The relationship between auditory efferent function and frequency selectivity in Man

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Abstract

The auditory efferent system consists of two populations of fibres. The lateral system connects with the inner hair cells and the medial system connects with the outer hair cells. Past work has suggested that the efferent system may act via the action of the outer hair cells to control cochlear mechanics and, in doing so, contribute to the high degree of frequency selectivity observed in the auditory system. This hypothesis has not, in the past, been fully examined in humans.

In this study, normally hearing human subjects were examined to see if there was a link between efferent activity and frequency selectivity. The first experiment investigated whether there was a link between efferent action and frequency selectivity at 1kHz. Efferent activity was assessed using the contralateral suppression of transient and distortion product otoacoustic emissions (OAEs). Emissions were evoked by tones and clicks and suppressed by contralateral white noise, narrow band noise and tones. The suppression was examined in specific frequency bands as well as over the entire response range. Frequency selectivity was estimated using the notched-noise masking technique and 'roex' filter shape modelling. No conclusive relationship was found.

The possibility that the efferent system might play a part in frequency selectivity only when activated by the contralateral ear was considered by testing the auditory filter during contralateral white noise. No consistent relationship was found between efferent function, and either the 1kHz filter shape measured during contralateral stimulation, or the change in filter shape. However, contralateral white noise did cause a significant broadening of the 1kHz filter via what was thought to be the action of the efferent system.

The timing of the onset of the contralateral efferent effect was examined using OAEs in order to allow stimulus timings to be set so that the efferent system would be effective during filter shape testing. The onset latency was found to be between 17 and 20ms.

Further analysis of the auditory filter shape during contralateral stimulation was undertaken by stimulating the contralateral ear with narrowband noises at 500Hz, 1kHz and 2kHz. This also provided no clear proof of a link with efferent activity.

Finally, the filter shape at 2kHz was considered. Significant relationships emerged between the shape of the filter and the activity of the efferent system in this region. This implied that efferent control of frequency selectivity is not constant over the length of the basilar membrane.

Overall, the effect of the efferent system on frequency selectivity in normally hearing humans was not found to be straightforward, and was found to vary with frequency. However, the results were consistent with the theory that the medial portion of the efferent system acts in such a manner as to dampen the motion of the basilar membrane.
Chapter 1

Introduction
1. Introduction

The auditory efferent system consists of an extensive network of neural fibres that extend from the brainstem to the organ of Corti in the cochlea. The importance of these pathways lies in the fact that responses to auditory signals can be modified before they reach the brain. Although the efferent system has been the subject of numerous studies, its exact function is still not clear. It seems unlikely that such a comprehensive system of fibres would be redundant. Physiological and behavioural evidence suggests that its role may be to protect the ear against damage from loud sounds (Rajan 1992) or to aid in localising (Fisch 1970) or attending to sounds (Giard et al. 1994). It has also been suggested that it may help in detection of sounds in background noise. It is this latter role that is to be investigated in this thesis.

The human ear exhibits a high degree of frequency selectivity. In other words, it is able to pick out particular frequencies from a signal successfully. Energy input is required in order to enable the system to behave in this manner. The outer hair cells in the cochlea seem to be involved in these active processes. The efferent system makes connections to the outer hair cells. Thus, the hypothesis behind this thesis is that the efferent system may control, or at least be involved in, frequency selectivity via the action of the outer hair cells.

Past work has found that efferent activity is implicated in discrimination of signals in noise, both in animals (Kawase et al. 1993; Winslow and Sachs 1988) and in humans (Micheyl and Collet 1996). The tuning of the auditory system in animals can be measured directly, at the level of the nerve or basilar membrane. In humans, the task is less straightforward, although it is possible to get an estimation of the shape of the auditory filter which is representative of the frequency selectivity present in the system. There have however, been no studies where the frequency selectivity has been examined in this manner and related to the functioning of the auditory efferent system in humans.

The objective therefore of this thesis is to explore the possible role of the auditory efferent system in frequency selectivity in normal hearing humans. Measurements which assessed the level of activity of the efferent system were compared to measurements of frequency selectivity in the same subjects. Detailed analysis of the results from both
types of tests was carried out to investigate the possibility of a link that could point to an involvement of the efferent system in frequency selectivity.
Chapter 2

Background Theory
2. Background Theory

2.1 The Anatomy and Physiology of the Human Ear

In this section, the anatomy and physiology of the ear are discussed, with the emphasis towards those aspects that are relevant to frequency selectivity.

The structure of the ear can be divided into 3 major sections: the outer, middle and inner ear. Figure 2-1 shows this basic structure.

Figure 2-1: The structure of the ear in humans (from Pickles 1988)

2.1.1 Outer and Middle Ear

The outer ear consists of the pinna, narrowing to form the concha and external auditory meatus. The structure of the resonant cavity thus formed, causes a relative increase in sound pressure at the tympanic membrane for frequencies between 2 and 7 kHz. The
shape of the pinna also provides clues as to the direction of the sound source. Spectral modulation occurs due to interference of components of the acoustic wave reflected from the walls of the pinna. This modulation varies with position of the source and thus provides localising clues.

The sound wave travels along the external auditory meatus to the tympanic membrane. From here the sound is transmitted to the cochlea via movement of the ossicular chain which is composed of three bones: the malleus, incus and stapes, which are linked together. The stapes is attached to the oval window of the cochlea. The middle ears acts as an impedance coupler between the low impedance of the air in the external auditory meatus and the higher impedance of the fluids in the cochlea, preventing large transmission losses due to reflection.

Different frequencies are transmitted by the middle ear with different efficiencies. The transfer function can be measured by comparing the pressure at the tympanic membrane to that at the cochlear duct, behind the oval window (Nedzelnitsky 1980, see Figure 2-2).

**Figure 2-2: Transfer function of the middle ear (Nedzelnitsky, 1980)**

![Transfer Function Graph](image)

The maximum gain is around 1kHz and away from this frequency the gain drops off, giving a bandpass type of frequency response. Transmission is reduced above 1kHz due to the ossicular mass and the change of vibration pattern of the tympanic membrane into many modes. Resonances in the middle ear cavity also cause some losses. At frequencies below 1kHz, there is reduced transmission due to elastic stiffness of the middle ear structures, and compression and expansion of the air in the middle ear. This
has a greater effect at lower frequencies because the displacement of the air is greater and therefore the forces are also greater.

The **middle ear muscles**, the stapedius muscle and the tensor tympani muscle, also have some control over the amount of energy transmitted through the middle ear. The stapedius muscle is connected to the neck of the stapes and acts to stiffen the ossicular chain and the tympanic membrane. It is innervated by the facial (VIIth) cranial nerve. The tensor tympani muscle is attached to the malleus and contraction again causes stiffening of the ossicular chain and the tympanic membrane. It is innervated by the trigeminal (Vth) cranial nerve. Activation of the middle ear muscles affects primarily the transmission of frequencies below 1-2kHz. The reflex is activated by loud sounds (>75dBSL), vocalisation, general movement, tactile stimulation or voluntarily in some cases.

There are a number of hypothesised functions of the reflex: 1) protection of the inner ear against loud sounds; 2) stabilisation of cochlea input at low frequencies for intensities of up to 20dB above reflex threshold; 3) reduction in the masking of high frequency sounds by those of low frequency, helping in the perception of complex stimuli; 4) reduction in the effect of middle ear resonances on the frequency response of the middle ear.

Therefore, in summary, the only relevant effect that the middle ear has on the frequency selectivity of the auditory system is to perform an overall bandpass transformation of the frequency response spectrum.
2.1.2 Cochlea

2.1.2.1 Structure

The cross-sectional structure of the inner ear or cochlea is shown in Figure 2-3.

Figure 2-3: Cross-section of the cochlea (from Pickles 1988)

The scala vestibuli and the scala tympani are filled with perilymph and are connected at the apex of the cochlea at the helicotrema (see Figure 2-4).

Figure 2-4: Schematic diagram of the cochlear duct (from Pickles 1988)

The inner compartment of the cochlea is called the scala media. It is filled with endolymph, which has a high $K^+$ and low $Na^+$ concentration. The resting potential of the
endolymph is highly positive (+80mV) whereas the perilymph in the other two compartments is at 5-7mV relative to the plasma. The positive potential of the endolymph is thought to arise from a Na⁺/K⁺ ATPase ion pump in the stria vascularis. The perilymph is very similar to normal extracellular fluid.

The scala vestibuli opens onto the oval window, which is connected to the stapes footplate. Thus, movement of the stapes produces displacement of the oval window and therefore the fluid in the scala vestibuli. The movement of fluid is transmitted via the scala tympani to the round window. This flow of fluid causes a displacement in the basilar membrane. The structure of the organ of Corti showing the basilar membrane is shown in Figure 2-5.

**Figure 2-5: Cross-section of the organ of Corti (from Pickles 1988)**

Two types of receptor cell are found in the organ of Corti; the inner and outer hair cells. There are 3,500 inner hair cells in one row and 12,000 outer hair cells in 3-5 rows. The hair cells have many fine stereocilia attached. The longest of the outer hair cell stereocilia are connected to the tectorial membrane, which is attached only at one end. The hairs of the inner hair cells are fitted loosely into a groove called Henson's stripe on the lower surface of the tectorial membrane. Thus, as the basilar membrane is deflected relative to the tectorial membrane there is a shearing force applied to the stereocilia.

In response to this movement, K⁺ ion channels are opened and closed allowing release of K⁺ from the stereocilia into the cell body. The resting potential of the inner hair cells is -45mV and of the outer hair cells is -70mV. In the inner hair cells, depolarisation due
to the K⁺ flow, triggers the release of transmitter from the base of the hair cell, instigating an action potential in the afferent auditory nerve fibres which end there. The inner hair cells’ function therefore seems to be the conversion of acoustic mechanical stimuli into electrical impulses, which are then transmitted to the cortex. The function of outer hair cells is not yet certain.

The afferent and efferent nerve fibres make connections at the base of the hair cells and provide a link to the higher centres of the brain. These pathways are described in section 2.1.3.

2.1.2.2 Transient Cochlear and Neural Potentials

Three different cochlear potentials can be recorded at the round window or across the cochlear partition, in response to stimuli:

(a) The cochlear microphonic

The cochlear microphonic (CM) is believed to arise from the current flow mainly through the outer hair cells, in response to acoustic stimulus. This a.c. response approximately follows the stimulus.

(b) The summating potential

A d.c. shift in the average potential can be measured for the duration of the stimulus which is called the summating potential (SM). The shift can be positive or negative in direction and is thought to be derived from the d.c. flow from both the outer and inner hair cells.

(c) The compound action potential

The compound action potential (CAP) is recorded at the onset of a stimulus. It is the massed synchronised contributions of the action potentials of the fibres activated by the stimulus. The first wave of the response can be measured after approximately 1ms and is called N₁, and the second wave (N₂) can be measured at about 2ms.
2.1.2.3 Frequency Selectivity in the Cochlea

The term *frequency selectivity* refers to the ability to resolve sinusoidal components from a complex signal. The high degree of frequency selectivity present in the auditory system is essential in order to distinguish different sounds successfully. The human ear is capable of resolving two frequencies greater than 10% of the centre frequency away from each other. However, when tones are presented successively we are able to detect much smaller differences in frequency (as low as 0.2-0.3% at 1kHz). This is referred to as *frequency discrimination*.

(a) Basilar Membrane Mechanics

Incoming acoustic signals are transferred to the inner ear, as a hydromechanical wave, where they are coded according to frequency for nerve transmission. Studies, using the Mössbauer effect, of the motion of the basilar membrane (BM) during travelling wave displacement, have shown that each portion of the BM peaks in displacement at a particular frequency (Sellick et al. 1982). Frequencies just outside a certain band are many times less likely to have this effect and thus a highly tuned bandpass filter is indicated.

This variation of resonant frequency with position along the cochlear partition is brought about by the changes in stiffness and inertia. At the base of the cochlea the system is predominantly stiffness limited and at the apex, it is mostly mass limited. The travelling wave moves from the base towards the apex and grows in amplitude because the stiffness limited system moves first. Resonance occurs when the stiffness limitation equals the mass limitation. High frequencies resonate at the base because the inertial forces are higher. The wave slows down approaching the point of resonance and therefore damping occurs effectively, thus reducing the amplitude of the wave beyond this point.

The most accurate mathematical models describing the tuning of the cochlea seem to involve active mechanisms (e.g. Neely and Kim 1983). The addition of mechanical energy to the travelling wave by an active process would increase the amplitude of vibration of the basilar membrane at the characteristic frequency. Although the basilar membrane provides some passive tuning, this does not account for all the tuning effects.
seen. For example, Sellick et al. (1982) have shown that after death, the cochlea is only broadly tuned (see Figure 2-6).

**Figure 2-6: Tuning curves from guinea pig cochlea in various conditions**

Thus, when alive, both the sensitive and the broadly tuned characteristics are effective. The active mechanism suggested would act as an amplifier, increasing the energy of the travelling wave and thus sharply tuning the response. It would also seem likely that the production of otoacoustic emissions (see section 2.2) is linked with this active process. The active mechanism is discussed further in section (d).

(b) Tuning of hair cells

The responses of both outer and inner hair cells to different frequencies are shown in Figure 2-7. The form of the tuning curve, for both types of hair cell, is very similar to that measured from the motion of the basilar membrane. The lower side of the tuning curve tails off gradually and the upper side has a steep gradient. The tip of the curve at the characteristic frequency has a low threshold.
(c) Tuning of auditory nerves

The frequency selectivity of the auditory nerve shows similar characteristics to that of the basilar membrane and the hair cells. Individual fibres are sharply tuned to a particular characteristic frequency. The tuning curves become increasingly asymmetric at higher frequencies. As with the hair cell and basilar membrane tuning curves, in the asymmetric cases, the high frequency tail of the curve is very steep whereas the lower tail gradually slopes off. Increase in stimulus intensity causes an increase in the firing rate of the fibres, which follows a sigmoidal relationship. For lower frequency tones (below about 5kHz) there is also phase locking to the response. The neural coding of pitch seems to depend on both temporal and spectral features i.e. both the patterns of firing of the neurons and the distribution of firing across different neurons play a part.

(d) Active Processes and Outer Hair Cells

The presence of an active process seems essential to explain the high degree of frequency selectivity of the auditory system. Although there are still uncertainties regarding the workings of this mechanism, it seems clear that it is very vulnerable to physiological change. Figure 2-6 showed that tuning deteriorated in the post mortum cochlea suggesting that a source of energy is required. Experiments involving the use of kanamycin, an ototoxic antibiotic which selectively damages outer but not inner hair
cells, have indicated that the outer hair cells play a crucial role in the maintenance of normal tuning. Work by Robertson and Johnstone (1979), using this technique, showed that the auditory nerve tuning curves lost sensitivity and sharpness, and shifted to a lower frequency, when the outer hair cells were lost (Figure 2-8).

Figure 2-8: Change in tuning of the auditory nerve fibre with kanamycin administration, which acts on the outer hair cells. From Robertson and Johnstone (1979), adapted by Pickles (1988)

![Graph showing the change in tuning of the auditory nerve fibre with kanamycin administration.](image)

Electrical stimulation of the crossed olivocochlear bundle (COCB) in guinea pigs and cats was also found to degrade the tuning curves of inner hair cells (Brown et al. 1983) and of the auditory nerve (Guinan and Gifford 1988b). The COCB predominantly makes connections with the outer hair cells, and therefore this evidence again indicates that the latter are important in frequency selectivity.

The motility of outer hair cells in response to stimulation has been demonstrated (Ashmore 1987), adding weight to the theory that they may be part of a feedback mechanism with the basilar membrane. The ability of the cochlea to actually produce sound, or otoacoustic emissions, as mentioned earlier, is also a by-product of the presence of an active mechanism.
Russell and Nilsen (1997) estimated the location of the amplifier of cochlear mechanics in guinea pigs to be a 1.25nm portion of the basilar membrane when measuring non-linear, saturating vibrations at 15kHz. It has been suggested though, that this mechanism may not contribute evenly to peripheral tuning along the cochlear partition. Human auditory filters, at close centre frequencies were measured using the roex(ρ,ρ) method (see section 3.1) and were found to be very different from each other (Fagelson and Champlin 1997).

Therefore, in summary, it is clear that the shape of the tuning characteristics of the hair cells and the auditory nerve are very similar to that of the basilar membrane. It is likely that the overall frequency selectivity of the auditory system is determined at the level of the basilar membrane. Current evidence suggests that the fine-tuning observed is due to an actively driven feedback loop, at the level of the basilar membrane, which is accomplished via the motile properties of outer hair cells.

2.1.3 Auditory Neural Pathways

The afferent pathway relays the electrical impulses from the cochlea to the brain. Each ear has about 30,000 fibres which connect mostly with the inner hair cells (type I fibres). One hair cell is innervated by many fibres. Only 5-10% of fibres (type II) connect with outer hair cells (see Figure 2-9).

Figure 2-9: Connections of hair cells to afferent nerve fibres. OHC, Outer hair cells; IHC, Inner hair cells; SG, Spiral ganglion. (From Spoendlin (1978), Fig.8)
The efferent nerve fibres carry nerve impulses descending to the cochlea. This means that higher centres are able to have some control over the cochlea and therefore the incoming information. The efferent fibres make connections with the outer and inner hair cells. Each outer hair cell has many efferent nerve connectors, with those at the base having more than those at the apex. The projections to the inner hair cells synapse with the afferent fibres rather than directly with the hair cell itself. The fibres terminating at the outer hair cells tend to have large endings, which cover the base of the hair cell as well as the afferent terminals.

2.1.3.1 The Afferent Pathways

The pathway that the ascending afferent nerves take from cochlea to brainstem is shown schematically in Figure 2-10.
Figure 2-10: Pathway of the afferent auditory pathways. Cross section through the junctional zone between the pons and the medulla. From Noback and Demarest (1981)
2.1.3.2 The Efferent Pathways

The route of the descending efferent pathways is shown schematically in Figure 2-11. The efferent system will be discussed in greater detail in section 2.3.

Figure 2-11: The efferent auditory pathways. From Noback and Demarest (1981)
2.2 Otoacoustic Emissions

2.2.1 Existence of otoacoustic emissions

The discovery that the cochlea actually emits sound as well as receiving it has revolutionised research into hearing. Gold had suggested in 1948 that a feedback mechanism, consisting of both a mechanical-to-electrical and an electrical-to-mechanical stage, was the source of the high degree of frequency selectivity exhibited in the cochlea. However, it was not until Kemp (1978) demonstrated that energy emitted by the human cochlea could be recorded in the ear canal, that there was more interest in Gold’s hypothesis. These sounds are called otoacoustic emissions (OAEs). The argument that active processes control the mechanical state of the cochlea, gathered further weight with the discovery that outer hair cells change shape as a voltage is applied (Brownell et al. 1985). In addition, they seemed to be the source of the reverse transduction process (electrical-to-mechanical energy) which could affect motion of the basilar membrane (Mountain and Hubbard 1989).

The exact mechanisms, which lead to the production of OAEs from active processes within the cochlea, are still to be elucidated. However, OAEs are proving to be a useful tool in analysing the details of cochlear mechanics. The measurement of OAEs has the advantage of being a non-invasive objective procedure that can easily be applied to humans as well as animal models. The test has the benefit of examining only the sensory elements of the hearing pathway, without confounding the results with information about the neural pathways.

OAEs are very sensitive to physiological changes in the cochlea. Therefore, even mild pathological conditions can abolish OAEs entirely. Many problems with hearing, such as those due to noise exposure, may involve damage to the actively assisted vibration of the basilar membrane and therefore OAEs provide a valuable insight into the cochlea in these cases. Thus, the normal and the abnormal cochlea can be studied using OAEs in order to further our understanding of the exact mechanisms involved in the hearing process (for review see Probst et al. 1991).

There are four types of OAEs, which can be classified by whether an external stimulus is required to evoke them and if so, what the nature of that stimulus is. Only the first three types of OAE discussed were used in this study.
2.2.1.1 Transient otoacoustic emissions (TOAE)

These emissions are elicited by a brief stimulus, usually a click. The response emitted by the ear is then recorded, averaged and frequency analysed using a fast Fourier transform (FFT) procedure.

It appears that all normally hearing adults would exhibit measurable TOAEs if tested in ideal conditions (Probst et al. 1991). However, with hearing losses greater than 25-30dBHL the TEOAE is unrecordable, with the most important frequency region being 1-2kHz for TOAE generation (see Probst et al. 1991 for review). The latency of the emissions is related to their frequency components. Lower frequencies are emitted later than higher ones, with the frequencies around 1kHz being emitted after about 10ms (e.g. Kemp and Chum 1980). The growth of emission amplitude with stimulus level is approximately linear until stimulus levels of greater than about 20-30 dBSL, at which point, the response saturates (e.g. Kemp 1978).

2.2.1.2 Spontaneous otoacoustic emissions

Spontaneous otoacoustic emissions (SOAEs) are acoustic signals generated by the cochlea without any stimulus. They are narrow band emissions and can be found in approximately a third of normally hearing ears (see Probst et al. 1991 for review). SOAEs are most commonly found in the vicinity of 1-2kHz, although this may be influenced by the transfer function of the middle ear, which is best in this frequency range (Kemp 1980). SOAEs can be measured directly with no external stimulus. However, in practice it is more convenient, since the response is more robust, to measure the SOAEs emitted after a click stimulus. In this situation the SOAEs are synchronised to the stimulus and are therefore often called synchronised spontaneous otoacoustic emissions (SSOAE). SOAEs appear to be very stable in frequency with time, and therefore it would seem likely that non-uniformities of some type at distinct points of the organ of Corti may lead to their generation.

2.2.1.3 Distortion product otoacoustic emissions

Distortion product otoacoustic emissions (DPOAE) are produced as a result of non-linear processes acting on the incoming acoustic signal to create signals of different frequencies. DPOAEs were first noted by Kemp (1979) in human ears. If the stimulus
consists of two tones, \( f_1 \) and \( f_2 \) (primary frequencies), where \( f_1 < f_2 \), combination tones are produced. The most common DPOAE to study because it is usually the strongest, is that at a frequency of \( 2f_1 - f_2 \), the cubic distortion tone.

DPOAEs have advantages when studying the details of cochlear mechanics in that the place that the response originates is a localised area of the cochlea. There may be more than one source of DPOAEs on the basilar membrane (Whitehead et al. 1992). Much of the DPOAE energy is thought to come from the region around the travelling wave peaks of the stimuli (\( f_1 \) and \( f_2 \)) and probably more predominantly from the region of \( f_2 \) (Brown and Kemp 1984). It is thought that the source may be in the region of maximum overlap of the travelling wave envelopes of the 2 primary tones, which would place it near to \( f_2 \). A second source may be from the DP site (Brown et al. 1996). The exact mechanism of DP production is not defined. However, it is certain that the situation is complex, and the interference (constructively or destructively) of the overall emission with emissions from any part of the cochlea, will affect the outgoing signal. There are therefore many influences on the emitted response.

### 2.2.1.4 Stimulus-frequency otoacoustic emissions

Stimulus frequency otoacoustic emissions (SFOAEs) can be generated by presentation of a low level tonal stimulus. The response is a steady state emission of additional energy at the same frequency as the stimulus. There has been much less research work on this class of emissions in comparison with other types, especially in pathological ears (for review see Probst et al. 1991). For this reason, SFOAEs were not tested in this study and therefore are not discussed in detail.

### 2.2.2 How OAEs have been used to study the auditory efferent system

It has been found that the magnitude of TOAE responses can be reduced by the simultaneous presentation of contralateral acoustic stimuli (Collet et al. 1990). This effect was assumed to take place via the efferent fibre connections to the opposite ear since it could occur at levels below the acoustic reflex threshold. There are many other experiments that have studied this effect and they will be discussed in section 2.3.2.2.
2.3 The Auditory Efferent System

2.3.1 Detailed Anatomy

As early as 1893 (Held), it was known that efferent neural pathways relayed information from the brain to the cochlea. However, the anatomy of the pathway was described in greater detail by Rasmussen (1946). The efferent input to the cochlea consists of the olivocochlear bundle, which passes from the superior olivary complex structures on both sides of the brain. The contralateral fibres (crossed olivocochlear bundle, COCB) cross over the dorsal surface of the brainstem, below the floor of the fourth ventricle. The crossed and the uncrossed (ipsilateral) fibres then join together. Before leaving the brainstem, some fibres end in the cochlear nuclei and the rest travel along the vestibular nerve until they transfer into the auditory nerve and enter the cochlea (Figure 2-12).

Figure 2-12: Schematic diagram showing the course of the auditory efferent system (broken line) to the cochlea, and the afferent system (solid line).  

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1 Abbreviations used are: LOCS- lateral olivocochlear system, MOCS- medial olivocochlear system, CN- cochlear nucleus, SOC- superior olivary complex, TB- trapezoid body, IC- inferior colliculus, SM- stapedius muscle, VIIMN- seventh nerve motor nucleus
By using transport techniques, involving radioactively labelled amino acids, two populations of olivocochlear neurons have been found (Guinan et al. 1983):

i) The lateral olivocochlear neurons (LOC) (54% of the total number of olivocochlear fibres) are mainly (approximately 90%) ipsilateral and synapse with cochlear afferent neuron dendrites close to the inner hair cells. They are thin, unmyelinated and originate from the lateral superior olivary complex.

ii) The medial olivocochlear neurons (MOC) (40% of the total population) project mainly contralaterally (80%) from the region around the medial nuclei of the superior olivary complex and terminate beneath the outer hair cells of the organ of Corti. These are large, myelinated fibres.

Most efferent units were found, in the cat, to have binaural inputs (Liberman 1988).

This structure suggests that it may be possible for the efferent system to exert control over both the hair cell and the transmission of the impulse to the afferent nerve. Moreover, it would seem likely that these two very distinct populations of fibres would have different roles to play in the hearing process.

2.3.2 Physiology

Studies to evaluate the function of this extensive pathway of fibres have used electrical and acoustic stimuli to activate the efferent nerves in both animal and human subjects. This review of the physiology of the efferent system classifies current knowledge according to the effects that efferent activation has on different levels of the auditory pathway. Firstly, the effects to the afferent responses and cochlear potentials will be discussed, followed by efferent induced otoacoustic emission changes.

2.3.2.1 Effects of efferent activation on afferent responses and cochlear potentials

Early experiments (Desmedt 1962; Galambos 1956) established that the crossed olivocochlear bundle (COCB) has an inhibitory effect on the eighth nerve action potential. Electrical stimulation of the floor of the fourth ventricle caused a suppression in the action potential (N1), as did selective stimulation of the MOC fibres (Gifford and Guinan 1987). These findings have also been replicated using acoustic stimulation in cats (Buno 1978) and in humans (Folsom and Owsley 1987; Prasher and Gibson 1984).
Kawase and Liberman (1993) found both a decrease and an *increase* in cochlear action potential (CAP) for tones masked with ipsilateral noise. The effect depended on frequency. Enhancement of CAP in the cat was largest for tones from 8 to 16 kHz and suppression was largest for 2 to 8 kHz. Kawase et al. (1993) showed that discharge rates to a masked tone could be increased whilst the rates to the masker decreased, by the addition of contralateral sound, indicating a role in signal discrimination.

Some other cochlear potentials are also affected by activation of the efferent system. An increase in the cochlear microphonic (CM) was observed under electrical stimulation in animals (Fex 1962; Gifford and Guinan 1987; Mountain 1980) and a drop in endocochlear potential (EP) was also found (Fex 1967; Gifford and Guinan 1987). The CM is thought to be produced predominantly by the outer hair cells and the stria vascularis is the presumed generator of the EP.

The discharge rate of auditory nerve fibres during efferent stimulation was found to be reduced, (see part A, Figure 2-14) and this effect was greatest for fibres responding to their own characteristic frequency, during electrical stimulation (Wiederhold 1970) and during contralateral acoustical stimulation (Warren and Liberman 1989b), demonstrating the frequency specificity of the effect. The tonotopic organisation of the efferent projections is also evidenced by the work of Liberman and Brown (1986) who found that the fibres are tuned to the region that they innervate. The decline in the suppression away from the characteristic frequency leads to a widening of the fibre's frequency tuning curve and therefore a reduction in the "Q" value (Guinan and Gifford 1988b). Fibres with low spontaneous rates (SR) were also found to show the greatest reduction in discharge rate (Guinan and Gifford 1988b). Stimulation of the MOC was found to reduce the SR activity mostly around 10kHz and to suppress responses to sounds more at lower characteristic frequencies (CF) in cats (Guinan and Gifford 1988a). The former result was thought to be due to the fact that the greatest innervation of outer hair cells by medial efferent fibres is at this frequency. The latter finding was attributed to the efferents acting on the outer hair cells to damp the motion of the basilar membrane and this effect being transmitted apically. By recording potentials from electrodes at the round window and in the skull of guinea pigs in the presence of no ipsilateral stimulation, the 'ensemble background activity' was found to be reduced during contralateral white noise stimulation (Da-Costa et al. 1997).
Earlier studies found that the effect that the efferent system had on auditory nerve responses was maximal at low ipsilateral sound levels (Desmedt 1962; Galambos 1956; Wiederhold 1970). More recently however, measurements of single afferent fibre responses to varying levels of sound stimulation showed that the largest MOC evoked suppression was at moderate to high sound levels (Guinan and Stankovic 1996). The difference between this and the earlier results was thought to be due to the fact that in the earlier studies the CAP was being measured, and this would have been dominated by responses from high spontaneous rate fibres and from fibres responding to sounds not at their CF. As mentioned earlier, both these conditions are not optimal for measuring efferent effects since low spontaneous rate fibres show greatest suppression (Guinan and Gifford 1988b) as do those fibres responding to their CF (Warren and Liberman 1989b; Wiederhold 1970). However, the results of Guinan and Stankovic (1996) have also been shown for basilar membrane motion in guinea pigs, measured using a laser interferometer (Russell and Murugasu 1997).

The decrease in output from the afferent fibres seems to be accounted for by the reduction in the mechanical responses of the inner hair cells (Brown and Nuttall 1984). However, the increase in attenuation at higher levels discussed above, does not support the hypothesis that changes in BM motion are solely responsible for the effect. Nevertheless, as mentioned, it has been found that OCB activation does cause damping of the motion of the basilar membrane (Russell and Murugasu 1997). This was also demonstrated by measurement of the cochlear microphonic potential under contralateral acoustic stimulation in the mustached bat (Henson et al. 1995). However, the slow contraction of the outer hair cells produced by stimulation of the efferent system was only found to give small deflections of the stereocilia, equivalent to a transverse movement of the organ of Corti of less than 1.5nm (Patuzzi and Rajan 1990). It is possible therefore that the changes in BM motion may partly explain the effects seen, but also another, as yet unfound, mechanism may be involved.

By sectioning of the pathway at different levels in animals, some workers have tried to deduce the effects of the olivocochlear bundle (OCB) on hearing. A section in the floor of the fourth ventricle ipsilateral to the ear to be studied, at the vestibular nerve or at the internal auditory meatus (see B in Figure 2-13), can eliminate the entire OCB (both crossed and uncrossed components), whereas a midline section (see A in Figure 2-13)
will cut only the crossed component. The latter will remove most of the MOC whilst sparing most of the LOC.

**Figure 2-13:** Schematic diagram of a transverse section through the brainstem at the level of the fourth ventricle showing the positioning of: (A) a section at the midline cutting the crossed OCB and, (B) a section off midline where both the crossed and uncrossed OCB are cut. LSO- lateral superior olive, MSO- medial superior olive.

Sectioning of the entire OCB was shown to give threshold (Bonfils et al. 1986a) and tuning curve changes (Bonfils et al. 1986a; Carlier and Pujol 1982) and to abolish CAP suppression during contralateral acoustic stimulation (Liberman 1989). However, Liberman (1990) found no change in threshold, tuning curves or rate-level functions in cats, after complete OCB sectioning at the floor of the fourth ventricle, and Littman et al. (1992) found no change in guinea pig tuning curves after entire OCB section. The variation in results obtained could be due to inter-species differences in physiology and anatomy or from the variety of experimental methods employed, some perhaps causing damage to the OCB. However, many papers indicate that the uncrossed component of the OCB, rather than the crossed component, may be the source of the changes seen, via
the action of the outer hair cells (Bonfils et al. 1986a; Bonfils et al. 1986b; Kawase and Liberman 1993; Warren and Liberman 1989a).

To summarise, when activated either electrically or acoustically the olivocochlear bundle has the effect of reducing or sometimes increasing the activity in the afferent nerves. The effect seems to be frequency specific and thus the tuning of the cochlea can be altered.

2.3.2.2 Effects of efferent activation on otoacoustic emissions

Although the above studies have shown that OCB activation can alter the responses of the auditory nerve fibres to sound, the precise mechanisms subserving these effects are not yet clear. However, information from the work on otoacoustic emissions provides evidence that the OCB can affect the active processes involved in cochlear mechanics.

As discussed in section 2.2, otoacoustic emissions are thought to be produced by the active processes occurring in the cochlea, probably involving the outer hair cells. Since the MOC neurons synapse at the base of these cells, it is possible that the efferent system can directly alter the cochlear mechanics. Thus, otoacoustic emissions have become a useful and convenient tool in studying efferent function.

Electrical stimulation of the crossed olivocochlear bundle in animals produces changes in the distortion product (DP) emissions from the cochlea (Guinan 1986; Mountain 1980; Siegel and Kim 1982). This is important evidence showing that the OCB can, in fact, alter cochlear mechanics. By changing the movement of the organ of Corti, the mechanical stimulus imparted to the inner hair cells is altered. Thus, this may indicate a mechanism by which the incoming efferent signals can alter afferent responses to sounds.

Contralateral acoustic stimuli have been shown to reduce the amplitude of transient evoked otoacoustic emissions (TEOAEs) (Collet et al. 1990), and spontaneous otoacoustic emissions (SOAEs) (Mott et al. 1989; Moulin et al. 1992) in humans. This reduction in response implies that the MOC alters the activity of the outer hair cells in such a way as to dampen the production of otoacoustic emissions. Lesions to part of this feedback loop should in theory then reduce the suppression of OAEs. This was in fact found to be the case, when inner hair cells were selectively damaged in chinchillas using
carboplatin, and the TEOAE amplitude was enhanced, even though hearing thresholds were elevated (Wake et al. 1996). Distortion product otoacoustic emissions (DPOAEs) seem to show an increase or a decrease in response amplitude in humans (Moulin et al. 1992).

The effect of contralateral auditory stimulation on DPOAEs was found to be stronger in the vicinity of spontaneous otoacoustic emissions (Moulin et al. 1992) which seems to disagree with the theory that SOAEs are produced at an area of low efferent damping activity. Tests on TEOAEs have shown that greater levels of efferent suppression occur with lower levels of ipsilateral stimulation (Hood et al. 1996; Ryan and Kemp 1996; Veuillet et al. 1996). This has also been shown with DPOAEs as well as finding that mid frequency regions give the greatest suppression (Moulin et al. 1993). The variation of efferent effect with stimulus levels agrees with the earlier studies of afferent nerve responses but not the more recent work (see section 2.3.2.1) which found that there was a larger effect at higher levels.

Contralateral sound has also been shown to produce a shift in phase of TEOAEs (Ryan et al. 1991), a shift in frequency of SOAEs (Mott et al. 1989; Moulin et al. 1992) and a decrease in the latency of DPOAEs (Giraud et al. 1997b). No signs of efferent fatigue were found for stimuli of up to 4 minutes duration (Giraud et al. 1997c), measured using TEOAEs. The frequency specificity of the contralaterally evoked suppression was demonstrated, at least for middle frequencies, with both TEOAEs (Rossi et al. 1993; Veuillet et al. 1991) and DPOAEs (Chery Croze et al. 1993). Amplitude modulation of contralateral stimuli (a phenomenon common in environmental situations) has also been found to alter the ipsilateral TOAE response (Maison et al. 1997b).

Low level contralateral stimuli were found to decrease the variability in the amplitude of TEOAE between tests, and the variability during contralateral stimulation was found to be related to the suppression in the response (Maison et al. 1997a). Since other methods of attenuating the response, such as reducing the stimuli level, also increased the variability, the MOC was assumed to be the cause of the variability decrease by stabilising active processes in the cochlea. The argument was strengthened when no effect was found in patients who had undergone a vestibular neurectomy and who would have had their efferent fibres cut at the same time as the vestibular nerve. This result fits in with the hypothesis that the efferent system acts via the outer hair cells to dampen the
motion of the basilar membrane. With a dampened basilar membrane, the whole system would be tightened and there would be less possibility of variability in the response.

There is some disagreement concerning the existence of a gender or laterality effect. A smaller suppressive effect was noted in right ears and in females by McFadden (1993) in his review paper, whereas the olivocochlear system was found to be more active on the right side, without any effect of gender, by Khalfa and Collet (1996). Large intersubject variability has been observed with normals (Collet et al. 1992; Norman and Thornton 1993).

The evidence that the above effects are mediated via the efferent system is quite convincing. In animals, Puel and Rebillard (1990) found that the DP suppressive effect was eliminated by a midline section of the brainstem involving the COCB and Kujawa et al. (1993) inhibited the suppression with strychnine, curare and atropine, antagonists of olivocochlear efferent activity. Similarly, gentamicin administration in guinea pigs abolished contralateral suppression of DPs (Avan et al. 1996). Gentamicin is thought to block medial efferent action at the level of the outer hair cell synapses.

Few studies have investigated ipsilateral suppression of OAE responses. The ipsilaterally responsive loop consists of the crossed OCB and thus contains 2/3 of the OCB population rather than the 1/3 of fibres that are activated in the majority of efferent studies using contralateral stimulation. It is important to separate out any suppressive effects that may be due to processes occurring in the cochlea other than those which are induced by the efferent system. After the first few milliseconds a longer latency suppression was observed in TEOAEs (Tavartkiladze et al. 1996) and this was assumed to be evidence for a MOC ipsilateral effect. However, only one subject was tested in this study. Analysis of DPOAEs over time in cats showed that there was a rapid adaptation of the response by 6dB caused by the primary tones (Liberman et al. 1996). Sectioning the COCB abolished the effect and thus it was attributed to action of the ipsilateral MOC reflex.

By suppressing DPOAEs with a third ipsilateral tone, which was swept across frequency, a suppression tuning curve was measured. This has been thought to be an indicator of frequency selectivity and it was found that contralateral stimulation did indeed broaden the tuning curve (Williams and Brown 1995).
Further research has addressed the question as to whether the efferent system only affects cochlear mechanics when stimulated or continuously in a tonic level of control. TOAEs were measured in chinchillas before and after COCB section (Kakigi et al. 1997) and it was found that the amplitude of most frequency components of the response (except that at 1kHz) increased i.e. the suppressive effect of the COCB had been removed. Similar results were found when measuring DPOAEs after COCB section in cats (Liberman et al. 1996). There have however, been reports of no change in DPs after OCB section in the guinea pig (Littman et al. 1992). If the efferent system does indeed provide a tonic level of control of cochlear mechanics it may either originate via a feedback loop from the ipsilateral stimulus or from a spontaneous level of activity continuously present in the OCB system.

Chang and Norton (1997) suggested that activation of the efferent system might cause modulation of adaptive processes in the cochlea via shifts in the dc operating bias of the outer hair cells. They found that in guinea pigs the quadratic distortion product \( f_2-f_1 \), which decays over time (Kirk and Johnstone 1993), declined at a faster rate and with a greater magnitude in the presence of contralateral broadband noise.

In patients who had had a vestibular neurectomy, no suppression of TEOAEs was found (Williams et al. 1993; Williams et al. 1994). Patients with lesions affecting the efferent pathway in the vestibular nerve or at the superior olivary complex have also been shown to have reduced or absent suppression (Prasher et al. 1994).

Some interesting results have come from examining efferent function in certain clinical groups. In a subject with hyperacusis, the normal suppression of TEOAEs was not observed (Collet et al. 1992), suggesting an inefficient efferent system failing to dampen responses to auditory signals. A study of a group of noise induced tinnitus patients showed abnormal suppression of TEOAEs compared to a normal group (Attias et al. 1996). Tests of efferent function may therefore prove to become useful in clinical diagnosis.

The ability of otoacoustic emissions to give a non-invasive insight into cochlear mechanics has meant that the popularity of OAEs as a tool for examining the OCB has increased dramatically over the last few years. The work so far has provided important evidence that OCB activation does actually affect cochlear mechanics. This has been
shown by reductions in TEOAEs and SOAEs and changes to DPOAEs in both animal and human subjects. The effect was also found to be frequency specific.

2.3.2.3 Effects of efferent activation on efferent fibres themselves

There have been only a few studies on acoustically evoked responses of the efferent fibres in animals. These studies are informative in that they give specific information about the timing of activation of fibres under certain stimulus paradigms. Fex (1962) demonstrated that in response to contralateral sound the efferent fibres, recorded in the vestibulocochlear anastomosis, produced regular spike trains. The fibres were found to have a high degree of frequency selectivity similar to that found in afferent fibres (Cody and Johnstone 1982a; Liberman and Brown 1986). The latter also established that most of the fibres measured were excited by only one ear and had no spontaneous activity. Binaural units were often found to have spontaneous discharge activity and were normally associated with low CF regions of the cochlea.

Differences were noted between the electrical properties of the MOC and the LOC neurons using cell patch recordings (Fujino et al. 1997). In response to depolarising current pulses, LOC fibres were found to exhibit a long first interspike interval, whereas MOC fibres had a long latency until the first spike.

2.3.2.4 Possible Influence of the Acoustic Reflex Pathway

Stimulation of the olivocochlear bundle electrically or acoustically could also stimulate the nerve fibres innervating the stapedius muscle. Contraction of this muscle would cause attenuation of the signal passing through the middle ear. This may be a confounding factor in studies where recording of efferent function involved transmission of sound through the middle ear i.e. not those where measurements were taken directly from the efferent nerve fibres. In order to be sure that results observed in the above studies were due to the olivocochlear bundle rather than the acoustic reflex, many of the workers either cut the tendons, paralysed the muscle or tested subjects with no acoustic reflex.

The suppression of OAEs has been demonstrated in subjects with absent acoustic reflex (Veuillet et al. 1991). It has also been suggested that features of the effect, such as
frequency specificity and the ability to be activated by low contralateral intensities, rule out the possibility of the effect being caused by the middle ear muscles alone.

2.3.2.5 Neuro-transmitters

The lateral and medial efferent nerves were defined anatomically, but have also been found to have differing neurochemistry. At the synapses between the medial efferents and the outer hair cells acetylcholine (ACh), calcitonine gene related peptide (CGRP) and, at the cochlea apex, γ-amino-butyric acid (GABA) have been localised. The lateral efferent synapses with the afferent auditory nerve beneath the inner hair cells have been found to contain not only ACh, CGRP and GABA, but also enkephalins, dopamine and dynorphins (for reviews see Eybalin 1993; Pujol 1994). Since so many neurotransmitters have been found to be present, it has been suggested that the lateral efferent system be further subdivided into populations dependent upon their neurochemistry. Release of combinations of these neurotransmitters in differing physiological conditions may well be indicative of both efferent systems playing a variety of roles in audition.

2.3.2.6 Summary

Although direct electrical stimulation of the olivocochlear bundle provides a convenient way of studying the effects on the cochlea of efferent activation, it is not without its problems. Firstly, although it may appear a good method of specifically stimulating the nerve in question, it is hard to rule out spread of the stimulation to other fibres, for example specific stimulation of the LOC or MOC in certain locations. Secondly, one must be careful when interpreting results from electrical stimulation with regard to the 'real life' situation of acoustic stimulation. The stimulus rates and intensities used in some of these studies may have been markedly different from those encountered physiologically. It is also worth noting that the results from work involving acoustical stimulation have the advantage of being more readily comparable between animal and human models. However, some valuable information can still be acquired from the studies using electrical stimulation, and acoustical stimulation results have in general agreed with those findings.

It must also be noted that the use of anaesthetics can reduce tonic activity in the efferent nerves as measured by studying TOAEs (Harel et al. 1997). Increases in OAE level may therefore occur and so results from experiments involving anaesthesia must be treated
with caution. One study has also examined the relative strengths of the olivocochlear reflex in humans and anaesthetised cats (Puria et al. 1996). It was found that the human OCB reflex was at least as strong, if not stronger, than the reflex in cats.

Bringing the work of the previous sections together, we can get a clearer picture of how the efferent system operates. In summary, the MOC when activated seems to be able to alter afferent responses, via the outer hair cells and their ability to alter cochlear mechanics. Generally the MOC seems to inhibit responses in auditory nerve fibres and the most common hypothesis about this mechanism is that it occurs via a damping of the motion of the basilar membrane by the outer hair cells. There is however, evidence that other mechanisms may also play a part in MOC evoked inhibition. Electrical stimulation of efferents in cats attenuated the afferent responses to tone bursts and the equivalent attenuation was found to be largest at mid dB SPL levels (Guinan and Stankovic 1996). If reduction of basilar membrane motion was the only cause of the inhibition observed then one would expect the equivalent attenuation to be greatest at low sound levels. This is because reduction in the motion of the basilar membrane is a result of the compressive non-linearity of the system tending towards linearity. The reduction in basilar membrane motion is therefore greatest at low sound levels (Sellick et al. 1982).

There are some other possible mechanisms by which MOC activation may cause an inhibition in afferent nerve activity (Guinan and Stankovic 1996). Firstly, the coupling between the basilar membrane motion and the inner hair cell stereocilia deflection may be reduced by distortion of the organ of Corti. Secondly, movement of the outer hair cells may cause small deflections of the inner hair cell stereocilia and efferent activation could reduce this coupling. Finally, electrical potentials set up by MOC activation may affect inner hair cell transmitter release. However, only the latter mechanism could possibly produce a maximal auditory nerve inhibition at mid to high sound levels. Given the present evidence from OAE studies though, the explanation for the efferent induced reduction in afferent responses seems most likely to be due a change in BM motion with the added influence of one or more other factors.

There is still little information on the functioning of the lateral olivocochlear system (LOC), since many of the above tests involve possible activation of both the MOC and the LOC. Gifford and Guinan (1987), by stimulating the MOC separately, suggested that small effects which had been previously attributed to the LOC could be accounted for by
2.3.3 Possible roles of the auditory efferent system

So far the work discussed has been concerned with elucidating the physiology of the auditory efferent system. Although some speculation of possible roles in hearing can be made from this information, the following studies have examined the validity of certain hypotheses concerning efferent function.

2.3.3.1 Protection

Past work has suggested that the efferent system could play a role in protecting the cochlea against acoustic injury. By exposing guinea pigs to loud tones, Cody and Johnstone (1982) found that contralateral acoustic stimulation of the same frequency could reduce the temporary threshold shift (TTS) encountered. This was not due to acoustic reflex pathway activation since a muscle relaxant had been used. Further evidence that the effect was efferent mediated came from the elimination of the reduction in TTS after the administration of efferent activity blocking strychnine. Electrical stimulation of the OCB was found to reduce the TTS only for levels of acoustic stimulation between 110-130dBSPL (Takeyama et al. 1992), and for high frequencies (8 and 10kHz) and short durations (1 and 2 minutes) (Reiter and Liberman 1995). Puel and Vassout (1992) found that the CAP was changed more after loud tone bursts at a rate of 1 per second than at 17 per second. This was attributed to full efficiency of the protective function at 17 per second stimulation, whereas at the slower rate there was time for the cochlea to return to its original state between each tone burst. Cutting the OCB at the level of the fourth ventricle blocked the protective effect providing more evidence that the MOC fibres play a role in protection. This was also shown to be the case in chinchillas for both TTS and permanent threshold shift (PTS) (Zheng et al. 1997a; Zheng et al. 1997b). Contradictory evidence however, came from Liberman (1992) who failed to identify any difference in the hearing of cats exposed to loud sounds either with or without section of the OCB or any protection against permanent hearing damage in guinea pigs (Liberman and Gao 1995). Rajan (1992), in his review of the protective mechanism, noted how there may be a "memory
component" to the protective mechanism, which is activated by afferent input. Stimulation of the brainstem, which would normally have caused a reduction in TTS for some time, did not produce this effect after sectioning of the COCB fibres. He suggested that the protective effect may operate via a lower brainstem reflex arc, which may be influenced by activity from higher centres, and more recently, Rajan (1995) noted that binaural exposure gave greater OCB protection than monaural exposure. He also found that the activation threshold for the efferent induced protection seemed to be dependent on the frequency of the damaging sound in cats (Rajan 1995a), and that an initial exposure to noise was able to prime the OCB to protect the cat cochlea 35 minutes later even when the second stimulus would not normally have evoked a protective response (Rajan 1996).

There have been some studies in humans of possible efferent induced protection. Scharf et al. (1994) measured the TTS in one subject who had undergone vestibular neurectomy. After presentation of a 1kHz tone at 90 dBSPL for 15 minutes only slightly less TTS was present in the operated ear than in the unoperated one. Otoacoustic emission suppression, by contralateral sound, was compared in subjects with noise induced hearing loss (NIHL) and those with other forms of sensori-neural hearing loss by Collet et al. (1991). No difference in the efferent induced suppression was found and no correlation was found between TTS and emission suppression in the NIHL group. However, there was no control group with which to compare the results in the latter part of this study and thus no conclusion about the involvement of the MOC in protection could be drawn. The suppression of TEOAEs was also measured by Veuillet et al. (1992) and it was noted that at frequencies around 4kHz no suppression occurred. A link between this and the vulnerability of the cochlea in this region was suggested.

The lack of information on human subjects relates to the difficulties in testing quantifiable noise damage. One can conclude, however, that it is possible that the efferents may play their part in protection by altering cochlear mechanics via the outer hair cells, thus reducing the travelling wave magnitude. Ideally, subjects prior to noise damage should be tested and efferent function related to cochlear damage after a period of noise exposure. Defining the degree of the efferent involvement in hearing protection may prove to be important in industrial noise exposure.
2.3.3.2 Improved Signal Detection and Discrimination

It is possible that the efferent system could help in signal discrimination in noise by suppressing responses to the background noise, therefore improving the signal to noise ratio. This theory is supported by the work of Winslow and Sachs (1987) who concluded that OCB stimulation caused a reduction in the response of single auditory fibres to masking noise (see Figure 2-14). Thus, the efferent system could play a role in signal detection in noise by restoring the dynamic range of the cochlea to almost that which it would have had without the addition of the background noise.

Figure 2-14: Rate Intensity functions for an auditory nerve fibre in response to a tone, with (dashed line) and without (solid line) electrical stimulation of the COCB. (A) No masking noise, (B) with continuous masking. Arrow show points where the tone increased the firing rate by 20 spikes/s. From Winslow and Sachs (1987) adapted by Pickles (1988).

Early behavioural studies on animals showed that sectioning of the OCB caused a reduction in a trained monkey's ability to discriminate between vowel sounds in noise (Dewson 1968) and between different frequencies (Capps and Ades 1968). More recently, vowel format discrimination in noise in cats has been found to deteriorate after bilateral OCB sectioning (Hienz et al. 1998). However, other studies on cats have revealed that COCB section causes no deterioration in either detection of single tones in noise (Igarashi et al. 1972; Trahiotis and Elliott 1970), frequency discrimination (Igarashi et al. 1979b) or intensity discrimination (Igarashi et al. 1979a). The lack of change may be due to the fact that the uncrossed olivocochlear bundle (UOCB) was not sectioned and, as mentioned earlier, some workers consider the UOCB to be responsible
for many of the effects reported experimentally. The discrepancy in the results could also be explained by considering that by steepening the rate-level function shown in Figure 2-14, COCB activation could improve the discrimination of the signal from the background noise. However, at low levels, at the base of the function (shown by the arrows), the initial increase in firing rate with increasing signal level would still occur at the same point. In this way, straightforward detection of low-level tones in noise would not show any efferent effect, but complex suprathreshold discrimination tasks would be improved.

More recently, Kawase et al. (1993) found that a contralateral acoustic stimulus could increase the afferent neuron discharge rates to a tone in a masker while the rate of activity to the masker decreased, indicating a role in signal discrimination in noise. Electrical stimulation of the COCB of a cat was found to affect intensity discrimination in noise (Winslow and Sachs 1988). In this study, the efferent system was thought to enhance intensity discrimination by restoring dynamic range and sensitivity to the cochlea. When the OCB was sectioned at the floor of the fourth ventricle it was found that intensity discrimination in background noise deteriorated only at mid frequencies (8kHz) and not at 1kHz (May and McQuone 1995).

With normally hearing human subjects, a correlation has been observed between detection thresholds to simple and complex multi tones in noise, and OAE suppression (Micheyl and Collet 1996; Micheyl et al. 1995b). Detection of speech in noise was shown to be related to MOC activity as measured by OAE suppression (Giraud et al. 1997a). This study also found that the addition of contralateral noise improved intelligibility of speech in noise in normal subjects but not in patients whose OCB is cut after vestibular neurectomy. This implies that the efferent system plays an anti-masking role thus improving speech in noise intelligibility. Additionally, patients with King-Kopetzky syndrome, where the only auditory complaint is a difficulty in discriminating speech (particularly in background noise), have been shown to have significantly less TEOAE suppression than a control group (Zhao et al. 1997). A decline in the function of the efferent system with age was found using the test of OAE suppression, which may explain the common age related complaint of difficulty in hearing against background noise (Castor et al. 1994). These results all indicate a role for the OCB in detection of signals in noise, however there have been studies of patients whose OCB is cut after vestibular neurectomy, which have found no deterioration in detection (Scharf et al.
This latter study also found no deterioration in frequency selectivity, loudness adaptation, and intensity and frequency discrimination.

Contralateral noise altered intensity discrimination in humans and this change was related to MOC function as measured by TEOAE suppression (Micheyl et al. 1997b), thus agreeing with the previous studies on animals (May and McQuone 1995; Winslow and Sachs 1988) but not the findings from vestibular neurectomy patients (Scharf et al. 1997). A study comparing groups of musicians and non-musicians found that efferent suppression of OAEs was higher in the musicians group (Micheyl et al. 1995a; Micheyl et al. 1997a). This interesting result raises the possibility that 'auditory training' could alter physiological mechanisms, thus improving our hearing.

Another suggestion has been that the efferent system may aid in temporal resolution. Giraud et al. (1997) found that the latency of DPOAEs was reduced during contralateral acoustic stimulation in normally hearing human subjects. They postulated that this may have occurred due to a shortening in the time taken for the basilar membrane to build up a peak, and if this was the case, then it would enable the auditory system to follow temporally fluctuating sounds more rapidly.

2.3.3.3 Lateralization

The literature is inconclusive regarding the efferent system and sound lateralization, as there have been few studies with varying results. Early work (Fisch 1970), indicated that subjects post vestibular neurectomy, had deficits in their ability to lateralise sounds. However, changes in the hearing sensitivity may have accounted for this. More recently, Scharf et al. (1997) found no defects in a subject's ability to judge the position of a sound after vestibular neurectomy.

2.3.3.4 Attention

The effect of attention on hearing and its link with the efferent system has been studied by a number of workers. The earlier work on animals by Glenn and Oatman (1977) examined the effect of a visual task on the ABR in cats. The amplitude of the ABR was found to decrease during the task and the latency of N1 increased. Oatman and Anderson (1977) also found a decrease in response amplitude, with mid frequency tones suppressed more, and detected no change in the cochlear microphonic (CM).
In more recent work with humans, Brix (1984) tested the influence of selective auditory attention on the ABR of 100 subjects, and found a decrease in the I-V interval. However, the length of the ABR test causes problems with regard to attention span, so with the advent of OAE testing this has made it easier to measure the subtle changes involved in a shorter length of time. During a visual task, a decrease in the amplitude of the dominant frequency of evoked OAEs was measured (Puel et al. 1988), but no change was found in DP or stimulus frequency emissions (Avan and Bonfils 1992). The latter two studies however, involved comparing the attentional task with a passive task which may have confounded the attentional effects with general alertness. Froehlich et al. (1993) found a decrease in the amplitude of evoked OAEs, which was maximum at approximately 1-2kHz for a visual task and at 2-3kHz for an auditory task, suggesting that attention in different modalities acts on different parts of the cochlea. Using a test where the direction of auditory attention was changed, tone evoked OAEs were found to be greater at the target frequency when attention was directed towards that ear (Giard et al. 1994). However, there have been other tone evoked OAE studies demonstrating no influence of the efferent system in selective attention (Michie et al. 1996). Scharf et al. (1997) found that after numerous tests on vestibular neurectomy patients there was significant change in the detection of unexpected signals in noise. This was attributed to an impairment in selective attention, and suggested that the efferent system was the cause of the change.

It thus seems to be the case that input from higher centres can affect the level of efferent action on the cochlea. This may prove useful when wishing to concentrate on a particular signal in a particular ear. It is therefore clearly important to control the attentional state of the subject when testing the efferent system.

### 2.3.3.5 Synchronisation

Berlin et al. (1993) suggested that the efferent system could be involved in synchronisation of the afferent activity, after testing a number of subjects with normal thresholds at 2kHz, robust OAEs but absent ABR and acoustic reflexes. The authors thought however, that it was more likely that these findings were due to a deficit in afferent function.
2.3.3.6 Summary

Although the effects of OCB activation have been studied in detail in both animals and humans, the role that the MOC, and especially the LOC system play in hearing, have still to be clarified.

Past studies have demonstrated that activation of the efferent neurons leads to a reduction in N1 response, an increase in the cochlear microphonic, a decrease in the endocochlear potential, desensitisation of the inner hair cells and changes in cochlear mechanics shown by studies of otoacoustic emissions. The actions of the efferent nerve were found to be frequency specific. With regard to the role that this system plays in the hearing process, there is evidence that it may be involved in protection of the ear against damagingly loud sounds and the detection of signals in noise. There is also evidence that the efferent system may facilitate a method by which the higher centres of the brain can affect incoming signals at the level of the cochlea, for example in situations where attention is needed to a particular input. The use of OAEs is very valuable in studying these situations.

The function of the LOC is still particularly poorly understood. It seems unlikely that such a system would have no influence on hearing.

2.4 Measurement of Frequency Selectivity in Humans

In animals, frequency selectivity can be investigated by measurements of nerve fibre responses to different stimuli and by direct measurements of the motion of the basilar membrane. Since these methods are inappropriate for human studies psychoacoustic techniques have been developed to enable measures of frequency selectivity to be obtained.

Frequency selectivity can be described as the ability to discriminate a particular signal in the presence of others. Masking techniques are therefore commonly employed in tests of frequency selectivity. Fletcher (1940) measured the threshold of a tone in masking noise as the bandwidth of the noise was increased. It was found that the threshold increased to a point before reaching a plateau. This was taken to indicate that only the narrow band of frequencies close to the probe tone were effective as maskers. This bandwidth was
named the ‘critical band’. Fletcher (1940) made the assumption that the filter which determined how much of the masker and signal could pass, was rectangular. He suggested that the basilar membrane was the origin of a bank of overlapping such filters responding to different frequencies.

However, noises outside the critical band are able to mask the probe signal. This is indicative of the fact that the auditory filter is not rectangular in shape but has sloping sides. One method for finding the shape of the auditory filter involves measurement of the detection threshold for a fixed level probe signal with a masker (tone or narrow band noise) which varies in frequency. The resulting threshold graph is called a psychophysical tuning curve (PTC). The shape of the PTC is very similar to that of the tuning curves measured directly from nerve fibres therefore indicating that both methods are examining the same physiological process.

In response to any stimulus, the auditory system uses the auditory filter that is best for the input. It is important when measuring the shape of the auditory filters that all the measurements are taken from the same filter. It may be the case that in some listening conditions the subject automatically uses a filter of a different centre frequency since this provides an improved signal to noise ratio. This is called off-frequency listening.

In order to minimise off-frequency listening, notched-noise can be used as the masker. Noise is also preferable to a sinusoidal signal as the masker because it reduces the effect of beats, which can be used as a cue. Therefore, a preferable method to use, and the one chosen for this study, is that called the notched-noise method.

The filter shape however, is not symmetric especially at higher and lower levels. In order to measure the asymmetry of the filter, maskers which are asymmetric about the signal frequency are used. By measuring the threshold of the signal at various notch widths, the shape of the auditory filter can be estimated. The details of the experimental method used will be described in more detail in section 3.1.

Studies into the frequency selectivity of the human auditory system using psychoacoustic techniques have shown that as the centre frequency rises the bandwidth of the filter also rises (Glasberg and Moore 1990). The bandwidth for frequencies above 1kHz is approximately 10-17% of the centre frequency. The relationship between the equivalent rectangular bandwidth (ERB) and frequency (F) is fitted quite closely by
The ERB is an approximation of a filter. It assumes its shape to be rectangular and to be able to pass the same power as the original filter.

The bandwidth also increases with age and with level of the signal. There is an accompanying increase in the filter asymmetry in the latter case mostly due to the lower skirt of the filter becoming shallower. At lower levels, the filter is approximately symmetrical on a linear frequency scale.

2.5 Thesis Rationale

The auditory efferent system consists of an extensive network of fibres running to the cochleae. So far, there is little firm evidence about the exact nature of its function in hearing. It seems unlikely that it has no purpose, although past studies have certainly been inconclusive in finding a specific role. The importance of the efferent system in hearing is that it is able to modify auditory signals as they are received before reaching the cortex.

As can be seen from the work discussed in section 2.3.3 studies have been carried out which have focused on the possible role of the efferent system in protection against loud sounds, attention, lateralisation and in signal discrimination in noise. It is entirely possible that some of these roles may actually be combined e.g. attending to a sound in order to increase the signal to noise ratio. This study examined the possible contribution that the efferent system may play in finely tuning the cochlea, since there has been little conclusive work on this subject, especially in humans.

The hypothesis under examination is that the efferent system could act, instigated by either signals from the cortex or from the contralateral ear, stimulating the outer hair cells to change shape or stiffness and thus changing the mechanics of motion of the cochlear partition in response to incoming stimuli. This would then force a modification of the travelling wave peak and thus more or less inner hair cells would be stimulated to different degrees. The ascending signal could therefore be changed. In order to enhance frequency selectivity, this would involve efferents modulating outer hair cell stiffening to different extents along the length of the cochlea, to increase coupling of energy at the
signal frequency and/or decrease coupling away from the signal frequency. We know that active processes are involved in maintaining a high degree of frequency selectivity (see section 2.1.2.3(d)) and that the medial efferent fibres synapse at the outer hair cells. Since outer hair cells have been implicated in the provision of energy input into cochlear mechanics it seems feasible that the efferent system may have some control over this mechanism and therefore the tuning of the cochlea. Via this mechanism, it may be possible for the cortex to control frequency selectivity, e.g. in situations where attention to a stimulus is required, or for a brainstem level olivocochlear reflex to modify signals in one ear after stimulation from the other. In order for this reflex loop to be useful in aiding frequency selectivity for transient stimuli, this would have to occur quickly. Thus, the timing of the activation of the modification to tuning is crucial and must be short. For this reason, we also examined the latency of activation of the olivocochlear response. Finding this latency also allowed us to set the stimulus timing parameters for the contralateral sounds in the tuning experiments.

As mentioned, there has been relatively little work in linking efferent activity with auditory tuning, particularly so in humans. However, there has been work in animals where the COCB has been cut or electrically stimulated which has caused a degradation in tuning. Figure 2-15 shows the effect on inner hair cell tuning curves of COCB stimulation by electric shocks. The tuning curve is raised in threshold at the tip and broadened slightly. Frequency selectivity changes were also found in the auditory nerve fibre responses after electrical stimulation (Guinan and Gifford 1988b). Since tuning curves from inner hair cells and eighth nerve responses are similar to the mechanical tuning of the cochlear partition (Sellick et al. 1982), it is not unreasonable to expect any effects of the efferent system to show up as changes in these studies.
However, recently, more direct measurements of the motion of the BM have been made using laser interferometry (Murugasu and Russell 1996). During COCB electrical stimulation, similar results were found to those of the auditory nerve and inner hair cell tuning, with a desensitisation of the tip of the tuning curve. A downward shift in frequency of the tuning curve was also seen sometimes, by up to 500Hz. The broadening of the tuning curve was not found to be consistent.

Electrical stimulation does not mimic the physiological situation very realistically, however. Therefore, other studies have looked at the effect of sectioning the efferent fibres or by stimulating them acoustically.

Examining the tonic efferent input (or the ongoing activity present without stimulation), some studies have looked at the effect of cutting different portions of the nerve on the tuning. Sectioning at different levels of the OCB pathways revealed changes in nerve fibre tuning in guinea pigs only when the UOCB was sectioned (Bonfils et al. 1986a; Bonfils et al. 1986b). Carlier and Pujol (1982) also found that CAP tuning curves in cats were broadened by sectioning the OCB at the level of the vestibular nerve. However, some similar studies have found that OCB section has no effect on the tuning (Liberman 1990; Littman et al. 1992; Rajan et al. 1990). It is unclear why the results of these
experiments differ. One can only assume that there must have been differences in experimental technique or incomplete sectioning of the efferent fibres in some cases. It is hard to find an alternative explanation for the observed changes to the tuning though, if they were not due to lack of efferent input.

Cutting the efferent nerve has also been shown to affect those behavioural measures that are reflections of auditory tuning. Discrimination of speech in noise by rhesus monkeys was found to deteriorate after COCB section (Dewson 1968), as were pure tone masked thresholds in cats (Trahiotis and Elliott 1970). Ablation of the COCB has also been associated with a deterioration in squirrel monkeys' ability to perform a frequency discrimination task (Capps and Ades 1968), although Igarashi et al. (1979) found that there was no difference in cats. The only similar studies in humans (Scharf et al. 1997; Scharf et al. 1994) observed no change in tuning in patients who had had their OCB sectioned during a vestibular neurectomy.

Recently, a study on the development of hearing in cats after sectioning the OCB (Walsh et al. 1998) has shown that they develop broader tuning curves.

So, the past work on animals has come to no real conclusion about the involvement of the efferent system in frequency selectivity. Certainly, the results from experiments involving electrical stimulation of the efferents show effects more clearly than those with acoustic stimulation or those where the nerve was cut. Unfortunately, electrical OCB stimulation is not an alternative when wishing to examine the effect in humans. Therefore, in order to study the same question in human subjects the use of OAEs has proved to be valuable as a non-invasive tool giving an objective insight into cochlear function. The effect of efferent activation on distortion products has been examined (Williams and Brown 1995). Assuming distortion product suppression by a third ipsilateral tone to be a measure of frequency selectivity, they found that contralateral acoustic stimuli produced a broadening of the suppression tuning curve.

Reviewing the work in section 2.3.2.2, it can be assumed that the contralateral suppression of TEOAEs can be used as a measure of the strength of action of the MOCB on the peripheral auditory system. This test gives robust results and is easy to test in human subjects. For these reasons, it has been chosen for use in this study. Micheyl and co-workers also used this test in order to give a guide as to the involvement of the OCB in detection of signals in noise. A correlation was found between the degree
of contralateral suppression of TEOAEs and detection thresholds of 2kHz tones in noise whilst a contralateral masker was present (Micheyl and Collet 1996). Those subjects with greater contralateral TEOAE attenuation were found to have better detection performance.

We have chosen to look at the influence of the efferent system on frequency selectivity measured by a psychoacoustic technique of auditory filter shape measurement. Brown et al. (1993) compared the DPOAE tuning curve generated when one of the primaries was swept and the other was kept constant and found that there was some correlation between this and the psychoacoustically measured filter bandwidth. However, they encountered problems with variability and noise in measuring the DP curve bandwidth. An objective test of frequency selectivity would have been ideal to use in our study but it was considered that there were no reliable such test available at present and thus psychoacoustic tests would make a good alternative.

The only study which has combined measurement of TEOAEs and a psychoacoustic measure of filter shape in the same subjects is that of Micheyl and Collet (1994). They found that quality of the tuning of the auditory filter was related to the size of TEOAEs and to the presence or absence of SOAEs. Larger TEOAEs were associated with a lower quality factor.

In this study, we aim to go further than this latter work. We have not only measured the relation of active processes (measured via TEOAEs, SOAEs and DPOAEs) to frequency selectivity, since it is important to determine this first, but have also looked at the possible link with efferent system activity (measured via contralateral suppression of OAEs). We have used a psychoacoustic test that is thought to give more accurate measures of auditory filter shape than that used by Micheyl and Collet (1994). In our method, we employed the notched-noise technique of filter shape estimation, which reduces the effect of off-frequency listening. We have examined how contralateral stimulation affects the auditory filter shape and have related this to efferent function.
Chapter 3

Equipment and General Methods
3. Equipment and General Methods

3.1 Auditory Filter Shape Measurement

3.1.1 Modelling of Auditory Filter Shape

As previously discussed, the tuning of the cochlea can be thought of as a series of bandpass filters. The shape can be modelled fairly accurately when the skirts on either side of the filter are approximated by an exponential function and the peak is flattened (see Figure 3-1).

Figure 3-1: Approximation of auditory filter shape

This is called the rounded exponential function (ROEX) and, according to Patterson et al. (1982), has three forms depending on the range of the filter. The simplest of these is given by the equation

\[ W(g) = (1 + pg)e^{-pg} \]

where the parameter \( p \) determines not only the width of the passband but the gradient of the skirts of the filter. The parameter \( g \) determines the distance of the point of interest from the centre frequency, and is normalised to the centre frequency. The threshold
curve can be found by integrating the filter equation. The equivalent rectangular bandwidth (ERB) can be found using

\[
ERB = \frac{4f_0}{p}
\]

This single parameter filter model Roex(p) has side tails which decrease indefinitely. Thus, in order to give a more accurate model of these tail areas, a three parameter filter was suggested by Patterson et al. (1982). This Roex(p,w,t) filter is given by

\[
W(g) = (1 - w)(1 + pg)e^{-pg} + w(1 + tg)e^{-pg}
\]

where \(w\) determines at what stage there is a change over in which term dominates and is usually much less than 0.5. The shape of the tail of the filter is determined by \(t\).

In practice it is difficult to accurately determine \(w\) and \(t\) and therefore the Roex (p,r) model can be used.

\[
W(g) = (1 - r)(1 - pg)e^{-pg} + r
\]

Here the parameter \(r\) imposes a limitation on the dynamic range. For each of the above equations, the filter can be asymmetric by allowing \(p\) to have different values on each side of the filter. Thus, \(p_l\) and \(p_u\) can be substituted in to the equations for the lower and the upper branches respectively. Patterson and Nimmo-Smith (1980) described a method by which the filter shape could be found which best fits the measured data. Starting values for \(p_l\) and \(p_u\) were first assumed and then the values were altered until the difference between the actual and the predicted values was minimised via the least squares method.

The computer program “Polyfit” described by Rosen and Baker (1994) was used to fit a Roex (p,r) model to the data measured. Data was inputted via a text file containing not only the threshold values, but the details of the maskers, the probe level, frequency and whether a fixed probe or fixed masker paradigm had been used. The parameters generated described the shape of the best fitting auditory filter and were as follows:

a) \(p_l\) - determines the rate of fall off of the lower skirt of the filter

b) \(p_u\) - determines the rate of fall off of the upper skirt of the filter

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c) $k$ - measure of detector efficiency at filter output i.e. signal-noise ratio (dB)

d) $r_l$ - gives a dynamic range limitation for the lower slope of the filter

e) $r_u$ - gives a dynamic range limitation for the upper slope of the filter

f) The 3dB bandwidth of the filter

In the command that ran the program, different flags could be assigned values to instruct the fitting procedure to take certain factors into account. The number of polynomial terms used to describe a parameter could be specified. This is useful when trying to fit data from more than one level at one frequency (Rosen and Baker 1994), but in this case there was only one level and therefore only one coefficient per parameter was needed. Off-frequency listening was provided for by the fitting procedure and was allowed to extend to ±15% of the probe frequency. For simplicity, $r_l$ and $r_u$ were constrained to being equal, although $p_l$ and $p_u$ were allowed to vary independently.

3.1.2 The Notched Noise Method

As mentioned earlier, a method of notched-noise masking was chosen to estimate the shape of the auditory filter, whereby the detection threshold of the probe signal in various masker notch widths was measured. By using this method, one can minimise the effects of off-frequency listening and of additional cues gained from beats. In addition, the use of asymmetrically positioned notches allowed the asymmetry of the filter to be described.

The measurement of auditory filter shapes using the notched noise method can become very time consuming when many masker configurations are used. In order to make the test suitable for clinical use the test must be shortened in some way. Stone et al. (1992) suggested that the 13 or 19 notch widths commonly used in laboratory studies could be reduced to 5, allowing the asymmetry and sharpness of the auditory filter to still be estimated with reasonable accuracy. Leeuw and Dreschler (1994) found that 5 or 7 notches gave similar standard deviations as 13 notches, but when the smaller number of notches is used the thresholds should ideally be measured twice.²

---

¹ Use of a bandwidth measurement at a wider point of the auditory filter (e.g. 10dB bandwidth), may have provided a more accurate estimation due to its proximity with the widths of the notches used.

² Use of only 5 notches meant that each filter skirt was effectively determined by only 3 points. However, in the interest of saving testing time, this possible loss of accuracy was considered necessary.
Thresholds were therefore measured with five notches. This procedure was repeated to give two sets of data for each notch, in order to minimise the error in the bandwidth estimation. The equipment generated an average threshold and standard deviation for each notched noise test. A final value for the threshold was calculated from the original data by pooling all of the data from both tests and finding the mean and standard deviation. This allowed runs with higher standard deviations (having probable lower accuracy) to be given less weighting in the final average, rather than giving all runs equal weighting.

Rosen and Baker (1994) found that filter models where the parameters depended on probe level were much more successful at describing the data than those in which the parameters depended on masker level. Thus, for this study the experiments were carried out keeping the probe level fixed and varying the masker level to find threshold.

The level of the notched noise masker tracked up and down automatically via the TDT equipment. The set up was a ‘two alternative forced choice’ scenario (2AFC). The subject listened to two stimuli (Figure 3-2) both of which contained the notched noise (350ms in length) but only one of which contained the probe tone burst (250ms in length, starting 50ms after the onset of the noise burst). The rise and fall times for the masker and the probe tone were 5ms.¹

Figure 3-2 Arrangement of notched-noise and probe tone in time

¹ The ramp was introduced to eliminate high frequency transients, although a 10ms ramp may have been more effective in doing this.
The subject then responded as to whether they thought they had heard the probe tone in the first interval or the second. Using this method has the advantage of eliminating some of the bias of standard threshold testing. For example, an over cautious subject, fearing giving a false positive response, may only press the button for a signal when they are sure they can hear it because it is some way above their threshold. In the 2AFC procedure, they have to make a choice for each pair of responses. Likewise, the subject who is afraid of missing any stimuli and presses the button frequently, giving many false positive responses, will give a more accurate result with the 2AFC procedure since they have to respond every time. The 2AFC procedure also has the advantage that presentations that include the test signal are presented frequently. It is possible, using other techniques, for there to be many presentations in a row where the subject cannot hear the stimuli, and therefore may forget what they are listening for.

The occurrence of the tone burst in the first or the second interval was random and controlled by the program. The subject was then prompted to respond by pressing a key on the computer keyboard depending on when they thought they had heard the tone: “Z” for the first interval, “M” for the second. Visual feedback was given to the subject via the computer monitor showing whether they were correct or incorrect with their choice. Since individuals’ strategies varied, it was found useful to provide visual feedback as well as a verbal description of the task, as this often made the test easier to learn.

An adaptive procedure was used to change the masker levels. This meant that the level of a presentation was determined by the response to the preceding presentation. The level of the notched noise masker was increased after 3 correct answers and decreased after one incorrect answer. This technique of threshold estimation gave the 79.4% correct point of the psychometric function (Levitt 1971). The step size started initially at 10dB and decreased to 2dB after 3 reversals. This allowed the threshold to be approached quickly but found accurately, meaning that each run could start with levels where the tone was easily detectable for the subject. Only the data acquired from the last even number of reversals after 3 complete reversals was used to calculate the mean threshold.
The threshold of the tone was measured with five different narrow band maskers. Figure 3-3 shows the frequency distribution of the maskers around a probe tone at 1kHz. The width of the noise bands was 0.4 of the probe frequency. All noise bands were flat in level across frequency. Condition 1 had no-notch in the noise, and in conditions 2-5, the
bands of noise were positioned asymmetrically around the probe tone. This was to allow the asymmetry of the filter shape to be assessed. The notches were defined using the normalised value $g$,

$$g = \frac{|f - f_0|}{f_0}$$

where $f_0$ is the probe frequency and $f$ is the frequency of the nearest edge of the masker to the probe tone. The $g$ values for the five notches are shown in Table 3-1.

### Table 3-1: Notched-noise masker $g$ values

<table>
<thead>
<tr>
<th>Masker Number</th>
<th>Lower $g$</th>
<th>Upper $g$</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>2</td>
<td>0.1</td>
<td>0.3</td>
</tr>
<tr>
<td>3</td>
<td>0.3</td>
<td>0.1</td>
</tr>
<tr>
<td>4</td>
<td>0.5</td>
<td>0.3</td>
</tr>
<tr>
<td>5</td>
<td>0.3</td>
<td>0.5</td>
</tr>
</tbody>
</table>

The starting level for the masker was varied between conditions to keep the dB difference between the start and expected threshold value approximately equal. This helped to reduce the time taken to track to threshold.

The number of trials for each notched-noise condition was set to 55. This meant that a good compromise was achieved between length of test and number of points that could be used to find the mean threshold. It was very important to reduce the length of the test to shortest time that could still give a good measure of the auditory filter shape. Runs of 55 trials took 2-3 minutes to complete and usually generated 6-12 values from which to find the threshold. The interval between the two trials was set to 100ms and the inter-trial delay was set to 300ms. Again, this minimised the test time whilst still making it manageable for the subject. The five notched-noise conditions were repeated with each subject in order to increase the accuracy of the final result. The data from a run was only accepted when the standard deviation of the mean threshold from that run was less than
3dB. This usually meant that a couple of runs would need to be repeated. The other
criterion for accepting data was that the mean thresholds for the two runs of the same
condition were within 5dB of each other. If not, another run was performed. Thus, it
usually took approximately 45 minutes to get a complete set of data, although this did
depend on the individual subject's response times.

3.1.2.1 Stimulus set up for measuring the auditory filter at 1kHz

The probe signal of 1kHz was set at a level of 25dBSPL and a starting phase of zero.
This level was achieved by attenuating the maximum signal by 77.6dB.

It was decided not to vary the level of the probe tone to compensate for the differences
in individuals pure tone thresholds. The well documented microstructure in human
auditory threshold (Elliot 1958) could mean that thresholds in the frequency range of
interest may vary by up to 12dB from that measured at the standard audiometric
frequencies. In these experiments, we are interested in the auditory filter and therefore it
is important to consider how the auditory threshold would vary within this frequency
range. Zwicker (1990) measured the periodicity in continuous tonal SPL and found it to
be 0.4 Bark. The 'Bark' represents one critical bandwidth and relates to a constant
length along the basilar membrane of 1.1mm. Assuming the critical bandwidth that was
used to derive the 'Bark' is approximately equal to the width of the auditory filter, as
measured in these experiments, it can be concluded that the auditory threshold may
undergo 2.5 full cycles of variation with a possible peak-to-trough threshold difference
of 12dB within one filter width. Thus, altering the level of the probe for each subject
would only make any sense if a highly detailed auditory microstructure was determined
beforehand. It has been shown that the peak-to-trough difference of the microstructure is
less pronounced at higher levels (Kemp 1979b) but at the levels used here it is unlikely
that the microstructure would have flattened out. Another consideration is that although
using a constant dBSPL would mean that the dBSL level may differ between subjects, it
does mean that since the input level of the stimulus to the cochlea will be approximately
the same for each subject (apart from differences in stimulus level reaching the cochlea
due to outer and middle ear attenuation), the mechanics of the cochlea in each case are
operating at the same point. This will therefore minimise differences in the spread of
masking that occur with changes in level.
The masker level was then varied to find the threshold.

The frequencies of the notched noise maskers are shown in Table 3-2 for the probe tone at 1kHz. The starting levels for the maskers were 1.8, 16.8, 26.8, 36.8, 31.8dB/Hz which were achieved by attenuating the signals by 60, 45, 35, 25, and 30dB for conditions 1-5 respectively. The initial value was varied for each condition so that there was always approximately the same change in dB level needed to reach threshold.

Table 3-2: Notched-noise masker frequencies for a 1kHz probe tone

<table>
<thead>
<tr>
<th>Masker Number</th>
<th>Lower g</th>
<th>Upper g</th>
<th>Lower band (Hz)</th>
<th>Upper band (Hz)</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>0</td>
<td>0</td>
<td>600-1000</td>
<td>1000-1400</td>
</tr>
<tr>
<td>2</td>
<td>0.1</td>
<td>0.3</td>
<td>500-900</td>
<td>1300-1700</td>
</tr>
<tr>
<td>3</td>
<td>0.3</td>
<td>0.1</td>
<td>300-700</td>
<td>1100-1500</td>
</tr>
<tr>
<td>4</td>
<td>0.5</td>
<td>0.3</td>
<td>100-500</td>
<td>1300-1700</td>
</tr>
<tr>
<td>5</td>
<td>0.3</td>
<td>0.5</td>
<td>300-700</td>
<td>1500-1900</td>
</tr>
</tbody>
</table>

3.1.2.2 Stimulus set up for measuring the auditory filter at 2kHz

The procedure for measuring the auditory filter at 2kHz was exactly the same as that described previously for measuring the filter at 1kHz. The 2kHz probe tone was set to a level of 22.5dB/Hz. The frequencies of the notched-noise maskers are shown in Table 3-3. The widths of the noise bands were increased to 800Hz in order to keep their bandwidth at 0.4 of the probe frequency, as was the case at 1kHz.
Table 3-3: Notched noise maskers for testing the filter at 2kHz

<table>
<thead>
<tr>
<th>Masker Number</th>
<th>Lower g</th>
<th>Upper g</th>
<th>Lower band (Hz)</th>
<th>Upper band (Hz)</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>0</td>
<td>0</td>
<td>1200-2000</td>
<td>2000-2800</td>
</tr>
<tr>
<td>2</td>
<td>0.1</td>
<td>0.3</td>
<td>1000-1800</td>
<td>2600-3400</td>
</tr>
<tr>
<td>3</td>
<td>0.3</td>
<td>0.1</td>
<td>600-1400</td>
<td>2200-3000</td>
</tr>
<tr>
<td>4</td>
<td>0.5</td>
<td>0.3</td>
<td>200-1000</td>
<td>2600-3400</td>
</tr>
<tr>
<td>5</td>
<td>0.3</td>
<td>0.5</td>
<td>600-1400</td>
<td>3000-3800</td>
</tr>
</tbody>
</table>

3.1.3 Equipment

Tucker -Davis Technologies (TDT) System II hardware was chosen, purchased and set up to provide all the stimuli. Control was via a processing card in an attached P.C. and the equipment could be programmed using software supplied with it. All testing was carried out in a sound-treated room, the walls of which were double layered and insulated in order to attenuate external sounds. The computer monitor, keyboard, mouse and headphones were the only equipment inside the booth. The computer itself had to be kept outside the room since the noise from the fan was too loud. The TDT hardware modules were also kept outside the room, since they were attached by fibre optic cables to the computer. Connecting cables were passed through a small hole in the double wall of the booth for the monitor, keyboard, mouse and headphones.

3.1.3.1 Hardware

The TDT equipment consisted of 8 modules and a processing card in the P.C. The modules were mounted in two device caddies called XBUS (XB1). Each caddie held four devices and provided the power, communication and synchronisation for the units. The two XBUSs were linked together by a ribbon cable and these were then linked to the array processing card in the P.C. (AP2) via 6 fibre optic cables (see Figure 3-4). The optical fibre link module (OIL) was situated on the back of one of the XB1s. The fibre optic cables allowed error free transmission of data over greater distances. Power was
provided from the mains via a transformer (PWS25) mounted on the back of the XB1. The AP2 used digital signal processing technology and allowed the user to program the external devices. It had data and signal storage capabilities.

The first stage for the signal, after arriving at the XBUS via the fibre optic link, was to pass through the digital-to-analogue converter (DA1). The DA1 could sample at 170kHz and had 16-bit stereo conversion accuracy. The probe tone signal passed through one channel whilst the masker passed through the other. Both signals were then fed into the anti-aliasing filter (FT5). The FT5 consisted of two passive lowpass filter blocks with corner frequency placement accuracy of 1%. The unit was op-amp buffered at the input and the output allowing for impedance matching. Each signal was then passed through one of the programmable attenuators (PA4). These were single channel devices with a range of 0 to 99.9dB. The attenuation resolution was 0.1dB and the accuracy was 0.05dB. Switching transients were almost eliminated by use of a sample and hold amplifier, which froze the output during changes. The PA4s were programmed via the P.C. and the XBUS, but could also be programmed manually from the front panel of the device. After attenuation, the signals were summed in the weighted signal mixer (SM3). The gain for each channel could be altered, but in this experiment, both channels were kept equal. The final output stage was the headphone buffer (HB6). The input to the headphone buffer was a BNC type cable in either the left of the right ear inputs. A cable was constructed which consisted of a 0.25in stereo jack plug for the output from the HB6 and two mono 0.25in sockets into which the headphone leads could connect. This cable was passed through the small hole in the wall of the sound proof room. The headphones were the audiometry standard type (TDH49). Due to the very low impedance of these headphones (~10Ω) an extra 10dB of attenuation was required at the HB6. A switch on the front panel allowed this to be achieved. The HB6 also included an extra monitor output, which allowed the tester to sit outside the booth and hear how the test was progressing. The layout of all the above units is shown in Figure 3-4.
The low impedance of the headphones unfortunately gave some signal-to-noise problems. Since they were so sensitive, this meant that a loud signal could be played out without using the full range of the DA1. Therefore, when it came to attenuate the signal the voltage was already so low that it went right down into the noise floor. This meant that there was a great deal of noise breakthrough and distortion. The original program had the two signals mixed before the DA1 and this one signal was then attenuated by only one PA4. To allow both the tone and the masker to use the full range of the DA1,
they were separated into different channels. In order to allow for this, a new program was developed by the author, which separated the probe signal from the masker.

The waveform generator (WG1) produced stimuli for the contralateral ear. This module was programmable from the unit itself. White noise was generated and the amplitude and duration were altered manually. Additional attenuation was required to bring the level of the signal down to a suitable point. This was provided by a step attenuator. Synchronisation with the ipsilateral stimuli was provided via an external trigger from the DA1 module. Figure 3-5 shows the experimental set up of the modules used. In order to match the impedance of the attenuator to the modules on either side of it, one 600Ω resistor was placed in series after the WG1 and one parallel before the HB6.

Figure 3-5: Schematic diagram of equipment set up to produce contralateral noise

![Schematic diagram of equipment set up to produce contralateral noise](image)

The attenuator was found to have an internal attenuation of 6dB and so in order to give 30dB of attenuation, the level was set to 24dB. The signal was then passed to the HB6.

In order to produce narrowband noises for the contralateral ear, the white noise signal from the WG1 was also passed through the programmable filter (PF1). The filter frequencies could be programmed into the module itself, allowing for only the selected narrow band of frequencies to be passed.

3.1.3.2 Software

Programs were developed under the “Xperimenter” software, which drove the hardware. “Xperimenter” is an environment designed specifically for running psychoacoustic
experiments. The program allowed the above stimuli to be produced, defined the tracking procedure and specified the data that should be used for finding the threshold.

In order to run the experiment a "condition file" was produced. Each condition file contained condition blocks controlling the trials. The condition block was in turn divided into three sections:

a) the general parameter section- e.g. number of trials

b) the variable section- e.g. parameters for tracking of level

c) the signal section- e.g. definition of signal frequency and duration

Each condition block began with the keyword COND and ended with ENDCOND. Lines starting with a semi-colon were ineffective and for information only.

The statements that define variables were defined by a keyword followed by one or more arguments. All statements are contained within one line except COND, TRACK and SIGNAL. Each of these requires a few lines of statements, which are contained within starting and finishing keywords e.g.

TRACK: 1 -65.0 -90.0 0.0

BIG_STEP: -10

SMALL_STEP: -2

NUM_UP: 1

NUM_DOWN: 3

NUM_BIG_REVS: 3

NUM_TOS_REVS: 3

ENDTRACK

The signals are defined within the SIGNAL/ENDSIGNAL keywords. The type of signal, frequency (Hz), duration (ms), time delay (ms) and starting phase (°) can all be defined here. In the case of the notched noise maskers, two bands of noise are defined by the LINBAND statement, the edge frequencies of which are specified in the STEP
command\textsuperscript{1}. The level of attenuation of the noise was defined by the TRACK command since this specified how the levels changed with each trial. The level of attenuation was kept equal across the frequencies giving a flat noise spectrum.

The Xperimenter software includes a number of paradigms that define the way the signals are combined and presented. The stimuli in the interval in which the signal appears are called the “signal” and the stimuli presented when the probe signal is not present are called the “standard”. The masker is played in channel A of the D/A converter and the probe tone is played in channel B. The P1 paradigm was the simplest arrangement that allowed the probe and masker to be presented as required.

\begin{center}
\begin{tabular}{|l|}
\hline
\textbf{P1 Paradigm} \\
\hline
\textbf{Signal} :- \\
\hspace{1cm} Channel A= Signal 1 (masker) \\
\hspace{1cm} Channel B= Signal 2 (probe tone) \\
\textbf{Standard}:- \\
\hspace{1cm} Channel A= Signal 3 (masker) \\
\hspace{1cm} Channel B= Signal 4 (probe tone not present) \\
\hline
\end{tabular}
\end{center}

However, in order to present a stimulus to the contralateral ear, which was synchronised to the ipsilateral stimuli, a different arrangement was required.

In the P2 paradigm, a third signal can be presented. This third signal was set to produce no sound, but just to act as a time reference. The contralateral stimulus was produced by the WG1 module and was triggered by the signals from the DA1. Thus, by use of the time reference signal, the start time of the contralateral noise could be varied with respect to the ipsilateral stimuli.

\textsuperscript{1} The STEP statement allows parameters to vary over multiple runs. In this case each run was separated into a different condition file and so each STEP statement contains only one set of parameters.
P2 Paradigm

Signal:-
  Channel A= Signal 1 (masker) + Signal 3 (silence/time reference)
  Channel B= Signal 4 (probe tone)

Standard:-
  Channel A= Signal 5 (masker) + Signal 7 (silence/time reference)
  Channel B= Signal 8 (probe tone not present)

Results were logged to disk following each set of trials and a graph of the results was displayed. The file in which the results are saved was a text file. The subject details were displayed along with the average dB level and standard deviation calculated from the tracking procedure. The dB level for each individual trial was then shown with the values used in calculating the average marked with an asterisk. Thus these values could be noted and combined with the results from the repeat measurement to make one average value.

Appendix 1 contains the program that ran all the 1kHz filter measurements. This program was based on the P2 paradigm, which permitted an extra time reference to be added. This allowed the contralateral noise to be presented before the start of the ipsilateral stimuli. The same program was also used without any contralateral noise in the experiment in chapter 5. For measurements of the auditory filter at 2kHz the program in Appendix 2 was developed. This is similar to the other program except for the frequencies of the probe and maskers and that it is based on the P1 paradigm, since no contralateral stimulus was being used.

3.1.3.3 Calibration

Within the Xperimeter program the calibration mode can be specified. This allows the user to calibrate at the start of the first run, at the start of every run or not at all. The signal blocks 15 and 16 are then used to define the calibration tone for channels A and B respectively:

; Calibration TONE for Channel-A
The attenuation is set to zero and, in this case, a 1kHz tone is generated in each channel separately. All levels in the Xperimeter program are specified with respect to the user defined zero dB reference and refer to “spectral level” (per hertz), not the overall level. The relationship between overall level and spectral level is given approximately by,

\[
\text{Overall Level} \approx \text{spectral level} + 10\log\text{BW}
\]

where BW is the bandwidth in hertz of the noise band.

The zero dB spectral level was therefore measured in dBSPL, and from this all other values could be referenced back to this level. This was achieved by attaching the headphone unit (TDH49) to an artificial ear acoustic coupler. The coupler was a Brüel & Kjær (B&K) type 4153 with a 0.5 inch microphone and a 2.5cm³ cavity. The headphone was fitted over the coupler so that there was no sound leakage and a weight of 550g was placed on the top to maintain the positioning. The acoustic coupler was in turn attached to a B&K type 2230 sound level meter, set to measure at 1kHz with a 1/3 octave filter.

Calibration of the sound level meter was achieved using a tone generator designed specifically for calibration purposes. It produced a tone at 1kHz of 94dBSPL. The tone generator fitted over the microphone in the acoustic coupler and the sound level meter was adjusted until it read the correct level.

The zero dB reference tone was then measured from each channel. For measurements of the auditory filter at 1kHz, zero dB was found to be 61 in channel A (from which the masker was produced) and 102.5dBSPL in channel B (which produced the probe tone). These values were found to be 63.5dBSPL and 102.5dBSPL respectively for the filter.
measurement at 2kHz. Thus, all attenuation levels were referenced back to these levels. However, there are two places where a signal can be attenuated. The programmable attenuator controls the level of the overall signal block and attenuation is applied on a per D/A channel basis. Each component of the signal block also has an attenuation level, which is applied digitally. The absolute level for a signal is therefore given by,

\[ \text{Level} = \text{Calibration Level} + \text{Programmable Attenuator Level} + \text{Component Level} \]

For each experimental set up, the system calculates a “calibration factor”. This represents the scaling of the waveforms required to utilise the full dynamic range of the 16 bit D/A converters. The system searches the program to find the highest value that may be generated, and produces a calibration factor for each channel. Therefore every time a new program was used, the above calibration procedure was carried out.

The output of the system was checked with a signal analyser. The purity of the signal and the output spectrum of the notched noise were both found to be satisfactory.

The repeatability of the test was examined in two ears. Each ear was tested 4 times. The inter-test variability was then compared to the inter-subject variability to check for consistency. The mean and standard deviations are shown in Table 3-4. The auditory filter shape measurement was found to be sufficiently repeatable in the light of the spread of bandwidths to be expected from a normal population of subjects. A graph showing the median and quartiles for the data from the two ears tested repeatedly in comparison to the data from 39 different ears, is shown in Figure 3-6.

<table>
<thead>
<tr>
<th></th>
<th>Number of tests</th>
<th>Mean 3dB bandwidth (Hz)</th>
<th>Standard deviation</th>
</tr>
</thead>
<tbody>
<tr>
<td>Ear 1</td>
<td>4</td>
<td>90.22</td>
<td>2.99</td>
</tr>
<tr>
<td>Ear 2</td>
<td>4</td>
<td>92.65</td>
<td>4.18</td>
</tr>
<tr>
<td>Whole subject group</td>
<td>39</td>
<td>117.35</td>
<td>33.50</td>
</tr>
</tbody>
</table>
3.2 Otoacoustic Emission Measurement

3.2.1 Transient Evoked Otoacoustic Emissions (TOAEs)

3.2.1.1 Equipment

The Otodynamics ILO92 otoacoustic emission measurement system was used to record TOAEs and to analyse the results. This system is described by Kemp et al. (1990).

The emission probe consisted of a transducer to produce the stimulus and a microphone (Knowles Ltd.) positioned close to the probe tip to enable accurate recording of OAE response. An air leak pipe allowed pressure equalisation. The probe sat in the ear canal enclosed by a sponge tip to minimise noise contamination. Careful positioning of the probe was required to acquire a good response. The fit of the probe in the ear canal could be examined prior to each measurement using the “checkfit” stage of the program. The ILO92 is a twin probe system and both probes were used in the test of contralateral stimulation on TOAE response. The second probe provided the contralateral stimulus.
The output box to which the probes connect was in turn connected to a card in the PC. The output box was positioned inside a sound proof room attached through the wall by cables to the PC. Using the accompanying software one could design the stimuli and recording parameters and analyse the results.

Calibration of the entire system was achieved by using a 1cc probe cavity supplied with the equipment. The probe tip was inserted into the cavity and a special synthetic OAE stimulus was generated. The stimulus consisted of three tones of different frequency. The results could then be compared from session to session in order to check that calibration had been maintained. More specific testing of the hardware could be achieved using the “TEST92” software. Firstly, the link between the computer and the ILO92 card could be tested. Given that this test is passed, the gain of the amplifier is tested to check that errors are within those specified in the documentation. The attenuators in channels A and B are then checked for accuracy across the range of attenuation required.

3.2.1.2 Stimulus Set up and Testing Procedure

Two tests were performed using TOAEs. These were the standard test for TOAE presence and a test of the contralateral stimulus effect on TOAEs.

(a) Standard test for presence of TOAEs

The standard test was performed first to give a quick idea of whether TOAE were present, since there would be little point in going through the longer test with contralateral stimulation if they were not. The stimulus was clicks since they stimulate a wide frequency range. The standard clicks that the ILO92 provides have a bandwidth of 5kHz, a duration of 80μs and a level of approximately 85dBSPL. Clicks occurred at a rate of 50 per second and were in groups of four. The first three clicks were identical, with the fourth click being inverted and three times greater than the others in magnitude (Figure 3-7). This was called the “non-linear” mode and allowed the OAE to be recorded without contamination from the stimulus or from ringing in the meatus. The responses from these linearly behaving systems are cancelled out by the summation of the four responses.

1 The auto adjust facility was used in order to provide the same stimulus level for each subject.
Following each click, sound was recorded for 2.5-20ms. 260 sweeps were recorded and averaged alternately into two buffers, A and B. When 260 sweeps were recorded that were quieter than the noise rejection threshold, the data in buffers A and B were compared. Differences in the data in A and B represented the background noise present, and the data which was present in both A and B was used to calculate the overall level of the response.

The inter-test variability was assessed by measuring the standard TOAE response from one ear repeatedly (on many different occasions), and comparing this with the responses from a group of many different ears. The mean emission from one ear tested 6 times was 10.9dB with a standard deviation of 0.2dB. The mean response from a group of 39 normal subjects was 11.9dB with a standard deviation of 4.3dB, thus showing that the inter-test errors were negligible in comparison with the spread of data one could expect from a normal population.

(b) Test of contralateral suppression of TOAEs

The test of contralateral stimulus on TOAE level was performed using both channels of the ILO92. Channel A provided the stimulus to the test ear and recorded the response, whereas channel B provided the stimulus to the contralateral ear. The stimulus used to evoke the emission was clicks or tones and the contralateral suppressing stimulus was
either white noise or tones. The contralateral stimuli were presented continuously during the ipsilateral stimulation in order to suppress the response.

Clicks were presented in the “linear” mode from the ILO92. In this mode, all four clicks are identical. Linear clicks are known to give a better signal-to-noise ratio than the non-linear clicks. However, in this mode, the contaminating probe and meatal ringing responses are not cancelled out. The level of the click was therefore reduced to 63±3dBSPL in order to reduce the presence of these unwanted responses. Thus, the same time window as in the standard test (2.5-20ms) could be studied. The stimulus was adjusted to this level for each subject individually.

The tests consisted of recordings of 60 sweeps, alternately with and without contralateral stimulation. Five sets of 60 sweeps were captured with the contralateral stimuli, and 5 without. The suppression was calculated by subtracting the total response energy (dB) with contralateral stimulation from the condition without contralateral stimulation.

Veuillet et al. (1991) used an alternative method of calculating the suppression during contralateral stimulation. Their method involved measurement of the suppression during various levels of ipsilateral stimulation with and without contralateral noise. The suppression was then expressed as the equivalent attenuation (EA) of the ipsilateral stimulus level that would give the same suppression as the contralateral sound. The EA was calculated as the mean difference between the two lines (with and without noise) as shown in Figure 3-8.

In this way, they hoped to compensate for the differences in original sizes of OAEs before suppression encountered in different subjects. However, the two lines are not parallel since greater suppression is found at lower ipsilateral intensities (Hood et al. 1996; Ryan and Kemp 1996; Veuillet et al. 1996) and therefore this method seems to confound the different growth functions of the two stimulus conditions. Therefore, in this study, since it was important to make the overall test time as short as possible, the quicker method of simply taking the difference in the response levels between the ‘with’ and the ‘without’ noise situation was used.
Past work has suggested that attentional state may affect OAEs. A decrease in TOAE amplitude was found to be maximal at 1-2kHz for a visual task and at 2-3kHz for an auditory task (Froehlich et al. 1993). For tone evoked emissions, the amplitude was found to be greatest whilst attention was directed towards the test ear (Giard et al. 1994). Due to these reported effects of attention on OAE measurements, subjects were asked to read during the test. This was thought to be the best way of ensuring equal attention levels between subjects. The subject was also requested to relax but to sit as still and quietly as possible. Reading was also found to help keep the subjects from moving around as much during the test.

The reliability of the test was assessed by measuring TOAE suppression in 8 subjects on two separate occasions (test 1 and test 2 in Figure 3-9). The suppression measured from a group of 39 normal subjects was 1.7dB, with a standard deviation of 0.9dB. It can be seen from Figure 3-9 that the inter-test variability was low in comparison, thus indicating that the test was reliable and repeatable.
3.2.2 Synchronised Spontaneous Otoacoustic Emission (SSOAE) Measurement

A test for SSOAEs was performed on each subject. The same hardware as described above to record TOAEs was used (IL092). Spontaneous otoacoustic emissions are acoustic signals that can be measured in the ear canal without an evoking stimuli. However, measurement can prove difficult unless the responses are synchronised in time. The IL092 uses this method and therefore the response is technically a synchronised spontaneous emission (SSOAE). The SSOAE recorded are therefore much more robust and clinically useful than unsynchronised spontaneous emissions.

Smaller clicks than in the case of TOAEs are used to synchronise the emissions. They have a duration of 40μs and occur every 80ms. The response is recorded between each click, except for the period immediately after the click, and a short time before the next click for data transfer (approximately 3ms). This much longer time window is averaged and a Fast Fourier Transform (FFT) is performed and displayed showing the SSOAE peaks by frequency. The frequency of the peaks could be measured, with an error of...
12.2Hz, since this is the minimum bin size. However, it has been noted (Ostergaard 1996) that the frequencies displayed by the software are actually out by 12.2Hz and are in fact 12.2Hz less than quoted. This discrepancy though has little effect on this study.

As before, 260 non-noise rejected sweeps were averaged. The presence or absence of SSOAEs was noted and also whether the frequencies of the emissions, if present, were in the vicinity of 1kHz. Peaks between 0.75 and 1.5kHz were counted as being close.

3.2.3 Distortion Product Otoacoustic Emission Measurement

3.2.3.1 Equipment

The equipment required for measurement of distortion products (DPs) was essentially the same as that described in section 3.2.3.1. for the testing of TOAEs and SSOAEs. However, the probe used for DP measurement differs in its design to that used for measurement of TOAEs and SSOAEs. The DP probe uses both channels A and B and has an extra tube contained within the probe in order that the two primary tones may be carried.

3.2.3.2 Stimulus Set up and Testing Procedure

Once the probe is placed in the ear canal the ILO92 software is started, which firstly checks that the fit is good. The ideal fit would give a flat frequency spectrum from the click stimulus. When satisfied with the fit, the program then automatically adjusts the stimulus levels by examining the response at 1kHz and then adjusting the other frequencies with respect to this by using internal probe calibration tables.

Collection of a “DP-GRAM” involves cycling through the primary frequency sequence and measurement of the distortion product 2f1 -f2. The level of f1 was 60dBSPL and the level of f2 was 54dBSPL with an f2/f1 frequency ratio of 1.21. These parameters have been found to be optimal for DPOAE production (for review see Probst et al. 1991).

The DP level as a function of frequency is built up. Data is acquired in sections of about a second at each frequency and the noise level is also assessed. The stimuli can be allowed to cycle through as many times as required to get good data and new data is pooled with the old data in order to make an average for each frequency pair. As the
data is acquired, a graph is shown which displays not only the DP level with frequency but the level of the noise (Figure 3-10). The solid shaded areas show the noise contamination and the hatched area shows the statistical 2 standard deviation limit of the noise.

Figure 3-10: Example Distortion Product Gram

Figure 3-10 shows a standard DP-gram which sweeps a wide range of frequencies and measures at 4 points per octave. The fine structure of the DP was also measured around 1kHz since this was the frequency of interest in the rest of the experiment. In order to examine the fine structure, the frequency range swept was limited to 0.75-1.5kHz and 17 points were tested.

The subjects were instructed to sit quietly and relax, as with the TOAE measurement. Again, the subjects were asked to read in order to keep attentional state as equal as possible between subjects.

The inter-test variability was low, as shown by Figure 3-11. This shows the DPgram measured from the same ear on two separate occasions. The DP amplitudes are very similar showing high reliability.
Figure 3-11: Repeat DPGram measurements on the same ear showing good inter-test reliability.

(a) Standard tests for DPOAEs

Two standard tests were used in these experiments in order to examine the amplitude of DPOAEs. As mentioned, the DPGram and the fine structure were both tested with the stimulus parameters above.

(b) Contralateral suppression of DPOAEs

The two standard tests were also both carried out in the presence of contralateral stimuli in order to examine the effect of the efferent system activation via the MOCB.

Contralateral sound stimulation was presented via TDH39 headphones attached to a calibrated audiometer. The contralateral stimuli were presented at a level of 50dBHL. The stimuli were either continuous third octave narrow band noise centred at 1kHz or tones (1kHz). The other earphone of the headphone set was placed in a comfortable position away from the ear in which the DPs were being recorded.
The DP amplitude recorded during contralateral stimulation was subtracted from that recorded without contralateral stimulation in order to examine the effect that the efferent system had.

The DP levels were then recorded again without contralateral stimulation to check for stability.

### 3.3 Pure Tone Audiometry

Pure tone audiometry was carried out using the British Society of Audiology’s recommended procedure (Audiology 1981). For the pure tone audiogram (PTA) ‘normal’ was defined as thresholds better than 20dBHL for frequencies between 250Hz and 8kHz at octave intervals.

### 3.4 Tympanogram

Tympanometry was also carried out following the British Society of Audiology’s recommended procedure (Audiology 1992). This procedure allowed the mobility of the ear drum to be measured along with the middle ear pressure. The technique involved fitting an air-tight probe into the outer ear and sweeping the pressure from +200daPa to -400daPa at a rate of 50daPa/s. A probe tone of 226Hz was used and the impedance of the ear drum could therefore be assessed with respect to the amount of energy reflected back into the ear canal. The reciprocal of the impedance is the admittance of the eardrum and this is directly proportional to the compliance. A peak of eardrum compliance could be found at the point where the pressure in the outer ear matches that of the middle ear. Unusually poor OAE levels can be due to the eardrum being of abnormal mobility, and therefore it is important to carry out this test. ‘Normal’ tympanometry was defined as eardrum compliance between 0.3 and 1.6 cm$^3$ and middle ear pressure between -50 and +50daPa.
3.5 Acoustic Reflex Thresholds

The acoustic reflex is the term used to describe the involuntary contraction of two middle ear muscles (the stapedius and the tensor tympani muscles) in response to auditory stimuli, vocalisation, tactile stimulation or body movement. Generally, the reflex contraction of the stapedius muscle occurs above a certain intensity threshold for acoustic stimuli. The stapedius muscle connects the wall of the middle ear to the stapes, the most medial of the bones in the ossicular chain. The tensor tympani muscle is attached to the malleus near the tympanic membrane. Contraction of the muscles causes stiffening of the ossicular chain which produces a reduction in the transmission of low frequency sounds from the outer ear to the cochlea.

The stapedius muscle is innervated by the facial or seventh cranial nerve and the tensor tympani muscle is innervated by the trigeminal or fifth cranial nerve. The reflex arc consists of:

1. The cochlear nerve (VIII) from the hair cells to the cochlear nucleus
2. Projection to the medial superior olivary complex
3. Motor neurons to the muscles themselves

The contralateral pathway also consists of another synapse in the medial superior olivary complex. Thus, testing both ipsi and contralateral acoustic reflexes gives an idea of the integrity of this whole pathway (Stach 1987). Existence of any lesion affecting the pathway in a subject would rule them out from being considered normal in this study.

The procedure for testing the acoustic reflex utilises the same principle as that used to measure the tympanogram i.e. measurement of the change in the acoustic impedance of the eardrum. The probe tone used was 226Hz as before. The reflexes were measured at 0.5, 1, 2 and 4kHz. So, as the reflex was elicited the ossicular chain stiffens and the impedance (and therefore the compliance) of the ear drum changes. Thus, the lowest level of stimuli at each frequency that gave a repeatable change in the compliance was considered to be the acoustic reflex threshold.

Ipsilateral and contralateral acoustic reflex thresholds were analysed as normal with regard to the levels as defined by Prasher and Cohen (1993).
Chapter 4

Plan of Experiments
The experiments performed have been divided into the five chapters that follow. The first experiment (Chapter 5) was carried out in order to examine the initial basic hypothesis that the efferent system may be involved in frequency selectivity. This experiment looked at whether the 'strength' of efferent action in a particular subject was related to the cochlear frequency selectivity. This was carried out at 1kHz. Efferent action 'strength' was assessed by measurement of the contralateral suppression of otoacoustic emissions. Both transient, and the more frequency specific distortion products, were tested, as were OAEs of both types without contralateral stimulation and SOAEs. These latter tests were performed in order to investigate whether general activity of the cochlea was also implicated. Frequency selectivity was assessed using the notched noise psychoacoustic test described earlier to estimate the shape of the auditory filter.

The next experiment investigated the possibility that the efferent system might only play a role in frequency selectivity when actually activated. The filter shape was thus tested during contralateral white noise stimulation. However, firstly it was important to examine the timing of the onset of the contralateral efferent effect (Chapter 6). The results from this experiment were useful for two reasons. Firstly, they allowed the timing of the contralateral stimuli to be set in the experiments that followed so that one could be sure the efferent system was effective during the filter shape measurement. Secondly, the results threw light on whether the timing of the effect would allow it to be at all effective in frequency selectivity.

Chapter 7 describes the experiment that investigated what effect contralateral white noise had on the shape of the auditory filter at 1kHz, and whether the changes due to the white noise, were related to the activity of the efferent system. The subsequent experiment (Chapter 8) was a more detailed analysis, whereby various narrow band noises were used as the contralateral stimuli. It was hoped that this might mimic the auditory stimuli situations encountered in the environment more realistically.

Finally, Chapter 9 examined the auditory filter at 2kHz and its relation to efferent activity. All the preceding tests described had been involved in testing the filter shape at 1kHz. Therefore, in order to check whether the level of correspondence between the
OAE and the psychoacoustic tests was consistent across frequency this final experiment examined the relationship at 2kHz.
Chapter 5

Investigation of the Possible Link between Auditory Filter Shape at 1kHz and Otoacoustic Emission Magnitude and Suppression.
5. Investigation of the Possible Link between Auditory Filter Shape at 1kHz and Otoacoustic Emission Magnitude and Suppression.

5.1 Introduction

Given the hypothesis that the efferent system may be involved in tuning in the cochlea via the action of the outer hair cells, in this chapter we investigate whether this may be demonstrated in normal human subjects. The experiment involved testing subjects to see whether there is any relation between the degree of efferent activity and the shape of the auditory filter. The aim was to investigate whether those subjects with a high degree of efferent activity, measured via the contralateral suppression of otoacoustic emissions, had narrower auditory filters and therefore better tuning abilities. The auditory filters were measured using the psychoacoustic technique of notched noise masking.

5.2 Method

5.2.1 Subjects

Subjects were selected from those people responding to a notice placed in the university. Most subjects were therefore undergraduate or postgraduate students who were naive to the testing being carried out. Only subjects with normal hearing thresholds (see section 3.3) and tympanometry (see sections 3.4 and 3.5) were included in the study. Thirty nine subjects, aged between 18 and 35 years (mean age 23.8, standard deviation 4.0) were tested. Of this group, 21 were female and 18 male. The ear to be tested was selected randomly, which resulted in 21 right ears and 18 left ears being tested. This randomised ear selection was carried out because McFadden (1993) had found that there is an asymmetry in the functioning of the efferent system.

Aspirin has also been found to affect the frequency selectivity of the human auditory system in the past. One study showed that filters measured using a notched noise psycho-acoustic technique were broader when aspirin was administered (Carlyon and Butt 1993) and another study found changes in the resonant frequency of DPOAE tuning.
curves (Brown et al. 1993b). In this study therefore, subjects were asked not to take aspirin prior to the experiment.

5.2.2 Measurement of Auditory Filter Shape at 1kHz

Each subject’s auditory filter shape was estimated using the technique described in 3.1.2.

5.2.3 Transient Otoacoustic Emission (TOAE) Measurement

Both the standard test for presence of TOAEs, and the contralateral suppression of TOAEs, were performed on each subject and have been described earlier (see section 3.2.1.2).

In this experiment, a number of different stimuli were used to evoke and suppress the OAE. Four tests were performed using different stimulus combinations (Table 5-1).

Table 5-1: Combinations of stimuli tested with TOAEs

<table>
<thead>
<tr>
<th>Test</th>
<th>Ipsilateral Ear Stimulus</th>
<th>Contralateral Ear Stimulus</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Click</td>
<td>White noise</td>
</tr>
<tr>
<td>2</td>
<td>Click</td>
<td>1 kHz tone</td>
</tr>
<tr>
<td>3</td>
<td>1 kHz tone pips</td>
<td>1 kHz tone</td>
</tr>
<tr>
<td>4</td>
<td>1 kHz tone pips</td>
<td>White noise</td>
</tr>
</tbody>
</table>

The contralateral white noise and 1kHz tone were presented continuously throughout ipsilateral recording. The threshold to both stimuli were measured for each subject and the levels accordingly set to 40 and 50dBSL for the white noise and the 1kHz tone respectively. These levels were selected to be well below normal acoustic reflex thresholds (approximately 70dBHL) and to minimise cross-over via air or bone conduction. The white noise was chosen since it stimulates a broad area of the cochlea and therefore should produce the maximum suppression. The 1kHz tone was chosen so that only efferents responding to this frequency would be activated which may have more relevance to the test of auditory filter shape at 1kHz.
Tone evoked emissions were also recorded at 1kHz. This concentration of the ipsilateral energy at this frequency was also tested since the results were being compared to the auditory filter shape which was also measured at 1kHz. It was thought that more specific testing of the cochlea might eliminate some variations in the results comparing the TOAE and filter shape measurements. These variations may occur if efferent function is not uniform over the length of the cochlea.

In order to keep the time taken to perform the test to a minimum the four different combinations of stimuli were used in the order shown in Table 5-1. This sequence allowed the fewest number of adjustments to be made to set up the stimuli between each test, therefore keeping the total testing time to a minimum.

5.2.4 Synchronised Spontaneous Otoacoustic Emission (SSOAE) Measurement

Each subject was tested for the presence of synchronised spontaneous otoacoustic emissions. The equipment and methods used have been described in section 3.2.2.

5.2.5 Distortion Product Otoacoustic Emission Measurement

The DP-gram and fine structure at 1kHz of 26 normal subjects were measured. Fifteen subjects were also tested with a contralateral tone of 1kHz and twelve subjects were tested with contralateral narrow band noise at 1kHz. The equipment and methods used are described in section 3.2.3.

5.3 Results

The mean 3dB bandwidth of the auditory filter at 1kHz for the 39 subjects tested was 117.35Hz (standard deviation 33.50Hz, range 75.47-188.91Hz).\(^1\) For all subjects the bandwidth of the filter was compared to the magnitude and suppression of OAEs in different conditions. These results are presented in sections 5.3.1 to 5.3.6. The results of the comparisons of more detailed parameters describing the filter shape to the OAE results are presented in section 5.3.7. Finally, other factors that may influence the results are discussed.

\(^1\) Similar results were noted by Glasberg and Moore (1990), who found an ERB of 133Hz at 1kHz
The distribution of the 3dB bandwidth data was tested with the Kolmogorov-Smirnov test. The Z-statistic was 1.367 and the asymptotic 2-tailed significance level was 0.048 showing that the data was significantly different from a normal distribution. Therefore, non-parametric tests were used when examining the bandwidth data.

### 5.3.1 TOAE magnitude

The mean TOAE response magnitude for the 39 subjects tested in this experiment was 11.88dBSPL (s.d.=4.27, range 4.30-21.50dB). The response was not found to be correlated with the 3dB bandwidth (correlation coefficient=0.046, significance = 0.783).

### 5.3.2 TOAE suppression

The results for the four different suppression tests carried out are summarised in Table 5-2. The table shows the number of subjects tested (one subject was not tested on the last 2 conditions) and the mean suppression for each condition with its standard deviation.

<table>
<thead>
<tr>
<th>Suppression Test</th>
<th>n</th>
<th>Mean suppression dB (standard deviation)</th>
<th>1 sample t-test (test value=0) p value</th>
<th>Correlation with Auditory Filter Bandwidth</th>
</tr>
</thead>
<tbody>
<tr>
<td>Ipsi click-Contra white noise</td>
<td>39</td>
<td>1.65 (0.85)</td>
<td>&lt;0.001</td>
<td>-0.118</td>
</tr>
<tr>
<td>Ipsi click-Contra 1kHz tone</td>
<td>39</td>
<td>0.37 (0.34)</td>
<td>&lt;0.001</td>
<td>0.047</td>
</tr>
<tr>
<td>Ipsi 1kHz tone – contra white noise</td>
<td>38</td>
<td>0.57 (0.50)</td>
<td>&lt;0.001</td>
<td>-0.099</td>
</tr>
<tr>
<td>Ipsi 1kHz tone – contra 1kHz tone</td>
<td>38</td>
<td>0.16 (0.18)</td>
<td>&lt;0.001</td>
<td>-0.224</td>
</tr>
</tbody>
</table>

In order to check that in each case there was actually a measurable suppression the one-sample t-test was carried out with a test value of zero. All four conditions were found to have distributions with p values less than 0.001, and therefore were significantly greater than zero (Figure 5-1).
The data was then compared to the 3dB bandwidth of the auditory filters at 1kHz in the same subjects. The non-parametric Spearman’s rho correlation coefficient (shown in Table 5-2) was calculated, to check for a linear dependency, along with the 2-tailed significance value of the correlation. There were no statistically significant linear correlation coefficients between any of the OAE stimulus results and the 3dB bandwidth of the auditory filter. A graph of the suppression of click evoked TOAEs by contralateral white noise and its relationship to the 3dB bandwidth of the auditory filter is shown in Figure 5-2.
The graph also shows little evidence of any non-linear dependence. The other three suppression combinations are also shown as scatterplots with 3dB bandwidth, and likewise, no non-linear dependence is obvious (Figure 5-3 for clicks suppressed by tones, Figure 5-4 for tones suppressed by white noise and Figure 5-5 for tones suppressed by tones).
Figure 5-4: Scatterplot of the suppression of 1kHz tone evoked TOAEs by white noise versus 3dB bandwidth of the auditory filter

Figure 5-5: Scatterplot of suppression of 1kHz tone evoked TOAEs by 1kHz tones versus 3dB bandwidth of the auditory filter

The repeatability of the TOAE suppression test (clicks and white noise) was measured in 8 ears as discussed in section 3.2.1.2(b). The suppression was measured on two separate occasions. Using the paired sample t-test to check for similarity between the two tests, it was found that there was no significant difference between the suppression
results from the two occasions and the variability was low in comparison to the population variability.

5.3.3 TOAE magnitude and suppression in specific frequency bands

The suppression of TOAEs was examined in narrower frequency bands as well as looking at the overall response as in section 5.3.2. This was in order to take account of the possibility that the efferent system may not be equally effective at all frequencies. Since the auditory filter was measured at 1kHz, the frequency regions of the OAE responses around 1kHz and around 2kHz were examined in more detail.

The response from the standard TOAE test was divided into half octave frequency bands and the response falling into the bands around 1 and 2kHz was compared to the auditory filter bandwidth in the same subjects. For the suppression measurements, the averaged responses with contralateral stimulation and without, were filtered. The difference between the responses in the two conditions after filtering was then calculated. The first bandpass filter cut out frequencies apart from those between 781 and 1513Hz and the second filter cut out everything except a bandpass range of 1513-2978Hz (i.e. approximately octave filters centred at 1kHz and 2kHz).

The Spearman’s rho bivariate correlation coefficients are shown in Table 5-3, for the relationship between the 3dB bandwidth and the filtered responses from the standard TOAE test and the suppression of TOAEs.

Table 5-3: Correlation between 3dB bandwidth, and TOAE standard response and suppression, after filtering

<table>
<thead>
<tr>
<th>Stimuli</th>
<th>1kHz region</th>
<th>2kHz region</th>
</tr>
</thead>
<tbody>
<tr>
<td>Click evoked TOAE-standard test</td>
<td>0.082</td>
<td>-0.003</td>
</tr>
<tr>
<td>Ipsil click-Contra white noise</td>
<td>0.001</td>
<td>-0.158</td>
</tr>
<tr>
<td>Ipsil click-Contra 1kHz tone</td>
<td>0.112</td>
<td>0.144</td>
</tr>
<tr>
<td>Ipsil 1kHz tone – contra white noise</td>
<td>-0.191</td>
<td>-0.184</td>
</tr>
<tr>
<td>Ipsil 1kHz tone – contra 1kHz tone</td>
<td>0.094</td>
<td>-0.108</td>
</tr>
</tbody>
</table>
The results show no statistically significant linear correlation between the responses in these frequency regions and the auditory filter bandwidth.

5.3.4 Temporally windowed TOAE suppression

It has been shown that increased levels of suppression, of the order of 3-4dB, can be observed by analysing the emission only in the later parts of the recording period (Berlin et al. 1993b). With the hypothesis that this may emphasise differences between individual suppression levels, the results were further analysed by restricting the time window to only take into account data recorded 10-20ms after the onset of the stimulus. At these latencies, the main frequencies contained within the response tend to be those up to about 2kHz (Tognola et al. 1997). These are also the predominant components of the overall response and the frequencies that are transmitted best by the middle ear, so therefore windowing the response can be thought of as similar to filtering the response. Windowing also had the advantage of eliminating any contamination from stimulus artefact that may have been present early in the response. The suppression of the response in the same frequency zones (751-1513 and 1513-2978Hz) as before was examined.

The Spearman's rho correlation coefficients are shown in Table 5-4 for the data filtered into the two frequency bands. The correlation coefficients show the relationship between the TOAE suppression and the 3dB bandwidth of the auditory filter in each case.

<table>
<thead>
<tr>
<th>Stimuli</th>
<th>751-1513Hz</th>
<th>1513-2978Hz</th>
</tr>
</thead>
<tbody>
<tr>
<td>Ipsi click - Contra white noise</td>
<td>0.070</td>
<td>-0.007</td>
</tr>
<tr>
<td>Ipsi click - Contra 1kHz tone</td>
<td>0.151</td>
<td>0.097</td>
</tr>
<tr>
<td>Ipsi 1kHz tone – contra 1kHz tone</td>
<td>-0.134</td>
<td>0.106</td>
</tr>
<tr>
<td>Ipsi 1kHz tone – contra white noise</td>
<td>-0.107</td>
<td>-0.012</td>
</tr>
</tbody>
</table>

Again, there appeared to be no statistically significant linear relationship between the degree of suppression in the last 10ms of the response and the 3dB bandwidth of the auditory filter.
5.3.5 SOAEs

The subjects were divided into two groups depending on whether they had SOAEs or not. Of the 39 subjects tested, 19 were found to have SOAEs in the test ear. The 3dB bandwidth of the auditory filter was compared in the two groups to see whether the presence of SOAEs was related. The groups were compared with the independent samples t-test because the distribution of the bandwidths in both groups was found to be normal (Kolmogorov-Smirnov test) and the variances could be assumed to be equal (Levene's Test). The mean bandwidth for the group of subjects without SOAEs was 113.80Hz (s.d=33.38) and for those with SOAEs it was 121.08Hz (s.d=34.14). However, this difference was not found to be significant (p=0.505) at the p=0.05 point. A boxplot of the comparison in 3dB bandwidth between the two groups is displayed in Figure 5-6, where the median, quartiles and extreme values are shown for each group.

Figure 5-6: Boxplot comparing the 3dB bandwidth of the auditory filter in subjects with and without spontaneous otoacoustic emissions (SOAEs)

The scatterplot of the bandwidth of the auditory filter versus the suppression of click evoked TOAEs by contralateral white noise (Figure 5-2) was examined with regard to the distribution of the subjects with and without SOAEs. The data is shown again in
Examining the prevalence of SOAEs with frequency, especially around 1kHz, it was found that in all but one of the subjects who had SOAEs, they occurred in the octave band around 1kHz. Making the assumption that only those SOAEs in the vicinity of the frequency of the auditory filter being tested could affect it, two groups were formed containing those subject with SOAEs around 1kHz (n=18) and those without (n=21). This made little difference to the results from the previous two groups. The mean bandwidth in subjects with SOAEs around 1kHz was 120.8Hz and for those without it was 114.4Hz. Again this difference was not statistically significant (p=0.553).
5.3.6 DPOAEs

5.3.6.1 DP magnitude

DP-grams spanning a wide range of frequencies and the fine structure around 1kHz were measured from 26 subjects. In order to compare the DP results to the filter bandwidth, 7 measures of the DPs were examined. The 7 measures were:

a) The level of the DP in dB when \( f_2 = 1\)kHz (since the site of DP production is thought to be nearest to \( f_2 \) as discussed in section 2.2.1.3)

b) The level of the DP when \( f_2 = 2\)kHz

c) The mean DP response from all the frequencies of the fine structure sweep. This gave an overall DP level in the 1kHz region (\( f_2 = 745\) - 1501Hz).

d) The mean of the fine structure sweep responses below 1kHz (\( f_2 = 745\)Hz - 977Hz)

e) The mean of the fine structure sweep responses above 1kHz (\( f_2 = 1013\)Hz - 1501Hz)

f) The difference between the mean high frequency response level and the mean low frequency response (i.e. (e)-(d) above). This gave a measure of the general trend of the DP response in the 1kHz region.

g) The standard deviation of the DP level in the fine structure sweep. This was taken as a measure of the flatness of the response in the 1kHz region.

The 3dB auditory filter bandwidth and the 7 DP measures above were all found to follow a normal distribution. Therefore, the parametric measure of correlation (Pearson's correlation coefficient) was calculated when comparing the DP measures with the filter bandwidth. The results of this comparison are shown in Table 5-5. The greatest correlation with the filter bandwidth is the level of the DP at 1kHz. However, this correlation, along with all of the other results, is not statistically significant at the p<0.05 point.
Table 5-5: Correlation of DP measures and 3dB bandwidth of auditory filter

<table>
<thead>
<tr>
<th>DP Measurement (dB)</th>
<th>Mean (dB)</th>
<th>Standard Deviation</th>
<th>Correlation Coefficient</th>
</tr>
</thead>
<tbody>
<tr>
<td>DP at 1kHz</td>
<td>3.59</td>
<td>8.45</td>
<td>0.363</td>
</tr>
<tr>
<td>DP at 2kHz</td>
<td>6.45</td>
<td>6.93</td>
<td>0.178</td>
</tr>
<tr>
<td>Mean (745-1501Hz)</td>
<td>4.94</td>
<td>6.02</td>
<td>0.127</td>
</tr>
<tr>
<td>Low Mean (745-977Hz)</td>
<td>3.53</td>
<td>5.39</td>
<td>0.132</td>
</tr>
<tr>
<td>High Mean (1013-1501Hz)</td>
<td>5.92</td>
<td>6.89</td>
<td>0.117</td>
</tr>
<tr>
<td>High - Low</td>
<td>2.56</td>
<td>4.08</td>
<td>-0.030</td>
</tr>
<tr>
<td>Standard Deviation</td>
<td>3.49</td>
<td>1.14</td>
<td>0.131</td>
</tr>
</tbody>
</table>

A few DP levels were below the noise floor. In these cases, it was decided to use the level of the limit of the noise floor (at 2 standard deviations) instead. Obviously, the actual DP level is at some point below the noise floor, and therefore we do not know its value. However, if these points were excluded, it would give a false estimation of the overall DP level (e.g. the mean would be too high). Although the noise floor values used are not the correct DP values, they were therefore used as a compromise solution to enable the most accurate estimation of DP level to be made.

5.3.6.2 DP suppression

The suppression was measured with contralateral narrow band noise at 1kHz and a contralateral tone at 1kHz. If the DP level dropped below the noise floor at particular frequencies on either the ‘with’ or ‘without’ contralateral stimulation condition, the suppression value was excluded since it was likely to be inaccurate. Therefore, the number of valid measurements varies at each frequency.

The mean and standard deviation of the suppression of the 2f₁-f₂ distortion product ‘DPGram’ by a contralateral 1kHz tone, are shown for each f₂ frequency measured, in Table 5-6. The number of ears tested at each frequency is also shown (n).
Table 5-6: Suppression of 2f₁-f₂ distortion products by a contralateral 1kHz tone - DPGram

<table>
<thead>
<tr>
<th>F₂ Frequency (Hz)</th>
<th>n</th>
<th>Mean Suppression dB (s.d.)</th>
<th>1 sample t-test (test value=0) p value</th>
<th>Correlation with 3dB auditory filter bandwidth</th>
</tr>
</thead>
<tbody>
<tr>
<td>708</td>
<td>14</td>
<td>-0.37 (2.24)</td>
<td>0.545</td>
<td>0.039</td>
</tr>
<tr>
<td>842</td>
<td>15</td>
<td>1.05 (1.21)</td>
<td>0.005*</td>
<td>-0.179</td>
</tr>
<tr>
<td>1001</td>
<td>12</td>
<td>0.22 (1.46)</td>
<td>0.618</td>
<td>0.259</td>
</tr>
<tr>
<td>1184</td>
<td>14</td>
<td>0.26 (3.35)</td>
<td>0.778</td>
<td>0.470</td>
</tr>
<tr>
<td>1416</td>
<td>15</td>
<td>0.56 (1.78)</td>
<td>0.243</td>
<td>0.510</td>
</tr>
<tr>
<td>1685</td>
<td>14</td>
<td>0.48 (1.79)</td>
<td>0.335</td>
<td>0.355</td>
</tr>
<tr>
<td>2002</td>
<td>15</td>
<td>1.39 (1.81)</td>
<td>0.010*</td>
<td>0.375</td>
</tr>
<tr>
<td>2380</td>
<td>15</td>
<td>1.21 (1.34)</td>
<td>0.004*</td>
<td>0.285</td>
</tr>
<tr>
<td>2832</td>
<td>15</td>
<td>0.53 (2.01)</td>
<td>0.321</td>
<td>0.038</td>
</tr>
<tr>
<td>3369</td>
<td>15</td>
<td>0.33 (1.71)</td>
<td>0.463</td>
<td>-0.194</td>
</tr>
<tr>
<td>4004</td>
<td>14</td>
<td>0.31 (1.84)</td>
<td>0.543</td>
<td>0.117</td>
</tr>
<tr>
<td>4761</td>
<td>13</td>
<td>0.02 (1.54)</td>
<td>0.958</td>
<td>0.535</td>
</tr>
<tr>
<td>5552</td>
<td>13</td>
<td>-0.53 (2.55)</td>
<td>0.467</td>
<td>0.487</td>
</tr>
<tr>
<td>6299</td>
<td>15</td>
<td>-0.42 (1.99)</td>
<td>0.428</td>
<td>0.177</td>
</tr>
</tbody>
</table>

The suppression levels were low overall and the only frequencies with suppression that was statistically significant (P<0.05) for the group were 842, 2002 and 2380Hz. The mean suppression levels along with the 95% confidence intervals for each frequency are shown in Figure 5-8.

Figure 5-8: Mean suppression of 2f₁-f₂ distortion product by contralateral 1kHz tone – DPGram (showing 95% confidence intervals)
The suppression was compared to the bandwidth of the auditory filter for the subjects tested. The correlation was calculated at each f2 frequency. The suppression data as well as the filter bandwidth for this group of subjects were all found to follow a normal distribution and therefore Pearson’s correlation coefficients were calculated. The correlation coefficients are shown in Table 5-6. The correlation coefficients were, in general, found to be positive, implying that a higher degree of suppression was associated with a wider bandwidth. However, none were statistically significant.

Table 5-7 shows the results for the suppression from a contralateral 1kHz tone when the fine structure of the DPgram is examined at the frequencies around 1kHz.

**Table 5-7: Suppression of 2f1-f2 distortion products by a contralateral 1kHz tone – Fine structure around 1kHz**

<table>
<thead>
<tr>
<th>F2 Frequency (Hz)</th>
<th>n</th>
<th>Mean Suppression dB (s.d.)</th>
<th>1 sample T-test (test value=0) p value</th>
<th>Correlation with 3dB auditory filter bandwidth</th>
</tr>
</thead>
<tbody>
<tr>
<td>745</td>
<td>12</td>
<td>1.58 (2.44)</td>
<td>0.047* (0.036)</td>
<td></td>
</tr>
<tr>
<td>781</td>
<td>14</td>
<td>0.27 (1.91)</td>
<td>0.603 (0.052)</td>
<td></td>
</tr>
<tr>
<td>818</td>
<td>14</td>
<td>0.84 (2.06)</td>
<td>0.149 (0.029)</td>
<td></td>
</tr>
<tr>
<td>854</td>
<td>14</td>
<td>0.20 (2.29)</td>
<td>0.749 (0.029)</td>
<td></td>
</tr>
<tr>
<td>891</td>
<td>14</td>
<td>0.79 (1.93)</td>
<td>0.147 (0.104)</td>
<td></td>
</tr>
<tr>
<td>928</td>
<td>14</td>
<td>-0.15 (1.52)</td>
<td>0.717 (0.094)</td>
<td></td>
</tr>
<tr>
<td>977</td>
<td>12</td>
<td>-0.27 (1.03)</td>
<td>0.387 (0.043)</td>
<td></td>
</tr>
<tr>
<td>1013</td>
<td>13</td>
<td>-0.06 (1.74)</td>
<td>0.900 (0.044)</td>
<td></td>
</tr>
<tr>
<td>1062</td>
<td>13</td>
<td>0.87 (1.03)</td>
<td>0.010* (0.236)</td>
<td></td>
</tr>
<tr>
<td>1111</td>
<td>14</td>
<td>0.29 (2.73)</td>
<td>0.701 (0.201)</td>
<td></td>
</tr>
<tr>
<td>1160</td>
<td>14</td>
<td>-0.35 (1.87)</td>
<td>0.496 (0.086)</td>
<td></td>
</tr>
<tr>
<td>1208</td>
<td>13</td>
<td>-0.36 (1.82)</td>
<td>0.487 (0.380)</td>
<td></td>
</tr>
<tr>
<td>1257</td>
<td>15</td>
<td>-0.08 (2.04)</td>
<td>0.881 (0.578*)</td>
<td></td>
</tr>
<tr>
<td>1318</td>
<td>14</td>
<td>0.05 (2.82)</td>
<td>0.948 (0.335)</td>
<td></td>
</tr>
<tr>
<td>1379</td>
<td>15</td>
<td>1.41 (3.85)</td>
<td>0.179 (-0.296)</td>
<td></td>
</tr>
<tr>
<td>1440</td>
<td>14</td>
<td>0.76 (2.16)</td>
<td>0.207 (0.096)</td>
<td></td>
</tr>
<tr>
<td>1501</td>
<td>15</td>
<td>-0.17 (1.28)</td>
<td>0.607 (0.291)</td>
<td></td>
</tr>
</tbody>
</table>

The 1 sample t-test was used to check whether the suppression was significantly greater than zero. This was found to be the case only at f2 frequencies of 745 and 1062Hz.

Figure 5-9 shows the mean suppression for the group and the 95% confidence intervals.

* significant at p<0.05
The Pearson correlation coefficients are shown in Table 5-7 for the comparison between the suppression at the frequencies examined in the fine structure and the 3dB bandwidth of the auditory filter in the same subjects. There was a statistically significant positive correlation at one frequency (1257Hz). The correlation coefficient was 0.578 and the p value was 0.024. This is statistically significant at the p<0.05 level, but since there are multiple comparisons the result should be viewed cautiously. The Bonferroni test suggests that the p value that one considers to be significant, should be divided by the number of comparisons. Therefore here, 17 comparisons are made and p becomes 0.05/17≈0.003. This makes the criteria for accepting a significant correlation much stricter and casts some doubt over the validity of this one significant result at 1257Hz.

DP suppression was then tested with contralateral narrow band noise at 1kHz to see if this was a more effective suppresser. The results are shown in Table 5-8 for the standard DPGram frequencies.
Table 5-8: Suppression of 2f1-f2 distortion product by contralateral 1kHz narrow band noise - DPGram

<table>
<thead>
<tr>
<th>F2 Frequency (Hz)</th>
<th>n</th>
<th>Mean Suppression dB (s.d.)</th>
<th>1 sample T-test (test value=0) p value</th>
<th>Correlation with 3dB auditory filter bandwidth</th>
</tr>
</thead>
<tbody>
<tr>
<td>708</td>
<td>10</td>
<td>0.67 (2.84)</td>
<td>0.475</td>
<td>-0.238</td>
</tr>
<tr>
<td>842</td>
<td>11</td>
<td>0.74 (2.00)</td>
<td>0.250</td>
<td>0.002</td>
</tr>
<tr>
<td>1001</td>
<td>12</td>
<td>0.61 (1.38)</td>
<td>0.154</td>
<td>0.366</td>
</tr>
<tr>
<td>1184</td>
<td>12</td>
<td>1.16 (1.28)</td>
<td>0.010*</td>
<td>-0.258</td>
</tr>
<tr>
<td>1416</td>
<td>12</td>
<td>-0.74 (3.28)</td>
<td>0.450</td>
<td>0.373</td>
</tr>
<tr>
<td>1685</td>
<td>12</td>
<td>0.08 (1.86)</td>
<td>0.880</td>
<td>0.460</td>
</tr>
<tr>
<td>2002</td>
<td>12</td>
<td>0.33 (1.73)</td>
<td>0.519</td>
<td>0.436</td>
</tr>
<tr>
<td>2380</td>
<td>12</td>
<td>0.04 (1.21)</td>
<td>0.907</td>
<td>0.179</td>
</tr>
<tr>
<td>2832</td>
<td>12</td>
<td>0.12 (1.75)</td>
<td>0.822</td>
<td>-0.149</td>
</tr>
<tr>
<td>3369</td>
<td>12</td>
<td>-0.04 (1.62)</td>
<td>0.931</td>
<td>0.433</td>
</tr>
<tr>
<td>4004</td>
<td>11</td>
<td>-0.03 (1.10)</td>
<td>0.936</td>
<td>0.550</td>
</tr>
<tr>
<td>4761</td>
<td>10</td>
<td>0.02 (2.37)</td>
<td>0.979</td>
<td>-0.019</td>
</tr>
<tr>
<td>5652</td>
<td>11</td>
<td>0.75 (2.18)</td>
<td>0.278</td>
<td>0.380</td>
</tr>
<tr>
<td>6299</td>
<td>11</td>
<td>-0.29 (2.52)</td>
<td>0.710</td>
<td>-0.060</td>
</tr>
</tbody>
</table>

The only frequency with statistically significant suppression is 1184Hz, above the centre frequency of the narrow band noise. The mean suppression of the group and the 95% confidence intervals are shown in Figure 5-10.

Figure 5-10: Mean suppression of 2f1-f2 distortion product by contralateral narrowband noise - DPGram (showing 95% confidence intervals)
Comparing the suppression at each frequency with the 3dB bandwidth showed no significant correlation at any frequency.

The results for the fine structure measurements of suppression with a narrowband noise suppresser are shown in Table 5-9.

**Table 5-9: Suppression of 2f1-f2 distortion product by contralateral 1kHz narrow band noise – fine structure around 1kHz**

<table>
<thead>
<tr>
<th>F2 Frequency (Hz)</th>
<th>n</th>
<th>Mean Suppression dB (s.d)</th>
<th>1 sample t-test (test value=0) p value</th>
<th>Correlation with 3dB auditory filter bandwidth</th>
</tr>
</thead>
<tbody>
<tr>
<td>745</td>
<td>10</td>
<td>-0.32 (3.00)</td>
<td>0.744</td>
<td>-0.197</td>
</tr>
<tr>
<td>781</td>
<td>12</td>
<td>-0.46 (2.41)</td>
<td>0.524</td>
<td>0.353</td>
</tr>
<tr>
<td>818</td>
<td>12</td>
<td>0.87 (2.00)</td>
<td>0.162</td>
<td>0.341</td>
</tr>
<tr>
<td>854</td>
<td>11</td>
<td>0.64 (3.14)</td>
<td>0.516</td>
<td>-0.423</td>
</tr>
<tr>
<td>891</td>
<td>11</td>
<td>0.58 (1.55)</td>
<td>0.241</td>
<td>-0.020</td>
</tr>
<tr>
<td>928</td>
<td>11</td>
<td>-0.48 (1.64)</td>
<td>0.352</td>
<td>-0.109</td>
</tr>
<tr>
<td>977</td>
<td>11</td>
<td>-0.84 (1.58)</td>
<td>0.109</td>
<td>0.196</td>
</tr>
<tr>
<td>1013</td>
<td>12</td>
<td>0.08 (2.21)</td>
<td>0.898</td>
<td>-0.069</td>
</tr>
<tr>
<td>1062</td>
<td>11</td>
<td>0.91 (1.84)</td>
<td>0.132</td>
<td>0.163</td>
</tr>
<tr>
<td>1111</td>
<td>12</td>
<td>-0.27 (1.83)</td>
<td>0.623</td>
<td>0.441</td>
</tr>
<tr>
<td>1160</td>
<td>12</td>
<td>0.78 (2.17)</td>
<td>0.238</td>
<td>0.056</td>
</tr>
<tr>
<td>1208</td>
<td>12</td>
<td>0.63 (2.47)</td>
<td>0.399</td>
<td>0.530</td>
</tr>
<tr>
<td>1257</td>
<td>12</td>
<td>0.57 (2.03)</td>
<td>0.354</td>
<td>0.150</td>
</tr>
<tr>
<td>1318</td>
<td>12</td>
<td>-0.17 (2.89)</td>
<td>0.845</td>
<td>0.376</td>
</tr>
<tr>
<td>1379</td>
<td>12</td>
<td>-0.22 (2.22)</td>
<td>0.742</td>
<td>0.237</td>
</tr>
<tr>
<td>1440</td>
<td>12</td>
<td>0.31 (2.83)</td>
<td>0.713</td>
<td>0.107</td>
</tr>
<tr>
<td>1501</td>
<td>12</td>
<td>-0.34 (1.78)</td>
<td>0.519</td>
<td>0.318</td>
</tr>
</tbody>
</table>

No frequencies had a group suppression that was significantly different from zero. The mean suppression for the group and the 95% confidence intervals are shown in Figure 5-11.
The greatest correlation between the suppression and the auditory filter bandwidth was found at 1208Hz (see Table 5-9) and again this correlation was found to be in the positive direction. However, this was not statistically significant.

5.3.7 Other parameters describing the shape of the auditory filter

So far only the 3dB bandwidth of the auditory filter has been examined in relation to the suppression of TOAEs. This gives an indication of the degree of tuning of the filter, but does not take into account any of the other aspects of the shape of the filter, such as the gradient of the slopes on either side, or the asymmetry. The parameters describing the upper and lower skirts of the filter ($p_u$ and $p_l$) were therefore analysed along with $k$, the detector efficiency and $r$, the dynamic range limitation. The symmetry of the filter was assessed by comparing the upper and lower slopes. A symmetry index (SI) was devised to measure this, whereby

$$SI = \frac{p_l}{p_u}$$
For the 39 subjects tested, the mean SI was 1.22 with a standard deviation of 0.29. The majority of the subjects (71.8%) had a SI of greater than 1 i.e. the lower slope of the auditory filter had a higher gradient than the upper slope. The SI was found to approximate a normal distribution (see Figure 5-12).

Figure 5-12: Histogram of the Symmetry Index of the auditory filter shape in 39 normal subjects, showing the normal curve.

5.3.7.1 TOAE magnitude and suppression

These measures were compared to the TOAE standard test response magnitude as well as the results from the four combinations of TOAE suppression. The Spearman correlation coefficients and their corresponding two-tailed significance p values are shown in Table 5-10. There were 39 subjects tested in the TOAE magnitude test and in the first two suppression categories and 38 in the last two suppression categories.
Table 5-10: Correlation of TOAE magnitude and suppression with parameters describing auditory filter shape

<table>
<thead>
<tr>
<th>OAE Measurement</th>
<th>P (lower)</th>
<th>P (upper)</th>
<th>k</th>
<th>r</th>
<th>Symmetry (SI)</th>
</tr>
</thead>
<tbody>
<tr>
<td>TOAE magnitude</td>
<td>-0.062</td>
<td>-0.033</td>
<td>-0.056</td>
<td>-0.015</td>
<td>0.052</td>
</tr>
<tr>
<td>Ipsi click-Contra white noise</td>
<td>0.087</td>
<td>0.208</td>
<td>0.106</td>
<td>-0.074</td>
<td>0.023</td>
</tr>
<tr>
<td>Ipsi click-Contra 1kHz tone</td>
<td>-0.017</td>
<td>-0.002</td>
<td>0.062</td>
<td>0.091</td>
<td>0.080</td>
</tr>
<tr>
<td>Ipsi 1kHz tone – contra white noise</td>
<td>0.144</td>
<td>0.299</td>
<td>0.259</td>
<td>-0.073</td>
<td>-0.015</td>
</tr>
<tr>
<td>Ipsi 1kHz tone – contra 1kHz tone</td>
<td>0.071</td>
<td>0.172</td>
<td>0.235</td>
<td>0.061</td>
<td>-0.042</td>
</tr>
</tbody>
</table>

None of these more detailed measures of the shape of the auditory filter were found to be correlated with any of the suppression results or with the standard TOAE response magnitude.

5.3.7.2 TOAE magnitude and suppression in specific frequency bands

As before (see section 5.3.3), the TOAE magnitude and suppression from the 39 subjects were analysed in select frequency bands around 1kHz and 2kHz. The results were then compared to the other parameters that describe the shape of the auditory filter to examine whether there was any relation. The results of the Spearman’s rho correlation calculations are shown in Table 5-11. The results show no statistically significant correlation between any of the parameters under test.

Table 5-11: Correlation of TOAE magnitude and suppression at 1 and 2kHz with parameters describing auditory filter shape

<table>
<thead>
<tr>
<th>OAE Measurement</th>
<th>Stimuli</th>
<th>Frequency Band (kHz)</th>
<th>P (lower)</th>
<th>P (upper)</th>
<th>k</th>
<th>r</th>
<th>Symmetry (SI)</th>
</tr>
</thead>
<tbody>
<tr>
<td>TOAE magnitude</td>
<td>1</td>
<td>0.164</td>
<td>-0.016</td>
<td>0.020</td>
<td>-0.252</td>
<td>-0.188</td>
<td></td>
</tr>
<tr>
<td></td>
<td>2</td>
<td>-0.009</td>
<td>0.011</td>
<td>-0.116</td>
<td>0.045</td>
<td>0.089</td>
<td></td>
</tr>
<tr>
<td>Ipsi click-Contra white noise</td>
<td>1</td>
<td>-0.032</td>
<td>0.091</td>
<td>0.032</td>
<td>-0.198</td>
<td>-0.036</td>
<td></td>
</tr>
<tr>
<td></td>
<td>2</td>
<td>0.062</td>
<td>0.273</td>
<td>0.159</td>
<td>-0.007</td>
<td>-0.117</td>
<td></td>
</tr>
<tr>
<td>Ipsi click-Contra 1kHz tone</td>
<td>1</td>
<td>-0.112</td>
<td>-0.083</td>
<td>0.014</td>
<td>-0.006</td>
<td>0.068</td>
<td></td>
</tr>
<tr>
<td></td>
<td>2</td>
<td>-0.132</td>
<td>-0.105</td>
<td>-0.116</td>
<td>0.085</td>
<td>-0.088</td>
<td></td>
</tr>
<tr>
<td>Ipsi 1kHz tone– Contra white noise</td>
<td>1</td>
<td>0.121</td>
<td>0.235</td>
<td>0.187</td>
<td>-0.101</td>
<td>0.012</td>
<td></td>
</tr>
<tr>
<td></td>
<td>2</td>
<td>0.050</td>
<td>0.206</td>
<td>0.205</td>
<td>0.067</td>
<td>-0.061</td>
<td></td>
</tr>
<tr>
<td>Ipsi 1kHz tone– Contra 1kHz tone</td>
<td>1</td>
<td>-0.070</td>
<td>-0.035</td>
<td>0.044</td>
<td>0.115</td>
<td>-0.087</td>
<td></td>
</tr>
<tr>
<td></td>
<td>2</td>
<td>-0.020</td>
<td>0.252</td>
<td>0.183</td>
<td>0.088</td>
<td>-0.164</td>
<td></td>
</tr>
</tbody>
</table>
5.3.7.3 Temporally windowed TOAE suppression

The parameters $p_l$, $p_u$, $k$, $r$, and the SI were then compared with the above suppression values after having been temporally windowed to include only the portion of the response from (10-20ms) after stimulus onset (as in section 5.3.4). The results are shown in Table 5-12.

As before there was no statistically significant correlation between any of the OAE measurements and the auditory filter shape parameters.

Table 5-12: Correlation of temporally windowed TOAE suppression at 1 and 2kHz with parameters describing auditory filter shape

<table>
<thead>
<tr>
<th>OAE Measurement</th>
<th>Stimuli</th>
<th>Frequency Band (kHz)</th>
<th>$P$ (lower)</th>
<th>$P$ (upper)</th>
<th>$k$</th>
<th>$r$</th>
<th>Symmetry (SI)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Ipsilateral click-</td>
<td>1</td>
<td>-0.021</td>
<td>-0.004</td>
<td>0.020</td>
<td>-0.333</td>
<td>0.018</td>
<td></td>
</tr>
<tr>
<td>Contra white noise</td>
<td>2</td>
<td>-0.065</td>
<td>0.137</td>
<td>-0.043</td>
<td>-0.293</td>
<td>-0.249</td>
<td></td>
</tr>
<tr>
<td>Ipsilateral click-</td>
<td>1</td>
<td>-0.147</td>
<td>-0.139</td>
<td>-0.157</td>
<td>-0.118</td>
<td>0.006</td>
<td></td>
</tr>
<tr>
<td>Contra 1kHz tone</td>
<td>2</td>
<td>-0.079</td>
<td>-0.111</td>
<td>-0.021</td>
<td>-0.110</td>
<td>-0.052</td>
<td></td>
</tr>
<tr>
<td>Ipsilateral 1kHz tone-</td>
<td>1</td>
<td>0.074</td>
<td>0.190</td>
<td>0.138</td>
<td>-0.134</td>
<td>-0.036</td>
<td></td>
</tr>
<tr>
<td>Contra white noise</td>
<td>2</td>
<td>-0.003</td>
<td>0.102</td>
<td>0.060</td>
<td>-0.190</td>
<td>-0.142</td>
<td></td>
</tr>
<tr>
<td>Ipsilateral 1kHz tone-</td>
<td>1</td>
<td>0.065</td>
<td>0.229</td>
<td>0.137</td>
<td>0.031</td>
<td>-0.009</td>
<td></td>
</tr>
<tr>
<td>Contra 1kHz tone</td>
<td>2</td>
<td>-0.068</td>
<td>-0.007</td>
<td>-0.030</td>
<td>0.020</td>
<td>-0.100</td>
<td></td>
</tr>
</tbody>
</table>

5.3.7.4 SOAEs

Each of the parameters which describe aspects of the shape of the auditory filter was compared between the groups of subjects with and without SOAEs. Nineteen out of 39 subjects has SOAEs in the test ear.

The mean and standard deviations of the 5 parameters describing the shape of the auditory filter are shown in Table 5-13 for the groups of subjects with and without SOAEs. The independent samples t-test was used to compare the difference between the means of each of the two groups, except for $r$ which was found not to be distributed normally, and thus the Mann-Whitney test was used. The significance values resulting from this test are shown in the last column of the table. Although there are differences in the mean values, they are not significantly different.
Table 5-13: Comparison of the values of the parameters describing the shape of the auditory filter in subjects with and without SOAEs

<table>
<thead>
<tr>
<th>Filter shape parameter</th>
<th>With SOAEs</th>
<th>Without SOAEs</th>
<th>Asymptotic significance (2-tailed)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Mean</td>
<td>Standard deviation</td>
<td>Mean</td>
</tr>
<tr>
<td>P(lower)</td>
<td>33.58</td>
<td>13.24</td>
<td>36.33</td>
</tr>
<tr>
<td>P(upper)</td>
<td>27.71</td>
<td>5.42</td>
<td>28.51</td>
</tr>
<tr>
<td>K</td>
<td>9.69</td>
<td>3.04</td>
<td>9.87</td>
</tr>
<tr>
<td>R</td>
<td>-47.74</td>
<td>19.31</td>
<td>-42.64</td>
</tr>
<tr>
<td>Symmetry Index</td>
<td>1.18</td>
<td>0.29</td>
<td>1.26</td>
</tr>
</tbody>
</table>

5.3.7.5 DPOAEs

(a) DP magnitude

The 7 measures of DP magnitude outlined in section 5.3.6.1 were compared to the 5 parameters which describe the shape of the auditory filter to see if there was any relation. There were 26 subjects in the group. The results of the Spearman's rho correlation coefficients and the p values are shown in Table 5-14.

Table 5-14: Correlation of DPOAE magnitude measurements with parameters describing auditory filter shape

<table>
<thead>
<tr>
<th>DPOAE Measurement</th>
<th>P (lower)</th>
<th>P (upper)</th>
<th>k</th>
<th>r</th>
<th>Symmetry (SI)</th>
</tr>
</thead>
<tbody>
<tr>
<td>DP at 1kHz</td>
<td>-0.342</td>
<td>-0.357</td>
<td>-0.172</td>
<td>-0.146</td>
<td>-0.246</td>
</tr>
<tr>
<td>DP at 2kHz</td>
<td>-0.237</td>
<td>-0.166</td>
<td>-0.098</td>
<td>0.002</td>
<td>-0.134</td>
</tr>
<tr>
<td>Mean (745-1501Hz)</td>
<td>-0.132</td>
<td>-0.070</td>
<td>0.105</td>
<td>-0.050</td>
<td>-0.051</td>
</tr>
<tr>
<td>Low Mean (745-977Hz)</td>
<td>0.002</td>
<td>-0.150</td>
<td>0.110</td>
<td>0.115</td>
<td>0.184</td>
</tr>
<tr>
<td>High Mean (1013-1501Hz)</td>
<td>-0.141</td>
<td>0.023</td>
<td>0.135</td>
<td>-0.113</td>
<td>-0.137</td>
</tr>
<tr>
<td>High-Low</td>
<td>-0.128</td>
<td>0.355</td>
<td>0.251</td>
<td>-0.375</td>
<td>-0.441*</td>
</tr>
<tr>
<td>Standard deviation</td>
<td>-0.203</td>
<td>0.069</td>
<td>-0.234</td>
<td>-0.255</td>
<td>-0.184</td>
</tr>
</tbody>
</table>

* statistically significant, p<0.05
None of the parameters are significantly correlated except the relation between the symmetry index and the 'high-low' DP measurement. The correlation coefficient in this case is -0.441 and the 2-tailed p value is 0.024.

(b) DP suppression

The amount of suppression of DPs at each f2 frequency was compared to p(lower), p(upper), k, r, and the symmetry index in the same subjects. This was done for both the DPs suppressed with a contralateral 1kHz tone or a 1kHz narrow band noise. The subject groups were the same as previously described in section 5.3.6.2. The results tables show the Pearson correlation coefficients and the significance values for each frequency, for the standard DPGram measurements as well as the fine structure around 1kHz, and also the suppression produced by both types of contralateral stimuli used. These tables can be found in Appendix 3.

A number of significant correlations were found. The following graphs show the distribution of the significant correlations with f2 frequency. Figure 5-13 displays the results for the 'DPGram' measurement with both a 1kHz tone suppresser and a 1kHz narrow band noise suppresser. More frequencies have correlated suppression with filter shape parameters for the 1kHz tone suppresser than for the narrowband noise suppresser. P(upper) was found to be correlated with the suppression due to a 1kHz tone at numerous frequencies, especially between 1 and 2kHz. Examining the suppression due to the narrow band noise in Figure 5-13, significant correlation was also present for p(upper) in this region. Thus, it seems that efferent suppression acting in the region of 1-2kHz, affects the gradient of the upper slope of the 1kHz auditory filter. This is further emphasised by the finding of an agreement between the two separate tests (tone and NBN suppresser) which were performed on subject groups that differed (only about half of the subjects participated in both tests).

In the region from 4-6kHz, the suppression correlates with all of the auditory filter shape parameters.

The frequencies around 1kHz were examined in greater detail by measurement of the fine structure during contralateral 1kHz tone and 1kHz narrow band noise stimulation. The distribution of f2 frequencies where significant correlation occurred, with the 5
parameters that describe the shape of the auditory filter, are shown in Figure 5-14. Again, \( p(\text{upper}) \) correlates with the suppression at a couple of frequencies.

All of the significant correlations are negative in direction, except for one low frequency correlation of \( r \) with suppression. This again adds weight to the argument that there is in fact some interaction between the suppression and the filter shape parameters. This interaction acts such that an increase in suppression is associated with a decrease in \( p(\text{upper}) \) and to some extent a decrease in \( p(\text{lower}) \), \( k \), \( r \), and the symmetry index.
Figure 5-13: Graphs showing the frequencies of significant correlation between suppression of DPs and auditory filter shape parameters - DPgram

DP suppressed by 1kHz tone

DP suppressed by 1kHz narrowband noise
5.3.8 Influence of other factors

5.3.8.1 Pure tone hearing threshold

The mean pure tone hearing threshold level in the ear which was tested was 1.1dBHL (s.d. = 3.4) for the 39 subjects showing that the group as a whole had normal levels of hearing at 1kHz. This threshold was then compared to the results of the OAE and the
filter shape measurements to see whether there was any relation. The Spearman's rho correlation coefficient for the relationship between the 3dB bandwidth and the hearing threshold was 0.245 with a p value of 0.149. This was not a significant correlation. There was also no significant correlation for p(upper), p(lower), k, r, the symmetry index or any of the measures made using otoacoustic emissions.

5.3.8.2 Gender

21 females and 18 males were tested. There was no significant difference in any of the parameters describing the auditory filter shape between the sexes. Regarding otoacoustic emissions, the TOAE amplitude was found to be significantly greater (p=0.02, independent samples t-test) in females (mean=13.32dB) than in males (mean=10.19dB). The TOAE amplitude at 1kHz was found to follow this trend (p=0.017), but the difference was not significant at 2kHz. The suppression of OAEs was not found to be significantly different, for any of the stimuli used, in males and females, disagreeing with the results found by Khalfa and Collet (1996) and McFadden (1993).

5.3.8.3 Ear laterality

The differences between the right and the left ear were examined. The right ear of 21 of the subjects was tested whereas the left ear of 18 subjects was tested. There were no significant differences between right and left ears for any of the auditory filter shape parameters. The TOAE amplitude was found to be greater in the right ear (mean=13.34dB) than the left ear (mean=10.17dB) with a significant p value of 0.019 on the independent samples t-test. This trend was also found for the TOAE amplitude in the 1kHz region (p=0.033) and in the 2kHz region (p=0.015). No significant differences between the ears were found for the distortion product measurements. For the suppression of TOAEs, the only stimulus combination for which there was a significant ear difference was for tone evoked emissions suppressed by 1kHz contralateral tones (p=0.042). The prevalence of SOAEs was found to be greater in the right ears that were tested than the left ears (significance value 0.025 on Fisher's exact test). These latter results are all in agreement with laterality results found previously with OAEs (Khalfa and Collet 1996; Khalfa et al. 1997; McFadden 1993).
The OAE suppression results were compared with the ability to detect a 1kHz tone in noise. This measurement was taken from the threshold of the 1kHz tone in the condition having a masker with no notch in the auditory filter shape measurements. No significant correlation was found between detection of the tone in noise and the suppression of OAE in any of the stimulus conditions.

**5.3.8.5 Age**

The mean age of the 39 subjects was 23.8 years (s.d.=4.0) with the minimum age being 18 and the maximum 35. There was found to be a weak but significant correlation between age and the suppression of clicks by 1kHz tones (Pearson’s correlation coefficient=-0.38, significance=0.017). This indicated that suppression of OAEs using these stimuli decreased with age. However, there was no linear relationship between any of the other measures of suppression, or any of the parameters describing the shape of the auditory filter, and age of the subject.

**5.4 Discussion**

A group of 39 audiometrically normal human subjects were tested in this study. The experiment was carried out in order to ascertain whether the effectiveness of an individual’s auditory efferent system was related to the tuning characteristics of their auditory systems, measured psychoacoustically.

The medial efferent system is assumed to produce a reduction in the mechanical feedback generated by the outer hair cells, the cochlear amplifier. In turn, the selective amplification of the motion of the basilar membrane seems intrinsically linked to the sharp tuning of the cochlea. It is hypothesised, that the medial efferent system exerts control over the cochlear amplifier, in a manner that aids detection of tones in a background of noise. Past work has indicated that at the auditory nerve, stimulation of the medial efferent system seems to reduce the response to low level ‘non-essential’ background noise. This thereby increases the signal to noise ratio and range over which the nerve can code the response to the tonal target signal (Kawase et al. 1993; Kawase
and Liberman 1993). From a frequency perspective, the efferent system affects the tuning of the auditory fibres (Guinan and Gifford 1988b), therefore influencing the effectiveness by which a background noise masks a signal.

Firstly, the results from the standard, non-suppressed OAE measurements are discussed. No evidence was found for a statistically significant linear relationship between the 3dB bandwidth, lower slope (pl), upper slope (pu), r, k or the symmetry index, and TOAE magnitude, SOAE presence or DP magnitude. The only exception was the ‘high-low’ measure of the DPs (indicating the general trend of the DP magnitude across frequency), which was weakly but negatively correlated with the symmetry index of the auditory filter. The frequency ranges used to calculate the ‘high-low’ measure were 745-977Hz and 1013-1501Hz. These are approximately the same frequency ranges as the pass band covered by the auditory filter at 1kHz, and therefore the measures can be compared more readily (making the assumption that the f2 frequency is close to the point of DPOAE generation). The direction of the correlation indicates that in subjects where the high frequency DP magnitude is smaller in relation to the low frequencies, the high frequency side of the auditory filter is shallower in gradient in comparison to the low frequency side. This may lead one to assume that the more active regions of the cochlea allow for sharper tuning of the portion of the auditory filter in that region. However, the lack of significant correlation coefficients between the mean high and low DP magnitudes individually and the filter shape parameters points against this. It is possible that the balance of the active processes occurring over the length of the cochlea involved in the tuning of the filter (and maybe beyond) is the important factor in determining the degree of tuning exhibited. One must also consider the possibility that the correlation had occurred by chance. With the large numbers of correlation calculations being made it is likely that some ‘false-positive’ results will be found. However, the overall lack of significant correlation between the active processes present in the cochlea and the tuning does not in itself indicate that the efferent system is not involved in some way.

Examining the results from the suppression of TOAEs showed that for the group as a whole, the suppression from all four tests was significant. No evidence of a linear relationship was found with either the bandwidth, or any of the parameters describing the shape of the auditory filter at 1kHz. This was the case for all four of the TOAE suppression tests, even after filtering to include only frequencies around 1kHz or 2kHz.
and after windowing to include only the last 10ms of the response window. It is also
important to note that examination of the distribution of results graphically, revealed no
other non-linear relationships.

Consideration of the hypothesis whereby the medial efferent system could control the
tuning in the cochlea, by modifying the damping of the basilar membrane motion, one
might expect there to be a relationship between the strength of the efferent suppression
measured in TOAEs and auditory tuning. The lack of a relationship points either to
different physiological mechanisms underlying the two processes or to some feature of
the measurement techniques.

Examination of the reliability of the TOAE and the psychoacoustic tests reveals that
both tests were satisfactorily repeatable. It is unlikely therefore that inter-test variability
was masking a true relationship.

The levels of the contralateral stimuli were such that effects from air and bone
conduction on the ipsilateral cochlea would be minimal, and in any case, past work from
unilaterally deaf subjects (Collet et al. 1990) showed that suppression did not occur in
these patients. The levels were also well below the stapedius reflex thresholds of all the
subjects tested. Again, previous studies have indicated that TOAE suppression effects
were not due to the stapedius reflex since the suppression was demonstrable in subjects
without the reflex (Veuillet et al. 1991). Although a small reflex effect, at a level too
small to be measured by conventional tympanometry, cannot be ruled out at these levels,
the past studies on TOAE suppression indicate that it is unlikely that it played a great
role in these results.

Comparison of these results with past studies is difficult since virtually all of the
previous work has been carried out using paradigms where the efferent system is either
stimulated or sectioned. As seen earlier, many of these results from animals have found
that the efferent system does influence the tuning of the auditory system. The only
comparable study (Micheyl and Collet 1994), which uses a simplified method for
assessing the auditory filter shape, found that the tuning was related to the presence of
SOAEs and to the magnitude of TOAEs. These results differ from the results that we
found. However, most of the statistically significant differences were found by them for
the auditory filter measured 2kHz and not at 1kHz. Other studies which have found
relations between TOAE suppression and detection in noise tasks have all involved
contralateral masking (Giraud et al. 1997a; Micheyl and Collet 1996; Micheyl et al. 1995b) and therefore again, differ from this study.

In this experiment, we have compared the detection of a tone in an ipsilateral masker with the suppression of ipsilateral TOAEs by a contralateral masker. The lack of significant correlations could be because the two tests involve different populations of nerve fibres. The TOAE suppression test utilises a contralaterally effective loop whereas the tuning curve test examines an efferent loop which, if it is involved at all in frequency selectivity, would be ipsilateral in site of action. The assumption that the experiment makes, is that a high level of activity in one population of efferents in a particular subject, would be associated with a high level of activity in the other population. This therefore may be one reason why no relationship was found between the two tests.

Another possible explanation for the results could be that the efferent system is not involved to any great degree in frequency selectivity when both tone and masker are only present in one ear. This situation is unlike most encountered in everyday circumstances, since environmental background noise generally affects both ears. The efferent system may only be effective in aiding tuning when there is binaural noise. The experiment described in chapter 7 was therefore devised to enable investigation of this.

The DPOAE suppression measured was generally small, and examination of the group data showed significant suppression at very few frequencies, although these frequencies were all in the mid-range. These suppression levels were comparable to those encountered by Chery Croze et al. (1993). However, these mean levels were less important than the relationship between the DPOAE suppression and the shape of the auditory filter in individual subjects.

The 3dB bandwidth of the auditory filter was only found to be correlated with the DP suppression at one frequency (1257Hz). This was in the positive direction implying that greater efferent activity at that frequency was associated with a wider filter. However, this result alone does not give much credibility to a possible link. Examination of the other five parameters describing the shape of the auditory filter provides some more interesting results.

The upper slope of the auditory filter at 1kHz ($p_u$) was found to be negatively correlated with DPOAE suppression at a number of different frequencies between 1 and 2kHz.
This was the case for both the 1kHz tone and the 1kHz narrow band noise suppressers. The negative correlation indicated that subjects with greater efferent suppression of DPOAEs had shallower upper filter slopes at 1kHz. The fact that this negative correlation is found at many frequencies and with both types of suppresser (and therefore different subject groups) increases confidence in the validity of the correlation. The relationship with the gradient of the lower slope of the filter (p_u) was less strong, with a significant correlation (also negative) occurring at only one frequency in the 1-2kHz region. The measure of detector efficiency, k, and the dynamic range limitation parameter, r, also had only a couple of frequencies where a significant correlation was encountered. Although the lower number of significant correlations for these latter two parameters offers us less confidence in a relationship, the negative direction of the correlation indicates that a higher level of efferent activity may lead to better detection efficiency and a greater dynamic range. This is in keeping with the results from previous studies in animals (Kawase et al. 1993; Kawase and Liberman 1993). The general trend of these results whereby greater efferent activity levels are associated with a widening of the tuning of the auditory filter are also in agreement with past studies. Efferent stimulation was found to broaden auditory nerve fibre tuning curves (Guinan and Gifford 1988b) and DP suppression tuning curves (Williams and Brown 1995).

The tonotopic organisation of the efferent system would lead one to expect that contralateral stimuli at 1kHz would suppress the ipsilateral response most in the region of 1kHz. Inspection of the mean suppression values across frequency in these results does not point to this interaural correspondence clearly. However, the frequencies of most of the correlations with filter shape (particularly with p_u) occur upwards from 1kHz. Tracing of the pathways of nerve fibres in animals have found that the connections that single medial efferent neurons make with outer hair cells are spread out over about an octave, but the distribution of these is biased towards the basal end of the cochlea (Liberman and Brown 1986). Thus, the place of maximum action of the medial efferent system stimulated at 1kHz may be at a slightly higher frequency. It is therefore possible that in these results, although the group suppression was not significant, the frequencies where the efferent system had maximal effect were between 1 and 2 kHz and this is why these are the frequencies that show the most correlation. In other words, the suppression caused by 1kHz stimuli is not great enough at other frequencies to give an accurate estimation of the strength of efferent activity. In any case, assuming a DP
generation site close to \( f_2 \), which is probably not entirely accurate, the results provide
evidence that strength of efferent activity in the 1-2kHz region is related to the gradient
of the upper slope of the auditory filter. This seems logical since the upper side of the
filter spans some of this frequency region.

An unexpected result was the occurrence of several significant correlations between 4
and 6kHz. All five parameters were significantly correlated in the negative direction
with frequencies in this region. This result is harder to explain since the site of action of
these efferents is far from the site of the auditory filter being tested. It is possible that the
stronger the activity of the efferent system in this region the greater the effect (damping)
on the travelling wave as it passes this point on the basilar membrane on its way to the
1kHz region.

Looking at the individual slopes of the filter and the symmetry index could provide
information that analysis of the 3dB bandwidth may mask. If, for example, one
compared two filters, one of which had a steeper lower side but a shallower upper side,
the bandwidth may be the same even though the filters are different.

Investigation of other factors that may have confounded the results firstly showed that in
our normal population, pure tone threshold was not related to any of the test results.
Gender differences were also found not to be confounding the results. Although females
had greater TOAE amplitude than males, there was no significant difference in the
auditory filter shapes. Similarly, right ears were found to have greater TOAE magnitude
and SOAE prevalence than left ears, but there were no such differences in the auditory
filter shapes. This is in agreement with past studies (Khalfa and Collet 1996). However,
in order for the present study to be comprehensive, measurements would be needed from
both ears of the same subject. The detection threshold of a 1kHz tone in noise was also
found to be unrelated to all of the OAE amplitude and suppression results. This agrees
with previous studies which have not found efferent activity to be related to detection in
noise when the detection task did not involve contralateral or binaural maskers (Micheyl
and Collet 1996; Scharf et al. 1994; Scharf et al. 1993). Likewise, in animals, there was
no detrimental effect of OCB sectioning on detection in monaural noise (Trahiotis and
Elliott 1970). Past results indicate that the efferent system is only involved in detection
in noise when stimulated binaurally. This follows from the electrophysiological
findings, which have shown that most OCB neurons respond best to binaural stimuli
(Liberman 1988). Regarding any confounding effects of age, the range of ages tested was very narrow but nevertheless, one of the TOAE suppression tests (clicks suppressed by tones) was negatively correlated with age. A reduction in OAE suppression with age has also been shown in past studies (Castor et al. 1994). However, there was no difference in the auditory filters of the subjects tested here, that was correlated with age, and the narrow spread of ages in this study do not make it possible to conduct a definitive analysis from these results. Therefore, none of the factors investigated were found to be interacting with the results.

5.5 Conclusion

The aim of this study was to establish whether the tuning of the human auditory system was related to the activity of the auditory efferent system, as measured by its effectiveness at suppressing otoacoustic emissions.

Generally, the results provided little evidence of any influence of the efferent system over monaurally measured frequency selectivity. A few results did indicate a link. However, the overall lack of a consistent relationship must make one view the few significant results with caution.

Nevertheless, examination of the suppression of distortion products provided some evidence that subjects who demonstrated a higher level of medial efferent activity at certain frequencies, were more likely to have wider tuning curves, due mostly to a decrease in the gradient of the upper slope. This result is in keeping with past studies showing an increase in tuning curve width during OCB stimulation. Since the efferent system seems able to effect cochlear mechanics, disruption of the fine tuning observed is a logical outcome.

The monaural auditory filter measurement in this experiment will not activate maximally the efferent neurons that are binaurally or contralaterally sensitive, and thus the OAE study examines the effectiveness of a different population of fibres. The next step is therefore to test the auditory system under contralateral stimulation in the hope that the same portion of the efferent system will be activated as in the OAE experiments.
Chapter 6

The Onset Latency of Auditory Efferent Suppression of Otoacoustic Emissions
6. The Onset Latency of Auditory Efferent Suppression of Otoacoustic Emissions

6.1 Introduction

When testing the effect of contralateral noise on the auditory filter it is important to know how long it takes for the sound to activate the efferent system and then affect cochlear mechanics in the opposite ear. Once this is known, one can set up the experimental paradigm to make sure that the efferent system is active over the period of time that the stimulus is presented. Additionally, knowledge of the timing of the effect is useful in assessing whether it would be useful in frequency selectivity where it would need to adjust quickly to adapt to changing environmental sounds. The timing of the onset of efferent suppression has received scant attention in past work, especially in humans.

Some indication of the timing involved may be ascertained from earlier work, mostly on animal models, assessing both the onset time and the overall timescale of response.

6.1.1 Onset Time

The past studies on the onset time of efferent activation can be divided into groups depending on the site of activation measured. First to be considered is the time taken for efferent activation to affect afferent responses. The suppression of the N1 response was shown to occur between 20 and 40 ms after the start of electrical stimulation at the floor of the medulla of a cat (Galambos 1956). The latency of inhibition of discharge rate of single afferent fibres after electrical shocks to the COCB was found to increase with an increase in characteristic frequency (Wiederhold and Kiang 1970). More recently, the efferent induced suppression of the ensemble background activity of the VIIIth nerve was measured in guinea pigs, by placing an electrode on the round window (da Costa et al. 1997). The latency of the suppression was found to be 10ms or less.

Next is the latency of activation of the efferent fibres themselves. The activation time of different fibres in the cat COCB ranged from 5-40ms when stimulated with short tone pips (Fex 1962). Cody and Johnstone (1982) recorded from the efferent fibres in Rosenthal's canal in the guinea pig and found minimum latencies to range between 7
and 35 ms for different fibres, whilst a minimum latency of 10 ms was found when investigating the olivocochlear bundle (OCB) response to tone bursts in cats (Liberman and Brown 1986). This latency increased as the sound intensity approached threshold. Gummer et al. (1988) measured efferent nerve responses to amplitude modulated tones in the guinea pig. Recordings were taken at the basal turn of the cochlea making no distinction between crossed and uncrossed efferents. They found that group phase delays were shorter (8.2 ms) and more tightly distributed than minimum onset latencies (24.2 ms) measured from the same fibres. The group phase delay describes the latency for a change to occur in the steady state (i.e. continuous noise) and the minimum onset latency is comparable to studies above.

Measurements of the onset of efferent effect on cochlear potentials have also been made. The latency of the negative potential of the scala media induced by electrical stimulation of the COCB was measured. Fex (1967) found a range of latencies of 12-40 ms from different experiments in the cat, and concluded that this measurement represented the activity of the outer hair cells. Later, Konishi and Slepian (1971) carried out a similar experiment on guinea pigs, finding an average latency of 10 ms. Contralateral broadband stimulation was found to cause changes in the cochlear microphonic with a latency of 11 ms in the mustached bat (Henson et al. 1995). Sridhar et al. (1995) have found evidence for 'slow' changes to cochlear responses in the guinea pig along with the 'fast' effects already observed. The slow effect was found to have time constants three orders of magnitude slower than the fast effect. It was suggested that whilst the fast effect may be involved in modulating responses to acoustic stimuli, the slow effect may play a role in protection from acoustic injury.

Finally, a couple of studies have looked at the time taken for the efferent system to affect OAEs. Lind (1994) measured the latency of contralateral suppression of transient otoacoustic emissions in normal human subjects. The latency range found (40-140 ms) was wider, and had a higher maximum value, than those established in the above studies. Recently, Pratt et al. (1998) used contralateral clicks to actually evoke OAEs. This was assumed to be via the action of the efferent system and was found to take 12-22 ms to occur.
6.1.2 Overall Timescale of Response

Other studies have examined the time taken for the effect to take place and decay. Using electrical stimulation of the COCB, effects on the receptor potentials of the inner hair cells were found to take 50-250ms to reach maximum and decay (Brown and Nuttall 1984). Warren and Liberman (1989) presented sound to the contralateral ear of cats as the responses of single afferent fibres were recorded and found that the suppression took 250ms to develop fully and 80ms to recover. Sectioning of the COCB in the floor of the fourth ventricle made little difference to the suppressive effect and thus it was suggested that the suppression caused by contralateral sound occurred via the uncrossed OCB. It is possible therefore, that some of the above results measured from the COCB may prove to be of less value in understanding the role of the efferent system.

Many of the previous studies have been conducted on animals using electrical stimulation. The use of acoustic signals has the advantage of being much more physiologically realistic than electrical stimulation. This study examined the timing of the onset of the suppression effect of contralateral noise on click evoked otoacoustic emissions (CEOAE) in humans.

6.2 Method

6.2.1 Subjects

35 ears of 25 subjects (8 male and 17 female, mean age 26.1 +/-5.3 years) were tested. Each test of the main experiment involved different groups of subjects and many subjects participated more than once for different tests. All subjects had normal pure tone thresholds (better than 20dBHL at octave frequencies between 0.5-8kHz) and ipsilateral and contralateral stapedial reflex thresholds (Prasher and Cohen 1993). Subjects were also tested to ensure the suppression of CEOAEs in the presence of continuous 50dBSL contralateral white noise.
6.2.2 Apparatus and stimulus protocol

The Otodynamics ILO92 twin probe analyser provided all auditory stimuli, and was used to record and analyse the resulting responses. The tests were conducted in a sound proof room.

The contralateral suppression of click evoked OAEs was carried out in the manner described in section 3.2.1.2(b). The ipsilateral stimulus was, as before, linear clicks at 63+/-3dBSPL. The contralateral stimulus was a 5ms burst of white noise at 50dBSL. This was well below the acoustic reflex threshold. Evidence from past work on unilaterally deaf subjects (Collet et al. 1990) suggests that acoustic cross-talk may not be a factor of importance as suppression is not observed in these subjects.

The ILO92 produces clicks in groups of four. The data acquisition part of the programme was modified to allow each of the four clicks to be averaged and analysed separately. The experiment was conducted using five different time delays of the noise burst in the contralateral ear. This delay was measured with respect to the first click of each group of four in the ipsilateral ear. This allowed emission measurements to be made at various intervals after the onset of the noise burst. Figure 6-1 shows the five different timings used. The delay in time (D) between the first click and the noise burst was 14, 13, 8, 3 and 0ms for tests 1 to 5 respectively. Tests 1 and 2 were carried out initially because an onset effect early, in the first 10ms, was expected. Tests 3, 4 and 5 were then performed to give a more complete view of the process. In tests 1 to 4, the delay of the contralateral noise burst meant that the first of each set of four clicks occurred before the noise. These results were included and referred to with negative times (-14, -13, -8 and -3ms) to differentiate them from those clicks that occurred at or after the onset of the contralateral noise burst.
Figure 6-1: Stimulus arrangement where $D$ is the delay of the noise burst, and $C_n$ are the times between onset of the noise and the click. The table shows the timings that could be measured for each of the five test paradigms. Click times shown with a "-" sign were before the onset of the contralateral noise burst.

<table>
<thead>
<tr>
<th>Test number</th>
<th>Noise Delay after Click 1 $D$(ms)</th>
<th>Time between onset of noise and click (ms)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>C1</td>
</tr>
<tr>
<td>1</td>
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</tr>
<tr>
<td>2</td>
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<td>-13</td>
</tr>
<tr>
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<td>-8</td>
</tr>
<tr>
<td>4</td>
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<td>-3</td>
</tr>
<tr>
<td>5</td>
<td>0</td>
<td>0</td>
</tr>
</tbody>
</table>

The short duration of the noise burst was chosen so that each click was exposed to the same duration of noise. A longer noise burst would have meant that the clicks occurring later in the sequence would have been influenced by a longer sound than those occurring earlier. This allowed measurement of OAEs from clicks very soon after the noise burst and thus meant that even very short onset times could be assessed. The short length also allowed greater flexibility in the relative positioning of the noise burst in the analysis period of 20ms.
In order to allow for a complete recovery of the suppression to avoid any summating effect, a gap was inserted between each group of four clicks. Since Brown and Nuttall (1984) found that 50-250ms was required for the OCB effect on inner hair cell receptor potential to reach maximum and decay, and Warren and Liberman (1989) established that efferent fibre suppression took 250ms to develop and 80ms to recover, a gap of 500ms was considered appropriate. There is some evidence that efferent activity can last for several minutes after stimulation (Liberman 1988). However, these long lasting effects occur after continuous stimulation lasting many minutes and at much higher levels than used in this study. As each of the four clicks in a group were separated by 20ms, an inter-group delay of 420ms was inserted in order to make a total delay of 500ms.

6.2.3 Procedure

6.2.3.1 Continuous Contralateral Noise Suppression (CCNS) Test

First, a basic test of efferent suppression with continuous masking noise was performed using the ILO92 (as described in section 3.2.1.2(b)). Sets of 60 sweeps were recorded alternately with and without contralateral white noise. The intensities used were 63+/−3dBSPL for the linear clicks and 50dBSL for the white noise. Five sets of 60 sweeps for each condition (noise/no noise) were recorded and averaged. Suppression was calculated by subtracting the total response energy (dB) with contralateral noise from the no noise condition.

6.2.3.2 Test of Efferent Latency (EL)

This was the main experiment for this study. The intensities used were as above. The threshold of the noise bursts was established for each subject in order to set a level of 50dB above sensation level. Eight runs were recorded, consisting of 260 sets of clicks each. The four runs with contralateral noise and four runs without noise were alternated. The initial run was randomly chosen.
6.2.4 Analysis

Data was recorded with a time window of 2.5 to 20ms after each click and the spectrum of frequencies analysed lay between 0 and 6kHz. For every subject, the data from each group of four runs of 260 sweeps was summed, to obtain one set of data with a total of 1040 sweeps. This was carried out for both the 'with contralateral noise burst' (N) and the 'without noise' (Q, quiet) conditions. So, for every ear and each test there were four N response values (N1-N4) and four Q response values (Q1-Q4), i.e. one for each click.

The four Q readings were checked for any ipsilateral suppression effects in test 2 of the main experiment (i.e. the four click readings recorded without contralateral noise, to which the 13ms delayed noise would be compared). The Q1-Q4 values were averaged separately over the ears examined.

No significant differences between any of the mean Q values were found and therefore the four Q values were considered to be equivalent and were averaged to obtain a mean Q value (Qav). The difference between Qav and each N value represented the suppression for each click. This was given by the expression

\[ S_x = Q_{av} - N_x \]

where \( N_x \) is the response value for click \( x \) with contralateral noise and \( S_x \) is the suppression (dB) for that click.

The suppression data was checked to make sure it was distributed normally and then tested with the one sample t-test to determine if it was significantly different from zero.

Increased levels of suppression of the order of 3-4dB have been shown to be present by restricting analysis to the time period after 8ms (Berlin et al. 1993b). Accordingly, in this study the response was analysed between 10 and 20ms with a 2.5ms rise time (windowed data). The same statistical tests as above were then applied.

The onset latency of efferent induced suppression of OAEs could therefore be examined by comparing the suppression encountered at different times.

Finally, in order to investigate the variability encountered in the results the data was analysed further:
i) The possibility of a change in the probe fit over the duration of the test was examined. The total response data at 27ms for each run of the 'no contralateral noise' situation was averaged over the different subjects. These four 'no noise' populations would be equivalent if there was no significant change in the probe fit. The two sample t-test was used to confirm this.

ii) To compare the initial suppression test using continuous contralateral masking noise with the suppression measured from the main part of the experiment, correlation coefficients were calculated. The data sets compared to the initial test were those at 26 and 27ms since suppression was present at these times.

### 6.3 Results

All subjects had normal pure tone thresholds (better than 20dBHL between 0.5 and 8kHz), normal ipsilateral and contralateral stapedial muscle reflex thresholds and greater than 1dB of suppression of CEOAEs with the preliminary test in the presence of continuous contralateral white noise.

The total response (non-windowed) data is presented in Table 6-1 and the windowed data in Table 6-2, for times from 0 to 60ms after the onset of the noise burst and -14, -13, -8 and -3ms before the noise bursts. The suppression mean and standard deviation of the population for each timing are shown as are the number of ears tested in each case. The suppression values ranged from -0.05 to 0.24 and -0.19 to 0.62dB for the total response and the windowed data respectively. The data distribution was judged to be normal, permitting the use of the one sample t-test, which gave the significance values shown in the tables. Mean suppression which was significantly greater than zero (i.e. p<0.05) occurred at 20, 26, 27, 46 and 47ms after the noise burst and also at 52ms for the windowed data.
Table 6-1: Mean total response suppression data at various click times

<table>
<thead>
<tr>
<th>Time between onset of noise and click (ms)</th>
<th>Number of Ears Tested</th>
<th>Mean Suppression (dB)</th>
<th>Standard Deviation of Suppression</th>
<th>t-test significance level</th>
</tr>
</thead>
<tbody>
<tr>
<td>-14</td>
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</tr>
<tr>
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<tr>
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<td>0.63</td>
</tr>
<tr>
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</tr>
<tr>
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<tr>
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<tr>
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<tr>
<td>60</td>
<td>13</td>
<td>0.18</td>
<td>0.40</td>
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</table>

* significant at p<0.05
** significant at p<0.01
Table 6-2: Mean windowed suppression data at various click times

<table>
<thead>
<tr>
<th>Time between onset of noise and click (ms)</th>
<th>Number of Ears Tested</th>
<th>Mean Suppression (dB)</th>
<th>Standard Deviation of Suppression</th>
<th>t-test significance level</th>
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<tr>
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<td>0.32</td>
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</table>

The windowed response shows the suppression much more strongly than the total response. This is shown in Figure 6-2, where the windowed and the non-windowed data are compared for test 1. The suppression is statistically significant for both the windowed and the total response data at 26 and 46ms and not at -14 and 6ms. The mean windowed values for tests 1, 2 and 5 are shown in Figure 6-3 a, b and c respectively. The different tests are not directly comparable, because they are measured from different subject populations, and therefore not plotted on the same graph. The graphs show the increase in mean suppression between 6 and 26ms, 7 and 27ms, and 0 and 20ms. The onset latency of the efferent suppression of OAEs thus appears to lie between 7 and 20ms. Tests 3 and 4 showed much lower levels of suppression in comparison to the other tests, as shown in Figure 6-4 for the windowed data.

* significant at p<0.05
** significant at p<0.01
Figure 6-2: Comparison of suppression values for the total response data and the windowed data (10-20ms). Data shown is for each of the four clicks of test 1. The windowed data shows higher amplitudes of suppression than the non-windowed data. The standard error is also shown for each set of data.
Figure 6-3: Suppression results from the windowed data of tests 1, 2 and 5. a) Test 1- suppression increases markedly between 6 and 26 ms. b) Test 2- suppression increases between 7 and 27 ms. c) Test 5- suppression increases between 0 and 20 ms. The onset of suppression is therefore between 7 and 20 ms. The standard error is also shown for each set of data.
Investigation of the change in the probe fit with time gave a two sample t-test significance level of 0.901 when comparing the first and the last run. These runs were compared because they would have the greatest difference if the probe had become loose with time.

The suppression at 26 and 27ms from the total response data of the EL test was compared with that achieved with the CCNS test using continuous noise on the same subjects. The correlation between the two sets of data was found to be 0.507 for 27ms and -0.364 for 26ms with significance values of 0.246 and 0.271 respectively, showing that they were not correlated.

To check for the first clicks suppressing the later clicks (ipsilateral suppression), the Q1-Q4 values were examined for test 2. The average values ranged from 7.68 to 7.85dBdSPL. There was no significant difference between the values and therefore no evidence of ipsilateral suppression was found.
6.4 Discussion

6.4.1 Onset Time

The principal finding to emerge from this study is that the contralateral sound activated suppression of CEOAEs occurs at or before 20ms. This result is in line with previous estimates of suppression onset latency observed in animals. The time taken for acoustic stimulation to activate efferent fibres was found to be 5-40ms by Fex (1962), 7-35ms by Cody and Johnstone (1982), 10ms or greater by Liberman and Brown (1986) and Gummer et al. (1988) measured a mean value of 24.2 +/- 12.5ms. Whereas, the time for efferent stimulation to produce afferent effects was found to be 20-40ms by Galambos (1956) and <10ms by da Costa et al. (1997). The time for efferent stimulation to create changes in the cochlear potentials was found to be 12-40ms by Fex (1967), 10ms by Konishi and Slepian (1971) and 11ms by Henson et al. (1995).

A possible mechanism for the process that we have measured, starts with the stimulation of the contralateral ear and activation of the afferent system via hair cell receptors. The efferent system is then activated, stimulating the outer hair cells and thus causing a change in the otoacoustic emission. Therefore, the latency measured in this experiment may be expected to be approximately the sum of the time taken when stimulating with contralateral acoustic stimulation and recording the changes in efferent nerves (Cody and Johnstone 1982a; Fex 1962; Liberman and Brown 1986), and the time taken when stimulating the efferent nerve directly and recording from the afferent nerve (Galambos 1956) or the cochlea (Fex 1967; Konishi and Slepian 1971). The range of possible onset times that we measured do conform to the above hypothesis. However, it should be borne in mind that the experimental design in this study did not include a final afferent pathway as in the study by Galambos (1956), and thus the latency measured might be expected to be shorter, although there is the additional time for the emission to be emitted from the cochlea before being measured in the canal (<10ms, since the emission returns and is recorded within 20ms of the stimulus onset). Alternatively, the time for excitation of the cochlea to reach the superior olivary complex (SOC) is about 4ms, as measured from auditory brainstem measurements, and presumable the time for the signal to pass from the SOC to the cochlea is approximately the same. Adding some time for the process to occur in the cochlea which alters the mechanics (e.g. reverse transduction) gives a total latency time very similar to that which we have found.
It is unlikely that the effect observed is due to acoustic cross-over via air or bone conduction. Not only were the levels low to minimise this, but the latency measured was far too long to be due to cross-over.

The most marked OAE suppression occurred at 20, 26, 27, 46, 47 and 52ms. All the suppression values are small but consideration of the windowed data between 10 and 20ms has the benefit of revealing the suppression to a greater degree (Figure 6-2). Using this time window also has the advantage that there is much less chance of stimulus artefact affecting the results.

The suppression appears to start at some time after 17ms but before 20ms. It is important to note however, that the range found in this study of 17-20ms describes the possible time zone within which the suppression starts, whereas the ranges of times quoted in some of the above studies show the extent of the onset latencies measured from different fibres.

In this study the trend of increase in suppression between 17 and 20 ms was found in a large proportion of the ears tested: 76% for test 1, 93% for test 2 and 77% for test 5 for the windowed data. At present it is not clear whether there is a gradual growth of the suppressive effect or whether it begins at some point between the two times and rises sharply. A gradual growth may occur if different efferent fibres have different latencies (Cody and Johnstone 1982a; Fex 1962), especially as the stimulus was white noise in this study and this would be expected to activate many fibres of different characteristic frequencies.

It is possible that the onset of suppression occurs after the initial set of clicks with a time of 517 to 526 ms instead of 7 to 26ms. However, this appears unlikely, as the longest onset times found in previous work are of the order of 40ms.

More recently, Lind (1994) attempted to measure the onset of suppression in humans using CEOAEs. The contralateral noise was produced by a computer interface and was much longer (80ms) than that used in this study. Results were gathered from the four clicks separately and two conditions were studied: one with the batch of clicks following
the noise and one with the click batch 40ms after the start of the noise. As with our results the last 10ms of the emission was found to give larger suppression values. Large variation was found in the onset times obtained which ranged from between 40 to 140ms. These results show much longer latencies than measured by the workers mentioned previously. It is possible that OAE measurement may give slightly longer latencies than direct measurements from the nerve, but this delay would be in the order of 10ms and therefore does not fully account for the discrepancies. However, with Lind's experimental design, 40ms was the minimum time that could be examined and there were only 8 possible measurement times. One must also consider that the 2nd, 3rd and 4th clicks in the train of four would have been affected by progressively longer lengths of the contralateral noise burst and would therefore not show equivalent suppression.

Recently, another study has considered the latency of the efferent suppression of TOAEs (Tavartkiladze et al. 1996). However, they examined the ipsilaterally evoked suppression. Given that the neural path taken was probably fairly similar in length to that when suppressed contralaterally, it is perhaps not surprising that they found a similar latency to that reported here (15ms).

### 6.4.2 Lack of Significant Suppression at Certain Points

At 32, 37 and 40ms the suppression was not significant and was reduced in comparison with that at 27 and 46ms (Figure 6-4). There are four possible explanations for this: a) the suppressive effect is only activated when afferent input from the ipsilateral ear is received within a certain critical time interval after the contralateral noise. From the present data, this critical time interval would seem to be between 7 and 12ms. When the first click is 7ms after the contralateral noise burst then the suppressive effect is activated and takes time (between 7 and 20ms after noise burst) to develop. If, however, the first click occurs later (12ms after the noise burst) then the effect is not activated and the emissions are not suppressed. However, the mean values at 32 and 37ms do show some, non-significant, suppression. This may be explained by the gradual decline in the suppressive effect after the 'critical interval'. If an ipsilateral click is received before the suppression has totally diminished then some residual suppression of OAEs may be
seen. A similar mechanism was suggested (Rajan 1992) for the protective role of the
efferent system, although over a much longer time span. In this latter study, it was found
that stimulation of the floor of the fourth ventricle of guinea pigs produced reduced
temporary threshold shift (TTS) in response to loud sounds even when the loud noise
was delayed after the stimulation. By sectioning the COCB after stimulation but before
the noise, the TTS reduction was prevented, showing that storage of this effect was at a
central site. b) There are two separate mechanisms contributing to OAEs: a 'quick' reflex
type response and a slower effect involving the influence of higher centres via the
efferent system to the outer hair cells. It is possible that the dip in the suppression may
represent the point of overlap between the two mechanisms. c) The gap between the
onset of the noise burst and the click actually influences the latency of the suppression.
This is supported by the fact that in test 3 the suppression was significant only at the
final click for the windowed data, i.e. the latency was longer because the noise to click 2
interval was long (12ms). The functional basis of such a mechanism is unclear. d) It is
also feasible and perhaps most probable that the lack of suppression at these points
represents the variability in the results.

6.4.3 Variability of Suppression

The suppression of OAEs observed was small as indicated earlier but in comparison the
extent of variability across subjects was quite high. In order to find the source of the
variation, the data was subjected to further analysis.

6.4.3.1 Probe Fit

Due to the long length of the test it was considered that change in the probe fit with time
might affect the degree of emission recorded thereby increasing the variability. The two
sample t-test was used to compare the first and last run of the no noise condition and the
result showed that there was no significant change in the two readings. The results
indicate that the probe probably moved very little between the beginning and the end of
the test session. However, the change in the probe fit in the same subject over different
test sessions, may have lead to some variability in the emissions recorded and therefore
the suppression levels derived.
6.4.3.2  Comparison with the Continuous Contralateral Noise Suppression (CCNS) Test

The suppression values in the main test of efferent latency (EL) were much smaller than those observed with continuous contralateral noise in the first screening test. It is possible that the short duration of the contralateral noise may have had an effect, as it has been shown that with short stimuli there is a reduction of suppression (Liberman and Brown 1986). In addition, the values would have been expected to be smaller, because it is the onset of suppression that is being measured in the EL test, as opposed to the maximum value measured in the CCNS test.

The data from the EL test was not found to be correlated with that of the CCNS test at 26 and 27ms when comparing suppression within the same subjects. The difference in the results found by the two tests could be due to several reasons. The suppression measured in the CCNS test is in response to continuous contralateral stimulation and thus maximum suppression will have been reached for a prolonged period of time. In contrast, the EL test looks at the very small changes that occur as the suppression starts to take effect. It is possible to hypothesise from this that different people may have different onset times, which are not related to the absolute value of the suppression finally achieved. Unfortunately, due to the fact that in the test paradigm used, specific points in time were measured rather than continuous monitoring of the suppression, it is not possible to determine whether there is a variability in onset times across individuals.

An alternative explanation may be that the difference is due to a change in alertness between the two tests. During the protracted EL test, some subjects could easily have become less attentive compared to the much shorter CCNS test.

Having discussed this, however, it is noted that the range of suppression values found across subjects in both suppression tests was similar. This is demonstrated by the fact that the standard deviation of the suppression in the CCNS test was 0.697 compared to a mean standard deviation of 0.373 found in the total response suppression data after 20ms (values from before 20ms were excluded since suppression did not necessarily occur there). The suppression recorded in the EL test was much smaller than that of the CCNS test and therefore the standard deviation forms a much higher percentage of the result. It was difficult to standardise the attentiveness of the different subjects and this
may be a contributing factor to the range of suppression levels seen, since attention has been shown to affect OAE level (Froehlich et al. 1993). It is therefore unclear as to how much of the variability observed is due to inherent differences in each subject's efferent system.

### 6.5 Conclusion

This study has demonstrated a means of estimating the onset time of efferent suppression in humans. The onset was found to be between 17 and 20 ms after the start of a contralateral noise burst. Questions still remain about the source of the variations in the results and whether they can be eliminated or whether they are an inherent part of the suppression process or the testing procedure. Further work is necessary to address these problems along with a more detailed analysis of the onset slope and the confirmation and possible cause of the lack of significant suppression encountered at some of the measurement times.

However, this evidence indicates that the fast activation time observed in animals is similar in humans. The speed with which the efferent system is able to affect cochlear mechanics is important in the processing of auditory stimuli. If, in fact the efferent system is involved in controlling the fine tuning of the cochlea, then a quick activation time such as this would enable rapid response to the constantly changing acoustic environment. Thus allowing for optimal performance of the auditory system.
Chapter 7

Efferent Activation with White Noise during Filter Shape Measurement
7. Efferent Activation with White Noise During Filter Shape Measurement

7.1 Introduction

It is possible that the efferent system only affects frequency selectivity when activated contralaterally or binaurally. In this chapter, the investigation of the effect on the auditory filter of activating the efferent system contralaterally, is reported. This has then been compared to the effect that contralateral stimulation has on OAEs.

There is some proof from the literature that the efferent system does not play a measurable role in frequency selectivity without some extra stimulation (either electrical or acoustic). In general, the studies where the efferent system was shown to have an effect on frequency selectivity were those where the OCB was stimulated (Brown et al. 1983; Guinan and Gifford 1988b; Murugasu and Russell 1996) whereas when the nerve was cut the result varied (see section 2.5). A possible interpretation of these results is that the ipsilateral stimulus alone, is not enough to activate the efferent system. Therefore, experiments where the OCB was cut and the change to an ipsilateral response was measured tended to show no effect. This may have been because the efferent system was not activated when the response was measured before sectioning. This would also explain why many of the experiments performed by Scharf et al. (1997) and Scharf et al. (1994) showed no change after cutting the efferent nerve in patients who had undergone a vestibular neurectomy. Most of the tests that they carried out only used ipsilateral stimuli, and for the test that they carried out with binaural noise, there were only two subjects. More evidence for this line of reasoning comes from the work of Liberman (1988), who found that most efferents in the cat have binaural inputs.

Studies on humans have shown changes to frequency selectivity during contralateral activation of the efferent system. Micheyl and Collet (1996) found that OAE suppression was only related to detection of tones in noise whilst a contralateral noise was present, and Williams and Brown (1995) showed that contralateral stimulation broadened the DP tuning curve, which was thought to be a measure of auditory frequency selectivity.
In this experiment therefore, we examine how the tuning of the human auditory system, measured psychoacoustically, changes during efferent stimulation, and if these changes are related to the efferent effect measured using otoacoustic emissions.

7.2 Methods

7.2.1 Subjects

A subset of the subjects tested in chapter 5 was tested in this experiment. In total, 24 subjects were tested, 12 of whom were male and 12 female. Their ages ranged from 19 to 29 years of age and the mean age was 23.2 years (standard deviation 2.58). The ear to be tested was chosen randomly, resulting in 50% of the studies being carried out on the right ear and 50% on the left ear. This was done in order to eliminate ear symmetry bias which has been suggested to exist for efferent function (McFadden 1993).

All the subjects had normal pure tone audiometry thresholds, tympanometry and acoustic reflex thresholds as described in sections 3.3, 3.4 and 3.5.

7.2.2 Measurement of Auditory Filter Shape

The auditory filter shapes were measured with and without contralateral noise. The same method was used to find the filter shape as that described in section 3.1. The contralateral stimulus used was white noise of 65 dBSPL produced by the WG1 module of the TDT equipment. In order to give some idea of how this related to levels in dBHL or dBSL, three ears were tested to find their perception thresholds for white noise on the Tucker-Davies equipment and on a standard audiometer. The mean perception threshold on the TDT equipment was at an attenuation level of -48.3dB. However, these subjects all had white noise thresholds measured on the audiometer of -5dBHL, implying that they had thresholds 5dB better than a normal population. Thus the threshold level for a normal population for white noise on the TDT equipment is approximately equal to an attenuation level of -43.3dB, meaning that the 65dBSPL level of the contralateral noise in this experiment equated to a level of about 43.3dBL. This was approximately the same level as that used for the contralateral stimulus in the OAE experiments and was, as mentioned earlier, below the threshold of the acoustic reflex.
In Chapter 6, it was found that the latency of activation of the efferent system was between 7 and 20 ms. The timing of the contralateral stimulus was adjusted to take this into account, so that the OCB would be activated before the arrival of the ipsilateral stimulus. The onset of the contralateral stimulus was therefore set to start 50ms before the onset of the ipsilateral stimuli. Its duration was 400ms and therefore it finished at the same time as the noise in the ipsilateral ear. The contralateral noise was present both for the conditions where the probe tone was present and those where it was not.

The order of testing of the two filter shapes, with and without contralateral noise, was varied between subjects, and if possible, the two runs for each of the five threshold measurements were tested alternately with the five from the other condition. This change in order was carried out to minimise the effects of tiredness and conditioning with time. However, the results were reliable as retest measurements showed (see section 3.1.3.3).

In order to verify that any change observed in the filter shape was not due to acoustic cross-talk via air or bone conduction, two ears were tested with a foam ear plug inserted into the ear canal of the contralateral ear. White noise was presented as above and the filter shape was measured. The ear plug provided about 30dB of attenuation.

7.2.3 Otoacoustic Emission Measurements

TOAEs, SOAEs, DPOAEs and contralateral suppression of TOAEs were performed on all subjects. The methods are the same as those described in sections 5.2.3, 5.2.4 and 5.2.5.

7.3 Results

The auditory filters of 24 subjects were tested at 1kHz with and without contralateral white noise. The mean bandwidth without contralateral noise was 115.59Hz (s.d.=32.03) and with contralateral noise was 138.42Hz (s.d.=34.57). This difference was found to be significant using the Wilcoxon signed ranks test (2 tailed significance=0.016).

The bandwidth of the auditory filter tested during contralateral noise ($BW_{\text{wn}}$) was found to be distributed normally using the one sample Kolmogorov-Smirnov test (2 tailed asymptotic significance=0.31). However, as before, the bandwidth tested without
contralateral noise was found not to be normally distributed (2 tailed asymptotic significance=0.049). The change in bandwidth when contralateral noise was applied was also found to be normally distributed (significance=0.374). The change in bandwidth ($\Delta BW_{wn}$) was defined as

$$\Delta BW_{wn}(Hz) = (\text{bandwidth of filter with contralateral white noise})Hz - (\text{bandwidth of filter without noise})Hz$$

The mean difference in bandwidth was 22.82Hz with a standard deviation of 35.96Hz. The distribution of $\Delta BW_{wn}$ across subjects is shown in Figure 7-1.

**Figure 7-1: Histogram showing the distribution of $\Delta BW_{wn}$ and the normal distribution.**

Analysis showed that p(lower), p(upper), k, r and the symmetry index were all significantly different when contralateral white noise was applied. These results are shown in Table 7-1. P(lower) and p(upper) were both smaller during contralateral stimulation indicating that the slopes of the sides of the filter had got shallower. The significant decrease in k shows that the detection efficiency at the output of the auditory filter was better, and the decrease in r indicates a greater dynamic range under contralateral stimulation. The reduction in SI expresses the shift in the symmetry of the filter towards low frequencies.
Table 7-1: Mean 1kHz auditory filter parameters with and without contralateral white noise and p values for testing the difference of the means.

<table>
<thead>
<tr>
<th>Filter Shape Parameter</th>
<th>No noise</th>
<th>During contralateral white noise</th>
<th>Test for significant difference (p value)</th>
</tr>
</thead>
<tbody>
<tr>
<td>P(lower)</td>
<td>35.85</td>
<td>28.05</td>
<td>0.002**</td>
</tr>
<tr>
<td>P(upper)</td>
<td>27.57</td>
<td>24.68</td>
<td>0.015*</td>
</tr>
<tr>
<td>k</td>
<td>9.08</td>
<td>7.51</td>
<td>0.040*</td>
</tr>
<tr>
<td>r</td>
<td>-43.86</td>
<td>-50.60</td>
<td>0.032*</td>
</tr>
<tr>
<td>Symmetry Index</td>
<td>1.28</td>
<td>1.11</td>
<td>0.003**</td>
</tr>
</tbody>
</table>

The mean auditory filter shape from the 24 subjects tested, is shown graphically in Figure 7-2, along with the mean auditory filter tested during contralateral white noise. The increase in bandwidth during contralateral noise can be seen, and additionally, the greater change in p(lower) than p(upper).

Figure 7-2: Mean auditory filter shape from 24 normal subjects with and without contralateral white noise

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** statistically significant, p<0.01
* statistically significant, p<0.05

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7.3.1 Cross-talk

The mean bandwidth \((n=2)\) of the filter measured during presentation of contralateral white noise whilst the opposite ear was blocked with an ear plug was found to be 97.13Hz, whereas the mean bandwidth for these two ears without the earplug was 104.97Hz. The mean bandwidth measured from these ears without any contralateral noise was 92.07Hz. Therefore, attenuating the input to the contralateral ear reduced the broadening of the filter, indicating that cross-talk was not the sole cause of the changes observed. Certainly, in these cases, 7.84Hz of the widening was not due to cross-talk. It is possible that none of the widening was due to cross-talk, since the earplugs only had an attenuation of about 30dB. This meant that the contralateral signal was still audible and would therefore be activating the efferent system even with the earplug in the ear canal, although to a lesser degree. This may explain why the bandwidth with the ear blocked is not as low as when there was no noise at all, although a small effect of cross-talk cannot be eliminated from these results. However, the interaural attenuation for supra-aural earphones in the 1kHz region is about 60dBLH (Hall and Mueller 1996). As mentioned earlier, the level of the white noise used in this study was found to equate to about 43dBLH, which indicates that there was no cross-over via air or bone conduction.

7.3.2 TOAE magnitude

The mean TOAE magnitude for the 24 subjects tested in this experiment was 11.73dB with a standard deviation of 4.19dB. The TOAE magnitude was not found to be correlated to the bandwidth tested during contralateral white noise (Pearson’s correlation coefficient=0.126, \(p=0.559\)) or with \(\Delta BW_{wn}\) (Pearson’s correlation coefficient=0.225, \(p=0.290\)).

7.3.3 TOAE suppression

A summary of the results of the four TOAE suppression test results on the 24 subjects tested in this experiment are shown in Table 7-2. All suppression tests had mean values that were significantly greater than zero. The Pearson’s correlation coefficients are also
shown for the relationship between the suppression tests and the variables $BW_{\text{wn}}$ and $\Delta BW_{\text{wn}}$.

Table 7-2: Summary of results of TOAE suppression tests and relation to $BW_{\text{wn}}$ and $\Delta BW_{\text{wn}}$

<table>
<thead>
<tr>
<th>TOAE Suppression Stimuli</th>
<th>n</th>
<th>Mean Suppression (dB) and standard deviation</th>
<th>1-sample t-test (test value 0) P value</th>
<th>Correlation with $BW_{\text{wn}}$</th>
<th>Correlation with $\Delta BW_{\text{wn}}$</th>
</tr>
</thead>
<tbody>
<tr>
<td>Ipsi click-Contra noise</td>
<td>24</td>
<td>1.65 (0.77)</td>
<td>&lt;0.001</td>
<td>-0.197</td>
<td>-0.170</td>
</tr>
<tr>
<td>Ipsi click-Contra tone</td>
<td>24</td>
<td>0.44 (0.35)</td>
<td>&lt;0.001</td>
<td>-0.140</td>
<td>-0.064</td>
</tr>
<tr>
<td>Ipsi tone-Contra noise</td>
<td>24</td>
<td>0.48 (0.44)</td>
<td>&lt;0.001</td>
<td>-0.096</td>
<td>-0.338</td>
</tr>
<tr>
<td>Ipsi tone-Contra tone</td>
<td>24</td>
<td>0.14 (0.18)</td>
<td>0.001</td>
<td>-0.219</td>
<td>-0.295</td>
</tr>
</tbody>
</table>

The results show no evidence of a linear relationship between the suppression of TOAEs and the bandwidth of the auditory filter during contralateral white noise. There is also no linear relationship with the change in the bandwidth caused by the contralateral white noise. Scatterplots showing the lack of any other type of relationship between the different tests are shown in Figure 7-3 to Figure 7-6.

Figure 7-3: Scatterplot of the relationship between the suppression of click evoked OAEs by white noise and the 3dB bandwidth of the auditory filter during contralateral noise.
Figure 7-4: Scatterplot of the relationship between the suppression of click evoked OAEs by a 1kHz tone and the 3dB bandwidth of the auditory filter during contralateral noise.

Figure 7-5: Scatterplot of the relationship between the suppression of 1kHz tone evoked OAEs by white noise and the 3dB bandwidth of the auditory filter during contralateral noise.
Figure 7-6: Scatterplot of the relationship between the suppression of click evoked OAEs by a 1kHz tone and the 3dB bandwidth of the auditory filter during contralateral noise

7.3.4 TOAE magnitude and suppression in specific frequency bands

The relationship between the bandwidth of the auditory filter tested during contralateral white noise and the TOAE magnitude and suppression in frequency bands around 1kHz and around 2kHz were examined. The frequency bands were the same as those described in section 5.3.3. The results are shown in Table 7-3.

Table 7-3: Pearson's correlation coefficients for the relationship between bandwidth of the auditory filter during contralateral white noise and TOAE magnitude and suppression in two different frequency regions

<table>
<thead>
<tr>
<th>Stimuli</th>
<th>1kHz Region</th>
<th>2kHz Region</th>
</tr>
</thead>
<tbody>
<tr>
<td>Click evoked TOAE-standard test</td>
<td>0.169</td>
<td>0.002</td>
</tr>
<tr>
<td>Ipsi click-Contra white noise</td>
<td>0.023</td>
<td>-0.265</td>
</tr>
<tr>
<td>Ipsi click-Contra 1kHz tone</td>
<td>0.197</td>
<td>-0.024</td>
</tr>
<tr>
<td>Ipsi 1kHz tone-Contra white noise</td>
<td>-0.088</td>
<td>-0.029</td>
</tr>
<tr>
<td>Ipsi 1kHz tone-Contra 1kHz tone</td>
<td>-0.172</td>
<td>-0.146</td>
</tr>
</tbody>
</table>
Statistical testing found no significant linear correlation between any of the frequency-limited measures and BW\(_{wn}\) at either frequency.

The same analysis was carried out on the change that occurred in the bandwidth (\(\Delta BW_{wn}\)) and the OAE filtered results. These are shown in Table 7-4.

Table 7-4: Pearson’s correlation coefficients and significance values for the relationship between \(\Delta BW_{wn}\) and TOAE magnitude and suppression in two different frequency regions

<table>
<thead>
<tr>
<th>Stimuli</th>
<th>1kHz Region</th>
<th>2kHz Region</th>
</tr>
</thead>
<tbody>
<tr>
<td>Click evoked TOAE-standard test</td>
<td>0.085</td>
<td>0.128</td>
</tr>
<tr>
<td>Ipsi click-Contra white noise</td>
<td>-0.086</td>
<td>-0.185</td>
</tr>
<tr>
<td>Ipsi click-Contra 1kHz tone</td>
<td>0.252</td>
<td>-0.329</td>
</tr>
<tr>
<td>Ipsi 1kHz tone-Contra white noise</td>
<td>-0.374</td>
<td>-0.111</td>
</tr>
<tr>
<td>Ipsi 1kHz tone-Contra 1kHz tone</td>
<td>-0.424*</td>
<td>-0.223</td>
</tr>
</tbody>
</table>

The only result which shows a statistically significant relationship is that for the 1kHz region of the suppression of 1kHz tone evoked emissions suppressed by 1kHz tones (p=0.039). This correlation is negative in direction.

7.3.5 Temporally windowed TOAE suppression

As in section 5.3.4, the suppression was windowed in time to include only the response between 10 and 20ms after the stimulus onset. The windowed suppression was then compared to the bandwidth of the auditory filter during contralateral white noise (BW\(_{wn}\)) in Table 7-5 and to the change in the bandwidth (\(\Delta BW_{wn}\)) in Table 7-6.

* statistically significant, p<0.05
Table 7-5: Pearson's correlation coefficients for the relationship between BW_{wn} and the TOAE suppression data after filtering by frequency and windowing in time (10-20ms after stimulus onset)

<table>
<thead>
<tr>
<th>Stimuli</th>
<th>1kHz Region</th>
<th>2kHz Region</th>
</tr>
</thead>
<tbody>
<tr>
<td>Ipsi click- Contra white noise</td>
<td>-0.044</td>
<td>0.128</td>
</tr>
<tr>
<td>Ipsi click- Contra 1kHz tone</td>
<td>0.078</td>
<td>0.051</td>
</tr>
<tr>
<td>Ipsi 1kHz tone-Contra white noise</td>
<td>0.079</td>
<td>-0.071</td>
</tr>
<tr>
<td>Ipsi 1kHz tone-Contra 1kHz tone</td>
<td>0.272</td>
<td>-0.131</td>
</tr>
</tbody>
</table>

Table 7-6: Pearson's correlation coefficients for the relationship between ΔBW_{wn} and the TOAE suppression data after filtering by frequency and windowing in time (10-20ms after stimulus onset)

<table>
<thead>
<tr>
<th>Stimuli</th>
<th>1kHz Region</th>
<th>2kHz Region</th>
</tr>
</thead>
<tbody>
<tr>
<td>Ipsi click- Contra white noise</td>
<td>-0.073</td>
<td>-0.361</td>
</tr>
<tr>
<td>Ipsi click- Contra 1kHz tone</td>
<td>-0.033</td>
<td>-0.393</td>
</tr>
<tr>
<td>Ipsi 1kHz tone-Contra white noise</td>
<td>0.029</td>
<td>-0.147</td>
</tr>
<tr>
<td>Ipsi 1kHz tone-Contra 1kHz tone</td>
<td>0.469*</td>
<td>-0.071</td>
</tr>
</tbody>
</table>

Again, there are no strong correlations between the suppression measurement and the BW_{wn}. The only correlation which is statistically significant, as before, is that for the linear relationship between the tone evoked TOAEs suppressed by tones, in the 1kHz region and ΔBW_{wn} (p=0.021). However, in this case, the correlation is in the positive direction.

* statistically significant, p<0.05
7.3.6 SOAEs

Of the 24 subjects tested, 12 were found to have SOAEs and 12 were not. All subjects with SOAEs, had at least one SOAE present in the 1kHz region. $BW_w$ and $\Delta BW_w$ were compared between the two groups to see whether there was any difference. The two independent sample t-test was used since the data in the groups were found to be normally distributed.

The mean $BW_w$ was higher in the group without SOAEs (142.9Hz) than the group with (133.9Hz). However, this difference was not found to be significantly different. The mean change in bandwidth $\Delta BW_w$ was also higher in the group with SOAEs (33.0Hz) than in the group without (12.7Hz), although again the difference was not statistically significant.

7.3.7 DPOAEs

Fourteen of the 24 subjects tested in this experiment were also tested with DPOAEs. The seven DPOAE measures described in section 5.3.6 were also used in this experiment to examine the relationship of DPOAE magnitude to the $BW_w$ and the $\Delta BW_w$.

The means and standard deviations for each of the 7 DPOAE measures are shown in Table 7-7 along with Pearson’s correlation coefficients and p values for the relationship between these parameters and $BW_w$ and $\Delta BW_w$.

Table 7-7: Relationship between DPOAE measures and $BW_w$ and $\Delta BW_w$

<table>
<thead>
<tr>
<th>DPOAE Measure</th>
<th>Mean (dB)</th>
<th>Standard Deviation</th>
<th>$BW_w$</th>
<th>$\Delta BW_w$</th>
</tr>
</thead>
<tbody>
<tr>
<td>DP at 1kHz</td>
<td>3.56</td>
<td>8.88</td>
<td>0.220</td>
<td>-0.081</td>
</tr>
<tr>
<td>DP at 2kHz</td>
<td>8.27</td>
<td>7.63</td>
<td>0.576*</td>
<td>0.320</td>
</tr>
<tr>
<td>Mean (745-1501Hz)</td>
<td>5.47</td>
<td>5.34</td>
<td>0.242</td>
<td>0.148</td>
</tr>
<tr>
<td>Low Mean (745-977Hz)</td>
<td>4.37</td>
<td>4.75</td>
<td>0.391</td>
<td>0.362</td>
</tr>
<tr>
<td>High Mean (1013-1501Hz)</td>
<td>6.23</td>
<td>6.21</td>
<td>0.144</td>
<td>0.023</td>
</tr>
<tr>
<td>High-Low</td>
<td>1.80</td>
<td>3.90</td>
<td>-0.259</td>
<td>-0.417</td>
</tr>
<tr>
<td>Standard deviation</td>
<td>3.55</td>
<td>1.17</td>
<td>-0.066</td>
<td>-0.126</td>
</tr>
</tbody>
</table>

* statistically significant, p<0.05
A statistically significant positive linear relation was found between the BW<sub>wt</sub> and the level of the DP at 2kHz (p=0.031). None of the other measures were related in a linear fashion.

### 7.3.8 Other parameters describing the shape of the auditory filter

OAE measurements were compared with more detailed aspects of the auditory filter shape during contralateral noise. The same 5 parameters were examined as in section 5.3.7.

#### 7.3.8.1 TOAE magnitude and suppression

The comparisons between the TOAE magnitude, the four different suppression tests and the five parameters which describe the shape of the auditory filter during contralateral white noise stimulation are shown in Table 7-8.

The results show no evidence of any linear correlation between any of the above variables. Examination of the scatter plots between these variables revealed no other non-linear relationship.

Table 7-8: Correlation of TOAE magnitude and suppression with parameters describing auditory filter shape during contralateral white noise (Pearson correlation coefficient except for results from r which used Spearman’s rho)

<table>
<thead>
<tr>
<th>OAE Measurement</th>
<th>P (lower)</th>
<th>P (upper)</th>
<th>k</th>
<th>r</th>
<th>Symmetry (SI)</th>
</tr>
</thead>
<tbody>
<tr>
<td>TOAE magnitude</td>
<td>-0.153</td>
<td>-0.216</td>
<td>-0.232</td>
<td>-0.318</td>
<td>-0.051</td>
</tr>
<tr>
<td>Ipsi click-Contra white noise</td>
<td>0.166</td>
<td>0.197</td>
<td>0.263</td>
<td>-0.094</td>
<td>0.111</td>
</tr>
<tr>
<td>Ipsi click-Contra 1kHz tone</td>
<td>0.156</td>
<td>0.196</td>
<td>0.166</td>
<td>0.053</td>
<td>0.083</td>
</tr>
<tr>
<td>Ipsi 1kHz tone – contra white noise</td>
<td>0.063</td>
<td>0.110</td>
<td>0.111</td>
<td>0.014</td>
<td>0.022</td>
</tr>
<tr>
<td>Ipsi 1kHz tone – contra 1kHz tone</td>
<td>0.205</td>
<td>0.327</td>
<td>0.105</td>
<td>0.130</td>
<td>0.044</td>
</tr>
</tbody>
</table>
7.3.8.2 TOAE magnitude and suppression in specific frequency bands

The above analysis was carried out in greater detail by analysing frequency bands around 1 and 2kHz as described in section 5.3.3. The correlation coefficients and significance values are shown in Table 7-9. There were no statistically significant linear correlations in either the 1 or the 2kHz region.

Table 7-9: Correlation of TOAE magnitude and suppression at 1 and 2kHz with parameters describing auditory filter shape during contralateral white noise (Pearson correlation coefficient except for results from r which used Spearman’s rho)

<table>
<thead>
<tr>
<th>OAE Measurement</th>
<th>Stimuli</th>
<th>Frequency Band (kHz)</th>
<th>P (lower)</th>
<th>P (upper)</th>
<th>k</th>
<th>r</th>
<th>Symmetry (SI)</th>
</tr>
</thead>
<tbody>
<tr>
<td>TOAE magnitude</td>
<td>1</td>
<td></td>
<td>-0.158</td>
<td>-0.212</td>
<td>-0.197</td>
<td>-0.286</td>
<td>-0.089</td>
</tr>
<tr>
<td></td>
<td>2</td>
<td></td>
<td>-0.074</td>
<td>-0.105</td>
<td>-0.206</td>
<td>-0.187</td>
<td>0.005</td>
</tr>
<tr>
<td>Ipsi click-Contra white noise</td>
<td>1</td>
<td></td>
<td>-0.070</td>
<td>-0.021</td>
<td>0.116</td>
<td>-0.246</td>
<td>-0.099</td>
</tr>
<tr>
<td></td>
<td>2</td>
<td></td>
<td>0.281</td>
<td>0.242</td>
<td>0.175</td>
<td>0.130</td>
<td>0.263</td>
</tr>
<tr>
<td>Ipsi click-Contra 1kHz tone</td>
<td>1</td>
<td></td>
<td>-0.120</td>
<td>-0.181</td>
<td>-0.181</td>
<td>-0.154</td>
<td>-0.069</td>
</tr>
<tr>
<td></td>
<td>2</td>
<td></td>
<td>0.086</td>
<td>0.120</td>
<td>0.010</td>
<td>0.194</td>
<td>0.011</td>
</tr>
<tr>
<td>Ipsi 1kHz tone-Contra white noise</td>
<td>1</td>
<td></td>
<td>0.093</td>
<td>0.105</td>
<td>0.094</td>
<td>0.003</td>
<td>0.062</td>
</tr>
<tr>
<td></td>
<td>2</td>
<td></td>
<td>-0.056</td>
<td>0.061</td>
<td>0.054</td>
<td>-0.118</td>
<td>-0.117</td>
</tr>
<tr>
<td>Ipsi 1kHz tone-Contra 1kHz tone</td>
<td>1</td>
<td></td>
<td>0.189</td>
<td>0.244</td>
<td>0.110</td>
<td>0.146</td>
<td>0.087</td>
</tr>
<tr>
<td></td>
<td>2</td>
<td></td>
<td>0.106</td>
<td>0.258</td>
<td>0.084</td>
<td>0.015</td>
<td>-0.048</td>
</tr>
</tbody>
</table>

7.3.8.3 Temporally windowed TOAE suppression

As in section 5.3.7.3, comparisons were made between the parameters describing the shape of the auditory filter during contralateral white noise, and the TOAE suppression measurement after windowing to include only the response which occurred from 10 to 20 ms after the onset of the stimulus. The results are shown in Table 7-10. The linear correlation between the parameters was not significant except for the relationship between k and the suppression of a 1kHz tone evoked TOAE by a contralateral tone in the 1kHz region of the response (p<0.05). The correlation was negative indicating that an increase in suppression was associated with a decrease in k.
Table 7-10: Correlation of TOAE suppression (windowed from 10-20ms) at 1 and 2kHz with parameters describing auditory filter shape during contralateral white noise (Pearson correlation coefficient except for results from r which used Spearman’s rho)

<table>
<thead>
<tr>
<th>OAE Measurement</th>
<th>Frequency Band (kHz)</th>
<th>P (lower)</th>
<th>P (upper)</th>
<th>k</th>
<th>r</th>
<th>Symmetry (SI)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Ipsi click-Contra white noise</td>
<td>1</td>
<td>0.045</td>
<td>0.056</td>
<td>0.253</td>
<td>-0.234</td>
<td>0.012</td>
</tr>
<tr>
<td></td>
<td>2</td>
<td>-0.180</td>
<td>-0.062</td>
<td>0.195</td>
<td>-0.140</td>
<td>-0.222</td>
</tr>
<tr>
<td>Ipsi click-Contra 1kHz tone</td>
<td>1</td>
<td>0.001</td>
<td>0.062</td>
<td>0.091</td>
<td>-0.095</td>
<td>-0.101</td>
</tr>
<tr>
<td></td>
<td>2</td>
<td>-0.169</td>
<td>0.124</td>
<td>0.411</td>
<td>-0.120</td>
<td>-0.334</td>
</tr>
<tr>
<td>Ipsi 1kHz tone-Contra white noise</td>
<td>1</td>
<td>-0.041</td>
<td>-0.166</td>
<td>-0.041</td>
<td>-0.252</td>
<td>0.076</td>
</tr>
<tr>
<td></td>
<td>2</td>
<td>0.035</td>
<td>0.201</td>
<td>0.215</td>
<td>-0.093</td>
<td>-0.120</td>
</tr>
<tr>
<td>Ipsi 1kHz tone-Contra 1kHz tone</td>
<td>1</td>
<td>-0.227</td>
<td>-0.289</td>
<td>-0.404*</td>
<td>-0.072</td>
<td>-0.147</td>
</tr>
<tr>
<td></td>
<td>2</td>
<td>0.076</td>
<td>0.259</td>
<td>0.207</td>
<td>0.151</td>
<td>-0.113</td>
</tr>
</tbody>
</table>

7.3.8.4 SOAEs

The parameters that describe the shape of the auditory filter during contralateral white noise stimulation were compared in subjects with and without SOAEs. There were 12 subjects in each group. The results are presented in Table 7-11. No statistically significant differences were found between the 2 groups.

Table 7-11: Comparison of the values of the parameters describing the shape of the auditory filter during contralateral white noise in subjects with and without SOAEs (all t-test results except those for r which used Mann Whitney U Test)

<table>
<thead>
<tr>
<th>Filter shape parameter</th>
<th>With SOAEs</th>
<th>Without SOAEs</th>
<th>2-tailed significance</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Mean</td>
<td>Standard deviation</td>
<td>Mean</td>
</tr>
<tr>
<td>P(lower)</td>
<td>28.47</td>
<td>10.34</td>
<td>27.62</td>
</tr>
<tr>
<td>P(upper)</td>
<td>25.35</td>
<td>4.25</td>
<td>24.01</td>
</tr>
<tr>
<td>k</td>
<td>8.15</td>
<td>2.28</td>
<td>6.87</td>
</tr>
<tr>
<td>r</td>
<td>-51.37</td>
<td>19.88</td>
<td>-49.82</td>
</tr>
<tr>
<td>Symmetry Index</td>
<td>1.10</td>
<td>0.25</td>
<td>1.12</td>
</tr>
</tbody>
</table>

* statistically significant, p<0.05
7.3.8.5 DPOAEs

Features of DPOAEs were compared with the five parameters describing the shape of the auditory filter during contralateral white noise. The DPOAE descriptive parameters investigated were the same as those outlined in section 5.3.6.1. Fourteen subjects were tested. The correlation coefficients and significance values are shown in Table 7-12. All quoted values were Pearson’s correlation coefficients, except those for r which were Spearman’s rho correlation coefficients.

A number of filter shape parameters (pi, r and SI) were found to have a significant negative linear correlation with the distortion product measured when f2 was 2kHz (p values 0.037, 0.009, and 0.026 respectively). k was also found to be positively correlated with the ‘high-low’ parameter (p= 0.036). None of the other possible relationships were found to be statistically significant.

Table 7-12: Correlation of DPOAE magnitude measurements with parameters describing auditory filter shape

<table>
<thead>
<tr>
<th>DPOAE Measurement</th>
<th>P (lower)</th>
<th>P (upper)</th>
<th>k</th>
<th>r</th>
<th>Symmetry (SI)</th>
</tr>
</thead>
<tbody>
<tr>
<td>DP at 1kHz</td>
<td>-0.296</td>
<td>-0.071</td>
<td>0.161</td>
<td>-0.130</td>
<td>-0.468</td>
</tr>
<tr>
<td>DP at 2kHz</td>
<td>-0.562*</td>
<td>-0.432</td>
<td>-0.264</td>
<td>-0.670**</td>
<td>-0.591*</td>
</tr>
<tr>
<td>Mean (745-1501Hz)</td>
<td>-0.289</td>
<td>-0.105</td>
<td>0.141</td>
<td>-0.336</td>
<td>-0.422</td>
</tr>
<tr>
<td>Low Mean (745-977Hz)</td>
<td>-0.440</td>
<td>-0.273</td>
<td>-0.108</td>
<td>-0.323</td>
<td>-0.514</td>
</tr>
<tr>
<td>High Mean (1013-1501Hz)</td>
<td>-0.187</td>
<td>-0.008</td>
<td>0.264</td>
<td>-0.323</td>
<td>-0.342</td>
</tr>
<tr>
<td>High-Low</td>
<td>0.252</td>
<td>0.331</td>
<td>0.564*</td>
<td>-0.332</td>
<td>0.093</td>
</tr>
<tr>
<td>Standard deviation</td>
<td>0.138</td>
<td>-0.044</td>
<td>-0.028</td>
<td>-0.138</td>
<td>0.288</td>
</tr>
</tbody>
</table>

* statistically significant, p<0.05
** statistically significant, p<0.01
7.3.9 Influence of other factors

The influence of other various factors on the shape of the auditory filter during contralateral white noise and on the OAE measures were assessed, as in section 5.3.8.

7.3.9.1 Pure tone threshold

The mean pure tone threshold at 1kHz in the test ear of the subjects tested in this experiment was 2.0dBHL with a standard deviation of 3.3dB. There was found to be no significant correlation between this and any of the measures of auditory filter shape during contralateral noise, \( \Delta BW_{wn} \) or with any of the OAE suppression measures.

7.3.9.2 Gender

In this experiment 10 males and 14 females were tested. There was no significant difference between the sexes for the \( BW_{wn} \) or \( \Delta BW_{wn} \).

7.3.9.3 Ear Laterality

12 right ears and 12 left ears were tested. \( BW_{wn} \) and \( \Delta BW_{wn} \) were compared for the two ears but no significant differences were found.

7.3.9.4 Threshold in noise

The ability to detect a 1kHz tone in the presence of an ipsilateral narrow band masker and a contralateral white noise was compared to the TOAE suppression results. The threshold was taken from the first condition of the filter shape measurement where there was no notch in the ipsilateral masker. No relationship was found between suppression of TOAEs and the threshold of detection in bilateral noise.

7.3.9.5 Age

The mean age of the 24 subjects tested was 23.17 years with a standard deviation of 2.58 years. The age of the subjects was not found to be correlated with \( BW_{wn} \) or \( \Delta BW_{wn} \)
7.3.9.6 Order of testing

The filter shape threshold measurements consisted of two sets of five runs each. These sets were alternated with and without contralateral noise. The first set was randomly assigned to be a ‘with’ or ‘without’ contralateral noise test. Fourteen of the 24 subjects were tested with no contralateral noise first, 8 were tested with contralateral noise first and 2 subjects had the tests on different occasions. Excluding the 2 subjects who were tested on separate days, the data from the other subjects was compared to investigate whether the order of testing had an influence over the results.

The order of testing was found to have no significant effect over the bandwidth measured with or without contralateral noise.

7.4 Discussion

The main finding to come from this study is the statistically significant increase in width of the 1kHz auditory filter under contralateral white noise stimulation. The mean increase in the 3dB bandwidth of the filter was 22.82Hz from testing a group of 24 audiometrically normal subjects. The increase in bandwidth was due to both the lower and the upper slopes of the filter becoming significantly shallower. This was also accompanied by an increase in the dynamic range (r) of the filter and an increase in the detection efficiency at the output of the filter (k).

These effects seem likely to be due to the activation of the efferent system by the contralateral noise. The broad band stimulation would activate efferent fibres of wide ranging best frequency. The medial efferent fibres activated are hypothesised to facilitate a damping of the motion of the basilar membrane. In this case therefore, damping would occur across a wide range of frequencies, at the basilar membrane of the contralateral cochlea. This damping would affect the fine balance of amplification of basilar membrane motion acting in order to maintain the high degree of tuning commonly observed. Thus, with this kind of wide-ranging damping, one could expect the tuning to deteriorate. In order for the tuning to be enhanced by medial efferent damping, the action of the damping would have to very frequency specific, for example,
just in the side skirts of the filter leaving the centre frequency to receive greater amplification than the surrounding frequencies.

A decrease in k was observed during contralateral noise. This result indicates that the efferent system may in fact alter the detection efficiency at the filter output and therefore the performance of the auditory system, by changing the threshold signal to noise ratio. It is unclear how this change may be facilitated physiologically. The increase in r, the dynamic range, during contralateral noise suggests that damping of the BM motion may emphasise the difference between the activation at the centre frequency of the filter and the frequencies outside the filter.

Past studies have also shown a broadening of tuning during efferent stimulation. Auditory nerve fibre tuning curves were found to be wider with electrical efferent stimulation (Guinan and Gifford 1988b) and contralateral acoustic stimulation widened the DP suppression tuning curves (Williams and Brown 1995). Other studies have been less conclusive about this issue. For example, Murugasu and Russell (1996) found that electrical stimulation of the OCB caused the BM tuning curve response to widen in some cases but not others. This is in keeping with the present results since not all subjects demonstrated the reduction in tuning.

The changes in quality of tuning were not, in general, found to be related to the degree of efferent function measured using otoacoustic emissions or to the unsuppressed OAE levels. A small number of significant correlation coefficients were found, but the results were not consistent and therefore likely to be due to chance. The DP level measured at 2kHz was found to be positively related to the bandwidth during contralateral white noise stimulation. Correlations were also found for p(lower), r and SI. This was not surprising since these parameters are related to each other and to the bandwidth. This result indicated that the cochlear amplifier in the region of 2kHz was involved in determining the shape of the filter during contralateral white noise.

This raises the question as to why the seemingly efferent induced broadening of the auditory filter is not related to the OAE results. The first point to consider is the possibility that the filter broadening was not mediated by the efferent system. The level of the stimulus would indicate that cross over by air or bone conduction was unlikely, and blocking the ear reduced the widening effect.
Auditory filter shape measurements have shown that an increase in level leads to the lower side of the filter becoming shallower, but the upper side becoming steeper (Glasberg and Moore 1990). In this experiment, the upper side of the filter was found to become shallower.

Another feature of the results that does not fit with a crossover explanation is that the detection efficiency (k) was found to increase during contralateral stimulation. Detection efficiencies should be expected to remain constant for a particular frequency measured at different levels (Rosen and Baker 1994), thus indicating that we are observing an effect more complex than cross-over.

Activation of the middle ear reflexes is also unlikely to be the cause of the widening. The crossed acoustic reflex threshold for BBN in a normal population was found to be 82 dBSPL (Hall and Weaver 1979) which is well above the level that is used here. In any case, tuning is generally found to broaden with increasing level. Attenuation of the signal entering the cochlea by the middle ear reflex would effectively reduce the level and therefore theoretically produce a narrowing of the filter. It is therefore unlikely that this is the cause of the changes to the filter shape encountered.

There is a possibility that the contralateral signal may be causing masking of the ipsilateral signal, more centrally, such as in the superior olivary complex. Central masking effects from contralateral sounds have been found to be very small (Zwislocki et al. 1967). However, in order to examine this, a test was devised whereby there was a delay between the presentation of the contralateral sound and the ipsilateral stimuli so that the signals would not coincide as they travelled centrally. Unfortunately, this made the task too confusing for the subjects tested and no reliable results could be recorded. There is little past work on the differences between central masking and the efferent effects, and the role of central masking is unknown in this study. However, given the damping effect that contralateral efferent stimulation has on OAEs it seems unlikely that the efferent system has no influence over the filter shapes measured during contralateral noise, whether there is some additional central masking present or not. This may however be an extra variable in the psychoacoustic test that meant that the results did not relate to the OAE tests.

Another possibility is that the activation of the efferent system caused a change in the best frequency of the auditory filter. This may make the modelled filter shape at 1kHz
appear wider and the fitting procedure would not give an accurate estimation of the width. Any relationship to the efferent activity measured using OAEs may therefore not show up. A shift in frequency of 500Hz was noted by Murugasu and Russell (1996) in some of the BM tuning curves measured.

Investigation of other factors (gender, age, ear tested, order of testing) found no evidence that the results may be confounded by any other variable.

Other work has shown relationships between detection of tones in noise (during contralateral noise) and efferent function (Micheyl and Collet 1996). However, this correlation was only found at 2kHz and not at 1kHz. This may show possible differences in efferent function along the length of the cochlea. This issue will be examined later (Chapter 9).

Both the OAE tests and the auditory filter tests should have been measuring the effect of the same group of efferent fibres i.e. those that act on the ipsilateral ear after activation from the contralateral ear. However, it is possible that the effect of white noise on the filter is too generalised in frequency. For this reason, in the next chapter, more frequency specific contralateral stimulation was used.

### 7.5 Conclusion

This study shows that contralateral white noise alters the shape of the auditory filter in the opposite ear of audiometrically normal subjects. This was in keeping with the results from chapter 5, which showed greater efferent suppression of DPOAEs associated with a widening rather than a narrowing of the filter. It seems likely that the damping action of efferent system on the motion of the BM is involved. However, the broadening effect is not related to the contralateral suppression of transient otoacoustic emissions, which is assumed to be due to the action of the medial efferent system. It is unclear from this study whether the lack of correspondence between the results is indicative of differing underlying physiological processes or whether some feature of the measurement techniques has masked the relationship.
Chapter 8

Efferent Activation with Narrow Band Noise during Filter Shape Measurement
8. Efferent Activation with Narrow Band Noise during Filter Shape Measurement

8.1 Introduction

In the previous chapter, the effect of contralateral white noise on the shape of the filter was examined. This was tested in relation to the effectiveness of the efferent system as measured via OAEs. The white noise stimulation produced a broadening of the filter, an effect that was suggested to be due to activation of the efferent system.

The change that the white noise caused however, was not, in general, found to be related to the OAE measurements. The aim of this chapter therefore was to investigate in further detail, changes in the filter shape due to contralateral sound and its possible relationship to the functioning of the efferent system. It is possible that the activation caused by the white noise was too widespread in frequency to show a relationship with the OAE studies. In this chapter, we examine the effect of selective stimulation of the efferent system by narrow bands of contralateral noise.

In the following experiments, both ears are stimulated by narrow band signals. This bears more resemblance to the 'real-life' situation, where one attempts to hear a signal in background noise. The majority of the time, both ears will have similar input. Therefore, if in fact, the efferent input from the opposite ear aids in signal detection, then in most cases this will be when the efferent system is stimulated at similar frequencies to those that surround the target signal in the ipsilateral ear. As mentioned in section 2.3.2.2, contralateral efferent action seems to be frequency specific at mid frequencies (Chery Croze et al. 1993; Rossi et al. 1993; Veuillet et al. 1991). The auditory filter at 1kHz was being examined and thus narrow bands of noise around this frequency are likely to have most effect in altering the filter shape. The filter shape was therefore tested with and without contralateral narrow band noise at 1kHz, 2kHz or 500Hz and compared with OAE results.
8.2 Methods

8.2.1 Subjects

12 subjects were tested with contralateral (1/3 octave) narrow band noise at 1kHz and 2kHz during measurement of the auditory filter at 1kHz. Six of the subjects were female and six were male. Their ages ranged from 19 to 29 years with a mean of 23.3 and a standard deviation of 2.8. Eleven of these subjects were additionally tested with contralateral (1/3 octave) narrow band noise at 500Hz.

All the subjects had normal pure tone audiometry thresholds, tympanometry and acoustic reflex thresholds as described in sections 3.3, 3.4 and 3.5. As before, the ear to be tested was chosen randomly which resulted in 50% being tested on each side.

8.2.2 Measurement of Auditory Filter Shape

The shape of the auditory filter at 1kHz was measured using the same method as that described in section 3.1. The contralateral narrow band noise stimuli were generated using the WG1 module of the TDT equipment to produce white noise and control the duration (400ms, starting 50ms before the ipsilateral stimuli) as in section 7.2.2. The white noise was then passed through a programmable filter (PF1) and band limited to include only the frequencies within a 1/3 octave band around each of the 3 frequencies. The levels of the 1kHz, 2kHz and 500Hz narrow band noise stimuli were 47.5, 51 and 44.5dB SPL respectively. The thresholds of 10 ears were tested for these narrow band noises produced by the TDT equipment and compared to the thresholds from the same stimuli produced by an audiometer. The mean thresholds on the TDT equipment were -37.8, -40 and -30dB attenuation for the narrow band noises at 1kHz, 2kHz and 500Hz respectively. The mean thresholds for the same ears tested on the audiometer were 2, -1 and 1.5dBHL respectively. Therefore considering the narrow band noise levels in terms of dBHL for a normal population, the 1kHz, 2kHz and 500Hz levels become equivalent to 39.8dBHL, 39dBHL and 31.5dBHL respectively. The 1 and 2kHz narrow band noises were therefore at a similar level to the contralateral white noise stimuli used in the previous OAE and filter shape measurements, and the 500Hz was approximately 7.5dB quieter.
Filter shapes were measured during contralateral narrow band noise to examine whether narrow band efferent activation may relate any more strongly than white noise to the efferent suppression measured via OAEs. The difference between these filter shapes and those measured without any contralateral stimulation was also considered.

8.2.3 Otoacoustic Emission Measurements

TOAEs, SOAEs, DPOAEs and contralateral suppression of TOAEs were performed on all subjects. The methods are the same as those described in sections 5.2.3, 5.2.4 and 5.2.5.

8.3 Results

The mean and standard deviations for the 3dB bandwidth of the auditory filter tested during various contralateral narrow band noises are shown in Table 8-1. The bandwidths for the same subjects during no contralateral stimulation and during contralateral white noise are also shown, for purposes of comparison. This data is also shown graphically in Figure 8-1. The data for all the test conditions shown was found to be normally distributed using the Kolmogorov-Smirnov test.

Table 8-1: Mean and standard deviations for 3dB bandwidth of 1kHz auditory filter tested during contralateral narrow band noise (NBN) at 1kHz, 2kHz and 500Hz. Bandwidths for same subjects during no contralateral noise and during white noise are also shown for comparison.

<table>
<thead>
<tr>
<th>1kHz Filter shape test condition</th>
<th>N</th>
<th>Mean bandwidth (Hz)</th>
<th>Standard deviation (Hz)</th>
</tr>
</thead>
<tbody>
<tr>
<td>1kHz NBN</td>
<td>12</td>
<td>115.3</td>
<td>33.2</td>
</tr>
<tr>
<td>2kHz NBN</td>
<td>12</td>
<td>102.8</td>
<td>18.9</td>
</tr>
<tr>
<td>500Hz NBN</td>
<td>11</td>
<td>114.4</td>
<td>29.3</td>
</tr>
<tr>
<td>No noise</td>
<td>12</td>
<td>110.4</td>
<td>30.6</td>
</tr>
<tr>
<td>White noise</td>
<td>12</td>
<td>131.8</td>
<td>35.6</td>
</tr>
</tbody>
</table>
The mean bandwidth for the filter measured during contralateral narrow band noise (NBN) at 1kHz and at 500Hz are both slightly greater than the bandwidth with no contralateral noise, whereas the mean bandwidth during 2kHz NBN was slightly lower. However, it can be seen that the mean bandwidths measured during any of the contralateral NBN conditions are not markedly different from the bandwidth measured with no contralateral noise. The paired sample t-test showed that the differences were not significant. There were also no significant differences between the bandwidths measured during the three different frequencies. Analysis of variance also showed no significant differences between the groups.

The difference in Hz between the 3dB bandwidth measured without contralateral NBN and with contralateral NBN, was calculated for each subject at each NBN frequency. The difference parameters were defined as follows,

\[ \Delta BW_{1\text{NBN}}(\text{Hz}) = \text{(bandwidth of filter with contralateral 1kHz NBN)}\text{Hz} - \text{(bandwidth of filter without noise)}\text{Hz} \]

\[ \Delta BW_{2\text{NBN}}(\text{Hz}) = \text{(bandwidth of filter with contralateral 2kHz NBN)}\text{Hz} - \text{(bandwidth of filter without noise)}\text{Hz} \]

\[ \Delta BW_{500\text{NBN}}(\text{Hz}) = \text{(bandwidth of filter with contralateral 500Hz NBN)}\text{Hz} - \text{(bandwidth of filter without noise)}\text{Hz} \]
The mean and standard deviations of the difference in the bandwidths are shown in Table 8-2. All 3 groups of data could be approximated by a normal distribution and were analysed to see if the data was significantly different from zero. This was not found to be the case and the p values are also shown in Table 8-2.

Table 8-2: Summary of difference parameter values for bandwidth with and without contralateral narrow band noise, including 1 sample t test significance values (test value=0)

<table>
<thead>
<tr>
<th>Difference parameter</th>
<th>Mean</th>
<th>Standard deviation</th>
<th>p-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>ΔBW1nbn</td>
<td>4.88</td>
<td>39.10</td>
<td>0.674</td>
</tr>
<tr>
<td>ΔBW2nbn</td>
<td>-7.60</td>
<td>34.29</td>
<td>0.459</td>
</tr>
<tr>
<td>ΔBW500nbn</td>
<td>2.97</td>
<td>44.27</td>
<td>0.829</td>
</tr>
</tbody>
</table>

The mean bandwidth during contralateral white noise in these subjects was greater than during NBN. When comparing the NBN results with the white noise results, the paired sample t-test showed that the differences were not significant for the NBN at 1kHz and at 500Hz but were significant at 2kHz (p=0.044).

8.3.1 TOAE magnitude

The mean TOAE magnitude for this group of 12 subjects was 10.7dB with a standard deviation of 3.9dB. Pearson’s correlation coefficients were calculated to compare the bandwidth of the auditory filter during NBN to the TOAE magnitude. The bandwidth of the filters tested with 1kHz NBN (BW1nbn) and with 2khz NBN (BW2nbn) were not found to have a statistically significant linear correlation. However, the TOAE magnitude did have a negative linear correlation (-0.724, p=0.012) with the bandwidth measured during contralateral 500Hz NBN (BW500nbn). TOAE magnitude was not correlated with ΔBW1nbn, ΔBW2nbn or ΔBW500nbn.
8.3.2 TOAE suppression

The mean, standard deviations and significance values for the 1 sample t test (test value=0) are shown in Table 8-3 for the four different TOAE suppression tests. The results shown are for the 12 subjects tested with 1kHz and 2kHz NBNs. The results differed little with the one subject removed who did not participate in the 500Hz NBN test. All suppression values are significantly greater than zero except the ipsi 1kHz tone evoked OAE suppressed by a contra 1kHz tone. All were found to be distributed normally.

Table 8-3: TOAE suppression results

<table>
<thead>
<tr>
<th>TOAE suppression test</th>
<th>Mean suppression (dB)</th>
<th>Standard deviation</th>
<th>1 sample test p value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Ipsi click - contra white noise</td>
<td>1.667</td>
<td>0.780</td>
<td>0.000</td>
</tr>
<tr>
<td>Ipsi click - contra 1kHz tone</td>
<td>0.492</td>
<td>0.387</td>
<td>0.001</td>
</tr>
<tr>
<td>Ipsi 1kHz tone - contra white noise</td>
<td>0.500</td>
<td>0.594</td>
<td>0.014</td>
</tr>
<tr>
<td>Ipsi 1kHz tone - contra 1kHz tone</td>
<td>0.142</td>
<td>0.228</td>
<td>0.054</td>
</tr>
</tbody>
</table>

The Pearson correlation coefficients were calculated in order to examine the possible existence of a linear relationship between the suppression results and the 3dB bandwidth results during contralateral NBN and also the difference in the bandwidth in the noise and the no noise situations. These results are shown in Table 8-4. None of the correlations were statistically significant.

Scatterplots of this data revealed no other non-linear relationships. These are shown in Appendix 4.
Table 8-4: Pearson’s correlation coefficients for the relationship between 4 measures of TOAE suppression and the 3dB bandwidth of auditory filters measured during contralateral NBN or the change in bandwidth from the filter measured with no noise.

<table>
<thead>
<tr>
<th>Suppression Test</th>
<th>Pearson’s Correlation Coefficients</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>BW_{1\text{nbn}}</td>
</tr>
<tr>
<td>Ipsi click – Contra white noise</td>
<td>-0.073</td>
</tr>
<tr>
<td>Ipsi click – Contra 1kHz tone</td>
<td>-0.151</td>
</tr>
<tr>
<td>Ipsi 1kHz tone – Contra white noise</td>
<td>0.297</td>
</tr>
<tr>
<td>Ipsi 1kHz tone – Contra 1kHz tone</td>
<td>-0.169</td>
</tr>
</tbody>
</table>

8.3.3 TOAE magnitude and suppression in specific frequency bands

The TOAE magnitude and suppression in specific frequency bands around 1kHz and 2kHz was examined as in section 5.3.3. This was compared to the 3dB bandwidth during NBN and the difference in the bandwidth between the NBN and no noise conditions. The existence of a possible linear relationship was examined by calculating the Pearson’s correlation coefficients.

The results for the filter tested during contralateral 1kHz NBN are shown in Table 8-5. There were no significant correlation coefficients.

Table 8-5: Pearson’s correlation coefficients for the relationship between BW_{1\text{nbn}} and \Delta BW_{1\text{nbn}} and TOAE magnitude and suppression in 2 frequency bands

<table>
<thead>
<tr>
<th>Stimuli</th>
<th>1kHz Region</th>
<th>2kHz Region</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>BW_{1\text{nbn}}</td>
<td>\Delta BW_{1\text{nbn}}</td>
</tr>
<tr>
<td>Standard TOAE</td>
<td>0.210</td>
<td>0.174</td>
</tr>
<tr>
<td>Ipsi click- Contra white noise</td>
<td>-0.059</td>
<td>-0.449</td>
</tr>
<tr>
<td>Ipsi click- Contra 1kHz tone</td>
<td>-0.028</td>
<td>-0.016</td>
</tr>
<tr>
<td>Ipsi 1kHz tone- Contra white noise</td>
<td>0.217</td>
<td>-0.180</td>
</tr>
<tr>
<td>Ipsi 1kHz tone- Contra 1kHz tone</td>
<td>-0.208</td>
<td>-0.421</td>
</tr>
</tbody>
</table>
The results for the filter tested during 2kHz contralateral NBN are shown in Table 8-6. The only correlation which was found to be significant, at the p<0.05 level, was that of ΔBW_{2nbn} and the suppression of click evoked TOAEs by white noise in the 1kHz region (p=0.014). The correlation was in the negative direction.

**Table 8-6: Pearson’s correlation coefficients for the relationship between BW_{2nbn} and ΔBW_{2nbn} and TOAE magnitude and suppression in 2 frequency bands**

<table>
<thead>
<tr>
<th>Stimuli</th>
<th>1kHz Region</th>
<th>2kHz Region</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>BW_{2nbn}</td>
<td>ΔBW_{2nbn}</td>
</tr>
<tr>
<td>Standard TOAE</td>
<td>-0.502</td>
<td>-0.281</td>
</tr>
<tr>
<td>Ipsi click-Contra white noise</td>
<td>-0.421</td>
<td>-0.686*</td>
</tr>
<tr>
<td>Ipsi click-Contra 1kHz tone</td>
<td>0.111</td>
<td>0.069</td>
</tr>
<tr>
<td>Ipsi 1kHz tone-Contra white noise</td>
<td>-0.111</td>
<td>-0.476</td>
</tr>
<tr>
<td>Ipsi 1kHz tone-Contra 1kHz tone</td>
<td>0.474</td>
<td>-0.017</td>
</tr>
</tbody>
</table>

Table 8-7 shows the Pearson’s correlation coefficients for the filter tested with contralateral 500Hz NBN.

**Table 8-7: Pearson’s correlation coefficients for the relationship between BW_{500nbn} and ΔBW_{500nbn} and TOAE magnitude and suppression in 2 frequency bands**

<table>
<thead>
<tr>
<th>Stimuli</th>
<th>1kHz Region</th>
<th>2kHz Region</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>BW_{500nbn}</td>
<td>ΔBW_{500nbn}</td>
</tr>
<tr>
<td>Standard TOAE</td>
<td>-0.523</td>
<td>-0.345</td>
</tr>
<tr>
<td>Ipsi click-Contra white noise</td>
<td>0.079</td>
<td>-0.307</td>
</tr>
<tr>
<td>Ipsi click-Contra 1kHz tone</td>
<td>-0.288</td>
<td>-0.239</td>
</tr>
<tr>
<td>Ipsi 1kHz tone-Contra white noise</td>
<td>-0.246</td>
<td>-0.489</td>
</tr>
<tr>
<td>Ipsi 1kHz tone-Contra 1kHz tone</td>
<td>-0.315</td>
<td>-0.430</td>
</tr>
</tbody>
</table>

** statistically significant at p<0.01 level
* statistically significant at p<0.05 level
No significant correlation coefficients were found except that for the relationship between the TOAE amplitude, when measured in the ½ octave around 2kHz, and the BW\textsubscript{500nbm}. The correlation was strongly negative and was highly significant (p=0.003). Examining the dependence graphically (Figure 8-2), it is clear that the data points are not distributed evenly. Excluding the one outlying point, the others are clustered such that those with little TOAE energy in the 2kHz region have high bandwidths and those with a larger amount of 2kHz energy have narrower bandwidths. The strength of the correlation and the significance is a good indication of a real relationship, even given the large number of correlation calculations performed (i.e. 10 calculations for BW\textsubscript{500nbm}, therefore should consider p values less than 0.005 significant). The equivalent correlation for ΔBW\textsubscript{500nbm} was also negative and significant. Since BW\textsubscript{500nbm} and ΔBW\textsubscript{500nbm} are related, it is not surprising that they are both significant.

**Figure 8-2: TOAE magnitude in ½ octave around 2kHz compared to 3dB bandwidth of 1kHz auditory filter measured during contralateral 500Hz narrow band noise**

8.3.4 Temporally windowed TOAE suppression

The filtered suppression data was windowed to include only that part of the response recorded between 10 and 20ms after the onset of the stimuli (as in section 5.3.4). Table 8-8 shows the Pearson correlation coefficients calculated from the comparison of this OAE data with the auditory filter bandwidths during 1kHz contralateral NBN. The
difference between the bandwidths with and without noise was also compared to the OAE data.

Table 8-8: Pearson’s correlation coefficients for the relationship between \( BW_{1nbn} \) and \( \Delta BW_{1nbn} \) and TOAE suppression in 2 frequency bands after temporally windowing (10-20ms)

<table>
<thead>
<tr>
<th>Stimuli</th>
<th>1kHz Region</th>
<th></th>
<th>2kHz Region</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>( BW_{1nbn} )</td>
<td>( \Delta BW_{1nbn} )</td>
<td>( BW_{1nbn} )</td>
<td>( \Delta BW_{1nbn} )</td>
</tr>
<tr>
<td>Ipsi click-Contra white noise</td>
<td>0.007</td>
<td>-0.576</td>
<td>-0.102</td>
<td>-0.232</td>
</tr>
<tr>
<td>Ipsi click-Contra 1kHz tone</td>
<td>-0.371</td>
<td>-0.340</td>
<td>-0.601</td>
<td>-0.861*</td>
</tr>
<tr>
<td>Ipsi 1kHz tone-Contra white noise</td>
<td>0.178</td>
<td>-0.109</td>
<td>0.120</td>
<td>0.238</td>
</tr>
<tr>
<td>Ipsi 1kHz tone-Contra 1kHz tone</td>
<td>0.186</td>
<td>0.314</td>
<td>-0.570</td>
<td>-0.419</td>
</tr>
</tbody>
</table>

Generally, the variables were not found to be related in a linear fashion. The only exception however, is the negative correlation (-0.861) between \( \Delta BW_{1nbn} \) and the click evoked TOAEs suppressed by a 1kHz tone, filtered around 2kHz and windowed (p=0.028).

The results from the equivalent calculations with the filter shape data measured during 2kHz contralateral NBN, are shown in Table 8-9. The only statistically significant correlation was that of \( \Delta BW_{2nbn} \) vs. the suppression of click evoked TOAEs by contralateral white noise in the 1kHz region. In this case the correlation coefficient was -0.744, with a p value of 0.034.

* statistically significant, p<0.05
Table 8-9: Pearson's correlation coefficients for the relationship between $\text{BW}_{2\text{nbn}}$ and $\Delta\text{BW}_{2\text{nbn}}$ and TOAE magnitude and suppression in 2 frequency bands after temporally windowing (10-20ms)

<table>
<thead>
<tr>
<th>Stimuli</th>
<th>1kHz Region</th>
<th></th>
<th>2kHz Region</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>$\text{BW}_{2\text{nbn}}$</td>
<td>$\Delta\text{BW}_{2\text{nbn}}$</td>
<td>$\text{BW}_{2\text{nbn}}$</td>
<td>$\Delta\text{BW}_{2\text{nbn}}$</td>
</tr>
<tr>
<td>Ipsi click-Contra white noise</td>
<td>-0.288</td>
<td>-0.744*</td>
<td>-0.398</td>
<td>-0.291</td>
</tr>
<tr>
<td>Ipsi click-Contra 1kHz tone</td>
<td>-0.189</td>
<td>-0.007</td>
<td>0.686</td>
<td>0.014</td>
</tr>
<tr>
<td>Ipsi 1kHz tone-Contra white noise</td>
<td>-0.293</td>
<td>-0.457</td>
<td>0.097</td>
<td>0.206</td>
</tr>
<tr>
<td>Ipsi 1kHz tone-Contra 1kHz tone</td>
<td>-0.364</td>
<td>-0.023</td>
<td>0.497</td>
<td>0.309</td>
</tr>
</tbody>
</table>

The data for the auditory filter measured during 500Hz contralateral NBN is shown in Table 8-10. None of the correlation coefficients were significant except $\text{BW}_{500\text{nbn}}$ vs. the suppression of click evoked TOAEs by a 1kHz tone in the 1kHz region. The correlation coefficient was 0.778 and the p value was 0.023.

Table 8-10: Pearson's correlation coefficients for the relationship between $\text{BW}_{2\text{nbn}}$ and $\Delta\text{BW}_{2\text{nbn}}$ and TOAE magnitude and suppression in 2 frequency bands after temporally windowing (10-20ms)

<table>
<thead>
<tr>
<th>Stimuli</th>
<th>1kHz Region</th>
<th></th>
<th>2kHz Region</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>$\text{BW}_{500\text{nbn}}$</td>
<td>$\Delta\text{BW}_{500\text{nbn}}$</td>
<td>$\text{BW}_{500\text{nbn}}$</td>
<td>$\Delta\text{BW}_{500\text{nbn}}$</td>
</tr>
<tr>
<td>Ipsi click-Contra white noise</td>
<td>0.185</td>
<td>-0.307</td>
<td>-0.085</td>
<td>-0.159</td>
</tr>
<tr>
<td>Ipsi click-Contra 1kHz tone</td>
<td>0.778*</td>
<td>0.502</td>
<td>0.386</td>
<td>-0.200</td>
</tr>
<tr>
<td>Ipsi 1kHz tone-Contra white noise</td>
<td>-0.452</td>
<td>-0.547</td>
<td>-0.604</td>
<td>-0.209</td>
</tr>
<tr>
<td>Ipsi 1kHz tone-Contra 1kHz tone</td>
<td>-0.211</td>
<td>-0.023</td>
<td>0.308</td>
<td>0.281</td>
</tr>
</tbody>
</table>

8.3.5 SOAEs

Of the 12 subjects tested in this experiment, 5 had SOAEs (all of whom had SOAE present in the 1kHz region). $\text{BW}_{1\text{nbn}}$, $\text{BW}_{2\text{nbn}}$ and $\text{BW}_{500\text{nbn}}$ were examined using the

* statistically significant, p<0.05
independent samples t-test to see whether there was any difference between those subjects with SOAEs and those without. There was only a significant difference for $BW_{300\text{nb}}$, where the group with SOAEs had a mean bandwidth of 94.48Hz (s.d.=8.12Hz), and those without SOAEs had a mean bandwidth of 131.04Hz (s.d.=30.60Hz). Levene’s test showed the variances to be unequal ($p=0.003$) and the resulting t-test statistics, comparing the means, were found to have a p value of 0.032.

$\Delta BW_{1\text{nb}}$, $\Delta BW_{2\text{nb}}$ and $\Delta BW_{500\text{nb}}$ were also examined in a similar fashion. Again, only $\Delta BW_{500\text{nb}}$ was found to have significantly different means for the groups with and without SOAEs. The variances were found to be equal ($p=0.534$, from Levene’s test) and the t-test comparing the means gave a p value of 0.046. The mean from the group with SOAEs was $-25.25Hz$ (s.d.=36.83Hz) and the mean from the group without SOAEs was 26.48Hz (s.d.=37.07Hz).

### 8.3.6 DPOAEs

A subset of 9 of the 12 subjects tested in this experiment were tested with DPOAEs. As in section 5.3.6.1, 7 parameters derived from the DP response were examined. Their mean and standard deviation values for the 9 subjects are shown in Table 8-11.

<table>
<thead>
<tr>
<th>DPOAE Measure</th>
<th>Mean (dB)</th>
<th>Standard Deviation</th>
</tr>
</thead>
<tbody>
<tr>
<td>DP at 1kHz</td>
<td>6.02</td>
<td>7.08</td>
</tr>
<tr>
<td>DP at 2kHz</td>
<td>10.47</td>
<td>5.22</td>
</tr>
<tr>
<td>Mean (745-1501Hz)</td>
<td>6.68</td>
<td>4.95</td>
</tr>
<tr>
<td>Low Mean (745-977Hz)</td>
<td>4.91</td>
<td>4.60</td>
</tr>
<tr>
<td>High Mean (1013-1501Hz)</td>
<td>7.92</td>
<td>5.65</td>
</tr>
<tr>
<td>High-Low</td>
<td>3.07</td>
<td>3.47</td>
</tr>
<tr>
<td>Standard deviation</td>
<td>3.36</td>
<td>1.05</td>
</tr>
</tbody>
</table>

Pearson’s correlation coefficients were calculated for the linear relationship between these 7 DPOAE measures and $BW_{1\text{nb}}$, $BW_{2\text{nb}}$, $BW_{500\text{nb}}$, $\Delta BW_{1\text{nb}}$, $\Delta BW_{2\text{nb}}$ and $\Delta BW_{500\text{nb}}$ (Table 8-12).

It can be seen that $\Delta BW_{1\text{nb}}$ correlates significantly with many of the DPOAE measures. The p values are 0.001, 0.041, 0.008, 0.031 and 0.009 for the measures DP at 1kHz, DP
at 2kHz, mean, mean low frequency and mean high frequency respectively. Obviously, many of these variables are related to one another, but nevertheless, the strength of the correlation indicates that there is some negative relation between the level of DPOAE in this region and the change encountered in the bandwidth of the auditory filter when contralateral 1kHz NBN is added.

The only other statistically significant correlation was that between $BW_{2\text{nnb}}$ and the standard deviation of the DP level. The correlation was negative in direction, indicating that the greater the variability or irregularity of the DP level, the smaller the bandwidth of the filter measured during 2kHz NBN.

Table 8-12: Pearson’s correlation coefficients for relationship between DPOAE measures and auditory filter shape bandwidth (and change in bandwidth) during contralateral NBN

<table>
<thead>
<tr>
<th>DPOAE Measure</th>
<th>Pearson’s Correlation Coefficients</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>$BW_{1\text{nnb}}$</td>
</tr>
<tr>
<td>DP at 1kHz</td>
<td>-0.340</td>
</tr>
<tr>
<td>DP at 2kHz</td>
<td>-0.257</td>
</tr>
<tr>
<td>Mean (745-1501Hz)</td>
<td>-0.666</td>
</tr>
<tr>
<td>Low Mean (745-977Hz)</td>
<td>-0.649</td>
</tr>
<tr>
<td>High Mean (1013-1501Hz)</td>
<td>-0.622</td>
</tr>
<tr>
<td>High-Low</td>
<td>-0.120</td>
</tr>
<tr>
<td>Standard deviation</td>
<td>0.371</td>
</tr>
</tbody>
</table>

8.3.7 Other parameters describing the shape of the auditory filter

The OAE results were then compared with more detailed parameters describing the shape of the auditory filters measured during contralateral NBN. The five filter shape parameters have been described earlier, in section 5.3.7.

** statistically significant, p<0.01
* statistically significant, p<0.05
8.3.7.1 TOAE magnitude and suppression

The TOAE standard test magnitude and the four suppression test results were compared with the five measures describing the shape of the auditory filter. The results of the Pearson’s correlations performed are shown in Table 8-13 for the filter tested with contralateral 1kHz NBN. The results show no evidence for the existence of a linear relationship between any of the variables.

Table 8-13: Pearson’s correlation coefficients for the relationship between parameters describing the filter shape measured during 1kHz contralateral NBN and TOAE magnitude and suppression.

<table>
<thead>
<tr>
<th>TOAE Test</th>
<th>P (lower)</th>
<th>P (upper)</th>
<th>k</th>
<th>r</th>
<th>Symmetry (SI)</th>
</tr>
</thead>
<tbody>
<tr>
<td>TOAE magnitude</td>
<td>-0.077</td>
<td>-0.286</td>
<td>-0.391</td>
<td>-0.401</td>
<td>0.052</td>
</tr>
<tr>
<td>Ipsi click-Contra white noise</td>
<td>0.073</td>
<td>0.008</td>
<td>-0.177</td>
<td>-0.098</td>
<td>0.115</td>
</tr>
<tr>
<td>Ipsi click-Contra 1kHz tone</td>
<td>0.112</td>
<td>0.026</td>
<td>-0.040</td>
<td>0.107</td>
<td>0.238</td>
</tr>
<tr>
<td>Ipsi 1kHz tone – contra white noise</td>
<td>-0.278</td>
<td>-0.400</td>
<td>-0.392</td>
<td>-0.495</td>
<td>-0.165</td>
</tr>
<tr>
<td>Ipsi 1kHz tone – contra 1kHz tone</td>
<td>0.137</td>
<td>0.065</td>
<td>-0.090</td>
<td>0.140</td>
<td>0.193</td>
</tr>
</tbody>
</table>

Table 8-14 shows the results for the filter measured during contralateral 2kHz NBN. Again, none of the comparisons showed any existence of a linear relationship.

Table 8-14: Correlation coefficients for the relationship between parameters describing the filter shape measured during 2kHz contralateral NBN and TOAE magnitude and suppression. (Pearson’s correlation coefficients, except for r where Spearman’s rho)

<table>
<thead>
<tr>
<th>TOAE Test</th>
<th>P (lower)</th>
<th>P (upper)</th>
<th>k</th>
<th>r</th>
<th>Symmetry (SI)</th>
</tr>
</thead>
<tbody>
<tr>
<td>TOAE magnitude</td>
<td>0.306</td>
<td>0.217</td>
<td>-0.327</td>
<td>0.077</td>
<td>0.339</td>
</tr>
<tr>
<td>Ipsi click-Contra white noise</td>
<td>0.145</td>
<td>0.325</td>
<td>-0.140</td>
<td>-0.148</td>
<td>-0.014</td>
</tr>
<tr>
<td>Ipsi click-Contra 1kHz tone</td>
<td>-0.373</td>
<td>-0.309</td>
<td>-0.522</td>
<td>0.092</td>
<td>-0.404</td>
</tr>
<tr>
<td>Ipsi 1kHz tone – contra white noise</td>
<td>0.125</td>
<td>0.164</td>
<td>-0.153</td>
<td>0.176</td>
<td>0.082</td>
</tr>
<tr>
<td>Ipsi 1kHz tone – contra 1kHz tone</td>
<td>-0.410</td>
<td>-0.222</td>
<td>-0.537</td>
<td>0.155</td>
<td>-0.510</td>
</tr>
</tbody>
</table>
The results for the auditory filter measured during 500Hz contralateral NBN are shown in Table 8-15. The only pair of variables to show a statistically significant linear correlation are p(upper) and TOAE magnitude. The correlation is positive with a p value of 0.006. This result therefore indicates that the correlation observed earlier (section 8.3.1) between the TOAE magnitude and BW\textsubscript{500nbn} was mostly due to the upper skirt of the filter (p(upper)).

Table 8-15: Pearson’s correlation coefficients for the relationship between parameters describing the filter shape measured during 500Hz contralateral NBN and TOAE magnitude and suppression.

<table>
<thead>
<tr>
<th>TOAE Test</th>
<th>P (lower)</th>
<th>P (upper)</th>
<th>k</th>
<th>r</th>
<th>Symmetry (SI)</th>
</tr>
</thead>
<tbody>
<tr>
<td>TOAE magnitude</td>
<td>0.596</td>
<td>0.765**</td>
<td>0.378</td>
<td>0.544</td>
<td>0.420</td>
</tr>
<tr>
<td>Ipsi click-Contra white noise</td>
<td>0.197</td>
<td>0.381</td>
<td>0.093</td>
<td>0.154</td>
<td>0.084</td>
</tr>
<tr>
<td>Ipsi click-Contra 1kHz tone</td>
<td>0.308</td>
<td>0.074</td>
<td>0.175</td>
<td>0.212</td>
<td>0.377</td>
</tr>
<tr>
<td>Ipsi 1kHz tone – contra white noise</td>
<td>0.110</td>
<td>0.145</td>
<td>0.028</td>
<td>0.144</td>
<td>0.088</td>
</tr>
<tr>
<td>Ipsi 1kHz tone – contra 1kHz tone</td>
<td>0.174</td>
<td>0.115</td>
<td>0.130</td>
<td>0.137</td>
<td>0.179</td>
</tr>
</tbody>
</table>

8.3.7.2 TOAE magnitude and suppression in specific frequency bands

The TOAE data was analysed further by examining two frequency bands around 1kHz and 2kHz, as described in section 5.3.3.

The results for the relationship of this data to the parameters describing the auditory filter shape measured during contralateral 1kHz NBN are shown in Table 8-16. No significant correlation coefficients were evident.

** statistically significant, p<0.01

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Table 8-16: Pearson’s correlation coefficients for the relationship between parameters describing the filter shape measured during 1kHz contralateral NBN and TOAE magnitude and suppression in two frequency bands.

<table>
<thead>
<tr>
<th>TOAE Test</th>
<th>Frequency band (kHz)</th>
<th>P (lower)</th>
<th>P (upper)</th>
<th>k</th>
<th>r</th>
<th>Symmetry (SI)</th>
</tr>
</thead>
<tbody>
<tr>
<td>TOAE magnitude</td>
<td>1</td>
<td>-0.119</td>
<td>-0.227</td>
<td>-0.389</td>
<td>-0.270</td>
<td>-0.050</td>
</tr>
<tr>
<td></td>
<td>2</td>
<td>-0.224</td>
<td>-0.436</td>
<td>-0.483</td>
<td>-0.498</td>
<td>-0.082</td>
</tr>
<tr>
<td>Ipsi click-Contra white noise</td>
<td>1</td>
<td>0.072</td>
<td>0.000</td>
<td>-0.025</td>
<td>-0.312</td>
<td>0.106</td>
</tr>
<tr>
<td></td>
<td>2</td>
<td>0.031</td>
<td>-0.086</td>
<td>-0.302</td>
<td>0.000</td>
<td>0.119</td>
</tr>
<tr>
<td>Ipsi click-Contra 1kHz tone</td>
<td>1</td>
<td>0.004</td>
<td>-0.133</td>
<td>-0.076</td>
<td>-0.066</td>
<td>0.135</td>
</tr>
<tr>
<td></td>
<td>2</td>
<td>0.300</td>
<td>0.127</td>
<td>-0.014</td>
<td>0.272</td>
<td>0.418</td>
</tr>
<tr>
<td>Ipsi 1kHz tone – Contra white noise</td>
<td>1</td>
<td>-0.185</td>
<td>-0.319</td>
<td>-0.328</td>
<td>-0.484</td>
<td>-0.076</td>
</tr>
<tr>
<td></td>
<td>2</td>
<td>-0.294</td>
<td>-0.384</td>
<td>-0.455</td>
<td>-0.242</td>
<td>-0.179</td>
</tr>
<tr>
<td>Ipsi 1kHz tone – Contra 1kHz tone</td>
<td>1</td>
<td>0.204</td>
<td>0.098</td>
<td>0.013</td>
<td>0.024</td>
<td>0.269</td>
</tr>
<tr>
<td></td>
<td>2</td>
<td>0.144</td>
<td>0.048</td>
<td>-0.115</td>
<td>0.281</td>
<td>0.233</td>
</tr>
</tbody>
</table>

Table 8-17 shows the results for the auditory filter measured during 2kHz contralateral NBN. Again, none of the correlation coefficients were statistically significant.

Table 8-17: Correlation coefficients for the relationship between parameters describing the filter shape measured during 2kHz contralateral NBN and TOAE magnitude and suppression in two frequency bands. (Pearson’s correlation coefficients except r which uses Spearman’s rho)

<table>
<thead>
<tr>
<th>TOAE Test</th>
<th>Frequency band (kHz)</th>
<th>P (lower)</th>
<th>P (upper)</th>
<th>k</th>
<th>r</th>
<th>Symmetry (SI)</th>
</tr>
</thead>
<tbody>
<tr>
<td>TOAE magnitude</td>
<td>1</td>
<td>0.460</td>
<td>0.461</td>
<td>-0.053</td>
<td>0.025</td>
<td>0.422</td>
</tr>
<tr>
<td></td>
<td>2</td>
<td>0.161</td>
<td>0.004</td>
<td>-0.476</td>
<td>0.232</td>
<td>0.269</td>
</tr>
<tr>
<td>Ipsi click-Contra white noise</td>
<td>1</td>
<td>0.353</td>
<td>0.497</td>
<td>0.283</td>
<td>-0.014</td>
<td>0.197</td>
</tr>
<tr>
<td></td>
<td>2</td>
<td>0.121</td>
<td>0.228</td>
<td>-0.283</td>
<td>-0.082</td>
<td>0.021</td>
</tr>
<tr>
<td>Ipsi click-Contra 1kHz tone</td>
<td>1</td>
<td>-0.068</td>
<td>-0.086</td>
<td>-0.202</td>
<td>0.322</td>
<td>-0.065</td>
</tr>
<tr>
<td></td>
<td>2</td>
<td>-0.428</td>
<td>-0.271</td>
<td>-0.561</td>
<td>0.012</td>
<td>-0.492</td>
</tr>
<tr>
<td>Ipsi 1kHz tone – Contra white noise</td>
<td>1</td>
<td>0.097</td>
<td>0.139</td>
<td>-0.187</td>
<td>0.130</td>
<td>0.051</td>
</tr>
<tr>
<td></td>
<td>2</td>
<td>0.081</td>
<td>0.167</td>
<td>-0.311</td>
<td>0.221</td>
<td>0.009</td>
</tr>
<tr>
<td>Ipsi 1kHz tone – Contra 1kHz tone</td>
<td>1</td>
<td>-0.489</td>
<td>-0.370</td>
<td>-0.565</td>
<td>0.194</td>
<td>-0.531</td>
</tr>
<tr>
<td></td>
<td>2</td>
<td>-0.287</td>
<td>-0.094</td>
<td>-0.541</td>
<td>-0.032</td>
<td>-0.414</td>
</tr>
</tbody>
</table>

Table 8-18 shows the results for the auditory filter measured during contralateral 500Hz NBN. P values showed most of the correlation coefficients not to be statistically significant. The only exceptions were the relationship between the TOAE magnitude around 2kHz and p(lower), p(upper) and r. The p values for these were 0.021, 0.003 and
0.042 respectively. Comparing these results to those for 500Hz NBN in the previous section, shows that the correlation observed there is due more to the TOAE energy in the 2kHz region than at 1kHz.

Table 8-18: Pearson’s correlation coefficients for the relationship between parameters describing the filter shape measured during 500Hz contralateral NBN and TOAE magnitude and suppression in two frequency bands.

<table>
<thead>
<tr>
<th>TOAE Test</th>
<th>Frequency band (kHz)</th>
<th>P (lower)</th>
<th>P (upper)</th>
<th>k</th>
<th>r</th>
<th>Symmetry (SI)</th>
</tr>
</thead>
<tbody>
<tr>
<td>TOAE magnitude</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>1</td>
<td>0.390</td>
<td>0.596</td>
<td>0.141</td>
<td>0.303</td>
<td>0.237</td>
<td></td>
</tr>
<tr>
<td>2</td>
<td>0.683*</td>
<td>0.801**</td>
<td>0.529</td>
<td>0.620*</td>
<td>0.519</td>
<td></td>
</tr>
<tr>
<td>Ipsi click—Contra white noise</td>
<td></td>
<td>-0.215</td>
<td>0.057</td>
<td>-0.210</td>
<td>0.026</td>
<td>-0.302</td>
</tr>
<tr>
<td>2</td>
<td>0.409</td>
<td>0.434</td>
<td>0.139</td>
<td>0.295</td>
<td>0.345</td>
<td></td>
</tr>
<tr>
<td>Ipsi click—Contra 1kHz tone</td>
<td></td>
<td>0.305</td>
<td>0.082</td>
<td>0.036</td>
<td>0.298</td>
<td>0.371</td>
</tr>
<tr>
<td>2</td>
<td>0.186</td>
<td>0.123</td>
<td>0.023</td>
<td>0.379</td>
<td>0.197</td>
<td></td>
</tr>
<tr>
<td>Ipsi 1kHz tone—Contra white noise</td>
<td></td>
<td>0.167</td>
<td>0.182</td>
<td>0.075</td>
<td>0.177</td>
<td>0.140</td>
</tr>
<tr>
<td>2</td>
<td>0.211</td>
<td>0.262</td>
<td>0.086</td>
<td>0.134</td>
<td>0.173</td>
<td></td>
</tr>
<tr>
<td>Ipsi 1kHz tone—Contra 1kHz tone</td>
<td></td>
<td>0.273</td>
<td>0.216</td>
<td>0.277</td>
<td>0.303</td>
<td>0.256</td>
</tr>
<tr>
<td>2</td>
<td>0.129</td>
<td>0.095</td>
<td>0.034</td>
<td>0.102</td>
<td>0.136</td>
<td></td>
</tr>
</tbody>
</table>

8.3.7.3  * Temporally windowed TOAE suppression*

The TOAE data was windowed to include only that part of the response that was recorded between 10 and 20ms after the onset of the stimulus.

The results of the comparison between the windowed TOAE data and the auditory filter measured during contralateral 1kHz NBN are shown in Table 8-19. None of the Pearson’s correlation coefficients calculated were statistically significant except for the relationship between the parameter r and the windowed suppression of 1kHz tone bursts by 1kHz tone in the 2kHz frequency band. The p value for this correlation was 0.017.

* statistically significant, p<0.05
** statistically significant, p<0.01
Table 8-19: Pearson’s correlation coefficients for the relationship between parameters describing the filter shape measured during 1kHz contralateral NBN and TOAE suppression in two frequency bands after windowing from 10-20ms.

<table>
<thead>
<tr>
<th>TOAE Test</th>
<th>Frequency band (kHz)</th>
<th>P (lower)</th>
<th>P (upper)</th>
<th>k</th>
<th>r</th>
<th>Symmetry (SI)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Ipsi click-Contra white noise</td>
<td>1</td>
<td>-0.081</td>
<td>-0.014</td>
<td>0.202</td>
<td>-0.520</td>
<td>-0.125</td>
</tr>
<tr>
<td></td>
<td>2</td>
<td>-0.080</td>
<td>0.328</td>
<td>0.025</td>
<td>0.246</td>
<td>-0.333</td>
</tr>
<tr>
<td>Ipsi click-Contra 1kHz tone</td>
<td>1</td>
<td>0.349</td>
<td>0.269</td>
<td>0.422</td>
<td>0.095</td>
<td>0.390</td>
</tr>
<tr>
<td></td>
<td>2</td>
<td>0.365</td>
<td>0.559</td>
<td>0.488</td>
<td>0.526</td>
<td>0.244</td>
</tr>
<tr>
<td>Ipsi 1kHz tone-Contra white noise</td>
<td>1</td>
<td>-0.152</td>
<td>-0.222</td>
<td>-0.270</td>
<td>-0.484</td>
<td>-0.099</td>
</tr>
<tr>
<td></td>
<td>2</td>
<td>-0.205</td>
<td>-0.100</td>
<td>-0.397</td>
<td>0.123</td>
<td>-0.246</td>
</tr>
<tr>
<td>Ipsi 1kHz tone-Contra 1kHz tone</td>
<td>1</td>
<td>-0.233</td>
<td>-0.236</td>
<td>-0.345</td>
<td>-0.036</td>
<td>-0.186</td>
</tr>
<tr>
<td></td>
<td>2</td>
<td>0.441</td>
<td>0.538</td>
<td>0.331</td>
<td>0.729*</td>
<td>0.361</td>
</tr>
</tbody>
</table>

Next, the parameters describing the shape of the auditory filter measured during contralateral 2kHz NBN were compared to the temporally windowed TOAE suppression results. These results are shown in Table 8-20. The symmetry index was negatively correlated with two of the OAE results. For the suppression of clicks by 1kHz tones in the 2kHz region, the correlation was -0.870 with a p value of 0.024. For the suppression of 1kHz tones with 1kHz tones in the 2kHz region, the correlation coefficient was found to be -0.744, with a p value of 0.014.

* statistically significant at p<0.05
Table 8-20: Correlation coefficients for the relationship between parameters describing the filter shape measured during 2kHz contralateral NBN and TOAE suppression in two frequency bands after windowing from 10-20ms (Pearson’s correlation coefficients except r which uses Spearman’s rho)

<table>
<thead>
<tr>
<th>TOAE Test</th>
<th>Frequency band (kHz)</th>
<th>P (lower)</th>
<th>P (upper)</th>
<th>k</th>
<th>r</th>
<th>Symmetry (SI)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Ipsi click-</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Contra white noise</td>
<td>1</td>
<td>0.250</td>
<td>0.313</td>
<td>0.491</td>
<td>-0.407</td>
<td>0.151</td>
</tr>
<tr>
<td></td>
<td>2</td>
<td>0.151</td>
<td>0.675</td>
<td>0.539</td>
<td>-0.771</td>
<td>-0.103</td>
</tr>
<tr>
<td>Ipsi click-</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Contra 1kHz tone</td>
<td>1</td>
<td>0.185</td>
<td>0.394</td>
<td>0.449</td>
<td>-0.238</td>
<td>-0.024</td>
</tr>
<tr>
<td></td>
<td>2</td>
<td>-0.792</td>
<td>-0.453</td>
<td>-0.481</td>
<td>0.087</td>
<td>-0.870*</td>
</tr>
<tr>
<td>Ipsi 1kHz tone –</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Contra white noise</td>
<td>1</td>
<td>0.299</td>
<td>0.275</td>
<td>0.001</td>
<td>-0.021</td>
<td>0.276</td>
</tr>
<tr>
<td></td>
<td>2</td>
<td>-0.159</td>
<td>0.040</td>
<td>-0.574</td>
<td>0.050</td>
<td>-0.261</td>
</tr>
<tr>
<td>Ipsi 1kHz tone –</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Contra 1kHz tone</td>
<td>1</td>
<td>0.336</td>
<td>0.342</td>
<td>0.012</td>
<td>0.197</td>
<td>0.292</td>
</tr>
<tr>
<td></td>
<td>2</td>
<td>-0.581</td>
<td>-0.228</td>
<td>-0.319</td>
<td>0.152</td>
<td>-0.744*</td>
</tr>
</tbody>
</table>

Finally, the parameters describing the shape of the auditory filter measured during contralateral 500Hz NBN were compared to the windowed TOAE suppression results. These results are shown in Table 8-21.

The windowed suppression of clicks by 1kHz tone was significantly correlated with p(lower) in the 2kHz region and p(upper) in the 1kHz region. The correlations were both negative in direction and the p values were 0.047 and 0.014 respectively.

Table 8-21: Pearson’s correlation coefficients for the relationship between parameters describing the filter shape measured during 500Hz contralateral NBN and TOAE suppression in two frequency bands after windowing from 10-20ms.

<table>
<thead>
<tr>
<th>TOAE Test</th>
<th>Frequency band (kHz)</th>
<th>P (lower)</th>
<th>P (upper)</th>
<th>k</th>
<th>r</th>
<th>Symmetry (SI)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Ipsi click-</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Contra white noise</td>
<td>1</td>
<td>-0.238</td>
<td>-0.076</td>
<td>0.012</td>
<td>-0.255</td>
<td>-0.297</td>
</tr>
<tr>
<td></td>
<td>2</td>
<td>-0.454</td>
<td>0.489</td>
<td>0.032</td>
<td>-0.600</td>
<td>-0.638</td>
</tr>
<tr>
<td>Ipsi click-</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Contra 1kHz tone</td>
<td>1</td>
<td>-0.688</td>
<td>-0.814*</td>
<td>-0.667</td>
<td>-0.706</td>
<td>-0.434</td>
</tr>
<tr>
<td></td>
<td>2</td>
<td>-0.817*</td>
<td>0.174</td>
<td>0.137</td>
<td>-0.076</td>
<td>-0.797</td>
</tr>
<tr>
<td>Ipsi 1kHz tone –</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Contra white noise</td>
<td>1</td>
<td>0.333</td>
<td>0.535</td>
<td>0.214</td>
<td>0.412</td>
<td>0.177</td>
</tr>
<tr>
<td></td>
<td>2</td>
<td>0.424</td>
<td>0.638</td>
<td>0.453</td>
<td>0.150</td>
<td>0.246</td>
</tr>
<tr>
<td>Ipsi 1kHz tone –</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Contra 1kHz tone</td>
<td>1</td>
<td>0.094</td>
<td>0.278</td>
<td>-0.164</td>
<td>0.280</td>
<td>-0.005</td>
</tr>
<tr>
<td></td>
<td>2</td>
<td>-0.457</td>
<td>-0.144</td>
<td>-0.262</td>
<td>-0.035</td>
<td>-0.531</td>
</tr>
</tbody>
</table>

* statistically significant, p<0.05
8.3.7.4 SOAEs

As mentioned earlier, of the 12 subjects examined 5 had SOAEs and 7 did not. The five parameters that describe the shape of the auditory filter were compared between these two groups to explore whether there was any difference between those that had SOAEs and those that did not.

The results are shown in Table 8-22 for the filter shape measured during contralateral 1kHz NBN. None of the parameters are significantly different between the 2 groups.

Table 8-22: Comparison of the values of the parameters describing the shape of the auditory filter during contralateral 1kHz NBN in subjects with and without SOAEs (all independent samples t-test results)

<table>
<thead>
<tr>
<th>Filter shape parameter</th>
<th>With SOAEs</th>
<th>Without SOAEs</th>
<th>2-tailed significance</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Mean</td>
<td>Standard deviation</td>
<td>Mean</td>
</tr>
<tr>
<td>P(lower)</td>
<td>34.01</td>
<td>14.23</td>
<td>37.08</td>
</tr>
<tr>
<td>P(upper)</td>
<td>27.29</td>
<td>3.98</td>
<td>28.72</td>
</tr>
<tr>
<td>k</td>
<td>8.61</td>
<td>2.91</td>
<td>10.62</td>
</tr>
<tr>
<td>r</td>
<td>-44.68</td>
<td>17.62</td>
<td>-41.27</td>
</tr>
<tr>
<td>Symmetry Index</td>
<td>1.21</td>
<td>0.36</td>
<td>1.26</td>
</tr>
</tbody>
</table>

The results for the parameters describing the shape of the auditory filter measured during 2kHz contralateral NBN are shown in Table 8-23. Again, none of the parameters showed a significant difference between the 2 groups.
Table 8-23: Comparison of the values of the parameters describing the shape of the auditory filter during contralateral 2kHz NBN in subjects with and without SOAEs (all t-test results except those for r which used Mann Whitney U Test)

<table>
<thead>
<tr>
<th>Filter shape parameter</th>
<th>With SOAEs</th>
<th>Without SOAEs</th>
<th>2-tailed significance</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Mean</td>
<td>Standard deviation</td>
<td>Mean</td>
</tr>
<tr>
<td>P(lower)</td>
<td>39.58</td>
<td>7.88</td>
<td>41.17</td>
</tr>
<tr>
<td>P(upper)</td>
<td>28.74</td>
<td>2.58</td>
<td>28.87</td>
</tr>
<tr>
<td>k</td>
<td>8.81</td>
<td>1.97</td>
<td>10.82</td>
</tr>
<tr>
<td>r</td>
<td>-35.24</td>
<td>4.01</td>
<td>-43.52</td>
</tr>
<tr>
<td>Symmetry Index</td>
<td>1.37</td>
<td>0.18</td>
<td>1.41</td>
</tr>
</tbody>
</table>

The 11 subjects that were tested with contralateral 500Hz NBN were also split into two groups depending on whether they had SOAEs or not. Five had SOAEs and six did not. Table 8-24 shows the results for the parameters describing the shape of the auditory filter measured during contralateral 500Hz NBN. There was no significant difference between the two groups for any of the parameters except p(upper). Here the group with SOAEs had a significantly larger value of p(upper).

Table 8-24: Comparison of the values of the parameters describing the shape of the auditory filter during contralateral 500Hz NBN in subjects with and without SOAEs (all independent samples t-test results)

<table>
<thead>
<tr>
<th>Filter shape parameter</th>
<th>With SOAEs</th>
<th>Without SOAEs</th>
<th>2-tailed significance</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Mean</td>
<td>Standard deviation</td>
<td>Mean</td>
</tr>
<tr>
<td>P(lower)</td>
<td>41.83</td>
<td>4.31</td>
<td>31.31</td>
</tr>
<tr>
<td>P(upper)</td>
<td>31.25</td>
<td>2.42</td>
<td>25.33</td>
</tr>
<tr>
<td>k</td>
<td>10.80</td>
<td>1.44</td>
<td>8.19</td>
</tr>
<tr>
<td>r</td>
<td>-37.41</td>
<td>3.23</td>
<td>-49.58</td>
</tr>
<tr>
<td>Symmetry Index</td>
<td>1.34</td>
<td>0.09</td>
<td>1.20</td>
</tr>
</tbody>
</table>

** statistically significant, p<0.01
8.3.7.5 DPOAEs

The parameters describing the shape of the auditory filter during contralateral NBN were then compared to the DPOAE parameters. Nine subjects took part in this section of the experiment.

Table 8-25 shows the Pearson correlation coefficients for the DP parameters compared with the five filter shape parameters measured during contralateral 1kHz NBN. There were no statistically significant correlations.

Table 8-25: Pearson correlation coefficients of DPOAE magnitude measurements with parameters describing auditory filter shape during contralateral 1kHz NBN

<table>
<thead>
<tr>
<th>DPOAE Measurement</th>
<th>P (lower)</th>
<th>P (upper)</th>
<th>k</th>
<th>r</th>
<th>Symmetry (SI)</th>
</tr>
</thead>
<tbody>
<tr>
<td>DP at 1kHz</td>
<td>0.144</td>
<td>0.366</td>
<td>0.327</td>
<td>0.153</td>
<td>-0.005</td>
</tr>
<tr>
<td>DP at 2kHz</td>
<td>0.133</td>
<td>0.302</td>
<td>0.237</td>
<td>0.027</td>
<td>-0.002</td>
</tr>
<tr>
<td>Mean (745-1501Hz)</td>
<td>0.540</td>
<td>0.623</td>
<td>0.606</td>
<td>0.425</td>
<td>0.448</td>
</tr>
<tr>
<td>Low Mean (745-977Hz)</td>
<td>0.538</td>
<td>0.564</td>
<td>0.583</td>
<td>0.486</td>
<td>0.496</td>
</tr>
<tr>
<td>High Mean (1013-1501Hz)</td>
<td>0.497</td>
<td>0.605</td>
<td>0.569</td>
<td>0.356</td>
<td>0.384</td>
</tr>
<tr>
<td>High-Low</td>
<td>0.073</td>
<td>0.203</td>
<td>0.124</td>
<td>-0.086</td>
<td>-0.047</td>
</tr>
<tr>
<td>Standard deviation</td>
<td>-0.323</td>
<td>-0.295</td>
<td>-0.344</td>
<td>-0.470</td>
<td>-0.344</td>
</tr>
</tbody>
</table>

Table 8-26 shows the results for the correlation calculations between the DPOAE measures and the parameters describing the auditory filter shape during 2kHz NBN. P(lower) and p(upper) were both found to be positively correlated with the standard deviation measure of the DPOAE. The p values were 0.031 and 0.017 respectively.
Table 8-26: Pearson correlation coefficients of DPOAE magnitude measurements with parameters describing auditory filter shape during contralateral 2kHz NBN

<table>
<thead>
<tr>
<th>DPOAE Measurement</th>
<th>P (lower)</th>
<th>P (upper)</th>
<th>k</th>
<th>r</th>
<th>Symmetry (SI)</th>
</tr>
</thead>
<tbody>
<tr>
<td>DP at 1kHz</td>
<td>-0.408</td>
<td>-0.141</td>
<td>-0.119</td>
<td>-0.007</td>
<td>-0.548</td>
</tr>
<tr>
<td>DP at 2kHz</td>
<td>-0.082</td>
<td>0.123</td>
<td>-0.027</td>
<td>0.263</td>
<td>-0.198</td>
</tr>
<tr>
<td>Mean (745-1501Hz)</td>
<td>-0.332</td>
<td>-0.091</td>
<td>-0.224</td>
<td>0.051</td>
<td>-0.466</td>
</tr>
<tr>
<td>Low Mean (745-977Hz)</td>
<td>-0.416</td>
<td>-0.269</td>
<td>-0.338</td>
<td>-0.041</td>
<td>-0.482</td>
</tr>
<tr>
<td>High Mean (1013-1501Hz)</td>
<td>-0.257</td>
<td>0.019</td>
<td>-0.141</td>
<td>0.099</td>
<td>-0.418</td>
</tr>
<tr>
<td>High-Low</td>
<td>0.152</td>
<td>0.391</td>
<td>0.220</td>
<td>0.222</td>
<td>-0.014</td>
</tr>
<tr>
<td>Standard deviation</td>
<td>0.712*</td>
<td>0.763*</td>
<td>0.630</td>
<td>0.486</td>
<td>0.607</td>
</tr>
</tbody>
</table>

Table 8-27 shows the results for the filter shape measured during 500Hz contralateral NBN. There were no statistically significant correlations.

Table 8-27: Pearson correlation coefficients of DPOAE magnitude measurements with parameters describing auditory filter shape during contralateral 500Hz NBN

<table>
<thead>
<tr>
<th>DPOAE Measurement</th>
<th>P (lower)</th>
<th>P (upper)</th>
<th>k</th>
<th>r</th>
<th>Symmetry (SI)</th>
</tr>
</thead>
<tbody>
<tr>
<td>DP at 1kHz</td>
<td>-0.231</td>
<td>0.272</td>
<td>0.190</td>
<td>0.015</td>
<td>-0.470</td>
</tr>
<tr>
<td>DP at 2kHz</td>
<td>-0.104</td>
<td>0.574</td>
<td>0.222</td>
<td>0.112</td>
<td>-0.468</td>
</tr>
<tr>
<td>Mean (745-1501Hz)</td>
<td>-0.166</td>
<td>0.340</td>
<td>0.174</td>
<td>0.000</td>
<td>-0.429</td>
</tr>
<tr>
<td>Low Mean (745-977Hz)</td>
<td>-0.143</td>
<td>0.256</td>
<td>0.145</td>
<td>0.260</td>
<td>-0.351</td>
</tr>
<tr>
<td>High Mean (1013-1501Hz)</td>
<td>-0.166</td>
<td>0.361</td>
<td>0.176</td>
<td>-0.149</td>
<td>-0.439</td>
</tr>
<tr>
<td>High-Low</td>
<td>-0.064</td>
<td>0.241</td>
<td>0.090</td>
<td>-0.576</td>
<td>-0.221</td>
</tr>
<tr>
<td>Standard deviation</td>
<td>-0.189</td>
<td>-0.034</td>
<td>-0.290</td>
<td>-0.467</td>
<td>-0.213</td>
</tr>
</tbody>
</table>

* statistically significant, p<0.05
8.3.8 Influence of other factors

8.3.8.1 Pure tone threshold

The mean 1kHz threshold level in the test ear of the 12 subjects tested was 2.5dBHL with a standard deviation of 3.4dB. The pure threshold was not found to be related to the bandwidths of the auditory filters tested during any of the contralateral NBNs.

8.3.8.2 Gender

In this experiment six males and six females were tested. There was no significant difference between the sexes for the bandwidths tested with any of the contralateral NBNs.

8.3.8.3 Ear Laterality

Six right ears and six left ears were tested. No significant differences were found for the filters tested during contralateral 1kHz or 2kHz NBN. However, for the filter tested during contralateral 500Hz NBN, the mean bandwidth for the left ear was 138.88Hz (s.d.=26.53) and for the right ear was 94.04Hz (s.d.=7.62). This was a significant difference (p value 0.03) when tested with the independent samples t-test. This may be linked to the fact that the 1kHz pure tone thresholds of the test ear, were significantly worse in the subjects whose right ear was tested, than those whose left ear was tested (mean left 0dB, mean right 5dB, p value 0.07). However, the group sizes were small and the bandwidth was not found to be correlated with pure tone threshold.

8.3.8.4 Threshold in noise

The detection threshold for 1kHz tones in noise during the three different contralateral NBNs were compared to the results from the four TOAE suppression tests. None were significantly related for the thresholds measured during contralateral 500Hz NBN or 2kHz NBN. The threshold measured during contralateral 1kHz NBN was found to be positively related to the suppression of clicks by white noise (p=0.026) and to the suppression of 1kHz tones by 1kHz tones (p=0.048), with correlation coefficients 0.635 and 0.580 respectively. Closer examination of the frequency bands of the suppression revealed that the threshold was only significantly related to the suppression in the 2kHz
region. In fact, the correlation was significant in the 2kHz region for all four of the suppression tests, even in the 2 tests where the overall suppression was not related (see Table 8-28).

### Table 8-28: Pearson correlation coefficients for the relationship between the suppression of TOAEs in the 2kHz region and the tone in noise threshold during contralateral 1kHz NBN

<table>
<thead>
<tr>
<th>Suppression Test</th>
<th>Correlation coefficient</th>
<th>P value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Ipsilateral clicks-contralateral white noise</td>
<td>0.783</td>
<td>0.004**</td>
</tr>
<tr>
<td>Ipsilateral clicks-contralateral 1kHz tone</td>
<td>0.735</td>
<td>0.016*</td>
</tr>
<tr>
<td>Ipsilateral 1kHz tone-contralateral white noise</td>
<td>0.628</td>
<td>0.029*</td>
</tr>
<tr>
<td>Ipsilateral 1kHz tone-contralateral 1kHz tone</td>
<td>0.630</td>
<td>0.028*</td>
</tr>
</tbody>
</table>

The correlation coefficients are all positive indicating that in these subjects, those with higher suppression levels in the 2kHz region were better at detecting tones, in what was effectively binaural 1kHz NBN.

### 8.3.8.5 Age

The mean age was 23.3 years with a standard deviation of 2.8 years. The age of the subjects was not found to be correlated with any of the bandwidths measured during contralateral NBN.

### 8.4 Discussion

The aim of this experiment was to acoustically stimulate the efferent system in specific frequency bands to see what effect this had on the auditory filter measured at 1kHz. The filters tested during contralateral NBN at 500Hz and 1kHz were found to be slightly, but not significantly wider than the filter tested with no contralateral stimulation. In

** statistically significant, p<0.01

* statistically significant, p<0.05
contrast, when tested during 2kHz NBN, the filter was found to be (non-significantly) narrower. Comparing with the results from the previous chapter where white noise was the contralateral stimulus, NBN had a much smaller effect on the filter shape. This is in agreement with past work on the effect of contralateral noise on the TOAE amplitude. (Norman and Thornton 1993) found that NBNs were much less effective suppressers than white noise. Presumably, this was because fewer medial efferent fibres were activated by the NBN.

Although changes to the filter shape were small in this study, we examined whether, on an individual basis, efferent activity level was related to the filter shape during efferent input at various frequencies. The frequency specific nature of action of the efferent system on OAEs has been described previously (Chery Croze et al. 1993; Mott et al. 1989; Veuillet et al. 1991). Thus, NBN contralateral stimulation should produce maximal efferent effect on the auditory filter in the corresponding frequency range.

However, the filter shapes during contralateral NBN were not in general related to the strength of efferent activity measured by OAE suppression or to the non-suppressed OAE level. The change in width of the filter between the ‘with’ and ‘without’ noise situations was also not related.

A few parameters did show some correspondence between the OAE and the filter shape tests. Since so many correlation coefficients were calculated, one has to treat the odd significant correlation with caution. Therefore, only results that had numerous or particularly strong correlations will be discussed further. The BW\textsubscript{500bn} was found to be negatively related to the TOAE amplitude. This was particularly strong for the relationship between the TOAE amplitude in the 2kHz region and the upper slope of the filter. It is not clear why this may be the case, since the efferents are presumably being activated in the 500Hz region for the filter shape test.

The DP level, at 1kHz, 2kHz and averaged across the frequency range, was correlated negatively with ΔBW\textsubscript{1bn}. This result implied that the greater the activity of the cochlear amplifier in this region, the more the auditory filter was resistant to change when presented with contralateral 1kHz NBN. This seems counter intuitive since the efferent system generally is most effective at suppressing BM motion and auditory nerve activity.
at the best frequency and therefore point of greatest activity (Murugasu and Russell 1996; Williams and Brown 1995).

For the auditory filter measured during contralateral 2kHz NBN, the symmetry index was found to be negatively correlated to the suppression of click or tone evoked TOAEs by contralateral tones. The relationship was only significant in the 2kHz region and when windowed temporally. The direction of the relationship indicates that in subjects with higher levels of efferent function in the 2kHz region (when the efferent system is stimulated by a 1kHz tone), the lower slope of the filter is shallower in comparison to the upper slope i.e. the filter is skewed towards the lower frequencies. This follows logically if one considers that the action of the efferent system stimulated by 2kHz NBN may be to damp the motion of the BM in the 2kHz region specifically. This frequency specific action could feasibly therefore sharpen one side of the filter. However, this result is not backed up by a significant relationship between p(upper) and the OAE suppression results.

Another interesting result to come from this work is the relationship between the suppression of TOAEs and the masked threshold of the 1kHz tone during contralateral NBN stimulation. Significant correlation coefficients were only found for the threshold of the detection of a 1kHz tone in noise during contralateral 1kHz NBN. Since the ipsilateral masker was also narrow band, the masking noise in both ears was approximately the same. There were significant relationships for the suppression of clicks evoked OAEs by white noise and for the suppression of 1kHz tone evoked OAEs by 1kHz tones. However, when examining the suppression more carefully, it was found that the relationship only held for the suppression in the 2kHz region, but that in this region the relationship was statistically significant for all four of the OAE suppression tests. The direction of the result implied that the subjects in whom the contralateral noise produced greater efferent activity in the 2kHz region were able to hear the tone better in the masking noise. Thus, in these subjects, the efferent system may act more effectively to dampen the BM motion, and therefore the neural response, to the ipsilateral masker at higher frequencies than 1kHz whilst leaving the signal frequency itself less damped. Previous work has suggested an anti-masking role for the efferent system (Kawase et al. 1993; Kawase and Liberman 1993; Liberman 1988). Micheyl and Collet (1996) carried out a similar comparison to the present study, and compared the suppression of TOAEs with the detection of tones in dichotic (broadband) noise. A
relationship was found, although only for the detection of the 2kHz tone, which they interpreted as suggesting that the OCB is involved in the detection of tones in noise. No such relationship was found for the detection of a tone at 1kHz. It is possible that the detailed analysis of the OAE suppression results carried out here helped us to reveal a relationship at 1kHz, which was hidden to them. In addition, we used signals that were more specific in frequency than Micheyl and Collet (1996). From the previous chapter, we also found no relationship with broadband signals. Stimulating the efferent system over a wide range of frequencies may add variability to an already sensitive measure, especially if, as is likely, the efferent system is not equally active over the length of the BM.

The same possible explanations for the general lack of a relationship between the OAE and the psychoacoustic tests apply as in the previous chapter. Obviously, the results may indicate that the efferent system is not involved in frequency selectivity. However, one must consider other factors. As before, the stimulus levels used mean that acoustic cross over or activation of the acoustic reflex were unlikely to be interfering with the results. It is possible that a change in shape of the filter did occur due to efferent activation, but that the change was so small that other test errors swamped the result. Another consideration is that the efferent system may cause a shift in the best frequency of the filter. If this was the case, the filter modelling procedure would not be able to accurately estimate the shape of the filter and therefore a relationship to the OAE results would be lost.

Finally, some of the past studies on humans have found a link between efferent activity and detection of tones in noise (Micheyl and Collet 1996) or between OAE magnitude and psychoacoustic tuning (Micheyl and Collet 1994), at 2kHz but not at 1kHz. In order to test whether there was some anomaly between the efferent activity at the two frequencies, the relationship between efferent function and psychoacoustic tuning was measured at 2kHz and is described in the next chapter.
8.5 Conclusion

This study did not show significant changes to the shape of the 1kHz auditory filter by the addition of contralateral NBN at 500Hz, 1kHz or 2kHz. There was some evidence however, for an involvement of the efferent system (particularly around 2kHz) in the detection of 1kHz tones in binaural 1kHz NBN. The results indicated that greater efferent activity was related to better detection-in-noise performance.

Assessing the results as a whole, there was little evidence for a link between efferent activity, as measured by OAE suppression, and psychoacoustic tuning when the efferent system was stimulated in these specific frequency bands.
Chapter 9

The Auditory Filter Shape at 2kHz and the Possible Link with OAE Magnitude and Suppression
9. The Auditory Filter Shape at 2kHz and the Possible Link with OAE Magnitude and Suppression

9.1 Introduction

Past studies do not indicate that there are substantial differences in OCB physiology or anatomy between 1kHz and 2kHz apart from the fact that in animals, efferent medial innervation generally increases at high frequencies. (Liberman 1988). Although there remains no similar anatomical proof in humans, physiological studies have provided some curious results. Micheyl and Collet (1996) found a relationship between TEOAE suppression and detection of tones in noise only at 2kHz and not at 1kHz, and also between psychoacoustic tuning curve measurements and OAEs mainly at 2kHz rather than at 1kHz or 4kHz (Micheyl and Collet 1994). Additionally, studies such as Scharf et al. (1997), which found no difference in frequency selectivity after vestibular neurectomy, made measurements only at 1kHz.

It seems unlikely that the results of Micheyl, noted above, were due to differences in the functioning of the measuring equipment between 1kHz and 2kHz. Given this past work and the inconclusive results from the studies at 1kHz presented in the previous chapters, it was decided to carry out some tests at 2kHz. This would help to ascertain whether the lack of association between the OAE and the psychoacoustic tests was consistent across frequency.

9.2 Methods

9.2.1 Subjects

Nine subjects were tested in this experiment, five of whom were female and four were male. The mean age was 22.4 years (s.d.=2.4 years). The ear to be tested was assigned randomly, resulting in the testing of 6 right ears and 3 left ears. All the subjects had normal pure tone audiometry thresholds, tympanometry and acoustic reflex thresholds as described in sections 3.3, 3.4 and 3.5.
9.2.2 Measurement of Auditory Filter Shape

The filter shape at 2kHz was estimated using the technique described in section 3.1.2.2.

9.2.3 Otoacoustic Emission Measurements

TOAEs, SOAEs, DPOAEs and contralateral suppression of TOAEs were performed on all subjects. The methods are the same as those described in sections 5.2.3, 5.2.4 and 5.2.5. In addition, TOAE suppression tests were performed using the same procedure with a 70dBSPL 2kHz tone evoking the emission and with a 50dBSL 2kHz tone suppressor.

9.3 Results

The mean 3dB bandwidth was found to be 202.71Hz with a standard deviation of 56.27Hz. The Kolmogorov-Smirnov test showed the bandwidth data to be approximately normal in distribution.

9.3.1 TOAE magnitude

The mean TOAE response level for the nine subjects tested in this experiment was 12.18dB (s.d.=3.38). The data was found to be distributed normally and the Pearson’s correlation coefficient was 0.517 when compared to the bandwidth of the filter at 2kHz. However, the p value was 0.154 and therefore the correlation was not statistically significant.

9.3.2 TOAE suppression

The mean and standard deviations for the four TOAE suppression tests are shown in Table 9-1. The 1 sample t-test results are shown which show that the suppression for all four tests was significantly greater than zero. Data from all the tests were found to be normally distributed. Also shown are the Pearson’s correlation coefficients for the comparison with the 3dB bandwidth of the auditory filter at 2kHz. None of the suppression tests were found to be correlated with the 3dB bandwidth of the 2kHz auditory filter.

1 Rosen and Baker (1994) found the 3dB bandwidth at 2kHz to be 261Hz. However, this was measured at a higher level and therefore one would expect it to be wider than that measured here.
Table 9-1: TOAE suppression results including Pearson correlation coefficients for relationship with 3dB bandwidth of 2kHz auditory filter.

<table>
<thead>
<tr>
<th>TOAE Suppression Stimuli</th>
<th>Mean Suppression (dB)</th>
<th>Standard Deviation</th>
<th>1-sample t-test (test value 0) p value</th>
<th>Correlation with 3dB bandwidth</th>
</tr>
</thead>
<tbody>
<tr>
<td>Ipsi click-Contra noise</td>
<td>1.94</td>
<td>0.67</td>
<td>0.000</td>
<td>0.563</td>
</tr>
<tr>
<td>Ipsi click-Contra 1kHz tone</td>
<td>0.59</td>
<td>0.34</td>
<td>0.001</td>
<td>0.141</td>
</tr>
<tr>
<td>Ipsi 1kHz tone-Contra noise</td>
<td>0.66</td>
<td>0.61</td>
<td>0.012</td>
<td>-0.075</td>
</tr>
<tr>
<td>Ipsi 1kHz tone-Contra 1kHz tone</td>
<td>0.21</td>
<td>0.21</td>
<td>0.016</td>
<td>-0.210</td>
</tr>
</tbody>
</table>

9.3.3 TOAE magnitude and suppression in specific frequency bands

The TOAE magnitude and suppression in the 1kHz and 2kHz regions (see section 5.3.3) were compared with the 3dB bandwidth of the auditory filter measured at 2kHz. The results of the Pearson's correlation coefficients calculated are shown in Table 9-2. None of the correlations were statistically significant.

Table 9-2: Pearson's correlation coefficients for the relationship between the 3dB bandwidth of the 2kHz auditory filter and TOAE magnitude and suppression in 2 frequency bands

<table>
<thead>
<tr>
<th>Stimuli</th>
<th>Correlation Coefficient</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>1kHz Region</td>
</tr>
<tr>
<td>Standard TOAE</td>
<td>0.491</td>
</tr>
<tr>
<td>Ipsi click-Contra white noise</td>
<td>0.379</td>
</tr>
<tr>
<td>Ipsi click-Contra 1kHz tone</td>
<td>0.193</td>
</tr>
<tr>
<td>Ipsi 1kHz tone-Contra white noise</td>
<td>-0.117</td>
</tr>
<tr>
<td>Ipsi 1kHz tone-Contra 1kHz tone</td>
<td>-0.221</td>
</tr>
</tbody>
</table>
9.3.4 Temporally windowed TOAE suppression

As in previous chapters (see section 5.3.4) the suppression data was windowed to include only that part of the response recorded between 10 and 20ms after the onset of the stimuli. The results of the correlation of this data with the 2kHz filter bandwidth data are shown in Table 9-3. There were no statistically significant correlations.

Table 9-3: Pearson’s correlation coefficients for the relationship between the 3dB bandwidth of the 2kHz auditory filter and TOAE suppression in 2 frequency bands after temporally windowing (10-20ms)

<table>
<thead>
<tr>
<th>Stimuli</th>
<th>1kHz Region</th>
<th>2kHz Region</th>
</tr>
</thead>
<tbody>
<tr>
<td>Ipsi click- Contra white noise</td>
<td>0.080</td>
<td>0.419</td>
</tr>
<tr>
<td>Ipsi click- Contra 1kHz tone</td>
<td>-0.063</td>
<td>0.349</td>
</tr>
<tr>
<td>Ipsi 1kHz tone- Contra white noise</td>
<td>0.270</td>
<td>0.092</td>
</tr>
<tr>
<td>Ipsi 1kHz tone- Contra 1kHz tone</td>
<td>0.179</td>
<td>-0.022</td>
</tr>
</tbody>
</table>

9.3.5 SOAEs

Of the 9 subjects tested 5 were found to have SOAEs, and the mean 3dB bandwidth for these subjects was 220.94Hz (s.d.=72.75). Four subjects did not have SOAEs and the mean bandwidth for these subjects was 179.92Hz (s.d.=11.89). However, this difference was not statistically significant (p value=0.307).

9.3.6 DPOAEs

The distortion products of all 9 subjects were tested. These were the same 9 subjects that were tested for DPs in section 8.3.6 and the summary of the mean and standard deviations for the 7 parameters used to measure the DPs can be found in Table 8-11.

Pearson’s correlation coefficients were calculated for the relationship between these parameters and the 3dB bandwidth of the 2kHz auditory filter. These results are shown in Table 9-4. None of the correlation coefficients were statistically significant.
Table 9-4: Pearson’s correlation coefficients for relationship between DPOAE measures and 2kHz auditory filter bandwidth

<table>
<thead>
<tr>
<th>DPOAE Measure</th>
<th>Correlation Coefficient</th>
</tr>
</thead>
<tbody>
<tr>
<td>DP at 1kHz</td>
<td>0.222</td>
</tr>
<tr>
<td>DP at 2kHz</td>
<td>0.411</td>
</tr>
<tr>
<td>Mean (745-1501Hz)</td>
<td>0.575</td>
</tr>
<tr>
<td>Low Mean (745-977Hz)</td>
<td>0.487</td>
</tr>
<tr>
<td>High Mean (1013-1501Hz)</td>
<td>0.579</td>
</tr>
<tr>
<td>High-Low</td>
<td>0.285</td>
</tr>
<tr>
<td>Standard deviation</td>
<td>0.020</td>
</tr>
</tbody>
</table>

9.3.7 Other parameters describing the shape of the auditory filter

9.3.7.1 TOAE magnitude and suppression

The 5 parameters which describe the shape of the auditory filter at 2kHz were compared with the TOAE magnitude and the 4 TOAE suppression tests. The Pearson correlation coefficients are shown in Table 9-5. Only one of the correlation coefficients was found to be significant. This was the relationship between the TOAE magnitude and the parameter $k$. A negative correlation was found which had a $p$ value of 0.014.

Table 9-5: Pearson’s correlation coefficients for the relationship between parameters describing the 2kHz auditory filter and TOAE magnitude and suppression.

<table>
<thead>
<tr>
<th>TOAE Test</th>
<th>P (lower)</th>
<th>P (upper)</th>
<th>$k$</th>
<th>r</th>
<th>Symmetry (SI)</th>
</tr>
</thead>
<tbody>
<tr>
<td>TOAE magnitude</td>
<td>-0.366</td>
<td>-0.527</td>
<td>-0.777*</td>
<td>-0.559</td>
<td>-0.209</td>
</tr>
<tr>
<td>Ipsi click-</td>
<td>-0.543</td>
<td>-0.551</td>
<td>-0.332</td>
<td>-0.584</td>
<td>-0.468</td>
</tr>
<tr>
<td>Contra white noise</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Ipsi click-</td>
<td>-0.262</td>
<td>-0.134</td>
<td>-0.004</td>
<td>0.004</td>
<td>-0.319</td>
</tr>
<tr>
<td>Contra 1kHz tone</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Ipsi 1kHz tone – contra</td>
<td>0.076</td>
<td>0.085</td>
<td>-0.004</td>
<td>0.074</td>
<td>0.050</td>
</tr>
<tr>
<td>white noise</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Ipsi 1kHz tone – contra</td>
<td>-0.003</td>
<td>0.177</td>
<td>0.365</td>
<td>0.361</td>
<td>-0.108</td>
</tr>
<tr>
<td>1kHz tone</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

* statistically significant, $p<0.05$
9.3.7.2 TOAE magnitude and suppression in specific frequency bands

Table 9-6 shows the correlation coefficients for the comparison between the TOAE magnitude and suppression analysed in frequency bands around 1kHz and 2kHz and the five parameters describing the shape of the 2kHz auditory filter. The only correlation that was found to be statistically significant was that of the relationship between the suppression of click evoked OAEs by white noise in the 2kHz region with the parameter r.

**Table 9-6: Pearson’s correlation coefficients for the relationship between parameters describing the 2kHz auditory filter shape and TOAE magnitude and suppression in two frequency bands.**

<table>
<thead>
<tr>
<th>TOAE Test</th>
<th>Frequency band (kHz)</th>
<th>P (lower)</th>
<th>P (upper)</th>
<th>k</th>
<th>r</th>
<th>Symmetry (SI)</th>
</tr>
</thead>
<tbody>
<tr>
<td>TOAE magnitude</td>
<td>1</td>
<td>-0.357</td>
<td>-0.456</td>
<td>-0.610</td>
<td>-0.615</td>
<td>-0.263</td>
</tr>
<tr>
<td></td>
<td>2</td>
<td>-0.112</td>
<td>-0.282</td>
<td>-0.652</td>
<td>-0.256</td>
<td>0.027</td>
</tr>
<tr>
<td>Ipsi click-Contra white noise</td>
<td>1</td>
<td>-0.338</td>
<td>-0.349</td>
<td>-0.216</td>
<td>-0.407</td>
<td>-0.300</td>
</tr>
<tr>
<td></td>
<td>2</td>
<td>-0.598</td>
<td>-0.565</td>
<td>-0.515</td>
<td>-0.697*</td>
<td>-0.572</td>
</tr>
<tr>
<td>Ipsi click-Contra 1kHz tone</td>
<td>1</td>
<td>-0.217</td>
<td>-0.130</td>
<td>-0.142</td>
<td>-0.135</td>
<td>-0.283</td>
</tr>
<tr>
<td></td>
<td>2</td>
<td>-0.359</td>
<td>-0.114</td>
<td>0.034</td>
<td>0.126</td>
<td>-0.495</td>
</tr>
<tr>
<td>Ipsi 1kHz tone – Contra white noise</td>
<td>1</td>
<td>0.122</td>
<td>0.118</td>
<td>0.078</td>
<td>0.117</td>
<td>0.104</td>
</tr>
<tr>
<td></td>
<td>2</td>
<td>-0.356</td>
<td>-0.323</td>
<td>-0.385</td>
<td>-0.345</td>
<td>-0.351</td>
</tr>
<tr>
<td>Ipsi 1kHz tone – Contra 1kHz tone</td>
<td>1</td>
<td>0.073</td>
<td>0.158</td>
<td>0.333</td>
<td>0.379</td>
<td>0.038</td>
</tr>
<tr>
<td></td>
<td>2</td>
<td>-0.456</td>
<td>-0.231</td>
<td>-0.072</td>
<td>-0.052</td>
<td>-0.550</td>
</tr>
</tbody>
</table>

9.3.7.3 Temporally windowed TOAE suppression

The suppression data was windowed to include only that part of the response recorded from 10 to 20ms after the stimulus onset. The data was compared with the 5 parameters describing the shape of the 2kHz auditory filter and the Pearson correlation coefficients are shown in Table 9-7. None of the correlation coefficients were found to be statistically significant.
Table 9-7: Pearson’s correlation coefficients for the relationship between parameters describing the 2kHz auditory filter shape and TOAE suppression in two frequency bands after windowing from 10-20ms.

<table>
<thead>
<tr>
<th>TOAE Test</th>
<th>Frequency band (kHz)</th>
<th>P (lower)</th>
<th>P (upper)</th>
<th>k</th>
<th>r</th>
<th>Symmetry (SI)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Ipsi click-Contra white noise</td>
<td>1</td>
<td>0.061</td>
<td>-0.105</td>
<td>0.130</td>
<td>-0.141</td>
<td>0.195</td>
</tr>
<tr>
<td></td>
<td>2</td>
<td>-0.309</td>
<td>-0.447</td>
<td>-0.110</td>
<td>-0.563</td>
<td>-0.162</td>
</tr>
<tr>
<td>Ipsi click-Contra 1kHz tone</td>
<td>1</td>
<td>-0.067</td>
<td>0.134</td>
<td>0.384</td>
<td>0.124</td>
<td>-0.240</td>
</tr>
<tr>
<td></td>
<td>2</td>
<td>-0.508</td>
<td>-0.423</td>
<td>-0.076</td>
<td>-0.160</td>
<td>-0.489</td>
</tr>
<tr>
<td>Ipsi 1kHz tone – Contra white noise</td>
<td>1</td>
<td>-0.086</td>
<td>-0.302</td>
<td>-0.289</td>
<td>-0.362</td>
<td>0.088</td>
</tr>
<tr>
<td></td>
<td>2</td>
<td>-0.184</td>
<td>-0.187</td>
<td>0.171</td>
<td>-0.043</td>
<td>-0.108</td>
</tr>
<tr>
<td>Ipsi 1kHz tone – Contra 1kHz tone</td>
<td>1</td>
<td>-0.207</td>
<td>-0.158</td>
<td>-0.284</td>
<td>-0.179</td>
<td>-0.229</td>
</tr>
<tr>
<td></td>
<td>2</td>
<td>-0.282</td>
<td>-0.032</td>
<td>0.256</td>
<td>0.263</td>
<td>-0.401</td>
</tr>
</tbody>
</table>

9.3.7.4 SOAEs

The five parameters describing the shape of the auditory filter at 2kHz were compared in the two groups of subjects with and without SOAEs. The results are shown in Table 9-8. The significance values from the independent samples t-test show that none of the parameters were significantly different between the two groups.

Table 9-8: Comparison of the values of the parameters describing the shape of the 2kHz auditory filter in subjects with and without SOAEs (independent samples t-test)

<table>
<thead>
<tr>
<th>Filter shape parameter</th>
<th>With SOAEs</th>
<th>Without SOAEs</th>
<th>2-tailed significance</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Mean</td>
<td>Standard deviation</td>
<td>Mean</td>
</tr>
<tr>
<td>P(lower)</td>
<td>37.02</td>
<td>11.75</td>
<td>43.34</td>
</tr>
<tr>
<td>P(upper)</td>
<td>29.16</td>
<td>5.53</td>
<td>33.06</td>
</tr>
<tr>
<td>k</td>
<td>10.33</td>
<td>2.65</td>
<td>12.71</td>
</tr>
<tr>
<td>r</td>
<td>-44.38</td>
<td>15.17</td>
<td>-36.98</td>
</tr>
<tr>
<td>Symmetry Index</td>
<td>1.24</td>
<td>0.21</td>
<td>1.31</td>
</tr>
</tbody>
</table>

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9.3.7.5  **DPOAEs**

The seven DPOAE parameters were compared with the five parameters describing the shape of the auditory filter at 2kHz. Table 9-9 shows the Pearson correlation coefficients, none of which were statistically significant.

<table>
<thead>
<tr>
<th>DPOAE Measurement</th>
<th>P (lower)</th>
<th>P (upper)</th>
<th>k</th>
<th>r</th>
<th>Symmetry (SI)</th>
</tr>
</thead>
<tbody>
<tr>
<td>DP at 1kHz</td>
<td>-0.259</td>
<td>-0.328</td>
<td>0.057</td>
<td>-0.121</td>
<td>-0.122</td>
</tr>
<tr>
<td>DP at 2kHz</td>
<td>-0.390</td>
<td>-0.510</td>
<td>-0.266</td>
<td>-0.346</td>
<td>-0.205</td>
</tr>
<tr>
<td>Mean (745-1501Hz)</td>
<td>-0.663</td>
<td>-0.665</td>
<td>-0.301</td>
<td>-0.384</td>
<td>-0.523</td>
</tr>
<tr>
<td>Low Mean (745-977Hz)</td>
<td>-0.605</td>
<td>-0.578</td>
<td>-0.299</td>
<td>-0.245</td>
<td>-0.495</td>
</tr>
<tr>
<td>High Mean (1013-1501Hz)</td>
<td>-0.642</td>
<td>-0.660</td>
<td>-0.278</td>
<td>-0.432</td>
<td>-0.497</td>
</tr>
<tr>
<td>High-Low</td>
<td>-0.226</td>
<td>-0.291</td>
<td>-0.064</td>
<td>-0.372</td>
<td>-0.140</td>
</tr>
<tr>
<td>Standard deviation</td>
<td>0.093</td>
<td>0.056</td>
<td>-0.041</td>
<td>-0.224</td>
<td>0.066</td>
</tr>
</tbody>
</table>

9.3.8  **Relationship to TOAEs elicited and/or suppressed at 2kHz**

The three additional TOAE suppression tests were also analysed by comparing them to the filter shape results. Firstly, the summary of the mean and standard deviations of the data is shown in Table 9-10. Eight subjects of the nine tested for the filter shape at 2kHz were tested for the first two suppression tests and seven for the final test.

One test did not give results which were high enough to be significantly greater than zero. This was the suppression of 2kHz tone evoked OAEs by a 2kHz contralateral tone. However, the other two tests did have suppression significantly greater than zero. These results are also shown in Table 9-10.
Table 9-10: Summary of TOAE suppression results

<table>
<thead>
<tr>
<th>TOAE Stimuli</th>
<th>n</th>
<th>Mean Suppression (dB)</th>
<th>Standard Deviation</th>
<th>1 sample t-test p value (test value=0)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Ipsi 2kHz tone - Contra white noise</td>
<td>8</td>
<td>0.51</td>
<td>0.53</td>
<td>0.030*</td>
</tr>
<tr>
<td>Ipsi 2kHz tone – Contra 2kHz tone</td>
<td>8</td>
<td>0.05</td>
<td>0.13</td>
<td>0.316</td>
</tr>
<tr>
<td>Ipsi click – Contra 2kHz tone</td>
<td>7</td>
<td>0.40</td>
<td>0.24</td>
<td>0.005**</td>
</tr>
</tbody>
</table>

The correlation coefficients for the comparison of the TOAE results with the filter shape results can be seen in Table 9-11.

Table 9-11: Pearson correlation coefficients for the relationship between TOAE suppression tests and 2kHz auditory filter parameters

<table>
<thead>
<tr>
<th>TOAE Stimuli</th>
<th>3dB Bandwidth</th>
<th>P (lower)</th>
<th>P (upper)</th>
<th>k</th>
<th>r</th>
<th>Symmetry (SI)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Ipsi 2kHz tone - Contra white noise</td>
<td>0.895**</td>
<td>-0.881**</td>
<td>-0.916**</td>
<td>-0.911**</td>
<td>-0.766*</td>
<td>-0.718*</td>
</tr>
<tr>
<td>Ipsi 2kHz tone – Contra 2kHz tone</td>
<td>0.308</td>
<td>-0.537</td>
<td>-0.220</td>
<td>-0.231</td>
<td>-0.161</td>
<td>-0.709*</td>
</tr>
<tr>
<td>Ipsi click – Contra 2kHz tone</td>
<td>-0.742</td>
<td>0.808*</td>
<td>0.665</td>
<td>0.671</td>
<td>0.723</td>
<td>0.835*</td>
</tr>
</tbody>
</table>

It can be seen that the suppression of 2kHz tone evoked OAEs by contralateral white noise is strongly correlated with the 3dB bandwidth of the 2kHz auditory filter, p(lower), p(upper), k and slightly less strongly with r and the SI. The p values for these correlation coefficients are 0.003, 0.004, 0.001, 0.002, 0.027 and 0.045 respectively. The results indicate that higher suppression is associated with a wider, less finely tuned filter.

The SI is also significantly correlated with the suppression of 2kHz evoked OAEs by a 2kHz contralateral tone and to click evoked OAEs suppressed by a contralateral 2kHz tone.

** statistically significant, p<0.01
* statistically significant, p<0.05
tone. In the former the correlation is negative in direction (p value=0.049) and in the latter case the correlation is positive in direction (p value=0.02). Finally, the suppression of click evoked OAEs by a 2kHz tone is positively correlated with p(lower) with a p value of 0.028.

9.3.9 Influence of other factors

9.3.9.1 Pure tone threshold

The mean 2kHz pure tone threshold level in the test ear of the 9 subjects tested was 3.3dBHL with a standard deviation of 5.6dB. The pure threshold was not found to be related to the bandwidth of the auditory filter tested at 2kHz.

9.3.9.2 Gender

In this experiment 4 males and 5 females were tested. There was no significant difference between the sexes for the bandwidth of the auditory filter at 2kHz.

9.3.9.3 Ear Laterality

6 right ears and 3 left ears were tested. No significant differences were found for the filter tested at 2kHz.

9.3.9.4 Threshold in noise

The detection threshold for 2kHz tones in noise during was compared to the results from the TOAE suppression tests. None were significantly related.

9.3.9.5 Age

The mean age was 22.4 years with a standard deviation of 2.4 years. The age of the subjects was not found to be correlated with the bandwidth of the auditory filter at 2kHz.
9.4 Discussion

The 3dB bandwidth of the auditory filter measured at 2kHz was found to be wider than that measured at 1kHz. This is to be expected from the past literature (Glasberg and Moore 1990). However, the bandwidth was not found to be related to the OAE magnitude or suppression in any of the conditions measured in the previous 1kHz experiments.

Examining the filter shape in more detail found two relationships that were statistically significant. Firstly, the TOAE magnitude was found to be negatively correlated to k, the detection efficiency. Secondly, the suppression of click evoked OAEs by white noise in the 2kHz region was found to be negatively related to r, the dynamic range of the filter. Given that many of the other relationships were not found to be correlated, the significance of these single results is therefore reduced.

However, the comparison of the filter shape to the OAE suppression tests which involved 2kHz ipsilateral and/or contralateral stimuli are much more conclusive. There appears to be a consistent and strong relationship between the 2kHz auditory filter and the amount of suppression produced by a white noise stimulus on 2kHz tone evoked OAEs. The direction of the relationship is such that greater efferent activity as measured by the OAE suppression results is correlated with broader auditory filters. This greater bandwidth seems to be produced by having both shallower upper and lower sides of filters. However, the symmetry index is negatively correlated, implying that the filter is skewed towards the lower frequencies more in subjects with higher suppression levels. This was also the case for the suppression of 2kHz tone evoked OAEs by 2kHz tones. The greater filter widths in these subjects is also associated with lower values of r and k. This indicates that larger suppression is linked to a greater dynamic range of the filter and a better detection efficiency. The results imply that the efferent system may be acting, via the active mechanisms involving the outer hair cells, to damp the motion of the BM, and that the effect of this damping is to cause a deterioration in the tuning. These results are in keeping with past studies which have shown that OCB stimulation causes a widening of neural (Guinan and Gifford 1988b) and DP (Williams and Brown 1995) tuning curves.
The suppression of click evoked OAEs by 2kHz tones was also related to the lower side of the filter and to the symmetry index. Interestingly though, in contrast to the above results, these were related in the opposite direction. It is possible that the efferent population stimulated in this test (being of narrower frequency range than when white noise was used as the stimulus) is actually, in the case of the 2kHz filter, involved in improving the tuning. It may be that the variability in efferent effectiveness along the length of the cochlea combined with the select nature of BM damping required to give enhancement in tuning could produce these results. In other words, a greater level of efferent activity in this very select region could cause a damping in only the tail of the filter (most likely the lower side, since the upper slope is not significantly correlated) thus narrowing the curve. However, the 'general' efferent activity measured by stimulating with white noise, would affect the whole system, thus preventing the detail from select frequencies of interest to be recorded. The 'general' damping of BM motion would lead to a broadening of the tuning, since the peak of the tuning curve would also be affected.

Examination of other factors (pure tone threshold, age, gender, ear) showed no evidence for confounding influences on these results.

Comparison of these results to those for the auditory filter at 1kHz shows that there seems to be a much stronger relation between the level of activity of the efferent system and tuning at 2kHz. Past studies have also found this to be the case using different test protocols. Micheyl and Collet (1996) found that OAE suppression was related to the detection of tones in noise at 2kHz but not at 1kHz. The only other paper which examined the relationship between OAEs and a psychophysical measure of the auditory filter (Micheyl and Collet 1994), mainly found significant results for the 2kHz tuning curve rather than at 1kHz and 4kHz.

Other work has indicated that active mechanisms may have greater influence at higher frequencies. Fagelson and Champlin (1997) measured auditory filter shapes at neighbouring frequencies in the 1kHz and the 4kHz regions. Correlation between the filters was affected by level to a greater extent at 4kHz. They argued that BM displacement is controlled more by passive mechanisms at lower frequencies and thus active non-linear mechanisms may compensate by being more prevalent at high frequencies. Rosen and Stock (1992) tested the effect of level on auditory filters from
125-1000Hz and concluded that the greater change at higher frequencies was due to non-linear processes being more pronounced towards the cochlear apex. In the cat, Guinan and Gifford (1988) found that threshold shifts during efferent stimulation, peaked at higher frequencies. They also noted that tuning curves, in general, were widened by efferent stimulation, but that below 2kHz the results were more variable since some of the tuning curves became narrower. This implied that as frequency increases, in this region of the cochlea, the widening effect of the efferent system on the tuning curves increases. In addition, efferent induced discharge rate suppression was found to be greater in nerve fibres of higher characteristic frequency (Wiederhold 1970) as was the anti-masking effect observed by Kawase et al. (1993). Finally, anatomical studies, albeit in animals, of medial efferent neurons, have tended to show an increase in innervation towards higher frequencies (Liberman 1988).

It seems unlikely that the contrasting results gained at 1kHz and 2kHz could be solely due to differences in the operation of the measuring equipment between these two frequencies. In this frequency range the receiving and output characteristics of the equipment were approximately uniform. Additionally, a smaller group of subjects was tested in this experiment than in the previous chapters. However, the results were still statistically significant.

It is possible that the efferent system has greater innervation and/or greater action on the BM at higher frequencies. A greater degree of activity in the 2kHz region may have been enough to compensate for possible inaccuracies in the experiment that may have masked the effect in the 1kHz region.

An alternative explanation is that the outer hair cells may have been physiologically compromised in distinct areas of the cochlea. It is well known that the outer hair cells are physiologically vulnerable. If small sections of outer hair cells are damaged (e.g. by noise) the tuning in that region may be altered dramatically, even though the tuning in the adjoining regions is normal. If this was the case, the auditory filter may be wider at this frequency but when the OAE suppression is tested, a less select group of outer hair cells will contribute to the emission, and the suppression may appear normal. In this experiment, it is possible that the subjects tested had less outer hair cell damage in the 2kHz region than at 1kHz and therefore the OAE suppression test represented the outer hair cell and efferent function in the region of the auditory filter more accurately. Thus,
the relationship between the two tests could be observed. There is some evidence from past work that this line of reasoning may have some validity. Fagelson and Champlin (1997) found that filters at close neighbouring frequencies differed in their frequency selectivity, particularly at low levels where an active feedback mechanism would have most effect.

The observation that the filter shape at 2kHz was related to the suppression when stimulated or suppressed at 2kHz, but not when the emission stimulation or suppression was at 1kHz or over a wide band of frequencies, is noteworthy. This also indicates uneven effectiveness of outer hair cell active feedback and efferent control of BM motion over the length of the cochlea.

### 9.5 Conclusion

A relationship was found between the level of efferent activity, as measured by the suppression of OAEs, and the shape of the 2kHz auditory filter. When compared to the suppression of 2kHz tones by contralateral white noise, an increase in efferent activity was associated with a broader filter. This would be expected if one considers that the broad band efferent interference would affect the high degree of frequency selectivity naturally provided by the selective amplification of BM motion. A higher level of suppression of click evoked emissions by a 2kHz tone was however associated with narrower filter shapes. It was considered that this suppression measure represented a much more select group of efferent fibres, which possibly act slightly off the centre frequency of the filter thus suppressing only the response in the side of the filter. Damping selectively off-frequency may allow for enhanced tuning.

In contrast, results from the previous chapters showed a lack of similar relationships at 1kHz and the present chapter showed a lack of correlation of the 2kHz filter with the OAE suppression tests involving 1kHz or broadband stimulation. This leads one to suspect that irregularities in the active feedback contribution to BM motion, or irregularities in the influence of efferent activation, are present along the length of the cochlea and account for the results observed.
Chapter 10

Discussion
10. Discussion

Past studies have indicated that the efferent system can affect auditory tuning. Elimination of efferent input to the cochlea was found to affect behavioural, psychophysical and physiological measures of frequency selectivity (Capps and Ades 1968; Carlier and Pujol 1982; Dewson 1968; Trahiotis and Elliott 1970). Electrical OCB stimulation has produced changes in the tuning of inner hair cells, afferent nerve fibres and BM motion (Brown et al. 1983; Guinan and Gifford 1988b; Murugasu and Russell 1996). This set of experiments explored the possible link between efferent function and auditory frequency selectivity in humans.

In this study, the auditory filter shape was found to broaden significantly under contralateral white noise stimulation. This is consistent with the past evidence from electrophysiological and OAE measurements, which have indicated that the medial efferent system acts to dampen the motion of the BM, causing a suppression in nerve fibre responses. Reduction in the active mechanical feedback to the cochlea has a deteriorating effect on the tuning. This has been shown to occur in past studies where the outer hairs cells were destroyed causing the tuning curve to broaden (Robertson and Johnstone 1979).

The possibility that the broadening may be partially due to a general increase in level, due to the contralateral noise, can not be entirely ruled out. However, evidence from blocking of the contralateral ear, the broadening of the upper slope of the filter (not normally associated with a straightforward increase in level) and the change in the detection efficiency (k), indicate that cross-over is not the sole cause of the broadening. It is also possible that central masking may have caused the tuning curves to broaden. However, past studies have shown central masking effects to be small (Zwislocki et al. 1967). Middle ear reflexes are unlikely to have produced a broadening of the auditory filter. Even if the level of the contralateral stimuli was loud enough to activate the reflex, this would have had the effect of attenuating the incoming filter shape stimuli. Filter shapes measured at lower levels are narrower rather than broader and thus this does not account for the effects seen.

How efferent induced broadening of cochlear tuning is helpful in everyday situations of frequency discrimination in competing background noise is unclear. In order for
damping of BM motion to actually improve tuning of the auditory filter, it would have to be restricted to certain frequency ranges of the cochlea. Suppression of BM motion would have to occur in the tails of the filter leaving the best frequency undamped. Evidence from auditory nerve fibre rate-level measurements has indicated that the efferent system may influence detection of signals in noise by restoring the dynamic range of operation of the fibres (Kawase et al. 1993; Winslow and Sachs 1987). Thus, the change in intensity caused by addition of the signal of interest can be coded more effectively. There is evidence for this line of reasoning from the present results, which have shown an increase in the dynamic range of the auditory filter during contralateral stimulation.

The detection efficiency (k) at the filter output was also found to increase significantly during efferent stimulation, and additionally, those subjects with greater efferent OAE suppression had better detection efficiency (at 1kHz). This means that these subjects find it easier to hear the probe tone in the background noise. This again may be linked to an efferent induced restoration of the dynamic range. This result in turn ties in with the finding that the threshold of detection of the tone in noise during 1kHz NBN stimulation in the contralateral ear was related to the efferent activity measured from OAE suppression. Similarly, Micheyl and Collet (1996) found that better detection of tones in noise was associated with greater level of efferent function, although this was at 2kHz.

The effects observed in this study could be attributed to the action of the medial portion of the OCB. It is the medial system that has been studied predominantly in past studies and has been found to have a suppressive effect on BM motion and nerve responses. The action of the lateral system is still unknown. In contrast to the medial fibres, which synapse at the outer hair cells in a relative diffuse frequency range of the cochlea (over about an octave), the lateral system tends to project to the type I afferents in a uniform and tonotopically precise manner. Thus, it is plausible that, whilst the medial efferents cause a general wide spread damping of BM motion, reducing the response to background noise, the lateral efferents act to disinhibit the response at the signal frequency. The effect of this on the auditory filter would be one of increasing the dynamic range. The bandwidth of the filter would become narrower only if the lateral efferent action was exceptionally frequency specific. Given that the lateral efferents are likely to act over a range of frequencies, the tip of the filter would be found to broaden whereas the tails would be suppressed. The results from this experiment fit in neatly
with this theory, whereby the bandwidth and dynamic range of the filter were greater with higher levels of efferent activity.

Many of the variables compared in these experiments proved not to be significantly correlated, indicating that the relationship between the two tests was not strong or straightforward. However, examination of the significant results from the different tests reveals that in the majority of cases the direction of correlation is in keeping with the theory that the efferent dampens BM motion, widening the auditory filter and increasing the dynamic range and detection efficiency. The shape of the 1kHz auditory filter measured without any contralateral noise was not, in general, found to be related to the OAE magnitude or suppression. Past evidence had suggested that tests involving only monaural stimuli did not activate the efferent system sufficiently for effects to be observed, possibly because most efferents have binaural inputs (Liberman 1988). However, closer examination of the results, particularly the DPOAE suppression data, revealed some strong and consistent correlations.

Addition of contralateral white noise caused a significant widening of the filter shape. This was not related to efferent OAE suppression and it was considered that this might be due to the broadband nature of the efferent activation in the filter shape test. Three different narrowband noise stimuli were then used as a more frequency specific way of activating the efferent system. These stimuli were much less effective in changing the shape of the auditory filter, and maybe because of this, there was still little evidence of a strong link.

The relationship between efferent function and frequency selectivity seemed to be more apparent at 2kHz than at 1kHz. This is in keeping with past studies, which have shown similar differences between these two frequencies (Micheyl and Collet 1996). This may reflect a lack of uniformity in the efferent innervation and/or efferent effectiveness along the length of the cochlea. Another possibility is that selective damage to small groups of outer hair cells may compromise tuning in specific areas, but may be too small to observe in OAE studies. It may also explain why some of the other past studies on humans have shown little relationship between the efferent system and frequency selectivity, since they were only tested at 1kHz (Scharf et al. 1997; Scharf et al. 1994).

The signal-in-noise stimuli encountered in day to day situations are generally rapidly changing. In order for the efferent system to provide any useful changes to cochlear
mechanics, it needs to react quickly to these changes. Measurement of the latency of efferent action showed this to be the case. Efferent stimulation started to suppress active process in the contralateral cochlea within 17 to 20 ms. This result suggested that the activation time in humans was similar to that which had been previously recorded in animals (Cody and Johnstone 1982a; Fex 1962; Fex 1967; Gummer et al. 1988; Konishi and Slepian 1971; Liberman and Brown 1986).

The detailed examination of the OAE results in comparison to the psychoacoustic test of auditory filter shape has not been attempted by any previous published studies. The filtering of the TOAEs did not show any great trends in the data that were hidden by examining the whole frequency response. TOAEs are not greatly frequency specific in the nature of production and represent contributions from active processes over broad frequency range of the cochlea. It was considered that by restricting the frequency range of the response, the functioning of a narrower region of the cochlea could be considered with less confounding influence from other regions. This may well have been the case in these results but the specificity was still not good enough to show a strong relationship with the auditory filter in that region. Additionally, windowing the response from 10 to 20ms did not overall help to clarify the picture, although the suppression tended to be larger as observed in the latency experiment.

The results from DPOAE suppression showed results that were more promising. DPs are much more frequency specific in nature of production and thus, although a direct frequency map can not be assumed, are probably able to reveal more information about cochlear function in a particular place. There appeared to be no link between frequency selectivity and the presence or absence of SOAEs. One might expect that a localised disturbance in active mechanisms, such as that producing SOAEs, would interfere with the fine tuning of the cochlear amplifier. This did not seem to be the case, although further detailed examination of the frequency of the SOAEs may prove otherwise.

Given that contralateral stimulation broadened the auditory filter significantly, and that this was likely to be due to the effect of the efferent system, it is curious that there is not a stronger relation between the filter shape and the OAE results. Although both measurements examine changes in the cochlea, the psychoacoustic study is also dependent upon interactions at all stages of the neural pathway to the cortex. Additionally, the results rely on a subjective response. OAE measurements have the
advantage of being objective, however, they are influenced to a greater extent by the status of the middle ear, since to record a response, the signal has to pass through the middle ear twice. Factors such as scarring of the eardrum, wax, effusion and disease would all affect the results of OAE measurements more.

The measurement of filter shape, by its very nature, analyses cochlear function over a narrow range of frequencies. The OAE measurements, particularly the TOAEs, are more likely to be influenced by contributions from active processes occurring at other points in the cochlea. Constructive and destructive interference of the incoming and outgoing travelling waves mean that the response from a particular place is complicated by many other factors. Therefore, this introduces a source of variability between the two types of measurement, meaning for example that local changes to say outer hair cell function may affect the auditory filter but be unmeasurable by OAEs. Furthermore, the stimuli used in the two tests were similar but not identical. Use of the same stimuli may help to bring out a well-hidden relationship.

One must also consider the possibility that the action of the efferent system may have been to change the centre frequency of the auditory filter. There is conflicting evidence that this might be the case from the past literature. Williams and Brown (1997) found that contralateral stimulation did not alter the centre frequency of the DP tuning curve, but changes in the centre frequency of BM tuning have been noted (Murugasu and Russell 1996). The measurements of filter shape in the present experiments were all designed to measure the filter shape changes at the same centre frequency. It would be interesting to make filter shape measurements at neighbouring frequencies to see how they were affected.

Attentional effects may have also interfered with the accuracy of the tests. Attempts were made to control attention during the OAE studies, by making subjects read whilst being tested. However, in the psychoacoustic test the subjects were attending to the stimuli thus allowing for the possibility of additional central input via the efferent system to the cochlea. Studies where the efferent system has been stimulated electrically have found that the maximum rate for the stimulus to drive the fibres is at 100/s, whereas the maximum effect on the cochlea occurs at 400/s (Fex 1962; Liberman and Brown 1986). This suggests that central efferent input may work alongside OCB reflex input to produce maximum effects on frequency selectivity in the cochlea.
Chapter 11

Concluding Remarks
11. **Concluding Remarks**

The objective of these experiments was to study the possible role of the auditory efferent system in human frequency selectivity that had been suggested by previous animal studies. Past work on human subjects has not made the comparison between comprehensive studies of OAEs, and detailed analysis of the shape of the auditory filter.

The results from these studies tend to confirm the data from past studies on animals, which have suggested that activation of the efferent system produces a broadening of tuning in the cochlea and an increase in the dynamic range. In addition, the relationship between efferent activity and frequency selectivity seem to be stronger at 2kHz than at 1kHz, also supporting previous findings in humans.

However, the evidence for a link between efferent activity and frequency selectivity was by no means conclusive of a strong relationship. Further work is required to investigate the details of this relationship. Firstly to verify the nature of the effect at 1kHz, and secondly to confirm the frequency dependence of the relationship. The OAE and the psychoacoustic tests employed here are measuring aspects of a highly complex system, which makes analysis of the results difficult. By minimising the factors which produce variability between the psychoacoustic and the OAE tests future studies should help to resolve this issue.

Control of attention levels may help to equalise central input for the two tests. If the OAE test could be organised so that the subject was attending to the signal evoking the OAE then this would aid in reducing this form of variability.

Psychoacoustic tests suffer from being subjective and requiring concentration from the subjects. Development of a DP test for frequency selectivity, such as that used by Abdala et al. (1996), using a third ipsilateral suppresser tone, may provide a more objective measure.

The suppression of distortion products produced interesting results, and further detailed analysis at other frequencies, of the type undertaken here, would be useful to present a full picture of the relationship across frequency.
Another obvious next step is to study the efferent effect on frequency selectivity at other frequencies, since the present results suggest that it is not constant across frequency. Particularly interesting will be the results from studies at higher frequencies because the trend indicated by this study is that there is a greater input from the efferent system in controlling frequency selectivity as frequency increases. Another feature of the effect, which should be examined, is the possible change in centre frequency of the auditory filter, which has been suggested by some past studies. Additionally, use of exactly the same masker in both ears may help to reveal a relationship, because the frequencies over which the efferent system would be activated, and therefore provide damping, would correspond to the masker.

The anti-masking effect provided by the OCB was noted by Kawase et al. (1993) to occur only at higher levels. It would therefore be interesting to look at the effect on the auditory filter when measured at different levels. By doing this, the system could be assessed at a different point in its dynamic range with varying levels of saturation of active processes and nerve fibre responses.

A possible reason for the lack of clarity in the results is that in normally hearing subjects, the range of differences in efferent operation and frequency selectivity are not great enough to show a relationship, given the inherent errors in the experimental procedures. Subjects with either very under or over active efferent systems may provide interesting results. Patients, who have undergone a vestibular neurectomy for Ménière’s disease, where the efferent nerve is severed, are an interesting group to test. Although they do not in general report difficulties in frequency selectivity, it is possible that the ‘good ear’ is compensating. Past studies on these patients have not looked at the auditory filter shape in as much detail as the present study. Another interesting group to test may be patients reporting hyperacusis. Some past studies have attributed this oversensitivity to sound to be due to dysfunction of the efferent system. Other groups of patients with specific lesions to the efferent pathway, such as some multiple sclerosis or acoustic neuroma patients could also be examined.

It would also be interesting to study the effect of the efferent system on development of frequency selectivity. The efferent system may somehow guide the development of tuning for the most important sounds encountered. There is some evidence for this in
that broader tuning curves developed after sectioning of the OCB in neonatal cats (Walsh et al. 1998).

In summary, this thesis presents evidence that the efferent system may be involved in frequency selectivity in humans. The relationship does not appear to be particularly strong or straightforward but this may be due to the experimental methods employed. Publications resulting from this work are included in Appendix 5.
Chapter 12

References
12. References


Appendix 1
13. Appendix 1: Program written to test auditory filter at 1kHz

;---------------------------------------------------------------------
; Fixed Probe Setup - 5 notches, for use with or without contralateral noise
;---------------------------------------------------------------------
;
; COND: 1 1
; PLOT: 0
; Condition #1 run 55 trials for 5 different noise notch separations
; Signal frequency is 1000Hz

NUM_TRIALS: 55
DEFAULT_GATE_TIME: 5
INTER_INT_DELAY: 100.0
INTER_TRIAL_DELAY: 250.0
CAL_MODE: 1

TRACK: 1 -60.0 -90.0 0.0
BIG_STEP: -10
SMALL_STEP: -2
NUM_UP: 1
NUM_DOWN: 3
NUM_BIG_REVS: 3
NUM_TOSS_REVS: 3
ENDTRACK

STEP: 1 600.0
STEP: 2 1000.0
STEP: 3 1000.0
STEP: 4 1400.0

; Signal definition
SIGNAL: 1 350.0 TRACK1 50.0
LINBAND: 0.0 0.0 STEP1 STEP2
LINBAND: 0.0 0.0 STEP3 STEP4
ENDSIGNAL

; Signal definition
SIGNAL: 3 400.0 -99.0 0.0
; this only gives a time reference
TONE: -99.0 1000.0 0.0
ENDSIGNAL

; Signal definition
SIGNAL: 4 250.0 -77.6 100.0
; this is the probe tone
TONE: 0.0 1000.0 0.0
ENDSIGNAL

; Standard definition
SIGNAL: 5 350.0 TRACK1 50.0
LINBAND: 0.0 0.0 STEP1 STEP2
LINBAND: 0.0 0.0 STEP3 STEP4
ENDSIGNAL

; Standard definition
SIGNAL: 7 400.0 -99.0 0.0
; only a time reference
TONE: -99.0 1000.0 0.0
ENDSIGNAL

; Signal definition
SIGNAL: 8 250.0 -77.6 100.0
; tone absent
TONE: -99.0 1000.0 0.0
ENDSIGNAL

; Calibration TONE for Channel-A
SIGNAL: 15 300.0 0.0 0.0  
TONE: 0.0 1000.0 0.0  
CALIBRATE_LEVEL: 61  
ENDSIGNAL  

; Calibration TONE for Channel-B  
SIGNAL: 16 300.0 0.0 0.0  
TONE: 0.0 1000.0 0.0  
CALIBRATE_LEVEL: 102.5  
ENDSIGNAL  

ENDCOND  

;-----------------------------------------------------------------------------  
; Fixed Probe Setup -2nd notch, for use with or without contra noise  
;-----------------------------------------------------------------------------  

; COND: 2 1  
; PLOT: 0  
; Condition #2 run 55 trials for 5 different noise notch separations  
; Signal frequency is 1000Hz  
NUM_TRIALS: 55  
DEFAULT_GATE_TIME: 5  
INTER_INT_DELAY: 100.0  
INTER_TRIAL_DELAY: 250.0  
CAL_MODE: 0  

TRACK: 1 -45.0 -90.0 0.0  
BIG_STEP: -10  
SMALL_STEP: -2  
NUM_UP: 1  
NUM_DOWN: 3  
NUM_BIG_REVS: 3  
NUM_TOSS_REVS: 3  
ENDTRACK  

STEP: 1 500.0  
STEP: 2 900.0  
STEP: 3 1300.0  
STEP: 4 1700.0  

; Signal definition  
SIGNAL: 1 350.0 TRACK1 50.0  
LINBAND: 0.0 0.0 STEP1 STEP2  
LINBAND: 0.0 0.0 STEP3 STEP4  
ENDSIGNAL  

; Signal definition  
SIGNAL: 3 400.0 -99.0 0.0  
; this only gives a time reference  
TONE: -99.0 1000.0 0.0  
ENDSIGNAL  

; Signal definition  
SIGNAL: 4 250.0 -77.6 100.0  
; this is the probe tone  
TONE: 0.0 1000.0 0.0  
ENDSIGNAL  

; Standard definition  
SIGNAL: 5 350.0 TRACK 1 50.0  
LINBAND: 0.0 0.0 STEP1 STEP2  
LINBAND: 0.0 0.0 STEP3 STEP4  
ENDSIGNAL  

; Standard definition  
SIGNAL: 7 400.0 -99.0 0.0
ENDCOND

; Fixed Probe Setup - 3rd notch, for use with or without contra noise
; COND: 3 1
; PLOT: 0
; Condition #3 run 55 trials for 5 different noise notch separations
; Signal frequency is 1000Hz
NUM_TRIALS: 55
DEFAULT_GATE_TIME: 5
INTER_INT_DELAY: 100.0
INTER_TRIAL_DELAY: 250.0
CAL_MODE: 0

TRACK: 1 -35.0 -90.0 0.0
BIG_STEP: -10
SMALL_STEP: -2
NUM_UP: 1
NUM_DOWN: 3
NUM_BIG_REVS: 3
NUM_TOSS_REVS: 3
ENDTRACK

STEP: 1 300.0
STEP: 2 700.0
STEP: 3 1100.0
STEP: 4 1500.0

; Signal definition
SIGNAL: 1 350.0 TRACK1 50.0
LINBAND: 0.0 0.0 STEP1 STEP2
LINBAND: 0.0 0.0 STEP3 STEP4
ENDSIGNAL

; Signal definition
SIGNAL: 3 400.0 -99.0 0.0
; this only gives a time reference
TONE: -99.0 1000.0 0.0
ENDSIGNAL

; Signal definition
SIGNAL: 4 250.0 -77.6 100.0
; this is the probe tone
ENDSIGNAL

; Standard definition
SIGNAL: 5 350.0 TRACK1 50.0
LINBAND: 0.0 0.0 STEP1 STEP2
LINBAND: 0.0 0.0 STEP3 STEP4
ENDSIGNAL

; Standard definition
SIGNAL: 7 400.0 -99.0 0.0
; only a time reference
TONE: -99.0 1000.0 0.0
ENDSIGNAL

; Signal definition
SIGNAL: 8 250.0 -77.6 100.0
; tone absent
TONE: -99.0 1000.0 0.0
ENDSIGNAL

; Calibration TONE for Channel-A
SIGNAL: 15 300.0 0.0 0.0
TONE: 0.0 1000.0 0.0
CALIBRATE_LEVEL: 61
ENDSIGNAL

; Calibration TONE for Channel-B
SIGNAL: 16 300.0 0.0 0.0
TONE: 0.0 1000.0 0.0
CALIBRATE_LEVEL: 102.5
ENDSIGNAL

ENDCOND

; Fixed Probe Setup -4th notch, for use with or without contra noise

COND: 4 1
PLOT: 0
Condition #4 run 55 trials for 5 different noise notch separations
Signal frequency is 1000Hz
NUM_TRIALS: 55
DEFAULT_GATE_TIME: 5
INTER_INT_DELAY: 100.0
INTER_TRIAL_DELAY: 250.0
CAL_MODE: 0

TRACK: 1 -25.0 -90.0 0.0
BIG_STEP: -10
SMALL_STEP: -2
NUM_UP: 1
NUM_DOWN: 3
NUM_BIG_REVS: 3
NUM_TOSS_REVS: 3
ENDTRACK

STEP: 1 100.0
STEP: 2 500.0
STEP: 3 1300.0
STEP: 4 1700.0

; Signal definition
SIGNAL: 1 350.0 TRACK1 50.0
LINBAND: 0.0 0.0 STEP1 STEP2
LINBAND: 0.0 0.0 STEP3 STEP4
ENDSIGNAL

; Signal definition
SIGNAL: 3 400.0 -99.0 0.0
; this only gives a time reference
TONE: -99.0 1000.0 0.0
ENDSIGNAL

; Signal definition
SIGNAL: 4 250.0 -77.6 100.0
; this is the probe tone
TONE: 0.0 1000.0 0.0
ENDSIGNAL

; Standard definition
SIGNAL: 5 350.0 TRACK1 50.0
LINBAND: 0.0 0.0 STEP1 STEP2
LINBAND: 0.0 0.0 STEP3 STEP4
ENDSIGNAL

; Standard definition
SIGNAL: 7 400.0 -99.0 0.0
; only a time reference
TONE: -99.0 1000.0 0.0
ENDSIGNAL

; Signal definition
SIGNAL: 8 250.0 -77.6 100.0
; tone absent
TONE: -99.0 1000.0 0.0
ENDSIGNAL

; Calibration TONE for Channel-A
SIGNAL: 15 300.0 0.0 0.0
TONE: 0.0 1000.0 0.0
CALIBRATE_LEVEL: 61
ENDSIGNAL

; Calibration TONE for Channel-B
SIGNAL: 16 300.0 0.0 0.0
TONE: 0.0 1000.0 0.0
CALIBRATE_LEVEL: 102.5
ENDSIGNAL

ENDCOND

; Fixed Probe Setup -5th notch, for use with or without contra noise
COND: 5 1
PLOT: 0
Condition #5 run 55 trials for 5 different noise notch separations
Signal frequency is 1000Hz
NUM_TRIALS: 55
DEFAULT_GATE_TIME: 5
INTER_INT_DELAY: 100.0
INTER_TRIAL_DELAY: 250.0
CAL_MODE: 0

TRACK: 1 -30.0 -90.0 0.0
BIG_STEP: -10
SMALL_STEP: -2
NUM_UP: 1
NUM_DOWN: 3
NUM_BIG_REVS: 3
NUM_TOSS_REVS: 3
ENDTRACK

STEP: 1 300.0
STEP: 2 700.0
STEP: 3 1500.0
STEP: 4 1900.0

; Signal definition
SIGNAL: 1 350.0 TRACK1 50.0
LINBAND: 0.0 0.0 STEP1 STEP2
LINBAND: 0.0 0.0 STEP3 STEP4
ENDSIGNAL

; Signal definition
SIGNAL: 3 400.0 -99.0 0.0
; this only gives a time reference
TONE: -99.0 1000.0 0.0
ENDSIGNAL

; Signal definition
SIGNAL: 4 250.0 -77.6 100.0
; this is the probe tone
TONE: 0.0 1000.0 0.0
ENDSIGNAL

; Standard definition
SIGNAL: 5 350.0 TRACK1 50.0
LINBAND: 0.0 0.0 STEP1 STEP2
LINBAND: 0.0 0.0 STEP3 STEP4
ENDSIGNAL

; Standard definition
SIGNAL: 7 400.0 -99.0 0.0
; only a time reference
TONE: -99.0 1000.0 0.0
ENDSIGNAL

; Signal definition
SIGNAL: 8 250.0 -77.6 100.0
; tone absent
TONE: -99.0 1000.0 0.0
ENDSIGNAL

; Calibration TONE for Channel-A
SIGNAL: 15 300.0 0.0 0.0
TONE: 0.0 1000.0 0.0
CALIBRATE_LEVEL: 61
ENDSIGNAL

; Calibration TONE for Channel-B
SIGNAL: 16 300.0 0.0 0.0
TONE: 0.0 1000.0 0.0
CALIBRATE_LEVEL: 102.5
ENDSIGNAL

ENDCOND
Appendix 2
14. Appendix 2: Program written to test auditory filter at 2kHz

;-----------------------------------------------------------------------------
; Fixed Probe Setup -5 notches
;-----------------------------------------------------------------------------
;
; COND: 1 1
; PLOT: 0
; Condition #1 run 55 trials for 5 different noise notch separations
; Signal frequency is 2000Hz
NUM_TRIALS: 55
DEFAULT_GATE_TIME: 5
INTER_INT_DELAY: 100.0
INTER_TRIAL_DELAY: 300.0
CAL_MODE: 1

TRACK: 1 -65.0 -90.0 0.0
BIG_STEP: -10
SMALL_STEP: -2
NUM_UP: 1
NUM_DOWN: 3
NUM_BIG_REVS: 3
NUM_TOSS_REVS: 3
ENDTRACK

STEP: 1 1200.0
STEP: 2 2000.0
STEP: 3 2000.0
STEP: 4 2800.0

; Signal definition
SIGNAL: 1 350.0 TRACK1 0.0
LINBAND: 0.0 0.0 STEP1 STEP2
LINBAND: 0.0 0.0 STEP3 STEP4
ENDSIGNAL

; Signal definition
SIGNAL: 2 250.0 -80.0 50.0
TONE: 0.0 2000.0 0.0
ENDSIGNAL

; Standard definition
SIGNAL: 3 350.0 TRACK1 0.0
LINBAND: 0.0 0.0 STEP1 STEP2
LINBAND: 0.0 0.0 STEP3 STEP4
ENDSIGNAL

; Standard definition
SIGNAL: 4 250.0 -80.0 50.0
TONE: -99.0 2000.0 0.0
ENDSIGNAL

; Calibration TONE for Channel-A
SIGNAL: 15 300.0 0.0 0.0
TONE: 0.0 2000.0 0.0
CALIBRATE_LEVEL: 63.5
ENDSIGNAL

; Calibration TONE for Channel-B
SIGNAL: 16 300.0 0.0 0.0
TONE: 0.0 2000.0 0.0
CALIBRATE_LEVEL: 102.5
ENDSIGNAL

ENDCOND
### Fixed Probe Setup - 2ND notch

**Condition #2 run 55 trials for 5 different noise notch separations**

- **Signal frequency is 2000Hz**
- **NUM_TRIALS:** 55
- **DEFAULT_GATE_TIME:** 5
- **INTER_INT_DELAY:** 100.0
- **INTER_TRIAL_DELAY:** 300.0
- **CAL_MODE:** 0

**Track Parameters:**

- **BIG_STEP:** -10
- **SMALL_STEP:** -2
- **NUM_UP:** 1
- **NUM_DOWN:** 3
- **NUM_BIG_REVS:** 3
- **NUM_TOSS_REVS:** 3

**Steps:**

- **STEP:** 1 1000.0
- **STEP:** 2 1800.0
- **STEP:** 3 2600.0
- **STEP:** 4 3400.0

**Signal Definitions:**

- **SIGNAL:** 1 350.0 TRACK1 0.0
  - **LINBAND:** 0.0 0.0 STEP1 STEP2
  - **LINBAND:** 0.0 0.0 STEP3 STEP4

**Standard Definition:**

- **SIGNAL:** 2 250.0 -80.0 50.0
  - **TONE:** 0.0 2000.0 0.0

**Calibration TONE for Channel-A**

- **SIGNAL:** 15 100.0 0.0 0.0
  - **TONE:** 0.0 2000.0 0.0
  - **CALIBRATE_LEVEL:** 63.5

**Calibration TONE for Channel-B**

- **SIGNAL:** 16 100.0 0.0 0.0
  - **TONE:** 0.0 2000.0 0.0
  - **CALIBRATE_LEVEL:** 102.5

**ENDCOND**

### Fixed Probe Setup - 3rd notch

---

---
Condition #3: Run 55 trials for 5 different noise notch separations.
Signal frequency is 2000 Hz.

**Parameters:**
- **NUM TRIALS:** 55
- **DEFAULT_GATE_TIME:** 5
- **INTER_INT_DELAY:** 100.0
- **INTER_TRIAL_DELAY:** 300.0
- **CAL_MODE:** 0

**Track 1:**
- **BIG_STEP:** -10
- **SMALL_STEP:** -2
- **NUM_UP:** 1
- **NUM_DOWN:** 3
- **NUM_BIG_REVS:** 3
- **NUM_TOSS_REVS:** 3

**Steps:**
1. **600.0**
2. **1400.0**
3. **2200.0**
4. **3000.0**
5. **-80.0**

**Signal Definitions:**
- **SIGNAL 1:**
  - **TRACK1:** 0.0
  - **LINBAND:** 0.0 0.0 **STEP1** **STEP2**
  - **LINBAND:** 0.0 0.0 **STEP3** **STEP4**
- **SIGNAL 2:**
  - **STEP5:** 50.0
  - **TONE:** 0.0 2000.0 0.0
- **SIGNAL 3:**
  - **TRACK1:** 0.0
  - **LINBAND:** 0.0 0.0 **STEP1** **STEP2**
  - **LINBAND:** 0.0 0.0 **STEP3** **STEP4**
- **SIGNAL 4:**
  - **STEP5:** 50.0
  - **TONE:** -99.0 2000.0 0.0

**Calibration Tones:**
- **SIGNAL 15:**
  - **TONE:** 0.0 2000.0 0.0
  - **CALIBRATE_LEVEL:** 63.5

- **SIGNAL 16:**
  - **TONE:** 0.0 2000.0 0.0
  - **CALIBRATE_LEVEL:** 102.5

**End Condition**

---

**Condition #4:**

**Parameters:**
- **NUM TRIALS:** 55
- **DEFAULT_GATE_TIME:** 5
- **INTER_INT_DELAY:** 100.0
- **INTER_TRIAL_DELAY:** 300.0
- **CAL_MODE:** 0

**Track 1:**
- **BIG_STEP:** -10
- **SMALL_STEP:** -2
- **NUM_UP:** 1
- **NUM_DOWN:** 3
- **NUM_BIG_REVS:** 3
- **NUM_TOSS_REVS:** 3

**Steps:**
1. **600.0**
2. **1400.0**
3. **2200.0**
4. **3000.0**
5. **-80.0**

**Signal Definitions:**
- **SIGNAL 1:**
  - **TRACK1:** 0.0
  - **LINBAND:** 0.0 0.0 **STEP1** **STEP2**
  - **LINBAND:** 0.0 0.0 **STEP3** **STEP4**
- **SIGNAL 2:**
  - **STEP5:** 50.0
  - **TONE:** 0.0 2000.0 0.0
- **SIGNAL 3:**
  - **TRACK1:** 0.0
  - **LINBAND:** 0.0 0.0 **STEP1** **STEP2**
  - **LINBAND:** 0.0 0.0 **STEP3** **STEP4**
- **SIGNAL 4:**
  - **STEP5:** 50.0
  - **TONE:** -99.0 2000.0 0.0

**Calibration Tones:**
- **SIGNAL 15:**
  - **TONE:** 0.0 2000.0 0.0
  - **CALIBRATE_LEVEL:** 63.5

- **SIGNAL 16:**
  - **TONE:** 0.0 2000.0 0.0
  - **CALIBRATE_LEVEL:** 102.5

**End Condition**

---
; Condition #4 run 55 trials for 5 different noise notch separations
; Signal frequency is 2000Hz
NUM_TRIALS: 55
DEFAULT_GATE_TIME: 5
INTER_INT_DELAY: 100.0
INTER_TRIAL_DELAY: 300.0
CAL_MODE: 0

TRACK: 1 -30.0 -90.0 0.0
BIG_STEP: -10
SMALL_STEP: -2
NUM_UP: 1
NUM_DOWN: 3
NUM_BIG_REVS: 3
NUM_TOSS_REVS: 3
ENDTRACK

STEP: 1 200.0
STEP: 2 1000.0
STEP: 3 2600.0
STEP: 4 3400.0
STEP: 5 -80.0

; Signal definition
SIGNAL: 1 350.0 TRACK1 0.0
LINBAND: 0.0 0.0 STEP1 STEP2
LINBAND: 0.0 0.0 STEP3 STEP4
ENDSIGNAL

; Signal definition
SIGNAL: 2 250.0 STEP5 50.0
TONE: 0.0 2000.0 0.0
ENDSIGNAL

; Standard definition
SIGNAL: 3 350.0 TRACK1 0.0
LINBAND: 0.0 0.0 STEP1 STEP2
LINBAND: 0.0 0.0 STEP3 STEP4
ENDSIGNAL

; Standard definition
SIGNAL: 4 250.0 STEP5 50.0
TONE: -99.0 2000.0 0.0
ENDSIGNAL

; Calibration TONE for Channel-A
SIGNAL: 15 100.0 0.0 0.0
TONE: 0.0 2000.0 0.0
CALIBRATE_LEVEL: 63.5
ENDSIGNAL

; Calibration TONE for Channel-B
SIGNAL: 16 100.0 0.0 0.0
TONE: 0.0 2000.0 0.0
CALIBRATE_LEVEL: 100.0
ENDSIGNAL

ENDCOND

; Fixed Probe Setup -5TH notch
;-----------------------------------------------------------------------
COND: 5 1
PLOT: 0
; Condition #5 run 55 trials for 5 different noise notch separations
; Signal frequency is 2000Hz
NUM_TRIALS: 55
DEFAULT_GATE_TIME: 5
INTERJNT_DELAY: 100.0
INTER_TRIAL_DELAY: 300.0
CAL_MODE: 0

TRACK: 1 -35.0 -90.0 0.0
BIG_STEP: -10
SMALL_STEP: -2
NUM_UP: 1
NUM_DOWN: 3
NUM_BIG_REVS: 3
NUM_TOSS_REVS: 3
ENDTRACK

STEP: 1 600.0
STEP: 2 1400.0
STEP: 3 3000.0
STEP: 4 3800.0
STEP: 5 -80.0

; Signal definition
SIGNAL: 1 350.0 TRACK1 0.0
LINBAND: 0.0 0.0 STEP1 STEP2
LINBAND: 0.0 0.0 STEP3 STEP4
ENDSIGNAL

; Signal definition
SIGNAL: 2 250.0 STEP5 50.0
TONE: 0.0 2000.0 0.0
ENDSIGNAL

; Standard definition
SIGNAL: 3 350.0 TRACK1 0.0
LINBAND: 0.0 0.0 STEP1 STEP2
LINBAND: 0.0 0.0 STEP3 STEP4
ENDSIGNAL

; Standard definition
SIGNAL: 4 250.0 STEP5 50.0
TONE: -99.0 2000.0 0.0
ENDSIGNAL

; Calibration TONE for Channel-A
SIGNAL: 15 100.0 0.0 0.0
TONE: 0.0 2000.0 0.0
CALIBRATE_LEVEL: 63.5
ENDSIGNAL

; Calibration TONE for Channel-B
SIGNAL: 16 100.0 0.0 0.0
TONE: 0.0 2000.0 0.0
CALIBRATE_LEVEL: 102.5
ENDSIGNAL

ENDCOND
15. Appendix 3: Correlation of DP suppression and auditory filter shape parameters

Table 15-1: Pearson’s correlation coefficients and significance values for the relationship between DP suppression (contralateral 1kHz tone) at various F2 frequencies and auditory filter shape parameters - DPGram

<table>
<thead>
<tr>
<th>F2 Frequency (Hz)</th>
<th>P (lower)</th>
<th>P (upper)</th>
<th>k</th>
<th>r</th>
<th>Symmetry (SI)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Correlation coefficient</td>
<td>P value</td>
<td>Correlation coefficient</td>
<td>P value</td>
<td>Correlation coefficient</td>
</tr>
<tr>
<td>708</td>
<td>-0.054</td>
<td>0.855</td>
<td>-0.095</td>
<td>0.746</td>
<td>-0.105</td>
</tr>
<tr>
<td>842</td>
<td>0.262</td>
<td>0.345</td>
<td>0.177</td>
<td>0.529</td>
<td>0.330</td>
</tr>
<tr>
<td>1001</td>
<td>-0.225</td>
<td>0.482</td>
<td>-0.286</td>
<td>0.367</td>
<td>-0.060</td>
</tr>
<tr>
<td>1184</td>
<td>-0.428</td>
<td>0.127</td>
<td>-0.530</td>
<td>0.051</td>
<td>-0.293</td>
</tr>
<tr>
<td>1416</td>
<td>-0.405</td>
<td>0.134</td>
<td>-0.721*</td>
<td>0.002</td>
<td>-0.453</td>
</tr>
<tr>
<td>1685</td>
<td>-0.240</td>
<td>0.406</td>
<td>-0.548*</td>
<td>0.042</td>
<td>-0.354</td>
</tr>
<tr>
<td>2002</td>
<td>-0.194</td>
<td>0.489</td>
<td>-0.643*</td>
<td>0.010</td>
<td>-0.331</td>
</tr>
<tr>
<td>2380</td>
<td>-0.179</td>
<td>0.522</td>
<td>-0.382</td>
<td>0.159</td>
<td>-0.111</td>
</tr>
<tr>
<td>2832</td>
<td>0.011</td>
<td>0.969</td>
<td>-0.212</td>
<td>0.448</td>
<td>0.085</td>
</tr>
<tr>
<td>3369</td>
<td>0.216</td>
<td>0.440</td>
<td>0.016</td>
<td>0.956</td>
<td>0.272</td>
</tr>
<tr>
<td>4004</td>
<td>0.037</td>
<td>0.899</td>
<td>-0.327</td>
<td>0.254</td>
<td>0.013</td>
</tr>
<tr>
<td>4761</td>
<td>-0.456</td>
<td>0.118</td>
<td>-0.598*</td>
<td>0.035</td>
<td>-0.551</td>
</tr>
<tr>
<td>5652</td>
<td>-0.634*</td>
<td>0.020</td>
<td>-0.359</td>
<td>0.228</td>
<td>-0.699*</td>
</tr>
<tr>
<td>6299</td>
<td>-0.241</td>
<td>0.386</td>
<td>0.006</td>
<td>0.982</td>
<td>-0.142</td>
</tr>
</tbody>
</table>

Table 15-2: Pearson’s correlation coefficients and significance values for the relationship between DP suppression (contralateral 1kHz tone) at various F2 frequencies and auditory filter shape parameters - Fine Structure around 1kHz

<table>
<thead>
<tr>
<th>F2 Frequency (Hz)</th>
<th>P (lower)</th>
<th>P (upper)</th>
<th>k</th>
<th>r</th>
<th>Symmetry (SI)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Correlation coefficient</td>
<td>P value</td>
<td>Correlation coefficient</td>
<td>P value</td>
<td>Correlation coefficient</td>
</tr>
<tr>
<td>745</td>
<td>-0.157</td>
<td>0.627</td>
<td>-0.021</td>
<td>0.948</td>
<td>-0.032</td>
</tr>
<tr>
<td>781</td>
<td>0.456</td>
<td>0.101</td>
<td>0.479</td>
<td>0.083</td>
<td>0.259</td>
</tr>
<tr>
<td>818</td>
<td>-0.030</td>
<td>0.918</td>
<td>-0.107</td>
<td>0.715</td>
<td>-0.310</td>
</tr>
<tr>
<td>854</td>
<td>-0.024</td>
<td>0.936</td>
<td>-0.094</td>
<td>0.748</td>
<td>-0.168</td>
</tr>
<tr>
<td>891</td>
<td>-0.114</td>
<td>0.699</td>
<td>-0.161</td>
<td>0.582</td>
<td>-0.301</td>
</tr>
<tr>
<td>928</td>
<td>-0.140</td>
<td>0.634</td>
<td>-0.096</td>
<td>0.744</td>
<td>-0.064</td>
</tr>
<tr>
<td>977</td>
<td>0.487</td>
<td>0.108</td>
<td>0.393</td>
<td>0.206</td>
<td>0.296</td>
</tr>
<tr>
<td>1013</td>
<td>0.114</td>
<td>0.710</td>
<td>-0.011</td>
<td>0.972</td>
<td>0.251</td>
</tr>
<tr>
<td>1062</td>
<td>0.180</td>
<td>0.556</td>
<td>0.167</td>
<td>0.587</td>
<td>-0.049</td>
</tr>
<tr>
<td>1111</td>
<td>-0.182</td>
<td>0.533</td>
<td>-0.284</td>
<td>0.325</td>
<td>-0.290</td>
</tr>
<tr>
<td>1160</td>
<td>-0.072</td>
<td>0.806</td>
<td>-0.187</td>
<td>0.522</td>
<td>-0.055</td>
</tr>
<tr>
<td>1208</td>
<td>-0.369</td>
<td>0.215</td>
<td>-0.413</td>
<td>0.161</td>
<td>-0.281</td>
</tr>
<tr>
<td>1257</td>
<td>-0.528*</td>
<td>0.043</td>
<td>-0.584*</td>
<td>0.022</td>
<td>-0.218</td>
</tr>
<tr>
<td>1318</td>
<td>-0.348</td>
<td>0.222</td>
<td>-0.391</td>
<td>0.167</td>
<td>-0.269</td>
</tr>
<tr>
<td>1379</td>
<td>0.271</td>
<td>0.329</td>
<td>0.366</td>
<td>0.179</td>
<td>0.218</td>
</tr>
<tr>
<td>1440</td>
<td>-0.024</td>
<td>0.935</td>
<td>-0.203</td>
<td>0.487</td>
<td>0.020</td>
</tr>
<tr>
<td>1501</td>
<td>-0.230</td>
<td>0.410</td>
<td>-0.473</td>
<td>0.075</td>
<td>-0.391</td>
</tr>
</tbody>
</table>

* Significant at p<0.05 level
Table 15-3: Pearson’s correlation coefficients and significance values for the relationship between DP suppression (contralateral 1kHz narrowband noise) at various F2 frequencies and auditory filter shape parameters - DPogram

<table>
<thead>
<tr>
<th>F2 Frequency (Hz)</th>
<th>P (lower) P value</th>
<th>P (upper) P value</th>
<th>k Correlation coefficient</th>
<th>r Correlation coefficient</th>
<th>Symmetry (SI) P value</th>
</tr>
</thead>
<tbody>
<tr>
<td>708</td>
<td>0.091 0.803</td>
<td>0.298 0.403</td>
<td>-0.034</td>
<td>0.925 0.316</td>
<td>-0.085 0.815</td>
</tr>
<tr>
<td>842</td>
<td>0.039 0.910</td>
<td>0.087 0.798</td>
<td>-0.257</td>
<td>0.446 0.154</td>
<td>-0.101 0.768</td>
</tr>
<tr>
<td>1001</td>
<td>-0.265 0.405</td>
<td>-0.492 0.104</td>
<td>-0.234</td>
<td>0.464 0.339</td>
<td>-0.047 0.486</td>
</tr>
<tr>
<td>1184</td>
<td>0.280 0.378</td>
<td>0.198 0.538</td>
<td>0.069</td>
<td>0.830 0.305</td>
<td>0.190 0.554</td>
</tr>
<tr>
<td>1416</td>
<td>-0.324 0.305</td>
<td>-0.565 0.056</td>
<td>-0.182</td>
<td>0.571 0.251</td>
<td>0.020 0.952</td>
</tr>
<tr>
<td>1685</td>
<td>-0.377 0.228</td>
<td>-0.621* 0.031</td>
<td>-0.283</td>
<td>0.372 0.342</td>
<td>-0.029 0.928</td>
</tr>
<tr>
<td>2002</td>
<td>-0.423 0.171</td>
<td>-0.499 0.099</td>
<td>-0.429</td>
<td>0.164 0.498</td>
<td>-0.188 0.559</td>
</tr>
<tr>
<td>2380</td>
<td>-0.135 0.676</td>
<td>-0.305 0.335</td>
<td>-0.266</td>
<td>0.403 0.128</td>
<td>0.003 0.992</td>
</tr>
<tr>
<td>2832</td>
<td>0.058 0.855</td>
<td>0.065 0.840</td>
<td>0.216</td>
<td>0.500 0.369</td>
<td>-0.009 0.977</td>
</tr>
<tr>
<td>3369</td>
<td>-0.457 0.135</td>
<td>-0.487 0.108</td>
<td>-0.287</td>
<td>0.366 0.371</td>
<td>-0.239 0.454</td>
</tr>
<tr>
<td>4004</td>
<td>-0.548 0.081</td>
<td>-0.478 0.137</td>
<td>-0.747*</td>
<td>0.098 0.332</td>
<td>-0.407 0.214</td>
</tr>
<tr>
<td>4761</td>
<td>0.011 0.975</td>
<td>0.074 0.839</td>
<td>-0.329</td>
<td>0.354 0.900</td>
<td>-0.045 0.902</td>
</tr>
<tr>
<td>5652</td>
<td>-0.353 0.287</td>
<td>-0.355 0.284</td>
<td>-0.501</td>
<td>0.116 0.313</td>
<td>-0.213 0.528</td>
</tr>
<tr>
<td>6299</td>
<td>0.040 0.920</td>
<td>0.177 0.603</td>
<td>0.011</td>
<td>0.975 0.046</td>
<td>0.058 0.866</td>
</tr>
</tbody>
</table>

Table 15-4: Pearson’s correlation coefficients and significance values for the relationship between DP suppression (contralateral 1kHz narrowband noise) at various F2 frequencies and auditory filter shape parameters – Fine structure around 1kHz

<table>
<thead>
<tr>
<th>F2 Frequency (Hz)</th>
<th>P (lower) P value</th>
<th>P (upper) P value</th>
<th>k Correlation coefficient</th>
<th>r Correlation coefficient</th>
<th>Symmetry (SI) P value</th>
</tr>
</thead>
<tbody>
<tr>
<td>745</td>
<td>0.270 0.451</td>
<td>0.039 0.914</td>
<td>0.098</td>
<td>0.787 0.320</td>
<td>0.274 0.443</td>
</tr>
<tr>
<td>781</td>
<td>-0.288 0.364</td>
<td>-0.452 0.140</td>
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<td>0.184 0.097</td>
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</tbody>
</table>

* significant at p<0.05
Appendix 4
16. Appendix 4: Scatterplots of the relationship between suppression of click evoked OAE by white noise and auditory filter shapes measured during contralateral narrow band noise

Plots A-C show the relationships between the suppression of click evoked OAEs by white noise, and the bandwidths of the auditory filter measured during 1kHz, 2kHz and 500Hz contralateral NBN respectively. Plots D-F show the comparison between the filter shape bandwidths (during 1kHz, 2kHz and 500Hz NBN respectively) and the suppression of click evoked OAEs by 1kHz tones. Plots G-I show the equivalent plots for the suppression of 1kHz tone evoked OAEs by white noise, and plots J-L show the relationships for the suppression of 1kHz tone evoked OAEs by 1kHz tones.
Figure 16-1: Scatterplots of the relationship between 4 measures of TOAE suppression and the 3dB bandwidth of auditory filters measured during contralateral NBN (for details see text)
Appendix 5
17. Appendix 5: Published work

Publications resulting from this work:


Copies of these papers are included in this appendix.

Presentations at conferences:

1. British Society of Audiology’s short papers meeting on Experimental Studies of Hearing and Deafness.


2. 3rd European Conference on Audiology, Prague (1997)- “Possible role of the auditory efferent system in frequency selectivity”

   “Involvement of the auditory efferent system in frequency selectivity”
As early as 1893, it was known that neural efferent pathways relayed information from the brain to the cochlear. The importance of these pathways lies in the fact that responses to auditory signals can be modified before they reach the brain, although the precise functional roles remain unclear.

The anatomy of the pathway was described in greater detail by Rasmussen in 1946. The efferent input to the cochlear consists of the olivocochlear bundle which passes from the superior olivary complex structures on both sides of the brain. The contralateral fibres (crossed olivocochlear bundle) cross over the dorsal surface of the brainstem below the floor of the fourth ventricle. The crossed and the uncrossed (ipsilateral) fibres then join together. Before leaving the brainstem, some fibres end in the cochlear nuclei and the rest travel along the vestibular nerve until they transfer into the auditory nerve and enter the cochlear (Figure 1).

By using transport techniques, involving radioactively labelled amino acids, two populations of olivocochlear neurons have been found.

1. The lateral olivocochlear neurons (54% of the total number of olivocochlear fibres) are mainly (approximately 90%) ipsilateral and synapse with cochlear afferent neuron dendrites close to the inner hair cells. They are thin, unmyelinated and originate from the lateral superior olivary complex.

2. The medial olivocochlear neurons (40% of the total population) project mainly contralaterally (80%) from the region around the medial nuclei of the superior olivary complex and terminate beneath the outer hair cells of the organ of Corti. These are large, myelinated fibres.

Most efferent units were found, in the cat, to have binaural inputs. This structure suggests that it may be possible for the efferent system to exert control over both the hair cell and the transmission of the impulse to the afferent nerve. Moreover, it would seem likely that these two very distinct populations of fibres would have different roles to play in the hearing process.

This paper will deal firstly with studies examining how the efferent system functions; specifically what stimulation affects it and how. The second part of the paper discusses the work which has dealt with finding a function for the efferent system.

Studies of efferent system physiology

Studies to evaluate the function of this extensive pathway of fibres have used electrical and acoustic stimuli to activate the efferent nerves in both animal and human subjects. Due to the huge numbers of papers which have set out to describe the physiology of the efferent system, in this review the work has been classified according to the effects that efferent activation has on different levels of the auditory pathway.

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HOW STIMULATION OF THE EFFERENT SYSTEM AFFECTS THE AFFERENT NERVES AND COCHLEAR POTENTIALS

Early experiments established that the crossed olivocochlear bundle has an inhibitory effect on the eighth nerve action potential. Electrical stimulation of the floor of the fourth ventricle caused a suppression in the action potential (N1), which was maximal for low intensity auditory stimuli. Selective stimulation of the medial olivocochlear fibres also gave these results. These findings have also been replicated using acoustic stimulation in cats and in humans. Kawase and Liberman found both a decrease and an increase in cochlear action potential for tones masked with ipsilateral noise. The effect depended on frequency. Enhancement of the cochlear action potential in the cat was largest for tones from 8 to 16 kHz and suppression was largest for 2 to 8 kHz. Kawase et al. showed that discharge rates to a masked tone could be increased whilst the rates to the masker decreased, by the addition of contralateral sound, indicating a role in signal discrimination.

Looking at the auditory brainstem response, Reid and Thornton found that various levels of masking noise had no effect in human subjects. Lavernhe-Lemaire and Beutter also found no effect of contralateral masking, but found that ipsilateral masking reduced the amplitude of wave I and lengthened the interwave latency I–V. It may be possible that the auditory brainstem response is not sufficiently sensitive to pick up the small changes associated with efferent activity.

Some other cochlear potentials are also affected by activation of the efferent system. An increase in the cochlear microphonic was observed under electrical stimulation in animals and a drop in endocochlear potential was also found. The cochlear microphonic is thought to be produced predominantly by the outer hair cells and the stria vascularis is the presumed generator of the endocochlear potential.

The discharge rate of auditory nerve fibres during efferent stimulation was found to be reduced, and this effect was greatest for fibres responding to their own characteristic frequency, during electrical stimulation and during contralateral acoustical stimulation, demonstrating the frequency specificity of the effect. The decline in the suppression away from the characteristic frequency leads to a widening of the fibre’s frequency tuning curve and therefore a reduction in the Q value. Fibres with low spontaneous rates were also found to show the greatest reduction in discharge rate. Stimulation of the medial olivocochlear fibres was found to reduce the spontaneous activity mostly around 10 kHz and to suppress responses to sounds more at lower frequencies in cats. The latter finding was attributed to the efferents acting on the outer hair cells to damp the motion of the basilar membrane and this effect being apically.

It has recently been found that olivocochlear bundle activation does cause damping of the motion of the basilar membrane, as hypothesized. This was demonstrated by measurement of the cochlear microphonic potential under contralateral acoustic stimulation in the mustached bat. However, the slow contraction of the outer hair cells produced by stimulation of the efferent system was only found to give small deflections of the stereocilia equivalent to a transverse movement of the organ of Corti of less than 1.5 nm. A desensitization of the inner hair cells was also noted.

By sectioning the pathway at different levels in animals, some workers have tried to deduce the effects of the olivocochlear bundle on hearing. A study of a section in the floor of the fourth ventricle ipsilateral to the ear, at the vestibular nerve or at the internal auditory meatus, can eliminate the entire olivocochlear bundle (both crossed and uncrossed components), whereas a midline section will cut only the crossed component. The latter will remove most of the medial olivocochlear fibres whilst sparing most of the lateral ones.

Sectioning of the entire olivocochlear bundle was shown to give threshold and tuning curve changes and to abolish cochlear action potential suppression during contralateral acoustic stimulation. However, Liberman found no change in threshold, tuning curves or rate-level functions in cats after complete sectioning of the cochlear bundle at the floor of the fourth ventricle and Littman et al. found no change in guinea pig tuning curves after section. The variation in results obtained could be due to inter-species differences in physiology and anatomy or from the variety of experimental methods employed, some perhaps causing damage to the olivocochlear bundle. However, many papers indicate that the uncrossed component of the bundle, rather than the crossed component, may be the source of the changes seen, via the action of the outer hair cells.

To summarize, when activated either electrically or acoustically the olivocochlear bundle has the effect of reducing or sometimes increasing the activity in the afferent nerves. The effect seems to be frequently specific and thus the tuning of the cochlear can be altered.

HOW STIMULATION OF THE EFFERENT SYSTEM AFFECTS OTOACoustIC EMISSIONS

Although the above studies have shown that activation of the olivocochlear bundle can alter the responses of the auditory nerve fibres to sound, the precise mechanisms subserving these effects are not yet clear. However, information from the work on otoacoustic emissions provides evidence that the olivocochlear bundle can affect the active processes involved in cochlear mechanics.

Otoacoustic emissions are thought to be produced by active processes occurring in the cochlear, probably involving the outer hair cells. Since the medial olivocochlear neurons synapse at the base of these cells it is possible that the efferent system can directly alter the cochlear mechanics. Thus, otoacoustic emissions have become a useful and convenient tool for studying efferent function.
Electrical stimulation of the crossed olivocochlear bundle in animals produces changes in the distortion product emissions from the cochlear.\textsuperscript{16,31,32} This is important evidence showing that the bundle can in fact alter cochlear mechanics. By changing the movement of the organ of Corti, the mechanical stimulus imparted to the inner hair cells is altered. Thus, this may indicate a mechanism by which the incoming efferent signals can alter afferent responses to sounds.

Contralateral acoustic stimuli have been shown to reduce the amplitude of transient evoked otoacoustic emissions (TEOAEs),\textsuperscript{33} and spontaneous otoacoustic emissions (SOAEs)\textsuperscript{34,35} in humans. This reduction in response implies that the medial olivocochlear neurons alter the activity of the outer hair cells in such a way as to dampen the production of otoacoustic emissions. Distortion product otoacoustic emissions (DPOAEs) seem to show an increase or a decrease in response amplitude in humans.\textsuperscript{36}

The effect of contralateral auditory stimulation on DPOAEs was found to be stronger in the vicinity of spontaneous otoacoustic emissions\textsuperscript{37} which seems to disagree with the theory that spontaneous emissions are produced at an area of low efferent damping activity. Tests on TEOAEs have shown that greater levels of efferent suppression occur with lower levels of ipsilateral stimulation.\textsuperscript{36-38} This has also been shown with DPOAEs as well as finding that mid frequency regions give the greatest suppression.\textsuperscript{39}

Contralateral sound has also been shown to produce a shift in phase of transient emissions\textsuperscript{40} and a shift in frequency of spontaneous emissions.\textsuperscript{34,35} The frequency specificity of the contralaterally evoked suppression was demonstrated, at least for middle frequencies, with both transient\textsuperscript{41,42} and distortion product emissions.\textsuperscript{43}

There is some disagreement concerning the existence of a gender or laterality effect. A smaller suppressive effect was noted in right ears and in females by McFadden\textsuperscript{44} in his review paper, whereas the olivocochlear system was found to be more active on the right side, without any effect of gender, by Khalfa and Collet.\textsuperscript{45} Large intersubject variability has been observed with normals.\textsuperscript{46,47} This variability may be suggestive of inter-subject differences in efferent function rather than due to problems of the measurement technique itself.

The evidence that the above effects are mediated via the efferent system is quite convincing. In animals, Puel and Rebillard\textsuperscript{48} found that the distortion product suppressive effect was eliminated by a midline section of the brainstem involving the crossed olivocochlear bundle and Kujawa \textit{et al.}\textsuperscript{49} inhibited the suppression with strychnine, curare and atropine, antagonists of olivocochlear efferent activity. There have, however, been reports of no change in the distortion products after olivocochlear bundle section\textsuperscript{28} showing that although the efferent system may alter cochlear mechanics when stimulated by, e.g., sound, it may not play a continuous tonic role in cochlear operation.

In patients who had a vestibular neurctomy, involving section of the efferent fibres, no suppression of transient emission was found.\textsuperscript{50,51} Patients with lesions affecting the efferent pathway in the vestibular nerve or at the superior olivary complex have also been shown to have reduced or absent suppression.\textsuperscript{52} A fast reflex type pathway, not involving the higher centres of the brain, is implicated in the suppression of transient emissions, by the onset of contralateral suppression taking around 20 ms to occur.\textsuperscript{53} However, it is possible that this reflex type pathway can be influenced by higher input as discussed below in the section headed 'Attention'.

Some interesting results have come from examining efferent function in certain clinical groups. In subjects with hyperacusis, no decrease in emission amplitude was found,\textsuperscript{54} suggesting an inefficient efferent system failing to dampen responses to auditory signals. A study of a group of patients with noise induced tinnitus showed abnormal suppression of TEOAEs compared to a normal group.\textsuperscript{54} Tests of efferent function may therefore prove to become useful in clinical diagnosis.

The ability of otoacoustic emissions to give a non-invasive insight into cochlear mechanics has meant the popularity of OAEs as a tool for examining the olivocochlear bundle has increased dramatically over the last few years. The work so far has provided important evidence that activation of the olivocochlear bundle does actually affect cochlear mechanics. This has been shown by reductions in TEOAEs and SOAEs and changes to DPOAEs in both animal and human subjects. The effect was also found to be frequency specific.

HOW STIMULATION OF THE EFFERENT SYSTEM AFFECTS THE EFFERENT NERVES

There have been only a few studies on acoustically evoked responses of the efferent fibres in animals. These studies are informative in that they give specific information about the timing of activation of fibres under certain stimulus paradigms. Fex\textsuperscript{15} demonstrated that in response to contralateral sound the efferent fibres, recorded in the vestibulocochlear anastomosis, produced regular spike trains. The fibres were found to have a high degree of frequency selectivity similar to that found in afferent fibres.\textsuperscript{55,56} The latter also established that most of the fibres measured were excited by only one ear and had no spontaneous activity. Binaural units were often found to have spontaneous discharge activity and were normally associated with low frequency regions of the cochlea.

Although direct electrical stimulation of the olivocochlear bundle provides a convenient way of studying the effects on the cochlear of efferent activation, it is not without its problems. First, although it may appear a good method of specifically stimulating the nerve in question, it is hard to rule out spread of the stimulus to other fibres. Second, one must be careful when interpreting results from electrical stimulation with regard to the 'real life' situation of acoustic stimulation.
The stimulus rates and intensities used in some of these studies may have been markedly different from those encountered physiologically. It is also worth noting that the results from work involving acoustical stimulation have the advantage of being more readily comparable between animal and human models. However, some valuable information can still be acquired from the studies using electrical stimulation, and acoustical stimulation results have in general agreed with those findings.

Bringing the work of the previous sections together, a clearer picture of how the efferent system operates can be obtained. In summary, the medial olivocochlear bundle when activated seems to be able to alter afferent responses, via the outer hair cells and their ability to alter cochlear mechanics. There is still little information on the functioning of the lateral olivocochlear system, since many of the above tests involve possible activation of both the medial and lateral neurons. Gifford and Guinan, by stimulating the medial bundle separately, suggested that small effects which had been previously attributed to the lateral neurons could be accounted for by spread of activity to the uncrossed medial fibres. No other effects of the lateral olivocochlear system have been defined.

Tables 1 and 2 summarize the past work on the effect that efferent stimulation has on afferent responses and on otoacoustic emissions.

**POSSIBLE INFLUENCE OF THE ACOUSTIC REFLEX PATHWAY**

Stimulation of the olivocochlear bundle electrically or acoustically could also stimulate the facial nerve fibres innervating the stapedius muscle. Contraction of this muscle would cause attenuation of the signal passing through the middle ear. This may be a confounding factor in studies where recording of efferent function involved transmission of sound through the middle ear, i.e., not those where measurements were taken directly from the efferent nerve fibres. In order to be sure that results observed in the above studies were due to the olivocochlear bundle rather than the acoustic reflex, many of the workers above either cut the tendons, paralysed the muscle or tested subjects with no acoustic reflex.

The suppression of otoacoustic emissions has been demonstrated in subjects with an absent acoustic reflex. It has also been suggested that features of the effect, such as frequency specificity and the ability to be activated by low contralateral intensities, rule out the possibility of the effect being caused by the middle ear muscles alone.

**Studies on the role of the efferent system in hearing**

So far the work discussed has been concerned with elucidating the physiology of the auditory efferent system. Although some speculation of possible roles in hearing can be made from this information, the following studies have examined the validity of certain hypotheses concerning efferent function.

**PROTECTION**

Past work has suggested that the efferent system could play a role in protecting the cochlear against acoustic injury. By exposing guinea pigs to loud tones Cody and Johnstone found that contralateral acoustic stimulation of the same frequency could reduce the temporary threshold shift encountered. This was not due to acoustic reflex pathway activation since a muscle relaxant had been used. Further evidence that the effect was efferent mediated came from the elimination of the reduction in temporary threshold shift after the administration of efferent activity blocking strychnine. Electrical stimulation of the olivocochlear bundle was found to reduce the threshold shift only for levels of acoustic stimulation between 110-130 dBSPL, and for high frequencies (8 and 10 kHz) and short durations (1 and 2 min.). Puel and Vassout found that the cochlear action potential was changed more after loud tone bursts at a rate of 1/s than at 17/s. This was attributed to full efficiency of the protective function at 17/s stimulation, whereas at the slower rate there was time for the cochlear to return to its original state between each tone burst. Cutting the olivocochlear bundle at the level of the fourth ventricle blocked the protective effect providing more evidence that the medial olivocochlear fibres play a role in protection. Contradictory evidence however, came from Liberman who failed to identify any difference in the hearing of cats exposed to loud sounds either with or without section of the olivocochlear bundle. Rajan in his review of the protective mechanism, noted how there may be a 'memory component' to the protective mechanism, which is activated by afferent input. Stimulation of the brainstem, which would normally have caused a reduction in temporary threshold shift for some time, did not produce this effect after sectioning of the crossed olivocochlear fibres. He suggested that the protective effect may operate via a lower brainstem reflex arc, which may be influenced by activity from higher centres, and more recently, Rajan noted that binaural exposure gave greater olivocochlear bundle protection than monaural exposure.

There have been some studies in humans of possible efferent induced protection. Scharf et al. measured the temporary threshold shift in one subject who had undergone vestibular neurectomy. After presentation of a 1 kHz tone at 90 dBSPL for 15 min only slightly less threshold shift was present in the operated ear than in the unoperated one. Otoacoustic emission suppression, by contralateral sound, was compared in subjects with noise induced hearing loss and those with other forms of sensori-neural hearing loss by Collet et al. No difference in the efferent induced suppression was found and no correlation was found between temporary threshold shift and emission suppression in the group with noise induced loss group.
Table 1. Effect that efferent pathway stimulation has on afferent responses

<table>
<thead>
<tr>
<th>Study</th>
<th>Result</th>
</tr>
</thead>
<tbody>
<tr>
<td>Electrical stimulation</td>
<td></td>
</tr>
<tr>
<td>Galambos (1956)</td>
<td>Reduction of N1 in cats</td>
</tr>
<tr>
<td>Desmedt (1962)</td>
<td>Increase in CM in cats and guinea pigs</td>
</tr>
<tr>
<td>Fex (1962)</td>
<td>Reduction in endocochlear potential in cats</td>
</tr>
<tr>
<td>Mountain (1980)</td>
<td>Desensitization of inner hair cells in guinea pigs</td>
</tr>
<tr>
<td>Fex (1967)</td>
<td>Maximum reduction in response for fibres at CF in cats</td>
</tr>
<tr>
<td>Wiederhold (1970)</td>
<td>Maximum reduction in spontaneous rate at 10 kHz</td>
</tr>
<tr>
<td>Guinan and Gifford (1988a)</td>
<td>Greater reduction in response at lower CFs in cats</td>
</tr>
<tr>
<td>Guinan and Gifford (1988b)</td>
<td></td>
</tr>
<tr>
<td>Patuzzi and Rajan (1990)</td>
<td>Small deflections of stereocilia in guinea pigs</td>
</tr>
<tr>
<td>Contralateral sound stimulation</td>
<td></td>
</tr>
<tr>
<td>Buno (1978)</td>
<td>Reduction of nerve activity in cats</td>
</tr>
<tr>
<td>Folsom and Owsley (1987)</td>
<td>Greatest reduction in response at CF in cats</td>
</tr>
<tr>
<td>Kawase and Liberman (1993)</td>
<td>Suppression of CAP greatest at 2–8 kHz</td>
</tr>
<tr>
<td>Kawase et al. (1993)</td>
<td>Discharge rate to masker decreased whilst rate increased to tone in cats</td>
</tr>
<tr>
<td>Bonfils et al. (1986a)</td>
<td>OCB section caused abolition of CAP suppression plus threshold and tuning curve changes in guinea pigs</td>
</tr>
<tr>
<td>Liberman (1989)</td>
<td>Abolition of CAP suppression after OCB section in cats</td>
</tr>
<tr>
<td>Liberman (1990)</td>
<td>No change in threshold, tuning curves or rate level function with OCB section in cats</td>
</tr>
<tr>
<td>Littman et al. (1992)</td>
<td>No change in tuning curves with OCB section in guinea pigs</td>
</tr>
<tr>
<td>Warren and Liberman (1989b)</td>
<td>Suppression of nerve activity in cats, changes in tuning curves in guinea pigs and changes in CAP amplitude in cats, due mostly to uncrossed portion of OCB</td>
</tr>
<tr>
<td>Bonfils et al. (1986a,b)</td>
<td></td>
</tr>
<tr>
<td>Kawase and Liberman (1993)</td>
<td></td>
</tr>
<tr>
<td>Reid and Thornton (1983)</td>
<td>No effect on contralateral ABR in humans</td>
</tr>
<tr>
<td>Laverne-Lemaire and Bautter (1991)</td>
<td></td>
</tr>
</tbody>
</table>


However, there was no control group with which to compare the results in the latter part of this study and thus no conclusion about the involvement of the medial olivocochlear neurons in protection could be drawn. The suppression of transient emissions was also measured by Veuillet et al.\(^6\) and it was noted that at frequencies around 4 kHz no suppression occurred. A link between this and the vulnerability of the cochlear in this region was suggested.

The lack of information on human subjects relates to the difficulties in testing quantifiable noise damage. One can conclude, however, that it is possible that the efferents may play their part in protection by altering cochlear mechanics via the outer hair cells, thus reducing the travelling wave magnitude. Ideally, subjects should be tested prior to noise damage and efferent function related to cochlear damage after a period of noise exposure. Defining the degree of the efferent involvement in hearing protection may prove to be important in industrial noise exposure.

**IMPROVED SIGNAL DETECTION AND DISCRIMINATION**

It is possible that the efferent system could help in signal discrimination in noise by suppressing responses to the background noise, therefore improving the signal to noise ratio. This theory is supported by the work of Winslow and Sachs\(^67\) who concluded that stimulation of the olivocochlear bundle caused a reduction in the response of single auditory fibres to masking noise. Early behavioural studies on animals showed that sectioning of the bundle caused a reduction in a trained monkey's ability to discriminate between vowel sounds in
Table 2. Effect that efferent pathway stimulation has on otoacoustic emissions

<table>
<thead>
<tr>
<th>Study</th>
<th>Result</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Electrical stimulation</strong></td>
<td></td>
</tr>
<tr>
<td>Mountain (1980)</td>
<td>Changes in DPOAEs in guinea pigs, chinchillas and cats</td>
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<tr>
<td>Siegel and Kim (1982)</td>
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<td>Guinan (1986)</td>
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<td><strong>Contralateral sound stimulation</strong></td>
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<tr>
<td>Collet et al. (1990)</td>
<td>TEOAE amplitude reduction*</td>
</tr>
<tr>
<td>Mott et al. (1989)</td>
<td>SOAE amplitude reduction and shift in frequency*</td>
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<td>Moulin et al. (1992)</td>
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<tr>
<td>Moulin et al. (1992)</td>
<td>DPOAE amplitude reduction*</td>
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<tr>
<td>Moulin et al. (1993)</td>
<td>Maximum change to DPOAEs with low ipsilateral stimulation level and at mid frequencies*</td>
</tr>
<tr>
<td>Ryan et al. (1991)</td>
<td>Shift in phase of TEOAEs*</td>
</tr>
<tr>
<td>Veuillet et al. (1991)</td>
<td>Frequency specificity demonstrated*</td>
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<td>Rossi et al. (1993)</td>
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<td>Chéry-Croze et al. (1993)</td>
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<tr>
<td>Puel and Rebillard (1990)</td>
<td>COCB section eliminated DPOAE suppression in guinea pigs</td>
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<tr>
<td>Kujawa et al. (1993)</td>
<td>Efferent antagonists inhibited suppression in guinea pigs</td>
</tr>
<tr>
<td>Littman et al. (1992)</td>
<td>No change after OCB section in guinea pigs</td>
</tr>
<tr>
<td>Williams et al. (1993, 1994)</td>
<td>No suppression after efferent section*</td>
</tr>
</tbody>
</table>


noise and between different frequencies. However, other studies on cats have revealed that surgical section of the crossed olivocochlear bundle causes no deterioration in either detection of single tones in noise, frequency discrimination or intensity discrimination. The lack of change may be due to the fact that the uncrossed olivocochlear bundle was not sectioned and, as mentioned earlier, some workers consider the uncrossed component to be responsible for many of the effects reported experimentally. More recently, Kawase et al. found that a contralateral acoustic stimulus could increase the afferent neuron discharge rates to a tone in a masker while the rate of activity to the masker decreased, indicating a role in signal discrimination in noise. Electrical stimulation of the crossed olivocochlear bundle of a cat was found to affect intensity discrimination in noise. In this study, the efferent system was thought to enhance intensity discrimination by restoring dynamic range and sensitivity to the cochlear. When the olivocochlear bundle was sectioned at the floor of the fourth ventricle it was found that intensity discrimination in background noise deteriorated only at mid frequencies (8 kHz) and not at 1 kHz.

With human subjects, a correlation has been observed between detection thresholds to simple and complex multi-tones in noise, and otoacoustic emission suppression. These results from normal subjects indicate a role for the olivocochlear bundle in detection of tones in noise, but studies of patients whose bundle is cut after vestibular neurectomy, have found no deterioration in detection. The latter study also found no deterioration in frequency selectivity, loudness adaptation, and intensity and frequency discrimination. Nonetheless, a decline in the function of the efferent system with age was found using the test of otoacoustic emission suppression, which may explain the common age related complaint of difficulty in hearing against background noise. A study comparing groups of musicians and non-musicians found that efferent suppression of emissions was higher in the musicians group. This interesting result raises the possibility that "auditory training" could alter physiological mechanisms, thus improving our hearing.

LATERALIZATION

The literature is inconclusive regarding the efferent system and sound lateralization, as there have been few studies with varying results. Early work indicated that subjects, post vestibular neurectomy, had deficits in their ability to lateralize sounds. However, changes in the hearing sensitivity may have accounted for this. More recently, Scharf et al. found no defects in a subject's ability to judge the position of a sound after vestibular neurectomy.

ATTENTION

The effect of attention on hearing and its link with the efferent system has been studied by a number of workers. The earlier work on animals by Glen and Oatman examined the effect
of a visual task on brainstem response in the case. The amplitude of the auditory brainstem response was found to decrease during the task and the latency of N1 increased. Oatman and Anderson\(^6\) also found a decrease in response amplitude, with mid-frequency tones suppressed more, and detected no change in the cochlear microphonic.

In more recent work with humans, Brix\(^44\) tested the influence of selective auditory attention on the auditory brainstem response of 100 subjects, and found a decrease in the I–V interval. However, the length of the ABR test causes problems with regard to attention span, so with the advent of oto-acoustic emission testing this has made it easier to measure the subtle changes involved in a shorter length of time. During a visual task, a decrease in the amplitude of the dominant frequency of evoked emissions was measured,\(^8\) but no change was found in distortion product or stimulus frequency emissions.\(^6\) The latter two studies however, involved comparing the attentional task with a passive task which may have confounded the attentional effects with general alertness. Froehlich \(et al.\) found a decrease in the amplitude of evoked emissions which was maximum at approximately 1–2 kHz for a visual task and at 2–3 kHz for an auditory task, suggesting that attention in different modalities acts on different parts of the cochlear. Using a test where the direction of auditory attention was changed, tone evoked emissions were found to be greater at the target frequency when attention was directed towards that ear.\(^6\) However, there have been other tone evoked emission studies demonstrating no influence of the efferent system in selective attention.\(^8\) Scharf \(et al.\) found that after numerous tests on patients after vestibular neurectomy there was significant change in the detection of unexpected signals in noise. This was attributed to an impairment in selective attention, and suggested that the efferent system was the cause of the change.

It thus seems to be the case that input from higher centres can affect the level of efferent action on the cochlear. This may prove useful when wishing to concentrate on a particular signal in a particular ear. It is therefore clearly important to control the attentional state of the subject when testing the efferent system.

**Synchronization**

Berlin \(et al.\) suggested that the efferent system could be involved in synchronization of the afferent activity, after testing a number of subjects with normal thresholds at 2 kHz, robust otoacoustic emissions but absent ABR and acoustic reflexes. The authors thought, however, that it was more likely that these findings were due to a deficit in afferent function.

**Conclusions**

Although the effects of activation of the olivocochlear bundle have been studied in detail in both animals and humans, the role that the medial bundle, and especially the lateral system play in hearing, have still to be clarified.

Past studies have demonstrated that activation of the efferent neurons leads to a reduction in N1 response, an increase in the cochlear microphonic, a decrease in the endocochlear potential, desensitization of the inner hair cells and changes in the cochlear mechanics shown by studies of otoacoustic emissions. The actions of the efferent nerve were found to be frequency specific. With regard to the role that this system plays in the hearing process, there is evidence that it may be involved in protection of the ear against damagingly loud sounds and the detection of signals in noise. There is also evidence that the efferent system may facilitate a method by which the higher centres of the brain can affect incoming signals at the level of the cochlear, for example in situations where attention is needed to a particular input. The use of otoacoustic emission is very valuable in these situations.

Future work should be directed at clarifying the function of the lateral olivocochlear system. It seems unlikely that such a system would have no influence on hearing. Psychoacoustical and behavioural studies should shed some more light on the nature of higher effects on efferent activation and on possible roles in loudness and pitch perception. Through further analysis of the subtle changes in auditory nerve response, cochlear mechanics and human behavioural studies, the functional role of this system may be elucidated.

**References**


Auditory efferent effects and functional relevance

cochlear bundle in the cat. I. Pure-tone threshold and perceptual signal-to-noise ratio. *Acta Otolaryngol.* (Stockh.) 73, 455-466


Latency of Contralateral Sound-evoked Auditory Efferent Suppression of Otoacoustic Emissions

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The suppression of transiently evoked otoacoustic emissions by contralateral sound stimulation is thought to occur as a result of the action of the efferent pathway from the superior olivary complex to the cochlea via the medial olivo-cochlear neurons. The purpose of this study was to determine the time taken for this pathway to activate the suppressive mechanism in response to contralateral sound in normal human subjects. The time for onset of suppression was found to be between 7 and 20 ms. Key words: activation time, auditory efferent suppression, latency, otoacoustic emissions.

INTRODUCTION

There are two types of efferent fibres which provide an input to the cochlea. The lateral efferent fibres are thin, unmyelinated and originate from the lateral superior olivary complex. They run mainly ipsilaterally and synapse with the cochlear afferent neuron dendrites close to the inner hair cells. The medial efferent fibres, which are large and myelinated, originate in the medial nuclei of the superior olivary complex. They project mainly contralaterally, crossing the floor of the fourth ventricle, before they synapse at the base of the outer hair cells of the organ of Corti. There are, however, some efferent units which respond more strongly to contralateral stimulation (1).

The medial olivocochlear neurons therefore provide a pathway which may facilitate interdependence of the cochleae. Studies of the medial efferent pathway in animals have largely been conducted using electrical stimulation of the crossed olivocochlear bundle (COCB) in the floor of the fourth ventricle, which has shown a reduction in the action potential of the eighth nerve, in response to an acoustic stimulus (2, 3). Acoustic stimulation of the contralateral cochlea has also been shown to reduce responses of afferent fibres in animals (4, 5) and in humans (6, 7).

Otoacoustic emissions are thought to arise from the action of the outer hair cells upon which the fibres of the medial olivocochlear bundle (MOCB) synapse, thereby allowing a means of investigating the efferent pathway. Several workers, (e.g. 8, 9) have shown that transiently evoked otoacoustic emissions can be suppressed in humans by contralateral acoustic stimulation. This suppression is considered, at least partly, to be mediated by the medial efferent system, although the middle ear reflexes may possibly be a confounding factor. However, the presence of suppression in subjects without middle ear reflexes (9), and its abolition by MOCB section during vestibular neurectomy (10), make it less likely that middle ear reflexes play a major part.

The role that the efferent system plays in hearing is still unknown and the timing of the delay between one ear receiving a sound and the onset of suppression of otoacoustic emissions (OAE) in the contralateral ear has received scant attention. Information about the latency of the efferent effect on the cochlea could provide an insight into the neural pathways and the possible role of higher cortical centres in this phenomenon.

Some indication of the timing involved may be ascertained from earlier work, assessing (a) onset time and (b) overall timescale of response.

Onset time

Afferent response measurement. The suppression of the N1 response was shown to occur between 20 and 40 ms after the start of electrical stimulation at the floor of the medulla of a cat (2). The latency of inhibition of discharge rate of single afferent fibres after electrical shocks to the COCB was found to increase with an increase in characteristic frequency (11).

Efferent response measurement. The latencies of activation of different fibres in the cat COCB ranged from 5 to 40 ms when stimulated with short tone pips (12). Cody and Johnstone (13) recorded from the efferent fibres in Rosenthal's canal in the guinea pig and found minimum latencies to range between 7 and 35 ms for different fibres, whilst a minimum latency of 10 ms was found when investigating the olivocochlear bundle (OCB) response to tone bursts in cats (14). This latency increased as the sound intensity approached threshold. Gummer et al. (15) measured efferent nerve responses to amplitude modulated
tones in the guinea pig. Recordings were taken at the basal turn of the cochlea, making no distinction between crossed and uncrossed efferents. They found that group phase delays were shorter (8.2 ms) and more tightly distributed than minimum onset latencies (24.2 ms) measured from the same fibres. The group phase delay describes the latency for a change to occur in the steady state (i.e. continuous noise) and the minimum onset latency is comparable to studies above.

Cochlear potential measurement. The latency of the negative potential of the scala media induced by electrical stimulation of the COCB has also been measured. Fex (16) found a range of latencies of 12–40 ms from different experiments in the cat, and concluded that this measurement represented the activity of the outer hair cells. Later, Konishi and Slepian (17) carried out a similar experiment on guinea pigs, finding an average latency of 10 ms.

Otoacoustic emission measurement. Lind (18) measured the latency of contralateral suppression of transient otoacoustic emissions in normal human subjects. The latency range found (40–140 ms) was wider and had a higher maximum value, than those established in the above studies.

Overall timescale of response
Other studies have examined the time taken for the effect to take place and decay. Using electrical stimulation of the COCB, effects on the receptor potentials of the inner hair cells were found to take 50–250 ms to reach maximum and decay (19). Warren and Liberman (5) presented sound to the contralateral ear of cats as the responses of single afferent fibres were recorded and found that the suppression took 250 ms to develop fully and 80 ms to recover. Sectioning of the COCB in the floor of the fourth ventricle made little difference to the suppressive effect and thus it was suggested that the suppression caused by contralateral sound occurred via the uncrossed OCB. It is possible therefore that some of the above results measured from the COCB may prove to be of less value in understanding the role of the efferent system.

Most previous work has been conducted on animals using either electrical or contralateral acoustic stimulation and a variety of response measures. The use of acoustic signals has the advantage of being much more physiological than electrical stimulation. This study examined the timing of the onset of the suppression effect of contralateral noise on click evoked otoacoustic emissions (CEOAE) in humans.

MATERIALS AND METHODS

Subjects
Thirty-five ears of 25 subjects (8 male and 17 female, mean age 26.1 ± 5.3 years) were tested, although many subjects participated more than once for different tests. All subjects had normal pure-tone thresholds (better than 20 dBHL at octave frequencies between 0.25 and 8 kHz) and ipsilateral and contralateral stapedial reflex thresholds (20). Subjects were also tested to ensure the suppression of CEOAEs in the presence of continuous 50 dBSL contralateral white noise.

Apparatus and stimulus protocol
The Otodynamics ILO92 twin probe analyser provided all auditory stimuli, and was used to record and analyse the resulting responses. The tests were conducted in a soundproof room.

The stimulus in the test ear consisted of linear clicks of 80 µs duration, presented at a rate of 50 Hz and at a peak equivalent level of 63 ± 3 dBSPL. The click intensity was reduced to this level to eliminate the “ringing”, which arises at higher intensities with linear clicks. This also allowed the complete time window of 2.5–20 ms to be studied without the need for cancellation of the early part of the response.

Acoustic cross-talk and acoustic reflexes are factors that need to be eliminated before the suppression effect can be attributed to action of the efferent pathways on the cochlea. Evidence from past work on unilaterally deaf subjects (8) suggests that acoustic cross-talk may not be a factor of importance, as suppression is not observed in these subjects. The stapedial reflex is also unlikely to be solely responsible for the effects seen, as emission suppression has been demonstrated in subjects without the stapedial reflex (9) and the frequency specificity of the suppression effect (21) is not characteristic of the action of the middle ear. However, in order to minimize interaural transmission and activation of the stapedial reflex, which normally occurs above 70 dBHL, the contralateral stimulus was presented at 50 dBSL. The stimulus consisted of a 5 ms burst of white noise.

The ILO92 produced clicks in groups of four. The data acquisition part of the programme was modified to allow each of the four clicks to be averaged and analysed separately. The experiment was conducted using five different time delays of the noise burst in one ear with respect to the first click of each group of four in the other ear, thus allowing emission measurements to be made at various intervals after the onset of the noise burst. Fig. 1 shows the five different timings used. The delays in time (D) between the first click and the noise burst were 14, 13, 8, 3 and 0 ms.
for tests 1 to 5 respectively. Tests 1 and 2 were carried out initially because an onset effect early, in the first 10 ms, was expected. Tests 3, 4 and 5 were then performed to give a more complete view of the process. In tests 1 to 4, the delay of the contralateral noise burst meant that the first of each set of four clicks occurred before the noise. These results were included and referred to with negative times (−14, −13, −8 and −3 ms) to differentiate them from those clicks that occurred at or after the onset of the contralateral noise burst.

The short duration of the noise burst was chosen so that each click was exposed to the same duration of noise. A longer noise burst would have meant that the clicks occurring later in the sequence would have been influenced by a longer sound than those occurring earlier. This allowed measurement of OAEs from clicks very soon after the noise burst and thus meant that even very short onset times could be assessed. The short length also allowed greater flexibility in the relative positioning of the noise burst in the analysis period of 20 ms.

In order to allow for a complete recovery of the suppression to avoid any summating effect, a gap was inserted between each group of four clicks. Since Brown and Nuttall (19) found that 50–250 ms was required for the OCB effect on inner hair cell receptor potential to reach maximum and decay, and Warren and Liberman (5) established that afferent fibre suppression took 250 ms to develop and 80 ms to recover, a gap of 500 ms was considered appropriate. There is some evidence that efferent activity can last for several minutes after stimulation (1). However, these long-lasting effects occur after continuous stimulation lasting many minutes and at much higher levels than used in this study. As each of the four clicks in a group were separated by 20 ms, an inter-group delay of 420 ms was inserted in order to make a total delay of 500 ms.

Procedure

Continuous contralateral noise suppression (CCNS) test. First, a basic test of efferent suppression with continuous masking noise was performed using the IL092. Sets of 60 sweeps were recorded alternately with and without contralateral white noise. The intensities used were the same as in the main experiment (i.e. 63 ± 3 dBSPL linear clicks and 50 dBSL white noise). Five sets of 60 sweeps for each condition (noise/no noise) were recorded and averaged.
averaged. Suppression was calculated by subtracting the total response energy (dB) with contralateral noise from the no noise condition.

**Test of efferent latency (EL).** In the main study the intensities used were as above. The threshold of the noise bursts was established for each subject in order to set a level of 50 dB above sensation level. Eight runs were recorded, consisting of 260 sets of clicks each. The four runs with contralateral noise and four runs without noise were alternated. The initial run was randomly chosen.

**Analysis**

Data were recorded with a time window of 2.5 to 20 ms after each click and the spectrum of frequencies analysed lay between 0 and 6 kHz. For every subject the data from each group of four runs of 260 sweeps were summed to obtain one set of data with a total of 1040 sweeps. This was carried out for both the with contralateral noise burst (N) and the without noise (Q, quiet) conditions. So, for every ear and each test there were four N response values (N1–N4) and four Q response values (Q1–Q4), i.e. one for each click.

The four Q readings were checked for any ipsilateral suppression effects in test 2 of the main experiment (i.e. the four click readings recorded without contralateral noise, to which the 13 ms delayed noise would be compared). The Q1–Q4 values were averaged separately over the ears examined.

No significant differences between any of the mean Q values were found and therefore the four Q values were considered to be equivalent and were averaged to obtain a mean Q value (Qav). The difference between Qav and each N value represented the suppression for each click. This was given by the expression

\[ S_x = Qav - N_x \]

where \( N_x \) is the response value for click \( x \) with contralateral noise and \( S_x \) is the suppression (dB) for that click.

The suppression data were checked to make sure they were distributed normally and then tested with the one sample \( t \)-test to determine if they were significantly different from zero.

Increased levels of suppression of the order of 3–4 dB have been shown to be present by restricting analysis to the time period after 8 ms (22). Accordingly, in this study the response was analysed between 10 and 20 ms with a 2.5 ms rise time (windowed data). The same statistical tests as above were then applied.

The onset latency of efferent induced suppression of OAEs could therefore be examined by comparing the suppression encountered at different times.

Finally, in order to investigate the variability encountered in the results the data were analysed further:

(i) The possibility of a change in the probe fit over the duration of the test was examined. The total response data at 27 ms for each run of the no contralateral noise situation were averaged over the different subjects. These four “no noise” populations would be equivalent if there was not significant change in the probe fit. The two sample \( t \)-test was used to confirm this.

(ii) To compare the initial suppression test using continuous contralateral masking noise with the suppression measured from the main part of the experiment, correlation coefficients were calculated. The data sets compared to the initial test were those at 26 and 27 ms since suppression was present at these times.

**RESULTS**

All subjects had normal (better than 20 dBHL between 0.25 and 8 kHz) pure-tone thresholds, normal ipsilateral and contralateral stapedial muscle reflex thresholds and greater than 1 dB of suppression of CEOAEs with the preliminary test in the presence of continuous contralateral white noise.

The total response (non-windowed) data are presented in Table I and the windowed data in Table II, for times from 0 to 60 ms after the onset of the noise burst and −14, −13, −8 and −3 ms before the noise bursts. The suppression mean and standard deviation of the population for each timing are shown, as are the number of ears tested in each case. The suppression values ranged from −0.05 to 0.24 and −0.19 to 0.62 dB for the total response and the windowed data respectively. The data distribution was judged to be normal, permitting the use of the one sample \( t \)-test, which gave the significance values shown in Tables I and II. Mean suppression, which was significantly greater than zero (i.e. \( p < 0.05 \)) occurred at 20, 26, 27, 46 and 47 ms after the noise burst and also at 52 ms for the windowed data.

The windowed response shows the suppression much more strongly than the total response. This is shown in Fig. 2, where the windowed and the non-windowed data are compared for test 1. The suppression is statistically significant for both the windowed and the total response data at 26 and 46 ms and not at −14 and 6 ms. The mean windowed values for tests 1, 2 and 5 are shown in Fig. 3a, b and c, respectively. The different tests are not directly comparable, because they are measured from different subject populations, and therefore not plotted on the
Table I. **Mean total response suppression data at various click times**

<table>
<thead>
<tr>
<th>Time between onset of noise and click (ms)</th>
<th>No. of ears tested</th>
<th>Mean suppression (dB)</th>
<th>Standard deviation of suppression</th>
<th>t-test significance level</th>
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<td>0.40</td>
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* Significant at \( p < 0.05 \).

same graph. The graphs show the increase in mean suppression between 6 and 26 ms, 7 and 27 ms, and 0 and 20 ms. The onset latency of the efferent suppression of OAEs thus appears to lie between 7 and 20 ms. Tests 3 and 4 showed much lower levels of suppression in comparison to the other tests, as shown in Fig. 4 for the windowed data.

Investigation of the change in the probe fit with time gave a two sample \( t \)-test significance level of 0.901 when comparing the first and the last run.

Table II. **Mean windowed suppression data at various click times**

<table>
<thead>
<tr>
<th>Time between onset of noise and click (ms)</th>
<th>No. of ears tested</th>
<th>Mean suppression (dB)</th>
<th>Standard deviation of suppression</th>
<th>( t )-test significance level</th>
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* Significant at \( p < 0.05 \).
Fig. 2. Comparison of suppression values for the total response data and the windowed data (10-20 ms). Data shown are for each of the four clicks of test 1. The windowed data show higher amplitudes of suppression than the non-windowed data. The standard error is also shown for each set of data.

These runs were compared because they would have the greatest difference if the probe had become loose with time.

The suppression at 26 and 27 ms from the total response data of the EL test was compared with that achieved with the CCNS test using continuous noise on the same subjects. The correlation between the two sets of data was found to be 0.507 for 27 ms and -0.364 for 26 ms with significance values of 0.246 and 0.271 respectively, showing that they were not correlated.

To check for the first clicks suppressing the later clicks (ipsilateral suppression), the Q1–Q4 values were examined for test 2. The average values ranged from 7.68 to 7.85 dB SPL. There was no significant difference between the values and therefore no evidence of ipsilateral suppression was found.

DISCUSSION

Onset time

The principal finding to emerge from this study is that the contralateral sound activated suppression of CEOAEs occurs at or before 20 ms. This result is in line with previous estimates of suppression onset latency observed in animals. The time taken for acoustic stimulation to activate efferent fibres was found by Fex (12) to be 5–40 ms, by Cody and Johnstone (13) to be 7–35 ms, by Liberman and Brown (14) to be 10 ms or greater, and Gummer et al. (15) measured a mean value of 24.2 ± 12.5 ms.

Galambos (2) found the time for efferent stimulation to produce afferent effects to be 20–40 ms. The time for electrical stimulation of the efferent nerves to create changes in the cochlear potentials was found by Fex (16) to be 12–40 ms and by Konishi and Slepian (17) to be 10 ms.

A possible mechanism for the process that we have measured starts with the stimulation of the contralateral ear and activation of the afferent system via hair cell receptors. The efferent system is then activated, stimulating the outer hair cells and thus causing a change in the otoacoustic emission. Therefore, the latency measured in this experiment may be expected to be approximately the sum of the time taken when stimulating with contralateral acoustic stimulation
and recording the changes in efferent nerves (12–14), and the time taken when stimulating the efferent nerve directly and recording from the afferent nerve (2) or the cochlea (16, 17). The range of possible onset times that we measured does conform to the above hypothesis. However, it should be borne in mind that the experimental design in this study did not include a final afferent pathway, as in the study by Galambos (2), and thus the latency measured might be expected to be shorter, although there is the additional time for the emission to be emitted from the cochlea before being measured in the canal (<10 ms, since the emission returns and is recorded within 20 ms of the stimulus onset).

The most marked OAE suppression occurred at 20, 26, 27, 46, 47 and 52 ms. All the suppression values are small but consideration of the windowed data between 10 and 20 ms has the benefit of revealing the suppression to a greater degree (Fig. 2). Using this time window also has the advantage that there is much less chance of stimulus artifact affecting the results.

Owing to the lack of suppression at 32 and 37 ms the results at 12 and 17 ms were not considered when looking at the onset time, since they were recorded in the same tests as the values at 32 and 37 ms and may have had a lower magnitude of suppression due to other reasons, discussed later. Therefore the suppression appears to start at some time after 7 ms but before 20 ms. It is important to note, however, that the range found in this study of 7–20 ms describes the possible time zone within which the suppression starts, whereas the ranges of times quoted in some of the above studies show the extent of the onset latencies measured from different fibres.

In this study the trend of increase in suppression between 7 and 20 ms was found in a large proportion of the ears tested: 76% for test 1, 93% for test 2 and 77% for test 5 for the windowed data. At present it is not clear whether there is a gradual growth of the suppressive effect between 7 and 26 ms or whether it begins at some point between the two times and rises sharply. A gradual growth may occur if different efferent fibres have different latencies (12, 13), especially as the stimulus was white noise in this study and this would be expected to activate many fibres of different characteristic frequencies.

It is possible that the onset of suppression occurs after the initial set of clicks with a time of 507 to 526 ms instead of 7 to 26 ms. However, this appears unlikely, as the longest onset times found in previous work are of the order of 40 ms.

More recently, Lind (18) attempted to measure the onset of suppression in humans using CEOAEs. The contralateral noise was produced by a computer interface and was much longer (80 ms) than that used in this study. Results were gathered from the four clicks separately and two conditions were studied: one with the batch of clicks following the noise and one with the click batch 40 ms after the start of the noise. As with our results the last 10 ms of the emission was found to give larger suppression values. Large variation was found in the onset times obtained, which ranged from 40 to 140 ms. These results show much longer latencies than measured by the workers mentioned previously. It is possible that OAE measurement may give slightly longer latencies than direct measurements from the nerve, but this delay would be in the order of 10 ms and therefore does not fully account for the discrepancies. However, with Lind’s experimental design, 40 ms was the minimum time that could be examined and there were only eight possible measurement times. One must also consider that the 2nd, 3rd and 4th clicks in the train of four would have been affected by progressively longer lengths of the contralateral noise burst and would therefore not show equivalent suppression.

Lack of significant suppression at certain points
At 32, 37 and 40 ms the suppression was not significant and was reduced in comparison with that at 27 and 46 ms (Fig. 4). There are four possible explanations for this: (a) The suppressive effect is only activated when afferent input from the ipsilateral ear is received within a certain critical time interval after the contralateral noise. From the present data this critical time interval would seem to be between 7 and 12 ms. When the first click is 7 ms after the contralat-
eral noise burst, then the suppressive effect is activated and takes time (between 7 and 20 ms after noise burst) to develop. If, however, the first click occurs later (12 ms after the noise burst) then the effect is not activated and the emissions are not suppressed. However, the mean values at 32 and 37 ms do show some, non-significant, suppression. This may be explained by the gradual decline in the suppressive effect after the “critical interval”. If an ipsilateral click is received before the suppression has totally diminished, then some residual suppression of OAEs may be seen. A similar mechanism was suggested (23) for the protective role of the efferent system, although over a much longer time span. In this latter study it was found that stimulation of the floor of the fourth ventricle of guinea pigs produced reduced temporary threshold shift (TTS) in response to loud sounds even when the loud noise was delayed after the stimulation. By sectioning the COCB after stimulation but before the noise, the TTS reduction was prevented, showing that storage of this effect was at a central site. (b) There are two separate mechanisms contributing to OAEs: a “quick” reflex type response and a slower effect involving the influence of higher centres via the efferent system to the outer hair cells. It is possible that the dip in the suppression may represent the point of overlap between the two mechanisms. (c) The gap between the onset of the noise burst and the click actually influences the latency of the suppression. This is supported by the fact that in test 3 the suppression was significant only at the final click for the windowed data, i.e. the latency was longer because the noise to click 2 interval was long (12 ms). The functional basis of such a mechanism is unclear. (d) It is also feasible and perhaps most probable that the lack of suppression at these points represents the variability in the results.

Variability of suppression

The suppression of OAEs observed was small, as indicated earlier, but in comparison the extent of variability across subjects was quite high. In order to find the source of the variation the data were subjected to further analysis.

Probe fit. Because of the long length of the test it was considered that change in the probe fit with time might affect the degree of emission recorded, thereby increasing the variability. The two sample t-test was used to compare the first and last run of the no noise condition and the result showed that there was no significant change in the two readings. The results indicate that the probe probably moved very little between the beginning and the end of the test session. However, the change in the probe fit in the same subject over different test sessions may have led to some variability in the emissions recorded and therefore the suppression levels derived.

Comparison with the continuous contralateral noise suppression (CCNS) test. The suppression values in the main test of efferent latency (EL) were much smaller than those observed with continuous contralateral noise in the first screening test. It is possible that the short duration of the contralateral noise may have had an effect, as it has been shown that with short stimuli there is a reduction of suppression (14). Also, the values would have been expected to be smaller because it is the onset of suppression that is being measured in the EL test as opposed to the maximum value measured in the CCNS test.

The data from the EL test were not found to be correlated with those of the CCNS test at 26 and 27 ms when comparing suppression within the same subjects. The difference in the results found by the two tests could be due to several causes. The suppression measured in the CCNS test is in response to continuous contralateral stimulation and thus maximum suppression will have been reached for a prolonged period of time. In contrast, the EL test looks at the very small changes that occur as the suppression starts to take effect. It is possible to hypothesize from this that different people may have different onset times, which are not related to the absolute value of the suppression finally achieved. Unfortunately, owing to the fact that in the test paradigm used specific points in time were measured rather than continuous monitoring of the suppression, it is not possible to determine whether there is a variability in onset times across individuals.

An alternative explanation may be that the difference is due to a change in alertness between the two tests. During the protracted EL test, some subjects could easily have become less attentive compared to the much shorter CCNS test.

Having discussed this, however, it is noted that the range of suppression values found across subjects in both suppression tests was similar. This is demonstrated by the fact that the standard deviation of the suppression in the CCNS test was 0.697 compared to a mean standard deviation of 0.373 found in the total response suppression data after 20 ms (values from before 20 ms were excluded since suppression did not necessarily occur there). The suppression recorded in the EL test was much smaller than that of the CCNS test and therefore the standard deviation forms a much higher percentage of the result. It was difficult to standardize the attentiveness of the different subjects and this may be a contributing factor to the range of suppression levels seen, since attention has
been shown to affect OAE level (24). It is therefore unclear as to how much of the variability observed is due to inherent differences in each subject's efferent system.

This study has demonstrated a means of estimating the onset time of efferent suppression in humans. The onset was found to be between 7 and 20 ms after the start of a contralateral noise burst. Questions still remain about the source of the variations in the results and whether they can be eliminated or whether they are an inherent part of the suppression process or in fact the testing procedure. Further work is necessary to address these problems along with a more detailed analysis of the onset slope and the confirmation and possible cause of the lack of significant suppression encountered at some of the measurement times.

REFERENCES


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