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I am submitting herewith a dissertation written by Eva Kinsey Saffer entitled "Auditory Evoked Potentials: ABR, MLR, and the Late Auditory Responses for Children With and Without Histories of Otitis Media." I have examined the final copy of this dissertation for form and content and recommend that it be accepted in partial fulfillment of the requirements for the degree of Doctor of Philosophy, with a major in Speech and Hearing Science.

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We have read this dissertation
and recommend its acceptance:

Accepted for the Council:

Associate Vice Chancellor
and Dean of the Graduate School

AUDITORY EVOKED POTENTIALS: ABR, MLR, AND THE LATE AUDITORY RESPONSES FOR CHILDREN WITH AND WITHOUT HISTORIES OF OTITIS MEDIA

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ABSTRACT

A consequence of chronic otitis media in early childhood is a reduction of sensory stimulation of the peripheral and central auditory system. This reduction in sensory stimulation may be viewed as a form of auditory deprivation. Three groups included for this study were children with minimal history of otitis media (NL), children with history of otitis media (OMH), and children with active otitis media (AOM). The purpose was to compare the measurements of the auditory brainstem response (ABR), middle latency response (MLR), and the late auditory response (LAR) across the groups to determine if there were effects of sensory deprivation on these responses.

Shorter latency for the waves I and III were identified for the NL and OMH groups when compared to the AOM group. The interwave interval latency for III-V was shorter for the AOM group when compared to the NL and OMH groups. No differences were identified between the NL and OMH groups. Waveform replication and identification were 100% for the NL group for the MLR. The numbers of waves present for the OMH group were 81.25% for the right ear and 62.5% for the left ear. The wave identification was 77.78% for the AOM group. The results show that otitis media influences the morphology and replication of the waves of the MLR. The wave N1 latency of the LAR was longer for the AOM group compared to the NL group. The P2 latency was longer for the left ear for the AOM group compared to the NL group.

Consequences of otitis media were reflected in delayed wave latencies. ABR latencies on the whole were not affected by history or presence of otitis media. The percentage of subjects with MLRs was smaller for the OMH or those with AOM. LARs were delayed in latency for subjects with history of otitis media and even more delayed for subjects with active

otitis media. It was hypothesized that these changes for the LAR represent alterations in the maturation pattern. The rank order pattern identified with the AOM group exhibiting the longest latencies and the OMH group second with longer latencies than the NL group suggest a slowly recovering system. Re-testing of the subjects in one year may provide additional information about recovery of the LAR latency.

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CHAPTER I

INTRODUCTION AND STATEMENT OF PURPOSE

A consequence of chronic otitis media in early childhood is a reduction of sensory stimulation of the peripheral and central auditory system. The reduction in the auditory signal caused by otitis media may be viewed as a form of auditory deprivation. During the early years (birth to three), many structures within the central nervous system are immature and still developing (Fria and Doyle 1984; Moller and Jenetta, 1985; Musiek, Verkest, and Gollegly, 1988). It has been demonstrated that auditory deprivation in the critical periods compromises the normal development and maturation of the peripheral brainstem, mid-brain and cortical structures along the auditory pathway (Clopton and Silverman, 1977, Webster and Webster, 1979; Trune and Morgan, 1988). Batkins, Groth, Watson, and Ansberry (1970) suggested that adequate levels of environmental stimulation are necessary for the development of sensory-neural responsivity.

There is considerable support for the alteration of the central auditory pathway in animal literature. Structural changes within the auditory brainstem of animals, such as smaller neuron size and shorter dendritic tree length, have been identified following the artificial reduction of sound to the auditory system. These changes occur in ipsilateral anteroventral cochlear nucleus and lateral superior olive and in the contralateral medial nucleus of the trapezoid body and the inferior colliculus (Webster and Webster, 1977, 1979; Blatchley, Williams and Coleman, 1983; Webster, 1983; Trune and Morgan, 1988; Pasic and Rubel, 1991).

The auditory evoked potentials (AEPs) provide information about the location of abnormalities affecting the

central nervous system pathways and they may provide objective demonstration of dysfunction in the sensory pathways even when the clinical examination is normal (Hillyard and Picton, 1987). The auditory brainstem response (ABR), middle latency response (MLR) and the late auditory response (LAR) may provide information regarding the differential susceptibility (if any) to auditory deprivation as a function of the level in the auditory pathway.

It might be expected that the children with active otitis media and histories of otitis media would have longer latencies for the auditory evoked potential measures than children without significant history of otitis media. The expected latency shift is supported in the animal deprivation research where deprivation of sounds caused longer latency in young rats (McGinn and Henry, 1975). The specific aim of this study was to compare the ABR, MLR, and LAR indices across three groups of children: minimal history of otitis media (NL), history of otitis media (OMH), and active otitis media (AOM) to determine the effects of auditory deprivation on these responses.

PURPOSE OF STUDY

The purpose was to determine whether the effect of auditory deprivation as caused by early otitis media could be measured between subject groups in the latency measurements of the waves of the auditory evoked potentials (Waves I, III, and V of the ABR; Na and Pa for the MLR; and N1, P2, N2, and P3 of the LAR). The null hypothesis states that: 1. There will be no differences in the latency of the waves I, III, and V of the ABR between the subjects in the NL, OMH, and AOM groups. 2. There will be no differences in the latency of the waves Na and Pa of the MLR between the subjects in the NL, OMH, and AOM groups. 3. There will be no differences in the latency of the waves N1, P2, N2, and P3 of the LAR between the subjects in the NL, OMH, and AOM groups.

CHAPTER II

REVIEW OF THE LITERATURE

The following review of the literature presents topics related to auditory deprivation. The varieties of definitions of auditory deprivation in the literature make it especially difficult to compare studies. The variations ranged from removal from sound environment with no structural changes in the animals to structural alterations within the animals. Changes from conductive type auditory deprivation were reviewed. The ABR, MLR, and the LAR as measures of change in the auditory system were described.

AUDITORY DEPRIVATION

The peripheral auditory system is functional well before birth with the neuromaturation continuing into the second or third year of life (Fria and Doyle, 1984; Moller and Jennetta, 1985). The central auditory system anatomy in deprived animals is influenced by peripheral system dysfunction during a species specific critical developmental period (Mc Ginn and Henry, 1975; Clopton and Silverman, 1977, 1978; Webster and Webster, 1979; Webster, 1983; Blatchley, 1983; Trune and Morgan, 1988). If the peripheral structures do not adequately stimulate the central auditory structures, then the physiological response may be altered. Anatomical changes in animals deprived of auditory stimulation show smaller cell areas in ipsilateral anteroventral cochlear nucleus and contralateral trapezoid body, superior olivary complex, and inferior colliculus (Perier, Alegria, Buyse, D'Alimonte, Gilson and Serniclaes, 1984; Trune and Morgan, 1988; Pasic and Rubel, 1991).

Webster and Webster (1977, 1979, 1982,1983) observed that both postnatal auditory deprivation and experimentally produced conductive hearing loss in mice resulted in incomplete maturation of most brainstem neurons. The size of the ventral cochlear nucleus, medial nucleus of the trapezoid body, and the inferior collicular nucleus were reduced ipsilateral to the ear with conductive loss compared with the control side that had not been impaired. There was a critical period, before 45 days of age during which acoustic stimulation had a more pronounced effect on neuronal maturation than the same stimulation given between 45 and 90 days. The changes observed were only partially reversible in animals that were submitted to auditory deprivation until 45 days of age and then placed in a normal acoustic environment.

Neural plasticity was demonstrated by Webster and Webster (1977, 1979) and Pasic and Rubel (1991). The changes in the cochlear nucleus cell size were partially reversible in the animals that were submitted to auditory deprivation and then placed in a normal acoustic environment. Pasic and Rubel (1991) blocked the auditory nerve action potentials and measured the spherical cells of the antroventral cochlear nucleus ipsilateral to the manipulation. The animals showed a decrease in cell size of 16 percent. They allowed the animals to recover for seven days following the blockade for 24 or 48 hours, after which cell size returned to the previous levels. Even though the action potential blockage does not represent the same type of auditory deprivation as otitis media, it does show a recovery of function when auditory stimulation was returned.

In contrast, Tucci, Born, and Rubel (1987) did not find significant effects of a conductive or mixed conductive and sensorineural hearing loss on anatomical and physiological properties of the chicken auditory system. The animals underwent either a columella (ossicle) removal, or an oval window puncture. The tympanic membrane puncture and

columella removal conductive hearing loss were not associated with either cell area changes in the nucleus magnocellularies or changes in spontaneous neuronal activity. The oval window puncture (sensorineural hearing loss) was associated with a cell area reduction of 20% as well as marked decline in activity within the auditory nuclei. The researchers concluded that auditory deprivation, as produced by a purely conductive hearing loss, did not affect the ear in the same manner as deafferentation. They suggested that the previous researchers may not have obtained a pure conductive component in experiments (Webster and Webster 1977, 1979).

Several inferences can be drawn from the animal studies. The deprivation of stimulation from conductive hearing loss can lead to structural changes within the auditory brainstem. The animal studies identified periods of particular sensitivities to auditory stimulation that varied between species. Tucci, Born, and Rubel (1987) and Pasic and Rubel (1991) have demonstrated that alterations caused by conductive pathology was temporary.

AUDITORY EVOKED POTENTIALS

Electrophysiological measures of the brainstem, midbrain and cortical structures allow a non-invasive assessment of the possible effect of reduced auditory stimulation in children. The auditory evoked potentials (AEP) consist of a series of scalp recorded responses generated by the auditory mechanism. Each wave reflects the synchronized activity of a large number of cells responding to the sounds. As many as fifteen AEPs have been identified within the first 500 msec post stimulus onset (Picton, Hillyard, Krausz, & Galambos, 1976). These potentials are labeled in terms of their latency. Figure 1 depicts on a linear time scale the major AEPs that can be recorded from the human scalp. They consist of three major groups of potentials, the ABR, the MLR, and the LAR. The ABR can be identified in the 2-10 msec time

epoch. The thalamus and auditory cortex are the proposed sites for the middle latency responses at 8-50 msec. The late auditory responses that occur in the range 50-300 msec in the mature central nervous system arise from the primary and secondary areas of the cerebral cortex.

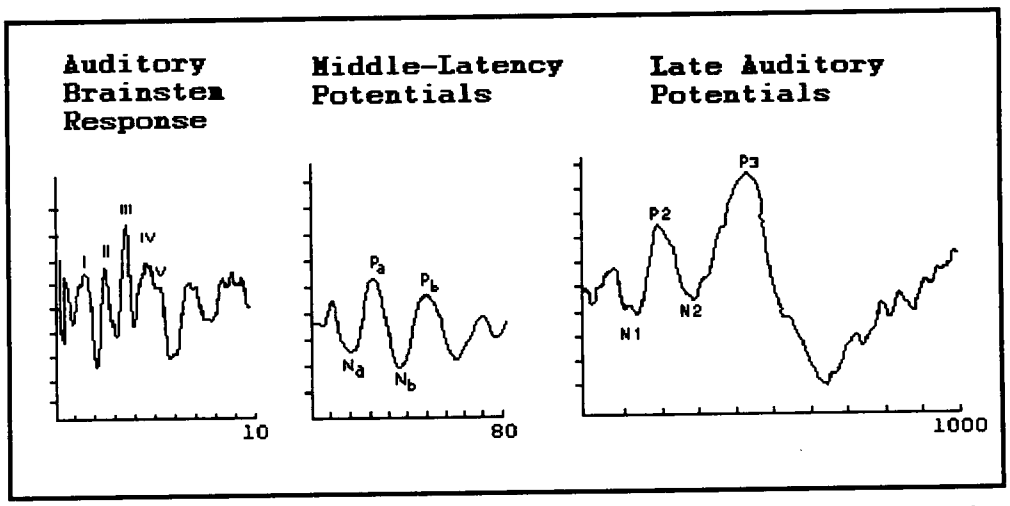


Figure 1. Three primary time epochs of the auditory evoked potentials. The abscissa represents latency in milliseconds. The ordinate represents amplitude in micro-volts.

The Auditory Brainstem Responses

Jewett and Williston (1971) described the origin of the far field scalp recorded ABR. The ABR has been observed as a series of potentials occurring within 10 msec after presentation of a click stimulus. Peak identification and classification include waves I through V. The potentials are generated by the auditory nerve (waves I and II) and multiple fiber groups in the pons (waves III, IV, and V) (Moller and Janetta, 1985; Legatt, Arezzo, and Vaughn, 1988). The ABR has two principal applications: the evaluation of peripheral auditory system function and the evaluation of the neural

function of the acoustic nerve and the brainstem (Jacobson, 1985).

The maturation of the auditory system is incomplete at birth for brainstem nuclei and cochlear regions. The various ABR indices have different maturational rates. Wave I is the first to reach adult latency at approximately three months of age. Waves III and V continue to decrease in latency until age two or three years. The I-V interwave interval, which measures neural conduction time, does not reach adult values until almost three years of age (Murry, 1988; Gorga, Kaminski, Beauchaine, Jesteadt, and Neely, 1989; Eggermont, 1985; Fria and Doyle, 1984). The longer time required for latency stabilization of the later waves indicates a caudorostral development pattern in the brainstem (Musiek, 1989).

There is a consensus that conductive pathology prolongs wave component latency for unmasked air conduction stimuli. Finitzo-Hieber, Hecox, and Cone (1979), Fria and Sabo (1980), and McGee and Clemis (1982) identified prolonged wave latency for conductive pathology with no changes in the interwave intervals. Fria and Sabo (1980) carefully defined the populations by including only patients with otitis media validated by surgery. Their conclusions were that the ABR latency was a sensitive indicator of otitis media and the ABR data can be used to estimate the amount of conductive hearing component.

The ABR has been used to predict which children had histories of otitis media. Retrospective electrophysiologic studies of children with histories of otitis media have identified prolonged absolute wave III and V and interpeak I-V latencies (Folsom, Weber, and Thompson, 1983). The children (age six to ten) tested by Folsom et al were otologically clear and had normal hearing sensitivity at the time of the test. Lenhardt, Shaia, and Abedi (1985) reported on two eight year old girls with chronic recurrent otitis

media who demonstrated normal latencies for Wave I, but prolonged waves III and V relative to the laboratory norms. They hypothesized that the changes in the evoked potentials did not reflect structural damage but slowly recovering system.

Chambers, Rowan, Mathies, and Novak (1989) performed ABR in children with previous otitis media and compared the results with a matched control with no history of otitis media. Their subjects were children who had just had tympanostomy tubes inserted due to the documented histories of OME. They found prolonged absolute wave III and V latencies and I-III interpeak latency differences in children with histories of otitis media.

Gunnarson and Finitzo (1991) evaluated children, age five to seven, with longitudinally documented records of early otitis media. Hearing levels and otitis history were documented from birth. The children were free of otitis media at the time of the testing. Group classification was based on the infant ABR test. The normal group had normal ABR at infancy. Group B had wave V present but prolonged in latency and group C had absent wave V at 30 dB. The results indicated that the normal control group differed from both groups for the latencies of waves III and V and III-I and V-I interwave interval. The latencies were prolonged for the groups B and C for waves III and V.

There are reports in the literature of changes in the latency of the ABR components with conductive hearing loss. These are due to changes in the hearing thresholds. ABR data have been used to estimate the amount of conductive hearing component (Finitzo-Hieber, Hecox, and Cone, 1979; Fria and Sabo, 1980; McGee and Clemis, 1982; Stockard and Curran, 1990). The ABR latencies were reported to remain altered after resolution of the otitis media (Folsom, Weber, and Thompson, 1983; Lenhardt, Shaia, and Abedi, 1985; Chambers,

Rowan, Mathies, and Novak, 1989; Gunnarson and Finitzo, 1991).

The Middle Latency Response

The MLRs occur between 10 and 80 msec, and are composed of positive and negative waves Na, Pa, Nb and Pb. Three components are dominant, two centered at or near the vertex and one centered in posterior temporal areas over the auditory cortex (Kileny, 1983; Ozdamar and Kraus, 1983; Vaughan and Ritter, 1970, Kraus and McGee, 1988). The most prominent feature of the MLR is a positive peak, Pa, in the 25 msec to 35 msec latency region and a 53 msec positive peak, usually called Pb. Pa is preceded by a less prominent negative peak centered at the vertex called Na (Cacace, Satya-Murti, and Wolpaw, 1990).

The generators for the MLR have not been clearly defined. Evidence has mounted in support of multiple generators in the auditory cortex as the bases for these potentials (Picton and Hillyard, 1974; Kraus, Smith, Reed, Stein, & Cartee, 1985; Pelizzone, Hari, Makela, Huttunen, Ahlfors, and Hamalainen, 1987; Cohen, 1982; and Woods, Clayworth, Knight, Simpson, Naeser, 1987). Temporal cortex involvement had been demonstrated in data from normal adults (Cohen, 1982; Ozdamar and Kraus, 1983; Wolpaw and Wood, 1982; Wood and Wolpaw, 1982; Kileny, 1983).

The course of MLR development in humans has been shown to extend throughout the first decade of life, with a steady increase in MLR detectability over that time (Kraus, Smith, Stein, and Cartee, 1985). The MLRs have been reported for infants and neonates as young as 31 weeks gestational age. Mendel, Adkinson, and Harker (1977) reported that infant MLRs had shorter peak latencies than adult MLRs. Mendelson and Salamy (1981) found no significant latency difference from 60 subjects in four age groups (premature infants, newborns, children and adults). Kraus, Smith, Stein, and Cartee,

(1985) completed a study on the age effects on the MLR in several categories of subjects. Considerable variability among the subjects younger than 10 years of age was identified. Na was present in about 60 to 70 percent of the children less then one year of age. The presence of MLR waves increased with age until Na and Pa waves were present in nearly 100% of the subjects at ages 10 to 12 years. In the age range from 8-11 years of age the MLR was frequently "abnormal" in a significant percentage of uninvolved children. Kraus et al (1985) based abnormal waveforms on two criteria. Na and Pa were considered absent if bioelectric activity occurred below or above the baseline that was determined by the ABR. Second the waves were judged on the basis of the characteristic appearance of adult MLRs which included a broad negative trough beyond the ABR followed by a vertex positive response. The variation in the results of the identification of the MLR in children may be from the different filters and testing procedures. Scherg (1982) and Kileny (1983) reported that narrow filtering of the brain's electrical activity could interact with early (ABR) activity to produce an artificial response in the 25 to 30 msec latency range.

Use of the MLR has been limited due to the controversy surrounding the generator sources and the variability in normal subjects. The subjects with central auditory processing disorders (CAPD) showed a prevalence of abnormal MLRs. The MLR in CAPD takes the form of an inappropriate rate dependence rather than a present or absent phenomenon (Jerger, Oliver, Chmiel, 1988). The MLR waves in children with central auditory processing disorders would be present with slower rates and absent with increases in the rate of the stimulus. The middle latency responses have been shown to be affected by early otitis media (Gunnarson and Finitzo, 1988). Gunnarson and Finitzo documented prospectively from birth through 18 months the amount of conductive component

that was caused by the otitis media. The children were free of otitis media and had normal hearing at the time of the testing. Children with early otitis media had a lower percentage of detectable PA waves than normal controls.

The information from the MLR may be limited due to the variability of the response. The response is absent in a significant percentage of cases even with normal hearing. The data analysis has been restricted to the determination of the presence or absence of the waveforms. If MLRs are present less often in OMH listeners than in normal listeners, it may be that deprivation slowed maturation.

Late Auditory Response

The LARs are composed of three relatively high amplitude waves: N1, P2, and N2. The N1 is a negative trough occurring near 100 msec. The P2 is a positive wave at approximately 200 msec followed by a negative trough, the N2 at 250 msec. Following the N2 component is the P3, or P300, component. It is the most prominent and the best characterized of the components related to higher level processing. The P3 consists of a passively elicited component, P3a (Squires 1983, Polich 1989) as well as a task related, P3b response (Donchin, 1978). The P3 of the LAR reflects psychological processes underlying the analysis of sounds (Donchin, Ritter, and McCallum, 1978).

Neurophysiology of the LARs is still being explored. Mapping the LAR across the head indicated that the potentials are widely distributed across central and frontal scalp regions. The broad distribution of the LAR suggested that the waves are a composite of activity generated simultaneously in a number of cortical regions. These regions include the primary auditory cortex and temporal and frontal association areas (Kileny, 1985; Mason and Mellor, 1984; Musiek and Baran, 1987; and Picton, Hillyard, Kraus and Galambos, 1976; Kurtzberg, 1989; Wood and Wolpaw, 1982).

The LAR latency vary as a function of age (Courchesne, 1979; Musiek, 1989; Donchin, 1978; Hillyard and Picton, 1987; Mason and Mellor, 1984; and Musiek 1989; Ohlrich, Barnet, Weiss, and Shanks, 1979; Finley, 1983, Polich, Howard, and Starr, 1985; Pfefferbaum, Ford, Wenegrat, Roth, and Kopell, 1984). The LAR decreases in latency from birth until the teenage years and then at about the third decade the latency increases in a rather systematic manner (Musiek, 1989; Courchesne 1978; Goodin Squires, and Henderson, 1978; Pfefferbaum, Ford, Wenegrat, Roth and Kopell, 1984; Polich, Howard, and Starr, 1985). These shifts are quite substantial: as much as 200 msec for N2 between birth and 40 months of age. Conflicting results have been reported on identification of the LAR in young children. Identification of N1 and P2 were unreliably obtained in subjects at 6 years of age (Martin, 1988). Johnson (1989) reported adult like latencies and amplitudes for N1 and P2 in school age children. The discrepancies may be related to the definition of the time line where the wave would be identified. Reports for children younger than six years of age were sparse.

Researchers have suggested that the subject be involved in a behavioral task while the LARs are recorded (Neville, 1988; Donchin, Ritter, McCallum, 1978). However, not all LARs require a conscious perceptual decision (Friedman, Simson, Ritter, and Rapin, 1975). LARs can be tested passively (no subject response) or actively (subjects are required to respond to target stimuli) (Polich, 1989). The late auditory response is observed in response to infrequent stimuli or unexpected changes in a repetitive stimulus sequence, even when the subject is instructed to ignore the stimuli. The N2 and the P3a may reflect some component of an alerting or orienting response to unpredictable or unexpected changes in ambient stimulation (Squires and Hecox, 1983). Polich (1989) found that the P3 components from the passive

sequence procedure were morphologically quite similar to those obtained from the active discrimination task.

The automatic responses of the LAR make these potentials a good clinical tool for testing young children. Many children with otitis media frequently have been identified as exhibiting difficulty with central auditory processing. Children with central auditory processing disorders showed increased latencies of N1, P2, and P3; and smaller amplitudes for P3 when compared to control children without CAPD (Jirsa and Clontz, 1990). The effects of chronic otitis media on the late cortical potentials were investigated by Seikel, Brandt, and Ferraro (1989). Seikel et al identified differences in ear of presentation. The groups differed in responses based upon ear of presentation. The control group had longer P2 latencies for stimuli delivered to the right ear than left, whether those stimuli were words or tones. The otitis media group responded with longer P2 latencies to tonal stimuli delivered to the left ear. Right ear presentation did not show differentiation between the tonal and verbal stimuli in the P2 latency. Speech stimuli produced shorter latency than the tones for the P2 and N2 waves.

Identification of group differences in these potentials may help establish clinical application in these areas. The present investigation defines auditory deprivation as a reduction in the auditory stimulation caused by transient otitis media. Deprivation during the critical period would produce the greatest effect on the developing auditory system. Alterations of the auditory pathway that were reported in the animal studies identified increased latency of the auditory potentials (Batkins, Groth, Watson, and Ansberry, 1970). Use of the auditory evoked responses for identification of characteristics in children with otitis media might establish a base for prediction of later development. In the present study, activity in the auditory

nervous system was studied using responses that arise from peripheral as well as central sources. This was done to determine if changes due to otitis media can occur at some loci within the auditory system and not at others.

CHAPTER III

METHODS AND PROCEDURES

The review of the literature identified studies that suggested that reduced auditory stimulation may affect neural development of the auditory system. The effect of these changes may be measurable using the auditory evoked potentials. The latencies of the components of the ABR, MLR, and LAR were used to evaluate three groups of children minimal history of otitis media (NL), otitis media history (OMH), and active otitis media (AOM). The descriptions of subjects, general procedures and specific procedures are presented in this chapter.

SUBJECTS

Subjects were recruited from an otolaryngology clinic and a pediatric clinic in San Antonio, Texas. The ethnic background for the subjects was mixed (black, hispanic, and white). The data were collected from June 1993 to March 1994. The informed consent was obtained before acquisition of history and testing. Three groups of children were used for identification of latency change of auditory evoked potentials as the result of otitis media. For this study otitis media was defined as an inflammation of the middle ear without reference to etiology or pathogenesis. The children did not exhibit obvious mental retardation, any condition known to be associated with retarded development, major congenital anomalies such as cleft palate, or chronic illness.

The subjects identified as minimal history of otitis media (NL), consisted of 11 children (4 girls, 7 boys) who

ranged in age from 4 years 0 months to 4 years 9 months (mean = 4 years 5 months). These children had fewer than four ear infections during the first four years of life. The medical history was established by reports from the child's pediatrician and parent interviews. The children have not been evaluated by an otolaryngologist or had P.E. tubes for ear infections. This is considered to be normal. Hearing levels were less than 15 dB HL (re ANSI, 1969) with an air bone gap of less than 10 dB HL for 500 to 4000 Hz. The mean pure tone threshold for 500, 1000, and 2000 Hz for the normal group was 10.6 and 11.3 dB for the right and left ear respectively. The speech reception thresholds (SRTs) were within 10 dB of the pure tone air conduction thresholds suggesting good test reliability. Speech stimuli are more interesting to children which could result in a lower measure for the SRT. The SRT may be more representative of the auditory threshold for this group. The pure tone average was used for determination if the children met the criterion for inclusion in the groups. A negative otologic history does not rule out the presence of a history of otitis media because some children are asymptomatic. Because it was not possible to longitudinally assess these children from an early age, it was possible that some of them had episodes of asymptomatic middle ear effusion of which there were no records.

The subjects identified as otitis media history (OMH), consisted of 16 children (4 girls, 12 boys) who ranged in age from 4 years 0 months to 5 years 0 months (mean = 4 years 5 months). Inclusion in this group was based upon multiple recurrent episodes of otitis media with fluid (range four to six) with the initial episodes occurring at or before 12 months of age. Confirmation of this history was established by reports from the child's pediatrician and otolaryngologist. The pediatricians utilized a referral criterion of six or more ear infections in one year. All the

children in this group had received myringotomies with tubes. At the time of the testing the children were otitis free. Hearing levels were less than 15 dB HL (re ANSI, 1969) with an air bone gap of less than 10 dB HL for frequencies at octave intervals 500 to 4000 Hz. The mean pure tone threshold for 500, 1000, and 2000 Hz was 10.5 dB for the right ear and 10.0 dB for the left ear. The SRTs were within 10 dB of the pure tone air conduction thresholds suggesting good test reliability.

The subjects identified as active otitis media (AOM), consisted of 9 children (4 girls, 5 boys) who ranged in age from 4 years 0 months to 5 years 0 months (mean = 4 years 6 months). Inclusion criterion included confirmation of active otitis media by examination by an otolaryngologist. The otolaryngologist's decision was based upon otoscopy, tympanometry, and audiometry. The inclusion criteria were type B tympanogram and an air bone gap greater than 10 dB HL for 500-4000 Hz. The mean pure tone thresholds for 500, 1000, and 2000 Hz were 24.2 dB for the right ear and 27.6 dB for the right ear. The SRTs were within 10 dB of the pure tone air conduction thresholds suggesting good test reliability.

Audiometric and tympanometric evaluation were completed to determine present status of the hearing and middle ear system. All audiological tests were completed in a sound treated booth (Industrial Acoustics). A Grason-Stadler Model 16 audiometer with Telephonics TDH 50P earphones was used to complete the pure tone and speech audiometry. The audiometer was calibrated for output intensity, linearity of attenuation, harmonic distortion, lack of transients or noise when the tone is interrupted, and frequency, in accordance with ANSI 1969 specifications. Testing was conducted at 250, 500, 1000, 2000, 4000 and 8000 Hz. The 1000 Hz tone was re-tested to establish test re-test reliability. The test was considered reliable if the re-test at 1000 Hz was within plus

or minus 10 dB of the original threshold. The SRT were obtained as a reliability check for the air conduction testing. The SRT was within 10 dB of the pure tone average of 500, 1000, and 2000 Hz. There were no cases of disagreement between the pure tone average and the SRT. The mean pure tone average and speech reception thresholds for the groups are shown in
Table 1.

Impedance measures were taken using a Grason-Stadler Model 33 Version 1 Middle ear Analyzer. The impedance meter was calibrated for intensity and harmonic distortion of the probe tone using the manufacturer's calibration procedure. Tympanograms were plotted from + 200 to - 300 da Pa. Classification of normal tympanograms followed the revised guidelines for acoustic immittance screenings (ASHA, 1985). The abnormal results include flat tympanogram and low static compliance (<.2 ml).

TABLE 1. Means for Pure Tone Average (PTA) and Speech Reception Thresholds (SRT) for the Right (R) and Left (L) Ears for the Three Groups. Measurements are in decibels. Air Bone Gap for the Groups.

GROUP	MEAN PTA FOR AIR CONDUCTION		MEAN PTA FOR BONE CONDUCTION		AIR BONE GAP		MEAN SPEECH RECEPTION THRESHOLD	
	R	L	R	L	R	L	R	L
NL N=11	10.6	11.3	8.18	7.88	2	4	7.3	8.2
OMH N=16	10.5	10.0	6.25	6.15	4	4	6.6	9.1
AOM N=9	24.2	27.6	3.33	3.52	21	24	22.8	25.6

The data were collected in one session of approximately one and a half hours. Each subject participated in all the tests except for subject one. She participated in the otoscopic evaluation, audiological testing, tympanometric examination and the late auditory response test, but the ABR and MLR were not completed. Other missing data were due to lack of replicability of results which may have been due to poor test conditions or to the absence of responses.

The subjects' age was held constant at four to five years. The NL group consisted of children with minimal history of otitis media, no reports of medical problems with the ears, and normal hearing. The OMH group had previous history of otitis media and were treated with tubes and frequent medical follow-up. The OMH children were clear of otitis media at the time of the test and had normal hearing. The AOM group were diagnosed with otitis media on the day of the test and had an average hearing loss of 24.2 dB for the right ear and 27.6 dB for the left ear.

METHOD AND INSTRUMENTATION

General procedures are presented in the initial section with specific procedures for each of the auditory evoked potentials discussed in separate sections. The ABR, MLR, and the LAR were measured utilizing the Nicolet Compact Four (Model CA 2000) Electrodiagnostic equipment with Telephonics TDH 39P earphones. The electrodes were routed to the CA2000 using the ET 403 Biomedical head box. Waveforms were replicated and stored on a floppy disk for analysis. Reliability was assessed by comparing two samples of trials recorded from the same subjects under comparable conditions. No behavioral response was required from the subject.

The intensity levels selected for this study were 75 dB nHL for the NL and OMH group and 85 dB nHL for the AOM. The intensity adjustment for the children with auditory thresholds greater than 16 dB would help in the

identification of the target waves and possibly compensate for attenuation caused by the otitis media. At higher intensity levels a post auricular reflex could be triggered interfering with the identification of the MLR component at the 12 msec latency range. It is important to realize that changes in levels of cochlear stimulation influence the latency of the earlier ABR and the MLR waves (Jacobson, 1985; Picton, Hillyard, Kraus, Galambos, 1976; Hall, 1992).

Stimulus intensity has not been a significant factor influencing the latency of the late potentials (Goodin, Squires, and Starr, 1983; Squires and Hecox, 1983). The cortical responses can even be elicited in the absence of sensory stimulation if the signal is an omitted click in a train of clicks presented at equal intervals. In this case, the P300 occurs as expected, about 300 msec following each omitted click, but is not preceded by brainstem or late evoked potential waves (Courchesne, 1979).

Electrode locations for the auditory evoked potentials followed the International 10/20 system (Jasper, 1958). The 10/20 locations were abraded with OMNI ECG and EEG skin prepping paste to reduce the impedance of the skin and Medicotest Blue Sensor disposable electrode pads were placed in the appropriate positions. The impedance was measured utilizing the CA2000 internal impedance check. Mean electrode impedance was under 10k ohms with no more than 5k ohms between the electrodes. The recordings were completed with the patient reclining comfortably with the head fully supported.

Evoked Potential Stimulus Recording Systems

Auditory Brainstem Response

The ABRs were recorded in response to 1500 click stimuli presented with alternating polarity at 11.4 clicks per second. The standard electrode configuration was utilized

for the recording of the ABR. The positive electrode was placed on vertex (Cz), the negative electrode was placed on the mastoid process of the test ear (A1 and A2), and the ground was forehead (Fpz). The bandpass filter settings were 150 and 3000 Hz. The analysis time of 10 msec was selected.

Middle Latency Response

The MLRs were recorded in response to click stimuli presented with alternating polarity at 11.4 clicks per second. The analysis time window of 80 msec was selected. The electrode sites were the vertex (Cz) as positive, the forehead (Fpz) as common and the mastoid placement (A1 and A2) as negative. Kileny (1983) reported that narrow filtering of the brain's electrical activity could introduce into the average MLR waveform non existing components, the product of filter distortion. The bandpass filter settings were 10 and 1000 Hz. The slower rate of 11.4 stimulus /second was utilized to avoid rate effects identified by Kraus, Smith, Reed, Stein, and Cartee (1985). In children, the increase in stimulus rate reduced the identification of the MLR waveform.

Late Auditory Responses

The primary requirements necessary to elicit the LAR are that the subject must process stimulus information and that on some occasions the stimulus information is some way "unexpected" (Squires and Hecox, 1983). The frequent versus rare stimuli are distinguished by a difference in frequency (Goodin, Squires, Henderson, and Starr, 1978; Goodin, Squires, and Starr, 1983; Hillyard, Hink, Schwent and Picton, 1973). The frequencies selected for this study were 750 Hz and 2000 Hz. The frequency difference has been used to elicit late auditory potentials in children (Squires and Hecox, 1983; Martin, Barajas, Fernandez, and Torres, 1988; Polich, 1986).

The N1, P2, N2, and P3 responses were recorded utilizing an oddball paradigm. The frequently occurring (80%) stimulus was a 750 Hz tone and the rarely occurring (20%) stimulus was a 2000 Hz tone. The analysis window selected was 800 msec. The electrode sites included P3 and P4 as positive, the Fpz as common and A1 and A2 as negative. The late auditory responses have been shown to have larger amplitude at midline and parietal regions (Courchesne, 1983). This electrode configuration allowed recording from each hemisphere when a binaural stimulus was presented. The binaural stimulus presentation allowed the examiner to reduce the time of testing for the cortical response.

The bandpass filter settings were 1 to 30 Hz. Random eye movements could not be directly monitored due to equipment limitations. The filter bandpass setting from 1-30 Hz has been shown to be sufficient to record the auditory evoked potentials and at the same time narrow enough to help reduce artifact from eye movements (Jirsa, 1990). The subjects watched a video screen during the testing to provide visual fixation (Rockstroh, Elbert, Birbaumer, and Lutzenberger, 1982; Jirsa, 1990). The visual task has been shown to eliminate oculographic artifacts in adults (Sklare and Lynn, 1984). The subjects were alert throughout the recording. The responses to the frequent stimulus and the responses to the rare stimulus were collected in separate memory blocks.

Response Evaluation for the Auditory Evoked Responses

The presence or absence of waveforms of the auditory evoked response was judged on the basis of the characteristic appearance of adult waves. The response was considered abnormal or absent if it showed atypical waveform morphology or if replication of the waves could not be obtained. Visual peak identification was used for data analysis (Callaway, Halliday, and Herning 1983). Measurement of latency was

computed from onset of the stimulus to the highest peak on the wave. If the peak was not obvious, latency was estimated using the intersection of lines of "best fit" on either side of the component peak (Finley, 1985). If the peak was bifid, the largest of the two peaks was selected as the point to determine the latency.

Auditory Brainstem Response

ABR measurements included absolute latencies for waves I, III, and V, as well as interwave interval calculations for I-III, III-V, and I-V. Wave I was identified as the absolute positive peak that had the most positive point between 1.5 and 2.5 msec. Wave III was identified as the absolute positive peak that had the most positive point between 3.3 and 4.8 msec. Wave V was identified as the absolute positive peak that had the most positive point between 5.3 and 6.5 msec. The interwave interval calculations were the differences in latency between waves I and III, III and V, and I and V.

Middle Latency Response

MLR measurements included absolute latency for Na, Pa and Nb. The MLR wave forms were identified by peak identification. The characteristic MLR waveform appearance included a broad negative trough (Na) followed by a vertex-positive Pa component. Na was defined as an absolute negative peak that had the most negative point between 8 and 30 ms. Pa was defined as an absolute positive peak that has the most positive point between 20 and 50 ms.

Late Auditory Response

The LAR measurements included the latency for N1, P2, N2, and P3. The latency for N1 was identified as the largest negative trough between 60 and 250 msec and P2 latency was identified as the highest positive peak between 130 and 350

msec following N1. Latency for N2 was identified as the largest negative trough following P200 between 250 and 500 msec. The P3 latency was identified as the largest amplitude positive peak between 300 and 800 msec following N2.

DATA ANALYSIS

The first objective in all waveform analysis is confirmation that a response is present. ABR and MLR waveform repeatability consisted of two or more averaged waveforms, when overlaid, that were very similar. The presence or absence of waves was judged on the basis of the characteristic appearance of adult waveforms and the criterion latency range that was described earlier.

For present responses, latency was measured from onset of the stimulus to the highest peak on the wave. Each wave identified (I, III, V, Na, Pa, N1, P2, N2, and P3) was treated as a separate experiment for the purpose of clarity of description and subsequent analysis. The interwave intervals of I-III, III-V, and I-V were calculated. Data were reported in terms of each group latency of the auditory evoked potentials that yielded mean scores. The differences among these means that represented each groups performance were analyzed using an Analysis of Variance (Winer, 1971). A pre-determined level of significance accepted for this study was .05. A model that would incorporate analysis of both the left and the right ear together would have been more appropriate. Due to the small sample size and missing data it was not possible to use that model for analysis. A one way analysis of variance for the right and for the left ear data was used. The number of subjects used in the analysis for the evoked potentials varied due to missing data. The middle and late potentials were often absent for some children. This caused a difference in the numbers of subjects reported and the number of data points analyzed for significant differences between the groups.

CHAPTER IV

RESULTS

The results of the waveform identification and the statistical comparisons between group means are presented in this chapter. A summary of the individual data may be found in Appendices A, B, and C.

RELIABILITY RESULTS

Reliability measures were taken for the audiometric test and the evoked potentials. The 1000 Hz tone was re-tested to establish test re-test reliability for the pure tone testing. The test was considered reliable if the re-test at 1000 Hz was within plus or minus 10 dB of the original threshold. All the children responded within plus or minus 10 dB of the original threshold. The speech reception thresholds (SRT) were obtained as a reliability check for the air conduction testing. Reliability of the subject's responses to the auditory evoked potentials was assessed by repeating the stimuli a second time to all the subjects. The responses from the second administration were compared to the initial responses. A response was considered reliable if the responses waveforms could be superimposed. Only repeatable waveforms were considered a response for the auditory evoked responses.

The audiometric data were considered reliable for all children. The test re-test at 1000 Hz and the SRT were within plus or minus 10 dB of the original 1000 Hz Threshold for all children. The auditory evoked potentials that were included in the analysis contained responses that were

replicated. All other responses were not considered reliable and were not used in the calculations.

AUDITORY BRAINSTEM RESPONSE RESULTS

Determination of presence of the waves were based on the following criterion. The waveforms had to have the characteristic peaks within the specified latency range and the replication of the waves must be very similar when they are superimposed. The ABR waveform identification and replication were good for all subjects. Typical ABR waveforms for the children in the groups are shown in Figure 2. The main components as shown in Figure 2 are waves I (1.5-2.5 msec), III (3.3-4.8 msec), and V (5.3-6.5 msec). The children in the NL group and the OMH group had all the waves present with good replication. The children in the AOM group had good replication of the wave forms with slight variations in the morphology. The wave IV and V were more fused in the AOM group.

The data analyzed included latency measures of waves I, III, and V. In addition the interwave interval for I-III, III-V, and I-V were calculated. The analyses were carried out separately for each ear for the absolute latency of waves, I, III, V and the interwave intervals of I-III, III-V, and I-V. The group scores were analyzed statistically using a one way analysis of variance (ANOVA) to determine if there was nonchance variation in any of the means of the latency measurements at a .05 level of significance.

The group mean latencies and standard deviations for waves I, III, and V were calculated and are shown in Table 2. The means for the NL and the OMH group were essentially the same. The mean latencies for waves I, III, and V were longer for the AOM group (means I = 2.08 msec for the left ear and 2.05 msec for the right ear; means III = 4.27 msec for the right and left ear; means V = 6.16 msec for the left ear and 6.0 msec for the right ear) compared to the NL and the OMH

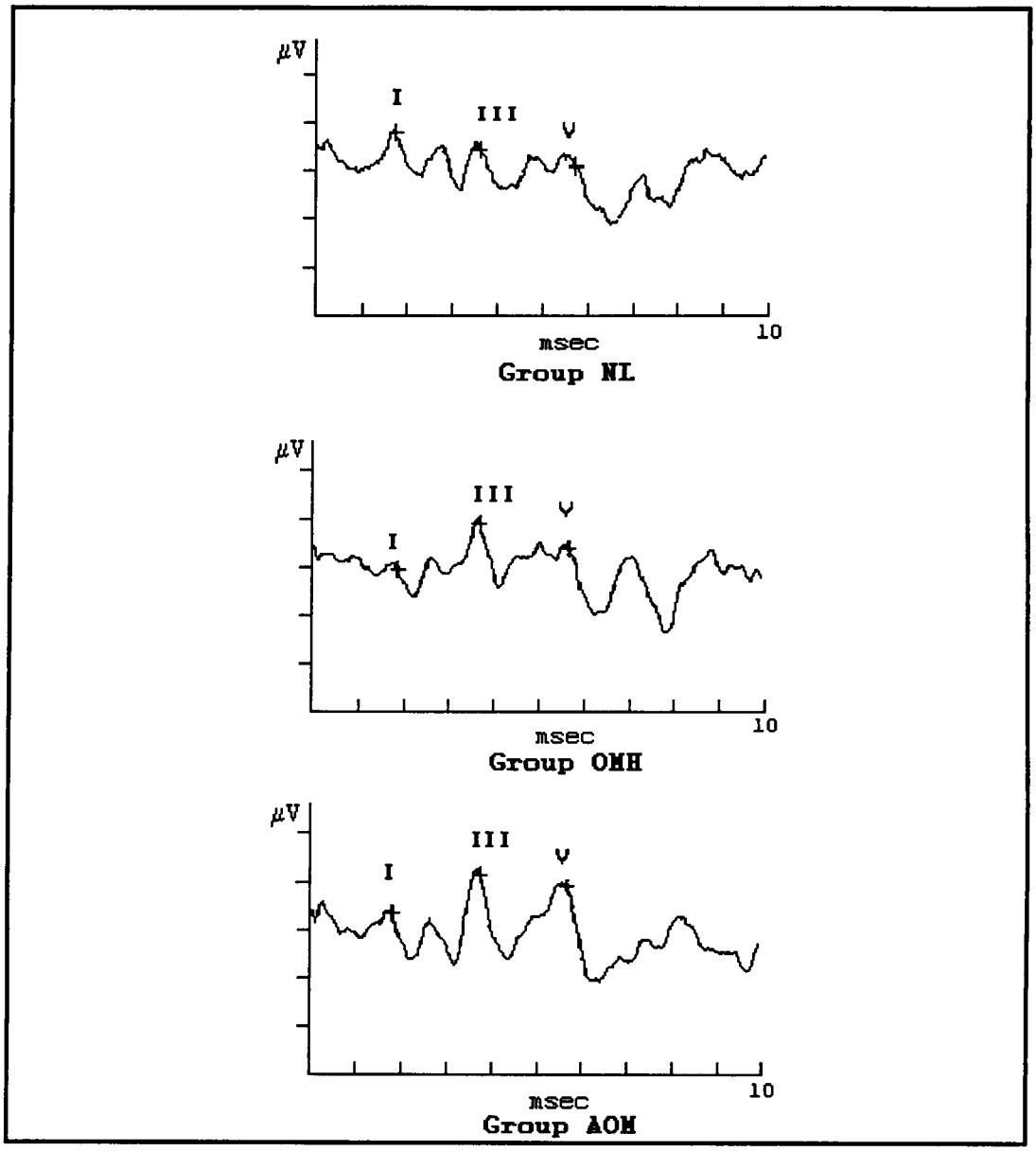


Figure 2. Representative Auditory Brainstem Responses for each Group. Wave I, III, and V are labeled. Groups are minumal history of otitis media (NL), history of media (OMH), and active otits media (AOM).

Table 2. Means and Standard Deviations for ABR Waves I, III, and V by Group and Ear. The measures are latency in milliseconds. The groups include Minimal History (NL), Otitis Media History (OMH), and Active Otitis Media (AOM).

		I		III		V	
Group	Measure	Left	Right	Left	Right	Left	Right
NL	Mean	1.840	1.872	3.960	3.884	5.804	5.928
N=10	SD	0.154	0.165	0.206	0.165	0.236	0.164
OMH	Mean	1.803	1.843	3.915	3.923	5.915	5.923
N=16	SD	0.123	0.119	0.270	0.198	0.340	0.235
AOM	Mean	2.080	2.053	4.271	4.271	6.160	6.000
N=9	SD	0.302	0.295	0.490	0.409	0.464	0.415

group (mean I = 1.8 msec for the left ear and 1.87 msec for the right ear; means III = 3.96 msec for the left ear and 3.88 msec for the right ear; means V = 5.8 msec for the left ear and 5.93 msec for the right ear).

The mean latency and standard deviations for the interwave intervals are shown in Table 3. The group scores were analyzed statistically using a one way analysis of variance (ANOVA) to determine if there was nonchance variation in any of the means of the latency measurements at a .05 level of significance.

The mean latencies for the interwave intervals I-III for the NL and OMH groups were essentially the same at about 2.116 msec for the left ear and 2.012 msec for the right ear. The AOM group means were shorter for the interwave interval I-III (2.06 msec for the left ear and 2.076 msec for the right ear) compared to the NL and OMH groups. The interwave interval III-V mean latencies for the NL and OMH groups were about 1.836 msec and 2.044 msec for the left and right ears respectively. The mean latencies for the interwave interval III-V were shorter for the AOM group (1.889 msec for the left ear and 1.729 msec for the right ear) compared to the other

groups. The interwave interval I-V mean latencies for all three groups were similar at 3.960 msec to 4.113 msec for the left and right ears respectively. As shown in Table 2, the standard deviations were similar between the three groups.

Table 3. Means and Standard Deviations for ABR Interwave Intervals of I-III, III-V, and I-V by Group and Ear. The measures are latency in milliseconds. The groups include Minimal History (NL), Otitis Media History (OMH), and Active Otitis Media (AOM).

		I-III		III-V		I-V	
Group	Measure	Left	Right	Left	Right	Left	Right
NL	Mean	2.116	2.012	1.836	2.044	3.960	3.756
N=10	SD	0.168	0.166	0.186	0.118	0.249	0.960
OMH	Mean	2.113	2.080	2.000	2.000	4.113	4.080
N=16	SD	0.216	0.137	0.212	0.179	0.257	0.206
AOM	Mean	2.060	2.076	1.889	1.729	3.955	3.947
N=9	SD	0.156	0.393	0.120	0.361	0.159	0.202

Tables 4 and 5 present a summary of the analysis of variance (ANOVA) and the Scheffe' post hoc test for the latency measures of waves I, III, and V. The ANOVA indicated a statistically significant difference between groups for each ear for waves I and III. The Scheffe' procedure was used to test differences between group pairs at a .05 level of significance. The Scheffe' follow-up analysis revealed that the mean latency for waves I and III for the right and left ear for the NL were significantly different from the mean latency of these waves for the group with AOM. The mean latency for waves I and III for the right and left ear for the OMH group were significantly different from the latency of these waves for the group with active otitis media.

It can be seen in Table 2 that the mean latencies for the NL and the OMH groups were shorter than the mean latencies for the AOM group. The prolongation in the latency for wave I for the AOM group compared to the NL group was .24 msec for the left ear and .18 msec for the right ear. The latency shift for wave I between the AOM and OMH group were similar at .28 msec and .21 msec for the left and right ear respectively. The prolongation in the latency for wave III for the AOM was .31 (left ear) and .39 (right ear) when compared to the NL group and .36 (left ear) and .35 (right ear) when compared to the OMH group.

Table 4. Summary of ANOVA to test for differences in mean latency for the ABR waves I, III, and V between the minimal history of otitis media (NL), otitis media history (OMH), and active otitis media (AOM) groups. The right and left ear were calculated separately. The significant F values are marked with an asterisk.

LEFT EAR				RIGHT EAR			
SOURCE	**df**	**MS**	**F**	**Source**	**df**	**MS**	**F**
ANOVA for the ABR Wave I							
Between Groups	2	0.216	6.183*	Between Groups	2	0.136	3.756*
Within Groups	31	0.035		Within Groups	32	0.036	
ANOVA for the ABR Wave III							
Between Groups	2	0.390	3.677*	Between Groups	2	0.446	6.572*
Within Groups	32	0.068		Within Groups	32	0.068	
ANOVA for the ABR Wave V							
Between Groups	2	0.315	2.542	Between Groups	2	0.019	.249
Within Groups	32	0.124		Within Groups	32	0.077	

Table 5. Summary of Scheffe' Post Hoc test for differences between group pairs for the ABR waves I and III for the minimal history (NL), otitis media history (OMH), and active otitis media (AOM) groups. The right and left ear were calculated separately. The significant F values are marked with an asterisk.

Scheffe' Comparison Between Group Pairs: Wave I					
Hypothesis	**LEFT EAR**	**Critical Value**	**Hypothesis**	**RIGHT EAR**	**Critical Value**
NL-OMH=0	0.5304	2.5690	NL-OMH=0	0.5230	2.5652
AOM-NL=0	2.7920*		AOM-NL=0	2.7530*	
AOM-OMH=0	3.5920*		AOM-OMH=0	3.5418*	
Scheffe' Comparison Between Group Pairs: Wave III					
NL-OMH=0	-0.3710	2.5690	NL-OMH=0	0.4281	2.5652
AOM-NL=0	3.2300*		AOM-NL=0	2.5957*	
AOM-OMH=0	3.2028*		AOM-OMH=0	3.2765*	

Tables 6 and 7 present a summary of the ANOVA and the Scheffe' post hoc test for the latency measures of the interwave intervals I-III, III-V, and I-V. A significant difference between groups was shown for the III-V interwave interval for the right ear. The Scheffe' follow-up analysis revealed that the mean latency of the interwave interval III-V for the right ear for the NL and the OMH groups were significantly different from the latency for the AOM group.

The latency of the interwave interval III-V for the right ear was shorter for the AOM group when compared to the NL and the OMH groups. The latency difference was .315 msec for the NL group and .271 msec for the OMH group. No other interwave interval differences were identified as significant.

Table 6. Summary of ANOVA to test for differences in mean latency for the ABR interwave intervals I-III, I-V, and III-V between the minimal history of otitis media (NL), otitis media history (OMH), and active otitis media (AOM) groups. The right and left ear were calculated separately. The significant F values are marked with an asterisk.

LEFT EAR				RIGHT EAR			
Source	**df**	**MS**	**F**	**Source**	**df**	**MS**	**F**
ANOVA for the ABR Interwave Interval I-III							
Between Groups	2	0.009	.246	Between Groups	2	0.016	.287
Within Groups	31	.036		Within Groups	32	0.055	
ANOVA for the ABR Interwave Interval I-V							
Between Groups	2	0.101	1.818	Between Groups	2	0.323	1.116
Within Groups	31	0.056		Within Groups	32	0.290	
ANOVA for the ABR Interwave Interval III-V							
Between Groups	2	0.090	2.628	Between Groups	2	0.283	5.488*
Within Groups	32	0.034		Within Groups	32	.052	

Table 7. Summary of Scheffe' Post Hoc test for differences between group pairs for the ABR interwave interval III-V between the minimal history of otitis media (NL), otitis media history (OMH), and active otitis media (AOM) groups. The right and left ear were calculated separately. The significant F values are marked with an asterisk.

Scheffe' Comparison Between Group Pairs: Wave III-V Interwave Interval of the ABR					
Hypothesis	**LEFT EAR**	**Critical Value**	**Hypothesis**	**RIGHT EAR**	**Critical Value**
NL-OMH=0	NA	NA	NL-OMH=0	0.4787	2.5652
AOM-NL=0	NA		AOM-NL=0	3.0064*	
AOM-OMH=0	NA		AOM-OMH=0	2.8522*	

MIDDLE LATENCY RESPONSE RESULTS

Determination of presence of the waves were based on the following criterion. The waveforms had to have the characteristic peaks within the specified latency range and the replication of the waves must be very similar when they are superimposed. The typical MLR waveforms for the children in the groups are shown in Figure 3. The waves for Na, Pa, and Nb were marked. The waves that were identified consisted of a negative component in the 12-20 msec region labeled Na. The MLR component Pa was a rounded single peak followed by a negative trough. The waveform morphology and replication ranged from good to absent.

The percentage of waves that were identified (number present/number recordings) of the Na and Pa was calculated for each group. Table 8 shows a summary of the percentage of waves that were present for the MLR waves Na and Pa for the right and left ear between the groups. Waves Na and Pa were identified in 100% of the children in the NL group (N = 10). For the OMH group (N = 16), the waves were present 81.25% of the time for the right ear and 62.5% of the time for the left ear. Seven subjects of nine (77.78%) in the AOM group had repeatable waveforms. The waves for the OMH and AOM groups were less well formed. There was a trend of decrease in discernible wave components for the MLR for the OMH and the AOM groups.

For the waves that were identified the group mean latencies and standard deviations were calculated and are shown in Table 9. Poor waveform morphology and poor replication resulted in the MLR wave components being identified as absent. This resulted in missing data for the data analysis. The statistical calculations of group differences using the ANOVA were not completed due to the small data set due to missing data.

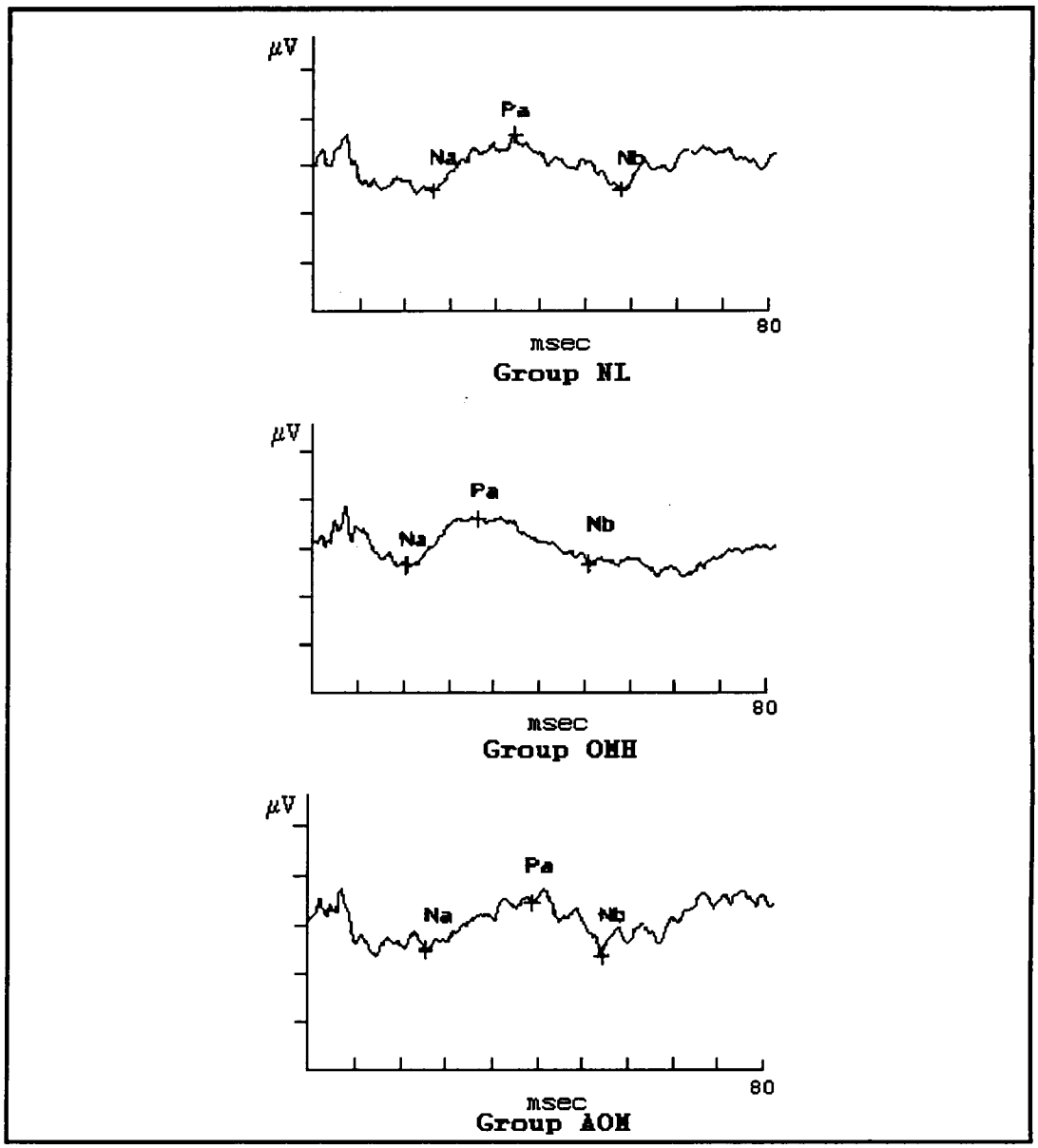


Figure 3. Representative Middle Latency Responses for each Group. Waves Na and Pa are labeled. Groups are minumal history of otitis media (NL), history of otitis media (OMH), and active otitis media (AOM).

Table 8. Percentages of waves present for the MLR Waves Na and Pa for the Minimal History (NL), Otitis Media History (OMH), and the Active Otitis Media Groups (AOM).

GROUP	NA		PA	
	LEFT	RIGHT	LEFT	RIGHT
NL N=10	100%	100%	100%	100%
OMH N=16	62.5%	81.25%	62.5%	81.25%
AOM N=9	77.78%	77.78%	77.78%	77.78%

Table 9. Means and Standard Deviations for the Middle Latency Response Na and Pa by Group and Ear. The measures are latency in milliseconds. The groups include Minimal History (NL), Otitis Media History (OMH), and Active Otitis Media (AOM).

Group	Measure	Na		Pa	
		Left	Right	Left	Right
NL	Mean	18.704	17.351	30.524	31.040
	SD	4.119	2.233	3.232	2.743
	Number	10	10	10	10
OMH	Mean	17.536	18.440	31.008	29.760
	SD	2.909	3.174	4.738	3.173
	Number	10	13	10	13
AOM	Mean	18.143	19.109	33.000	33.829
	SD	3.506	2.535	4.536	3.262
	Number	7	7	7	7

LATE AUDITORY RESPONSE RESULTS

Determination of presence of the waves were based on the following criterion. The waveforms had to have the characteristic peaks within the specified latency range and the replication of the waves must be very similar when they are superimposed. The waveform morphology of the late auditory response was fair to poor for many of the children. The representative waveforms for the children in the groups

are shown in Figure 4. The waves N1 (60-250 msec), P2 (130-350 msec), N2 (259-500 msec), and P3 (300-800 msec) were marked for the rare stimuli. The characteristic waveform exhibited three prominent components N1, P2, and N2. The P3 wave component was not easily identified for the groups due to the short time window.

The data analyzed included latency of waves N1, P2, N2, and P3. The group mean latencies and standard deviations were calculated and are shown in Table 10.

Table 10. Means and Standard Deviations of the Late Auditory Responses N1, P2, N2, and P3 by Group and Electrode Site. Groups represented are Minimal History (NL), Otitis Media History (OMH), and Active Otitis Media (AOM). The measures are latency in milliseconds.

		N1		P2		N2		P3	
Group	Measure	P3/L	P4/R	P3/L	P4/R	P3/L	P4/R	P3/L	P4/R
NL	Mean	148	158	201	214	262	265	539	529
	SD	39	47	49	60	56	68	106	99
	Number	11	11	11	11	11	11	11	11
OMH	Mean	177	179	240	242	292	298	590	570
	SD	23	28	37	46	50	63	93	83
	Number	14	14	14	14	14	14	12	13
AOM	Mean	205	205	263	258	313	316	624	630
	SD	34	22	46	26	66	40	91	83
	Number	10	10	10	10	10	10	6	7

Table 10 shows that the latencies for the AOM group were longer (means N1 = 205 for the P3 and P4 sites; means P2 = 263 for P3 and 258 for P4; means N2 = 313 for P3 and 316 for P4; means P3 = 624 for P3 and 630 for P4) compared to the NL and the OMH groups. The OMH group had longer latencies (means N1 = 177 for P3 and 179 for P4 sites; means P2 = 240 for P3 and 242 for P4; means N2 = 292 for P3 and 298 for P4; means P3 = 590 for P3 and 570 for P4) compared to the NL group.

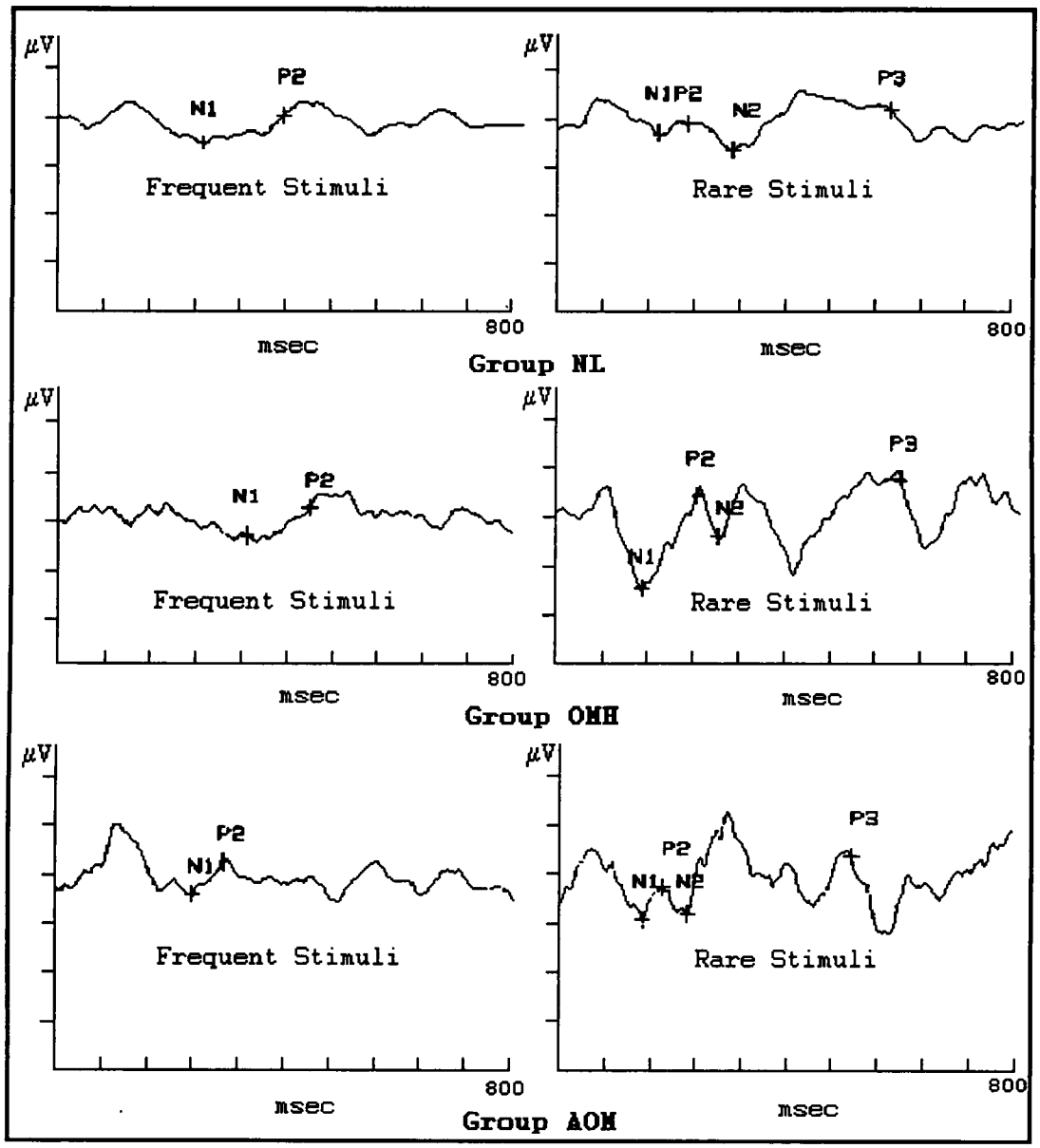


Figure 4. Representative Late Auditory Responses for the groups. Waves N1, P2, and P3 are labeled. Groups are minumal history of otitis media (NL), history of otitis media (OMH), and active otitis media (AOM).

The group latencies were analyzed statistically using a one way ANOVA to determine if there was nonchance variation in the means of the latency measurements at a .05 level of significance. Table 11 presents a summary of the ANOVA for the latency measures of waves N1, P2, N2, and P3. The results of the ANOVA analysis indicated that statistically significant differences existed between the groups for N1 for both electrode sites. A significant difference between the groups was shown for P2 for the left electrode site.

The Scheffe' procedure was used to test differences between group pairs. The results shown in Table 12 show that the latencies for the N1 for both electrode sites were significantly different for the NL group when compared to the AOM group. The latency measurements for P2 for the left electrode site were also significantly different for the NL group when compared to the AOM group. No other comparisons were found to be significant.

It can be seen in Table 10 that the mean latencies for the AOM group were longer than the mean latencies for the NL and the OMH groups. The prolongation in the latency for wave N1 for the AOM group compared to the NL group was .57 msec for the left (P3) electrode site and .47 msec for the right (P4) electrode site. The latency shifts for N1 between the AOM and OMH group were similar at 28 msec and .26 msec for the left and right electrode sites respectively. The prolongation in the latency for wave P2 for the AOM was .52 msec (P3) and .44 msec (right ear) when compared to the NL group, and .36 msec (P4) and .35 msec (P3) when compared to the OMH group. Figure 5 shows a graph of the mean latency for the waves N1, P2, N2 and P3 for each group. The graph illustrates the trend of longer latency for the auditory potentials for the OMH and AOM groups compared to the NL group. The rank ordering of the groups from NL to AOM can be seen.

Table 11. Summary of ANOVA to test for differences in mean latency for the late responses N1, P2, N2, and P3 between the Minimal History of Otitis Media (NL), Otitis Media History (OMH), and Active Otitis Media (AOM) groups.

LEFT P3				RIGHT P4			
Source	**df**	**MS**	**F**	**Source**	**df**	**MS**	**F**
ANOVA for Wave N1							
Between Groups	2	8722.5	8.581*	Between Groups	2	5903.2	5.280*
Within Groups	32	1016.5		Within Groups	32	1118.1	
ANOVA for WAVE P2							
Between Groups	2	10275.1	5.439*	Between Groups	2	5315.3	2.451
Within Groups	32	1889.0		Within Groups	32	2168.5	
ANOVA Wave N2							
Between Groups	2	6918.9	2.123	Between Groups	2	7131.3	2.050
Within Groups	32	3259.5		Within Groups	32	3479.5	
ANOVA WAVE P3							
Between Groups	2	15781.5	1.642	Between Groups	2	21653.6	2.734
Within Groups	26	9608.6		Within Groups	28	7921.3	

Table 12. Summary of Scheffe' Post Hoc test for differences between group pairs for the LAR waves N1 and P2 between the Minimal History of Otitis Media (NL), Otitis Media History (OMH), and Active Otitis Media (AOM) groups.

Scheffe' Comparison Between Group Pairs: Wave N1 of the LAR					
Hypothesis	LEFT P3	Critical Value	Hypothesis	RIGHT P4	Critical Value
NL-OMH=0	2.3238	2.5652	NL-OMH=0	1.5809	2.5652
AOM-NL=0	4.1369*		AOM-NL=0	3.2476*	
AOM-OMH=0	1.9940		AOM-OMH=0	1.7898	
Scheffe' Comparison Between Group Pairs: Wave P2 of the LAR					
NL-OMH=0	2.2258	2.5652	NL-OMH=0	NA	NA
AOM-NL=0	3.2338*		AOM-NL=0	NA	
AOM-OMH=0	1.2466		AOM-OMH=0	NA	

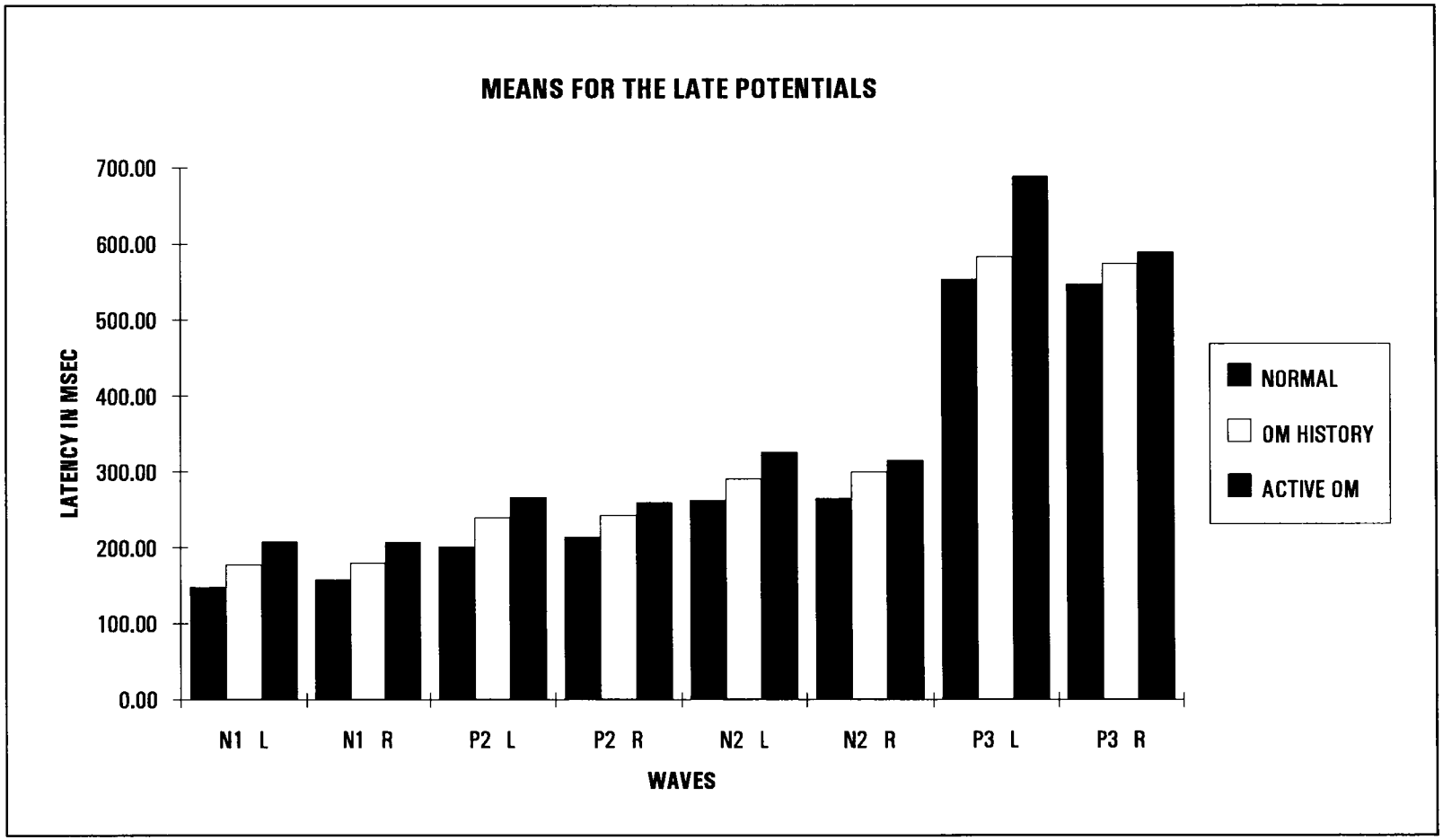


Figure 5. Graph of the Mean Latency for Waves N1, P2, N2 and P3 of the Late Auditory Responses for the Left (L) and Right (R) Electrode Sites. A trend of longer latency is shown for the responses for the OM History and Active OM groups.

The statistical analysis revealed shorter latency for the waves I and III for the NL and OMH groups when compared to the AOM group. The interwave interval latency for III-V was shorter for the AOM group when compared to the NL and OMH groups. No differences were identified between the NL and OMH groups.

Waveform replication and identification were good for the NL group for the MLR. The numbers of waves present for the OMH group were 81.25% for the right ear and 62.5% for the left ear. The wave identification was 77.78% for the AOM group. The results show that otitis media influences the morphology and replication of the waves of the MLR. No statistical analyses were completed due to the reduced data set caused by absent waveforms.

Waveform morphology varied for the LAR. A statistically significant difference was identified between the NL and AOM groups for latencies of N1 for each ear and P2 for the left ear. The wave N1 latency was longer for the AOM group compared to the NL group. The P2 latency was longer for the left ear for the AOM group compared to the NL group. No other analyses were found to be significant. A rank order trend was identified that shows that the NL group had shorter latencies with the OMH group second and the AOM with the longest latencies.

CHAPTER V

DISCUSSION AND IMPLICATIONS

The auditory evoked responses were obtained in time lines of the ABR, MLR, and LAR. These potentials were selected to cover the range of the auditory system from the cochlea to the cortex. The specific aim of this study was to compare the ABR, MLR, and LAR indices across three groups of children (NL, OMH, and AOM) to determine the effects of otitis media a form of auditory deprivation on these responses. This chapter presents discussion with implications for further research.

DISCUSSION

It was proposed that the children with history of otitis media and children with active otitis media have greater variability and longer latency for the auditory evoked potentials than children with minimal history of otitis media. A possible model for the auditory deprivation caused by otitis media could include a slowing of the maturation process of the auditory system. Identification of alteration of the auditory evoked potentials for the active otitis media children would provide evidence that the auditory system is not functioning the same as children with minimal history of otitis media. The auditory evoked potential measures may identify recovery of function in the children with history of otitis media who are now clear of infection and have normal hearing. A trend in the present study was identified that showed changes in the morphology of the waves for the MLR and the LAR and increases in the latency of some of the waves for the ABR and LAR for children with active otitis media. The

children with history of otitis media were not different from the active otitis media group or the minimal history group.

Auditory Brainstem Response

The earliest components of the auditory evoked response, ABR, have proved to be highly reliable and their minimal variability has facilitated the construction of normative tables against which subject data can be compared (Jacobson, 1985, Hall, 1992). ABR waveforms were readily identified for all subjects in this study. The statistical analysis revealed longer latency for the waves I and III for the AOM group when compared to the NL and OMH groups. The interwave interval for III-V was shorter for the AOM group for the right ear than for the NL or the OMH groups.

Though there were some significant differences in the ABR latencies, there is no clear pattern of prolongation of latencies to indicate that otitis media or history of otitis media significantly alters auditory processing in the brainstem. The expected result for the absolute latency for the ABR was an increase in the latency for waves I, III and V for the subjects in the AOM group and the OMH group when compared to the NL group. The stimulus level pre-selected as 85 dB nHL for the AOM group may have resulted in reduced cochlear stimulation as compared to that for the NL and the OMH groups. It is felt that the latency shift for the AOM represents a peripheral effect caused by the hearing loss.

If active otitis media affects the brainstem maturation and development, then it should have been reflected in the prolonged I-III or III-V interwave intervals. The shorter latency for the interwave interval III-V was difficult to explain. It was not an expected result. It has been reported that a conductive hearing loss causes a change in the absolute latency of the waves I, III, and V with no change in the interwave interval (Fria and Sabo, 1980). Minor inconsistency or error in calculation of wave latency

could mean the difference between a normal and an abnormal interpretation. A greater latency shift with declining intensity may occur for wave I or wave III than for wave V in normal adults, resulting in a shortening of the I-V latency interval. The reduction of the effective intensity at the cochlea for the AOM group could result in a shortening of the interwave interval III-V.

The prolonged absolute latency merely reflects a conductive component and reduced intensity reaching the cochlea. A prolonged latency of the interwave intervals I-III and III-V would reflect changes in the maturation or development of the brainstem. The prolonged interwave intervals were not identified in the present study. It could be that the early intervention with tubes for the OMH group allowed recovery of the brainstem responses. Since the brainstem responses mature by the age of two years, the maturation could be complete.

Group differences between the NL and the OMH groups were not identified for the present study. The lack of differences in ABR latencies for NL and OMH groups suggests that OMH does not affect brainstem processing on a permanent basis. Other retrospective studies of children with histories of otitis media have found changes in the ABR latencies. Folsom, Weber, and Thompson, (1983) identified prolonged absolute wave III and V and interpeak I-V latencies for the group with otitis media history when compared to their controls. The children were otologically clear and had normal hearing sensitivity at the time of the test. The children who participated in the study completed by Folsom et al (1983) were 6 to 10 years of age. Lenhardt, Shaia and Abedi (1985) reported abnormal delays in the latencies of waves III and V and prolonged I-III interwave intervals for two children with histories of recurrent otitis media at age five. Chambers, Rowan, Matthies, and Novak, (1989) found group differences for the absolute wave III latencies and I-

III interpeak latency between children with histories versus those without. Chambers et al (1989) tested the otitis media history group four months after tube placement for otitis media with effusion. Gunnarson and Finitzo (1991) studied prospectively three groups of children age five years to seven years. Hearing levels and otitis history were documented from birth. The normal group had normal ABR at infancy. Group B had wave V present but prolonged in latency and group C had absent wave V at 30 dB. The controls differed from both groups with early hearing loss for wave III and wave V latencies and interpeak I-III and III-V latencies.

Only one study did not identify latency differences for children with histories of otitis media. Seikel, Brandt, and Ferraro (1989) did not find a group difference in the ABR latencies between normal hearing subjects and subjects with early histories of otitis media. The subjects who participated in the study completed by Seikel et al (1989) were seven years of age and medically clear of otitis media for an average of 52 months.

The variety of ABR results for studies using children with histories of otitis media may be related to the fluctuating nature of otitis media and the variation in the amount of conductive hearing loss that each child experiences (Bess, 1985; Fria, Cantekin, and Eichler, 1985). The length of time with otitis media and the last episode would be important variables. The children in the OMH group for the present study represent a sub-group of children with history of otitis media. They had episodes of otitis media before age one and they had received aggressive treatment (myringotomies and tubes) with frequent follow-up medical care. The lack of significant differences for the OMH group suggest that the medical management of the otitis media may have prevented residual effect of the auditory deprivation at the level of the brainstem.

Middle Latency Response

In the present study, MLR waveform replication and identification for Na and Pa were good for the subjects in the NL group. The waveform replication and identification were fair to absent for the subjects in the OMH and AOM groups. Waves Na and Pa were consistently identified in all the children (100%) in the NL group (N = 10). For the OMH group (N = 16), the waves were present 81.25% of the time for the right ear and 62.5% of the time for the left ear. Seven subjects of nine (77.78%) in the AOM group had repeatable waveforms for both ears. No statistical analyses were completed due to the reduced data set caused by absent waveforms. The mean data available showed no observable differences. Kraus, Smith, Reed, Stein, and Cartee (1985) found that the presence or absence of MLR components Na and Pa varied systematically with age. The probability that identifiable responses are obtained decreases with age. One possible explanation of the present MLR results is that the OMH and AOM groups are similar to those of younger children. One possible explanation of the present results is that the OMH and AOM group MLR results are similar to those of younger children. If this were so, then the effects of OM on the MLR would be the same as delay maturation.

The MLR results suggested that the effect of otitis media may not be restricted to changes in latency. Factors that influence waveform morphology include stimulus intensity and maturity of the auditory pathway. The AOM group had a hearing loss for which there may not have been adequate compensation. However, the OMH group did not have a change in the hearing sensitivity when they were compared to the NL group. The waveform morphology for the OMH group was poor and the percentage of present waves was lower than for the NL group. This would suggest that the hearing loss was not a factor in the change in the morphology. Maturity of the

auditory system should not be a factor because the age of the subjects was controlled.

Gunnarson and Finitzo (1988) concluded that the MLRs were influenced by early otitis media. The children, age five to seven, with early otitis media had a lower percentage of detectable Pa waves than normal controls (Gunnarson and Finitzo, 1988). Their findings indicated that the responses remained altered even after resolution of early conductive problems.

The alterations in the MLR response may not be limited to the traditional measures of latency. The trend identified in the current study suggested that otitis media reduced the number of discernible waves of the MLR. The effect of otitis media continues even with the resolution of the hearing loss. A larger data set would be required to assess changes in waveform morphology that were identified for the MLR. The MLR waveform morphology show an influence of the auditory deprivation caused by otitis media. The effect of the auditory deprivation has remained for the OMH group even though they have been aggressively treated from an early age.

Late Auditory Response

Even in subjects who are normal audiologically and neurologically, the late auditory responses vary substantially (Duncan-Johnson and Donchin, 1982; Musiek, Verkest, Gollegly, 1986). In addition to individual differences, direct comparison across studies for the late potentials is not always possible due to different methodological approaches.

The late auditory evoked responses of N1, P2, and N2 were present for all subjects. Wave P3 was not consistently identified in any of the groups. Two significant differences were identified. Wave N1 was longer in latency for the AOM group for both electrode sites when compared to the NL group. Longer latency was identified for P2 for the left electrode

site for the subjects in the AOM group than for the subjects in the NL group. No other analyses were found to be significant. Figure 5 (page 40) shows a consistent rank ordering of the three groups. The AOM group had longer latencies for all of the LAR waves than the other groups. The OMH group had longer latencies than the normal group. The direction of the ranking was consistent with the predicted change as the result of the auditory deprivation caused by otitis media.

When events occurred which were not explicitly pre-categorized and in which each subject uses his/her own internal rules to categorize the rare or frequent stimuli, subjects of different ages produced responses dominated by different waves. N1 and P2 waves were the most prominent waves in infants and children, while P3 waves were the most prominent waves in adults (Courchesne, 1978). The N1 and P2 waves were dominate for the children in this study. However, Martin, Barajas, Fernandez, and Torres (1988) found the N1 and P2 component of the late auditory responses to be the most unreliable components in children age range from 6-14 years.

It has been proposed that the N1 wave reflects early stimulus selection process (Hillyard and Picton, 1987; Picton and Hillyard 1974). An increase in latency for N1 of the LAR would not have been expected since N1 represents acoustic perception. In the present study, differences in the latency for the N1 between the NL group and the AOM group may represent differences in ability to attend to an auditory stimuli. Feagans (1987) proposed a model of the effects of otitis media in which the degraded auditory stimulation leads to poorer attention to auditory signal. Gravel (1992) expanded on this suggesting that when auditory deprivation occurs during critical periods of communication development, the foundation for higher order listening skills, in addition to attention, may not be firmly established.

The P2 wave represents early manifestation of processes related to the categorization (perception) of the rare events (Squires and Hecox 1983). The present study identified a significant difference for the left electrode site between the NL and the AOM groups. It was thought that the significant change for the P2 for the left hemisphere site only could be related to differences in the pure tone thresholds between the right and left ear for the AOM group. The mean threshold for the left ear was 27.6 dB compared to the 24.2 dB for the right ear. The mean latency by electrode site from Table 8 (page 35) shows that the AOM group had a five msec difference between left and right (263-258 msec). This would suggest that the difference in the pure tone thresholds would not have contributed to the latency difference for the left electrode site.

The N2 latency is related to the duration of stimuli that is a reflection of a nonspecific mismatch process, such as the orienting reflex (Squires and Hecox 1983). No significant differences were identified for the N2 component of the late auditory responses between the groups. The P3 was not consistent within this age group. The presence of the P3 component provided evidence that the subject was attending to the stimulus sequence. The latency range for the P3 for the children in the four year old age range has not been clinically identified. The P3 component has been related to the timing of stimulus evaluation processes (Hillyard and Picton, 1987). The time window of 800 msec may not be sufficient to identify normal variations in stimulus evaluation for the P3 component for four year old children. Absences of a P300 component may be related to transient subject inattention to the rare stimulus, rather than to the subject's inability to attend selectively to auditory stimuli.

The dominance of the N1 and P2 waves suggest an immature auditory system. The increase in the N1 latency may suggest

that the development of attending skills may be delayed for the children in the AOM group. The increase in the latency for P2 for the left hemisphere site may represent difficulty in categorization of differences between the two tones. The P3 response was not consistently identified for these children. The time window could be too short to allow for identification.

IMPLICATIONS

It is suggested that the compromise of the normal development and maturation of the peripheral brainstem, mid-brain and cortical structures along the auditory pathways (Clopton and Silverman, 1977) could be identified using the auditory evoked potentials. The pattern of results for the ABR were not clearly supportive of alterations in the brainstem. The changes in the absolute latency suggest inadequate stimulation at the cochlea. Shortening of the interwave intervals for the III-V were also consistent with the reduction of effective stimulation. The pattern of lower percentage of identification of Na and Pa for the MLR suggest that the active otitis media and history of otitis media may affect the development of the associated mid brain and auditory cortex. The trend identified for the LAR shows that the time that it takes a stimulus to be perceived is longer for the OMH group and even longer for the AOM group. A recovery appears to occur for the children in the OMH group.

Factors that contribute to the variability and increased latency of the auditory potentials include unmyelinated fibers, smaller axonal diameter and incomplete synaptic connection, all of which reduce synaptic efficiency in the central auditory system (Eggermont, 1985; Fria and Doyle, 1984). The auditory system is functional well before birth with the neuromaturation continuing into the second or third year of life (Fria and Doyle, 1984; Moller and Jennetta, 1985). The last step in the development of the nervous

system is completion of myelin sheaths around the processes and cell bodies of the neurons. With completion of this process, axons reach their maximal velocity of conduction representing the mature state (Willard, 1990). The ascending sequence of myelination in the brainstem is shown with the temporal sequence of maturation of the evoked potentials peak I before peak III, followed by peak IV etc. (Willard, 1990).

Though, there were some significant differences in the ABR latencies, there is no clear pattern of prolongation of latencies to indicate that otitis media or history of otitis media significantly alters auditory processing in the brainstem. It was felt that the latency shift for the AOM represents a peripheral effect caused by restricted stimulation at the cochlea. It is felt that the latency shift for the AOM represents a peripheral effect caused by the hearing loss.

One possible explanation of the present MLR results is that the OMH and AOM groups are similar to those of younger children. The Pa and Na morphology for younger children is poorer and the percentage of wave forms that can be identified increases as age decreases (Kraus et al 1985). If this were so, then the effects of OM on the MLR would be the same as delay maturation.

The dominance of the N1 and P2 waves suggest an immature auditory system (Courchesne, 1978). The morphological changes and the latency increase may represent immaturity of the auditory system caused by lack of stimulation. It is speculated that MLR and the LAR waveform variability identified in this study may be related to the interruption of the myelination process resulting in a possible slowing of the processing. This might be studied longitudinally in children with chronic otitis media.

The evoked potentials also provided information about recovery of function after alterations have occurred. The children in the OMH group shared characteristics with both

the AOM group and the NL group. This suggested that time that the children have been otitis free could allow partial recovery of function. Sensory deprivation effects may be reduced because of the aggressive treatment that had been provided.

It is unknown at this time which (if any) of the children tested in this study might experience difficulty with speech, language, or auditory skills due to the otitis media history. A follow up study of the children in this study would provide information concerning the development of the auditory evoked potentials. A collaboration with a speech-language pathologist for evaluation of the linguistic abilities of the children should be included.

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APPENDIXES

APPENDIX A

INDIVIDUAL DATA FOR THE AUDITORY BRAINSTEM RESPONSE WAVES I, III, V AND THE INTERWAVE INTERVALS FOR I-III, III, V, AND I-III

		AUDITORY BRAINSTEM RESPONSE DATA SUMMARY													
	GROUP	SUBJECT #	SEX	I L	I R	III L	III R	V L	V R	I-III L	I-III R	I-V L	I-V R	III-V	III-V
1	NL	ONE	F												
2	NL	FIFTEEN	F	1.72	1.84	3.72	3.76	5.60	5.64	2.00	1.92	3.88	3.80	1.88	1.88
3	NL	TWENTY EIGHT	F	1.72	1.72	3.68	3.72	5.64	5.80	1.92	2.00	3.88	4.08	1.96	2.08
4	NL	TWENTY NINE	F	1.96	2.00	4.08	4.00	5.92	6.04	2.12	2.00	3.96	4.04	1.84	2.04
5	NL	FOURTEEN	M	1.88	1.84	4.12	4.00	5.88	6.20	2.24	2.16	4.00	4.36	1.72	2.20
6	NL	EIGHTEEN	M	1.96	1.92	4.12	3.96	6.08	6.00	2.16	2.04	4.12	4.08	1.92	2.04
7	NL	SIXTEEN	M	1.84	1.96	3.92	3.96	5.32	5.96	2.08	2.00	3.48	4.00	1.40	2.00
8	NL	NINETEEN	M	1.76	1.72	3.68	3.56	5.72	5.72	1.92	1.84	3.96	4.00	2.04	2.16
9	NL	TWENTY THREE	M	1.76	1.72	4.20	4.08	6.08	5.96	2.44	2.36	4.32	4.24	1.88	1.88
10	NL	TWENTY SEVEN	M	1.64	1.76	3.92	3.80	5.92	6.00	2.28	2.04	4.28	4.24	2.00	2.20
11	NL	THIRTY	M	2.16	2.24	4.16	4.00	5.88	5.96	2.00	1.76	3.72	3.72	1.72	1.96
	GROUP	SUBJECT #	SEX	I L	I R	III L	III R	V L	V R	I-III L	I-III R	I-V L	I-V R	III-V	III-V
12	OMH	TWO	F	1.92	1.92	3.80	3.96	6.04	6.04	1.88	2.04	4.12	4.12	2.24	2.08
13	OMH	THREE	F	1.80	1.84	3.88	3.76	5.56	5.40	2.08	1.92	3.76	3.56	1.68	1.64
14	OMH	FIVE	F	1.48	1.76	3.48	3.84	5.24	5.76	2.00	2.08	3.76	4.00	1.76	1.92
15	OMH	THIRTY TWO	F	1.80	1.88	4.00	3.96	6.04	5.96	2.20	2.08	4.24	4.08	2.04	2.00
16	OMH	SIX	M	1.76	1.88	4.04	4.24	5.92	6.16	2.28	2.36	4.16	4.28	1.88	1.92
17	OMH	SEVEN	M	1.92	1.88	4.00	3.88	6.32	6.16	2.08	2.00	4.40	4.28	2.32	2.28
18	OMH	EIGHT	M	1.76	1.84	3.76	3.68	5.84	5.72	2.00	1.84	4.08	3.88	2.08	2.04
19	OMH	NINE	M	1.76	1.64	3.72	3.64	5.60	5.76	1.96	2.00	3.84	4.12	1.88	2.12
20	OMH	TEN	M	1.88	1.72	4.52	3.84	6.20	6.04	2.64	2.12	4.32	4.32	1.68	2.20
21	OMH	ELEVEN	M	1.72	1.76	3.88	3.92	5.88	6.16	2.16	2.16	4.16	4.40	2.00	2.24
22	OMH	TWELVE	M	1.96	1.96	4.40	4.12	6.76	6.08	2.44	2.16	4.80	4.12	2.36	1.96
23	OMH	THIRTEEN	M	1.84	1.84	3.60	3.68	5.84	5.80	1.76	1.84	4.00	3.96	2.24	2.12
24	OMH	FOUR	M	1.64	1.68	3.72	3.84	5.76	5.64	2.08	2.16	4.12	3.96	2.04	1.80
25	OMH	TWENTY ONE	M	1.96	1.96	4.00	4.12	5.96	6.20	2.04	2.16	4.00	4.24	1.96	2.08
26	OMH	THIRTY ONE	M	1.80	1.80	3.76	3.96	5.76	5.76	1.96	2.16	3.96	3.96	2.00	1.80
27	OMH	THIRTY FOUR	M	1.84	2.12	4.08	4.32	5.92	6.12	2.24	2.20	4.08	4.00	1.84	1.80
	GROUP	SUBJECT #	SEX	I L	I R	III L	III R	V L	V R	I-III L	I-III R	I-V L	I-V R	III-V	III-V
28	AOM	TWENTY	F	2.16	2.04	4.40	3.88	6.16	6.12	2.24	1.84	4.00	4.08	1.76	2.24
29	AOM	TWENTY TWO	F	1.60	1.76	3.64	3.64	5.64	5.44	2.04	1.88	4.04	3.68	2.00	1.80
30	AOM	TWENTY FOUR	F	1.72	1.84	3.68	3.80	5.68	5.72	1.96	1.96	3.96	3.88	2.00	1.92
31	AOM	THIRTY FIVE	F	2.12	2.40	4.24	4.60	6.00	6.12	2.12	2.20	3.88	3.72	1.76	1.52
32	AOM	SEVENTEEN	M	1.96	2.08	4.20	4.20	5.96	5.96	2.24	2.12	4.00	3.88	1.76	1.76
33	AOM	TWENTY SIX	M	2.40	2.44	4.48	4.64	6.52	6.80	2.08	2.20	4.12	4.36	2.04	2.16
34	AOM	TWENTY FIVE	M	2.44	2.40	4.20	4.72	6.04	6.40	1.76	2.32	3.60	4.00	1.84	1.68
35	AOM	ELEVEN	M	2.24	1.80	4.28	4.60	6.28	5.72	2.04	2.80	4.04	3.92	2.00	1.12
36	AOM	THIRTY THREE	M		1.72	5.32	4.36	7.16	5.72		1.36		4.00	1.84	1.36

APPENDIX B

INDIVIDUAL DATA FOR THE MIDDLE LATENCY RESPONSE WAVES NA, PA, AND NB

		MIDDLE LATENCY RESPONSE DATA SUMMARY						
	GROUP	SUBJECT #	NA L	NA R	PA L	PA R	NB L	NB R
1	NL	ONE						
2	NL	FOURTEEN	17.60	19.20	28.80	33.28	45.76	48.96
3	NL	FIFTEEN	16.32	12.80	28.16	30.40	32.96	50.24
4	NL	EIGHTEEN	18.56	15.36	32.32	29.12		41.60
5	NL	SIXTEEN	29.12	19.84	35.84	33.60	48.32	47.68
6	NL	NINETEEN	13.12	17.92	24.96	33.60	33.28	48.00
7	NL	TWENTY THREE	15.36	21.20	28.16	27.20	32.64	33.60
8	NL	TWENTY SEVEN	19.00	18.88	31.00	29.44	39.36	41.28
9	NL	TWENTY EIGHT	19.20	17.92	34.56	34.56	49.60	54.08
10	NL	TWENTY NINE	19.20	18.24	31.36	28.16	39.68	41.92
11	NL	THIRTY	18.56	16.00	30.08	27.20	37.76	42.88
	GROUP	SUBJECT #	NA L	NA R	PA L	PA R	NB L	NB R
12	OMH	TWO	17.28	19.52	31.04	35.20	46.08	51.20
13	OMH	THREE						
14	OMH	FIVE						
15	OMH	SIX	16.64	22.72	37.12	31.04	49.60	42.56
16	OMH	SEVEN	11.52	18.56	21.44	30.08	33.28	36.48
17	OMH	EIGHT	15.68	17.96	27.52	26.88	33.92	34.56
18	OMH	NINE		15.36		29.12		41.60
19	OMH	TEN	19.20	19.52	27.52	28.16	44.16	37.12
20	OMH	ELEVEN	21.12	19.20	33.60	33.28	51.20	44.16
21	OMH	TWELVE		16.96		31.04		
22	OMH	THIRTEEN	18.56	18.24	29.44	27.52	34.56	37.12
23	OMH	FOUR	16.96	13.76	34.56	23.68		
24	OMH	TWENTY ONE		12.80		26.56		41.28
25	OMH	THIRTY ONE	21.76	22.40	31.68	31.68	43.20	41.92
26	OMH	THIRTY TWO						
27	OMH	THIRTY FOUR	16.64	22.72	36.16	32.64	58.24	44.48
	GROUP	SUBJECT #	NA L	NA R	PA L	PA R	NB L	NB R
28	AOM	SEVENTEEN	22.04	18.88	38.72	36.48	53.44	49.28
29	AOM	TWENTY						
30	AOM	TWENTY TWO	12.48	20.80	33.60	38.08	53.12	51.52
31	AOM	TWENTY SIX						
32	AOM	TWENTY FOUR	16.00	16.32	31.36	32.00	48.96	47.04
33	AOM	TWENTY FIVE	17.28	22.08	35.84	36.80	54.72	52.80
34	AOM	ELEVEN	17.92	16.32	24.60	30.40	45.76	42.88
35	AOM	THIRTY THREE	22.72	17.28	35.52	32.96	50.24	44.16
36	AOM	THIRTY FIVE	18.56	22.08	31.36	30.08	49.92	48.00

APPENDIX C

INDIVIDUAL DATA FOR THE LATE AUDITORY RESPONSES WAVES N1, P2, N2, AND P3

		LATE AUDITORY RESPONSE DATA SUMMARY									
	GROUP	SUBJECT #	SEX	LATE CORTICAL RESPONSE LATENCY AND AMPLITUDE							
				N1 L	N1 R	P2 L	P2 R	N2 L	N2 R	P3 L	P3 R
1	NL	ONE	F	99.2	108.8	163.2	166.4	188.8	188.8	486.4	444.8
2	NL	FIFTEEN	F	179.2	172.8	208.0	201.6	300.8	246.4	579.2	534.6
3	NL	TWENTY EIGHT	F	118.4	150.4	169.6	182.4	236.8	204.6	726.4	547.2
4	NL	TWENTY NINE	F	185.6	147.2	284.8	179.2	368.0	230.4	508.8	473.6
5	NL	FOURTEEN	M	156.8	172.8	220.8	256.0	291.2	329.6	499.2	688.0
6	NL	EIGHTEEN	M	96.0	99.2	144.0	169.6	192.0	220.8		
7	NL	SIXTEEN	M	150.4	195.2	249.6	307.2	297.6	387.2	486.4	496.0
8	NL	NINETEEN	M	214.4	144.0	265.6	188.8	313.6	252.8	476.8	668.8
9	NL	TWENTY THREE	M	147.2	233.6	169.6	310.4	214.4	348.8	694.4	576.0
10	NL	TWENTY SEVEN	M	172.8	217.6	195.2	262.4	249.6	307.2	448.0	585.6
11	NL	THIRTY	M	102.4	92.8	144.0	131.2	233.6	195.2	630.4	448.0
	GROUP	SUBJECT #	SEX	N1 L	N1 R	P2 L	P2 R	N2 L	N2 R	P3 L	P3 R
12	OMH	TWO	F	169.6	182.4	211.2	211.2	230.9	227.2	563.2	697.6
13	OMH	THREE	F	153.6	153.6	198.4	198.4	224.0	246.4	502.4	483.2
14	OMH	FIVE	F	179.2	214.4	240.0	316.8	307.2	396.8	499.2	550.4
15	OMH	THIRTY TWO	F	214.4	217.6	275.2	265.6	313.6	288.0		
16	OMH	SIX	M	160.0	182.4	233.6	224.0	307.2	304.0	544.0	540.8
17	OMH	SEVEN	M	156.8	140.8	256.8	246.4	281.6	288.0	582.4	604.8
18	OMH	EIGHT	M	198.4	204.8	265.6	249.6	291.2	304.0	688.0	640.0
19	OMH	NINE	M	134.0	131.2	172.8	166.4	220.8	220.8	560.0	585.6
20	OMH	TEN	M	169.6	160.0	240.0	272.0	342.4	332.8	707.2	704.0
21	OMH	ELEVEN	M	188.8	198.4	304.0	320.0	384.0	396.8	761.6	537.6
22	OMH	TWELVE	M	204.8	201.6	256.0	249.6	320.0	294.4	467.2	460.8
23	OMH	THIRTEEN	M	195.2	192.0	249.6	275.0	297.6	396.8	537.6	464.0
24	OMH	TWENTY ONE	M	195.2	156.8	272.0	208.0	345.6	249.6		502.4
25	OMH	THIRTY ONE	M	163.2	169.6	188.8	188.8	227.2	230.4	665.6	633.6
26	OMH	THIRTY FOUR	M	182.4	185.6	230.4	249.6	265.6	313.6	499.2	630.4
27	OMH	FOUR	M								
	GROUP	SUBJECT #	SEX	N1 L	N1 R	P2 L	P2 R	N2 L	N2 R	P3 L	P3 R
28	AOM	TWENTY	F	163.2	176.0	227.2	233.6	268.8	275.2	617.6	
29	AOM	TWENTY TWO	F	204.8	230.4	291.2	284.8	288.0	364.8		
30	AOM	TWENTY FOUR	F	211.2	198.4	230.4	246.4	243.2	275.2		617.6
31	AOM	THIRTY FIVE	F	204.8	188.8	265.6	262.4	342.4	364.8	620.8	608.0
32	AOM	SEVENTEEN	M	208.0	211.2	284.8	256.0	316.8	281.6		643.2
33	AOM	TWENTY SIX	M	281.6	227.2	371.2	297.6	464.0	361.6	768.0	652.8
34	AOM	TWENTY FIVE	M	179.2	179.2	211.2	214.4		304.0		
35	AOM	ELEVEN	M	182.4	224.0	256.0	288.0	368.0	342.4	768.0	528.0
36	AOM	THIRTY THREE	M	233.6	230.4	259.2	249.6	316.8	272.0	668.8	486.4

VITA

Eva Kinsey Saffer was born in Elco, Nevada on August 18, 1954. She attended elementary schools in Perryton, Texas and was graduated from Perryton High School in May 1972. The following summer she entered Frank Phillips Junior College, and in May of 1974 she received an Associate of Arts Degree. She went on to attend Texas Tech University, receiving a Bachelor of Science degree in Education in May 1976. In the Fall of 1976, she began study toward a Master of Science degree in Audiology at Texas Tech University. She was awarded the Master's degree in May of 1979. She received the Certificate of Clinical Competency from the American Speech and Language Association in 1980.

After four years in private practice, she entered the Graduate School of the University of Tennessee, Knoxville, in September of 1985. During this time she was a clinical supervisor with the Speech and Hearing Center at the University of Tennessee.

The author is a member of the American Speech and Hearing Association, Texas Speech and Hearing Association, Alexander Graham Bell Association, and the Self Help for the Hard of Hearing.