

OTOACOUSTIC EMISSIONS MEASUREMENT IN MÉNIÈRE'S DISEASE

THESIS

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in
Audiology

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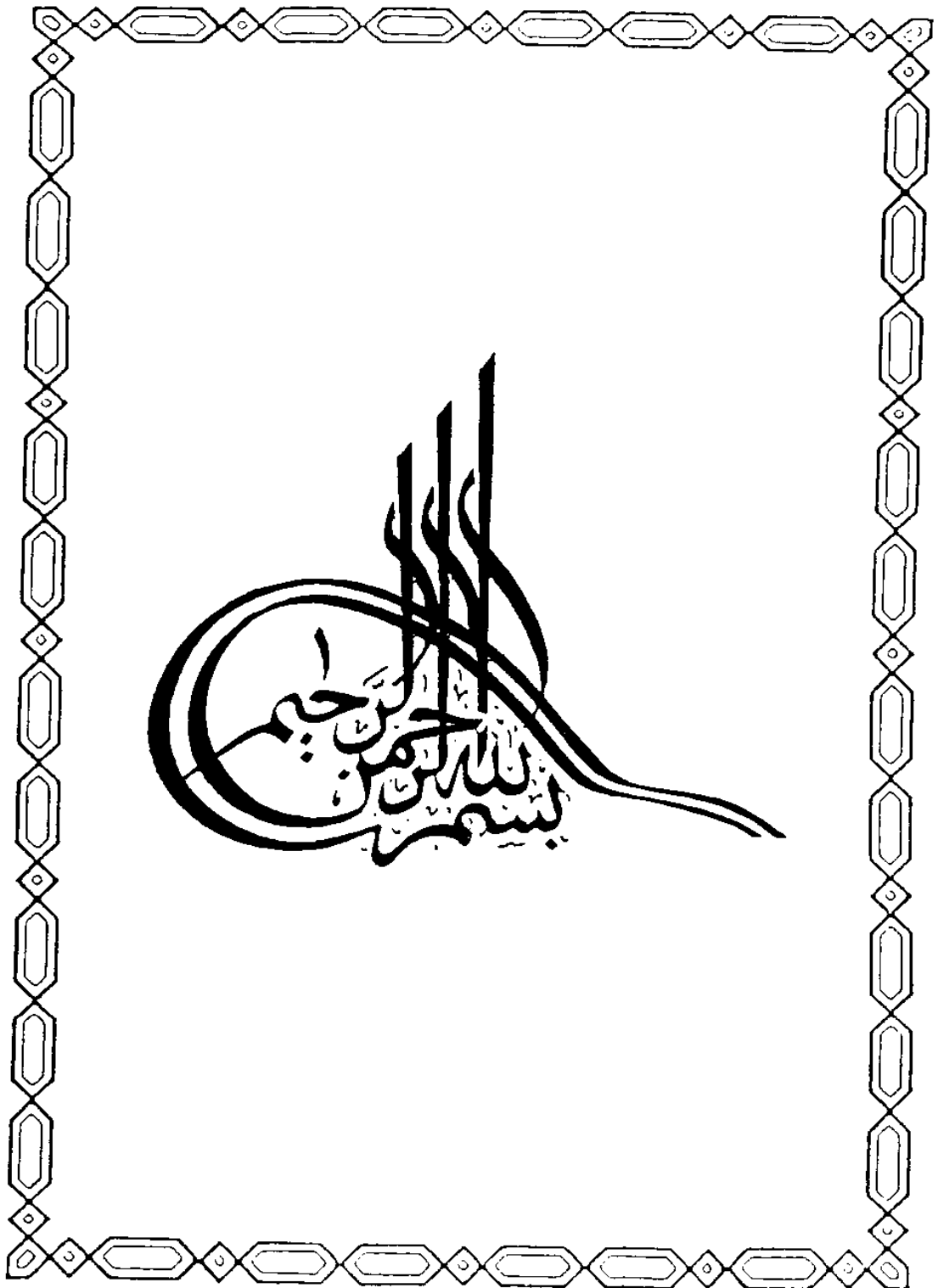
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بسم الله الرحمن الرحيم

سنريهم آياتنا في الآفاق وفي أنفسهم

حتى يتبين لهم أنه الحق أو لم يكف

بربك أنه على كل شيء شهيد

صدق الله العظيم

فصلت الآية (٥٣)

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To..
My Parents...
Who Try Every Moment to
Offer Me A Best Life..

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Introduction and Rationale

INTRODUCTION AND RATIONALE

Ménière's disease is a disease of the inner ear which was first described by Prosper Ménière in 1861. He described a condition that is characterized by sudden and recurring episodes of vertigo with nausea and vomiting, together with hearing loss and tinnitus (Colman, 1987).

In addition to this classical presentation of Ménière's disease atypical presentation may occur, including cochlear Ménière's disease, vestibular Ménière's disease, tumarkin otolithic crisis and Lermoyez syndrome (Balkany et al., 1980).

Ménière's disease is not an uncommon clinical disorder, Matsunaga (1976) gave an average incidence of 0.5% of the disease in patients attending ear, nose and throat clinics in various hospitals in different countries. Nedzelski et al. (1986), reported that Ménière's disease is responsible for 10% of visits to dizziness unit at Sunnybrook Health Science Center, University of Toronto. A study done by El-Sayed et al. (1992) showed that dizziness is a common symptom encountered among adult Egyptian patients attending the Audiology Unit Otolaryngology Departement (24.6%). He found also that Ménière's disease was the common cause of peripheral vertigo (27.3%) among adults Egyptian patients.

Although Ménière's disease is a well recognized clinical entity, an accurate diagnosis is often difficult to make, and usually based on clinical picture. The difficulty become more obvious in early stages or when clinical presentation has atypical picture.

Electrocochleography showed to be helpful in the objective identification and monitoring of Ménière's disease and endolymphatic hydrops (*Ruth et al., 1988*).

The summing potential/action potential (SP/AC) amplitude ratio proved to be the best index of SP measurements in the diagnosis of Ménière's disease providing that the patient has at least 25 dB low frequency sensorineural hearing loss. Meanwhile, the diagnostic significance of SP/AC complex is reduced when pure tone average at 250, 500 and 1000Hz exceeds 55dB (*Kamal et al., 1995*).

Otoacoustic emissions (OAEs) are sounds generated by the cochlea, and can be measured in the outer ear canal (*Kemp, 1978*).

Recent observations suggest that OAEs, can be considered as an excellent way to monitor the cochlear active mechanics (*Probst et al., 1991*). Minor changes in the cochlear function that may not result in changes in the pure tone audiogram, can lead to significant changes in OAEs (*Kemp, 1988*).

Additionally, transient evoked OAEs, combined with glycerol test provide a simple and non-invasive method that could be used for assessing the reversibility of hydrops in the early stages of Ménière's disease (*Tanaka et al., 1990*).

Accordingly, this work address the hypothesis that otoacoustic emissions could have a role in the objective diagnosis of Ménière's disease specially in the early or atypical cases.

<i>Aims Of The Work</i>	

AIMS OF THE WORK

1. To define the response pattern of otoacoustic emissions both transient and distortion product in subjects with Ménière's disease, if any.
2. To study early changes in the non affected ear of Ménière's disease subjects by otoacoustic emissions measurements.
3. To measure the effect of glycerol test on changes of otoacoustic emissions both transient and distortion products, if any.

<i>Review of Literature</i>	

IDIOPATHIC ENDOLYMPHATIC HYDROPS (Ménière's disease)

Definition:

Ménière's disease is defined as a disorder of the membranous labyrinth that consists of fluctuating sensorineural hearing loss, episodic vertigo, tinnitus and less frequently aural fullness (*Schessel and Nedzelki, 1993*).

Historical Background:

In 1861, Ménière's described the autopsy findings of a young girl who experienced sudden, complete deafness and severe vertigo before death. A brain lesion was suspected. At autopsy however, he found bleeding within the labyrinth, the brain was normal. This report first drew attention to the inner ear as the cause of vertigo (*Linstrom, 1992*).

During his lifetime, Vincent Van Gogh was thought to have epilepsy and "madness". However, when investigators reviewed 96 personal letters Van Gogh wrote to family and friends between 1884 and 1890, they found that the correspondence described symptoms typical of labyrinthine vertigo coupled with nausea, vomiting, noise intolerance. These episodes were separated by symptom-free periods. Van Gogh reported positional vertigo, fluctuating hearing loss, tinnitus, hyperacusis (*Arenberg et al., 1990*).

In 1927, Guild clearly demonstrated longitudinal flow of endolymph, referring to the endolymphatic sac as a site of "outflow of endolymph". In guinea pigs Portmann (1927) described surgery