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Behavioral and electrophysiological assessment of children with a specific temporal processing disorder

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BEHAVIORAL AND ELECTROPHYSIOLOGICAL ASSESSMENT OF CHILDREN WITH A SPECIFIC TEMPORAL PROCESSING DISORDER

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DEDICATION

What could possibly be worse than writing a dissertation? Perhaps living with someone who is trying to complete one! For their love, patience and understanding, I dedicate this project to my family. I am blessed to have a supportive husband who did everything from picking up articles at the library, putting together racks in my lab, learning to cook, and serving as a subject when I was trouble-shooting the BIC- ITD measurements. Thank you, Louis! I am also very blessed to have a wonderful daughter, Lana, who perhaps felt somewhat neglected during the latter part of this process. I love you and I look forward to spending more time together!

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TABLE OF CONTENTS

LIST OF TABLES

LIST OF FIGURES

ABSTRACT

Auditory processing disorders (APDs) have received considerable attention over the past few decades. Much of the attention has focused on the controversy surrounding the operational definition of APD, the heterogeneous nature of APD, and an appropriate test battery for APD assessment. Temporal processing deficits are one characteristic of APD and are the focus of the present investigation. This investigation reports behavioral and early electrophysiological measures in a group of children with specific temporal processing difficulties and an age-matched control group. In an effort to better describe the subjects, two language tests and the SCAN-C were administered. Significant differences were found in the language tests, SCAN-C, and behavioral tests of temporal processing. No significant differences in ABR waveform latency were found between the control and experimental group. Significant amplitude differences were found, albeit small. Binaural interaction was present in both groups. Based on the results of the present well-controlled investigation of children with temporal processing disorders, there is no indication that the auditory brainstem response recording to click stimuli is efficient in providing additional diagnosis of APD.

CHAPTER 1

INTRODUCTION

Auditory processing disorders (APDs) have received considerable attention over the past few decades. APD is not a new entity in audiology. For many years, professionals have been aware that some individuals with normal results on tests of peripheral function report difficulty understanding speech. Since APD involves processing of auditory signal, audiologists are called upon to make this diagnosis of APD based upon a battery of tests.

Much of the recent attention has focused on the controversy surrounding the operational definition of APD, the heterogeneous nature of APD, and an appropriate test battery for APD assessment. This resurgent interest in APD has generated a clinical demand for improved diagnostic methods as well as evidence-based, effective treatment plans for APD.

APDs are wide-spectrum disorders. Investigators have attempted to document the heterogeneous nature of APDs by sub-grouping APD or describing the characteristics in terms of commonalities (Bellis & Ferre, 1999; Katz, 1992; Musiek & Gollegly, 1988). Although this may be beneficial in management, no sub-grouping system or model is universally accepted. In addition, APD may exist with other learning, language, or reading disorders. This comorbidity has created controversial debate of an appropriate diagnosis of APD with other learning disorders or if the diagnosis of APD should only be made when it is a single entity.

Most investigations of APD have not described the specific auditory deficits or

characteristics of their subjects. This may have led to some of the conflicting results in both behavioral and electrophysiological measures in children with APD. The research reported in this dissertation represents is a first-step in addressing some of the confounding issues surrounding APD. Specifically, this investigation will address a sub-group of children with APD who have specific temporal processing deficits.

Temporal processing refers to the time aspects of an auditory or acoustic signal. Phillips (1995) defines temporal processing in several ways including determination of a sound source or "spatial percept," or determination of the pitch of a sound, and the perceptual segregation of two successive acoustic events. Temporal processing is important in the discrimination of duration and variations in pitch, which are critical to following the prosody of speech and music perception (Phillips, 1995).

Temporal processing deficits have also been associated with learning disabilities. Tallal's work (Tallal, Miller & Fitch, 1993; Tallal, Miller, Bedi, Byma, Wang et al., 1996; Merzenich, Jenkins, Johnston, Schreiner, Miller, & Tallal, 1996) demonstrates that impaired temporal processing may result in language disorders, speech processing disorders and reading disorders. Tallal reports, "The phonological and language difficulties of language-learning impaired children may result from a basic deficit in processing rapidly changing sensory inputs" (Tallal et al, 1996, p. 81). These investigators hypothesize that impaired temporal processing disrupts the normal development of an efficient phonological system and these phonological difficulties result in language and reading disorders.

Poor temporal processing is one of the characteristics of APD and is a key component of auditory function (Chermak & Musiek, 1997). Temporal processes are

critical in a number of auditory functions "including auditory discrimination, binaural interaction, pattern recognition, localization/ lateralization, monaural low-redundancy speech recognition, and binaural integration" (Show, Seikel, Chermak, & Berent, 2000, p. 67).

The underlying physiological neural mechanisms for temporal processing may be assessed by behavioral and electrophysiological means. Behavioral tests "stress" the auditory system by degrading the acoustic environment or signal by introducing background or speech noise or by filtering the signal. Behavioral tests may require multiple auditory processes such as attention, memory, and perception (Jirsa & Clonz, 1990). Further, behavioral tests may be confounded by learning, attention, fatigue, hearing sensitivity, intelligence, developmental age, motivation, motor skills, language experience, and language impairments (Jerger & Musiek, 2000).

Electrophysiologic recordings of the central and peripheral neural auditory pathway, specifically in the early latency Auditory Brainstem Response (ABR) and the derived Binaural Interaction Component (BIC) recordings, objectively assess neural functions that are believed to be involved in early neural coding for temporal processes (Hall, 1992). The ABR reflects synchronous firing of neurons of cranial nerve VIII and lower brainstem structures. The BIC reflects neural activity which is hypothesized to reflect such binaural processes as localization and lateralization (Debruyne, 1984; Dobie & Wilson, 1985; Hendeler, Suires & Emmerich, 1990). Other investigators report that the BIC may objectively reflect ongoing binaural processing (Fowler & Swanson, 1988; Jiang & Tierney, 1996). Together, these electrophysiologic recordings provide information about the integrity of the lower central auditory pathways that are also

involved in auditory processing and the requisite capabilities of the auditory system to encode information.

This investigation is based upon the premise that there is a subgroup of children who are "at risk for" or have been diagnosed with APD who have specific temporal processing deficits. Children with temporal processing difficulties have difficulty processing the temporal aspects of speech and other non-linguistic acoustic stimuli. Preliminary temporal coding may be observed in the early firing pattern of the VIII nerve and auditory brainstem nuclei. This preliminary coding underlies higher order, time-related perceptual processes. This deficit in temporal processing will be assessed by behavioral tests and by electrophysiologic, objective measures reflective of synchronous neuronal firing in the peripheral and central auditory pathway. The specific aims are:

SPECIFIC AIM 1: Is there dys-synchrony in the peripheral nerve and auditory brainstem pathway in children at risk for APD, as evidenced by differences in the ABR? Published reports of APD and electrophysiologic recordings are conflicting. Some investigations have reported latency and amplitude differences in the ABR recordings (Sohmer and Student, 1978). Others have shown no differences or abnormalities in these recordings (Shimizu, Brown, Capute, & Mahoney, 1981). The hypothesis is that children who perform abnormally on behavioral tests for auditory processing may also show abnormalities in electrophysiologic recordings at the auditory nerve and brainstem levels. This hypothesis is based upon the fact that auditory processing begins in the peripheral auditory system where tonotopically arranged hair cells send electrical impulses to the central auditory system. At the level of the cochlear nucleus, neurons are temporally coded for the onset and termination of a sound; temporal

processing continues through a "bottom-up" process through the mid-brain and auditory cortices (Efron, 1963).

SPECIFIC AIM 2: Is there dys-synchrony in the peripheral nerve and auditory brainstem pathway in children at risk for APD, as evidenced by differences in the BIC with and without interaural time differences? The Binaural Interaction Component may be a useful tool in evaluating "binaural processes such as localization, lateralization, and fusion" (Hall, 1992, p. 163). However, there is lack of research on the BIC in children with APD. Gopal and Pierel (1999) reported significant differences in the amplitude and latency of the BIC in children "at-risk" for APD. These investigators suggest this difference reflects a lack of binaural inhibition in the brainstem. Clearly, further research is warranted.

Investigators have also employed interaural time differences (ITDs) to further study the electrophysiological binaural response and the derived BIC. These investigations have reported a degradation of the BIC with an increase in ITD. In addition, BICs with ITDs have been paired with the behavioral task of lateralization. In one investigation, the BIC response was visible as long as the signal was fused (Furst, Levine & McGaffigan, 1985).

The hypothesis for this specific aim is that children with APD will show amplitude and latency differences in the BIC. Additionally, interaural time differences will degrade the BIC in children with APD to a greater degree in comparison to the control group.

In addition, this investigation will determine if there is a relationship between the performance on the behavioral tests for temporal processing and the electrophysiologic

recordings?

Temporal processing may be assessed behaviorally by such tests as the Masking Level Difference (MLD), Pitch Pattern Test, Duration Pattern Test, Random Auditory Gap Detection, and Time Compressed Speech Test. The hypothesis is that deficits that are demonstrated behaviorally in the temporal processing ability of children at risk for APD will also be seen in results of electrohysiologic tests. This hypothesis is based upon Musiek and Gollegly's (1988) sub-grouping of APD based upon an underlying neurophysiological deficit or neuromaturatioanal delay, neuromorphological disorder, maturational delay of the CNS, and neurologic diseases and insults. Therefore, the abnormal performance on behavioral assessment may be related to abnormalities in the the auditory pathway and will be objectively assessed by electrophysiologic recordings of the peripheral and auditory pathway.

This research proposal holds great clinical significance in the assessment of APD. There is a lack of data reporting both behavioral and electrophysiological measures in children with APD. Electrophysiological measures are recommended in the minimal APD test battery the American Speech-Language Hearing Association (1996) and the American Academy of Audiology (Jerger & Musiek, 2000). However, there is no research to support their recommendation. This research will be the first to provide both behavioral and electrophysiological measures in a well-defined group of children with temporal processing deficits.

CHAPTER 2

REVIEW OF LITERATURE

The literature reviewed in this chapter provides a general over-view of auditory processing disorders. This chapter has been subdivided into four major sections. The first section introduces the reader to the history of auditory processing disorders, sub-types of auditory processing, the etiology of APD, and comorbid conditions. The second and third sections describe the behavioral and electrophysiological assessment of APD. In order to better understand the underlying physiology of APD, a brief description of the anatomy and physiology of the central auditory nervous system is included. Finally, a section detailing the significance of the present investigation is included.

A. HISTORICAL OVERVIEW

Concern with auditory processing disorders dates back to the 1950s. Bocca, Calaero, & Cassinari (1954) and Bocca, Calaero, Cassinari & Migliavacca (1955) were first to report that patients with temporal lobe lesions had complaints of difficulty understanding speech. Frustrated with the inability of peripheral auditory assessment procedures to uncover any disturbance, this group of Italian physicians developed a monaural low redundancy speech test. Further, they reported that patients with temporal lobe lesions also had difficulty discriminating between sounds, even though they had normal peripheral hearing sensitivity. These investigators reported that patients with temporal lobe disorders acted as though they had a hearing loss and

concluded that unilateral lesions of the temporal lobe could impair integration and synthesizing ability of the central auditory nervous system (Musiek & Baran, 1987).

A few years later, Kimura (1961a, b) administered digit triads dichotically to subjects with temporal lobe lesions. She reported deficits in the ear contralateral to the temporal lobe lesion. She also reported ipsilateral ear deficits in subjects with left hemisphere lesions. Other investigators have reported difficulty understanding speech with brainstem lesions (Jerger & Jerger, 1974), and dysfunction in interhemispheric transfer of auditory information by way of the corpus callosum (Damasio & Damasio, 1979; Keith, 1977, 1981a, 1981b; Sparks & Geschwind 1968). Lesions in the superior temporal lobe are also found to be associated with abnormalities in phonemic perception (Luria, 1973).

Terminology

It was not until the late 1960s and 1970s that the term central auditory processing disorder was used to describe children with similar symptoms as adults with a central auditory nervous system lesion (Chalfant & Scheffelin, 1969; Katz & Illmer, 1972; Manning, Johnson & Beasley, 1977; Martin & Clark, 1977; Sweetow & Reddell, 1978; Willeford, 1977). Since then, interest has continued to grow as numerous articles, conferences, books and special committees have been devoted to this topic.

One of the controversies surrounding APD has been the terminology used to describe the disorder. "Central" has been used to distinguish the VIII nerve, brainstem and cortical areas as the anatomical site of dysfunction in contrast to the cochlea as a "peripheral" site of lesion. Central auditory processing is used interchangeably with central auditory function, central auditory perception, auditory language processing, and

auditory language learning. This has caused many investigators to adopt APD which relates to no specific anatomical site of dysfunction (Jerger & Musiek, 2000). However, other investigators continue to use "central" to emphasize the disorder occurs central to the peripheral hearing mechanism (Bellis, 2003). Other terminology used to describe auditory processing disorders include central hearing loss, auditory perception disorder, central deafness, word deafness, auditory agnosia, auditory memory deficit, auditory sequencing problem, and auditory dysfunction.

One of the problems in defining auditory processing disorders is that it is a description of symptoms of functional deficits (ASHA, 1996). Auditory processing stated simply is "what we do with what we hear" (Katz, 1992). Butler (1983) defined auditory processing as the abstraction of meaning from an acoustic signal and the retrieval of that meaning. The 1996 ASHA Task Force defines central auditory processing as "the mechanisms and processes responsible for the following behavioral phenomena: sound localization and lateralization; auditory discrimination; auditory pattern recognition; temporal aspects of audition including temporal resolution, temporal masking, temporal integration, temporal ordering; auditory performance decrements with competing acoustic signals; and auditory performance decrements with degraded acoustic signals." A central auditory processing disorder is an observed deficiency in one or more of the above-listed behaviors (ASHA, 1996, p.41).

The 2000 Bruton Consensus Conference on the "Diagnosis of Auditory Processing Disorders in School Aged Children" defined an auditory processing disorder as "a deficit in the processing of information that is specific to the auditory modality. The problem may be exacerbated in unfavorable acoustic environments. It may be

associated with difficulties in listening, speech understanding, language development, and learning. In its pure form, however, it is conceptualized as a deficit in the processing of auditory input" (Jerger & Musiek, p. 468).

Sub-groups of APD

Investigators have attempted to document the heterogeneous nature of APDs by sub-grouping APD or describing the characteristics in terms of commonalities (Bellis & Ferre, 1999; Katz,1992; Musiek & Gollegly, 1988). Although this may be beneficial in management, no sub-grouping system or model is universally accepted.

The Buffalo Model (Katz, Smith, & Kurpita, 1992) focuses on the relationship between patterns of performance on one particular test of auditory processing, and learning difficulties in children. This model contains four subtypes: Decoding, Tolerance-Fading Memory, Integration, and Organization. Decoding describes individuals who "have difficulty keeping up with the flow of communication, have poor phonemic skills, are slow responders, often have articulation errors, have difficulty following directions, and have weak oral reading and spelling skills" (Steker, 1992, p. 61).

Persons with tolerance-fading memory have difficulty understanding speech with competing background noise and have short-term memory problems. These individuals are often described as impatient and are easily over-stimulated. They tend to have poor reading comprehension and may have handwriting difficulty. Persons with integration problems have difficulty integrating the auditory modality with other non-verbal aspects of speech such as word finding, morphological and syntactical errors, or an expressive language disorder. Organization describes persons who have difficulty

sequencing events and have sequencing errors. These individuals are often disorganized at home or school. Often a person will exhibit characteristics of more than one sub-type.

Musiek & Gollegly (1988) report three types of APD in children with learning disabilities. These three types are based upon an underlying neurophysiological deficit or neuromaturational delay: neuromorphological disorder, maturational delay of the CNS, and neurologic diseases and insults. These types are theoretical and have not been directly investigated due to the invasive nature of necessary research procedures.

The Bellis/Ferre model of APD (Bellis and Ferre, 1999) is based upon the underlying neurophysiology of the brain and the relationship among different types of APD and language, learning, and communication difficulties. This model proposes five subtypes of APD, which are: Auditory Decoding Deficit, Prosodic Deficit, Integration Deficit, Associative Deficit and Output Organization Deficit. Again, these theories and subtypes are conceptual descriptions of the academic problems of children. "Auditory decoding refers to persons with "poor auditory closure abilities, characterized by poor performance on tests of monaural low redundancy speech and speech-in-noise" (Bellis, 1996, p. 186). Integration Deficit refers to difficulties in interhemispheric transfer. Associative Deficit refers to "an underlying inability to apply the rules of language to incoming acoustic information" (Bellis, 1996, p. 189). Output-Organization Deficit is a deficit in organizing, planning, and sequencing responses. Again, it is possible that a person may have more than one sub-type.

The present research investigation further subgroups children with APD into a group with specific temporal processing difficulty. Temporal processing deficits have previously been identified in children with language and learning disorders. As noted in

the introduction, Tallal reports, "The phonological and language difficulties of language-learning impaired children may result from a basic deficit in processing rapidly changing sensory inputs" (Tallal et al, 1996, p. 81).

There are limited data regarding normative temporal processing ability in school- age children. Gap detection thresholds in pre-school and school-age children have been reported by McCroskey and colleagues. Davis and McCroskey (1980) reported a reduction in gap detection thresholds with an increase in age. Temporal order judgments have been reported at 36.1 ms for children 7 to 14 years of age (Lowe & Campbell (1965), compared to 20 to 25 ms for adults (Hikrsh, 1959; Hirsh & Sherrick, 1961; Pisoni, 1977).

Discrimination scores of time-compressed speech in school-aged children also improve with age (Beasley & Maki, 1976). Allen (1997) reports that temporal auditory discrimination and detection is often more variable in school-age children than adults. Certainly, it is evident that there are improvements in temporal related auditory tasks with age. This lack of normative temporal auditory data in school-aged children has been addressed by the ASHA Task Force on Central Auditory Processing Disorders.

Etiology

Historically, brain lesions were thought to be the underlying cause of APD. Persons with similar symptoms were thought to have some central auditory pathway lesion. As pointed out in the previous section, one sub-grouping of APDs was based upon theorized neuroanatomical and neurophysiological etiologies (Musiek and Gollegly; 1988). Causes of APD in children are not completely understood. Often,

these children do not show any neurological disease or show any neurological abnormality (Schain, 1977).

Not all cases of APD have an underlying structural deficit, therefore, APD may be difficult to diagnose with computerized tomography or magnetic resonance imaging scans of the brain. Researchers have suggested that the problem underlying APD "may be invisible to many neurologic and radiolgic studies" (Musiek & Lamb, 1994, p. 198).

Other prenatal or perinatal factors that may be indicated in APD are: hyperbilirubinemia, ototoxic drugs, anoxia, low birth weight, RH incompatibility, prematurity, abnormal secretion that affects brain cell development prior to birth, and unspecified birth problems (Willeford & Burleigh, 1985). Maternal factors which may adversely affect the development of the central nervous system include diabetes, rubella, syphilis cytomegaloviruses, and toxemia (Willeford & Burleigh, 1985). Hereditary factors may also play an important role (Willeford & Burleigh, 1985; Bellis, 2002). Future brain imaging studies such as functional magnetic resonance imaging may prove of value in further understanding the mechanisms involved in brain function and auditory processing in normal children and children with APD.

Comorbidity of APD

There is an intimate relationship between language, attention, and auditory skills. Auditory processing disorders often coexist with learning disabilities, language disorders, attention deficit disorders, and dyslexia (Chermak & Musiek, 1997; Caccace and MacFarland, 1998). All of these groups are heterogeneous in nature. However, it is important to note that not all children with a language, learning or attention disorder will have an auditory processing disorder. APDs have also been linked with children

with chronic otitis media (Brown, 1994; Gravel & Wallace, 1992; Hall & Grose, 1993; Hall, Grose & Pillsbury, 1994) and with the elderly and aging population (Committee on Hearing, Bioacoustics and Biomechanics Working Group on Speech Understanding and Aging, 1988; Stach, Spretnjak, & Jerger, 1990). This has led some investigators to question if auditory processing deficits underlie language disorders, or if auditory processing disorders are but one type of language disorder (Keith 1981a, 1981b; Rees, 1973, 1981; ASHA 1996).

Controversy exists about the label of APD in children with multi-sensory deficits. Some investigators argue that if multi-sensory deficits are present, then the diagnosis of APD is inappropriate and the diagnosis is only appropriate where there is a single auditory deficit (Cacace & McFarland; 1998). However, given the interconnections of the nervous system and the influence of higher-level functions such as language, cognition and attention, the single modality-specific definition for APD is not logical (Bellis, 2003).

Oral language acquisition depends upon the efficient processing of acoustic stimuli (ASHA, 1996). An auditory perception account of the etiology of children with specific language impairments has been proposed. This theory posits that some children with specific language impairments have difficulties in perceiving rapid acoustic events and have difficulty in processing auditory information of brief duration relative to surrounding segments (Tallal, 1976; Tallal & Piercy, 1973; Tallal & Stark, 1981). This difficulty will not only affect phoneme recognition, but also affect the listener's ability to segment speech. Leonard (2001) reported that the primary flaw of

the auditory perception account was that it does not account for the full range of linguistic problems of children with specific language impairments.

A degraded acoustic environment may hinder speech processing. This degraded environment has also been theorized to be one of the etiologies of specific language impairments in that the amount and type of linguistic input necessary for optimal language acquisition is not present (Cramblit & Siegel, 1977; Lasky & Klopp, 1982). However, it is important to note that not all children with specific temporal processing deficits show language or speech disorders.

There are two contrasting models regarding the influence of lower order perceptual processing and higher order cognitive processing on language and learning disabilities (Keith, 1981). Models describe how listeners "perceive the acoustic signal, conduct auditory analysis involving complex pattern recognition; match acoustic patterns to some internal representation(s); extract meaning from strings of lexical representations; and construct a message level interpretation (Craig, 1977, p. 73). Bottom-up processing is a term used in information processing which describes the cochlea and the brain's analysis of neural coding through the cortex (Chermak & Musiek, 1997). Top-Down Processing refers to the influence of higher level cognitive or language related knowledge on the interpretation of incoming sensory information.

B. BEHAVIORAL ASSESSMENT

General Overview

An auditory processing assessment must accomplish three things: first, it must be determined if auditory processing is affected; second, if auditory processing is deficient, then the severity of the APD must be assessed; third, the clinician must

determine if the APD can account for the person's communication and learning difficulties. These three goals of an APD assessment are not without controversy.

Investigators and clinicians suggest that the diagnosis of APD is made based upon the pattern of responses in a battery of tests. In addition, the severity of APD is difficult to address in that most of the behavioral tests are not standardized. Instead, normal cut-off values are given. Lastly, it is clinically impossible to determine the contribution of APD to the individual's learning difficulties if there are comorbid conditions. Based upon information from the APD assessment, the clinician must make management recommendations, such as assistive listening devices, suggest appropriate compensatory techniques, and recommend remedial therapy programs that will improve auditory processing skills.

Behavioral tests have been used extensively in the diagnosis of APD. Historically, behavioral tests were developed for site-of-lesion testing. Because of similar symptoms, these tests were later used to assess auditory processing. This approach with children is based on the assumption that these children with similar symptoms are neuro-developmentally immature or there is an abnormality in the central auditory nervous system, or function as if they have a lesion (Keith & Jerger, 1991).

Although use of a test-battery is recommended for the diagnosis of APD (Katz, 1992; Musiek & Lamb, 1994), there is no statistically compelling evidence indicating which tests should be included in the battery, which tests correlate with other tests, or the cost-effectiveness of each test (Singer, Hurley, & Preece, 1998). The test battery must stress the central auditory nervous system at various levels to identify areas of weakness. Clearly, data are limited.

The following review will describe the tests commonly used in APD assessment. A discussion of peripheral hearing loss is included as part of the assessment battery. Following that, the behavioral tests discussed in this review are divided into four sections: binaural integration/ interaction tests, monaural low-redundancy speech tests, dichotic speech tests, and monotonic tone tests.

Peripheral Hearing Evaluation

It should be noted that peripheral hearing sensitivity should be documented so that any peripheral hearing loss is ruled out. Peripheral hearing loss can effect the outcome of many auditory processing tests, as audibility is key in understanding speech. Included in the peripheral hearing assessment recommended by both the American Speech-Language-Hearing Association Task Force (1996) and the American Academy of Audiology Consensus Conference (2000) are pure tone air and bone conduction, immittance audiometry, which includes tympanometry and ipsilateral and contralateral acoustic (middle ear muscle) reflexes and speech audiometry. Otoacoustic emissions are often included and provide valuable information about the integrity of the outer hair cells and may provide valuable diagnostic information about auditory neuropathy or dys-synchrony (Berlin, Hood, Cecola, Jackson, & Szabo, 1993; Hood, Berlin, Hurley, Cecola, & Bell, 1996; Musiek & Jerger, 2000), which share some of the symptoms and may be mis-identified as APD.

APD Screening

Another area of concern has been the use of tests designed for screening as a diagnostic tool. Such tests provide an overview of speech- language, educational, and

cognitive function, but should not be used as a single diagnostical test for APD assessment (Musiek, Gollegly, Lamb & Lamb, 1990).

The Screening Test for Auditory Processing Disorders in Children (SCAN-C) is the only audiological test that has been designed for the specific purpose of screening for APD (Keith, 1986). It consists of four sub-tests: Filtered Words, Auditory Figure Ground, Competing Words and Competing Sentences. No specific sub-test for temporal processing is included. Temporal processes are employed, however, in the sub-tests. Results of the SCAN have been shown to correlate with findings on selected tests of auditory processing, i.e., the Staggard Spondaic Words test and the Competing Sentence Test (Keith, 1986). The SCAN-C has published norms for individuals from ages 5 to 11 years of age and provides valuable information about the maturation of the auditory system. Results of the SCAN-C should be considered with all other information to determine if further testing is necessary.

Binaural Integration Tests

Binaural integration tests are also referred to as binaural interaction tests. This group of tests requires the integration of auditory information from both ears. These tests are sensitive to brainstem lesions; however, they can be affected by higher auditory centers.

Masking Level Difference

The Masking Level Difference (MLD) is a widely used test of temporal processing and binaural interaction. The MLD compares the threshold of two binaural signals: either a low-frequency tone (500 Hz) or speech embedded in noise. The thresholds for the signals are measured in noise while the noise is in-phase

(homophasic- No) and out-of- phase (antiphasic- Nπ) with the signal, or while the signal is in phase (homophasic- So) and out-of-phase (antiphasic Sπ) with the noise (Hirsh, 1948; Olsen, Noffsinger, & Carhart, 1976; Olsen, Noffsinger, & Kurdziel, 1975). In most cases, there is a release of masking, or improvement in threshold, either when the noise or signal is out-of-phase between the two ears. This release of masking occurs because the listener perceptually can separate the competing signal. The stimulus appears to originate from a different source while out-of-phase. The MLD is mediated by the lower brainstem. The MLD has been shown to be abnormal in patients with brainstem lesions (Olsen et al, 1976; Lynn, Gilroy,1977); whereas cortical lesions have shown no effect on the MLD (Cullen & Thompson; 1974).

There are limited data reporting MLDs in children. The MLD has been shown to be smaller in children with histories of protracted otitis media (Pillsbury, Grose, & Hall, 1991; Hall & Grose, 1993). However, after medical intervention for the otitis media, the MLD returned to normal (Hall & Grose, 1993). Rosenthal & Wohlert (1973) reported smaller MLDs in a group of aphasic children. In addition, the MLD is reduced in children with auditory perceptual difficulties (Sweetow & Reddell ,1978). However, Wayras & Battin (1985) did not report a reduced MLD in learning disabled children but attributed this finding to the wide heterogeneity of learning disabled children. Roush & Tait (1984) also found normal MLDs in children with APD.

Rapidly Alternating Speech Perception

Willeford (1976) first introduced the Rapidly Alternating Speech Perception (RASP) test. This test requires the integration of segments of speech. Unintelligible sequential bursts of information are delivered to the right and left ears at periodic

intervals. The most common clinical stimuli have been sentences although monosyllabic words have been used and may be preferred by reducing redundancy. The rapidly alternating message is easily understood in normal listeners. The RASP+ has been used in site-of lesion testing and in APD evaluations, but with questionable efficacy. Studies have shown that the RASP tests may not be sensitive to all brainstem lesions. Musiek (1983) and Lynn & Gilroy (1977) report only a small percentage of subjects showed abnormal results on this test. In addition patients with interhemispheric or corpus callosum lesions performed normally on this test (Lynn & Gilroy, 1977). Therefore, other tests with greater specificity to diagnose brainstem lesions are normally chosen. Willeford and Billger (1978) have found abnormal results in only a small percentage of children with APD.

Binaural Fusion

These tests employ stimuli that have been filtered into two separate segments that are then presented simultaneously to the two ears of the subject. Generally, the filtering of the stimuli causes the stimuli to be unintelligible in one ear alone, but with the combined, filtered information from the other ear and assuming the auditory system is functioning correctly, the information is spectrally fused and recognition occurs. Stimuli most often used are monosyllabic words or spondees.

Data from investigations using binaural fusion tests are conflicting. Matzker (1975) reported normal results for subjects with cortical lesions. However, subjects with brainstem involvement have abnormal test results. Other studies report that only a small percentage of patients with known brainstem lesions perform abnormally on the binaural fusion task (Lynn & Gilroy, 1977; Smith & Resnick, 1972). These conflicting

results may be in part due to the small sample size and subject selection, as well as the difference in the presentation level.

Auditory processing tests such as the Binaural Fusion Test and the Rapidly Alternating Speech Perception Test require binaural fusion, binaural localization, and/or binaural integration. Information from the two individual ears must interact. This information is mediated by the superior olivary complex (Tobin, 1985). In addition, the Masking Level Difference (MLD), a test of brainstem integrity and higher brainstem centers, requires binaural integration also (Lynn, Gilroy, Taylor & Leiser 1981). With questionable sensitivity, the Binaural Fusion Test and the RASP test are not widely used clinically, whereas, the MLD is widely employed.

Monaural Low-Redundancy Speech Tests

Monaural low-redundancy tests have been used extensively in the evaluation of APD. These tests use stimuli that have been degraded, modified, or distorted in the frequency, temporal, or spectral domain to reduce redundancy. Because speech is so redundant, the normal listener can recognize speech even when parts are missing. However, subjects with central auditory dysfunction cannot easily recognize this modified speech.

Low-Pass Filtered Speech

Filtered speech reduces the redundancy of the signal. Several investigations have reported the use of low-pass filtered speech in assessing the auditory processing of subjects with intracranial lesions. Using various stimuli, (i.e. monosyllabic words, spondees, digits) and varying the frequency characteristics of the stimuli, as well as presentation levels, investigators have reported contralateral deficits in patients with

temporal lobe lesions (Baran & Musiek, 1991; Bocca et al., 1954,1955; Hodgson, 1967; Jerger, 1960; Lynn & Gilroy, 1977; Musiek, Baran & Pinheiro, 1994). Subjects with interhemispheric pathway involvement have performed normally on filtered speech tests (Baran, Musiek, & Reeves, 1986; Lynn & Gilroy, 1977; Musiek & Chermak, 1994; Musiek, Pinheiro, & Musiek, 1985). No consistent pattern has been reported in subjects with brainstem involvement. This may be due to the level, size of the lesion, and whether it is an extra-or intra-axial lesion (Musiek & Guerkink, 1982). Elliot & Katz (1979) reported familiarity with target words as well as the cutoff frequency will affect the test results.

Time Compressed Speech

Compressed speech alters the temporal and frequency characteristics of the signal. Historically, the first compressed speech tests were accomplished by having the speaker read the passage faster or by increasing the playback speed of the tape recorder. Soon after, electromechanical alterations and later digital computer editing of natural speech were used to distort the temporal and frequency components of speech. In normal listeners, a compression ratio of 60% is the cutoff for normal performance (Beasley & Maki, 1976). However, some clinicians feel that the 60% compression ratio is difficult for normal listeners and employ a 45% ratio clinically (Bellis, 1996; 2003). This test of reduced temporal redundancy is sensitive to dysfunction at all levels of the central auditory pathway (Pinheiro & Musiek, 1985; Thompson & Abel, 1992a, 1992b). Additionally, investigators have employed multiplicative effects by using time-compressed speech in a reverberant background (Wilson, Preece, Salamon, Sperry, &

Bornstein, 1994). The reverberant background further reduces the redundancy of the signal.

Speech-in-Noise

One of the most common characteristics of individuals with APD is the adverse effect of background noise on communication. There are many clinically acceptable ways to assess speech in noise, including different types of stimuli (monosyllabic words or sentences), different types of noise (white, speech, cafeteria, or babble), and different signal-to-noise ratios. Research indicates that linguistic materials (i.e. multi-talker speech babble background noise) are more effective maskers than speech-spectrum noise, even though the frequency spectrum and amplitude may be similar (Sperry, Wiley & Chial, 1977). In addition, speech babble is also more effective than narrow band or white noise (Sperry et al., 1977).

Introducing ipsilateral noise is one method of reducing the redundancy of speech stimuli. Patients with CNS lesions have shown reduced performance for the ear contralateral to the lesion in the auditory cortex (Heilman, Hammer, & Wilder, 1973). Olsen, Noffsinger, & Kurdziel (1975) reported that 50% of subjects with temporal lobe lesions performed within normal variability range. Therefore, speech-in-noise tests may not be useful in specific identification of a cortical site of lesion.

Dichotic Speech Tests

Dichotic Speech Tests were first introduced in 1961 by Kimura. Different stimuli are presented simultaneously to the two ears. Kimura developed a model to describe how the central auditory nervous system processes dichotic stimuli. The contralateral pathways are more numerous; therefore, the contralateral pathway will be

dominant. Dichotic speech tasks have employed a number of stimuli including digits, nonsense syllables, spondees, monosyllabic words, and sentences. These tests, as a group, are reported to be sensitive to cerebral and interhemispheric compromise (Musiek, Kibbe, & Baran, 1984) and brainstem involvement (Katz, 1962; Jerger & Jerger, 1974; Keith, 1977; Musiek, 1983).

Competing Sentences

The Competing Sentences Test was developed by Willeford (1977). Individuals must attend to stimuli in one ear and ignore the competing message. Approximately fifty percent of subjects with central auditory nervous system (CANS) lesions had abnormal performance in the ear ipsilateral to the lesion whereas subjects with temporal lobe lesions showed contralateral deficits (Baran & Musiek, 1991; Lynn & Gilroy, 1972, 1975, 1977; Musiek 1983, 1983b; Musiek, Baran, & Pinheiro, 1994). This test has been shown to be less sensitive to cerebral lesions than performance on the Staggered Spondaic Word Test or Dichotic Digit Test (Lynn & Gilroy, 1972, 1975, 1977; Musiek, 1983b).

Staggered Spondaic Words (SSW)

Katz (1962) first described the Staggered Spondaic Word (SSW) test. This test consists of spondaic words that are presented dichotically in a staggered manner so that the second syllable of the first spondee is overlapped with the first syllable of the second spondee. This test has been shown to be sensitive to brainstem and cortical lesions (Katz, 1962). It has also been widely used with children.

Dichotic Digits

The Dichotic Digits Test (Musiek, 1983) is a dichotic test in which digits are presented simultaneously to both ears. Results of investigations using the dichotic digits indicate contralateral deficits in subjects with right temporal lobe lesions and bilateral or contralateral deficits in subjects with left hemisphere lesions (Baran & Musiek, 1991; Musiek, 1983). Left ear deficits have been reported in subjects with interhemispheric compromise and are more frequently reported than right ear deficits (Musiek et al., 1994). This test is not highly linguistically loaded and is easy and quick to administer. One criticism of this test is that it offers no normative data, only cut-off ranges for normal and abnormal scores (Katz, Johnson, Brandner, Teryl, Ferre, et al., 2002).

Dichotic CVs

Berlin & Lowe (1972) introduced Dichotic Consonant Vowel Test for central auditory nervous system assessment. Although this test is lightly linguistically loaded, it's difficult because of the similarity in the CVs (pa, ba, ta, da, ka, and ga). In addition, one version of the Dichotic CV test had a 15, 30, 60 or 90 msec delay in the presentation of the second stimulus. Normal individuals improve with a delay of 30 msec or more. However, no improvement with delays was reported in subjects with temporal lobe lesions (Berlin & Lowe, 1972). Investigators have reported either contralateral ear deficits or bilateral deficits with left hemisphere compromise (Berlin & Lowe, 1972; Mueller, Beck & Sedge, 1987).

Monotonic Tone Tests or Temporal Patterning Tests

Although temporal processes are critical in a number of auditory behaviors, there are limited clinical tests used to assess temporal processing abilities. These tests

are based on the assumption that important acoustic signals such as speech vary over time. If a person is to extract meaning from these acoustic signals, the listener must be able to detect very small and rapid time variations. The most commonly used tests are the Pitch Pattern Sequence Test, or Frequency Pattern Test, Duration Pattern Test, and Brief Tone Audiometry.

Pitch Pattern Sequence Test (PPST)

Pinheiro (1977) first reported the use of the Pitch Pattern Sequence Test to assess pattern perception and temporal sequencing skills. The tones consist of a low frequency tone and a high frequency tone. This test is "not designed to assess fine temporal acuity per se but rather to assess the listener's ability to perceive a pattern of auditory events occurring over time" (Bellis & Ferre, 1999, p. 321). Listeners are able to respond in three modes: humming, verbal or pointing to the correct sequence of "low-high" or "high-low" tones. Musiek (1983) reported that learning-disabled individuals could hum the correct response but did not do well in the verbal or pointing modes. Pinheiro (1977) found a significant deficit in the ability of dyslexic children and a control group of normal children. Some investigators have inferred information about the myelination of the corpus callosum when linguistic labeling is involved (Musiek, 1983).

Duration Pattern Test

The Duration Pattern Test (Pinheiro & Musiek, 1985) is very similar to the Pitch Pattern Test. The frequency of the stimulus tones are the same, however, the duration of one of the tones is different from the other two. The listener must respond verbally, humming, or pointing to the correct sequence of "long" and "short" tones.

Gap Detection/ Auditory Fusion Test

Temporal resolution may also be investigated using gap detection thresholds. Gap detection reflects the ability of the auditory system to detect a brief silent interval in white noise. This test requires temporal fusion of the auditory system. Gap detection thresholds may be obtained by using the Random Gap Detection Test, Auditory Fusion Test-Revised (McCroskey & Keith, 1996). Investigators have found larger auditory fusion thresholds in children with language, learning, and reading disorders (McCroskey & Kidder; 1980; Isaacs, Horn, Keith, & McGrath, 1982). Gap detection thresholds systematically decrease with increasing age from three to nine years (McCroskey & Keith, 1996). Gap detection thresholds remain stable throughout adulthood until the fifth decade of life, and then increase with age (McCroskey & Keith, 1996).

Brief Tone Test

One test that has not yet been used in the assessment of APD is the Brief-Tone Test (Cranford, Stream, Rye & Slade, 1982). This test evaluates the ability to discriminate the frequency of short duration tone pulses. Cranford, Thompson, Hoyer & Faires (1997) reported larger frequency difference limens (DLF) in children with protracted histories of middle ear effusion. In addition, this test has been shown to be sensitive in patients with temporal lobe damage (Cranford et al, 1982; Cranford, 1984), in elderly subjects (Cranford & Stream, 1991), and more recently in children diagnosed as having reading disorders (Walker, Shinn, Cranford, & Givens, 2002).

Behavioral Tests Specific to Temporal Processing

The 1996 ASHA Task Force lists the following six behavioral processes in auditory processing: sound localization and lateralization, auditory discrimination, auditory pattern recognition, temporal aspects of audition, including resolution, masking, integration, and ordering, auditory performance decrements with competing acoustic signals, and degraded auditory performance with degraded acoustic signals. However, it is difficult to find one specific auditory behavioral test that will assess only one of these auditory behaviors. Usually, one test may require multiple auditory behaviors. Again, the validity and reliability, and lack of normative data are areas of concern in behavioral assessment of APD (ASHA, 1996).

C. ELECTROPHYSIOLOGIC ASSESSMENT

The use of the ABR in the assessment and diagnosis of APD has been recommended by the 1996 ASHA Task Force on Central Auditory Processing Consensus Development and by the 2000 Consensus Conference on the Diagnosis of Auditory Processing Disorders in School-Aged Children. The primary advantage of electrophysiological measures is that they are objective, requiring no active participation from the listener. The inclusion of electrophsyiologic tests for APD has been controversial. Published research on the auditory brainstem response (ABR), middle latency response (MLR), late latency response (LLR) including the P300 recordings, in subjects with APD has been conflicting and has not shown marked differences. Electrophysiological recordings add expense and time to the evaluation and the results of electrophysiological recordings will not influence the management

plan for the child. However, these objective measures may strengthen the diagnosis of APD.

Bioelectrical activity is generated in both the presence and absence of sensory stimulation. The central auditory pathway is a complex network of organized interconnections between nerves resulting in neuroelectric fields. When neurons are collectively excited, synchronous discharges are evoked resulting in the generation of a measurable overall field (Jacobson, 1985). This electrical activity can be recorded using electrodes placed at either near or distant surfaces.

Auditory evoked potentials have been recognized as a valuable diagnostic tool in assessing the integrity of the central auditory pathway (Jacobson, 1985; Hall, 1992). Electrophysiologic assessment offers objective evidence about the integrity of peripheral and central auditory pathways. These measures are not influenced by extraneous factors and may offer advantages to behavioral tests.

Auditory evoked potentials are classified according to when in time they occur. The auditory brainstem response (ABR) is a short latency response (less than 10 msec post-stimulus onset) which provides objective evidence of the integrity of the auditory brainstem. The middle latency response (MLR), late latency response recordings including the P300 and Mismatch Negativity (MMN) provide information about the cortical and sub-cortical areas of the auditory pathway and will not be included in this review.

Auditory Brainstem Response

The ABR is a far-field potential. This is to say that electrodes are distant from the generator sites. Recording this potential is non-invasive, using scalp electrodes.

The ABR consists of seven major components (waves I through VII) which occur within 10 milliseconds after stimulus onset (Jewett, 1970; Jewett, Romano, & Williston, 1970; Jewett & Williston, 1971). These investigations reported that the waves are the resulting electrical activity of central auditory mechanisms. Waves I and II are associated with neural activity of the VIII cranial nerve and the cochlear nuclei. Wave III is associated with neural activity of the VIII cranial nerve through the superior olivary complex. Wave V is attributed to the lateral lemniscus activity (Jewett & Williston, 1971; Moller, Janetta, & Moller, 1982).

Maturation must be considered when using the ABR as a diagnostic tool. ABRs can be recorded as early as 27 weeks conceptional age. Infant ABR recordings have a much longer latency than adult ABRs (Hecox & Galambos, 1978; Galambos & Hecox, 1978). However, the recording does not reach adult maturity until approximately 18 months (Galambos & Hecox 1978). The decrease in wave latency with increased age is assumed to be a reflection of the maturation of the central auditory nervous system (Hecox & Galombos, 1978; Galambos & Hecox, 1978). Presumably, maturation is influenced by an increase in myelination, and an increase in the number of secure synapses in the auditory pathway.

Although the latencies of the ABR waveforms may reach adult values, Lauter, Oyler, & Lord-Maes (1993) found increased variations in the amplitude of ABR recordings in children. These investigators conclude that ABR characteristics are changing and do not reach adult form until the age of 15. They also suggest these electrophysiological measures are indicative of immaturities in the auditory brainstem.

It is important to note that children with histories of protracted otitis media may

have prolonged ABR wave latencies (Folsom, Weber, & Thompson, 1983; Gunnerson & Finitzo 1991; Gravel & Wallace, 1995; Hurley & Hurley, 1995). Otitis media is often associated with a conductive hearing loss that may produce an "auditory deprivation." This auditory deprivation has been shown to alter the normal development of the auditory structures of altricial animals (animals with an immature auditory system at birth) and produce anatomical and physiological changes in the auditory structures (Webster & Webster, 1977) though results have been less clear in precocial mammals, including humans.

Gender effects have been reported in the ABR. However, the gender effect in infants has been debated. Stockard & Stockard (1979) reported no gender differences in newborns. Contrary to this, Cox, Hack, & Metz (1981) reported shorter latencies in female pre-term infants. Gender differences in adults have been attributed to such factors as better hearing sensitivity, body temperature, smaller head size, brain dimensions in females, and biochemical differences between sexes (Allison, Wood, & Golf, 1983; Michalewski, Thompson, Patterson, Bowman, & Litzelman, 1980; Stockard & Sharbrough, 1980; Hare, Wood, Manyam, Gerner, Ballenger, & Probst, 1982). Additionally, Don, Ponton, Eggermont, & Masuda (1993) reported shorter cochlear response times in the ABR of females than males. They attributed this finding to better neural synchrony. These investigators contend that if one could factor the stiffness gradient of the female cochlea to be 13% greater while the tonotopic organization remains constant; the female cochlea is 13% shorter than the male cochlea. They reported that this hypothesis is consistent with anatomical findings of a shorter cochlea in females.

The sensitivity of the ABR test to detect lesions in the auditory brainstem was first reported by Starr & Achor (1975). Since that time more than 100 clinical papers have reported the use of ABR in identifying space-occupying lesions (Hall, 1992). Advances in imaging (i.e. better sensitivity with MRI and a decrease in the cost of MRI) have replaced the ABR as the test of choice in identifying retrocochlear lesions.

ABR has also been used in the diagnosis of central nervous system disorders such as demyelinating diseases (Jerger, Oliver, Chmiel, & Rivera, 1986), degenerative diseases (Harkins 1981), or asynchronous disorders such as auditory neuropathy/dys-synchrony (Hood, 1998; Starr, Picton, Sininger, Hood, & Berlin,1996). Unlike imaging techniques which have superior spatial resolution and are useful in identifying structural defects, the ABR has excellent temporal resolution and is useful in detecting central auditory nervous system disorders (Hall, 1992). However, it is important to note that for some disorders such as space occupying lesions or multiple sclerosis, the magnetic resonance imaging may be the test of choice.

ABR research findings in APD are conflicting. This could in part be due to the heterogeneity of the subject population. Sohmer and Student (1978) reported abnormal ABR results with children with minimal brain dysfunction. In contrast, Shimizu, Brown, Capute, & Mahoney (1981) reported normal ABRs in children with minimal brain dysfunction. Worthington (1981) reported little or no correlation between ABR results and diagnosed APD children. However, Worthington, Beauchaine, Peters, & Reiland (1981) reported abnormal ABR results with 30% of subjects with moderate to severe language and/or developmental delays. Protti (1983) reported that 2 of 13 subjects identified by behavioral tests had “positive” (abnormal) ABR results. Protti

concluded that ABR may not be sensitive to all brainstem disorders but is probably more appropriate for APD diagnosis than behavioral tests. One of the reasons the ABR may not be different in children with APD may be in part due to the test stimuli employed, which consist of clicks, filtered clicks, tone pips, and tone bursts, rather than speech-like stimuli. Early latency ABR responses merely reflect the auditory mechanism's ability to recognize a signal; not the processing functions reflected by the late potentials (Brugge, 1975).

Recently, Gopal and Kowalski (1999) using the method of slope vectors reported morphological differences in the ABR recordings of children at risk for APD and normal children. The method of slope vectors is a statistical tool used to objectively evaluate morphological differences in the width of the ABR waves by statistically comparing the width of the wave. This investigation stemmed from their clinical experience of visually observing broader, less well-defined ABR waveforms in children with APD. Previously, morphological analysis has not received much attention. "If reliable and valid methods of morphological analysis are developed, gross and subtle alterations in ABR waveform morphology may lead to identifications of differences in the brainstem responses among subjects" (Gopal & Kowalski, p.86). The poor morphology as demonstrated by the APD group may reflect poor synchrony in the central auditory pathway.

Binaural ABR Recordings, Binaural Interaction Component, and Ear Asymmetries

Binaural stimulation in ABR recordings was initially used to enhance wave peaks. Other investigators believed that diagnostic information such as the localization of possible brainstem disorders could be obtained from the binaural recording (Levine,

1981). Binaural stimulation causes changes in ABR recordings. These changes have been reported as 1) an increase in the amplitude of the waveforms, 2) a decrease in the latency of the ABR wave peaks, and 3) morphological changes in the wave form peaks occurring approximately 4 msec post-stimulation (Blegvad, 1975; Davis, 1976).

Kemp and Robinson (1937) were the first to report electrophysiological recordings to binaural stimuli. These recordings were made from electrodes implanted in the lateral leminscus of the cat. They reported a binaural interaction component (BIC) as the difference in the binaural and summed monaural amplitudes and this difference was a consequence of the occlusion effect.

Central neural interaction of the ABR potentials to binaural stimulation was first reported by Jewett (1970). He reported morphological differences in the interaction of the ABR potentials from binaural stimulation in comparison to monaural stimulation in the cat. The binaural wave IV amplitude was smaller with binaural stimulation than the summed monaural responses. Jewett reported that this difference was a result of "convergence at this level or of mutual inhibition from the two ears" (Jewett, 1970, p. 616).

In theory, the amplitude of the right recording added to the amplitude of the left recording should equal the amplitude of the binaural recording. However, this is not the case. In fact, generally speaking, there is a difference waveform which can be derived by subtracting the binaural response recording from the summed monaural recording, or vice versa. This difference waveform usually occurs at a latency in the approximate range of wave V in humans and this difference waveform is thought to show objective evidence for binaural interaction.

Investigations have reported the BIC in humans and in animals (Debruyne, 1984; Dobie & Berlin, 1979; Gardi & Berlin, 1981; Levine, 1981). Other parametric investigations have reported the effect of stimulus intensity (Wilson, Kelly-Ballweber, & Dobie, 1985), interaural stimulus intensity (Dobie & Berlin, 1979: Arslan, Prosser & Michelini, 1981), stimulus rate (Wilson et al., 1985), interaural time delays (Arslan et al., 1981; Berlin and Dobie, 1979; Decker and Howe, 1981; Wrege & Starr, 1981), and ear asymmetries in the binaural ABR (Berlin, Hood, & Allen, 1984; Decker & Howe, 1981, 1982; Levine & McGaffigan, 1983).

In addition, the BIC has been successfully recorded in infants (Hosford- Dunn, Mendelson, & Salamy, 1981; McPherson, Tures, & Starr; 1989). These investigations indicated a present BIC in all subjects. The BIC has also been investigated in children with positive history of frequent conductive hearing loss, attributed to otitis media during infancy (Gunnarson & Finitzo, 1991). This investigation reported difficulty in discerning the BICs in children with positive histories of conductive hearing loss.

Researchers also studied the effect of interaural time differences (ITD) on the BIC. As previously reported in psycho-acoustic studies, click stimuli with ITDs in the 0-1 ms range are perceived as a single sound moving toward the leading ear (Babkoff & Sutton, 1965). With an increase in ITD, two separate sounds are heard (Babkoff & Sutton, 1965).

Dobie and Berlin (1979) investigated stimulus intensity, interaural intensity and interaural time differences in the BIC of guinea pig. They found a BIC in the region of 3.5 to 4 msec region which is consistent with the wave IV. The BIC was still present with a 20 dB interaural intensity difference. They also varied the interaural time

differences from +/-3000 μsec and reported significant changes on the amplitude and latency of the BIC when the interaural time difference was greater than 1000 μsec.

Similar findings of the BIC with ITDs were reported by Wrege and Starr (1981) in human subjects. They investigated ITDs of 50, 200, 500, 900, 1400, and 2000 microseconds. The delayed right monaural response was added to the left monaural response and then subtracted from the binaural response. This report found an increase in BIC latency and a decrease in amplitude as the ITD increased. Several wave peaks were not present when the ITD was greater than 500 microseconds. The BIC was almost non-existent when the ITD was at 900 μsec.

Arslan et al. (1981) also investigated the BIC with interaural time delays of the binaural stimulus. These investigators reported morphological changes in the latency range of 3.5 to 6.5 msec when the ITD was greater than 2 msec.

Furst, Levine, & McGaffigan (1985) investigated the change in perception and the BIC for dichotic clicks with varying ITDs. They found that the first major peak of the BIC was present when the image was fused, this correlated with the perceptual task of lateralization. As the ITD increased, the first wave of the BIC, (β) was degraded.

Few parametric studies of the BIC exist. DeChicchis (1981) examined such variables as gender, age, and intensity on the BIC in humans. He reported the BIC latency which usually occurs in the latency range of wave V, was shorter in the young female group. Dechicchis also employed two independent judges to visually judge the presence or absence of a BIC. He reported poor intra-judge and inter-judge reliability in detecting the presence of the BIC. This lack of agreement is disturbing and may limit the clinical usefulness of this difference trace.

Decker and Howe (1981) also investigated the BIC in two experiments. In the first experiment, the right and left ear responses were added to obtain a predicted waveform. The predicted response was altered by computer manipulation so that it would lead the binaural response in time on some occasions and lag the binaural response on other occasions. A polarity reversal occurred in the difference trace when the binaural trace leads the predicted response, meaning the BIC was negative. The difference trace was positive when the binaural trace lags the predicted.

The second experiment was performed to determine if the binaural response was more dependent upon the right or left response. The left and right ear monaural responses were subtracted individually from the binaural response. Fifty percent of the subjects showed no difference between traces; fifty percent showed differences suggesting an "auditory tract preference". Decker and Howe defined an auditory tract preference when the binaural trace mirrors only one of the monaural recordings. They argue that diotic stimuli are not transmitted symmetrically through the auditory pathway. If the preferred auditory tract has a shorter latency, the difference trace will be in a negative direction. Further, they argue that the difference trace is only valid if there is equal weight in the binaural recording. It is important to note that this investigation has not been substantiated by other investigations.

Berlin et al. (1984) reported asymmetries in the early, middle, and late auditory evoked potentials. They found the asymmetries in the binaural recordings were not related to peripheral differences. They further reported that asymmetries in the BIC were totally dependent upon the right and left asymmetries in the monaural responses.

This is demonstrated by vertex to ear lobe recordings (Cz-A1; Cz-A2) in a binaural stimulation.

The BIC may be a clinically useful tool in evaluating "binaural processes such as localization, lateralization and fusion" (Hall, 1992 p. 163). Binaural electrophysiologic studies may also be used to investigate some of the specialized brainstem processing for binaural hearing (Berlin et al, 1984; Hall, 1992; Wrege & Starr, 1981). In addition, the BIC may be a promising tool in assessing APD. Gopal and Pierel (1999) reported significant differences in the amplitude of the BIC in a group of children with APD. They postulate that this may reflect inadequate binaural inhibitory interactions.

Another interesting finding in the Gopal and Pierel investigation is that four of nine experimental subjects showed no binaural inhibition. In most subjects, when the monaural recordings are added together, the amplitude of the wave V of the summed average is larger than the binaural response. This finding has been substantiated by other investigators who also report the amplitude of the wave V binaural recording is larger than the monaural recordings, but smaller than the summed monaural recordings (Ainslie & Boston, 1980; Debruyne, 1984; Kelly-Ballweber & Dobie, 1984; McPherson et al, 1989; McPherson & Starr, 1993; 1995). Gopal and Pierel (1999) hypothesized that lack of binaural inhibition may reflect "reduced inhibitory processes at higher levels of the auditory brainstem;…it is more than likely that the deficit lies in the functional properties of neurons stimulated binaurally" (p. 83). Further studies are needed.

Recently, Delb, Strauss, Hohenbert & Plinert (2003) reported a decrease in the occurrence of BIC with increasing ITD in both the experimental and control groups. However, there was a significant difference in the occurrence of the BIC between the control and experimental group. This investigation also reports that the indication of the presence or absence of the BIC as an indication of APD achieved a sensitivity and specificity of 76% in this investigation.

One of the difficulties in reviewing the literature of the BIC is the nomenclature and different methods of obtaining the BIC. Several investigations describe a method where the right and left responses were "digitally added" this response is then referred to as the "summed" response. However, a "summed" response on one commercially available evoked potential system is essentially an "averaged" response.

In addition, investigators have not specified whether they have maintained equal number of averages in the derivation process. In other words, if two runs of 2000 click presentations to the right and two runs of 2000 click presentations to the left ear are digitally added, the result will be a total of 8000 monaural click presentations. Investigations do not report the number of click presentations to the binaural response. If this summed response of 8000 monaural click presentations is subtracted from a binaural response, then it would take four runs of 2000 clicks to maintain an equal number of click presentations. This is an important consideration in the signal to noise ratio of this recording. It is important to note that a fixed number of stimuli does not guarantee the same amount of residual noise (Elberling & Don, 1984; Elberling &Wahlgreen, 1985).

D. UNDERLYING PHYSIOLOGY

Temporal processing requires synchronous discharge of neurons of the peripheral and central auditory pathway. In order for a clinician to understand how to assess and manage a child with APD, it is necessary to review how temporal information is transmitted via the peripheral and central auditory pathways. Although this investigation will use both behavioral assessment and electrophysiological measures, an overview of the anatomy through the auditory cortex is presented.

Sound undergoes complex processing by intricate neural mechanisms and neural networks. These mechanisms and networks are composed of structures located in the brainstem, subcortex, primary and association areas of the auditory cortex and the corpus callosum. These structures are responsible for transmitting, enhancing or inhibiting, reshaping, refining and assigning recognition and meaning to the once air-borne vibrations. Much of the auditory processing is a preconscious event. However, the result is an auditory perceptual event (ASHA, 1996).

Auditory Periphery

The displacement of the basilar membrane is reflected in the excitation pattern of the VIII nerve. Fibers innervating the basal end of the cochlear are sensitive to or "tuned" to high frequencies. Fibers innervating the more apical end of the cochlea are tuned to low frequencies.

Temporal information about the stimulus is preserved in the neuronal firing patterns. Although neurons do not fire on every cycle of the stimulus, they fire at the same phase of the waveform. This synchronization is precise enough to pass intelligible speech. Rose, Galambos & Hughes (1960) report the upper limit of phase locking to be

around 4-5 kHz and further suggest that the phase-locked response is the way neurons code for monaural pitch and binaural localization (Rose, Kitzes, Gibson & Hind, 1974).

The auditory division of the VIII nerve is composed of type-I and type-II fibers. The type-I fibers are myelinated and the majority (88%) synapse with inner hair cells (Webster, 1995). The type-II fibers are unmyelinated and synapse with outer hair cells. The VIII cranial nerve enters the brainstem at the level of the cerebellopontine angle (CPA). Each fiber divides and sends branches to the three divisions of the cochlear nucleus.

Cochlear Nucleus

Representation of ipsilateral frequency, timing, and level cues are maintained in the cochlear nucleus. The cochlear nucleus (CN) has three divisions: the anterior ventral cochlear nucleus (AVCN), the posterior ventral cochlear nucleus (PVCN), and the dorsal cochlear nucleus (DCN). The CN is tonotopically organized. In other words, frequency representation is highly organized. The CN neurons receive input from the ipsilateral auditory nerve. In each division of the CN, a different type of cell is predominant. These different types of cells have different response patterns to sound stimulation. These response patterns include primary-like, Onset, chopper, pauser, and build-up responses (Pfeiffer, 1966).

The temporal features of a sound are exhibited in the firing pattern response of cochlear nucleus neurons (Stillman, 1980). The phase-locking ability of the AVCN is similar to the cochlear nerve fibers (Rose et al., 1974); Moushegian, Rupert, & Whitcomb, 1964). The neural firing pattern demonstrates temporal resolution at this early stage of processing in the brainstem.

Superior Olivary Complex

The majority of the fibers from the CN decussates and terminates in the superior olivary complex (SOC). The SOC is composed of several different groups of nuclei. However, only the three which are important to auditory processing will be reviewed in this paper. The lateral superior olivary nucleus (LSO) receives ipsilateral input from the AVCN and contralateral innervation from the contralateral AVCN and PVCN via the medial nucleus of the trapezoid body (MNTB). The medial superior olivary nucleus (MSO) receives input from the ipsilateral and contralateral AVCN. The MNTB receives the majority of its innervation from the contralateral CN.

The SOC is the first place in the auditory pathway where binaural integration occurs. This demonstrates an anatomical basis for binaural listening (Willeford & Burleigh, 1985). Binaural hearing has advantages. It improves performance in most auditory tasks and is essential in other auditory tasks. Hearing with two ears makes localization possible. Interaural time differences and interaural intensity differences are critical cues in localization on the horizontal plane. Interaural time differences and interaural intensity differences are reflected in SOC firing patterns, reflecting information used in localization, a process underlying many auditory tasks (Masterson, Thompson, Bechtold, & Robards, 1975; Boudreau & Tsuchitani, 1970). Discrimination of sounds in a noisy environment is also improved with binaural hearing. In addition, binaural hearing helps in selective listening to one speaker, the so-called "cocktail party" effect.

Lateral Lemniscus

The lateral lemniscus (LL) is the primary auditory pathway in the brainstem. It is composed of three nuclei, the dorsal, ventral, and intermediate. The lateral lemniscus contains fibers arising bilaterally from the SOC. The right and left lateral lemnisci are connected by Probst's commissure. The lateral lemnisci are generally thought to be transmission lines for ascending and descending fibers through the brainstem. Tonotopic organization is maintained in the lateral leminiscus as low frequencies are represented in the dorsal lateral lemniscus and high frequencies are represented in the ventral lateral lemniscus. Neurons in the dorsal lateral lemniscus respond to binaural stimulation, and neurons in the ventral nucleus respond to contralateral stimulation.

Inferior Colliculus

From the lateral lemniscus, neural fibers progress to the inferior colliculus (IC). The IC is the largest auditory structure in the brainstem (Oliver & Morest, 1984). The IC is an "obligatory relay nuclear complex" which transmits auditory information to higher levels (Noback, 1985) and it receives descending information from the auditory cortex. The IC is composed of a central area, which contains auditory fibers and the pericentral nucleus, or belt, which contains somatosensory and auditory fibers. The commissure of the IC connects the right and left IC. The IC also receives fibers from the CN and SOC in addition to the contralateral IC.

The IC is also tonotopically organized (Merzenigh & Reid, 1974). Investigators report good frequency resolution from neural fibers at this level in the brainstem (Aitken & Webster, 1972; Aitken, Webster, Veale, & Crosby, 1975). In addition, many

neurons of the IC are time-and spatially sensitive (Pickles, 1988; Knudson & Konishi, 1978) and sensitive to binaural stimulation (Benerento & Coleman, 1970).

Medial Geniculate Body

The medial geniculate body (MGB) is a thalamic-level structure. It is divided into three nuclei: the ventral, dorsal, and medial. Neurons in the ventral medial geniculate body respond to acoustic stimulation, while other divisions respond to both auditory and somatosensory stimulation (Pickles, 1988). The dorsal and medial divisions of the MGB receive projections from pericentral IC and the medial division of the MGB receives projections from the SOC, and nuclei of the LL.

Tonotopic organization has been shown in the ventral portion of the MGB (Aiken & Webster, 1972). Many cells in the MGB are binaurally sensitive and also respond to interaural intensity disparities (Aiken and Webster, 1972). Keide, Kallert, Korth & Humes (1983) theorize that the MGB may play an important role in the processing of speech due to the neuronal firing response pattern to frequency modulation.

Reticular Formation

The auditory system is connected to the reticular formation (RF), or reticular activating system. The RF is involved in altering the level of consciousness and in sustaining the function of consciousness (Moruzzi & Magoun, 1949). The RF is responsible for "alerting" the brain to incoming stimuli, but is not limited to auditory stimuli.

Ayres (1972) theorizes that children with learning disabilities may have a RF which fails to discriminate between stimuli that should be processed and those that

should be inhibited, thus creating a sensory overload which interrupts processing. Because the RF is also a multi-sensory system, the ability to attend to an auditory stimulus may be interrupted if there are changes in the environment (Ayres, 1972). Therefore, researchers hypothesize the RF may play an important role in listening in noise (Chermak & Musiek, 1997).

Auditory Cortex and Auditory Association Cortex

Auditory information is projected from the MGB through the internal capsule and into the primary auditory cortices. The primary auditory cortex is located on the transverse gyri of Heschl, in the Sylvian fissure on the superior surface of the temporal lobe. The primary auditory cortex is tonotopically organized.

Auditory information processed in Brodmann's areas 41 and 42 is then passed to area 22. Area 22 is an association auditory cortex. Area 22 lies within the posterior two-thirds of the superior temporal gyrus and the planum temporale.

A large tract called the arcuate fasciculus is made up of projection neurons from area 22 and some adjacent temporal lobe areas. The arcuate fasciculus brings information to the inferior cortex or Brodmann's areas 44 and 45, which is also called Broca's area. This area is responsible for the motor processing of speech. On route to Broca's area, the arcuate fasciculus "interacts with the angular and supermarginal gyri" (Webster, 1995, p. 256). The angular area 39) and supermarginal (area 40) gyri are multi-modality association cortices capable of integrating what is heard, what is seen, and what is felt (Webster, 1995).

Geshwind (1979) and Geshwind and Levitsky (1968) have reported asymmetries in the planum temporale. In most humans, the left planum temporale is much larger.

This finding is related to the fact that in most people area 22 plays a role in speech comprehension. Netsky (1986) reports that this asymmetry is evident in fetuses as early as 29 weeks gestation, suggesting that the substrate for language development is present before birth. Anatomical differences are also found in the Sylvian fissure, which is found to be larger in the left. Additionally, the number of Heschl's gyri in each hemisphere may range from one to three and may differ between hemispheres (Musiek & Reeves 1990).

Binaural representation is evident in each temporal lobe with each hemisphere receiving projections from both ears. There is still much unknown about the function of the auditory cortex and research continues on the effects of cortical deficits. The auditory cortex may be involved in a number of auditory tasks. Pickles summarized eight hypotheses of auditory cortex functions as follows:

> "(1) it may be necessary for the analysis of complex sounds;
> (2) it subserves sound localization and the representation of 'auditory space'
> (3) it is necessary for selective attention to auditory stimuli on the basis of source position
> (4) it serves to inhibit inappropriate motor responses:
> (5) it serves to identify stimuli on an absolute basis:
> (6) it is necessary for the discrimination of auditory temporal patterns;
> (7) it is necessary for short-term memory when one auditory stimulus has to be related to another later in time
> (8) it is necessary for auditory tasks that are difficult" (Pickles, 1988, pp 233-234.)

Corpus Callosum

The corpus callosum carries information from the right and left hemispheres and connects the right and left auditory cortices. The corpus callosum is responsible for transmitting information between the two hemispheres. The auditory area lies anterior to the splenium in the posterior half (Chermak & Musiek, 1997). Some auditory tasks,

such as dichotic listening tasks require that information from the two hemispheres be integrated. Recent investigations report a decrease in the size of the corpus callosum in individuals with attentional deficits in comparison to normal subjects (Hynd, Semrud-Lorys, Novey, Eliopulos, 1990). Differences in the size of the corpus callosum have also been reported in children with specific language impairments (Leonard, Eckert, Lombardino, Morris, Hynd, Alexander, et al., 1993; Leonard, Eckert, Lombardino, Oakland, Kranzler, Mohr, et al., 2001).

Maturational Factors

Maturation affects the function of the central auditory pathway. The peripheral auditory system matures before the central auditory system (Stockard & Stockard, 1979, 1983). Maturation of the auditory pathway occurs in a caudal to rostral manner (Chermak & Musiek, 1997). Myelination, arborization and synaptogenesis affect maturation (Chermak & Musiek, 1997). Myelination of the central auditory pathway occurs at different times for different regions.

Yakovlev and LeCours (1967) describe the myelination patterns using a Loyez (silver) staining technique. They report myelination of the prethalamic auditory tracts are complete by 5-6 months chronological age. The post-thalamic pathways are not completely myelinated until 5-6 years and the corpus callosum and other auditory association areas do not complete myelination until 10-12 years, or older.

This is also reflected in the latency of the auditory evoked potentials. Early waveforms, such as the auditory brainstem response (ABR) reach adult values by age two (Hecox and Galambos, 1974). However, later waveforms do not reach adult maturity until adolescence (Musiek & Gollegly, 1988). Additionally, amplitude

measures of the ABR waveforms do not reach adult values until puberty (Lauter, et al. 1993). This investigation supports Musiek & Gollegly's report that myelination of the auditory brainstem is continuous throughout childhood and not complete until puberty.

Myelination may affect performance on auditory tasks and "it is likely that the difference in children's performance on certain auditory tests may be related to differences in the amount of myelination in critical regions of the brain" (Chermak & Musiek, 1977, p. 63). It is important to consider that the rate of myelination may also be variable (Yakovlev & LeCours, 1967; More, 1983). As neurons mature, branching of the axons and dendrites occurs. "In the very early maturational course, growing axons make their way to specific areas of the immature brain. After reaching its destination the axon develops branching (i.e., arborization) and each branch has a bulbus termina. These bulbs in turn make synapses with dendrites (Kalil 1989)" *as cited in Chermak & Musiek, p. 64.* Arborization may be influenced by maturity, or other environmental factors such as stimulation and deprivation (Kalil, 1989).

E. SIGNIFICANCE

There is a clear need for research in the assessment of APD. This investigation is timely. There is a significant lack of data reporting both behavioral and electrophysiological measures in children with temporal processing deficits. This investigation will be a first-step in addressing some of the confounding issues. In summary this investigation will answer the following:

1. Is there dys-synchrony in the peripheral nerve and auditory brainstem pathway in children at risk for APD, as evidenced by differences in the ABR?

2. Is there dys-synchrony in the peripheral nerve and auditory brainstem pathway in children at risk for APD, as evidenced by differences in the BIC and BIC with increased interaural time differences?

CHAPTER 3

METHODS

A. SUBJECTS

The subject pool for this research was comprised of 24 experimental and 24 control male subjects between the ages of 7 and 12 years of age. Four potential subjects were excluded; one had abnormal middle ear measurements; one had a sensorineural hearing loss, and two did not meet the behavioral criteria addressed in a later section. Gender specificity was imposed because of the gender effect on wave latency in the electrophysiologic recordings (Cox et al., 1981). All subjects gave informed assent and had parental or legal guardian consent, as approved by this university's Institutional Review Board. (See Appendix C.) All subjects had normal peripheral hearing as assessed by normal pure tone audiometric thresholds from 500 to 4000 Hz $\leq$ 15 dB HL (re: ANSI, 1989) and normal middle ear pressure and static admittance as evidenced by normal (type A) tympanograms.

Behavioral and elecrophysiological data were collected on 24 males who are potentially at risk for APD, as evidenced from self-referrals to the Louisiana State University Health Sciences Center Speech and Hearing Clinic for an APD evaluation. The experimental subjects had abnormal scores on at least three behavioral tests, described in the following section. These behavioral tests employ specific temporal processing skills.

Twenty-four age-matched males comprised the control group. The control group was recruited from families and friends of the LSUHSC Department of Communication Disorders faculty and staff. Members of the control group were allowed to have only

one abnormal score on one of the behavioral tests for temporal processing; all other behavioral scores were within normal limits. In addition, the parent or legal guardian of the control subjects answered, "No." to the following questions:

1. Has your child ever failed a speech or language screening?
2. Has speech-language therapy ever been recommended for your child?
3. Has your child ever been diagnosed with attention deficit or attention deficit with hyperactivity disorder?
4. Has your child ever been referred to a reading or educational specialist?

Additional demographic information from both groups was obtained. The educational level of the mother and father was obtained and grouped into five categories: 1) did not finish high school, 2) finished high school, 3) some college; 4) college graduate, and 5) post-graduate degree. Information about the type of school each subject attended was also obtained. The type of the school each subject attends was obtained and grouped into four categories: 1) public school, 2) private school, 3) parochial school, and 4) home-schooled.

B. DESCRIPTIVE TESTS

In order to further describe the subjects in this study for audiences such as audiologists, speech-language pathologists, and educational and reading specialists, a commonly used screening instrument for auditory processing disorders and two language tests was administered.

SCAN-C

The Screening Test for Auditory Processing in Children (SCAN-C) is a widely used screening instrument for APD. The rationale for administering this test was that it

would aid in describing the subjects. The SCAN-C does not have a subtest for temporal processing. The SCAN-C determines auditory development in children and screens for efficient and inefficient auditory processing performance (Keith, 1996). The SCAN-C contains four subtests, Filtered Words, Auditory Figure Ground, Competing Words, and Competing Sentences. The raw score, standard score and percentile rank for each subject was reported for each sub-test.

Language Tests

To further describe the subjects, two commonly used language tests were administered: the Peabody Picture Vocabulary Test-III (Third Edition) (PPVT-III) and two subtests of the Oral and Written Language Scales (OWLS). The PPVT-IIII is designed as a measure of an individual's receptive vocabulary. In addition, it is an achievement test of the level of a person's vocabulary acquisition. The Listening Comprehension subtest of the OWLS is designed to measure the understanding of spoken language. The Oral Expression Scale is designed to measure the understanding and use of spoken language.

C. EXPERIMENTAL MEASURES

Behavioral Tests

Behavioral testing was administered in a sound treated room. All behavioral tests, with the exception of the MLD, were digitally recorded on commercial compact discs. The clinical audiometer, Interacoustics 40 was be calibrated to the 1000 Hz calibration tone on each individual CD before administering the behavioral tests. The recorded stimuli will be routed through the clinical audiometer and presented at 55 dB

HL and delivered through EAR 3A insert earphones. The presentation order for the behavioral tests was counterbalanced to eliminate any order effect.

Masking Level Difference

The Masking Level Difference (MLD) was derived by measuring the masked threshold for a 500 Hz tone. Thresholds were obtained for S_oN_o (homophasic) and $S_\pi N_o$ (antiphasic) conditions. The 500 Hz pure tone signal was generated using the Interacoustics 40 audiometer. The narrow band noise, also generated by the Interacoustics 40 audiometer, had a 146 Hz band of noise centered at 500 Hz with a 12 dB per octave roll-off. The 500 Hz signal was set to70 dB HL. Signal attenuation of the narrowband noise was in 1 dB steps. Thresholds were obtained by averaging the threshold of four ascending and four descending trials for a total of eight trials. The MLD was defined as the difference in threshold between homophasic and antiphasic stimuli. The MLD was considered abnormal if it is less than 10 dB (Sweetow and Reddell, 1978; Roush and Tait, 1984).

Pitch Pattern Test

The child version of the Pitch Pattern Test, which requires auditory discrimination, temporal ordering and pattern recognition, was administered. This test is digitally recorded on a compact disc and is available from AudiTec of St. Louis. This test consists of 120 pattern sequences made up of three tone bursts, two are the same frequency and one is different. The pure tones were 1122 and 880 Hz. The subject repeated the pattern by verbalizing “high or low”. Thirty monaural trials were presented at 55 dBHL. This test was scored on the percentage correct. The subject’s

scores were compared to the normative cut-off data by Musiek (1985) and interpreted as normal or abnormal. Normative data are provided in Appendix B.

Duration Pattern Test

The duration pattern test is very similar to the Pitch Pattern Test. This test also requires temporal ordering and pattern recognition. This test is digitally recorded on a compact disc and is available from AudiTec of St. Louis. However, the tones do not vary in frequency, but vary in duration as being either long (500) ms or short (200) ms. Thirty monaural trials were presented at 55 dBHL. The subject repeated the pattern by verbalizing "long or short". The test was scored on the percent correct. Again, the subject's scores were compared to the normative cut-off data by Musiek (1985) and provided in Appendix B.

Discrimination of Time Compressed Speech

Time compression alters the temporal characteristics of speech by reducing the duration of the signal without affecting the frequency characteristics (Fairbanks, Everitt, & Jaeger, 1954). Time compressed (45%) NU-6 word lists were presented monaurally at 55 dB HL. Test scores were reported as percent correct. Discrimination scores above 90% were considered normal (Beasley, Schwimmer, and Rintelmann, 1972).

Gap Detection

Gap detection thresholds were measured by the Random Gap Detection Test. This test requires temporal resolution of the auditory system. The Random Gap Detection is a revision of the Auditory Fusion Test-Revised. This test is digitally recorded and is available from AudiTec of St. Louis. This test consists of a calibration tone, a practice subtest and four subtests at 500, 1000, 2000, and 4000 Hz. Each pure

tone is seventeen msec in duration. Stimuli with interstimulus intervals of 0, 2, 5, 10, 20, 25, 30, and 40 milliseconds were randomly presented. Stimuli were presented binaurally at 55 dBHL. The gap detection threshold was the lowest interval where the subject consistently identified two tones, rather than one tone. A composite gap detection threshold was obtained by averaging the gap detection threshold at 500, 1000, 2000, and 4000 Hz. Composite thresholds greater than 20 msec indicate temporal processing deficits that could interfere with speech perception and phoneme recognition (McCroskey and Keith, 1996). Keith (2000) reported that some children with temporal processing difficulties will be unable to hear the gap or respond appropriately; therefore, the test should be terminated after three unsuccessful practice sessions. An abnormal result on this test was a composite gap detection threshold that was greater than 20 mec or the inability to respond appropriately on three practice sessions.

D. GROUP COMPARISON OF DESCRIPTIVE AND BEHAVIORAL TESTS

A Chi-Square analysis was employed to determine if there were significant differences between the group's demographic data. An analysis of variance (ANOVA) was used to determine if significant differences exist in the SCAN-C subtests, OWLS subtests, and in the MLD and Gap Detection Threshold Tests. Behavioral tests were interpreted as normal or abnormal. A Chi-Square analysis was used to determine if there are differences in the behavioral tests.

E. ELECTROPHYSIOLOGIC RECORDINGS

Electrophysiologic recordings were obtained while the subject rested comfortably in a chair and watched silent videos (animated videos with captioning) or played a hand-held video game with no audible sound.

Stimulus

Testing was completed using the Tucker Davis Workstation System III. Test stimuli consisted of 100 μsec condensation clicks with a rate of 11.1 /sec, presented at 70 dB peak SPL via insert ER3A earphones.

Two stimulation sequences consisting of 2000 click presentations were recorded for each test situation. Therefore, each test situation had a total of 4000 presentations. The protocol consisted of two recordings of right, left and binaural (diotic) stimulations of 2000 clicks. Additionally, dichotic conditions in which the right stimulus is delayed by ITD intervals of .1, .4, .9, and 1.9 ms were obtained. Conditions were counterbalanced across subjects to reduce order effects.

Recordings

Recordings were made with five surface electrodes attached to the skin at the vertex (positive), ipsilateral mastoid (negative), and nape of the neck. The forehead served as ground for all recordings. Electrode impedance was below 5 kΩ. Three channel recordings were obtained: 1) vertex to mesial earlobe, 2) vertex to contralateral ear, and 3) vertex to midline (Cz-Oz). The forehead (Fpz) served as ground. The response was averaged over a 12 msec window. The response was amplified and filtered (bandpass 10-3000 Hz). The 10 Hz cut-off filter was chosen to enhance wave V amplitude. Artifact rejection was employed.

Measures

Peak-to-trough amplitude and latency of Waves I, III, and V were measured for each subject in the ipsilateral and mid-line channels. Each recording was then

compared to published, normative data (Joseph, West, Thornton, and Hermann, 1987; Musiek, Josey, & Glasscock, 1986) for each subject.

F. STATISTICAL ANALYSIS OF THE ABR

A repeated measures analysis of variance was employed to determine if there are any differences in amplitude and latency between the control and experimental groups. This analysis addressed Specific Aim 1 to determine if there were differences between the experimental and control groups in the ABR recordings. Additional independent variables will be mode of stimulation, and recording site. An alpha level of .05 will be used to determine statistical difference.

BIC and BIC with ITDs

The BIC was derived by digitally adding the averaged midline monaural right and averaged midline monaural left recordings and subtracting the averaged binaural response. BICs were derived for five conditions, one diotic and four dichotic ITDs of .1, .4, .9, and 1.9 msec with the right ear as the lag ear. The right monaural recording was digitally shifted to match the ITD of the binaural recording. Because of system noise problems, the binaural difference trace was digitally low-pass filtered with a corner frequency at 1000 Hz using the Biosig BioAmp Filters, which have a 40 dB per octave roll-off.

Two reviewers, the investigator and an independent examiner, reviewed the derived waveform to determine if the BIC was present. . In order to aid in the detection of the BIC, a two millisecond window was marked, 0.5 msec before the wave V and 1.5 post wave V. The examiners visually inspected the binaural difference waveform to determine if: 1) there was a positive peak in the 2 msec region, 2) if there was a

negative peak or trough in the 2 msec region, or 3) if there was no binaural interaction present. If the two investigators disagreed about the presence or absence of the binaural interaction component, a third independent judge examined the questionable binaural difference waveform.

A quantitative analysis was employed to determine if there were significant differences in the occurrence of the binaural interaction component between the control and experimental group.

CHAPTER 4

RESULTS

An important consideration before beginning this investigation was to recruit children for the control and experimental groups that were similar in age and socioeconomic level. The control group has a mean age of 8 years and 6 months. The experimental group has a mean age of 8 years and 8 months. An analysis of variance (ANOVA) indicates no significant differences in age between the two groups [F(1, 46)=.143, p=.707]. A Chi Square analysis indicates no significant differences between the education level of the mother [X^2 = 1.66, 3, p=.645], educational level of the father, [X^2 = 2.462, 4, p=.651], or the type of school the subject attends [X^2=1.667, 3, p=.644]. Therefore, there are no statistical differences between the two groups in demographic composition. Individual demographic data are included in Appendix B.

A. DESCRIPTIVE TESTS

Another goal in this study was to recruit and test similar experimental and control subjects who had normal receptive vocabulary as evidenced by their standard scores on the PPVT-IIII. The experimental group has a mean score of 102.33 and the control group has a mean score of 115.29. Although both groups have clinically "normal" scores, an analysis of variance indicates a significant difference between the two groups [F(1, 46) = 15.396, p=.001]. The mean and standard deviation for the PPVT-III and the OWLS subtests are displayed in Figure 1.

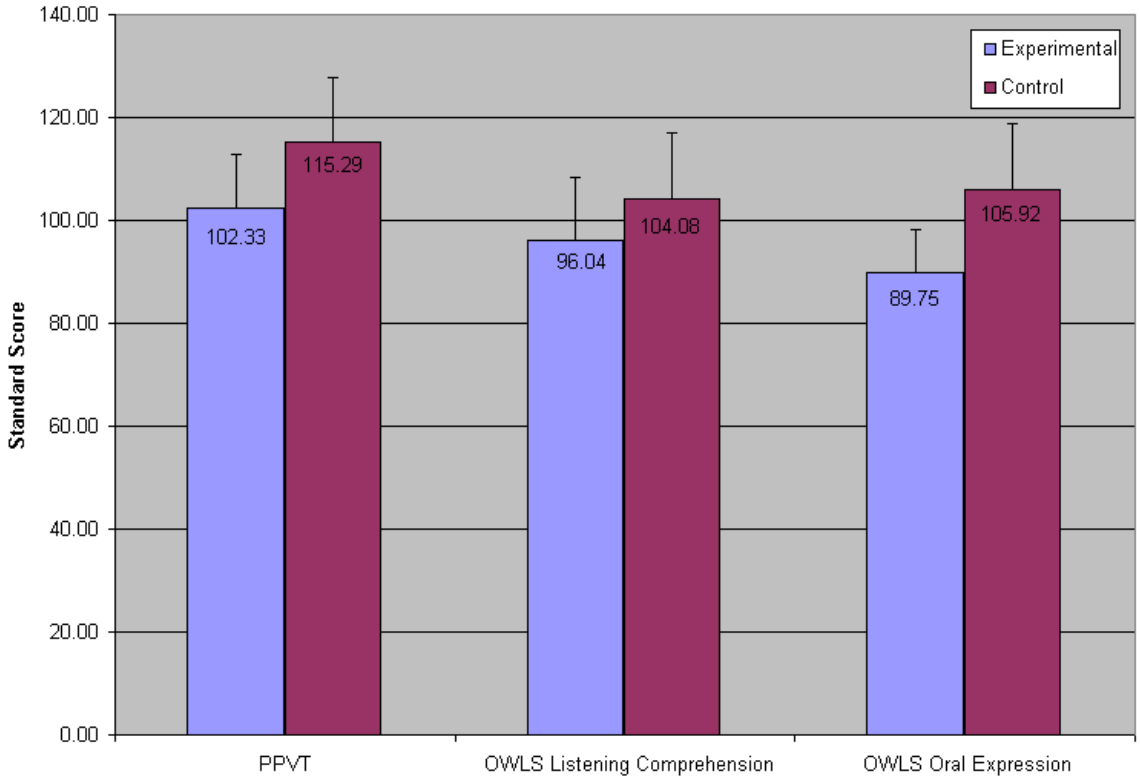

Figure 1. Means and standard deviations for the control and experimental groups on the language tests.

The Listening Comprehension and Oral Expression subtests of the OWLS were also administered. The experimental group has mean scores of 96.04 and 89.75 for the Listening Comprehension and Oral Expression subtests, respectively. The mean score for the control group is 104.08 and 105.92 for the Listening Comprehension and Oral Expression subtests, respectively. An analysis of variance (ANOVA) indicates significant differences between the groups for the Listening Comprehension subtest [F (1, 46) = 4.830, p=.001] and the Oral Expression subtest [F (1, 46) =26.125, p=.001].

The mean standard score and standard deviation for each SCAN-C subtest are listed below in Table 1. An analysis of variance indicates a significant

difference between the Filtered Words [F (1, 46) =8.902, p=.005], Auditory Figure Ground [F (1, 46) = 22.37, p=.0001], and Competing Words subtest [F (1, 46) =25.470, p=.0001]. However, there is no significant difference in the Competing Sentences subtest [F (1, 46) =2.09, p=.155].

Table 1. Mean scores and standard deviation for the control and experimental groups on the SCAN-C.

	Filtered Words	Auditory Figure Ground	Competing Words	Competing Sentences
Control Mean	11.46	9.96	11.04	10.17
Control St.Dev	1.74	1.94	1.99	1.99
Experimental Mean	9.21	6.63	7.79	9.04
Experimental St.Dev	3.26	2.86	2.45	3.25

B. BEHAVIORAL TESTS OF TEMPORAL PROCESSING

For statistical comparisons, the behavioral tests for temporal processing are interpreted as either normal or abnormal. Each subject's individual test scores are listed in Appendix B. In addition, the reference for scoring and interpreting each behavioral test is also listed in Appendix B. A Chi Square analysis, shown in Table 2, indicates significant differences between the control and experimental groups for each of the behavioral tests. This finding suggests that the two groups differ in their temporal processing.

Table 2. Chi Square analysis for behavioral tests of temporal processing in the control and experimental groups.

Test	X^2	df	p
Time Compressed Speech Right	9.36	1	.002
Time Compressed Speech Left	19.05	1	.001
Pitch Pattern Right	40.33	1	.001
Pitch Pattern Left	27.00	1	.001
Duration Pattern Right	27.19	1	.001
Duration Pattern Left	16.45	1	.001
Masking Level Difference	12.63	1	.001
Random Gap Detection	31.45	1	.001

C. ELECTROPHYSIOLOGIC MEASURES

A repeated measures analysis of variance indicates no significant differences in wave latency between the control and experimental group [F (2, 27) =1.25, p=.303]. No significant latency differences are found between the right, left, or binaural modes of stimulation [F (2, 2), = 1.639, p=.208] in this investigation.

The control group exhibits greater amplitude measurements for ABR waves I, III, and V than the experimental group. The amplitudes are significantly different for Waves I and III in the right, left, and binaural modes. The results of the Least Significant Difference post-hoc test are displayed in Table 3. The mode of stimulation is also significant [F (2, 27 = 8.105, p=.001]. Greater amplitudes were obtained for binaural stimulation for both groups.

Table 3. The results of the Least Significant Difference post-hoc analysis for the amplitude of the Cz-earlobe ABR recordings.

Wave	df	p
Right I	1	.005
Right III	1	.004
Right V	1	.056
Left I	1	.020
Left III	1	.006
Left V	1	.115
Bin I	1	.039
Bin III	1	.003
Bin V	1	.173

Wave I group mean latency and amplitude for the Cz- ipsilateral ear lobe trace for the right, left, and binaural stimulation modes are displayed in Figure 2 and Figure 3, respectively. Greater amplitude is noted in binaural stimulation for both groups. The amplitude of Wave I is significantly greater in the control group. (See Table 3.)

Wave III group mean latency and amplitude for the Cz- earlobe trace for the right, left and binaural stimulation traces are displayed in Figures 4 and 5, respectively. Interestingly, the experimental group has shorter wave latencies. The control group has significantly greater wave III amplitudes. (See Table 3.) Waveform amplitude is greater for binaural stimulation than monaural stimulation for both groups.

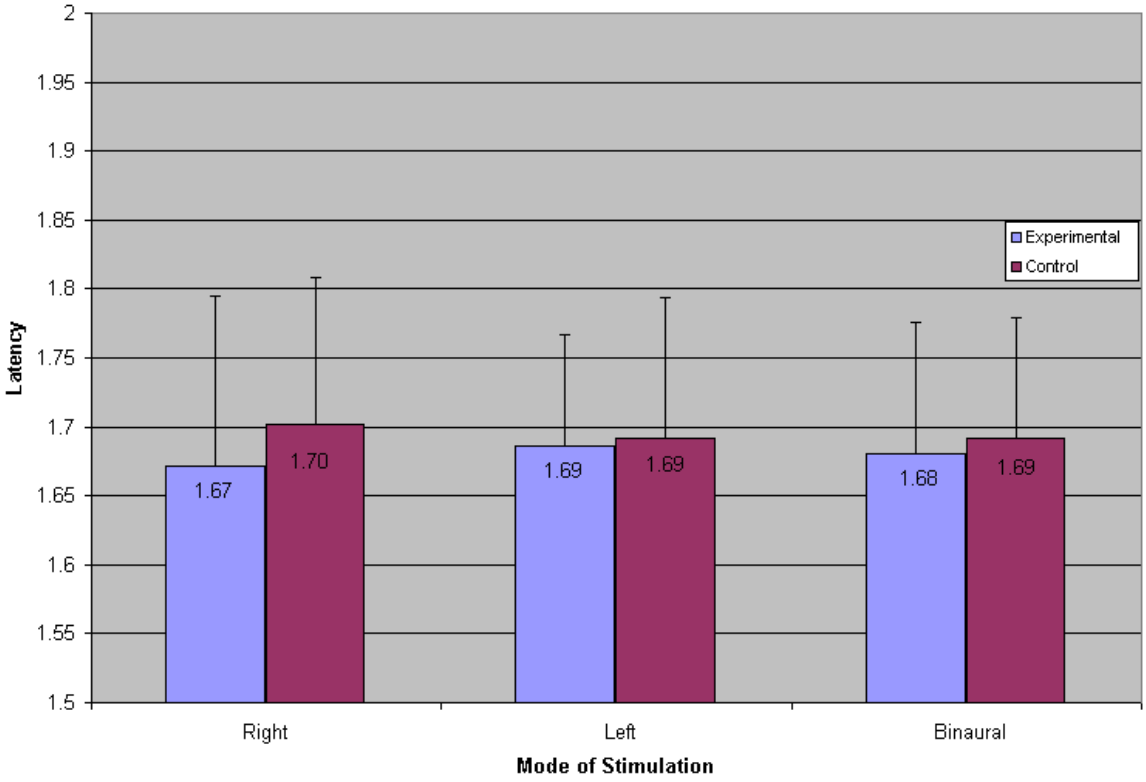

Figure 2. Group means and standard deviations for Wave I latency for right, left and binaural stimulation.

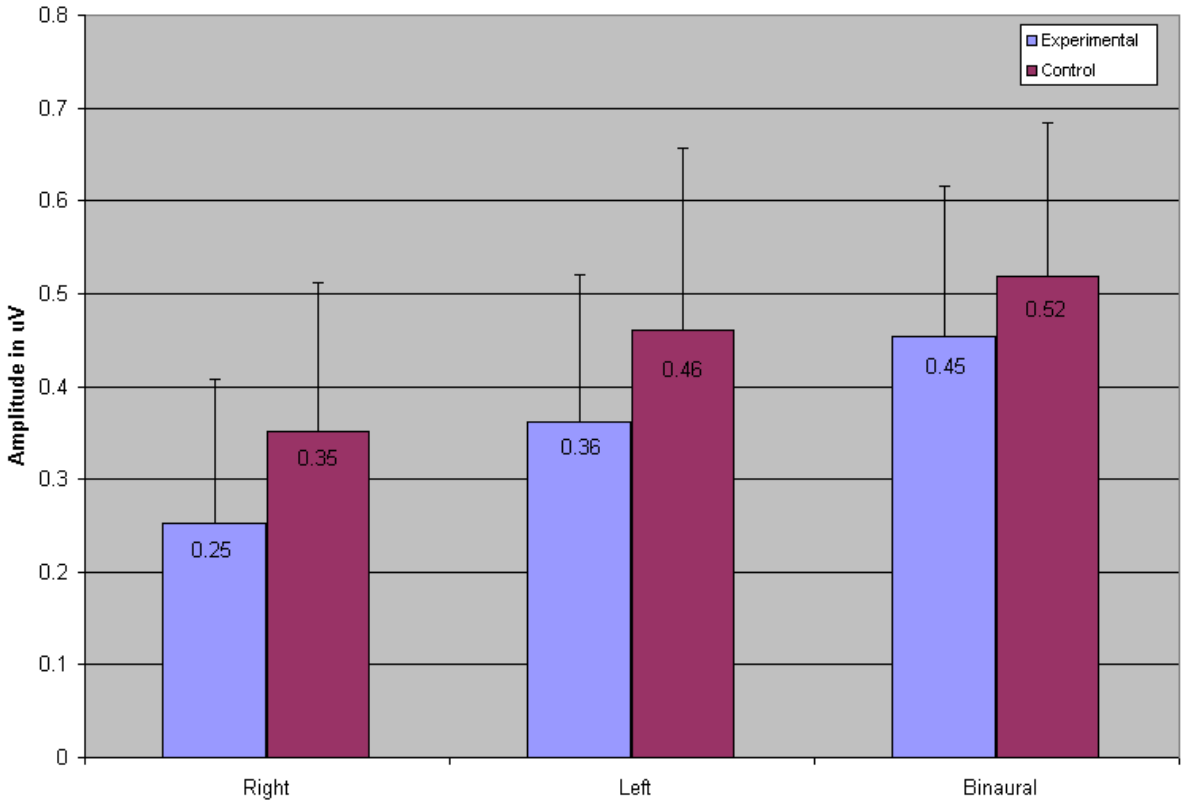

Figure 3. Group means and standard deviations for Wave I amplitude for right, left and binaural stimulation.

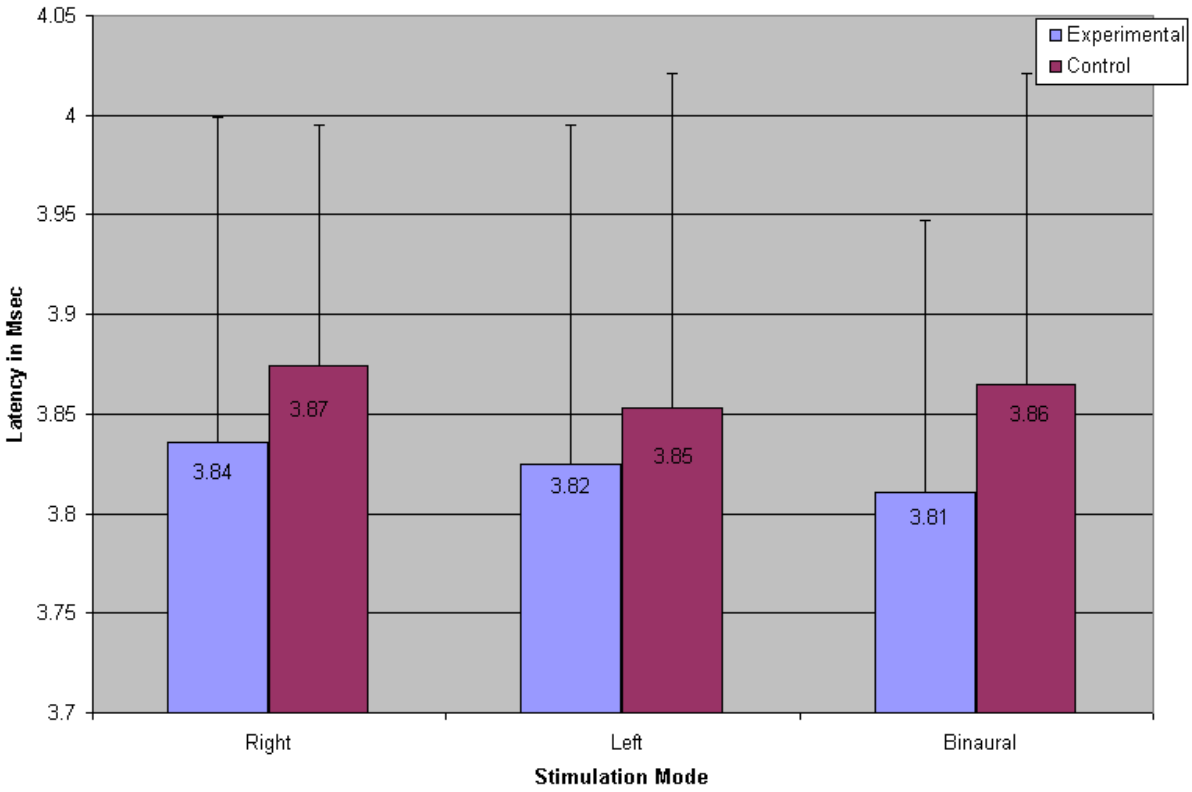

Figure 4. Group means and standard deviations for wave III latency for right, left and binaural stimulation.

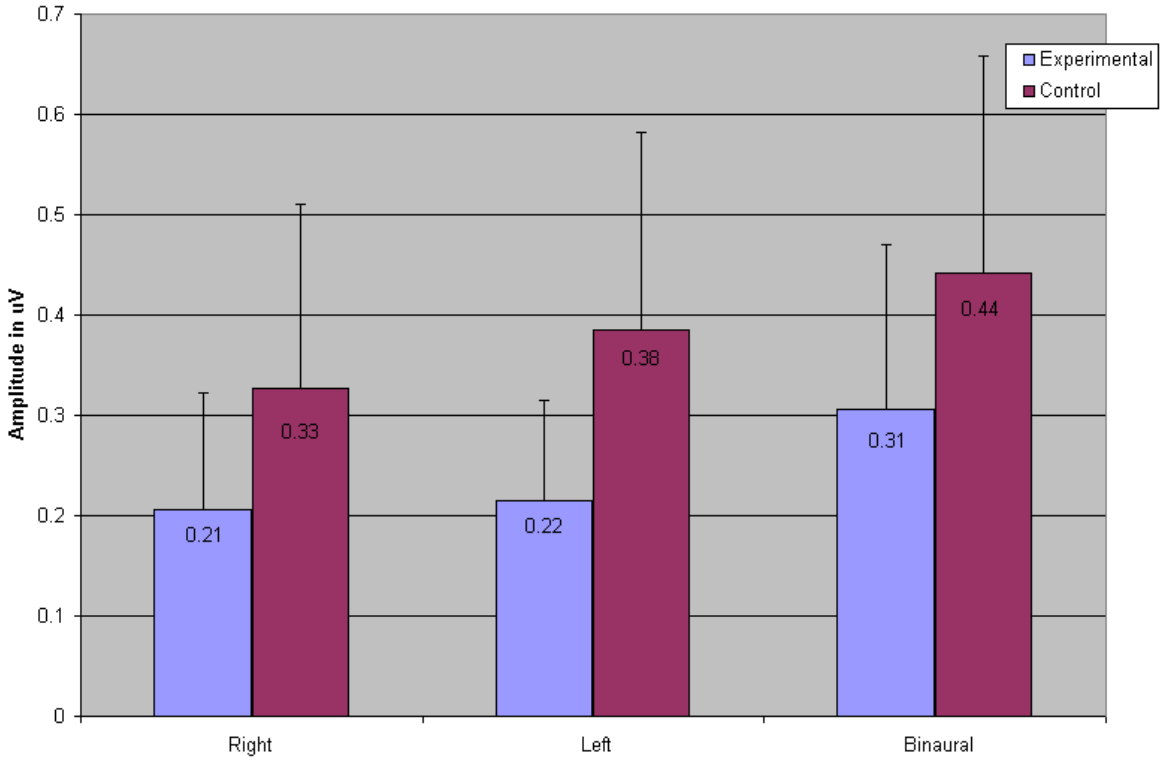

Figure 5. Group means and standard deviations for Wave III amplitude for right, left and binaural stimulation.

Group mean latency and amplitude measurements for wave V in the Cz-earlobe recording are displayed in Figures 6 and 7, respectively. No statistical difference in wave latency is indicated. The control group has greater wave V amplitudes than the experimental group, although not significant. (See Table 3.) The binaural mode of stimulation exhibits greater amplitude than monaural stimulation, as expected.

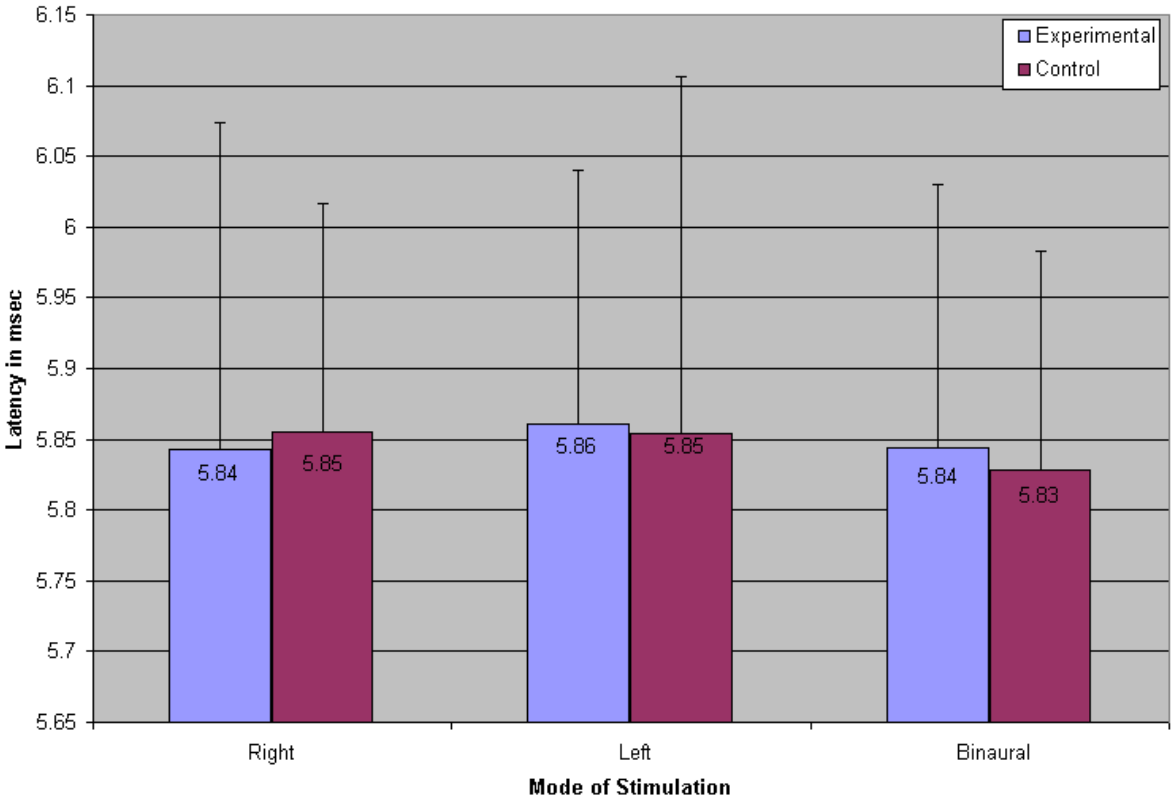

Figure 6. Group means and standard deviations for Wave V latency for right, left and binaural stimulation.

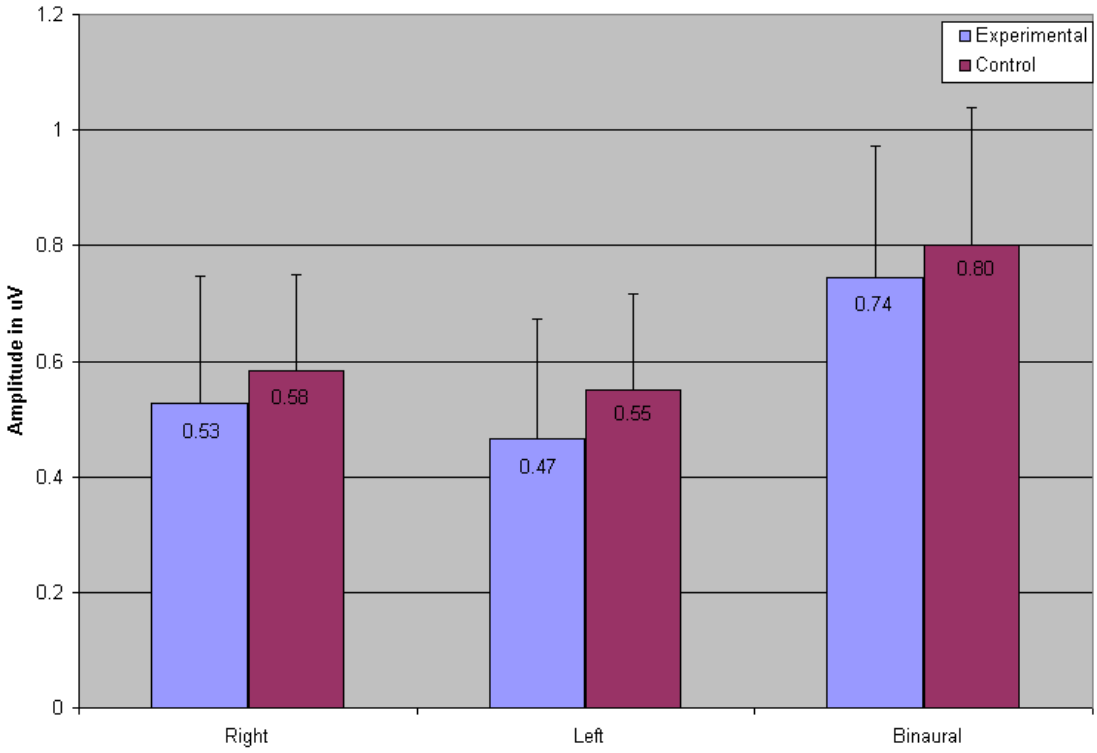

Figure 7. Group means and standard deviations for Wave V amplitude for right, left and binaural stimulation.

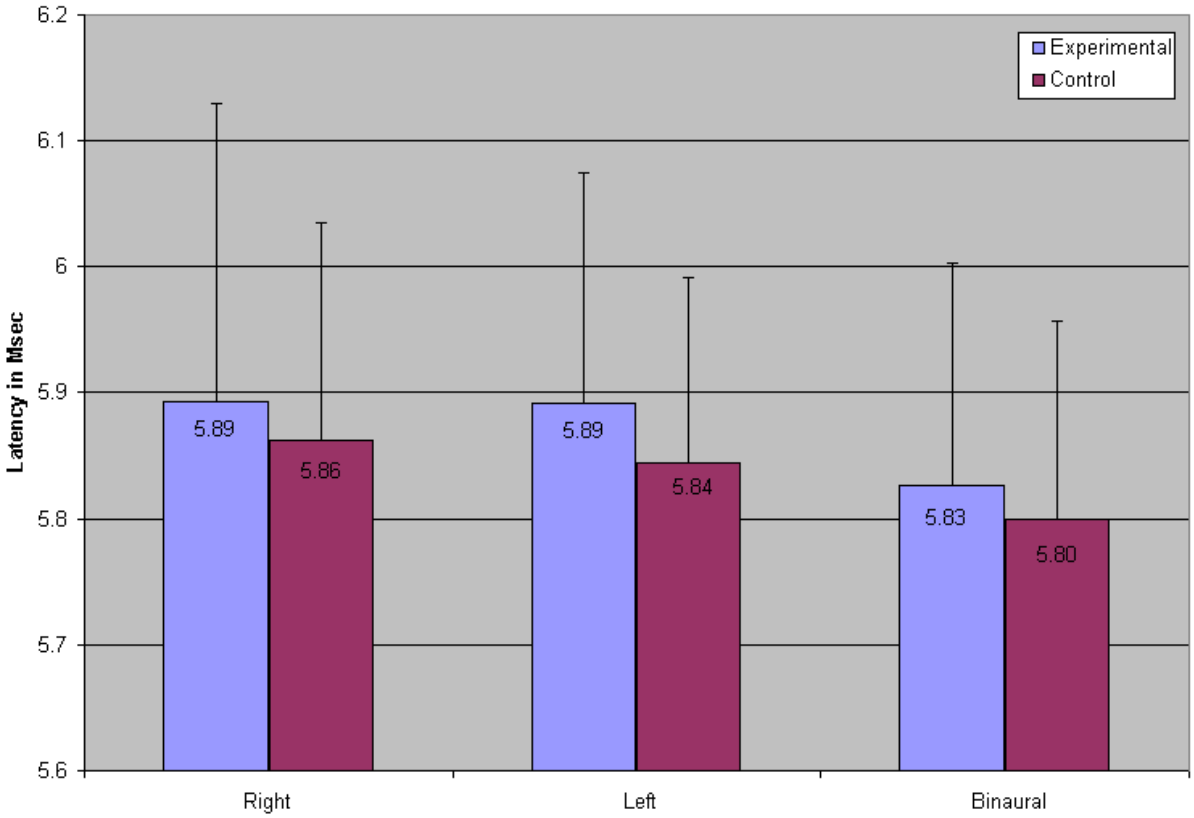

Figure 8. Group means and standard deviations for mid-line Wave V latency for right, left, and binaural recordings.

Figures 8 and 9 display Wave V mean latency and amplitude measures for midline (Cz-Oz) recordings for the right, left and binaural stimulation modes, respectively. Wave V latency from binaural recordings is slightly shorter than monaural recordings. However, the mode of stimulation was not a significant main effect on latency [F (2, 18) = 1.248, p = .311].

Greater amplitude is shown for the control group in the left and binaural stimulation modes, but not in the right monaural condition. Wave V amplitude is greater for binaural stimulation. The mode of stimulation was significant in amplitude measures [F 2, 17) = 52.119, p = .001].

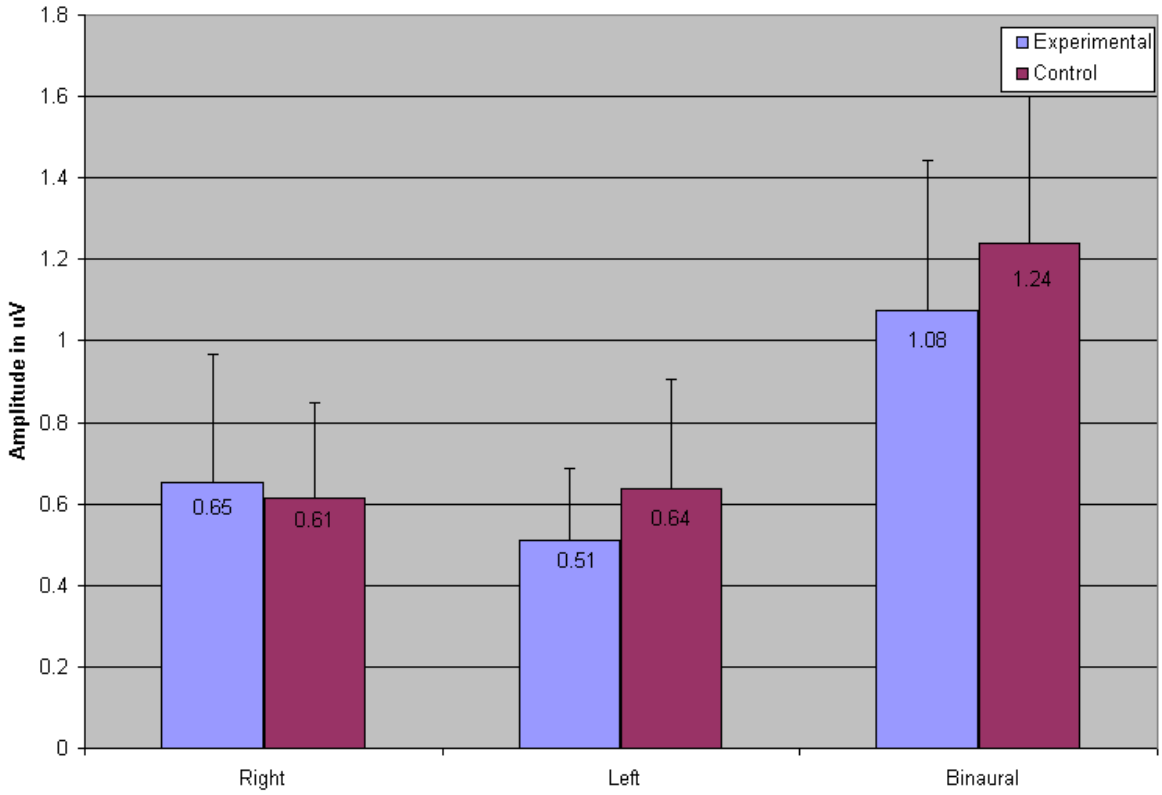

Figure 9. Group means and standard deviations for Wave V amplitude for the mid-line recording.

Binaural ITD

Midline latency and amplitude measurements of binaural wave V with an ITD of 0, 0.1, 0.4, 0.9, and 1.9 msec are displayed in Figure 10 and 11, respectively. In addition, an example of a midline recording of each binaural ITD for one subject is shown in Figure 12. A repeated measures analysis of variance indicates no significant differences in latency between the control and experimental groups [F (4,42) = .814, p=.523], although the experimental group has slightly longer wave V latencies.

There are no significant differences in the Wave V ITD amplitude measure between the groups [F (4, 42) = 2.209, p = .001]. Wave V latency increased and amplitude decreased with an increase in the ITD for both groups.

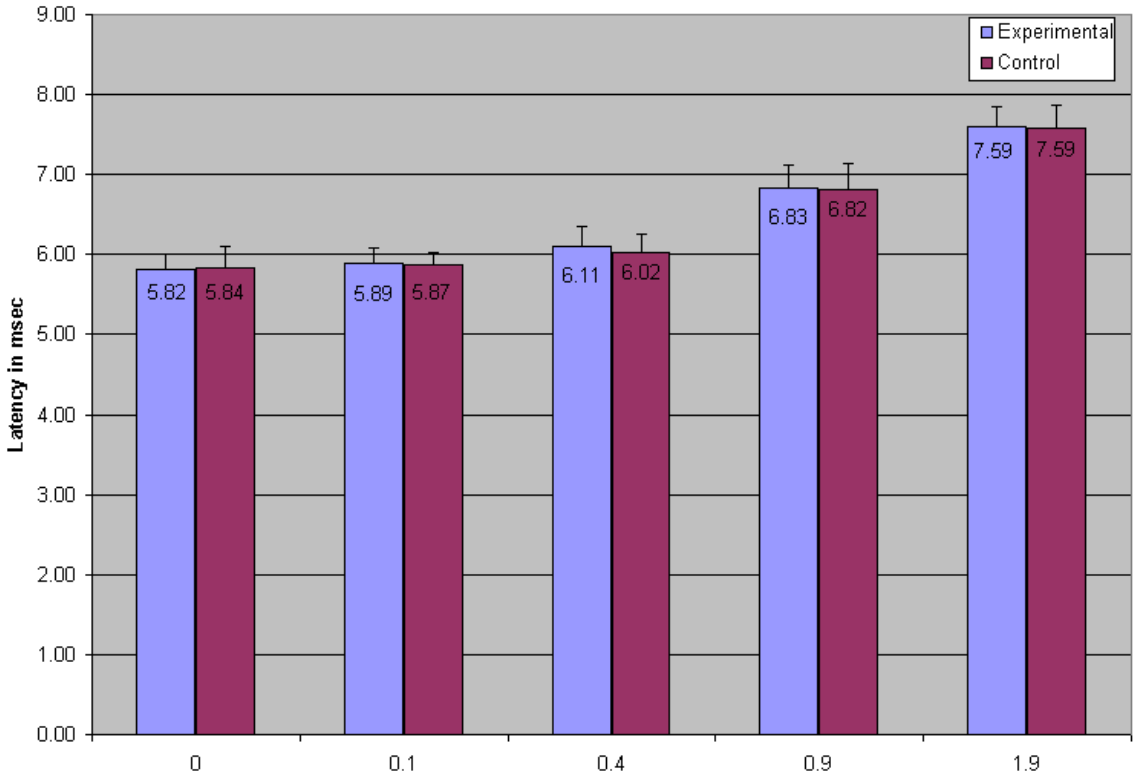

Figure 10. Group means and standard deviations Wave V mid-line recording at each ITD condition.

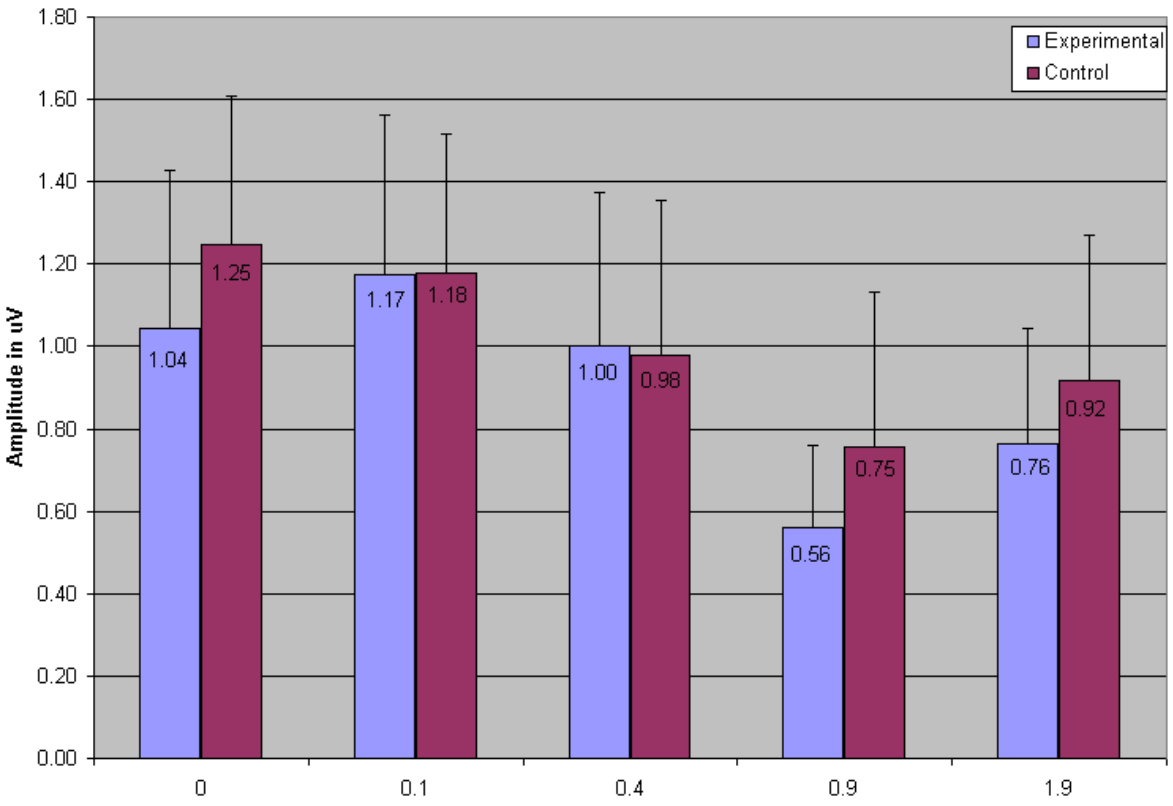

Figure 11. Group means and standard deviations for Wave V amplitude at each ITD condition.

Figure 12. An example of one subject's midline binaural ITD recording. A cursor is placed on wave V.

D. BINAURAL DIFFERENCE WAVEFORM

The binaural difference waveform was derived from the midline Cz-Oz tracings. The binaural interaction waveform was examined by the principal investigator and by a second independent judge. Previous investigations have shown that the BIC occurs within the region of waves IV to VI. Markers were placed on the binaural difference waveform at .5 msec before wave V peak latency and at 1.5 msec post wave V peak latency. This gave a 2 msec window

for reviewers to determine if a binaural interaction component was present. This qualitative analysis was done due to the inherent noise conditions of the binaural difference waveforms. These reviewers reported if there was any binaural interaction present. This is to say they reported if there was the presence of a peak, the presence of an inverted peak (trough), or the absence of any binaural interaction (flat line). An example of each is shown in Figures 13, 14, and 15, respectively. Out of 240 binaural difference waveforms, the judges disagreed on 3 waveforms. Thus, intra-judge reliability was 98.75%. A third independent judge reviewed the 3 waveforms in question.

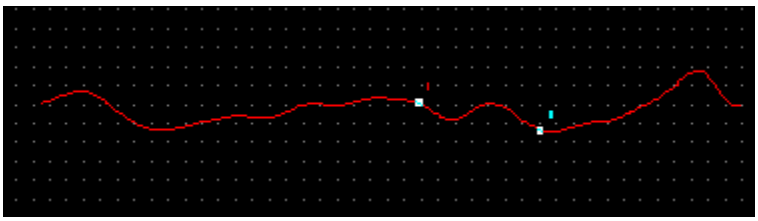

Figure 13. An example of a BIC peak.

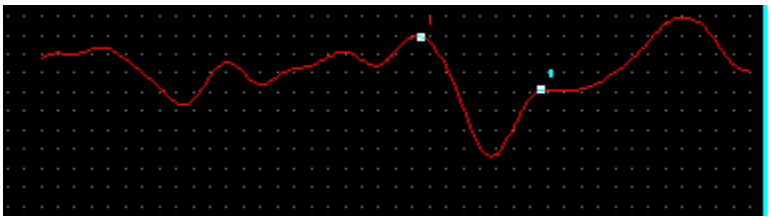

Figure 14. An example of a BIC trough.

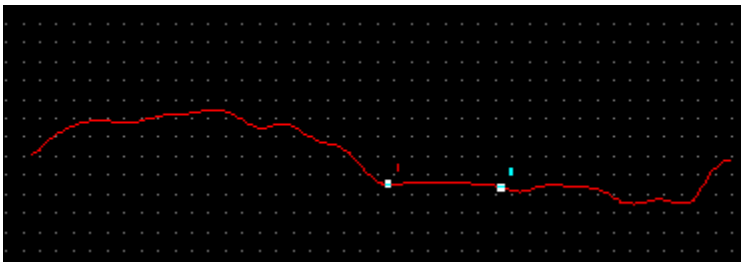

Figure 15. An example of an absent BIC.

A Chi Square analysis, shown in Table 4, indicates no significant differences in the presence of the Binaural Interaction Component between the two groups at any ITD.

Table 4. Results of the Chi-square analysis for the presence or absence of the binaural interaction component for each ITD.

	X^2	df	p
0 ITD	1.061	1	.247
.1 ITD	.105	1	.500
.4 ITD	.444	1	.370
.9 ITD	3.419	1	.068
1.9ITD	.462	1	.333

CHAPTER 5

DISCUSSION

The objective of the present study was to examine the early electrophysiological recordings (ABR and BIC) to determine if there was dys-synchrony in children with a specific temporal processing disorder. Since the lower brainstem auditory centers are responsible for early encoding of timing parameters, and these centers are the generators of the ABR, then early electrophysiologic recordings in children with APD may differ from normal children. Although it has been assumed that auditory evoked potentials are the electrophysiologic correlates of auditory processes (ASHA, 1996), the nature of this relationship has not been verified. The present investigation obtained important new information related to whether the ABR should be part of the APD test battery.

One of the difficulties in reviewing published investigations of APD is the frequent, inadequate definition of the study subjects. The present investigation provides descriptive data about the language abilities and specific temporal processing abilities of each subject. The temporal processing deficits of the subjects in this investigation are clearly defined by behavioral measurements.

A. DESCRIPTIVE TESTS

Some investigations contend that APD is only one type of language disorder. [See Reese (1981) and Cacace & McFarland (1998) for further elaboration.] It was not in the scope of the present investigation to further explore

a causality or relationship between auditory processing and language. Future research may one day allow investigators and clinicians to quantify the specific degree that auditory processing contributes to a child's academic difficulties.

A normal score on the PPVT-III was criteria for admission of all subjects into the present investigation (Standard Score =100 +/- 1 standard deviation of 15). The control group had a significantly greater group mean standard score on PPVT-III, and Listening Comprehension and Oral Expression Subtests of the OWLS. However, it must be noted that the each of the experimental subjects had scores on the PPVT-III that were within one standard deviation of a standard score of 100. Clinically, all of the subjects would be classified as within normal limits.

It is interesting to look at the specific areas where these children had difficulties. Fourteen of the experimental subjects had scores that were within normal limits on all of the language tests-- the PPVT-IIII, Listening Comprehension subtest and Oral Expression subtest of the OWLS. Five experimental subjects had abnormal scores on the Listening Comprehension subtest of the OWLS. Four experimental subjects had abnormal scores on the Oral Expression subtest of the OWLS. One experimental subject had abnormal scores on both the Listening Comprehension subtest and the Oral Expression subtest, but a normal score on the PPVT-III. There was only one subject in the control group had an abnormal score on the Listening Comprehension subtest of the OWLS. (Individual subject scores are listed in Appendix B.)

The widely used SCAN-C test is the only audiological test designed for APD screening of children ages five through eleven. This test determines if additional APD testing is necessary. The control group's mean standard score was greater on all sub-tests of the SCAN-C. However, there were significant group differences on the Filtered Words, Auditory Figure Ground, and Competing Words subtests of the SCAN-C. No significant difference was found on the Competing Sentence sub-test of the SCAN-C between the control and experimental groups. This could be due to the redundancy and contextual clues in the Competing Sentences sub-test. The SCAN-C test has been criticized for not having a specific sub-test for temporal processing. However, temporal processes are critical to a number of auditory perceptual tasks including the tasks assessed in the specific sub-tests of the Scan-C.

Careful inspection of individual subject data (reported in Appendix B) indicate that one experimental subject's SCAN-C subtest scores were essentially within normal limits--one standard deviation of the mean. Three control subjects had SCAN-C scores on one individual subtest that was below the mean. This investigation supports Keith's (1986) position that the SCAN-C is only a screening test and the results of this test should be viewed as complimentary to other behavioral and audiological findings.

B. BEHAVIORAL TESTS FOR TEMPORAL PROCESSING

APD is a wide spectrum disorder. Thus, the heterogeneity among listeners with APD limits the evaluation of behavioral test performance. Clinicians and investigators have sub-grouped or sub-typed APD. It is not expected that all

children with APD will perform similarly on all behavioral tests of APD. In this investigation only behavioral tests that specifically target temporal processing were administered. The results of the behavioral tests indicate that the control and experimental groups differ in their temporal processing ability.

Careful inspection of individual subject data (see Appendix B) strongly suggests that a diagnosis of APD should not be made based upon one single behavioral measure, but rather a test battery (ASHA, 1996; Bellis, 2003; Jerger & Musiek, 2000). The subject selection criterion for this study required that each experimental subject had failed at least three of the five behavioral tests. Two potential experimental subjects did not meet this criterion and were not included in this study. In addition, inspection of individual subject data in Appendix B indicates that eight control subjects had scores on one behavioral test that would be interpreted as abnormal.

The heterogeneity in APD complicates efforts to standardize an APD test battery. To date, there is no consensus or agreed-upon "gold-standard" APD test-battery. In this investigation, five behavioral tests which specifically target temporal processing were administered. Other tests which assess such auditory processes as dichotic listening or monaural low-redundancy tests were not administered. It is often recognized that children with APD often have deficits in more than one category. Therefore, children in this investigation with temporal processing deficits might demonstrate deficits in other auditory processing areas. Other APD tests which target additional auditory processes would be useful in recommending remedial programs or therapies.

C. ELECTROPHSYIOLOGICAL MEASURES

Electrophysiological measures were recommended by the 1996 ASHA Task Force on Central Auditory Processing Consensus Development and were recommended as part of the Minimal APD battery by the 2000 Consensus Conference on the Diagnosis of Auditory Processing in School-Aged Children. Electrophysiological tests in the APD evaluation may aid in the diagnosis or aid in validating the results of the behavioral test battery (Bellis, 2003; Chermak and Musiek, 1997). Unfortunately, there have been no published investigations which support or negate the inclusion of electrophysiological measures in the APD battery.

The results of the investigation reported here indicate no significant differences in the latency of ABR waves I, III, and V between the experimental and control group. There were also no significant differences in the wave V latency between the midline and Cz-mesial recording site. This is consistent with previous investigations which report no latency differences in the ABR recording from various recording sites (Hall, 1992; Hashimoto, Ishiyama, Yoshimoto & Nemoto, 1981). In addition, there were no significant differences in latency in the mode of stimulation, right, left or binaural. This is consistent with previous investigations of monaural versus binaural stimulation (Dobie & Norton, 1980; Hosford-Dunn, Mendelson & Salamy, 1981).

Wave I and wave III latencies were within normal limits for all control and experimental subjects (Musiek, Josey, & Glassock, 1986). Wave V latencies were within normal limits (Musiek et al., 1986) for all but two experimental

subjects. Careful inspection of individual ABR waveform latency data indicates that two experimental subjects had Wave V latencies that were 2 standard deviations greater than the experimental group mean latency value. Thus, this electrophysiological data adds objective evidence to support the diagnosis of an auditory processing disorder with a possible neurophysiological etiology in each of these two cases. It is also noted that both of these experimental subjects had a positive history of middle ear infections, as evidenced by having had pressure equalization tubes, and infant jaundice. Additional information of other abnormal or soft neurological signs was not mentioned in the case history. However, a neurological referral is appropriate based upon the abnormal auditory brainstem response. Other experimental subjects who had both infant jaundice and a history of middle ear infections had ABR recordings that were within normal limits.

Amplitude measures for waves I, III, and V were within normal limits (Musiek, et al., 1986) for both the control and experimental group. Amplitude measures were larger for the control group in the right, left, and binaural conditions than the experimental group.

Sohmer and Student (1978) reported abnormal ABR latency results in 16 subjects with minimal brain dysfunction. Subjects placed in this category had traits of hyperactivity, learning difficulty and coordination defects. Additionally, the Sohmer & Student investigation reported ABR latency abnormalities in other broad-spectrum disorders such as autism, and mental retardation.

The results of the current investigation are in agreement with Worthington (1981) who reported no differences in the ABR latencies between controls and

children with APD. This lack of difference is in contrast to the investigation by Worthington et al (1981) which reported abnormal ABR latencies in 8 out of 18 subjects with severe developmental and or/language delays. Conductive hearing loss accounted for an additional five abnormalities. The other three abnormalities related to right and left asymmetries which were greater than .3 msec in three subjects. Subject selection criterion for these studies was not reported.

Protti (1983) reported increased ABR latencies in 2 of 13 subjects with APD. Again, the type of APD, or how the diagnosis of APD was made, was not specified in this paper. However, Protti's work supports suggestions that electrophysiological measurements in the APD evaluation may provide objective evidence to support the diagnosis of APD.

Gopal and Pierel (1999) measured Wave V latency and amplitudes for 9 subjects who were diagnosed with a moderate to severe language impairment. Additionally, these subjects also had a composite score on the SCAN or SCAN-A that was greater than 1 standard deviation below the mean, indicating these subjects were "at-risk" for APD. The present investigation found no significant differences in the amplitude or latency measurements between the children "at-risk" for APD and a normal group. The BIC results of this study will be reported in a later section.

The present investigation is also consistent with Mason & Mellor (1984) who reported latency and amplitude measurements in eight children diagnosed with a language disorder and six children with motor speech disorders. No significant group differences in latency were reported. The amplitudes of the

ABR were was smaller in the language delay and motor speech group than the normal group. Only the amplitude of the wave V in the Cz-A1 recording was significantly different.

The ABR is recorded from surface electrodes, sometimes referred to as "far-field" recording. The amplitudes of the ABR recording will depend upon the conductivity of the tissue and the distance of the electrode from the generator site. It is worth noting once more that each group's mean amplitude measures were within normal limits. It is also important to note that ABR amplitude is more variable than peak latency (Lauter et al, 1993). Inherent noise conditions may also affect the amplitude of the ABR. In addition, other factors such as head size, the thickness of the skull, and electrode placement will affect the amplitude of the ABR.

D. BINAURAL WAVE V ITD

Waveform morphology, latency and amplitude may be affected when introducing a contralateral stimulus with a delay. This interaural delay may create a desynchronization of neuronal firings or result in a distortion of the normal ABR waveform. The results of this investigation are similar to the findings of Arslan et al. (1981) who reported morphological changes in the ABR recording when the ITD was greater than 2 msec. The results of this investigation found as the ITD increased, the Wave V latency increased and the amplitude decreased for both groups. There were no significant differences between the control and experimental group in wave V latency as a function of ITD.

A significant difference between groups in wave V amplitude for the .9 ITD condition was shown. A gradual decrease of .27 •V in amplitude was observed as the ITD increased from 0-.4 msec in the control group. This is similar to .17 •V decrease in amplitude as the ITD increased from .1 to .4 msec in the experimental group. An abrupt decrease in amplitude was observed as the ITD increased from .4 to .9 msec. Around 1 msec, the image is no longer fused, therefore, the amplitude reduction at .9 msec ITD is exhibited. The control group had a decrease of .23 •V in wave V amplitude, while the experimental group had a decrease of .44 •V in wave V amplitude.

E. BINAURAL INTERACTION COMPONENT

One of the difficulties in investigating the binaural interaction component is that the BIC is not a robust response and is difficult to measure. The amplitude of the BIC response is approximately .25-50 •V or about 10-20% of the amplitude of wave V (Hall, 1992). To add to the difficulty of investigating a very small potential, the BIC may not be present in all normal subjects (Dobie & Berlin, 1979; Dobie, 1982; DeChiccis, 1981).

An additional difficulty in BIC investigations is the instability of the response and potential background noise. Methodology differences are evident in published studies. Terminology such as "added, averaged, and summed" is sometimes confusing and has been used interchangeably in some past, published investigations. There are also differences in the number of monaural and binaural stimuli used in deriving the binaural difference waveform. A large number of stimuli have often been recorded to improve the signal to noise ratio. In one

report Wrege & Starr (1981) collected 12,000 stimuli and Furst, et al. (1985) recorded responses to 6400 sweeps. It is important to note that a fixed number of stimuli does not guarantee the same amount of residual noise (Elberling & Don, 1984; Elberlin &Wahlgreen, 1985). Therefore, the additional data test time and the noise make recording the BIC in a group of active, school-age children less than ideal.

A particular complication in BIC investigations is the identification of the prominent peaks. Early BIC peaks in the latency range for wave II and III were noticeable in this investigation. These early BIC waves have previously been reported in midline recordings (Wrege & Starr, 1981; Wilson et al., 1985). Activity from the distal portion of the VIII nerve and in part from the cochlear nucleus has been linked to Wave II. Binaural interaction is very difficult to explain from the VIII nerve and cochlear nucleus.

Conventional wisdom has it that the BIC occurs within a 4-6 msec window, in the latency of wave V. In this investigation, markers were placed .5 msec before wave V and 1.5 msec after wave V so that a 2 msec window was evident for the two reviewers to determine if a BIC was present in the Wave V region.

Previous investigations have reported the absence of the BIC when the ITD was greater than 1 msec. However, in one particular investigation by McPherson and Starr (1993) a figure with a BIC present at 1.6 ITD is displayed. Therefore, the BIC may be present at ITDs greater than 1 msec. Other investigators have also employed a behavioral localization task with the

electrophsyiological ITD recording. These investigators report an absent BIC when the signal is no longer fused (Furst et al, 1985).

Although there have been investigations of the BIC with infants, there are limited data describing the BIC in children. Gunnarson & Finitzo (1991) reported absent BICs in children with histories of otitis media.

Gopal and Pierel (1999) reported longer latencies and significantly smaller amplitudes of the BIC in children "at-risk" for APD. One difference in the present investigation and the Gopal & Pierel study was that significant differences in ABR amplitude were reported in the present investigation, but not in Gopal & Pierel's report, although wave V amplitudes were greater for the control group. Another limitation of the Gopal & Pierel report is they reported data for only nine subjects who were "at-risk" for APD. These nine subjects had been diagnosed with a language disorder, but no specific tests for APD were administered.

Delb et al. (2003) reported a significant difference in the occurrence of the BIC in children aged 6 –12 years with APD. However, they did not report their criterion to determine the presence or absence of a BIC. Delb et al reports a 76% sensitivity and specificity in using the presence of the BIC as an indicator of APD. However, these investigators did not report any difficulty or contra-indications of recording BICs in school-age children.

The present investigation reported no difference in the occurrence of a binaural interaction component between the control and experimental group. Two independent examiners judged the binaural difference waveform to determine if there was any indication of binaural interaction present. Excellent intra-judge

reliability was reported. This is in disagreement with the investigation by DeChiccis (1981) who reported poor intra-judge reliability between two examiners. The excellent intra-judge reliability in this investigation may be related to the BIC experience of these judges. Delb et al (2003) reported only the presence of the • wave--the first prominent peak of the binaural interaction component.

F. FUTURE INVESTIGATIONS

This investigation did not attempt to address the relationship between temporal processing ability and language ability. However, it is noted that although all of the children in this investigation had normal receptive vocabulary, some children demonstrated difficulty in oral expression or listening comprehension, while others did not. Additional information about the subject's reading abilities might be of interest.

Temporal processing ability in children with language and reading disorders have been an area of interest for many years. Efficacy of auditory training has been investigated using both behavioral and electrophysiological measures (Yencer, 1998). Documented changes in the neuronal firing patterns provide objective evidence for plasticity in the central auditory nervous system.

The 1996 ASHA Task Force listed four areas of research priority in APD; Basic Science, Assessment, Management, and Professional Practice. Eight specific issues were further described, including the development of a minimal test battery. An updated report from the ASHA Task Force is not yet available.

One important measure of temporal processing used in this investigation

was gap detection, as measured by the Random Gap Detection Test. The task-design may, in part, limit this test. The subject is to listen to a tone and indicate if there was one sound or two sounds. The gap detection threshold is the lowest interval in msec where 2 sounds are heard. This works well when subjects understand the task. However, there are no directions in interpreting test results where a subject hears 2 sounds in the item with a 10 msec gap, but only reports hearing 1 sound in the test items with a 15 and 20 msec gap.

A noted clinical observation in this investigation was that many of the subjects began guessing or would fixate on either a "1"or "2". Several subjects reported they heard no difference in any of the tones during three practice sessions, therefore, it is assumed that their gap detection thresholds are greater than 40 msec. An alternative paradigm would be a gap detection using a discrimination model. Subjects would listen to three tones, then indicate which one is different, an "odd-man out" paradigm. If such a test were developed in an adaptive procedure, three-interval forced choice, the gap detection threshold could be obtained in less time.

Previous investigations have linked otitis media and jaundice as risk-factors for APD. It was not a specific aim in the present research to investigate a relationship between APD and pre-disposing factors. However, anecdotal information of some "risk-factors" was reported in the case histories. Seven experimental subjects reported jaundice; ten experimental subjects reported a protracted history of otitis media by having pressure equalization tubes previously placed. A regression analysis indicated no significant differences in the latency

and amplitude of the ABR recording for these subjects ($p > .05$). Further exploration of risk factors including genetic factors could offer valuable insight to the etiology and remediation of APD.

Previous reports in the literature (Hannely, Jerger & Rivera, 1983; Jerger, Hannley & Rivera, 1983) have reported abnormal wave III recordings in patients with abnormal MLDs. In the present investigation, nine experimental subjects had abnormal MLDs. A regression analysis indicated a significant difference in the latency ($p = .032$) as well as the amplitude ($p = .033$) in the latency of the ABR recording in subjects with an abnormal MLD. Further investigations of children with an abnormal MLD and APD may be useful in understanding possible etiologies of APD.

Late auditory evoked potentials are believed to represent the sensory processing that takes place between peripheral encoding of the acoustic stimulus and conscious perception. Late auditory evoked potentials demonstrate different maturational patterns. Late auditory potentials have previously been used to investigate special populations such as language impairment (Tonquist-Uhlen 1996; Neville et al, 1993) learning disorders (Cunningham, Nicol, Zecker, Bradlow & Kraus, 2001) and ADHD (Cunninham et al, 2001).

Of particular interest to future investigations is the report of Jirsa & Clontz (1990). They reported an increase in latency and a decrease in amplitude of the P300 response in a group of children with APD when compared to normal individuals. This decrease was not evident in the N1 or P2 response. However, the description or characteristics of APD were not discussed.

CHAPTER 6

SUMMARY

This investigation reported behavioral and early electrophysiological measures in a group of children with specific temporal processing difficulties and an age-matched control group. In an effort to better describe the subjects, two language tests and the SCAN-C were administered. Significant differences were found in the group mean standard score on the language tests. However, the mean standard score for both the experimental and control group were within normal limits. There were also group mean differences on three sub-tests of the SCAN-C. In addition, the groups differed in their performance on behavioral tests of temporal processing. No significant differences in ABR waveform latency were found between the control and experimental group. There were significant amplitude differences, albeit small. The presence of the BIC was found in both control and experimental subjects.

The results of this investigation are clinically significant. Based on the results of the present well-controlled investigation of children with temporal processing disorders, there is no indication the auditory brainstem response to click stimuli is efficient in providing additional diagnosis of APD.

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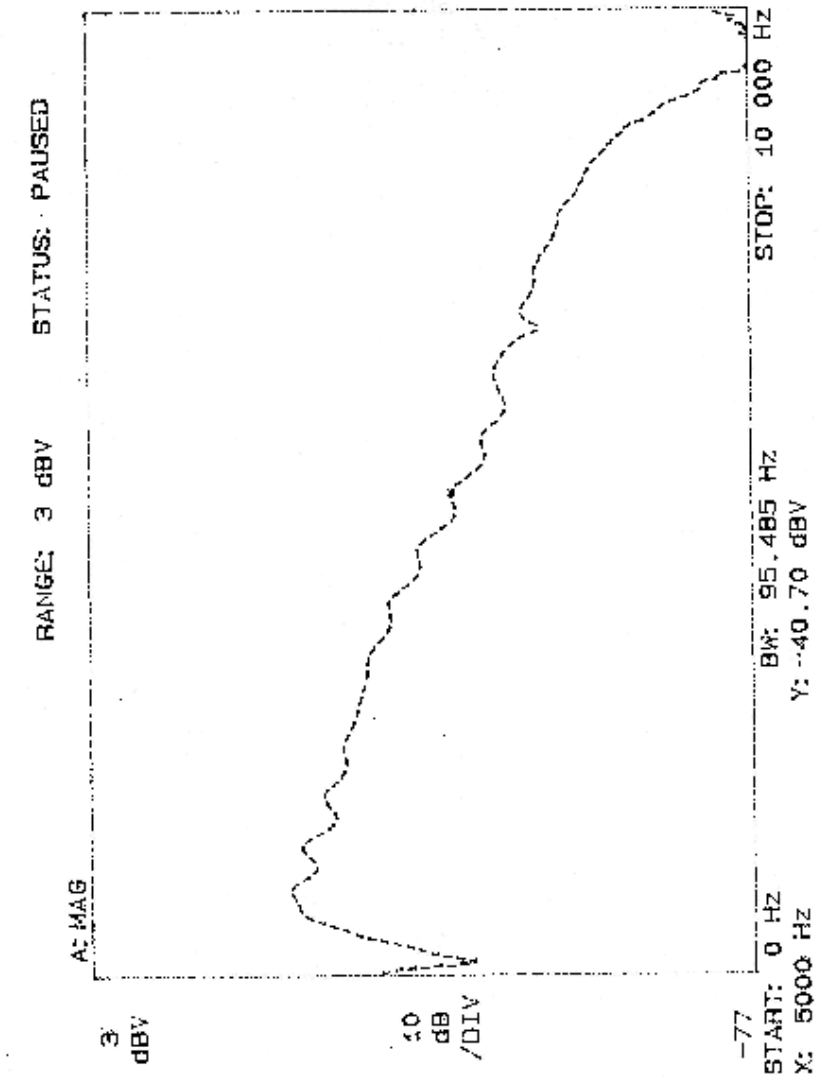
APPENDIX A
ACOUSTICAL SPECTRUM FOR ABR CLICK



APPENDIX B
SUBJECT DATA

Demographic Data

Subject	Age	group	Edu. Mom	Edu. Dad	School	Subject	Age	group	Edu. Mom	Edu. Dad	School
E1	7.70	1.00	2	4	1	C1	9.50	2.00	1	2	1
E2	9.40	1.00	3	3	1	C2	7.90	2.00	2	1	1
E3	7.10	1.00	3		1	C3	11.00	2.00	1	1	1
E4	8.60	1.00	1	1	1	C4	10.30	2.00	1	2	1
E5	8.70	1.00	2	3	4	C5	11.10	2.00	1	1	4
E6	8.80	1.00	3	3	1	C6	10.60	2.00	3	4	1
E7	8.20	1.00	1	1	1	C7	9.90	2.00	2	3	3
E8	8.60	1.00	2	3	2	C8	8.10	2.00	3	3	2
E9	8.80	1.00	1		3	C9	10.70	2.00	2	3	4
E10	10.30	1.00	2	2	1	C10	8.50	2.00	3	3	3
E11	7.60	1.00	1	1	1	C11	7.80	2.00	3	3	1
E12	7.80	1.00	1	1	1	C12	9.30	2.00	4	3	4
E13	7.50	1.00	1	1	1	C13	7.60	2.00	2	2	1
E14	7.70	1.00	2	3	1	C14	9.80	2.00	1	2	1
E15	7.20	1.00	3	3	1	C15	7.40	2.00	1	2	1
E16	7.20	1.00	1	1	1	C16	7.11	2.00	3	3	1
E17	7.80	1.00	3	3	1	C17	7.90	2.00	3	4	2
E18	9.80	1.00	1	2	3	C18	7.00	2.00	2	3	1
E19	8.50	1.00	2	2	4	C19	8.20	2.00	1	1	1
E20	11.70	1.00	2	1	4	C20	8.10	2.00	3	2	1
E21	11.40	1.00	3	4	3	C21	7.40	2.00	3	4	2
E22	11.50	1.00	1	0	1	C22	9.40	2.00	2	1	1
E23	9.10	1.00	3	3	1	C23	8.50	2.00	2	1	1
E24	7.30	1.00	1	1	3	C24	8.70	2.00	2	3	1

Descriptive Tests

SUBJECT	Age	SS PPVT	SS OWLS List	SS OWLS Oral	SCAN. FW	SCAN AF(	SCAN CW	SCAN CS
E1	7.70	124	118	98	14	8	12	12
E2	9.40	100	107	105	12	9	11	6
E3	7.10	119	114	100	12	10	9	11
E4	8.60	90	87	83	12	3	6	8
E5	8.70	107	107	89	6	6	12	10
E6	8.80	113	103	97	11	6	8	10
E7	8.20	112	95	92	13	6	10	8
E8	8.60	90	110	84	11	8	9	10
E9	8.80	100	100	100	7	7	6	8
E10	10.30	88	80	86	10	8	8	13
E11	7.60	103	85	87	7	3	5	16
E12	7.80	104	78	94	12	11	6	5
E13	7.50	91	92	73	10	6	5	5
E14	7.70	105	84	82	8	8	4	11
E15	7.20	90	80	90	1	1	6	4
E16	7.20	102	100	93	10	13	9	9
E17	7.80	105	108	98	5	8	10	16
E18	9.80	112	105	96	13	4	10	11
E19	8.50	101	108	86	9	8	9	9
E20	11.70	93	103	85	8	6	6	6
E21	11.40	120	81	97	12	9	6	9
E22	11.50	102	95	88	5	4	10	8
E23	9.10	96	85	78	5	3	4	4
E24	7.30	89	80	73	8	4	6	8

SUBJECT	Age	SS PPVT	SS OWLS List	SS OWLS Oral	SCAN. FW	SCAN AF(	SCAN CW	SCAN CS
C1	9.50	113	83	116	9	11	10	9
C2	7.90	118	109	102	11	9	10	13
C3	11.00	104	103	104	11	11	7	11
C4	10.30	98	103	104	12	11	11	9
C5	11.10	127	116	98	7	9	8	9
C6	10.60	105	99	116	11	10	12	10
C7	9.90	108	119	94	13	9	13	10
C8	8.10	144	138	150	11	9	12	16
C9	10.70	115	95	92	13	5	13	9
C10	8.50	100	95	95	13	7	9	8
C11	7.80	103	103	101	10	10	12	9
C12	9.30	119	120	106	13	14	14	9
C13	7.60	105	99	100	10	11	13	9
C15	9.80	104	97	104	12	9	10	11
C15	7.40	100	85	98	9	10	10	10
C16	7.11	125	122	118	13	13	14	14
C17	7.90	123	104	112	9	9	13	11
C18	7.00	110	104	95	13	8	8	8
C19	8.20	127	99	103	11	8	12	11
C20	8.10	121	104	101	12	12	10	10
C21	7.40	128	119	120	13	11	12	12
C22	9.40	139	94	120	14	12	13	10
C23	8.50	113	101	105	12	10	9	8
C24	8.70	118	87	88	13	11	10	8

Behavioral Tests

SUBJECT	T. Com. R	T. Comp L	P.P. Right	P.P. Left	D. P. R	D.P. L	Gap Det	MLD
E1	56	64	0	77	0	0		5
E2	68	76	30	47	0	10		5
E3	92	96	23	20	17	23		8
E4	84	68	0	27	0	0		11
E5	36	76	0	0	0	0		7
E6	72	56	30	7	20	20		11
E7	92	92	7	33	13	27		13
E8	100	100	30	30	0	0		14
E9	64	68	0	0	53	77		4
E10	72	68	40	43	10	10		10
E11	36	52	0	0	0	0		4
E12	72	64	0	0	0	0	27.5	8
E13	48	48	0	0	0	0	13.75	6
E14	44	60	10	27	13	7		7
E15	16	0	0	0	0	0		5
E16	56	46	63	57	7	27	4.75	9
E17	52	56	30	73	3	3		10
E18	68	68	33	23	27	23	5.5	7
E19	72	44	33	21	33	51		10
E20	60	76	27	0	67	67		10
E21	72	76	56	61	56	67		10
E22	76	72	20	13	13	20	6.75	10
E23	56	64	43	60	7	67	40	6
E24	32	40	0	0	0	0		10

SUBJECT	T. Com. R	T. Comp L	P.P. Right	P.P. Left	D. P. R	D.P. L	Gap Det	MLD
C1	88	80	100	100	76	67	8	11
C2	96	96	40	33	27	18	15	11
C3	100	92	93	97	93	90	8.75	12
C4	92	100	100	100	57	57	11.25	11
C5	88	88	77	60	97	100	10	11
C6	100	100	70	70	97	93	5	14
C7	92	96	97	80	30	40	8.75	12
C8	84	96	100	100	97	97	6.25	10
C9	82	80	60	37	13	33	7.5	11
C10	84	88	100	100	100	100	5	10
C11	96	92	100	100	100	100	10	10
C12	84	92	74	90	70	83	5.5	11
C13	92	92	53	53	70	83	6.25	13
C15	88	88	20	13	97	77	8.75	10
C15	96	100	40	67	63	60	6.75	10
C16	92	100	83	53	100	77	4.75	11
C17	84	92	90	93	73	70	5.25	14
C18	88	92	40	40	57	53	11.25	11
C19	88	88	90	92	100	100	15	14
C20	88	92	93	100	83	40	12.5	10
C21	100	92	90	83	30	20	8.75	12
C22	96	100	97	100	100	100	2.75	11
C23	100	96	10	37	60	53	7.5	10
C24	92	92	57	33	47	43	5	11

T. Com.R. = Time Compressed Speech Right Ear
T. Comp. L. = Time Compressed Speech Left Ear
P.P. Right = Pitch Pattern Right Ear
P.P. Left = Pitch Pattern Left Ear
D.P.R. = Duration Pattern Right Ear
D.P. L. = Duration Pattern Left Ear
Gap. Det.= Random Gap Detection
MLD = Masking Level Difference

Interpretation for Behavioral Tests
Within normal limits= 0; Abnormal =1

Experimental	T. Com. R	T. Comp L	P.P. Righ	P.P. Left	D. P. R	D.P. L	Gap Det	MLD
E1	1	1	1	0	1	1	1	1
E2	1	1	1	1	1	1	1	1
E3	1	0	1	1	1	1	1	0
E4	0	1	1	1	1	1	1	0
E5	1	1	1	1	1	1	1	1
E6	1	1	1	1	1	1	1	0
E7	0	0	1	1	1	1	1	0
E8	0	0	1	1	1	1	1	0
E9	1	1	1	1	0	0	1	1
E10	1	1	1	1	1	1	1	0
E11	1	1	1	1	1	1	1	1
E12	1	1	1	1	1	1	0	0
E13	1	1	1	1	1	1	0	1
E14	1	1	1	1	1	1	1	1
E15	1	1	1	1	1	1	1	1
E16	1	1	0	0	1	1	0	0
E17	1	1	1	0	1	1	1	0
E18	1	1	1	1	1	1	0	1
E19	1	1	1	1	1	0	1	0
E20	1	1	1	1	0	0	1	0
E21	1	1	1	1	1	1	1	0
E22	1	1	1	1	1	1	0	0
E23	1	1	1	1	1	0	1	1
E24	1	1	1	1	1	1	1	0

Control	T. Com. R	T. Comp l	P.P. Righ	P.P. Left	D. P. R	D.P. L	Gap Det	MLD
C1	0	0	0	0	0	0	0	0
C2	0	0	0	0	1	1	0	0
C3	0	0	0	0	0	0	0	0
C4	0	0	0	0	1	1	0	0
C5	0	0	0	0	0	0	0	0
C6	0	0	0	0	0	0	0	0
C7	0	0	0	0	1	1	0	0
C8	0	0	0	0	0	0	0	0
C9	0	0	0	1	0	1	0	0
C10	0	0	0	0	0	0	0	0
C11	0	0	0	0	0	0	0	0
C12	0	0	0	0	0	0	0	0
C13	0	0	0	0	0	0	0	0
C14	0	0	0	0	0	0	0	0
C15	0	0	0	0	0	0	0	0
C16	0	0	0	0	0	0	0	0
C17	0	0	0	0	0	0	0	0
C18	0	0	0	0	0	0	0	0
C19	0	0	0	0	1	1	0	0
C20	0	0	0	0	0	0	0	0
C21	0	0	0	0	0	1	0	0
C22	0	0	0	0	0	0	0	0
C23	0	0	1	1	0	0	0	0
C24	0	0	0	1	0	0	0	0

Electrophsyiological Latency Data

Subject	Cz I R	CZ III R	Cz V R	Cz I L	Cz III L	Cz V L	Cz I- Bin	Cz III Bin	Cz V Bin	Oz V R	Ov V Left	Oz V Bin
E1	1.6	3.7	5.6	1.7	3.6	5.6	1.6	3.7	5.6	5.95	5.82	5.82
E2	1.79	3.73	5.62	1.75	3.69	5.62		3.73	5.58	5.62	5.62	5.58
E3	1.55	3.52	5.49	1.59	3.56	5.74	1.47	3.64	5.62	5.54	5.7	5.62
E4	1.59	3.77	5.74	1.63	3.69	5.7	1.63	3.69	5.7	5.78	5.74	5.66
E5	1.71	3.73	5.99	1.75	3.6	5.95	1.67	3.64	5.9	5.99	5.99	5.82
E6	1.67	3.73	5.74	1.63		5.9	1.71	3.69	5.58	5.66	5.9	5.58
E7	1.47	3.85	5.74	1.71	3.81	5.9	1.67	3.85	5.86	5.74	5.99	5.82
E8	1.88	3.85	5.78	1.71	3.85	5.74	1.75	3.85	5.74	5.82	5.74	5.74
E9	1.59	3.93	5.86	1.67	3.85	5.9	1.63	3.85	5.82	5.9	5.86	5.86
E10	1.47	3.97	6.07		3.56	5.99	1.63	3.89	5.9	5.99	5.99	5.86
E11		4.18	6.11		4.06	5.82		4.01	5.95	6.23	5.9	5.99
E12	1.71	3.85	6.07		4.01	5.99	1.71	4.06	5.86	6.07	6.03	5.9
E13	1.51	3.77	6.03	1.55	3.93	5.86	1.67	3.77	6.03	6.11	5.95	6.03
E14	1.67	3.97	5.95	1.75	3.89	5.9	1.84	3.85	5.9	6.07	5.86	5.9
E15	1.75	3.73	5.62	1.71	4.01	5.86	1.63	3.69	5.74	5.74	5.66	5.7
E16	1.88		6.27		4.1	6.27	1.84		6.23	6.4	6.32	6.11
E17	1.63	3.81	5.7	1.79	3.77	5.66	1.61	3.81	5.7	5.7	5.82	5.66
E18	1.63	3.81	5.66	1.63	3.81	5.74	1.88	3.85	5.74	5.66	5.74	5.74
E19	1.67	3.93	5.66	1.63	3.69	5.78	1.63	3.77	5.74	5.7	5.78	5.74
E20	1.92	4.01	5.82	1.88	4.06	5.99		4.006	5.99	5.86	6.07	5.95
E21	1.71	3.6	5.66	1.63	3.6	5.54	1.63	3.6	5.62	5.7	5.62	5.58
E22	1.71	3.85	5.78			5.95		4.01	5.99	5.86	6.03	5.95
E23	1.59	4.16	6.4	1.71	3.85	6.27	1.67	3.93	6.27	6.44	6.27	6.27
E24	1.67	3.64	5.62	1.63	3.93	5.74	1.67	3.64	5.95	5.9	5.99	5.95

Subject	Cz I R	CZ III R	Cz V R	Cz I L	Cz III L	Cz V L	Cz I- Bin	Cz III Bin	Cz V Bin	Oz V R	Ov V Left	Oz V Bin
C1	1.63	3.93	5.78	1.75	3.73	5.7	1.79	3.89	5.78	5.86	5.78	5.74
C2	1.84	3.77	5.95	1.84	3.89	6.07	1.67	3.89	6.07	5.95	6.07	6.07
C3	1.71	4.1	5.95	1.75		5.9		4.14	5.9	5.95	5.9	5.86
C4		3.85	5.66		3.93	5.74	1.67	3.89	5.66	5.49	5.78	5.66
C5	1.75	3.89	5.78	1.67	3.6	6.78	1.63	3.6	5.74	5.78	5.86	5.7
C6	1.79	3.81	5.86	1.75	3.64	5.85	1.75	3.93	5.86	5.9	5.95	5.82
C7	1.63	3.77	5.66	1.59	3.81	5.74	1.55	3.73	5.66	5.62	5.66	5.66
C8			6.27	1.75	3.63	5.82	1.63	3.73	5.7	6.19	5.86	5.74
C9	1.79	3.81	5.82		3.81	5.82	1.79	3.89	5.9	5.86	5.86	5.9
C10	1.75	3.77	5.86	1.63	3.85	5.82	1.67	3.77	5.78	5.82	5.82	5.78
C11	1.76	3.89	5.91		3.77	5.5	1.63	3.81	5.7	5.91	5.54	5.7
C12	1.4	3.48	5.96	1.61	3.83	5.5	1.6	3.81	5.7	5.9	5.5	537
C13	1.71	3.69	5.95	1.71		5.9	1.71	3.73	5.9	5.9	5.9	5.82
C14	1.67	3.97	5.95	1.59	3.93	5.82	1.71	3.93	5.95	5.99	5.86	5.9
C15	1.67	3.97	6.03	1.63	3.97	5.78	1.71	3.89	5.95	5.99	5.86	5.99
C16	1.67	3.89	5.82	1.63	3.93	5.78	1.67	3.89	5.82	5.86	5.9	5.78
C17	1.79	4.01	5.86	1.75	4.22	5.82	1.79	4.14	5.86	5.74	5.86	5.66
C18		3.97	5.86		4.01	5.95	1.6	4.1	6.07	5.95	6.03	6.03
C19	1.75	3.85	5.7	1.67	3.93	5.7	1.71	3.85	5.74	5.74	5.74	5.7
C20	1.75	4.01	5.9	1.71	4.1	6.07	1.75	4.01	5.99	6.03	6.07	5.95
C21	1.84	4.06	6.11	1.75	4.06	6.11	1.79	4.06	6.11	6.19	6.07	6.11
C22	1.59	3.85	5.54	1.59	3.77	5.62	1.55	3.73	5.58	5.62	5.62	5.58
C23	1.55	3.73	5.82	1.47	3.64	5.82	1.51	3.6	5.78	5.86	5.86	5.74
C24		3.64	5.62	1.92	3.69	5.54	1.84	3.69	5.54	5.62	5.58	5.49

Electrophsyiological Amplitude Data

Subject	Cz I R	CZ III R	Cz V R	Cz I L	Cz III L	Cz V L	Cz I- Bin	Cz III Bin	Cz V Bin	Oz V R	Ov V Left	Oz V Bin
E1	0.253	0.206	0.527	0.361	0.215	0.466	0.453	0.306	0.666	0.434	0.558	0.79
E2	0.161	0.073	0.483	0.18	0.29	0.502		0.3	0.7	1	0.535	1.25
E3	0.204	0.154	0.755	0.471	0.384	0.364	0.683	0.432	0.396	0.755	0.364	1.15
E4	0.204	0.294	0.502	0.388	0.164	0.376	0.29	0.41	0.76	0.75	0.376	0.81
E5	0.291	0.244	0.572	0.275	0.158	0.192	0.451	0.415	0.662	0.382	0.59	1.38
E6	0.445	0.139	0.751	,308		0.19	0.52	0.492	0.79	0.574	0.317	1.62
E7	0.057	0.266	0.508	0.358	0.064	0.399	0.34	0.2	0.7	0.41	0.275	1.02
E8	0.201	0.228	0.491	0.551	0.292	0.558	0.46	0.26	1.05	0.517	0.757	1.41
E9	0.454	0.281	0.811	0.404	0.267	0.913	0.65	0.39	1.04	1.47	0.63	1.99
E10	0.209	0.146	0.163		0.154	0.154	0.166	0.21	0.511	0.304	0.448	0.94
E11		0.197	0.509		0.236	0.473		0.29	0.98	0.687	0.473	1.04
E12	0.097	0.109	0.261		0.112	0.158	0.548	0.164	0.46	0.516	0.15	0.574
E13	0.151	0.09	0.486	0.202	0.179	0.68	0.4	0.13	1.03	0.54	0.68	0.96
E14	0.048	0.093	0.265	0.196	0.141	0.346	0.189	0.248	0.389	0.82	0.504	0.53
E15	0.644	0.577	0.687		0.348	0.864	0.665	0.848	0.865	1.34	0.864	1.08
E16	0.098		0.587	0.395	0.271	0.54	0.272		0.757	0.54	0.663	0.508
E17	0.326	0.381	0.781			0.521	0.569	0.485	0.664	0.65	0.37	1.21
E18	0.383	0.198	0.531	0.227	0.1	0.621	0.41	0.22	0.94	0.51	0.66	1.37
E19	0.347	0.186	0.434	0.697	0.402	0.367	0.65	0.21	0.81	0.243	0.285	0.52
E20	`	0.1	1.02	0.2	0.17	0.74		0.14	1.16	1.02	0.74	1.51
E21	0.329	0.128	0.473	0.348	0.26	0.522	0.41	0.25	0.73	0.416	0.54	1.16
E22			0.053			0.37		0.15	0.7		0.456	1.11
E23E	0.101	0.248	0.634	0.27	0.24	0.339	0.323	0.242	0.695	0.765	0.662	1.09
E24	0.308	0.196	0.374	0.62	0.074	0.53	0.618	0.249	0.32	0.32	0.369	0.81

Subject	Cz I R	CZ III R	Cz V R	Cz I L	Cz III L	Cz V L	Cz I- Bin	Cz III Bin	Cz V Bin	Oz V R	Ov V Left	Oz V Bin
C1	0.38	0.75	0.89	0.361	0.856	0.743	0.48	1.08	1.11	1.09	0.932	1.75
C2	0.188	0.52	0.565		0.801	0.516	0.308	0.524	0.544	0.516	0.516	0.56
C3	0.048	0.057	0.488	0.105		0.474		0.087	0.593	0.129	0.665	1.08
C4		0.582	0.633	0.419	0.535	0.712	0.42	0.36	0.87	0.712	0.56	1.43
C5	0.362	0.179	0.374	0.142	0.203	0.268	0.289	0.457	0.636	0.479	0.296	1.21
C6	0.12	0.056	0.577	0.342	0.073	0.486	0.21	0.17	0.78	0.746	0.486	1.43
C7	0.62	0.333	0.582	0.729	0.499	0.55	0.635	0.54	0.727	0.475	0.411	0.91
C8			0.488	0.481	0.322	0.388	0.532	0.507	0.636	0.311	0.366	0.94
C9	0.39	0.35	0.52		0.21	0.63	0.42	0.53	0.83	0.703	0.63	1.129
C10	0.329	0.441	0.833	0.471	0.47	0.761	0.515	0.713	1.11	0.833	0.398	1.58
C11	0.321	0.326	0.342		0.217	0.543	0.437	0.331	0.753	0.475	0.683	0.859
C12	0.35	0.32	0.583	0.46	0.319	0.55	0.52	0.43	0.81	0.6	0.59	1.2
C13	0.223	0.08	0.485	0.137		0.458	0.341	0.109	0.871	0.457	0.68	1.79
C14	0.678	0.473	0.593	0.701	0.416	0.706	0.638	0.57	0.875	0.741	0.707	1.38
C15	0.205	0.245	0.457	0.451	0.338	0.398		0.389	0.615	0.457	0.51	0.69
C16	0.351	0.311	0.272	0.642	0.453	0.405	0.63	0.406	0.511	0.279	0.483	0.79
C17	0.341	0.301	0.536	0.464	0.257	0.5992	0.536	0.266	0.429	0.731	0.606	1.37
C18		0.08	0.56		0.25	0.48		0.23	0.68	0.531	0.66	1.11
C19	0.557	0.46	0.538	0.705	0.402	0.558	0.814	0.54	0.773	0.759	0.657	1.1
C20	0.307	0.352	0.608	0.675	0.436	0.159	0.552	0.53	0.606	0.65	1.56	0.95
C21	0.501	0.308	0.644	0.586	0.325	0.511	0.819	0.573	1.014	0.42	0.388	1.54
C22	0.435	0.176	0.946	0.487	0.139	0.863	0.55	0.24	1.43	0.968	0.99	1.62
C23	0.424	0.262	0.787	0.574	0.571	0.682	0.73	0.55	1.09	0.849	0.667	1.55
C24	0.263	0.541	0.695	0.263	0.302	0.735	0.51	0.47	0.96	0.82	0.836	1.77

Electrophysiological ITD Data

Subject	V 0 oz	V 0 ozamp	V .1oz	.1 oz am	V .4 oz	.4 oz am	V .9 oz	.9 oz am	V 1.9 oz	1.9 oz am
E1	5.82	0.79	5.86	0.9	5.95	0.849	7.06	0.75	7.71	0.616
E2	5.58	1.25	5.7	1	5.95	0.88	6.44	0.67	7.38	0.82
E3	5.62	1.15	5.66	0.98	5.7	1.12	6.56	0.438	7.67	0.599
E4	5.66	0.81	5.74	1.43	5.9	1.44	6.6	1.16	7.59	1
E5	5.82	1.38	5.99	1.19	6.15	0.79	6.52	0.56	7.1	0.26
E6	5.58	1.62	5.66	0.89	5.9	1.17	6.93	0.73	7.77	0.67
E7	5.82	0.70	5.86	1.32	6.63	0.38	7.43	0.462	7.67	1.08
E8	5.74	1.41	5.78	1.3	5.95	0.75	6.63	0.51	7.29	0.27
E9	5.78	1.99	5.9	2.17	6.03	1.73	6.44	0.79	7.63	0.71
E10	5.86	0.94	6.11	1.35	6.36	1.2	7.01	0.6	7.92	0.61
E11	5.95	0.98	5.9	1.52	6.4	0.83	6.52	0.73	7.51	1.09
E12	5.90	0.57	6.03	0.421	6.4	0.45	6.93	0.226	7.22	0.408
E13	6.03	0.96	5.99	1.5	6.07	1.12	6.69	0.587	7.59	0.69
E14	5.90	0.53	5.9	0.315	6.4	0.285	6.73	0.513	7.34	0.47
E15	5.70	1.08	5.9	1.21	6.11	0.94	6.89	0.5	7.67	0.96
E16	6.11	0.51	6.36	1.41	6.23	1.43	7.43	0.324	8.08	0.931
E17	5.66	1.21	5.78	1	5.99	0.998	6.64	0.32	7.55	0.91
E18	5.74	1.37	5.82	1.11	5.9	1.41	6.73	0.34	7.51	1.17
E19	5.74	0.52	5.86	0.66	6.03	0.68	6.69	0.45	7.71	0.57
E20	5.95	1.51	5.99	1.61	6.15	1.5	6.77	0.47	7.63	1.25
E21	5.58	1.16	5.62	1.1	5.95	0.84	6.81	0.72	7.47	0.6
E22	5.95	1.11	5.95	1.31	6.11	1.38	7.1	0.488	7.67	0.95
E23	6.27	0.70	6.4	1.17	6.56	0.801	7.47	0.393	8.21	1.1
E24	5.95	0.81	5.66	1.28	5.78	1.11	6.85	0.7	7.34	0.6

SUBJECT	V 0 oz	V 0 ozamp	V .1oz	.1 oz am	V .4 oz	.4 oz am	V .9 oz	.9 oz am	V 1.9 oz	1.9 oz am
C1	5.74	1.75	5.74	1.82	5.86	1.46	6.81	0.62	7.55	1.1
C2	6.07	0.56	6.11	0.77	6.27	0.385	7.01	0.308	7.63	0.57
C3	5.86	1.08	5.86	1.04	5.86	1.08	7.38	0.363	7.63	0.696
C4	5.62	1.43	5.7	1.12	5.82	0.84	6.52	0.21	7.3	1.17
C5	5.70	1.21	5.78	1.16	6.15	1.13	6.56	0.75	7.63	0.893
C6	5.82	1.43	5.82	1.34	5.99	1.09	6.93	0.69	7.59	0.61
C7	5.66	0.91	5.62	0.96	5.62	0.971	6.69	0.56	7.51	0.67
C8	5.74	0.94	5.82	0.71	5.82	0.39	6.81	0.33	7.43	0.27
C9	5.90	1.29	5.95	1.17	6.23	0.81	6.85	0.62	7.71	0.61
C10	6.78	1.58	5.86	1.18	6.15	0.69	6.85	1.22	7.59	0.964
C11	5.70	0.86	5.75	0.91	6.2	0.3	6.86	0.93	7.23	0.919
C12	5.80	1.00	6.07	1.34	6.19	1.41	7.01	0.568	7.96	1.05
C13	5.82	1.79	5.9	1.15	6.07	1.06	6.93	0.54	7.75	0.669
C14	5.90	1.38	5.95	1.19	5.99	1.08	6.97	0.86	7.8	0.94
C15	5.99	0.69	5.95	0.76	6.56	0.38	6.93	0.48	7.92	0.914
C16	5.78	0.79	5.82	0.84	5.9	0.71	6.97	0.77	7.06	0.644
C17	5.66	1.37	5.78	1.24	5.82	1.26	6.11	1.12	7.55	1.17
C18	6.03	1.11	6.07	1.21	6.23	1.1	6.89	0.301	7.88	0.536
C19	5.70	1.10	5.86	1	5.99	1.44	6.85	0.66	7.34	1.18
C20	5.95	0.95	6.03	1.17	6.23	0.76	6.97	1.52	7.8	0.94
C21	6.11	1.54	6.15	1.61	6.23	0.948	7.14	1.3	7.92	0.7
C22	5.58	1.62	5.62	1.93	5.7	1.54	6.52	0.71	7.18	1.48
C23	5.74	1.55	5.99	0.82	5.95	0.98	7.14	1.18	8	1.61
C24	5.49	1.77	5.58	1.82	5.66	1.64	5.86	1.49	7.18	1.71

APPENDIX C
REFERENCE NORMS FOR BEHAVIORAL TESTS

Test	Reference	Criteria
Time Compressed Speech	Beasley, D.S., & Maki, J. (1976)	Scores > 90% considered to be within normal limits
Pitch Pattern Test	Pinneiro & Musiek (1985) Bellis (2003)	7 yr. >/=35% 8 yr. >/= 40% 9 yr. >/= 65% 10 yr. >/= 72% 11 yr. >/= 75%
Duration Pattern Test	Pinneiro & Musiek (1985) Bellis (2003)	7 yr. >/= 25% 8 yr. >/= 40% 9 yr. >/= 65% 10 yr. >/= 72% 11 yr. >/= 75%
Masking Level Difference	Sweetow, R. & Reddell, R. (1978)	MLD thresholds 10 dB or greater are within normal limits
Random Gap Detection Test	Keith, R. (2000)	20 msec cut-off for normal

APPENDIX D

INFORMED CONSENT FORM

LOUISIANA STATE UNIVERSITY HEALTH SCIENCES CENTER IN NEW ORLEANS

Study Title: Early Electrophysiologic Recordings of Children at Risk for a Central Auditory Processing Disorder.

Performance Site: LSUHSC Speech and Hearing Clinic, 9th Floor, Rooms A13 & A16

Names and Telephone Numbers of Investigators: **Annette Hurley, M.S., CCC-A, FAAA**
Office**:** (504) 568-4348/4340
24 Hour Phone Number: Home: (225) 294-5206

Purpose of the Study:

This is a research study. The purpose of this study is to investigate behavioral and electrophysiologic recordings from the central auditory pathway. Only the data obtained from the routine audiological tests will be used for research purposes.

Description of the study:

A routine comprehensive audiometric evaluation will be performed on your child. This comprehensive evaluation will include pure tone thresholds, speech audiometry and immittance audiometry. Behavioral testing for APD will also be administered. This requires that your child listen and repeat filtered and degraded speech signals and tones. For electrophysiologic testing, your child will sit comfortably in a reclining chair during tests. Electrodes will be placed on his forehead and ears. The electrodes, which are small metal discs, are held in place by surgical tape after the surface of the skin has been cleaned. During the test, your child will be asked to remain still while he listens to tones through headphones. None of the tones will be too loud for comfort. He may signal at any time, for any reason if he would like to stop.

Benefits to Subject:

He will have contributed to the understanding of auditory processing.

Risks to Subject:

The study procedures are routine and have no known health risks.

Alternatives to Study Participation:

Not to participate in this study.

Subject Removal:

Failure to show up for three appointments without notification will be cause for removal from this study.

Subject Right to Refuse To Participate or Withdraw:

Participation is voluntary. Refusal to participate will involve no penalty or loss of benefits to which the subject is otherwise entitled, and the subject may discontinue participation at any time without penalty or loss of benefits to which the subject is entitled. Study subjects may refuse to participate or withdraw from the study at any time without jeopardizing, in any way, their medical treatment at this institution in the present or future. Should significant new findings develop during the course of the research which may relate to the subject's willingness to continue participation, that information will be provided to the subject.

Subject's Right to Privacy:

The results of the study may be released to the funding agency, the American Academy of Audiology. The results of the study may be published. The privacy of subjects will be protected and they will not be identified in any way.

Release of Information:

The medical records related to the study are available the sponsoring agency, the American Academy of Audiology, the Food and Drug Administration, and the LSUHSC IRB. While every effort will be made to maintain your privacy, absolute confidentiality cannot be guaranteed. Records will be kept private to the extent allowed by law.

Financial Information:

A. Participation in this study will not result in any additional charges above and beyond those routinely incurred by patients with similar conditions.

B. Subject Payment. Subjects who are self-referred to the clinic for an auditory processing disorder evaluation will receive this

comprehensive evaluation and report at no-charge. Patients who are not seeking such an evaluation will be reasonably compensated for their time and travel at rate of $10.00 per hour.

Signatures: The study has been discussed with me and all my questions have been answered. I understand that additional questions regarding the study should be directed to investigators listed on page 1 of this consent form. I understand that if I have questions about subject's rights or concerns, I can contact the Chancellor of LSU Health Sciences Center at (504) 568-4801. I agree with the terms above, acknowledge I have been given a copy of the consent form and agree to participate in this study. I understand that I have not waived any of my legal rights by signing this form.

_____ _____
Signature of Subject Date

_____ _____
Signature of Witness Date

Thee study subject has indicated to me that the subject is unable to read. I certify that I have read this consent form to the subject and explained that by completing the signature line above the subject has agreed to participate.

_____ _____
Signature of Reader Date

_____ _____
Signature of Person Administering Consent Date

_____ _____
Signature of Principal Investigator Date

The study subject is a child and I certify that I am his/her legal guardian.

Legal Guardian Name	Legal Guardian Signature	Date

ASSENT:

You will be asked to listen to words through headphones. Some of the words will difficult to understand because there will be noise in the background or other people speaking. You will listen and repeat the words. It will be okay for you to guess if you are not sure. You will be seated comfortably in a reclining chair during testing. Electrodes will be placed on your forehead and ears. The electrodes, which are small metal discs, are held in place by surgical tape after the surface of the skin has been cleaned. During the test, you will be asked to remain still while you listen to tones through headphones. During testing, you may watch silent videos. None of the tones will be too loud for comfort. You may signal at any time, for any reason, if you would like to stop.

_____

Child's Name & Age Child's Signature Date

Reason for not obtaining child assent:

VITA

Annette Hurley was born in Meridian, Mississippi. She received a Bachelor of Science degree in Liberal Arts from the University of Southern Mississippi in 1988. She continued her education at the University of Southern Mississippi and earned a Master of Science degree in audiology in 1990. She completed her clinical fellowship year at Louisiana State University Medical Center, Department of Otorhinolaryngology and Biocommunication, at Charity Hospital, New Orleans, Louisiana in May, 1990. She continued to work at Louisiana State University Medical Center for eight years, first as a clinical audiologist in the Lion's Clinic. Later, she began working as a research audiologist at Kresge Hearing Research Laboratory of the South under the incredible mentorship and wonderful supervision of Drs. Charles I. Berlin and Linda J. Hood.

Annette began pursing a Doctor of Philosophy degree in hearing science in August, 1996. During her doctoral studies, Annette worked as a course instructor in the Department of Communication Sciences and Disorders. She left Louisiana State University in 1998 and joined the faculty at Southeastern Louisiana University in Hammond, Louisiana. She was a recipient of the American Academy of Audiology Student Investigator Research Award in 2002. Currently, Annette is very happy to have returned to Louisiana State University Health Sciences Center, where she is now employed in the Department of Communication Sciences.