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The Basics, the Science, and the Future Potential of Otoacoustic Emissions

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The human ear is an exceptionally sensitive and delicate sensory device. It collects sound energy, manipulates it, and projects it onto an array of only 3000 sensory hair cells in a spatial pattern designed to overcome a fundamental biologic problem. The problem is that everyday sounds carry potentially useful information at an information rate thousands of times beyond the capacity of nerve fibers to transmit. The shortfall is similar to the difference in data capacity between the first dial-up modem computer connections and the broadband cable or wireless connections we enjoy today, and it is a problem all animals face. The hearing of frogs, and to a lesser extent of birds, is limited by the data transmission capacity of nerve tissue. But mammals have a mechanism that largely overcomes this limitation. The problem has been overcome not with a high-speed data link to the brain, but rather by a mechanism capable of subdividing, compressing, and extracting the essential information from sound before it is converted into neural code. The finer details of the mechanism that allows the mammalian cochlea to achieve this feat is the subject of intensive auditory research. Of most relevance to this book is the fact that in the great majority of cases of sensory hearing loss there is degradation of this mechanism. A means of examining this mechanism in operation is therefore an essential component of audiology.

◆ OAEs and Their Clinical Usefulness

The phenomenon known as otoacoustic emission (OAE) provides us with an objective “window” onto this uniquely important auditory mechanism. This window, albeit an oblique window, exists because in the process of manipulating and amplifying the energy it receives from the middle ear the cochlea loses control of a small amount of energy, throwing it back to the middle ear. This gives rise to sounds in the ear canal that can easily be recorded by a microphone inserted in the sealed ear canal. These signals

tell us indirectly about the progress of applied stimulation inside the cochlea, and from this the state of health of the cochlea can be inferred.

The possibility of screening for hearing loss with OAEs was quickly recognized (Johnsen and Elberling, 1983) but only adopted in major programs many years later (White and Behrens, 1993). Clinical applications also began very slowly after nearly a decade of laboratory research. The two OAE recording techniques originally proposed for clinical applications by Kemp et al (1986) remain the most commonly used today. **Figure 1-1** illustrates these two methods: the click or transient evoked otoacoustic emission (TEOAE) method and the distortion-product otoacoustic emission (DPOAE) method. A TEOAE response (**Fig. 1-1A**) typically consists of a complex sound waveform obtained in response to repeated clicks, which can be subsequently analyzed to chart OAE strength as a function of frequency (**Fig. 1-1B**). Additional examples are provided on the CD-ROM with this book (see How to Use the CD-ROM, which precedes Chapter 1). DPOAEs are “intermodulation” distortion tones (explained later) that the cochlea generates in response to a close pair of stimulus tones (**Fig. 1-1C**). During the DPOAE measurement process the frequency of the stimulus pair is stepped across the hearing range of interest, and the intensity of one selected distortion product emission (usually of frequency $2f_1 - f_2$) is recorded as a function of frequency forming a DP-gram (**Fig. 1-1D**). Each technique has specific advantages. The DPOAE technique can capture OAEs up to higher frequencies than TEOAE. Both techniques suffer from low-frequency noise interference from the patient and the test room, but the TEOAE method can be a little more effective at low frequencies. TEOAEs show subtle changes in cochlear status more readily, and the technique is more suited to simultaneous observations across frequency than DPOAE. DPOAEs can be measured with more intense stimulation than is practical with TEOAEs, and this is useful in recording responses from ears with slightly elevated threshold.

Otoacoustic emissions have proved to be especially valuable in audiology because in the vast majority of ears with hearing impairment preneural processing is degraded, leading in most cases to an absence of sound emission. The noninvasiveness, simplicity, and speed of otoacoustic emission recording have contributed greatly to their widespread use in newborn hearing screening (Norton et al, 2000a, and Chapter 13 in this book). But their use extends beyond screening. OAEs arise from the physiologic and physical properties of sensory outer hair cells (see Chapter 2), which are the most vulnerable components of the auditory system. This has made otoacoustic emission recording a key element of the diagnostic audiologic test battery and an integral part of many auditory research programs. Relative increases and decreases in cochlear activity can be monitored. The clinical role of OAEs is expanding as laboratory research develops better methods of evoking OAEs and interpreting OAE data.

Protective and even restorative treatments for sensory hearing impairment are beginning to be developed. The evaluation and application of such treatments will require a means of precisely monitoring the working characteristics and status of patient's cochleae. As understanding of the genetic factors underlying early loss of hearing grows sensitive, techniques will be needed to define and explore auditory phenotypes. Otoacoustic emissions have the potential to do this, and so their clinical and research role is set to increase.

This chapter focuses on the essentials and fundamentals of otoacoustic emissions and on the science behind them. The history of the discovery of OAEs and of the development of OAE science and technology will be used to introduce and illustrate issues that are important to a full understanding of OAEs and for an appreciation of their future potential and limitations. No attempt is made to give a comprehensive review of research in any area but many of the topics introduced here are dealt with in depth in other chapters of this book.

The cochlea is a dynamic and active mechanism—an “active micromachine”—that needs to be visualized at work to be properly understood. Accordingly, this book is accompanied

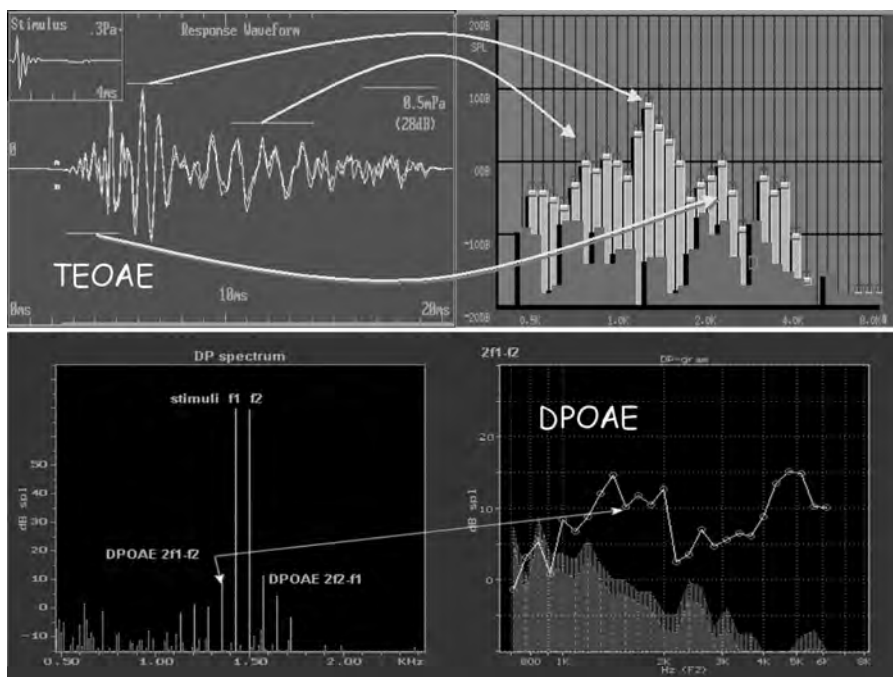


Figure 1-1 Illustration of the two most common OAE recording methods. **(A,B)** TEOAE recording. **(A)** The ear canal sound pressure waveform from a healthy adult ear following the presentation of a click stimulus of intensity 84 dB SPL peak equivalent (approximately 60 dB SL, sensation level) presented at a rate of 50 per second. The small panel inset shows the waveform of the stimulus in the ear canal on the same time scale but on an approximately one-thousandth amplitude scale. The small oscillations seen in the stimulus waveform (**A**, top left) reflect ear canal and middle ear properties. The OAE response is delayed with respect to the stimulus and spread over 20 ms. Its maximum amplitude is 0.5 mPa (28 dB SPL). In practice, TEOAE responses to repeated click presentations are averaged to overcome noise contamination. Two independent measurements are usually obtained and graphically overlaid to demonstrate reproducibility. Their correlation is computed as a measure of noise contamination. The first few milliseconds of the response waveform is typically set to zero (windowed out) to remove any contamination by the acoustic response of the ear canal and middle ear. This contamination is further reduced by scaling and subtracting responses to clicks at two, typically 10 dB different, stimulus intensities, called “nonlinear stimulation” (see Kemp et al, 1986). The example shown took 20 seconds to record. **(B)** The frequency analysis of this TEOAE waveform (light bands) showing that this response contains a wide range of frequencies up to 5 kHz. The darker bands show the noise level obtained by analysis of the differences between two independent recordings. TEOAE responses can be analyzed retrospectively in wide or narrow frequency bands. The smaller the analysis bandwidth, the lower the measured sound pressure level. The resolution here is one-eighth octave bands. The resolution limit is a bandwidth of $\Delta F = 1/T$ Hz, where T is the time sweep length. In this example $T = 20$ ms and $\Delta F = 50$ Hz. Frequency components present in the click stimulus are differentially delayed in the response (frequency dispersion). Arrows show the approximate correspondence of features in the waveform and in the spectrum. Lower frequencies (longer waves) emerge later in the response. The highest frequencies are from the earlier part of the response. Some of the highest frequencies are lost in the windowing process at the start and this limits the high-frequency range of the technique. **(C,D)** DPOAE recording. **(C)** The spectrum of sound present in the ear canal during sustained stimulation by a pair of tones of frequencies $f_1 = 1425$ Hz and $f_2 = 1500$ Hz, each of 70 dB SPL (the two tallest spectral lines). The shorter spectral lines on either side of the stimuli and spaced one interstimulus frequency interval apart are the distortion product emissions. **(D)** The line is the intensity of the closest lower frequency distortion product [frequency = $f_1 - (f_2 - f_1) = 2f_1 - f_2$ (as it is usually written)] plotted as the stimulus frequencies are stepped (or swept) across the auditory range. The filled area indicates the noise present at other non-DPOAE frequencies. The resolution here is one-eighth octave as with the TEOAE example. With DPOAE the resolution is determined by the frequency steps applied during the stimulus during recording. For demonstration purposes in this example, the ratio f_2/f_1 has been made quite small (1.05:1) to evoke multiple DPOAEs. The normal clinical ratio is 1.2:1, which evokes only two or three significant DPOAEs. The high-frequency limit of the DPOAE technique is set by the probe transducers and by the acoustic difficulties of achieving accurate sound levels at the eardrum. (Note: These TEOAE and DPOAE examples are not from the same ear.) See also Kemp 1998.

by a CD-ROM containing animated or audioenhanced versions of many of the illustrations in this chapter, which will help in the appreciation of the science behind OAEs. These may also be useful in teaching programs (see How to Use the CD-ROM, which precedes Chapter 1; this CD-ROM also contains a 15-minute video interview playable on a PC, in which the discovery of OAEs and various topics raised in this chapter are discussed and further illustrated).

OAE in Clinical Practice Today: A Commentary

The longest standing and most common application of OAE is in screening—mostly but not exclusively confined to newborns. This application relies on the empirical finding that OAEs are generally absent in moderate and more severe sensory deafness. OAE was perhaps the first audiometric screen to be proved in controlled trials involving a total of tens of thousands of infants (Kennedy et al, 1998; Norton et al, 2000b; White and Behrens, 1993). Screening applications require OAE detection to be fast, reliable, sensitive, and tolerant to background noise. Both TEOAE and DPOAE techniques can easily meet these requirements, largely because healthy infant ears emits strong OAEs—up to 30 dB SPL. The biggest drawback of the technique for screening is its inability to distinguish between sensory loss and minor middle ear dysfunction such as might be due to birth fluid and debris, which greatly attenuate OAEs. This can lead to high (approximately 20%) false-positive rates in newborns' tests in the first few hours after birth, but thereafter specificities exceeding 95% and sensitivities in the well-baby population exceeding 99% are commonly claimed in well-baby programs. The very nature of the OAE signal lends itself to frequency specific analysis and, unlike screening auditory brainstem response (ABR), most OAE screening is frequency specific to some degree. This is good because it allows attention to be focused on the frequency range most important for speech and language development (e.g., 1.5 to 4 kHz). In general OAE screening techniques are based on detection and not quantification of OAE activity, and this does not require elaborate stimulus manipulations or response analysis. OAE screening instruments do not in general have the flexibility required for clinical applications more complicated than screening.

In clinical applications we exploit specific characteristics of OAEs. The expectation in many applications is that OAEs provide a quantitative frequency-specific measure of cochlear activity closely correlated with hearing threshold. In other types of application the expectation is that OAEs will faithfully record subtle changes in cochlear status of relevance to hearing function. To what degree are these expectations justified?

The frequency specific character of OAEs is central to many clinical applications but it is often misunderstood. OAEs can give us a measure of cochlear emission activity as a function of frequency. It is logical to expect that an OAE response to a stimulus centered on, say, 2 kHz arises directly from the sensory cells, which play a part in the hearing of 2 kHz. The fact that the OAE response is actually at, or in the case of DPOAE near to, the frequency of the applied stimulus encourages this assumption. Such an assumption is known to be false for clinically recorded cochlear microphonic (CM) responses, which mainly come from the basal portion of the cochlea whatever the stimulus frequency. The assumption is much more accurate for the OAE response than for the CM, but with important reservations. To associate OAE frequency with the sensory cells that service that frequency is to associate OAE frequency with place in the cochlea. But the frequency of an OAE does not uniquely identify a specific place in the cochlea just as a stimulus of a particular frequency does not excite only one single point on the basilar membrane (BM). The place responsible for an emission is not necessarily the exact frequency place for hearing. In a typical clinical DPOAE measurement with, for example, f_1 at 2 kHz and f_2 at 2.4 kHz, the $2f_1 - f_2$ distortion we measure is probably generated by several hundred outer hair cells extending over a region of the BM most sensitive to frequencies between 2.4 and 3.4 kHz (a half-octave range). Depending on the stimulus configuration most of this distortion