CLICK EVOKED OTOACOUSTIC EMISSIONS IN DIABETIC PATIENTS

Thesis
submitted in partial fulfilment for
Master Degree in Audiology

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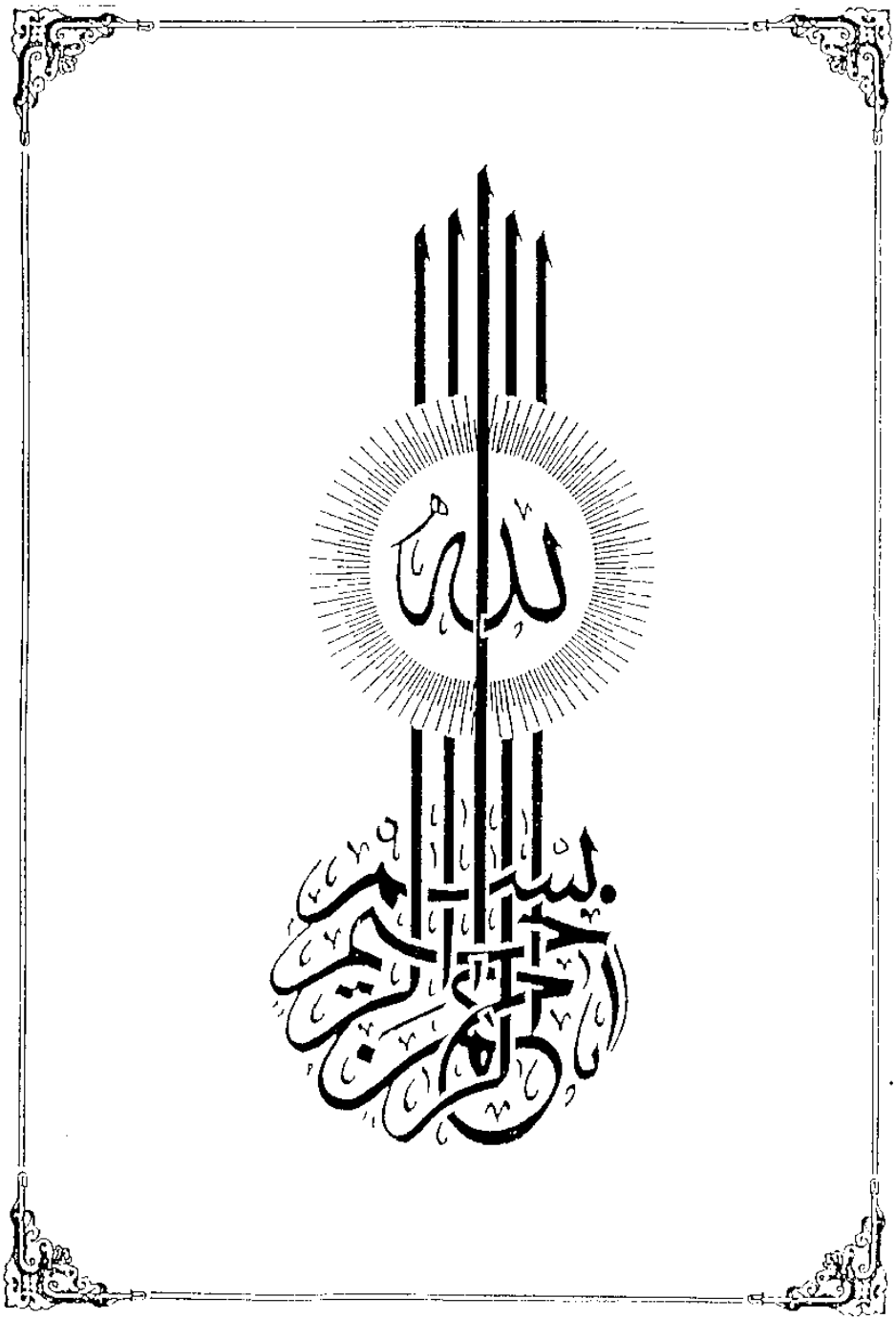
1994

بسم الله الرحمن الرحيم

سُبْحَانَكَ لَا عِلْمَ لَنَا إِلَّا مَا عَلَّمْتَنَا

إِنَّكَ أَنْتَ الْعَلِيمُ الْحَكِيمُ

سورة البقرة - آية ٣٢



Acknowledgment

I would like to express may deepest gratitude to Dr. Nadia Kamal *Assistant professor of Audiology, Ain Shams University,* who honored me by her kind supervisions, continuous help and valuable advices. Without her advices, this work could not be conducted.

No word can fulfill the feeling of gratitude and respect I carry to Dr. Nagwa Hazza, *Lecturer of Audiology, Ain Shams University,* for her great directions all through this work with a scientific personality and a kind heart.

My sincere gratitude and Acknowledgment is addressed to professor Dr. Ahmed Abd El Monem, *Prof. and chairman of E.N.T. department, El Minia University* for his continuous help and great support.

Before all, I would like to express my sincere thanks and deepest gratitude to our Prof. Dr. Salah Soliman, *Prof. and chairman of Audiology Unit, Ain Shams University.* He gave me and may colleagues the chance to learn a lot from him.

Also, I wish to express may unlimited thanks to all members of *Audiology unit, Ain Shams University* for their kind help and cooperation.

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Diabetes mellitus is a major disease affecting at least 5% of the population in developing countries **(Ghalioungi and Gharieb, 1978).**

The great majority of diabetic patients belong to the primary or the idiopathic diabetes which consists of two main types: Juvenile onset or insulin dependent diabetes mellitus (IDDM) and maturity onset or non insulin dependent diabetes mellitus (NIDDM) **(Irvine and Strong, 1978).**

Zelenka and Kozak (1965), suggested that diabetes mellitus affects the inner ear early in its course even before the disease become manifest clinically. Many studies have indicated that diabetes mellitus may lead to a lesion in the inner ear characterized by a slowly progressive, bilaterally symmetrical, sensorineural hearing loss **(Cerami et al., 1988).** The chain of experiments done by **Koide et al. (1958, 1960)** showed that the inner ear is dependent on glucose metabolism and it is primarily aerobic.

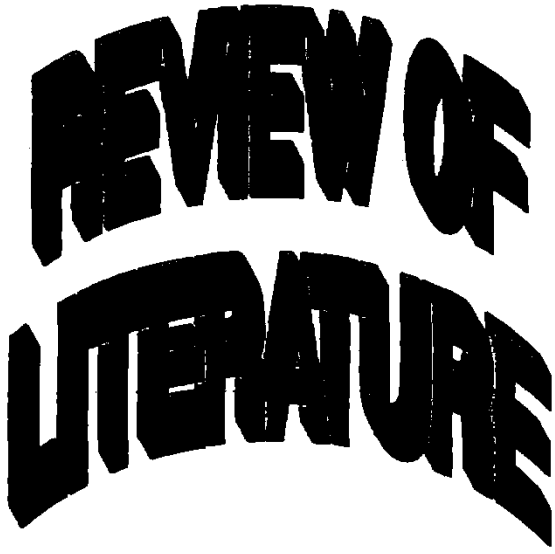
Otoacoustic emissions has shown to be an objective and non invasive tool to study cochlear function **(Martin and**

Lonsbury-Martin, 1993) . Also it is easy to perform, sensitive and has high test-retest reliability.

Recent observations suggest that otoacoustic emissions are produced by the motile activity of the outer hair cells **(Brownell, 1990).**

Both basic sciences and clinical studies strongly indicate that otoacoustic emissions provide objective measures of specific contribution that micromechanical activity of the outer hair cells makes to cochlear function **(Probst et al., 1991).** A study was done by **Triana et al. (1991)** on experimental rats, and a significant loss of outer hair cells was noted in the diabetic group of these animals.

Thus otoacoustic emissions, being originating from the outer hair cells and provide a very sensitive, objective and non invasive method to study cochlear function, could inform us about the cochlear status in diabetic patients



OTOACOUSTIC EMISSIONS (OAEs)

Otoacoustic emissions can be defined as the acoustic energy produced by the cochlea and recorded in the outer ear canal. This definition implies a cochlear origin of otoacoustic emissions generators **(Probst et al., 1990).**

Historical Background:

Over 30 years ago, **Gold (1948)** proposed the hypothesis that the sharp frequency selectivity exhibited by the cochlea results from a feedback system. This system consists of a mechanical to electrical transduction process coupled to an electromechanical conversion process. Such mechanism suggested the possibility of detecting this process in the form of sound in the ear canal. However **Gold's** hypothesis was never taken seriously until **Kemp (1978)** demonstrated that energy was indeed emitted by the cochlea and that it was recordable as acoustic vibrations in the ear canal using specialized method and equipment.

Classification of OAEs:

OAEs can be classified according to several logical schema. The most accepted one which defines emissions type according to the type of acoustic stimulation that best evokes them **(Martin et al., 1990).**

One major emission type is referred to as a spontaneous otoacoustic emission (SOAE). The remaining emissions are all evoked by deliberate application of acoustic stimulation. These evoked emissions are distinguished by the particular type of stimulation that elicits them. The first of these is termed a transient evoked otoacoustic emissions (TEOAEs), because this emission is elicited by clicks or other brief stimuli. Another emission evoked by a continuous, low level pure tone is designated as a stimulus-frequency otoacoustic emission (SFOAEs), since the emission frequency is identical to that of the eliciting stimulus. Finally, the simultaneous presentation of two tones to the ear results in the emission of a third frequency referred to as distortion product otoacoustic emission (DPOAEs).

Cochlear Origin of Otoacoustic Emissions:

The discovery of OAEs was first met with much skepticism **(Wilson, 1984)**. The emissions were believed initially to be an artifact, possibly related to middle ear activity. Today, the cochlear source of OAEs generation is well documented and generally accepted **(Probst et al., 1991)**.

One of evidences supporting the cochlear origin of OAEs comes from studies investigating the effect of ototoxic drugs on various emission types. Such drugs are toxic to the cochlear structure and their ingestion has been shown to lead to reduction or elimination of OAEs. The effect of aspirin, a drug with reversible ototoxicity, has been tested in both animal and human subjects. Studies in humans revealed that SOAEs were temporarly reduced or abolished by aspirin administration **(McFadden and Plattsmier, 1984; Wier et al., 1988)**. Additionally, depending on the emission under examination, TEOAEs & SFOAEs were affected, although not as dramatically as the effects observed on SOAEs **(Long & Tubis, 1988 and Johnsen and Elberling 1992)**. Moreover, **Long and tubis (1988)** have further shown that all emission types are reduced by aspirin. **Brown et al. (1989)** reported that the aminoglycoside antibiotic gentamycin, a proved ototoxic agent, led to elimination of DPOAEs in guinea pig. This finding was confirmed by electron microscopy where damage to the outer hair cells were evident **(Brown et al., 1989)**.

Another evidence demonstrating cochlear origin of OAEs, came from the observation that all four classes of OAEs are influenced by excessive acoustic stimulation. The primary effect of exposure to loud sounds is a reduction in the emission amplitude. Wheres the findings in human ears have

been based on relatively short exposure that induced temporary threshold shift (TTS) and reduction in the amplitude of SOAEs **(Norton et al., 1989)** or TEOAEs **(Kemp, 1981, 1982, 1986)**, studies in experimental animals generally have employed longer exposure to loud sounds resulting in more substantial cochlear damage and elimination of all classes of OAEs **(Evans et al., 1981, Anderson and Kemp 1979, Wilson and Evans, 1983)**.

Another line of evidences of cochlear origin of OAEs, comes from experimental studies in guinea pigs which showed that SOAEs and TEOAEs were reversibly abolished by hypoxia **(Zwicker and Manley 1981 and Evans et al., 1981)**. Also **Kemp and Brown (1984)** reported that anoxia or hypoxia led to a reduction in the DPOAEs amplitude and this reduction suggests the in influence of a metabolic dependent process on the generation of OAEs. This relation is not compatible with the notion that OAEs originate within the middle ear.

Other evidence that OAEs are generated by the cochlea is derived from the findings of studies with hearing impaired humans. **Kemp (1978)** reported that emissions such as SOAEs and TEOAEs are generally undetectable if hearing losses are greater than 25-30 dBHL. The same author reported that the emissions are frequency dependent i.e. no

TEOAEs are found in frequency regions where hearing loss is greater than 30 dBHL, while at the same time, emissions can still be present in adjacent frequency region, within the same ear, where hearing thresholds remain relatively normal **(Kemp et al., 1986, Probst et al., 1987)**. These two findings that OAEs are dependent on hearing threshold and the tested frequency, provide additional evidence supporting the proposal that the generation of OAEs occurs within the frequency analyzing portion of the peripheral auditory system i.e. within the cochlea.

Also the findings of studies of suppression phenomena, in which the amplitude of an OAE is reduced by stimulation with additional tone, support the cochlear origin of OAEs. This finding was documented by many investigators both in human beings **(Brown and Kemp 1984, Bargones and Burns 1988)** and in animal experimentation **(Evans et al., 1981, Martin et al., 1987, Manley et al., 1987)**. These studies demonstrated that the amount of emission amplitude reduction or suppression is dependent on the frequency and intensity of the suppressing tone and this suggested that the emission are highly tuned in a manner similar to that measured for single auditory nerve fiber. This implies that the generation of emissions is related to tuned elements which are generally believed to be localized in the cochlea.

Evidence Opposing a Neural Origin of OAEs:

An evidence opposing a neural origin of OAEs came from experiments done by **Arts et al. (1990)** on gerbils. **Arts et al.** used tetrodotoxin (TTX) which is a sodium channel blocker that inhibits synaptic transmission. The click-evoked A.B.R. was abolished when TTX was placed in the round window niche, whereas DPOAEs input-output function was not significantly altered.

Siegel and Kim (1982) and Martin et al. (1988) have show that DPOAEs are not altered when 8th nerve was severed in experimental animals. Thus, it clearly appears that the mechanisms responsible for OAEs generation are peripheral to 8th nerve.

Finally, the very interesting clinical observation that TEOAEs can be detected in cases of central hearing loss with hearing threshold above 30 dBHL even when no A.B.R. waves could be detected. This finding is a strong evident opposing the neurol origin of OAEs and a clear document supporting the cochlear origin of OAEs.

Middle Ear Consideration:

It is important to appreciate that, before OAEs can be recorded in the ear canal, it is essential that the