SOME STUDIES OF SPONTANEOUS AND EVOKED ACOUSTIC EMISSIONS FROM THE HUMAN EAR

GRAHAM JOHN SUTTON

A THESIS SUBMITTED FOR THE DEGREE OF
DOCTOR OF PHILOSOPHY

UNIVERSITY OF KEELE
1987

ABSTRACT

Several studies of spontaneous and evoked oto-acoustic emissions (SOAEs and EOAEs) and their relationship to normal and pathological hearing mechanisms are described.

1. A computer model was developed to study the theory that the EOAE is produced by summation of responses from activity distributed along the cochlea. It was found that, whilst with a regular cochlea and mapping the summed response is small owing to phase cancellation, any irregularity of mapping or structure or sensitivity gives rise to a sharply-tuned response showing many of the characteristics of EOAEs.

2. A search for SOAEs in 17 normal subjects revealed a prevalence of 53% (42% of ears), which was significantly higher among the females.

3. A group of 49 clinical tinnitus sufferers was investigated to test the hypothesis that SOAEs might be responsible in some cases. 3 such cases were found, all with tonal tinnitus and normal hearing at the tinnitus pitch (although 1 had a bilateral loss at other frequencies). The SOAEs in both studies were found between 680 and 9610 Hz at levels up to 24 dB SPL. They were not found at frequencies where hearing threshold was greater than 20 dB, and no consistent association with audiometric abnormalities was noted, implying that they should be seen as part of normal hearing variation, rather than the result of minor pathology.

4. Suppression effects were studied for (a) SOAEs in 2 ears and (b) both click-evoked and continuous-tone evoked OAEs in another ear. A number of features were observed, most notably multiple lobes in many of the suppression tuning curves. The implications of these results for theories of OAE generation are discussed.

LIST OF CONTENTS

1. INTRODUCTION AND REVIEW

A dramatic change has recently occurred in our conception of the cochlea. Whereas it used to be thought of as a purely passive transducer, converting sound into electrical nerve impulses, it is now known that it can also actually emit sounds, demonstrating the presence of energy-releasing active mechanical elements. This emission of sound can be seen in almost all normal ears as a delayed 'echo' response to a click stimulus, but even more dramatically as continuous low-level whistles emerging from a high proportion of ears. The discovery of these *oto-acoustic emissions*, *evoked* (by stimulation) or *spontaneous* (without stimulation) has, together with other recent findings, revolutionised our understanding of how the ear analyses sound.

This thesis is concerned with the prevalence and properties of these oto-acoustic emissions (OAEs) and the light they shed on cochlear processing.

Because it is believed that OAEs are intimately related to the mechanisms responsible for frequency analysis in the cochlea, our current state of knowledge of this subject will first be described briefly. After that the properties of OAEs and theories on their origin are summarised. Then follow the descriptions of the studies, in 4 chapters, and a concluding section.

1.1 FREQUENCY ANALYSIS IN THE COCHLEA

The ability to separate and resolve the different frequency components present in a complex sound is one of the crucial functions of the hearing mechanism, on which depends our ability to distinguish vowels and to understand speech. It has long been known from psychophysical experiments that the ear operates as a bank of overlapping narrowly tuned filters (Fletcher 1940, Zwicker et al 1957). It was at first thought that most of this filtering was carried out at higher levels of the auditory pathway, with the cochlea acting as little more than a microphonic transducer. However over the past 20 years, with the single unit nerve fibre recordings of Kiang et al (1965) and others, it has become clear that it is the *cochlea* which contains the filter bank, and which performs this frequency-filtering task. The culmination of much beautiful and detailed work by many researchers in tracking down the location and mechanisms of these filters within the cochlea has been the discovery (Khanna and Leonard 1982, Sellick et al 1982) that in fact all tuning may well be mechanical, present in the vibration pattern of the cochlear partition; i.e. before transduction to the haircells and nerve fibres. It also appears that a nonlinear, physiologically-vulnerable and mechanically-active process is involved, feeding energy back onto the cochlear partition to amplify and sharpen the mechanical response. Oto-acoustic emissions are believed to be generated by the same process.

Our view of the cochlea has thus changed from a passive linear transducer of sound to an amplifying nonlinear processor with very sharp filtering. Somehow stimulation of the haircells by movement of

the cochlear partition in return affects that motion. The following sections briefly review why this profound change has come about. Fuller reviews may be found in Pickles (1982) and Dallos (1981) (although these are already slightly outdated).

1.1.1 Basic structure of the ear

Sound arriving at the ear is gathered by the outer ear (pinna) and funnelled down the ear canal (or meatus) to the eardrum. The resulting eardrum vibrations are transmitted through the bones of the middle ear (malleus, incus and stapes) to the oval window and thus to the fluids of the inner ear, the cochlea. Here they cause a wave-like displacement in the cochlear partition (or basilar membrane - BM - often used interchangeably in this context), leading to stimulation of the sensory haircells and the generation of nerve impulses which are eventually perceived as sound.

1.1.2 Mechanical tuning and the 'second filter'

The travelling wave vibration pattern on the cochlear partition for a pure tone input shows a single peak whose position changes with frequency - low frequencies peak near the apex, high frequencies near the base (tonotopic organisation). Early observations in cadaver ears (von Békésy 1944, published in collected form 1960), and later in live animals (e.g. Johnstone et al 1970, Wilson and Johnstone 1975) showed rather broad patterns, with each point along the BM acting like a low-pass or a broad band-pass filter, and complete linearity.

On the other hand responses found in single cochlear nerve fibres showed very sharp tuning (Kiang et al 1965, Evans and Wilson 1973) which degenerated into broad tuning on anoxia, insult etc. This gap between broad mechanical tuning and sharp neural tuning led to the hypothesis that the broad mechanical 'first filter' of the BM was followed by a physiologically-vulnerable 'second filter' prior to neural transduction (Evans and Wilson 1973).

However Rhode (1971) and Rhode and Robles (1974) cast doubt on the need for a second filter by reporting sharper mechanical tuning than other workers, as well as nonlinearity in the response (it being sharper at low levels than high); and Russell and Sellick (1978) showed that if a second filter existed it had to be before the input to the inner haircell, so ruling out neural involvement. Finally with the recent reports of Sellick et al (1982), Khanna and Leonard (1982), and Robles et al (1985) the evidence is overwhelming that mechanical vibration of the BM can after all be as sharply tuned as auditory nerve fibres. The lack of sharp tuning seen in earlier results must now be put down to the extreme vulnerabilty of the mechanisms to even subtle manipulation.

With this discovery of sharp mechanical tuning the main reason for the concept of a separate second filter has disappeared. However Dallos (1981) has argued that some extra processing beyond the mechanical level may still be needed to account for the amplitude and phase plateaux above the best frequency seen in mechanical, but not in neural, data. One can also point to data in other animals: for example there is an electrical second filter in turtle haircells (Crawford and

Fettiplace 1981); haircell stereocilia microresonances contribute to tuning in alligator lizard (Holton and Hudspeth 1983), and the existence of similar structure variations make it possible they do so in man also (Strelioff et al 1985). Also the second filter concept can still be useful if it is referred to the physiologically-vulnerable mechanism by which the broadly tuned passive mechanics are sharpened up. The change is that it now must be seen as having its effect 'backwards' on the mechanical vibration, rather than 'forwards' on the nerve fibre input.

1.1.3 Active processes in the cochlea - role and function

How the mechanical responses are sharpened is still a matter of great interest and debate, but it appears to involve an active (energy-releasing) mechanism with positive feedback. Incorporation of such a mechanism in any system has several effects, the most important of which are to increase its frequency selectivity and sensitivity (and thus the dynamic range). Gold (1948) suggested that an active process was needed to account for earlier experimental results (Gold and Pumphrey 1948), but his arguments were based on some questionable assumptions and interpretations (see Green et al 1975, Wilson 1984) and were therefore received with scepticism. Also the problems of maintaining stability in a system with positive feedback made such a mechanism seem implausible. In fact sharp mechanical tuning may not necessarily imply an active process (Diependaal et al 1986), but there is much more direct evidence for this in the discovery of EOAEs (Kemp 1978) and SOAEs (Kemp 1979b). That SOAEs do require an active mechanism and are not just the product of passive amplification of background

noise is confirmed by stochastic analysis (Wit 1986). Even with EOAEs, Kemp and Chum (1980a) calculated that more energy could be produced by the cochlea than was sent in. Thus with OAEs some mechanism is actively responding to stimulation by causing the BM to move.

Clearly this is exactly the type of mechanism that is required to give the mechanical sharpening, and since both sharp mechanical tuning and OAEs display the same physiological vulnerability (§1.1.2 and 1.2.5), it is parsimonious to assume that the same active process underlies both. The former is believed to be its prime function, with EOAEs and SOAEs being epiphenomenal (since it is extremely unlikely that idiosyncratic emission of sound could be functional).

One consequence of an active process is to that our estimates of the BM vibration amplitude at threshold are much greater (by the amount of amplification). Disquiet was often expressed previously at the extreme smallness of estimates extrapolated from high level measurements (e.g. 1/500th diameter of a hydrogen atom in Békésy 1960). However as Davis (1983) points out this does not solve the difficulty since the active mechanism must still be triggered by the amplitude implicit in the passive system.

Various models have been proposed in general terms for the sharpening process (e.g. Zwicker 1979, Kim et al 1980, Davis 1983), using amplifying mechanisms (or equivalently negative damping) to feed energy back into the travelling wave. However the problem as to how stability is normally maintained in the ear with a large amount of positive

feedback still remains. And whilst SOAEs are evidence that instability can develop, these seem not to be the result of purely local feedback, since they are affected by mechanical influences on the middle ear (see §1.2.9).

1.1.4 Site of the active process

It is inner haircells (IHCs) to which the great majority of afferent (ascending, centripetal) nerve fibres are connected (Spoendlin 1978), and which therefore relay the sensory messages. The role of the much more numerous outer haircells (OHCs), which are mainly innervated by the efferent (descending, centrifugal) fibres from the crossed olivo-cochlear bundle (COCB), has long been a mystery. It now appears that it is the OHCs which sharpen the mechanical tuning as well as generate the cochlear microphonic. Thus sectioning the efferent bundle broadens the tuning of the afferent fibres (Carlier and Pujol 1983); electrical stimulation of COCB causes changes in the tuning of cochlear nerve fibres (Kiang et al 1970), and in basilar membrane mechanics and the generation of distortion products detectable acoustically in the ear-canal (Mountain 1980, Siegel and Kim 1982). Hubbard and Mountain (1983) also found that injecting alternating curent into scala media caused acoustical effects in the ear canal in gerbil. This 'reverse functioning' of the cochlea shows that electrical-to-mechanical transducers are present which could affect the BM response. Thus our picture is of the OHCs modifying and sharpening the input to the IHCs.

The mechanism by which the OHCs move the BM is not yet known. The presence of contractile proteins suggest the possibility of motility of

the stereocilia (as proposed by Anderson and Kemp 1979) which are in contact with the tectorial membrane), or of the cell bodies for which there is evidence in isolated OHCs (see Lim 1986 for recent review, also Wilson 1984). Such motility might also be consistent with Wilson's (1980df) hypothesis of slight volume changes of OHCs as they are activated. Davis (1983) suggested that the cochlear microphonic might drive the OHCs into motion by means of a reverse piezo-electric effect.

The efferent system is too slow-acting for it to be directly involved in the generation of oto-acoustic emissions or sharpening, but it may be able to control the overall level of response (Kemp 1981, 1986).

1.2 EVOKED AND SPONTANEOUS OTO-ACOUSTIC EMISSIONS - REVIEW

1.2.1 Discovery

In 1948, Thomas Gold predicted on theoretical grounds that tonal oscillations might be found emerging from some ears, but unfortunately was not able to confirm this using the experimental techniques of the time. Flottorp (1953) also attempted, and failed, to find an objective correlate of his 'idiotone' (an evoked transient tonal tinnitus).

There were however a handful of cases in the clinical literature in which very strong tonal emissions were audible emerging from the ear, either by auscultation or even sometimes to the unaided ear of a nearby observer. These remarkable cases were reported as 'objective tinnitus', a well-known clinical phenomenon usually involving low frequency vascular bruits or clicking due to muscle spasms etc, but not usually high frequency tones. Such tonal emissions were reported by Loebell

(1962), Citron (1969), Kumpf and Hoke (1970), Glanville et al (1971) and Huizing and Spoor (1973). However these cases were seen as isolated pathological oddities, until Kemp (1978), sealing a microphone into the ear canal to make a much more sensitive detection system, reported the discovery of 'cochlear echoes' (or evoked oto-acoustic emissions - EOAEs), detectable in nearly all normal ears. Subsequently Kemp (1979a) also reported the discovery of continuous tonal emissions (spontaneous oto-acoustic emissions - SOAEs) at much lower levels than the earlier cases, in a large proportion of normal ears. In retrospect we can also recognise that some results of Møller (1960) show the influence of £OAE£.

EOAEs have also been detected in monkey (Anderson and Kemp 1979, Wit and Kahmann 1982), chinchilla (Zurek and Clark 1981), guinea pigs (Zwicker and Manley 1981), caiman crocodile (Strack et al 1981), frog (Palmer and Wilson 1982), cat (Wilson and Evans 1983), and most recently starling (Manley et al 1987). SOAEs have also been detected in many species, including chinchilla (Zurek and Clark 1981), guinea pig (Evans et al 1981), dog (Ruggero et al 1982), frog (Palmer and Wilson 1982), and bat (Kossl and Vater 1985), and there are anecdotal reports in a cat and a pony (Evered and Lawrenson p133).

In retrospect some features of the classic reports of Wegel (1931) Flottorp (1953) and Ward (1955), in which observations on tinnitus, monaural diplacusis and aftertones were made, point towards a source in SOAEs, although this can now only be speculation. Running contrary to such speculation, another report on a case of monaural diplacusis (Formby and Gjerdingen 1981) observed some of the same effects

(irregular beating, rapid tone decay, an area where a tone was inaudible) but failed to detect any SOAE near the centre of the diplacusis where there was also a hearing loss of some 50 dB.

1.2.2 Detection methods

EOAEs can be detected in response to clicks, tone bursts, or continuous tones. Fig.1-1 illustrates how. A sensitive microphone is sealed into the ear canal to minimise enclosed volume and thus maximise the sound pressure generated, necessary because the eardrum is an inefficient loudspeaker. The stimulus may be presented to the ear, either via a driver also sealed into the ear canal or via an earphone over the ear. Sound is transmitted through the middle ear bones and sets up a travelling wave ('TW') in the cochlea (here shown 'unrolled'). The EOAE generated in the cochlea causes movements of the stapes in the oval window which are transmitted back through the middle ear and detected by the microphone as pressure variations in the ear canal. The response is usually high-pass filtered to remove muscular and vascular noise.

For transient stimuli the latency of the response allows it to be separated from the stimulus (Fig.1-1a). Many responses are usually averaged to overcome the signal-to-noise problem (Kemp 1978, 1979a), but can also be seen without averaging if a more sensitive microphone is used (Wilson 1980a). For click stimuli the whole of the cochlea is excited and the EOAEs (called click-evoked oto-acoustic emissions, cochlear echoes or Kemp echoes) contain energy spread over a wide range of frequencies. If tone-bursts rather than clicks are used the responses emerging are highly frequency-specific.

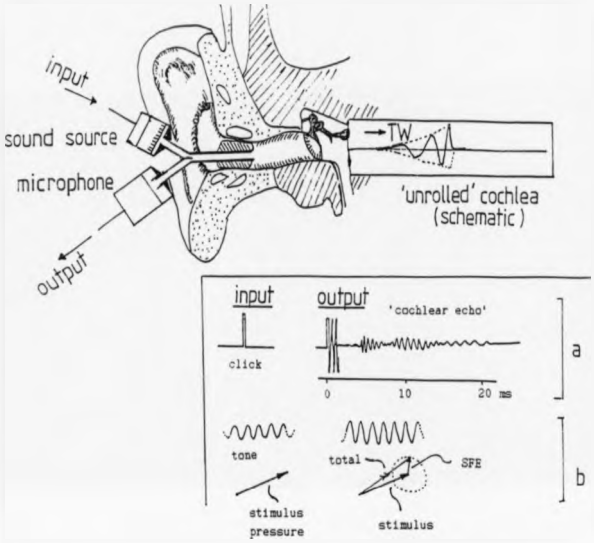

Fig. 1-1 Typical method of recording EOAEs, showing sound source and microphone sealed into the ear canal, and the travelling wave ('TW') in a schematically 'unrolled' cochlea.
(a) EOAE resulting from a click stimulus is the cochlear echo.
(b) EOAE resulting from a continuous tonal stimulation is the synchronous stimulus-frequency emission (SFE), which adds vectorially to the stimulus.

In contrast to these transient responses to transient stimuli, a steady-state tonal stimulus will evoke a steady state emission at the same frequency as the tone and synchronous with it (Kemp and Chum 1980b), known as a stimulus-frequency emission (SFE). This can be observed by its interference with the stimulus, leading to peaks and dips in the total meatal SPL as a function of frequency. Put another way, the SFE pressure adds vectorially to the stimulus pressure (Fig.1-1b), and as frequency is swept the SFE vector rotates rapidly, so that the resultant vector amplitude varies cyclically. In a Nyquist plot (the amplitude-phase locus of meatal SPL vectors - e.g. Palmer and Wilson 1982), lobing is seen. More accurate measurements can be made by extracting the SFE from the stimulus, using techniques based on the nonlinearity of the response (see §5.2).

SOAEs are detected using the same microphones sealed into the ear canal, although of course no stimulus source is needed in this case. Whilst SOAEs are often audible on the amplifier output without processing, some sort of narrow-band filtering is usually used for better separation from the background noise, sensitivity, and accurate measurement (see §3.1 for details).

PROPERTIES OF OAEs

1.2.3 Prevalence

The prevalence of EOAEs in normal human ears is close to 100% (Kemp 1978, Wit and Ritsma 1979, Rutten 1980, Johnsen and Elberling 1982b, Probst et al 1986). SOAEs are found in 25 - 45% of normal ears (Wilson

1980c, Zurek 1981, Schloth 1982, Wier et al 1984, Rabinowitz and Widin 1984, Strickland et al 1985, Probst et al 1986, §3).

1.2.4 Waveforms latencies and phase

Each ear has its own unique EOAE signature which is highly stable over years (Kemp 1978, 1982). The responses to clicks and tone-bursts are observed typically 5-20 ms post-stimulus, after the stimulus artefact and middle ear resonances have decayed, although some 'ring on' longer than this. These latencies are much too long to originate in the highly-damped structures of the middle ear (Kemp 1978). The waveform inverts precisely with stimulus polarity (Anderson 1980) ruling out the possibility of any neural involvement in generation. Responses often appear as short wave-groups of a particular frequency (see for example Wilson 1980c Fig.2). High frequency components emerge before low on average. Latencies at different frequencies are more accurately found from the EOAE phase slopes. Expressing these in terms of number of waves delay removes variation simply due to the frequency band, and gives delays of 10-15 waves over 0.8 to 4 kHz (Wilson 1980c, Wit and Ritsma 1980, Rutten 1980). These latencies greatly exceed estimates of travel time along the BM. The phase curves are characterised by long straight sections (Wilson 1980c, Kemp and Chum 1980ab) each of which is associated with a band of strong response. (It should be noted that in measurement paradigms where no time-windowing is used, 'cochlear resonances' (see §1.2.13) may confuse the picture by introducing phase inflections - e.g. lower level curves in Kemp and Chum 1980b, Fig 3.)

Some of the results initially reported as click-evoked OAEs appear in retrospect to be SOAEs that have been fully or partially synchronised by the stimulus (Kemp 1978, Zwicker 1979, Wit and Ritsma 1980, Wit et al 1981), and this can lead to confusion, particularly if input-output functions are examined (e.g. Zwicker 1979, and see discussion in Ruggero et al 1983).

1.2.5 Frequencies and bandwidths

In the frequency domain for all types of EOAEs, there appear to be regions about a critical band wide where the response is strong, often separated by deep notches (Wilson 1980c, Kemp and Chum 1980ab, Kemp 1982). In some measurement paradigms a fine structure of amplitude peaks and dips spaced at about 50 to 100 Hz can be seen superimposed on this (Kemp and Chum 1980b, Wilson 1980c, Kemp 1980, Zwicker and Schloth 1984): this is believed to be due to multiple internal reflection (see §1.2.13), and should not be taken to represent the underlying strength of the generator.

EOAEs are usually strongest between 1 and 2 kHz; this is thought to be because the middle ear is most efficient in reverse transmission here (see Kemp 1980). However they have been detected at least up to 6.6 kHz in man (Anderson 1980), and up to 9 kHz in one subject below (§4.5.8). Most SOAEs also occur in 1-2 kHz, but have been found up to 15 kHz (Wilson and Sutton 1983).

SOAEs are not absolutely pure tones with zero bandwidth but fluctuate like narrow-band noise processes. Their bandwidths were first resolved

by Kemp (1981) as between 1.2 and 4.7 Hz (3 dB bandwidth), and by Rabinowitz and Widin (1984) between 1.4 and 12 Hz (10 dB bandwidth). The bandwidth is determined by the amount of energy recirculated back to the generator (Kemp 1981). Wit (1986) was able to show that the amplitude probability distribution of an SOAE required an active process and was not consistent with passive filtering of extraneous noise.

Most SOAEs are fairly stable, although their frequencies and relative levels may change somewhat from session to session. Some however are unstable, and fluctuate rapidly (typically once or twice a second), appearing and disappearing or switching between 2 quasi-stable states at different frequencies (Wilson and Sutton 1983, Burns et al 1984, Jones et al 1986).

1.2.6 Emissions at distortion product frequencies

If one continuous tone is fed into the ear a stimulus-frequency emission (SFE) may be detected in the ear canal; if two tones are fed in then the ear canal signal also contains intermodulation components, the strongest being at the cubic difference tone (CDT) frequency $2f_1-f_2$ (Kemp 1979a, Wit et al 1981, Wilson 1980e) but there are also many others (Kemp and Brown 1986 in man). The same distortion products are found in the round window electrical recordings (cochlear microphonic). The precise origin of these is still controversial. It appears that the most effective suppressors are near the primaries (Kemp and Brown 1983, Brown and Kemp 1984), suggesting that this is the site of origin. On the other hand the CDT component is strongest when it occurs at a frequency where the SFE is strongest (Kemp 1979a, Wilson 1980e, Wit et

al 1981, Schloth 1982) pointing to it originating from the CDT site. In fact both sites may be contributing. Behaviour of these distortion products also often appears to differ from that of SFEs suggesting that different mechanisms may be involved (Kim 1980, Kemp and Brown 1983, Kemp 1986).

The consequence of most interest here is that, if one imagines the 2 external tones replaced by 2 SOAEs, one might predict that an SOAE may sometimes occur at $2f_1-f_2$. Indeed this is found, and where there are several SOAEs in one ear they are sometimes in this frequency relation (Strickland et al 1985, Jones et al 1986, §3 and §4 below). Similar relationships can be found amongst the high level emissions measured by Glanville et al (1971) and Wilson and Sutton (1983).

1.2.7 Level dependence

EOAEs saturate strongly as stimulus level is increased and are limited to about 20 dB SPL (Kemp 1978, Wilson 1980c, Johnsen and Elberling 1982ab, Schloth 1982). Input-output functions approach linearity at the lowest levels (Wit and Ritsma 1979, Kemp and Chum 1980a, Wilson 1980c, Schloth 1982, Zwicker 1983, Grandori 1985) but the slopes fall below unity above about 10 dB SL. Similarly, a few cases excepted (e.g. Glanville et al 1969), most SOAEs have levels below 20 dB SPL.

1.2.8 Effects of Pathology - Physiological Vulnerability

Both EOAEs and SOAEs are highly vulnerable to cochlear insult by various agents. They are not found at frequencies where there is significant (more than about 15-25 dB) sensorineural hearing loss (Kemp 1978, Rutten 1980, Johnsen and Elberling 1982a, Kemp et al 1986).

Aspirin has been found to reduce the amplitude of EOAEs (Johnsen and Elberling 1982a, Long et al 1986) and abolish SOAEs (McFadden and Plattsmier 1984, Long et al 1986). Furosemide and ethacrynic acid also reduce or abolish EOAEs in laboratory animals (Anderson and Kemp 1979), as does hypoxia and death (Wilson 1980d, Wilson and Evans 1983). Exposure to loud noise also depresses EOAEs in animals (Anderson and Kemp 1979, Wilson and Evans 1983) and in humans (Kemp 1981 1982, Fritze and Kohler 1986). On the other hand Zurek and Clark (1981) and Clark et al (1984) have found that noise exposure can apparently *produce* SOAEs in chinchillas, and McFadden and Plattsmier (1984) found an extra SOAE in one of their aspirin subjects after the effects had worn off.

The implications of these results are that the OAE generator mechanism is easily knocked out by any cochlear insult, and must therefore be associated with the physiologically-vulnerable part of the cochlear filter (what used to be called the second filter). On the other hand at least some emissions may arise from minor local pathology, provided the mechanism remains intact over some adjacent part of the cochlea.

1.2.9 Effects of middle ear changes

Neither SOAEs nor EOAEs are observed where there is significant conductive loss (Kemp 1978, Kemp et al 1986), presumably because this attenuates them to below detectability. Stiffening the middle ear system usually reduces amplitude and increases frequency of SOAEs, whether this is done by changing the pressure in the meatus (Wilson and Sutton 1981, Kemp 1981, Schloth 1982, Schloth and Zwicker 1983), or by activation of the stapedial reflex (Schloth 1982, Schloth and Zwicker

1983, Rabinowitz and Widin 1984). However the effects are not always so simple or consistent, and sometimes new SOAEs are seen under the altered condition (Wilson and Sutton 1981). These results are interpreted as being due to a combination of (a) the extra attenuation for the SOAE through the middle ear and (b) the impedance change at the cochlea-stapes boundary which affects the phase of the wave reflected back into the cochlea, thus causing a change in SOAE frequency. The anomalous cases may be due to shifts in the balance of energy between neighbouring SOAEs.

Changes in meatal pressure or intracochlear pressure (through postural changes) also affect EOAEs as observed directly (Wilson 1980c) (although this was not found by Johnsen and Elberling 1982b), and indirectly through loudness maxima (Kemp 1979b) and threshold fine structure (Wilson 1980c, Wilson and Sutton 1981, Schloth 1982) (see §1.2.13 below).

1.2.10 Synchronisation and frequency-locking of SOAEs.

The phase of an SOAE can be synchronised by a click stimulus of sufficient level (Wilson 1980c). This is also shown in Fig.2 of Kemp (1981), which shows more clearly that the SOAE is only affected some 8 ms after the synchronising click. Short tone bursts also synchronise the SOAE (Wilson and Sutton 1981), from which one can infer that a continuous tone of the same amplitude should synchronise the SOAE continuously - i.e. the SOAE should be pulled in to the frequency of a tone nearby in frequency. Such frequency-locking is observed and is very sharply tuned near the SOAE frequency (Wilson 1980c, Wilson and

Sutton 1981), with a very low threshold where only a very small energy is required to phase lock the SOAE (Wit 1986); this implies a close link between the generator and the late stages of cochlear filtering. When full frequency-locking occurs the actual SOAE level appears to be unaffected, although suppression may be expected at higher levels of the external tone (Wilson and Sutton 1981).

For intensities not quite sufficient to obtain full phase-locking, partial synchronisation is observed - phase-locking occurs for a certain period before noise and random fluctuations allow the SOAE to escape and free-run until the phases are again sufficiently close to re-establish locking (Wilson and Sutton 1981, Zurek 1981). Where the SOAE is audible subjectively as a tonal tinnitus, this alternate locking and unlocking to the external tone is heard as irregular beats (Wegel 1931, Flottorp 1953, Ward 1955, Wilson 1980c, Zurek 1981, Wilson and Sutton 1981, Schloth 1982), whose rate depends on the level of the external tone and which are a property of partial frequency-locking of a self-oscillating system (Kharkevich 1962).

Synchronisation can also be seen in click-evoked OAEs. The results reported by Kemp and Chum (1980a) on the interaction of two such emissions can also be interpreted as synchronisation of the response to the first click by the second (later) click. The second click can be crudely thought of as 'resetting' the generator, with a finite action time and dependent on relative levels.

1.2.11 Suppression of OAEs

All types of OAEs can be suppressed by external sounds. The suppression effect is sharply tuned in frequency (Kemp 1979a, Kemp and Chum 1980a, Wilson 1980c, Wilson and Sutton 1981, Wit and Ritsma 1980, Wit et al 1981, Zurek 1981), and the resulting 'suppression tuning curves' (STCs) are very similar in shape to other psychoacoustical measures of frequency selectivity and to neural tuning curves seen in animal studies. Often however the tip of the STC occurs at a slightly higher frequency than the OAE.

In the time domain, for click-evoked OAEs, two moderate click stimuli interact nonlinearly only if less than about 6 ms apart, with maximal effect when simultaneous (Kemp and Chum 1980a). Kemp's (1981) results on the effect in the time-domain of a click on an SOAE are best interpreted as synchronisation of the SOAE by the click (see §1.2.10). Schloth (1982) (also in Schloth and Zwicker 1983) also showed a 6 ms delay to suppression onset of an SOAE by a tone burst, and that the time course of suppression followed an exponential curve with a 13 ms time constant, which was the same for onset and recovery. On the other hand Zurek and Clark (1981) found that the recovery time constant in chinchilla was much longer (80-120 ms) than that for onset.

SOAEs often shift in frequency under suppression (Wilson and Sutton 1981). This probably accounts for at least some of the cases where 2 (or more) lobes are observed in the STCs, but not all (e.g. Evans et al 1981, Wilson and Sutton 1983). (See §5 for fuller review).

A different type of suppression effect is seen in Zwicker's (1981, 1983a) experiments. A triggered low-frequency masker decreases the emission evoked by a click differently at different points within the period of the masker; this effectively shows the effect on OAE generation of the displacement bias (toward scala tympani or scala vestibuli) caused by the masker. Measurement of the suppression effect at different masker phases give 'suppression period patterns' which almost exactly mirror the equivalent 'masking period patterns' for threshold elevation. This reinforces the view linking OAE generation with hearing sensitivity and that the same active process is responsible for both.

1.2.12 Habituation and Fatigue

The strength and form of the cochlear echo is practically the same whatever the click repetition rate, up to 200 s^{-1} (Rutten 1980, Kemp 1982, Grandori 1985). This rules out synaptic involvement in EOAE generation. Similarly SOAEs maintain their levels over indefinite periods. However exposure to loud noise or tones does fatigue the mechanism and reduce the EOAE, which then recovers over 10-15 minutes. Zwicker (1983b) showed a very close correspondence between recovery from temporary threshold shift and the EOAE magnitude. Kemp (1981, 1982) found that during recovery a 'bounce' or enhancement of the echo or SOAE occurred a minute or so after exposure. This suggests that there may be an overall gain control operating through the efferent system, as modelled by Kemp (1986).

1.2.13 Multiple internal reflection and 'whole cochlear resonances'

The existence of a boundary (at the oval window) at which partial reflection of an OAE back into the cochlea can occur has many consequences. These can be most clearly understood by considering the click-evoked OAE. As it emerges from the cochlea the oval window moves and the effect is transmitted backwards through the middle ear to be detected by the microphone in the ear canal. At the same time the movement of the oval window also launches a new travelling wave on the basilar membrane. Equivalently one may say that as the emission reaches the cochlea/middle ear boundary (by whatever method of propagation from its site of generation) there is likely to be some impedance mismatch, which will result in part of the response being transmitted through the middle ear to the meatus and part being reflected back into the cochlea. The secondary travelling wave acts as a new stimulus when it arrives at the EOAE generation site, causing a new EOAE, and so on. Direct evidence of this is found in occasional subjects (GFP in Wilson and Sutton 1981, Fig.1) as a multiple echo, in which the primary echo is followed by further delayed subsidiary versions.

This multiple internal reflection in the time domain has a consequent effect in the frequency domain, which is to superpose a regular pattern of peaks and dips in the EOAE spectrum, with a frequency spacing equal to the reciprocal of the delay time: thus 10 ms delay gives 100 Hz spacing (e.g. Kemp and Chum 1980b, fig.3). These have been termed 'whole cochlear resonances' by analogy to the acoustic resonances of a tube, but it is important that they should not be confused with the inherent resonances at each place on the basilar membrane. The fine-

structure is an overlay on the EOAE mechanism, arising from it being housed within a resonating cavity, and is a property of the cavity (the delay time) rather than of the EOAE generator itself. It is present in *any* method of looking at EOAEs, with the exception of time-windowing of a click-evoked OAEs where the spectrum free of fine structure should be seen to the extent that secondary reflections have been windowed out. The sharpness of these 'cavity' resonances is determined by the proportion of energy reflected back to the generator (Kemp 1980).

Other consequences of this phenomenon are that variations with identical frequency spacing may also be seen in:

(1) the pure-tone audiogram (Elliot 1958, Thomas 1975, Kemp 1979b, Wilson 1980c, Schloth 1982, Cohen 1982, Zwicker and Schloth 1984, Long 1984);

(2) the subjective loudness of a quiet tone of constant SPL (Kemp 1979b, Wilson 1980c);

(3) the SPL of a tone driven by a constant velocity source into the sealed ear canal (Kemp 1979ab, Wilson 1980ac);

Even without multiple internal reflection, interference between the SFE and the stimulus will lead to some ear-canal SPL variations (3), although as Kemp (1980) pointed out these would be sinusoidal: cochlear resonances sharpen them up.

Historically it was this auditory fine-structure which originally led Kemp to develop the cochlear resonance hypothesis and to predict the existence of EOAEs (Kemp and Martin 1976, Kemp 1978, 1979b, 1980). All these are low level effects, and smooth out at higher stimulation

levels, as the EOAE saturates. The objective nature of effect (3) allows one to show that it still occurs at levels well below (35-50 dB below) psychoacoustical threshold (Wilson 1980b), thus ruling out any involvement of neural processes in generation.

The multiple-reflection hypothesis is also supported by : the frequency regularity of the fine structure (e.g. Kemp 1979b, Wilson 1980c); the predictability of sound pressure peaks and dips from the EOAE phase (Wilson 1980c); the correlation between fine-structure in the audiogram and in the EOAE spectrum (Zwicker and Schloth 1984); and the effects on the fine structure of meatal pressure and postural effects which change the impedance matching (see §1.2.9).

An alternative hypothesis put forward by Manley (1983) proposing that each sensitivity peak represents an independent sharply-tuned resonance with its basis in cochlear structure cannot explain these findings, and also implies an inherent sharpness of tuning of a single element far greater than found by other means.

1.2.14 Inter-relation between click-evoked EOAEs, SFEs, and SOAEs

Zwicker (1983a) verified that EOAE responses to tone bursts could be predicted by summing responses to single sinusoids, in line with the linearity of the behaviour of EOAEs at low levels (§1.2.7). One would therefore predict that click-evoked OAEs and continuous-tone evoked OAEs (SFEs) are the same phenomenon in different guise and should therefore show the same frequency distribution. This is in contrast to Ruggero et al's (1983) suggestion that SFEs are uniform whilst echoes

are irregular. The evidence however shows that SFEs do also show irregular frequency distribution (e.g. Kemp and Chum 1980b).

The multiple internal reflection (or cochlear resonance) concept is the key to understanding how SOAEs arise from EOAEs. Self-sustaining oscillation can occur if the energy reflected back to the EOAE generator from the oval window is sufficient and in phase. The greater the loop gain the sharper the resonance and the more likely an SOAE is to develop. In fact (as Kemp 1981 pointed out) one can consider SOAEs as resonant enhancements of background cochlear noise. SOAEs are thus found coincident with loudness enhancements and threshold fine-structure minima (Kemp 1979ab, Schloth 1982, Zwicker and Schloth 1984, Jones et al 1986). It is also clear from this that there is a minimum spacing of 1/(delay time) for (simultaneous) SOAEs in an ear (Schloth 1982, Zwicker and Schloth 1984).

1.3 GENERATION AND PROPAGATION OF OAEs

A number of problems relating to OAEs and the active process remain unexplained. For example, what is the active mechanism and how does it sharpen the BM response? Why, when sharpening is presumably occurring for all frequencies, is the frequency distribution of EOAEs so arbitrarily non-uniform? Why are EOAEs limited to about 20 dB SPL when sharpening is still occurring at high levels (Evans 1977)? How does the long latency of EOAEs arise? How does the OAE propagate from the cochlear site of generation to the oval window?

Whilst the actual mechanisms of the active process generating OAEs are still unclear (see Wilson 1984 for a review), theories have been put forward to explain in general terms the non-uniform frequency distribution and the propogation of OAEs.

1.3.1 The irregularity hypothesis

Kemp (1978, 1979ab) suggested that EOAEs arose from reflection of the travelling wave from points where there was a large change in the mechanical impedance of the BM over a short distance. He further suggested that the resonant enhancement caused by the active process near the peak of the travelling wave would lead to such an impedance discontinuity. However Zwislocki (1983) showed that there should be no wave reflection from even a very sharp vibration maximum in a cochlear model. Another problem is that because the sharpening process is presumably occurring at all frequencies this mechanism would lead to a uniform frequency distribution of EOAEs, and the latency (as defined by the change of phase with change of frequency) would be very low because the phase of the travelling wave to the peak is independent of frequency (Kemp 1986). (In fact this low-latency energy leakage from the travelling wave peak may be the dominant mechanism in gerbil ears - Kemp 1986).

If however there was an irregularity of tuning or mechanical properties at a *fixed* position on the BM then a latent EOAE concentrated round the frequency for that place might be expected (Wilson 1980d, Kemp 1986). Such irregularities might occur at random points along the BM. Each of these would lead to a contribution to the EOAE in the frequency region

associated with that place, so that the total click-evoked echo would consist of contributions from all such irregularities. In fact there are two different views of these irregularities following from the alternative models of OAE propagation in the cochlea.

1.3.2 OAE propagation within the cochlea

Two models for how the EOAE is transmitted from site of generation to stapes have been proposed. In Kemp's (1978) model transmission is by means of a reverse travelling wave which arises from reflection of the normal forward travelling wave by the impedance irregularities, in a manner analogous to the partial reflection of an electromagnetic wave from an impedance mismatch in a waveguide (although in the cochlea this would be an 'assisted' or 'active' reflection). Whether in fact reverse travelling waves are feasible has been much debated. De Boer et al (1986) have shown that there is a fundamental asymmetry in propagation, and that whilst reverse waves can be produced they always undergo reflection as they travel, which leads to substantial attenuation. The existence of OAEs in frogs (Palmer and Wilson 1982), which have no BM, also points against a reverse travelling wave.

The alternative model proposed by Wilson (1980df) included the idea that propogation was by a direct compressional wave. He proposed that the whole of the normal cochlea was capable of EOAE generation, but that due to phase cancellation of the responses from different points no net response was seen in a perfectly uniform cochlea. Any irregularity or imperfection in structure would however disturb this cancellation and lead to a net summed response. The specific mechanism

suggested was that slight volume changes occurred in haircells in synchrony with their excitation. Thus the net summed response caused by an irregularity would be a net oscillatory volume change within the cochlear duct which would directly move the oval window by compression. It now appears though that such volumetric changes would not be sufficient to give some of the higher SPLs seen for SOAEs. However the general model of 'distributed activity /summed response' is still applicable to other mechanisms - for example if OHC stereocilia amplify the BM vibration then the net BM vibration would be the net summed response in the model, again moving the stapes by compression (see also §2.1).

In this latter model the localised irregularity merely reveals (by disturbing phase cancellation) the underlying activity which is distributed along a considerable length of the BM. This is in contrast to Kemp's (1978, 1979ab) model where the irregularity reflects the travelling wave and acts like a localised source or resonator.

In both models when the EOAE arrives at the oval window energy is partially transmitted through the middle ear to the meatus, and partially reflected back into the cochlea (the resulting vibrations relaunch a new forward travelling wave), leading to restimulation of the generator etc - i.e. multiple internal reflection (§1.2.13).

One clear difference between the models is that a reverse travelling wave would have a reverse travel time whilst this would be negligible for the compression effect with the 'summed response' model. In fact

one motive of Wilson's model was to explain the results of an experiment (Wilson 1980d) in which the acoustical cochlear echo emerged with the same latency as its round window electrical correlate, which seemed to rule out any reverse travel time. Similarly Evans et al (1981), studying the suppression of an SOAE, found both acoustic and round window electrical signal to have the same suppression latencies. There has however been some debate about the interpretation of these results and whether they necessarily imply zero reverse travel time (Brown and Kemp 1985).

As Kemp (1980) pointed out, the two propagation mechanisms give different predictions of the parameter for internal feedback of the OAE into the cochlea, but the data are not strong enough to distinguish them. Also the existence of a reverse travelling wave would have dramatic effects on the resultant travelling wave pattern at low levels, with the development of partial standing waves and local nulls.

1.4 OVERVIEW OF THESIS

This thesis describes a number of investigations into both spontaneous and evoked oto-acoustic emissions and their relation to normal hearing and pathology. These are:

§2. A computer modelling study of Wilson's (1980df) 'distributed activity/summed response' model for generation of EOAEs.

§3. A study of the prevalence of spontaneous oto-acoustic emissions (SOAEs) in a small group of normal-hearing adults;

§4. A study of the relationship of SOAEs and tinnitus in a population of clinical tinnitus sufferers.

§5. Three studies of suppression of OAEs by pure tones

§5.1 suppression of SOAEs in 2 ears;

§5.2 suppression of continuous-tone evoked OAEs (stimulus-frequency emissions) in 1 ear;

§5.3 suppression of click-evoked OAEs (cochlear echoes) in the same ear.

2. MODELLING OF COCHLEAR ECHOES USING A SUMMED RESPONSE APPROACH

2.1 INTRODUCTION

As outlined above (§1.3.2) Wilson (1980df) suggested that the active mechanical response generating EOAEs occurred throughout the length of the cochlea, not just at local 'hotspots', with the sum of all this activity (representing the response seen) acting directly by a compressional wave on the oval window without the involvement of a reverse travelling wave. It was suggested that this summed response would normally be small due to phase shifts, but that any irregularity in structure or mapping would disturb this phase cancellation and lead to the appearance of a narrow band response at the frequency for the place of the irregularity. Although Wilson (1980df) did suggest a specific mechanism (haircell swelling), this 'distributed activity/summed response' model is much more general in its applicability. For example, because stapes volume displacement at any instant equals net BM volume displacement, any active sharpening process affecting BM motion may be coupled (by compression wave) to the stapes. In this case the summed response represents this net BM displacement after sharpening.

A simple computer simulation was set up to explore the characteristics of this type of 'summed response' model, and to see if the presence of an irregularity would give rise to responses with some of the observed features of cochlear echoes, in particular their frequency concentration into narrow bands and long latencies - the latter in view of the fact that this type of model lacks a reverse travel time to

account for any of echo latency. Active and non-linear processes do not explicitly feature in the cochlear modelling, but could easily be incorporated.

Most of the work described in this chapter has been published previously (Sutton and Wilson 1983).

2.2 METHODS

2.2.1 Basilar membrane model

The first requirement for this modelling is a simulation of the amplitude and phase of the travelling wave along the basilar membrane (BM). The one-dimensional BM model described in de Boer (1980) was used for this, from an earlier pre-publication version of this report. This was chosen in preference to the more sophisticated 2- or 3-dimensional models because it is much simpler to implement and more economical in computer time, while still displaying all the necessary features with sufficient accuracy for the purposes of this study. It was implemented in FORTRAN (based on de Boer's listing) on a GEC 4080A computer: the complete program, including the second filter and summed response modifications, is listed in Appendix 1.

The length of the BM was divided into 1000 sections, rather than de Boer's 250, for greater accuracy, making the incremental distance 0.035 mm. The type of responses produced by the model are controlled by the impedance function, defined at each point as:

$$Z(x) = (C_o/i\omega)\, e^{-\sigma x} + i\omega M_o + R_o e^{-\mu x}$$

where x is distance along the BM, ω is radian frequency, C_o is stiffness, M_o is mass density, R_o is resistance, and α is a constant. The parameter values used are those given by de Boer (1980) as being approximately representative of values for man, *viz.* $C_o=10^9$ dyn.cm^{-3}, $M_o=0.05$ g.cm^{-2}, and $\alpha=3$ cm^{-1}. This form of Z(x) gives a true resonance at the value of x where stiffness and mass components cancel. The resistive term is defined in the form

$$R_o = \delta\ (M_o C_o)^{\frac{1}{2}}$$

where δ is the damping constant, so as to give a loss factor δ constant with respect to x, making the travelling wave pattern invariant as a function of frequency and position. A change of 1 octave shifts the pattern along by 4.62 mm. Two models were implemented: a low damping model (with $\delta=0.05$) which gave a fairly resonant type of response, and used by de Boer (1984); and a high damping model (with $\delta=0.2$) which gave responses more like those seen by Bekesy (1960). Calculated frequency responses of one point on the BM (the resonance point for 1 kHz) are illustrated in Fig.2-1 as 'BM', for both values of δ.

2.2.2 'Second filter' sharpening stage.

To this basic response a 'second filtering' stage was added to sharpen the responses. This work was carried out before it was confirmed that the travelling wave is very sharply tuned. However, whilst this has made the original concept of the second filter redundant (see §1.1.2), the extreme physiological vulnerability of the very sharp BM response means it can still be considered as a basic broad filter sharpened up by a second stage, so the model can still be applied.

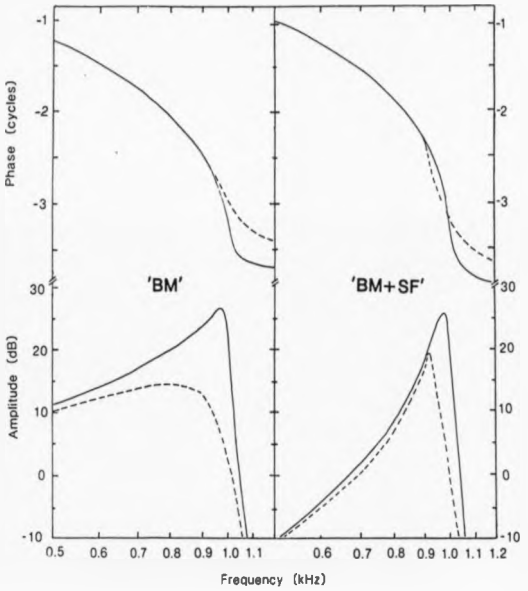

Fig.2-1 Phase and amplitude responses for cochlear models, relative to stapes motion. **Left:** for one point on basilar membrane (BM) with damping factor 0.05 (solid lines) and 0.20 (dashed lines). **Right:** after second filter (BM+SF), centred at 1001 Hz with Q=8 for low damping, and at 929 Hz with Q=20 for high damping.

The model second filter (SF) was a simple resonant second order LCR filter with a Q value of 8, chosen to make the combined filter bandwidth about 140 Hz at 1 kHz (i.e. Q_{10dB}=7), matching psychophysical measures (see Evans and Wilson 1973). At each point along the BM it is assumed that there exists an SF whose best frequency is specific to that point. It is necessary to decide the placement of the SF relative to the travelling wave pattern: for example the SF tuned to 1 kHz could be positioned either

(i) at the point of maximum amplitude of the travelling wave pattern for 1 kHz ,

or (ii) at the point of mechanical resonance of the BM (i.e. the point at which the reactive components of Z(x) cancel for 1 kHz.

It was decided rather arbitrarily to adopt the second option for the low damping modelling (δ=0.05), thus centring the SF at the point where the envelope has fallen by 3.6 dB on the HF cutoff side. There is some evidence that this is approximately the correct positioning for this type of model (most recently in the pre- and post-mortem data of Sellick et al 1982). The SF response was normalised at unity gain at its centre frequency. This gives the total responses shown in Fig.2-1 as 'BM+SF', and the resulting vibration patterns at a particular instant are shown in Fig.2-2a (BM) and 2-2b (BM+SF).

For the δ=0.2 high-damping model, a Q value of 20 was needed to give the same Q_{10dB} bandwidth to the combination. In this case the SF was centred at 3 dB down on the HF cutoff (i.e. below resonance in this case) as a compromise between options (i) and (ii). The resulting responses are illustrated on the right of Fig.2-1.

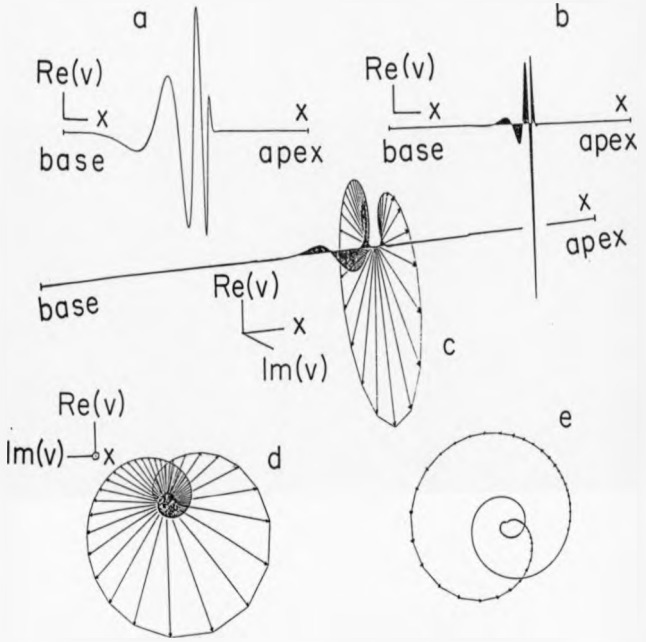

Fig.2-2 Illustration of summed response (SR) derivation for a 1 kHz input (δ=0.2, Q=20). Re(v) and Im(v) are the real and imaginary parts of the (complex) excitation, x is distance along BM. (a) BM vibration pattern. (b) BM+SF excitation pattern (net area under curve is proportional to the instantaneous SR). (c) Representation of (b) in complex vector form. (d) Projection of (c) looking down the x-axis. (e) Derivation of SR by laying vectors end-to-end (not same scale). SR is distance between ends of chain (small with no irregularity).

For a given input frequency the program calculated at each point the amplitude and phase of the BM response as in Fig.2-1; the effect of the SF at each point is then incorporated. The resulting 'BM+SF' response is taken to represent 'activity' at that point, and can be illustrated in vector form as in Fig.2-2c. This 'activity' can represent either neural excitation or (as we can now suppose) mechanical excitation, or hypothesised OHC swelling, or the associated electrical potential arising. The resultant pattern should be imagined as rotating about the x-axis at rate ω, figs.2-2b and 2-2d being 2-dimensional projections of it.

2.2.3 Calculation of 'summed response'

Summing all these vectors then gives the resultant 'summed response' (SR) representing the net mechanical response transmitted back through the middle ear as a stimulus-frequency emission, or alternatively the unweighted cochlear microphonic response. This vector summation is illustrated in Fig.2-2e (not on the same scale) where the vectors of Fig.2-2d are laid end-to-end to give a phasor diagram (familiar in optical physics). The gap between the ends of this curve gives the vector representing the SR. In the case illustrated (with regular mapping) this is very small.

The finite element length does not introduce substantial errors provided that adjacent vectors do not differ much in phase or amplitude. In fact the maximum phase and amplitude changes between adjacent points (for BM+SF for a 1 kHz input) were respectively 27° and 2.8 dB (near resonance). It was verified that a 4000 point computation

gave results that were very similar, and the slight accuracy improvement did not warrant the increase in computing time.

2.2.4 Impulse responses

Impulse responses for BM, BM+SF and SR were computed from the frequency responses by adding together cosine waves with the appropriate amplitudes and phases (10 Hz steps were used, over a sufficient bandwidth). Clearly this assumes linearity: nonlinear behaviour of the BM would mean that its impulse response could not be found this way, but given the impulse response for the total cochlear filter the SR to a click would still be the appropriate sum of these over the length of the cochlea (see §2.4).

2.3 RESULTS

2.3.1 With regular mapping

With a 'normal' BM and logarithmic frequency mapping of the SFs along the BM, the summed response (SR) - the vector sum of the total 'activity' along the cochlea - is extremely small and nearly uniform with frequency; in other words the vectors effectively cancel out. This near-perfect cancellation only occurs with the presence of the SF, which gives an extra π radians shift over the length of the cochlea. With only the first stage BM filter and no SF, the SR is proportional to the volume displacement of the stapes and is therefore not negligible. In this case the corresponding impulse response is of very short delay, and the effect of this is effectively to alter the impedance of the cochlea as seen from the stapes: it does not produce a delayed echo-like response.

2.3.2 With small mapping irregularity

The regular mapping was disturbed by incorporating a small irregularity in the form of an 8-element plateau. This gives 8 elements (equivalent to a length of 0.28 mm) all with their SFs centred on 1003 Hz instead of decreasing monotonically from 1022 Hz to 985 Hz (see Fig.2-3). (For δ=0.2, because the irregularity is at the same physical location and the SF mapping is different, the tuning is to 929 Hz). The effect of this (as shown in Fig.2-4) is to greatly enhance the summed response (by 34 dB) (relative to the regular mapping case) in a narrow frequency band (50 Hz and 30 Hz respectively for the two δ-values). The resulting frequency response is a little sharper than the 'BM+SF' curve (dashed line). The phase slope also exceeds that for 'BM+SF'. It will also be seen that in the low damping case there is a notch in the amplitude curve above the peak: this arises due to cancellation between the local response and the small non-zero response from the regular mapping. The link between the two parts of the phase curve above and below this notch is indeterminate since the amplitude is of the same order as the rounding errors of the computation.

The corresponding model impulse responses are shown in Fig.2-5. The curve for SF shows the exponential decay characteristic of a simple resonant filter; the BM curve shows a short wavefront delay followed by a response determined by the damping factor; and 'BM+SF' shows the characteristics of the more bandpass filter shape. Finally for 'SR' (which represents the model click-evoked cochlear echo) there is the striking feature of a slow build-up giving a 14 cycle delay to peak response, compared with 3 cycles for 'BM' and about 8 or 6 cycles for

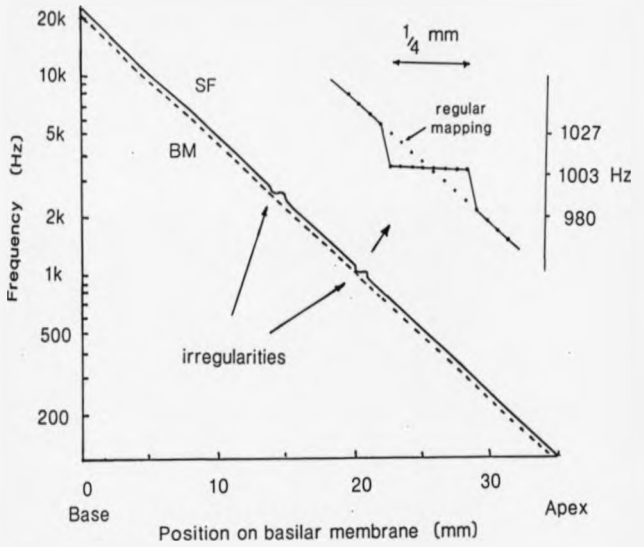

Fig.2-3 Mapping of SF tuning on to distance along BM (solid line). Two 'plateau' irregularities are shown present, (one shown enlarged: 8 elements =1/4 mm). BM peak tuning values are shown (dashed) with an exaggerated basalward shift relative to resonance frequency.

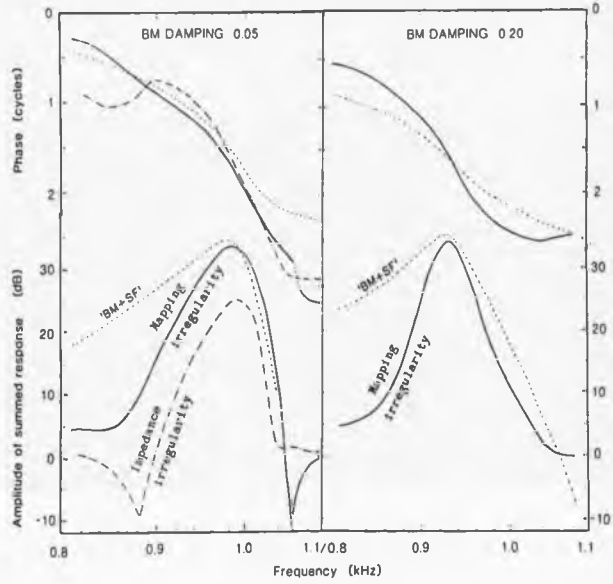

Fig.2-4 SR phase and amplitude for both damping models. Solid lines: with 8-element plateau in SF mapping. Dot-dash lines: with 8-element plateau in BM impedance. Dashed: BM+SF responses for comparison. (Amplitude zero is for regular mapping, phase zero is arbitrary).

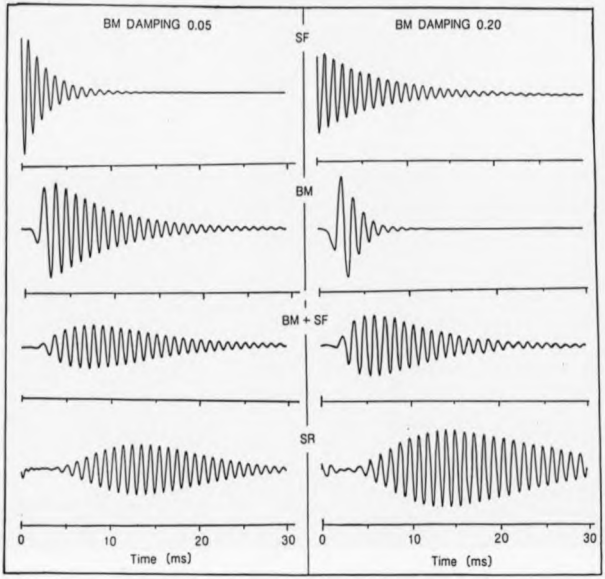

Fig.2-5 Impulse responses for both damping models. **Top to bottom:** for SF alone; for BM alone; for BM+SF at same point; for SR with 8-element SF mapping irregularity.

'BM+SF' (depending on δ). This is very comparable with observed latency of the cochlear echo in man.

2.3.3 Effect of 'shape' of irregularity

Eliminating the sudden discontinuities of the plateau by making the gradient change smoothly altered the results very little, except that a wider irregularity was required to give a comparable response. Changing the slope of the irregularity region was found solely to alter the magnitude but not the phase slope of the SR. Hence the character of the results do not depend critically on the nature of the mapping irregularity. It was therefore decided to use the simple plateau irregularity in all subsequent investigations.

2.3.4 Effect of width of irregularity

Fig. 2-6 shows the effect of altering the width of the irregularity from 3 to 15 elements, which is principally on the bandwidth of the SR, with little change in the phase slope. The impulse response corresponding to the 15-element case is shown, showing a slightly lower latency to peak than for 8 elements (Fig.2-5, SR, left).

2.3.5 Effect of two irregularities

Fig.2-7 illustrates the effect on SR frequency and impulse responses of inserting two plateau regions (each of 8 elements) in the mapping at various separations. This shows distinguishable frequency bands when the plateau centres are separated by more than about 15 elements, but merging when closer. Since this is a linear model this is as expected, with the two SR contributions simply adding vectorially. In other

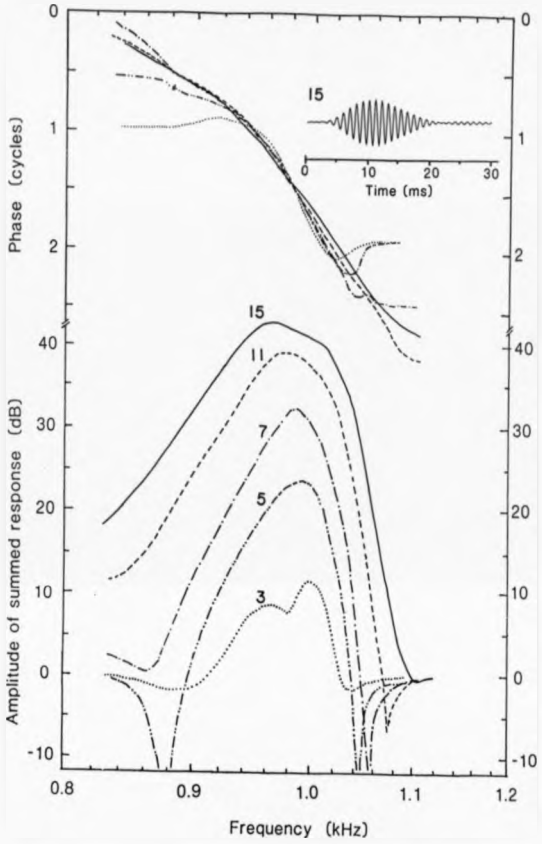

Fig.2-6 Effect of altering width of irregularity (from 3 to 15 elements) on *SR* phase and amplitude (low damping model). Impulse response is illustated for 15-element case.

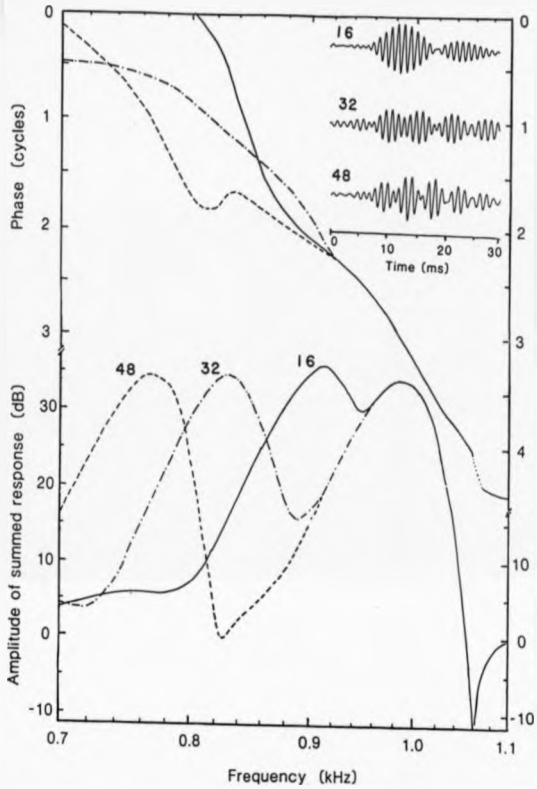

Fig.2-7 Effect of two irregularities at different separations (16 to 48 elements) on SR phase and amplitude (low damping model). Corresponding impulse responses are also shown.

circumstances the vector addition might as easily lead to a sharp notch in the spectrum as is sometimes observed experimentally (see §5).

2.3.6 Effect of irregularity in BM impedance

So far we have been examining the effect of an irregularity in the SF mapping. An alternative is to put an irregularity in the impedance of the cochlear partition, leaving the SF mapping uniform. For this an 8-element section of the BM was assigned a constant impedance (an impedance plateau) corresponding to that at the centre of the region (rather than having stiffness and resistance decreasing exponentially). One interesting consequence of this was that the BM phase and amplitude data showed some ripples, indicating that some reflection of the travelling wave was occurring from the impedance discontinuities at the region boundaries and leading to a partial standing wave. The estimated reflection coefficient was 0.1; this is unlikely to seriously affect the validity of the modelling.

The resulting SR is illustrated by the dashed line in Fig.2-4. It is again very large and the characteristics are similar to those of the SF mapping irregularity, but the phase slope is steeper (equivalent to about 20 cycles delay to maximum) and shows a reversal in part of the spectrum.

2.3.7 Simulation of cochlear damage

Any insult to part of the cochlea would probably knock out the active process and the contribution to the SR from that part. In the model eliminating the response from a small region in an otherwise normal

cochlea gives a large frequency-specific SR. The SR is significant even with one model element not contributing, (Fig.2-8, dot-dash line labelled '1') which does, as theory predicts, have the same shape as BM+SF. Widening the damaged region to 8 elements broadens the SR bandwidth, and in the extreme case where all the basal contribution is eliminated (an attempt to model a high-frequency hearing loss) one obtains a response following 'BM' (solid line labelled 'HF'). These results show that the SR is also very susceptible to any non-uniformity of the sensitivity of the haircells - in other words an appreciable SR will arise with any variation of haircell integrity.

2.4 SIMPLE THEORETICAL TREATMENT OF SUMMED RESPONSE

The group latency of any impulse response is determined by the amplitude-weighted average phase slope (Goldstein et al 1971). From the results in Fig.2-4, it can be seen that the long group latency of the SR impulse response (compared with the cochlear filter BM+SF) is primarily the result of a higher maximum phase slope and of the sharper amplitude response which 'picks out' the higher-sloped sections of the phase curve.

An intuitive insight into how the above results arise, and predictions for other model conditions, can be gained from a simple theoretical treatment. From a few simple assumptions and approximations, it will be shown that:

1. Provided the SR for a uniform mapping can be considered negligible, only the region of the irregularity itself need be considered.

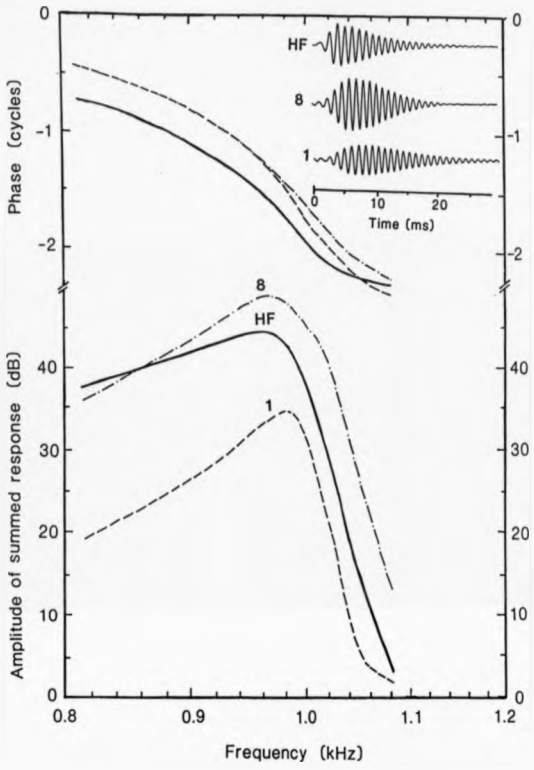

Fig.2-8 Effect of damaged (no SR contribution) region in the model on SR phase and amplitude (low damping model). **Dashed line:** 1 element damaged at 1 kHz place. **Dot-dash line:** 8 elements at 1 kHz place. Solid **line:** entire basal (high frequency - HF) end, up to 1 kHz place, damaged.

2. The phase of SR is to a first approximation that of the excitation at the centre of the irregularity. Hence the phase slope of SR is primarily determined by the phase slope of the cochlear filter, but there is an additional contribution from the way the SR arises.

3. The SR impulse response can more simply be found by summing together the impulse responses of the individual cochlear filters.

The procedure for calculating the SR (at an input frequency f) followed up to now can be written mathematically as:

$$SR(f) = \Sigma_x \; A(x,f)\, e^{i\theta(x,f)}$$

where A and θ are the amplitude and phase responses of the total cochlear filter (BM+SF in the above model).

A simplification is possible because the SR for a uniform map is nearly zero in the above 2-stage filter model. This allows the trick of subtracting the SR for the uniform map (= 0) from the right hand side of the equation (1) giving:

$$SR(f) = \underbrace{\Sigma_x \; A(x,f).e^{i\theta(x,f)}}_{\text{with irregularity}} - \underbrace{\Sigma_x \; A(x,f).e^{i\theta(x,f)}}_{\text{without irregularity}}$$

The next step is to see that all terms are identical except those within the bounds of irregularity itself. Therefore one need only consider this region - in other words the total SR is approximately the difference between the resultant excitation vector for the irregularity region and that for the same region without the irregularity. Thus for

the 8-element irregularity case, one need only do the summation on the 8 neighbouring vectors over this region.

SR phase

Next, to a first approximation over a small irregularity, the amplitude of excitation may be taken as constant, and the phase as linear. The phase of the resultant vector over the region would then be the same as that of the central vector contribution, and be identical for both regular and irregular mappings. Thus the SR, which is the difference between these resultant vectors would also have the same phase as the central element of the region, and the SR phase-frequency variation is therefore the same as 'BM+SF'.

In practice the SR phase slope at maximum amplitude is found to exceed that of 'BM+SF' slightly, owing to the breakdown of the 'constant amplitude' assumption above. The long latency seen in the SR impulse response is thus mainly inherent in the high phase slope of the cochlear filter, but also comes partly from an additional phase slope arising from the way the SR is compounded. The effect is also magnified because the SR amplitude response is sharper than 'BM+SF' and this picks out the steeper-sloped section of the phase curve, adding to the group delay.

SR amplitude

The same simplifications also allow one to show that the magnitude of the SR (with an irregularity in SF mapping) is proportional to

$$A.\ \phi'.\ (\varphi' + \tfrac{1}{2}\phi')$$

where ϕ' and ψ' are respectively the phase slopes of SF and BM over the irregularity, and A is the amplitude of the central vector. This matches the results of the full computation (for an 8-element plateau) quite well (within ±4 dB over the 10 dB bandwidth), considering the approximations made..

SR impulse response

The SR impulse response h has so far been found from SR(f) by:

$$h_{SR}(t) = \Sigma_f \{ SR(f).e^{i2\pi ft} \}$$

$$= \Sigma_f \{ \Sigma_x [A(x,f) \, e^{i\Phi(x,f)}].e^{i2\pi ft} \}$$

In fact the order of the summations can be reversed giving:

$$h_{SR}(t) = \underset{\text{all cochlea}}{\Sigma_x} [\Sigma_f \{ A(x,f).e^{i\Phi(x,f)}.e^{i2\pi ft} \}]$$

$$= \Sigma_x H(x,t)$$

$$= \Sigma_f H(f,t)$$

(summation over logarithmic frequencies)

where H(f.t) is the impulse response of the individual cochlear filter with characteristic frequency f. In other words the SR impulse response could be found simply by summing the impulse responses of all the individual cochlear filters. This is true for any cochlear model, not just the BM/SF one considered above. Where it also holds true that the SR for a uniform mapping is negligible at times of interest (say >4ms) one can write similarly to before:

$$h_{SR}(t) = \underset{\text{with irreg}}{\Sigma_f H(f,t)} - \underset{\text{without irreg}}{\Sigma_f H(f,t)}$$

where the sum is over the whole range of frequencies of tuning. But since the two expressions only differ over the region of the irregularity itself (which is say a 'plateau' tuned to the centre frequency f* instead of varying from f_1 to f_2) this becomes:

$$h_{SR}(t) = I_f [\, H(f^*,t) - \bar{H}(f,t) \,]$$

Writing $f=f^*+\Delta f$ and expanding $H(f^*+\Delta f,t)$ in a Taylor series for a small irregularity then substituting above, one obtains:

$$h_{SR}(t) \propto \frac{d^2H(f,t)}{df^2}$$
$$\propto t^2.H(f,t) \text{ approximately,}$$

which gives an extra group delay by weighting the later components over the earlier.

2.5 MODELLING USING A 2-SLOPE COCHLEAR FILTER

The simplifications discussed above (calculation of the SR from summing cochlear filter responses, etc) are more easily employed if one models using a filter whose impulse response has a known analytic form. One such, used by Goldstein et al (1971), has 2 segments of constant slopes (S_1 and S_2 dB/octave). This has an impulse response given by the approximate analytic form (Duifhuis 1973):

$$H_1(f,t) = (2\beta f/n!).(\beta ft)^n.e^{-\beta ft}.\cos(2\pi ft)$$

where $n = (S_1+S_2)/12 - 1$, and $\beta = \pi^2/(2+\ln(n+1))$.

The slopes were chosen to fit the previous BM+SF curve (with δ=0.05) at peak and -10dB points, and were 64 and 188 dB/octave (n=20). The resulting filter shape and minimum phase response are shown in Fig.2-9. The total phase change is much greater than for the δ=0.05 filter (66 *cf* 26 radians), but the phase slopes in the resonance region are very similar.

For a uniform mapping of filters the SR impulse response is the sum of all $H_1(f,t)$ over the cochlea. Putting $f=f_0e^{-\alpha x}$, then $df=-\alpha f dx$, and the required sum is the integral of $H_1(f,t)dx$, i.e. the integral of

$H_1(f,t)df/f$, which can be shown to be proportional to $1/t$. Thus the regular mapping SR does not sum to zero here because of the lack of an SF. However this has zero delay, and is negligible at times of interest (say >5ms). Thus we obtain that the SR impulse response peaks $2/\beta$ waves later than $H_1(f,t)$.

This can be seen in Fig.2-9 where the impulse response for the 1 kHz 2-slope filter is plotted for comparison with the SR derived for an 8-element 'plateau' irregularity at that frequency. The group latencies are about 10 and 11 ms respectively. The extra latency for the SR (compared with the cochlear filter) is not as marked as for the other model: this is because the more symmetrical shape of $H_1(f,t)$ means that envelope maximum and centre of gravity are closer together. The SR amplitude at 1 kHz is about 45 dB above that for the regular mapping, whilst in the time domain the impulse response is about 80 dB greater than the uniform response at 11 ms.

2.6 DISCUSSION

The above has shown that some of the features of the cochlear echo observed experimentally can be successfully modelled if it is assumed that some globally active mechanism (such as haircell swelling) is occurring in the cochlea. It has been shown that any slight irregularity due to randomness in the tuning or sensitivity of individual hair cells, or in BM physical characteristics, gives rise to a greatly enhanced summed response specific in frequency to the region of the irregularity. For model values chosen to match critical bands the calculated bandwidth and latency values for the SR turn out to

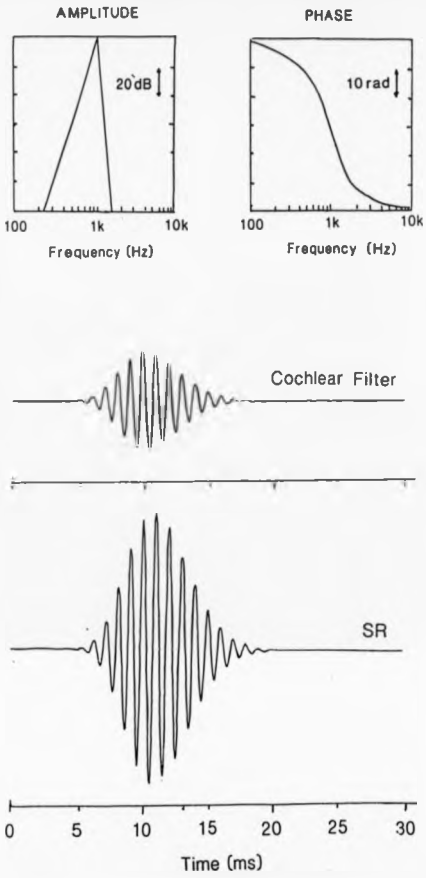

Fig.2-9 2-slope filter model with slopes 64 and 188 dB/octave centred at 1 kHz. **Top:** amplitude and phase responses. **Middle:** impulse response of filter. **Bottom:** impulse response for SR with 8-element irregularity in mapping.

correspond well with those of the cochlear echo. This is without recourse to a reverse travel time.

On the other hand, whilst the SR impulse responses of Fig.2-5 resemble the cochlear echoes seen in a few ears (e.g. subject GFP, Fig.1 in Wilson and Sutton 1981), most ears have echoes with only a few waves in a particular frequency band, although still with a long delay. However the results obtained with the 2-segment filter model (Fig.2-9) are closer to reality in this respect. Also, as seen in Fig.2-7, it is possible for a short wave group to arise from interaction and cancellation of the SR from two or more irregularities.

In the model strong responses are obtained not only from the irregularities of mapping etc, which might be slight variations inevitable in any biological structure, but also from areas of cochlear pathology, where the active process is knocked out (Fig.2-8). These responses are also tuned, more or less according to the size of the damaged region. This might account for the occurrence or enhancement of SOAEs after overstimulation (Kemp 1981, Zurek and Clark 1981) and of SOAEs near sharp audiometric drops (Wilson and Sutton 1981, this thesis §4).

2.6.1 Significance of SR for regular 'undisturbed' cochlea

It is noteable that while the two-stage (BM/SP) model gives negligible SR with an undisturbed cochlea, the one stage models (BM alone, or 2-slope filter) do not. In the 'BM alone' case this can be seen to be because the fluid volume displaced by the stapes is taken up by BM

displacement; i.e. the summed BM displacement (or SR) is proportional to the stapes volume displacement. In both cases however this SR component is non frequency-specific and non-delayed, and therefore the response merely acts to alter the effective input impedance of the cochlea as seen from the stapes. This component is thus equivalent to the 'wave-fixed' component discussed by Kemp (1986), which may be the dominant mechanism for SOAEs in gerbil and perhaps for distortion product emissions. Latent echo-like responses are however still produced by the presence of a 'place-fixed' irregularity, as observed for the 2-slope filter model (Fig.2-9).

2.6.2 Problem of saturation of CAE mechanism

One problem in understanding the cochlear echo is that while it is assumed that it arises as a result of an active process whose function is to sharpen the frequency response of the cochlea over a wide dynamic range (Evans 1977), the cochlear echo saturates at modest levels. However with a irregularity in sensitivity it is possible that as stimulus level is raised the difference in sensitivity between neighbouring elements would decrease; this would then reduce the relative strength of the SR and lead to an effective saturation of the cochlear echo without requiring the underlying active process to saturate at this level.

2.6.3 Possible model refinements

In the light of current knowledge it would be preferable to use a 1-stage cochlear model in which sharp BM responses were obtained by positive feedback (as in Kim et al 1980, Davis 1983, etc). Secondly, in the above no attempt has been made to model the effect of feedback of

the SR to the middle ear and reflection back into the cochlea, which is required to explain threshold minima, loudness enhancements, and the occurrence of SOAEs. The appropriate conditions would be where the SR amplitude was appreciable and in phase with stapes vibration. One can however see that the frequency spacings for threshold minima etc predicted by the model would be about 70 Hz at 1 kHz, which is close to experimental values (Kemp 1979, Wilson 1980bc, Schloth 1982). However the long straight phase slopes found experimentally (e.g. Wilson 1980c, §5.2), responsible for the regularity of these effects, were not found in the model responses.

3. SPONTANEOUS OTO-ACOUSTIC EMISSIONS IN A SMALL NORMAL-HEARING POPULATION

At the outset of this work, the only published data on prevalence of SOAEs, following Kemp's (1979a) initial brief report of their discovery in an unspecified number of normal ears, were those in Wilson (1980c), who tested 40 ears and found SOAEs in 10. However it was made clear that this was not a very exhaustive search. Therefore to extend the data a careful search was made in a small group of normal-hearing subjects.

3.1 DETECTION AND MEASUREMENT OF SOAEs

To detect and measure SOAEs one needs a sensitive microphone, low-noise amplification and a suitable analysing system. The systems used for this and the tinnitus study (§4) are first described.

3.1.1 Microphones

Sub-miniature microphones as used in hearing aids (Knowles types EA 1842 and the slightly larger BT 1753), sealed into the meatus with an E-A-R acoustic earplug, were used for some measurements. More often a version of Wilson's (1980a) 'sensitive microphone' was used, which has much lower electrical noise. This is a cheap commercial 11.5 mm electret condenser microphone modified as per Wilson (1980a) except that the back volume was not increased in this case. This was attached to a Duraluminium tube (4 mm i.d. and about 20 mm long) at the end of which an E-A-R plug was impaled for sealing comfortably into the ear canal. This is referred to below as the 'ES/Dural' microphone.

In some later measurements a Bruel and Kjaer 4155 pre-polarised electret microphone was used; it was coupled to the ear either with a soft rubber conical ring or with an adapter consisting of a modified microphone cover fitted with a plastic tympanometer eartip. The signal was passed through an FET follower circuit before amplification.

3.1.2 Pre-amplifier

A pre-amplifier box, giving low-noise amplification and switchable high-pass filtering of the microphone signal, and supplying the bias voltage where required, was used in all systems (Wilson 1980a).

3.1.3 SOAE signal analysis

The presence of low-level quasi-tonal signals can be detected by several methods:

(a) Audibility

Listening to the amplified microphone output uses the filtering capability of the experimenter's ear to separate the tone from the background noise. This simple direct method was always tried in the first instance (on headphones) as a first check for any very strong SOAEs, but is not sensitive enough for lower-level emissions.

(b) Dual-lockin system

In the lockin amplifier the incoming signal is multiplied with a square-wave reference signal, and any signal component at the fundamental frequency gives a DC output (extracted by a simple RC filter) which represents one vector component of the signal. By using two such lockins (Brookdeal 401) ganged together in quadrature phase relation, the DC vector outputs can be displayed as X and Y on an

oscilloscope. Thus a tone in the signal shows up as a spot rotating about the origin; as the reference frequency is tuned closer to the signal frequency the rate of rotation slows and the circle diameter increases. When the spot is near-stationary, frequency and level can be measured. The RC integrating times used for the SOAE scans were 30 ms (8 Hz bandwidth) or 100 ms (2.5 Hz). One disadvantage of this technique is that with a single frequency f_o at the input, spurious responses are seen at $f_o/3$, $f_o/5$, etc, so that care must be taken to distinguish these sub-harmonics from genuine SOAEs. This detection system was used for all SOAEs reported in §3 and §4.

(c) Slave filter system.

Occasionally analysis was carried out using a B&K 2020 heterodyne slave filter connected to a B&K 1024 sweep frequency oscillator. This acts as a constant bandwidth filter (selectable between 3.16 and 100 Hz) with centre frequency set by the oscillator. The output, monitored on a chart recorder, rises when the analysing band contains an SOAE.

3.1.4 Stimulus-frequency emission analysis.

The dual-lockin system can also detect the EOAEs evoked by a steady tone (SFEs) by using the external tone frequency as the lockin reference signal. A single stationary spot is seen on the CRO screen, with the vector from origin to the spot representing amplitude and phase of the total ear canal signal (stimulus-plus-SFE). The locus of such points as frequency is varied is smooth at high levels where the stimulus dominates, but at low levels loops or cusps may be seen, demonstrating the presence of SFEs. The external tone can be introduced either via a Sennheiser HD414 earphone over the ear (with the

microphone still in place), or via a driver-microphone placed on one arm of a small stainless steel Y-tube, (with the microphone on the other arm and the common end sealed in the meatus through an E-A-R plug).

3.1.5 Calibration

The responses of the 3 microphone systems in free-field are shown in Fig.3-1a. However this type of calibration is inappropriate at higher frequencies for the RS/Dural microphone because of resonances of the metal coupler tube, so that SPL at the diaphragm will differ from that from that in the meatus for short wavelengths. (The smaller dimensions of the other microphones mean their critical frequency is well above those of usual interest). A closed-field calibration was therefore performed, using a simple damped cavity to simulate the ear canal. This was a hard plastic tube about 2 cm long and of 1 cm diameter, and partly filled with acoustic foam to damp the cavity. The enclosed volume was about 1 cm^3. Tones were introduced by a driver at one end of the cavity and the test microphone was coupled to the other end. The SPL was monitored by a Knowles BT-1753 microphone just in front of the test microphone. The resulting calibration curve is shown in Fig.2-1b. In fact the shape of this follows the free-field curve quite closely over most of the range. SPLs were derived from this coupler curve for the RS/Dural, but taken direct from the free-field sensitivity of the 4155 (without coupler - i.e. SPL at diaphragm) and of the Knowles.

Noise floors were measured with each microphone sealed in this coupler for the dual-lockin analysis system (see Fig.3-2).

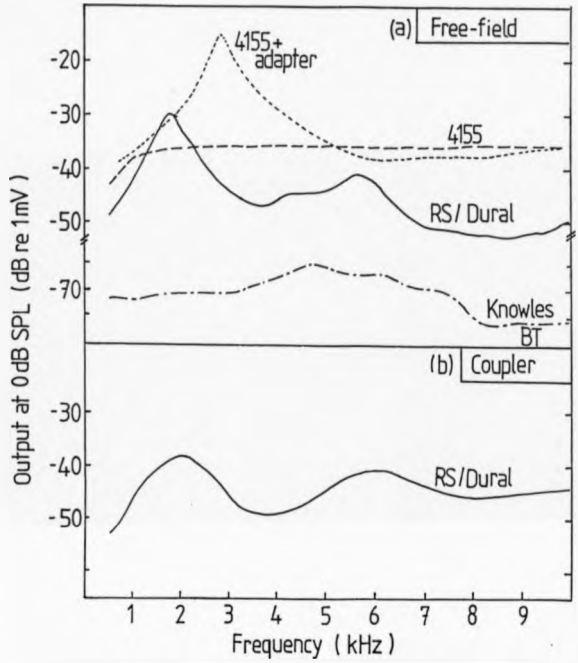

Fig.3-1 Calibration curves for microphone systems. Top: Measured in free field. Bottom: Measured in coupler.

3.1.6 General considerations - the SPL of SOAEs

The SPL of an SOAE is not an absolute invariant quantity, but greatly depends on the conditions of measurement - how and with what it is measured. There are 3 main points to be considered. Firstly, by sealing the ear canal we are deliberately and artificially increasing the SPL. Secondly, the impedance of the microphone as seen from the cochlea is likely to affect the level of an SOAE, and may even determine whether it can sustain itself. Thirdly at higher frequencies SPL may vary with position in the ear canal due to wavelength effects.

1. Effect of sealing the ear canal

If we model the eardrum as a vibrating piston (area A, radius r) at the end of a tube (length l), with the tube open at the far end the pressure produced for low frequencies is approximately $\omega\rho(l+0.6r)v_o$ (Randall 1951), where ω is radian frequency, ρ is the density of air, and v_o is the maximum speed of the piston. Sealing the tube to enclose a volume V increases this to $\rho c^2 A v_o/\omega V$ (c is the velocity of sound, which is greater by a factor $Ac^2/(\omega^2 V(l+0.6r))$. With $A=0.5cm^2$, $V=0.7cm^3$, $l=2cm$, $r=0.4cm$, $c=330\ ms^{-1}$ this is about 20 dB at 1 kHz.

2. Effect of microphone loading back on generator.

The above assumes a constant volume velocity source. In fact the impedance loading offered by the microphone and meatal volume will react back on the generator mechanism and affect its output, - in other words the impedance seen by the SOAE generator will be altered by that of the microphone and ear canal volume. SPLs may therefore differ according to which microphone is used and even between different placements of the same microphone.

3. High frequency effects.

At high frequencies the pressure will no longer be constant throughout the meatus. 3 cm (a typical eardrum-microphone separation) is 1/4 of a wavelength at 3600 Hz. Fortunately this is not a problem for most SOAEs which are below 3 kHz. For higher frequency SOAEs probe tube measurements near the eardrum are really required to give meaningful SPLs, but this technique was not pursued here.

3.2 METHODS

3.2.1 Selection of Subjects

The 17 subjects were friends or colleagues of the author, mainly students, aged between 18 and 45. None of these was aware of any hearing problems, although audiometry subsequently revealed that one (SFi) had a 30-dB bilateral high-frequency loss (probably noise-induced) and another (SD) had a 20-dB unilateral low/mid-frequency loss (cause unknown). They have however not been excluded below; neither was one case with a healed perforation (ML), nor another (AG) who had grommets for childhood middle ear effusion. There was no other known otological history. No subjects were aware of any tinnitus except on an occasional basis, although one (GNW) had become aware of mild tonal tinnitus in quiet following earlier experiments.

3.2.2 Search for SOAEs

A scan for SOAEs was made from 100 Hz to 10 kHz using the 'RS/Dural' microphone with the output fed into the dual lockin amplifier analysis system (§3.1.2). Frequencies and levels were measured 'live', but tape recordings were also usually made for later re-analysis. Pure-tone

Békésy audiograms (air conduction) were obtained for many, but not all subjects (see §4.4 for details of methodology). Subjects were seated in a quiet soundproofed room for the tests.

3.3 RESULTS

3.3.1 Prevalence

53 SOAEs were detected in 9 subjects (14 ears) of the 17 subjects (33 ears) tested - details are given in Table 3-1. 53% of subjects (or 42% of ears) therefore had at least one SOAE. The prevalence is higher in females than males (6 out of 6 females, *cf* 3 out of 11 males): this difference is statistically significant at the 1% level (Bailey 1959, p.61, exact test for 2x2 tables, double-tailed), as it is in terms of ears (11 out of 12 female ears, *cf* 3 out of 21 male ears).

3.3.2 SOAE frequencies and levels

The frequency and amplitude of the SOAEs detected is illustrated in Fig.3-2 (dots), relative to the noise floor of the BS/Dural microphone system. (Crosses and plus signs represent SOAEs found in tinnitus subjects - §4). The frequency range is from 684 Hz to 6187 Hz, and up to 24 dB SPL in level, with the vast majority, as usual, around 1-2 kHz.

Two ears contained SOAE components (f_1, f_2, f_3) in the relation $f_1=2f_2-f_3$, discussed below (§3.4.3).

TABLE 3-1
PREVALENCE OF SOAEs IN NORMAL SUBJECTS

SUBJECTS: 9 / 17 (53%) (MALES 3 / 11; FEMALES 6 / 6)

EARS 14 / 33 (42%) (MALES 5 / 17; FEMALES 9 / 16)

No SOAEs detected in:
JC L/R SD L/R SG L/R DL L/R HL L/R MM L/R
GS L/R DW L/R

Subject		Frequency (Hz)	Level (dB SPL)	Subject		Frequency (Hz)	Level (dB SPL)
AC	L	-		JS	L	not tested	
	R	1620	+1		R	1360	-2
		1883	+1			1592	-7
						4808	+5
CD	L	-					
	R	1540	+12	AS	L	1060	-3
		1972	+1			2390	-10
		2257	-15		R	1184	+1
		3441	+4			1713	-4
						2870	-1
SFi	L	-				3220	-2
	R	1490	+7				
				HW	L	684	+5
AG	L	1369	-2			1028†	-4
		1648	+1			1183†	+11
		1860	-10			1335†	-7
		1999	0			1566	-1
		2430	-17			1757	-9
		3219	-6			1909	+5
	R	991	+2		R	986	+24
		2813	-11			1378	+21
		3179	+9			1786	+21
IML	L	954†	0	GMW	L	3089	-5
		1204†	-1			3251	-7
		1452†	+10			3962	+8
		1656	-9			4765	+4
		1784	-10			5157	-6
	R	1255	+8		R	2611	+19
		1348	+9			3142	+4
		1654	-1			3819	+5
						4630	0
						6187	-5

† These SOAEs are in the relation $f_1=2f_2-f_1$

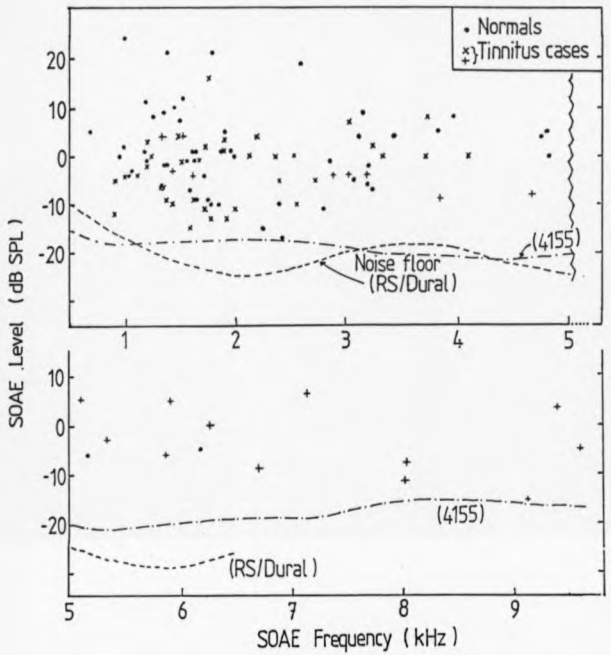

Fig.3-2 Distribution of SOAEs detected in all subjects. **Dots:** normal hearing subjects (RS/Dural microphone). **Crosses:** tinnitus subjects (RS/Dural microphone). **Plus signs:** tinnitus subjects (4155 microphone). The noise floors are also shown.

3.3.3 Hearing threshold levels at SOAE frequencies

The HTL at the SOAE frequency is given in Table 3-2 for those cases for whom Békésy audiograms were available. The range is from -12 to +18 dB HTL, with mean at 0.2 dB.

TABLE 3-2

HEARING THRESHOLD LEVEL AT SOAE FREQUENCIES

	No of SOAEs	
-15 to -11 dB HTL	1	
-10 to -6 dB HTL	7	
-5 to -1 dB HTL	9	
0 to +4 dB HTL	5	Mean = 0.2 dB HTL
+5 to +9 dB HTL	5	S.D. = 7.6 dB
+10 to+14 dB HTL	1	
+15 to+19 dB HTL	2	

(No data on 4 subjects)

3.4 DISCUSSION

3.4.1 Prevalence of SOAEs in the normal-hearing population

In this small sample of 17 subjects, SOAEs were found in 53% (42% of ears). A summary of other data in the literature is given in Table 3-3.

TABLE 3-3

SOAE PREVALENCE IN NORMAL SUBJECTS

Author	Material	In Subjects (N sample)	In Ears (N)
Wilson 1980c	Adults	-	25% (40)
Zurek 1981	Adults	48% (27)	37% (54)
O'Brien 1981	Adults	11% (80)	5% (160)
Schloth 1982	Adults	44% (64)	34% (128)
Hammel 1983*	Adults	34% (?)	21% (?)
Rabinowitz & Widin 1984	Adults	- (12)	42% (24)
Wier et al 1984	Adults	38% (47)	27% (92)
Strickland et al 1985	Children	40% (50)	31% (100)
" " "	Infants	38% (21)	26% (38)
Probst et al 1986	Adults	- -	43% (12)
Cianfrone & Mattia 1986	Adults	31% (52)	26 (104)
This work	Adults	53% (17)	42% (33)

*as reported by Strickland et al (1985)

Considering the variety of microphone and analysis systems used (and hence noise floors), the agreement between these figures is quite good. The figures in the sample here are thus similar to, if slightly higher than, the median of the other data. SOAEs thus seem to occur in about 1/3 to 1/2 of all normal ears.

3.4.2 Sex difference in prevalence

A significantly higher prevalence of SOAEs in women was found here, and is supported by other data. Zurek's (1981) data, whilst not statistically significant, were biassed in the same direction (females 57%, males 20%, normal hearing sample). The same applies to Hammmel (1983, as reported in Strickland et al 1985) (females 40%, males 28%). Strickland et al's (1985) data did reach statistical significance for their sample of children (females 52%, males 24%) and showed a non-significant difference (females 50%, males 14%) for their infants. The reasons for this sex difference are not yet clear. There is however now considerable evidence of sex differences in various other aspects of hearing function (reviewed in Dengerink et al 1984). One possibility is that these are due to hormonal differences. There is some evidence for such hormonal influences in Wilson's (1985, 1986) data on SOAE frequency changes through the menstrual cycle. Although these were found to be significantly correlated with body temperature, diurnal frequency changes were not, and other data (Zurek 1981, Wilson 1985, Wit 1985) show no direct effect of temperature on SOAE frequency: therefore if temperature is not mediating the frequency changes, hormonal levels may be. Wilson (1985) has speculated that these may act centrally, affecting the cochlea through the efferent system (which may be responsible for overall control of the active mechanisms -§1.1.4). However, turning back to the question of SOAE prevalence, there are no reports of variation in the *number* of SOAEs through the menstrual cycle, but this has not been directly investigated and would seem to be a worthwhile line of study.

No significant difference in prevalence between left and right ear has been found here or elsewhere.

3.4.3 Inter-related SOAEs

The triads of SOAE components in $f_1=2f_2-f_3$ frequency relation were also found in one of the tinnitus subjects (S4), and in the data of Glanville et al (1971), Wilson and Sutton (1983), and explicitly noted by Burns et al (1984), Strickland et al (1985), and Jones et al (1986). Presumably the f_2 and f_3 SOAEs behave as the 'primaries' and interact on the basilar membrane (just as would two external tones) to give rise to a $2f_2-f_3$ (CDT) travelling wave which travels to its own place (Kim 1980) and there excites the OAE generating mechanism, causing a CDT SOAE. As would be expected one of the primary SOAEs in each triad was relatively strong. If this model is correct one would expect that the CDT SOAE would behave differently from other SOAEs under suppression, being affected by tones at the primaries as well as its own frequency. There is some evidence of this (Burns et al 1984). Other differences in behaviour would be expected if, as Wilson (1977) suggested, the CDT is not propagated along the BM as a normal tone but via another structure. The frequency ratio of the primary SOAEs (f_3/f_2) was in the range 1.2 to 1.3, which is about the ratio for which maximal CDT EOAEs are obtained (e.g. Wilson 1980e).

Another type of interrelation is where an ear occasionally switches between 2 distinct sets of SOAEs (Wilson and Sutton 1983, Burns et al 1984, Jones et al 1986): these were termed by the latter authors 'linked noncontiguous SOAEs'. However whilst some SOAEs here were

clearly very variable in level, the narrow band analysis methods used did not easily allow study of covariation of different components.

3.4.4 Are SOAEs associated with truly normal hearing or minor pathology?

The problem as to whether SOAEs are naturally-occurring epiphenomena of truly normal hearing, or are rather the symptom of minor (perhaps sub-clinical) cochlear damage, will be discussed fully in §4.6.4. For the moment it may be noted that in these subjects the mean threshold at SOAE frequency was 0 dB HTL and was never worse than 18 dB HTL. Neither was there any consistent evidence of SOAEs being associated with local threshold notches etc.

3.4.5 Would SOAE prevalence be higher with a more sensitive detection system?

By definition any SOAEs present at levels below the noise floor of the microphone/amplifier system remain undetected, even if noise due to breathing, blood-flow etc is absent. An interesting question is, if we could push this floor further down would we then discover many more SOAEs, and would the prevalence approach 100% (rather than just at 30-50%) among normal subjects? The alternative, consistent with current ideas of SOAE generation, is that whilst the existence of an SOAE is determined by the amount of feedback, its level is determined by the saturation characteristics of the generator. This would imply that there is a minimum SPL for each SOAE to sustain itself, and one would not expect many more lower-level SOAEs.

Light may be shed on this question by examining the distribution of SOAE SPLs relative to the noise floor. If it were true that there were many 'hidden' SOAEs below current noise floors, with SOAE incidence increasing with decreasing SPL, one might find a crowding of SOAEs near the floor and a one-sided distribution tailing off towards higher SPLs. On the alternative 'minimum self-sustaining SPL' hypothesis however, the distribution would peak at some SPL, perhaps considerably above the noise floor.

Table 3-4 shows the height of the SOAEs above the noise floor; the range is 7 dB to 42 dB, with a mean at 22 dB (SD=8.0). A chi-square test shows that the distribution is not significantly different from a Gaussian (χ^2=2.9, DF=4). Pooling in the data for the SOAEs detected in the tinnitus subjects (S4) (crosses/plusses) gives a mean at 19 dB re noise floor (SD=7.9); again the result is consistent with a normal distribution (χ^2=6.8, DF=5). These distributions therefore peak well above the noise floor.

TABLE 3-4

LEVELS OF SOAEs RELATIVE TO NOISE FLOOR

Height above floor	No of SOAEs: Normals only	Normals & tinnitus
0-4 dB	0	2
5-9 dB	1	4
10-14 dB	7	24
15-19 dB	15	30
20-24 dB	12	20
25-29 dB	9	19
30-34 dB	5	7
35-39 dB	1	1
40-44 dB	3	4

One factor not taken into account above is that it was found experimentally that an injected steady tone generally needed to be about 5-8 dB above the absolute noise floor to be reliably detected, and this would have to be higher for fluctuating tones. Since many SOAEs do fluctuate this makes interpretation less straightforward.

Other workers' results appear contradictory at first sight. For example Zurek (1981) shows a distribution peaking at +15 dB above his noise floor and few below that peak, whilst Schloth (1982) shows a decided

clustering near the noise floor. These differences may however be due to Schloth's using a time-averaging FFT analyser: here the SPL measured for an intermittent and/or frequency-unstable SOAE (often the case) is an average value below its SPL when 'on' (the average depending on the exact time course of its fluctuations). The analysis method used in this thesis and by Zurek on the other hand records the 'on' (peak) SPL of the SOAE. Data collected using time-averaging may therefore be misleading in looking at this question. Recently however Strickland et al (1985) have published results from short-term FFTs showing a distribution peaking well above (about 15 dB above) minimal detectability.

The balance of evidence therefore suggests that the number of SOAEs found would not increase much with lower noise floors.

3.4.6 Why are most SOAEs inaudible?

Most SOAEs remain unheard by their owners. This was also true here, although some subjects did become aware of them after listening to a tape recording. The SPL of an external applied tone to give the same cochlear excitation as an SOAE can be estimated by correcting for the middle ear transmission properties (Kemp 1980, after Zwislocki 1962, Schloth 1982) to be from 10-40 dB greater than the SOAE meatal SPL according to frequency. One would therefore expect this correction to bring many SOAEs above the audibility threshold (typically 0-10 dB SPL), so that their inaudibility seems at first surprising.

Two factors may be important in explaining this. One is that such low level tones are readily masked by ambient noise, so that only in very quiet rooms is there the opportunity to hear them. The other is adaptation: evidence for this comes from Schloth (1982) (also in Schloth and Zwicker 1983) who found that a normally unheard SOAE could be made audible by shifting it (by ear canal pressure) by about 10 Hz. This suggests the hypothesis that the ear adapts to a steady emission, and a sensation is only produced when the steady state is altered. Clearly this could not apply to SOAEs which fluctuate or switch in frequency: one would expect therefore that such SOAEs would be heard more than steady ones.

4. SPONTANEOUS OTO-ACOUSTIC EMISSIONS IN A TINNITUS POPULATION

4.1 INTRODUCTION AND REVIEW

SOAEs have been shown to be present in 30-50% of normal human ears (see §3). Among those who have SOAEs, some are (or can become) aware of them as faint tonal tinnitus (Kemp 1979a, Wilson 1980c, Zurek 1981). However awareness of this often only begins after the SOAE is discovered and the owner's attention has been directed to it (e.g. on a tape recording), it is usually only heard in quiet surroundings, and is rarely troublesome. The question arises as to whether SOAEs might be implicated in any cases of more troublesome tinnitus, as seen widely in ENT departments.

Tinnitus ('ringing in the ears', 'head noises', etc) has been recognised since antiquity, and affects a considerable proportion of the population. To many sufferers it is a severe problem causing great distress. There has been much investigation and there is a voluminous literature, but little of it has advanced our understanding or helped sufferers (for reviews see Hazell 1979, Goodwin 1979). The main reason for this lack of progress is probably that tinnitus is not a disease but a symptom which may arise from pathology of almost any site along the auditory pathway. Thus there are almost certainly many different types of tinnitus, arising from different mechanisms, and findings for one source of tinnitus may not hold for any other. The best way forward would seem to be to try to identify distinct types of tinnitus with different sources, which would allow the properties of each type to be studied and specific strategies developed for management of each sub-

group of sufferers. In general this is difficult because sites of lesion can usually only be tentatively identified owing to the crudity of diagnostic procedures, but SOAEs give a possible new means of exploration into cochlear functioning.

To investigate the possibility that those with SOAE-related (cochlear mechanical) tinnitus might form such a distinct sub-group among a clinical tinnitus population, we have examined a group of sufferers. In all these the complaint predated any testing, thus excluding those not-uncommon subjects who become aware of SOAEs as tinnitus only after their attention is directed to it: falling into this category are 2 subjects (GNV and EFE) who were reported in an interim report of this work (Wilson and Sutton 1981) as having acoustic correlates of tinnitus (GNV was however included in §3).

4.1.1 Definition of terms

Tinnitus is the condition where a sound is heard without any relevant external stimulus, and is also used to describe the sensation itself. In the past the phenomenon in which a sound is detectable to an observer emerging from the subject's ear (unaided or with a microphone) or head (by auscultation using a stethoscope) has been called 'objective tinnitus', whether or not the subject was aware of the sound. This usage is now deprecated, and in this thesis the term 'tinnitus' is reserved for the subjective sensation of sound (and the general condition) and does not refer to any externally detected correlate. These latter are described as SOAEs, bruits, etc, as appropriate (Evered and Lawrenson 1981, p.300).

The terms *'tinnitus sufferer'* and *'tinnitus population'* are here restricted to those who complained of it before the tests, and whose awareness of their tinnitus was sufficiently strong for them to have come forward in response to adverts, or consulted their GP and an ENT specialist.

4.1.2 Tinnitus and Pathology

Tinnitus can arise from damage or disturbance to the normal hearing mechanisms at almost any point along the auditory pathway. It may be brought on by trauma or insult (through noise or head injury), various drugs (e.g. ototoxics, aspirin), and almost all diseases causing deafness or damage to the ear, both conductive and sensorineural (e.g. otosclerosis, ossicular discontinuity, presbyacusis, Menières Disease, acoustic neuroma), as well as many causing damage to the central nervous system (e.g. syphilis). Other likely causes include patches of demyelination and abnormalities in the blood supply of the stria vascularis. However patients with no other signs of pathology or audiological abnormality may also have tinnitus (although this does not rule out undetected sub-clinical damage to peripheral hearing mechanisms).

In practice the site of lesion cannot be pinpointed in many cases, and the very concept of a localised lesion may sometimes be misleading. For example sectioning the VIIIth nerve often fails to alleviate - and sometimes worsens - tinnitus which appeared to be peripheral (House and Brackmann 1981). It may be that some initially peripheral tinnitus can 'travel up' the auditory pathway and become imprinted centrally. This

is not so surprising if one thinks of the auditory mechanism as a unified whole in which activity at peripheral and central levels is interdependent, rather than as simply passing a signal sequentially through various processing stages: on this view nerve section may seriously alter the activity in the central sites. (See also discussion Evered and Lawrenson p.279-286).

4.1.3 Properties of peripheral tinnitus

Tinnitus - even that of peripheral origin - often does not behave like a 'real' external sound. For example, in masking studies it is often found that tonal tinnitus may be masked as easily by remote frequencies as nearby, if it can be masked at all (Feldmann 1971, Shailer et al 1981, Burns 1984), although in some cases the same frequency tuning as for an external tone is found (Shailer et al 1981). Similarly whilst a physical wide-band noise cannot be masked by a pure tone, this can occur with tinnitus (Feldmann 1971). Contralateral masking is also often effective with tinnitus (Feldmann 1971, Tyler and Conrad-Armes 1980). Pitch-matching to tonal tinnitus may show much more variation than matching to an external tone in the same subjects (Burns 1984). Also unlike a 'real' tone, only rarely are beats heard between a tonal tinnitus and a closely-tuned ipsilateral pure tone (Vogel 1931, Evered and Lawrenson 1981, p.281).

From this it may be concluded that not all tonal peripheral tinnitus has the same origin, and that the pattern of neural activity associated with some tinnitus is very different from that associated with an external tone of the same apparent pitch.

4.1.4 Theories of origin of peripheral tinnitus

A number of suggestions and analogies have been made as to the origins of peripheral tinnitus. The most intuitively obvious hypothesis is that tinnitus results from an increase in neural activity at some site, due to for example 'hyper-irritable' haircells (Stevens and Davis 1938), with the tinnitus perhaps corresponding to the characteristic frequency of the affected site, although the neural excitation would be unlike that for a real tone. Similarly Fowler (1940), and Davis and Silverman (1970), drew an analogy between noise-induced tinnitus and the itching or tingling caused by skin bruising. Supporting this, audiometric threshold notches are sometimes found at the tinnitus pitch (e.g. Douek and Reid 1968), but there has been much (unresolved) debate about whether such notches might be due to masking by the tinnitus rather than being 'true' hearing loss. Fowler (1940) also noted the frequent correspondence between tinnitus pitch and a sharp drop-off in the audiogram, and suggested this was due to a border effect between normal and reduced activity, analogous to putting one's leg in a hot bath where the strongest sensation is felt at the interface. This was also suggested by Kiang et al (1970), who found reduced or eliminated spontaneous neural activity in the region of threshold loss induced by ototoxics, with very sharp transition between normal and damaged regions. On the other hand Evans et al (1981) found *higher* spontaneous discharge rates after salicylate administration: this appeared to be true for all fibres, irrespective of characteristic frequency, so ruling out a border effect for this pathology. They therefore concluded that salicylate-induced tinnitus was probably associated with increased cochlear activity. Consistent with this, Jastreboff et al (1986) have

recently reported higher spontaneous activity in single units in the inferior colliculus following salicylate administration (in guinea pig).

Gold's (1948) speculations about active processes led him to suggest that some tinnitus might be self-oscillation of an active feedback mechanism, anticipating modern ideas of SOAEs. Another mechanism, proposed by Tonndorf (1980), was of partial decoupling of hair-cell stereocilia from the tectorial membrane, which would raise their random noise input to the haircell and hence cause an increase in haircell activity (and tinnitus). Møller (1984), emphasising the importance of temporal coding, suggested that any breakdown of insulation between neighbouring fibres would cause 'crosstalk' and correlation of these fibres' spontaneous activity, leading to tinnitus.

Clearly these different hypotheses and origins need not be mutually exclusive - there are certainly several types of peripheral tinnitus.

4.1.5 Descriptions of tinnitus

The sounds heard by patients with tinnitus are extremely varied, from simple tones to highly complex combinations, often described in graphic terms. They may be constant or intermittent, steady or pulsatile or varying, loud or quiet, and bothersome or not. Whilst it is often difficult to translate the subjective descriptions into more universal categories, it appears that the most common forms are tonal, tonal with a background of noise, and broad-band noise (Heller and Bergman 1953, Vernon and Meikle 1981).

4.1.6 Management

Tinnitus often causes severe distress, loss of ability to concentrate, sleep disturbance and depression. Management is initially reassurance that there is no serious underlying pathology, and this is sufficient for many; for others, a masking device (usually producing a wide-band noise), or hearing aid, or a combination, is often successful (e.g. Vernon and Meikle 1981). Success has also been claimed for many other treatments, including biofeedback (House 1981), although many studies could be criticised for failing to control for the known strong placebo effect.

4.1.7 Prevalence of Tinnitus

The prevalence found for tinnitus by a survey depends on what question is asked, how, and where. If placed in a quiet room and invited to listen carefully the vast majority of normal-hearing individuals will report some tinnitus (Heller and Bergman 1953). Similarly nearly everyone has experienced transient tonal tinnitus at some time. Therefore most surveys are carried out by questionnaire and exclude this trivial degree of tinnitus. Those carried out by the Institute of Hearing Research (1981) and the Office of Population Censuses and Surveys (1981) have shown that (after exclusion of those of short transient tinnitus or temporary noise-induced tinnitus etc), the prevalence of tinnitus in the general population is about 15-17%, still remarkably high. Whilst this figure still includes many cases in whom the condition would be considered sub-clinical, about 2% had continuous tinnitus (OPCS) and about 1% (IHR) reported severely annoying tinnitus. There is a hard core of about 0.5% (IHR) whose life is severely

affected. Prevalence increases with age and is higher in manual than in non-manual social-economic groups (which can be accounted for, at least in part, by the association with hearing difficulty). Tinnitus is slightly commoner in women than men (IHR 1981, OPCS 1981). Whilst tinnitus can occur either transiently or permanently in individuals without any obvious ear pathology or hearing problems, it is also a very common accompaniment to deafness - according to Fowler (1944) 86% of patients with ear disease also had tinnitus, whilst the OPCS survey found that tinnitus was reported by 33% of those with self-reported hearing difficulties.

4.2 HYPOTHESES

A population of tinnitus sufferers as defined above has been investigated with the primary aim of seeing if ear canal recording would reveal SOAEs that could be held to account for all or part of the tinnitus sensation. (Other objective sources of tinnitus do not concern us here and no such cases were seen.)

The hypotheses being tested here are, explicitly:

(1) Some cases of clinical tinnitus are caused by SOAEs.

In these cases we therefore expect one or more SOAEs to be found which could account for the tinnitus sensation. Because presumably the cochlear excitation pattern caused by an SOAE is similar to that caused by a pure tone of that frequency, in such cases we would expect the tinnitus to be pure-tonal, closely pitch-matched to the SOAE frequency and localised to that ear. Where apparent acoustic correlates were found, confirmation was sometimes sought by other methods: e.g.

subjective recognition of the SOAE, and the irregular beats heard when an SOAE interacts with an external tone (Kemp 1979b, Wilson 1980c).

With the techniques used here only correlates of tonal tinnitus can be sought. Whilst it is conceivable that some wider-band tinnitus might have an acoustic source (Kemp 1982) and some SOAEs have wider bandwidths than others, (Kemp 1981), more sophisticated methods (e.g. correlation between 2 microphones) would be needed to extract these from electrical noise.

(II) SOAEs will not occur at frequencies where there is significant hearing damage.

It is believed that SOAEs require the physiologically-vulnerable active process to be functional at the place corresponding to their frequency (see §1.2.8). We take 'significant' damage to be indicated by a hearing threshold of greater than 20 dB HTL. It is however recognised that hearing threshold is not the only, or most sensitive, measure of integrity of cochlear function (frequency resolution may be more sensitive - Pick and Evans 1983): however where threshold is abnormal other measures are also likely to be so.

Loudness and annoyance measurements were also made to try to place any positive acoustic correlate cases in the spectrum of severity.

4.3 SELECTION OF SUBJECTS

49 subjects were seen, whose awareness of tinnitus was sufficently strong for them to come forward in response to adverts etc, or to

consult their GP and an ENT specialist. Whilst, from our knowledge of SOAEs in normal-hearing individuals, acoustic correlates of tinnitus appear most likely *a priori* to be found in subjects with unilateral tonal tinnitus and with good hearing, such subjects are rare and the sample was not restricted to these: in fact subjects presented with a wide range of aetiologies and hearing losses.

There are two distinct sources by which subjects were obtained. 21 subjects were referred from the North Staffordshire Hospital Central ENT Outpatients' Department by the consultant ENT surgeons and their colleagues. All subjects were made aware that this was basic research and could not expect any direct personal benefit, and the study was approved by the hospital ethical committee. This is not in any sense a random sample of tinnitus patients in an ENT Department, because of

(1)intentional bias: we emphasised to the consultants that we were particularly, although not exclusively, interested in tonal tinnitus cases with good hearing, and who were likely to be cooperative and competent observers and experimental subjects;

(2) self-selection: the individuals having consulted their GP and an ENT surgeon, and then volunteered to participate in research.

(3) other factors, which almost certainly played a large part in the consultants decision as to who refer on - for example psychological suitability and nuisance value. There is often, justifiably, a fear that over-investigation of a tinnitus can aggravate the problem.

Within this group, all were invited for tests, although a few (6) withdrew, were unavailable, or did not attend.

The other 28 subjects either (a) replied to adverts in the University newspaper and on campus noticeboards, or (b) volunteered after items on local radio or after a talk to a local branch of the British Tinnitus Association, or (c) were known to colleagues as sufferers. At first all volunteers from these sources were tested, but later some preselection was carried out by means of a questionnaire (Appendix 2) sent out to them. Of these 28 subjects, 18 were seen unscreened and 10 after this questionnaire screening. 7 other subjects dropped out before testing and a further 16 were not invited.

In practice there is no clear distinction between these 2 sub-groups of subjects (the 'ENT referrals' and the 'volunteers') since at least 15 of the 'volunteers' had also seen an ENT specialist. No significant differences were found in the results and they are therefore treated as one group below.

4.4 TEST PROCEDURES

A full picture of the otological history of each subject (including noise and physical trauma, earaches, operations, etc) was first obtained along with a description of the tinnitus and the factors affecting it, usually by questionnaire (Appendix 2). Because time was often limited and every case offered individual features, the battery of tests employed was not the same for all subjects, and sometimes not as complete as one would wish. However in most cases, the following tests were carried out:

Otoscopic examination and tympanometry

Air-conduction Békésy pure-tone audiometry

Tinnitus pitch and loudness match

Scan for SOAEs

Minimal masking levels by tones, narrowband or wideband noises, and residual inhibition test

Loudness and Annoyance ratings (7-point scales)

Each of these will be described in more detail.

Otoscopic examination and tympanometry

An attempt was usually made to visualise the eardrum to verify that the ear canal was not completely blocked with wax (which would abolish or reduce the amplitude of SOAE recordings), but detailed inspection of the tympanic membrane was not generally attempted. In some cases there were known perforations or mastoid cavities. Tympanograms were recorded on most subjects (probe frequencies of both 220 Hz and 660 Hz) on a Grason-Stadler 1723 middle ear analyser to reveal cases of middle ear pathology or damage which would render any cochlear SOAEs undetectable.

Air conduction (Békésy) audiograms

Air conduction pure-tone audiometry was carried out using a Rion AA-38 Békésy audiometer with Rion headphones (later TDH-49P earphones), recording from 100 Hz to 10 kHz. The frequency sweep rate was about 3 min/octave and the attenuation change rate was 4 dB/s. The self-reported better ear was tested first. Where necessary wide-band noise was used to mask the contralateral ear, the level being set equal to that of the hearing threshold in the test ear. This is a crude masking method which may lead to error in some cases; therefore in those cases

with interaural differences of more than 40 dB, results were checked with the audiograms carried out in the hospital Audiology department using the more rigorous 'plateau' masking method with narrow-band noise.

The audiometer was recalibrated for TDH-49P earphones on an IEC 318 coupler (to BS 2497) midway through the study. Comparisons showed slight errors in the previous calibration (between 2 and 7 dB according to frequency). For simplicity, a uniform correction of 5 dB has been applied to these earlier thresholds.

Pitch and Loudness Match

Pitch-matching of the dominant component of the tinnitus was performed contralaterally (Vernon and Meikle 1981) through Sennheiser HD414 earphones, except where hearing was very poor in that ear. Where tinnitus was bilateral each ear was matched separately. The source was a continuously-variable oscillator, controlled by the experimenter. Because the loudness of the matching tone may affect its perceived pitch (particularly where there is a hearing loss) pitch and loudness matches were interleaved in order to minimise this (Vernon and Meikle 1981), and keep the tones in the range 5-30 dB SL (sensation level, i.e. dB above threshold).

Pitch-matching was by the method of limits (the subject repeatedly being asked to say whether the tone was 'too high' or 'too low' relative to the tinnitus pitch). Having identified an initial pitch match, a loudness match was found and expressed in dB SL. Checks were

then made for octave confusion both above and below the pitch match. Where the octave was preferred, a further octave in the same direction was offered for consideration, until the subject clearly indicated his preference. At least 2 repeats of the matching procedure were usually performed, starting anew from a remote frequency. In addition some subjects were allowed to adjust the frequency and level themselves (method of adjustment) but this was always followed by a repeat match carried out by the experimenter.

Some subjects felt their tinnitus could not be matched to a pure tone or were dissatisfied with the final tonal match; they were offered narrow-band noises (10 to 300 Hz wide) or failing that, wide-band noises with different degrees of spectral slope.

It is known (Tyler and Conrad-Armes 1983, Burns 1984) that pitch-matching to tinnitus is a difficult task and may often show large variation. In this sample some subjects' pitch matches were very variable, and did not stabilise to an asymptotic value despite many repeats. Such findings may indicate either (a) that a pitch cannot be properly assigned to the tinnitus, or (b) that there is diplacusis or pitch distortion confounding the procedure, or (c) that the subject is very poor in this type of judgement.

Scan for SOAEs

The dual-lockin analysis system (described in §3.1.3) was used to search for SOAEs. The microphone (usually RS/Dural) was inserted into the meatus until it was secure and apparently making a reasonable seal.

The subject was asked to keep as still as possible while the scan was made. The frequency range scanned was from 100 Hz up to either 5 kHz or 10 kHz, with the RC time constant of the averager at 30 ms (8 Hz bandwidth), with particular attention paid in the region of the tinnitus pitch match. Tape recordings were also made for later analysis, perhaps with higher resolution.

In a few cases a search was made for SFEs (as described in §3.1.4) using low-level (10 or 20 dB SL) probe tones swept slowly over the frequency range of interest.

Masking and Residual Inhibition tests

Minimal masking levels (Vernon and Meikle 1981) and thresholds were measured for various tones and/or narrow-band noises, and white noise, presented via headphones to the ipsilateral ear, often allowing classification into Feldmann's (1971) categories. Whichever masker (white noise or narrow-band noise at the pitch match) seemed the most effective was then presented at 10 dB above the minimal masking level for 1 minute to test for residual inhibition (Vernon and Meikle 1981), the subject being asked at the end of this time if there was any change in the tinnitus loudness or quality. The results of these tests allowed us to report back to the referring specialist on the possible usefulness of a tinnitus masker.

Loudness and Annoyance ratings

As described above a loudness match was obtained to an external pure tone. However it is a common finding (e.g. Goodwin 1979, Vernon and

Meikle 1981) that such sensation level matches rarely exceed 20 dB SL, and correlate poorly with subjective loudness, due to recruitment etc. Therefore subjects were also asked to rate the average loudness of their tinnitus on a 7-point scale (1="very quiet", 7="very loud"); and also how annoyed they were by it generally (1="not at all annoyed", 7="very annoyed").

4.5 RESULTS

4.5.1 The material

The 49 subjects comprised 35 males and 14 females, with ages ranging from 17 to 84 years. Details of each subject and the individual test results may be found in Appendix 3.

4.5.2 Aetiology

Table 4-1 lists the presumed likely aetiology of the tinnitus (or the event believed by the subject to have precipitated it) as judged from the clinical history and audiometry. In some cases there was an identifiable disease condition; in others the subject was sure that the onset of their tinnitus was related to some particular event, although objectively this might be coincidental. Therefore, despite the full history taken, the given aetiology remains speculative in many cases (not unusually for this condition).

3 cases had perforations coexisting with the main aetiology listed: CD (same ear as tinnitus), RDo and DK (contralateral ear).

TABLE 4-1

AETIOLOGY

Noise Trauma	(chronic)	10	(PA, FB, JB, IB, CC, KC, HE, JvH, EJ, CJo)
	(acute)	3	(CG, JWM, DW)
Presbyacusis		5	(GB, PB, RUD, GG, JHo)
Viral/Vascular		5	(DG, MH, EL, ITM, TR)
Head Injury	(sensorineural)	3	(IC, RHo, PP)
	(conductive)	1	(CJa*) *ossic. discontinuity
Menière's disease		3	(AB, RDo, TAS)
Mastoidectomy (radical)		2	(WB, JR)
Fenestration		1	(AF)
Cleft palate surgery		1	(CD) (also perforation)
Tooth extraction		1	(KP)
Wax removal		1	(JD)
Furosemide		1	(MWh)
Rubella		1	(NM)
Middle ear infection		1	(AR)
Anxiety/psychological		2	(BB, AM)
Temporo-mandibular joint dysfunction		1	(RHe)
Unknown		7	(TAB, Bo, DK, ML, AP, TVS, MWa)

4.5.3 Hearing threshold levels

Each subject's overall hearing loss has been classified on the basis of the 6-frequency average hearing threshold level (HTL) over 0.25, 0.5, 1, 2, 4, 8 kHz, in the (stronger) tinnitus ear, or in the better-

hearing ear where tinnitus was bilateral. (Thresholds exceeding 95 dB were assigned this value.) The breakdown is given in Table 4-2:

TABLE 4-2

MEAN HTL IN TINNITUS EAR

< 20 dB	17	(PA, TAB, PB, RUD, CG, JvH, RHe, RHo, EJ, JM, ITM, AM, KP, PP, AR, TVS, MWa)
20 - 40 dB	15	(AB, GB, FB, CC, IC, KC, RDo, DG, GG, JHo, CJa, MM TAS, MWb, DW)
40 - 70 dB	9	(JB, IB, CD, JD, HE, MH, CJo, DK, EL)
70 - 95 dB	2	(AF, TR)
> 95 dB	2	(WB, JR)
No data	4	(BB, Bo, ML, AP)

However, many of the 17 in the '< 20 dB' category had audiometric abnormalities such as local notches, high-frequency droops, etc, so that they cannot be considered as forming a clinically-normal sub-group. In fact only 1 subject (KP) meets 'strict clinical normality' criteria defined as:

> HTL < 20 dB at all frequencies (continuous scan) between 250 Hz and 8 kHz in either ear;
>
> and interaural difference at any octave frequency < 20 dB.

4 others (TAB, AM, ITM and MWa) fell just outside. 39 further cases had a sensorineural hearing loss (3 with coexisting eardrum perforations and 3 with absent middle ears), and 2 (RHe and CJa) had pure conductive losses. No audiometric data were available for 3 subjects (BB, AP, Bo)

(the consultant's verbal report of the loss was available for the fourth 'no data' case of Table 4-2).

For the unilaterals and those with tinnitus stronger in one ear, hearing levels in the tinnitus ears were almost always worse than in the non-tinnitus ear (Table 4-3). This confirms that tinnitus is mostly a symptom associated with deafness.

TABLE 4-3

DIFFERENCE IN MEAN HTL BETWEEN TINNITUS AND NON-TINNITUS EAR

Pure unilateral cases

WORSE HTL IN TINNITUS EAR :	21 cases	Mean diff. = >24 dB *
BETTER HTL IN TINNITUS EAR :	1 case	

(Data incomplete in 4 cases)

Tinnitus stronger one side

WORSE HTL IN TINNITUS EAR :	9 cases	
BETTER/SAME HTL IN TINNITUS EAR :	6 cases	Mean diff. = >6 dB *

(Data incomplete in 1 case)

*'>' because some thresholds were beyond range of audiometer)

Probably the most relevant measure for maximising the chances of finding SOAE-related tinnitus is a low HTL at the frequency of the tinnitus pitch match. Table 4-4 shows that, based on the best estimates of tinnitus pitch, 11 subjects had HTLs better than 20 dB in the dominant tinnitus ear.

TABLE 4-4

HTL AT TINNITUS PITCH

< 20 dB	11	(FB, TAB, PB, RUD, CG, RHe, JH, AM, HH, KP, MWa)
> 20 dB	35	(all others)
No data	3	(BB, Bo, AP)

4.5.4 Localisation of tinnitus

Breakdown of the tinnitus localisation is given in Table 4-5. 26 subjects identified their tinnitus as being purely unilateral, while the other 23 had some bilateral involvement. Of these latter however, most could identify one ear as having the stronger sensation, so that 21 had tinnitus purely left or left-louder, and 21 purely right or right-louder.

TABLE 4-5

LOCALISATION OF TINNITUS

PURE UNILATERAL	26	(BB, WB, AB, FB, JB, TAB, PB, CC, IC, RUD, CG, DG, JvH, RHe, MH, CJa, CJo, ML, JH, KP, AP, PP, TR, JR, TAS, MVh) [15 Left, 11 Right]
ONE EAR STRONGER	16	(PA, GB, Bo, IB, KC, CD, JD, RDo, AF, JH, EJ, DK, ITM, AR, TVS, DW) [6 Left, 10 Right]
BOTH ABOUT EQUAL	4	(HE, GG, AM, MWa)
IN HEAD	3	(RHo, EL, HH)

4.5.5 Tonality

The tonality of the tinnitus sensation was determined both from the initial description and from the degree of satisfaction expressed with the final pitch match. In Table 4-6 'pure-tonal' means tinnitus was either pure-tonal or contained tonal component(s).

TABLE 4-6

TONALITY OF TINNITUS

PURE-TONAL	26	(PA, WB, Bo, IB, CC, KC, CD, JD, RDo, HE, CG, DG, GG, JvH, RHo, CJa, EJ, CJo, DK, JM, MM, AM, KP, AR, MVh, DV)
IMPURE TONE OR NARROWBAND NOISE	17	(BB, AB, PB, JB, TAB, PB, RUD, AF, RHe, JHo, EL, ITM, IC, AP, PP, TVS, MVe)
WIDEBAND NOISE	6	(GB, MH, ML, TR, JR, TAS)

4.5.6 Prevalence of SOAEs

Of the 49 subjects (93 ears) tested, 12 (18 ears) revealed a total of 58 SOAEs. As would be expected in a population with so much coexisting hearing damage, this prevalence is lower than in normals (§3). No SOAEs were detected in any of the 8 ears with conductive pathology, in line with expectation. A full breakdown of the SOAEs found is given in Table 4-7 with the SOAE frequency, the tinnitus pitch match, and the audiogram. SOAEs were found from 0.9 kHz to 9.6 kHz, with levels up to 16 dB SPL, and are illustrated in Fig.3-2 (§3) by crosses (RS/Dural microphone) and plus signs (4155 microphone). (The SPLs sometimes differ from those in Wilson and Sutton 1981 due to the different calibration used). Note the triads of SOAEs in the CDT frequency relationship $f_1=2f_2-f_3$, as discussed in §3.4.3; in AM's case however the relative levels suggest that the 1349 Hz emission is an independent SOAE not a CDT 'satellite'.

TABLE 4-7

PREVALENCE OF SOAEs IN TINNITUS SUBJECTS

SUBJECTS: 12 / 49 (24 %) **EARS:** 18 / 93 (19 %)

Subj		Freq (Hz)	Level (dB SPL)	Audiogram (dB HTL) 0.25	0.5	1	2	3	4	6	8 kHz	Tinn.ear/ Pitch match
JB	R	1723	-11	45	35	20	10	30	65	75	75	L/5100
TAB	L	1766	+16	5	0	-5	-10	-10	-5	-5	15	
		1907	+1									
	R	1345	-7	10	5	-5	-10	-10	0	-10	15	R/380
		2008†	-11									
		2202†	+4									
		2399†	-5									
		2534	0									
		3256	+2									
IC	R	1488	+4	25	25	5	5	30	60	60	55	R/8900
RUD	R	1517	-1	5	0	-5	-5	-10	5	30	60	R*/3300
		1902	+3									
JvH	L+	899	-12	-5	-15	-10	5	40	60	45	35	L/10400
	R+	910	-5	-5	-10	-15	-15	-5	0	0	15	
RHe	R	1423	-10	5	0	-10	-5	-15	-5	-15	0	L/1600
		2137	0									
		2739	-5									
NN	L+	6274**	0	30	55	55	55	40	30	15	0	In head/
		8040**	-11									To L 5260 6600
	R+	5903**	+5	20	40	45	45	40	30	15	5	To R 8600
		9147	-16									8140/6685

\+ These measurements carried out with 4155 microphone (others with BS/Dural)
* Indicates stronger tinnitus ear in subjects with some tinnitus in both.
** Correlate to tinnitus.
† Components in relation f_1-$2f_2$-f_3

CONTINUED OVER ....

TABLE 4-7 (continued)

Subj		Freq (Hz)	Level (dB SPL)	Audiogram (dB HTL) 0.25	0.5	1	2	3	4	6	8 kHz	Tinn.ear/ Pitch match
AM	L+	1615**	-4	5	5	-5	-5	-15	-10	5	15	Both/1610
		4691	-8									1250/9260
		5866	-6									1600/6365
		6716**	-9									8575/6900
		9610	-5									
	R+	1349†	+4	0	-5	-10	-10	-10	-15	0	10	
		1432†	-3									
		1513†**	+4									
		2901	-4									
		3071	-4									
		3220	-4									
		3851	-9									
		5096	+5									
		5330	-3									
		7156	+6									
		8052	-8									
		9415**	+3									
KP	L	1205	+3	5	0	0	-10	0	-10	-5	0	
		1656	-1									
		1925	-13									
		2367	0									
		3342	0									
		3713	-2									
	R	1107	-4	0	0	-5	-15	0	-10	-5	0	R/1265
		1245**	0									
		1730	+2									
		2561	-10									
		3054	+7									
		3725	+8									
		4098	0									
TR	L	1380	-9	-5	-5	-5	10	20	35	35	45	R/Wideband
AR	R	1337	+5	0	0	0	-5	0	0	-5	35	R*/7600
NWb	L	1587***	-15	15	10	5	5	50	65	80	80	L/1600 2300/3600
	R	1001	-4	20	10	-5	10	50	60	55	55	
		1204	-2									
		1622	-9									
		1778	-13									

4.5.7 Hearing levels at SOAE frequencies

The HTL at the SOAE frequency (estimated from the Bekesy tracing to the nearest 5 dB) ranged from -15 to +15 dB ISO, with a mean of -1 dB (Table 4-8). Hence in no case was an SOAE found at frequency where the HTL was greater than 15 dB, supporting hypothesis II.

TABLE 4-8

HTL AT SOAE FREQUENCIES

-15 dB	3 SOAEs	
-10 dB	10	
-5 dB	22	Mean = -1 dB
0 dB	12	S.D. = 8 dB
+5 dB	8	
+10 dB	5	
+15 dB	5	
>15 dB	0	

4.5.8 Cases with acoustic correlates of tinnitus

Three cases (KP, MM, AM) were found whose tinnitus appeared to have an acoustic correlate. Very significantly, all 3 positive cases were in a sub-group of 6 who had both pure-tonal tinnitus *and* good hearing at the pitch match. The average HTLs (see Table 4-4) were -5, 31 and -5 dB respectively. All 3 were young women. Although one of the pitch matches of a further subject (MWh) corresponded to an SOAE, the higher frequency matches seemed to represent the stronger sensations, so that this case has not been called a correlate.

Approaching the data from the other direction, SOAEs were detected in 12 subjects: in 3 of these the SOAE was in the wrong (contralateral) ear and in a further 6 it was at the wrong frequency to account for the tinnitus, leaving the 3 cases of correspondence. The individual features of these cases will be examined.

Subject KP (Fig.4-1)

(Preliminary report in Wilson and Sutton 1981)

This was a 20-year old female with clinically normal hearing in both ears, except for an apparent low frequency droop below 200 Hz on the left, and slight irregularities near 3 kHz. (The audiogram illustrated for this and other subjects was drawn by eye through the Békésy traces.) The tinnitus was tonal, "like an off-tune radio", right-sided and was first noted after a wisdom tooth extraction 1 month previously. 6 SOAEs were found in the left and 7 in the right ear, one of which was close to the pitch match.

Subject [redacted] (Fig.4-2)

(Preliminary report in Wilson 1986)

This was a 21-year old female with a moderate bilateral low-to-mid tone loss (U-shaped audiogram), and normal HTLs above 6 kHz. The probable aetiology was congenital rubella, but there was also a 9-year history of earache and discharge accompanied by a facial rash. The tinnitus had been present for a long time but had worsened over the past 2 years. It was multi-tonal ("ringing notes of various frequencies, each one pulsating in level") with a background of "rushing and hissing atmospherics", and seemed to be in the head. 4 high frequency SOAEs

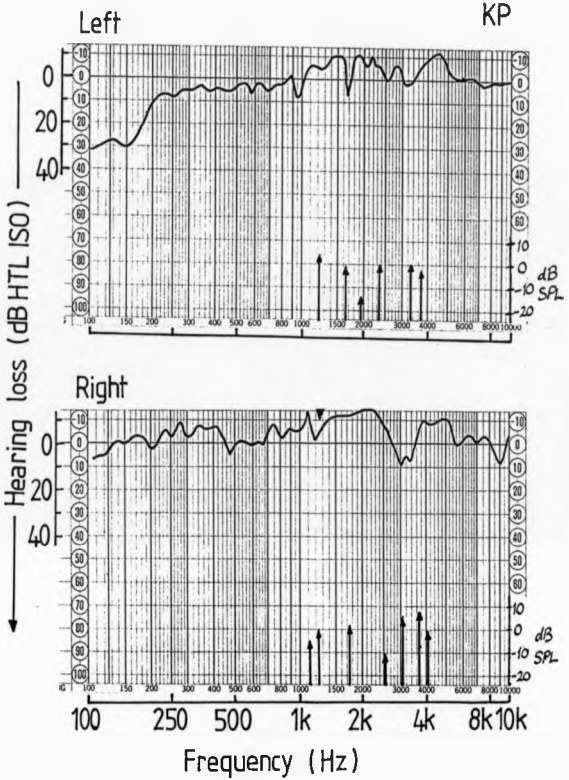

Fig.4-1 Békésy audiograms for subject KP. Tinnitus pitch match is shown by downward arrowhead; SOAEs by vertical arrows.

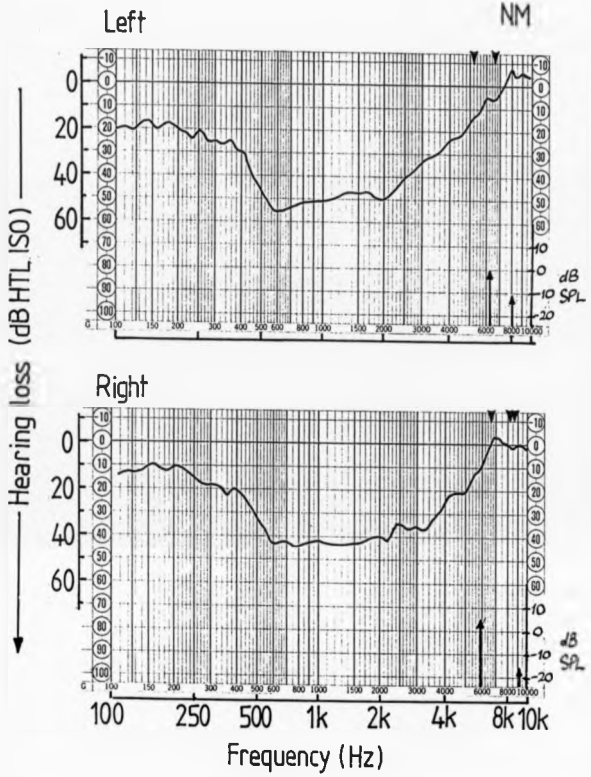

Fig.4-2 Békésy audiograms for subject NM. Tinnitus pitch matches (contralateral) are shown by downward arrowheads; SOAEs by vertical arrows.

were found (2 in each ear), ranging from 5.94 to 9.2 kHz, some varying in level. The tinnitus localisation appeared to be due to binaural fusion of the left 6274 Hz SOAE with the 5940 Hz right SOAE.

Using the 4155 microphone on the Y-tube with the 4134 as driver (see §3.1.4) very strong stimulus-frequency evoked emissions were found in this ear at high frequencies (Fig.4-3, from Wilson 1986). This is surprising in view of the presumed attenuation of the middle ear system at these frequencies, and implies high cochlear levels of these SFEs.

Subject AN (Fig.4-4)

This was a 17-year old female who first noted bilateral tonal tinnitus during a series of anxiety attacks 1 year before. Hearing was clinically normal in both ears, although there were 25-35 dB notches at 8 kHz. The tinnitus was described as "a sharp whisting sound", but it later became clear that it was multi-tonal. It was heard about equally in both ears. Pitch matches were very variable (1 to 9.3 kHz) and the subject was not an ideal observer, but it appeared that the dominant components of the tinnitus were round 1600, 6700 and 9000 Hz. A rich crop of SOAEs was detected - 5 in the left and 12 in the right ear - over a very wide frequency spectrum (1.6 to 9.6 kHz). Many were variable and unstable, switching between different frequencies. Again at the high frequencies we infer high levels in the cochlea. An SOAE was found corresponding with each of the main pitch match regions.

As mentioned above, confirmation of the relation between SOAE and tinnitus was sought by other methods for these 3 cases. All 3 claimed

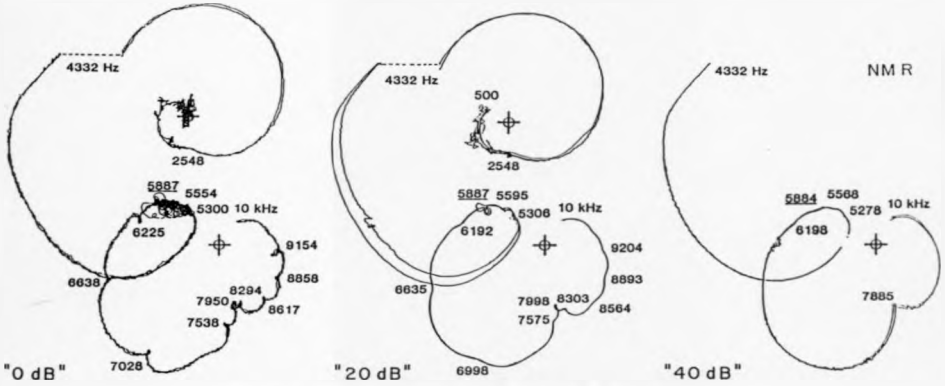

Fig.4-3 Presence of SFEs in right ear of subject NR shown in Nyquist (vector) plots by interference loops between SFE and continuous tone stimulus, for 3 stimulus levels. Note presence of SOAE at 5887 Hz. Scale (and position of origin) was altered at 4332 Hz.

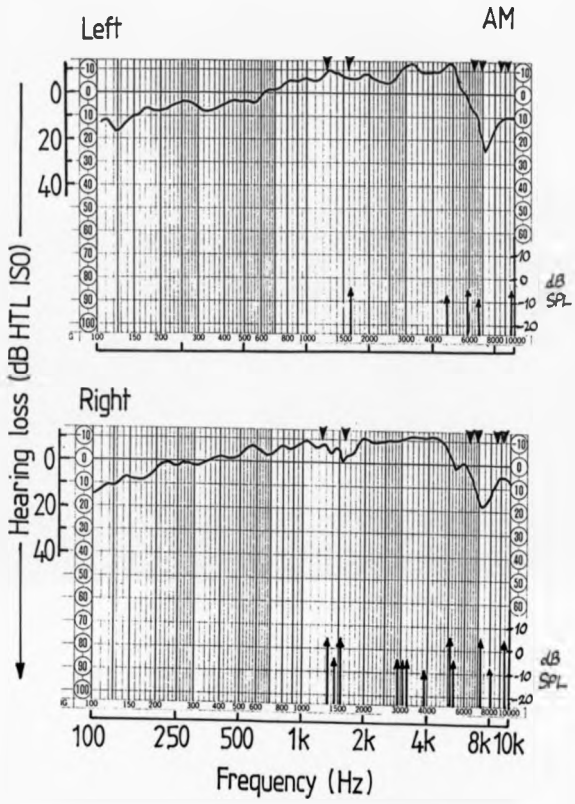

Fig. 4-4 Békésy audiograms for subject AM. Tinnitus pitch matches are shown by downward arrowheads; SOAEs by vertical arrows.

to recognised the SOAE recording as at least part of their tinnitus sensation. Subject MM was clearly aware of irregular beating with an nearby external tone, whereas KP and AM were not. The condition of 'cancellation' of the tinnitus and external tone was not achieved (Vegel 1931, Ward 1955, Wilson 1980cd), but little time was spent attempting the critical adjustments of frequency and level of the external tone often required. For subject KP, ear canal pressure shifted tinnitus pitch in the same direction as the SOAE. Masking tests for AM showed that 50-70 dB was required to mask the tinnitus irrespective of masker frequency: whilst one would not expect this pattern for tinnitus caused by one SOAE, it might arise from a multi-component multi-tonal tinnitus.

The results indicate beyond reasonable doubt that the SOAE(s) are the source of the tinnitus in subjects KP and MM, and probably also in subject AM, although the variability of AM's pitch matches mean that some doubt must remain in this case. Hypothesis I is thus confirmed. Thus the overall prevalence (on this non-random sample) of cochlear mechanical tinnitus is 3/49 (6%). However among those subjects who had both (a) tinnitus which was pure-tonal (or had at least a tonal component) *and* (b) threshold better than 20 dB HTL at the tinnitus pitch match, the prevalence is no less than 3/6 (50%). It must be said that these conditions are met in only a small proportion of the whole clinical tinnitus population, both in this study and even more so generally; therefore one may conclude that the vast majority of tinnitus has no acoustic correlate and arises from other sources of non-vibratory origin.

4.5.9 Loudness and Annoyance of tinnitus

Results for mean and SD for the different ratings are given in Table 4-9. Ratings for campus volunteers and ENT referrals showed negligible differences, so all have been pooled. The cases showing acoustic correlates gave moderate ratings (although there was no data for KP). None of the cases rating maximum 7 on loudness (8 cases) or on annoyance (10 cases) had an objective correlate for the tinnitus. This is consistent with the suggestion that cochlear mechanical tinnitus is not very severe, and that the most troublesome types of tinnitus coexist with greater degrees of hearing damage and loss.

TABLE 4-9

TINNITUS LOUDNESS AND ANNOYANCE RATINGS

LOUDNESS MATCH AT PITCH MATCH (dB SL)

(N=39) Mean = 11 dB SL

S.D. = 5 dB SL

LOUDNESS RATING (SCALE OF 7)		ANNOYANCE RATING (SCALE OF 7)	
(N=30)	Mean = 4.4	(N=31)	Mean = 4.5
	S.D. = 1.7		S.D. = 1.9

4.5.10 Audiometric features, tinnitus pitch and SOAEs

A number of authors have noted that tinnitus pitch matches often coincide with audiometric notches or on the edge of a sharp audiometric fall-off (Fowler 1940, Douek and Reid 1968), and such correspondence was found with a number of subjects here (PB, CC, RUD, DG, RHo, CJo, JM, HM, ITM, AM, PP, AR, TVS, MVh). In other cases there was no clear

relationship. The relationship of SOAEs to audiometric features is discussed in §4.6.4 and §4.6.5. No SOAEs were detected in any of the 8 ears with known conductive pathology, in line with expectation.

4.6 DISCUSSION

4.6.1 Prevalence of cochlear mechanical tinnitus

Three cases of acoustic correlates of clinical tinnitus have been found among the 49 subjects tested. Data reported by various other workers are summarised below in Table 4-10 (again where tinnitus appeared to predate testing). (Wilson and Sutton 1981 included an interim report of the work in this chapter).

TABLE 4-10

DATA ON RELATIONSHIP OF SOAEs AND CLINICAL TINNITUS

	Sample size	No of subjects with SOAEs	with correlates
Wilson and Sutton (1981)* *Exc. GMW, EPE, GFP	8	3	1
Zurek (1981)	6	2	0
O'Brien (1981)	20	2	0
Tyler & Conrad-Armes (1982)	25	1	0
Hazell (1984)	53	?	2
This work (inc. Wilson and Sutton 1981)	49	12	3

It is noteable that it is the 2 largest studies which have reported positive results, presumably because such cases are relatively uncommon (3/49 or 6% here, where there was a deliberate bias towards likely cases). SOAEs themselves are less prevalent among tinnitus sufferers than among normals, because of the association of tinnitus with hearing loss.

Therefore it is concluded that cases of cochlear mechanical tinnitus do occur among clinical tinnitus sufferers, and are not confined to subjects with completely normal hearing, as shown by case NM. SOAE-related tinnitus may be widespread among those with tonal tinnitus and normal hearing at the tinnitus pitch, and it appears well worth-while searching for acoustic correlates in such cases; it is often reassuring to the patient that a real and objective source of tinnitus can be identified.

Because aspirin abolishes SOAEs (McFadden and Plattsmier 1984) it should abolish tinnitus that is cochlear mechanical - this could therefore be used to provide confirmation in suspected cases.

Tinnitus from this source seems rarely to be very troublesome, presumably because it is usually easily masked by ambient noise. Thus the vast majority of clinical tinnitus has no acoustic correlates and arises from other sources of non-vibratory origin.

4.6.2 Possible mechanisms of SOAE-related tinnitus

Tyler and Conrad-Armes (1982) argued against the likelihood of cochlear mechanical tinnitus and "patients with tinnitus of cochlear origin" having SOAEs as follows:

1. SOAEs require normal haircell function,
2. The presence of tinnitus 'of cochlear origin' requires abnormal haircell functioning,

therefore 3. The presence of cochlear tinnitus is not associated with SOAEs.

However because an SOAE can arise from any region of normal haircell function in the cochlea and does not require that the whole cochlea be undamaged, (1) and (2) do not imply (3) if the 'haircell functioning' referred to is in a different physical region. Only where there is hearing damage at all frequencies is the conclusion valid.

The finding that aspirin abolishes SOAEs while producing tinnitus (McFadden and Plattsmier 1984) only demonstrates that aspirin-caused tinnitus is not cochlear mechanical; it does not, as those authors claimed, make the existence of SOAE-related tinnitus less likely.

One needs to explain why, whilst most SOAEs are unheard or heard only faintly in quiet, in some subjects they cause non-trivial tinnitus. Possible explanations include:

(a) that a long-standing SOAE becomes noticeable and troublesome when some hearing loss subsequently develops, because it is no longer so easily masked by ambient noise;

(b) that a new SOAE develops, either due to changed boundary conditions in the cochlea (caused by otosclerosis for example) or due to local cochlear damage (Evans et al 1981, Zurek and Clark 1981, Ruggero et al 1983, McFadden and Plattsmier 1984). These new SOAEs might be more noticeable to their owner while others are not because (i) adaptation processes affect long-standing SOAEs (Schloth 1982, Schloth and Zwicker 1983), or (ii) that they are of a different 'pathological' type, which might be be less coupled to stapes motion and stronger locally on the cochlear partition than a 'normal' SOAE. However the evidence for 2 types of SOAE is scanty (see discussion §4.6.5).
(c) that psychological factors cause some subjects to perceive SOAEs as much more bothersome than others, and/or that some traumatic personal event can amplify the perception of an SOAE. There is no doubt of the importance of psychological factors in determining a patient's reaction to tinnitus and their ability to cope with it, which varies greatly, even with similar loudness ratings (e.g. House 1981). It is also a common clinical finding that tinnitus is first noted after a bereavement: this may be explained as a change in perception rather than physiology.

It is not possible to say with any certainty which of these apply to the 3 cases here, for we do not know if the SOAEs were always there or developed later. AM might be consistent with (a) if the SOAEs were present before the bilateral 25-dB notches near 7 kHz in her audiogram; if however they arose together one might consider the possibility that they were 'pathological' SOAEs. In fact the onset of tinnitus after anxiety attacks suggests (c) as the likeliest reason for AM. Similarly

EN had a marked hearing loss over most of the spectrum, but this predated the tinnitus by many years, so making (a) less likely. KP had no hearing loss of note.

4.6.3 Possible other types of cochlear mechanical tinnitus

It is possible that some other tinnitus may also be of cochlear mechanical origin but without acoustic correlates being detectable by current methods. For example:

1. In otosclerosis, where tinnitus often disappears after stapedectomy (Glasgold and Altmann 1966, House and Brackmann 1981). Any oscillation in the cochlea would be attenuated below detectability by the middle ear loss.
2. Some non-tonal tinnitus might have wider-band acoustic correlates, which are more difficult to extract from background noise.
3. Kemp's (1981) 'type II', where the intracochlear vibration source would be decoupled from the rest of the cochlea (the result of local feedback), and thus not transmit to the meatus.

The existence of such types remains, for the moment, purely speculative.

4.6.4 Are all SOAEs pathological?

One key question is whether SOAEs should be viewed as a naturally-occurring epiphenomenon of truly normal hearing, or as 'pathological'; the result of minor cochlear damage (what one might call 'cochlear bruises'), perhaps in the form of localised outer haircell damage (Ruggero et al 1982, 1983, Clark et al 1984). Clark et al's (1984)

report, in which SOAEs were found in 2 out of 28 noise-exposed chinchillas, with cochlear punctate damage at the appropriate place, does show that pathological damage can be linked to SOAEs, but not that all are thus linked. In any case this paradigm may not be very relevant to most SOAEs (this is further discussed below in §4.6.5).

To examine the hypothesis that 'all SOAEs are pathological' we must be careful about what is implied in the notion of pathology. To be valid and coherent the above statement must mean not only (i) that some measurable deviation from ideal function accompanies all SOAEs *but also* (ii) that SOAEs will not be found in a group of ears defined as 'normal' by an independent method. This is essential to avoid circularity in the argument about normality.

Looking first at the evidence on prediction (i) as found in thresholds, it must first be noted that some threshold fine structure is in any case to be expected (usually including a sensitivity peak at the SOAE frequency) due to the 'whole cochlear resonance' effect (see §1.2.13), but this is not related to underlying function. Aside from this, some minor audiometric elevations have been found associated with SOAEs (e.g. Wilson 1980c fig.10, Ruggero et al 1983) as well as more marked notches for the rarer high-level emissions (Glanville et al 1971, Huizing and Spoor 1973, Wilson and Sutton 1983). However for the subjects in this thesis abnormalities were not consistently seen at or near SOAEs, despite the Békésy audiometry which reveals features that would be missed by a 6-point clinical audiogram. Some abnormalities were observed for some subjects in §4, (e.g. KP right, 3 kHz; AM right

7 kHz), but in most cases they were not (e.g. AM right, 3 kHz). Similarly in the normal subjects (§3) signs of pathology were mostly uncorrelated with SOAEs. The threshold at the SOAE frequency, the best measure of hearing function here available, averaged 0.2 and -1.1 dB HTL for the normal and tinnitus subjects respectively, and was never worse than 20 dB HTL.

Since however it is clear that sensitivity to pure tones does not tell us everything about cochlear function, it could be argued that abnormalities might be revealed only in other, more sensitive measures, such as frequency resolution (e.g. Pick and Evans 1983). This caveat notwithstanding, there is no evidence here of local pathology in the vicinity of most SOAEs.

Turning to prediction (ii) there are three crucial pieces of evidence:

(a) the same SOAE prevalence is found in children and infants (Strickland et al 1985), even though they have had less opportunity to accumulate outer haircell damage. Also the average number of SOAEs per ear in this study was significantly *higher* than for adult ears;

(b) the same near-100% prevalence of cochlear echoes is found in neonates (Johnsen et al 1983) as in adults;

(c) the prevalence of SOAEs is higher in females (§3.4.2), although it is males who tend to sustain more pathology through noise etc (e.g. Hinchcliffe 1959).

These data (particularly (a)) strongly argue for most SOAEs (and SOAEs) being considered as 'normal', and effectively refute the hypothesis

that all SOAEs should be regarded as 'pathological'. 'Pathology' implies abnormality, and to insist that about 40% of all unselected newborn ears are 'abnormal' begs the question. Whilst we could accept the notion that a large proportion (even a majority) of adults might have pathological hearing - as we do in the case of presbyacusis - we cannot with neonates (particularly as those in the above studies were not apparently in 'at-risk' groups). Even if it were shown that each SOAE was associated with a few missing haircells, and this might be clearly be viewed as a departure from the ideal, it could not fairly be called 'pathological' if it occurred in so many virginal ears. The situation is analogous to finding an audiometric threshold of (say) 10 dB HTL - this is viewed as part of the normal spread, not pathology.

4.6.5 Are there 2 types of SOAE - normal and pathological?

The above conclusion that all SOAEs are not pathological does not rule out the possibility that *some* may be. There is, as noted above, evidence associating some SOAEs with pathology, and the question remaining is whether all SOAEs have the same underlying mechanism or whether some require a different type of mechanism, related to local pathology, to explain them - i.e. is there one type of SOAE or two?

The evidence linking some SOAEs to pathology comes from:

(a) the existence of some very strong SOAEs with levels up to 60 dB SPL, which seem an order removed from the normal low-level SOAEs which rarely exceed 20 dB SPL. Also these are often associated with marked audiometric notches (Glanville et al 1971 and Wilson and Sutton 1983, Huizing and Spoor 1973).

(b) Some data appear to show different behaviour for some SOAEs, notably in the form of their suppression tuning curves which have multiple lobes in contrast to the normal pattern (Evans et al 1981, Ruggero et al 1982, Wilson and Sutton 1983).

(c) The apparent association of some SOAEs with transitions from normal to elevated threshold (Wilson 1980c, case HM above, Ruggero et al 1982, 1983) or slight audiometric abnormalities (Wilson 1980c, fig.10). It is not really clear however whether these SOAE are 'caused' by the adjacent hearing loss or are independent of it.

(d) The report of Fritze (1983) of higher prevalence of SOAEs in a group with 'mild cochlear losses' (up to 25 dB HTL) than a group with 'normal hearing'.

(e) The induction of SOAEs by noise exposure in chinchillas (Zurek and Clark 1981, Clark et al 1984), and (temporarily) in man (Kemp 1981, Fritze and Köhler 1986); Clark et al (1984) investigated histopathology and found cochlear lesions near the positions corresponding to the SOAE frequencies, although many animals without SOAEs also had these. McFadden and Plattsmier (1984) also found a new SOAE following recovery from effects of aspirin.

One important point should be emphasised initially: merely to show that an SOAE is sometimes associated with pathology or damage, or even caused by the pathogen, does not prove that the SOAE is itself of a different type with a different pathological mechanism. The irregularity hypothesis (§1.3.1) suggests that SOAEs arise because of minor local irregularities at some points along the cochlea. Such an irregularity might take the form of a few missing haircells or other

structural variation. If one accepts this then pathological cochlear damage might be considered as merely differing in degree (i.e. as a large acquired irregularity), as shown for the summed response model (§2). Thus a pathology-related SOAE may in fact be produced by the same mechanism as a normal SOAE, and merely precipitated by the cochlear damage. Therefore neither the association of notches or other audiometric features with SOAEs, nor the demonstration of noise-induced SOAEs *necessarily* implies a different pathological mechanism. Differences of behaviour and properties are needed to demonstrate this: which leaves the evidence of (a) and (b) to be assessed.

The very strong SOAEs - absolute levels and notches

The existance of these high-level SOAEs (Glanville et al 1971, etc) coupled with their associated audiometric notches might suggest a different type of mechanism. However the accumulating literature on SOAEs in normal subjects is revealing a wide spread of SPLs, and whilst most are still below 10 dB SPL, some are found with higher levels - up to 24 dB SPL in our subjects, 26.5 dB SPL in Burns et al (1984), and 29 dB SPL in Strickland et al (1985). These somewhat bridge the gap between the normal low-level and the high-level emissions and raises the likelihood that the latter simply represent the extreme end of the distribution of normal SOAEs, rather than a different 'pathological' type.

The data showing an audiometric notch near these SOAEs are not straightforward to interpret. It can be argued that this notch, rather than being 'essential' (reflecting true pathology and the cause of the

SOAE) might be caused by masking of the test tone by the SOAE, or be due to an adaptation effect. However Wilson and Sutton (1983) concluded that masking could not account for the whole of the elevation. Similarly there appears to be no threshold elevation near most SOAEs (rather sometimes a fine-structure threshold *minimum* occurs at the SOAE frequency - Kemp 1979b, Wilson 1980c, Schloth 1982, Zwicker and Schloth 1984) and thus no evidence of masking in these cases. This implies that the notch is genuine, rather than a masking effect, but again, on its own this is not proof of a different mechanism. An adaptation effect is suggested by the finding (Schloth 1982, Schloth and Zwicker 1983) of an SOAE which was only audible when shifted in frequency by a few hertz. This could only be a plausible explanation for an SOAE which was quite stable in frequency.

Differences in suppression behaviour of SOAEs. Some of the results showing multi-lobed STCs for SOAEs (Zurek 1981, Ruggero et al 1983) may be artefactual due to frequency shift of the SOAE out of the filter band (see §5), although some are definitely not (Evans et al 1981, Wilson and Sutton 1983). However in §5.1.and §5.2 multi-lobed STCs for a normal low-level SOAE and for SFEs are reported, so making it less likely that these indicate a different pathological mechanism. Instabilities of frequency and level are also common to both low and high level SOAEs.

Summarising, there still seems insufficient data to decide the question. In the meantime Occam's razor suggests that one should continue to work on the simplest hypothesis - that all SOAEs reflect

the same 'normal-hearing' mechanism. One useful experiment would be to look at the effect of aspirin ingestion in an individual with an SOAE in an audiometric notch. This should abolish the SOAE and allow us to answer the 'chicken-and-egg' question about whether the notch is a masking effect or not.

5. SUPPRESSION OF SPONTANEOUS AND EVOKED OTO-ACOUSTIC EMISSIONS

The study of the suppression of OAEs, whether spontaneous or evoked by clicks or continuous tones, is a powerful method for probing the properties of these emissions and the mechanisms underlying them.

Most data in the literature are in the form of suppression tuning curves (STCs) which show how readily the emission (SOAE or EOAE) at a fixed frequency is suppressed by tones at other frequencies. Using appropriate methods, suppression experiments can be carried out on either click-evoked OAEs or continuous-tone evoked OAEs (SFEs) and STCs derived. A complementary approach is to examine the effect on the whole spectrum of EOAEs by a single frequency suppressor (as in Kemp and Chum 1980a).

Reports on the suppression of SOAEs and EOAEs (echoes and SFEs) include those of Kemp (1979ab, 1981, 1982), Wit and Ritsma (1979, 1980), Kemp and Chum (1980ab), Wilson (1980c), Kemp (1981), Wilson and Sutton (1981, 1983), Zwicker and Manley (1981), Evans et al (1981), Zurek (1981), Zurek and Clark (1981), Wit et al (1981), Schloth (1982) (and Schloth and Zwicker 1983), Zwicker (1983), Ruggero et al (1982, 1983), Rabinowitz and Widin (1984), Burns et al (1984).

Most of these reports which have given STCs show a single-lobed curve, with sharp tuning matching neural and psychophysical tuning curves, whose tip is a little above the emission frequency. There are however some exceptions to the general rule, in that some of the above authors

have found STCs with more than one lobe for SOAEs. In the case of Wilson (1980c) it was clear that this was not true suppression, but was the result of the SOAE shifting frequency out of the analysing filter: the same probably applies to Zurek (1981) - later measurements on the same ear (Rabinowitz and Widin 1984) showed a simple single-lobed STC. Similarly the multiple lobes of Ruggero et al (1983) for a high-frequency SOAE represent the combined effects of suppression and frequency-shifting. There remain the reports of Evans et al (1981) showing 2 widely-separated lobes for a guinea pig SOAE, and of Wilson and Sutton (1983) showing 3 or more lobes for an exceptional high-level SOAE: both of these represent genuine suppression.

There have been no double or multiple-lobed STCs reported for EOAEs, although there are slight indications of some in Kemp and Chum's data (1980b). This report also showed a clustering of the tips of STCs near certain frequencies. The displacement of the STC tip towards frequencies higher than the emission is common to all reports except Wit and Ritsma (1979) (who found no displacement) and Ruggero et al (1982) (who, in a dog's ear, found a displacement towards lower frequencies for an SOAE which was associated with a high-frequency hearing loss).

To add to the limited data, experiments were carried out on 2 strong stable SOAEs in 2 ears, and also on both cochlear echoes and SFEs in another ear (the author's right), with normal hearing and no detectable spontaneous emissions. The results are reported in 3 sections, and are discussed together at the end.

5.1 SUPPRESSION OF SOAEs

Experiments were carried out on 2 strong stable SOAEs (at 1.77 kHz and 5.90 kHz) in subjects TAB and NN respectively.

5.1.1 Methods

Subject TAB

The essentially-normal audiogram for the left ear (the non-tinnitus ear of a subject reported in §4) is illustrated in Fig.5-1. The SOAE under study was measured at 1770 Hz (9 dB SPL) at the time of testing; there was a second weaker and less stable SOAE at 1907 Hz (3 dB SPL).

The SOAE was monitored using a Knowles EA-1842 miniature microphone sealed into the ear canal, amplified and analysed using the dual-lockin analysis system (§3.1.3). The suppressor tones were introduced via a Sennheiser HD414 earphone over the pinna, and their levels determined from the microphone output.

Subject NN

The audiogram for the right ear of this subject (again a tinnitus subject reported in §4) was shown in Fig.4-2; it is U-shaped with a maximum loss of about 45 dB, but normal at high frequencies. The SOAE at 5900 Hz (level on this occasion 8 dB SPL), audible as tinnitus, was studied. A second much weaker SOAE at 9147 Hz (at about -16 dB SPL) was detected on a tape recording.

The SOAE was monitored using a B&K 4155 microphone system coupled to the ear canal by a small stainless steel Y-tube (see §3.1.4).

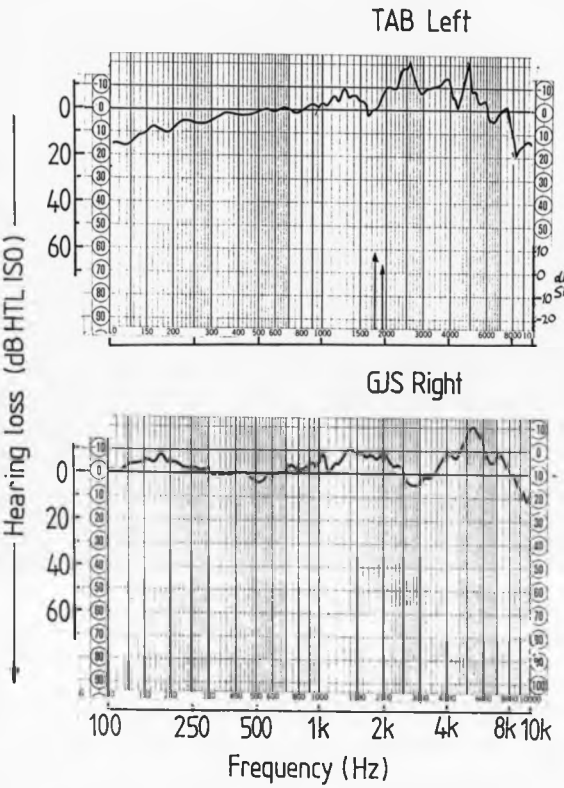

Fig.5-1 Békésy audiograms for TAB (left ear), and GJS (right ear).

Suppressor tones were fed into the other arm of the Y-tube from a B&K 4134 driver-microphone. The 4155 microphone output was amplified and analysed on the B&K 2020 slave filter system (§3.1.3) with bandwidth set at 10 Hz, monitoring on a chart recorder. Suppressor SPLs were measured from the 4155 output with the lockin synchronised to the suppressor frequency.

The analysing filter was kept tuned to the SOAE frequency at all times so that the derived STC represented 'true suppression' and not frequency-shift of the SOAE out of the filter (Wilson and Sutton 1981). Levels of suppressor tones were slowly increased until the SOAE was reduced by 3 dB, and the new frequency of the SOAE in this suppressed state was also noted.

5.1.2 Results

Subject TAB

The STC is shown in Fig.5-2 (bottom) for a 3 dB suppression criterion. It shows 2 distinct minima; one just above the SOAE frequency at about 2 kHz and one at 2.8 kHz. The rate of suppression was markedly more rapid at the midpoint 'maximum' at 2.4 kHz than at these minima.

The frequency shift of the SOAE during suppression, measured as the change in SOAE frequency that occurred immediately the suppressor was turned off, is also illustrated (top). Suppressors from 1.9 to 2.8 kHz all shifted the SOAE frequency downwards, maximally (0.4%) at 2.4 kHz. Above the second lobe (2.8 kHz) the shift was upwards.

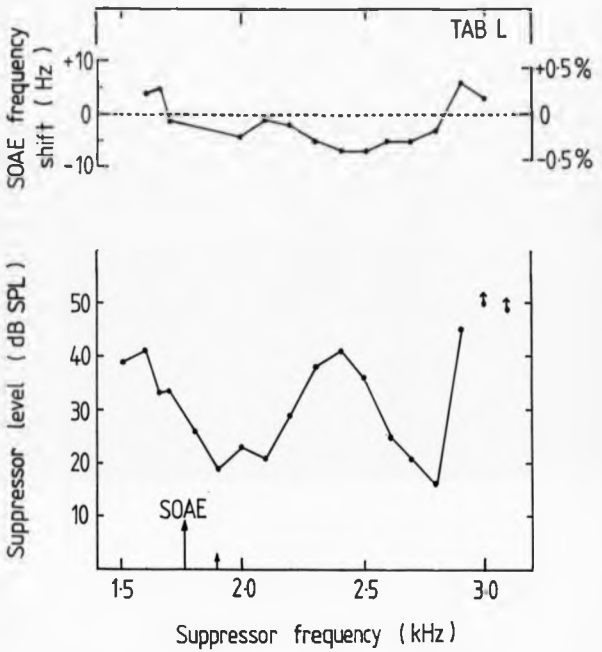

Fig.5-2 Suppression of 1770 Hz SOAE in subject TAB (left ear). Bottom: Suppression tuning curve, 3-dB criterion. Top: Change in SOAE frequency due to this level of suppressor tone.

Subject ■■

Results are shown in Fig.5-3 (as for Fig.5-2 except that at the top the actual SOAE frequency under suppression, rather than the shift, is illustrated). The STC shows fine structure at least 2 local minima at 6.2 and 7.0 kHz. Frequency shifts were always downwards, and maximally 0.5%.

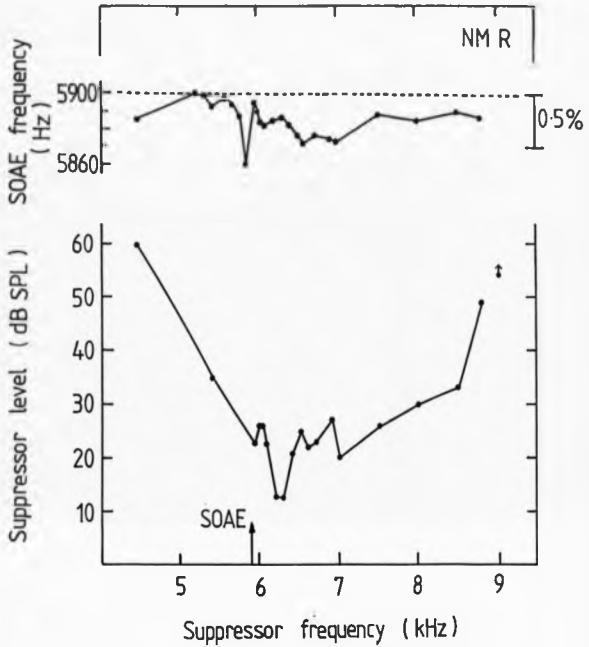

Fig.5-3 Suppression of 5900 Hz SOAE in subject HM (right ear). Bottom: Suppression tuning curve, 3-dB criterion. Top: SOAE frequency with this level of suppressor tone on.

5.2 SUPPRESSION OF SFEs

5.2.1 Methods

The dual-lockin system for SOAE detection was adapted to detect SFEs as described in §3.1.4. This however displays the total ear canal pressure vector, including the stimulus. To extract the SFE vector a method similar to Kemp and Chum's (1980b), based on the saturating property of SFEs, was used. The aural signal is dominated by the stimulus at high stimulus levels, but at lower levels the contribution of the SFE, adding vectorially to the stimulus, becomes increasingly significant. By cancelling the total ear canal pressure vector at high stimulus levels, the SFE vector is then revealed at lower levels. The effect of various suppressor tones on this SFE, and thus the STC, can then be determined.

The equipment used is illustrated in block diagrammatic form in Fig.5-4. Stimulus (S) and suppressor (M for 'masker') tones were taken from oscillators (Brookdeal 9471 for S, Levell for M) with attached frequency counters, added, amplified (Radford) onto a B&K 4134 driver-microphone polarised at 200 V. This was fitted onto one arm of the Y-tube assembly (§3.1.4) sealed into the ear canal with a bored E-A-R plug. A sensitive microphone (Wilson 1980a, §3.1.1) was fitted on the other arm of the Y-tube, and the output was amplified and then fed into a second electronic adder. The second input to this box came from the S oscillator via a phase shifter to allow cancellation at high levels. The signal output was then fed to the Brookdeal 401 dual-lockin system controlled by the S oscillator, with the display on a CRO. For a stimulus input of constant frequency, one obtains a stationary spot

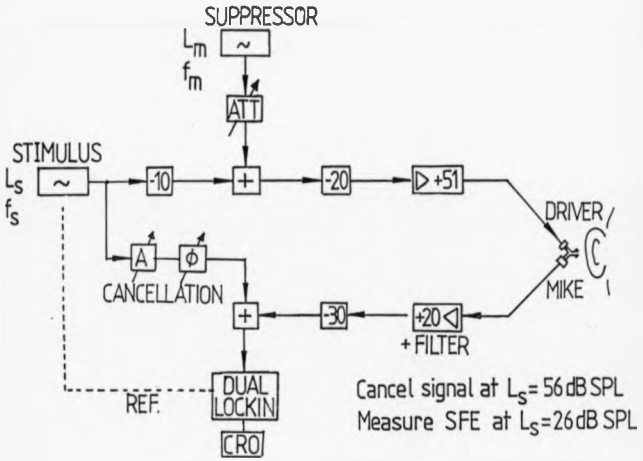

Fig.5-4 Block diagram showing equipment used for measurement of SFEs. Values in boxes are dB of amplification or attenuation according to sign. 'A' and 'ϕ' are amplitude and phase adjustment to achieve cancellation at the lockin input.

whose distance and direction from the origin represents the vector magnitude and phase of the total signal in the ear canal.

Calibration of the meatal SPLs produced by stimulus and suppressor tones was carried out subsequently using a Knowles BT-1753 microphone sealed into the ear canal under the Y-tube assembly. Three runs were performed, removing and replacing the Y-tube from the ear each time: the mean value was used to give a conversion from attenuator setting to SPL. A check using tone threshold measurements gave results agreeing to within 3 dB. Simultaneous recordings of the output of the sensitive microphone allowed SFE amplitudes to be converted to SPLs.

The procedure, having obtained a stable seal in the ear canal, was as follows. The S frequency was set, and its level increased to 56 dB SPL. The 'cancellation' phase and amplitude controls were then adjusted until the CRO spot was at the origin as closely as possible, thus cancelling the stimulus component. The S voltage was then switched down, revealing the SFE as a movement of the CRO spot away from the origin, the resulting vector representing magnitude and phase of the SFE. This was in most cases quite stable and highly repeatable.

Several series of measurements were first made of the magnitude and phase of the SFE - i.e. with no suppressor tones - for a constant voltage onto the driver microphone. Measurement frequencies were usually sufficiently closely-spaced to eliminate phase ambiguity. For the suppression measurements, various SFE frequencies from 910 to 1480 Hz were chosen arbitrarily. The SFE was first measured and then

the suppressor (M) tone was introduced. For each f_m, the subjective threshold was determined, and then the level needed to suppress the SFE by 3 dB and 6 dB (at least 3 measurements were made for the 3 dB criterion).

Checks were made in the test ear to ensure that no overloading, which would cause false apparent suppression, occurred at higher suppressor levels.

5.2.2 Results

5.2.2.1 Audiometry

The Békésy audiogram of this (normal) ear is illustrated in Fig.5-1 (bottom). Thresholds were also measured for the suppressor tones through the probe system (see §5.2.2.3).

5.2.2.2 Amplitude and Phase of SFEs

Fig.5-5 and Fig.5-6 show respectively the amplitude and phase of the SFEs. The stimulus tone level was 26 dB SPL at 1 kHz, rising to 32 dB SPL at 2 kHz (top of fig.5-5). Results shown were measured on a number of different days over a period of about 3 months. The measured phase curves include the stimulus phase variation of about 0.73 cycle/kHz, shown at top of Fig.5-5: one can see why this is so (despite cancellation) if one considers that altering the phase of the stimulus tone must identically alter the phase of the vector measured.

The SFE amplitude shows regions of strong response as usual. Amplitude showed some variation of up to 12 dB, most marked around 1300-1500 Hz.

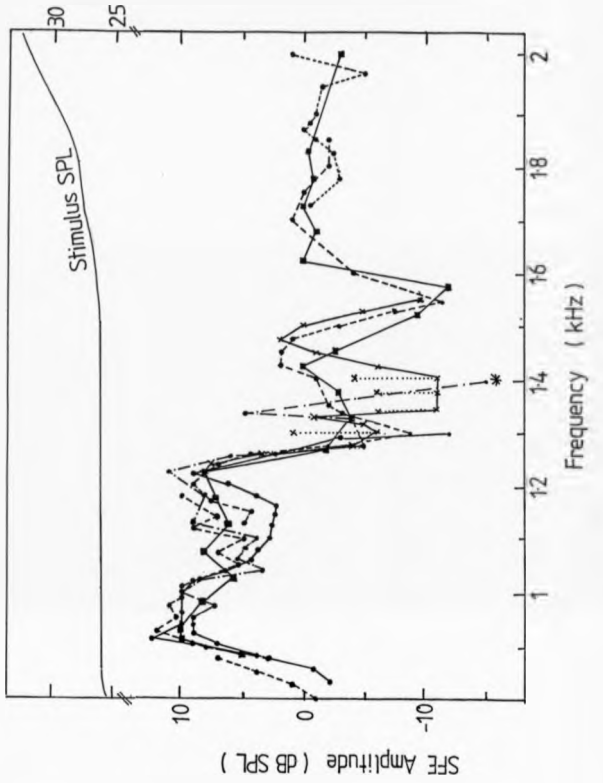

Fig.5-5 Amplitude of SFEs in subject GJS (right ear), as measured on several occasions over a 3-month period. Level of stimulus tone is also shown.

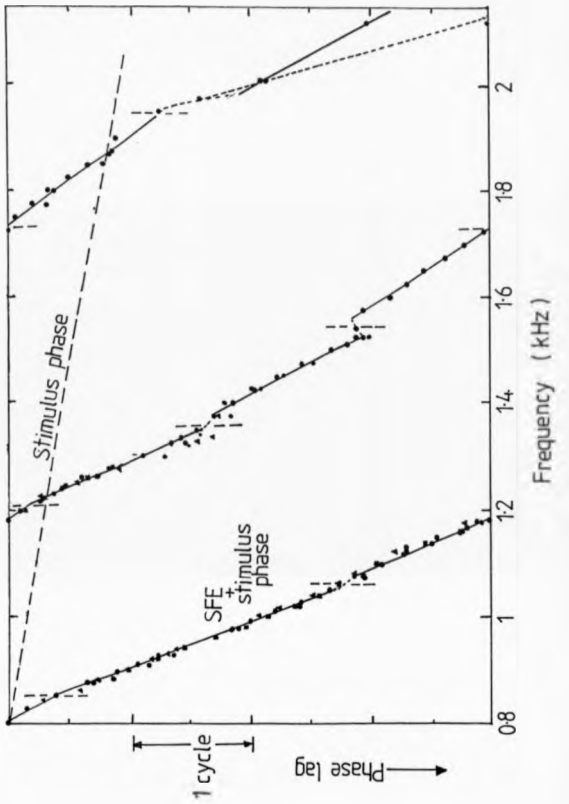

Fig.5-6 Phase of SFEs in subject GJS (right ear), as measured on several occasions over a 3-month period. The curves should be corrected by the phase of the stimulus (shown at top).

Here the SFE was often unstable and would periodically switch between 2 quasi-stable states (crosses indicate these). During one measurement at 1400 Hz (marked by a star) an apparent oscillatory component was observed at about 1360 Hz, near the frequency of the very narrow amplitude peak measured in that series. This was not an SOAE since it disappeared when the stimulus was switched off. Measurements at frequencies above 1700 Hz were also unstable.

Phase measurements show good stability over time: measurements made on different days almost all agreed within 0.1 cycle. The inherent accuracy of an individual measurement was about ± 0.01 cycle at best where the SFE was large, and poorer where the SFE was near the noise floor.

As found previously by Wilson (1980c) for tone-burst evoked OAEs, and by Schloth (1982) for SFEs, the phase curve is well-fitted by straight-line segments as shown: the dashed lines indicate the divisions. Phase ambiguities often occur at the dividing points, so that the connections are here arbitrary. The slopes (corrected for stimulus phase) are extracted to Table 5-1.

TABLE 5-1

SFE PHASE SLOPES

Frequency Range	Cycles/kHz	Group latency
850 - 1050	11.0	10.5 waves
1100 - 1180	11.8	13.4
1200 - 1375	8.0	10.2
1400 - 1525	7.3	10.5
1575 - 1725	5.9	9.8
1725 - 1950	5.1	9.3
1950 - 2100	7.3 or 13.6	14.6 or 27.1

These values agree well with other phase slopes and with latencies of click-evoked OAEs (Wilson 1980c, Kemp and Chum 1980b, Schloth 1982). The paucity (and instability) of data at the extreme high-frequency band mean that there is ambiguity here; however the lower phase slope in the table is much more plausible.

5.2.2.3 Suppression Tuning Curves

STCs for both 3-dB and 6-dB (in 1 case 10-dB) criteria are illustrated in Figs.5-7 and 5-8 for the different frequencies of SFEs. Most points are means of 3 determinations - in most cases the range of these was less than 3 dB, but at the notches this was sometimes up to 10 dB. The thresholds for the suppressor tones determined as attenuator settings during the measurements, and converted as per the STC levels, are shown on the same scale.

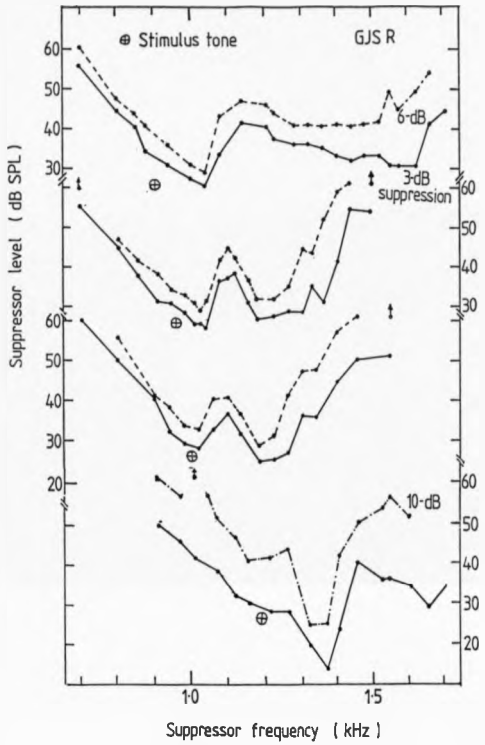

Fig.5-7 Suppression tuning curves for SFEs in GJS (right ear), 3-dB and 6-dB criterion (in one case 10-dB), for SFE frequencies (top to bottom) 910 Hz, 960 Hz, 1000 Hz, 1193 Hz. All 3-dB points are means of at least 2 (usually 3) determinations.

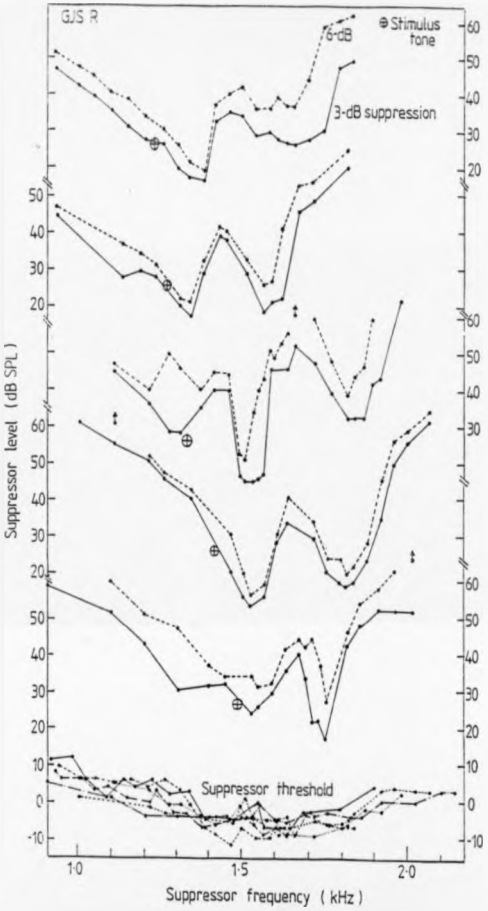

Fig.5-8 As for Fig.5-7, for SFE frequencies of 1220 Hz, 1260 Hz, 1320 Hz, 1405 Hz, 1480 Hz. Bottom: Threshold determinations for suppressor tones.

The main findings of these experiments are as follows.

(a) Multiple lobes in the STCs

It can be seen that many of the STCs for both criteria have 2 (and sometimes 3) lobes, typically 0.2-0.3 octaves apart, and separated by local maxima where suppression levels were up to 30 dB greater.

(b) Clustering of tips of the STCs

The tips of the STCs tend to be clustered near certain frequencies (see Table 5-2): these frequencies are thus efficient suppressors for a wide range of SFEs. However as can be seen in the table, this clustering is not absolute, and the lobe position does vary somewhat with SFE frequency.

TABLE 5-2

FREQUENCIES OF STC MINIMA

f_e	Frequency of minima (3 dB curves)
910	1040 1550?
960	1040 1180 1360
1000	1020 1180 1340?
1195	1375 1525? 1660
1220	1375 1525 1640
1260	1335 1550
1320	1300 1520 1850
1405	1510 1795
1480	1295 1520 1740 2000

(c) SFE phase changes under suppression by some frequencies

Whilst for most suppressor frequencies the SFE phase did not vary while being suppressed, for some there were shifts of up to 90°: these were always in the direction of increasing phase lag. The most marked effects occurred for suppressor frequencies at or near maxima in the STCs (i.e. where the SFE was hard to suppress) - as the level was increased the SFE vector would rotate with little or no amplitude change before suppression began.

(d) SFE enhancement by some 'suppressor' tones

With some particular frequencies of suppressor tone, as the level was increased, the SFE amplitude was at first not suppressed, but increased, by up to 6 dB. As with SFE phase shifts, this usually occurred at frequencies where the STC had a local maximum.

5.3 SUPPRESSION OF CLICK-EVOKED OAEs

A broadband click stimulus evokes all frequencies of EOAE at once. The spectrum of this cochlear echo may then be found by FFT, and changes in this spectrum caused by a suppressor tone can be observed. By this approach one can observe the effect of one frequency of suppressor on the whole frequency range of EOAEs, which is complementary to study of SFE suppression (giving the effect of a range of suppressor frequencies on one frequency of EOAE). The following series of experiments was carried out while the author was a guest scientist at the Institut für Elektroakustik, Technische Universität, München, and the results have appeared elsewhere (Sutton 1985).

5.3.1 Methods

A Knowles ID 812 miniature microphone and a small acoustical driver were built into a custom earmould, giving a small and reproducible enclosed volume. Vaseline was used to give a good seal into the meatus.

The electrical signal onto the driver was 1 cycle of a 2 kHz sine wave, triggered at a rate of about 45 per second. The spectral density of the acoustical signal was calculated as follows. The threshold of a 2-kHz continuous tone is 5 dB SPL in this ear. Threshold for a single cycle was found to be 20 dB higher. The spectrum of a single-cycle stimulus (pressure amplitude P_o, period T) has a maximum spectral density of P_oT (Randall 1977). For a just audible click this is thus -41 dB SPL/Hz, at 2 kHz. The spectrum of the click as measured in the ear canal (using the same analysis system as for the echoes) is shown (at an arbitrary level) in the top curve of Fig.5-10, and can be seen to be reasonably

uniform over the frequency range of interest (0.8-2.5 kHz). (For the actual response/stimulus ratio see §5.3.2.1).

The suppressor tone was fed from a Beyer DT48 earphone through plastic tubing. The microphone output was filtered (high pass at 0.5 kHz, followed by band pass 1-3 kHz) and amplified before being fed to a digital waveform averager with a 40 μs sampling interval. For most results 1000 responses (but for some series 4000) were averaged and the resulting waveform sent to an FFT computer (sampling interval 100 μs). Preliminary experiments showed that very little echo occurred after 20 ms in this ear; time-windowing of 4-20 ms was therefore generally used to exclude the initial signal and to optimise signal-to-noise ratio. This windowing convolves the EOAE spectrum with a sinx/x function with interlobe spacing 62.5 Hz (Kendall 1977): some of the spectral ripples observed may be due to this.

Cancellation of the suppressor tone

The suppressor signal must be eliminated from the averaged signal if the EOAE is to be clearly visible. This was achieved by varying the phase of the suppressor tone with respect to the stimulus: its phase was set to advance exactly 0.1 cycle between successive stimuli, thus ensuring complete cancellation for any multiple of 10 stimuli.

One possible drawback with this cancellation method - also applying to Kemp and Chum's (1980a) similar method where the suppressor phase is allowed to free-run - arises because EOAEs can be synchronised by external tones. It is conceivable that the suppressor might

synchronise part of the response (probably any late 'secondary' echoes which would be expected to be less strongly phase-linked to the click stimulus). Any EOAE thus synchronised would like the suppressor, disappear on averaging and would appear, wrongly, to have been suppressed. An attempt was made to validate the cancellation procedure by comparing results derived using it with those derived using a different procedure in which the suppressor phase was held constant (relative to the stimulus), and cancellation performed subsequently by subtraction from the total averaged signal. This check proved inconclusive due to problems of overloading and of adequately cancelling the suppressor. The phase-variation technique was therefore employed in all the experiments due to lack of a practical alternative.

Experiments were carried out in a quiet test booth. However, spectral peaks due to mains harmonics from the equipment (particularly at 1600 Hz) were occasionally observed owing to imperfect cancellation. Checks were also made against overloading in any part of the system.

For all results click stimulus (S) magnitudes are expressed in sensation levels (i.e. dB above subjective threshold), denoted by SL_s. The sensation-level magnitude of the suppressor (M for masker) tone is denoted by SL_m and its frequency by f_m. An unsuppressed EOAE spectrum was always recorded before runs with increasing levels of suppressor. In many cases another 'unsuppressed' spectrum was recorded at the end of the series: there were no significant differences between these 'before' and 'after' spectra and the mean is shown in these cases

(details are given in each figure caption). At some of the higher SL_M values cancellation was not perfect and a residual peak results at f_M.

5.3.2 Results

5.3.2.1 EOAEs without suppressors

The basic characteristics of the EOAE in this ear - in particular its dependence on stimulus level and its variability - were investigated first. Fig.5-9 illustrates the level-dependence. At top are shown averaged waveforms (excluding 0 to 4 ms where the averager was overloaded) for SL_E=16 and 40 dB (respectively -25 and -1 dB SPL/Hz). Below are shown the EOAE spectra for SL_E=10 dB up to 40 dB in 6 dB steps (details of extra filtering and time windowing are given in the figure caption). Calibration of responses relative to the stimulus was found by comparison of the echo FFTs with that for the click stimulus. The EOAE response was maximally -18 dB with respect to stimulus for the 16 dB SL clicks (at 1250 Hz), and -28 dB for the 40 dB SL clicks (at 1000 Hz).

Several distinct areas of strong response can be seen each with different level dependencies. For the region below 1300 Hz the response grows approximately linearly with level, and saturates strongly at high levels. Above 1400 Hz responses grow non-linearly even at SL_E=16 dB, and most again saturate at the higher levels. The 1600-1900 Hz region is an exception to the general pattern showing a roughly equal (though less than 1:1) growth between each curve and still increasing at SL_E=40dB.

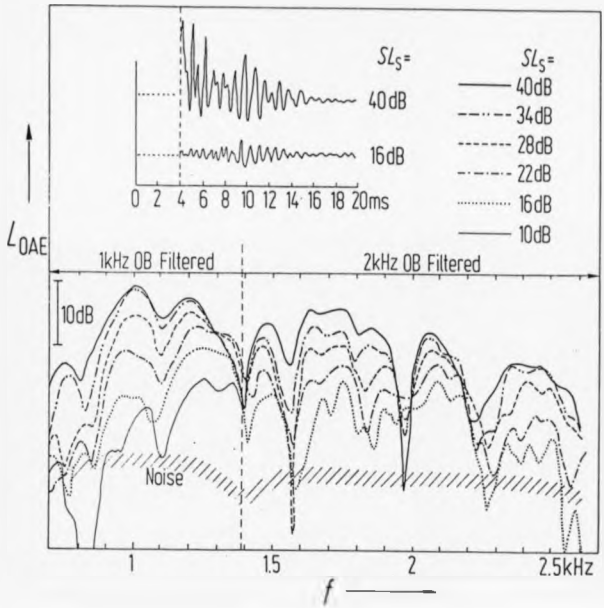

Fig.5-9 Top: Waveforms of EOAEs for SL_e=16 dB and 40 dB. **Bottom:** Spectra of EOAEs for SL_e=16 to 40dB in 6-dB increments. Responses in 0.8-1.4 kHz are 1 kHz octave band filtered with a 7-20 ms time window; responses above 1.4 kHz are 2 kHz octave band filtered with a 4-20 ms time window. 1000 waveforms were averaged for each spectrum. The approximate position of the noise floor is also shown.

It can be seen that the precise location of each peak and dip changes systematically with level, but that the direction of change is not the same everywhere. Also with increasing level, the deep notches at 1400 Hz, 1950 Hz and 2300 Hz tend to deepen and sometimes cross lower SL_e curves: where this crossing occurs there must be a negatively sloping input-output function. This effect is also clearly seen in Fig.5-10 at 1.4 kHz and will be discussed later. To check that these deep notches were not artefactual, experiments were made changing variously time-windowing, sampling rate and probe assembly: the notches always occurred at identical positions.

Variability and repeatability of the unsuppressed EOAE were assessed by examining a number of spectra (about 20) recorded over a period of several days. Median, 10th and 90th centiles of these data are illustrated in Fig.5-10. It is not surprising that variability is clearly much less at SL_e= 40 dB than at 16 dB, this reflecting the influence of the noise floor (illustrated). There was also some longer-term variability of the EOAE spectrum of up to 6 dB over the 4 week period of these experiments, probably due to changes in middle ear function.

5.3.2.2 Suppression of echoes with SL_e=40dB

Fig.5-11 shows the EOAE spectra for SL_e=40 dB under suppression by tones of frequencies from 1000 to 2000 Hz and levels of 40 and 50 dB SL. No significant suppression was observed for SL_m=20 or 30 dB. The repeatability of these results was high, as illustrated in (a) of this figure where the spread of results over 3 runs for SL_m=50 dB is shown.

The following features can be noted:

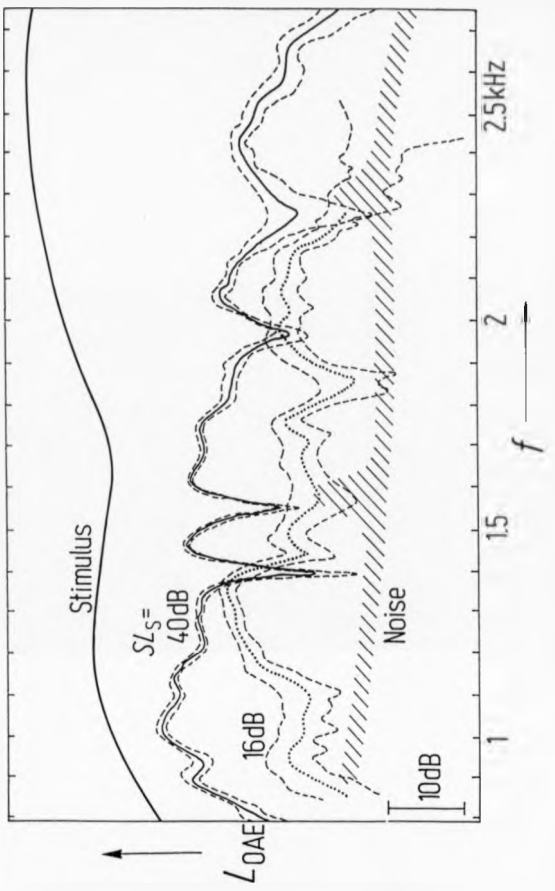

Fig.5-10 **Top:** Spectrum of stimulus click as recorded in the meatus (shown at an arbitrary level). **Bottom:** Repeatability of EOAE spectra (no suppression) at SL_S=16 dB and SL_S=40 dB (N=17 and 21 spectra respectively). Medians are shown (solid or dotted), with 10th and 90th centiles (dashed). The noise floor shown is the top 80th centile of 11 spectra recorded under the same conditions, but with no stimulus.

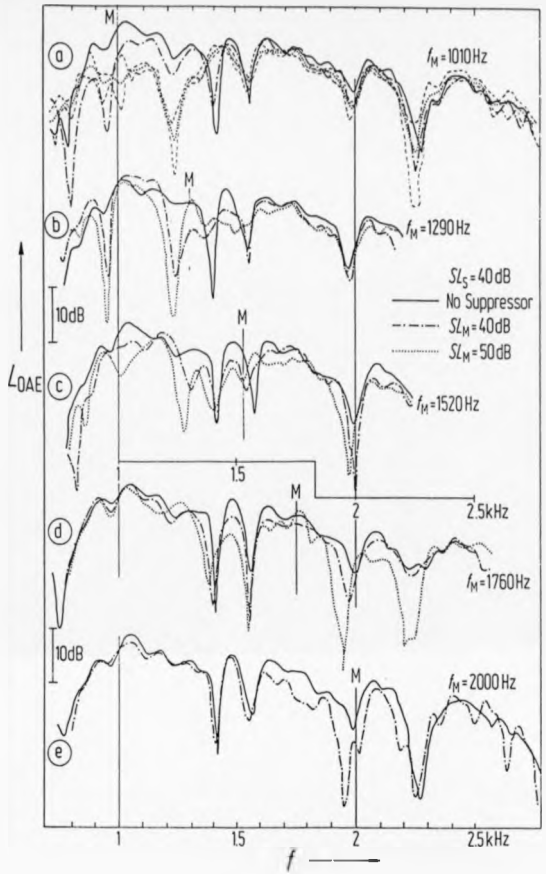

Fig.5-11 Effects of suppressor tones (M) on EOAE spectrum for SL_S=40 dB. Top to bottom: (a)f_M=1010 Hz, (b)f_M=1290 Hz, (c)f_M=1760 Hz, (d)f_M=2000 Hz, with levels SL_M=40 and 50 dB. 1000 waveforms averaged for each spectrum. 'No suppressor' curves are the means of 2 (for (b), 3) runs. In (a) SL_M=50 dB is the mean of 3 runs, and the range is shown by dashed lines; in (b) SL_M=40 dB is the mean of 2 runs.

(1) Suppression effects seem to spread considerably above and below f_M. Although the effects above f_M are not surprising, those below are, if one considers the excitation pattern of the suppressor tone in the cochlea.

(2) The maximum suppression at low SL_M tends to occur a little below f_M, analogous to the offset of STCs for SFEs. This is most clearly seen for f_M=1010 Hz, 1290 Hz and 2000 Hz at SL_M=40 dB.

(3) There are signs of periodicity in many of the suppressed spectra, which implies that there are, for each f_M, frequencies which are relatively sensitive and frequencies which are relatively insensitive to suppression. In particular the response at 900 Hz, 1250 Hz and 1950 Hz appear to be readily affected by several of the suppressors. The exception to this is for f_M=1520 Hz, where 900 Hz, 1200 Hz and 1400 Hz are 'insensitive' whilst 1050 Hz, 1250 Hz and 1500 Hz are 'sensitive'. The region around 1500 Hz appears to be sensitive to all the suppressors except f_M=2000 Hz. A 'sensitive' frequency would be expected to correspond to a broadly tuned STC for that EOAE frequency.

(4) The spectral notch at 1400 Hz shifts under suppression, and sometimes becomes a peak, thus giving rise to an apparent enhancement of EOAE here.

5.3.2.3 Suppression of echoes with SL_e=16 dB

The same wide range of suppressor frequencies was used, with SL_M from 20 to 50 dB. A selection of the results is shown in Fig.5-12, for which 4000 (rather than 1000) responses were averaged.

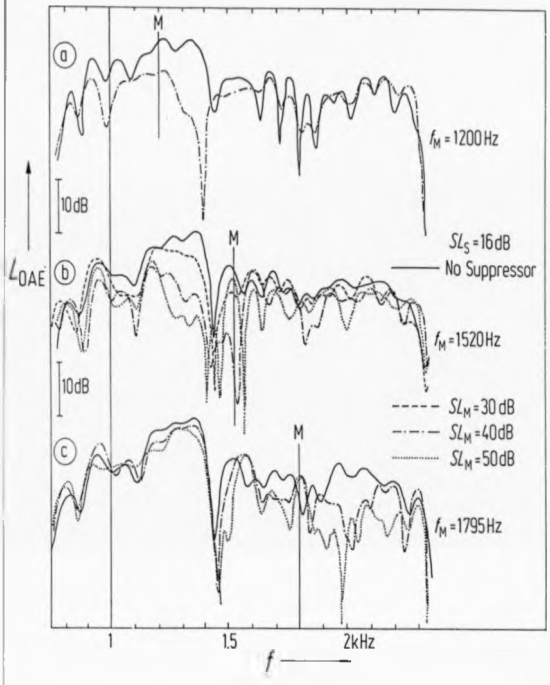

Fig.5-12 Effects of suppressor tones (M) on EOAE spectrum for SL_S=16 dB. Top to bottom: (a)f_M=1200 Hz, (b)f_M=1520 Hz, (c)f_M=1795 Hz, with levels SL_M=30 dB, 40 dB, 50 dB. 4000 waveforms were averaged for each spectrum.

As with SL_e=40 dB there is evidence of sensitive and insensitive frequency regions, notably sensitive regions near 1350 and 1950 Hz. On the other hand maximal suppression in this series of results does not usually occur below f_m except with f_m=1520 Hz. Again a shift in the position of the spectral peaks and notches can be seen.

5.3.2.4 More detailed study of 1100 to 1400 Hz region

The concentration of effects in this frequency region seemed to merit more attention, so a detailed series of measurements was made at SL_e=16 dB with suppressor frequencies spaced about 45 Hz apart and at levels SL_m=20 dB, 30 dB and 40 dB. The order of testing of f_m was randomised, and runs were performed with increasing SL_m.

A selection of results is shown in Fig.5-13; sections of Fig.5-12 are also relevant, in particular (a) and (b). It appears that for $f_m$below 1150 Hz (i.e. below the region) the curves for different SL_m are approximately parallel, - that is, the whole region is affected about equally by the suppressor. When however f_m is within or above the region (i.e. f_m>1150 Hz, see also Fig.5-12b) the spectral shape of the response changes with frequencies near f_m being preferentially suppressed. Maximal suppression does not occur at f_m but at the nearest sensitive frequency.

Suppression effects centre on 1335 Hz, this being not only the most effective suppressor frequency (Fig.5-13d) but also the frequency most sensitive to remote influence. If these results were transcribed to a set of STCs (for the suppression of particular EOAE frequencies) the

Fig.5-13 Effects of suppressor tones (M) on strong part of EOAE spectrum for SL_S=16 dB. (a)f_M=1010 Hz, (b)f_M=1105 Hz, (c)f_M=1290 Hz, (d)f_M=1335 Hz, (e)f_M=1380 Hz, with levels SL_M=20, 30, 40 dB. 1000 waveforms averaged for each spectrum. 'No suppressor' curves are the means of 2 runs.

tips of these would nearly all occur at or near 1335 Hz (cf §5.2, and Kemp and Chum 1980b fig.8). Periodicity is also apparent in the suppressed spectra which would correspond to multiple lobes in STCs.

5.4 DISCUSSION

5.4.1 Comparison of results for SFEs and click-evoked EOAEs

Since it appears that the OAE mechanism behaves linearly for low levels (see §1.2.7), one would expect a similar frequency distribution for click-evoked echoes and SFEs. However a different view was expressed by Ruggero et al (1983), predicting that SFEs would occur uniformly at all frequencies in contrast to click-evoked OAEs.

Before attempting a comparison one must first ask what level of click stimulus is equivalent to a given level of continuous tonal stimulation. Earlier the spectral density of the click stimulus was calculated as -25 and -1 dB SPL/Hz (for SL_e=16 and 40 dB) at 2 kHz, falling by 4 dB at 1 kHz. In fact the effective excitation for the EOAE generator would be the level in the bandwidth of the auditory filter, so that a more relevant measure would be in dB SPL/critical band. Taking 200 Hz as an average critical bandwidth for this frequency region (Evans and Wilson 1973) gives 17 and 41 dB SPL/critical band, which values are then roughly the same as the sensation levels. Hence a given SL of click-evoked echo should be comparable with the same SPL of continuous tone evoked SFE.

In fact the SFE amplitude (Fig.5-5) (for 26 dB SPL at 1 kHz) does compare quite well with and the SL_e=28dB echo spectrum (Fig.5-9), showing many of the same features. The overall shape around 0.8-1.3 kHz is similar, and the response/stimulus ratio (at 1.2 kHz) is nearly identical for both (about -18 dB). In both paradigms, four areas of strong response seem to be identifiable (around 1000Hz, 1200Hz,

1450 Hz, and 1700 Hz) and the spectral notch at 1550 Hz is present. There are however differences - for example the notch at 1400-1425 Hz in the echo spectrum appear to be at 1300 Hz in most SFE measurements. However it was very clear that SFEs were very unstable and variable in this frequency region, with amplitude and phase seen to periodically switch between 2 states, and for 2 sets of data the SFEs do show a notch at 1400 Hz.

The one set of echo suppression results (Fig.5-13) with suppressors closely enough spaced to allow the form of STCs to be deduced implies a clustering of STC tips near 1335 Hz, for emissions between 1200 and 1400 Hz: this agrees with results for the SFEs.

5.4.2 Summary of main findings

The key findings of these suppression studies are:

1. Deep notches in the amplitude spectrum of EOAEs.
2. Multiple-lobed STCs for both SOAEs and SFEs, with low-threshold suppression occurring for some suppressors well above the emission frequency.
3. Clustering of the lobes of the STCs for SFEs near certain frequencies.
4. Phase changes of SFE under suppression.
5. Enhancement of SFE amplitude (by up to 6 dB) at some frequencies before suppression.

The implications of these results for models of OAE generation and the irregularity hypothesis are discussed below. It will be argued that many of the above findings are explicable if one assumes that in some

ears there are 2 or more closely-spaced irregularities along the cochlear partition, and that they tend to support the 'distributed activity', rather than the 'localised resonator', interpretation of irregularities (see §1.3.1). Alternatively, the multiple lobes might arise from the influence of a reverse travelling wave on the cochlear excitation pattern.

5.4.3 Implications of spectral notches

Similar deep spectral notches are seen in other data (e.g. Wilson 1980c, Kemp and Chum 1980a), but were usually assumed to be the result of fine structure from whole-cochlear resonances (§1.2.13). However the results from the echo measurements here exclude this explanation, because (a) the time-windowing used minimises the secondary echoes, and (b) the notches were more marked at 40 dB SL than 16 dB SL, contrary to what one would expect (see Kemp and Chum 1980b).

In fact the spectral notches are reminiscent of an interference phenomenon, and it is proposed that they arise because of cancellation at these frequencies between the responses from different strong response regions. This implies closely-spaced irregularities. The frequency-shift of the notches with stimulus level changes and under suppression is consistent with this model: as the relative contribution of the two spectral regions alters, so the frequency at which cancellation occurs also alters. One can also account for the negative input-output slopes at the notches, and the apparent stimulatory (enhancement) effect of a suppressor tone seen at the notch in the echo results.

5.4.4 Implications of suppression by frequencies higher than the emission.

The results demonstrate - for SOAEs, echoes and SFEs - low-threshold suppression effects by tones with frequencies well above the emission (frequency ratios up to 1.4 for the SFEs, 1.6 for TAB's SOAE), whose travelling wave excitation should be minimal at the emission frequency 'place' (due to the steep apical slope of the pattern). This poses a severe problem for models in which the OAE generator is viewed as a single localised resonator at that place. One way round this is to suggest that several of these local regions are being fed by some more central energy source with limited output; thus a suppressor peaking in one region could rob energy from another region and thus cause suppression there. However it is not at all clear how this could occur physically. On the other hand if activity is distributed over a considerable length of the cochlea (see §1.3.2), then these suppression effects are much more easily explained.

5.4.5 Multiple lobes in STCs and clustering of tips

Previously the two reports of multiple-lobed STCs that were definitely not due to frequency-shifting effects (Evans et al 1981, Wilson and Sutton 1983) appeared to be merely odd exceptions to the general rule of single-lobed STCs with sharp high-frequency cutoffs (Wilson 1980c, Kemp and Chum 1980b, etc). No multiple lobes have previously been found for EOAEs. In contrast the results here imply that multiple lobes may be common for both EOAEs and SOAEs.

Clustering of the STC tips was also observed by Kemp and Chum (1980b) for SFEs, but they found no phase changes during suppression. Outside the realm of overstimulation, only Kemp and Chum (1980a) have previously reported (tentatively) a possible increase in EOAE amplitude (enhancement or stimulatory effect) caused by an external tone.

Three possible explanations for these effects will be considered.

5.4.6 Multiple lobes - could they arise from cochlear resonances?
One must first consider the possibility that the multiple lobes (and clustering) arise from whole cochlear resonances (due to multiple internal reflection - see §1.2.13): this would lead to some suppressors having greater effective intracochlear levels, and hence being more effective, than others. Indeed Long (1984), studying masking effects on probe tones near a threshold minimum, found clear multiple lobes in psychophysical tuning curves almost certainly arising from this effect. However this explanation seems inadequate to explain the results here for 3 reasons:

1. One would expect the frequency spacing of the lobes to be the same as for other fine structure (about 100 Hz), as it was in Long (1984), but this is not the case here (see Table 5-2). Nor are the lobe frequencies absolutely invariant as one would expect.

2. Fine structure variations are maximally about 15 dB at low signal levels, and rapidly smooth out at higher levels (Fig.12 of Wilson 1980c, Fig.7 of Kemp 1979b, Fig.4 of Long 1984). The peak-to valley ratios here were up to 30 dB, for 20-30 dB SL probes.

3. Measurements of tone threshold during the SFE experiments (Fig.5-9) revealed little fine structure, and no obvious correlation with the frequencies of the STC minima.

5.4.5 Multiple lobes - possible explanation based on closely-spaced irregularities.

Many of these effects could be due to the presence of 2 (or more) irregularities close together in the cochlea. For a stimulus frequency whose excitation pattern spanned both irregularities, the SFE would be made up of contributions from both. In the simplest example, if the contributions from the 2 irregularities are in phase, then the most effective suppressor frequencies would be those centred on the irregularities, leading to a double-lobed STC. One would also expect clustering of STC tips near 'irregularity frequencies'. For other SFE frequencies the contributions might partially cancel: certain suppressors could then differentially knock out one contribution resulting in an enhancement of the SFE before suppression.

In other examples the exact effect will depend on phase relationships. In general one can picture the SFE as a vector sum of elemental contributions. If the maximal effect of the suppressor were on an element not parallel to the resultant SFE, the phase of the SFE would change during suppression.

This model does not however explain the coincidence of SFE enhancements with maxima of the STCs, nor could it account for the multiple lobes of

Evans et al (1981) or Wilson and Sutton (1983) where the lobe separations were much greater (frequency ratios of up to 3.0).

5.4.6 Multiple lobes - possible explanation based on a standing wave. If reflection of the cochlear travelling wave took place from an irregularity, causing a reverse travelling wave (§1.3.2), then for a tonal input this would lead to a standing wave pattern (at least partial) along the BM, and nulls and maxima in the excitation pattern. Based on the distributed activity model, the contribution to the EOAE would be least from the nulls and most from the maxima, so that suppressor tones peaking at these nulls would be less effective than those peaking at the maxima: hence multi-lobed STCs might be seen. However the spacing of the nulls (and hence lobes) would be determined by the BM phase basal to the irregularity, and one would not expect clustering of the STC tips. Nor would one expect EOAE spectral notches to arise.

More explicit modelling would be useful before ruling out this standing wave hypothesis, but the multiple-irregularity model (§5.4.5) seems able to account for more of the observations, making this more attractive as a single hypothesis.

5.4.7 Significance of offset of STC tip from emission frequency
For both SOAEs and SFEs, the main STC tip is consistently above the emission frequency, in line with nearly all other data in the literature. As Kemp and Chum (1980b) pointed out, suppression reveals tuning only up to the nonlinearity, and not any extra filtering after

it, so that the offset could be explained by an additional linear filter after the nonlinearity tuned to a consistently slightly lower frequency. This argument still appears to hold even with this extra filtering provided by an active feedback onto the cochlear mechanical response. If this were so however one would expect STCs to reflect the broad tuning *prior* to this extra filtering, whereas in fact STCs are mostly very sharply tuned. This suggests that the nonlinearity responsible for suppression is *after* the active sharpening process. It may also be noted that the suggestion of a lower-tuned 'second filter' process is opposite to Davis' (1983) theory of the cochlear amplifier operating on the apical slope of the broadly-tuned passive travelling wave pattern (which means a higher-tuned 'second filter').

An alternative explanation has been put forward by Ruggero et al (1983) who suggested that the SOAEs arise from small localised regions of pathology and that the STC tip is offset into the better threshold region. This idea is however based on the assumption that all SOAEs are associated with minor pathology, which may be vigorously disputed from other evidence (see §4.6.4).

6. OVERALL CONCLUSIONS

1. Many of the features of the cochlear echo - notably its long delay and frequency concentration into narrow bands - can be successfully modelled on the basis of a summed response approach, in which activity is distributed over a considerable length of the cochlea, and with randomly-occurring irregularities of structure (or mapping or sensitivity) disturbing the normal phase cancellation. The latency of the cochlear echo on this model is primarily the result of cochlear filtering delays, but augmented because the distributed activity leads to cancellation of the early part of the response.

2. SOAEs were found in 53% of a small sample of normal-hearing subjects (42% of ears), consistent with the figure of 1/3 to 1/2 found by others. SOAEs appear to be significantly more prevalent in females: if this is due to hormonal factors then one might expect the number of SOAEs in a given female ear to vary with the menstrual cycle - this would be worth studying. Analysis of the distribution of SOAEs relative to the noise floor provisionally suggests that the prevalence figure would not increase much with a more sensitive detection system.

3. Most SOAEs have frequencies between 1 and 2 kHz and levels below 10 dB SPL in the sealed meatus, although a few are stronger (up to 24 dB SPL here) with frequencies up to 10 kHz. Most are inaudible, probably due to masking by ambient noise, and possibly adaptation.

4. In these studies SOAEs were not found at any frequency where the hearing loss exceeded 20 dB, and there was no consistent association of SOAEs with local pathology, at least as revealed by history or Békésy audiograms. This and other evidence suggests that they should be viewed as an epiphenomenon of normal hearing mechanisms, not as pathological.

5. Currently the evidence for a different type of 'pathological' SOAE requiring a different mechanism is scanty, despite the occasional subject found with audiometric notches at the SOAE frequency (Glanville et al 1971 and Wilson and Sutton 1983, Huizing and Spoor 1973, subject AM here). An interesting experiment in such a case would be to see if the audiometric notch disappeared when the SOAE was abolished by aspirin.

6. SOAEs are sometimes found in groups of three in the frequency relation $f_1=2f_2-f_3$. The behaviour of the CDT SOAE would be expected to differ from others, and studying this might prove useful in probing the generation and propogation of CDTs in the cochlea (Burns et al 1984).

7. Some cases of clinical tinnitus have been found with SOAE correlates. Such subjects may not have completely normal audiograms, but all 3 cases found here had normal hearing at least at the tinnitus pitch, and pure-tonal (or multi-tonal) tinnitus. Whilst SOAEs will only be implicated in a small proportion of tinnitus cases seen in ENT departments, they are worth looking for given these presenting factors. Cochlear mechanical tinnitus seems not usually to be very troublesome. Most tinnitus arises from other, non-vibratory, sources, and is

sometimes associated with audiometric notches or the border between normal and damaged regions, consistent with Kiang et al's (1970) hypothesis.

8. Deep notches in the high-level amplitude spectra of cochlear echoes are suggestive of cancellation effects between different frequency regions of strong response. It is suggested that these arise from the presence of several distinct irregularities closely spaced along the cochlear partition.

9. Suppression tuning curves have been found with marked multiple lobes for SOAEs and SFEs. Such curves are not therefore confined to high-level or possible pathological SOAEs, but may occur for normal low-level OAEs. The lobes of the STCs for the SFEs tended to cluster near certain frequencies. These results do not seem explicable on the basis of 'whole cochlear resonances'. Other effects of the external tone, including amplitude increases and phase shifts have been found with SFEs. Again these effects might be due to the presence of closely-spaced irregularities in the cochlea. Alternatively, the multiple lobes might be plausibly explained if reflection of the travelling wave was occurring causing a standing wave and nulls in the excitation pattern, but this would not seem to predict the lobe clustering. More data are required to see if such patterns are more common than previously suspected.

10. Suppression was often found by tones with frequencies considerably above that of the OAE. This implies that the activity responsible for a

(single frequency) OAE is distributed over a considerable length of the cochlea, rather than being confined to single localised resonators, although the sharp high-frequency cutoffs in most STCs in the literature might favour the latter.

11. Results here confirm that the main lobe of the STC is usually tuned just above the emission frequency. The explanation that this is due to extra filtering following the nonlinearity (Kemp and Chum 1980b) is hard to reconcile with the sharpness observed for most STCs. The implication of this explanation - that the active sharpening process is tuned consistently slightly lower than the first passive filter - is opposite to the model of Davis (1983), which suggests that it is consistently higher (maximally operating on the apical slope of the passive travelling wave pattern).

ACKNOWLEDGEMENTS

I would like to thank my supervisor Dr Pat Wilson for much help and many invaluable discussions during this work, and Prof E Zwicker who supervised the work (§5.3) during my 3 months as a guest researcher in Munich. Thanks are also due to Mr Kapur, Mr Little, and Mr Stuart, consultant ENT surgeons of the North Staffordshire Central Outpatients' Department, for diagnosing and sending their tinnitus patients. The work at Keele was supported by the Science and Engineering Research Council, and that at Munich by Deutsche Forschungsgemeinshaft through Sonderforschungbereich 50, Kybernetic, München.

Warmest thanks also to my friends, particularly Ingrid and Linda, whose encouragement kept me going.

REFERENCES

ANDERSON SD (1980). Some ECMR properties in relation to other signals from the auditory periphery. Hear Res 2, 273-296.

ANDERSON SD AND KEMP DT (1979). The evoked cochlear mechanical response in laboratory primates - a preliminary report. Arch Otorhinolaryngol 224, 47-54.

BAILEY NTJ (1959). Statistical methods in Biology. English Universities Press, London.

BEKESY G VON (1960). Experiments in Hearing. McGraw-Hill, New York.

BROWN AM (1986). Some experimental observations of responses evoked from the cochlea during two-tone stimulation. ORL 48, 124-134.

BROWN AM AND KEMP DT (1984). Suppressibility of the $2f_1$-f_2 stimulated acoustic emissions in gerbil and man. Hear Res 13, 29-37.

BROWN AM AND KEMP DT (1985). Intermodulation distortion in the cochlea: could basal vibration be the major cause of round window CM distortion. Hear Res 19, 191-198.

BURNS EM (1984). A comparison of variability among measurements of subjective tinnitus and objective stimuli. Audiology 23, 426-440.

BURNS EM STRICKLAND EA TUBIS A AND JONES K (1984). Interactions among spontaneous oto-acoustic emissions. I Distortion products and linked emissions. Hear Res **16**, 271-278.

CARLIER E AND PUJOL R (1982). Sectioning the efferent bundle decreases cochlear frequency selectivity. Neurosci Letters **28**, 101-106.

CIANFRONE G AND MATTIA M (1986). Spontaneous otoacoustic emissions from normal human ears: preliminary report. Scand Audiol Suppl.25 121-127.

CITRON L (1969). Observations on a case of objective tinnitus. Excerpta Med Int Cong Ser **189**, 91.

CLARK WW KIM DO ZUREK PM AND BOHNE BA (1984). Spontaneous oto-acoustic emissions in chinchilla ears: correlation with histopathology and suppression by external tones. Hear Res **16**, 299-314.

COHEN MF (1982). Detection threshold microstructure and its effect on temporal integration data. J Acoust Soc Am **71**, 405-409.

CRAWFORD AC AND FETTIPLACE R (1981). An electrical tuning mechanism in turtle cochlear hair cells. J Physiol **312**, 377-412.

DALLOS P (1981). Cochlear physiology. Ann Rev Psych **32**, 153-190.

DAVIS H (1983). An active process in cochlear mechanics. Hear Res **9**, 79-90.

DAVIS H AND SILVERMAN SR (1970). Hearing and Deafness (3rd edition). Holt Rinehart and Winston, New York.

DE BOER E (1980). Auditory Physics. Physical principles in hearing theory I. Physics Reports **62**, 87-174

DE BOER E KAERNBACH C KÖNIG P AND SCHILLEN T (1986). Forward and reverse waves in the one-dimensional model of the cochlea. Hear Res **23**, 1-8

DENGERINK JE DENGERINK HA SWANSON S THOMPSON P AND CHERMAK GD (1984). Gender and oral contraceptive effects on temporary auditory effects of noise. Audiology **23**, 411-425.

DIEPENDAAL RJ VIERGEVER MA AND DE BOER E (1986). Are active elements necessary in basilar membrane mechanics? J Acoust Soc Am **80**, 124-132.

DOUEK E AND REID J (1968). The diagnostic value of tinnitus pitch. J Laryngol Otol **82**, 1039-1042.

DUIFHUIS H (1973). Consequences of peripheral frequency selectivity for nonsimultaneous masking. J Acoust Soc Am **54**, 1471-1488.

ELLIOT E (1958). A ripple effect in the audiogram. Nature **181**, 1076.

ENGSTRÖM H AND GRAF W (1952). Recording of objective tinnitus. Acta Otol **41**, 228-234.

EVANS EF (1977). Frequency selectivity at high signal levels of single units in cochlear nerve and nucleus. In: Psychophysics and Physiology of Hearing (Evans EF and Wilson JP, eds), Academic Press, 185-192.

EVANS EF AND WILSON JP (1973). The frequency selectivity of the cochlea. In: Basic mechanisms in hearing (Møller A, ed). Academic Press, 519-551.

EVANS EF, WILSON JP AND BORERWE TA (1981). Animal models of tinnitus. In: Tinnitus (Evered D and Lawrenson G, eds). Pitman, London, 108-138.

EVERED D AND LAWRENSON G (EDS) (1981). Tinnitus (Ciba Symposium No 85). Pitman Medical, London.

FELDMANN H (1971). Homolateral and contralateral masking of tinnitus by noise-bands and pure tones. Audiol **10**, 138-144.

FLETCHER H (1940). Auditory Patterns. Rev Mod Phys **12**, 47-65.

FLOTTORP G (1953). Pure-tone tinnitus evoked by acoustic stimulation: the idiophonic effect. Acta Otol **43**, 396-415.

FORMBY C AND GJERDINGEN DB (1981). Some systematic observations on monaural diplacusis. Audiol **20**, 219-233.

FOWLER EP (1940). Head noises: significance, measurement and importance in diagnosis and treatment. Arch Otolaryngol. **32**, 903-914.

FOWLER EP (1944). Head noises in normal and disordered ears. Significance, measurement, differentiation and treatment. Arch Otol **39**, 498-503.

FRITZE W (1983). On the frequency-distribution of spontaneous cochlear emissions. In: Hearing - Physiological basis and Psychoacoustics (Klinke R and Hartmann R, eds). Springer-Verlag. 77-81.

FRITZE W AND KÖHLER W (1986). Our present experience on spontaneous cochlear emissions. Scand Audiol Suppl 25, 129-137.

GLANVILLE JD COLES RRA AND SULLIVAN BM (1971). A family with high-tonal objective tinnitus. J Laryng Otol **85**, 1-10.

GLASGOLD A AND ALTMANN F (1966). The effect of stapes surgery on tinnitus in otosclerosis. Laryngoscope **76**, 1524-1532.

GOLD T AND PUMPHREY JR (1948). Hearing I. The cochlea as a frequency analyser. Proc Roy Soc B **135**, 462-491.

GOLD T (1948). Hearing II. The physical basis of the action of the cochlea. Proc Roy Soc B **135**, 492-498.

GOLDSTEIN JL BAER T AND KIANG NYS (1971). A theoretical treatment of latency, group delay and tuning characteristics for auditory nerve responses to clicks and tones. In: Physiology of the auditory system (Sachs MB, ed) National Educational Consultants, Baltimore. 133-141.

GOODWIN PE (1979). The loudness of tinnitus. PhD thesis, University of Denver, Colorado.

GRANDORI F (1985). Nonlinear phenomena in click and toneburst evoked oto-acoustic emissions from human ears. Audiol **24**, 71-80.

GREEN DM WIER C AND WIGHTMAN (1975). Gold and Pumphrey revisited, again. J Acoust Soc Am **57**, 935-938.

HAZELL JWP (1979). Tinnitus. In Scott-Brown's diseases of the ear, nose and throat (4th edition), Vol 2 (Ballantyne J and Groves J, eds). Butterworth, Sevenoaks. 81-91.

HAZELL JWP (1984). Spontaneous cochlear acoustic emissions and tinnitus. Clinical experience in the tinnitus patient. J Laryngol Otol Suppl 9, 106-110.

HELLER MF AND BERGMAN M (1953). Tinnitus Aurium in normally hearing persons. Ann Otol Rhinol Laryngol **62**, 73-83.

HINCHCLIFFE R (1959). The threshold of hearing of a random sample rural population. Acta Otol **50**, 411.

HOLTON T AND HUDSPETH AJ (1983). A micromechanical contribution to cochlear tuning and tonotopic organisation. Science **222**, 508-510.

HOUSE JW AND BRACKMANN DE (1981). Tinnitus : surgical treatment. In Tinnitus (Evered D and Lawrenson G, eds). Pitman, London, 204-211.

HOUSE PR (1981). Personality of the tinnitus patient. In: Tinnitus (Evered D and Lawrenson G, eds). Pitman, London, 193-198.

HUBBARD AE AND MOUNTAIN DC (1983). Alternating current delivered into the scala media alters sound pressure at the eardrum. Science 222,510-2

HUIZING EH AND SPOOR A (1973). An unusual type of tinnitus. Arch Otolaryngol **98**, 134-136.

INSTITUTE OF HEARING RESEARCH (1981). Epidemiology of tinnitus. In: Tinnitus (Evered D and Lawrenson G, eds). Pitman, London, 16-34.

JASTREBOFF PJ AND SASAKI CT (1986). Salicylate-induced changes in spontaneous activity of single units in the inferior colliculus of the guinea pig. J Acoust Soc Am **80**, 1384-1391.

JOHNSEN NJ AND ELBERLING C (1982a). Evoked acoustic emissions from the human ear. I. Equipment and response parameters. Scand Audiol **11**, 3-12.

JOHNSEN N AND ELBERLING C (1982b). Evoked acoustic emissions from the human ear. II. Normative data in young adults and influence of posture. Scand Audiol **11**, 69-77.

JOHNSEN N BAGI P AND ELBERLING C (1983). Evoked acoustic emissions from the human ear. III. Findings in neonates. Scand Audiol **12**, 17-24.

JOHNSTONE BM TAYLOR KJ AND BOYLE AJ (1970). Mechanics of the guinea pig cochlea. J Acoust Soc Am **47**, 504-509.

JONES K TUBIS A LONG GR BURNS EM AND STRICKLAND EA (1986). Interactions among multiple spontaneous oto-acoustic emissions. In: Peripheral Auditory Mechanisms (Allen JB et al, eds), Springer-Verlag, 266-273.

KEMP DT (1978). Stimulated acoustic emissions from within the human auditory system. J Acoust Soc Am 64, 1386-1391.

KEMP DT (1979a). Evidence of mechanical nonlinearity and frequency-selective wave amplification in the cochlea. Arch Oto-Rhino-Laryngol. 224, 37-45.

KEMP DT (1979b). The evoked cochlear mechanical response and the auditory microstructure - evidence for a new element in cochlear mechanics. Scand Audiol Suppl 9, 35-47.

KEMP DT (1980). Towards a model for the origin of cochlear echoes. Hearing Research 2, 533-548.

KEMP DT (1981). Physiologically active cochlear micromechanics - one source of tinnitus. In: Tinnitus (Evered D and Lawrenson G, eds), Pitman, London. 54-76.

KEMP DT (1982). Cochlear echoes - implications for noise-induced hearing loss. In: New perspectives in noise-induced hearing loss (Henderson D et al, eds), Raven Press, New York, 189-207.

KEMP DT (1986). Otoacoustic emissions, travelling waves and cochlear mechanisms. In: Cellular mechanisms in hearing (Flock A and Wersäll J, eds), Elsevier, Amsterdam, 95-104.

KEMP DT BRAY P ALEXANDER L AND BROWN AM (1986). Acoustic emission cochleography - practical aspects. Scand Audiol Suppl 25, 71-95.

KEMP DT AND BROWN AM (1983). A comparison of mechanical nonlinearities in the cochleae of man and gerbil from ear canal measurements. In: Hearing - Physiological bases and psychophysics (Klinke R and Hartmann R, eds) Springer-Verlag, 82-88.

KEMP DT AND BROWN AM (1986). Wideband analysis of otoacoustic intermodulation. In Peripheral Auditory Mechanisms (Allen JB et al, eds), Springer-Verlag, 306-313.

KEMP DT AND CHUM R (1980a). Properties of the generator of stimulated acoustic emissions. Hearing Reseach 2, 213-232.

KEMP DT AND CHUM R (1980b). Observations on the generator mechanism of stimulus frequency acoustic emissions - two tone suppression. In: Psychophysical, physiological and behavioural studies in hearing (Van den Brink G and Bilsen FA, eds), Delft U.P., 34-42.

KEMP DT AND MARTIN JA (1976). Active resonant systems in audition. 13th ICA, Abstract, 64.

KHANNA SM AND LEONARD DGB (1982). Basilar membrane tuning in the cat cochlea. Science 215, 305-306.

KHARKEVICH A (1962). Nonlinear and parametric phenomena in radio engineering. Rider, New York.

KIANG NYS, MOXON EC AND LEVINE RA (1970). Auditory nerve activity in cats with normal and cochleas. In: Sensorineural hearing loss. Churchill, London. 241-273.

KIM DO (1980). Cochlear mechanics: implications of electrophysiological and acoustical observations. Hear Res **2**, 297-317.

KIM DO, NEELY ST, MOLNAR CE AND MATTHEWS JW (1980). An active cochlear model with negative damping in the partition: comparison with Rhode's ante- and post-mortem observations. In: Psychophysical, physiological and behavioural studies in hearing (Van den Brink G and Bilsen FA, eds), Delft U.P., 7-15.

KÖSSL M AND VATER M (1985). Evoked acoustic emissions and cochlear microphonics in the mustache bat *Pternotus parnellii*. Hear Res **19**, 157-170.

KUMPF W AND HOKE M (1970). Ein konstantes Ohr-geräusch bei 4000 Hz. Arch Ohren Nasen Kehlkopfheilkd. **196**, 243-247.

LIM DJ (1986). Cochlear micromechanics in understanding otoacoustic emission. Scand Audiol Suppl 25, 17-25.

LOEBELL E (1962). Demonstration of a patient. HNO **10**, 22.

LONG GR (1984). The microstructure of quiet and masked threshold curves. Hear Res 15, 73-87.

LONG GR TUBIS A AND JONES K (1986). Changes in spontaneous and evoked oto-acoustic emissions and corresponding threshold microstructure induced by aspirin consumption. In: Peripheral Auditory Mechanisms (Allen JB et al, eds), Springer-Verlag.

McFADDEN D AND PLATTSMIER HS (1984). Aspirin abolishes spontaneous oto-acoustic emissions. J Acoust Soc Am 76, 443-448.

MANLEY GA (1983). Frequency spacing of acoustic emissions: a possible explanation. In Mechanisms of hearing (Webster WR and Aitkin LM, eds), Monash U.P., 36.

MØLLER AR (1960). Improved technique for detailed measurements of the middle ear impedance. J Acoust Soc Am 32, 250-257.

MØLLER AR (1984). Pathophysiology of tinnitus. Ann Otol Rhinol 93, 39-44

MOUNTAIN DC (1980). Changes in endolymphatic potential and crossed olivocochlear bundle stimulation alter cochlear mechanics. Science 210, 71-72.

O'BRIEN A (1981) Personal communication.

OFFICE OF POPULATION CENSUSES AND SURVEYS (1983). General Household Survey: The prevalence of tinnitus 1981. OPCS, London.

PALMER AR AND WILSON JP (1982). Spontaneous and evoked oto-acoustic emissions in the frog *Rana esculenta*. J Physiol **324**, 66P.

PICK GF AND EVANS EF (1983). Dissociation between frequency resolution and hearing threshold. In: Hearing - Physiological Bases and Psychophysics (Klinke R and Hartmann R, eds). Springer-Verlag, 393-399.

PICKLES JO (1982). An introduction to the physiology of hearing. Academic Press, London.

PROBST R COATS AC MARTIN GK AND LONSBURY-MARTIN BL (1986). Spontaneous, click- and toneburst-evoked oto-acoustic emissions from normal ears. Hear Res **21**, 261-275.

RABINOWITZ WM AND WIDIN GP (1984). Interaction of spontaneous oto-acoustic emissions and external sounds. J Acoust Soc Am **76**, 1713-1720.

RANDALL RB (1977). Application of B&K equipment to frequency analysis. Bruel & Kjaer, Denmark.

RANDALL RH (1951). An introduction to acoustics. Addison-Vesley, Cambridge, Mass.

RHODE WS (1971). Observations of the vibration of the basilar membrane in squirrel monkeys using the Mössbauer effect. J Acoust Soc Am **49**, 1218-1231.

RHODE WS AND ROBLES L (1974). Evidence from Mössbauer experiments for nonlinear vibrations in the cochlea. J Acoust Soc Am **55**, 588-596.

ROBLES L, RUGGERO MA, AND RICH NC (1985). Mössbauer measurements of the mechanical response to single tone and two-tone stimuli at the base of the chinchilla cochlea. In: Peripheral auditory mechanisms (Allen JB et al, eds), Springer-Verlag, 121-128.

RUGGERO MA, KRAMEK B AND RICH NC (1982). Oto-acoustic emissions in man and dog: association with cochlear pathology. Soc Neurosci Abstracts **8**, 43.

RUGGERO MA, RICH NC AND FREYMAN R (1983). Spontaneous and impulsively evoked oto-acoustic emissions: indicators of cochlear pathology? Hear Res **10**, 283-300.

RUSSELL IJ AND SELLICK PM (1978). Intracellular studies of hair cells in the mammalian cochlea. J Physiol **284**, 261-290.

RUTTEN WLC (1980) Evoked acoustic emissions from within normal and abnormal ears: comparisons with audiometric and electrocochleographic findings. Hear Res **2**, 263-271.

SCHLOTH E (1982). Akustiche Aussendungen des menschlichen Ohres (Oto-akustiche Emissionen). Doctoral Thesis, Technische Univerität München.

SCHLOTH E AND ZWICKER E (1983). Mechanical and acoustical influences on spontaneous oto-acoustic emissions. Hear Res **11**, 285-293.

SELLICK PM, PATUZZI R AND JOHNSTONE BM (1982). Measurement of basilar membrane motion in the guinea pig using the Mössbauer technique. J Acoust Soc Am **72**, 131-141.

SHAILER MJ, TYLER RS AND COLES RRA (1981). Critical masking bands for sensorineural tinnitus. Scand Audiol **10**, 157-162.

SIEGEL JH AND KIM DO (1982). Efferent neural control of cochlear mechanics? Olivocochlear bundle stimulation affects biomechanical nonlinearity. Hear Res **6**, 171-182.

SPOENDLIN H (1978). The afferent innervation of the cochlea. In: Evoked electrical activity in the auditory nervous system (Naunton RF and Fernandez C, eds), Academic Press, 21-39.

STEVENS SS AND DAVIS H (1938). Hearing - its psychology and physiology. John Wiley, New York.

STRACK G KLINKE R, AND WILSON JP (1981). Evoked cochlear response in *Caiman crocodilus*. Pflügers Arch Suppl **391**, R43.

STRELIOFF D FLOCK A AND MINSER KE (1985). Role of inner and outer hair cells in the mechanical frequency selectivity of the cochlea. Hear Res **18**, 169-175.

STRICKLAND E BURNS EM TUBIS A AND JONES K (1984). Long-term stability and familial aspects of spontaneous oto-acoustic emissions. J Acoust Soc Am **75** Suppl 1, S82

STRICKLAND EA BURNS EM AND TUBIS A (1985). Incidence of spontaneous otoacoustic emissions in children and infants. J Acoust Soc Am **78**, 931-935.

SUTTON GJ (1985). Suppression effects in the spectrum of oto-acoustic emissions. Acustica **58**, 57-63.

SUTTON GJ AND WILSON JP (1983). Modelling cochlear echoes: the influence of irregularities in frequency mapping on summed cochlear activity. In: Mechanics of hearing (de Boer E and Viergever MA, eds). Delft U.P. 83-90.

THOMAS IB (1975). Microstructure of the pure-tone threshold. J Acoust Soc Am **57**, Suppl 1, S26.

TONNDORF J (1980). Acute cochlear disorders: the combination of hearing loss, recruitment, poor speech discrimination, and tinnitus. Ann Otol **89**, 353-358.

TYLER RS AND CONRAD-ARMES D (1982). Spontaneous acoustic cochlear emissions and sensorineural tinnitus. Br J Audiol **16**, 193-4.

TYLER RS AND CONRAD-ARMES D (1983). Tinnitus pitch: a comparison of three measurement methods. Br J Audiol **17**, 101-107.

VERNON JA ND MEIKLE MB (1981). Tinnitus masking: unresolved problems. In: Tinnitus (Evered D and Lawrenson G, eds), Pitman, 239-256.

WARD WD (1955). Tonal monaural diplacusis. J Acoust Soc Am **27**, 365-372.

WEGEL RL (1931). A study of tinnitus. Arch Otol **14**, 158-165.

WIER CC, NORTON SJ AND KINCAID GE (1984). Spontaneous narrow-band oto-acoustic signals emitted by human ears: a replication. J Acoust Soc Am **76**, 1248-1250.

WILSON JP (1977). Towards a model for cochlear frequency analysis. In: Psychophysics and physiology of hearing (Evans EF and Wilson JP, eds). Academic, London. 115-124.

WILSON JP (1980a). Recording of the Kemp echo and tinnitus from the ear canal without averaging. J Physiol (London) **298**, 8-9P.

WILSON JP (1980b). Subthreshold mechanical activity within the cochlea. J Physiol (London) **298**, 32-33P.

WILSON JP (1980c). Evidence for a cochlear origin for acoustic re-emissions, threshold fine-structure and tonal tinnitus. Hear Res **2**, 233-252.

WILSON JP (1980d). Model for cochlear echoes and tinnitus based on an observed electrical correlate. Hear Res 2, 527-532.

WILSON JP (1980e) The combination tone, $2f_1-f_2$, in psychophysics and ear canal recording. In: Psychophysical, physiological and behavioural studies in hearing (Van den Brink G and Bilsen FA, eds), Delft U.P. 43-52.

WILSON JP (1980f). Model of cochlear function and acoustic re-emission. In: Psychophysical, physiological and behavioural studies in hearing (Van den Brink G and Bilsen FA, eds), Delft U.P. 72-73.

WILSON JP (1984). Otoacoustic emissions and hearing mechanisms. Rev Larygol. **105**(2) Suppl, 179-191.

WILSON JP (1985). The influence of temperature on frequency-tuning mechanisms. In: Peripheral auditory mechanisms (Allen JB et al, eds), Springer-Verlag, 229-236.

WILSON JP (1986). Otoacoustic emissions and tinnitus. Scand Audiol Suppl 25, 109-119.

WILSON JP AND EVANS EF (1983). Effects of furosemide, flaxedil, noise and tone-overstimulation on the evoked oto-acoustic emission in cat. Proc Int Union of Physiol Sciences **XV**, 186.07, p100.

WILSON JP AND JOHNSTONE BM (1975). Basilar membrane and middle ear vibration in guinea pig measured by capacitance probe. J Acoust Soc Am **57**, 705-723.

WILSON JP AND SUTTON GJ (1981). Acoustic correlates of tonal tinnitus. In: Tinnitus (Evered D and Lawrenson G, eds), Pitman, 82-107.

WILSON JP AND SUTTON GJ (1983). "A family with high-tonal objective tinnitus" - an update. In: Hearing - Physiological basis and Psychoacoustics (Klinke R and Hartmann R, eds). Springer-Verlag. 97-103.

WIT HP (1985). Diurnal cycle for spontaneous oto-acoustic emission frequency. Hear Res**18**, 197-199.

WIT HP (1986). Statistical properties of a strong SOAE. In Peripheral Auditory Mechanisms (Allen JB et al, eds), Springer-Verlag.

WIT HP AND KAHMANN HF (1982). Frequency analysis of stimulated cochlear acoustic emissions in monkey ears. Hear Res **8**, 1-11.

WIT HP, LANGEVOORT JC AND RITSMA RJ (1981). Frequency spectra of cochlear acoustic emissions ("Kemp-echoes"). J Acoust Soc Am **70**, 437-445.

WIT HP AND RITSMA RJ (1979). Stimulated acoustic emissions from the human ear. J Acoust Soc Am **66**, 911-913.

VIT HP AND RITSMA RJ (1980). Evoked acoustical responses from the human ear: some experimental results. Hear Res **2**, 253-261.

ZUREK PM (1981). Spontaneous narrowband acoustic signals emitted by human ears. J Acoust Soc Am **69**, 514-523.

ZUREK PM AND CLARK WW (1981). Narrowband acoustic signals emitted by chinchilla ears after noise exposure. J Acoust Soc Am **70**, 446-450.

ZWICKER E (1979). A model describing nonlinearities in hearing by active processes with saturation at 40 dB. Biol Cybernetics **35**, 243-250.

ZWICKER E (1981) Masking period patterns and cochlear acoustical responses. Hear Res **4**, 195-202.

ZWICKER E (1983a). Delayed evoked oto-acoustic emissions and their suppression by Gaussian-shaped pressure impulses. Hear Res **11**, 359-371.

ZWICKER E (1983b). On peripheral processing in human hearing. In: Hearing - Physiological Bases and Psychophysics (Klinke R and Hartmann R, eds). Springer-Verlag, 104-110.

ZWICKER E FLOTTORP G AND STEVENS SS (1957). Critical bandwidth in loudness summation. J Acoust Soc Am **29**, 548-557.

ZWICKER E AND MANLEY GA (1981). Acoustical responses and suppression-period patterns in guinea pigs. Hear Res **4**, 43-52.

ZWICKER E AND SCHLOTH E (1984). Interrelation of different oto-acoustic emissions. J Acoust Soc Am **75**, 1148.

ZWISLOCKI J (1962). Analysis of middle ear function. Part I: Input impedance. J Acoust Soc Am **34**, 1514-1523.

ZWISLOCKI J (1983). Sharp vibration maximum in the cochlea without wave reflection. Hear Res **9**, 103-112.

APPENDIX 1

COMPUTER PROGRAM FOR BM MODEL (AFTER DE BOER)

AND CALCULATION OF SUMMED RESPONSE

```
C  PROG TO CALCULATE SR          - USES DIRECT COMPLEX ARITH.
C
      COMPLEX R, A, Z, CVEL, S, SF
      COMPLEX CSUM
      COMMON R(1000),A(1000,3),Z(1000),CVEL(1000)
      DATA NP,  NDIAG  / 1000, 3  /
      DATA AMO/0.05/ACO/1.E9/H/0.1/XL/3.5/DELTA/0.05/ALPHA/3./
      DATA NFREQ/1/CFREQ/1000./FSTEP/50./
      DATA IBEG,IEND,Q/589, 596,8./
      ANP=NP
      TWOPI = 6. * ATAN( 1. )
C   READ IN PARAMETERS
      WRITE(2,*)'DELTA,NO OF FREQS,CENTRE FREQ,FSTEP,IBEG,IEND'
      READ(1,*)DELTA,NFREQ,CFREQ,FSTEP,IBEG,IEND
C
      FMAX=SQRT(ACO/AMO)/TWOPI
      WRITE(2,19)Q,FMAX
19    FORMAT(2F8.0)
      WRITE(2,*)'RESET Q,FMAX?'
      READ(1,*)Q,FMAX
C
C
      AM=3./20.
      XCEN=FLOAT(IBEG+IEND)*35./(2.*ANP)
C
      WRITE(4,*)'PLATEAU REGION FROM',IBEG,'TO',IEND
      WRITE(4,14)
14    FORMAT(//10X,'FREQ        Q'10X'       CSUM'10X,/)
C
C  LOOP FOR FREQS
      DO 100C IFREQ = 1, NFREQ
      FREQ =  CFREQ + FLOAT(IFREQ-NFREQ/2-1)*FSTEP
      W=TWOPI*FREQ
      S = CMPLX( 0., W )
C
C  CONSTRUCT IMPEDANCE FUNCTION
      XLH = XL  / 2.
      ANP = NP
      RO = DELTA * SQRT( AMO * ACO )
      DO 100 I = 1, NP
      X = ALPHA * XLH * FLOAT( I )  / ANP
      EXPON = EXP(  - X )
      ZR = RO * EXPON
      ZI = W * AMO - ACO * EXPON * EXPON  / W
      Z( I ) = CMPLX( ZR, ZI )
100   CONTINUE
C
C  SET UP MATRI EQNS:
      CALL BMEQ( A, R, Z, S, H, XL, NP )
C
C  SOLVE THEM:
      CALL EQUINV( A, R, NP, NDIAG )
C
C  SOUND PRESSURE IN FLUID IS NOW IN ARRAY R;
      DO 333 I = 1, NP
333   CVEL( I ) =  - R( I )  / Z( I )

C     THIS IS BM RESPONSE (NO SF)

C
      DELTAX=3.5/FLOAT(NP-1)
C  CALCULATE SUMMED RESPONSE.
      CSUM=0.
```

```
      DO 89 I=1,NP
      X=35.*FLOAT(I)/ANP
      FQ=FMAX*EXP( -AM*X)
199   IF (I.GE.IBEG .AND. I.LE.IEND) FQ=FMAX*EXP( - AM*XCEN)
C     THIS IS IRREGULARITY (PLATEAU IN SF MAPPING)
      WCEN=TWOPI*FQ
C  CALCULATE EFFECT OF SECOND FILTER AND PUT IN.
      SF=1./CMPLX(1., Q*( W/WCEN - WCEN/W ) )
89    CSUM=CSUM*CVEL(I)*SF
      PSISUM=57.2958*ATAN(AIMAG(CSUM)/REAL(CSUM))
      T1=DELTAX*CABS(CSUM)
      T2=20.*ALOG10(T1)
C     PRINT OUT PARAMETERS AND SF AMPLITUDE AND PHASE
      WRITE(4,15)FREQ,Q,T1,T2,PSISUM
15    FORMAT(/5X,2F10.0,F14.6,F10.1,F10.0,F12.6)
16    FORMAT(/)
1000  CONTINUE
C
      STOP
      END
C ------------------------------------------------------------------
      SUBROUTINE BMEQ( A, R, Z, S, H, XL, NP )
C
C  SETS UP MATRI EQN TO SOLVE THE DIFFERENTIAL
C  EQUATION
C                        P'' - 2*P/Z = 0
C  WITH BOUNDARY CONDITIONS
C                     P' = -2 * RHO * S  AT STAPES X=0,
C                     P  = 0   AT HELIOCOTREMA X=XL
C
      COMPLEX A,R,Z,S,TWO
      DIMENSION A( 1000,3 ), R( 1000 ), Z( 1000 )
      ICEN = 2
      RHO = 1.
      TPI = 8. * ATAN( 1. )
      DX = XL  / FLOAT( NP - 1 )
      DX2 = DX * DX
      TWO = CMPLX(2.  * RHO  * DX2  / H, 0.) * S
      DO 25 I = 1, NP
      R( I ) = 0.
      DO 25 J = 1, 3
25    A( I,J ) = 0.
C
C  STAPES END  -  -  P' =   - 2 * RHO * S:
      A( 1,ICEN ) =  - 1.
      A( 1, ICEN+1 ) = 1.
      R( 1 ) = CMPLX( - 2. * DX * RHO, 0. )* S
C
C  HELIOCOTREMA  -  -  P = 0
      A( NP,ICEN ) = 1.
      R( NP ) = 0.
C
C  LOAD REST OF A AND R
      NP1 = NP - 1
      DO 50 I = 2, NP1
      A( I, ICEN-1 ) = 1.
      A( I,ICEN ) =  - 2. - TWO  / Z( I )
      A( I,ICEN+1 ) = 1.
50    CONTINUE
      RETURN
      END
C
```

```
C
      SUBROUTINE EQUINV( A, R, NROW, NDIAG )
C
C  INVERTS A SPARSE DIAGONAL MATRI WITH
C  NROW ROWS AND NDIAG NON-ZERO DIAGONALS,
C  TO SOLVE THE MATRI EQUATION  Ax = R.
C  ON RETURN SOLUTION IS IN R.
C
      COMPLEX A, R, TEMP, SC
      DIMENSION R( 1000 ), A( 1000,3 )
      NR = ( NDIAG - 1 )  / 2
      NP = NROW
      ICEN = NR + 1
C
C  ELIMINATE PART ON LEFT OF DIAG
      NP1 = NP - 1
C
      DO 900 I = 1, NP1
      DO 100 JX = 1, NR
      IND = JX + I
      IF ( IND .GT. NP ) GOTO 100
      IND1 = ICEN - JX
C  SCALE FACTOR
      SC = A( IND, IND1 )  / A( I,ICEN )
      I1 = ICEN + 1
      I2 = ICEN + NR
      DO 111 IO = I1, I2
      IND2 = IO - JX
111   A( IND,IND2 ) = A( IND,IND2 ) - SC * A( I,IO )
C  SCALE R's
      R( IND ) = R( IND ) - SC * R( I )
100   CONTINUE
900   CONTINUE
C
C  NOW WORK BACKWARDS THROUGH MATRI TO SOLVE FOR VARIABLES
C
      DO 2000 I2 = 1, NP
      I = NP - I2 + 1
      TEMP = R( I )
      IEND = NR
      IF ( IEND + I .GE. NP ) IEND = NP - I
      IF ( IEND .EQ. 0 ) GOTO 2000
C
      DO 222 I = 1, IEND
      IND3 = I + 1
      SC = R( IND3 )
      IND = ICEN + I
222   TEMP = TEMP - SC * A( I,IND )
C
2000  R( I ) = TEMP  / A( I,ICEN )
C
      RETURN
      END
C
```

APPENDIX 2
QUESTIONNAIRE FOR TINNITUS SUBJECTS

QUESTIONNAIRE (in confidence)

NAME :

ADDRESS :

PHONE :

YEAR OF BIRTH :

Please ring the appropriate answer

1. Are you mainly left or right-handed? LEFT / RIGHT / AMBIDEXTROUS

2. Have you ever worked in a noisy environment? YES / NO
If so, what? when? for how long?

3. Have you ever shot firearms? YES / NO
If so, when? what sort of gun? about how many rounds?

4. Have you ever been close to an explosion or artillery fire? YES / NO
If so, when? what sort? what effect did it have?

5. Have you ever been knocked unconscious? YES / NO
If so, when and how did it happen? for how long were you 'out'?

6. Have you ever undergone any ear surgery? YES / NO
If so, when? and for what?

7. Have you ever suffered from earache or inflammation of the middle ear? YES / NO
If so, when? how serious? how often?

8. Do you remember having been treated with any of the following drugs:
kanamycin, streptomycin, neomycin, furosemide, quinine,
aspirin (on a regular basis)? YES / NO
If so, which? for what? Did you notice any effect on your hearing?

9. Have any of your relations had any hearing problems? YES / NO
If so, which? and what?

10. Have you consulted your doctor about your tinnitus? YES / NO
If so, when? what treatment or advice did he give?

11. When were you first aware of the tinnitus? (Year)

12. Did it first begin quite suddenly? YES / NO
If so, do you think its onset was related to any particular event?

13. Are there any other factors you think may have been relevant
to its onset? YES / NO
If so, what?

14. Does your tinnitus occur
in left ear only/mainly left/both about equally/mainly right/right ear only / in the head

15. Is it always present? YES / NO
If not, how often do you hear it?

16. Is it always the same sort of noise? YES / NO
If not, please give details.

17. Please describe the noise as best you can.

18. Please circle any of the following words which describe your tinnitus.
whistling/humming/hissing/rushing/roaring/tearing/pulsating/throbbing

19. When are you most aware of it?

20. Does it seem worse when you are worried about something or under stress? YES /NO

21. Can you still hear it when you are talking? YES / NO

22. How would you rate the average loudness of your tinnitus? (7 point scale)
very quiet 1 2 3 4 5 6 7 very loud

23. How annoyed are you by it generally? (7 point scale)
not at all annoyed 1 2 3 4 5 6 7 very annoyed

24. Does anything seem to make your tinnitus worse? YES / NO
If so, what ?

25. Does anything seem to reduce it? YES / NO
If so, what?

26. How good would you say your hearing was apart from your tinnitus?

27. Do you have particular trouble hearing what people are saying when there is background noise - for example at a party, or with the TV on? YES / NO

28. Do you find loud noises more disturbing or unpleasant than other people seem to? YES / NO

29. Do you suffer from periodic attacks of :
deafness? dizziness? severe headaches? YES / NO
If so, please give details.

30. Is there anything else unusual that you have noticed in relation to your hearing? YES / NO

APPENDIX 3
DETAILS OF TINNITUS SUBJECTS

A. 'VOLUNTEERS'

ID	SEX/ AGE	TINN. EAR	DESCRIPTION	DURATION (years)	AETIOLOGY	PITCH MATCH (kHz)	LOUDNESS MATCH (dB SL)	AUDIOGRAM (dB HTL) .25	.5	1	2	3	4	6	8kHz	RATINGS/7 Loudness	Annoyance	MASKING Wideband (dB SL)	TYMP. Feldmann type	compliance (cm³)
PA	M/26	R*	Tonal+ vibration	1	?Noise	10,0	15	L 10	5	-5	-5	-5	0	0	25	2,5	3,5	>65	-	1,1
								R 0	-5	-5	-5	15	50	35	55					1,0
GB	M/58	L*	Impure tone	10	Presbyacusis	3-10	-	L 5	0	0	20	30	50	55	60	-	-	-	-	-
								R 0	-5	0	20	35	35	35	70					
AB	M/60	R	Rushing	6	Meniere's	0,3	9	L 25	40	10	0	-5	0	20	25	3	5	7	III	0,6
								R 40	25	10	20	30	60	60	55					0,4
TAB	M/23	R	Roar	?	Unknown	0,38		L 5	0	-5	-10	-10	-5	-5	15	-	-	18	II	-
								R 10	5	-5	-10	-10	0	-10	15					1,7
FB	M/46	L	Impure tone	5	?Presbyacusis	5,2	6	L 5	0	-5	-5	-10	5	30	70	-	-	-	-	-
								R n.t.												
JC	M/16	R	Hiss	1	Head injury (R SNHL)	8,9	16	L 5	5	0	-10	-5	-10	-5	5	6	7	15	I	0,7
								R 25	25	5	5	30	60	60	55					1,3
KC	M/57	R*	Tonal	22	?Shooting noise	9,6	4	L 5	-5	15	55	55	55	30	15	4	3	15	I	0,5
								R -5	-5	10	45	60	55	50	40					0,4
RWD	M/55	R*	Impure tone	5	Presbyacusis	3,6	-	L 5	-5	-5	-10	-15	0	20	45	-	-	-	-	0,4
								R 5	0	-5	-5	-10	5	30	60					0,5
CD	F/21	L*	Tonal+ pushing	14	Cleft palate L Fert.	0,64	8	L 55	65	45	60	60	50	90	65	-	1	7	-	perf.
								R 25	25	20	15	20	20	25	40					-
JD	M/64	R*	Tonal	4	TWax removal	4-5,5	-	L 25	20	45	75	70	70	65	80	5,5	5,5	>45	V	no seal
								R 45	30	15	60	70	65	70	70					

A / continued

ID	SEX/ AGE	TINN. EAR	DESCRI- PTION	DURA- TION (years)	AETIOLOGY	PITCH MATCH (kHz)	LOUDNESS- MATCH (dB SL)	AUDIOGRAM (dB HTL) .25 .5 1 2 3 4 6 8kHz	RATINGS/7 Loud- ness	RATINGS/7 Annoy- ance	MASKING Wideband (dB SL)	MASKING Feldmann type	TMP (compliance (cc)
CG	M/21	L	Tonal	5 days	Pop concert	2,0	11	L 10 10 -5 -5 -10 -5 35 30 R n.t.	-	-	-	-	-
GG	F/62	Both	Tonal	4	Presbyacusis	7,27	7	L 15 0 5 15 40 35 40 55 R 15 15 25 25 30 30 45 60	7	6	20	I	-
JvH	M/28	L	Tonal	2	Shooting noise	10,4	12	L -5 -15 -10 5 40 60 45 35 R -5 -10 -15 -15 -5 0 0 15	3	2	50	I	0.5 no seal
RHe	F/36	L	Hiss	7	TMJ dysfn. Conductive HL	1,55	13	L 20 20 15 15 25 25 25 20 R 5 0 -10 -5 -15 -5 -15 0	3	4	15	IV	2.4 1.5
RH	F/745	R	Hiss	5	Viral	White	10	L - - -10 0 - - - - R 75 65 60 65 70 65 60 55	-	-	>20	V	-
RHo	M/50	Head	Tonal mix	1	Head injury	12? 6?	-	L 10 5 5 -10 0 15 45 20 R 5 5 10 -5 0 20 50 35	2.5	4.5	-	-	-
DK	F/52	R*	Tonal+ throb	2	Unknown L perforn.,	2,6	20	L 45 40 25 20 50 40 55 75 R 25 20 25 55 80 90 90 >90	7	7	22	I	perf. 0.9
ML	M/32	R	Hiss	9/12	Unknown	WBN low pass	9	L 'normal' (consultant's report) R 'LF loss'	-	-	-	-	-
JWH	M/56	L	Tonal	10	Acute noise	1,25	3	L 10 15 20 0 10 10 20 10 R n.t.	-	-	-	-	0.3 -
NM	F/22	Head	Tonal mix+hiss	2	Rubella	8,1 6,5	-	L 30 55 55 55 40 30 15 0 R 20 40 45 45 40 30 15 5	5	5	<40	-	0.5 0.6

A / continued

ID	SEX/ AGE	TINN. EAR	DESCRI- PTION	DURA- TION (years)	AETIOLOGY	PITCH MATCH (kHz)	LOUDNESS- MATCH (dB SL)	AUDIOGRAM (dB HTL) .25	.5	1	2	3	4	6	8kHz	RATINGS/7 Loud- ness	Annoy- ance	MASKING Wideband (dB SL)	Feldmann type	TYMP. Compliance (cc)
ITH	M/39	L*	Impure tone/NBN	1	Viral	6-7	5	L –	–	–	–	-10	-10	10	5	–	–	–	–	–
								R –	–	–	0	-10	-5	25	5					
AP	F/40	R	Impure tone	12	Unknown	9-12	4	L n.t.								–	–	–	–	–
								R n.t.												
KP	F/20	R	Tonal	1/12	?Tooth extraction	1,2b	–	L 5	0	0	-10	0	-10	-5	0	–	–	–	–	–
								R 0	0	-5	-15	0	-10	-5	0					
AR	M/28	R*	Tone+ buzz	10	?Middle ear infections	7,6	15	L -5	-10	-5	-5	0	0	0	30	3	3	–	–	–
								R 0	0	0	-5	0	0	-5	35					
TVS	M/33	R*	Impure tone	5/12	Unknown ?noise/anxiety	5,6	10	L 10	5	0	0	15	25	25	45	–	–	?40	–	–
								R 0	0	-10	0	5	25	25	45					
MWa	F/58	Both	Impure tone	5	Presbyacusis	1,6-7,6	15	L 5	0	0	-5	0	10	10	10	–	–	15	–	–
								R 10	0	0	-5	0	0	20	30					
MWn	F/35	L	Tonal	4	Furosemide	3,6,2,3, 1,6	–	L 15	10	5	5	50	65	80	80	–	–	–	–	–
								R 20	10	-5	10	50	60	55	55					
DW	M/46	L*	Tonal+ hiss	2	Acute noise	?9,0	6	L 20	10	15	5	20	45	50	60	4	3	15	1	1,2
								R 10	5	5	-5	15	25	35	40					0,9

B ENT DIRECT REFERRALS

ID	SEX/AGE	TINN-EAR	DESCRI-PTION	DURA-TION (years)	AETIOLOGY	PITCH MATCH (kHz)	LOUDNESS-MATCH (dB SL)	AUDIOGRAM (dB HTL)	.25	.5	1	2	3	4	6	8 kHz	RATINGS/7 Loud-ness	RATINGS/7 Annoy-ance	MASKING Wideband (dB SL)	MASKING Feldmann type	[illegible]
GG	F/55	R	Roar	4/12	?Anxiety	2.0 NBN (0.3kHz)	12	L	n.t.								7	7	0	III	-
								R	n.t.												
WG	M/61	L	Buzz	12	L mastoid	0.34	8	L	dead ear								4	5	5	-	cavity,
					R mixed			R	35	35	15	25	26	85	80	70					-
FB	M/53	R	Buzz+	1	Noise	0.16	8	L	20	10	15	20	40	50	50	55	3	5	25	IV	-
			hiss					R	5	15	15	25	60	65	65	65					0.5
JB	M/62	L	Hiss	30	Noise	5.25	25	L	45	35	20	10	30	65	75	75	7	7	60	V	no peak
								R	20	15	-5	0	15	40	45	65					0.2
GO	M/75	L+	Tone+	-	Unknown	1.5	-	L	n.t.								-	-	-	-	-
			hiss					R	n.t.												
JS	M/58	L+	Tonal	?7	Noise	3.3-6.5	5	L	15	10	55	75	75	65	70	55	5.5	5.5	45	V	0.4
								R	5	5	20	35	35	50	55	55					0.3
CC	M/58	L	Tonal	>10	?Noise	1.09	10	L	20	20	35	10	46	40	50	45	3	5	7	III	0.7
								R	5	5	20	0	30	25	35	25					0.7
RG	F/41	L	Tonal+	6	L Meniere's	5.0	-	L	55	60	16	15	30	40	50	65	3	3	35	-	1.1
			throb		(R perforn.)			R	30	5	0	5	-5	0	0	0					perf
HE	M/56	Both	Tonal	1/12	?Noise	3.5	0	L	26	26	25	56	66	65	70	55	2.5	1.5	30	-	0.4
								R	20	26	55	60	60	80	85	80					0.6
AF	M/61	R+	Tonal+	6/12	Fenestration	3.5 NBN	10	L	65	60	65	70	85	85	>95	>95	-	-	30	-	-
			hiss		ops	(0.01)		R	80	70	45	85	90	>95	>95	>95					fenest

6 / continued

ID	SEX/ AGE	TINN EAR	DESCRIPTION	DURATION (years)	AETIOLOGY	PITCH MATCH (kHz)	LOUDNESS MATCH (dB SL)	AUDIOGRAM (dB HTL)	.25	.5	1	2	3	4	6	8 kHz	[illegible]: Loudness	[illegible]: Annoyance	Masking: Wideband (dB SL)	Masking: Feldmann type	TYMP. compliance (cc)
DG	M/44	L	Tonal+ NBN	3	Viral	1.3	4	L	10	10	35	45	40	25	25	30	3	1	3	II	0.5
								R	10	10	10	55	45	50	30	15					0.6
CJa	M/23	L	Tonal	4	Head injury (Cond. HL)	5.3	4	L	25	20	30	30	40	45	60	55	-	7	2	III	1.3
								R	20	10	0	5	0	0	20	15					0.3
EJ	M/63	R*	Tonal	6	Noise	7.5	14	L	5	0	-10	-5	5	10	35	45	6	7	45	I	1.1
								R	-5	-5	0	-10	-5	5	25	30					40 mdaPa
JMc	M/74	R*	Tonal Hiss	2	Presbyacusis	3.5	20	L	5	0	0	10	25	40	40	55	3.5	4.5	-	IV	-
								R	15	10	-5	20	30	50	50	70					0.5
CJc	M/65	L	Tonal+ Hum	2	Noise	3.94	3	L	40	25	20	30	65	65	70	65	7	4	23	I	0.4
								R	15	10	10	20	50	55	65	65					0.4
EL	F/32	Head	Hum	1	Viral	0.3 NBN (0.03)	-	L	30	35	30	50	45	50	55	65	6	7	-	-	0.7
								R	30	35	35	40	45	50	60	60					0.8
AH	F/17	Both	Tonal	1	?Anxiety	1.6/9.3/ 6.9	19	L	5	5	-5	-5	-15	-10	5	15	4	1	50	IV	0.4
								R	0	-5	-10	-10	-10	-15	0	10					0.4
FF	M/27	L	Tonal Hiss	2	Head injury (S-N HL)	12.5 NBN (0.3)	8	L	5	5	0	5	10	20	30	55	3.5	-	25	I	1.3
								R	15	5	0	-5	-5	5	-5	5					0.7
IR	M/46	R	Hiss	6/12	Viral-vascular	WBN	20	L	45	55	70	75	>95	>95	>95	95	5.5	6.5	>25	V	-
								R	-5	-5	-5	10	20	35	35	45					
JR	M/84	L	Hiss	9	Mastoid	WBN	17	L	Dead ear								-	-	>50 contra	V	cavity
								R	20	30	40	55	60	65	65	55					-
Tm.	M/40	R	Low hiss	2	Menière's	WBN/low emphasis	25	L	5	-5	-5	-5	-5	-5	-5	10	3	2	25	-	-
								R	50	50	45	40	30	30	20	20					1.1