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The Utilization of Transient Evoked Otoacoustic Emissions and Tympanometry in Hearing Screening of Hearing-Impaired Children

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The Utilization of Transient Evoked Otoacoustic Emissions and Tympanometry in Hearing Screening of Hearing-Impaired Children

Juvy Lee

Abstract

In this study 82 subjects aged between 6 to 12 years were included in a hearing screening in Hong Kong school for the Deaf (primary division) There are two parts in the screening, one aspect involved the use of tympanomtery to find out the prevalence of middle ear disorders There were 9 children who failed in the tympanometry No significant differences were noted for gender, age or side of the ear The aspect involved transient evoked otoacoustic emissions (TEOAEs) to determine the number of hearing impaired children with an intact cochlea Two children had TEOAEs that indicated intact cochlear functioning Moreover, distortion-product otoacoustic emissions (DPOAEs) were also administered to confirm the TEOAEs results and obtain more information on specific frequencies One child had bilateral cochlear functions and one child had a clear unilateral cochlear function Clinical implications and future direction of research will be discussed

Juvy Lee

The utility of evoked otoacoustic emissions and tympanometry screening in hearing-impaired children

Introduction

Sensorineural hearing loss can be subdivided into two types: sensory hearing loss and neural loss. Sensory hearing loss originates in the inner ear (cochlea) while the neural hearing loss is caused by disorders in the auditory nerve. The cochlea is responsible in firing nerve impulses upon stimulation. The auditory nerve plays an important role in transmitting information from the cochlear to the brain. However, it is very difficult to distinguish between hearing loss due to cochlear disorder from problems in the auditory nerve. Therefore, sensorineural loss is assumed to originate in the cochlea in most of the cases (Thomas, 1984; Suchfull et al., 1996).

A clinical procedure called Evoked Otoacoustic emissions (EOAEs) is effective in identifying people with hearing loss as no EOAEs can be recorded in individuals with hearing loss greater than 30–40 dB regardless of age and gender (Probst et al., 1991). Moreover, it is also applicable in distinguishing individuals with sensory hearing loss from auditory nerve disorder effectively.

Recently, there have been a number of reports identifying people with severe or profound sensorineural hearing loss with intact cochlear function by using EOAEs (Prieve, et al.; 1991. Katona, et al.; 1993, Laccourrey, et al., 1996; Konradsson, 1996

& Cullington et al., 1998). They reveal that there may be a small population with sensorineural hearing loss actually caused by retrocochlear impairment (i.e., auditory nerve, brainstem or cortex), not the cochlea itself.

In this study, EOAEs screening was carried out in a school for the deaf in Hong Kong. These children are those who have severe to profound sensorineural hearing loss in most of the cases (Yuen, 1997). We are interested in investigating if there is any subject would have presence of EOAEs, which shows that hearing loss is due to auditory nerve disorder instead of cochlear impairment

Hearing mechanism

The auditory system consisted of outer ear, middle ear, inner ear and a portion of nervous system that contributes to auditory sensation and the integration of auditory information with other sensory or motor systems respectively (Glattke, 1978). It is shown in Appendix 1. The outer ear comprises auricle and external ear canal through which sounds can reach the tympanic membrane at the end. Therefore, the outer ear collects and directs sounds into the middle ear. The middle ear begins at the other side of tympanic membrane. It is an air-filled space with three tiny bones. The acoustic energy is transformed from the sounds directed by the outer ear, into mechanical energy and delivered to the oval window of inner ear, i.e. the cochlea.

Since the cochlea is a fluid-filled organ with sensory receptors, movement of the oval window establishes pressure waves in the perilymph of the vestibular duct. They cause distortions of the basilar membrane towards the round window of the tympanic duct (Martini et al., 1995). The sensory receptors that sit on the basilar membrane, i.e., outer hair cells and inner hair cells also move as well. The inner hair cells fire nerve impulses, which travel to the brainstem and central nervous system via the auditory nerve. On the other hand, the wave pressures in cochlear fluids are released through the round window to the middle and outer ear. This is a backward transmission. Therefore, the sound transmissions in the auditory system are a bi-directional, not an uni-directional one (Margolis & Trine, 1997) (Appendix 2).

Forward transmission plays an important role for hearing as it transmits sounds to the cochlea and retro-cochlear system while backward transmission is transmitting sounds that are generated from the cochlea, i.e. OAEs, to the outer ear canal.

What is otoacoustic emission?

Otoacoustic emissions are the sounds that can be recorded in the external ear canal by an insert probe, which is connected to an analyzer. These emissions are inevitable by-product of those processes that are essential to hearing, but not essential to hearing (Norton, 1992). The otoacoustic emissions are made by the active movements of

outer hair cells (Prieve et al., 199; Norton, 1992). These movements set the cochlear fluids in motions and yield energy. Then, the energy is released through the middle ear and transmitted to the outer ear. Since the ear is hermetically sealed with an inserted probe, the energy released through the middle ear cause compressions and rarefactions of the air column in the outer ear. These air column movements are the OAEs (Otodynamics, 1997). The presence of OAEs indicates that the preneural cochlear receptor mechanism, i.e., the pathway from outer ear hair cells that was in the cochlea to the outer ear, are able to respond to sound in a normal way (Kemp et al., 1990).

Otoacoustic emissions are a reliable test as they are measurable in all the individuals with normal and near normal ears (Kemp, et al., 1990). These emissions can be emitted spontaneously or after stimulation. Since the evoked emissions have clinical value, two of types of evoked emissions, i.e., TEOAEs and DPOAEs, are used in identifying the status of cochlea in this study.

Transient otoacoustic emissions (TEOAEs)

TEOAEs are the otoacoustic emissions evoked by tone bursts or clicks. Clicks are broad band noise and they can stimulate the entire cochlea to give responses. A board response spectrum can be recorded in individuals with normal cochlear

functioning. This could also maximize the probability of detecting a response after a brief sampling period (Glattke et al., 1998). On the other hand, tone bursts could only cause a restricted frequency response as it has a narrow frequency range. People with hearing loss greater than 30dB normally do not have TEOAE (Probst et al.,1991 & Stach, 1998).

Distortion products otoacoustic emissions (DPOAE)

Two primary tones that vary in frequency are presented to an ear simultaneously. The normal cochlea responds by producing energy at additional frequencies, i.e., distorted products, which are picked up by the probe. Responses are generated from different areas of the cochlea when the primary-tone frequencies are varied (Martin, 1997). This allows DPOAEs to track precisely the frequency boundary between regions of normal versus impaired outer hair cells (Owens, et al., 1992). However, no response can be measured when the hearing loss is greater than 40 dB HL (Stach, 1998).

Why tympanometry was used?

In order to obtain an accurate TEOAE, normal middle ear functioning is a prerequisite (Owens, 1992, Martin; 1997, Margolis, et al., 1997). This is because conductive

pathology attenuates input stimulus as well as the output TEOAEs. The negative middle pressure caused by middle ear disorder may cause stiffness in the tympanic membrane or displacement of middle ear structures (Robinson, 1993). This can cause a greater attenuation on low frequency emissions of the TEOAEs (Robinson et al., 1991; Owens et al., 1992; Trine, et al., 1993). Any changes in middle ear pressure thus may affect the amplitude, reproducibility, and spectral characteristics of the TEOAEs (Trine et al., 1993) and potentially influence the reliability of the test in turn (Marshall et al., 1997). As a result, there may be an increase in the occurrence of false positive failures (Trine et al., 1993) as some of the otoacoustic emissions are attenuated. However, it is not possible to use OAE measures to separate conductive from sensorineural loss at present. In order to rule out the possibility that the absence of EOAEs was due to conductive component, tympanometry was used before using TEOAEs and DPOAEs.

Moreover, tympanometry is particularly important for children with known sensorineural hearing loss as they are at risk for middle ear disorder and/or more likely to suffer harmful developmental sequelae from the conductive hearing loss that usually accompanies middle ear disorders (AAA, 1997). In addition, the literature about the prevalence of middle ear disorders is rare especially in Chinese population. Therefore, it is valuable to investigate the prevalence of middle ear disorders in

school-age children in Hong Kong by using tympanometry.

Principle of tympanometry

Tympanometry is an objective method for evaluating the mobility of the tympanic membrane and the functional status of middle ear (Northern, 1996). Three tubes in a metal probe are connected to a miniature loudspeaker, a miniature microphone and an air pump seperately. The loudspeaker emits a low frequency pure tone while the microphone picks up the sound in the ear canal. The air pump pumps in and out the air in the external ear canal to create either positive or negative air pressure.

Method

Subjects

Eighty-one students from Hong Kong School for the Deaf participated in the study. The children were in primary division, ranging from 6 to 12 in age. The subjects were recruited by sending a consent form to the school describing the study. This form was then sent to the parents or guardians. Children were volunteers and no selection of subjects was carried out regarding history or ear status. Consent forms were received before the hearing screening.

The subjects received otoscopic examination followed by tympanometry and TEOAEs. DPOAEs was used only in those subjects with the presence of TEOAEs.

For those subjects who were found to have possible outer or middle ear disorders received a follow up in 4 to 6 week's time according to the screening guidelines recommended by ASHA (1990). Both of the tympanometry and OAEs were administered during the follow up.

The screening instrument

Otoscopic examination

Otoscopic examination was carried out before tympanometry and EOAEs in order to rule out any observable outer and middle ear disorders.

Tympanometry

Immitance measurement was be administered by using an automatic tympanometer (GSI-37). This instrument used a 226± 3% Hz probe tone with 85.5 dB SPL intensity. The pressure was swept from positive to negative with the sweep rate was 600 daPa/sec except near tympanogram peak where sweep rate slows to 200 daPa/sec. The pressure ranged from +200 to –400 daPa. It is calibrated before and after the screening. The tympanograms were printed out after the administered tympanometry on both ears of each subject

The subjects' tympanograms were categorized into three basic shapes. Type A

tympanogram described a normal middle ear pressure, as the peak was located between –150daPa and +100daPa. If the no clear peak is measured, then, it was a type B tympanogram. Lastly, a type C tympanogram indicated the presence of significant negative middle ear pressure with a distinct peak less than -150daPa (Appendix 3).

The statistical analysis was performed by using the Fisher Exact Test (two-tail). A p value of less than 0.05 was considered to be significant. The number of ears was used as the unit in the analysis.

Transient otoacoustic emissions (TEOAE)

TEOAE instrument consisted of a measuring probe containing a loudspeaker to stimulate the ear, a microphone to record all the sounds in the ear canal, and a signal separating process that can discriminate between sounds emerging from the cochlea and other sounds (Kemp, 1997). The probe fitted with a disposable plastic tip is inserted into the ear canal. The probe sealed the ear canal tightly to maximize TEOAE collection and exclude ambient noise (Kemp, 1997). The system and probe were calibrated before testing using standard procedures.

The measurement was carried out in a sound attenuated room with the background noise at 45dBA because the threshold of the TEOAEs was critically dependent the

noise in the recording environment (Rhoades et al. 1998; Glattke et al., 1998). The measurement system used was the Otodynamic ILO 88/92 Analyzer hardware and software (version 5.6). The Quickscreen mode was used and 100 sets of click stimuli were presented at a rate of 50Hz, target intensity 80± 3 dB SPL by using a non-linear mode. The electric pulse duration applied to the transducer was 80 μs. The noise rejection level was set at 47 dB SPL. When the noise was above the rejection level, the sampling will stop until the noise level was below the noise rejection level again. The linear growth component of the emissions was rejected as artifact. Only the residual non-linear component of non-linear growth was considered as true cochlear derived emissions. The responses were stored in two separate memory buffers by sampling. ILO software generated a Fast Fourier Transformation (FFT) between 0 to 6 kHz with a resolution of 50Hz. The Fast Fourier Transformation of the otoacoustic emission was used to find reproducible peaks in the emission spectra.

All the recordings are stored on floppy disc. The measured signals were considered as true responses if they are at least 3dB SPL above the average noise level and their reproducibility is above 50%.

Distortion products otoacoustic emissions (DPOAE)

The instrumentation was the same with measuring TEOAE. Two continuous sounds with a frequency ratio of f2/f1 at 1 22 were presented by a probe. The same intensity of f1 and f2 of 70 dB SPL in the external ear canal was used. DPOAE intensity at 2f1-f2 will be measured form 0 5kHz to 6 kHz in an intensity range between –10 and 30dB SPL. The measured signal was significantly different from the background noise if it was at least 3 dB SPL above the average noise level

Results

TEOAEs and DPOAEs

TEOAEs were recorded in two students (three ears) out of 81 subjects. DPOAEs were administered to these two students to confirm the presence of TEOAEs as well as to get more information on specific frequencies.

Student 1 has been found to have the presence of TEOAEs and DPOAEs bilaterally. The stimuli level of TEOAEs was 92 dB peak SPL in left and 93 dB peak SPL in right ear. They were measured in all frequency components except 2.4 kHz in the right ear. Higher amplitudes were recorded at high frequency components. However, only high frequencies (above 3 kHz) were measured in DPOAEs. Also, there was an increase in amplitude towards high frequencies. TEOAEs were recorded in the left ear with a wide frequency range (about 1kHz to 6kHz) The highest amplitude

measured was 20dB at around 4KHz. DPOAEs were measured in the frequency range of 2 kHz to 6kHz. The highest amplitude was measured at about 6kHz with approximately 20dB SPL (Appendix 4).

The other subject, Student 2, was found to have clear TEOAEs and DPOAEs on the right ear which are restricted in high frequencies only (above 3 kHz) with a stimulus level of 84 dB peak SPL. The amplitudes were unusually large, up to 27dB at 4 kHz in TEOAEs. A similar frequency range was observed in DPOAEs with approximately 5 to 15 dB in amplitude (Appendix 5).

Case Studies

Case 1

Student 1 was identified to have hearing loss around one year of age. He was diagnosed to have severe bilateral sensorineural hearing loss. Birth, medical and family histories were insignificant for hearing impairment. A detail audiological evaluation was offered to the Student 1 in order to determine the exact site of disorder. The audiological evaluation results as followed. Student 1 had severe sensorineural hearing loss bilaterally. Word recognition scores were poor in both aided and unaided condition, which were 30% and 20%, respectively. Impedance tympanometry revealed Type A tympnograms bilaterally. Clear TEOAEs and

DPOAEs were recorded in both ears. No synchronous responses have been recorded for either ear by using ABR with maximum stimulus level. Cochlear responses were observed that reliably inverted with reversal of stimulus polarity. No discernable MLR responses were obtained for both ears. Clear Late Responses (N1-P2) were observed for both ears with either ispi- or contra- stimulation. All responses were with similar N1 and P2 latencies, and were within normal limits.

The Student 1's mother reported that her child did not like to use the hearing aids as the amplified sounds delivered by the hearing aids were too loud. Student 1 seldom put on hearing aids at home. During the audiological evaluation, it was found that the student relied heavily on speech reading.

Case 2

Student 2 was identified as having hearing impairment when he was about 17 months old in the Pamala Youde Polyclinic. Birth, medical and family histories were insignificant for hearing impairment. Pure tone audiometry showed that he had a profound sensorineural hearing loss in the right ear and a moderate sensorineural hearing loss on the left ear. Weber test lateralized to the left at 1kHz while lateralizing to the right at 0.5kHz. Speech recognition scores for monosyllables were very poor (10% for with aids and 20% without aids). Impedance tympanometry

revealed Type A tympnograms bilaterally. Clear TEOAEs and DPOAEs were recorded only in the right ear. He had no acoustic reflexes upon either ipsilateral or contralateral stimulation.

No synchronous responses have been recorded for either ear from ABR. Middle Latency Responses (MLR) testing was completed with high stimuli intensity. Clear Na, Pa, Nb and Pb responses were observed for both ears with either ipsi- or contra-stimulation. All responses were with comparable latencies, which were within normal limits. Repeatable N1-P2 responses from Late Response (N1-P2) testing were observed for both ears with either ipsi- or contra- stimulation. All responses were with comparable N1 and P2 latencies, however, they were all significantly delayed.

The same complain about the loudness of the hearing aids also reported from the mother of Student 2. She reported that her child could not tolerate the amplified sounds delivered from the hearing aids. Student 2 did not use the hearing aids at home unless doing homework.

Sininger et al. (1995) and Starr et al. (1996) suggest some symptoms that always seen in presumed auditory neuropathy such as: having mild to moderate hearing loss, absent to severely abnormal ABRs to high level stimuli, presene of EOAEs that do not suppress with contralateral noise, poor word recognition, absence of acoustic reflexes to both issilateral and contralateral tones at 110 dB HL and absence of

masking level differences (MLDs). When comparing the audiological evaluations of these two students with above symptoms, we found that they are consistent with the symptoms of having auditory neuropathy. Student 1 had elevation of auditory thresholds, absence of ABRs to high level stimuli. The absence of ABR suggestss that patient is likely to have cochlear or auditory nerve disorder or both. Moreover, there was presence of Late latency responses only but not the ABRs and MLR. It revealed that cortical function was probably normal but not inner ear/brainstem function. However the presence of EOAEs in both ears ruled out the possibility in cochlear damage. It revealed that the student might have auditory neuropathy and brainstem disorders bilaterally.

On the other hand, Student 2 had an increase in hearing threshold, presence of EOAEs, absence of ABRs and acoustic reflexes at high stimuli level, and poor word recognition ability on the right ear. It revealed that the patient is likely to have cochlear or auditory nerve disorder or both. Moreover, only MLR without Long latency responses suggested that normal brainstem function is likely but not normal inner ear/brainstem or cortical function. Since the EOAEs were recorded in the right ear, the student might have unilateral auditory neuropathy on the right ear. He might also have cortical dysfunction.

Tympanometry

There were 81 respondents, 95% of the total sample pool, recruited in the study. The result of tympanometric screening is shown in Table 1. Among the 144 tested ears, 93.06% of them passed the screening criteria, while 6.94% failed. The distribution is shown in Fig.1. Nine subjects failed in the first tests and five of them remained having abnormal tympanograms in the follow up. Those subjects who failed in tympanometry twice were recommended to have otological referrals as they had higher risk in having middle ear disorders. 6 ears were reported with type B tympanogram while 4 with type C were recorded.

Table 1. Tympanometry screening results

Tympanometry	Number of ears	Percentage (%)
Type A	134	93.06
Type B	6	4.17
Type C	4	2.78
Total	**144**	**100**

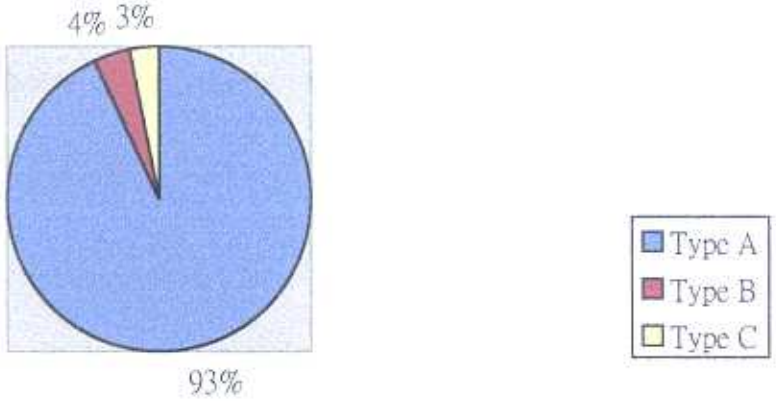

Fig. 1. Percentage in different types of tympanogram

3 females but 6 males failed in the screening. Although the failure rate in male was slightly higher than female, however, no significant difference in gender was noted ($p= 0.515$). The incidence of the left and right ears the difference was also not significant ($p= 0.331$).

The children were divided into two groups. Children aged from 6 to 8 were in the younger group while the older group was consisted of children aged 9 to 12. The differences found in age group was also not statistically significant ($p=0.270$). The tympanometry failure rate by age is shown in Figure 2

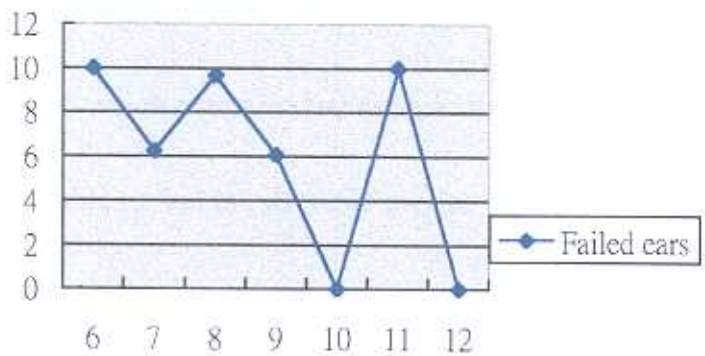

Fig. 2. Tympanometry failure rate by age

When looked at the incidence of the middle ear across the age range, no inverse relationship of age with the incidence rate was observed. This contrasted with the previous study reported by McPherson (1991).

Discussion

Evoked otoacoustic emissions (EOAEs)

In the present study, two children were identified to have possible auditory neuropathy as they had TEOAEs and DPOAEs but without ABRs Noteworthiness was the use of high stimuli intensity of TEOAEs (about 90 dB SPL peak), especially in Student 1. Such high stimulus intensity might increase the stimulus artifact problem (Kemp et al , 1990). However, we have confidence in the validity of the responses because DPOAEs were also recorded at 70 dB SPL, our target intensity level, for both Student 1 and Student 2. Moreover, the recorded DPOAEs in both students were above background noise more than 3 dB, which revealed that they were true responses. The linear response rejection method in the software would reject the artifacts, which minimized stimulus artifact problems (Kemp et al., 1990).

It is invaluable to find out the more precise causes of hearing loss in hearing-impaired children since this may be a crucial factor in designing the rehabilitation program Hearing-impaired children with cochlear impairment will very often benefit from the use of hearing aid(s) (McCandless et al., 1979) and may be suitable for an aural/oral educational program. However, children with retrocochlear hearing loss do not benefit from amplification by using hearing aids (Sininger et al., 1995). Moreover, McCandless et al., (1979) reported that hearing-impaired people with neural- or central-type lesions have a worse speech discrimination with aids than without them. This demonstrated that people with retrocochlear hearing loss might not benefit from

using hearing aids. Hearing aids are inappropriate for these individuals because there are greater inherent distortions of the auditory environment in those with retrocochlear loss (McCandless et al , 1979) This leads them profit less from hearing aids as a result. Moreover, the hearing aid(s) will amplify sounds and send the amplified sounds to a normal cochlea in such cases (Hood et al , 1994) This will not assist the disordered nerve or brain area to process such sounds better. On contrary, they will damage normal cochlear structures (Sininger et al , 1995). Therefore, the benefit of using hearing aids is greatly determined by the site of disorders.

In the present study, two of the hearing-impaired students showed that they might have auditory neuropathy. If they have auditory neuropathy, the rehabilitation methods may have to be reconsidered. It is because these students may not be benefited from using hearing aids anymore. Hood et al. (1996) and Sininger et al. (1995) suggest that using a low-gain FM system inside the classroom would be useful. This device improves the signal-to-noise ratio, and gives an advantage of the signal over the noise. Therefore, children can receive clear messages from the teacher regardless of physical distance. Personal FM system instead of hearing aid is also recommended for these

Moreover, speech trainers are recommended for these children in auditory training at

school because they can amplify speech to each ear separately with very little distortion even at high intensity (Tweedie, 1987). In addition, some options suggested by Hood & Berlin (1996) were; giving extra visual support to supplement the auditory stimuli simultaneously, keeping the sentence short and simple, gaining the child's attention before talking to them, and monitoring the child's comprehension of the message. On the other hand, augmentative communication such as manual communication and speech reading training may have to be considered (Sininger et al., 1995). Cued speech may also be useful in facilitating the children to acquire speech, too. However, any rehabilitation strategies used should be fit to the individual child and carefully evaluated—in light of wide variability in the performance of children with auditory neuropathy.

Tympanometry

There were 9 students failed in first tympanometry screening. Among them 5 out of nine remained fail in the follow up tests. Spontaneous recoveries of middle ear disorder were shown in those five subjects. Therefore, tympanometry can be a useful tool to monitor the progress of infected ears (Owens et al., 1992). Moreover, 4 subjects out of nine had spontaneous recovery in possible middle ear pathology. Therefore, referral was only given to those who failed twice in tympanometry in 4 to 6 weeks' time, which could greatly avoid over-referral (Asha, 1990). It could also

differentiate those individuals with transient or self-limiting episodes from those with chronic middle ear effusion (Roush et al., 1997). In all, this can help in reallocating and utilizing limited resources wisely.

In reviewing the literature, a number of scholars have found that there is an inverse relationship between the incidence of middle ear disorders and age (Stool et al, 1980, McPherson, 1991; Brookhouser, 1993). However, the present study contradicts the result. It may due to age range differences among the studies. In previous studies, the subjects were from kindergarten to junior schools. Report from National Center for Health Statistics (1973) cited in Asha (1985) shows that the otitis media is the most frequently in children between birth and age 2. However, incidence of middle ear pathology declines markedly after 6 to 7 years of age. Since the age range of the subjects in the present study were from 6 to 12, which was out of the peak incidence, the inverse relationship in the incidence of middle ear disorders with age was not shown.

Porter studied the incidence of middle ear disorders in hearing impaired children in 1974. When comparing the failure rate in deaf children aged 6 to 10, 7% in the present study with 23% cited in his study, an enormous difference is noticed. This huge discrepancy may due to genetic differences suggested by Tong et al., (1997) quoted in Rushton et al. (1997). Rushton et al. (1997) found that Chinese children

had a significantly lower prevalence than Caucasian children.. However, when comparing the 1.95% prevalent rate noted in a group of six-year old local students with normal hearing (Rushton et al., 1997), the incidence of possible middle ear disorder in the hearing-impaired children was high. The possible reason might be related to the season that the screening was carried out. The study was conducted in December, which was winter in Hong Kong. Some children might have influenza, which affect the middle ear functioning. This might lead them to fail in the tympanometry. Moreover, only a small sample size, which might not be representative, in each group was extracted.

Middle ear disorders can cause conductive hearing loss, which lowers the hearing threshold from 20 to 40dB (Brookhouser, 1993). Although the hearing loss caused by the middle ear infections is usually temporary, it may lead to permanent hearing impairment if it leaves untreated. The severity of hearing loss may increase. Moreover, Ruben and Math (1978) cited in Brookhouser et al. (1993) suggest that additional amplification is required to overcome as little as a 20 dB conductive hearing loss in children with varying degrees of preexisting sensorineural hearing loss. If the conductive hearing loss persists, a child will not get full benefit from using hearing aids for their residual hearing (Rood et al., 1981). This will deprive language experience and result in some difficulty acquiring speech and language

(Asha, 1985). This may also attribute to the difficulties in learning, as children cannot receive all the messages through hearing. Since it is a critical period for educational, social, speech and language development, tympanometry is highly recommended in routine hearing screening in a deaf school setting.

Clinical Implications

Evoked otoacoustic emissions is an effective tool in assessing the cochlear status mentioned in the present study. It can also distinguish individuals with cochlear damage versus auditory nerve disorder within a short testing period (total screening time was approximately 12 minutes per child). After identifying children who are having sensory hearing loss from neural loss, one may determine the rehabilitation approach for the hearing-impaired children. On the other hand, the use of TEOAEs together with DPOAEs could crosscheck the results in making a diagnosis. The validity of the responses recorded has a higher confidence level.

In present study, we also find that the prevalence of middle ear disorders is higher in hearing impaired children than in normal subjects. The subjects who failed in the tympanometry did not notice plausible middle ear disorder Therefore, it is highly recommended to use tympanometry as a routine hearing health screening tool in schools for hearing-impaired children. Moreover, it is a useful tool to monitor the

progress of infected ears.

Limitations

Two subjects were identified to have an intact peripheral auditory system as TEOAEs and DPOAEs in the present study. A detailed audiological assessment was performed to determine the site of the disorder. The results revealed that these children might have auditory neuropathy. However, we cannot draw a conclusion about the cause of the hearing impairment at this stage because the presence of a space-occupying tumor has not been ruled out. Therefore, radiological examination such as computed tomographic (CT) scan or magnetic resonance imaging (MRI) is recommended. Moreover, contralateral suppression of otoacoustic emissions can be administered to indicate the status of afferent-efferent connection. There is no afferent-efferent disconnection in patients with cochlear disorder (Sininger et al., 1995). It is expected that our two subjects have a disconnection in this pathway, if auditory neuropathy is present.

On the other hand, the restricted in subject age range, carried out the screening in only one season and small sample size would affect the results from tympanometry screening.

Directions in Future research

There were approximately 10 cases have been reported in the literature of OAEs in

the presence of severe or profound hearing impairment in the world wide (Cullington & Brown, 1998). However, the prevalence of auditory neuropathy might be higher than what we think as two children were identified to have possible auditory neuropathy in such a small sample size. Therefore, it is invaluable to investigate the prevalence of auditory neuropathy in our community. On the literature suggests that hearing aids are not useful for individuals with auditory neuropathy. The treatment and management for these individuals are still unclear (Sininger et al., 1995). If the incidence of auditory neuropathy is not rare, further research on the rehabilitation for children with auditory neuropathy is important because they have unmet communication needs. Otherwise, their hearing impairment may hinder their communication in future. Moreover, research on the prevalence of middle ear disorder in hearing-impaired children is rare. Since the prevalence in Asian may be very different from that in Western countries, it is valuable to build up a database for Asian children.

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Appendix 1

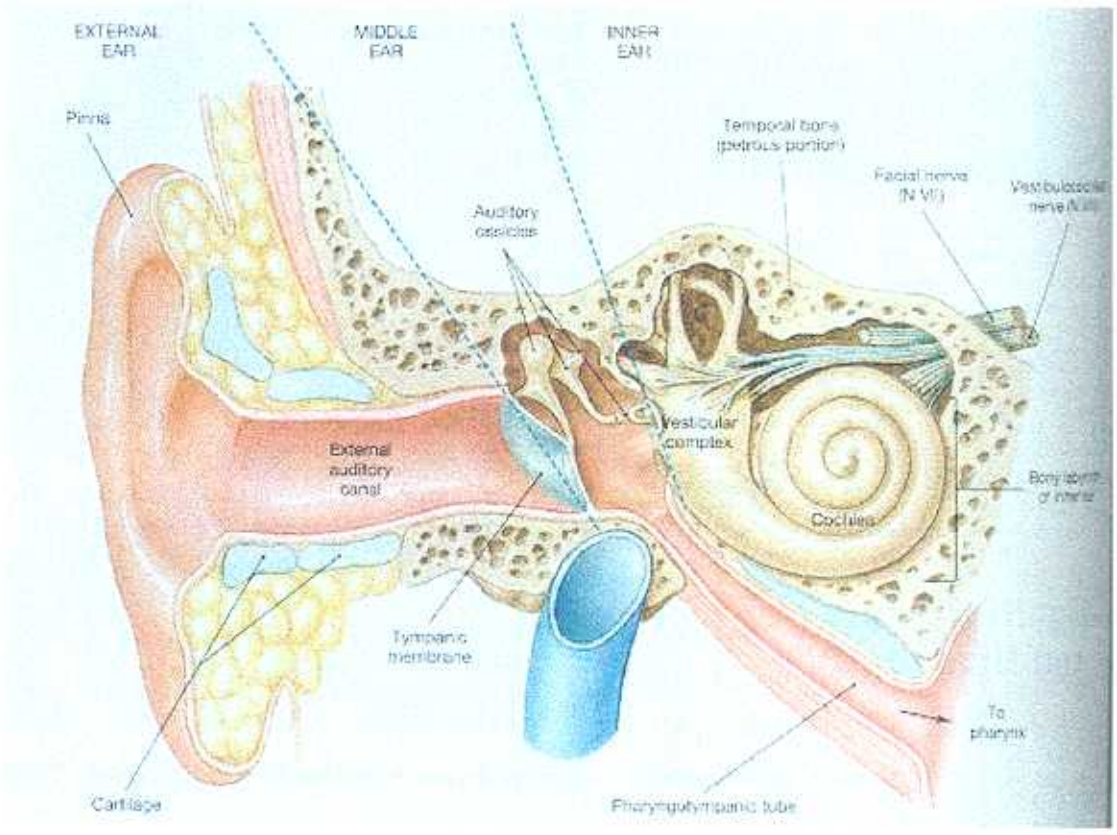


Fig.1. Anatomy of the ear. (Martini et al., 1995)

Appendix 2

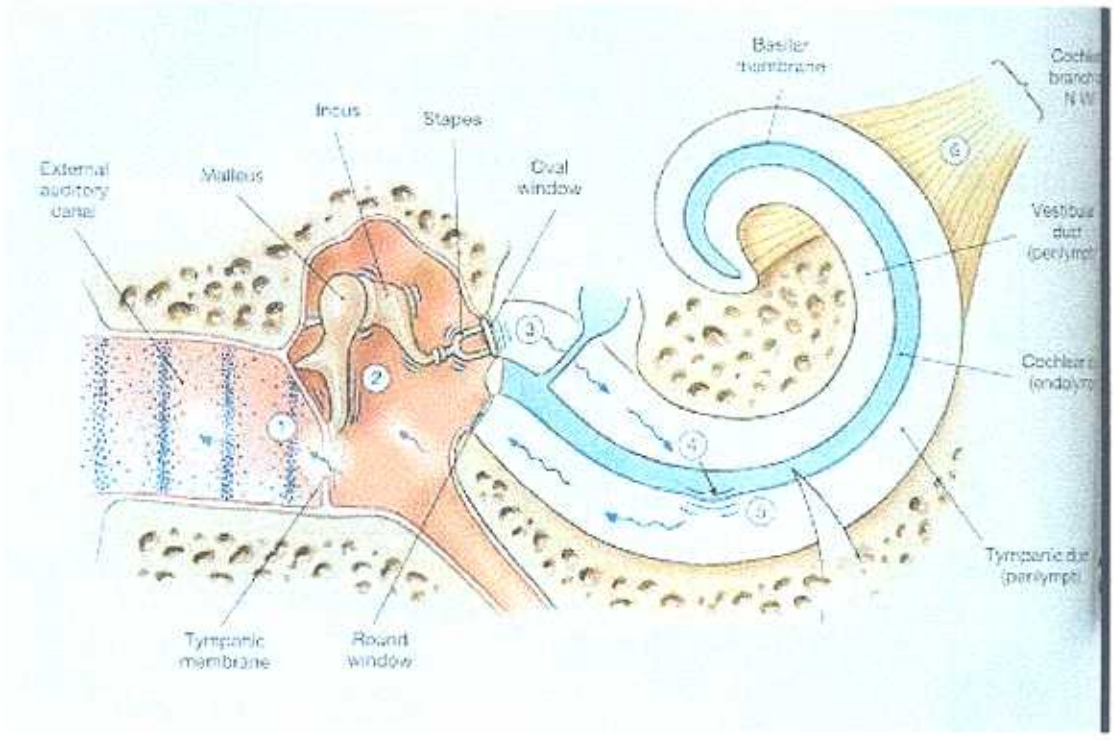


Fig. 2. Bi-direction transmissions of sounds in auditory system (Martini et al., 1995)

Appendix 3

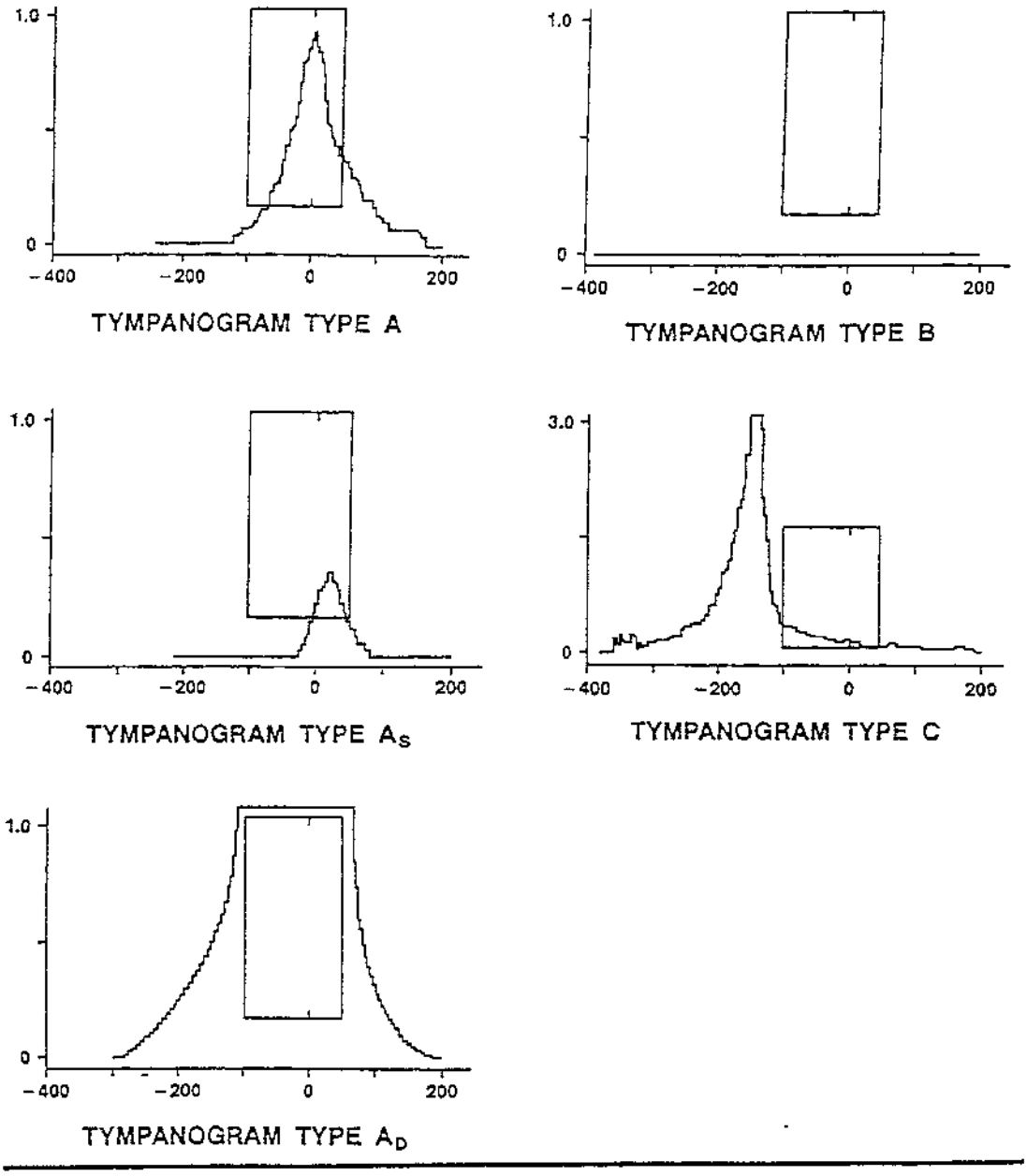
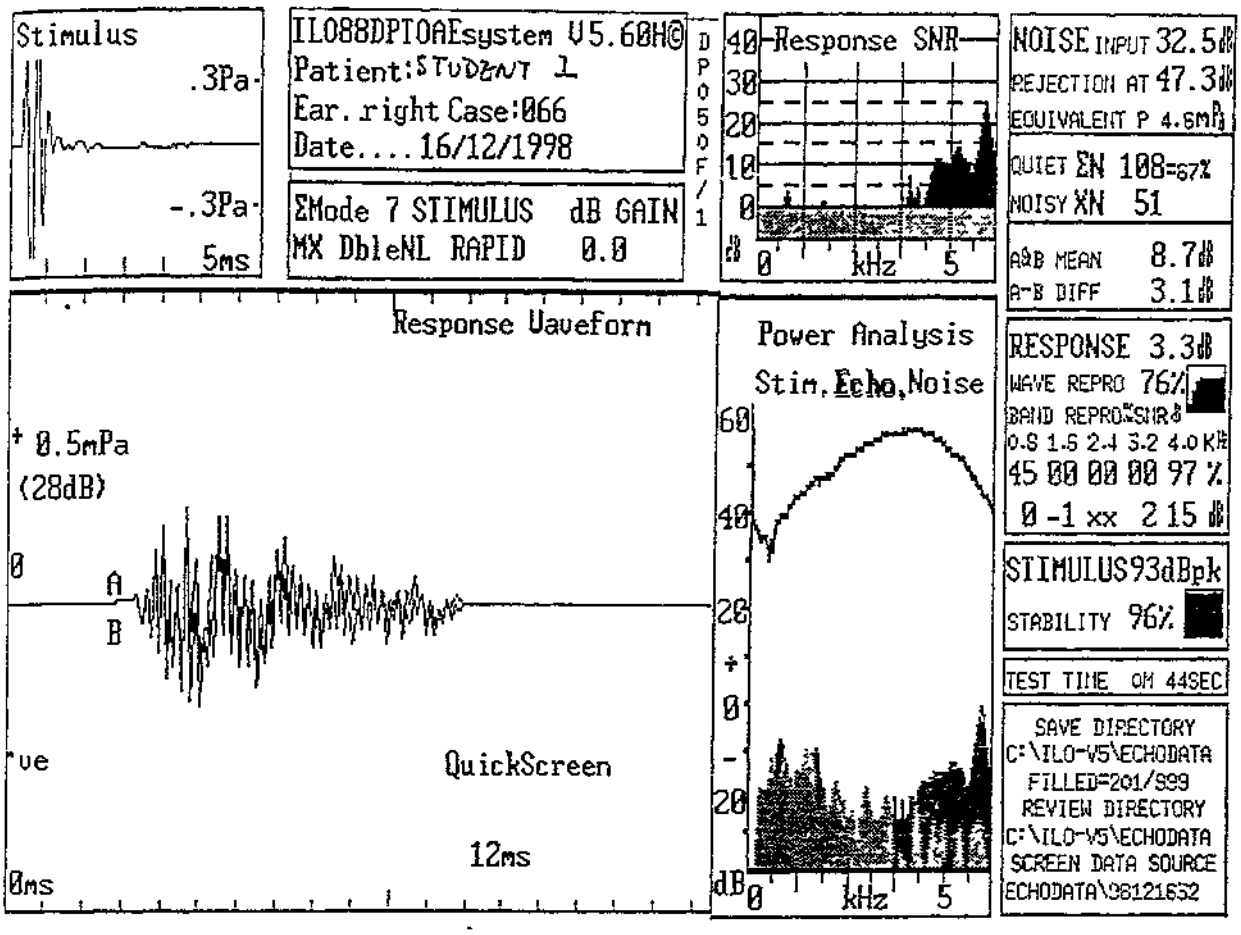


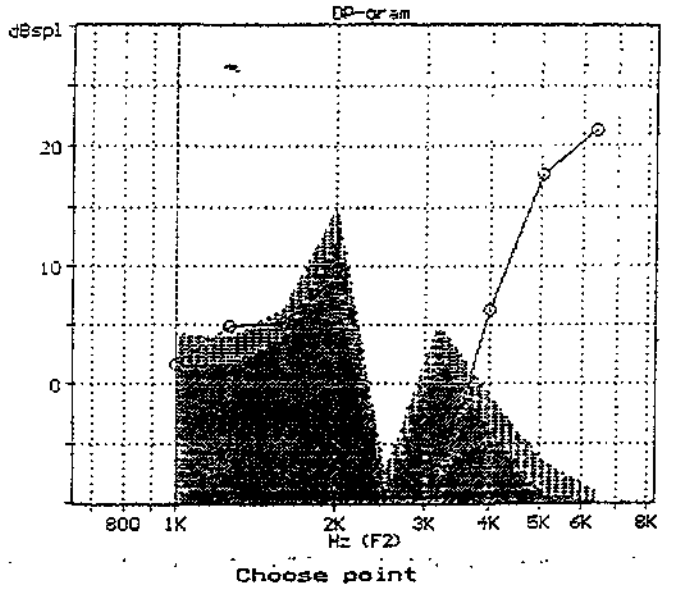
FIGURE 5-1. Jerger's classification of tympanograms. Type A patterns demonstrate normal middle ear pressure: type B tympanograms are associated with nonmobile tympanic membranes; type C tympanograms show significant negative middle ear pressure.

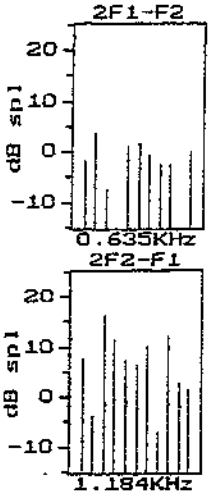
Northern, J L, 1996

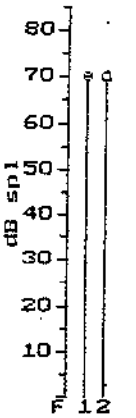
Appendix 4


Stimulus
.3Pa
-.3Pa
5ms
ILO88DPTOAEsystem V5.60H©
Patient:STUDENT 1
Ear.right Case:066
Date....16/12/1998
ΣMode 7 STIMULUS dB GAIN
MX DbleNL RAPID 0.0
D P O S O F / 1
Response SNR
40
30
20
10
0
dB
0 kHz 5
NOISE INPUT 32.5dB
REJECTION AT 47.3dB
EQUIVALEINT P 4.6mPa
QUIET ΣN 108=67%
NOISY XN 51
A&B MEAN 8.7dB
A-B DIFF 3.1dB
RESPONSE 3.3dB
WAVE REPRO 76%
BAND REPRO%SNR
0.8 1.5 2.4 3.2 4.0 KHz
45 00 00 00 97 %
0 -1 xx 2 15 dB
STIMULUS93dBpk
STABILITY 96%
TEST TIME 0M 44SEC
SAVE DIRECTORY
C:\ILO-V5\ECHODATA
FILLED=201/999
REVIEW DIRECTORY
C:\ILO-V5\ECHODATA
SCREEN DATA SOURCE
ECHODATA\38121652
Response Waveform
+ 0.5mPa
(28dB)
0
A
B
ve
QuickScreen
12ms
0ms
Power Analysis
Stim, Echo, Noise
60
40
20
+
0
-
20
dB
0 kHz 5

F1= 818Hz F1=70.2dBspl 2F1-F2= 1.6dBspl S/N= 0.1dB(86%), =-2.6dB(97%)
F2=1001Hz F2=69.4dBspl 2F2-F1= 6.5dBspl S/N=-6.4dB(86%), =-9.4dB(97%)
F2/F1=1.22 Sum No.=21/48 Test time=67s DP phase(φ)= 2.5°
Recalled ->STUDENT I .DPG

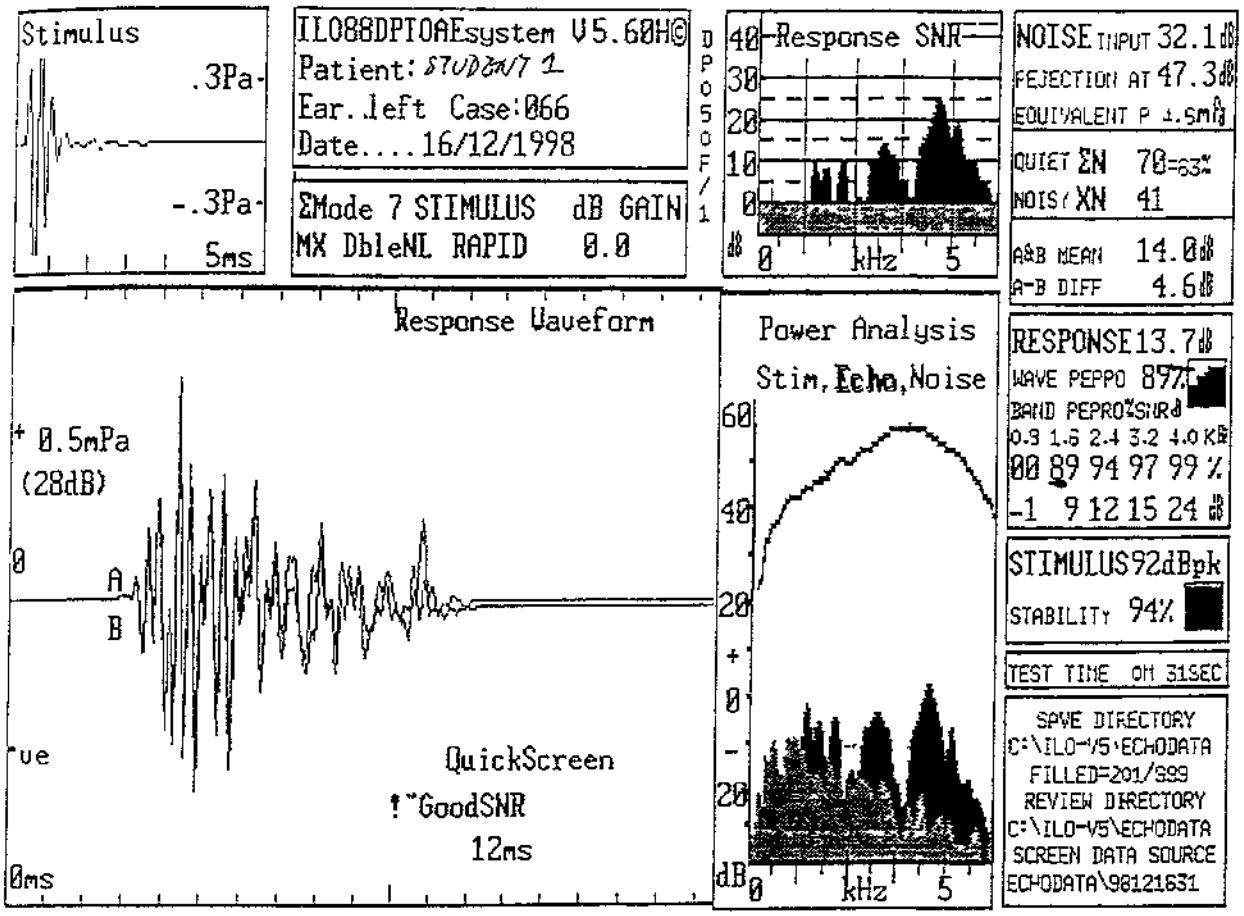




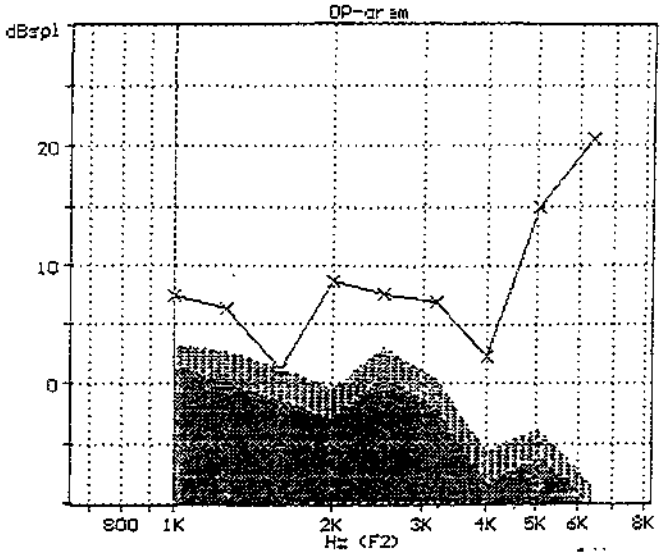


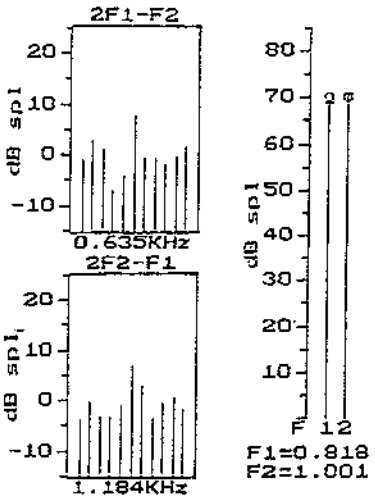
F1=0.818
F2=1.001

Choose point


Stimulus
.3Pa
-.3Pa
5ms
ILO88DPIOAEsystem V5.60H©
Patient: STUDENT 1
Ear.left Case:066
Date....16/12/1998
ΣMode 7 STIMULUS dB GAIN
MX DbleNL RAPID 0.0
DPOAE/1
Response SNR
40
30
20
10
0
dB
0
kHz
5
NOISE INPUT 32.1dB
REJECTION AT 47.3dB
EQUIVALENT P 4.5mPa
QUIET ΣN 70=63%
NOISY XN 41
A&B MEAN 14.0dB
A-B DIFF 4.6dB
Response Waveform
+ 0.5mPa
(28dB)
0
A
B
-ve
QuickScreen
!"GoodSNR
12ms
0ms
Power Analysis
Stim, Echo, Noise
60
40
20
+
0
-
20
dB
0
kHz
5
RESPONSE13.7dB
WAVE REPRO 87%
BAND REPRO%SNRdB
0.8 1.6 2.4 3.2 4.0 KHz
80 89 94 97 99 %
-1 9 12 15 24 dB
STIMULUS92dBpk
STABILITY 94%
TEST TIME 0M 31SEC
SAVE DIRECTORY
C:\ILO-V5\ECHODATA
FILLED=201/999
REVIEW DIRECTORY
C:\ILO-V5\ECHODATA
SCREEN DATA SOURCE
ECHODATA\98121631

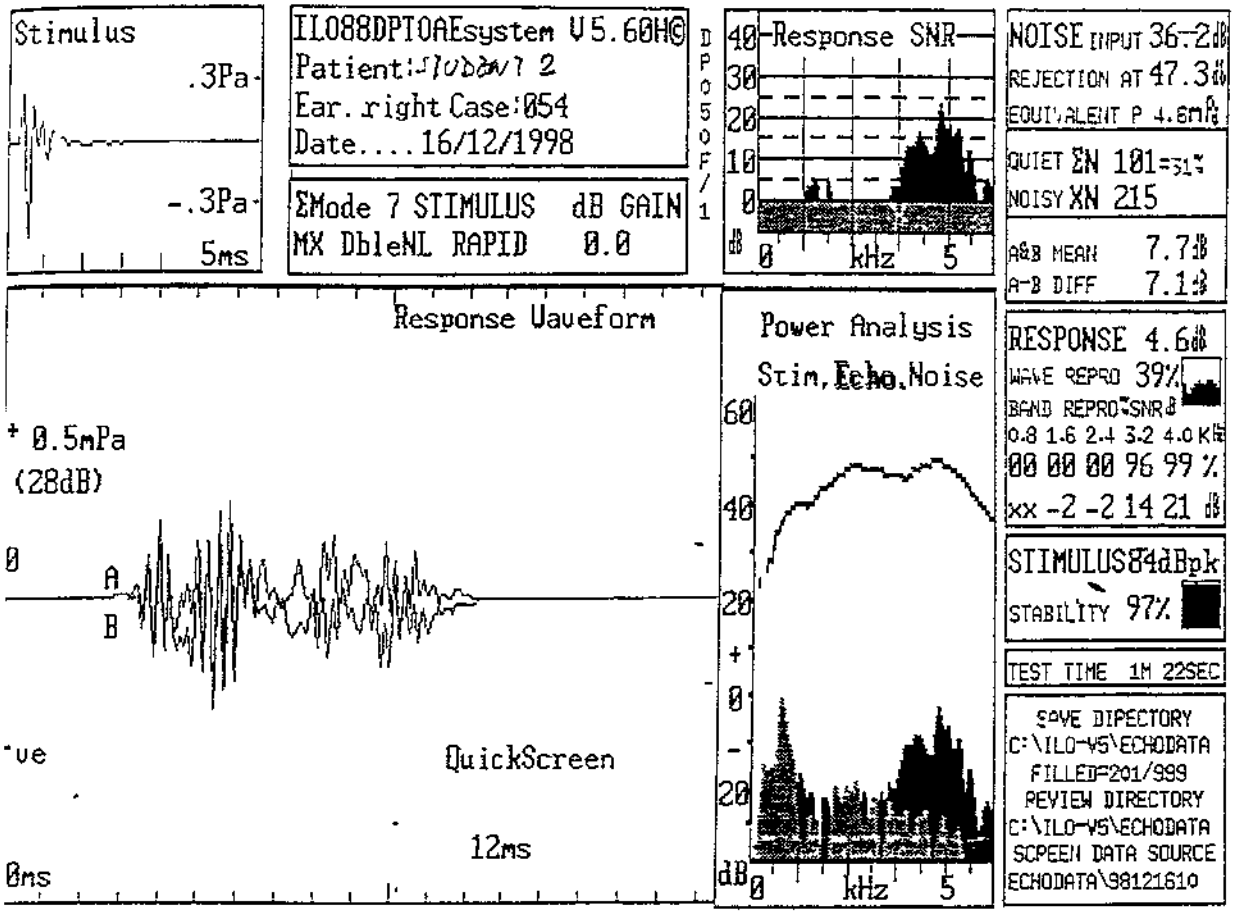
F1= 818Hz F1=68.8dBspl 2F1-F2= 7.5dBspl S/N= 6.0dB(86%), = 4.2dB(97%)
F2=1001Hz F2=70.8dBspl 2F2-F1= 6.7dBspl S/N= 5.9dB(86%), = 4.3dB(97%)
F2/F1=1.22 Sum No.=43/48 Test time=67s DP phase(φ)= -25.1°
Recalled ->STUDENT1.DPG





Choose point

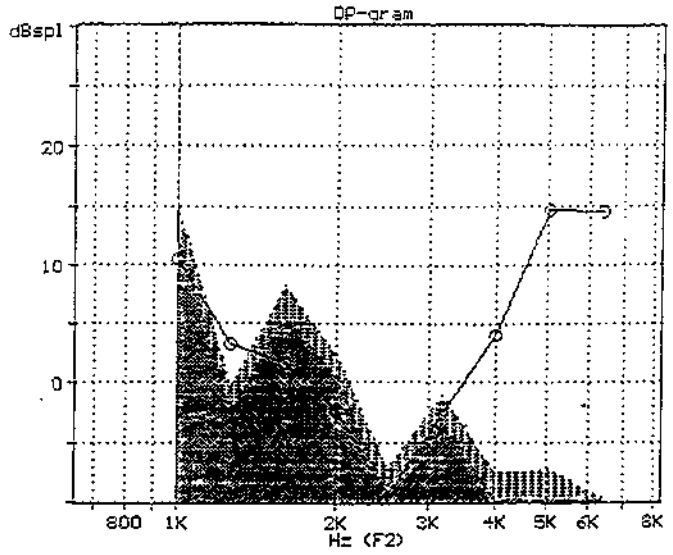
Appendix 5


Stimulus
.3Pa
-.3Pa
5ms
ILO88DPTOAEsystem V5.60H©
Patient:
Ear. right Case:054
Date....16/12/1998
ΣMode 7 STIMULUS dB GAIN
MX DbleNL RAPID 0.0
DPOSOF/1
Response SNR
40
30
20
10
0
dB
0 kHz 5
NOISE INPUT 36.2dB
REJECTION AT 47.3dB
EQUIVALENT P 4.6mPa
QUIET ΣN 101=31%
NOISY XN 215
A&B MEAN 7.7dB
A-B DIFF 7.1dB
RESPONSE 4.6dB
WAVE REPRO 39%
BAND REPRO%SNRdB
0.8 1.6 2.4 3.2 4.0 KHz
00 00 00 96 99 %
xx -2 -2 14 21 dB
STIMULUS84dBpk
STABILITY 97%
TEST TIME 1M 22SEC
SAVE DIRECTORY
C:\ILO-V5\ECHODATA
FILLED=201/999
REVIEW DIRECTORY
C:\ILO-V5\ECHODATA
SCREEN DATA SOURCE
ECHODATA\98121610
Response Waveform
+ 0.5mPa
(28dB)
0
A
B
ue
QuickScreen
12ms
0ms
Power Analysis
Stim, Echo, Noise
60
40
20
+
0
-
20
dB
0 kHz 5

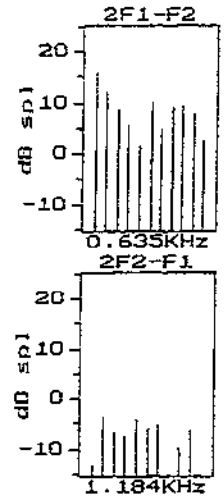
22 MAR 2006

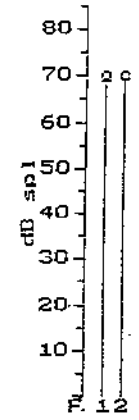
vS | -IL092(F)-Otoacoustic Distortion Product Analyser-12/17/98©TOdynamics

F1= 818Hz F1=68.8dBspl 2F1-F2= 10.4dBspl S/N=-2.0dB(86%), =-4.5dB(97%)
F2=1001Hz F2=69.4dBspl 2F2-F1= -5.8dBspl S/N=-1.3dB(86%), =-4.0dB(97%)
F2/F1=1.22 Sum No.=17/48 Test time=67s DP phase(ϕ)= -94.7°
Recalled -> STUDENT 2 .DPG



Choose point





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F2=1.001