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**THE DEVELOPMENT OF AN OBJECTIVE MEASURE OF CENTRAL
AUDITORY FUNCTION USING LINGUISTICALLY RELEVANT AUDITORY
STIMULI**

**by
ANDRE MARCOUX**

**A thesis submitted to
The Faculty of Graduate Studies and Research
In partial fulfillment of
The requirements for the degree of**

Doctor of Philosophy

Department of Psychology

**Carleton University
Ottawa, Ontario
February 2001
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Acceptance of the thesis:

**The development of an objective measure of central auditory function using
linguistically relevant auditory stimuli**

submitted by

André Marcoux

in Partial fulfillment of the requirements for
the degree of Doctor of Philosophy

Chair

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Abstract

A distinctive feature of central auditory processing disorders (CAPD) is the difficulty of speech discrimination in situations of decreased audibility. The hypothesis evaluated in the present investigation was that the long-latency auditory evoked potential, mismatch negativity (MMN), would be different in CAPD patients and control participants in conditions of noise. Such a measure could augment or replace many behavioral tests of CAPD, an advantage when testing individuals who cannot provide a behavioral response to auditory stimuli. Seven control participants with no history of auditory impairment were compared to seven participants with a confirmed diagnosis of CAPD. Group selection was based on previous medical history and performance on the SCAN-A screening test. Evoked potentials to the speech sounds /ba/ and /da/ were recorded under four conditions: quiet (NoNoise) and three levels of noise (65 dB, 70 dB, and 75 dB). Behavioral measures of discrimination of the speech sounds were also obtained for each condition. As the level of noise was increased, the MMN response of patients was of lesser amplitude and greater latency in comparison to control participants. However, the MMN amplitude of the patient group was also significantly lower than controls across in the NoNoise condition. A post-hoc analysis of the N1 component also indicates a general reduction in amplitude in the patient group. Nevertheless, the MMN latency data is in accord with present knowledge of MMN, where the passive discrimination response is similar for patients and controls in the NoNoise condition, but there is a marked decrease in

passive discrimination occurs for the patient group as noise is increased. The MMN latency data also correlates with the recorded behavioral discrimination scores: patients and controls actively and passively discriminated speech sounds equally well in the NoNoise condition; patients actively and passively discriminated speech sounds more poorly than controls in the noise conditions. The MMN latency index was correlated with behavioral discrimination scores. These results indicate that MMN in conjunction with other auditory evoked potentials can assist in the assessment and description of CAPD.

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The development of an objective measure of Central Auditory Function using linguistically relevant auditory stimuli

This document describes the use of the mismatch negativity technique for assessing the discrimination of speech stimuli in noise and its potential as an objective indicator of central auditory processing. The first section of the introduction defines central auditory processing disorders (CAPD) and explains the importance of the diagnosis of such disorders. The second section describes in detail the inventory of tests which are available to diagnose CAPD as well some of the advantages and disadvantages which are associated with these tests. The third section defines the need for electrophysiological tests of CAPD and asserts the potential of the mismatch negativity (MMN) response to objectively and reliably demonstrate certain central auditory processing abilities.

Central Auditory Processing Disorders

Central Auditory Processing Disorder (CAPD) is not a label for a well-defined pathology but rather a description of functional deficits (ASHA, 1996). These deficits are normally revealed in patients despite the fact that they can hear (i.e. have normal auditory sensitivity). CAPDs have numerous etiologies and present themselves in a variety of ways, such as difficulty understanding speech in noise or competing speech backgrounds, distractibility, inattentiveness and short attention, poor auditory memory, difficulty understanding verbal instructions, difficulties in reading and general academic underachievement in areas of social competency such as reading, spelling, and mathematics (Jerger,

Martin & Jerger, 1987; Musiek, Geurkink, & Keitel, 1982; Olsen, Noffsinger, & Kurdziel, 1975). The number and degree of symptoms associated with specific cases of CAPD normally differs from one individual to another. However, patients with CAPD normally require better signal-to-noise ratios to achieve satisfactory auditory performance during dichotic listening, selective attention and temporal processing (Chermak, Vonhof, & Bendel, 1989). In sum, CAPD refers to a deficit in one or more processes responsible for the following behaviors: sound localization and lateralization; auditory discrimination; auditory pattern recognition; temporal aspects of audition including temporal resolution, temporal masking, temporal integration, and temporal ordering; auditory performance with competing acoustic signals; and auditory performance with degraded acoustic signals (ASHA, 1996).

Possible comorbid deficits such as attention deficit disorders and learning disabilities may also amplify the cognitive, social and emotional deficits observed in CAPD (for review, see Chermak & Musiek, 1997), thus making the diagnosis of CAPD quite challenging. Although difficult to ascertain whether the comorbid disorder is a result of CAPD or vice-versa, the comorbidity of disorders results from the involvement of non-dedicated processes, such as memory, attention and cognitive abilities, which are common to both CAPD and its comorbid disorders.

Misdiagnosed and left untreated, the possible deleterious effects of language-based disorders such as CAPD could involve life-long delays in cognitive, social and emotional areas. Clearly, an early diagnosis of CAPD would

be of great importance in order to promote appropriate listening and learning skills before the onset of serious academic delays. Tests are therefore of great importance in assessing deficits in auditory performance so that proper management strategies may be established. It is common practice to perform central auditory tests on children diagnosed with a learning disability (Martin, Champlin, & Chambers, 1998). However, because CAPD often manifests itself with other comorbid conditions such as a learning disorder, many cases of CAPD may go undetected for a number of years. It is therefore imperative to determine whether CAPD is truly contributing to the overall condition or whether it appears as a symptom of a comorbid condition (Chermak & Musiek, 1992; Keller, 1992).

As a result of its heterogeneous nature, even the concept of auditory processing disorder has been challenged as being a non-specific umbrella diagnosis (Peck, Gressard, & Hellerman, 1981). Although the fundamentals underlying the etiology, assessment and management of CAPD have been established, their validation is often questioned (McFarland & Cacace, 1995; Silman, Silverman, & Emmer, 2000). The following sections will offer an overview of the various tools which are available to assess CAPD.

Central auditory testing

Sensitivity and specificity. Test efficiency is derived from sensitivity and specificity indices (Turner, 1991). Sensitivity (hit-rate) in signal detection terms refers to the percentage of test results that indicate an abnormality when an abnormality truly exists. Specificity is the percentage of test results that indicates

that no abnormality exists when indeed no abnormality exists, which corresponds to correct rejection in signal detection terms. Although the best possible scenario in CAPD testing is to have both high sensitivity and specificity, this is not always possible. By changing the pass/fail criteria, one can increase sensitivity but doing so will tend to reduce specificity. Thus the pass/fail criteria will determine the sensitivity and specificity of a given test. Depending on clinical demands and clinical consequences, one may opt to increase either sensitivity or specificity by modifying the pass/fail criteria. One may need to decide if it is more desirable to diagnose as many patients as possible with a given pathology at the expense of also diagnosing patients who do not have this pathology (false positives) or whether it is more desirable to limit the number of false negatives at the expense of missing a few patients which may have the pathology of interest (false negatives).

The following CAPD tests all differ in their sensitivity and specificity for the detection of lesions of the central auditory nervous system (CANS). One must therefore refer to the literature concerning specific tests in order to assess these indices. Whenever possible, a summary of reported indices are noted. The clinician must therefore decide which tests will assess the area of interest with sufficient sensitivity and specificity during the selection of CAPD tests.

Test battery. Since many interdependent auditory processes are involved in the perception of an auditory event, a clinician may choose to select a number of tests, enabling the examination of a subset of these processes. A number of central auditory tests, either behavioral or electrophysiological, are currently

available, each with its strengths and limitations. Although most tests are valuable, they differ in their efficiency, validity and reliability. The optimal use of these tests is achieved by combining a number of central auditory tests in order to yield more information regarding CAPD. By doing so, one may be better suited to observe the marked difference in performance between normal individuals and patients with lesions of the central auditory nervous system (CANS), thereby establishing the foundation of CAPD testing. Nevertheless, one may choose specific tests to be included ad-hoc following suspicions of lesions in a specific area of the CANS (Pinheiro & Musiek, 1985).

Influence of cochlear hearing loss. The presence of cochlear or peripheral hearing loss greatly affects the performance of patients on tests of CAPD. It is therefore important to document the patient's hearing loss before any CAPD assessment takes place. Several investigations have studied the effects of cochlear hearing loss on CAPD test results. These have shown a decrease in test performance during most tests that will be described later in this section, including the Compressed Speech test, Low-Pass Filtered Speech (LPFS) test, Speech-in-Noise tests, Dichotic Sentences test, Dichotic CVs test, Dichotic Digits test, Staggered Spondaic Word (SSW) test, Masking Level Difference (MLD) test and all electrophysiological tests (Fifer, Jerger, Berlin, Tobey, & Campbell, 1983; Grimes, Mueller, & Williams, 1984; Kurdziel, Noffsinger, & Olson, 1976; Miltenberger, Dawson, & Raica, 1978; Musiek, Gollegly, Kibbe, & Verkest-Lenz, 1991; Noffsinger, 1982; Olsen, Noffsinger, & Carhart, 1976; Orchik & Burgess, 1977; Roeser, Johns, & Price, 1976; Speaks, Niccum, & Van Tasell, 1985).

However, some tests are more affected than others. For example, the Dichotic Sentences test and the Dichotic Digits test appear to be less affected by cochlear hearing loss than other behavioral CAPD tests (Fifer et al., 1983; Musiek et al., 1991; Speaks et al., 1985). Great caution must be given to the administration and interpretation of CAPD testing when cochlear hearing loss has not been previously documented.

Behavioral tests of CAPD

Behavioral tests remain the evaluation of choice for CAPD. However, these tests have practical constraints which do not make them adequate for several types of populations including very young children, children with attention and/or learning disorders, cochlear implant users, patients with speech production or motor disabilities, the elderly, and non-native English speakers. These tests are often not adequate for assessing their target population (i.e., those with CAPD) since many of the impairments described above are often comorbid in the CAPD population. These tests are often too long, too difficult to explain and they all possess an underlying cognitive task which may be too difficult to achieve in certain populations regardless of their auditory abilities. Motivation may also impact the performance during the testing session (Silman et al., 2000). In sum, it appears that central auditory tests are often not suitable for the CAPD population, rendering the diagnosis of this pathology rather challenging. However, behavioral tests of CAPD permit the assessment of complex central processing tasks which are not currently accessible by

electrophysiological tests. Some of the most utilized behavioral CAPD tests are described below.

Monaural low-redundancy speech tests. These types of tests were the first to be used in the assessment of CAPD. The use of speech material to evaluate CAPD is well justified. The case history of the CAPD patient normally reveals difficulty understanding speech, especially in conditions of background competition, and difficulty assimilating complex messages. These phenomena exist in spite of normal peripheral (cochlear) sensitivity. The use of speech stimuli contrasts with the use of tone stimuli. Because of the large amount of variation in intensity, frequency and temporal characteristics within the spectral envelope of speech signals as opposed to non-speech stimuli, there is little predictive power of an individual's ability to understand speech from a pure-tone threshold evaluation (Elliot, 1963; Harris, 1965; Marshall & Bacon, 1981; Preminger & Wiley, 1985). Suprathreshold speech tests are therefore necessary in order to assess an individual's auditory processing abilities with regard to speech, due to its omnipresence in the patient's environment and to the considerable acoustic complexity of the speech spectra (Olsen & Matkin, 1991).

However, it has often been documented that CAPD patients will perform normally during simple speech-recognition tests. These findings instigated the development of tests to assess central (cortical) auditory function, first described by Bocca, Calearo, and Cassinari (1954). These investigators determined that conventional tests of speech recognition were not suitable for the detection of lesions of the central auditory system. They concluded that only through speech

tests, modified to reduce message redundancy, could the integrity of the central auditory processes be evaluated (Bocca, Calearo, Cassinari, & Migliavacca, 1955; Calearo & Lazzaroni, 1957; Bocca, 1958; Calearo & Antonelli, 1968).

Therefore, in order for central auditory processing capabilities to be assessed, more complex or more difficult auditory (verbal or non-verbal) material must be presented to the patient in order to overtax the auditory system's normal processing capabilities.

Bocca and Calearo (1963) differentiated between extrinsic and intrinsic redundancy of the stimulus message as constituting the basis for differential diagnosis at the central auditory levels, where redundancy means "unnecessary" repetition. Normally, much redundancy exists during the processing of an auditory event, both extrinsic and intrinsic, and through a reduction of this redundancy the CANS can be properly overtaxed to reveal processing disorders.

Intrinsic redundancy refers to the redundancy of anatomical structures and pathways within the auditory system and to the encoded neural counterparts of the stimulus message, all of which are not necessary during speech perception.

The most simple illustration to demonstrate intrinsic redundancy is that because humans have two ears and each ear has complex ascending connections to the central auditory cortex, binaural information undergoes a duplication in auditory processing (i.e., parallel processing), one ipsilateral and one contralateral to the source of the auditory stimulus. A second level of intrinsic redundancy can be illustrated by the numerous contralateral projections which send auditory information to both the ipsilateral and contralateral ascending

pathways of the CANS (e.g., information received by the right ear reaches both the right [ipsilateral] auditory cortex and the left [contralateral] auditory cortex), and since both pathways are symmetrical, this results in a second level of parallel processing of auditory information. These contralateral projections are found at the acoustic stria, the commissure of Probst, the commissure of the inferior colliculus, and the corpus callosum.

A reduction in intrinsic redundancy of the auditory system will compromise the duplication of auditory information along the ascending auditory anatomy. Intrinsic redundancy can be affected in three major ways in CAPDs. Most CAPDs result from neuromorphological disorders such as polymicrogyri (i.e., underdeveloped and misshapen cells) and heterotopias (i.e., misplaced cells), most likely in the left hemisphere and the auditory region of the corpus callosum (splenium) (Musiek, Gollegly, & Ross, 1985). Other cases of CAPDs result from maturational delays of the central auditory system (Musiek, Kibbe, & Baran, 1984; Musiek et al., 1985) and from neurological disorders and neurodegenerative disorders (Musiek, Baran, & Pinheiro, 1992; Musiek et al., 1985).

Because of the duplication of auditory structures and pathways, it is probable that an alteration of the structures responsible for the processing of auditory information may not affect the processing of most auditory events despite the compromised nature of the communication and timing of activity within and across auditory regions. However, when speech material with low extrinsic redundancy is presented, the compromised central auditory system may

not have the necessary resources to process this abnormally distorted information.

Extrinsic redundancy refers to the large number of overlapping cues within the speech signal, all of which do not absolutely need to be perceived in order for an auditory event to be recognized. During speech tests, the speech items which offer the most extrinsic redundancy are sentences. Each word within the sentence offers some context for the other words. Monosyllabic words, on the other hand, have substantially less extrinsic redundancy since they do not have the context provided by other words (or from other syllables for that matter). It is therefore more difficult to correctly recognize isolated monosyllables as opposed to sentences. Therefore, one way extrinsic redundancy can be reduced is by utilizing shorter linguistic units. One can furthermore reduce extrinsic redundancy by utilizing nonsense syllables during speech-recognition testing (e.g., farg). Nonsense syllables do not have an entry in the mental lexicon and therefore have a minimal amount of extrinsic redundancy. The speech items must therefore be recognized by relying entirely on the recognition of each separate phoneme (or combinations of phoneme sequences [e.g., biphones] determined by probabilistic phonotactics [see Vitevitch & Luce, 1999]).

A reduction in extrinsic and/or intrinsic redundancy will affect the perception of speech events. Therefore, redundancy is beneficial to the speech perception process. However, as stated previously, individuals will perform near normal levels during speech-recognition tests if only one type of redundancy is reduced (i.e., reduced extrinsic redundancy with normal intrinsic redundancy and

vice-versa). Only once both intrinsic and extrinsic redundancies are reduced will declining performance be observed during speech tests (Mueller & Bright, 1994).

Other methods of reducing the redundancy of speech items have been the object of most behavioral tests of CAPD. The Low-Pass Filtered Speech (LPFS) test consists of monaurally presenting words which have been low-pass filtered with a filter cut-off frequency of 500 Hz (Willeford, 1976, 1977). The listener's task is to repeat the speech item to the tester. The speech-recognition score is obtained by calculating the percentage of correct responses. Although results vary with the test stimuli employed and the frequency cut-off and roll-off characteristics of the acoustic filters, this test normally reveals contralateral deficits in patients with lesions of the temporal lobe (Baran & Musiek, 1991; Musiek, Baran, & Pinheiro, 1994). Essentially normal results have been shown in patients with lesions of the interhemispheric pathways and no consistent result has been measured in patients with lesions of the brainstem (Baran, Musiek, & Reeves, 1986; Gilroy & Lynn, 1974; Karlsson & Rosenhall, 1995; Lynn, Benitez, Eisenbray, Gilroy, & Wilner, 1972; Lynn & Gilroy, 1971, 1977; Musiek et al., 1984; Musiek, Wilson, & Pinheiro, 1979). Studies have indicated that the LPFS has moderate sensitivity to the presence of CANS lesions and that it is not useful in locating the specific location of the lesion.

The Compressed Speech test consists of shortening the duration of words or sentences (Beasley, Forman, & Rintelmann, 1972; Beasley, Schwimmer, & Rintelmann, 1972). Test performance decreases with increased compression. Deficits in test performance indicate contralateral ear deficits to lesions of the

CANS, with occasional ipsilateral deficits in patients with lesions of the left CANS (Baran & Musiek, 1991; Baran, Verkest, Gollegly, Kibbe-Michal, & Rintelmann, 1985; Kurdziel et al., 1976). Results vary depending on deficits in patients with lesions of the brainstem (Calearo & Antonelli, 1968; Quaranta & Cervellera, 1977). Despite the lack of normative data for this test, it appears that it is moderately sensitive to lesions of the CANS but not particularly helpful in determining the location of the lesion (Baran et al., 1985; Karlsson & Rosenhall, 1995). The Synthetic-Sentence Identification with Ipsilateral Competing Message (SSI-ICM) test consists of presenting a number of non-meaningful sentences (e.g., Small boat with a picture has become) to one ear while presenting a meaningful discourse in the same ear at various message-to-competing discourse ratios (Jerger & Jerger, 1974). The listener's task is to find the corresponding number of the sentence that is heard (from a closed-set type-written list) and verbally relate the answer to the examiner. The SSI-ICM is highly sensitive to lesions of the brainstem but produces essentially normal results in patients with cortical lesions (Antonelli, Bellotto, & Grandori, 1987; Jerger & Jerger, 1974; Jerger & Jerger, 1975; Stephens & Thornton, 1976).

Noise, an extraneous source of auditory interference, competes with other auditory information, thereby acting as a masking agent and reducing message redundancy. The introduction of an ipsilateral competing noise or message can seriously impair the word recognition process, thus making this task more laborious (Heilman, Hammer, & Wilder, 1973; Jerger, Martin, & Jerger, 1987; Morales-Garcia & Poole, 1972; Olsen et al., 1975). Normally, however,

individuals are able to attenuate the effect of noise competition in certain listening situations. Warren (1970) noted that since many extraneous sounds are present in most listening environments, humans must have some way of blocking or suppressing this interfering information during the perception of auditory information. Warren noted that at the phonemic level certain phonemes could easily be distorted or misunderstood in the presence of extraneous sounds. However, Warren concluded from a rather simple study that humans were able to perceptually restore speech sounds that had been obliterated with noise, hence the term phonemic restoration (Warren, 1970). In fact, individuals are still quite capable of achieving auditory discrimination in the presence of competing noise if sufficient contextual information is present to unconsciously fill-in-the blanks (Marcoux & Logan, 1999; Samuel, 1981, 1996). However, individuals who may have disorders of the auditory system will tend to perform more poorly during such tests. Research has shown that listeners with reduced hearing function will show a sharp reduction in speech-recognition scores with small decrements in extrinsic redundancy (i.e., increments in the level of noise presented with speech items). Although, certain researchers have found that adding noise to a speech recognition test adds complexity to the interpretation of results without adding any sensitivity to speech-recognition testing (Danhauer, Doyle, & Lucks, 1985, 1986), speech-recognition testing against background competition enhances the sensitivity of speech-recognition tests by separating the performance of normal listeners and those with auditory disorders (Cohen & Keith, 1976).

There have been many efforts made to develop speech in noise tests to

enhance the sensitivity of audiometric speech tests in detecting and demonstrating difficulties in auditory function with regard to word recognition and other communication skills (Bilger, Nuetzel, Rabinowitz, & Rzeczkowski, 1984; Elliott, 1995; Kalikow, Stevens, & Elliott, 1977; Morales-Garcia & Poole, 1972; Olsen et al., 1975). Researchers and clinicians have not been able to agree on what levels of noise should be used during these tests and this lack of uniformity has resulted in a lack of norms for these tests. Mueller and Bright (1994) describe these tests as being some of the most misused tests of central auditory function. Tests which use ipsilateral noise competition reveal reduced performance in the contralateral ear of patients with lesions of the auditory cortex (Heilman et al., 1973; Morales-Garcia & Poole, 1972), reduced performance in the ipsilateral ear of patients with extra-axial lesions of the brainstem (Dayal, Tarantino, & Swisher, 1966) and in one or both ears of patients with intra-axial lesions of the brainstem (Morales-Garcia & Poole, 1972; Noffsinger, Olsen, Carhart, Hart, & Sahgal, 1972). As with other tests in this category, tests with ipsilateral competition are moderately sensitive in detecting CANS lesions but are not helpful in determining the location of the lesion.

Temporal processing tests. Temporal processing is an important component of central auditory function (Bornstein & Musiek, 1984; Pinheiro & Musiek, 1985). The assessment of temporal processing is measured after presenting two or more successive acoustic stimuli. The listener's task is to repeat these stimuli in the order in which they were initially presented. The Frequency Pattern test (Musiek & Pinheiro, 1987; Pinheiro, 1977) test consists of

presenting a series of three brief 150 ms tones, which are a combination of 880 Hz or 1122 Hz sinusoids with a 200 ms interstimulus interval. The various combinations yield six patterns. The listener must repeat the combinations in the correct order using two modalities, one verbal and the other hummed or gestured. The Duration Pattern test (Musiek, Baran, & Pinheiro, 1990) exploits temporal processing in two ways: ordering of tonal stimuli and duration discrimination. This test uses combinations of three 1000 Hz tones which vary in duration (either 250 ms or 500 ms with a 300 ms interstimulus interval). The various combinations yield six patterns. The listener must repeat the combinations in the correct order using the verbal modality only. Both the Frequency Pattern test and the Duration Pattern test have a sensitivity of approximately 85% (i.e., 85% of test results were abnormal when a true abnormality exists) and a specificity of approximately 88% (i.e., 88% of test results were normal when no true abnormality exists) (Musiek et al., 1990; Musiek & Pinheiro, 1987). These rates were established using listeners with cortical lesions and for those with mild-to-moderate sensorineural hearing loss.

Other less utilized tests of temporal ordering are the Two-Element Ordering test (Swisher & Hirsh, 1972), and the Brief-Tone Ordering task (Cranford, 1984). The Two-Element Ordering test consists of ordering two different short acoustic stimuli. Swisher and Hirsh documented the elevated time of response for CAPD patients compared to normal patients. This test is sensitive to cerebral lesions, especially of the left temporal lobe. The Brief-Tone Ordering task consists of establishing a frequency difference limen (DL) for both short and

long tones. The frequency difference limen consists of the smallest detectable difference between two frequencies. Results show that the DL for long tones is similar for normal and CAPD patients but that the DL for short tones is much smaller in normal patients than in CAPD patients.

Tests of binaural integration and separation. Many tests which use dichotic presentation (i.e., different stimulus presented to each ear) are sensitive to CAPD. The listener's task during dichotic tasks is to either separate the messages from both ears by extracting a target event from the dichotic messages or to integrate the messages from both ears. Two of the most widely used binaural separation dichotic tests are the Synthetic Sentence Identification with Contralateral Competing Message test (SSI-CCM) (Jerger & Jerger, 1975) and the Competing Sentences (Willeford, 1976) test. The SSI-CCM, like the SSI-ICM, consists of presenting a number of non-meaningful sentences (e.g., Small boat with a picture has become) together with a meaningful discourse. However, unlike the SSI-ICM, where both the sentences and the discourse are presented to one ear, the SSI-CCM consists of presenting the sentence to one ear while presenting the discourse to the contralateral ear at various message-to-competing discourse ratios. The listener's task is to find the corresponding number of the sentence that is heard (from a closed-set type-written list) and verbally relate the answer to the examiner. The SSI-CCM is sensitive to cortical lesions, where a reduction in scores is observed when sentences are presented to the ear contralateral to the lesion. The Competing Sentences test consists of repeating a target sentence presented to one ear while a competing sentence is

presented to the contralateral ear. This test is less sensitive than the SSI-CCM since linguistic context is available to the listener.

Commonly used dichotic tests of binaural integration are the Staggered Spondaic Words (SSW) test (Katz, 1962), dichotic digits, dichotic consonant-vowels (CVs), and the dichotic sentence identification test (DSI). The SSW consists of presenting different spondee words to each ear with a staggered onset, where the first half of one stimulus is presented in a non-competing condition, followed by a competing condition with the first half of the second word, followed by the second half of the second word presented in a non-competing condition. The listener's task is to repeat both spondee words. The scoring procedure of the SSW is complex. However, Katz has demonstrated a correlation between certain SSW results and the site of CANS lesion, either in the brainstem, primary auditory cortex or associated auditory cortical areas (Katz, 1962, 1968, 1970; Katz, Basil, & Smith, 1963; Katz & Ivey, 1994). Studies have demonstrated sensitivity of approximately 70% for patients with lesions of the CANS or of the brainstem. However, less desirable hit-rates for patients with brainstem involvement have also been reported (Musiek & Guerlink, 1982; Rintelmann & Lynn, 1983). The dichotic digits test (Kimura, 1961a, 1961b) consists of presenting two digits (i.e., numbers 1 to 10, except 7) simultaneously, one in each ear, and asking the listener to repeat both digits. This test is quite sensitive to lesions of the temporal lobe, showing contralateral deficits in patients with right temporal lobe lesions and bilateral or contralateral deficits in patients with left temporal lobe lesions (Baran & Musiek, 1991, Musiek et al., 1994).

Studies have reported hit-rates of approximately 80% and false-positive rates of under 15% in patients with lesions of the temporal lobe (Musiek, 1983; Musiek et al., 1991). The dichotic CV test (Berlin, Lowe-Bell, Janetta, & Kline, 1972) is similar to the dichotic digits test except that the following CV (consonant-vowel) combinations are used: /ba, pa, da, ta, ga, ka/. The onset of the presentation of the two CVs can either be simultaneous or slightly staggered. This test is mostly useful for diagnosis of lesions of the temporal lobes. Similar hit-rates to the dichotic digits test are observed using the dichotic CV test (Olsen, 1977; Olsen & Kurdziel, 1978). Furthermore, a sensitivity of 93% and a specificity of 55% have been reported in patients with left temporal lobe lesions (Mueller, Beck, & Sedge, 1987). The dichotic sentence identification test (DSI) is a modification of the SSI-CCM which attempts to correct the silent gaps of the SSI-CCM competitive narration by using instead a paired competitive sentence. Again the listener identifies the numbers of the two sentences from a closed set. Results have suggested that the DSI is sensitive to lesions of the auditory cortex (Fifer et al., 1983; Mueller et al., 1987).

Tests of binaural interaction, lateralization, and localization. The tests included in this category differ somewhat from one another. Their commonality is that the ipsilateral and contralateral auditory systems must interact after the test item has been heard in order for the correct response to be obtained. The Masking Level Difference test (MLD) consists of presenting speech or tones to both ears at the same time that a noise is being delivered binaurally (Licklider, 1948). The patient is tested under two conditions, a homophasic and an

antiphasic condition. In the homophasic condition the speech stimuli and the noise are presented to both ears in phase (S_0N_0). In the antiphasic condition, one of the two signals is presented 180° out of phase and the other signal is presented in phase between the two ears (e.g., $S_\pi N_0$, where the signals are presented out of phase while the noise is presented in phase, or S_0N_π , where the signals are kept in phase and the noise is presented out of phase). Typically, when the signal or noise is out of phase, there is a release from masking and the thresholds at which the signal can be detected improves. The MLD corresponds to the difference in threshold between the homophasic and either antiphasic conditions.

Patients with lesions of the lower brainstem show little release from masking in the antiphasic condition whereas patients with lesions of the cerebrum perform similarly to normal patients on the MLD. Studies have reported sensitivity and specificity of more than 70% for patients with brainstem lesions (Karlsson & Rosenhall, 1995; Lynn, Gilroy, Taylor, & Leiser, 1981).

Tests of lateralization consist of presenting clicks with an interaural time differential or an interaural intensity differential (Groen, 1969; Hausler & Levine, 1980; Levine, et al., 1993; Matathias, Sohmer, & Biton, 1985; Pinheiro & Tobin, 1969; Quine, Regan, & Murry, 1983). With time differentials, the click that leads in time will attract the resulting sound image formed by both clicks to the side of the head to which the leading click is presented. Correspondingly, with intensity differentials, the click with the greater intensity will attract the resulting sound image to the side of the head to which the louder click is presented. Sensitivity

and specificity data are not available for these types of tests. However, the test has been successfully applied to diagnose multiple sclerosis (with or without CANS involvement) (Hausler & Levine, 1980; Levine et al., 1993; Quine et al., 1983). These patients did not direct the sound images to one specific side of the head but perceived them as being heard at mid-line. Tests of auditory localization are similar to those of lateralization but require listeners to pinpoint the exact location of the sound image as opposed to simply observing on which side of the head the sound image is formed (Moore, Cranford, & Rahn, 1990; Sanchez-Longo & Forster, 1958). Initial data showed a sensitivity of 57% and a specificity of 80% in the identification of lesions of the CANS from a population of 50 patients (Sanchez-Longo & Forster, 1958). A sensitivity of 88% was obtained in patients with multiple sclerosis (Cranford, Boose, & Moore, 1990).

Electrophysiological tests of CAPD

Electrophysiological tests are often used to supplement the behavioral component of a test battery for the evaluation of CAPD. The advantage of these procedures is that they often do not require the attention or active participation of the individual being tested and require no higher-order cognitive resources. (For a review of electrophysiological tests, see Hall, 1992). Overall, electrophysiological tests reduce the possibility of behavioral contamination caused by behavioral and pathological manifestations comorbid to CAPD such as hyperactivity, inattentiveness, learning disabilities and non-native language (Jerger, Chmiel, Tonini, Murphy, & Kent, 1999; Jerger & Allen, 1998; MacFarland

& Cacace, 1995; Sloan, 1986). Thus, patients who are unable to perform normally during behavioral CAPD tests because of their inability or unwillingness to properly respond could potentially be assessed using electrophysiological tests. For this reason, it is important that electrophysiological correlates of auditory processing contribute to a better definition and delineation of disorders of auditory processing in populations where behavioral tests are difficult to administer (Cone-Wesson & Wu, 1992; Gravel, Kurtzberg, Stapells, Vaughan, & Wallace, 1989; Jerger & Jerger, 1975; Jirsa & Clontz, 1990).

The Auditory Brainstem Response (ABR) was designed to diagnose lesions of the auditory pathways from the auditory nerve to the lateral lemniscus of the brainstem (Starr & Achor, 1975). The ABR consists of five waves, labelled I to V, which represent the synchronous activity generated by the auditory nerve and the brainstem (Hall, 1992; Moller, 1985). Waves I and II of the ABR represent the generation of activity from the auditory nerve, while wave III is generated by the cochlear nucleus of the lower brainstem and the IV-V wave complex is generated by several auditory centers, including the superior olivary complex and the lateral lemniscus (Moller, 1985).

The ABR is elicited by clicks or tone pips. In order to obtain a waveform that can then be interpreted, the elicited response is recorded within 12 ms after onset of the stimulus, averaged over thousands of trials and filtered to eliminate unwanted neuro-muscular or environmental electrical activity. The ABR is recorded from electrodes which are strategically placed on the scalp, forehead and on each mastoid, following the 10-20 International Electrode System, in

order to create differentials in potential (i.e. dipoles).

The most useful ABR indices of retrocochlear pathology are the interpeak latency interval index and the interpeak amplitude index (Elidan, Sohmer, Gafni, & Kahana, 1982; Giffiths, Chambers, & Bilger, 1989). The ABR remains a test of choice to evaluate the integrity of the brainstem and is often used during CAPD assessment. The sensitivity and specificity of the ABR is highest when applied to the diagnosis of acoustic neuromas and tumors of the eighth nerve (Hall, 1992; Musiek, 1991; Musiek, McCormick, & Hurley, 1996). However, the ABR is also quite useful when applied to the diagnosis of lesions of the brainstem, with a sensitivity of approximately 80% (Musiek & Lee, 1995).

The Middle Latency Evoked Response (MLR) (Geisler, Frishkopf, & Rosenblith, 1958) is an auditory evoked potential which reflects the synchronous activity of multiple generators including the thalamocortical auditory pathway, and to a lesser degree from the inferior colliculus, the mesencephalic reticular formation and the auditory reception area of the temporal lobe (Kraus, Kileny, & McGee, 1994).

Like the ABR, the MLR is also elicited by clicks or tone pips but requires a slightly more complex electrode montage. Since the ipsilateral and contralateral auditory pathways start to diverge from the mid-line as they approach the temporal lobes, hemisphere-specific electrodes are necessary (e.g., C3, C4, with Cz) (see Figure 4) in order to evaluate the possibility of unilateral lesions of the CANS which may not be detectable with a mid-line montage.

Averaging of the MLR, in a time period between 12 ms and 50 ms, reveals

both positive and negative voltage waves (i.e., Na, Pa, Nb, Pb).

In order to assess CAPD, the clinician relies on the presence of the MLR and on hemispheric differences of latency and amplitude. The best index in the diagnosis of CAPD is the comparison of Na-Pa amplitudes obtained for electrodes placed over each hemisphere. Normally, the electrode closest to the lesion will record potentials of smaller amplitude as opposed to the electrode placed over the contralateral hemisphere, regardless of the ear receiving stimulation. This phenomenon is termed the electrode effect, and differences of more than 50% are indicative of lesions of the CANS (Musiek, Baran, & Pinheiro, 1994). Ear effects, which show reduction of amplitude when one ear is stimulated, regardless of electrode site, can also be useful MLR index, although less valuable than the electrode effect (Shehata-Dieler, Shimizu, Soliman, & Tusa, 1991). Sensitivity and specificity data are unavailable regarding the use of the MLR towards the diagnosis of CAPD.

Long latency potentials have been investigated for many years. These responses can consist of stimulus-related exogenous potentials (SRPs) and/or event-related endogenous potentials (EPs).

The SRPs consist primarily of a negative peak, N1, occurring from 80 to 100 ms, a positive peak, P2, occurring at 140 to 220 ms, and a small negative peak, N2a, at about 280 to 320 ms (Kileny, 1985). The EPs, which are elicited by internal cognitive activity through any given sensory modality, consist of a variety of highly variable waveforms occurring beyond N2a (Musiek, Verkest, & Gollegly, 1988). The exogenous potentials (SRPs) N1 and P2 are directly dependent on

stimulus characteristics and are independent of the listener attending to or actively processing the stimulus. The N1 and P2 complex were the first late potentials to be used (Davis, 1939). The N1 is generated by the primary auditory cortex (Knight, Hillyard, Woods, & Neville, 1980; Vaughn & Ritter, 1970) while the P2 originates either from the temporal lobes and the limbic system (Hari, Aittoniemi, Jarvinen, Katila, & Varpula, 1980; Vaughn & Ritter, 1970) or from the primary auditory cortex, along the Sylvian fissure on the side contralateral to stimulation (Baumann, Rogers, Papanicolaou, & Saydjari, 1990). These components are elicited by presenting repeated auditory stimuli (e.g., /ba, ba, ba/, etc.). The N2a exogenous potential, also termed the mismatch negativity (MMN) is generated by the primary auditory cortex. The MMN is elicited by presenting repeated auditory stimuli within an oddball paradigm (e.g., /ba, ba, ba, ba, ba, da, ba, ba/, etc.), where infrequently occurring deviant stimuli (e.g., /da/) are embedded within a series of frequently occurring standard stimuli (e.g., /ba/). The N1 and P2 are obligatory responses and are elicited by either the audible standard or deviant stimuli (Näätänen, 1995). The Mismatch Negativity (MMN) (or N2a) is a discriminative response to the difference between the standard and deviant stimuli, and is thus only elicited with presentation of the deviant stimuli (Aaltonen, Niemi, Nyrke, & Tuhkanen, 1987; Sams, Aulanko, Aaltonen, & Näätänen, 1990). The MMN appears as either an enlarged N1, a second negative peak, or a reduction in P2 amplitude (Picton, 1990). This potential will be the main focus of discussion in the next section.

The P3, an endogenous potential, occurs approximately 300 ms after

onset of the deviant or significant stimulus within the oddball paradigm. What separates this potential from the exogenous potentials is that the listener must actively detect the deviant stimulus within the oddball paradigm (Sutton, Braren, Zubin, & John, 1965). The generators of the P300 are not known although support is given to the involvement of the hippocampus and/or the posterior temporal lobes as contributors to this potential (McPherson, 1996). Clearly, areas of the brain which are involved in memory could contribute to the generation of the P300. The ideal electrode montage in recording the P300 is Fz, Cz, and Pz.

Late evoked potentials can be elicited by clicks, tone pips, and yet more complex stimuli such as speech. However, speech materials are not readily used with this type of testing because speech is comprised of complex patterns of frequency, intensity and timing which involve a multitude of central structures and connections, which in turn will only produce an electrophysiological response which is excessively difficult to control and quantify. Therefore, simple auditory stimuli are used while measuring late evoked potentials. However, few data are available on the sensitivity and specificity of these potentials. Furthermore, there is little consensus on which attributes of these potentials should be used as indicators for CNS involvement. Latency, amplitude, ear effects and electrode effects have been used in various studies (Knight et al., 1980; Musiek, 1991; Musiek et al. 1992).

Because of the lack of data and strong support for electrophysiological testing when diagnosing CAPD (with exception of the ABR), and because of the expensive equipment used to record these potentials, the use of late evoked

potentials has been limited. Furthermore, because the central processes responsible for deficits in speech and language occur in auditory regions which respond to auditory stimuli after several hundreds of milliseconds, the shorter, more documented electrophysiological measures, such as the ABR, are of little use to evaluate CAPD. Furthermore, the use of clicks and tone bursts is insufficient to probe central auditory processes. In sum, complex measures, such as long-latency potentials, combined with complex auditory stimuli such as speech, are necessary for the electrophysiological assessment of CAPD.

Position of the problem

An electrophysiological test would be the test of choice during the assessment of CAPD. The advantage of using these tests would be that they do not require the active and sustained participation of the listener and are therefore not contaminated relative to the level of attention or cognitive resources the individual may possess. Furthermore, electrophysiological tests are most appropriate for listeners who are unable to respond because of age, motor impairments or who are unable to comprehend or grasp complex behavioral tasks. Because of the well-documented comorbidity of certain other deficits, such as hyperactivity, attention deficits and learning disabilities with CAPD, it is important to obtain an uncontaminated measure of central auditory processing abilities in order to give an unconfounded diagnosis of CAPD. Although electrophysiological tests may be theoretically preferred during the assessment of CAPD, the use of these tests has not been extensive because of the limited

amount of auditory stimuli compatible with electrophysiological procedures. In sum, the nature of auditory stimuli routinely used in conjunction with electrophysiological tests is too simple to overtax the central auditory system in the same manner as some of the more complex stimuli used in behavioral tests, thus limiting the number of auditory processes which may be explored. The MMN is the first electrophysiological test that offers the flexibility of using complex stimuli, such as words, while testing central auditory function without the attention or active participation of the listener. Thus, the MMN is as suitable candidate to bridge the advantages of electrophysiological testing with the advantage of studying processes of central auditory dysfunction as presently done using behavioral testing with complex stimuli.

The following sections will review pertinent issues regarding central auditory dysfunction and how MMN, a late auditory evoked potential, can be applied as an assessment tool. The use of the MMN using speech stimuli is appropriate because of its potential contribution towards a better assessment and understanding of central auditory dysfunction. Later sections will present a methodology for using complex auditory stimuli in conjunction with the MMN measure to probe central auditory dysfunction.

Mismatch Negativity: An objective measure of auditory processing

What is Mismatch Negativity? MMN is an exogenous event-related potential (ERP) which has previously been applied to the assessment of auditory stimulus detection and discrimination (Kraus, et al., 1993c). MMN is a neuronal

response that is elicited by an infrequent change in the physical attributes of an acoustic waveform. In the prototypical MMN paradigm, an MMN is elicited by occasionally replacing a repetitive auditory stimulus (standard) by an infrequent auditory stimulus (deviant). The MMN has been elicited by small acoustic differences in auditory stimuli which may deviate with respect to differences in tonal frequency as small as 8 Hz (Näätänen, Gaillard, & Mäntysalo, 1978; Sams, Paavilainen, Alho, & Näätänen, 1985), differences in intensity as little as 6 dB (Näätänen, Paavilainen, Alho, Reinikainen, & Sams, 1987), and complex phonetic changes (Aaltonen et al., 1987). The MMN can occur when the acoustic difference is near psychological/behavioral threshold and is thus an indicator of behavioral discrimination (Sams et al., 1985). The MMN is relatively unaffected by the level of attention or the active participation of the listener and is thus said to be elicited automatically (Näätänen, 1992).

The effects of attention and relevance of the stimulus on the mismatch negativity response. Näätänen (1975) proposed that any stimulus deviation per se, regardless of its meaning or significance, could produce a cognitive response (EP) that could be measured from the scalp of human subjects. Näätänen and his colleagues conducted two experiments in which they were able to illustrate that infrequent (deviant) stimuli, differing either in tonality or frequency, produced a negative shift in the cortical response compared to frequent (standard) stimuli (Näätänen et al., 1978). In one of those experiments, which used dichotic stimulus presentation, the participants were instructed to count the deviant stimuli in only one ear (relevant stimuli), while ignoring the stimuli in the other ear

(irrelevant stimuli). Standard and deviant stimuli were presented randomly in sequence and in location (left or right ear). The irrelevant stimulus sequence included deviant stimuli that were physically equivalent to the deviant stimuli (targets) of the attended input sequence. Note that this negative component appears whether the deviant was presented in the attended (left) or unattended ear (right).

Surprisingly, it was found that the deviant stimuli in both the attended and unattended stimulus sequence elicited a negativity at the 120 to 200 ms latency range, which could not be seen in the response to the standard stimuli. This was contrary to what had been observed in previous studies, which suggested evoked potentials were greater in the attended modality. Näätänen et al. (1978) proposed that:

“it may well be that a physiological mismatch process caused by a sensory input deviating from the memory trace (“template”) formed by a frequent background stimulus is such an automatic basic process that it takes place irrespective of the intentions of the experimenter and the subject, perhaps even unmodified by the latter. This view is supported by the fact that the mismatch negativity was similarly observed for both the attended and unattended sides. Hence, we may here be dealing with a deviation effect rather than relevance effect, whereas the much larger P300 in the EPs to the attended signals than to the unattended signals certainly represents a relevance effect”.

Characterization of the mismatch negativity response. Close examination of the mismatch response shows a clear waveform difference that is not detected by simple N1-P2 measurements. The MMN, which usually peaks between 100-200 ms after deviant-stimulus onset, is represented by a negative wave which overlaps the negative N1 (peak latency about 100 ms from stimulus onset) and subsequent positive P2 components which are elicited by both standard and deviant sounds (Näätänen et al., 1978). The MMN is a small negative deflection that is superimposed on the waveform in the latency range of the P2 wave. Its amplitude varies in proportion to the degree of deviance, the probability of occurrence of the deviant stimulus, and the rate of stimulus presentation (Näätänen, 1992). With increased difference between standard and deviant stimuli, and/or a decrease in probability of occurrence of the deviant stimulus the MMN amplitude increases until it reaches a plateau whereas its latency and duration continue to shorten. The MMN is largest at interstimulus intervals (ISI) of less than 4 s, becomes smaller over longer ISIs, and disappears with ISIs larger than 10 s (Näätänen et al., 1987). The MMN is obtained by subtracting curves in which the corresponding time points of the evoked potential (EP) to standards are subtracted from the EP to deviants (Näätänen et al., 1978).

Generation of the mismatch negativity response. The MMN amplitude is relatively large when recorded by scalp electrodes over the temporal areas. It was therefore suggested by Näätänen et al. (1978) that the MMN reflects specific auditory stimulus discrimination processes taking place in the primary and associative auditory cortices. A second intracranial generator, the frontal cortex,

was also proposed. The sensory-specific auditory cortex perceptually detects stimulus change, whereas the subsequent frontal activation might be associated with attention switch to stimulus change (Näätänen & Michie, 1979).

More direct evidence for the involvement of the auditory cortex in MMN generation was provided by the first magnetoencephalographic (MEG) recordings of the MMN (Hari, et al., 1984; Sams, et al., 1985), which located the equivalent current dipole of the MMNm (the magnetic equivalent of the MMN). These studies showed that the MMN is probably generated at the supratemporal plane at or in the vicinity of the primary auditory cortex. Other MEG studies have confirmed this observation and provided evidence for the separability of the MMNm and N1m (the magnetic equivalent of the supratemporal N1) generator loci (Alho, 1995).

Subsequent electric and magnetic studies also showed that the exact location of the MMN provided evidence for different generator sites in the auditory cortex for MMNs to deviations of different auditory attributes (i.e., frequency, intensity, duration) (Alho, et al., 1996; Paavilainen, Alho, Reinikainen, Sams, & Näätänen, 1991).

Näätänen and Michie (1979) had originally proposed that the MMN may also be composed of a frontal-lobe component, which may direct the listener's attention to the deviant stimuli. In 1990, Giard, Perrin, Pernier, and Bouchet mapped electrical brain generators during a typical MMN experiment. These topographic maps provided evidence for both auditory-cortex and predominantly right-hemispheric frontal involvement in MMN generation. They interpreted the

frontal activation in terms of an involuntary attention switch to the eliciting acoustic change, constant with Näätänen and Michie (1979) interpretation. They also concluded that the electrical response from the auditory cortex was bilateral but larger contra-than ipsilaterally to the stimulated ear, which reflects the involvement of auditory centers beyond the brain-stem cross-over (Giard et al., 1990).

The mismatch negativity response as a probe into auditory processing.

The MMN becomes very useful in light of several research and clinical issues: a) The MMN is elicited by any discriminable change in a repetitive sound that can be typically detected at the conscious level. b) The MMN provides an objective measure of one's discrimination ability for different simple and complex (such as speech) sound features. c) The MMN can be elicited without attention, and is therefore uncontaminated by attentional variations of attention-dependent physiological components of auditory function. This objectivity can furthermore be maintained while testing uncooperative or unresponsive individuals. d) Because central auditory representations are formed during MMN generation, the MMN provides a unique window to view the neurophysiological processes underlying normal hearing. These representations are physiological reflections of acoustic processing by the central auditory system and the detection of deficits may give a better idea of the precision of tone and speech discrimination. e) Finally, the MMN also provides a method for studying auditory short-term memory, which is of crucial importance for accurate speech processing and comprehension and is also a major reason central auditory dysfunctions

(Näätänen, 1995).

The MMN has been viewed as a prime candidate for an objective test of central auditory processing ever since it was shown that the MMN is present when the deviant stimuli are just discriminable from the standard stimuli but not when the difference is not perceptible, given that the listener is focused on the discrimination task (Aaltonen et al., 1987). Furthermore, the MMN a) occurs when the listener discriminates between stimuli at intensities similar to those used in normal speech, b) can be recorded when the differences between the deviant and standard stimuli are just noticeably different, and c) occurs whether the listener is attending to the stimuli or not (Sams et al., 1985). This is of considerable importance considering that previous research has shown that children with learning disabilities require larger acoustic differences to discriminate speech sounds than normal children (Elliott & Hammer, 1988; Elliott, Hammer, & Scholl, 1989). For example, acoustic cues that signal place of articulation appear to be particularly vulnerable when learning disabilities affect auditory processing of these phonemes (Rosen, 1992). Physiological research has demonstrated how central auditory neurons play an important role in decoding fine temporal aspects of the auditory signal (Phillips & Farmer, 1990).

The MMN may help define processing capabilities of the central auditory cortex. For example, it may be used to delineate certain learning disabilities which are presently still widely misunderstood and grouped together under a non-specific disorder label (Kraus & McGee, 1994). The MMN could provide a means to distinguish those children with central sensory deficits from children

with disorders which may have different etiologies. Using the MMN along with the CAPD patient's behavioral response may eliminate many potential contaminant factors encountered during CAPD assessment using behavioral tests alone.

It is also important to note that both adults and school-age children show a robust MMN, despite developmental differences, which may be used to explain the different morphologies of the preceding P1-N1 waves (Courchesne, 1990; Kraus, McGee, Carrell, Sharma, & Nicol, 1993a). When comparing the difference in morphology of the MMN component in adults and children, no significant differences in peak latency, duration or onset-to-peak amplitude were observed, although the MMN of children was generally larger compared to adults (Csépe, 1995). In an experiment by Kraus, et al. (1995a), using just-perceptibly different variants of the phoneme /da/, MMN was obtained once the adults and children behaviorally noticed that the variant stimuli did not resemble the phoneme /da/.

The mismatch negativity response and processing of speech. The discrimination of acoustic features of the speech signal is a prerequisite to normal speech perception (Stevens & Blumstein, 1978; Walley & Carrell, 1983). Various acoustic features of the speech signal are encoded at distinctly different areas along the auditory pathway (Carney & Geisler, 1986; Mendelson & Cynader, 1985; Miller, Barta, & Sachs, 1987). As an example, the encoding of fundamental frequency (the primary frequency from which other harmonics of speech arise from) is first achieved at peripheral levels of the auditory system, including the auditory nerve (8th cerebral nerve) and brain stem (Carney & Geisler, 1986) but is not achieved at the auditory cortex (Phillips, 1993). Single

neurons in auditory cortex seem to specialize in the processing of frequency changes rather than the precise frequencies themselves, such as encountered during certain speech formant transitions (Mendelson & Cynader, 1985; Makela, Hari, & Linnakivi, 1987; Shamma & Vranic, 1993). The fact that various acoustic cues are processed at different loci along the auditory pathway is reflected in clinical studies of persons with auditory processing problems. Although these patients all have difficulties in processing speech, the perception of acoustic features between patients does not fit a single mold. The lack of uniformity between patients with such disorders has been observed using short-duration spectral changes, as in /da/-/ga/ (Elliott & Hammer, 1988; Phillips & Farmer, 1990; Rosen, Walliker, Brimacombe, & Edgerton, 1989; Turner & Robb, 1987) or when studying differences in the duration of spectral transition (Auerbach, Allard, Naeser, Alexander, & Albert, 1982; Rosen et al., 1989).

Because the MMN has its origins in the neocortex (as reflected by its superimposed appearance at the N1-P2 complex, which is generated by the auditory cortex), it taps into the neural encoding of acoustic properties at a central level (Csépe, Pantev, Hoke, Hampson, & Ross, 1992; Kraus, et al., 1994; Scherg, Vajsaar, & Picton, 1989). Thus, the MMN reflects neuronal activity as a function of changes in the acoustical features of speech. In recent years, MMN has been shown to be elicited by changes in acoustic features, such as frequency, intensity and duration within the speech signal (Aaltonen, et al., 1987; Alho, Sams, Paavilainen, Reinikainen, & Näätänen, 1989; Kraus, et al., 1993c; Näätänen, et al., 1997; Sams et al., 1985; Sharma, Kraus, McGee, Carrell &

Nicol, 1993). The MMN has the potential to objectively measure aspects of speech processing and is suitable in measuring discrimination of just-perceptibly different speech contrasts (Kraus, et al., 1993b; Näätänen, et al., 1997).

Most studies using the MMN for the study of speech processing have looked primarily at phoneme categorization. This refers to the way speech sounds along a continuum are categorized into two or more distinct categories despite equivalent physical distance between individual stimuli (e.g., /da/-/ga/).

Categorization is thought to be an underlying mechanism in phonetic processing and unique to speech (Liberman, Harris, Hoffman, & Griffith, 1957; Liberman, Cooper, Shankweiler & Studdert-Kennedy, 1967; Studdert-Kennedy & Shankweiler, 1970) while others believe that categorical perception is a general auditory perceptual process since it is also apparent during the perception of non-speech stimuli (Pastore, 1976; Schouten, 1980). The most widely accepted view on phoneme categorization is that the human auditory cortex may contain neural networks (i.e. top-down efferent systems) that are activated by speech specific acoustic features which would result in categorization of phonemes. While certain studies suggest that humans have an innate sensitivity to certain acoustic features (Stevens, 1981), others have demonstrated how "label-learning" is achieved by learning which acoustic features belong together in a phonemic class. This promotes the development of a phonemic detector, corresponding to a specific set of acoustic features, which facilitates the subsequent perception of that phoneme (Ades, 1977; Cutting, 1978; Studdert-Kennedy, 1993; Werker & Tees, 1992). The perceptual magnet effect also

demonstrates how repeated exposure to a particular set of acoustic features will cause the development of a perceptual prototype. Thus nonprototypical stimuli of the same phonemic category are perceived as more similar to the category prototype than to each other (Kuhl, 1991). Hence it is easier to discriminate phonemes belonging to two different categories than it is to discriminate two exemplars which belong to the same phoneme category, regardless of acoustic differences (Liberman et al., 1957). Thus, a more time-consuming reprocessing based extensively on acoustic information is required to discriminate exemplars which lie within the same category boundary on the phonemic continuum (Fujisaki & Kawashima, 1971; Pisoni & Tash, 1974). Top-down information, such as phonemic knowledge, reduces the cortical processing workload once a more defined expectation of the incoming signal is formed.

The MMN has been used to study speech processing because of its pre-attentive nature. Although many models of speech perception and spoken-word recognition emphasize the categorization process, the MMN reflects speech processing of speech which is not affected by top-down influences. By focusing the attention of the listener towards another task during the study of phoneme categorization, the experimenter can reduce the effect of cognitive top-down linguistic resources. The MMN therefore reflects the pre-perceptual processing of speech. The MMN has been observed for stimulus pairs of equal acoustic difference from both within and across phonemic category boundaries. Thus, the MMN would appear to reflect the processing of acoustic aspects of the speech stimulus and not phonetic processing into categories (Kraus, McGee, Sharma,

Carrell, & Nicol, 1992; Sharma et al., 1993). Based on this research, the MMN would appear to reflect a response to speech discrimination and not speech identification. Furthermore, these experiments may imply that acoustic processing of phonemes occurs independently of categorical processing.

Based on these facts, it would appear that speakers of various languages would be able to pre-perceptually process English phonemes in a similar fashion, based strictly on its acoustic features, and presumably obtain similar MMN responses. However, cross-language and training studies have shown that MMN responses may in fact be affected by top-down neural networks for speech and thus may reflect categorical processing of phonemes. Näätänen, et al. (1997) have shown that the MMN is capable of being enhanced using native prototypes as deviant stimuli. This study was able to show that using an Estonian prototype as a deviant stimulus and a non-prototype common to both Estonian and Finnish speakers as the standard stimulus, the MMN was enhanced only in Estonian speakers when compared to using another non-prototype as the deviant stimulus. Näätänen and colleagues concluded that neural phoneme traces can be activated during categorical (phonemic) processing and consequently affect the MMN response. Training studies have shown that a MMN that was once absent because listeners could not discriminate between phonemes, was then observed after the listener was trained to discriminate between phonemes. It therefore appears that MMN could be obtained once a neural trace of the foreign phoneme was formed (Kraus et al., 1995b). Therefore, MMN may not strictly reflect precategorical, purely acoustic processing but may also reflect adaptive

changes in the perceptual system. A recent study suggests that the pre-attentive discrimination process of the MMN reflects both auditory (sensory) and phonetic (categorical) factors of speech items (i.e., vowels) (Winkler, et al., 1999). Thus, it may not be completely pre-categorical or pre-perceptual as originally thought.

Although phoneme continuum studies also imply certain discrimination capabilities, clinicians are also concerned with the discrimination of lexically relevant speech items such as words, since efficient communication strategically uses words, sentences and even larger speech segments in order to verbalize an intent. It is unclear at this point whether the MMN reflects strictly acoustic processing of words or whether top-down cognitive and lexical influences will modulate the response.

Neuroanatomic pathways for encoding acoustic speech contrasts. The study of patients with lobe-specific cortical damage has revealed that the left auditory cortex typically deals with the processing and discrimination of complex acoustic features such as speech (Fitch, Brown, O'Conner, & Tallal, 1993; Zattore, Evans, Meyer, & Gjede, 1992). Some MMN studies have shown symmetrical hemispheric responses to speech stimuli in adults (Aaltonen, et al., 1994; Tremblay, Kraus, Carrell, & McGee, 1997) and in school-aged children (Kraus, Koch, McGee, & Nichol., 1999). Others have shown asymmetrically larger MMNs to stop consonants over the right hemisphere (Csépe, 1995), while larger MMNs were obtained over the left hemisphere for vowels (Csépe, 1995) and syllables (Alho, et al., 1998). Non-native training studies also provide evidence towards hemispheric dominance during speech processing. Tremblay

et al. (1997) were able to show symmetrical MMN responses to non-native speech contrasts that became enhanced over the left hemisphere with training. This suggests that the left hemisphere is somewhat more involved in the processing of the native language (Näätänen, et al., 1997).

Magnetoencephalography studies have shown the equivalent MMNm response to be predominant over the left hemisphere during speech processing. However, MEG studies have shown that with masking of speech items, there is a clear shift of activity from the left to the right hemisphere during speech processing (Shtyrov, Kujala, Ahveninen & Näätänen, 1999). This hypothesis had previously been suggested when inactivation of the right hemisphere during electroconvulsive therapy caused a reduction of speech discrimination in noise, while inactivation of the left hemisphere had no significant effect (Balanov & Deglin, 1970).

The MMN can also be used to demonstrate processing features of localized regions of the auditory system using patients with confirmed auditory lesions. Aaltonen, Tuomainen, Laine, and Niemi (1993) showed that aphasics with posterior temporal lobe lesions had MMNs to pure tones but not to a short duration (42 ms) vowel contrast. Csépe (1995) found that Wernicke's aphasics had perceptual impairment and absent MMN to consonant contrasts, whereas Broca's aphasics did not. MMNs to vowels and tonal contrasts were intact.

Topographic studies suggest that MMN responses elicited by different acoustic features arise from different generators within the auditory system (Giard, Lavikainen, Reinikainen, Perrin, & Näätänen, 1995; Levänen, Hari,

McEvoy, & Sams, 1993). Electric and magnetic MMN data that show systematic differences between MMN fields elicited by frequency, intensity, and duration differences, providing evidence for multiple MMN sources. For example, Kraus et al. (1993b) describe a patient with a primary complaint of difficulty hearing in noise who showed an abnormal ABR, suggesting a disruption of central auditory processing. They found that this patient had much difficulty in discriminating speech contrasts differing spectrally in formant transitions (as seen between /da/ and /ga/) but showed no difficulties in discriminating speech contrasts differing temporally in formant transition duration (as seen between /ba/ and /wa/). The selective impairment was evident in both behavioral testing and during MMN recordings. Thus, it can be concluded that different fine acoustic attributes are processed and encoded in different regions of the auditory system, as reflected by the different MMN generation sites (Kraus et al., 1993b).

As with acoustic features, speech features are also processed distinctively by specific brain regions and often show hemispheric specialization (Auerbach et al., 1982; Phillips & Farmer, 1990; Zattore et al., 1992). Segmental (consonants and vowels) and suprasegmental (prosodic) aspects of speech may also be encoded at different points along different cortical pathways (Phillips & Farmer, 1990; Phillips & Hall, 1990), by identifying the corresponding MMNm. The use of MMNm, which maps and identifies the cortical topographical source of MMN generation may have implications for the diagnosis and rehabilitation of CAPD.

The suitability of the mismatch negativity response to assess central auditory dysfunction. The duration of auditory stimuli should be kept rather short

in order to elicit MMN with large amplitudes (i.e., in the range of 50 ms) (Paavilainen, Jiang, Lavikainen, & Näätänen, 1993). However, some exceptions have been noted. Winkler, Tervaniemi, and Näätänen (1997) showed that MMN to changes in fundamental frequency were only present in stimuli with a stimulus duration of over 150 ms and another study demonstrated that MMN to changes in stimulus intensity increased as a function of stimulus duration from 4 to 300 ms (Paavilainen et al., 1993). Furthermore, since phonemes (i.e., stimulus duration of 400 ms) have been shown to elicit MMN (Martin, Kurtzberg, & Stapells, 1999), other speech stimuli such as filtered speech (Bocca et al., 1954; Bocca et al., 1955), compressed speech (Bocca et al., 1958; Calero & Lazzaroni, 1957), speech in noise (Morales-Garcia & Poole, 1972) and interrupted speech (Jerger & Jerger, 1974), could also be used within the MMN methodology provided that the duration of these stimuli is kept under 500 ms. English phonemes and one or two syllable words are often less than 500 ms and thus would not seem to conflict with the limitations of the MMN paradigm.

MMN can be elicited to both synthetic (Aaltonen et al., 1987; Kraus, 1992, 1993a, 1993b, 1993c, 1993d; Sams et al., 1990; Sharma et al., 1993) and natural speech (Muller-Gass, Marcoux, Logan & Campbell, 2001; Sandridge & Boothroyd, 1996). While synthetic speech offers a considerable amount of analytical control over stimulus parameters, natural speech is a more valid stimulus because it contains all the subtle acoustic cues that listeners use in their daily interactions with others. However, the uncontrolled variations within natural speech are likely to cause a measurable amount of unexplained

variability during MMN analysis (Sandridge & Boothroyd, 1996). Nevertheless, since CAPD is also a realistic manifestation, its assessment is more ecologically relevant when natural speech tokens are used.

The application of MMN towards the assessment of CAPD using a speech-in-noise paradigm

Overview of present investigation. Careful selection of an auditory stimulus must be done to investigate CAPD using the MMN. Although the MMN itself has not shown limitations as to what the physical attributes of the auditory stimulus should be, the MMN methodology does limit the choice of auditory stimuli. Recall that the MMN is generated once a deviant stimuli is discriminated from a standard template formed in memory. Thus, it is imperative for the task to reflect the discrimination of one infrequent stimulus from a repetitive frequent stimulus. The behavioral CAPD tests of binaural interaction, lateralization, localization, integration or separation are not suitable for the MMN methodology since they require the simultaneous presentation of different auditory stimuli and do not depend entirely on the discrimination process. However, other behavioral CAPD tests, such as the Compressed Speech Test, the Filtered Speech Test and the Speech-in-Noise Test, are in essence discrimination tasks, which would fit the requirements of the MMN methodology rather well. Since the MMN has not been used extensively in the assessment of CAPD, it is imperative to explore its possibilities with caution by transposing the simplest of behavioral tests to this electrophysiological paradigm. Although these behavioral low-redundancy

speech tests do not have the highest sensitivity for the CAPD diagnosis, they possess simple methodologies that are adequate for this type of exploratory study.

In the present study the redundancy of these natural speech tokens was reduced, following the rationale of low-redundancy behavioral speech tests of CAPD, using speech noise in order to degrade, or reduce the extrinsic redundancy of speech items. Although behavioral tests of Speech-in-Noise typically do not have a high hit-rate for CAPD, they are nonetheless easily transposed to the MMN paradigm. Furthermore, methodologies similar to those of Speech-in-Noise tests have already been the object of MMN studies.

Research using noise as part of the deviant stimulus has reported the occurrence of a MMN to the deviant constructed by the addition of noise to the standard simple tone (Nordby, Hammerborg, Roth & Hugdahl, 1997) or a segment of the standard complex tone (Marcoux, Muller-Gass, Logan, & Campbell, 1999). Other studies have used continuous masking noise in order to explore the effects of reduced extrinsic redundancy on the entire length of the speech stimuli. Martin, Kurtzberg and Stapells (1999) were able to show, using the MMN procedure, a reduction in discrimination of English phonemes when ipsilateral white noise was used as a masking agent. Salo, Lang, and Salmivalli (1994) used contralateral speech noise and found a reduction in MMN amplitude based on central masking on speech discrimination. Collectively, these studies suggest that noise will also hinder the discrimination of other speech items, such as words, depending on whether these items can be used within the MMN paradigm. Such a methodology

would make possible an objective electrophysiological procedure for evaluating the processing of speech items and observing the effects of a reduction in extrinsic redundancy of the speech items.

Hypotheses. The present investigation used a procedure similar to Martin et al. (1999) in order to study the effects of degradation of the speech items on discrimination performance in both a normal, control group and an experimental CAPD group (i.e., reduced intrinsic redundancy of the auditory system). As discussed previously, a reduction in both intrinsic and extrinsic redundancy are necessary to obtain a decrease in performance during low-redundancy speech tests, such as the Speech-in-Noise test.

The combination of MMN and a speech-in-noise manipulation raised several questions. Because the degradation of speech items reduces their extrinsic redundancy of the speech items, will this degradation affect the discrimination of speech items in a similar fashion in normal patients and CAPD patients, who possess a reduced intrinsic redundancy of the auditory system? Can such a procedure be used as a probe for the assessment of CAPD? The results obtained during the MMN task were compared to a behavioral CAPD test of Speech-in-Noise in order to determine whether the MMN could objectively measure the reduction in word discrimination in increasingly noisy conditions.

METHOD

Participants

Seven patients with CAPD (age 25-43 years, 2 females) and seven age-

matched controls (age 20–43, 3 females) were chosen to participate in this study. The SCAN-A (Screening Test for Auditory Disorders-Adults) was administered to all volunteers prior to subject selection. The SCAN-A is composed of the following subtests: Filtered words, Auditory figure-ground (speech-in-noise), Competing words, and Competing sentences. The SCAN-A can rapidly be used to describe auditory processing disorders (Keith, 1994) and the results compared to established norms in order to quantify the level of deficit one may possess. Inclusion criteria for patients consisted of: a) previous diagnosis of CAPD by a registered audiologist; b) low ($<85/130$) SCAN-A total test score; c) low ($<7/16$) Auditory figure-ground subtest score; and d) normal hearing sensitivity (25dB ISO or less) for pure tones ranging from 250 to 4000 Hz. Control participants included in this study obtained SCAN-A total test scores that were considered normal ($>100/130$), and had normal hearing sensitivity (20 dB ISO or less) for pure tones ranging from 250 to 2000 Hz. SCAN-A scores for both groups appear in Table 1. Pure-tone audiometric thresholds for both groups appear in Table 2. Participants acknowledged that the experiment carried no risk or harm by signing a consent form which had been previously approved by the appropriate ethics committees.

Insert Figure 1 here

Insert Tables 1 and 2 here

Stimuli

The syllables /ba/ and /da/ were selected because of their use in previous research. These syllables differ in place of articulation of their initial phonemes and thus differ in the frequency domain. The stimuli were created by digitizing natural tokens produced by a male talker. The tokens were then processed using linear prediction coefficient (LPC) analysis, and synthesized tokens regenerated using the LPC polynomial roots (Maiste, Wiens, Hunt, Scherg, & Picton, 1995). The tokens were digitized using the Signalize program using a 22 kHz sampling rate (11 kHz anti-aliasing filter, 12 dB/octave), edited to 150 ms duration by eliminating the final portion of the steady-state vowel and tapering the offset of the vowel, and calibrated in peak-to-peak equivalent SPL (dB ppe SPL) relative to a 1000 Hz pure tone.

The frequencies for the steady-state portion of the vowel /a/ ($F_0=110$ Hz, $F_1=700$ Hz, $F_2=1170$ Hz, $F_3=2380$ Hz) and formant transitions were determined by spectrographic analysis. The transitions for /ba/ began at 362 Hz for F_1 , 1080 Hz for F_2 , and 2320 Hz for F_3 ; the transitions for /da/ began at 376 Hz for F_1 , 1620 Hz for F_2 , and 2480 Hz for F_3 . Figure 1 provides graphic and spectrographic representations of the speech items.

Insert Figure 1 here

Peak intensity of the speech sounds /ba/ and /da/ was 65 dB SPL. These stimuli were presented in a quiet condition (NoNoise) and in continuous ipsilateral background speech noise that varied in intensity. The intensity of the masking noise was either 65 (Noise65dB), 70 (Noise70dB), or 75 dB SPL

(Noise75dB) in different conditions. The masking noise was produced by an audiometer (Interacoustics AC33). The intensity of the noise was confirmed using a sound level meter (Brüel & Kjaer).

Procedure

During the EEG recording session, participants were seated in a sound attenuated room and instructed to read a self-selected book. Participants were asked to minimize eye and head movements. Both speech sounds and noise were presented to the right ear via an insert earphone (EAR-3A). The stimuli /ba/ and /da/ were delivered using a digital-to-analog (D/A) rate of 16 kHz. The quiet condition was presented twice to each listener, once with /ba/ and once with /da/ serving as deviant. Pilot data indicated no significant difference in the MMN between these deviants in both participant groups. Therefore, /ba/ was selected as the standard and /da/ as the deviant for all noise conditions. Each condition consisted of two blocks of 600 stimuli: 510 standards ($p=0.85$) and 90 deviants ($p=0.15$). Each condition was replicated, yielding a total of 1200 trials per condition. The ten blocks were presented in random order. A stimulus was presented every 1100 ms. The order of the presentation of stimuli was pseudo-randomized, with the provision that two deviants could not occur successively and that the first two stimuli could not be deviants. The entire duration of the procedure did not exceed 2 hours. Following EEG recording, the listener was instructed to attend to the stimuli and signal the occurrence of the deviant by pressing a computer key. During this behavioral task, 200 stimuli (160 standards,

40 deviants) were presented per condition.

EEG recording

EEG activity was recorded from 5 channels (Fz, Cz, Pz, CP3, CP4) according to the International 10-20 system using an Electro-Cap International Inc. custom electrode cap. Two additional channels were recorded from individual tin cup electrodes placed on the left and right mastoids (LM, RM). The nose served as a reference (Alho, Paavilainen, Reinikainen, Sams & Näätänen, 1986). A vertical EOG was recorded from electrodes placed at the infra and supra-orbital ridges of the right eye. A horizontal EOG was recorded from electrodes placed at the outer canthus of each eye. The ground electrode was attached to the forehead. Inter-electrode impedances were below 5 kOhms. Data was acquired continuously (586 Hz sampling rate), with a high filter set at 30 Hz and a time constant of 2 s. EOG artifact was corrected using an algorithm operating in the frequency domain. The EEG signals were later reconstructed into discrete epochs beginning 100 ms prior to stimulus onset and continuing for the 800 ms following it. ERPs were digitally low-pass filtered using an inverse FFT algorithm with a 12 Hz cutoff frequency.

Data measurement and statistical analysis

Single-trial responses were averaged separately for standard and deviant stimuli. Replications were averaged together for each condition. Difference waves were obtained by subtracting, point-by-point, the standard from the deviant waveforms at the Fz site in the same condition. MMN peak amplitude

was scored in a time window from 80 to 250 ms, for each control participant, and from 80 to 400 ms, for each patient. These time windows were determined from the grand-averaged waveforms. Determination of response presence/absence required the agreement of two experienced judges. The presence of the MMN was measured, in the difference waveform, under the following assumptions: a) the MMN was required to have a greater amplitude at more frontal sites as opposed to more posterior sites, b) the inversion of the MMN at both mastoid sites, and c) the MMN occurred within the reference window. The MMN latency was measured as the point of greatest amplitude on a single peak within the response window which corresponded to the mastoid inversion. Visual inspection of the MMN was used to determine response validity although other methods of validation may have resulted in increased detection rates (McGee, Kraus & Nicol, 1997). Two-way repeated measures ANOVAs (Noise condition, Participant group) were carried out on the MMN amplitude and latency data at Fz, as well as on the behavioral discrimination scores. Furthermore, one-way (Speech stimulus) repeated measures ANOVAs were performed for each group in the quiet conditions to compare differences in the MMN at Fz when /ba/ and /da/ served as deviants. Differences were considered to be significant at $p < 0.05$. Where appropriate, Hyndt-Feldt correction factors were applied to the degrees of freedom and adjusted probabilities reported. Neuman-Keuls post-hoc comparisons were performed to examine significant effects. Correlational analyses between behavioral discrimination scores and MMN measurements were considered to be significant when $p < 0.01$.

RESULTS

Deviant stimulus

Both /ba/ and /da/ were used as the deviant stimulus in separate conditions with no masking noise. The phoneme used as a deviant had no significant effect on MMN peak amplitude ($F(1,6)=1.66$, $p>0.25$; $F(1,6)=2.51$, $p>0.17$) and latency ($F<1$; $F(1,6)=1.60$, $p>0.70$) for either controls or patients, respectively. Standard and deviant waveforms at Fz, Cz, Pz and LM for all noise conditions in which /da/ served as a deviant are presented in Figure 2, for control participants, and Figure 3 for patients. These figures illustrate the effect of level of noise on MMN amplitude and latency at various electrode sites. Grand-averaged difference waveforms at Fz are superimposed for controls and patients in Figure 4. This figure illustrates the reduction in MMN amplitude across all noise conditions for patients in comparison to control participants. Furthermore, a relatively similar MMN latency is observed for both groups in the No Noise condition. However, a more marked increase in MMN latency is observed for patients in subsequent noise conditions in comparison to control participants.

Insert Figures 2, 3, and 4 here

MMN peak amplitude

A significant interaction was found between participant group and noise condition for MMN peak amplitude, $F(3,36)=5.65$, $p<0.01$, $\epsilon=1.00$. Simple effects

testing indicated a significant MMN peak amplitude difference between groups at the NoNoise and Noise65dB conditions. In these conditions, patients displayed significantly reduced MMN amplitude as compared to control participants. Due to heterogeneity of within-group variances, a one-way repeated measures ANOVA was conducted on each of the groups. A significant effect of noise condition was found for the control group only, $F(3,18)=18.70$, $p<0.01$, $\epsilon=0.66$. Planned comparisons indicated that as the level of noise was increased, the MMN peak amplitude of control subjects decreased steadily. MMN mean amplitudes of controls and patients as a function of noise intensity are displayed in Figure 5 (left panel).

Insert figure 5 here

MMN peak latency

A significant interaction was also found between participant group and noise condition for MMN latency, $F(3,36)=5.95$, $p<.01$, $\epsilon=1.00$. Simple effects testing indicated that in the NoNoise condition, controls and patients displayed similar MMN latencies. However, in all conditions in which noise was added, patients had significantly longer MMN latencies as compared to control participants. Due to heterogeneity of within-group variances, a one-way repeated measures ANOVA was conducted on each of the groups. For both controls, $F(3,18)=12.12$, $p<0.01$, $\epsilon=0.40$, and patients, $F(3,18)=24.24$, $p<0.01$, $\epsilon=1.00$, a significant effect of noise condition was revealed. Planned comparisons indicated that as the level of noise was increased, MMN peak latency of both groups was

delayed. MMN mean latencies of controls and patients as a function of noise intensity are displayed in Figure 5 (right panel).

Behavioral data

The interaction between participant group and noise condition for hit-rate was significant, $F(3,36)=3.34$, $p<0.05$, $\epsilon=0.74$. Simple effects testing revealed that in the NoNoise condition, controls and patients obtained similar hit-rates. However, in all conditions in which noise was added, patients had significantly lower hit-rates as compared to control participants. While patients' hit-rates significantly decreased from the NoNoise to the Noise65dB condition, controls' hit-rates did not. Both groups showed significant reductions in hit-rate as noise was further increased. Mean percentage of hits and false positives for both groups are shown in Table 3.

Insert Table 3 here

Correlations between MMN and behavioral measures

The relationships between the MMN and behavioral discrimination scores were examined using Pearson product-moment correlations. For the control group discrimination scores correlated well with both the MMN peak amplitude index (.47) and the MMN peak latency index (-.42). However, discrimination scores obtained for the patient group only correlated well with the MMN peak latency index (-.67) and not with the MMN peak amplitude index (-.10).

Attentional variables

Attention was controlled during the experiment in both groups by diverting the attentional focus of each participant towards a reading task. Attentional focus was monitored and confirmed with on-line monitoring of ocular movement. Furthermore, visual inspection of individual data across all conditions did not reveal a P2b-P3a component which follows and/or overlaps the MMN when the participant's attention is either voluntarily focussed towards the auditory stimulus or involuntarily shifted towards a salient, obtrusive auditory stimulus which is difficult to ignore (Marcoux et al., 1999). Attention was therefore adequately controlled during the experiment.

DISCUSSION

Summary of results

The results of the present investigation evaluated whether the choice of deviant stimulus among the chosen syllable pair /ba/ and /da/ had a significant effect on MMN measurements since standard and deviant stimuli are usually interchanged in order to eliminate order effects. Martin et al. (1999) examined the effects of masking noise on MMN peak amplitude and peak latency. They presented 28 conditions consisting of 1200 stimulus presentations which had a duration of 1100 ms each. The length of the testing session would thus be at least 10 hours per participant, much too long from a clinical perspective. In contrast, the length of the testing session was minimized in the present investigation, especially important for CAPD patients who had a low distraction threshold. Pilot testing revealed that the choice of deviant stimulus had no effect on MMN peak amplitude or MMN peak latency. Furthermore, there was no difference in MMN between patients and control participants, indicating that both /ba/ and /da/ are discriminated equally well when used as the deviant within the oddball paradigm. Therefore, the /da/ stimulus was used as the sole deviant in all conditions. This manipulation permitted the total testing time, including set-up and behavioral measures, to remain under 3 hours.

The reduction in audibility following an increase in the masking noise level resulted in a reduction of behavioral discrimination scores and a change in MMN

morphology. This result is consistent with previous results obtained by Martin et al. (1999) where a reduction in audibility resulted in a reduction in MMN amplitude and an increase in MMN latency.

Fit between data and theory

Behavioral trends were the same for both groups: as noise increased, hits decreased. Performance was also similar for both groups in the quiet condition. However, patients showed overall lower discrimination scores in the noise conditions. They also displayed a sharper decrease in hits from the NoNoise to the Noise65dB condition. These results are in accord with present knowledge of CAPD (Chermak & Musiek, 1997). Patients and controls discriminate speech in a similar manner in a quiet condition, but patients will show a marked decrease in performance as the level of noise is increased. For the behavioral discrimination task used in this experiment, hits were always above 90% and false positives were below 1%, regardless of the condition and group. Although significant differences were observed between groups, most behavioral tests of CAPD provide a larger spread of results between normal and abnormal scores. Clearly, the present task is not standardized but it is important to note that the simplicity of the task may not provide the necessary complexity to explore central auditory deficits.

MMN latency effects were similar to the behavioral data. Both groups showed a prolonged latency as intensity of the masking noise increased. However, while patients had longer latencies than controls across noise

conditions, no significant difference in latency between groups was observed in the quiet condition. These findings are also in accord with present knowledge of CAPD and behavioral performance, where both patients and controls have similar results in quiet conditions, but with patients showing more marked changes than controls in noisy conditions. This physiological effect may be explained by means of a neurophysiological model, designed to account for the reduction of intrinsic redundancy in CAPD (Starr, et al., 1991). The assumption behind this model is that central auditory processes, such as auditory discrimination and performance with competing signals, amongst others, are responsible for generating the auditory evoked potentials (Chermak, Hall, Baran, Sloan & Musiek, 1997). However, in CAPD the neuronal signal which, because of abnormalities along the auditory pathways, possesses a distorted representation of the original acoustic signal. What results is an irregular timing of events leading to a distortion in neural coding (ASHA, 1996; Starr, et al., 1991). Thus, the model explains the "bottom-up" component of CAPD. However, it has been demonstrated that children with language disorders could have similar "bottom-generated", sensory evoked potentials as controls (e.g., ABR and MLR) but show significantly different cortical potentials (Korpilahti & Lang, 1994). This finding offers evidence that cortical potentials, such as the MMN, are not solely a result of the previously altered "bottom-up" potentials which rely highly upon neural synchrony, but may also reflect "top-down" or central processes. The incoming auditory stimuli involve the deployment of top-down mechanisms, such as learning and memory, during speech processing (ASHA, 1996). The incoming

bottom-up signal is therefore compared to the listener's experience and expectation and provided with corrections and constraints in order to permit appropriate perception and recognition (McClelland & Elman, 1986). Therefore, although the temporal encoding distortion remains the most widely accepted explanation for central auditory disorders, research points beyond this explanation to a deficit in cortical pre-conscious processing (Leppänen & Lyytinen, 1997). Of course, a combination of these explanations is also possible.

A degradation of the acoustic speech signal increases the demand for top-down processes in order to achieve the same level of perception (Marslen-Wilson & Tyler, 1980). In so doing, the perception of a degraded acoustic signal is often possible depending on the amount of contextual support. The most probable explanation in light of the poor performance of the patient group in the present study is that masking noise may have caused an enhancement of the temporal encoding disruption. In the presence of noise, speech perception relies greatly on temporal encoding (Miller et al., 1987).

The present results also indicate a continuous decrease in MMN peak amplitude in the control group as the level of noise is increased. The MMN peak amplitude from patients did not change as a result of noise masking. It would appear that while there is a steady decrease in MMN peak amplitude in the control group, the patient group reveals a decrease in amplitude only from the NoNoise to the Noise65dB condition, followed by a highly variable increase in MMN peak amplitude in subsequent conditions. When isolating the two first conditions, a significant difference between groups can be visualized. It appears

that although the level of noise does cause a decrease in MMN amplitude in both groups, the patient group appears to reveal a generally more marked decrease in amplitude when compared to the control group.

Differences in MMN amplitudes between the control and patient populations are difficult to explain using the previously discussed neurophysiological model of temporal distortion. It is also accepted that late-latency auditory potentials such as the MMN are independent of neural synchrony, which may be disrupted in CAPD and may cause a reduction in amplitude of earlier potentials. Previous studies have not reported decreased MMN amplitudes using speech stimuli in CAPD (Kraus, et al., 1993b). One possible explanation could be the result of a fading memory trace in CAPD. Näätänen (1995) has postulated that a strong comparison process between the memory trace of the standard stimulus and the novel deviant stimulus will sharpen the MMN morphology. Nevertheless, it appears that the MMN latency index may be most suitable in identifying and assessing CAPD patients based on its theoretical foundations and its correlation with behavioral scores of both control participants and patients.

Hyde (1997) suggests that the analysis of earlier auditory evoked potentials may be useful during the interpretation of later potentials such as the MMN. In order to explore the decrease in MMN peak amplitude in the CAPD population, the N1 potential, which is generated prior to the MMN was examined and analyzed post-hoc. The N1, like the MMN, is a late auditory evoked potential. The N1 represents an early detection stage of the auditory process, which also

involves attention, memory, classification and recognition. The MMN is indicative of stimulus change. Thus, during the oddball paradigm, an N1 is obtained to both the standard /ba/ stimulus and the deviant /da/ stimulus. Our investigation, as seen in Figure 6, revealed an N1 component reduced in amplitude, in all conditions, in the patient group in contrast to the control group. However, no significant latency differences were observed between groups.

Insert figure 6 here

Previous research has documented amplitude and latency characteristics of N1 in patient populations. Patients with lesions of the superior temporal gyrus showed significantly reduced N1 components, as compared to a control group. Furthermore, significant latency differences were not observed (Knight, Scabini, Woods, & Neville, 1988). Similar results were noted in children with auditory processing difficulties (Pinkerton, Watson, & McClelland, 1989). Reduced N1 amplitude patterns were also recorded in adults with CAPD (Schönweiler, Wübbelt, Tolloczko, Rose, & Ptak, 2000). Other studies have shown a reduction in N1 amplitude accompanied by an increase in N1 latency in children with language impairment (Tonquist-Uhlen, Borg, Persson, & Spens, 1996) and in children with CAPD (Jirsa & Clontz, 1990). It is important to note that these studies utilized pure tones or tone bursts rather than speech sounds to evoke the N1. The N1 component has also been shown to be robust to noise masking (Martin, Sigal, Kurtzberg & Stapells, 1997). Thus, it appears plausible to expect a reduction in N1 amplitude in auditory processing disorders while latency characteristics remain similar or become somewhat prolonged compared to that

of controls. Furthermore, the reduction in amplitude of the N1 component in the patient population does not appear to be affected by the level of noise but rather by the underlying pathology. The ramifications of the present post-hoc analysis on N1 latency and amplitude lead to a different, more cautious interpretation of our MMN results. In the present study, it is mostly the reduction in MMN peak amplitude results that reflect the N1 detection potential. The increase in MMN latency index should be interpreted as having a more central origin, since an increase in N1 latency was not observed in the present study and have not always been noted in CAPD and other related pathologies.

The present results indicate that CAPD patients may have: a) a “bottom-up” component which is reflected in the reduction of N1 amplitude, and subsequently MMN amplitude; and b) a “top-down” or more central component which is reflected by an increase in MMN latency which cannot be attributed to an increase in the previous N1 potential. In light of these findings, an explanation could be based on the heterogeneity of CAPD patients. Although all patients were diagnosed with CAPD and all reported difficulty in noisy environments, the comorbidity of other pathologies, such as language impairments, developmental language-related disorders, was not controlled when selecting participants for the patient group. This explanation is unlikely upon examination of individual data, which clearly resemble the group data. A second, more ambitious explanation is that CAPD may manifest itself not only with a distortion of temporal neural encoding but also with difficulties in short-term memory and perceptual processing. The latency results of the present study indicated that while there

were no significant differences in N1 between controls and patients, there was a marked difference in MMN between groups. The following results would therefore suggest a disorder affecting the discrimination stage that was not apparent at the stage of stimulus detection. As mentioned previously, a fading memory trace in CAPD may be responsible. However, this explanation is unlikely since a reduction in short-term memory required for MMN should theoretically lead to a reduction in MMN amplitude which would not be explained by a reduction in N1. Since the MMN may reflect perceptual processes that are not consciously apparent (Kraus & Cheour, 2000), it is possible that pre-perceptual processing is affected in CAPD. For example, Winkler et al. (2000) demonstrated evidence for pre-perceptual categorical processing. According to this study, between-category discrimination permits the use of phonetic categorical information which elicits a more intense MMN response. As phonetic information is removed, such as would be the case in the present study where speech sounds are degraded, discrimination is achieved based on acoustic information. This type of discrimination elicits a less intense MMN response. Therefore, CAPD patients, because of continuous temporal distortions of speech sounds, may not possess well-defined categorical prototypes which could potentially facilitate discrimination. Although little is known about pre-conscious auditory processes, the MMN, in combination with an N1 analysis, may provide valuable information concerning auditory "cognition" when no conscious attention is directed towards the auditory task (Leppänen & Lyytinen, 1997).

Directions for future research

The methodology used in the present investigation was successful in differentiating control participants from CAPD patients. Although, when looking at the inventory of behavioral CAPD tests, a discrimination task between two syllables appears rather simple, it is important to note that an electrophysiological paradigm, such as the MMN, imposes serious restrictions regarding the choice of auditory stimuli. Nevertheless, it appears that this rather “simple” task is sufficient for probing central auditory dysfunction.

Patients who participated in the present study were all adults with idiopathic CAPD. The main concern that originally led them to consult with an audiologist was their reduced ability to understand in a noisy setting. However, most cases of CAPD are detected during the first academic years by a parent or teacher, who would have detected a learning problem or what would appear to be a lack of attention during verbal communication. Thus, it is rather rare that adults are first given the diagnosis of CAPD during adulthood, unless diagnosed with a developmental or acquired form of CAPD. An important question is whether a methodology, such as developed in the present study would be a help towards the assessment of pediatric CAPD. Theoretically, the method would be adequate although children would have more difficulty being cooperative during the long, uninterrupted testing session. Thus, it may be difficult to assess very young children who are unwilling to remain calm during the installation of the electrode array and the long testing session. Furthermore, because of its small amplitude and large variability, the MMN, although quite

identifiable in group data, is difficult to identify in individual data. Nevertheless, much research is needed with the pediatric population since the sequelae of CAPD commence during the important years of speech and language development. Therefore, it is important for future research to consider these restrictions which are inherent in the collection of long-latency evoked potentials.

As enumerated in the introduction of this dissertation, a multitude of behavioral CAPD tests are available in order to probe various aspects of central auditory processing. Future research may also wish to address whether evoked potentials are sufficient in order to explore these various aspects.

The results of the present study indicated that MMN is an important part of the electrophysiological assessment of CAPD. As discussed in the introduction, MMN is a potential probe into CAPD because it reflects passive sensory processing in addition to reflecting stimulus change. It is therefore a very useful neurophysiological tool since stimulus change is what undoubtedly characterizes the acoustics of human speech. However, in designing the study I did not anticipate having to gather more caudal evoked potentials such as the N1. Fortunately, the N1 could be analyzed post-hoc. The present results suggest that processes reflected by N1, which precede sensory memory processing (reflected in MMN), be added to electrophysiological studies of CAPD in order to interpret higher levels of processing.

Although the relationship between an abnormal N1 potential and auditory processing deficit is possible, the N1 cannot be regarded as an indicator of pre-conscious auditory cognition (Leppänen & Lyytinen, 1997). Thus, future

electrophysiological studies of CAPD should evaluate to sub-cortical and cortical potentials. Furthermore, although difficult, homogeneous groups of patients should be utilized in order to determine objective indices of CAPD. In the present study, most patients were given an idiopathic diagnosis of CAPD, thus making this group rather heterogeneous. Nevertheless, all reported similar difficulties and individual electrophysiological data resembled the group data. While I did not completely anticipate the present results, they warrant the collection of additional data in order to determine whether CAPD affects pre-conscious, cognitive processing in conjunction with lower distortion of neural encoding.

Future investigations of MMN and CAPD should also record the P300, a more latent, electrophysiological waveform. Unlike the MMN, the P300 can be elicited using visual and tactile stimuli. Therefore, the absence of P300 during an auditory task would confirm the response from the auditory modality.

Furthermore, a P300 would confirm that the central auditory processing functional deficits of the patient population are indeed linked to CAPD and not to a comorbid disorder which affects non-dedicated processes also found in CAPD. However, the advantage of using the MMN paradigm is the ability of assessing auditory processing without the contribution of cognitive confounds. Nevertheless, P300 should be recorded in conjunction with MMN if attention cannot be sufficiently withdrawn from the auditory task.

Future research should also explore the use of additional signal-to-noise ratios in order to determine a level of masking noise which could be used in isolation. In determining such a level, the MMN paradigm used in the present

investigation could have a clinical utility. However, at the present time, this type of methodology is much too long to be used in any realistic fashion in the clinical setting. Furthermore, the present study limited itself to reaching signal-to-noise ratios of no less than -10. Although this level of masking permitted a separation of the control and patient groups when examining MMN latency and behavioral scores, it is my opinion that lower signal-to-noise ratios could be used in order to optimize the separation between groups.

Conclusion

In sum, it is plausible to make the following assumptions: a) the distortion of neural encoding is present in CAPD patients as reflected by the N1 amplitude index; b) as the audibility of speech sounds is reduced, CAPD patients show a more marked decrease in MMN amplitude and a more marked increase in MMN latency, which indicates that as the temporal distortion of speech items is increased, temporal distortion of neural encoding is also increased; and c) CAPD patients may demonstrate a pre-conscious auditory processing deficit as reflected by the difference between the N1 and MMN latency indices. Thus, CAPD patients may possess fewer pre-conscious cognitive resources, such as categorical processing, in comparison to controls.

The greatest challenge of adapting such a methodology towards the assessment of CAPD lies with the methodology itself. Late-evoked potentials, such as the MMN are rather small and variable and thus, are difficult to identify in individual data. Nevertheless, the MMN latency index appears to correlate rather

well with active discrimination scores in both the control and patient groups. The MMN amplitude index is perhaps affected by levels of auditory processing which precede passive discrimination.

This study was a first attempt towards an adaptation of a central auditory processing task to an electrophysiological paradigm. Although further research is required in order to verify these results, data from the present investigation should convince the reader of the potential of the MMN measure as an objective assessment of CAPD. Furthermore, electrophysiological studies may shed some light regarding the underlying neurological bases of central auditory dysfunction.

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Tables

Table 1

Participant	Filtered Words	Figure-Ground	Competing Words	Competing Sentences	Total Test Score
C1	12*	12*	14*	9*	111*
C2	11*	12*	12*	12*	113*
C3	13*	12*	10*	12*	113*
C4	15*	13*	13*	12*	125*
C5	13*	12*	12*	9*	112*
C6	13*	13*	14*	12*	123*
C7	12*	12*	15*	12*	121*
P1	1	1	5	6	48
P2	3	2	4	6	52
P3	9*	2	11*	9*	83
P4	7*	6	10*	8*	83
P5	11*	5	7*	1	69
P6	4	6	7*	9*	73
P7	6	5	5	6	65

*normal test score

Table 1. Standardized SCAN-A sub-test and total-test scores for control (C) and patient (P) participants.

Table 2

Participant	Threshold at 250Hz (dBISO)		Threshold at 500Hz (dBISO)		Threshold at 1000Hz (dBISO)		Threshold at 2000Hz (dBISO)		Threshold at 4000Hz (dBISO)		Threshold at 8000Hz (dBISO)	
	Left	Right	Left	Right	Left	Right	Left	Right	Left	Right	Left	Right
C1	10	15	10	10	5	5	0	5	15	15	25	10
C2	15	10	5	0	-5	0	0	10	5	10	10	5
C3	10	5	10	10	0	0	-5	5	0	5	15	25
C4	5	10	0	-5	-10	-5	-5	-5	-5	5	5	5
C5	10	15	10	20	5	10	0	5	0	10	5	15
C6	15	10	5	5	0	5	10	0	5	10	10	10
C7	15	20	10	10	5	5	15	5	10	15	10	20
P1	10	10	10	15	10	0	0	10	10	10	20	15
P2	20	15	10	15	10	5	5	15	10	10	10	5
P3	15	10	5	15	5	10	5	15	25	20	25	30
P4	10	5	5	10	-5	0	-10	-5	0	-5	0	5
P5	10	15	5	0	0	0	0	-5	0	0	10	0
P6	5	10	0	-5	5	0	0	10	5	5	15	20
P7	15	15	5	10	15	5	10	10	15	20	15	25

Table 2. Pure-tone audiometric thresholds for control (C) and patient (P) participants.

Table 3

Condition	% of hits		% of false positives	
	Controls	Patients	Controls	Patients
No Noise	98.6	98.2	0.1	0.2
Noise 65dB	98.6	94.6	0.1	0.3
Noise 70dB	96.4	92.5	0.1	0.5
Noise 75dB	93.6	91.1	0.4	0.9

For all conditions n= 7

Table 3. Percentage of hits and false positives in control and patient groups for all experimental conditions in which /da/ served as deviant.

Figures

Figure 1

Figure 1. Sound spectrogram and formant analysis of speech sounds /ba/ and /da/.

Figure 2

Figure 2. Grand-averaged standard and deviant waveforms at Fz, Cz, Pz, and LM for control participants for all conditions.

Figure 3

Figure 3. Grand-averaged standard and deviant waveforms at Fz, Cz, Pz, and LM for CAPD patients for all conditions.

Figure 4

Control and Patient Difference Waveforms

Figure 4. Superimposed grand-averaged difference waveforms for control participants and CAPD patients for all conditions.

Figure 5

Figure 5. Mean MMN peak amplitude and latency values for control participants and CAPD patients for all conditions.

Figure 6

Figure 6. Mean N1 peak amplitude and latency values for control participants and CAPD patients for all conditions.