Otosclerosis is a disease process of the ear that stiffens the stapes annular ligament and results in footplate immobilization. This produces a characteristic loss in bone-conducted (BC) hearing of about 20 dB between 1 and 2 kHz, known as "Carhart's notch", for which the specific mechanisms responsible have not yet been well understood. In this study, it is hypothesized that this observed pattern of hearing loss results from interactions between compressional and inertial mechanisms of BC hearing. Differences in the basilar-membrane velocity between a normal and otosclerotic human ear were calculated in response to compressional vibration of the cochlear capsule, translational vibration of the skull bone in various directions, and combinations of the two, using an anatomically accurate 3-D finite element model of the middle ear, cochlea, and semicircular canals. Compressional and inertial BC stimuli were found to both be necessary to capture the full behavior of clinical data, with the compressional component dominating below 0.75 kHz, the inertial component dominating above 3 kHz, and the notch between 1 and 2 kHz resulting from the suppression of an ossicular resonance due to stapes fixation. [Work supported by grant R01-DC07910 and R01-DC05960 from the NIDCD of NIH.]
Introduction

Otosclerosis is a disease that stiffens the stapes annular ligament, resulting in air-conducted (AC) hearing impairment. In addition, it produces a bone-conducted (BC) hearing impairment of approximately 20 dB near 2 kHz, which is called Carhart’s notch (Carhart, 1950). While Carhart attributed this phenomenon to “mechanical factors associated with stapedial fixation” (Carhart, 1971), Tonndorf (1971) suggested that the Carhart’s notch at 2 kHz is caused by the loss of the middle-ear inertia due to the otosclerotic condition close to the resonance point of the ossicular chain. According to Stenfelt and Goode (2005), five factors potentially affect BC hearing, with inertia of the cochlear fluid and middle-ear mass, as well as bone compression among the major factors. We hypothesized that Carhart’s notch is affected by bone compression as well as by the inertia of the cochlear fluid and middle-ear mass. In this study, we investigated the effects on BC hearing of each factor separately, inertia and bone compression, in addition to the effects of the two factors in combination.

Methods

Using a 3-D finite element (FE) model (Kim et al., 2013; Figure 1), otosclerosis was simulated by stiffening the stapes annular ligament. Two different input stimuli were applied to the FE model, consisting of inertial stimulation of the middle-ear ossicles and the cochlear fluid by shaking the whole structure, and bone-compressional stimulation by applying a dynamic pressure to the outer bony surface of the cochlea. Concerning the inertial input, our aim was to test the existence of Carhart’s notch under the otosclerotic condition, regardless of the direction of the shaking stimulus. Therefore, two different stimulus directions were defined to be normal to the basilar-membrane (BM) surface, at distances of 0.8 mm and 5.8 mm from the base of the cochlea, respectively. These directions are indicated as \( d_1 \) and \( d_2 \) throughout this study. Next, the calculated hearing-loss results for the FE model, under the otosclerotic condition and for the different BC-excitation methods, were shown and compared with clinical data (Carhart, 1950).
Results

*BC excitation by inertia alone and by bone compression alone*

While the clinical data shows a 20 dB hearing loss localized at 2 kHz, the FE BC results due to inertial excitation show 20–30 dB of hearing loss all the way from 0.25 to 2 kHz (Figure 2A). Due to this discrepancy between the FE results and clinical data, we additionally need to consider another form of BC input, that is, bone compression. The effects of bone-compressional input on BC hearing were investigated and the results are shown in Figure 2(B). The FE results show no hearing loss at any of the simulated frequencies (0.25–10 kHz).

![Figure 2](image)

**FIGURE 2.** Hearing loss (defined as the difference of the maximum BM velocity between the normal condition and the otosclerotic condition) for the two different BC-excitation methods, which are (A) only inertia inputs, to the middle-ear ossicles and the cochlear fluids; and (B) only bone-compressional input, represented by applying dynamic pressure on the outer bony surface of the cochlea. The two different shaking directions for the inertial inputs are indicated by $d_1$ and $d_2$. In Figure 2(B), distinction between $d_1$ and $d_2$ is meaningful since only bone-compressional input is applied.

*BC excitation by both inertia and bone compression*

While the hearing loss was observed at low frequencies (below 0.5 kHz) in Figure 2(A), this hearing loss was somewhat decreased by applying both inertial and bone-compressional inputs, as shown in Figure 3(A). The combinational BC input caused about 8–25 dB of hearing loss for the $d_1$-directional vibration case, and 5–15 dB of hearing loss for the $d_2$-directional vibration case.

![Figure 3](image)

**FIGURE 3.** Hearing loss when both the inertial and bone-compressional stimuli are applied. In (A), the pressure ($P$) representing the bone compression is maintained at a constant magnitude of 1 Pa regardless of the input frequency; whereas in (B) the
pressure representing the bone compression is made to depend on the input frequency, according to Eq. (2). The value of \( k \) in Eq. (2) is a tuning factor used to modify the effects of bone thickness in the FE model, and was set 0.005 to produce these results.

The relationship between pressure and displacement for a simplified plane wave

While the magnitude of the pressure reaching the outer bony surface of the cochlea is supposed to depend on the input frequency, and an actual pressure wave traveling through the skull is significantly complex, we simplify the pressure wave in the model by defining it as a plane wave assuming that the wave length is significantly larger than the length of cochlea, according to the following relationship:

\[
P = iωd × ρc = 2πfid\sqrt{Eρ},
\]

where \( P \) is the pressure on the outer bony shell of the cochlea; \( i \) is the square root of -1; \( ω \) is the angular velocity; \( d \) is the bone displacement due to shaking the whole FE structure, which is assumed to be 0.1 nm in this simulation; \( E \) is the bone elasticity, assumed to be 14.1 GPa; \( ρ \) is the bone density, assumed to be 2200 kg/m\(^3\); \( f \) is the input frequency; and \( c \) is the speed of sound in bone, which is equal to the square root of \( E \) over \( ρ \). When we substitute the above values for each variable, \( P \) has the following relationship to the input frequency \( f \):

\[
P = 0.349\\pi f × k,
\]

where \( k \) is a tuning factor due to the relative amplitudes of the inertial and compressional inputs, which is unknown at this time.

Figure 3(B) shows the hearing loss of the FE simulation, with \( k = 0.005 \). When the frequency dependence of the bone compression is included, the simulation results indicate that there is little (about 0–7 dB) hearing loss below 0.5 kHz, and significant hearing loss (about 7–20 dB), between 1 and 2 kHz, which signifies greater consistency with the clinical data.

Conclusions

Based on the simulation results, we conclude that the BC hearing pathway is mainly governed by compressional input to the inner ear at low frequencies (below 0.5 kHz), whereas at high frequencies (above 1 kHz) it is mainly governed by inertial input to the middle ear and cochlear fluid. With the pathologically otosclerostic condition, the consequent alteration and mixing of the inertial and compressional BC inputs leads to the characteristic decrease in the BC threshold near 2 kHz known as Carhart’s notch.

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