

# Otoacoustic emissions, their origin in cochlear function, and use

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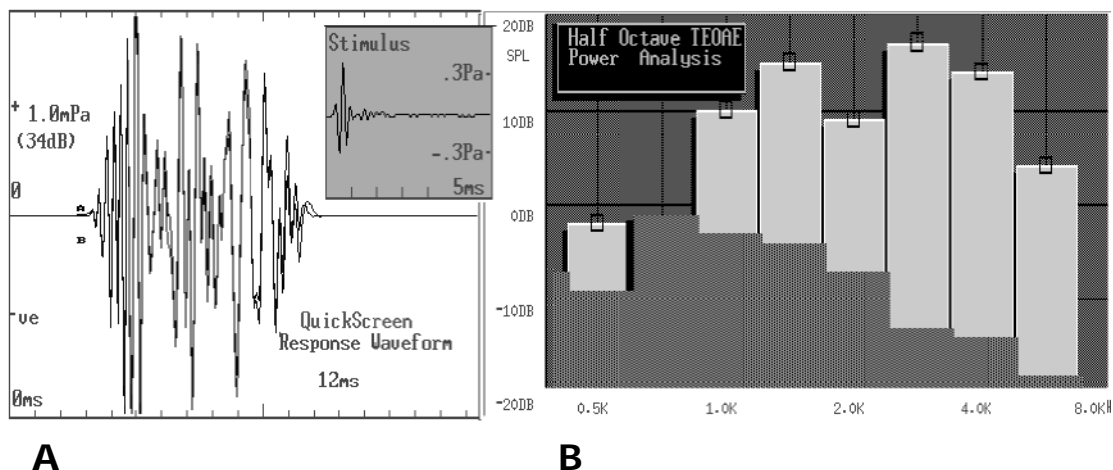
Otoacoustic emissions (OAEs) are sounds of cochlear origin, which can be recorded by a microphone fitted into the ear canal. They are caused by the motion of the cochlea's sensory hair cells as they energetically respond to auditory stimulation. OAEs provide a simple, efficient and non-invasive objective indicator of healthy cochlear function and OAE screening is widely used in universal new-born hearing screening programmes. As part of the audiological diagnostic test battery, OAEs can contribute to differential audiological diagnosis, they can be used to monitor the effects of treatment and they can be helpful in the selection of hearing aids and of surgical options. As a research tool, OAEs provide a non-invasive window on intracochlear processes and this has led to new insights into the mechanisms and function of the cochlea and also to a new understanding of the nature of sensory hearing impairment. This chapter provides a broad introduction to OAEs and their applications together with a detailed description of the relationship between OAEs and cochlear mechanisms.

Otoacoustic emissions (OAEs) are sounds which arise in the ear canal when (paradoxically) the tympanum receives vibrations transmitted **backwards** through the middle ear **from** the cochlea. These vibrations occur as a by-product of a unique and vulnerable cochlear mechanism which has become known as the 'cochlear amplifier' and which contributes greatly to the sensitivity and discrimination of hearing. Figure 1A shows an example of a strong, but otherwise typical, transient evoked otoacoustic emission (TEOAE) produced by a healthy new-born ear in response to a click stimulus.

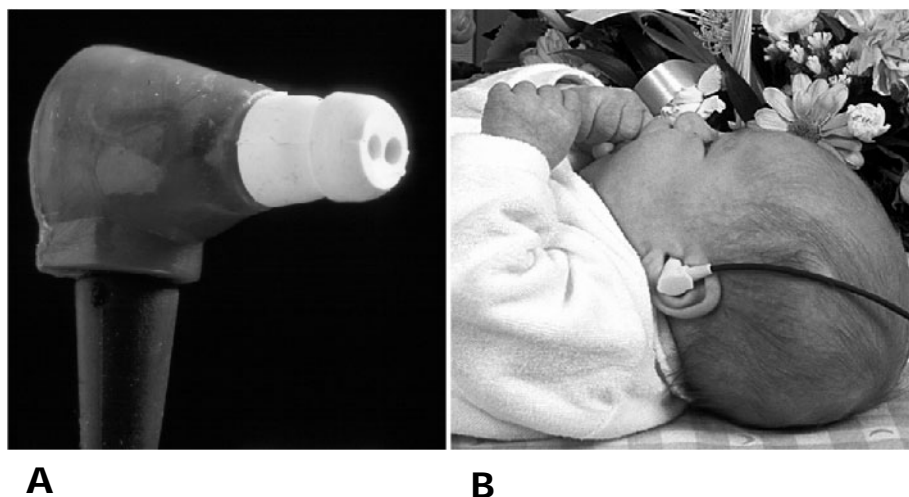
OAE recordings are made *via* an ear canal probe which is deeply inserted into the ear canal as shown in Figure 2. Click stimuli of around 84 dB SPL peak equivalent (p.e) level normally evoke a robust TEOAE response only if hearing threshold is 20 dB HL or better<sup>1-3</sup>. Unlike other audiometric tests, it is not necessary for the stimulus to be near to threshold levels to detect departures from normal function using OAEs. Middle ear status affects OAEs and can prevent their detection<sup>4</sup>.

The oscillatory sound pressure waveform seen in TEOAE responses (as in Fig. 1A) actually corresponds to the motion of the eardrum being

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**Fig. 1** A strong TEOAE response from a new-born infant. (A) The TEOAE wave form in response at an acoustic click stimulus (shown inset) peaking at 84 dB SPL. The oto-acoustic response level exceeds 30 dB SPL. (B) A frequency analysis of the same TEOAE response showing the energy present per half-octave band. The lower shaded area indicates noise contamination. The wave form is blanked from 0–3.5 ms to remove stimulus artefacts, and again after 12 ms when the next stimulus was applied. A ‘non-linear’ pattern of different stimulus intensity is usually employed to minimise stimulus artefacts<sup>46</sup>. Multiple stimulus presentations and response averaging are usually required to extract TEOAE responses from background noise. Stimulus repetition rates of 50–100 s<sup>-1</sup> are usual and recording times vary from a few seconds to a few minutes.

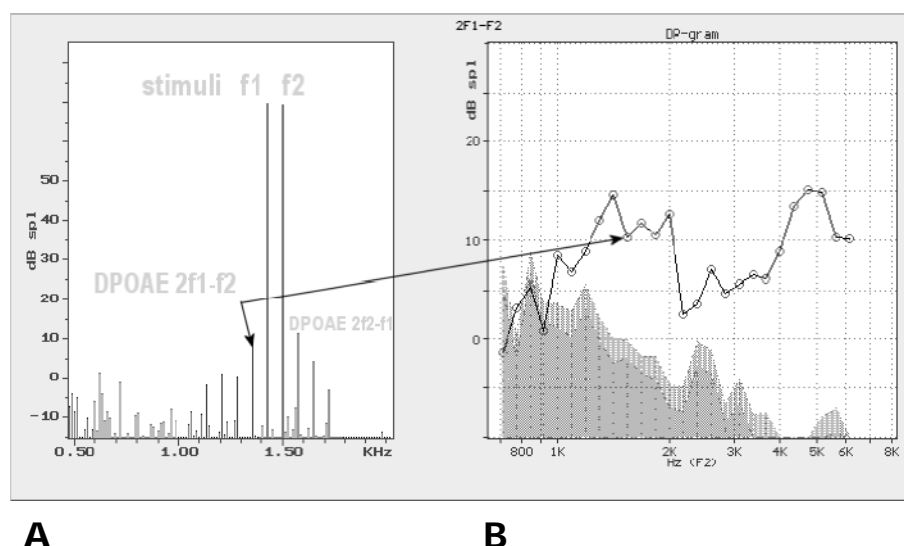


**Fig. 2** (A) A TEOAE probe containing miniature sound source and microphone transducers. The soft disposable tip carries sound ports for the stimulus and for the microphone. DPOAE probes have an additional stimulus port. In some probes, all ports feed a single sound tube. (B) The probe needs to be deeply inserted in the ear canal for maximum OAE capture and noise exclusion, with the cable positioned so as to avoid noise production on movement.

pushed backwards and forwards by fluid pressure fluctuations generated inside the cochlea. The sealing of the ear canal by the probe increases the recorded OAEs' sound pressure below 3 kHz, as otherwise drum vibrations would simply move air in and out of the ear canal. The response is long and complex because responses from different parts of the cochlea arrive at the ear canal at different times and at different frequencies. Although clicks are 'wide-band' stimuli, exciting the whole of the cochlea, TEOAE responses can give a frequency specific indication of cochlear status. By splitting the response into frequency bands after recording (Fig. 1B) separate responses from different parts of the cochlea are obtained. TEOAE responses are strongest and easiest to detect in the primary speech frequency band, 1–4 kHz. In young ears, TEOAEs extend up to 6–7 kHz<sup>5</sup>, but many clinically normal adult ears give weak TEOAEs (less than 3 dB SPL), with no substantial response above 4 kHz.

TEOAEs are highly sensitive to cochlear pathology and in a frequency-specific way. Frequencies at which hearing thresholds exceed 20–30 dB HL are typically absent in the TEOAE response<sup>1,6,7</sup>. Because of their sensitivity to cochlear dysfunction, TEOAEs have found wide-spread application in new-born hearing screening programmes<sup>8</sup>. Healthy infant ears typically produce strong OAE levels of 15 dB SPL to more than 30 dB SPL. Little signal processing is required to extract these responses from noise and fully validated frequency-specific measurements can often be made in a few seconds. This contrasts with recordings of the auditory brain-stem response (ABR), which require electrodes and which must be extracted from the relatively much stronger EEG background signal over a longer period of signal averaging. However, OAE signals are very sensitive to minor conductive losses caused by middle ear fluid and ear canal debris in neonates, so that sometimes, in the first few hours after birth, an ABR can be recorded when an OAE cannot. Also, since the ABR is sensitive to both cochlear and retro-cochlear pathology, ABR testing would appear to be preferable to OAE for infant screening. However, in practice, sensory hearing impairment in the low-risk new-born population appears to be overwhelmingly of the sensory transmissive type (see below), which is readily detectable by measurement of OAEs. This, together with favourable ergonomic and economics factors, means that OAEs are a reliable and highly cost-effective tool for universal new-born hearing programmes. Nevertheless, where there is a known risk of neurological damage, ABR testing is also essential.

The healthy ear produces OAEs not only in response to clicks, but to any applied sound including tones. A second method of OAE recording, using tonal stimulation and called 'distortion product otoacoustic emissions' (DPOAEs) is in wide-spread clinical use<sup>9</sup>. Non-linear intermodulation between the two stimulus tones inside the cochlea generates several new acoustic frequency components, which can travel



**Fig. 3** Distortion product OAE recording requires the application of two pure tone stimuli. The lower frequency tone is commonly applied at a level of 60–70 dB SPL while the higher frequency tone is applied at a lower level of 50–70 dB SPL. (A) The sound spectrum in a healthy human ear canal during stimulation by two closely spaced pure tones,  $f_1 = 1425$  Hz and  $f_2 = 1500$  Hz, both at 70 dB SPL. The spectral lines between 1100–1800 Hz on either side of the stimulus lines are intermodulation tones created by the cochlea. Each is separated from its neighbour by an interval of  $[f_2 - f_1]$ . Clinical DPOAE analysis typically tracks the level of just one of these distortion products ( $f_{dp} = f_1 - [f_2 - f_1] = 2f_1 - f_2$ ) as  $f_1$  and  $f_2$  are stepped across the frequency range of interest. (B) This allows a 'DP-gram' to be constructed. A stimulus frequency separation  $f_2/f_1 \sim 1.2$  results in the strongest DPOAEs. The lower shaded portion (right) indicates the noise contamination.

to the ear canal. Healthy ear canal distortion levels can be above 20 dB SPL. Figure 3 provides an example of a clinical DPOAE analysis and illustrates its derivation.

Non-linear intermodulation between two tones is a purely mechanical process and distortion products satisfy the frequency relationship  $f_{dp} = f_1 + N(f_2 - f_1)$  where  $N$  is any positive or negative whole number<sup>10</sup>. Each distortion component can be separated from the stimuli by preset signal frequency analysis. The intensity of one particular component at  $f_{dp} = f_1 + (-1)[f_2 - f_1]$ , (simplifying to  $2f_1 - f_2$ ) is used as an indicator of cochlear status, plotted as a function of frequency in the 'DP-gram' (see Fig. 3B). DPOAE generation is much reduced and usually absent if there is significant sensory hearing loss<sup>11</sup>.

The TEOAE and DPOAE techniques compliment each other. DPOAEs offer a wider frequency range of observation (above 10 kHz) with less sensitivity to minor and sub-clinical conditions in adults. More powerful excitation is practical with continuous tones (up to 75 dB SPL), allowing

DPOAEs to be recorded with moderate losses when no TEOAE can be detected. However, DPOAE recordings provide no greater frequency specificity than TEOAEs despite the use of pure tones. At best, both responses reflect the intrinsic frequency resolution of the cochlea, which is around one-quarter octave (see also Fig. 5).

Although OAEs are a good indicator of hearing loss, it is important to remember that an OAE examination is not a hearing test. It is a test of an essential and vulnerable cochlear function as described below. It is tempting to believe that OAE intensity relates directly to cochlear 'strength', but this is not so. Like electrophysiological measurements of auditory function, the observed OAE response intensity can be strongly affected by the quality of the coupling between sensor and patient (*i.e.* the OAE probe fit or electrode placement) and by spurious non-auditory factors which determine the external field produced by internal physiological activity. Intensity is a primary factor in OAE detectability but, like the ABR, it is the **presence** of a detectable OAE response to a particular stimulus that is clinically important and not its strength.

Measurement of DPOAEs at multiple stimulus levels can establish the OAE 'growth rate'. Healthy ears tend to exhibit a DPOAE growth rate of 1 dB of OAE per 1 dB of stimulus or less. Ears with some impairment show steeper growth while instrumental artefacts tend to have the greatest rates of growth. Single DPOAE observations can be misleading and results need to be averaged across a frequency range. Estimates of the degree of threshold elevation have been attempted using DPOAE intensity and growth functions<sup>12,13</sup>, but obtaining the 'threshold of detectability' of DPOAE against background noise adds little further information. While DPOAEs relate to threshold across a population, they are at best a very unreliable and imprecise indicator of an individual's hearing threshold. This is because, unlike the ABR, OAEs are presynaptic responses. Their origin precedes the 'threshold' imposed by the inner hair cell's transduction process. Use of higher stimulus intensities increases OAE detectability and can provide useful evidence of residual hair cell activity, but does not probe any deeper into the auditory pathway.

The field of OAE research has grown enormously since their discovery 25 years ago<sup>3,10</sup>. Comprehensive overviews of OAEs, measurement technology and applications can be found elsewhere<sup>9,14</sup>. The purpose of this chapter is to present and explain OAEs to potential users in the context of cochlear function and from this to derive a logical basis for their clinical application.

## How do otoacoustic emissions arise?

Detectable OAE sound pressure is produced by motions of the eardrum which are extremely small. For example, an eardrum oscillation of only

$10^{-10}$  m (the diameter of an atom of hydrogen) will create a 'large' OAE of intensity 34 dB SPL (1 mPa) in a 1-ml ear canal volume. A common misconception is that OAEs are 'radiated' by the cochlea and transmitted through the middle ear cavity. This is not the case. Motions of the oval and round window membranes during OAE generate negligible sound pressure, because their areas are too small and their motions oppose each other.

To understand how OAEs arise, why vibrations emerge from the cochlea, and why OAEs are indicative of good hearing, it is necessary to consider what normally happens as stimulation enters the cochlea.

### *The cochlea as a stimulus delivery system*

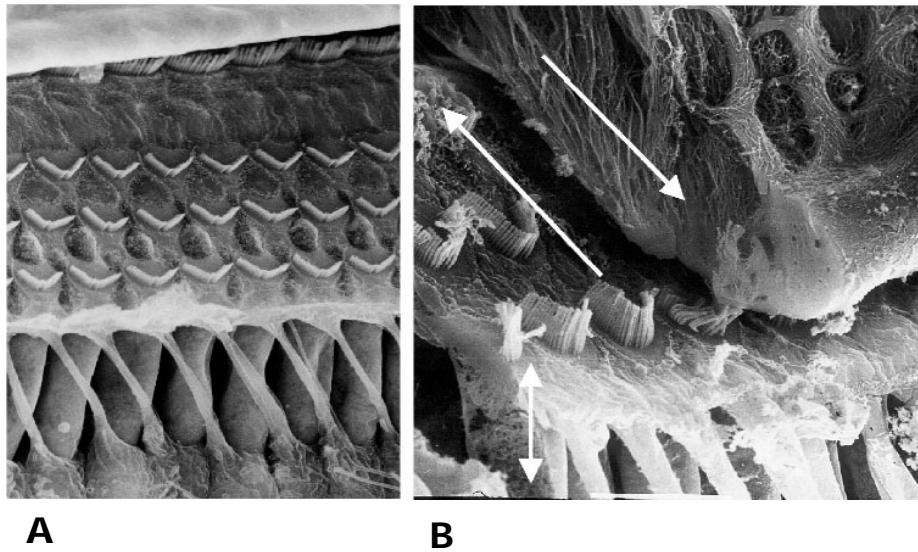
The importance of the outer and middle ear mechanisms in collecting sound energy and conveying it to the cochlea is well understood. Pathology in these areas affects hearing through stimulus attenuation, which can be severe but never profound. Unlike sensory loss, conductive loss introduces no distortion, so it can be very effectively corrected by amplification.

Even healthy ears have a slight 'conductive loss' because not all of the available sound energy enters the cochlea. The proportion which does enter the cochlea depends on how efficiently the middle ear mechanism couples the low acoustic impedance of air to the high mechanical impedance of the fluid-filled cochlea. The round window plays an important role in releasing cochlear fluid pressure caused by stapes displacement thereby greatly reducing the cochlear input impedance. This increases cochlear fluid motion which eventually excites the inner hair cells (Fig. 4). In contrast, acoustic pressure in the cochlear perilymph is almost instantly transmitted to every cell, but causes no sound sensation.

Fluid motion in the basal regions of scala vestibuli and tympani, synchronously with stapes vibration, necessarily displaces the basilar membrane (BM). Only the basal part of the BM moves initially because, although quite stiff, it has little inertia. Being narrow in relation to the cochlear duct, it moves relatively little fluid mass.

Induced transverse oscillations of the basal BM begins to propagate apically. Oscillatory exchanges occur between fluid motion energy and the energy held in elastic BM displacement. Adjacent BM sections are excited as fluid displaced from, for example, the upward motion of the BM at one place forces the next more apical and less stiff place downwards and so on, resulting in a travelling wave (Fig. 5A). The travelling wave (TW) conveys stimulus energy towards the apex at less than 1/100th of the speed of sound in air. For example, the 2 cm journey of a 500-Hz stimulus to its cochlear place takes around 10 ms, accounting for the substantial latency of OAEs<sup>15</sup>.

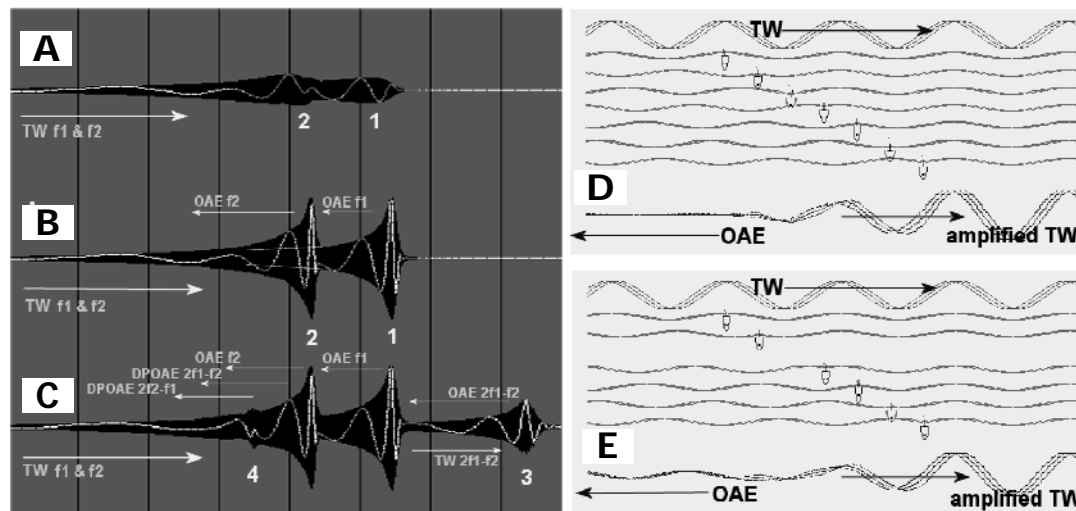
The BM becomes progressively less elastically stiff and wider away from the base. Less elastic stiffness means less force opposing displacement, so



**Fig. 4** Two scanning electron micrograph views of the organ of Corti and its sensory hair cells (courtesy of Prof. A. Forge) showing the extremely orderly arrangement of sensory cells and the reason for heavy viscous damping. **(A)** Stereocilia of the inner (back row) and outer (front three rows) hair cells which protrude from the reticula lamina. Sectioning (front) shows the separated cylindrical OHC bodies within the rigid box-like structure. **(B)** A freeze-etched specimen showing the tectorial membrane (TM) in its functional position (with some damage in preparation). The tectorial membrane is anchored to a non-vibrating point near the modiolus and also rests on the stereocilia of the outer hair cells. The organ of Corti moves as a whole with BM motion, causing linear and shear motion relative to the TM. This results in oscillatory radial fluid flow within the narrow sub-tectorial space between scala media and the inner sulcus. The flow impinges on IHC stereocilia, which do not touch the TM but form an almost continuous 'fence' to arrest any fluid flow (see rear of A). Their deflection excites the IHCs causing transduction of stimulus information into neural code. This fluid motion takes place in the very narrow space (less than  $10\ \mu\text{m}$ ) and it is here that viscosity readily absorbs energy from the travelling wave. OHC stereocilia are deflected against the TM into which they are embedded, the buttressed 'W' configuration ensuring that considerable force can be exchanged. Excitation of the OHCs generates synchronous mechanical forces which are then transmitted to the BM replacing lost energy and sustaining the TW.

TW amplitude can increase. Increased width and fluid contact area means more inertia, which acts to sustain motion and so opposes the elastic forces restraining displacement. Consequently, wave amplitude increases with distance along the BM. It is potentially maximum at the place where (for the particular stimulus frequency) the force of inertia equals and cancels the elastic restoring force, *i.e.* at 'resonance'.

Because inertial forces increase with frequency, the place along the BM at which this peak in TW amplitude occurs is progressively nearer to the base for higher frequency stimuli. The forward speed of the TW also reduces as inertial forces increase and stiffness decreases, and it eventually comes to a



**Fig. 5 (A–C)** A schematic illustration of the development of the TW along the BM and the sources of OAE. Panels show computed TW envelopes and instantaneous wave patterns in response to two prolonged pure tones with frequencies  $f_1$  and  $f_2$  ( $f_2/f_1 = 1.5$ ). The TW progresses from the base (left) towards the apex (right). In (A), natural damping absorbs most of the stimulus energy before any clear separate excitation peaks for  $f_1$  and  $f_2$  can develop. This corresponds to the ‘dead’ cochlea as studied by von Békésy. Sharp ‘images’ of stimuli  $f_1$  and  $f_2$  can be seen in (B), where damping has been largely removed by linear OHC amplification. In reality, OHC motility is non-linear and this results in intermodulation distortion products being created under the entire  $f_2$  envelope (including  $2f_1-f_2$  and  $2f_2-f_1$ ) as in (C), which then travel to their frequency places at points 3 and 4. (D,E) Show how OHC energy is fed into the TW to replace that lost by damping, and how OAEs can arise. In (D), seven uniformly spaced electromotile hair cells each ‘radiate’ a new bi-directional travelling wave exactly in synchrony with the stimulus TW (top), which has unrealistically constant amplitude for the purposes of this schematic. In the forward (apical) direction, these wavelets are automatically in synchrony resulting in an ‘amplified’ TW (bottom of (D)). OHC wavelets in the reverse (basal) direction cancel each other so that no OAE is generated. In (E) the array is unbalanced, e.g. by a missing or less active hair cell. Amplification still occurs, but the imbalance results in a basal-ward TW (bottom trace). A ‘place fixed’ or ‘stimulus frequency’ OAE (SFOAE) is created. More complete cochlear models have shown that, in order to recreate experimentally observed TW envelopes, amplification needs to occur over only a limited region of the BM, somewhat basally to the actual TW peak. SFOAEs probably emanate from this same region, as indicated in (B), but there is no guarantee of this. In the non-linear case (C), DP energy can escape in a similar way to SFOAEs, i.e. from places 3 and 4 in (C), to form DPOAEs. But with  $f_2/f_1$  between  $\sim 1.15$ – $1.3$ , a substantial amount of the DP  $2f_1-f_2$  escapes directly from inside the  $f_2$  envelope, as indicated in (C). These are termed ‘wave fixed’ emissions<sup>23,25,47</sup>. This figure emphasises the complex origins of OAEs and reasons why frequency-specific OAEs are not strictly ‘place’ specific.

virtual stop at ‘resonance’. A good way to understand the characteristic TW shape (Fig. 5B) is to realise that, as its longitudinal (i.e. apical) velocity reduces, its energy must become compressed into a smaller length of BM. Consequently, its transverse oscillations must increase in amplitude.

Increased transverse motion in the wave causes increased viscous drag in the organ of Corti (see Fig. 4), which removes energy at an increasingly rapid rate. At some place, ‘energy loss’ begins to dominate ‘energy concentration’ and the wave intensity falls precipitously as its energy is



absorbed by the organ of Corti. The overall result is an asymmetric peak of excitation for each frequency component (Fig. 5B).

### *The cochlea as an imaging system*

The TW envelope represents the excitation intensity applied to the organ of Corti as a function of distance along the length of the cochlea. The organ of Corti mechanism then converts BM motion to fluid motion across the IHC stereocilia, leading to neural excitation (see Fig. 4). The tonotopic re-distribution of stimulus energy achieved by the TW is essential in the mammalian cochlea because it facilitates the neural representation of stimulus frequencies well above the maximum nerve fibre firing rate of around 2 kHz. Although the TW is usually credited with frequency separation, the TW envelope is also an 'image' or 'map' of acoustic intensity against acoustic 'source size'. This follows because acoustic radiation efficiency is strongly related to the size of the radiating object, with sound from large objects tending to be dominated by low frequencies and peaking near the apex and with sounds from small objects tending to be dominated by high frequencies, peaking at the base. The functional significance of this is obvious.

Thought of as an 'imaging system', it becomes clearer why the 'quality' or 'sharpness' of the TW envelope is paramount. The resolution of the cochlear imaging mechanism can be characterised by the 'height' and 'width' of the TW excitation peak for each pure-tone component (see Fig. 5A,B).

### *The need for a 'cochlear amplifier'*

In discovering the travelling wave, von Békésy observed the cadaver cochlea to have very poor 'imaging' qualities<sup>16</sup>. He found the travelling wave peak in response to a pure tone stimulus to extend over a third or more of the entire cochlear length (see Fig. 5A). In the healthy living cochlea, the TW peak for low-level pure tone stimulation is much sharper<sup>17,18</sup>. The TW peak covers less than 1 mm and a shift in frequency of just one-third of an octave moves the TW peak to stimulate an entirely different set of sensory cells (see Ashmore, this volume).

The reason for the poor imaging quality of a dead (or deaf) cochlea (Fig. 5A) is high levels of viscous damping. If viscous damping is low, then the TW peak will be sharp and its amplitude will be large. Hearing will be more sensitive and cochlear frequency resolution (and selectivity) will be more acute in a cochlea with less damping.

It is not that cochlear fluids become any 'thicker' or more viscous than water in the deaf or dead cochlea. These viscous losses are physically

inevitable because: (i) sensory cells must absorb stimulus energy to operate; and (ii) in order to reach the inner hair cells, cochlear fluid motion must take place in the extremely constricted sub-tectorial space (Fig. 4B). The energy lost from the TW due to viscous fluid drag in the sub-tectorial space plus energy absorbed by the hair cell themselves is very substantial. In fact the majority of the incident stimulus energy is actually lost before reaching its appropriate frequency place. Under purely mechanical forces, the cochlea cannot develop a strong and sharp TW image.

The mammalian cochlea has evolved a unique mechanism for replacing TW energy lost by viscosity, at least for weak stimuli. The idea of a 'cochlear amplifier' to overcome physical limitations was first proposed by Gold<sup>19</sup>. It was revived following the discovery of OAEs<sup>3,20</sup>, but became a credible possibility only after the discovery of hair cell motility by Brownell<sup>21</sup>. Electro-motility (Ashmore, this volume) is the only known functional characteristic of the outer hair cells, which out-number the inner hair cells by 3:1 (Fig. 4A).

Figure 5 illustrates how OHC motility results in TW amplification. In so far as the OHC motile forces exactly oppose the forces of viscous drag, the degrading effect of the drag on the TW 'image' can be regarded as being neutralised. If OHC forces exceed that necessary to overcome viscosity, then excitation will be increased above that delivered by the stimulus, providing the possibility of amplification – as implied in Figure 5B. This process of TW enhancement in the cochlea by stimulated mechanical energy release parallels with what happens to light inside the laser.

#### *Oto-acoustic emissions and the 'imperfect' cochlear amplifier*

The cochlear amplifier is physically essential to the high sensitivity of hearing and to the formation of a sharp ~0.25 octave resolution tonotopic 'image' of the acoustic environment along the length of the cochlea. As Figure 5 indicates, OAEs are a by-product of this 'cochlear amplifier'. They arrive in the ear canal as a result of BM disturbances that escape from the cochlear amplifier mechanism and travel away from the sensory cells back to the base of the cochlea. Here the 'up-and-down' motion of the BM exerts a differential oscillating fluid pressure on the oval and round windows causing vibration of the ossicles and ear drum and hence OAEs.

OAEs can only be generated if the cochlear amplifier mechanism is present and to some degree operational. But, paradoxically, the reasons why vibrations are sent back to the base to form OAEs all relate to natural imperfections in this mechanism. What kind of amplifier imperfections result in OAEs?

As illustrated in Figure 5B, if OHC motility is not completely uniformly distributed, a stimulus frequency OAE will be generated. In a

travelling wave amplifier, any lack of uniformity in construction or function places an upper limit on the level of stable amplification. By scattering amplified energy back to the base of the cochlea, energy is wasted and appears as stimulus frequency OAEs.

It is not only spatial imperfections that can generate OAEs. If the forces exerted by OHCs on the BM do not exactly follow the stimulus waveform (*i.e.* if the OHC electromotility is 'non-linear'), they will add distortion signals to the forward travelling wave, which are one cause of aural combination tones. As explained in Figure 5, OHC electromotility distortion can also propagate back to the middle ear *via* two competing routes, to cause 'wave' and 'place' fixed 'distortion product' OAEs in the ear canal<sup>22,23</sup>. TEOAEs contain both stimulus frequency and distortion product OAEs according to Yates and Whithnell<sup>24</sup>.

Another type of 'imperfection' arises from positive feedback, leading to instability and self-oscillation and is a common property of TW amplification systems. In the cochlea, most energy travels apically and is absorbed, but any energy that escapes basally to reach the base can be partially reflected back, forming a new forward TW. This can re-stimulate the OHC mechanism. Under conditions of high amplification, endless recirculation of the TW leads to sustained oscillation inside the cochlea and to spontaneous OAE of one or more pure tones into the ear canal.

#### *Oto-acoustic emissions as a 'window' on cochlear function*

To what extent can the functional status of a cochlea be characterised and quantified using OAEs? OAEs are only a by-product of cochlear function. As we have seen, the factors which govern the escape of energy to produce OAEs relate to 'imperfections' of the cochlear amplifier, *i.e.* non-linearity and irregularities. Also, several different cochlear locations may contribute to a single frequency component of an OAE and these may fortuitously summate or interfere with each other (Fig. 5C). The transmission back to the ear canal also depends on individual middle ear characteristics. The interplay of all these factors cannot yet be accurately modelled, not least because most parameters are unknown. It is not surprising, therefore, that individual healthy ears differ greatly in the level and the spectrum of the OAEs they exhibit. Stimuli of slightly differing frequency or spectral composition can give rise to quite different OAE patterns. Taking an 'average' OAE characteristic over a range of stimuli provides a more meaningful description of cochlear status, but even so the intensity of OAEs alone is a very imperfect index of cochlear status.

The 'frequency' at which an emission can be evoked is more significant. OAEs are frequency-specific responses and tend to emerge only in

frequency bands where hearing is near normal. This provides a useful pointer to normally and abnormally functioning parts of a cochlea. But, as Figure 5A–C clearly demonstrates, with OAEs, frequency specificity does not always ensure ‘place’ specificity, even when pure tone stimuli are used, as with DPOAEs.

Two general rules apply to DPOAE production which are relevant to clinical applications. In any non-linear system, intermodulation distortion is always strongest when the two interacting signals have similar levels at the non-linearity. In the cochlea, the relative intensity and relative frequency of the two stimuli determine where along the organ of Corti this ‘physically ideal’ condition is met. Referring to Figure 5B, even when the input levels of  $f_1$  and  $f_2$  are the same, making TW ‘ $f_1$ ’ and TW ‘ $f_2$ ’ intensities similar at the base, at the place where TW ‘ $f_2$ ’ peaks its intensity is much larger than for the TW ‘ $f_1$ ’. Making the lower-frequency stimulus relatively more intense than  $f_2$  at the input will shift the place of greatest intermodulation towards the  $f_2$  peak of the TW and so enhance DP production. Increasing the  $f_1$  level too much will be counter-productive. Therefore, for each stimulus level, there is an optimum stimulus intensity ratio for maximum DP production.

DP production does not ensure DP emission, which is governed by the second rule. The extent to which all the elemental OHC DP sources reinforce each other to produce a strong backwards DP travelling wave (and hence a DPOAE) depends on the spatial distribution of DP phases. This depends on the relative travelling wave velocities of  $f_1$  and  $f_2$  at the non-linearity. When the ratio  $f_2/f_1$  is nearly one (e.g. 1.05),  $f_1$  and  $f_2$  TW velocities are very similar at all points. The phase distribution of DP elements then necessarily forms a forward (apical) TW with little DP sent backwards to form a DPOAE. Even so, some DPOAE will escape via the SOAE route, as illustrated in Figure 5C. For large  $f_2/f_1$  (e.g. 1.5), the densely packed phase changes within the  $f_2$  envelope generate an undulating DP phase distribution that will be largely self-cancelling and little DP wave will propagate from that region. However, because there is a minus sign in ‘ $2f_1-f_2$ ’, for  $f_2 > f_1$ , the spatial phase gradients of TW ‘ $f_1$ ’ and TW ‘ $f_2$ ’ counteract each other in  $2f_1-f_2$  DP production. Consequently, at some optimum  $f_2/f_1$  ratio (around 1.2), the relative velocities of TW ‘ $f_1$ ’ and TW ‘ $f_2$ ’ are such that the spatial distribution of DP elements actually becomes that of a backward travelling wave over a considerable length of OHCs. Maximum DPOAE is delivered to the ear canal via this ‘wave-fixed’ mechanism<sup>25</sup>. Interestingly, there is no optimum frequency ratio for the ‘alternative’ DPOAE  $2f_2-f_1$  (see Fig. 5C), which emanates from a place basal to both  $f_1$  and  $f_2$  peaks over a wide range of  $f_2/f_1$  ratios. The clinical significance of this DPOAE has not been fully evaluated.

Clinical DPOAE measurements are generally made with both stimulus intensity and frequency ratios optimised for maximum DPOAE  $2f_1-f_2$

intensity<sup>26</sup>. As Figure 3 illustrates, many different DPOAEs co-exist and their generation is intimately linked to the operating characteristics of the outer hair cells. It is possible that we will one day be able to reconstruct OHC operating characteristics from DPOAE data.

Spontaneous OAEs (SOAEs) are typically highly stable pure tones of level -10 to 30 dB SPL, which are found in 30–40% of healthy young ears<sup>27,28</sup>. Their presence indicates simply a 'chance' combination of factors. Strong TW amplification must co-exist with irregularities to cause a strong wave to be returned to the base and the proportion reflected back into the cochlea by the middle ear must, after re-amplification and re-emission, be sufficient to sustain a continuous oscillation of the middle ear and along a substantial section of the BM. The round trip travel time also has to be exactly right for this to occur and so can happen only at one precise frequency, just as in the laser. The precise frequency of an SOAE does not imply an origin at a precise place in the cochlea, but only a particular co-incidence of travel time and reflection from an ill-defined region of high OHC activity. But, because of their intrinsic stability and critical dependence on cochlear status, SOAEs are, when present, particularly sensitive indicators of metabolic and physiological changes in the cochlea.

All OAE forms show a high degree of sensitivity to changes in cochlear status. Exposure to noise levels causing temporary threshold shift depresses TEOAEs<sup>29,30</sup> and low stimulus level DPOAEs<sup>31</sup>. Changes in cerebrospinal fluid pressure induced by posture changes affect SOAE frequency and evoked OAE intensity – probably by their influence on cochlear fluid pressure and stapedial position<sup>32</sup>. Drugs known to depress hearing, including aspirin and quinine, also depress OAEs, and loop diuretics known to depress the endocochlear potential also depress OAEs<sup>33,34</sup>.

OAEs also exhibit a physical analogue of 'masking' where the perception of one sound is blocked by another (see BJM Moore, this volume). This may indicate that some forms of masking originate preneurally in the cochlea. Tracing the suppression of an OAE response to one tone by adjusting the intensity and frequency of a second suppressor tone allows an OAE suppression-tuning curve to be constructed<sup>35–37</sup>. The sharpness of such curves confirms the close association between OAEs and auditory function, and demonstrates that sharp mechanical tuning is present at the cochlear level.

Perhaps the most interesting suppression effect is the slight depression of OAE level (by 0.5–3 dB) caused by noise or irregular acoustic stimulation applied to the contralateral ear<sup>38,39</sup>. First discovered by Collet *et al* in 1990, this effect has been identified as being largely due to the influence of the medial cochlear efferent system. This terminates directly on the OHC bodies and presumably normally plays a role in the day-to-day maintenance of effective OHC status. The contralateral

suppression effect is best seen on TEOAEs evoked by a uniform train of click stimuli of less than 74 dB SPL p.e., but can also be seen on DPOAEs evoked by primaries below 60 dB SPL. The neural mechanism and function remain somewhat obscure. Absence of a contralateral OAE suppression effect can be the result of a brain stem lesion, but Collet has also reported it in certain stages of sleep, and it can be absent in some healthy individuals. It has even been suggested that absence of contralateral OAE suppression may correlate with autism and dyslexia<sup>40-42</sup>. The phenomenon of contralateral OAE suppression is not well understood.

In general, OAE responses carry a large amount of information about the status, activity and environment of OHCs, which we are currently unable to interpret. OAEs tend to be dominated by microscopic details of little relevance to hearing. Nevertheless, OAEs provide the only detailed non-invasive window on the cochlea and by their very presence confirm normal presynaptic cochlear function. Although useful today, if we can learn how to extract definitive data on OHC status from OAE data, then their clinical importance will be greatly enhanced.

## Key points for clinical practice

OAEs are already an essential part of the audiological diagnostic test battery<sup>43</sup>. Key points for clinical use are summarised below.

### Recording otoacoustic emissions

- Advanced OAE techniques need a sound attenuating booth, but useful OAEs can be made in a quiet office environment. Background room noise levels of 40 dB or below are recommended. A good probe fit helps block out external noise, although this effect is minimal with neonate ears. Short bursts of more intense noise (e.g. speaking to the patient, or a cough), which can be detected and rejected by the instrumentation, are less troublesome than continuous or reverberant sounds. Patient movement is also not a problem with OAEs provided it does not result in cable-rub noise. Jaw action, swallowing and vocalisations cause ear canal noises, which can prevent good OAE recordings.
- Patency of the ear canal is essential for successful recordings. Obstruction by wax in older patients, or by fluid or birth debris in neonates, or by collapse of the canal in the latter, prevent OAEs from reaching the ear canal and are major causes of OAE recording failure. Sleep and sedation have minimal effects on OAEs<sup>44</sup>.

### Types of otoacoustic emissions

- There are two widely used OAE measurements: transient evoked OAEs (TEOAEs) and dual-tone evoked distortion product OAEs (DPOAEs).

DPOAE measurements are better suited to advanced clinical investigation on adult patients, even though DPOAE analysis is complex and interpretation is difficult. The DP technique is more flexible and potentially more powerful than TEOAE analysis, having a wider useful frequency range. Waveform-based TEOAE measurements, as originally used in universal new-born hearing screening programmes, are also useful as a sensitive initial screen prior to full clinical examination. TEOAEs are also more sensitive to cochlear status changes manifested in subtle changes in the TEOAE waveform. DPOAE instruments can be used for screening with an appropriately low stimulus level (*e.g.* 65/55 dB SPL), but DPOAE screening instruments are generally not flexible enough for clinical applications.

### Middle ear factors

- OAE detection is affected by conductive losses and OAEs will be absent if there is effusion, glue, otosclerosis or ossicular dislocation. Moderate negative pressure and tympanic perforations not exceeding 30% result in the attenuation of only the lower frequency OAEs. Grommets do not greatly affect OAEs. Absent OAEs can re-appear following effective middle ear treatment or surgery if the residual conductive loss is very small and the cochlea is normal. Large and unusual ear canals and perforations can disturb stimulus delivery by 'ringing' and so prevent successful recording. This does not arise with neonates, but their ear canals are extremely small and this needs to be accommodated in the selection of probe size and stimulation intensity.

### Oto-acoustic emissions and the nature of sensory hearing loss

- OAEs come exclusively from outer hair cells which do not themselves activate primary auditory nerve fibres, yet a strong relationship exists between the absence of OAEs and hearing loss. This forces a re-definition of the term 'sensory hearing loss'.
  - † **Sensory transmissive loss** can be defined as hearing loss resulting from dysfunction of outer sensory hair cell group. Absence of the 'cochlear amplifier' allows natural damping to remove most stimulus energy from the cochlear travelling wave and lowers the resolution of the cochlear imaging mechanism. Inefficient transmission of excitation to the IHCs causes loss of hearing sensitivity and frequency selectivity. Since there remains a pathway for stimulation to reach the IHCs, profound hearing loss cannot be caused by OHC dysfunction alone. Total OHC failure is estimated to cause no more than 60 dB hearing loss. Loss of OAEs with a normal middle ear indicates sensory transmissive loss.
  - † **Sensory transduction loss** can be defined as hearing loss resulting from failure of inner hair cells to respond and activate the synapsed auditory nerves. This could give rise to any degree of hearing loss from mild to profound since the auditory nerves themselves have no sensitivity to sound stimulation. Loss of frequency selectivity would not necessarily accompany threshold elevation in a pure sensory transductive loss, and OAEs would be normal.

- It is clear from the high correlation between sensory loss and OAE absence that most sensory losses are of the sensory transmissive type. This makes sense, as the outer hair cell mechanism is both highly specialised and highly vulnerable to degradation by excessive noise, anoxia or ototoxic agents. OHCs selectively amplify weak stimuli which would otherwise fall below the threshold for IHCs to trigger a neural response and as a consequence the symptoms of sensory transmissive loss necessarily include loudness recruitment in addition to threshold elevation and reduced frequency selectivity (see BJM Moore, this volume)<sup>45</sup>.

#### Interpretation of otoacoustic emissions

- The presence of robust evokeable OAEs across the key speech frequency range (1.0–4 kHz) indicates a useful degree of normal function in both the middle ear and cochlea and further indicates that speech and language development will not be greatly impeded by peripheral auditory dysfunction. For clinical purposes, it is useful to record OAE status as a function of frequency, averaged over one-half or one-third octave frequency bands. Higher resolution has little physiological meaning.
- The absence of OAEs without middle ear pathology or acoustic obstruction strongly indicates sensory transmissive hearing loss. Depending on the type and intensity of stimulation, OAEs can reveal threshold elevations as small as 20 dB HL and the frequency 'resolution' of OAEs can be as good as one-half octave. The amount of threshold elevation cannot be predicted with any useful accuracy, but if DPOAEs are present with TEOAEs absent, this suggests mild-to-moderate loss only.
- OAEs are normally very stable with time and are valuable as a sensitive monitor of changes in cochlear (and middle ear) status over time, *e.g.* in relation to sudden hearing loss, Ménière's disease or noise trauma.
- Although OAEs can differ enormously between healthy ears, they are usually quite similar in the left and right ears. Substantial left-right differences may, therefore, indicate pathology.

#### Differential diagnosis

- OAEs are expected to be present in sensory transductive, neural, central and psychogenic hearing losses. OAEs can be either present or absent with 8th nerve tumours, depending on whether the cochlear blood supply has been compromised. If present, OAEs indicate the possibility of hearing recovery with a conservative surgical approach<sup>43</sup>.
- OAEs are preneural responses indicating healthy cochlear status and cannot be used to detect sensory transductive or neural hearing losses. With neonates, the absence of an OAE response in clear dry ears should be treated as a strong risk factor for sensory hearing impairment. However, other risk factors need to be considered before presence of an



OAE is taken as evidence of normal hearing. Hyperbilirubinaemia or any risk of neurological damage requires that an ABR test also be conducted.

- Auditory neuropathy is indicated by the presence of normal OAEs but the absence of normal ABR responses. In such rare cases, the application of hearing aids with high amplification may be counterproductive, so in infants ABR and OAE testing should precede hearing aid selection.

### Special applications

- OAEs can be slightly depressed by contralateral noise stimulation if the medial cochlear efferent system is operational. The significance of the absence of this effect is not clearly understood but may help clarify the nature of certain neural pathologies<sup>48</sup>.
- The objective nature of OAEs can be useful in the investigation and management of inorganic hearing loss by demonstrating normal cochlear function to the patient.
- Serious tinnitus is almost never associated with OAEs, but rather with their absence (see Baguley, this volume). Spontaneous OAEs can sometimes be perceived as tinnitus and occasionally cause unnecessary anxiety. Typically, in such cases, hearing threshold is normal and the tinnitus is mild, tonal and easily maskable by noise. Patients can be reassured by the objective demonstration of spontaneous OAEs.

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