4pPP1. Hearing impaired cochlear response simulation

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A model is introduced which allows the vibration of the basilar membrane to be estimated for different degrees of hearing loss, using a discrete lumped parameter implementation of the human cochlea. The hearing losses are assumed to be caused by the combined malfunction of the outer hair cells (OHCs), the inner hair cells (IHCs) and the endocochlear potential driving the system. OHC loss and damage to endochoclear potential are modelled by a reduction of the cochlear amplifier gain, which is obtained by reducing the feedback gain of the OHCs. IHC loss is modelled as an overall reduction in measured response. The distribution of OHC and IHC loss along the cochlea are derived using an iterative method, which matches the output vibration amplitude of the model to that assumed to generate the hearing impaired audiogram.

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INTRODUCTION

It is important to build models of the cochlea, because of the difficulties in directly measuring its active characteristics in vivo, particularly when it is hearing-impaired. Active linear models of the cochlea such as the ones presented by Allen[1], Kanis and de Boer[2] or Neely and Kim[3], allow the response of the cochlea to be predicted for an input of a given frequency, and hence the frequency response of the cochlea at a given position along the basilar membrane (BM) length can be obtained. These models account for the longitudinal coupling of the fluid chamber, as well as the micromechanical behaviour of individual parts of the cochlear partition. Such models are typically constructed using a set of discrete elements, whose mechanical properties vary continuously along its length. The response of these models is then obtained by solving a set of difference equations for one frequency at a time. By varying the mechanical and active characteristics of these models, the magnitude of the responses can be modified, and hence allow to reproduce different degrees of hearing impairment.

There are models that account for the reduced loudness summation in people with cochlear hearing loss[4, 5], but no previous work has been done, as far as we are aware, from the point of view of a discrete elements cochlear model. Nowadays the process of hearing aid adjustment is based on the characteristics of a hearing impaired persons audiogram, which is not considered to be a very accurate approach, since the same audiogram can be obtained due to hearing loss (HL) of different natures[6]. A model that reproduces the origins of the hearing deficit may therefore, for example, be of use in the specification of a hearing aid.

Several forms of HL can occur inside the cochlea, these are mainly caused by damage to the outer hair cells (OHC) or the inner hair cells (IHC)[7], although it is also believed to occur due to the loss of endocochlear potentials, which affects the operation of both OHC and IHC[6]. It is assumed here that a normal cochlea presents an active amplification that goes from 45 dB at the base to 20 dB at the apex, as reported by Robles and Ruggero[8], hence hearing loss up this figure are assumed to be caused by a reduction in the performance of the OHC. Greater losses are then assumed to be generated by a loss of function in the IHCs as well.

COCHLEAR MODELS

A discrete element model of the cochlea can be divided into two main domains: the fluid coupling which accounts for the inertia of the fluid inside the two cochlear chambers, and the micromechanics which accounts for dynamic behaviour of the BM. Although the purpose of this paper is not to explain the whole operation of these models, a brief description is included here for background.

The fluid coupling due to 1D pressure variation was initially calculated by Neely[9], using a finite difference formulation, but a more recent analytic formulation derived by Elliott et al.[10] is used here. The fluid coupling matrix, $Z_{FC}$, is thus defined, so that the vector of pressures at $N$ points along the cochlea, can be expressed as a function of the BM velocity at these points, $v$, in the matrix form

$$\mathbf{p} = Z_{FC} \mathbf{v}$$

(1)

The micromechanics used here are these of the Neely and Kim 1986 model[3]. This micromechanical model, which is shown in Fig. 1, is a two degree of freedom system, in which one mass represents the BM and the other mass represents the tectorial membrane (TM). The action of the cochlear amplifier is introduced by a feedback loop proportional to the relative displacement of BM and TM. The admittance of a single micromechanical element, reproducing the cochlear partition at a given position, is given by

$$Y_p(\omega) = Z_1(\omega) + Z_2(\omega) \left( \frac{Z_3(\omega) - \alpha Z_4(\omega)}{Z_2(\omega) + Z_3(\omega)} \right),$$

(2)
where

\[ Z_1(\omega) = \frac{k_1}{j\omega} + C_1 + j\omega M_1, \quad Z_2(\omega) = \frac{k_2}{j\omega} + C_2 + j\omega M_2, \]  \hspace{1cm} (3)

\[ Z_3(\omega) = \frac{k_3}{j\omega} + C_3, \quad Z_4(\omega) = \frac{k_4}{j\omega} + C_4, \]  \hspace{1cm} (4)

where \( \gamma \) represents the gain of the feedback loop used to model the cochlear amplifier. The values for each of the elements of the micromechanical systems, which are the same values as used by Ku et al. [11] are shown in Table. 1. These parameters are used here because they replicate better the response of a human cochlea, instead of the original parameters given by Neely and Kim, which were established to simulate a cat cochlea. The vector of BM velocities in response to the pressures at each position can then be written as

\[ \mathbf{v} = \mathbf{v}_S - \mathbf{Y}_{BM} \mathbf{p}, \]  \hspace{1cm} (5)

where \( \mathbf{v}_S \) is the vector of velocities due to the stapes and \( \mathbf{Y}_{BM} \) is a diagonal matrix with elements given by Eq. 2. The response of the coupled cochlea is then obtained by combining the matrix description of fluid coupling, and BM dynamics, to give

\[ \mathbf{v} = (\mathbf{I} + \mathbf{Y}_{BM} \mathbf{Z}_{FC})^{-1} \mathbf{v}_s, \]  \hspace{1cm} (6)

The stability of the coupled system can be determined using a state space formulation of the same model[12]. The maximum active enhancement obtained with the model using these
parameters is shown in Fig. 2. This has been calculated by running the model when it is entirely passive, so $\gamma = 0$, and fully active, so $\gamma = 1$, and taking the difference in the BM responses at the characteristic place over a number of excitation frequencies.

The distribution of the feedback gains in the micromechanical elements along the cochlea are adjusted in the next section to reproduce the reduction in sensitivity due to hearing impairment. There is not a one-to-one correspondence between the reduction in feedback gain at a particular point in the cochlea and the reduction in sensitivity at this characteristic frequency, so that a global optimisation must be used.

**SIMULATION OF HEARING LOSS**

**Obtaining the cochlear amplifier gains**

The hearing impaired cochlear responses presented here are the outcome of an iterative program, which finds the active gain distribution along the cochlear length that minimises the error between the BM calculated with a coupled model with a given set of active gains and a target velocity distribution of the cochlear partition, assumed for the hearing impaired.

The distribution of HL over frequency given by the standard ISO 7029, *Statistical distributions of thresholds as a function of time* [13], have been used to define the target loss of sensitivity. This standard gives an estimation of the HL assumed to occur in different percentages of the population due to ageing. The values of HL estimated to occur for 50 percent of the female population are replicated here, as shown in Fig. 3.

Fig. 2 shows that the maximum active gain obtained with the model varies between around 20 dB at low frequency, to around 40 dB at high frequency, which is consistent with measurements [8]. The maximum HL that can be attributed to OHC loss, however, are limited by the model's active gain. It is assumed here that the HL at 125 Hz, for each age, are due to IHC malfunction, and that this is constant all along the BM. The HL due to OHC are thus assumed to be that shown in the left hand side plot of Fig. 3 minus the amount at 125 Hz.

Whilst that the HL due to the OHC are clearly reflected in a reduction of the BM velocity, this is not the case for the HL due to IHC damage, where it is the measured response that is reduced [7]. It is possible however to model the malfunctioning of the IHC by plotting the neural tuning curves with an elevated threshold along all the frequency band.

The envelopes of the BM velocity assumed to occur only to OHC malfunction are shown in
FIGURE 3: Median HL expected in 50% of females at different ages, as given by the standard ISO 7029 (left) and the corresponding BM velocity envelope distributions calculated from the envelope for the active cochlea, on the left hand side of Fig. 2 (20 years), minus the HL assumed due to the OHCs at different ages (right).

The right hand side plot of Fig. 3. This velocities have been obtained by choosing the output of the fully active model to be that of a 20 years old cochlea, $v_{20}$. The velocities which correspond to other degrees of hearing impairment are reduced according to the dB reduction of the HL that correspond to a certain age

$$v_{HL} = v_{20}10^{-HL/20}. \quad (7)$$

The obtained distribution of velocities is obtained up to 8 kHz, which corresponds to the highest frequency presented in the standard ISO 7029[13].

**Hearing Impaired Dynamics**

A first attempt at reproducing these losses was made by adjusting the feedback gain, $\gamma$, to match the HL at each frequency, and assigning this gain value to the point of the cochlea with a characteristic frequency equal to that of the excitation. When the coupled response of the cochlea was calculated again, however, the model was found to be unstable. It was found that a smooth decrease on the cochlear amplifier gains between adjacent elements is needed in order to assure stability. This lead to the use of a polynomial to shape the amplifier gains along the length of the cochlea. Fig, 4 shows the reduction in BM velocity due to the reduction of cochlear amplifier gains needed to obtain an envelope of velocities which is close to Fig. 3.

The gains for the active amplifier have been obtained by fitting a $4^{th}$ polynomial, optimizing its coefficients until the envelope of the responses showed a reasonable agreement with that of Fig. 3, leaving mean errors of 0.2, 0.6 and 0.7 dB for 50, 60 and 70 year old respectively. Other trials have been performed with other polynomials of lower and higher order, but the most reasonable results have been obtained with a $4^{th}$ order one. The results are based on median HL up to only 8 kHz, but the gain curves continue to decrease towards more basal locations, as shown in Fig. 4. This is consistent with measurements in other mammal species cochleas, where it has been seen that the degradation of OHC for noise induced mice grows towards the base[14].

Neural tuning curves can be calculated from this model, with different degrees of HL. These are shown in Fig. 5 at a distance 8.5 mm from the base, which corresponds to a characteristic frequency of approximately 5.5 kHz. The hearing threshold is increased constantly at all frequencies, due to IHC malfunction, and by a large amount at the characteristic frequency due to the OHC gain reduction, which corresponds to a reduction of about 30 dB. It is also observed how the characteristic frequency of this point on the BM is somehow reduced due to HL, as observed in physical measurements[8].
**FIGURE 4:** The solid lines show the BM velocity responses against frequency calculated to approximate a 50 year old cochlea (top left), a 60 year old cochlea (top right) and a 70 year old cochlea (bottom left), compared to the responses of a 20 years old cochlea (dashed lines). The dotted line represent the envelope of the response, as in the right plot of Fig. 3. The optimised distributions of cochlear amplifier gains, used to obtain these results, are plotted as a function of position along the cochlea at the bottom right graph of the figure.

**FIGURE 5:** Neural tuning curves associated with the HL given by the standard ISO 7029 at a position 8.5 mm from the base.

**Frequency selectivity**

As well as the sensitivity of the BM being reduced by HL, it is known that the frequency selectivity is also reduced. This can be quantified by calculating the sharpness of the equivalent filters, generated for various degrees of HL, for example.

The tuning of auditory filters is normally expressed in terms of equivalent rectangular bandwidth (ERB), also often called the critical bandwidth[15], which corresponds to the
bandwidth of a perfect bandpass filter which has the same white noise power as the specified filters. The results presented here are plotted as the quality factor using this ERB metric, \( Q_{ERB} \), which is defined as

\[
Q_{ERB} = \frac{f_C}{ERB(f_C)}
\]

where \( f_C \) represents the characteristic frequency, i.e., the frequency at which a point of the BM is more sensitive.

Fig. 6 shows the estimates of \( Q_{ERB} \) that are obtained for various degrees of HL. The values corresponding to a BM of 20 year old are comparable with the estimates based on otoacoustic emissions by Shera et al.\[16, 17\]. The results of the model match very closely the measurements of Shera et al. between 6 and 10 kHz, but show a lower \( Q_{ERB} \) below 6 kHz.

The loss of frequency selectivity due to the increase in bandwidth, and hence reduction in \( Q \), is clearly seen in Fig. 6, for the model corresponding to the HL of Fig. 3, and primarily affects the high frequencies.

**CONCLUSIONS**

A model has been presented that can be adjusted to reproduce the reduction in sensitivity due to age related hearing losses. This is achieved by calculating distributions of cochlear amplifier gain, reproducing OHC loss, and reducing the overall sensitivity, to represent loss of IHC function, that best match the frequency dependence of the hearing loss at various ages.

The model can also be used to quantify the selectivity of hearing, in terms of the equivalent rectangular bandwidth quality factor, \( Q_{ERB} \). The estimates of \( Q_{ERB} \) match previous estimates for a healthy cochlea reasonably well above 6 kHz. The predictions with a hearing-impaired cochlea, corresponding to an age of 70, show how the frequency selectivity is approximately halved at higher frequencies, in comparison to the normal case.

Although further development of the model is required, to more accurately model the micromechanics, and better estimates of the active enhancement in the normal cochlea could be used, the methods used to reproduce the hearing loss would still be similar to these outlined here. An advantage of such a model is that the effect of hearing loss on other aspects of cochlear behaviour, such as otoacoustic emissions[\[18, 11\]], or the two tone suppression that is involved in masking, could also be predicted. It may be that such studies would provide methods of distinguishing between different sources of hearing loss, and hence the prescription of more effective hearing aids for individual patients.
REFERENCES


