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BACKWARD MASKING WITH SIMULTANEOUS EARLY, MIDDLE AND LATE EVOKED POTENTIALS

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BACKWARD MASKING WITH SIMULTANEOUS

EARLY, MIDDLE AND LATE EVOKED POTENTIALS

By:

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Declaration

I hereby declare that the work contained in this report has never been submitted for a degree in any other university. To the best of my knowledge, this report contains no material previously published or written by another except where due reference is made within the report itself.

I further declare that the ethical procedures and principles determined by the University of Montana's document on human research and experimentation have been adhered to in the preparation of this report.

Signed   Date October 31, 2016

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Silas Smith, M.S., Summer, 2016   Psychology / Commuicative Sciences and Disorders

Interdisciplinary Program

Backward Masking with Simultaneous Early, Middle and Late Evoked Potentials

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Abstract

Auditory processing disorders (APDs) affect a diverse range of people. These types of disorders impair auditory function, despite the outer, middle and inner ear maintaining proper function and health. APD is not necessarily related to auditory thresholds. When people with APD have difficulty discriminating sounds in connected speech, it may be due in part to an effect called Backward Masking (BM). Masking occurs when one stimulus inhibits another, which can lead to a variety of impairments. The neural locus of APDs is for the most part unknown, including the specific conditions which cause BM. A better understanding of these processes would lead to a greater ability to provide an intervention and therapy for APD. Electrophysiological responses have been well documented in a forward-masking paradigm, but not so in a backward masking paradigm. The significance of these responses is yielded through electrode signal input, a large degree of amplification and summation analyses of brain wave data. In this research a latency and amplitude deviance was detected in the early and middle stages of the auditory evoked response. Our data has revealed that the backward masking effect is observable at approximately the 90-250 msec range given the appropriate stimulus parameters. The temporal conditions of this effect lead to the conclusion that the BM effect occurs in the midbrain to the auditory cortex.

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CHAPTER ONE

Introduction and Historical Overview of Backward Masking

The central focus of this research is to observe the waveform morphological changes of the whole auditory evoked potential during a backward masking procedure. It may be possible to objectively measure the electrophysiological Backward Masking (BM) effect in human and animal models using auditory evoked potentials. The design of study will allow measurement of the early (0-10 msec), middle (10-100 msec), and late (100-350msec) auditory evoked potentials. This process will allow us to observe the differential electrophysiological responses of evoked potentials during the BM task.

Backward masking refers to the process of raising the sensory threshold for a target stimulus by means of an interfering signal *after* the target stimulus. BM is not unique to the auditory system. Masking effects are similarly exhibited in other perceptual senses as well (Raab, 1963). In simpler and shorter terms, BM is defined by later stimuli affecting an earlier stimulus. Masking effects have been documented as early as 1902, when the discovery of the Broca-Sulzer phenomenon established that the effect of length of viewing exposure was related to the apparent luminescence of an object (Raab, 1963). BM has demonstrated high significance for the study of Auditory Processing Disorders or APD's, including but not limited to several learning impairments. For example, children who stutter have a significantly higher threshold for BM, and the higher masking thresholds correlate with rates of dysfluency (Howell, Rosen, Hannigan & Rustin, 2000). There is no relation between the impairment of auditory feedback and the structural integrity of the auditory system; therefore, it is believed that the stuttering impairment occurs due to a dysfunction of central auditory processing in the brain (Howell et al,,

2000). It has also been shown that children with dyslexia were similarly impaired, and have significantly higher BM thresholds than matched control groups (Rosen & Manganari, 2001).

It has been hypothesized, and supported by a body of evidence that the BM event disrupts temporal processing at the level of the brainstem (Wright, Lombardino, King, Puranik, Leonard & Merzenich, 1997; Tahaei, Ashayeri, Pourbakht, Kamali & Jahanshahi, 2014). This temporal processing ability may be "mapped" through an analysis of auditory evoked potentials, measuring brainstem, midbrain and cortical electrophysiological functioning during an auditory task. An example of an early auditory evoked potential – Auditory Brainstem Response (ABR 0-10 msec.) is shown in Figure 1. Each of the peaks in this waveform correspond to specific loci within the brainstem. An example of auditory Middle Latency Response (MLR 10-100 msec) is shown in Figure 2 below. The response origins have been found to be in the midbrain. Figure 3 is an example of a late auditory evoked potential (100-300 msec). The neural origins of responses in this latency range are clearly cortical.
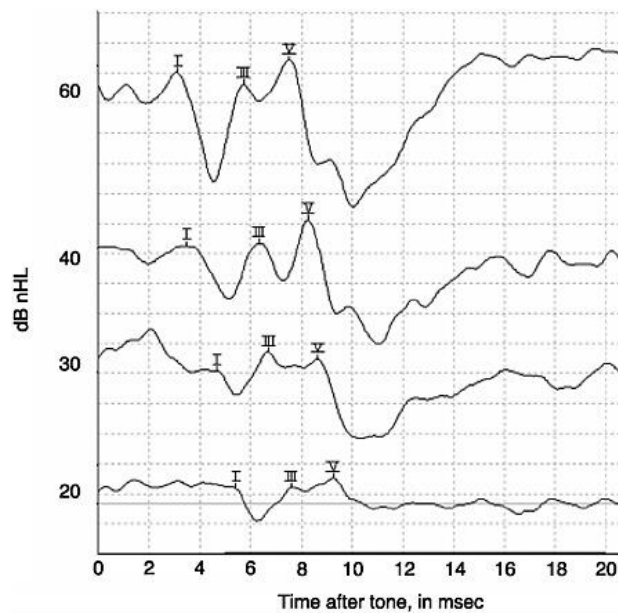
Figure 1: Early Auditory Brainstem Response (0-10 msec) (Dobie, 2004, p. 97).
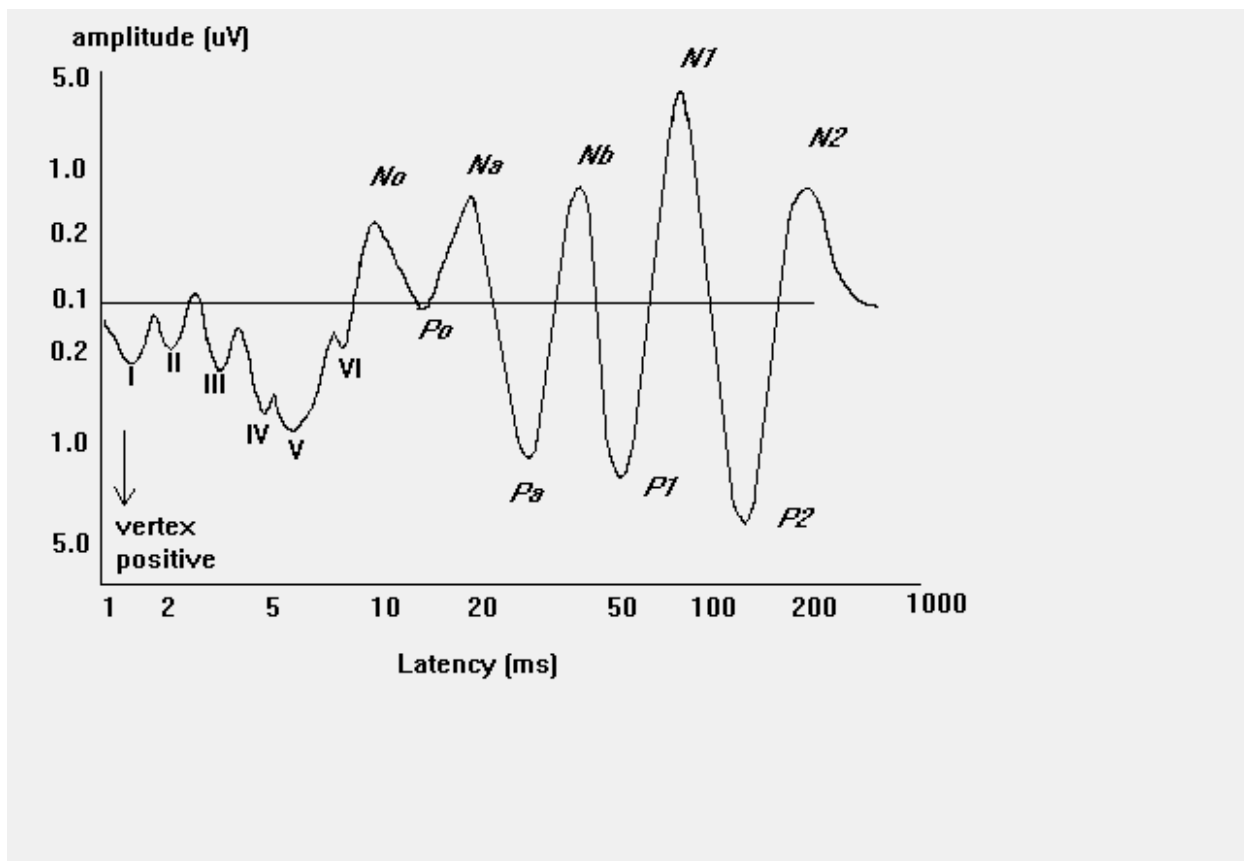
Figure 2 (Above): Early, middle and late auditory evoked potentials, as shown by (Barlow, 1982, p. 124).

A study of the latencies and amplitudes of the waveforms in the entire evoked potential will allow us to determine what structure or structures in the brain are responsible for the BM effect. The repetition rate and the technical issues related simultaneous acquisition of early, middle and late evoked potentials have been solved by Johnson and Yonovitz (2008). The ability to acquire the early, middle, and late components requires a fast sample rate (15625 samples per second) and a slow repetition rate. While other studies have speculated as to where

specifically in the brain where the BM effect occurs, our study will take an expanded approach, allowing us to determine in totality, which neurological pathways are affected.
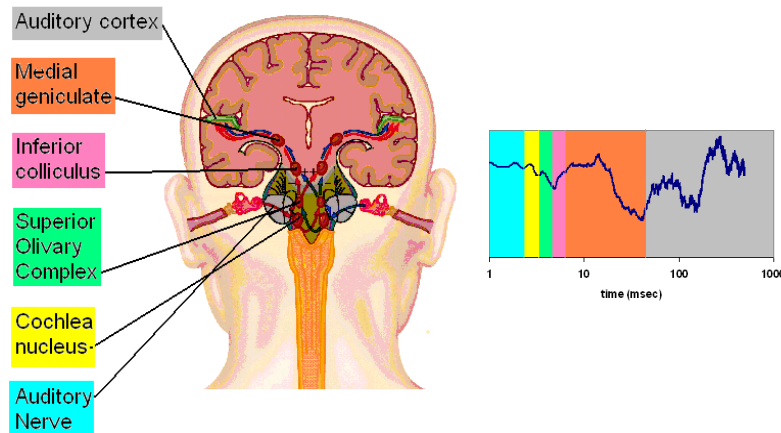
Figure 4: The auditory pathway and the neural generators for the specific waveforms. Adapted from (Kalat, 2007, p. 200)

Sounds are aurally received as more than one individual component or frequency, at least in the majority of real-world situations. The first reporting of human ability to hear multiple sounds at once was reported in 1843 (Colman, 2008). The first report of a disorder in hearing more than, or perceiving more than one noise at once did not occur until much later. Miller (1947), reported that there are inconsistencies in the ability to respond to multiple frequencies at once. Miller reported three different aspects to the masking effect: relative intensity of both masker and tonal noise, frequency of stimuli, and temporal separation of stimuli. There have been succeeding research endeavors that have cemented these findings as well (Samoilova 1956, Howell et al., 2000). Much of the early research regarding masking and indeed the interference of speech in general, was driven largely by two primary factors: The invention and progression

of the telephone, and World War II. These unique circumstances necessitated the understanding and circumvention of the disruption and interference of sound. Miller reported that the greatest interference occurs on a constant, pure-tone signal ranging from 1000-4000 Hz. Miller reviewed a host of different masking noises to reveal which paradigms yielded the most significant masking effects. This report assessed speech noises, pure tones, complex tones and even music, toting that (p. 112) "since much of the popular dance music of the day is (to some people) noisy and annoying, the possibility that it interferes seriously with speech was worth investigating" (Miller, 1947). It was found, however, that music was rather unobtrusive in a masking paradigm unless multiple sources of music were played simultaneously. Miller reported that low frequency masking noises were able to mask the full spectrum of audition, while high-frequency masking noises only interfered with a partial domain of audition. It was stated that this disparity is due to high-frequency noises being weaker in energy, and thus easier to produce, allowing also for easier masking. Miller's early work in auditory masking was an important foundation for further endeavors in the field.

The first acknowledgement of what is now known as Central Auditory Processing Disorders (CAPDs)was published in 1954, in a book simply titled: *Auditory Disorders in Children*, by Helmer Myklebust. Myklebust made a simple, yet novel discovery: that auditory deficits can, and do occur in individuals who present normal audiograms. In simpler terms, those who can hear *single* tones at normal thresholds sometimes fail to hear *multiple* tones at a normal level. This simple discovery led to a great deal of research, and frustration which both continue to this day. In order to understand the foundations and true consequences of these disorders, a basic understanding of auditory functioning must be known (Musiek, 2007).

Although the etiology is relatively unknown, CAPD's affect a numerous and diverse population of people across the world. CAPD's are defined as a disruption in auditory processing that occurs in the auditory pathway post-ceding the cochlear response. CAPD's are also generally believed to occur before cortical (semantic) processing. (Griffiths, 2002). Although it is hopeful that a true causality to central auditory processing disorders may be defined, it must be admitted that they may result from a number of different factors and may manifest in a number of different ways (as is true with the majority of speech and language disorders).

There is a sufficient body of research to promote and enable the current developments of research into the realm of APD's. One of the central tenants of this research is the significance of temporal processing and backward masking in such disorders (Musiek, 2007). It is clear though, that there is much to be learned, unlearned and reworked in this field; the research and etiologies delving into CAPD's are far from resolved.

CHAPTER TWO

Review of Literature – Backward Masking and Central Auditory Processing Disorders

Temporal Masking and Backward Masking – Subjective Assessments

There is a reliable history of subjective evidence that has proven the relevance of BM to language disorders. The first recorded instance of auditory BM was reported by Miller (1947). Miller tested auditory thresholds in a forward masking procedure for periodic tone bursts which were preceded with masking stimuli of varying intensity. It was found that when a tone was preceded by the masking signal, the threshold for audibility of the tone was significantly higher (i.e. poorer). The effects of auditory BM were further expanded on by Samoilova (1956), who reported that these masking effects were intensified when the amplitude of the masking signal was raised. Samoilova also reported that masking effects were increased when the duration of the pure-tone stimulus was abbreviated, and when the interval between the stimulus and masking noise was decreased (Raab, 1963). This research marks the first subjective assessments of masking signals. Samoilova determined the relevant parameters of pure-tone stimuli length were 20 to 100 msec, with an Inter-Stimulus Interval (ISI) of length 1-100 msec, masking amplitude of 10-100 dB and masking noise frequency of 650-6000 Hz. The maximum amount of masking amplitude achieved in these experiments was 70 dB at an ISI of 2 msec and pure tone stimulus of 20 msec in length according to subjective assessments.

Another early study of backward masking, and in fact titled *Backward Masking*, was published by Pickett (1959). Pickett touched upon Samoilova's earlier work in 1956, reporting that when the ISI decreased to 1 msec, the threshold was lowered (improved) to 60 dB. Pickett reported results distinctly similar to those of Samoilova three years previously, opening the door for a continued study of the backward masking phenomenon. Both of these authors stated that

there is a clear, and even a relatively linear correlation between ISI length, with exceptions for very short ISI's and masking level, pure tone length and masking level, and masking amplitude and threshold.

There have been many succeeding experiments and studies that have provided a well-established foundation for the backward masking phenomenon (Musiek, 2007). Subjective assessments for the effect have allowed a greater understanding of the role of temporal processing in audition.

Age Reports in Backward Masking

It is well-known that many aspects of audition change with age, this remains true with BM. Several studies have shown that BM performance declines over age, as does audition in general. However, even with normal hearing, aging ears - generally show defective BM functions. In 1993, a study aimed to corroborate the previous evidence for age related increasing BM thresholds. These researchers reported a robust evidencing of digenesis in BM function related to age both in terms of decibel threshold and inter-stimulus interval effects. These researchers reported that the younger group performed significantly better. Also reported was an interaction between age-related digenesis and ISI (Cobb, Jacobson, Newman, Kretscher & Donnelly, 1993).

In 1999, two researchers examined the progressive age-related effects in the backward masking paradigm (Gehr & Sommers, 1999). They reported robust findings of age effects in the data taken. Higher BM thresholds in two groups of individuals were measured in a subjective BM task using a 10 msec sine wave (.5 kHz) and a masking broadband noise (50 msec). There

was clear evidence of correlation between age and backward masking thresholds. These researchers found that in the younger group, with an inter-stimulus interval between tone and masking noise in the region of 6-8 msec and beyond, the BM effects were almost nonexistent. In comparison, the older age group exhibited backward masking effects even at the longest measured inter-stimulus interval (20 msec) (Gehr & Sommers, 1999).

Backward masking has been shown to follow auditory development (Hartley, Wright, Hogan & Moore, 2000). Temporal resolution – the relationship between speed of stimuli and accuracy of processing was the focus of this research. The main goal of study to measure the hypothesized improved temporal resolution thresholds in 10 year olds relative to 6 year olds. This plan of study followed the assumption that temporal resolution, and auditory performance in general, are improved in that particular range of development. It is reported that auditory function equivalent to an adult's is not achieved until around age 11 or so on average (Hartley et al., 2000) There was a reported 34 dB threshold advantage attributed to the older group. Age-related improvements were seen in auditory backward masking in 6 to 10 year old children. (Hartley et al., 2000). This evidence of causality between age and temporal/backward masking thresholds corroborated previous publications that have reported similar data. In this study, a correlation between lower IQ and increased backward masking thresholds was also reported, this aligns with Wright's work 3 years earlier which showed a 45 dB backward masking threshold elevation between older and younger groups. It should be noted though, that it is true that auditory function is improved in the 6-10 year old age range, however, the cause is not known. It may include factors genetic, external, or a combination of the pair; findings in that area must be regarded with some caution.
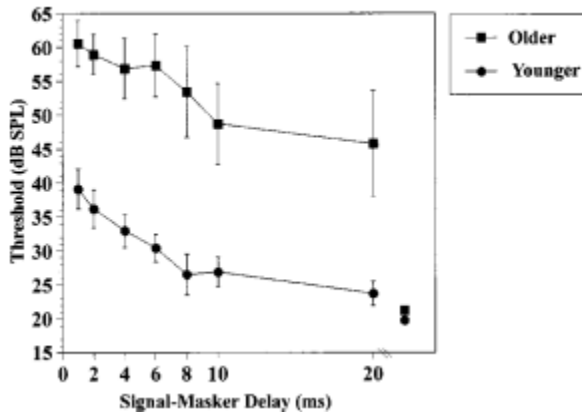
Figure 5: The objective BM thresholds for older and younger comparison groups (Gehr & Sommers, 1999, p. 2794)
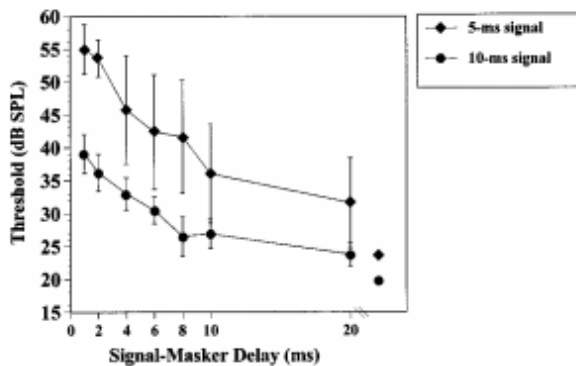
Figure 6: The comparison of 5msec and 10msec stimuli in the younger group (Gehr & Sommers, 1999, p. 2796).

There have been many historical studies claiming the significance of the improvement of auditory and language process during childhood development, and these researchers showed that backward masking function/processing is similarly developed along these years; pointing to, a significance in the context of audition and auditory processing (Hartley et al., 2000).

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It is clear that the auditory system, and indeed cognition in general undergoes marked improvement before the early teenage years. It has been shown that the auditory system is developed fully by age 11 (on average). Buss, Hall, Grose and Dev (1999) aimed to sequentially test the auditory system in 14 individuals as maturation was reached, and backward masking was the paradigm used to exhibit auditory temporal resolution ability. These researchers measured forward, simultaneous and backward masking. They hypothesized that younger children/individuals show greater variance in threshold detection, and higher thresholds in all 3 masking paradigms.

Two groups were studied, a younger age group of children aged 5 to 11 and an older group of adults aged 23-43. They tested bandwidth masking frequency as a variable with a 10 msec pure-tone stimulus of 1,000 Hz. These studies reported that there was great variance to be found under the BM paradigm, and relatively less so in the forward and simultaneous masking conditions. A reliable trend was reported in the data, in that masking performance is generally improved in children who develop normally from ages 5 to 11. This was proven true for backward, forward and simultaneous masking conditions. It is worth noting also that the degree to which masking performance was elevated was similar between paradigms (backward, forward and simultaneous).

Buss et al. (1999) claimed that this data provides evidence that the processing deficit is not due to basic auditory system function/processing based on the fact that there were no differences in results between the backward and forward/simultaneous masking conditions. The researchers asserted that attention-switching processes are a direct influence on the backward masking response, which indicates a disorder of central processing, and not a deficit in general audition. (Buss et al., 1999).

These studies show a succinct correlation between BM and auditory discrimination ability and development/digenesis. The BM phenomenon is present in all, although individuals with greater auditory confusion (i.e. those older in age) clearly show elevated thresholds for this effect.

Backward Masking, Dysfluency and Dyslexia

Howell et al. (2000) published a study correlating BM performance to the rate of dysfluency in children who stutter. These researchers proposed that the affect was due to a disruption of the auditory feedback loop. There is a marked increase of central auditory processing disorders in people who stutter, however, there is no difference in peripheral hearing evidenced. These researchers assessed professionally diagnosed stutterers as to whether their thresholds for the backward masking affect were different from those without any symptoms of CAPD. Performance on simultaneous masking assessments was also observed. Researchers used a subjective measure to report the threshold for effect. These researchers used a 40 dB masking noise with a 300 msec duration. The tone stimulus was a 1,000 Hz sine wave with a duration of 20 msec. Stimulus presentation was monaural. The ISI was 800 msec. These researchers found a distinct difference in the backward masking thresholds between the stuttering groups and the control groups in the backward masking condition. While the simultaneous masking condition remains relatively stagnant across the two groups, the disparity between groups in the backward masking condition is clear. People who are affected by stuttering experience masking of pure-tone stimuli much sooner (that is to say, at higher dB levels) than the control group. There is also a much wider degree of variability on the stuttering group, according to the box plot presented by

(Howell et al., 2000). There is an outlier belonging to the stuttering group that has a much lower backward masking threshold than either group. This suggests that there are additional unknown factors that are enveloped in the backward masking phenomenon, although it is clear that on average, the masking thresholds are much "worse". It should also be noted that in the simultaneous masking condition, there is apparently a wider degree of variability in the stuttering group as well, although the averages are much more similar to the control group under this condition.
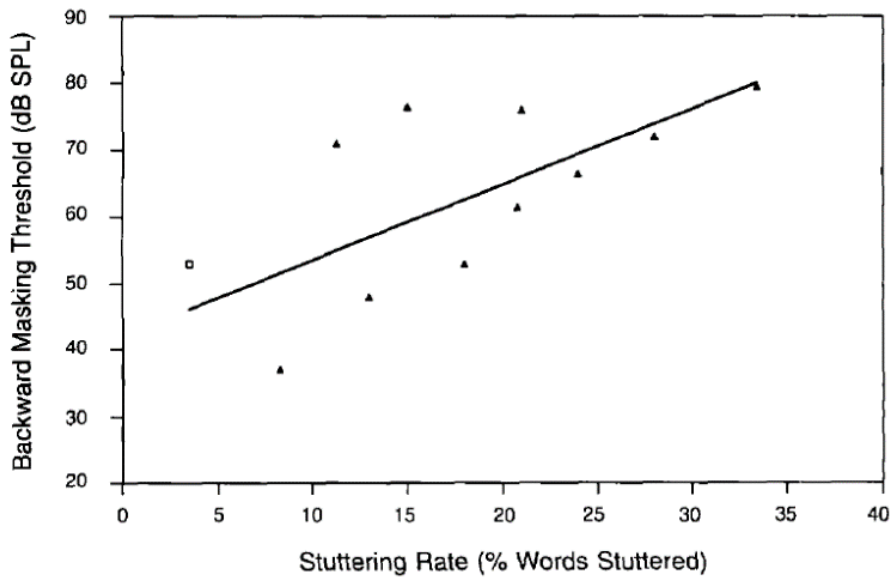
Figure 7: Correlation between stuttering rate and backward masking thresholds (Howell et al., 2000 p. 355)
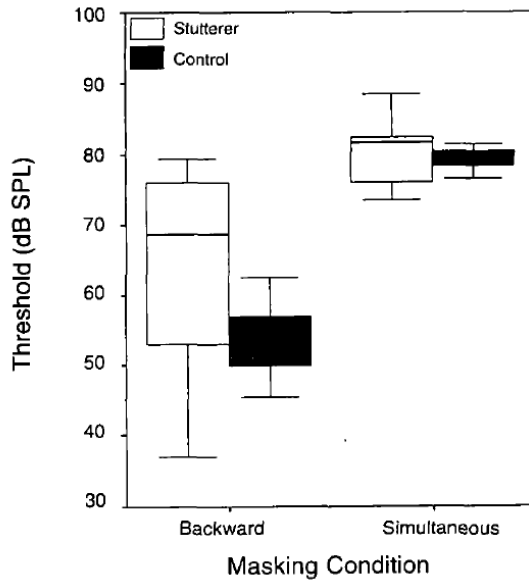
Figure 8: Backward and Simultaneus Masking Threshold Comparison (Howell et al., 2000, p. 355)

The figure above was also presented by Howell in the same study. These researchers evidenced a clear, linear correlation between the stuttering rate of individuals, and poorer backward masking thresholds. This robust evidence has been cited as one of the central supports for the relationship between backward masking and CAPD.

Backward Masking and Schizophrenia

BM, and forward masking as well have been shown to be correlated to schizophrenia. One of the common symptoms of Schizophrenia is experiencing auditory hallucinations (among other effects). These researchers reported that schizophrenics performed similar to the control group in a simultaneous masking condition, as do children with language learning impairments. However, in both a forward, and backward masking assessment, sufferers of schizophrenia showed significantly elevated thresholds. Furthermore, those who were more affected by

symptoms of schizophrenia, i.e. needing increased residential care, showed increased backward masking. Although the etiology for schizophrenia is truly unknown, backward masking is at the very least correlated to the dysfunction, and may share some significant causal factors. Auditory masking experiments in schizophrenia (Kallstrand, Montnemery, Nielzen & Olsson, 2002).

Backward Masking and Mental Ability

Researchers reported a correlation again between higher mental ability and the P300 wave, particularly in the amplitude and latency of the evoked potential (Beauchamp & Stelmack, 2006). Researchers measured this elusive variable of "higher ability" in terms of discriminatory response time, and specificity/accuracy. These researchers also reported discrepancy in the latency of the Mismatch Negativity (MMN) response in a deviant-stimuli task. This research also stated that the effects are due to an increased ability to access short term working memory that are necessitated by audition, as well as many other activities. They also stated that this resolution/discrimination task is autonomous in nature. Backward masking again was the paradigm investigated for the measurement of auditory resolution. Higher mental ability was deemed attributable to subjects with higher degree of accuracy in responses and faster response time. As others have since reported, when presented with a short enough ISI, the latencies of the evoked potentials became shorter, rather than longer.

These authors concluded that the nature of backward masking to these discriminatory processes is inherent. They noted that backward masking is an aptly appropriate task to measure response times in a deviant stimuli paradigm. These researchers explored the ISI parameters ranging from 25-150 msec, and white-noise masking stimuli ranging from 800 Hz to 1 kHz. The deviant stimulus was a pure-tone stimulus that varied between 633 Hz, 666 Hz and 700 Hz.

Backward Masking in Landau-Kleffner Syndrome

In 1998, a case study was published that explored the temporal processing difficulties that an individual with Landau-Kleffner Syndrome (LKS) exhibited. A specific type of acquired aphasia is manifested in a language disorder accompanied by convulsions. Researchers aimed to identify exactly what sort of lingual/non-lingual deficits occur in this disorder. It was found that William (the afflicted individual) had normal pure-tone audiometric thresholds, and maintained normal middle, outer and inner ear function. However, it was reported that the subject experienced discriminatory deficits when presented with BM condition (Vance & Rosen, 1998). The most afflicted stimuli were lingual in nature, although some non-lingual stimuli were masked as well, they were not masked to the degree that the lingual stimuli were.

This exposition evidenced yet another language disorder related, or at least correlating to temporal resolution characteristics LKS has been associated with lesions in the temporal lobe, specifically in the auditory cortex. The disorder is also associated with lesions bilaterally in the parietal lobe, superior temporal gyri, and the sylvian fissure. That being said, there is no conclusive definition as to the etiologies of this disorder.

These researchers explored a variety of auditory and language/communicative paradigms. The subject of this case study had apparently normal development until age 3, when his performance dropped dramatically, due to an unknown etiology. William experienced a variety of disabilities - auditory comprehension was affected early on in development, as was speech, although speech abilities were partially intact at times. EEG testing revealed the diagnosis of LKS in this particular individual. Further electrophysiological testing showed nothing significant – MRI evaluations did not detect anything unusual either. William was tested under a common

assessment aimed to determine whether individuals process auditory stimuli in a "top-down" or "bottom-up" style learning process. This yields (sometimes) the root functionality of auditory comprehension in an individual (Vance & Rosen, 1998).

Results from the case study are as follows: the individual showed normal auditory function in audiometric pure-tone assessment and auditory brainstem response measurement. William performed also at normal levels for a same/different auditory perception task. In a test involving auditory discrimination and attentional processes – The individual exhibited significant difficulties as compared to normal thresholds for children his age. William also exhibited difficulties on a test (clinical evaluation of language fundamentals – revised) involving receptive and expressive language skills. The subject performed at the skill level equivalent of an 8 year-old when he was 14. William also (at age 14) exhibited difficulties articulating speech, although speech processes were mostly intact.

In the realm of non-linguistic auditory processing tasks, William displayed relatively normal functioning. Auditory gap-detection task results were mixed, the subject displayed a deficit in the right ear, but not the left. In BM tasks, both simultaneous and backward William yielded very poor results. This being one of the most significant findings in the study. In assessments linguistic in nature, William performed very much worse than the control group. In auditory discrimination tasks (both word and non-word) William did not fare well, and performed equally poorly on a lexical decision task (Vance & Rosen, 1998).

APD and the Auditory Evoked Potential – Objective Assessments

It must be noted that the exact neural origins/processes for central auditory processing disorders are yet to be discovered. The best current models are based on a conglomerate of research agreed upon by current, devoted minds (Musiek, 2013). One special difficulty in the research of CAPDs is that cortical activity is markedly different in humans and animals. Research in this narrow field must, for the most part, use human subjects. This research is therefore limited in manipulability and nature of variables observed, regardless of whatever relevance they may or may not have. It is in this context, that subjective studies have come to prove especially important in the field of auditory processing research. However, as technological advancements have developed, there have been ventures into electrophysiological markers for auditory processing, primarily in through EEG assessments. Musiek (2013) reported on the relevance provided to central auditory processing by certain key features in the auditory brainstem response recorded through EEG testing. According to a slew of electroencephalographic data, Musiek (2013) stated the specific importance of waves III, IV, and V which mark functioning, or lack thereof, in the brainstem. These malformations in the waveform are likely due to brainstem lesions that affect the central auditory nervous system.

There has been a limited degree of recent developments in ABR measurements, due primarily to the fact that there are more precise methods (e.g. fMRI) that are used to test the most prevalent neural idiosyncrasies. In fact, the main *current* prevalent clinical area where ABR assessments are appropriate is for infant hearing screenings, cochlear implant and hearing aid assessments (when behavioral responses are not usually reliable), as well as the detection of small tumors. However, the few stalwart research ventures in the ABR field have been promising, in that there has been apparent detection of the BM phenomenon in ABR and evoked potential testing (Marler & Champlin, 2005). Auditory brainstem responses in this study were

tested concerning children with Language Learning Impairments (LLIs). Marler & Champlin (2005) hypothesized that the greatest morphological significance of the backward masking affect is in the "wave V" of the ABR. These researchers found that the waveform morphology of the two groups were not significantly different when measurements were taken under a no masking condition. However, when tested under a backward masking condition, the LLI group had a reportedly significant delay of the wave V response as hypothesized. Despite being a successful study, there were no formative conclusions drawn to the causality of the backward masking effect, only apparent evidence that the effect can be objectively measured. It must also be noted that the backward masking affect is of extremely small amplitude, and requires a very precise measurement.

De Pascalis and Varriale (2012) reported a study of a late evoked potentials using the mismatched negativity response (MMN), and mental ability in a backward masking paradigm. They defined this improvement by a measurement of ISI. Those who could hear the "masked" noise with a relatively shorter ISI were posited to have higher mental ability. These researchers proposed that the MMN response to a BM paradigm involves a process they termed "preconscious discrimination". It has been shown that a larger MMN response indicates that sensory discrimination processes are improved. This response (figure shown below) is related to auditory processing, even to the level of deviating morphologically based on grammatical and semantic changes. These researchers also investigated the effects on the p300 wave. It was hypothesized that the amplitude of the P300 would be greater, and the latency of the mismatched negativity response would be shorter when there is no masking condition present. When there is a masking stimulus present, it is hypothesized that the MMN would have greater latency, and the P300 would have a decreased amplitude; correlating to the intensity of the masking stimulus.

The P300, as the name suggests, occurs around 300 msec after onset; it has been highly

correlated with consciousness tasks. The p300 is also conveniently the 3rd reliable positive peak

in an evoked potential response. This event has been deemed a "task-relevant" response,

meaning it manifests as an event-related action potential, as a result of a conscious action. This

waveform is usually measured by an oddball task paradigm, where responses to outlier stimuli

are focused upon. There is a finite amount of attention-processes available. Attentional processes

are strained when there is one more stimulus that needs direct involvement. The p300 has been

shown to decrease in amplitude under such conditions (De Pascalis & Varriale, 2012).

Figure 8 (Below): P300 Evoked Potential Response (Kolodziej, 2012. p. 435)
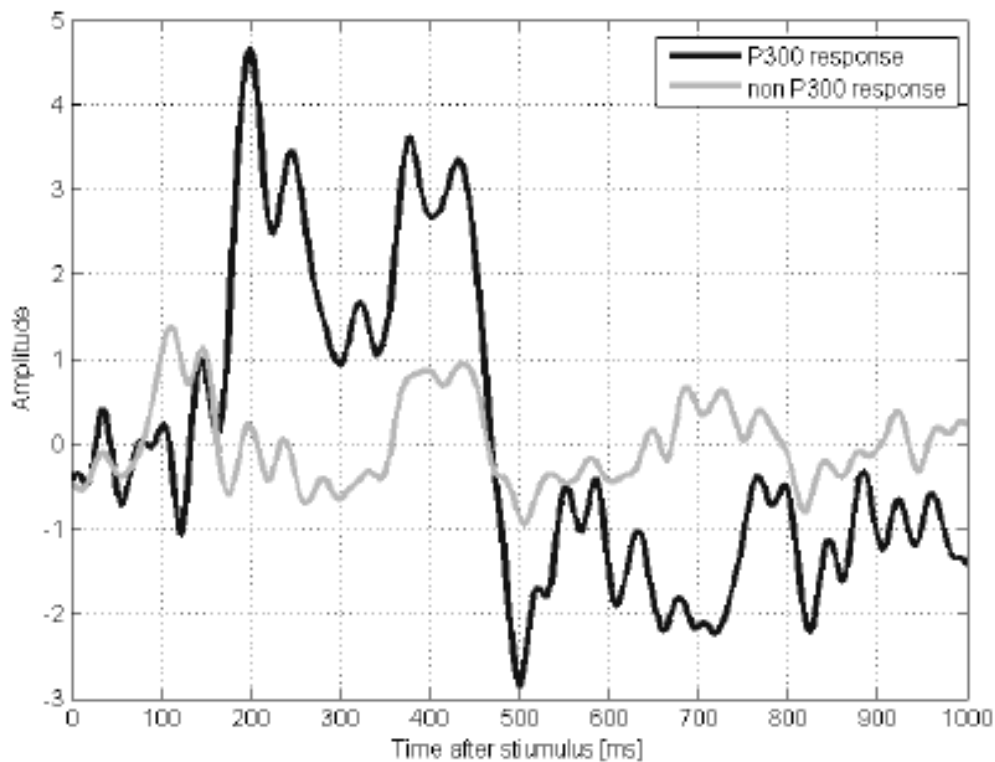
Figure 9 (Below): Comparison of MMN Responses for Impaired and Unimpaired Groups (Natanen et al., 2011, p. 3441).
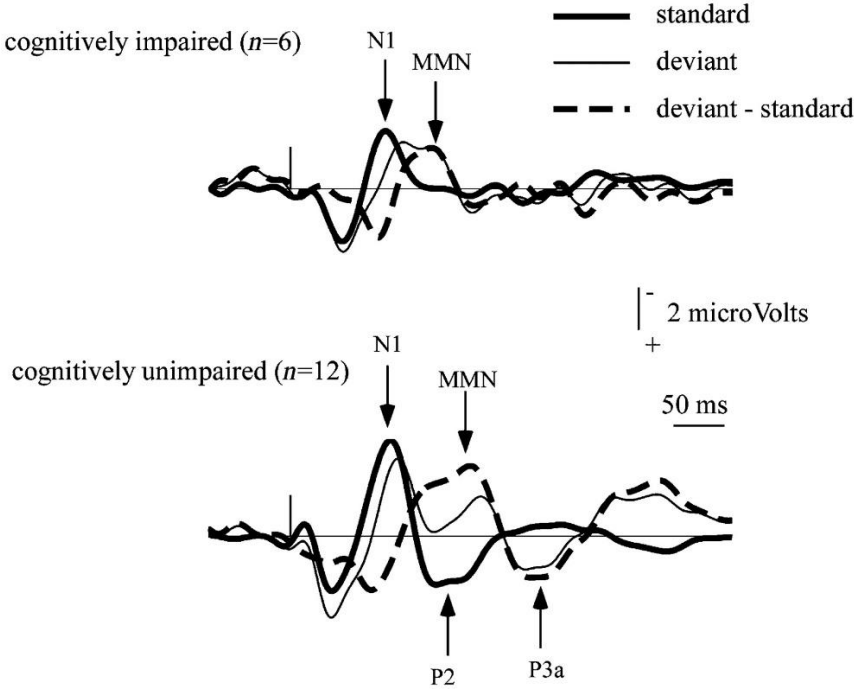
The MMN is a late evoked potential that is very reliable in recordings. This is because it does not require the subject to be conscious (De Pascalis & Varriale, 2012). This response is not subject to deviations caused by attention processes, or cortical activity, and can be measured simultaneous to activity of any sort. During experimentation, participants were asked to read a book (Beauchamp, 2006). The MMN manifests as a negative waveform, and is hypothesized to be the autonomous response to auditory stimuli, and is responsible for temporal resolution. (De Pascalis & Varriale, 2012) stated that the mismatched negativity response is reliably larger in individuals with higher auditory discrimination abilities. The BM function is highly correlative to the (MMN), which follows the logic of the theorized autonomous temporal

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resolution/discrimination hypothesis of the MMN wave. During various backward masking

tasks, the MMN has been extinguished entirely under certain paradigms. Data from the De

Pascalis and Varriale (2012) study showed significant effects between higher mental ability and

shorter length of the mismatched negativity response. Amplitude of MMN response was also

shown to be higher in these subjects with higher mental ability. However, MMN latency

decreased when the inter-stimulus interval was decreased, opposing the expectations of the

researchers.

It was found that the latency of the MMN was significantly shorter when the ISI was

decreased. The authors of this research alluded to the idea that the tones were processed as a

"gestalt", a single perceived noise composed of a number of other tones/noises that are

compounded (as opposed to a pure tone stimulus). This follows the assumption that the MMN

evoked potential is "passive" meaning the processes are initiated subconsciously (De Pascalis &

Varriale, 2012).

In another study regarding intelligence and auditory processing speeds, Beauchamp and

Stelmack (2006) reported that under a BM condition, the individuals with a "higher mental

ability" had better auditory discrimination between the tone and masking noise, as well as having

a faster neural response time. These researchers also reported that the higher mental ability group

had greater average P300 wave amplitudes, and shorter average latency on the P300 and MMN

waveforms. This particular study reported that the intensity of the amplitude, and the length of

the ISI were contributing factors to the significance of the differences.

Although not specifically tested under a backward masking paradigm an ABR task

evidenced that subjects with Persistent Developmental Stuttering (PDS) have a significantly

different evoked potential than subjects with "normal" language functioning (Tahei, Ashayeri, Pourbakht, Kamali and Mohammed, 2014). This current research demonstrates an affect that aligns with the hypothesis stating temporal resolution effects manifest in the central auditory pathway. This same hypothesis, although not stated as definite causality, was these researchers' primary explanation for the differing ABR effects. This study is of importance because the true cause of stuttering is not known, although it is known that the peripheral auditory system is unaffected (at least due to the stuttering). It is believed that the cause of stuttering is disruption in the auditory feedback loop is due to central auditory processing dysfunction (Howell, 2000). Effects were observed in the latency shift in the onset and offset of the waveform stimuli. Researchers observed markedly significant increased latencies in waves V, A, and O. It was also apparently observed that the V, A waves had a smaller degree of inclination. During data analysis, a strong correlation was drawn between the degree of stuttering present in speech, and the degree of latency in waves A and O. There was an apparent decrease of *synchronicity* as well in the PDS group, where peaks of waves were aligned with less consistency. The study also pointed to the fact the waves specifically related to spectral encoding were unaffected in the stuttering group. This again points to temporal processing as the causal factor for stuttering (Tahei et al., 2014).

One study (Kumar & Singh, 2015) showed that children with CAPD in a range of ages 8 to 12 have significantly different ABR potentials than those with "normal" auditory processing. The study was extensive, assessing 336 children in total, and performing MANOVA analysis to yield statistical significances of the experiment. The analyses revealed that the latencies of waves V and waves A were delayed. As well, they showed that the slope of waves V and A had a

smaller degree of inclination in those affected by CAPD. These researchers also reported that the

first formant was reduced in amplitude when compared to the control group. (Kumar & Singh,

2015). These researchers remarked that in previous studies (including some studies performed by

the researchers themselves), the waveforms most significantly affected by CAPD were waves V,

A, C, D, E, F and O. Also reported in other studies was the reduced degree of inclination in the

V/A waves.

(Banai, Hornickel, Skoe, Nicol, Zecker and Kraus (2009) reported robust evidence that

reading skill is correlated with subcortical auditory function by use of ABR. While it has long

been theorized, and held a limited body of research that phonological processing is key to the

reading process. (Banai et al., 2009) provided data that defines a correlation between reading and

central auditory processing skills. These researchers demonstrated that phonological decoding is

apparently directly correlated with the latency of auditory processing morphological waveforms.

This is a particularly important study because it develops a real understanding of the relationship

between reading and subcortical processes. Banai et al. (2009) correlated scores on reading

comprehension tests with subcortical measurements of ABRs to provide statistical evidence, that

latency delays in peaks V, A, C, D, E, F, and O are apparently correlated to reading

ability/function. i.e. those with greater latency delays on the waveforms through

electroencephalographic measurement on average, had lower reading ability.

Another nominal study established that there is a correlation between subcortical

brainstem functions and performance on central auditory processing assessments (Billiet &

Bellis, 2011). This study again established a link between phonological processing, reading

comprehension and auditory ability. This study showed with specific significance that dyslexia is

especially related to CAPD, and that ABRs may be able to diagnose CAPD's in an objective

manner, as opposed to current subjective tasks. This particular study narrowed the focus on the

dyslexia (one, if not the most prevalent CAPD). This study reported that 30% of children with

learning problems related to language (including but not exclusive to auditory processing

problems) have significantly different ABR measurements. This study took 32 children with

normal hearing sensitivity, and a professional diagnosis of dyslexia ranging in age from 8 to 12

years, and correlated their phonological processing (reading) skills with their evoked brainstem

potential recordings. These researchers showed that the ABR measurements for those diagnosed

with dyslexia are significantly different than the ABRs in individuals with "normal"

phonological processing. This corroborates the most current research on APDs and abnormal

brainstem responses. Consistently, it has been found that the brainstem response is at least one of

the factors that must be considered when reviewing central auditory processing disorders. This

study, again in corroboration with the apparent best, current research, has shown that waves A, C

and O are the locus of abnormalities in the brainstem response relating to dyslexia.

In an attempted objective recording measure of the BM task, Van Dijk and Backes,

(2002) used functional magnetic resonance imaging (fMRI) to assess the backward masking task

in adults with normal hearing. They recorded individuals in both a BM and simultaneous

masking condition (as a control). They reported several apparently significant effects in the

comparison between masking condition; those being: greater recorded activity in the cerebellum,

left inferior parietal lobe, posterior cingulate cortex and left inferior frontal cortex in the

simultaneous masking condition than in the backward masking condition. There was reportedly

greater activity in the *backward* masking condition in the anterior cingulate cortex and the

anterior temporal poles (laterally). These researchers cited this evidence as reason to believe that

simultaneous and backward masking respectively activate different neural regions and processes.

They went on to state that it is plausible to think that different lingual deficits may be caused by differently affected areas (Van Dijk & Backes, 2002).

Researchers reported a correlation again between higher mental ability and the p300 wave, particularly in the amplitude and latency of the evoked potential (Beauchamp, 2006). They measured this elusive variable of "higher ability" in terms of discriminatory response time, and specificity/accuracy. These researchers also reported discrepancy in the latency of the MMN response to a deviant-stimuli task. These authors reported that the effects are due to an increased ability to access short-term working memory that are necessitated by auditory and indeed many other activities. They stated, and later evidence supports (De Pascalis & Varriale, 2012) that this resolution/discrimination ability is autonomous in nature (happens in the brainstem). Backward masking again was the parameter investigated for the measurement of auditory resolution. Higher mental ability was deemed attributable to subjects with higher degree of accuracy in responses, and faster response times. As others previously, and have since reported, when presented with a short enough ISI, the latencies of the evoked potentials became shorter, rather than longer as expected.

These authors stated the nature of backward masking to these discriminatory processes is inherent. They also noted that backward masking is an apt and appropriate task to measure response times in a deviant-stimuli paradigm. These researchers explored ISI parameters ranging from 25-150 msec, and white-noise masking stimuli ranging from 800 Hz to 1 kHz. The deviant stimulus was a pure-tone stimulus that varied between 633 Hz, 666 Hz, and 700 Hz.

Haskins (2008) showed that chicks exposed to lead had auditory brain responses similar to ABRs found in children with language learning impairments. It has been well evidenced that

low levels of exposure to heavy metals like mercury and lead and cause central auditory processing disorders. This particular study is extremely relevant because it shows that the ABRs are very consistent in the resemblance of lead exposed chicks and language impaired humans.

BM has proven to be of high significance in regard to CAPD's. The BM effect has been correlated to language impairment, age-related auditory degeneration, and even in ailments such as schizophrenia. Although it may not be causal to all of these, backward masking is, at the very least, present in many language disorders, and remains a phenomenon worth investigating. The contributions that may yield from a greater understanding of BM are astounding. The ambiguity in definition and diagnosis of many neurological impairments could potentially be revealed through electrophysiological recordings of the BM procedure. It is blatantly clear that BM processing is an ability that is hindered in individuals with a variety of impairments. It is yet to be discovered though, the exact nature and functioning of BM neurologically.

Hypothesized Models for Auditory Processing Disorders

Although there is no agreement to, and likely no simple or single cause of auditory processing disorders, there continues to be valuable revisions as new research and technological advancements emerge. In the meantime, there are several hypothesized models that aim to encapsulate the effects and etiologies based on known evidence.

There is a host of recent studies suggesting that there is a comorbidity of auditory processing disorders and impaired phonemic discrimination in the central auditory system (Marler, Champlin & Gillam, 2002). However, the physiological cause or causes of the disruption is not known. The next step in the research of APDs is to determine why and how the stimuli "overlap" and interfere in the brain (Wright et al., 1997, Musiek, 2013). Researchers in

2002 reported a nominal study in which the source of language impairment in children was proposed to be a disruption in auditory processing and spectral resolution. These researches aimed to develop a more precise model to explain the deficits in learning and comprehension when individuals were otherwise unimpaired. They tested two masking condition with eight language impaired children, and eight children in the control group with reportedly "normal" language development. There were two especially relevant findings in the data acquired from this research. The first was that perception for the language impaired individuals was disrupted *more than* when the masking noise and tonal stimulus were similar in frequency (Marler et al., 2002). The second important aspect was the reported effect of higher backward masking thresholds in the language impaired group. This study concluded that children who are impaired in their language ability have a varying degree of difficulty discriminating two sounds within a short timeframe. They also suggested that the specific frequency of a backward masking tonal stimulus and noise affects behavioral results (Rosen and Manganari, 2001). Researchers aimed to determine if BM effects are different under speech and non-speech conditions for auditory stimuli. Researchers tested a group of 8 dyslexic individuals and a control group of similarly aged non-dyslexic individuals. Researchers determined first that forward masking levels were not significantly different between groups; backward masking thresholds however, were markedly heightened in the dyslexic group. Following this confirmation, they developed a method that tested if these elevated BM thresholds were the cause of dyslexia-related disruption. The authors theorized that if BM is the root of speech misperception, phonemes that contain consonant preceding a vowel will be affected to a higher degree under this condition as opposed to the consonant postceding the vowel. If backward masking was the root of the disruption, morphemes such as "ob" and "od" would not be affected as much as morphemes like "bo" and

"ba". However, these researchers found no discrepancies when the change in speech phonemes occurred secondary. Speech recognition should have been adequate on the changed term, because being second in sequence, it would not be subject to backward masking. Although these authors noted that there was initially a better measured ability in the non-dyslexic in terms of better general language ability. Under the backward masking condition, there was no discernable difference. These authors stated that this determines the backward masking task to be irrelevant in this paradigm to basic speech discrimination, but not complex speech noises. It is obvious that sweeping terms are not all that define speech perception, or the backward masking task for that matter.

There are several theories regarding the relationship of temporal resolution difficulties to speech and language impairments. The backward masking related impairments were originally attributed to difficulty in fundamental-frequency discrimination in rapid tasks (Reed, 1999). However, in a test developed measuring fundamental frequency discrimination ability in speech versus non-speech acoustic sounds, the non-speech phonemes, even with complex frequency shifts were not affected in a temporal resolution paradigm. This evidences the notion that the phenomenon (BM) is speech-specific (Rosen & Manganari, 2001). These researchers asked the question:

"could a non-speech deficit in children with dyslexia be used to predict performance in speech contrasts?"

(Rosen & Manganari, 2001) also remarked on the difficulty of capturing the predictive power of two stimuli that acoustically are very different. The non-speech broadband masking noise is much different than "real-speech" noises. These authors touched also on the model

proposed by Wright et al. in 1997 that the disability yielded itself in the rapidity of acoustic speech.

In a recent publication, researchers hypothesized that based on previous data, that BM disrupts *phonological* processes, and that these phonological interruptions are the driving factor behind temporal impairment (Heath, Hogben & Clark, 1999). This disruption is presented in disabled readers with a comorbid language disorder. Accordingly, they stated that the temporal processing deficits were *not* present in disabled readers without a comorbid language delay (oral). These researchers aimed to reach a more definite conclusion as to these effects and etiologies.

Data was taken on a sample of 7 to 10 year olds; it was found that disabled readers with a co-occurring oral language delay experienced disruption in auditory temporal processing. However, the individuals without oral language delay, even those with reading disorders showed normal temporal processing thresholds. These authors proceeded to state that there is a plausible correlation between oral/phonological processing and these temporal thresholds. This theory proposed by Heath et al. in 1999 states that the loss of rapid/temporal acuity that is present in auditory processing disorders deters the individuals' phonological awareness.

In another widely cited study, Marler et al. (2002) reported a comprehensive review of auditory memory in children with a language impairment by means of a backward masking task. These researchers defined an auditory temporal processing disorder as "an impaired ability to separate sounds in time." Marler et al. (2002) contended that higher backward masking thresholds are correlated with language impairment and therefore delayed perceptual learning in children. To prove this, these researchers measured backward masking thresholds in children with LLI. Marler et al. (2002) stated: "The question remains whether the disruption was at a

sensory, memory, or cognitive level." Marler et al. (2002) at first replicated the findings of other psychoacoustic assessments of children with LLI, with good footing, the researchers solidified the model of backward masking related to CAPDs. This research furthered the model by measuring the backward masking affect objectively as well as subjectively through electrophysiological recordings. This research used stimuli consisting of a 10 msec 1 KHz pure-tone signal with a 5 msec rise/fall envelope followed immediately by a 150 msec narrow-band masker (.6 KHz – 1.4 KHz). Stimuli were presented monaurally. These researchers observed that both behaviorally and objectively, there were statistically significant differences between the language-impaired group and the control group. Higher backward masking thresholds were observed in the LLI group, and the MMN electrophysiological response was delayed and reduced in amplitude. Marler et al. (2002) asserted that the disruptions in the MMN response was due to a disruption of early auditory memory. They stated that there are two cortical regions associated with the mismatch negativity response: small specialized regions in the auditory cortex that process varying aspects of acoustic stimuli, and independent stimulus processing region of the frontal lobe, which may also play a role in attention-switching processes. The MMN disruption also supports the model of impaired auditory encoding.

Naatanen, Kujala & Winkler (2011) reviewed a model of auditory processing regarding "conscious perception". Primary causality of auditory discrimination and processing was deemed to be related to the MMN and N1 evoked potentials in the brain. This study confirmed several others that have evidenced, that central auditory processing is related to certain evoked potentials. The focus of this study in particular was to determine which auditory processes are conscious and which are not.

In a recent attempt to model the auditory pathway through ABR, (Johnson, Nicol, Zecker & Kraus, 2007) described in detail the nature of this paradigm. These researchers linked two theories regarding auditory processing into a single comprehensive model. The two theories included in the research were the "source-filter model of acoustics" and the "cortical sensory processing streams model". The source filter model refers to the constant filtering of speech stimuli in the vocal tract when speech is produced. The cortical sensory processing theory is the more relevant model, at least in regards to the focus of this paper. The sensory processing theory was first shaped in the context of the human visual system. It was proposed (and later evidenced) that there are two separate, but simultaneously functioning pathways that are used to process visual information. These pathways (dorsal and ventral) are both used to identify objects, but they are focused on different aspects of visual stimuli. Some time later, research was published that evidenced a similarly functioning system in the auditory pathways (Romanski, Tian, Fritz, Mishkin, Goldman-Rakic, Raushecker, 1999). Johnson et al, (2007) went on to define the brainstem response to a complex sound as: "a gauge both of spectrum encoding – which is indicative of the overarching organization scheme of the auditory pathway – and of periodicity encoding". These researchers stated that the brainstem response is replicable and reliable in individuals. Johnson et al. (2007) also reported that the early waves in the auditory pathway (3 msec or less) were especially relevant in diagnosis when presented with an auditory stimulus. These authors went on to say that the process of encoding of frequencies is yielded in the brainstem in an amplitude and latency shift of waveform peaks (Steinschneider, Schroeder, Arezzo & Vaughan, 1993). This study, in addition to observation of early waveforms, observed the "frequency-following response" waveform (15-150 msec). It was reported that the FFR is accurate to the point where an EEG taken of the potential following a speech stimulus can be

amplified and presented audibly as the same stimulus. The proposed loci of the FFR are the

lateral lemniscus and inferior colliculus, although there is still some debate on the matter. The

stimulus presented in the experiment was a complex speech sound 40 msec in length. Below is

the hypothesized "mapping" of the auditory pathway as proposed by Kraus & Nicol (2005). As

evidenced in previous research, specific latency differences are shown between waves and

amplitude of waves that constitute the "expected" brainstem response to the speech stimulus.
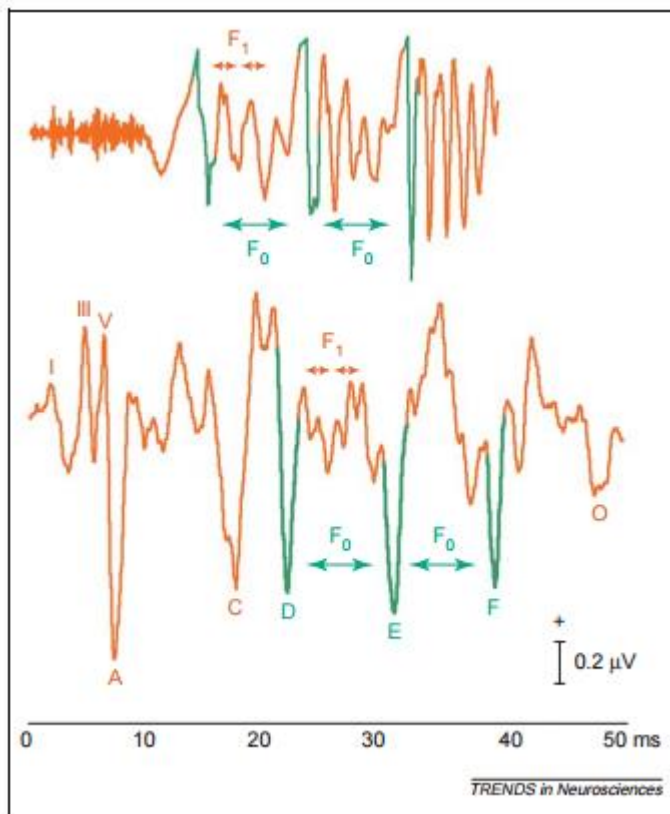
Figure 9: Early/middle auditory evoked potential with labelling of specific waveform attributes
(Kraus & Nicol, 2005, p. 179).

Johnson, Nicol, Zecker and Kraus (2008) completed a study that progressed the

knowledge of the relationship between language impairments and the backward masking

phenomenon. Researchers reported that children with an assortment of language related

discrepancies suffer from a lacking ability in temporal resolution, i.e. sounds/tones in quick

succession were perceived with greater difficulty. These researchers proposed that these effects happen in the low-level auditory pathway. These researchers measured an objective backward masking test and then formed two groups, one of better and one of poorer auditory temporal resolution. The groups were then measured in an objective manner through auditory brainstem evoked potentials. The primary variable in this experiment was the ISI. These researchers stated that this deficit in temporal resolution is not due to a cortical deficit but an "encoding" deficit in the brainstem related to acoustic cues. The rapidity of the morphology in speech is the key contributor to this auditory confusion. They proposed that if the evoked potential response to a backward masking task would determine if this phenomenon occurs at a subcortical level, or later. These authors purported that between 5, and 10 percent of children with normal peripheral audition are afflicted with some degree of language-related learning disorder. They also reported that these effects are antagonized with increasingly rapid successions of auditory stimuli.

In a comprehensive review published in 2014, the underlying etiologies of central auditory processing disorders were yielded under a factor analysis statistical method. These researchers agreed that there is not a concrete definition available for central auditory processing disorders; they did concede though, that CAPD is marked primarily by the peripheral auditory system maintaining facility and normal functioning on a pure-tone audiogram (Ahmmed, Ahmmed, Bath, Ferguson, Plack & Moore, 2014). These researchers attempted to complete a totally comprehensive statistical analysis as to the true cause/correlatively to central auditory processing disorders. From these statistical analyses, 3 primary factors were manifested. The first and most prevalent of the 3 these researchers termed "general auditory processing". The other two, "working memory" and "processing speed." These three driving forces behind central auditory processing disorders were manifested statistically by means of factor analysis. The

"general auditory processing ability" was deemed according to a battery of tests involving backward and simultaneous masking, frequency discrimination and accuracy, and speech processing. "Working memory" was determined by tasks of executive attention, cognitive-related batteries, and listening tests. Processing speed was measured by motor-related input speeds relative to certain tasks. The authors remarked that there is obvious variance in symptoms and abilities, but hoped still to tie together underlying causes and their subsequent effects. They first aimed to define APD, however, and remarked that generally, the presence of a hearing problem while a normal pure-tone audiogram is maintained marks this particular disorder (Ahmmed et al., 2014).

The terming of such disorders remains somewhat murky however, as many co-occurring disabilities are present in many of these affected individuals. The American Speech and Hearing Association has taken the position that APD is not higher-order in nature i.e. due to cortical malfunctions. However, some more recent research endeavors have explored the idea that a significant affect in evoked potential tests is a result of attentional processing differences, which are inherently "higher-order". Short-term working memory yielded significant correlates as well (Ahmmed et al., 2014). Although there is not consensus, there are a few likely factors that this study aimed to encapsulate.

Reading abilities were also given importance to in this study, and have been a factor in much of APD research current and historic. Again, given the complexity and elusive nature of the reading process, it has been highly disputed if and how auditory processing disorders are related to neurological functioning. The authors of this factor analysis noted that some recent studies have shown no significant relationship between auditory comprehension and reading. It may be that APD occurs alongside many reading disabilities due to some common etiology. At

any rate, there can be no definitive conclusions drawn at this point. This study aimed to develop the most appropriate battery of tests that inclusively measure and assess APD. They insisted a multimodal approach to this assessment is necessary, despite the admitted murkiness in definition that leads to complications. These authors stated that factor analyses have been performed on auditory processing assessments previously. However, the analyses were done on "out-dated" batteries. These analyses did yield two primary contributing factors though, "binaural separation/competition" and "composite monaural low-redundancy degradation" (Ahmmed et al., 2014).

Mcanally and Stein (1996) published a paper that posited a controversial opinion – that dyslexia is not a higher-order cortical disorder, but a disorder of early processes occurring in the brainstem. These authors stated two findings that demonstrated this hypothesis. The first being the significance of backward masking functioning and frequency discrimination; The second being the measured evoked potentials. These researchers assessed 23 dyslexic adults, as marked by the difference between nonverbal intelligence and reading ability. These researchers stated that dyslexic individuals exhibit poorer performance relatively when compared with control groups in the presence of rapid auditory tones. It was stated, according to the data taken, that the inter-stimulus interval between the tone and masker did not yield a great effect between the dyslexic group and the control group. Temporal encoding was the factor most discrepant between the control group and the dyslexic group. Temporal encoding refers to the accuracy of the coding of stimuli onset/offset in the brain.

Frequency of stimuli in temporal encoding has been hypothesized to be the result of phase-locked nerve fibers. These fibers fire at the same rate as the auditory input for tones 5 kHz and below. It was stated that the dyslexic group under the masking condition had significant

difficulties detecting frequency changes in the tonal noise around 1 kHz. This suggests that the impairment resulting in language disorder in dyslexic individuals is a result of a disruption in the temporal encoding of these phase-locked discharges, which in turn effects frequency discrimination.

These researchers found that the greatest masking effects were achieved during a binaural masking condition, where the phase difference of the tone was 180 degrees inter-aurally presented. This gives support to the idea that phase-effects are related to the disorders presented in dyslexia.

An objective assessment of the dyslexic individuals was presented in an evoked potential task. Measured was the far field potential: an evoked potential that has been shown to measure directly the firing of the phase-locking neurons. These synapses occur in the brainstem, and it has been shown that lower amplitude in the far field potential correlates with reduced accuracy in phase synchronicity. Evidenced in the data taken in this study, was a significantly lower amplitude for the far-field potential. (Latency of the potential was not significantly affected). The fact that the latencies of the waveform did not differ from the control group lends support that the effect occurs in the brainstem (Ahmmed et al., 2014).

It has long been touted that central auditory processing is not a disorder due to peripheral hearing. Musiek (2013) takes an interesting approach to this model that is not in direct opposition, but claims the process is not quite so simple. Musiek stakes that the greatest masking effect indeed is not due to peripheral dysfunction. Masking effects occur predominantly in a range of 10 msec and below, resolving in the brainstem. However, it has also been shown that BM effects are yielded from the 15-25 msec range as well, and these effects may be related to basilar membrane functioning in the peripheral auditory system. Musiek also cited authors who

state that forward masking is more prevalent to peripheral auditory functioning. There is an issue with this model though, in that individuals fitted with a cochlear implant – in other words, those lacking any peripheral processing at all show similar forward masking thresholds.

Marler (2002) considered auditory memory, specifically its relevance to low-level processing. These researchers specified that the backward masking effects primarily operate on complex (non pure-tone) acoustic stimuli that are nonlinguistic in nature. They provided both objective and subjective data to defend their case. Marler (2002) tailored two models to encompass auditory processing disorders: The first being a sensory approach, stating that the temporal disruption experienced in CAPD is due to an incomplete rendering of acoustic waveforms due to some quality of features in the auditory system. The second approach cites low-level auditory memory as the central tenet for temporal disruption. These theories state that the disruption manifests during the encoding/storage of memory processes. This early auditory memory is highly correlated with the MMN response. This potential is not cortical i.e. higher order in constitution.

These authors stated that an affect in the N1 morphological potential would indicate a sensory disruption. An affect in the MMN would indicate a disruption of low-level auditory memory. After electroencephalographic measurements and data analysis, it was found that the N1 potential was intact, and that the MMN was significantly delayed temporally, and diminished in amplitude. Provided this data, these authors stated that low-level auditory memory is pays a key contribution to central language impairments. These researchers described a model based on neural encoding into memory that does not take sensory mechanisms into account. In 2005 Marler continued his studies in the auditory processing field. Marler (2005) made an addendum to his earlier research, reported that the wave V response is significantly reduced in addition to

the MMN response. It was therefore proposed that attentional activity is incorporated in the response. Marler (2005) also remarked that these disruptions appear to be pre-linguistic, meaning they occur before language areas are cortically activated. It is likely then, that the disruption occurs in the brainstem. A misfiring of synapses in certain context may produce auditory temporal disruptions.

There has been great a deal of evidence alluding to the idea that auditory stimuli are processed hierarchically. This invokes the idea that primary auditory/language areas have higher neural activation than non-primary areas in a linearly correlated fashion (during a speech/language task). As well, stimuli that are more complex in nature retain greater neural activation (Hall, Johnsrude, Ingrid, Haggard, Palmer, Akeroyd & Summerfield, 2002). In an fMRI paradigm, determined that a multi-frequency harmonic tone yields greater neural activation in a few key areas when compared with to a pure-tone stimulus. Heschl's gyrus showed higher activation in the right temporal lobe, and the supratemporal plane showed higher activation in both the right and left hemisphere. These researchers cited this evidence, along with previous research to the theory that the auditor cortex is formed hierarchically (Hall et al., 2002).

Escera Leung, and Sumie (2014) purveyed a theory that states that the auditory hierarchy starts as low as the brainstem. They accomplished this using a deviance detection based paradigm. Reported in the data was that the evoked potential related to detection of a deviant/unexpected stimuli was marked by an aberrance in the Mean Latency Response (approximately 10-80 msec after onset) that was distinct from the deviance marked in the MMR response (approx. 100-240 msec after onset), and in the brainstem as well. In other words, the waveform morphology was different for each pathway/response, and the notion that the disruption manifests differently in separate auditory regions aligns with the theory of auditory

hierarchy. These researchers also reported similar findings of deviance detection evoked potentials in tested animals.

It must be noted that it takes exact and minute measuring techniques to find significant results in the span of a few microseconds. There have been articles stating there are no significant effects to be found concerning backward masking and certain language impairments. These studies raise a deal of questions on measurement, reliability and validity.

Training/Attentional Processes relevant to CAPDs

There have been some relevant experiments that have attempted to clinically improve performance on temporal resolution tasks in language impaired children. Some have displayed significant improvement in such endeavors. At the very least, it is worth noting that training may affect the backward masking procedure. It should be noted that although individuals with and without CAPD exhibit training benefits on backward masking/temporal resolution tasks, those without language impairment show greater potential for improvement.

Merzenich, Jenkins, Johnston, Schreiner, Miller and Tallal (1996) reported that certain cognitive processes, language learning included, can be dramatically improved by means of behavioral training. These improvements were demonstrated subjectively and objectively in an electrophysiological procedure. These researchers evidenced this data to hypothesize that the language disorders related to those temporal deficits are rooted, and manifested from a history/context of poor learning. Temporal/perceptual development may be the causal factor to these language impairments.

In this experiment, researchers attempted to train children with a professionally diagnosed language delay in an attempt to lessen the temporal resolution disruption. They used two different training methods, although both methods were manipulated in an audiovisual realm, presented in the form of a game. The games were reportedly designed to engage the individuals as much as possible, to evidence as much affect as possible on the training variable and with the age range (5-10) and the individuals' unique abilities in mind. The first game the authors labelled a "perceptual identification task." This task involved two auditory tonal stimuli played in rapid succession. The second game involved the training of phonetic awareness in the language-impaired individuals. In the first trial of the experiment, training took place over 4 weeks with each individual receiving 19-28 training sessions of length 20 minutes. Five of the seven children tested in the first session exhibited language-learning related benefits, the majority of whom showed increasing benefits as the training continued. Two of the seven children that underwent training even surpassed normal thresholds. Before and after training, the "Tallal Repetition Test" was given, this test being an agreed upon method for assessing temporal processing abilities. The Tallal test showed significant improvement in temporal processing/sequencing abilities. These authors reported that the greatest advantage experienced after training was in the detection of brief stimuli, and under a brief ISI condition. The second test involving phonemic awareness established beneficial results as well. Six of the seven participants performed markedly better after undergoing training. This comprehensive study corroborates previous evidence that temporal processing and some language-related delays/impairments appear to be related.

CHAPTER THREE

Statement of the Problem

The main basis for this research is simple in nature, but requires great caution to circumvent. The auditory evoked potential is only a few microvolts in amplitude, and is susceptible to interference and deviations due to hardware technicalities, and from the individuals themselves in the form of cognitive activity. The main problem lies within the specificity that must be applied to the parameters of experimentation. The variables must be manipulated in a way that produces the greatest observable effect from an extremely small input source. Furthermore, there are some assumptions that this hypothesis follows according to the most prevalent and historic evidence. It is assumed that the auditory brainstem response is a functioning measurement of auditory integrity at the sub-cortical level. It is also assumed that the backward masking response will affect audition enough to detect within reasonably precise measurement protocols. It is also assumed that with sufficient reliability/validity testing before data is recorded, that the hardware, software and methods used in the experiment will reveal backward masking effects within the waveform data. It is not guaranteed that these assumptions hold true, but the BM effect and its causalities remains worth investigating. It has been made plain through recorded evidence, that BM is highly relevant to CAPD's. It has also been clearly evidenced that individuals with CAPD's exhibit auditory evoked potentials that are distinctly different from individuals without CAPD. However, the electrophysiological bridge between BM and CAPD's that would identify the true causality of such disorders has not yet been properly identified.

General Questions Addressed

1) Can the BM effect be measured objectively in humans through the use of evoked potentials?

2) Using early, middle and late evoked potentials where is the brain locus for the BM effect?

CHAPTER FOUR

Methodology

This study has been approved by the University of Montana Institutional Review Board for the Protection of Human Subjects in Research (IRB). The informed consent for this research is shown in the Appendix.

<u>Subjects</u>

Subjects were volunteers from The University of Montana. The participants were 6 college-aged female students with no history of auditory dysfunction. Pure tone hearing sensitivity for each subject was assessed and was within normal limits. The only restriction was exclusion for non-normal hearing and a previous diagnosis of APD. The mean age and range of the subjects was 22.4 and 20-25, respectively.

<u>Stimuli</u>

There were two different classes of stimuli, 10 msec pure tones and rectangular pulses. The pure tone stimuli used a Blackman envelope to minimize extraneous frequencies created by the brief tonal signal (see spectral analysis, figure 14). The rectangular pulses were a rarefaction click with a duration of 100 µsecs. Each of these was followed by an interval of silence, the Inter-Stimulus Interval (ISI). The noise stimulus was a 50 msec broad band noise with a linear rise-fall time. The spectrum of this noise is shown in figure 15. The noise and tonal stimulus was 70 dB HL. The rectangular pulse stimulus was 70 dBnHL. The ISI was 10 msec. A control condition consisted of silence and no stimulus was presented. A total of 4000 stimuli were utilized. Each of the four stimulus conditions including the control condition were presented randomly each being 1000 presentations. The temporal integrity of the signal (tonal or rectangular pulse was maintained and is shown if figures 10-12.

Figure 10: Pure-Tone and Masker (temporal integrity)

Figure 11: Isolated Pure-Tone Stimulus (temporal integrity)
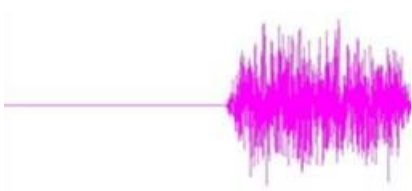
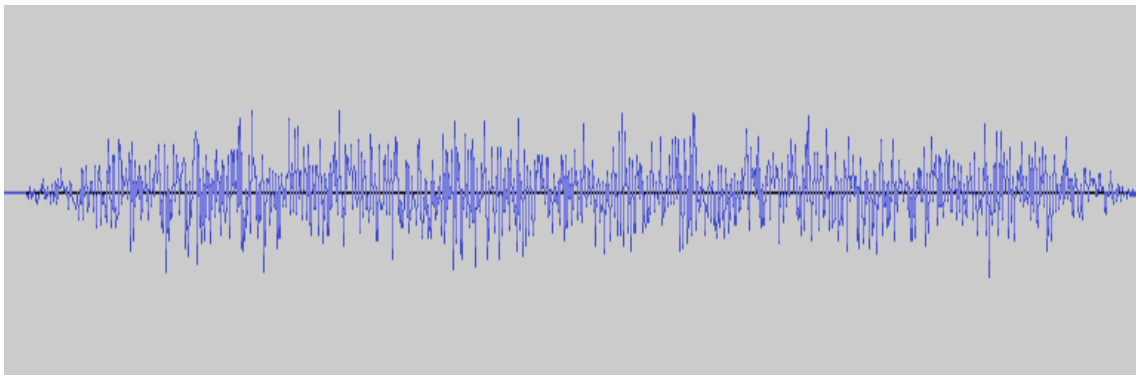

Figure 12: Isolated Masking Stimulus (temporal integrity)
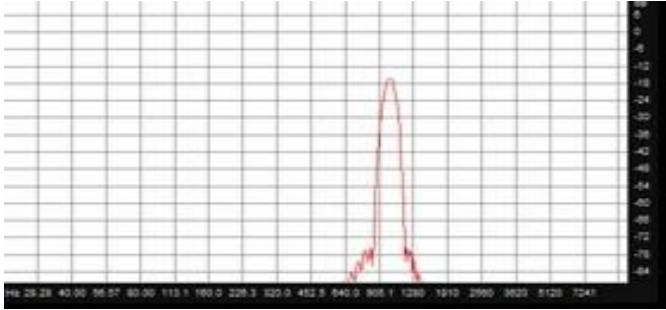

Figure 13: Isolated Masking Stimulus
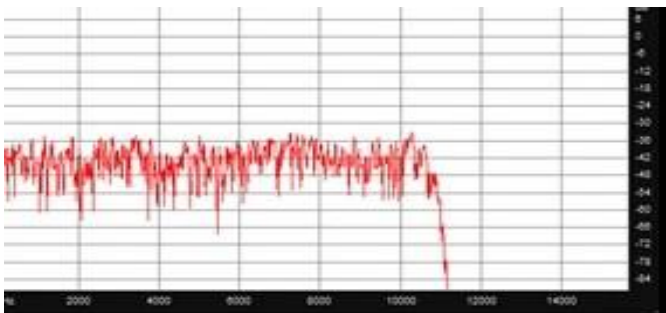
Figure 14: Spectral analysis of the tonal signal

Figure 15: Spectral analysis of the noise signal

Apparatus

The generation of electrically and acoustically noise free stimuli were paramount to evoked potential analysis because of artifact possibilities. Three different types of apparatus were considered. The first consisted of a Hewlett Packard (HP3000) Arbitary Waveform Generator. A computer program was implemented that presented randomly each of the 4000 stimuli. Unfortunately, a design fault in the instrument generated a spurious signal upon each load of the signal (HP, 2016, personal communication). An additional attempt was made to

construct an arbitrary waveform generator for specific use in this project, however, again noise levels were deemed too high to proceed. A third method proved successful and was used in the study. This method consisted of simultaneous use of A/D and D/A coding within the evoked potential program (see program, Appendix B). For this procedure the program was able to generate both the required random stimulus as well as acquire the bioelectric potentials simultaneously. All programs were either written in Microsoft Quickbasic 4.5 or Quickbasic 64.

Procedure

For the electrophysiological task a biological preamplifier (Grass, P5J)) was used with gold-plated electrodes, at the vertex (Cz, referenced to the left ear mastoid) to obtain the evoked potentials. The right ear mastoid was used as ground. Inter-electrode impedance was below 6k ohms. A total of 4,000 EEG time epochs were used to determine the summed evoked potentials. Each summed evoked potential was composed of 1000 EEG epochs (4 x 1000). The inter-stimulus interval was 1 sec. Thus, the subject listening task was 4000 sec or 66.7 min.

Evoked Potential Analysis

Each evoked potential was acquired at a sample rate of 31250 samples/sec. This sample rate was sufficient to allow both early, middle and late evoked potentials to be acquired. Early evoked potentials over a 20 msec period had 312 sample points. The inter-trial interval of 1 sec was sufficient to provide independence of trials for the late evoked potentials.

Statistical Analysis

All statistical analysis was accomplished using Statistical Package for the Social Sciences (SPSS, v14).

**CHAPTER FIVE**

Results and Discussion

Results

Six subjects were tested for both the tonal and pulse stimuli. This analysis does not provide a formal consolidation of the data. The data were for each of the subjects were compared and found to yield similar results. A typical subject of the six tested is elaborated in this section.

Each Evoked potential time epoch (500 msec) was composed of 16625 samples. Four thousand time epochs were obtained and represented four specific responses from four unique stimuli.   A program (QB 64) derandomized these responses. Average responses and measures of central tendency were obtained for the four specific responses.

Figure 16 (below) shows the full 500 msec evoked response when the subject is presented with the "tone" and "noise" sequentially during a single stimulus. Plus/minus 2 standard errors are shown also. The entire stimulus length is 70 msec, the shown evoked response is for a 500 msec interval. The tonal stimulus (10 msec) is theoretically masked by the noise stimulus (50 msec), which is presented following a 10 msec inter-stimulus interval (silence) following the tone. The range of this response was approximately 8-20 microvolts. It should be noted that this is the "average" of the response over 1,000 trials, as described in the methodology.
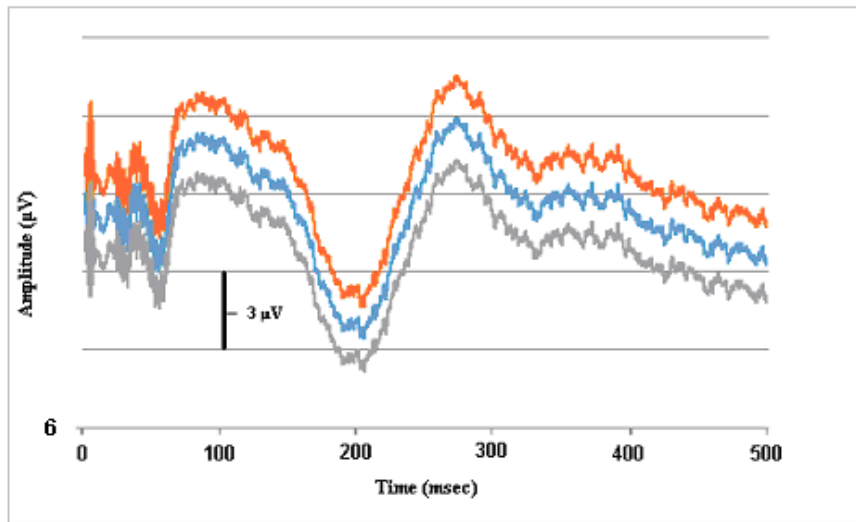
The graph in figure 17 (below) shows the 500 msec evoked response when subject is presented only with the "tone" stimulus of length 10 msec (+/- 2 standard error). The "tone alone" response measures approximately from amplitude 11-20 microvolts. This shows the early, middle and late response to the tonal stimulus of length 10 msec. This measure is primarily a reference to the principal trials of the experiment.
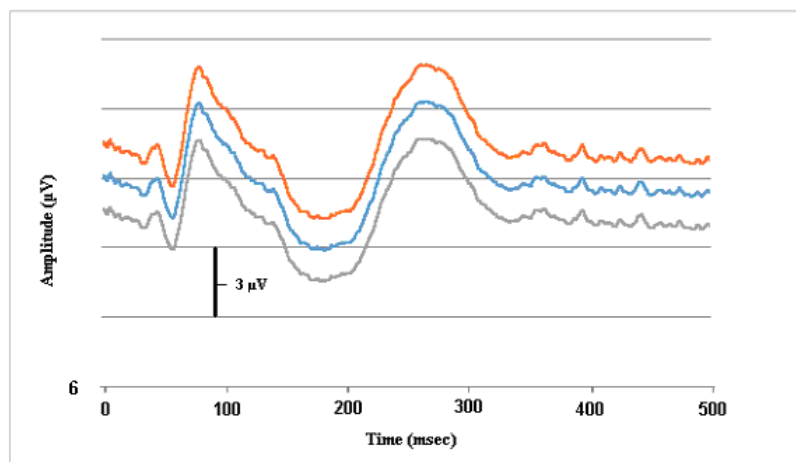
The graph in figure 18 (below) shows the 500 msec evoked response when a subject is presented with the noise alone (+/- 2 standard errors). According to the same paradigms as the "tone plus noise" as well as the "tone alone", the entire length of the stimulus was 70 msec; the "noise alone" only is present in the last 50 msec of the stimulus. Responses for the "noise alone" range in the 8-21 microvolt range. Again, this graph represents the "summation" of 1,000 individual trials.
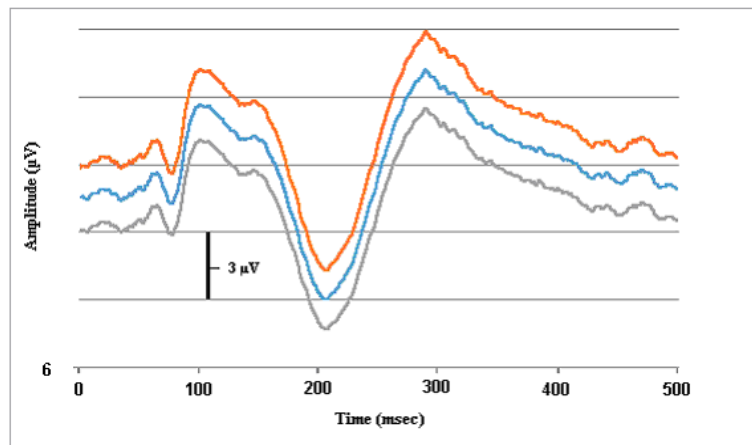

Figure 19 (below) shows the 500 msec evoked response when a subject is presented with a control paradigm of 70 msec silence (+/- 2 standard error). This response ranges in approximate amplitude 12-17 microvolts.
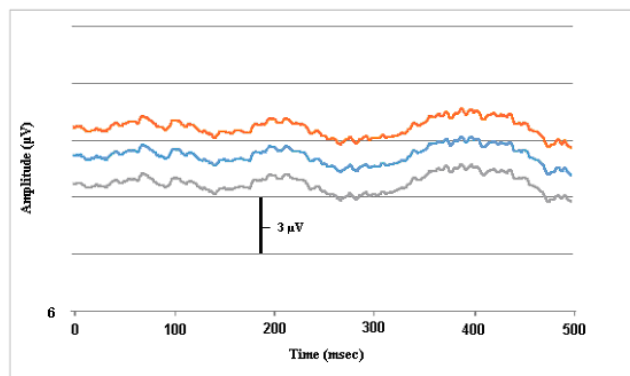
Figure 20 (below) shows the tone alone (in blue), and the derived response of the "tone plus noise" minus the "noise alone". This compares the tone alone, the response with no backward masking effect – to the theoretical tonal response acted upon by the BM effect. It should be noted that the reduction in amplitude seen in the derived response is an artifact of subtracting from the already reduced masked response.
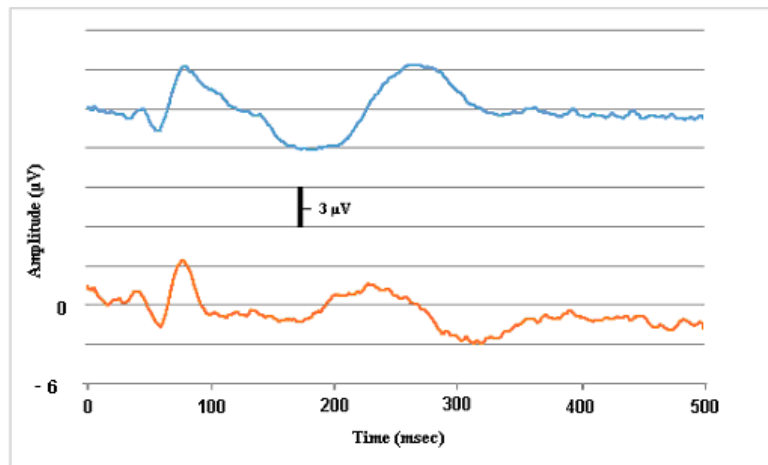

Figure 21 (below) shows the noise alone (in blue), and the "tone plus noise" minus the "tone alone. This paradigm manipulates the data in an inverse manner to the previous graph; The isolated noise is yielded through the subtraction of the tone alone from the combined "tone plus noise" stimulus.
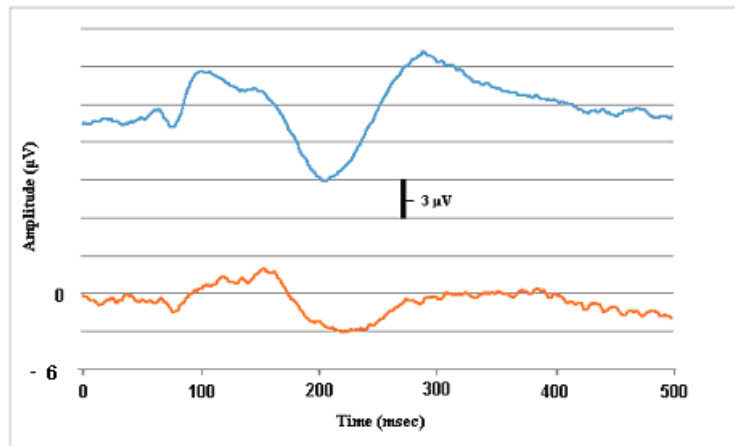
Figure 22 (below) shows a comparison of the combined "tone plus noise stimulus" to the "tone alone" stimulus summed with the "noise alone." Theoretically, if there is no backward masking effect, these two responses should be certainly comparable, if not the same altogether. Alternatively, if the BM effect is not present – the single "tone plus noise" stimulus should be no different than the sum of the isolated tone alone and noise alone. The 2nd graph is of greater relevance and similarity of morphology. The reasoning for this is that by summing the isolated tone and noise components, the baseline electrophysiological measurement is essentially doubled (regardless of any simultaneous stimuli). In other words, there is a relatively constant amplitude for an individual, stimuli that excite these responses only serve to create greater deviations around this "average" of electrophysiological activity. Thus, when the 2 waveforms are summed

together, it is proper to divide the sum by 2.
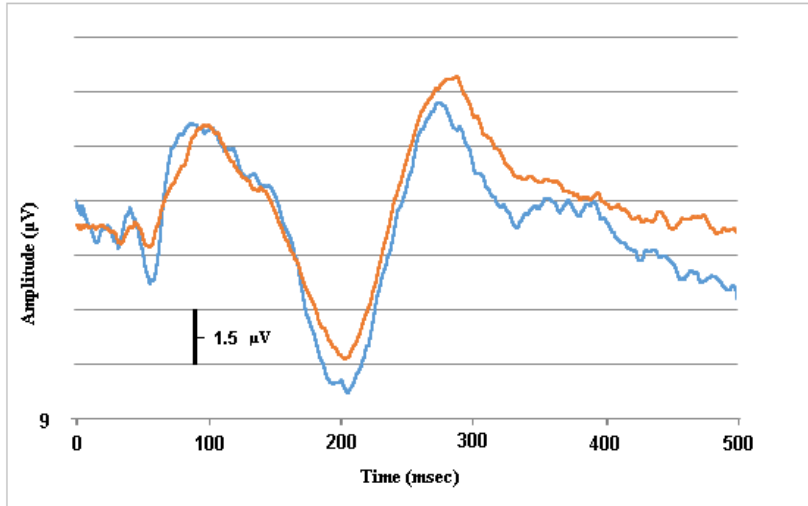
Shown in figure 23 (below) is the middle latency (200 msec) response for the 2 msec "pulse" stimuli followed by a 10 msec ISI and a 50 msec noise stimulus. This is the measured response that theoretically hosts the backward masking effect, and therefore a diminished "pulse" response. Also shown are +/- 2 standard errors.
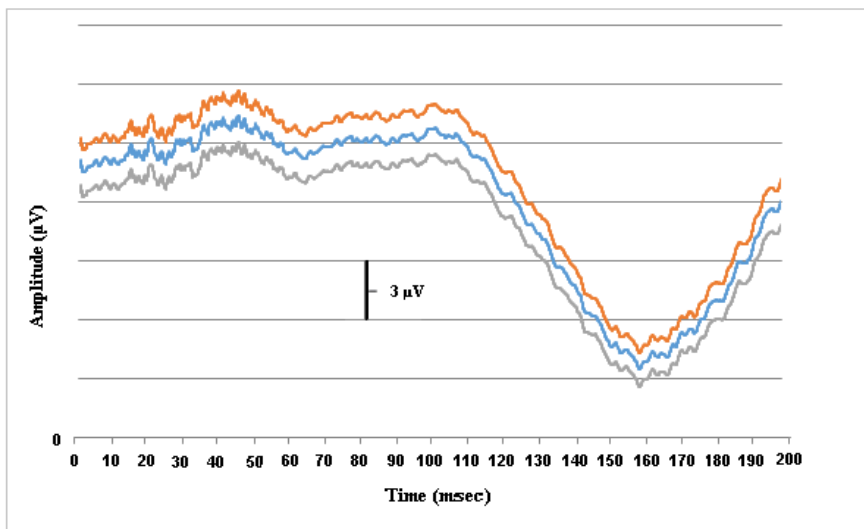
Figure 24 (below) shows the middle latency response of the pulse stimulus alone, consisting of the isolated 2 msec pulse stimuli with no noise stimulus. Also shown are +/- 2 standard errors.
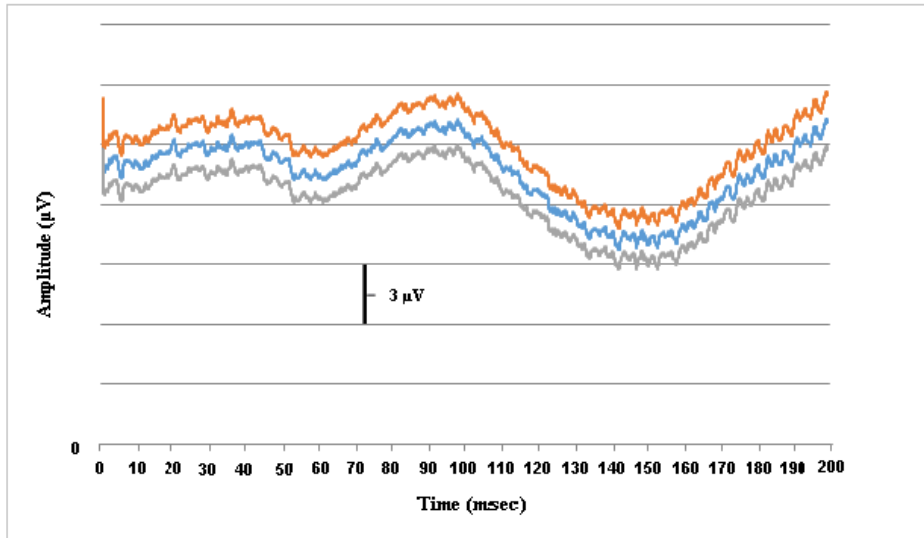

Figure 25 (below) shows the isolated noise alone stimulus, without the pulse stimuli. This graph serves primarily as a reference, to show that the evoked potential for the noise alone is indeed different than the potentials for the other stimuli.
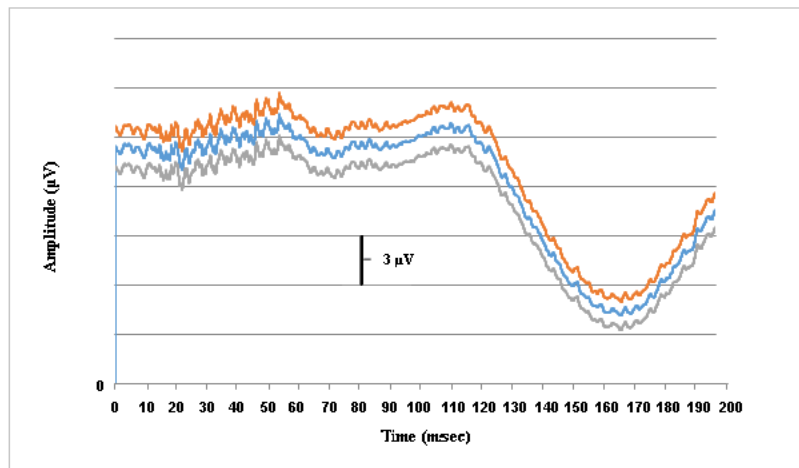
Figure 26 (below) is a control condition of the "silence" stimuli with same paradigms as the other conditions, except for the lack of any stimulus whatsoever. This represents the baseline electrophysiological response (for one individual). It should be noted that the silence paradigms for the tonal and pulse responses are markedly similar (even among different subjects) which confirms the validity of this experiment.
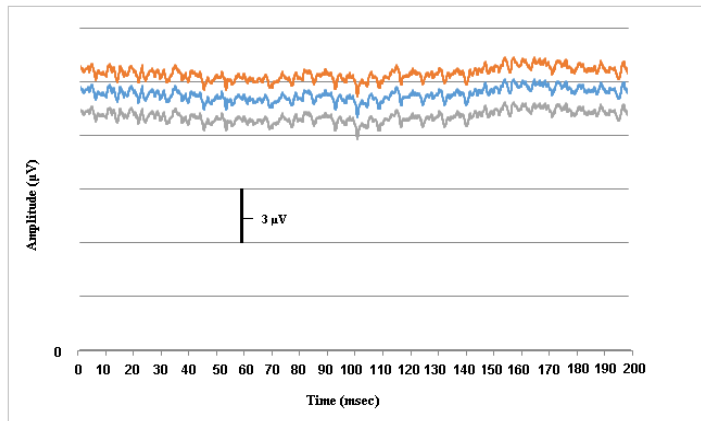
Shown in figure 27 (below) is the late latency (500 msec) response for the 2 msec "pulse" stimuli followed by a 10 msec ISI and a 50 msec noise stimulus. This is the entire course of the measured response that theoretically hosts the backward masking effect, and therefore a diminished tonal response. Also shown are +/- 2 standard errors.

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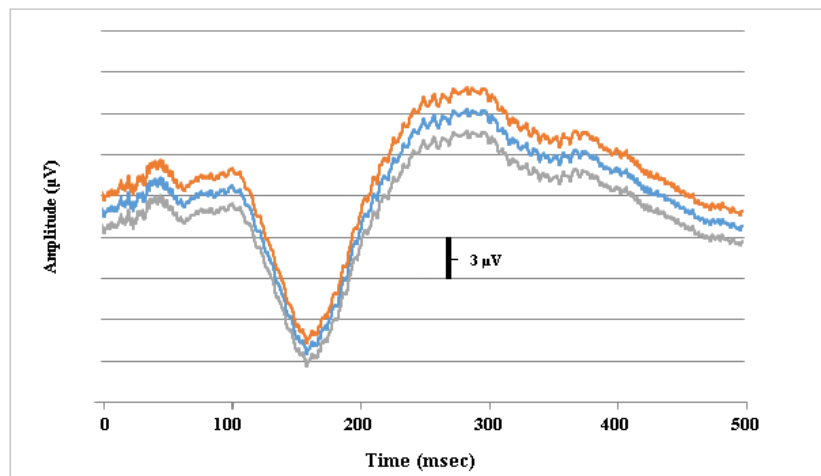Figure 28 (below) shows the late latency response of the pulse stimulus alone, consisting of the isolated 2 msec pulse stimuli with no noise stimulus. Also shown are +/- 2 standard errors.
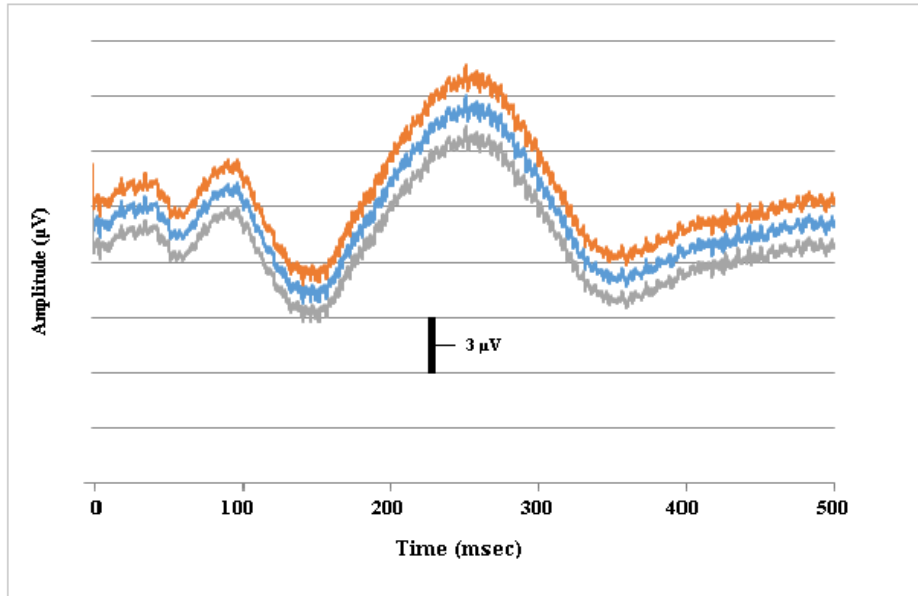

Figure 29 (below) shows the isolated noise alone stimulus, without the pulse stimuli. This graph serves mostly as a reference, to show that the evoked potential for the noise alone is indeed different than the potentials for the other stimuli.
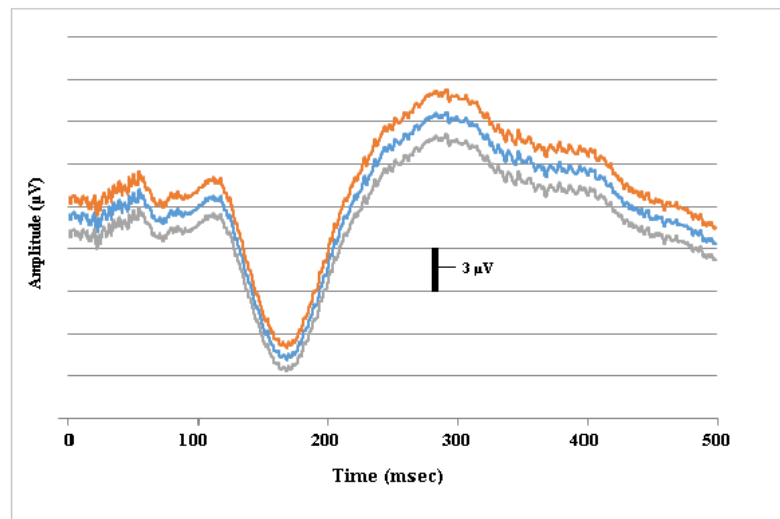
Figure 30 (below) is a control condition of the "silence" stimuli with same paradigms as the other conditions, except for the lack of any stimulus whatsoever. This represents the baseline electrophysiological response (for one individual). It should be noted that the silence paradigms for the tonal and pulse responses are markedly similar (even among different subjects) which confirms the validity of this experiment.

Figure 31 (below) compares the noise alone response, and a "derived" response that consists of the "pulse plus noise" minus the "pulse alone." By performing this subtraction, the evoked response to the noise alone is theoretically yielded. It should be noted that the reduction in amplitude seen in the derived response is an artifact of subtracting from the already reduced masked response.
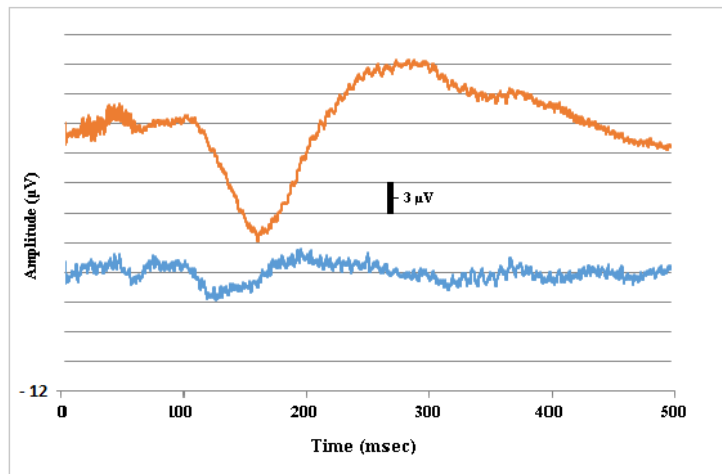
Figure 32 (below):

Compared here are the noise alone response, and a "derived" response that consists of the "pulse plus noise" minus the "noise alone." By perf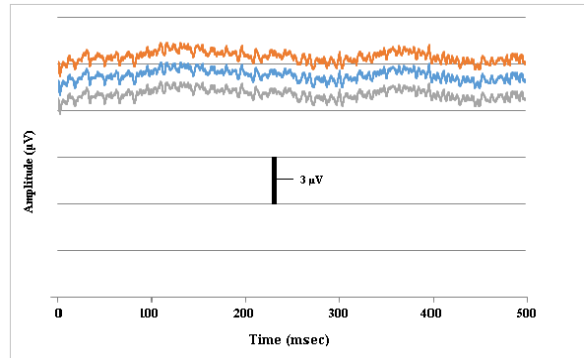orming this subtraction, the evoked response to the noise alone is theoretically yielded. It should be noted that the reduction in amplitude seen in the derived response is an artifact of subtracting from the already reduced masked response.

This paradigm yields the significance of the pulse alone, when compared with the derived response of the "pulse plus noise" minus the noise alone. By manipulating these responses, the backward masking effect is brought forth. The pulse plus noise (the masked stimulus) is left when the noise response is subtracted. The effect of the noise stimuli is left residual while it's actual evoked response is subtracted. This graph therefore compares the "unmasked" pulse alone – to the "masked" pulse plus noise.
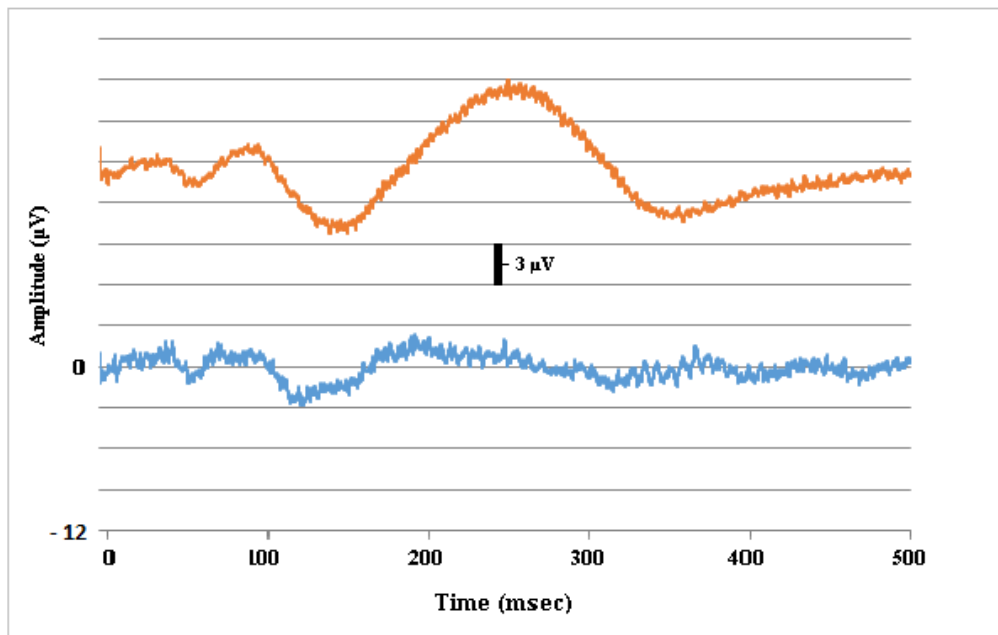
Figure 33 (below) shows the derived response consisting of the pulse plus noise minus the noise

Alone for the middle latency response (200 msec). This graph represents the 2 msec pulse

response when acted upon by the backward masking effect compared to the pulse alone (with no

noise contributing to the backward masking effect.
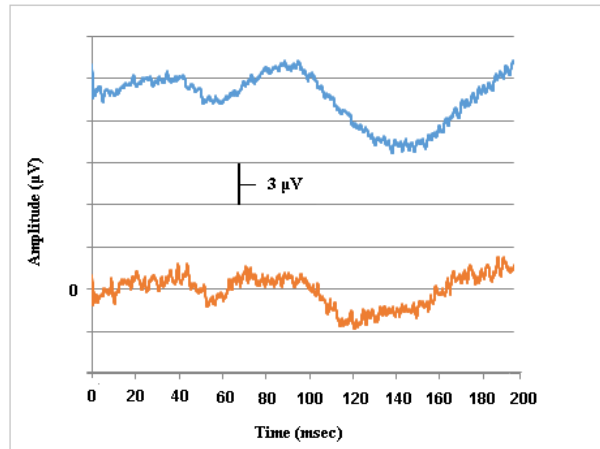
Figure 34 (below) shows the derived noise effect compared to the isolated noise effect.

This is a control condition to prove that the response for the noise alone stimulus is different

from the same stimulus when combined with others, and indeed it is.
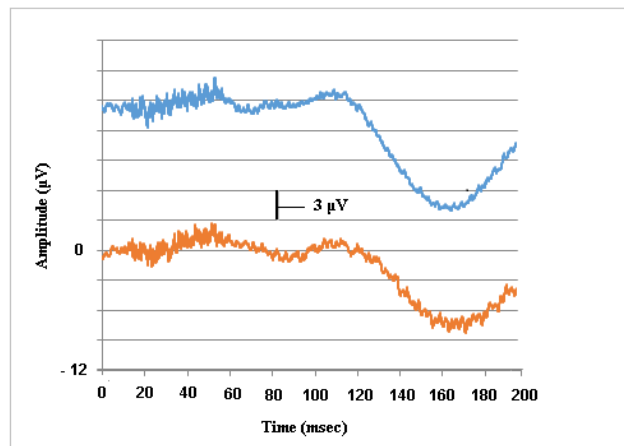
Shown in figure 35 (below) is the early latency (20 msec) response for the 2 msec "pulse" stimuli followed by a 10 msec ISI and a 50 msec noise stimulus. This is the early course of the measured response that theoretically hosts the backward masking effect, and therefore a diminished tonal response. Also shown are +/- 2 standard errors.
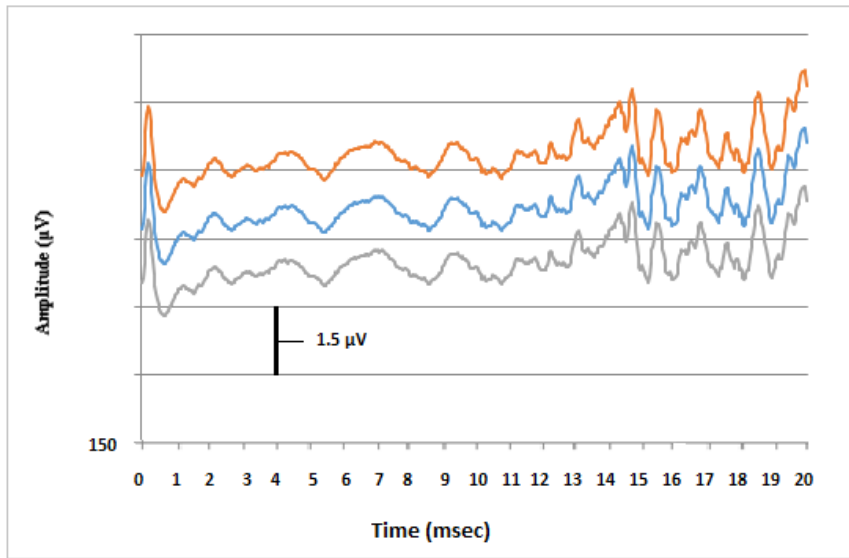
Figure 36 (below) shows the early latency response of the pulse stimulus alone, consisting of the isolated 2 msec pulse stimuli with no noise stimulus. Also shown are +/- 2 standard errors.
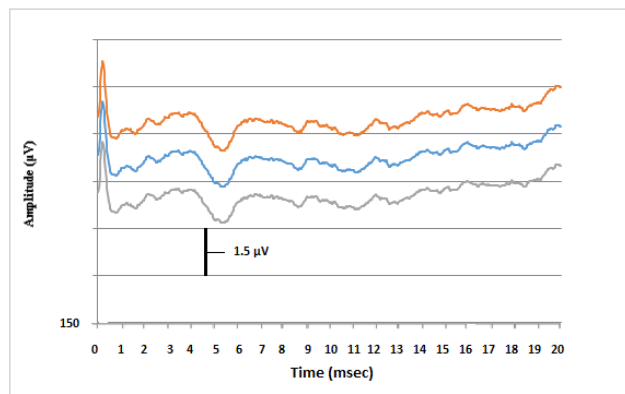
Figure 37 (below) shows the isolated noise alone stimulus, without the pulse stimuli. This graph serves mostly as a reference, to show that the evoked potential for the noise alone is indeed different than the potentials for the other stimuli.
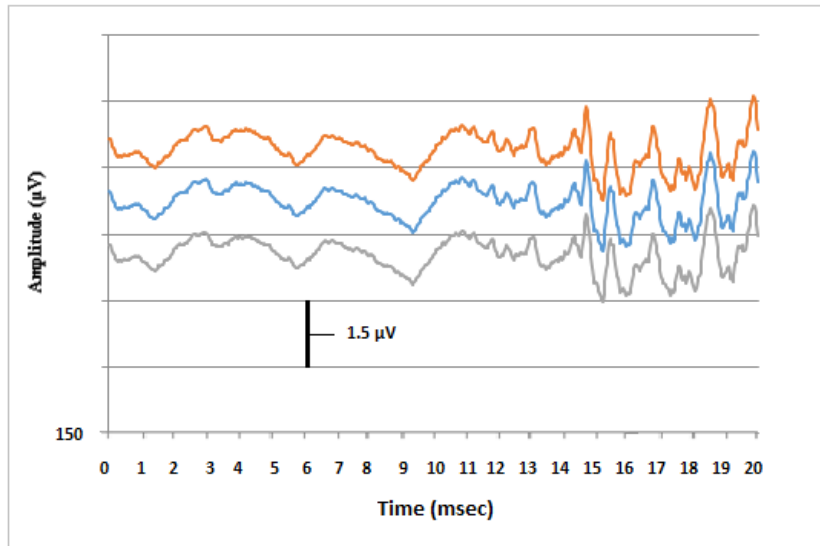
Figure 38 (below) is a control condition of the "silence" stimuli with same paradigms as the other conditions, except for the lack of any stimulus whatsoever. This represents the baseline electrophysiological response (for one individual). It should be noted that the silence paradigms for the tonal and pulse responses are markedly similar (even among different subjects).
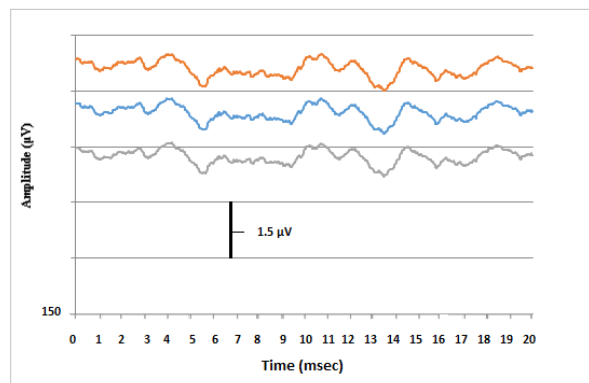
Discussion

Possibly the greatest significance of this experiment is yielded in figure 32. The peak shown approximately 250 msec is clearly present, and in fact the largest component of the morphology of the response to the pulse stimulus when presented alone. However, in the second graph, when the backward masking effect is acting upon the pulse stimulus, the peak is clearly not present. Furthermore, the second (approx. 150 msec) and third (approx. 90 msec) largest peaks are diminished in amplitude as well. In fact, the only peak not affected is the first. According to current models of the auditory system, this places the disruption at the level of the auditory cortex, and furthermore, and these disruptions continue through half of the response period attributed to the auditory cortex. Thus, according to our methods of measuring evoked potentials over 4,000 randomized trials of 4 different stimuli, through data filtering and manipulation, it appears that the backward masking effect occurs in the auditory cortex.

Figure 20 shows the backward masking effect present during the tonal paradigm. This condition consists of tone 10 msec in length, followed by an ISI of 10 msec and noise stimuli of 50 msec. The comparison of the tone plus noise response to the pulse response is fundamentally different. This makes absolute sense, in that the length of the initial stimulus is much longer. Although the ISI remains the same length, when compared these two graphs essentially show the difference in the backward masking effect when the stimulus is 2 msec vs. 10 msec. The responses in the range of 180-300 msec do show a marked difference from the tone alone condition. The response around 60 msec also is steeper when going negative. This presents a confirmation of a significant aspect of backward masking previously reported upon: that a prior

stimulus of greater amplitude and length yields a stronger response when combined with the noise stimulus.

While necessary to the experiment, the other graphs represent no contextually significant effects, and therefore do not play a great deal of relevance in a discussion. The data does suffice to say though, that backward masking occurs in a paradigm when a tone is proceeded in quick succession by a noise stimulus. It should be noted though, that the evoked potentials for the noise alone and silence stimuli followed the same morphological patterns in the pulse and tonal trials of the experiment. This confirms the validity and reliability of the study, even in different individuals.

CHAPTER SIX

Summary

Through experimentation, the effects of the backward masking yielded differential electrophysiological measurements with the manipulation of stimuli. After analysis of data, using summation techniques with over 1,000 time epochs of single-electrode recordings, we have shown the electrophysiological variations are dependent on the backward masking condition in comparison to baseline activity. The backward masking effect manifests approximately in the 90-250 msec range, with stimulus of appropriate amplitude and ISI length. According to the current models of the auditory pathway, this places the location of the disruption in midbrain to the auditory cortex. This study is the preliminary step in exploring the neurological role backward masking plays in auditory disorders, and will be further researched to hopefully yield the spectrum of knowledge from etiology to treatment of such disorders.

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APPENDIX A

SUBJECT INFORMATION AND INFORMED CONSENT

Title:
Backward Masking and Evoked Potentials

Project Directors:

Al Yonovitz, Faculty Supervisor; 32 Campus Drive, Department of Communicative Sciences and Disorders; (406) 243-2408

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Taylor Perius, Research Assistant: 32 Campus Drive, Department of Communicative Sciences and Disorders: (406) 224 2412

Special instructions:
This consent form may contain words that are new to you. If you read any words that are not clear to you, please ask the person who gave you this form to explain them to you.

Purpose:
The purpose of this study is to investigate auditory processing issues as it relates to temporal masking. The study will present to you both tone sounds and noise sounds. All of these sounds are presented at a comfortable level. This study will allow us to determine if we can measure the amount of temporal masking by measuring your brain waves.

Procedures:
You will first be given a hearing screening which will take approximately 10 minutes. If you agree to take part in this research study you will be seated in a sound-attenuated booth. Three electrodes will be placed onto your head. These are the same techniques that are used in clinical testing of brain waves. Earphones will be placed over your ears and adjusted until comfortable. Your only task is to sit quietly and listen.

Risks/Discomforts:
There is no anticipated discomfort for participating in this study, so risk to participants is minimal. You will be given breaks when needed.

Benefits:

You will receive a free hearing screening and gain the knowledge of your personal hearing thresholds. Participation may contribute to our knowledge of auditory processing disorders may result in advances in the field of Communicative Sciences and Disorders.

Confidentiality:

Your records will be kept confidential and will not be released without your consent except as required by law. You will be assigned a code number. Only the principle investigators will have access to the link between the code number and the name of the subject. This code number will be kept in a locked file cabinet. Even the data assigned to a code number will be protected. At the end of the experiment, including the publication, all identifying information will be destroyed. If the results of this study are written in a journal or presented at a meeting, your name will not be used.

Voluntary Participation/Withdrawal:

Your decision to take part in this study is entirely voluntary, and you may leave the study at any time for any reason.

Compensation for Injury:

In the event that your child is injured as a result of this research you should individually seek appropriate medical treatment. If the injury is caused by the negligence of the University of Montana or any of its employees, you may be entitled to reimbursement or compensation pursuant to the Comprehensive State Insurance Plan established by the Department of Administration under the authority of M.C.A., Title 2, Chapter 9. In the event of a claim for such injury, further information may be obtained from the University's Risk Manager (406-243-2700; kathy.krebsbach@umontana.edu) or the Office of Legal Counsel (406-243-4742; legalcounsel@umontana.edu). (Reviewed by University Legal Counsel, May 9, 2013)

Questions:

If you have any questions about the research before or after the study, contact:

Al Yonovitz (406) 243-2408
Silas Smith(360) 359-2940
Nicole Aline (406) 403-6164
Kendall Alley (406) 491-4671
Taylor Perius (206) 224-2412

If you have any questions regarding your rights as a research subject, you may contact the Chair of the IRB through the University of Montana Research Office at 243-6672.

Statement of Consent:

I have read the above description of this research study. I have been informed of the risks and benefits involved, and all my questions have been answered to my satisfaction. Furthermore, I have been assured that any future questions I may have will also be answered by a

member of the research team. I voluntarily agree to take part in this study. I understand I will receive a copy of this consent form.

_____

Printed Name of Subject

_____   _____

Subject's Signature   Date

Control Programs
Pulse Program:

```
CLS
REM Al and Silas 11/14/2015
DIM Ppn%(15625)
DIM Pa%(15625)
DIM NAP%(15625)
DIM SIL%(15625)
DIM play1%(15625)

DIM rand1a(4000)
DIM ong(615)
DIM hbyte(615)
DIM lbyte(615)
DIM hbyte1%(15625)
DIM lbyte1%(15625)
DIM val1%(15625)

OPEN "Ppncon.txt" FOR INPUT AS #3
FOR hh = 1 TO 15625
   INPUT #3, datval
   Ppn%(hh) = datval
NEXT hh
CLOSE #3

OPEN "Pacon.txt" FOR INPUT AS #3
FOR hh = 1 TO 15625
   INPUT #3, datval
   Pa%(hh) = datval
NEXT hh
CLOSE #3

OPEN "Napcon.txt" FOR INPUT AS #3
FOR hh = 1 TO 15625
   INPUT #3, datval
   NAP%(hh) = datval
NEXT hh
CLOSE #3

OPEN "SILcon.txt" FOR INPUT AS #3
FOR hh = 1 TO 15625
   INPUT #3, datval
   SIL%(hh) = datval
NEXT hh
```

```
CLOSE #3

conver = 13
conver1 = 100
trials = 4000
basead = &H220
intersample = 250000
OUT basead + 13, 0
begin:
CLS
PRINT
PRINT
PRINT "* * * * * SELECT ONE * * * * *"
PRINT
PRINT "        1) INITIATE SAVE DATA"
PRINT
PRINT "        2) SELECT NUMBER OF TRIALS"
PRINT
PRINT "        3) ONGOING EEG"
PRINT
PRINT "        4) OBTAIN EP DATA"
PRINT
PRINT "        5) EXIT (MUST EXIT)"
PRINT
GOTO keyval
GOTO begin

keyval:
a$ = INKEY$
IF a$ = "1" THEN GOTO SDATA
IF a$ = "2" THEN GOTO RANDOMSEL
IF a$ = "3" THEN GOTO ONEEG
IF a$ = "4" THEN GOTO OBDATA
IF a$ = "5" THEN GOTO QUIT
GOTO keyval

END

ONEEG:
SCREEN 2
LINE (0, 0)-(639, 0)
LINE (639, 0)-(639, 199)
LINE (639, 199)-(0, 199)
LINE (0, 199)-(0, 0)

cont:
```

```
FOR j = 0 TO 614
   OUT basead + 10, 2
   OUT basead + 12, 0
   FOR t = 1 TO conver1
   NEXT t
   hbyte(j) = INP(basead + 5)
   lbyte(j) = INP(basead + 4)
NEXT j

FOR j = 0 TO 614
   ong(j) = (((hbyte(j) AND 15) * 256) + lbyte(j)) - 2048
NEXT j

x = 10
FOR j = 0 TO 614
   PSET (x, 100 - INT(ong(j) / 21))
   x = x + 1
NEXT j
FOR t = 1 TO 20000
NEXT t
x = 10
FOR j = 0 TO 614
   PSET (x, 100 - INT(ong(j) / 21)), 0
   x = x + 1
NEXT j

a$ = INKEY$
IF a$ = "Q" OR a$ = "q" THEN GOTO subbegin
GOTO cont

subbegin:
SCREEN 0
GOTO begin

OBDATA:
CLS
FOR jtri = 1 TO trials
   REM Getting the first random number

   SIL$ = STR$(rand1a(jtri))
   sil1$ = RIGHT$(SIL$, 1)

   REM This always sets pulse alone
   sil1$ = "2"

   IF sil1$ = "1" THEN GOTO one
```

```
IF sil1$ = "2" THEN GOTO two
IF sil1$ = "3" THEN GOTO three
IF sil1$ = "4" THEN GOTO four

one:
FOR hhh = 1 TO 15625
   play1%(hhh) = Ppn%(hhh)
NEXT hhh
GOTO cont30

two:
FOR hhh = 1 TO 15625
   play1%(hhh) = Pa%(hhh)
NEXT hhh
GOTO cont30

three:
FOR hhh = 1 TO 15625
   play1%(hhh) = NAP%(hhh)
NEXT hhh
GOTO cont30

four:
FOR hhh = 1 TO 15625
   play1%(hhh) = SIL%(hhh)
NEXT hhh
GOTO cont30

cont30:

REM Let's delay
FOR del1 = 1 TO intersample
NEXT del1

FOR j = 1 TO 15625
   OUT basead + 10, 2
   OUT basead + 12, 0
   REM Send out data DAC value

   REM send out the stimulus
   OUT &H378, play1%(j)

   FOR t = 1 TO conver
   NEXT t
   hbyte1%(j) = INP(basead + 5)
   lbyte1%(j) = INP(basead + 4)
```

```basic
    NEXT j
    REM write random number to EP file
    randx% = rand1a(jtri)
    PUT #1, , randx%

    FOR j = 1 TO 15625
       val1%(j) = (((hbyte1%(j) AND 15) * 256) + lbyte1%(j)) - 2048
       PUT #1, , val1%(j)
       REM PRINT #4, val1%(j)
    NEXT j
    LOCATE 8, 30
    PRINT "                ";
    LOCATE 8, 30
    PRINT jtri, randx%;
NEXT jtri
CLOSE #1
GOTO subbegin

RANDOMSEL:
CLS
PRINT
PRINT "NUMBER OF TRIALS 1000,2000,3000,4000: ";
INPUT numtrial
IF numtrial = 1000 THEN GOTO get1000
IF numtrial = 2000 THEN GOTO get2000
IF numtrial = 3000 THEN GOTO get3000
IF numtrial = 4000 THEN GOTO get4000

get1000:
OPEN "rand1000.txt" FOR INPUT AS #2
FOR j = 1 TO 1000
   INPUT #2, dat1
   rand1a(j) = dat1
NEXT j
CLOSE #2
GOTO begin

get2000:
OPEN "rand1000.txt" FOR INPUT AS #2
FOR j = 1 TO 1000
   INPUT #2, dat1
   rand1a(j) = dat1
NEXT j
CLOSE #2
```

```
OPEN "rand2000.txt" FOR INPUT AS #2
FOR j = 1001 TO 2000
   INPUT #2, dat1
   rand1a(j) = dat1
NEXT j
CLOSE #2
GOTO begin

get3000:
OPEN "rand1000.txt" FOR INPUT AS #2
FOR j = 1 TO 1000
   INPUT #2, dat1
   rand1a(j) = dat1
NEXT j
CLOSE #2

OPEN "rand2000.txt" FOR INPUT AS #2
FOR j = 1001 TO 2000
   INPUT #2, dat1
   rand1a(j) = dat1
NEXT j
CLOSE #2

OPEN "rand3000.txt" FOR INPUT AS #2
FOR j = 2001 TO 3000
   INPUT #2, dat1
   rand1a(j) = dat1
NEXT j
CLOSE #2
GOTO begin

get4000:
OPEN "rand1000.txt" FOR INPUT AS #2
FOR j = 1 TO 1000
   INPUT #2, dat1
   rand1a(j) = dat1
NEXT j
CLOSE #2

OPEN "rand2000.txt" FOR INPUT AS #2
FOR j = 1001 TO 2000
   INPUT #2, dat1
   rand1a(j) = dat1
NEXT j
CLOSE #2
```

```
OPEN "rand3000.txt" FOR INPUT AS #2
FOR j = 2001 TO 3000
   INPUT #2, dat1
   rand1a(j) = dat1
NEXT j
CLOSE #2

OPEN "rand4000.txt" FOR INPUT AS #2
FOR j = 3001 TO 4000
   INPUT #2, dat1
   rand1a(j) = dat1
NEXT j
CLOSE #2
GOTO begin

SDATA:
CLS
PRINT "* * * * * Save Data * * * * *"
PRINT
PRINT "Enter File Name: (eg NNEP.dat";
INPUT sfilename$
OPEN sfilename$ FOR BINARY AS #1
GOTO begin

QUIT:
PRINT "QUIT"
CLOSE #1
END
```

End pulse program

Tone Program:

```
CLS
REM Al and Silas 11/14/2015
DIM TA%(15625)
DIM tpn%(15625)
DIM NA%(15625)
DIM SIL%(15625)
DIM play1%(15625)

DIM rand1a(4000)
DIM ong(615)
DIM hbyte(615)
DIM lbyte(615)
DIM hbyte1%(15625)
DIM lbyte1%(15625)
DIM val1%(15625)
```

```
OPEN "Tpncon.txt" FOR INPUT AS #3
FOR hh = 1 TO 15625
   INPUT #3, datval
   tpn%(hh) = datval
NEXT hh
CLOSE #3

OPEN "Tacon.txt" FOR INPUT AS #3
FOR hh = 1 TO 15625
   INPUT #3, datval
   TA%(hh) = datval
NEXT hh
CLOSE #3

OPEN "Nacon.txt" FOR INPUT AS #3
FOR hh = 1 TO 15625
   INPUT #3, datval
   NA%(hh) = datval
NEXT hh
CLOSE #3

OPEN "SILcon.txt" FOR INPUT AS #3
FOR hh = 1 TO 15625
   INPUT #3, datval
   SIL%(hh) = datval
NEXT hh
CLOSE #3

conver = 13
conver1 = 100
trials = 4000
basead = &H220
intersample = 250000
OUT basead + 13, 0
begin:
CLS
PRINT
PRINT
PRINT "* * * * * SELECT ONE * * * * *"
PRINT
PRINT "       1) INITIATE SAVE DATA"
PRINT
PRINT "       2) SELECT NUMBER OF TRIALS"
PRINT
PRINT "       3) ONGOING EEG"
PRINT
```

```
PRINT "        4) OBTAIN EP DATA"
PRINT
PRINT "        5) EXIT (MUST EXIT)"
PRINT
GOTO keyval
GOTO begin

keyval:
a$ = INKEY$
IF a$ = "1" THEN GOTO SDATA
IF a$ = "2" THEN GOTO RANDOMSEL
IF a$ = "3" THEN GOTO ONEEG
IF a$ = "4" THEN GOTO OBDATA
IF a$ = "5" THEN GOTO QUIT
GOTO keyval

END

ONEEG:
SCREEN 2
LINE (0, 0)-(639, 0)
LINE (639, 0)-(639, 199)
LINE (639, 199)-(0, 199)
LINE (0, 199)-(0, 0)

cont:
FOR j = 0 TO 614
   OUT basead + 10, 2
   OUT basead + 12, 0
   FOR t = 1 TO conver1
   NEXT t
   hbyte(j) = INP(basead + 5)
   lbyte(j) = INP(basead + 4)
NEXT j

FOR j = 0 TO 614
   ong(j) = (((hbyte(j) AND 15) * 256) + lbyte(j)) - 2048
NEXT j

x = 10
FOR j = 0 TO 614
   PSET (x, 100 - INT(ong(j) / 21))
   x = x + 1
NEXT j
FOR t = 1 TO 20000
NEXT t
```

```
x = 10
FOR j = 0 TO 614
   PSET (x, 100 - INT(ong(j) / 21)), 0
    x = x + 1
NEXT j

a$ = INKEY$
IF a$ = "Q" OR a$ = "q" THEN GOTO subbegin
GOTO cont

subbegin:
SCREEN 0
GOTO begin

OBDATA:
CLS
FOR jtri = 1 TO trials
   REM Getting the first random number

   SIL$ = STR$(rand1a(jtri))
   sil1$ = RIGHT$(SIL$, 1)

   REM This always sets tone alone
   sil1$ = "2"

   IF sil1$ = "1" THEN GOTO one
   IF sil1$ = "2" THEN GOTO two
   IF sil1$ = "3" THEN GOTO three
   IF sil1$ = "4" THEN GOTO four

   one:
   FOR hhh = 1 TO 15625
     play1%(hhh) = tpn%(hhh)
   NEXT hhh
   GOTO cont30

   two:
   FOR hhh = 1 TO 15625
     play1%(hhh) = TA%(hhh)
   NEXT hhh
   GOTO cont30

   three:
   FOR hhh = 1 TO 15625
     play1%(hhh) = NA%(hhh)
   NEXT hhh
```

```
    GOTO cont30

    four:
    FOR hhh = 1 TO 15625
       play1%(hhh) = SIL%(hhh)
    NEXT hhh
    GOTO cont30

    cont30:

    REM Let's delay
    FOR del1 = 1 TO intersample
    NEXT del1

    FOR j = 1 TO 15625
       OUT basead + 10, 2
       OUT basead + 12, 0
       REM Send out data DAC value

       REM send out the stimulus
       OUT &H378, play1%(j)

       FOR t = 1 TO conver
       NEXT t
       hbyte1%(j) = INP(basead + 5)
       lbyte1%(j) = INP(basead + 4)

    NEXT j
    REM write random number to EP file
    randx% = rand1a(jtri)
    PUT #1, , randx%

    FOR j = 1 TO 15625
       val1%(j) = (((hbyte1%(j) AND 15) * 256) + lbyte1%(j)) - 2048
       PUT #1, , val1%(j)
       REM PRINT #4, val1%(j)
    NEXT j
    LOCATE 8, 30
    PRINT "                ";
    LOCATE 8, 30
    PRINT jtri, randx%;
NEXT jtri
CLOSE #1
GOTO subbegin

RANDOMSEL:
```

```
CLS
PRINT
PRINT "NUMBER OF TRIALS 1000,2000,3000,4000: ";
INPUT numtrial
IF numtrial = 1000 THEN GOTO get1000
IF numtrial = 2000 THEN GOTO get2000
IF numtrial = 3000 THEN GOTO get3000
IF numtrial = 4000 THEN GOTO get4000

get1000:
OPEN "rand1000.txt" FOR INPUT AS #2
FOR j = 1 TO 1000
   INPUT #2, dat1
   rand1a(j) = dat1
NEXT j
CLOSE #2
GOTO begin

get2000:
OPEN "rand1000.txt" FOR INPUT AS #2
FOR j = 1 TO 1000
   INPUT #2, dat1
   rand1a(j) = dat1
NEXT j
CLOSE #2

OPEN "rand2000.txt" FOR INPUT AS #2
FOR j = 1001 TO 2000
   INPUT #2, dat1
   rand1a(j) = dat1
NEXT j
CLOSE #2
GOTO begin

get3000:
OPEN "rand1000.txt" FOR INPUT AS #2
FOR j = 1 TO 1000
   INPUT #2, dat1
   rand1a(j) = dat1
NEXT j
CLOSE #2

OPEN "rand2000.txt" FOR INPUT AS #2
FOR j = 1001 TO 2000
   INPUT #2, dat1
   rand1a(j) = dat1
```

```
NEXT j
CLOSE #2

OPEN "rand3000.txt" FOR INPUT AS #2
FOR j = 2001 TO 3000
   INPUT #2, dat1
   rand1a(j) = dat1
NEXT j
CLOSE #2
GOTO begin

get4000:
OPEN "rand1000.txt" FOR INPUT AS #2
FOR j = 1 TO 1000
   INPUT #2, dat1
   rand1a(j) = dat1
NEXT j
CLOSE #2

OPEN "rand2000.txt" FOR INPUT AS #2
FOR j = 1001 TO 2000
   INPUT #2, dat1
   rand1a(j) = dat1
NEXT j
CLOSE #2

OPEN "rand3000.txt" FOR INPUT AS #2
FOR j = 2001 TO 3000
   INPUT #2, dat1
   rand1a(j) = dat1
NEXT j
CLOSE #2

OPEN "rand4000.txt" FOR INPUT AS #2
FOR j = 3001 TO 4000
   INPUT #2, dat1
   rand1a(j) = dat1
NEXT j
CLOSE #2
GOTO begin

SDATA:
CLS
PRINT "* * * * * Save Data * * * * *"
PRINT
PRINT "Enter File Name: (eg NNEP.dat";
```

```
INPUT sfilename$
OPEN sfilename$ FOR BINARY AS #1
GOTO begin

QUIT:
PRINT "QUIT"
CLOSE #1
END
```