AGE RELATED CHANGES IN EVOKED OTOACOUSTIC EMISSIONS AND RELATIONSHIP OF EARCANAL VOLUME TO AMPLITUDE

AN INDEPENDENT PROJECT WORK SUBMITTED
IN PART FULFILMENT FOR THE
FIRST YEAR M.Sc.
(SPEECH AND HEARING)
TO THE UNIVERSITY OF MYSORE.

Register Number M 9523

ALL INDIA INSTITUTE OF SPEECH AND HEARING
MYSORE - 570006
1996

DEDICATED TO MY PARENTS
"PARENTS AND LOVE DIFFER ONLY IN NAME
FOR THE MIRACLES THEY WORK ARE THE SAME"

CERTIFICATE

This is to certify that this Independent Project entitled AGE RELATED CHANGES IN TRANSIENT EVOKED OTOACOUSTIC EMISSIONS AND RELATIONSHIP OF EARCANAL VOLUME TO AMPLITUDE *is the bonafide work in part fulfilment for the first year* **"MASTER OF SCIENCE (SPEECH AND HEARING)",** *of the student with* **Register Number M9523**

Dr. (Miss) S.NIKAM
DIRECTOR
All India Institute of Speech & Hearing
MYSORE - 570006.

CERTIFICATE

This is to certify that this Independent Project entitled AGE

RELATED CHANGES IN TRANSIENT EVOKED OTOACOUSTIC EMISSIONS AND RELATIONSHIP OF EAR CANAL VOLUME TO AMPLITUDE *has been prepared under my supervision and guidance.*

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MYSORE
MAY, 1996

DECLARATION

I hereby declare this Independent Project entitled AGE RELATED CHANGES IN TRANSIENT EVOKED OTOACOUSTIC EMISSIONS AND RELATIONSHIP OF EAR CANAL VOLUME TO AMPLITUDE is the result of my own study under the guidance of Dr. (Miss) S.NIKAM, Director, All India Institute of Speech & Hearing, Mysore, and has not been submitted earlier at any University for any other Diploma or Degree.

Mysore

MAY, 1996 [REG.NO. M9523]

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Finally, I thank all the subjects of the study. . . . without them this project would not have been possible.

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INTRODUCTION

The existence of otoacoustic emissions was first demonstrated by Kemp (1978) from the Institute of Laryngology and Otology in England. The original reports of Kemp (1978) were greeted with a mixture of excitement and scepticism. His findings have provoked significant research and a greater insight into cochlear mechanisms. Much of the early work was concerned with replicating Kemp s findings. His original observations have since been confirmed and further investigated by a number of researchers across the globe.

Otoacoustic emissions refer to a release of, often, very low intensity audiofrequency energy. However, a few celebrated cases of moderately intense spontaneous emissions, originally described as objective tinnitus, have been reported (Glanville, 1971). They appear to originate from within the cochlea and propagate through the middle ear structures to the external auditory meatus. It is there, with the aid of a sensitive microphone and signal analysis techniques, that they are measured.

Following the discovery of otoacoustic emissions, the traditional view of the cochlea functioning solely as a passive organ, receiving acoustic energy, transducing it into electrical signals and transmitting in one direction only was no longer tenable. Evidence for an active process contributing to the production of otoacoustic emissions was found, first, in the existence of spontaneous emissions, and secondly, in the observation that in certain

instances stimulated emissions contain more energy than that of the stimulus.

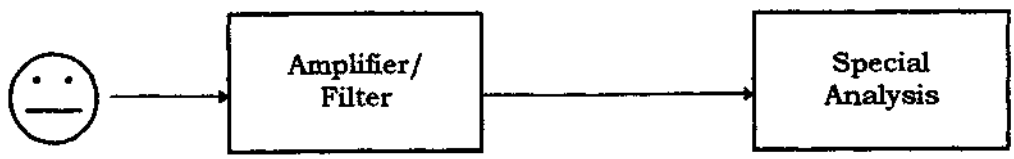
Otoacoustic emissions are now thought to reflect the activity of active biological mechanisms within the cochlea responsible for the exquisite sensitivity, sharp selectivity and wide dynamic range of the normal auditory system. There is strong evidence that these mechanisms are the outer hair cells, at least in the mammalian cochlea (Kiang, Moxon and Levine, 1970; Khanna and Leonard, 1986; Liberman and Dodds, 1984; Sellick, Patuzzi and Johnstone, 1982). Absence of outerhair cells is a condition associated with a lack of otoacoustic emissions (Wilson^ 1980; Khanna and Leonard, 1986), supporting the hypothesis that the outer hair cells are responsible for the generation of otoacoustic emission.

Types of otoacoustic emissions

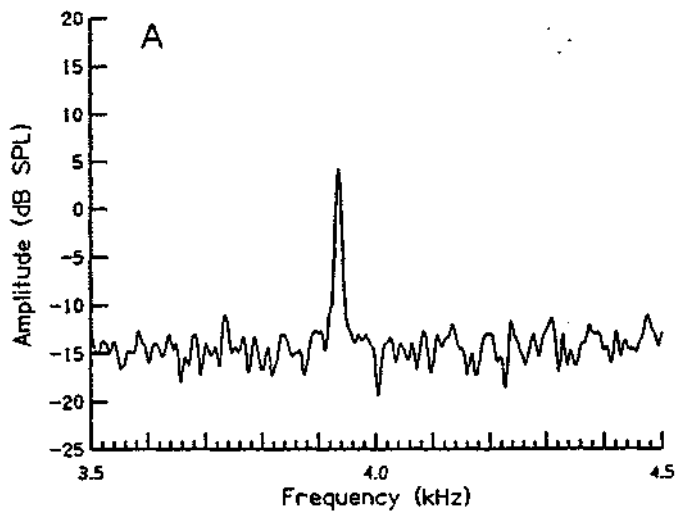
OAEs are manifest in two fundamental forms depending on the conditions in which they occur.

1. **Spontaneous otoacoustic emissions (SOAE):** These occur in the absence of any deliberate stimulation of the ear. These can be detected in nearly 50% of all ears with normal hearing by sealing a sensitive miniature microphone into the external auditory meatus (Kemp, 1979; Zurek, 1981).

Figure 1. Schematic diagram of a representative system for measuring SOAEs from human ears.



Fig



2. Evoked Otoacoustic Emissions (EOAE)

These occur in response to the presentation of acoustic stimuli. They can be detected in nearly all the ears with normal hearing by sealing a

sensitive microphone and miniature speaker into the ear canal (Bonfils et.al.. 1986). EOAEs are of three types.

(i) **Transient-evoked otoacoustic emissions (TEOAE)**

These are frequency-dispersive emissions, occurring in response to a transient acoustic stimulus such as a click or a tone burst. The schemata for measurement is shown in Figure 3.

Figure 3. Schematic diagram of instrumentation to measure TEOAE.

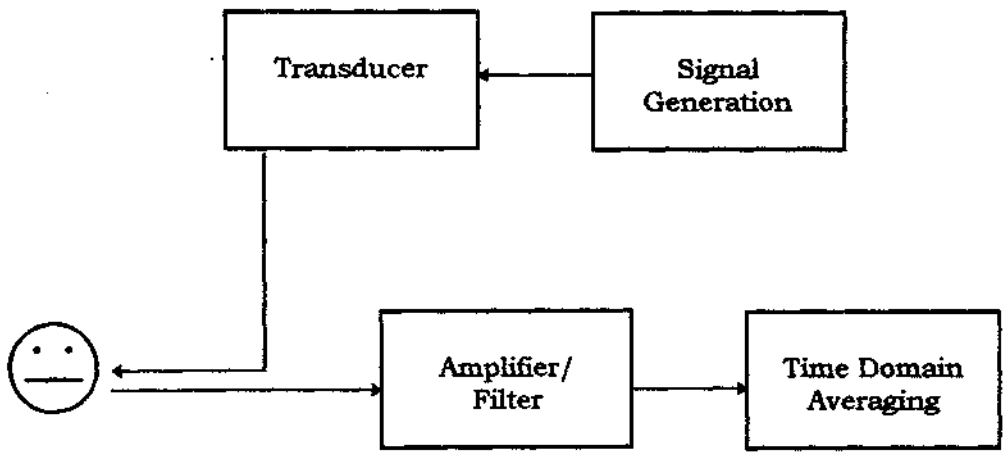
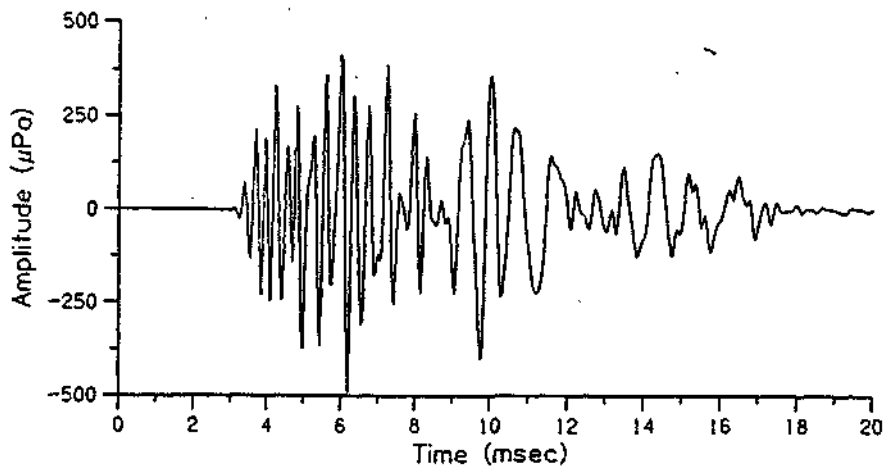


Figure 4. A typical TEOAE Response.



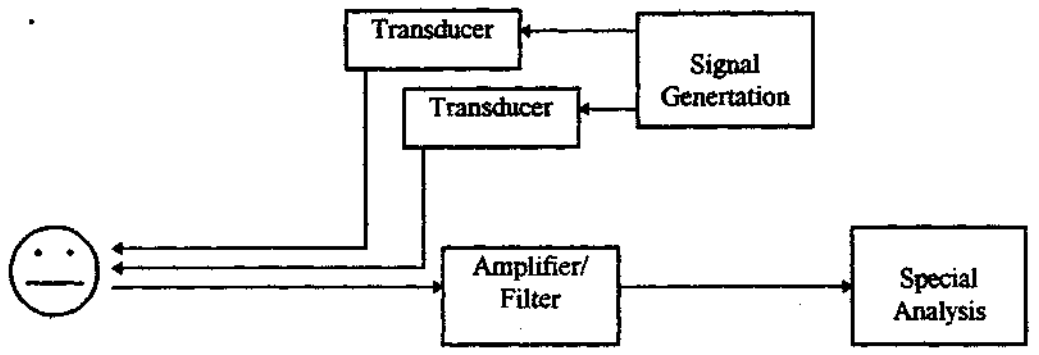
The emission may last upto 25 msec, or longer. The initial component (with a delay of 0-5 msec) is mainly to the impulse response of transducers and that of the outer ear, middle ear and passive parts of the cochlea (Kemp, 1980). The latter part is the TEOAE. In other words, the measured response is determined by the evoking stimulus and the recording parameters as well as the status of the peripheral auditory system.

The important parameters of the emission are group latency, threshold, amplitude of the response and spectral components. With age, there is a reduction in the amplitude and in the higher frequency components (Kemp, 1980)

(ii) **Distortion product otoacoustic emissions (DPOAE)**

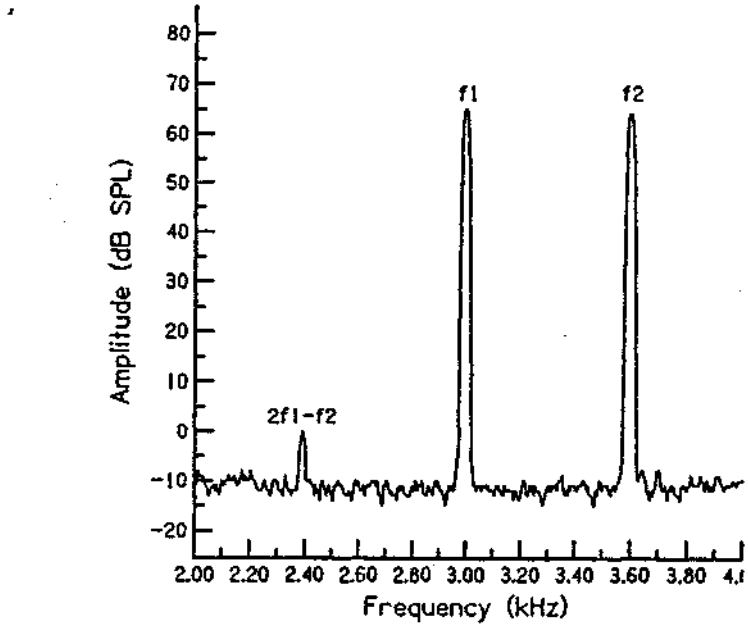
These are generated in response to two continuous pure tones closely separated in frequency by a prescribed difference (in Hz) and presented simultaneously to the ear. Emitted distortion production at intermodulation frequencies such as $f_1 + f_2$ and $2f_1 - f_2$ are measured.

Figure 5. Schematic diagram of instrumentation of measure DPOAE.



The typical distortion product obtained is shown in figure 6. f_1 and f_2 are the stimuli and $2f_1 - f_2$ is the DPOAE generated by the cochlea.

Figure 6. A typical DPOAE pattern, f_1 and f_2 are the stimuli and $2f_1 - f_2$ is the emission generated by the cochlea.



(iii) **Stimulus frequency evoked otoacoustic emission (SFOAE):**

These occur as a synchronous response to a continuous tonal stimulus and are at the same frequency as the stimulus. There is thus a lack of temporal (as in TEOAE) or spectral (as in DPOAE) separation in these emissions. Therefore, sophisticated equipment is required to measure SFOAE.

Figure 7. Schematic diagram for SFOAE measurement.

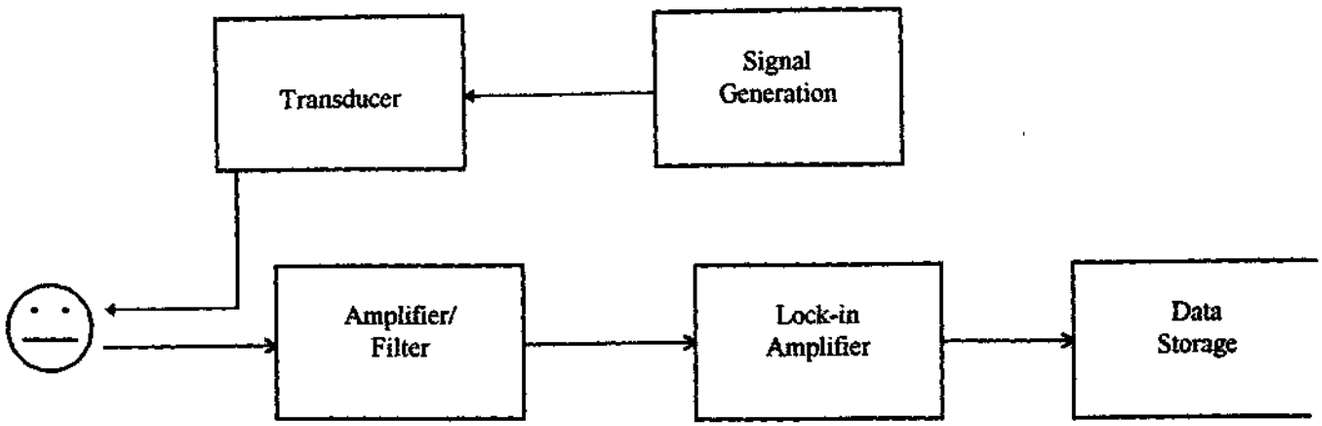
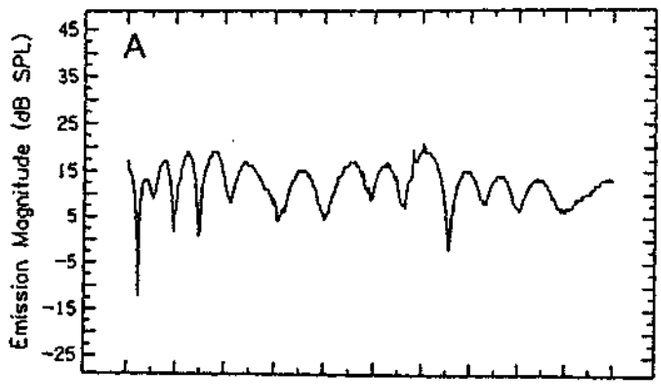


Figure 8: A typical SFOAE response. The stimulus was swept continuously from 1000 - 2000 Hz at 27 dBSPL.



Potential Clinical Applications of TEOAEs.

Most of the research in the past two decades has concentrated on the transient-evoked otoacoustic emissions due to their potential as clinical tools. They are present in 98% of the ears of normal hearing individuals (Bonfils et al, 1990). In addition, they have highly individual and repeatable spectra, suggesting their applicability in long-term monitoring of an individual's cochlear status. This type of monitoring could be applicable for those at risk for cochlear damage, such as patients treated with ototoxic drugs or exposed to high levels of noise at their work place.

It has been reported that even patients with mild cochlear impairment fail to show any emissions. Therefore, a second possible application is the identification of subtle cochlear pathology in patients complaining of hearing difficulty, but with normal puretone thresholds.

A third and as yet little investigated use is as a diagnostic tool, differentiating between cochlear and purely retrocochlear lesions. Lutman (1989) and Prieve et al (1991) reported the presence of evoked emissions in patients with profound hearing loss. They then suggested that the possible pathology might have been retrocochlear. Similarly TEOAEs can be used to differentiate between organic and non-organic hearing losses.

The most promising application so far has been the use of evoked otoacoustic emissions as a screening device for the identification of normal

cochlear status, especially in neonates and infants. TEOAEs have the added advantage of being easy to use, rapid, objective, non-invasive and occurring in almost all normal ears.

TEOAEs are gaining momentum as acceptable clinical tools. Infact, the Rhode Island Hearing Assessment Program (Vohr et al: 1990) has screened over 12,000 infants over five years.

Need for the Study

Before any instrument can be applied clinically, normative values have to be obtained. This is essential because the audiologist requires norms which can be compared with the emission values of patients to decide whether the latter's cochlear status is impaired. However, it is not sufficient to obtain the emission values of the normal hearing population without considering the age of the subjects. Several researchers have observed that the amplitude of transient evoked otoacoustic emissions reduces with age. Therefore, it is important to obtain normative data on TEOAEs at different ages. It is also important to investigate whether the emission amplitude of children and adults differ in a statistically significant manner.

Earlier reports on normative data have speculated on the reason for age related changes. The more robust emissions of children may be due to their healthier cochleae or due to the fact that the external auditory meatus is shorter and straighter, offering

better coupling between the microphone and tympanic membrane. Though it is tempting to conclude that the outer hair cell function deteriorates with age even in children, it is difficult to experimentally verify this in humans. A different way to solve this riddle is to study the relationship between earcanal volume and emission magnitude. A high correlation would indicate that the cochlear contribution to age related changes is minimal. Such a study would contribute immensely to our understanding of the mechanism by which TEOAEs are generated.

Purpose of this Study

The study has a fourfold purpose:

1. To establish norms for children and adults for transient evoked emissions.
2. To study age and gender related differences, if any, in terms of the amplitude and spectral characteristics of the emission.
3. To investigate the correlation between the amplitude of the emission and ear canal volume. This is to test the hypothesis that pediatric ear canals tend, on average, to be smaller than those of adults, causing a greater sound pressure level (SPL) to be developed within the pediatric ear canal, which may be revealed in the higher response amplitudes.
4. To outline the methodology of transient evoked otoacoustic emission measurement and the likely difficulties that one might encounter while using it as a clinical tool.

REVIEW OF LITERATURE

Much of the clinical work related to otoacoustic emissions has focussed on transient evoked emissions. This is mainly because they provide broad-band, cochlea-wide information. In a clinical situation, this means, the most information in the shortest time. Another reason for the widespread work on transient evoked otoacoustic emissions (TEOAEs) is the fact that the earliest commercially available hardware and software (the ILO 88, Otodyramius Ltd., U.K) was optimized to measure click evoked emissions. In addition, the finding that 96-100% of normal hearing individuals have recordable TEOAEs (Probst et al; 1990) gave impetus to their studies.

TEOAEs have taken a mammoth share of the research attention in otoacoustic emissions. They have been studied in a variety of cases. (a) Screening for peripheral auditory system dysfunction in neonates and infants; (b) Monitoring the effects of noxious agents, such as ototoxic drugs and intense noise exposure on the cochlea; (c) Effects of aging; (d) Separating the cochlear and the neural components of sensorineural hearing loss.c. In the assessment of pseudolyspocusis and high frequency cochlear

hearing loss; (f) Assessing fluctuating hearing loss, with or without therapeutic regimens.

In the present survey of literature, the research has been classified under the following heads:

1. Occurrance/prevalence studies
2. Site and mechanism of origin
3. Instrumentation
4. Characteristic features
5. Factors which influence the TEOAE response
6. Bone conduction evoked otacoustic emission
7. Normative findings (a) in adults
   (b) in infants
8. Clinical data.

1. Occurrence of TEOAEs

TEOAEs are measurable in essentially all normal hearing persons with normal middle ears and normal cochleae (Kemp, 1978; Johnson and Elberling, 1982; Granderi, 1985; Alexander and Brown, 1986, Probst, 1986). The existence of emissions in all normal ears makes it a sensitive tool to detect even minor changes

in the hearing status (Norton and Neely, 1987; Bonfils and Piron, 1988).

Though 100% occurrence has been found in adults, it is slightly lower in neonates and infants. Bonfils et al; (1990) measured TEOAEs in neomates ranging from 2 hours to 4 days ninety eight percent of the tested ears had emissions. There was no significant difference in the occurrence between one and four days postpartum, but the occurrence increased within the first 24 hours. Kok, Van Zanten and Brocarr (1992) and Vohr et al (1993) observed a 100% increase in ears with emissions when the ears were first tested 3 to 51 hours after birth and repeated at least 24 hours later. Engdahl et al (1994) observed TEOAEs in 96% of the tested ears of 3 to 4 day old infants. Delaying testing until after the first postnatal day resulted in a 13% higher pass rate.

Giender differences have not been found in the occurrence of TEOAEs (unlike spontaneous emissions, where females are reported to have a higher incidence of emissions (Kok et al; 1982).

Johnsen and Elberling (1982) and Coren and Habestain (1990) reported that the interear variability of TEOAE was the same as intersubject variability. Therefore, TEOAEs of each ear are statistically relatively independent.

2. Site and Mechanism of origin

After otoacoustic emissions were discovered, the traditional view of the cochlea as a purely passive organ, receiving acoustic energy, transducing it into electrical signals and transmitting in one direction was no longer tenable. Evidence for an active process contributing to the production of otoacoustic emissions was found, first, in the exsistence of spontaneous emissions and secondly, on the observation that stimulated emissions contain more energy than the stimulus (Wilson, 1987).

The riddle of the site of origin of otoacoustic emissions was solved with few contradictions. In fact, much before the discovery of otoacoustic emissions, Gold (1948) had suggested the existence of active bio-mechanical cochlear feedback while trying to explain the fine frequency selectivity in the cochlea.

Numerous observations support a cochlear origin of otoacoustic emissions:

(i) The emissions are independent of synaptic transmission and are preneural. When the auditory nerve activity was blocked chemically (Forts, Norton and Rubel, 1990) or physically by severing it (Seigel and Kim, 1982; Martin, Lonsbury - Martin, Probst and Coats, 1987), otoacoustic emissions could be measured though neural responses to sound were absent.

(ii) Otoacoustic emissions are unaffected by stimulus rate, unlike neural responses.

(iii) Evoked otoacoustic emissions are frequency despersive, that is, the higher the emission frequency, the shorter the latency, which is consistent with coding along the basilar membrane. Their amplitudes also grow non-linearly with stimulus level.

(iv) Otoacoustic emission tuning or suppression curves are similar to psychophysical tuning curves.

(v) The emissions are vulnerable to various agents such as ototoxic drugs, intense noise and hypoxia, which are known to affect the cochlea.

(vi) They are absent in frequency regions with cochlear hearing losses exceeding 40 - 50 dBHL and present when hearing sensitivity is normal.

Many authors earlier felt that the existence of otoacoustic emissions was a pathological phenomenon associated with tinnitus (Ruggero et al; 1983, Clerk et al; 1984; Fridge and Kohler, 1986). Ruggero et al (1983) suggested that localized hair cell damage too minor to be detected by conventional audiometric technique mainifested itself as spontaneous and evoked otoacoustic emissions.

Kemp (1986) considered the phenomenon to be due to a leakage of energy from the functional forward travelling wave due to some mechanical perturbation.

Wilson (1980) suggested that the hair cells or supporting cells underwent volume changes when stimulated by sound. This could be due to the movement of ions and gave the cochlea its bidirectional transduction property.

Brownell (1983) demonstrated that the outer hair cells have electromotile properties. Further evidence was provided by Brownell, Bader, Bertrand and de Ripapierre (1985) ; Brownell and Kachar (1986); Brundin et al. (1989). It was observed that the actin and myosin filaments in the stereocilia interact under electrical stimulation and set the outer hair cells to oscillate at audible frequencies. Brownell and Kachar (1986) demonstrated that even after cellular stores of adenosine triphosphate were depleted, the outer hair cells oscillated in response to auditory stimulation. They concluded that the conversion of electrical potential energy to mechanical energy caused the oscillations.

Further evidence came from studies on crossed olivocochlear bundle stimulation. It modulates the hair cell receptor potential and not their membrane resistance (Brown and Nuttan, 1984)

An outer hair cell involvement was also suggested by comparing the otoacoustic emissions produced by mammal and non mammal (avian) vertebrates (Assad, Hacohen and Corey, 1989; Crawford and Fittiplace, 1985). Only stimulated otoacoustic emissions were obtained in non mammals and these were of lower magnitude and frequency. It was suggested that these arose from electrically evoked movements of the stereoclair bundle. Mammalian otoacoustic emissions could occur at frequencies nearly an order of magnitude higher hinting at structural features unique to the mammalian inner ear. The organ of Corti is a mammalian specialization.

To summarize, otoacoustic emissions are generated due to the electromotile properties of the outer hair cells.

3. Instrumentation

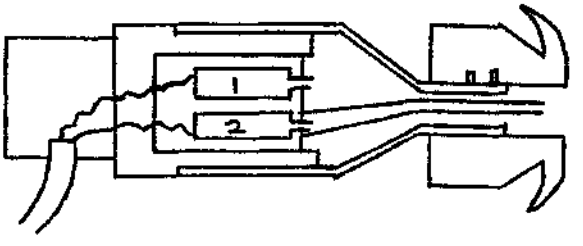
Any system to measure transient evoked otoacoustic emissions must incorporate a means for presenting stimuli, and picking up the emissions through a probe microphone filtering and anlalyzing the wave form and a visual display. This is schematically represented in Figure 3.

The probe microphone contains a sensitive, low noise microphone and a miniature sound source. About 500 to 2000 stimuli are

presented in order to improve the signal-to-noise ratio. The emissions are amplified and then averaged. That is, TEOAEs are obtained using synchronous, time-domain averaging techniques similar to those used to measure auditory evoked potentials. Here the acoustic waveform is averaged, and not the electrical waveform. The ear canal sound pressure is amplified by a factor of 100-10000 and high-pass filtered at 300-400 Hz. It is then digitized at a rate of 40 to 50 kHz.

Kemp, Ryan and Bray (1990) have provided a comprehensive guide to the effective use of otoacoustic emissions. They concluded that a useful way to obtain evoked otoacoustic emissions from a large part of the cochlea simultaneously, including all the byproducts of nonlinearity and intermodulation was to provide a very short but strong broadband **stimulus.** Repetition (about every 20msec) and synchronous averaging allowed the signal-to-noise ratio of the complex otoacoustic emission waveform to be enhanced as required. TEOAEs could then be processed to simultaneously provide information over a wide range of frequencies.

Kemp, Ryan and Bray (1990) emphasized the importance of a good fitting **probe** (Figure 9). This is important to capture high



1 ear phone
2 microphone

frequency (above 3 kHz) emissions and when the stimulus spectrum shows a sharp peak (around 2 kHz) and trough (around 4 Khz) and no low frequency energy below 1 kHz. A large amount of environmental noise is also admitted.

Kemp, Ryan and Bray (1990) recommended click **stimuli** for TEOAE measurement, since these give information over a wide frequency range. They suggested a 85 dBSPL peak with a bandwidth of 5 kHz. They added that the level of stimulator drive voltage should be reduced by a factor of 10 in neonates. This is due to their greatly reduced meatal volume. Tone burst stimuli should be used with nonlinear differential processing.

Keep et al. (1986) observed that there are typically three sources of **noise** that may affect recording: instrumentation (microphone included), environment and the subject. Instrumentation noise has stationary properties and is reduced with synchronous averaging. Environmental noise is reduced using a sound proof cabin and/or with a noise rejection threshold and synchronous averaging. Patient noise is more difficult to treat: it is often of low frequency and is produced by swallowing, breathing, snoring, teeth grinding, heartbeating and cable rub. High amplitude patient noise can be eliminated by the noise

rejection method, while low level noise can be reduced by digital high-pass filtering (Lutman, 1993).

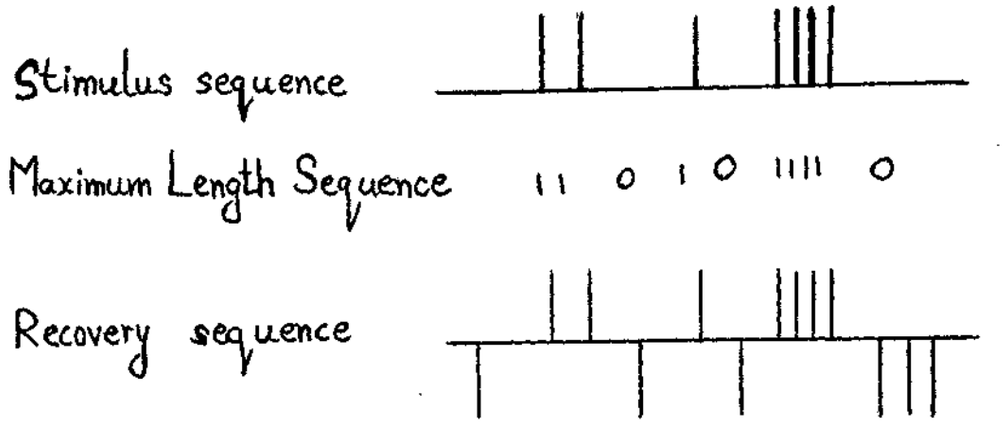
In fact Bray (1994) has been optimistic in claiming that the subject noise problem can be solved and the present state of art in measurement gives valid recording from the typical child patient with characteristic noise.

Recording of the emission can be done with realtime recording or time averaged recording. Wit et al (1981) compared the two and found that these yielded different input-output curves. They suggested time-averaged recording for clinical purposes.

Thornton et al. (1993) noted that a practical problem of using evoked otoacoustic emissions with neonates and young children was that to obtain a good recording, responses must be averaged over a period of a minute or so; the child must be quiet for that length of time. Sometimes 10-15 minutes elapse before such a sample can be obtained since the equipment rejects sweeps that are contaminated by movement artifacts or noise. A solution to this is reducing the test time. They have suggested the use **of maximum length sequences (MLS),** in which a particular sequence of clicks and silences is presented. For example the click

stimulus sequence, MLS and recovery sequence are shown in Figure 11.

Figure 10. MLS Presentation:



They suggested a on-the-fly recovery procedure where each incoming digital sample is multiplied by values in the recovery sequence before averaging. It increased test speed, upto 3000-5000 clicks/second could be presented.

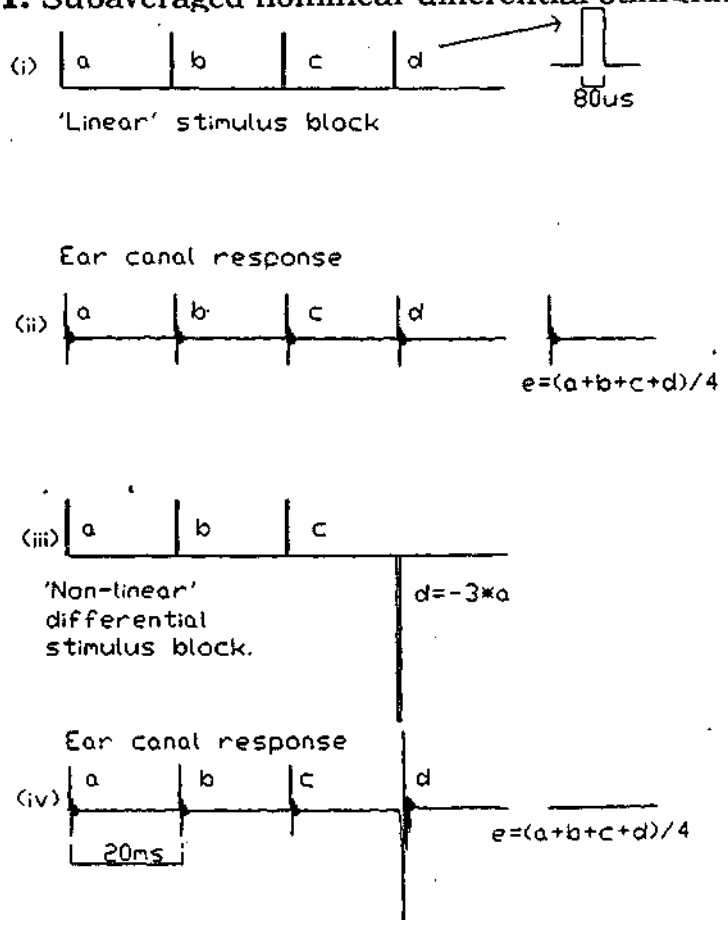
Thornton (1993) reported that maximum length sequence clicks enabled response of a duration greater than the time between stimuli to be recorded and deconvolved to produce an uncontaminated response. Conventional recordings at stimulus rates of 33/S and 50/S were taken together with maximum length sequence recordings at rates upto 840/S. The waveforms correlated very well. Although the emission showed some

adaptation at the highest stimulus rate this did not prevent a significant reduction in test time.

Detection of an emission is commonly based on either visual inspection and cross-correlation between replicate recordings or cross-spectral analysis. These methods overlook the residual noise level. Tognola et al (1995) recognized the need for improving methods of detecting TEOAEs and developed an optimal high-pass filtering technique (600-800 Hz) using digital off-line filtering.

With continuous otoacoustic emission techniques, spectral analysis is used to separate the emission from the stimulus and/or noise. The time delay between the stimulus and the emission also serves this purpose. The nonlinear properties of otoacoustic emissions enable identification and differentiation from the stimulus sound and the middle ear reponse. Kemp, Ryan and Bray (1990) used the subaveraged nonlinear differential stimulus method to control the contammination introduced by ringing of the stimulus in the meatus. Signal-to-noise ratio was enhanced by the method (Figure 11).

Figure 11: Subaveraged nonlinear differential stimulus method.



(i) The ear canal probe is driven by rectangular pulses of 80μs duration, which are all of equal amplitude in the linear mode of operation.

(ii) The train of similar ear canal responses can be averaged to enhance signal-to-noise ratio.

(iii) In the nonlinear differential method, every fourth stimulus is inverted and is three times greater in amplitude. This does not contain any probe or meatal response or noise artifact.

The envelope technique is the least sophisticated to detect emission peaks from the output waveform (Johnsen and Elberling

1982). The recording is double rectified followed by zero phase shift low-pass filtering. Cepstrum analysis is more complex. It is the logarithmic power spectrum of the original time series. The envelope and cepstrum techniques can be used in defining group latencies.

The response magnitude is often the RMS value, though some times the peak-to-peak amplitude is halved (Johnsen and Elberling, 1982).

The currently avilable otoacoustic emission measurement systems are listed below:

1. POEMS Programmable Otoacoustic Emission Measurement Systems. Institute of Hearing Research, Nottingham, U.K.
2. Peters AP 200 OAE Processor. Institute of Audiology, Netherlands.
3. Celester 503. Maseen Electronics, Denmark.
4. Ranger system Etymotic Research, Illinois.
5. VIRTUAL MODEL 330. Portland, Oregon.
6. ILO88 Otodynamics Ltd., U.K.
7. SCOUT DPOAE System; Biologic Systems Corporation, USA.

5. Characteristic Features of TEOABs

TEOAEs are measurable in essentially all normal hearing persons with normal middle ears and normal cochleae (Kemp, 1978; Johnsen and Elberting, 1982; Granderi, 1985; Alexander and Brown, 1986; Probst, 1986; Norton and Neely, 1987; Bonfils and Piron, 1988). Kemp et al (1978) reported that TEOAEs are generally not observed in ears where threshold shifts as small as 30 dBHL have occurred. They used clicks with an intensity of 80 dBSPL. However, TEOAEs may be observed in patients with thresholds upto 50 dBHL if higher intensity stimuli are used (Norton and Stover, 1994).

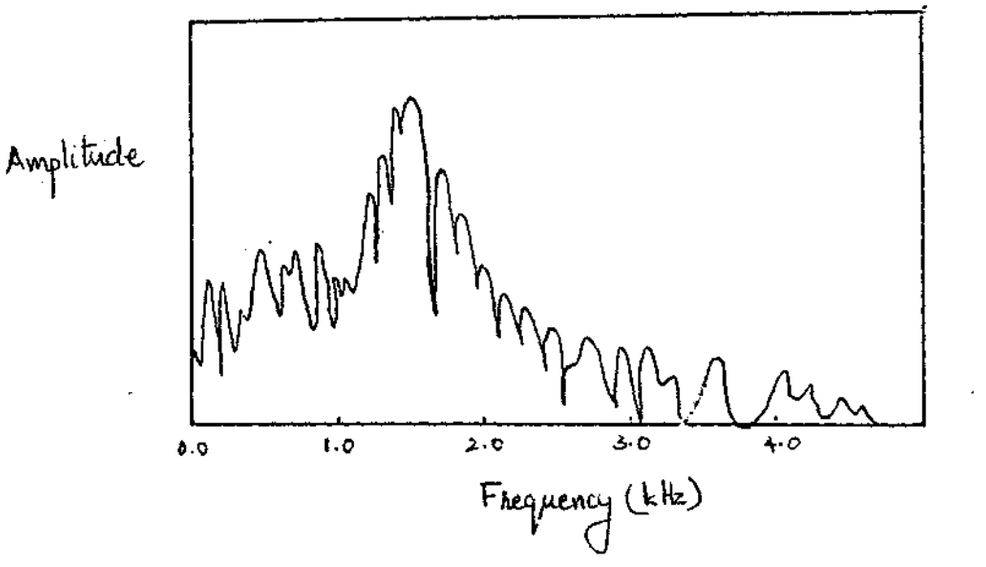
TEOAEs exhibit broad band Spectrum with high component frequencies (Bonfils et al, 1990). They are most frequently measured between 500 Hz and 4000 Hz (Elberling et al., 1985). A typical TEOAE response is shown in Figure 4.

The frequency content of the response is determined by the spectrum of the evoking stimulus. This is true of both the level and bandwidth of the stimulus (Granderi, 1985). The response components have decreasing latencies with increasing stimulus frequencies (Norton and Neely, 1987). One of its most important

characteristics is that it is frequently dispersive, that is, high frequencies emerge sooner than low frequencies. This frequency dispersion is consistent with frequency coding along the basilar membrane, with high frequencies being coded basally whereas low frequencies are coded apically (tonotopic organization).

The emission of a particular frequency originates from the cochlear location tuned to that frequency. However, one should keep in mind that there are other factors which influence the emission, namely, the time window, filtering characteristics and type of stimuli (clicks or tone bursts) [Bonfils, Piron, Uziel and Pujol, 1988). In other words, the measured response is determined by the evoking stimulus, recording parameters and status of the peripheral auditory system. The most determining factor, through, is the spectrum of the evoking stimulus (Granderi, 1985). Since emissions can be evoked at most locations (if not all) in the normal cochlea, the broader the stimulus spectrum, the broader is the emission spectrum (Figure 12)

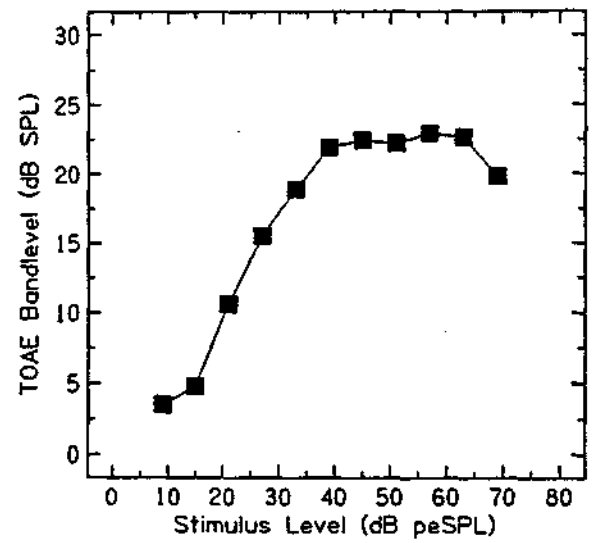
Figure 12: The frequency spectrum of a TEOAE



NOTE: Most of the energy lies between 1-2 KHz.

There is a **threshold** for the TEOAE response. It is the lowest level at which the response waveform and spectrum are judged to be visually different from those of the baseline (no stimulus condition). The amplitude grows nonlinearly as a function of the stimulus level beyond the threshold. After a certain level, it saturates. With further increases in the stimulus level, no further increase in emission amplitude is observed.

Figure 13: The saturating input-output function.



The key parameter of a TEOAE is **group latency** because peak-to-peak amplitude and frequency content are defined only for a specific latency. The response has oscillations of 20-40msec. duration following the stimulus. The original response is relatively weak; so an averaged waveform (time domain) and spectrum (frequency domain) is obtained. The magnitude of the response should be 3dB above the noise floor across all frequencies to be clearly discernible.

The TEOAE begins to drop out at an octave or half octave before the frequency at which there is hearing loss. They are

altered in both the frequency composition and amplitude (Figure 14) when the cochlea is affected by:

a. Noise exposure

b. Acoustic trauma

c. Ototoxic drug administration

d. Surgery

e. Aging

f. Contralateral auditory stimulation.

5. Bone Conduction evoked Otoacoustic emission:

This aspect was not researched until 1988 when Rossi and Solero attempted to study the role of the osscicular chain in the transfer of EOAE to the eardrum. Normal-hearing subjects (N=6) and patients with unilateral otosclerosis (N =4) were studied. In normal-hearing subjects, EOAE by bone conduction stimulation showed the same characteristcs as those evoked by air conduction. The morphological features remained unchanged over a period of four months and their amplitude increased nonlinearly with increasing stimulus intensity. In subjects with unilateral

otosclerosis, no EOAE could be elicited by air-conduction stimulation from the otosclerotic ear before surgery, whereas they could be recorded by bone conduction stimulation. After stapedectomy, EOAE could be obtained by airconduction stimulation too. These results suggest that the ossicular chain is important but not essential in the transfer of the EOAE to the eardrum.

Rossi et al. (1989) noted that on an average, the bone conduction evoked emission was 10 dBHTL greater than the air conduction EOAE threshold.

Collet et al. (1989) agreed with Rossi and Solero (1988) that bone conduction evoked OAEs are comparable to air conduction evoked OAEs. They also concluded that the bone conduction stimulated only the cochlea and was not somatosensory.

Bone conduction evoked OAEs have a potential application in studying EOAEs in patients with a conductive pathology. More research needs to be done in this area.

6. Factors influencing otoacoustic emissions:

In the past decade, extensive research has been done on the factors influencing transient evoked otoacoustic emissions. This is because the TEOAE was recognized as a potentially useful clinical tool and the search was on for the optimum testing conditions. In the present review, the studies have been mentioned in the following sequence:

1. Instrumentation.
2. Stimulus parameters,
   a) Frequency
   b) Intensity
   c) Contralateral stimulation
3. Patient variables
   a) Attention
   b) Posture
   c) Middle ear characteristics.
4. Age and gender effects, and
5. Induced changes.

Now that there is a variety of commercially available **instruments,** the Audiologist must avoid drawing conclusions across studies since no two instruments are replica of each other. In fact some recent studies have concentrated on instrumentation related factors which affect the TEOAE response (Zwicker, 1990; Lutman et al, 1994., Thonton et al., 1994)

Zwicker (1990) described how the acoustical impedence of the probe could influence both the amplitude and waveform of the emission. Lutman et al (1994) corroborated the above study and demonostrated that the acoustic characteristics of the probe could modify the measured response. Further, if the probe acted as a reactive load, oscillation at a particular frequency was seen. This could be confused with a TEOAE response even though no significant oscillatory behavior would occur without the reactive load provided by the probe.

Thornton, Kimm, Kennedy and Cafarelli-Dees (i994) identified the instrumentation related factors which affect the TEOAE response. Of these, the form of stimulus, the characteristics of the microphone, amplifiers and filter were identified as the major ones. Data were collected from 64 neonates

(3 days post partum) on the ILO88 (Otodynamics Ltd., U.K.) and the POEMS system (Institute of Hearing Research, Nothingham, U.K). The ILO 88 TEOAEs consistently had larger high frequency components and higher correlations between repeat recordings.

The influence of various **stimulus** parameters and their efficacy in evoking emissions has been studied (Wit et al., 1979; Zwicker, 1983; Elberling et al., 1985; Norton and Neely, 1987; Thornton, 1993).

Wit et al., (1979) investigated the influence of tone burst **frequency** on the emission amplitude. At the same stimulus level stimuli of higher frequency generated much smaller emissions. They found similar results with filtered clicks (Wit et al., 1981). Elberling (1985) evaluated TEOAEs in response to various tonal stimuli in normal hearing adults (48 to 90 years). They noted that changing the stimulus frequency had only a minor effect on the power spectra. The click was a better stimulus than the tone burst since it gave wider frequency information.

Probst et al. (1986) studied the efficacy of different stimulus types in eliciting otoacoustic emissions. The click (0.1 ms pulse) and toneburst (0.5, 1.0, 1.5, 3.0 Khz) were used. Two patterns

were observed: (i) 18% of the ears showed short broad band click evoked otoacoustic emissions lasting less than 20 ms after stimulus onset.

(ii) 82% of the ears had emissions lasting greater than 20 ms. They found the click to be a better stimulus.

Zwicker (1983) studied the relationship between stimulus **intensity** and the emission. The two were proportional upto 20 dBSL above which the emission level saturated. Wit et al (1979) noted that at low response levels, the relation between stimulus level and response level is approximately linear. Norton and Neely (1987) investigated the relation between toneburst frequency (0.5, 0.75, 1.0, 1.5, 2.0 Khz) and intensity. The saturation curve was noted at all the frequencies. The level at which saturation occurred was lower at higher frequencies. In addition, the spectra of the TEOAE resembled those of the evoking stimuli.

In an attempt to hasten the testing procedures high **repetition rates** have been experimented on (Thornton, 1993; Elberling, 1994). Thornton (1993) noted the effect of varying click repetition rate (338 to 840/S). A higher repetition rate did not

contaminate the response. It also reduced the test time to a few seconds in adults and neonates.

The influence of **contralateral stimulation** on the TEOAE has been widely studied (Collet, 1989; Ruggero, 1983; Ryan, 1991; Collect et al., 1992; Norman, 1993; Williams et al., 1993; Collet et al., 1994; Ryan et al, 1995). Otoacoustic emissions seem to be an ideal tool to study the influence of contralateral auditory stimulation because (i) this may involve the ipsilateral olivocochlear bundle, (ii) One subsystem of the olivocochlear bundle, the medial olivocochlear bundle, synapses directly with the outer hair cells of the organ of Corti, and (iii) the outer hair cells are involved in the genesis of otoacoustic emissions.

The results of various studies on contralateral acoustic stimulation have been summed up by Collet et al (1994).

i) Alteration (mainly a decrease) of emission amplitude,

ii) Alteration of the response spectrum (upward shift in the frequencies), especially with spontaneous emissions,

iii) Alteration of phase,

iv) The effect depends on the intensity of the contralateral stimulus,

v) The effect inversely depends on the intensity of the ipsilateral stimulation,

vi) The frequency specificity of the suppressive effect. The amount of supression increases with the bandwidth of the noise, especially for noises centered around 1-2 Khz. Wide band noise had greater suppressive effects than narrow band noise.

Williams et al., (1993) studied the effects of contralateral stimulation following vestibular neurectomy and found that TEOAEs were unaffected.

It is of utmost importance to identify the **patient-related variables** which may affect the response. To Johnsen et al., (1982) investigated the effect of **posture** (lying down, sitting and standing) in a group of healthy adults (21 to 42 years). They found emission to be unaffected by posture changes. Antonelli and Grandori (1986) studied the influence of the relative position between the head and the body on the responses (sitting down on a chair, and lying onto a reclinable bed at angles of 0, - 20 and -40 degrees, with respect to the horizontal plain). The response amplitudes were reduced when the subjects were held in reclining positions. The latency of the response was greater in the reclining positions and it decreased in the order 0 to 20 to 40 degrees.

Wilson (1980) had also reported a decrease in amplitude and a time shift whenever the subjects position was changed. However, the changes were noted in waveforms only if the new position was held for a time interval of convenient duration.

Meric et al. (1993) compared the influence of **auditory attention** tasks on emission levels and found no significant effect. With repetitive measures, the amplitude was seen to increase during the second and third sessions and linear saturation was seen.

Meric et al. (1993) compared the TEOAE amplitudes of subjects with and without **spontaneous otoacoustic emissions.** There was a significant difference; the subjects with spontaneous emissions had higher TEOAE amplitudes.

Wada (1993) investigated the relationship between TEOAEs and **middle ear** dynamic characteristics. He concluded that TEOAEs are most distinctly detected at the middle ear resonant frequency and in normal subjects whose middle ear mobility is moderate.

Engdahl et al (1994) studied the possible effect of diurnal middle ear pressure variations on the TEOAE amplitude. They observed naturally occurring systematic diurnal variations in pressure. They suggested recording TEOAEs after tympanometry so that the measurement is done at peak acoustic admittance. They added that this is especially important when monitoring small changes in cochlear function by means of TEOAEs. Engdalal et al. (1994) studied intrasubject reproducibility and short term variability of the TEOAE amplitude. The intrasubject variability in terms of minutes and hours was less than ldB and there were no systematic diurnal variations in amplitude.

The TEOAE response changes with **age** (Kemp, 1980; Collet et al., 1990; Kemp, Ryan and Bray, 1990). The response amplitude decreased by 10 dBSL in adults. Neonetal emissions typically extended fairly uniformly from l to 5 kHz whereas adult responses had less power at high frequencies and more below 1 Khz. Robinette (1992) who conducted an exhaustive study on age differences found a statistically significant age effect. The mean amplitude decreased sequentially in dBSPL across age groups from 9.7 dB for the 20 to 29year old group to 7.2 dB for the 60 to 80 year old group.

Robinette (1992) found a statistically significant **gender** effect. The mean TEOAE amplitude was larger for women by 2.8 dB.

Some authors have **induced** changes in the auditory system to study their effects on TEOAEs (Anderson and Kemp, 1979; Johnson et al., 1980; Robinson et al. , 1991 Hauses et al. , 1992; Holtz et al. , 1993).

Anderson and Kemp (1979) reported that **anoxia** and **ototoxic** diureties coursed a reversible depression of otoacoustic emissions in monkeys . Wilson (1980)s tated that a decrease after nembutal overdose occurred in the otoacoustic emissions in cats. Johnsen et al (1980) induced sensorineural hearing loss in two subjects by acetyl salicylate ingestion. The thresholds were elevated and the response pattern was altered.

Hauses et al (1992) noted a reduction in amplitude with **general anaesthesia** in a group of normal hearing adults (18 to 52 years). More subjects showed reduced amplitude with N_2O inhalation (9/10) than with non - N2O anaesthetic inhalation (7/9). The authors attributed this to gas diffusion in the middle ear.

Holtz (1993) noted reduced TEOAE amplitude in the frequency range of 2 to 4 kHz with **noise.**

Robinson (1991) studied the variation of TEOAE response with **ear canal pressure.** The response typically reduced for positive and negative pressures.

7. Normative Data.

The TEOAE characteristics in normal individuals have been studied extensively (Johnsen et al., 1982, 1983, 1988; Weir, 1984; Bray and Kemp, 1987; Bonfils et al., 1988; Collect et al., 1990; Kemp, Ryan and Bray, 1990; Norton and Widen, 1993; Stover, 1993; Engdahl, 1994; Thornton et al., 1994). The studies have covered several age groups (neonates, adults and geriatrics)

(i) Infants Johnsen and Elberling (1983) noted the responses in neonates with normal otoscopic and tympanometric results at 48-96 hours post partum. They observed clear and reproducible responses from all ears at 50 dB SPL. The latencies, amplitude and input-output functions were in the same range as that of adults. But many researchers (Kemp, Ryan and Bray, 1990., Collet et al; 1990., Norton and Widen, 1990; Kok et al, 1992; Engdahl et al., 1994) report significant differences between infants and adults.

Johnseh et al. (1989) studied developmental changes in TEOAEs and observed that the latency and amplitude were unchanged. In some ears the frequency content of the dominant part of the TEOAE was changed. They concluded that postnatal changes occur in the cochlea. Norton and Widen (1990) in a survey of librature, summarized the findings with regard to developmental changes in TEOAEs.

(i) The amplitude reduces with age (1 Bray and Kemp, 1987; Norton and Widen, 1990; Norton, 1993).

(ii) The energy spectrum tends to shift to lower frequencies (Kemp, Ryan and Bray, 1990; Norton and Widen, 1990; Norton, 1993).

(iii) The latency tends to to increase with age (Johnsen and Elberling, 1983)

Norton and Widen (1990) observed that the possible age-related differences influencing this were: interalia, anatomy of the external auditory meatus, middle ear impedance and cochlear function Kemp, Ryan and Bray (1990) , while talking about instrumentation, stated that the main differences are not all attributable to the different probe designs used. The very different meated and tmponomatric configuration of neonate and adult ears may have an influence. They noted that neonate responses were, on the average, stronger than

adult responses by 10 dB. They attributed this to be due to the enhancement of coupling between the tympanic membrane and microphone caused by the very small meatal volume. They added that increased emission activity in infants could not be ruled out. Neonatal emissions were observed to extend fairly uniformly from 1 to 5 kHz. Adults had less high frequency power and more power below 1 kHz, and invariably one or two missing frequency brands or notches.

Kok et al., (1992) attributed the higher amplitude in neonates and infants to the higher prevalence of spontaneous otoacoustic emissions in infants younger than 18 months. They observed that the amplitude reduced in the midfrequency region and high frequency reduction occurred later. They noted that from 3 to 51 hours post partun the occurrence of TEOAEs was 50%, but from 25 hours in the same group, the occurrence was 100%.

Engdahl et al., (1994) conducted a longitudinal study of the reproducibility of TEOAE in the first year of life. On the third and fourth postnatal days, TEOAEs were recorded successfully in 192 ears of 100 full-term neonates. A follow up study was performed on 35 of those infants at the ages of three, six and 12 months. The number of infants presenting decreased linearly with postnatal age. The

amplitude was found to reduce with age. The time required for testing each infant and the number of ears in which TEOAEs could not be identified increased with age. Otomicroscopic changes indicating secretory otitis media were found in all the ears not showing a TEOAE response.

Obstruction of the ear canal, either partial or total, is common in the earliest post natal stage (Cavanangh, 1987), and this is regarded as one of the factors causing failure to record TEOAEs in infants (Chang et al., 1993; Norton, 1993; Vohr et al., 1993). The results of Engdahl's (1994) longitudinal study confirm that the third or fourth day is the best time to record emissions. Further justification for testing at this age is:

(a) All hospital births are theoretically available for testing. Failure to keep appointments increases linearly with time after discharge from the hospital.

(b) The number of unsuccessful TEOAE recordings increased with postnatal age.

(c) The time required for testing was least in the first postnatal week.

(d) Secretory Otitis media is common at six to tweleve months of age.

(e) The middle ear pressure is normal at two to four days. At three monthly intervals during the first year of life negative middle ear pressure was noticed (Tos et al., 1979), and the TEOAE amplitude was reduced in such conditions (Maeve et al., 1992; Engdahl, 1993).

(ii) Adults

Studies on normal adults (Johnsen and Elberling, 1982, Collect et al., 1990; Stover, 1993) indicate that responses can be traced down to or below the psychoacoustic thresholds. Different response patterns have been noted for different ears with the same audiogram configuration . Collect et al (1990) noted a decrease in the presence of emissions and the frequency peak in spectral analysis decreased with age (60 to 83 years) and the emission threshold increased. Stover (1993) demonstrated that none of the age effects (20 to 80 year old subjects)were independcent of hearing sensitivity on any type or parameter.

8. Clinical Data:

Much of the clinical work related to OAEs has focussed on click evoked emissions This is primarily because they provide broad band, cochlea-wide information. In clinical situations, this means the most information in the shortest time. The commercial availability of hardware and software, the ILO88 which is optimized for measuring

transient evoked otoacoustic emissions (TEOAE) is another reason for it being the most widely researched . The clinical data is discussed under the following headings.

1. Psychoacoustic thresholds
2. Audiogram configuration
3. Unilateral hearing loss
4. Conductive pathology
5. Sensorineural hearing loss.
   - (a) Noise induced loss
   - (b) Minieres disease
   - (c) Ototoxicity
   - (d) Otosclesosis
   - (e) Acoustic neuroma
   - (f) Idiopathic sudden deafness
6. Central Auditory disorders
7. Pseudohypocusis
8. Neonatal screening.

Psychocaoustic thresholds

In the presence of hearing loss, TEOAEs have been shown to decrease in incidence as hearing thresholds increase (Kemp, 1978; Kemp et al., 1986; Tanaka, 1987; Bonfils et al., 1988; Stevens, 1988;

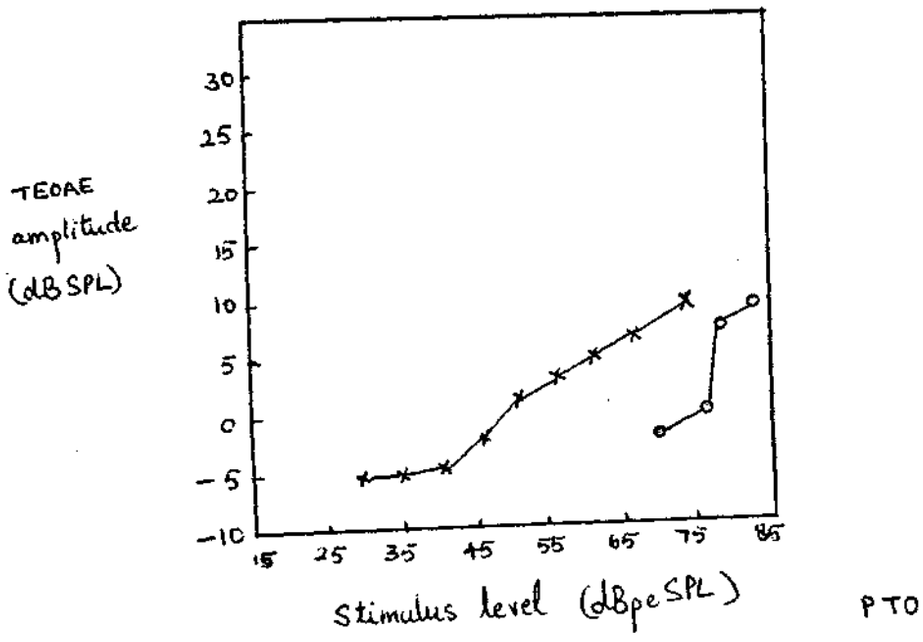
Bonfils and Uziel, 1989; Collet, Gartner and Moulin, 1989, Tanaka, 1990).

Stevens (1988), on an age range of 16 to 85 years, found correlation between psychoacoustical threshold and stimulus level needed to obtain a recordable emission. But the correlation was not significant to make it a useful measure of hearing loss. Tanaka et al. (1989) concluded that TEOAEs are useful to clinically determine the degree of inner ear impairment (age range was 13 to 35 years).

Generally, if the hearing loss exceeds 40-50 dB, an emission cannot be evoked to a transient stimulus. Kemp et al. (1986) reported that the upper limit is 30 dBHL for a 80 dB pSPL, 80 µs click. At higher stimulus levels the limit appears to be 50 dBHL.

Norton and Widen (1990) studied the input-outputfunction (TEOAE amplitude as a function of stimulus level).

Figure 14. Input-output function at two threshold levels



PTO

-X- normal hearing adult with 15 dBHL thresholds

-O- 12 year old child with 40 dBHL thresholds (sensorineural hearing loss)

Stover and Norton (1992) reported good correlation between psychophysical thresholds and TEOAEs for the same stimuli. Responses to suprathreshold stimuli decreased as sensitivity decreased. However, in mild to moderate losses, TEOAEs may appear to be within normal limits for high level stimuli. If one is interested in sensitivity, one may need to measure emission at several stimulus levels and determine the emission threshold. If interested only in the cochlear reserve or integrity, one may use a single high-level stimulus.

There are exceptions to the above results and cases have been reported with normal hearing showing absence of TEOAE and with hearing loss showing TEOAE responses. Lutman et al (1989) reported a eleven year old child with profound sensorineural hearing loss showing TEOAE. They hypothesized the lesion to be retrocoachlear, but did not conduct any confirmatory tests. Prieve et al., (1993) reported findings of TEOAE in a 33 year old woman with severe to

profound sensorineural hearing loss. They assumed a group of surviving outer hair cells in some region of the cochlea with corresponding inner hair cells being intact to be the source of the emission. They also added that the hearing loss may be due to neural damage. They concluded that TEOAEs are a true indicator of site of lesion.

Audiometric frequencies

Collet et al., (1989) reported statistically significant correlation between TEOAE threshold and hearing loss at 1 kHz. They concluded that the presence of TEOAE indicates middle frequency functional integrity of the outer hair cells of the organ of Corti. Absence of TEOAE is hard to interpret. Stover and Norton (1992) studied the relationship between the audiogram and tone-burst evoked OAEs at the octave frequencies at 80 dB pl SPL in a young adult with sensorineural hearing loss. The amplitude was measured. It provided a good snapshot of the audiogram configuration. The click evoked OAE contained energy from 1000 to 3500 Hz.

Where the audiogram shows frequency bands of normal hearing, emissions are usually evoked at those frequencies by a click stimulus. With high-frequency loss emissions are usually obtained

upto the frequency of first loss. There is strong evidence for a high degree of frequency specificity in otoacoustic emissions. Despite the nonlinear saturating characteristics of the emissions with respect to intensity, well separated frequency bands react linearly. Close frequency bands (within one critical band) do interact nonlinearly, as the existence of distortion product emissions shows. (Kemp, Ryan and Bray, 1990).

Lind and Randa (1989) investigated whether a simple technique with a single repeated recording at fixed stimulus intensity could give information enabling differentiation between high frequency and low/medium frequency hearing losses. The latency was measured. They concluded that it can be used to evaluate the presence of low/medium frequency hearing loss exceeding 40 dBHL. Collet et al (1992) reported that the TEOAE spectrum and sensorineural hearing loss are significantly positively correlated. They added that, however, it is not possible to establish an audiogram by spectrum analysis.

Johnson (1993) studied the relationship between audiogram configuration, puretone average and the recurrance of TEOAE. When the audiogram was flat not a single subject with thresholds below 40 dBHL showed absence of TEOAE, and not a single subject who

showed absence of TEOAE had thresholds below 30 dBHL. In sloping audiogram patterns the thresholds at 1 and 2 kHz were considered important for generating TEOAE.

Robinette (1992) studied TEOAE parameters in case of low and high frequency cochlear hearing losses. In pure sensorineural hearing loss there was a linear relationship between the TEOAE threshold and the mean audiometric threshold for frequencies between 1 kHz and 4 kHz (Bonfils et al., 1986). Hence TEOAE thresholds could give information on the audiometric threshold for mid frequencies (1-4 Khz). Nevertheless, this audiometric interest is strongly limited by the disparity of TEOAEs when the puretone threshold for these frequencies is greater than 30 dBHL.

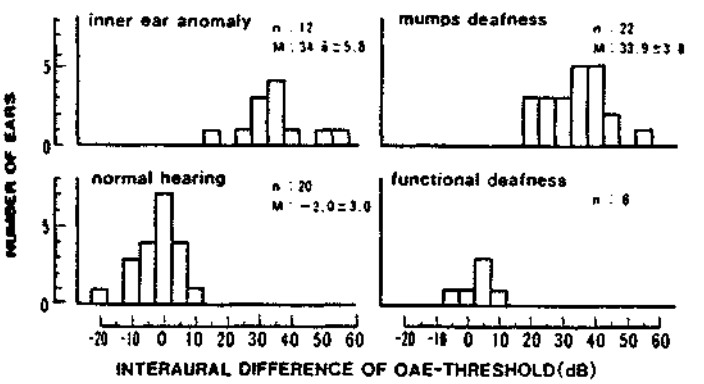
Robinette (1992) found that emission in high frequency cochlear hearing loss generally extend from below 1 Khz to a frequency between the knee of the downward audiometric slope and the highest frequency within the normal hearing range (0-25 dBHL).

However, there is disagreement regarding the "frequency specificity" of the TEOAE. This is because of the transient properties of the stimulus.

Unilateral hearing loss

It is felt by many researchers that TEOAEs can be used to detect unilateral losses, especially in infants where behavioral observation audiometry (BOA) does not indicate a unilateral loss. Tanaka et al (1987) reported the interaural amplitude difference in TEOAE to be a useful indicator in unilateral cochlear pathology rather than the threshold value itself. They also noted a high positive correlation between interaural psychoacoustic threshold differences and those of TEOAE. There were no interaural differences in unilateral functional hearing losses. Tanaka et al., (1990) reported the mean interaural difference to be 35 dBHL in unilateral profound hearing loss . For example, a 10 year old boy who had sudden deafness in the left ear after mumps had a mean audiometric threshold of 11.3 dB in the right ear and 70 dB in the left ear. The OAE threshold was 10 dBnHL in the right ear and 50 dBuHL in the left ear. Similar interaural differences were found in unilateral cases of Miniere's disease and cerebellopontine angle tumors. Tanaka and Suzuki (1990) illustrate this in the histograms (Figure 15).

Figure 15: Histograms showing interaural difference in TEOAE threshold in subjects with unilateral hearing loss. A. inner ear anomaly; B. mumps deafness; C.normal hearing; and D. functional hearing loss (Tanaka and Suzuki, 1990).



Conductive hearing loss

In cases of middle ear pathology TEOAEs may not be measurable because they are not effectively transmitted by the middle ear. Generally, if the air-bone gap for puretone thresholds exceeds 30-35 dB, TEOAEs cannot be measured (Norton and Stover, 1994). However, emissions can be measured in ears with patent pressure equalization tubes if the air-bone gap is small and the middle ear cavity is healthy.

In Ostosclerosis TEOAEs have never been observed when the mean audiometric thresholds for 0.5 Khz and 1 khz were greater than 30 dBHL. Bonfils and Trotoun (1989) observed that after stapes

surgery, TEOAEs appeared in cases whose audiometric thresholds were less than 30 dBHL.

In serous otitis media, TEOAEs were recorded only when audiometric thresholds were lower than 30-35 dBHL (Bonfils, Uziel and Nancy, 1988). When TEOAEs were recordable, the emission spectrum gave additional information, that is only high frequencies (above 1.5 - 2 kHz) were present. This frequency pattern seemed specific to enstachain tube dysfunction.

Wada (1993) studied the influence of middle ear dynamic characteristics on TEOAEs. Those included mobility of the middle ear, Eustachain tube patency and the middle ear resonant frequency. TEOAEs were detected most distinctly at the middle ear resonant frequency and in normal hearing subjects whose middle ear mobility was moderate. Kemp, Ryan and Bray (1990) stressed the importance of ruling out middle ear pressure or fluid changes before failing a TEOAE screen. They reported that pressure imbalance reduced emission energy below about 2 kHz and possibly increased it above 3 kHz. This could be replicated by artifically controlling meatal pressures.

Engdahl et al (1994) studied the reproducibility and short term variability of TEOAE (intra subject) in terms of amplitude and the possible effect of diurnal middle ear pressure variations. There were no systematic diurnal variations noted. However, TEOAE amplitudes were observed to vary with naturally occurring middle ear pressure changes. Hence they suggested combining tympanometry with TEOAE recording, thus making it possible to make measurements at peak acoustic admittance. This is especially important when monitoring small changes in cochlear function by means of TEOAEs.

In testing newborns debris including wax in the external ear canal can reduce and block TEOAEs (Chang, Vohr, Norton and Lekas, 1993). Collapsed ear canals can also interfere with emission measurements (Norton and Stover, 1994).

Sensorineural hearing loss

Ever since the cochlea was identified as the source of OAEs, it is considered a reliable predictor of sensorineural hearing loss, especially cochlear pathology (Kemp, 1978; Kemp et al., 1980; Harris et al., 1982; Stover, 1982; Bonfils et al., 1989 Norton et al., 1990).

Bonfils et al (1989) studied the clinical applicability of TEOAEs as objective indicators of cochlear pathology in the age range of

fourteen to seventy four years. They found it to be a reliable technique for the objective study of normal micromechanical activity within the cochlea and for the detection of subtle changes in cochlear disease.

Norton et al. (1990) opine that TEOAEs, can be used (a) as a screening tool for cochlear dysfunction across individuals, (b) to monitor changes over time in cochlear status within and ear. Tanaka et al., (1990) found the sensitivity of the test to be 96% for cochlear losses. Here the pathology was confirmed by other tests of cochlear function.

The presence of TEOAEs when the person has sensorineural hearing loss can be an indication of retrocochlear pathology (Lutman et al., 1989, Prieve et al., 1991). Bonfils et al (1988) reported that TEOAEs could be used clinically for (a) objective assessment of sensorineural hearing loss, (b) diagnosis of retrocochlear pathology.

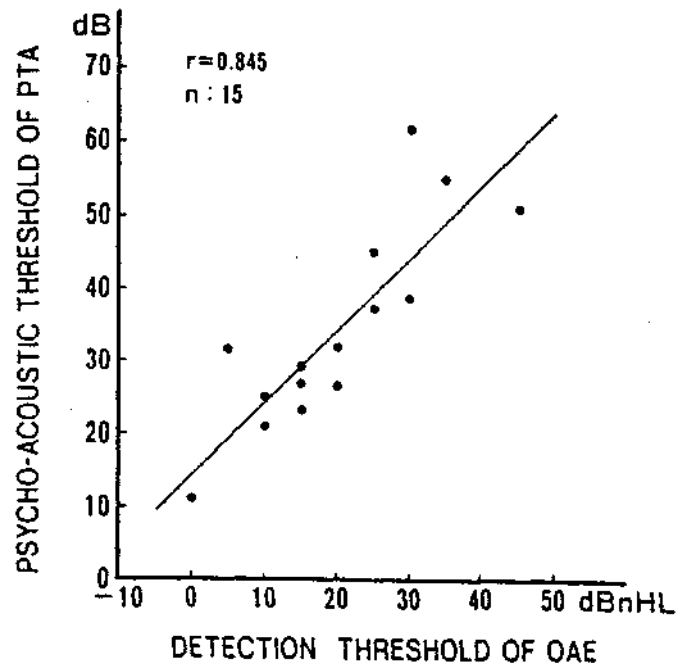
Noise induced hearing loss

Animal studies where the animals have been tested for emissions after prolonged exposure to noise have revealed absent or diminished TEOAEs (Kossl et al., 1985; Homer et al., 1985; Lenior et al., 1987).

Kemp (1982) measured TEOAEs in young adults after exposure of 80 dBSPL broad band noise for 1 hour. The amplitude of TEOAE response was inversely related to the degree of temporary threshold shift (TTSJ and increase non liniearly with time post-exposure. The decreases in amplitude with noise exposure were consistent (Norton and Hayes, 1990).

Tanaka et al. (1990) reported TEOAEs to be useful in predicting noise susceptibility. The scatterplot shows the relationship between psychoacoustic threshold and OAE threshold in 15 ears of noise-induced hearing damage (Figure 16).

Figure 16. Scatterplot of relation between psychoacoustic threshold and detection threshold of TEOAE)



The correlation was found to be 0.85. In ears with notch type hearing loss that represented the initial stages of noise-induced hearing damage, otoacoustic emissions were detectable at one or two octaves lower that of the usual dip frequency. Thus, the otoacoustic emission threshold was not a crucial parameter. But the duration of otoacoustic emission within 20 msec. after stimulus onset was prolonged according to the increase of stimulus intensity and this appeared more prominent in the ears with dip type hearing loss.

Miniere's disease

The findings regarding emissions in Mimere's disease are contradictory. [Elevated emission thresholds have long been reported (Johnsen and Elberling, 1982; Rossi, Solero and Rolando, 1989] Bonfils et al. (1988) suggested that TEOAEs could be used clinically for staging Miniere's disease by recording glycerol induced changes. Norris et al. (1990) reported absence of emissions in endolymphatic hydrops induced in chinchillas. Rupture of the Reissner's membrane caused the emissions to disappear. But this may be due to trauma caused to the organ of Corti while inducing membrane rupture. Tanaka, Suzuki and Tsueno (1990) reported improvement in TEOAE thresholds with glycerol administration.

Harris et al. (1992) studied patients with Miniere's disease in the age range of twenty to seventy years. Clicks and tonebursts were used to elicit emissions. With clicks, emissions were recorded in twentysix out of th;irtyone subjects in the affected ear, and in twentynine out of thirty one in the unaffected ear. With tone bursts as stimuli, emissions were recorded in twenty eight out of thirty one subjects in the affected ear and thirty out of thirty one in the unaffected ear. This shows that TEOAE responses are not affected in Miniere's disease and tone bursts are more sensitive to the emissions.

Acoustic Neuroma:

Tanaka and Suzuki (1990) found interaural amplitude differences in a case of unilateral cerebellopontine angle tumour. Martin Robinette (1992) evaluated 61 acoustic neuroma patients pre-operatively and measured TEOAEs in 31 of them. For 19 of these patients, TEOAEs were expected because hearing thresholds for most frequencies were within the normal range. But 12 patients who had mild to moderate hearing losses showed TEOAEs. So TEOAEs were positive suggesting retrocochlear lesions only for 20% of these patients.

The use of TEOAE tests with auditary nerve tumor patients can assist in differentiating cochlear versus neural pathology pre-operatively and post-operatively. For example, if TEOAEs present pre-operatively are absent port-operatively, it can be inferred that the surgery may have caused cochlear damage.

Ototoxicity/ Drug-induced changes

Johnsen and Elberling (1980) observed elevated thresholds and altered response patterns when a sensorineural hearing loss was induced by ingesting acetyl salicylate. The TEOAEs reappeared after the drug-intake was stopped and hearing had returned to normal.

Johnsen and Elberling (1982) Mc. Fadden and Plattsier (1984) and Long et al., (1988) noted that large dloses of aspirin or a long-term treatment caused SOAEs to disappear and TEOAEs to be diminished. The TEOAEs persisted longer and recovered sooner.

Kossl and Vater (1985) administered nembulal and halothane to the bat and observed a decrease of both the amplitude and frequency of OAEs. Anderson and Kemp (1975) studied monkeys and found that minutes after injecting ethacrynic acid, the OAE amplitude decreased by 15 dB.

However, the clinical applicability of TEOAEs to detect otootoxicity seems to be limited by the frequency range of TEOAEs. In ototoxicity the inner ear damage predominantly affects the basal turn of the cochlea (that is, high frequencies). As TEOAEs are optimally suited to observe mid-frequency activity of the cochleal (1-4 kHz), they do not seem adapted to an early detection of ototoxicity, which could be achieved with more efficiency by high frequency audiometry.

Idiopathic Sudden Deafness

Temporal bone findings of sudden idiopathic deafness revealed hair cell degeneration in the cochlea of vascular origin (Schuknecht, 1974; Gussen, 1976). Tanaka, Suzuki and Tsueno (1990) demonstrated that the distribution and mean value of interaural differences was similar to that in inner ear anomalies or mumps deafness. Therefore (they concluded) most cases of sudden deafness may be caused by inner ear impairment.

6. Central Auditory Disorders

This aspect has not be researched extensively, mainly since otoacoustic emissions are known to originate from the cochlea and the role of the central nervous system is presumed to be negligible. Bonfils et al. (1990) observed that evoked otoacoustic emissions are

always present in infants with lesions involving the central nervous system. Lafreneire et al. (1991) attempted to characterize the emissions from neonatal and infant subjects at risk for hearing loss. Transient evoked otoacoustic emissions and distortion product otoacoustic emissions were of low amplitude or absent in subjects with suspected central hearing loss.

The feasibility of using TEOAEs as an in-patient check of hearing status in children recovering from bacterial meaningitis in the age range of 3 to 16 years was studied by Fornum et al (1993). They found a 100% specificity - all those who failed the test had subsequent hearing loss.

7. Pseduohypocusis

TEOAEs are valuable in identifying functional hearing loss. Robinette (1992) found TEOAE assessment useful in the 12 cases that he studied. He described a 41-year old man with a bilateral severe sensorineural hearing loss speech reception. Thresholds were 35 dB bilaterally and word recognition scores were normal. His behavioural responses to stimuli were delayed. TEOAEs were present bilaterally and following careful reconstruction, normal behavioural thresholds were obtained.

Tanaka and Suzuki (1990) illustrate the interaural differences in TEOAEs in cases of functional unilateral hearing loss (Figure 17).

8. Neonatal Screening

The population incidence of severe and profound congenital sensorineural hearing loss is between one and two per thousand (Pecham, 1980; Davis and Wood, 1992). The importance of early detection of affected infants and their habilitation by six months of age is widely acknowledged. The speech intelligibility of infants fitted with hearing aids before the age of six months has been reported to be superior to that of infants fitted after this age (Markides, 1986) and oral language production abilities are improved by early intervention (Raun-Kahawan and Davis, 1992). There is also evidence that a lack of auditory experience during early infancy can result in a permanent loss of hearing sensitivity (Fisch, 1990).

For these reasons, there is urgent need to consider screening of all infants for auditory impairment. This screen needs to be of high specificity to avoid the unnecessary parental anxiety and work load on audiology services created by false-positive results. The screen

also needs to be simple to administer to a large number of neonates, preferably under ward conditions, if high uptake is to be ensured.

TEOAEs are extremely robust in normal hearing, full term new born babies. By contrast, a new born who has a moderately severe sensorineural hearing loss would not show measurable TEOAEs.

Results from some large clinical trials (Maxon, Norton, White and Brehens; 1991; Volur et al. 1994) indicate that transient OAEs can be a rapid, sensitive tool for detecting hearing loss in both full-term and at risk new borns.

The use of otoacoustic emissions for screening hearing function in neonates and infants has been suggested (White et al, 1980; Kemp, et al, 1981; Tanaka et al, 1986; 1989; Bonfils et al, 1990; Stevens et al 1990; Dolhen et al; 1991; Baldwin, 1992; Fortum et al, 1993; Meredith et al, 1994; Engdahl et al; 1994). The problems associated with behavioural tests, high risk registers and auditory brainstem response (ABR) testing give impetus to the research on the feasibility of otoacoustic emissions for infant screening.

Behavioural tests have low coverage, low specificity and low sensitivity for hearing loss (Davis, 1992). In addition, many mild unilateral and fluctuating hearing losses are not detected.

Davis et al (1995) report a specificity of only 64% with high risk registers. Mank and Behrans (1993) found the specificity to be less than 50%.

The frequently cited problems regarding ABR testing are:(i) the time required for automatic recording is approximately twenty minutes, added to this is the time required to obtain consent from the parents and shift the baby to a test room; (ii) fifty present of the mothers refused consent either due to concern about the implications of failing the screen or anxiety about the use of scalp electrodes (Hunter et al, 1994).

A fews researchers feel OAEs can be a substitute to ABR (Tanka, et al, 1982, Kemp et al, 1992, Stevens et al, 1990). However, other researchers feel OAE screeming should be combined with other tests like ABR and behaviour observation audiometry (Stevens et al, 1990; Uzil and Piron, 1991, White et al, 1993 ; Kennedy et al, 1994).

Tanaka et al (1989) assessed the diagnostic value of TEOAE in children ranging from six years to fifteen years. The TEOAE thresholds obtained were as follows:

Normal hearing	-	5.9 dBnHL
Mild hearing loss	(25 - 40 dBHL)	6.2 dBnHL

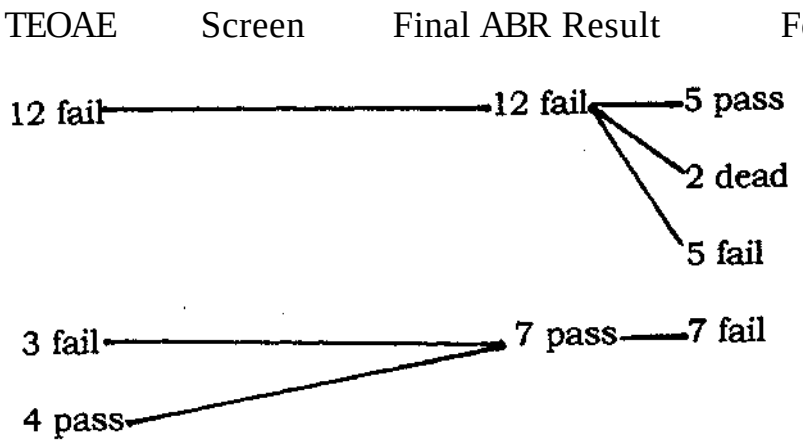
Severe hearing loss (70 - 90 dBHL) 37.2 dBuHL.

They concluded that TEOAEs do serve as valuable diagnostic indicators of inner ear function in children.

Stevens et al (1990) studied the possibility of using TEOAE to identify hearing-impaired neonates. The proportion of NICU (neonatal intensive care unit) infants producing recordable TEOAEs was 80% while 98% of the normal infants produced recordable emissions. The selectivity of TEOAE to ABR was 84% and the sensitivity when compared to ABR was 93% upto 3 months of age. They also found the testing time to be shorter. They provide the following comparison.

	EOAE	ABR
Mean test time	12.1 minutes	21.0 minutes
Recording conditions		
(i) Maximum test room noise	30dBA	
(ii) State of infant	Long periods with absence of noise above 30 dBA	40 dBA Long periods with absence of muscle activity.
Proportion successfully tested		
(i) 0-6 weeks post due date	100%	
(ii) 7-12 weeks	92.3%	
(iiil) Above 12 weeks	71.4%	

Comparison of ABR results upto 3 months of age with follow-up tests results from 8 months of age shows that some of those who initially failed ABR passed it at a later stage. This lack of specificity was not seen in TEOAEs.



TEOAE test shows high sensitivity to ABR upto 3 months of age, the sensitivity to follow-up results is much lower at 55% for infants failing followup at 30 dBHL and 67% for infants failing follow-up at 40 dBHL. This is not surprising considering the separation in time and the possible development of hearing impairment after birth..

Combining TEOAEs with ABR to create a two-stage screening test of high specificity has been suggested by many researchers in light of the promises that it holds in detecting hearing loss at birth (Bonfils et al, 1990; Kennedy et al, 1990; Stevens et al, 1990; Mziel and Piron, 1991, White et al, 1993). Stevens et al (1993) add that

TEOAE should be the initial method to screen for hearing loss: test failures should be followed by ABR.

Hemter et al (1994) studied the feasibility of OAE detection followed by ABR as a universal neonatal screening test for hearing impairment. The procedure was feasible in 95% of the babies born in a hospital. It required two testers working in 12 hour shifts to screen all babies born. The specificity was 70%; only 8.5% of the babies (who had bilateral failures) required ABR confirmation. In an earlier study it was 6%. The high OAE failure was attributed to the young age of the neonates (fluid in the middle ear, fluid or vernix in the earcanal; Northrop et al, 1986) ,or/and developmental phenomenon in the cochlea in the first days of postnatal life (Thornton et al, 1993) or high noise levels (Kemp, 1982).

Recently the emphasis has shifted to longitudinal data to determine specificity and clinical applicability. The Rhode Island hearing assessment showed promising results (Volur et al, 1990) of the TEOAE as a screening tool after screening over 12,000 infants over five years. Meredith et al (1994) screened high-risk neonates for five years between 1988 and 1993. They found an overall failure rate of 27.7%. The failure rate for low birth weight babies was significantly

higher (45.9%) than for babies in other at-risk categories. Sensitivity-was 100% and the specificity was 72.3% (There was a high false-positive rate. This higher failure rate in premature infants was earlier noted by Stevens (1990) and Uziel et al (1991). It may be due to flattened heads and distorted ear canal shape. High incidence of middle ear effusion in babies who have been intubrated may also be a reason.

To summarize, TEOAEs are now widely accepted as objective, efficient and noninvasive method for screening auditory function in infants.

METHODOLOGY

The study was c o n d u c t e d the frame work of the survey metnod and a sample was chosen. The methodology is described under the following headings.

1. Subjects
2. Instruments
3. Test environment
4. Calibration
5. Procedure
6. Statistics.

1. Subjects

The subjects were volunteers from neighbouring schools, and postgraduate students and staff of the All India Institute of Speech and Hearing. The transient evoked otoacoustic emissions were measured if the subjects had hearing within the normal range (Pure tone average better than 25 dBHL; reference ANSI 1969). Further, they had to have speech discrimination scores not less than80% and normal tympanograms ('A' Type).

In the case of young children for whom pure tone testing could not be done, the acoustic reflex threshold was taken as a measure of hearing sensitivity (acoustic reflex threshold within 95 dBSPL).

In addition, all the subjects were carefully screened in a verbal interview for negative history of otological disease, noise exposure, otoxic drug use, metabolic diseases associated with hearing loss and family history of hearing impairment. The risk factors for hearing impairment as defined by the Joint Committee on Infant Hearing (1994) were recorded, if present (Appendix I).

Fiftythree subjects (24 males, 29 females) whose age ranged from 50 days to 28 years (mean age =10.86 years) were selected. They were arranged into seven age groups as follows: below 3.0 years (N=5; Mean =1.5 years, Mode = 2 years), 3.0 to 5.11 years (N = 9 ; Mean, 4.2 years, Mode = 5 years),6.01 to 8.11 years (N = 5; Mean = 8.2 years; Mode = 8 years), 9.0 toll. 11 years (N = 7, Mean =10.years ; Mode= 10 years), 12.0 tol4.11 years (N = 5; Mean = 12.8 years, Mode = 12.5 years), 15.0 to 17.11 years (N = 5, Mean = 16.6 years, Mode = 17 years) and above 18.0 years(N = 17 ; Mean = 22.4 years, Mode = 21 years). The details are compiled in Table - 1.

Table - 1. Age and Sex Distribution of the sample.

Group	Range	Age (Years)		Sex distribution		Number of ears tested.
		Mean & (Standard de viation	Mode	Males	Females	
	0-2.11	1.5 (0.72)	2	1	4	7
II	3.0-5.11	4.2 (0.87)	5	2	7	10
III	6.0-8.11	8.2 (0.98)	8	3	2	9
IV	9.0-11.1	10.3 (0.46)	10	4	3	10
V	12.0-14.11	12.8 (0.75)	12.5	3	2	10
VI	15.0-17.11	16.6 (0.49)	17	2	3	10
VII	18 and above	22.4 (2.48)	21	9	8	30

2. **Instruments**

Four instruments were used in the study; (a) to measure transient evoked otoacoustic emissions, (b) to measure the physical volume of the ear canal and acoustic reflexes, (c) to screen hearing sensitivity for puretones, and (d) to measure the ambient noise level of the test room.

(a) The transient evoked otoacoustic emissions were measured using a Bio-logic Scout Plus System (Software version 1.22) in standard default operational mode. The stimuli were 100 us rec-

tangular pulses with a presentation rate of 4/second. The stimulus level varied from 64 to 110 mPa with a mean of 70 mPa. Eight samples per buffer and 128 sweep sets were recorded. The spectrum level range was 40 dBpSPL and the spectrum frequency range was 0 to 8 kHz. The stimuli were presented in blocks of four where three stimuli of one polarity were added to a fourth stimulus of opposite polarity three times the amplitude so that the stimulus artifact was minimized.

The ER-IOC probe with appropriate eartip size was used. Each response was bandpass filtered from 5656.900 Hz. (low pass filter frequency) to 2000.000 Hz (High pass filter frequency) in order to reject artefacts. The artefact rejection threshold was 0.977 mPa. The response was sampled from 5.000 to 19.000 mPa. The responses were stored after completion of 256 averages.

(b) A microprocessor based automatic immittance meter with a visual display (Grason-Stadler GS1-33, Version 2 Middle Ear Analyser) was used to obtain the physical volume and acoustic reflex thresholds.

(c) A two-channel diagnostic audiometer (Grason - Stadler GSI-10 Audiometer) with TDH-49 earphones mounted on supra-aural

MX41AR ear cushions was used to screen hearing sensitivity for puretones.

(d) A type O precision sound level meter (Bruel and Kjaer 2209) connected to a one inch microphone (Bruel and Kjaer 4145) was used to measure the ambient noise level.

3. Environment

All measurements were made in air conditioned sound treated rooms where the ambient noise level did not exceed 39.25 dBSPL. This is within permissible limits according to ANSI 1969. The noise level was measured with a sound level meter (Bruel and Kjaer 2209) to which a one-inch microphone (Bruel and Kjaer 4415) was attached. The sound level meter was mounted on a tripod and set in the 'C' weighting network and 'slow' mode. It was oriented in four different directions and the measurements were made. The four values were averaged to arrive at the ambient noise level.

Calibration

In the instrument used to measure transient evoked otoacoustic emissions (Bio-logic Scout Plus System), calibration was the second phase of testing. The attenuator was automatically adjusted to achieve the target output level.

The automatic immittance meter (Grason Stadler Middle Ear Analyzer 33, Version 2) was calibrated according to the procedure specified in the instruction manual.

The audiometer (Grason Stadler GS 10) was calibrated according to the standard procedure as given in the Instruction Manual and ISO standards for frequency output, attenuator output and attenuator linearity.

To calibrate the sound level meter it was set to the linear mode attached to a one-inch microphone (Bruel and Kjaer 4144). A piston phone (Bruel and Kjaer 4220) with an output of 124 dBSPL at 250 Hz was placed on the sound level meter. The needle deflection was adjusted to match the output of the piston phone.

5. Procedure:

Initially the subjects were screened in a verbal interview for a history of otological disease, noise exposure, ototoxic drug use, metabolic diseases associated with hearing loss and a family history of hearing impairment. If any risk factors for hearing impairment were present as defined by the Joint Committee on Infant Hearing (1994), these were recorded.

Next, the subjects were screened for hearing loss. They were instructed to raise their index finger in response to auditory stimuli presented through earphones. The modified Hughson West lake procedure was used and thresholds were obtained at octave frequencies 0.25 to 8.00 kHz. If the thresholds in any one ear exceeded 25 dBHL, the subject was not taken up for further testing. Ten monosyllables were presented at 40 dBSL. The subjects were instructed to repeat these and the responses were scored in percentage. The subject was not tested for otoacoustic emissions if he scored poorer than 80%.

In young subjects (under 3 years) the acoustic reflex threshold was measured at 1.0 kHz. Subjects whose ipsilateral acoustic reflex thresholds did not exceed 95 dBHL were chosen for study. Behavioral observation audiometry was used for the youngest subject, who was 50 days old.

The transient evoked otoacoustic emission measurement was carried out after the subject was seated comfortably and instructed not to move or talk during the test. The measurement was done in three phases:

(i) Checkfit: A transient broad frequency stimulus was presented to the ear and the measured response was displayed. The fit of the

probe in the ear canal was adjusted to obtain the flattest possible spectrum.

(ii) Calibration, which has been described earlier.

(iii) Measurement. The stimuli (whose parameters have been described under instrumentation) were presented automatically. The stimulus spectrum, the response over time and the response spectrum were displayed on the screen during the test. The responses were stored after the test was completed.

The automatic immittance meter was set in the 'Tymp' mode to obtain the ear canal volume. The pressure was automatically varied in a hermetically sealed ear canal by means of a pressure transducer located within the probe box. The volume in ml. appeared on the display.

6. Statistics

The mean, standard deviation and range were calculated for each age group on each parameter (echo amplitude, background noise level, testing time, reproducibility and ear canal volume). The Mann-Whitney U test was used to calculate significance of the difference between means of groups I to VI. The 't' test was used to calculate significance of the difference between the means of adults

and children. The correlation between earcanal volume and echo amplitude was calculated using the linear correlation method for each group. The Karl-Pearson's product moment method was used to calculate the correlation for the whole group.

RESULTS

The purpose of the present study was to investigate age-related changes in transient-evoked otoacoustic emissions in terms of the amplitude, noise floor, response waveform and reproducibility. The study also aimed at correlating the external auditory meatus volume with the magnitude of the response. The purpose of studying the dependence between the two was to investigate the extent of contribution, if any that ear canal volume could make to amplitude.

Altogether 86 ears were tested and all the ears had detectable TEOAE responses, giving a 100% detectability rate. In 11.63% of the samples, the emission amplitudes were below 0.0 dB.

1. **The response waveform on visual inspection.**

Figure 17 (a) - (g) shows the time-domain average waveforms from seven subjects ranging in age from 50 days (a) and 28 years (g). In each trace the abscissa is 20 millisecond long with the first 4 milliseconds zeroed. As is apparent, the amplitude and temporal characteristics of the TEOAEs are different across the seven ears. The amplitude of the emission falls rapidly with time in figures (d) to (g). It is also clear that each peak is broader and the time gap between two successive peaks increases from (a) to (g). The large interpeak interval

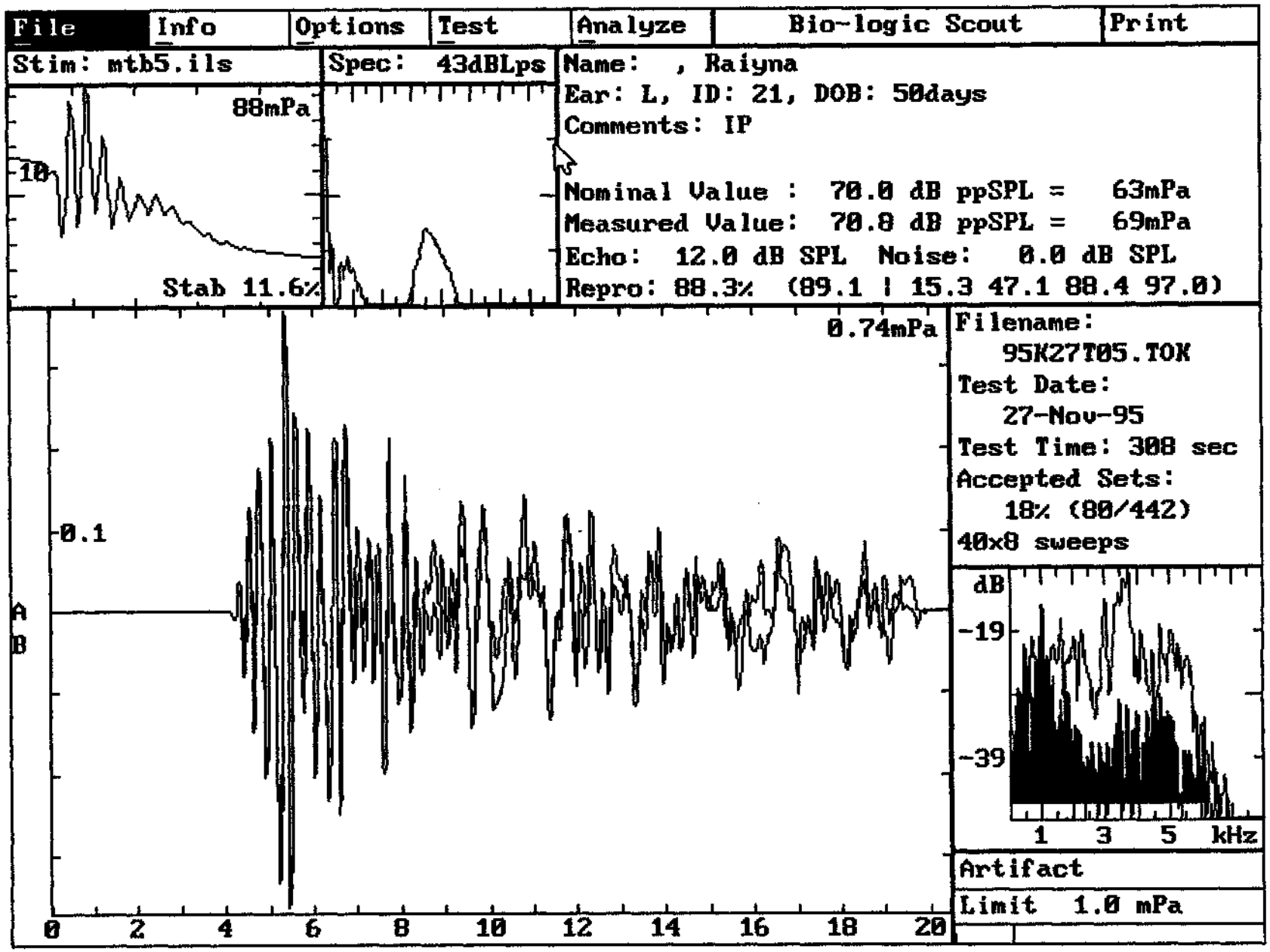


Figure 17(a). Waveform of subject aged 50 days.

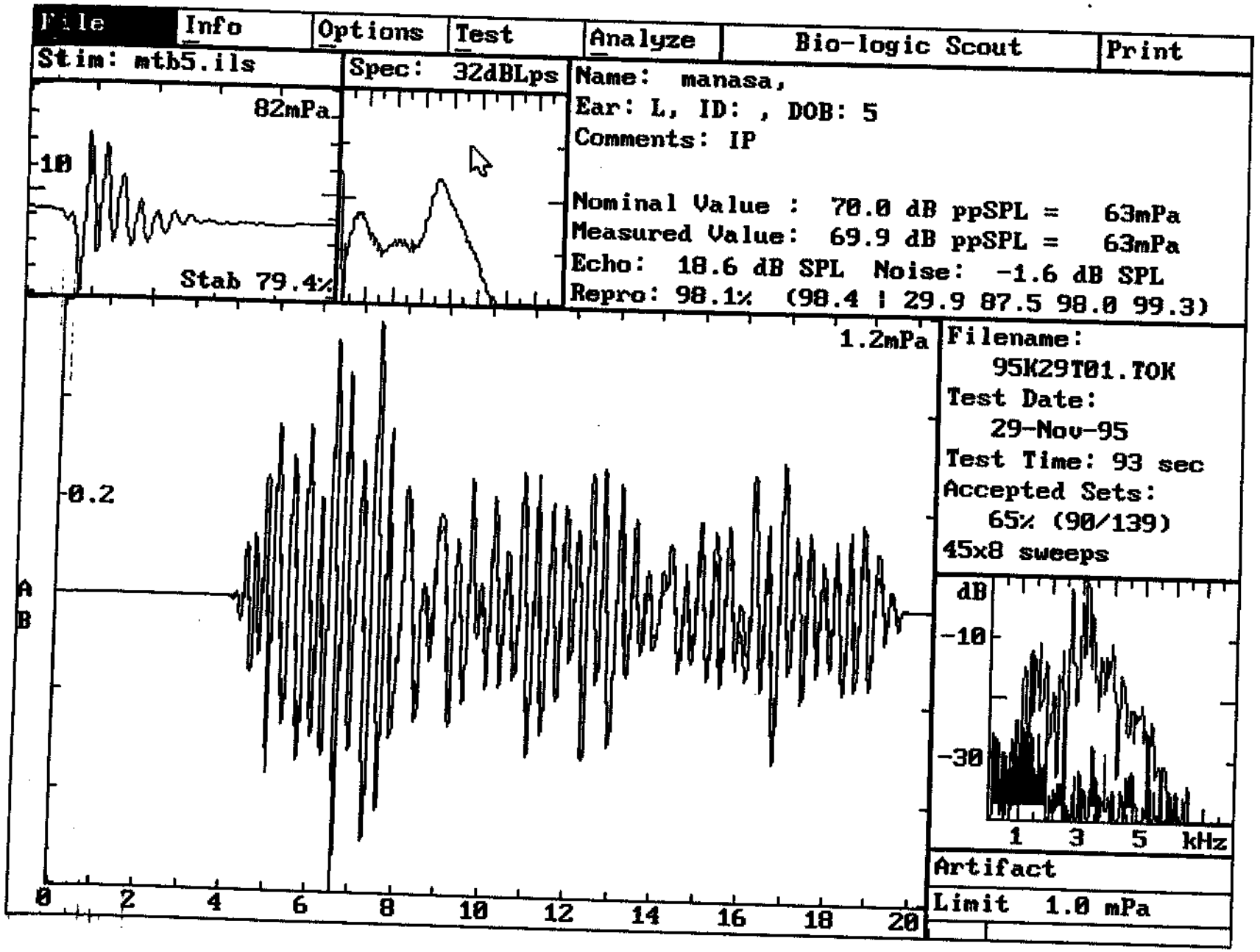


Figure 17(b). Waveform of subject aged 4.0 years.

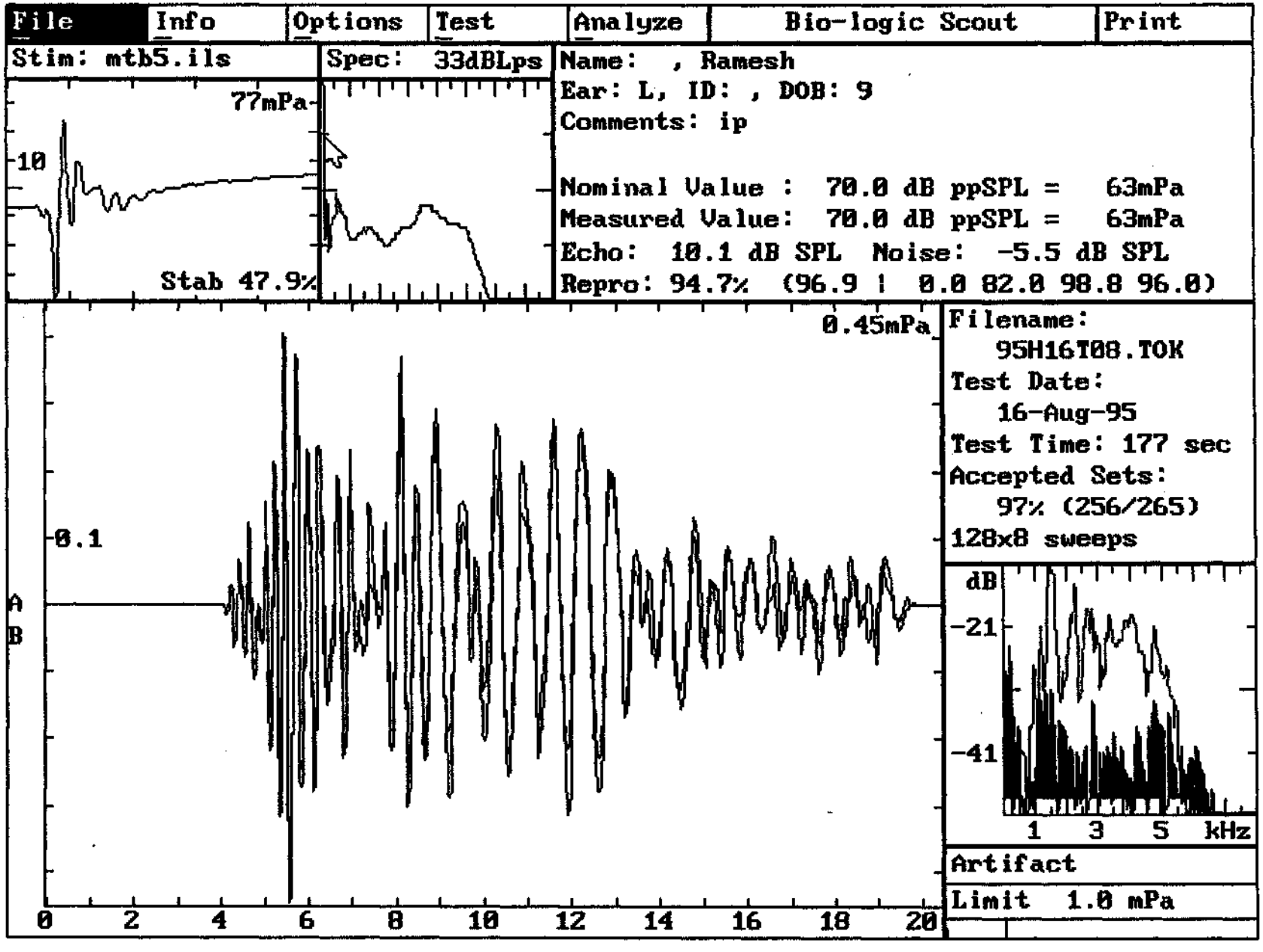


Figure 17(c). Waveform of subject aged 90 years.

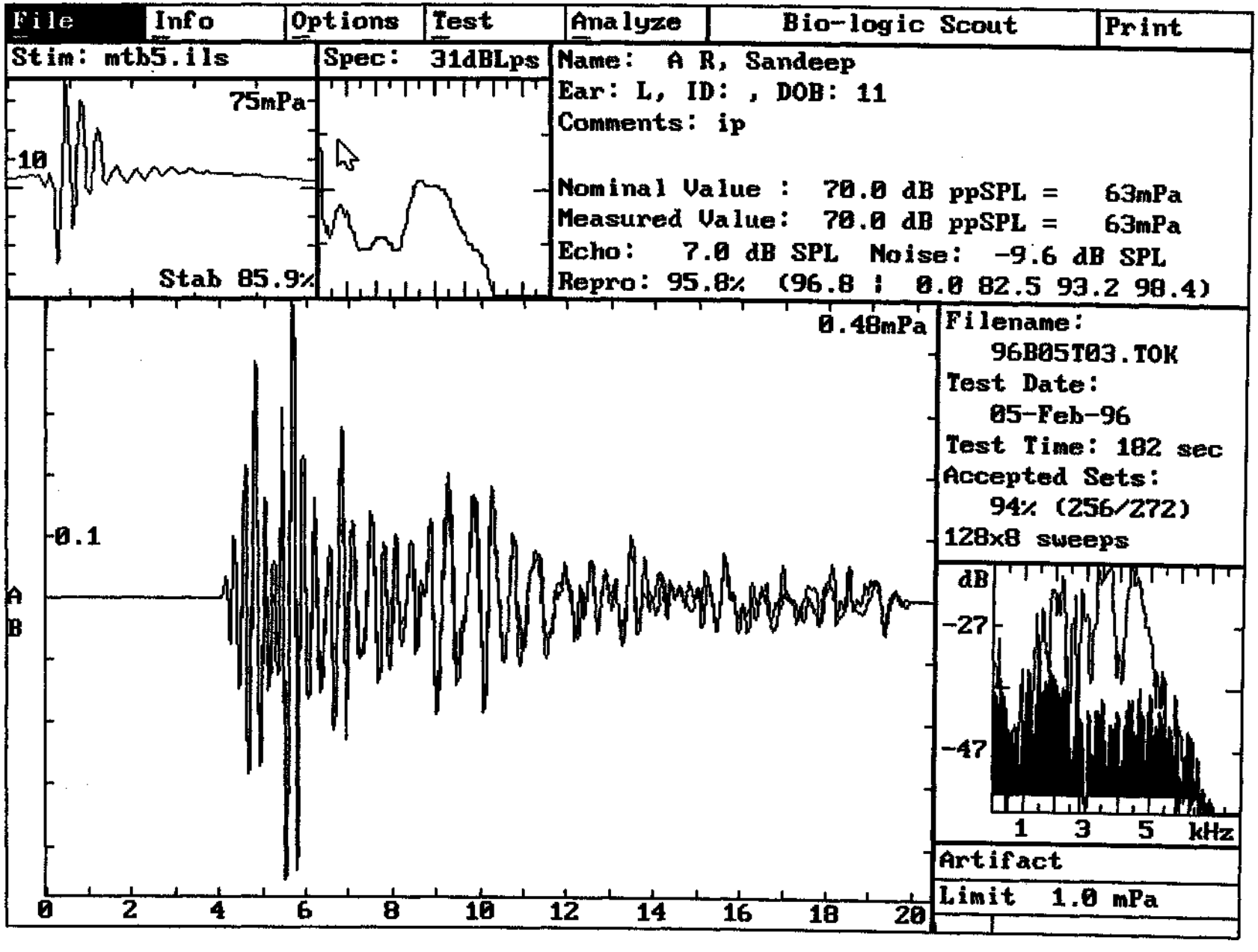


Figure 17 (d). Waveform of subject aged 11 years.

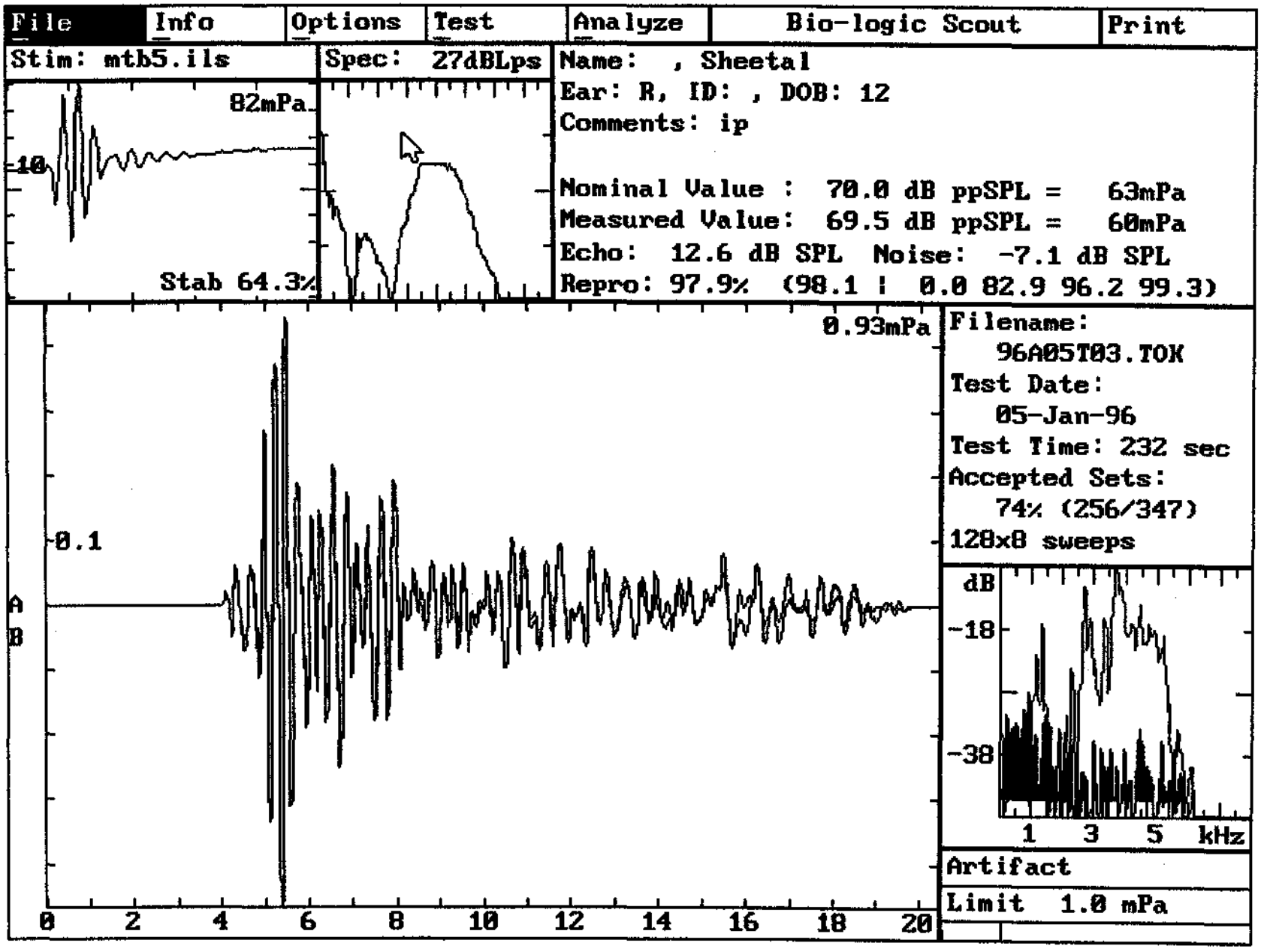


Figure 17(e). Waveform of subject aged 12.5 years.

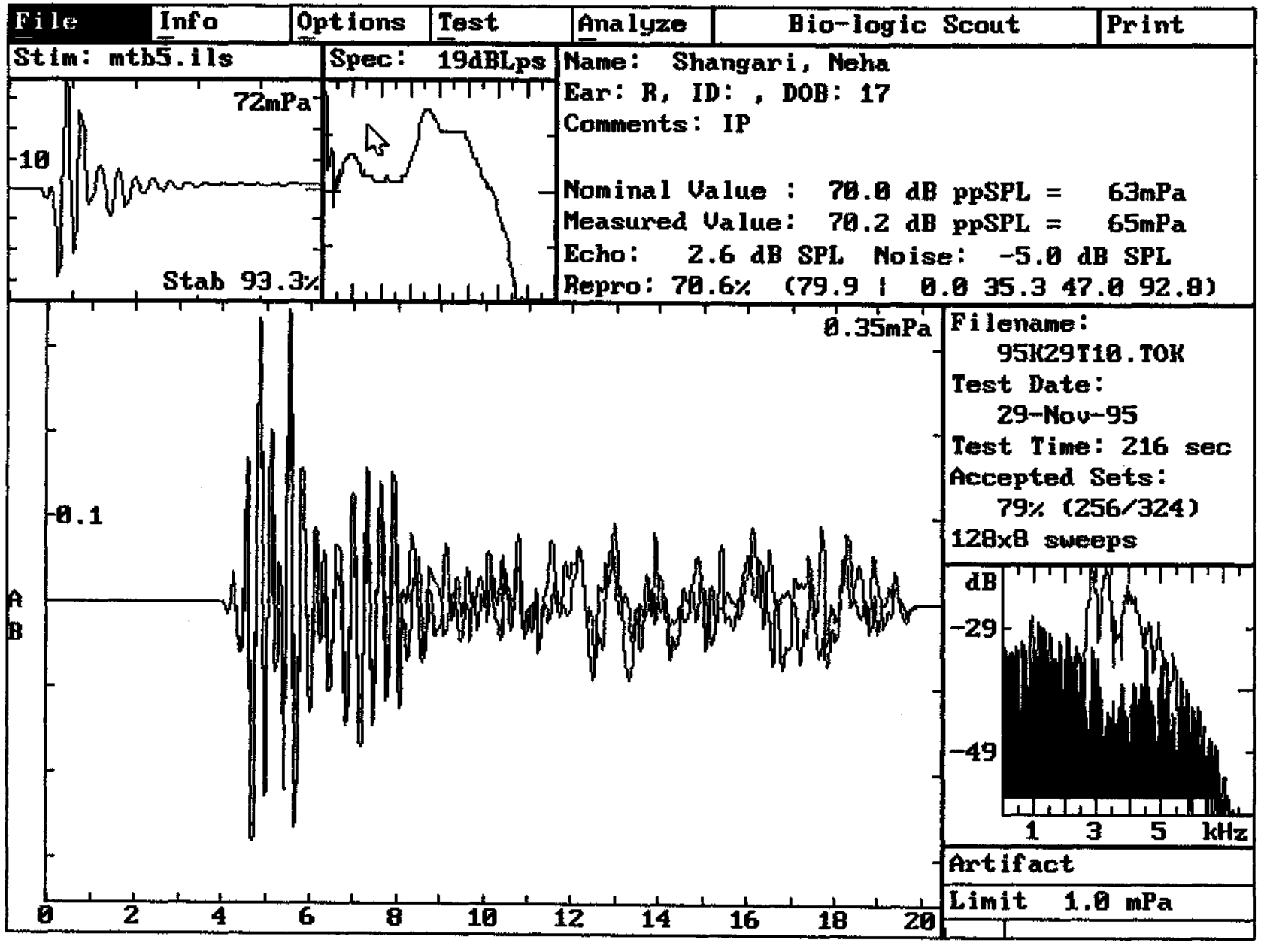


Figure 17(f). Waveform of subject aged 17 years.

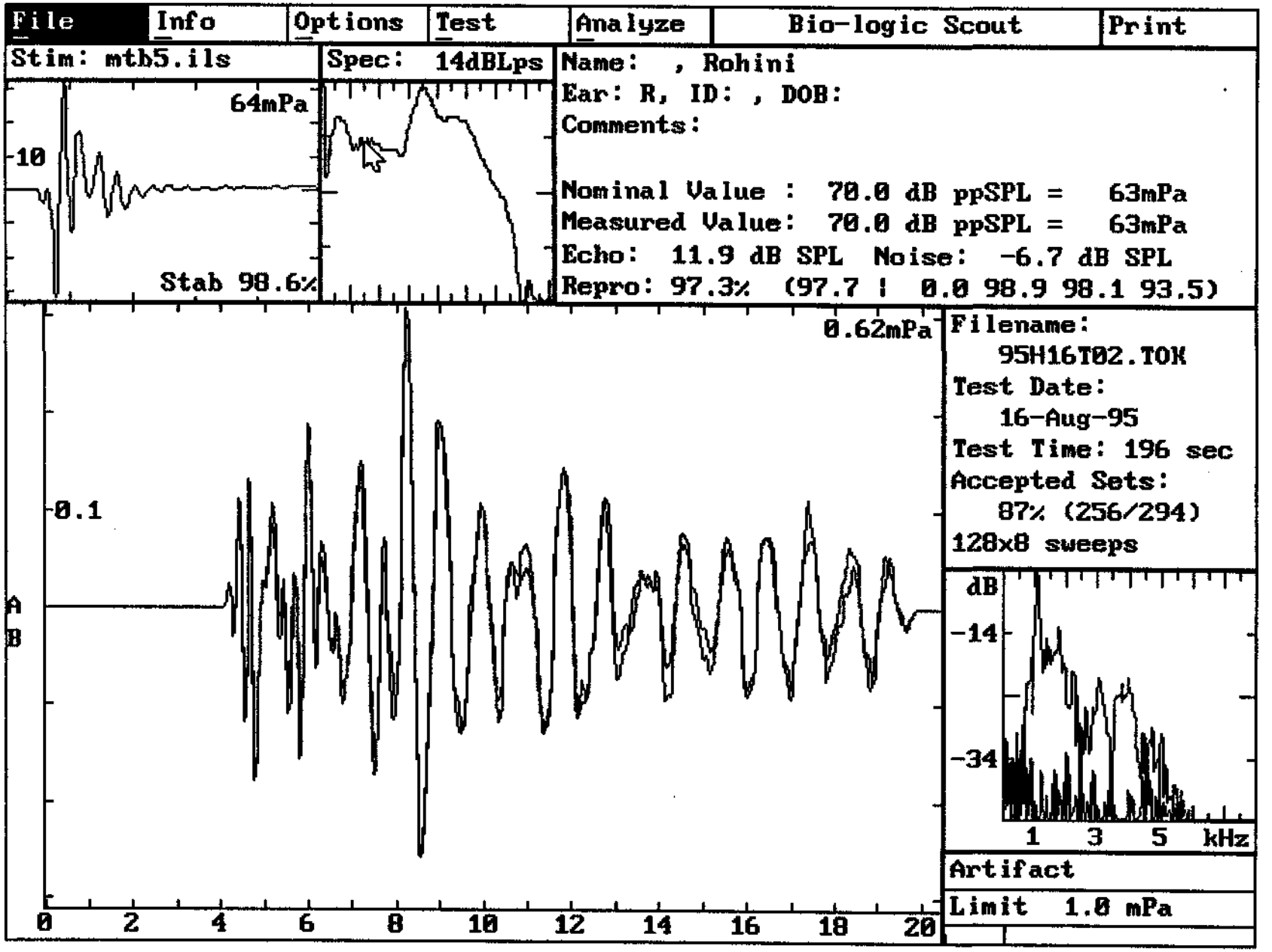


Figure 17(g). Waveform of subject aged 28 years.

is very apparent in (g) which is the emission wave nospail form of a 28 year old healthy female subject.

The response spectrum (solid line) and background noise (shaded area) are shown on the lower right corner in each of the figures 8 (a) - (g). In (a) and (b) the emission is spread beyond 5 kHz. In (c) - (f) the emission is limited to below 5 kHz. In (g) the spread is only upto 4 kHz. This illustrates that the frequencies at which emissions occur become more restricted with age. Moreover in (e) and (g) distinct notches are seen in the spectrum at 2 kHz. in (e) and closer to 4 kHz in (g).

Further, the extent of the shaded area (the noise floor) decreasedf with age.

2. **Amplitude**

Table 2 shows the mean, standard deviation and range of the emission amplitude in dB for all the age groups, adults and children male and female separately. The average amplitudes steadily decrease with age from 14.98 dB for Group I to 4.71 dB for group VII as illustrated in Figure *1%*. The Mann-Whitney U test was used to calculate the significance of the difference between the means of adjacent age groups. The amplitude differences were significant only between groups II (3.0 - 5.11 years) and III (6.0 - 8.11). The U values

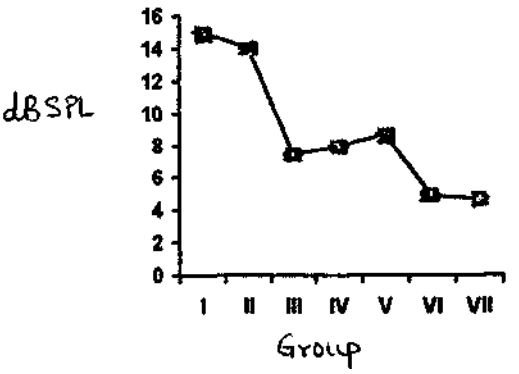
are given in Table 3. The 't' test was used to calculate the significance of the difference in amplitude between adults (group VII) and children (group I - VI). The difference is significant at the 0.05 level.

One-way analysis of variance (ANOVA) was used to calculate the inter ear and inter subject variability in amplitude. The adult male subjects group VII was taken as representative and the F value was calculated taking the amplitudes of the left and the right ears separately. The calculated F value was 0.0035 (Table 4), whereas the F value required for signifcance is 4.49 ($P < 0.05$). Therefore the inter ear variability is not significantly different from the intersubject variability.

Table 2. Variation of average emission amplitude with age.

Group	Age	Echo amplitude (dB)		
		Mean	Standard deviation	Range
I	0-2.11	14.98	4.87	12.0 - 25.0
II	3.0-5.11	14.02	4/60	8.0-22.1
III	6.0-8.11	7.46	6.98	-5.2 - 16.6
IV	9.0- 11.11	7.94	6.98	-5.9 - 12.8
V	12.0- 14.11	8.66	2.51	5.6 - 12.6
VI	15.0- 17.11	4.91	6.39	-5.8 - 15.7
Children	0.0- 17.11	9.41	6.35	-5.9 - 2.5
VII	18 & above	4.71	5.67	-4.9 - 15.6
Males	0.0-above 18	4.77	3.90	- 5.8-17.1
Females	0.0 - above 18	9.62	5.76	0.9-22.1

Figure 18. Averaged amplitude of each age group



Groups being compared	U Value	Minimum tabled value	Interpretation
I and II	25	45	Not significant
II and III	66	58	Significant (P=0.005)
III and IV	37	82	Not significant
IV and V	52	82	Not significant
V and VI	32	82	Not significant
Adults 8B I-VI	32	t = 1.85	Significant

* P=0.05

Table 3. The results of the Mann-Whitney U test used to calculate significance of the difference between the Groups I to VI. The last set of scores is the obtained *"V* value required for signifance when the adult group was compared with children (groups I to VI).

TABLE 4. The results of ANOVA for inter ear and inter subject variability in amplitude of otoacoustic emissions for the adult male subjects

Source	SS	df.	S2	F
Between groups	0.1152	1	0.1152	0.0035
Within groups	526.42	16	32,90	
TOTAL	526.53	17	33.1052	

*p , 0.05

3.Noise floor

The background noise levels for the seven age groups are presented in Table 5. The values steadily decrease with age, except for Groups IV and V, which show a slight increase (Figure 19)

Table 5. Age and noise level (dB)

Gorup	Age (Years)	Background noise (dB)		
		Mean	Standard Deviation	Range
I	0.00-2.11	- 1.64	3.8	-6.3 to + 6.6
II	3.0-5.11	-1.69	5.62	-8.7 to -13.1
III	6.0-8.11	-6.46	1.71	-8.8 to - 2.9
IV	9.0- 11.11	-5.20	2.42	-9.6 to - 2.4
V	12.0- 14.11	-5.57	1.38	-7.6 to -3.0
VI	15.0- 18.11	-.7.29	2.53	-11.5 to-3.2
Children	0.0- 17.11	-4.11	2.04	-11.5 to 13.1
VII	18 & above	-9.13	1.72	-11..7to-4.7
Males	0.00 -above 18	-7.12	2.84	-11.9 to 0.2
Females	0.0 - above 18	-.641	3.6	-11.5 to 6.6

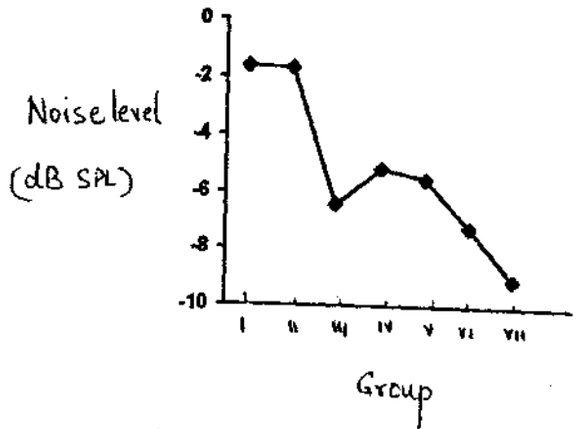


Figure 19. Average noise level of each age group.

4.Testing time

The average time in seconds required to complete the test in one ear, standard deviation and range values are given in Table 6. The testing time decreased with increasing age from 244.4 sec. for group I to 185.9 sec. for group VII, except for Groups IV and V where it is higher (also see Figure 2 c).

Table 6. The time taken to test (seconds) subjects in different age groups.

Group	Age (Years)	Testing time (seconds) Mean	Standard Deviation	Range
I	0.0-2.11	244.4	55.7	179 - 30.8
II	3.0-5.11	233.5	129.1	183 - 5.59
III	6.0-8.11	211.8	31.98	177 - 276
IV	9.0- 11.11	244.22	63.51	183.407
V	12.0- 14.11	226.3	12.53	218.251
VI	15.0- 17.11	194.8	15.6	172 - 220
Children	0..00- 17.11	225.83	17.8	183.558
VII	18.0 & above	185.91	19.4	173-2.51
Males	0.00 to above 18	200.97	22.7	172-308
Females	0.0 to above 18	208.13	31.7	173-558

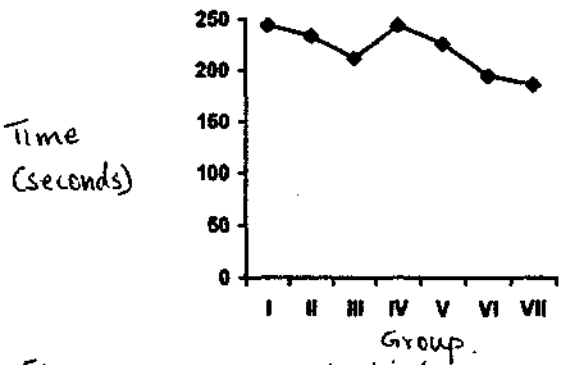


Figure 20. Average testing time (seconds) of each age group

5. Reproducibility

The reproducibility of the wave form over time in percentage is presented in Table 7 and Figure 21 . The average value exceeds 75% in all the age groups except group IV where it is only 40.73 percent. This is due to the very low reproducibility scores of one subject. However, there does not seem to be any age-related trend. This is seen in Figure 22, which shows TEOAE amplitude as a function of reproducibility decreases as amplitude increases. That means, smaller TEOAEs are more affected by noise than larger ones

Table 7. The age and reproducibility

Group	Age (Years)	Reproducibility % Mean	Standard Deviation	Range
I	. 0.00 -2.11	88.7	10.71	63.98
II	3.0 -5.11	75.29	30.03	24.1-99.7
III	6.0 -8.11	76.79	35.71	12.3 - 99.2
IV	9.0- 11.11	40.73	39.87	16.8 - 96.7
V	12.0- 14.11	93.99	4.37	86.4 - 98.9
VI	15.0- 17.11	79.53	22.67	27.7 - 99.6
VII	18 & above	85.87	16.39	30.2 - 99.8
Males	0.00 -18 above	87.33	20.52	20.2 - 99.8
Females	0.0 -above 18	89.48	24.14	12.3 - 99.2

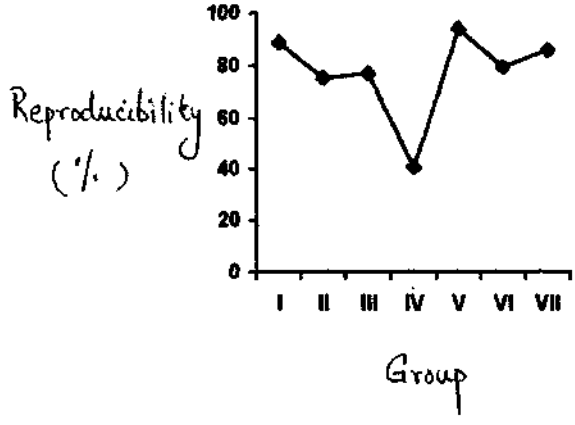


Figure 21. Average reproducibility (%) of each age group.

6. Ear Canal volume and its correlation with amplitude

The average physical volume of the ear canal is presented in Table 8. This increases with age. The increase is greatest between groups II and III, and IV and V, it is least for groups V, VI and VII.

The linear coefficient of correlation between ear canal volume and amplitude is negative for all the groups (Table 9). It is high for groups I (r = -.91) and II (r =-0.73). In groups III to VII it is lower. The coefficient of correlation for the whole group was calculated by the Karl-Pearson's Product Moment Method and it is 0.62.

The trend is illustrated graphically (figure 23)

Table 8. The physical volume of the ear canal and age.

Group	Age	Physical volume of ear canal (ml)		
		Mean	Standard Deviation	Range
I	0.0 -2.11	0.50	0.10	0.4-0.7
II	3.0 -5.11	0.54	0.11'	0.4 - 0.7
III	6.0 -8.11	0.85	0.16	05-1.1
IV	9.0- 11.11	0.84	0.15	0.7 - 1.0
V	12.0 - 14.11	1.05	0.13	09 - 1.3
VI	15.0- 17.11	1.25	0.16	10.1.5
VII	18 & above	1.32	0.32	1.0- 1.8

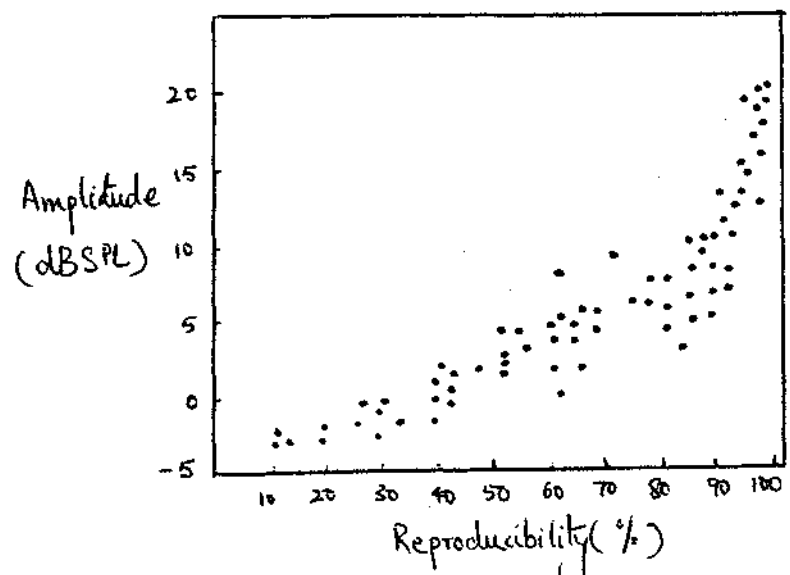


Figure 22. Amplitude (dBSPL) as a function of reproducibility (%)

Figure 23. Average earcanal volume of each age group.

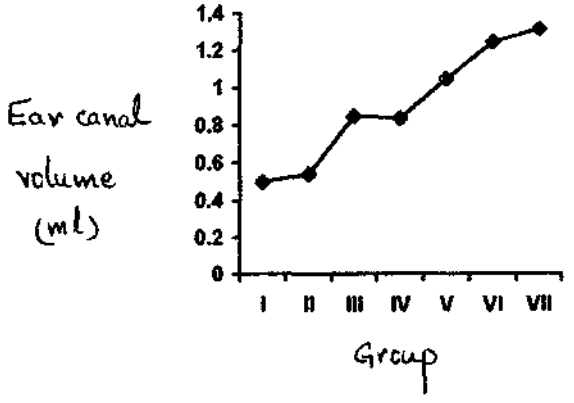


Table 9. The linear coefficient of correlation and age.

Group	Age (Years)	Correlation coefficient
I	0-2.11	0.91
II	3.0-5.11	0.83
III	6.0-8.11	0.54
IV	9.0- 11.11	0.48
V	12.0- 14.11	0.41
VI	15.0- 17.11	0.48
VII	18.0 and above	0.47

DISCUSSION

The TEOAE characteristics in normal individuals have been studied extensively, both in children and adults (Johnsen et al., 1982, 1983, 1988; Weir, 1984; Bray and Kemp, 1987; Bonfils et al, 1988; Collet et al., 1990; Kemp, Ryan and Bray, 1990; Norton and Widen, 1993; Stover, 1993; Engdahl, 1994; Thornton et al., 1994}. Each of the results is discussed separately.

Prevalence

The present study found TEOAEs in 100% of the subjects. This has been reported by several authors. Table 10 summarizes the occurrence values reported in literature. Though it is tempting to conclude that all healthy ears display TEOAEs, the failure of all the studies to have a 100% occurrence rate (Grandori, 1983; Probst et al., 1986; Stevens, 1988) should not be overlooked. This raises the question of what exactly causes an otoacoustic emission (other than a healthy cochlea). Martin, Probst and Lonsbury - Martin (1990) felt that the failure to detect TEOAEs from normally hearing individuals could occur under clinical conditions due to the varied anatomical properties of the ear canal or middle ear, equipment - related difficulties or subject-generated noise problems. It must be emphasized that they did not attribute the failure to a midly

disordered cochlea. Martin, Probst and Lonsbury Martin (1990) further stated that the spectrum of the stimulus is extremely important in eliciting TEOAEs. They reported of a subject in whom TEOAEs were not detected in response to clicks but were present in response to tone bursts. If TEOAEs are to be used as clinical tools it is important that they have a 100% occurrence in healthy ears and audilogists recognize conditions which bring about a failure to record TEOAEs.

Table 10. Prevalence of TEOAEs as reported in literature.

Study	No. of ears tested	No. of ears TEOAEs	% ears TEOAEs
Kemp (1978)	35	35	100
Grandori(1983)	23	22	96
Kemp et al.(1986)	150	150	100
Probst et al.(1986))	28	27	96
Bonfils et al.(1988)	262	262	100
Stevens (1988)	36	35	97

Emission Waveform

In the present study, a) the peaks in the waveform were spread to a greater extent over time as age increased, and (b) the frequency spectrum of the emission became restricted with age. Norton and Widen (1990) compared the TEOAE waveforms of normal hearing subjects ranging from 1.5 months to 20.5 yars. They found similar

differences in the frequency spectrum and temporal characteristics. Johnsen and Elberling (1989) conducted a longitudinal study where the subjects were tested as neonates and again at four years of age. The emissions of the four year olds had a relatively restricted frequency spectrum and the peaks of the waveform were further apart.

The above findings clearly demonstrate that the frequency of the waveform changes with age and it may lead us to conclude that the hearinfg function changes even in childhood (the deterioration in the hearing ability of geriatrics is not debated here). But humans are generally considered precocial with regard to auditory function becase most of the peripheral auditory system development is complete before birth. Therefore the changes observed in the TEOAE spectrum could be due to changes in the external and middle ear transfer characteristics. The resonance of the ear canal in neonates may modulate the stimulus and response spectrum and create a dominant high frequency component Kinger (1981) found that the resonant frequency of neonatal ear canals ranged from 5.3 to 7.2 kHz and that of adults was 2.7 kHz at an average. The neonates ear canal resonant frequency decreased to 2.7 kHz by 20 months of age.

Amplitude

The present study found a steady decrease in amplitude with age which was significant between

a) groups II (3.0 -5.11 years) and III (6.0 -8.11 years)

b) adults (above 18.0 years) and children (below 17.11 years)

c) males and females.

Bray and Kemp (1987), Norton and Widen (1990) and Norton (1993) found a similar reduction in amplitude with age. Johnsen and Elberling (1989) tested 20 subjects at birth and again at four years of age. They noted no differences in amplitude for any of the subjects. In the present study the average amplitude of group I (0.0 - 2.11 years) was 14.98 dBSPL (S.D = 4.60). This agrees with the findings of Johnsen and Elberling (1989) mentioned above.

Norton and Widen (1990) noted that on average, the neonatal responses were stronger than adult responses by 10 dB SPL. In the present study, the average responses of group I (0.0 - 2.11 years) were 10.27 dBSPL stronger (14.98 - 4.71 dBSPL) than the average responses of group VII (above 18.0 years).

But there is high individual variability as evident from the large range values of each age group (Table 2). High individual variability was also reported by Bray and Kemp (1987) and Norton and Widen

(1990). Thus is an overlap in values between groups VII and I too. There one cannot set cut-off values for adult and child emissions. This implies that when a minor cochlear pathology may reduce the amplitude of an emission without causing it to disappear altogether, this cannot be detected. Therefore, the claim by many researchers that minor cochlear pathology can be detected is questionable.

The cochlea is influenced by ambient auditory input, as is evident from studies where cochlear damage has been inflicted by noise exposure. Therefore the question arises whether everyday auditory experiences actually cause deterioration in the auditory system (Rubel, 1985). This is unlikely because it is not reflected in the behavioral thresholds of children, which actually improve with age (Northern and Downs 1984).

In the present study female subjects were found to have a larger mean amplitude (9.62 dBSPL) than the male subjects (4.81 dBSPL). Kok et al (1982) similarly reported female subjects to have a higher amplitude. Other studies do not report any gender differences.

ANOVA revealed that the inter ear variability was the same as intersubject variability. Johnsen and Etherling (1982) and Coren and Hakstian (1990) reported that the two ears of each individual are statistically relatively independent.

Noise, Reproducibility and Testing-time

The decrease in background noise that was noted in the present study was anticipated due to two reasons:

a) With age, the subjects became more co-operative and ceased to be restless.

b) Many of the young subjects fell asleep during the test. This resulted in heavy breathing and body movements.

The children of groups IV and V were very restless, as is apparent in their high average noise levels.

Reproducibility is related to noise as is evident from figure. Norton and Widen (1990) also noted an increase in reproducibility with a decrease in the background noise level.

The decrease in testing time that was noticed may also be because they become more co-operative with age. During the course of testing it was observed that the emission had a higher amplitude initially than later. Brownell (1983) talked about the possibility of reduction in the electromotile property of outer hair cells after continuous stimulation. The present observation supports this. Cutting down on the testing time would therefore give a better emission magnitude and also enhance the reproducibility since the subjects become restless after two-three minutes.

Have experimented with a presentation rate of 860 stimuli per second these were equally effective in eliciting TEOAEs.

Ear canal volume and its correlation with amplitude

The ear canal volume increased with age upto twelve years after which it did not change significantly. This corresponds with the high correlation coefficient in the younger age groups. One may safely conclude that the small ear canal volume of groups I and II does contribute to some extent to their high emission amplitudes.

The postnatal changes in the conductive apparatus (external and middle ear) are well documented. There are obvious differences in size, shape and tissue of the neonate and infant ear canals as compared to adult ears. The tympanic membrane is more horizontal in neonates than adults and the tympanic ring is incomplete. The infant external ear is more cartilagenious and therefore more compliant. Kruger (1987) calculated that the effective length of the ear canal in neonates was 12 mm and in adults was 32 mm. A majority of the changes in resonant frequency and length occurred in the first two years.

Feigin, Kapin and Stelmachowicz (1989) measured the sound pressure level generated in the ear canal using a probe microphone in children from age one month to five years. They found that infants

and children had consistently greater real ear-coupler differences, that is, higher ear canal sound pressure levels, than adults. These differences, which average 4 dB, gradually decreased with age, but were still present at five years. Nelson-Barlow, Auslander, Rines and Stelmachowicz (1988) found no differences between the 8 year olds and the adults.

The above literature supports the findings of the present study. That is, the coefficients of correlation for groups IV (9.0 -11.11 years), V (12.0 - 14.11 years), VI (15.0 - 17.11 years) and VII (above 18.0 years) do not vary significantly. Bray and Kemp (1987) were among the first to attribute the larger TEOAE amplitude in children to the smaller ear canal size.

Ear canal acoustic obscure otoacoustic emissions in other ways, notably by the occurrence of cross-talk and standing waves (Siegel, 1993, 1994, 1995). Siegel (1994) noted that low-noise microphones designed to measure otoacoustic emissions from the human ear canal typically sampled the sound filed in the canal 15 to 20 mm away from the eardrum. The input sound levels are usually defined as "sound-pressure level at the ear drum". But standing waves produce a spatially non-uniform pressure for frequencies above 2.3 kHz. Siegel demonstrated this by simulating the measurement condition. He also

showed that large (± 20 dB) errors in the estimated ear drum sounds pressure level values occurred when the position of the sound source was varied. He suggested that the measurements be made near the eardrum.

Earlier work (Siegel, 1993) revealed that the ear drum sound pressure level is underestimated by 15-20 dB for stimulus frequencies near 5-7 kHz. That means the actual sound pressure level at the ear drum exceeds the desired level by 15 - 20 dB. Since otoacoustic emission values vary nonlinearly with stimulus intensity it is difficult to estimate the differences in emission behaviour caused by the above (Siegel, 1994).

There are also individual differences (Siegel, 1994) due to variations in probe placements and ear canal lengths. Group data will show less pronounced deviations since the measured value could be greater or lower.

This is critical in explaining the systematic differences between adult males and females and between infants and adults (Siegel, 1994) in emission amplitudes.

Siegel (1995) studied the possibility of internal coupling (cross-talk) between the sound source and probe microphone when the probe is placed in the human ear canal. The sound source tube or

microphone inlet was blocked with modelling clay. The resulting sound pressure values were compared with the "unblocked" condition, indicating cross-talk. Therefore, the accuracy of "in the ear" acoustic calibrations is questioned.

Further, he found that the cross talk levels could be brought down by using harder-walled sound source tubing.

SUMMARY AND CONCLUSION

The study aimed at:

1. Establishing norms for children and adults for transient evoked otoacoustic emissions.
2. Documenting age and gender related changes, if any.
3. Investigating the correlation between the amplitude of the emission and ear canal volume, in turn noting any contribution of ear canal acoustics to the measurements.
4. Outlining the methodology of transient evoked otoacoustic emission measurement and the likely difficulties that one might encounter while using it as a clinical tool.

Fifty three subjects (24 males, 29 females) whose ages ranged from fifty days to twenty eight years were tested after ruling out hearing loss.

Transient evoked otoacoustic emissions were measured using the Bio-logic Scout Plus System (Software Version 1.22) in a sound treated room. The ear canal volume was measured using an automatic immittance meter with visual display (Grasion stadler GSI-33, Version 2 Middle Ear Analyzer)

The obtained data were subjected to statistical analyses (mean, standard deviation, Mann-Whitney U test, ANOVA and Karl-Pearson's Product Moment Coefficient of correlation). The analysis revealed the following:

1. A hundred percent occurrance. The emission amplitudes were below O.OdB in 11.63% of the ears.
2. The emission spectrum became restricted in frequency with increasing age. The peaks in the wave form became more widely spaced with increasing age.
3. The average emission amplitudes steadily decreased with age. But there is wide individual variability within age groups. The interear variability is not significantly different from the intersubject variability.
4. The time background noise levels steadily decrease with age.
5. The time required to test decreased with increasing age.
6. The reproducibility of the waveform over time is above 75% in all the age groups except the 9.0 to 11.11 years.
7. The ear canal volume correlates highly with amplitude of emission under six years of age.

Implications of the study

1. The higher amplitude of emission in children may not be solely due to healthy cochleae. The smaller ear canal volumes in children may be contributory factor. This has a major theoretical implication.

2. It can be used clinically as a screening tool. The emission cannot be indicative of mild pathology because the normal values encompass a wide range. This carries immense clinical significance.

Limitations of the Study

1. The sample frame was healthy children from neighbouring schools and adult subjects from the All India Institute of Speech and Hearing, fulfilling certain rigid criteria as stated in the methodology. The generalizability of these results is limited to this extent.
2. The probe tips of the Biologic Scout Plus System (Version 1.22) used to measure transient evoked otoacoustic emissions and the Grason-Stadler GSI-33 Version 2 Middle Ear Analyser used to measure the physical volume of the ear canal differed in size. This introduced a systematic difference in the measured physical volume and the actual volume of the ear canal when the otoacoustic emissions were measured.

BIBLIOGRAPHY

Antonelli A. and Grandori, F. 1986 Long term stability, influence of the head position and modelling consideration for evoked otoacoustic emissions. Sandinavian Audiology, Suppl. 25: 97-108.

Bonfils, P., Avon, P., Martine, F. and Marie, P. 1990. Clinical significance of OAEs: A perspective. Ear and Hearing, 11: 155-156.

Bonfils, P., Fra ncois, M., Avan, P., Londero, A., Trotoux, J. and Narcy, P. 1992 Spontaneous and evoked otoacoustic emissions in pre-term monates. Laryngoscope, 102 (2): 182-187.

Bonfils, P., Uziel, A., Piron, and Pryot, P. 1988. Evoked OAEs from adults and infants. Clinical applications. Acta Otolaryngologica, 105: 445-449.

Bonfils, P., Uziel, A. and Pujol, R. 1988, Screening for auditory dysfunction in infants by evoked otoacoustic emissions. Archives of Otolaryngology and Head and Neck Surgery, 114:887-890.

Bonfils, P., and Uziel, A. 1989, Clinical interest of evoked otoacoustic emissions. Annals of Otology, Rhinology and Laryngology, 98:326-331.

Bonfils, P., Bertrand, Y. and Uziel, A. 1988. Evoked OAEs: Normative data and presbyacusis. Audiology, 27: 21-35.

Bonfils, P., Dumont, A., Marie, P., Francois, M., Narcy, P. 1990. Evoked otoacoustic emmission in newborn hearing screening. Laryngoscope, 100: 186-189.

Pray, P. J., and Kemp, D.T. 1987. An advanced cochlear echo suitable for infant screening. British Journal of Audiology, 210:191-

Bright,K.E., 1994. Handson Helpful Hints for OAE measurement. The Hearing Journal, 47(2) : 72:75.

Brownell, W.E. 1990 OHC Electromotility and OAEs. Ear and Hearing, I(2) : 82-93.

Cane, M.A., Donoghue, G.M., Lutman, M.E. 1992. The feasibility of using OAEs to monitor cochlear function during acoustic neuroma surgery. Scandinanian Audiology, 21(3) : 173-177.

Chang, K.W., Vohur, B.R., Norton, S.J. and otoacoustic lekas, M.D. 1993. External and middle ear status related to evoked emissions in neonates. Archives of Otolaryngology and Head and Neck Surgery, 119:276-282

Collet, L., Chanal, J.M., Hellal, H.., Gartner, M. and Moryon, A. 1989. Validity of bone conduction stimulated ABR, MLR and OAEs. Scandinavian Audiology, 18 (1): 43-47.

Collet, L., Gartner, M., Moutlien, A., Kaufmann, I., Disant, F., and Morgon A. 1989. Evoked OAEs and Sensorineural hearing loss. Archives of Otolaryngology and Head and Neck Surgery. 115: 1060-1062.

Collet, L., Moulin,A., Gartner, M. and Morgon, A. 1990. Age related change in evoked OAEs. Annals of Otology, Rhinhology and Laryngology,99 (12): 993-997.

Collet, L., Veuillet, E., Chanal, J.H. and Morgon, A. 1991. Evoked OAEs. Correlates between spectrum analysis and audiogram. Audiology, 30(3): 164-173.

Collet, L., Veuillet, E., Bene, J. and Morgon, A. 1992. Effects of contralateral white noise on click evoked emissions in normal and sensorineural ears towards an exploration of the medial olivo cochlear System. Audiology, 31:1-7.

Collet, L., Levy,V., Veuillet, E., Trury, E. and Morgon, A.M. 1993, Click evoked otoacoustic emissions and hearing threshold in sensorineural hearing loss. Ear and Hearing, 14: 141-143.

Collet, L., Veuillet, E., Moulin, A., Morlet, T., Giraud, A.L., Micheyl, C, Chery-Gross.A. 1992 Contralateral auditory stimulation and

otoacoustic emissions a review of basic data in humans. British Journal of Audiology, 298: 213-218.

Cope. Y., and Lutman M.E. Otoacoustic Emissions. In Mc. Cormick B. (ed.) "Pediatric Audiology, 0-5 years." 1994:221-247. A.I.T.B.S. Publishers and Distributors. Krishan Nagar, New Delhi.

Davis, H. 1993. An active process in cochlear mechaines. Hearing Research, 9: 79-90.

Devries, S.M. and Recker, T.N. 1992. Otoacoustic emissions: Overview of measurement methodologies. Seminars in Hearing, 13(1) : 15-21.

Dolhen, P., Hennaux, C, Chantry, P. and Hennebert, D. 1991. The occurrance of evoked OAEs in a normal adult population and neonates. Scandinavian Aodiology, 20 (3): 2-3-205.

Elberling, C., Parbo, J., Johnsen, M.J. and Bagi, P. 1985. Evoked otoacoustic emissions, Clinical applications. ActaOtolaryngologica, 421:77-85.

Engdahl, B., Arnesan, A.R., Mair, I.W.S 1994. Reproducibility and short-term variability of transient evoked otoacoustic emissions. Scandinavian Audiology, 23(2) : 99-104.

Engdahl, B., Arnesan, A.R., Mair, I.W.S. 1994. Otoacoustic emissions in the first year of life. Scandinavian Audiology, 23 (3): 195-201.

Fabiani, M 1993. Evoked Otoacoustic emissions in the study of adult sensorineural hearing loss. British Journal of Audiology, 27(2): 131-139.

Fortum, H., Farnsworth, A., Davis, A. 1993. The feasibility of evoked OAEs as an in-patient hearing check after meningitis. British Journal of Audiology, 27(4): 227-233.

Furst, M. and Lapid, M.A. 1988. A cochlear model for acoustic emissions. Journal of Acoustical Society of America, 84: 222-229.

Gobsch, H., Kevanishvili, Z., Gamgebeli, Z., Gvelesian, T. 1992. Behavior of delayed evoked OAEs under forward masking paradigm Scandinavian Audiology, 21(3) : 143-149.

Grandori, F. 1985. Non-linear phenomena in click and tone-burst evoked otoacoustic emissions from human ears. Audiology, 24: 71-80.

Guelke, R.W. and Bum, A.E. 1985. A mechanism for stimulated otoacoustic emissions in the cochlea. Hearing Research, 19(3) : 185-191.

Harris, F.P. and Probst, R. 1991. Reporting click-evoked and distortion - product otacoustic emission results with respect to the pure tone audiogram. Ear and Hearing, 12 (6): 399-405.

Harris, F.P., Probst, R. and Wenger, R.1991. Repeatability of transcantty evoked OAEs in normally hearing humans. Audiology, 30(3): 135-142.

Harris, F.P. and Probst, R. 1992 Transiently evoked otoacoustic emissions in patients with Menieres disease. ActaOtolaryngologica, 112(1): 36-45.

Hunter, M.F. Kimm, L., Cafarelli - Dees, D.C., Kennedy, C.R., Thornton, A.R.D. 1994. Feasibility of Otoacoustic Emission Detection followed by ABR as a universal neonatal screening test for hearing impairment. British Journal of Audiology, 28:47-51.

Johnsen, N. J. and Elberling, C. 1982. Evoked Otoacoustic emissions from the human ear. Equipment and Response Parameters Scandinavian Audiology, 11: 3-12.

Johnsen, M.J., Bagi, P. and Elberling, C. 1983. Evoked OAEs from the human ear. III Findings in neonates. Scandinavian Audiology, 12: 17-24.

Johnsen, N.J. Bagi, P. Parbo, J. and Elberling, C. 1988. Evoked OAEs from the human ear. IV. Final results on 100 neonates Scandinavian Audiology, 17: 27-34.

Johnsen, N.J., Parbo, J. and Elberling, C. 1989. Evoked Otoacoustic emission from the human ear. V. Developmental changes Scandinavian Audiology, 18(1): 59-62.

Johnsen, N.J., Parbo, J. and Elberling, C. 1993 Evoked acoustic emissions from the human ear VI. Findings in cochlear hearing impairment. Scandinavian. Audiology, 22(2): 87-96.

Johnsen, N.J. and Elberling, C. 1982. Evoked OAEs from the human ear and influence of posture. Scandinavian Audiology, 11(1): 69-77.

Kemp, D.T. 1978. Stimulated acoustic emissions from within the human audiary system. Journal of Acoustic Society of America, 65: 1386-1391.

Kemp, D.T. 1979. The evoked cochlear mechanical response and the auditory microstructure - evidence for a new element in cochlear mechanics. Scandinavian Audiology, Suppl. 9:35-47.

Kemp D.T. 1980 Towards a model for the origin of cochlear echoes. Hearing Research, 2:533-548.

Kemp, D.T. 1986. Otoacoustic emissions, Travelling waves and cochlear mechanisms. Hearing Research, 22: 95-104.

Kemp, D.T. 1990, Otoacoustic emissions - basic facts and applications, Audiology in Practise, 7(3).

Kemp, D.T. and Chum, R. 1980. Properties of the generator of stimulated acoustic emissions. Hearing Research, (2): 213-232.

Kemp, D.T., Ryan, S., Bray, P. 1990. A guide to the effective use of otoacoustic emissions. Ear and Hearing, 11 (2): 93-105.

Kemp, D.T. and Ryan, S. 1993. The use of transient evoked otoacoustic emissions in neonatal hearing screening programs. Seminars in Hearing, 14 (1): 15-23.

Kim, D.O. 1980, Cochlear mechanics: implications of electrophysiological and acoustical observations. Hearing Research, 2:297-317.

Kok, M.R., Van Zanten, G.A., and Brocaar, M.P. 1992. Growth of evoked Otoacoustic emissions during the first days post-partum: a preliminary report. Audiology, 5: 140-149.

Kossl, M. and Vater, M. 1985. Evoked acoustic emissions and cochlear microphonics in the Mustache Bat, Pteronotus Parnellii. Hearing Research, 19(2): 157-171.

Lafreniere, C.C. Jung, M.D., Smurzynski, J., Leonard, G., Kim, D.O. and Sasek, J. 1991. Distortion product and Click-evoked OAEs in healthy newborns, Archives of otolaryngology and Head and Neck Surgery, 117: 1382-1389.

Lim, D.J. 1986. Cochlear micromechanics in understanding otoacoustic emissions. Scandinavian Audiology, Suppl. 25: 17-25.

Lind, O. and Randa, J. 1989. Evoked acoustic emissions in high frequency versus low or medium frequency hearing loss Scandinavian Audiology, 18 (1): 21-27.

Long, G.R. and Tubis, A. 1988. Modification of spontaneous and evoked OAEs and associated psycho-acoustic microstructure by aspirin consumption. Journal of Acoustical Society of America, 84: 1343-1353.

Lutman, M.E. 1990. Evoked otoacoustic emissions in adults: Implications for screening. Audiology in Practise, 6(3) : 6-8.

Lutman, M.E. Sheppard, S. 1990. Quality estimation of click evoked otoacoustic emissions. Scandinavian Audiology, 19(1): 3-9.

Lutman, M.E. 1993. Reliable identification of click-evoked OAEs using signal processing techniques. British Journal of Audiology, 27: 103-108.

Lutman, M.E., Mason, S.M., Sheppard, S. Gibbin K.P. 1991. Differential diagnostic potential of OAEs: A case study Audiology, 28 (4): 205-211.

Martin, G.K., Probst, R. and Lonsbury-Martin, B.L., 1990. OAEs in Human ears - Normative findings. Ear and Hearing, 11: 106-120.

Martin, G.K., Lonsbury-Martin, B.L., Probst R. and Koats, A.C. 1985. Spontaneous otoacoustic emissions in the non-human primate: A survery Hearing Research, 20(1):: 91-95.

Meredith, R., Stephens, D. Hogan, S., Cartlidge, P.H.T. Drayton, M. 1994 Screening for hearing loss in an at-risk neonatal population using evoked otoacoustic emissions. Scandinavian Audiology, 23(3): 187-194.

Meric, C., and Collectic, L. 1993, Comparative influence of repeated measurements and of attention on electroc otoacoustic emissions. Acta Otolaryngologica, 130 (4) : 471-477.

Micheyl, C, Collect, L. 1994. Interrelations between psychoacoustical tuning curves and sponatneous and evoked otoacoustic emission. Scandinavian Audiology, 23(3): 171-178.

Morgan, D.E. and Canalis, R.F. 1991, Auditory screening in infants Otolaryngologica Clinics of North America, Clinical Audiology, 2: 277-284.

Moulin, A., Collet L., Veuillet, E., Morgon, A.M. 1993. Inter-relations between transiently evoked otoacoustic emissions, spontaneous otoacoustic emissions and acoustic distortion products in normally hearing subjects, Hearing Research, 65: 216-233.

Naeve, S.L, Margolis, R.H., Levine, S.C., and Fournier, E.M. 1992. Effect of ear canal air pressure on evoked otoacoustic emissions. Journal of Acoustical Society of America, 91: 2091-2095.

Norton, S.J., Widen, J.E. 1990. Evoked Otoacoustic Emissions in Normal-Hearing Infants and Children. Emerging Data and Issues. Ear and Hearing, 11: 121-127.

Norton, S.J. and Neely, S.T. 1987. Tone burst-evoked otoacoustic emissions from normal hearing subjects. Journal of Acoustical Socciety of America, 81: 1860-1872.

Norton S.J. 1993. Application of Transient evoked otoacoustic emissions to plediatric populations. Ear and Hearing, 14: 64-73.

Norton, S.J. 1992. Cochlear function and otoacoustic emissions, Seminars in hearing, 13 (1): 1-14.

Norton, S.J. and Stover L.J. 1994.Otoacoustic emissions In Katz , J. ed. Handbook of Clinical Audiology 4th edition William and Wilkins Co., Baltimore, U.S.A.

Norton, S.J., Schmidt, N.R. and Stover L.J. 1998, Tinnitus and OAEs Is there a link ? Ear and Hearing 11(2): 159 - 167.

Patuzzi, R. 1993, OAEs and the categorization of cochlear and retrocochlear lesions British Journal of Audiology, 27 (2): 91-97.

Penner, M.J. 1988, Audible and annoying SOAEs: A case study, Archives of Otolaryngology and Head and Neck Surgery, 114: 150-153.

Prieve, B.A. 1992. Otoacoustic emissions in infants and children: Basic characteristics and clinical applications. Seminars in Hearing, 13(1).

Prieve, B.A., Gorga, M.P. Schmidt, A. Neely, S. Pters, J. Schultes L. and Jesteadt W. 1993. Analysis of transient - evoked otoacoustic emissions in normal-hearing and hearing-impaired ears. Journal of AcousticalSociety of America, 6.

Probst, R. and Harris P 1993. Transiently evoked and distortion product OAEs, Comparison of results from normally hearing and hearing-impaired human ears. Archives of Otolaryngology and Head and Neck Surgery, 119 (8): 858-860.

Probst, R., Coats, A.C. Martin, G.K. and Lonsbury-Martin B.L. 1986. Spontaneous, Click and tone-burst evoked oto-acoustic emissions from normal ears. Hearing Research, 21 (3): 261-277.

Probst, R., harris, F.P. and Hauser R. 1993. Clinical Monitoring using OAEs. British Journal of Audiology, 27 (2): 85-91.

Probst, R., Lonsbury-Martin, B.L., martin G.K. 1991. A review of otoacoustic emissions. Journal of Acoustical Society of America, 89 (5): 2027-2067.

Reshef, Haran I., Attias, J. and Furst M. 1993. Characteristics of click-evoked OAEs in ears with normal hearing and noise-induced hearing loss. British Journal of Audiology, 27 (6): 387-397.

Robinette, M.S. and Fraser, G. 1991. Evoked otoacoustic emissions in a patient with a profound sensori-neural hearing loss. Archives of Otolaryngology and Head and Neck Surgery.

Robinette, M.S. 1992. Clinical observation with transient evoked otoacoustic emissions with adults. Seminars in Hearing, 13 (1).

Robinson, P.M. and Haughton P.M. 1991. Modification of evoked OAEs by changes in pressure in the external ear. British Journal of Audiology, 25 (2): 131-135.

Rossi, G. and Solero P. 1988, Evoked otoacoustic emissions (EOAEs) bone conduction stimulation, Acta Otolaryngologica, 105: 591-594.

Rossi, G., Solero, P. Rolando, M. and Olina M. 1988. Delayed oto-acoustic emissions evoked by bone-conduction stimulation: Experimental data on their origin, characteristics and transfer to the external ear in man. Scandinavian Audiology. 17 Suppl. 29: 1-24.

Ruggero, M.A. Rich, N.C. and Fryman, 1993, Spontaneous and impulsively evoked OAEs: indicators of cochlear pathology. Hearing Research, 10: 283-300.

Rutten, W.L.C. 1980 Evoked acoustic emissions from within normal and abnormal human ears: comparison with audiometric and electrocochelographic findings. Hearing Research, 2: 263-271.

Ryan,S., Kemp, D.T. and Hinchcliffe, R. 1991. The influence of contralateral acoustic stimulation on click evoked OAEs in humans. British Journal of Audiology, 25 (6): 391-399.

Siegel, H.J. 1993. cited in Sigel, H.J. 1994. Ear Canal standing waves and High-frequency sound calibration using otoacoustic emission probes, JASA, 95: 2589-2597.

Siegel, H.J. 1994. Ear Canal standing waves and high-frequency sound calibration using otoacoustic emission probes. JASA, 95, 2589-2597.

Siegel, H.J. 1995. Cross-talk in otoacoustic emission probes. Ear and Hearing, 16 (2), 150-158.

Siegel, H.J. and Kim, D.O. 1982 Efferent neural control of cochlear mechanics ? Olivocochlear bundle stimulation affects cochlear biomechanical non-linearity. Hearing Research, 6(2): 171-182.

Smurzynski, J. and Kim D.O. 1992. Distortion-product and click evoked OAEs of normally hearing adults. Hearing Research, 58 227-240.

Spektor, Z., Leonard, G., Kim, D.O. Jung, M.D. and Smurzynski, J. 1991. OAEs in normal and hearing impaired children and normal adults. Laryngoscope, 101:965-974.

Stevens, J.C., Hutchenson, J., Connell J, Soffin, J.T. 1990 Click Evoked Otoacoustic Emissions in Neonatal Screening, Ear and Hearing, 11(2) : 128-134.

Stevens, J.C. Webb, H.D., Huthinson, J.H., Connell, J., Smith M.F. and Buffin J.T. 1991. Evaluation of Click-evoked OAEs in the newborn. British Journal of Audiology, 25 (1):" 11-15.

Tanaka, Y., Ouchi, T., Arai, Y. and Suzuki, J. 1987. Otoacoustic emissions as an indicator in evaluating inner ear impairments. Acta Otolaryngologica, 103: 644-648.

Tanaka, Suzuki and Tsueno. 1990 Evoked Otoacoustic Emissions in sensorineural hearing impairment. Its clinical implications. Ear and Hearing, 11(2): 134-143.

Thornton, A.R.D. 1993. High rate otoacoustic emission. Journal of Acoustical Society of America, 94: 132-136.

Thornton, A.R.D.,1993. Click evoked otoacoustic emissions: New Techniques and applications. British Journal of Audiology, 27: 109-5.

Thornton, A.R.D., Folkard, T.J., Chambers, J.D. 1994. Technical aspects of recording evoked otoacoustic emissions using maximum length sequences. Scandinavian Audiology, 23 (4): 225-232.

Thornton, A.R.D., KimmL, Kennedy C.R., Caffarelli-Dees, D. A. 1994 Comparison of neonatal evoked otoacoustic emissions obtained using two types of apparatus, British Journal of Audiology, 28: 99-109.

Thornton, A.R.D. and Slaven A. 1993. The effect of stimulus rate on the contralateral inhibition of evoked otoacoustic emissions. Journal of Acoustical Society of America, 34: 132-136.

Thornton, A.R.D., Kimm, L., Kennedy, C.R. and Caffarelli—Dees D. 1993. External and middle ear factors affecting evoked OAEs in neonates. British Journal of Audiology, 27 (5): 319-329.

Thornton, A.R.D., and Norman M. 1993. Frequency analysis of the contra lateral suppression of evoked OAEs by narrow band noise. British Journal of Audilogy, 27 (4) : 281-291.

Tognola, G., Ravazzani, P., Grandori, F.1995. An optimal filtering technique to reduce the influme of low frequency noise on click-evoked otoacoustic emissions. British journal of Audiology, 29(3) : 153-160.

Truy, E., Vevillet, E. Collet, L. and Morgon, A. 1993. Characrteristics of transient OAEs in patients with sudden idiopathic hearing loss. British Journal of Audiology, 27 (6) : 379-387.

Uziel, A., Piron, J.P. 1991. Evoked OAEs from normal new borns and babies admitted to an intensive care unit. Acta Otolaryngologica, Suppl. 482, 85-91.

Van dijk, P. and Wit, H.P. 1987. The occurrence of click-evoked Otoacoustic emissions (Kemp echoes) in normal-hearing ears. Scandinavian Audiology, 16: 62-64.

Wada, H., Ohyama, K., Kobayashi, O., Nawhria, S., Kioke. 1992. Relationship between evoked otoacoustic emissions and middle ear dynamic characteristics. Audiology, 32 (5) : 282-292.

Weir, C.C., Norton, S.J. and Kincaid, G.E., 1984. Spontaneous narrow band otoacoustic signals emitted by human ears: a replication. JASA, 76 (4): 1248-1250.

Williams, E.A., Brookes, G.B. and Prasher, D.K. 1993, Effects of contralateral acoustic stimulation on OAEs following vestibular neuractomy. Scandinavian Audiology, 22 (3) : 197-205.

Wilson, J.P. 1980 a. Evidence for a cochlear origin for acoustic re-emissions, threshold fine-structure and tonal tinnitus Hearing Research, 2: 233-252.

Wilson, J.P. 1980 b. Model for cochlear echoes and tinnitus based on an observed electrical correlate. Hearing Research, 2:527-532.

Wilson, J.P. 1986. Otoacoustic emissions and tinnitus. Scandinavian Audiology, Supplementum, 25: 109-119.

Wit, H.P. and Ritsma, R.J. 1979. Stimulated acoustic emissions from the human ear. Journal of Acoustical Society of America, 66: 911-913.

Wit, H.P. and Ritsma, R.J. 1980. Evoked acoustical responses from the human ear: Some experimental results, Hearing Research, 2: 253-261.

Wit, H.P. Langevoort, J.C. and Ritsma, R.J. 1981. Frequency spectra of cochlear acoustic emissions (Kemp Echoes). Journal of Acoustical Society of America, 70: 437-445.

Zenner, H.P. 1980. Motility of outer hair cells as an active action-mediated process. Acta Otolaryngologica, 105: 39-44.

Zenner, H.P. 1986. Motile response in outer hair cells. Hearing Research, 22: 83-90.

Zenner, H.P. 1993. Possible roles of outer hair cell D.C. movements in the cochlea, British Journal of Audiology, 27 (2) : 73-79.

Zwicker, E. 1983, Delayed evoked otacoustic emission and the suppression by Gaussian shaped pressure impulses. Hearing Research, 11: 359-371.

Zwicker, E and Schorn, K. 1990. Delayed and evoked otoacoustic emission - an ideal screening test for excluding hearing impairment in infants. Audiology, 29(5): 241-251.

APPENDIX

Joint Committee on Infant Hearing 1994 Position Statement.

A. Risk Criteria: Neonates (birth . 28 days)

The risk factors that identify those neonates who are at risk for sensioneural hearing imparimentinclude the folloiwng:

1. Family history of congenital or delayed onset childhood sensiorineural impairment.

2. Congenital infection known or suspected to be associated with sensiorineural hearing impairment such as toxoplasmosis, syphilis, rubella, cytomegalovirus and herpes.

3. Craniofacial anomalies including morphologic abnormalities of the pinna and ear canal, absent philtrum, low hairline, etc., tera.

4. Birth weight less than 1,500 grams (3.3 lbs.)

5. Hyperbilirubinemia at a level exceeding indication for exchange transfusion.

6. Ototoxic medications including but not limited to the aminoglycosides used for more than 5 days (e.g., genatmicin, tobramycin, Kanamycin, streptomycin) and loop diuretics used in combination with aminoglycosides.

7. Bacterial meningitis.

8. Severe depression at birth, which may include infants with Apgar scores of 0-3 at 5 minutes or those who tail to initiate spontaneous respiration by 10 minutes or those with hypotonia persisting to 2 hours of age.

9. Prolonged mechanical ventilation for a duration equal to or greater than 10 days (e.g., persistent pulmonary hypertension).

10. Stigmata or other findings associated with a syndrome known to include sensiorineural hearing loss (e.g. Waardenburg or Usher's Syndrome).

B. Risk Criteria: Infants (29 days - 2 years:

The factors that identify those infants who are at risk for sensorineural hearing impairment include the following:

1. Parent/ caregiver concern regarding hearing, speech, language and/or developmental delay.

2. Bacterial meningitis.

3. Neonatal risk factors that may be associated with progressive sensorineural hearing loss (e.g., cytomegalvirus, prolonged mechanical ventilation and inherited disorders).

4. Head trauma especially with either longitudinal or transverse fracture of the temporal bone.

5. Stigmata or other findings associated with syndromes known to include sensorineural hearing loss (e.g. Waardenburg or Usher's Syndrome.)

6. Ototoxic medications including but not limited to the aminoglycosides used for more than 5 days (e.g., gentamicin, tobramycin, kanamycin, streptomycin) and loop diuretics used in combination with aminoglycosides.

7. Children with neurodegenerative disorders such as neurofibromatosis, myoclonic epilepsy, Werdnig-Hoffman disease, Tay-Sach's disease infantile Gaucher's disease. Nieman-Pick disease, any metachronatic or any infantile demyelinating neuropathy.

8. Childhood infectious diseases known to be associated with sensorineural hearing loss (e.g., mumps, measles).

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