Chapter 2

Physiological correlates of hearing impairment

2.1 Introduction

People suffering from a hearing loss of cochlear origin often show different performance than normal in measures of loudness perception, intensity discrimination, frequency selectivity, temporal resolution and speech perception (Moore, 1995; Florentine et al., 1993; Holube, 1993; Glasberg and Moore, 1989; Festen and Plomp, 1983; Moore, 1983) even when compared at the same sensation level (SL) with normal subjects. Usually, large intersubject variability is seen in the results of different psychoacoustic experiments (for instance the slope of the loudness function or auditory–filter bandwidth) even for subjects with a similar spectral characteristic (sloping or flat) and amount of hearing loss, and same etiology. This is usually not the case for subjects with a conductive hearing loss, where psychoacoustic performance is hardly altered when the comparison is made at the same SL. There are three specific perceptual changes associated with cochlear hearing loss which are important for the scope of this study. Firstly, hearing–impaired subjects usually show reduced sensitivity in detecting pure tones in quiet, i.e., they have raised absolute thresholds. A second change, which is quite often used for diagnostic purposes, concerns the perception of loudness. In patients suffering from a cochlear hearing loss, the rate of growth of loudness with increasing stimulus level is usually steeper than in normal–hearing subjects; the same range of subjective loudness as normal is achieved over a smaller range of stimulus levels than normal. This effect, known as recruitment (Fowler, 1936), is usually not seen in subjects suffering from a purely conductive hearing loss. Several different forms of recruitment have been reported in the literature (Brunt, 1994), including complete recruitment (where loudness reaches its "normal" value at high sound levels), over–recruitment (where loudness exceeds the "normal" value at high sound levels) and partial recruitment (where loudness never reaches normal values). The third change is that hearing–impaired subjects usually show reduced frequency selectivity, i.e., broadened auditory filters (Moore, 1995; Hétu and Tran-Quoc, 1995; Laroche et al., 1992; Glasberg and Moore, 1986; Tyler, 1986). These three

changes seldom occur separately and might thus reflect damage of a common underlying mechanism. In this chapter the physiological correlates underlying these alterations will be discussed.

2.2 Physiology of hearing impairment

Loudness recruitment is solely observed in subjects suffering from a sensorineural hearing loss. This is due to the way acoustic signals are processed by the auditory system. The peripheral auditory system transforms and encodes the mechanical energy of a sound, e.g., speech, into electrical pulse trains. This neural pulse train is conveyed to the brain, i.e., the auditory cortex, via the auditory nerve and different higher stages of the auditory pathway. Basically, three different stages contribute to peripheral processing. The first stage consists of outer and middle ear. The outer ear mainly acts as a spatial filter gathering and attenuating sounds from different directions. From the outer ear, the sound is transmitted through the ear canal to the middle ear consiting of the tympanic membrane and the ossicular chain. The ossicular chain consists of three very small bones (hammer, anvil and stirrup) the last of which (stirrup or stapes) is connected to the oval window and thus to the fluid–filled inner ear, i.e., the cochlea. Overall, the outer and middle ear act as pure linear attenuators and therefore damage to these organs solely causes a loss of sensitivity but no performance changes in psychoacoustic tasks like, e.g., recruitment.
In the second stage of auditory signal processing, in the inner ear or cochlea, the mechanical energy of the sound wave is converted to neural spike trains. The mechanical vibration of the tympanic membrane and the ossicular chain is transformed to a motion of the liquid in the cochlea via the vibration of the oval window. This liquid motion drives the basilar membrane (BM) on which a complex–structured receptor organ is situated, the Organ of Corti. This membrane performs a decomposition of the signal into its spectral components (similar to a Fourier transformation). The mechanical displacement of BM is converted by the Organ of Corti via displacement of the receptor cells (inner and outer hair cells) into neural spike trains on the auditory nerve. Thus, a very basic view of auditory signal processing in the cochlea is as a bank of linear bandpass filters each with a certain bandwidth ("critical bandwidth"). Further processing is carried out independently in each filter. It is assumed that outer hair cells contribute in a still unresolved way to the mechanics of this organ, while inner hair cells act as pure passive receptors. Unlike the outer and middle ear, the cochlea does not solely act as a simple attenuator but represents the first stage of auditory signal processing. Therefore, damage to this organ has serious consequences for auditory signal processing and thus causes strong alterations in psychoacoustic performance, e.g. recruitment.
The third stage of auditory signal processing consists of the cochlear nerve and different auditory nuclei which convey the neural spike trains to the auditory cortex. In these nuclei, further signal processing, like binaural processing or extraction of sound envelop, is perfor-

med. However, the exact performance changes following damage to these organs are still unresolved.
The physiological basis and causes of cochlear injuries have been extensively studied (see (Ruggero et al., 1995; Ruggero, 1992a; Saunders et al., 1991; Pickles, 1988), for reviews). Cochlear hearing loss can result from a great variety of different causes, including noise exposure, ototoxic drugs (salicylates, aminoglycocides, diuretics), chemical solvents (Franks and Morata, 1995), and autoimmune diseases (Zenner, 1993a; Zenner, 1993b). Surprisingly, they mainly seem to produce initial damage at the same site in the Organ of Corti, affecting the transduction processes in outer and inner hair cells (Ruggero et al., 1995; Patuzzi, 1992; Saunders et al., 1991; Pickles, 1988), although the exact morphological changes in the Organ of Corti can differ. The results of these injuries are fourfold: loss of sensitivity (elevation of auditory threshold); loss of sharp tuning, i.e., the typical tip–tail characteristic is altered (broadened auditory filters); loss of compressive nonlinearity in the BM input–output functions; and a reduction of other nonlinearities such as two–tone suppression, generation of cubic difference tones and otoacoustic emissions. These alterations might be caused by a common underlying mechanism, thus being intimately linked to each other and not appearing separately.

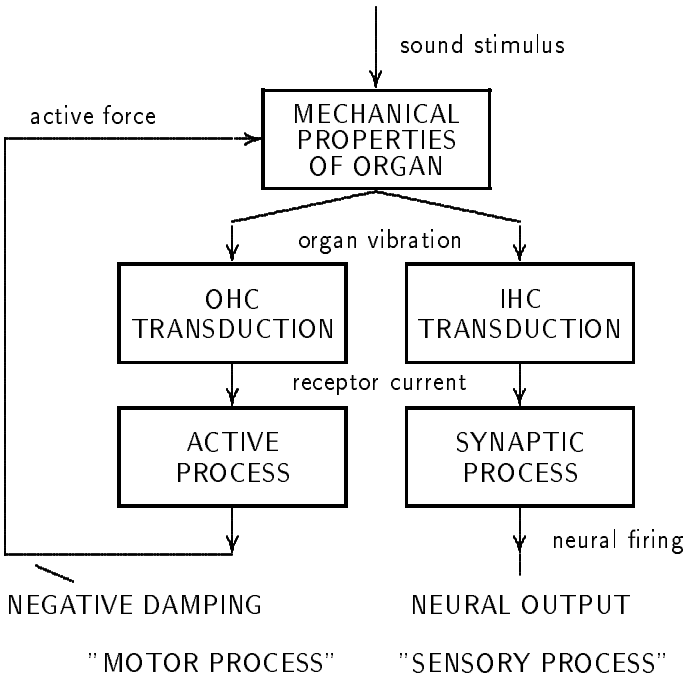

Fig. 2.1: *Schematic model of Organ of Corti function. OHCs are thought to provide some mechanical support to basilar membrane vibration while the IHCs are thought to be pure sensory receptors which encode auditory information.*

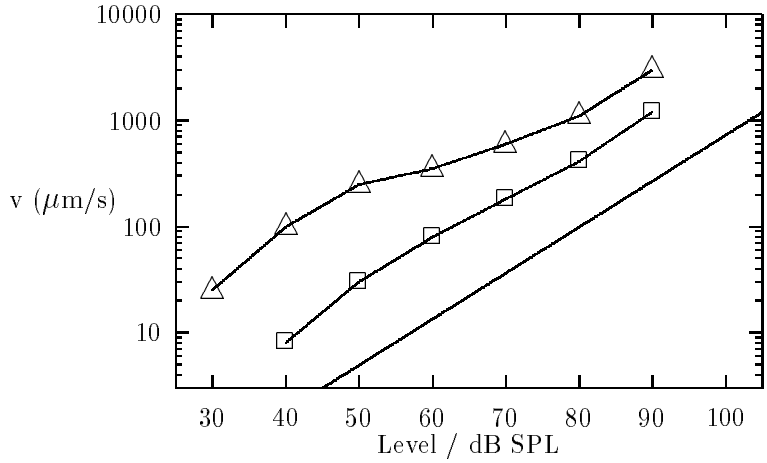

Fig. 2.2: *Input/output function of normal (△) and abnormal (□) basilar membrane. Basilar membrane velocity (μ m/s) is plotted versus stimulus intensity (dB SPL). The solid line exemplifies a linear input–output function. The main consequence of cochlear damage is a linearization of BM input–output function. Adapted from Ruggero and Rich (1991).*

The micromechanics of Organ of Corti/BM vibration are still not completely understood (Allen and Neely, 1992; Dallos, 1992; de Boer, 1991). A schematic model of Organ of Corti function is shown in Fig. 2.1. However, it seems clear that inner hair cells (IHC) are responsible for "sensory processes", i.e., the encoding of auditory information, while the outer hair cells (OHC) strongly influence BM vibration thus representing motor or active processes. These motor processes are interpreted as being the cause of the nonlinearity of the BM transfer function, yielding high sensitivity of auditory neurons around their characteristic frequencies (CF), the sharp tuning seen in auditory nerve tuning curves (tip–tail shape) (Patuzzi and Robertson, 1988) and the frequency–selective nonlinearities of the auditory nerve (Ruggero, 1992b). The main consequence of damage to OHCs is a linearization of BM responses (cf. Fig. 2.2) causing the loss of sensitivity, loss of sharp frequency tuning (cf. Fig. 2.3) and loss of the auditory nonlinearities mentioned above (Ruggero et al., 1995). The loss of OHCs and the consequences of this for BM response, have quite often been discussed. However, damage only to IHCs has seldom been observed. Liberman and Dodds (1984) described a few cases in which the majority of OHCs were apparently normal and only IHCs were damaged. Their data indicate that the shapes of neural tuning curves seem to be hardly altered (cf. Fig. 2.3), but thresholds for all frequencies are raised, suggesting that the motor processes are still present. Similar findings were presented by Siegel and Relkin (1987) although they did not damage the stereociliae of inner hair cells but blocked the synaptic transduction between inner hair cells and neurons by perfusion of a magnesium–rich salt into the scala tympani. Blocking synaptic transduction probably causes a shift of threshold, since a higher stimulus level is needed to achieve a given amount of transmitter release. Damage of stereociliae, however, might yield complete

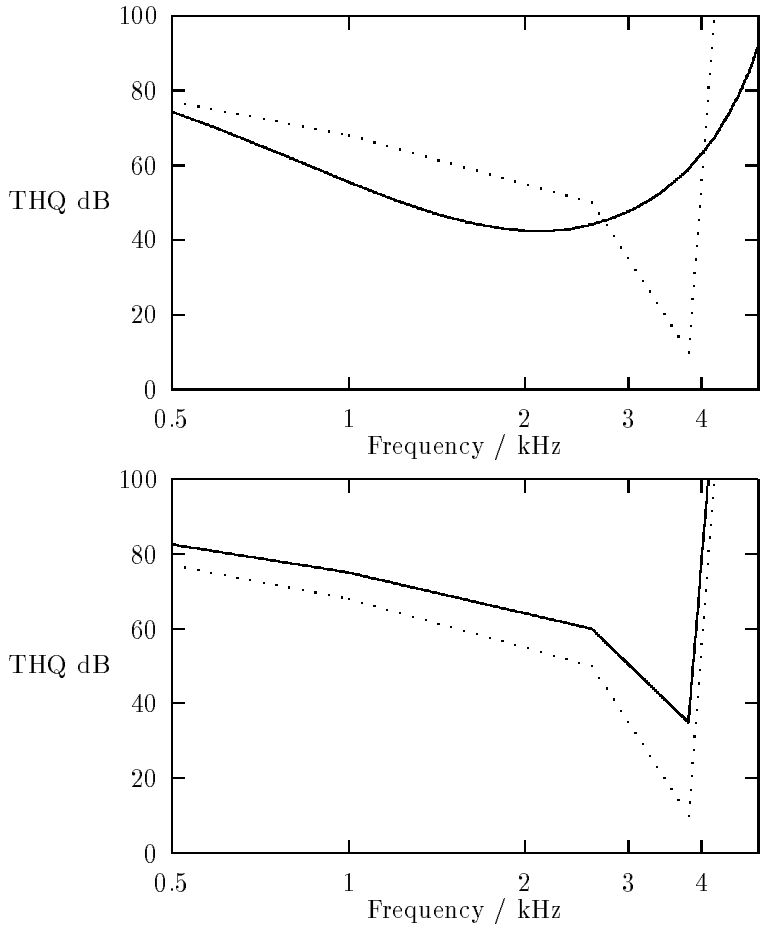

Fig. 2.3: *Different damage pattens of OHC and IHC in the Organ of Corti and their influence on neural tuning curves. Threshold of nerve fibers (THQ) is plotted versus frequency. The dotted line represents the normal and the solid line the abnormal tuning curves. Upper panel: Damage to OHCs only. The neural tuning curves are raised in threshold and significantly broadened. Lower panel: Damage to IHCs only. The neural tuning curves are not broadened but only raised in threshold. Schematic drawing adapted from experimental results obtained by Liberman and Dodds (1984).*

deafness (no excitation of neural spikes by IHCs), while blocking the synaptic transduction only causes a threshold shift.

These findings (lack of nonlinear processes when outer hair cell are damaged, "motor losses"; only threshold shift when inner hair cells are damaged, "sensory losses") have been the basis for some speculations about the consequences of different types of hearing loss for psychoacoustical performance (Patuzzi, 1993b; Patuzzi, 1993a). Patuzzi speculates that for pure "sensory" losses the resulting changes might be comparable to changes usually seen in a conductive hearing loss. Thus, threshold shifts should occur in these cases but no marked performance changes should be observed in psychoacoustic tasks, i.e., there should

be no recruitment, no broadening of the auditory filters (except to the extent that normal filters broaden with increasing level) and little alterations in speech perception. For pure OHC loss, however, psychoacoustical performance should be strongly altered, i.e., there should be recruitment, broadened auditory filters and reduced speech perception. Thus different damage pattens in the Organ of Corti (different amount of OHC and IHC loss) could result in different performance in psychoacoustic tasks although producing the same threshold shift in the audiogram. According to that assumption a large amount of OHC loss and only few IHC loss might, e.g., result in a steep loudness function, while only small amount of OHC loss and large amount of IHC loss could result in weakly steepened loudness function. Different damage pattens could thus account for part of the variability seen in psychoacoustic experiments with impaired subjects with similar audiometric threshold shifts and similar etiology.
Recently, Takeno *et al.* (1994) described some experiments in which they used a drug (carboplatin) damaging only inner hair cells (which is species specific). Experiments using this drug could help to further clarify the role of IHCs and OHCs in the mechnics of the Organ of Corti. Furthermore it would shed new light on whether some of the variability usually seen in psychoacoustic data obtained from hearing–impaired subjects could be explained by different damage patterns in the Organ of Corti.

2.3 Conclusions

In summary, it is likely that two components contribute to the alterations in cochlear processing of sounds due to cochlear damage: Firstly, an "active" component which is due to damage of OHCs yielding a less compressive basilar membrane input–output function ("compression loss"). This component would cause strong alterations of psychoacoustic performance, e.g., loudness recruitment. The second component is assumed to be a pure passive component, similar to a conductive hearing loss. Both, loss of OHC and loss of IHC contribute to this component ("sensitivity loss"). It is assumed that this component only causes weak alterations in psychoacoustic performance, thus no or only weak loudness recruitment should occur. These physiological findings have some important implications for the model describing loudness perception in impaired listeners proposed in chapter 7.