MIDDLE LATENCY RESPONSE (MLR) IN TEMPORAL LOBE AND BRAINSTEM DISORDERS

THESIS

Submitted in Partial fulfillment

of The M.D. Degree

in Audiology

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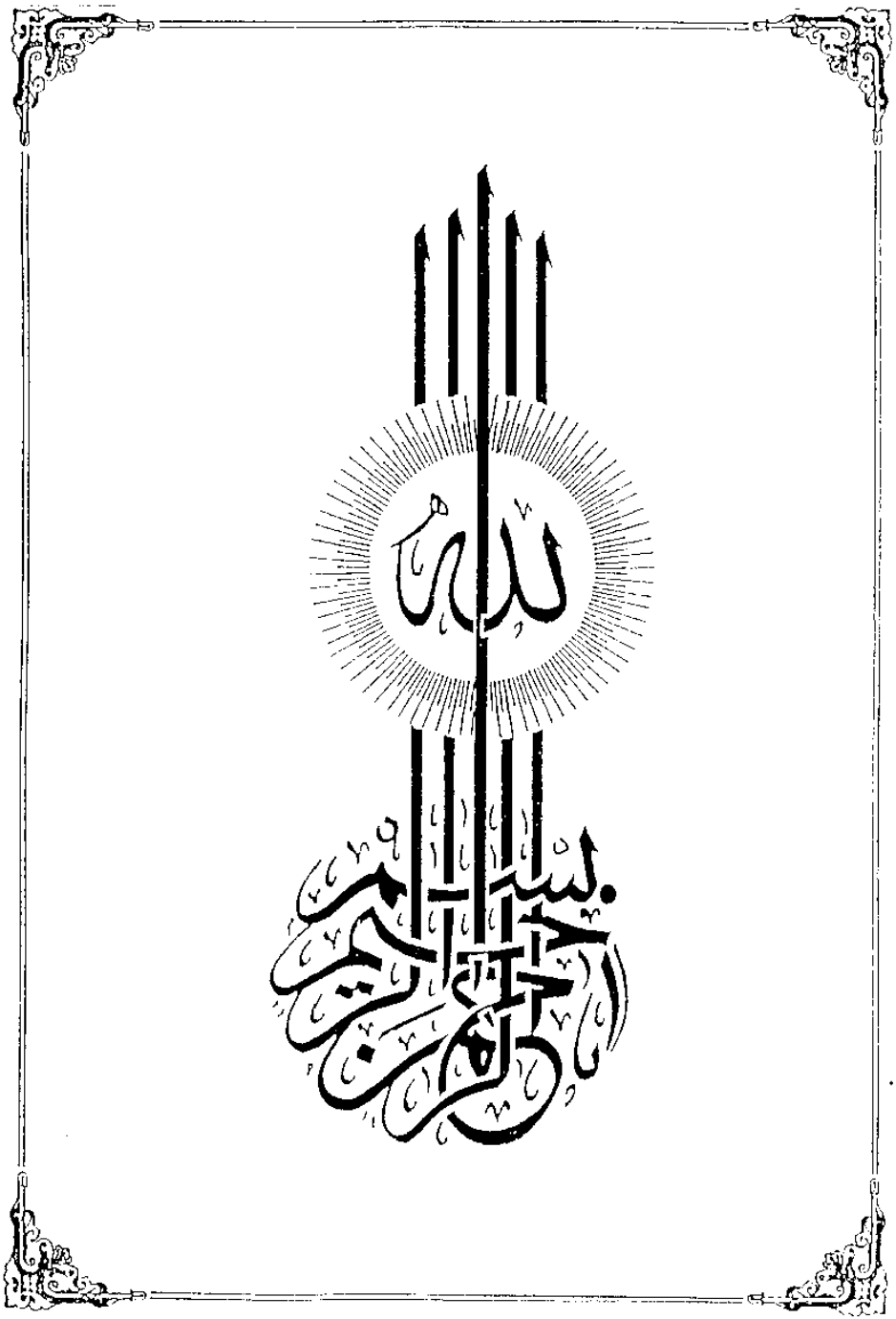

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INTRODUCTION

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INTRODUCTION AND RATIONALE

Assessment of both the locus and the extent of pathology within the peripheral auditory nervous system has become a relatively straightforward and routine procedure for many years. Major strides in the assessment and diagnosis of retrocochlear lesions have also been achieved in recent years.

Progress in diagnosis of central auditory disorders, on the other hand, has not kept pace with the advancement made in assessment of disorders of the peripheral auditory system. Beginning in the early fifties, clinical research studies of central auditory dysfunction showed slow progress which was attributed to three reasons (Rintelmann, 1985); The dearth of precisely documented central auditory nervous system (CANS) lesions in patients tested by audiologists, the lack of sensitivity of conventional auditory tasks for identifying the CANS lesions, and most importantly, the resistance of CANS to exhibit disruption on auditory tasks due to anatomic and physiologic complexity of the central auditory pathway.

Several techniques have been developed for the diagnosis of the site of lesion within the CANS. These were either behavioural tests (central auditory tests) or electrophysiologic procedures such as auditory evoked potentials (AEPs).

The central auditory test battery included speech or non-speech tasks that were devised to diagnose central auditory dysfunction through the introduction of complex or distorted signals. Although these tests seemed easy to apply , there were

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many limitations to the use of these procedures (Olsen and Palm,1986) including: 1) Lack of consistent normative data due to the great variability among tests and subjects. 2) The questionable validity of these tests in evaluating children, specifically those with learning disabilities, or elderly subjects . 3) Peripheral hearing loss, when present, can have a confounding effect on the administration and interpretation of these tests.

Auditory evoked potentials (AEPs) are a series of far field, volume conducted reflections of the stimulus related changes that take place in raw EEG with the presentation of an acoustic stimulus to the listener's ear. These event related potentials can be recorded from the scalp. The earliest potential appear within few milliseconds of stimulus onset and form a sequence of waves extending for several hundreds of milliseconds thereafter (Squires and Hecox, 1983).

Recognized first by Davis (1939), AEPs were promising to be a useful and objective clinical tool which can reveal normal or abnormal neural functions as opposed to the analysis of structure which is the domain of radiology.

There have been several approaches to the classification of AEPs. One common classification is based on the latency "epoch" of the response (Picton et al, 1974; Picton and Fitzgerald

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1983). The various epochs are designated as "first" (0-2 msec), "fast" (2-10 msec), "middle" (10-50 msec), "slow" (50-300 msec) and "late" (300+ msec). Often, the presumed site of origin of a particular AEP tends to take precedence over other classifications , thus, it is more common to describe the fast response as the auditory brain stem response (ABR). However, since the origin of certain AEPs components is not yet settled, they are still described by their latency, e.g: the middle latency response.

During the past decade the AEPs have been used successfully as an objective means for threshold determination and in providing information as to the location and severity of auditory nervous system disorders.

The auditory brainstem responses (ABRs), due to their replicability, stability and localizing properties are, at present, the most widely used component of the AEPs. The ABR has proven to be an excellent tool in the diagnosis of eighth's nerve lesions (Brackmann,1977; Josey et al.,1980; Moller and Moller, 1983), in providing useful information to a variety of pathologies involving the brainstem such as demyelinating disease, degenerative disorders, tumors and vascular lesions (Stockard et al.,1977; Musiek et al., 1984) and in evaluation of comatosed patients (Kaga et al.,1985).

However, the ABR, which have origins at levels ranging from the eighth nerve to the rostral brainstem, sample only an anatomically and functionally restricted portion of the auditory

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pathway (Lev and Sohmer, 1972; Hashimoto et al.,1981). Moreover, in the presence of cochlear hearing loss, specially in the high frequency range, there may not be readable response by ABR. This limits the use of this response for neuro-otologic diagnosis in those patients.

First described by Geisler et al. (1957), the middle latency response (MLR) was among the first AEPs recorded from the human scalp. Nevertheless, Bickford et al.(1964) and Mast (1965) indicated that this response reflects solely myogenic rather than neurogenic activity and consequently the procedure did not receive enthusiastic interest among researchers. Later studies, however, have proven that the response was neurogenic and not myogenic in origin (Harker et al.,1977).

In recent years, with the increased popularity of the ABR, the MLRs have received renewed attention in both audiological and neurological investigations . The potential utility of these responses were strengthened by studies showing that the response offers a good compromise between reliability, sensitivity, frequency selectivity and resistance to sleep and mild sedatives (Davis, 1981).

MLR is now reported to have a significant value in threshold determination, particularly for low frequency threshold measurements (Musiek and Geurkink, 1981; Maurizi et al.,1984).

Subsequently, the possible utility of MLR as a test for lesions central to the brainstem and as an adjunct to ABR in diagnosis of brainstem lesions has attracted the attention of investigators. However, the use of MLR in neuro-otological

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diagnosis is still limited by the lack of knowledge about the specific response generators. Although this issue has been addressed in several studies, no conclusive evidence could be obtained yet. Picton et al. (1974) listed several possible neural generators including the thalamus, association cortex in frontal, parietal and temporal lobes. Some investigators have pointed to the auditory cortex as the generator of all or at least some of the MLR components (Kaga et al.,1980; Cohen, 1982, Özdamar et al., 1982).

The use of MLR in detection of cortical dysfunction is still in the experimental stage. And inspite of the limited number of studies published regarding this issue, there have been conflicting reports on the effects of temporal lobe lesions on MLR and accordingly the extent of temporal lobe structures generating the AEP. While Özdamar et al.,(1982) indicated total absence of the response in a patient with bilateral temporal lobe lesions, Parving et al.,(1980) reported normal responses in a patient who also had bilateral temporal lobe lesions. Kraus et al.(1982) reported abnormal responses in patients with unilateral temporal lobe lesions, however, the response remained intact in some patients inspite of substantial temporal lobe damage. Recently, two reports have been published on MLR in patients with lesions involving the temporal lobes (Kileny et al.,1987; Woods et al.,1987) who almost reached opposite conclusions as regards the effects of lesions and the response generators.

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Musiek et al.(1984) indicated that a prerequisite to MLR evaluation of cortical lesions is to rule out or, at least, account for any auditory periphery, or brainstem dysfunction which might contaminate the MLR results in these patients.

At present, before widespread clinical application of the MLR as a test for lesions involving the CANS, there is a need to study this response in patients with various documented lesions involving the brainstem and the temporal lobe, controlling for other factors which might affect the response such as age of the subject and peripheral hearing loss.

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AIMS OF THE WORK

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The objectives of the present work were:

1) To study the effects of temporal lobe lesions on the latency and amplitude of the MLR.

2) To determine the effects of brainstem lesions on MLR latency and amplitude.

3) To investigate whether the effects of documented lesions on the response would provide significant information pertinent to its generators.

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REVIEW OF LITERATURE

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