on the top of the OC is called the reticular lamina (RL). These tight junctions form an ionic barrier between the SM and the OC. In contrast, the BM appears to be reasonably permeable to ions with the result that the ionic composition of the fluid within the OC is very much like that of the perilymph, which fills the SV and the ST. One group of cells that make up part of the RL is the Deiter’s cells. Their cell bodies are located beneath the OHCs and form cups within which the OHCs are seated. Each Deiter’s cell has a long thin process, called phalangeal process, that projects to the RL. Each of phalangeal process ends in a platelike structure, and these structures are interdigitated between the OHCs.
The limbus is a structure that protrudes from the upper surface of the spiral lamina and provides the major attachment point for the tectorial membrane (TM). The TM is an extracellular matrix and extends from the limbus slightly beyond the outmost row of OHCs. The tallest hairs of the OHCs appear to be embedded in the TM. Whether the IHC stereocilia are in contact with the TM remains unresolved (Lim, 1980; Ulfendahl et al., 2001). Another unresolved issue is whether the outer edge of the TM is connected to the RL.

The stapes provides the sound pressure input to the SV from the middle ear. At the apical end of the cochlea, the SV is connected to the ST via the helicotrema, which equalize the static pressures between the two scalae. The ST has a membrane-covered contact with the middle ear cavity at the round window.

### 2.2. Mechano-electrical transduction

The vibrations of the tympanic membrane are finally transmitted to BM vibrations in the cochlea through the cochlea fluid. On the BM, the OC transforms the BM vibrations to action potentials in auditory nerve fibers. As a result, we perceive the sounds. This process is known as mechano-electrical transduction of the IHC.

A number of physiological and functional studies concerning the IHC have provided considerable information about the mechano-electrical transduction. Figure 2.4 shows the schematic illustration of the IHC and vibration mode of the OC. The IHC has a three-row structured hair bundle called stereocilia on the top of the cell. The stereocilia of the IHC are thought to be tilted by viscous drag of fluid streaming between the TM and the RL (Kimura, 1966; Engstrom, 1978; Lim, 1972). The deflection of the stereocilia is supposed to cause opening of K+ channels in the apical region of the stereocilia and influx of K+ ions into the cell (Davis, 1958; Pickles, 1984), which depolarize the intracellular potential of the IHC (Hudspeth and Corey, 1977; Russell et al., 1986). As a result, the neurotransmitter is released and action potentials are produced in auditory nerve fibers. Katz and Miledi
(1967) demonstrated that the IHC discharges impulses in proportion to IHC's depolarization.

2.3. Frequency selectivity of the cochlea

The cochlea is known to have sharp frequency selectivity. When the vibration of the stapes is transmitted to the BM through the cochlea fluids, the vibration of the BM takes the form of waves that travel down the BM (von Békesy, 1960). Figure 2.5 shows the schematic of the traveling wave. When a pure tone is transmitted to the BM, the region of its maximum amplitude of the vibration is related to the frequency of the tone. In other words, each has maximum amplitude at a specific frequency called characteristic frequency (CF). More basal regions of the cochlea have higher CFs, while the characteristic place of lower frequencies shifts progressively toward the apex. Figure 2.6 (a) displays that the traveling waves on the BM have a peak near the base when high-frequency sounds enter the cochlea. By contrast, low frequency sounds develop the traveling waves on the BM which have a peak near the apex. Figure 2.6 (b) shows a schematic drawing of the place-CF map in the human. The CF distributions differ within species.
Figure 2.1. Schematic of the human auditory system. The external auditory meatus conducts sound to the tympanic membrane. The vibration of the tympanic membrane is mechanically transmitted by the ossicles (malleus, incus and stapes) to the cochlea.
Figure 2.2. Schematic of the cochlea and its cross section. The three fluid-filled tubes (scala vestibuli, scala media and scala tympani) are separated from each other by Reissner’s membrane and basilar membrane. The organ of Corti contains sensory cells which detect sound signals.
Figure 2.3. Structure of the organ of Corti. The organ of Corti contains sensory and supporting cells. The inner hair cell and three rows of outer hair cells are sensory cells. The tectorial membrane is an extracellular matrix and covers the organ of Corti. Auditory nerve fibers enter the organ of Corti from modiolus.
Figure 2.4. Schematic illustration of the IHC and vibration mode of the organ of Corti. The IHC has a three-row structured hair bundle called stereocilia on the top of the cell. The stereocilia of the IHC are thought to be tilted by viscous drag of fluid streaming between the tectorial membrane and the reticular lamina (Kimura, 1966; Engstrom, 1978; Lim, 1972).
Figure 2.5. The vibration pattern of the BM. The spiral-shaped BM is straightened out. When the vibration of the tympanic membrane is transmitted by stapes to the BM, the vibration of the BM takes the form of waves that travel away from the stapes and toward the cochlear apex.
Figure 2.6. Frequency selectivity of the cochlea.

(a) The vibration mode of the BM. The traveling waves on the BM have a peak near the base when high-frequency sounds enter the cochlea, while low frequency sounds develop the traveling waves on the BM which have a peak near the apex.

(b) Schematic drawing of the place-characteristic frequency (CF) map in the human. The CF distributions differ within species.
3. Model

3.1. Geometry

The OC at the basal turn in the gerbil cochlea, the distance of which is 2.5-3.0 mm from the base, was modeled as shown in Fig. 3.1. Table 3.1 indicates geometric parameters of the OC model which were determined based on the measurement in the hemicochlea of the gerbil (Edge et al. 1998). Meshing was done at a subcellular level using a triangular element, by which the number of nodes and elements were 1,274 and 2,139, respectively. The fluid within the Corti tunnel was treated as an elastic body without shear stiffness. Although there is no element in the sub-tectorial space which is a narrow space between the TM and RL, the viscous force was considered analytically on the assumption that Couette flow occurs in this space. The effect of the mass of the fluid in the sub-tectorial space was assumed to be negligible, because the volume of this space is inconsiderable in comparison with that of the SV.

To simulate the behavior of the lymph fluid and its interaction with the OC, models of the lymph fluid in the SV and the ST were constructed as shown in Fig. 3.2. The dark area of each model corresponds to the OC. As the fluid pressure distributions in the scalae are influenced by the lymph fluid in the longitudinal direction, three-dimensional fluid models were constructed. The longitudinal width of each model was determined to be 48 μm, a value which is less than one-fourth of the wavelength of the traveling wave (Ren, 2002). In consideration of the modiolus and the cochlear wall, the left and right boundaries of these models were fixed. Longitudinal boundaries of both models, the boundary of the SV model at 150 μm from the BM and that of the ST model at 120 μm from the BM were also fixed because it was assumed that the lymph fluid did not move across those boundaries. Mesh having intervals of 6 μm made it possible to evaluate the pressure distribution around the OC in the scalae. As a result, the SV model and the ST model have 11,200 and 8,000 cubic elements, respectively.
3.2. Mechanical properties

Young's moduli of individual portions in the model were based on measurements *in vitro* and *in situ*. Young's modulus applied to the model was $6.0 \times 10^2 \text{ N/m}^2$ at the TM (Zwislocki, 1989), $1.0 \times 10^4 \text{ N/m}^2$ at the OHCs (Ulfendahl, 1998), $1.0 \times 10^7 \text{ N/m}^2$ at the phalanx (Laffon, 1996) and $1.0 \times 10^9 \text{ N/m}^2$ at the pillar cells (Tolomeo *et al.*, 1997). Young's moduli of the IHC and the Deiters' cells were assumed to be the same as those of the OHCs and the phalanxes, respectively. Young's moduli of the Kimura's membrane, which is the undersurface of the TM, and the RL were assumed to be $1.0 \times 10^6 \text{ N/m}^2$ and $1.0 \times 10^9 \text{ N/m}^2$, respectively, because these structures would be stiff enough to support adjacent structures. As the reported Young's modulus for the cortical bone has been shown to be $2.0-2.2 \times 10^{10} \text{ N/m}^2$ (Ashman *et al.*, 1984), that of the osseous spiral lamina, which is also a bone, was assumed to be $2.0 \times 10^{10} \text{ N/m}^2$. Young's modulus of the stereocilia was assumed to be $1.0 \times 10^7 \text{ N/m}^2$ (Zetes and Mountain, 1997).

Although Young's moduli of the BM and Hensen's cell would have a profound effect on the dynamic behavior of the OC, those of the gerbil have not been reported. To determine Young's moduli of these portions, the stiffness of the OC obtained by numerical analysis was compared with that of the gerbil measured by Naidu and Mountain (1998). According to their experiment, the stiffness of the OC was 2–4 N/m at a point beneath the OHCs when the OC at the basal turn was statically deflected. If the cells were removed from the BM, the stiffness of the BM was assumed to be 1.3–2.6 N/m at a point beneath the OHCs. Using the BM model, which was a reduced representation of the OC model as shown in Fig. 3.3, Young's modulus of the BM was determined to be $1.0 \times 10^7 \text{ N/m}^2$. Using the OC model as shown Fig. 3.1, Young's modulus of the Hensen's cells was then determined to be $5.0 \times 10^3 \text{ N/m}^2$.

The Poisson's ratio of soft cells and the TM, which is composed of the extracellular matrix, was assumed to be 0.49 because these portions are nearly incompressible, whereas those of hard cells and the osseous spiral lamina were assumed to be 0.3 because this value
is commonly used in structure analysis.

To guide the eye, the OC model shown in Fig. 3.1 was colored with shades of gray, a different shade being used for each portion having the same mechanical property. For the lymph fluid models, mechanical properties were assumed to be the same as those of water. Table 3.2 shows all mechanical properties of the models.

3.3. Formulation

In the OC model, it was assumed that the cross section of the OC maintains its plane surface when external force is applied to the OC. Therefore, the model of the OC was formulated under the plane strain condition. The equation of the motion of the structure by the finite-element process is represented by the following matrix differential equation:

\[
[M] \frac{\partial^2 \mathbf{u}}{\partial t^2} + [C] \frac{\partial \mathbf{u}}{\partial t} + [K] \mathbf{u} = \mathbf{f},
\]

(1)

where \([M]\), \([C]\) and \([K]\) are the mass, damping and stiffness matrices, respectively, \(\mathbf{u}_s\) is the structural displacement vector, \(\mathbf{f}\) is the force vector and \(t\) is the time. The damping matrix \([C]\) is derived from the linear combination of mass and stiffness matrices, i.e.,

\[
[C] = \alpha[M] + \beta[K],
\]

(2)

where \(\alpha\) and \(\beta\) are Rayleigh damping parameters. In the Newmark-\(\beta\) method (Newmark, 1959), the displacement vector at the end of a time interval can be expressed in terms of the displacement, velocity and acceleration vectors at the beginning of the interval as follows:

\[
\begin{align*}
\left[ [K] + \frac{2}{\delta t} [C] + \frac{4}{(\delta t)^2} [M] \right] \mathbf{u}_{n+1} &= \mathbf{f}_{n+1} + [M] \left( \frac{\partial^2 \mathbf{u}_n}{\partial t^2} + \frac{4}{\delta t} \frac{\partial \mathbf{u}_n}{\partial t} + \frac{4}{(\delta t)^2} \mathbf{u}_n \right) + [C] \left( \frac{\partial \mathbf{u}_n}{\partial t} + \frac{2}{(\delta t)^2} \mathbf{u}_n \right),
\end{align*}
\]

(3)

where \(\delta t\) is the time interval and \(n\) is the time step. The velocity and acceleration vectors at the end of a time interval can be expressed in terms of the velocity and acceleration vectors at the beginning of the time interval and the displacement vector at the end of the time.
interval by the relations
\[
\frac{\partial \mathbf{u}^{n+1}}{\partial t} = -\frac{\partial \mathbf{u}^n}{\partial t} + 2 \frac{\partial t}{\partial t} \left( \mathbf{u}^{n+1} - \mathbf{u}^n \right),
\]
(4)
\[
\frac{\partial^2 \mathbf{u}^{n+1}}{\partial t^2} = -\frac{\partial^2 \mathbf{u}^n}{\partial t^2} - 4 \frac{\partial \mathbf{u}^n}{\partial t} + 4 \frac{\partial \mathbf{u}^{n+1}}{\partial t^2} \left( \mathbf{u}^{n+1} - \mathbf{u}^n \right).
\]
(5)

Using Eqs. (3), (4) and (5), values of the vectors at time step \( n + 1 \) can be obtained from the previously determined values of the vectors and the known value of the force vector.

In the lymph fluid models, as characteristic values are \( L = 170 \mu m \) and \( U = 1 \) mm/sec, where \( L \) is the length of the BM and \( U \) is the approximate maximum fluid velocity in the vicinity of the BM which was estimated from experimental data (Olson, 2001), the Reynolds number (Re) of the lymph fluid model is defined as
\[
Re = \frac{\rho UL}{\mu} \approx 0.17,
\]
(6)
where \( \rho \) is the density of water (1 g/cm\(^3\)). In this range of Reynolds number, an incompressible and viscous flow can be assumed, and thus an incompressible Navier-Stokes equation was used to analyze the dynamic behavior of the lymph fluid. The incompressible Navier-Stokes equation is given by
\[
\frac{\partial \mathbf{v}_f}{\partial t} + (\mathbf{v}_f \cdot \nabla) \mathbf{v}_f + \frac{1}{\rho} \nabla p_{OC} - \nu \Delta \mathbf{v}_f = 0,
\]
(7)
where \( \mathbf{v}_f \) is the fluid velocity vector, \( p_{OC} \) is the fluid pressure caused by the OC motion, \( \nu \) is the kinetic viscosity of the fluid, \( t \) is the time, and the gradient operator \( \nabla \) and the Laplacian operator \( \Delta \) are defined in the following form
\[
\nabla = \left( \frac{\partial}{\partial x}, \frac{\partial}{\partial y}, \frac{\partial}{\partial z} \right), \quad \Delta = \frac{\partial^2}{\partial x^2} + \frac{\partial^2}{\partial y^2} + \frac{\partial^2}{\partial z^2}.
\]
(8)

Using a Marker-and-Cell (MAC) method (Harlow, 1965), the fluid is decomposed into rectangular parallelepiped cells and the pressure is discretized at the center of the cell. Discretizing the diffusion term and convection term explicitly and the pressure term implicitly in Eq. (8), the discrete Navier-Stokes equation is derived as follows:
\[
\frac{\mathbf{v}_f^{n+1} - \mathbf{v}_f^n}{\delta t} + (\mathbf{v}_f^n \cdot \nabla) \mathbf{v}_f^n + \frac{1}{\rho} \nabla p_{OC}^{n+1} - \nu \Delta \mathbf{v}_f^n = 0,
\]
(9)
where $\delta t$ is the time interval and $n$ is the time step. Rewriting Eq. (9) leads to

$$v_f^{n+1} = v_f^n - \delta t \left\{ (v_f^n \cdot \nabla) v_f^n + \frac{1}{\rho} \nabla p_{OC}^{n+1} - \nu \Delta v_f^n \right\}.$$  

(10)

Taking divergence of Eq. (10) leads to

$$\nabla \cdot v_f^{n+1} = \nabla \cdot v_f^n - \delta t \nabla \cdot \left\{ (v_f^n \cdot \nabla) v_f^n + \frac{1}{\rho} \nabla p_{OC}^{n+1} - \nu \Delta v_f^n \right\}.$$  

(11)

Following the continuity equation of fluid, $\nabla \cdot v_f^n = 0$. By contrast, to reduce numerical error, the first term of the right side $\nabla \cdot v_f^n$ is allowed to remain. As a consequence, Eq. (11) becomes

$$\Delta p_{OC}^{n+1} = \rho \left\{ \frac{1}{\delta t} \nabla \cdot v_f^n - \nabla \cdot \left\{ (v_f^n \cdot \nabla) v_f^n - \nu \Delta v_f^n \right\} \right\}.$$  

(12)

Substituting the known value of fluid velocity vector $v_f^n$ into Eq. (12), $p_{OC}^{n+1}$ can be obtained, and then $v_f^{n+1}$ is obtainable from Eq. (10).

In this study, as the structural model (OC) and fluid model (Lymph fluid) were constructed separately, the fluid-structure interaction between the model of the OC and that of the lymph fluid was considered by applying a staggered approach. As shown in Fig. 3.4, the procedure for coupling the fluid and structure equations is as follows: In time step $n = 1$, by multiplying the initial pressure $p_{\text{in}}$, with the area of the fluid-structure interface (F-S interface) of the OC model, the force vector $f^1$ over F-S interface of the OC model is obtained. Applying the force vector $f^1$ to Eq. (3), the displacement vector $u^1$ can be obtained, and then substituting it into Eq. (4), the velocity vector $\partial u^1/\partial t$ is obtainable. Assuming $\partial u^1/\partial t = v_f^0$ at the F-S interface, the fluid pressure $p_{OC}^1$ caused by the OC motion is obtainable by Eq. (12). In time step $n = 2$, assuming Couette flow in the sub-tectorial space, the shear stress vector $\tau^1$ exerted on the TM and the RL is given by

$$\tau^1 = \mu \frac{v_{\text{RELATIVE}}^1}{2h},$$  

(13)

where $v_{\text{RELATIVE}}^1$ is the relative velocity vector between the TM and RL, $h$ is the clearance.
between the TM and the RL and \( \mu \) is the viscosity of water \((1 \times 10^{-3}\text{ kg/m} \cdot \text{s})\). Multiplying this shear stress vector \( \tau^1 \) in the sub-tectorial space by areas of the TM and RL, and multiplying previously obtained fluid pressure \( p_{oc}^1 \) and the initial pressure \( p_{in}^2 \) in time step \( n = 2 \) by the area of the F-S interface, the force vector \( f^2 \) in time step \( n = 2 \) is obtained. This obtained force vector \( f^2 \) is applied to Eq. (3). By repeating the above procedure, the time history of the movement of the OC and that of the pressure distribution in each scala are obtained.

Numerical calculations were performed on a COMPAQ DS-20E using a 64-bit floating point number representation. Calculation was executed for 4 cycles for each sinusoidal frequency and took 2 hours.
Figure 3.1. Model of the organ of Corti discretized with finite elements. The number of nodes is 1274 and the number of elements is 2139. Each shade of gray in the model indicates the portion which has the same mechanical property. Scale bar represents 50 μm.
Figure 3.2. Models of the lymph fluid. (a) Scala vestibuli. (b) Scala tympani. Model dimensions are given in µm. Dark area in each model corresponds to the OC.
Figure 3.3. Model of the BM. This model is a reduced representation of the OC model shown in Fig. 3.1. Using this model, the stiffness of the BM is calculated to determine the Young's modulus of the BM. The arrow indicates the point where the force is applied and the stiffness is evaluated.
Figure 3.4. Scheme of the fluid-structure interaction using the staggered approach. In time step 1, initial pressure $P_{\text{INT}}^1$ caused by the fast and slow waves is applied to the equations of the OC (Eqs. (3) and (4)) and the velocity of the OC $\frac{\partial u_s}{\partial t}$ is obtained. Then, applying this velocity $\frac{\partial u_s}{\partial t}$ to the equation of the lymph fluid (Eq. (12)) as a fluid velocity $v_f^0$ over a fluid-structure interface, the fluid pressure $P_{\text{OC}}^1$ in each scala caused by the movement of the OC is obtained at the same time step. In step 2, this obtained fluid pressure $P_{\text{OC}}^1$, the initial pressure $P_{\text{INT}}^2$ in time step 2 and shear stress $\tau^1$ exerted on the TM and RL are applied to the equation of the OC. By repeating the above procedure, the time history of the movement of the OC and that of the pressure distribution in each scala are obtained.
Table 3.1. Geometric parameters of the OC model. BM, TM, IPC, OPC and RL are the abbreviations of basilar membrane, tectorial membrane, inner pillar cell, outer pillar cell and reticular lamina, respectively. RL/BM angle indicates an angle between RL and BM.

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>BM width</td>
<td>170 µm</td>
</tr>
<tr>
<td>BM maximum thickness</td>
<td>35 µm</td>
</tr>
<tr>
<td>TM width</td>
<td>120 µm</td>
</tr>
<tr>
<td>TM maximum thickness</td>
<td>35 µm</td>
</tr>
<tr>
<td>OPC height</td>
<td>70 µm</td>
</tr>
<tr>
<td>IPC height</td>
<td>55 µm</td>
</tr>
<tr>
<td>RL/BM angle</td>
<td>20 degrees</td>
</tr>
<tr>
<td></td>
<td>Young's modulus (N/m²)</td>
</tr>
<tr>
<td>--------------------------</td>
<td>------------------------</td>
</tr>
<tr>
<td>BM</td>
<td>$1.0 \times 10^7$</td>
</tr>
<tr>
<td>Deiters' cell</td>
<td>$1.0 \times 10^7$</td>
</tr>
<tr>
<td>Hensen's cell</td>
<td>$5.0 \times 10^3$</td>
</tr>
<tr>
<td>Inner hair cell</td>
<td>$1.0 \times 10^4$</td>
</tr>
<tr>
<td>Kimura's membrane</td>
<td>$1.0 \times 10^6$</td>
</tr>
<tr>
<td>Osseous spiral lamina</td>
<td>$2.0 \times 10^{10}$</td>
</tr>
<tr>
<td>OHC</td>
<td>$1.0 \times 10^4$</td>
</tr>
<tr>
<td>Phalanx</td>
<td>$1.0 \times 10^7$</td>
</tr>
<tr>
<td>Pillar cell</td>
<td>$1.0 \times 10^9$</td>
</tr>
<tr>
<td>RL</td>
<td>$1.0 \times 10^9$</td>
</tr>
<tr>
<td>Stereocilia</td>
<td>$1.0 \times 10^7$</td>
</tr>
<tr>
<td>TM</td>
<td>$6.0 \times 10^2$</td>
</tr>
</tbody>
</table>

The density and viscosity of the lymph fluid are $1.0 \times 10^3$ kg/m³ and $1.0 \times 10^{-3}$ Pa·s, respectively, which are equal to those of water.
4. Validation of the model: Static displacement

When a sinusoidal force of 2.0 Hz and $1.0 \times 10^{-6}$ N was applied to the bottom of the BM, the amplitude and angle of each point within the OC model was calculated. This stimulation was equivalent to that of the hemicochlea experiment (Hu et al., 1999), i.e., the application of oscillatory force via a glass paddle. Experimentally obtained trajectories of each measurement point (points in Fig. 4.1 (a)) and those of numerical results are shown by thin and thick lines in Fig. 4.1 (b), respectively.

A comparison between numerical and experimental results revealed that the numerically obtained amplitude and angle at the innermost OHC and outer pillar cell were nearly the same as those obtained by the experiment. However, angles of the outermost OHC top, IHC top, TM and middle OHC top differ from the experimental values by 16.7 degrees at the outermost OHC top, 13.2 degrees at the IHC top, 27.9 degrees at the TM and 15.1 degrees at the middle OHC top. These discrepancies would result because the angle between the RL and the BM of the model was different from that in the hemicochlea. From the above-mentioned comparison, it was confirmed that the mechanical properties of the model were appropriate.
Figure 4.1. Trajectories of cochlear structures. (a) Points of measurement when a sinusoidal force of 2 Hz and $1.0 \times 10^{-6}$ N is applied to the bottom of the basilar membrane (arrows), such force being similar to the experiment using the hemicochlea. (b) Trajectories of each point within the OC. The horizontal axis is parallel to the BM, and the vertical axis is perpendicular to the BM. Thin lines are the experimental results (Hu et al., 1999) and thick lines are the numerical results.
5. Prediction of the characteristics of the fast and slow waves

When a fluid pressure fluctuation is induced by vibration of the stapes, two types of pressure waves occur in the cochlea. Lighthill (1981) theoretically characterized these pressure waves. One is a fast wave which is uniformly distributed over cross-sections in both scalae and propagates at the velocity of sound. The other is a slow wave which follows a traveling wave on the BM. The slow wave exists in the vicinity of the BM and has equal magnitude with opposite phases at either side of the BM (Lighthill, 1981). Olson (1999) suggested the existence of two modes of pressure waves from her measurement of intracochlear pressure. As the OC is driven by these pressure waves, the frequency characteristics of them were predicted to simulate the dynamic behavior of the OC. The procedure for predicting the frequency characteristics of the fast and slow waves, which are initial pressures applied to the OC model, is as follows: First, the frequency characteristics of the fast wave in the SV (P_{fast-SV}) were estimated by an analytical method. Second, taking account of the experimentally obtained fluid pressure in the ST (Olson, 2001), the frequency characteristics of the fast wave in the ST (P_{fast-ST}) were estimated. Finally, by comparing these experimental data with the numerical results, the frequency characteristics of the slow wave (P_{slow}) were estimated.

When it is assumed that the cochlea is a tapered closed tube, i.e., the cross-sectional area of the cochlea becomes smaller towards the apex, the magnitude of the fast wave \( f(x) \) can be described as follows (Lighthill, 1981):

\[
f(x) = f(0)\frac{J_0[\omega c_0^{-1}(L-x)]}{[J_0(\omega c_0^{-1}L)]},
\]

where \( f(0) \) is the magnitude of the fast wave at the base, \( L \) is a length of the cochlea, \( x \) is the distance from the base, \( J_0 \) is the first zero of the Bessel function, \( \omega \) is the angular frequency and \( c_0 \) is the velocity of sound in water. In this study, the length of the cochlea \( L \) was set to be 11.1 mm (Müller, 1996). Figure 5.1 (a) is a schema of the gerbil cochlea indicating the
location for which the model was constructed, and Fig. 5.1 (b) shows the magnitude of \( f(x) \) relative to \( f(0) \) as a function of frequency in the gerbil cochlea at \( x = 3 \) mm. The magnitude of the fast wave increases with increasing frequency and quarter-wavelength resonance occurs at 48 kHz. The frequency characteristics of \( P_{\text{fast-SV}} \) were estimated as follows:

1. The fluid pressure in the vicinity of the stapes was estimated to be 105 dB SPL when the sound stimulus of 80 dB SPL was applied to the ear canal due to the gain of 25 dB in the middle ear. Therefore, it was estimated that the magnitude of \( P_{\text{fast-SV}} \) was 105 dB SPL at 1 kHz and increased gradually with increasing frequency and reached 112 dB SPL at 40 kHz in accordance with Eq. (14).

2. The phase difference of \( P_{\text{fast-SV}} \) relative to the pressure near the stapes is zero for the entire frequency range because the footplate of the stapes is connected to the basal end of the SV and the fast wave propagates at the velocity of sound.

Figure 5.2 (a) shows the experimental data on the fluid pressure in the ST in the basal turn in the gerbil cochlea when a pure tone of 80 dB SPL was applied to the ear canal (Olson, 2001). In that experiment, advancing and retracting the pressure sensor to and from the BM while keeping it perpendicular to the BM, the fluid pressures in the ST were measured at intervals of approximately 20 \( \mu \)m. The base point of the distance from the BM was determined by touching the BM with the sensor. Taking these experimental data into account, the frequency characteristics of \( P_{\text{fast-ST}} \) were estimated as follows:

3. In the experimental data, the difference of the magnitude of the fluid pressure between nearest-neighbor measuring points for a certain frequency becomes small with increasing distance from the BM, except for frequencies close to the characteristic frequency (CF) of 16 kHz. Because \( P_{\text{slow}} \) exists in the vicinity of the BM in contrast to \( P_{\text{fast-ST}} \) which is uniform in the ST, this behavior implies that the magnitude of \( P_{\text{slow}} \) does not have a significant effect on the pressure at points far from the BM, i.e., \( P_{\text{fast-ST}} \) is dominant. Therefore, from the experimental data at 120 \( \mu \)m from the BM, it was
estimated that the magnitude of $P_{\text{fast-ST}}$ is 90 dB SPL at 1 kHz, 93 dB SPL at 5 kHz, 102 dB SPL at 30 kHz and 111 dB SPL at 40 kHz. The magnitudes of $P_{\text{fast-ST}}$ between 5 kHz and 30 kHz were estimated using linear interpolation.

(4) The phase difference of $P_{\text{fast-ST}}$ relative to the pressure near the stapes was estimated based on the experimental data at 102 μm from the BM, i.e., 60 degrees below 10 kHz and 0 degrees above 16 kHz.

On the other hand, as $P_{\text{slow}}$ is regarded as being fluid pressure propagation caused by the fluid flux in the vicinity of the BM, the frequency responses of $P_{\text{slow}}$ are similar to those of the traveling wave on the BM. It is widely accepted that the magnitude of the traveling wave increases with increasing frequency up to the CF, and then decays sharply. Therefore, the frequency characteristics of $P_{\text{slow}}$ were assumed to be as follows:

(5) The phase delay of the traveling wave was assumed to originate at 1 kHz. And then, following the experimentally obtained phase difference of the fluid pressure at 7 μm from the BM, where $P_{\text{slow}}$ is dominant, it was estimated that the phase difference of $P_{\text{slow}}$ was -180 degrees at 18 kHz, resulting in destructive interference with $P_{\text{fast-ST}}$ which was observed as a pressure notch in the experimental data. Above 22 kHz, the phase difference of $P_{\text{slow}}$ was estimated to be -360 degrees. Therefore, the phase differences of $P_{\text{slow}}$ over the entire frequency range were estimated as shown in Fig. 5.3 (b) using cubic interpolation.

(6) Applying the magnitude and phase of $P_{\text{fast-SV}}$ and $P_{\text{fast-ST}}$ and the phase of $P_{\text{slow}}$ to the model, the magnitude of $P_{\text{slow}}$ was estimated so as to fit the pressure distribution in the ST obtained by our numerical analysis with that of the experimental data. At 1 kHz, it was estimated that the magnitude of $P_{\text{slow}}$ is the same value as that of $P_{\text{fast-SV}}$ because both of them are caused by vibration of the stapes. The magnitude of $P_{\text{slow}}$ then gradually increased with increasing frequency and reached a maximum of 125 dB SPL at the CF. The magnitude of $P_{\text{slow}}$ was followed by 122 dB SPL at 18 kHz and 110 dB SPL at 22 kHz. Above 22 kHz, the magnitude of $P_{\text{slow}}$ was determined to be 70 dB SPL,
a limit beyond which the magnitude of $P_{\text{slow}}$ does not have an effect on the pressure distribution in the ST.

Figure 5.2 (b) shows the numerically obtained pressure distribution in the ST when these predicted pressure waves, which are shown in Fig. 5.3 (a) and (b), were applied to the model. The pressure peak at 16 kHz, the pressure notch at 18 kHz, the pressure increase from 30 kHz and the phase of pressure relative to the pressure near the stapes were consistent with those of the experimental data. However, the depth and sharpness of the pressure notch at 18 kHz were different. The reason for this discrepancy might be the difference between the boundary condition in the model of the ST and the actual situation in the real cochlea at 120 μm from the BM. Consequently, it can be said that the magnitude and phase of the fast and slow waves were appropriately predicted for the most part.
Figure 5.1. Frequency response of the fast wave at a specific point in the gerbil cochlea. (a) Schema of the cochlea and the site where the model was constructed. The total length of the gerbil cochlea is 11.1 mm and the OC at 3 mm from the base (shaded area) is modeled. (b) Magnitude of the fast wave as a function of frequency which is derived from Eq. (14). Magnitude is relative to the sound pressure at the base.
Figure 5.2. Magnitude and phase of the fluid pressure in the ST versus frequency when a pure tone of 80 dB SPL was applied to the ear canal. The key indicates the distance from the BM. (a) Experimental data from the basal turn where the CF is about 16 kHz (Olson's figure 7. c and d, 2001). (b) Numerical results. Magnitude is relative to the stimulus level in the ear canal. Phase is relative to the pressure near the stapes.
Figure 5.3. Numerically obtained frequency characteristics of the slow wave, the fast waves in the SV and the ST when the magnitude and phase of the fluid pressure in the scala tympani are shown in Fig. 5.2 (b). The slow wave was applied to the F-S interface of the OC model and the fast waves were applied to the fluid models uniformly. (a) Magnitude. (b) Phase.
6. Velocity of the basilar membrane as a function of frequency

Using a laser interferometer microscope, Ren and Nuttall (2001) measured the velocity of the BM at the basal turn in the gerbil cochlea as a function of frequency. As the active force generation by the OHC was not included in the model, the post-mortem BM velocity in their study was compared with the numerically obtained velocity of the BM. The CF at the location of the measurement was approximately 10 kHz. The velocity of the BM was analyzed at the radial position of the second row of outer hair cells indicated in Fig. 6.1 (a), where the laser beam was focused on the BM in the experiment. Figure 6.1 (b) shows a comparison between the numerically obtained velocity of the BM and experimental one as a function of frequency. Although the frequency where the BM velocity has a maximum value is different between the CF of the model and that of the experiment, namely, 16 kHz and 10 kHz, respectively, the numerical result was similar to the experimental data.

Figure 6.2 shows velocity trajectories at each point on the OC, i.e., at the Claudius cell, Hensen cell, upper surface of the TM, IHC top and bottom of the TM. Velocities at all position reached a maximum at the frequency of 16 kHz. The angle of the trajectory relative to the BM was about 48 degrees at the IHC top and about 84 degrees at the bottom of the TM. Moreover, the trajectories at the IHC top and Hensen cell became ellipses with increasing frequency up to CF. It was concluded that the OC rotates around a point near the foot of the inner pillar cell. Moreover, the velocity trajectories of the point at Hensen cell became ellipse and opened wider with increasing frequency up to CF. This behavior corresponds to that of experiment (Hemmert et al., 2000).

As mentioned above, the numerical results corresponded to experimental data. Therefore, the model and the predicted pressure waves appear to be reliable.
Figure 6.1. Velocities of the basilar membrane when a pure tone of 80 dB SPL is applied to the ear canal. (a) Velocity measurement points on the BM. OHC2 is the abbreviation of the OHC of the second row. (b) Numerically obtained velocity of the BM and the experimental one (Ren and Nuttall, 2001) at the position which is indicated in (a). The CF of the model and that of the experiment are 16 kHz and 10 kHz, respectively.
Figure 6.2. Velocity trajectories at points on the OC for each stimulus frequency. (a) Measuring points. (b) Claudius cell.
Figure 6.2. Velocity trajectories at points on the OC for each stimulus frequency. (c) Hensen cell. (d) TM.
Figure 6.2. Velocity trajectories at points on the OC for each stimulus frequency. (e) IHC Top. (f) Bottom of TM.
7. Conclusions

A two-dimensional fluid-structure interaction micromechanical model of the OC was constructed using the FEM, and unknown mechanical properties of the model were validated with the experimental results of trajectories within the OC in the hemicochlea. By comparison between numerical results and those of intracochlear pressure measurement, the magnitude and phase of the fast and slow waves were predicted. The dynamic behavior of the OC was then simulated. Conclusions are as follows:

(1) When a pure tone of 80 dB SPL is applied to the ear canal, the magnitude of the fast wave in the SV, which is 105 dB SPL at 1 kHz, increases with increasing frequency and reaches 112 dB SPL at 40 kHz. The phase of the fast wave in the SV is 0 degrees for the entire frequency range. The magnitude of the fast wave in the ST also increases with increasing frequency, but the magnitude in the ST is lower than that in the SV, i.e., 90 dB SPL at 1 kHz and 111 dB SPL at 40 kHz. The phase of the fast wave in the ST is +60 degrees in the low frequency range and becomes 0 degrees above the CF. The magnitude of the slow wave is the same as that of the fast wave in the SV at 1 kHz (105 dB SPL), and increases gradually with increasing frequency until it reaches a maximum of 125 dB SPL at the CF. It then falls sharply to 70 dB SPL. The phase of the slow wave starts at 0 degrees and is significantly delayed near the CF and becomes -360 degrees above the CF.

(2) The OC shows a rotational movement around a point near the foot of the inner pillar cell.

(3) It was confirmed that the availability of the model to simulate the dynamic behavior of the OC.
Acknowledgements

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