Mismatch Negativity responses to early-acquired sounds in children with speech sound disorders

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ABSTRACT

To examine whether children with speech sound disorders (SSD) have sparsely specified phonological representations, the present study recorded neural responses to early-acquired speech sounds in children with SSD and their typically developing controls (ages 4-6 years). Event-related brain potentials (ERPs) were recorded while children listened to speech syllables containing two early-acquired sounds: /b/ and /d/. While both the typically developing (TD) children and children with SSD demonstrated Mismatch Negativity (MMN) responses, the responses of the TD children were significantly larger. The identification of the smaller MMN responses suggests that children with SSD may have less specified phonological representations, which may impact their ability to correctly produce speech sounds. In addition, when all of the children's data were pooled together, the MMN responses were strongly correlated with measures of speech production. These results are consistent with the hypothesis that the MMN reflects acoustic-phonetic processing, which appears to be less developed in children with SSD.

Keywords

EEG; ERP; MMN; speech sound disorders; children; phonological representations

1. INTRODUCTION

When a child has a functional speech sound disorder (SSD), there is a failure by the child to use speech sounds that are age and dialect appropriate. Specifically, children with SSD have difficulty producing, using, and integrating sounds (i.e., phonemes) of the target system (Gierut, 1998). A SSD is not simply a lack of control of the speech articulators; it is an impairment in *acquiring* the phonology of a language. Generally, a SSD can affect a speaker's production and/or mental representation of speech sounds. Specifically, a SSD may be *phonetic* in nature, meaning that the difficulty lies in how sounds are produced, thereby involving a motoric component; and/or the difficulty may be *phonemic* in nature, implying that the disorder may have a cognitive or linguistic basis, thereby affecting how speech sound information is stored, represented, and retrieved in the mental lexicon (Gierut, 1998). It is presently unknown what underlying mechanisms might account for SSD. One possible explanation is that children with SSD cannot produce speech sounds correctly because they have poorly specified phonological representations in their mental lexicons, which are the result of inaccurate speech sound perception. Thus, speech sound production errors may stem from imprecise speech perception and sparse phonological representations.

1.1 Phonological representations.

The identification of phonemes through the comparison of distinctive features has been the underlying premise of phonological analysis for many decades (Chomsky & Halle, 1968; Clements & Hume, 1995; Halle, Vaux, & Wolfe, 2000; Jakobson, Fant, & Halle, 1952; Lahiri & Reetz, 2010; McCarthy, 1988). The descriptive nature of the distinctive features has changed over the years from describing the perceptual and acoustic nature of sounds, to focusing on the articulation of sounds, to examining the natural classes of sounds. These distinctive features form

the basis for the phonological representations that are accessed during lexical representation tasks, such as verbal communication.

While phonological representations have been a part of the phonology literature since the inception of Generative Phonology (Chomsky & Halle, 1968), which proposed that all speech sounds have both an underlying (perceptual) phonological representation and a surface (production) phonological representation, their distinctive feature composition has been widely debated. Specifically, it has been questioned whether phonological representations are fully specified in terms of the distinctive features, or whether some aspects of the phonological representations may be less detailed. For example, some approaches suggest that the phonological representations must be completely faithful to what is heard, thus typically requiring the storage in the mental lexicon of very detailed and specific representations for each production variant a listener encounters (Bybee, 2003; Johnson, 1997, 2005; Pisoni, 1993; Ranbom & Connine, 2007).

An alternative account suggests that a much more abstract phonological representation is created, stored, and accessed during speech perception and production. In other words, only contrastive or not otherwise predictable phonological information (i.e., distinctive features) must be stored for each phoneme encountered (Archangeli, 1988; Cornell, Lahiri, & Eulitz, 2011; Dinnsen, 1996; Eulitz & Lahiri, 2004; Gierut, 1996; Lahiri & Reetz, 2002, 2010; Steriade, 1995; Wheeldon & Waksler, 2004). Thus to make speech processing easier, adults are hypothesized to not store all aspects of all phonemes in their underlying representations within the mental lexicon. Only the phonemically contrastive distinctive features are stored; the features that are common and similar across phonemes are not stored because they are predictable. In other words, being less specified suggests that a sound contains some, but not all, of the distinctive

features of a more specified sound. Three primary models of sparse or underspecified representations have been proposed: contrastive underspecification (Steriade, 1995), radical underspecification (Archangeli, 1988), and the Featural Underspecified Lexicon (FUL) (Lahiri & Reetz, 2002, 2010).¹

While there has been objection to the idea of underspecification and it has been suggested that full phonological specification should be expected (Halle et al., 2000), asymmetries and markedness differences do exist across features and phonemes (Lahiri & Reetz, 2010). For example, certain English phonemes are considered to be more complex and marked² than others (Gierut, 2007): affricates (e.g., 'ch') are more marked than fricatives (e.g., /s/ and /z/), which are in turn more marked than oral plosives (e.g., /b/ and /d/) (Cataño, Barlow, & Moyna, 2009; Dinnsen & Elbert, 1984; Elbert, Dinnsen, & Powell, 1984; Gierut, Simmerman, & Neumann, 1994; Ingram, Christensen, Veatch, & Webster, 1980; Schmidt & Meyers, 1995). Underspecification is one way to account for the markedness phenomenon, with less marked phonemes assumed to be less specified. Thus, marked or complex sounds require the storage of more distinctive features in their phonological representations than do less complex sounds.

Underspecification theory also addresses phonological development, with one perspective suggesting that children's initial underlying representations only contain a small number of

¹ At a basic level, the first two models differ in terms of how they address marked and unmarked values in that contrastive underspecification only specifies non-redundant features, whether they are marked or unmarked, while radical underspecification specifies only the marked values of contrastive features. Both radical underspecification and FUL assume that during development children's initial phonological representations contain only universally specified distinctive features; the phonological representation becomes more complex as more vowels or consonants are identified and need to be distinguished from each other. While radical underspecification assumes that only unpredictable features are specified, with the predictable features being filled in by rules, FUL has clear predictions about how features are specified. It predicts that first the ARTICULATOR node is considered, with CORONAL existing, but underspecified as compared to other place of articulation features. If the ARTICULATOR features are not sufficient to differentiate lexical representations, then TONGUE HEIGHT contrasts will be addressed.

² Marked sounds are typically more complex phonologically (i.e. in terms of articulation and production), are acquired later in speech acquisition, and occur in fewer world languages.

universally specified distinctive features, with the representation becoming more complex, in terms of feature composition, as the child learns to identify the featural contrasts that differentiate phonemes (Archangeli, 1988; Dinnsen, 1996; Gierut, 1996). Thus, while adults' underlying phonological representations are sparse, children's underlying representations are initially even sparser. Importantly, typically developing (TD) children are hypothesized to have more specified underlying phonological representations than children with SSD (Dinnsen, 1996; Gierut, 1996).

While TD children are able to refine their underlying phonological representations to adult-like levels, it is quite likely that children with SSD may have difficulty creating accurate phonological representations due to their inaccurate perception of speech sounds (McGregor & Schwartz, 1992). In other words, faulty representation (i.e., memory traces in the mental lexicon) of the speech signal in the central auditory processing centers (Kraus, 2001) may be an underlying mechanism of SSD. This suggests that if a child with SSD does not adequately perceive, identify, and store all of the discriminating or distinctive features of a sound, the child's phonological representation of that sound may be underspecified. Thus, sounds sharing similar phonetic features (e.g., voicing, articulatory placement, and/or articulatory manner) require accurate speech perception so that the appropriate distinctive features of each sound are correctly stored in its phonological representations. The phonological representation, whether detailed or sparse, can then be accessed during both speech sound perception and production.

During speech perception, a presented sound is compared with the stored (in memory) phonological representations of all the sounds a listener knows in order to identify the given sound; this phoneme identification can go awry if a phonological representation does not contain enough detail to differentiate two sounds. For example, both /b/ and /d/ are voiced oral

stops/plosives that differ only by their place of articulation, with /b/ being produced bilabially at the lips and /d/ being a coronal sound produced with the tongue tip touching the alveolar ridge. If the place of articulation is not stored in the phoneme's phonological representation, it is possible that the child may misidentify or misperceive a /b/ as a /d/, or vice versa. Similarly, a child's production of a sound is also dependent on the stored phonological representation. If the phonological representation does not contain all of the relevant distinctive features, the child's production of the sound may also be incorrect because the representation did not specify all of the sound's features that needed to be produced. Thus similar to the perception example, a /b/ could be incorrectly produced as a /d/, if the phonological representation did not specify that the sound must be produced bilabially.

1.2 Electrophysiological indices of speech perception.

Prior behavioral studies have demonstrated that children with SSD are better able to perceive prototypical adult speech (Rvachew, Rafaat, & Martin, 1999), as compared to synthetic speech (Edwards, Fox, & Rogers, 2002; Monnin & Huntington, 1974) or child-produced speech (Chaney, 1988; Hoffman, Stager, & Daniloff, 1983). In these studies, the children are often required to identify whether a target sound is produced correctly. Unfortunately, behavioral tasks can only provide information with which to extrapolate possible differences underlying auditory processing. It is possible that children with SSD can differ from their TD peers at many points during the processing of auditory information, and behavioral tasks cannot provide data regarding this time course of processing that underlies the perception of auditory information.

In contrast, event-related potentials (ERPs) are an excellent tool to use when assessing perceptual processing of speech sounds. Given their attention-independent nature, ERP measures are free of behavioral confounds such as memory and cognition and are a good tool to use with

young children who are often non-compliant with behavioral testing. Moreover, the excellent temporal resolution of ERPs makes it an ideal tool for identifying different stages of perceptual and cognitive processing. Examining specific ERP peaks that represent different stages of speech perception such as sound encoding (e.g., P2: Čeponienė, Alku, Westerfield, Torki, & Townsend, 2005; Čeponienė, Torki, Alku, Koyama, & Townsend, 2008; Crowley & Colrain, 2004), sound integration (e.g., N2: Čeponienė et al., 2001; Čeponienė, Cummings, Wulfeck, Ballantyne, & Townsend, 2009), and sound discrimination (e.g., the Mismatch Negativity, MMN: Näätänen & Winkler, 1999; Picton, Alain, Otten, Ritter, & Achim, 2000) may help identify where in the process of perceiving sounds a child with SSD differs from his/her typically developing peers. In addition to providing excellent information regarding the exact timing of auditory perception, ERPs are able to identify whether populations of children can be differentiated by the neural sources involved in the perception of speech sounds. ERPs are able to identify cortical scalp distribution differences, such as when an effect is localized over just a few electrode sites over the left hemisphere as compared to when it is widespread across the scalp.

While no published research has examined the neural underpinnings of speech perception in children with SSD, studies involving children with language impairment, reading disorders, and/or childhood apraxia of speech have identified atypical electrophysiological discriminatory responses to speech sounds (Froud & Khamis-Dakwar, 2012; Kraus et al., 1996; Paul, Bott, Heim, Wienbruch, & Elbert, 2006; Sharma et al., 2006; Uwer, Albrecht, & von Suchodoletz, 2002). Given that developmental disorders such as SSD, language impairment, and reading disabilities have large amounts of overlap (Pennington & Bishop, 2009), it would seem quite plausible that children with SSD would also have differences in the underlying neural responses representing the discrimination of speech sounds.

One specific ERP peak that has been found to be useful in identifying fine-grained auditory distinctions is the Mismatch Negativity (MMN) (Näätänen & Winkler, 1999; Picton et al., 2000). The MMN is an attention-independent neurophysiological response elicited by an acoustically different (deviant) stimulus when presented in a series of homogenous (standard) stimuli – the oddball paradigm (Näätänen, Gaillard, & Mäntysalo, 1978; Näätänen, 1995). Thus, the MMN is an automatic change detection response in the brain and is thought to reflect stimulus discrimination and sensory memory (Sams, Paavilainen, Alho, & Näätänen, 1985); it can be elicited by any discriminable acoustic contrast: a change in intensity, frequency, or duration, or a change in a complex sound like a chord, speech syllable, or an auditory pattern. The MMN typically is observed 100-350 ms after stimulus onset (Čeponienė et al., 2002; Csépe, 1995; Korpilahti, Krause, Holopainen, & Lang, 2001; Morr, Shafer, Kreuzer, & Kurtzberg, 2002; Näätänen, Paavilainen, & Reinikainen, 1989; Shafer, Morr, Kreuzer, & Kurtzberg, 2000), suggesting that low-level neural mechanisms exist to distinguish between certain acoustic and/or phonological contrasts. Moreover, the MMN can be sensitive to language-specific speech sound representations (Kraus, McGee, & Koch, 1998a, 1998b; Näätänen, 2001; Näätänen et al., 1997; Winkler et al., 1999).

The MMN has also recently been used to demonstrate the presence and absence of phonological underspecification in adults. For example, within the FUL approach to underspecification, it is assumed that certain consonantal places of articulation (e.g., coronal, such as /d/) are less specified than other places (e.g., labial, such as /b/) of articulation (Cornell et al., 2011; Cornell, Lahiri, & Eulitz, 2012; Eulitz & Lahiri, 2004; Friedrich, Eulitz, & Lahiri, 2006; Gaskell & Marslen-Wilson, 1996, 1998; Lahiri & Reetz, 2002, 2010; Snoeren, Gaskell, & Di Betta, 2009; Wheeldon & Waksler, 2004; Zimmerer, Reetz, & Lahiri, 2009). Larger and

earlier MMN responses have been observed when there was a conflict between the phonological representations of two sounds. In other words, when a more specified sound, such as /b/, was the standard in a MMN oddball paradigm, large MMN responses were elicited by the less specified sound, /d/, since /b/ specified the bilabial place of articulation, which /d/ violated by being coronal in nature. Alternatively, when the less specified sound (/d/), served as the standard, the more specified sound (/b/) did not elicit a MMN due to the fact that the place of articulation was not specified by /d/ so no conflict between the phonetic features was identified (e.g., Eulitz & Lahiri, 2004). Thus following the underspecification theory's interpretation of neural responses, the MMN could be a useful measure to use when examining the specificity of phonological representations in children with SSD. Smaller MMN responses could be indicative of less specification of the standard sound's phonological representation.

1.3 The present study.

No previous research has examined electrophysiological brain responses reflecting attention-independent speech sound discrimination of children with SSD. It is presently unknown why children with SSD do not produce certain sounds correctly; however, one possible reason may be that they cannot perceptually distinguish the distinctive features of phonemes, which results in a less detailed phonological representation. The purpose of this study was to determine whether children with SSD had typical or atypical auditory neural discrimination responses to two early-acquired sounds, /b/ and /d/, that they could produce, as compared to their same-age peers. Based on previous research involving children with language impairment, reading impairment, and childhood apraxia of speech, it was hypothesized that children with SSD would elicit small or no MMN responses to the speech sounds, as compared to typically developing children, possibly implicating a neural mechanism underlying SSD.

In the present study's oddball MMN paradigm, the syllable /ba/ was the standard and /da/ was the deviant. Based on previous ERP work (e.g., Eulitz & Lahiri, 2004; Lahiri & Reetz, 2002, 2010), it was predicted that the distinctive features from the standard syllable, /ba/ would be extracted and stored (briefly) in memory as an underlying phonological representation of that syllable. Alternatively, the deviant syllable, /da/, would serve as the surface phonological representation by being the most recent auditory input. The phonetic features extracted from the deviant would be compared to those of the standard, which would have been extracted and stored in memory.

Thus, the phonological variation in the syllable pairs of the present study was predicted to result in different neural responses. Importantly, the children were predicted to be less sensitive to changes following standard sounds composed of more sparse phonological representations, as compared to standard sounds composed of more detailed, specific representations. Thus, while both groups of children were presented with the same standard syllable, /ba/, if the /ba/ had a less detailed phonological representation in children with SSD, it would result in a smaller MMN response.

2. MATERIALS AND METHODS

2.1 Participants.

Twenty-four children between the ages of 4 and 6 years of age completed the ERP study (Table 1): 12 children (3 female) had speech sound disorders (SSD) and 12 children (4 female) were typically developing (TD). All participants were right-handed monolingual English speakers. They were screened for neurological disorders, hearing, uncorrected vision, emotional,

and behavioral problems. Hearing was tested with a portable audiometer using pure tones of 500, 1000, 2000, and 4000 Hz. Thresholds of 20-dB or lower were required to pass. All children also passed an oral-peripheral mechanism exam (Robbins & Klee, 1987). All participants signed informed consent in accordance with the University of North Dakota Human Research Protections Program.

The children with SSD all had a previous diagnosis of SSD, but to ensure their speech production difficulties, a common, standardized test of speech production was administered. The TD children were required to have a minimum standard score of 85 on the *Goldman-Fristoe Test of Articulation – 2* (GFTA-2; Goldman & Fristoe, 2000), while the children with SSD were required to have a maximum standard score of 80 on the GFTA-2 (Goldman & Fristoe, 2000) (Table 1). Three measures of speech production ability were calculated from children's performance on this standardized articulation test. First, the GFTA-2 Raw Score was the number of errors, out of the target 77 consonant sounds, that each child had on the test. The GFTA-2 Standard Score was based on the number of errors that each child had, while taking into account the child's gender and age; thus, this was a normalized measure. The GFTA-2 Percentage of Consonants Correct (PCC) measure (e.g., Shriberg, Austin, Lewis, McSweeney, & Wilson, 1997; Shriberg & Kwiatkowski, 1982; Shriberg, 1993) was calculated by identifying the number of consonants the child produced correctly out of 152 (the total number of consonants contained in all of the words on the entire GFTA-2).

< Table 1 about here >

In addition to the above assessments, the children with SSD received additional standardized testing of their receptive picture vocabulary skills and nonverbal cognition abilities using the *Peabody Picture Vocabulary Test-IV* (PPVT-IV; Dunn & Dunn, 2007) and the Brief IQ Screener

on the *Leiter International Performance Scale – Revised* (Leiter-R; Roid & Miller, 1997). These measures were used to ensure that the children with SSD only had speech production impairments, without concomitant language impairments and/or intellectual disabilities. All of the children with SSD were within the normal range on these measures (Table 1).

2.2 Stimuli.

Syllables (consonant + /a/) were pronounced by a male North American English speaker. The syllables were digitally recorded in a sound isolated room (Industrial Acoustics Company, Inc., Winchester, UK) using a Beyer Dynamic (Heilbronn, Germany) Soundstar MK II unidirectional dynamic microphone and Behringer (Willich, Germany) Eurorack MX602A mixer. The syllables were digitized at 44.1 kHz with a 16-bit sampling rate. The average intensity of all the syllable stimuli was normalized to 65 dB SPL.

Four different syllables (one standard and three deviants) were used in the study. The *Standard Syllable* for all children was “ba” (/ba/), which contained a consonant that every child produced correctly in a brief sound stimulability assessment (Powell & Miccio, 1996)³. Three deviant syllables were presented to each child. Two of the deviants were specific to each child with SSD and were based on the sound inventories of each child - that is, these were sounds that the child with SSD could not produce correctly (e.g., “ra”, “sa”, “sha”, “tha”, “cha”, “fa”, “ga”); these two deviants varied across children. The third *Deviant Syllable*, “da” (/da/), was the same for all children and contained a consonant that every child produced correctly in a brief sound stimulability assessment (Powell & Miccio, 1996). For the purposes of this paper, only the

³ The children were asked to watch, listen, and say what the experimenter said. Both /b/ and /d/ were produced with three different vowels, /a/, /i/, and /u/, in three contexts: consonant-vowel (e.g., /ba/), vowel-consonant-vowel (e.g., /aba/), and vowel-consonant (e.g., /ab/). In this quick and simple production task, the children were judged to be able to accurately produce both consonants in all 9 situations.

children's responses to /ba/ and /da/ will be discussed since all children could produce those sounds and all children were presented with those sounds in the ERP paradigm.

The two syllables of interest, /ba/ and /da/, are phonetically very similar. They are both voiced oral stops/plosives. They differ in terms of their place of articulation, with /b/ being produced bilabially (at the lips) while /d/ is produced with the tongue tip/blade touching the alveolar ridge behind the top teeth (a coronal production). Both sounds are acquired early in development, with both being produced by 85% of 2-year-olds (Goldman & Fristoe, 2000).

To better characterize the syllable stimuli, the first three formants of three different parts of each syllable were measured using Praat acoustic analysis software (Boersma & Weenick, 2013). Specifically, acoustic measurements of the consonantal sound, the consonant-vowel transition, and the vowel sound were measured for each of the syllables (Table 2; Figure 1). Acoustic measurement was a multi-step process. First, the onset of the vowel was identified by visual judgment through examining the steady state of the formants subsequent to a transition. The consonant-vowel transition was then identified as the period of time in which the formants rose or fell prior to the steady vowel state; the point at which there was no more change in formant slope was identified as the onset of the transition. The consonant sound was then determined to be whatever acoustic information occurred prior to the onset of the transition. Once each of the three stages was identified, the midpoint of each was calculated. The formant frequencies were then measured at this midpoint.

< Table 2 about here >

< Figure 1 about here >

The syllables initially varied slightly in duration, due to the individual phonetic make-up of each consonant. Syllable duration was minimally modified (by shortening the vowel duration) so

that all syllables were 375 ms in length. Each syllable token used in the study was correctly identified by at least 15 adult listeners.

2.3 Stimulus Presentation.

The stimuli were presented in blocks containing 237 standard stimuli and 63 deviant stimuli (21 per deviant), with 5 to 8 blocks being presented to each participant. Each block lasted approximately 6 minutes and the children were given a break between blocks when necessary, which was typically 2-3 breaks per session. Within the block, the four stimuli were presented using an oddball paradigm in which the three deviant stimuli (probability = 7% for each) were presented in a series of standard stimuli (probability = 79%). Stimuli were presented in a pseudorandom sequence and the onset-to-onset inter-stimulus interval varied randomly between 600 and 800 ms. The syllables were delivered by stimulus presentation software (Presentation software, www.neurobs.com). The syllable sounds were played via two loudspeakers situated 120 cm in front and 30 degrees to the right and left from the midline in front of a participant, which allowed the sounds to be perceived as appearing from the midline space. During the study, the children sat alone or in their parent's lap in a sound-treated room and watched a silent cartoon video of their choice. Typically the recording of the ERPs took approximately 1 hour.

2.4 EEG Recording and Averaging.

Sixty-six channels of continuous EEG (DC-128 Hz) was recorded using ActiveTwo data acquisition system (Biosemi, Inc, Amsterdam, Netherlands) at a sampling rate of 256 Hz. This system provides “active” EEG amplification at the scalp that substantially minimizes movement artifacts. The amplifier gain on this system is fixed allowing ample input range (-264 to 264 mV) on a wide dynamic range (110 dB) Delta-Sigma ($\Delta\Sigma$) 24-bit AD converter. Sixty-four channel scalp data were recorded using electrodes mounted in a stretchy cap according to the

International 10-20 system. Two additional electrodes were placed on the right and left mastoids. Eye movements were monitored using FP1/FP2 (blinks) and F7/F8 channels (lateral movements, saccades). During data acquisition, all channels were referred to the system's internal loop (CMS/DRL sensors located in the centro-parietal region), which drives the average potential of a subject (the Common Mode voltage) as close as possible to the Analog-Digital Converter reference voltage (the amplifier "zero"). The DC offsets were kept below 25 microvolts at all channels. Off-line, data were re-referenced to the average of the left and right mastoid tracings.

Prior to data averaging, sporadic artifact rejection of the continuous EEG was completed using EEGLAB (Delorme & Makeig, 2004). This involved marking and rejecting the time periods during which sporadic artifacts occurred (i.e., random head movements, muscle movements related to speaking, excessive electrode activation stemming from pressing the head into the back of the chair, etc.). After sporadic artifact rejection, independent-component analysis (ICA) (Jung et al., 2000) was completed so that the experimenters could identify eye blink and saccade components; these components were then deleted from the continuous EEG. The remaining artifactual trials due to excessive muscle artifact, amplifier blocking, and overall body movements were rejected from further analyses using the simple voltage threshold measure in ERPLAB (Luck & Lopez-Calderon, 2012). The voltage limits for all children were set at -100 to 100 microvolts; none of the children's data needed additional/higher threshold measures. Epochs containing 100 ms pre-auditory stimulus and 800 ms stimulus time were baseline-corrected with respect to the pre-stimulus interval and averaged by stimulus type: Standard Syllable and Deviant Syllable. The data were low-pass filtered at 30 Hz and high-pass filtered at 0.05 Hz, using 2-way least squares FIR filters. On average, the remaining individual data contained 751

(SD = 127) Standard Syllable (/ba/) trials (TD M = 774 (SD = 292); SSD M = 728 (SD = 127)) and 91 (SD = 27) Deviant Syllable (/da/) trials (TD M = 91 (SD = 36); SSD M = 91 (SD = 19)).

2.5 ERP Measurements.

ERP responses to the Standard (/ba/) and Deviant (/da/) Syllable trials were analyzed. The MMN was measured at the electrodes where it was robust in the grand-average difference wave waveforms. Difference waves were created by subtracting the response elicited by the standard stimulus from the response elicited by the deviant stimulus.

While all electrodes were examined, the MMN is typically maximal over fronto-central midline electrode sites (e.g., Fz, FCz, and Cz) (Näätänen, Teder, Alho, & Lavikainen, 1992). Accordingly, the MMN was measured at these three electrodes. Visual inspection of the grand average waveforms suggested that the MMN peak appeared between approximately 300 and 400 ms post-syllable onset. The MMN peak was then measured individually in each participant within this time window.

Three different dependent variables were used for statistical analysis of the MMN peak: MMN latency, MMN mean amplitude, and MMN rectified area; all three were measured using the ERPLAB Toolbox (Luck & Lopez-Calderon, 2012). The peak latency was determined as the latency of the most negative point in the difference waveform of the three electrodes (Fz, FCz, and Cz) of each participant in the designated search windows. The mean amplitude of the MMN was measured over a 40 ms window centered at the peak latency of the three electrodes for each participant. To clarify, the peak latency detection program in ERPLAB identified the most negative point (i.e., largest MMN response) within the 300-400 ms post-syllable onset time window in each individual participant's difference wave waveforms. The mean and rectified

amplitudes were then measured using the previously identified peak latency as the middle time point for the 40 ms analysis time windows. Thus, the timing of the mean and rectified amplitude measurements varied slightly across participants, depending on the MMN latency.

The rectified area measurements were also included because of the large inter-individual variability in MMN amplitudes in children (Rinker et al., 2007). The rectified area refers to the area under the ERP waveform curve in a give time window. In other words, the ERP waveform was combined with the zero-voltage baseline to form a set of polygons, one for each set of consecutive points with the same polarity. Thus, the area measure summed together the areas of the individual polygons, ignoring the polarity of the waveform used to create each polygon. This was equivalent to *rectifying* the waveform (turning every negative value into a positive value) and then computing the integral over the measurement window (Luck & Lopez-Calderon, 2012). The rectified area measurements are presented in microvolt seconds (μ Vs) because area has a height (μ V) and a width (seconds). For example, a 1 μ V value over a 100 ms period would have a rectified area of 0.1 μ Vs. The rectified area was calculated for the same 40 ms time window centered on each individual participant's peak MMN latency. There were strong correlations between both the MMN Mean Amplitude and Rectified Area measurements (Fz: $r = -.690$, $p < .0001$; FCz: $r = -.809$, $p < .0001$, Cz: $r = -.428$, $p < .04$).

One sample t-tests were first used to measure whether the MMN mean amplitudes and rectified areas of each group at each electrode were significantly different from zero. Cohen's d effect sizes and effect size correlations (r_{Y1}) were also calculated for each t-test. The group differences of the MMN latency, mean amplitude, and rectified area measurements were examined in separate Group (SSD, TD) x Electrode (Fz, FCz, Cz) repeated measure ANOVAs.

Partial eta squared (η^2) effect sizes are also reported for all significant effects and interactions.

When applicable, Geiser-Greenhouse corrected p-values are reported.

In addition, since the phonological representations of the children were of interest, pre-planned correlations examining the children's age, speech production ability (GFTA-2 Raw Scores and Percent Consonants Correct (PCC)), and MMN measures were completed. Three sets of correlations were completed: one for each group (SSD, TD) separately, and one with both groups combined together. None of the correlations for the individual groups were significant, thus only the combined group correlations are reported below.

3. RESULTS

In both participant groups, the ERP waveforms elicited by the Standard and Deviant stimuli consisted of a large auditory P1/P2 positivity at ca. 150 ms and a large negativity, the auditory N2, discernable at ca. 300 ms (Figure 2). In the difference wave, both groups of children demonstrated a MMN response between 300 and 400 ms (Figure 2).

< Figure 2 about here >

3.1 MMN Latency.

No main effect of Participant Group was observed ($p > .24$). Thus, the MMN latency did not differ between children with SSD and their TD peers (Table 3). The MMN latency did not differ across the three electrodes ($p > .23$). No Group x Electrode interaction was observed ($p > .49$). None of the correlations between MMN latency, children's age, and speech production ability was significant.

< Table 3 about here >

3.2 MMN Mean Amplitude.

One sample t-tests revealed that the TD children had a significant MMN mean amplitude at Fz ($t(11) = -3.076$, $p < .02$, $d = 1.854$, $r_{Y1} = .680$), FCz ($t(11) = -4.629$, $p < .002$, $d = 2.791$, $r_{Y1} = .813$), and Cz ($t(11) = -3.609$, $p < .005$, $d = 2.176$, $r_{Y1} = .736$). For the children with SSD, a significant MMN mean amplitude was observed at FCz ($t(11) = -3.064$, $p < .02$, $d = 1.848$, $r_{Y1} = .679$) and Cz ($t(11) = -5.028$, $p < .0001$, $d = 3.032$, $r_{Y1} = .835$), but not at Fz ($t(11) = -1.968$, $p < .08$, $d = 1.187$, $r_{Y1} = .510$) (Table 3).

No main effect of group was found ($p > .30$). Thus, overall the children with SSD and the TD children had similar MMN mean amplitudes. This lack of significant differences was most likely due to the wide range of mean amplitudes within groups (TD range: $-9.31 \mu V$ to $3.23 \mu V$; SSD range: $-7.34 \mu V$ to $3.10 \mu V$). The overall MMN mean amplitude did not differ across the three electrodes ($p > .22$). In addition, a strong trend for a Group x Electrode interaction was observed ($F(2,44) = 3.319$, $p < .06$, $\eta^2 = .131$). However, separate post-hoc t-tests did not find any significant group differences at any of the electrodes ($p > .11 - .40$). Post-hoc repeated measure ANOVAs completed separately for each group also did not reveal mean MMN amplitude differences across electrode sites for children with SSD ($p > .12$) or TD children ($p > .12$).

The correlation involving the MMN mean amplitude at electrode Fz and children's age was significant ($r = -.417$, $p < .05$), with older children having slightly larger Fz MMN mean amplitudes (Figure 3). No other correlations involving the mean amplitude, age, and children's speech production abilities were significant.

< Figure 3 about here >

3.3 MMN Rectified Area.

One sample t-tests revealed that the TD children had a significant MMN rectified area measurements at Fz ($t(11) = 5.489$, $p < .0001$, $d = 3.310$, $r_{Y1} = .856$), FCz ($t(11) = 5.654$, $p <$

.0001, $d = 3.409$, $r_{YI} = .863$), and Cz ($t(11) = 6.589$, $p < .0001$, $d = 3.973$, $r_{YI} = .893$). The children with SSD also had a significant MMN rectified area measurements at Fz ($t(11) = 2.607$, $p < .03$, $d = 1.572$, $r_{YI} = .618$), FCz ($t(11) = 3.063$, $p < .02$, $d = 1.847$, $r_{YI} = .678$), and Cz ($t(11) = 2.781$, $p < .02$, $d = 1.677$, $r_{YI} = .643$) (Table 3).

Overall, the TD children had larger MMN rectified area measurements than did the children with SSD ($F(1,22) = 7.013$, $p < .02$, $\eta^2 = .242$). The overall MMN rectified area measurements did not differ across the three electrode sites ($p > .09$). A Group x Electrode interaction was also found ($F(2,44) = 4.079$, $p < .03$, $\eta^2 = .156$). Separate post-hoc t-tests revealed that the group differences were significant at Fz ($t(22) = -2.503$, $p < .03$, $d = 1.029$, $r_{YI} = .458$) and FCz ($t(22) = -3.091$, $p < .006$, $d = 1.269$, $r_{YI} = .536$), but not at Cz ($t(22) = -1.161$, $p > .25$, $d = 0.474$, $r_{YI} = .230$). Post-hoc repeated measure ANOVAs completed separately for each group revealed MMN rectified area differences across electrode sites for TD children ($F(2,22) = 3.904$, $p < .04$, $\eta^2 = .262$), driven by the fact that FCz elicited significantly measurements than Cz. No electrode site differences were found for children with SSD ($p > .28$).

The correlations between MMN rectified area and children's age and speech production ability suggest that while age was not related to the rectified area measurements, children's speech production ability was significantly correlated with the area measurements. Recall that two different speech production accuracy measurements were calculated with the GFTA-2: 1) the raw score, which refers to the number of errors each child had on the target 77 consonant sounds of the test and 2) the percent consonants correct (PCC), which was calculated by individually assessing *each* consonant produced in *all* of the GFTA-2 words (152 consonants). While the PCC measure provides a better overall assessment of a child's speech production ability due to the larger number of consonant sounds it samples, the raw score is what a

practicing speech-language pathologist is most likely to calculate due to time constraints. Moreover, the two measures were strongly correlated within the present sample ($r = -.979$, $p < .0001$). Children with more errors on the GFTA-2 had smaller Fz ($r = -.447$, $p < .03$) and FCz ($r = -.473$, $p < .03$) rectified area measurements (Figure 4). Similarly, children who produced more consonants correct on the GFTA-2 had greater Fz ($r = .499$, $p < .02$) and FCz ($r = .547$, $p < .007$) rectified area measurements (Figure 5). Thus, the MMN did appear to capture the relationship between speech production and speech perception.

< Figure 4 about here >

< Figure 5 about here >

4. DISCUSSION

This study examined children's electrophysiological brain responses reflecting attention-independent discrimination of auditory syllables containing two early-acquired English phonemes, /b/ and /d/, that they could produce. This was the first study to use the excellent temporal resolution of electrophysiological methods (i.e., ERPs) to examine the neural mechanisms underlying speech sound processing in children with speech sound disorders (SSD) and their typically developing (TD) peers. The oddball MMN paradigm was used to identify possible differences in the phonological representations of children with SSD and TD children; a standard syllable /ba/ and a deviant syllable /da/ were used. The children with SSD demonstrated smaller MMN responses than did their TD peers, suggesting that while they could correctly produce the target sounds /b/ and /d/, their phonological representations for those sounds might not be as detailed as the representations of children without speech impairments. The larger MMN responses of the TD children suggest that they were able to access well-established language-specific phonological representations, indicative of typical phonological development.

4.1 Specification in phonological representations.

It was expected that within the oddball stimulus paradigm of the present study, the distinctive features extracted from the deviant syllable, /da/, would be compared with the distinctive features extracted from the standard syllable, /ba/, which were stored in memory. Detectable phonological variation between the two stimuli was predicted to result in different patterns of neural responses. Given that the amplitude of any ERP response broadly represents the degree to which underlying neural generators are active during a cognitive task (Otten & Rugg, 2005), a smaller ERP amplitude response would indicate that fewer neural resources were used during a task while a larger ERP response would suggest more neural involvement. Moreover, if fewer neural resources are allocated for a task, it is likely that the task would not be as effectively completed as if more resources were used.

The present study's cognitive task involved the extraction of phonological information from speech sounds and measured neural involvement via the MMN. Thus, a smaller MMN could indicate that fewer distinctive features were perceived, identified, and stored, resulting in a more sparse phonological representation (Eulitz & Lahiri, 2004). Alternatively, larger MMN responses could be indicative of more neural involvement; additional neural allocation might allow for more distinctive features to be adequately perceived, resulting in a more detailed phonological representation. As was predicted, children with SSD demonstrated a smaller MMN response than did their TD peers. Thus, consistent with previous behavioral research (Dinnsen, 1996; Gierut, 1996), the phonological representations of children with SSD, as measured by the MMN, were not as specified as those of typical children.

Previous adult ERP underspecification studies have used a full crossing of /b/ and /d/, in that both sounds served as both standards and deviants in the MMN oddball paradigm, in order to

determine whether /b/ or /d/ had a less specified phonological representation (Cornell et al., 2011, 2012; Eulitz & Lahiri, 2004; Friedrich et al., 2006; Gaskell & Marslen-Wilson, 1996, 1998; Lahiri & Reetz, 2002, 2010; Snoeren et al., 2009; Wheeldon & Waksler, 2004; Zimmerer et al., 2009). While it would have been ideal to be able to examine the specific phonological representations of the phonemes /b/ and /d/, due to ERP recording time constraints, this study chose to focus on the overall population group differences in the specification of the standard sound, /b/. Thus, without a full crossing, it is difficult to know whether children with SSD have a typical or atypical representations of /b/ and/or /d/, with a likely possibility being that both /b/ and /d/ are underspecified in this population. Future studies will explicitly contrast /b/ and /d/ (as both standard and deviant stimuli), in order to determine whether typically developing children and children with SSD have a less specified representation of /d/, as compared to /b/. Currently the data suggests that children with SSD have overall less specified phonological representations than do their TD peers.

Since both syllables, /ba/ and /da/, contained the same vowel, the ERP results were interpreted in terms of differences in the two consonants, /b/ and /d/. However, since the syllables were naturally produced, there were subtle variations in the productions of the /a/ in the two syllables. Thus, it is a possibility that some of the MMN response differences were due to the acoustic variations within the vowels. While this is somewhat unlikely due to listeners' ability to categorize subtle variations of a sound into a larger sound category via categorical perception (Clayards, Tanenhaus, Aslin, & Jacobs, 2008; Kuhl, Tsao, & Liu, 2003; Kuhl, Williams, Lacerda, Stevens, & Lindblom, 1992; Kuhl, 2004; Liberman, Harris, Hoffman, & Griffith, 1957; Liberman, 1996; Polka & Werker, 1994; Werker & Tees, 1984), it is important to consider all possibilities when working with children with known speech sound impairments. To

ensure that adults perceived these two productions as the same vowel, 43 undergraduates in a Clinical Phonetics course were asked to identify the two vowels. Both vowels were overwhelmingly identified as /a/ (95% identified it in /ba/; 93% identified it in /da/); of the three adults who did not identify it as /a/, two identified it as the very similar vowel, /ɔ/. Thus, even though there were some subtle acoustic differences between the two vowels, the vowels in both syllables were perceived and categorized as the same vowel by the vast majority of adults. Future studies that explicitly control the speech stimuli (e.g., by splicing the same vowel onto each consonant) will control for these possible acoustic confounds.

4.2 Scalp distribution differences.

Three midline electrodes were examined in this study, with group differences being observed over the two electrodes most commonly associated with the MMN response: Fz and FCz. Unfortunately, large variation in mean amplitudes within and across groups limited scalp distribution analyses. Since the MMN has been well studied, much is known about its overall scalp distribution patterns in healthy adults (Garrido, Kilner, Stephan, & Friston, 2009; Näätänen, Astikainen, Ruusuvirta, & Huotilainen, 2010; Näätänen, 2001; Winkler, 2007). Moreover, MEG and MRI studies completed in conjunction with ERP studies have provided evidence that the MMN is generated by a fronto-temporal network, often located in the auditory cortex (Alho, Huotilainen, & Näätänen, 1995; Doeller et al., 2003; Garrido et al., 2009; Hari et al., 1984; Korzyukov, Winkler, Gumenyuk, & Alho, 2003; Liebenthal et al., 2003; Molholm, Martinez, Ritter, Javitt, & Foxe, 2005; Näätänen et al., 2010; Opitz, Rinne, Mecklinger, von Cramon, & Schröger, 2002; Rinne, Degerman, & Alho, 2005; Rinne, Alho, Ilmoniemi, Virtanen, & Näätänen, 2000).

Visual inspection of the scalp distribution of the mean amplitudes measured at the approximate mean peak latency windows for the MMN (TD: 300-340 ms; SSD: 325-365 ms) in Figure 2 suggests that the TD children and children with SSD were using potentially different neural networks during the processing of the /ba/-/da/ mismatch. The TD children demonstrated a typical MMN scalp distribution, with a slightly right fronto-central peak area of activation. Alternatively, the children with SSD had a prominent central area of activation over the vertex (i.e., electrode Cz). These differences in scalp distribution could indicate that atypical neural generators were involved in the speech perception of children with SSD. For example, adolescents with SSD have been found to have hypoactivation of speech perception areas in the right medial temporal gyrus (Tkach et al., 2011). Future studies with a larger participant sample will be able to better address these potential scalp distribution differences.

4.3 The speech perception and speech production relationship.

It was most interesting that though the children with SSD could consistently and correctly produce /b/ and /d/, their MMN responses were still significantly smaller than those of their TD peers. This suggests that while children's speech impairments are often most apparent in their incorrect productions of more complex, later developing sounds, such as /r/ or /s/, (Gierut, 2007), a fairly widespread impairment involving the acoustic-phonetic processing of all sounds might be present in this population. The correlation measures also support the idea that children with SSD have a more extensive acoustic-phonetic processing deficit that involves more than just the sounds that they have difficulty producing. Specifically, the children's speech production abilities were strongly correlated with the MMN Fz and FCz rectified area measures. These results support previous claims that the MMN is an index of acoustic and/or phonological contrasts (Čeponienė et al., 2002; Csépe, 1995; Korpilahti et al., 2001, 2001; Näätänen et al.,

1989; Shafer et al., 2000). More importantly, these results suggest that children with speech production impairments appear to have some difficulty with the perception of acoustic-phonetic contrasts of similar sounds that they can produce correctly. Future studies involving other sounds that share acoustic-phonetic properties, such as the approximants (e.g., /r/, /l/, /w/), or more complex/late-acquired sounds will further clarify whether children with SSD have a widespread speech sound perception deficit.

4.4 Caveats and future directions.

This study is the first to examine the neural responses to speech sounds in children with SSD. As with any new population being studied, a variety of experimental observations and caveats arose through the course of the study, which can inform future research directions.

One interesting observation with this study was the statistical difference between the MMN mean amplitude MMN measurements and the rectified area MMN measurements. There should not have been much difference between the two types of measurements if the MMN included only negative potentials for all children. However, since the rectified area measurements calculate the entire area under the waveform curve (negative and positive), they may differ from mean area measurements if all or part of the mismatch response includes positive potentials. In other words, the fact that group differences were identified with the rectified area measurements, and not the mean amplitude measurements, suggests that some or all of the children with SSD had at least partial positive mismatch responses (P-MMR).

While no previous ERP study has involved children with SSD, a recent study examined the speech sound discrimination abilities of children with childhood apraxia of speech (CAS) (Froud & Khamis-Dakwar, 2012). CAS typically is characterized as a disorder of motor learning and/or motor programming, though deficits in phonological representation and processing may also be

involved (Froud & Khamis-Dakwar, 2012). Froud and Khamis-Dakwar (2012) observed that while the TD children demonstrated a large MMN, the children with CAS demonstrated a P-MMR to the phonemic contrast in the same general time window as the MMN. Since this positive response is typically seen only in much younger children (Rivera-Gaxiola, Silva-Pereyra, & Kuhl, 2005), the P-MMR was interpreted as an immature pattern of mismatch responding characteristic of much younger children (Froud & Khamis-Dakwar, 2012). Thus, the presence of a P-MMR suggests that children might be beginning to establish separate distinguishing phonemic categories, but may not have fully established phonological representations. Future studies involving children with SSD will look more closely at their individual responses to see if they demonstrate positive and/or negative mismatch responses. The positive versus negative mismatch response may even have the potential to help differentiate subgroups of children in terms of the severity of their disorder.

Another interesting finding in the present study was the appearance of a very early negativity (appearing 0-50 ms post-stimulus onset) in response to the /da/ deviant in the ERP waveforms of the TD children. Since the deviant elicited the negativity, the early negativity was also present in the MMN difference wave. While this negativity was outside the MMN time window of interest, its prominence in the waveform suggests that its origin(s) should be hypothesized.

A variety of possible reasons for this negativity were examined. The age of the participants was not a possible reason for the negativity, as the children were all very similar in age both within and across groups. Moreover, the TD children and children with SSD were tested as they were recruited; thus, recording sessions were not “blocked” by participant group, meaning that both groups of children were randomly recorded and no overt ERP recording differences between groups were noted. It is possible that this early negativity was due to noise in the ERP

signal, possibly due to small numbers of trials. However, there was no difference in the number of standard ($t(22) = -.412, p > .68$) or deviant ($t(22) = -.368, p > .71$) trials between the two groups.

The current working hypothesis is that the early negativity is due to a combination of the acoustical differences in the experimental stimuli and participant characteristics. In terms of acoustical stimulus differences, close examination of the stimulus waveforms and spectrograms suggests that the release of the two stops have different latencies, with the occlusion of the stop being included in the recording of /ba/ but not in /da/. This subtle difference would result in a slight VOT differences between conditions and it is possible that this slight acoustic difference may explain the small early effect in the ERP.

In terms of participant differences, children with SSD have been shown to have poor sensitivity to acoustic cues (Hoffman, Daniloff, Bengoa, & Schuckers, 1985) and might use nonstandard acoustic cues to define contrasts across phonemes (Rvachew & Jamieson, 1995). To clarify, several different acoustic cues help differentiate phonemes (Johnson, Pennington, Lowenstein, & Nittrouer, 2011), and it is possible that the acoustic properties that a child attends to changes throughout development (Johnson et al., 2011; Nittrouer, Manning, & Meyer, 1993). For example, it has been shown that young children initially attend to dynamic acoustic properties, such as formant transitions, while older children attend to more specific acoustic cues, such as silent gaps (indicating vocal tract closure) or phoneme duration (Johnson et al., 2011; Nittrouer & Studdert-Kennedy, 1987; S Nittrouer, 1992, 1996; Nittrouer & Miller, 1997). Thus, the weighting of acoustic cues may be related to the accurate development of phonological representations (Nittrouer et al., 1993).

In other words, in the present study, the two groups of children may have attended to different acoustic cues in the two stimuli, /ba/ and /da/, resulting in different patterns of responses. The TD children might have perceived the subtle acoustic difference at the beginning of the /da/, while the children with SSD did not attend to that cue, which then resulted in the early negativity in response to /da/ in the waveforms of the TD children. If this is the case, it is possible to interpret the early negativity as an indication of an extra processing load placed on the auditory system (Sussman, Steinschneider, Gumenyuk, Grushko, & Lawson, 2008). Thus, the TD children perceived the early acoustic stimulus difference in /da/ and attempted to process it; alternatively, the children with SSD did not attend to that acoustic cue and thus did not engage in the processing of that information. That is, an absence of the early negativity might be indicative of no processing effort allocated to the task at that point in time, as speech sound discrimination of that acoustic cue was not identified as a task to complete (due to poor perception).

If the children with SSD used nonstandard acoustic cues to define the contrasts for /b/ and /d/ (Hoffman et al., 1985; Nittrouer et al., 1993; Rvachew & Jamieson, 1989), it is also possible that these inaccurate phonological representations also affected their production of the sounds. Recall that the experimenters used a stimulability probe (Powell & Miccio, 1996) to assess the children's productions of /b/ and /d/, and children were judged to be 100% stimutable for those sounds in simple consonant-vowel combinations. However, it is possible that subtle acoustic differences were also present in the /b/ and /d/ productions of children with SSD and TD children.

For example, small differences in voice onset time (VOT) (Fabiano-Smith & Bunta, 2012; Macleod & Glaspey, 2014; Yu et al., 2014) might have been present. To clarify, three different

stages of stop voicing have been found in children (Macken & Barton, 1980). First children have no differentiation in their production of voiced and voiceless stops (e.g., /p/ and /b/); both are produced with short-lag VOT. Children then begin to differentiate voiced and voiceless stops, however both types of productions still fall within the adult category of short-lag voicing. Finally, children fully differentiate voiced and voiceless stops. While the differentiation of voiced and voiceless stops is expected around 2 years of age, this doesn't necessarily mean that children are producing the sounds with adult-like VOT contrasts, as it has been found that children's VOT values are longer than those of adults (Fabiano-Smith & Bunta, 2012; Johnson & Wilson, 2002; Lisker & Abramson, 1964). Thus, it is possible that the VOT values of the TD children might have been closer to adult-like productions than those of the children with SSD. Future studies can possibly examine this acoustic-perception relationship in more detail.

Finally, the scope of the present study was fairly narrow, in that it only compared one standard syllable and one deviant syllable. As a result, claims regarding the specificity of phonological representations must be tempered until more studies can be completed that examine many speech sounds from a variety of phoneme classes (i.e., not just stops, but also fricatives, liquids, vowels, etc.). In addition, there is no information regarding this population's responses to non-speech stimuli, such as pure tones. Given the evidence suggesting the MMN response is deviant in children with known language disabilities in response to tones (Ahmmed, Clarke, & Adams, 2008; Korpilahti & Lang, 1994; Rinker et al., 2007), it is a possibility that children with SSD may also have atypical responses to non-speech stimuli. Future studies will examine the responses of children with SSD to pure tones and other non-linguistic stimuli, such as environmental sounds, in order to determine whether they have a specific speech sound perceptual impairment or a more generalized non-linguistic auditory processing impairment. It is

a possibility that the neural responses to all auditory sounds in children with SSD may be attenuated, as compared to their typically developing peers.

If future research programs continue to identify perceptual processing deficits in children with SSD, this would suggest that speech treatment programs should include perceptual training, along with speech sound production training. In other words, deficits in acoustic-perceptual processing might be the source of speech production problems in children with SSD (Guenther, Hampson, & Johnson, 1998; Guenther, 1995; Munson, Baylis, Krause, & Yim, 2009; Perkell et al., 2000). That is, if children have difficulty encoding auditory information, this would result in the formation of an incorrect phonological representation; then, when children go to produce a word, they would retrieve the wrong phonological information, resulting in an inaccurate word production (Edwards & Lahey, 1998; Locke & Kutz, 1975). In support of this idea, it has been found that speech perception training can improve children's speech production (Rvachew, 1994; Rvachew, Nowak, & Cloutier, 2004). Thus, the usefulness of speech perception tasks in the assessment and treatment of SSD appears to be an avenue to explore further.

5. CONCLUSION

Based on prior studies of children with language impairment, reading disorders, and childhood apraxia of speech, it was hypothesized that children with SSD would have less specified phonological representations of speech sounds. It was predicted that this phonological underspecification would be characterized by small or no MMN responses during a speech sound discrimination task. The findings reveal that children with SSD did show smaller discriminatory responses to early-acquired speech sounds that they could produce correctly, as compared with TD peers. The present study is the first to use electrophysiological methods to examine the

neural mechanisms underlying SSD and provides some initial insight into how phonological representations might be established in children with SSD.

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Figure Captions

Figure 1. Adult-produced /ba/ and /da/ syllables used in the ERP study. Formant distinctions for the two oral stops are visibly different when comparing the second (F2) formant, which is indicative of place of articulation differences (bilabial for /b/ and coronal/alveolar for /d/). See Table 2 for formant measurement information.

Figure 2.

Section A. Top row: Grand-average ERPs elicited by the standard syllable, /ba/ (dotted line), and the deviant syllable, /da/ (thin black line) of the typically developing (TD) children. The deviant-minus-standard difference waves are represented by the thick black line. The MMN was most prevalent in the 300-400 ms window. Bottom row: Mean amplitudes of the MMN centered around its peak latency (300-340 ms); the voltage topography of the MMN ranged from $-3\mu\text{V}$ (dark blue) to $+0.5\mu\text{V}$ (dark red).

Section B. Top row: Grand-average ERPs elicited by the standard syllable, /ba/ (dotted line), and the deviant syllable, /da/ (thin black line) of the children with speech sound disorders (SSD). The deviant-minus-standard difference waves are represented by the thick black line. The MMN was most prevalent in the 300-400 ms window. Bottom row: Mean amplitudes of the MMN centered around its peak latency (325-365 ms); the voltage topography of the MMN ranged from $-3\mu\text{V}$ (dark blue) to $+0.5\mu\text{V}$ (dark red).

Section C. The deviant-minus-standard difference waves of the typically developing (TD) children (dotted line) and the children with speech sound disorders (SSD). Magnitude differences between the two groups were prevalent for both the MMN responses.

Figure 3. Fz Mean Amplitude x Participant Age correlational scatter plot. Older participants had slightly larger MMN responses at Fz than did younger children ($r = -.417$, $p < .05$).

Figure 4.

Section A. Fz Rectified Area x GFTA-2 Raw Score correlational scatter plot. Children with more errors on the GFTA-2 had smaller Fz rectified area measurements ($r = -.447$, $p < .03$).

Section B. FCz Rectified Area x GFTA-2 Raw Score correlational scatter plot. Children with more errors on the GFTA-2 had smaller FCz rectified area measurements ($r = -.473$, $p < .03$).

Figure 5.

Section A. Fz Rectified Area x GFTA-2 Percent Consonants Correct correlational scatter plot. Children who produced more consonants correct on the GFTA-2 had larger Fz rectified area measurements ($r = .499$, $p < .02$).

Section B. FCz Rectified Area x GFTA-2 Percent Consonants Correct correlational scatter plot. Children who produced more consonants correct on the GFTA-2 had larger FCz rectified area measurements ($r = .547$, $p < .007$).

Table 1. Characteristics of the SSD and TD subject samples (standard deviation)

	TD (n=12)	SSD (n=12)
Mean Age	5.17 (0.97) t(22)=-0.581, p > .56	4.94 (0.97)
GFTA-2 Standard Score	103.83 (6.45) t(22)=-5.460, p < .0001	77.83 (15.18)
GFTA-2 Raw Score (i.e., number of errors)	9.50 (8.42) t(22)=4.840, p < .0001	31.75 (13.52)
GFTA-2 Percent Consonants Correct (PCC)	89.83 (9.22) t(22)=-4.510, p < .0001	64.83 (16.84)
Leiter-R Standard Score	N/A	109.40 (15.43)
PPVT-IV Standard Score	N/A	108.40 (7.89)
Hearing	Within normal limits	Within normal limits

- The *Goldman Frisbie Test of Articulation -2* (GFTA-2; Goldman & Frisbie, 2000), *Peabody Picture Vocabulary Test – IV* (PPVT-IV; Dunn & Dunn, 1997), and *Leiter International Performance Scale – Revised* (Leiter-R; Levine, 1997) yield standard scores with M=100 and SD=15.

Table 2. Acoustic measurements of the consonantal sound, the consonant-vowel transition, and the vowel sound were measured for the two syllables used in the ERP study: /ba/ and /da/. See text for more details.

	Standard:	Deviant:
	/ba/	/da/
<i>Consonantal Segment</i>		
F1 (Hz)	827	449
F2 (Hz)	974	1826
F3 (Hz)	2676	2877
Time start (ms)	0	0
Time end (ms)	38	40
Measurement time (ms)	25	27
<i>CV Transition</i>		
F1 (Hz)	940	793
F2 (Hz)	957	1388
F3 (Hz)	2720	2629
Time start (ms)	38	40
Time end (ms)	71	104
Measurement time (ms)	63	78
<i>Vowel Segment</i>		
F1 (Hz)	839	942
F2 (Hz)	1180	1155
F3 (Hz)	2678	2740
Time start (ms)	71	104
Time end (ms)	375	375
Measurement time (ms)	243	244

Table 3. MMN peak latency, mean amplitude, and rectified area measurements at three fronto-central electrodes in typically developing (TD) children and children with speech sound disorders (SSD).

		MMN measurements (SEM)		
		Fz	FCz	Cz
Peak Latency	SSD	344 ms (10)	346 ms (10)	343 ms (9)
	TD	327 ms (14)	325 ms (14)	320 ms (13)
Mean Amplitude	SSD	-1.423 μ V (.723)	-2.106 μ V (.687)*	-2.556 μ V (.508)*
	TD	-2.933 μ V (.953)*	-3.890 μ V (.840)*	-1.924 μ V (.533)*
Rectified Area	SSD	.062 μ Vs (.024)*	.061 μ Vs (.020)*	.069 μ Vs (.025)*
	TD	.155 μ Vs (.028)*	.176 μ Vs (.031)*	.103 μ Vs (.016)*

**significantly different from zero at $p < .05$ or lower*

Ba
/ba/

Da
/da/

A.

*TD Children MMN Scalp Distribution
 300-340 ms post-syllable onset*

B.

*Children with SSD MMN Scalp Distribution
 325-365 ms post-syllable onset*

C.

A.

B.

A.

B.