Phonemic Categorization of Eight-to-Ten Year Old Children with an Articulation Disorder

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PHONEMIC CATEGORIZATION OF EIGHT-TO-TEN YEAR OLD CHILDREN
WITH AN ARTICULATION DISORDER

by
Marjorie A. Smith

A thesis submitted to the faculty of
Brigham Young University
in partial fulfillment of the requirements for the degree of
Master of Science

Department of Communication Disorders
Brigham Young University
August 2009
This thesis has been read by each member of the following graduate committee and by majority vote has been found to be satisfactory.

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ABSTRACT

PHONEMIC CATEGORIZATION OF EIGHT-TO-TEN YEAR OLD CHILDREN WITH AN ARTICULATION DISORDER

Marjorie A. Smith
Department of Communication Disorders
Master of Science

Phonemic categorization is the ability to discriminate and organize speech sounds into categories. This ability begins soon after birth and continues to refine as an individual matures. An association between categorical labeling and phonological awareness has been reported. A strong link between perception and production has been established. The present study examined phonemic categorization of two groups of four listeners. Eight-to ten-year-old children with an articulation disorder were compared with typically speaking peers to determine if the two groups differed in their ability to categorize speech sounds. Behavioral and electrophysiological measures were used to ascertain if any differences existed. These measures were obtained in response to four stimulus pairs (/pa/-/ta/, /ta/-/ka/, /pa/-/ka/, /sa/-/ʃa/). Three of the pairs (/pa/-/ta/, /ta/-/ka/, /pa/-/ka/) differed by place of articulation only and the fourth pair (/sa/-/ʃa/)
consisted of sounds that are more commonly found in error for the age group of the participants. Behavioral data showed differences in reaction time between the two groups as well as between correct and incorrect responses. Electrophysiological data including the mismatch negativity showed that both groups perceived a distinction between the stimuli presented, but the normal control group generally displayed a higher $SD$ for peak latency and amplitude. The normal control group also generally displayed a higher mean amplitude. These results suggest a difference between the two groups in the underlying processes of phonemic categorization. Specifically, these results support that the normal control group’s ability to distinguish and categorize speech sounds is better established than that of their peers with an articulation disorder.
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As children develop speech and language, they are simultaneously developing a wide variety of cognitive processes integral to speech and language development. One of the many areas of development is termed phonemic categorization, or categorical labeling. Phonemic categorization is the ability to not only discriminate speech sounds but also to organize these speech sounds into appropriate categories on a consistent basis (Simon & Fourcin, 1978). It was noted by Pursell, Swanson, Hedrick, and Nabelek (2002) that categorical labeling becomes more refined as an individual matures. Three groups were included in their study: one group of graduate students, one group of older children, and one group of younger children. It was also reported that not all of the participants in their study displayed categorization typical of their respective age group. For example, some young children demonstrated categorization that was closer to that of older children and adults, and some older children and adults demonstrated categorization that was closer to that of younger children. Pursell et al. also reported an association between categorical labeling and phonological awareness. This was of particular import because of the role of phonological awareness in the development of literary skills.

The ability to categorize speech sounds begins almost as soon as an infant is born. This ability changes significantly by six months of age (Kuhl, Williams, Lacerda, Stevens, & Lindblom, 1992). Neonates are able to discriminate speech sounds with highly predictable patterns. This ability is not limited to their native tongue, but stretches across all speech sounds. It has been suggested that infants are born with an innate neuronal network that is preprogrammed to discriminate and categorize speech sounds (Nakisa & Plunkett, 1998).
Categorical perception is a phenomenon of both the auditory and visual systems. It has been proposed by Moore (2002) that categorical perception in neonates is a function of the brain stem. The auditory pathway of the brain stem is functionally mature at the time of birth, while the cortex has yet to undergo significant maturation. Categorical perception by the visual system was explained by Özgen (2004) using the example of a rainbow. A rainbow is a result of white light breaking into a continuum of light wavelengths within the visible spectrum. Even though a rainbow contains the entire visible spectrum, individuals group or categorize the rainbow into color classes such as red or blue.

The maturation sequence of the auditory cortex was documented by Moore (2002) as a result of examining postmortem brain tissue of individuals ranging in age from the 16th gestational week to 27 years. The information was gained from immunostaining of the postmortem brain tissue and determining the depth of mature axons within the auditory cortex. Specifically, immunolabeling of axonal neurofilaments was used, as neurofilament proliferation immediately precedes myelination. Moore used immunolabeling of axonal neurofilaments as a measure of the onset of auditory function. It was determined that mature neurons were only present within the most superficial layer of the auditory cortex up to approximately four months of age. Axons with neurofilaments began appearing in the white matter of the temporal lobe between four months and one year of age. At approximately two years of age, a light plexus of axons became apparent within the deeper layers of the cortex, which increased in density between three and five years of age. After five years of age, mature axons continued to increase in quantity within the superficial layers of the auditory cortex until
approximately 12 years of age, when the concentration of mature neurons within the auditory cortex was equivalent to that of young adults. Due to the neurons continuing to increase in density, Moore suggested that the human auditory cortex does not reach full maturity until late childhood. Given this, Moore noted a significant improvement in perceptual skills coinciding with the maturation of the auditory cortex. Hartley, Wright, Hogan, and Moore (2000) supported the idea of increasing auditory temporal resolution with age. This increase in auditory processing was noted to be steady across late childhood. Therefore, age, as well as speech and linguistic experience, should be considered in studies examining phonemic categorization of adults.

McQueen (1991) investigated lexical effects on phonetic categorization. Specifically, this was done with two separate experiments that examined categorization using a combination of words and nonwords (i.e., fish and fiss). McQueen also investigated the effect of stimulus quality. Speech categorization among adult participants was determined using responses of a forced choice between the phonemes /s/ and /ʃ/. Words were paired into the following groupings: word-nonword, nonword-word, and nonword-nonword. The stimulus words remained the same between the two experiments, but in the second experiment the stimuli were passed through a low-pass filter with a cutoff set at 3000 Hz, causing degradation of the stimulus. It was observed that the acoustic quality of the stimulus had an impact on whether or not a shift in categorization function emerged secondary to the lexical status of the stimuli. The many differences observed in phonemic categorization secondary to changes in stimuli, such as decreased acoustical information, leads to questions regarding factors influencing phonemic categorization.
Perception/Production Relationship

Another concept that must be considered regarding phonemic categorization is the relationship between speech perception and speech production. One theory regarding the correlation between speech perception and speech production is a motor theory of speech perception and production as presented by Liberman and Mattingly (1985). The motor theory consists of two main components. The first component is that objects of speech perception are intended phonetic gestures of the speaker and that these gestures are represented in the brain as motor commands for the movement of articulators. The second component is dependent on the first component, and it states that speech perception is represented as a motor command. If speech perception is represented by the same motor command as speech production, then there must be a relationship between speech perception and speech production. Liberman and Mattingly suggested that this relationship is further supported by the idea that what is heard when listening to speech represents the same motor sequences completed when speaking.

One of the main arguments against the motor theory is that speech is based on sounds and not on gestures (Ohala, 1996). Ohala maintained that individuals associate sounds with the entities that produce them without understanding how the sounds are produced. For example, an individual can identify a specific sound as being created by a siren without knowing specifically how that sound was produced.

Saltuklaroglu, Kalinowski, Dayalu, Stuart, and Rastatter (2004) provided insights into the relationship between speech perception and speech production and its connection to stuttering. Several participants in their study were able to inhibit stuttering behaviors after presentation of voluntary repetitions and before speech production. These results were obtained by using ten adults who presented with stuttering as participants in four
experimental conditions: passive pseudostuttering, passive fluent speech, active pseudostuttering, and active fluent speech. The experimental conditions were obtained by a fluent individual reading a passage that was recorded on video and was then played back to the participants within the study. The fluent individual was instructed to read using either fluent speech or pseudostuttering in the form of repetitions. The participants were then instructed to read a passage and either passively listen to the same passage read by the fluent individual or to actively attempt to imitate the behaviors displayed during the previously read passage. One thought considered by Saltuklaroglu et al. was that this effect could have been the result of fear reduction behaviors, but, based on mirror neurons’ impact on fluent imitations, they instead came to the conclusion that the effect was the result of a relationship between speech perception and speech production influenced by mirror neurons.

Mirror neurons are activated when an action is observed. These mirror neurons are located in the same areas of the cortex that would fire if the action were executed. This fact regarding the location of activation of mirror neurons has led to many theories regarding learning and mirror neurons, such as the idea that mirror neurons might further influence learning via a possible impact on imitation and mental practice (Buccino, Binkofski, & Riggio, 2004). It would follow that if mirror neurons are activated by observation, these same neurons could be activated by mental practice. It would also be anticipated that the activation of mirror neurons might positively impact performance since mirror neurons are a subset of the neurons that are activated when a task is performed.
Groenen, Maassen, and Crul (1998) investigated differences in perception as measured by identification of speech sounds between control participants and participants with misarticulations (an articulation disorder). Groenen et al. undertook two experiments using children, adolescent, and adult participants. It was observed that both misarticulating children and misarticulating adolescents displayed poorer phonetic processing, or an inability to consistently identify speech stimuli. It was reported that by shortening the duration of the transition between the second and third formants the ability to categorize the speech sounds decreased. Stimuli were created using a resynthesis procedure previously used by these authors. Groenen et al. concluded that among the participants over seven years of age with an articulation disorder, phonetic processing deficits were due to a difference of phonetic processing instead of delayed phonetic processing development. This conclusion was attributed to a lack of evidence in their study supporting a significant developmental pattern for consonant perception. This lack of a developmental pattern was present in both children with an articulation disorder and those in the control groups. Groenen et al. also suggested that, given the result of the study, normally developing children acquire perceptual abilities before speech production abilities.

Edwards (1974) investigated four hypotheses specifically related to speech perception and speech production in child phonology. The four hypotheses that were investigated included voicing distinction in stops preceding voicing distinction in fricatives, perception preceding production in the acquisition of unmarked-marked opposition (i.e., /s/ and /ʃ/), velar spirant (i.e., /x/) acquisition at the appropriate time in phonological development even though they are absent in the native language, and
phonemic perception preceding production in the acquisition of glides. The study was conducted using participants under the age of four years. Edwards reported several conclusions that provide significant insights regarding the perception-production relationship. One of the conclusions presented by Edwards was that speech perception develops gradually and with a predictable pattern. Edwards also concluded that perception of a given sound difference was generally acquired before the production of the same sound difference. For example, a child would be able to perceive a difference between the English glides before being able to produce this difference. It was also noted that the order of development for speech perception and speech production were not always identical. Edwards also concluded that phonemic perception was not completely developed at the time that the child begins to develop language, but continued to develop as the child matures.

It was suggested by Bailey and Haggard (1980) that perceptual boundaries are based primarily on acoustic information. This can have a significant impact on measuring the relationship between speech perception and speech production, as acoustic features are only present in measures of production. With the lack of a common unit of measure, there is significant difficulty in conceptually quantifying the relationship between perception and production. Bailey and Haggard reported that children who required a long VOT for identification of voiceless sounds produced voiceless phonemes with a long VOT, which supports the theory of a relationship between speech perception and speech production. These findings were based on perception and production measures from over 100 children ranging in age from 20 to 50 months, with stimuli presented both pictorially and auditorily.
**The Mismatch Negativity**

Event-related potentials (ERP) are measurable brainwaves in response to sensory stimuli, including auditory stimuli such as speech. ERPs have been shown to be a reliable way to evaluate categorization of speech sounds. Four different experiments within the same study (Maiste, Wiens, Hunt, Scherg, & Picton, 1995) investigated several aspects of using ERPs, including the mismatch negativity (MMN). The MMN is a preattentive response to a stimulus that is physically different than the preceding stimulus. It is most commonly elicited using an *oddball* paradigm where two physically different stimuli are presented with different frequencies. One stimulus, often referred to as the standard stimulus, might occur 85% of the time, while the other stimulus, referred to as the deviant stimulus, would occur 15% of the time. In an oddball paradigm the MMN occurs in response to the deviant stimulus. The four experiments evaluated attention, mismatch, continuum, and acoustic versus phonetic aspects of the stimulus used to elicit an MMN.

The first experiment was designed to evaluate the association between evoked potentials and the categorization of speech sounds made by the participants. The results indicated that the processing taking place was more complex than simple categorization. It was reported that the amplitude of the N2 wave varied similarly to reaction time, leading Maiste et al. (1995) to conclude that the N2 wave was related to the difficulty of the task, or the effort required for discrimination. Specifically, Maiste et al. reported that the amplitude of the N2 wave increased with increased reaction time.

The second experiment utilized the MMN and was designed to evaluate the waveforms of participants ignoring the stimuli in an attempt to eliminate the effects of attention on the waveforms produced. The results of the second experiment provided evidence that an MMN could be elicited by changes in speech stimuli. It should be noted
that the MMN elicited by changes in speech stimuli was smaller than the MMN typically elicited by changes in intensity or frequency. Further, the results of the second experiment did not report a categorical MMN in all cases. For example, a deviant /da/ stimulus did elicit a definite MMN, whereas a deviant /ba/ stimulus did not elicit an MMN.

The third experiment used the same protocol as the first experiment, but took place over a longer period of time in an attempt to determine a categorical boundary for the MMN response. Maiste et al. (1995) were unable to confirm or disconfirm the presence of a categorical boundary for the MMN response. It was reported that a deviant /da/ stimulus within an oddball paradigm resulted in a smaller MMN response than did a deviant /ba/ stimulus given the conditions of the third experiment. The asymmetry between the MMN elicited by a deviant /da/ stimulus and the MMN elicited by a deviant /ba/ stimulus made it very difficult to attribute the MMN strictly to categorization. If the MMN response could be attributed directly to categorization, then the same response should have been produced regardless of the deviant stimulus. One possible explanation for the difference in the MMN responses of the deviant stimulus was the acoustical difference between /ba/ and /da/.

The fourth experiment was designed to examine the difference in scalp distribution in the MMN elicited by phonetic stimuli and by a simple acoustic deviance in the same phonetic stimuli. It was noted that the MMN responses elicited by a phonetic difference were very small. Further, it was observed that the phonetic stimuli resulted in a
less pronounced MMN than did acoustically deviant stimuli. The phonetic stimuli were from different ends of the phonetic continuum (i.e., /ba/ and /da/) and the acoustically deviant stimuli varied by the intensity at which they were presented.

Generally it was noted that ERPs were adequately sensitive to match categorical differences noted by participants. It was also observed that ERPs allowed for additional information regarding categorization of speech sounds. One additional insight provided by the first and second experiments was that longer reaction times were present with stimuli near categorical boundaries.

Kraus, McGee, Sharma, Carrell, and Nicol (1992) reported that an MMN can be elicited using speech stimuli containing phonetic differences. The authors were able to attain an MMN response in children and young adults using the syllables /da/ and /ga/. The specific stimuli used were not classic exemplars of the phonemes /da/ and /ga/ but were instead more similar, decreasing the phonetic differences between them. Given the MMN responses, Krause et al. concluded that the MMN was adequately sensitive to use with speech stimuli, even with reduced phonetic differences. They concluded that the human brain is capable of detecting acoustic changes in speech stimuli that are not represented in psychometric evaluation. It is possible that this may be primarily a result of automatic detection of frequency changes based on the ability of the MMN to show perceptible changes in frequency. Therefore, the primary basis for the response to phonetic differences may be the detection of frequency changes between the stimuli, as suggested by Maiste et al. (1995).
It is preferable that participants be passively attending to stimuli during the elicitation of an MMN. Generally this is accomplished by having the participant read or watch a video during presentation of the stimuli. It has been cautioned that the task that participants undertake during the study, and the participants themselves, should be closely monitored, as some tasks such as reading or watching a video can induce sleepiness, which will affect the reliability of the MMN. Several other considerations for the elicitation and recording of the MMN were suggested. One of the considerations presented was the frequency of occurrences of deviant stimuli. Lang et al. (1995) suggested that a decreased frequency of the deviant stimulus can result in an increase in the amplitude of the MMN.

It has been noted that in children the time between the presentation of the stimulus and the ERP can exceed 300 ms, especially if long-duration stimuli are used, even though the adult latency range is typically between 80 and 250 ms. As a result, proper length of recording should be ensured when participants include children (Lang et al., 1995). Although it is preferable that participants not be paying attention to the stimuli when eliciting an MMN, it has been suggested that by having participants pay attention to the stimuli, perception can be monitored along with the evoked potentials. The additional information gained with this method may outweigh the potential influence of active processing on the MMN. Due to the influence of active processing and a possible overlap of processes, the MMN may not be as easy to discern (Maiste et al. 1995).

One consideration for the use of the MMN as a clinical and research tool is the grouping of sound processing within the brain. Although this is most closely related to the processing of tones, it is also important in the processing of speech and other auditory
stimuli. A study conducted by Shinozaki et al. (2000) noted that there were at least two processing functions within the auditory cortex, which can process information simultaneously. These results were determined following investigation of the MMN elicited in healthy adult participants presented with tones in five different conditions. The conditions included single high tones, single low tones, alternating tones with the deviant stimulus occurring 15% of the time, alternating tones with the deviant stimulus occurring 50% of the time, and overlapping tones. These authors also noted that this simultaneous processing was highly influenced and in some cases made possible by grouping tones that had a similar acoustical frequency. The results also supported the theory of needing higher levels of concentration to detect deviant stimuli in alternating conditions than in single tone conditions.

Barrett and Fulfs (1998) studied gender effects in young healthy adults between 18 and 35 years of age (10 men and 9 women). Each participant was included in both experimental and control conditions. In the experimental condition standard stimuli and deviant stimuli differed in the intensity at which they were presented. The control condition contained no deviant stimuli. Their results indicated that there was no significant effect of gender on peak latency. There was however, a significant effect on peak-to-peak amplitude and two different measures of area under the curve used in this particular study. The conclusions were that separate normative data were not necessary for males and females in relation to the MMN latency, but that separate norms should be considered when evaluating peak-to-peak amplitude and the area under the curve.

Sandridge and Boothroyd (1996) conducted an MMN study designed to evaluate the use of speech stimuli. Their study included presenting adult participants with a
combination of naturally produced speech in CV syllables and pseudovowels.
Pseudovowels were created by extracting one cycle each from the vowels /I/ and /E/ and
replicating the cycle over 200 ms. This resulted in a complex, steady-state tone with a
natural vowel spectrum. Specifically, vowel height in natural syllables, consonant place
in natural syllables, and vowel height in isolation in pseudovowels were investigated.
These authors reported that pseudovowels were the most effective in eliciting an MMN.
They observed that although the MMN was elicited using natural speech syllables, these
were less robust than those elicited by using pseudovowels. There was also less
variability among the results of the MMN elicited by pseudovowels. These authors
attributed the differences noted to acoustical differences between natural speech and
pseudovowels. This conclusion was based on a number of studies that have found that the
MMN displays adequate sensitivity to detect subtle acoustical changes, including changes
in frequency.

Sharma, Kraus, McGee, Carrell, and Nicol (1993) had results that also called into
question the usefulness of eliciting an MMN response with speech stimuli. Sharma et al.
included adult participants and investigated the MMN both across and within phonemic
categories. It was observed that the MMN was elicited in all of the participants in both
conditions. The results that they reported provide further evidence that an MMN response
is caused by the processing of acoustical information contained within the stimuli. The
most significant finding they reported supporting this conclusion was that an MMN was
consistently obtained in response to stimuli that could be identified as the same phoneme.
This response could only occur if the processing that elicits an MMN was based on small
acoustical differences, specifically those that would not be otherwise perceptible.
The present investigation used the MMN to detect the presence or absence of differences in phonemic categorization among eight- to ten-year-old children. Specifically, the investigator intended to assess whether or not differences, including differences between phoneme classes, exist between the phonemic categorization of children with an articulation disorder and typically speaking peers within this age group. The ability of participants to perceive and categorize the phonemes was assessed using a combination of the MMN and behavioral measures. A difference, either qualitative or statistically significant, between the articulation disordered group and the normal control group in measures of the MMN amplitude and latency, scalp distributions, or reaction time constituted a difference in phonemic categorization. The stop consonants /p/, /t/, and /k/ and the fricatives /s/ and /ʃ/ were used to detect the presence of differences in phonemic categorization.

**Method**

**Participants**

The current study was approved by Brigham Young University’s Institutional Review Board for human subjects. Eight participants were recruited to form two groups: one group presenting with an articulation disorder and one control group. Each group consisted of four participants between the ages of eight and ten years. Participants were recruited by contacting parents of children identified with an articulation disorder and receiving services at local elementary schools. Control group participants were recruited by contacting parents of students at local elementary schools. All participants were administered the Goldman-Fristoe Test of Articulation 2nd Edition (GFTA-2, Goldman & Fristoe, 2000) to confirm the presence or absence of an articulation disorder. The results
of the GFTA-2 were also used to gain information regarding specific errors characteristic of participants’ articulation disorders. All participants had bilateral hearing thresholds equal to or better than 25 dB HL at 250, 500, 1000, 2000, 4000, and 8000 Hz (ANSI, 1996).

**Stimuli**

A female native English speaker recorded the stimulus syllables. Stimuli were digitally recorded in a sound-isolated chamber using a low impedance dynamic microphone (DPA 4011) positioned approximately 6 inches from the speaker’s mouth. All recordings were made at 44.1 kHz with 16-bit quantization. Stimuli consisted of naturally produced speech. Voiceless stop consonants /p/, /t/, and /k/ and fricatives /s/ and /ʃ/ were used to assess phonemic categorization within and between phoneme classes.

Stop consonants and fricatives were paired with /ɑ/ to form a CV syllable. Natural speech was recorded and edited to CV syllables to reduce the likelihood of the final consonant interfering with categorization of the initial consonant. The words recorded and edited to make the CV syllables were *pot*, *tot*, *cot*, *sot*, and *shot*. Each word was recorded ten times and, following editing of the stimuli, three speech-language pathology graduate students rated each of the productions on a scale of 1 to 9, with 9 being an ideal production. The productions with the highest mean ratings were used as the experimental stimuli. No single production was rated below 5 by any rater. Two graduate students listened to the stimuli as presented to the participants. These students were asked to rate the stimuli for extraneous factors that may indicate that the stimuli were different, such as intonation or vowel length. None of the stimuli were rated higher than 3 on a 1 to 10 scale, with 1 indicating no extraneous factors present, by either graduate student. Therefore, it was
determined that there were no differences in extraneous factors, such as intonation or vowel length, between the test stimuli.

*Procedure for Electrophysiological Experiment*

Participants were comfortably seated in a sound-isolated chamber. They were asked to sit quietly and push a button to indicate whether the stimulus pairs presented were the same or different. One button was used to indicate a pair of matched stimuli and another button was used to indicate a pair of mismatched stimuli. Stimuli were presented using insert phones at 60 dB HL. The MMN was obtained by presenting a standard stimulus that occurred 85% of the time and a deviant stimulus that occurred 15% of the time using an oddball paradigm. The paired comparisons consisted of /pa/-/ta/, /ta/-/ka/, /pa/-/ka/, and /sa/-/ʃa/. Each trial consisted of 392 stimulus presentations. Each participant was presented with a series of practice trials before data were collected. The length of the practice set was determined by the participant’s understanding of the task, as evidenced by the participant’s need to be reminded to make a choice following stimulus presentation. The paired comparisons were randomized both during each participant’s trial and between participants.

Participants were fitted with a NeuroScan 32-electrode cap. Each electrode was filled with an electrolyte to reduce impedance to 10 kOhms or less. A NeuroScan computer using Scan 4.2 software was used to collect and analyze the evoked potentials. Peak latency and amplitude were recorded during analysis. The individual waveforms were used to compute grand averages that were then used to generate scalp distributions. Scalp distributions illustrating the differences between the normal control group and the articulation disordered group were created by subtracting the grand average of the normal
control group from the grand average of the articulation disordered group for each comparison. The raw electrical potentials were bandpassed from 0.05 to 70 Hz.

Results

Normal Control Group

Mismatch Negativity of Phonemic Classification

Comparisons of /pa/-/ta/, /ta/-/ka/, /pa/-/ka/, and /sa/-/ʃa/ elicited an MMN in both the normal control group and the articulation disordered group. The means and standard deviation of the normal control group is in general agreement with other studies that used the MMN elicited by phonemes (Kraus et al., 1992; Sharma et al., 1993) Table 1 shows the minimums, maximums, means, and standard deviations for the MMN amplitude and peak MMN latency for each of the four comparisons for the normal control group. Descriptive statistics for control comparisons of the same phonemes (/pa/-/pa/, /ta/-/ta/, /sa/-/sa/) were not included since an MMN was not elicited; therefore, the measures were not available for either the normal control group or the articulation disordered group. Several key differences and similarities were noted between the MMN amplitudes and MMN latencies displayed by the normal control group. The minimum MMN amplitude for the comparison of /ta/-/ka/ was approximately 6 µV lower than the lowest minimum MMN amplitude for each of the other three comparisons (/pa/-/ka/, /pa/-/ta/, /sa/-/ʃa/). The peak MMN latency for the /ta/-/ka/ comparison was nearly twice the longest maximum peak MMN latency of the remaining three comparisons (/pa/-/ka/, /pa/-/ta/, /sa/-/ʃa/). The standard deviation of the peak MMN latency for the comparison
of /tɑ/-/kɑ/ was over three times greater than the other three comparisons (/pɑ/-/kɑ/, /pɑ/-
/tɑ/, /sɑ/-/ʃɑ/). There was pronounced variability in the standard deviations of all of the
comparisons. The minimum peak MMN latency for the comparison of /sɑ/-/ʃɑ/ was
34 ms longer than the latest of the remaining three comparisons (/pɑ/-/kɑ/, /pɑ/-/tɑ/, /tɑ/-
/kɑ/). The minimum, maximum, and mean MMN amplitudes for the comparison of /pɑ/-
/kɑ/ were all similar as evidenced by a standard deviation of less than .25 µV. Likewise,
the mean MMN amplitudes for all four comparisons were similar. The minimum MMN
amplitudes of the comparisons /pɑ/-/kɑ/, /pɑ/-/tɑ/, and /sɑ/-/ʃɑ/ were also similar.

Table 1

<table>
<thead>
<tr>
<th>Comparison</th>
<th>Measurement</th>
<th>Minimum</th>
<th>Maximum</th>
<th>Mean</th>
<th>SD</th>
</tr>
</thead>
<tbody>
<tr>
<td>/pɑ/-/kɑ/</td>
<td>Amplitude (µV)</td>
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<td>10.89</td>
<td>10.72</td>
<td>.23</td>
</tr>
<tr>
<td></td>
<td>Latency (ms)</td>
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<td>156.00</td>
<td>136.00</td>
<td>28.28</td>
</tr>
<tr>
<td>/pɑ/-/tɑ/</td>
<td>Amplitude (µV)</td>
<td>7.91</td>
<td>17.94</td>
<td>11.40</td>
<td>5.67</td>
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<tr>
<td></td>
<td>Latency (ms)</td>
<td>130.00</td>
<td>150.00</td>
<td>137.33</td>
<td>11.02</td>
</tr>
<tr>
<td>/tɑ/-/kɑ/</td>
<td>Amplitude (µV)</td>
<td>1.38</td>
<td>28.61</td>
<td>11.92</td>
<td>14.62</td>
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<tr>
<td></td>
<td>Latency (ms)</td>
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<td>306.00</td>
<td>186.00</td>
<td>104.23</td>
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<tr>
<td>/sɑ/-/ʃɑ/</td>
<td>Amplitude (µV)</td>
<td>8.84</td>
<td>10.80</td>
<td>9.82</td>
<td>1.38</td>
</tr>
<tr>
<td></td>
<td>Latency (ms)</td>
<td>206.00</td>
<td>208.00</td>
<td>207.00</td>
<td>1.41</td>
</tr>
</tbody>
</table>
Scalp Distribution Maps

The grand averages for each group and each comparison were analyzed using scalp distributions. These averages were represented by a scalp distribution map as well as the differences between groups within each comparison. The comparisons represented by the maps were /pɑ/-/pɑ/, /pɑ/-/ka/, /pɑ/-/ta/, /ta/-/ka/, and /sa/-/ʃa/. The grand average difference files for each condition were used to create inter-participant difference files by subtracting the normal control group from the articulation disordered group for each comparison condition. Negativity on an inter-participant difference scalp distribution indicated that the normal control group had greatest influence, whereas positivity indicated the articulation disordered group had greatest influence. Each figure contains 25 maps representing 1000 ms after presentation of the stimuli in 40 ms increments.

Normal control group, /pɑ/-/pɑ/ control comparison. Figure 1 illustrates minimal activity differences throughout the scalp distribution for this recording. However, some slight frontal positivity occurred starting at 360 ms, continuing through 480 ms. Also, a slight negativity occurred over the right temporal region between 480 ms and 520 ms. Some negative activity also occurred over the temporal region intermittently from 600 ms to 840 ms, but this negativity was barely above background activity.

Normal control group, /pɑ/-/ka/ comparison. Figure 2 illustrates strong positive activity starting at 280 ms and diminishing by 360 ms. This positivity was most pronounced between 260 ms and 320 ms and occurred over the central parietal and temporal regions, but was strongest over the left hemisphere. Concurrently, some negative activity was seen over the left temporal region. Slight positive activity was also
Figure 1. Scalp distribution of the normal control group for the /pa/-/pa/ control comparison.
Figure 2. Scalp distribution of the normal control group for the /pa/-/ka/ comparison.
present over the left temporal occipital region between 360 ms and 480 ms. A slight positivity was seen over the central frontal region starting at 400 ms and diminishing by 520 ms. This positivity was strongest between 480 ms and 520 ms. Negative activity began over the central parietal region at 400 ms and continued to increase in area to include the temporal region and increase in amplitude until reaching its maximum amplitude between 520 ms and 560 ms. This negativity was generally central but appeared over a slightly larger area of the right hemisphere at its maximum amplitude. This central negativity diminished by 680 ms. Starting at 640 ms, a small temporal area displayed positivity that was not present after 800 ms. Between 720 ms and 840 ms, frontal negativity occurred. Some slight temporal frontal negativity occurred between 840 ms and 960 ms, which was almost imperceptible between 880 ms and 920 ms. Stronger temporal frontal negativity occurred between 960 ms and 1000 ms.

Normal control group, /pa/-/ta/ comparison. Figure 3 illustrates background activity until 280 ms. A slight to moderate positivity occurred over the central parietal region between 280 ms and 320 ms. Negative activity started at 360 ms and increased in amplitude and area until 440 ms. The negativity was generally over the central parietal to temporal areas, but was slightly stronger over the left hemisphere until 440 ms, when it began demonstrating increased amplitude over the right hemisphere. After reaching its maximum amplitude at 440 ms, the negativity decreased and was no longer present at 600 ms. Starting at 600 ms, positive activity was present over the left central parietal region until 800 ms. The positivity reached its maximum amplitude and area between 680 ms and 760 ms. Some very slight positive activity was seen over the central parietal
Figure 3. Scalp distribution of the normal control group for the /pa/-/ta/ comparison.
region between 800 ms and 960 ms as well as over the right temporal region starting at 840 ms, reaching its greatest amplitude between 960 ms and 1000 ms.

*Normal control group, /tə/-/kə/ comparison.* Figure 4 illustrates an area of negativity over the right temporal occipital area starting at 200 ms. Beginning at 240 ms this negativity was seen over both the right and left temporal occipital areas and was present until 1000 ms. Between 240 ms and 360 ms, and again between 480 ms and 600 ms, this negative activity was also seen over the right and left temporal regions. A slight to moderate central parietal positivity was seen starting at 240 ms, diminishing completely by 320 ms. This positivity was primarily over the left hemisphere and reached its maximum amplitude between 280 ms and 320 ms. An area of negative activity was seen over the right frontal parietal area at 320 ms and increased in area and amplitude to include the right and left central parietal regions, diminishing by 480 ms. A moderate to strong negativity was seen starting over the right temporal region at 480 ms and included large areas over the left temporal region starting at 520 ms. This negativity diminished completely by 680 ms. Starting at 600 ms, an area of negativity was seen over the left temporal frontal area. This negativity increased in amplitude and then decreased slightly in amplitude at approximately 680 ms, subsequently increasing in area to cover most of the frontal region and again increased in amplitude, diminishing by 1000 ms. A small area of positivity started developing at approximately 840 ms over the central posterior parietal region and increased in area and amplitude until 1000 ms.

*Normal control group, /sə/-/fə/ comparison.* Figure 5 illustrates negative activity starting at 200 ms and continuing at varying amplitudes until 1000 ms over the right posterior temporal area, occasionally occurring over the right occipital area as well.
Figure 4. Scalp distribution of the normal control group for the /\textipa{t}/-/\textipa{k}/ comparison.
Figure 5. Scalp distribution of the normal control group for the /sɑ/-/ʃɑ/ comparison.
Central right frontal positive activity was seen between 280 ms and 400 ms. A very small area of positivity occurred over the right temporal region between 360 ms and 400 ms. At 320 ms, negative activity started over the left central temporal parietal area, and it continued to increase in amplitude and area to include most of the right and left temporal and parietal areas until 560 ms. The strongest and most widespread negativity was seen between 480 ms and 560 ms. Positive activity started over the left posterior frontal area and increased in amplitude and area to include the right and left frontal areas until 840 ms. Negative activity was seen starting over the left frontal region at 880 ms and increased in area to include most of the anterior half of the scalp by 1000 ms.

Articulation Disordered Group

Mismatch Negativity of Phonemic Classification

Table 2 shows the minimums, maximums, means, and standard deviations for the MMN amplitude and peak MMN latency for the articulation disordered group for each of the four comparisons. A number of similarities and differences were noted between each comparison. The minimum MMN amplitude (-1.97 µV) for the comparison of /pa/-/ka/ was lower than any of the other minimum MMN amplitudes, and the maximum MMN amplitude was approximately twice that of the next highest maximum MMN amplitude. The standard deviations of the MMN amplitude and peak MMN latency for the comparison of /pa/-/ka/ were greater than the standard deviations of the remaining three comparisons (/pa/-/ta/, /ta/-/ka/, /sa/-/ʃa/). The minimum, maximum, and mean for the peak MMN latency measures were similar for the comparison /pa/-/ta/ as well as the comparison /sa/-/ʃa/. This similarity was most pronounced for the comparison of /sa/-/ʃa/.
The MMN amplitude measures for the /sæ/-/ʃæ/ comparison were also similar as evidenced by a standard deviation lower than .5 µV. The standard deviations for the MMN amplitude and the peak MMN latency for the comparison of /sæ/-/ʃæ/ were small when compared to those of the other three comparisons (/pæ/-/kæ/, /pæ/-/tæ/, /tæ/-/kæ/).

Table 2

<table>
<thead>
<tr>
<th>Comparison</th>
<th>Measurement</th>
<th>Minimum</th>
<th>Maximum</th>
<th>Mean</th>
<th>SD</th>
</tr>
</thead>
<tbody>
<tr>
<td>/pæ/-/kæ/</td>
<td>Amplitude (µV)</td>
<td>-1.97</td>
<td>29.48</td>
<td>11.90</td>
<td>13.31</td>
</tr>
<tr>
<td></td>
<td>Latency (ms)</td>
<td>92.00</td>
<td>212.00</td>
<td>140.50</td>
<td>50.92</td>
</tr>
<tr>
<td>/pæ/-/tæ/</td>
<td>Amplitude (µV)</td>
<td>5.09</td>
<td>8.61</td>
<td>7.38</td>
<td>1.98</td>
</tr>
<tr>
<td></td>
<td>Latency (ms)</td>
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<td>164.00</td>
<td>150.67</td>
<td>11.72</td>
</tr>
<tr>
<td>/tæ/-/kæ/</td>
<td>Amplitude (µV)</td>
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<td>4.84</td>
</tr>
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<td>158.00</td>
<td>120.00</td>
<td>33.65</td>
</tr>
<tr>
<td>/sæ/-/ʃæ/</td>
<td>Amplitude (µV)</td>
<td>6.65</td>
<td>7.29</td>
<td>6.97</td>
<td>.45</td>
</tr>
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<td></td>
<td>Latency (ms)</td>
<td>206.00</td>
<td>208.00</td>
<td>207.00</td>
<td>1.41</td>
</tr>
</tbody>
</table>

Scalp Distribution Maps

Articulation disordered group, /pæ/-/pæ/ control comparison. Figure 6 illustrates a slight negativity over the central parietal area starting at 440 ms, diminishing by 560 ms with the strongest negativity between 440 ms and 480 ms. A more pronounced negativity was seen over the left central frontal region between 560 ms and 960 ms. This negativity
Figure 6. Scalp distribution of the articulation disordered group for the /pa/-/pa/ control comparison.
started more over the left frontal region and moved to a more central frontal position with increased latency.

*Articulation disordered group, /pa/-/ka/ comparison.* Figure 7 illustrates a slight to strong left central frontal negativity between 160 ms and 400 ms and also between 600 ms and 1000 ms. Central parietal negativity occurred starting at 160 ms, increasing in amplitude and area to include the left temporal region. This negative activity diminished by 240 ms. Slight positive activity was present over the right temporal region between 240 ms and 260 ms. A small right temporal parietal area displayed slight positivity between 360 ms and 400 ms. An area over the left temporal region and a small area over the right parietal area were affected by negative activity between 400 ms and 440 ms. Starting at 520 ms, slight to strong negativity was displayed over the right and left temporal parietal regions. This negative activity diminished by 720 ms. A slight to moderate negativity occurred over the central parietal area beginning at 760 ms, diminishing by 920 ms. Slight right temporal negative activity was seen between 640 ms and 760 ms. Right temporal positive activity occurred between 960 ms and 1000 ms.

*Articulation disordered group, /pa/-/ta/ comparison.* Figure 8 illustrates slight negative activity between 200 ms and 280 ms over the central parietal area. Very slight negative activity was also seen at various places over the central parietal area between 280 ms and 400 ms. Minimal negative activity started to develop over the central parietal region at 400 ms and increased in area to include the occipital area until diminishing completely by 560 ms. An area of negativity began developing over the left central area at 280 ms and continued to increase in area and amplitude until 1000 ms. Positive activity was present over the left temporal occipital area starting at 480 ms, diminishing by
Figure 7. Scalp distribution of the articulation disordered group for the /pa/-/ka/ comparison.
Figure 8. Scalp distribution of the articulation disordered group for the /pa/-/ta/ comparison.
760 ms. A slightly stronger positivity was present over the right temporal occipital area starting at 640 ms, completely diminishing by 880 ms. Positive activity was also present over the central frontal parietal area between 680 ms and 760 ms. Between 840 ms and 880 ms, positive activity was present over the central parietal occipital area. A slight negativity also occurred over the left occipital area between 920 ms and 1000 ms.

\textit{Articulation disordered group, /t\aa/-/ka/ comparison.} Figure 9 illustrated positive activity which was barely perceptible between 160 ms and 200 ms over the right and left occipital regions. Between 280 ms and 300 ms, moderately strong positive activity occurred over the left central parietal region and slight positive activity stretched up to the central frontal area, diminishing by 360 ms. Negative activity appeared at 360 ms over the right central parietal region and increased in amplitude and area to include most of the right and left central parietal regions, diminishing by 440 ms. Also, starting at 360 ms, a small area of positivity occurred over the posterior left temporal area between 480 ms and 600 ms. An area of strong positivity also occurred over the right central parietal area between 520 ms and 720 ms, with the strongest positivity between 520 ms and 600 ms. Positive activity started over the left central occipital region at 560 ms and continued to 1000 ms, but it was not seen between 680 ms and 760 ms. This positivity increased in amplitude and diminished between 560 ms and 680 ms. It again increased in amplitude between 760 ms and 1000 ms, reaching its maximum amplitude and area between 960 ms and 1000 ms. Positive activity was seen over the right posterior temporal region between 600 ms and 800 ms.

\textit{Articulation disordered group, /s\aa/-/f\aa/ comparison.} Figure 10 illustrates slight broad positive activity over the central parietal region between 80 ms and 280 ms. A
Figure 9. Scalp distribution of the articulation disordered group for the /ta/-/ka/ comparison.
Figure 10. Scalp distribution of the articulation disordered group for the /sa/-/ʃa/ comparison.
small area of positivity was present over the right frontal area between 80 ms and 200 ms and again between 280 ms and 400 ms. Slight positivity was present over the right occipital area between 280 ms and 320 ms. Minimal negative activity was also present over the left temporal area between 400 ms and 440 ms. Moderate to strong positive activity was present over the left central frontal area between 440 ms and 640 ms. Negative activity was seen beginning at 400 ms, which was over the right anterior parietal region which continued to increase in area and amplitude through 720 ms. Although this negativity did increase in area to include the left hemisphere, it was most prominent over the right hemisphere. Starting at 680 ms, left frontal negativity was seen. This negative activity reached its maximum amplitude between 840 ms and 880 ms and remained active through 1000 ms. Strong positive activity was present over the anterior central parietal area between 720 ms and 800 ms. Very slight negative activity occurred over approximately the same area between 840 ms and 880 ms. Slight negativity was also present over the left temporal occipital region between 880 ms and 920 ms. An area of negativity began developing over the right temporal occipital region at 840 ms and continued to increase in amplitude through 1000 ms.

*Differences Between the Normal Control Group and the Articulation Disordered Group*

*Mismatch Negativity of Phonemic Classification*

Differences were apparent between the normal control group and the articulation disordered group for the comparisons of different phonemes (see Tables 1 and 2). The minimum MMN amplitude resulting from the comparison of /pɑ/ - /kɑ/ for the normal control group was 10.56 µV, as compared to -1.97 µV for the articulation disordered group. Interestingly, this difference did not occur between the maximum MMN
amplitudes of the two groups. There were also several differences in the evoked potentials for the comparison of /pa/-/ta/. The normal control group had a maximum MMN amplitude of 17.94 µV while the articulation disordered group displayed a maximum MMN amplitude of 8.61 µV. Although there were some differences in the peak MMN latency values of the two groups, the peak MMN latency standard deviations for the two groups differed by less than 1 ms. Several differences appeared between the two groups for the comparison of /ta/-/ka/. Of note, the standard deviations of the normal control group and the articulation disordered group were different for all measures. The most pronounced difference was seen between the standard deviations for peak MMN latency. The differences between the two groups were slightly less pronounced for the comparison of /sa/-/ʃa/. It should be noted, however, that the standard deviations were different, indicating more variability in the normal control group, which showed greater variability across all measures.

Scalp Distribution Maps

Comparison of normal control group and articulation disordered group, /pa/-/pa/
control comparison. Figure 11 illustrates that neither group exhibited any pronounced positive activity, and the positive activity that was displayed was generally either short in duration or very localized. The normal control group displayed weaker negative activity in general as compared to the articulation disordered group. Of particular note was the moderately strong left and central frontal negativity displayed by the articulation disordered group, which was not seen in the normal control group.
Figure 11. Scalp distribution of the comparison of the normal control group and articulation disordered group for the /pa/-/pa/ control comparison.
Comparison of normal control group and articulation disordered group, /pa/-/ka/ comparison. Figure 12 illustrates that the scalp distributions for the /pa/-/ka/ comparison were similar in that both groups displayed moderately large areas of negative activity over similar areas between approximately 400 ms and 680 ms. Although both groups displayed similar negative activity, it was noticeably stronger in the normal control group. The normal control group did display some negative activity, which was generally over much smaller areas of the frontal cortex and was only intermittently present between 600 ms and 1000 ms. The articulation disordered group displayed strong left central frontal negativity between 160 ms and 400 ms and again from 600 ms to 1000 ms. Overall, the normal control group displayed more instances of positive activity occurring over the left temporal occipital region than the articulation disordered group. The articulation disordered group did have instances of temporal occipital positivity, but these instances of positivity were less numerous than those in the normal control group and occurred over the right hemisphere. The normal control group displayed strong central positivity between 280 ms and 320 ms, during which time the only activity displayed by the articulation disordered group was negative. The articulation disordered group displayed a moderate central positivity between 800 ms and 880 ms; however, this was not observed in the normal control group.

Comparison of normal control group and articulation disordered group, /pa/-/ta/ comparison. Figure 13 illustrates similar early negative activity by both the normal control group and the articulation disordered group. Although this negativity was similar in both groups, the negativity started approximately 160 ms earlier in the articulation
Figure 12. Scalp distribution of the comparison of the normal control group and the articulation disordered group for the /pa/-/ka/ comparison.
Figure 13. Scalp distribution of the comparison of the normal control group and the articulation disordered group for the /pa/-/ta/ comparison.
disordered group was stronger, but it covered a greater area in the normal control group. The normal control group displayed moderate central positivity between 280 ms and 320 ms, during which time the only activity present in the articulation disordered group was negative. The normal control group also displayed slight positive activity over the left temporal occipital area at the beginning of the broad negativity, while the articulation disordered group did not display positive activity in this area until much later. Positive activity over the left temporal occipital region developed in the articulation disordered group starting at 560 ms, with similar activity appearing in the normal control group starting at 600 ms. This positivity was stronger in the normal control group and diminished earlier than in the articulation disordered group. Both groups also displayed central positivity between approximately 640 ms and 800 ms, but this positivity was more widespread and stronger in the normal control group. Negative activity occurred over the left frontal region starting at 280 ms and continuing through 1000 ms in the articulation disordered group, whereas negativity over this area was only present between 840 ms and 1000 ms in the normal control group, and was slightly more centrally located.

Comparison of normal control group and articulation disordered group, /ta-/ka/ comparison. Figure 14 illustrates that the articulation disordered group had a small area of slight positivity over the right and left temporal occipital regions between 160 ms and 200 ms, which was not observed in the normal control group. Starting at 200 ms, the normal control group developed a moderate to strong negativity over the right and left temporal regions, which continued until 1000 ms and was not observed in the articulation disordered group. Both groups developed broad central parietal areas of positive activity starting at approximately 240 ms, but this occurred slightly later and included more of the
Figure 14. Scalp distribution of the comparison of the normal control group and the articulation disordered group for the /ta/-/ka/ comparison.
right frontal area in the articulation disordered group. This positivity completely diminished in both groups by 360 ms. Between 360 ms and 520 ms, the articulation disordered group developed a very small left anterior temporal area of positive activity, which was not seen in the normal control group. Starting at 320 ms, the normal control group began developing negative activity at varying amplitudes over either the central parietal region or the right and left temporal regions until 680 ms. Between 360 ms and 440 ms, the articulation disordered group also displayed a central parietal negativity, but this negativity was more localized and much weaker than the negativity seen in the normal control group. Strong left to central frontal positivity developed in the articulation disordered group between 480 ms and 600 ms. Other areas of positivity were also seen in the articulation disordered group between 520 ms and 1000 ms, while there was no positive activity seen in the normal control group between 320 ms and 840 ms. Beginning at 840 ms a small area of slight to moderate positivity developed over the anterior central parietal area in the normal control group, which was similar in location to the positive activity seen in the articulation disordered group. This positive activity covered a larger area and was much stronger in the articulation disordered group. Both groups developed central frontal negative activity at varying amplitudes beginning at approximately 640 ms and ending at 1000 ms. This negativity was strongest in the normal control group between 760 ms and 880 ms, while it was strongest in the articulation disordered group between 920 ms and 1000 ms.

Comparison of normal control group and articulation disordered group, /sæ/-/ʃə/

Comparison. Figure 15 illustrates activity earlier in the articulation disordered group and consists primarily of weak positivity. Both groups developed right frontal positivity
Figure 15. Scalp distribution of the comparison of the normal control group and the articulation disordered group for the /sɑ/-/ʃɑ/ comparison.
between 280 ms and 400 ms, but this activity was stronger in the articulation disordered group. Both groups also displayed central negative activity, which was greater in area and amplitude in the normal control group, starting at approximately 320 ms. This negative activity also started and ended later in the articulation disordered group. The normal control group displayed a small right temporal occipital area of negativity between 240 ms and 720 ms, as well as from 760 ms and 1000 ms. This type of negative activity occurred in the articulation disordered group as well, but lasted for a shorter period of time and was slightly more anterior between 840 ms and 1000 ms. Both groups also displayed positive activity between approximately 680 ms and 840 ms. This positive activity started earlier and ended later in the normal control group, and was more anterior than it was in the articulation disordered group. This positivity was stronger in the articulation disordered group, but was more posterior in position and lasted for only 80 ms.

**Behavioral Results**

All of the participants included in the articulation disordered group exhibited articulation errors on /r/ (see Table 3). One of the participants also exhibited articulation errors on /s/. Although the youngest in the normal control group was one year and five months older than the youngest in the articulation disordered group, it would be anticipated that /r/ would be well established by eight years of age (Sanders, 1972). Comparisons of stop consonants were used because only placement varied from one sound to the next, even though these sounds were not in error in the articulation
disordered group. The comparison of /sa/-/fa/ was included because errors on /s/ are more common among the age range investigated.

Table 3

Description of Articulation Errors Exhibited by Participants

<table>
<thead>
<tr>
<th>Group</th>
<th>Participant</th>
<th>Age (Years:Months)</th>
<th>Gender</th>
<th>Articulation Errors</th>
</tr>
</thead>
<tbody>
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<td>Articulation Disordered</td>
<td>1</td>
<td>9:3</td>
<td>F</td>
<td>/r/ (initial [correct but exhibited increased length and stress], medial, and final), /br/, /fr/, /gr/, /kr/, /tr/</td>
</tr>
<tr>
<td></td>
<td>2</td>
<td>10:2</td>
<td>F</td>
<td>/r/ (initial, medial, and final), /br/, /dr/, /fr/, /gr/, /kr/</td>
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<tr>
<td></td>
<td>3</td>
<td>8:8</td>
<td>F</td>
<td>/r/ (initial, medial, and final), /br/, /dr/, /fr/, /gr/, /kr/, /tr/</td>
</tr>
<tr>
<td></td>
<td>6</td>
<td>8:0</td>
<td>F</td>
<td>/r/ (initial, medial, and final), /s/ (initial, medial, and final), /br/, /dr/, /fr/, /gr/, /kr/, /tr/</td>
</tr>
<tr>
<td>Normal Control</td>
<td>4</td>
<td>10:1</td>
<td>M</td>
<td>No Errors</td>
</tr>
<tr>
<td></td>
<td>5</td>
<td>10:2</td>
<td>M</td>
<td>No Errors</td>
</tr>
<tr>
<td></td>
<td>7</td>
<td>9:5</td>
<td>F</td>
<td>No Errors</td>
</tr>
<tr>
<td></td>
<td>8</td>
<td>10:7</td>
<td>F</td>
<td>No Errors</td>
</tr>
</tbody>
</table>

A one-way ANOVA was completed for reaction time and each of the comparison results. There was a significant difference in the mean reaction time for all conditions in
the articulation disordered group compared to the normal control group,

\[ F(1, 4126) = 67.65, p \leq .001 \] (see Table 4). There was a significant difference,

\[ F(1, 4036) = 105.762, p \leq .001, \]

for reaction time of correct responses in the articulation disordered group compared to the normal control group. A significant difference,

\[ F(1, 2062) = 26.046, p \leq .001, \]

was observed for the reaction time of incorrect responses compared to that of correct responses within the normal control group. A significant difference, \[ F(1, 2062) = 64.245, p \leq .001, \] was also seen for reaction time of incorrect responses.

Table 4

*Descriptive Statistics for Reaction Time in ms (n=4)*

<table>
<thead>
<tr>
<th></th>
<th>Min</th>
<th>Max</th>
<th>Mean</th>
<th>SD</th>
<th>SE</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Between Groups</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>All Conditions</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Normal Control Group</td>
<td>112.0</td>
<td>19,218.0</td>
<td>959.1</td>
<td>900.9</td>
<td>19.8</td>
</tr>
<tr>
<td>Articulation Disordered Group</td>
<td>119.0</td>
<td>61,969.0</td>
<td>1,315.8</td>
<td>1,751.9</td>
<td>38.6</td>
</tr>
<tr>
<td><strong>Correct</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Normal Control Group</td>
<td>112.0</td>
<td>15,664.0</td>
<td>950.0</td>
<td>808.9</td>
<td>17.9</td>
</tr>
<tr>
<td>Articulation Disordered Group</td>
<td>119.0</td>
<td>13,051.0</td>
<td>1,257.5</td>
<td>1,075.9</td>
<td>24.1</td>
</tr>
<tr>
<td><strong>Incorrect</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Normal Control Group</td>
<td>466.0</td>
<td>19,218.0</td>
<td>2,062.9</td>
<td>4,435.8</td>
<td>1,075.8</td>
</tr>
<tr>
<td>Articulation Disordered Group</td>
<td>211.0</td>
<td>61,969.0</td>
<td>2,905.8</td>
<td>7,299.8</td>
<td>854.4</td>
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<tr>
<td><strong>Within Groups</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Normal Control Group</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Correct</td>
<td>112.0</td>
<td>15,664.0</td>
<td>950.0</td>
<td>808.9</td>
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</tr>
<tr>
<td>Incorrect</td>
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<td>4,435.8</td>
<td>1,075.8</td>
</tr>
<tr>
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<td>854.4</td>
</tr>
</tbody>
</table>
responses compared to that of correct responses within the articulation disordered group. However, there was not a significant difference, $F(1, 88) = .208, p = .65$, between the reaction times of incorrect responses from the normal control group and those from the articulation disordered group.

**Summary of Findings**

The normal control group and the articulation disordered group varied in each of the measures used in this study to quantify phonemic categorization. In the case of the MMN latency and amplitude, differences were not only found between the normal control group and the articulation disordered group, but also between phoneme comparison groupings. Similarly, scalp distribution maps resulted in noticeable differences from one participant group to the other given the same phoneme comparison, as well as differences from one phoneme comparison to another within the same participant group. Significant differences between and within groups were present for reaction time. The one exception was reaction time for incorrect responses, which did not differ significantly between the normal control group and the articulation disordered group.

**Discussion**

The present study examined phonemic contrasts in two different groups of eight- to ten-year-old children using four conditions (/pa/-/ta/, /ta/-/ka/, /pa/-/ka/, /sa/-/ʃa/). Three of the four conditions (/pa/-/ta/, /ta/-/ka/, /pa/-/ka/) used stop consonant discrimination and the fourth condition (/sa/-/ʃa/) used discrimination of a phoneme more commonly in error among the age group investigated. Stop consonants were used because all distinctive features stay constant with the exception of place of articulation. Within
each condition an oddball paradigm was used to elicit the MMN. Behavioral
discrimination data were also collected using a button push. The data collected were
analyzed for any significant differences between children diagnosed with an articulation
disorder and typically speaking peers.

The Mismatch Negativity

Descriptive statistics of peak amplitude and latency of the MMN elicited in each
group showed a difference in the variability of each group. The normal control group
generally exhibited a higher standard deviation for both the MMN amplitude and the
MMN latency. Conversely, the articulation disordered group exhibited higher standard
deviations on all measures of reaction time. The standard deviations for the MMN
amplitude and MMN latency of the /pa/-/ka/ comparison and the MMN latency of the
/pa/-/ta/ comparison were higher in the articulation disordered group. However, the
standard deviations for the normal control group were noticeably higher than those of the
articulation disordered group. For example, the standard deviation for the MMN latency
of the /ta/-/ka/ comparison was 33.65 ms for the articulation disordered group, compared
to 104.23 ms for the normal control group. This might suggest that the underlying
processes involved in discriminating the speech sounds presented (/pa/-/ta/, /ta/-/ka/,
/pa/-/ka/, /sa/-/ʃa/) were more consistent among children in the articulation disordered
group than children in the normal control group. The variability observed among the
children in the normal control group may have resulted from the ability to make
comparisons of the stimuli presented still being established. However, the consistency
among the children in the articulation disordered group may have resulted from being unable to make a comparison between the stimuli presented.

Analysis of the mean MMN amplitude indicated a higher mean MMN amplitude in the normal control group for three of the four comparisons (/pa/-/ta/, /ta/-/ka/, /sa/-/ʃa/). This higher mean MMN amplitude in the normal control group indicates a stronger and therefore more established response to these comparisons (/pa/-/ta/, /pa/-/ka/, /sa/-/ʃa/) in the normal control group. Analysis of the mean MMN latency indicated a longer MMN latency in the articulation disordered group for three of the four comparisons (/pa/-/ta/, /ta/-/ka/, /pa/-/ka/, /sa/-/ʃa/). This longer MMN latency in the articulation disordered group may be indicative of a less established response than the response seen in the normal control group; hence, a longer processing time is required to make a response. The more established a response, the shorter the latency of the response.

Scalp Distributions

Scalp distributions (brain maps) were created to determine if the processing of the task differed across the scalp between the two groups, thus indicating different neural processes involved in the task. Careful analysis of the scalp distributions indicated that the articulation disordered group displayed frontal activity more often than the normal control group. This difference between groups may indicate that the articulation disordered group was using the frontal lobe more actively. The frontal lobe is responsible for executive functions, including decision making. Specifically, frontal lobe processing includes determining what components of stimuli should be attended to and what the motor response should be (Goldman-Rakic, 1996). The increased frontal involvement
seen in the articulation disordered group indicated increased activation of areas responsible for various aspects of decision making and attention to stimuli. Increased involvement is suggestive of less-established comparisons, as increased attention to the stimuli and/or active decision making were required by the articulation disordered group.

In the situations where the normal control group and the articulation disordered group displayed similar activity, the articulation disordered group typically had longer MMN latencies than the normal control group. This difference in the MMN latency exhibited by the articulation disordered group was accompanied by the significant difference in reaction time for all conditions in the articulation disordered group. The increase difference in reaction time may have been due to the increased cognitive processing required by the articulation disordered group to make a decision as to whether the stimuli presented were the same or different. Increased processing time was also exhibited by the articulation disordered group for the /sɑ/-/ʃɑ/ comparison, as evidenced by the mean MMN latency for the /sɑ/-/ʃɑ/ comparison. This was approximately 50 ms greater than the longest mean MMN latency of the other three comparisons (/pɑ/-/tɑ/, /tɑ/-/ka/, /pɑ/-/ka/) within the articulation disordered group. This difference in the articulation disordered group indicates increased processing for the /sɑ/-/ʃɑ/ comparison, which is of note as these sounds (/s/ and /ʃ/), particularly /s/, are in error more often than the other sounds investigated (/p/, /t/, and /k/). There was not a difference in the mean latency for the /sɑ/-/ʃɑ/ comparison in the normal control group.
Behavioral Results

Overall, the articulation disordered group exhibited increased processing time and reduced response amplitude, as evidenced by multiple measures, including the MMN latency, the MMN amplitude, and reaction time. These differences are suggestive of comparison responses that are less well established than those of the normal control group. The difference in the establishment of comparison responses between the normal control group and the articulation disordered group may indicate possible reasons why some children exhibit difficulty with sound production. It could be reasoned that in order to make a sound correctly, a child would have to be able to consistently distinguish between similar sounds. Just as important, if not more so, is the ability to make these distinctions quickly. The increased processing time exhibited by the articulation disordered group may indicate that children with an articulation disorder take longer than typically speaking peers to make this distinction. Children with an articulation disorder taking more time to make distinctions between sounds could also be seen in the increased frontal processing time exhibited by the articulation disordered group. The general findings indicate there may be a basic difference in development of responses between children with an articulation disorder and typically speaking peers when they are presented with speech stimuli.

Implications For Future Research

A limitation of the current study includes the small number of participants. That is, the participants included were not precisely age and gender matched beyond the relatively narrow age range investigated. Likewise, a broader comparison of phonemes, especially those contained within the participant’s individual error, would provide more in depth insight into this process.
Additional research using larger populations and a greater variety of age groups might better define similarities and differences between children with an articulation disorder and typically speaking peers. Future studies could provide additional information regarding specific neurophysiological and behavioral differences in phoneme distinction between children with an articulation disorder and their typically speaking peers.

Although further research is needed in this area, the current study provides information that contributes to our understanding of the possible neurological components of an articulation disorder. These findings also have the potential to assist in the future development of more quantitative means of clinical assessment of communication disorders, as well as improving the quality of services that are offered to children with an articulation disorder.
References


Journal of the Acoustical Society of America, 63(3), 925–935.
Parental Informed Consent for Child to Act as a Human Research Subject

Name of Participant: __________________________ Date of Birth: _________

**Purpose of Study**
This research is designed to examine categorization of speech sounds in children with articulation disorders using measures of electrical brain activity. Participation in this study will help teachers and scientists better understand the brain’s ability to process and categorize speech sounds and will be useful to professionals who are responsible for diagnosing and treating articulation disorders. Your child has been invited to participate in a research study conducted by graduate student, Marjorie Smith, and Dr. David L. McPherson. Your child has been invited to participate in this research study as he/she is between 8 and 10 years of age and has been diagnosed with an articulation disorder or has not been diagnosed with a speech or language disorder.

**Procedures**
Based upon these criteria your child will be assigned to one of two groups. The study will be conducted in room 111 of the John Taylor Building on the campus of Brigham Young University. The testing at BYU will consist of one 1-2 hour session including orientation and a basic hearing test. Your child may ask for a break at any time during testing.

Surface electrodes (metal discs about the size of a dime) will be used to record electrical activity of your child’s brain. These discs will be applied to the scalp with a cream or gel and are easily removed with water. Blunt needles will be used to help apply the electrode gel but will never be used to puncture the skin. Your child may feel uncomfortable using the cap as it adds slight pressure to the scalp and having gel on his or her face and head. If your child is uncomfortable, he or she will be assured that they will only have the cap on for a short period of time. If your child has a negative reaction to the electrodes, the electrodes and gel will be removed. Discomfort from the electrode cap immediately dissipates upon removal of the cap and the gel is easily removed with warm water.

The electrode cap simply measures the electrical activity of the brain and does not emit electricity. These measurements are of normal, continuous electrical activity in the brain.

Your child will wear the electrode cap while he/she listens to 2000 speech sounds and/or syllables, during which time the electrical activity of his/her brain will be recorded on a computer. Your child will be asked to give responses during parts of the test.
The procedures used to record the electrical brain activity are standardized and have been used without incident in many previous investigations. The combination of speech sounds and syllables presented is experimental, but the recording procedure is not.

**Risks**

There are very few potential risks from this procedure and all are extremely rare. The risks of this study include possible allergic reactions to the conductive gel or to the skin prepping gel. There is also a possibility for an allergic reaction to the electrodes. If any of these reactions occur, a rash would appear. Another unlikely risk is a small abrasion on the scalp when the blunt needle is used to place electrode gel. Treatment would include removing the electrode and gel and exposing the site to air, resulting alleviation of the irritation. Testing procedures would be discontinued in the event of any adverse reaction.

**Benefits**

Benefits from participating in this study include an assessment of hearing and articulation. You will be notified if any clinical deficits are found in these areas. There may be no direct benefit to you or your child. However, the information obtained will be beneficial to professionals involved in treating speech and hearing disorders.

**Confidentiality**

Participation in this study is voluntary and your child may refuse to participate or withdraw at any time. All information obtained from testing is strictly confidential and is protected under the laws governing privacy. No information specifically pertaining to your child, other than reporting of test results will be released without your signature. All identifying references will be removed and replaced by control numbers in any disclosed or published data. Data collected will be stored in a secured area accessible only to personnel associated with the study.

**Other Considerations**

There are no charges incurred for participation in this study. Your child will receive a coupon for ice cream whether or not he/she completes the study. There is no treatment or intervention involved in this study.

The procedures listed above have been explained to you and your child by: ______________________ in a satisfactory manner and any questions relating to such risks have been answered. If there are any further questions or concerns regarding this study, you may ask any of the investigators or contact Marjorie Smith, Graduate Student, Communication Disorders, 885 N. 900 E. #4, Provo, Utah 84604; phone (208) 317-6471; email: parkay2ms@msn.com or David McPherson, Ph.D., Communication Disorders, 129 Taylor Building, Provo, Utah 84602; phone (801) 422-6458; email: david_mcpherson@byu.edu.

If there are any questions regarding your rights as a participant in this research project, you may contact Christopher Dromey, PhD, Chair of Institutional Review Board, 133
I give permission for my child to participate in the study explained above.

_________________________  ____________
Signature of Parent/Guardian   Date