AN INVESTIGATION INTO THE EFFECTS OF SEX DIFFERENCE AND CONTRALATERAL MASKING ON THE MONAURAL BRAINSTEM AUDITORY EVOKED RESPONSE (BAER) OBTAINED IN A GROUP OF NORMAL HEARING INDIAN UNDERGRADUATE UNIVERSITY STUDENTS.

by

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*For my wife, **Devageeranee**, my daughter*
***Nadine** and my son **Jonathan**.*

ABSTRACT

The objectives of the investigation were to establish diagnostic reference data; and to examine and report on the effects of sex difference and contralateral masking on the normal BAER. BAERs were elicited from the target (R) ear using clicks presented at 70dBnHL both in the absence and presence of three (50, 60 and 70dBHL) levels of contralateral broadband masking noise. Relevant latency and amplitude data were obtained from 60 selected normal hearing Indian undergraduate female (N=30; $\bar{X}$ age = 20.33 years) and male (N=30; $\bar{X}$ age = 21.33 years) students aged between 18 and 25 years ($\bar{X}$ age = 20.73 years). Diagnostic reference data were established for the absolute latencies of peaks I to VI; relative latencies of peaks I-III; III-V and I-V; absolute amplitudes of peaks I and V and the relative amplitude ratio of peaks V:I. The application of the MANOVA revealed an overall significant ($p < 0,05$) sex difference effect while no significant differences were observed between the masked and non-masked normal BAER. Furthermore, there were no significant overall interactional effects of sex difference and masking on the BAER. These results are discussed in terms of the literature and implications for clinical application and further research.

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CHAPTER 1

1.1 INTRODUCTION

During the past decade and a half, there has been a formidable increase in the use of specialized audiological test procedures in otoneurological diagnosis. Since the initial description of a procedure to record auditory evoked far-field electrical potentials from the human scalp (Jewitt and Williston 1971), the measurement of the brainstem auditory evoked response (BAER) has become the most recent electrophysiological procedure to be integrated into testing protocols (Jacobson, 1985b). This procedure subsequently had a major impact on the disciplines of audiology, otology and neurology, (Schwartz and Berry, 1985). The development of the BAER has focused on two principal areas of application :

i. the evaluation and diagnosis of peripheral auditory problems and related pathology, and

ii. the assessment of the neural integrity of the acoustic nerve and caudal levels of the brainstem afferent auditory pathway (Hecox and Jacobson, 1984).

However, despite the reported robustness and stability of the BAER as a reliable assessment tool, it is critical to the effective use of this measurement to have diagnostic reference data that are collected within the individual laboratory or clinic. Several investigators have reported on the myriad of variables that can potentially alter one or more of the important parameters of the BAER, and hence lead to misinterpretation. It is, therefore, appropriately suggested by Schwartz and Berry (1985), that it is not advisable for any clinician to depend on published diagnostic reference data for interpreting BAERs. This emerges from the opinion that there is a lack of uniformity in BAER measurement variables among investigators in various clinics and laboratories around the world.

Among the numerous variables cited in the literature as having an influence on the elicitation of the BAER in normal subjects, are differences in sex (Beagly and Sheldrake, 1978; McClelland & McCrea, 1979; Stockard et al., 1978; 1979; and Jerger & Hall, 1980) and the apparent influence of contralateral masking (Ozdamar and Stein, 1981; Reid and Thornton, 1983; and Reid et al., 1984).

Although there are published diagnostic reference data on the influence of differences in sex on the BAER, Jerger et al. (1980), stressed that this variable should be routinely considered in the generation of "normal values" for any clinic or laboratory. As regards the influence of contralateral masking on the elicitation of the normal BAER, Ainslie and Boston (1980), have stated that previous investigators have not clarified the effect of this variable on the BAER for monaural stimuli.

Therefore, it seems important to establish diagnostic reference data on the normal BAER within a clinic and to assess how the variables of differences in sex and the use of contralateral masking would influence the monaurally elicited BAER.

1.2 MOTIVATION

The following factors motivated this study.

i. Although there is available information on diagnostic reference data on the normal BAER (Martin & Moore, 1977; Rowe, 1978; Chiappa et al., 1979; Fria, 1980 and Schwartz & Berry, 1985), such information is lacking in South Africa for any age or population group. Furthermore, due to the absence of standards to specify recording parameters and methods used to measure the BAER, (Schwartz and Berry, 1985), it is imperative that the Audiology Clinic at the University of Durban-Westville establish it's own diagnostic reference data based on it's own test equipment and protocol. BAER interpretation maybe confounded by the influence of various factors viz. differences in:

   a. electrical and electromagnetic field variations between clinical/laboratory sites
   b. the use of different stimuli
   c. recording and analysis parameters
   d. electrode placement
   e. transducer type and
   f. transducer placement.

   The above may lead to small but significant changes in peak latency, amplitude and morphology. Therefore, a study planned to elicit the BAER in a group of normal people under controlled conditions would provide relevant information, leading to the establishment of suitable diagnostic reference data for the interpretation of BAER in the Audiology Clinic at the University of Durban-Westville. These data may be appropriate for patients in the age range 18 to 25 years.

ii. There appears to be evidence of a sex effect on the BAER amongst normal males and females (Beagly & Sheldrake, 1978; Stockard et al., 1977; Jerger & Hall, 1980 and Michalewaski et al., 1980). These investigators suggest that females generally present with earlier response latencies and large peak V amplitudes than males of the same age. It has been hypothesized that this observed phenomenon is due to females having a smaller head circumference and a foreshortening of the brainstem pathway between the auditory nerve and midbrain (Stockard et al.,1978). Regardless of the explanation for this difference, the clinical implication of these data is that each facility should generate individual diagnostic reference latency and amplitude values between sexes. The practice of applying female norms to male patients could lead to interpretations of delayed waveform latency when none actually exists (Jerger & Hall, 1980 and Schwartz & Berry, 1985). This would then lead to an interpretation error. Therefore, a study to examine this difference would contribute towards establishing separate diagnostic reference values for males and females, thereby preventing the occurrence of potential errors in the interpretation of BAERs in the Audiology Clinic at the University of Durban-Westville.

iii. There have been reports suggesting that the use of contralateral masking has an effect on the monaurally elicited BAER amongst normal hearers (Reid & Thornton, 1983; Reid et al. 1984, and Ozdamar & Stein, 1981). However, these investigators have not clarified the effects of cross-talk especially with respect to certain characteristics of the BAER. Therefore, a study examining the effects of contralateral masking on certain characteristics of the monaurally BAER in normal hearers would help to add and or clarify information on the influence of this variable.

iv. In conducting a comprehensive review of selected available literature, it has been noted that there are no readily available studies reporting on the combined effects of differences in sex and of the influence of contralateral masking on the monaurally elicited BAER among normal hearers. Therefore, a study designed to examine the interactional influence of these variables on the monaurally elicited BAER would serve to contribute new and additional information to the field of BAER audiometry.

It is apparent that a study designed to examine the normal response characteristics amongst a group of male and female subjects will permit the researcher to:

a. establish appropriate diagnostic reference data for the clinical interpretation of the BAER in the Audiology Clinic at the University of Durban-Westville in a selected age group.

b. to study and to report on the influence of differences in sex and of contralateral masking on the monaurally elicited BAER amongst normal hearers.

In so doing, the study may, provide relevant information which would serve to contribute to the literature in this field.

1.3 AIMS OF THE INVESTIGATION

The following are the specific aims of this study:

i. To establish diagnostic reference data among a group of normal hearing Indian university students aged between 18 and 25 years with respect to certain characteristics of the monaural BAER viz. the:

   a. absolute latencies of peaks I to VI
   b. relative (interpeak) latency (IPL) of peaks I-III, III-V and peaks I-V.
   c. peak to trough amplitudes of peaks I and V
   d. amplitude ratio obtained by comparing the absolute amplitudes of peak I and V.

ii. To compare the monaural BAER between a group of male and female Indian university students with respect to the undermentioned characteristics viz. the:

   a. absolute latencies of peaks I to V
   b. relative (interpeak) latencies of peaks I-III, III-V and peaks I-V
   c. peak to trough amplitude of peaks I and V
   d. amplitude ratio of peak V compared to peak I.

iii. To determine the effect of contralateral masking on the monaurally elicited BAER among undergraduate Indian students. This is to be done by comparing the BAER recorded from the target ear both in the absence and presence of three intensity levels (50, 60 and 70dBHL) of contralateral masking. The following characteristics of the BAER will be examined; the:
   a. absolute latencies of peaks I to V
   b. relative (interpeak) latencies between peaks I-III, III-V and I-V
   c. absolute amplitude of peak V
   d. amplitude ratio of peak V compared to peak I.

iv. To investigate the interactional effects of sex difference and of contralateral masking on the monaurally elicited BAER with respect to the following; the:
   a. absolute latencies of peaks I to V
   b. relative latency of peaks I-III, III-V and I-V
   c. absolute amplitude of peak V
   d. amplitude ratio of peak V compared to peak I.

1.4 HYPOTHESES

In order to fulfill aims i, ii, iii and iv listed above, the following hypotheses will be tested :

i. BAER data are obtainable for Indian university students; 18 to 25 years.

ii. There is a significant difference between the monaural clinical BAER recorded from the target ears of males and females with respect to the following charateristics;

the:

   a. absolute latencies of peaks I to V
   b. relative latencies of peaks I-III, III-V and I-V
   c. absolute amplitude of peak V
   d. amplitude ratio of peak V compared to peak I.

iii. There is a significant difference between the clinical BAER recorded from the target ear in the presence of contralateral masking, and that recorded in the absence of contralateral masking among a group of undergraduate, students with respect to the following characteristics; the:

   a. absolute latencies of peaks I to V
   b. relative latencies of peaks I-III, III-V and I-V
   c. absolute amplitude of peak V
   d. amplitude ratio of peak V compared to peak I.

iv. There is a significant difference in the clinical BAER recorded from the target ear in both the presence and absence of contralateral masking, between male and female Indian undergraduate students with respect to the following charateristics; the:

   a. absolute latencies of peaks I to V
   b. relative latencies of peaks I-III, III-V and I-V
   c. absolute amplitude of peak V
   d. amplitude ratio of peak V compared to peak I.

1.5 CONCLUSION

In this chapter, the problems to be investigated have been identified. In addition, the aims and hypotheses have also been presented. In the ensuing chapters, the definition of concepts, the relevant research findings and the design of the investigation will be discussed and presented.

CHAPTER 2

DEFINITION OF CONCEPTS, HISTORICAL DEVELOPMENT AND DESCRIPTION OF THE BAER.

2.1 DEFINITION OF CONCEPTS

In view of the fact that the area under study is highly specialized, it is appropriate to review and to clarify concepts, terms and abbreviations that are used in the preparation of this dissertation. The following is a presentation of these concepts, terms and abbreviations together with their definitions and points of clarification.

i. EEG : refers to the electroencephalogram which is a recording of spontaneous, random bio-electric activity (in the absence of sensory stimulation) which is generated by the central nervous system (Jacobson et al., 1985). Berger (1929), as cited by Glasscok et al. (1987), first described EEG activity as "semi-rhythmic electrical patterns of varying amplitudes and frequency, which are dependent on the state of the subject".

ii. EP : refers to evoked potential. Hampton (1984), defines an EP as an electrical by-product of activity in the peripheral and central neural pathways in response to an external stimulus.

iii. AEPs : refers to auditory evoked potentials. These are minute but consistent stimulus-related changes in the raw EEG tracings when presenting acoustic stimuli to the ear. These minute changes are then extracted from the ongoing EEG by using the principle of algebraic summation of electrical activity following repeated auditory stimulation (Clark et al., 1961). The AEP waveform revealed by summation usually has wave components (peaks and troughs) which are described by their amplitude and latency characteristics (Jacobson et al., 1985).

iv. NOMENCLATURE AND CLASSIFICATION OF AEPs : According to Jacobson et al. (1985), there is no standard set of terms or classifications that applies to AEPs. There have been several approaches to classifying AEPs. However, descriptions used, usually include at least one or more of the following aspects: response latency; physiologic description; anatomical origin; stimulus response relationships and electrode placement. A summary of AEP classification and their various wave nomenclatures is presented in Table I.

Table 1 : A classification system of auditory evoked potentials and their descriptions. Adapted from Jacobson et al., (1985).

Common name	Physiological description	Anatomy source	Latency epoch	Latency range (m/s)	Stimulus-response	Electrode response
Cochlear microphonic (CM)	Receptor	Hair cells	First	0	Sustained	Near-field
Summating potential (SP)	Receptor	Hair cells	First	0	Sustained	Near-field
Action potential (AP) (N_1 N_2) (ECochG)	Neurogenic	Auditory nerve	First	<2	Transient	Near and Far-field
Auditory brainstem response (ABR) (I-VII)	Neurogenic	Auditory nerve Brainstem	First Fast	<10	Transient	Far-field
Frequency following response (FFR)	Neurogenic	Brainstem	Fast	Tone duration	Sustained	Far-field
Middle latency response (MLR) (No,Po,Na,Pa,Nb,Pb)	Neurogenic	Thalamus Auditory cortex	Middle	8-50	Transient	Far-field
Event-related potential (40Hz)	Neurogenic	Brainstem-Thalamus Auditory cortex	Fast? Middle?	12-50	Transient? Sustained?	Far-Field
Slow-vertex response (SVR) P_1, N_1, P_2, N_2)	Neurogenic	Cerebral Cortex (primary and association	Slow	50-300	Transient	Far-field
Sustained cortical potential (SCP)	Neurogenic	Cerebral Cortex (P & A)	Slow	Tone duration	Sustained	Far-field
Late positive component (P_{300})	Neurogenic	Cerebral Cortex (P & A)	Late	250-350	Perceptual	Far-field
Cognitive negative variation (CNV)	Neurogenic	Cerebral Cortex (association)	Late	300+	Perceptual	Far-field

m/s = milli-seconds

v. BAERs : refers to brainstem auditory evoked responses. (Chiappa et al., 1979). The BAER is a set of five to seven positive peaks recorded from the scalp during the first 10-12m/sec following click stimulation (Jewett et al., 1970, 1971). They represent far-field evoked potentials originating in the auditory nerve (VIIIth cranial nerve) and brainstem and are conventionally labelled in Roman numerals as suggested by Jewett et al. (1970, 1971), cited by Gastone et al., (1987).

It is important to note that this auditory evoked response has been referred to and labelled differently by different writers in the literature. Effectively, they all refer to the same AEP. A list of these references is given below :

a. BAEPs : refers to Brain Stem Auditory Evoked Potential (Gastone et al., 1987).

b. ABR : refers to Auditory Brain Stem Response (Jacobson et al., 1985; Fria 1980; Glasscock et al. 1987, and Jerger & Hall, 1980).

c. BER : refers to Brainstem Electrical Responses (Gibson and Ruben, 1978).

d. BEP's : refers to Brainstem Evoked Potentials (Picton et al., 1977).

For the purposes of this dissertation the abbreviation BAER as suggested by Chiappa et al. (1979), and Rowe (1978), will be used. This would allow the writer to use a consistent term throughout the presentation of this dissertation.

vi. MONAURAL : referring to ONE ear.

vii. CONTRALATERAL : refers to the ear opposite the target ear.

viii. MASKING : refers to the introduction of a noise that prevents a listener from hearing another sound. Typically in audiometry, masking is presented to the non-test ear. Masking may also lead to a worsening of the auditory threshold for one sound by the introduction of another sound in the same ear (Katz, 1985).

ix. CLICK : refers to an abrupt broad band sound with a rapid onset that is produced by exciting a headphone with a rectangular voltage pulse. This pulse has a duration which is equal to 100µsec. (Jacobson et al., 1985).

x. All of the following; CROSS-HEARING, CROSS-TALK AND CROSSOVER, refer to a situation which occurs during audiometry in which an acoustic stimulus presented to one ear is transmitted to the opposite ear, usually by vibration of the skull. (Katz, 1985).

xi. INTER-AURAL ATTENUATION : refers to the amount by which the intensity of a sound is reduced as it travels across the head from the test-ear to the non-test ear (Hodgson, 1980). It is considered to be at least 40dB for air-conducted pure tones and 0dB for bone-conducted pure tones (Katz, 1985). For click stimuli the mean air-conducted inter-aural attenuation was found to be 63dB (Ozdamar and Stein, 1981), as cited by Reid et al., (1984).

xii. dBHL : Decibel hearing level - refers to the level in (dB) of a sound relative to 0dBHL, which is equal to the average hearing threshold for young adults with normal hearing (ANSI 1969), as cited by Katz, (1985).

xiii. dBsl : Decibel sensation level - refers to any level of sound (dB) above an individual's threshold of audibility for that sound (Katz, 1985).

xiv. LATENCY : refers to the time relationship between any response and the stimulus eliciting that response. (Fria, 1980).

xv. ABSOLUTE LATENCY : refers to the time relationship between stimulus onset and the associated response (Fria 1980).

xvi. RELATIVE OR INTERPEAK LATENCY : refers to the time difference between two component peaks eg. the I-V interpeak latency (Fria, 1980).

xvii. AMPLITUDE : refers to the height of a given wave component, and it is usually measured in microvolts (μV) from the peak of the wave to the following trough. This measurement is sometimes referred to as absolute amplitude (Fria, 1980). Refer to figure 7 p. 38 for an illustration of this measurement.

xviii. RELATIVE AMPLITUDE : refers to the absolute amplitude of wave components which are expressed in relation to one another, eg. comparing the absolute amplitude of wave component V to that of wave component I. This comparison is usually expressed as a ratio, ie. V:I (Fria, 1980).

2.2 HISTORICAL PERSPECTIVE

The origins of the BAER can be traced to the animal experiments of Caton, who in 1875 first reported on the presence of electrical activity in the brain of rabbits, as cited by Glasscock et al.,(1987). Subsequent animal experiments conducted by Danilevsky in 1877 demonstrated spontaneous electrical activity in dogs. Between 1883 and 1891 von Marxow (1890), and Gotch et al. (1891), as cited by Glasscock et al. (1987), pursued the recording of electrical activity of the brain in a variety of animal experiments. However, it was Pravdich-Neminsky who in 1913 first photographed the record of an animal electroencephalogram (EEG), as cited by Glasscock et al., (1987). During this early period of research, experiments relating to evoked potentials were restricted to the use of animals. This may have been due to the lack of sophisticated stimulation techniques, recording techniques and equipment. Furthermore, the then social stigma of "interferring with human heads" may have prevented researchers from using human subjects in their quest to elicit and describe evoked potentials.

Nevertheless, according to Jacobson (1985), the pioneering work of Berger in 1929 led to the recording of the first human EEG. This effort was followed by the work of Loomis, Harvey and Harbot (1938), who first reported on alterations in human EEG patterns brought about by the introduction of sensory stimulation. Thereafter, several workers attempted to identify specific changes in the EEG in response to tactile, visual or auditory stimulation. However, their work was hampered due to the fact that the specific changes were largely obscured by the background fluctuations of the EEG peaks (Gibson and Ruben, 1978).

As a consequence, various techniques were developed to eliminate unwanted bio-electric activity and/or physiological noise from the response recordings. To this end, several attempts were made to extract the "wanted evoked potential" (EP) from the EEG pattern (Dawson, 1954 and Geisler et al., 1958). This process of extracting stimulus-related bio-electric events from ongoing EEG activity set the stage for future research and clinical developments in evoked potential measurements.

Clark (1958), and his co-workers (Clark et al. 1961), developed the "averaged response computer" which enabled the user to accumulate and to extract desired responses. This computer operates on the principle of algebraic summation of bio-electric events elicited by stimulus synchronisation (time locked repetition). This operation functions through a process of analog to digital conversion, whereby EEG voltage is converted and expressed as a numerical value. Furthermore, because the evoked potential is predicated on event related stimuli, it assures a constant time relationship to the signal onset. In contrast, the ever present unwanted noise is random. In this way, the unwanted noise has no time relationship to stimulus onset and thus the EPs can be extracted from the noise of the random and ongoing EEG activity. Currently, this process of digital averaging and summation is favoured by most workers in the field of evoked response measurements (Jacobson, 1985 and Gibson et al., 1978). This process enables one to clearly extract the wanted response from the random ongoing EEG and biological noise.

The advent of the signal averaging computer, improved recording and signal processing techniques as well as the increased interest among researchers, led to important advances in the clinical investigation of auditory evoked potentials (AEPs) (Jacobson et al., 1985).

The decade of the 1960's was particularly significant in the development of AEPs. According to Jacobson et al. (1985), early interest in AEPs was focused on a "slow" (50-200 msec latency) EP, thought to be of cortical origin (Davis et al., 1963). Other AEPs having longer latencies and thought to be related to auditory perception were concurrently identified (Walter et al., 1964). Furthermore, the work of Ruben et al. (1960), and Teas et al. (1962), reflected on the action potential of the cochlear nerve while Goldstein and Rodman (1967), Mendel and Goldstein (1969), studied the middle latency responses occurring between 15 to 50 msec.

The late 1960's and early 1970's were dominated by investigation and clinical application of the brainstem auditory evoked response. According to Jacobson (1985a), the brainstem auditory evoked response should be considered an outgrowth of research activity conducted in the exploration of the cochlear microphonic (CM), the action potential (AP), and the cortical responses. Although early investigators monitoring the CM and AP utilized invasive electrode techniques these were not readily available for routine clinical use. As a result, attention was centred on alternative procedures to surgical recording methods. Among those investigating non-invasive electrode placements were Sohmer and Feinmesser (1967), as cited by Glasscock et al. (1987), who offered the first account of EPs generated from the brainstem. They reported a series of four wave components, the first two peaks comprising the N1-N2 complex of the acoustic nerve action potential (AP). The latter two peaks were of doubtful origin and it was surmised that the responses were either repetitive firing of the acoustic nerve or neural discharge patterns from the auditory brainstem pathway. However, it was due to the efforts of Jewett (1970), and colleagues (Jewett and Romano, 1972; Romano and Williston 1970; Jewett and Williston, 1971) that a clearer picture of the brainstem auditory evoked response emerged. They provided evidence that peaks II through to IV were in fact brainstem responses which had been picked up by using non-invasive far-field electrodes. In 1971, Jewett and Williston demonstrated that the normal BAER consisted of five to seven vertex-positive peaks occurring within the first 10 msec following click stimulation. See figure 1 below for the early classic response as described by Jewett et al., (1971).

According to Jewett et al. (1971), this wave series was consistent among a group of normal hearing subjects that they studied. Furthermore, on repeated testing the response series was consistent within each subject. Peak V was the most prominent component of the response, and the most robust in it's resistance to the effects of stimulus repetition rate. Peak VI was a fairly consistent part of the response, while peak VII occurred inconsistently across subjects. In addition, it was discovered that moving the vertex electrode seven centimetres anteriorly or laterally, did not affect the response, thereby confirming that the BAER is a far-field response ie. the potentials arise from distant neural generator sites. (Jewett et al., 1971).

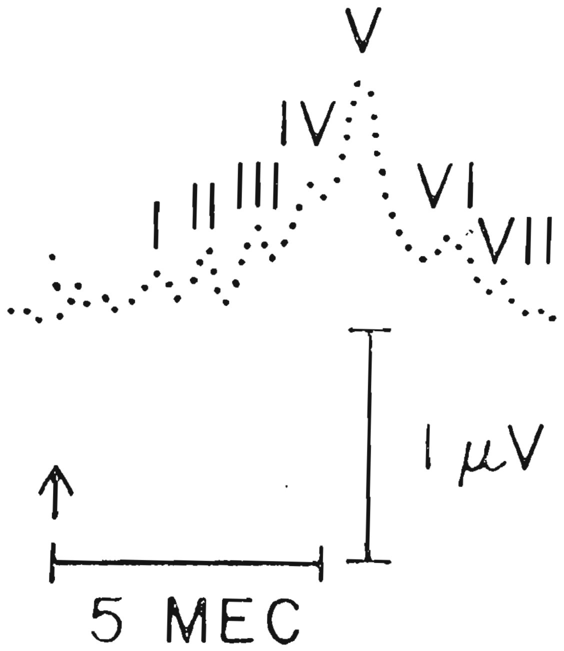


Fig. 1 : Early classic response (Jewett et al., (1971). Adapted from Fria, (1980).

2.3 DESCRIPTION OF THE BRAINSTEM AUDITORY EVOKED RESPONSE (BAER).

Glasscock et al. (1987), described the BAER as a fast response series occurring in the first 10 to 12 msec after stimulation. Jacobson, (1985) concurs with the above and adds that the BAER is a far-field response by virtue of the fact that the monitoring electrodes attached to the scalp are removed from the site of the electric field source. Effectively, the potentials picked up by the scalp electrodes are transduced electrical impulses arising as a result of acoustic (click) stimulation. In other words, the electrical activity at the scalp relates directly to processes in the nerve cells and fibre tracks of the auditory periphery, cochlear nerve and the brainstem (Glasscock et al., 1987). The activity in the neural "generators" (see page 29) is then transmitted to the auditory cortex via the auditory pathway (see page 24 for a description of the pathway). The "auditory" electrical activity at the cortex is embedded within the ongoing EEG (representing about 1% of the total brain activity) (Stockard et al., 1978).

The scalp electrodes, therefore, pick up electrical potentials which have passed through cerebro-spinal fluid; the membrane covering the brain and internal skull; the bony skull and tissues of the scalp (Hampton, 1984). The response waveform is made visible via time-locked averaging following stimulation (Glasscock et al., 1987). Hence, the BAER profile has been described as a volume conducted response (Jewett et al., 1971).

A review of the literature reveals that the response peaks are often labelled with Roman numerals after the convention of Jewett et al., (1971). There are five to seven peaks in a normal subject occurring at 1 msec intervals beginning at about 1.5 to 2 msec (Fria, 1980; Jacobson 1985, and Glasscock, et al., 1987). According to Glasscock et al. (1987), the BAER profiles synchronous discharge to an appropriate stimulus at threshold and suprathreshold levels.

Figure 2 illustrates the normal averaged volume conducted BAER pattern reflecting the different peaks and their respective approximate latencies.

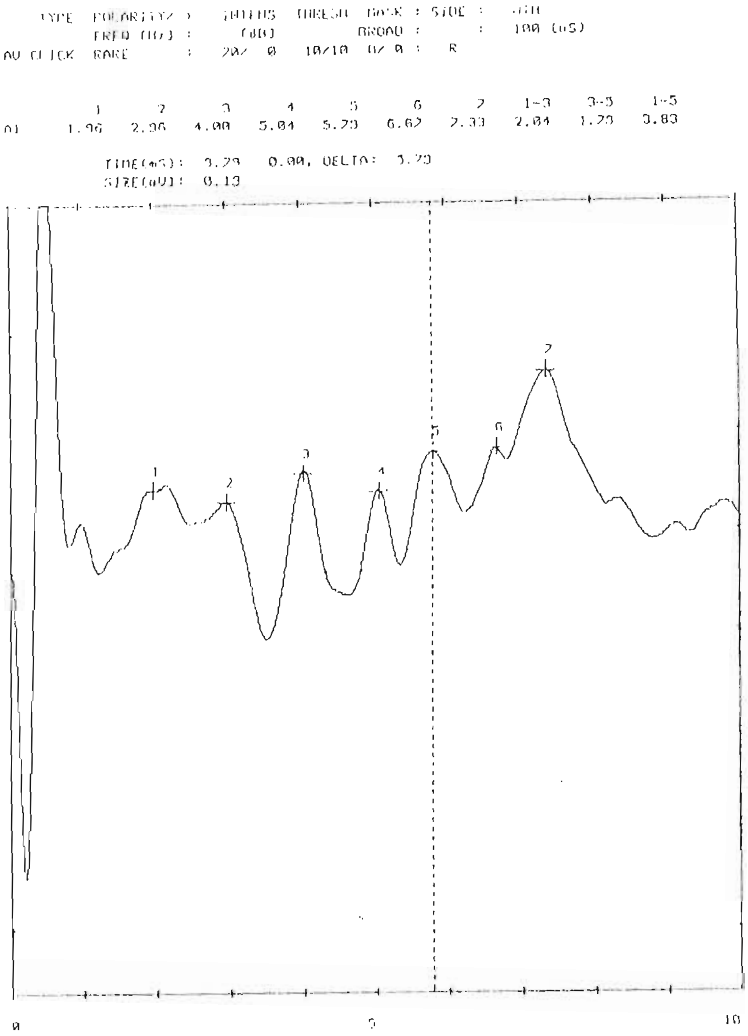


Fig. 2 : Normal average volume conducted BAER - peaks and latencies. Obtained from a 23 year old female subject.

2.4 ANATOMICAL CONSIDERATIONS

The usefulness of the BAER in clinical work depends upon knowing the anatomy of the brainstem, the described auditory afferent pathway and the origins of the various components of the response. A brief review of information available in these areas is therefore presented in the ensuing discussion.

2.4.1 Gross anatomy of the auditory brainstem

Musiek and Baran (1986), offer the following gross anatomical description of the brain stem. The brainstem is composed of several structures which encompass the ascending auditory pathway. Proceeding in a caudal to rostral direction, the auditory brainstem is composed of the cochlear nuclei, superior olivary complex, the lateral lemniscus in the pons, the inferior colliculus in the mid-brain and the medial geniculate body in the thalamus. There are also other structures posterior to and within the brainstem that play a role in auditory function, e.g. the reticular formation which is a medial structure within the brain stem extending from the mid-brain to the spinal cord. It is made up of diffuse aggregations of nerve cells positioned in the midst of dense nerve fibre tracts in the brainstem. This brainstem structure has multiple direct and indirect inputs from various brainstem auditory nuclei. The reticular formation appears to play a major role in auditory alertness, reflexes and habituation.

2.4.2 Afferent (ascending) auditory pathway

Electrical impulses generated from the receptor hair cells in the cochlea are transmitted to the cerebral cortex via the acoustic nerve and subsequent neurons in the brainstem and mid-brain (Jacobson et al., 1985). This pathway includes at least four major synaptic nuclei. The major components of the pathway are duplicated on either side of the cranial midline. These neuronal connections serve to propagate impulses and also play an important role in the interpretation and integration of sensory stimuli (Jacobson et al., 1985). Figure 3, shows a rough synaptic scheme of the afferent pathway.

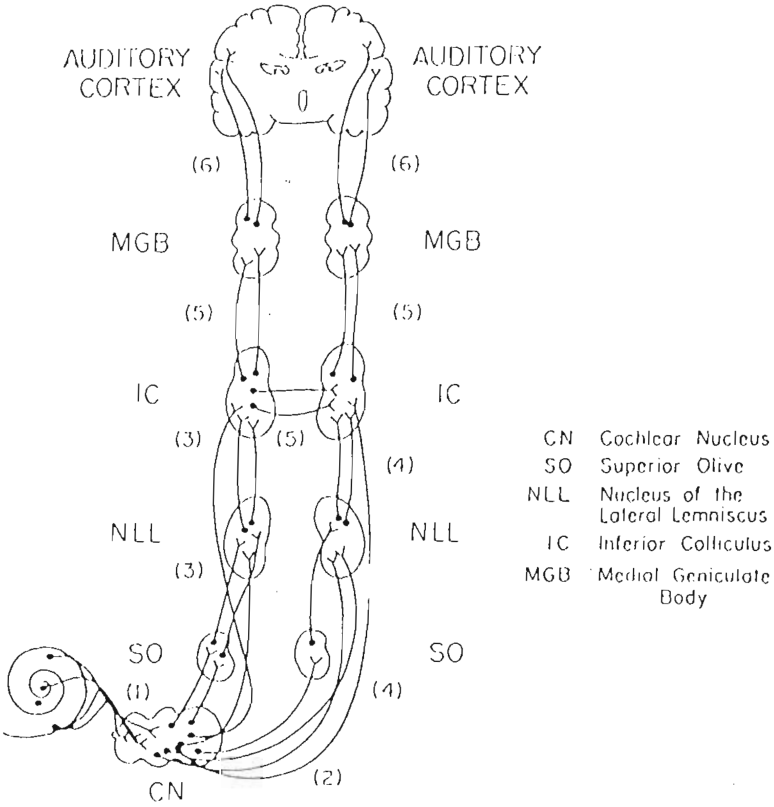


Fig. 3 : Synaptic scheme of the ascending auditory pathway. Adapted from Zimmerman, (1985).

The acoustic nerve consisting of some 30,000 nerve fibres serves to transmit afferent neurologic information from the inner and outer hair cells of the cochlea to the brainstem. These nerve fibres enter the brainstem at the level of the pons and terminate with secondary neurons in one of the divisions of the cochlea nucleus (CN:1) (Mountcastle, 1980). The CN is divided into three sections, viz. the dorsal cochlea nucleus (DCN), the anteroventral cochlea nuclear (ACN) and the posteroventral cochlea nucleus (PCN) (Brugge, 1980 and Warr, 1982). Second order neurons leave the CN in three acoustic stria, viz:

a. The dorsal stria (Manokov's area:2) originates in the DCN and passes through the reticular formation to the opposite side to join the medial portion of the contralateral lemniscus and inferior colliculus (IC) (Osen, 1969 and Bredberg, 1981). Fibres from the dorsal stria also project ipsilaterally, to synapse at the IC (Warr, 1966 and 1969), as cited by Jacobson et al.,(1985).

b. The intermediate stria (commisure or tract of Held) arises from the posteroventral cochlear nucleus, sending fibres bilaterally to the Superior Olivary Complex (SOC) and lateral lemniscus (Osen, 1969). Therefore, the intermediate stria provides secondary avenues for decussation (crossing fibres).

c. The principal bundle, the ventral stria (trapezoid body) project fibres from the anteroventral cochlea nucleus to decussate the midline and terminate at the trapezoid body nucleus, the medial superior olivary, and continues to the lateral lemniscus and IC (4). Additional fibres ascend ipsilaterally to the lateral superior olive. Thus, there is parallel and bilateral representation to the IC (3 and 4) (Zimmerman, 1985).

The next major relay centre is the superior olivary complex (SOC), consisting of three major nuclei viz. the superior olive, the medial nucleus and the nucleus of the trapezoid body. There is an intricate network of interneurons within this complex (Jacobson et al., 1985). Furthermore, the superior olive is the first anatomic site of integration of DICHOTIC auditory input, and conveys signals from both cochleas to the more rostral brainstem structures.

According to Goldberg and Brown (1968), the medial olive receives input from both cochlea and is sensitive to low frequency stimuli. Van Noort (1969), as cited by Jacobson et al. (1985), suggests that the lateral superior olive receives input from the ipsilateral ventral cochlea nucleus and according to Brodal (1981), is sensitive to high frequency inputs. Ascending auditory fibres (3)from the superior olivary complex and the cochlea nuclei then travel through the lateral lemniscus (LL) tract to synapse at the IC. The LL has two major nuclei viz. the inferior ventral nucleus and the dorsal nucleus. The inferior ventral nucleus receives projections from the ventral cochlea nucleus and bilateral innervation from the olivary complex (van Noort 1969, and Warr, 1969), as cited by Jacobson et al., (1985). The dorsal nucleus is supplied with bilateral input from the lateral and medial superior olives and the dorsal cochlea nucleus. There is also a connection between the dorsal lemniscal nuclei on either side of the brainstem called the commissure of Probst, which allows for further decussation for select ascending fibres (Jacobson et al., 1985).

The next major relay station is the inferior colliculus (IC) and serves to receive second and third order neurons (Osen 1972; Adams, 1979). The IC has two divisions, viz. the:

a. central nucleus which is further subdivided into the dorsal and ventral regions and

b. lateral nucleus (Morest, 1964).

The ventral region of the central nucleus receives sensory innervation from the contralateral ventral cochlea nucleus via the lateral lemniscus (Osen, 1972). Less is known about the other nuclei. However, each division shows inter-neuronal connections and the two inferior colliculi communicate via the commissure of the inferior colliculus (Jacobson et al., 1985). According to Zimmerman (1985), at the inferior colliculus the auditory pathway ascends both ipsilaterally and contralaterally (5). The contralateral pathway crosses the commissure of the IC to the IC and MGB on the opposite side. The brachum of the IC is then the ipsilateral pathway to the MGM (Jungert 1958, and Brodal, 1981).

The medial geniculate body in the thalamus is the final synaptic relay station of the primary sensory pathway to the auditory cortex. All ascending auditory fibres synapse at the geniculate nuclei. The geniculate nuclei, viz. the ventral, dorsal and medial, then project ipsilateral fibres to terminate in the homolateral gyrus of Heschl (Brodal, 1981).

It must be noted that the above description of the primary afferent auditory pathway is perhaps simplified. Several researchers have described it as being extremely complex, having ipsilateral, and contralateral ascending fibres which is further complicated by the presence of several inter-connections as it ascends to the cortex.

2.4.3 Anatomical neural generator sites of the BAER

From the outset, various investigators have speculated about the origins of the BAER component peaks (Fria, 1980). Consequently, several investigators have attempted to verify experimentally the neural generators of the BAER. Hall in 1984, contends that there is no consensus on the location of neural generator sites. Accordingly, he suggests that the resultant lack of consensus regarding the neural generators of the series of peaks in the BAER, seems to be directly related to the complex nature of the ascending auditory system in man.

A review of literature pertaining to the decade of the 1970's, depicts the notion that sequential activation of the brainstem structures (eighth nerve, cochlear nuclei, superior olivary complex, nuclei and tracts of the lateral lemniscus, inferior colliculus and the medial geniculate body) are associated with each wavelet or peak of the BAER. That is, peak I with the cochlear nerve, peak II with the cochlear nucleus, peak III with the superior olivary complex, peak IV with the lateral lemniscus, peak V with the inferior colliculus and peak VI with the medial geniculate body. Figure 4 depicts this one to one correspondence.

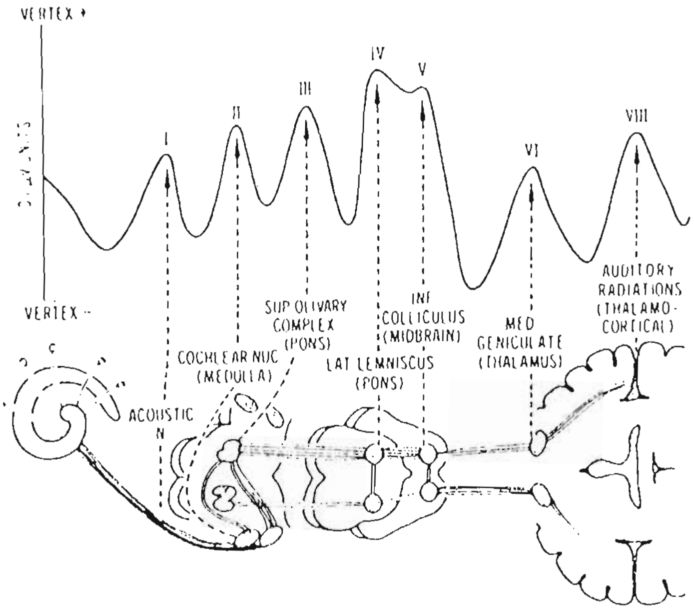


Fig. 4 : The presumed correspondence between BAER component peaks (I to VII) and anatomical structures in the primary acending pathway. Adapted from Fria, (1980).

However, in recent years, this simple schema has come under closer scrutiny, and recent research suggests that most of the BAER wave components have multiple, concurrent generators (Achor and Starr 1980; Britt and Rossi 1980, and Mϕller et al., 1981).

Subsequently, Hall (1984), reviewed the literature and presented the views of the early 1980's. It was generally felt and accepted that the wave I component was a far-field representation of the action potential of the auditory nerve. This contention was supported by Achor and Starr, (1980); Buchwald and Huang, (1975); Hashimoto et al., (1981); Jewett et. al., (1971) and Møller et al., (1981). As regards the peak II component, clinical and experimental data provided by Achor and Starr, (1980); Hashimoto et al. (1981), and Møller et al., (1981), suggested that it was generated by the intracranial portion of the auditory nerve and perhaps by the cochlear nucleus. However, Møller et al., (1981) argued that peak I originates from the distal portion of the eighth nerve and peak II from the proximal part of the nerve as it enters the brainstem. Presently, it is generally accepted that peak I originates from the distal portion of the coclear nerve (Møller and Janetta, 1985).

The generator sites for wave components III, IV, V and VI were disputed. Peak III was traditionally associated with neural activity in the superior olivary complex contralateral to the stimulus (Buchwald and Haung, 1975). However, Achor and Starr, (1980) reported on substantial activity in the ipsilateral superior olivary complex, while Gardi and Bledsoe, (1981) concluded that peak III arises from the contralateral medial nucleus of the trapezoid body. Therefore, there has been no consensus as to the origin of peak III. Hall in 1984, attributed it's origin to activity in the brainstem, probably in the pons, with no clear generator site. This contention is supported by Hashimoto et al., (1981). The generator site for peak IV is less definite and may be attributed to activity in the lateral lemniscus and/or it's nuclei (Hall, 1984). However, Hashimoto et al., (1981) and

Kevanishvili (1980), suggested that it was possible that peak IV arose from the pons, and in fact, was subleminscal. Traditionally, peak V was associated with activity in the inferior colliculus (Buchwald & Huang, 1975; Hashimoto et al., 1981; Starr and Hamilton, 1976; Stockard and Rossiter, 1977). This theory is supported by intracranial depth electrode recordings in man (Hashimoto et al., 1981). However, Achor and Starr in 1980, contested this theory by recording a peak V component obtained after destroying the inferior colliculus in cats. This suggested that peak V is probably generated by sub-inferior colliculi structures. The latter is supported by Kevaneshvili, (1980; 1981).

Finally, there is the long standing confusion as to whether the BAER peaks III to V arise from brainstem structures either ipsilateral to or contralateral to the stimulus (Berry et al. 1976, and Møller and Janetta 1982 and others). This position has not been clarified as yet and remains a topic for future research.

As regards peak VI, there is no agreement as to it's origin and may, in fact, be the result of combined activity arising in the inferior colliculus and the medial geniculate body.

It can be concluded that as at 1984, the general consensus was that peaks I and II emerged from the eight nerve itself, whilst no specific generator sites could be attributed to peaks III to VI. It may however, be presumed that the brainstem components (peak III to VI) arise from multiple concurrently active neural sources within the brainstem, rather than by a successive activation of auditory pathways and nuclei.

Currently, Mollers' (1985) work, as cited by Musiek at Baran (1986), on human intra-cranial recordings, represents an important advance in establishing the origins of BAER components. Mϕller's investigation indicates that peak I is generated from the lateral- distal aspect of the auditory nerve. Peak II originates from the medial - proximal aspect of the cochlear nerve. Peak III probably has more than one generator, as do most of the other subsequent peaks of the BAER. However, Mϕller states that the cochlear nucleus is the principal generator for peak III. Peak IV, according to Mϕller, also has multiple generator sites, but that it arises primarily from the superior olivary complex with a stronger influence coming from the contralateral side of the brainstem. According to Mϕller (1985), peak V is generated primarily from the lateral lemniscus with some contribution from the inferior colliculus. No mention is made of the generator site for peak VI but it is reasonable to asssume that the inferior colliculus is the primary contributor to this peak. Figure 5 depicts a simple scheme of current thinking relating to the origins of the peak components of the BAER.

However, Musiek and Baran (1986), warn that this is still an oversimplified scheme for deriving origins of the

BAER. They state that the BAER is generated from synchronous discharges along the auditory nerve and brainstem pathway, and that observed responses may arise from a number of different structures in the brainstem and not from any one structure.

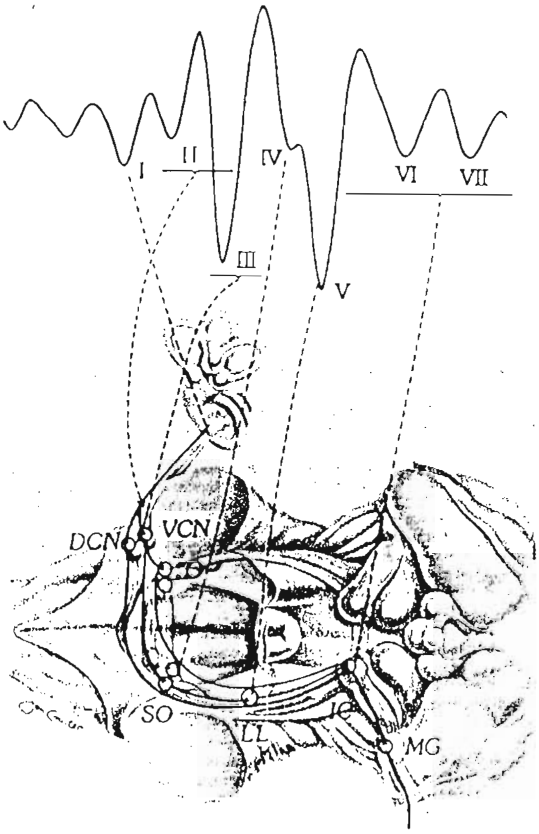


Fig. 5 : Schematic illustration of the neural generators of the BAER in man. Adapted with permission from Møller and Janetta, (1985).

Nevertheless, Musiek and Baran (1986), do suggest that there is "a good possibility that the first five peaks of the BAER may be generated within the auditory nerve and pons."

The above is a discussion of the salient anatomical considerations as related to the BAER. The ensuing presentation will reflect on the normal aspects of the

BAER, encompassing a review of some of the non-pathological factors that may alter and/or confound the elicitation and interpretation of the normal BAER.

2.5 CHARACTERISTICS OF THE NORMAL BAER

The use of the BAER for clinical and research purposes requires the ability of one to recognize abnormal results. Such recognition depends on a knowledge of normal BAER characteristics (Fria, 1980). Furthermore, the clinician must also be cognizant of the variability of normal characteristics both between and within subjects, and of variability due to non-pathologic factors (Fria 1980). According to Schwartz and Berry (1985), the major physical characteristics of the BAER that are used in interpretation include :

1. Waveform morphology,
2. Latency in milliseconds, including:
    - a) absolute latency and
    - b) relative (interpeak) latency and
3. Amplitude in microvolts, including:
    - a) absolute amplitude and
    - b) V:I amplitude ratio

Diagnostic decisions are often based on alterations of one or more of these response parameters.

2.5.1 Waveform morphology

According to Fria (1980), and Schwartz and Berry (1985), morphology refers to the visual appearance or actual shape of the averaged response. It is a more subjective parameter than either latency or amplitude, because morphology cannot be quantified and is, therefore, at best a qualitative descriptor.

In it's clinical application, due cognizance should be given to labelling the peaks. The convention is to follow Jewett & Williston's (1971), suggestion of using Roman numerals to identify the series of peaks. Furthermore, normal morphologic variations must be considered in interpretations. Several investigators have observed that peaks IV and V often are fused together to form the "IV-V" complex (Fria, 1980). However, this is not always apparent (Schwartz and Berry, 1985). Figure 6, exemplifies the morphologic variations that one can obtain on normal subjects. Other morphologic variations include a complete absence of a peak as seen when peak IV combines totally with peak V (as seen in C) or there may be a bifurcation of peaks I and/or III as seen in figure 6, H and G respectively. Of particular importance is that response morphology can be affected by age, pathology and the intensity level of the stimulus i.e. a high stimulus level leads to a larger amplitude measurement and vice-versa (Schwartz and Berry, 1985). Essentially, alterations in peak morphology represent "soft" clinical signs of pathology (Schwartz and Berry, 1985).

Normal variations in the ABR. (A) wave V is riding on the down-shoulder of wave IV; (B) wave IV is riding on the up-shoulder of wave V; (C) waves IV and V appear as a broad undulation (M configuration) with wave V amplitude less than IV; (D) wave V amplitude is greatly reduced from peak IV; (E) the classic IV/V complex; (F) waves I and II amplitude greater than IV/V; (G) bifid wave III; (H) bifid wave I and fused IV/V.

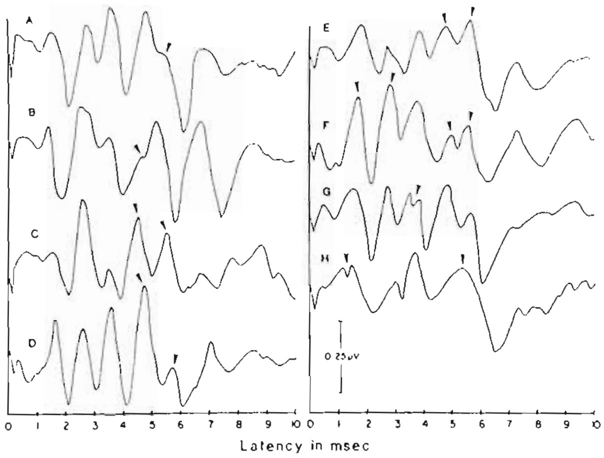


Fig. 6 : Morphological variations in the normal BAER. Adapted from Chiappa et al., (1979).

2.5.2 Peak response latency

The time relationship between the onset of the eliciting stimulus and response is commonly referred to as latency (Fria, 1980 and Schwartz and Berry, 1985). In BAER testing latency may be designated into:

a. absolute latency and

b. relative or interpeak latency.

Absolute latency, according to Schwartz and Berry (1985), is the time period (msec) between the onset of the acoustic stimulus and the peak of the averaged response. Interpeak latency (IPL), however, refers to the time difference in msec between two primary peaks (e.g. I-III; III-V; or I-V ; Fria, 1980). Figure 7 illustrates the manner in which absolute and relative latencies and amplitudes of the BAER are measured (Schwartz and Berry, 1985).

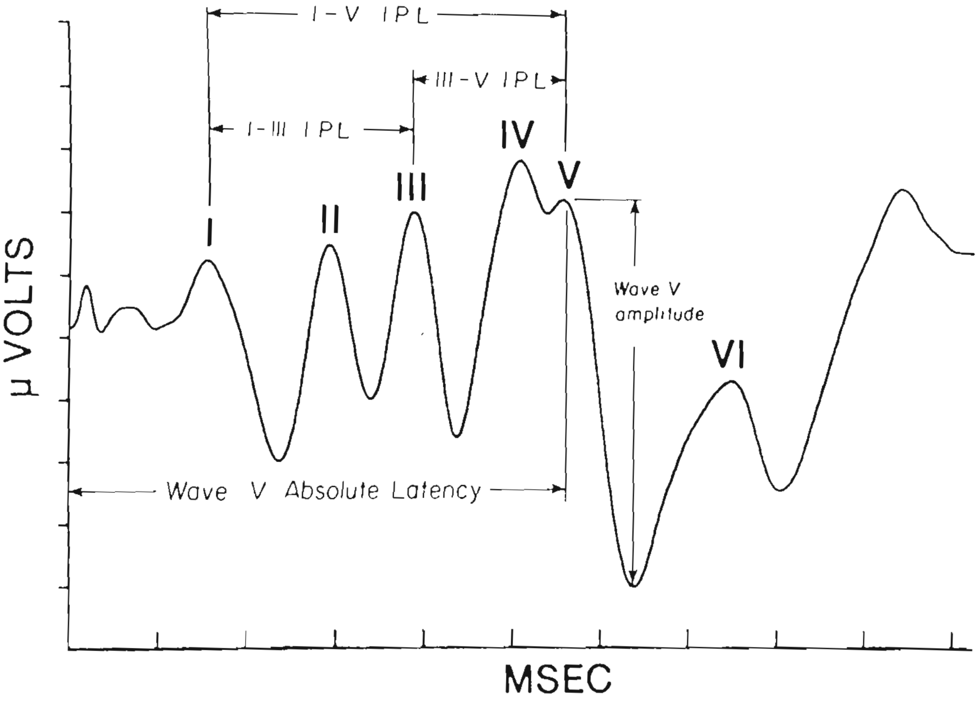


Fig. 7 : Measurements of absolute and relative latencies and amplitudes. Adapted from Schwartz and Berry, (1985).

Table 2 : Normative ABR latency data across 11 laboratories. Adapted from Schwartz & Berry, (1985).

Laboratory/Clinic	1	2	3	Filter settings (Hz)	Wave latency (ms)							
					I	II	III	IV	V	I-III	III-V	I-V
Jewett and Williston, (1970)	60-75	?	?	10-10,000	1.5	2.6	3.5	4.3	5.1	-	-	-
Lev and Sohmer, (1972)	65	?	?	250-5000	1.5	2.5	3.5	-	5.0	-	-	-
Picton et al., (1974)	60	?	10	10-3000	1.5	2.6	3.8	5.0	5.8	-	-	-
Starr and Achor, (1975)	65	alt.	10	100-3000	1.6	2.8	3.8	4.8	5.5	-	-	-
Stockard and Rossiter, (1977)	60	rar.	10	100-3000	1.9	3.0	4.1	5.2	5.9	2.1	1.9	4.0
Rosenhamer et al., (1978)	60	?	16.6	180-4500	1.7	2.9	3.9	5.2	5.9	2.26	2.0	4.27
Row, (1978)	60	?	10	100-3000	1.9	2.9	3.8	5.1	5.8	1.97	1.97	3.94
Gilroy and Lynn, (1978)	75	?	11	150-3000	1.55	2.67	3.60	4.69	5.40	2.05	1.9	3.83
Beagley and Sheldrake, (1978)	70	?	10	250-3200	2.1	3.3	4.3	5.3	6.1	2.2	1.8	4.0
Chiappa et al., (1979)	60	alt.	10	100-3000	1.7	2.8	3.9	5.1	5.7	2.1	1.9	4.0
Schwartz and Berry, (1985)	75dB (nHL)	rar.	11.1	75-1500	1.65	2.85	3.8	4.99	5.66	2.05	1.85	4.00

1 = Stimulus intensity level in dB
2 = Stimulus polarity
3 = Repetition rate (cps)

Table 2, summarizes the absolute and IPL's of responses recorded from normal hearers as reported by 11 different investigators. The number of subjects, intensity of the clicks, filter settings, stimulus polarity and repetition rates used in each study are indicated. Although not indicated on the table, the standard deviation for each of the absolute latencies was reported by the following researchers (Starr and Achor, 1975; Rosenhammer et al., 1978; Rowe, 1978; Stockard et al., 1978 and Chiappa et al., 1979) to be approximately 0,3 msec. An average of the mean standard deviation for the interpeak latencies was 0,2 msec (Fria, 1980). However, the observed variation between studies might reflect differences in the number of subjects evaluated, and/or click intensity, filter settings, stimulus polarity and repetition rates employed. Despite these differences, the data in Table 2 demonstrate a notable trend. The peaks occur at approximately 1 msec intervals from roughly 1,5 msec to 6.1 msec in response to high intensity clicks.

In the light of clinical application, a review of the literature clearly reveals that the absolute latency of peak V, the rostral component of the BAER, has received widespread attention. The importance of peak V relates to its robust character and reliability under varying measurement conditions (Schwartz and Berry, 1985).

Crucial to the differential diagnosis of space occupying lesions, either intrinsic or extrinsic to the brainstem, is the time difference between two peaks viz. I-III;

III-V and I-V, referred to as interpeak latencies. This time difference, in part, reflects the neural conduction time and has been referred to as "central transmission" or "brainstem transmission" (Schwartz and Berry, 1985). According to Fria (1980), the I-III interpeak latency value indicates transmission time through the ponto-medullary junction and lower pons, the III-V value indicates transmission time from caudal pons to caudal midbrain levels and the I-V interpeak latency value reflects the time needed for the impulses to travel from the auditory nerve to the caudal midbrain. A review of the literature on IPLs reveals that an approximate 2,0 msec difference exists between peaks I-III and III-V and a difference of about 4,00 msec for the I-V IPL in normal hearers.

2.5.3 Waveform amplitude

Waveform response amplitude refers to the height of a given peak component and it is usually measured in microvolts from the peak of the wave to the following trough (Fria, 1980). This measurement is referred to as the absolute amplitude of a peak. See figure 7 for an illustration of this measurement. According to Schwartz and Berry (1985), the use of the measurement of amplitude has not met with a great deal of clinical success, owing to the variability of the measure. Rowe, (1978), states that amplitude values do not appear to be normally distributed, are highly susceptible to myogenic activity and noise levels, are difficult to replicate and are influenced by minor alterations in recording technique. Consequently, the measurement of absolute amplitude does not enjoy the stability and reliability when compared to it's latency counterpart (Schwartz and Berry, 1985).

The alternative to the absolute amplitude measure which has gained increased clinical acceptance in recent years, is the calculation of the relative I-V amplitude ratio. In normal patients, peak V is usually greater in amplitude than peak I, resulting in an amplitude ratio >1.00 (Chiappa et al., 1979; Rowe, 1978; Starr and Achor, 1975). Hence, a I-V ratio of <1.00 is considered abnormal (Musiek et al., 1985). However, Schwartz and Berry are of the opinion that further research is required into investigating the confounding effects of such variables as stimulus polarity, repetition rate, filter characteristics, electrode sites etc., prior to the general use of this relative amplitude measure in clinical practice.

In summary, it is clear that the accurate and reliable interpretation of the BAER, is based on the quantitative and qualitative assessment of waveform latency, amplitude and morphology. Of these, the measurement of absolute and IPLs offer greatest clinical value in assessing the integrity of the auditory system. For the broader clinical use of the amplitude measures, additional research is required to identify all sources of normal variability, prior to their adoption as useful measures. In addition, though the assessment of waveform morphology is subjective in nature, it can provide valuable information and insight into lesions in the retro-cochlear and central auditory system, but it is best performed by an experienced clinician (Schwartz and Berry, 1985).

2.6 NON-PATHOLOGICAL FACTORS INFLUENCING THE NORMAL BAER

Due to variations in recording parameters and methods used to measure the BAER, it is imperative that each individual clinical facility establish it's own reference data (Schwartz and Berry, 1985). Several investigators have reported on the myriad of variables that can potentially alter one or more of the important parameters of the BAER and hence lead to misinterpretations. Therefore, it is important to review some of these influencing variables and to note their effects on the normal BAER. These variables maybe broadly divided into:

a. technical parameters

b. procedural parameters and

c. subject parameters.

2.6.1 Technical parameters

2.6.1.1 Stimulus type

According to Hall (1984), the click, which has an instantaneous onset and very brief duration, is well suited for generating synchronous neuronal firing required to elicit the BAER. Any increase in rise time tends to increase the latency of the BAER, decrease it's amplitude and cause the morphology to deteriorate (Hall, 1984).

Furthermore, Cobb et al. (1978), Huang and Buchwald (1978), and Stapells and Picton (1981), have found that stimulus rise time in excess of 5 msec may fail to generate a recognizable response. Furthermore, since the BAER is an onset response, changes in stimulus duration and fall (decay) time have no influence on the response (Hall, 1984).

2.6.1.2 Stimulus polarity

The results of empirical investigations concerning the effects of stimulus polarity on the BAER have been ambiguous (Schwartz and Berry, 1985). Terkildsen et al. (1973), showed no consistent latency differences between BAER recordings using both rarefaction and condensation click stimuli. However, Orntz and Walter (1975), reached the opposite conclusion when they described a consistent shortening in peak V latency on the order of 0,4-0,8 msec to rarefaction versus condensation clicks. Generally, research studies have failed to reveal any systematic alteration in the peak latency of peak V with click phase inversion (Borg and Lofqvist, 1982). This suggests that one could use both rarefaction and condensation clicks to identify peak V with no substantial change.

However, several studies have suggested significant effects on the earlier peaks i.e. I-IV. Stockard et al. (1979), and Ruth et al.(1982), have shown that peak I tends to show decreased latency, increased amplitude and improved resolution when using rarefaction clicks. This shortening of peak 1 latency obviously, effects subsequent interpeak latencies viz. I-III, III-V and I-V latencies.

Furthermore, Stockard et al. (1979), found that changing click polarity from rarefaction to condensation influences the morphology of the IV-V peak complex. They found that in using rarefaction as opposed to condensation clicks, peak IV became more prominent. Stockard et al. (1979), suggests that alternating clicks should be used to minimize electrical and mechanical artifacts and also to resolve peak I, or to determine it's absence. Schwartz and Berry (1985), concur with the above, and state that alternating clicks tend to produce optimal responses and do not significantly change the response characteristics of the BAER. However, Hall (1984), suggests that one should routinely document stimulus polarity in normative and clinical BAER measurements and that one should not rely solely on a single polarity stimulus. Therefore, it is apparent that normative data for each polarity should be obtained in each clinical setting.

2.6.1.3 Stimulus repetition rate

Numerous investigators have reported on the effects of changing repetition rate on the latency, amplitude and morphology of the BAER. Jewett and Williston (1971), first described the alteration in morphology as a function of increased repetition rate from 2.5 to 80per sec. Pratt and Sohmer (1976), reported decreased amplitudes and increased latencies of peaks I to V with increasing repetition rates. This finding was supported by Zollner et al., (1976).

Generally, increases in rate of stimulation above approximately 20per sec result in a diminution in amplitude for the early components of the BAER (peaks I-III) with little effect on the more rostral component, peak V, until stimulus rate exceeds approximately 30per sec (Schwartz and Berry, 1985). More importantly, the latency of essentially all BAER components appears to increase by approximately 0,4 msec as repetition rate inceases from 10 to 80per sec (Van Olphen et al. 1979, and Stockard et al., 1979).

For clincial use, it appears that the latency of peak V is not seriously affected until stimulus rate exceeds 30per sec (Hyde et al., 1976). Although this is the case, it is suggested that for routine clinical assessment repetition rates of 10 to 20per sec. should be used (Schwartz and Berry, 1985, and Stockard et al., 1978). This would serve to preserve all component peaks and at the same time have no effect on latency, morphology and amplitudes of peaks. However, faster rate presentations are desirable to detect incipient abnormalities of the brainstem pathway (Gerling and Finitzo-Heiber 1983, and Stockard et al., 1978).

2.6.1.4 Stimulus intensity

The most striking characteristic of the BAER is it's sensitivity to the intensity of the acoustic stimulus. A decrease in stimulus intensity results in an increase in response latency and a decrease in the amplitudes of all BAER component peaks (Jewett and Williston, 1971; Hecox and Galambos, 1974; Picton et al., 1977 and others). Rose (1984), and Worthington et al. (1980), as cited by Schwartz and Berry (1985), have calculated the probability of detecting various peak components as a function of stimulus intensity for normal hearers. In both studies, visual detection of peak V was possible in 75% of the cases at intensities between 10 and 20dBsl and approached 100% at high stimulus levels. Peak III, however, was identifiable at levels of approximately 30dBsl in 50-60% of subjects. Peak I required an intensity of above 50dBsl for easy detection. Therefore, it would appear that one has to present fairly high intensities for all peak components to be visible, especially for peak I, since detection of this peak is crucial for evaluating VIIIth nerve function and for calculating brainstem transmission time (Schwartz and Berry, 1985).

Stockard et al. (1978), reported that peak I latency increased more than peaks III and V when stimulus intensity was decreased. Consequently, interpeak latency values involving peak I (i.e. I-III and I-V) were shorter at lower stimulus levels. The average decrease in the I-III interpeak latency was, 0.19 msec and for I-V it was

0.34 msec. This has an important bearing on clinical diagnostic work.

As regards amplitude reduction, Stockard et al. (1978) observed a 33% reduction in amplitude of the peak IV-V complex when the stimulus was reduced by 50dB; while the same reduction in intensity was associated with a 90% decrease in peak I amplitude. Consequently, the V:I relative amplitude ratio increased with decreasing stimulus intensity. This finding supported Starr and Achors' (1975), original contention.

Generally, it is evident that there is a definite intensity / latency / amplitude effect in eliciting the BAER. That is, as intensity decreases, latency increases and the amplitude of all component peaks decrease.

2.6.1.5 Filter characteristics

As with most bio-electric potentials, the BAER is embedded in a background of competing electrical activity. In fact, the BAER is approximately 1% of the size of the ongoing EEG (Stockard et al. 1978). Consequently, it is essential that the frequency response of the recording system be set to reject the maximal amount of electrical interference. In order to optimize BAER clarity, the signal to noise ratio should be reduced. This is done by band pass filtering (Schwartz and Berry, 1985). However, according to Hall (1984), there is conclusive evidence that the BAER varies as a function of filter settings. A standard amplifier band pass of 100 or 150 to 3000Hz is usually recommended for routine clinical measurement of the BAER. Several investigators have demonstrated that by lowering the high frequency limits of the band to below 3000Hz, it tends to round off the averaged response peaks, with corresponding and significant reduction of BAER latency resolution, thereby offering no advantage to the measurement (Hall, 1984). Likewise, a high pass filter setting of above 150-300Hz is undesirable because it significantly reduces amplitudes and recognizability of the later peak components. If, however, one extends the low frequency cut off to 10 to 40Hz, it serves to enhance peak V, but has the disadvantage of including unwanted neuromuscular activity. This can obscure other component peaks, thereby making interpretation difficult. According to Hall (1984), a 150-3000Hz band pass is adequate, without compromising peak morphology, latency or amplitude. However, he suggests that there should be consistency in filter settings within a facility, and that one should document any deviations from these settings.

2.7 PROCEDURAL PARAMETERS

2.7.1 Time domain averaging

There are several methods of eliminating unwanted electrical activity and improving signal-to-noise ratio from the desired BAER. These include: bandpass filtering (already discussed), artifact rejection, and electrode placement. The single most powerful tool is time domain averaging (Thornton, 1982). The absolute number of averages needed for clear response resolution is dependent on the amplitude of the BAER and on the amount of unwanted cerebral and non-cerebral activity. To avoid disorganization of waveform morphology and decreased response amplitude, Schwartz and Berry (1985), recommend that:

a. at least two averages of 2000 responses be obtained with greater averaging required for threshold measures.

b. a control trial be performed as a baseline comparison with the actual response and

c. artifact rejection of events that exceed the limits of the A.D convertor to be employed.

If the above are not considered then the obtained BAER may be difficult to interpret, especially at lower intensity levels where resolution of the response is poorer.

2.7.2 Transducer types (earphones)

According to Fria (1980), a difference in stimulus transducer can also account for varied reports of normal BAER parameters. A number of studies have used the TDH-39 earphones with MX41/AR cushions, while others have employed the TDH-49 or even more novel transducers. A problem arises because different earphones can have different characteristics eg. a rectangular electric pulse delivered to two different earphones can produce significantly different acoustic signals which can in turn influence the latency of the response obtained (Fria, 1980). According to Schwartz and Berry (1985), an inadequately dampened earphone especially around the resonant frequencies (3000 and 6000Hz) may result in a latency prolongation, reflecting the physical properties of the transducer rather than the function of the auditory system. This could obviously lead to an interpretation error. Therefore, it is recommended that earphones and their physical characteristics be routinely checked before performing BAER testing.

2.7.3 Electrode location

The recording of auditory evoked potentials requires the placement of 3 or 4 surface electrodes, two or three of which are connected to the preamplifier inputs with the other serving as ground. In BAER testing, electrodes are arranged such that the electrical potential difference is measured between pairs of electrodes (bipolar derivation). In single channel recording, 3 electrodes are used, two of which are connected to the preamplifier and the 3rd serving as ground. The active (positive) is placed on the vertex (C_z) or high forehead (F_z), while the reference (negative) is placed on a neutral site such as the ipsilateral earlobe (A_1, A_2) or mastoid (M_1, M_2), with the contra-lateral earlobes or mastoid serving as the site for the ground electrode. See chapter 4, p. 126, figure 9 for the locations for the above named sites.

According to Hall (1984), in volume conducted evoked response recording (as in BAER), the precise location of the vertex (active or positive) electrode is not important. He states that equivalent results are obtained with this electrode located along the midline, from forehead to the occipital region. The only observed difference in moving this electrode from the vertex is that as the electrode is placed further away from the vertex, there is a slight decrease in amplitude of peak V. However, significant changes in BAERs occur with changes in the reference electrode position.

According to Hall (1984), the following changes in the BAER as function of reference electrode position have been observed. N.B. the positive electrode on the vertex remained constant.

a. If the reference electrode is placed on the ipsilateral ear lobe, then peak one is clearly observed, but if the reference electrode is placed on the sternum or on the contralateral earlobe the peak I diminishes in amplitude.

b. Peaks IV and V are not distinctly separate with an ipsilateral earlobe reference electrode, but becomes distinct with a sternum or contralateral earlobe electrode.

c. Peak V shows greatest amplitude if the reference electrode is placed on the sternum than on the ipsilateral or contralateral earlobe.

d. Peak components following peak V appear more distinct with a sternum reference than with an ipsilateral or contralateral earlobe electrode.

Furthermore, Stockard et al. (1978), found that the BAER parameters were markedly altered when recordings were referenced to the contralateral earlobe. They found that peaks I and II decreased in amplitude, peak IV and V were clearly separated and the peak V latency increased. Similar findings were reported by Thornton, (1975). In addition, Terkildsen et al. (1973), and Chiappa et al. (1979), suggested that a non-cephalic site for the reference electrode is unfavourable due to problems related to increased myogenic interference, and inferior signal-to-noise conditions.

It, therefore, seems that there is no single electrode montage that best records all of the major BAER components. However, Schwartz and Berry (1985), indicate that the most acceptable clinical compromise is achieved with a vertex-to-earlobe arrangement. Here again, they suggest that each clinic or laboratory should establish it's own reference data using the same electrode montage.

2.7.4 Bilateral recording of the BAER

Simultaneous ipsilateral/contralateral and binaural measurements of the BAER have received limited research attention despite having great potential to facilitate proper identification of component waveforms (Schwartz and Berry, 1985).

Rosenhamer and Holmkvist (1982), and Thornton (1975), have shown that recordings from the ear opposite that under stimulation shows a peak I which is either diminished in amplitude or is absent. In the same way, peak III is more attenuated than peak II (Stockard et al., 1978). As regards latency, peak III shows a shorter latency and peak V is prolonged on the order of 0.15 msec relative to it's ipsilateral counterpart. Furthermore, the I-III IPL is shortened and the I-V IPL is increased for recordings obtained or the ear contralateral to click stimulation.

An interesting feature is seen during binaural stimulation. There is a marked increase in amplitude of all component peaks, and peak V shows an approximate 50% increase in amplitude. (Dobie and Berlin, 1979).

2.7.5 Effect of contralateral masking

An indepth study of this variable will be presented in Chapter 3 as it forms an integral part of this project.

Thus far, the relative large variation in waveform latency, amplitude and morphology due to technical and procedural factors have been highlighted. This undoubtedly stresses the need for each clinic or laboratory to establish it's own test protocol and data. In the ensuing discussion further evidence is provided to illustrate how the normal BAER parameters may be influenced by individual subject differences, pharmacologic factors, and/or physiologic characteristics which are not related to neurologic disease or auditory pathology.

2.8 EFFECT OF SUBJECT CHARACTERISTICS ON THE NORMAL BAER

2.8.1 Age

During the past decade there have been an number of studies demonstrating the marked alteration in response latency, amplitude and morphology as a consequence of age. It is now well established that infant responses consist of three vertex-positive peaks (I, III and V) having latencies and amplitudes that differ from corresponding adult values (Stockard et al., 1978; Jerger and Hall 1980, and Jacobson et al., 1981). The consensus of these studies is that immaturity in the auditory system is reflected in the BAER by absolute and IPL delays of the three primary components, and a peak I amplitude which is twice that found in adults (Schwartz and Berry, 1985). Such maturational effects become resolved by approximately 12-18 months of age when the BAER begins to assume adult characteristics (Hall 1984, and Schwartz and Berry, 1985).

At the opposite end of the age continuum, data related to changes in BAER characteristics for individuals beyond 60 years are both sparse and conflicting. Beagly and Sheldrake (1978), found no increase in latency across subjects grouped according to decades of age from 11 to 79 years who showed normal hearing. Conversely Rowe (1978), showed that adults in the age range 51-74 years had latency delays in the order of 0.30 to 0.50 msec when compared to adults in the age range 17-33 years. This finding was supported by Thomson et al. (1978), who stated that peak V latency increases at a rate of 0.1 msec per decade of life. However, Rosenhamer et al. (1980), reported no significant differences in peak latency between individuals in the sixth and seventh decades and young adults.

While there is no overwhelming agreement among investigators as to the relationship between old age and the BAER waveform, latency or amplitude, it is reasonable to assume that such alterations could be observed as a result of expected physiological changes that occur with the central auditory system and cochlea with increasing age.

Therefore, in recognition of differences in BAER patterns in infants, in normal hearing young adults and of the dubious findings within the geriatric population, it is imperative that age-relevant reference data commensurate with the population to be studied be acquired, before using the BAER as a routine clinical tool.

2.8.2 Body temperature

Stockard et al. (1978), have reported that hypothermia tends to delay latencies and IPLs of the BAER by approximately 0.7 to 0.9 msec. It is, therefore, suggested that BAER testing be conducted when the subject has a normal or near normal body temperature. No reports have been forthcoming on the effect of excessively increased body temperature on the BAER.

2.8.3 Mental state

Hall (1984), states that the mental state of "normal" subjects do not influence the BAER and is perhaps the reason for the widespread clinical popularity of this evoked response. Normal BAER parameters are reliably recorded if the subject is relaxed, and/or is in a natural state of sleep. This is also true of narcolepsy (Hellekson et al., 1979) and metabolic comas (Hall et al. 1982). This implies that the BAER may be reliably elicited in subjects irrespective of their state or stage of sleep. However, subjects who have suffered auditory deprivation (Decker and Howe 1981), Autism (Rosenblum et al. 1980), and mental retardation (Squires et al. 1980), have all shown differential and abnormal BAER patterns. Furthermore, subjects with Down's Syndrome, though having normal hearing sensitivity show unusually short peak I to III and I to IV latency intervals but prolonged IV to V intervals (Squire et al., 1980).

Therefore, when working with such populations it is important to be aware of possible inherently abnormal brainstem auditory function (Hall, 1984).

2.8.4 Drugs

Most anesthetic agents and CNS depressants, such as barbiturates, exert little or no influence on the BAER even at high doses (Hall 1984, and Stockard et al., (1978). However, alcohol, ethanol and diazepam may alter BAER latency and or amplitude (Rosenhamer et al. 1980, and Adams et al., 1982). Furthermore, Phenytoin, an anticonvulsant, may significantly prolong BAER latencies (Green et al., 1982). Javel et al. (1982), have stated that Lidocaine, (a local anesthetic) may produce prolonged brainstem transmission time. Finally, Toulene tends to produce severe BAER abnormalities as seen in paint sniffers (Metrick and Brenner, 1982). Furthermore, all known ototoxic drugs such as gentamycin, kanamycin, streptomycin have been related to BAER abnormalities (Stockard et al., 1978).

In view of these varied effects of drugs, it is advisable to carefully document the type and dosage of medications in subjects undergoing BAER testing.

2.8.5 Sex effects

A number of investigators (Beagly and Sheldrake, 1978; Jerger and Hall, 1980; McClealland and McCrae 1979, and Stockard et al., 1978) have all shown that there is a marked sex effect on the normal BAER.

Generally, females show shorter latencies and larger amplitudes than males. The difference is greater for peak V than for the earlier peaks, and also produces shorter IPL within females.

The clinical implication of this differential effect is that it is advisable to establish different reference data for males and females to avoid mis-interpretations

(Hall, 1984). The effect of this variable on the normal BAER will be studied in detail in the ensuing chapter.

CONCLUSION

The foregoing chapter highlighted the various concepts, terms and nomenclatures associated with auditory evoked potential testing. Furthermore, a resumé was provided of the historical development of the BAER together with a review of anatomical correlates to be considered in BAER audiometry. In the latter part of the chapter the myriad of variables and the many non-pathologic factors that could influence the measurement parameters of the BAER were exemplified. While some of these variables may appear to have only minimal effects, the synergistic consequences across variables can lead to serious interpretation errors.

In the next chapter an indepth study of the influence of two variables viz. contralateral masking and sex differences on the normal monaural BAER is to be made. These variables are the subject of the present investigation and therefore, a thorough literature review relating to their effects on the normal monaural BAER will be conducted. This would serve to highlight both the past and present knowledge of these variables in the field of BAER testing.

CHAPTER 3

REVIEW OF LITERATURE

MASKING AND SEX RELATED DIFFERENCES IN BAER TESTING

INTRODUCTION

It is evident from the preceding chapter that the BAER in normal hearers is influenced by various technical, procedural and testee effects. Among the many variables cited, suspicion has been cast on the potential influence of contralateral masking and differences in sex on the normally elicited BAER. The forthcoming discussion will reflect on:

i. the stimulus-related variable, viz. contralateral masking, encompassing it's use and suggested effects in BAER testing, and

ii. the subject-related variable, viz. the effect of differences in sex both in the absence and presence of contralateral masking in BAER testing.

Each of the above mentioned variables will be explored in terms of firstly their significance to general clinical audiometric evaluations and then specifically, as related to BAER testing. Thus, a review of pertinent studies will serve to highlight the need for further investigation of these two variables. In so doing, the motivation for this project will become apparent.

3.1 MASKING IN THE CLINICAL CONTEXT

3.1.1 Definition of clinical masking

The American National Standards Institute (ANSI) Standard on Acoustical Terminology S1.1 - (1960), as cited by Studebaker (1973), defines masking as "...the amount by which the threshold of audibility of a sound is raised by the presence of another (masking) sound..." Meyer (1959), added that there was a concomitant reduction in the loudness of a stimulus which occurs under certain circumstances upon the introduction of other signals. Scharf (1964), as cited by Studebaker (1973), used the term "partial masking" to refer to this loudness reduction. Carter and Kryter (1962), as cited by Studebaker (1973), stated that "masking refers to the limits placed on the recognition of a sound by the presence of another sound, when the time and frequency characteristics of both are known". This definition therefore includes intra-aural distortion products as one of the consequences of both stimuli.

Deatheridge and Evans (1969), stated that masking is "the process by which the detectability of one sound, the signal, is impaired by the presence of another sound, the masker". As regards the physiological correlates of masking, Teas et al. (1962), stated that masking noise tends to eliminate part of the cochlear nerve AP in electrocochleography and concluded that, "since noise acts primarily by eliminating portions of the normal response at times appropriate to the frequency characteristics and level of the noise... higher levels remove more of the AP, but they do not appear to change the timing of the response that remains".

Generally, these definitions are concerned basically with the concept of interference with the primary auditory signal by a secondary source and the elevation and/or shift in threshold.

3.1.2 Rationale for use of masking.

The application of clinical masking is often essential during audiometric testing. The need to mask arises when an individual being tested demonstrates substantial threshold differences between ears (Goldstein and Newman, 1985). Because the goal in audiometric measurements is to obtain valid and reliable results, the audiologist must evaluate each ear independently. This separate evaluation of each ear is important for diagnostic and rehabilitative decisions. Therefore, in the clinical setting, audiologists rely on the use of masking noises to ELEVATE the non-test ears (NTE) threshold without interfering with or influencing the audiometric results of the test ear (TE) (Goldstein and Newman, 1985). The rationale for this is to eliminate the participation of the NTE when evaluating the TE. Specifically, a second sound source (usually some type of noise presented via the air-conduction mode through an earphone) is employed to shift the sensitivity of the non-test cochlear to prevent the NTE from responding when presenting a signal to the TE. This therefore, implies that the use of masking is essential in cases of unilateral or asymmetric bilateral hearing loss (Sanders, 1978; Hodgson 1980 and Goldstein and Newman, 1985).

Furthermore, when a signal is presented to the poorer ear (TE) at a sufficiently loud intensity level, the possibility of crossover exists. Without the employment of masking in the better ear (NTE), subjects will respond to hearing the signal in the (NTE), since air conducted stimuli that have crossed over from the poorer ear will actually shadow (mimic) the thresholds of the better ear. These "mimicked" responses of the better ear are elevated by the amount of the interaural attenuation at each frequency. When this crossover occurs, then the obtained thresholds for the poorer ear (TE) are better than the "TRUE" thresholds. Therefore, without the application of appropriate masking procedures, validity of the test findings are questionable. The consequences of failing to mask or the inappropriate use of masking, have potentially serious negative ramifications on both medical and audiological management (Goldstein and Newman, 1985).

3.2 MECHANISM OF CROSSOVER

Crossover responses may essentially occur via a peripheral mechanism, i.e. via bone conduction (Hodgson, 1980; Sanders, 1978 and Goldstein and Newman, 1985) and/or via a central mechanism, i.e. via neural interaction (Zwislocki, 1953; Dirks and Malmquist,1969 and Linden et al., 1959).

3.2.1 Peripheral crossover

Peripheral crossover, according to Hodgson, (1980) depends on the:

i. intensity of the test signal
ii. sensitivity of the NTE's cochlea
iii. reduction of the signal as it travels across the head (i.e. interaural-attenuation).

The crossover route ordinarily is by bone conduction. Zwislocki (1953), established that, even for a signal which originates from an earphone, the crossover route to the NTE is by bone conduction. i.e. the vibration of the earphone-cushion which is pressed against the skull tends to stimulate the cochlea of the NTE by bone conduction. This crossover is more likely to occur if the intensity of the test signal is high and it exceeds the interaural-attentuation for that stimulus frequency. However, the interaural-attentuation level may vary from frequency to frequency and may range from 40dB to 80dB i.e. the difference between effective intensity of the signal emanating from the earphone and the signal at the non-test cochlear maybe anywhere between 40dB to 80dB.

However, because of the variability in interaural-attentuation values Hodgson (1980), suggests that we ignore the specific frequency values and conservatively generalise that the expected interaural-attentuation for any air conducted signal to be about 50dB to 60dB. For bone conduction, however, the interaural-attentuation value is essentially 0dB as the bone oscillator which vibrates against the skull is more likely to stimulate the better cochlear regardless of the mastoid on which the vibrator is placed i.e. little or no damping effect is experienced with bone conducted signals. Therefore, masking should always be considered when there is a ±5dB difference in thresholds between the air conducted pure tone signals and bone conducted pure tone signals.

It is, therefore, evident from the above discussion that clinical masking should be used whenever there is any danger of the test signal being heard in the non-test ear (Sanders, 1978).

3.2.2 Central masking

Central masking according to Hodgson (1980), occurs when there is a shift in the threshold of the test ear, relative to it's threshold in quiet. This occurs even if the level of the masking noise is insufficient for actual physical crossover to take place. Thus, an observed threshold shift when the level of masking is less than the magnitude of interaural-attentuation is attributable to central masking. The mechanism of which is presumed to lie in the higher pathway of the central auditory system where "neural interaction" between ears causes a threshold shift of the test ear due to the masking noise introduced in the non-test ear, (Hodgson, 1980). The amount of central masking increases as the masking level increases. The clinical effect of central masking though negligible, may change threshold by 5dB to 15dB (Hodgson, 1980). Zwislocki (1953), estimated the maximum threshold shift from central masking to be 5dB. However, Dirks and Malmquist (1969), reported central masking of 10dB to 12dB and Linden et al. (1959), found as much as 15dBs of central masking. This possibility of a central masking effect dictates that one should use no more masking than that which is necessary i.e. the masking used must be effective without compromising the response parameters.

3.3 DESCRIPTION OF MASKING SIGNALS USED IN AUDIOLOGICAL ASSESSMENTS

The question of what kind of masking noise to use requires a consideration of the kind of noises available and of the masking effectiveness of each (Sanders, 1978). The key issue in the selection of the type of masking noise is that of "relative masking efficiency", i.e. the ratio of the shift in threshold in the non-test ear relative to the overall intensity of the noise. The objective is to achieve the largest shift in threshold with the least overall noise intensity (Goldstein and Newman, 1985). According to Hodgson (1980), audibility of a signal is best prevented by the presentation of another signal having a similar frequency composition because the type of masking noise used depends to a large extent on the test stimulus selected.

Over the years, various types of masking noises were used in audiometric assessments and the following are some of the more frequently used.

3.3.1 Complex noise

This consists of a low fundamental frequency and amplified harmonics. It has a buzzing low pitch quality. The noise concentrates energy in the low frequencies and is a good masker of low frequency signals. Therefore, in the presence of high intensity complex noise, crossover hearing can still occur for high frequency tones. This type of noise is inadequate for use in BAER testing as the test signal viz. the transient click is essentially a high frequency signal.

3.3.2 White noise

This type of masking noise is composed of energy present randomly at all frequencies with approximately equal energy spread across the frequency range (Goldstein and Newman, 1985). Hodgson (1980), states that because of it's broad and nearly flat spectrum, white noise is an efficient masker for most pure tones used in clinical audiometry. However, there are two problems :

i. In masking a pure tone, a great amount of energy offered by white noise is unused since only the energy in frequencies immediately surrounding the test tone is useful in masking that tone. The unused energy contributes to perceived loudness and often patients will object to the loudness of white noise; especially at an intensity level necessary to prevent crossover hearing.

ii. The use of white noise is related to the differential sensitivity of the ear across frequency. Therefore, white noise will produce different amounts of masking for each test frequency and will be least effective for low frequency signals, where the ear's sensitivity is poorest in normals. This implies that white noise is not an effective masker for all pure tones but offers best utility for masking speech stimuli.

Since speech, by nature, is broad in frequency composition, appropriate speech noise is extracted from white noise (i.e. in the frequency range 250Hz to 4000Hz) by using suitable high pass and low pass filters. Similarly, white noise offers itself for use in masking click stimuli used in BAER testing. Broadband noise extracted from ongoing white noise by using appropriate filter settings is used effectively to mask the high frequency click transient used in BAER testing (Chiappa et al., 1979).

It is, therefore, apparent that white noise, when appropriately filtered is valuable in masking speech stimuli (by using speech noise) and click stimuli (by using broadband noise).

3.3.3 Narrow band noise

Here again, narrow band noise can be obtained by filtering white noise. These bands, centered around each audiometric test frequency, contain energy that masks pure tones efficiently. i.e. the band used to mask each pure tone is an individual noise source. This, then leads to large threshold shifts in the non-test ear with the least amount of intensity. Therefore, the patient is able to tolerate the noise with no objection to loudness (Hodgson, 1980). However, narrow band noise is not effective for BAER testing as the individual bands do not adequately cover the high frequency composition of the click transient.

In summary, effective masking for pure tones is offered by narrow band noise. Masking of speech is best achieved by using speech noise and broadband noise is best used for masking the transient click stimuli used in BAER testing. All of these noises are easily extracted by the appropriate filtering of white noise.

3.4 SECONDARY CLINICAL CONSIDERATIONS IN MASKING

Although the application of masking in the non-test ear to eliminate crossover of the test stimulus is standard protocol in clinical audiology (Schwartz and Berry, 1985), the clinician must be aware of two secondary procedural effects of masking, viz. overmasking and undermasking.

3.4.1 Overmasking

The concept of overmasking is based on interaural attenuation (IA). If masking is introduced to the non-test ear and is increased in intensity to the level of IA and beyond, it will eventually cross over to reach and mask the test cochlea. This in effect produces an ipsilateral masking condition in the test ear whereby the noise obliterates the test signal (Goldstein and Newman, 1985). The net result of which is to make the threshold of the test ear seem poorer than it really is, and this may lead to inaccurate test results (Hodgson, 1980). In order that overmasking is avoided, clinicians should use only as much masking as the IA would allow (Goldstein and Newman 1985).

3.4.2 Undermasking

According to Goldstein and Newman (1985), undermasking is also based on IA values. It refers to insufficient masking to produce the necessary threshold shift in the non-test ear. Undermasking can occur when:

i. the level of the noise is over-estimated (as in the case of a calibration error), or

ii. an insufficient level is delivered to the non-test ear because of a calculation error or of an under-estimation of the extent of the crossover signal.

The latter is likely to occur especially when conducting supra-threshold audiometric tests eg. BAER. If undermasking goes unnoticed the validity of obtained results in the test ear becomes questionable (Goldstein and Newman, 1985). Therefore, the clinician must select adequate levels of masking for the non-test ear to prevent it from responding for the test ear.

It is therefore, apparent that both overmasking and undermasking can influence audiometric test results, which calls for careful clinical use of masking.

Furthermore, the validity of all audiologic test procedures, and their resulting differential diagnosis and management are at risk, especially if masking is necessary but overlooked or inappropriately employed. (Goldstein and Newman, 1985). It is also apparent, that even if correct and appropriate masking is used, it may have an unfavourable influence on audiologic test results. This is especially attributed to central masking factors. As mentioned earlier, central masking implies that the shifts in thresholds are mediated through the central nervous system. According to Linden et al. (1959), central masking is attributable to attenuation caused by neural activity. This process may be understood by the following explanation; "The efferent fibres interconnect the superior olivary nucleus, on each side, and the contralateral cochlea; ...stimulation of the superior olivary area weakens the afferent impulses from the opposite cochlea," thereby requiring more signal intensity to override this attenuation offered by neural activity (Linden et al., 1959).

This observed central effect of masking seems to influence many of the audiologic tests, and it is important for all clinicians to be aware of the nature of these possible influences.

3.5 EFFECTS OF MASKING ON DIFFERENT AUDIOLOGIC TEST PROCEDURES

3.5.1 Effects on pure tone testing

Several studies have noted shifts in pure tone thresholds in the presence of low levels of masking. Dirks and Malmquist (1964), as cited by Goldstein and Newman (1985), noted a 1dB to 3dB shift while Linden et al. (1959), found air conduction and bone conduction threshold shifts of between 5dB and 15dB. Studebaker (1962), found the greatest shift for bone conduction thresholds to be between 7dB and 12dB at 2000Hz. All of the above findings were attributed to central masking effects.

3.5.2 Effects on speech audiometry

Martin (1966), found that contralateral masking presented below the level of crossover shifted speech thresholds in normal hearing subjects by approximately 4dB to 8dB. In 1966, Martin reported that high level masking produced a 5dB shift in the speech thresholds for spondees among normal hearers. Coles and Priede (1970), reported results on masking for speech discrimination whereby they found masking for speech discrimination whereby they found central masking effects of up to 1% intelligibility decrement for every 3dB sensation level of wide band contralateral masking noise. It, therefore, appears that central masking may affect both speech thresholds and speech discrimination among normals. However, these changes are regarded as being minimal as they do not seriously compromise overall test results.

3.5.3 Effects on the Short Increment Sensitivity Index (SISI) Test

Central masking has been found to cause slight increases in SISI scores at 1000Hz and significant changes at 4000Hz (Blegvad and Terkildsen, 1967). The intensity of the tone employed, as compared with the intensity of the masking (signal-to-noise ratio), has been proven to affect results (Swisher et al., 1969). Both normal-hearing subjects and those with conductive pathologies were seen to shift from negative SISI scores without masking to questionable and positive scores with masking (Shimizu, 1969).

3.5.4 Effects on Tone Decay Testing

Shimizu (1969), and Coles and Priede (1970), found that subjects tended to demonstrate significant apparent tone decay at 1000Hz and above when masking was present as compared when masking was absent. This suggests that subjects appeared to have cochlear nerve problems, whereas in fact they all had normal auditory function.

3.5.5 Effects on Bekesy Audiometry

Dirks and Norris (1966), claimed that masking also affected Bekesy audiometry, especially when the tone and masking were either both pulsed or both continuous. Furthermore, as frequency increased, greater threshold shifts, separation of tracings and narrowed excursions appeared to occur due to central masking (Blegvad, 1967). In addition, Blegvad (1967), found that type tracings appeared to shift from type I to II, from type I to IV and from type II to IV in the presence of masking.

It is apparent that masking, though an essential test procedure in audiologic evaluations, may appear to influence and alter "normal" test results. It is, therefore, reasonable to assume that if masking affects other audiometric tests as discussed, it would also have some influence on the normal BAER.

As such, the focus of the ensuing discussion will be directed towards reviewing the literature that led to the need for examining the effects of masking and of those studies that specifically relate to assessing the effects of the clinical use of contralateral masking on the normal BAER.

3.6 REVIEW OF LITERATURE ON MASKING IN BAER TESTING

The effects of presenting a masking noise to the non-test ear on the response obtained from the test ear are critical to the successful interpretation of any audiometric test. Therefore, it is imperative that one is aware of the limitations of the unmasked and masked test results in order to localize a lesion effectively or to determine the degree of impairment accurately. This point of view is in accordance with that expressed by Humes and Ochs, (1982).

Prior to the actual introduction of routine clinical masking in BAER testing, an important question had to be answered i.e. whether it is necessary to use masking in

BAER testing? In an attempt to answer this question, two of the earliest studies viz, that of Finitzo-Hieber et al. (1979), and Chiappa et al. (1979), produced conflicting reports. Finitzo-Heiber et al. (1979), studied the BAER's in two adults with unilateral sensory neural deafness. They reported that in both subjects, normal BAERs were obtained in the normal ear at a click presentation level of 60dBsl. However, both subjects failed to show repeatable BAERs when the impaired ear was stimulated at very high intensity levels of 100dB to 117dB pe SPL (peak equivalent sound pressure level). The authors, therefore, concluded that contralateral masking may not be necessary during BAER testing. The implication of this was that there was NO danger of acoustic cross hearing (cross-talk) in BAER testing. Their contention was questioned later the same year by Chiappa et al., (1979).

These authors reported on different findings in two subjects who had complete unilateral non-functioning ears. They recorded recognizable waveforms which had increased latencies upon stimulation of the poorer ear and normal responses upon stimulation of the better ear. They then introduced masking in the better ear and found that the previously obtained response in the poor ear was completely abolished. Based on these findings, Chiappa et al. (1979), recommended the use of contralateral masking in the clinical application of BAER, especially in monaural BAER testing. The implication of their study is that there is the possibility of acoustic cross-talk contributing to a monaurally elicited BAER. Therefore, masking should be used when eliciting the monaural BAER.

Subsequent to Chiappa et al.'s (1979) study, four studies, those of Ainslie and Boston, (1980); Ozdamar and Stein, (1981); Humes and Ochs, (1982) and Reid et al.,(1984) further confirmed the findings of Chiappa et al. (1979), and suggested that acoustic cross hearing may occur when eliciting the monaural BAER.

Ainslie and Boston (1980), used a different method to verify the effects of acoustic cross hearing. They compared the monaural BAER (both ears stimulated separately) both with and without masking against binaurally elicited responses in four normal hearing subjects. They, subsequently reported that observed differences between unmasked monaural, masked monaural and binaural responses were due to the effect of "acoustic cross-talk". They suggested that contralateral masking be considered when eliciting the monaural BAER.

Ozdamar and Stein (1981), have shown that wide band clicks, which are commonly used in the recording of BAERs may cross over to the non-test ear. They found that when the difference in click thresholds between the two ears exceeds approximately 60dB, BAERs may be recorded from the non-test ear. Their results demonstrated that, in patients with a unilateral hearing loss a crossover response may be present using a click at 70dBHL and is definitely present using a stimulus at 90dBHL. In view of this observed crossover response they also recommended the use of contralateral masking, especially in patients who are suspected of having a unilateral hearing loss.

Humes and Ochs (1982), have claimed the existence of a similar crossover response effect. They studied four subjects with unilateral hearing loss. All of the subjects produced BAERs in the poorer ear, but stated that these responses varied in latency, amplitude and morphology to those obtained in the good ear. Thus, their findings appear to concur with those of Chiappa et al., (1979); Ainslie and Boston (1980), and Ozdamar and Stein, (1981).

Reid et al. (1984), reported on their success in eliciting BAERs from five unilaterally deaf subjects whilst presenting clicks at 90dBsl to the impaired ear. All subjects demonstrated clear wave V peaks, but at delayed latencies on the order of 7.5 msec. However, when masking at 50dBsl was introduced into the good ear, the previously observed BAERs were abolished in all 5 subjects. They then concluded that the presence of a BAER in the impaired ear without masking was due to a crossover effect. Therefore, in order to obtain an accurate assessment of the status of the impaired ear, they recommend that the good ear be occupied by using an appropriate masking noise.

The strong recommendation that emerges from the four studies quoted, appears to firmly support the need for the use of clinical masking, especially when eliciting the monaural BAER in patients who have a unilateral sensory neural hearing loss.

In accepting the foregoing conclusions, researchers found it reasonable to speculate on the effects of contralateral masking on the monaurally elicited BAERs in normal hearers. The premise for such speculation lies in the following argument, as put forward by Reid et al., (1984). "If responses from the contralateral ear can produce an ABR waveform in patients with a unilateral hearing loss, it seems likely that such contralateral responses will contribute to the ipsilaterally recorded ABR waveform in normally hearing people." In order to determine the effect of such a contribution, researchers began to compare the ABR waveforms obtained both with and without contralateral masking at various stimulus levels. This then led to studies which were specifically designed to examine the effects of contralateral masking on the monaurally elicited BAER in normal hearers.

3.7 REVIEW OF SPECIFIC STUDIES CONDUCTED TO EXAMINE THE EFFECTS OF CONTRALATERAL MASKING ON THE NORMAL BAER

3.7.1 CHIAPPA, GLADSTONE AND YOUNG, (1979).

The first of these studies which recognized the need for examining the effects of the clinical use of contralateral masking on the normal BAER was conducted by Chiapppa et al., (1979). They studied the BAERs in 12 normal, hearing adults whose age range was from 15 to 51 years. Click evoked BAERs, were obtained in both unmasked and masked conditions for a stimulus presentation level of 60dBsl (re: threshold for a click stimulus which was stated to be within 5dBHL for subjects tested). The masking noise comprised of white noise presented at a "sensation level" of 60dB. The reference used to establish sensation level was not specified, i.e. it was not clear if 60dBsl referred to a threshold for white noise, or if it was referenced to the threshold of the click stimulus. Nonetheless, they were unable to observe any significant effects of contralateral masking noise on the latency or amplitude measurements made. It is, however, unclear as to whether absolute or relative or both latency measures were made. Furthermore, the same vague statement is made about amplitude measurements, i.e. no mention is made about whether absolute, relative or both types of amplitude measurements were made. Further, no mention is made about which of the peaks were studied, i.e. whether all or specific peaks were studied.

However, they reported that 12 of their subject's ears (55%) produced waveform morphological changes in the peak IV-V complex. In referring to an earlier figure (fig. 6 p. 37) they reported that 4 ears changed in pattern from B to C; 3 ears changed in pattern from A to E; 2 ears changed in pattern from A to D and one ear each changed from C to B; B to D and C to D. These changes in morphology of the peak IV-V complex were attributed to the presence of masking in the non-test ear. Unfortunately, these authors did not offer an explanation for the observed morphological changes to the peak IV-V complex, nor did they speculate on the possible influence of cross-talk or central masking. It is also apparent that their study did not account for any sex difference in their overall results both with and without the introduction of contralateral masking.

3.7.2 HUMES AND OCHS, (1982)

These researchers studied the effects of contralateral masking on the monaurally elicited BAER in 10 normal hearing adults (5 men and 5 women) whose age ranged from 23 to 33 years with a mean age of 26 years. All subjects were described to have had normal hearing as evidenced in pure tone air and bone conduction thresholds of better than 15 dBHL (ANSI, 1969) for octave frequencies of 250Hz to 8000Hz and with normal tympanograms bilaterally.

Click stimuli were presented at 40, 60 and 80dBsl and with each click stimulus level, broadband masking noise was presented to the contralateral ear at 20 and 40dBsl. These were referenced to normal thresholds obtained on pure tone testing. They restricted their measurements to peak V latency and amplitude. They reported that there were no statistically significant differences in their measurements between the unmasked and masked conditions across all masking levels used ($p > 0.01$). However, it is unfortunate that no consideration was given to measuring the effects of masking on:

i. the remaining peaks

ii. the interpeak latencies
iii. absolute amplitudes
iv. relative amplitude i.e. the peak V:I ratio and
v. waveform morphology

Furthermore, despite having an equal number of males and females in their small sample, no consideration or report was made or the relative effects of sex differences on the BAER both in the absence and presence of contralateral masking.

3.7.3 ROSENHAMER AND HOLMKVIST, (1983)

These authors examined the effects of contralateral white noise masking on monaural BAER in 11 normal hearing, young female subjects. Their age range extended from 24 to 37 years and their mean age was 32 years. Their pure tone thresholds were less than 20dBHL at the six standard frequencies from 125Hz through 4000Hz and less than 25dBHL at 6000Hz and 8000Hz.

The clicks were presented at a constant level of 70dBHL. White noise masking was introduced at four different levels viz. 60, 70, 80 and 90dBHL. The click stimuli were introduced in the right ear while the white noise was presented in the left ear. The researchers restricted their measurements to absolute latencies of peaks I, II and V and the amplitude ratios of peak V compared to peaks I and III respectively. These amplitude ratios were determined by visual inspection only. Their results indicated that there was no significant change to peak I latency in the presence of the four levels of masking. The latency of peak III was significantly prolonged only at the noise level of 90dBHL. The latency of peak V was significantly prolonged at the noise levels of 80 and 90dBHL. The average latency prolongation was in the order of 0,05 msec.

They attributed their findings to central masking effects rather than due to cross-talk. Furthermore, they reported no significant changes in terms of amplitude ratios. However, these researchers did not consider the effects of masking on inter-peak latencies, nor did they give any consideration to the combined effects of masking and differences in sex on their obtained results.

3.7.4 REID AND THORNTON, (1983)

In their study, BAERs were obtained in 8 normally hearing subjects, 3 females and 5 males, who were aged between 22 and 29 years. No mean age is given.

In this experiment the click stimulus level was kept constant at 70dBsl. No clear reference is made as to whether sl was related to pure tone threshold or to click threshold. Contralateral masking was introduced to the non-test ear at levels of 15, 30, 45, 60, 70 and 80dBsl. Here again no references are given as to which threshold was used to establish sensation level. Furthermore, the type of contralateral masking noise is not defined i.e. whether white noise, broad band, or narrow band noise etc., was used.

In reporting their finding, they state that the analysis of their results show no statistically significant effect of contralateral masking noise upon the normal BAER. Their analysis was restricted to absolute amplitude and latency measures of peak I and V.

In explaining the absence of significant effects of contralateral masking on the monaural BAER, Reid and Thornton (1983), draw from the hypothesis put forward by Gersuni (1971), and his associates. These authors proposed that a different mechanism and pathway within the auditory system exists for short duration sounds and for longer duration sounds. They speculated that, if wide band clicks produces neural activity within the "onset responding " short duration part of the auditory pathway and the continuous broad band masking noise causes neurons in the long duration part of the pathway to fire, then perhaps, for this reason, contralateral masking will not have an effect on the BAER.

However, a detailed examination of brainstem transmission time as evidenced by interpeak latencies of peak I-III, III-V and I-V may have clarified and possibly elaborated on the above speculation put forward by Gersuni (1971), i.e. Reid and Thornton ought to have studied interpeak latency effects of te BAER by comparing them both in the absence and presence of contralateral masking noise.

In addition, they also neglected to study the effects of differences in sex on the BAER both in the presence and absence of contralateral masking.

3.7.5 REID, BIRCHALL AND MOFFAT, (1984)

In their study, auditory brainstem responses were recorded from 9 normally hearing subjects, 6 females and 3 males, aged between 19 and 29 years. No mean age is given. In their procedure, they presented clicks at 70, 80, and 90dBsl to one ear, both with and without 50dBsl of contralateral masking i.e. the masking level was kept constant while the stimulus level was varied. It is noted that no clear reference is made as to how sensation level was obtained both for the click level of presentation as well as for the masking level presentation. In addition, the type of masking noise used has not been described i.e. whether white noise, broad band or narrow band noise was used.

The measurements studied by these researchers included peak-to-peak amplitude and latency values of waves I-VI. These values were then analysed statistically and it was found that for waves I-V there were no significant latency or amplitude differences between the results obtained with and without masking at each stimulus level. However, they noted that there was a 19% increase in the amplitude of peak V with masking (at 50 dBsl) with a click level of 90dBsl. Furthermore, peak VI amplitude was significantly reduced with masking when the click stimulus level was set at 90dBsl, but there was no significant effect at lower click stimulus levels. The peak VI amplitude under the high stimulus level condition was reduced by 26%.

Their explanations for such findings were related to the theory that the presence of peak V from the contralateral ear is in someway affecting peak V and VI from the test ear. They elaborate by stating that it is as though the contralateral response, i.e. a delayed peak V, is combining with the ipsilateral response and thus affecting the amplitude of peak VI and of peak V. However, this explanation does not take into account any changes in the frequency spectrum of the click as it travels from one ear to the other. Generally, they attribute their significant findings to the effects of acoustic crossover rather than due to central masking factors.

It is important to note that Reid et al. (1984), have also neglected studying certain other aspects of the BAER in the presence of contralateral masking. These include:

i. Interpeak latencies

ii. Relative amplitudes i.e. the peak V:I ratio especially in view of the fact that they found a 19% reduction of the peak V amplitude in one of their treatments.

iii. No consideration was given to male vs female responses.

3.7.6 PRASHER AND COHEN, (1984)

They obtained BAERs from 12 subjects with normal hearing sensitivity. No reference is made to age and to the number of male versus female subjects that were used. Furthermore, in their procedure several treatments of masking and non-masking were used. However, of interest is their use of continuous white noise in one of their treatments as opposed to pulsed white noise in another. In other words in one treatment continuous white noise was presented to the non-test ear while clicks were presented to the test ear. In the other treatment, pulsed white noise was used instead of continuous white noise.

It is unfortunate that these researchers did not specify the intensity levels of the clicks and of the 2 types of masking noises used.

In presenting their results they state that the BAERs to click stimulation remained unaffected by the presence of continuous white noise in the contralateral ear. However, they reported that when white noise was presented in the pulsed mode to the non-test ear and clicks to the test ear, then peak V of the BAER diminished in amplitude in comparison to only monaural click stimulation. All other components of the waveform remained unaffected by the presence of pulsed white noise in the contralateral ear.

The change in the amplitude of peak V in the presence of pulsed white noise is explained in terms of it being a manifestation of central masking i.e. due to binaural neural interaction rather than due to crossover hearing via bone conduction.

They cite Zwislocki (1979), in suggesting that pulsed noise is a more effective central masker than continuous noise, thereby producing a centrally mediated change in the peak V amplitude. They further suggest that the mediation of the central masking effect is in the region of the inferior colliculus. (i.e. the presumed site for the generation of peak V). However, this suggestion is at variance with the recent finding by Musiek et al. (1986), who suggested that peak V may derive from the lateral leminiscus. Thus, this apparent conflict in opinions provides room for further research on the actual site and mediation of central masking. Generally, the manner in which Prasher and Cohen (1984), present their information makes it difficult for one to replicate their study and to verify their findings. This is especially apparent in their failure to disclose click intensity and masking intensity levels used; to discuss how amplitude and latency measurement were conducted; the number of male and female subjects in their sample; their age range; equipment used, and several other procedural aspects.

Table 3 on the next page reflects a summary of the studies that have examined the effect of contralateral masking on the normal BAER.

Table 3: Summary of studies on the effects of contralateral masking on the normal BAER

AUTHORS	Chiappa, Gladstone & Young (1979)
NO. OF SUBJECTS	12, Number of Male vs Female is not mentioned.
CLICK INTENSITY LEVEL	60dBsl
TYPE OF MASKING NOISE USED CONTRA-LATERALLY	White noise
MASKING INTENSITY LEVEL/S	60dBsl
BAER PARAMETERS EXAMINED	*1. Latency (not clear if absolute or relative) 2. Amplitude (not clear if absolute or relative) *3. Morphology IV-V Complex
RESULTS AND FINDINGS	*1. No significant effects were reported. *3. Significant change in observed patterns in 55% of ears tested.

AUTHORS	Humes & Ochs (1982)
NO. OF SUBJECTS	10, 5 Males and 5 Females
CLICK LEVEL	40; 60 & 80dBsl
MASKING NOISE	Broadband Noise used contralaterally
MASKING LEVEL/S	20 & 40dBsl
BAER PARAMETERS EXAMINED	*1. Absolute latency of peak V. *2. Absolute amplitude.
RESULTS & FINDINGS	*1&2. No significant effects were reported ($p > .01$)

Table 3 cont./...

AUTHORS	Rosenhamer & Holmkvist (1983)
NO. OF SUBJECTS	11, 11 Females
CLICK LEVEL	70dBHL
MASKING NOISE	White Noise used contralaterally
MASKING LEVEL/S	60, 70, 80 & 90dBHL
BAER PARAMETERS EXAMINED	*1. Absolute latencies I, III & V. 2. Absolute amplitude of peak IV-V complex. 3. Relative amplitudes I:III; III:V & I:V.
RESULTS AND FINDINGS	*1. No significant changes to latency of peak I. The latency of peak II was prolonged only at 90dB of contralateral masking Latency of peak V was prolonged at 80 & 90dB of masking. No changes were observed on shape and amplitudes of response.

AUTHORS	Reid & Thornton (1983)
NO. OF SUBJECTS	8, 3 Females and 5 Males
CLICK LEVEL	70dBsl
MASKING NOISE	Not described
MASKING LEVEL/S	15; 30; 45; 60; 70 & 80dBsl
BAER PARAMETERS EXAMINED	1.Absolute Latencies I & V 2.Absolute Amplitudes I & V
RESULTS AND FINDINGS	No significant effects were reported for all latency and amplitude measures made.

Table 3 cont./...

AUTHORS	Reid, Birchall & Moffat (1984)
NO. OF SUBJECTS	9, 6 Males and 3 Females
CLICK LEVEL	70; 80 & 90dBsl
MASKING NOISE	Not described
MASKING LEVEL/S	50dBsl
BAER PARAMETERS EXAMINED	1.Absolute Latencies I-VI 2.Absolute Latencies I-VI
RESULTS AND FINDINGS	No significant latency effects were reported for peaks I to VI. However, with 90dBsl of clicks and 50dBsl of masking peak V amplitude increased by 19% and peak VI amplitude decreased by 26%. No significant changes in amplitude were seen at 70 & 80dBsl of masking.

AUTHORS	Prasher & Cohen (1984)
NO. OF SUBJECTS	12, Number of Males vs Female subjects not mentioned
CLICK LEVEL	Not presented
MASKING NOISE	1.Continuous white noise used contralaterally 2.Pulsed white noise used contralaterally
MASKING LEVEL/S	Not presented
BAER PARAMETERS EXAMINED	Not clearly specified - simply refer to brainstem responses.
RESULTS AND FINDINGS	No significant effects were reported in BSR with continuous white noise. However, peak V diminished in amplitude in the presence of pulsed white noise. NO other effects were noted.

It is, therefore, apparent from the various studies above, that there is no clear or conclusive evidence for, or evaluation of, the potential influence of contralateral masking on the monaurally evoked BAERs. This contention is drawn from the fact that previous investigators have not clarified the effects of cross-talk and/or central masking on the BAER for monaural stimuli. Variations in reported findings may be due to several contributing factors, some of these may include the following:

i. differences in the number of subjects studied.
ii. differences in the age ranges of the subjects studied.
iii. no consideration is given to the differences in male vs female components of the BAER.
iv. differences in stimulus intensity levels used.
v. differences in masking intensity levels used.
vi. differences in type of masking noise used.
vii. differences in stimulation parameters used.
viii. differences in recording procedures used.
ix. differences in measurement procedures used.

The foregoing provides motivation for a well controlled study to aid in the documentation and clarification of the effects of contralateral masking on the monaural BAER. Furthermore, despite the documented evidence that there exists a difference in the normal BAER obtained between males and females, none of the studies reviewed, paid any attention to the potential influence of this variable on normal BAER elicited both in the presence and absence of contralateral masking.

It is appropriate, therefore, to review the salient studies that reflect on sex difference and to examine and document these differences.

3.8 A BRIEF OVERVIEW OF SEX-DIFFERENCES IN CNS FUNCTION AND ANATOMY

The issue of sex differences in central nervous system (CNS) function and anatomy has been the subject of interest and attention by researchers in different disciplines. Data from selected studies suggest that males and females differ on certain measures of CNS function and anatomy.

Trotman et al. (1979), as cited by Wexler et al., (1988) have claimed the females show greater bilateral flexibility during self-generation tasks than males who were found to be more rigid in their performance. Sex differences have also been described in asymmetry of facial motor behaviour (Alford et al. 1981), and in dichotic listening tasks (Dawe et al. 1986), as cited by Wexler et al., (1989). Mc Glone (1980), reviewed the extant clinical, normative, anatomical, electrophysiological, developmental and animal data and concluded that the male brain may be more asymmetrically lateralized than that of the female, both in verbal and non-verbal functions. Stockard et al. (1981), found a sex difference effect on visually evoked potentials. They found that females showed shorter P1 latencies (p = 0,03; t-test) than males. The basis for which was speculatively attributed to shorter visual pathway length - the nasion to the inion distance in females than males. On average females had a 3.9 cm shorter distance ($p < 0,01$, t-test) than males. In addition they felt that the higher deep body (and brain) temperatures found in females may have contributed to the observed sex difference effect. Unfortunately, they did not elaborate on the latter factors.

Witelson (1989), studied 56 human brains (40 female and 16 male - after autopsies were conducted) and reported on marked sex differences in both the size and weight parameters. Female brains were significantly smaller in weight ($x = 1263g$, $t = 6.0$, df54, $P < 0,0001$) than in males ($x = 1439g$). This finding was consistent with those of Dekaban (1978), who found female brain weight to be 1280g and that of males to be 1410g.

Such evidence has led Witelson to claim that males and females differ significantly in brain size and weight. Holloway (1980), claimed that larger brain size in males might be expected since males are taller and have larger bodies. He stated that brain weight correlates significantly with height and body weight ($r = 0.47$ and 0.29 respectively).

Bearing in mind the above demonstrated sex differences in other CNS functions and anatomical regions, it is conceivable that similar differences may exist in auditory function and anatomy.

The ensuing discussion will reflect on studies that have considered sex difference in:

i. conventional auditory performance tests and

ii. the BAER.

3.9 THE INFLUENCE OF SEX RELATED DIFFERENCES IN CONVENTIONAL AUDIOMETRIC TESTING

According to Jerger and Hall (1980), there is mounting evidence that differences in sex (male vs female) is a factor in both behavioural and impedance audiometry. Generally, it has been found that females tend to perform better on various auditory tests. This tendency indicates that females have better auditory sensitivity than males. However, there is no clear-cut explanation for this observed sex difference effect.

3.9.1 Pure tone audiometry

Research conducted by Hayes and Jerger, (1979); Bunch and Raiford (1931), and Corso (1963), have indicated the following:

i. pure tone sensitivity for high frequency pure tone signals is usually better in women than in men.

ii. Pure tone sensitivity for low frequency pure tone signals is usually better in men than in women.

3.9.2 Speech audiometry

Sex difference in performance on diagnostic speech audiometric procedures have also been documented. Hayes and Jerger (1979), have reported that females tend to perform better on speech testing tasks than do males.

3.9.3 Impedance audiometry

Several researchers have reported on the influence of sex related differences on impedance audiometric measurements. Jerger et al., (1972); Hall (1979), and Hall (1984), have all shown that static compliance measures are greater in male than in female subjects. There is, however, no documented evidence to reflect on apparent differences in tympanometry and acoustic reflex measures (Jerger and Hall (1980).

It is unfortunate, however, that there are no clear-cut reasons to explain these sex difference effects on conventional tests. Researchers have speculated on general physiological differences, hormonal differences, and differences in dimensions of the head and ears among males and females. Morever, there is no conclusive evidence to explain the differences. Be that as it may, it is important that clinicians take cognisance of these sex related differences especially when establishing and using normative data.

As a result of the observed sex-related effects on conventional auditory tests, researchers were then prompted to examine these effects on the normal BAER. The ensuing presentation reviews pertinent studies that have reflected on the effect of this subject related variable on the normal BAER.

3.10 THE INFLUENCE OF SEX RELATED DIFFERENCES ON THE NORMAL BAER

According to Jerger and Hall (1980), the potential influence of differences in sex on the BAER have been "grossly unappreciated". It was established that only 6% of the 37 studies examined, reported on BAER data as a function of sex-difference related influences. In the light of the above, Jerger and Hall strongly recommend that careful consideration must be given to this subject related variable in future research and clinical applications of the BAER.

3.11 STUDIES REFLECTING ON THE INFLUENCE OF SEX DIFFERENCE ON THE BAER

3.11.1 BEAGLEY AND SHELDRAKE, (1978)

As part of their study on differences in Brainstem response latency with age and sex, these authors tested 70 subjects ranging in ages from 14 to 79 years. Attention was focussed primarily on the measurement of the latency of peak V as a function of three intensity levels, viz. 60, 70 and 80dB. The Wilcoxon matched pair signed ranked correlation test was applied to their data. The results obtained reflected that in each case the difference was highly significant ($p < 0,00003$) in favour of females having shorter latencies for peak V than males.

However, no explanation or speculation on the reason for their obtained difference was afforded. It was recommended that this sex-difference should be considered in diagnostic applications.

3.11.2 STOCKARD, STOCKARD AND SHARBROUGH, (1978)

In reporting on nonpathologic factors influencing Brainstem auditory evoked potentials, these researchers presented sex-difference related data obtained in 50 normal young adults, 25 males and 25 females. However, they did not present the age ranges or the mean age for each group. The clicks were presented at 60dBsl at a rate of 10per sec and the level of the clicks were referenced to hearing thresholds for pure tones.

Their attention was focussed on interpeak latency (IPL) differences between males and females i.e. peaks I-III; III-V and I-V. Table 4 below reflects a summary their findings.

Table 4 : Sex differences in interpeak latencies : means and (sd). Adapted from Stockard, Stockard and Sharbrough, (1979)

IPLs	Males	Females	Diff.
I-III	2.12 (0.13)	2.11 (0.19)	0.01 (NS)
III-V	1.95 (0.14)	1.82 (0.12)	0.13 ($p < 0.002$)
I-V	4.06 (0.20)	3.94 (0.25	0.12 ($p < 0.05$)

These researchers concluded that females have significantly short IPLs than males. Furthermore, they speculated that this difference might be related to shorter anatomical distances in the corresponding segments of the auditory pathway, as a result of the smaller average head size and brain of females. It was felt that this may result in a shorter length in the auditory pathway from the acoustic nerve to the mid-brain in females, thereby accounting for the sex differences in IPL. They do, however, acknowledge that this explanation is speculative, and that other unknown factors may also be relevant. Nevertheless, as a consequence of this observed difference in IPLs between males and females, Stockard et al. (1978), suggest that it is "worthwhile" to have separate IPL norms for males and females. Thus, the female IPL norms cannot be applied to male patients and male IPL norms cannot be applied to female patients.

3.11.3 McCLELLAND AND McCREA, (1979)

As part of their study on Intersubject Variability of the Auditory Evoked Brain Stem Potentials, these researchers focused attention on the contribution of sex differences on the normal BAER. Responses were obtained for 39 adult subjects, consisting of 21 females (x age 22.1) and 18 males (x age 21.5). Stimulation ranged from 80dBnHL to 20dBnHL at a click rate of 10per sec. Their measurements focused on the latency of peak I, III and V and on the interpeak latency of peaks I to V.

In examining the latency for peak 1 for the two groups it was reported that there was no significant differences when the t-test was employed.

In the case of peak III a small sex difference was found, whereby males showed a slight delay in response latency when compared to their female counterparts. Peak V showed an extension of the peak III trend with clear sex differences emerging at all intensities, especially at 80dBnHL i.e. males showed consistently longer peak V response latency times than females. Table 5 below depicts this trend and at the same time shows that the IPL of peak I-V is different for the groups at 80dBnHl. In applying the t-test, McClelland and McCrae have shown that at all three intensity levels (40, 60, and 80dBnHl) male responses were significantly later than female responses at the 0.1% level.

Table 5 : Latency statistics for peak V and peak I to V interval at 80dBnHL. Adapted from McCleland and McCrae, (1979).

Intensity	Males mean = SE	Females mean = SE
Latency of peak V at:		
80dBnHL	5.95 = 0.03	5.65 = 0.03
60dBnHL	6.21 = 0.04	5.96 = 0.05
40dBnHL	6.93 = 0.08	6.54 = 0.06
Peak I to peak V interval at 80dBnHL	4.19 = 0.04	3.92 = 0.03

According to the authors, these sex differences are largely attributable to difference in the peak V to peak I interval, and are due to gender difference in conduction time within the central auditory pathway. However, they do not specify the cause of the differences in conduction time, nor do they speculate on this issue. Obviously further research is necessary in this respect.

3.10.4 KJÆR, (1979).

In his study, on difference of latencies and amplitudes of brain stem evoked potentials in subgroups of normal material, he focuses his attention on both latency and amplitude measures in males and females. The responses were obtained from 40 normal subjects aged between 13 and 48 years. Click stimuli were presented at 75dBHL. The rate of click presentation has not been stipulated.

He reported that male subject latencies of peaks III to VII were significantly longer than those of female subjects ($p < 0,0005$). The elongation increased from 0,09 to 0,44 msec for peaks III to VII. Female subjects had amplitudes significantly larger ($p < 0,05$) than male subjects although the variations were wide. He also reported that the relative latencies of peaks I-III; III-V; and I-V were all significantly shorter in females than in males ($p < 0,0005$). He attributed the sex difference effect to body length, whereby male subjects were on average 13 cm taller than female subjects.

He recommended that due to the demonstrated sex effect, one should establish separate normal values for males and females.

3.11.5 MICHALEWASKI, THOMPSON, PATTERSON, BOWMAN AND LITZELMAN, (1980).

The above authors, like Jerger and Hall (1980), studied both latency and amplitude differences between males and females. They presented their findings based on responses obtained from 20 normal hearers (7 males and 13 females) whose ages ranged from 20 to 40 ($\bar{x}$ = 30) years. Clicks were presented at 60, 70 and 80dBsl at a rate of 10,15 and 20per sec. Their measurements focused on absolute latencies and of absolute amplitudes for all seven peaks of the BAER.

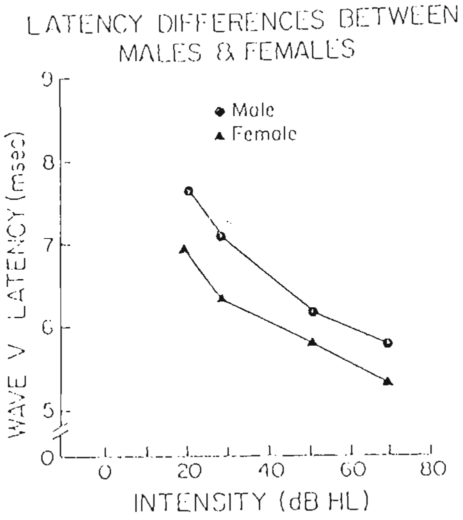
An overview of their results indicated that there were overall sex differences in the amplitudes of peaks IV, V, VI and VII, and an overall sex effect on the latency of peak V. The amplitudes of the named peaks were generally larger in females than in males and the latency of peak V was shorter in females than in males.

They attributed the results to differences in the relative distances of the anatomical generators of the BAER. They, therefore, concur with Stockard et al.'s (1978), suggestion. Furthermore, they state that because the precise origin of the sex difference effect is not clearly known, those researchers attempting to develop normative data for the BAER should consider the possible influences of sex differences.

3.11.6 JACOBSON, NOVOTNY AND ELLIOT, (1980)

In their report on Clinical Considerations in the Interpretation of Auditory Brainstem Responses, these authors draw attention to the effect of sex differences in the BAER. Without describing their procedure, they report of their findings obtained from 10 males and 5 females. They focused attention on peak V latency difference as a function of stimulus intensity among males and females.

It is evident from the following figure (fig. 8) that there is a significant difference between the latencies of males and females even as a function of intensity.



Intensity dBHL	Mean Latency (msec) to Wave V		
	Male	Female	M - F
70	5.720	5.409	0.311
50	6.179	5.775	0.404
30	7.013	6.324	0.689
20	7.678	6.926	0.752

N=10,5 males & 5 females ANOVA: F=24.8; df 1,8; P<.005

Fig.8 : BAER peak V latency - intensity function for males and females. Mean latencies and their differences are tabulated (Jacobson et al., 1980).

The authors state that the female peak V latencies were significantly (p < 0,01) shorter than male latencies at each intensity level (20, 30, 50 and 70dBHL). Their findings support those of Stockard et al. (1978), viz. that these differences are associated with the distance between common synaptic junctions of the auditory pathway i.e. females have a shorter distance between synaptic junctions, thereby accounting for the overall obtained latency values. Here again, Jacobson et al. (1980),

[illegible]

recommend the establishment of independent norms for both males and females.

3.11.7 JERGER AND HALL, (1980)

These authors, unlike previous studies quoted, studied both latency and amplitude difference between males and females. Their findings were based on responses obtained from 92 normal subjects who ranged in age from 20 to 79 years. However, no mention is made about the number of males or females studied in respect of the sex difference effect.

Attention was focused on the peak V latency and amplitude measures. They found that the average peak V latency for males was 0,14 msec longer than for females. Likewise, female subjects displayed waveform amplitudes of a magnitude of 0,080 to 0,120µV greater than those of their male counterparts. However, they did not state whether these values were statistically significant or not. Like Stockard et al. (1978), these authors speculate that due to the relatively smaller dimensions of the female CNS, neural transmission time of the BAER are reduced when compared to males. Consequently, they feel that the actual basis for the conspicuous sex difference effect deserves further investigation.

3.11.8 JERGER AND JOHNSON, (1988)

As part of their study on the interaction of age, gender and sensorineural hearing loss on ABR latency, these researchers focussed their attention on the sex difference effect on peak V latency. Their study was a retrospective one involving 325 subjects, 174 of whom were males ($\bar{x}$ age 47.5 years) and 151 females ($\bar{x}$ age 48 years). Clicks were presented at different intensity levels viz. 70, 80, 90, 100 and 103dBHL depending on the degree of hearing loss present. The rate of click presentation was at 11.1per sec.

An overview of their results revealed that peak V latency was significantly ($p < 0,5$) shorter in females than males. They postulated that at least 3 factors contributed to shorter latencies in females than males, viz. differences in head and brain size; differences in whole body temperature and differences in the hormonal milieu. However, they did not elaborate on their suggestions. Further research on the sex difference effect on the BAER is therefore, imperative.

It is apparent from the above review of the literature that there exists a significant difference in the BAER parameters between males and females. In the light of the presence of this sex difference effect, it has been strongly recommended by all researchers consulted, that due consideration should be given to this subject related variable in BAER testing and interpretation. According to Schwartz and Berry (1985), the clinical implication of these data is that each facility should generate individual latency and amplitude values between genders, since the application of female norms to male patients could lead to interpretation of delayed waveform latency where none actually exists (Type I error).

Table 6 below reflects a summary of the studies that have examined the effect of sex difference on the normal BAER.

Table 6 : Summary of studies on the effect of sex difference on the normal BAER

AUTHORS	Beagley & Sheldrake (1978)
NO. OF SUBJECTS	70, No. of male & female Subjects not mentioned.
CLICK INTENSITY LEVEL	60, 70 and 80dB.
BAER CHARACTE- RISTICS EXAMINED	Absolute latency of peak V.
RESULTS AND FINDINGS	Females had significantly shorter ($p < 0,0003$) peak V latency than males. No explanation for their finding is given.

AUTHORS	Stockard, Stockard & Sharbrough (1978)
NO. OF SUBJECTS	50, 25 Males and 25 Females
CLICK INTENSITY LEVEL	60dBsl
BAER CHARACTE- RISTICS EXAMINED	Interpeak latencies of peaks I-III; III-V & I-V.
RESULTS AND FINDINGS	I-III - not significant, III-V - significant ($p < 0,0002$), I-V - significant ($p < 0,05$). Females had shorter interpeak latencies than males. These are attributed to shorter auditory pathways in females.

Table 6 cont./...

AUTHORS	McClelland & McCrae (1979)
NO. OF SUBJECTS	39, 21 Females and 18 Males.
CLICK INTENSITY LEVEL	80dBnHL to 20dBnHL
BAER CHARACTERISTICS EXAMINED	Absolute latencies of peaks I, III & V Interpeak latency of peaks I-V.
RESULTS AND FINDINGS	No significant differences on peaks I & III. Males showed longer peak V latencies than females at 40, 60 and 80dBnHL. Interpeak latency I-V was shorter in females - reported at 0,1% level. Findings were attributed to faster conduction times in the female auditory pathway - no elaboration.

AUTHORS	Kjaer (1979)
NO. OF SUBJECTS	40, No mention is made of no. of males vs females
CLICK INTENSITY LEVEL	75dBHL
BAER CHARACTERISTICS EXAMINED	All absolute latencies I-VII Interpeak latencies of peaks I-III; III-V and I-V. Amplitude of peak V
RESULTS AND FINDINGS	Peaks III to VII were significantly longer in males ($p < 0,0005$). All interpeak latencies were longer in males ($p < 0,0005$). Amplitude of peak V was larger in females than in males ($p < 0,05$). He attributed the sex difference effect to longer body length in males by 13 cm.

Table 6 cont./...

AUTHORS	Michalewaski et al. (1980)
NO. OF SUBJECTS	20, 7 Males and Females
CLICK INTENSITY LEVEL	60, 70 & 80dBsl
BAER CHARACTERISTICS EXAMINED	Absolute latencies of peaks I-VII. Absolute amplitudes of peaks I-VII.
RESULTS AND FINDINGS	Shorter peak V latencies in females than males. Larger amplitudes in females than males. No significance levels were provided. No explanation is given but concur with Stockard et al., (1978)

AUTHORS	Jacobson, Novotny & Elliot (1980)
NO. OF SUBJECTS	15, 10 Males and 5 Females
CLICK INTENSITY LEVEL	20, 30, 50 & 70dBHL
BAER CHARACTERISTICS EXAMINED	Absolute latency of peak V.
RESULTS AND FINDINGS	Female peak V latencies were significantly shorter ($p < 0,01$) than males. They concur with Stockard et al. 's (1978) suggestion.

Table 6 cont./...

AUTHORS	Jerger & Hall (1980)
NO. OF SUBJECTS	92, No mention is made of no. of males and females
CLICK INTENSITY LEVEL	80dBHL
BAER CHARACTERISTICS EXAMINED	Peak V latency Absolute amplitude of peak V.
RESULTS AND FINDINGS	Peak V latency was 0,14 msec longer in males than females. Female amplitude measures for peak V were between 0,08 V to 0,12 V larger than for males. No levels of significance were given. Attribute findings to smaller dimensions of CNS leading to shorter neural conduction times in females than in males.

AUTHORS	Jerger & Johnson (1988) (Retrospective study).
NO. OF SUBJECTS	325, 174 Males and 151 Females
CLICK INTENSITY LEVEL	70, 80, 90, 100 & 103dBHL depending on degree of hearing loss.
BAER CHARACTERISTICS EXAMINED	Peak V latency
RESULTS AND FINDINGS	Peak V latency was significantly ($p < 0,05$) shorter in females than in males. Attributed findings to 1. differences in head and brain sizes 2. differences in whole body temperature 3. differences in hormonal milieu.

At this stage it is relevant to note that despite the known controversial effects of contralateral masking (as discussed earlier) and the known sex effect, there is a lack of research on the combined effects of these two variables on the normal BAER. Therefore, appropriate research directed at investigating the combined effects of these two variables on the normal BAER would be valuable. Such research will contribute to the body of information relating to the non-pathological factors that influence the normal BAER.

3.12 SUMMARY AND CONCLUSIONS

The initial part of this chapter dealt with a review of relevant information on the concept of clinical masking in audiology. It is evident that masking is an essential test procedure used in audiometry. It enables the clinician to obtain independent and reliable test results from the test ear without the contribution and confounding influence of the non-test ear, especially when stimuli are presented at high intensity levels.

Furthermore, while use of masking is sometimes essential, attention has also been drawn to the possible adverse effects of masking on audiometric test procedures. In this respect the effects of undermasking, overmasking and central masking were discussed and highlighted. Following the review of literature on masking, rationale were presented for the inclusion of clinical masking in BAER testing. The primary rationale emerged from BAER studies done on subjects having unilateral sensori-neural hearing losses. Briefly, the clinical use of masking was strongly advocated by several researchers who observed crossover BAERs when the impaired ear was stimulated in unilaterally deaf subjects. Supporting evidence to this effect has been presented.

Ensuing from the above, this chapter then addressed the question of whether clinical masking of the contralateral ear has an effect on the normal monaural BAER or not. It is apparent from the review of available literature that there is some controversy as to whether or not contralateral masking has an effect on the normal monaural BAER. There appears to be variations in the reports reviewed.

Generally, the studies reviewed tend to differ from each other in that they vary with respect to the following:

i. number of subjects assessed in each study
ii. the age of the subjects studied
iii. stimulation, recording and interpretative indices used
iv. intensity and type of masking noise used
v. lack of acknowledging the effect of differences in sex on the normal BAER.

Furthermore, a major problem encountered with most of the studies reviewed is the rather small sample size tested i.e. the number of subjects used in each study ranged from 7 to 12. This leads to restricted generalizability of results.

In addition to the above, the latter part of the chapter focussed on the effect of sex differences on various audiometric procedures with special emphasis on the normal BAER.

It is apparent from the studies reviewed that there is a sex difference effect on the response parameters of the BAER. There appear to be differences in both latency and amplitude measures of the normal BAER among males and females. The general conclusion among investigators is that females have shorter latencies and larger amplitudes than their male counterparts. These observed differences have been speculatively attributed to females having a smaller head circumference, combined with a foreshortening of the brainstem pathway between the auditory nerve and the mid-brain.

Finally, the need to study the combined effects of the two variables viz. that of contralateral masking noise and that of difference in sex on the normal BAER was highlighted.

In the next chapter, a description of the actual investigation will be advanced. The chapter will reflect on the aims of the study, the research questions put forward and on the methodological aspects of the research project.

CHAPTER 4

METHODOLOGY

4.1 THE INVESTIGATION

The objectives of this study are as follows:

i. The first aim of the study was to establish reference data for the monaurally elicited BAER among a group of Indian undergraduate students who participated in this project.

ii. The second aim was to investigate whether sex differences exist with regard to the monaurally elicited BAER among the group of Indian undergraduate students who took part in this study.

iii. The third aim was to determine the effect of contra-lateral masking at levels of 50, 60 and 70dBHL on the monaurally elicited BAER among the group of Indian undergraduate university students.

iv. The fourth aim was to investigate the interactional effects of sex difference and of contra-lateral masking on the monaurally elicited BAER in these subjects.

These aims led to the generation of the hypotheses which were presented in Chapter 1. In attempting to realize the aims and to test the formulated hypotheses, it was necessary to execute the investigation in two sessions, viz (i) screening and selection of subjects and (ii) conducting the brainstem auditory evoked response test. The rest of this chapter will focus on the screening and selection of subjects and on the auditory tests and procedures employed. Furthermore, a detailed presentation of the equipment used will be given.

4.2 INVESTIGATIVE PROCEDURES EMPLOYED IN THE CURRENT STUDY

Three major investigative procedures were employed to obtain the relevant data. These were:

i. Administration of a pre-test case history interview questionnaire. (APPENDIX A)
ii. Administration of an audiometric screening test battery.
iii. Elicitation of the BAER.

It should be noted that the investigative procedures (i) and (ii) above were used primarily to screen and select subjects for BAER testing.

4.3 APPARATUS EMPLOYED IN THE INVESTIGATIVE PROCEDURES

4.3.1 The pre-test case history questionnaire

(see appendix A)

This questionnaire, based on selected content areas as suggested by Emerick and Hatten (1979), and Rosenberg (1978), was drawn up by the reseacher. It was utilized to obtain details regarding identification, age, sex, academic level and year of study at university, a self report on hearing status, history of hearing problems and relevant neuro-medical information.

4.3.2 Apparatus for administration of the audiometric screening test battery

4.3.2.1 Sound proof booth

An isolated Industrial Acoustics Company (IAC) twin-audiometric sound proof booth of double wall construction meeting the noise level requirements as set by ANSI (1977), was used as the test environment for pure tone and speech audiometric testing.

4.3.2.2 Clinical audiometer

A twin-channel micro-processor based clinical diagnostic audiometer, the Grayson Stadtler GSI 10 was used for pure-tone (air and bone conduction) and speech audiometry. The accessory items of this equipment that were utilized included:

i. Bone vibrator A radio-ear B-71 bone conduction vibrator and

ii. Earphone A pair of TDH-50P telephonics adjustable earphones with MX41 cushions.

The audiometer was technically calibrated to meet standards set by ANSI (1969), in February 1988.

4.3.2.3 Otoscope

A Welch-Allen battery operated otoscope was used for the otoscopic examinations.

4.3.2.4 The impedance meter

A clinical middle-ear analyser; the Grayson-Stadtler GS1 1723, equipped with a probe-tip and a TDH-49P earphone was used to administer the impedance test battery. The meter was calibrated according to ISO-389 (1975) specifications and was technically calibrated in January 1988.

4.3.2.5 Spondiac word list (C.I.D. W-1 & C.I.D. W-2

The C.I.D. W-1 and W-2 spondiac word lists published by the Central Institute for the Deaf (USA) (cited in Martin, 1981) was used to establish speech reception thresholds (SRT) (see appendix B)

4.3.2.6 Phonetically balanced word lists

The C.I.D. W-22 phonetically balanced monosyllabic word lists were used for speech discrimination testing (SDT). These lists were published by the Central Institute for the Deaf, (USA) as cited in Hodgson, (1980). (see appendix C)

4.3.3 Apparatus used for the elicitation of the BAERs

4.3.3.1 Auditory evoked response audiometer

A Cadwell Quantom 84, computer based soft-ware controlled auditory evoked response audiometer was utilized in the elicitation of the BAERs. This system included the following essential accessory pieces of equipment:

i. Pre-amplifier A low-noise differential biological Cadwell Quantom 84 twin channel pre-amplifier with a gain of 10

ii. Headphone A pair of TDH-39P electrodynamic headphones having a flat frequency response housed in Amplivox free-field audio-cups was used to deliver click stimuli to the target ear and masking noise to the non-test ear.

iii. Programmed disk A RUN QT 84 single sided double density 3.5 inch floppy disk was used. This disk contained the BAER programme used to start and to run the system.

iv. Printer A built in ALPS printer was used to print a permanent record of the subjects' BAERs.

4.3.3.2 Electrodes

High quality self-adhesive silver-chloride electrodes as suggested by Chiappa et al. (1979), were used to pick up and to relay electrical impulses to the pre-amplifier.

4.3.3.3 Electrode gel

Standard EEG electrode gel (Colloidon) was used (Chiappa et al, 1979).

4.3.3.4 Omni-prep

Omni-prep skin preparing paste was used to clean the electrode sites of all debris.

4.3.3.5 Couch

A standard patient-examination couch on which subjects rested was used during evoked response testing.

4.3.3.6 Anechoic chamber

An electro-magnetically screened anechoic sound treated chamber meeting noise-level requirements as set by ANSI (1977), and as suggested by Reid et al. (1983), was used as the test environment for the elicitation of the BAERs.

4.3.4 Materials for recording data

4.3.4.1 Pure tone and speech audiometry

A pure tone and speech audiogram designed by the Department of Speech and Hearing Therapy, University of Durban Westville was used to manually record pure tone and speech audiometric test results. (see appendix D).

4.3.4.2 Impedance audiometry

Custom designed (Grayson-Stadtler 1723) recording cards were used to record all impedance audiometric test results (see appendix E).

4.3.4.3 Brainstem auditory evoked responses

Custom designed rolls of ALPS printer paper for the Cadwell Quantom 84 system was used to record BAER data.

4.4 Investigative procedures

The investigative procedures in this study took place over two sessions.

Session I

This session had two phases viz. phase I and phase II.

Phase I

In this phase potential subjects were required to complete the pre-test case history questionnaire.

Phase II

In this phase potential subjects underwent an audiometric screening assessment involving a test battery approach. The information and test results obtained during session I was used to select or exclude subjects in terms of the following criteria:

i. Hearing Acuity

All subjects had normal hearing acuity bilaterally as determined by the results of the conventional audiometric screening test battery and as reported by the subject.

ii. Neurological Integrity

All subjects had a negative history of central neurological abnormalities (Reid et al, 1983; Chiappa et al, 1979 and Rowe, 1978). This was necessary to eliminate the effects of neuropathology on hearing as the establishment of diagnostic reference data was part of the study. This information was gleaned from the administration of the pre-test case history interview questionnaire.

iii. Age

All subjects fell within the age range of 18 to 25 years. This was necessary as it has been found that hearing acuity is at it's peak between the ages of 16 to 30 years (Jerger, 1970 and Rowe, 1978). Furthermore, this age range was selected as it was felt that the subjects in this age range would easily tolerate the prolonged nature of the testing process.

iv. Use of Drugs

Although the commonly used drugs such as sedatives and stimulants do not apparently have an effect on the BAER (Gibson and Ruben, 1978), all subjects selected did not consume any known ototoxic drugs previously in their life. Furthermore, all subjects had a negative history of acute or chronic alcohol or ethanol intoxication as these tend to prolong the peak V latency or the peak I to V interpeak latency, (Stockard et al., 1978).

V. Dominant ear

All subjects were right handed to ensure consistency for this variable. Subjects had to fill a questionnaire designed to elicit evidence that they were right handed.
(See appendix F).

4.4.1 Description of the subject selection process

4.4.1.1 Description of procedure for session I:

Phase I - Completion of Pre-Test Case History Questionnaire.

A total of 350 questionnaires were distributed for completion among undergraduate Indian students. Of these 143 (40.85%) were returned. From these 143, 17 (11.8%) questionnaires were spoilt or inadequately completed. Eleven (7.7%) respondents were over the age of 25, while 33 (23.07%) did not wish to participate in the project. Eight (5.5%) respondents were excluded due to adverse medical and/or neurological case histories. The remaining 74 respondents comprising of 35 males and 39 females were then given appointments for participating in phase II of session I and in session II of the testing programme.

Phase II - Audiometric screening assessement.

From the remaining 74 respondents 33 males and 36 females kept their appointments to participate in phase II of session I. However, on administering the handedness questionnaire one female subject was excluded because she showed evidence of being left handed. Therefore, in total 68 subjects were still available for further assessments.

4.4.1.2 Procedures for pure tone audiometry

Pure tone thresholds were obtained bilaterally via:

i. air conduction in the octave frequencies from 250Hz to 8000Hz and

ii. bone conduction in the octave frequencies from 250Hz to 4000Hz

Carhart's and Jerger's (1959), ascending - descending method of threshold exploration was utilized to determine pure tone thresholds, as cited in Hodgson, (1980).

4.4.1.3 Speech audiometry

i. Speech reception threshold (SRT).
The standardized procedure as suggested by Hodgson, (1980) was used to establish (SRT).

ii. Speech discriminition testing (SDT).
Phonetically balanced CID-W22 word lists were presented live by the tester at the following supra-thresholds; 20dBs; 30dBsl & 40dBsl as suggested by Berger (1978), cited in Rose, (1978).

4.4.1.4 Impedance audiometry

The nature of the test was explained to each subject before testing commenced. Furthermore, prior to testing an otoscopic examination was conducted to rule out external and ear canal abnormalities. The following sub-tests were conducted:

i. Tympanometry
ii. Static Compliance Measures
iii. Acoustic Reflex Threshold Testing

4.4.1.4.1 Instructions.

Each subject was advised against moving of the head and mouth, speaking and/or swallowing during the test. They were requested to sit as still as possible. Each subject was also requested not to respond to the probe tone or to the high intensity acoustic stimuli presented during acoustic reflex threshold testing. This was done to prevent a surprise body response or a startle response which could confound the recording and interpretation of test results (Hodgson 1980).

4.4.1.4.2 Tympanometry

Using an appropriate sized probe tip, an acoustic seal was obtained and then the integrity of the middle-ear system was assessed by recording a tympanogram automatically over a pressure range of +200 MMH_2O and -200 MMH_2O using a 220Hz probe tone (Grayson-Stadtler 1723 instruction manual 1983).

4.4.1.4.3 Static compliance measures

The data for the static compliance measures were obtained from the tympanogram in accordance with the procedure outlined by Jerger & Northern, (1980).

4.4.1.4.4 Acoustic reflex threshold testing

This test was performed at the point of maximum compliance as displayed by the tympanogram (Martin, 1981). The presence of an acoustic reflex was defined as a change in middle-ear compliance of greater than 0.01 ml directly following acoustic stimulation.

Contralateral reflex thresholds were elicited for steady pure tones in the octave frequency range of 250Hz to 4000Hz . The ascending method of threshold exploration beginning at 70dBHL was used to determine thresholds (Hodgson, 1980). The results were recorded manually on the impedance recording card.

4.4.2. Criteria for interpreting the results obtained during session I - PHASE II

4.4.2.1 Pure tone audiometry

The ANSI (1969) scales, as cited by Green (1978), was used to evaluate hearing acuity. Pure tone thresholds ranging from 10dB to 26dB for all test frequencies was regarded as normal. Any threshold above 26dB was regarded as abnormal. Furthermore if a bone-air-gap greater than 10dB was obtained, then the subject was regarded as having an abnormal result.

4.4.2.2 Speech audiometry

4.4.2.2.1 Speech reception threshold (SRT)

The obtained SRT had to be within ±5dB of the pure tone average (Hodgson, 1980).

4.4.2.2.2 Speech discrimination testing

A maximum discrimination score of between 90% and 100% at 35dBsl was regarded as normal (Goetzinger, 1978; cited in Martin, 1981).

4.4.2.3 Impedance audiometry

4.4.2.3.1 Tympanometry

Tympanograms were classified according to the system advocated by Jerger, (1970). Only subjects with normal TYPE "A" tympanograms with maximum compliance occurring in the pressure range of +50mmH_2O to -50mmH_2O were regarded as normal (Jerger , 1970).

4.4.2.3.2 Static compliance measures

Only measures ranging between 0,28cc and 2,5cc were considered to be within normal limits for adults (Jerger and Northern, 1980).

4.4.2.3.3 Acoustic reflex thresholds

Reflex thresholds of 70dBsl to 90dBsl were considered to be within normal limits (Jerger et al., 1972). Any threshold above or below this range was regarded as abnormal.

From the 68 subjects who were assessed in Session I Phase II, two males and three females were excluded from the sample due to their having middle ear problems as evidenced in abnormal pure tone, speech audiometric and impedance test results. As as result a total of 63 subjects comprising of 31 males and 32 females were assessed in Session II. Session II involved the elicitation of BAERs which occurred half an hour after Phase II of Session I for each subject.

4.4.3 Description of procedure for session II : BAER TESTING

All subjects were assessed in the supine position, with appropriate head propping by using a pillow to minimize postural muscle activity in the neck and head (Chiappa et al. 1979, and Bauch et al. 1980). The nature of the test was briefly explained to each subject prior to actual testing.

4.4.3.1 Instructions to subjects

Subjects were encouraged to relax and to fall asleep during the recording session (Martin and Moore, 1977) as this would reduce myogenic influences on the recordings. Therefore, actual testing commenced only if subjects appeared to be relaxed or asleep.

4.4.3.2 Electrode placement (Preparation of subject)

The electrode sites were thoroughly cleaned of all debris with omni-prep skin preparing paste. Thereafter, the electrode sites were lightly abraded (Chiappa et al., 1979 and Coats & Jerger 1980), so as to assist in reducing skin resistance (impedance) to below 3000Ω. The self-adhesive silver-silver chloride electrodes were then arranged so that the electrical potential difference was measured between a pair of electrodes (bipolar derivation) as suggested by Schwartz and Berry, (1985). The positive (active) electrode was placed on the high forehead position (Schwartz and Berry, 1985) or on the Fz position as suggested by Martin (1977), in accordance with the International 10-20 system proposed by Jasper (1958), cited by Schwartz and Berry (1985). See Figure 9 below.

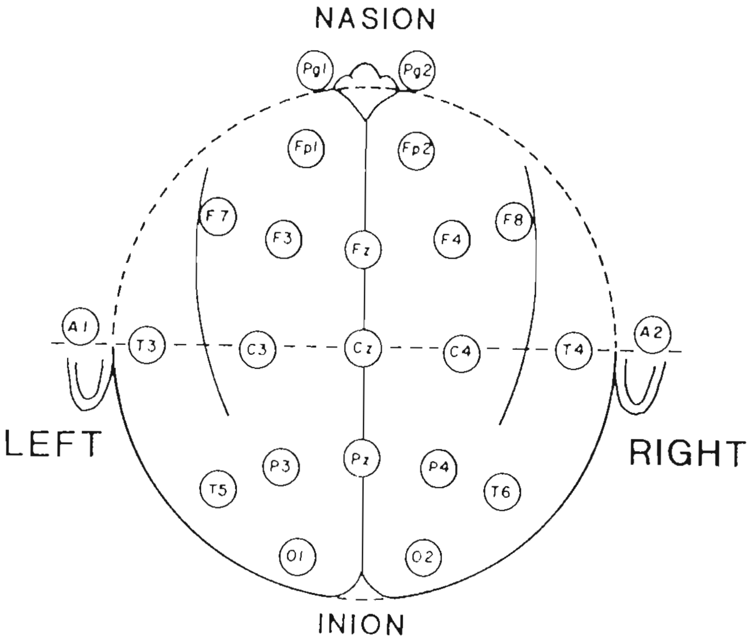


Fig. 9 : Electrode positions in accordance with the International 10-20 system proposed by Jasper, (1958).

The negative (reference) electrode was placed on the ipsilateral mastoid (T4) while the contralateral mastoid (T3) was used as the site for the common (ground) electrode (Schwartz & Berry, (1985). The positive (active) electrode was placed on the forehead just below the hairline (Chiappa et al., 1979). Prior to fixing of the electrodes to the skin, standard EEG electrode paste (collodion) was applied between the electrode and the surface of the skin. Thereafter, the electrodes were fixed firmly to the skin.

4.4.3.3 Pre-amplifier functional set-up

The recording electrodes were connected to a high gain (10^4) differential, low noise biological pre-amplifier (the Cadwell Quantum 84 pre-amplifier). The electrodes were tested for impedance after 10 minutes of allowing the electrodes to settle. Impedance had to be below 3000Ω before testing commenced (Chiappa et al., 1979).

4.4.3.4 Filter settings

In order to optimize response clarity by reducing signal-to-noise ratio a low cut pass band filter setting of 100Hz and a high cut band pass filter of 3000Hz was chosen. These filter settings have been suggested both by the Cadwell programme for BAER testing as well as by Jerger et al., (1985).

4.4.3.5 Stimulus presentation

Click stimuli with a duration of 100μsec were presented monaurally to the target ear via a TDH-39P earphone which was housed in an Amplivox free field audio-cup. The headphone position was held constant for all subjects.

4.4.3.6 Stimulus intensity

The stimuli were presented at 70dBnHL in order to observe the best waveform that the ear is capable of producing (Jerger et al., 1985).

4.4.3.7 Click rate

A rate in the vicinity of 10-20 clicks is normally appropriate to ensure observation of all component waves and to obtain "normal" latencies (Jerger et al., 1985). A click rate of 11.29per sec was presented in this project. This is in keeping with the programmed test protocol of the Quantum 84 (Quantum 84 clinical instruction manual, 1984).

4.4.3.8 Polarity of the stimulus

An alternating click polarity was used in order to maintain signal constancy from subject to subject (Jerger et al., 1985 and Reid et al., 1983).

4.4.3.9 Masking noise and intensity levels

Broadband noise as suggested by Parker and Thornton (1975), was used to mask the non-test ear during the masking condition. Broadband noise was selected as it is a good match for masking all click frequencies equally (Parker and Thorton, 1975). In view of Ozdamar and Stein's (1981), claim that the transcranial attenuation of sound traveling to the contralateral ear is approximately 60dBHL for click stimuli, the researcher chose to assess the effects of three levels of masking intensity on the monaural auditory evoked brainstem response. These intensities were arbitarily chosen as 50dBHL, 60dBHL and 70dBHL.

4.4.3.10 Sweep time

The sweep time was set at 1 so that each of the 10 divisions of the screen represented 1 msec. Therefore, the BAER was observed over a time frame of 10 msec poststimulus.

4.4.3.11 Number of clicks presented per trial

A total number of 2048 clicks were presented to ensure waveform build up and clarity.

4.4.3.12 Recording of responses

All responses were recorded using the built in ALPS printer with the procedure outlined in the Quantum 84 clinical instruction manual (1984).

4.4.3.13 Artifact rejection

This was switched on and artifacts were automatically rejected by the evoked potential system.

The actual test run was initiated as soon as all test parameters were set. Prior to any stimulus presentation a control run was done to allow for comparing and identifying of true responses. NB. See appendix I for a summary of the test protocol used in this study.

4.4.4 Measurements made

Amplitude and latency measurements were made and recorded automatically by the built in computer of the Quantum 84 evoked potential system. These measurements were made for all 63 subjects who participated in Session II. The measurements made included the following:

4.4.4.1 Absolute latencies

The absolute latencies of peaks I to VI in msec in the absence of masking was recorded from the target ear to provide relevant data to realize the four aims of this study.

The absolute latency of peaks I to V in the presence of the three levels of contralateral masking were recorded from the target ear to provide relevant data to realize aims three and four of this study.

These were made from stimulus onset to the positive peak of each component wave of the BAER.

4.4.4.2 Relative or interpeak latencies

The relative or interpeak latencies in msec of peaks I-III, III-V and I-V both in the absence and presence of the three intensity levels of contralateral masking were recorded from the target ear to provide relevant data to realize the four aims of this study.

4.4.4.3 Absolute amplitude

Peak to trough amplitudes of peaks I and V in microvolts, both in the absence and in the presence of the three intensity levels of contralateral masking were recorded from the target ear to provide sufficient relevant data to realize the four aims of this study.

These were measured from the positive peak to the following negative trough of each component wave of the BAER.

4.4.4.4 Amplitude ratio

This ratio was obtained by comparing the amplitude of peak V to peak 1. The ratio was obtained by dividing the amplitude of peak V by the amplitude of peak I (Musiek and Gollegly, 1985).

This amplitude ratio was calculated for the target ear both in the absence and presence of the three intensity levels of contralateral masking. Data for the calculation of this ratio was obtained from 4.4.4.3 above. The obtained values were used to realize aims one, two, three and four of this study.

4.4.5 Final selection of subjects

A total of 63 subjects underwent BAER testing in session II. However, the responses obtained from one male and two females were excluded from the final analysis of the results. One female and the one male subject were excluded because the investigator was unable to reduce inter-electrode imped-

ance to below 3000Ω, while the responses obtained from the last excluded female subject were erratic showing considerable baseline shifts. This subject was unable to tolerate lying still for the duration of the test.
Thus, a total of 60 subjects with an average age of 20.73 years; comprising of 30 males whose average age was 21.33 years and 30 females whose average age was 20.33 years, contributed relevant data for the purpose of this study.

Table 7 below illustrates the details of the subjects who finally contributed data for the completion of the project.

Table 7 : Subject Characteristics

	No.	$\bar{x}$ AGE (years)
Males	30	21.3
Females	30	20.3

The obtained raw data from the 60 subjects were then subjected to various statistical tests and procedures. The results thereof will be presented in the forthcoming chapter.

CHAPTER 5

RESULTS OF THE INVESTIGATION

The results of the investigation will be presented in four parts. Part one a presentation of the diagnostic reference data for the clinical interpretation of the BAER, while part two will focus on the sex effect on the BAER obtained from the group of normal hearers. Part three will concentrate on the effect of contralateral masking on certain aspects of monaural BAER, while part four will consider the effects of both sex differences and contralateral masking on selected aspects of the monaural BAER.

NB. 1) All significant values obtained are reported at the 0,05 α level in parts two, three and four of the study.

2) The results for part one of the study emerged from the use of conventional statistical procedures which allowed for the generation of means (X), ranges and standard deviations. The results for parts two, three and four were generated by using the multivariate analysis of variance (MANOVA) as suggested by Lazarus (1989); Murray (1989) and Maharaj (1989). The MANOVA statistical procedure is renowned as being a more stringent and robust measure to test for significant differences between and among multiple dependent variables.

5.1 Part one : The establishment of diagnostic reference data for the clinical interpretation of the BAER

The data for analysis were obtained from 60 normal hearing Indian female (N = 30) ($\bar{X}$ age 20.33) and males (N = 30) ($\bar{X}$ age 21.33) aged between 18 and 25 years. ($\bar{X}$ age = 20.73). These data, reflect measurements of absolute and relative latencies of peaks I to VI in msec and absolute amplitudes of peaks I and V and the relative amplitude of peak V compared to peak I in micro-volts. All raw data (Refer to Appendix G) are representative of monaural responses obtained from the right ear of each subject in the absence of masking. The data, which were subjected to appropriate statistical treatments are reflected for (30 females and 30 males) i.e. the combined group (N=60) in Table 8 and for 30 females and 30 males in Table 9.

Table 8 below summarises the statistical analyses made on the various latency and amplitude measurements obtained from the combined group (N=60).

TABLE 8 : Summary statistics for absolute and relative latencies in milli-seconds and absolute and relative amplitude measurements in micro-volts of the monaural BAER for the combined group. (N=60)

BAER Measures	Statistical Measures		
Absolute Latencies (msec)	$\bar{X}$	Range	SD
Peak I	2.08	1.87-2.29	0.08
Peak II	3.02	2.17-3.37	0.18
Peak III	4.12	3.71-4.52	0.17
Peak IV (N=54)	5.13	4.42-5.83	0.30
Peak V	6.00	5.50-6.58	0.23
Peak VI	7.49	6.54-8.21	0.32
Relative Latencies (msec)			
Peak I-III	2.01	1.69-2.41	0.15
Peak III-V	1.88	1.48-2.41	0.18
Peak I-V	3.90	3.45-4.37	0.22
Absolute Amplitudes (µV)			
Peak I	0.17	0.08-0.36	0.05
Peak V	0.24	0.08-0.50	0.08
Relative Amplitude (µV)			
Peak V:I	1.50	0.55-3.85	0.67

N.B. For Peak IV, Combined group N = 54

Table 8 above reflects the diagnostic reference data for the various latency and amplitude measurements obtained from the combined group (N=60).

Table 9 reflects a summary of the statistical analysis conducted for the various latency and amplitude measurements obtained among 30 female and 30 male subjects.

This table was drawn up for two specific purposes, viz;

i. to reflect diagnostic reference data for the normal BAER in male and females separately, and

ii. to allow the investigator to use the data in the table to assess the effect of sex difference on the normal BAER in 30 female and 30 male subjects.

Table 9 : Summary statistics for absolute and relative latencies in milli-seconds and absolute and relative amplitude measurements in micro-volts of the monaural evoked BAER in females (N=30) and males (N=30)

BAER Measures (P=Peak)	Statistical Measures					
	Females (N=30)			Males (N=30)		
Absolute Latencies	$\bar{X}$	Range	SD	$\bar{X}$	Range	SD
P I	2.06	1.87-2.19	0.07	2.11	1.92-2.29	0.10
P II	3.02	2.71-3.29	0.14	3.04	2.79-3.37	0.14
P III	4.08	3.71-4.52	0.18	4.16	3.87-4.50	0.16
P IV	5.10	4.42-5.83	0.37	5.17	4.58-5.52	0.21
P V	5.98	5.54-6.58	0.24	6.03	5.50-6.42	0.22
P VI	7.44	6.75-7.96	0.30	7.55	6.54-8.21	0.33
Relative Latencies						
P I-III	2.00	1.74-2.41	0.16	2.03	1.69-2.37	0.15
P III-V	1.87	1.48-2.41	0.18	1.87	1.56-2.29	0.17
P I-V	3.84	3.49-4.37	0.22	3.91	3.45-4.33	0.23
Absolute Amplitude						
P I	0.17	0.08-0.36	0.05	0.16	0.09-0.29	0.05
P V	0.25	0.09-0.44	0.08	0.22	0.08-0.50	0.08
Relative Amplitude						
P V:1	1.50	0.74-2.50	0.51	1.50	0.55-3.85	0.81

N.B. For Peak IV; Female No. = 26
Male No. = 28

Table 9 represents the means, ranges and standard deviations for the various BAER measurements as obtained for the females and males respectively.

These tables (8 and 9), therefore, represent the diagnostic reference data related to the first aim and hypothesis of the study.

5.2 Part Two : The effect of sex difference on the normal monaural BAER

The results in table 9 were further analysed to see if there were any sex differences with respect to certain characteristics of the monaural BAER. The characteristics studied were as follows:

i. absolute latencies of peaks I to V

ii. Relative latencies of peaks a) I-III
  b) III-V
  c) I-V

iii. Absolute amplitude of peak V

iv. Amplitude ratio of peaks V:I

Sex differences in the BAER have been previously reported by Jerger et al. (1980) and Stockard et al. (1978).

In this study, the effect of sex difference on certain aspects of the monaural BAER was analysed by using the multivariate analysis of variance (MANOVA) statistical procedure. The obtained values were generated by using the SAS/STAT computer program, (1988). (See appendix H for the instructions used to run the MANOVA statistical program).

In analysing the raw data for an overall sex difference, table 10 reveals the MANOVA test criteria and exact F statistic suggesting no overall sex effect.

Table 10 : <u>MANOVA test criteria and exact F statistic for the hypothesis of no overall sex effect</u>.

	S = 1		M = 4	N = 110.5	
Statistic	Value	F	Num DF	Den DF	Pr > F
Wilks' Lamba	0.8782	3.0912	10	223	0.0010
Pillai's Trace	0.1217	3.0912	10	223	0.0010
Hotelling-Lawley Trace	0.1386	3.0912	10	223	0.0010
Roy's Greatest Root	0.1386	3.0912	10	223	0.0010

Table 10 above, reveals a consistent agreement among the four statistics that there is an overall sex difference effect on the normal monaural BAER.

Table 11 below provides a summary for the F-test on the effect of sex difference on the various latency measures of the normal monaural BAER.

Table 11 : <u>The F-test for the effect of sex difference on various latency measures of the normal monaural BAER.</u>

Absolute: Peaks	Statistical Values				
	DF	Type 1 SS	Mean Square	F-value	Pr > F
I	1	0.075	0.075	7.70	0.0060
II	1	0.037	0.037	1.27	0.6680
III	1	0.478	0.478	14.54	0.0002
IV	1	0.517	0.517	0.16	0.6900
V	1	0.226	0.226	4.46	0.0357
Relative: Peaks					
I-III	1	0.193	0.193	8.37	0.0042
III-V	1	0.000	0.000	0.00	0.9920
I-V	1	0.135	0.135	2.74	0.0990

It is apparent from Table 11 that there is a significant sex difference effect on the following latency measures of the normal BAER;

i. Peak I showed a significant effect at 0,0060 (F = 7.70; DF = 1;59)

ii. Peak III showed a significant effect at 0,0002 (F = 14.54; DF = 1;59)

iii. Peak V showed a significant effect at 0.0257 (F = 4.46; DF = 1;59)

iv. Relative latency of peaks I-III showed a significant effect at 0,0042 (F = 8.37; DF = 1;59)

In all of the above latency measures, females showed that they had significantly ($p < 0.05$) shorter latencies than their male counterparts. The mean differences for the following peaks were;

i. peak I = 0.05 msec
ii. peak III = 0.08 msec
iii. peak V = 0.05 msec
iv. the relative latency of peaks I-III = 0.03 msec.

These mean differences were gleaned from Table 9.

The other measures viz., peaks II and IV and the relative latencies of peaks III - V and I -V showed no significant effects.

Table 12 below reflects the F-test on the effect of sex difference on the selected amplitude measures of the normal monaural BAER.

Table 12 : <u>The F-test for the effect of sex difference on two selected amplitude measures of the normal monaural BAER</u>

Amplitude measures	Statistical Values		
	DF	F-value	Pr > F
Absolute amplitude Peak V	1	1.64	0.2011
Relative amplitude Peak V:I	1	0.27	0.6008

The statistical results reflected in Table 12 reveal that there were no significant sex-difference effects on the two amplitude measurements made.

5.3 Part Three : The effect of contralateral masking on the normal monaural BAER

The raw data (See appendix G) relating to latency and amplitude measurements were analysed to see if there were any significant effects of contralateral masking under three (50, 60 and 70dBHL) conditions of masking on the monaurally elicited BAER. The characteristics of the BAER that were studied were the same as for hypothesis II.

A review of selected available studies that have addressed this question appear to be at variance as to whether masking does or does not have effects on the monaural BAER. Chiappa et al. (1979), and Humes & Ochs (1982), report on an absence of making effects while Rosenhamer and Holmkvist (1983), report on the presence of masking effects on the monaural BAER.

In this study, the effects of contralateral masking on the monaural BAER was examined by using the Multivariate Analysis of Variance (MANOVA) statistical procedure. The results were generated by using the SAS/STAT computer program (1988).

The results presented in Table 13 reflect the summary of the MANOVA and exact F-statistics for overall masking effect, at 50, 60 and 70dBHL.

Table 13 : MANOVA test criteria and F-Statistic for the hypothesis of no overall masking effect, at 50, 60 and 70dBHL.

	S=3 M=3 N=110.5			
Statistic	Value	F	Num DF	Pr > F
Wilks' Lamba	0.9343	0.5112	30	0.9867
Pillai's Trace	0.0669	0.5135	30	0.9863
Hotelling-Lawley Trace	0.0689	0.5092	30	0.9871
Roy's Greatest Root	0.0386	0.8699	10	0.5622

Note: F-Statistic for Roy's Greatest Root is an upper bound.

Table 13 above, reveals a consistent agreement among the four statistics that there is No overall masking effect on the normal monaural BAER at the intensity levels reflected.

5.4 Part Four : Interactional effects of difference in sex and contralateral masking on the monaurally elicited BAER

The obtained data (see appendix G) relating to latency and amplitude measurements were analysed to see if there were any interactional effects of sex difference and contralateral masking on the monaurally elicited BAER. The characteristics of the monaural BAER that were studied were the same as for hypotheses II and III.

A thorough review of selected literature did not yield information regarding the effect of sex and masking on the monaurally elicited BAER.

In this study, this question was addressed by examining the interactional effects by using the Multivariate Analysis of Variance (MANOVA) statistical procedure. The results were generated by using the SAS/STAT (1988) computer program.

The results presented in Table 14 below reflects a summary of the MANOVA TEST CRITERIA and F- Approximations for overall sex and masking effects on the monaurally elicited BAER.

Table 14 : MANOVA test criteria and F approximations for the hypothesis of no overall sex and masking effects.

	S=3	M=3	N=110.5	
Statistic	Value	F	Num DF	Pr > F
Wilks' Lamba	0.9553	0.3427	30	0.9997
Pillai's Trace	0.0452	0.3445	30	0.9996
Hotelling-Lawley Trace	0.0461	0.3410	30	0.9997
Roy's Greatest Root	0.0272	0.6139	10	0.8013

Note: F-Statistic for Roy's Greatest Root is an Upper Bound.

Table 14 above reveals a consistent agreement among the four statistics that there is NO overall sex and masking effect on the monaural BAER.

5.5 SUMMARY

The raw data obtained in this study were subjected to various statistical procedures and resulted in the generation of information summarized below.

1. Table 8 reflects the diagnostic reference data obtained from the combined group of normal hearing Indian students (N = 60). The group had a mean age of 20.73 years.

2. Table 9 reflects the diagnostic reference data obtained from normal hearing males and females separately. Thirty females with a mean age of 20.33 years and thirty males with a mean age of 21.30 years contributed the information for the generation of the clinical diagnostic reference data for females and males.

3. Part two of this section examined the effects of sex difference on the normal BAER. The results revealed that there was a significant sex effect on the absolute latency of peaks I, III and V and on the relative latency of peaks I-III (see Table 11). Females tended to show consistently shorter latency values for the above named latency measures. There were no significant effects on the amplitude measures studied (See Table 12). The MANOVA Test Criteria and exact F-statistic in Table 10 show consistent agreement that there was an overall sex effect on the normal BAER obtained in the group under study.

4. Part three of this section examined the effect of contralateral masking on the normal monaural BAER. Table 13

illustrates the MANOVA Test Criteria and exact F-test for the hypothesis of no overall masking effect on the monaural BAER under the masking conditions specified. The results revealed that contralateral masking has no significant effect on the various latency and amplitudes measures studied.

5. Part four of this section focused on assessing the effects of both sex differences and contralateral masking on the normal monaural BAER. Table 14 shows the MANOVA Test Criteria and F-approximations for the hypothesis of no overall sex and masking effect. The results revealed that there were no sex and masking interactional effects on the various latency and amplitude measures studied.

The results presented in chapter 5 will be discussed in the ensuing chapter.

CHAPTER 6

DISCUSSION

The objectives of this investigation were to establish diagnostic reference data for the BAER in a group of normal hearing young Indian university students, and to investigate the effects of sex difference and contralateral broadband masking on the normal monaural BAER. Each of the aims presented, generated the hypotheses stated in chapter 1. The results presented in the previous chapter will now be integrated and discussed with respect to the aims and hypotheses which motivated this study.

6.1 **HYPOTHESIS I:** There are reference data for Indian students; 18-25 years.

Aim : TO ESTABLISH DIAGNOSTIC REFERENCE DATA FOR THE BAER IN A GROUP OF INDIAN UNIVERSITY STUDENTS.

This aim emerged from the strong recommendation made by various research authors consulted, eg., Schwartz and Berry (1985), who stated that it is critical to the measurement (BAER) that appropriate diagnostic reference data be collected within each laboratory or clinic. This recommendation stems from the evidence that the normal BAER is influenced by many non-pathological factors (See chapter 3 for the relevant discussion).

In addition, the data derived in this investigation is a reflection of the particular test system (Caldwell Quantom 84) and test protocol to be used in the Audiology Clinic at the University of Durban-Westville. Furthermore, the generation of the diagnostic reference data was essential as it allowed for the investigation of the effects of sex difference and contralateral broadband masking on the normal monaural BAER. (ie. to test hypotheses ii, iii and iv stated in Chapter 1.).

The results presented in part 1, table 8 in chapter five, provide a summary of the latency and amplitude measures made on the monaural BAER elicited in 60 normal hearing young students. The diagnostic reference data reflected in table 8 (see page 135) is to be discussed in relation to previously published data under the following headings:

a) Absolute Latencies
b) Relative Latencies
c) Absolute Amplitudes
d) Relative Amplitude Ratio

6.1.1 ABSOLUTE LATENCIES

Table 8 (see page 135) reflects the overall group absolute latency values for peaks I to VI. The obtained range extends from 2.08 to 7.49 msec in response to clicks presented at 70dBnHL. The latency range for peaks I to V was 2.08 to 6.00 msec. Beagley and Sheldrake (1978), in reporting their "normative data" obtained from 5 male and 5 female subjects (age range 21-30 years) for peaks I to V show a similar latency range of 2.1 to 6.1 msec in response to clicks presented at 70dBsl.

However, in comparing the findings of the above studies to those obtained in other clinics and laboratories (See table 2, page 39), it is evident that there are small variations between and among the values reported. These variations may be attributed to differences in intensity, polarity of clicks, repetition rates, filter setting and other aspects of test protocols used. The fact that these discrepancies between and among clinics and laboratories exist, highlights the need for each facility to generate it's own diagnostic reference data.

An examination of the individual studies reported in Table 2 (see page 39) revealed two major differences:

a. Some researchers did not always clearly define the reference intensity level for the clicks used, ie. whether the clicks were presented in dBsl, dBnHL, dBspl or dBHL. This aspect is considered important as it reflects on exactly how loud a click is presented. Furthermore, it is well documented that there is a direct relationship between intensity level and the latency of the peaks obtained in the BAER (Rowe 1978, Stockard et al. 1978), ie. as the intensity of the click increases, the latencies of the peaks decreases and vice-versa (Coats, 1978; Galambos & Hecox, 1978 and Moore 1983). It is, therefore, suggested that investigators clearly define the reference intensity levels used. This suggestion is supported by the American Electroencephalic Society guidelines for clinical evoked potential studies (1984). Adherence to these guidelines would permit the uniform use of reference intensity levels and would allow for the comparison of reference data between and among clinics and laboratories.

b. Investigators did not always clearly define the polarity of clicks used. BAERs are affected by the acoustic phase of clicks (Gastone et al. 1987). Rarefraction clicks evoke shorter peak I and V latencies (Coats and Martin 1977); although these vary considerably among subjects. Condensation clicks, however, appear to delay peak I and V latencies (Schwartz and Berry 1985), while alternating clicks do not seriously compromise BAERs, but serve to enhance the clarity of the response (Schwartz and Berry, 1985). It is, therefore, recommended that investigators clearly define the acoustic phase of clicks used in the generation of "normal" reference data. This would facilitate the development of uniform procedures and hence allow for inter-facility comparison.

It is conceivable that among the variables mentioned earlier, both variation in intensity reference levels and the polarity of clicks used, may principally account for the differences in normal values reported in Table 2 (see page 39) and in this investigation.

In considering the appearance of individual peaks, all six peaks except for peak IV were consistently elicited in this investigation. Six subjects (2 males and 4 females) did not produce clear and measurable peak IV latencies. According to Beagley and Sheldrake (1978), peak IV tends to be a more labile peak while Rowe (1978), states and it may, in some normal subjects be absent. This may be attributed to the fact that peak IV sometimes tends to fuse with peak V thereby making it indistinct (Chiappa et al. 1979).

Peak I was consistently elicited in a latency range of 1.87 to 2.29 msec. This finding is consistent with Picton's (1986), recommended range of 1.4 to 2.5 msec.

The mean of 2.08 msec, however, appears to be slightly delayed when compared to the means reflected in Table 2 (see page 39). Among the other variables mentioned earlier, this may be attributed to variations in testing and measurement protocols used in the different laboratories and clinics, particularly to differences in intensity levels and click phases used.

Generally, the absolute latencies of peaks II, III, IV and V show close approximation with those presented by Beagley and Sheldrake (1978). However, in comparison with the other studies reflected in Table 2, these latencies appear to be slightly delayed, but the inter-facility standard deviations show close agreement. This provides support for the claim that the BAER is a stable and reliable measure at moderate to high intensity levels.

The inspection of the peak V latency, which, according to Schwartz & Berry (1985); Stockard et al. (1978), should occur within 4.00 msec after peak I, reveals that the finding of this study is consistent with the above i.e. peak V (see table 2) had a mean latency period of 3.90 msec after peak I. According to several researchers, e.g. Beagley and Sheldrake (1985); Stockard et al. (1978) and Picton (1986), peak V is the most consistent and prominent of the BAER, and is probably most useful diagnostically. It's appearance at a mean latency period of 6.00 msec in this study conforms well with findings reported by Picton et al. (1974), at 5.8 msec; Stockard Rossister (1977), at 5.9 msec; Rosenhamer et al.

(1978), at 5.9 msec and of Rowe, (1978) at 5.8 msec. Furthermore, the standard deviation of the peak V latency as reported by all these researchers did not differ significantly from that of this study ie. a standard deviation of 0.23. Clearly, peak V latency appears to be robust in character. It is reliable and stable even under varying measurement conditions. This contention is supported by the evidence presented in Table 2. It is, therefore, not surprising to note that peak V (a rostral component of the BAER) has received widespread clinical attention in differential diagnosis of otoneurologic disorders, as well as for the estimation of hearing sensitivity (Schwartz and Berry, 1985).

It is apparent that the reference data for the absolute latency of the various peaks in this study are in close agreement with those presented by Beagley and Sheldrake (1978), whilst appearing to be slightly delayed when compared to those of other facilities seen in Table 2. These variations in absolute latency values between and among clinics and laboratories are expected to occur especially in view of differences in test conditions and variations in stimulus parameters (e.g. when actual intensity levels vary). This contention is supported by Chiappa et al. (1979), and Coats, (1978). However, peak V has again demonstrated it's robust nature by showing it's stability under varying test conditions. The finding that there were some overall variations between this study and of those summarised in Table 2, illustrates and highlights again the need for each clinic or test facility to establish it's own diagnostic reference data.

6.1.2 RELATIVE OR INTER-PEAK LATENCIES

Table 8 (see page 135) also reflects the mean relative latency values obtained in the group of 60 normal hearing students. These include the values for the relative latencies of peaks I-III; III-V and peaks I-V. Crucial to the differential diagnosis of space occupying lesions, either intrinsic or extrinsic to the brainstem, is the time difference between peaks. These time differences are reflected by the time intervals between the following:

i. peaks I-III as representing peripheral transmission time from stimulus onset to the ponto-medullary junction in the lower pons (Stockard et al., 1978)
ii. peaks III-V as reflecting central transmission time from caudal pons to the midbrain (Schwartz and Berry, 1985) and
iii. peaks I-V as representing both peripheral and central transmission time from stimulus onset to the midbrain (Schwartz and Berry, 1985).

Peripheral transmission time is determined by middle-ear function, cochlear mechanics, cochlear transduction, synaptic and cochlear nerve conduction velocity while central transmission time is associated with fibre conduction velocity and synaptic transmission of brainstem tracts and nuclei (Cornacchia et al., 1983).

The mean relative latency values obtained in this investigation were as follows;

peaks I-III = 2.01 msec
peaks III-V = 1.88 msec
peaks I-V = 3.90 msec

These values coincide well with previously published data as reflected in Table 2. Furthermore, these values fit in well with the suggested values presented by Schwartz and Berry (1985); these being ±2 msec for peaks I-III; and III-V and ±4 msec. for peaks I-V in normal hearing subjects. According to Rowe (1978), these relative latency values should not vary between and among laboratories and clinics using the same <u>rate</u> of stimulus presentation.

In summary, this investigation established relative latency values which are similar to those found in other clinics and laboratories having used a click stimulation rate of 10-12 clicks per sec (see Table 2). The lack of variability in these measures between and among facilities using the same click rate, makes them robust measures of peripheral and central transmission time within the auditory system. Therefore, the interpeak latency measures are suitable for assessing pathologies which may affect the transmission of auditory impulses in the peripheral and brainstem part of the auditory system.

6.1.3 ABSOLUTE AMPLITUDES

In referring to Table 8 (see page 135), two absolute amplitude measurements were considered in this investigation. These were the absolute amplitudes of peaks I and V. The obtained values in μV were used for the computation of the more clinically acceptable amplitude measure. i.e. the amplitude Ratio of peak V compared to peak I.

There is consensus among researchers, viz. Schwartz & Berry, (1985); Rowe, (1978), and Chiappa et al. (1979), that absolute amplitude measures are not normally distributed; are highly susceptible to myogenic activity and noise levels; are difficult to replicate, and are easily influenced by minor alterations in recording techniques. Consequently, the measurement of absolute amplitudes do not enjoy the stability and reliability of their latency counterparts (Schwartz and Berry, 1985).

In this study, the mean peak I amplitude value was 0.17μV and that of peak V was 0.24μV. Chiappa et al. (1979), presented a mean peak I amplitude value of 0,28μV and a mean peak V value of 0.47μV. Stockard et al. (1978), published a mean value of 0,23μV for peak I and 0,35μV for peak V. It is clear that there are no close approximations between and among reported measures.

These reported variations in amplitude measures between and among normal hearers may be attributed to the present system of signal averaging and use of artifact rejection (Fernandes, 1989). Theoretically, a wanted evoked potential is extracted from ongoing EEG by signal averaging and the use of artifact rejection. That is, by increasing the signal-to-noise ratio. Waveform and amplitude build up is, therefore, a product of time-locked averaging together with the rejection of other contaminating artifacts, e.g. myogenic and other cerebral activity.

The problem is that there is no consensus among researchers on how much of averaging and/or artifact is required before a response is judged as acceptable or not. According to Hyde (1985), the choice of the number of clicks presented for averaging is often "based on popular consensus rather than on quantitative rationale". Due consideration has not been given to the influence of differences in "internal noise levels" among normal hearers when reference data are established. That is, some normal subjects may have higher internal noise levels, requiring longer periods of averaging with greater number of averages within a trial before eliciting an appropriate response than subjects who have lower internal noise levels (Hyde, 1985). Therefore, a choice of either 1048, 2000 or 2048 clicks to elicit a suitable averaged response may not be appropriate for all normal hearers. Furthermore, since the amplitude of a response is partly dependent on the number of averages that occur in a trial, it is reasonable to assume that response amplitudes will differ between and among individuals. This, therefore, may account for the variability in amplitude measurements that are reported in the literature.

Similarly, the use and control of artifact rejection to eliminate unwanted noise is not consistent in studies that have reported on normal amplitude values. It is, therefore, not surprising to find variations in the reported amplitude values between and among studies.

The consistent and appropriate application of signal averaging and management of artifact rejection needs to be given careful attention in future research. Attention needs to be focused on decisions pertaining to; the:

i. actual number of averages required in a trail (i.e. 1048, 2000 or 2048 clicks) before a response is regarded as representative of a "true neurogenic" response.

ii. Use and control of artifact rejection so that the final response is truely representative of the BAER without being contaminated by other artifacts.

A reasonable course of action, is to set the artifact rejection limits so that little of the "well behaved" (low variance) activity is rejected, while all of the high variance (bursts of electromyogenic noise) activity is. This may be done by "tuning" the rejection level while observing the displayed activity, so that only about 5-10% of the good activity is rejected.

Perhaps, the manufacturers of evoked potential systems need to incooperate additional desirable features that will allow for the display of the input EEG during averaging, rejection of trails in which large voltage artifacts occur and an assessment of amplitude variability within an averaging run.

The above may assist in establishing appropriate reference data for amplitudes which may be used routinely in BAER interpretation.

In the interim however, an important aspect to consider when inspecting the peak amplitude values of I and V, is that the peak V value is almost always larger than the peak I value in normal hearers. It has been suggested by Chiappa et al. (1979), that if the reverse occurs then it is quite likely that the result is indicative of an abnormal BAER.

In view of the reported variations in absolute amplitude measurements reported by different investigators, it is suggested that researchers wishing to use absolute amplitude measures for diagnostic purposes, must establish reference data which are particular to that test facility (Schwartz and Berry 1985). The writer concurs with the above but adds, that further consideration be given to the appropriate use of signal averaging and artifact rejection in the establishment of reference data for amplitude measurements. Thereafter, such reference data should be applied widely to assess how otoneurologic pathologies influence the measures, and to document the obtained patterns for ongoing comparisons.

6.1.4 RELATIVE AMPLITUDE - THE PEAK V:I AMPLITUDE RATIO

Table 8 (see page 135) reveals, that the mean amplitude ratio obtained in this investigation was 1.50. This is consistent with the findings of Chiappa et al., (1979); Rowe, (1978); and that of Starr & Achor (1975), who have all reported that a value greater than 1.00 be considered as normal. In order to detect abnormality, Musiek et al. (1984), state that amplitude ratio should be less than 1.00. Stockard et al. (1978), however, state that a complete absence of peak V in the presence of peak I is an indication of relative amplitude abnormality.

Differing in this opinion, Starr and Achor (1975), state that a peak V:I amplitude ratio of less than 0,5 at 55 dBsl is abnormal. Later in 1978, Stockard et al., suggested that a peak V absolute amplitude value which is reduced by more than 3 sd from the normal mean, together with a peak I amplitude that is larger than peak V, and an inter-trial variation of less than 10% are all necessary for the peak V:I amplitude ratio be defined as abnormal. Chiappa et al. (1979), agree with Starr and Achor (1975), that 10 of their 104 normal subjects displayed a peak I amplitude which was larger than peak V: The findings of this investigation are in part agreement with Starr and Achor (1975), and with Chiappa et al. (1979), since 12 subjects (5 females and 7 males) displayed peak I amplitudes which were larger than peak V, although the overall mean was 1.50. The observed differences in amplitude ratios appear to be due to normal variations that occur within and among normal individuals. This contention is in keeping with Stockard et al.'s (1977), statement that "alterations of BAER morphology in the absence of quantifiable latency or absolute amplitude abnormality are not considered abnormal per sé, because of the variability of BAER waveforms within and among normal individuals."

However, Schwartz and Berry (1985), are of the opinion that there is a dearth of well documented literature concerning the use of the V:I amplitude ratio in a large pathologic population. They suggest that considerable research is needed on the confounding effects of such variables as stimulus polarity, repetition rates, filter characteristics, electrode sites etc., prior to the general use of this measure in clinical practice. The investigator concurs with the above recommendation. Due consideration should also be given to inter and intra individual variations when examining amplitude data. Furthermore, an improvement in signal averaging and artifact control may aid in resolving in the issue of obtaining variable amplitude measures in normal hearers.

6.2 SEPARATE DIAGNOSTIC REFERENCE DATA FOR MALES AND FEMALES

In response to the suggestion made by several researchers, viz. Stockard et al., (1978); McClelland & McCrae, (1979); Jacobson et al., (1980); Jerger & Hall (1980), and Jerger & Johnson 1988), that diagnostic reference data be established separately for males and females, the raw data was further treated to reflect this separation.

Table 9, reflects the means, ranges and standard deviations for the various BAER measurements as obtained from 30 females and 30 males.

On inspection and comparison of the mean absolute latency values obtained for the two groups, it is evident that for all six peaks, females tended to show shorter latencies values than males. This is also evident for the peak I-III and peak I-V relative latency values.

The absolute and relative amplitude measures show no such differences, implying that there are no observable differences between sexes for these measures in this investigation. However, further research focusing on the appropriate use of signal averaging and artifact rejection may produce realistic amplitude measures in normal hearers. Once this has been achieved, it is suggested that the effect of sex difference on amplitude measurements be reassessed.

The question of whether there is a significant sex difference effect on the normal BAER, is to be addressed by hypothesis ii of the study. The discussion of the findings thereof will be presented later in this chapter.

In the interim, the fact that there are observed latency differences between the sexes as seen in Table 9 (see page 136), is supportive of the suggestion that separate diagnostic reference data be established for the two sexes. The establishment of such data, would prevent the clinician from applying inappropriate sex related reference data to interpret the BAER.

SUMMARY : Diagnostic reference data

The reference data established in this investigation are in part similar to those reported by Beagley and Sheldrake, (1978). The similarity in absolute and relative latency values between the two studies are probably due to the use of similar testing protocols. However, slight variations were noted when the present reference data for absolute and relative latencies were compared to other facilities as presented in Table 2.

These variations may be attributed to differences in test protocols used. The primary variables that may have contributed to the observed variations are differences in intensity levels and acoustic phase of clicks used. However, it was noted that the absolute latency of peak V remained less susceptible to the influence of differences in test protocols. Therefore, peak V appears to be a more robust and reliable measure in BAER testing. This contention is well supported by the findings of several previous researches e.g., Stockard et al., (1977); Rowe, (1978); Schwartz & Berry (1986) and others.

The relative latency measures of peaks I-III; III-V and I-V displayed little or no variation when compared to the other studies. This, according to Rowe (1978) is acceptable, especially if similar click presentation rates are used. Furthermore, relative latency measures are not entirely dependent upon stimulus intensity levels (Rowe, 1978). The finding that these peripheral and central transmission times remain consistent between and among facilities, allows for their use as reliable measures in diagnostic BAER audiometry.

The absolute amplitude measures obtained in this investigation (peak I and V) displayed greater variation when compared to previous research findings. A survey of selected studies, shows no close agreements in the values presented. Schwartz & Berry, (1985); Rowe, (1978), and Chiappa et al. (1979), all concur that absolute amplitude measures are not normally distributed and are highly susceptible to different variables (e.g. muscle potentials, noise, intensity of stimulus). It is, therefore, clinically acceptable to observe variations in values as reported by different clinics and laboratories. Thus it appears that at this stage, absolute measures of amplitudes are not suitable for diagnostic purposes. However, further research into the appropriate use of signal averaging and artifact rejection may contribute towards obtaining clinically useful reference data for amplitude measurements.

In presenting the finding on the peak V:I amplitude ratio, a mean value of 1.50 was obtained in this investigation. This finding is in keeping with the suggestion made by several researchers e.g., Chiappa et al., (1979); Rowe, (1978) and Starr & Achor (1975), that the norm for RA should be 1.00 or greater. However, there is some controversy in the literature as to the actual lower cut off point for a normal response, as some normal hearers show a larger peak I amplitude than peak V.

The last aspect relating to the establishment of diagnostic reference data focuses on presenting separate data for males and females. An examination of the data in Table 9 (see page 136), reveals that females show shorter mean absolute and relative latencies than males. The amplitude measures presented reveal a similar mean value for both sexes with overlaps occurring in the ranges. However, the sex difference effect needs to be re-assessed relative to the appropriate use of signal averaging and artifact rejection as discussed earlier.

The observation that there were differences between the sexes is strongly supportive of the suggestion that separate reference data be established for the sexes (Stockard et al., 1978; Jerger & Hall, 1980 and Michalewaski et al., 1980). This would allow for the accurate clinical interpretation of the BAER obtained in the two sex groups.

In concluding the discussion on the establishment of diagnostic reference data, it is the investigator's opinion that the obtained data be applied to a wide clinical population of known pathological conditions. This would serve to assess the clinical efficiency of the established reference data.

The question of whether sex difference has significant effects of the normal BAER is to be discussed in the ensuing part of this chapter.

6.3 **HYPOTHESIS II:** There is a significant difference between the monaural clinical BAER recorded from the target ears of males and females with respect to the following characteristics; the:

a) absolute latencies of peaks I to V.
b) relative latencies of peaks I-III; III-V and I-V.
c) absolute amplitude of peak V.
d) amplitude ratio of peak V compared to peak I.

The finding in this study supports the above hypothesis. Table 10 (see page 138) representing the MANOVA test criteria and exact F statistic for the hypothesis of no overall sex effect, reveals a consistent agreement among the four statistics (Wilk's Lamda, Pillai's Trace, Hotelling-Lawlay's Trace and Roy's Greatest Root) that there is an overall sex difference effect on the normal BAER in this study.

The overall finding of this study is consistent with several investigators who addressed the question of whether sex difference affects the BAER. Kjaer (1979), in presenting his motivation for expecting a sex difference effect on the normal BAER, states that many biological differences exist between the two sexes, leading to different patterns of diseases and physiological data and cites Pedersen and Klemar's (1974) finding, of differences in haemoglobin values between females and males as an example of an existing biological difference between the sexes. In the CNS, sex differences in brain size and weight have been described, e.g. Witelson (1989), and Dekaban, (1978). Holloway (1980), reported on significant correlations between brain size & weight and height & body weight in males. McGlone (1980), has also reported on differences in hemispherical asymmetry between the sexes (see Chapter 3, 3.8 for an overview of these studies). Bearing the above in mind, and considering Jewett et al.'s (1971), description of the BAER as a volume conducted response, it was reasonable to expect a sex difference effect on the normal BAER.

This expectation has been confirmed by findings of this study. Furthermore, there is general consensus among the following investigators, viz. Stockard et al., (1978); Beagley & Sheldrake, (1978); Kjaer, (1979); McClelland & McCrae, (1979); Jacobson et al., (1980); Jerger & Hall, (1980); Michalewaski et al. (1980), and Jerger & Johnson (1988), that there is a definite sex difference effect on the normal BAER.

It is evident from the various studies that the sex difference effect may influence one or more of the following measures of the BAER :

i. absolute latencies of peaks
ii. relative latencies of peaks
iii. absolute amplitudes of peaks.

The ensuing discussion will focus on speculative arguments and explanations for the observed sex difference effect on the BAER.

6.3.1 THE SEX DIFFERENCE EFFECT : ABSOLUTE LATENCIES

In this study females displayed statistically significant shorter latencies for peaks **I**, (F=7.70 ; DF=1;59 ;$p<0.05$), **III** (F=14.54 ; DF=1;59 ; $p<0.05$) and **V** (F=4.46 ; DF=1;59 ; $p<0.05$) and shorter (though not statistically significant) latencies for peaks II and IV (See Table 11, page 138).

The overall finding of shorter latencies amongst females, suggests that transmission of click stimuli at 70dBnHL is faster in females than in males. Several researchers, viz. Beagley & Sheldrake, (1978); Kjaer, (1979); Jerger & Hall (1980); Jacobson et al., (1980); Michalewaski et al. (1980), and Jerger & Johnson (1988), published similar findings, but focused their attention on the absolute latency of peak V. (i.e. the most consistent and robust absolute peak measure, Schwartz and Berry, 1985). This tendency for a shorter peak V latency in females was also demonstrated at several different intensity levels eg. Michalewaski et al. (1980), at 60, 70 and 80dBsl; Jerger and Hall (1980), at 70 and 90dBHL and Jerger and Johnson (1988), at 20, 40, 60 and 80dBHL.

However, none of the investigators consulted, have provided a definitive explanation for the observed sex difference effect. Hall (1984), summarises the situation by stating "The basis of the sex effect remains unknown". Nevertheless, various postulations and speculative arguments have been put forward in an attempt to explain the sex difference effect of obtaining shorter latencies in females than in males. As such, several factors may be operational in contributing to the sex difference effect.Michalewaski et al. (1980), Beagley & Sheldrake (1978), and Jerger & Hall (1980), concur with Stockard et al. (1978), who stated that the sex difference effect may be attributed to "differences in the anatomical distances of structures along successive stages of the auditory pathway". This contention speculatively relates to the presence of shorter distances between common synaptic junctions of the auditory pathway in females as compared to males. Kjaer (1979), attributes the sex difference effect to differences in body length in that he found males to be 13 cm taller than females. Although, he did not elaborate on this suggestion, it would appear that males would have had larger heads, brain & body size and mass than females. This relates well to Holloway's (1980) claim described earlier. It may imply that the volume conducted BAER would show longer latencies and smaller amplitudes in males than in females, i.e. a greater distance and volume would have to be traversed by the impulses to the pick up scalp electrodes in males.

Drawing purely, from subjective visual observations that the Indian females who participated in this study:

a. appeared to be smaller in stature and

b. appeared to have smaller body weights than their male counterparts, the present investigator is inclined to concur with the speculation put forward by Stockard et al. (1978), and Kjaer, (1979).

It is, therefore, speculated that in view of Witelson's, (1989); Dekaban's (1978), and Holloway's (1980), claims of larger brain size & mass and body height & mass in males, the BAERs may be different from those of females. This follows from the assumption that the volume conducted peak latencies of the BAER would have to traverse a larger volume and distance in the heads of males than in females.

However, correlates of these anatomical differences, for example, height, weight, and head size were not collected in this investigation and are thus not available for analyses or study. This, therefore, does not allow for the making of definite scientific judgements. However, the inclusion of such data and analyses in a future research project may aid in clarifying these speculations.

Another speculative argument that may account for the sex difference effect on absolute latencies is drawn from recent findings relating to auditory neuro-chemistry. According to Caspary (1986), as cited by Musiek (1989), the application of an excitatory neurotransmitter (ASPARATE) to a neuron of the cochlea nucleus results in an increased firing rate for acoustic stimulation and spontaneous activity, while the application of an asparate antagonist results in the opposite effect. Assuming that such neuro-transmitters exist in the different synaptic junctions of the auditory system, it is conjectured that females may be partial to having a higher concentration of excitatory neuro-transmitters at the different synaptic junctions than males (this may be due to females having smaller head size and volumes). It is then envisaged that upon acoustic stimulation, a higher or faster excitatory effect is present in the synaptic junctions in females, thereby leading to shorter latency measures for the different peaks. However, this is purely a conjectural point of view, requiring further neuro-chemical research to assess whether the sex difference effect is in any way related to differences in neural substrates between males and females.

In the same light, Musiek (1989), cites Caspary (1986) in stating that certain auditory nuclei eg. the cochlear nuclei are sites for high metabolic activity. It is quite conceivable that females may show a high rate of metabolism in the different parts of the auditory system than males, thereby accounting for shorter peak latencies in the BAER. Furthermore, some credibility to the above argument is given by Jacobson et al.'s, (1985) description of the function of the myelin sheath which surrounds the cochlear nerve. They state that the myelin sheath has the effect of increasing the efficiency and speed of signal transmission via saltatory conduction (conduction of impulses across the nodes of Ranvier of the cochlear nerve) especially during an increased period of metabolic activity (as seen during acoustic stimulation). It is, therefore speculated, that a higher rate of metabolism together with a more efficient myelinated cochlear nerve in females, may allow for faster transmission of impulses than in males, and hence giving rise to shorter peak latencies in the BAER's in females. Further research, related to auditory nerve metabolism, cochlear nerve transmission and neurochemical analyses between sexes may clarify the observed sex difference effect.

In view of the demonstrated significant sex difference effect on the absolute latency measure, it is considered acceptable to establish separate diagnostic reference values for males and females. This contention is in keeping with the recommendations made by Stockard et al. (1978), and Schwartz & Berry, (1985).

6.3.2 SEX DIFFERENCE EFFECT : RELATIVE LATENCY MEASURES

The peaks I-III relative latency measure emerged as being statistically different (F=8.37 ; DF=1;59 ; p<0,05) with respect to the sex difference effect. Females displayed a faster mean difference time of 0,03 msec than their males counterparts. These results are reflected in Table 9 (see page 136). Kjaer (1979), reported a similar finding, except that he also found shorter relative latencies for peaks III-V and I-V. He attributed his finding to males being taller by 13cm when compared to females in this study.

The finding of this study suggests that females have a shorter peripheral transmission time than males over the distance extending from the onset of the click to the ponto-medullary junction in the lower pons. However, central transmission time extending from caudal pons to the midbrain, i.e. interpeak latency of peaks I-V did not reach significance, despite males tending to have the longer latency value of 3.91 msec compared to 3.84 msec in females (F = 2.74, df = 1:59; p = 0.09).

It would appear from the finding of this study that peripheral conduction time as determined by middle ear function, cochlea mechanics, cochlea transduction, synaptic and cochlea nerve conduction velocity (Cornacchia et al. 1983), are different in both sexes. It is quite conceivable that one or more of the above physiological mechanisms in the auditory periphery may act alone or in combination to facilitate transmission time, more so in females than in males. In addition, it would appear that Stockard et al.'s (1978) postulation, of shorter anatomical distance between synaptic junction in females may also account for the observed sex difference effect. Extending this postulate, it would appear that females have a shorter distance between the outer ear and the ponto-medullary junction than their male counterparts, thereby accounting for the shorter relative latency for peaks I-III.

However, the writer is unable to invoke appropriate measures to account for the latter, but maintains that one of several mechanisms may be operational in accounting for the sex difference effect, including; the possibility of favourable neural substrate activity; higher metabolism, and a more efficient cochlea nerve transmission (as discussed earlier) in females than in males. The exact mechanism responsible for the sex difference effect remains unclear and open to further question and research.

The lack of a significant sex difference effect on the relative latency measures of peaks III-V and I-V implies that the central brainstem transmission time, as determined by the fibre conduction velocity and synaptic transmission within the brainstem tracts and nuclei (Cornacchia et al. 1983), are essentially the same for both sexes.

However, males did show a slightly longer interpeak latency value for peaks I-V which are in agreement with the findings of Kjaer (1979), and Stockard et al., (1978). Therefore, there appears to be consensus that the interpeak latencies between males and females are different, though not always statistically significant.

In view of the above, it is worthwhile and acceptable to establish separate interpeak latency reference data for males and females.

6.3.3 SEX DIFFERENCE EFFECT : AMPLITUDE MEASURES

The amplitude measures conducted in this study viz.,

a) the absolute amplitude of peak V and

b) the relative amplitude (RA) of peak V compared to 1, showed no significant ($p>0,05$) sex difference effect (See Table 12, page 139).

Three studies viz: those of Kjaer (1979); Jerger & Hall (1980), and Michalewaski (1980), have all reported larger amplitude values for peak V in females than in males. Jerger and Hall (1980), found that on average peak V amplitudes was 0.08µV larger in females than in males (no mention is made as to whether this was statistically significant or not). Michalewaski found that female peak V amplitudes were 0.29μV larger than in males (no mention is made as to whether this was statistically significant or not) Kjaer (1979), found that the amplitude of peak V was 0.12μV larger in females, and was reported as being statistically significant ($p < 0,0005$). In this study the difference was 0.03μV in favour of females having larger amplitudes. However this was not statistically significant ($p > 0,05$).

It is apparent that wide discrepancies exist in the studies which have reported on the actual magnitude of the mean differences in peak V amplitudes between the sexes. Kjaer (1979), states that there are wide individual variations in this measure among normal hearers, and that at best a gross estimate can only be given as to whether a recording has a normal or some degree of reduced amplitude. However, further research into the effective use of signal averaging and artifact rejection may aid in establishing realistic measures in amplitudes between the sexes (see pages 155-157 for relevant discussion). Following the availability of such data, one can clearly establish the effect of sex difference on amplitude measures.

Here again, discrepancies between and among test facilities are exemplified, and the need for individual clinics to establish there own diagnostic reference data is evident.

In considering the relative amplitude measure, the selected literature reviewed shows a lack of studies reporting of the sex difference effect on this measure. In the present study, there were no significant sex difference effects on the relative amplitude measure, (F = 0.27, df = 1:59; p = > 0.60). However, in view of the controversy that exists over what value should be normal and as to what constitutes a measure of abnormality the investigator suggests that the reference data that are generated should be applied widely in pathological populations to assess their ability to differentiate normal from abnormal results.

SUMMARY : Sex Difference Effect.

The overall findings of this study are consistent with several investigations that have addressed the question of whether there is a sex difference effect on the normal BAER or not. There is general consensus among the following investigators Stockard et al., (1978); Beagley & Sheldrake, (1978); Kjaer, (1979); McClelland & McCrae (1979); Jacobson et al., (1980); Jerger & Hall, (1980); Michalewaski et al., (1980); Hall (1984), and Jerger & Johnson (1988), that there is a definite sex difference effect on the normal BAER. The above investigators have reported either shorter absolute and relative latencies and/or larger amplitude values amongst females when compared to their male counterparts.

This study revealed that the absolute latencies of peaks I; III and V and the relative latency of peaks I-III were significantly shorter in females than in males. There were no significant effects observed on the amplitude measures conducted. Further research requirements in this respect have been suggested.

There were discrepancies between this study and among other studies that reported on the sex difference effect in terms of the different BAER characteristics studied. This highlights the need for each test facility to establish their own sex related diagnostic reference data.

In accounting for the overall sex difference effect, there were no clear and definite explanations given by previous researches. However, several authors including the present investigator have put forward speculative arguments in an attempt to explain and to support the general finding for an overall sex effect on the normal BAER.

A summary of each investigators explanation for the observed sex difference effect, in which females tended to have shorter latency and or larger amplitude values than males, is given below.

1. STOCKARD ET AL. (1978), attribute the sex difference effect to shorter anatomical distances in the corresponding segments of the auditory pathway, in view of smaller average head size and brain of females. No further elaboration is given.
2. BEAGLEY & SHELDRAKE, (1978); JACOBSON ET AL., (1980); McCLELLAND & McCRAE (1979), simply state that they concur with Stockard et al.'s suggestion.

3. KJÆR (1979), attributes their findings to the differences in body length, whereby males were about 13 cm taller than females in their study. No further elaboration is given.

4. JERGER AND HALL (1980), concur with Stockard et al.'s suggestions and state that "due to relatively smaller dimensions of the female CNS, neural transmission time of the ABR is reduced". They suggest that the actual basis for the conspicuous sex difference deserves further investigation.

5. MICHALEWASKI ET AL. (1980), also concurred with Stockard et al. (1978), in stating that "differences in the anatomical distance of the structures along successive stages of the auditory pathway may contribute to the latency difference between the sexes." No elaboration in made.

6. GASTONE ET AL. (1987), state that male-female differences in the normal BAER are attributed to different body and brain size. No futher information is given.

7. HALL (1984), speculates on such variables as head size, body temperature and hormonal status as contributing to the observed sex difference.

8. JERGER AND JOHNSON (1988), tend to agree with Hall and suggest as possible bases for the overall sex effect, to be related to differences in head and brain size, difference in whole body temperature and differences in the hormonal milieu.

The latter two researchers have mentioned the possible influences of body temperature and hormonal factors. Correlates of these were not evoked in this study, but these two factors, deserve further research.

The present investigator has added to the above speculative arguments by focusing on the possible influence of; differences in brain size & mass as affecting volume conduction; differences in neural transmitter substrates; differences in metabolic rates, and allied saltatory conduction times of impluses between the sexes as contributing to the overall sex difference effect. Other factors may be responsible, but it is clear that further consideration and research is required in order to discover the basis for the sex difference effect.

Despite the lack, at this stage, of a conclusive argument for the sex difference effect on the normal BAER, it has been confirmed by this study, that such an effect exists. Whatever, the explanation for the sex difference effect, it is recommended that separate diagnostic reference data for males and females be established in each clinic or test facility. Application of female data to interpret male BAERs will result in false positives, while the application of male data to female patients will result in false negatives. The above contention parallels Stockard et al.'s (1978), and Michalewaski et al.'s (1980), recommendation to generate separate reference data for both sexes so as to avoid the potential type I error in diagnostic work.

6.4 HYPOTHESIS III: There is a significant difference between the BAER recorded from the target ear in the presence of contralateral masking, and that recorded in the absence of contralateral masking among a group of Indian university students with respect to certain characteristic of the monaural BAER.

The findings of this study do not support the above hypothesis. The use of three levels (50, 60 and 70dBHL) of contralateral broadband masking introduced to the non-test left ear had no significant effect ($F=0.51$; $DF=1;59$; $p > 0.05$) on the various characteristics of the monaural BAER elicited on the test (right) ear presented with clicks at 70dBnHL. The latency and amplitude (absolute and relative) measures considered in the study showed NO significant changes between the non-masked and the three masked conditions. Table 13 (see page 141) revealed a consistent agreement among the four named statistics, that there were NO overall masking effects on the monaural BAER obtained from 60 normal hearing Indian university students.

The results of this investigation, therefore support those obtained by previous investigators, viz. Chiappa et al., (1979); Humes & Ochs (1982), and Reid & Thornton, (1983). It is to be noted that this investigation was similar to the above three studies in that the masking stimulus used was steady broadband noise introduced at levels of 70dBHL and below. In comparison, Roshenhamer & Holmkvist, (1983); Reid et al. (1984), and Prasher & Cohen (1984), reported significant changes in the BAER under masked conditions at levels greater than 75dBsl. Furthermore, the type of masking noise used differed from the present study. Rosenhamer and Holmkvist used white noise; Cohen used white pulsed noise while Reid et al. (1984), do not describe the masking noise used.

It is apparent that the type and intensity of masking noise plays a role in determining whether or not contralateral masking has an effect on the monaural BAER. In this respect, it is evident that continuous broadband noise below 70dBHL does not significantly effect the monaural BAER, whereas the use of high intensity (> 75dBsl) of white noise or pulsed white noise and high pass filtered noise does have an effect on the monaural BAER. An explanation for the effect of the latter variables cannot be evoked as these were not studied in this investigation.

However, in considering the findings of this investigation of no overall masking effect, the writer wishes to consider Rosenhamer and Holmkvist's (1983) argument, "...that at least, theoretically masking noise at sufficiently high levels may effect the monaurally elicited brain stem reponse via four possible mechanisms:

a. by eliciting the stapedial reflex
b. by air-conducted crossover

c. by bone-conducted crossover and

d. by central masking".

The writer, drawing form relevant research findings, will discuss how the above mechanisms may in fact be operational in controlling rather than contributing to contralateral masking effects at intensity levels of 50, 60 and 70dBHL from influencing the monaural click evoked BAER. It is the writer's opinion that one or more of the mechanisms to be discussed below may be responsible for the findings of this study.

6.4.1 Mechanism via the acoustic stapedial reflex.

It has been well established by various researchers (Jepsen, 1951; Deutch, 1972; Jerger et al., 1972; and Borg & Counter 1989), that the acoustic reflex is bilaterally elicited by sufficiently loud sounds. It has been demonstrated that the reflex is evoked at between 70 and 90dBsl in normals for pure tones (Petersen & Linden 1972, and Jerger et al. 1972), and at 10-12dB's lower for broadband noise (Flottorp et al., 1971). The contraction of the stapedius muscle stiffens the middle ear system which in turn reduces sound transmission to the inner ear by 20 dB or more (Borg and Counter, 1989). The effect of this action protects the inner ear receptor cells from sustained loud noises that might otherwise cause hearing loss (Borg and Counter, 1989).

It is, therefore, logical to assume that in presenting contralateral broadband masking at 50, 60 and 70dBHL, the stapedius muscle will contract bilaterally. This would stiffen the middle ear system thereby attenuating the level of the click and causing the evoked BAER to be compromised. This compromise may appear in the form of delayed latency or reduced amplitude measures.

Research by Mϕller (1972), and Borg & Counter (1989), suggest that the attenuation caused by the contraction of the stapedius muscle is more selective than previously thought. Borg and Counter (1989), state that the contraction of the stapedius muscle stiffens the middle-ear system thereby attenuating the low frequency components of complex sounds, more than it does the high frequency components. This supports Mϕller's (1972), contention that the attenuation caused by the contraction is rather small (< 5dB) in the high frequency region.

Consequently, and noting that the click used to elicit the BAER is high frequency in composition, the contraction of the stapedius muscle using intensity levels of 50, 60 and 70dBHL of masking noise should have little or no attenuation effect on the transmission of the click stimuli to the inner ear.

There is also the contention that the click level itself, if sufficiently loud would elicit the stapedial reflex and thereby attenuate the intensity of the clicks, the effect of which may compromise the latencies and amplitudes of the BAER. Rosenhamer and Holmkqvist (1983), found that clicks presented at 70dBHL produced a small-amplitude contraction decaying to zero well before the end of the epoch, and concluded that such a contraction was inadequate to attenuate the click level. Therefore, the click elicited stapedial reflex would not compromise the BAER to any significant way. The writer concurs with the above in view of the fact that the click level in this study was presented at 70dBnHL.

Thus, the above arguments support the finding of this investigation that the click elicited monaural BAER at 70dBnHL does not show an overall significant change both in the absence and presence of 50, 60 and 70dBHL of contralateral broadband masking.

6.4.2 Mechanism of crossover of broadband masking by air conduction.

Reid et al. (1984), and Rosenhamer & Holmkvist (1983), suggest that contralateral masking, if sufficiently loud will cross over into the click stimulated ear and thereby influence the obtained BAER in that ear.

However, it has been documented by Studebaker (1973), that the interaural attenuation for broadband noise using closely fitted electromagnetically, shielded earphones is approximately, 50dB. Thus, it is reasonable to assume that in using 50, 60 and 70dBHL of contralateral masking, only ± 0, 10 and 20dB of broadband noise would have crossed over to the click stimulated ear in this study.

Furthermore, it is well established that during the process of crossover, high frequency components of the masking noise are more attenuated than low frequency noise (Studebaker, 1973). This would imply that a lesser amount of high frequency noise would have crossed over to the click stimulated ear, and thereby have little or no influence on the high frequency click evoked monaural BAER. This argument would account for a lack of a significant effect of contralateral masking at 50, 60 and 70dBHL in the monaural BAER as evidenced in this study.

6.4.3 Air-to-bone mechanism of crossover of contralateral broadband masking noise.

The contention of acoustic cross-talk via bone conduction resulting in the stimulation of the test cochlear directly and influencing the evoked BAER is inherent in Rosenhamer & Holmvist (1983), and Reid et al.'s (1984), argument. They suggest that contralateral masking noise will crossover via bone conduction and produce a cochlear masking effect, and hence reduce the test cochlear's ability to process and to transmit the click stimuli.

However, it has been shown by Thummler et al. (1981), that the air to bone attenuation of broadband noise is at least 60 to 70dB. Therefore, this route would only produce no more than 0-10dB of cochlear masking in the click stimulated ear. This amount is hardly sufficient to cause any observable change in the BAER in the test ear. The above contention is supported by Thummler et al.'s

(1981), findings.

It is reasonable to assume that the cross via air to bone conduction would not affect the monaurally elicited BAER at intensity levels of 50, 60 and 70dBHL. This contention is supportive of the finding in this investigation.

In addition to the arguments given in 6.4.2 and 6.4.3 above, another mechanism inherent in the signal averaging process may account for the lack of a significant effect of contralateral masking on the monaural BAER. In using an "averaged response computer", one is able to accumulate and extract desired responses (Gibson and Ruben, 1978). The computer operates on the principle of algebraic summation of bio-electric events elicited by stimulus synchronisation (time lock repetition). In other words, the evoked potential is predicated on event related stimuli, thereby assuring a constant time relationship to the response of the signal. In contrast, unwanted noise is random. In this way, the unwanted noise has no time relationship to stimulus onset, and thus the evoked potential is elicited without the confounding effects of unwanted noise.

Bearing this in mind, it is reasonable to assume that contralateral masking noise in the non-test ear, although crosses over by exceeding the interaural attenuation values, will be out of phase with the click stimuli presented to the test ear. In effect then, the crossed over masking noise, whether via air or bone conduction, will be averaged out by the computer. It is, therefore, plausible to use the above argument to support the finding that contralateral masking at the peripheral level, has no overall effect on the monaural BAER.

6.4.4 Mechanism of central masking.

The mechanism of central masking is presumed to lie in the high pathways of the central auditory system where "neural interaction" between ears may cause a threshold shift of the test ear due to masking noise introduced in the non-test ear (Hodgson, 1980). Several authors have attributed shifts in pure tone thresholds in the presence of contralateral masking of up to 15dB (Hodgson, 1980). In view of this observed phenomenon, Rosenhammer and Holmkvist (1983), postulated that contralateral masking may reduce click evoked neural input and break up it's synchronisation, and also reduce signal to noise ratio in the central auditory pathway by adding to the back ground spontaneous activity. This postulate may be viable if it can be clearly shown that both the click induced impulses and the masking induced impulses travel along a single auditory pathway in the brainstem. In this way it would be plausible to expect some neural interaction to occur and cause central masking to occur, thereby compromising the monaural BAER.

However, evidence exists to suggest that there is a different mechanism and pathway within the auditory system which maybe responsible for the "handling" of short duration sounds (clicks for example) and longer duration sound (continuous masking) separately (Gersuni, 1971). He postualted that if click stimuli produce neural activity within the "onset responding" short duration part of the auditory pathway and continuous broadband masking causes neurons in the long duration part of the auditory pathway to fire, then there would be no "neural interaction" and, therefore, no central masking effect. Reid et al. (1983), have adopted this hypothesis to explain the lack of contralateral masking effects on the monaural BAER in their study.

The above contention is further supported by Evan's suggestion in 1974, "that the auditory system divides into two subsystems that can be differentiated anatomically and functionally, at the brainstem level at least, and maybe related to the processing of localisation and pattern formation, respectively". Furthermore, the findings of Stephens (1973; 1975), supports Gersuni's hypothesis of a two mechanism auditory system. Wynn (1977), produced evidence which suggests the presence of two pathways conveying auditory information to the brain. He proposed that a stochastic mechanism may be responsible for channelling this information into either a slow or fast pathway. A number of physiological studies (e.g. Gersuni, 1971) have shown that a dual system of neural units, which respond either to onset of a stimulus or to it's sustained presence exists in the lower brainstem.

Lending credence to the above, recent research findings by Musiek & Baran (1986), and Caspary (1986), as cited by Musiek (1989), have identified different cell types in the cochlear nucleus of man. These cells yield different discharge response patterns as seen in post-stimulatory histograms. Musiek (1989), states that these cells function either to modify or to preserve incoming impulses of the auditory nerve in a predictable manner. Post-stimulus histograms reveal that one of the cell types viz. the "stellate" cells yield a repetitive "on off" response and another cell type, the "bushy" cell preserves the auditory nerves firing pattern termed "primary like" (Kiang, 1975).

In view of Gersuni's hypothesis of a dual system of auditory units, and in the light of Kiang's (1975), description of specific cell function, as reported by Musiek (1989), it is conceivable that click stimuli used in BAER testing is "handled" by the "stellate" cells while continuous contralateral masking is processed by the "bushy" cells. This would then probably give rise to different functional pathways handling auditory information selectively and separately. The writer contends, that it is perhaps for the above postulation, that contralateral masking has <u>NO</u> significant effect on the monaural BAER, as evidenced in this investigation.

It is conceded that, it is very difficult to make accurate inferences at this stage about underlying neural and neuronal events occurring in the auditory system. However, research focussing on the use of microelectrodes, neurochemical analysis and electron microscopic studies of the auditory system in response to acoustic stimulation may reveal a clearer picture of the auditory physiology involved. This contention is supported by Jacobson et al., (1985).

SUMMARY : Effects of contralateral masking on the monaural BAER.

In the foregoing discussion, it is evident that the finding of this investigation do not support hypothesis III as stated in Chapter 1. The investigator has put forward plausible explanations and arguments for rejecting the hypothesis. It must be borne in mind that these explanations and arguments have been derived from existing research finding and speculations regarding the physiology of the auditory system. Undoubtedly, this system is complex in both it's structure and function (Musiek 1989), and it is accepted that the explanations given may be open to question and further research.

However, the writer maintains, that no matter what the explanation for the lack of a significant effect of contralateral broadband masking on the monaural BAER maybe, the present finding has important clinical implications.

It is evident, that contralateral broadband masking noise presented at levels of 50, 60 and 70dBHL will not significantly affect the monaural click evoked BAER. Therefore, clinicians should feel safe to use these levels of contralateral broadband masking in the audiological clinic at the University of Durban-Westville in patients, at least for the age range 18-25 years. This would lead to the elicitation and interpretation of "true" monaural BAERs especially in patients who have unilateral sensori-neural hearing losses.

6.5 HYPOTHESIS IV: There is a significant difference in the clinical BAER recorded from the target ear in both the presence and absence of contralateral masking, between male and female Indian undergraduate students with respect to the following characteristics; the:

a) absolute latencies of peaks I to V.

b) relative latencies of peaks I-III; III-V and I-V.

c) absolute amplitude of peak V.

d) the amplitude ratio of peak V compared to peak I.

The finding of this study does not support the above hypothesis. A comparison of the normal click elicited BAER at 70dBnHL in the absence and presence of three levels (50, 60 and 70dBHL) of contralateral broadband masking in 30 female and 30 male subjects did not reveal any significant interactional effects. This implies that both the stimulus related variable (contralateral masking) and the subject related variable (sex difference) did not show significant interactional effects on the normal characteristics of the monaural BAER.

Table 14 (see page 143) reveals a consistent agreement among the four statistics, that there were NO overall sex and masking interactional effects on the characteristics of the normal monaural BAER studied.A thorough review of selected literature did not yield information regarding the interactional effects of sex difference and contralateral masking on the normal monaurally elicited BAER. Therefore, the finding of this study adds new information to the body of literature relating to the non-pathological factors that influence the normal BAER.

The lack of a significant interactional effect (sex difference and contralateral masking) on the BAER implies that the manner in which the auditory system processes click stimuli in the presence of three levels of contralateral masking (50, 60, and 70dBHL) is the same in both males and females.

The explanation for the lack of a significant interactional effect, may be gleaned from the earlier arguments made (see page 182-189), whereby the present investigator attempted to account for the rejection of hypothesis III of this study.

It is, therefore, suggested that the manner in which the acoustic reflex, the mechanisms of crossover by air-conduction and of air-to-bone conduction, the implications of effective computer averaging and the mechanism of central masking contributes toward counteracting the possible effects of contralateral broadband masking on the monaural BAER, is the same for both males and females.

The clinical significance of the demonstrated absence of an interaction (sex difference and contralateral broadband masking) effect on the monaural BAER, is that clinicians may safely use contralateral broadband masking at levels of 50, 60 and 70dBHL without significantly compromising the monaural click evoked BAER at 70dBnHL in both males and females in the age range 18-25 years.

Furthermore, the study incidentally revealed that broadband masking noise used at 50, 60 and 70dBHL may be more appropriate in masking the non-test ear than using either continuous white noise used by Rosenhamer and Holmkvist, (1983), pulsed white noise used by Prasher & Cohen, (1984), or high-pass filtered noise used by Klien (1986), at intensity levels greater than 75dBsl.

CHAPTER 7

SUMMARY AND CONCLUSIONS

The objectives of this investigation were to establish diagnostic reference data; to study and report on the effects of sex difference and contralateral masking on the normal BAER. BAERs were elicited from the target (R) ear using clicks presented at 70dBnHL, both in the absence and presence of three (50, 60 and 70dBHL) levels of contralateral broadband masking noise. Relevant latency and amplitude data were obtained from 60 selected normal hearing Indian undergraduate female (N=30; $\bar{x}$ age = 20.33 years) and male (N=30; $\bar{x}$ age = 21.33 years) students aged between 18 and 25 years ($\bar{x}$ age = 20.73 years).

7.1 Diagnostic Reference Data

Diagnostic reference data were established for both the combined group (N=60) and separately for females and males. The obtained data relates to the following characteristics of the normal BAER; absolute latencies of peaks I to VI, relative latencies of peaks I-III; III-V and I-V, absolute amplitudes of peaks I and V, and the relative amplitude of peaks V:I. Similarities and differences between this study and of those reported in the literature were noted and discussed. The similarities in absolute latency measures were attributed to close approximations between testing protocols used, whilst variations were primarily related to, among other variables, differences in reference intensity levels and the polarity of clicks.

Despite the difference between this study and of those reflected in Table 2, (see page 39) the absolute latency of peak V remained resistant to variations in stimulus, recording and "normal subject" variables. Therefore, this measure appears to be robust and may be reliably used in otoneurological diagnosis and for estimating hearing sensitivity.

The relative latency values generated are consistent with those reported in the literature (See table 2), and this is attributed to the fact that click presentation rates used are similar, i.e. 10-12 clicks per sec., in each of the studies. It is, therefore, suggested that clinicians may confidently use these measures to assess otoneurological pathologies that may upset the conduction of impulses in the auditory periphery (e.g. multiple sclerosis), provided that the click rate used is 10 to 12per sec.

Differences in amplitude measures between this study and among other studies were noted. These variations were, among other factors, attributed to the manner in which signal averaging and artifact rejection have been manipulated in obtaining the averaged BAER. Further research in this respect has been suggested. However, the relative amplitude value of 1.50 obtained in this study is consistent with those reported in the literature. This implies that the RA measure is less variable in normals and therefore, may be used as a more sensitive measure of brainstem auditory function than absolute amplitude measures.

In view of the demonstrated differences in reference data between and among clinics and laboratories, the writer is of the opinion that clinicians should exercise caution in using reference data established elsewhere, especially if reported testing protocols differ in stimulation, recording and normal subject variables e.g. age. Therefore, it is recommended that each clinic generates it's own reference data commensurate with it's needs. Furthermore, noting that this study fell short of giving due consideration to age - related data across the continuum, inter-aural latency differences, use of different repetition rates, stimulus intensity reference levels and click polarity, future research considering the above, needs to be conducted to extend the present reference data base.

The need for consensus to be reached among researchers and clinician with respect to test protocols used in BAER testing cannot be overemphasized. Perhaps, an international conference involving the various disciplines that use this test procedure should be held, in order to formulate a standard guideline or protocol for the use and interpretation of the BAER. This would facilitate inter-clinic and/or laboratory comparisons, and perhaps aid in resolving some of the controversies that exist in BAER testing and interpretation.

In the interim, it is important that researchers and clinicians clearly define the parameters of their test protocols in establishing reference data. In addition, such data should be applied within populations having known otoneurological pathologies to assess the extent to which the reference data is able to differentiate normal from pathological ears. The latter is also applicable to the reference data generated in this study.

7.2 The Sex Difference Effect

The results of this study revealed that there was and overall sex difference effect on the normal BAER (See Chapter 5 & 6 for statistical information and discussion, respectively). This finding is consistent with those reported by several researchers (see Chapter 6). Speculative arguments and explanations were offered for the observed sex difference effects. Suggestions for future research were also made. Despite the lack of a definitive explanation of the sex difference effect, the writer suggests that it has important clinical implications. These are, that;

i. it is acceptable and worthwhile to establish separate reference data for males and females
ii. each clinic or laboratory should generate it's own data in view of the confounding effects of other non-pathological factors mentioned earlier
iii. awareness by clinicians of the sex difference effect would prevent diagnostic errors which could arise when female norms are applied to interpret male BAERs and vice-versa.

7.3 Effects of Contralateral Broadband Masking on the Monaural BAER

This study reveals that the monaural click evoked BAER at 70dBnHL does not change significantly in the absence and presence of three (50, 60 and 70dBHL) levels of contralateral broadband masking noise. Speculative arguments and explanations relating to the acoustic reflex mechanism, crossover of masking noise via air and bone conduction, effective computer averaging and the central masking mechanism have been presented to support the finding of this investigation.

The writer suggests that no matter what explanations may arise for the above finding, it has important clinical implications. Clinicians may feel safe to use contralateral broadband masking at 50, 60 and 70dBHL in eliciting the monaural BAER at 70dBnHL. This is, at least applicable for the Audiology Clinic at the University of Durban-Westville, particularly in patients in the age range 18-25 years. This would lead to the elicitation and interpretation of "true" monaural BAERs, especially in patients who have unilateral sensorineural hearing losses.

7.4 Interactional Effects of Sex Difference and Contralateral Broadband Masking

A comparison of the normal click evoked BAER at 70dBnHL in the absence and presence of three (50, 60 and 70dBHL) levels of contralateral broadband masking in the subjects did not reveal any significant interactional effects. This finding contributes to the body of literature on non-pathological factors that influence the normal BAER, since a perusal of selected literature did not yield information regarding the interactional effects of sex difference and contralateral masking on the normal BAER.

The clinical significance of this finding is that clinicians may safely use such masking noise at the stated intensity levels without significantly compromising the click evoked BAER at 70dBnHL. This may be applicable to males and females in the age range 18-25 years in the Audiological Clinic at the University of Durban-Westville.

Furthermore, broadband masking noise at the stated intensity levels may be more appropriate in masking the non-test ear than using white noise, pulsed white noise or high pass filtered noise presented at high intensity levels (> 75dBsl). Further research relative to these types of noises and their effects on the monaural BAER may contribute information to the literature dealing with non-pathological factors that affect the normal BAER.

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APPENDIX A

PRE-TEST CASE HISTORY INTERVIEW QUESTIONNAIRE

Dear Student

A study, aimed at surveying the auditory (hearing) skills of university students is to be conducted. Kindly indicate with a cross [X] whether you are willing to participate in such a project or not. Please remember that all information including test results will be treated in strict confidence by the researcher. Your consent to participate in this project will be greatly appreciated.

☐ - I am willing to participate in this project.

☐ - I am not willing to participate in this project because :

....

....

If you are willing to participate in this project please complete the following questionnaire carefully.

INSTRUCTION

1. Where applicable, you must answer questions by marking the appropriate block with an X.
2. There are THREE sections, (A, B, & C) to this questionnaire, please answer all the questions contained in each section.
3. Y = YES

   N = NO

appendix A cont./...

SECTION A

BIOGRAPHICAL DATA

1. SURNAME : ....

2. FIRST NAMES: ....

3. DATE OF BIRTH: ....

4. AGE: .... YEARS: .... MONTHS: ....

5. SEX : ☐ M ☐ F

6. DEGREE/DIPLOMA: ....

7. YEAR OF STUDY AT UNIVERSITY

1st	2nd	3rd	4th	5th

8. RESIDENTIAL ADDRESS WHILST AT UNIVERSITY..................

....

9. TELEPHONE NO. WHILST AT UNIVERSITY........................

SECTION B

GENERAL MEDICAL AND NEUROLOGICAL HISTORY

1. Do you or have you suffered from a medical problem?

☐ Y ☐ N

If yes, state the nature and duration of the problem or condition.

....

2. Do you or have you suffered from a neurological problem?

☐ Y ☐ N

If yes, state the nature and duration of the problem or condition.

....

appendix A cont./...

3. Have you or are you receiving treatment for any of the above?

Y	N

If yes, state the nature of the treatment.

....

4. If you are taking or have taken any form of medication, state:

a. Name of the drug as you know it;

....

b. Dosage taken :

5mg	5ml	or	10mg	10ml	or	>10mg	>10ml

c. Frequency of consumption:

1	2	3	4	>4	times a day

d. Duration of treatment :

<1	2	3	>3	months

5. Have you been hospitalized or have you been treated for a prolonged period for any medical, surgical or neurological condition?

Y	N

If yes, please describe.

....

....

6. Do you suffer from headaches?

Y	N

If yes, answer the following;

a. Type

MIGRAINE	TENSION	DON'T KNOW	OTHER

(if other, state ....)

b. Onset

MORNING	MIDDAY	AFTERNOON	NIGHT

c. Duration

< 1 HOUR	> 1 HOUR

d. Frequency

1	2	3	4

times a day or is it CONTINUOUS

e. Severity

MILD	MODERATE	SEVERE	VERY SEVERE

appendix A cont./...

7. Do you feel dizzy? Y | N

If yes, answer the following;

a. Onset

MORNING	MIDDAY	AFTERNOON	NIGHT

b. Duration

< 1 MIN	> 1 MIN

c. Nature : Do you feel any sensation in which you tend to fall to the

RIGHT	LEFT	FORWARD	BACKWARD

d. Do you feel that objects around you tend to spin? Y | N

e. Do you feel that you are spinning inside ? Y | N

f. Frequency:

1	2	3	4

times a day or is dizziness a CONTINUOUS feeling?

g. Severity:

MILD	MODERATE	SEVERE	VERY SEVERE

C. AUDIOLOGICAL (HEARING) HISTORY

1. Do you have any problem hearing? Y | N

If yes, please describe your difficulty/ies

....

....

2. Did you have any pain in your ear/s recently? Y | N

What diagnosis was given to you for this if you visited a doctor?

....

3. Did you have any discharge coming from your ear/s recently? Y | N

appendix A cont./...

4. Did you suffer from or are suffering from any other ear, nose or throat problem? | Y | N |

5. Does anyone in your family have a hearing problem? | Y | N |

6. Do you hear any unusual sounds or noises in your ear/s? | Y | N |

If yes, please answer the following;

....

....

a. ear: | LEFT | RIGHT | BOTH |

b. nature: | HIGH PITCHED | LOW PITCHED | RINGING | PULSATILE |

| ROARING |

c. Onset : | MORNING | MIDDAY | AFTERNOON | NIGHT |

d. Frequency: | INTERMITTENT | CONTINUOUS |

e. Severity: | SOFT | LOUD | VERY LOUD |

f. Does this worry you? | Y | N |

7. Do you live near or in a noisy area? | Y | N |

If yes, state the source and type of noise that you are exposed to.

....

....

8. Do you practise any hobbies which involve exposure to very loud noise? | Y | N |

If yes, please describe

....

....

appendix A cont./...

9. Did you injure your head and/or ear/s at any time? Y N

If yes, please describe

....

....

10. Are you oversensitive to loud noises i.e. do they annoy, irritate or cause you any discomfort? Y N

If yes, please elaborate

....

....

....

I wish to inform you that the data supplied will be screened and selected individuals will be required to undergo a complete audiological (hearing) evaluation. As you have already indicated your willingness to participate in such a programme, I will be contacting you at the given address/telephone number.

<u>THANK YOU FOR YOUR TIME AND INTEREST IN COMPLETING THE ABOVE QUESTIONNAIRE</u>.

C.D. GOVENDER
LECTURER/AUDIOLOGIST (DEPT. OF SPEECH AND HEARING THERAPY)
ENQUIRIES TEL. 820 2140 OR EXT. 2140

APPENDIX B

UNIVERSITY OF DURBAN-WESTVILLE
DEPARTMENT OF SPEECH AND HEARING THERAPY

CID SPONDEES

Airplane
Sunset
Armchair
Duckpond
Toothbrush
Eardrum
Greyhound
Schoolboy
Mousetrap
Northwest
Iceberg
Horseshoe
Farewell
Grandson
Pancake
Railroad
Playground
Hardware
Mousetrap
Oatmeal
Hotdog
Hothouse

Greyhound
Schoolboy
Inkwell
Whitewash
pancake
mousetrap
eardrum
Headlight
birthday
duckpond
sidewalk
hotdog
padlock
mushroom
hardware
workshop
horseshoe
armchair
baseball
stairway
coyboy
iceberg

northwest
railroad
playground
airplane
woodwork
oatmeal
toothbrush
farewell
grandson
drawbridge
doormat
hothouse
daybreak
sunset
workshop
schoolboy
padlock
railroad
northwest
armchair
eardrum
headlight

EAR:	P T A	S R T
RIGHT:		
LEFT:		

APPENDIX C

Adapted from Egan, (1948).

W-22 WORD LISTS

PB-50—LIST 1

1. ace
2. ache
3. an
4. as
5. bathe
6. bells
7. carve
8. chew
9. could
10. dad
11. day
12. deaf
13. earn (urn)
14. east
15. felt
16. give
17. high
18. him
19. hunt
20. isle (aisle)
21. it
22. jam
23. knees
24. law
25. low
26. me
27. mew
28. none (nun)
29. not (knot)
30. or (oar)
31. owl
32. poor
33. ran
34. see (sea)
35. she
36. skin
37. stove
38. them
39. there (their)
40. thing
41. toe
42. true
43. twins
44. yard
45. up
46. us
47. wet
48. what
49. wire
50. you (ewe)

PB-50—LIST 2

1. ail (ale)
2. air (heir)
3. and
4. bin (been)
5. by (buy)
6. cap
7. cars
8. chest
9. die (dye)
10. does
11. dumb
12. ease
13. eat
14. else
15. flat
16. gave
17. ham
18. hit
19. hurt
20. ice
21. ill
22. jaw
23. key
24. knee
25. live (verb)
26. move
27. new (knew)
28. now
29. oak
30. odd
31. off
32. one (won)
33. own
34. pew
35. rooms
36. send
37. show
38. smart
39. star
40. tare (tear)
41. that
42. then
43. thin
44. too (two, to)
45. tree
46. way (weigh)
47. well
48. with
49. yore (your)
50. young

PB-50—LIST 3

1. add (ad)
2. aim
3. are
4. ate (eight)
5. bill
6. book
7. camp
8. chair
9. cute
10. do
11. done (dun)
12. dull
13. ears
14. end
15. farm
16. glove
17. hand
18. have
19. he
20. if
21. is
22. jar
23. king
24. knit
25. lie (lye)
26. may
27. nest
28. no (know)
29. oil
30. on
31. out
32. owes
33. pie
34. raw
35. say
36. shove
37. smooth
38. start
39. tan
40. ten
41. this
42. though
43. three
44. tie
45. use (yews)
46. we
47. west
48. when
49. wool
50. year

PB-50—LIST 4

1. aid
2. all (awl)
3. am
4. arm
5. art
6. at
7. bee (be)
8. bread (bred)
9. can
10. chin
11. clothes
12. cook
13. darn
14. dolls
15. dust
16. ear
17. eyes (ayes)
18. few
19. go
20. hang
21. his
22. in (inn)
23. jump
24. leave
25. men
26. my
27. near
28. net
29. nuts
30. of
31. ought (aught)
32. our (hour)
33. pale (pail)
34. save
35. shoe
36. so (sew)
37. stiff
38. tea (tee)
39. than
40. they
41. through
42. tin
43. toy
44. where
45. who
46. why
47. will
48. wood (would)
49. yes
50. yet

PB-50—LIST 5

1. add
2. bake
3. bathe
4. beck
5. black
6. bronze
7. cheat
8. choose
9. curse
10. drive
11. feed
12. flap
13. good
14. Greek
15. grudge
16. high
17. hill
18. inch
19. kid
20. lend
21. love
22. mast
23. nose
24. odds
25. owls
26. pass
27. pipe
28. puff
29. punt
30. rear
31. rind
32. rode
33. roe
34. scare
35. shine
36. shove
37. shy
38. sick
39. solve
40. thick
41. thud
42. trade
43. true
44. tug
45. vase
46. watch
47. wink
48. wrath
49. yawn
50. zone

APPENDIX D

UNIVERSITY OF DURBAN-WESTVILLE

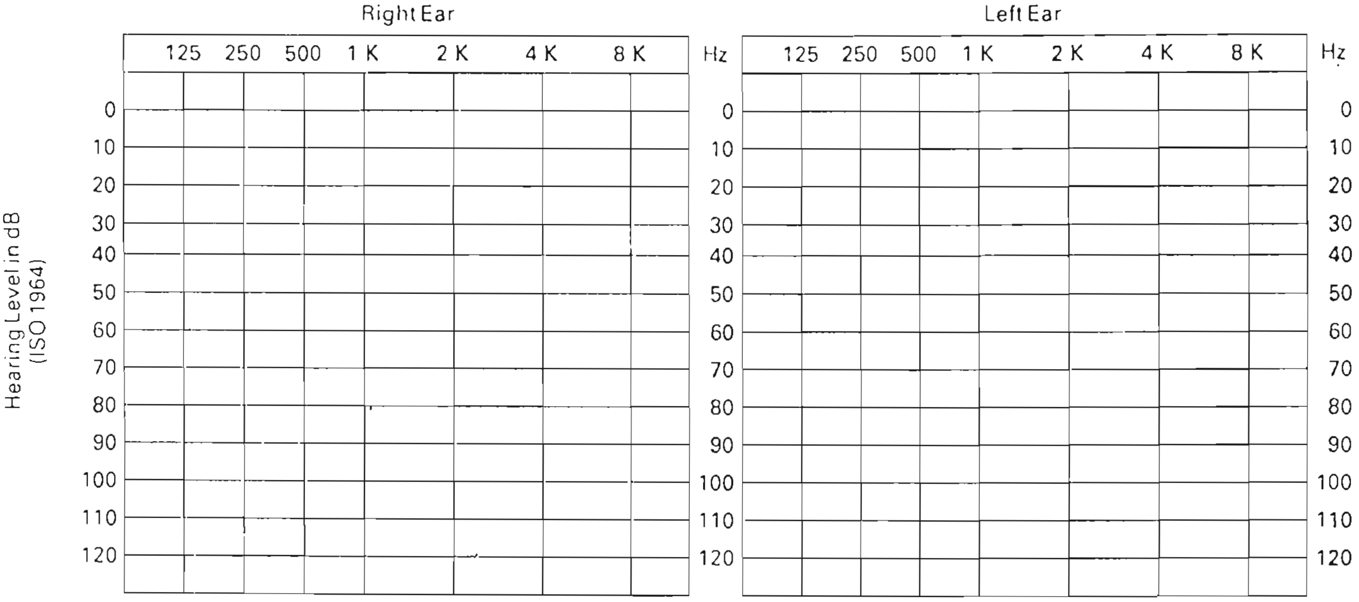
AUDIOMETRIC EXAMINATION

DEPARTMENT OF SPEECH AND HEARING THERAPY

NAME .... PATIENT NO. ....

AUDIOLOGIST .... DATE ....

PURE TONE AUDIOGRAM



NBN dB Masking								
	AC							
	BC							

AC							
BC							

AUDIOMETRIC WEBER

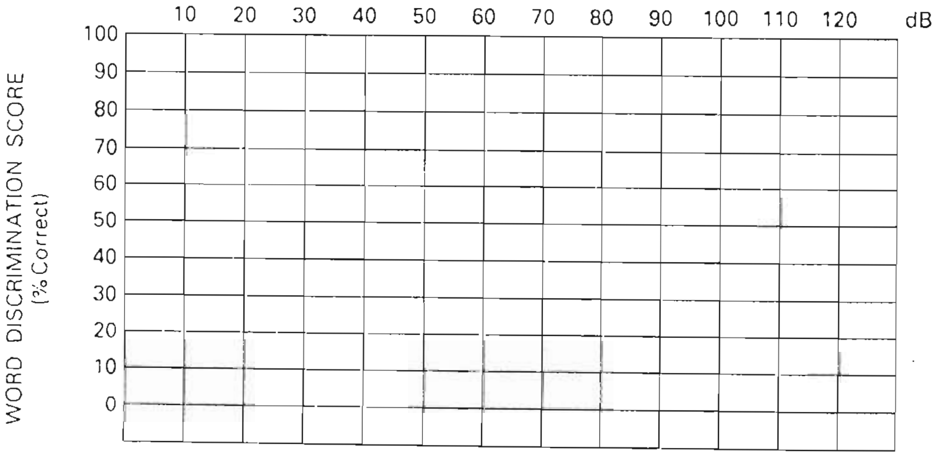
250	500	1 K	2 K	4 K	Hz

R–Right C–Central L–Left

TEST RELIABILITY

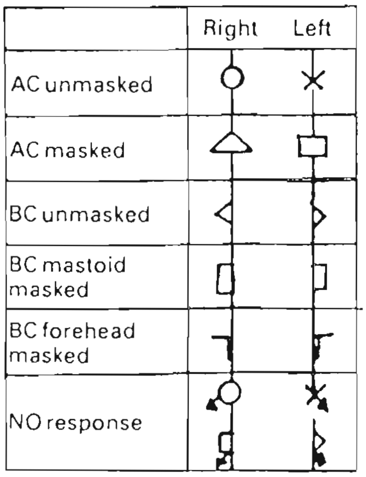
GOOD	FAIR	POOR

SPEECH AUDIOGRAM



WN MASKING													
													dB

AUDIOGRAM KEY



EAR	PT	SRT	% max disc	MCL	DL	Dynamic range
Right						
Left						

APPENDIX E

GSI Grason-Stadler

1723 Middle-Ear Analyzer

LEFT	RIGHT

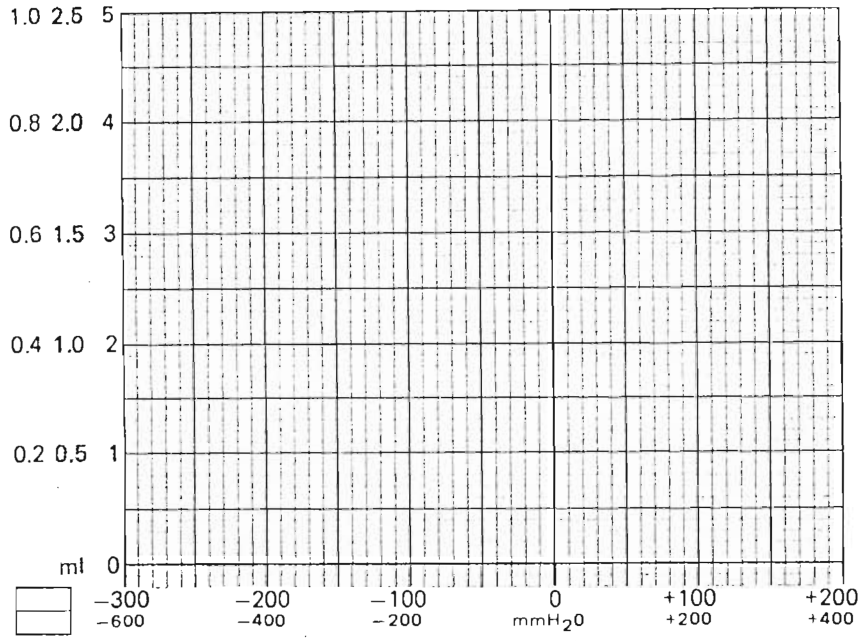
NAME NO. AGE

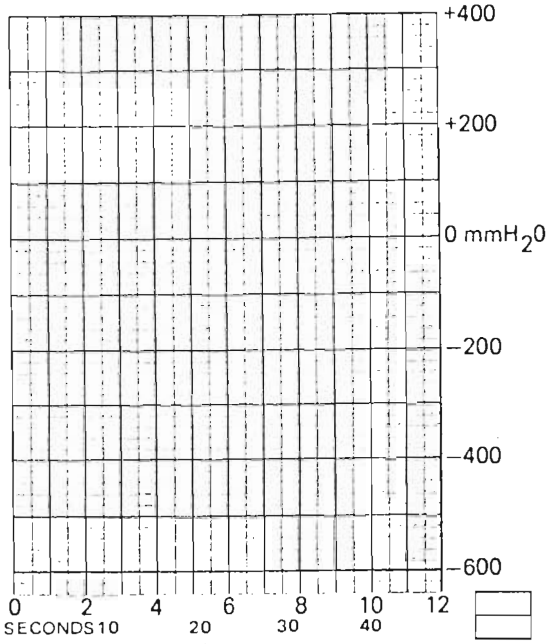
EXAMINER DATE TIP SIZE

SENSITIVITY*						FREQ.		RECORD SPEED	
1.0	2.5	5.0	Y	B	G	220	660	NORM.	SLOW

REFLEX SENSITIVITY	.5	1.0

250	500	1K	2K	4K	LB	HB	BB	STIMULUS
								IPSI HL
								CONTRA HL





*MULTIPLY SENSITIVITY
X 3 AT 660 Hz

TYMPANOGRAM

REFLEX EUSTACHIAN TUBE

CHART NO. 1723-9600

REV 5

GSI Grason-Stadler

1723 Middle-Ear Analyzer

LEFT	RIGHT

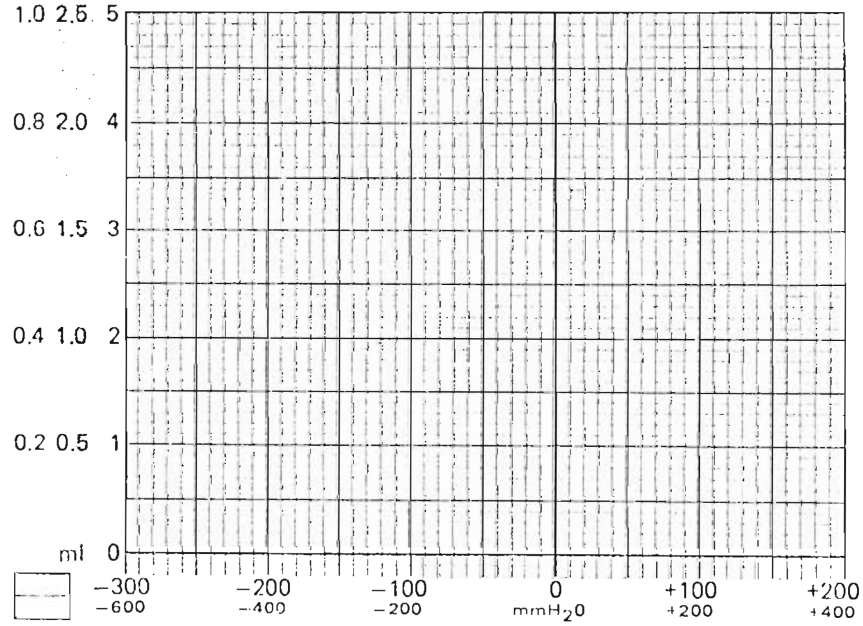
NAME NO. AGE

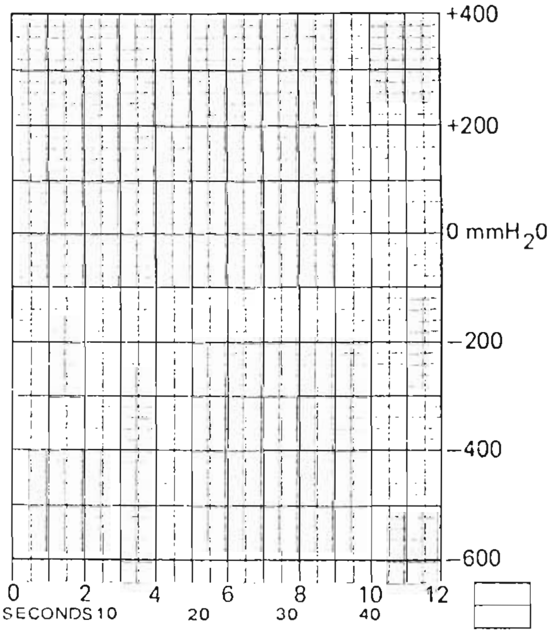
EXAMINER DATE TIP SIZE

SENSITIVITY*						FREQ.		RECORD SPEED	
1.0	2.5	5.0	Y	B	G	220	660	NORM.	SLOW

REFLEX SENSITIVITY	.5	1.0

250	500	1K	2K	4K	LB	HB	BB	STIMULUS
								IPSI HL
								CONTRA HL





*MULTIPLY SENSITIVITY
X 3 AT 660 Hz

TYMPANOGRAM

REFLEX EUSTACHIAN TUBE

CHART NO. 1723-9600

REV 5

APPENDIX F

HANDEDNESS INVENTORY (Annet's - Adapted & modified by Lazarus, 1983; 1985)

NAME :........................... SEX:........... AGE:.......

INDICATE HAND PREFERENCE	ALWAYS LEFT	USUALLY LEFT	NO PREFERENCE	USUALLY RIGHT	ALWAYS RIGHT
1. To write a letter legibly	....	....	....	....	....
2. To throw a ball to hit a target.	....	....	....	....	....
3. To play a game requiring the use of a racquet	....	....	....	....	....
4. At the top of a broom to sweep dust from the floor.	....	....	....	....	....
5. At the top of a spade to move sand	....	....	....	....	....
6. To hold a match when striking it.	....	....	....	....	....
7. To hold a scissors to cut paper.	....	....	....	....	....
8. To hold thread to guide through the	....	....	....	....	....
9. To deal playing cards	....	....	....	....	....
10. To hammer a nail into wood.	....	....	....	....	....
11. To hold a toothbrush while cleaning teeth.	....	....	....	....	....
12. To unscrew the lid of a jar.	....	....	....	....	....

13. Are either of your parents left handed? If yes, which ....

14. How many siblings do you have? Male:....... Female:.........

15. What is your birth order? First:.... Middle:.... Last:.......

16. Which eye do you use when using only one eye to look through a telescope?..................

17. Have you ever suffered any severe head trauma? ....

APPENDIX G

Raw BAER data for 60 subjects (30 males and 30 females).

A = Females and males alternately.
B = Contralateral masking conditions viz. 0, 50, 60 and 70dBHL.
C = Absolute latency of peak I in msec.
D = Absolute latency of peak II in msec.
E = Absolute latency of peak III in msec.
F = Absolute latency of peak IV in msec.
G = Absolute latency of peak V in msec.
H = Relative latency of peak I-III in msec.
I = Relative latency of peak III-V in msec.
J = Relative latency of peak I-V in msec.
K = Relative amplitude of peak I:V.
L = Absolute amplitude of peak V.

A	B	C	D	E	F	G	H	I	J	K	L
F	00	2.04	2.96	3.87	4.79	5.54	1.83	1.66	3.49	1.29	0.22
F	50	2.00	2.92	3.87	4.79	5.67	1.87	1.79	3.66	1.57	0.22
F	60	2.04	2.92	3.83	4.87	5.62	1.79	1.79	3.58	1.04	0.24
F	70	2.08	2.87	3.83	4.92	5.67	1.74	1.83	3.58	0.76	0.16
M	00	2.17	3.08	4.12	5.04	6.00	1.95	1.87	3.83	0.68	0.17
M	50	2.17	3.04	4.17	5.12	6.25	1.99	2.08	4.04	1.05	0.22
M	60	2.08	3.17	4.12	5.00	6.17	2.04	2.04	4.08	0.78	0.18
M	70	2.12	3.08	4.21	5.12	6.04	2.08	1.83	3.91	0.57	0.13
F	00	1.96	3.04	4.21	5.25	5.87	2.24	1.66	3.91	1.73	0.26
F	50	2.00	2.92	4.33	5.04	5.92	2.33	1.58	3.91	1.92	0.25
F	60	2.00	3.08	4.46	0.00	6.04	2.45	1.58	4.04	1.06	0.17
F	70	2.08	3.00	4.08	5.08	6.25	1.99	2.16	4.16	0.91	0.21
M	00	2.00	2.79	4.04	5.21	5.83	2.04	1.79	3.83	0.89	0.16
M	50	2.04	2.87	4.08	5.12	6.04	2.04	1.95	3.99	1.22	0.22
M	60	2.08	2.96	4.08	5.12	5.87	1.99	1.79	3.79	1.00	0.14
M	70	2.04	3.04	4.08	5.17	5.83	2.04	1.74	3.79	0.93	0.14
F	00	2.09	2.84	4.00	5.17	6.09	1.83	2.00	3.91	1.36	0.19
F	50	2.13	2.88	4.13	5.21	6.00	1.91	1.79	3.79	1.07	0.16
F	60	2.17	2.88	4.09	5.29	5.96	1.83	1.79	3.71	1.33	0.16
F	70	2.13	2.92	4.13	5.25	5.96	1.91	1.75	3.75	1.07	0.16
M	00	2.12	3.00	4.50	5.46	6.42	2.37	1.91	4.29	2.09	0.23
M	50	2.12	3.04	4.50	0.00	6.37	2.37	1.87	4.24	1.47	0.25
M	60	2.17	3.25	4.46	0.00	6.33	2.29	1.87	4.16	1.69	0.22
M	70	2.21	3.04	4.37	5.75	6.50	2.16	2.12	4.29	2.33	0.35
F	00	2.12	2.95	3.95	4.95	5.70	1.74	1.65	3.49	0.93	0.25
F	50	2.08	2.95	3.91	4.91	5.74	1.74	1.74	3.57	1.13	0.27
F	60	2.12	2.95	3.91	4.91	5.78	1.70	1.78	3.57	0.96	0.23
F	70	2.12	2.95	3.91	4.87	5.78	1.70	1.78	3.57	0.88	0.21
M	00	2.04	2.87	3.96	5.00	5.50	1.91	1.95	3.45	0.55	0.16
M	50	2.08	2.87	3.96	5.04	5.54	1.87	1.82	3.45	0.69	0.18
M	60	2.04	2.92	4.00	5.04	5.75	1.95	1.99	3.70	0.54	0.15
M	70	2.00	2.96	3.96	5.00	5.67	1.95	1.54	3.66	1.29	0.22

Appendix G cont./...

F	00	2.15	3.19	4.11	4.65	6.07	1.85	1.85	3.81	1.17	0.27
F	50	2.15	3.07	4.23	4.69	5.90	1.98	1.55	3.64	1.78	0.32
F	60	2.07	3.07	4.11	5.02	5.94	1.94	1.73	3.77	1.67	0.35
F	70	2.07	3.15	4.11	4.94	5.90	1.94	1.69	3.73	1.22	0.22
M	00	1.92	2.87	4.12	5.00	5.96	2.20	1.83	4.04	1.56	0.25
M	50	1.96	2.92	3.96	5.04	5.92	1.99	1.95	3.95	1.06	0.17
M	60	1.87	2.83	4.12	5.17	5.92	2.24	1.79	4.04	1.16	0.22
M	70	1.92	2.87	4.00	0.00	5.96	2.08	1.95	4.04	2.11	0.36
F	00	2.00	2.96	4.00	0.00	6.00	1.99	1.99	3.99	1.06	0.19
F	50	1.96	2.96	3.96	4.83	6.17	1.99	2.20	4.20	1.13	0.17
F	60	2.00	2.92	3.92	5.29	6.12	1.91	2.20	4.12	1.20	0.18
F	70	2.00	3.00	4.00	5.29	6.17	1.99	2.16	4.16	1.21	0.17
M	00	2.23	3.19	4.40	0.00	6.07	2.06	1.56	3.73	2.67	0.24
M	50	2.19	3.15	4.36	0.00	6.15	2.06	1.69	3.85	2.15	0.25
M	60	2.19	3.19	4.23	0.00	6.13	1.94	1.81	3.85	2.15	0.28
M	70	2.23	3.15	4.40	0.00	6.23	2.06	1.73	3.89	2.30	0.30
F	00	2.12	3.21	4.21	4.96	5.96	2.08	1.74	3.83	1.65	0.33
F	50	2.17	3.04	4.25	4.96	6.08	2.08	1.83	3.91	1.45	0.32
F	60	2.20	3.08	4.25	4.96	6.08	1.95	1.83	3.79	1.87	0.28
F	70	2.12	3.04	4.25	5.04	6.04	2.08	1.83	3.91	2.07	0.31
M	00	2.12	3.12	4.21	5.33	6.04	2.08	1.83	3.91	1.00	0.17
M	50	2.12	3.17	4.17	5.50	6.04	2.04	1.87	3.91	1.44	0.26
M	60	2.08	3.17	4.17	5.58	6.08	2.08	1.91	3.99	0.86	0.19
M	70	2.08	3.25	4.17	5.25	6.29	2.08	2.12	4.20	1.53	0.26
F	00	1.96	3.00	3.87	5.00	5.96	1.91	2.08	3.99	0.81	0.29
F	50	2.04	2.96	4.04	0.00	5.96	1.99	1.91	3.91	0.58	0.21
F	60	2.04	3.04	4.00	0.00	5.96	1.95	1.96	3.92	0.88	0.28
F	70	2.04	3.04	4.04	0.00	6.00	1.99	1.95	3.95	0.94	0.30
M	00	2.12	3.17	4.00	4.58	5.92	1.87	1.91	3.79	3.85	0.50
M	50	2.12	2.87	4.04	4.58	5.92	1.92	1.87	3.80	3.92	0.51
M	60	2.32	3.08	4.04	4.54	5.92	1.62	1.87	3.49	3.77	0.49
M	70	2.17	3.25	4.04	4.58	5.92	1.87	1.87	3.74	2.39	0.43
F	00	1.87	2.71	3.71	4.42	5.54	1.83	1.83	3.66	2.21	0.31
F	50	1.83	2.71	3.75	4.33	5.54	1.91	1.79	3.70	1.91	0.21
F	60	1.83	2.62	3.75	4.46	5.58	1.91	1.83	3.74	1.92	0.25
F	70	1.83	2.67	3.71	4.46	5.54	1.87	1.83	3.70	2.27	0.25
M	00	2.08	3.12	4.29	5.46	6.33	2.20	2.04	4.24	1.10	0.11
M	50	2.08	3.29	4.29	5.37	6.29	2.00	1.99	4.21	1.44	0.13
M	60	2.42	3.21	4.29	5.37	6.25	1.87	1.95	3.83	1.67	0.20
M	70	2.29	3.21	4.29	5.54	6.17	1.99	1.87	3.87	1.40	0.14
F	00	2.12	3.08	4.29	5.04	5.92	2.16	1.62	3.79	1.81	0.29
F	50	2.12	3.12	4.00	5.08	5.87	1.87	1.87	3.74	1.69	0.27
F	60	2.08	3.17	3.96	5.08	5.92	1.87	1.95	3.83	1.75	0.28
F	70	2.12	2.92	4.08	5.21	5.87	1.95	1.74	3.74	1.33	0.24
M	00	1.96	2.87	3.87	5.50	5.50	1.91	1.62	3.54	1.08	0.28
M	50	2.00	2.87	3.87	5.58	5.58	1.87	1.70	3.58	1.15	0.31
M	60	2.00	2.92	3.87	5.50	5.50	1.87	1.62	3.49	1.21	0.34
M	70	2.00	2.97	3.83	5.62	5.62	1.83	1.79	3.62	1.17	0.34
F	00	2.09	3.04	3.96	4.59	5.79	1.79	1.75	3.62	2.06	0.35
F	50	1.96	2.84	3.96	4.54	5.79	1.91	1.75	3.79	1.93	0.29
F	60	2.12	3.00	3.96	4.59	5.79	1.66	1.75	3.50	1.77	0.32
F	70	2.09	2.92	4.00	4.67	5.84	1.83	1.75	3.66	2.31	0.37

Appendix G cont./...

M	00	2.08	3.00	4.21	5.25	6.29	2.12	2.08	4.20	3.20	0.32
M	50	2.12	2.96	4.21	5.29	6.17	2.08	1.95	4.04	2.50	0.25
M	60	2.08	3.00	4.25	5.29	6.17	2.16	1.91	4.08	2.10	0.21
M	70	2.04	2.83	4.17	5.29	6.29	2.12	2.12	4.24	1.90	0.19
F	00	2.12	3.29	4.21	5.62	6.08	2.08	1.87	3.95	1.00	0.15
F	50	2.17	3.25	4.25	0.00	6.04	2.08	1.79	3.87	1.47	0.25
F	60	2.12	3.29	4.21	5.50	6.29	2.08	2.08	4.16	0.68	0.13
F	70	2.21	3.21	4.25	5.04	6.29	2.04	2.04	4.08	1.35	0.23
M	00	2.17	3.08	4.25	5.29	6.00	2.08	1.74	3.83	1.40	0.21
M	50	2.12	3.08	4.33	5.29	6.00	2.20	1.66	3.87	1.61	0.21
M	60	2.12	3.17	4.29	5.37	6.17	2.16	1.87	4.04	1.30	0.17
M	70	2.12	3.21	4.42	5.37	6.17	2.29	1.74	4.04	1.30	0.17
F	00	2.00	2.83	3.96	5.04	5.92	1.95	1.95	3.91	0.75	0.09
F	50	2.04	2.87	3.96	4.62	6.21	1.91	2.24	4.16	1.54	0.20
F	60	2.04	2.75	3.92	4.67	5.92	1.87	1.99	3.87	1.91	0.21
F	70	1.96	2.71	4.04	5.12	6.45	2.08	2.41	4.49	2.00	0.20
M	00	2.12	2.92	4.08	5.08	6.17	1.95	2.08	4.04	1.27	0.19
M	50	2.04	2.96	4.12	5.12	6.17	2.00	2.04	4.12	1.40	0.21
M	60	2.12	3.00	4.12	5.12	6.21	1.99	2.08	4.08	1.33	0.20
M	70	2.17	2.92	4.17	5.21	6.25	1.99	2.08	4.08	1.06	0.17
F	00	2.12	3.21	4.21	5.42	6.42	2.08	2.20	4.29	0.74	0.14
F	50	2.08	3.17	4.17	5.37	6.29	2.08	2.12	4.20	0.92	0.12
F	60	2.21	3.21	4.17	5.42	6.33	1.95	2.16	4.12	0.65	0.09
F	70	2.21	3.25	4.25	5.42	6.42	2.04	2.16	4.20	1.50	0.14
M	00	2.00	2.96	4.21	5.25	6.17	2.20	1.95	4.16	1.80	0.18
M	50	1.96	3.00	4.21	5.46	6.17	2.24	1.95	4.20	1.20	0.18
M	60	2.04	3.25	4.29	0.00	6.29	2.24	1.99	4.24	1.36	0.19
M	70	2.00	2.96	4.29	5.50	6.21	2.29	1.91	4.20	0.88	0.14
F	00	1.96	3.00	3.92	5.21	5.92	1.95	1.99	3.95	1.41	0.24
F	50	2.00	3.00	3.96	5.33	6.00	1.95	2.04	3.99	1.47	0.25
F	60	2.00	3.00	4.00	5.29	6.04	1.99	2.04	4.04	1.93	0.27
F	70	2.04	3.00	3.96	5.25	6.17	1.91	2.20	4.12	1.53	0.26
M	00	2.00	3.21	4.21	5.25	6.21	2.20	1.99	4.20	2.08	0.25
M	50	2.04	3.08	4.17	5.33	6.29	2.12	2.12	4.24	1.70	0.22
M	60	2.08	3.17	4.25	5.33	6.29	2.16	2.04	4.20	1.78	0.25
M	70	2.12	3.29	4.25	5.25	6.42	2.12	2.16	4.29	1.39	0.25
F	00	2.190	2.90	4.52	0.00	6.11	2.23	1.48	3.81	2.42	0.29
F	50	2.19	2.86	4.40	5.02	6.07	2.10	1.56	3.77	2.50	0.25
F	60	2.27	3.15	4.44	5.07	6.11	2.06	1.56	3.73	1.84	0.35
F	70	2.19	3.32	4.40	5.11	6.07	2.10	1.56	3.77	2.80	0.28
M	00	2.12	2.96	4.00	5.00	6.12	1.87	2.12	3.99	1.00	0.21
M	50	2.04	2.83	4.00	5.17	6.33	1.95	2.33	4.29	1.92	0.25
M	60	2.00	2.87	4.12	0.00	6.12	2.12	1.99	4.12	1.08	0.14
M	70	2.12	3.00	4.25	5.12	6.37	2.12	2.12	4.24	1.92	0.23
F	00	2.09	2.75	4.25	5.38	6.58	2.08	2.04	4.21	1.80	0.10
F	50	2.14	3.29	4.25	5.29	6.34	1.83	2.00	3.91	1.50	0.15
F	60	2.14	2.21	4.46	5.13	6.38	2.04	1.83	3.96	1.07	0.16
F	70	2.25	3.17	4.59	5.42	6.42	2.25	1.75	4.08	1.15	0.15
M	00	2.15	2.90	3.98	4.94	6.23	1.73	2.14	3.98	1.13	0.17
M	50	2.15	2.69	3.98	4.98	6.02	1.73	1.94	3.77	1.75	0.28
M	60	2.07	2.90	4.27	5.19	6.23	2.10	1.85	4.06	0.77	0.10
M	70	2.07	2.90	4.19	5.02	6.19	2.02	1.89	4.02	1.31	0.17

Appendix G cont./...

F	00	2.00	2.96	4.04	5.00	6.08	2.04	2.04	4.08	0.94	0.17
F	50	2.08	2.79	4.04	4.87	6.37	1.95	2.33	4.29	0.85	0.17
F	60	2.08	3.04	4.12	4.83	6.29	2.04	2.16	4.20	1.17	0.14
F	70	2.04	2.79	4.08	4.83	6.21	2.04	2.12	4.16	0.79	0.11
M	00	2.17	3.37	4.33	5.21	6.17	2.16	1.83	3.99	0.70	0.14
M	50	2.17	3.25	4.29	5.25	6.27	2.12	1.91	4.04	1.14	0.16
M	60	2.25	3.12	4.25	5.25	6.33	1.99	2.08	4.08	1.00	0.17
M	70	2.21	3.33	4.33	5.42	6.46	2.12	2.12	4.24	1.20	0.18
F	00	2.04	3.00	4.12	5.21	5.96	2.08	1.83	3.91	1.47	0.28
F	50	2.04	3.00	4.12	5.21	5.92	2.08	1.79	3.87	1.06	0.25
F	60	2.00	2.96	4.17	5.21	5.92	2.16	1.74	3.91	1.62	0.21
F	70	2.12	3.00	4.12	5.21	5.96	1.99	1.74	3.83	1.73	0.26
M	00	2.23	2.94	4.02	5.02	5.82	1.69	1.69	3.48	1.89	0.36
M	50	2.23	2.98	4.02	5.11	5.82	1.69	1.69	3.48	1.56	0.25
M	60	2.11	2.90	4.07	5.11	5.90	1.85	1.73	3.69	1.61	0.29
M	70	2.19	3.07	4.15	5.15	6.02	1.85	1.77	3.73	1.06	0.17
F	00	2.00	3.25	4.42	5.21	6.12	2.41	1.70	4.12	2.50	0.25
F	50	2.08	3.25	4.37	5.17	6.21	2.29	1.83	4.12	2.08	0.27
F	60	2.08	3.37	4.29	5.33	6.17	2.20	1.87	4.08	1.39	0.25
F	70	2.08	3.33	4.33	0.00	6.17	2.24	1.83	4.08	1.21	0.17
M	00	1.92	2.92	4.00	5.08	5.83	2.08	1.83	3.91	1.00	0.28
M	50	2.00	3.00	3.96	5.21	5.83	1.95	1.87	3.83	1.09	0.25
M	60	1.96	2.92	4.00	0.00	5.87	2.04	1.87	3.91	1.46	0.35
M	70	1.96	2.92	4.00	5.29	5.87	2.04	1.87	3.91	2.06	0.35
F	00	2.00	2.96	3.96	4.87	5.87	1.95	1.91	3.87	1.83	0.42
F	50	2.00	2.96	3.87	5.00	5.83	1.87	1.95	3.83	1.18	0.23
F	60	2.00	3.00	3.87	5.00	5.87	1.87	1.99	3.87	1.03	0.33
F	70	2.04	2.96	3.96	4.92	5.96	1.91	1.99	3.91	0.89	0.25
M	00	2.00	3.04	4.04	5.25	6.33	2.08	2.29	4.33	0.77	0.17
M	50	2.00	3.00	4.08	5.25	6.46	2.08	2.37	4.45	0.77	0.17
M	60	2.00	3.00	4.04	5.33	6.46	2.04	2.41	4.45	2.43	0.17
M	70	2.08	3.00	4.04	5.29	6.00	1.95	1.95	3.91	0.70	0.14
F	00	2.12	3.21	4.17	0.00	5.92	2.04	1.74	3.79	2.20	0.44
F	50	2.08	3.21	4.12	0.00	5.83	2.04	1.70	3.74	2.44	0.39
F	60	2.17	3.21	4.50	0.00	5.97	2.33	1.37	3.70	1.35	0.31
F	70	2.12	3.25	4.29	0.00	5.92	2.16	1.62	3.79	1.65	0.33
M	00	2.17	3.04	4.12	5.42	6.04	1.95	1.87	3.87	1.25	0.15
M	50	2.17	3.08	4.12	5.17	6.12	1.95	1.83	3.95	2.00	0.20
M	60	2.17	3.08	4.17	0.00	6.17	1.99	1.95	3.99	1.83	0.22
M	70	2.21	3.04	4.25	0.00	6.08	2.04	2.08	3.87	1.43	0.20
F	00	2.08	3.17	4.42	5.79	6.29	2.33	1.87	4.20	1.27	0.28
F	50	2.21	3.37	4.42	5.50	6.17	2.20	1.74	3.95	1.17	0.21
F	60	2.21	3.25	4.50	5.37	6.37	2.29	1.87	4.16	1.33	0.28
F	70	2.17	3.21	4.37	0.00	6.21	2.20	1.83	4.04	1.45	0.32
M	00	2.27	3.36	4.40	5.52	6.15	2.02	1.89	4.06	0.57	0.08
M	50	2.27	3.32	4.61	5.61	6.23	2.23	1.89	4.10	0.57	0.08
M	60	2.27	3.44	4.57	5.52	6.23	2.19	1.87	3.98	0.50	0.07
M	70	2.44	3.32	4.36	5.73	6.19	1.81	2.52	4.64	0.91	0.10
F	00	2.08	3.08	3.96	0.00	5.92	1.87	1.95	3.83	1.31	0.17
F	50	2.04	2.87	3.96	5.00	6.00	1.91	2.04	3.95	2.00	0.24
F	60	2.00	2.87	3.96	5.21	5.96	1.95	1.99	3.95	1.05	0.16
F	70	2.08	2.87	4.00	5.00	6.00	1.91	1.99	3.91	1.56	0.10

Appendix G cont./...

M	00	2.21	3.00	4.37	5.17	6.08	2.16	1.70	3.87	0.59	0.13
M	50	2.17	3.29	4.54	5.17	6.12	2.37	1.58	3.95	1.57	0.22
M	60	2.21	3.17	4.50	5.21	6.29	2.29	1.79	4.08	0.90	0.19
M	70	2.17	3.25	4.33	5.12	6.33	2.16	1.99	4.16	1.06	0.19
F	00	2.04	2.83	3.79	4.42	5.71	1.74	1.91	3.66	1.71	0.24
F	50	1.96	2.96	3.75	4.37	5.79	1.79	2.04	3.83	2.06	0.37
F	60	2.00	2.71	3.83	4.46	5.79	1.83	1.95	3.99	2.07	0.29
F	70	1.96	2.67	3.75	4.42	5.67	1.79	1.91	3.70	2.43	0.34
M	00	2.12	2.96	4.04	5.04	5.96	1.91	1.91	3.83	2.80	0.28
M	50	2.17	2.92	4.17	5.21	5.96	1.99	1.79	3.79	2.50	0.25
M	60	2.17	3.04	4.17	5.08	6.00	1.99	1.83	3.83	1.22	0.22
M	70	2.12	2.95	4.21	5.25	6.04	2.08	1.83	3.91	1.94	0.31
F	00	2.12	3.04	4.08	5.37	6.50	1.95	2.41	4.37	1.21	0.17
F	50	2.29	3.12	4.08	5.33	6.42	1.79	2.33	4.12	0.82	0.14
F	60	2.29	3.08	4.21	5.25	6.37	1.91	2.16	4.08	1.28	0.07
F	70	2.17	3.45	4.21	5.37	6.50	2.04	2.29	4.33	1.00	0.10
M	00	2.12	3.00	4.12	5.08	6.04	1.99	1.91	3.91	1.22	0.22
M	50	2.17	2.96	4.08	5.17	6.00	1.91	1.91	3.83	1.43	0.20
M	60	2.21	2.92	4.12	5.17	6.12	1.91	1.99	3.91	1.53	0.23
M	70	2.12	3.08	4.25	5.08	6.04	2.12	1.79	3.91	2.45	0.27
F	00	2.12	3.12	3.96	4.75	5.67	1.83	1.70	3.54	1.32	0.25
F	50	2.17	2.92	4.00	4.87	5.75	1.83	1.74	3.58	1.00	0.20
F	60	2.12	3.12	4.00	4.92	5.75	1.87	1.74	3.62	1.25	0.20
F	70	2.21	3.04	4.00	4.96	5.79	1.79	1.79	3.58	1.05	0.22
M	00	2.29	3.33	4.37	5.37	6.12	2.08	1.74	3.83	2.18	0.35
M	50	2.21	3.21	4.33	0.00	6.21	2.12	1.87	3.99	1.89	0.34
M	60	2.25	3.21	4.25	5.25	6.25	1.99	1.99	3.99	1.61	0.29
M	70	2.21	3.29	4.33	0.00	6.25	2.12	1.91	4.04	1.68	0.27
F	00	1.96	2.96	4.04	5.29	5.92	2.08	1.87	3.95	1.75	0.28
F	50	1.96	2.96	4.04	5.79	6.25	2.08	2.20	4.29	1.35	0.27
F	60	1.92	2.92	4.08	6.04	6.50	2.16	2.41	4.58	1.36	0.30
F	70	1.92	2.83	4.00	0.00	6.12	2.08	2.12	4.20	1.58	0.30
M	00	2.08	3.08	4.25	4.87	5.96	2.16	1.70	3.87	1.63	0.26
M	50	2.08	2.87	4.29	5.08	6.21	2.20	1.91	4.12	2.18	0.24
M	60	2.17	2.79	4.29	4.96	6.12	2.12	1.83	3.95	1.47	0.22
M	70	2.08	2.79	4.25	4.83	6.08	2.16	1.83	3.99	1.57	0.22
F	00	2.00	3.04	4.04	4.83	6.00	2.04	1.95	3.99	2.28	0.32
F	50	2.12	3.00	4.17	4.83	6.04	2.04	1.87	3.91	2.50	0.35
F	60	2.00	3.08	4.04	4.83	6.04	2.04	1.99	4.04	2.89	0.26
F	70	2.00	3.08	4.08	4.87	6.00	2.08	1.91	3.99	2.45	0.27
M	00	2.04	2.92	4.08	5.04	5.71	2.04	1.62	3.66	1.47	0.22
M	50	2.04	2.96	4.04	5.12	5.75	1.99	1.70	3.70	1.33	0.20
M	60	2.04	3.00	4.08	5.17	5.87	2.04	1.79	3.83	1.40	0.21
M	70	1.96	3.00	4.08	5.17	5.87	2.12	1.79	3.91	1.92	0.25
F	00	2.12	3.17	4.21	5.29	6.04	2.08	1.83	3.91	1.00	0.21
F	50	2.21	3.21	4.29	5.46	6.17	2.08	1.87	3.95	0.83	0.19
F	60	2.21	3.25	4.25	0.00	6.25	2.04	1.99	4.04	1.26	0.24
F	70	2.29	3.37	4.37	5.46	6.21	2.08	1.83	3.99	0.90	0.19
M	00	2.27	3.07	4.36	0.00	6.02	1.98	1.56	3.64	1.44	0.26
M	50	2.07	2.90	4.36	0.00	6.11	2.19	1.64	3.94	2.44	0.22
M	60	2.02	2.94	4.52	0.00	6.11	2.39	1.48	3.98	1.75	0.21
M	70	2.07	3.23	4.27	5.36	6.04	2.10	2.02	4.23	1.31	0.17

APPENDIX H

Instructions to run the MANOVA statistical procedure on the SAS/DAT computer program (Murray, M. 1989).

```
data cyril;
input sex $ mask y1-y10;
cards;
;(Raw data)
run;
proc print;
run;
proc glm;
class sex mask;
model y1-y10 = sex mask sex*mask;
manova h=sex / printh htype=1;
manova h=mask / printh htype=1;
manova h=sex*mask / printh htype=1;
run;
```

APPENDIX I

BAER - TEST PROTOCOL

TECHNICAL AND PROCEDURAL CONSIDERATIONS

STIMULUS : clicks - 100 µsec. duration

TRANSDUCER : electrodynamic TDH-39P earphones housed in free field audio-cups.

ELECTRODES : self-adhesive silver-silver chloride.

EVOKED RESPONSE AUDIOMETER : Cadwell Quantum 84

ELECTRODE SITES : positive - F_z high forehead
negative - ipsilateral mastoid
ground - contralateral mastoid

POLARITY : alternating

REPETITION RATE : 11,29per sec

FILTER PASS BAND : 100Hz - 3000Hz

SWEEP TIME : 1 division = 1 msec

TIME FRAME : 10 msec post-stimulus

NO. OF CLICKS PER TRIAL : 2048

NO. OF TRIALS : Minimum-Two to ensure waveform repeatability.

CONTRA-LATERAL MASKING : 50dBHL, 60dBHL & 70dBHL

LEVEL OF TEST EAR STIMULUS : kept constant at 70dBnHL

ARTIFACT REJECTION : switched on

RECORDING OF RESPONSES : by built in ALPS Printer

TEST ENVIRONMENT : ANECHOIC CHAMBER - electromagnetically screened low noise levels ANSI (1979).

PATIENT STATE : appeared to be relaxed or asleep lying in a supine position on a standard patient couch.

NB. : A control run prior to stimulation was done to allow for comparing and identifying true responses.