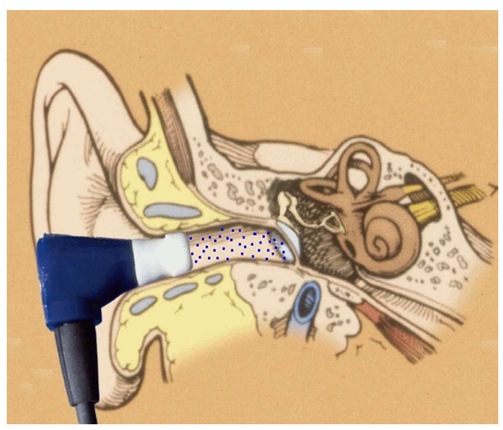
OAE Theory and Practice

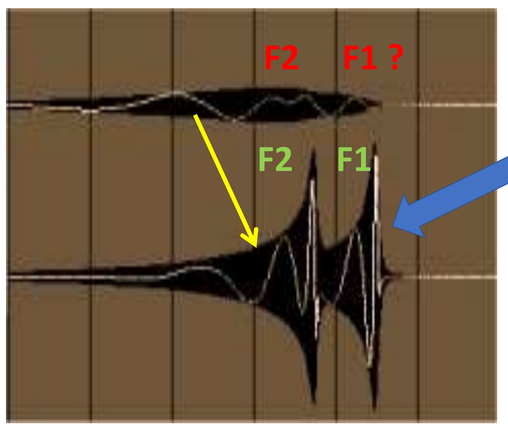
Do we understand OAEs?
Are recordings optimal?

EAR CANAL ACOUSTICS



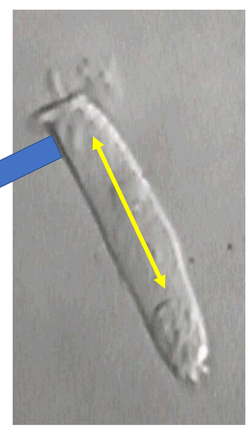
It seemed simple once. You presented a known stimulus level to the ear, checked the level through the probe microphone and recorded the emission. But reflectance has shown us that we can't accurately control the stimulus to the cochlea by simply setting the sound pressure at the probe. The probe fit, the impedance of the probe, ear canal and ear drum all influence what the cochlea receives and OAE level we record.

TRAVELLING WAVES

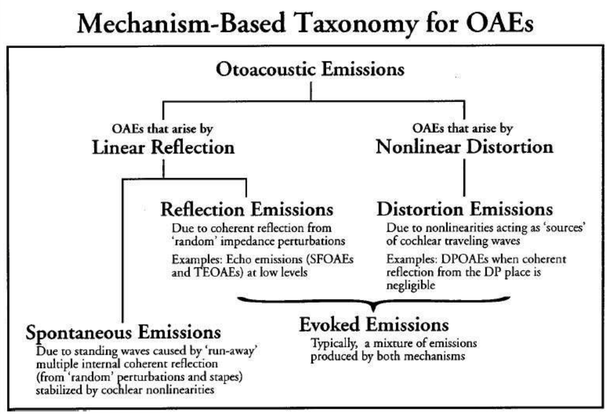


Acoustic stimulation starts travelling waves on the basilar membrane distributing stimulus frequencies tonotopically to hair cells. Before OAEs everyone subscribed to von Bekesy's conclusion from cadavers ears that waves from close frequencies f1 ad f2 were not well separated (top). Waves died away quickly and never reflected back out. But in the early 1980s Johnstone and others saw strong sharp wave separation in healthy cochleae. Brownell and others found that outer hair cell were electro-motile and this active process greatly enhanced the travelling waves. (bottom). Surely OAEs must come from this 'cochlear amplifier'. But how did that amplifier actually work?

ELECTROMOTLITY

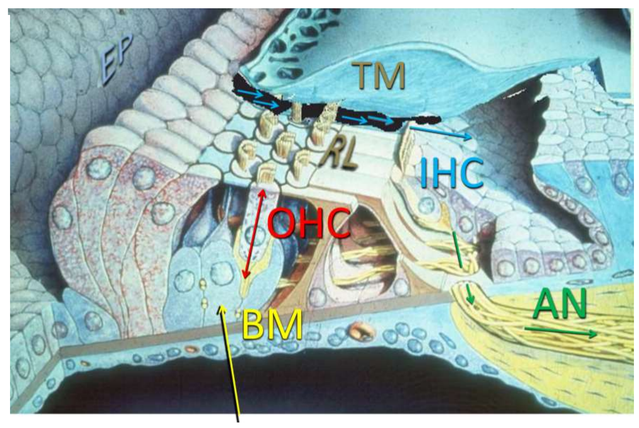


OAE GENERATION



By the early 1990s most thought OAEs were 'explained' by OHCs and the 'cochlear amplifier. But how did OAEs escape? Kemp had identified two types of emission (wave and place fixed) and Brown had identified two interfering components in DPOAEs. In 1995 Zweig and Shera explained TE and SFOAEs as due to coherent reflection of the travelling wave from small irregularities. In 1998 Shera and Guinan (above) explained that distortion emissions didn't need reflection but could be reflected at their frequency place – explaining Brown's 'two sources' of DPOAE.

ORGAN OF CORTI MICROMECHANICS



The organ of Corti is the 'engine of the cochlea'. It determines hearing quality and sensitivity. It is also the source of OAEs. It's a micro-machine that converts 'up/down' basilar membrane (BM) motion into 'horizontal' fluid motion between reticular lamina (RL) and tectorial membrane (TM) deflecting the stereocilia of the inner hair cells (IHC) which then triggers auditory nerve (AN) spikes. The mechanism is power-assisted by outer hair cell (OHC) electro-motility, drawing energy from the endocochlear potential (EP). OAEs arise when some of this energy escapes.

GOLD THOUGHT OF IT FIRST – IN 1948!

In 1948 Thomas Gold (left) wrote that the cochlea NEEDED to have a built-in amplifier and positive feedback to achieve sharp tuning and overcome cochlear fluid viscosity. He said the proof of his idea would be sound emissions from the ear!

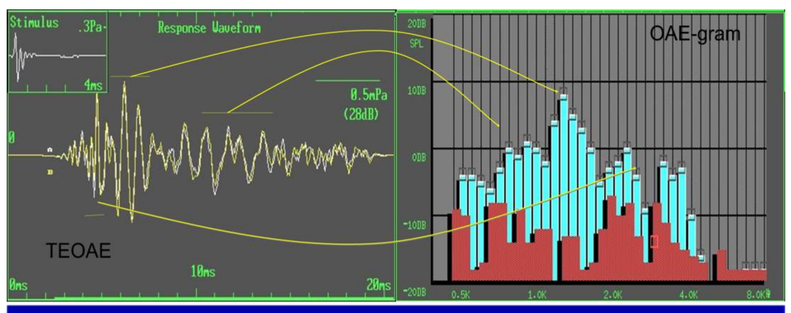
Gold proposed piezoelectric motility in the tectorial membrane as the 'active process'. **He predicted spontaneous acoustic emissions** would occur when the amplifier became momentarily unstable and generated feedback and tinnitus.

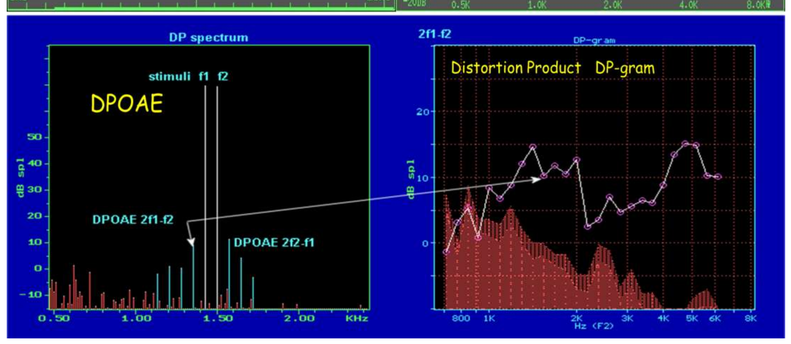
Gold tried unsuccessfully to detect such emissions during transient tinnitus using a telephone microphone pressed to the ear. We know now that such tinnitus isn't caused by OAEs.

Von Bekesy didn't accept Gold's theory and he was happy to believe neural processing provided fine frequency tuning.

When Gold heard stimulated emissions and DPOAEs had been recorded he said '*My theory didn't predict that*'. For Gold's theory to work the cochlea had to be smooth and linear.

TEOAEs AND DP-GRAMS, THE TWO COMMONLY USED METHODS FOR RECORDING OAEs



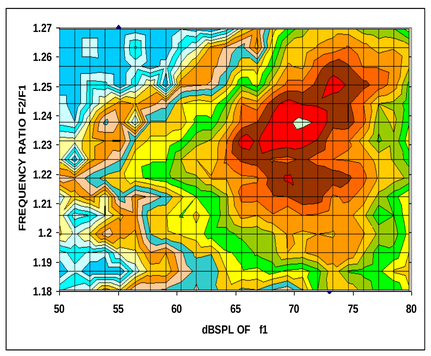


These two recording methods have changed little in the 30 years since they entered clinical service in 1988.

For Transient Evoked OAEs (top) the ear canal acoustic response to clicks is captured as a waveform (top left) and then transformed into a spectrum display (right) showing OAE intensity with frequency. Different parts of the cochlear response with different frequencies at different times so its possible to record from them all simultaneously and then separate them by signal processing. Basal high frequency responses arrive first, lower frequency responses later, as indicated by the yellow lines. TEOAEs are frequency-specific. They are widely used for infant screening.

DPOAEs (bottom) are identified by frequency analysis of ear canal sounds. Multiple intermodulation distortions can emerge in response to two closely spaced tones f1 & f2. To construct a DP-gram the level of just one of these distortions, one with a frequency 2f1-f2 is plotted as f1 & f2 are incremented in steps through the test. Many other OAE measurements are possible.

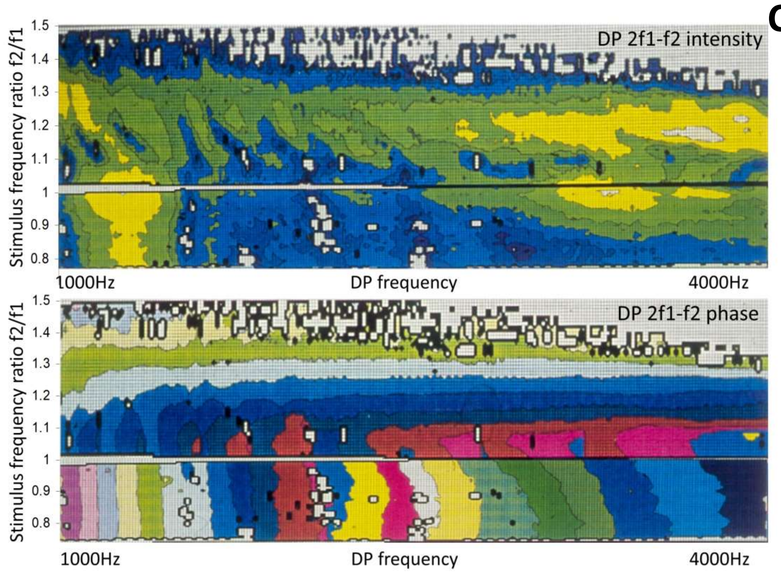
WHY DO WE MAKE L1 > L2 ? AND F2/F1=1.2?



When recording DPOAEs we make the lower frequency tone f1 stronger than f2 especially for low stimulus levels. That's because f1 has to travel further to reach its peak and so is weaker as it passes over f2 waves. Most distortion is made where the f1 & f2 <u>waves</u> are equal, usually not when L1=L2 .

Travelling waves get much sharper at lower levels so L1 needs to be made much larger then L2. The 'equal waves' principle means the amount of distortion generated also depends on how close f1 and f2 are. Closer f1 & f2 need more similar L1 and L2 levels. (see 'Travelling Waves' above). The contour map above shows how DPOAE dBSPL depends on the frequency and intensity ratio of the two stimuli f1 and f2. Here, for an f2 level of 65dBSPL, the strongest DPOAE is achieved with a frequency ratio of 1.24 with an f1 level of 70dBSPL. But this optimum relationship varies with stimulus frequency, level and between subjects. <u>Current instruments don't allow for this</u>.

CURRENT METHODS ONLY SCRATCH THE SURFACE



In 2001 Knight and Kemp devised a way of mapping DPOAEs that revealed the different character of wave fixed 'distortion' and place fixed 'reflection' emissions. Shown left, this DP map shows DPOAE intensity (top) and phase (bottom) against DP frequency for a range of f2/f1 ratios. Ratios above about 1.15 give mostly 'distortion' wave fixed emissions. Ratios below 1.1 down to 0.8 don't allow distortion to escape by direct emission. Only 'reflected' DP emissions are present (vertical phase bands)

Conventional clinical DPOAEs represent only one horizonal line through this map at f2/f1=1.2 - and so only scratch the surface when it comes to what OAEs can tell us about the individual cochlea. And this map cover one L1,L2! (Note 2f2-f1 for ratios <1)

The DPOAE mapping method has proved valuable in research by Stagner Martin and Martin, aimed at finding out how and where emissions originate. Adding a third tone changes the map because of suppression. Subtracting the old from new map reveals exactly where the changes occurred. It turns out not all DPOAEs come from the f2 and DP places.

Individuals can have quite different DPOAE maps and there is great potential for the mapping technique to reveal subtle changes in cochlear condition e.g. as a result of noise exposure, or ototoxic drug or just age. However OAE mapping take time!